PASCOE'S PRINCIPLES & PRACTICE OF EQUINE DERMATOLOGY



SECOND EDITION

DEREK C. KNOTTENBELT

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PASCOE'S

PRINCIPLES & PRACTICE OF

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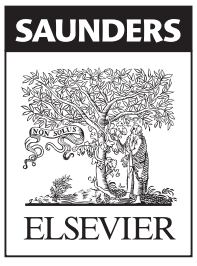
Second Edition

DEREK C. KNOTTENBELT OBE, BVM&S, DVM&S, DIPECEIM, MRCVS

Professor in Equine Medicine
The Philip Leverhulme Equine Hospital
University of Liverpool, UK

With a contribution by

JOHN W. MCGARRY MSc, PhD
Senior Experimental Officer/Parasitologist
Veterinary Parasitology Group
Department of Veterinary Pathology, Faculty of Veterinary Science
University of Liverpool, UK



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Foreword

Despite my early practice years being occupied with general veterinary practice, my real interest was always in equine practice. I was immersed in equine reproductive medicine and surgery. For me, the challenges of equine dermatology developed following the successful initial isolation of equine herpesvirus 3 (coital exanthema) and my interest and subsequent investigation into the occurrence of its pustular appearance on the genitals of both mares and stallions.

My interest was further stimulated by dermatophilosis (rain scald/mud fever) occurring throughout the year, but always related to rainfall. I was intrigued one wet winter, which is unusual for Queensland, by some spectacular outbreaks occurring on the long winter coats of the horses. This disease, while present during the summer, is usually missed due to the finer short hair coats. The summer type infection characteristically shows infection of the skin following water run-off patterns of the hair coat.

The widespread occurrence of dermatophytosis ('girth' or 'jockey' itch) in young racing horses became an absorbing interest over many years. The recognition of its highly infective nature, fomite transmission on the girth, jockey's boots, whips and saddles as well as brushes and curry combs, were all thoroughly investigated. Many of the enigmatic epidemiological questions were answered by the discovery of its longevity on the saddle girth in particular, allowing the spread from stored breaking-in saddles and girths for over 12 months after the initial contamination. This led to dramatic improvements in management and prevention.

My widening interest in all equine dermatological conditions was further stimulated by consulting with pathologists, particularly Danny Scott and the late Tony Stannard (USA), both of whom virtually founded equine dermatopathology. I cannot emphasize too strongly the importance of access to the dermatopathologists for discussion, guidance and interpretation of histopathological findings. Sharing the case details makes it more interesting for them too!

Clinical dermatology is a wonderful example of how satisfying it can be to apply clinical acumen, and observation aligned with the tenacity to understand the pathogenesis can lead to the satisfaction of confirmation through laboratory diagnosis. All the best components of practice are available to the equine dermatologist! Clinical dermatology lends itself to the almost obsessional use of a camera and today, with digital cameras and the increasing availability of better photographic equipment, the final images are very satisfying both as a clinical record and as a means of archiving interesting and ordinary conditions.

What does the spectre of climate change mean to our horses' skin? There will be shifts in disease occurrences – already we are seeing a southward drift of diseases such as pythiosis which is occurring 600–1000 km south of where it was restricted to 30 years ago.

So, what of the future?

Research in equine dermatology has been slow, probably due to the small number of purely equine dermatologists and the constant shortage of research funds. One of our greatest forward steps has been the slowly widening information and knowledge on equine sarcoids, their almost certain link with bovine papillomavirus DNA, and the increased interest in achieving a better outcome through genetic relationships of affected horses, or perhaps even the eventual isolation of specific genetic locations of immunity or susceptibility.

We need much more research into insect bite hypersensitivity – another disease that occurs universally and is a major welfare issue. The production of an effective desensitizing vaccine for insect hypersensitivity would help to prevent the intense irritability of our horses and the frustrations of their owners.

There will be countless new challenges and more opportunities in equine dermatology in the future and I would encourage practitioners to view dermatology as a challenging and interesting specialty, rather than a clinical annoyance!

I am very pleased that my name is being used in the title of this book. From the inception of our previous *Manual of Equine Dermatology*, much progress has been made in our understanding of equine dermatology and more clinicians are recognizing the significance of skin disease. This new book has a heavy practical bias and so it reflects my own philosophy in approaching cases that offer challenges and interest. It's great to know the pathogenesis and the background but ultimately we need answers – whether this is a treatment or a more accurate prognosis or both. I expect everyone who reads it will find it useful and that it will generate discussion, investigation and solutions!

Reg Pascoe AM

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Preface

This book represents the development of the previous *Manual of Equine Dermatology* and at the outset I would like to pay a tribute to Reg Pascoe, who has remained a true friend and a wise adviser to me and to countless others. His immense breadth of knowledge and experience in all areas of equine science is inspirational and his willingness to share, help, advise and commiserate has been my inspiration. I take on this book with the certain knowledge that I would be a fraction of the clinician I am without having met Reg, and the book would be a fraction of what it is without his contribution.

To the equine dermatologist the late Tony Stannard was an absolute saint! His quiet and authoritative advice was based on countless cases over many years and he remained up to his untimely death one of the most astute minds I have known. He persistently made the point that many equine diseases are given names which are derived from 'similar' diseases in other species but that there are often significant differences in the pathogenesis, pathology and clinical features. In many cases, for example sarcoidosis, systemic lupus erythematosus and urticaria, the differences are really sufficient to warrant separate names and so it has become common practice to prefix the diseases with 'equine'. He repeatedly made the point that it is probably unwise therefore to interpret the name of a disease as an indication of an identical pathological and clinical syndrome in other species. Consequent extrapolation of diagnostic or treatment protocols could be very disappointing.

The continuing popularity of the horse for both sport and pleasure has led to an ever-increasing need for veterinarians to update and upgrade their knowledge of equine diseases. Although the skin is the most visible and one of the largest organs of the body, its state of health is often overlooked. Skin disease in horses can be singularly distressing to many owners and so it is not surprising that if we, as animal health professionals, fail to solve problems, owners resort to often unproved and even harmful remedies to treat their horses. This frequently leads to a clinical deterioration and often an alteration in the clinical appearance of the condition. It can become increasingly difficult to reach either a definitive diagnosis or a successful outcome. I hope this book will address at least some of the problems of diagnosis and treatment.

The aim of this book is give the reader:

1. Confidence in his/her ability to recognize the normal variations and the changes that are implicit in dermatological disease (whether primary or secondary).
2. An effective clinical approach that minimizes the risks of a misdiagnosis and consequent errors of clinical management, and maximizes the good results and definitive diagnostic and therapeutic outcomes.
3. An opportunity to quickly access the methods of diagnostic confirmation and appropriate treatment.

The layout of the book is therefore designed to help students and veterinary surgeons with an interest in equine dermatology to improve their diagnostic and treatment skills and to help the busy (and perhaps the 'not so interested') practitioner make a rapid and effective dermatological investigation.

Chapters covering the basic understanding of skin anatomy and normal function (which is vital to the understanding of disease), general clinical examination of the horse (which is very important in dermatological cases), diagnostic tests (which are an almost inevitable event in many skin diseases) and the general principles of dermatological therapy (which is the ultimate demand of the owner) provide the basis for the subsequent sections.

It is easier to mistreat a rare disease than a common one but mistreatment of a common disease is almost unforgivable!

As an aid to the busy practitioner, each disease has a 'key points' box summarizing the major features of the condition. Within the box are numbered codes that should allow a busy practitioner to get instant information about the likelihood of the condition under the particular circumstances. Thus if the disease is seasonal and restricted to summer months (e.g. insect bite hypersensitivity) the code suggests that in winter this disease is very unlikely. Of course, familiarity with the codes will improve the value of the book overall but I have tried to make them as obvious as possible. The Syndromes section (Part III) provides a simple summary of the various major presenting signs encountered in equine dermatology and a differential list with cross references to the diseases themselves. This is a development of the flow charts in the previous edition which provided little extra information for the clinician beyond the name of the disease.

I am very pleased to include a section on the clinical recognition of skin mites, flies and other parasites contributed by Dr John McGarry of the University of Liverpool. Many parasitic conditions are highly frustrating and this is frequently because the parasite is not identified and the life cycle is not understood. Understanding the life cycles is important because it is possible then to identify 'weak points' where control measures can be applied. I hope this section (in Chapter 3) will help clinicians identify the various and often problematic parasites that afflict horses.

The hoof is basically just an extension of the skin and so I feel that it is justified to include a special section on disorders of the foot (excluding the orthopaedic and other internal disorders).

In spite of the generally high prevalence of cutaneous neoplasia in horses, equine cutaneous oncology is for the most part a poorly covered but important topic. Skin neoplasms are a persistent source of both clinical frustration and practice income! However, many of the specific neoplastic disorders are very rare; common things occur commonly! Accordingly, a separate chapter has been included with detailed diagnostic, treatment and prognostic information.

The 'syndrome' chapter includes a new approach to differential diagnosis with tables containing symbols/keys to help identify the common conditions and the salient features of differentiation. I hope this will encourage clinicians to further explore the differential diagnoses of the common broad categories of disease. These tables are intended to facilitate the recognition of unusual manifestations of common conditions as well as the rarer diseases, which can easily be

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overlooked. They are inevitably oversimplified and contain diseases which individual veterinary surgeons might never encounter and they should therefore be interpreted with care and clinical understanding.

The specific diseases are covered individually to provide a broad clinical understanding of many of the equine skin conditions. I have attempted to place greater emphasis on the commoner disorders while at the same time recognizing that the rarer ones are often more difficult to diagnose and treat. I hope that the reader will understand this relative emphasis and that we need to include the rarer disorders and those with limited geographical distributions. In each disease there is a list of possible differentials, and a brief note as to how these can be separated is given. These are specially wide in their variety so that as reference is made to the book in different areas and different diseases more information can be acquired and retained. Reference to the literature is not exhaustive – Danny Scott and William Miller's book *Equine Dermatology* contains every reference that a researcher might wish for!

Equine dermatology is considerably aided by visual appraisal but this can be a trap for the unwary, often leading to the diagnosis of rare conditions when, in fact, the horse may have a modified form of a common disease. Photographic material significantly enhances the value of a textbook on dermatology and I have attempted to illustrate as many of the disorders as possible with high-quality colour illustrations derived from many sources including Reg Pascoe (of course), Tony Stannard, Danny Scott, Marianne Sloet and many others, too numerous to list here (see acknowledgements). There are many new pictures in this edition and I hope that they will enhance the value of the book on the basis that a 'picture is worth a 1000 words'. It is not, however, possible to show every disease and variation of each disease and so clinicians should interpret the illustrations as a limited indication of the range of clinical appearances of the condition.

The wide variety of disease conditions means inevitably that there is some difficulty in trying to indicate which are common and which are rare. I have attempted to indicate the more common conditions but there is obviously a marked geographical variation in the prevalence of dermatological diseases. Furthermore, the breed, colour and type of horse as well as the specific husbandry practices have a significant effect on the incidence of disease. Veterinary surgeons will inevitably become aware of the more common diagnostic options in their own localities.

Air transportation and faster and more frequent movement of horses between countries mean that a wider range of disease conditions can be expected to be presented for diagnosis and treatment. It is particularly important to be aware of the possibility of spread of serious epidemic disease from this source. I have tried to include these wherever possible while recognizing that many veterinary surgeons may never encounter them. Furthermore, hitherto geographically restricted diseases may appear almost anywhere in the modern world. A geographical restriction code has been included in the key points boxes for those diseases that are currently regarded as restricted to limited areas of the world. Readers should interpret these with care and recognition that they are for the most part reliant upon literature reports alone.

Initially, readers might like to use the first part of the book as revision of the basic aspects of dermatological diagnosis and treatment. When faced with a dermatological case, reference could then be made directly to the chapter on syndromes. Having considered the most likely diagnosis from this, specific details regarding the confirmation of diagnosis and the suggested treatment can be obtained from the section on conditions, which is divided initially into the infectious and non-infectious diseases. I have included a short sentence or brief notes in the differential diagnosis of each condition to provide the busy practitioner with some diagnostic help. I have tried to vary these a bit so the reader can gradually learn more about the various diseases that are included in the differential diagnosis list. I hope this helps!

Inevitably in a clinically orientated book, there will be large areas of theoretical information that are not present. I have intentionally avoided the inclusion of histology and pathology, apart from the section that relates to the likely pathological descriptions a clinician will encounter in surgical pathology reports, because this is usually of incidental interest to the clinician. The reader's attention is drawn to *Veterinary Immunology: An Introduction* by Ian Tizard published by WB Saunders, Philadelphia, USA, 2000. This is an exceptional and very understandable text that covers aspects of immunology carefully and comprehensively. Similarly James Rooney and John Robertson's book *Equine Pathology* published in 1999 by Iowa Sate University Press, Ames, USA, provides an understandable and carefully balanced pathological description of many of the important skin disorders. *Pathologic Basis of Veterinary Disease* by Don McGavin and James Zachary (Mosby, 2006) is an amazing resource – it is a fascinating read and is commended to every practitioner whether dermatologist or not. *Equine Dermatology* by Danny Scott and William Miller (WB Saunders, 2003) is the definitive reference text in equine dermatology and it is inevitable that anyone wishing to pursue a specialist interest in dermatology will have a copy of this dermatological 'bible'.

Equine dermatology is a poor relation when it comes to professional enthusiasm and research effort but it can be a rewarding, challenging and lucrative specialty. The technical requirements for a dermatologist are minimal, desperate (midnight) emergencies are very rare and with effective treatment many cases can be cured or at least managed adequately for purpose. Furthermore it is a visual extravaganza! I hope this book will add to the available information on equine dermatology and will bring new enthusiasts into the subject.

I commend this book to the reader with humility and the recognition that many people know a great deal more than me about much of the material. I am still learning something new every day and often lots of things every day – 'the more we see, the more we realize we need to learn'. I welcome and encourage opportunities to share information in the field of equine dermatology with any interested party and I hope that this book will generate discussion and develop the specialism of equine dermatology.

In the words of the late Tony Stannard:
Equine dermatology is fun … if you try it, you will believe it!

Derek Knottenbelt

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Acknowledgements

Firstly I wish to pay tribute to my wife Morna who has yet again tolerated the late night vigils of the aspiring author with resigned good humour – her counsel as a small animal dermatologist and her constructive manuscript criticisms have always been very valuable and invariably correct!

Reg Pascoe continues to inspire us all and he has provided significant numbers of pictures and the textural components of this book carry an inherent 'Pascoe' style derived from his input into the previous edition – he is the definitive clinicians' clinician. The name of this book is not only a tribute to his clinical and dermatological contributions to the profession but to his amazing enquiring mind.

It would also be quite impossible to fail to recognize Danny Scott in the construction of any dermatology book – his inspiration as a dermatologist is legendary. His outstanding *Equine Dermatology* written with William Miller and published in 2003 has to be the definitive guide. My other heroes in equine dermatology are Marianne Sloet and Rob Pilsworth who have provided challenge and advice for many years. I am grateful to Professor Don Kelly and Dr Udo Hetzel for their help with pathological support.

I remain extremely grateful to my good friend Denis Duret, IT Officer of the Faculty of Veterinary Science, University of Liverpool for his continuing help with the figures and flow charts and other help throughout this project. Barrie Edwards prepared some of the graphic drawings and I am very grateful for this help.

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Dedication

This book is dedicated to Reg and Joy Pascoe and its title is a tribute to them both. When I first met them I knew I was in the presence of special people and that sentiment has increased with each of our all too infrequent meetings.

I wish them a long and fruitful 'walk-about' in their retirement – lots of cool and relaxing dawns, amazing sunsets, warm evenings and plenty of new experiences!

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‘Key point’ codes

A ‘key points’ box headed by a set of coded numbers is included for each disease described in Part II of this book. These boxes provide basic information only, but they should allow a degree of disease recognition and give effective and rapid guidance on basic therapy. Thus, if the disease is seasonal and restricted to the summer months (e.g. insect bite hypersensitivity), the code suggests that in winter this disease is very unlikely. Familiarity with these codes will improve the value of these boxes.

Key points:

Code number	Descriptor	Description
1	**Geographical restriction**	Geographical restrictions are sometimes absolute (i.e. the disease can only be found in the defined area) but in other circumstances are due to specific control measures that have been used to prevent introduction of disease to a population of horses. As a result of easy and universal rapid travel for horses, it is unwise to assume that a geographically restricted disease cannot ever be found outside those boundaries where the disease currently occurs.
2	**Hereditary**	Diseases/disorders with a genetic origin that can be passed on from parent to offspring. There are variations in the heritability of the conditions.
3	**Congenital**	Congenital disorders and malformations are not necessarily hereditary. Most of the conditions are present at or shortly after birth but some (e.g. hereditary equine regional dermal asthenia) are possibly hereditary and only appear after some months or years.
4 (0+) 4 (1+) 4 (5+) 4 (15+) 0–1 years 1–5 years 5–15 years >15 years	**Age-restricted**	The rough age of any restriction is shown in the symbol. Conditions that have a congenital origin are usually, but not always, manifest at or soon after birth. Some conditions are restricted to older or younger horses.
5	**Seasonality**	Diseases that are seasonally restricted are usually fairly well recognized and the causes can usually be attributed to some aspect of the weather, management or the seasonal presence of a causative agent such as *Culicoides* spp. midges or particular feed substances.
6	**Contagion**	Indicates that the disease can be spread from horse to horse. Spread can be either direct or indirect through fomites but the term does not apply when vectors are involved. Most contagious conditions are infectious but some infectious conditions are NOT contagious.
7	**Other animals**	A disease that is transmissible to other animals (other than human).
8	**Zoonosis**	A disease that is transmissible to humans.
9	**Nodular**	Diseases characterized by focal skin thickenings larger than 0.5 mm in diameter.
10	**Alopecia**	Hair loss is present within the area of the condition. This may, of course, be focal. Alopecia only refers to failure of hair production or pathological shedding of hair; the term includes hairs damaged by disease process but not hair that is rubbed out by harness or by self-trauma.
11	**Crusting**	The presence of dried exudate (including plasma, blood, pus or accumulated body fluids of any sort such as milk, urine) that has dried into and onto the skin and hair. This is markedly different from scaling in both appearance and clinical implication, although both can exist at the same time.

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Key points: continued

Code number	Descriptor	Description
12	**Scaling**	Excessive exfoliation of the superficial layers of the epidermis results in the clinical appearance of scaling. An alternative name for this is seborrhoea. This is markedly different in both appearance and clinical implication from crusting, although both can exist at the same time.
13	**Pigmentary changes**	A significant indicator of skin disease. Some conditions cause profound changes in hair colour or in skin colour, or both.
14	**Single focus**	A single isolated or occasional few isolated lesions are present.
15	**Multifocus**	Disorders that have several (often many) similar foci on the skin or mucous membranes. Involvement of different tissues, such as feet and skin or coronary band, chestnut and oral mucosae, is included.
16	**Generalized**	A generalized disease occurs over wide areas of the body; there may or may not be areas where focal lesions occur within those regions.
17	**Pain**	An important clinical sign in dermatology, which can vary from mild to extreme. The same condition can cause more or less pain in different horses, as some appear to have a low pain threshold while others, such as ponies and donkeys, have a very high one. It must be differentiated from pruritus.
18	**Pruritus**	A significant clinical sign in dermatology. There are many causes of pruritus, some of which are infectious in origin. Pruritus is defined as the unpleasant sensation that provokes the desire to itch or rub; it must be differentiated from pain.
19	**Systemic effects**	Cutaneous disease can have systemic effects and systemic disease may have cutaneous effects. Both of these circumstances are denoted by this code.
20	**Persistent**	A persistent disorder is one that does not resolve at all, even though there may be some degree of variation in severity.
21	**Intermittent**	Applies to conditions that have an unpredictable recurrence at very irregular intervals.
22	**Recurrent**	Applies when the condition recurs at regular or irregular intervals. Seasonality may be regarded as a form of recurrence but this category is only used when the condition resolves completely between episodes.
23	**Pathognomonic pathology**	Examination of samples collected either by fine needle aspirate or biopsy will reveal the true diagnosis in all (or at least the large majority of) cases. Sometimes very specialized tests will be required, such as immunohistochemical staining of sections.
24 Good; 24 Guarded/ Moderate; 24 Poor; 24 Hopeless	**Prognosis**	The prognosis of any disease or disorder will vary markedly from animal to animal but there are some general rules that apply. Green = good prognosis; yellow = guarded or moderate; orange = poor; red = hopeless prognosis. Prognosis will, of course, vary according to the availability of treatments; some diseases will have a good prognosis even if they are not treatable.
25	**Reportable**	The reportable diseases will vary in different parts of the world, but most countries have a statutory policy for the eradication and/or control of serious (usually infectious) diseases.

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1 Introduction

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More than 100 years ago, the noted medical pathologist Virchow (1860) portrayed the skin as a protective covering for more delicate and functionally sophisticated internal viscera. At that time, the skin was primarily treated as a passive barrier to fluid loss and mechanical injury. The modern approach is to consider the skin as a highly complex organ in which precisely regulated cellular and molecular interactions govern many crucial responses to environmental changes.

The skin has many functions and apart perhaps from the liver, it could be said to have the most diverse range of 'duties' of all the major organs. Each of these functions is involved either individually or with others in the pathogenesis of skin disease (Table 1.1). The skin plays a major role in:

1. Protection against physical, chemical and microbiological agents
2. Protection of the internal milieu against fluid, protein and electrolyte loss, and from external wetting/maceration (waterproofing)
3. Thermoregulation (sweating and heat conservation)
4. Blood pressure regulation
5. Synthesis of vitamin D
6. Sensory functions (touch and other specialized receptors)
7. Storage of fat, water, vitamins, carbohydrates and protein
8. Immune functions within both the inner and outer layers of the skin.

In addition, the skin provides a means of identification and individual recognition. Embryologically the skin is modified to create specialist structures such as the cornea and the hoof.

1. *Physical protection.* This is afforded by the inherent strength and elasticity of the integument. The skin varies in its thickness (being thickest on the legs and back and thinnest on the eyelids, the scrotum and medial thighs). The integrity of the physical barrier is important for protection against traumatic insult, solar radiation, and potential pathogens (viruses, bacteria, fungi and other parasites). Protection against bacterial attack is afforded not only by the physical barrier of the skin – the sebum and cellular components play an active role in preventing bacterial invasion and recognition of potentially damaging pathogens. Breaks in the skin integrity and alterations in its cellular and immunological function can result in pathological

Table 1.1 The major functions of the skin that affect dermatological health

Function	Mechanism/cells responsible
Physical protection:	
Chemical/physical insult	Keratinocytes
Ultraviolet radiation	Melanocytes
Antigens/haptens	Langerhans cells, dermal lymphocytes, polymorphonuclear cells, mast cells
Microorganisms (virus/bacteria/fungi/parasites)	Keratinocytes, Langerhans cells, mononuclear phagocytes/mast cells
Prevention of water/electrolyte/protein loss (internal waterproofing)	Keratinocytes
External waterproofing (protection against external wetting)/lubrication	Keratinocytes/sebaceous glands/hair
Shock absorption	Collagen elasticity in dermis/subcutaneous fat
Sensation	Range of specialist sensory organs for pain, touch, heat, moisture, etc.
Thermoregulation (prevention of external heat gain and internal heat loss)	Dermis/subcutaneous fat/blood vessels/lymphatics/sweat glands
Calorie/energy reserve	Subcutaneous fat
Vitamin D synthesis	Keratinocytes
Individual recognition	Hair follicles (colour patterns)/sebaceous glands

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infections. For example, fungal infections such as dermatophytosis are common in horses but these infections require skin damage/trauma before disease can develop. Immunocompromising disease such as Cushing's disease can result in opportunistic skin infections including bacterial, fungal and parasitic diseases.

A healthy skin is a strong ally in the fight against infection and skin insult.

2 *Protection of internal milieu.* The skin provides an effective barrier against evaporation. The skin is also virtually waterproof as a result of the physical arrangement of the epidermis and the secretions of the sebaceous glands. Where the skin is damaged over an extensive area (e.g. in burns and skin wounds) there may be a significant loss of blood and/or blood proteins. This might result in a debilitated animal that is less able to fight potential pathogens.

The integrity and health of the skin is a major factor in the maintenance of the internal milieu.

3 *Thermoregulation.* The skin has a significant blood supply (being one of the largest body organs) and thermoregulation is a major function. In cold conditions the cutaneous blood vessels are narrowed to preserve heat loss and in high temperatures they are dilated to help heat loss. Subcutaneous fat also provides a thermoregulatory function. Sweat glands provide an additional heat loss mechanism – horses have a rather unusual regional and patchy sweating ability that results from cutaneous vasodilation and catecholamine release. Some areas have a greater ability to sweat than others. In donkeys the mechanism for sweating is significantly different. They will only start to lose sweat when their body temperature has risen significantly and they retain their sweat at the skin level as opposed to the outside of the hair coat. This means that donkeys are better able to withstand high temperatures and water deprivation.

The skin is the major thermoregulatory organ for the horse.

4 *Blood pressure regulation.* Cutaneous vasodilation and vasoconstriction are a major means for control of blood pressure. The mechanisms for this function are controlled by catecholamine release and autonomic nerve functions.

5 *Vitamin D synthesis.* It is easy to forget that the skin plays a major role in calcium homeostasis within the body and in this way has an influence on the function of many major organs. The vitamin D precursor 7-hydroxycholesterol is converted to the vitamin D precursor pre-vitamin D_3, which in turn is a vital component of the active form of vitamin D.

Vitamin D synthesis is dependent on exposure of the skin to sunlight (ultraviolet light).

6 *Sensory functions.* The skin is the major organ of touch and thermal sensation. There is a plethora of sensory nerve endings in the skin that allow perception of heat, cold, wet, dry, pressure and pain as well as many other sensations. Skin that is deprived of its nerve innervation may suffer insult without any self-protective reflexes. Also the neurological functions are responsible for sweating and blood supply.

The skin is the biggest sensory organ of the body and as such is a vital component of survival.

7 *Metabolic store.* The subcutaneous fat deposits are an important active aspect of weight maintenance. In some cases the deposition of fat can be excessive and in others there may be an absence of fat. Under normal conditions fat deposition takes place in summer and during periods of plentiful food while mobilization of skin fat occurs in cold weather and in nutritional deprivation.

The subcutaneous fat depots are important survival mechanisms.

8 *Immunological function.* The skin plays a very active role in the recognition and recall of infectious and non-infectious challenges. Historically the skin was regarded as a passive organ but this is far from the case (see below, p. 11).

The skin is a major immunological organ and impaired immunity has profound effects on the health of the skin and the animal as a whole.

The cosmetic appearance of the skin is important both for individual recognition and in some cases for the function of the domesticated horse. Under normal conditions a healthy equine skin is lustrous and non-greasy. The mane and tail hairs are lustrous, long, strong and very flexible. In winter the body hair coat is longer, somewhat greasier to the touch and often lacks lustre. The mane and tail hairs do not alter between summer and winter.

Cosmetic problems often cause more concern to owners than some more serious diseases. This means that owners may overlook serious conditions while over-emphasizing some trivial ones.

Note

Without resort to any sophisticated equipment, every clinician has ample opportunity to visually examine and palpate the skin in every case!

Dermatological disease is one of the commonest clinical presentations in equine practice. This is possibly due to the fact that skin is the largest organ system, it may be altered by a number of exogenous and endogenous factors, and owners usually recognize cutaneous lesions easily. It is easy to overlook the fact that the hoof, ergots, chestnuts and cornea are modifications of the skin and their diseases could justifiably be included in dermatology. However, it is also true to say that many cutaneous diseases are overlooked, trivialized or belittled as being 'cosmetic' nuisances whilst they are more often significant than not. There are many cutaneous diseases that have systemic effects and many systemic disorders that have significant and diagnostically helpful cutaneous manifestations; in some cases the skin signs are both the earliest evidence of disease and indeed the most obvious. This makes differentiation between primary

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and secondary skin disease a significant aspect of the diagnostic process and it remains disappointing that the skin is often overlooked during clinical investigations.

Obvious lesions in the skin are usually easily identified but subtle changes in skin temperature, thickness, colour, hair density and sensitivity can sometimes be difficult to locate and investigate in horses. Deviations from normal may reflect primary skin disease, for example viral, bacterial or fungal infections or insect hypersensitivity, and/or secondary manifestations of primary systemic disease, such as the cutaneous photosensitization manifestations of advanced liver failure. The latter are relatively common. Also, the skin may act as a significant indicator of other systemic disease and may reflect the horse's general health status. Clinicians and owners often report 'a staring harsh coat quality' in horses with significant systemic diseases. The same signs could be induced by measures applied by the owner also; for example repeated washing in severe degreasing shampoos, detergents and soaps can result in a grossly abnormal skin appearance and texture.

Note

The skin provides a great deal of visual information about its own health and about the health of the animal it covers. No other organ system is so easy to assess visually and palpably.

The key to successful treatment is accurate diagnosis. The basis of disease diagnosis must, by necessity, be guided by a thorough knowledge of what may be regarded as normal. This is usually a skill acquired by regular and careful scrutiny of many ostensibly normal horses so that subtle (possibly early) abnormalities can be identified quickly. Late diagnosis may sometimes make the investigation far easier – the lesions are advanced and therefore may be very typical for any particular condition. However, in these late stages there may be secondary changes that may confuse a diagnosis even if specialized tests are applied. Early lesions may be very subtle and this can introduce diagnostic and therapeutic problems. Furthermore, early diagnosis is often difficult because of the limited number of ways in which skin can react to the full range of infectious and non-infectious challenges; different diseases can have remarkably similar overt appearances (Fig. 1.1).

Careful and critical assessment of presenting clinical signs and an understanding of their significance in relation to a disease process, combined with a basic understanding of skin morphology and function, allow the clinician to categorize skin disease into broad groups such as nodules, alopecia, change in hair/skin colour. This categorization allows a clinician to reach an accurate diagnosis more easily and accurately than if attempts are made to guess a diagnosis without knowing all the facts. However, in practical circumstances, there are few cases in which a single category/syndrome is strictly applicable because most skin

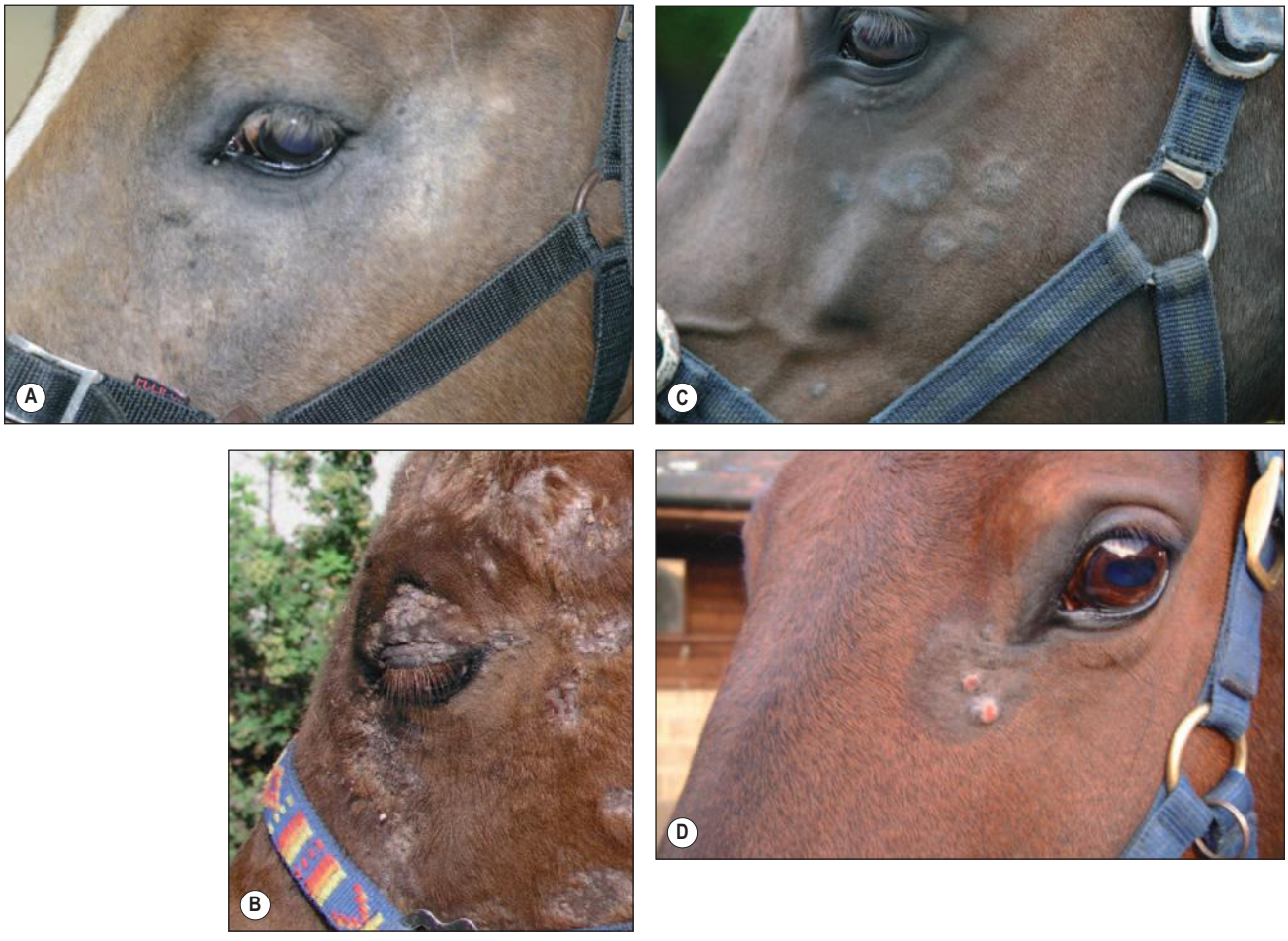


Figure 1.1 Four different periorbital alopecic and flaking disorders. They have a broadly similar appearance but there are very different implications for diagnosis, treatment and prognosis. **(A)** alopecia areata; **(B)** pemphigus foliaceus; **(C)** dermatophytosis/ringworm; **(D)** mixed sarcoid.

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diseases either have multiple presenting signs or are complicated by secondary changes that mask the underlying primary condition. Also, categorizing skin diseases into defined syndromes as opposed to identifying the primary cause of the condition carries dangers. Significantly different diseases can be remarkably similar to the casual observer (Fig. 1.1).

A single term for the clinical appearance of a cutaneous lesion may be very descriptive but subtle variations may perhaps not then be clearly identifiable. A broad categorization of a type of lesion is also not usually helpful. For this reason terms such as 'eczema' and dermatitis should be avoided as far as possible even though owners will often be satisfied with such an approach simply because these terms are commonly used in humans. For example, a diagnosis of 'nodular skin disease' could cover several distinctly different clinical entities and each of these would surely require very different therapy (Fig. 1.2 and Table 1.2).

The written description of various skin lesions can be both poorly understood and poorly used, and the specialized vocabulary of the dermatologist may be daunting. Familiarity with the dermatological vocabulary allows a rational scientific dialogue between veterinary practitioners at all levels and dermatopathologists, reducing confusion and materially improving diagnostic accuracy. These terms and descriptions make the description of well-recognized lesions more universally understood by both dermatopathologists and clinicians. A short glossary of the common terms and expressions is included at the end of this chapter (p. 21). Their use serves to reduce confusion in written reports and dialogue and increases understanding of skin conditions but can lead to oversimplification. Although specific descriptive terms can sometimes be open to misinterpretation, regular usage will minimize this risk.

Normal equine skin

The normal anatomical structure of equine skin broadly follows the classical descriptions shown in many textbooks of anatomy (Fig. 1.3). However, there have been fewer studies

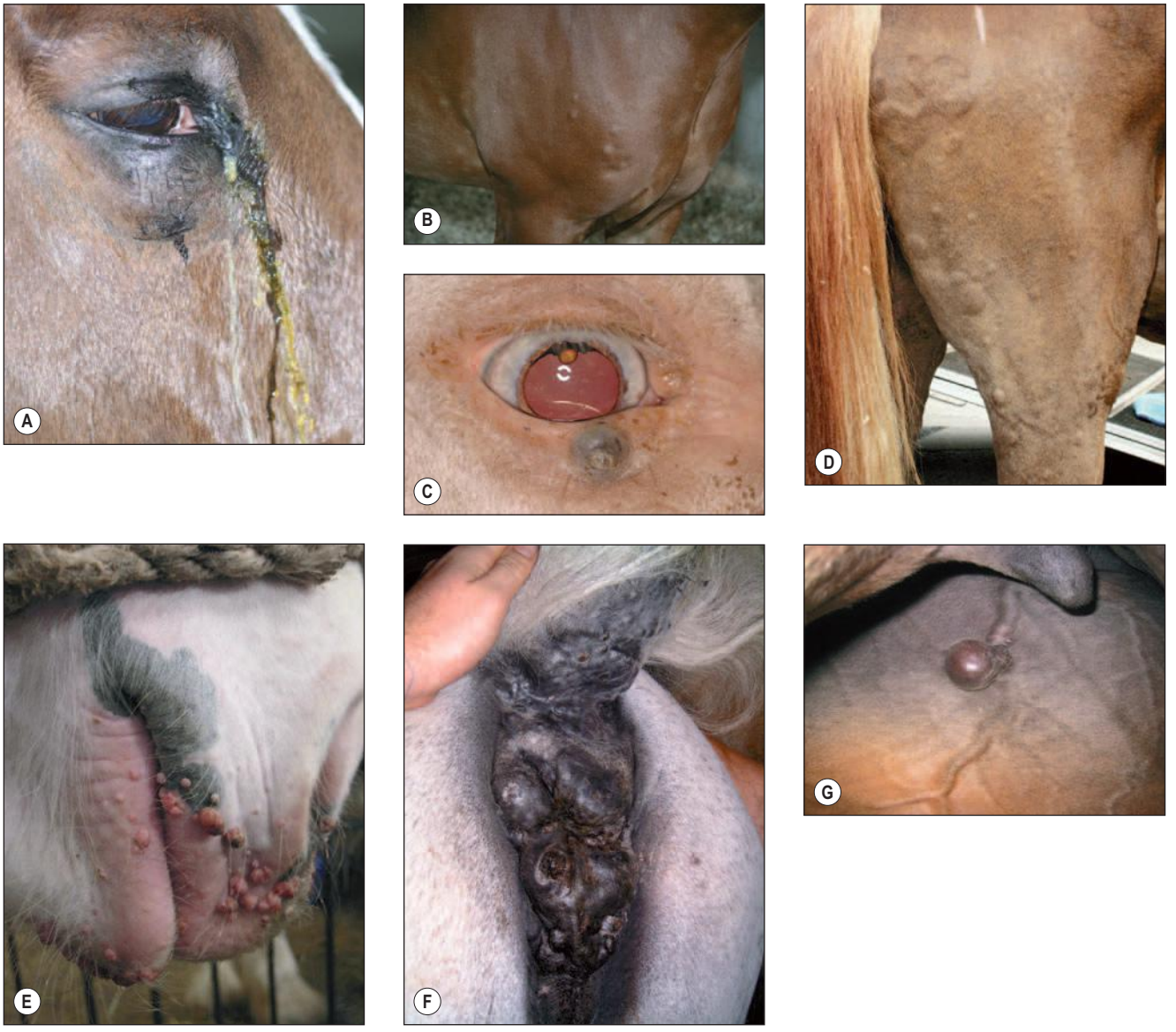


Figure 1.2 The seven skin nodules shown here are very different diseases, they have different prognoses and each requires a very different treatment. **(A)** subconjunctival squamous cell carcinoma; **(B)** fly bite reactions; **(C)** cutaneous melanoma; **(D)** urticaria; **(E)** viral papilloma; **(F)** malignant melanoma; **(G)** sarcoid.

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Table 1.2 Examples of some skin nodules illustrating the differences in treatment and prognosis

	Disease type	Treatment	Prognosis
Squamous cell carcinoma	Neoplastic	Radiation required	Poor
Fly bite reaction	Transient local hypersensitivity reaction	None required	Excellent
Localized melanoma	Cutaneous neoplasm	Surgical preferred	Very variable
Urticaria	Transient/recurrent allergic response	Medical treatment may be required	Good
Viral papilloma	Viral fibropapilloma	Treatment may not be required – surgical removal can be helpful	Excellent
Malignant melanoma	Cutaneous neoplasm with metastatic potential	Neither medical nor surgical treatment can help	Very poor

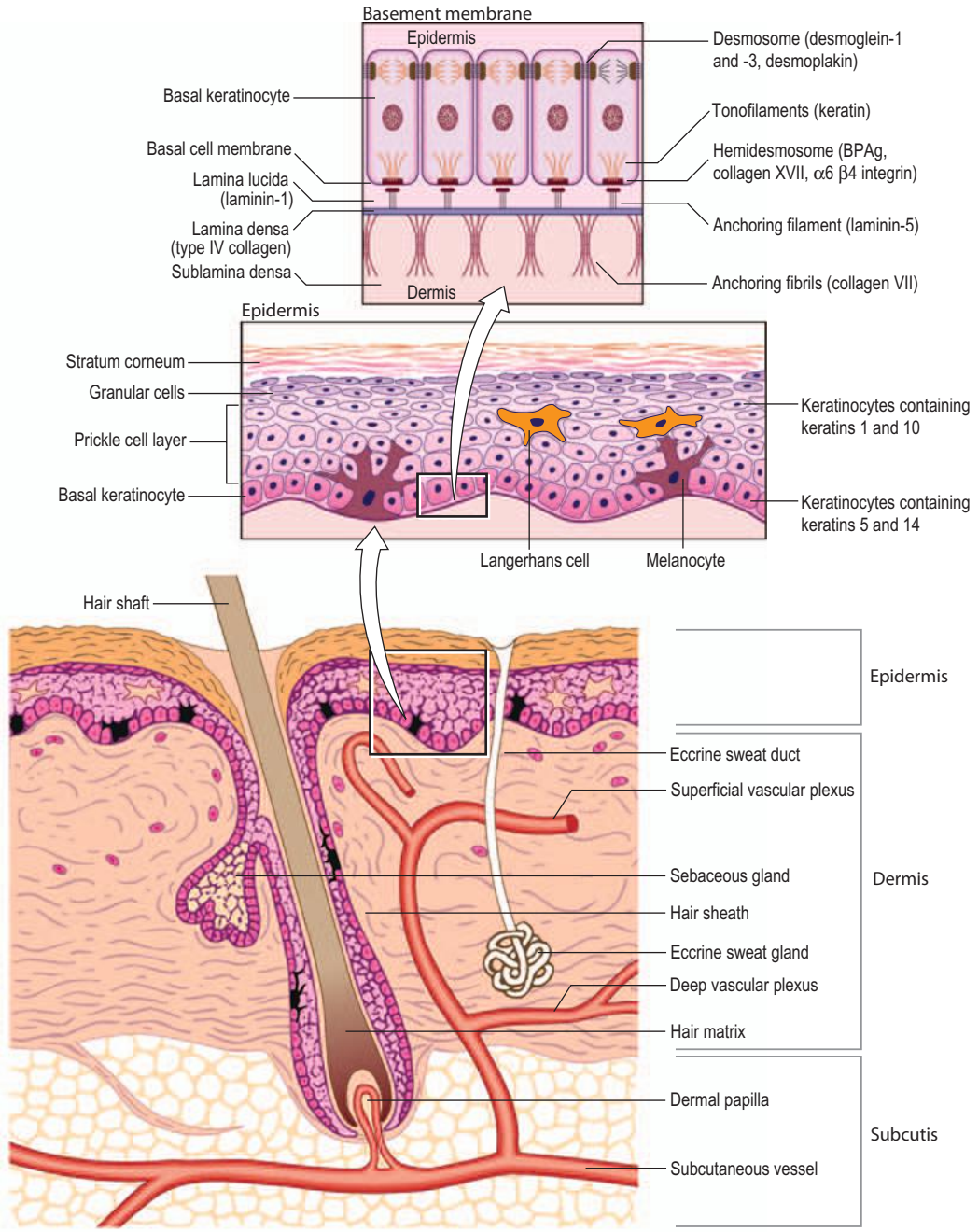


Figure 1.3 Structure of normal skin. (Adapted from Boon N A et al, 2006, *Davidson's Principles and Practice of Medicine*, 20th edn. Churchill Livingstone, Edinburgh; with permission.)

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on equine skin than for many other species and so significantly less is known about its particular function, the cutaneous blood supply and particular cellular behaviour patterns. Extrapolation from the other species seems an unreliable way to understand the skin of a highly adapted species but until more research is supported equine dermatology will remain behind that of other species in this respect.

Skin structure varies greatly at different sites and among the different equine species. There are some significant differences, however, in the horse. For example, the skin is particularly well endowed with sweat glands and the horse sweats more easily and in different patterns from most other species. In addition there are specialized skin structures such as the eyelashes, the sensory or tylotrich hairs and the ergots and chestnuts.

Evolutionary adaptations have had a significant effect on the structure and function of equine skin. There are significant differences in the skin of the various members of the equidae, with the donkey in particular being particularly well adapted to extremes of heat and dryness. Without skin adaptations the donkey could not survive in the climates where its relationship to man is crucial. The donkey survives the rigours of extreme climates by having the ability to increase its body core temperature by 1–2°C without harmful effects and its mechanisms for thermoregulation through sweating are highly efficient, involving minimal water loss for maximal cooling effects. Even within the horse species there are significant differences in skin thickness and hair coat growth patterns that may have an influence on the prevalence of some of the disorders. In most horses, however, the natural periodicity of hair coat growth is adapted for the circumstances the horse is in. In hot climates, even the winter coat is short and thin whereas in cold climates the same horse would grow a long, dense, protective and warming hair coat. Even the use of rugs and blankets can have a profound effect on hair growth patterns.

Seasonality of coat character is an area that is poorly understood in the horse, but the changes are very predictable both in extent and type. The growth patterns of the hair coat have a seasonal relationship that is strongly and predictably dependent upon the daylight hours but also significantly affected by other factors such as temperature.

Colour patterns and the position of hair whorls are used widely as forms of identification but there are some important variations. For example, a foal may be born ostensibly chestnut or bay and then turn grey and some breeds such as the Appaloosa may change the patterning and clarity of their skin colour spots with age.

The mane and tail have a different type of hair – both have thicker, stronger and longer hair. Disease may affect one or more components of the skin/hair types on a horse; the mane may be spared while the body hair is badly affected or the legs may be spared while the body and mane can be severely diseased. White-skinned limbs have a deserved reputation for greater susceptibility to some photoactivated and some infectious disorders but the reasons for this are not known; the physiology and microscopic structure of the white skin is indistinguishable from pigmented skin apart from the obvious lack of melanocytes.

The skin is continuous with mucous membranes at the body openings and this is a common site for subtle dermatological signs to develop. The same can be said of specialized skin junctions such as the coronary band.

Gross and histological anatomy of the skin

The skin consists of epidermis, dermis, hair follicles, sebaceous glands, sweat glands and digital appendages, for example chestnuts, ergots and hooves. One of the most difficult aspects of dermatological investigation facing the veterinarian is establishing what is normal for an individual horse. Within the species there are also significant breed and even individual differences in hair coat density and quality and even distribution and so it is important that the breed, season and colour are always considered. An apparent dermatological disorder could in fact be normal for that animal and whilst most 'genetically' based changes are present from birth there are some that develop in later life.

Note

- The haired skin is thickest over the dorsum and lateral aspect of limbs and is thinnest on the eyelids, ventrum and medial aspect of the thighs.
- The subcutis or panniculus connects epidermis and dermis with the underlying fascia and musculature.

There is increasing evidence that all the cells that comprise the skin including the keratinocytes have active roles as well as passive ones.

Epidermis

The epidermis is an avascular stratified squamous epithelium and is firmly attached to the dermis by a complex basement membrane (Fig. 1.3). This structure is important in some well-recognized skin diseases such as the pemphigus complex.

Keratinocytes comprise over 95% of the epidermal cells. They are attached to each other by complex adhesion proteins known as desmosomes. They synthesize insoluble proteins called keratins of two main basic types. These are conventionally described as 'basic (type 1)' and 'acidic (type 2)'. Individual keratins of each type pair up to form filaments which provide the basic structure to the skin (cytoskeleton). They also synthesize lipid-rich cornified material primarily responsible for the protective barrier and waterproofing functions of skin. They are derived from stem cells in the basal layer of the epidermis and move outwards, gradually losing their shape (becoming flatter). The nucleus degenerates and they become degenerate flattened anuclear cells in the most superficial horny layer of the skin where they are surrounded by the lipid-rich hydrophobic material that 'cements' them together. Finally the cells are sloughed/shed from the surface as fine scales (dander/dandruff/scale). Some pathological conditions are characterized by excessive scaling and flaking but a continuous low level of shedding of defunct keratinocytes, keratin strands and lipid is a normal physiological function of skin. A slightly scurfy skin character is normal on horses.

Classically the epidermis is divided into defined layers although this is a gradual change rather than a defined, abrupt one.

- The stratum corneum is the outermost epidermal layer and comprises many sheets of flat keratinized (anuclear) cells (keratinocytes) that eventually slough from the surface. Non-haired skin tends to have more layers in

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the stratum corneum than haired skin. This layer varies markedly in many disease conditions, being either excessively thick or abnormally thin.

- The stratum lucidum is absent in haired areas and is a thin layer of compacted, flattened, fully keratinized cells covering the granular layer or stratum granulosum. It consists of effete cells with basophilic keratohyalin granules. The stratum granulosum is absent or minimal in haired skin but prominent in hairless (glabrous) skin.
- Beneath these layers is the stratum spinosum, which consists of a zone of polyhedral cells attached by desmosomes; these intercellular bridges can be recognized histologically as 'spines'. The innermost layer of the skin is the germinal layer or stratum basale, consisting of a single layer of mitotically active cuboidal to columnar cells resting on the basement membrane. There are abundant stem cells distributed in the stratum basale.

Intermixed in the basal cell layer are melanocytes, Langerhans cells and Merkel cells.

- *Melanocytes* synthesize melanin pigment from tyrosine, package it in the intracellular melanosomes and transfer the pigment to surrounding keratinocytes and hair bulbs via their dendritic processes, thereby imparting colour to the skin and hair. They are also present in the basal layers of the stratum spinosum.
- *Langerhans cells* are dendritic cells derived from bone marrow that circulate between the epidermis and the local lymph nodes. They act primarily as immune surveillance cells and are the major antigen-presenting cells of the skin. Antigens are presented to T lymphocytes and may be vital in viral and bacterial infection and in tumour cell recognition. It is possible to stain for these cells specifically but they cannot be identified on normal haematoxylin and eosin stained sections.
- *Merkel cells* are neuroendocrine cells and function as slowly adapting mechanoreceptors for touch sensation. A few Merkel cells are also found in the basal layer of the epidermis.
- *Intra-epidermal lymphocytes* are known to be T cells in the mouse and human (and likely to be similar in horses). They monitor the skin surface for extraneous potentially harming antigens.

Dermo-epidermal junction and basement membrane

The basement membrane acts as an anchoring system for the epidermis but it still allows the relatively free movement of cells and nutrients between the dermis and the epidermis. In the horse, the basement membrane zone is thick and relatively easy to define histologically. This is typically straight in hirsute animal skin due to the extra anchoring function of the hair follicles. The structure of the basement membrane is identifiable by electron microscopy and is typical of all basement membranes under epithelia. It attaches to the basal cell layer of the epidermis by hemidesmosomes.

Dermis

The dermis is the vascular part of the skin that structurally and nutritionally supports the epidermis. It also supports blood vessels, nerves and adnexal structures including the hair follicles and sweat glands. It is divided into superficial and deep layers; the superficial dermis has finer, loosely arranged collagen fibres while the deeper dermis has a denser collagen fibre arrangement. Its thickness varies at different anatomic sites and this accounts for the main differences in the physical nature of the different sites on the body. In the horse, the dermis is thinnest in the eyelids, the medial thigh and axillae. In the neck, gluteal and sacral regions a deeper third layer is present, which is composed of fine collagenous fibres interwoven with fine elastic and reticular fibres.

Fibroblasts comprise the majority of the cellular components of the dermis; fibroblasts synthesize the extracellular matrix fibres and the ground substance. Mast cells, mononuclear phagocytes (macrophages), lymphocytes and Langerhans cells are distributed throughout. Eosinophil and neutrophil leukocytes are occasionally distributed in normal dermis.

The dermis consists mainly of collagen (type I and type III) and elastic fibres (elastin and reticulin) in a ground substance largely containing glycosaminoglycan, hyaluronic acid and dermatan sulphate, all of which are synthesized by fibroblasts. The production and catabolism can be influenced by endocrine factors although these are probably less obvious in the horse than in other species.

Cutaneous arteries give rise to three intercommunicating vascular plexuses. The deep plexus is located at the subcutaneous-dermal junction, the middle plexus lies at the level of the sebaceous glands and the superficial plexus sends capillary loops to the dermo-epidermal junction. Lymphatics are present in the superficial dermis and around adnexae. The rich supply of nerves to the epidermis and dermis consists largely of general somatic, afferent, myelinated and non-myelinated fibres.

Adnexae

The adnexal structures such as hair, sweat glands and hoof are in effect specialized epithelial structures with embryonic ectodermal origins. They develop during gestation so that at birth all the major skin appendages apart from the hair shafts are present in adult form. The hair types vary; the mane and tail hair is thicker and longer than body hair. Seasonal shedding of hair affects only body and limb hair.

Mane and tail hair are less influenced by endocrine factors but are more affected by insults that affect keratin such as selenium toxicity. This is a partial explanation at least of the different responses that occur in different diseases.

Hair follicles Forms of hair follicles vary in different animals. The hair follicles of the horse are simple; single hair follicles of different sizes are evenly distributed. Simple follicles are deeply rooted in the dermis, and usually have a single hair shaft, sebaceous and sweat glands and arrector pili muscles. The hairs themselves consist of the internal root sheath, external root sheath, dermal papilla and hair matrix. The root of the hair has a terminal bulb which attaches to the dermal papilla. The hair shaft has an outer cortex (cuticle) of densely packed keratinized cells containing most of the pigment that imparts the characteristic colour. The inner medulla is a looser mass of cuboidal or flattened cells.

Growth of hair is seasonal in animals, with the time of growth and mitotic activity termed the anagen phase. Microscopically, anagen hair follicles have a roughly spindle-shaped dermal papilla wrapped over the hair matrix in the form of a 'ball and claw'. A transitional phase, during which

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cellular proliferation ceases, is the catagen phase. The follicle then enters a resting state, the telogen phase, after which mitotic activity and new hair production resume and the old hair is shed.

Tactile hairs, which include sinus and tylotrich hairs, are also prominent at some anatomic locations on horses including the periorbital and facial skin. They are much heavier, coarser shafts and the follicles have a prominent nerve and blood supply. The former act as slow-adapting mechanoreceptors and generally are located on the muzzle, above the eyes and on the lips and throat. The latter function as rapid-adapting mechanoreceptors and are scattered among regular body hairs.

Sweat glands

There are two types of tubular glands in the skin: apocrine (paratrichial) glands associated with primary hair follicles and eccrine (atrichial) glands found on glabrous skin (e.g. hoof).

Apocrine gland activity is grossly visible in horses as they sweat after exercise or during high temperature. It is believed that the ability of the horse to sweat in limited regions is dependent on the local delivery of catecholamines. This can arise either because the blood has large amounts (as in excitement or exercise) or when the cutaneous blood supply is increased sufficiently. Increases in cutaneous blood supply in local areas occur in some autonomic disorders such as Horner's syndrome when the affected side of the face/neck sweats significantly relative to the normal unaffected side. Grass sickness (equine dysautonomia) is an autonomic neuropathy characterized by patchy sweating over the body and neck in particular.

Sebaceous glands

These are holocrine glands derived from hair follicles and their ducts discharge sebum into the upper part of the hair follicle. The horse has large, branched sebaceous glands that are especially numerous on the lips and around the vulva and particularly large in the mane, prepuce and mammary gland areas. Secretion of sebum is governed by endocrine factors in most species but the mechanisms are not established in horses.

Arrector pili muscles

These are smooth muscles that extend from the connective tissue sheath of hair follicles to the superficial dermis. Muscle contraction causes erection of hairs and expression of sebaceous glands. These muscles are not well developed in horses.

Blood vessels and nerves

The equine skin is very well supplied by both blood vessels and nerves. The latter explains the considerable sensory and tactile nature of equine skin. The blood supply is controlled largely by autonomic nerves and can be locally variable; this accounts for the horse's ability to sweat in patches. Patchy sweating is a normal equine phenomenon that relates more to the blood supply than to the overall concentration of catecholamines in the skin and this is why vasodilation results in sweating even in the absence of extra catecholamine. Autonomic nerve dysfunction can result in localized areas of continuous sweating – usually the affected areas are warmer than the adjacent normal skin and this can usually be palpated or can be confirmed using thermal imaging techniques.

Subcutis (hypodermis, panniculus)

Below the dermis is the subcutis (or hypodermis). It consists primarily of fat and loose connective tissue. The subcutis attaches the dermis to adjacent muscle or bone and comprises adipose tissue, collagen and elastic fibres. The former insulates against temperature variation, the latter provides flexibility. The subcutis is also an effective shock absorber for impact protection and cushions the more sensitive structures beneath.

In horses there are areas where subcutaneous fat accumulation is more obvious such as the sides of the body, buttocks and neck regions; only rarely can fat be found on the limbs or face. Deposition of subcutaneous fat is an important energy reservoir and its extent is used to provide an index of body condition. Deposition can occur irregularly in some areas and over-fat horses often have excessive subcutaneous fat deposits in the neck/crest region and over the buttocks and tail head. In some diseases there is an abnormal redistribution of fat. For example, in pituitary pars intermedia dysfunction (PPID/equine Cushing's disease) there is a diagnostically significant pattern of fat deposition in which fat is laid down in the retrobulbar regions and in the abdomen at the expense of the neck and body wall.

Special appendages (hoof/ergot, chestnut)

There are several significant cornified/horny appendages including the hooves, the ergots and the chestnuts. These structures tend to be viewed as rather casual 'skin appendages' but not only can they reflect important clinical signs of disease (e.g. laminitic rings) and nutritional abnormalities (e.g. biotin and methionine deficiencies) or toxicity (e.g. selenium toxicosis) but they have significant implications for diagnosis of some important diseases. The margin between these structures and the adjacent skin is often a site where abnormality can be demonstrated in some significant autoimmune conditions. For example, in pemphigus foliaceus and some autoimmune coronitis disorders there may be marginal inflammation along the skin border and often much more extensive changes than in normal skinned regions. The chestnuts provide a good source of tissue for diagnostic pathology where the coronet cannot easily or safely be sampled.

In solipeds such as the horse, the hooves consist of the wall, sole and frog. The sole and wall are composed of keratin. The wall consists of three layers, the inner one of which interdigitates with the dermis and acts as an anchor. The deeper portion of the dermis blends with the periosteum of the third phalanx. The frog is a dense fibro-elastic structure that has a major shock-absorbing function. Both the walls/sole and the frog are subject to significant pathological changes. An excellent description of the anatomy of the foot is available in Pollitt (1995).

Hair growth patterns

Some months before birth hairs develop over the body. Premature or dysmature foals always have a poorly developed hair coat and this is used as one of the indices of gestational age. The character of the hoof is also significant – at birth the hoof is soft but is recognizably 'hoof'. It hardens rapidly after birth. Premature foals have a soft rubbery hoof capsule often with finger-like extensions from the sole

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margin. The hair of a newborn foal is soft and is usually short; in some specific lines of some breeds a longer curly coat is present. This is a 'normal' trait in these animals.

Abnormal growth patterns do occur and the timing and nature of these are important diagnostic features. There are seasonal variations on the normal that may relate to breed and environment but some changes are regarded as pathological, for example seasonally related alopecic conditions. However, little is known about equine hair and its alterations in disease in horses.

The horse has four types of hair:

1. The temporary hair that is shed in cycles governed by changes in daylight length. This comprises the large majority of the body and limb hair.
2. The tylotrich hairs are distributed throughout the body hair. They are recognizably larger than normal body hair and have a sensory function.
3. The permanent hair of the mane and tail. This is not shed in a coordinated way and grows continuously at the rate of about 2–5 mm per month. Eyelashes (vibrissae) are also classified in this category but clearly these do not grow as fast and are inherently shorter.
4. Tactile/sinus hairs. These are larger and more isolated hairs mostly found on the face, eyelids and ears. They are associated with sensory nerve endings and play an important tactile role.

The hair coat of the horse varies with breed and with seasonal as well as environmental influences. Horses that live in cold conditions tend to have longer hair than those living in permanently hot conditions – although the moulting cycle still takes place. A horse that lives in the arid deserts of Africa does not grow a long hair coat at all and those living in very cold climates tend to have denser and longer coats at all stages in the year; both of course do still shed seasonally. Some breeds will grow very thick winter coats when in their natural environment of very cold conditions and this can be confused for abnormal hirsutism. Horses that move from one type of climate to another will adapt to the new circumstance over a few hair-shedding cycles.

Skin and hair alterations in disease states

The response the skin shows to insult or disease is reflected in changes in its physical appearance or texture. Alterations in the hair density or colour are also common signs of skin disease, but in some cases these occur in the absence of any apparent local disease process.

Changes in physical appearance might be very significant to the dermatologist but taken alone may serve to confuse the possible diagnosis and treatment. Although there are some pathognomonic changes in some diseases that make diagnosis relatively straightforward, there are many disorders that are almost indistinguishable clinically. This emphasizes the need for detailed history and clinical examination as well as the careful selection of further diagnostic procedures. Usually some pathological evidence can be obtained provided that the samples are truly representative of the abnormal condition. Possibly the most difficult change to detect and assess pathologically are the pigmentary changes in the absence of hair loss.

The skin can be an indicator of systemic disease; for example, a horse with generalized lymphoma may have obvious cutaneous nodules that may on their own resemble other conditions such as nodular sarcoid or melanoma. Liver disease may manifest as cutaneous jaundice or photosensitization affecting the white parts of the body. The cutaneous signs of equine systemic lupus erythematosus-like syndrome and equine sarcoidosis (generalized granulomatous disease) may be by far the most obvious visible evidence of the conditions but these cutaneous symptoms can resemble other conditions also and in many such cases the changes are subtle. There are also skin diseases with systemic implications; and it is therefore important again that a full clinical examination is performed when investigating a dermatological complaint.

Note

It is easy to overlook the skin when dealing with more 'significant' clinical signs but careful dermatological examination can be very helpful in identifying systemic disease and systemic signs of cutaneous disease can also be helpful diagnostically.

Inflammation

The inflammatory response is probably the most important single aspect of skin disease. It can be defined as the response of living tissue to injury (Cotran et al 1989). This response can only occur in vascularized tissues and is essential for protection against invading pathogens and for instigating the repair processes following injury or disease. It is also an essential defence mechanism and an important pathological process with both desirable and undesirable consequences. In some types of injury, such as simple lacerated/incisional wounds, inflammation leads specifically and directly to repair. In infected tissues the response leads first to the removal of necrotic tissues, infectious organisms and foreign matter and then to repair. In some infections the process of elimination of bacteria may lead to the release of enzymes which have tissue-destructive properties. This process, which may also involve other causes of tissue destruction, can result in disruption of the normally beneficial inflammatory response.

Inflammation is a highly complex process which proceeds in a coordinated fashion to produce the response in the injured tissue. One of the main values of inflammation is the repair of injured tissues after foreign matter and infection have been eliminated. A detailed knowledge of the particular inflammatory responses in an organ and the cells involved in the process provides the clinician with the means to manipulate the response and to develop new and more effective therapeutic regimens.

Failure of the normal defence mechanisms to respond adequately to an insult can result in infection and progressive tissue damage. The inflammatory process can also sometimes be destructive and in other cases it is an inappropriate response that results in disease. An inappropriate, overactive response can also result in tissue damage as a result of (autoimmune) self-damage.

The inflammatory process is largely similar for a wide range of applied insults – it can be regarded as the end point of damage however it is caused. The responses seen

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in the skin are generally, therefore, not pathognomonic for a particular cause. However, there are some responses which show significant and identifiable differences that make diagnosis simpler. These conditions are often easily recognized, both clinically and pathologically.

There are several distinct ways in which the skin can respond to an applied insult. The first of these is the inflammatory response and the others largely fall into the categories of injury or hypersensitivity. However, it is important to realize that a skin laceration, for example, will be accompanied by inflammation and, indeed, its resolution (healing and repair) depends upon the existence and efficiency of the inflammatory response. Similarly, a hypersensitivity response, to an insect bite for example, will inevitably result in an acute inflammatory response. The intensity and duration of the response will depend in large part on the nature and severity of the insult and any complicating factors which develop and on the specific/individualized reactions in the affected animal. It is entirely possible (indeed likely) that the same insult applied to different individuals will result in significantly different responses. Even self-inflicted trauma (such as might arise from pruritus) and mild infections will have a profound influence on the overall response. It is therefore often difficult to establish the primary problem when such complicating factors exist.

Typical of all aspects of physiological response, the processes are linked seamlessly and undue reliance upon categories of response is likely to lead to errors of interpretation. Inflammation can conveniently be divided into acute and chronic forms, although this division is arbitrary and elements of both are almost always present at some point in the healing/repair process. The balance between the two is probably not clinically definable, but acute inflammation usually evolves into chronic inflammation with or without a repair phase. In some cases there is no detectable acute phase and the injury appears to pass directly into a chronic response. Chronic inflammation is usually of longer duration than acute inflammation and is characterized by the presence of mononuclear cells (macrophages, lymphocytes and plasma cells) and marked fibroblastosis.

The acute inflammatory response

Note

Acute inflammation is the natural rapid response to an injurious process that supports host defence mechanisms at the site of the injury through delivery of plasma proteins and cells.

There are three major components in the acute inflammatory process:

1. increased blood flow
2. extravasation of blood proteins and leukocytes
3. emigration of leukocytes and their activation in the damaged tissues.

Potential stimuli for acute inflammation in the skin and subcutis include:

1. infections (viral, bacterial, fungal and parasitic)
2. trauma (blunt and penetrating)
3. physical and chemical injury (thermal injuries (burns, freeze), radiation, chemicals)
4. tissue necrosis (no matter what cause)
5. foreign bodies (splinters, metal, glass, etc.)
6. hypersensitivity (immune) reactions.

Each of these dermal insults can result in distinctive features that can provide useful diagnostic information but the underlying process is the same and so differentiation based on the physical appearances can sometimes be misleading.

Following an insult to the skin that involves a vascular response to injury (such as physical injury, insect bite or a response from interactions within the body itself), a transient period of vasoconstriction develops. This is followed by vasodilation and an increase in arteriolar permeability both to cells and to bioactive mediators. This results in the extravasation of fluid and proteins (and blood cells if the damage is sufficiently severe). There is a resultant relative haemoconcentration and increased viscosity of the blood which slows the circulation down. This allows stasis of erythrocytes and platelets and margination of leukocytes to occur. Once attached to the vascular endothelium they migrate into the extravascular space.

Neutrophils and monocytes identify and attack noxious particles such as foreign matter and bacteria, removing them by phagocytosis. However, most bacteria must first be coated with serum proteins (opsonins) (which have also leaked into the tissue fluid as a result of the vascular damage). Opsonins include IgG (antibodies to antigens encountered previously) and complement. Once phagocytosed, the bacteria are destroyed in the cell itself, but some of the products of this breakdown and the lysis of cells can escape into the region of the injury and here they may exacerbate the tissue destruction. A cascade of biophysiological responses is instigated which culminates in the development of one or more of the cardinal signs of inflammation. These are heat, pain (or pruritus), swelling and (less obviously in the skin of horses) redness.

The cells involved in the acute inflammatory process are largely delivered to the site via the circulation. They include granulocytes (neutrophils, eosinophils and basophils), mast cells, monocytes and lymphocytes. The neutrophil is the predominate cell in acute inflammation, particularly when bacterial infection is present. It is also a common cell type in skin that has been traumatized (either by external insult or from self-inflicted trauma such as follows rubbing and scratching).

The neutrophil These contain two major types of granules which are involved in the inflammatory process. While these cells generally have a predominately beneficial effect on acutely inflamed tissue, disruption of the cells can cause the release of destructive enzymes, which can lead to further cell death. Furthermore, they can elicit the production of free radicals, hydrogen peroxide and hypochlorous acid, which are all harmful to the tissues.

Other granulocytes Eosinophils, basophils and mast cells release a variety of lipid and peptide mediators which instigate aspects of the inflammatory response. The eosinophil seems to be particularly involved in the defence mechanisms for parasitic infections/insults while the basophil and mast cell are probably involved more in allergic or immediate hypersensitivity responses.

Macrophages These are probably the most important cell type in the inflammatory responses. They are especially

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important in chronic inflammation. They are produced by monocytes in the bone marrow and are delivered to the damaged tissues by the circulation. Monocytes are long-lived tissue cells which can differentiate under suitable stimulus into macrophages with secretory and phagocytic properties. They have a highly active phagocytic function and their role in the process of defence inflammation involves many bioactive substances (including plasminogen activation factor, collagenase, interleukin-1, tumour necrosis factor and complement).

Activated macrophages also produce oxygen molecules (including free radicals) and eicosanoids (including prostaglandins and thromboxane). The majority of the products from macrophage metabolism and destruction augment inflammation, and while this may result in significant defence against microorganisms it may also cause further tissue destruction.

Macrophages also play a role as an intermediary in the development of humoral response/antibody production by carrying and presenting antigens to T lymphocytes.

Lymphocytes These cells are important for identification of foreign and auto-antigens. B cells recognize the antigens and will respond by producing antibody-forming T cells. T cells also recognize antigens and will proliferate in response to specific antigens.

Chronic inflammation

Coordination of the vascular and cellular responses is important in the inflammatory process. This is provided by a series of mediators which are derived from the circulation (complement, albumin, fibrinogen and kinins) and from cells in the tissue (either migratory cells or cells comprising the local vasculature or other tissues). Cyclooxygenases catalyse the breakdown of arachidonic acid and this culminates in the production of prostaglandins or thromboxane. The prostaglandins are mediators of the vascular phases of inflammation causing profound vasodilatation, and with histamine and kinins cause increased vascular permeability. In addition to the pro-inflammatory effects, these chemokines also have significant suppressive effects on B and T lymphocytes as well as interleukin-2. Lipoxygenase proteins also support the bonding of oxygen, but in this case to fatty acids (and arachidonic acid in particular). This reaction proceeds to the production of leukotrienes, which are important chemo-attractants for leukocytes. The interrelationships of these chemical processes are very complex.

A large number of factors are capable of influencing the inflammatory process including the non-steroidal anti-inflammatory drugs (such as aspirin, phenylbutazone, ketoprofen and flunixin meglumine). These drugs act by inhibiting cyclooxygenase and individual drugs have greater or lesser effect in this direction. Corticosteroids also have a profound effect on the process by inhibiting the synthesis of prostaglandins and leukotrienes. They have an important difference from the non-steroidal drugs in that they affect both cyclooxygenase and lipoxygenase production. Thus, the two classes of drugs can be used to disrupt different aspects of the process and are particularly useful when one side or the other of the process is having a deleterious or harmful effect on the tissue.

Growth factors and cytokines are complex bioactive proteins which influence the behaviour of cell types, through either stimulation or inhibition. The relative proportions of these are vital in the coordination of the repair processes which follow inflammation. Uncoordinated production with consequent alterations in cell behaviour are blamed for many abnormal or idiosyncratic responses in the skin including the failure of some skin wounds to heal.

Proteinases (including the metalloproteinases which rely upon zinc in particular) are some of the most significant mediators of injury and tissue destruction. In some cases they are responsible for bacterial and cell debris degradation but they have a significant and desirable role in collagenolysis and natural disruption of the extracellular matrix in healthy, healing tissue. The role of these proteins in skin diseases of the horse has not yet been established but their study is currently one of the most exciting areas of research.

It is probable that all aspects of the inflammatory process have both advantages and disadvantages. The balance and coordination of the process appears to be the most significant aspect in promoting the beneficial while suppressing the harmful effects. All therapeutic measures applied to the repair of skin injuries and the curing of skin disease have to take account of these processes and drugs or manipulations which sacrifice the beneficial aspects must be viewed with some caution.

The extent of the inflammatory response outlined above is also influenced by various other types of response which precede inflammation. These are usefully categorized into four types of reaction which depend variously on the type of insult that is applied. An in-depth description of these responses can be found in most texts on immunology and pathology. Although some skin diseases can be attributed to a single one of these responses, most have aspects of several and so the categorization is arbitrary. It is, however, helpful at a basic level.

Mechanisms for immunologically mediated skin disease

In common with immune processes throughout the body, immunity and resistance to disease or insult on the skin surface is reliant upon cellular and humoral components. The first part of the defence system is the major physical barrier and then a series of delicately controlled immune and cellular processes produce a protective inflammatory response. There are mechanisms to 'recognize' previous challenges so that defences can be mounted rapidly. This relies upon the delivery of defined and specific antigen fragments to the antigen-sensitive cells of the immune system. Once the appropriate fragments are incorporated into the cellular recognition mechanisms, the immune system will respond by producing antibodies to them. The fundamental components of this process are receptors (antigen-presenting molecules called histocompatibility molecules) on cell surfaces. Most of these molecules are coded for by genes in the major histocompatibility complex (MHC). The histocompatibility molecules are entirely dependent on the specific genetic structure of the individual animal. Thus, there are species and individual susceptibility and resistance to disease that depend upon the MHC.

The MHC has particular importance to equine dermatology because of the way it is linked to certain of the major

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cutaneous disorders such as the equine sarcoid. In mammals the MHC is a very large gene complex. Each MHC contains three classes of gene: class I, class II, class III. Although they all have a role, the major groups in equine skin disease are the class I and class II genes and these are the main mechanism for controlling and regulating the immune responses. There are two class I loci (ELA-A and ELA-B). ELA-A has more than 20 accepted alleles whilst the ELA-B locus has fewer. Two class II loci have been identified (DR and DQ). All these code for MHC molecules on the surface of antigen-presenting cells including the cutaneous dendritic (Langerhans) cells and cutaneous macrophages. MHC class II proteins are expressed on cutaneous dendritic (Langerhans) cells, and in horses nearly all adult T cells, including the specific dermal population. Class III genes code for complement molecules and although these are of significance they have a lesser role in dermatological disease than the others.

The structural constraints limit the ability of the MHC to bind to proteins and so the efficiency of protein binding to the MHC molecules is necessarily a limitation to the resistance to infectious and other disease. A much larger number of MHC molecules would seem to be a desirable objective to enable a much wider range of disease resistance but this also has potential threat as well – recognition of a self-antigen would be potentially catastrophic. The primary function of the MHC molecules is to control immunological function. If a challenging protein cannot bind to an MHC molecule there will be no immune response and disease may ensue. Susceptibility to insect bite hypersensitivity (IBHS/sweet itch) is linked to the presence of ELA-Aw7 – perhaps the most susceptible horses are Icelandic, Shires and Welsh ponies and they are all in this group. Similarly susceptibility to sarcoid is linked closely (almost 100%) to ELA-A3, ELA-A15 and ELA-Dw13. Lipizzaner horses are solidly immune because they have no 'susceptibility' genes, and they therefore may have an ability to recognize and bind the (putative) causative virus.

A basic understanding of the immune processes involved in maintaining 'normality' is important for any clinician. Inappropriate immune responses that result in pathological states are commonly termed hypersensitivity disorders. The responses still result in inflammation but the cause is different to the inflammation resulting from trauma or infections. Derangements that are induced by overt and easily understood insult such as trauma and infection are far more understandable but more complex diseases often have underlying immunological complications relating to the aetiology. Controlling or treating these conditions can be a complex process but in common with most therapy, there are undesirable effects that could result in significant problems. These are more evident when dealing with immune-mediated disease.

The skin is the first line of defence against microbial and toxic insults. The efficiency of this system means that almost no pathogenic microorganisms and few toxins can penetrate the normal intact skin. Potential pathogens on the skin are inhibited from gaining access by the physical barrier and an active cellular process driven by the cutaneous dendritic (Langerhans) cells (see also p. 7) that are interspersed between the keratinocytes of the epidermis. These cells are responsible for recognizing pathogenic substances and organisms and presenting them to the dermal T lymphocytes. Although they have an independent nature they also depend heavily on the keratinocytes themselves – for many years the keratinocytes were only considered to provide an impervious barrier to external insults but this is far from the case. The keratinocyte is an active player in the immunological protective processes of the skin. Both Langerhans and keratinocyte cells have abundant MHC class II molecules on their surface and in response to these they will release significant amounts of interleukin mediators (IL-1, IL-6, IL-8) that further enhance the activity and numbers of dermal T lymphocytes. It is when this response is unregulated that disease arises as opposed to a curative reaction. An example of this process is when a parasite bites the skin. The antigen of the saliva is bound to the Langerhans cells and presented directly to the dermal T lymphocytes; the resulting mediator-driven inflammatory cascade results in a prominent local inflammation culminating in severe pruritus.

An additional protective mechanism is afforded by humoral immunity on the skin surface. IgA is thought to be produced locally by skin lymphocytes and protect the skin surface by immune exclusion. However, little is known about this specifically in horses.

The protective functions of the skin can be breached by other insults also – strong ultraviolet light and some chemicals can cause significant derangement (or possibly even destruction) of the Langerhans cells. In these circumstances secondary infection and an inappropriate response can arise. For example, certain plant proteins are known to adhere to the skin or to be deposited in the skin haematogenously (phylloerythrin, hypericin, etc.), and when these are bound to dermal immune cells and then subjected to ultraviolet radiation the inflammatory response is excessive. This is termed photosensitization. A good example of this is the condition known as photoactivated pastern and cannon leukocytoclastic vasculitis (see p. 273). It is suggested that pollens and other plant proteins bind to the skin-forming complexes that can be activated by ultraviolet light and so there is a pattern of pathological inflammation that reflects the combined pattern of contact and the pattern of exposure to light.

Hypersensitivity diseases, whether triggered exogenously or autogenously, can be classified on the basis of the fundamental immunological mechanism that mediates the disease (Table 1.3). Classically the Coombs system is used to categorize the responses. This approach recognizes four types of hypersensitivity response, i.e. types of inappropriate response reflecting in pathological variations. This is helpful insofar as it identifies the spatial and temporal nature of the specific pathogenesis and hopefully therefore the possible therapeutic interventions that are likely to be helpful. Whilst these are well-characterized and fall into well-described pathology dogma, the reality is that the classification may be too narrow and many conditions have elements of more than one type of 'reaction' and there are many circumstances when the fundamental process is complicated by aspects of another one or by coexistent or consequent inflammatory changes. For example, a condition that results in alopecia may be a result of primary degranulation of mast cells and eosinophils, but the local inflammation will alter the process and secondary self-trauma from itching and infection will change that still further.

The pro-inflammatory processes are linked both spatially within the tissues (tissue responses are linked and interdependent) and temporally, some being more immediate and

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Table 1.3 The relationship between the hypersensitivity responses and some of the recognized hypersensitivity diseases of the horse

Disease	Type 1 (immediate)	Type 2 (cytotoxic)	Type 3 (immune complex)	Type 4 (delayed, cell mediated)
Anaphylaxis	++ + +			
Atopy	+++			
Food hypersensitivity	+++			
Contact hypersensitivity				++
Urticaria	+++		+++	
Pemphigus foliaceus		+++		
Pastern and cannon leukocytoclastic vasculitis		+++	++	
Purpura haemorrhagica				+++
SLE-like syndrome			+++	
Drug eruptions	+	+++	+	+
Insect bite hypersensitivity	++			+++

some being delayed so that the time difference between onset of the response and the appearance of the clinical syndrome may add to the confusion. Taking the analogy of a horse that has an acute onset of a highly pruritic condition such as insect hypersensitivity, the horse may only be examined some hours or even days after the onset of the pathology when the signs become severe.

Nevertheless the system does help in our understanding of the responses that are associated with the immune system. Tissue damage may be evoked by antigens derived from outside the body (exogenous) and also in some cases from endogenous (internal) antigens. Although the mechanisms involved may be the same and the consequential secondary effects may be similar, it is important to make the distinction because the former are possibly useful defensive mechanisms for external challenges but the latter are inappropriate in many cases. The exogenous challenges may be avoidable or preventable but the endogenous ones are far more complex and the suppression of the responses may bring unwanted complications such as immunosuppression. The autoimmune conditions are an important group of skin disorders in all species and this applies equally to the horse.

A growing number of disorders of the horse (and in particular those of the skin) are being attributed to autoimmune aberrations (see Table 1.5). Much of the evidence is unfortunately, extrapolated directly from other species including man, and so where a similar pathophysiology is suggested the equine disorder has frequently 'picked up' the human name for the disease. However, there is little evidence that the diseases are identical and so the same name may not be entirely justified. This is simply because so little is known about the conditions in horses. For example, systemic lupus erythematosus (SLE) is a well-recognized (if rare) multisystemic immune-mediated condition in humans and dogs. A broadly similar multisystemic disease also affects horses and so this has been given the same name. However, so far no lupus cells have been found in horses and so it is wise to adjust the name of the condition to SLE-like syndrome until the true aetiopathogenesis can be established. This approach serves also to focus the research effort on those diseases of the horse for which the pathogenesis is unclear or totally unknown.

Coombs sensitivity reactions

Classically hypersensitivity responses are grouped into four distinct types. These have been given a numerical classification (1, 2, 3, 4), although the descriptive name is probably far more useful to the clinician in recognizing the basic pathophysiology of the various responses. It is tempting to classify hypersensitivity disorders into these distinct categories but there are many conditions that have characteristics of more than one hypersensitivity type. The reason this classification is important, however, is that the type of response also directs the clinician at the focus of the pathology so that treatment can be equally focused (Table 1.4).

The environment of the horse is teeming with molecular and infectious challenges that are capable of inducing an immune response. Antigens can be exogenous (including microorganisms, pollen, chemicals and drugs, etc.) and the immune response that arises as a result of exposure can be subclinical (usually no detectable clinical evidence) to minor irritations such as pruritus (itch) or minor skin rashes up to severe life-threatening disease such as asthmatic attacks. The basic pathophysiology of hypersensitivity disease is that contact with an antigen leads inevitably to a protective immune response but, this response can also be potentially damaging to the tissues of the host animal.

Severe hypersensitivity reactions are unusual in horses but they do include some immune-mediated vasculitis conditions such as purpura haemorrhagica. Most hypersensitivity reactions of this type in horses are caused by a well-described

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Table 1.4 Common disorders (not necessarily all cutaneous) associated with the various hypersensitivity states and the pathophysiology of each of the four Coombs types

Coombs type	Disorders	Immune mechanisms
1 Anaphylactic (immediate)	Anaphylaxis Urticaria Allergic rhinitis Acute airway allergy (asthma)	Cytotropic IgE antibody causes immediate degranulation of basophils and mast cells and the release of vasoactive amines that recruit other cells to the site. Vascular permeability increases with sequestration of proteins
2 Cytotoxic	Autoimmune conditions Transfusion reactions Neonatal isoerythrolysis	Formation of IgM and IgG which bind to antigens on target cell surfaces and result in phagocytosis of the cells or lysis of cells by CD8 (or antibody-dependent cellular cytotoxity)
3 Immune complex	Arthus reaction SLE-like syndrome Immune-mediated thrombocytopenia/anaemia Drug eruptions	Antigen–antibody complexes result in complement activation which attracts neutrophils and release of lysosomal enzymes and other tissue destructive substances
4 Cell-mediated (delayed)	Skin tests for tuberculosis/glanders Contact allergies Transplant/graft rejection	Sensitized T lymphocytes cause release of lymphokines and T-cell-mediated cytotoxicity

(but poorly defined) series of Coombs hypersensitivity reactions. The reactions are usually mediated either by antigen/humoral antibody or by various cell-mediated immunological mechanisms. In some circumstances antibody-mediated responses can be initiated by endogenously derived antigens; this is called autoimmunity. This circumstance is obviously more difficult to control than conditions caused by exogenous antigens. In the latter case, the disease can usually be controlled simply by avoidance of exposure to the defined antigens. For the autoimmune conditions avoidance is of course impossible and so the diseases tend to be far more complex, require longer-term management and inevitably carry a worse prognosis. However, some are relatively mild and then management can be effective.

Coombs type 1 (immediate) hypersensitivity

Type 1 hypersensitivity is a rapidly developing immune-mediated reaction that characteristically develops within minutes of the challenge; this gives it its alternative name of immediate hypersensitivity reaction (Fig. 1.4). It results from the rapid release of a barrage of vasoactive and spasmogenic substances that act on blood vessels and smooth muscle in particular and so alter their function considerably. Usually this response requires prior exposure to the antigen (allergen) and development of a specific antibody for this response to develop.

The antibodies that cause type 1 hypersensitivity belong to the IgE group. Most of the antigens (allergens) are delivered through mucous membranes or the skin but parenteral injections can also be responsible. In some animals the IgE is produced constantly and high concentrations can build up. This possibly genetic disorder is called atopy and is an important dermatological syndrome (see p. 262).

The mechanism responsible for type 1 hypersensitivity reactions is focused around mast cells. Where antigen binds to mast cell bound IgE, the cells are induced to degranulate and release their contents (primarily histamine) into the surrounding tissues. This precipitates an inflammatory cascade that involves the arachidonic acid ladder and induces vasoconstriction, bronchial constriction and localized destruction of nearby cells with activation of the complement cascade also. Sometimes the response is extremely rapid (within seconds or minutes) and circulating basophils will also be triggered to degranulate en masse.

The clinical effects of type 1 hypersensitivity reactions relate mainly to the vasoactive molecules and the extent of the signs will depend on both the location and the extent of the process. Where the response is severe and widespread and exceeds the compensatory mechanism the term anaphylaxis is used; the reaction develops into a full anaphylactic response – collapse, pallor, tachycardia and catastrophic falls in blood pressure can develop. A similar severe response can be seen in some cases but this is not mediated immunologically and so the term anaphylactoid response is applied to those. An example of this is the acute anaphylaxis that is reported to occur occasionally when using protein extracts of bacillus Calmette–Guérin for treatment of equine sarcoids. The severity and danger associated with this type of response means that it is invariably harmful rather than protective. However, at least some antigen is eliminated and this is also the suggested mechanism for resistance to some helminth parasites.

Where the response is milder the signs are correspondingly moderated by counteractive/compensatory processes. Where the signs are manifest in the skin, local oedematous swellings (hives, urticaria) occur. Other examples are allergic rhinitis resulting in a serous nasal discharge and nasal irritation, and food allergies resulting in diarrhoea and rectal/anal irritation.

Typical examples of this response include anaphylaxis, atopy and food hypersensitivity.

Coombs type 2 (cytotoxic) hypersensitivity

Type 2 hypersensitivity is mediated by antibodies directed towards antigens present on the surface of cells or other tissue components. In some cases the antigens are intrinsic to the cell

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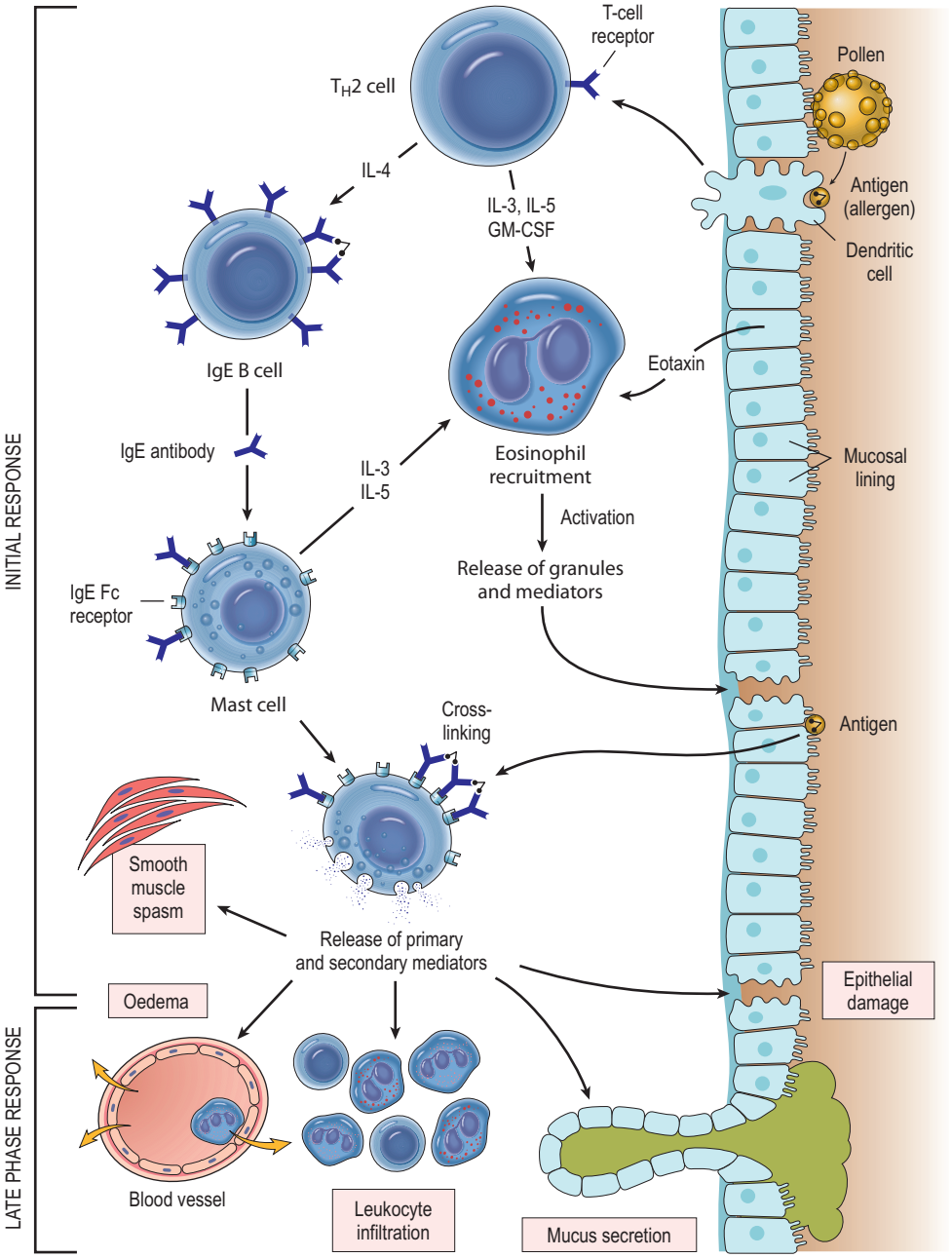


Figure 1.4 A schematic diagram showing the theoretical pathogenesis of a type 1 hypersensitivity reaction. The late phase response is mediated by leukocytes in filtration and tissue injury and so takes a longer time to develop than the immediate response. The diagram shows the response to a mucosal allergen challenge by a pollen grain. (Adapted from Kumar V, Abbas A, Fuasto N 1999 *Robbins and Cotran Pathologic Basis of Disease*, 7th edn, WB Saunders, Philadelphia; with permission.)

itself or extrinsic where antigens are adsorbed onto the surface of a cell. The humoral antibody binding to the surface of the cell prepares these cells for phagocytosis by macrophages and other active cells or to lysis. There are three different ways in which the subsequent response is induced (Fig. 1.5).

1 *Complement-dependent reactions.* These lead either to the lysis of the target cell or to phagocytosis. Antibody (IgM or IgG) binds to the cell and so activates the complement system that then disrupts the cell membrane causing lysis, or the cells are rendered liable to phagocytosis (opsonization). This type most commonly involves blood cells (leukocytes, erythrocytes and platelets are all vulnerable). This response can also be shown in the renal glomeruli. There are also some drug reactions that have this outcome if the drug is bound to the surface of cells in a suitable fashion. All forms of autoimmune haemolytic anaemia, thrombocytopenia and blood transfusion reactions are due to these responses.

2 *Antibody-dependent cell-mediated cytotoxicity.* Here the antibody-mediated damage involves monocytes, neutrophils, eosinophils or natural killer (NK) cells. The targeted antibody-covered cells are destroyed by non-sensitized cells and induce cell lysis without phagocytosis. This response is mediated by both IgG

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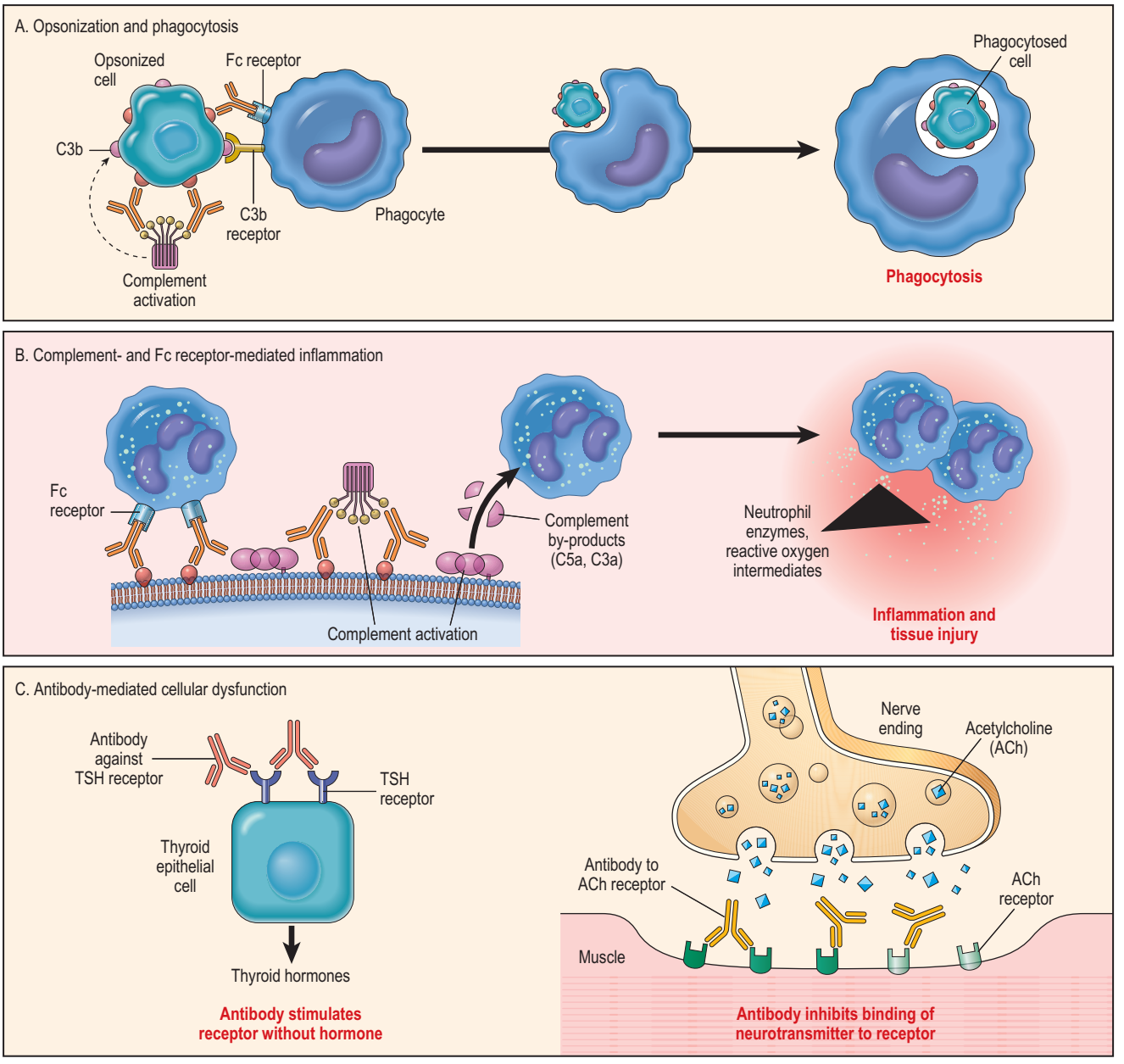


Figure 1.5 These three diagrams illustrate the theory behind the three major mechanisms involved in type 2 (antibody–antigen) hypersensitivity. **(A)** Antibody and complement-opsonized cells are phagocytosed by macrophages and neutrophils. **(B)** Antibody binding to Fc receptors of leukocytes and complement breakdown products induce an inflammatory response. **(C)** Antireceptor antibodies disturb their normal function. This example involves a well-identified human condition (Graves' disease) and this form has not been reported in horses as a cause of any skin-related disorder. (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn, WB Saunders, Philadelphia; with permission.)

and to a lesser extent IgE and may be involved in anti-parasite responses or tumour destruction.

3 *Antibody-mediated cellular dysfunction.* Again antibodies are directed against cell surface receptors and impair or dysregulate the function of the cell without actually destroying it. This type has little known equine relevance.

Examples of this response include the pemphigus complex of diseases, haemolytic disease of foals (neonatal isoerythrolysis), and immune-mediated and drug-mediated thrombocytopenia or anaemia. Drug eruptions may also be associated with this type of reaction.

Coombs type 3 (immune-complex-mediated) hypersensitivity

Type 3 (immune-mediated) hypersensitivity is induced by antigen–antibody complexes that cause tissue damage as a result of the activation of plasma mediators primarily of the complement group. The antigen–antibody complexes (immune complexes) can exist freely in the circulation (circulating immune complexes): the resulting toxic reactions can occur within the circulation or can be deposited in capillary beds, basement membranes or other extravascular sites (in-situ immune complexes). The presence of these

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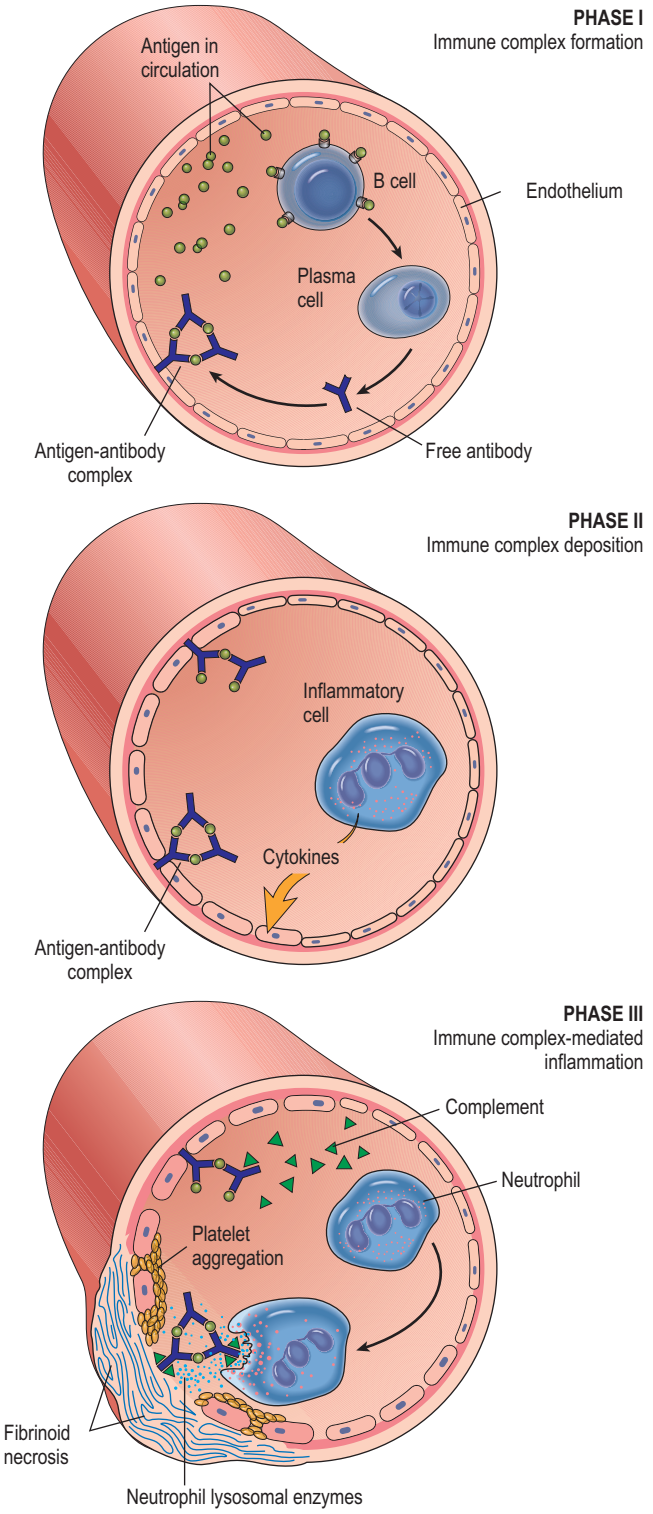


Figure 1.6 Schematic illustration of the pathogenesis of the type 3 (immune complex) hypersensitivity reaction. Note that this response results in vascular damage. (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn, WB Saunders, Philadelphia; with permission.)

antigen–antibody complexes in the circulation or in tissues is not, however, synonymous with disease – the process may be continuing without any untoward effects and it may be that this is a normal mechanism for antigen removal. However, in pathological states these antigen–antibody complexes are deposited on circulating cells (e.g. red cells or platelets) or the cells of major organs. Deposition onto vascular endothelial cells is particularly common. Their deposition at these sites is strongly attractive to neutrophils, and when these are disrupted they release tissue destructive hydrolytic and proteolytic enzymes which induce tissue damage (Fig. 1.6). The consequent damage to blood vessel

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walls causes small haemorrhages (petechial and ecchymotic haemorrhages) and leakage of protein, which results in localized oedema. Serious secondary systemic effects can arise. Cell damage can therefore occur without specific antibody to the cells themselves. The pathogenesis of the immune-mediated tissue injury is shown in Fig. 1.7.

The classical Arthus reaction (local immune complex disease) is a good demonstration of this type of response. It is a localized area of tissue necrosis (usually in the skin) that results from an acute immune complex vasculitis. When an antigen to which the animal is sensitized (i.e. the animal has existing circulating antibodies to the antigen) is injected or otherwise deposited into the skin, immune complexes are formed as the large amount of antibody accumulates. These precipitate and trigger an inflammatory response typical of a type 3 reaction. This reaction takes several hours to develop (in contrast to the type 1 immediate IgE-mediated response); it reaches a peak around 4–10 hours post injection. The local response is often referred to as a fibrinoid necrosis and shows eosinophil infiltration with moderate–severe oedema, local haemorrhage, microvascular thrombosis and subsequent necrosis.

This is the presumed mechanism for the autoimmune haemolytic anaemia which accompanies internal infections or neoplasia. Significant dermatological conditions involving type 3 hypersensitivity (immune-complex hypersensitivity reactions) include photoactivated pastern and cannon leukocytoclastic vasculitis, purpura haemorrhagica and systemic lupus erythematosus-like syndrome.

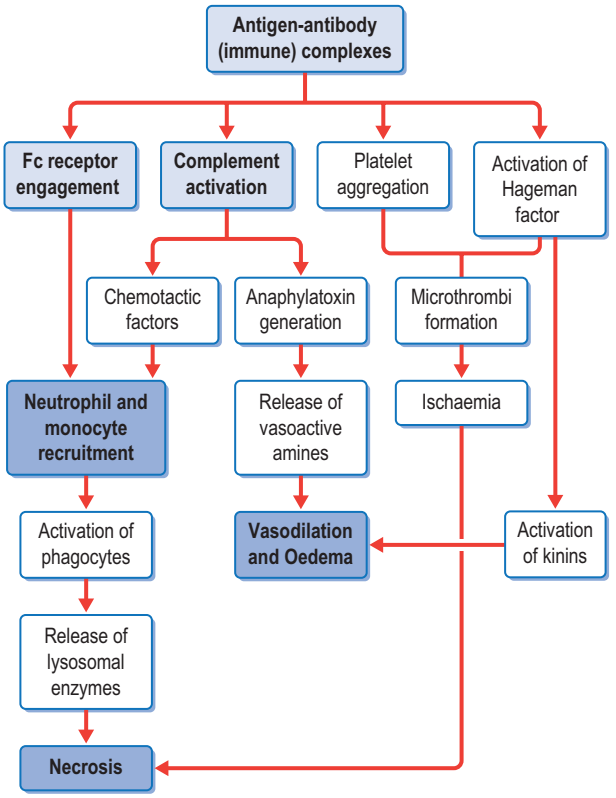


Figure 1.7 Pathogenesis of the immune-complex-mediated tissue injury characteristic of the type 3 hypersensitivity response. (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn. WB Saunders, Philadelphia; with permission.)

Coombs type 4 (delayed/cell-mediated) hypersensitivity

Type 4 hypersensitivity (cell-mediated responses) can be defined as a slowly developing cell-dependent immunological response to an antigen (usually exogenous or injected) that is bound to antigen-presenting cells (including Langerhans cells in the skin CD4 and T cells). The monocytes/macrophages (or Langerhans cells in the case of skin responses) then present the antigen to the lymphoreticular tissue where T lymphocytes respond by releasing mediators that cause tissue damage and generalized systemic effects such as extensive pruritus. Interferon-γ, interleukin-2 and tumour necrosis factor are all major players in the pathogenesis. As the reaction involves the activation of sequential cell-based responses, the hypersensitivity reaction/response is delayed for up to 48–72 hours (or sometimes longer). The type 4 (cell-mediated) hypersensitivity is the principal immunological pattern for many viruses, bacteria (including *Mycobacterium tuberculosis*), fungi, protozoa and parasites. This reaction is employed in the intradermal tuberculin test in cattle and humans and the intradermopalpebral mallein test for glanders in horses and it may be involved in graft rejection and tumour immunity (Fig. 1.8). The response can be used therapeutically in the treatment of sarcoid with bacillus Calmette–Guérin (see p. 406).

The intradermal tuberculin and mallein tests demonstrate the events well. When the antigen is injected into the dermis of a previously sensitized patient, the site of the injection develops into a reddened (this may not be obvious in veterinary species), swollen and oedematous focus over the following 2–3 days. This is recognized histologically as an accumulation of mononuclear cells in the dermis and subcutis and in particular around the vessels (perivascular cuffing). Oedema results from increased microvascular permeability. In this way protein and fibrin are deposited into the site and the latter is responsible for the indurated nature of most of these responses. Later, the 'cuffed' venules and capillaries develop a marked endothelial hypertrophy and possibly even hyperplasia. The CD4+ helper T lymphocytes can be identified histologically with immunoperoxidase stains. If the antigen is persistent or non-biodegradable, the mononuclear response is replaced by macrophages and this may then develop into a granuloma. Therefore the later stage pattern of inflammation is termed granulomatous inflammation.

Where the antigen is applied to the skin (contact allergy) these antigens may bind to the Langerhans cells in the dermis and the resulting complexes trigger a characteristic type 4, T-cell-mediated response. By 24 hours post exposure, mononuclear cells will have infiltrated the dermis in a typical perivascular cuffing, with intense pruritus. Provided that the antigen exposure is no longer present, the response will usually subside within a day or two.

Drug eruptions and insect bite hypersensitivity (IBHS/sweet itch), fungal granuloma, the mallein intradermopalpebral test and several skin tests for other conditions such as *Histoplasma* spp. are examples of this response. Responses to migrating *Gasterophilus* larvae may also manifest in this way.

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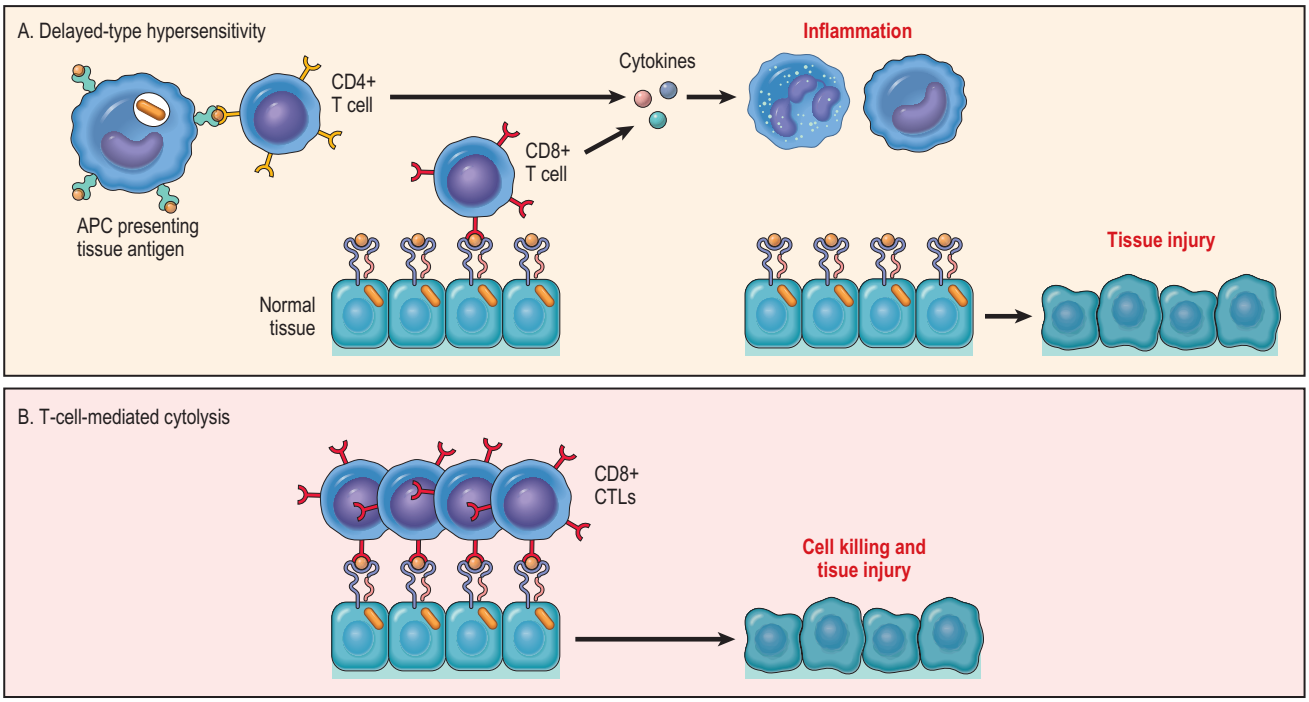


Figure 1.8 In type 4 (delayed) hypersensitivity responses the clinical effects develop slowly because of the need to attract sensitized activated lymphocytes to the site. This can often take some days to develop fully and is histologically distinctive with granuloma formation. (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn, WB Saunders, Philadelphia; with permission.)

Immune-mediated skin reactions

Disorders of the immune system fall into several defined categories:

1. Hypersensitivity conditions, which form the mechanism of immune-based injury in many dermatological and other system disorders (see above).
2. Autoimmune conditions, which are caused by immune reactions to self-antigens and tissues (see p. 265).
3. Immunological deficiency diseases, which result from distinct (usually genetically determined or acquired) defects in the immune system. Some important conditions, such as the lethal white syndrome, lavender foal disease and Fell pony immune deficiency syndrome, have important primary and secondary dermatological signs. These signs may be the only ones that are easily detected by a primary clinician.
4. Amyloidosis – a poorly understood disorder that can affect the skin and has an immunological basis (see p. 295).

Autoimmune disease

Immunological tolerance is a state in which an immune response to a specific antigen cannot be mounted by the individual. Self-tolerance means that the body does not mount any immunological response against its own cells or proteins. This process is clearly critical to survival and can be explained by several recognized pathological mechanisms including clonal deletion (loss of self-reactive T and B lymphocytes during their maturation in the thymus), clonal anergy (suppression of the activity of lymphocytes by continued exposure), and peripheral suppression by T cells. There is evidence for the existence of autoimmune disease in all species including the horse. These conditions are a result of inappropriate autoimmunity to self-antigens. The clinical states reflected by a loss or bypass of immune tolerance usually arise from several defined mechanisms and these vary for each autoimmune disease.

Note

Autoimmunity implies loss of self-tolerance.

It is also true that autoantibodies can sometimes be found in the body without any apparent clinical consequence. The existence of autoantibodies in a remarkable number of apparently normal animals suggests that this may be an effective physiological (non-pathological) mechanism for the removal of some antigens that arise following organ damage and in particular for the removal of self-idiotypes and aged cells This means that criteria have to be established to warrant a diagnosis of autoimmune disease (as opposed to incidental or even beneficial autoantibody production) – the simple existence of an autoantibody is not synonymous with 'disease'.

The autoimmune disorders include a broad spectrum of pathological possibilities, ranging from those conditions in which a single (monoclonal) antibody to a specific cell/organ is involved, i.e. organ-specific disease such as pemphigus foliaceus, to disorders that involve complex groups of antibodies to several or possibly even many antigens resulting in a more generalized disease such as equine systemic lupus erythematosus-like syndrome (Table 1.5). In the

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Table 1.5 The accepted autoimmune skin diseases of the horse

Single organ/tissue disease
Pemphigus: Pemphigus foliaceus Pemphigus vulgaris Bullous pemphigoid
Discoid lupus
Coronitis complex
Pastern and cannon leukocytoclastic vasculitis
Eosinophilic coronitis – stomatitis syndrome
Multisystemic disease
Systemic/cutaneous lupus erythematosus-like syndrome (SLE-like syndrome)
Drug eruptions
Chronic/generalized granulomatous disease (sarcoidosis)
Multisystemic eosinophilic epitheliotropic dermatitis

former case the target of the immune-mediated reaction is a single body cell or protein and so the signs are predictably alike in all horses affected by the same condition. In the latter, in contrast, the panel of symptoms can be wider and there can therefore be wide variations in the clinical syndrome. In SLE-like syndrome, the autoantibodies act against the nuclear material in a wide range of cells types so many organs are involved – it is one of the classical multi-organ disorders in all species.

Note

Autoimmunity can be to *single* self-antigens and so produce a defined, single, clinical or non-clinical symptom in a single organ or *multiple* when many organs or many molecules in a single organ can be affected.

Although the precise mechanisms for the loss or bypass of self-tolerance are not known, from a clinical perspective they appear to develop spontaneously and at random – the predisposing factors are seldom if ever obvious. The basic pathophysiology depends on genetic and immunological processes. The most accepted hypothesis for sudden appearance of autoimmunity relates to the exposure of hidden self-antigens. Infectious agents including viruses, bacteria and fungal organisms may also be implicated. In the latter case the fundamental mechanism may involve the microbial antigens becoming intimately associated with the host cells and the resulting immunologically active complex bypasses the T-cell tolerance. Viruses in particular may also induce B-cell mutations and so may induce formation of autoantibodies. In a few cases viruses can directly suppress immunity and so allow the normal T-cell protection to be bypassed. It is entirely possible that some of the drug eruptions are due to similar processes including drug binding to create an antigenic complex, or immune suppression.

An added complication is that many autoimmune conditions wax and wane in their severity; this implies that the relationship between the positive and negative regulatory processes is unstable. In some diseases an antibody is produced to the antibody and in theory this process can continue indefinitely but there is an apparent negative feedback mechanism, which could explain the 'settling' of some of these responses. In some cases remissions can take place for many years and then for no apparent reason there is a rapid deterioration. Other conditions may remain far more 'constant' with signs that vary only mildly. One form of coronitis appears to follow this latter path.

In spite of the importance of autoimmune skin (and other) diseases in horses, relatively little is known about how they are triggered and under what circumstances. Conventionally, for a diagnosis of autoimmune disease to be made, therefore, three criteria are required. Only when these three criteria are satisfied can a diagnosis of autoimmune disease be accepted.

1. The existence of an autoimmune reaction
2. Clinical and pathological evidence that this reaction is NOT secondary to organ damage but is in fact a primary pathological event
3. The absence of another explanation for the disease condition/symptoms (i.e. a diagnosis of exclusion).

Unfortunately these three criteria are seldom met in equine dermatology, even in disorders that are assumed to be autoimmune, so classical criteria may not strictly be applicable. However, in some of the 'well-recognized' pathologies such as equine SLE-like syndrome and pemphigus foliaceus, the pathogenesis is usually assumed and the diagnosis accepted.

Pathological description of skin responses

General considerations

Ultimately the clinician wants a diagnosis upon which he/she can focus treatment. The pathological reports on skin biopsy specimens returned by dermatohistopathologists should always contain a morphological description of the salient features of the changes presented. 'Incomprehensible' technical pathological descriptions without a 'presumptive diagnosis' are often quoted as a major reason why clinicians do not submit pathology specimens for histology and therefore the clinician can benefit greatly from a basic understanding of the various pathological changes that may be used in the morphological description of biopsies submitted.

There are few diseases that are so pathognomonic that a pathologist can give a definitive decision without any background information and it is therefore unfair to expect the pathologist to be able to 'spot' diagnose most of the conditions. The pathologist can only help if the full clinical information is presented and the correct specimens have been taken and handled correctly. Of course, the corollary of this is that the pathologist must be prepared to recognize that not all cases are 'textbook' in their signs and so there may necessarily be limited information available.

Examination of the patterns of reactions occurring in the skin are key to the interpretation of the responses and so provide information to the pathologist about the location, type and extent/severity of the condition. The patterns of disease are used to facilitate diagnosis but it is important to recognize that these are simply the common manifestations.

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Dermatopathology

Even in normal 'healthy' animals cells must adapt constantly to changes in their environment. There are well-defined physiological (hormonal, endocrine or mediator-driven) mechanisms that govern these changes and perhaps the best example of this is in the seasonal shedding of hair that is a feature of the horse. The hair that grows from an ostensibly identical hair follicle is much longer and faster growing in the winter-coated horse. The mechanisms for the shedding in spring and autumn are governed by endocrine mechanisms triggered by changes in daylight hours. Horses that live in hotter climates have a thinner hair coat covering and the difference between the summer and winter coat is less obvious. It is also possible to manipulate some of the normal changes; the coat can be kept thinner and shorter by providing heavy blankets in the colder winter months.

Although many of the normal adaptations that are made in equine skin are recognized, little is actually known of the mechanisms for them. Cell adaptation may involve up- or down-regulation of specific cellular receptors or alteration in the volume and type of proteins. Thus in a state where the skin is repeatedly challenged the epidermis will become thickened (hypertrophied) and may contain much more keratin than normal skin adjacent to it. The extracellular matrix in a chronically inflamed lesion on the skin is noticeably different from the normal state and the fibroblasts may alter significantly to become non-secretory fibrocytes. Many of the underlying mechanisms are also shared in pathological disease circumstances. Thus, there are some disorders that result in failure to shed the hair coat and others that result in thinning of the skin and hair loss.

In many circumstances the natural adaptive mechanisms will permit cells to survive and avoid injury in an adverse circumstance. Cellular adaptation is an important state that lies somewhere between normal unstressed cells and the severely compromised, injured cell that is typical of the diseased state.

Some of the common adaptive changes that affect the skin include:

1. *Hyperplasia/acanthosis.* Hyperplasia constitutes an increase in the absolute number of cells in the skin so that it is thicker, i.e. there are simply more cells of a normal type. Physiological hyperplasia of the skin is usually reactive or compensatory to some mild/physiological stimulus. Pathological hyperplasia arises as result of endocrine alterations or trauma or other repeated or chronic inflammatory changes. The most significant hyperplasia is that of the stratum spinosum called acanthosis (see below). The ultimate physiological response is called a hamartoma which is an abnormal accumulation of a normal cell type, usually but not necessarily, in a normal anatomical location; the outcome of course can be grossly abnormal (i.e. pathological). Hyperplasia of the epidermis is a consistent feature of almost all chronic inflammatory skin diseases. An example of this occurs in the vascular hamartoma (sometimes termed cavernous haemangioma/vascular naevus) of young foals (often Thoroughbred and Arabians) (see p. 237).
2. *Hypertrophy.* Hypertrophy refers to an increase in the size of the individual cells such that there is an increase in the overall size of the organ – it is usually less obvious than hyperplasia. It does not involve any increased cell numbers – the cells are abnormally large as a result of the synthesis of extra intracellular structural components. Again hypertrophy can be physiological (such as muscle hypertrophy in response to training) or pathological, arising from abnormal stimuli. There is a necessary limit to the size that individual cells can reach.

Note

- Hypertrophy and hyperplasia are often closely related and can develop at the same time.
- Hypoplasia: this is a *decrease* in the absolute number of cells and is a rare manifestation in the skin.

3. *Atrophy.* Atrophy refers to the shrinkage of individual cells as a result of loss of cell substance. This results in a shrinkage of the size of the organ or structure. In the skin atrophy is relatively common. Atrophic cells are recognizable pathologically from the increased number of autophagic vacuoles. Atrophy may proceed to the point where the cells die.
4. *Metaplasia.* Metaplasia is a (usually) reversible change in which one adult cell type is replaced by another. It may be adaptive or pathological. A common form of metaplasia is that which arises in the columnar epithelium of the respiratory tract, which becomes a squamous epithelium in response to chronic airway disease. Fibrous metaplasia occurs also in muscles that undergo denervation atrophy. The potential for conversion from one cell type to another appears to be inherent in stem cells. Although normality can be restored if the inductive stimulus is removed, prolonged exposure can result in a persistent change with a tendency to neoplastic transformation.

Gross pathology

Accurate description of the clinical appearance of the skin at a macroscopic level is critical, since before biopsy lesions are, in effect, the gross pathology. Correlation between the gross and histological appearances is often critical in formulating diagnoses and in understanding pathogenesis.

Clinical terms for specific types of lesions seen in the horse

It is important to describe lesions accurately even if a definitive diagnosis cannot be attained. An accurate description of a skin lesion is helpful for clinical descriptive purposes but it also provides a degree of descriptive commonality between clinicians and dermatopathologists. However, whilst these definitions are useful when describing a clinical condition, many lesions have characteristics of several single types. Examples of each type of lesion are shown in other chapters of this book.

Primary lesions

Macule A small (<1 cm diameter) circumscribed, flat, (usually) impalpable area characterized by its flatness and usually

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distinguished from surrounding skin by colour change in the skin (hypo- or hyperpigmentation).
Patch A macule >1 cm diameter.
Papule A small (<1 cm diameter), circumscribed, solid, slightly raised mass in skin less than 0.5 cm in diameter. A papule is always palpable as an abnormality of the normal skin texture/structure. Papules may be discoloured (pink or red). They can be associated with insect bites etc., and may be pruritic. (See Fig. 5.2.)
Plaque A solid, elevated, flat-topped, regular or irregular thickening of the skin >2 cm in diameter but without substantial depth arising as result of coalescence of papules. (Usually allergic in origin, e.g. urticarial lesions.) A plaque which has a closely packed irregular projecting surface is termed a vegetation.
Vesicle These are sharply demarcated, raised lesions <0.5 cm in diameter, filled with a clear watery fluid. Large vesicles are called 'bullae'. They can be epidermal or subepidermal. It is easy to appreciate that the thin overlying epidermis makes them very fragile and liable to rupture from even minor trauma – these are therefore rarely seen intact (e.g. early equine coital exanthema, blistering etc.).
Bulla A fluid-filled raised area greater than 0.5 mm across.
Blister Common term for vesicle or bulla.
Pustule A small, well-defined and circumscribed visible mass in the skin is filled with purulent material and inflammatory debris. They are usually yellow in colour but may have a green or dark brown hue. (See Fig. 5.11.)
Abscess This is a localized collection of pus in a cavity >12 cm diameter formed by disintegration of tissue. (See Fig. 3.31.)
Wheal A well-defined, circular, flat-topped, evanescent lesion formed as the result of dermal oedema of variable size, which usually causes no changes in the overlying skin or hair coat. There may be pruritus and variable blanching of the skin (in suitably coloured animals). They usually appear and disappear rapidly (e.g. urticarial reactions). (See Fig. 11.3.)
Nodule A raised, circumscribed (round) solid lump >1 cm in diameter which usually extends into the deeper layers of the skin (e.g. sarcoid, melanoma, neurofibroma, eosinophilic granuloma, some allergic responses). (See Fig. 18.23.)
Cyst A smooth, well-defined, circumscribed structure lined with epithelium and containing solid or liquid matter secreted by cells in the lining. Cystic structures in the skin are unusual, although the nasal atheroma and dorsal midline cutaneous dermoid cysts are encountered on occasion. (See Fig. 10.21.)
Tumour Mass of (usually) neoplastic origin (benign or malignant) (e.g. sarcoid, melanoma, papilloma, etc.).
Papilloma A 'nipple-like' mass of varying size (1–20 mm or more), projecting from the skin.
Petechiae Pinhead-sized macules of blood in the skin (or mucous membranes).
Ecchymosis A larger extravasation of blood into the skin, often with an irregular outline.
Haematoma A large accumulation of free blood under the skin arising from gross subcutaneous bleeding.
Comedo (plural comedones) A plug of keratin and sebum wedged in a dilated pilosebaceous orifice.
Epidermal collarette A particular circular (collar-like) arrangement of separated, peeling epithelium (scale) that usually reflects the prior existence of a pustule or vesicle/blister.
Telangiectasia/lymphangiectasia The visible dilation of small cutaneous blood/lymphatic vessels.
Naevus A well-defined developmental hyperplastic skin defect derived from any of the components of the skin including blood vessels, keratinocytes and glands.
Hamartoma A focal malformation resulting from faulty development of an organ that resembles a neoplasm, but which is composed of an abnormal mixture of normal tissue elements, or an abnormal proportion of a single normal tissue element that would normally be present in that site. In contrast to a neoplasm, a hamartoma develops and grows at virtually the same rate as normal components. It is normally applied to those tissue masses that have no compressive or other implications for adjacent tissues/structures.
Chorista/choristoma A focus of histologically normal tissue that is located in a site remote from the organ or structure from which it derived. A choristoma is a mass formed by maldevelopment of a type of tissue not normally found at that site.
Hirsutism Excessive/abnormal volume, density and length of the hair coat.

Secondary lesions

Alopecia Partial or complete loss of hair from whatever cause. Most alopecia is secondary but it can be primary. The term alopecia is not applied to rubbed or broken hair.
Scale/flake Accumulation of loose, light, epidermal debris (dry, horny, plate-like excrescence); usually the result of imperfect cornification and shedding from the skin surface. Usually the material is grey or white but it is sometimes discoloured yellowish/brown by sebum, serum or blood. Scale often has a soft, slightly greasy texture and squeezing the scales onto a piece of blotting paper can often produce a 'greasy spot' (Table 1.6). (See Fig. 14.4; p. 329.)
Crust A solid, dry, adherent accumulation of dried serum, blood, pus or scales sometimes including hairs. Wetting causes softening and then erythrocytes and other significant cells may be seen. Crusting may also contain significant

Table 1.6 The clinical and microscopic differences between scaling/flaking and crusting

	Appearance	Texture	Microscopic
Scaling	Scale like rafts of light material Grey/pale cream	Dry/soft No change with soaking (float on water)	Keratin/sebum May have cells such as erythrocytes, acanthocytes and bacteria
Crusting	Solid granules Brown, red-orange	Hard, gritty Soften with soaking (sink in water)	Red, brown/yellow irregular granules Soaked residue with erythrocytes, leukocytes and haemoglobin

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diagnostic cells such as acanthocytes in the pemphigus complex, and microorganisms, which can be a very helpful diagnostic aid. Crust feels 'gritty' and granular. When examined under low power microscopy, crusts often have a brown or red-orange colour and irregular three-dimensional shapes.

Scar/cheloid A scar is a result of deep skin damage where the normal structures are replaced by fibrous tissue. The older the scar the less obvious it becomes as a result of scar contraction and reorganization of the damaged tissues. (See Fig. 12.21.) Abnormal or inappropriate scarring may be hypertrophic or cheloid. (See Fig. 12.22.)

Erosion An erosion is a shallow skin defect not penetrating the basement membrane (it does not result in scarring). (See Fig. 9.12.)

Ulcer An ulcer is an erosion that penetrates at least into the upper part of the dermis and may extend the full depth of the skin (scar formation is inevitable when healing has taken place). (See Fig. 9.5.)2

Lichenification Thick, hard skin usually the result of repeated trauma which is often hyperpigmented and alopecic. Marked folding and thickening is often present.

Excoriation An ulcer or erosion produced by skin trauma or scratching and characterized by breakage of the epidermis, causing a raw linear or poorly defined area.

Fissure Splitting of skin (often with bleeding) and loss of natural elasticity. (See Fig. 6.22.)

Sinus A cavity or channel that permits the escape of pus or blood.

Fistula A tract that extends between two body 'cavities' such as the skin and a bursa or joint, the mouth and the nose (oronasal fistula), or the skin and the mouth (orocutaneous fistula).

Hypo-/hyperpigmentation Damaged skin resulting in changes in natural pigment distribution in the immediate vicinity. This can be either white (leukoderma/leukotrichia) or black (melanoderma/melanotrichia). (See Fig. 10.18.)

Basic histopathology

Whilst there are many different causes of tissue damage in the skin, it only has a limited repertoire of reactions to this damage; few skin conditions have defined and absolutely pathognomonic histological features and so a diagnosis can only be made when the histological features are considered in conjunction with the clinical history and the physical examination (macroscopic features, distribution and duration of lesions).

The term 'dermatitis' is commonly used clinically to describe a wide variety of inflammatory skin conditions with many different causes. From a histological perspective non-specific and specific changes can be recognized – sometimes histological changes are detected which can support a definitive diagnosis or the most probable cause. For example, the presence of acanthocytes in the epidermis is suggestive of pemphigus and the specific staining of fungal organisms can confirm a diagnosis of dermatophytosis.

The various skin disorders manifest the basic changes in different combinations and with varying degrees of severity.

Dermatohistopathology has a specialized vocabulary, examples of which are given below.

Epidermal terms

Alterations in epidermal growth or differentiation

Hyperkeratosis is an increase in the thickness of the stratum corneum and occurs in two forms. In orthokeratotic hyperkeratosis (also referred to as hyperkeratosis), the cells within the outer layers of the epidermis are anuclear, and, in parakeratotic hyperkeratosis (also referred to as parakeratosis), the cells contain nuclei. Both hyperkeratosis and parakeratosis are common non-specific terms describing responses to the effects of chronic stimuli (e.g. superficial trauma, and inflammation). They may occur as alternating layers in the stratum corneum due to variable waves of epidermopoiesis. An interesting form of hyperkeratosis occurs in the equine sarcoid as a result of secondary keratinocyte changes (see p. 391).

Pseudocarcinomatous or pseudoepitheliomatous hyperplasia mimics some features of squamous cell carcinoma but without invasion through the basement membrane and with differentiation of the epidermal cells. It can be associated with underlying chronic dermal suppuration, granulomatous or neoplastic processes and ulcers.

Necrosis is the death of cells within living tissue. Nuclei undergo pyknosis, karyorrhexis or karyolysis and loss of intercellular bridges with resultant rounding-up of cells. A normal-sized or swollen eosinophilic cytoplasm can be a means of identifying individual necrotic keratinocytes in histological sections. Causes include physical injury (lacerations, thermal burns), chemical injury (irritant contact dermatitis) and ischaemic injury (vasculitis, thromboembolism).

Apoptosis refers to programmed cell death. Apoptotic keratinocytes have brightly eosinophilic cytoplasm, a pyknotic nucleus with chromatin condensed along the margin, decreased size, and detachment from neighbouring keratinocytes. Apoptosis is seen with diseases such as lupus erythematosus, erythema multiforme and squamous cell carcinoma.

Alterations in epidermal cell adhesion

Oedema may occur *inter*cellularly or *intra*cellularly. *Intercellular oedema (spongiosis)* of the epidermis is characterized by a widening of the intercellular spaces with accentuation of the intercellular bridges, giving the epidermis a spongy appearance. If severe, this may lead to the formation of spongiotic vesicles within the epidermis. Even when severe, this is often a difficult change to detect because much of the fluid runs out during tissue handling. *Intracellular oedema (hydropic degeneration, vacuolar degeneration)* is characterized by increased size, cytoplasmic pallor, and displacement of the nucleus to the periphery of affected cells. This is a common feature of acute or subacute inflammatory dermatoses and is especially common in dermatophilosis and acute contact dermatitis. Intracellular oedema of the basal epidermal cells is usually associated with drug eruptions, erythema multiforme and toxic epidermal necrolysis.

Acanthosis is the term given to thickening of the epidermis usually due to an increase in the thickness of the stratum spinosum (prickle cell layer). It is a common feature of chronic inflammatory conditions of the skin. The thickening is often prominent in the rete pegs, which are commonly expanded and elongated with prominent interdigitating dermal papillae.

Acantholysis is the loss of cohesion between epidermal cells and the development of microscopic, intra-epidermal clefts, vesicles and bullae. It may be caused by, for example,

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oedema, proteolytic enzymes released by neutrophils or eosinophils in inflammation.

Acantholysis occurs at several levels in the skin and the location is important from a pathological and diagnostic perspective. It is a notable feature of the autoimmune pemphigus complex dermatoses.

Acanthocytes are microscopically distinct, 'free' nucleated epidermal cells within the vesicles with an intact nucleus and often a deeply eosinophilic cytoplasm. They are usually distinctive against the paler staining keratinocytes. Acantholysis and acanthocytes are important diagnostic features in immune-mediated diseases such as the pemphigus complex, some dermal neoplasms and some infections such as vesicular stomatitis and coital exanthema. The pemphigus complex can be subclassified into its various forms on the basis of the location of the acantholysis/acanthocytes (see p. 264).

Inflammatory lesions of the epidermis

Exocytosis is the migration of leukocytes or erythrocytes into the epidermis. Leukocyte exocytosis is common in inflammation and may be accompanied by spongiosis (intercellular oedema). Erythrocyte exocytosis is associated with trauma and circulatory disturbances such as purpura or vasculitis.

Pustules and microabscesses are intra-epidermal or subepidermal accumulations of inflammatory cells and fluid. Cells may be predominantly neutrophils (superficial microbial infections), eosinophils (parasitic disease, allergies) or, rarely, mononuclear cells.

A crust is a desiccated coagulum of plasma proteins, leukocytes, erythrocytes, epithelial squames and often microorganisms. Most crusts are not specifically diagnostic but careful preparation may reveal significant cells (e.g. acanthocytes, leukocytes, erythrocytes), and bacteria and fungi (e.g. dermatophilosis and dermatophytosis).

Alterations in epidermal pigmentation

Hyperpigmentation is excess melanin deposited within the epidermis. It is a common non-diagnostic finding in chronic inflammatory and hormonal dermatoses. It is rarely encountered in horses.

Hypopigmentation refers to decreased melanin in the epidermis. It may be associated with congenital or acquired idiopathic defects in melanization.

Pigmentary incontinence refers to the loss of melanin pigment from the basal layer of the epidermis due to damage to the cells of the basal layer and subsequent accumulation of the pigment in macrophages in the upper dermis. Pigmentary incontinence may be a non-specific lesion associated with inflammation; however, it is also seen with diseases that specifically damage the stratum basale, such as lupus erythematosus or discoid lupus.

Leukotrichia and leukoderma refer to decreased pigmentation of hair and skin, respectively.

Dermis

Degenerative disorders

Collagen degeneration characterized by increased granularity and staining intensity, develops in disorders associated with infiltrates of eosinophils, such as reactions to insect bite, mast cell tumour and eosinophilic collagenolytic granuloma.

Atrophic dermatosis is characterized by varying degrees of epithelial and connective tissue atrophy. The most common disorders in this category are endocrine, nutritional and developmental dermatoses. Microscopic changes include hyperkeratosis, epidermal atrophy, follicular atrophy and inactive hair follicles.

Inflammation

Dermal oedema is a common accompaniment to inflammation in the skin recognized by dilated lymphatics, widened spaces between blood vessels and separation of collagen bundles. Severe oedema may result in subepidermal vesicles and bullae, epidermal necrosis and stretching of collagen fibres, for example urticaria due to type 1 and type 3 hypersensitivity reactions.

Granulation tissue is proliferation of fibroblasts and blood vessels with prominent endothelia, for example ulcers undergoing repair.

Dermal deposits

Cutaneous (mucosal) amyloidosis appears in haematoxylin and eosin (H&E) stained sections as amorphous eosinophilic material. Often this is difficult to detect in normal sections, however.

Cellular infiltrates

These may be described in terms of the types of cells present and the pattern of cellular accumulations, so-called pattern analysis.

Neoplasia

The skin is the most common site for neoplasia in the horse. The various primary and secondary tumour types associated with equine skin are described in Chapter 18 (p. 377) (Baker & Leyland 1975, Cotchin 1977, Johnson 1998).

References

Baker J R, Leyland A 1975 Histological survey of tumours of the horse with particular reference to those of the skin. Veterinary Record 96:419–424

Cotchin E 1977 A general survey of tumours in the horse. Equine Veterinary Journal 9:16–21

Cotran R S, Kumar V, Robbins S L 1989 Inflammations and repair. In: Cotran R S, Kumar V, Robbins S L (eds) Robbins' pathological basis of disease, 4th edn. WB Saunders, Philadelphia, pp 39–86

Johnson P J 1998 Dermatologic tumors (excluding sarcoids). Veterinary Clinics of North America (Equine Practice) 14:625–658

Pollitt C 1995 Color atlas of the horse's foot. Mosby-Wolfe, London

Virchow R 1860 Cellular pathology. John Churchill, London, p 33

Further reading

Baker J R, Leyland A 1975 Histological survey of tumours of the horse, with particular reference to those of the skin. Veterinary Record 96:419–424

Carlton W W, McGavin M D 1995 Integumentary system. In: Thomson's special veterinary pathology, 2nd edn. Mosby-Year Book, St Louis, pp 461–511

Cotchin E 1977 A general survey of tumours in the horse. Equine Veterinary Journal 9:16–21

Dellmann H D 1993 Textbook of veterinary histology, 4th edn. Lea and Febiger, Philadelphia

Goodrich L, Gerber H, Marti E, Antczak D F 1998 Equine sarcoids. In: Veterinary Clinics of North America (Equine Practice) 14:607–623

Jubb K V F, Kennedy P C, Palmer N 1993 The skin and appendages. In: Pathology of domestic animals, 4th edn. Academic Press, San Diego, pp 531–738

McGavin M D, Zachary J F 2006 Pathologic basic of veterinary disease, 4th edn. Mosby, Philadelphia

Scott D W, Miller W H 2003 Equine dermatology. WB Saunders, St Louis, MO

Talukdar A H, Calhoun M L, Stinson A W 1972 Microscopic anatomy of the skin of the horse. American Journal of Veterinary Research 33:2365–2390

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2

The approach to the equine dermatological case

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There are many important aspects involved in the clinical investigation of a dermatological case. Often, the main clinical evidence of skin disease and the reason for the owner seeking a veterinary consultation is visually obvious in some form or another but the skin is easily overlooked as an important mirror for the health of the horse. Also it is often the most abused organ as a result of neglect, repeated washing, over-energetic grooming, poor harness and blanket hygiene, and of course the horse's own propensity for trauma. This means that often the primary clinical signs have been either obliterated or so altered and confused that it is hard to know whether any particular sign is the primary one.

It is remarkable how many times a diagnosis can be achieved even in long-standing and difficult cases when extra time and care are applied in the process of clinical investigation. The primary or first clinical evidence and information on the progression (improvement/deterioration) of skin disease can usually be defined with the help of a really thorough history. In cases where the historical progression of signs cannot be established, repeated clinical assessments will often provide useful information.

Intuitive supposition is often the mainstay of clinical practice – sometimes the signs are pathognomonic but even very experienced clinicians can make diagnostic errors when important aspects of the clinical process are not followed. A solid black spherical mass in the perineal skin of an aged grey mare may be assumed to be a 'typical' melanoma but it could be one of several different other options as well as one of several different types of melanoma (Fig. 2.1). Also a superficial glance at the skin may be grossly misleading. A close and detailed examination is an essential part of the diagnostic process even when the condition is apparently quite obvious. Often there are occasions when there are two distinct conditions present in the same field of view and then the clinician could easily overlook one of them (Figs 2.2 and 2.3).

Without a logical and systematic approach, there are few more frustrating situations than the diagnosis and treatment of skin disease in horses; treatment commonly fails when it is not focused on the cause of disease or recurrences develop when the pathogenesis and epidemiology are not considered. It is therefore essential that before any diagnosis is made, all the available facts are considered as a whole. Owners of horses commonly become distressed when skin problems fail to resolve, or even to follow a predicted course of treatment. Sometimes this leads to further frustration and often desperate measures such as seeking advice from non-professional sources or resorting to homeopathy. This may be a result of misinformation, misdiagnosis or in some circumstances improper treatment. There are some conditions that are extremely difficult to treat/manage and if the owner is not aware of these problems, there may be an unrealistic expectation.



Figure 2.1 This large black nodule complex developed from long-standing smaller nodules similar to the ones in the tail base, over a period of around 2 months in this 16-year-old grey horse. Although the lesion was initially diagnosed as 'melanoma' it was in fact a highly malignant aggressive undifferentiated melanosarcoma with a high mitotic rate. Prompt removal of all the nodules was undertaken.

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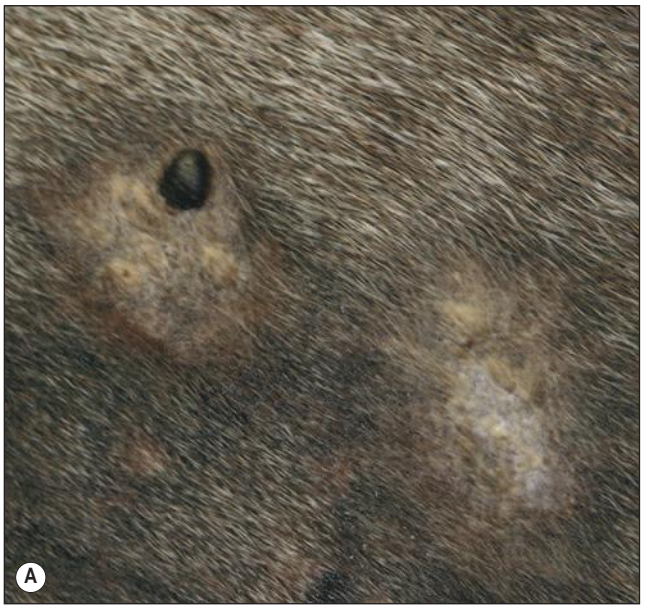




Figure 2.2 **(A)** This 3-year-old grey horse was presented for investigation of alopecic areas on the lateral neck. Close examination of this area showed a noticeably different black-coloured nodule. Further inspection confirmed that there were in fact many more of these small nodules. Biopsy confirmed both occult and verrucose sarcoid and melanocytoma. The treatment options for the two conditions are significantly different. **(B)** A diagnosis of melanoma was made for the lesions marked with arrows but the wider alopecic area was sarcoid.

CD

A realistic and helpful prognosis and effective treatment usually require an accurate diagnosis. Without this diagnosis, any treatment becomes little better than an educated guess. For example, the clinical similarities between some forms of the occult sarcoid, linear keratosis and dermatophytosis (ringworm) may lead to an error of treatment choice. The simple expedient of a thorough, exhaustive history and a careful clinical examination involving the whole horse will invariably lead to a correct diagnosis, correct treatment and a satisfied owner. Even if the condition is not resolvable, or carries a poor prognosis, the owner will be satisfied that a proper diagnosis has been achieved. In the days of information availability it is even more important to develop clinical acumen. Symptomatic treatment is often palliative rather than curative and does little to improve our understanding of equine dermatological disease; often this approach relies on natural 'healing' mechanisms and when this does not take place there is opportunity for complaint and recrimination.



Figure 2.3 This horse was presented for investigation of this pale wart-like area affecting the inner skin of both pinnae. A diagnosis of pinnal acanthosis (see p. 136) was made clinically. During the close examination a verrucose sarcoid was identified at the ear margin (arrow). Again the clinical approach is very different for the two conditions – the sarcoid has a much more threatening nature, and management options for the two conditions are very different.

It is tempting to overlook apparently trivial skin changes, but where two or more conditions coexist, each may have very different clinical demands, significance and prognoses **(Fig. CD2 • 1A–H)**.

A problem-orientated approach is an effective way of exploring a dermatological case; the diagnostic process is directed towards the identification of the recognizable problems which the horse has. The problems that are recognized do not necessarily have be dermatological – indeed, a comprehensive problem list will help enormously in both the investigative process and the selection of further tests. It is, for example, possible to overlook the more generalized clinical signs of liver disease when faced with a horse suffering from 'actinic' sunburn dermatitis. Recognition that the horse has lost weight, has icteric mucous membranes and a plaque of ventral oedema, can be vital aids to a diagnosis. However, it is also true that some other signs can be misleading and that is when clinical excellence comes to the fore.

Once the problem list has been established, a broad differential diagnosis can be formulated. Careful elimination of the various options may lead directly to a convincing diagnosis but it is a fact that the broad scope for diagnosis and the rather narrow panel of available signs means that further tests are often required in dermatological diagnosis (see Chapter 3). It is also important to recognize that the use of further tests will not necessarily lead to a diagnosis and so sensible judgements have to be made at all stages in the procedure and the owner apprised of the difficulties and the 'likely diagnosis'. The maxim 'communication is all' is particularly applicable to equine dermatology. An accurate diagnosis can, however, often be made.

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Experience and training enable the veterinarian to provide a prognosis: Is the condition treatable or not, and is it worth treating? If a targeted, specific treatment is available, economic and effective this may be used. In some circumstances treatment may be worse than the disease and then other aspects should be considered. The overriding concern has to be the welfare of the animal. Often owners will wish for 'cosmetic' treatments but when this conflicts with the welfare needs of the horse such treatment may not be ethical.

Symptomatic treatment alone often masks important signs and so it is vital that any concurrent or recent therapy of any type is declared. The clinical signs of insect bite hypersensitivity may be significantly altered by concurrent steroid therapy for an airway obstruction.

When approaching a skin disorder the clinician must establish whether the condition is a primary dermatological disorder (even if there are secondary systemic signs and consequences) or whether the skin signs are secondary to a primary systemic disease. For example, failure to recognize that a severe photo/actinic dermatitis was the result of advanced liver disease might allow the veterinarian to suggest that the horse had a trivial disorder that simply required local topical treatment. Conversely it might in fact be a case of simple sunburn and then an unsupported conclusion that the horse had advanced liver failure could be equally dangerous. The problems may be primarily dermal, and some of these may have systemic signs which could easily mislead the clinician. Furthermore, significant skin disease can arise as a secondary manifestation of a primary systemic disease. A pruritic horse can rapidly cause a lesion at the site – the basic question to ask then is: Was the lesion present before the pruritus or was the pruritus the first sign with a lesion developing subsequent to self-trauma? In the former case the primary sign is the lesion while in the latter it is the pruritus. The investigation and differential diagnostic possibilities are very different.

History taking (anamnesis)

The history of both the animal and the presenting problem are important independently derived parts of all clinical procedures. These should preferably be established and recorded before any clinical assessment or judgement is made. At the outset the history should be directed at the general history of the horse rather than at the presenting complaint. This provides the clinician with background information about the duration of ownership, the management and disease history of the horse. During the collection of this information it is sometimes hard to keep the owner focused away from the presenting complaint. Failure to obtain an adequate history often leads to errors of interpretation and wrong diagnosis.

The value of an individually formulated examination sheet (such as is shown in Fig. 2.7) cannot be overstated – it allows a structured, thorough questioning without omissions. However, even this may not always be totally comprehensive and the use of a structured form can sometimes be too prescriptive for unusual circumstances and presentations so they should be used with care. No information is too trivial – subtle facts may ultimately be the most significant. It is important to avoid undue repetitions but it may be necessary to ask questions in different ways to obtain the most helpful and reliable answers.



Figure 2.4 This horse presented with all the signs of insect bite hypersensitivity (IBHS/'sweet itch') and the owner took strenuous measures to protect the horse from the *Culicoides* bites. However, its feed was placed directly adjacent to the overgrown, damp, forested area in which the midges bred. In this case the horse did not in fact have any hypersensitivity but was being bitten unmercifully by myriad *Culicoides* midges. The signs described were 'typical' of IBHS but the simple expedient of observing the horse in its normal environment provided very helpful information and a simple curative solution!

The duration of ownership will carry an important message – recent purchase from an unwarranted sale with no 'papers' means that there will be no long-term information about the case and in fact it might have been sold because of the problem! The converse is the aged horse that the owner has owned since birth; everything should be known about it and the history may be so full that it becomes difficult to establish what is important and what is not. Nevertheless the extra information is far more helpful than the absence of a history. A pre-purchase examination at the time of acquisition should have identified if any problem was visible at that time. This examination can be assumed to be a complete and thorough examination of all body systems and the documentation may be supportive of a post-acquisition problem or it may have identified a significant or assumed insignificant problem even if the examination failed to make a diagnosis. Clinical descriptions can be very helpful in retrospect. For example, a small area of alopecia and hyperkeratosis on the medial thigh of a horse could be assumed to be a rub mark from a strap but if this corresponds some 2 years later with a proliferative fibroblastic mass it might well have been an occult/early sarcoid at the time. Descriptions are probably more help than a diagnosis that is wrong. If a diagnosis of ringworm (dermatophytosis) had been made in the example above the connection between the two lesions could have been overlooked because almost all dermatophyte lesions resolve spontaneously over some months.

One of the most overlooked yet important aspects of skin disease is the management system employed by the owner (Fig. 2.4). Changes in management immediately prior to the onset of a significant urticaria might suggest some aspect of the change could be responsible. Failure to ask the right historical questions can be very limiting. It is often helpful in long-standing cases to ask the client to provide a chronological list of the disease's progress, variation of feed or environment or of rider tack or harness which has been used. Management changes such as feed, water, bedding, pasture and transport, or unusual circumstances such as access to toxic waste or preservative-treated fence posts, should be established early.

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The history should establish all the relevant long-term information about the animal such as previous disease or illness, surgery, medication and the vaccination and worming status. Work and management details (including bedding types and feed regimens) provide useful information, particularly when considered with the time of onset of the condition.

Careful reviews of in-contact horses (or other species) and the environment are useful additions.

The concept of contagion between horses and between horses and the other species in the same place must be identified. Whilst some diseases may appear to be contagious because several animals are affected equally, there are some important non-infectious conditions that arise from management changes. For example the use of a strong chemical wash might affect all the horses and give the impression that the condition is highly contagious.

The history should include an assessment of in-contact horses as well as other species. Medication history and routine procedure details (vaccination, worming, dental and farriery) should be recorded. Consideration should be given to changes in management, peer group and contact species health and feeding. The long-term history should therefore be exhaustive and should include any significant abnormality of any body system at any time in the past.

The local environment is also important and it can be helpful to examine the horse in its own circumstance (see Fig. 2.4). Local disease surveillance can help enormously in some circumstances. For example, epizootic lymphangitis is endemic in some parts of Ethiopia but absent in others. In spite of the breed predilection of the Icelandic horse for insect bite hypersensitivity, the condition is very rare in Iceland because there are few (if any) *Culicoides* spp. insects there. Local knowledge is often helpful.

Owners may be inclined to blame environmental pollution for some skin diseases but these are few and far between and most such suggestions are born out of frustration and the desire to blame something/someone else. Local environmental conditions can also be important. Veterinarians may need, for example, to examine the pasture closely for photodynamic plants and toxic weeds; of course their recognition will require basic botanical skills also. *Mimosa* spp. and *Leucaena* spp. plants can cause specific dermatological problems. Seleniferous plants that concentrate selenium could be very significant if the horse(s) is already receiving selenium feed supplements.

Note

- Are many horses similarly affected since a local factory was constructed and are the environmental aspects of the factory being properly addressed?
- Are clinical signs and epidemiology consistent with toxicity?

Owners seldom recognize the importance of a full history and may become impatient and keep returning to the problem.

The owner's complaint

One of the most helpful contributions to the dermatological case is the 'owner's complaint'. This is the clinical problem as perceived by the owner and is a useful starting point for the process of obtaining the history of the specific complaint.

What is the owner worried about?

Although this can be misleading it does help to focus attention on the presenting problem and this should be recorded verbatim. Often the owner will continue to comment on this problem even during the acquisition of a history and so it may be best to delay this question until the general history has been obtained.

Having established the presenting complaint, the clinician can now obtain a detailed history of the complaint. This is often termed the focused history or somewhat misleadingly 'the short-term or specific disorder history'.

The specific disorder history

A careful chronological history of the presenting complaint is then obtained. The objective of the targeted history is to establish the duration and progression of the condition.

What were the first signs noticed and when were these seen?

The first signs are often the most important but they can be the most difficult to pinpoint simply because many signs are insidious in onset. Sometimes the first sign is overlooked. For example, an area of pastern dermatitis could be what the owner perceives as the first 'disease' situation but asking if it arose as a result of scratching and rubbing might result in an answer 'Yes, he had been rubbing and biting at his leg for some weeks, but he has done that for a long time!' In some cases the first sign is very noticeable even though the presented state may be very different.

The timing of onset relative to management or environmental changes is often critical to the diagnostic process. For example, horses that are pruritic during turn-out periods in summer but improve when they are indoors and in winter may have insect bite hypersensitivity. Exposure to certain plants and foods may precipitate an attack of urticaria.

Skin disease is often overlooked by an owner until it is in an advanced state and often some 'treatment' will have been applied – possibly repeatedly. Therefore attempts to establish the date of onset and the primary clinical signs are often frustrated.

How have these progressed (with or without treatment)?

Progression of disease is a critical issue but this is affected markedly by attempts at treatment. Particular attention should be paid to previous diagnostic tests, diagnoses and attempts at treatment (by owners, well-meaning lay people and/or previous veterinary surgeons). The nature of practice sometimes makes this difficult to establish, but it is important to determine whether any measures have had significant effects on the course of the condition (for better or worse) and questions may have to be rephrased to get a true answer. For example, asking 'What medication has been applied already?' may get a quite different answer from 'Have you put anything on the horse?' Of course I could ask 'What is this medication that I can see/smell?'… even if I can't!

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Failure of an owner to declare/admit to any previous treatments and applications can be frustrating. For example, if the clinician is not aware that the owner has already attempted treatment with anti-parasitic medication, he or she might be misled by the failure to find ectoparasites during subsequent clinical and laboratory investigations. A horse may be presented for investigation of a painful multifocal skin disease that appears from both history and clinical investigations to be bacterial in origin. The owner may have washed the horse immediately prior to presentation to enhance its appearance at the hospital and then cultures are almost bound to be unhelpful. Similarly owners will often use lay 'advice' and treatment from unqualified sources. This can result in significant complications and masking of the primary sign by secondary changes. Diseases which have been subjected to repeated (and sometimes ill-conceived) treatment attempts are particularly common in equine practice and a truthful, long-term history of the animal is very helpful. Careful, exhaustive inquiry of the client is a time-consuming exercise but is clearly essential.

Are other animals also involved?

Several animals (including other species in some cases) can be equally or similarly affected either through contagion or having been challenged by the same aetiological process. However, simply because several animals exhibit the same sign it does not necessarily mean that contagion is *certainly* involved although that is a strong possibility.

Note

Including the historical information on a 'dermatological examination form' provides a record of these events. It is often possible to come to a broad conclusion as to the nature of the case based upon the history alone (Fig. 2.5).



Figure 2.5 When presented with a non-specific sign such as this area of alopecia in the girth region, the history and clinical features are critical. Collection of samples will depend on the options available. The alopecia could have been due to rubbing, dermatophytosis, sarcoid, alopecia areata or scarring or several other conditions. Careful enquiry revealed that it had developed following an episode of traumatic rubbing and consequent bacterial dermatitis. There was little point in further tests because of the extent of the self-trauma.

For example, contact with other horses known to be affected by dermatophytosis ('ringworm') might be suggestive of the problem, but failure to continue the investigation by clinical assessment is potentially dangerous.

The signalment or basic animal description is also an important aspect because each of the aspects breed, sex, age, type, size and colour has some relationship to some skin disorders.

- *Breed* matters because there are certain inherited skin disorders such as the mane and tail dystrophy in Appaloosa horses and the fading/pinky Arabian syndrome (Table 2.1).
- *Colour* also matters because there are colour-related diseases (Table 2.2). Grey horses are liable to melanoma. Non-pigmented skin on the face and perineum is inherently predisposed to squamous cell carcinoma and predominately white-skinned horses are more liable to actinic (sunburn or ultraviolet-light-induced photosensitivity). White-skinned distal limbs have a justifiably poor reputation for bacterial infections. It is nonetheless dangerous to assume, for example, that because squamous cell carcinoma occurs more often on white skin, pigmented skin cannot be affected.

Table 2.1 Some of the breed-related disease conditions*

Breed	Disease predilection
Shire	Insect bite hypersensitivity (sweet itch)
	Vitiligo
	Coronary band dystrophy
Arabian	Fading/pinky Arab syndrome
	Vitiligo
	Lavender foal disorder
Icelandic	Insect bite hypersensitivity (sweet itch)
Fell pony	Fell pony immune deficiency syndrome (russet coat syndrome)
Appaloosa	Mane and tail dystrophy
Welsh	Insect bite hypersensitivity (sweet itch)
Belgian	Epidermolysis bullosa
	Linear keratosis
Quarterhorse	Hereditary equine regional dermal asthenia (HERDA/hyperelastosis cutis)
	Linear keratosis
	Reticulated leukotrichia
Thoroughbred	Atopy
	Vitiligo

* Many breed societies have policies regarding the breeding of horses with known genetic problems but others accept the consequences. There are still many disorders for which the heritability is uncertain to say the least. This list is by no means exhaustive. Being aware of the breed predilection can be a strong ally but it may also bias the clinician towards making the wrong diagnosis where the signs are similar for different diseases.

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Table 2.2 Some of the disease conditions related to colour*

Colour	Disease predilection
Grey	Melanoma
Albino/cremello	Actinic dermatitis (sunburn)
	Squamous cell carcinoma
	Melanoma
Coloured/(white) non-pigmented skin	Actinic dermatosis
	Photoactivated immune dermatitis (pastern and cannon leukocytoclastic vasculitis)
	Photosensitization
	Squamous cell carcinoma
	Bacterial infection (limbs/face)

*The list is by no means exhaustive but serves to illustrate the importance of the skin and coat colour of the patient. Note that there are conditions that affect different coloured regions on the same horse.

Table 2.3 Some of the disease conditions related to age*

Age	Disease prediction
Newborn < 3 months	Congenital disorders. e.g. epidermolysis bullosa/cutaneous agenesis/congenital curly coat syndrome (Paint breed)
	Vascular hamartoma/cavernous haemangioma
	Lavender foal/lethal white syndrome/Fell pony syndrome
	Congenital viral papilloma
	Cutaneous naevus
Young (<5 years)	Atopy
Middle age (5–15 years)	Mane and tail dystrophy (Appaloosa)
	Cutaneous melanoma
	Cutaneous histiocytic lymphosarcoma
	Insect bite hypersensitivity
Aged (>15 years)	Equine Cushing's syndrome
	Squamous cell carcinoma (penile/vulvar)
	Equine cutaneous melanoma

*The list is by no means exhaustive but serves to illustrate the importance of knowing the age of the patient.

- There are some other conditions that have been suggested as being more common in certain colours of horse. Thus, for many years there was an impression that sarcoids were commoner on grey horses but large epidemiological surveys have largely refuted this.
- *Age* also has a significant implication (Table 2.3). For example, grey horses over 12 years of age will almost certainly have a melanoma somewhere. The implications of a melanoma-like lesion in a young horse are quite different – in fact the condition can be regarded as clinically similar but very different pathologically and therapeutically. Similarly if a newborn foal is found with significant skin deficits the diagnostic options are far narrower than would be the case in older horses. In the former in the absence of any trauma, cutaneous agenesis could be the diagnosis while in the latter a large skin deficit would likely be of traumatic origin. Equine pituitary pars intermedia dysfunction is a very common disorder with cutaneous signs in old horses (>15 years of age); the condition does not occur in horses under 10–12 years of age.
- The *sex* of the patient can be important in the dermatological case – clearly there will be sex-related dermatological diseases affecting the genitalia but there are some important conditions that are more prevalent in one sex. For example, mast cell tumours are reported to be far more common in males than females. An entire breeding stallion that developed multiple vesicles and pustules on the preputial skin might suggest coital exanthema, especially if concurrently there were mares that had been served by him with similar vulvar lesions. This case illustrates the importance of correlating the findings from the history with the clinical details and the findings.

There are of course difficulties with some of these preconceptions. For example, melanoma and squamous cell carcinoma can be significantly more dangerous if seen in younger horses and assumptions on the likely diagnosis can be misleading if this is based only on the old-age-related more benign states. Similarly, an Appaloosa with a thin mane and tail may have a serious toxicity problem relating to selenium supplementation rather than the benign condition of mane and tail dystrophy.

Fig. 2.6 outlines a suggested approach to the dermatology case.

Example of a dermatological examination form

(Fig. 2.7)

This form is included to illustrate the type of form which might prove useful in investigation of dermatological problems in horses. A suitably abbreviated form can be prepared to cover re-examinations at subsequent dates. Clinicians are invited to copy or modify this to suit their own needs.

The clinical (physical) examination

Having completed a history a full physical/clinical examination should be performed. This must include a consideration of all the body systems and the vital signs (body temperature, heart and respiratory rates and capillary refill time and mucous membrane colour, body weight and condition score).

Note

There may be significant dermatological signs with systemic diseases and dermatological diseases with systemic signs.

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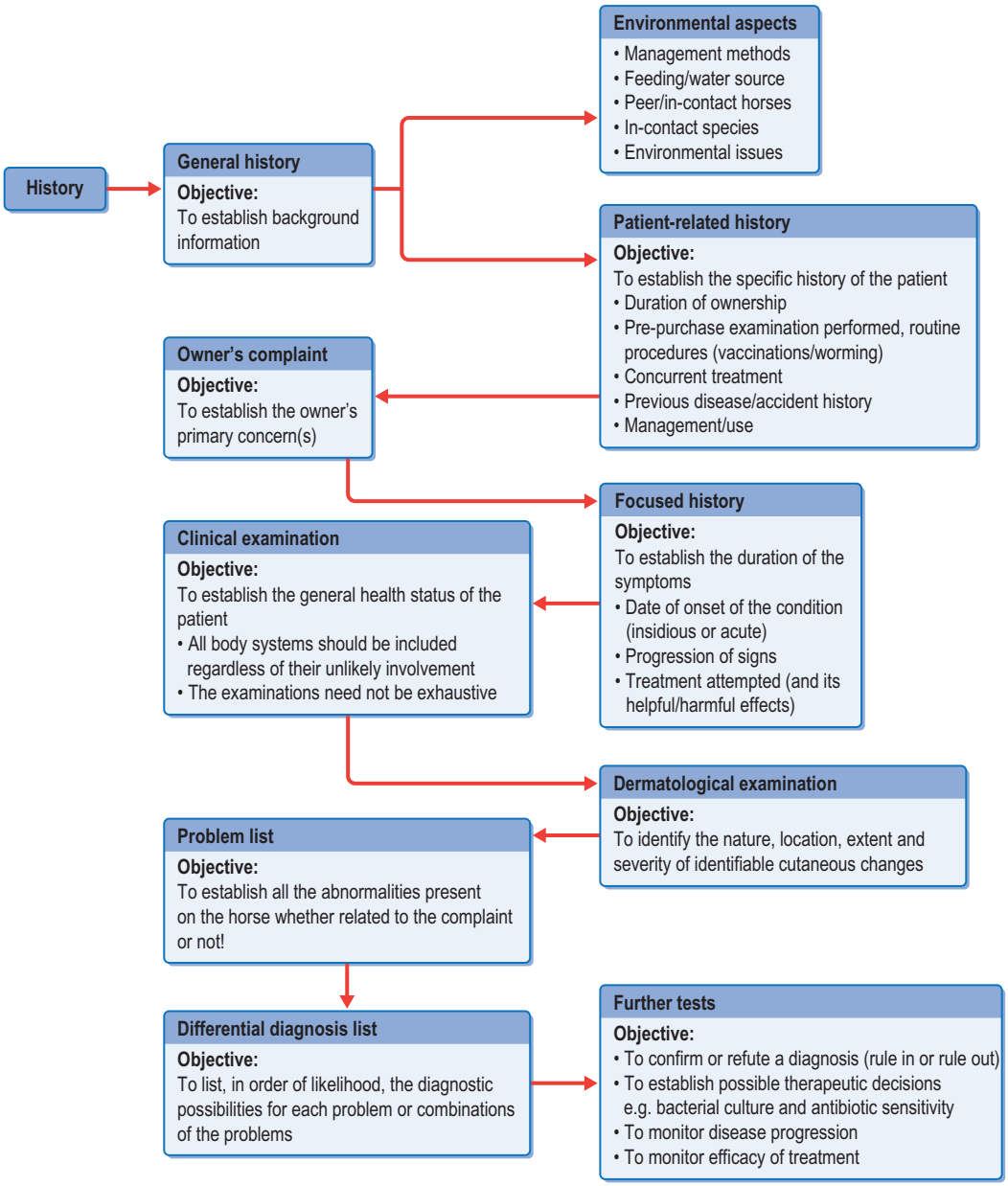


Figure 2.6 A suggested approach to the dermatology case.

The primary objective of the physical examination is to establish what systems are involved primarily or secondarily. The clinical examination should begin with the assessment of the general health, demeanour and bodily condition of the horse. An early overall view of the horse in good light is useful and might help to establish the distribution of any obvious skin abnormalities as well as many aspects of the other major body systems.

An older horse may have a serious bacterial skin infection as a result of immunocompromise from equine Cushing's disease. Dealing with the skin infection on its own may be successful temporarily but management of the whole case should have better long-term results.

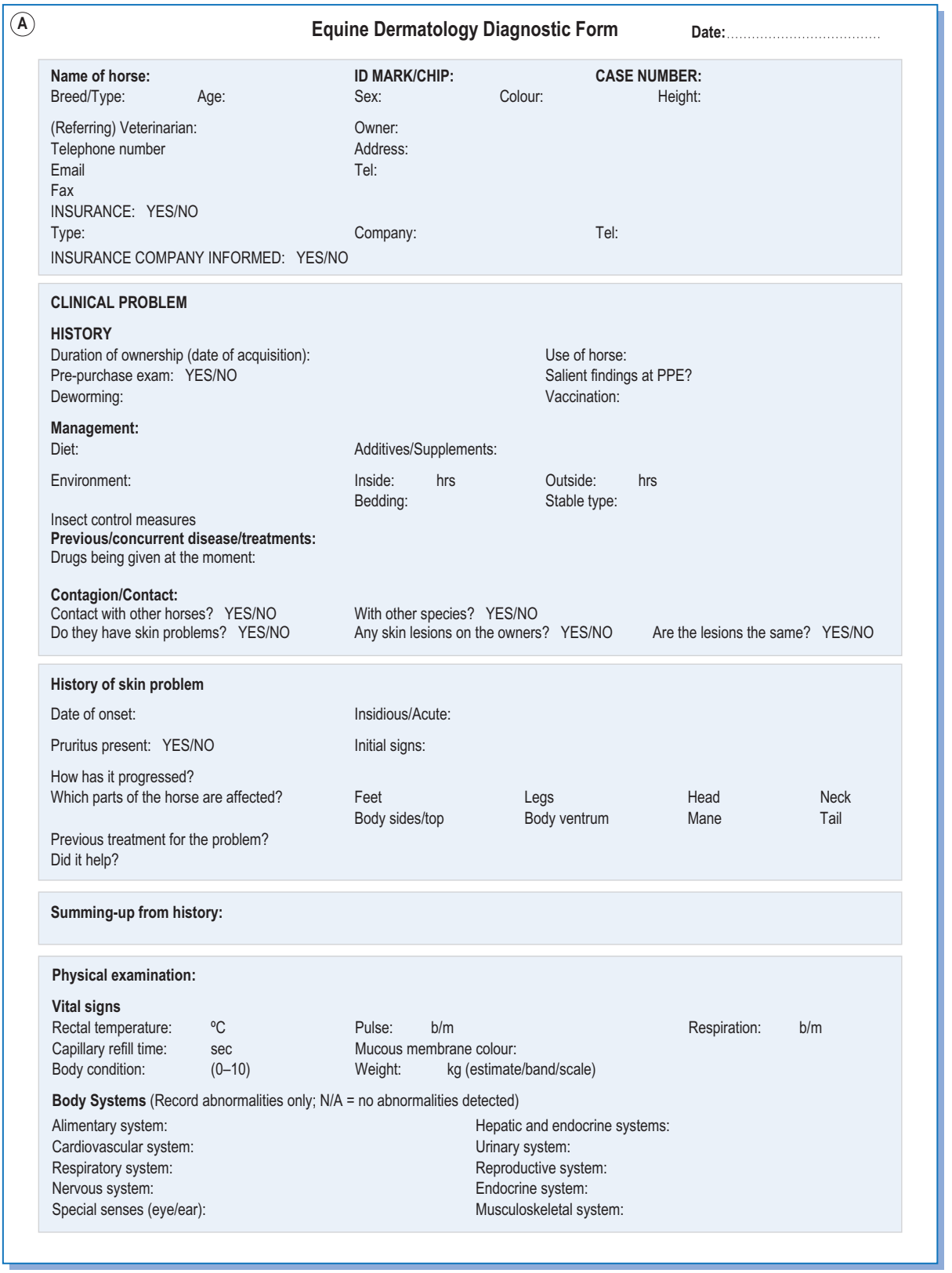
Similarly a horse with photoactivated dermatitis may have serious liver disease or may simply have been exposed to a primary photodynamic plant such as *Hypericum perforatum* (St John's wort) or buckwheat. The prognosis for the two possibilities is markedly different and yet both may cause a very similar clinical presentation. Of course the other possibility is that the horse has been exposed to very strong sunshine and has simply been sunburnt!

Failure to perform a full clinical examination of all the body systems can lead to errors of diagnosis.

For example, a horse showing evidence of photosensitization might also exhibit icterus (jaundice) and ventral oedema and the underlying primary problem may be hepatic failure. Subsequent treatment of the dermatological problem alone may be disappointing without the primary disease being addressed.

In practice, however, an exhaustive clinical examination involving all the body systems is seldom performed. Although in reality they are seldom actually required, the clinician should remember that no channel should be completely forgotten. It should be possible from a relatively superficial clinical examination and an accurate and complete history to establish whether any particular body system might be worth a more detailed examination.

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(A)

Equine Dermatology Diagnostic Form

Date:.....

Name of horse: **ID MARK/CHIP:** **CASE NUMBER:**
Breed/Type: Age: Sex: Colour: Height:

(Referring) Veterinarian: Owner:
Telephone number Address:
Email Tel:
Fax
INSURANCE: YES/NO
Type: Company: Tel:
INSURANCE COMPANY INFORMED: YES/NO

CLINICAL PROBLEM

HISTORY
Duration of ownership (date of acquisition): Use of horse:
Pre-purchase exam: YES/NO Salient findings at PPE?
Deworming: Vaccination:

Management:
Diet: Additives/Supplements:

Environment: Inside: hrs Outside: hrs
Bedding: Stable type:
Insect control measures
Previous/concurrent disease/treatments:
Drugs being given at the moment:

Contagion/Contact:
Contact with other horses? YES/NO With other species? YES/NO
Do they have skin problems? YES/NO Any skin lesions on the owners? YES/NO Are the lesions the same? YES/NO

History of skin problem

Date of onset: Insidious/Acute:

Pruritus present: YES/NO Initial signs:

How has it progressed?
Which parts of the horse are affected? Feet Legs Head Neck
Body sides/top Body ventrum Mane Tail
Previous treatment for the problem?
Did it help?

Summing-up from history:

Physical examination:

Vital signs
Rectal temperature: ºC Pulse: b/m Respiration: b/m
Capillary refill time: sec Mucous membrane colour:
Body condition: (0–10) Weight: kg (estimate/band/scale)

Body Systems (Record abnormalities only; N/A = no abnormalities detected)
Alimentary system: Hepatic and endocrine systems:
Cardiovascular system: Urinary system:
Respiratory system: Reproductive system:
Nervous system: Endocrine system:
Special senses (eye/ear): Musculoskeletal system:

Figure 2.7 (A, B) Example of a dermatological examination form.

Many of the common skin diseases are primary and present with sufficient clinical signs and history to justify an abbreviated clinical examination. However, there is then a significant danger with problem skin diseases, and especially those which do not fall neatly into recognizable patterns, that important aspects of the general health of the patient might be missed.

The condition may be localized or more generalized. This in itself aids the diagnosis, as some conditions are usually (or always) localized whilst others are more usually

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B

Dermatological examination

Pruritus: Absent Present Mild Moderate Severe

Was lesion present before pruritus? YES/NO/UNKNOWN Was pruritus present before lesion? YES/NO/UNKNOWN

Seborrhoea: YES/NO

EXTENT : MILD/MODERATE/SEVERE CHARACTER: Dry/Greasy

LOCALIZED / GENERALIZED

State locations:

Primary lesions:

(circle) macule papule patch pustule vesicle
bulla wheal nodule tumour

Secondary lesions:

erythema scale crust fissure alopecia
comedone ulcer erosion excoriation
lichenification hyperpigmentation

Hair

Colour change?: YES/NO/NOT OBVIOUS Easily epilated?: YES/NO Greasy coat? YES/NO Dry coat? YES/NO

Skin texture: Normal Thickened Thin Dry Inelastic

Problem list:

Differential diagnosis (in order of likelihood):

Diagnostic tests and results (ring tests performed and * if test performed 'in house')

Name of laboratory: Report number: phone:
Date of dispatch: Date of result/report: written:

1. Skin scrapings:
2. Sellotape preparation:
3. Groomings:
4. Culture/sensitivity:
   - Viral culture/ID:
   - Fungal swab/culture:
   - Yeast prep/culture:
   - Bacterial swab:
   - *Dermatophilus* prep/culture:
5. Needle aspirate:
6. Biopsy:
   - Bacterial culture:
   - Histopathology:
   - Immunofluorescence:
7. Serology:
8. Other:

Final diagnosis:

Treatment prescribed (in detail):

Photographs (number/location/type):

Revisits: YES/NO

Date: Sheet number: Case number:

Figure 2.7 (A, B) (Continued)

generalized. In some cases careful assessment of the location may help significantly with a diagnosis. Thus an area of apparent alopecia may simply be the result of a rug rub – placing the rug onto the animal and testing for contact with the suspicious area can be helpful (Fig. 2.8).

Note

Simple observation and palpation are the 'forgotten diagnostic tools' of the clinical examination. It is often possible to make a diagnosis when the simple clinical procedures are performed completely and carefully.

In any case the clinician should establish the extent and location of the problem and then the variety or similarity of the lesions. It is easy to assume that a case has a single disorder when in fact there may be two or even more present concurrently and these may or may not be connected. Furthermore, specific lesions may look similar from a distance but an illuminated magnifying glass **(Fig. CD2 • 2)** or even an auriscope **(Fig. CD2 • 3)** is another excellent method of making a closer examination of a small area of the skin. The latter, however, covers a very small area at a time. In this way the skin can be examined down to its surface and any movements of parasites, scale or crusting will be obvious. There are sophisticated fibreoptic and

CD

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Figure 2.8 This horse presented for pre-purchase examination with an area of alopecia over both shoulders. This coincided with the pressure points of the rugs it was wearing and magnified skin examination showed no evidence of alopecia or inflammation. Hair rubbing such as this is not alopecia.



Figure 2.9 This horse was admitted to hospital for removal of a localized proliferative lesion on the glans penis following a cursory examination. On preparation for surgery extensive pre-carcinomatous dysplastic changes were detected higher up in the prepuce. This changed the prognosis significantly and meant that much more extensive surgery and ongoing treatment was necessary.

digital camera systems but they are expensive and largely unnecessary. They do, however, provide the possibility of a picture to either assess later or to store as an indicator of the 'current position'. A camera with a macro-facility is certainly a useful aid.

Clearly, there are justifiable short-cuts which are acceptable after experience has been gained, but clinicians should be particularly wary of jumping to conclusions in dermatological disease either by assuming a diagnosis or even assuming that the skin is the only structure involved. Generalized pruritus is sometimes the most obvious external symptom of paraneoplastic syndromes associated with serious (often small) internal neoplasia (see p. 383). Thus, an examination of the musculoskeletal system and a lameness work-up or a detailed reproductive examination should not be needed in most cases. However, some serious skin disorders have systemic signs that may result in joint inflammation and there are important dermatological diseases that affect the external genitalia. An assessment of the neurological system may be specifically helpful in some cases such as photosensitization when, for example, hepatic encephalopathy may be present. A focal area of pruritus on the neck could even be associated with a cervical vertebral fracture **(Fig. CD2 • 4A–D)**.

CD

It is important to examine as much of the animal as possible. Even where there may be an obvious lesion in one particular location, a complete examination is always helpful in establishing the full clinical presentation and therefore the complete clinical picture. For example, during a cursory examination for blood loss from the prepuce a horse may have an obvious proliferative lesion on the glans penis. A presumptive diagnosis of squamous cell carcinoma or squamous papilloma can be suggested but failure to examine the whole of the penile and preputial skin may mean that the choice of treatment is wrong (Fig. 2.9).

Where skin disease appears as a consequence of internal disease, local skin treatment is likely to be ineffective (or palliative) unless the underlying disease is resolved. Furthermore, successful treatment of the underlying disease may result in rapid resolution of the skin disease without the need for extensive local treatment beyond obvious supportive measures. From a casual perspective a rectal examination may seem to be an unusual dermatological investigation but this can and perhaps should be done in most cases where neoplasia and some generalized inflammatory conditions are suspected. A brief overall view of most of the body systems may allow elimination of much of the time-consuming clinical examination.

Note

In dermatological cases a thorough clinical examination can be particularly rewarding.

Following a broad physical examination, a more detailed dermatological examination is carried out. The nature, number, size and distribution of the lesions should be recorded. Reference to the type and size of lesions is based upon the accepted morphological descriptions of the common skin lesions (see Chapter 1), which should be readily recognized by the clinician. It is often useful to refer to these recognized lesion types when discussing the case with colleagues or with a pathologist who might become involved in the case.

The specific dermatological examination is obviously the major part of the clinical process of investigation of skin disease and again the method for this can be laid out on a prepared form (see Fig. 2.7) so that little is overlooked. The form can be used again for reference if the problem fails to resolve. The physical examination may require to be repeated at a later stage, particularly when an apparently simple skin disease fails to respond to treatment, and it is important to record all the findings of the initial and subsequent examinations. Additionally, reference to previous records provides information on the extent and type of changes since the last examination. The owner (and veterinarian) might be under the impression that the condition has deteriorated whereas

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in fact it might have improved or new lesions might have developed with a different distribution. A chronological record is particularly useful to pathologists in identifying the likely age of a lesion or change in the skin.

Note

Skin diseases are often very photogenic and photographs provide an exact description which cannot always be effectively provided by a written account. A photographic or video record is a simple, useful and under-utilized diagnostic and clinical asset in dermatology.

In addition, photographic material provides an amazing reference resource.

There may be indications for specific organ function tests. These might include haematological analysis, serum biochemistry, abdominocentesis, faecal and urine analyses, rectal examination, ultrasonography and gastrointestinal absorption tests. The dermatologist should be particularly wary of the trap of a 'haematological diagnosis'. There are few primary skin diseases which exhibit significant haematological findings. However, some diseases with secondary dermatological changes have important haematological changes. For example, equine pituitary pars intermedia dysfunction (PPID/equine Cushing's disease) commonly exhibits a panel of pathognomonic signs including hirsutism, polydipsia/polyuria and secondary skin infections of all types but haematology changes including hyperglycaemia and hypercortisolaemia will make the diagnosis even more robust. Photosensitization can be a cardinal sign of advanced hepatic disease and the diagnosis can be supported by haematological evidence of liver failure and then ultrasonography and liver biopsy can be justified.

The problem list

Following the history taking, clinical examination and recording of the 'owner's complaint', a problem list should be compiled. It serves to focus attention on the defined problem(s) which have been identified. The list can be as detailed as the clinician feels is useful. It provides a concise summary of the salient features of the case as well as the findings of historical and clinical investigations. It is potentially dangerous to ignore the rare conditions because even these, by definition, appear from time to time.

It is useful to categorize the condition if possible into one or more of the recognized syndromes or major disease patterns (see below). While it may be more correct scientifically to make an approach based on the causal agent or agents, these are commonly not obvious and their identification often requires further tests.

It is often possible at this stage to detect the presence or absence of systemic disease involvement (whether related to the dermatological problem or not). Although this may in some cases be misleading, it may still have profound influences on treatment options. Thus, a pony with laminitis may be precipitated into a life-threatening acute episode following a single corticosteroid dose administered to treat a mild inflammatory skin disease. Similarly, a horse with liver failure may show no skin disease until it is subjected to high levels of ultraviolet light/sunlight and may then not benefit from being turned out into sunlight as part of an attempt to treat a suspected or confirmed dermatophytosis (ringworm).

The term syndrome is defined as an aggregate of clinical features, signs or symptoms which form the picture of a disease entity. This is therefore not a term that is theoretically applicable to the features of dermatological disease. In the horse the range of clinical signs is limited for a wide range of disparate disorders and so for the purposes of this text they will be termed syndromes. For each syndrome a number of possible diagnoses can be suggested – the differential diagnosis.

The clinical problem can usually therefore be classified into one or more of the major classes outlined below. Further breakdowns of these broad categories of disease can be found in Chapter 19.

1. *Pruritus.* Itching or irritation of the skin which encourages behavioural changes such as self-inflicted trauma.
2. *Alopecia and other alteration of hair quantity or quality.* Complete or partial baldness (loss of hair) or alterations in hair density or quality or over a variable area. Alopecia is not the traumatic rubbing/breaking of the hair.
3. *Nodular disease.* A circumscribed or diffuse thickening of the skin of variable size and extent. Nodules can be broadly divided into inflammatory nodules, non-inflammatory nodules and neoplastic nodules.
4. *Dry dermatosis* (*scaling and flaking, crusting*). An accumulation of dry scales and crusts arising from excessive flaking of the skin or from accumulation of dried exudate on the surface of the skin.
5. *Moist dermatosis* (*weeping and seeping or crusting*). An accumulation of fluid exudate or dried exudate (crust) on the skin surface arising directly from skin damage/inflammation or another pathological process.
6. *Pigmentary alterations.* Changes in the normal pigmentary pattern of the hair coat and/or the skin.
7. *Pastern dermatitis.* Inflammatory and non-inflammatory changes in the coronary band and pastern skin are very common in horses and whilst some are linked to other skin and systemic diseases this is a recognized category of dermatological pathology.
8. *Abnormal sweating patterns*. Sweating abnormalities reflect alteration in autonomic control of cutaneous blood vessels or excessive circulating catecholamines.

One or more of these major forms of skin disease may be presented and this should be considered with other clinical features such as the anatomical location, the duration and progression and the epidemiology of the problem.

The inclusion of multiple recognizable skin abnormalities in the problem list and the derived differential diagnosis list is almost inevitable. For example, scaling and crusting over the fetlock region and facial and limb pruritus might be recognized. A horse may be pruritic and have obvious excoriation and exudation as a result but the affected area may have nodules that are the primary cause, or possibly they may be unrelated. Sometimes there are certain connections between the signs – a horse presented with insect bite hypersensitivity (IBHS) may have generalized skin thickening with lichenification with rugae in the upper neck skin and hair loss where the rubbing has broken the hair.

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A few open papular lesions may be present with exudate, and even focal (nodular) skin thickening may be present in some affected areas.

It is always easy to assume that the horse has only a single disease entity and that a particular disease only has one set of clinical signs but this is a trap for the unwary clinician. Multiple diseases are relatively common. They may be completely unrelated and have completely different histories and clinical implications. However, they can be indirectly related and in some cases the main sign may be secondary rather than associated with the primary underlying disease – an immunocompromised horse may be presented with anaemia due to a severe lice infestation. A horse may have a long-standing viral acanthosis in its ears but then develop pruritus associated with a psoroptic mite infestation. There is no reason why a horse could not have sarcoids and eosinophilic granuloma with collagen degeneration as well, for example. The unwary clinician may be drawn to the most obvious sign and link the symptom to it – this would clearly be wrong! The reason why this is an important concept is that the treatment for the various conditions may be conflicting. Corticosteroids administered to control IBHS-related pruritus in a horse with Cushing's disease or laminitis could be very harmful. An alternative strategy may need to be constructed.

Similarly, an assumption that a particular disease has a completely predictable clinical presentation is a trap. Almost every disease entity has variations in clinical signs. The variations can even be present in localized lesions. A sarcoid may show several different forms of the disease in a single 'focus' (Fig. 2.10).

The diagnostic process is made even more difficult when a logical diagnostic process is not applied. The complexity of the options should not distort or bias the diagnostic process. Although these specific points serve to identify the character of the problem(s) present in the case, in themselves they provide little information on the aetiology, diagnosis or treatment options. Their main use is in providing a basis for some of the differential diagnoses that will need to be considered. Each condition should be initially considered in its own right and the combined lists examined to see whether there are common diagnostic possibilities. The next stage in the process is to establish a list of differential diagnoses, i.e. specific diseases which may be present or which have the features identified.

Of course the clinician will seek to achieve a definitive diagnosis but in many cases this is not possible directly from the problem list – further tests are commonly used in dermatological diagnosis.



Figure 2.10 This sarcoid shows the variations that are possible within the broad diagnosis of sarcoid. This sarcoid complex has nodular, verrucose (warty) and occult types (see also p. 367).

Differential diagnosis

After completion of the problem list, a differential diagnosis list consisting of probable and possible diseases can be assembled. Flow charts (algorithms) can be useful for both inexperienced and experienced clinicians to develop the differential diagnosis. Very few diseases are predictably identical in all cases and so serious errors can be made by attempting to make a disease fit a single or few clinical features. Subtle and more obvious variations are usually present to trap the unwary clinician. Nevertheless experience will enable a degree of intuitive supposition.

Note

What is common in one geographical area is not necessarily common in others and some conditions are commoner in certain management systems.

No possible differential diagnostic options should be totally ignored. The most likely conditions will probably be placed at the top of the differential diagnosis list with the less likely ones placed lower down. The options will vary widely in different geographical areas, at different times in the year and under different management conditions. For example, urticaria is relatively common in horses being fed very high cereal rations and others only occur in defined seasons.

Some conditions can justifiably be left out of serious consideration because the diseases only exist in certain areas of the world.

For example, glanders is currently geographically very restricted and possibly could be justifiably ignored in the United Kingdom – however, the dangers of such an approach are obvious! Deep mycosis such as Florida leech disease occurs in a restricted area in the USA, South America and Australia and has not been recorded elsewhere. There are other areas where it is theoretically possible and it may then be placed low on the list. Insect bite hypersensitivity is very common in Scotland but is not recognized in Iceland even though the Icelandic pony has a high genetic predisposition to the disease, simply because in Iceland there are no *Culicoides* spp. midges, whereas in Scotland their numbers are astronomical!

Thus, some diseases are common in some areas and almost never encountered in others. Disease is no respecter of national boundaries and within countries there may be some regions where a particular condition is common – epizootic lymphangitis due to *Histoplasma farciminosum* infection, for example, is relatively common in the Highland

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areas of Ethiopia but rarely affects horses or donkeys in the Rift Valley regions. Fistulous withers due to *Onchocerca cervicalis* is common in Mali but rare in adjacent countries with similar climates, but a clinically similar 'classical' fistulous withers can be caused by *Brucella* spp. bacteria in other countries where this exists. Geographical restriction of diseases is, however, becoming much less significant as rapid, worldwide transport of horses has become commonplace. Unusual diseases may appear and epidemics of highly contagious diseases can also occur more readily. It is, of course, impossible in a book of this type to list every variation in every disease.

After considering the clinical signs, the problem list and the differential diagnosis list, it may be possible to identify a specific and definitive diagnosis and take appropriate therapeutic measures. Experience and cognitive intuition are often very supportive of a diagnosis. For example, experienced clinicians can usually make a diagnosis of melanoma or sarcoid where the presented lesions have an almost pathognomonic appearance **(Fig. CD2 • 5)**.

Similarly horses that are presented with pruritus that is worse outside in summer months than inside and in winter months can be assumed to have insect bite hypersensitivity (IBHS/'sweet itch'). There are of course dangers in some of the assumptions – the diagnosis could be wrong! In most circumstances, however, a short-list of possible diagnoses whose clinical features are basically similar and which are not separable on clinical grounds alone will be considered. Repeated examinations may help to narrow the diagnostic options but under many circumstances special (further) tests will be performed to either confirm a presumptive diagnosis or 'rule out' some of the options. A biopsy taken from an ulcerated lesion on an eyelid could be a squamous cell carcinoma or a sarcoid and a biopsy should differentiate them easily. Of course on occasions the true diagnosis will have been overlooked – the eyelid lesion may turn out histologically to be a mast cell tumour. This assumes of course that the correct samples were taken and that the pathologist was given the correct information.

Investigative tests (see Chapter 3)

From the differential diagnosis list, a number of further investigative methods can be identified either to confirm or to eliminate ('rule out') specific conditions. Supportive diagnostic tests are often used in dermatological cases because of the outward clinical similarity of many conditions and the limited range of clinical signs detectable through clinical examination alone.

There is a significant danger in proceeding straight to this stage without any previous clinical examination or historical information, thus relying totally on the services of a bacteriologist, pathologist or clinical pathologist. This is not an acceptable practice in dermatological cases and the specialist concerned carries an unfair and unjustifiable responsibility for diagnosis.

The available diagnostic tests are described in Chapter 3.

Diagnostic confirmation

The diagnosis is that part of the investigation which leads to the identification of a specific disease process. Some conditions are classified by names that may or may not be instantly recognizable whilst others are, unfortunately, given a more vague morphological or pathological 'diagnosis'. For example, a diagnosis of pemphigus foliaceus can be very helpful because the disease has a recognizable and scientifically investigated pathology and evidence-based treatments, but a 'diagnosis' of 'dermatitis' or even worse 'moist eczema' is not anything like as useful. Owners may be impressed by any vague/broad name for a disease but this approach does nothing for the long-term development of dermatology as a specialty.

If a diagnosis cannot be achieved safely, the clinician should at least try to categorize the condition into one of the major recognized syndromes (listed above). Consideration should then be given to the selection of the most appropriate further tests and procedures. A direct leap into symptomatic treatment should not be the objective although on rare occasions there will be little practical alternative.

CD

It is unwise and unfair to expect a clinical pathologist, bacteriologist or dermatohistopathologist to support a diagnosis without access to all relevant clinical and historical information. A photocopy of the case record sent with any specimens is very helpful and saves time and effort – provided of course that the records are more than just a record of a visit and a price for the services.

Various dermatological diagnostic tests are available; some are simple and some more complicated. Regular contact with pathologists often provides very valuable information and a good rapport is to be encouraged. Unless the diagnostic tests are standard and performed frequently, it is often very useful to consult a specialist pathologist (or technician) before embarking on them. For example, skin biopsy in particular appears to be simple but there are specific types of biopsy technique or fixatives which may be useful in particular cases (see Chapter 3). A pathologist's decision on the orientation, site and type of biopsy can often be significantly different from the standard clinical approach.

Once a decision has been taken with respect to the selection of further tests, the clinician must obtain all the relevant equipment to perform the procedures. A wide range of tests is available and most of the regular ones are described in detail in Chapter 3.

If, after all the possible investigations have been completed, a diagnosis still cannot be reached, a thorough review of both the clinical and historical features should be performed. This is made very much simpler if a full standard record has been taken at each examination. Regular access to good textbooks and well-illustrated clinical papers in refereed journals is very helpful but often cannot be relied upon to provide a diagnosis. It is often helpful to discuss the case with a colleague or a pathologist or a specialist dermatologist who might have different experiences and a different perspective on it.

Note

Professional isolation is not a useful clinical ideology. Discussion and sharing of information through any forum at all is the way in which problems are solved.

'There is nothing new – someone somewhere has seen it before.'

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Recording the findings

There is little point in repeated clinical investigations if proper records are not kept. The time spent in recording the history, the results of the clinical examination and the results of diagnostic tests is always well spent. It is unrealistic to expect the owner to remember what was said and it is unlikely that a busy clinician will recall the exact details of any case some weeks later. Furthermore a good clinical record will enable a colleague or specialist to study the progression (deterioration/improvement) of the case clearly and accurately. The threat of litigation and acrimony has never been greater and a good supporting documented case file is possibly the strongest ally a clinician can have in this adversity. An added benefit is the ability to derive case study and publication material for retrospective studies and case discussions. In this way sensible judgements can be made about prognosis, treatment and diagnosis. Many equine skin diseases have little published information and so for these at least it is difficult to find helpful information on the likely results of treatment.

The value of a pre-printed proforma sheet that includes the correct information and guidance cannot be overstated – it need not be complicated but it should be thorough. The form should have a logical structure that enables the information to be recorded in a consistent way. It is easy to construct 'forms' that simply include a series of disparate questions with no logical progression but these often confuse rather than help. Use of tick boxes and computer-friendly systems will help enormously. It is even possible now to enter clinically relevant data instantly onto hand-held/palm-top computers so that paperless records can be used. These can be sent electronically to the hospital recording system or specialists in seconds and so clinical results become more usable and diagnoses can be reached earlier. The concept of recording is helped by the proper training of the clinicians so that lesions can be quickly identified and categorized correctly. A photographic or even video record of the case and the salient features add to the value enormously because the skin is one of the most visual organ systems. Modern hand-held computers and cameras mean that all the information can be collated and stored instantly. The results of further tests and a record of the treatment can be appended either electronically or in hard copy (or both). Repeat visits and owner reports of changes and even a series of photographs can help enormously.

Evidence-based medicine

> *If you always do what you've always done, you will always get what you always got.*

An owner of a horse has the right to expect that his/her veterinarian is up-to-date, well-informed and able to correctly interpret the scientific literature to the benefit of the patient. Evidence-based medicine has become the standard by which clinical practice is governed and audited. It provides the basis for informing the client about the various options that are available for diagnosis and treatment. The principles apply as much to dermatology as to other systematic clinical medicine and as much to the horse as to other species.

Evidence-based medicine is the process of systematically finding, appraising and interpreting contemporaneous research findings as the basis for clinical decisions. It is a means of identifying gaps between research evidence and clinical practice that should be interpreted with care and if possible the evidence should be improved by focused research effort. The application of evidence-based medicine means that expensive/ineffective/harmful decision making can be avoided. It encourages and supports the practice of quality-controlled medicine that uses the information derived from properly conducted scientific trials, published in peer-reviewed journals. It can be applied in any situation where there is good documented data on clinical diagnosis, prognosis and treatment.

Evidence-based medicine enables the implementation of proven results on new techniques, drugs or procedures derived from combined results from the open sharing of information. The frank exchange of information on both successes and failures is, however, an ideal that is very difficult to deliver. No clinician really wants to openly admit to repeated failure and may wish to 'protect' his/her successes, especially when this relates to the development of a new drug that may have cost an enormous amount to develop. The whole concept relies on high quality audited responses and self-critical analysis, free of bias that provides the client with confidence that the best thing is in fact being done for their horse.

The problem with this approach is the generally poor quality of the research, the lack of properly conducted scientific studies and the enormous amount of anecdotal and ill-informed 'information' that is easily available now. Where the published evidence for treatment in particular is poor or non-existent, treatments are often based on a 'hit-and-miss' approach – if a treatment works it must confirm the 'diagnosis'! Whilst the response to well-directed specific treatment may indeed help to establish a diagnosis, this is not a reliable method; it can be counterproductive and misleading.

Where treatments consistently fail either the diagnosis is wrong, or the treatment choice is wrong, or there is no established effective treatment or the way in which the treatment is applied is wrong. There are plenty of options for the failure of treatment.

The steps involved in evidence-based medicine are:

1. To formulate clear clinical diagnostic, prognostic or therapeutic questions from the patient's problem(s).
   - **a** What is the best/proven way to confirm the diagnosis?
   - **b** What advice can be given to the owner regarding the prognosis?
   - **c** What treatment is known to have the best clinical basis and results?
2. To search the literature for relevant clinical articles.
   - **a** Recent refereed papers and reports of properly conducted trials provide the best source of information.
   - **b** Anecdotal 'internet-based' advice is possibly the worst source unless this is backed up by a reputable clinical institute.

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3 To evaluate and critically appraise the evidence for its validity/usefulness. If the evidence is both *valid* and *relevant* the information can be transferred to patients.
   a A single study may be sufficient to warrant a change in approach but the full circumstances have to be considered. The number of cases, the specific construction of the trial and the statistical tests all provide a basis for reliability.
   b Experimental studies are not always applicable in practice circumstances.
4 To implement useful/applicable findings in clinical practice to the benefit of the patient.

Frustrated clients often turn in desperation to completely irrational approaches such as homeopathy that are touted as solutions to all problems. As an example, homeopathic treatment has no scientific backing or justification – no clinical benefit beyond the placebo effect has been shown to accrue in any species, in any disease condition and under any circumstances. Although it cannot do anything harmful, neither can it do anything helpful. Dilution of homeopathic remedies in water has not been critically compared to the current gold standards for any known disease and is not an accepted tested medical principle. The reality is that the willing acceptance of a useless 'therapy' is not a choice that the animal can take for itself; it requires a properly trained, thinking, scientific and caring veterinarian – even the owner is not able to assume clinical responsibility for the animal but can of course do so for him/herself. Failure to provide proper evidence-based treatment is in itself often harmful – the disease may progress to the point of being untreatable by any conventional means or at least results in continuation of the suffering. Therefore, using evidence-based medicine, homeopathy cannot be justified under any conditions, i.e. it does harm by omission rather than commission. The fact that the homeopathic philosophy has an explanation for every possible outcome is something that inherently generates deep scepticism – such a 'treatment' cannot then be assessed using any recognized clinical testing method and so comes to rely on the perception of the owner alone; the animal has no ability to 'report'. The only way to counter the 'irrational' approaches of homeopathy, 'faith healers' and other charlatans is to provide the best possible evidence-based medicine and the only way we can do that is by open scientific discussion. Likewise, the way the alternative 'professions' can gain credibility is to allow themselves to be subjected to proper clinical audit.

Evidence-based medicine creates confidence in the veterinary profession in general, and the individual practitioner who can support his/her clinical approaches on the basis of the results of large numbers of audited cases involving the correct selection of treatment/procedure. It also provides a solid legal defence for any clinical approaches that have been taken. It does not limit an individual's right to perform any particular treatment or to attempt to expand our knowledge through scientific study – it simply forces 'unusual or new' procedures to be explored and tested against the standard methods in a proper scientific way that imparts clinical confidence.

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3

Diagnostic/investigative procedures

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A careful review of the differential diagnosis list will direct the clinician to the most appropriate further diagnostic aids or tests which can be used either to confirm suspicions of a diagnosis or to eliminate ('rule out') specific disorders. The use of further tests as a 'general scanning' method is not really useful and should be avoided. The clinician should recognize that the further test is being performed for a specific reason and that the findings will clarify the chosen treatment or will alter the treatment depending on the findings. If the proposed treatment is not dependent on the findings then it is probably hard to justify any further tests at all.

Note

If the treatment is not going to alter whatever the results of further tests then the test should probably not be performed – at best it will be a waste of time and money!

Basic tests, which can be carried out in a simple practice laboratory using a microscope, basic equipment and simple stains, can often provide useful diagnostic information in uncomplicated disorders. A positive diagnosis from a practice laboratory can be very rewarding, but a false (negative or positive) result may be embarrassing. Unless handling and processing of specimens is performed regularly, it may be wiser to send specimens to a recognized veterinary laboratory for assistance with diagnosis. Most commercial and university laboratories are well equipped and staffed with trained pathologists and technicians. If all the relevant historical and clinical information is supplied, they can be a powerful ally in establishing a diagnosis in many cases or in establishing 'rule outs', and particularly so in complex or unusual conditions. However, as there are few specialist equine pathologists and even fewer equine dermatohistopathologists, some difficulties may be experienced in obtaining a correct interpretation of the specimens. Discussion with an experienced pathologist is, however, invariably helpful and he or she may have access to colleagues who might also be willing to assist. The use of human dermatopathologists should be considered only in the last resort. They are usually very keen to help and are often highly skilled but are seldom familiar with horse conditions and, unless they are in active contact with colleagues who can assist them with diagnoses, results can sometimes be very misleading and occasionally embarrassing.

Note

Consultation and close cooperation with a pathologist is a highly rewarding process in many cases – it provides discussion and understanding of the problems each party faces in establishing a useful diagnosis.

The equipment required to collect specimens for diagnostic purposes in shown in Table 3.1. This list is really very basic and there is no real justification for collection of inadequate samples. The return from the pathologists is proportional to the care with which samples are collected and handled.

A basic practice laboratory should have the equipment listed in Table 3.2. Sophisticated or expensive equipment is not usually necessary in practice, and for procedures that require the more advanced expertise of a pathologist the specimen should be sent directly to that specialist. Without the experience or skill required to perform sophisticated tests, possession of sophisticated apparatus is often wasteful

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Table 3.1 Equipment that can be used to collect dermatological samples from the skin

- Clippers (preferably electric rechargeable type) with disinfectant spray lubricant
- Magnifying lens, preferably with incorporated illumination (an otoscope is useful in providing both light and magnification in a convenient form)
- 'Denture' toothbrush or soft nailbrush
- Petri dishes or collection pots for collection of specimens
- 'Flea comb'
- Clear adhesive tape
- Scalpel blades
- Biopsy punches (6/8 mm)
- Suture material/skin staples
- Sterile cotton swabs
- Small spoon (cup) curette
- Scissors
- Artery clamps (haemostats)
- Microscope slides (with frosted ends) and pencil for marking
- Bacterial swabs (with transport medium))
- Sterile specimen bottles
- Bottles containing 10% formal saline (and/or other fixatives)
- Histological specimen baskets with appropriate fixatives
- Slides and cover slips
- Surgical scrub solution and sterile saline
- Camera (with high resolution (fine) capability)

Table 3.2 Basic equipment for a simple practice laboratory

- Hand magnifying lens (possibly with a built-in light source; a normal otoscope provides both a light source and magnification)
- Dark ceramic tile
- Simple (preferably binocular) optical microscope with integral light source and ×4, ×10, ×20, ×40 and ×100 objectives
- Stereo dissecting microscope with zoom lens and appropriate light source (this is a very useful instrument)
- Pipettes (preferably disposable, plastic)
- Potassium hydroxide (KOH) solution (10%)
- Stains suitable for the range of tests routinely performed in the laboratory (including Gram's, Ziehl–Neelsen, methylene blue, Wright–Giemsa, Giemsa, Leishman's, lactophenol–cotton blue, etc.
- Spatula, forceps and platinum loop (for bacteriology)
- Gas or spirit Bunsen burner
- Bacteriological plates, incubator (and suitable stains) if cultures are to be performed 'in-house'
- Camera

and frustrating. There is little merit in sending prepared samples to a pathologist (such as mounted and stained sections) unless a second opinion is being sought – indeed the pathologist may not then be able to establish the true orientation of the specimen and might be unable to explore other stains and other orientations of the sample. When a pathologist receives a fixed specimen with a full history and a good clinical description of the clinical case (possibly even with photographic images of the sampled lesions) he/she will usually cut the specimen into the most appropriate 'blocks' for processing and in that way the best possible diagnostic information can be gleaned. Many of the problems encountered in diagnosis are the result of inadequate information, inappropriate specimen selection and inappropriate interpretation of the results of poorly performed analysis.

Note

Some culture media are simple and commercially prepared kits are helpful. They require little technical skill or knowledge (e.g. dermatophyte test medium (DTM) or Fungassay fungal culture medium), but rely on careful reading of the instructions.

Although it is tempting to take every conceivable sample from every case, this approach cannot be justified; often it also confuses the issue. A sensible selection of tests should be made which is economical, relevant and most likely to lead to a diagnosis. Some general principles can be applied. For example, biopsy of suspected bacterial or fungal skin diseases should be taken from truly representative early lesions. Secondary bacterial contamination is common in horses. Similarly, sampling very old, traumatized, open or healing lesions may not reveal the causative organism in cases of dermatophytosis (ringworm). Notwithstanding the need to select carefully, however, groomings, hair samples (and even though they are very marginal in value possibly skin scrapings also) should probably be taken from all horses with pruritic skin disease.

Parasite recognition

The parasites that affect horses are usually fairly easy to recognize. Some are pathogens while others are opportunist pathogens (Table 3.3).

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Table 3.3 Parasites that can be collected from the skin surface, their predilection sites and relative ease of detection

	Parasite	Predilection sites	Visibility
Lice	*Werneckiella* (*Damalinia*) *equi* *Haematopinus asini*	Lateral neck/dorsum	Naked eye (lice and eggs)
Mites	*Chorioptes equi*	Distal limb (feathered horses worse affected)	Microscope/magnifying glass
	Psoroptes cuniculi/*equi*	Tail/head	
	Demodex equi	Eyelid/face	
	Sarcoptes scabiei	Any site (body/head/neck mainly)	
	Dermanyssus gallinae	Face/distal limb/body	
	Trombicula autumnalis *Acarus* spp. (forage mites)		
Ticks	*Ixodes* spp. *Ripicephalus* spp. *Dermacentor* spp.	Perineal skin/ears/body/distal limb	Naked eye (otoscope required for ear examination)
	Otobius megnini	External ear	
	Amblyomma spp.	Perianal skin	
Insects	*Culicoides*/Mosquitoes	Tail base/neck/head/ventrum	Naked eye Traps and nets
	Simulium spp.	Internal pinna/ventral midline/prepuce	
	Gasterophilus spp.	Eggs on hairs of legs/face and neck Body/neck/face	
	Stomoxys spp./*Tabanus* spp.	Body	
	Hippobosca	Perianal skin/perineum	
	Fly larvae	Wound sites	Naked eye
Helminths	*Strongyloides westeri* (larvae) *Oxyuris equi* (eggs) *Habronema musca*	Limbs (foals) Perianal skin Facial skin/wounds/urethral fossa, conjunctiva	Naked eye Microscope (×10)

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ARTHROPODS THAT CAUSE EQUINE DERMATOSES

John W. McGarry

Ectoparasites affect horses throughout the world and include mites and ticks, the biting flies and sweat flies, fly larvae that invade the skin and lice that chew skin or suck blood. These arthropod groups possess different types of feeding mouthparts and have evolved diverse life cycles, being associated permanently or temporarily with their host.

Besides causing direct cutaneous damage, some arthropods, most notably the ticks and biting flies, are vectors of important disease pathogens. Furthermore, ectoparasite feeding activity can be highly irritating and cause disrupted feeding and behavioural difficulties.

Some species of surface-feeding and biting flies also transmit nematodes that can cause skin or eye disease.

Mites

Mites and ticks are arachnids (class Arachnida). Arachnids possess eight legs in the adult and nymphal stages but only six in the larval stages. In arachnids the head and thorax are indistinct and fused and the legs protrude from the body margin. The insects (class Insecta) such as flies and lice have an obvious head, thorax and abdomen and they always have six legs emerging from the thorax region.

The majority of mites are ubiquitous and live freely in very diverse habitats. The relatively small number that have evolved to become permanent skin ectoparasites are responsible for significant skin damage in domestic animals.

Some mite species are temporary ectoparasites, visiting animals to suck blood for relatively short periods. Parasitic mites irritate the skin by feeding on skin scales, lymph exudates, sebaceous secretions or blood. Those known as the 'burrowing' mites tunnel into the skin (e.g. *Sarcoptes scabiei*) or invade hair follicles and sebaceous glands (*Demodex* spp.)

Mites that feed on the skin surface are known as 'non-burrowing mites' and include *Chorioptes equi* and *Psoroptes ovis*. The mouthparts of *P. ovis* can pierce the skin and mites then feed on serous exudates. Both burrowing and non-burrowing mites cause dermatitis which can progress to a severe skin condition called 'mange'. Blood-sucking mites such as *Neotrombicula autumnalis* are temporary ectoparasites and spend most of their life cycle in the environment. Some are casual or opportunistic equine parasites such as *Dermanyssus gallinae*, the red poultry mite, which is a blood-feeding mite. Again these spend more time off the equine host than on it. The implications from this are that they may be difficult to find and therefore diagnose; the clinical signs may be present without the parasite.

Identification of mites can be complicated due to their diverse morphologies and the existence of numerous free-living types but a useful character seen in the species that cause mange is the presence of a 'thumb print'-like pattern on the dorsal cuticle.

Sarcoptes scabiei

This is a borrowing mite and the single species in the genus. There are a large number of host-adapted forms worldwide which affect domestic and wild animals. Mites are transmitted during physical contact.

Identification

S. scabiei are round and flattened (Fig. 3.1A) with a rounded head region and short front legs. The first pairs of legs have tiny claws and end with a stalk (the pretarsus) bearing a sucker. The two pairs of posterior legs, which bear long hairs (setae), do not extend beyond the body margin; the dorsal surface bears cuticular folds giving rise to a 'thumb print' pattern and is covered with scales and strong spines recognizable in histological sections (Fig. 3.1B).

Life cycle

The entire life cycle takes place on the host. Following mating the female mite tunnels into the sub-corneal layer of the skin. Each tunnel (1.0–2.0 cm in length and just below the skin) contains a single female mite, her eggs and excreted material. The female mite lays around three eggs per day and eggs hatch to six-legged larvae within the tunnels after 3–4 days. The larvae exit the tunnels and successive moulting to eight-legged protonymph, tritonymph and female (600 μm) and male adult (300 μm) stages occurs. Newly moulted adult females mate immediately and create new tunnels by eating skin and tissue fluids. The total egg-to-adult life cycle can be as little as 11 days but up to 3 weeks is usual.



Figure 3.1 **(A)** The typical appearance of *S. scabiei*, showing characteristic setae on the back legs, the pedicle with a small sucker on the front leg, and scales on the dorsal cuticle. **(B)** The histological appearance can be recognized with the parasites located in dermo-epidermal tunnels.

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Significance

Sarcoptic mange is a rare but severe condition in horses, largely confined to immunocompromised animals. The disease was historically a very significant cause of epidemic debilitating equine disease and was reportable. Nowadays it is singularly rare and more often associated with immunocompromised horses in contact with other affected animal species.

Burrowing and feeding activity causes intense itching and scratching. Pruritic papular lesions result. These rupture and cause hair loss and crusting.

Dermal pathology can extend over the entire body but the most severe forms of the condition are more often seen on the head and neck (see p. 195).

Psoroptes equi

Psoroptes equi is a non-burrowing mite. There is a large number of host-adapted strains in different parts of the world which vary in pathogenicity but recent genotyping has suggested the existence of distinct species. Two of these are associated with horses – *P. equi* and *P. natalensis*. The latter is thought to have originated in South Africa but is now present in Europe. Infestation with *P. equi* in horses is reported from Europe, Australia and North America.

Mites are transferred during physical contact but off-the-host survival may also be a significant route of transmission, particularly in winter.

Identification

The body of *P. equi* is oval-shaped and the head region is pointed (Fig. 3.2). All stages of this mite are identified by the presence, on the first two pairs of legs, of a three-segmented stalk which ends with a trumpet-shaped sucker. The males are identified by the presence of copulatory suckers in addition to the above features, which are specific to the genus *Psoroptes*. These characters enable differential identification from *Chorioptes equi*, a more common mite of equines, and from incidental forage mites, some of which have a broadly similar morphology.



Figure 3.2 *Psoroptes* spp. mite showing the three-segmented stalk and sucker on the first two pairs of legs; the male has copulatory suckers. Females tend to be smaller than the males and eggs may be seen in the abdomen.

Life cycle

Eggs are relatively large (250μm) and are produced at about two to three per day. Successive growth and moults occur: eggs, larvae, protonymph, tritonymphs and adults. Adults are often seen in mating pairs in skin scrapings and old moulted cuticles are often abundant. Adults measure approximately 750μm (females) and 450μm (males) and are therefore visible to the naked eye when viewed against a dark background. The population may double every 7–8 days.

Significance

Psoroptes equi infestations in the horse occur in regions of thick hair such as the tail and mane but also on the face, ears and other parts. A fluid exudate, which dries leaving crusts, is produced. Thickening and hair loss can occur and is particularly noticeable along the margins of the pinnae. In severe infestations large numbers of active mites can be recovered from the edge of lesions. Psoroptic mange may result in emaciation, head shaking and pruritus (see p. 193).

Chorioptes equi

Chorioptes bovis is the only species of *Chorioptes* in temperate climates but there are host-adapted strains; other species probably occur worldwide. The name *Chorioptes equi* is used to describe chorioptid mites on equines. Chorioptic mange results from chewing (rather than skin piercing) activity and is considerably less severe than psoroptic or sarcoptic mange but is far more common. Like psoroptic mange, this condition tends to be a winter disease and feathered horses are more liable to the condition.

Identification

Females (Fig. 3.3) (300–400μm) and males (300μm) are significantly smaller than *P. equi* and have a rounded head region. *Chorioptes* do not have the jointed stalk of *Psoroptes* and the ending is cup-shaped as opposed to trumpet-shaped. There are long whip-like setae on the reduced third pair of legs in the female. In the male, the two broad posterior lobes are square-shaped and bear numerous long hairs. In heavy infestations all stages of the life cycle including eggs are readily encountered and adult *C. equi* are often seen as mating pairs.

Life cycle

The life cycle of *C. equi* is similar to that of *P. ovis*. Egg, larval, two nymphal and adult stages occur. The period from egg to adult takes approximately 3 weeks. Mites may survive off the host and transmission may occur via the bedding as well as by direct contact.

Significance

The mites prefer the lower parts of the limb and sometimes the head and base of the tail. Horses with feathered legs are frequently affected. Erythema, crusting, ulceration and alopecia with itching is common. Infected horses frequently stamp their legs (see p. 191).

Demodex spp. (follicle mites)

Clinical disease due to *Demodex* spp. is extremely rare in horses and the parasite is usually regarded as a commensal

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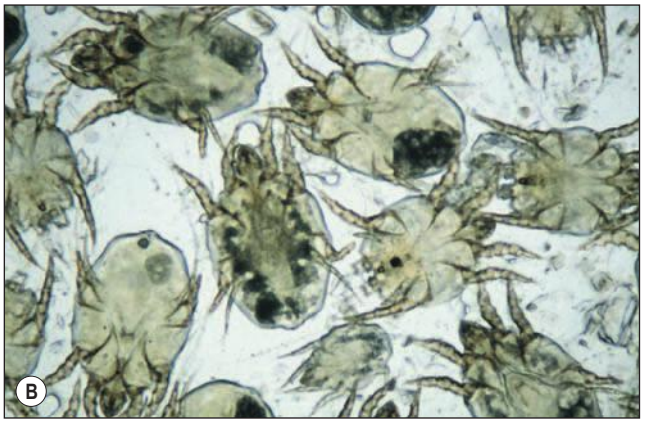


Figure 3.3 **(A)** *Chorioptes equi* are usually easy to recognize by their physical appearance (though very similar to *Psoroptes*). **(B)** There are invariably many very active parasites to be seen. It is important to ensure that other parasitic mites are not confused with non-parasitic forage mites, which may also be present in the skin brushings. (Figure courtesy of Dr Marianne Sloet.)

and facultative pathogen. Two species of host-specific *Demodex* affect horses; *D. equi* is found on the body and the larger species, *D. caballi*, infests the eyelids and muzzle.

Identification

Demodex spp. mites are minute (100μm) with a tapered and pointed abdomen, the cuticle of which is striated. The stumpy legs, which end in claws, and the cigar-shaped bodies are recognizable in stained skin sections (Fig. 3.4).

Life cycle

Demodex spp. usually exist as harmless commensals in the hair follicles and the sebaceous glands of the skin. The life cycle, which takes 18–24 days to complete, consists of eggs, two larval stages (a protonymph and tritonymph), and the adult stages. Males are rarely seen but all the life cycle stages may be present in a single hair follicle. *Demodex* spp. are unable to survive away from the host's body and so transmission is very slow; it is suggested that infection is acquired by foals at or around birth but little is known about the disease in horses.



Figure 3.4 Typical *Demodex* mites obtained from skin scrapings (see p. 63); they can sometimes be found in a histological section of normal skin.

Significance

Although *Demodex* spp. are usually regarded as non-pathogenic, significant clinical disease can result if the health and immune status of the animal is compromised. Most cases are reported to be in horses receiving long-term glucocorticoid therapy. Infestation may present as scaling and alopecia; papules and pustules may form on the face, shoulders, neck and limbs (see p. 196).

Trombicula spp. (harvest bugs, berry bugs, chiggers)

The species that infest horses and companion animals are *Trombicula* (*Neotrombicula*) *autumnalis*, in Europe, *Trombicula* (*Eutrombicula*) *alfredduges* and *Trombicula* (*Eutrombicula*) *splendens* in the New World, and *Trombicula* (*Eutrombicula*) *sarcina* in Australasia. Only the larval stage is parasitic. These mites are not host specific and will feed from available hosts including horses and humans. They are prevalent in areas with chalky soils and are seasonal, showing most activity during late summer and autumn.

Identification

Trombiculids (Fig. 3.5) are very small (200μm) and are sometimes noticed in groups as pale orange-brown specks on the skin. Close examination will reveal six-legged larvae having a scutum and body covered with very small feathered hairs.

Life cycle

The adults and nymphs are non-parasitic and predate upon the eggs and larvae of various types of soft-bodied insects. Eggs are laid in well-drained soil and newly emerged larvae quest, in a similar manner to ticks, on the tops of grass stems awaiting a passing host. The mouthparts pierce the skin, producing an exudate on which they feed for several days. In northern Europe, there is one generation of

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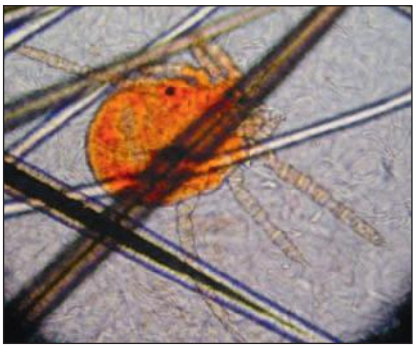


Figure 3.5 *Trombicula* spp. larvae are small, orange in colour and can be seen on the skin. Only the larvae attack mammalian hosts, where they can cause a hypersensitivity response (see p. 198).

larvae each year which are very active in late- to mid-autumn. Horses are parasitized through contact with grasses when feeding and during exercise.

Significance

Infestation, usually on the limbs and face, causes pruritus with erythema, and sometimes a severe exudative dermatitis out of proportion to the severity of the infestation. Hypersensitivity may result with pustules, scabbing and hair loss (see p. 198).

Forage mites

These comprise the many free-living species associated with damp straw and hay. They usually feed on microorganisms and organic matter. Populations can reach very high levels in a matter of weeks when environmental conditions are suitable and they can be a significant cause of dermatitis. There are many species but the commonest forage mite species is probably *Acarus siro* (*Tyroglyphus farinae*).

Identification

These soft-bodied mites are oval-shaped and typically possess numerous and very long whip-like hairs on the posterior body margin (Fig. 3.6). Teeth are usually prominent on the palps. These features are important in distinguishing forage mites from the mites that cause mange.

Significance

When in contact with horse skin, mites may cause a hypersensitivity-related dermatitis or even abrade the skin to cause a temporary erythema. There may also be pruritus, papules and crusts. The most obvious lesions occur close to the hay or straw that harbour the mites, i.e. the face and distal limbs (see p. 198).

Dermanyssus gallinae (poultry red mite)

Dermanyssus gallinae is a temporary blood-feeding mite of domestic and wild birds. In the absence of avian hosts, however, this species is able to feed on many types of animal, including humans and horses. Stables located near poultry houses or birds' roosts may be a source of hungry poultry, especially if bird hosts are absent. *Dermanyssus gallinae* is a nocturnal ectoparasite, spending the daylight hours hidden in crevices and cracks in stable walls and wooden beams.

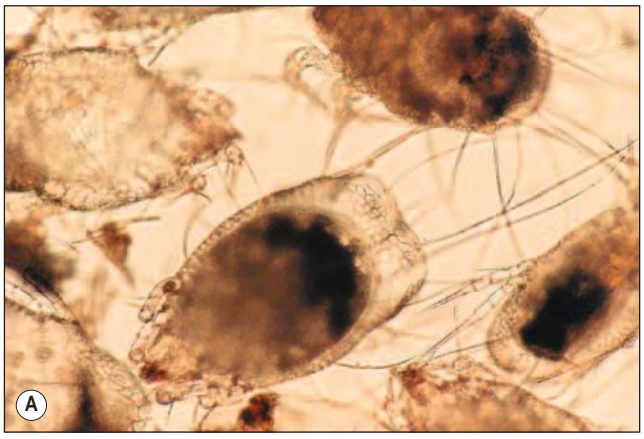




Figure 3.6 A variety of mites occur in bedding and forage and on pasture. **(A)** These parasites are *Acarus siro* and are common in hay and straw. **(B)** This mite is a typical bedding-related mite. These are sometimes significant causes of limb and face dermatitis and irritation.

Problems caused by red mites are likely to be transient; *D. gallinae* populations do not establish in horse accommodation and require continued poultry contact.

Identification

Adults are relatively large – up to 1 mm in length. The body cuticle is tougher than that of the soft-bodied permanent species such as *Psoroptes* spp. and the legs are spider-like legs that protrude well beyond the body margin (Fig. 3.7). A diagnostic feature that separates this mite from the similar types (below) is the anal shield, which is large and D-shaped **(Fig. CD3 • 1)**; this feature is only seen if the specimen is cleared (see section examination for mange mites of horses). Poultry mites are fairly easy to see with the naked eye or with a magnifying glass, appearing bright red when fully blood-engorged and almost black when the blood meal is more than 24 hours old. Pigeons' nests within or near stables may be a transient source of *D. gallinae* in autumn when young birds are fledged.

CD

Life cycle

Mites are fully fed within a few hours of arriving on their host. They then drop off quickly. Eggs are laid near nests and roosts. Newly hatched larvae do not feed but undergo successive moults to the nymph and adult stages in as little as 7–10 days. Populations can build up rapidly in bird accommodation.

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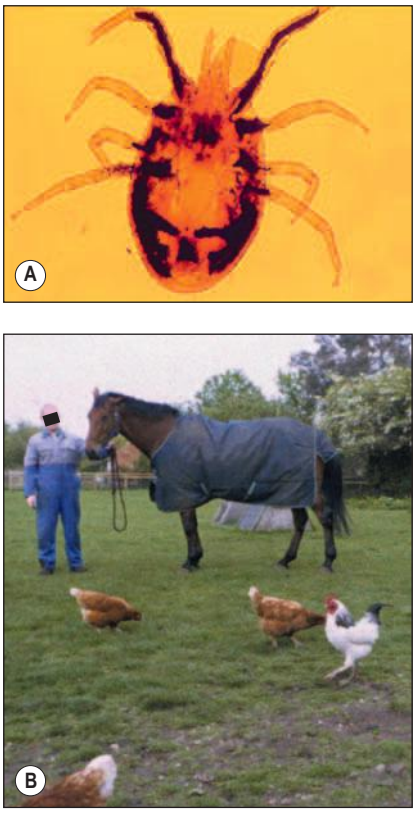


Figure 3.7 (A) *Dermanyssus gallinae* found on the distal limb of a horse showing facial and limb pruritus. **(B)** The horse was stabled in contact with domestic poultry.

D. gallinae populations can survive for up to 35 weeks in the absence of their usual avian hosts; accommodation can therefore remain infested for long periods. Horses (and humans) are likely to be bitten by dispersing hungry mites.

Significance

There may be pruritus, papules and crusts. Diagnosis is difficult because mites are seldom recovered from animals (see p. 199).

Other opportunistic blood-sucking mites

These include *Laelaps* spp. (e.g. the spiny rat mite), *Eulaelaps stabularis*, *Haemogamasus pontiger*, *Allodermanyssus sanguineus* (the house mouse mite) and *Ornithonyssus bacoti* (the tropical rat mite). These species have a similar morphology, life cycle and behaviour to *Dermanyssus gallinae* but are normally associated with rodents rather than poultry.

Identification

The legs are long and the body shape very similar to that of *D. gallinae*. The anal plate is pear-shaped and not D-shaped as in *D. gallinae*. The bodies of these species are densely covered with short hairs **(Fig. CD3 • 2)**.

CD

Life cycle

These species are usually associated with rodents and their nests but they will feed on the blood of other animals. They may become problematic if rodent populations near stables are high.

Significance

There may be papules and crusts with pruritus. Diagnosis is difficult because mites are seldom recovered from animals. The implications of finding them of course is that rodent populations are high and control should be instituted.

Examination for mange mites of horses

Examination of tissue may reveal all the living life cycle stages, including eggs, larvae, nymph and adult stages and moulted cuticles. Examination of old lesions may reveal dead and degenerated ectoparasites or parts of these.

Where infestation with non-burrowing mites (e.g. *Psoroptes* and *Chorioptes*) is suspected, repeat surface samples should be obtained using a strong comb or preferably a stiff brush (see p. 60). Deeper skin scrapings using a scalpel blade are necessary to recover burrowing mites such as *Sarcoptes* and *Demodex*.

Brushings and scrapings can be examined dry, under a stereoscopic (dissecting) microscope. Mites are best seen against a dark background, particularly when particles of debris adhere to the mite's body hairs. Movement is usually very easy to observe.

In severe mange, often where scab material is present, the extracted tissue can be macerated in 10% potassium hydroxide for 10 minutes at 37°C. The resulting fluid is placed on a microscope slide with a cover slip and examined under low power (×10). Higher magnification is needed to see *Demodex* mites.

Semi-permanent preparations of burrowing and non-burrowing mites, which are soft bodied, may be made by placing them directly into lactophenol or Berlese fluid on a microscope slide with a cover slip. To make preparations of mites such as *Dermanyssus gallinae*, which have tough cuticles like those of fleas and may have fed, clearing is necessary by incubation in 10% potassium hydroxide for 1 hour followed by washing and dehydration in a series of alcohols. The specimen is then cleared in xylene and mounted on a glass slide using a resinous mountant (DPX or similar).

Ticks

Hard ticks (family Ixodidae) and soft ticks (family Argasidae) are blood-feeding ectoparasites of animals and birds found throughout the world. Hard and soft ticks of animals are classified in nine major genera found in temperate, topical and subtropical parts of the world (see p. 212). These two groups exhibit different feeding behaviour, life cycles and morphologies. Ticks cause serious mechanical damage, irritation, and inflammation to the skin. Infestations can also induce hypersensitivity.

Hard ticks are important as vectors of livestock diseases such as babesiosis. They possess a tough dorsal covering, the scutum.

All stages of ticks (both hard and soft) possess a characteristic hypostome, a toothed structure which anchors the tick into the skin. No other arthropod groups, e.g. mites, possess this feature. Feeding hard ticks tend to aggregate and remain attached to the host for several days, usually provoking a significant skin reaction. Deeper lesions are caused by species in the hard tick genera *Amblyomma* (Africa, North and South

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America, Asia) and *Hyalomma* (Asia and Africa) because they possess very long mouthparts.

Lesions caused by hard ticks may also predispose to 'screw-worm' myiasis. Other ixodid genera that might be found on horses worldwide include *Boophilus* spp. (Africa, Asia, South America, Australasia); *Ixodes* spp. (Palaearctic, USA, Australia and Australasia); *Dermacentor* spp. (Palaearctic, North America); *Haemaphysalis* spp. (Europe, Asia, Africa and Australia), *Rhipicephalus* spp. (mainly Africa).

Soft ticks (family Argasidae) are more common in drier regions and are of generally lesser veterinary importance. They have a leathery, folded and pitted dorsal surface. Adult soft ticks feed frequently and within a matter of minutes. They may also feed on horses, e.g. *Otobius megnini*, the spinose ear tick, found in western and south-western North America and Canada. Another soft tick genus, *Ornithodorus*, may also attack horses.

Identification

There are no distinct body regions. The head and thorax are fused to form the basis capituli. Examination of the structure and shape of the mouthparts can confirm the specimen as a tick and is also useful in determining the genus.

Some tick genera have a metallic, shiny scutum whereas others do not. Species of ticks in subtropical/tropical genera *Ambylomma* and *Hyalomma* are often very large – up to 1.0 cm in size even when flat and unengorged – and can be easily identified by their overall size and very long mouthparts. The scutum of *Amblyomma* spp. has a metallic appearance; that of *Hyalomma* spp. is dull and non-metallic. Both these genera are usually found in the perianal region and the underside of animals and may appear like bunches of grey grapes, 1–1.5 cm in size when full of blood. *Ixodes* spp. are non-metallic, dark red, and have long mouthparts; *Ixodes* spp. occur in temperate climates.

Boophilus spp. are the smallest of all the tick genera. They are non-metallic bluish grey in colour and possess much reduced mouthparts. *Dermacentor* spp. are a metallic-coloured species, the mouthparts of which are club-shaped. *Haemaphysalis* spp. are non-metallic and have laterally expanded palps. *Rhipicephalus* spp. are non-metallic, reddish in colour with a characteristic hexagonal shape to the basis capituli. *Rhipicephalus* spp. may be found inside the ears.

Soft ticks such as *Otobius* and *Ornithodorus* spp. appear crab-like and have a leathery, textured cuticle. The mouthparts are recessed under the tick and cannot be seen from above.

Life cycles

Hard and soft ticks comprise the parasitic stages of larva, nymph and adult. Soft ticks have a multi-host developmental cycle. Each stage of the soft tick life cycle feeds several times and for only a few minutes on each occasion. Hard ticks are far more important and their life cycle is now described in detail. Adult hard ticks spend the first 24 hours on the host without imbibing blood and at this point females are more or less flat and 5–10 mm in size. Females attach to the host, attract males and become fully engorged with blood after 6–12 days. Engorged adult ticks may reach 1 cm in size (Fig. 3.8); they then drop off the host and produce thousands of eggs in vegetation.

The resulting hatched larvae are very small (less than 1.0–1.5 mm in size), and become only slightly larger when blood-feeding is completed within 2–5 days. Engorged larvae drop off the host and moult to nymphs. Nymphs are 1–3 mm in their unfed state but swell to 3–6 mm following feeding. Nymphs are fully blood-engorged after 3–8 days and then drop off to moult to the adult stage.



Figure 3.8 An engorged ixodid tick.

In this way, most hard tick species parasitize three different animal hosts in order to complete their life cycle (three-host ticks), which may be as short as a few months in the subtropics or as long as 3 years in temperate climates.

In contrast, *Boophilus* spp. are 'one-host' ticks, feeding and moulting on the same host. The larva remain on the host, mature and drop off the host as an engorged adult.

Some species of *Hyalomma* and *Rhipicephalus* spp. are two-host ticks.

Significance

Hard ticks tend to feed at specific sites, especially the ears, face, neck, axillae, groin, distal limbs and tail, but although the different species have preferential feeding sites, ticks may be found anywhere on the body. Due to the time spent on the host, one-host and two-host species are likely to induce a more severe host reaction than three-host species. The cutaneous signs associated with hard tick feeding in horses include focal necrosis and a variable (mild–severe) inflammatory response. Papules, pustules and ulcerations occur. The larvae of *Boophilus microplus* are reported to cause a local hypersensitivity reaction with papule formation at the site of feeding. Tick feeding can cause abscesses by the introduction of surface bacteria. There may be subsequent infection with *Staphylococcus*. The lesions created by ticks may be attractive to screw-worm (fly) larvae in the tropics and subtropics (see p. 212).

The larvae and nymphs of the soft tick *Otobius megnini* can produce a severe otitis externa.

Flies

Flies are insects in the order Diptera. Insects differ from arachnids (the mites and ticks) in possessing a distinct head with compound eyes, a thorax which bears the legs, and an abdomen. There are a single pair of wings and three pairs of legs.

All species of Diptera have a complex life cycle with a complete metamorphosis: larvae or 'grubs' hatch from eggs which are laid on various types of organic substrate and increase in

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Figure 3.9 Flies cause worry/nuisance and are a major cause of skin pathology. Many species are involved and it is almost impossible to list all those responsible for some pathology. **(A)** Muscid flies (*Hydrotea irritans*) congregating around the eye. The horse will usually be unconcerned. **(B)** A wide variety of flies, including *Lucilia* spp. (green), attacking a purulent and exudative wound on the eyelid.



Figure 3.10 Fly worry. A small verrucose sarcoid on the lateral sheath region was tormented by flies (*Musca* spp.) and made far worse.

size by feeding and moulting through three instars. Feeding flies can cause severe irritation, dermatitis and hypersensitivity. (Eosinophilic) collagenolytic granuloma within the deep dermis may be associated with blood-sucking flies.

Surface-feeding flies ('nuisance' or 'sweat' flies) are secretophagous, i.e. they feed around the eyes and face or any other parts of the body that produce secretions. The blood-sucking types are known as 'biting flies' and possess mouthparts that penetrate or lacerate the skin. Several important surface-feeding and biting fly species that occur throughout the world can be found in the family Muscidae. The biting flies include other important types – the black-flies (family Simulidae), midges and sandflies (Ceratopogonidae) and mosquitoes (Culicidae). The biting flies also include the large and aggressive 'horse flies' (Tabanidae) with mouthparts that slash rather than pierce the skin to feed on blood.

Flies are a persistent problem for many reasons. Some simply cause worry, others transmit serious diseases and others cause direct pathology by either tissue destruction or debilitating parasitism (Figs 3.9 and 3.10).

Biting flies

Family Muscidae – *Stomoxys calcitrans*, the 'stable fly' or 'biting house fly'; *S. niger*; *S. sitiens*

Stomoxys calcitrans is the most important species found worldwide. *S. niger* and *S. sitiens* are common species in Africa and east Asia, respectively. Stable flies are known to transmit equine infectious anaemia and 'surra' (a form of trypanosomiasis). They (and the non-biting *Musca* spp.) are vectors of the stomach worm *Habronema* spp. and the eye worm *Thelazia* spp. in horses, the larvae of which cause cutaneous lesions. (see p. 217).



Figure 3.11 *Stomoxys calcitrans*, the stable fly.

Identification

Stomoxys spp. are 5–7 mm, brown/grey and house-fly-like in appearance (Fig. 3.11). The abdomen is chequered and the thorax striped. Males and females are similar and possess a typical strong proboscis which is black, shiny and forward pointing. The palps are short and thin and very small compared to the length of the proboscis.

Life cycle

Both males and females are active throughout the day; individual female flies take several blood meals in order to mature their eggs and this interrupted feeding pattern is an important aspect of disease dissemination.

Stomoxys spp. breed in sheltered animal manure mixed with straw and urine but other varieties of rotting matter provide suitable breeding substrates, provided it is protected from rain. Larvae feed and moult from the first instar to the second and then to the third instar. Larval development may take as little as 7 days. The third-stage larva pupate and adult development within this is temperature-dependent. *Stomoxys* spp. overwinter as puparia in temperate regions but breed all year round in warm climates. In northern Europe, population levels peak in August/September and

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Figure 3.12 *Lyperosia irritans*. Note that flies are very small, being around 4–5 mm long. The wings are set very flat.

flies can be seen pestering housed and grazing horses. They can be recognized at rest 'sunning' themselves on exposed walls, farm machinery and beams.

Significance

Stable flies inflict a painful bite and are one of the most annoying pests of horses, mostly attacking the lower body and legs. Horses react by stamping the legs, biting and shivering the skin. The presence of cattle and pigs, on which this species also readily feeds, can influence the numbers of *S. calcitrans* pestering horses.

Lyperosia irritans (=*Haematobia irritans*), 'hornflies'; *L. exigua*

L. exigua is found in warmer countries and is a major pest of cattle and horses in the USA. It is also common throughout Europe. The species mainly feed on cattle but will also feed on horses.

Identification

Lyperosia spp. are the smallest of the biting muscids. The palps are the same length as the proboscis. The head is rather small compared to the overall body of the fly and the wings are elongated (Fig. 3.12). Flies may appear embedded in the hair along the backline.

Life cycle

The life cycle of *Lyperosia* spp. is highly adapted to ectoparasitism; the flies congregate to feed and rest on the backs of animals. Flies leave the host to deposit eggs in fresh dung as soon as it is dropped on pasture. The life cycle in dung is similar to that of the other species, so that new populations may emerge every 2–4 weeks, depending on temperatures and conditions. Adult flies do not infest animal accommodation; *Lyperosia irritans* does not bite horses inside stables.

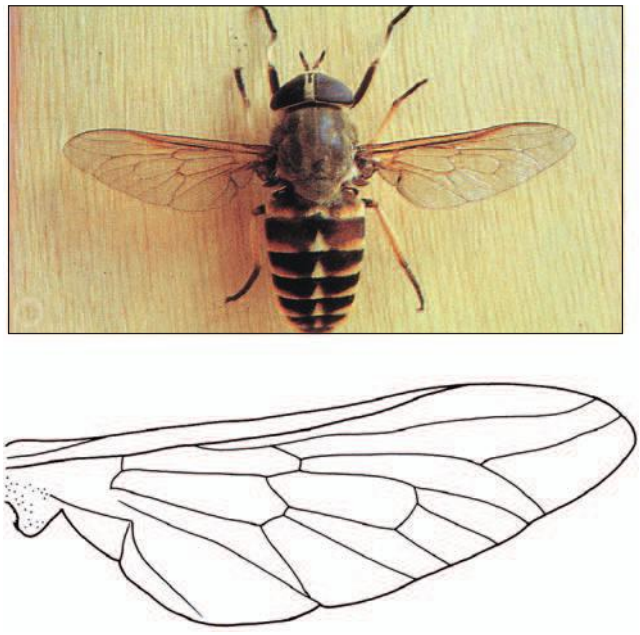


Figure 3.13 *Tabanus* spp. are some of the largest flies and can reach 1.5 cm long. The wing pattern is a good identifying feature.

Significance

The flies can cause irritation. Severe infestations may cause anaemia. Presence of cattle pasture may influence the numbers pestering horses.

Family Tabanidae: *Tabanus* (=*Hybomitra*) spp. (horse flies, thunder flies); *Haematopota* spp. (cleggs); *Chrysops* spp. ('green heads')

These species inflict a painful bite as the mouthparts are specialized and adapted for slashing the skin and releasing blood which is sponged up from the wound.

Identification

Flies in the family Tabanidae are all large and stout-bodied, between 10 and 15 mm in size. *Tabanus* spp. are the largest of the Tabanidae, and measure 1–2 cm (Fig. 3.13). Like all tabanids the eyes are semi-moon-shaped and iridescent, the antennae are three-jointed and the wings are clear. *Haematopota* spp. are smaller **(Fig. CD3 • 3A)** and the wings are mottled. In *Chrysops* spp. **(Fig. CD3 • 3B)**, the wings are usually banded.

CD

Life cycle

The larvae are carnivorous and develop from eggs laid underneath hanging vegetation in or near wet areas, muddy patches or marshes. There is usually only one generation of adult flies annually in temperate climates; emergence starts on warm days in early summer and feeding activity, on warm, humid days, is greatest in July and early August. Tabanids overwinter as late-stage larvae in Europe. Adult emergences overlap in tropical and subtropical zones, but are influenced by wet and dry times of the year. Females of some tabanid species do not require an initial blood meal to produce eggs, a phenomenon known as autogeny.

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Significance

Despite their large size, tabanids fly silently and appear on any part of the horse's body but very often it is the lower legs and the heels which are bitten. *Haematopota* spp. are also attracted to the sides of the face and neck and other parts where the host may have difficulty in dislodging the attacker. Tabanids usually make several attempts to bite and readily move between animals, making them effective vectors of disease pathogens. Feeding results in bleeding which attracts nuisance flies. Horses tolerate tabanids to different extents but may flee from the persistent attention (gadding).

Family Glossinidae: *Glossina* spp. (tsetse flies)

There are 22 species of tsetse flies, found only in sub-Saharan Africa across tropical forests or savannah zones, occurring within 'fly belts' within the overall tsetse fly range. They are aggressive blood-feeders and leave large visible bite sites with oedema and a purple central spot. They are responsible for transmission of trypanosome parasites.

Identification

CD

Glossina spp. are similar in colour to house flies but have a slim body and are larger **(Fig. CD3 • 4)**. They possess a forward-pointing proboscis and hold their wings over the dorsal abdomen in a scissor-blade posture. The wings also have a characteristic hatchet cell not seen in other blood-sucking or nuisance fly species.

Life cycle

Both males and females feed several times during a highly specialized reproductive strategy – a single, ground-dwelling larva is produced during each 21-day cycle. Adults emerge from puparia after 30 days. Several larvae are produced by a single fly.

Significance

Skin lesions and intense annoyance result from feeding behaviour, the extent of which depends on the level of fly challenge, but their principal importance is as vectors of cyclically-transmitted animal trypanosomes.

Family Hippoboscidae: 'keds' or 'forest flies'; *Hippobosca equina*, *H. rufipes*, *H. maculata*

Hippoboscids are blood-sucking flies that superficially resemble very large lice. *Hippobosca equina* mainly bites horses and donkeys and has a worldwide distribution. *H. rufipes* is found mainly in Africa and *H. maculata* throughout the tropics and subtropics.

Identification

Adult flies are approximately 10 mm in length and are pale reddish brown (Fig. 3.14). They have small heads with bulging eyes and the proboscis is forward-pointing. Hippoboscids feed on the host for long periods (see p. 210).

Life cycle

Both sexes feed on blood. Females leave the host and deposit a single larva in soil or leaf litter. A female may produce up to six larvae in her life time. Flies are most abundant in the summer months in temperate zones.



Figure 3.14 *Hippobosca equi*. These parasites are extremely tough – it is almost impossible to kill them by hand! It is ill advised to try to dislodge or kill them by slapping the horse's skin. The horse may resent the hit more than the flies.

Significance

The preferred feeding sites are the perineum and between the hind legs. Flies may be well tolerated by donkeys and horses that are exposed to high, persistent challenge throughout the fly season. This species can be a source of intense irritation in other circumstances, e.g. when horses are moved into infested areas (see p. 210).

Family Culicidae (mosquitoes): *Anopheles* spp., *Aedes* spp., *Culex* spp., *Culiseta* spp.

There are over 3000 mosquito species in two distinct subfamilies – the anophelines, which include *Anopheles* spp., and the culicines, which comprise *Aedes* spp., *Culex* spp., *Culiseta* spp. and others.

Some species transmit disease pathogens, e.g. equine encephalitis, and they contribute to the overall problem of fly attacks. Many mosquitoes species have a wide host range and readily attack grazing horses. Mosquitoes are active at dusk and during the night but some, such as *Aedes* spp., also bite during the day.

Identification

Adult are 3–10 mm long, slender with long legs, threaded antennae and scales on the wings. Mosquitoes have a very long proboscis flanked by palps.

Life cycle

Only females feed on blood. Eggs are laid in batches on still surface water. The larvae and puparia of anophelines require unpolluted fresh water sites whereas culicines breed in stagnant, organic-rich water. The eggs of *Aedes* spp. have adapted to survive desiccation and are laid on moist substrates and not directly on water itself.

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Mosquito larvae and pupae may be easily recognized as they rest and breath just under the water surface and 'loop' away downwards if disturbed. There is a distinct summer activity pattern in temperate zones and woodland with ponds or salt marshes providing breeding sites. In tropical/subtropical zones there may be large increases in fly activity weeks after the seasonal rains.

Significance

Mosquitoes can significantly contribute to fly 'nuisance'. Animals may vary in their ability to tolerate mosquito bites; some individuals may simply show local redness and swelling whereas others may have a severe hypersensitivity to saliva (see p. 284).

Family Ceratopogonidae: *Culicoides* spp. (midges); family Psychodidae: *Plebotomus* spp., *Lutzomyia* spp. (sandflies)

Culicoides spp. tend to swarm in significant numbers and attack most frequently at dawn and dusk. They are a recognized cause of severe allergic dermatitis in susceptible horses and are also important disease vectors. Sandflies are slightly larger (5 mm) and occur in dry areas of the world, feeding at night.

Identification

Culicoides spp. are very small flies (1–4 mm) and may not be noticed flying around or feeding on horses. Many species have mottled wings **(Fig. CD3 • 5)**. Sandflies are small flies with hairy wings which are held erect above the thorax while feeding.

Life cycle

Culicoides spp. breed in any decaying vegetable matter, notoriously in compost heaps. Sandfly breeding sites are leaf litter, humus and associated decaying matter. The life cycle is typically dipteran and can be completed in 3 weeks or less.

Significance

The very common skin disorder insect bite hypersensitivity ('sweet itch' Queensland itch) is frequently caused by hypersensitivity responses to the bites of *Culicoides* (see p. 284). This is a predictable highly pruritic condition in susceptible horses with self-inflicted skin damage through rubbing on posts, etc. The withers, mane, tail, face, ventral midline and other regions are often affected, leading to hair loss, excoriation and crusting which leads to hyperkeratosis and thickening of the skin.

Sandflies may also cause significant dermatitis in horses where this fly is endemic but this is usually transient (although some hair loss may persist) and usually far less severe than insect bite hypersensitivity (see p. 209).

Family Simulidae: *Simulium* spp. (black-flies, buffalo flies)

Flies may feed in huge numbers in certain locations in temperate countries, such as in the northern parts of the USA, Canada and eastern Europe.

Identification

Simulium spp. are small flies (2–4 mm) with a characteristic humped thorax. The wings are clear and the antennae short. Adults are dark in colour but some species are brown rather than black.

Life cycle

Eggs are deposited in gelatinous aggregations on vegetation in slow to fast running waters, in which the larvae and puparia develop. The adults emerge in late summer and bite the underside of horses or inside the ears, depending on the species. Black-flies are active throughout the day.

Significance

Simulium spp. can cause general dermatitis at feeding sites and crusting, particularly inside the ears and in the ventral midline (see p. 210). They may be vectors of the virus that causes pinnal acanthosis (see p. 136). Swarm attacks can be fatal to horses.

Surface-feeding flies ('nuisance flies' or 'sweat flies')

There are several families that contain 'nuisance flies' occurring worldwide. The most important families in temperate zones are the Muscidae and Fannidae.

CD

Musca autumnalis (the face fly) and other *Musca* spp.

CD

Musca autumnalis, the 'face fly', is a very common exophilic species in Europe, central Asia, parts of Africa and North America **(Fig. CD3 • 6)**. *Musca sorbens*, 'the bazaar fly' (Africa, Pacific Islands and Oriental regions, Australia), and *Musca vetustissima*, the Australian bushfly (Australia), are other important species.

Identification

Females of *Musca* spp. have the same gross appearance as *Stomoxys* spp. but the proboscis is of the non-biting type, ending in a pad for mopping up secretions. In *Musca* spp., the two longest wing veins join at the wing margin forming a V-shape. Males also feed and have a yellow abdomen.

Life cycle

The life cycle of *Musca* spp. is very similar to that of *Stomoxys* spp. However, *Musca* spp. breed in animal dung on pasture or in other decaying organic matter. The life cycle may take as little as 14 days in the summer, with up to four generations in one season. Flies overwinter as adults.

Significance

Feeding occurs on the face and muzzle and around the corners of the eye. Eye irritation can lead to kerotoconjunctivitis (fly eye or 'pink eye') either directly or through transferred bacterial infections.

Musca spp. are also vectors of the stomach worm *Habronema* spp. the larvae of which cause granulomatous cutaneous lesions around the face (see p. 217). *Musca* spp. also transmit the eye worm *Thelazia* spp.

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Hydrotaea irritans, 'the sheep head fly', and other *Hydrotaea* spp.

H. irritans is by far the major nuisance fly on grazing horses in the summer months in Europe and across the northern hemisphere (it is not present in the USA).

Hydrotaea albipunta is another very common species, confined to the corners of the eyes. There are many other species of *Hydrotaea* and related genera which pester horses in various parts of the world.

Identification

CD

Female *H. irritans* are 4–5 mm in size and have a pale green abdomen with a yellow wing base **(Fig. CD3 • 7)**. Males are slightly larger and black in colour. *H. albipunta* resemble *H. irritans* but are 3–4 mm in size and the abdomen has a bluish hue.

Life cycle

The larvae and puparia of *H. irritans* develop in soil and there is a single emergence each year. Males emerge in mid-July and swarm around horses outside stables. Females emerge several weeks later, feeding on secretions around the eyes and muzzle but the entire body may be attractive. The mouthparts are adapted to abrade the skin and this species may initiate small wounds or enlarge existing ones, particularly on warts and sarcoids. They often feed on blood released around the head of a biting fly. *H. irritans* may be present on horses until late October. *H. albipunta* has a similar life cycle to *Musca autumnalis*, breeding in animal dung on pasture, and there may be more than one generation each year.

Significance

H. irritans causes major annoyance. The intensity of fly challenge due to *H. irritans* may be increased by the presence of adjacent cattle and sheep pastures, particularly near wooded river meadows, on which populations of these flies are particularly abundant. *H. irritans* causes intense irritation and associated cutaneous damage. Feeding around the corners of the eye can result in a conjunctivitis (fly eye) and they may be a vector for *Moraxella* spp. bacteria.

CD

Morellia spp.; *Drymeia* spp.; *Fannia* spp.

Morellia spp. and *Drymeia* spp. breed in dung on pasture; *Fannia* spp. (family Fanniidae) are associated with decaying vegetation, compost heaps, etc. *Morellia* spp. are metallic black/blue in colour (7–8 mm) with grey-dusted thoracic stripes **(Fig. CD3 • 8A)**; *Drymeia* spp. closely resemble *H. irritans* but are darker (see **Fig. CD3 • 8B**). *Fannia* spp. are usually small, dark brown species with a pointed abdomen **(Fig. CD3 • 8C)**.

CD

All three genera feed exclusively around the eye and muzzle, contributing to annoyance and eye pathology in temperate countries.

CD

Myiasis flies

Myiasis is the invasion of living tissue by fly larvae and is a serious problem in livestock throughout the world. Fly larvae complete three instars while feeding and moulting in the hosts' tissues. When wounds are involved, myiasis is said to be 'traumatic'; where larvae mature inside a boil, the form of myiasis is termed 'furuncular' (see p. 207).

The family Calliphoridae contains several species which can cause cutaneous myiasis in horses. The following species are 'facultative', i.e. they usually breed in carrion but the flies may opportunistically lay eggs on animals. Flies in this family may have metallic green, blue or dark blue sheens on the thorax and abdomens. In temperate regions, *Lucilia* is the most important genus; the larvae of *Lucilia sericata* can initiate a lesion and *Lucilia* is known as a 'primary' myiasis fly. Temperate climate calliphorids also include *Calliphora* spp. and *Phormia* spp. Their larvae can be involved in secondary invasion of tissues, i.e. the tissues have already been invaded by primary myiasis species. Other less important facultative myiasis species are the flesh flies, *Sarcophaga* and *Wohlfahrtia* spp., which are non-metallic, grey-coloured with chequered patterns on the abdomen.

Myiasis in horses due to the calliphorid genera *Cochliomyia* and *Chrysomyia*, the 'screw-worms', is more common and serious in the tropics and subtropics than in temperate zones. These species, which are obligate parasites of animals (they do not breed in carrion), deposit eggs on any available open wound (including tick bite lesions), and their larvae are highly invasive and more pathogenic than facultative species.

Facultative species: *Lucilia* spp.; *L. cuprina* ('greenbottles')

In sheep these species usually initiate a 'strike' (primary myiasis) whereby eggs are laid on faecally soiled skin. In other animals eggs are deposited near old poorly healing wounds or in damaged areas producing odours that are attractive egg-laying cues. *Lucilia sericata* is mainly found in Europe and Asia; *L. cuprina* is mainly found in South Africa, Australia and New Zealand.

Identification

Lucilia sericata are metallic green flies (Fig. 3.15 and **Fig. CD3 • 9A**) and measure approximately 10.0 mm. Their eggs are creamy white, approximately 1 mm in size and may be seen in groups or scattered on the skin. Second- and third-stage larvae a are 5–10 mm in size and are segmented and barrel-shaped. The puparia are tan-brown.

Life cycle

Most of the calliphorid myiasis species have similar life cycles. Eggs are deposited singly or in batches on the skin or lesion. In the case of *Lucilia*, first-stage larvae hatch within 18–24 hours and abrade the surface, feeding on serous exudates. There is a moult to the second stage after 24 hours and a further moult to the third stage after the same period, as the larvae feed and grow. Third-stage larvae **(Fig. CD3 • 9B)** may therefore be seen in lesions in less than 3 days. Larvae drop to the ground and pupate, with flies emerging 10–14 days later, but events are temperature-dependent. In northern Europe, there may be several waves of fly activity during the summer months but in warmer countries there may be no special pattern.

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Figure 3.15 *Lucilia sericata* (arrow) feeding on a recently dead carcass. Note other species of fly are also present.

Significance

The larvae only develop in necrotic exudative tissue. They do not attack or destroy live tissue but there may be some secondary metabolic effects of the infestation. Severely infested animals may be unresponsive, isolate themselves and do not feed. Death may result from septicaemia. The larvae are used therapeutically in the treatment of some exudative and sloughing wounds in humans and in horses.

Obligate species: *Cochliomyia* (=*Callitroga*) spp. ('army screw-worm fly'), *C. macellaria* and *Chrysomyia bezziana*

Cochliomyia hominivorax is endemic in South America and Mexico and the Caribbean. *Chrysomyia bezziana* occurs in Afrotropical, Middle East, Australia and Oriental zones of the world although they are also reported from South America.

Identification

C. hominivorax **(Fig. CD3 • 10A)** and *Chrysomyia bezziana* are bluish-green, with longitudinal black stripes on the thorax and orange-brown faces and eyes. Larvae **(Fig. CD3 • 10B)** are larger than those of *Lucilia* spp. The trunks leading from the posterior spiracles are darkly pigmented in *C. hominivorax*; these are not present in *C. macellaria*. In *Chrysomyia bezziana*, the segments of the third-stage larvae are broad and are circled by strong spines.

Life cycle

C. hominivorax and *Chrysomyia bezziana* are attracted to existing wounds in which the eggs are laid. The larval progression and life cycle is similar to *L. sericata* although larval feeding is more aggressive and invasive; feeding occurs deeper in the tissues. *C. macellaria* is usually a facultative species and is often involved in secondary myiasis.

Significance

Larvae typically feed head down deep in tissues so that only the posterior spiracles are visible and large foul-smelling areas are created. Tissues show progressive degeneration and become liquefied. If untreated, death may ensue.

Obligate species: *Cordylobia anthropophaga* (the 'tumbu fly')

The larvae of this calliphorid fly may affect animals in sub-Saharan Africa. Wild and domestic animals including horses may be infested by the larvae of these species.

Identification

C. anthropophaga adults **(Fig. CD3 • 11A)** are non-metallic, brown-grey with vestigial mouthparts. Larvae are barrel-shaped and covered in small spines **(Fig. CD3 • 11B)**.

Life cycle

C. anthropophaga has evolved an unusual life cycle strategy. Eggs are laid on the ground and hatch as a warm-blooded mammal passes by; the larvae burrow into the skin. A boil develops in the skin in which the mature larva develops over a period of months.

Significance

This species produces furuncular ('bot fly-like') myiasis. Swellings in the skin initially cause itching, followed by severe pain and distress as the larvae increase in size. Lesions, which may leak serous fluid, appear on the upper body, neck, back, flanks and tail and are often together in groups.

Family Oestridae

The family Oestridae contains flies the mature larvae of which are known as 'bots' or 'warbles'. These lesions develop from eggs deposited by flies on hairs on the horse's body. The larvae burrow into the skin and migrate within the animal tissues over a period of weeks or months. Infestations do not cause the extensive damage caused by blowflies and are not usually life-threatening. Important myiasis species are *Gasterophilus* spp., (the horse bot fly), *Dermatobia hominis* and *Hypoderma* spp. The latter two are mainly parasites of cattle but horses are also affected.

Gasterophilus spp. ('horse bots')

G. intestinalis, *G. nasalis* and *G. haemorrhoidalis* occur worldwide. *G. pecorum* occurs in Europe, Africa and Asia. Other species of minor importance are *G. inermis* and *G. nigricornis*. The life cycles of the different species vary only slightly.

Identification

Adult *Gasterophilus* spp. (Fig. 3.16 and **Fig. CD3 • 12**) are up to 2.0 cm, bumble-bee-like and hairy with a protruding ovipositor (a tube at the end of the abdomen for laying eggs). The wings are banded. *Gasterophilus* spp. eggs can be seen with the naked eye on horse hairs, being pearly white and 1.0–2.0 mm in size **(Fig. CD3 • 12A and B)**. The mature larvae are orange/red, cylindrical with spines on the segments (see Fig. 3.16B and **Fig. CD3 • 12C**).

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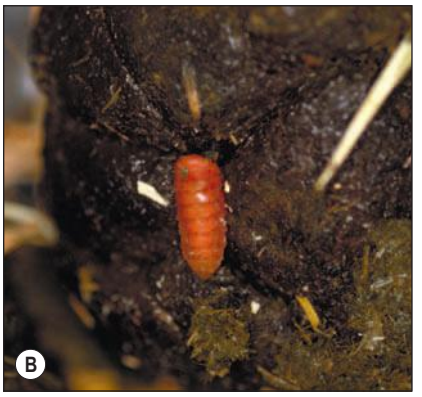


Figure 3.16 (A) Adult *Gasterophilus intestinalis* fly. **(B)** Third-stage (mature) larva in faeces. These are the outwardly visible signs of the infestation.

Life cycle

Adult *Gasterophilus* spp. are active for a period of weeks only in late summer in temperate regions, and persist in efforts to lay eggs around the legs or lip area causing considerable discomfort to the animal as they proceed; *G. intestinalis* is attracted to the forelegs and shoulders of horses. *G. nasalis*, the throat bot, and *G. haemorrhoidalis*, the nose bot, lay their eggs around the intermandibular area and around the lips, respectively. The larvae penetrate the tongue and buccal mucosa. The final site is the gastric tissue **(Fig. CD3 • 12D)**, apart from the larvae of *G. pecorum* which mature attached to the soft palate **(Fig. CD3 • 12E)**. Larvae are passed in faeces and pupate on the ground.

Significance

Fly activity (particularly due to *G. haemorrhoidalis*) is distressing as persistent attempts are made to lay eggs on hairs. The pathogenicity of the larvae is debatable. *G. pecorum* has been reported to be a (rare) cause of death when large numbers of larvae have attached to the soft palate and prevent feeding. Migrating early stage *Gasterophilus* spp. larvae may rarely cause facial and gingival ulcers and sometimes subcutaneous tracts are recognized (see p. 205).

Hypoderma spp. ('warble flies')

The main hosts of *Hypoderma* spp. are cattle and deer. Aberrant infestations of horses with *Hypoderma* spp. sometimes occur (in some areas of the USA horses are regularly affected) and a proportion of larvae will develop to maturity in the back, causing problems if they mature in the saddle region. Three species, *Hypoderma bovis*, *H. lineatum* and *H. diana*, are present mainly in the northern hemisphere and active in June and July.

Identification

Adults resemble 'bumble-bees' – the abdomen is covered with orange/yellow hairs **(Fig. CD3 • 13A)**. The eyes are prominent but the mouthparts vestigial. Mature larvae are approximately 2.5 cm, dark brown, with prominent segments which do not have spines **(Fig. CD3 • 13B)**.

Life cycle

H. bovis adults are active for a few weeks only and lay eggs singly on the hairs of the legs. *H. lineatum* lays eggs in rows **(Fig. CD3 • 13C)**. Larvae migrate and develop over a 10-month period within the host tissue. The final predilection site is the backline where lumps or 'warbles' form (see p. 207). Third-stage larvae eventually emerge from the lesion and pupariate on the ground.

Significance

When adult flies approach animals to lay eggs, animals panic and flee ('gadding') and this may result in serious self-injury. If maturing larvae die in situ, neurological and other problems can result.

Lice

Lice are insects belonging to the order Phthiraptera and are highly host-adapted and host-specific, spending their entire life on the host. Lice are flattened dorsoventrally and have claws for grasping hairs or feathers. There are two suborders:

1. Lice in the Anoplura suck blood and are called sucking lice.
2. Lice in the Mallophaga feed on tissue and are called chewing lice.

Haematopinus asini (the 'horse-sucking louse')

Identification

H. asini has a pointed head and strong legs with prominent claws (Fig. 3.17). This is a large louse (approx. 0.5 cm), yellow/brown in colour with dark markings on the lateral margin of the abdominal segments.

Life cycle

Lice have a hemimetabolous life cycle – there is no true metamorphosis and the immature instars resemble small versions of the adult. Lice in the two suborders have similar life cycles. Eggs, which can be seen with the naked eye, are glued onto hairs (Fig. 3.17B). They are whitish in colour, operculate (i.e. have a lid) with breathing holes. Eggs produce the nymphs which have a similar overall shape to adults. Sucking lice take many blood meals each day and can survive off the host for short periods only.

Significance

Lice tend to have preferential feeding sites where they congregate and cause intense irritation. *H. asini* are commonly found on the head, neck, back and inner legs. Heavy infestations of sucking lice can cause anaemia (see p. 216).

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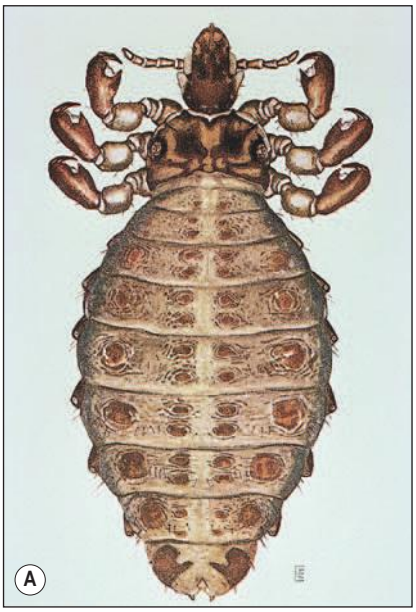






Figure 3.17 **(A)** Adult *Haematopinus asini*. **(B)** The myriad of lice included mature adults and developing younger individuals. The lice were just visible to the naked eye. **(C)** The eggs are harvested by simple skin brushings. The affected donkey was markedly anaemic and hypoproteinaemic.

Werneckiella (*Damalinia*/*Bovicola*) *equi* (the 'chewing louse of horses')

Chewing lice are smaller than sucking lice. *W. equi* measures 1–2 mm. This species is the commonest chewing louse of horses in temperate climates.

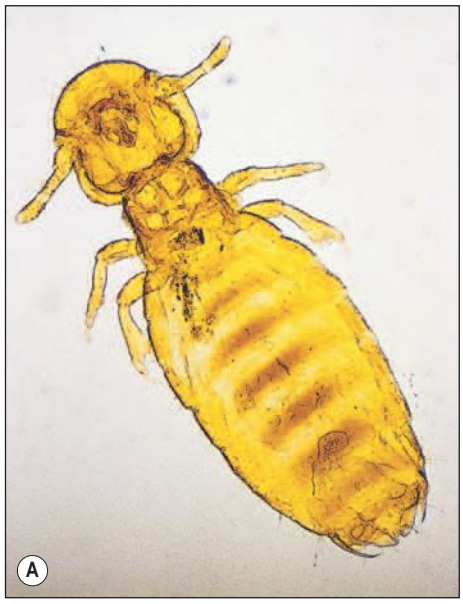






Figure 3.18 **(A)** *Werneckiella (Damalinia) equi* adult. This is a surface scale-feeding louse that occurs in vast numbers **(B)** and is highly contagious between in-contact horses, especially in winter months. **(C)** Sometimes they are difficult to see but a blanket or rug will usually bring them to the surface quickly and many will stick to the rug when it is removed (bottom right). The eggs ('nits') may be visible with a magnifying glass but can be identified easily with a fine 'nit comb'. Hair brushings are an effective way of harvesting the eggs and the lice themselves.

Identification

The obvious differences between *W. equi* and *H. asini* are seen in the shape of the head; the former has a large, round head approximately as wide as the body (Fig. 3.18), while the latter has a pointed head. *W. equi* is a light brown/cream colour whilst *H. asini* is dark nutmeg brown. The head of *W. equi* bears a pair of three-segmented antennae and the claws are small. The lice are just visible to the naked eye

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and may be much more obvious against a dark hair coat because of their creamy yellow/light brown colour.

Life cycle

The life cycle is similar to that of *H. asini.*

Significance

Little skin damage is evident when small populations of *W. equi* are present but heavy infestations often result in intense itching, alopecia and skin infections (see p. 215).

Fleas

Fleas are insects belonging to the order Siphonaptera. Horses may occasionally be bitten by fleas which normally infest other animals. An example is *Ctenocephalides felis*, the common cat flea, which is rather cosmopolitan in host choice. The burrowing flea species *Echidnophaga gallinacea* (the stick tight flea of poultry) and *Tunga penetrans* (the chigger flea) are reported to infest horses in warm countries.

Nematode parasites causing skin disease

Habronema muscae; *H. microstoma* (worldwide)

Muscid fly-transmitted larvae of the spiruroid nematode *Habronema* spp. are a cause of cutaneous habronemosis or 'summer sores' in equines in warm countries.

Identification

Lesions have a characteristic presentation around the eyes or face (see p. 218) but may appear at other fly feeding sites.

Life cycle

Adult worms in the horse's stomach produce larvae that are passed in faeces. These nematode larvae are ingested by muscid fly larvae present in dung. The nematodes pass into the adult fly during metamorphosis and develop into the infective stage, which invades the skin as the fly feeds.

CD

Significance

Granulomatous lesions form on the face, and nematodes can be seen in stained sections with notable eosinophilia. Ulceration of the eyelids is associated with eye feeding activities (see p. 218).

Thelazia lacrymalis

Thelazia lacrymalis is found in areas of Europe and North America.

Identification

The small thin white worms, 1.0–2.0 cm in length, are found in the ocular region, nasolacrimal ducts and within the conjunctival sac.



Figure 3.19 An adult *Onchocerca cervicalis* worm being extracted from a suppurating and necrotic nodule in the withers region of a donkey in Mali. The worm itself is almost invisible (arrows).

Life cycle

Adult worms infect the eyes and are viviparous – first-stage larvae are produced and picked up by muscid flies during feeding. These larvae develop to the third-stage instar (L3) within the ovaries of the fly and are transmitted to the eye region when the fly feeds, thus developing to an adult.

Significance

Worms and larvae in the eyes cause lacrimation and conjunctivitis. In heavy infections the cornea may become cloudy and ulcerated. Adults can survive in the eye for several years.

Onchocerca cervicalis

Onchocerca cervicalis is a filarial nematode transmitted by *Culicoides* spp. and other biting flies. The worms cause lumps in the skin of the withers and more extensive skin lesions on the neck, face and distal limb **(Fig. CD3 • 14A)**. Nodules in the lower limbs and ligamentum nuchae may be fibrous.

Identification

Adult worms measure several centimetres and are thin and white **(**Fig. 3.19 and **Fig. CD3 • 14B**). They are usually coiled up within nodules and can be extracted from these relatively easily. However, they are very long (often up to 30–50 cm or more) and incredibly thin, being no thicker than a strand of very fine cotton thread, so it does take some skill and experience to find them and extract them from a mass of granulating and necrotic tissue. They also decay very rapidly outside the host so it is not easy to collect specimens (see p. 222).

Life cycle

Adult worms produce microfilariae in the skin. When ingested by a biting midge, the microfilariae develop into

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the third stage which is infective to other horses when the fly feeds again. Adult worms may take several months to develop to maturation within the nodule.

Significance

The site of the bite initially develops into a diffuse swelling which becomes a palpable lump. These lumps may calcify or become infected with bacteria, leaving open purulent wounds (see p. 222).

Strongyloides westeri/*Pelodera strongyloides*

Strongyloides westeri and *Pelodera strongyloides* cause similar skin disease through skin penetration. These are free-living nematodes that are facultative pathogens of the gastrointestinal tract of horses. Skin disease occurs when large numbers of larvae penetrate the skin usually of the legs in unhygienic conditions (see p. 220).

Identification

The larvae are difficult to identify without careful reference to parasitological textbooks. Washings can be collected from suspected cases by enclosing the whole limb in a plastic sleeve and washing the skin firmly with warm water agitation. The washings are suspended for an hour or two and the deposit examined under a dissecting microscope. The larvae are highly active and can be readily seen, usually in profusion.

Life cycle

Infection is acquired when the third-stage larvae penetrate the skin (usually in regions in contact with wet, muddy, unhygienic conditions) and then undergo a long visceral migration or by direct ingestion of the infective third-stage larvae. The larvae of *S. westeri* are also thought to be shed into milk and so a foal may be infested in that way. The adults develop in the large colon and shed eggs onto the pasture. The eggs are indistinguishable from strongyle or cyathostomin eggs.

Significance

The penetration sites (usually on the distal limb with *S. westeri* and on the limbs, body, belly and neck with *P. strongyloides*) are pruritic and erythematous and affected foals will usually bite and worry at the sites (see p. 222). *S. westeri* has been blamed for introducing *Rhodococcus equi* to the mesenteric lymph nodes because the organism has been identified in the free-living larvae (Dewes 1972). Adults are seldom affected but some transient distal limb pruritus cases may be associated with *P. strongyloides* in adult horses maintained in poor, wet, unhygienic conditions.

Oxyuris equi (pinworm/threadworm)

This helminth parasite inhabits the large and small colon of horses and causes dermatological disease through the egg-laying habits of the adult female worm. The eggs are laid on the perianal skin where both the process of laying and the 'adhesive' used to fasten the eggs to the skin are causes of perineal irritation and itching. In some cases the signs can be severe (see p. 224). Many experienced dermatologists have never encountered this condition – it is rare in environments where avermectin wormers are used regularly but can be a significant cause of morbidity.

Note

This parasite is in no way related to the human threadworm, *Enterobius vermicularis*.

Identification

CD

The adult worms are seldom seen but they have a characteristic unusual shape **(Fig. CD3 • 15A)**. The eggs can be harvested using the clear adhesive tape procedure (see p. 66) and are obvious when viewed under even a low power microscope **(Fig. CD3 • 15B)**. They are usually present in large numbers in rafts and sheets ('rust') .

Life cycle

The eggs laid on the perianal skin by the adult female worms fall to the ground after some days, where they hatch and develop into third-stage larvae. These are eaten directly from the pasture. Usually the eggs are laid in large numbers and they are shed intermittently from the skin surface so pastures may be widely infected with a low egg burden.

Significance

Oxyuris equi causes perianal irritation – often severe. This can result in persistent severe itching and self-trauma around the anus. The itching is probably caused more by the adhesive the eggs are surrounded with than the movement of the adult females but it is easy to understand why the horse can be severely distressed by the condition (see also p. 224).

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SAMPLES FOR DERMATOLOGICAL INVESTIGATIONS

Groomings

Indications

This is a simple and effective test for detection of ectoparasites such as lice and surface-feeding mites such as *Chorioptes equi* (see Table 3.3) and for collection of crusts and scale for microscopic examination. It should be applied to all horses presented with pruritus. It is also useful for differentiating scale and crust (see p. 22).

CD

The advantages of the brushing/grooming tests include:

1. ease and non-invasive nature of the procedure
2. speed of sampling of a large area so that low numbers of parasites can be detected more effectively
3. only cheap and simple (disposable) equipment is required.

The only disadvantage is that it may over-interpret the significance of a few parasites in some cases but this can be overcome by parasite identification. The question that needs to be asked is: 'How likely is this parasite to be a cause of the problem being investigated?'

Method

A stiff brush such as a nail scrubbing brush or preferably a 'denture-type' toothbrush is used to sweep skin debris and hair particles into a suitable container (such as a plastic Petri dish) (Fig. 3.20) or onto a piece of stiff cardboard, thick paper or even a dustpan held below an area of affected skin. The groomings can be placed directly onto a dark tile for immediate examination using a simple (illuminated) magnifying glass or placed into a clean, closed, but not airtight, plastic container for later microscopic examination.

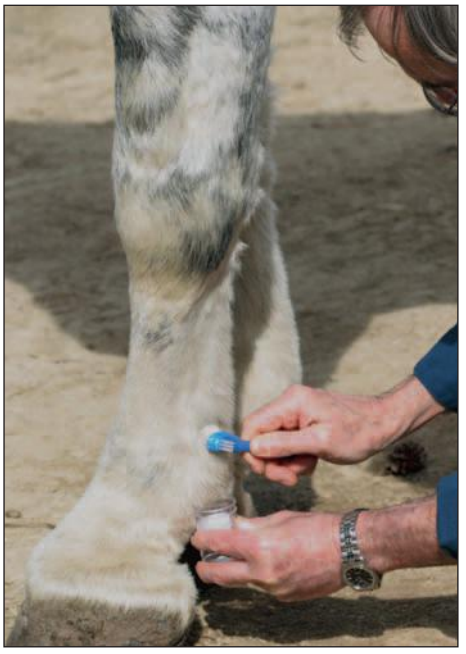


Figure 3.20 Groomings being taken with a stiff (denture) brush from the palmar cannon region of a horse presented for investigation of pruritic seborrhoea. The groomings are collected in a Petri dish or a wide-mouthed, sealable plastic pot held under the area.

Interpretation

Lice and other large parasites may be identified macroscopically (particularly when placed on a dark ceramic tile). Movement of parasites is often the easiest feature to notice and this is usually more obvious if the sample is warmed gently.

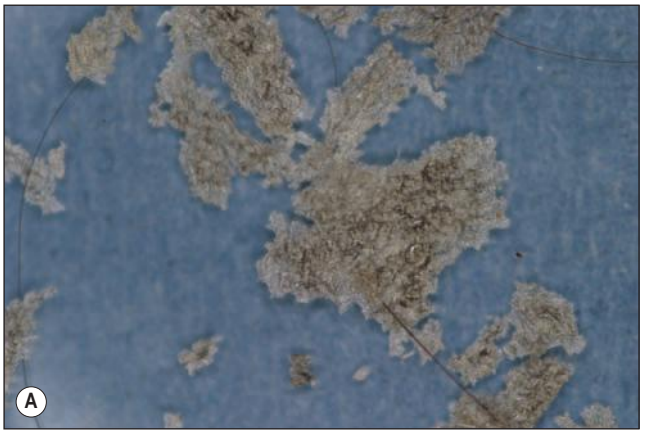
A magnifying glass is helpful but a binocular 'dissecting' microscope is an excellent method of examination for smaller parasites and their eggs **(Fig. CD3 • 16)**. It allows the rapid examination of a large amount of material under ideal lighting conditions.

Groomings also permit the accurate differentiation of 'scale' and 'crust' (Fig. 3.21).

Hair sampling

Indications

Hair sampling is indicated when there are altered growth patterns, broken hairs, crusts, scales and actual loss of hair (alopecia). As dermatophytes are associated with hair follicles, there are advantages in plucking the hair with a pair of haemostat forceps (artery clamps) as opposed to using a



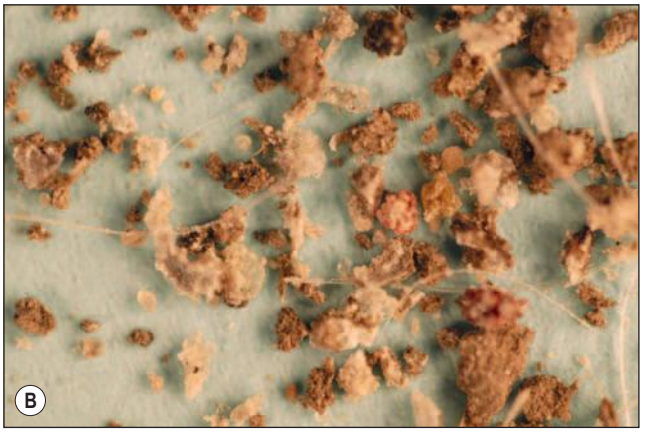


Figure 3.21 Scale **(A)** and crust **(B)** can be differentiated under a low power dissecting microscope. Note the reddish discoloration of some of the crusts indicating some degree of blood loss.

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skin scraping. Horses suspected of dermatophyte infection (ringworm) or *Dermatophilus* spp. infection are examples in which hair sampling is useful.

The microscopic study of hair quality has not been well established in horses but it is possible to achieve some important diagnoses by macroscopic and microscopic examination (light and electron microscopy) of hair shafts and bulbs.

The advantages of the method include the ease of sampling (usually there is no significant resentment) and the simplicity of collecting a large number of hairs. However, there are dangers in sampling hairs that have a lesser diagnostic value as a false negative or a false positive result may be obtained.

The method is most applicable to dermatophyte infections (ringworm) but is not generally useful for bacteriological cultures. However, some *Dermatophilus congolensis* infections can be identified.

Method

1 Lightly clean the area with 70% alcohol on a swab but do not scrub or abrade the skin.
2 Pluck hair and associated scabs from the margins of a number of fresh lesions within the cleaned area (Fig. 3.22 and **Fig. CD3 • 17A–D**).
3 Place the harvested crusts and hairs in numbered sterile bottles, leaving the caps slightly loose; if transported, use cellulose tape to secure lids without sealing the container and place in numbered/identified sterile bottles.

Note

For fungal culture the bottles should not be airtight as this might encourage overgrowth of saprophytic fungi.

4 Examine the samples in an appropriate fashion (see below) in the practice laboratory or dispatch to a diagnostic laboratory with a full history.

Interpretation

Positive results are very satisfying, but failure to demonstrate the organisms (false negative results) may be due to the sampling of an old lesion. Submission of fresher samples and scabs for culture to the microbiology laboratory can help.

Typical branching mycelia-like organisms and spores attached to or associated with the hair shafts will be seen in positive ringworm samples.



Figure 3.22 Hair plucking. It is important not to 'clamp' the hair too tightly.

Direct examination of hair and skin samples for dermatophyte organisms

Materials required

- Clearing agents
  - 10% potassium hydroxide (KOH) solution
  - Chlorolactophenol (made by mixing 25 mL liquid phenol, 50 g chloral hydrate and 25 mL liquid lactic acid in a container in a fume cupboard; it should be stored in a dark glass bottle and is ready for use in 2–3 days).

Note

All clearing agents are seriously caustic – skin/eye contact must be avoided. In the event of skin contact the area should be washed with copious running water. If the eyes are splashed medical opinion must be sought immediately after washing the eye with copious water.

- Fungal stains such as chlorozol, lactophenol–cotton blue (these can be applied to emphasize mycelium or spores).

CD

Method

1 Place 'teased-out' specimen hairs on slide and carefully apply a few drops of the selected clearing agent.
2 With chlorolactophenol clearing is more or less immediate; apply cover slip and examine immediately.
3 When 10% potassium hydroxide (KOH) is used the slide should be warmed to 45–55°C for 15–20 seconds; apply a cover slip and examine under the microscope.

Note

- Repeated/prolonged warming is not required and the sample should NOT be boiled – the correct temperature is comfortable warmth when the under-surface of the slide is held against the back of the hand.
- *Great care should be taken to avoid the caustic agents touching the skin when warming the slide.*
- Spread of the caustic onto the bottom or sides of the slide will also cause serious damage to the microscope!

4 Examine under low-power magnification (×4 or ×10) first to identify a normal hair for reference and obvious abnormal hairs. Re-examination with a higher magnification (×40) can help to confirm the location of the spores.

Interpretation

Infected hairs are pale and swollen compared with normal hairs.

Fungal spores are very small and may be present as chains on (*Trichophyton* spp.) or within (*Microsporum* spp.) the hair shaft.

- Mixed populations of Gram-positive cocci consisting of *Streptococcus* spp. and chains and clumps of *Staphylococcal* spp. bacteria may occasionally be found in cases of pastern folliculitis but their significance may be marginal.

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- *Dermatophilus congolensis* is a Gram-positive, branching filamentous bacterium. The bacteria divide longitudinally and horizontally, forming parallel rows of coccoid bodies ('railway-line organisms'; see Fig. 6.28). Specific details for direct smear examination for *Dermatophilus congolensis* are given below.

Specific examination techniques for *Dermatophilus congolensis*

Impression smears taken directly from the underside of freshly removed scabs can be heat fixed (the slide should be passed briefly over a flame until the slide feels just too hot to rest on the back of the hand). It is then cooled and stained with methylene blue, Gram's, Giemsa or Wright–Giemsa (Diff-Quik) stain.

- Place a drop or two of saline on a clean microscope slide.
- Clip off excess hairs from the crust sample and place the crusts into the saline drops. Gentle teasing out of the sample can hasten the procedure.
- Allow the specimen to macerate/soften for a few minutes and then remove the larger particles and hairs. Mix the residues into the saline until there is a distinct milkiness.
- Spread out the material on the slide and allow to air dry.
- Heat fix the slide by gentle warming to 50–60°C (50°C is just too warm for the back of the hand).
- Stain with methylene blue, Gram's, Giemsa or Wright–Giemsa (Diff-Quik) stain.
- Examine under low power (×40) or oil immersion (×100) magnification.

Note

Examination of direct smears for dermatophytes and dermatophilosis can be time-consuming and even then may not lead to a positive identification. All specimens should therefore be cultured for positive confirmation (see culture methods, p. 65).

Examination of hair for dermatophytes is facilitated by using mineral oil or, better still, a clearing agent such as 10% potassium hydroxide (KOH) or chlorolactophenol.

Trichogram

Collection of hairs for direct examination of their cyclical status and the features of the hair shaft and the anagen:telogen ratios is not well established in horses. Given the number of disorders that are characterized by altered hair growth or density this is surprising; the approach is widely used in small animal dermatology.

The hair of a newborn foal is recognizably different and the stage of gestation can be indicated by the state of the hair growth and density. Premature foals and those with delayed fetal development and those suffering from pre-partum hypothyroidism have reduced hair density and reduced hair shaft diameter and strength. Hairs grow in cyclical fashion and the biggest difference is between the summer and winter coats. The change is driven by photoperiodicity. Normal hair shafts are uniform in diameter but taper towards the tip.

There are significant differences also between various breeds (some are curly coated and some are straight, some have long hairs while others are shorter); this is also altered by environmental factors. For example. the hair coat of some mountain and moorland breeds of ponies, such as the Exmoor in the UK, have been studied in detail and found to have a distinctly different hair coat pattern and structure as a foal, a yearling, an adult and an aged horse (Speed 1960). There is a lesser variation in the mane and tail hairs, as might reasonably be expected (Song et al 1991).

- Anagen (growing phase) hairs can be recognized by a relatively plump shiny, glistening hair bulb. The root is soft so it may bend during drying.
- When there is early cessation of growth (catagen) hairs have a longer, thinner hair bulb – sometimes with a constriction within the bulb itself.
- In the true catagen stage the hair bulb is small and more 'onion shaped'.
- Telogen (resting stage) hairs are straight and have an apparently rough surface. The root is club- or spear-shaped and usually non-pigmented.

Method

To obtain a representative sample of hair for generalized skin disorders in which hair loss or abnormal hair character is present, a protected haemostat is used to pluck hair from typically affected areas. (Hair shafts can be damaged by the serrated grip of a haemostat so it is useful to protect the hair by covering the serrations with a sheet of paper.) It is important to sample from several areas so that a truly representative sample is collected. The hairs are placed carefully on a flat piece of greaseproof paper and then wrapped prior to despatch.

The hairs are examined under a dissecting microscope immersed in an oil or liquid paraffin drop and the anagen: telogen ratio calculated from at least 200 hairs. The sample is examined carefully for evidence of broken or damaged hairs and for abnormal curling and distortions.

Interpretation

Currently there is limited information to glean from sampling of hairs. However, study of the hair shafts might identify the difference between hairs that have been broken by, for example, rubbing/pruritus and those that have been cut. Hairs that have been chewed off either by parasites or by the horse itself can look similar. Hairs that are easily plucked are usually either in the stages of shedding or have an abnormal follicular development.

Currently useful features include:

- The stage of growth, the length and quality of the hair shaft and the presence of broken hair shafts. Normal horses have a variety of hair shaft growth types with approximately 50/50 distribution of telogen and anagen hair shafts. No normal animal should have *all* telogen or *all* anagen hairs. Where this is present anagen/telogen defluxion or suspended hair growth disorders are suspected. Large numbers of telogen hairs are taken to suggest endocrine or metabolic disorders (Scott & Miller 2003).
- Abnormal curling, thinness or distortions suggest metabolic or nutritional problems.

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- A dry staring coat is usually a sign of prolonged poor nutrition. This can be the result of an abnormal general nutritional state but is more often an indicator of systemic disease. Possibly the most obvious changes that occur with respect to the general appearance of the hair are an excessive dryness/brittleness typically seen in anhidrosis and seborrhoeic (greasy) changes that occur in a number of ill-defined circumstances in some horses. Some toxins such as arsenic, mercury and selenium cause serious and recognizable changes in the hair shafts.
- The hair shafts (and hoof clippings) may provide a useful forensic indicator of poisonings and other toxicity if suitable toxicological analysis can be undertaken.
- Broken hair shafts indicative of pruritus have an obvious jagged free end.
- Hairs infected with dermatophytes have obvious spores either within the hair shaft (endothrix spores) or outside the shafts (ectothrix spores).
- Parasite eggs can be recognized with the naked eye in some cases, e.g. *Gasterophilus* eggs or louse eggs (nits) or under the low power microscope (mite eggs).

Skin scrapings

Skin scrapings are primarily used to identify the burrowing mites *Sarcoptes* or *Demodex*, which are rare in the horse, and sometimes bacterial and dermatophyte infections. Advantages of the procedure include the sampling of deeper epidermal and superficial dermal cells. The disadvantages are that the method samples a very small area and it may be resented by the horse.

From a parasitological perspective, the technique is not as useful as in many other species because the only significant burrowing mites on horses are *Sarcoptes scabiei* var. *equi* and *Demodex equi*. Both of these are found only in very particular circumstances (see pp. 195 and 196). Where these two are a significant problem they can be identified on scrapings taken from the deeper layers of the skin. Nevertheless, there is some justification in performing a skin scraping in all cases showing pruritus. Where *Demodex* spp. mites are found the clinical significance has to considered carefully. However, both *Demodex* spp., and *Sarcoptes scabiei* are found in immunocompromised horses from time to time. Skin scrapings may also be useful in the detection of nematode larvae such as *Pelodera* spp. or *Strongyloides* spp. Trombiculid mites ('harvest mite' or scrub-itch mite) are also occasionally seen in a scraping but the mites often fall off after feeding, leaving either a bare (alopecic) patch or a slightly itchy area without the presence of mites.

It is advisable to collect samples from as many fresh areas as possible owing to the difficulty in obtaining mites in many samples. There is no defined number of samples to take, however; more is always better than too few. Single samples often provide a false negative result.

It is very unlikely that surface-feeding mites and lice will be harvested by skin scrapings taken from very small areas of the surface skin. Sometimes small pieces of the parasites can be obtained but they are hard to see and even harder to identify; brushings are far more logical.



Figure 3.23 A deep scraping being taken from an area of pruritus. Note the shielded blade and the extent of the scraping. Note also how little area this harvests. Apart from *Demodex equi* and *Sarcoptes* spp., which are extremely rare, parasites are seldom found in this way. Some cases of dermatophytosis and deep infection may be diagnosed.

Bacterial and fungal cultures may be useful provided that no antiseptic washes have been used prior to sampling.

Several different types of scraping can be obtained.

Superficial scrapings Hair and macerated tissue is collected from the superficial layer of skin but there are few occasions when this procedure is helpful. The sampling zone is very small and the chances of finding significant parasites are very low. Sometimes it is helpful for obtaining specimens for fungal culture or bacterial culture and direct microscopy for fungal spores on hair shafts. The process does not cause bleeding.

Psoroptes, *Chorioptes* and *Dermanyssus gallinae* (poultry or fowl mite), and lice may be found in these samples under a low-power microscope or stereo (dissecting) microscope. With the aid of higher magnification, dermatophyte organisms (ringworm) may also be seen, but these specimens often require clearing in warm 10% potassium hydroxide first. The clinical significance of a fungal organism seen in these specimens cannot be assessed without culture.

Deep scrapings These collect material from the epidermis and superficial dermis. Sampling is deep enough for bleeding to occur and so blood cells are invariably present in the specimen (Fig. 3.23).

Demodex, *Sarcoptes* and to a lesser extent *Chorioptes* may be found in these samples under low-power microscopy. Fungal and bacterial elements may also be identified under higher magnification, but the pathological significance of any of these will vary with the presumptive diagnosis. Culture from the scrapings can be helpful but requires that no antiseptic has been applied.

Hair and scab scrapings These are taken to recover the superficial and deeper hair particles and scabs. They are technically very similar to hair pluckings but are obtained by scraping the area. It is a useful technique for fungal and bacterial culture as they may be less contaminated with commensal organisms from the surface layers of the skin.

Method

1 Lightly moisten the area with light mineral oil (liquid paraffin). Antiseptics should be avoided as far as possible if bacterial cultures are to be performed. If fungal cultures are intended then a wipe-over with

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an alcohol swab will reduce the number of contaminant bacteria and saprophytic and incidental fungi. If a viral condition is suspected then the area should be simply wiped with water – alcohol can have significant deleterious effects in the detection of virus particles.

2 *Superficial scraping*. A new, sterile #23 scalpel blade is held at right angles to the skin. A spoon curette is a useful alternative instrument as the accumulated debris is collected into the spoon as the procedure is continued. The selected area is scraped and hair and superficial skin are obtained and placed directly onto a microscope slide. Samples for culture can be placed into sterile labelled universal bottles for transport to the laboratory or into bacterial or viral transport medium.

Deep scraping. The skin is pinched firmly between finger and thumb and the skin is scraped with a #23 blade (or a spoon curette) held at right angles to the surface until bleeding occurs. The sample is placed on a slide for immediate examination or into a sterile labelled universal container or suitable viral/bacterial transport media.

3 Direct examination can be performed by adding an extra drop of oil to the sample and then a cover slip is placed carefully so as to avoid artefactual air bubbles, etc.

4 If dermatophyte (ringworm) infections are suspected, the sample can be cleared with 10% potassium hydroxide (warmed for 10 minutes) or chlorolactophenol.

5 The slide is scanned under low power (×4–×10). Most mites will still be alive and movement is then easily seen. Examination under higher power (×40) is used to identify mites, mite eggs and any fungal elements.

Interpretation

In most instances positive findings provide a positive diagnosis of disease. However, care should be exercised when *Demodex equi* or *Demodex caballi* are found as they can be commensals in clinically normal horses.

Even multiple extensive scrapings can be unrewarding and may suggest a negative result even in positive cases. Multiple samples will minimize but not eliminate the risk of these false negative results. There is no universally effective way of concentrating parasites to make diagnosis more accurate, although combinations of clearing agents and centrifugation can be helpful in some cases; they can also cause serious damage to the parasites and so make detection difficult.

Swabs/scrapings for culture

Viral detection

Viral culture or detection using electron microscopy or fluorescent antibody methods, etc. often require specialized sample collection methods. Scabs and infected tissue packed on ice are often useful for some diseases such as the pox and papilloma viruses. Scrapings and swabs should be harvested from the periphery of lesions because that would normally be where the lesions are youngest and the amount of virus present correspondingly greater (Gibbs et al 1980).

If any sample is to be subjected to viral detection methods, the laboratory should be consulted first. Specialized transport media and handling may be required. The samples will usually require to be kept in a viral transport medium and kept cold (±4°C).

Dry swabs rubbed over a lesion are virtually useless for culture of either bacteria or viruses. However, modern immunological methods do sometimes enable detection of some significant diagnostic material from even a few cells. For example, sarcoid may be diagnosed from superficial swabs taken from the surface of ulcerated lesions using a PCR method for bovine papilloma virus DNA (Martens et al 2001). Similar methods are used increasingly for the laboratory detection of rabies (conjunctival swabs), poxviruses and herpesviruses, all of which have some dermatological significance.

Bacterial culture

The horse's skin carries a plethora of commensal bacteria. Swabs taken from the surface of lesions are therefore often unrewarding or difficult to interpret and have a low diagnostic value.

Needle aspirates of pus and debris from the deeper layer of the diseased area consistently provide a more restricted and relevant culture population from which clinically and pathologically significant pathogens may be identified and subcultured more easily.

Bacteriological culture and bacterial sensitivity tests are universal laboratory procedures, but both are of limited value in the horse. It is as well to remember that drugs applied topically would usually have to be absorbed into the tissue to reach the organisms and topical antibiotics commonly do not reach the minimum inhibitory concentration, so that sensitivity results need to be interpreted with care. Furthermore, sensitivity profiles issued from laboratories may not take account of the species of animal involved and so there may be significant safety and efficacy implications in the displayed profile information.

Method

1 Locate an intact pustule or the most recently infected area, which displays the most characteristic clinical signs. Do not wipe or use antiseptic over the area.

Note

For fungal culture, samples must be obtained from the rim of active lesions and placed in a sterile container and in this case commensal bacteria may be reduced by a single wipe with a surgical spirit swab (see below).

2 Obtain a swab either:
- by using a sterile disposable needle to open the pustule prior to taking the swab for culture, or
- by using a sterile needle to obtain an aspirate for culture from deep within the tissue where deeper infection may be present.
- Alternatively, use a biopsy sample (see biopsy sampling techniques, p. 66) and obtain a swab for culture from the deep portion of biopsy (see Fig. 3.29). This technique should definitely be used if a deep bacterial folliculitis (or a deep mycosis) is suspected.

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Note

Viral transport media usually include antibiotics and so are useless for bacterial cultures.

Dry swabs are also virtually useless as they are liable to desiccation and significant alterations of bacterial culture are possible within hours.

3 Bacteriological samples are always placed directly into transport medium to forward to the laboratory. The transport medium is essential and a suitable type should *always* be used for the suspected pathogens.
   - If anaerobic bacteria such as *Clostridia* spp. are suspected, samples should be collected into anaerobic transport media for immediate dispatch. Most such media are specialized and need to be freshly prepared. The laboratory should be consulted for suitable anaerobic media and advice on handling the sample before the procedure is performed.
   - A specific method for culture of *Dermatophilus congolensis* is given below.

Laboratory culture is usually left to specialist technicians. The swab is plated out directly onto an appropriate medium or into broth. Sensitivity cultures may be useful, but delays are inevitable as they regularly take 24–48 hours longer than the identification of cultures (and sometimes longer if subculturing is required).

Interpretation

Apart from *Dermatophilus* spp., *Staphylococcus* spp. and occasional *Streptococcus* spp., the horse's skin abounds with commensal and contaminating bacteria, which reduces the value of skin cultures. However, if a biopsy sample is taken, a small portion can be aseptically removed and placed in transport medium for culture. This is often more useful.

Parenteral use of suitable antibiotics as identified by sensitivity testing may be inappropriate or unsafe in the horse, so great care must be used in the selection of suitable drugs for skin conditions. Some laboratories are willing to set up specific equine sensitivity profiles employing only those drugs that are suitable and available for the species but may need to be encouraged to do so. Some aspects of sensitivity testing by PCR genetic screening can be also helpful.

Examination techniques for Dermatophilus congolensis

Isolations from older or healing lesions present more difficulties and the useful, if time-consuming and complex, technique outlined below can be used to confirm the presence of *D. congolensis:*

- Small pieces of the scabs are placed in bijou (5 mL) (screwtop) bottles, and moistened with 1 mL distilled water and allowed to stand open for 3–4 hours on the bench at room temperature. This softens and rehydrates the crusts.
- The open bijou bottles are then transferred to a wide-mouthed jar and sealed in an atmosphere of about 20% carbon dioxide produced either directly from a cylinder or by burning a candle within the jar.

Note

The stages up to this point can be usefully performed in practice and will add significantly to the sensitivity of the culture in the laboratory.

The bijou bottles can be posted safely to the laboratory.

- After 15–30 minutes, the bottles are carefully removed and samples are taken from the surface water with a bacteriological loop and seeded onto 5% ox-blood agar.
- The samples are incubated at 37°C in 20% carbon dioxide atmosphere for 24–48 hours.
- Abundant, small, embedding, haemolytic, rough, fimbriated colonies, typical of *D. congolensis*, are obtained, usually in relatively pure culture.
- Identity may be confirmed by examining stained smears obtained from the colonies.

Fungal culture

Specimens for fungal culture must be collected correctly.

The site can be swabbed with an alcohol-based antibacterial solution to limit the bacterial overgrowth.

Specimens for dermatophyte culture should be placed in a paper envelope or a sterile (unsealed/non-airtight) plastic universal for transport to the laboratory. Sealed containers tend to encourage overgrowth of non-pathogenic fungi such as mucor. They seldom require any transport medium. However, where deep mycosis is suspected, swabs may be taken from biopsy specimens and in these circumstances transport medium may be helpful in preventing desiccation (see Fig. 3.29).

A positive culture of a pathogenic fungal organism is diagnostic for infection and appropriate treatment can then be started.

1 Remove samples from sterile bottles with sterile forceps.
2 Culture directly on dermatophyte test medium (DTM), which is based on Sabouraud's dextrose agar with pH indicator (methyl or phenol red is usually used) and antibacterial agents to prevent overgrowth of bacterial contaminants (suitable prepared media are available commercially from laboratory suppliers). Press the sample firmly into the surface of the medium but do not bury it.

Note

Some fungi require high concentrations of B vitamins and better cultures are achieved if these are added to the medium at the outset. A single drop of an injectable vitamin B complex solution is helpful.

3 Incubate at room temperature for 14 days with a loose cap. Incubate duplicate set of plates at 37°C for *Trichophyton verrucosum*.
4 Check cultures daily for colour change and growth of fungal colonies.
5 If necessary a sample is submitted to a specialist laboratory for confirmation and evaluation of doubtful samples.

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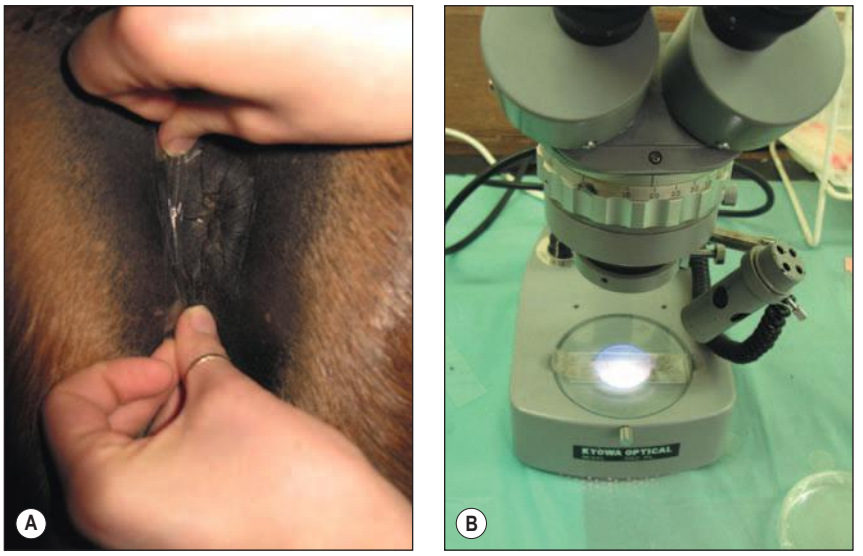


Figure 3.24 (A) Acetate tape preparation. **(B)** Examination under a dissecting microscope is simple and effective.

Interpretation

The identification of the specific species of dermatophyte present can be difficult and it is best left to a specialized technician.

Pathogenic dermatophytes metabolize protein early and so reduce the pH of the amber alkaline medium to a distinctive red colour indicative of an acid medium, usually within 3–5 days (before any colony is actually visible). Saprophytic fungi metabolize carbohydrate and only induce this change after 14 days or more (Grant 1991).

CD

Dermatophyte colonies are white/beige and are seldom if ever dark in colour. A positive identification can therefore be made in most instances, if at the same time as the colour change occurs (or within 24 hours of it) a white to beige, powdery or fluffy colony begins to appear (Evans & Stannard 1986). Light brown or dark, black/green colonies are invariably negative even if the medium colour changes to red. False results are uncommon, however; the characteristics of the culture are pathognomonic and examination of the macroconidia/spores will also support the diagnosis. Mistakes are usually the result of contamination or errors of interpretation.

Acetate (clear, adhesive) tape preparation

This is primarily used to detect *Oxyuris equi* eggs around the anus, vulva and perineum. It may also be useful in detecting surface-living lice and certain parasitic mites and their eggs.

Hair should routinely be clipped with scissors to allow the application of the adhesive tape to the skin and base of the hairs (Fig. 3.24A).

Method

1. If appropriate, clip hairs short (but do not shave) and gently remove any excessive crust.
2. Press clear adhesive tape firmly and evenly to skin and hair. Repeated dabbing of the tape onto the skin may help sometimes.
3. Remove tape and carefully place it sticky side down on a microscope slide over a drop of mineral oil so as to avoid air bubbles.
4. Place a drop of mineral oil over the tape and carefully apply a cover slip over the entire mount.
5. Examine for parasites and/or eggs under low power or a dissecting microscope (Fig. 3.24B).

Interpretation

Positive identification of *Oxyuris* eggs (90μm × 30μm) with their characteristic operculum at one end, or live or dead mites is diagnostic (see **Fig. CD3 • 15B**). Artefactual bubbles and pollen grains, etc. can be misleading. A negative finding is of course inconclusive. *Chorioptes equi* mites can often be 'harvested' in this way.

This may be a useful exercise to show to clients but it is time-consuming and can be unrewarding. Brushings have a far higher success rate for identifying parasites.

Tissue biopsy

Biopsy specimens obtained from skin lesions are used for several purposes and the technical aspects of obtaining a suitable specimen will vary. The objective is to collect a diagnostically significant specimen on the first occasion. There is considerable merit therefore in consulting with the pathologist at an early stage and it is sometimes helpful to ask the pathologist to be present during the procedure so that the surgeon can get the best advice on sampling. For example, it is sometimes very helpful to obtain a particular orientation of biopsy such as a shave biopsy or to use a special fixative. Furthermore, a full history and clinical report (possibly including a photograph of the condition and the biopsy site) should always be provided with the sample so that the pathologist is aware of the true nature of the case and can adjust the investigations so as to maximize the value of the procedure. It is not helpful to 'test the skill' of the pathologist by providing no or scanty information.

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The response of skin to a variety of insults can be histopathologically similar. Biopsies are often non-specific and therefore disappointing to both clinician and pathologist. For example, severe sunburn can be very similar to some types of squamous cell carcinoma. There may be no visible specific cause for superficial perivascular dermatitis and then the clinician needs to consider the meaning of such a finding in the light of the clinical picture. Sometimes rule-outs are just as significant. For example, a suspected diagnosis of pemphigus may be refuted by a biopsy – provided of course that the sample was truly representative and collected appropriately.

It is probably unrealistic to expect a single biopsy to provide all the possible results. If a primary diagnosis is required, it is usual to biopsy a representative mature or early lesion; the exception to this is any vesicular disease, which should be biopsied entire as early as possible in the course of this disease.

Skin biopsy has certain limitations. It can be very useful for papules or pustules but is much less so for ulcers and crusts. Biopsy of chronic lesions, superficial inflammatory changes and lichenified, crusted dermatoses often reveals little extra information because they are invariably secondarily affected by chronic changes.

Self-inflicted trauma is a common event in equine skin disease and is particularly so in pruritic cases. Vigorous or even milder rubbing can cause significant alterations, with primary lesions rapidly being converted into secondary lesions which are, naturally, non-specific. The salient features of the primary condition may be obliterated completely. Similarly in some conditions (especially the pemphigus group) it is easy to cause damage to the biopsy at the time of sampling by clipping and surgical preparation, so that a non-diagnostic sample is obtained.

Biopsy of a part of a large mass requires careful selection to avoid artefacts; the margins may be unrepresentative of the main mass and the central portion may be necrotic and fail to yield anything diagnostically useful. However, there are no hard and fast rules and regular biopsy usage may provide valuable clinical experience from which long-term benefits can accrue.

Note

While a definitive diagnosis is ideal, the elimination of other suspected diagnoses can be of importance in difficult cases.

Indications

Skin biopsies are taken:

- to establish a specific diagnosis
- to eliminate other clinical diagnoses
- to follow the course of the disease
- to confirm the completeness of excision of a tumour (McGavin & Fadok 1984).

Biopsy specimens are used for several pathological purposes including:

- histological diagnosis
- cytology
- special tests
  - immunohistochemistry/immunofluorescence.
  - bacterial/fungal culture and direct examinations (see above).
- virus identification (using PCR or ELISA or direct electron microscopy and culture).

Types of biopsy

Shave biopsy Shave biopsies are sometimes useful for the vesico-bullous conditions such as pemphigus vulgaris and pemphigus foliaceus in some anatomical sites such as the coronary band when full thickness biopsies are not possible.
Punch biopsy A column of tissue is removed with a commercially available disposable biopsy punch. These are available in several different sizes but specimens of less than 6–7mm diameter are probably less useful than larger ones. Pathologists are often significantly hindered by having very small specimens which may not be fully representative of the diseased tissue.
Wedge biopsy An outward-facing wedge with only a small amount of normal skin may be more sensible in some circumstances. The important portion is the abnormal tissue and the small segment of normal skin serves to provide the pathologist with an orientation of the lesion and an example of the normal skin for comparison. There is no material advantage in sampling the margin of the lesion and sending a large section of adjacent normal skin. The pathologist needs all the abnormal tissue that is available. A separate biopsy of adjacent normal skin may be sent if requested by the pathologist.
Excisional biopsy This is used when normal and abnormal tissue are required in the sample or when the lesion is so small that biopsy of a portion is difficult. It is also used when the lesion present (e.g. a vesicle or pustule) is likely to be adversely affected by partial biopsy. It is also indicated when malignancy and dissemination of tumour may follow partial biopsy.
Fine needle aspirate This is used to obtain a sample of cells or fluids from a lesion. Interpretation of cells obtained in this way is a very specialized technique often requiring an experienced cytological pathologist. The method of obtaining the cells is important as they are easily damaged beyond recognition. The pathologist should be consulted on the correct sampling and preservation methods.
Impression smears These have limited value. Many skin lesions have surface bacterial contamination (which may or may not be of clinical significance). Exfoliative and inflammatory cells are regularly encountered and again they may or may not be of significance. Interpretation is particularly difficult and is a very specialized procedure.

Tissue fixatives

The correct handling of tissues samples (whether cells or tissues) is essential if a good diagnosis is to be achieved. Prompt immersion of tissue in an appropriate fixative (formalin or glutaraldehyde if electron microscopy is required) is essential. Fixatives require a finite time to exert their effects. This may be dependent on the type and size of the specimen (and the extent of penetration required) but is seldom less than 24–36 hours. Pathologists are justifiably reluctant to process specimens at an earlier stage and more rapid results may require frozen sections.

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Note

- Following fixation, tissue becomes very rigid and inelastic – specimens shoved into narrow-neck bottles often cannot be removed for processing without breaking the container. Wide-neck containers of a suitable size must therefore be used at all times.
- The volume of fixative should be at least 10 times the volume of the tissue submitted.
- Samples to be sent in the post that are not urgent can be fixed for 4–5 days and then the formalin can safely be poured off to allow easier, cheaper and safer posting.

Fixative solutions:

- Buffered, neutral formal saline is used routinely. At least 10 times the volume of fixative to specimen must be present.
- Michel's medium was formerly used to preserve specimens for immunohistochemical investigations. This is now no longer required in many cases and consultation with the pathologist will establish its value in any particular case.
- Frozen sections are sometimes used to obtain a very rapid result. They require particular circumstances and handling of the specimen. Where this is required a pathologist or technician must be present to handle the tissue. There are few circumstances when frozen sections are required in horses.
- Glutaraldehyde is the preferred fixative for electron microscopy. Tissue must be cut into very small cubes (1–1.5 mm) and fixed as soon as possible.

Method

Suitable restraint with tranquillization if necessary should be used to allow handling of the horse and safety of the surgeon. Combination of an α_2-adrenoreceptor agonist (such as romifidine or detomidine) and an opiate analgesic (such as butorphanol) often allows minor surgery without the need for extensive local anaesthetic infiltration which itself may adversely affect the results of biopsy.

If at all possible it is better not to use any local anaesthesia although clearly that may vary with the needs of the case and the type of biopsy being obtained. For example, a biopsy of the nose or the skin of the distal limb is unlikely to be tolerated at all while a biopsy of a fleshy outgrowth such as granulation tissue or fibroblastic sarcoid may require no analgesia at all.

Although there is a strong temptation to clip, scrub and sterilize the site of a skin biopsy, this should be avoided as far as possible. A cursory (long) clip and a wash with soap and water if the area is heavily contaminated is the most that should be done. Shaving and vigorous cleaning/scrubbing prior to biopsy will usually induce detectable changes that may mask the pathology and furthermore any bacterial cultures may be significantly affected. Owners may be somewhat surprised by the lack of sterilization, but careful instruction should avoid any subsequent complication. The small biopsy sites left after using a biopsy punch will normally heal very quickly and do not normally need to be closed at all. Indeed, closure of a correctly taken biopsy can be counterproductive as any infection is likely to be sealed into the biopsy site.



Figure 3.25 A shave biopsy being taken from the coronary band of a horse with a severely inflamed coronitis identified as pemphigus foliaceus. The area was desensitized using a standard abaxial sesamoid regional nerve block.

A small skin biopsy will heal rapidly by contraction and normal second intention healing. A large biopsy such as an excisional biopsy perhaps will need some form of closure in many cases.

- Regional perineural anaesthesia will avoid any possible complications from local injection of an irritant/acidic chemical such as mepivacaine hydrochloride. Alternatively, a small volume of 2% mepivacaine hydrochloride can be placed deep to the lesion to be sampled using a 25G needle. A ring block of 2% mepivacaine hydrochloride can be used for larger lesions or where more extensive biopsy is required.
- Where local anaesthetic infiltration is unavoidable it is important to use as small a volume as possible and place it either deep to the tissues to be sampled or a few centimetres away from it. A useful tip is to leave the needle in situ after injecting the local anaesthetic so that the exact placement is recognizable after the required 5-minute wait.

Note

Many biopsies are quite correctly taken without any clipping or surgical preparation (which may distort or alter the results); a suitable systemic antibiotic can be administered immediately after the sample has been taken.

Topical antiseptic medication is also probably justified after the procedure.

Shave biopsy

A shave biopsy is a useful method for the biopsy of difficult sites, such as the coronary band, that is not, however, commonly used because there are specific technical difficulties for the pathologist.

The selected region can be anaesthetized using routine regional nerve blocks and so the technique is safe and easy and there is little risk of misleading inflammatory changes from local anaesthetic solution infiltration. The epidermis is shaved parallel to the surface in layers (Fig. 3.25).

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Figure 3.26 **(A)** A wedge skin biopsy being taken from a suspected occult/verrucose sarcoid lesion in the groin of an anaesthetized horse. **(B)** An excisional biopsy of an isolated cutaneous nodule. **(C)** It was closed with staples but this could be viewed as both unnecessary and a risk if the skin was not surgically prepared. It will, of course, depend on the size of the biopsy wound and the location.

Note

This method has some significant technical disadvantages and where possible a punch or wedge biopsy should be used instead. The heel bulb area is a less dangerous site for a full thickness biopsy; if this area is affected in addition to the coronary band, then this is clearly a preferable site for biopsy.

Method

- After suitable restraint and regional anaesthesia, a series of shallow shaves are collected parallel with the skin surface. It is essential to avoid any interference with the specimen during the procedure, so surgical scrubs and washes are not recommended; the removal of superficial scale and crust and epidermis by surgical preparation may render the biopsy valueless.
- Sampling continues until spots of blood are seen indicating exposure of the dermis. Scissors should not be used to cut the specimen off. The thin pieces of tissue are easily traumatized so they should not be gripped with sharp or crushing instruments.
- Suturing is not possible.
- A clean dressing will help to limit the secondary infection risks.
- Samples are placed directly into fixative solutions and submitted for histological examination. An accurate diagrammatic orientation of the biopsy should be made immediately to assist the pathologist.

Wedge or excision biopsy

A wedge of tissue is obtained by full thickness incisions. This is a standard approach that is widely used, but it is easy to collect the wrong tissue. Conventionally the clinician aims to include some normal skin in the biopsy but this should not dictate the extent or location of the biopsy. It is seldom helpful in any case. The pathologist requires a carefully taken representative sample of the lesion – if normal skin is included it should certainly not be the larger portion of the biopsy. In cases where a piece of normal skin is required, this should usually be taken separately from an adjacent piece of normal skin.

Method

- The chosen site should not be surgically prepared. The less the interference the better the result. It is easy to remove crust and superficial epidermis and so mask the true nature of the lesion. Ideally no clipping or cleaning should be performed at all.
- After suitable restraint and local/regional anaesthesia a scalpel is used to excise a representative piece or tissue. If normal skin is included the orientation should be described so that the pathologist can prepare the tissues correctly (Fig. 3.26).
- The wound is likely to require closure with staples or sutures in most cases because the sample will be relatively large.
- Antibiotics and dressings may be indicated.
- The tissue is placed directly into the fixative. It is often helpful to use a tissue cage to maintain the specimen in its proper flat orientation (see Fig. 3.28).

An *excisional biopsy* is similar but involves the removal of the whole lesion, which may involve rather more surgery if it is large. An elliptical incision with an appropriate margin for the type of tissue is made with a suitable scalpel to include all tissue down to the cutaneous muscle (see Fig. 3.26B and C).

Even though the skin is not aseptically prepared, sterile equipment should always be used. Scissors should not be used to cut the specimen free of the subcutis and neither should the specimen be gripped with sharp or crushing instruments.

For wedge and excisional samples, sutures, staples or n-butyl cyanoacrylate adhesives may be used to close the wound depending on the size and nature of the defect (see Fig. 3.26C). An accurate diagrammatic orientation (or preferably a photograph) of the biopsy should be made immediately to assist the pathologist.

Punch biopsy

Disposable skin punches are available commercially in various sizes ranging from 4 mm to 9 mm diameter. The punch is used to remove a core of abnormal tissue consisting of epidermis, dermis, subcutaneous fat and panniculus muscle. For equine skin 6–9 mm diameter punches are usually selected.

The punch is used to incise the skin using a single direction push-turn action. The punch should be rotated in one direction. This will minimize the stretch artefacts. Back and forth movements should not be used as this increases stretch artefacts.

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Figure 3.27 Punch biopsy technique. **(A)** A small bleb of local anaesthetic is carefully placed under or away from the chosen site. **(B)** The needle is left in place to mark the correct site. **(C)** The biopsy is taken carefully to minimize artefactual damage to the specimen. The skin defect is left to heal by secondary intention.

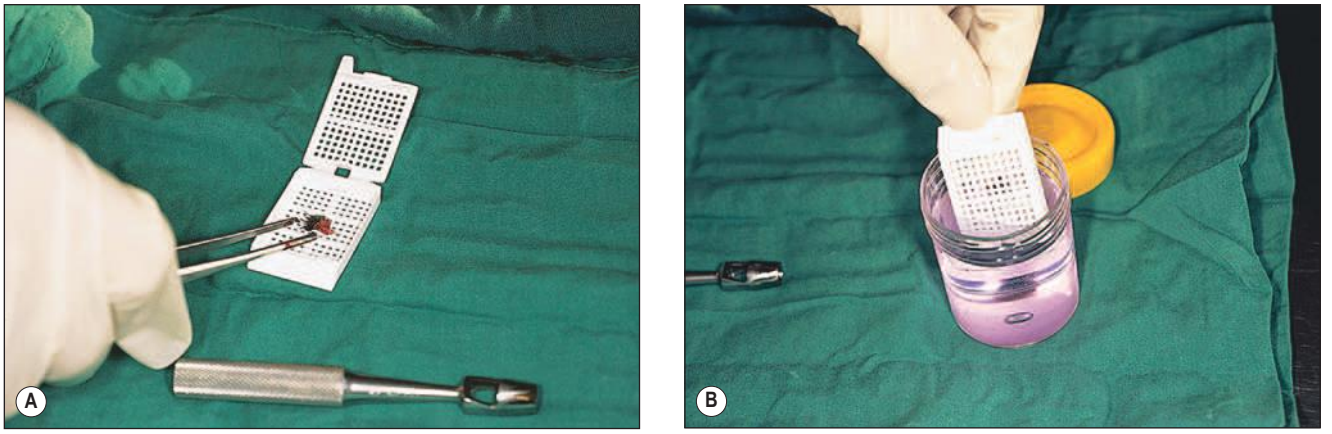


Figure 3.28 **(A)** A biopsy specimen being loaded into a specimen mesh to prevent distortion. **(B)** The specimen enclosed in the mesh being inserted into the fixative solution for dispatch to the pathologist.

Note

Significant iatrogenic (misleading) inflammation is caused by all local anaesthetic solutions. Ideally a regional block should be used in the limbs and face where there may be some resentment but at body sites it is usually quite possible to obtain samples without the use of local anaesthesia if suitable sedation (with an opioid analgesic) is used.

It is far better to obtain a diagnostic biopsy on the first occasion even if the horse has a small, short pain. Where local anaesthesia is required, a regional block or inverted 'L' block can be used. A very small bleb of local anaesthetic can be placed deep to the biopsy site. The needle is left in situ for the 5–10 minutes required so the site is not 'lost' (Fig. 3.27).

The specimen can be easily (and significantly) traumatized by rat-tooth forceps or by excessive pressure/squeezing by forceps and these should therefore not be used. The specimen is often attached by a fine core of subcutaneous fascia/fat. A 25G needle can be used to carefully lift the specimen so that the underlying attachments can be cut with the scalpel blade (not scissors). The sample is rapidly transferred to fixative.

Suturing is not usually required and indeed, in view of the lack of sterile technique, suturing can in fact be a disadvantage; closing a small site like this may increase the chances of infection. Leaving the site open results invariably in a rapid natural healing without complication. A dressing may be helpful in limiting the risks in some sites (e.g. on the lower leg region).

A specimen of adjacent normal skin may be obtained in a similar fashion for comparative purposes.

Specimen handling

Biopsy samples of skin which might become distorted during fixing should either be placed dermis down on a small square of cardboard for about 1 minute to adhere to the cardboard before being placed in fixative or be pinned gently to a piece of card in its natural shape. Alternatively, a specimen basket can be used to avoid distortion of tissues as fixation occurs (Fig. 3.28). These are simple and efficient means of preventing distortion without the need to pin the sample and are highly recommended.

1. Sample specimen with sterile swab for bacteriology and fungal culture if indicated (Fig. 3.29).
2. Place sample in appropriate fixative solution.
3. A further sample should be taken if a possible diagnosis of onchocercal microfilariasis (*Onchocerca cervicalis*) is suspected. The sample should be preserved fresh in a cooled container.

Precautions

Measures to improve the quality of the results from tissue biopsy include the following.

- Do not stretch the skin during biopsy. This causes stretch artefacts/distortion on both a macroscopic and microscopic scale. If a punch biopsy is to be taken, the

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Figure 3.29 A swab being taken from the depths of a biopsy specimen prior to the specimen being placed in 10% buffered formalin solution. Notice that the formalin pot is adjacent to the swabbing and open – this specimen revealed no culture almost certainly because of the potent sterilizing effects of formaldehyde. A repeat sample taken 3 days later was diagnostic. The formalin pots should be in another place entirely and the samples should not be handled together.

punch must be rotated in the same direction and must be very sharp.

- Use a scalpel, not scissors, to excise the subcutaneous tissue. This eliminates crushing artefacts along the line of incision.
- Try to avoid over-handling of the specimen and use fine plain forceps very gently or, preferably, not at all. A fine hypodermic needle is a useful way to remove/handle the specimen.
- Try to reduce specimen size to less than 1 cm to allow rapid and proper fixation.
  - Large samples should be serially cut into pieces < 1 cm and preserved in numbered separate containers with a diagram to help the pathologist orientate the specimen correctly.
  - Always use at least 10 times the specimen volume of formalin solution.
- Avoid *too small* specimens. Samples less than 4–6 mm in diameter are probably barely adequate and are particularly liable to artefactual distortions from stretching, compression or crushing during sampling and may be difficult to handle when fixed.
- Multiple sections are useful from large lesions and in some circumstances the complete specimen should be sent in a suitable container with at least 10 times the specimen volume of fixative. Solid intact specimens require to be cut open and advice should be obtained from the pathologist as to the best way of handling such a specimen.
- Remember that specimens will need to be removed from the container. A narrow-mouthed container (such as a glass bottle) means either that the specimen has to be handled roughly or that the container needs to be broken to get the fixed specimen out. Therefore, a suitable wide-mouthed, inert, clean container must always be used.
- Samples which have to be sent by post and for which there is no urgency can be fixed for a suitable time before being drained of fixative prior to posting. A day or two without the fixative will not usually harm the specimen – remember though to ensure that it is packed suitably to prevent desiccation. The pathologist will need to know that the sample has no fixative.

Biopsy technique (iatrogenic) artefacts are a serious limitation to diagnostic pathology. It is important that these are minimized as far as possible. There is little more disappointing than collecting a sample from a difficult horse and then finding out that the selection of the site or collection/handling errors have resulted in a non-diagnostic or grossly misleading sample. Not only does this mean that the procedure needs to be repeated with the extra expense and trouble but the time delay may mean that the results are often not as good. Some of the commoner artefacts are shown in Table 3.4.

Note

When dealing with a biopsy specimen that is going to provide several different specimens such as histology, bacteriology, fresh samples, etc. the method of collection must take account of the special requirements for each of these. In general, bacteriological swabs are the most sensitive and so this should be taken first, before any further handling is done. Any container of formalin solution must be kept well away from the sampling site – it is wise to leave it closed in another room until the bacteriological specimens have been taken. Only then should the sample bottle be opened well away from the swab. Formalin specimens and bacterial/viral/fungal swabs, pluckings or groomings must be handled, parcelled and posted separately.

Fine needle aspirate

Fine needle aspirates are widely used in other species and are being used more in horses since skilled cytopathologists are now available. The procedure, however, is often performed badly and the results are then disappointing. This is often a difficult procedure for solid lesions. It is important to sample the correct regions in the correct manner and to handle the specimens carefully.

Some important points for fine needle aspirates are:

1. *Identify the preferred site and prepare the skin carefully*, avoiding excessive trauma by avoiding scrubbing the lesion during preparation.
2. *Sample from the edge of the lesion*. Sampling from the centre of the lesion is not the best option. Many solid (neoplastic or inflammatory) lesions have a necrotic centre and so sampling from this may be easier but the results are less satisfactory. The best site is usually the marginal solid area adjacent to the normal tissues.
3. *Do not use a large needle*. A large needle will usually 'acquire' a high blood volume in addition to the cells and so it becomes much harder to find the pathology. A 21–23G needle is best.
4. *Do not use a large syringe*. The required vacuum is best achieved with a 2 or 5 mL syringe and a maximal vacuum of 2–4 mL before relaxing the negative pressure. Higher vacuums can cause major damage to cells and often the pathologically significant cells are the most sensitive to this.
5. Always use *new, unhandled (or freshly degreased) slides* to avoid grease marks and distortions.

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Table 3.4 Biopsy-induced artefacts in equine dermatology (after Logas & Barbet 1999)

Collection errors	Changes	Avoidance
Skin stretch artefact	Pyknosis/elongation/disruption of cells	Always rotate biopsy instruments the same way Avoid tissue tension during collection Use very sharp instruments
Compression/crushing	Crushed cells, connective tissue disruption	Avoid use of scissors (use scalpel blades). Use very sharp instruments Avoid use of forceps in handling specimen as far as possible
Contraction/rolling/distortion	Loss of orientation of specimen	Ensure large specimens are pinned out to avoid curling and orientated (photograph or drawing)
	Difficulty in handling fixed specimen to get correct block cut	Ensure fixative volume at least 10 times volume of specimen. Ensure container has suitable wide neck to allow specimen to be removed easily
Drying artefact	Desiccation and loss of cell differentiation	Fix samples as soon as possible after collection Keep wrapped in saline swab if delay is unavoidable
Inadequate specimen volume/size and location	Untypical tissue sampled	Ensure careful planning of tissues to be sampled. Consult pathologists if in doubt
	Higher chance of crush, tension or drying artefacts Failure to identify full range of tissues involved	Sample representative tissue
Iatrogenic inflammation	Acute inflammatory response in sample	Avoid scrubbing/clipping and avoid local anaesthetic if possible (use regional blocks or remote anaesthesia)

6 *Mark the slides with a lead pencil* – not a marker pen. Marker pen ink is dissolved by the alcoholic fixatives. It is best to mark the slides before preparing the aspirate.

Method

- Three or four very clean, degreased (new) glass slides are laid out on a bench and marked using a pencil on the frosted section with the case number and date and any other relevant information (Fig. 3.30A).
  - Identification marking should be done before using the slides so that the 'upper' surface can be easily identified throughout the processing.
  - Other types of marking pens will be dissolved by the alcoholic fixatives and stains.
- Where appropriate local anaesthetic agent can be injected beneath the mass – the needle can be left in situ so that its exact location is known (Fig. 3.30B).
- Attach the selected (23–25G) needle to the 2 mL syringe and insert the needle into the *margin of the lesion* (Fig. 3.30B).
- Repeated, short duration applications of suction to 2–3 mL are then applied.
- The needle is withdrawn and air is drawn into the syringe. Repeated jets of air are directed through the needle at the slides. Cells will be delivered to the slides (Fig. 3.30C).
- If advised by the pathologist, smearing of these can be performed but this often results in cellular distortions (Fig. 3.30D).

Cells from fluid-filled cavities (including bullae and abscesses) can be obtained using fine needles (Fig. 3.31). It is often helpful to identify fluid cavities by ultrasonography at the same time. This avoids indiscriminate, random exploration.

The sample obtained should be treated according to its purpose. Thus, samples for virus identification must be treated accordingly (consultation with the pathologist or virologist is advised) while those for bacterial culture can be introduced directly into either biphasic (blood culture) bottles or other transport media.

Most pathologists prefer that the slides are not fixed at all but rapidly air dried and packed carefully in a slide carrier for dispatch. In some cases alcohol fixation can be used; the pathologist may be able to give advice on this.

Staining of the specimens must be very carefully performed so that cells are not lost in the process – sometimes the pathological cells are few in number and hard to locate.

Interpretation

- Among the solid tumours only the melanoma provides an instant diagnosis – the black material is easily recognized even in unstained aspirates.
- The cytological examination of fine needle aspirates is a specialist procedure. The cells are often sparse and the variations may be subtle. It is wise to use the services of a skilled cytopathologist.

Impression smears

Impression smears are subject to considerable misinterpretation but for some lesions such as squamous cell carcinoma and some fungal skin infections they can be useful. The technique simply requires suitable exposed tissue.

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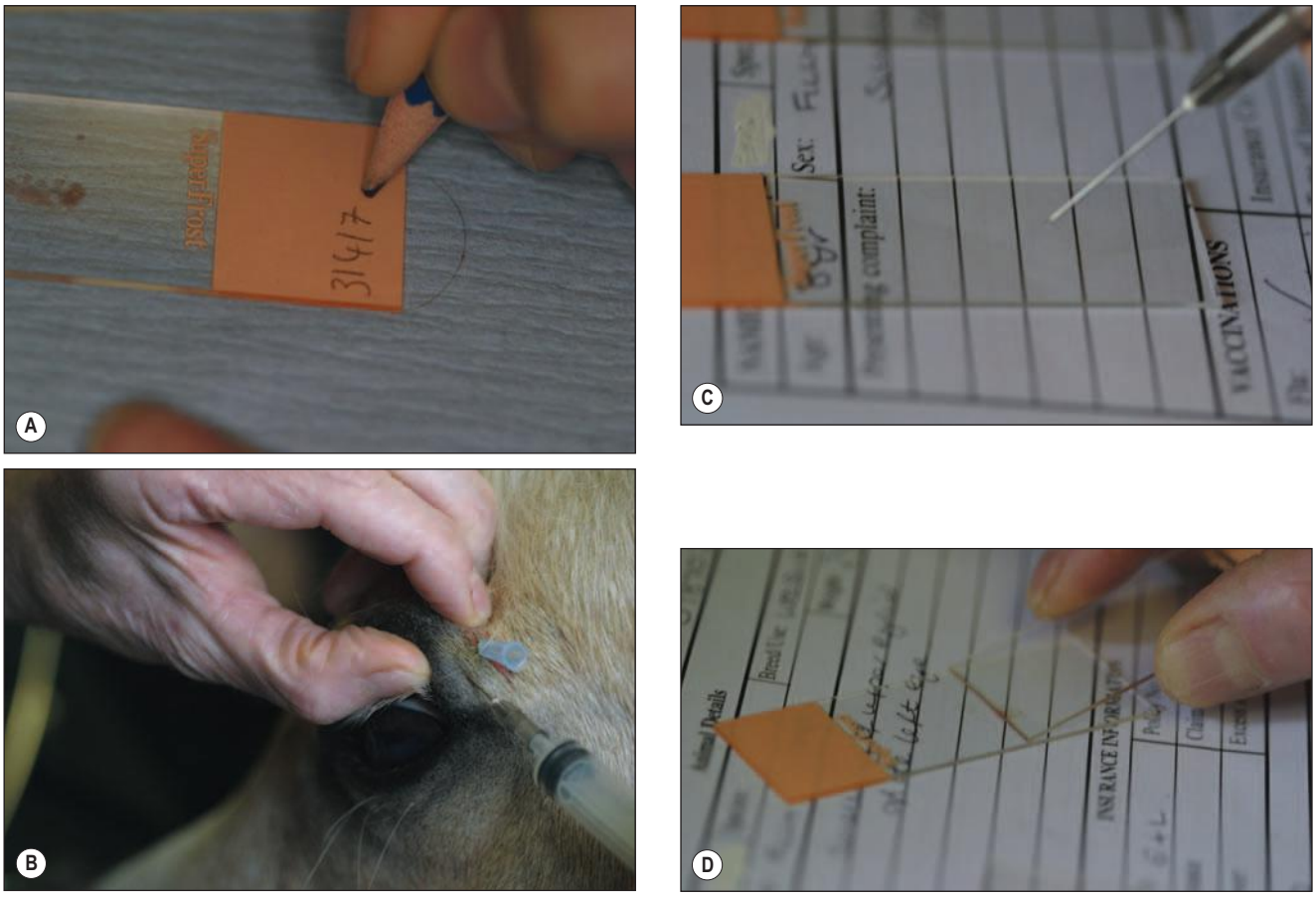


Figure 3.30 **(A)** Note the pencil marker! **(B)** A fine needle aspirate being taken from a solid nodule in the upper eyelid. Note the small syringe and the marginal placement of the needle. **(C)** The contents of the needle are 'jetted' onto the slide by ejecting air from the syringe. There is no need usually to further smear out the harvested cells. **(D)** The cells may be spread as here or left to dry in situ. Note that relatively few cells have been harvested but these do provide the best information. A large volume of blood or serum makes the whole procedure more difficult.



Figure 3.31 A needle aspirate being taken from a fluctuant cutaneous swelling. Culture and direct examination of a Gram-stained smear of the material confirmed that the swelling was an abscess.

Method

- Any atypical serocellular crust or debris or exudate is wiped carefully from the surface of the lesion using a dry sterile gauze swab.
- Sometimes it is helpful to gently squeeze the lesion to encourage exudate and exfoliation of cells.
- The slide is applied firmly to the exposed area and then removed. The slides are rapidly air dried or if indicated can be fixed by heat or alcohol immediately they are dry.
- Some tumours and subcutaneous nodules may require to be incised before the smear is taken (Fig. 3.32).

Interpretation

Diagnostically significant cells can be identified but often only a few significant cells are harvested. Ulcerated lesions often yield a variety of bacteria and normal tissue and blood cells (including white blood cells and erythrocytes) which may or may not be relevant to the underlying condition.

Different stains are used for different purposes; for example, Gram's stain can be used for bacterial identification while cell stains such as Wright–Giemsa can be used for cytology. Usually it is wise to check with the pathologist if staining or fixing is required immediately. Most will prefer that unstained, unfixed smears are submitted.

Impression smears may be very helpful for fresh *Dermatophilus congolensis* lesions (see p. 158) and for some fungal infections of the skin such as histoplasmosis (see p. 182) and cryptococcosis (see p. 183).

Abnormal cells such as acanthocytes can be indicative of pemphigus disorders.

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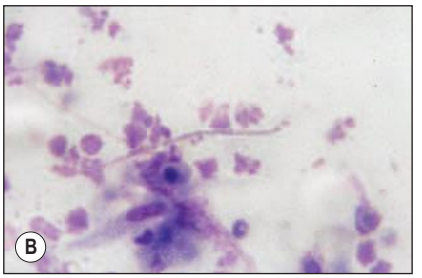


Figure 3.32 **(A)** An impression smear being taken from an incised solid skin nodule. The smear was unrewarding; only very few 'tumour' cells were identified among the blood cells and they were not of diagnostic value. **(B)** An impression smear showing typical *Dermatophilus congolensis* organisms.

Specific examination for *Onchocerca cervicalis* microfilaria

The suspected affected area is sampled by punch or excision biopsy technique as outlined above. As many horses in endemic areas have accumulations of *Onchocerca* located in the ventral midline region without apparent pathological effect, lesions away from this should be sampled, e.g. the head (particularly the face between the eyes and ears), neck, thorax and proximal regions of the front legs. In non-endemic areas there may be justification in sampling any lesion regardless of the area.

Method 1

1. A 6–8 mm biopsy punch is used to harvest a core of full thickness skin (as described above, p. 70) to obtain several samples from the lesions.
2. Place half of (each) biopsy in 10% buffered formalin for normal histology.
3. Place the second half (or halves) in a cooled plain container on ice. At the laboratory the sample is finely cut up (minced) and suspended in Tyrode's solution or phosphate buffered saline at 37°C for 3 hours.
4. The resulting mixture is filtered through a 250 μm sieve and the fluid centrifuged at 3000 rpm for 3–5 minutes.
5. The supernatant fluid is discarded and the deposit re-suspended in a drop of the fluid. Examine the re-suspended deposit with a 'hanging drop' or on a glass slide with ×60 and ×100 objectives for the presence of motile microfilariae.

Method 2

A wedge biopsy is taken of affected skin and placed in a Petri dish with saline and incubated at 37°C for 15–30 minutes.

The microfilaria may be seen under a low power microscope trying to escape from the biopsy specimen.

Interpretation

Live larvae are very easy to identify from the movement they cause in the drop or on the slide.

Occasionally live microfilariae can be found in the circulating blood – examination of buffy coat smears may be helpful (Fig. 3.33).

A positive larval finding from the biopsy site must be evaluated with the clinical signs shown by the horse. A diagnosis can be confirmed where the clinical condition of skin lesions with pruritus is substantiated by the presence of larvae in the biopsy sample.

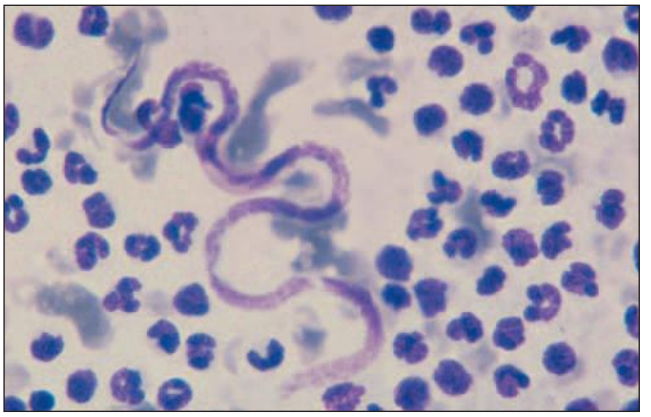


Figure 3.33 Microfilaria in the buffy coat of blood – this is particularly unusual.

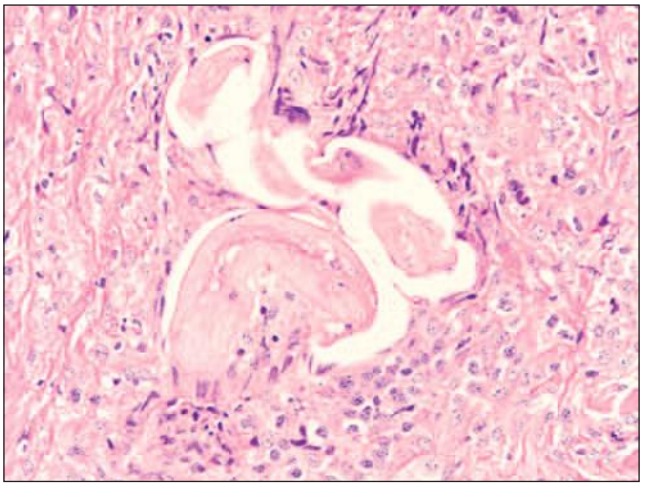


Figure 3.34 A histological section from a case of onchocercosis (see p. 222, Fig. 9.45AB).

Histopathological detection of the larvae is rather more difficult and they can easily be missed. However, if one or both is positive the diagnosis can be considered to be safe (Fig. 3.34).

Improving the diagnostic value of skin biopsy

The critical factor here is that the pathologist must be supplied with an appropriate typical specimen and all the correct information; the best results are obtained when a team effort is applied.

- The owner provides an honest and straightforward history.

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UNIVERSITY OF LIVERPOOL
DEPARTMENT OF VETERINARY PATHOLOGY

Request for:

Necropsy ☐
Surgical Pathology ☐
Cytology ☐

For hospital use only

University Clinician .....
University Hosp. No. .....
Insurance Co. request? Y/N

For laboratory use only

Pathology Lab. No. .....
Histology Ref. No. .....
Vet. Code .....
Date Received .....
Date of Necropsy .....
Interval between death & necropsy .....
Tissue(s)

Photographs

Owner ..... Ref. No. .....
Owner's address .....
Species ..... Breed .....
Age ..... Sex ..... Body Weight .....
Date & Time of death/euthanasia/surgery .....

Referring Veterinary Surgeon .....
Practice Name & Address

Tel. No. Fax. No.

Clinical Features (including anatomical site of surgical specimen)

Previous Path. Lab. No.

Differential Diagnosis/Clinical Impression

Please remember - it is a legal requirement that all specimens must be securely packaged
Please send samples to: Department of Veterinary Pathology, University of Liverpool, PO Box 147, Liverpool L69 3BX
Tel 0151-794-4265, Fax 0151-794-4268

Figure 3.35 A suitable submission form to accompany any surgical specimens submitted for histological or cytological examination.

- The veterinarian carries out a full clinical examination and records the findings carefully for the pathologist (Fig. 3.35). The type, distribution and number and extent of the lesions is stated (a photograph is helpful).
- The correct samples are taken and handled/submitted correctly in timely fashion.
- The pathologists handles the specimens carefully and interprets the findings correctly.

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Regrettably some clinicians seem to prefer to offer the challenge to the pathologists by providing misleading or incomplete information or by failing to provide anything at all! There is an unhelpful concept that the pathologist can and will enable a diagnosis to be made because the patterns of disease are always 'distinctive and pathognomonic'. This is absolutely not the case. The pathologist needs to become an integral part of the diagnostic team and he/she cannot provide a comprehensible and helpful report based on misleading or absent information.

Selecting the best biopsy site and maximizing the return

1. The best biopsy is obtained by careful selection of the site.
2. Select a truly typical lesion free of secondary changes or previous trauma/interference.
3. Try to harvest the whole lesion if a bulla or vesicle is found.
4. Select an area with a piece of ostensibly normal adjacent tissue – this is not always very important; it is far better to harvest a truly typical piece of the lesion and collect a piece of normal skin from an adjacent site.
5. Sample all the variations that are present – some may be secondary.
6. Obtain multiple biopsies, particularly if an autoimmune condition is suspected.
7. Do not clip or scrub and try to avoid intralesional local anaesthetic drugs – use a regional or localized remote block.

Information that should be supplied to the pathologist

- Veterinarian's name and contact details
- Owner's name, address (helpful also for follow-up reports)
- Name and signalment of the horse
  - Age, breed/type, sex, colour
- Clinical features and clinical history
  - Concurrent related or unrelated disease problems
  - Any evidence of systemic involvement or localized skin condition
  - Seasonality/recurrences under defined conditions (transient or chronic)
  - Pruritic/non-pruritic
  - Type of lesion (macule/papule/vesicle/ulcer, etc.): age of lesion; single or multiple; anatomical site, relation to tack, saddle
  - Evidence of contagion or similar signs in other horses under the same management
  - Suggested clinical differential diagnoses
  - Responses to previous treatment
  - Confirmation that the lesion submitted is typical.

The more information that is provided to the pathologist in terms of history and clinical signs the better the likely outcome. There is merit in supplying detailed close-up photographs of skin lesions before and after the biopsy with the documentary notes so that the pathologist can see exactly where the biopsy was taken from; sometimes the pathologist can even be present and can then select the sites most likely to be diagnostically useful. In this way the pathologist makes a meaningful contribution to the diagnosis. He/she might also come to appreciate the difficulties that the clinician faces when selecting material to sample. By the same token clinicians could usefully spend some time with a pathologist to gain an improved understanding of the limitations they face also.

The decision to obtain tissue samples (of any sort) is only the first step in the process. The specimens need to be taken in the correct way, handled in the correct way and then processed correctly. These technical aspects do require some thought and careful sampling will always yield better, more meaningful results.

Helpful aspects of tissue sampling include:

- Samples submitted should be as large as possible.
  - Minute samples are frequently non-diagnostic. However, it is not always possible to obtain large specimens (e.g. corneal biopsy).
- Inclusion of a normal edge or a sample of unaffected skin from an adjacent area is helpful but this should not be extensive – it is better to collect a bigger area of abnormal tissue and a smaller area of normal than vice versa.
- Careful biopsy technique.
- Correct handling of the samples to avoid artefactual damage to the tissue (see Table 3.4).
- Selection of the correct fixative (usually for most routine circumstances 10% formalin is the best option) and the correct volume of fixative (at least 10 times the volume of the tissue).

In return for the clinical effort, the pathologist needs to provide a full report on the findings including details of any histological changes (Fig. 3.36). The report should be as helpful as possible to the clinician and to another pathologist. Whilst many clinicians will actually only be interested in the 'bottom line' (diagnosis!) it is very important that this is supported by credible histological information that can be interpreted by another pathologist should this become necessary. The pathologist's report must be signed and dated (Fig. 3.36).

The clinician can reasonably expect:

- a comprehensive and comprehensible report (properly signed and dated) covering the pattern/distribution of the pathological changes, the cellular elements involved and a morphological diagnosis
- a suggested diagnosis (whether this agrees with the clinical impression or not) with a constructive comment on the rule-outs that were suggested by the clinician on the submission forms
- information on the possible cause and the likely prognosis.

In the case of neoplastic lesions the pathologist will usually provide:

1. a diagnosis or a presumptive diagnosis along with a morphological description of the lesion that can be understood by another pathologist
2. some indication as to the adequacy of excision (tissue margins)
3. the prognosis and any reported information on the clinical behaviour of the tumour type that is likely to be helpful in terms of prognosis.

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Pathology Lab. No Sp97-655

DEPARTMENT OF VETERINARY PATHOLOGY, UNIVERSITY OF LIVERPOOL
SURGICAL PATHOLOGY REPORT

Clinical Accession No: 15894 **Received:** 21.4.97
Species: Equine **Breed:** Thoroughbred **Date of Surgery:** 21.4.97
Age: 5yrs **Sex:** M(N) **Reported Typed:** 30.4.97 (JB)
Owner: [redacted] **Tissue:** Skin (X 3)
University Clinician: [redacted] **Photograph:** Skin surface and cut surfaces
Referring Veterinary Surgeon: [redacted] **Body Weight:**

Clinical Features:

Non-painful, non-pruritic, non-ulcerated lesions present since winter. Approx 25-30 dermal "lumps"/nodules over saddle area, thoracic wall and neck. Lumps developed over 4-8 weeks. (Also sarcoids on abdominal wall, thigh and viral papillomas on nose).
Sample marked 1 has small 'pore' (A).
? Nodular collagen necrosis; insect bite reaction; folliculitis/staphylococcal infection with scarring.

Histology reference: H646-97 **Previous Path Lab. No(s):**

MACROSCOPIC

A. Circular skin biopsy (7mm diameter X 5mm) with grey, haired surface in the centre of which is a pale hairless area (2mm diameter) with a central small pore. Cut surfaces are tough and pale with a central gritty yellow miliary focus adjacent to epidermal pore. Sections X 3 (NO RESERVE).

B. 2 similar skin biopsies with intact epidermal surfaces. Cut surfaces are tough and white with central gritty yellow foci. Sections X 6 (NO RESERVE).

MICROSCOPIC

All the sections are basically similar. Intact epidermis and unremarkable adnexae. Throughout superficial and deeper dermis are multiple nodular aggregations of inflammatory cells: macrophages, a few multinucleated giant cells, lymphocytes and eosinophilic leukocytes. In the centres of some are necrotic and calcified debris. In adjacent dermis there is collagen degeneration and focal mineralisation. Eosinophilic leukocytes are conspicuous in the tunica intima of a deep dermal vein. There is an implanted hair shaft fragment in one of the necrotic foci. Adjacent to one of the areas of degenerate collagen there is a zone of dermal fibroplasia and capillary activation.

Comment: The histological appearance is of multifocal necrotising and calcifying, eosinophilic granulomatous dermatitis, with dermal collagen degeneration. No evidence of cause is recognised.

[redacted] MA, PhD, BVSc, MRCVS, FRCPath, Dip1ECVP 25.4.97
(Telephone report: 1345; 25.4.97)

Figure 3.36 An example of the ideal type of report returned to the clinician.

When a pathologist reports that the tumour extends to the margin of excision, this is a helpful piece of information for the clinician; it is not the pathologist's fault that the tumour was not excised completely and neither usually was it the clinician's. This finding means that extra measures may be required or at least the prognosis adjusted accordingly. In any case the owner of the horse should be told immediately so that there is no future scope for recriminations.

Whilst some suggestions can be made on treatment there are significant dangers in pathologists proffering advice to

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VETERINARY SURGICAL BIOPSY FOLLOW-UP REPORT
Department of Veterinary Pathology
University of Liverpool
P.O. Box 147
LIVERPOOL L69 3BX

Dear .....

We examined and reported on the following specimen from your practice and would be grateful if you would fill in the follow-up sheet and return it to the above address as soon as possible. A stamped and addressed envelope is included for your reply.

Lab. Reference ..... Your Reference .....

Species & Breed .....

Specimen .....

Date received .....

Diagnosis .....

Follow-up

Alive or dead? .....

Unable to trace .....

Destroyed (Give reasons. If animal was not destroyed in your practice, do you know where it was destroyed?).

.....

.....

Was a post-mortem performed? .....

Figure 3.37 A good example of a simple 'follow-up' approach that can be made to the clinician. This may stimulate the clinician also to approach the client for information that helps all parties.

clinicians. It is an unfortunate truth that many times treatment is suggested that is either totally impractical or inappropriate. For example, 'wide surgical excision should be curative' when the lesion involves the whole upper eyelid. Similarly opinions of generality that are given for very rare, or even once in a lifetime cases, such as 'These very rare tumours usually respond to radiation treatment or wide surgical excision'. Whilst any information is helpful the clinician must interpret it with care and the pathologist should construct the report with similar care to avoid giving impractical or unproven, gratuitous advice.

One of the most neglected aspects in equine dermatology is the concept of 'follow-up'. The presumptive diagnosis or simply the pathological information may be helpful or it may not. The pathologist and the clinician need to cooperate to encourage dialogue and follow-up to establish whether the diagnosis led to a successful treatment or to failed treatment. Follow-up is often performed by pathologists in an attempt to improve their services and their knowledge. This can only help the clinician. Professional satisfaction and progress in knowledge would be much helped by having follow-up information at relevant times after the diagnosis and treatment have been provided (Fig. 3.37).

Clinicians and pathologists can and should cooperate to get the maximum value from biopsy of equine skin lesions. This will lead to improved diagnosis and management in individual cases, and the development of confidence and competence through shared experience of cases over time. The concept of evidence-based medicine must be the objective and only by accumulation of accurate data will progress be made. The value of a cooperative team effort cannot be overstated.

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SPECIALIZED/OTHER DIAGNOSTIC TECHNIQUES

Wood's lamp

The Wood's lamp test is a classically described method for the identification of some dermatophyte infections. The method relies upon a strong source of ultraviolet light which causes the dermatophyte spores to fluoresce a characteristic green colour. Although the test is seldom applied to horses because only *Microsporum* species (with their ectothrix spores) will fluoresce and because most dermatophytosis cases are due to *Trichophyton* spp. (which do not usually fluoresce due to their endothrix spores), there are some important principles to follow when using this method.

1. The lamp must be in good working order and must be allowed sufficient time to warm-up; it may take up to 15–20 minutes.
2. The examination must be performed in as near total darkness as possible.
3. Some dermatophytes only start to fluoresce after some minutes of exposure so a casual wave of the lamp is not helpful.
4. Individual hairs must be seen to fluoresce – suspicious hairs can be plucked out and examined directly under the lamp.
5. Some medications, soaps and antibiotics will also fluoresce strongly so prior medication is often not helpful. Other chemicals such as iodine applications remove the florescence.

Restriction/provocation tests

In cases where a feed allergy or reaction is suspected it may be useful to attempt to identify the cause by use of an exclusion/provocation test. Alfalfa or ryegrass hay is generally regarded as the starting point (although whether they could be regarded as hypoallergenic is debatable). The horse should be fed exclusively on a single dietary substance for 2–4 weeks (but it may be longer). Once the basic diet substance is tolerated and standardized, individual other components including the suspected material can be added at 2-week intervals. It is important to realize, however, that there are few recorded cases of confirmed diet-related skin disease. Environmental and other factors may need to be considered and the overall complexity of the problem can be very difficult to sort out.

Immunofluorescence

Tests using these techniques are available in specialized pathology laboratories. Some pathologists question the value of direct and indirect immunofluorescence methods in horses and prefer to rely on characteristic conventional histopathological changes. Biopsy samples should be placed in appropriate fixatives after discussion with the pathologists. In the absence of any guidance each biopsy should be divided into two and placed both in 10% formal saline and in Michel's solution (Evans 1992).

Immunocytochemistry

Increasingly sophisticated immunological methods are being applied to diagnostic processes. The availability of specific (monoclonal) antibodies has revolutionized diagnosis of many skin disorders in humans and other species. Although there are limited such opportunities in horses, the techniques are sometimes useful. Currently it is most valuable in the differentiation and confirmation of B- and T-cell lymphomas.

The autoimmune conditions can be confirmed using these methods with fluorescent antibody techniques.

Antinuclear antibody (ANA) testing

The antinuclear antibody (ANA) titre and lupus erythematosus (LE) cell preparation may assist in the diagnosis of equine systemic or cutaneous lupus erythematosus-like syndrome of horses (E-SLE). This test was originally developed to diagnose LE, but it may give positive reactions in a number of other conditions. It is a test performed on serum and detects antibodies directed against nucleolar DNA. High ANA titres (exceeding 1:160) are indicative of immune-mediated disorders. A high titre in combination with multisystem clinical signs is highly suggestive of a positive diagnosis for SLE (Swiderski & McLure 1995). While neither test is specific for SLE, a positive result serves to indicate a possible autoimmune dysfunction. Although the ANA test is the best test available for SLE (Fadok & Greiner 1990) there is some doubt as to its relevance in equine medicine because some cases which present all the clinical features of the diseases fail to show any detectable ANA antibody and others may develop the antibody, particularly subsequent to drug administration, without showing any cutaneous or systemic signs.

Serum (10 mL) or freshly clotted whole blood (20 mL) is required. It is probably wise to consult with the laboratory regarding the collection of the sample as some do not perform the test.

Allergy testing

Intradermal (in-vivo) skin testing

Intradermal skin testing in horses has been used in a very limited way. It requires the availability of specific allergens and experienced personnel to conduct and read the test. It should be read and interpreted in the light of the history and general examination of the horse. False negative and false positive tests do occur and some horses even show detectable reaction to the blank test sites. Clinically normal horses can also show marked positive skin reactions, giving a misleading impression of the relevance of the test. However, most dermatologists regard skin testing as the ultimate test of atopic disease (Littlewood 1997) but this condition is probably rare in the horse anyway.

Prior to skin testing the patient should not have been treated with drugs such as corticosteroids, antihistamines,

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tranquillizers or anaesthetics and non-steroidal anti-inflammatory drugs – a drug-free period of at least 6–10 days is advisable. Fractious animals can be safely and effectively sedated with an α_2-adrenoreceptor agonist (such as xylazine, detomidine or romifidine) immediately prior to the procedure. Phenothiazine ataractics (such as acepromazine) may have a significant suppressive effect on the test.

Many substances such as grass, tree and weed pollens, moulds, stable dust, dust mites, feathers and dander have the potential to cause inhalant allergies resulting in atopic pruritus and may be detected by intradermal testing.

Method

1. Two days before the test is to be performed, the hair over the lateral neck is clipped (avoiding any areas with obvious lesions) and gently washed with a bland soap. The area prepared depends on the number of agents to be tested. Each will need an area of about 3–4 cm radius to avoid possible overlapping reactions.
2. One day before the test, 0.05 mL of 1:100 000 histamine phosphate solution is injected intradermally into a site just outside the test area. Normally a wheal approximately 1–2 cm in diameter should develop within 30 minutes. No response suggests that the horse is 'non-responsive' and the test should be postponed until a reaction can be elicited by this test. If an inordinate reaction develops the procedure is probably unwise.
3. Prepare all reagents required, including emergency adrenaline. Fortunately, untoward reactions in the horse (anaphylaxis or urticaria) are very rare.
4. The full required range of allergens is reconstituted. Lyophilized allergen panels are available commercially. Once reconstituted they must be refrigerated and used within 3 months. It is not wise to use compounded (multiple) allergens containing several different substances as this fails to provide the narrow diagnosis which is required for subsequent desensitization.
5. The sites for the injections are identified by a non-irritant, indelible felt-tip marking pen and numbered (either on the animal or on a prepared 'map' of the site).
   - If palpable variations in skin thickness are present, the thickness of the skin at each site (as measured by dermal calipers) should be recorded.
   - Tuberculin testing calipers are crude but effective.
   - More accurate mechanical or electronic skin testing calipers are available.
6. Small amounts (usually 0.05 mL) of the various suspect allergens are then injected intradermally using a 25/26G 10 mm (intradermal) needle and a 1 mL tuberculin or insulin-type syringe.
7. Histamine (positive) and physiological saline (negative) controls are also used.
8. The extent of reaction at each numbered and identified site is measured (using skin testing calipers) and recorded after 15 and 30 minutes and at 4 hours after injection (Fig. 3.38). Reaction to allergens can then be compared with the two control sites.

Interpretation

A positive result is indicated by swelling at the site of the injection.



Figure 3.38 The appearance of a multiple allergen skin test performed in a horse some 3 hours previously. Note the positive oedematous nodular reactions shown as swellings at the site of the intradermal injection. The control positive and the negative sites show little or no reaction.

By convention a significant response is taken to be a swelling greater than, or equal to, twice the average increase between the negative and positive controls. A positive response does not necessarily correspond with pathological allergy but when considered with the clinical syndrome and the history it can be a useful additional help. Furthermore, the extent of the positive reaction is not necessarily proportional to the severity of the allergy.

There is little correlation between the histological changes occurring at the site of the tests and the clinical responses. At present interpretation should be made carefully. However, continued testing of cases may eventually provide a better defined panel of allergens for horses and stimulate further research into allergen vaccinations – currently only a few are available.

Note

Allergen neutralization testing and therapy involves the use of serial dilutions of the allergen to establish a dilution at which the allergen fails to induce a reaction in the skin. This is, somewhat surprisingly, often some way between the highest and lowest concentrations which show significant reactions. This dilution is then used by subcutaneous injection to 'neutralize' the allergic response. Good results have been claimed in the human and with some equine respiratory allergies, but the value for dermatological responses to inhaled or ingested allergens in the horse has yet to be established.

In-vitro allergy testing

Commercially available tests on serum use either the radioallergosorbent test (RAST) which employs radioimmunoassay with isotope-labelled antisera, or an enzyme-linked immunosorbent assay (ELISA) which uses antibody coupled to enzymes.

The 'extent of allergy' to an inhaled or ingested allergen is supposedly correlated with the amount of allergen-specific antibody (IgE) present in the serum sample. There have been insufficient published reports and results to allow true evaluation of its value in equine dermatology at the present time.

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The responses may be very different in contact allergies. Very careful evaluation of results is therefore necessary. There is little standardization between individual laboratories and so results can vary widely and indeed the results obtained from the same animal on different occasions can be very different. Therefore, both RAST and ELISA methods, while offering some extra information, must be interpreted very cautiously in the horse (Fadok 1995).

Method

1. Serum (10 mL) or freshly clotted whole blood (20 mL) is required for either testing procedure.
2. A full history and supportive information on possible exposures and treatment responses achieved must be provided. There is little advantage in testing horses that are concurrently under treatment with corticosteroid drugs.

Interpretation

The tests must be interpreted with considerable care. The implications from a positive test can encourage the use of vaccines and energetic avoidance strategies but given the restricted panel of IgE antibodies that are available specifically for horses, it is entirely possible that the 'positive' horse is responding to an allergen that has not been used in the test. Thus, efforts to use either avoidance strategies or vaccines may be an expensive waste of time. Where they work, however, there is some detectable benefit. Desensitizing vaccines alone are seldom if ever capable of resolving complex allergic conditions – avoidance of the allergen is clearly the best (though sometimes the most difficult) of all therapeutic options.

References

Dewes H F 1972 *Strongyloides westeri* and *Corynebacterium equi* in foals. New Zealand Veterinary Journal 20:82–84

Evans A G 1992 Dermatophilosis – diagnostic approach to non-pruritic, crusting dermatitis in horses. Compendium of Continuing Education for Veterinary Practitioners 14:1618–1623

Evans A G, Stannard A A 1986 Diagnostic approach to equine skin disease. Compendium of Continuing Education for Veterinary Practitioners 8:652–660

Fadok V A 1995 Overview of equine pruritus. Veterinary Clinics of North America, Equine Practice 11:1–9

Fadok V A, Greiner E C 1990 Equine insect hypersensitivity: skin test and biopsy results correlated with clinical data. Equine Veterinary Journal 22:236–240

Gibbs E P J, Smale C J, Voyle C A 1980 Electron microscopy as an aid to the rapid diagnosis of virus diseases of veterinary importance. Veterinary Record 106:451–453

Grant D I 1991 Fungal skin disease. In: Skin diseases in the dog and cat. Blackwell Scientific, London, p 54

Littlewood J D 1997 Diagnostic procedures in equine skin disease. Equine Veterinary Education 9:174–176

Logas D B, Barbet J L 1999 Examination of the skin. In: Colahan P T, Mayhew I G, Merritt A M, Moore J N (eds) Equine medicine and surgery. Mosby, St Louis, p 1797

McGavin M D, Fadok V A 1984 Factors limiting the usefulness of histopathological examination of skin biopsies in the diagnosis of large animal dermatoses. Veterinary Clinics of North America, Large Animal Practice 6:203–213

Martens A, De Moor A, Ducatelle R 2001 PCR detection of bovine papilloma virus DNA in superficial swabs and scrapings from equine sarcoids. Veterinary Journal 161:280–286

Scott D W, Miller W H Jr 2003 Hair examination. In: Equine dermatology. WB Saunders, Philadelphia, pp 85–88

Song M D et al 1991 Hair follicle cycling in the mane and tail region of horses. American College of Veterinary Dermatology 7:18–25

Speed J G 1960 The importance of the coat on Exmoor and other mountain and moorland ponies, living out of doors. British Veterinary Journal 116:91–99

Swiderski C E, McLure J J 1995 Immunodiagnostic assays. Veterinary Clinics of North America, Equine Practice 11:455–489

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Principles of dermatological therapeutics

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The treatment of skin disease provides many challenges for the clinician. These include the type of disease and the practicality of many of the normal therapeutic options when used in such a large animal. As with all treatment protocols, in selecting a treatment method the clinician should consider its likely benefits and any possible undesirable consequences; most medications that have at least one benefit (usually the therapeutic effect) will also have at least some potential for harm (the side- or unwanted effects). Thus, a surgical option might present itself, but the resulting wound might be impossible to close, or the resultant scar might cause significant functional problems that may be more serious than the condition itself. For example, a sarcoid in the upper eyelid or on the coronary band would be extremely difficult to remove surgically or treat with a necrotizing topical cream without some functional deficit. The treatment in such cases may be far worse than the condition. A much more careful choice of treatment would almost certainly have less secondary effect (Fig. 4.1). Of course, the pressures of practice often mean that owners want a 'cheap, quick fix' and are often intolerant of a more gentle approach or a more expensive option.

The use of corticosteroids in the management of a case of insect bite hypersensitivity (IBHS/sweet itch) would probably be contraindicated if the animal had a history of laminitis or was of a type that might make it susceptible to laminitis. These difficulties simply emphasize the need to consider all aspects of the case before selecting any particular treatment. The conflicts between treatment required to manage a disease condition and the potentially dangerous side-effects are a persistent challenge to veterinarians. Fortunately if all the proper criteria are employed for selection of a treatment modality, the number of cases suffering from side-effects is usually very low, and if the treatment is withdrawn because of an inherent minute risk, treatment would not be possible for almost any disease.

There is no evolutionary advantage in disease and so in normal circumstances the natural reparative mechanisms are deployed to attempt repair. Notwithstanding the therapeutic benefits of drugs they cannot substitute for a healthy immune and defence system. A healthy body is less inclined to disease and more inclined to heal well. The importance of a healthy diet and lifestyle is often overlooked in the management of disease. Simple nutritional management can help significantly in maintaining a healthy skin and hair, preventing disease, and supporting recovery from disease of any type. The benefits of omega oils and vitamins and minerals on skin health are well known; a healthy well-balanced diet is usually reflected in a healthy skin that is highly resistant to opportunistic pathogens. Nutritionally deprived and immunocompromised horses will always have greater susceptibility to disease and more difficulty with any therapy than healthy horses fed a healthy diet. The concept of nutritional deficiency and the requirement for supplemental vitamins, minerals and oils, etc. has become almost standard practice in horse management. The truth is, however, that the vast majority of healthy horses are very tolerant of minor and short-term deficiencies and will remain healthy even in the face of general or specific nutritional deprivation. Over-enthusiastic supplementation with any nutrient component can result in significant imbalances and this applies as much to the skin as to other organs and structures. For example, selenium supplementation is common but overdoses can lead to severe and even life-threatening disease. Unfortunately in many parts of the world, vitamin and mineral supplements, herbal remedies and feed additives that purport to 'enhance' immunity and resistance to disease are advertised in such a way that most horse owners are drawn to their use regardless of the actual value and regardless of the possibility of harm rather than benefit. Indeed most such supplements are simply a waste of money. Some can be harmful rather than beneficial.

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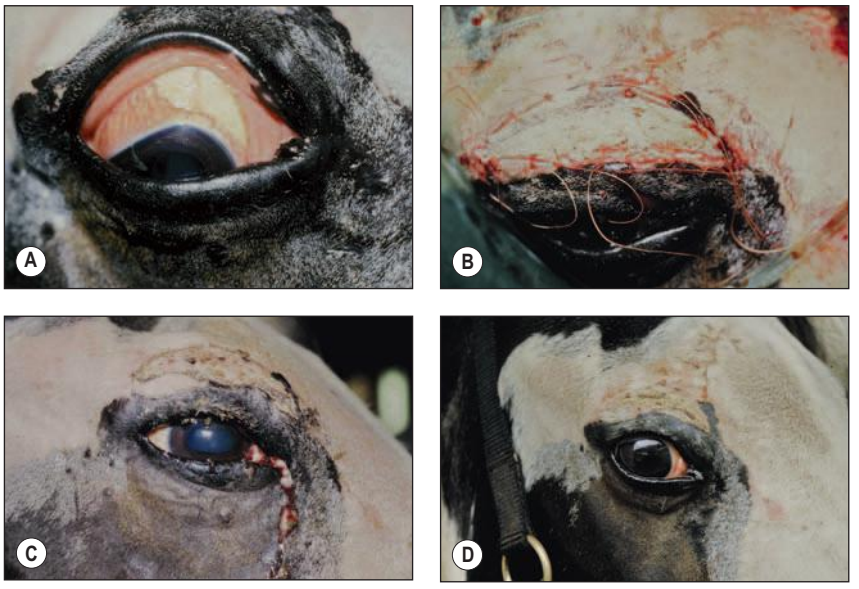


Figure 4.1 This coloured gelding was subjected to surgical removal of a type B nodular sarcoid on the central part of the upper eyelid. Scarring and wound contraction resulted in a severe functional disability that necessitated extensive cosmetic surgery involving full skin grafting. **(A)** Pre-surgery; **(B)** 9 days post-surgery; **(C)** 21 days post-surgery; **(D)** 4 months post-surgery. Although the ultimate outcome was very satisfactory, a more rational initial treatment would have been a better option. It might even have been better to leave it alone, rather than interfere in an inappropriate way.

Drugs are commonly used to treat skin disease but many have significant undesirable consequences, some of which may be life-threatening if they are used inappropriately – errors of overdosing, under-dosing, incorrect dose interval and inappropriate drug selection all have potential effects. For example, the use of a corticosteroid to treat a minor skin disorder in a pony with a tendency to laminitis may precipitate a catastrophic acute laminitic episode. Although corticosteroids have a generally bad reputation in horses this is possibly unfair because the risks of their administration in a horse that has no tendency to laminitis are very low – the benefits can, however, be considerable. Amitraz is a common ectoparasiticide for use on cattle and dogs but it has serious toxic effects on horses resulting in intestinal ileus.

Evidence-based medicine is a critical issue in modern veterinary practice. This approach takes account of properly conducted trials on treatments and management procedures and provides the best clinical information for the practitioner. The client expects the best approach to be taken and the practitioner needs to be aware of what that is. The options for treatment that owners can seek are becoming much wider with the information age and in many cases they seek what looks easy, cheap and convenient. In many cases the reality is the reverse; an attractive advertisement extolling the virtues of an untried material with no clinical supporting data on efficacy and safety can tempt a gullible and vulnerable owner, especially when the package price seems attractive and the promises of success are impressive. Even depriving an animal of proper and timely treatment could be construed to be negligence. The skilled clinician is able to keep up with current developments, to critically appraise published papers and analyse the results of trials and reports so that the patients can expect the best available advice and treatment. Almost no client-led litigation has taken place when the owner has been properly informed by a qualified veterinarian of the benefits and disadvantages of using a particular therapeutic strategy – regardless of its complexity or simplicity and regardless of the outcome.

The available therapeutic options available in any particular part of the world will necessarily vary and clinicians will need to be aware of the proprietary names of the therapeutic agents and compounds mentioned below. Regulations concerning the residues in tissue may be very important in some regions where equidae may be used for human food. However, every effort should be made to use the most effective treatment method for the specific condition at the outset. There is little more wasteful use of resource than using a treatment which does not work; whether this is an expensive or cheaper option is largely irrelevant. Time is wasted and the condition might get worse in the interim or the drug might have unwanted side-effects which make the case even more difficult to manage. It is the efficacy and safety that matter.

Possible reasons for failure of treatment to resolve the problem must be sought when it is clear that treatment is not proving effective. When therapy fails, a complete review of the case notes can help – also, sometimes the failure of a treatment or unexpected resolution can help to clarify the diagnosis. However, when treatments fail, there is usually a reason.

1. The diagnosis may be wrong and therefore the selected treatment is inappropriate. In dermatology this is a relatively common event because many of the clinical symptoms are representative of several very different disease conditions.
2. The diagnosis may be correct but the wrong treatment is used.
3. The diagnosis is right and the treatment selected is correct but it is being applied wrongly. As most dermatological therapy relies heavily on owner and patient compliance there is scope for further problems to develop. It is vital

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that data sheets for all the drugs are read and re-read to ensure that the best possible information is available regarding side-effects and drug conflicts.

4 The condition is untreatable. Under these conditions treatment is palliative at best but a worsening of the disease situation can occur if inappropriate drugs are used. It may in some circumstances serve the animal best if nothing is done at all.

5 The patient is not cooperative. Some treatments are painful or uncomfortable and application can either be compromised or in some cases is impossible. Even oral medications with steroids or antibiotics can be problematic. Many drugs work best when short intervals between doses/applications are made but the ideal circumstances may simply not be possible.

6 There is failure of owner compliance. The owner may be unable or unwilling to undertake the treatment for any one of a number of valid and invalid reasons. Sometimes there is a perception that the drugs being offered are 'harmful'. This applies in particular nowadays to corticosteroids. Although there is no scientific evidence that laminitis is an inevitable consequence of corticosteroid administration, a number of high profile cases have given this misconception some credence. Recent research confirms that the risks are very low but that certain types of case and certain types of horse may be more liable to complications.

Some owners fail to grasp the importance of following the instructions implicitly – this may lead to over- or under-dosing or the use of over- or under-strength topical medications. The owner may believe that the condition has resolved and so withdraw medications before the full effects can develop and some may stop the treatment before the full benefits are seen, believing that the treatment is not proving effective.

Pharmaceuticals described later in this chapter refer specifically to their use in dermatological treatment in the horse. However, many of the compounds have not been researched or tested effectively in horses – there is much anecdotal information but little evidenced-based guidance in equine dermatological therapy. Data extrapolated from other species may be useful in some cases, in others it may be less so. Furthermore, some drugs are not available or have licensing problems in many individual countries. Where specific licensing problems exist the reader is advised to check carefully with the regulations in force in his or her locality to avoid any problems with the use of unlicensed products. New drugs are being developed and others are being withdrawn from the market for safety or commercial reasons (or both) and the list given here cannot therefore be complete.

Generic drug names are used throughout in this text.

Anti-infective pharmaceuticals used in dermatology

Antiseptics/disinfectants

Many of the modern antiseptic compounds have good virucidal, bactericidal and fungicidal effects and some have useful sporicidal effects for spore-forming bacteria and fungi. The commonest compounds used in equine dermatology and wound management include chlorhexidine, povidone-iodine, iodophor, quaternary ammonium compounds, halogenated tertiary amines, inorganic halogenated peroxygen compounds and acidic iodines.

Some antiseptics and disinfectants can be used on the skin but others are harmful. There is no justification for applying chemicals to the horse's skin that are not specifically designed for topical application. Manufacturers' instructions should always be followed implicitly. The antiseptics are variously used for their antibacterial properties prior to surgical interference in aseptic surgery and also as wound antiseptics. Most compounds will have some harmful effect on the skin when used in abnormally high concentrations and the specific dilution instructions are vitally important. In any case, many of the iodine-based compounds work more effectively at lower concentrations than at the higher ones and it is very important to ensure correct dilution.

Most agents will have less or more activity against various microbes and the correct selection will enable a more rapid resolution or effect to be gained. Thus, chlorhexidine probably has less antifungal activity than povidone-iodine and some of the newer quaternary ammonium compounds and halogenated tertiary amines have a very wide range of activity against viruses, bacteria and fungal organisms but some are irritant if applied to the skin. Care must be taken to read the labels to check for the range of activity and the value and dangers of application to the skin. Some agents are used solely as environmental disinfectants in order to reduce the microbial challenge to animals.

Chlorhexidine diacetate/gluconate A phenol-related, biguanide antiseptic-disinfectant with few problems and no apparent systemic absorption or local effects. It has a wide range of antibacterial activity and probably has better control over *Staphylococcus aureus* than povidone-iodine. It has little or no fungicidal effect except at high concentrations and is used primarily as a surgical scrub and as an antiseptic solution (in alcohol).

The presence of pus or other organic material does not diminish its activity and it has good tissue properties with some residual effects in tissues, providing prolonged activity.

Routine use of 0.5% is recommended but, as a wound irrigation solution concentrations of 0.05% should be used. It is important to dilute the concentrate with water rather than saline or other electrolyte solutions which may cause significant precipitation of the compound in solution and consequent loss of effect.

Povidone-iodine A non-irritating, non-staining, iodophor which comprises iodine solubilized by surface-active agents. It has a broad spectrum of activity with virucidal, bactericidal (and sporicidal) and fungicidal effects which are not affected by blood, serum, necrotic tissue or pus. The rapid bactericidal effects are better at lower dilution – higher concentrations result in reduced bactericidal effects and greater tissue toxicity. The mild tissue toxicity effects are minimized by dilution to less than 0.05%. Solutions of 5% or more actively inhibit leukocyte migration into wounds, while very weak solutions appear to encourage neutrophil and monocyte migration. One per cent solutions cause death of fibroblasts but 0.01% has no detectable harmful effect on these cells.

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Acetic (and malic) acid Natural highly acidic chemical solutions which are widely marketed as wound irrigants. They are effective in the control of *Pseudomonas* spp. infections on skin and their use should be restricted to old wounds infected with this organism and use as a debriding agent. The solutions should not be applied to a fresh wound or wounds without *Pseudomonas* spp. complications as they cause severe deleterious effects on all cells associated with wound healing.

Hydrogen peroxide Available in various dilutions, it has been used widely for its disinfectant properties. It has only weak germicidal effects but an impressive foaming and deodorizing property as a result of liberation of nascent oxygen. The oxygen release can be employed usefully in the control of deep anaerobic infections. Concentrations higher than 5 volumes (v/v) are damaging to tissues, with worse cytological effects against tissue cells (including fibroblasts) than against bacteria. As the chemical can cause microvascular thrombosis, it can retard healing. It can, however, be effective at higher concentrations in the treatment of thrush in the cleft of the frog.

Hexachlorophene Used primarily in medicated shampoo but it is possibly less effective than many of the other agents and is becoming less available owing to its potential long-term toxicity.

The specific properties of the common agents are described in Table 4.1.

Fumigation sterilization of tack and equipment using formaldehyde gas

This is a very useful technique for sterilizing equipment and tack without damaging it and without the need to handle every single piece of it. Although this method is excellent, modern halogenated peroxygen compounds and quaternary ammonia disinfectants are probably safer and almost as effective.

Although formaldehyde is very dangerous, the method and precautions outlined below make the procedure safer and more effective.

Equipment required

- Protective clothing and mask are obligatory.
- A suitable amount of potassium permanganate (Condy's crystals) (usually about 100 g).
- A suitable volume of 40% (strong/neat) formalin solution (for 100 g of potassium permanganate about 250 mL is required).
- Airtight plastic bags of suitable size.
- Sealing clips for bags.

Precautions

Although the precautions outlined below may initially be daunting, they are sensible. However, the method is really very simple, very practical and highly effective. With reasonable care it is entirely feasible and safe in almost every stable situation.

- All procedures should be carried out in the open air under cover (ventilation is extremely important as the gas is very toxic).
- Ensure that bags are airtight.
- Avoid contact with either permanganate or formalin solution. It is advisable to wear a mask, gloves (preferably up to the elbow), rubber boots and protective waterproof clothing.

Method

1. Place the articles to be sterilized in a large plastic bag.
2. Place a plastic bowl with potassium permanganate crystals (Condy's crystals) into the bag.
3. Pour the 40% formalin solution onto the crystals.
4. Quickly seal the bag.
5. Leave undisturbed for at least 12 hours.
6. Open the bag in fresh air without breathing fumes – plastic bags can be easily split open and left in the open air.
7. Leave the bag open for 12 hours before handling any equipment inside it.

Note

- Although this is an excellent method for disinfecting equipment it can be a dangerous procedure.
- Formaldehyde gas can be seriously toxic to humans if inhaled in quantity or on repeated occasions.
- The strong formalin solution is also toxic and should not be touched.
- Proper protective measures ***must*** be taken at all times to ensure that no human hazard is present.

Antiviral agents

There are few if any specific antiviral agents currently in use in horses. In theory at least some of the new antiviral

Table 4.1 Relative activity of some of the common disinfectant compounds used in veterinary practice (the more stars, the greater the effect)

Agent	Antifungal activity	Antibacterial activity	Antiviral activity	Value as dermatological therapeutic
Chlorhexidine	*	***	–	***
Povidone-iodine	**	***	–	***
Halogenated tertiary amine	****	****	****	Uncertain
Inorganic peroxygen compound	***	****	****	Uncertain
Acidic iodine	**	****	****	Not for topical application

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drugs (such as aciclovir, trifluoridine, ganciclovir and vidarabine) would be expected to have significant benefits on virally induced skin disease and in particular the herpesvirus conditions. Only aciclovir has any equine reputation because the others are both expensive and largely unavailable in the amounts required for horses (both topically or systemically). Aciclovir is currently limited to treatment of putative herpetiform keratitis. Its action is specifically against herpesviruses.

Topical applications of a 5% aciclovir cream can be used on painful coital exanthema (EHV-3) lesions but there is little to be gained by this given that the condition is self-resolving in almost every case. A number of skin lesions including some defined erosive/ulcerative conditions of the lips and muzzle may respond to aciclovir applied topically but the implications of this are not clear. Oral dosing of aciclovir can also be used but no information is available as to its safety or efficacy.

The modern acidic iodine, tertiary amine and inorganic halogenated peroxygen disinfectants have strong antiviral properties but many are highly irritant to the skin and therefore are inappropriate for application. However, some tertiary amine compounds have been formulated into surgical scrub solutions and can therefore be justifiably used on the skin. Sterilizing the skin with powerful antiseptic solutions is not always an advantage but there are some viruses that are significant commensals.

Principles of antibiotic therapy in skin disease – the use of antimicrobials in wound management and skin disease

A fuller understanding of the action and function of antibiotics can be obtained from pharmacological texts. Antibiotics are commonly used by veterinarians in their daily practice – often to treat defined or suspected infections or as prophylaxis for various medical and surgical procedures. Antibiotics do not eliminate infection, rather they reduce the rate of bacterial replication to a degree which allows the host defences to eliminate the infectious agent. The use of antibiotics carries risks of potentially harmful side-effects and therefore they should be used with care.

Potential side-effects include:

- bacterial resistance
- anaphylactic/hypersensitivity reactions including cutaneous urticaria
- delayed drug-related skin eruptions which may occur up to 6 months after administration
- immunologically-mediated systemic effects
- overgrowth of other bacteria or fungal species which may be pathogens that are normally held in check by the commensal microorganisms, or opportunistic pathogens that take advantage of local immunosuppression
- gastrointestinal disturbances deriving from alteration of the delicate large bowel bacterial flora.

Since potential risks are involved in the use of antibacterial and antifungal drugs, it is important to continue asking the following questions to ensure correct and appropriate usage:

1 Is an antibacterial agent really indicated either prophylactically or therapeutically?
2 Should a culture and sensitivity test be performed before embarking on the medication and is there sufficient justification for the inevitable consequent delay in institution of the therapy?
3 Is the chosen agent appropriate for the case and the suspected (or confirmed) condition?
4 What are the correct dose, dosing interval and route of administration?
5 Is its use likely to result in early resistance and are there measures which can be taken to minimize this risk?
6 Is the duration of therapy appropriate and should any monitoring procedures be performed (to assess either resistance of pathogen or harmful side-effects on the patient)?

Prophylactic antibiotic therapy

The use of prophylactic antibiotics is necessary for surgical procedures that carry a significant risk of postoperative infection such as with wounds on the lower limb, in the perineum or around the mouth. Clean procedures are associated with a low risk of postoperative infection and therefore require less than 24 hours of antibiotic coverage. Since in all but surgical skin wounds some infection is likely, elective procedures involving skin wounds should be preceded by at least one full therapeutic dose of a suitable antibiotic. If full aseptic technique can be sustained throughout the procedure (such as in a surgical wound resulting from excision of a small tumour), additional therapy should not usually be necessary. If any break in aseptic technique occurs or the procedure involves complicating factors such as a synovial structure, continued antibiotic administration for up to 5 or more days may be prudent. The aim of prophylactic therapy is to achieve high levels of antibiotics in the skin at the time of surgery. Ideally the concentration of the agent should be 4–8 times the minimum inhibitory concentration (MIC) for the potentially infective bacteria. This requires administration of antibiotics about 30 minutes preoperatively for intravenous dosing and 1–2 hours preoperatively for intramuscular dosing. Care should be taken to ensure that antibiotic administration does not interfere with the anaesthetic protocol (e.g. potentiated sulphonamides should not be used concurrently with injectable α_2-adrenoreceptor agonist sedatives because the combination might induce cardiac dysrhythmias). In addition, the potential systemic toxic effects and the cost of the antibiotic should be considered.

Therapeutic antibiotic medication

Antibiotics are required when suspected or confirmed infection is present. Confirmation is obtained by culture of swabs taken from the site (see above) and subsequent culture, Gram stain and antimicrobial sensitivity test. In the absence of confirmation of an infection, clinical signs of inflammation (heat, pain, swelling, redness) or lack of response to supportive therapy are sufficient to warrant the use of antibiotics. As cultures often take several days to perform, it is often wise to pre-empt the findings by administration of a suitable and logical antibiotic and adjust the drug if necessary when results become available. A clinical judgement of the likely infectious organisms based on experience can usually be made.

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Many factors influence the function and efficacy of the drugs and the clinician needs to consider carefully the whole case and make a reasoned selection. In many cases, however, an informed guess has to be used initially. The main questions that need to be considered are:

- What organism(s) is most likely to be present given the history and cause of the disease?
- What is the *likely* antibiotic sensitivity profile of the suspected bacteria?

Streptococcus spp. are involved in a high proportion of equine skin conditions and most of these are fully or partially sensitive to penicillin. Therefore, it has become common practice to use full therapeutic doses of penicillin as a primary approach to skin antimicrobial therapy. This is entirely justifiable.

Both prophylactic and therapeutic antibiotics in skin disease rely on the delivery of the drug to the skin in therapeutic concentrations. Few antibiotics have been shown, when delivered to the skin of the horse, to achieve an effective minimal inhibitory concentration (MIC) and so careful appraisal of the effects of such therapeutic measures must be taken. There are some antibiotics that are potentially very harmful to horses and so extra care must always be taken to justify the drug selection; there is no point in controlling a skin infection if the drug results in a catastrophic bowel derangement.

The distribution of antimicrobial drugs in the skin depends on its formulation, lipid solubility, state of ionization, protein-binding capacity, route of administration and the delivery efficiency to the site. For instance, a drug that is more soluble in lipid may have better intracellular penetration and therefore prove more effective for use against intracellular bacteria.

The reasons for failure of a detectable therapeutic response to antibiotic therapy include:

1. inappropriate drug selection for the species of bacteria involved
2. inappropriate dose and dose intervals so that the drug never reaches a therapeutic concentration
3. inappropriate drug delivery dynamics/administration route
4. resistant (strains of) bacteria
5. inability of the drug to reach the site due to a compromise in the blood supply or the presence of fibrous tissue or other local problems
6. inactivation of the antibiotic due to enzymatic inactivation or an unsuitable pH in the tissue. Local factors such as the presence of destructive enzymes, acidic pH, low oxygen tension and intracellular organisms can have a marked effect upon the in-vivo efficacy of the drug, even when the in-vitro sensitivity is established.

Culture and sensitivity results allow the use of antimicrobials to which a particular bacterium is most sensitive in vitro. Enzymatic and pH-related inactivation can usually be avoided if abscess cavities are drained or if septic synovial structures are flushed to remove the acidic debris.

Rapid identification of the infecting organism can sometimes be made from a Gram-stained impression smear from the surface of infected skin, but in dermatological disease the heavy normal commensal bacterial flora of equine skin makes the interpretation difficult. Gram staining may only indicate the type of bacteria, whereas a culture will usually provide a positive identification.

Specific organisms involved in skin disease such as *Dermatophilus congolensis* may require specific culture methods and the correct method of sampling (see above) should always be observed so as to avoid errors of omission or commission.

Sensitivity testing by disc diffusion or micro-dilution methods can be used to establish whether the bacteria are susceptible, moderately susceptible, or resistant. New rapid methods of determining sensitivity using genetic technology are being developed in an attempt to facilitate correct selection at the outset of treatment.

The minimum inhibitory concentration is the lowest concentration that inhibits bacterial growth and is calculated from serial dilution of the bacteria grown. The antibiotic dose necessary to achieve four times the minimum inhibitory concentration in the blood can be calculated and administered, but delivery to the site may still be a problem. Even the circulatory and blood protein status can have an influence on the delivery of drugs including antibiotics.

Ideally, choice of antimicrobial agent will be based on culture and sensitivity results. However, the decision regarding immediate antibiotic administration must invariably be based on clinical judgement (Fig. 4.2). This requires an assessment of the location and nature of the condition,

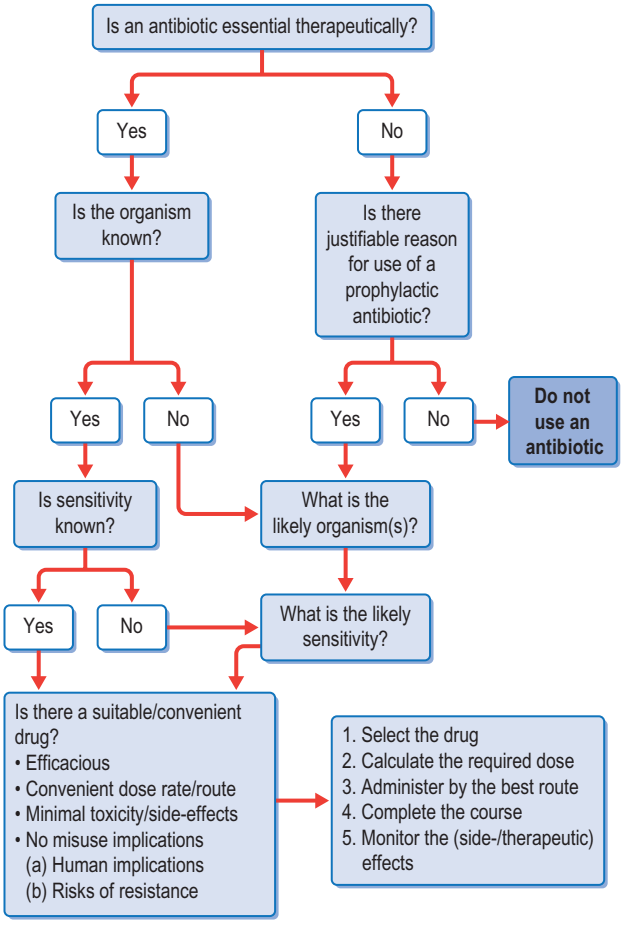


Figure 4.2 This algorithm illustrates the key decisions that should be taken when selecting an antibiotic for use in horses.

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and more importantly knowledge of the organism(s) most likely to be present. For instance, a kick wound may have a significant infection with *Escherichia coli* resulting from direct contamination but could also have pathological skin contaminant bacteria such as *Staphylococcus aureus*, *S. intermedius* or *S. epidermidis*. Once the potentially useful antibiotics are selected, the final choice may be based on the route or frequency of administration, the location of the infection, the pharmacokinetics of the drug, the cost of the drug, and the clinician's preference.

The pharmacokinetics should be considered before any antibiotic is administered to a horse. Ideally, a drug administered to treat a dermatological problem needs to be delivered to the skin in an effective concentration without any untoward effect on other organ systems. The correct dose is vital if the drug is to be effective – overdoses are wasteful and under-dosing with antibiotic is a potential factor in resistance development. To calculate the correct dose the weight of the horse should be established accurately if possible. The extent of local irritation, the rate of uptake from the site of injection or application and the excretion pathways carry significant risks if an inappropriate drug is administered. Thus, lincomycin and possibly oxytetracycline may have profound harmful effects on the bacterial flora and fauna of the large colon and intractable diarrhoea may ensue. Similarly, amitraz is an effective ectoparasiticide for dogs and cattle but is extremely toxic to horses, causing severe colic as a result of complete and often irreversible intestinal stasis.

The extent of protein binding can also influence the efficacy of a compound. A highly protein bound drug is unlikely to reach effective concentrations in the skin without very high systemic doses. A horse suffering from low blood proteins might sustain higher plasma concentrations for longer than a normal healthy horse, but effective uptake from the site of injection may be significantly delayed. The overall clinical status of the horse is therefore important when considering the drug and route of administration.

When should antibiotics be used in skin disease?

The number and type of bacteria in a wound or skin lesion are the major considerations governing wound contamination and infection (Spurlock & Hanie 1989). These can be further complicated by tissue trauma and necrosis. Impairment of the local blood supply and hence lowered delivery of immunoglobulins and leukocytes as well as the general condition of the horse are also important considerations. Old, debilitated horses often have poor responses to skin injury or insult. The use of systemic glucocorticoids also increases the risk of more severe bacterial invasion of the tissues.

There are many significant primary and secondary bacterial skin infections that occur without any prior skin trauma. Antibiotics may be indicated but often they are ineffective on the surface of the skin. Then a different route of application may need to be considered. Topical and systemic antibiotics are all used in the management of bacterial skin disease. Additionally, it is common practice to use prophylactic antibiosis but this carries risks and careful thought should always be given to the question 'Is an antibiotic justified in this case?'

For how long should antibiotics be used?

This is often related primarily to the tissue involved and the type of infection that is being treated. Involvement of bone, joints and tendon sheaths may require prolonged therapy (often up to 2–3 months), whereas, in superficial skin wounds, parenteral medication can usually be terminated once granulation has commenced. Where poor blood supply to a wound can be identified, therapy should nevertheless be prolonged rather than shortened. Some skin infections such as staphylococcal dermatitis can require prolonged courses, and the possibility of secondary infections with either other resistant bacteria or fungal infections should be considered.

What are the best routes for administration of antibiotics?

Prolonged or repeated intramuscular or intravenous medication can be distressing for some horses; even the first injection can be problematic, particularly if the injection is painful, and then there may be increasing subsequent difficulty. When there is difficulty with administration, doses may be missed or courses may be cut short and in either case efficacy can be adversely affected.

Pain can result from the material itself or from the volume of drug deposited at a single site or from incorrect injection technique (including errors of route such as intramuscular injection of an irritant substance designed to be injected intravenously). The benefits from multiple-site injections with a reduced individual volume have to be considered against the possible resentment arising from multiple injections.

There are few effective oral antibiotics for use in horses; only enrofloxacine and potentiated sulphonamides are widely used orally at the present time but some others are being developed. These two are also the only ones that have an injectable form as well as an oral form so that a loading dose can be administered parenterally with an oral dose being used to follow on.

There have been few attempts to establish pharmacodynamics and complications of the common oral antibiotics. Some drugs carry serious risks while in others there seems to be much less risk. However, anecdotal reports of serious complications arising from oral administration (and some parenteral) antibiotics makes the selection difficult. In practical terms the only available economical, safe, proven antibacterial agents in horses are the potentiated sulphonamides. These are used widely, being convenient, safe, easily administered and largely effective. However, distribution to the skin may be less efficient and achievement of effective antibacterial concentrations may be difficult.

How can the best (most effective) and most convenient antibiotic be selected?

Various factors need to be considered when selecting an antimicrobial drug.

- The *sensitivity* of the bacteria involved (or suspected). The in-vitro sensitivity to a given antibiotic does not guarantee in-vivo efficacy. Lack of response may be related to tissue concentrations of the active agent as well as the extent to which the organism is in contact with the drug. Thus, some organisms, for example *Rhodococcus equi*, are largely intracellular and may have a lipid capsule which inhibits antibiotic effects. Positive identification of the organism and its antibiotic sensitivity provide the best selection criteria. However, some laboratories

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include antibiotics that are either contraindicated in horses or are not available in suitable forms in sensitivity tests and others do not test specifically for those that are available and in common usage; results should therefore be interpreted with some care.

- Antibiotic *blood levels* affect the tissue levels because passive diffusion appears to be the most likely method of delivery of most antimicrobials. Efficacy is then subject to the activity in the presence of pus, tissue enzymes, acidity/alkalinity (pH) and fibrinous exudate, each of which can influence the clinical response.
- The relative benefits of *bactericidal or bacteriostatic* effects are important. Bactericidal antibiotics such as penicillin are usually preferred for skin diseases and wounds. In these cases there is then less reliance upon the body's own defence mechanisms. However, many bactericidal antibiotics are not effective, or are contraindicated in horses, or fail to reach the desired concentration in the desired site. This promotes the development of antibiotic resistance within the wound. Bacteriostatic antibiotics such as oxytetracycline rely ultimately on the natural defence mechanisms to remove the inhibited bacteria so there has to be a healthy cellular immune response.
- *Safety and freedom from side-effects* and idiosyncratic reactions are obviously important. Unpredictable (idiosyncratic) side-effects such as urticaria, anaphylaxis, etc. are entirely possible with any antibiotic but for the most part are commonest with the penicillin groups. Other drugs (such as lincomycin) have more predictable harmful side-effects (such as diarrhoea). Drug eruptions involving dermatological and systemic effects (such as immune-mediated vasculitis, systemic lupus erythematosus-like syndrome and thrombocytopenia) are less predictable and may follow the administration of any drug (although antibiotics probably have the greatest tendency to induce this) over a period of weeks or months following administration. Drug eruption is a rare but important skin condition in horses (see p. 288) and antibiotics have been implicated.
- The *cost* of the drug will also influence the choice. Many of the most effective, dermatologically active antibiotics are very expensive and involve regimens of treatment which make their use impractical. The cheapest drugs are usually basic penicillin, oxytetracycline and the potentiated sulphonamides. Fortunately, these are commonly sufficiently effective to make them valuable in the treatment of skin diseases.

In general the first-choice antibiotics are the simple penicillins as many dermatological infections in the horse are of *Streptococcus* spp. and most of these retain good or moderate sensitivity to penicillin. The synthetic penicillins such as ampicillin, amoxicillin and cloxacillin are less commonly used, often because of local reactions or impractical administration requirements. Aminoglycosides (such as neomycin and gentamicin) or cephalosporins (such as ceftiofur) are more useful if Gram-negative organisms are involved. The advanced, later-generation aminoglycosides such as amikacin are extremely expensive and usually impractical for dermatological cases. Long-term therapy is effectively limited to potentiated sulphonamides by mouth. These have the added advantage that courses of treatment can be started parenterally and continued for long periods.

Spectrum of activity

Some drugs have particular affinity for certain tissues, but unfortunately most seem to have a low affinity for skin. This may relate to the relatively low blood supply or other factors as yet undefined. Delivery of adequate therapeutic concentrations of antibiotics in the skin is sometimes difficult and frequently requires very high doses at frequent intervals.

Most equine skin diseases and wounds carry mixed infections but almost all will have a significant *Streptococcus* spp. component. Most if not all equine streptococci are penicillin sensitive or partially sensitive and so penicillin is widely used as first line of treatment in skin injuries and disease. However, kick injuries and wounds contaminated by faecal material will most likely have significant Gram-negative infections. Specific infections involving defined organisms may have characteristic sensitivity to antibiotics, but the widespread use of broad-spectrum antibiotics (often in unjustified circumstances) makes resistance a serious potential problem. In general, penicillin is ineffective against Gram-negative organisms but synthetic penicillins have improved activity. β-Lactamase-producing bacteria are resistant to penicillin and so infections with these organisms do not respond to it. Gram-negative organisms usually require aminoglycosides (preferably gentamicin); however, care must be taken to ensure delivery to the skin, which may be less than ideal.

The spectrum of activity also includes the activity of the drug against anaerobic or aerobic organisms. Metronidazole is a very useful drug for treatment of suspected or confirmed anaerobic skin infections (and is particularly useful topically in the foot region) but its tissue residue problems may reduce its availability.

Route of administration

The route of administration of an antibiotic may be dictated by the location of the condition, practitioner preference, environment of the animal, patient or owner compliance and economics. The duration of therapy needed may also influence the decision, since long-term intravenous administration requires a long-stay catheter, hospital environment and normal patent jugular vein(s).

The standard options available for administration of antibiotics to horses are:

1. parenteral route (intravenous; intramuscular)
2. oral administration
3. topical application.

Intravenous administration provides a rapid (almost instantaneous), high plasma concentration. This route often requires frequent dosing and may need special training to administer; it is usually therefore expensive. The need for special training can be avoided by the placement of an intravenous catheter; however, this may not be feasible outside the hospital and in any case catheter management can be difficult. It has been established that because its efficacy is concentration dependent rather than time dependent, gentamicin can be administered effectively using 6.6 mg/kg once daily (Magdesian et al 1994) rather than 2.2 mg/kg three times daily and this clearly makes its administration more economical (Geor 1997).

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All drugs administered to horses by the intravenous route should be suitably formulated and given slowly to avoid damage to the vein and possible delivery of a bolus of potentially irritant drug to any internal organ. The intravenous route may be chosen to avoid complications associated with tissue damage and local irritant/pain effects of intramuscular injection; all drugs administered by the intravenous route must be specifically formulated for that route.

Possible side-effects of intravenous drug administration include:

- jugular vein thrombosis/thrombophlebitis
- accidental intra-arterial injection
- accidental extravascular injection with local tissue effects on skin, connective tissue and other structures in the vicinity of the vein, for example the vago-sympathetic trunk
- bolus effects from over-rapid injection of a drug which might have effects on the heart or brain
- anaphylactic reactions.

Intramuscular administration may achieve good plasma concentrations but these may not be attained until 1–2 hours following administration depending on the formulation and the exact site of injection. Slower absorption, distribution and elimination require less frequent dosing than the intravenous route but levels may fluctuate. Although some special training is necessary for safe administration, clients can usually be trained to administer intramuscular injections. The expense is usually less than for intravenous medication. Large volumes of drug are, however, needed and these can be painful or warrant division of the dose into several sites. Patient resistance can make it difficult to complete longer courses.

Unwanted side-effects are rare but include:

- (transient) local swelling
- abscessation (either sterile or infected)
- damage to adjacent structures such as nerves, vessels and joints
- anaphylaxis (this is fortunately very rare but the risk is always present whenever a drug is administered parenterally).

Oral administration is perhaps the easiest for the client and the least traumatic for the animal. However, plasma levels of the drug are achieved slowly and it may be prudent to administer a loading dose of the drug by injection (if such a formulation exists). Furthermore, there are few antibiotics which can be given to horses by mouth. The logistics of administration of the doses which would be required are also problematical for many of the drugs used by this route in other species.

Where the oral route can be used the expense will vary with the medication used, but economies can be made simply by home administration. The cost of hospitalization and intravenous catheter placement can be avoided. Patient compliance is usually good and side-effects are usually minimal. Some drug interactions are important, such as the cardiac effects which are reported to occur when α_2-adrenoreceptor agonist sedatives (such as romifidine and detomidine) are administered with potentiated sulphonamides.

Topical antimicrobials are usually in the form of antiseptic solutions, antibiotic creams (often combined with various other pharmaceuticals such as corticosteroids or local anaesthetic agents) and so-called triple antibiotic ointment (a combination of bacitracin–neomycin–polymixin in an ointment base). Topical antibiotics may be useful as a method of antibiotic prophylaxis in surgery. However, it is preferable to apply them to the tissues at the time of incision. This will provide for maximum effect.

Duration of therapy

The duration of antibacterial therapy depends on the situation. In elective procedures, prophylactic antibiotics are useful when administered prior to surgery so that the skin concentrations are high at the time of the surgery. A refined clean procedure may require as little as one preoperative dose. By contrast, severely contaminated wounds involving synovial structures can require in excess of 21–28 days of intensive treatment. Although intravenous or intramuscular administration may be used initially, there are clear advantages in changing to the oral route. However, there are severe limitations in choice, and resistance or non-response to the oral formulations available for horses make long courses difficult. The change to the oral route should probably be delayed until patent sepsis has cleared. For traumatic wounds that do not involve bone or synovial structures, 3–5 days of treatment is usually sufficient. If a drain has been placed, therapy should continue for at least one day beyond the time of drain removal. If signs of infection are present, antibiotic therapy should continue for 2–3 days following resolution of the signs. Usually once drainage is established at the site of an incisional abscess, signs of infection will resolve rapidly.

Table 4.2 shows the commonly used antimicrobial agents and gives a summary of the regimens for their use. It is important to remember that none of these can be definitive and the clinician must make a careful assessment of the case as above before embarking on any therapeutic course. Furthermore, the clinician should be satisfied as to the safety and relevance of the regimen to be used.

Antifungal agents

A variety of antifungal agents are used in equine dermatological disease. These are either administered topically (several of the conazoles, nystatin and natamycin) or by mouth (griseofulvin and potassium or sodium iodide) or by injection (amphotericin B and sodium iodide). Generally antifungal agents work best when applied directly to the lesion, but extensive disease or deep mycosis is likely to resist topical application alone.

Sodium iodide and potassium iodide are underutilized, effective and safe antibacterial and antifungal agents when administered by intravenous and oral routes, respectively; the potassium salt must not be administered by intravenous injection. A dose of 10 g per 450 kg bodyweight once daily given in the feed has a detectable if mild broad-spectrum antibacterial and antifungal effect against *Aspergillus* spp. It is convenient and accurate to use a twice-daily dose of 25 mL of an aqueous solution of 160 g of potassium iodide dissolved in 400 mL of water. The solution can be mixed easily with the food and is generally totally unnoticed by the horse. Lesser doses can be effective also. The drug can

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Table 4.2 Commonly used systemic antibacterial agents used in equine dermatological disease[a]

Drug group	Agent	Route[b]	Dose (mg/kg)	Dose interval (q–*h)[d]	Notes
Penicillin	Na/K penicillin	i.v./i.m.	20–30[c]	6–8	Few problems
	Procaine penicillin	i.m.	20–30[c]	12–24	Few problems
	Benzathine penicillin	i.m.	20–30[c]	24–36	Few problems
	Ticarcillin (+clavulinate)	i.v.	50–75	6–8	Very expensive
	Ampicillin	i.v. (i.m.)	30–100	8	Risk of site reaction
	Amoxicillin	i.m.	30	6	Risk of site reaction
Aminoglycosides	Neomycin	i.m.	4–5	8–12	Possibly nephrotoxic
	Amikacin	i.v.	5–7	8–12	Possibly nephrotoxic; *very* expensive
	Kanamycin	i.v.	5	8	*Very* expensive
	Gentamicin	i.v. (i.m.)	3.3 (6)	8–12 (24)	Nephrotoxic; expensive
Cephalosporins	Ceftiofur	i.v. (i.m.)	2	6–8	Site reaction i.m.
	Cephalexin	p.o.	25–30	6–8	
Tetracyclines	Oxytetracycline	i.v.	5–10	8–12	Risk of shock if i.v. and diarrhoea
Amphoterics	Chloramphenicol	i.v.	30–50	6–8	Risk of bone marrow suppression/ resistance
Sulphonamides	Potentiated sulphonamides	i.v./i.m./p.o.	30 (total)	24	Site reaction Do not use with the α_2-agonist group sedatives. Very safe prolonged courses
Nitroimidazoles	Metronidazole	p.o./i.v.	15–25	6–8	Risk of diarrhoea
Macrolides	Erythromycin	p.o.	25	8	Risk of diarrhoea

A full antibiotic option list is available in most general veterinary texts such as *Current Therapy in Equine Medicine* (Robinson N E, ed), WB Saunders, Philadelphia and *The Saunders Equine Formulary* (Knottenbelt D C), WB Saunders, London.
[a]Uncomplicated superficial infections are probably best treated initially with penicillin or potentiated sulphonamides unless there is reason to suspect a Gram-negative or anaerobic infection. Use of other agents should be based on culture and sensitivity results but inevitably there will be occasions when other agents will be preferred. Antibiotic dosage regimens for horses are suggested but not definitive and may need to be adjusted in various clinical circumstances.
[b]Route: i.m., intramuscular; i.v., intravenous; p.o., by mouth.
[c]Dosages for penicillin are given in 10^3 i.u./kg.
[d]Dosing interval (q–*h), to be repeated *-hourly.

be continued until obvious signs of iodism develop (lacrimation, a sticky greenish naso-ocular discharge and later a scurfy, flaky skin are the major signs in most cases). If these signs develop, the compound can be withdrawn for 7 days and re-administered if needed. The effects are slow to develop and dramatic responses are unlikely in any deep-seated bacterial or fungal infection.

Amphotericin B is a very expensive drug which can be administered topically or intravenously. It has a wide antifungal range; most of these at least have in-vitro activity against species of *Blastomyces*, *Aspergillus*, *Histoplasma*, *Cryptococcus*, *Sporothrix* and *Zygomycetes*). It is fungistatic at low doses and fungicidal at high doses. It acts by binding to sterols in cell membranes and allows intracellular electrolytes (potassium in particular) to leak out. As bacteria and rickettsia have no sterols it is not active against these organisms.

It is highly bound to plasma proteins and has good distribution to all organs including skin. It is used most often for treatment of phycomycosis and other deep fungal infections. There is a significant risk of systemic toxicity, however, and so all cases receiving parenteral doses must be carefully assessed to confirm its value in the case itself and to check for ongoing systemic toxicity. Topical creams are also useful for application to localized fungal lesions.

Griseofulvin is a microsized-particle antifungal agent used in the treatment of superficial mycosis (such as dermatophytosis/ringworm). The drug has been used for many years and has gained a good reputation but recent results are not as satisfying. Reports of the efficacy of

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griseofulvin are anecdotal – no extensive trials have been reported in support of this. This may be due to resistance or another factor not yet established. It is a tasteless powder which is well tolerated for prolonged courses: courses of up to 30–40 days may be required in some cases. There is no detectable effect against *Aspergillus* spp. or other deep mycoses. It is potentially teratogenic and so should not be used during pregnancy.

The conazoles (including miconazole, ketoconazole, enilconazole, fluconazole and itraconazole) are used widely in the treatment of fungal infections and particularly in superficial mycoses such as trichophytosis and microsporosis (ringworm). The drugs do have a useful effect on *Aspergillus* spp. but delivery to the site may be more difficult. Topical application and systemic dosing can be used with relevant formulations. In spite of the obvious widespread use and efficacy there is little specific information about the efficacy and the pharmacokinetics of the conazole group in horses. This is particularly disappointing given that fungal diseases are relatively common and some have life-threatening potential.

Ketoconazole has some potential in the treatment of *Sporothrix schenckii* but its action is poorly documented. The drug is administered by mouth at 30 mg/kg twice daily. It is extremely expensive and has already largely been replaced in human medicine (for treatment of sporotrichosis) by itraconazole, which can be given to horses at 3 mg/kg twice daily by mouth. Again its expense is likely to limit its use to those fungal infections that fail to respond to other agents (including iodine salts).

A commercial mixture of 2% miconazole with 2% chlorhexidine has been shown to be useful as a whole-body antifungal shampoo and an environmental antifungal agent (Paterson 1997).

Enilconazole is very effective when applied topically to ringworm-infected horses (Paterson 1997). It is applied directly to the lesions and the surrounding skin every 3 days for three or four applications. Alternatively, the horse can be sprayed with the diluted solution. The drug is not absorbed from the skin and has no significant toxicity apart from the irritant effect of very strong solutions incorrectly applied.

Fluconazole and itraconazole can be administered orally but are largely untested in horses; they may have useful effects against some of the problematic deep fungal infections such as *Cryptococcus neoformans* and *Histoplasma capsulatum* var. *farciminosum* but little information is available for these circumstances.

Nystatin is a naturally derived antifungal antibiotic which is available for topical use as a powder or a cream. Oral use is not effective as the drug is not absorbed from the gut. Although it is anecdotally reported to be particularly effective against *Aspergillus* spp. and *Candida* spp. there are no reports of efficacy trials in horses.

Natamycin is an insoluble powder which, in suspension, is used to treat superficial mycosis and in particular *Trichophyton* spp. and *Microsporum* spp. infections (ringworm). The suspension is very safe and non-irritating and is therefore widely used. In high concentrations it can, however, cause skin inflammation and so strict adherence to manufacturers' instructions is required. Its strong sporicidal activity makes it very useful both for the direct treatment of affected horses and for sterilizing the stable and harness, etc. Again there are no reports of efficacy trials. Unless previous personal usage has given uniformly good results, natamycin and nystatin should be used with care until appropriate data are established.

Potassium monopersulphate can be used to control environmental fungal infections (Paterson 1997) and many of the available disinfectants (see above) have strong antifungal and sporicidal effects.

The common antifungal drugs and agents are shown in Table 4.3.

Ectoparasiticides

Ectoparasiticides are an essential aspect of equine dermatological practice. In spite of the overall clinical importance of ectoparasitic infestations, the therapeutic options have been much reduced with the removal of many organophosphate and chlorinated hydrocarbons from the drug lists. The ongoing need for effective topical medications has not led to the development of new chemicals and even fewer specific equine compounds are being produced. The choice of ectoparasiticidal compounds is important as misuse can have significant environmental and health and safety implications. To date there is no evidence of resistance but at least in theory, misuse can lead to this complication.

There are few, if any, licensed ectoparasiticidal products for topical application to horses in Europe, Australasia, the United States and Canada. In other parts of the world some of the more effective (but environmentally harmful) compounds remain in use. There is no doubt that many of the problematical compounds have been abused (environmental considerations are very important) but their value in veterinary treatment and the relief of animal suffering has been considerable. There are some compounds that are still available for human use such as malathion but they are not permitted in horses in case someone touches them or eats the horse!

There are some compounds including amitraz and arsenical cattle dips which are very harmful to horses and should never be used in any case.

The treatment of ectoparasites can be performed by systemic or topical means. The correct selection of the approach depends upon the nature of the infestation, the drug and the desired effect. For example, there is little merit in treating a single small local lesion with systemic treatment involving high systemic doses of a drug, and conversely topical application to large areas may be difficult and impractical and a systemic option might be more appropriate.

Prevention of exposure is often the pragmatic approach to insect challenge (Fig. 4.3).

Chlorinated hydrocarbons

In most countries production and use of these compounds is prohibited due to toxicity and residue problems. Gamma benzene hexachloride and bromocyclen are probably the last of these compounds which are available in some countries. There is little doubt that the compounds

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Table 4.3 Commonly used antifungal drugs for use in treating fungal skin infections in horses

Drug group	Agent	Route	Dose (mg/kg)	Dosing interval (q–*h)[a]	Notes
Iodines	Potassium iodide	Oral	10 g total dose	12	Safe and cheap but somewhat unreliable
	Sodium iodide	Oral/i.v.	10 g total dose	24	Cease if signs of iodism occur (lacrimation/scurfy coat) Activity against *Cryptococcus* spp., *Histoplasma* spp., *Blastomyces* spp., *Aspergillus* spp.
	Povidone-iodine	Topical Dilute	n/a	As required	More dilute solutions are better
Polyene agents	Griseofulvin	Oral	10	24 (×10–30 days)	Activity against *Trichophyton* spp., *Microsporum* spp. only
	Nystatin	Topical			Very toxic parenterally Activity against *Candida* spp., *Cryptococcus* spp., *Histoplasma* spp., *Blastomyces* spp. *Trichophyton* spp. *Aspergillus* spp.
	Natamycin	Topical			Activity against *Trichophyton* spp., *Microsporum* spp. Useful sporicidal activity
	Amphotericin B (Robinson 1987)[b]	Topical i.v. as solution in 5% dextrose *(not saline)*	0.3 0.5 0.6 0.6	Day 1 Day 2 Day 3 q 48 h for 3 days per week	Renal and hepatic toxicity. Used for phycomycosis and other deep/systemic mycoses; *Cryptococcus* spp., *Histoplasma* spp., *Blastomyces* spp., *Aspergillus* spp.
Imidazoles	Miconazole	Topical			Active against *Candida* spp., *Cryptococcus* spp., *Histoplasma* spp., *Trichophyton* spp. (with chlorhexidine)
	Ketoconazole	Topical			Active against *Candida* spp., *Cryptococcus* spp., *Histoplasma* spp., *Trichophyton* spp., *Microsporum* spp.
	Enilconazole	Oral			

[a]Dosing interval (q–*h), to be repeated *-hourly.
[b]Manufacturer's data sheet *must* be consulted carefully before using this drug. Preparation of the solution and rate of administration intravenously *must* be accurate.

were effective and that their loss has a significant implication in the treatment of parasitic infections. Some human anti-parasitic washes and shampoos, however, still have the chemicals and some of these may be safely used on horses where such an approach is legally and ethically acceptable.

Organophosphates

These compounds are now banned in many parts of the world due to the toxic effects on humans of prolonged or repeated exposure. Many of them are, however, still used in the control of insect pests on domestic pets and on agricultural crops and garden plants.

Commonly used insecticidal compounds which have been used for horses include dichlorvos, diazinon, malathion, phosmet and chlorpyrifos. Phoxim (Sebacil, Bayer, Germany) is still available in some parts of Europe and can be an effective compound on horses. These compounds are potentially toxic if used improperly and they may have damaging environmental effects also. All have been used topically in horses (usually as washes or dips but occasionally as pour-on compounds) and have good anti-ectoparasitic effects. Most of the compounds have a potent anticholinesterase activity but vary in their toxicity. Application must be strictly according to the manufacturer's instructions. Clinical signs of toxicity can develop rapidly following their use, particularly in overtired/exhausted horses or in hot humid weather or as a result of over-frequent dipping or from over-strength applications. Signs of toxicity include salivation, trembling or convulsions, constricted pupils, diarrhoea and collapse. Simultaneous use of other organophosphates (such as

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Figure 4.3 This horse suffered from severe insect bite hypersensitivity. He resented being stabled and so the owner provided a full set of clothing for the horse and turned it out into an open-fenced paddock. This resulted in cessation of signs and a happy pony.

dichlorvos-based anthelmintics) makes toxicity even more likely. Treatment of the toxic signs includes administration of atropine sulphate by injection and control of convulsions.

Natural insecticidal compounds

A number of natural compounds with variable insecticidal properties have been used over many years in treatment of ectoparasitism in horses. These include rotenone/derris, lime sulphur and natural pyrethrins. These compounds are usually derived from plants or naturally occurring chemicals. They are almost all very safe and non-toxic but their effects are often marginal. All these substances have a limited duration of effect which limits their therapeutic value. Lime sulphur has a particularly unpleasant smell and can cause burning and irritation of the skin when used in strengths over 5% but it has a significant effect on surface-feeding mites and on some dermatophytes.

As the more effective drugs are discontinued the value of these compounds may need to be reviewed.

Synthetic pyrethroids

Commonly used compounds include deltamethrin, permethrin, cypermethrin and fenvalerate. These compounds have almost no known toxicity at any dose and so are extremely safe. They are frequently used topically against skin parasites. Piperonyl butoxide is commonly added to preparations to enhance the activity. They have a higher efficacy and longer duration than the natural pyrethrins which they have largely replaced. The synthetic pyrethroids are particularly useful as repellents for *Culicoides* spp. midges and a few species of fly but must be applied at least weekly and immediately after rain wetting in order to achieve continuous protection. Cypermethrin and permethrin have been used as repellents in fly fringes and tags for attachment to head collars and the mane and tail hair (Fig. 4.4).



Figure 4.4 This horse has a cypermethrin-impregnated tag fastened to its forelock. Others were attached to its mane and tail to act as an effective fly repellent.

Other agents

1. *Selenium sulphide (1%).* A shampoo for the treatment of seborrhoea in pet animals, it has been used for the treatment of lice in particular, in horses (Paterson & Orrell 1995). It is claimed that the shampoo also reduces the secondary seborrhoea which is characteristic of lice infestation.
2. *Macrocyclic lactones (avermectin compounds).* These are commonly used for the treatment of helminth and bot (*Gasterophilus* spp.) parasites. Ivermectin and to a lesser extent moxidectin administered orally has been shown to have an effect in reducing the extent of egg laying by lice (*Werneckiella* (*Damalinia*) *equi* and *Haematopinus asini*) and mites (*Chorioptes* spp., *Sarcoptes* spp. and *Psoroptes* spp.) and is anecdotally reported to have some effect against the adult stages also, but the effects are unpredictable and generally it seems that it is unlikely to eliminate the parasites (Barth & Sutherland 1983). In some parts of the world injectable forms of doramectin are preferred as a means of controlling ectoparasites, especially those that are blood or lymph feeders (ticks, sucking lice and lymph-feeding mites).

   It is possible that later-generation avermectin compounds will be found to have a greater effect when administered either by mouth or by injection. There are no published trials of these (or any of the other compounds) in horses. To date there are no reports of resistance but continued use may encourage resistant strains of mites, lice and ticks.
3. *Insect growth regulators.* Most of these compounds have been developed for the control of fleas in small animals. They specifically target chitin, which is a major

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component of the insect exoskeleton and which does not exist at all in the mammalian body. Currently available insect growth regulators include benzoyl phenylurea derivatives and pyrimidine derivatives. Whilst these have been used on farm animals there is no study on their general application to horses. Possibly the best value is in their use in stables infested with fleas of various types.

4 *Lime sulphur.* Sulphur and compounds that contain it can be effective parasiticides and are relatively non-toxic and have few environmental concerns. Lime sulphur is a cheap and readily available wash in some countries and has been shown to be effective in horses for controlling surface mites and lice in particular. Sulphur washes of this type have a foul smell that lingers for a long time and they tend to stain white hair quite badly. Therefore they are largely unpopular with owners but are worth trying when other materials are either ineffective or unavailable.

Note

The amidine (formamidine) group of insecticidal compounds includes the molecule amitraz. Amitraz is highly effective against all the mites and ticks that could affect horses but it is *seriously toxic to horses* and therefore is specifically contraindicated.

Corticosteroids

General considerations for the use of corticosteroids

Corticosteroids are anti-inflammatory, immunosuppressive drugs which are frequently used in immune-mediated and pruritic skin diseases and in diseases where non-infective inflammation plays a major role in the aetiopathogenesis. They are powerful drugs and must be used carefully and correctly. Oral medication is usually preferable to parenteral injection as the former provides a better sparing effect on the pituitary–adrenal axis and thereby minimizes their secondary, undesirable effects.

The drugs are commonly employed in the treatment of insect bites, insect hypersensitivity, habronemiasis, allergies and in some immune-mediated diseases including vasculitis, urticaria, pemphigus (in all its forms), systemic lupus erythematosus-like syndrome and sarcoidosis.

Specific diseases do not always respond to standard treatment regimens and may require either a higher or lower than normal dose rate. Once remission is established, the dose should be gradually reduced over 2–3 weeks to the lowest dose which maintains the desired therapeutic effect. Once this dose is established, an attempt should be made to use alternate-day treatments as this will have a negligible effect on the pituitary–adrenal axis and will minimize side-effects.

Some of these drugs do not always have the expected effect in every case and alternatives should then be tried. Thus, it is usual to institute treatment with prednisolone as it has the fewest problems and is conveniently administered by the oral route. Dexamethasone should then be used if no effect is detectable within 24–48 hours. Again it is important to reduce the drug dose to a minimum effective dose as soon as possible.

It is important when selecting a corticosteroid to consider its specific pharmacokinetics. For example, prednisone is widely used in other species but it cannot be converted into an effective metabolite in horses. There is little evidence for its efficacy and it probably has no effect beyond placebo in most cases – it therefore has no material value in dermatological therapy. The drug distribution of methylprednisolone acetate is such that its value in dermatology is also very limited. Its supposed depot formulation results in poor volume distribution and unpredictable blood concentrations. Triamcinolone is possibly the compound that is associated with most of the largely irrational fear of corticosteroids in horses. Evidence-based medicine suggests that it is not as liable to induce laminitis as it was previously thought (Cornelisse & Robinson 2004, McCluskey & Kavenagh 2004). As might be expected, there are limits to the efficacy of any drug and potent glucocorticoids are no exception. Maximal doses should be accepted and not exceeded. Furthermore, there may be circumstances when the pharmacokinetics are impaired or altered and then the dangers may be far greater. No drug should ever be administered without due consideration for the possibility of side-effects and the possibility that standard doses may be excessive in some circumstances. As a good principle, drugs should only be used when specifically indicated and should be withdrawn as soon as they no longer have a genuine benefit. This principle applies as much to corticosteroids as to any other type of drug.

Whenever prolonged corticosteroid treatment courses are unavoidable, alternate-day therapy should begin as soon as the case becomes stable. Prednisolone and dexamethasone can be used in this way and so are the preferred choices in most cases. Dexamethasone and betamethasone have a longer duration of effect than prednisolone and so have less sparing effects but can still be used in alternate-day regimens. Anti-inflammatory (low) doses of prednisolone may not cause side-effects, whereas immunosuppressive (high) doses administered daily carry significant risks including immunosuppression (with a risk of infections), laminitis, polydipsia, polyphagia and behavioural alterations.

Prolonged (or sometimes even short) high-dose (or low-dose) courses may induce acute, often life-threatening laminitis, although there are few recorded confirmed scientific reports to this effect. The risks of laminitis should not be underestimated but are probably not as great as once feared. The risks appear to be much higher in cases which have active laminitis or which have had, or are susceptible to, laminitis. Under these conditions the use of corticosteroids should be considered particularly carefully. Triamcinolone seemingly has a greater potential for causing laminitis than other members of the group but is commonly only used in very small doses for the intralesional treatment of collagen necrosis nodules (and occasionally for intra-articular injections).

Complete failure of corticosteroid therapy should be followed by a careful reassessment of the case and, if the diagnosis is confirmed, treatment with aurothioglucose can be attempted as an alternative approach with the same objective. Azathioprine, a potentially toxic immunosuppressive drug, can also be used when corticosteroids are contraindicated for other reasons or fail to bring a resolution.

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Systemic corticosteroid therapy (Stannard 1994)

Clinical effects

The effects of corticosteroid therapy can be divided into three major categories. Each of the compounds has a slightly different bias in terms of both the desirable and the undesirable effects but these are probably of little overall clinical significance. However, some specific compounds such as triamcinolone may have potentially more dangerous side-effects. As a general principle, glucocorticoids are catabolic because they enhance the breakdown of protein tissues and they generally induce an increase in blood glucose levels by stimulating gluconeogenesis and glycogenolysis.

1 *Anti-inflammatory effect*
   - Decreased leukocyte activity (prevents release of lytic enzymes which would destroy cells and attract other inflammatory cells to areas of tissue damage)
   - Decreased leukocyte accumulation (monocytes and lymphocytes are sequestered into tissues)
   - Vascular stabilization (reduced capillary permeability)
   - Suppression (sequestration) of eosinophils
   - Stabilization of mast cells
   - Blockade of prostaglandin release.
2 *Immunosuppression effect*
   - Lymphocytopenia
   - Decreased T-lymphocyte function
   - Decrease in immunoglobulin and complement levels.
3 *Antimitotic effect (retarded cell division)*
   - Specific inhibition of fibroblasts.

Drug type

The types of corticosteroids available for use in horses can be divided into those which act for shorter or longer periods.

1 *Short-acting* (biological effects last for less than 12 hours), e.g. hydrocortisone, cortisone
   - Very rapidly metabolized
   - Little pharmacological effect and little clinical application.
2 *Medium-acting* (biological effects last for 12–24 hours), e.g. prednisolone or prednisone, triamcinolone, methylprednisolone and isoflupredone
   - Given daily at beginning of treatment
   - Suitable for alternate-day therapy.
3 *Long-acting* (biological effects last for over 48 hours), e.g. dexamethasone, betamethasone, flumethasone
   - Long half-life, smaller dosage required
   - Potential additive effect
   - Not usually suitable for alternate-day treatment.
4 *Slow-release*, e.g. methylprednisolone acetate
   - Convenient for general use
   - Can be used intralesionally
   - Possibly increased risk of side-effects including laminitis.

Route of administration

The preferred route of administration for each of the compounds varies.

Parenteral/injection The drug is injected intravenously, intramuscularly or subcutaneously. Over the period during which the drug remains in the bloodstream it exerts a continuous effect on the area of inflammation. There will also be continuous adrenosuppressive and mineralocorticoid effects. These will remain for as long as the drug is at concentrations capable of exerting the effect (even very low doses can have significant effects on the pituitary–adrenal axis). There will be a progressive, slow reduction in circulating drug concentrations and so there is no abrupt withdrawal of the drug.

Intralesional cortisone injection Very high local concentrations of the drug can be achieved locally without large overall doses and can therefore provide prolonged local effect. It is applicable to nodular lesions in particular. The overall dose of the compound used (usually triamcinolone acetonide or methylprednisolone actetate) should always be considered. Multiple small injections soon add up and it is easy to exceed the safe upper limit overall when many very small doses are being given. As a basic principle this has little to commend it – there are local and potentially systemic side-effects that are often more serious than the original condition. Injection locally commonly causes calcification and severe local immunosuppression can result in bacterial proliferation.

Oral corticosteroids The oral route is generally considered to be safer than injections. Provided that the dose is correct, absorption from the gastrointestinal tract is rapid and effective in delivering a therapeutic concentration in the blood. As a basic principle the safest, most effective drug should be used – this usually means prednisolone, which has a high safety margin in horses. The action of prednisolone stops quickly after therapy ceases. Therefore it is useful for alternate-day therapy and has minimal adrenal–pituitary axis suppression. Oral dexamethasone appears to have a rapid and useful intestinal absorption rate. However, the nature of the drug is such that it has a much longer duration in the bloodstream and so has a lesser sparing effect on the pituitary–adrenal axis than prednisolone. Nevertheless there are circumstances when its increased anti-inflammatory effects are valuable.

Note

All single daily doses of oral glucocorticoids should be administered in the morning.

In some clinical circumstances such as the supplemental use of omega-3 fats in atopic horses or the concurrent use of gold salts in pemphigus cases, it may be possible to reduce the dose significantly. Again the objective is to reduce the dose to the minimum effective daily or (preferably) the minimum effective alternate-day dose as soon as possible to spare the pituitary–adrenal axis.

There are also minimal mineralocorticoid side-effects such as polydipsia–polyuria, hyperglycaemia and alterations in appetite.

Topical dermocorticoids Topical application of corticosteroids is commonly practised. Lotions, creams, ointments and gels are available and are often combined with antibiotics and sometimes antifungal agents also.

The local concentration of the drug is high immediately after application but falls rapidly thereafter as the drug

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diffuses into the bloodstream. Systemic effects are minimal but maintenance of therapeutic effect requires repeated applications at short intervals. It is not often used in the horse except for localized single inflammatory lesions. The antimitotic effects are sometimes used in the control of abnormal cell replication in exuberant granulation tissue or superficial skin neoplasms.

They should always be applied thinly and evenly – over-applications serve no benefit and in fact may be harmful. One or two applications per day is usually enough – more repeated applications may adversely affect the skin immune processes and prolonged heavy applications can be severely immunosuppressive and may also cause skin thinning. These applications are probably contraindicated when there is overt infection unless the applications are accompanied by specific mechanisms to control or eliminate the infection. All applications should be made using gloved fingers – the drugs can equally be absorbed into the handler's body.

Recent developments of spray-on forms of hydrocortisone and betamethasone have allowed high local concentrations to be achieved with few systemic effects. These are, however, largely untested in horses at the present time but preliminary results are encouraging. It is always important to remember that even localized applications will have an inhibitory effect on local beneficial immune responses, so care must always be exercised.

Duration of therapy

Long-term therapy with corticosteroids can be required in some disorders, although it is generally accepted that this is undesirable. Where no alternative drugs class can be used and where long courses are unavoidable, 'the shorter the course the safer the regimen' is probably a justifiable maxim. The treatment course should be carefully monitored to ensure that the drugs are being administered in the most efficient manner while minimizing the risk of untoward effects.

1. Induction and daily dose are maintained high until clinical signs regress.
2. The daily dose is tapered off to obtain a minimal (effective) daily dose (MDD).
3. The maintenance dose is established by using double the MDD every alternate day and this is then adjusted (downwards usually but sometimes upwards) to establish the effective alternate-day dose (EADD).
4. The EADD is maintained to stabilize the condition.
5. Decrease dosage progressively to establish the minimum effective alternate-day dose (MEADD).

Dose factors

The individual drugs have dosages that are usually (although not invariably) effective, but this varies with the purpose for which the drug is being used. All single daily doses should be administered in the morning to correspond with the diurnal rhythm of endogenous cortisones.

1. *Anti-inflammatory activity*
   - Prednisolone: induction dose 0.8–2.2 mg/kg; maintenance dose 0.4–1 mg/kg
   - Dexamethasone: induction dose 0.1 mg/kg; maintenance dose 0.05 mg/kg.
2. *Immunosuppressive activity*
   - Prednisolone 2–4 mg/kg (prednisone is not of value in horses)
   - Dexamethasone 0.1–0.2 mg/kg.

Note

Treatment with either drug is continued until remission is obvious. If no change occurs within 10 days, an alternative treatment should be attempted.

Once remission is achieved the dose should be reduced by 20% every second week until a maintenance dose is achieved (this is usually 20% higher than the minimal effective dose).

Side-effects (or undesirable effects)

Side-effects are not common but are invariably important when they are present. They include:

- *Pituitary–adrenal axis disturbances*: Suppression of the pituitary–adrenal axis by negative feedback mechanisms.
- *Polydipsia and polyuria* (mineralocorticoid effects).
- *Psychological effects*: Mood changes.
- *Immunosuppression*: Increased susceptibility to infection (immunosuppression). This effect is of doubtful significance in horses.
- *Catabolism*: Catabolic effects, reflected in weight loss and poor hair coat and skin quality (this is usually a marginal effect in horses).
- *Withdrawal syndrome*: Abrupt withdrawal of the drug (even after a relatively short course) may, in theory at least, result in an adrenal insufficiency syndrome similar to Addison's disease (hypoadrenocorticism). This has, however, not been well documented in the horse, which would seem to be less susceptible to the effects than most other species. Nevertheless abrupt withdrawal of the drugs is not advisable.
- *Laminitis*: The true incidence of laminitis as a direct consequence of corticosteroid administration is hard to establish. Recent retrospective studies have largely discounted any direct (Cornelisse & Robinson 2004, McCluskey & Kavenagh 2004). There are, however, anecdotal reports of catastrophic laminitis following even small doses, and some high profile court cases that appear to take little account of the real evidence, but many horses receive these drugs (even in very high doses) without any untoward effect.

The major factors which appear to predispose to the iatrogenic occurrence of laminitis are:

- excessive doses (especially of the trimacinolone group)
- combinations of drugs used simultaneously or alternately
- active laminitis (acute, chronic, refractory) at the time of administration
- previous history of laminitis
- predisposing hoof conformation (large horse with small feet)

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- predisposed body type (e.g. fat, under-worked, unfit ponies)
- dystrophic calcification: this may occur when depot injections are used in the skin/subcutis (or when injections are made into tendons and ligaments).

Corticosteroid-like drugs

Ciclosporin Ciclosporin is a selective immunosuppressant calcineurin-inhibiting drug, with a low cytotoxicity and a high immunosuppressive effect that selectively inhibits interleukin-2 production by T lymphocytes. It has inhibitory effects on mediators including interferon-α, interleukin-3/4/5 and tumour necrosis factor and so it directly suppresses activation of macrophages, mast cells and monocytes and neutrophil adherence. It can therefore be considered to be an immunosuppressive anti-inflammatory drug.

There are no large-scale reports of its use in equine dermatology – it is bound to be prohibitively expensive in oral form at the suggested dose of 5–10 mg/kg q 12 h that is used in humans, dogs and cats, but at least in theory it should be useful for conditions such as atopy.

A large number of drugs including some antibiotics such as erythromycin react with the drug and so there are major difficulties with its use. The doses required for horses are extremely expensive.

Azathioprine This is a purine antagonist anti-metabolite with immunosuppressive properties. It affects cell replication through inhibition of RNA and DNA synthesis and mitosis. It has a greater activity on delayed hypersensitivity and cellular immunity than on humoral antibody responses. It is used largely as a second- or third-line immunosuppressant drug in the treatment of immune-mediated disease and is applicable when glucocorticoids are contraindicated or have been ineffective.

It has been used to treat difficult or refractory cases of pemphigus foliaceus and some other immune-mediated conditions including urticaria (Scott & Miller 2003).

The drug is administered at a dose rate of 2.5 mg/kg for the first two doses 24 hours apart and then q 48 h for maintenance.

The nature of the drug means that there are likely to be serious potential side-effects such as myelosuppression. It is also prohibitively expensive.

Aurothioglucose Where horses do not respond to glucocorticoids alone or laminitis is a high risk factor, the use of gold salts (aurothioglucose) therapeutically may be of advantage (Manning 1983). These compounds possibly decrease histamine release from mast cells and inhibit prostaglandin formation. It is particularly used to treat severe immune-mediated skin problems such as pemphigus in its various forms.

A test or induction dosage of 0.25 mg/kg aurothioglucose can be administered intramuscularly followed by maintenance dosages of 1 mg/kg aurothioglucose in multiple intramuscular injections at weekly intervals, continued until the disease is in remission (often after 6–8 weeks). Thereafter the aurothioglucose is decreased in frequency to every 2–4 weeks. Gold therapy should be discontinued if any untoward signs appear.

The drugs have a myelosuppressive side-effect in some horses. However, there are reports of successful treatment of some cases of pemphigus foliaceus in particular and in any case the drug may reduce the dependency on corticosteroids – a lower required dose and less frequent relapses are reported.

Pentoxifylline This is a methylxanthine immunodulatory drug with a wide range of pharmacological effects against mediators of inflammation. It may also inhibit T-cell adherence to keratinocytes and so it may have at least a theoretical benefit in some cases of atopy, erythema multiforme and other immune-mediated dermatoses including those characterized by pruritus. A dose of 10 mg/kg q 12 h is suggested but little is known of its benefits and side-effects.

Antihistamines

Antihistamines act by competitively inhibiting histamine at H_1-receptor sites. They do not inactivate or prevent the release of histamine but rather control the effect of the histamine on cells. They may also have significant sedative and anticholinergic effects.

Antihistamines appear to have little or no significant pharmacological effects in equine dermatology; their best effects are available before histamine is released (i.e. as a prophylactic measure) and this state is seldom applicable in the horse. Their effects are notoriously variable – some horses seem to respond well or even excessively while others show no material effect or unwanted side-effects. Tripelennamine hydrochloride and hydroxyzine hydrochloride are the only specific antihistamines with any clinically significant effects in horses. However, an effective antihistaminic effect is also shown by certain phenothiazine ataractics such as acepromazine and by corticosteroids in general.

Tripelennamine hydrochloride This is an ethylenediamine derivative with strong antihistaminic activity. It acts by blocking the effect of histamine on H_1-receptors on the cell but it does not inactivate the histamine or reduce its release from cells. It has limited effects at a dose rate of 1.1 mg/kg by intramuscular injection (at multiple sites) once or twice daily. The drug should not be administered intravenously (seizures, collapse or disorientation are possible). Intramuscular injection is often painful and causes significant swelling (and risks of abscessation).

Hydroxyzine hydrochloride This is a tricyclic antidepressant with H_1-blocking properties. The drug also has mild sodium channel blocking effects. It is probably the most effective (prophylactic) antihistamine. Dose rates of 1–1.5 mg/kg q 6–8 h are used. An empirical dose of 600 mg three times daily for a 450 kg horse can be tried safely. The dose should be reduced to a minimum daily level to control recurrence of the problem. Once this occurs the dose should be reduced to twice daily and then to once daily. Major side-effects can be sedation or hyperactivity and sometimes some 'personality' changes (Rosencrantz 1988) but usually there are few if any noticeable problems. Sedation that occurs following the first one or two doses usually disappears by the third or fourth dose. It cannot be given to pregnant mares as it has teratogenic properties.

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Other antihistamines that are sometimes listed for use in horses include chlorpheniramine (0.25–0.5 mg/kg q 8 h) and diphenhydramine (1–1.5 mg/kg q 8 h).

Corticosteroids have an antihistaminic effect also and when administered at the same time as antihistamines may enhance the effects of many of the rather unsatisfactory antihistamine drugs.

Dermatological shampoos/washes

The skin of normal healthy horses is in delicate balance between the host and the large variety of commensal bacterial species that inhabit the skin surface. The balance is a critical part of the maintenance of a strong barrier between the dermis and the outside challenges. Normal skin is exfoliative and so some scale and flake is an inevitable natural and healthy occurrence.

Topical materials that are applied to the skin of horses include various shampoos with defined and ill-defined properties or benefits. However, in spite of the importance of a 'good healthy skin' to horse owners, little is known about the best compounds, washes and lotions, etc. for the horse. Most information is extrapolated from small animals and humans and the reality is that the horse's skin is very different from both of these. This means that there are anecdotal reports of amazing successes and disasters in about equal numbers.

Shampoos There is an increasing and disturbing tendency towards repeated washing of horses and a corresponding burgeoning of the products available for cosmetic washing. At the same time the number of therapeutic topical washes is falling as fewer are licensed and found to be commercially viable. In spite of the plethora of useless (or possibly even harmful) materials that are available there are some that are mainstays of equine dermatology. Some are designed to alter skin pH, to degrease, to perfume and to deliver specific chemicals with a defined objective such as parasiticide and defoliant shampoos. Some shampoos and rinses can be applied with good therapeutic effects.

Delivery of a therapeutic effect to the skin is a major challenge to the horse owner and clinician. Not only is the size a problem but the hair coat of the horse has protective properties that prevent the wetting of the underlying skin. This is a particular problem on the back and the limbs especially in heavy horses with feathered legs. In many cases therefore delivery of any chemical requires several stages of management.

- Preferably the hair coat should be clipped off short so that the therapeutic effect can be maximized.
- The natural greasiness of the hair coat may need to be overcome using a degreasing material. In some cases dishwashing detergents may be acceptable for this purpose. They are cheap and have strong anti-greasing properties and are generally formulated to avoid any human skin damage. They tend therefore to be very bland. Their potency means that they should be used judiciously and only when degreasing is a desirable part of the treatment. There are, however, specifically designed equine degreasing shampoos but it is not clear what significant benefits they have to justify their considerable extra cost.
- Cosmetic (shining) shampoos are also commonly used. Hypoallergenic shampoos are used when the skin or the hair reacts adversely to the active ingredients in conventional shampoos.
- Emollients and moisturizers (hair conditioning ingredients), including chitosanide, colloidal oatmeal, fatty acids, lipids and glycerine, are frequently added to improve the shampoo's effects when individual horses require to be bathed frequently or where the hair has a brittle character or the skin is seborrhoeic. Frequent bathing of horses is taken to be when a whole body shampoo is used more frequently than once per month. Grey horses are probably subjected to more bathing procedures than other colours because they 'look dirty' much more quickly.

Modern shampoo technology has resulted in a wide group of shampoo-like materials that can be used to deliver specific chemicals such as parasiticides and hydrating and conditioning materials. All these (like all other topical applications) must be applied according to the manufacturers' instructions. Many require to be left on the hair/skin for extended periods to allow for the maximal effects.

When a shampoo is to be used for a genuine therapeutic effect it may be necessary to treat only one defined area or to treat one area differently from the rest. For example horses affected with chorioptic mange commonly have a major infestation on the limbs and head and far less on the body. Therefore extra attention may need to be applied to these areas. Antifungal shampoos can be used over the whole horse to ensure that a maximal effect is gained but the sites of lesions may benefit from a pre-wash treatment or a second wash after the whole body one has been carried out.

Rinses are liquid (usually aqueous) solutions of various chemicals that are left in contact with the skin to dry into the hair and onto the surface of the skin. Most are antiparasitic. The residual effects depend heavily on the nature and longevity of the active ingredient when in contact with the skin and exposed to local environmental challenges such as sunlight, rain, and sweating. A typical example is fipronyl, which is applied by a spray onto the skin in the treatment of chorioptic and other mite infestations (see below, p. 103). The solutions will adhere better to the skin and hair if they are applied after a degreasing wash and before the hair is completely dry. A few of the active ingredients adhere better to greasy skin and so the full implications of the rinse concerned should be appreciated before they are applied. Sometimes manufacturers will provide helpful advice in the instructions for use but often there is no specific advice for horses.

Powders containing synthetic pyrethroids (see below) have been manufactured mainly for the treatment of pediculosis (lice) but are generally inefficient and they should be used with caution. Inhalation of these powders by persons applying them is not an ideal circumstance!

Gels, lotions, creams, pastes and emulsions Various active ingredients are incorporated into these formulations with the intention of prolonging their effects. They are usually applied in a more focal manner and can contain various corticosteroids (used for localized anti-inflammatory effects), insect repellents such as benzyl benzoate, *Aloe vera* (for management of localized inflammatory responses) or protection of the skin from solar radiation (sunblock).

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Benzoyl peroxide is a follicular flushing agent designed for use in dogs. This material is a useful localized adjunctive wash in the management of bacterial pyoderma (and in particular bacterial pastern dermatitis) and localized areas of hyperkeratosis.

Table 4.4 shows the main washes and rinses that are available at the present time.

Insect control and management

Insects such as flies, mosquitoes, midges and ticks are a persistent cause of dermatological disease in horses worldwide, through a variety of mechanisms, and therefore their control is a major aspect of the prevention and treatment of skin disorders. Insects can affect the skin directly – the bites can be significant causes of worry, irritation, local reaction (e.g. bee or wasp stings), localized hypersensitivity reactions (e.g. insect bite hypersensitivity) and even severe systemic hypersensitivity reactions. For example, swarm attacks by *Simulium* sp. flies (black-fly) in the USA have resulted in deaths in horses. Furthermore, breaks in the skin and localized skin inflammation associated with exudation can provide some species of fly with an ideal place to lay eggs – the larvae that hatch here can cause significant disease. Insects cause worry through large numbers of insects on and around the horse; localized single or multiple bites/stings are common. Some insect bites cause serious local damage to the skin (e.g. spider bites) and others cause transient swellings/nodules and some degree of worry or

Table 4.4 Some of the main equine shampoos and topical rinses available and their main indications (CP = common practice use)

Group Nature	Types	CP	Indications/Comments
Cosmetic	Conditioning (colloidal oatmeal/lipids, fatty acids, chitosanoids)	✓	Repeated washing is required Best used when hair is fragile or brittle/dry and when seborrhoea is present
	Human hair shampoos	✓	Usually not appropriate for horses
Degreasing	Washing-up detergents		Degreasing of coat prior to use of medicated shampoo Not to be repeated
	Liquid soap		
Exfoliating	Selenium sulphide	✓	Seborrhoea/excessive scaling Keratinization defects Can cause dryness and epidermal hyperactivity
	Salicylic acid		
	Coal tar	✓	Also degreasing effects
Anti-pruritic	Colloidal oatmeal	✓	Adjunctive therapy for pruritic horses Note topical hydrocortisone aceponate and betamethasone also available
	Selenium sulphide	✓	
	Hydrocortisone		
	Diphenhydramine		
Antibacterial	Chlorhexidine 4%	✓	Antibacterial effects for skin surface, hair and in some cases follicular resident bacteria (pathogenic and non-pathogenic)
	Povidone-iodine	✓	
	Triclosan		
Antifungal	Chlorhexidine	✓	Dermatophytosis (trichophytosis/microsporosis) *Malassezia* sp. *Candida* spp. (*Candida albicans*)
	Povidone-iodine	✓	
	Miconazole/enilconazole	✓	
	Natamycin	✓	
	Nystatin		
Anti-parasitic	Synthetic pyrethroids	✓	Anti-ectoparasitic shampoos/rinses for control and prevention of insect infestations
	Malathion (0.5%)		
	Fipronyl (0.25%)	✓	
	Sulphur (lime sulphur)		
	Benzyl benzoate		

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pruritus. *Culicoides* (and probably some other species) also cause moderate to severe hypersensitivity responses that are probably the commonest 'allergic'/hypersensitivity disorders in horses (see Chapter 3).

Insects are also important vectors for viral, protozoal, fungal and parasitic diseases. Some of the resulting diseases are extremely serious and life-threatening, e.g. African horse sickness, equine infectious anaemia, equine encephalitides (WEE/EEE, VEE/West Nile virus), and epizootic lymphangitis (histoplasmosis). Surface-feeding and blood/lymph-feeding species of flies may be involved in the epidemiology of the equine sarcoid (see p. 387).

The majority of insect species are less significant but they do invariably cause some distress and worry for both horse and owner.

The implications for insect-related diseases are that prevention of contact between the vector/cause and the horse will break the natural history of the diseases concerned. Prevention of the cause is far better than cure – even when the damage is minimal. Therefore insect repellents and avoidance strategies are more significant methods of preventing the problem.

Having identified the species involved, control measures can be brought into force to prevent contact between the insects and the horse. In order to control the challenges it is important to know the life cycle of the insects concerned (see Chapter 3) – strategies can be formulated that focus on weak points in the life cycle or at least accessible vulnerable points in the life cycle.

Many general insect-controlling measures will involve potentially harmful environmental issues; in most circumstances the easy and effective options are by far the most environmentally unsound. For example, the widespread use of chemicals of the chlorinated hydrocarbon groups (DDT/BHC/dieldrin, etc.) to control fly and mosquito populations has led to major environmental, food chain and human health problems and so many of the most effective insecticidal chemicals have been removed from the marketplace. Even oral dosing with ivermectin can have a profoundly harmful effect on the pasture populations of insects and the water courses connected with them. Furthermore, even the most aggressive insecticides have seldom been effective in eliminating any species of insect under any circumstance and so alternative strategies have to be devised.

Additionally, many insects can travel considerable distances or be blown on the wind for up to 50–100 or more kilometres, and so any measures that are taken on one particular property may have an insignificant role in the reduction of the overall population numbers. Proximity to cattle farms, industrial areas and so on may mean that the challenge is virtually insurmountable. Then it becomes important to try to repel the flies from the individual horse.

Strategies may involve:

1. Elimination of the insect from the environment: This is often the most difficult control to carry out. Methods may include removal of the insects' breeding grounds such as stagnant water courses, old car tyres, forests, manure/bedding heaps. Properly composted organic material reaches sufficient heat to kill any breeding flies but anything less than this results in faster development. Placing black plastic over a manure heap may help to increase the temperature and keeps flies off.

Note

In the control of fly and mosquito populations the key word is *sanitation*.

It may be possible to remove bee hives and wasp nests from the proximity of horses to limit the chances of stings occurring. However, in many circumstances control of the general population of a particular insect species will not be possible.

2. Avoidance of contact either by removing the horse from the insect environment (such as stabling while insects are present) or by using 'clothing' or insect repellents and insecticides on the horse so that contact is at least minimized (see Fig. 4.3).

   Netting 'windows' in a stable and a ceiling fan creating a wind speed greater than 5 km/h, might prevent flies and mosquitoes from entering. Some flies will not enter or at least will be reluctant to do so (e.g. *Culicoides*) whilst others such as mosquitoes and stable flies will readily follow a horse into its stable or seek them out.
3. Moving the horse to another environment away from the insects or manipulating the environment so as to make it inhospitable to the insects. An example of this is the use of fans that create a wind speed of over 5 km/h in the stable – this is usually enough to prevent contact with *Culicoides* spp. midges. The horse may be moved from a low-lying field near a watercourse and wooded area (a suitable breeding site for *Culicoides* spp. and mosquitoes) to a high windy paddock where flies and midges cannot survive.
4. Killing the insects by use of traps and electrocution apparatus will minimize contacts but they are not very efficient. Baits and chemical traps can be more effective. The latter is a common mechanism for controlling tsetse flies, for example in Africa; pheromone traps draw the flies to a strong insecticidal mesh and the dead flies are removed immediately to prevent bird ingestion.
5. Rendering the skin of the horse inhospitable to the insects. This strategy, for example, involves close clipping in the case of *Chorioptes* spp. mites and application of organic oils (including benzyl benzoate) and greases that prevent insects from landing and/or feeding. Insect repellents form a major sales gimmick and many purport to eliminate or deter all insect contact. This is unrealistic.

In general, however, several of the measures need to be applied in almost all circumstances. Thus, for example, when a horse suffers from insect bite hypersensitivity ('sweet itch') simple stabling may not be protective as a few bites will cause serious problems – additional protection by clothing and direct repellents may be helpful. Reliance on 'clothing' or 'chemical' protection whilst leaving the horse in the environment where *Culicoides* midges are breeding is nonsensical.

As many controlling measures as can sensibly be achieved is the aim. Many of the possible strategies are, however, not feasible in many circumstances. An owner may not be able to move the horse away or the horse may not tolerate clothing of any sort and sometimes close clipping of 'feathered' legs is not an acceptable practice for owners of show horses.

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Note

The more tools you use for insect control the better it will be. No one method can be relied on to do the whole job and good sanitation is essential.

Chlorinated hydrocarbons These chemicals have potent long-term insecticidal effects but have largely been withdrawn on the grounds of environmental hazard. They persist in the environment for long periods (up to tens of years possibly) and many have serious implications for plant, bird, insect and animal life. Currently only malathion is available as a topical wash that is used sometimes to treat head lice in humans. Its use in horses is not permitted in many countries.

Organophosphates These have been completely withdrawn from the market and should not be used in spite of their potent insecticidal properties and impression of benign effects on the treated animal. Long-term human hazards are reported to be significant.

Fipronil This is a potent insecticidal phenylpyrazole compound that can be applied topically in a spray form and it has been shown to be effective in the treatment of chorioptic mange and pediculosis (lice infestation). The spray works best when applied to a nearly dry skin following a selenium sulphide wash. Topical 'spot' treatments for dogs and cats are not really useful in horses.

Pyrethroids These are based on a natural plant-derived parent molecule (pyrethrin) with insecticidal properties. Modern synthetic pyrethroids such as cypermethrin, permethrin and deltamethrin have been developed to improve their potency whilst still retaining their general lack of toxicity to animals. Synergistic chemicals including piperonyl butoxide are added to increase their potency. They are, however, extremely dangerous to fish and so should not contaminate natural water courses. Pyrethroid topical washes and powders (predominately permethrin) are available for treatment of ectoparasitic infestations. Permethrin, like the other pyrethroids, is very safe, and stable in ultraviolet light and heat. It is also valuable as an insect repellent and can be incorporated into brow fringes, blankets and tags/strips to provide some relief from insect attack (see Fig. 4.4). Similar materials can be hung in stables to limit insect populations in a closed space. There are also electrically driven vaporizers that heat cards impregnated with pyrethroid chemicals for environmental fumigation to prevent mosquito attack primarily.

One of the most effective mechanisms for the control of chorioptic mange mites in horses is the application of a pyrethroid-impregnated cat or dog flea collar around each pastern. This is not a substitute for proper elimination strategies but it can be of real help in prevention of reinfestation. The collar must be applied loosely and in such a manner that it cannot tighten, it *must* have an elasticized safety strip and it is wise to test the horse with a single one first to ensure that it does not react adversely to the collar.

Avermectin compounds There has been an increasing interest in the use of avermectin compounds in the management of parasitic skin infestations. Orally administered avermectins are very effective in treatment of the migrating and gastric larvae of *Gasterophilus* spp. flies (stomach bots, pharyngeal bots) (see p. 204). It has been reported anecdotally that oral or injected ivermectin also reduces the population of mites and some sucking lice such as *Haematopinus asini* and *Dermanyssus gallinae*. As might be expected, its efficacy is probably greater in parasites that suck blood or plasma or burrow in the skin. Surface-feeding parasites have a lesser exposure of course.

Injection of ivermectin carries a definite risk and there is no material therapeutic advantage over the oral form in these conditions – absorption from the gastrointestinal tract is extremely efficient. A dose of 200 μg/kg bodyweight administered orally and repeated after 14 days is suggested as being effective. In fact the method probably has only a marginal eradication effect on mites and lice. Rather they may have reduced activity and lay fewer eggs. Doramectin is an injectable avermectin that is relatively safe in horses. It appears to have a better overall effect on mites and lice than the other compounds when administered by deep intramuscular injection at 0.25 mg/kg.

Both ivermectin (at 200 μg/kg) and moxidectin (at 400 μg/kg) (and probably doramectin) have strong effects against circulating microfilariae and adult *Onchocerca cervicalis* parasites (see p. 221).

Topical application of a buffered artificial tears solution containing a drop of ivermectin injection is an effective method for treating ophthalmic forms of *Onchocerca cervicalis*, *Thelazia lacrimalis*, *Habronema musca* and *Setaria digitata*. Cutaneous habronemiasis and *Draschia* spp. infections (see p. 217) will usually respond well to a single topical application of a solution or cream containing a low concentration of ivermectin – direct application of the oral paste to the surface of the wound is an effective delivery method.

Insect repellents There is increasing concern over the use of insecticidal compounds – even extending to the pyrethroids. When this is taken with the fact that prevention of contact between the insect and the horse is by far the best approach, insect repellents have a pivotal role in the management of insect-related disease. Accepting the fact that insect breeding grounds should be either destroyed or at least controlled, the next major opportunity is to prevent the insect attacking the horse. This sounds a relatively easy process but in spite of a plethora of suggested materials including citronella, in-feed garlic, perfume etc., this is not anything like efficient enough. Some pyrethroids such as permethrin and deltamethrin have repellent properties and are used in fly tags, brow bands, blankets and in electrically powered (heat) vaporizers. Human 'insect repellent sprays' can be used but they seldom have a suitably long action and so they become both expensive and inconvenient.

Principles of immunotherapy in dermatology

The concept of immunotherapy relies upon the activation or stimulation of an inflammatory process using immunologically active agents. The 'beneficial' responses are due to altered tumour–host relationships improving the ability of the host to identify (immunologically) the tumour and reject it. Immune methods of treatment of skin disease are theoretically attractive but have strict limitations.

Immunotherapy can be effectively divided into two clinical categories, vaccination and immunomodulation.

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Vaccination

Treatment of some viral, bacterial and fungal skin disease by these methods is currently either universally useful or actively being explored. For example, treatment of virus papillomas by the use of an autogenous vaccine has gained a considerable reputation and the possible use of vaccines to prevent dermatophytosis is being explored with some promise. A vaccine is now licensed for use in horses in some parts of continental Europe. However, vaccines can only be expected to be effective when the response is directed effectively at the target organism or cell. Attempts to use autogenous vaccines for treatment of the equine sarcoid (on the assumption that it is caused by a virus which can be eliminated by antibody responses) have either consistently failed or proved unreliable. There is some suggestion that they may make the condition worse.

Immunomodulation

Active immunotherapy processes involve the production of antibody against tumour-associated antigens and the stimulation of the animal's intrinsic immune system. An example of this is the use of extrinsic proteins such as bacillus Calmette–Guérin cells or proteins or *Propionobacterium acnes* or acemanan (the active ingredient of *Aloe vera*). Active tumour-specific immunotherapy is not yet used in horses but is becoming more feasible in other species. So far attempts to produce a 'melanoma vaccine' from X-irradiated autologous tumour cells have not provided any convincing benefit. Melanoma vaccines with a BCG cell wall (Nomagen, American Home Products Corporation, Madison, USA) adjuvant is reported to have a beneficial effect (Jeglum 1997). In some cases such as the equine sarcoid, the process of crude autogenous vaccines may cause more harm than good.

Passive immunotherapy involves the delivery of previously sensitized immunological agents such as antibodies or, alternatively, cells that stimulate the production of antibodies against the tumour.

Restorative immunotherapy is a concept that involves the restoration of a depleted or impaired immune system in which there is a reduced functional capacity for either antibody production or recognition of abnormal cells. Drugs such as cyclophosphamide are used to inhibit cells (T lymphocytes and macrophages) that down-regulate mediated immunity – effectively therefore, they act as immune stimulants. This is supposedly the mechanism that is created by the H_2-blocking drug cimetidine that is reported to be an effective method of control of melanoma (Goetz et al 1990). Reports of efficacy are very mixed – some successes are reported (Hare & Staemphli 1994) but most cases fail to show any significant benefit. There is much controversy over the latter except where known immunocompromising processes such as hypercortisolaemia, immunodeficiency syndromes and drug suppression are present.

Vaccination

Indications

Vaccination for skin disease is not currently widely used with the exception of the equine viral papilloma ('grass wart'). Autogenous vaccines may be prepared directly from tissue removed from an active lesion and after suitable precautions during preparation to ensure sterility and safety the material is injected intramuscularly.

The vaccination method works well for some forms of juvenile viral papilloma but is contraindicated in the treatment of sarcoids. Although some reports have suggested that a benefit can be obtained in isolated sarcoid cases (Wheat 1964, Page & Tiffany 1967), other cases may become dramatically worse with the development of multiple or miliary lesions.

Method

A small portion of a papilloma is obtained by surgical excision and placed in a sterile plastic container for direct delivery to the laboratory. If delays are anticipated, the sample should be placed on ice until it reaches the laboratory. The vaccine is prepared by homogenization of the tissue and the virus is inactivated by sterilization (usually using formalin). Adjuvants can be added. The vaccine is tested for sterility before dispatch. Two (or more) injections are given 1–2 weeks apart. The responses are usually obvious by 3–6 weeks following the second injection.

Advantages

- Simple
- Cheap
- Effective for viral papilloma, particularly if multiple
- Creates long-term protective immunity.

Disadvantages

- Only for use in the same horse (autogenous)
- Very limited efficacy range
- Unpredictable quality/efficacy.

'Stimulation'/alteration of the immune system (immunomodulation)

These methods rely on the intense immunological response following local injection of protein cell wall extracts from mycobacterial organisms or killed or live mycobacteria, usually bacillus Calmette–Guérin (BCG). Cell wall extracts can be crude (usually 'milky' in appearance) or refined and are available commercially. Viable freeze-dried BCG reconstitutes to a clear aqueous solution whose concentration and volume can be varied by appropriate dilution.

Several other forms of mycobacterial protein have been developed in response to the concept that the viable (live) bacillus is likely to cause an anaphylactic response that may be fatal. The alternative approaches, said to be 'free of adverse effects' include purified cell wall extracts (Regressin-V, Vetrefarm Research Inc, USA; Ribigen-E, Ribi Immunochemical Research, USA) and protein-free extracts of cell skeleton (Nomagen, American Home Products Corporation, Madison, USA). Other bacterial extracts including a heat-killed protein preparation of *Propionobacterium acnes* (Eqstim, Neogen, USA) have been developed to exploit the effect for treatment of certain cutaneous neoplasms including the equine sarcoid and squamous cell carcinoma. However, there are limitations to their

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use in both conditions and most of the commercial products have fallen into disuse or been withdrawn after problems with production or use.

These protein and protein-free extracts have no direct effect on the cells themselves. The therapeutic effects rely on specific and non-specific effects. Simplistically, the latter non-specific effects occur when the protein material probably binds to cells directly and a delayed hypersensitivity response is mounted locally. The proteins are also intensely immunogenic – ingress of T cells, neutrophils, macrophages and NK cells results in tumour cells being killed 'incidentally' or non-specifically in a massive inflammatory response. T-cell recognition of the tumour cells may result in a longer effect. Therefore, the method is restricted to individually treated lesions – there is usually no detectable effect on untreated lesions on the same animal. Furthermore, it is probably only applicable to discrete tumours – diffuse surface sarcoids of the occult and verrucose types, for example, respond poorly while nodular lesions show a good effect (particularly around the eye). Treatment of multiple lesions is therefore difficult and requires a large volume of the protein. The material has little or no effect if injected around a lesion and requires to be injected truly intralesionally (Theon 1998). There is also a reported risk of anaphylactic reactions following repeated injections. Ideally, therefore, it should be injected intralesionally after suitable premedication has been given to prevent untoward systemic reactions (Rebhun 1987).

Several protocols are commonly used. This method has a good record against periocular nodular sarcoid, somewhat less effect on fibroblastic sarcoid, and is probably impractical for verrucose or occult sarcoid (Knottenbelt & Kelly 2000). Sarcoids away from the periocular region have a poorer response and those on the limb may be exacerbated by the procedure. The method has a poor record against squamous cell carcinoma in horses (whereas in cattle it has a significant if somewhat unpredictable beneficial effect). BCG and the related immunological methods are not effective against many of the other tumours including melanoma and neurofibroma.

Intralesional injection seems to be vital for satisfactory results with sarcoids and it is virtually impossible to inject a significant volume of the material into the skin surface of the verrucose and occult lesions. A McClintock type (TB-Testing Automatic 0.1 mL dose) or a dental syringe can be used to spread the material within the lesion (Fig. 4.5).

Attempts to treat equine sarcoid lesions with an undefined 'immunostimulant' failed to show any convincing benefit beyond a placebo effect (Studer et al 1997).

Surgical debulking of sarcoid lesions, in particular, prior to administration of BCG has been reported to improve the overall results (Theon 1998), but in the author's experience this is not repeatable. In general it seems unwise to use debulking with a sarcoid in particular.

Indications

The method has a good reputation for treatment of nodular and fibroblastic sarcoids around the eye but, somewhat surprisingly, is much less reliable elsewhere (Lavach et al 1985). Indeed there is some suggestion that its use away from the head may induce a more aggressive growth of the tumour mass. There is no apparent effect on lesions away from the injected ones (Owen & Jagger 1987, Vanselow 1988) – each lesion requires its own tumour-volume-related injection, making it a tedious and very expensive procedure for general use for sarcoid.

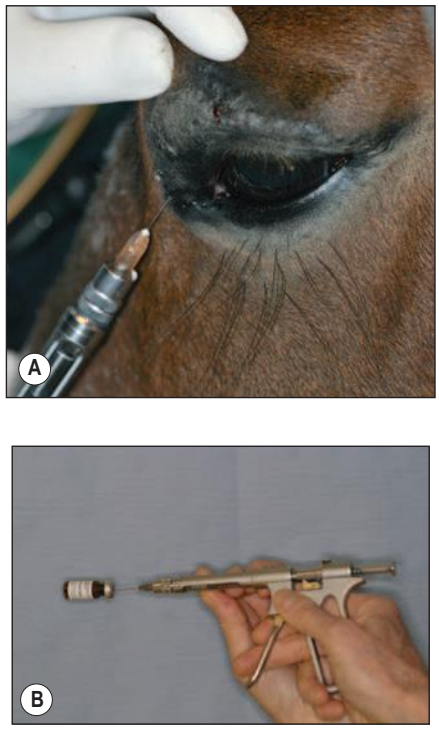


Figure 4.5 **(A)** A solution of BCG being injected into a modular sarcoid in the eyelid using a pressure volume-limited syringe **(B)** that dispensed 0.1 mL with each click. Similar equipment is used to perform intradermal injections during TB testing in cattle (TB syringe). Note the Luer-lock needle attachment, which prevents the system from bursting when very high pressure is applied.

Treatment of squamous cell carcinoma with BCG has a poor overall success rate; in the author's experience, less than 20% of squamous cell carcinomas will respond at all and even fewer will be 'cured'.

Method

Several different methods have been advocated and the chosen one will reflect the material used and the type of lesion being treated. In most protocols it is usually necessary to use up to 4–10 injections, although there are reports of single or two injections giving rapid, total resolution (Wyman et al 1977, Vaneslow et al 1988). Although it has been suggested that the effect is dose related (larger tumours need more material), the proper placement of the reconstituted proteins seems equally or more important.

There is also a genuine risk of anaphylaxis although this can be minimized by careful placement of the material and by suitable steroid and non-steroidal premedication (see protocol, Table 4.5).

- Lesions may be injected with reconstituted BCG vaccine or purified cell wall extracts at 7–10 day intervals.
- The protocol used by Liverpool University employs intralesional injection of the reconstituted freeze-dried,

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live BCG organisms at intervals of 7, 14, 21, 28, 28, 28 days. Thus the second injection is administered 1 week after the first. The third injection is given after a further 2 weeks and the fourth after a further 3 weeks. This protocol is described fully in Table 4.5. The advantage of this method is that fewer doses are used and the risks of anaphylaxis are minimized. It is also far cheaper than many of the other weekly regimens that use much larger doses of the BCG itself.

Note

- True intralesional injection is critical to the success and the avoidance of dangerous systemic/anaphylactic responses.
- The material is injected into the lesion with a fine needle and infiltrated to avoid reflux of the fluid. The volume of injectate should be adjusted to permit maximal antigen delivery into the tumour mass and the tumour–tissue interface.
- Injection is sometimes highly problematic due to the density of the tumour (especially in the case of sarcoid or neurofibroma).

Responses observed

- Local swellings are common.
- Repeated injections result in either exaggerated responses with further swelling or progressive shrinkage of the mass.
- Some nodular and fibroblastic sarcoids react by abscessation and discharge of purulent material; this may result in a chronic discharging sinus tract requiring surgical curettage.
- Anaphylaxis has been reported following repeated use of attenuated live BCG in particular. The first two injections are probably safe (provided that the method has not been used previously on this or another lesion) but thereafter appropriate precautions should be taken. This danger may be minimized by using BCG cell wall vaccine preparations and/or premedication with corticosteroids (e.g. dexamethasone at 0.05–0.1 mg/kg intravenously) and flunixin meglumine (at 1.1 mg/kg intravenously) at least 30 minutes before the injection is given. Antihistamines, such as tripelennamine hydrochloride (at 1 mg/kg intramuscularly), have also been used to premedicate horses prior to BCG injection. The value of the antihistamines is doubtful. True intralesional injection apparently also helps considerably.

Treatment of anaphylaxis Careful preparation must be made prior to the injection of any BCG or similar products, to permit immediate treatment of an untoward response. The reactions may develop instantly upon injection or within 20–40 minutes afterwards and so the operator should be ready in either case to deal with the emergency.

The preparations should include:

1. adrenaline (epinephrine) solution for injection (1:10 000 is usually available in 1–3 mL ampoules)
2. oxygen with delivery/demand valve
3. dexamethasone solution for intravenous injection (2 mg/mL) (treatment may require a dose of around 0.2 mg/kg).

Table 4.5 University of Liverpool protocol for periocular, nodular (and some fibroblastic) sarcoids (the method is not considered valuable for other types of sarcoid or for other neoplastic disorders)

1	Evaluate the history, size and type of tumour to be treated.
2	30–45 minutes prior to every single injection (including the first) the patient is premedicated intravenously with corticosteroids (e.g. dexamethasone at 0.05–0.1) mg/kg) and flunixin meglumine (at 1.1 mg/kg). *Note:* An alternative method should be considered if there is a history of use of the same method, or if the horse is known to have had an adverse reaction previously Antihistamines are of dubious value and are not used routinely.
3	Sedate and restrain the horse.
4	Perform careful surgical preparation of the site of injection.
5	Dilute one ampoule of lyophilized BCG viable bacillus into an appropriate volume of water (for small tumours <0.5 cm diameter the contents are diluted in 0.5 mL of water; for larger tumours one ampoule can be reconstituted with 1–1.5 mL) and inject intralesionally. *Use a 1 mL tuberculin-type syringe (or a dental pressure syringe with a Luer-lock needle) and 24–26 G needle of an appropriate length.
6	Repeat injection after 7 days (second dose after 1 week).
7	Repeat injection after 14 further days (third dose after a further 2 weeks).
8	Repeat injection after 21 further days (fourth dose given after a further 3 weeks).
9	Repeat injection after 28 further days (fifth dose after further 4 weeks).
10	Further doses are repeated at 4-week intervals.
11	Up to nine or ten injections may be required (5 is 'average').

* It is imperative that the entire contents are injected *intralesionally*. Subcutaneous injection in the vicinity of a sarcoid is to be avoided as far as possible as there is a risk of anaphylaxis.

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Note

- The treatment method has much to commend it but there are a significant number of failures and definite (however small) risks.
- The attending veterinarian should remain with the horse for 2 hours after the injection with adrenaline readily available.

Dosing should not stop until a suitable response is evident. Significant reactions developing after 3–5 injections indicate that treatment can be either stopped or delayed to allow assessment of the responses. A few cases suddenly resolve some months after the method has been abandoned due to lack of efficacy. The reasons for this are not clear.

Localized abscessation can develop and can be alarming with a persistent discharging sinus developing for over 12 months. Total surgical excision of the abscess may be the only reliable treatment to resolve this problem. In other cases the tumour gradually shrinks with the full effect taking up to 12 months. BCG treatment of sarcoids results in a very acceptable cosmetic effect and is safe for both operator and patient.

Best results are obtained in nodular sarcoids in and around the eyelids (Fig. 4.6). There is no detectable effect on untreated sarcoids on a horse on which one or more individual lesions are treated with BCG.

Advantages

- Convenient/simple and little equipment required
- Cheap (for single or few lesions)
- Can be administered to standing, sedated horse
- Effective in over 70–100% of palpebral nodular sarcoids.

Disadvantages

- Risk of anaphylaxis (Hepler & Lueker 1980). This is a serious if very rare complication. True intralesional injection using a suitable proportionate volume/concentration of BCG will minimize the risk.
- Repeated injections are required (increasing the risk of anaphylaxis and adding to the overall cost).
- Volume/mass dependent (larger lesions need more material). However, this may be less important than the distribution of the material within the lesion. Over-dilute solutions created by adding too much diluent reduce efficacy significantly.
- No effect is detected on lesions remote from the injected one, i.e. every lesion requires specific injection. This may be highly problematic and very expensive if there are many lesions.
- Must be injected intralesionally. This largely precludes its use in verrucose and occult lesions where intralesional injection is either impossible or very difficult.
- Limited efficacy range (not effective for squamous cell carcinoma or melanoma); little effect on sarcoids of verrucose or occult types or tumours of all types away from the periorbital region.
- Contraindicated for sarcoids on limbs. The reason for this is not clear.

General notes on treatment using immune methods

- Autogenous vaccines are strictly limited to the 'viral wart' or viral papilloma. Most of these cases will resolve spontaneously in around 8 weeks in any case, so there is no real advantage in their use.
- Use of autogenous vaccines for neoplastic lesions is entirely illogical – if it were as simple as this there would be no neoplasia. Furthermore, the use of autogenous vaccines in sarcoid therapy may be positively dangerous – this approach is contraindicated and it should never be used. New vaccine technologies are being explored but so far none has given any tangible benefit.
- Injections of freshly prepared BCG must be made for each administration and the dose should be related to the size and type of tumour.
- Squamous cell carcinoma in horses is much less responsive to BCG injections in all its forms than in cattle.
- *Corynebacterium parvum* can also be used effectively but is less predictable and is less available.

Immune 'stimulants'

Immune stimulants and immunomodulating compounds are an attractive concept. Immune-mediated diseases or those in which the immune process is in some way impaired (such as sarcoidosis) seem to be the ideal theoretical objective of such therapy. The major problem is that



Figure 4.6 This pony received a series of four injections of BCG into the fibroblastic palpebral sarcoid **(A)**. A significant reaction with marked swelling at the site developed after the third injection of 1 mL (1 ampoule only) and at the time of the fourth (which also showed a significant but transient swelling), it was noticeably smaller **(B)**. Following the fourth injection, the lesion rapidly disappeared with no recurrence at least up to 9 years **(C)**.

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no work has established the certain value of any of these chemicals. The commonest compound in use in the horse is levamisole. This is used widely as a livestock anthelmintic and is cheap and available. The efficacy of the drug is very doubtful, with little clinical evidence of improvement in immune process.

A number of protein derivatives of the mycobacterial cell wall are used as general, systemic stimulants of the immune system. They are injected intramuscularly or subcutaneously and have ill-defined biological effects. Most of the commercially available compounds have shown a singular lack of predictability of effects and side-effects and have been withdrawn from the market. There are, however, some good reports of their effect on tumours such as melanoma (see p. 421).

General immune stimulation of a scientifically unreported nature is supposedly derived from administration of several homeopathic medications. There are extensive anecdotal reports of the value of *Aloe vera* in particular but these are widely and justifiably viewed with some scepticism. The wide range of therapeutic claims for this and other natural medicines suggests that there is a basic biological response which could be beneficial under some conditions. The absence of critical scientific analysis of the effects of the materials needs to be addressed before any recommendations can be made. Most makers of these products are not willing to subject them to a proper blinded clinical trial and one can only surmise the reason for this reluctance.

Note

- There are also some less defined herbal products that are purported to stimulate deficient immune systems such as *Echinacea*, devil's claw and a number of other plant extracts. The value of naturally derived (plant) medications cannot be overlooked – many of our mainstays of medical practice have their origins in these, but so far there is little to suggest any benefits.
- The homeopathic approach to the immune system has neither detectable nor scientifically explicable effect and its effects are certainly no better than a placebo effect. Furthermore homeopathy has a devious way of explaining any outcome regardless of whether there is an improvement, deterioration or no change. Conventional medicine is not permitted to function on the same criteria. These methods have no scientifically proven results and evidence-based medicine suggests that they either have no effect at all or are, potentially at least, harmful because their use may delay proper intervention and so make the prognosis correspondingly worse.

Principles of dermatological chemotherapy and cancer medicine

Chemotherapy refers to the use of drugs to treat neoplastic disease. Although the options for chemotherapy in horses are limited, new concepts are developing as novel drugs and approaches are recognized. Several factors including the cost, the logistics and the untoward side-effects have limited the use of chemotherapy in horses. Although systemic chemotherapy is now well established in many other veterinary species and in humans, the use of antineoplastic drugs has not been widely practised in horses and there is little published evidence to support their use. The unacceptable side-effects and the exorbitant costs largely preclude their use as systemic agents. Therefore, currently chemotherapy is largely limited to topical applications and intra-tumoral chemotherapy for cutaneous tumours. The high prevalence of cutaneous tumours in horses makes this approach logical. The local approach is used to obviate the harmful effects of the drugs, reduce the cost and maximize the exposure of the tumour to high concentrations of the drugs whilst minimizing the exposure of normal tissue. Suitable drugs must have a steep dose–response curve, i.e. they must have a high response for a low dose. For this reason carriers can be used to prolong the effects of the drug, i.e. to hold the drug in the location where it is placed. Other strategies for enhancing the effects of local chemotherapeutic drugs include temporary occlusion of the blood supply, 'Botox' injections locally, cooling and sundry other supportive mechanisms.

Chemotherapeutic agents generally act by altering or stopping cell replication through damage to nucleic acids (DNA and or RNA) (Table 4.6). This restores apoptosis and prevents cell replication. The expectation is that this will then result in tumour regression. However, it is important to realize that the effects are not discriminatory for tumour cells and so all the chemotherapeutic agents will have some untoward effects on normal tissues. In theory at least the drugs exert a greater effect on the cells that are undergoing more rapid replication – however, some tumours are slow growing – the cells have a long cell cycle and so the effects on surrounding rapidly dividing cells (such as bone marrow, enterocytes, testicular cells, etc.) may be greater than expected. There are other aspects of the drug activity that may limit these effects including affinity of the drug for particular cell types. Ideally a drug should be cell-type specific and have a complete destructive effect without any side-effects. Currently there are no drugs that fulfil these criteria.

Currently the options include cisplatin (diamino platinum), which has a broad spectrum of activity against epithelial and mesenchymal tumours. The compound inhibits DNA synthesis and is currently mainly used to treat squamous cell carcinoma and sarcoid (Theon 1998). The efficacy of the material relies upon fresh preparation and formulation of stable emulsion in medical grade sesame oil. An aqueous solution is difficult to create but this is then mixed with sesame oil to create a stable emulsion containing 3.3 mg cisplatin per mL (10 mg cisplatin is dissolved in 1 mL water and 2 mL sesame oil). This emulsion is injected intralesionally to deliver 1 mg cisplatin per millilitre of tumour every 2 weeks for three or four applications. It is reported that a series of parallel lines of the emulsion is injected into the tumour to allow a high concentration to be delivered. However, many sarcoids are very dense and so are less amenable to the use of intralesional injection. Leakage from the site is common and as the material is very mutagenic and carcinogenic the technique requires very special care. It is not considered applicable to general practice use on safety grounds and should be restricted therefore to specialist oncology centres. The results, however, can be impressive (Theon et al 2007).

The choice of treatment for any condition will depend on many factors but the overriding concern is for the welfare of the animal and the evidence-based data about its condition. If, for example, the only available method of treatment

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Table 4.6 The major classes of chemotherapeutic drugs available in veterinary medicine (after Mair & Couto 2006)

Drug category	Activity	Examples	Dose	Route	Interval	Dermatological value
Alkylating agents	DNA changes and altered cell replication cycles	Carboplatin				N/K
		Chlorambucil	20 mg/m^2	i.v.	14 days	N/K
		Cisplatin	1 mg/mL tumour	i.l.	14 days	***
		Cyclophosphamide	200 mg/m^2	i.v.	14 days	N/K
		Melphalan	N/K		N/K	N/K
		Lomustine	N/K		N/K	N/K
Antibiotics with anti-tumour activity	Damage DNA/ interfere with nucleic acid synthesis	Actinomycin	N/K		N/K	N/K
		Bleomycin	N/K		N/K	*
		Doxorubicin	N/K		N/K	N/K
		Mitoxantrone	N/K		N/K	N/K
Plant alkaloids	Mitotic disruption	Vinblastine	N/K		N/K	N/K
		Vincristine	0.5 mg/m^2	i.v.	7 days	N/K
Anti-metabolites	Alteration of DNA/ RNA synthesis	Cytosine arabinoside	200–300 mg/m^2	s.c./i.m.	14 days	N/K
		L-Asparaginase	10 000–40 000 i.u./m^2	i.m.	14–21 days	N/K
		Methotrexate	N/K	N/K	N/K	****
		5-Fluorouracil	5 mg /mL tumour	i.l.	48 h	****
Taxanes	Alteration of mitosis	Taxol	N/K	N/K	N/K	n/k
Hormones	Immunosuppression	Prednisolone	1–2 mg/kg	Oral	48 h	****
Others		Cimetidine	3.5 mg/kg	Oral	q 12 h	*

N/K = no known studies; i.l., intralesional; * = relative value for dermatological oncology. Note that in horses little is established in the use of many of the suggested drugs.

has no chance of success then there is little justification in embarking upon it. Similarly in the case of a systemic neoplastic disease, there is little point in starting unless the full investment in terms of effort, care, persistence and finance is applied. Whilst the owner may be desperate for a solution/cure, the veterinarian needs to be very careful how he or she is advised; it is easy to be swept up in the emotions of the case and embark upon a hopeless regimen.

Figure 4.7 shows the decision-making tree that could be employed when considering the management of a neoplastic disease; the owner plays a major role in the process but is not the decision maker.

Following this protocol, for example, for a horse with an uncomplicated squamous cell carcinoma of the penis, surgical treatment can be justified because there is a proven effective method that provides an acceptable extension to the animal's life while at the same time improving the quality of its life. A more severe case warranting an en bloc resection of the entire prepuce and penis is a vastly bigger surgery with much higher costs and greater immediate dangers. The owner may not be prepared to tolerate the cosmetics of the procedure and then the decision would have to be taken as to whether the condition can be managed palliatively in such a way as to give the animal some useful and acceptable extension to its life. If the horse had palpably enlarged iliac and inguinal lymph nodes and pulmonary metastasis, the treatment may not be justifiable even if the owner was prepared to support the treatment.

There is documented evidence that radiation treatment of periocular sarcoid is an effective and highly successful treatment and therefore provided that the owner was prepared to pay for it, that course gives the animal the best long-term prognosis. As soon as the owner declares that they cannot pay for the best possible treatment the veterinarian is in a dilemma. Lesser treatments carry a lesser prognosis but the disease is likely to advance to the point where it becomes a

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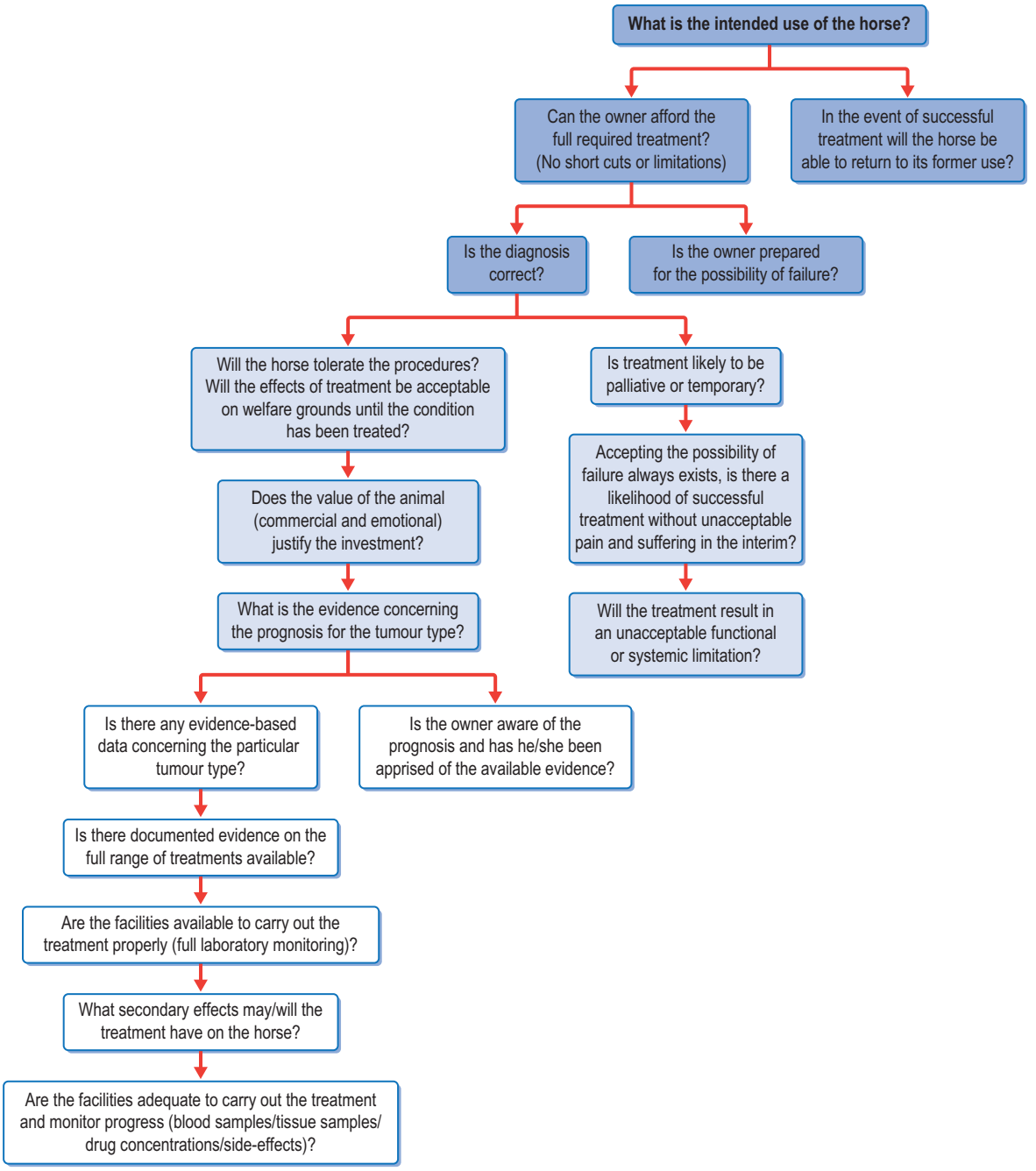


Figure 4.7 A decision-making tree for the management of a neoplastic disease.

welfare issue. Economics are a very important issue but it is the welfare of the horse that is paramount.

Cytotoxic/antimitotic therapy

Antimitotic therapy can be divided broadly into topical and systemic applications. Although, in theory, systemic antimitotic drugs such as cyclophosphamide and vincristine can be used to treat neoplastic and autoimmune skin disease, they are probably impractical in the horse under normal clinical conditions. Applications to skin neoplasia are even more restricted. Furthermore, the most aggressive tumours of the lymphosarcoma group are not currently considered treatable. Cutaneous histiocytic lymphosarcoma carries a somewhat better prognosis anyway and systemic therapy probably has little effect on the outlook.

Topical treatments have included 5-fluorouracil, cisplatin, cimetidine, methotrexate, carmustine, and cytosine arabinoside. Only the first three have been reported in significant numbers of tumours and even then the range of tumours involved is narrow. Several older materials have also been used but their use is gradually fading as new formulations and new molecules are developed to treat specific disorders.

Management of some cases of cutaneous lymphosarcoma by use of systemic chemotherapy has been reported (Couto 1994). This protocol included a triple drug approach. Cytosine arabinoside at 200–300 mg/m^2 was given subcutaneously every 1–2 weeks with chlorambucil at

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20 mg/m^2 orally every 2 weeks alternated with the cytosine. Prednisolone was given orally at 1–2 mg/kg q 48h. It is possible to substitute cyclophosphamide at 200 mg/m^2 intravenously once every 2 weeks for the chlorambucil. There are far too few reported cases and evidence-based reports on the outcome and until these are available and involve significant numbers of animals with comparative regimens, the justification for the treatment has to be questioned. It is important to realize too that many cutaneous lymphoma cases have a much better prognosis that true multicentric lymphosarcoma.

Note

The whole concept of equine chemotherapy is many years behind the other species – possibly justifiably but certainly disappointingly.

Veterinarians should remain sceptical of statements to the effect that any particular treatment 'usually works' when the number of cases in a series is very small. Nevertheless the only way to get large series is to encourage multicentre blinded trials.

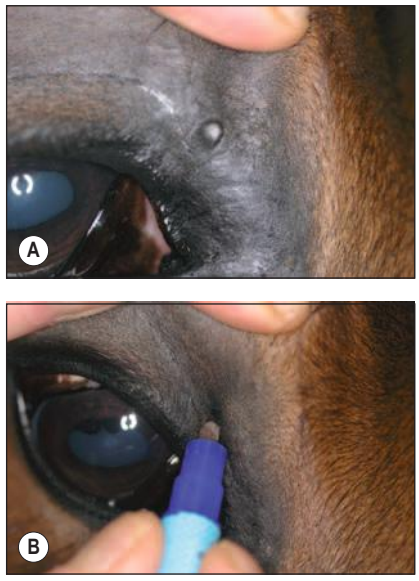


Figure 4.8 This very small skin lesion **(A)** was identified on a horse with many other sarcoids. A specific diagnosis was not attempted but the lesion was treated once with a styptic (silver nitrate) pencil **(B)** and disappeared over 3 weeks.

Topical/local therapy

Although surgical (or other excision) is the standard approach to many cutaneous neoplasms in particular, this is often unsuccessful and recurrence of the tumour is a frequent complication. For this reason, as well as for reasons of convenience, cost, feasibility and risks, topical and local methods are commonly employed either on their own or in conjunction with surgery (Theon et al 1997). There is singular advantage in local treatment of a single or few neoplastic lesions provided that the treatment carries at least as high a prognosis as other methods. As always, the selection of treatment will depend on many factors but increasingly topical and intralesional therapy is gaining credibility.

Topical cytotoxic and antimitotic agents form an important group of pharmaceuticals. Several forms are available but are currently effectively limited to treatment of viral papilloma, equine sarcoid, melanoma and some squamous cell carcinomas. Many and varied compounds of inorganic tissue poisons such as arsenic, mercury, platinum, and antimony and combinations of these have been used to treat cutaneous neoplasms including sarcoid and squamous cell carcinoma in particular. More recently, the use of podophyllin, methotrexate, fluorouracil and thiouracil and cisplatin has been described. All require some form of repeat applications, some on a daily basis for as long as 30 days.

Silver nitrate

Silver nitrate is cytotoxic and topical application is a very old method for treatment of very localized, small and superficial sarcoids, papillomas and skin tags. A solid silver nitrate 'styptic pencil' is the commonest way it is used (Fig. 4.8). The pencil is damped first and then applied directly to the mass. A single application is usually all that is required but a repeat application can be made 5–10 days later. The treated area will turn black and leathery and will slough away over some weeks, leaving an imperceptible scar. Larger areas should not be treated in this way and contact with human skin will cause blackening and superficial localized sloughing. Although this is an old method it still works and is simple and very cheap.

Podophyllum

This is a topical wart treatment for human use; it can be effective against a few localized and very small verrucose sarcoids and viral papillomas situated on the eyelids and periorbital skin. Limited effects make this almost a waste of time but there may be some applications in small superficial reactive hyperplastic tissues.

Formalin paste

A formalin-based paste is marketed in some parts of the world for the management of superficial tumours and sundry other indications. The material is intensely toxic to the skin and causes localized necrosis with formation of an eschar which sloughs in time. Like the other forms of topical treatment its use should be limited to papillomas and skin tags. It should certainly not be applied to open or granulating wounds and any contact with sensitive tissues such as the eye is potentially disastrous.

Cisplatin

Cisplatin (Platinol, Bristol Myers, USA; CisPlatin, Baker Pharmaceutical, UK) is an inorganic platinum-containing (heavy metal) molecule with strong broad-spectrum antineoplastic properties which are independent of the cell cycle; it acts by directly binding to DNA and inhibits its synthesis. The compound has a relatively narrow therapeutic range so doses are important. In species other than the horse, cisplatin is used widely as a systemic cancer therapy but there are serious potential side-effects on neurological, gastrointestinal and renal function. The need to target localized skin tumours in horses has led to the development of stable emulsions, impregnated sponges and slow release beads for placement in localized tumour sites. In this way the local concentrations are maintained for a more effective

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period and the risks of toxicity are minimized – the overall dose administered to the horse usually remains very low and well under the toxic upper limit.

Cisplatin is water-soluble and is available in this form commercially as a solution containing 1 mg/mL. The objective for all methods is to implant 1 mg cisplatin per cubic centimetre of tumour (i.e. approximately 0.3–0.4 mL of emulsion per cm^3) and to retain that in situ for as long as possible.

The dangers of cisplatin include mutagenicity and carcinogenicity and so these procedures should only be carried out in suitably equipped and licensed premises. Suitable operator protection includes double gloving, masks, caps, eye protection, aprons and a disposable overall suit. Contamination of the environment can occur and this is a potential extreme hazard. Horses should be hospitalized throughout the procedure and suitable disposal of faeces and urine (and contaminated bedding) has to be made.

Preparation of a stable emulsion

1 *Using pure cisplatin*. The best described uses in horses relate to the use of a stable emulsion prepared in medical grade sesame oil that delivers an overall dose rate of 3.3 mg/mL of emulsion. This can be prepared by mixing 10 mg pure cisplatin in 1 mL of water and 2 mL of purified filtered medical grade sesame oil, under pressure using a pumping method from one syringe to another via a three-way tap; after around 1 minute the emulsion should develop. This should remain stable without separating out on standing. This emulsion contains 3.3 mg/mL. Injection is made using a Luer-lock pressure syringe and a 20G needle.

Note

All forms of cisplatin are hazardous (carcinogenic) to humans but pure cisplatin is extremely dangerous and must only be handled by a properly qualified dispensing and formulating pharmacist with proper handling facilities.

Injection using only freshly made emulsion is made, either in the standing sedated horse or under general anaesthesia depending on accessibility of the site. As the emulsion is injected, the needle is withdrawn. In theory at least, lines of the emulsion are laid 1 cm apart and 1 cm in different planes at right angles. This may sound an easy process but leakage and the density of most sarcoids make this technique very difficult. Leakage is taken to indicate an 'adequate dose'! The process is repeated every 2 weeks or so for four to six injections (Theon 1998).

2 *Using a commercially available aqueous solution containing 1 mg/mL*. This material can be injected directly into the tumour but the requirement for 1 mg/cm^3 means that 1 mL of the solution must be infiltrated into 1 cm^3. Actually this is almost impossible; the potency of this concentration is low and the leakage and rapid absorption make this virtually useless. Furthermore, the injections must be repeated regularly over some days or weeks. Inclusion of adrenaline (epinephrine) in the solution at 1:100 000 may delay the redistribution somewhat. This system is also highly impractical.

3 *Impregnated biodegradable sponges and beads using concentrated cisplatin*. These are very logical and easier to handle than the injection forms. The sponges/beads are implanted into the mass to be treated usually after surgical removal and are left in situ either to degrade or to be removed after a defined time. The sponges will decay naturally, releasing sustained concentrations of cisplatin locally. Beads will release the cisplatin slowly but may need to be removed on completion of the course.

Slow-release biodegradable beads (3 mm diameter) made from calcium sulphate and dextran sulphate containing 7% cisplatin have been described in a study of treatment of various cutaneous neoplasms including spindle cell tumours/sarcoid, squamous cell carcinoma, melanoma and other tumours. Each bead contained 1.6 mg cisplatin and was placed at 2 cm intervals in the tumour bed (where debulking had been performed) or through the central portion of the tumour where tumours were < 1.5 cm diameter and debulking was not considered necessary. The overall success rate was over 83% (Table 4.7). The method appears to be far safer, more rational and far easier to perform than other methods using the material.

There are, however, very few reported series of cases subjected to these methods (Southwood & Baxter 1999, Hewes & Sullins 2006) and so it is difficult to suggest a true prognosis. Studies have also suggested that combination of cisplatin treatment with local interleukin-2 (IL-2) infiltration improves the prognosis for cases receiving intratumoral injection (Spoormakers et al 2003).

Cisplatin treatment is indicated for the treatment of several types of solid neoplastic masses that are not amenable for one reason or another to surgical removal. Reports suggest that it is an effective method for treatment of some types of sarcoid, squamous cell carcinoma, melanoma, soft tissue sarcoma, cutaneous lymphoma and hamartoma. Small masses appear to be more susceptible and carry a much better chance of success (see Table 4.7). Larger masses may need to be surgically debulked before cisplatin is introduced and where this is not feasible, then a more aggressive protocol with extra volume and extra doses may be successful (Theon et al 2007). Some tumours such as squamous cell carcinoma at mucocutaneous junctions at the mouth and eyelid or vulva appear to have a lower success rate. Overall success rates, however, for all tumours of all types and sizes are reported to carry a prognosis of 75–100% remission after 2 years with excellent cosmesis (Theon 1998). This is of course exceptionally promising so it is surprising that the method has not achieved a higher and more uniform success rate amongst various oncology centres. There are, however, very few scientific reports of its use but it has been shown to be effective when administered under a strict protocol which includes safety measures for the operators (Theon 1997) (Fig. 4.9). The drug is seriously toxic (nephrotoxic) and should be limited in overall volume/weight at any one time, although the small amounts used to treat localized tumours are probably unlikely to have any material harmful effect. It has a limited availability.

5-Fluorouracil

5-Fluorouracil is an anti-metabolite with direct cytotoxic effects and a usefully adjunctive contact allergy (delayed

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Table 4.7 The results obtained by cisplatin injections using a stable oily emulsion at 1 mg cisplatin/cm^3 or biodegradable beads (Theon 1998, Knottenbelt unpublished results, Hewes & Sullins 2006)

Tumour type	Number of cases	Tumour diameter (cm)	Concurrent debulking surgery	Success (%)	Reference
Sarcoid	27	⩽5	No	93	1
	65	5–30	No	24	2
	31	Not stated	Yes/no*	91	3
Squamous cell carcinoma	312	3–8	No/yes*	72–89	1
	15	1–15	No/yes*	33	2
	11	Not stated	Not stated	60	3
Melanoma	72	2–3	No	72	1
	23	1–24	No	0	2
	14	Not stated	Yes/no*	93	3
Cutaneous lymphoma	6	⩽5	No	100	1

*Debulking performed for larger tumours or in smaller masses in sites where excision/debulking was possible.



Figure 4.9 This very invasive fibrosarcoma/sarcoid tumour responded well to repeated injections of a stable wax containing 10 mg cisplatin/mL. **(A)** and **(B)** Start. **(C)** 14 months post-treatment.

hypersensitivity) activity. It selectively kills or impairs rapidly proliferating skin tumour cells through competitive inhibition of thymidylate synthetase activity (essentially resulting in a severe intracellular thymine deficit in the cells) and so the synthesis of DNA is prevented in the treated cells. It is available as a 5% topical cream base and as an aqueous solution of 25 mg/mL in multidose vials.

Squamous cell carcinoma cells are more thymine reliant and are therefore highly sensitive to this chemical. It therefore has a particularly selective activity against squamous cell carcinoma and in particular tumours with high cell replication rate. Normal skin cells are much less sensitive than rapidly dividing tumour cells. Where cells are partially affected they appear to become much more 'recognizable' to the immune system. This double effect results in an effective barrage against the tumour but the material does not appear to affect deeper tissues. Therefore depth is limited when topical applications are made.

The main indication for 5-fluorouracil is in the treatment of superficial squamous cell carcinoma as an alternative or adjunct to surgical excision. It can also be used in some occult and verrucose sarcoids.

There are two main forms of 5-fluorouracil that are applied in equine cutaneous oncology with or without surgical debulking. Topical 5% cream and an aqueous (intratumoral) injection are available methods using this material. Debulking is probably a sensible option prior to the use of either topical or intralesional injection, if the tumour is larger than 2–3 cm; in this event the chemotherapy regimen should only be started once bleeding has ceased.

All forms of the material should be treated with considerable respect and suitable handling precautions should be taken for all antimitotic and cytotoxic chemicals.

Topical ointments containing 5–20% 5-fluorouracil (Efudix, Roche, UK) have been found to be effective in the treatment of some very superficial occult and verrucose sarcoids and localized squamous cell carcinoma (Fortier & MacHarg 1994). Topical treatment of squamous cell carcinoma is an effective alternative to surgical excision in some genital cases in particular. Repeated applications of the material to the surface of the mass are required and this may sometimes be resented. A suitable protocol is twice daily applications for 7 days and then once daily application for 7 days. Following a rest period of 7 days during which the acute

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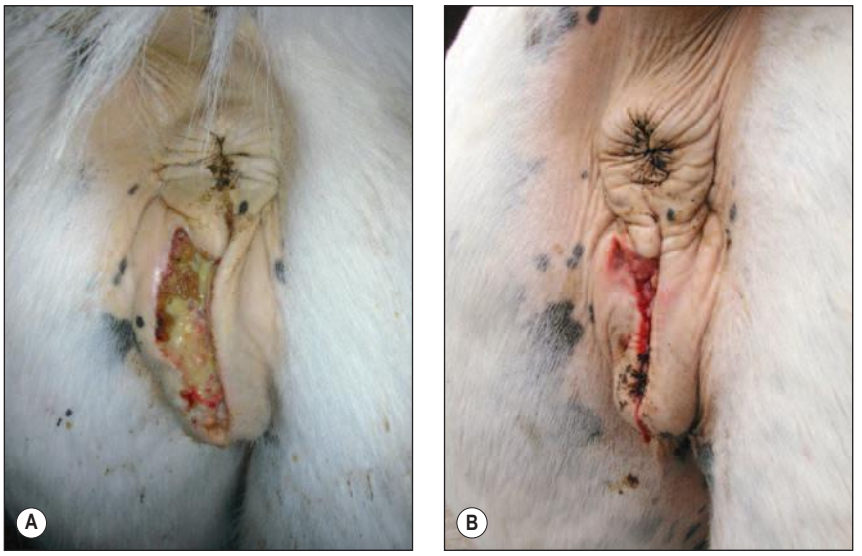


Figure 4.10 This labial vulvar squamous cell carcinoma developed in around 4 months and in that time caused considerable and increasing tissue destruction **(A)**. Topical treatment with 5% 5-fluorouracil was instituted daily for 7 months and then intralesional injections were made every week for 6 weeks. The lesion did not resolve completely but showed considerable improvement **(B)**. Management was relatively straightforward and was deemed to be worthwhile by the owner. Alternative treatments were not possible.

inflammation subsides, the treated area can be clinically assessed. Repeated courses may be required for up 7–12 months (Fig. 4.10) but superficial squamous cell carcinoma and some sarcoids **(Fig. CD4 • 1)** respond more rapidly to this regimen. Longer inter-treatment intervals can be used on penile lesions because the concentration of the drug can be maintained within the prepuce.

The effects of these applications include an acute inflammatory response with exudation (and hair loss) followed by sloughing over some weeks. Application does not appear to cause any discomfort but the treated area can become significantly inflamed.

Solution for injection containing 25 mg/ mL (designed for intravenous or intra-arterial use in human oncology) (Oncotain, DBL, Mayne Pharma plc, UK) (Doyle 1996). There are few reported studies on the use of injectable forms of 5-fluorouracil in tumours of horses. However, the author has had some success both with squamous cell carcinoma and with nodular and fibroblastic sarcoids. Remissions are slow, but persistent treatment can be successful in some cases (Fig. 4.11).

Arsenic paste

Historically a soap-based paste containing up to 25% white arsenic trioxide in a soft soap base has been used particularly for the treatment of equine sarcoids for over 100 years. Neat arsenic trioxide powder has also been dusted onto ulcerated tumours. The corrosive nature of the material makes it extremely dangerous topically with uncontrollable tissue necrosis. It is also extremely toxic systemically and there are serious safety issues with the handling of the chemical (especially in powder form).

AW4-LUDES

This material is an experimental combination of cytotoxic and antimitotic drugs, heavy metals, natural oils and steroids in various concentrations which has been shown to have some promise in the treatment of equine sarcoids (Fig. 4.12 and **Fig. CD4 • 2A–C**). It appears to have little or no value in the treatment of other forms of cutaneous neoplasia. Major complications can arise from extensive local cicatrization. Deep muscle damage can also be caused when treating periocular fibroblastic sarcoids (especially where these have been unsuccessfully treated previously by other means). Up to 70% resolution of individual treated lesions has been obtained in trials involving over 20000 cases at Liverpool University. The best results are obtained when AW4-LUDES is used as an initial treatment method. Repeat treatment and treatment of previously treated and unresolved lesions is much less successful. However, repeated treatments in these refractive cases are still recorded as being more likely to be successful than other compounds and treatments commonly used (Knottenbelt & Walker 1994).

Synthetic retinoids

This includes those synthetically derived compounds with a vitamin A-like activity. The retinoid pharmaceuticals include a very large and complex group of topical medications in several generations. Many medications in this group have been developed for human use in the management of several disorders of keratinocytes.

Retinoids modulate key pathogenic factors in mild–moderate plaque psoriasis in human and down-regulate expression of matrix metalloproteinases (MMPs), cytokines and other genes involved in cutaneous proliferative conditions and some inflammatory skin diseases including basal cell carcinoma (BCC) and (precancerous changes) in squamous cell carcinoma.

Tazarotene is a third generation of receptor selective retinoids (ethyl-6[2-(4,4-dimethylthiochroman-6-yl)-ethynyl]nicotinate) that the author has found to be useful in some superficial occult and verrucose sarcoid cases (Knottenbelt 2005). Its exact mechanism of action is not fully understood but it is known to restore normal cutaneous morphology, reduce inflammatory markers and diminish or down-regulate markers of epidermal hyperplasia and abnormal cell differentiation through actions on keratinocyte transglutaminase, involucrin and keratin. Its antitumour effects result from its ability to restore apoptosis in keratinocytes and to normalize the epidermal keratinocyte proliferation, and limit the lack of differentiation. In some cases the effects may be similar in fibroblasts, although that is not a reported effect. There are direct anti-proliferative effects on basaliomatous cells and arrested production

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Figure 4.11 This horse had received previous radiation brachytherapy for an invasive sarcoid in the upper and (medial) lower eyelid. He developed a large aggressive fibroblastic sarcoid on the cheek **(A)**. This was treated by repeated intralesional infiltrating injections of 10 mL (250 mg) 5-fluorouracil injectable solution daily for 7 days and then weekly for four treatments **(B)**. There was an extensive necrosis and sloughing of the lesion **(C)**, leaving a small fresh lesion **(D)** that was retreated later in the same way. Follow-up after 2 years shows no recurrence at this site.

of keratinocytes so that there is a reduction in abnormal/normal keratin production. The action on keratinocytes is likely to be useful in a number of disorders of keratinization but these are not yet known.

Tazarotene-induced gene-2 (TIG-2) is expressed in high levels in normal skin and in low levels in psoriatic skin. Expression is up-regulated following tazarotene application. The compound is thought to restore normal epidermal differentiation and therefore it could be useful in management of any of the proliferative or dysplastic keratinocyte disorders in horses. The clinical effects are a result of interaction with specific retinoid receptors within cells that result in altered cell behaviour. In this way they broadly induce reduced cell replication, alter the cytokine profiles that cells produce and some have anti-inflammatory effects. This rather broad spectrum of activity means that they have a wide range of clinical effects and so they have found use in many types of disease including some dermatological conditions in humans including Darrier's disease, psoriasis, basal cell carcinoma and discoid lupus, and in some cutaneous conditions in small animals. There are no reports of their specific use in horses.

Given that the effects are most obvious on keratinocytes and the effects on fibroblast are far less obvious, tazarotene 0.1% gel (Zorac 0.1% Gel/Tazorac 0.1% Gel (Allergan, UK)) can be used to reduce the overall area of verrucose or occult sarcoid and so reduce the extent of supportive primary sarcoid treatment (Fig. 4.13 and **Fig. CD4 • 3A–D**). A few cases with limited dermal involvement have resolved completely with this material in the author's case series, but retinoids in general should be viewed as adjunctive therapy.

CD

An incidental use of this material is in the management of some epidermal naevi (see p. 242) and in some cases of coronary band dysplasia (see p. 362).

The compounds are relatively expensive and they do have recognized teratogenic effects and so are precluded in pregnancy and must not be handled at all by pregnant humans either.

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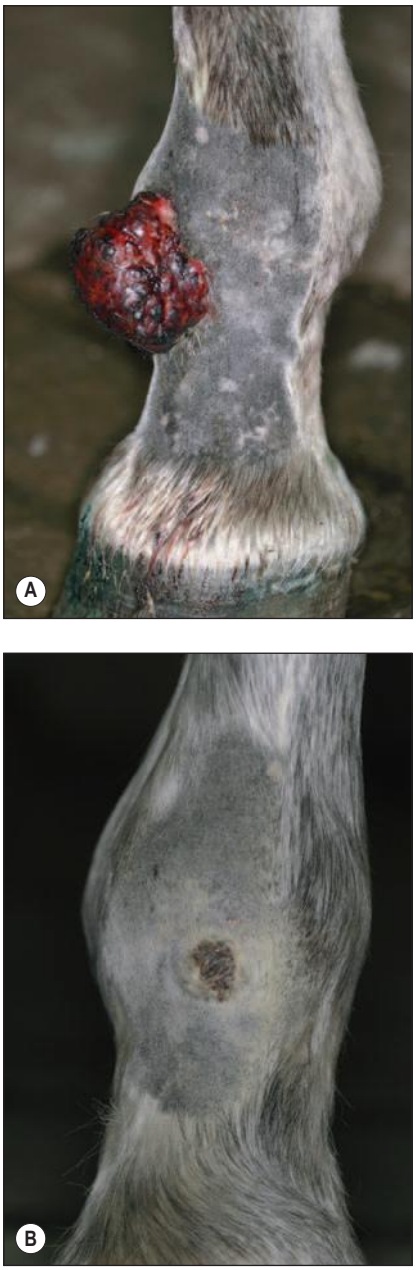


Figure 4.12 This type 1a pedunculated (non-rooted) sarcoid **(A)** was treated topically with AW4-LUDES over a period of 9 days. The second picture **(B)** shows the state after 3 months. A second milder course was applied on suspicion that the sarcoid was still active and after a further 7 weeks the site was a small flexible skin scar. No recurrence occurred for at least 4 years.

Prednisolone/corticosteroids

These compounds have a detectable antimitotic effect and can be used as supplements to other therapy for proliferative dermatological and conjunctival lesions. The usual precautions must be taken before and during use (checking for fungal/viral/bacterial infections and, in the eye region, corneal ulcers). The compounds have a specific inhibitory effect on fibroblasts (Bill 1997). Corticosteroid compounds applied topically to exuberant granulation tissue have a marked inhibitory effect on fibroblast replication but probably have no effect on sarcoid fibroblasts.

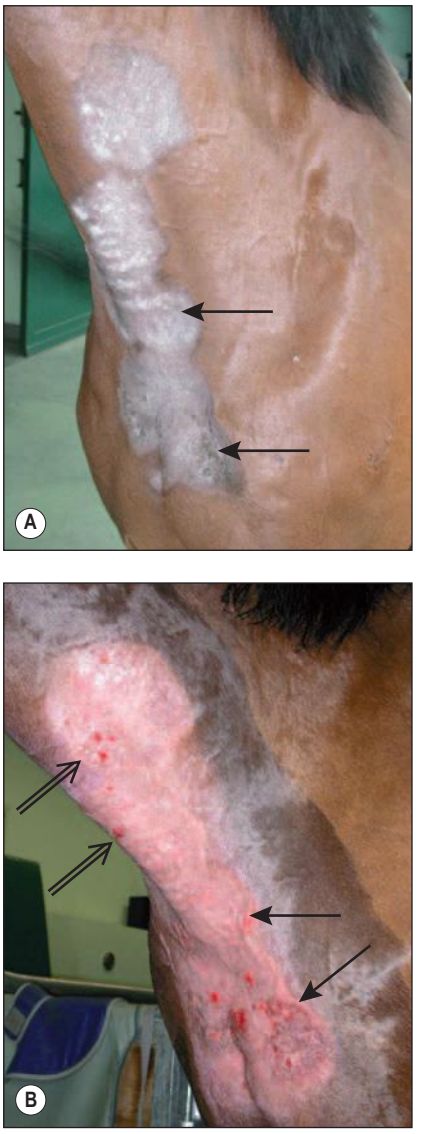


Figure 4.13 This extensive verrucose/occult sarcoid was clearly impossible to treat by any conventional means **(A)**. Topical applications of tazarotene as a 0.1% gel (Zorac, Allergan, UK) resulted in extensive epidermal sloughing, exudation and some discomfort. However, it was then possible to identify 12 localized sites where sarcoid was more obvious. **(B)** Some of these obvious superficial verrucose sarcoids did in fact resolve with this treatment alone. The solid arrows identify the obvious areas of sarcoid that were visible at the outset but the open arrows identify aggregates of sarcoid that were not obvious prior to treatment. These were treated with AW4-LUDES and resolved. (Figures courtesy of Dr Andrea Bertuglia.)

Systemic therapy

The range of systemic antimitotic drugs for use in horses is very limited. The specific antimitotic drugs used for treatment of generalized or extensive neoplasia in small animals and humans, such as 5-fluorouracil, are not feasible in the horse under normal conditions for reasons of cost and toxicity. Only cimetidine treatment of melanoma has been reported in a significant number of cases.

Cimetidine

Cimetidine (an H_2-receptor blocker used in the treatment and control of gastric ulceration syndrome in adult horses

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and foals) has been advocated for the treatment/control of melanomas in horses. The concept related to the benefit reported in human melanocytic disease, and initial reports suggested that it had a considerable benefit in some equine melanoma tumours. A remission rate of around 50–90% was reported (Goetz et al 1990). Subsequent reports have, however, largely refuted these findings and the efficacy is clearly a problem with such variable results in spite of apparently similar disease states and similar regimens being used (Warnick et al 1995). All the case series so far reported are very small and so the individual veterinarian needs to be satisfied with the choice based on one or other of the reports. The results from larger studies need to be reported. The premise that the equine melanoma is similar to the human condition is possibly a reason why there is a wide range of responses. Case selection may be the major limiting factor.

The mode of action is uncertain at present but its antineoplastic properties seem to depend on the anti-serotonin effects. It seems likely that activation of suppressor T cells (which are thought to alter the natural antineoplastic mechanisms in the body) is reduced by the drug. This allows the expression of the natural mechanisms which might be expected to control the development or proliferation of melanoma.

Oral treatment at up to 3.5 mg/kg three times daily (or somewhat less effectively 9 mg/kg once daily) should continue for 6 weeks; if there is no material change there is little value in continuing. However, if a detectable improvement is evident then the drug should be continued for 3–4 weeks after no further improvement is evident. Repeated courses may have a similar or different effect. The dose suggested for melanomas is a fraction of that required for treatment of gastric ulceration syndromes and other related H_2-blocking drugs are reportedly not effective at all.

Dermatological radiotherapy

Ionizing radiation destroys living cells and this property is employed to treat certain types of cutaneous (and other) neoplasia. The application to cutaneous oncology is important in providing a therapeutic option for tumours which might otherwise have no chance of resolution. The techniques have significant advantages in minimizing scarring and distortion due to cicatrization in anatomical regions where normal function is of vital importance. Skin tumours involving the eyelids and periorbital skin are probably the most frequently subjected to radiation therapy but masses at other sites may also be important.

The basic principle of the technique arises because electromagnetic radiation (γ radiation) and high-energy particles (α, β radiation) ionize tissues through which they are directed.

Following a course/dose of radiation the tissues are destroyed (after a variable period) and then a process of repair needs to take place. Normal reparative tissue develops from natural stem cells to replace the destroyed tissue. It then needs to become reorganized into a natural and therefore functional organ system with restoration of blood supply and oxygenation. This process is largely similar to normal wound repair mechanisms but the slower response typical of radiation damage means that there is a greater tendency for natural tissue cells to replace the damaged cells rather than for fibrous tissue scarring. Therefore the extent of scarring is usually much less and the cosmetic results are usually very acceptable. Some cells are, however, permanently altered by the radiation rather than killed by it. This might reflect the relative resistance of the cell type or the reduced radiation dose to which it is subjected. In the skin, melanocytes along the margins of a lesion treated with radiation will often be suppressed or altered so that white skin (leukoderma) and hair (leukotrichia) may replace the normal body colour.



Figure 4.14 Iridium-192 linear radiation sources inserted into periocular sarcoids.

The procedure carries both cost and safety implications (no matter how it is applied) and therefore the technique is usually restricted to small tumours in difficult sites such as the eye and periorbital skin and over joints. There is the added advantage that radiation can be used as 'fail-safe' technique. Tumours previously treated with less expensive techniques which have not succeeded may still be treated by this method with good prospects of success (within the inherent limitations of the method). Accurate dosimetry and careful use results in a better overall prognosis than with most other methods. It can be combined with surgical debulking (as in the treatment of corneal carcinoma in situ) so that less radiation needs to be applied. The full effects of radiation treatment may take several months to become apparent. Tumours treated in this way often show a progressive size reduction for up to 12 months, but in other cases considerable local necrosis occurs and abscess discharge may initially be alarming.

Radiation is delivered by brachytherapy or teletherapy, which reflect the method of generation of the radiation. There are only a few reports of the use of teletherapy for treatment of equine dermatological problems (Coombe 1994).

Brachytherapy Brachytherapy is most often used in the treatment of squamous cell carcinoma, neurofibroma and nodular and fibroblastic sarcoids and other superficial tumours situated in areas which are not amenable to surgery or which have recurred after previous attempts to remove them (by whatever means). The most frequent methods for this include a number of sources of β and γ radiation which are either implanted into the tumour mass (Turrel & Koblik 1983) or applied directly to the surface (Frauenfelder et al 1982). Iridium-192 (Fig. 4.14) and

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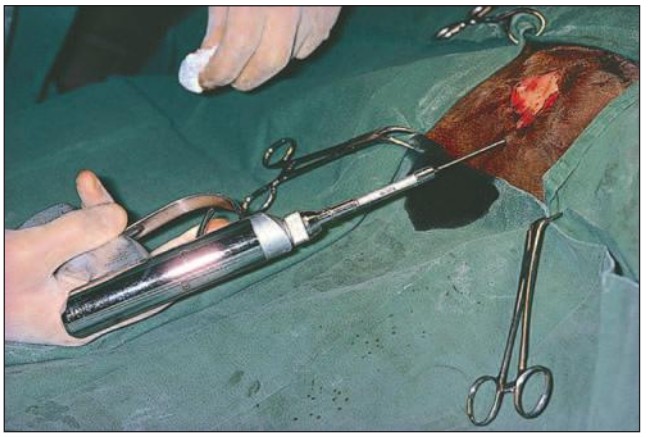


Figure 4.15 A Marston gun being used to insert radiogold (gold-198) pellets into an anaesthetized horse to treat a palpebral squamous cell carcinoma.



Figure 4.16 A strontium-90 plaque being applied directly to the surface of the cornea as adjunctive treatment to surgical keratectomy for a carcinoma in situ.

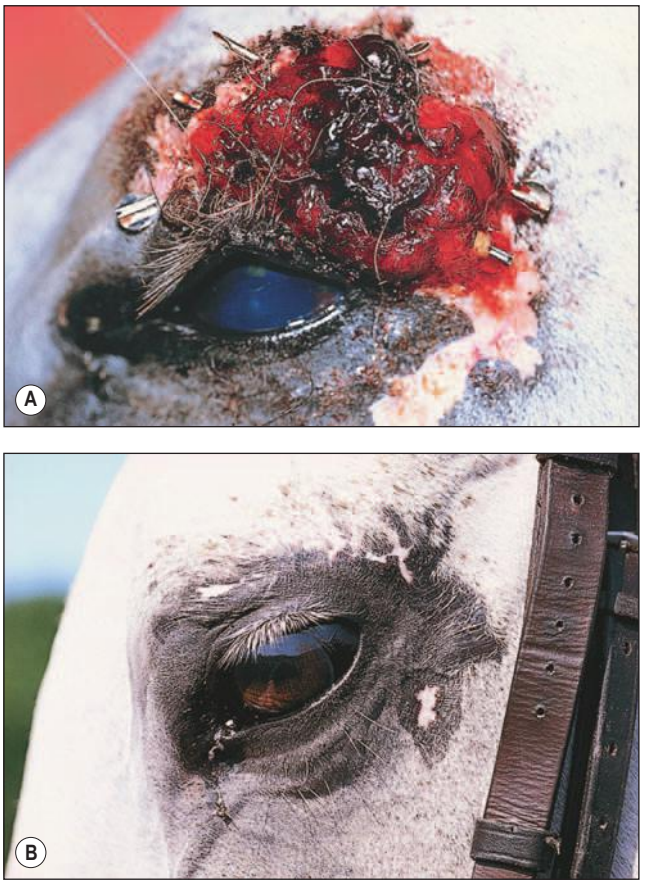


Figure 4.17 An aggressive fibroblastic sarcoid in the periorbital skin had been treated several times with a combination of surgery, BCG and cryosurgery without resolution. The introducers used to guide the linear iridium-192 sources are shown in **(A)** while the final appearance of the lesion some 12 months later is shown in **(B)**. Note the loss of hair and the limited white-skinned area above the upper eyelid which are typical of radiation therapy. In spite of the severity of the lesion, the functional and cosmetic effects are excellent.

gold-198 (Fig. 4.15) are γ emitters with radiation continuously emitted from the source through the target tissue. The dose of radiation can be adjusted by altering the strength of the source, the time of contact or its physical extent. For surface applications, β radiation is delivered from a strontium-90 (or similar isotope) plaque (Fig. 4.16). Radiation is applied in fractionated doses calculated to deliver the required 'lethal' overall dose over several days. The source is usually applied for a pre-calculated time once or twice daily so that the required dose is administered over a considerable period. Fractionation is more important for β radiation than for γ radiation.

The beneficial effects of radiation can be startling, with complete resolution, and although its use has severe restrictions it is a useful fall-back where other methods fail (Fig. 4.17).

Teletherapy A linear accelerator is used to generate radiation of specific types and power which is then beamed into the treatment area. The animal needs to be completely still and so cases subjected to this technique require general anaesthesia. Fractionation and multi-beam technology means that deep tumours can be treated. However, it is such a specialized technique that it is very rarely available, practical or applicable.

Advantages

- High primary success rate.
- Little disfigurement and scarring.
- Effective back-up option for other methods.
- Placement of iridium-192 wire brachytherapy can be easily achieved under sedation alone especially using after-loading techniques (this limits personal exposure).
- Strontium-90 is very safe when properly used and fractionated doses can be administered under sedation alone.
- Debulking leads to residual activity of dividing tumour cells, which are most susceptible to radiation, and to smaller treatment areas.

Disadvantages

- There is potentially serious personal danger to operator and staff.
- Specialist radiologist or radiophysicist is required.
- Accurate dosimetry requires detailed calculations.
- Special secure area for hospitalized horses is required.
- Cost of isotopes is very high.

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- Surgical debulking may be needed to limit the radiation dose.
- Limited size/volume of tissue can be treated at a time.
- General anaesthesia is often required for placement (particularly for gold-198 seeds).
- Some extensive reactions possible with tissue necrosis – may take a long time to resolve.
- Resultant leukoderma and leukotrichia (white scars).

Hazards and effects of radiation therapy

The toxicity and hazards of radiation therapy can be divided into the acute or immediate effects and the chronic (long-standing) or permanent effects which are usually (hopefully) restricted to the treated case (rather than the operator). In the former, the skin surrounding the radiated tissue becomes inflamed (swelling, heat, pain and redness). The fastest-dividing cells in the area show the earliest effects with superficial sloughing. In the case of the eye there may be a degree of transient corneal oedema over the treated area of cornea. In the treatment of dermatological tumours the acute reactions are of little consequence but may alarm a poorly counselled owner.

Chronic effects of radiation are more obvious and remain in the long term. They may have significant implications for operators who are repeatedly exposed to radiation of any description. These changes affect slowly dividing cells, e.g. bone, cartilage and the lens. Thus damage to the lens culminating in the development of cataract can be a secondary consequence of γ radiation in and around the eye. Radiation has significant effects on melanocytes and hair follicle germ cells, leaving white skin and hair to replace the body colour (Fig. 4.17B). Only in very rare cases is there any apparent long-term effect on local cell repair, and restoration of function is to be expected in almost all cases (unless the radiation dose is excessive) (see Fig. 4.17).

Systemic effects of a single course of radiation brachytherapy applied in the treatment of a skin neoplasm in the horse are seldom noticeable. There are no reports of any serious secondary effects even when repeated courses are applied, although this is a particularly unusual circumstance.

- Repeated exposure to ionizing radiation has potentially disastrous consequences for operators including the development of internal malignant neoplasia. *Any* cell can be transformed into a cancer by sufficient or inappropriate exposure to radiation.
- Unless specific reasons exist and suitable precautions can be taken, other means of treatment should be explored.
- Radiation is a dangerous 'last-ditch' resort even though it has very satisfying results for the most part! Its use *must* be very carefully considered in every case.

Principles of surgical treatment of skin disease

Surgical therapy most commonly applies to proliferative or neoplastic skin diseases. Before embarking on a surgical approach, careful consideration must be given to the prognosis. The type and character of a tumour has a profound bearing on the feasibility of surgical options. The character of the problem may render surgical options fraught with difficulty. Surgical excision may appear to be an attractive and simple option, but some conditions are probably only amenable to surgical excision with strict limitations. For example, nodular and small occult or verrucose sarcoids can be effectively treated by surgical excision (or cryosurgery), but there remains a high risk of recurrence. The secondary effects of surgery may be more serious even than the original disorder. While squamous cell carcinoma of the third eyelid or the cornea can often be treated by surgical excision, palpebral forms of the condition are not usually amenable to surgical removal because the defect and/or subsequent scarring may lead to clinically significant limitations of function.

In general, surgical excision is less feasible in areas where there is little 'spare skin' to allow primary closure of the defect while at the same time ensuring complete removal. The perceived limitations in such areas tend to encourage a conservative approach by the surgeon. Marginal excisions may result in a higher risk of tumour recurrence, particularly in ill-defined (and potentially malignant) tumour masses. In any case it may be very difficult to define the lesions sufficiently to be sure of complete removal.

Some classes of tumours are amenable to surgical removal with good recovery and low recurrence rates. For example:

- Lipomas and *subcutaneous* nodular sarcoids are easily removed and seldom create any untoward problem.
- Mast cell tumours can respond well to surgery even if surgical ablation is not complete.
- Melanomas have an unpredictable outcome: while many respond well to surgical ablation, some are in difficult or impossible sites and a few are highly malignant.
- Squamous cell carcinoma of the membrana nictitans (third eyelid) is frequently amenable to surgery. The availability of 'extra' mucosa permits primary closure after wide excision: it is usually possible to perform wide excision without risk of failure of closure or future cicatrization. With the possible exception of nictitans and some penile and vulvar forms, squamous cell carcinomas have a high recurrence rate following surgical ablation on its own and better results are obtained when combined with cryosurgery (see below), radiation or cytotoxic medication.

The surgeon should therefore take every possible step to ensure that an appropriate choice of therapy is made and should certainly consider every possible consequence before embarking on surgical excision of tumours in particular.

There are several surgical options available to the surgeon. These include ligation by strangulating ligature, electrocautery, surgical excision, cryosurgery, hyperthermia, and laser surgery. The relative value of these options for each specific disease for which it is a viable treatment option is given under the disease profiles in Part II.

Ligation by strangulating ligature

Indications

This is restricted to pedunculated, benign masses in sites where there is loose skin which allows the incorporation of

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Figure 4.18 Two elastic (lamb-castration) bands applied below a skin nodule (sarcoid) on the medial thigh. Note the 'safe' skin margin that maximizes the chance of success and the absence of significant tension on the skin beneath the bands. This is not applicable to all cutaneous tumours but is a widely performed practice for certain types of sarcoid in particular.

a significant margin away from the mass itself that can be included in the ligature. Suitable masses include pedunculated and isolated melanomas and certain pedunculated fibroblastic or nodular sarcoids (where it can be used in conjunction with other methods including topical cytotoxic creams).

Methods

Ligation can best be achieved by the use of Lycra in larger lesions and elastrator (castration) bands for smaller nodules (Fig. 4.18). Alternatives are heavy gauge elastic bands applied around the base of the lesion or the tight application of heavy suture material or string (Fig. 4.19). In some cases even a hair from the tail of the horse has been used.

Advantages

- Easy
- Cheap
- Convenient (requires little surgical skill)
- Small scar.

Disadvantages

- Only applicable to certain tumour types under controlled conditions in defined locations, i.e. there is 'spare' skin without tension, a safe margin and tolerance of skin removal.
- Imprecise.
- May need concurrent cytotoxic applications in addition for maximal benefit if there is some doubt about the extent of the base of the tumour.
- Risk of infection (tetanus in particular).
- Can be painful both at the time of application and later during sloughing.

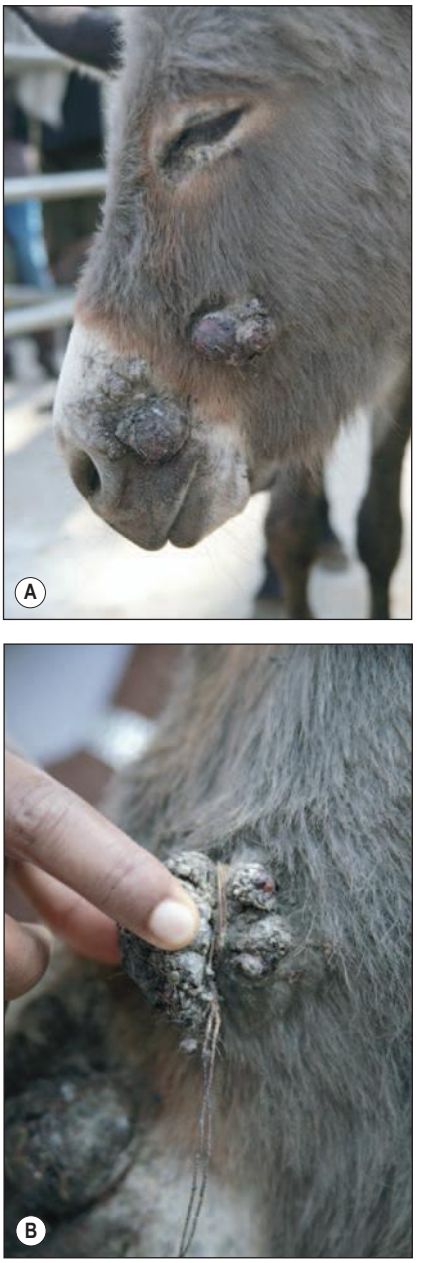


Figure 4.19 This donkey had two significant fibroblastic sarcoids on its face **(A)**. An attempt was made to ligate one of them with nylon twine **(B)**. Not only was the ligature not tight enough to cause anything other than an irritation to the tumour, it was not placed beneath the base of the tumour. In this circumstance the method cannot be expected to be successful.

- When sloughing finally occurs it is quite common for the skin to 'spring back' and leave a larger deficit than was expected. This occurs particularly when there is tension skin beneath the ligature. Provided that all the tumour tissue is incorporated into the ligature a good result can be expected.

Electrocautery/diathermy

Indications

Where single small masses are involved, ordinary electrocautery is satisfactory but recurrence can be expected

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because it is again impossible to define the margins of many tumours. Electric heat is produced in a focused probe that cuts tissue by burning. Newer high-frequency electrosurgical units are now available that can simultaneously cut and coagulate on their own or together. Another method using electric current greater than 10 000 Hz across two electrodes to destroy tissue between the two points is described. Supposedly at this level it is pain free and causes no muscle contractions or other electrical problems. Current around 3000 Hz causes heat and pain.

Method

With large electrocautery units, cutting, coagulation and fulguration are available and should be used; the tumour should be removed with cutting current followed by treatment of the area by fulguration as far as possible, particularly the skin edges.

Advantages

- Control of haemorrhage during surgery allows precise excision.
- Electrocoagulation reduces the chance of sideways migration of tumour cells or infectious agents and seeding of the wound bed.

Disadvantages

- Equipment can be expensive and cumbersome.
- Treatments may require general anaesthesia.
- Large vessels may require ligation.
- Cicatrization can be problematical if large areas of skin/tumour are removed.

Thermocautery

Indications

Use is limited to small tumours or where electrocautery is not available.

Method

The technique requires a source of heat such as a gas burner to heat metal probes. Electrically heated wires can be used (tendon 'firing irons' of linear or point configurations are the main example of this and are, regrettably, sometimes used to surgically excise tumours).

Advantages

- Removal of the entire area does remove the cause.
- Haemostasis is effective.
- Necrosis of the margins limits spread of tumour cells.
- Equipment is generally available.

Disadvantages

- Lack of precision: it is difficult to control.
- Cicatrization with contraction is significant especially around eyelids or mouth.

Radiofrequency hyperthermia

Malignant tumour cells are more sensitive to the effects of heat between 41 and 45°C because neoplastic cells tend to be hypoxic and acidotic and because tumour microvasculature is preferentially destroyed. This means that adjacent normal tissue is largely unaffected and so the method is applicable to small tumours on or around the eye. Although by no means all tumour cells are sensitive to this effect, the method has been used in treatment of squamous cell carcinoma (Grier et al 1980, Schwink 1987). Some other small tumours such as sarcoids have also been treated successfully (Grier et al 1980, Hoffman et al 1983, Latimer 1987). Larger tumours and aggressive invasive tumours of any sort are unlikely to be treated effectively with this method.

Tissue heating (to 50°C) over at least a 30-second period is achieved using a 2 MHz hand-held thermoprobe source of the radiofrequency energy. The tissue between the two electrodes is heated to the controlled temperature. The extent of tissue damage is limited to 2–4 mm depth over an area of 1 cm^2. The size of the probes means that invariably overlapping and repeated applications are needed. This is a very time-consuming process for larger tumour areas and volumes but is more appropriate for superficial ocular/orbital masses (e.g. surface neoplasia, squamous cell carcinoma, verrucose sarcoid and limited fibroblastic sarcoids; the latter may require a more potent energy source). Treatments usually require repeated courses over 3 weeks or more. Following the procedure, the treated tissue undergoes necrosis and sloughing over some weeks, leaving a healthy granulation bed. Recurrence will occur, however, if any tumour cells remain viable.

There is currently an increasing interest in the combination of hyperthermia and the concurrent use of various chemotherapeutic drugs. The effects of radiation are also said to be potentiated by hyperthermia. These potentiating effects may enable the method to be used in a wider variety of tumour cells, and sarcoid tumours are the obvious target for this approach being both superficial and easily recognized.

Success rates in some studies have suggested that the method has no material advantage over surgical excision alone in equine ocular squamous cell carcinoma and is no better than most other modalities for both squamous cell carcinoma and sarcoids; refractory cases require further treatment by other means. The method is most successful for shallow early tumours not previously treated. Better results are reported if surgical debulking is performed first.

Laser surgery

Indications

The laser is a concentrated monochromatic beam of infrared energy (10.6 μm), which is maximally absorbed by tissues with high water content and/or melanocytes. The rapid rise in intracellular temperature to over 100°C vaporizes the cells (photothermolysis). Historically CO_2 YAG lasers have been cumbersome and very expensive both to purchase and to maintain. Laser surgery is becoming more accessible since the advent of safer and cheaper diode machines (Fig. 4.20). In all cases there are significant health and safety regulations that must be employed to prevent any human eye or other damage. In the UK, qualifying premises with suitable controls are given a licence to use a surgical (high energy) laser. They cannot be used outside these particular circumstances.

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Method

The premises should be suitably prepared to prevent any possibility of laser reflections from shiny metallic surfaces. Most laser instruments have a red indicator target beam that is switched off during laser firing – when the red beam is visible there is no laser firing. The machine power, pulse width and fluence should be adjusted to suit the tissues being excised. Most machines are relatively simple to set up but experience is required to adjust the major functional parameters to minimize the damage to adjacent tissues such as bone, tendon, nerves and vessels.

After suitable surgical preparation, which usually includes general anaesthesia, the surgical team and anaesthetists/nurses and technicians are suitably protected with laser goggles. Careful strategic laser cutting allows meticulous, almost bloodless surgical excision (Fig. 4.21).

Advantages

- Precise surgical excision is possible.
- Less pain (nerve endings are obliterated and sealed during the procedure).
- Control of haemorrhage during surgery allows improved surgical technique (note that higher energy laser does not coagulate the vessels and if this is required a lower setting or ligation of large vessels is required).
- Laser vaporization reduces the chance of deposition of tumour cells or infectious agents such as fungi and bacteria in the wound bed during surgery.

Disadvantages

- Equipment can be expensive.
- Protective measures have to be taken (protective eye goggles/glasses and non-reflective room/equipment).
- General anaesthesia may involve oxygen and there is a risk of explosion in high oxygen conditions.
- Treatments may require general anaesthesia but under many circumstances procedures on the skin can be performed with suitable sedation, restraint and local anaesthesia.
- Bleeding may only start after some hours following surgery; the surgeon should be aware that larger vessels may require ligation.
- Cicatrization can be problematic if large areas of skin/tumour are removed.
- Wound healing is delayed due to 'skin margin die back'.

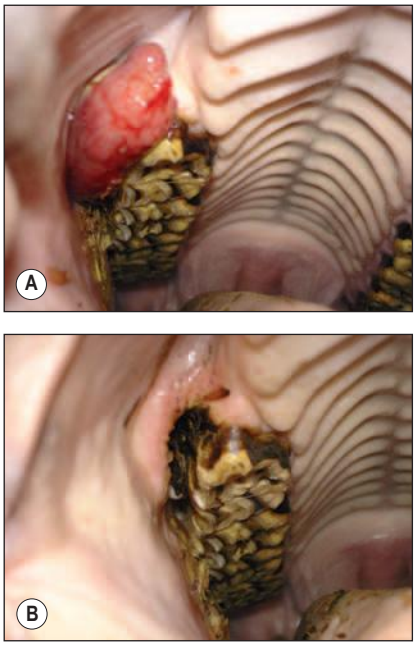


Figure 4.20 **(A)** This oral fibrosarcoma was removed from the margin of the upper gingival margin using a diode laser under standing sedation and a (posterior) maxillary root nerve block. **(B)** No recurrence was reported after 4 years.

Surgical/sharp excision

Indications

This is effectively restricted to small tumours and other lesions with a well-defined margin or with sufficient skin around them to permit effective excision and primary closure after surgery. It can effectively be applied to benign or inflammatory lesions at any suitable site including dermoid cysts, inclusion cysts, eosinophilic collagen necrosis lesions and some neoplastic lesions where the margins can be defined and a suitable closure/healing method is available. In some sites such as the upper eyelid, very particular care has be taken to avoid excessive removal of vital skin and musculature and avoid the formation of functionally limiting scarring. The latter include non-invasive squamous cell carcinoma (of the third eyelid, limbus or cornea, the penis and the vulva).

Some melanomas, lipomas and mast cell tumours are effectively removed by sharp surgery, but for the most part



Figure 4.21 **(A)** Laser surgical excision of a preputial fibroblastic sarcoid in a 9-year-old stallion. Note the bloodless field and the precise nature of the surgery **(B, C)**. Histology confirmed a safe margin around each lesion and in spite of the difficulty of the site, healing was uneventful.

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the equine sarcoid is not amenable to this treatment as there is a high rate of recurrence. A possible exception to this is the type A_1 nodular sarcoid. These nodules, which are entirely subcutaneous and surrounded by a loose fibrocellular capsule (see p. 392), can be effectively removed by carefully incising the overlying skin without penetrating the tumour at all. The loose capsule allows the mass to be gently squeezed from the surrounding tissues. During all surgical approaches to the equine sarcoid in particular the use of the clean surgical approach is very helpful in minimizing tumour seeding into the wound site. The one-cut, one-blade principle is used; no cutting or closure instrument is reapplied to the skin for a second time and provided that the margins of the sarcoid have not been damaged during the procedure the results are usually excellent. In some circumstances there is little or no option either because of the nature and location of the tumour or the lack of any feasible alternative approach (Fig. 4.22).

Where the excision is likely to leave a skin deficit which is impossible to close, alternative methods have to be employed (these may include skin grafting after surgical excision, and the use of tissue substitutes) or the reliance on second intention healing. Surgical excision is accordingly not generally applicable to ill-defined fibroblastic or occult/verrucose sarcoids or to palpebral squamous cell carcinoma; in some cases there are no viable alternatives, however **(Fig. CD4 • 4A–E)**.

Methods

Various surgical methods are applied in normal practice and are beyond the scope of this book.

Surgical debulking of large tumours prior to application of cryosurgery or the injection of cytotoxic or immunomodulating compounds or prior to radiation treatment is widely practised with variable results.

Advantages

- Easy
- Cheap
- Relatively quick
- Sometimes does not require general anaesthesia
- Convenient (requires little surgical skill)
- Small scar if primary closure can be achieved.

Disadvantages

- Imprecise for ill-defined masses
- Risk of dissemination of malignant cells
- Precise surgical techniques may require general anaesthesia
- Need for 'spare' or loose skin (anatomical limitations)
- Scarring may be significant to function, e.g. eyelids.

Cryosurgery/cryonecrosis therapy (cryotherapy)

The principles of cryotherapy are now widely appreciated. It is one of the commonest methods of management of skin masses in horses. However, it is also widely misused through failure to appreciate the actual demands that are required to obtain a good result. Cryotherapy involves the destruction of tissue by a rapid freeze and slow unassisted thaw process. In most cases the procedure is performed over three or more cycles. This process results in the formation of intracellular and extracellular ice crystals, intracellular dehydration and massive elevation in intracellular osmotic pressure, loss of cell membrane integrity and leakage of intracellular material and consequent tissue necrosis. The greatest effects are produced in microvasculature; thrombosis results in localized ischaemia and so any mass that is treated in this way is deprived of its blood supply.

CD

The key to successful cryosurgery is the achievement of a precise, controlled temperature throughout the tumour tissue. Because cryostat histology is not commonly available, it is wise to perform biopsies of some of the most difficult tumours to establish the likely margins. Having constructed a suitable topographical plan of the tumour, thermocouple probes should be placed at the margins and deep to the mass (Munroe 1988). The lesion should be frozen to between − 20 and − 30°C for more than 1–2 minutes

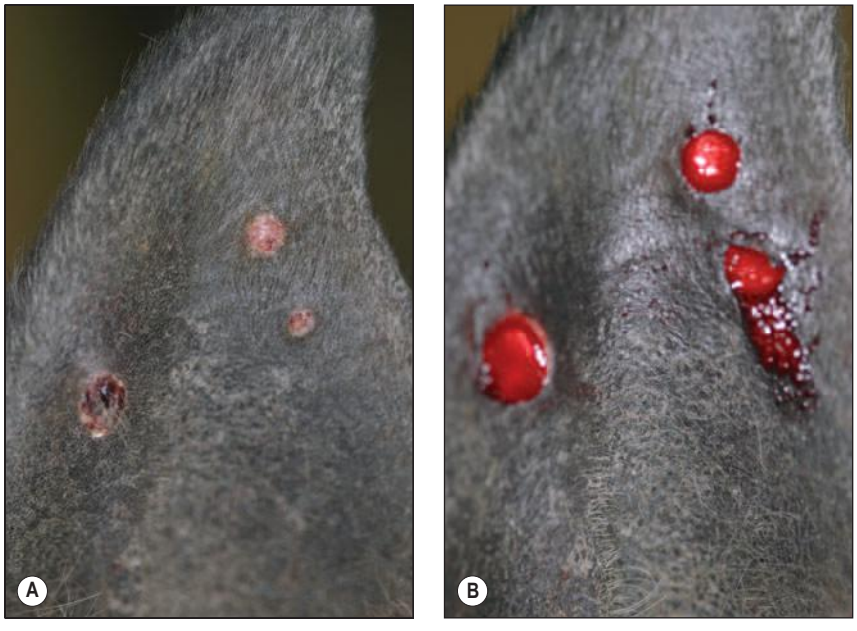


Figure 4.22 These fungal granulomas on the ear of a 5-year-old Andalusian grey mare were removed easily with punch biopsy instruments. **(A)** Before. **(B)** After.

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to achieve the maximal effect. The frozen tissue is left to thaw slowly (unassisted).

In many circumstances for tumours and other applications for cryosurgical treatment, the mass of the tissue can be removed surgically (debulking). This will mean that the extent of freeze is much reduced. However, severe bleeding will have a profound effect on the efficiency of the procedure and therefore the outcome. When attempts are made to freeze heavily bleeding sites, the ice ball usually involves only the free blood and limited tissue. Debulking a day or two before the cryosurgery is performed is a wise precaution but of course means that a double procedure is required. For suitable sites, application of a tourniquet or Esmarch bandage prior to the procedure (whether debulking is used or not) will dramatically improve the effects by reducing the 'central heating system' of the bloodstream and reducing the amount of bleeding.

In most circumstances it is possible to perform cryosurgery on a standing sedated horse but anatomical considerations and duration may dictate the type of restraint used. A single freeze–thaw cycle is seldom adequate and triple (or even quadruple) freeze–thaw cycles are recommended. If extensive tumours or large numbers of masses are to be treated the procedure is very tedious. It also becomes expensive, error ridden and less successful than when a single or few localized areas are treated.

Suitable equipment for cryosurgery in horses varies in sophistication from simple copper heat-sink systems immersed in liquid nitrogen, to sophisticated electronically regulated machines. Common veterinary equipment uses liquid nitrogen (−196°C) delivered either by a spray system or via a thermoprobe (Winthrow 1980). Systems that employ nitrous oxide are largely useless in horses because the tissues do not achieve the required temperature and tumours treated with these systems have a high recurrence rate.

Within 12–24 hours following the procedure the treated sites swell, and gradually over the ensuing 10–21 days become necrotic. A margin of demarcation with inflammation and oedema is usually obvious from around 5 days. The necrotic eschar separates progressively and sloughs (hopefully) leaving a healthy wound bed. Healing takes place by slow (retarded) second intention repair. The site will invariably be hairless, pink and scarred. Even the hairs along the margin of the treated site may be white.

Complications arise from under- or over-treatment and injury to blood vessels, nerves, bone and tendon. Improper technique leads to loss of too much tissue or lack of kill (Fretz & Barber 1987).

Indications

This technique is useful for some types of ocular and skin squamous cell carcinoma (except most palpebral and some vulvar and penile forms), melanoma and sarcoids (Joyce 1975). Localized granuloma, mast cell tumour and other masses can be treated also. The method is non-discriminatory and there are few cell types that can withstand the full three-cycle process.

Cryosurgical cautery of exuberant granulation tissue has also been reported.

Method

CD

The best results are obtained by use of liquid nitrogen at −196°C applied by spray (Fig. 4.23) or by use of a heat-sink probe **(Fig. CD4 • 5A and B)**.

The objective is to freeze the tissue rapidly and thaw it slowly to create the maximum possible cell-destructive effect. Modern pressurized, electrically controlled cryosurgery units have the ability to control the temperature of the probe and so provide a better means of limiting unwanted effects.

Thermocouples, placed 0.5–2 cm from the margin of the lesion (depending on the type of lesion being treated and the anatomic limitations), should always be used to limit the damage and maximize the effects. At least two or three freeze–thaw cycles with tissue temperatures of − 25°C to − 30°C should be applied to all tumours. Lower tissue temperatures will usually bring better results but will of course cause wider damage.

Better results can sometimes be achieved by surgically debulking large tumours to allow better depth of freeze. However, excessive bleeding may create problems and significantly limit the effect. Uncontrolled freezing should be limited by surrounding protective masks created by petroleum jelly (Vaseline) and/or a polystyrene cup or cotton swabs. Extensive, and often uncontrollable, sloughing and subsequent cicatrization limit its application to areas where these are unlikely to have significant effects.

1. Select an adequate 'heat' sink with an appropriate surface area (a copper rod or soldering iron enclosed in a polystyrene 'case' can provide an adequate sink).
   - Liquid nitrogen at − 196°C provides the best coolant.
   - Liquid nitrogen spray apparatus and electrically controlled, more sophisticated machines are commercially available.
2. Insert thermocouples in adjacent tissue (unfortunately these are not commonly used).
3. It is helpful to restrict the local circulation to the area using tourniquet if appropriate and possible.
4. Eliminate bulk by surgical trimming before freezing if indicated; this may enhance the effects of freezing but may cause extensive bleeding if a tourniquet or Esmarch bandage is not used to limit the blood supply.
5. Blood vessels (>1 mm) cut during debulking should be ligated prior to freezing.
6. Protect surrounding tissue with petroleum jelly (particularly if using spray methods).
7. Apply suitable cryogel to surface of mass.
8. Freeze tissues rapidly to below −25°C by applying the probe to the site.



Figure 4.23 A liquid nitrogen spray being used to freeze a fibroblastic sarcoid on the neck of a horse.

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9 Once the local temperature has fallen to below −25°C (as reflected by the thermocouples), the probe can be removed.
10 Allow to thaw slowly to body temperature (*do not* attempt to speed up the thaw by use of warm water, etc.).
11 Repeat the cycle 2–3 times.

Comments

- Under no circumstances should liquid nitrogen be poured onto the skin – this leads to totally uncontrolled 'freeze burning' of tissues to arbitrary and indiscriminate depths and to 'flow' damage in adjacent normal tissue (Fig. 4.24).
- The procedure can be very time-consuming – each lesion may take up to 15 minutes to complete the cycle, so limited numbers can be dealt with at a time (whether in the standing or anaesthetized horse).
- It is vital to take account of the anatomic location and the possible secondary/unwanted effects on nerves, blood vessels, periosteum, bone, muscles, etc.
- The full effects of the procedure will take some weeks to develop. Demarcation of the necrotic tissue is accompanied by some pain, oedema, haemorrhage and swelling.
- Treated areas will usually slough by 2–4 weeks and the resulting ulcer heals by second intention over the following 2–4 months. Failure or delay in the healing process may indicate that tumour cells may still be present.
- Hair follicles are destroyed by double cycle.
- Vitiligo and/or leukotrichia occur even with a single freeze–thaw cycle; scars accompanied by leukoderma occur with double or triple cycles. Owners must be advised of the likely cosmetic outcomes. The owner should be informed of the likely formation of white hair and/or scarring at the site (this is the same process used to freeze-mark the skin for identification).
- Bone can become devitalized if included in freeze–thaw cycles; regeneration may take 2 years or more and sequestration can prolong healing time still further.
- Deep infiltrative tumours are seldom satisfactorily treated by cryosurgery and multiple lesions make the process very time-consuming.
- The patient's temperament is important when considering standing sedation; the correct restraint will allow the full process to be performed efficiently; many cases will require general anaesthesia, particularly if the lesion(s) are located in difficult sites or when they are numerous.
- Tetanus antitoxin or toxoid booster may be required.
- The owner should be apprised of the process, the likely effects (swelling, sloughing and white skin/hair changes) and the required aftercare.



Figure 4.24 This case was subjected to direct application of liquid nitrogen and this resulted in extensive uncontrolled flow and tissue freeze. The procedure was only partially successful; there was widespread recurrence within weeks (arrows). (Figure courtesy of Bob Ordidge FRCVS.)

Advantages

- Convenient for small localized masses
- Cheap (limited facilities needed)
- Easily repeated if needed
- Predictable consequences (immediate and long-term)
- Can sometimes be performed under standing sedation and local anaesthesia
- Cryoantigen release into bloodstream has been reported with benefits at remote sites for treatment of the equine sarcoid (Lane 1977).

Disadvantages

- Tedious if multiple sites; three cycles required for each mass
- Limited applications
- Uncontrolled freezing can be very dangerous
- Need for thermocouples
- General anaesthesia if site/numbers inconvenient
- Swelling and slough of tissue
- White discoloration of hair/skin
- Scarring/cicatrization.

Electrochemotherapy (ECT)

Electrochemotherapy is an adjunctive mechanism for anti-cancer treatment to enhance the effect of chemotherapeutic agents such as cisplatin. The technique involves the delivery of pulsed electric fields to increase the permeability of the tumour cells to the drugs that are injected into the tumour itself. Currently this involves the use of cisplatin (see p. 111). There are reports of improved results when this method is applied to sarcoid tumour in particular (Tamzali et al 2001, Tamzali 2006).

Following induction of general anaesthesia the chosen antimitotic drug (usually cisplatin) is injected intratumorally and up to 1 cm from the tumour margin. Five minutes later the electric current is passed across the site by bringing the electrodes connected to a suitable delivery apparatus (PS15 Jouan Electropulsator, France) into contact with the skin. Eight pulses at 1 Hz frequency with a voltage

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of 1.3 kV are suggested. Multiple procedures are used to treat the entire tumour area.

The procedure appears to carry an improved prognosis when using either cisplatin or bleomycin. Impressive success rates of 100% are reported with no recurrences (Tamzali et al 2003).

The absolute requirement for repeated general anaesthesia (up to four or more times) at 2–3-week intervals is a major factor. Considering the technique is applicable to tumours in difficult locations and to difficult tumour types, this consideration is relatively minor. It is not currently widely practised but is a technique that may develop and become more feasible in hospital situations.

Photodynamic therapy (PDT)

Photodynamic therapy uses specific wavelengths of light to 'activate' photosensitizing drugs and thereby causes an acute inflammatory response and destruction of unwanted (tumour) tissue whilst still preserving the normal tissue.

A drug that is inactive (harmless) until it comes into contact with light of a specific wavelength is either applied locally or given parenterally. If the drug can be concentrated into the target cells (usually neoplastic/cancerous cells) and then the appropriate wavelength of light is applied, the drug becomes active and the resulting destruction of the cells kills the tumour.

The effect is selective according to the light and the compound used and the placement of the chemical in the tissues concerned. Although the method is used quite widely for management of skin (and some internal tumours) in other species including man it is a relatively unexplored therapy in horses. There are several ways in which the chemical can be delivered to the skin.

1. Systemically administered agents with a general tissue distribution.
2. Systemically administered photoactive substances bound to antibodies that target specific cells, i.e. the agent is bound only to target cells, or use of compounds such as haematoporphyrin that deposit more intensively in tumour cells than normal tissues.
3. Topically applied photodynamic agents such as hypericin (plant toxin derived from *Hypericum perforatum*) and d-aminolaevulinic acid-induced porphyrins (endogenously synthesized porphyrins).

Only the latter option has been used to any extent in horses.

The main constraint to be considered in this method is the extent to which light will penetrate tissues. Most light will only penetrate 1 – 3 mm in skin and so its use is usually limited to skin tumours and more particularly small superficial tumours and precancerous/dysplastic changes. There are reports of its use in early superficial sarcoid.

Gene therapy

Gene therapy is a technique that either corrects defective genes that are directly responsible for disease or using genes to overcome specific diseases by inserting them into the genetic structure of an animal so that a benefit can be derived from mediators expressed by the gene. There is a theoretical prospect of simple and effective treatments that avoid the use of potentially dangerous or at least harmful drugs.

Currently the main mechanisms for gene therapy are focused on vector delivery using viruses, such as retroviruses, adenoviruses and some herpesviruses that will incorporate without harm into the host cells. There are also some non-viral delivery approaches using either liposomes, which carry the therapeutic DNA directly into a target cell, or chemical linking of the gene (DNA) to a molecule that will bind specifically to targeted cell receptors. There are major ethical, cost and practical problems that will need to be overcome before this approach becomes clinically useful. Probably the earliest approach will be made to those disorders for which there is a known genetic basis either in terms of genetic susceptibility or where the genes themselves are altered by disease (e.g. neoplastic disorders). The equine melanoma appears to have the strongest genetic predisposition – over 90% of grey horses will eventually (at some stage) have at least one melanoma! Sarcoid also carries the prospect given that the genetic factors involved in the predisposition are becoming clear. In both these cases the prospect of a genetic therapy is highly attractive.

Although currently there is no published clinically useful gene therapy for horses for skin (or other) disease, it is likely that ultimately it will become more feasible as the equine genome is mapped and the genetic implications of disease are known.

References

Barth D, Sutherland I H 1983 Proceedings of the 12th World Veterinary Congress, Perth, Australia, p 14

Bill R 1997 Anti-inflammatories. In: Pharmacology for veterinary technicians. Mosby, London, p 277

Coombe K A 1994 Primary parotid lymphoma in a 10-year-old Hanovarian gelding. Equine Veterinary Education 6:91–94

Cornelisse C J, Robinson N E 2004 Glucocorticoid therapy and equine laminitis: fact or fiction. Equine Veterinary Education 16:90–93

Couto C G 1994 Lymphoma in the horse. Proceedings of the 12th Annual Forum of the American College of Veterinary Internal Medicine, San Francisco, pp 865–867

Doyle P N 1996 Chemotherapeutic modulation of equine skin tumours. In: White N A, Moore J N (eds) Current techniques in equine surgery and lameness. Saunders, Philadelphia, PA

Fortier L A, MacHarg M A 1994 Topical use of 5-fluorouracil for treatment of squamous cell carcinoma of the external genitalia of horses: 11 cases (1988–1992). Journal of the American Veterinary Medical Association 204:1183–1185

Frauenfelder H C, Blevins W E, Page E H 1982 Sr^{90} for the treatment of periocular squamous cell carcinoma in the horse. Journal of the American Veterinary Medical Association 180:307–309

Fretz P B, Barber S M 1987 Prospective analysis of cryosurgery as the sole treatment for equine sarcoids. Veterinary Clinics of North America 10:847–850

Geor R 1997 Aminoglycoside dosing. In: Robinson N E (ed) Current therapy in equine medicine 4. WB Saunders, Philadelphia, pp 476–478

Goetz T E, Ogilvie G K, Keegan K G et al 1990 Cimetidine for treatment of melanomas in 3 horses. Journal of the American Veterinary Medical Association 196:449–452

Grier R L, Brewer W G, Paul S R, Theilen G H 1980 Treatment of bovine and equine ocular squamous cell carcinoma by radiofrequency hyperthermia. Journal of the American Veterinary Medical Association 177:55–61

Hare J E, Staemphli H R 1994 Cimetidine for the treatment of melanomas in horses: efficacy determined by client questionnaire. Equine Practice 16:18

Hepler D I, Lueker D C 1980 Letter to the Editor. Journal of the American Veterinary Medical Association 176:390

Hewes C A, Sullins K E 2006 Use of cisplatin-containing biodegradable beads for treatment of cutaneous neoplasia in equidae: 59 cases (2000–2004). Journal of American Veterinary Medical Association 229:1–6

Hoffman K D, Kainer R A, Schideler R K 1983 Radio-frequency, current-induced hyperthermia for the treatment of equine sarcoid. Equine Practice 5:24–32

Jeglum K A 1997 Melanoma. In: Robinson N E (ed) Current therapy in equine medicine 4. WB Saunders, Philadelphia, PA, pp 399–400

Joyce J R 1975 Cryosurgery for removal of equine sarcoids. Veterinary Medicine for Small Animal Clinicians 70:200–203

Knottenbelt D C 2005 A suggested clinical classification for the equine sarcoid. Diagnostic Techniques in Equine Medicine 3:278–295

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Knottenbelt D C, Kelly D F 2000 The diagnosis and treatment of periorbital sarcoid in the horse: 445 cases from 1974 to 1999. Veterinary Ophthalmology 3:169–191

Knottenbelt D C, Walker J A 1994 Topical treatment of the equine sarcoid. Equine Veterinary Education 6:72–75

Lane J G 1977 The treatment of equine sarcoids by cryosurgery. Equine Veterinary Journal 9:127–128

Latimer C A 1987 Diseases of the adnexa and conjunctiva. In: Robinson N E (ed) Current therapy in equine medicine, 2nd edn. WB Saunders, Philadelphia, p 442

Lavach J D, Sullins K E, Roberts S M et al 1985 BCG treatment of periocular sarcoid. Equine Veterinary Journal 17:445–447

McCluskey M J, Kavenagh P B 2004 Clinical use of triamcinolone acetonide in the horse (205 cases) and the incidence of glucocorticoid induced laminitis associated with its use. Equine Veterinary Education 16:86–89

Magdesian K G, Hogan P M, Brumbaugh G et al 1994 Pharmocodynamics of gentamicin administered once daily by the intravenous and intramuscular routes. Proceedings of the 40th Annual Convention of the American Association of Equine Practitioners. P 115–116

Mair T S, Couto C G 2006 The use of cytotoxic drugs in equine practice. Equine Veterinary Education 18:149–156

Manning T O 1983 Pemphigus foliaceus. In: Robinson N E (ed) Current therapy in equine medicine 1. WB Saunders, Philadelphia, pp 541–542

Munroe G A 1988 Cryosurgery in the horse. Equine Veterinary Journal 1:14–17

Owen R H, Jagger D W 1987 Clinical observations on the use of BCG cell wall fraction for treatment of periocular and other equine sarcoids. Veterinary Record 120:548–550

Page E H, Tiffany L W 1967 Use of autogenous equine fibrosarcoma vaccine. Journal of the American Veterinary Medical Association 150:177

Paterson S 1997 Dermatophytosis in 25 horses – a protocol of treatment using topical therapy. Equine Veterinary Education 9:171–173

Paterson S, Orrell S 1995 Treatment of biting lice (*Damalinia equi*) in 25 horses using 1% selenium sulphide. Equine Veterinary Education 7:304–306

Rebhun W C 1987 Immunotherapy for sarcoids. In: Robinson N E (ed) Current therapy in equine medicine 2. WB Saunders, Philadelphia, pp 627–638

Robinson N E 1987 Table of common drugs: approximate doses. In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, p 761

Rosencrantz W 1988 Equine allergies and urticaria. Veterinary Forum 6:14–15

Schwink K 1987 Factors influencing morbidity and outcome of equine ocular squamous cell carcinoma. Equine Veterinary Journal 19:198–200

Scott D W, Miller W M 2003 Immune mediated disorders. In: Equine dermatology. WB Saunders, Philadelphia, PA, pp 489–490

Southwood L L, Baxter G M 1999 Management of skin tumours. In: Colahan P T, Mayhew I G, Merritt A M, Moore J N (eds) Equine medicine and surgery. Mosby, Philadelphia, PA, pp 1847–1863

Spoormakers T J, Klein W R, Jacobs J J et al 2003 Comparison of the efficacy of local treatment of equine sarcoids with IL-2 or cisplatin/Il-2. Cancer Immunology and Immunotherapy 52:179–184

Spurlock S L, Hanie E A 1989 Antibiotics in the treatment of wounds. Veterinary Clinics of North America. Large Animal Practice 5:465

Stannard A A 1994 Corticosteroid therapy. Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Studer U, Marti E, Stornetta D et al 1997 The therapy of equine sarcoid with a non-specific immunostimulator – the epidemiology and spontaneous regression of sarcoids. Schweiz Tierheilkund 139:385–391

Tamzali Y 2006 Tumeurs des paupieres: deux cas de sarcoides palpebraux. Le Nouveau practicien Veterinaire equine 50:50–52

Tamzali Y, Teissie J, Rols M P 2001 Cutaneous tumour treatment by electrochemotherapy: preliminary clinical results in horse sarcoids. Revue Medicine Veterinaire 152:605–609

Tamzali Y, Teissie J, Rols M P 2003 First horse sarcoid treatment by electrochemotherapy: preliminary experimental results. Proceedings of the American Association of Equine Practitioners 43:381–384

Theon A P 1997 Cisplatin treatment for cutaneous tumours. In: Robinson N E (ed) Current therapy in equine medicine 4. WB Saunders, Philadelphia, pp 372–377

Theon A P 1998 Intralesional and topical chemotherapy and immunotherapy. Veterinary Clinics of North America. Equine Practice 14:659–671

Theon A P, Pascoe J R, Madigan J E et al 1997 Comparison of intratumoral administration of cisplatin versus bleomycin for treatment of periocular squamous cell carcinoma in horses. American Journal of Veterinary Research 58:431–436

Theon A P, Wilson W D, Magdesian K G et al 2007 Long term outcome associated with intratumoral chemotherapy with cisplatin for cutaneous tumours in equidae: 573 cases (1995–2004). Journal of the American Veterinary Medical Association 230:1506–1513

Turrel J M, Koblik P D 1983 Techniques of after-loading iridium-192 interstitial brachytherapy in veterinary medicine. Veterinary Radiology 24:278–279

Vaneslow B A, Abetz I, Jackson A R B 1988 BCG emulsion immunotherapy of equine sarcoid. Equine Veterinary Journal 20:444–447

Warnick L D, Graham M E, Valentine B A 1995 Evaluation of cimetidine treatment for melanoma in seven horses. Equine Practice 17:16–20

Wheat J D 1964 Therapy for equine sarcoids. Modern Veterinary Practice 45:62

Winthrow S J 1980 General principles of cryosurgical technique. Veterinary Clinics of North America 10:779–786

Wyman M, Rings M D, Tarr M J, Aldin C L 1977 Immunotherapy in equine sarcoid: a report of two cases. Journal of the American Veterinary Medical Association 171:449–451

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5 Viral diseases

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Viral skin disease can arise from primary cutaneous infection or in some cases as part of a more generalized viral disease. Most of the primary skin viral conditions are regarded as benign but where there are cutaneous manifestations of systemic viral infections the implications may be more significant. In the latter case, the cutaneous signs may be the most obvious (or sometimes the only) clinically detectable evidence; examples include equine viral arteritis and Getah (caused by an alpha virus of the Togaviridae family in south-east Asia). These diseases have non-specific cutaneous signs that can be easily overlooked or misdiagnosed.

There are important principles associated with confirmation of diagnosis in many of the viral conditions. The collection of samples for viral identification by electron microscopy or for culture usually requires special procedures because the viruses may be relatively fragile, being easily affected by minor changes in pH, light exposure, disinfectants etc., and in any case may only be found in significant numbers in localized sites within the lesion or in 'early' lesions. Modern PCR and antigen ELISA tests can be very sensitive at detecting the presence of any virus particles (Martens et al 2001). Also serological evidence can sometimes be used where a positive viral detection is not possible; this is particularly useful for systemic viral conditions with cutaneous signs. Additionally, skin lesions caused by viral diseases may be confusingly contaminated or complicated by bacterial or fungal infections.

Probably the most important primary viral skin diseases are those caused by pox viruses or papillomaviruses. However, there is little specific information about most of these viruses. Vesicular stomatitis is an important disease because it is probably transmissible to other animals and possibly even to humans, and in ruminants and pigs the condition can resemble foot and mouth disease; it is reportable in most countries but it can easily pass unnoticed because the signs are usually transient and can be mild. There are also several other important diseases with cutaneous signs such as African horse sickness, rabies (see pruritus, p. 442) and viral arteritis. In most of these conditions the dermatological symptoms are themselves trivial compared to the main ones but they can provide useful diagnostic support.

Horse pox (vaccinia)

Profile

This is a very rarely encountered disease of equine skin that was once regarded as a serious, highly contagious disease. The disease is a benign, mildly contagious, cutaneous infection due to an as yet unclassified pox virus. The virus is similar to the cowpox virus. It seems likely that 'horse pox' was not in fact a specific equine viral condition but was probably vaccinia infection; vaccinia is a cowpox-derived laboratory strain used for human smallpox vaccination.

There is a potential zoonotic implication.

Key points: Horse pox (vaccinia)

1 6 9 11 12 15 20 21 23 24 25

1. Rare disease probably related to a specific but unidentified equine pox virus or vaccinia (the cowpox-derived attenuated virus used for human smallpox vaccination).
2. Localized forms are more likely and the muzzle-mouth is the commonest site. Pastern dermatitis can be present.
3. Diagnosis can be confirmed by electron microscopy and by PCR methods.
4. Lesions can be painful but are usually transient; early and mild cases are seldom reported to veterinarians.
5. There is no treatment and no vaccination. Immunity following infection is probably life-long.

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Clinical signs

The disease occurs in two forms. In the buccal form, pox-like lesions occur in the mouth and skin of the lips and nostrils (buccal horse pox/contagious pustular stomatitis) (Fig. 5.1). This form lasts for around 3 weeks and is not considered to be serious. In some cases it may be overlooked completely. A condition resembling this condition was reported in a donkey (Jayo et al 1986).

In the cutaneous form, papular lesions which later become patent vesicles and then pustules covered with a fragile scab develop particularly over the flexor surfaces of the lower limbs (leg pox). Vulvar and penile lesions (genital pox) and palmar pastern inflammation can also occur. Extensive body involvement is singularly rare.

Vesicles rapidly develop over a period of about 4 days and burst to become dark/black umbilicated ulcers and then pustules with surface crusts and exudate. The lesions may be painful to the touch but are not usually pruritic. This form may closely resemble molluscum contagiosum (see p. 133) and in fact it may be the same disease.

Mild systemic signs (pyrexia and depression) may be seen.

Lesions may be detected on humans in contact with affected horses.

Differential diagnosis

- Molluscum contagiosum: geographically largely restricted to South Africa.
- Papillomatosis: very common, easily recognized, spontaneously resolving condition of young horses at grass in particular.
- Immune-mediated disease, such as pemphigus complex: generalized signs are more usual.
- Viral papular dermatitis: rare, slowly resolving viral disease – can be very similar.
- Dermatophytosis (ringworm): characteristic epidemiology and size and shape of lesions – diagnostic mycology.

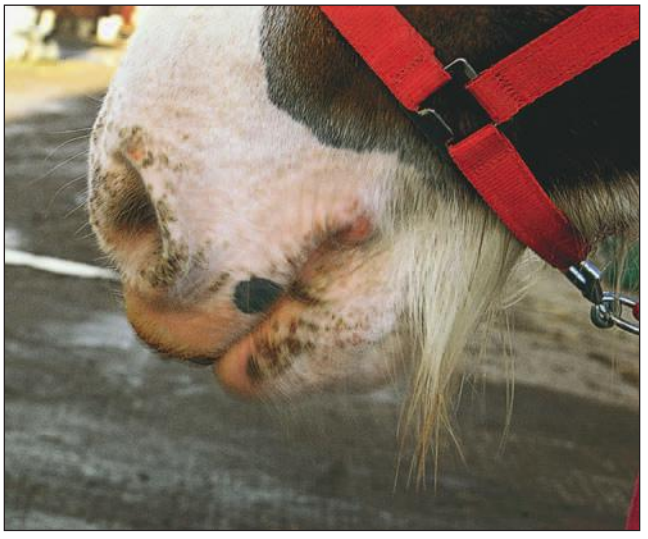


Figure 5.1 Typically mild, ulcerative, vesicular and scabby lesions of horse pox on the lips and nasal margin. This case showed mild discomfort only but actively resented palpation of the lesions. Diagnosis was confirmed by biopsy.

- Dermatophilosis: this is more exudative and occurs commonly; there is characteristic bacterial identification.
- Coital exanthema: a transient, venereal infection that usually affects only the external genitalia.
- Sarcoid: long-standing/persistent neoplastic condition with multiple clinical manifestations.

Diagnostic confirmation

- Clinical signs.
- Biopsy may show intracytoplasmic inclusion and 'ballooning' and sometimes microvesicle formation in keratinocytes in the stratum spinosum.
- Immunohistochemistry using indirect or direct fluorescent antibody stains on normal sections of affected skin.
- Virus isolation is characteristic and PCR methods can be confirmatory.
- Electron microscopic detection of the characteristic 'brick-shaped' virus particles.

Treatment

No specific treatment is available. Spontaneous remission with long-standing, solid immunity is usual after about 4 weeks. A few lesions may become infected with skin pathogens and then topical antiseptics may be useful.

No vaccine exists and indeed the number of cases and the mildness of the signs make this quite unnecessary.

Disinfection of the stable environment with a virucidal disinfectant such as a halogenated peroxygen compound may limit the spread.

Molluscum contagiosum

Profile

This is a mildly contagious cutaneous infection of horses of any age caused by an unclassified pox virus closely related to the horse pox group. Restricted geographically at present to South Africa but some cases may be encountered in other places including southern Europe (Scott & Miller 2003). Horse-to-horse transmission occurs slowly even when there is relatively close contact between infected and non-infected individuals. Spread by fomites is possible but less likely.

Key points: Molluscum contagiosum

1 5 9 12 15 16 20 23 24

1. Rare geographically restricted, largely to areas of southern Africa, viral (molluscipox) skin disorder affecting horses and humans.
2. Possibly transmitted to horses from humans or by direct close contact between horses or by insect vectors.
3. Miliary, transient or long-lived papular lesions with a waxy appearance occurring all over the body with most in the hairless skin such as the inguinal region, and face.
4. Diagnosis is easily confirmed by characteristic histopathology.
5. No treatment is warranted or available. Slow resolution may occur but some lesions may persist.

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A similar disease occurs in humans and the virus and the pathology are very similar (if not identical). Horses may be infected by humans (Thompson et al 1998).

Clinical signs

The condition is non-painful and non-pruritic. There are no apparent systemic effects.

Multiple, raised, well-defined grey/white papules with a waxy surface (1–6 mm) occur primarily in hairless or sparsely haired (glabrous) areas such as the penis, prepuce, scrotum, mammary glands, thighs (Fig. 5.2), axilla and muzzle. Other body areas such as the rump, neck and chest can also be affected and focal alopecia is a common sign **(Fig. CD5 • 1A and B)**. Individual lesions may coalesce and form larger aggregations closely resembling papillomas (see Fig. 5.6) (Moens & Kombe 1988).

Older lesions become smaller and umbilicated. They may develop a central pore from which a caseous plug is extruded. The lesions may become 'horny' and resemble viral papillomas.

In areas where hair is thicker, nodules are covered with powdery scabs which, when detached, remove the hairs.

Differential diagnosis

- Papillomatosis: transient fibro-papillomatous condition with a characteristic pattern of development in many parts of the world. Differentiation is difficult where both conditions are endemic; histology and electron microscopy is characteristic.
- Uasin Gishu disease/viral papular dermatosis: can be very similar in appearance but occurs in geographically distinct regions.
- Immune-mediated skin diseases (pemphigus complex/sarcoidosis, etc.): multifocal seborrheoic persistent autoimmune dermatosis.
- Occult/verrucose sarcoid: characteristic variations in clinical appearance and persistence with a tendency to exacerbation on interference.
- Dermatophytosis (ringworm): transient, alopecic and scaling dermatosis with characteristic epidemiology and mycological examinations.
- Horse pox: localized, transient and geographically distinct regions.
- Coital exanthema: transient venereal pustular condition restricted largely to external genitalia.

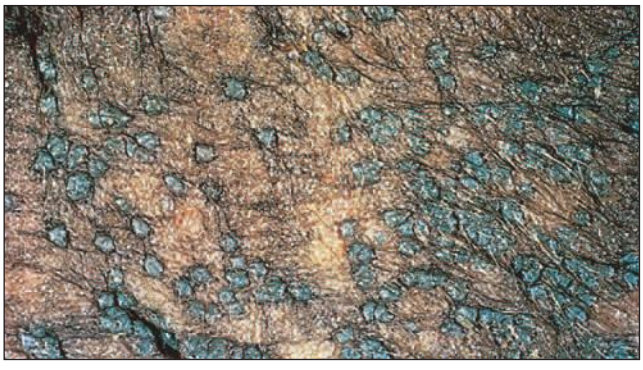


Figure 5.2 Molluscum contagiosum lesions showing the typical waxy, raised papules.

Diagnostic confirmation

- Characteristic histopathology showing epidermal hyperplasia and hypertrophy. Individual keratinocytes are swollen and contain large intracytoplasmic inclusion (molluscum) bodies **(Fig. CD5 • 2A–C)**. There is usually little or no dermal inflammation.

CD

Treatment

No specific treatment is available. Spontaneous remission with new hair growth has been reported to take up to 6 years and some sites remain hairless and thickened for many years longer. Complete resolution is not reported.

CD

Sensible precautions include isolation, hygiene controls between affected and non-infected horses and fly control.

Human infection probably does not occur from affected horses but the risk of this is not known definitively.

Vesicular stomatitis (red nose; sore nose)

Profile

This is a sporadic contagious viral disease of limited geographical distribution caused by Lyssa virus (family Rhabdoviridae). Most cases are encountered in spring and late summer. The virus is probably spread by insect vectors (biting, sucking and surface-feeding flies including *Culicoides* spp., *Simulium* spp. and muscid flies) and possibly by fomites (Knight & Messer 1983). The prevalence is highest in closely congregated animals.

Clinical signs

Inflammation and vesicle formation in mouth and tongue occur and to a lesser extent on the skin of the nose, mammary gland and prepuce (Fig. 5.3 and **Fig. CD5 • 3A–C**). Coronet and digital skin are also sometimes affected. Vesicles rupture leaving a very painful, small, ulcerated lesion.

CD

Major signs include inappetence, fever, depression and lameness. Extensive oral ulceration causes drooling of saliva and intense rubbing of the mouth and lips on mangers or doors. Lesions resolve over 3–7 days with solid immunity.

Scars of previous infection on the skin may be seen as focal depigmented spots (Donaldson & Ferris 1995).

Key points: Vesicular stomatitis

1 6 7 11 15 23 24 25

1. Geographically largely restricted to North and South America.
2. Probably most significant transmission is by biting insects but direct close contact is also a possible route. Feeding troughs and water buckets may be involved in the spread between horses.
3. The transient signs are usually restricted to the mouth and lips but are occasionally found elsewhere.
4. Miliary, small, painful vesicles and ulcers cause inappetence and salivation (often with some blood). Lesions on the coronary band can lead to lameness.
5. Treatment is not warranted – spontaneous resolution is the norm.

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Figure 5.3 Vesicular stomatitis. The intact vesicles are fragile and burst easily, leaving focal circular ulcerated areas that heal quickly. There may be pain, and oral lesions induce bloodstained salivation. (Figure courtesy of Paul Owen.)

Differential diagnosis

- Viral papular dermatosis: an unusual viral disease with transient unilateral papules.
- Molluscum contagiosum: a geographically restricted viral wart-like disorder showing waxy papules over extensive areas; resolution is usually very slow.
- Horse pox ('mouth pox' forms): transient vesicular and papular lesions mainly around the mouth and nose.
- Bullous pemphigoid/pemphigus vulgaris: a multifocal or diffuse (generalized) exfoliative autoimmune dermatopathy with characteristic histological findings.
- Contact irritants/chemical injuries: history of exposure is usually established and some can be detected directly.

Diagnostic confirmation

- Characteristic clinical features and history in endemic regions
- Biopsy is non-specific but the marked localized epidermal oedema and superficial perivascular dermatitis leading to epidermal necrosis can be suggestive
- Serology
- Virus isolation/identification is possible.

Treatment

None is possible or required.

Isolation of affected cases is wise and, if relevant, reporting of cases to the disease regulatory body (in view of possible transmissibility to cattle and confusion with foot and mouth disease and to humans). No vaccine exists.

Equine viral arteritis (EVA)

Profile

Equine viral arteritis is a contagious disease caused by a member of the Arterivirus family (formerly known as Togaviridae). It occurs sporadically in many parts of the world with a low prevalence, at a low level. The virus causes abortion, infertility and skin disease; some strains are relatively less virulent and infection may go unnoticed. Transmission is through the respiratory tract (by droplet and microaerosol) or urogenital tract (urine or semen).

The incubation period varies between 3 and 14 days.

Although its transmission is usually venereal, horses can be infected via the respiratory route. The virus is carried in the testicular tissue of carrier stallions. Mares have a transient infection and no carrier status is recognized.

Key points: Equine viral arteritis

1 Equine viral arteritis is a rare but important contagious disease of horses transmitted venereally by infected (carrier) stallions and by the respiratory route.
2 The disease causes minor skin lesions including oedematous plaques resembling urticaria and localized exudative dermatitis.
3 The major significance is reproductive (abortion).
4 Diagnosis is confirmed by serology and by viral isolation/identification in seminal fluid.
5 There is no treatment but an effective vaccination is available for restricted use. Affected stallions must be either castrated or killed as they act as a persistent source of infection.

Clinical signs

Cutaneous signs The signs include a localized or more generalized skin rash resulting from vasculitis with skin papules and plaques closely resembling urticarial lesions. These are relatively trivial and easily overlooked. However, they can be the only external evidence of the condition.

Systemic signs Variable, pyrexia and depression. Increased respiratory rate. Oedema of limbs and less frequently, head, ventral abdomen, scrotum, prepuce and conjunctiva ('red eye'). Coughing may or may not be associated with the disease. Reduced exercise tolerance in horses showing clinical disease. Abortion occurs in affected pregnant mares during or just after the febrile phase.

Differential diagnosis

- Urticaria/allergy (including food/contact allergy): transient or recurrent and sometimes more persistent urticarial wheals, usually occurring without any systemic signs; a few cases are mildly pruritic.
- Purpura haemorrhagica: history of recent viral or bacterial (streptococcal) infection; widespread petechial and ecchymotic mucosal haemorrhages, characteristic ventral oedema and skin exudation (angio-oedema).
- Insect bites/stings: single or multiple foci of urticaria with a central focus; history of attack may be known.
- Dermatophytosis: prolonged self-resolving superficial mycosis with characteristic epidemiology, mycology and histology with spontaneous resolution after 6–12 weeks.

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- Viral and bacterial respiratory infections.
- Other causes of abortion: most do not have concurrent primary skin disease.

Diagnostic confirmation

- Virus isolation from respiratory (upper respiratory tract swabs) or reproductive tracts (semen from 'shedder' stallions). Virus can also be isolated or identified in the buffy coat of blood and in urine as well as the placenta of aborted mares.
- Paired sera 14 days apart are necessary for the serum neutralization test (titre > 1:4 is regarded as positive).
- Laboratory findings include a mild leukopenia.
- Histopathology: necrosis of the media of small arteries and neutrophilic infiltration and thrombosis of smaller submucosal vessels. Viral antigen may be detected using fluorescent immunohistochemistry.

Treatment

No treatment is available.

Vaccinations are effective and can be used to control the spread of the disease in a population of susceptible horses. The carrier status of the stallion depends on testosterone and so castration or killing carrier stallions is the only means of elimination of the carrier state. Vaccination of infected stallions does not treat the carrier state. No carrier state is found in mares – the infection is transient and immunity is long-standing and solid.

Equine viral papular dermatosis (Uasin Gishu disease)

Profile

This is an infectious skin disease caused by an unspecified pox virus. The disease is named after the Uasin Gishu plateau in Africa where it was first encountered. It is recorded in some parts of the USA also.

Spread is direct or indirect by vectors such as flies. Incubation period is about 7 days with a course of 2–3 weeks.

Clinical signs

Sudden appearance of many anular papular lesions which quickly develop crusts with hair loss primarily over the trunk and scrotal skin (Fig. 5.4). Lesions are not pruritic or painful and become scaly after about 2 weeks. There are no systemic signs and most cases resolve within 4–6 weeks. A few cases have a persistent scaling or nodular lesion that may take years to resolve.



Figure 5.4 Unilateral viral papular dermatitis lesions on the scrotum of a stallion.

Differential diagnosis

- Dermatophytosis (ringworm): roughly circular or coalescent areas of alopecia and scaling with mild skin irritation and possibly a little pruritus. Characteristic epidemiology, mycology and histology with spontaneous remission after 6–12 weeks. Infected stables and tack remain a source of recurrent infections in susceptible horses.
- Sarcoid (occult): can be very similar but there are usually other typical sarcoid types present also.
- Horse pox: transient condition localized usually to the mouth and muzzle regions.
- Pemphigus foliaceus: multifocal, autoimmune exfoliative bullous disease.
- Molluscum contagiosum: viral papular lesions in horses in central and southern Africa; characteristic histology.
- Urticaria/insect bites: transient oedematous/urticarial lesions possibly with a central inflamed spot indicative of a sting or bite.
- Coital exanthema: transient venereal disease affecting external genitalia mainly.

Diagnostic confirmation

- Virus isolation from papular lesions is possible.
- Biopsy shows hyperplasia, superficial and deep dermatitis with ballooning degeneration and eosinophilic intracytoplasmic inclusion bodies.

Treatment

No specific treatment is needed. The disease has no systemic implications and resolves spontaneously.

Isolation of affected horses is probably wise and disinfection of premises may help limit its spread.

Key points: Equine viral papular dermatosis

6 9 15 23 24

1. A rare and poorly understood skin disorder of horses of all ages and types.
2. Acute development of non-painful, non-pruritic, crusting anular papules over limited areas (sometimes unilateral).
3. Lesions last around 2 weeks but a few cases may have more persistent lesions.
4. Spontaneous resolution in 2–4 weeks.
5. Neither treatment nor vaccination is warranted or available.

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CD

Congenital papillomatosis (neonatal wart/epidermal naevus)

Profile

The equine papillomavirus apparently has the ability to pass across the placenta to infect the skin of the fetus. Viral papillomas are not an uncommon finding on the skin of newborn foals and they may also be identified on the chorionic surface of the placenta. This may also be regarded as a naevus and some cases have no detectable viral state (see also p. 242).

Clinical signs

Most often a small, localized, cauliflower-like, flattened single wart or a few 'warts' ranging between 5 mm and 20 cm are identified on the skin of a newborn foal at any site on head, neck or trunk (Atwell & Summers 1977) (Fig. 5.5). They are usually single and no other signs are usually present. True congenital viral papilloma may also be identified on the placenta, so examination of the placenta can be helpful diagnostically.

Key points: Congenital papillomatosis

3 4♂ 9 12 14 20 23 24

1. A rare, incidental condition encountered in newborn foals.
2. Papillomatous lesions of variable size and number on any part of the body. Single lesions are most common. Examination of the placenta at birth is a useful aid to diagnosis; papillomatous lesions may be obvious. Some cases are severe.
3. Spontaneous resolution does not occur but surgical excision or cryosurgery is curative.
4. Recurrence does not occur; the presence of a congenital papilloma does not means that the foal will not get viral papilloma ('grass warts') or sarcoid later in life, so the condition may in fact represent a naevus rather than a papilloma.
5. Prevention is not possible and in any case the condition is an incidental cosmetic condition.



Figure 5.5 Congenital (viral) papilloma on a foal.

There is no implication to their presence apart from the cosmetic aspects. Occasionally they can become severe or extreme **(Fig. CD5 • 4A and B)**.

Differential diagnosis

- Congenital epidermal naevus: this can be very similar and the two conditions could be the same.
- Congenital vascular hamartoma/cavernous haemangioma: usually single on the distal limbs of (mainly) Arabian foals. Similar appearance but easily traumatized with heavy bleeding; slow expansion. Some extreme forms also found on the belly wall and face.

Diagnostic confirmation

- Diagnosis is usually based on clinical appearance but this is not really enough to differentiate it from the other important similar conditions which carry very different prognoses.
- Histopathology is an important early aid (the placenta can be helpful also).
- Virus isolation on fresh unpreserved samples is usually disappointing; sampling the placental lesions may be more rewarding.

Note

Serology is not helpful as seroconversion does not usually take place.

Treatment

Congenital viral papillomas are easily treated by ligation or surgical removal with little problem. Extensive lesions will necessarily create bigger challenges.

Podophyllin paste (50%) applied daily for 20 days may also be effective. Silver nitrate applications using a styptic pencil may be curative for very small lesions.

Spontaneous resolution seldom occurs.

Viral papillomatosis

Profile

Papillomatosis is a viral skin disease caused by a host-specific papovavirus (equine papillomavirus) affecting the basal cell layers of the epithelium. Usually the disease is encountered mainly in younger horses (6 months to 4 years) but all immunologically susceptible animals can be affected, so older horses that have not been exposed previously may be infected. Immunocompromised horses such as those diagnosed with pituitary pars intermedia dysfunction (PPID/equine Cushing's disease) (see p. 322) may also become infected.

The disease has an incubation period of approximately 60–70 days and is moderately contagious by direct and indirect contact between co-grazing horses (hence the name 'grass warts').

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Clinical signs

Young horses under 3–4 years of age are commonly affected. Sequential crops of yearlings and young horses can be affected on individual premises.

Multiple (often many) single or coalescent pink or grey vegetative lesions most often on muzzle (Fig. 5.6), lips, face (see Fig. 5.8). The distal limbs and paragenital regions can be affected also. Occasional cases are presented with extreme forms where the papillomas coalesce into plaque-like lesions which may bleed with minor trauma.

Lesions usually increase in number rather than the size of individual lesions but coalescence is common where many lesions occur in the same locality.

Key points: Viral papillomatosis

1 A very common, well-recognized viral disease of younger horses (1–4 years old) developing most often during their first year at grass. The condition is caused by a specific equine papillomavirus. Individual premises and fields may have sequential groups of affected young horses. The condition in adult horses is less common.
2 Multiple papular 'wart' lesions that may coalesce, particularly developing over the face, groin and less commonly at other sites such as the distal limb and neck. A few cases are severely affected. There are no systemic implications in most cases.
3 Diagnosis is relatively straightforward based on the clinical presentation and characteristic epidemiology. Biopsy is diagnostic. Viral isolation is not necessary.
4 Treatment is not usually warranted as spontaneous resolution usually occurs except in older horses where the lesions may persist. Surgical excision and topical antiviral and 'wart' creams can be effective. Autogenous vaccination can be effective.
5 The adult onset form is much more persistent. Prevention is difficult apart from avoidance of 'infected' premises and pastures.

Some cases can be severely affected and there may be bleeding and ulceration and then there may be systemic signs (Fig. 5.7).

Usually affected horses show no evidence of either systemic responses or local discomfort or pruritus.

In older horses the lesions tend to be more isolated and there are usually fewer of them. The groin and perineum **(Fig. CD5 • 5)** are more often affected than the face but any site can be affected in a hitherto non-exposed (non-immune) horse. The condition can affect older immunocompromised horses, such as those suffering from PPID/Cushing's disease.

CD

Single large lesions can be found on the vulvar lips or on the preputial skin or the free portion of the penis. Lesions on older horses are usually squamous papillomas and may be persistent. Most are progressive but some remain static for many years. There is some suggestion that squamous papilloma represents a precursor to squamous cell carcinoma

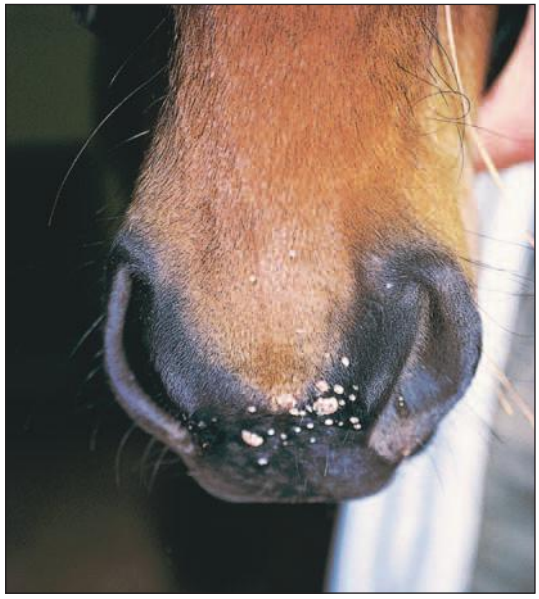


Figure 5.6 Viral papillomas on the muzzle of a grazing yearling Thoroughbred. Notice that some of the lesions have coalesced.





Figure 5.7 (A, B) A severe case of viral papillomatosis. Note the coalescing lesions and the superficial haemorrhaging. This yearling was mildly discomforted by the condition but recovered spontaneously over 6–7 weeks without any treatment.

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Figure 5.8 Healing discrete viral papillomas on the face of a yearling.

of the penis and prepuce of males or the clitoris and vulva in mares.

Differential diagnosis

- Pinnal acanthosis/aural plaques: distinctive persistent plaque and papular condition affecting the inner surface of the pinna and occasionally the ventral midline skin and lateral aspects of the sheath/thighs.
- Verrucose sarcoid: usually persistent and exacerbating over time; several different forms exist; seldom self-resolving; characteristic histology.
- Squamous cell carcinoma: aggressive, neoplastic disease with facial and vulvar proliferative and destructive (ulcerative) forms; precancerous changes can be squamous papilloma; distinctive histopathology.
- Molluscum contagiosum: geographically restricted, more persistent and characteristic histological features.
- Vaccinia/horse pox: rare vesicular/pustular dermatitis with a transient nature usually around the muzzle and mouth regions; virus isolation/identification by electron microscopy can be diagnostic.

Diagnostic confirmation

- Characteristic clinical appearance and history in young horses including the previous occurrence of the condition in other similar horses in previous years.
- Contagion (others of the same age affected).
- Biopsy for histopathology is diagnostic.

Treatment

CD

No treatment is usually required. Most lesions on young horses will resolve spontaneously after 3–4 months (Fig. 5.8 and **Fig. CD5 • 6A–D**).

Autogenous vaccines have a fairly good reputation but it takes some weeks to prepare the vaccine and complete the required course of injections (usually three to four are required at weekly intervals). It is uncertain whether there is definite clinical advantage from this method.

Note

Autogenous vaccines derived from a single horse should not be used on other animals even though they may be similarly affected.

Surgical removal of isolated lesions is feasible and curative. Sites where this is required on occasion include around the eyes and commissure of the mouth. Some lesions respond well to cryosurgery, radiofrequency hyperthermia or surgical removal.

Topical 'wart' medications including Posalfilin, formalin ointments, and silver nitrate pencils can be used to good effect in limited sized lesions in convenient locations.

Spontaneous or induced resolution is the norm for this condition and usually leaves no scar. However, a few affected areas can be hairless or sparsely haired and some pigmentary changes may occur (especially in perineal and inguinal regions).

Spontaneous resolution does not occur in most adult cases – the papillomas remain static or slowly expand. Autogenous vaccines are probably not helpful in adult horses. Surgical removal, cryosurgery or topical wart medications are the main options. Where an old horse is very badly affected, aspects of immunocompromise may need to be managed.

Prevention of infection in young horses is difficult – where a specific pasture is related to high prevalence a long period of rest possibly with co-grazing by ruminants can reduce the risk. However, the virus appears to survive for several years and so even this may not prevent all cases. The immunity following infection and spontaneous recovery appears to be solid and so the disease is usually tolerated as a cosmetic and transient nuisance only. Prior infection with equine papilloma does not prevent the development of sarcoid (see p. 387) or pinnal acanthosis (see below).

Pinnal acanthosis (hyperplastic aural dermatitis/aural plaques/fungal plaque)

Profile

Pinnal (papillary) acanthosis (pinnal hyperkeratosis/aural plaque/fungal plaque) is a very common condition in horses over 2 years of age, and is probably a clinically distinct form of papilloma caused by an as yet unidentified papillomavirus, other than the typical equine papillomavirus (Williams 1997).

Given that the clinical lesions are restricted in area to the medial pinnal skin and the groin and thighs (less commonly) and that the onset of the condition is largely restricted to the summer months, it seems likely that the causative (papilloma) virus is transmitted by blackflies (*Simulium* spp.). Studies suggest that *S. vittatum* and *S. argus* are involved (Fairley & Haines 1992). These species occur widely and have feeding habits that include preferred feeding sites in the inner surface of the pinnae and the ventral midline and sheath region of geldings in particular. They can also feed at other sites such as the medial thighs and ventrum – and particularly in the umbilical and preputial region of male horses and this corresponds with the development of the less common inguinal form of the condition.

Papillomavirus can be demonstrated in a high proportion of these plaques by electron microscopy and by immunostaining.

There is no evidence that this disease has any fungal component at all – the historical name 'fungal plaque' is therefore a misleading misnomer!

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Key points: Pinnal acanthosis

9 12 15 17 20 23 24

1. This is a very common cosmetic nuisance in horses worldwide. The disease is probably caused by a papillomavirus unrelated (at least immunologically) to the common equine papillomavirus. It is assumed from the distribution of the lesions that *Simulium* spp. flies are involved in the transmission of the condition.
2. Grey-pink, papular and coalescent plaque-like areas develop characteristically on the inner skin of the pinna. Similar lesions are sometimes found on the ventral midline anterior to the prepuce or on the glabrous preputial skin or the medial thighs (less significantly). More generalized cases do occur. Early developing stages can cause marked or mild ear discomfort and resentment of handling. A few cases have extreme changes with extensive flaking and scaling and even some ulceration. There are usually no systemic signs and even in the early stages the clinical effects are mild.
3. Diagnosis is usually based on clinical recognition but biopsy and electron microscopy with virus identification is possible. Some forms can closely resemble the common viral papilloma or the verrucose form of sarcoid.
4. Self-cure does not occur and autogenous vaccines are not helpful. Treatment is not warranted. Topical medications or surgical/cryosurgical removal can lead to severe ear resentment and seldom brings a cure. Imiquimod may be useful in some cases.
5. The commoner forms of the condition should be viewed as a cosmetic nuisance. Severe cases present major clinical and therapeutic dilemma.

Clinical signs

The earliest lesions are small (1–4 mm diameter), raised, well-demarcated, depigmented (pink, grey or cream) papules most often on the medial pinnal skin. The clinical appearance of lesions seen in the various anatomic locations does vary somewhat. However, the common features are localized, roughly circular, small grey-cream-pink papules in the epidermis.

In the ear form (aural plaques/pinnal acanthosis) they are raised, usually flat-surfaced, depigmented lesions of variable size (ranging from 1–3 mm to 5–10 mm diameter) on the inside surface of one or both ears (Fig. 5.9). They may be solitary and discrete or variably coalescent, and usually have marked scaling and flaking. In some more advanced cases they are thickened and hyperkeratotic with abundant exfoliating keratinocytes **(Fig. CD5 • 7)**. Lesions can be slow to develop or undergo periods of mild or more significant exacerbation from time to time. However, some remain static for years. It is unusual to have a single ear affected. The pinnae can be painful and handling is then invariably resented, especially in early developing lesions. Severe cases may be presented with ear discomfort or even overt rotatory or side-to-side head-shaking. Some of these cases appear to have marked pain associated with the condition and some have secondary otitis externa as a result of dermal exfoliation into the ear canal.

A very similar condition with typical plaque-like papules also occurs at the other preferred feeding site for the *Simulium* spp. flies such as on the ventral midline forward of the sheath, the external preputial skin, the scrotum and sometimes the medial thighs (Fig. 5.10).



Figure 5.9 The earliest signs of pinnal acanthosis (ear plaques/fungal plaques) appear as single small depigmented foci that may then expand and thicken and ultimately may coalesce into larger areas of hyperkeratotic and thickened skin.

On the ventral midline it can be confused with ventral midline dermatitis related to insect bite hypersensitivity. Mares seldom have more than a few lesions on the ventral midline of the condition, thus implying that the feeding habits of the fly involve odours and environments that are only present in males.

A severe form can affect individual animals with facial and body lesions; limb lesions can also be found but they tend to be more isolated and fewer in number. Usually these cases are severely affected with massive numbers of coalescing papular lesions. Whilst this form may resemble the more typical viral papilloma, the individual lesions still have a very different appearance. There is loss of pigmentation and hair and they seldom resolve. This may be associated with some forms of immunocompromise but little is known about this form.

It is possible to find lesions in the ventral midline/genitalia regions without lesions in the ear.

Differential diagnosis

CD

- Equine papillomatosis: transient similar disorder seldom encountered in the pinnal region and self-resolving in young horses during grazing seasons; epidemiology is distinctive; the histology of the two conditions is similar but depigmentation is less common in viral papillomatosis.
- Papular dermatosis/Uasin Gishu disease: geographically restricted papular skin lesions occurring on the body trunk and head.
- Molluscum contagiosum: geographically restricted and seldom involves either the ear or the ventral midline.
- Horse pox (genital form): restricted to genitalia, transient and self-resolving.
- Bullous pemphigoid: severe vesico-bullous disorder affecting the skin, mucocutaneous junctions and mucous membranes; characteristic histology.

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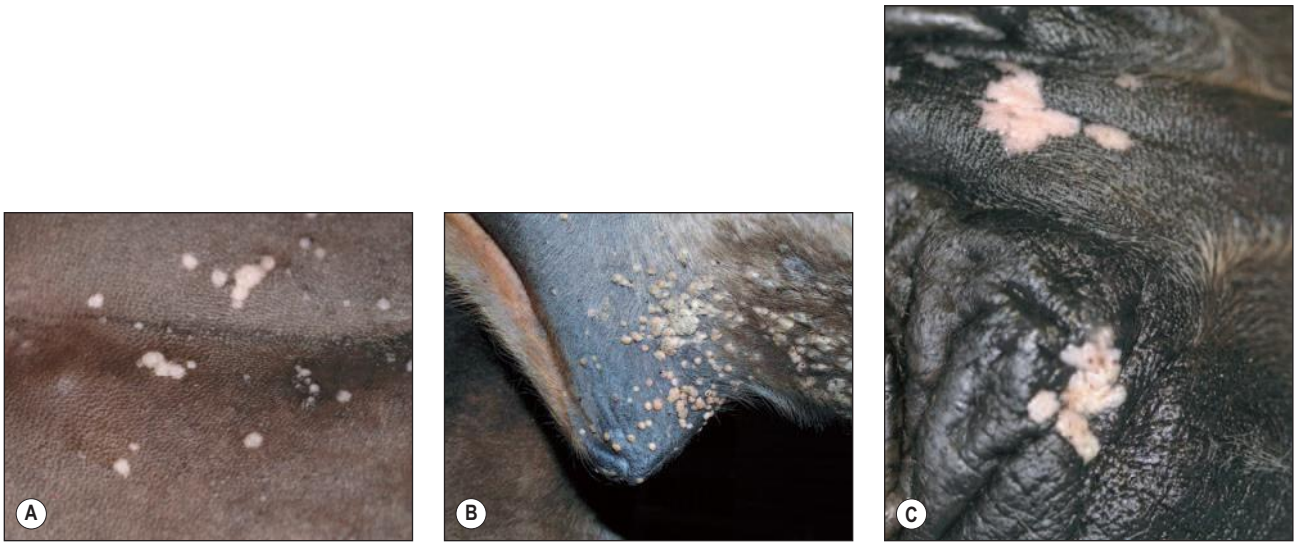


Figure 5.10 Viral papillomatous acanthosis on the ventral abdomen **(A)** and sheath **(B)** of a middle-aged gelding. **(C)** The skin of the ventral abdomen anterior to the sheath shown close up after clipping. Note the typical non-pigmented acanthosis lesions; note also the coalescence of the adjacent lesions possibly relating to the bite sites of an 'infected' fly. These lesions developed at the same time as the condition in the ears.

- Other genital infections (including coital exanthema/dourine): typical restriction to genitalia with more extensive inflammatory responses not restricted to the epidermis; epidemiological features.
- Vesicular stomatitis: rare localized vesicular disease of the mouth predominately.
- Verrucose sarcoid: can be similar but usually there are other typical lesions and they are in any case histologically distinctive.
- Squamous cell carcinoma/squamous papilloma (precancerous stages): squamous papilloma is restricted to the ocular and palpebral tissues and the skin of the male and female external genitalia; ulcerative and proliferative forms exist with exudation and bleeding with little pain.

Diagnostic confirmation

- Clinical signs are highly distinctive but sometimes difficult to differentiate clinically from other papular dermatoses.
- History may reveal challenge by *Simulium* spp. flies – most cases occur in late summer or autumn and become obvious by the following spring.
- Virus isolation, electron microscopy or immunostaining are only successful when samples are taken early in the course of disease (fresh lesions are therefore essential).
- Biopsy is distinctive but quite similar to viral papilloma apart from the singular depigmentation that is a strong feature.

Treatment

By contrast to viral papilloma, viral (pinnal/inguinal) acanthosis seldom resolves spontaneously and so there is inevitable pressure for a 'cure', especially in show horses. However, pinnal/ventral viral acanthosis (aural/ventral plaques) should not be subjected to treatment attempts – the cure is frequently worse than the disease and many cases become incurably head/ear shy. Attempts to treat these lesions with autogenous vaccines, retinoids, anti-wart ointments and some sarcoid treatments including imiquimod (Aldara) have usually failed to resolve them. There are anecdotal reports of successes with imiquimod.

Given that the condition is so benign (in most if not all cases) and given that the treatments often result in severe aural resentment there is little to justify any interference at all except in the most severe cases. Where the lesions are severe and have an effect on the horse, then a long period of rest is a far better approach than any attempt to surgically remove or otherwise treat the lesions.

Control

There is no vaccine at present. The condition probably cannot be directly transmitted between horses. The fly bite appears to be the main source of viral entry.

Equine coital exanthema

Profile

This is a contagious, venereal, viral skin disease caused by equine herpesvirus (EHV-3). (Pascoe et al 1969). Transmission occurs also through indirect contact (fomites) and by inhalation of virus-laden droplets. The incubation period is 5–7 days. Epidemiologically the disease is really only significant in breeding animals.

Immunity is solid and so most horses are only infected once.

Clinical signs

Multiple, rapidly developing papules (1–5 mm diameter), which develop necrotic tops and become obvious pustule-like lesions on the penis – principally in the area of the junction of parietal and visceral folds of the prepuce on the dorsal surface (the site of the most prolonged contact with the vulva during coitus) (Fig. 5.11). Some horses may also develop lesions around the nose and mouth.

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Key points: Equine coital exanthema

5 6 9 11 15 17 18 23 24

1 A viral venereal disease caused by equine herpesvirus 3 usually restricted to breeding animals. Transmission by indirect or droplet routes also possibly occurs.
2 Clinical signs are usually transient with small painful cutaneous vesicles on the external genitalia following infected coitus. There may be fever and transient local pain. The vesicles rupture leaving a small crater with a pustular centre. Healing lesions are usually depigmented.
3 Diagnosis is usually simple with a close record of recent breeding and can be confirmed clinically by the finding of similar lesions on the penile shaft and the vulva and perineum of the mare.
4 Treatment is not usually warranted – soothing antibacterial creams may speed recovery and local anaesthetic may reduce the discomfort. Breeding rest should be maintained until the lesions have healed completely.
5 Spontaneous resolution may occur over some days. Immunity is partial with recrudescence of latent infections occurring during times of stress in some horses.



Figure 5.11 Equine coital exanthema on a stallion's penis.

In mares similar lesions develop on the mucosal surface of the vulva and often extend to the skin of the vulva and perineum (Fig. 5.12); severe cases can also affect the anus and tail skin. Perineal swelling and oedema of the vulvar lips develop by the fourth day.

The lesions may be mildly pruritic but are not painful. The developing lesions closely resemble pustules but they do not produce identifiable liquid pus. It would seem that the initial cell necrosis is not followed by liquefaction. Healing lesions become covered with serum-encrusted scabs. Over 3–5 days oedema regresses and healthy granulation occurs by 10–14 days. Healing lesions in both mares and stallions may leave permanently depigmented spots on the skin (leukoderma).

Typically of latent herpesviruses, individual horses may show recurrent recrudescence during periods of stress (including transport, disease or pregnancy).



Figure 5.12 Equine coital exanthema on the vulval lips and perineal skin of a mare.

Differential diagnosis

- Papular dermatitis: geographically distinctive and without relationship to breeding; and restriction to the genitalia.
- Molluscum contagiosum: geographically restricted and not restricted to genitalia and breeding history.
- Horse pox (genital form): can be very similar but distinctive virus can be found on electron microscopy and PCR.
- Bullous pemphigoid: severe bullous disease with multifocal oral and skin lesions.
- Other genital infections, including dourine: more involvement of the genital tract.
- Vesicular stomatitis: transient vesicular disease affecting oral and skin sites with geographical restriction.
- Ventral and sheath forms of viral acanthosis: persistent or slowly resolving pink or grey papular lesions without other symptoms; may be extensive and miliary.
- Squamous cell carcinoma (precancerous stages).

Diagnostic confirmation

- Clinical signs.
- History.
- Virus isolation: only successful when samples are taken early in the course of disease (fresh lesions are essential).
- Paired sera 14–28 days apart can be used for serum neutralization test (SNT) and complement fixation test (CFT).

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Treatment

Breeding from infected animals should be stopped completely until at least 3 weeks after the lesions have healed.

The acute lesions can be cleaned carefully with an antiseptic solution and dressed with a local anaesthetic antibacterial cream such as human haemorrhoid cream. Scabs should be removed gently with a dilute hydrogen peroxide (5 vol) and individual spots medicated with strong iodine solution. A daily treatment with a cotton bud dipped in a soothing antibiotic/antiseptic ointment may be helpful but as most improve quickly this may be more resented than the condition.

The value of parenteral corticosteroids is doubtful in the management of any latent herpesvirus infection but topical applications can be helpful.

There is no vaccine at present. Most cases become permanently immune following recovery.

Sarcoid

See pages 387–407.

References

Atwell R B, Summers P M 1977 Congenital papilloma in a foal. Australian Veterinary Journal 53:229

Donaldson A I, Ferris N P 1995 Vesicular stomatitis. Equine Veterinary Education 7:205–206

Fairley R A, Haines D M 1992 The electron microscopic and imunohistochemical demonstration of papillomavirus in equine aural plaques. Veterinary Pathology 29:79–81

Jayo M J, Jensen L A, Leipold H W, Cook J E 1986 Poxvirus infection in a donkey. Veterinary Pathology 23:635

Knight A P, Messer N T 1983 Vesicular stomatitis. Compendium of Continuing Education 5:S517–S519

Martens A, De Moore A, Ducatelle R 2001 PCR detection of bovine papilloma virus DNA in superficial swabs and scrapings from equine sarcoids. Veterinary Journal 161:280–286

Moens Y, Kombe A H 1988 Molluscum contagiosum in a horse. Equine Veterinary Journal 20:143–145

Pascoe R R, Spradbrow P B, Bagust T J 1969 An equine genital infection resembling coital exanthema associated with a virus. Australian Veterinary Journal 45:166–170

Scott D W, Miller W H 2003 Viral and protozoal skin diseases. In: Equine dermatology. WB Saunders, St Louis, MO, pp 379–381

Thompson C H, Yager J A, Van Rensburg I B 1998 Close relationship between equine and human molluscum contagiosum viruses demonstrated by in situ hybridization. Research in Veterinary Science 64:157–159

Williams A M 1997 Papillomatosis: warts and aural plaques. In: Robinson N E (ed) Current therapy in equine medicine 4. WB Saunders, Philadelphia, pp 389–399

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Bacterial skin diseases are a common practice problem although fortunately there are relatively few clinical diseases associated with them and relatively few bacterial species that are regular causes of disease. An example of the confusion is provided by staphylococcal (bacterial) folliculitis (pyoderma); the commonest clinical presentation is circular crusting painful dermatitis that is very easily confused with some early forms of dermatophytosis. This means that while the diseases may be recognizable to an experienced clinician, there may be some confusing overlapping clinical presentations. Whilst there are some organisms such as various staphylococcal organisms that are regarded as pathogenic, there are many commensal organisms that may in certain circumstances cause disease. The former can cause disease, which is local usually but occasionally more generalized, in the absence of any overt immune compromise. The latter often require some local or generalized immunocompromise before disease develops.

Skin damage, which may be trivial or more obvious, and compromised local or general immunity can predispose to skin infections. The clinical objectives have to be to identify the organisms involved, to establish whether they are primary or secondary and then to control and eliminate the infection. In this way the primary skin diseases might rapidly resolve if a specific treatment option was available and convenient. There may be significant benefit in controlling secondary skin infection also. For example, an old horse with known pituitary pars intermedia dysfunction (Cushing's disease) might have a serious skin infection involving a normally commensal bacterial organism or may have serious disease from a pathogenic species. Controlling the infection(s) will certainly help but treating the underlying condition should be the main objective – provided of course this can be done at all.

The various different bacteria that are involved in skin infections often have a different location within the skin. Thus *Staphylococcus* spp. usually cause folliculitis (i.e. they remain, at least at first, in the follicles) but deeper extension results in a nodular condition commonly referred to as 'botryomycosis'. The misleading name gives the impression of a fungal lesion but this simply reflects the clinical similarity to *Actinomyces* (and other fungal granulomatous) infections. *Corynebacterium pseudotuberculosis* causes a deeper pyoderma and this can extend rapidly in lymphatic vessels and so results in a condition known as ulcerative lymphangitis.

There are some very serious bacterial skin diseases that are either potentially zoonotic (including multi-resistant/methicillin-resistant *Staphylococcus aureus* (Yasuda et al 2000, Cuny et al 2006) and *Burkholderia* (*Pseudomonas/Malleomyces*) *mallei*) or are life-threatening to the horse. An example of life-threatening bacterial disease is the anaerobic clostridial infections that affect horses in several different ways. Tetanus can develop from deep infection with *C. tetani* even in an apparently trivial skin or foot injury and several gas-forming clostridial species can cause massive necrotizing damage to the skin and subcutaneous tissue. Of course these diseases also illustrate the point that the specific circumstances of a wound may dictate the species of bacteria that can survive therein. Clostridia require anaerobic conditions and some other species require either acidic or alkaline environments.

In order to treat many of the bacterial skin diseases (whether primary or secondary) it is important to know the species involved and where possible its antibiotic sensitivity profile. There are few antibiotics that are available for horses and so careful/rational use is essential. Diagnostic bacteriology may be helpful but it can also be misleading – the underlying primary disease can be immunological, for example, and the detection of potentially pathogenic bacteria on the diseased skin may not be the most important aspect of the disease. Diagnostic investigations for bacterial disease are well established but it is important that a truly representative culture is achieved and therefore a presumptive diagnosis should be made and the sampling adjusted to take account of where the bacteria might best be identified. Deep swabbing from the site of a biopsy or culture of a whole biopsy specimen may be indicated. There may be some wish to surgically prepare the site before biopsy or swabbing but this will clearly have a very significant effect

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on the outcome of any attempt to culture the whole spectrum of bacteria involved (see p. 64).

If a diagnosis of bacterial skin disease is made and treatment applied it should resolve! If it does not, there are several reasons that can be considered.

1. The delivery of the drug to the site of infection is not effective either because the drug is not administered appropriately or because the site precludes delivery.
2. The drug is wrong for the bacterial species concerned or the wrong dose or route is being used.
3. There has been a change in the bacterial species or the sensitivity of the cultured organism.
4. The diagnosis is wrong.

In spite of the theoretical benefits of antibacterial medications, many skin diseases do not respond fully or in some cases at all. If treatment fails then a full review of the case should be carried out.

Some bacterial diseases such as glanders and sporotrichosis are very serious and statutory control measures may be required to eliminate the conditions.

Key points: Abscess

1. Cutaneous and subcutaneous abscesses are a regular clinical presentation. The foot is the most common anatomic site. Sterile abscesses also occur. Some remain localized, others are less defined.
2. Focal or diffuse swelling with signs of inflammation. Size varies. Single and multiple abscesses are encountered. Characteristic early painful focus that becomes softer and then bursts with purulent discharge. Purulent discharges tend to cause skin inflammation below the abscess site.
3. Diagnosis is based on clinical examination and ultrasound examination. Aspiration can help to confirm the diagnosis and the infective organism.
4. Treatment involves establishing drainage of the abscess site. Antibiotics may be appropriate in some sites and the tetanus status of the animal should always be considered.
5. In most cutaneous sites the prognosis is good but this may be varied when the underlying cause is difficult to resolve. Multiple abscesses can occur and when they are at different stages of development different approaches may need to be taken.

Abscess

Profile

An abscess is a circumscribed collection of inflammatory debris (pus) developing from acute or chronic localized infection and/or tissue destruction. Abscesses are relatively common in horses in particular locations – the foot is possibly the commonest site. Frequently in the early stages they are accompanied by pain, heat, swelling and perilesional inflammation. Latterly the inflammation becomes liquefied and the abscess becomes fluctuant. Then the overlying skin thins and finally ruptures, discharging the inflammatory debris.

Dermal abscesses may be infected or sterile – infection is not a prerequisite for abscessation but it is the most common cause. Infected abscesses can be caused by a variety of organisms; the more common organisms involved are *Streptococcus equi*, *Staphylococcus* spp. and *Corynebacterium pseudotuberculosis*. *Streptococcus equi equi* infection (strangles) often manifests as discharging abscesses associated with lymph nodes around the head (submandibular and parotid in particular). Systemic (haematogenous) dissemination of infective emboli (septicaemia/pyaemia) is a potential component of severe infections with *Streptococcus equi equi* (bastard strangles) and *Rhodococcus equi* and cutaneous abscess can be found with both infections. Abscesses due to *Clostridium* spp. are often associated with intramuscular injections and particularly with non-antibiotic and mildly irritant drugs. Sterile abscess usually develops in response to non-infective tissue necrosis. It can also develop if infection is controlled but inflammation and inflammatory debris persist.

The infecting organism usually gains access through skin wounds but can be disseminated into the skin through the blood and/or the lymphatic system or as extensions from infected foci in adjacent subdermal tissues. Ectoparasites may be responsible for transmission of infection into the skin, for example in some cutaneous abscesses such as those due to *Corynebacterium pseudotuberculosis* in the pectoral and chest regions and widespread small abscesses due to *Histoplasma farciminosum* (epizootic lymphangitis) (Welsh 1990).

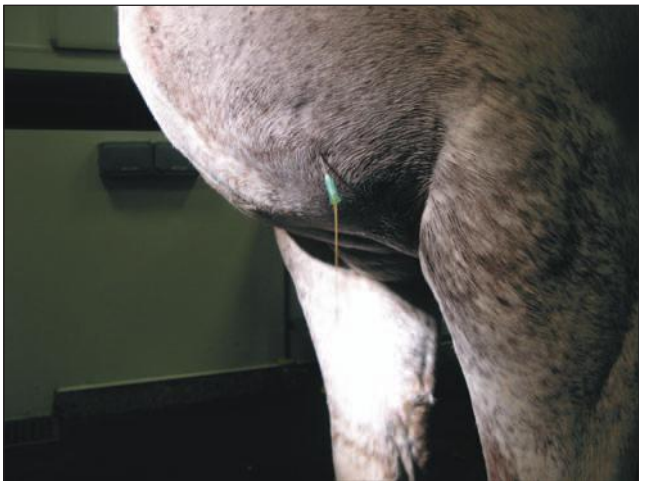


Figure 6.1 This large, hot swelling developed over some days following an intramuscular injection. Initially it was firm and painful on palpation. After several days a thinner area of skin was identified and under ultrasound guidance an incision was made into the abscess.

In immunocompromised horses (such as Cushing's disease cases) oral abscesses are particularly common from even minor infections of small abrasions.

Clinical signs

Clinical signs vary widely with location, number and cause. For example, an abscess within the hoof capsule causes extreme pain while a superficial abscess on the skin may be only painful on palpation.

There is a characteristic progression from a hard, painful nodule surrounded by an area of acute inflammation with heat and swelling (Fig. 6.1) to a softer texture with skin thinning in one site (usually). The fluid may fluctuate on palpation in the later stages.

The overlying skin becomes thinner and eventually bursts. The accumulated debris/pus is usually fluid in consistency at the stage of bursting and if in a suitable site it

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Figure 6.2 **(A)** This donkey had a large abscess on the shoulder. **(B)** The discharge caused extensive dermatitis down the skin.

may drain under gravity. In some cases there is no fluid pus and the site is made up of necrotic tissue that is far more difficult to remove.

Purulent discharges can cause significant skin damage (Fig. 6.2).

Multiple interlinking cutaneous abscessation involving relatively small individual abscesses is a feature of epizootic lymphangitis (see Fig. 7.20), glanders (see p. 161), sporotrichosis (see p. 178) and ulcerative lymphangitis (see p. 155) amongst some other conditions. The causative organisms and the epidemiological situation may help to establish the cause and the likely outcome.

Oral and facial abscesses are common in horses suffering from Cushing's disease (hyperadrenocorticism).

Clostridial abscess is characteristically associated with gas formation and has a less localized character. Pain and local swelling are often severe and there may be extensive sloughing of the skin and underlying necrotic muscle, etc. (Fig. 6.3). The affected horse is invariably very ill until the infection is controlled.

Chronic (cold) and sterile abscesses are usually well walled-off and may or may not be attached to the skin and there may be a relatively small discharging sinus tract leading to a much larger subcutaneous sterile (or infected) necrotic abscess. This circumstance quite commonly occurs with periapical abscessation of the mandibular and premolar maxillary cheek teeth. In the case of the maxillary teeth the skin sinus may be obvious over the apices of the teeth on the face. Discharging sinuses on the lower jaw may be much more difficult to identify **(Fig. CD6 • 1)**.



Figure 6.3 Clostridial infection of an injection site resulted in extensive skin necrosis and a prolonged recovery time with extensive scarring. (Figure courtesy of Mark Cubberley.)

Differential diagnosis

- Haematoma: these are soft and fluctuant from the outset and usually not painful apart from any primary injury that results in haematoma formation; the fluid nature is easy to appreciate.
- Cyst: usually not painful, inflamed or progressive, but some dermoid cysts can become secondarily infected and/or inflamed and then may develop into an abscess.
- Acute eosinophilic granuloma: solid, mildly painful and not usually progressive; they seldom soften and usually subside spontaneously.
- Hernia: the contents can be identified and most are reducible.
- Nodular neoplastic lesions, e.g. sarcoid/lymphosarcoma: tumours are usually well defined or have typical characteristics; some faster growing ones with necrotic centres and those with ulcerated surfaces can abscessate; tumours often have impaired local immunity and so secondary infections are common.

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Diagnostic confirmation

- Characteristic appearance and ultrasonographic features (fluid-filled, hypoechoic central portion with typical 'snow-storm' appearance).
- Aspiration of contents usually allows identification of the nature of the pus and the bacterial species involved.

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Treatment

Poulticing and hot compresses can be used to speed up the development of the mature abscess. There is no advantage in opening an abscess in the early (hard) stages except in the case of clostridial infection when oxygenation/aeration of affected tissues is important.

The value of antibiotics is dubious in most cases of single localized abscessation but where streptococcal infection is suspected (or proven) penicillin may be a singular help. Effective antibiotic delivery to the central necrotic part of a developing abscess is problematic. Furthermore, there is a general sentiment that early (premature) administration of appropriate drugs may markedly delay maturation of an abscess and delay final healing. In some cases early antibiosis may apparently result in exacerbation after withdrawal of the drugs. However, antibiotics can sometimes cure the condition before bursting without further complication or treatment.

Establishment of effective drainage of the mature abscess is probably the most important aspect of management, but the identity of the infecting organism will have a significant bearing on the value and choice of antibiotics whether administered systemically or locally. In many cases rapid improvement in the horse's demeanour follows surgical drainage. This is particularly noticeable in foot abscesses.

CD

The discharges from abscesses can be a potent source of infective material for other horses, either directly or via fomites and flies **(Fig. CD6 • 2A–C)**.

Following the development of an abscess, the surrounding skin may lose its hair and the superficial epidermis and the hair may slough off. This is possibly due simply to pressure and sustained local inflammation. These changes are invariably temporary but the site of the abscess may show a detectable scar (Fig. 6.4).

Purpura haemorrhagica (see p. 275) can be a sequel to prolonged abscessation (particularly that due to *Streptococcus equi equi*, *S. equi zooepidemicus*) whether or not the abscesses are satisfactorily treated and/or drained.



Figure 6.4 This horse had disseminated (metastatic) *Streptococcus equi* abscessation (bastard strangles) and developed multiple discharging cutaneous abscesses. Note the loss of hair and pigment in the area surrounding the abscess site.

Note

- The purulent discharge from most abscesses is laden with bacteria and this can be a significant source of infection for other animals and, indeed, other sites of injury on the affected horse.
- Horses suspected of having either strangles or Wyoming strangles should be isolated before and after abscesses are surgically drained. Discharging abscesses also attract flies (both surface feeders and myiasis species).
- The discharges and swabs, instruments etc., from the serious diseases such as strangles (*Streptococcus equi*), epizootic lymphangitis (*Histoplasma farciminosum*), glanders (*Burkholderia* (*Pseudomonas*/*Malleomyces*) *mallei*), sporotrichosis (*Sporothrix schenckii*) and *Corynebacterium pseudotuberculosis* must be effectively disinfected.

Cheek abscess

Profile

The specific development of single abscess or more usually multiple, small, interlinked abscesses on the side of the cheeks is a recognized entity. Acute abscess formation at or around the lateral commissure of the mouth is typical. It is possibly caused by dirty bits, dental damage to internal mucosa of the cheek or penetrating wounds due to barley or oat awns etc.

Key points: Cheek abscess

9 11 14 17 21 24

1. A specific entity affecting the cheeks as a result of either abrasion or penetrating foreign bodies from outside or within the mouth. Usually streptococcal organisms such *S. equi* var. *zooepidemicus* are involved.
2. Multiple, painful focal abscessation with small discharging sinus tracts and a generalized local painful swelling (suggestive of cellulitis) is presented. Abscessation seldom develops into a single focal mass and is more often a series of smaller linked tracts.
3. Diagnosis is based on clinical grounds and on culture from the discharging purulent material.
4. Treatment with antibiotics and irrigation is indicated. Antibiotic selection will depend on culture results. Most are streptococcal, however, and penicillin is usually a reasonable first choice approach. The cause must be addressed if it can be identified.
5. The prognosis is good but some local deformity, alopecia and loss of pigmentation may occur.

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Clinical signs

Acute, painful, hot swelling of the commissure of the mouth or adjacent cheek area is typically encountered. Multiple small interlinking sinus tracts with small areas of exudation over pointing abscess can be seen (Fig. 6.5).

Hair loss (alopecia), pigmentary changes and scarring of the skin overlying the abscess are common. Although oral lesions heal well for the most part, chronic ulceration at the site of cheek abscesses is also common. A few cases are slow to heal and then foreign bodies or repeated traumatic insults should be suspected. A careful examination of all tack and a dental examination must be performed.

Note

Oral abscessation (possibly involving the cheeks) is a common secondary feature of pituitary pars intermedia dysfunction (PPID/Cushing's disease/ pituitary-based hyperadrenocorticism) in horses over 15 years of age. Unless this is addressed, the affected area may not heal at all.

Differential diagnosis

- Bit injury in the mouth: usually this is focal and has an obvious cause.
- Buccal laceration from teeth inside the commissure: multiple buccal and possibly lingual ulceration usually present.
- Foreign bodies: these are difficult to identify.
- Tumours (ulcerated melanoma, squamous cell carcinoma or sarcoid in particular): usually these are firmer and better defined and are seldom patently infected.

Diagnostic confirmation

- Cuts or wounds inside the mouth adjacent to the abscess.
- Typical clinical features of heat, pain and swelling with an interconnected multifocal inflammatory nature.
- Culture of discharges and swabs taken from within the lesions usually identify *Streptococcus* spp. bacteria.
- Ultrasonography can help to differentiate this from tumours and other inflammatory and non-inflammatory disorders.



Figure 6.5 Cheek abscess. Note hair loss overlying the abscess.

Treatment

Further damage or irritation in the area should be avoided and where possible any causative factor (tack/bit or dental conditions) should be addressed. Working horses may be amenable to use of a bitless bridle, at least in the short term to allow the area to heal.

Alternating hot and cold fomentation will hasten development of the abscess, but it may be impossible to apply these principles to the lesion if the area is very painful and handling is resented. Sedation and careful drainage can be helpful.

Surgical lancing and drainage when the abscesses 'point' sufficiently is the treatment of choice. Early use of antibiotics should probably be avoided. Once the condition is in a suitable state (or if the inflammation is having a systemic effect on the horse), antibiotics should be administered. Penicillin is usually the first choice but the culture results may have a bearing on the final choice. Once healing starts it usually progresses rapidly.

Bacterial folliculitis and furunculosis (acne/ superficial pyoderma)

Profile

Folliculitis is inflammation of the hair follicle with accumulation of inflammatory cells within the follicle lumen. Most commonly it is caused by bacteria involving coagulase-positive *Staphylococcus* spp. (*S. aureus*, *S. intermedius* and *S. hyicus*) (Chiers et al 2003) and *Streptococcus* species are isolated. Multi-(methicillin)-resistant *Staphylococcus aureus* (MRSA) has recently been identified from healthy horses

Key points: Bacterial folliculitis and furunculosis

11 15 16 17 22 23 24

1. A relatively common occurrence – often with *Staphylococcus* spp. or *Streptococcus* spp. bacteria. Most cases appear to relate to hygiene or cutaneous immunity problems. Exposed, hairless (glabrous) skin surfaces are most often involved, e.g. the sheath/groin and the sides of the face, but the saddle region and body skin can be seriously infected.
2. A painful localized region in which papules can be 'felt' in the skin that develops into an inflamed, exudative and oedematous area is typical; these lesions are often circular and exudative in the early stages. Some develop into isolated individual pustules while others coalesce into abscesses or furunculosis.
3. Diagnosis is usually simple but the changes are not pathognomonic for any particular bacterial species; early lesions can easily be confused with various forms of dermatophytosis. Pain usually indicates *Staphylococcus* spp. bacteria but other species may be involved.
4. Treatment is sometimes problematic and recurrences are common. Local warm washes with antiseptic shampoos such as chlorhexidine and povidone-iodine, systemic antibiotics and oral potassium iodide are all helpful. Skin and stable hygiene are important. Treatment courses of several months may be required.
5. The prognosis is fair but some cases take a long time to resolve and the healing lesions are sometimes alopecic and/ or have permanently altered hair colour and quality.

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(Yasuda et al 2000) and whilst the significance of this to horses has yet to be established it is becoming more of a concern to veterinarians.

CD

As the infective process proceeds, degeneration of the hair follicle leads to infection of the surrounding dermis and subcutis. This is called furunculosis. The condition is usually related to areas of rubbing due to dirty or ill-fitting harness, rugs or saddle cloths. Most cases occur in late spring or early summer coincidental with hair shedding, humidity and increasing work with dirty or poorly maintained tack. Poorly groomed horses have a higher tendency to develop the condition. Horses in poor condition, or immunocompromised animals or those on medications likely to be immunosuppressive also seem more likely to be affected (Inokuma et al 2003).

Dermatophilus congolensis is a common cause of bacterial dermatitis and folliculitis and is considered in its own right (see p. 156). More rarely *Corynebacterium pseudotuberculosis* is responsible and this too is considered in its own section (see p. 155). Fungi including dermatophytosis and cutaneous mycetoma/fungal granuloma can also be involved (see p. 183).

Clinical signs

CD

Usually a rapid development of small (2–5 mm diameter), painful papules is reported – in the early stages these may be more easily felt than seen unless the hair is closely clipped (Fig. 6.6). The first sign may be the presence of roughly circular areas (2–6 cm diameter) of erect hairs with a small matted crust around the base.

The so-called truncal form is restricted to the body trunk and is commonly called Canadian horse pox (a considerable misnomer), contagious pustular dermatitis or contagious acne, and is associated with poor tack hygiene in early spring in particular. This form may represent a deeper penetration of the *Staphylococcus* spp. infection and is very similar to infection with *C. pseudotuberculosis*, the cause of ulcerative lymphangitis on the limbs and neck.

Palpation is usually resented and pain is usually marked. The papules develop an ulcerated central area with extensive local oedema and exudation (Fig. 6.7).

Where multiple infections coalesce locally, it becomes a carbuncle or boil; these occur in horses with saddle rash/scab (*Staphylococcus aureus*, *S. intermedius*), heat rash, cheek abscess (see Fig. 6.5) and 'pigeon-breast' (Wyoming strangles due to *Corynebacterium pseudotuberculosis*).

More diffuse coalescing folliculitis leads to a furunculosis and focal areas of necrosis, especially on the distal limbs. This may be a result of an inappropriate response to bacterial toxins or a hypersensitivity response **(Fig. CD6 • 3A–C)**.

There may be evidence of enlarged cutaneous veins and lymphatics leading from the affected sites (Fig. 6.8).

There is seldom pruritus and in uncomplicated cases there are no systemic signs of illness.

Healing lesions become flat and alopecic and may develop leukoderma and leukotrichia (white saddle or girth marks may arise from this as well as other causes including persistent trauma).

Some cases of dermatophilosis are indistinguishable from this condition and indeed could be grouped in the same category of folliculitis. This is particularly so for the common *Dermatophilus congolensis* infection that involves the pastern and cannon regions (mud fever) and the back (rain scald) (see also p. 156).

Some superficial lesions fail to heal and these may become deeply infected (furunculosis) with sinus tracts and discharging sinuses. Chronic granulation tissue diffusely infiltrated with microabscesses and sinus tracts is often classified as botryomycosis (pseudomycetoma) on the skin (see Fig. 6.10), or as scirrhous cord at the site of non-healing castration wounds **(Fig. CD6 • 4A and B)**.

A staphylococcal infection of the palmar/plantar pastern region (see p. 153) is a recognized entity in the pastern



Figure 6.6 Bacterial folliculitis due to *Staphylococcus intermedius* infection.





Figure 6.7 (A, B) This 9-year-old Shire-cross gelding developed a very painful folliculitis in the sheath area. There was a great deal of inflammatory swelling and severe resentment to palpation.

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dermatitis complex (see p. 471). Although this condition is caused by the same group of bacteria it is a recognizably different syndrome – usually poor hygiene is not the main instigation but skin maceration due to continued wetting and a primary superficial infection may be involved.

Differential diagnosis

- Dermatophilosis: this can be difficult to differentiate without bacteriological cultures and skin biopsies but it usually affects the dorsum of the back and the lower limb regions.
- Other bacterial pyoderma (e.g. *Streptococcus equi equi* – strangles).
- Fungal folliculitis: these are usually much more long-standing, slower progressing and isolated and are seldom painful; fungal organisms can be identified in skin biopsies.
- Dermatophytosis: this seldom shows significant pain and exudate but secondarily infected dermatophytosis lesions can be similar; the fungal organisms can be identified in skin scrapings and hair plucks.
- Onchocerciasis: this condition induces a more diffuse change in most cases and is seasonal and regionally restricted; the microfilariae or adult parasites can usually be identified directly or in biopsies; these lesions can be secondarily infected as well.
- Generalized granulomatous disease (enteritis/sarcoidosis) multi-organ involvement.
- Pemphigus foliaceus: generalized focal or diffuse junctional exfoliative disorder.



Figure 6.8 This was one of several young Thoroughbred horses in a racing yard that, over a period of some weeks, developed very painful cutaneous inflammatory foci associated with *Staphylococcus aureus* infection. Note the prominent lymphatics leading away from the sites. Careful attention to hygiene, warm water antibacterial washes, and a prolonged course of potassium iodide orally gradually resolved the problem. (Figure courtesy of Chris Riggs.)

- Greasy heel syndrome: complex of conditions resulting in a chronic verrucose thickening of the pastern with pseudo-tumour formation and granulation, infection and ulceration.

Diagnostic confirmation

- Clinical features and pain are usually characteristic but mixed infections are common.
- Biopsy is usually avoided if possible but a fine biopsy punch (3 mm) may be useful, particularly for culture.
- Bacteriological culture and sensitivity are important but deep biopsy may be needed for significant culture **(Fig.CD6 • 5A and B)**.

CD

Treatment

Three aspects must be considered during the management of a case of pyoderma. These are:

1 *Control of the specific lesions on the affected horse.* The affected areas should be clipped and washed with warm water and an antiseptic such as chlorhexidine or povidone-iodine surgical scrub solution. This should be left on the skin for some 5–10 minutes before being rinsed off thoroughly, again with warm water. Washes are useful also for controlling the spread of the organisms responsible **(Fig. CD6 • 5C and D)**.

CD

Proprietary antiseptic shampoos are available (usually containing chlorhexidine, povidone-iodine or hexachlorophene) but human equivalents should probably not be used.

Open ulcerated pustules and abscesses should be managed carefully and hydrosurgical debridement is an effective mechanism for the removal of much of the bacterial content of a focus of infection. Clearly this would only apply to limited areas.

Parenteral antibiotics are useful adjunctive therapy. Culture and sensitivity testing should always dictate the selection but the most common antibacterial used is potentiated sulphonamide because it is usefully effective and can be maintained for weeks if necessary by oral dosing for up to 2 weeks (White 1988).

Many isolates are resistant to parenteral penicillin but where they are shown to be sensitive this can be useful, at least initially. Enrofloxacine can be effective also but should not be used in horses under 2 years of age. Although vancomycin and other potent drugs in that class would probably be effective (Orsini et al 2005), their use represents irresponsible antibiotic use except in absolutely exceptional circumstances.

Note

Donkeys and mules are known to eliminate the drugs faster than horses so either the dose interval or the dose itself may need to be adjusted.

1 Topical dressing with silver sulfadiazine or where feasible silver wound dressings should always be considered. Modern adhesive dressings such as

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adhesive hydrocolloid (DuoDerm, Convatec, UK) are very useful, even on the body trunk, and can be combined with hydrofibre silver dressings (Aquacel Ag, Convatec, UK) to provide an excellent local antibacterial effect.

2 *Prevention of further outbreaks on the affected animal*. This usually involves significant improvements in the overall hygiene and the health status of the horse. However, even in the best management systems with the healthiest horses, recurrences are to be expected. Turning out into sunshine (but avoidance of wetting by rain) is helpful and where this cannot be done, ultraviolet and infrared lamps can be helpful. Continued washing is to be avoided as far as possible but may be unavoidable. Recurrences are nevertheless common.

3 *Control of the environmental contamination and prevention of spread to other horses in contact*. All tack and rug contact with the affected skin should be prevented. Washing and sterilizing of all tack, grooming equipment, rugs, etc. is strongly advised Tertiary amine disinfectants have strong antibacterial properties without much risk of damage to tack or other equipment. Fumigation of all tack with formaldehyde gas is often the best approach to sterilization of the equipment but is more difficult (see p. 86).

Severe outbreaks in stables have been controlled with autogenous vaccines but reliance on this is not advisable. However, in the investigation of a widespread problem in a stable yard, hygiene is imperative. Regular cultures and if possible genetic studies on the organisms involved may allow the identification of a carrier animal but even then hygiene is the critical issue. Stable managers should always address hygiene as a priority – individualizing and regular sterilization of tack, grooming equipment, water and feeding bowels and buckets and in particular rugs and harness are important aspects of control.

Surgical procedures such as castration, prosthetic laryngoplasty, check ligament desmotomy in stables that are known to have a major *Staphylococcus* spp. problem must be considered very carefully. In the event that MRSA is identified in any stable, the local environmental health office should be advised. The human implications are considerable but it is not yet clear how much human infection derives from horses and how much equine infection is related to carrier humans.

Bacterial granuloma (botryomycosis/staphylococcal pseudomycetoma/deep pyoderma)

Profile

This is a bacterial pyogranulomatous disorder most often associated with skin injury such as laceration, puncture or surgery. Most lesions occur on the limb or lip/chin regions. The clinical appearance is due to the formation of miliary interlinking microabscesses from *Staphylococcus* spp. infection. In spite of the names botryomycosis and pseudomycetoma, there is no evidence of a fungal aetiology.

Clinical signs

A slow or non-healing wound with induration of the wound margins (Fig. 6.9) is typical. A chronic purulent discharge from one or more sinuses within the wound site is common.

Exuberant granulation tissue forming rosettes of complex granulation tissue and multiple microabscesses with sinuses may develop (hence the derivation *botryo-* meaning grape-like) (Fig. 6.10). Small yellow-white granules/grains may be visible in the purulent discharge. Tracking along lymphatic vessels sometimes occurs. It can reach large (or even enormous) proportions. The condition is seldom painful or pruritic. Affected horses do not often show systemic illness, but very large lesions can be debilitating.

This is a possible cause for the development of a chronic non-healing wound at the site of open castration. Usually a thickened spermatic cord can be felt and imaged

Key points: Bacterial granuloma

9 11 14/15 17

1 This is a bacterial infection usually involving staphylococcal microabscessation. The concurrent chronic inflammation and (unhealthy) granulation tissue combine to produce exuberant masses. A specific form of this condition occurs as a non-healing, fistulated, discharging castration site when the enlarging mass forms at the end of the spermatic cord (a so-called champignon or mushroom).
2 Clinically relatively easy to recognize but bacterial causes can vary.
3 Biopsy is usually simple because the tissue has no blood supply – swabs should be taken from the base of the biopsy site rather than from the lesion surface.
4 Treatment involves removal of all infected tissue and restoration of the wound site to a surgical wound. Silver dressings are helpful in controlling the infection. Prolonged antibiotics are required. Oral potassium iodide is also helpful. (**Note**: Potassium or sodium iodide should not be given to pregnant mares under any circumstances.)
5 The prognosis depends on the feasibility and efficiency of removal of the infected tissues and then the degree to which repeated infection can be controlled. Many cases require more than one surgical procedure.



Figure 6.9 Bacterial granuloma (botryomycosis/staphylococcal pseudomycetoma) showing the typical non-healing indurated margins. The lesion showed an intermittent slight purulent discharge.

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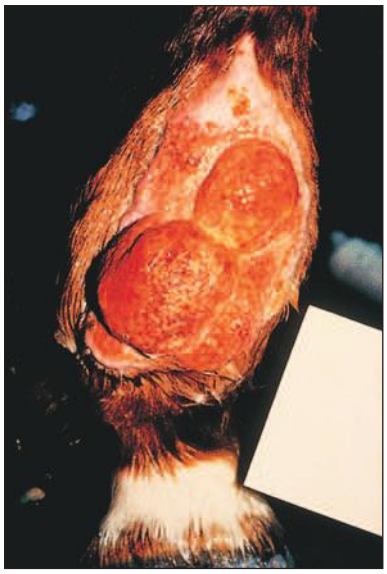


Figure 6.10 Bacterial granuloma (botryomycosis/staphylococcal pseudomycetoma) with multiple rosettes of pyogranuloma. *Staphylococcus aureus* was isolated from the multiple tracts and microabscesses which were found when the lesion was surgically removed.

ultrasonographically. This is the so-called 'champignon' (mushroom) condition **(see Fig. CD6 • 4A and B)**.

In a few cases the infection may become much more extensive, especially if the animal is immunocompromised in some way. Where this occurs the infection may extend to the local lymph node where gross enlargement can occur. This form is commonest in ponies with pituitary pars intermedia dysfunction (equine Cushing's disease) and the introduction of the infection usually requires some skin damage such as pruritus from insect bite hypersensitivity (Fig. 6.11). An accurate history of the horse and a thorough clinical examination are essential aspects of the case investigation.

Differential diagnosis

- Exuberant granulation tissue: from any other cause including foreign body reactions and haematoma/seroma formation.
- Fibroblastic (and mixed) sarcoid: sarcoid at distal limb sites can be *very* similar; usually the growth rate and appearance are typical and most cases have other sarcoid lesions; biopsy is diagnostic.
- Foreign body/sequestrum resulting in a sinus tract and chronic inflammation: these can be similar but



Figure 6.11 This 20-year-old Shetland pony suffered from insect bite hypersensitivity (sweet itch) and pituitary pars intermedia dysfunction (equine Cushing's disease). It developed an extreme form of deep pyoderma (botryomycosis) in which large subcutaneous abscesses and multiple furunculosis were present over the buttock region **(A–C)**. The lesions had a purulent exudate **(D)**. The prefemoral, inguinal and iliac lymph nodes were grossly enlarged and had staphylococcal abscessation. The same *S. aureus* organism was identified in pure culture from all sampled sites throughout. (Figures courtesy of Jane Lawson MRCVS.)

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are usually characterized by more discharge and less granulation.

- *Habronema musca* infestation of a wound: these tend to be flat but on the penile glans a proliferative lesion can develop.
- Bacterial fibrogranuloma/abscess: this is similar but involves other species of bacteria and is usually smaller and less exophytic.
- Pythiosis/deep fungal infections/fungal granuloma: geographically restricted and typical culture and biopsy results are pathognomonic.
- Squamous cell carcinoma (of the face): this tends to be more destructive and staphylococcal pyogranulomas are rare on the face.

Diagnostic confirmation

- History of injury or surgical procedures and subsequent failure of healing without response to normal therapeutic procedures.
- Histopathology is characteristic.
- Culture from deep tissue biopsy samples will establish the bacterial species involved and its antibiotic sensitivity.

Note

Extra care should be taken to obtain truly representative bacterial culture so sampling from the deeper parts of the lesions may be more meaningful than superficial surface swabs.

Biopsy from biopsy specimens or from the biopsy wound can be helpful. Modern PCR methods and staining can sometimes identify the species of bacteria involved.

Treatment

Careful surgical excision with debridement of all affected tissue is always the preferred option. Repeated procedures may be required to establish a normal healing wound bed. Concurrent prolonged antibiotic treatments depending on in-vitro sensitivity results are usually used. Trimethoprim sulphur combinations administered orally at 30–35 mg/kg total substance once or preferably twice daily for 30 days is the usual approach. In adult horses (those with inactive growth plates) enrofloxacine (at 7.5 mg/kg orally once daily) can be used in the event that the bacteria are not sensitive to the trimethoprim sulphur compounds. Oral potassium iodide (10 g daily for a 500 kg horse) or prolonged courses of antibiotics can provide useful assistance but cannot be relied upon solely.

Other possible reasons for a non-healing wound must be eliminated and if indicated these must be addressed.

Relapses or recurrences following sufficiently aggressive initial treatment are unusual. Consideration should be given to early grafting of the resulting granulating wound to encourage rapid wound closure.

Staphylococcal dermatitis

Profile

Several species of coagulase-positive *Staphylococcus* bacteria cause skin disease in horses. *Staphylococcus aureus* and *S. intermedius* are the most frequent isolates from lesions (Pascoe 1989). *S. hyicus* has been isolated from some 'greasy heel'-type lesions of the pastern and coronet. The role of MRSA in horses is not yet clear but several cases have been reported.

Key points: Staphylococcal dermatitis

9 11 15 17 19 22 23 24

1. Coagulase-positive staphylococci produce several syndromes, including saddle rash, folliculitis and pastern folliculitis.
2. The range of clinical conditions is wide; signs are usually pain and localized exudative dermatitis. Focal lesions such as pyogranulomas and abscesses also occur.
3. Diagnosis is based on the clinical features and cultural identification of the coagulase-positive staphylococcal organisms.
4. Treatment is always problematic. Clipping of overlying hair and antiseptic washes with warm water are indicated in all cases of diffuse dermatitis. Abscesses are drained and flushed. Systemic antibiotics can be helpful if based on sensitivity profiles.
5. The prognosis is guarded in all cases. Recurrences are common but a significant proportion of cases do resolve completely – often leaving some scarring and some changes in hair coat density and colour.

These bacteria produce a series of damaging endotoxins which are possibly the main factor in the skin damage, either directly due to dermonecrotoxins or due to immunological responses/hypersensitivity reactions. They cause several well-recognized inflammatory skin diseases including saddle rash, staphylococcal pyoderma /folliculitis (tail pyoderma, mucocutaneous pyoderma, acne), furunculosis, pastern folliculitis, pyoderma, cellulitis/lymphangitis complex, abscess and wound infections.

The conditions are contagious and are characterized by a highly painful dermatitis with acute folliculitis, furunculosis and crust formation. The lesions are commonest in areas of close tack or harness contact such as the back and the face. Most cases occur in spring and early summer, possibly related to hair shedding, higher ambient temperatures and the culmination of a winter of tack and skin neglect.

Clinical signs

1. *Staphylococcal pyoderma* (tail pyoderma/mucocutaneous pyoderma/acne; botryomycosis/pseudomycetoma). In most of these circumstances the changes are secondary to some form of trauma – however localized or minor. Focal or acute localized painful inflammatory foci are recognized at first. Provided that the infection remains localized to hair follicles the condition is termed folliculitis but once it breaks out from this into the surrounding dermis and subcutis, it becomes a furunculosis (Fig. 6.12). Once this affects the fascial planes and lymphatic vessels the terms cellulitis/lymphangitis respectively can be applied. Tail pyoderma shows painful pustules and larger exudative areas while the mucocutaneous condition shows extensive, painful cracking of the lip margins. Acne is a term given to the multiple focal pustular lesions that develop in the skin. These start as small tufted papules – often with only the hair direction indicating any change. In some circumstances the affected area remains localized, producing a staphylococcal microabscessing granulating nodule.
2. *Saddle rash*. This is a rapidly developing, localized, inflammatory, purulent dermatitis associated with severe pain rather than pruritus. It probably represents a particular form of furunculosis/cellulitis. Lesions are principally associated with harness areas and saddle cloths and most

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Figure 6.12 Furunculosis over the breast area of a young horse. A pure culture of *S. aureus* was obtained. Note the generalized areas of inflammation with small discharging tracts.



Figure 6.13 Staphylococcal dermatitis ('saddle rash') due to *Staphylococcus aureus* infection. The affected area of the saddle was extremely painful to the touch.

commonly affect the skin of the back, saddle (Fig. 6.13), loins and chest and occasionally the distal limb regions. Examination of the affected area is often markedly resented with avoiding behaviour (ducking, moving away or kicking/biting). Small (1–2 mm) painful lesions rapidly coalesce to produce large areas of exudative dermatitis with surrounding oedema (Fig. 6.14). Surface scabs and hair matting develop but little pus is present.

3 *Pastern folliculitis*. See page 153.

4 *Wound infections*. Wound infection with *Staphylococcus* spp. bacteria is common in horses. The role of MRSA is uncertain. Isolation of *S. aureus* from wounds requires immediate and appropriate antibiotic therapy, so culture is important.

5 *Staphylococcal abscess*. Localization of the infection with a hot, painful, central inflamed area is usually consistent with a developing abscess. Staphylococcal abscessation is painful initially but gradually the central portion softens and eventually bursts spontaneously, discharging a large volume of pus **(Fig. CD6 • 6)**.

6 *Impetigo*. This term is used in human medicine and is seldom applicable to horses. The condition is described as a staphylococcal infection of the exposed skin and the initial bullous state possibly arises from an immunological response to the toxins produced by the *Staphylococcus* spp. involved. Thus the clinical signs can be very different and there may be no identifiable staphylococcal organisms to culture or to stain in a smear. Usually the exudative and purulent stage follows the pruritic/immunological stage. In effect it is not significantly different clinically from the folliculitis/furunculosis syndromes described above.



Figure 6.14 This area of acutely painful dermatitis developed under the blankets over some weeks. The horse could not be examined without heavy sedation. Culture revealed a pure growth of *S. hyicus*. (Figure courtesy of Ian Camm.)

CD

Differential diagnosis

- Streptococcal folliculitis/*S. equi* abscessation: this can be very similar but tends to be less painful; culture and direct smears are required to differentiate the two conditions.
- Dermatophilosis: tends to be less painful and spontaneous resolution occurs if the regions can be kept dry; typical culture and direct smears.
- Dermatophytosis (*Trichophyton* spp.): usually non-painful, mildly pruritic and with characteristic epidemiology and direct and cultural microbiology.
- *Corynebacterium pseudotuberculosis* infection: this is rare and produces large abscesses; culture and direct smears are diagnostic.
- Histoplasmosis/epizootic lymphangitis (*H. farciminosum*): geographically restricted to parts of Saharan Africa; characteristic mycology.
- Chemical/medicant (over-strength or inappropriate) irritation or burns: history is usually clear but

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secondary infection can be a significant secondary problem.
- Other causes of 'greasy heel' syndrome (see p. 471).

Diagnostic confirmation

- History of unhygienic conditions, dirty tack and dusty stables.
- Typical distribution of lesions.
- Pain is often marked and exudation is usually less prominent. Matting of the hair is typical.
- Direct smear and culture from fresh lesions will identify the causative organism.

Treatment

Close clipping and thorough cleaning of affected area with chlorhexidine (or povidone-iodine) soapy solutions are required. However, these procedures may be very painful and require sedation and analgesia.

In some pastern folliculitis and some saddle rash cases, the crusts may be very adherent and then soaking the site with a hydrogel for 24–36 hours (if possible under dressing) can allow the clipping to take place more easily. Silver hydrofibre dressings (Aquacel Ag, Convatec, UK) can be helpful but in many cases the affected areas are not amenable to dressing application.

Washing with warm water and chlorhexidine, povidone-iodine or a tertiary amine antiseptic (surgical scrub) solution is probably sensible at an early stage.

Lancing and draining of defined abscesses is the treatment of choice. The abscesses can be flushed out repeatedly with low pressure saline irrigation.

Systemic or topical antibiotics have little effect. However, penicillin and trimethoprim sulphur drugs can be useful in some cases. Enrofloxacine and ceftiofur may be used. However, multiple resistance is common and culture is critical if topical and systemic antibiotics are to be used. Silver dressings are possibly one of the most effective ways of treating the conditions where application is practical. Topical silver sulfadiazine can be useful too but the material can sometimes mat the area so that exudate is not removed.

Sunshine or an ultraviolet lamp can be helpful for the saddle rash complex.

Thorough cleansing of all equipment and rugs, etc. is advisable.

Autogenous vaccines may be useful in theory at least if many horses are involved simultaneously but these have not been universally accepted as being effective.

Control

All affected cases should be isolated and all direct and indirect contact avoided including (grooming equipment and harness, etc.). All equipment should be sterilized repeatedly by washing in a tertiary amine disinfectant or fumigation with formaldehyde gas (see p. 86).

Streptococcal dermatitis/folliculitis

Profile

Streptococcal skin infections are a common event in horses throughout the world – they are possibly the commonest secondary infection of wounds and other skin damage. It is important to remember too that *Streptococcus equi* (var. *equi* and var. *zooepidemicus*) are common respiratory pathogens (the cause of strangles and its variants). *Streptococcus equi* var. *equi* is an obligate pathogen and does not have a commensal state whereas *Streptococcus equi* var. *zooepidemicus* is an opportunistic pathogen. Other cases may be caused by *Streptococcus equi zooepidemicus* or other streptococcal species including *S. equisimilis*.

There are numerous clinical presentations. The typical lesions are folliculitis, furunculosis and ulcerative lymphangitis; often diffuse cellulitis may be present in the surrounding tissues. All streptococcal organisms are highly contagious.

A hypersensitivity state has also been described as a possible cause of recurrent urticaria and most forms of purpura haemorrhagica (see p. 275) are probably associated with streptococcal antigen–antibody complexes.

Key points: Streptococcal dermatitis/folliculitis

9 11 14/15 17 21 23 24

1. Streptococcal bacteria are a common cause of primary and secondary skin infections as well as a common cause of subcutaneous abscessation. *S. equi* var. *equi* is an obligate pathogen while *S. equi* var. *zooepidemicus* is an opportunistic commensal pathogen.
2. Mildly to moderately painful skin infections and abscesses are characteristic. Streptococcal folliculitis is a common superficial skin infection while *S. equi* var. *equi* is a common cause of disseminated abscessation (bastard strangles).
3. Diagnosis is based on culture and typical epidemiological and clinical features.
4. All streptococci are at least partially sensitive to penicillin and so this is the first choice antibiotic. Drainage of abscesses is important where possible. Superficial infections can be resolved by using topical antibacterial washes with systemic antibiotics.
5. The prognosis is generally good apart from the few cases of disseminated infection with multiple internal (and possibly cutaneous) abscessation.

Clinical signs

Small, painful follicular infections around mouth (Fig. 6.15), vulva and in wounds are the defined non-strangles streptococcal infection.

Localized skin abscesses resulting from pyogenic infection of lymph nodes in the submandibular and parotid regions are typical of strangles. Disseminated infection can result in cutaneous abscesses at any site including the limbs and body trunk (as well as internally) Most of these abscesses are very painful and very inflamed, at least in the early stages (Fig. 6.16). Loss of hair and serum exudation may precede rupture of the abscess.

Differential diagnosis

- Staphylococcal folliculitis: this can be very similar but is generally more painful; bacterial culture is required to differentiate the two conditions.
- Dermatophilosis: largely diffuse dermatitis without obvious folliculitis/abscessation; exudate tends to be less purulent and less in quantity; pain is significantly less in most cases.

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- Cellulitis: diffuse pyogenic infection of connective tissue; can be similar or identical and staphylococcal infection may be involved at the same time.
- *Corynebacterium pseudotuberculosis* infection: focal abscessation or cording lymphatic nodular dermatitis are features.
- Glanders: severe debilitating disorder with characteristic geographical distribution and defined microbiology.

Diagnostic confirmation

- Needle-aspirate-derived smear shows Gram-positive cocci. (Note that direct impression smears may be misleading with mixed commensal and opportunistic pathogens being detected.)
- Culture for *Streptococcus* spp. is characteristic.



Figure 6.15 Streptococcal folliculitis (*Streptococcus equi* var. *equi*). The small pustular lesions were painful and there was little or no exudate.



Figure 6.16 Streptococcal septicaemia (*Streptococcus equi* var. *equi*) was isolated in pure culture from this purulent material which drained from a small abscess associated with localized lymphangitis.

Treatment

The affected area should always be clipped out and cleaned with chlorhexidine (or povidone-iodine or tertiary amine) antiseptic (surgical) scrub solution. Topical antibiotics and antiseptics may be effective in localized areas such as the mouth/face.

Defined abscesses should be lanced and drained when mature (i.e. when overlying skin is thin and contents are fluid). All affected horses should be isolated until the infection has totally resolved.

All streptococci are either sensitive or partially sensitive to penicillin. Therefore this is the antibiotic of choice. The problem is in delivering a suitable concentration to the site of the infection. In the case of strangles, the value of prolonged treatment with intramuscular penicillin is controversial.

Note

Horses may develop an immune-mediated vasculitis and purpura haemorrhagica (see p. 275) or immune-mediated thrombocytopenic purpura or haemolytic anaemia some weeks later (particularly if challenged by a second infection cycle) (Scott 1988, Rose & Hodgson 1993). The secondary effects can be extremely serious and sometimes cannot be differentiated from drug eruptions and reactions.

Dermatological signs are prominent in all these conditions.

Pastern folliculitis

Profile

This is an uncommon bacterial folliculitis that affects one or more of the pasterns and coronary bands and is caused by one or more of the following bacteria: *Staphylococcus aureus,*

Key points: Pastern folliculitis

5 11 14/15 17 22 23 24

1. An uncommon staphylococcal infection of the palmar/plantar pastern regions of one or more limbs. White legs are more often affected.
2. Initial papules and pustules rapidly coalesce into a wider painful inflammatory dermatitis with moderate exudate; scabs incorporate hair and become difficult to dislodge. The condition seldom results in systemic signs but localized oedema and lameness may be present.
3. Diagnosis depends on swabs taken from the surface of untreated areas and from squeezed skin exudate. The condition can be painful so regional anaesthesia may be helpful.
4. Treatment involves close clipping and antibacterial washes with warm water and warm water rinses. The region should be blotted dry, not rubbed, and the area kept dry thereafter. Silver dressings are very helpful in refractory cases. Strong or repeated antiseptic washes in cold water are not helpful.
5. The prognosis is good in most cases but some horses are left with skin fragility and a tendency to recurrence. This may reflect an inherent susceptibility to the condition.

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S. hyicus and possibly *S. intermedius*. Usually there is little evidence of involvement of other regions. The pathogenesis is unknown but some degree of local hypersensitivity is possibly involved. Individual horses may be liable to the disease and white-skinned limbs are more usually involved than coloured legs although the reason for this is far from clear.

The disease is a component of the pastern dermatitis/'greasy heel' syndrome (see p. 471).

Clinical signs

The condition is, at least initially, limited to posterior of pastern and bulbs of the heels on one or more (white-skinned) limbs (Fig. 6.17).

Very early cases consist of papules, which coalesce and produce large areas of ulceration and suppuration. Matting hair and oedema of the skin produce a swollen, painful exudative limb. Oedema may extend up the limb and deep spread of the infection can cause extensive cellulitis (diffuse, pyogenic infection of the connective tissues) with gross, painful swelling of the whole limb. The horse may exhibit lameness but systemic effects are usually absent.

Differential diagnosis

- Other forms of 'greasy heel' including:
  - Dermatophilosis: seldom only involves the palmar/plantar pastern and is less painful; typical bacteria can be found but the two conditions commonly coexist.
  - Mycetoma: deep dermal mycosis; more serious and extensive staphylococcal dermatitis really representing an advanced stage of this condition.

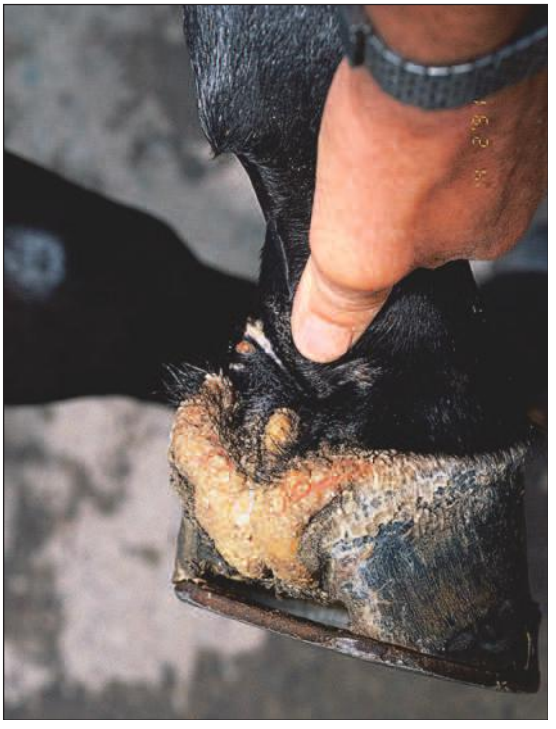


Figure 6.17 Pastern folliculitis. A mixed culture of *Staphylococcus aureus* and *S. hyicus* was obtained from swabs taken from biopsy specimens.

- *Fusobacterium* spp. cannon dermatitis: an unusual condition encountered on the cannon regions of racehorses; very painful disorder with a characteristic fetid odour.
- Coronary band dystrophy/vitamin A deficiency: this is restricted to coronary band.
- Eosinophilic coronitis–stomatitis syndrome: restricted to the coronary band, the margins of the ergots and chestnuts and the mouth initially at least; florid inflammatory changes with ulceration and secondary infection.
- Vasculitis (immune mediated): usually more generalized, with necrotizing circular or coalescent cutaneous lesions; purpura haemorrhagica is an example.
- Pastern and cannon leukocytoclastic vasculitis (photoactivated): this affects the lateral aspects of white hind limbs more than others and is usually secondarily infected; biopsy is characteristic.
- Photosensitization: this is a characteristic exaggerated inflammation resulting from primary photosensitizing plants/chemical ingestion or from hepatic disease; it affects all exposed white-skinned areas.
- Pemphigus foliaceus: this involves widespread skin lesions – often extensive and numerous; there is a characteristic histological finding.
- Chorioptic mange, with self-trauma: the parasites are easily found with skin brushings.
- Contact/irritant dermatitis: urine, unhygienic stable bedding and yards, chemical contacts; the history and clinical findings are characteristic; coloured limbs are equally affected.

Diagnostic confirmation

- Typical clinical appearance with significant pain and limited exudate creating adherent crusting (scab).
- Smears or culture of organism are usually definitive but often produce mixed cultures of facultative pathogens and commensals.

Treatment

Effective treatment is usually a painful process – sedatives and analgesia may be needed in severe cases involving more than one limb. A single limb may be treated using a regional nerve block. General anaesthesia may be worthwhile if the condition is extensive, very painful or involves more than one limb.

The affected areas must be close-clipped, cleaned and scrubbed with chlorhexidine or povidone-iodine antiseptic soaps. It is very important that these washes are done with warm water and that following a period of about 10 minutes the chemicals are washed off carefully with warm water. Repeated soaking of the skin is often contraindicated as skin maceration simply adds to the complications. The latest forms of silver-impregnated hydrofibre dressings (Aquacel Ag, Convatec, UK) can be applied directly to the clipped and cleaned area in a moist or dry state under a soft absorbent bandage. The dressings are best left undisturbed for the first 48 hours at least. Silver is known to have very strong broad-spectrum antibacterial (-cidal) effects and has useful additional antifungal properties. Many refractory cases can be treated using this material.

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Topical antibiotic creams and silver sulfadiazine ointments can be useful but on their own are almost useless. They can also cause softening and maceration of the skin. In all cases the critical issue here is to clip the hair very short and, in the absence of sliver dressings the area should then be kept clean and dry – an open approach is often the best, provided that the skin can be kept free of further bacterial contamination. Corticosteroid creams (often combined with antibiotics of various types) have been advocated and used frequently but whilst these may result in a significant immediate improvement they are usually counterproductive in the end. Repeated applications will significantly alter local immunity and may thin the epidermis significantly.

Severe cases may need parenteral antibiotics based on sensitivity tests, but antibiotic delivery and penetration are unpredictable.

Recovery is usually slow and difficult – persistence usually pays dividends in the end.

Ulcerative lymphangitis

Profile

This is a rare, sporadic, mildly contagious bacterial infection of the cutaneous lymphatics particularly of the lower (usually hind) limb skin, due usually (but not exclusively) to *Corynebacterium pseudotuberculosis*. Mixed infections can also be found.

C. tuberculosis is thought to be spread in summer months by flies – possibly from another infected animal or from 'infected' soil. The bacterium is seldom identified in wounds and so fly bites are possibly a major factor. Poor hygiene conditions may be a factor also.

Several other bacterial species, including *Staphylococcus* spp., *Streptococcus* spp., *Rhodococcus equi*, *Pseudomonas aeruginosa* and *Actinobacillus equuli*, have been implicated in isolated cases in different parts of the world, so the condition perhaps represents a cutaneous lymphatic response pattern to several different infections.

The disease can affect horses of any age.

Key points: Ulcerative lymphangitis

1 6 9 15 17 20 23 24

1. Rare, mildly contagious infectious disorder and cording nodular condition, due mostly to *Corynebacterium pseudotuberculosis* (but may involve other species also), mainly affecting the lower limb regions.
2. Initial signs are usually a diffuse painful oedema of the affected limb(s) and this non-specific sign can easily be misdiagnosed. Corded nodules that may (rarely) ulcerate and exude a small amount of purulent material are also found. The condition is seldom fatal but can be disfiguring and functionally limiting.
3. Diagnosis depends on histology and culture from biopsy materials to reveal the causative organism.
4. Treatment is with antibiotics and systemic or oral iodide treatments. Prolonged procaine penicillin and oral trimethoprim sulphonamide combinations can be helpful.
5. Most cases are disfigured and there may be regular recurrences with continued leg filling and progressive nodule development.

Clinical signs

Three major syndromes are recognized.

(a) *C. tuberculosis* abscess (pigeon breast/Wyoming strangles This form is not strictly a lymphangitis but is caused by the same organism. This condition is geographically restricted to some parts of the USA while the other forms have no recognizable restriction.

(b) Truncal, superficial (mild) corynebacterial pyoderma Initially small, mildly painful papules are noted. The lesions develop into roughly circular areas of hair loss and localized mild swelling with some pain. The areas may expand and coalesce to cause larger areas of hair matting and some hair loss and a transient change in skin/hair colour (Fig. 6.18).

(c) Chronic, deep (limb) form The sudden onset of moderate to severe oedema of the lower limb and swelling extending to elbow or stifle are the first signs of a problem. Most often the hind legs and quite commonly only one leg may be affected. The affected leg is painful but pain responses can be masked by the extensive limb bulk as a result of the oedema. Recurrences of the oedema are common. Serum exudate over the distal limb may occur with cording of the lymphatics of the proximal limb region.

The lymphatic vessels become corded, and firm (sometimes fluctuant) nodules develop along the path of the affected lymphatics. Ulceration and discharge of creamy, greenish pus occurs only occasionally and these may heal quite quickly leaving a small cutaneous scar (which may only be visible if the area is carefully clipped out). Multiple small, draining sores are characteristic of the disease especially in cases encountered in the USA (Fig. 6.19).

Most cases simply present with corded nodules that from time to time appear to be become actively inflamed again and recurrent ulcerations result in progressively more thickened scarred skin.

Differential diagnosis

- Sporotrichosis (*Sporothrix schenckii*): extremely rare, non-painful, non-pruritic, fungal infection of cutaneous wounds; the nodular corded lymphatics are very similar; the organism is almost impossible to find in exudates and biopsy is required.



Figure 6.18 This case is typical of the truncal form of *C. pseudotuberculosis* pyoderma. The earlier papular lesions are shown in this view.

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Figure 6.19 Ulcerative lymphangitis (chronic, deep, limb form). Extensive thickened and scarred lymphatic vessels and numerous ulcerative lesions. Note that the other limb is normal.

- Mycetoma (deep mycosis): localized, usually non-painful isolated skin nodules; biopsy is required.
- Systemic mycosis: deep fungal infections are rare and geographically restricted.
- Epizootic lymphangitis (histoplasmosis/*H. farciminosum*): geographically restricted with a far more aggressive behaviour; characteristic mycology and biopsy.
- Glanders: geographically restricted with far more exudative nature; concurrent respiratory signs and debility.
- Purpura haemorrhagica: vasculitis can be diffuse or focal with defined pathogenesis and previous streptococcal or viral respiratory tract infection.
- Staphylococcal abscess/cellulitis: diffuse and seldom cording; prominent pain and purulent exudate; easily defined bacterial culture.
- Wound healing failure/recurrent trauma: history of wounding and slow healing.
- Foreign body punctures (e.g. thistle and awns): isolated and transient.
- Cutaneous forms of lymphosarcoma: more extensive and systemic signs present usually; defined histology.
- Malevolent sarcoid: defined histology, other lesions on the horse.

Diagnostic confirmation

- Clinical appearance is characteristic.
- Culture from exudate may produce positive cultures of *Corynebacterium pseudotuberculosis*, *Rhodococcus equi*, *Streptococcus* spp. or *Staphylococcus* spp.

Treatment

Depending on the cause (culture results), appropriate antibiotics should be given. Intravenous medication with 5 g sodium iodide daily for 7 or more days or oral administration of potassium iodide can be given until signs of iodism occur (lacrimation and skin scaling). Repeated courses may be required.

Chronic lymphatic nodules can be removed with elastic ligatures around the base of the swelling. The prognosis is very guarded as recurrences are common and treatment is difficult and rather non-specific.

Dermatophilosis (rain scald/mud fever/rain rot)

Profile

Dermatophilosis is one of the most important and commonest bacterial inflammatory skin infections of the horse worldwide. *Dermatophilus congolensis* is a Gram-positive branching actinomycete that is a commensal and opportunistic pathogen of the skin of horses (and cattle and sometimes even humans). The organism does not survive in the environment and the normal dry healthy skin is very resistant to it; to become infected the local protective mechanisms (physical and immunological) have to be compromised to some degree. Chronically infected horses are a common source of infection but the organism is only contagious when the skin of the carrier horse is moistened; this releases the infective zoospores (White 2005). The bacterium can be transferred directly through close contact, via fomites and via insect bites.

For the infection to become established in the skin four conditions are usually required:

1. A source of infectious material (usually other chronically infected horses in close proximity or grooming brushes etc.)
2. A persistently moist skin or skin that is macerated by persistent soaking by rain, wetting, or sweating under rugs and harness is a typical history.
3. Local breaks in the physical integrity of the skin (usually through maceration from continued wetting/sweating) or immunological defence mechanisms localized or generalized immunocompromise.
4. Skin damage (abrasions, rubbing or inflammation from other infections or actinic dermatitis/sunburn.)

It is usually but not exclusively associated with prolonged moist conditions especially when the skin surface becomes macerated (Pascoe 1983). The highest incidence is in out-wintered horses or in wet, warm conditions. Large outbreaks can occur in closely managed horse herds (Yeruham 1996).

The infectious zoospore stage of the organism develops when the skin is continuously damp/wet and so the most obvious and serious infections occur over wet areas and on the distal limbs. When it occurs on the limbs in particular, it is worst on white-skinned areas.

The organism can be transmitted mechanically from horse to horse by grooming brushes and by biting and non-biting flies (Stannard 2000).

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Key points: Dermatophilosis

5 6 11 15 17 22 23 24

1 A very common infectious and contagious bacterial dermatitis due to the Gram-positive actinomycete *Dermatophilus congolensis*. The pattern of infection reflects the areas that are repeatedly or persistently soaked and the worst cases involve neglected horses maintained outside in winter under poor conditions.
2 Variable areas of superficial dermatitis to purulent multifocal–coalescent dermatitis over the back, head and neck. The pastern and cannon are often affected in unhygienic conditions. Initially the lesions are small and are often easier to feel than to see. This is a common presentation in summer conditions with a short hair coat. Winter conditions often induce a much more severe disease with a tessellated hair coat over large areas of the back and neck in particular. Removal of the adherent crusts reveals a purulent base with a rosy or silvery exposed dermis which is not usually obviously infected. Pain is usually mild unless there is secondary infection with staphylococcal or streptococcal organisms.
3 The condition is widely recognized but can be confirmed by direct smears or culture from the purulent exudate or crusts. Biopsies can be helpful also but the sites should not be clipped or scrubbed and the pathologist should be asked to include the crust in the block.
4 Most cases will resolve spontaneously provided that they are removed to a dry environment and groomed carefully to get rid of the crusts and scabs. Treatment principles are:
   a Removal from exposure to wetting (including bedding and pasture)
   b Removal of all overlying matted and damaged hair coat (by soaking and washing)
   c Washing affected area(s) in a diluted chlorhexidine surgical scrub and warm water
   d Rinsing thoroughly after 10 minutes using only warm water
   e Keeping the affected area dry from that point onwards for at least 3 weeks.
   f Systemic antibiotics and possibly topical antibacterial treatments can be helpful but corticosteroids are to be avoided if possible.
5 Control is critical. The disease is contagious and so affected horses should be isolated and all infected hair/crust should be disposed of carefully. Hygiene, nutrition, skin care and blankets and harness care should be improved. There is no significant immunity and so repeated infections do occur.

Clinical signs

Two distinct syndromes are described but there is a marked overlap and there are many variations. The distribution of the lesions reflects the areas which are subjected to moistening or wetting by rain (Fig. 6.20) or by sweating under rugs, etc. The head, neck, dorsum and sides of the abdomen and thorax and the pastern region are most often affected. The condition can also be more localized to limited areas of the body such as the dorsum (Fig. 6.21) and the dorsal cannon skin of the hind limbs in racehorses. In the early stages, the lesions (either on body or limbs or face) are often felt more easily than they are seen.

In spite of quite extensive skin damage, the condition is usually only mildly painful. Removal of scabs and crusts is mildly resented.

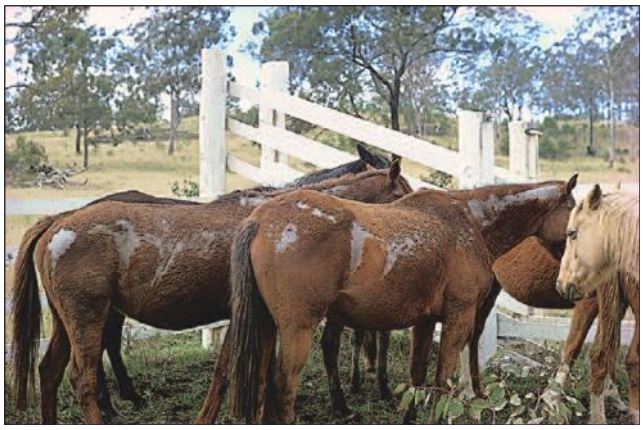


Figure 6.20 Dermatophilosis infection (*Dermatophilus congolensis*). The picture shows several horses with the lesions restricted to those areas which would be wetted by rain ('rain scald').



Figure 6.21 Dermatophilosis infection restricted to a limited area of the dorsum of a coloured horse.

General signs

Mild or severe, localized or generalized alopecia and crusting with little or no pruritus is typical. Severely affected horses may be lethargic and depressed with a poor appetite, weight loss, fever and enlarged lymph nodes.

Lesions on head and legs occur more prominently on white skin areas and can have severe erythema exacerbated by a type of photodermatitis associated with *D. congolensis* infection (Fig. 6.22).

Winter (long-coat) form The coat hair becomes matted/tessellated with an underlying purulent exudate. Skin crusting and flaking occurs – the crusts are often better felt than seen and removal often removes hair as well. Removal of the scab reveals an eroded, ulcerated or more rarely bleeding, purulent skin surface, typically with an ovoid shape, and the base of the scab has a moist purulent appearance (Fig. 6.23). The undersurface of the plucked scab is often concave and has the hair roots protruding through the scab, giving a characteristic 'paint brush' appearance (Fig. 6.24).

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Figure 6.22 Dermatophilosis infection exacerbated by a type of photosensitization. Note that the damage is restricted and does not involve all of the non-pigmented skin. In such cases liver function must always be assessed carefully.

CD



Figure 6.23 Dermatophilosis infection. Removal of a fresh scab reveals a 'pool' of pus and underlying silvery, hairless skin. The organism can easily be identified in the purulent material.



Figure 6.24 Dermatophilosis infection (chronic) showing typical 'paint-brush' dried exudate some way up the shaft of the hairs.

Most active lesions show thick, glutinous, creamy, white-yellow or greenish pus adhering between the skin and the overlying scab. The underlying skin is often remarkably clean and free of suppuration. Magnified examination reveals hair shafts growing back even at an early stage.

Moist eczematous lesions occur on foals kept in wet unhygienic stables and on the back of the pastern of older horses (part of the greasy heel syndrome) (Fig. 6.25).

Summer (short-coat) form Lesions tend to be smaller than the winter form with scabs enclosing a few hairs embedded in a dried exudate which is often fixed some way up the hair shafts (see Fig. 6.24). Scabs are palpable as small 'shot-like' lumps (1–2 mm diameter) often firmly adherent to the skin. Extensive superficial shedding of the hair coat is common, imparting a 'moth-eaten' appearance. In longer-standing circumstances the coat is easily dislodged by grooming (even rubbing with a hand). Extensive areas can be involved.

The palmar pastern area can also be affected by a dry scabbing and scaling dermatitis (Fig. 6.26) with scabs and crusts and some superficial exudate.

A distinctive clinical entity caused by the same bacterium occurs on the hind cannon of racehorses. The lesions are typical of the summer type of dermatophilosis with closely placed, small matted hair patches down the front of both hind cannons (Fig. 6.27). It is likely that this arises from superficial trauma induced during training in wet grass or on cinder tracks.

In some more severe forms there may be permanent changes in the hair coat pattern and colour **(Fig. CD6 • 7A and B)**.

Differential diagnosis

- Papular dermatitis: this is very rare and usually non-painful, non-exudative and not seasonally related.



Figure 6.25 Dermatophilosis infection of the pastern region which is commonly known as 'mud rash'. Notice the marked inflammation and exudation associated with the most severe areas. The organism can readily be identified in direct smears from the area, but mixed infections (with *Staphylococcus* spp. and *Streptococcus* spp.) are common.

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Figure 6.26 Dermatophilosis infection (chronic). Dried, matted and scabbed lesion over the bulbs of the heel and pastern.



Figure 6.27 Dermatophilosis infection affecting only the dorsal aspects of the hind cannon skin which was attributed to traumatic damage from cinder track and wetting through long grass on the gallops.

- Dermatophytosis: characteristic epidemiology, localized lesions at least at first with much less exudate and little or no discomfort; characteristic microbiology on direct and cultural examination.
- Larval nematode dermatitis: seasonal, pastern and coronet mainly involved and very transient; lesions usually restricted to the distal limb regions of foals.
- Chorioptic mange: easily identifiable parasites with main sign of chronic leg and body pruritus.
- Sunburn/actinic dermatosis (facial and pastern forms): restricted to white areas and exposure to sunlight.
- Rope/wire injury or other direct trauma (pastern forms): history of localized injury.
- Pemphigus foliaceus: generalized seborrhoeic condition with little pain; can be secondarily infected.

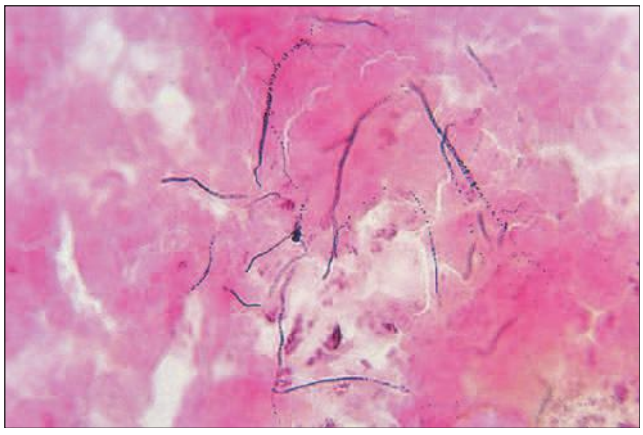


Figure 6.28 *Dermatophilosis congolensis*. Identified by Gram's stain on a smear taken from the purulent material under a scab. Higher magnification will reveal the railway-line appearance of the branching mycelial chains.

- Food allergy: pruritic and or urticarial lesions.
- Alopecia areata: largely asymptomatic with circular areas or diffuse alopecia; more often persistent but can be variable.
- Linear keratosis: characteristic, persistent, non-painful, non-inflammatory linear keratosis and possibly some corresponding alopecia.
- Sarcoidosis and generalized granulomatous disease (enteritis): generalized multisystemic disease with non-painful seborrhoeic, cutaneous signs; some pigmentary incontinence is recognized histologically and possibly clinically also.
- Pastern and cannon leukocytoclastic vasculitis: photo-exacerbated, restricted to non-pigmented skin of pasterns and cannons; can be secondarily infected with *Dermatophilus congolensis*.
- Contact dermatitis: known or suspected contact with toxic or damaging agent in affected areas.
- Pastern folliculitis: restricted to pastern and possibly coronet and cannon regions – can be very similar.
- Coronary band dystrophy: breed condition restricted to coronary band regions.
- Mercurial: 'blister'; known exposure through direct application; systemic poisoning can cause serious generalized skin damage with secondary infections.
- Scalding of skin (wound exudate, urine, (foal) diarrhoea): obvious signs and history.
- Anhidrosis: dry, non-infectious, seborrhoeic dermatitis seldom painful unless secondary infection is present; characteristic history.

Diagnostic confirmation

- Clinical appearance of matted hair encased with exudate and protrusion of hairs is almost pathognomonic. Lesions usually have well-defined anatomical distributions along the dorsum, lower limbs, face, back of the pastern and front of the hind cannons.
- Microscopic examination of a smear made from a fresh scab or fresh pus shows the typical branching 'rail-road' organism. The smears can be effectively stained with any of the commoner stains including Wright–Giemsa (Diff-Quik), Giemsa, methylene blue or Gram's stain (Fig. 6.28).

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- Culture requires specialized methods that provide a microaerophilic environment.
- Biopsy of affected skin should be taken from a carefully selected site. The site should not be clipped or scrubbed or wiped at all before sampling so as to preserve the crusts overlying the lesion. Ideally a freshly crusted lesion should be taken and the pathologist should be asked to include the crust in the block. A presumptive diagnosis of dermatophilosis will usually trigger the correct approach in the pathologist.

Local treatment

Most cases will resolve spontaneously if the skin is kept dry and more so if exposure to sunshine can be provided. The overriding approach must be directed to keeping the skin dry and free of repeated or persistent soaking. Grooming to remove all crusts, scabs and loose hair is desirable but this must be done carefully so as to avoid further skin trauma and only clean grooming equipment should be used. The condition is contagious and so all blankets, brushes, etc. must be individualized, cleaned, and if possible sterilized.

Affected areas on the body should be gently but thoroughly washed with chlorhexidine or povidone-iodine-based antiseptic washes (surgical scrub, for example) using *warm water* for both the washes and the rinses. Gentle warm water soaking of the skin will allow removal of most of the crusts and scabs and infected debris.

The skin should then be dried very carefully, avoiding any rubbing of the skin (possibly with the help of hair dryers set at lower temperatures or blotting with clean paper towels). *Following washing, the skin must be kept dry.*

Localized areas of dermatitis which may crack or exude serum can be treated with emollients containing antibiotics and (possibly) corticosteroids. Debriding creams/gels such as hydrogels and aqueous creams containing malic and propionic acid can be very helpful in the early stages to loosen and separate small, badly affected areas. The best softening material is a 50/50 combination of the malic acid debriding cream and a hydrogel left under a bandage for 24 hours.

Alternative treatments for large areas include:

- surgical scrub antiseptic soaps/scrub solutions (povidone-iodine, tertiary amine or chlorhexidine)
- 0.1% chloramine (a halamide disinfectant)
- 4% polyvinylpyrrolidine-iodine solution
- 4% povidone-iodine solution.

White lotion (zinc sulphate and lead acetate solution BPC) can be helpful for cracked and inflamed areas. A single steroid application can sometimes be justified but steroids should not be continued – the integrity of the local immune mechanisms is integral to the long-term resistance to the disease.

Quite commonly there is a secondary infection with either streptococci or staphylococci and then the extent of pain is usually much greater. The approach to these cases is largely similar but it is not usually possible to clip and debride the affected areas without more analgesia and some sedation. Furthermore, controlling the broader group of bacterial species involved may require a more aggressive initial approach to soften the crusts and reduce the bacterial burden, such as the use of a moistened hydrofibre dressing with silver (Aquacel Ag, Convatec, UK).

Infections on white-skinned areas of the nose and legs may be associated with secondary photodermatitis and despite antibiotic treatment may continue to exhibit dermatitis due to sunlight exposure; stabling or protection of the area from sunlight is a sensible measure and in many cases this results in a significant benefit through a reduction in skin wetting. A sunblock (factor 15 or higher) can be used on white skin in horses which have to be turned out.

Fetlock and pastern lesions Again the areas must be clipped and cleaned in a warm water surgical scrub solution. It is helpful to allow the solution to remain in contact with the skin for 10–15 minutes before rinsing it off with warm water.

Where the crusting (scab) is adherent and pain precludes full removal, a moistened hydrofibre silver dressing can be applied usefully. Simple dressings with wound hydrogels are also very effective means of softening the crusting sufficiently to allow cleaning and clipping of the hair. Milder cases may respond effectively to initial application of a hydrogel under a soft bandage for 24–36 hours. Where crusting is severe it may be necessary and/or helpful to use a more severe debriding system such as ointments containing malic acid. These can be applied under a light bandage and repeated every 24 hours until the crusting can be removed easily.

Note

Bandaging *must* be used with caution: it may increase the severity of the disease by keeping the skin moist and may also cut into the inflamed skin and induce even more exudation.

Contact with wet bedding or urine splashing or even hosepipe water sprays should be avoided.

Repeated or prolonged infection makes the skin even more liable to reinfection. Prophylactic protective bandaging of the fetlock and cannon regions should not be used if possible except during exercise itself.

Chronic scarring occurs with prolonged infections. Secondary lameness can occur.

Note

Over-treatment with harsh antiseptics can lead to physical and chemical dermatitis as a complication of the original infection and must therefore be avoided.

Topical application of corticosteroids may appear initially to bring a good response – usually the pain is reduced and swelling subsides. However, repeated applications cause local immunocompromise, skin thinning and greater susceptibility to repeated *D. congolensis* and commensal/opportunistic infection. A single application can, however, be a helpful measure and spray-on forms of hydrocortisone (see p. 98) are more helpful than creams and ointments.

Systemic treatment

Parenteral procaine penicillin administered intramuscularly daily for 3–5 days is sometimes helpful (Logas & Barbet 1999); *D. congolensis* is usually penicillin sensitive but delivery to skin surfaces is always problematic.

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Systemic or local treatment of generalized infection in large groups of horses is not usually feasible. Under these conditions the horses should be housed to prevent skin wetting and most cases will gradually resolve over 3–6 weeks. Sunlight may hasten resolution in these provided that the skin can be kept dry. Further rain or wetting usually causes a recurrence of the disease. In severe cases, individual treatment becomes necessary. Individual treatments can be very difficult without facilities to prevent skin wetting.

Note

Infections on white-skinned areas of the nose and legs may be associated with a primary actinic dermatitis or the affected region may be photo-exacerbated. Despite antibiotic treatment, therefore, the sites may continue to exhibit dermatitis due to exposure to sunlight. Stabling of the affected animal or protection of the affected area from sunlight becomes essential. Furthermore, primary or secondary exudative (photo- or other) dermatitis may act as a focus for replication of *Dermatophilus congolensis*.

All horses showing dermatitis limited to the white areas must be investigated for possible hepatic involvement or photodynamic plant poisoning.

Control

The condition is contagious and so affected cases should be isolated from unaffected animals. The spores can survive in wet ground and on skin surfaces for considerable periods so early intervention in affected animals is a major aspect of the control.

The disease tends to affect horses in poorly maintained conditions that are regularly or persistently soaked and whose legs are in repeated or persistent contact with wet, muddy conditions.

All affected horses should be removed from exposure to rain or any conditions where they are regularly soaked; even condensation from the inside of roofs can be enough to maintain the condition in the face of any other attempted treatment. Contact with wet grass and wet stable bedding should be avoided. Bedding should be clean, non-irritating and dry. Blankets and rugs that induce sweating can be a persistent source of infection; it is possibly better to leave the horse without rugs or at least to use a clean cotton sheet under the rugs every day.

Over-grooming of mildly affected horses should be avoided and any affected cases should be isolated. All grooming equipment and tack must be washed and preferably sterilized; formaldehyde fumigation or scrubbing/soaking with a tertiary amine disinfectant are effective measures. Individual grooming kits are advisable, in any case, in stable yards.

Note

There is no significant immunity to infection (Zaria 1993) and so repeated infections are common under poor hygiene conditions. When repeated cases occur, hygiene is the focus of attention.

Glanders ('farcy')

Profile

Glanders is a highly contagious, frequently fatal disease of Equidae (and other animals including humans) caused by a Gram-negative bacillus *Burkholderia* (*Pseudomonas/Malleomyces*) *mallei*. The organism is an obligate aerobic pathogen (parasite) which has a poor survival in the environment. It is easily killed by heat, dryness, chemicals and by desiccation.

The disease is highly contagious through direct contact (respiratory and ingested) and indirect contact including water troughs, feed mangers and stable environments; infection is probably acquired by ingestion or inhalation of infective material. Infected horses disseminate large amounts of infective material from the skin and respiratory tract exudates, which spread infection rapidly through a susceptible population. Subclinical cases and the very rare recovered cases may act as carriers for extended periods. Debilitated, malnourished, diseased and stressed horses seem more susceptible to the infection. Mules and donkeys show more severe signs with a more acute course. Apparently recovered animals remain carriers (probably for life).

The disease is characterized by formation of ulcerating nodules in the mucous membranes of the respiratory tract and internal organs. Nodules and ulcerating, discharging skin lesions, which result from cutaneous lymphatic involvement, are known as 'farcy'. The disease can cause devastating loss to the communities where it occurs; it is geographically restricted to certain parts of the Indian subcontinent, east Asia, Saharan Africa and eastern Europe.

The disease has been eradicated from large areas of the world by aggressive slaughter policies. Movement of animals

Key points: Glanders

1 6 7 8 9 11 15 19 20 23 24 25

1. This is a highly dangerous, contagious zoonotic disease with a high morbidity and high mortality. The disease is caused by *Burkholderia mallei* (*Pseudomonas/Malleomyces mallei*). The disease is geographically restricted and is a reportable disease in most countries from which it has been eliminated for many years. The chronic and debilitating nature of the condition and its failure to respond to any known treatment make a slaughter policy the only effective way of controlling it.
2. Clinical signs are restricted largely to the skin and respiratory tracts. Ulcerating cutaneous and mucosal nodules discharge a honey-coloured pus. Affected horses are febrile and frequently have a cough, lymphadenopathy (especially the submandibular and parotid lymph nodes). Death follows septicaemic disseminated infection or when there is severe debility.
3. Diagnosis is usually established from a combination of clinical signs, epidemiological features and the intradermopalpebral (Mallein) test. Particular care must be taken when handling suspected cases and potentially infective material to avoid human infection. Histoplasmosis (epizootic lymphangitis) is the only really significant differential diagnosis.
4. There is no effective treatment although a few cases may be less severely or even subclinically infected – these cases are invariably carriers.
5. The prognosis is hopeless and control by slaughter of infected animals is the only approach available.

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from endemic areas is invariably strictly controlled or more usually forbidden.

Note

People who have contact with affected horses (and especially veterinarians who might carry out close examinations) must take strict precautions to prevent human infection by direct contact or indirectly by contact with specimens or cultures of the organism. Gloves, masks and eye protection are essential. In the human, infection is highly fatal and has a shorter course where immunocompromise is present.

Clinical signs

Acute and chronic forms occur and some infected horses are subclinical carriers.

1 Acute form: This is the rarer form of the disease in horses but commonest presentation in donkeys and mules. Infected animals have an acute febrile respiratory tract infection with a cough, nasal discharge and epistaxis, and painful enlarged submandibular lymph nodes. Death occurs as result of septicaemia within a few days.

2 Chronic form: This form is characterized by skin disease as well as a more protracted respiratory and debilitating systemic disease with typical ulcerative cutaneous nodules. The onset is usually insidious with debility and skin nodules being the main presenting signs. Undulant fever, coughing, nasal discharges and epistaxis may be encountered. The skin lesions can occur at any site but are most common at sites of trauma such as the lateral aspect of the hind limbs and the neck (Fig. 6.29). Lesions start as subcutaneous nodules which ulcerate and discharge a characteristic honey-like secretion laden with infectious material. Granulation, healing and cicatrization follow before the next nodule breaks open. The cutaneous nodules arising from scarring and healing are termed 'farcy'. Progressive spread up lymphatics causes cording, lymphangitis and regional lymphadenopathy. Draining lymph nodes and lymphatic nodules may abscessate, producing deep sinus tracts. Gross thickening of the limb(s) may occur.



Figure 6.29 Glanders. Note the ulcerating cutaneous nodules that are worst along lines of contact with harness **(A)**. Extensive cutaneous involvement is common in the medial thighs **(B)** and the lips and face **(C)**. There is often involvement of the respiratory tract including the upper **(D)**, and lower airways. This disease is a serious welfare issue in horses and mules and is an important zoonosis. It is fortunately rare and geographically restricted at present at least. Strict controls are enforced to prevent its spread from endemic regions.

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Systemic signs of respiratory tract infections with mucopurulent (honey-like) nasal discharge with stellate nasal mucosal ulceration, cough, epistaxis and fever are present in most cases at some time or another.

General signs include malaise, poor exercise tolerance and weight loss. The disease is invariably fatal eventually (often after a prolonged illness).

Differential diagnosis

- Epizootic lymphangitis (*Histoplasma farciminosum*): this condition can be very similar and it is likely that in some countries the diagnosis is easily confused; culture and direct smears are helpful.
- Sporotrichosis (*Sporothrix schenckii*): much more benign condition with few or no respiratory signs but with prominent nodular-ulcerative lymphatic cording.
- Ulcerative lymphangitis (*Corynebacterium paratuberculosis*): a much more benign condition affecting the lower limb regions mostly.
- Respiratory tract infections (*Streptococcus equi equi*, etc.): characteristic microbiology and epidemiology; the acute form of glanders can be very similar.
- Septicaemic (bastard) strangles (*Streptococcus equi equi*): isolated cases with characteristic epidemiology and bacteriology.
- Malevolent sarcoid: this is a single, non-febrile, cutaneous neoplastic condition.
- Cutaneous histiocytic and generalized lymphosarcoma: sporadic, very rare disease with few similarities apart from ulcerative skin nodules.

Diagnostic confirmation

- Geographical region where the disease is known to be endemic.
- Characteristic debility, nasal discharge and inability to work, high mortality.
- Culture of *Burkholderia* (*Pseudomonas*) *mallei*), Gram-negative, facultative anaerobic rods, from discharges and biopsy material.
- The 'mallein (intradermo-palpebral) test' is currently the best means of diagnosis in the living horse. Mallein (a purified protein derivative of killed *Burkholderia* (*Pseudomonas*) *mallei*) is injected intradermally into the lower eyelid about 1 cm from its margin. In affected horses a marked painful swelling at the site after 24 and 48 hours, fever and an ocular discharge develop. The test reflects the marked hypersensitivity of affected horses but some severely affected and debilitated horses may not show a clear positive test result and then clinical assessment becomes the mainstay of diagnosis.
- Complement fixation.
- Bacterial isolation and identification by PCR methods.

Note

The disease can be fatal to humans and extreme care must be taken in sampling suspected cases. The laboratory must also be warned of the special nature of the specimens so that appropriate precautions can be taken.

Treatment

No treatment is available and in any case none is advisable. The reportable nature of the disease makes treatment irrelevant. Death is almost inevitable but can take several months or even some years, during which suffering is considerable and dissemination of the infection can be widespread.

No vaccines that are acceptably effective have been developed.

The disease remains a potential disaster for susceptible populations of horses and so movement from endemic areas is strictly controlled or forbidden. The disease has been eradicated from large areas of the world by statutory aggressive slaughter and quarantine policies.

Clostridial myo/dermonecrosis/malignant oedema/gas gangrene

Profile

Clostridial infections of skin and muscles occur occasionally in horses. Often the cause is a deep penetrating wound but some of the most serious cases follow intramuscular injections or smaller wounds. Cutaneous, subcutaneous and muscular infection occurs with *C. septicum*, *C. perfringens*, and some other species more rarely.

The cardinal features of these infections are their anaerobic nature and their exotoxin production.

Three toxins are probably involved in most cases:

1. α-toxin: necrotizing toxin
2. β-toxin: leukocyte destructive toxin
3. γ-toxin: promotes tissue destruction and spread of infection.

Tetanus and wound-related botulism are well-recognized non-dermatological clostridial infections.

Key points: Clostridial myo/dermonecrosis/malignant oedema/gas gangrene

11 14 17 19 24

1. Rare condition due to anaerobic proliferation in tissues of exotoxin-producing clostridial organisms including *C. perfringens* and *C. septicum*. Sites of skin injury, injections and surgery can be affected.
2. Signs of local tissue destruction and gas formation develop rapidly. The affected area is hot, painful and emphysematous. The toxins cause serious systemic signs of endotoxaemia. Severe tissue destruction continues until death unless suitable intervention takes place.
3. Diagnosis depends on histology and culture from biopsy materials to reveal the causative organism.
4. Treatment centres on establishing an aerobic wound site, so opening the area is critical and possibly flushing with diluted hydrogen peroxide. Systemic antibiotics including penicillin and metronidazole are helpful. Concurrent fluid therapy, flunixin meglumine and analgesics are essential.
5. Many cases die but others can be saved. Recovery and healing may take a long time depending on the site and the severity of the damage.

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Note

Dry gangrene is a non-infectious necrosis of tissue. Infection is seldom involved except at the margin of the necrotized tissues.

Clinical signs

The sudden onset of a severely inflamed, emphysematous local swelling following an injury or injection or surgery is typical. The horse will be severely affected metabolically at the same time with a high fever, depression and anorexia. The skin sloughs rapidly exposing dark black, emphysematous muscle and subcutis (Fig. 6.30 and see Fig. 6.3).

Differential diagnosis

- Staphylococcal abscess/cellulitis: diffuse and seldom cording; prominent pain and purulent exudate; easily defined bacterial culture.
- Wound healing failure/recurrent trauma: history of wounding and slow healing.
- Foreign body punctures, e.g. thistle and awns: isolated and transient.
- Dry (ischaemic) gangrene: the affected region, usually a limb, is cold to the touch and fails to bleed when pricked or cut; pain is minimal.

Diagnostic confirmation

- Clinical appearance and history are characteristic.
- Culture and toxin detection methods take far too long for anything useful to be gained.

Treatment

Treatment principles involve emergency establishment of an aerobic wound site and then maintaining effective antibiosis. Intravenous sodium benzylpenicillin is the treatment of choice. All clostridia are highly sensitive to penicillin but the extent of local necrosis may make delivery of the drug problematic. Therefore it is common practice to open the site and remove as much of the necrotic tissue as possible; this is often possible without any general anaesthesia; sedation and opioid drugs are usually enough. The site is then flushed with saline and possibly a dilute hydrogen peroxide solution (2.5–5 volumes dilution maximum). Direct injection of penicillin into the affected site can be used but will cause further damage.

Supportive fluid therapy and systemic non-steroidal anti-inflammatory drugs such as flunixin meglumine are indicated. In spite of the apparent severity of the infection, it is not the skin problem that results in the death of affected horses. The local damage and toxin production results in severe systemic signs.

The prognosis is very guarded – many cases die within a few hours but some recover and the sites can sometimes heal well.

Dry gangrene

Profile

Dry gangrene is really a non-infectious necrosis of the skin and the subcutaneous tissues that can arise from coagulative necrosis induced by heat, extreme cold, chemicals or toxins applied to the skin or by interruption of the arterial blood supply to a tissue or region. It forms the basis of some important therapeutic measures such as ligation of blood supply to hernial sacs by strangulating elastic bands, cryosurgery, hyperthermia and topical cytotoxic treatment for some tumours such as sarcoid. Topical cytotoxic/coagulative heavy metals and other substances may cause necrosis during sarcoid treatment, for example.

The commonest accidental form is vascular occlusion on a limb or the tail either by strangulating bands of string or rope or by traumatic disruption of the arterial blood supply such as to the tail. Over-tight bandages, wounds and tourniquets (intentional or accidental strangulating wire or ropes, etc.) are a common cause of dry gangrene in the limbs and tail.

The affected tissue sloughs, leaving the viable margin to close by second intention.



Figure 6.30 Clostridial infection of the buttock of a gelding following an intramuscular injection. In spite of the severity of the infection (see Fig. 6.3), healing with impressive wound contraction rapidly occurred following treatment to remove all necrotic tissue and antibiotic administration. (Figure courtesy of Mark Cubberley.)

Key points: Dry gangrene

14 19 20 24

1. Ischaemic necrosis or coagulative necrosis following application of heat, chemical or toxin.
2. Signs are usually related to the cause. Ischaemic gangrene results in a loss of vascular perfusion and the region concerned becomes cold and insensible. There is loss of function and pain is transient. Chemical, thermal or toxin-induced coagulative necrosis is mildly painful in the early stages and then a line of demarcation develops that identifies the sloughing tissues. The sloughing involves all affected tissues.
3. Diagnosis is usually simply on clinical examination and history. The distribution of signs depends on the cause.
4. Usually, in accidental cases, by the time the condition is recognized the changes are irretrievable. Iatrogenic necrosis is easily identified by the history.
5. The outcome depends on the extent of the damage and the relevance to the survival of the horse.

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Bacterial infection is not usually a significant factor except at the margin between healthy and necrotic tissues.

Clinical signs

Vascular obstruction or disruption results in loss of tissue vitality and necrosis. The extent can be very variable. For example, an over-tight bandage around the tail may cause complete vascular obstruction and death of the tail distal to the 'ligature'. On the other hand, it might simply result in superficial dermal necrosis and possibly even only a mild epidermal and melanocytic damage resulting in pigment loss and hair thinning. This is a common result of application of non-elastic exercise bandages in exercising horses. The natural swelling of the leg during exercise cannot occur and so the smallest vessels are occluded first. This then results in further oedema and swelling and finally a complete disruption of the arterial supply. Failure to release the 'tourniquet' in timely fashion results in necrosis distally and a sloughing of the devitalized tissue (Fig. 6.31). In some cases the open wound becomes infected and then the situation can be critical (Fig. 6.32).

Burns (Fig. 6.33) and cryosurgical sites as well as topical application of heavy metal and other compounds such as AW4-LUDES (Fig. 6.34), silver nitrate, arsenic trioxide, zinc chloride/XXTERRA, etc. compounds in the treatment of skin tumours also cause the development of localized coagulative necrosis (eschar).

Differential diagnosis

The loss of blood supply to any tissue is usually obvious – palpation of pulses, a cold (ambient) tissue temperature can be strongly supportive. However, if the skin only is involved, the temperature may be maintained by the underlying inflammatory process. The history of applications or procedures is usually obvious.

- Clostridial/gas gangrene: this is usually obvious by gas/emphysema or by significant local inflammation.

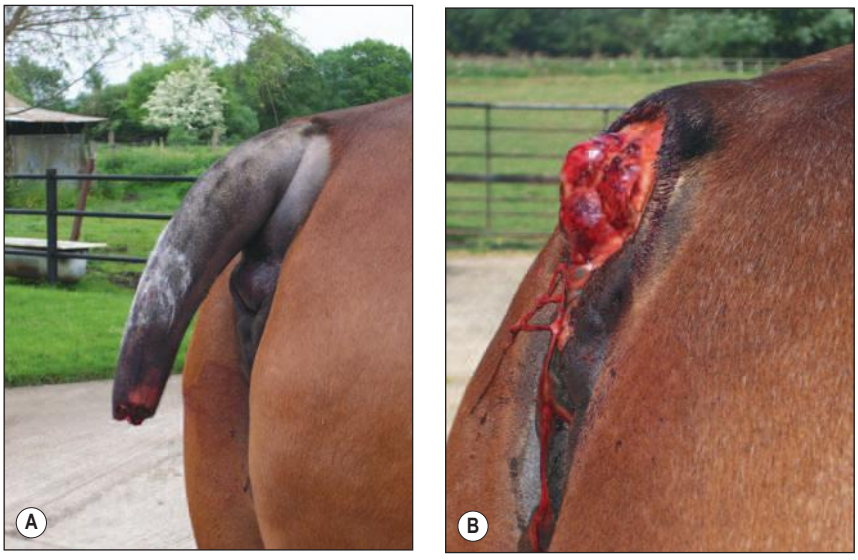


Figure 6.31 **(A)** Tail gangrene caused by an overtight bandage left on for too long. **(B)** The tail sloughed, leaving a granulating wound.



Figure 6.32 Dry gangrene as a result of vascular disruption resulted in a sloughing of the digit – the necrotized distal end of the proximal phalanx can be seen. (Figure courtesy of Ian Kelly.)



Figure 6.33 This horse shows typical coagulative necrosis of the superficial dermis as a result of a burn injury. The slough was very slow to clear and the scar was obvious for life.

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Figure 6.34 Dry gangrene (eschar) caused by application of AW4-LUDES as treatment for a fibroblastic sarcoid.

Diagnostic confirmation

- Clinical appearance and history are characteristic.
- Secondary bacterial infection in the proximal/viable tissues adjacent to the lesion is common but remarkably rarely are they critical issues.

Treatment

Where the approach is part of an intended treatment method, management is simply by analgesia and non-steroidal drugs where required. The pain associated with profound ischaemia only lasts a limited time so most vascular obstruction/disruption cases are examined too late in the course to do anything other than palliative care. The sloughing can be hastened in some cases.

Supportive antibiotics can be helpful. The local damage and toxin production can sometimes result in severe systemic signs.

The prognosis is highly dependent on the extent and the significance of the necrotizing tissues.

References

Chiers K, Decostere A, Devriese L A et al 2003 Bacteriological and mycological findings, and in vitro antibiotic sensitivity of pathogenic staphylococci in equine skin infections. Veterinary Record 152:138–141

Cuny C, Kuemmerle J, Stanek C et al 2006 Emergence of MRSA infections in horses in a veterinary hospital: strain characterisation and comparison with MRSA from humans. European Surveillance 11:44–47

Inokuma H, Kanaya N, Fijii K 2003 Equine pyoderma associated with malnutrition and unhygienic conditions due to neglect in a herd. Journal of Veterinary Medical Science 65:527–529

Logas D B, Barbet J L 1999 Inflammatory, infectious and immune disease. In: Colahan P T, Merritt A M, Moore J N, Mayhew I G (eds) Equine medicine and surgery, 5th edn. Mosby, St Louis, pp 1910–1927

Orsini J A, Snooks-Parsons C, Stine L et al 2005 Vancomycin for the treatment of methicillin resistant staphylococcal and enterococcal infections in 15 horses. Canadian Journal Veterinary of Veterinary Research 69:278–286

Pascoe R R 1983 Dermatophilosis. In: Robinson N E (ed) Current therapy in equine medicine, 1. WB Saunders, Philadelphia, pp 553–554

Pascoe R R 1989 Equine dermatoses, 3rd edn. University of Sydney Post Graduate Foundation in Veterinary Science No. 30. University of Sydney, Australia.

Rose R J, Hodgson D R 1993 Hemolymphatic diseases. In: Manual of equine practice. WB Saunders, Philadelphia, p 335

Scott D W 1988 Immunologic diseases. In: Large animal dermatology. WB Saunders, Philadelphia, p 321

Stannard A A 2000 Alopecia in the horse – an overview. Veterinary Dermatology 11:191–203

Welsh R D 1990 *Corynebacterium pseudotuberculosis* in the horse. Equine Practice 12:7–18

White S D 1988 Corynebacterial folliculitis in a horse. Journal of the American Veterinary Medical Association 193:89–90

White S D 2005 Equine bacterial and fungal diseases: a diagnostic and therapeutic update. Clinical Techniques in Equine Practice 4:302–310

Yasuda R, Kawano J, Onda H 2000 Methicillin resistant coagulase negative staphylococci isolated from healthy horses in Japan. American Journal of Veterinary Research 61:1451–1455

Yeruham I 1996 Outbreak of dermatophilus in a horse herd in Israel. Journal of Veterinary Medicine 43:393–399

Zaria L T 1993 *Dermatophilus congolensis* infection (dermatophilosis) in animals and man: an update. Comparative Immunology and Microbiology of Infectious Disease 16:179–184

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7

Fungal diseases

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A considerable range of infectious actinomycetes and fungi are recognized as potential causes of disease of the skin and internal organs in many species and in all cases they offer significant diagnostic and therapeutic challenges to the clinician. The causative organisms involved and the treatment options in the two broad groups of fungal pathogens are also very different. Fungal skin infections in horses range from some very common superficial disorders such as dermatophytosis (ringworm) caused by almost ubiquitous *Trichophyton* and *Microsporum* fungi, to the exotic and difficult disorders related to secondary wound infections such as pythiosis ('Florida leeches') due to *Pythium* spp., and conidiobolomycosis (black-grained mycetoma) caused by cutaneous and subcutaneous infection with *Conidiobolus coronata*. There is an increasing awareness, too, of the role of fungal skin infections on and within the skin. Some result in diffuse or localized areas of dermatitis while in more benign situations such as the temperate European climate some result in nodular fungal granuloma.

Classification of the fungal dermatological infections in other species is usually based on the location of the infection. This system is also applicable to equine mycotic disease (Table 7.1). Three broad groups of fungal skin disease can be identified:

1 *Superficial mycoses* are those in which the pathogen is confined to the stratum corneum (and does not affect hairs). As might be expected, there is little or no tissue reaction or inflammation and as result they tend to be rather benign. Some of the conditions are viewed as facultative pathogens, or even commensals, rather than primary pathogens and in either case there can be secondary seborrhoeic changes that result in excessive scale and flake on the skin surface. There is usually little immunological or cellular response associated with the organisms and no obvious seroconversion. A good example of this is *Malassezia* spp. infection, which has recently been recognized as a potentially significant skin infection in horses.
2 *Cutaneous mycoses* affect all keratinized tissue including hair, horn and skin, although most of the pathogens are confined to the non-living layers of the skin and the appendages. The organisms are capable of causing significant destruction of keratinized tissue and there are usually immunological responses in the host that result in obvious lesions and seroconversion with variable protective properties.
3 *Subcutaneous/deep mycoses* constitute a heterogeneous group of fungal diseases that are more deep-seated. They involve subcutaneous tissues, possibly in addition to the dermal/epidermal involvement. Whilst most of these remain localized (e.g. *Alternaria alternate*), some do spread insidiously to contiguous tissues, e.g. *Basidiobolus haptosporus* and *Conidiobolus coronatus* infections. Some spread via lymphatic vessels occurs, e.g. histoplasmosis (epizootic lymphangitis) due to *H. farciminosum* and sporotrichosis due to *Sporothrix schenckii*. There may be difficulties with diagnosis and treatment in this group of diseases. Seroconversion may take place but seldom seems to result in an effective protective process. Most of the conditions are chronic and progressive.

The deep mycoses are necessarily much more serious in most cases but are fortunately geographically restricted. Although some of the diseases are sporadic others occur in epidemics or are enzootic. For the most part, however, apart from the major disease epizootics, deep fungal infections are rare or very rare in horses.

Generally the clinical signs of cutaneous mycoses (both deep and superficial) are not pathognomonic in that they may closely resemble those elicited by other microorganisms and some other non-infectious pathological states. For example, some early dermatophytosis cases can closely resemble urticaria and even some forms of pemphigus. Epizootic lymphangitis can be easily mistaken for glanders (and vice versa). In places where the diseases are common, some conditions can be quickly recognized by veterinarians through a combination of historical, clinical and environmental/epidemiological findings. For example, pythiosis is singularly rare outside the southern states of the USA but within the region itself it is well recognized and usually rapidly diagnosed, and black grained mycetoma is a rare but recognized condition in the northern parts of Australia.

There are also some incidental and secondary fungal infections. Therefore, a definitive diagnosis is often dependent on the clear and unequivocal detection of a known pathogenic organism.

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Table 7.1 The main fungal and actinomycete infections of the skin of the horse

Group disease	Disease classification		Organisms (examples)*	Pathogenicity
Superficial mycosis			***Malassezia* sp.**	Secondary Facultative
Cutaneous mycosis	Dermatomycosis	Not reported in horses		
	Dermatophytosis	**Trichophytosis**	***T. equinum***	Secondary (trauma) Pathogenic
			T. verrucosum	
			T. canis	
			T. mentagrophytes	
		Microsporosis	***M. equi***	
			M. canis	
			M. gypseum	
	Cutaneous zygomycosis	Not reported in horses		
Subcutaneous mycosis	Mycetoma (eumycotic)	***Curvularia geniculata***		Secondary (subcutaneous introduction/trauma) Pathogenic
	Subcutaneous phaeohyphomycosis	*Alternaria alternate*		
		Drechslera spicifera		
		***Pythium* sp.**		
	Sporotrichosis		***Sporothrix schenckii***	Pathogenic
	Subcutaneous zygomycosis		***Basiodiobolus haptosporus***	Secondary (skin trauma)
			Conidiobolus coronatus	Pathogenic
Systemic mycosis	**Aspergillosis**		*A. fumigatus*	Mucosal secondary pathogen
			A. nidulans	
	Blastomycosis		*B. dermatiditis*	
	Cryptococcosis		***C. neoformans***	Secondary pathogen
	Histoplasmosis		***H. farciminosum***	Pathogen requires subcutaneous implantation

Significant dermatological disease states are highlighted in bold.
Adapted from Chandler F W, Kaplan W, Ajello L (1980) Taxonomy of the fungi and classification of fungal and actinomycotic disease. In: *Histopathology of Mycotic Diseases*. Wolfe Medical Publications, pp. 9–17.
*Examples are shown of the commoner reported organisms only. Rarely, others may be encountered.

Apart from the superficial dermatophytosis (ringworm) disorders, in which culture and direct microscopic examination of hair shafts and skin scrapings are diagnostic in most cases, the best definitive and practical procedure is biopsy and in some cases histological demonstration of the definitive organism. This also has some difficulty because the juvenile or early forms of some of the deeper mycoses are not 'typical'. New methods of immunohistochemistry involving immunofluorescence and PCR can be used in some cases.

Trichophytosis

Profile

Cutaneous mycosis (ringworm) due to *Trichophyton* spp. is a very common, highly contagious disease which affects horses of all ages. Younger horses are naturally less resistant and take longer to recover than older ones. Transmission is by direct or indirect contact with a source of infection.

The most common species are *Trichophyton equinum* var. *equinum* (TEvE), *T. equinum* var. *autotrophicum* (TEvA), and less commonly *T. verrucosum* (most often from direct or indirect contact with infected cattle) and *T. mentagrophytes* (most often derived from contact with infected rodents and cats) (Pascoe 1979, 1984). *T. equinum* var. *equinum* tends to be the predominate species in the northern hemisphere while *T. equinum* var. *autotrophicum* tends to dominate in the southern hemisphere and the Antipodes.

The spores are highly resistant to environmental destruction and may persist in stables and on tack, etc. for many years. Most cases occur in winter months when horses are closely grouped and groomed heavily using shared or unhygienic tack, harness, clothing and equipment. Wet, warm weather has also been associated with outbreaks but sunshine is a significant inhibitor of the fungi in general. The distribution of lesions in some cases suggests that biting flies may be a significant vector.

Infection relies upon the presence of active (live) spores and mechanical skin abrasion (even if very mild) and this is the reason for most lesions developing on girths and saddle and jockey boot friction areas. The spores become vegetative in the damaged stratum corneum and the fungal hyphae penetrate the anagen hair follicles. In *Trichophyton*

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species infection, the hyphae invade the hair shafts and relatively few spores are produced within the hair shafts (endothrix spores). As the fungal hyphae penetrate downwards towards the hair bulb, keratolytic enzymes are produced. These enhance and facilitate further penetration. Damage to the anagen hair shaft occurs so that the outer portion is shed; the fungus may be expelled with it. In order for the fungus to thrive the hair has to be actively growing and so as soon as the hairs enter the telogen stage the fungus cannot easily survive. At this stage the highly resistant spores are produced and may be shed into the environment with the hair shaft. Most cases resolve spontaneously as a result of hair shedding in telogen phase or as a result of hair breakage or following an immuno-excitatory inflammatory folliculitis; this can be a result of local hypersensitivity responses to the secretions of the fungus. In some cases the complex of secretory and induced inflammatory products results in a more florid inflammation – often with some mild irritation to the horse. Following infection, lesions are visible at around 7–21 days depending largely on the immune status of the horse. Reinfection of a single hair follicle and its hair shaft does not occur until the hair re-enters its anagen phase. If immunity is strong at this stage, the infection is unlikely to re-establish. Clinically the lesions may expand and continue to spread across the horse for some 2–4 months depending on the stage of hair growth and the extent of immunity. Immunity to *Trichophyon* spp. fungi is short-lived; there are some common antigens and so reinfestation with another *Trichophyton* species is less likely. However, there does not appear to be any significant correlation between circulating antibody concentrations and the extent of resistance to reinfection. The natural antifungal effects of healthy untraumatized skin seem to be at least as important in the overall resistance to the disease. Repeated degreasing shampoos are probably not helpful because sebum has a significant protective property.

Immunocompromised horses (such as those on steroid treatment or clinical cases of pituitary pars intermedia dysfunction (PPID/Cushing's disease)) are liable to recurrent, severe and often overwhelming infections.

The positive identification of the species involved (in all dermatophytosis cases) provides useful information on the likely source of infection. The treatment is unlikely to vary but as immunity is generally poor, avoidance of reinfection may depend on a combination of avoiding the source of the infection (where this can be achieved) and sterilization of the environmental challenges arising as a result of spore contamination of buildings, tack harness, rugs, etc.

Note

Although most species of dermatophyte are in theory at least transmissible to humans, this appears to be much less common with the specific equine species. The bovine and pet species of dermatophyte can infect horses and then the infectivity to humans may be greater than with the equine species. Human disease does nevertheless occur and it is always useful to enquire about any human skin disease and if anything is recognized, the person must be referred to a medical practitioner (carrying a note of the suspicion for the animal). Therefore it is important to establish the species involved in an outbreak so that proper measures can be taken to control it amongst the horses and limit the associated human risks.

Key points: Trichophytosis

6 7 8 10 12 15/16 22 23 24

1. Very common cutaneous mycosis due to *Trichophyton* spp. dermatophytes. Worldwide distribution. Spores are highly resistant so repeated infections occur in stables/yards. The fungus requires epidermal damage to gain entry and remains inside the hair follicle and on the hair shafts. The sites of infection reflect areas of superficial skin trauma such as tack and harness contact points. Generalized infection can follow simply from grooming with infected brushes or transferring the fungus to fresh sites on the same horse. The spores can survive for many years.
2. Early clinical signs are erect hairs, some local swelling/oedema with mild exudate in a few cases. As all hairs are involved within a local area, complete shedding of hair occurs and lesions are easily and completely epilated leaving a silvery exposed epidermis. Abrasions from jockeys' boots and girths are common sites for infection.
3. Diagnosis is relatively simple clinically but differential culture is important for control measures. Biopsy can be helpful.
4. Treatment involves isolation of affected horses and careful hygiene to prevent spread between horses and to humans. Topical antifungal washes are effective and oral griseofulvin can be given; it should not be given to pregnant mares. Environmental control is also important to limit the risks of spread so fungicidal disinfectants are used as sprays and washes for tack and harness, etc.
5. Control by vaccination is possible in some countries but early recognition and isolation of cases, and stable and personal hygiene are by far the best ways to prevent its spread. Individualized tack, harness and rugs, etc. are essential, especially in stables with a history of dermatophyte infection. When handling any case of dermatophytosis, gloves should always be worn.

Clinical signs

The earliest lesions appear as erect hairs in circular areas of 5–20 mm diameter (Fig. 7.1). There is often a degree of localized inflammation resulting in a thickening of the skin within the infected area; this can be urticarial in nature and there may be some exudate, which dampens the site. By day 7–10 post-infection hair can easily be plucked from the site (Fig. 7.2), leaving a silvery, slightly reddened circular

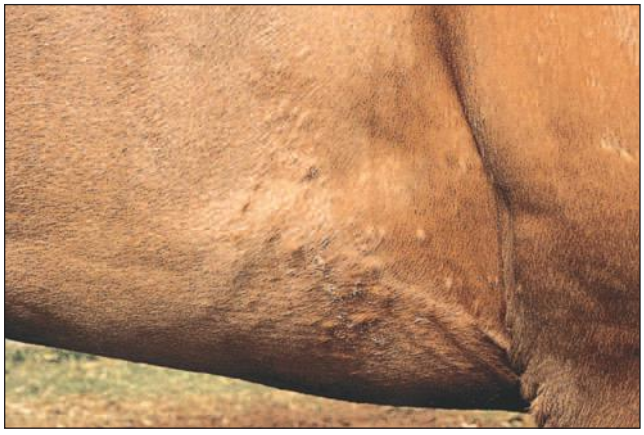


Figure 7.1 Ringworm. Early lesions of *Trichophyton equinum* var. *equinum* infection in the girth area (attributed to a contaminated girth some 5–7 days previously).

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Figure 7.2 Ringworm. Removal of the hair in a mat at 10 days after natural infection with *Trichophyton equinum* var. *equinum*.

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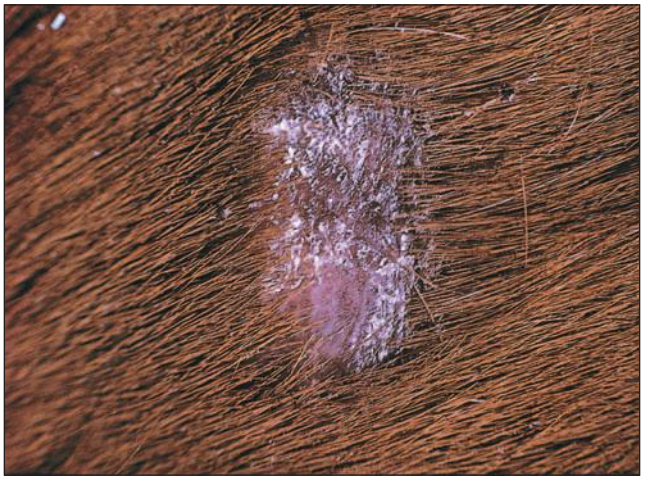


Figure 7.3 Ringworm. The typical silvery, slightly scaly appearance of a lesion due to *Trichophyton equinum* var. *autotrophicum* infection. The hair loss in this case occurred naturally and infection was considered to have arisen some 14 days previously.

area of exposed epidermis (Fig. 7.3). Hair loss also occurs naturally but this is less abrupt and so the lesions may not be obvious until 14–21 days. One of the cardinal signs is the ease with which the hair is removed – this is because most (probably all) hairs are affected within the lesion (Fig. 7.4).

The infected areas expand centrifugally and may lose the circular appearance, becoming diffuse and ill-defined.

Girth and shoulder/chest wall areas are common sites owing to infection from contaminated girths, riding boots, etc. (Fig. 7.5). Generalized infections are also common, particularly in younger horses (Fig. 7.6).

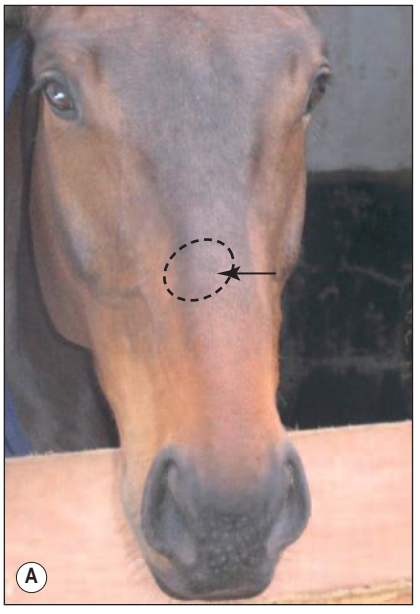
Different species of *Trichophyton* such as *T. verrucosum and T. mentagrophytes*, which are respectively usually derived from infected cattle and rodents, show some differences in the type of lesion (Fig. 7.7 and **Fig. CD7 • 1A–D**).

Lesions are pruritic only in the early stages of infection; however, the horse can be irritable if lesions are 'picked' with a fingernail (when performing this test, due hygiene precautions must be taken for self-protection). This response persists even 5–10 days after treatment if the lesion is still infected and is a useful aid to diagnosis when the hair has been shed and scale and crust are still present on the lesion.

The healing lesions are usually markedly alopecic, smooth and silvery in colour. There may be secondary infection under the shedding scab with accumulation of purulent material.

Differential diagnosis

- *Microsporum* infection: this is a similar condition epidemiologically and clinically but tends to involve only a proportion of the hairs in an infected area; plucking of the hair is therefore more difficult and is sometimes resented.
- Dermatophilosis (*Dermatophilus congolensis*): characteristic bacteria and epidemiology; tends to infect the back and lower limb legions mostly and lesions are more purulent.



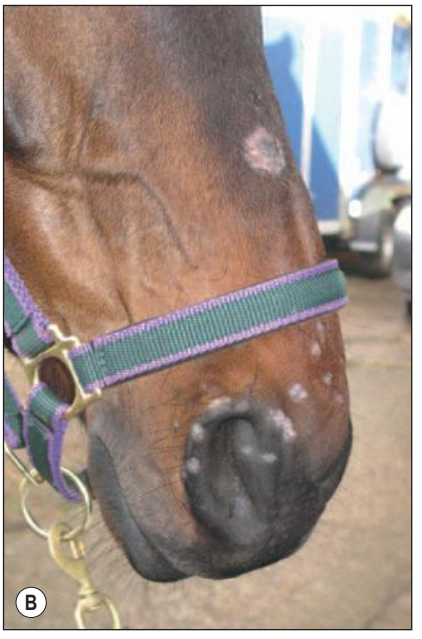


Figure 7.4 **(A)** This 5-year-old Warmblood had a very subtle, slightly pruritic raised lesion with elevated hairs on his nose (arrow). **(B)** By 7 days later a more typical ringworm lesion had developed and several smaller, slightly later lesions were developing on the muzzle.

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Figure 7.5 **(A)** Diffuse infection with *Trichophyton equinum* var. *autotrophicum* in the girth and chest wall areas due to infection from contaminated girths and riding boots. **(B)** The worst affected areas are in the regions where skin rubbing by the tack occurs the most.



Figure 7.6 Ringworm. Generalized infection with *Trichophyton equinum* var. *autotrophicum* in a 6-month-old Warmblood foal.



Figure 7.7 *Trichophyton verrucosum* infection derived from infected cattle. The cattle and the horse fed from the same metal hay rack. Note the generally verrucose nature of the lesions **(A)** and the mildly inflamed, hairless, shiny skin surface that is exposed when a scab is lifted off **(B)**. The severity and extent of the condition was noticeably worse in the region where the head collar made contact with the skin. The owner of this horse was also affected on her arms and neck.

- *Culicoides* allergy/hypersensitivity (sweet itch): severe localized pruritus mainly centred on the mane, tail and in some cases the ventral abdomen, associated with seasonal exposure to *Culicoides* spp. in particular.
- Insect bites: defined localized swellings associated with oedema and a central haemorrhagic spot.
- Mite or louse infestation: obvious parasites and more pruritus; localized to limbs and head and tail; no defined lesions usually present.
- Pemphigus foliaceus: this can be very similar clinically and even histologically and it may require special stains to identify the fungal elements.
- Sarcoidosis: generalized exfoliative seborrhoeic disease with little similarity; some generalized forms of trichophytosis can be similar but they tend to self-cure and mycology and skin scrapings are diagnostic.
- Granulomatous enteritis syndrome: systemic involvement with prominent scaling.
- Alopecia areata: similar circular areas but with characteristic absence of any inflammatory responses and sterile cultures; characteristic histology.
- Anhidrosis: single cases affected with typical history of a move to a warm climate; affected cases may develop secondary trichophytosis due to impaired local immunity.
- Actinic dermatitis: restricted to white regions, particularly on the nose, face and distal limb regions.
- Mercurial poisoning: history of applications.

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Figure 7.8 Hair plucking being taken from one of many circular alopecic, scaling lesions on a pony mare. Note that gloves are worn when handling the case and that hairs are plucked using artery forceps from the margin of the lesion.

- Wound/exudate/lacrimal scalding: history and obvious clinical evidence; may be secondarily affected with trichophytosis due to skin damage.

Diagnostic confirmation

- Characteristic clinical signs and history of contact with infected horses (or other species).
- Hair plucking from the margins of fresh lesions (Fig. 7.8) can be examined microscopically (possibly after clearing with chlorolactophenol or 10% potassium hydroxide solution). Hyphae and relatively few large endothrix spores will be seen.
- Staining with lactophenol cotton blue can assist the recognition of the hyphae.
- Infected hairs do *not* fluoresce under ultraviolet light.
- Culture of hairs plucked from the margins of lesions on Sabouraud's fungal medium. Medium with added phenol red provides an early indicator of dermatophytosis. Culture permits easy identification of the typical macroconidia.
- Skin biopsy is usually diagnostic of dermatophytosis but does not help to establish the species concerned (upon suspicion the pathologists should be informed so that special stains can be used to confirm the hyphae and spores).

Note

It is not easy to be certain of the species of fungus involved without culture. Cultures of hair plucking on commercially prepared Sabouraud's agar at 25°C show characteristic colonies and change in colour of medium. The species can be confirmed from the colony and conidial spore characteristics.

For *Trichophyton equinum* vitamin enrichment is important and this can be achieved by the addition of two drops of injectable vitamin B complex to the medium.

Treatment

Most cases will resolve spontaneously after 6–12 weeks (particularly if the horses are in sunshine) (Pascoe 1973a). Treatments do not shorten the course but may limit the spread of infection and limit the extent of environmental contamination. Subsequent immunity can last for an extended period but some cases can re-emerge following partial elimination from hair follicles.

Treatment is directed at the use of fungicidal treatment of the horse and sporicidal treatment of the environment. The infected areas should be clipped (taking care to disinfect the clippers at regular intervals and particularly thoroughly after each horse). All horses in contact should be considered for treatment at the same time and access to sunlight should be encouraged. The horse(s) may be washed with a fungicidal wash such as enilconazole or natamycin. Proprietary washes of these compounds are widely available. A 2% miconazole–2% chlorhexidine shampoo applied twice weekly has been shown to be effective in reducing the infectivity and so limiting an outbreak (Paterson 1997). Some tertiary amine surgical scrub solutions have a strong antifungal (but limited sporicidal) effect. Spot treatment of lesions (with the above solutions or miconazole) is probably not very useful in the horse in view of the rapid spread across the horse.

Individual lesions and the immediate surrounding hair may also be scrubbed for 1–2 minutes daily for 7–10 days with one of the following treatments:

- 10% povidone-iodine solution
- 2.5% lime sulphur in water
- 10% thiabendazole in water
- 2.5–10% tincture of iodine (painted on, not scrubbed)
- 0.3% Halamid
- tertiary amine disinfectant scrub solution.

Oral griseofulvin may be administered daily for 15–60 days (Hiddleston 1970) but the results of this alone are very variable. There are no reports of its efficacy and many specialists consider that it is of no material help. In any case, it probably does not reduce the infectivity of the spores and fungus-laden hairs. It should therefore not be used alone except perhaps in grazing horses. The drug is teratogenic and must not be used in pregnant mares.

Control

Prevention of spread between horses is important. All scabs and infected hairs should be carefully removed and burned.

Appropriately diluted washes of antifungal drugs such as natamycin, potassium monopersulphate and enilconazole are particularly useful as a spray (or fumigant) for the environment and infected equipment – most have strong sporicidal effects and this will reduce the chances of reinfection or infection of unaffected horses.

A number of modern disinfectants, including in particular the halogenated tertiary amines and inorganic peroxygen compounds, have potent antifungal effects and some are sporicidal. These should probably not be used on the horse unless appropriate instructions from the manufacturer are available.

The stable environment can be effectively disinfected by 'fogging' with potassium monopersulphate (using an industrial or horticultural fogging machine) or enilconazole distributed in the same fashion (Desplenter 1989).

Contaminated tack and other equipment may be washed in suitable fungicidal disinfectants; modern halogenated

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peroxygen compounds have a strong sporicidal and antifungal effect, but these can be unreliable. Preferably, all tack and equipment should be fumigated with formaldehyde gas (see p. 86).

Vaccination is available in some European countries to some species of dermatophytes including *T. verrucosum*. The vaccines rely upon common antigens in the various species of dermatophyte but, because natural immunity is short-lived, the vaccine is unlikely to induce a better immunity. Repeated vaccinations are therefore required. In spite of some reports of severe local reactions (including swelling, pain and abscessation), its efficacy is suggested as being good. There are few studies to support its use but repeated vaccinations are reported to prevent new infections developing in a contaminated environment and limit the severity of the diseases. Its major value probably lies in stables where repeated infections have occurred and where contamination is widespread and uncontrollable.



Figure 7.9 Ringworm. *Microsporum gypseum*-infected insect bites over the rump. Note that the pattern is consistent with biting flies. It was not possible to pluck the infected hairs easily.

Microsporosis

Profile

Ringworm due to *Microsporum gypseum*, *M. equinum* or *M. canis* (microsporosis) is less common than trichophytosis (see above). This disease is also highly contagious, being spread by direct and indirect contact with infected horses or through contaminated equipment or environment. It can also be spread by biting insects and skin abrasion (Pascoe & Connole 1974).

The organism can frequently be isolated from the soil or bedding over 6–12 weeks after infected horses have had access to it. The spores are probably very resistant to environmental conditions and may survive for years.

The pathogenesis is probably indistinguishable from trichophytosis (see p. 168).

Key points: Microsporosis

6 7 8 10 12 15/16 18 22 23 24

1 *Microsporum* spp. are less common in most circumstances than *Trichophyton* spp. infections. Isolated lesions are more common than extensive coalescing areas. The pathogenesis is probably the same as trichophytosis. The common species include *M. equinum*, *M. canis* and *M. gypseum*. The origin of the infection is an important aspect of the epidemiology and control. The spores and the active mycelia can survive for months in bedding, wooden poles and tack/blankets and harness.
2 Clinical signs show first as small expanding areas of localized oedema resembling urticaria (some cases are complicated by urticarial reactions). The lesions may have significant exudate in the early stages and may be mildly pruritic – the horse may rub the affected site against a solid object but does not bite at the lesions. Plucking of the hairs is resented because not all the hairs are equally affected and so many are still firmly attached. This makes the lesions far less distinctive.
3 Diagnosis is made on the clinical signs and the fungal cultures and microscopy of stained smears and cleared hairs. Differentiation from urticaria is relatively simple but secondary urticaria-like plaques can arise as a result of microsporosis. Other similar cutaneous lesions occur in some forms of pemphigus foliaceus, sarcoid and insect bite reactions.
4 Treatment involves local topical washes with fungicidal compounds such as natamycin, enilconazole, or miconazole. The whole body should be washed thoroughly and then the hair clipped off the defined lesions and these areas washed again. Environmental disinfection is important. Sunshine is a strong inhibitor of *Microsporum* spp. fungi.
5 The prognosis is excellent – most cases will resolve spontaneously within 6–12 weeks but the risk of spread to other horses and to humans means that treatment should be instituted.

Clinical signs

Small alopecic areas, most commonly on the face and legs, develop but lesions may also follow the distribution of insect bites elsewhere (Fig. 7.9). The lesions may be mildly exudative and many have an oedematous (urticaria-like) plaque within the affected skin.

Not all the hairs in a particular area will be equally affected and so, when a lesion is plucked, not all the hairs are shed. Plucking of the lesion is therefore more difficult and is often resented by the horse (Fig. 7.10).

This effect is fairly characteristic of microsporosis and is quite different from trichophytosis.

Some lesions are very inflamed and more pruritic than others (Fig. 7.11 and **Fig. CD7 • 2**). Where pruritus develops it is likely that some hypersensitivity is involved and of course this will result in localized spread of the infection and environmental contamination.

Lesions may be exudative and may even be overtly purulent as a result of secondary bacterial folliculitis. A few cases initially present with an urticaria-like wheal that has some exudation and crusting. Lesions are not pruritic but are positive to a scratch test, i.e. the horse will respond to gentle scratching of the area (when performing this test, due hygiene precautions must be taken for self-protection).

In donkeys infected with *Microsporum gypseum*, there may be significantly more hair loss and a much more aggressive verrucose nature **(Fig. CD7 • 3)**.

Note

It is always worth asking about possible infections amongst human contacts – the presence of typical lesions in human contacts **(see Fig. CD7 • 1C–E)** may be incidental of course but it could be helpful also.

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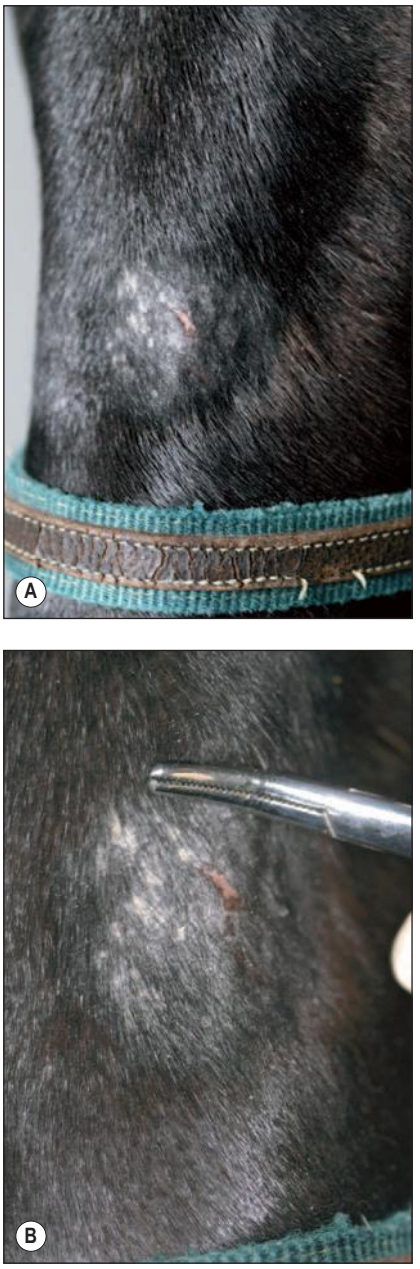


Figure 7.10 **(A)** This *Microsporum canis* lesion developed following a similar infection in the family dog and several humans including the rider. Notice the rather more diffuse area of involvement and the involvement of only a proportion of the hairs. **(B)** Plucking of hairs was difficult and resented.

Differential diagnosis

- Trichophytosis: this is a very similar clinical presentation but in this case the hair is uniformly affected and so plucking removes all the hair from an expanding circular lesion; typical cultural characteristics with a characteristic macroconidia are found; note that in donkeys the presenting signs with *Microsporum gypseum* infection are very similar to the lesions seen in *T. verrucosum* in horses: see **Fig. CD7 • 3** and Fig. 7.4.
- *Stomoxys calcitrans* (stable fly) bites: transient localized oedematous plaques – a central spot can usually be identified.



Figure 7.11 This severe *Microsporum* lesion developed over 6 days and was severely pruritic. The resultant self-trauma caused extensive superficial skin erosions.

- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch): characteristic moderate–severe seasonal pruritus that is worse when outside than inside; this can be complicated by secondary dermatophytosis due to the rubbing on poles and walls, etc.
- Mange mites: localized pruritic disorder with scale and crust in some cases; identifiable mites found in skin brushings.
- Lice infestation, biting or sucking species: identifiable parasites and mild–moderate pruritus with a moth-eaten hair coat.
- Onchocercal dermatitis: facial and ventral midline alopecic grey scaly areas; negative on fungal cultures.
- Occult sarcoid: can be very difficult clinically but protracted static course and other forms of the condition on the same animal are helpful to diagnosis.
- Pemphigus foliaceus: this can be very similar clinically and even histologically and it may require special stains to identify the fungal elements.
- Chemical irritation: history of exposure.
- Alopecia areata: benign static alopecic disorder with variable areas involved; very characteristic histology.

Diagnostic confirmation

- Clinical appearance of lesions in patterns according to the transmission method.
- *M. equinum* and some *M. canis* isolates may fluoresce under ultraviolet light (Wood's lamp). *M. gypseum* does *not* fluoresce.
- Hair samples should be examined after clearing with warm chlorolactophenol or 10% potassium hydroxide. Single or chains of ectothrix spores and hyphae may be seen.

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- Culture of hair plucking on a commercially prepared indicator (Sabouraud's agar) shows characteristic colonies and conidial spores with characteristic colour change.

Treatment

Treatment is as for trichophytosis (above) but the response to both systemic (oral) griseofulvin and topical antifungal/fungicidal washes is significantly slower and more variable. However, again the disease will usually resolve spontaneously in 4–12 weeks.

Control

As for trichophytosis (above).

Removal of affected horses from contaminated yards after treatment is probably advisable, but the organism appears to have less resistance to environmental factors and so, by contrast to trichophytosis, may not appear annually.

Disinfection of the stable and all equipment and tack is important and again formaldehyde gas is probably the most reliable method. Clippers used for several horses are a common source of infection – skin abrasions are almost inevitable. Therefore fungicidal disinfectant trays should be used repeatedly during clipping.

Malassezia dermatitis

Profile

This is probably the most superficial mycosis in horses and is caused by *Malassezia pachydermatis*. The organism has recently gained increased importance in equine dermatology (Nel & Bond 2002). The organism is probably found as a natural commensal of the equine skin and gains significance when there is local or systemic immunocompromise. It may become significant in persistent damp or exudative skin and so is most often identified around the foot and distal limbs of horses with pastern dermatitis (see p. 471).



Figure 7.12 Intermammary dermatitis in a mare that had a long history of perineal pruritus for which another explanation could not be found **(A)**. Removal of the debris and a single wash with miconazole and chlorhexidine resulted in a permanent cure **(B)**.

Key points: *Malassezia* dermatitis

1. *Malassezia pachydermatis* is a natural commensal of equine skin occurring at low levels in normal skin. Local or systemic immunocompromise can predispose to overgrowth of the organism.
2. Mares have the only apparent primary *Malassezia* dermatitis in a syndrome in which the intermammary skin debris becomes infected with the organism. Perineal pruritus is the main sign. Otherwise the signs are usually related to a primary skin disease or damage and pastern dermatitis seems the commonest secondary syndrome associated with detectable forms of *Malassezia*.
3. Diagnosis is simply made on the identification of the organism in smears and cultures.
4. Treatment is simple with miconazole washes. Restoration of skin health is important.
5. The prognosis is excellent but recurrences can occur unless the underlying disorder is managed correctly.

Clinical signs

The most significant syndrome is perineal and ventral abdominal pruritus due to intermammary debris (White 2005). *Malassezia* can usually be found here but it may not be the primary cause of the problem (Fig. 7.12).

Secondary *Malassezia* spp. may also be identifiable in the exudate from some cases of pastern dermatitis.

Differential diagnosis

- Perineal pruritus due to *Oxyuris equi* infestation: eggs are easily identified with adhesive tape tests on the skin around the anus.
- Perineal or hind quarter/tail head pruritus due to insect bite hypersensitivity (sweet itch/*Culicoides* hypersensitivity): characteristic seasonality and epidemiology.
- Pastern dermatitis: *Malassezia* spp. infection is invariably secondary (see p. 471).

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Diagnostic confirmation

Direct smears and culture are required for confirmation.

Treatment

The infection is probably benign.

The organism probably has little primary significance; the only primary condition is intermammary syndrome of pruritus and tail rubbing, which is easily treated by removal and miconazole/chlorhexidine washes.

Cases of pastern dermatitis in which the organism is identified should be washed once with a miconazole wash.

Alternariasis

Profile

Environmental fungal organisms do occasionally infect the skin of horses. *Alternaria* spp. dermatitis (alternariasis) occurs in more temperate countries and is an occasional pathogen infecting mildly damaged epidermis and dermis. The organism is a commensal on the skin and is more often found in spoil and stable environments.

Infection may be gained via open skin abrasions or biting insects may be involved.

A more severe form is possible if the animal is immunocompromised.

Clinical signs

The nodules are usually very slow growing and are neither pruritic nor painful. The ears and breast are more often involved but they can occur at any site (Fig. 7.13). More extensive nodular and dermal serpiginate thickening can occur in sites where skin trauma from pruritus has occurred (Fig. 7.14).

Differential diagnosis

- Cutaneous eosinophilic necrogranuloma with collagen degeneration (collagenolytic granuloma): usually develop along the back, trunk and neck, often multiple and usually very hard, small and commonly have a central calcified core under a small crust; usually very benign.
- Zygomycosis/*Basidiobolus* infection (*Basiodiobolus haptosporus*): geographically restricted to warm humid climates; ulcerative, granulomatous skin disease with interlinking cutaneous and subcutaneous cording nodules.
- Sporotrichosis (*Sporothrix schenckii*): multinodular, ulcerating disorder with characteristic geographic distribution and histopathology.
- Nodular sarcoid: invariably other types of sarcoid lesions are present and the distribution of lesions is

Key points: Alternariasis

9 15 20 23 24

1. An occasional cause of nodular skin disease. *Alternaria* spp. are free-living saprophytic fungi. Infection usually gains entry via small superficial abrasions but insect bite transmission is also possible.
2. Small (0.3–1 cm diameter), firm intradermal nodules with an area of alopecia and possibly crusting in early cases may be found. There is no obvious pain or pruritus associated with the nodules. A few cases have a larger nodule.
3. Diagnosis is difficult without biopsy and the lesions are clinically similar to several other causes of intradermal nodules such as collagenolytic granuloma, sarcoid and melanoma. Special stains are required to demonstrate the fungus in histological sections.
4. Treatment options are limited to surgical excision or benign neglect.
5. The prognosis is good but hygiene should be improved if the patient is repeatedly affected.



Figure 7.13 These small nodules developed slowly over some months in this Shetland pony's ears. The non-painful, non-pruritic nodules were biopsied and special stains and cultures from the central parts of the nodules confirmed *Alternaria* spp.



Figure 7.14 This horse developed a localized pruritus over the hind quarter following a pelvic fracture. This was assumed to be a result of neuritis but biopsies of the nodules revealed *Alternaria* spp. fungal elements in granulomatous nodules. They were surgically removed following partial response to oral potassium iodide.

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significantly different; much longer and more severe course in most cases.
- Melanoma: characteristic black nodules mostly encountered in grey horses in the perineum, preputial skin, eyelids and lip; biopsy is diagnostic.

Diagnostic confirmation

Biopsy and culture are required for confirmation.

Treatment

Where possible surgical removal is curative but recurrence can occur locally if any infection is left. Extensive areas are much more problematic. Cryosurgical necrosis and thermocautery can be used. Oral potassium iodide (5–10 g twice daily by mouth) for 3–6 weeks may improve the condition. Antibiotics are ineffective and systemic antifungal drugs are prohibitively expensive and only marginally effective.

The condition is very benign and so once the diagnosis is established a decision to leave the lesions alone can be justified.

Phaeohyphomycosis

Profile

This is a chronic subcutaneous and systemic fungal disease with small, multiple subcutaneous nodules, caused in tropical countries by *Drechslera spicifera* (Kaplan 1975). It differs from mycetoma and alternariasis (above) in that the hyphae remain discrete and do not aggregate into nodules. The fungus gains access to the skin via wounds. In temperate climates a similar condition (which may be indistinguishable from phaeohyphomyocosis) can occur (most often on the head and ears) from deep and chronic infection with a variety of less pathogenic fungi including *Alternaria* spp. (see p. 176).

Key points: Phaeohyphomycosis

1 5 9 10 11 13 14 15 17 18 19 20 23 24

1 Rare nodular skin disease usually associated with *Drechslera spicificera* and other 'black' moulds occurring in tropical countries in particular.
2 Signs restricted to dark, non-painful, multifocal, firm–solid, cutaneous nodules with overlying hair loss. Some are pustular and can ulcerate. Most lesions occur on the face.
3 Histological examination of excised lesions and stained smears from aspirates may reveal fungal hyphae and a granulomatous inflammation.
4 Treatment is limited to surgical removal of the nodules but iodides and topical antifungal disinfectants and conazoles may be helpful.
5 Prognosis is fair with some cases resolving spontaneously.

Clinical signs

Small, black or darkly coloured, denuded plaques and nodules primarily containing papules and pustules or multiple fibrotic subcutaneous nodules present on the sides of the neck, body (Fig. 7.15) and limbs. The lesions can, however, occur at any site.

Differential diagnosis

- Eosinophilic dermal necrogranuloma with collagen degeneration (collagenolytic granuloma): similarly benign nodular disease with almost no pathognomonic signs but with characteristic biopsy features.
- Molluscum contagiosum: geographically restricted and can look very similar clinically; biopsy is diagnostic.
- Mixed and nodular sarcoid: biopsy is diagnostic and usually other sarcoid types are present.
- Cutaneous lymphosarcoma: larger, more extensive nodules and cording of cutaneous lymphatics; occasional ulceration.
- Melanoma: characteristic black colour and definitive biopsy and fine needle aspirate; usually multiple and most often in the perineum of grey horses.
- Insect bite reactions: transient nodules with urticaria-like reactions; some cases can be more persistent.

Diagnostic confirmation

- Biopsy is essential (fungal elements may be identified directly on sections).
- Culture and identification of *Drechslera spicifera* from the deepest regions of the biopsy specimen on Sabouraud's dextrose agar (without antibiotic additions). Initially the colony grows rapidly and is white-grey but it soon changes to a brown-black colour.

Treatment

Treatment is likely to be prolonged and even in mild cases only marginally effective. Systemic iodide therapy (sodium iodide at 40 mg/kg as a 20% solution by slow intravenous



Figure 7.15 Phaeohyphomycosis. Multiple fibrotic subcutaneous nodules on the sides of the body. *Drechslera spicifera* was isolated from an excised lesion.

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injection once daily for 3–5 days then orally until cured or 5–10 g twice daily of potassium iodide by mouth until signs of iodism appear or a cure is achieved) may be partially effective. Amphotericin B or fluconazole therapy may also be useful but there are no studies on the efficacy. Topical application of etisazole in dimethyl sulphoxide (DMSO) can be effective (Evans 1990).

Mycetoma

Profile

True eumycotic mycetomas are caused by fungal contamination of wounds by free-living (soil and plant) fungi. The fungi cause chronic subcutaneous infections characterized by tumour-like lesions with extensive sinus tracts and fistulas. They commonly have granular components (so-called 'kunkers'). The most common fungi are *Curvularia geniculata* and *Pseudoallescheria boydii*. The former produces a very dark lesion known as a black-grained mycetoma (Boomker 1977, Miller 1980), while the latter produces a white-grained mycetoma. *Alternaria* spp. are also liable to produce small granulomas at the sites of contaminated skin injuries which could be termed mycetoma (see p. 176).

Actinomycotic mycetomas are due to such bacteria as *Actinobacillus* spp., *Nocardia* spp. and *Actinomyces* spp. and so are not strictly mycetomas.

Clinical signs

Ulcerating nodules occur on limbs, head or ventral abdomen with cording of lymphatic vessels. Lesions show a chronic seropurulent discharge from granulating ulcers. The condition is accompanied by mild pruritus. Some asymptomatic nodules, similar in appearance to papilloma but black or dark in colour, covered by ulcerated hairless skin may be present (McEntee 1987). Biopsy of the excised lesion readily identifies the unusual but characteristic colour and consistency of the lesions (Fig. 7.16).



Figure 7.16 Black-grained mycetoma (due to *Curvularia geniculata*). Surgically removed from the skin of a horse in northern Australia.

Differential diagnosis

- Pythiosis (*Pythium* spp.): geographically restricted severely invasive fungal infection of wounds.
- *Basidiobolus* infection (*Basiodiobolus haptosporus*): nodules are very similar.
- Sporotrichosis (*Sporothrix schenckii*): rare, geographically restricted nodular skin disease with lymphatic tracking.
- Glanders/farcy (*Burkholderia* (*Pseudomonas*) *mallei*): severe geographically restricted ulcerative nodular disease affecting the respiratory tract and skin in areas of skin trauma.
- Nodular sarcoid: slowly expanding lesions with nodules more common on the body and several different types present on the same horse; biopsy is characteristic.
- Melanoma: characteristic black nodules sometimes with an ulcerated surface predominately affecting grey horses.

Diagnostic confirmation

- Restricted geographical area.
- Physical examination is suggestive.
- Biopsy with special staining methods is required for confirmation.
- Isolation and culture of *Curvularia geniculata* or *Pseudoallescheria boydii* from deep within nodules or from biopsy.

Treatment

Some cases can be resolved by aggressive surgical removal of affected area, but recurrence is common. Oral potassium iodide (5–10 g twice daily by mouth) for 3–6 weeks may improve the condition.

Antibiotics are largely ineffective and systemic antifungal drugs are prohibitively expensive and only marginally effective.

Key points: Mycetoma

1 Broad group of sporadic and rare cutaneous and subcutaneous nodular disorders resulting from subcutaneous or cutaneous introduction of free-living saprophytic fungal organisms.
2 Subcutaneous and cutaneous nodules and corded cutaneous lymphatic vessels with a tendency to ulceration. Exudates are frequently seropurulent. Pruritus is common and then there may be superficial erosions and secondary bacterial infection.
3 Diagnosis is based on histological and cultural characteristics but some cases are very difficult.
4 Wide surgical excision can be effective in suitable areas but many cases occur on the limbs and face where this approach is impossible. Iodides, amphotericin B and fluconazole may help.
5 The prognosis is guarded. Improvements in overall health may be as effective as anything else.

Sporotrichosis

Profile

This is a chronic, progressive, sporadic infection of skin and subcutaneous/lymphatic tissue caused by *Sporothrix*

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Key points: Sporotrichosis

6 9 14 19 20 22 24 25

1. A serious but sporadic zoonotic disease of the skin caused by *Sporothrix schenckii*.
2. Ulcerative dermatitis and lymphatic cording are the main signs.
3. Diagnosis is solely by histological examination and examination of smears. Culture is very difficult.
4. Treatment with systemic or oral iodides can be successful. Organic iodides such as ethylene diamine dihydroiodide have been shown to be more effective.
5. The prognosis is guarded but depends on the immune capacity of the host – some cases recur and this may be associated with subsequent use of corticosteroids or other immunosuppressive states.

schenckii. The organism is a saprophytic free-living dimorphic fungus. Infection is usually introduced through small skin wounds (Evans 1990). The primary site is usually associated with a small wound on the distal limb region.

The disease is a zoonosis so care should be taken when handling suspected or proven cases.

Clinical signs

The primary lesion is a firm, nodular swelling at the site of a previous (usually) small wound, often at or below the fetlock. Over some weeks or months, corded lymphatics and painless, non-pruritic subcutaneous nodules (1–5 cm diameter) develop running proximally. Lesions can develop on the upper limb regions and body trunk.

Ulceration of surface of nodules sometimes occurs with creamy, white-grey, purulent discharge and surface encrustation and scabbing (Fig. 7.17). The cardinal signs are gradual extension from a single nodule or small cluster of nodules.

Repeated episodes over some months result in lymphatic cording (lymphangitis) in some cases only – medication with corticosteroids may result in recurrences and exacerbation of the condition. A few cases develop isolated nodules and so when nodules are investigated this condition should be considered.

Remarkably, the regional lymph nodes are seldom involved.

Differential diagnosis

- Ulcerative (bacterial) lymphangitis (*Corynebacterium paratuberculosis*).
- Epizootic lymphangitis (*Histoplasma farciminosum*): geographically restricted, debilitating, cording and ulcerating nodular disease affecting the skin of horses, donkeys and mules; organism can be identified in purulent discharges.
- Glanders (*Burkholderia* (*Pseudomonas*) *mallei*): geographically restricted, severely debilitating respiratory and cutaneous fungal nodular disease with ulceration and discharge of a honey-coloured seropurulent discharge.
- Mycetoma: isolated but often complex and slowly expanding cutaneous/subcutaneous nodules that seldom have lymphatic cording.



Figure 7.17 Sporotrichosis. Note the skin and subcutaneous/lymphatic nodules caused by *Sporothrix schenckii* infection of small wounds at sites of skin abrasion from harness.

- Malignant sarcoid: rare condition seldom seen in the same type of horse and in the same geographical regions; characteristic histology and usually there are other sarcoid types present also.
- Cutaneous histiocytic lymphosarcoma and lymphoma: very rare, debilitating cutaneous nodular disease with characteristic histology.

Diagnostic confirmation

- Characteristic cording and nodules on lymphatic vessels.
- Gram stain of exudate from impression smear identifies the causative organism.
- Culture on Sabouraud's agar.
- Biopsy is not usually diagnostic.

Treatment

Iodine therapy is the only available and useful medical approach.

Systemic iodide therapy (sodium iodide at 40 mg/kg as a 20% solution by slow intravenous injection once daily for 3–5 days then oral 10 g daily of potassium iodide until cured or 10 g of potassium iodide daily by mouth until signs of iodism appear.

Organic iodides such as ethylene diamine dihydroiodide administered orally can be useful but prolonged courses are essential (White 2005).

Topical iodine applied to ulcerated lesions has also been used.

The value of amphotericin B is uncertain.

Surgical removal of affected nodules can be performed and can be effective in limiting the disease but will seldom resolve it.

Histoplasmosis (epizootic lymphangitis)

Profile

Epizootic lymphangitis is an important, chronic, highly contagious disease of Equidae caused by *Histoplasma capsulatum* var. *farciminosum*. It is also known as 'African farcy' or pseudoglanders. It is currently restricted to some localized

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Key points: Histoplasmosis

1 5 6 8 9 11 15 19 20 23 24

1. A serious debilitating cutaneous fungal infection of horses, donkeys and mules in limited areas of North Africa, Asia and the Far East caused by *Histoplasma capsulatum* var. *farciminosum*. Altitude is a significant factor with cases encountered within a narrow altitude band.
2. Early cases show localized dermal or subcutaneous nodules which track along lymphatics and ulcerate. Severe forms show coalescing masses of ulcerated nodules with a creamy yellow purulent discharge. Ocular forms are also encountered.
3. Diagnosis is relatively easy in endemic areas but the signs can closely resemble glanders. Culture and smear characteristics from the pus are diagnostic.
4. Treatment is tedious and involves the individual incision and iodine flushing of each infected nodule. Oral and systemic iodides, fluconazole and topical fungicidal disinfectants can be effective for some cases. Severe cases are probably not treatable.
5. Vaccines are not available at present. Prognosis is good if treatment can be instigated in the earliest possible stages but hopeless in advanced cases.

areas of Ethiopia and Saharan Africa. In the affected regions it is a common cause of chronic, debilitating skin disease and sometimes systemic disease and death in working horses and mules. The disease has become a major economic problem in working horses in Ethiopia, Sudan and other parts of north-eastern Africa.

Donkeys were historically regarded as less susceptible but the disease is increasing in prevalence in donkeys.

The disease is transmitted by biting and surface-feeding flies and by direct contact with infected material. The organism is resistant to many environmental conditions and purulent exudates remain potentially infective for some weeks. The organism requires to be introduced into the skin either via a wound site or via an insect bite. It is not certain whether the inhalation route is significant in horses. The risks of transmission are higher in congregated horses and the risks are increased if the animals are debilitated through malnutrition or disease, or are otherwise immunocompromised.

There may be some zoonotic implications, especially where people are immunocompromised. The related *H. capsulatum* var. *capsulatum* can cause severe and highly fatal pulmonary disease in humans but the equine variant is much less pathogenic to humans.

CD

Clinical signs

Several different forms are recognized but they are all chronic and severely debilitating (Fig. 7.18). Severely affected horses eventually succumb and of course act as an infectious reservoir.

CD

Cutaneous form The earliest signs are isolated cutaneous and subcutaneous nodules that can easily be overlooked (Fig. 7.19). The earliest obvious cases have localized oedema and a few ulcerated nodules in the skin (Fig. 7.20). They are seldom painful or overtly inflamed. Recognition of this stage is important because it gives the best chance of treatment.



Figure 7.18 A severely affected, debilitated horse with epizootic lymphangitis. Note the most severely affected regions are the limbs, lower trunk and head.



Figure 7.19 An early case of epizootic lymphangitis. The nodules are prominent but have not yet burst out. Note that this mule in Ethiopia had a wound over the withers region also that was treated inappropriately.

The disease is characterized by suppurative lymphangitis primarily affecting the hind legs and neck, lips and other areas where harness abrasions occur such as the face, girth and hind limbs. Gross enlargement and inflammation of cutaneous (and other) lymphatic vessels with lymphadenitis is typical. Skin ulceration along the corded lymphatics occurs with a 'mouldy' odour from lesions. Often the lesions remain fairly localized in the earlier cases so that one region of tack contact becomes badly affected but the rest of the skin is unaffected **(Fig. CD7 • 4)**.

The severe forms of the disease are more generalized and the extent of skin involvement can be extreme with all areas of the skin being involved to variable degrees. Even in this form the lesions themselves are seldom painful on palpation **(Fig. CD7 • 5)**.

Most cases have no systemic signs, but the disease can affect the nasal cavity, lungs, pleural cavity, eyes and joints, which makes it clinically very similar to glanders. However, generally the absence of significant systemic signs and the definitive smear morphology of *Histoplasma farciminosum* readily differentiate this from glanders.

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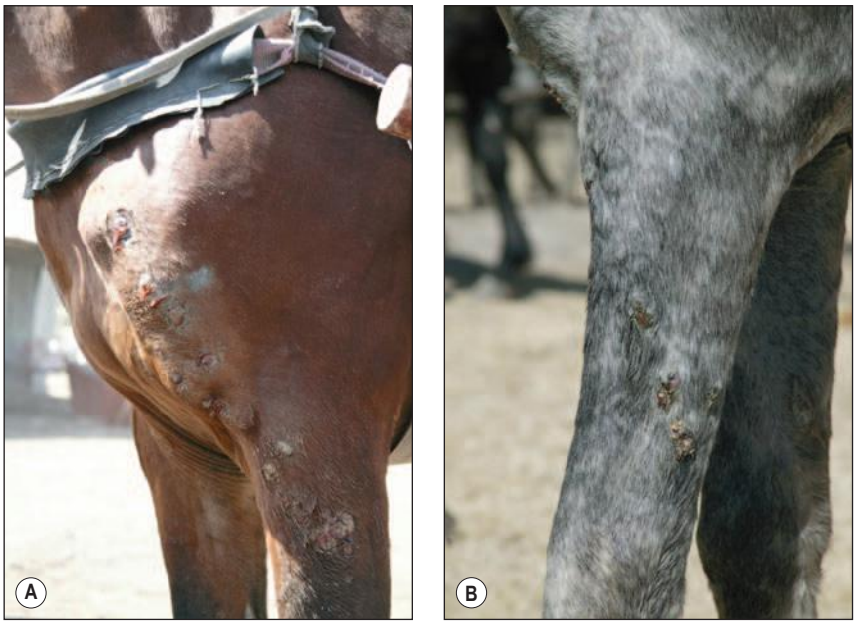


Figure 7.20 (A, B) Two early cases of epizootic lymphangitis. Note the localized swelling and the small, discrete, ulcerated nodules that correspond with the path of cutaneous lymphatics.

Ophthalmic/Ocular form Ocular histoplasmosis affects the conjunctiva and skin of the periorbital region; it is commoner in donkeys than horses (Fig. 7.21). The condition in donkeys is somewhat different in that the lesions are more proliferative and tend to cause obstruction of the nasolacrimal duct **(Fig. CD7 • 6)**.

Differential diagnosis

- Glanders: this is a very similar condition and can easily be mistaken for epizootic lymphangitis. There is more pulmonary involvement and serology and the 'mallein' (intradermopalpebral) test can be used to differentiate the two conditions.
- Ulcerative lymphangitis: sporadic localized ulcerative condition that has characteristic culture and smear identification of *C. pseudotuberculosis*.
- Sporotrichosis: sporadic geographically restricted disease with ulcerating nodules; can be similar to early cases of epizootic lymphangitis.
- Malevolent sarcoid: single cases with different implications and usually several different forms present on the horse.
- Cutaneous lymphosarcoma: sporadic, rare and individual condition without geographical restrictions; characteristic histology.

Diagnostic confirmation

- Direct impression smears from discharging lesions reveal the tissue form of the disease **(Fig. CD7 • 7)**.
- Biopsy and culture from biopsy on Sabouraud's agar can demonstrate the cultural forms of the fungus. However, growth is very slow; over 2–3 weeks is required.
- Serological testing is not yet established.

Treatment

Ideally the disease should be eradicated by an aggressive slaughter policy but, in most endemic areas, that is impractical. In endemic areas treatment is an important consideration.

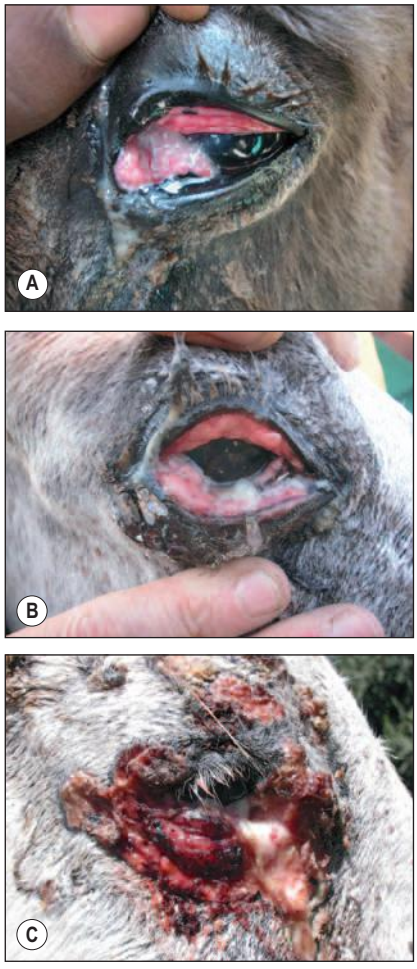


Figure 7.21 This series shows the range of ophthalmic disease that can be caused by *Histoplasma farciminosum* infection in horses and mules. **(A, B)** The two eyes of the same horse. There is extensive conjunctival and some corneal involvement and a marked ocular discharge. Note the voracious attacks of flies which fed in spite of the handling of the eyelids. **(C)** A case showing severely ulcerated periorbital skin.

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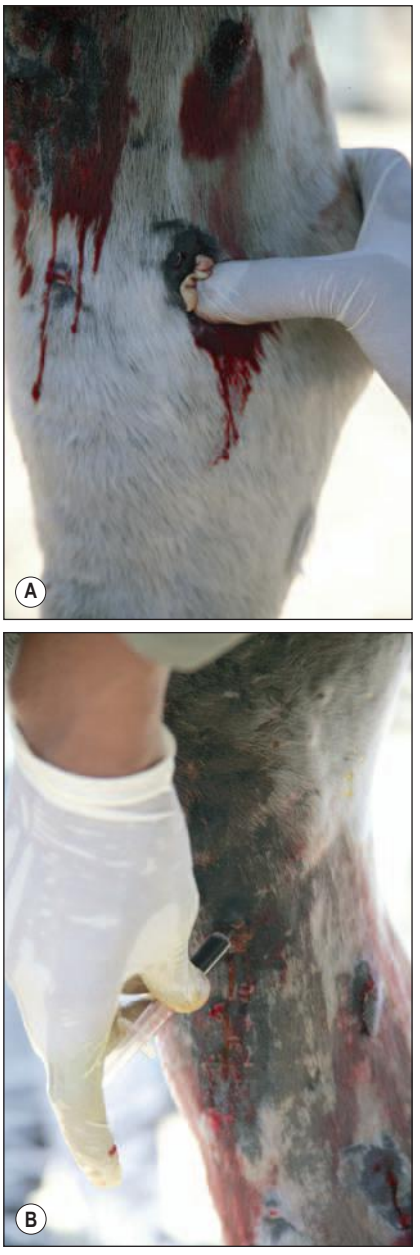


Figure 7.22 Treatment requires the incision of each nodule and expression of the purulent material **(A)** before flushing with strong iodine solution **(B)**.

Provided that treatment can be undertaken, early cases can be treated effectively but it does require considerable dedication and persistence. In theory at least the organism should be susceptible to amphotericin B, nystatin and possibly fluconazole. None of these are usually available in the areas where the disease occurs and in any case treatment would be prohibitively expensive. Other treatment options are limited by the access to the patients over several months or more.

In the early stages the nodules should be individually incised and flushed aggressively with strong (7.5%) iodine solution (Fig. 7.22). Repeated flushing with the iodine solution is required for up to six weekly treatments (and sometimes more). Persistence is the main factor involved in successful treatment – a single treatment is unlikely to help a great deal. Once the fungus is under control, granulation



Figure 7.23 Repeated treatments result in gradual healing of the nodules with granulation and epithelialization.

of the lesions occurs (Fig. 7.23) and the nodules resolve as small focal scars.

Some horses recover spontaneously and are then solidly immune. Animals that are treated effectively are probably also immune.

Control

Surgical drainage of the purulent material and naturally discharging nodules are laden with infective material. The organism can survive for many months in soil and in vegetable mater. Disinfection of the environment following treatment is essential – halogenated peroxygen and quaternary ammonia compounds, or other fungicidal disinfectants can be used.

In all cases fly control is a major aspect of control of spread but in endemic areas this is virtually impossible. Vaccination has been attempted but so far without success.

Cases occurring outside endemic regions are usually slaughtered to prevent spread. Treatment is not usually attempted with slaughter policies in force to control the spread of the condition.

Cryptococcosis (European blastomycosis/torulosis)

Profile

This is a very rare, chronic subcutaneous and systemic fungal disease caused by an encapsulated yeast, *Cryptococcus neoformans*, with several different variants that are geographically restricted. *Cryptococcus* spp. organisms are free-living, saprophytic yeast-like fungi that have a close connection to wild bird faeces and the associated detritus of bird habitats.

The fungus gains access to the skin via wounds and possibly via the inhalation route. Immunocompromise is a major risk factor in all affected species including man.

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Key points: Cryptococcosis

1 4 6 9 15 20 23 24

1 Very rare nodular skin disease associated with *Cryptococcus neoformans* found in different parts of the world and often associated with wild bird faeces and nesting sites.
2 Skin nodules develop around the mouth and the margins of the pinnae in particular. Ulceration occurs with discharge of a thick pus. Concurrent signs may be identified and the condition is worse in immunocompromised situations including concurrent cortisone therapy.
3 Diagnosis is based on biopsy and identification of the circular yeast-like organism.
4 Treatment is difficult at best but fluconazole and iodides by mouth may be useful. Amphotericin and flucytosine may be effective. Individual nodules can be surgically removed. Treatment of any concurrent disease or corticosteroid therapy is advised.
5 Prognosis is poor and there are zoonotic implications with some variants.



Figure 7.24 *Cryptococcus neoformans* var. *gatti* found in a donkey in Morocco. The donkey was severely debilitated through malnutrition and dental disease. There were concurrent blood-sucking lice and a heavy worm burden. The animal was probably immunocompromised.

The virulence of the organism relates to the specific polysaccharide capsule that prevents phagocytosis. Non-capsulated strains have much less virulence. Animals with a normal immune system seem unlikely to be affected. Some forms of immunocompromise may be involved in some cases although concurrent disease may not be obvious. In common with many other of the opportunistic deep fungal infections, concurrent corticosteroid therapy is known to exacerbate the infection. Some cases can lie dormant in the skin until some form of immunocompromise develops.

Clinical signs

Non-specific firm skin nodules, particularly in the lips and pinnae, that expand very slowly are typical. Ultimately these can ulcerate and produce a thick creamy pus. The surrounding tissue may be minimally inflamed (Fig. 7.24 and **Fig. CD7 • 8**).

Differential diagnosis

- Eosinophilic dermal necrogranuloma with collagen degeneration (collagenolytic granuloma): benign nodular disease easily recognized and very common; seldom occurs on the ear flaps and mouth region; typical histology.
- Molluscum contagiosum: geographically restricted and usually multiple with no tendency to ulcerate; biopsy is diagnostic;
- Mixed and nodular sarcoid: biopsy is diagnostic and usually other lesions of sarcoid types are present.
- Cutaneous lymphosarcoma: larger, more extensive nodules and cording of cutaneous lymphatics; occasional ulceration.
- Melanoma: characteristic black colour and definitive biopsy and fine needle aspirate; usually multiple and most often in the perineum of grey horses.
- Insect bite reactions: transient nodules with urticaria-like reactions; some cases are more persistent.

Diagnostic confirmation

- Biopsy is essential (fungal elements may be identified directly on sections) **(see Fig. CD7 • 8)**.

CD

Treatment

There are too few reported series on which to base any treatment advice (Chandra 1992). However, in other species fluconazole (and some other conazole medications) or amphotericin B therapy may also be useful but there are no studies on the efficacy, and dose rates are uncertain.

Surgical removal of isolated nodules can be effective.

Note

Nodules can remain dormant or expand very slowly unless and until an immunocompromise develops or corticosteroids are administered.

CD

Fungal granuloma/pythiosis (phycomycosis/basidiobolomycosis/bursatti)

Profile

This is a chronic, subcutaneous, fungal, ulcerative, granulomatous skin disease caused by *Pythium insidiosum* occurring in subtropical and tropical areas and affecting horses of all types and ages and both sexes. The disease is also called bursatti, Florida horse leeches and swamp cancer. The fungi are aquatic and so wetting and contact with infected plant material in water is essential. The fungi are actively drawn to sites of tissue damage (skin wounds are probably a prerequisite for infection) and skin wetting/maceration. Most cases therefore occur during warm wet seasons. Horses kept in persistently warm wet conditions such as swamps and low-lying water courses are more often affected. Most cases have been reported in Australia, the Gulf states of the USA including particularly Florida, and the Indian subcontinent.

Lesions most commonly occur on the limbs, abdomen, neck, lips and nasal margins and cases rarely show systemic involvement even when the extent of damage is severe.

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Key points: Fungal granuloma/pythiosis/basidiobolomycosis

1 11 15 18 21/22 24

1. A serious secondary, opportunistic infection of wounds resulting in chronic failure to heal caused by *Phythium insidiosum* fungi. Largely restricted to warm, wet climates where the skin may be persistently wet/macerated from soaking. *Basidiobolus haptosporus* causes a similar state.
2. Rapid expansion of a wound site with prominent fibrinous exudate is typical. The wound has extensive dense granulation tissue containing masses of yellow-grey gritty masses ('kunkers'). The fibrinous exudate hangs from the wound (giving it the name 'Florida leeches').
3. Diagnosis is by biopsy, smear recognition and culture of the organisms responsible. Recent ELISA and PCR methods have improved diagnosis significantly.
4. Treatment is limited to wide surgical excision combined with immunotherapy. Iodides are not as effective as for other fungal skin infections.
5. Vaccination holds some promise of effective prevention.

Basidiobolus haptosporus causes a similar disease to pythiosis but is confined to head and abdomen; no leg lesions have been recorded.

Clinical signs

Pythiosis Ulceration of skin or wound with pruritus manifest as biting and kicking at affected area(s). Dense granulation tissue containing masses of yellow-grey necrotic tissue which is sometimes calcified (known as 'kunkers'). The lesions (which are most often single and unilateral) may expand dramatically over a short period, reaching a very large size.

Characteristic sticky, stringy serosanguineous discharge which either mats into the hair or hangs from body wall in thick mucopurulent strands (Fig. 7.25). Lymphangitis with swelling and oedema develop in chronic cases, primarily on legs (Fig. 7.26) and ventral abdomen. Joints and tendons may be involved with chronic discharging sinus formation. Lymphadenopathy may occur if the fungal elements gain access to the draining nodes.

Pruritus may be present.

Basidiobolomycosis This disease has a very similar presentation to pythiosis but growths are more shallow and confined more to the neck and abdomen. Limb lesions are less common (Fig. 7.27).

Differential diagnosis

- Sarcoid (particularly if recurrent interference): localized tumour condition with characteristic histology.
- *Habronema musca* infestation in wounds: this can be very similar but tends to be less destructive and is far easier to treat. The larva can be identified in washings from the lesion.
- Neoplasia (including cutaneous lymphosarcoma): sporadic, non-infective and usually slow to expand. Systemic signs are common and histology is diagnostic.
- Mycetoma: probably similar but much less aggressive.
- Botryomycosis (staphylococcal pyogranuloma): biopsy and culture of *S. aureus* (usually) are diagnostic.
- Exuberant granulation tissue (from any cause) or indolent or non-granulating: can be difficult to differentiate if the granulation tissue is infected.



Figure 7.25 'Florida leeches' on the ventral abdomen. The subcutaneous fungal, ulcerative granulomatous lesions were caused by *Pythium insidiosum*.



Figure 7.26 Pythiosis. Typical lymphangitis with swelling and oedema of the leg. Lymphadenopathy was present in the inguinal and iliac lymph nodes.



Figure 7.27 Basidiobolomycosis. Severe case with extensive shallow ulceration and exudation confined to brisket and base of the neck. There were no limb lesions.

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Diagnostic confirmation

- *Pythiosis* usually has a history of access of horses to water-logged pasture or lagoon creeks; *basidiobolomycosis* occurs more in dry conditions with the organism probably living in the soil.
- Clinical appearance of characteristic mucopurulent strings and 'kunkers' in exudate or in cut surface of lesion.
- Biopsy (collected into 10% formol saline) shows increased collagen and intense eosinophilic infiltration.
- Fungal culture on Sabouraud's agar (using a fresh sample sent on ice) from early lesions identifies organisms responsible.
- ELISA and PCR methods hold promise of an improved diagnosis.

Treatment

Early treatment is essential as the prognosis is much poorer in chronic cases.

Surgical excision of the tumour under general anaesthesia is the most common and most successful treatment, particularly in chronic cases (Bridges & Emmons 1961, Restrepo et al 1973, Pascoe 1989, Moriello et al 1998). Complete removal of all affected tissue is essential if recurrences are to be avoided. Surgery should be performed again as soon as any new lesions develop (usually manifest as 1–5 mm diameter dark red or black spots). Surgery requires extreme caution on the limbs in particular and iodine-soaked pressure bandages should be applied after excision is complete. Repeat surgery is commonly required when the lesions occur around tendons and joints.

Intravenous sodium iodide (Pascoe 1973b) is a useful adjunct to surgery. Amphotericin B may be administered intravenously once daily and topically (McMullan et al 1977). For intravenous administration, amphotericin B is available as a vial containing 50 mg lyophilized amphotericin B. The required dose is dissolved in 1 litre of saline and slowly infused intravenously over 1 hour. Intravenous treatment is well tolerated at an average daily dose starting at 0.3 mg/kg rising to 0.6 mg/kg on day 3 and then every other day for the required time (10–80 days). For local treatment of the wound site or fresh lesions, 50 g amphotericin B in 10 mL sterile water + 10 mL dimethyl sulphoxide (DMSO) on a gauze dressing is applied daily for at least 1 week.

Significant benefit may be obtained by surgically removing the bulk of the fungal growth, followed by daily intravenous and topical administration of amphotericin B, with periodic extirpation of small necrotic tracts as necessary. Some strains of *Pythium* have shown resistance to amphotericin B (Eaton 1993).

Vaccination may provide therapeutic and prophylactic effects (Miller 1981, Newton & Ross 1993, Mendoza et al 2003) but vaccines are not manufactured or available commercially and need to be prepared by a suitably qualified laboratory.

Prognosis

Successful treatment depends on the age and physical condition of the horse, previous treatment, age/size/site of the lesion and whether there is bony involvement. Young fresh lesions of 2 weeks' duration respond well to immunotherapy alone. Older lesions respond poorly and all reported cases with bone involvement have died (Alfaro & Mendoza 1990).

References

Alfaro A A, Mendoza L 1990 Four cases of equine bone lesions caused by *Pythium insidiosum*. Equine Veterinary Journal 22:295–297

Boomker J 1977 Black grain mycetoma (maduromycosis) in horses. Ondesterpoort Journal of Veterinary Research 44:249

Bridges C H, Emmons C W 1961 A phycomycosis of horses caused by *Hypomyces destruens*. Journal of the American Veterinary Medical Association 138:579

Chandra V K 1992 Localised subcutaneous *Cryptococcus granuloma* in a horse. Equine Veterinary Journal 25:166–167

Desplenter L 1989 Dermatophytosis in animals: topical treatment and environmental control with enilconazole. Advances in Veterinary Dermatology. In: Proceedings of the First World Congress of Veterinary Dermatology, Dijon, pp 453–454

Eaton S A 1993 Osseous involvement by *Pythium insidiosum*. Compendium of Continuing Education for Practising Veterinarians 15:485–488

Evans A G 1990 Alterations in skin. In: Smith B P (ed) Large animal internal medicine. Mosby, St Louis, pp 209–238

Hiddleston W A 1970 The use of griseofulvin mycelium in equine animals. Veterinary Record 87:119

Kaplan W 1975 Equine phaeohypomycosis caused by *Drechslera spicifera*, cited by Scott D W (1988) Large animal dermatology, WB Saunders, Philadelphia

McEntee M 1987 Eumycotic mycetoma. Review and report of a cutaneous lesion caused by *Pseudallescheria boydii* in a horse. Journal of the American Veterinary Medical Association 191:1459–1461

McMullan W C, Joyce J R, Henselka D V, Heitmann J M 1977 Amphotericin B for the treatment of localised subcutaneous phycomycosis in the horse. Journal of the American Veterinary Medical Association 17:1293–1298

Mendoza L, Mandy W, Glass R 2003 An improved *Pythium insidiosum* vaccine formulation with enhanced immunotherapeutic properties in horses and dogs with phythiosis. Vaccine 21:2797–2804

Miller R I 1980 Black grained mycetoma in two horses. Australian Veterinary Journal 56:347

Miller R I 1981 Treatment of equine phycomycosis by immunotherapy and surgery. Australian Veterinary Journal 57:377–382

Moriello K A, DeBoer D J, Semrad S D 1998 Diseases of the skin. In: Reed S M, Bayly W M (eds) Equine internal medicine. WB Saunders, Philadelphia, pp 534–536

Nel A, James S A, Bond C J 2002 Identification and distribution of a novel *Malassezia* species years on normal equine skin. Veterinary Record 150:395–398

Newton J C, Ross P S 1993 Case notes and commentary: equine pythiosis – an overview of immunity. Compendium of Continuing Education for Practising Veterinarians 15:491–493

Pascoe R R 1973a Dermatophyte infection in the horse. Fellowship Thesis, Royal College of Veterinary Surgeons, London, UK

Pascoe R R 1973b The nature and treatment of skin conditions observed in horses in Queensland. Australian Veterinary Journal 49:35–40

Pascoe R R 1979 The epidemiology of ringworm in racehorses caused by *Trichophyton equinum* var. *autotrophicum*. Australian Veterinary Journal 55:403–407

Pascoe R R 1984 Infectious skin diseases of horses. Veterinary Clinics of North America, Large Animal Practice 6:27–46

Pascoe R R, Connole M D 1974 Dermatomycosis due to *Microsporum gypseum* in horses. Australian Veterinary Journal 50:380–383

Restrepo L F, Morales L F, Robledo M et al 1973 *Antioquia Medica* 23:13–25; cited by R I Miller (1980) Treatment of equine phycomycosis by immunotherapy and surgery. Australian Veterinary Journal 57:377–382

White S D 2005 Equine bacterial and fungal diseases: a diagnostic and therapeutic update. Clinical Techniques in Equine Practice 4:302–310

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8 Protozoal diseases

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There are limited protozoal infections that affect the equine skin. These are invariably also geographically restricted. Whilst some such as besnoitosis and leishmaniasis have quite prominent skin implications, conditions such as dourine (trypanosomiasis due to *T. equiperdum*) have generalized clinical signs that are usually of far greater importance than the cutaneous signs. However, the cutaneous signs may be an early indication of some of the conditions and may also be the only outward sign of disease. Therefore the possibility of a protozoal diagnosis should not be forgotten.

The mechanisms for the pathogenesis of protozoal skin disease are very poorly understood and seldom documented satisfactorily, possibly because the conditions affect a limited equid population and because the cutaneous signs are usually mild compared to the systemic signs.

The complications of diagnosis are matched only by the difficulty of treatments for most of these conditions. There are limited anti-protozoal drugs that can be safely used in horses and the most serious of the conditions, known as dourine, is a venereally transmitted trypanosome (*Trypanosoma equiperdum*) infection. It is widely feared because of its chronic, intractable and severely debilitating nature and its very high mortality.

Besnoitiosis (globidiosis/elephant skin disease)

Profile

This host-specific protozoal disease is due to either *Besnoitia besnotii*, or in donkeys *B. bennetti*, acquired by ingestion of feed contaminated with cat faeces, or by bites from blood-sucking insects and parenteral injection of blood from acute cases. Horses and donkeys have been reported affected in South Africa (Bigalke & Prozesky 1994) and North America (Terrel & Stookey 1973, Davis et al 1997).

Key points: Besnoitiosis

1. Rare, geographically restricted skin disease of horses and donkeys. Probably transmitted directly by biting insects.
2. Systemic signs including weight loss, depression and a nasal discharge may be present.
3. Multiple, small cutaneous papules with prominent flaking and scaling and more obvious papules in glabrous (hairless) skin. Some lesions have a glue-like exudate.
4. The organism in a biopsy of a topical papule is pathognomonic.
5. Potentiated sulphonamide may be effective when given for 30–60 days.

Clinical signs

Chronic, pruritic, alopecic dermatitis with generalized thickening and lichenification of the ventral abdominal skin from sternum to scrotum or neck and withers (Fig. 8.1 and **Fig. CD8 • 1**) is characteristic of the disease. Multiple

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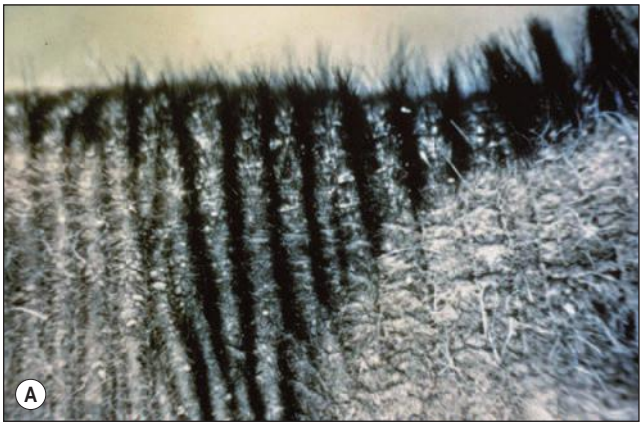




Figure 8.1 *Besnoitia bennetti* infection causing extensive dry scaling and thickening of the skin over the withers **(A)** and neck **(B)**. (Figures courtesy of Dr J J van Heerden.)

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papules may be present in subcutis of perineum and abdominal area. The hair is sparse and the skin is inclined to crack and ooze. The worst lesions are usually around the fetlock. Small nodules may be present on sclera, soft palate, pharynx and larynx.

Exercise intolerance and bilateral nasal discharge may be present (van Heerden et al 1993). Other signs include an enlarged oedematous prepuce in males.

Differential diagnosis

- Dourine (*Trypanosoma equiperdum*): geographic restriction and venereal transmission are features; very easy identification of the parasites.
- *Culicoides* spp. hypersensitivity (sweet itch) and ventral biting *Culicoides* spp. hypersensitivity: sporadic and rare in donkeys, prominent seasonal management related pruritus.
- Equine viral arteritis: transient viral infection with minimal skin signs.
- Sarcoidosis: sporadic seborrhoeic disease with systemic signs. Diagnosis largely by exclusion of alternatives.
- Sarcoid (occult/verrucose forms): characteristic histology and typical appearance with multiple lesions over focal and sometimes more extensive areas in typical sites.
- Heavy metal poisoning: access and identifiable toxicological analysis in hair and hoof in particular.

Diagnostic confirmation

CD

Biopsy reveals spherical *Besnoitia* cysts containing the bradyzoites in the dermis with interstitial fibrosis and a chronic plasma cell exudate (Robertson 1976) **(Fig. CD 8 • 1)**.

Treatment

Prolonged courses of potentiated sulphonamide (trimethoprim/sulfadiazine) by mouth or injection (30–60 days or more) may help significantly but there are too few reports to provide a definitive treatment.

Prevention

Prevent cat or rodent contact with feedstuffs. If biting insects are present, use insect screens around infected animals.

Leishmaniasis

Profile

The causative organism is an obligate intracellular parasitic protozoan belonging to several members of the genus *Leishmania* including *L. braziliensis*, and is transmitted between horses by a few species of biting sandflies such as *Lutzomyia cayennensis* and *Phlebotomus* spp. The disease can, in theory at least, be transmitted to humans but there is little information available on this aspect (Yashida 1990).

In endemic areas such as South and Central America (particularly Brazil), southern Europe, and the Middle East, the cutaneous form of the disease occurs frequently in horses, donkeys and mules of all types and ages (van der Lugt & Stewart 1994). Isolated cases have been reported from the USA (Ramos-Vara et al 1996) and South Africa but it is extremely rare in non-endemic regions.

Key points: Leishmaniasis

1 6 7 8 9 11 15 20 23 24

1. A rare, geographically limited, protozoal disorder with cutaneous lesions.
2. Isolated single or multiple ulcerated skin nodular lesions often with a 'piled-up' margin may be found on the body trunk or ears/scrotum or face.
3. Diagnosis is difficult but relies upon identification of the organism in biopsy or smears taken from an active lesion.
4. No information is available about possible treatments in horses.
5. Successful treatment with sodium stilbogluconate has been reported but there is little available information on the prognosis of affected horses.

Clinical signs

Single or multiple, raised, crusted or ulcerated, papules and nodules (0.5–1.5 cm diameter) are found on the head (particularly the muzzle, pinnae and periorbital skin), scrotum, legs and neck. The lesions may have a 'piled up' margin and a non-healing, ulcerated, depressed centre.

Differential diagnosis

- Dourine (*Trypanosoma equiperdum*): geographically limited but with some overlap; characteristic debilitating disorder with obvious parasites obtained from aspiration or biopsy of cutaneous lesions.
- *Gastrophilus* spp. larval (hypersensitivity) ulcers: transient, temperate climates mainly and usually restricted to lips and rostral face skin.
- Sarcoidosis: generalized multisystemic granulomatous disorder with no distinct skin lesions.
- Collagen (necrosis) granuloma: common very benign multifocal disorder with characteristic histopathology.
- Basal cell carcinoma: isolated single lesions with very distinctive histopathology.

Diagnostic confirmation

- Biopsy: multinucleated giant cells can be found and the amastigote stages of the organism may be visible within macrophage sections and in macerated tissues.
- Real-time PCR to detect leishmanial DNA has been used (Roao et al 2005).

Treatment

A single case report suggests that sodium stilbogluconate may be curative (Ramos-Vara et al 1996). Standard treatment is limited to antimony or arsenical compounds. Amphotericin B has been suggested as being effective but in most circumstances treatment is impractically complicated or expensive (or both).

Prevention

Only avoidance of fly contact and isolation of cases help to control the spread of the disease. Fortunately it is not common and remains geographically restricted but the potential role of the carrier horse may have implications for international disease control.

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Key points: Dourine

1 9 15 19 20 24 25

1. An important debilitating, chronic systemic disease with cutaneous signs geographically restricted to some areas of Central America, some areas of Africa and the Far East.
2. Clinically skin lesions are limited to localized areas of oedema (wheal-like) with exudate in some cases. There may be few, several or many. The systemic signs are far more evident (genital oedema, debility, inappetence and lethargy).
3. Diagnosis is made by identifying the *T. equiperdum* organisms in fluid aspirated from the cutaneous lesions – they are rarely found in blood. Serological tests can be helpful.
4. Treatment is not usually attempted or recommended. Euthanasia is the best option.
5. The prognosis is very poor and the venereal nature of the disease is a serious limitation to the breeding of affected individuals.

Dourine (*Trypanosoma equiperdum*)

Profile

Dourine is a venereal protozoal disease with cutaneous manifestations caused by *Trypanosoma equiperdum*.

The disease occurs in limited areas of Africa, the Middle East, South America and Asia. Horses have been reported affected in South Africa. The disease has been eradicated from large areas of the world including Europe and North America.

Clinical signs

The condition has an insidious onset following a prolonged incubation period after infective coitus. Venereal signs are most prominent with swelling of the male and female external genitalia and preputial or vulvar discharge.

Characteristic (pathognomonic) 'silver dollar' dermal plaques (2–10 cm diameter) may develop in some cases (Robertson 1976) (Fig. 8.2 and **Fig. CD8 • 2**). They develop first in the perineum and then at any site on the skin, with most obvious lesions on the flanks. Plaques are oedematous ('pit' on pressure) and individual lesions may last for hours or weeks. An enlarged oedematous penis and prepuce with purulent urethritis occurs in males.

Progressive lameness and gluteal muscle atrophy are the more visible general signs. Weight loss, loss of performance and terminal paralysis develop as a result of internal disease. Terminal central nervous signs are associated with invasion of the central nervous system. Death is almost inevitable.

Differential diagnosis

- Urticaria: transient possibly recurrent characteristic oedematous papules and plaques with rapid onset and equally rapid resolution following glucocorticoid administration.
- Insect bites: single isolated or multiple bites with recognizable central focus. Transient effects after acute onset. Possibly some discomfort and pruritus but seldom any systemic signs.

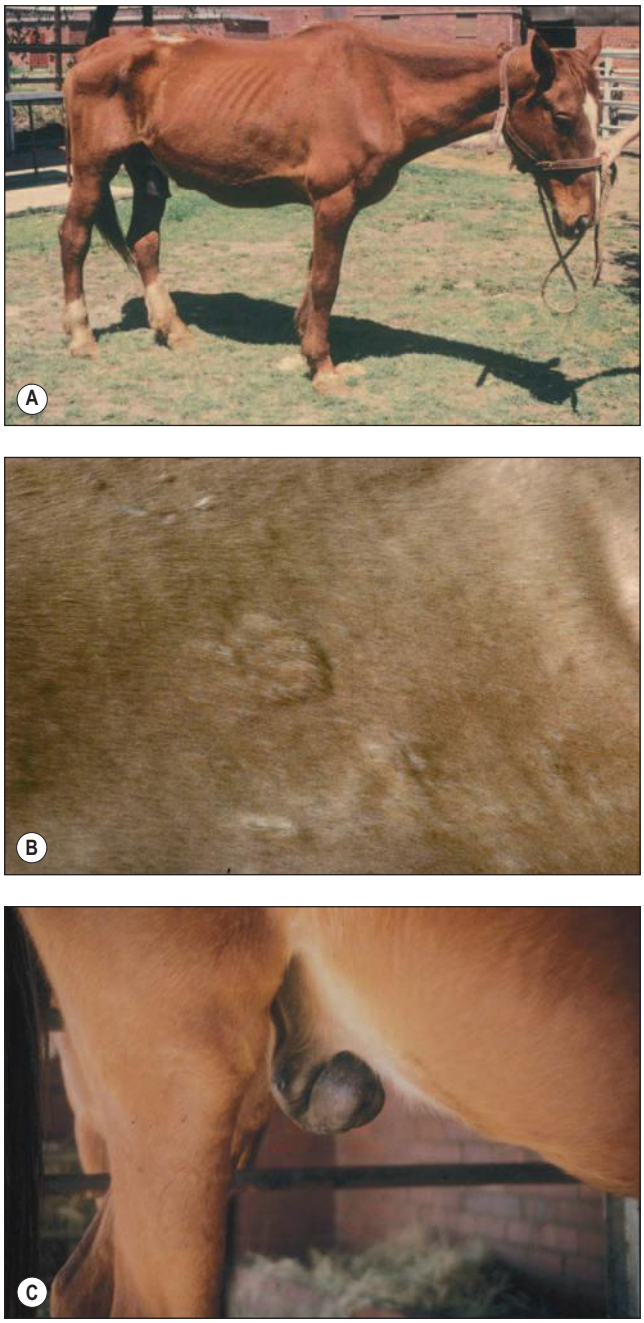


Figure 8.2 Dourine. **(A)** Chronic debility, weakness and lethargy are accompanied by preputial/penile oedema. **(B)** Characteristic (pathognomonic) 'silver dollar' dermal plaques (2–10 cm diameter) develop around 4 weeks after infected coitus. **(C)** Typical systemic and genital signs were obvious. (Figures courtesy of Dr J J van Heerden.)

CD

- Equine viral arteritis: transient viral infection with fever, peripheral oedema and positive serology; epidemiology is often characteristic.
- Lymphosarcoma: chronic or subacute debilitating neoplastic disease with cutaneous or lymph-node-related nodules. Seldom leukaemic.
- Purpura haemorrhagica: non-thrombocytopenic purpura; generalized ventral and dependent oedema with mucosal petechial haemorrhages following respiratory tract infection and possible central nervous signs.

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- Rabies: history of exposure/bite in endemic areas. Rapidly progressive hind-limb and other ascending neurological signs.
- Trauma of genitalia: signs limited to region and obvious evidence of localized injury.
- Other causes of chronic ill thrift and generalized oedema.

Diagnostic confirmation

- History and geographic location.
- Impression smears and sheath washings reveal characteristic *T. equiperdum* organism (characteristic movement can be seen with wet hanging drop preparation under low power).
- Inoculation of blood from infected horse into splenectomized or SCID mice.
- Complement fixation test (CFT).

Treatment

Homidium bromide, suramin and quinapyramine sulphate or diminazene aceturate can be used but all these carry inherent risks. Diminazene is poorly tolerated in donkeys and in many horses also. Isometamidium can be used to both treat and prevent dourine but the management of *T. equiperdum* is almost impossible because of the failure of the drugs to cross the blood–brain barrier and because treatment usually fails to sterilize the animal and so carrier status may be masked.

In most countries the disease has a statutory schedule status and affected animals are killed. Importation from endemic areas is strictly controlled.

Prevention

Eradication comprises control of breeding in endemic areas, quarantine and slaughter of infected horses. CFT is used to control importation of the disease into non-endemic areas.

Note

Horses, donkeys and mules with chronic infection with tsetse-fly (*Glossina* spp.)-borne trypanosomes such as *T. congolense*, *T. vivax* or *T. brucei* do have dermatological signs. These are trivial compared to the other symptoms associated with the infection. A harsh, dry coat with subcutaneous oedema and chronic or subacute debility with rapid death are common.

References

Bigalke R D, Prozesky L 1994 Besnoitiosis. In: Coetzer J A W, Thomson G R, Tustin R C (eds) Infectious diseases of livestock. Oxford University Press, Cape Town, pp 245–252

Davis W P, Peters D F, Dunstan R W 1997 Besnoitiosis in a miniature donkey. Veterinary Dermatology 8:139–143

Ramos-Vara J A, Ortiz-Santiago B, Segales J, Dunstan R W 1996 Cutaneous leishmaniasis in two horses. Veterinary Pathology 33:731–734

Roao N, Martins M J, Joao A, Campino L 2005 Equine infection with *Leishmania* in Portugal. Parasite 12:183–186

Robertson A 1976 Handbook on animal diseases in the tropics. British Veterinary Association, London

Terrel T G, Stookey J L 1973 *Besnoitia bennetti* in two Mexican burros. Veterinary Pathology 10:177–184

Van der Lugt J J, Stewart C G 1994 Leishmaniasis. In: Coetzer J A W, Thomson G R, Tustin R C (eds) Infectious diseases of livestock. Oxford University Press, Cape Town, pp 269–272

Van Heerden J, Els H J, Raubenheimer E J, Williams J H 1993 Besnoitiosis in a horse. Journal of the South African Veterinary Medical Association 64:92–95

Yashida E L A 1990 Human, canine and equine (*Equus caballus*) leishmaniasis due to *Leishmania braziliensis* in the southwest region of São Paulo State, Brazil. Mem Institute Oswaldo Cruz 85:133–136

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Metazoan/parasitic diseases

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Chorioptic mange (leg and tail mange)

Profile

This dermatological disease is caused by infestation with *Chorioptes equi* which primarily affects the distal limb regions but can extend to abdomen, axilla and groin. The parasites are scale-feeders and live on the surface of the skin. Adult mites can survive in bedding and stable floors for some months, especially if there is accumulated skin debris.

Mite populations are much higher over winter and signs are therefore more recognizable in housed over-wintered horses. This may relate to skin wetting, poor hygiene and the cooler environmental temperatures. The close proximity between housed horses in winter and the movement between stables of staff and tack and equipment means that epidemic infestation can occur in stables. The mites do not survive as well on sun-exposed pastures so populations tend to reduce markedly when horses are at pasture in summer. Carrier horses showing no clinical signs of infestation serve to perpetuate the infection from season to season. In pastured horses a few mites might be found, particularly in the heel bulb and coronary band regions – these cases may be a carrier source that perpetuates the infestation.

Heavy (draught) breeds with dense feathering on the legs are the most frequently affected but thin-skinned, non-feathered horses can be severely affected also – no horse in a challenging environment is exempt from the possibility of infestation. Apart from the tenuous relationship to heavy feathered breeds, there is no colour, sex or age predilection.

The condition is part of the verrucose pastern dermatitis (greasy heel syndrome (see p. 471). The disease is transmitted by direct and indirect contact. *Chorioptes equi* is possibly species-specific and certainly does not seem to infest in-contact humans, goats, sheep or cattle.

The parasites are easily recognized (see p. 46).

Key points: Chorioptic mange

5 6 12 15 18 22 23 24

1. Common cause of pruritus, worse in housed horses in winter months and commoner in feathered legs. The mites survive well in the stable environment especially where skin debris and dander are present and straw bedding is used. Carrier horses harbour a few mites around the distal limb over summer.
2. Usually many parasites (at varying stages and eggs) but clinical signs can be severe with only a few parasites. Pruritus not always proportional to severity of infestation!
3. Also affects body and neck/head and can be generalized, causing scaling and severe self-trauma. Carrier animals may show no signs.
4. Diagnosis is simple by examining brushings with magnifying glass or microscope.
5. Treatment involves repeated anti-parasitic washes, clipping of affected areas and stable hygiene. Important to treat all in-contact horses at the same time. Stable hygiene should be improved. Turn-out generally results in reduced mite numbers.

Clinical signs

Pruritus, irritation and restlessness with foot stamping and biting at legs are prominent early signs. Stabled horses in winter seem more liable to the infestation, with some remission over grazing periods. It is likely that some of these cases remain carriers, perpetuating the infestation within a stable yard. The affected horse seeks every opportunity to rub against or kick on posts, fences, concrete steps, walls and rails. Affected groups of horses can often be heard stamping their feet all night. Irritation is worse in warmer weather. Rubbing of the leg often causes the horse to bite or chew at the site (Fig. 9.1). Rubbing of the tail-base and face can also be encountered.

Patchy alopecia with broken hairs (possibly as result of rubbing and biting) and prominent scaling are common

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Figure 9.1 Chorioptic mange. This Shire horse mare bit her hind pastern in response to heavy infestation with *Chorioptes equi*. She persistently stamped her feet and rubbed them on the stable walls.

signs (Fig. 9.2). Exudation of serum with matting of leg hair, alopecia and scabs may develop over limited or extensive areas. Severe thickening of the skin and secondary bacterial infections arise from the long-standing, persistent self-inflicted trauma and this is one aspect of the verrucose pastern dermatitis syndrome that plagues many heavy draught breeds and the Shire and Clydesdale in particular (Fig. 9.1).

The severity of pruritus is not proportional to the severity of the infestation – some horses will rub a great deal with few mites while others with heavy burdens will show little (or possibly even no) pruritus. The condition is not restricted to horses with feathered limbs (Fig. 9.3).

Repeated episodes of self-inflicted damage can cause progressive thickening of the skin and exudation over the areas concerned. In some cases there may also be a degree of hypersensitivity to the mites. Secondary bacterial and fungal infections, fly strike (myiasis) can combine to produce a true verrucose pastern dermatitis (greasy heel) syndrome (see p. 471).

The head, neck and tail-base can be affected as part of the condition or on their own in a few cases. Whole-body (generalized) infestation results in a generally moth-eaten appearance with generalized pruritus, weight loss, irritability and poor exercise tolerance.

In-contact horses may be an important aspect of the clinical investigation both from a direct contagion viewpoint and because of the possibility that an individual may be a non-seasonally/persistently affected carrier animal. Straw bedding appears to be a significant exacerbating factor.

Differential diagnosis

- Atopy: persistent, variable location; mild to severe pruritus in the absence of any detectable cause in young horses.
- Insect bite hypersensitivity (including *Culicoides* spp. hypersensitivity/sweet itch): seasonally restricted and usually related to exposure circumstances with severe pruritus of neck and tail and less often ventral midline, head and ears.
- Dermatophilosis (*Dermatophilus congolensis*): not pruritic and usually obvious inflammatory lesions.





Figure 9.2 Chorioptic mange showing scaling **(A)** of the pastern region and ulceration **(B)** which was caused by self-trauma. This horse is the same as that shown in Figure 9.1.



Figure 9.3 Severe scaling of the distal limb regions in a Thoroughbred cross mare with a non-feathered leg. Note the extensive scaling and crusting with some areas of acute inflammation which were probably due to self-inflicted trauma. Profuse infestation was identified on her and on her stable companions even though they showed very little or no pruritus.

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- Dermatophytosis (*Trichophyton* spp.): only mildly pruritic and characteristic clinical progression and epidemiology.
- Trombiculidiasis (*Trombicula autumnalis*): mainly affects the limb and head regions; larval mites can be found on brushings or can be seen as orange-coloured moving parasites. Geographically limited to chalky soils.
- Sarcoptic mange (*Sarcoptes scabiei*): extremely rare condition usually restricted to immunocompromised horses. Extreme whole-body pruritus and mites identifiable in deep skin scrapings.
- Lice infestation (*Werneckiella* (*Damalinia*) *equi* and *Haematopinus* spp.): obvious pathognomonic macroparasites identified directly or on brushings.
- *Strongyloides westeri* and *Pelodera strongyloides* (parasitic) dermatitis: self-limiting condition affecting young foals, usually on wet/poor pasture; parasites can be identified in washings.
- Cattle tick (nymphal and larval stages): obvious localized visible parasites – not usually pruritic.
- Verrucose pastern dermatitis (greasy heel syndrome): restricted to distal limbs – more often white limbs; can be complicated by secondary parasitic infestations and bacteria.
- Pastern leukocytoclastic vasculitis: restricted, non-pruritic painful condition affecting white limbs only, initially lateral or medial aspects of cannon and pastern.
- Pastern folliculitis: painful, regionally restricted dermatitis.
- Mercurial and other blistering counter-irritants: exposure history, not pruritic.

Diagnostic confirmation

- Mites are easily recognized in groomings and scrapings from the affected regions – the ear margins are a good source of parasites in affected animals (see p. 46). They can usually be easily seen with a magnifying lens on a dark tile or under a low-power microscope.

Treatment

Careful clipping of long hair and removal of scabs followed by scrubbing of all affected areas with appropriate insecticidal shampoo or powder may be successful in reducing the numbers of mites and providing some relief of clinical signs. Organophosphates and chlorinated hydrocarbon insecticides are effective but are less available now.

Oral ivermectin paste (at 0.1 mg/kg daily for 7–10 days or 0.2 mg/kg twice at 2-weekly intervals) or moxidectin paste (at 0.4 mg/kg q 14 days) appears to help appreciably. However, this approach does not kill the mites – some do die but others are simply rendered less active and they lay fewer eggs. Therefore this alone does not cure the problem (Littlewood et al 1995). There is no advantage in injecting ivermectin over oral dosing – the intestinal absorption is rapid and provides the same blood concentrations very rapidly. There are significant dangers in injecting the drug into horses apart from its 'off label' use. Injectable doramectin show some promise as a therapeutic agent but again the effects are unreliable.

Topical washes of the affected horse and the in-contact animals are the main approach. Affected animals should be clipped out first (although in some cases this is anathema to the owners!). A preliminary warm wash with a selenium sulphide shampoo is a very helpful measure prior to the application of fipronil spray (FrontLine, Merial, UK) (see p. 93) or an anti-parasitic rinse. Application of a cat or dog flea collar impregnated with cypermethrin or permethrin is a very helpful approach (see p. 95) and provides a good measure of protection for some weeks. The collars should be tested on one limb first in case the animal shows a local reaction, and must be fitted with a locking buckle to avoid over-tightening and an elasticized section to prevent the collar 'snagging' (Fig. 9.4).



Figure 9.4 A cat flea collar impregnated with diazinon placed on the fetlock of a horse to control chorioptic mite reinfestation.

Stables where the disease is established show less response to treatments and isolation of infected horses from foals and yearlings should be considered. Mass treatment is useful in controlling large outbreaks or in stables where recurrent infections are common but reservoir hosts (and possible asymptomatic carriers) might make it impossible to eradicate in spite of the short viability of the mite off the host.

Psoroptic mange

Profile

This is a parasitic skin disease (body mange) primarily of young stabled horses caused by the non-burrowing scale- and fluid-eating mite *Psoroptes equi*. *Psoroptes* spp. mites do not appear to be particular about their hosts and so there may be fewer species than once was appreciated. However, it does appear that different feeding habits do occur and so this may be the main species differences. *Psoroptes hippotis* may be a separate species which may cause some cases of ear irritation and head-shaking. Older horses may become infected if infected younger horses are introduced into the stable. Mites can also be spread by head collars and rugs. Survival in a cool damp environment is possible for up to 2 months or more. Carrier status is not yet established but individual yards are recurrently affected. Degrees of hypersensitivity can occur also.

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Key points: Psoroptic mange

1 6 10 12 15 18 21 23 24

1. Uncommon cause of pruritus largely restricted to the head and ears and tail regions. There may be relatively few parasites.
2. Signs are mainly related to pruritus and localized skin thickening with hyperkeratosis. Severe excoriations can occur and this may be complicated by hypersensitivity reactions.
3. Diagnosis is by identification of the mite in ear debris and skin brushings and scrapings.
4. Treatment options are limited to topical anti-parasitic creams and washes and oral ivermectin or moxidectin. Treatment commonly has to be repeated frequently. Hygiene and avoidance of transfer to other horses is important.
5. It is worth checking other in-contact horses carefully. Once eliminated, the condition is relatively easy to avoid. A few horses become long-term carriers.



Figure 9.5 Psoroptic mange. *Psoroptes* spp. mites which caused extensive tail-rubbing and severe self-inflicted trauma. This was a severe case.

Clinical signs

Some affected horses show few or no signs at all but close inspection of the pinnal margin in particular may reveal thickening and hyperkeratosis. Obvious clinical signs attributed to the condition include head-shaking/rubbing and tail-rubbing. Moderate to severe pruritus mostly around the ears, mane, body and tail-head are non-specific but important signs (Fig. 9.5). Pruritus is more severe in warmer weather. Scaling (seborrhoea) and exudation are common, particularly along the margins of the ears along the mane and over the tail-base.



Figure 9.6 *Psoroptes hippotis*. Infestation of the skin of the ears causing a droopy, lop-eared posture. The margins of the ears showed extensive scaling and thickening.

Aural discharge, head-rubbing, head-shaking (with a side-to-side shake) and the ears held flat ('droopy' ears) are characteristic of *P. hippotis* (Fig. 9.6).

Tail-head infestation causes tail-rubbing with consequent poor hair density and broken and distorted tail hairs.

Irritability, particularly around the head, is often reported and some cases of head-shaking have been attributed to this infestation.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch): seasonally restricted pruritus affecting predominately the mane and tail regions but occasionally the ventral midline and head and ears.
- Chorioptic mange (*Chorioptes equi*): predominately affects the legs and head regions. Mites easily harvested by brushings.
- Lice (*Werneckiella* (*Damalinia*) *equi* and *Haematopinus asini*): easily recognized parasites in brushings.
- *Oxyuris equi* infestation: perineal/perianal pruritus only in unwormed horses.
- Stickfast flea, *Tyroglyphus* spp. mites: perineal and ear inflammation and irritation.
- Spinose ear tick (*Otobius megnini*), cattle tick larvae, other ticks: head-shaking, easily viewed auriscopically.
- Atopy (feed or inhaled allergy): non-seasonal, variable over body, onset in young horse.
- Conchal cyst: non-pruritic single discharging sinus tract on pinna or below.
- Idiopathic or photic head-shaking: characteristic neurological disease.
- Aural haematoma: very rare secondary event to shaking and rubbing.
- Over-strength insecticides: history of application – discomfort and inflammation localized, not pruritic.

Diagnostic confirmation

- Samples of ear wax or skin groomings can be examined with a hand lens on a dark tile – small white moving dots will be seen.
- Confirmation by microscopic identification of the mites (see p. 45).

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Note

Tranquillization may be necessary, as some affected horses resent ear handling. However, ear resentment is not a pathognomonic symptom.

Treatment

The general body and tail itch may respond to anti-parasitic shampoo containing organophosphate, chlorinated hydrocarbon or synthetic pyrethroid insecticides. It may be helpful to clip the areas affected before any washing. The effect of oral ivermectin or moxidectin is uncertain but it may be expected to have at least some effect. Injectable doramectin (off label) is untested but it could be effective. Fipronil is certainly worth a try.

Most horses resent any interference with their ears. Tranquillization may be required. All accumulated wax and debris should be cleaned from the ears. Where such products exist, proprietary canine anti-parasitic ear drops containing gamma benzene hexachloride, benzyl benzoate or synthetic pyrethroids can be used with good effect. However, some horses resent the drops more than the condition and can become permanently head-shy after even one or two applications. Direct applications of human pediculosis creams applied along the ear margins can be useful.

Treatment should be repeated in 10 days to ensure that any newly hatched mites are killed.

Sarcoptic mange

Key points: Sarcoptic mange



1. A very rare skin disease causing severe or extreme pruritus. Most affected horses are immunocompromised. Contact with affected pigs, small ruminants and humans can be a factor. Pruritus is a result of dermal burrowing and hypersensitivity responses.
2. Pruritus is usually severe, especially in compromised cases where generalized excoriations and severe and distressing itching occur. Severe damage and secondary skin infections are likely. Pruritus is worse if the skin is warmed by sunlight or by rugging.
3. Parasites can be identified in skin scrapings (seldom in brushings). Multiple skin scrapings will maximize the chance of finding the parasite, which is easily recognized. Biopsy may be lucky – a parasite or its track may be recognizable in the generally inflamed skin; it is often complicated by inflammation from self-trauma.
4. It is essential to address any underlying immunocompromising disorder at the outset. Treatment involves repeated anti-parasitic washes (lindane or malathion), clipping of affected areas and stable hygiene. Oral ivermectin or moxidectin may help and 'off label' doramectin injections can be justified. The source of the infestation should be sought.
5. The prognosis is poor and complicated by any underlying disease.

Profile

This is an uncommon, highly pruritic skin disease due to the burrowing mite *Sarcoptes scabiei*. Pruritus is a combined result of the burrowing mite and a hypersensitivity to its proteins. Transmission is by direct and indirect contact although the mite has a poor survival time off the host. It has been eradicated from large areas of the world and is now a rare disease, largely as a result of strict control measures. The removal of effective treatments makes it likely that it will reappear. The large majority of horses affected by this are immunocompromised in some way.

Contact with affected pigs and small ruminants may be a significant source of infection. Transmission to humans (and vice versa) is theoretically possible so it is worth asking if the handlers have any pruritic problems!

Clinical signs

This condition is characterized by intense, almost uncontrollable pruritus. Usually it is reported to start in one site but then rapidly spread to involve the whole body. Lesions frequently begin on the head, neck and ears and may then spread over the entire body (Fig. 9.7).

The earliest lesions are papules and vesicles on skin with intense pruritus. Rubbing and biting leads to progressive and extensive hair loss and excoriation with serum exudation/crusting and even bleeding. The skin may become very crusty and thickened (lichenified).

Secondary bacterial infections and myiasis can occur. Affected horses often have altered behaviour and temperament and may lose weight significantly, but some of these problems may be an underlying disorder.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch) or irritation from swarm insect attacks: characteristic regional pruritus occurring in seasonal patterns year on year; transient and better when avoiding possible contact with cause.
- Black-fly worry (*Simulium* spp.): bites are obvious and localized to head.
- Chorioptic mange (*Chorioptes equi*): localized, mild to moderate pruritus usually restricted to distal limbs and tail and head; parasites easily identified.
- Forage/scrub itch/chiggers/harvest mite irritation (*Trombicula* spp.): geographically limited, regional pruritus on legs and face. Parasites can be seen.
- Onchocerciasis (*Onchocerca cervicalis*) (particularly following ivermectin anthelmintic administration): localized mild pruritus in defined areas; geographically restricted.
- Larval cattle tick infestation: not pruritic – worry rather than pruritus, parasites obvious.
- Atopy (feed and inhalant allergy): young horse onset of mild to severe pruritus.

Diagnostic confirmation

- *Multiple deep* skin scrapings are usually required as there are often few parasites.

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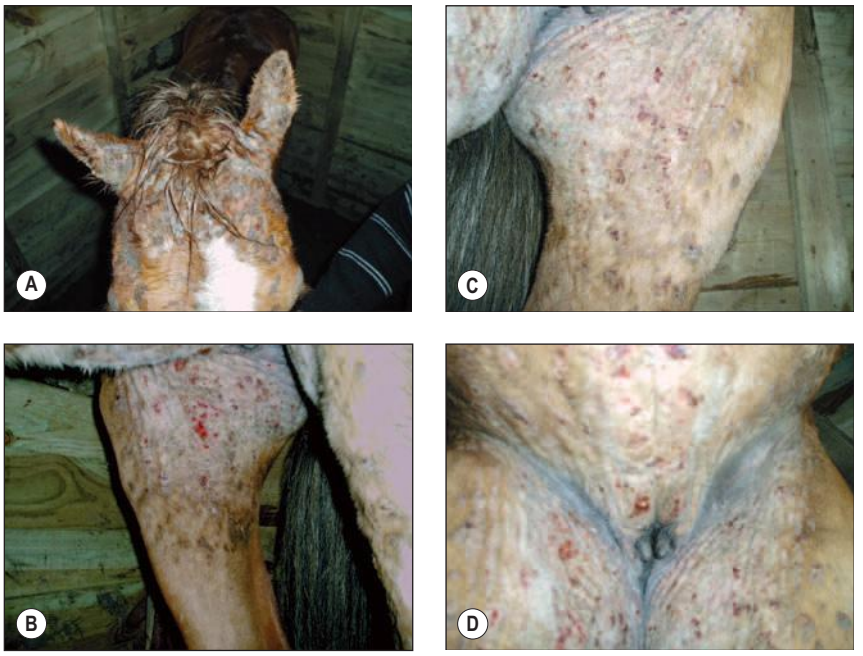


Figure 9.7 (A–D) This pony was presented with generalized severe pruritus, and *Sarcoptes scabiei* were identified in skin scrapings. Almost all affected horses are immunocompromised. (Figures courtesy of Dr Marianne Sloet.)

- Microscopic identification of *Sarcoptes scabiei* var. *equi* (see p. 44). A negative skin scraping may not be significant as the parasite is sometimes very difficult to locate.
- Biopsy of affected regions may reveal parasites or tracts but this is often complicated by inflammatory changes from self-trauma.

Treatment

Underlying imunocompromising disorders should be addressed – without this the condition will probably not respond usefully to any treatment.

Topical treatment with chlorinated hydrocarbons or organophosphate insecticides approved for use in horses has been the main approach but most such compounds are no longer commonly available. Malathion, lindane and organophosphate chemical washes may, however, be helpful where they are available. Oral ivermectin or moxidectin at normal dose rates repeated weekly for three doses may be effective. Injectable doramectin ('off label') should help. There is no information available on the therapeutic value and the risks associated with topical selamectin as used in dogs.

Washes containing synthetic pyrethroids are very variable in efficacy and usually fail to solve the problem. Similarly 2% lime sulphur has been advocated but it is unlikely to be effective on its own.

Repeated washes and dosing may be needed and it is important to treat in-contact horses at the same time even though these may not have any overt evidence of infestation.

Note

It is very important to remember that amitraz is toxic to horses and must not be used.

Relief from the pruritus may require tranquillization but this is a short-term expedient.

Corticosteroids will not help in the long term and may exacerbate the problem both in the short and long term.

The prognosis for affected horses is poor.

Control

The disease is reportable in some countries.

Mites can live up to 3 weeks in infested stables and fomites.

Early detection is critical if the condition is to be managed. Isolation of all infected horses and fumigation of all fomites, grooming materials, rugs and harness must be performed. Immediate prophylactic treatment of any potential in-contact horses and elimination of contacts with other species that may be involved may be wise.

Demodectic mange (demodicosis)

Profile

Demodex spp. mites are probably a commensal of the skin of all large animal species. The full life cycle is completed on the host and the parasites can only survive for a few hours at most away from the host. This means that spread between animals is difficult and slow (if at all) – it is best viewed as non-contagious.

The parasites only become clinically significant when the host animal is immunocompromised. Clinically this is a very uncommon disease even though mites can be demonstrated in apparently normal horses. *Demodex caballi* can be found in and around the eyelids and muzzle and *Demodex equi* from the body.

Clinical signs

The infestation is invariably asymptomatic in normal horses. Lowering of general body immunity by other conditions (such as debility, concurrent disease, stress or pituitary adenoma) may influence the likelihood of clinical signs developing.

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Key points: Demodectic mange

15 20 23 24

1. A rare skin infestation causing few symptoms. *Demodex* spp. are probably best regarded as a normal commensal (and opportunistic pathogen) of the skin of large animals. They may become pathogenic in immunocompromised horses.
2. Most cases are identified incidentally.
3. Parasites can be identified in skin scrapings (not in brushings) taken from the eyelids in particular. Multiple skin scrapings will maximize the chance of finding the parasite, which is very easily recognized. The parasite may be recognizable incidentally in biopsies taken for other reasons.
4. Treatment is not usually necessary – the role of regular avermectin anthelmintic doses is not established but this may reduce the overall prevalence still further.
5. The disease is incidental and seldom if ever significant except as part of an immunocompromise condition (stress, debility, steroid or lymphosarcoma).



Figure 9.8 This horse was severely immunocompromised and had evidence of liver failure. It exhibited persistent pruritus and pigmentary changes around the eyes. A deep scraping revealed copious live *Demodex* spp. mites. It was not certain that this was the cause of the problem, however. (Figure courtesy of Richard Frost.)

Patchy alopecia sometimes with scaling, papules and pustules particularly over the face, neck, shoulders and forelimbs may be associated with the condition and/or the underlying immunocompromise. Some cases of leukotrichia and (possibly) leukoderma around the eyes may be caused by this parasite (Fig. 9.8).

Differential diagnosis

- Other (bacterial) follicular dermatoses: identifiable organisms leading to a more distinctive panel of signs.
- *Onchocerca cervicalis* infestation: much more obvious signs.
- Vitiligo: characteristic asymptomatic loss of pigment around the eyes and face in particular.

Diagnostic confirmation

- Skin scrapings and biopsy are the only way to identify it. It is best to squeeze the skin firmly during the scraping procedure, which should continue until blood is drawn. The presence of large numbers of mites is suggestive of clinical disease while a single mite may not be significant.
- Biopsy may reveal the parasites (see p. 46).

Note

A positive skin scraping with characteristic clinical features is probably indicative of clinical disease but an alternative diagnosis should be sought unless the animal is immunocompromised.

Treatment

As the condition is usually asymptomatic, it may regress spontaneously. Usually it is not directly treated, though consideration should be given to reasons for impaired immunity and to the treatment of other concurrent disease.

Treatment with repeated avermectin and washes of an organophosphate may be curative if a genuine case is presented.

Note

Amitraz is commonly used to treat canine demodicosis (whether effectively or not) but this drug is very toxic to horses and MUST NOT be applied under any circumstances.

Regular oral doses of ivermectin or moxidectin probably will (gradually) remove the parasites and as this is a common practice now there may be far fewer horses with any mites at all.

Trombiculidiasis (scrub itch/chigger/harvest mite infestation)

Profile

Infestation with (*Neo*)*Trombicula* spp. ('harvest', 'chigger', forage, 'scrub itch' or 'heel bug' mites) is relatively common in defined geographical regions (usually associated with chalky and grassland soils) in late summer and autumn. Individual badly infested pastures are usually recognized and then avoidance of autumn grazing may be a useful (if often impractical) suggestion. The adult mites are free-living and feed on plants or invertebrates but the red-orange-coloured larvae require tissue fluids, normally derived from rodents and incidentally from horses and humans. The mites are only on the horse for short periods to feed and so even in severe infestations they may be difficult to find. Clinical disease is associated only with infestation by the larval mite.

A second group of mites behaving in a similar way are the so-called forage mites. These are usually larger and heavier and often have a more hairy appearance. They inhabit straw bedding and so clinical signs are restricted to housed horses. The individual behaviour of these opportunistic parasites varies and so the patterns of disease associated with them are unpredictable. Some are surface

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Key points: Trombiculidiasis

1 5 11 15 18 22 23 24

1. A common, usually transient, seasonal pruritic skin infestation of pastured horses. Regional differences occur in the species involved and the circumstances but most are restricted to chalky soils. Forage mites tend to affect stabled horses – especially those on straw bedding.
2. Mild to severe regionally restricted pruritus under defined management conditions. Usually affects the head and legs more severely but can involve the whole body. Changes in management usually improve matters.
3. Larval *Trombicula* spp. parasites can be identified directly as yellow-red or orange dots on the skin. Forage mite species are much larger and usually have a hairy appearance.
4. Treatment is helpful – permethrin washes or fipronil sprays are helpful. Cat flea collars around the legs can help also. The value of avermectin anthelmintic doses is not established but they may reduce the overall prevalence.
5. The disease is incidental and only rarely significant except as part of an immunocompromise condition (stress, debility, steroid or lymphosarcoma).

feeders and others are simply irritants as they move around in the hair coat (usually of the distal limbs).

Tyroglyphus spp. mites occur only in grain and can incidentally affect horses.

Clinical signs

(*Neo*)*Trombicula autumnalis* infestation is usually restricted to horses at grass (especially those grazing on chalky soils).

Individual bites are papular, inflamed and exudative and may coalesce to give much wider areas of exudative dermatitis. The condition usually affects the head (nose and face) and the lower legs in late summer and autumn (Fig. 9.9). Repeated and severe biting at the lower legs results in small or larger hairless areas which may become obviously inflamed and exudative after 2–3 days (Fig. 9.10). Irritability, leg-stamping, nose-rubbing and head-shaking reflect the intensely pruritic nature of the infestation – some of the response may reflect a hypersensitivity. Small papules and wheals develop on distal limbs, nose, neck **(Fig. CD9 • 1A–C)** and the ventral abdomen (where it can cause a form of ventral midline dermatitis). Exudation and superficial inflammation reflect both the local reactions to bites and the self-inflicted trauma. In some circumstances the whole body can be affected.

CD

Forage mite infestation will affect different parts of the body exposed to hay/straw and other forage sources. Some can affect the head and legs and others affect the body when the horse lies down. Overhead hay/straw storage may be a source of mites on the back. The mites tend to remain fairly localized so collection is easy but numbers may be low.

Differential diagnosis

- Poultry (red) mite (*Dermanyssus gallinae*) infestation: recognizable parasite, and chicken/bird contact is usual.
- *Stomoxys calcitrans* spp. irritation and bites: individual bites usually on the body and no residual attached parasite. The bite focus is usually dark purple in colour and very obvious.



Figure 9.9 Trombiculidiasis. Diffuse dermatitis of the face caused by *Trombicula autumnalis* infestation.



Figure 9.10 Trombiculidiasis. Extensive dermatitis which arose as a result of self-inflicted trauma due to severe and persistent trombiculid mite infestation. Note that the dermatitis was not restricted to the white areas.

- Onchocercal (microfilarial) dermatitis (*Onchocerca cervicalis*): chronic skin thickening and mildly pruritic. Geographically restricted.
- Chorioptic/Psoroptic mange (*Chorioptes equi*, *Psoroptes equi/hippotis*): distinctive parasites detected – may be present in addition.
- Tick larvae infestation: attached larger parasites; seldom pruritic.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): large parasites affecting the body trunk and neck mainly.
- Plant irritation (e.g. thistles): exposure related, transient, individuals involved – self-resolving.
- Contact sensitivity/allergy: exposure related, individual horse involved.

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Diagnostic confirmation

- Skin brushings are diagnostic – sometimes several different parasites are identified.
- As the mites fall off the host after feeding they are sometimes difficult to find, but aggregations of the red-orange mites feeding at one site make it easier to find on some cases. Individual lesions may be recognizable under a magnifying glass as a papule with a bright red/orange parasite in the centre.
- Extensive examination of groomings may be needed at an early stage in the onset of clinical signs. The use of a dark tile is helpful to show up the orange-red nymphs, although some can be almost colourless (see p. 47).
- Forage mites are very obvious, being much larger and fatter.

Treatment

Horses usually lose the infestation quickly and so should only be treated if absolutely necessary. It is better to remove the source of infestation if possible or to move the horses to a non-infested pasture.

Topical applications of sulphur washes, and permethrin powders and washes are helpful. Regular spraying with fipronil may be useful. Attention must be paid to the possible source of infection.

Forage mites may live in preserved forage or bedding so infestation can sometimes be 'out-of-season' and affect different regions of the body.

Poultry (red) mite (*Dermanyssus gallinae*)

Profile

This pruritic dermatological disorder is due to cutaneous infestation with the poultry (red) mite *Dermanyssus gallinae*. Direct or indirect poultry contact is always present.

Key points: Poultry (red) mite

11 12 15 18 21 23 24

1. A pruritic condition due to incidental/opportunistic bites of a variety of poultry blood-feeding ectoparasites including (most often) *Dermanyssus gallinae*. Poultry or pigeon exposure is always involved.
2. Pruritus restricted to specific regions such as the back, face and legs is the usual presentation. Some individuals will be more/less affected than others.
3. The parasites are easily identified but they do not stay on the horse for long. Therefore extensive and repeated brushings are often required. Circumstantial evidence is often strong! The mites may be found in and on wooden rails used for perching.
4. Treatment is not usually necessary – but washes and dusting with a permethrin compound will help limit the effects. Washing any wooden roosting/nesting sites and eliminating or treating the chickens is also helpful.
5. Removal of the contact between the horses and the poultry is obligatory and curative but removal of the poultry can in fact make matters worse (at least in the short term) unless the environment is cleaned as well.

The mites are blood-feeders and localized hypersensitivity responses may be encountered.

Clinical signs

There is severe, often progressive, pruritus over the face and distal limbs initially but often extending to the whole body. Horses become irritable and stamp and bite at the body and legs (Fig. 9.11). Some cases cause severe excoriation – the extent of pruritus may be disproportionate to the number of mites present. Small pruritic skin papules and crusts may be present in areas readily accessible to the mite (legs, face and ventrum).

Differential diagnosis

- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): characteristic parasites easily identified in brushings.
- Chorioptic, sarcoptic or psoroptic mange (*Chorioptes equi*, *Sarcoptes scabiei*, *Psoroptes equi/hippotis*): characteristic parasites recognized – may coexist.
- Insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch) and irritation (ventral biting species in particular).
- *Stomoxys calcitrans* fly bites/irritation: single or few and bite sites are more severe and isolated.
- Forage/'scrub itch', harvest (*Trombicula* spp.) mites: restricted areas affected and mites characteristic on visual inspection and brushings.
- Larval tick infestation: usually miliary tiny larval or nymphal ticks present; irritational rather than pruritic but can be severe. Recognizable parasites on brushings.
- Larval nematode dermatitis (*Strongyloides westeri*).
- Food allergy/atopy: rare, persistent, mild-to-moderate shifting pruritus in young horse without any detectable cause.

Diagnostic confirmation

- Poultry contact is always involved. The mites and their eggs can be found in bedding and crevices in walls and on wooden poles and poultry perches (see p. 48).



Figure 9.11 This mare developed severe facial and body trunk pruritus as a result of exposure to poultry. Poultry mites do not prefer to live on the horse and so they may not be found at all. Only a few mites were identified after extensive brushings were taken.

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- Groomings show small, mobile, red nymphal mites which are very visible on a black tile. Microscopic identification of mites.

Note

The mites are not always present in high numbers and so may be difficult or impossible to find.

Circumstantial evidence is a strong diagnostic factor.

Treatment

Washing or spraying of the horse with an approved (licensed) pyrethroid or fipronil insecticide can be very effective but it is usually necessary to repeat all treatments every 7 days in summer and 10–14 days in winter.

Remove horses from proximity to poultry or remove poultry from the stable environment. The effect of such separation may not be obvious for up to 6 months or more as the mites are very robust and can survive for some time in crevices in walls and floors and in wooden rafters on which the chickens once roosted. Treatment of buildings and all equipment with an appropriate insecticide may be more difficult than it sounds. Treatment of the poultry is sometimes helpful but even a few mites will survive to perpetuate the problem. Complete elimination of poultry, pigeons and other roosting birds is essential.

Fleas (stickfast or stick-tight fleas)

Profile

Flea infestations can occur either as result of contact with flea-ridden cats, dogs, rodents and wild animals in the stable yard or through contact with poultry infested with the poultry flea *Echidnophaga gallinacea*. The poultry flea prefers poultry but can feed off livestock whilst the cat/dog fleas species are much less host selective. In spite of the sometimes overwhelming population, horses are seldom affected clinically. This may reflect a tolerance of the bites rather than the fact that fleas would not feed on horses. All fleas are blood-feeders and so there can be some clinical effects from high challenge.

Key points: Fleas

5 15 18 22 24

1. Fleas from farm stock and domestic pets as well as wild animals can affect horses because they are not host-specific. The poultry flea can also infest horses but prefers its own host. Sporadic outbreaks can occur but once established the fleas are extremely difficult to eliminate. Fleas remain attached to the skin for some hours and so they can sometimes be seen.
2. The signs are seldom obvious but localized foci of mild to intense pruritus can be identified usually after the flea has left the site. Examination of the animals and birds in contact is helpful. Sometimes the humans will also be bitten and this is strong epidemiological evidence.
3. Diagnosis can be made by identifying and capturing the fleas. A small bleb of petroleum jelly will prevent them from escaping! Flea dirt can be found by combing the hair in the affected regions. The dander is placed on damp blotting paper and observed for red discolouring of the blotting paper as the 'flea dirt' dissolves.
4. Anti-parasitic washes may be effective but their effects are short and so they may have to be repeated often.
5. Control is the solution – either a concerted effort to treat the animals and poultry and the environment together using controlling sprays such as imidocloprid or separation of the source animals/birds from the horses are the main options.

Clinical signs

Prominent pruritus and secondary alopecia and excoriation occur where fleas attach. The resulting localized dermatitis is often roughly circular.

Most often the eyelids, face, ears, chest, shoulders and limbs are affected. Fleas are visible to the naked eye and are firmly attached to the skin.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch) or irritation from swarm attack: characteristic relationship to outdoor living in summer months; localized to neck and tail-head or ventrum (or both).
- Chorioptic, psoroptic and other mange mites: leg and head pruritus with characteristic patterns of itch and detectable mites.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): visible lice and eggs. Generalized pruritus and moth-eaten hair coat.
- Other ectoparasites, e.g. spinose ear tick (*Otobius megnini*): usually identifiable.

Diagnostic confirmation

Visible adult fleas, with mouth parts buried in the skin, may be seen. Parasites remain attached for some time and so they can be seen with an illuminated magnifying glass on the skin surface. 'Harvesting' the fleas for diagnosis can be difficult; a small bleb of petroleum jelly can be applied and then a pair of tweezers can be used to grab the parasite and place it in formalin for microscopic identification.

Treatment

Topical washes with insecticidal shampoo, flea powders or aerosols (licensed synthetic pyrethroids, organophosphates and chlorinated hydrocarbons are effective). There is no information on the value of ivermectin but as the fleas are blood-feeders it is reasonable to suppose that this or moxidectin would be a useful addition.

The fleas are very hardy and so elimination can be a real problem. Sustained treatment of source animals/birds and their own environments or significant and strict separation of horses from source animals including poultry can be effective. Often, however, removal of the others species simply results in a bigger challenge to the remaining animals. Ongoing control measures including regular stable spraying with anti-flea aerosols such as imidocloprid may be helpful but there is no reported information on the likely benefits. Poultry farmers and advice organizations may be

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a good source of information on the latest effective control strategies.

Culicoides/mosquito irritation

Profile

The *Culicoides* family (also known as 'no-see-ums', midges or gnats) includes many hundreds of species each with its own geographical location and breeding/feeding habits and some have a host preference. Many geographically restricted species of mosquito and *Culicoides* attack horses. Individual species of *Culicoides* tend to attack different areas of the horse (Pascoe 1973). It is important epidemiologically and clinically to know what *Culicoides* spp. midges there are in any particular geographical region.

Apart from their nuisance effects both *Culicoides* spp. and mosquitoes (*Culex* spp., *Aedes* spp. and *Anopheles* spp.) have an important second role in the transmission of some significant viral diseases (including the viral encephalitides such as West Nile virus and African horse sickness) and some other infectious diseases (including those caused by *Onchocerca cervicalis* and *Leishmania* spp.). These transmissions often require specific species of *Culicoides*; for example African horse sickness is probably transmitted only by *Culicoides imicola*. Both *Culicoides* and mosquitoes are also involved in the hypersensitivity disorders known as insect bite hypersensitivity (sweet itch/Queensland itch) (see p. 284) (Fadok & Greiner 1990).

The majority of the significant insects are blood-feeders and their life cycle demands warmth, moisture and suitable vegetation conditions for the individual species, so they tend to be very seasonal in most parts of the world. Where this is so, the diseases they transmit and the specific primary effects tend also to be seasonal. Their size and fragility mean that they are most active in calm conditions (with wind speeds less than 6–7 km/h). The various species have preferred feeding times and preferred feeding sites on the host animals. Thus, some feed in mornings, others in evenings. Some are ventral feeding, while others prefer to feed on the dorsum of the host.

Note

All horses may be attacked by *Culicoides* spp. insects and show signs of irritation, which leads to restlessness and even pruritus as a result of the bites. This should not be considered to be synonymous with the condition affecting horses that have an inordinate allergic response to the bite of relatively few of the midges or mosquitoes (less commonly).

Under favourable climatic conditions enormous populations of the midges and mosquitoes are present and challenged horses usually show severe irritation during these wave attacks. The irritation rapidly abates as the population of insects falls, leaving only those horses with hypersensitivity as a serious clinical problem. It is important to remember that horses can develop hypersensitivity responses to any mosquito or midge (or indeed any other biting insect).

Key points: *Culicoides*/mosquito irritation

5 15 18 22 24

1. This is a worldwide problem relating to the bites of *Culicoides* and other mosquito species. The condition results in a painful, papular and pruritic skin disorder and in some cases worry/behavioural changes. Swarm attacks are common but some individuals seem able to tolerate the multitudes of bites without any problem. The greater importance lies in the transmission of important diseases and to individuals that develop a hypersensitivity to the bites.
2. Swarm attacks cause more problems because of the large number of bites. Bites are restricted to the preferred feeding sites of the prevalent species. Individual bites are papular, pruritic and sometimes oedematous. Large numbers of bites in one location cause coalescent areas of erythema, pain and pruritus.
3. Diagnosis is usually simple. Known circumstances of exposure are almost impossible to avoid.
4. Avoidance by stabling or insect repellents can be effective to some extent. Severe attacks may warrant short-term systemic corticosteroid treatment. Localized bite sites may benefit from topical corticosteroids/antihistamines and local anaesthetic creams.
5. A relatively few cases develop progressive hypersensitivity problems (insect bite hypersensitivity). Eliminating populations of *Culicoides* is probably impossible but controlling numbers through traps, breeding site disruption and environmental management can help.

Clinical signs

Bites are followed immediately by short-term, localized pain, and pruritus develops within 2–12 hours. Papular erythematous foci with a surround area of oedema (wheals) develop but in many cases the skin changes are not really recognizable until self-trauma causes skin excoriation. The bite site itself is seldom visible with even a magnifying glass (but can be identified histologically) (cf. biting flies which leave an obvious central crust). Bites are usually restricted to defined anatomical sites of the preferred feeding habits of the species involved; the majority of species seem to affect the head, base of mane, the withers and tail. Ventral biting species bite the ventral abdomen or the upper medial limb regions (axillae/groin) and the distribution of the pruritus varies accordingly. Individual horses may be bitten mercilessly **(Fig. CD9 • 2A–C)**.

CD

Heavy attacks particularly by mosquitoes may precipitate an urticarial response over variable areas of the skin.

Secondary dermal bacterial or fungal infections can occur if self-trauma is significant.

In contrast to the allergic disorder which affects individuals, most or all of the horses in a group will usually be affected to some extent simply as a result of common exposure. A few individuals may not be affected in spite of the challenge of many bites. Self-inflicted trauma can cause moderate to severe superficial skin abrasions and excoriation with serum exudate (Fig. 9.12).

Individual horses may develop the allergic condition even after a single attack, thereafter remaining allergic and becoming progressively more affected in succeeding seasons. Some breeds are apparently more sensitive and others (such as the Icelandic and Welsh ponies and the Shire horse) are more inclined to develop an allergic reaction.

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Figure 9.12 *Culicoides* irritation. Acute inflammatory focus due to *Culicoides* spp. bites on the flank. The horse showed no sign of hypersensitivity. Similar intensely pruritic lesions are caused by mosquitoes and a few fly species.

The relationship between insect bites (of any type) and the common nodular skin disease 'collagen necrobiosis' or 'collagenolytic granuloma' (see p. 292) is not established. Some cases of this disorder have obvious tracts leading to the skin while others do not. The location of the nodules on the dorsum of the back and the sides of the neck tends to indicate that mosquito and *Culicoides* bites are at least less likely to be involved than other heavier biting insects.

Differential diagnosis

- Insect irritation, e.g. *Stomoxys calcitrans*, *Tabanus* spp., *Haematobius* spp., black-flies (*Simulium* spp.): this is virtually identical and the main variation will be in the size of the bite and the preferred feeding sites. The species may be identifiable if they can be caught/trapped. The bites from the larger flies tend to be bigger and deeper and commonly leave a small crust and a purple central spot at the site of a bite. A magnifying glass is usually needed to see them.
- Insect bite hypersensitivity (*Culicoides* spp. allergy/sweet itch): caused by the same insects but the response to a single or few bites is inordinate and much more persistent and rapid in onset.
- Dermatophilosis (*Dermatophilus congolensis*): more inflammatory and exudative, tends to be non-pruritic, slow onset and painful. Organism can be identified.
- Dermatophytosis (*Trichophyton equinum*, *Microsporum gypseum*): characteristic non-pruritic, self-limiting condition with characteristic epidemiology and mycology.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): obvious large parasites visible on horse and in groomings.
- Onchocerciasis (*Onchocerca cervicalis*): defined foci of mildly pruritic skin changes, long-lasting. Defined geographical and epidemiological circumstances in horses that have not received regular avermectin anthelmintic treatments.
- Contact hypersensitivity: defined exposure in one horse usually.
- Chemical irritation/scald/burn: defined history of exposure; non-pruritic.

Diagnostic confirmation

- Seasonal and environmental conditions are well recognized.
- Known presence of large numbers of *Culicoides* spp. or mosquitoes.
- Multiple horses are involved when the challenge by *Culicoides* midges is high. Individuals may become allergic to a few bites.
- Mixed populations of flies are also common so that it is usually impossible without trapping to establish the species involved. Use of a night-light trap or CO_2–octanol traps will allow identification of the species involved. The same traps can be used to reduce the populations of *Culicoides* midges significantly.

Treatment

Managemental avoidance of contact with *Culicoides* spp. and other mosquito insects is by far the best and simplest strategy where possible. Most *Culicoides* spp. feed during the morning and evening periods. Turning the horse out at night and in the heat of the day can be a really effective measure but most mosquitoes and some *Culicoides* spp. feed at night. *Culicoides* midges seldom enter buildings but some do and mosquitoes certainly do! The one thing they dislike most of all is wind – natural wind fans in the stable causing a wind of over 5 km/h are very helpful. Large fans in fields are often much appreciated by the horses, who quickly come to realize the benefit of standing in the draught. In some parts of the world where midges and flies are a real worry to horses, they may try to stand in roadways to make use of the wind of passing vehicles – this is an unwise manoeuvre for a horse!

Individuals with significant clinical problems may benefit from short-term systemic corticosteroid or antihistamine treatment. Local areas can be relieved by application of soothing lotions such as calamine lotion or steroid/antihistamine creams. Local anaesthetic sprays and creams can also help to reduce the self-trauma and distress. Usually the condition subsides quickly once further attack is prevented.

Use of permethrin and cypermethrin repellents or aromatic oils (such as citronella) and benzyl benzoate can be helpful but are not capable of preventing an attack completely. Overall body suits are available for grazing horses but they are expensive and tend to get damaged easily. Some horses are less tolerant of them. Some of these are impregnated with repellents. Suitable stable management and protective rugging, stables with fine mesh filters and overhead fans (to provide an air speed of over 5–10 km/h) are useful for individual horses.

Large groups of paddock horses may be protected by using smoky ('smudge') fires in and around the paddock. 'Midge traps' that emit a very low amount of CO_2 and octanol from a suction trap are highly effective in trapping millions of midges but they inevitably have a limited effect on the overall population. The more traps there are the better the effects will be!

Most *Culicoides* midges breed in water courses and in shady woodland areas and avoidance of these or

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Figure 9.13 This horse was suspected of having a *Culicoides* bite hypersensitivity (IBHS/sweet itch) and was totally covered with 'clothing'. Its feed was placed in the shaded area alongside a wooded stream. In fact the horse had to make a decision whether to eat and be plagued by midges or not to eat and have a comfortable skin. Removal of the horse to an open field meant that all the clothing could be removed – there was actually no hypersensitivity; the problem was simply a product of the numbers of midges worrying the horse!

controlling the flies within them by drainage and vegetation management, etc. can help significantly (Fig. 9.13). Often though it is simply best to move the horses well away from the danger areas during the seasons or times of the day when the midges are prevalent.

Ventral midline dermatitis

Profile

This is a specific insect-related dermatitis of grazing horses in summer months in fly- and *Culicoides*-infested areas. The condition is caused by the bites of a variety of insects including *Culicoides* spp., *Haematobia irritans*, *Stomoxys calcitrans* and *Tabanus* spp. Although one species may be prevalent the condition is usually caused by a mixture of insect species.

Key points: Ventral midline dermatitis

5 10 11 14 18 21 24

1. A recognized syndrome affecting younger horses predominately caused by fly or *Culicoides*/mosquito attack.
2. A highly pruritic ventral midline dermatitis develops soon after exposure. Pruritus may be intense and result in severe self-trauma, skin thickening, excoriation and secondary infection.
3. The diagnosis is simple but the cause is often more complicated.
4. Treatment is based on removal of the cause (either by management) or by use of effective fly repellents/and or clothing to prevent attack.
5. Prevention of attack is directed at limiting the contact by either reducing the population or preventing the insects from attacking (repellents and fans, etc.) or by avoidance such as moving the horse or stabling.

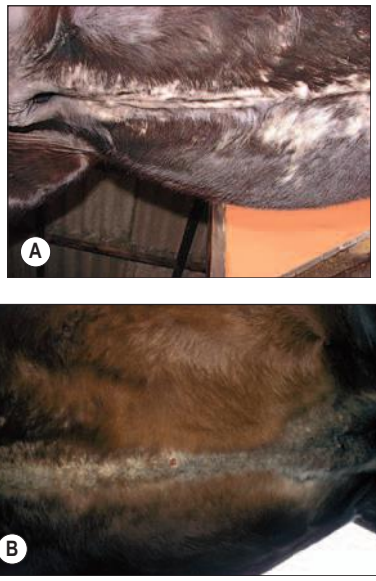


Figure 9.14 These two cases of ventral midline dermatitis started in June in the height of a wet warm period. The horses were kept outside and an abnormally large population of mosquitoes and midges was known to have been prevalent at the time. Fortunately the problem never progressed any further. Case **(A)** shows a relatively mild but more acute state while **(B)** shows longer-standing damage resulting in lichenification and hyperkeratosis.

The condition is commonest in younger horses (under 3–5 years of age); older horses seem much less prone to it.

Some individual cases develop a hypersensitivity to the bites of one or more of the species involved (not always *Culicoides* spp.) and then the condition can develop progressively year on year with increasing severity from minimal exposure (see insect bite hypersensitivity, p. 284).

Clinical signs

Ventral-biting species of fly, *Culicoides* midges and mosquitoes, bite the ventral abdomen along the midline (where the hair is less dense) and particularly around the umbilicus and area forward of the prepuce in the male horse. The bites from the major blood-feeding flies are larger and can have a small crust at the skin puncture site – this may be visible with a magnifying glass (but it is not always practical to look at the ventral midline of the horse so closely). A very high quality camera is a useful way to examine the ventral midline skin closely.

The bites leave sharply demarcated, inflamed areas which may be ulcerated, especially as a result of self-inflicted trauma. Individual bites may be visible but the numbers usually mean that there is coalescence. Pruritus varies from mild to extreme, with some horses showing bizarre behaviour and making frantic attempts to rub the ventral abdomen on troughs or fences. Bleeding and crusting with self-inflicted trauma causes skin thickening and alopecia (Fig. 9.14). The inflammatory response is often an attraction to surface-feeding flies and so the horse is also physically irritated. Localized leukoderma and leukotrichia and even long-term alopecia are common effects of the repeated local trauma **(Fig. CD9 • 3)**.

Usually most or all of the horses in a group will be affected to some extent but will vary markedly in their individual responses (by contrast to the allergic disorder,

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which invariably affects individuals severely from a single or few bites) **(Fig. CD9 • 4)**.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* spp. allergy/sweet itch): can be very similar in clinical presentation but the effects of swarm attack are short-lived and proportional to the number of bites.
- Onchocerciasis (*Onchocerca cervicalis*): an uncommon, geographically restricted condition in horses that have not had an avermectin worming programme.
- Dermatophilosis (*Dermatophilus congolensis*): seldom affects the ventral midline.
- Dermatophytosis (*Trichophyton equinum*, *Microsporum gypseum*): seldom affects the ventral midline and is much more restricted.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): seldom affects the ventral midline specifically.
- Poultry or trombiculid mite infestation of bedding: circumstantial evidence in housed horses with poultry; usually more restricted in extent and severity.
- Contact hypersensitivity: single horse usually and circumstantial evidence of contact – seldom restricted to ventral midline.
- Chemical irritation/scald/burn: single animals with an explicable cause such as urine scalding, wound exudate or known application of chemicals to defined regions.

Diagnostic confirmation

- Seasonal and environmental conditions are suggestive and several horses with a similar condition would be a strong support.
- Mixed populations of flies are also common so that it is usually impossible to establish the species involved without trapping and entomological classification.
- Biopsy of the lesions shows prominent eosinophilic infiltration and perivascular inflammation and small areas of superficial epidermal necrosis.

Treatment

Gentle washing and application of soothing creams (usually with corticosteroid) and prevention of further attacks are sensible measures.

Parenteral corticosteroids are sometimes required in severe cases.

Use of permethrin and cypermethrin repellents or aromatic oils (such as citronella) and benzyl benzoate can be helpful. However, in most circumstances it is almost impossible to prevent recurrent attacks. Thick applications of petroleum jelly or Stockholm tar are used along the ventral midline but make using and handling the horse unpleasant. Fly control measures can be used but are difficult when several species of flies are involved.

Suitable stable management and protective rugging and stables with fine mesh filters are useful for individual horses.

Large groups of paddock horses may be protected by using smoky ('smudge') fires in and around the paddock.

Gasterophilus spp. (bot fly) infestation

Profile

The genus *Gasterophilus* comprises several individual fly species that are significant in horses. These include *G. intestinalis*, *G. nasalis*, *G. haemorrhoidalis* and *G. pecorum*. The major concern for owners of affected horses usually relates to gastric or intestinal or (in the case of *G. pecorum*) the pharyngeal effects but there are significant dermatological implications.

The eggs are laid in characteristic sites for the species involved (Table 9.1 and see Chapter 3, p. 55).

The main dermatological effects, however, relate to skin penetration by the larval stages. The migration pathway is ill defined and the time taken can be very variable. Individual horses may be affected annually, often with increasing severity. This indicates that there may be some hypersensitivity responses involved. More information on the life cycle is given in Chapter 3.

Key points: *Gasterophilus* spp. infestation



1. *Gasterophilus* spp. flies lay their eggs in summer on the skin/hair. The larvae hatch and burrow into the stomach (or in the case of *G. pecorum* into the pharynx) using a tissue migration pathway originating in the mouth. Intestinal, enteric signs are usually absent.
2. The eggs can usually be seen on the hair of the legs, face and neck. Signs are variable. Some migration pathways are visible in the skin of the face as tortuous tracts that may have hair loss and thickening. Others cause focal, very 'punched-out', non-painful, non-pruritic ulcers on the lips, cheeks and gums. Gingival inflammation may be present.
3. Diagnosis is made on characteristic signs and the epidemiology. Clinically affected horses have not usually had avermectin anthelmintic treatment. Biopsy of the ulcers shows only a mild eosinophilic response. The larvae ('bots') can be seen in the faeces in springtime.
4. Treatment of the ulcers is by topical application of an avermectin paste and simultaneous dosing with oral ivermectin or moxidectin. Topical steroid cream is usually helpful but most ulcers resolve spontaneously in due course. The eggs can be artificially hatched by washing the legs with warm water or they can be scraped off the skin with a blunted knife or a safety razor.
5. Prevention is by regular worming in late winter to kill the gastric stages. Prevention of fly contact is difficult as the flies themselves are very aggressive.

Table 9.1 Characteristic egg-laying sites and larval development site for the various species of *Gasterophilus* (bots)

Species	Egg lay site	Larval site
G. intestinalis	Lower legs/shoulder	Stomach
G. nasalis	Intermandibular skin	Stomach
G. haemorrhoidalis	Face	Stomach
G. pecorum/*G. inermis*	Face	Pharynx

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Clinical signs

The eggs are attached to hair shafts often in large numbers (see **Fig. CD3 • 12A and B** and p. 55) and can usually be seen easily even when fewer in number (Fig. 9.15 and **Fig. CD9 • 5**).

The flies are very seasonal (summer and early autumn) and are somewhat geographically restricted. Some areas may be plagued by them while nearby there may be few or none, in areas where the flies exist. Horses at grass may be very upset by the flies and try to take avoidance action.

A relatively few horses have a hypersensitivity to the migrating larvae and a localized circular ulcerated lesion may develop in the skin around the mouth or on the lips and gums. Usually this is located in the skin of the upper lip or the mucosae of the upper lip (Fig. 9.16). The size of the lesion may vary but most are between 2 and 5 cm diameter. In some cases the area is more proliferative and appears like a granuloma. Quite commonly the lesions are bilateral and can be remarkably symmetrical. They are notable for their indolent clinical nature and refractory slow healing.

In *G. pecorum* infestation there is a major accumulation of the pupal stages in the pharynx where they can cause some difficulty with swallowing. Usually most affected horses are feral horses and ponies and so weight loss and even death may be overlooked.

Differential diagnosis

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- Foreign body granuloma (often from barley or grass awns): the ulcers are usually more inflamed and histological evidence of plant material can be found.
- Squamous cell carcinoma: carcinomas around the mouth region are usually highly aggressive and destructive. Biopsy is diagnostic.
- Basal cell carcinoma: ulcers can be very similar but biopsy is diagnostic. The tumour condition is progressive and persistent and is not seasonal.
- Sarcoid: not seasonal and seldom resolves spontaneously. Characteristic long course and usually other lesions of other sarcoid types are found elsewhere. Biopsy is diagnostic but the procedure carries risks.
- Onchocercosis (*Onchocerca cervicalis*): not common around the mouth and not an ulcerative condition. Geographically restricted.
- Epizootic lymphangitis (histoplasmosis; *H. farciminosum*): geographically restricted and usually large numbers of discharging ulcers in chains are found on the body, face, neck and limbs. Characteristically long disease course accompanied by debility and conjunctival/ophthalmic disease.



Figure 9.15 Eggs of the bot fly (*Gasterophilus* spp.) on the body **(A)** and leg **(B)**. Note that some species lay their eggs more diffusely than others and the location of the eggs is also species-related to some extent. Single isolated eggs also occur in some cases.



Figure 9.16 Typical ulcerated lesions on the lip and perioral lesions **(A, B)**. Note the lack of an inflammatory response and the almost indolent ulcer margin. Lesions also occur inside the mouth **(C)**. A short course of topical corticosteroid ointment usually brings a rapid resolution. Many resolve spontaneously but some remain static for months.

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- Melanoma (ulcerated): grey horses predominately, characteristic black coloration; not seasonal; usually multiple.

Diagnostic confirmation

- Seasonal and environmental conditions with known presence of the flies.
- Often bilateral and in conjunction with proliferative/ulcerative lesions on the buccal mucosa or gingival margins.
- Biopsy may reveal non-specific evidence of parasitic inflammation or may in some cases show the direct presence of parasitic larvae.

Treatment

Lesions associated with this condition are rare although many horses are infested. Whilst most lesions resolve spontaneously with time, others remain active – often for many months. Conservative approaches involve the administration of an avermectin wormer and topical application of the same paste to the lesion twice daily for 3 days. This can be followed by the application of a corticosteroid ointment for a few days. Usually resolution is slow, however. Where the tracts are seen in and under the skin of the face, avermectin therapy by mouth repeated at 14 days is probably effective. The difficulty comes when persistent alopecia, leukotrichia and thickening of the skin are seen in this cosmetically important location; fortunately this manifestation is extremely rare as these are probably untreatable. However, some do subside over months to years.

Where the conservative approach fails to resolve the problem, the lesion(s) can be surgically excised or subjected to cryosurgical necrosis.

Prevention of the condition can be achieved to some extent by fly control policies and by removing the eggs as far as possible when they are seen. This is achieved by warm water washing with soap and water and/or gentle scraping of the area with a blunted knife or similar.

Hypodermiasis (warble fly infestation)

Profile

Cutaneous nodules caused by migrating *Hypoderma bovis* and *H. lineatum* larvae (ox warble). The parasites are largely bovine but occasionaly horses are affected. The disease is restricted geographically to the northern hemisphere in areas where control measures for the disease in cattle and the adult fly are not in operation.

Younger horses and those in poor body condition are more likely to be affected. The parasites are bovine adapted and so, while they may cause disease in horses, they seldom mature fully. Extremely rarely aberrant migration can cause significant and unpredictable effects in the central nervous system or elsewhere.

The fly itself is a large and aggressive insect and females lay their eggs on the hair mainly on the legs. Single eggs indicate infestation by *H. bovis* while multiple piles of eggs indicate *H. lineatum*. Following hatching the larvae burrow through the skin (usually without any clinical effects) and over some months undergo a visceral migration to finally appear on the dorsal midline in the following spring.

Key points: Hypodermiasis

1 5 9 14/15 22 24 25

1. A predominately bovine parasite, geographically restricted to the northern hemisphere. The adult fly is large and the clinical manifestation is a cutaneous swelling on the back. Horses seldom have more than one or two maturing larvae.
2. Clinically the only evidence of the condition is a warm, firm skin swelling (2–3 cm diameter) along the dorsal midline. The nodules are painful on palpation. Only a few develop a small characteristic breathing pore (visible with a magnifying glass) – the majority fibrose and become a firm, painless cutaneous nodule.
3. Diagnosis is not easy because biopsy is probably not safe due to the risks of release of foreign proteins into the circulation.
4. Treatment is not usually undertaken apart from ensuring that an avermectin anthelmintic drug is administered. In some cases the pore can be enlarged and the larva removed. Alternatively the whole nodule can be surgically removed.
5. The condition is now extremely rare as a result of both bovine statutory control measures and the regular use of avermectin wormers.

The larval development results in a soft inflamed lump which eventually develops a small pore. After some 2 months the larvae leave the skin and pupate on the ground to complete the life cycle.

Clinical signs

There are seldom any signs during the skin penetration stage although a few horses may worry at the region for a day or two.

Early lesions are firm, well-demarcated, warm subcutaneous nodules along the dorsum (particularly in the withers region) in the spring months. Single or a few lesions are usually present but small aggregations may also be encountered. There may be some pain on palpation and saddle resentment.

Nodules develop a softer cyst-like structure with a small breathing 'pore' which may be visible and a few will produce a live larva (Fig. 9.17). Pruritus or more likely some resentment-worry may be present during the larval development. If the larva dies or is ruptured in situ, the horse may show a more severe reaction with inflammation and swelling. There is a theoretical possibility of an anaphylactic reaction but this has not been described in the literature.

Differential diagnosis

- Infectious granuloma/abscess: usually multiple and purulent; not restricted to midline back but can be a result of back injuries/penetrations, etc.
- Epidermoid and dermoid cyst: usually long-standing and in younger horses; can be inflamed in some cases.
- Neoplasia (mast cell, sarcoid or cutaneous lymphosarcoma): solid, usually non-painful and not restricted to dorsal midline; often others are present at other sites.
- Foreign body reaction: usually painful and obvious outer skin damage is commonly seen early.
- Staphylococcal folliculitis/abscess: not restricted to the focal area usually and very painful; progressive/spreading dermatitis; bacterial cultures are diagnostic.

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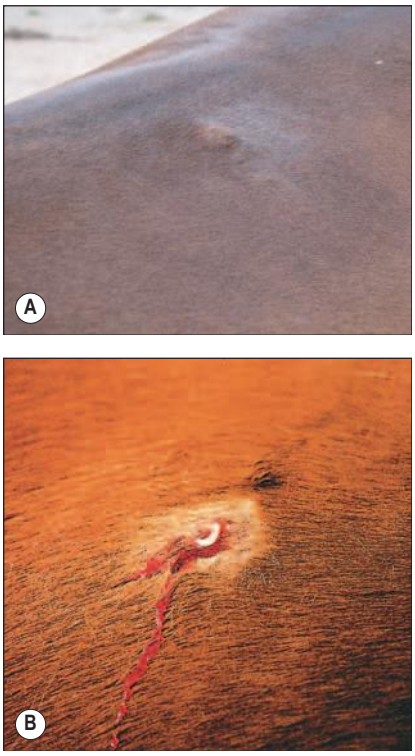


Figure 9.17 **(A)** *Hypoderma bovis* larvae under the skin producing an inflamed painful nodule. The nodule failed to mature into a larva but was identifiable using an 18 MHz ultrasound scanner. **(B)** This nodule produced a live larva.

- (Eosinophilic) collagen necrosis nodule.
- Panniculitis: rare inflammatory condition of subcutaneous fat; diffuse inflammation and pain.
- Eosinophilic granuloma (insect bite reaction): the nodules are usually multiple and variable in size and not restricted to the dorsal midline; they can be painful in some cases and they can be quite large; the small crust that is commonly found can resemble a pore but by this stage the nodules are small and hard.

Diagnostic confirmation

- Geographical location in winter months.
- Contact with affected cattle.
- Physical examination of nodular lesion with demonstrable breathing pores in skin or actual larva.
- High-frequency ultrasound examination is very useful – live *Hypoderma* spp. may be seen moving within the swelling.

Treatment

Medical treatment with ivermectin orally is effective (therefore in a horse where this is used routinely the condition probably will not occur). Topical application of organophosphate insecticides has been successful but these are not universally available. If the larva is in the later stages of development the skin can be opened and the grub removed.

There are anecdotal reports of anaphylaxis following drug-induced death of the parasite.

Surgical removal of the entire nodule is feasible but may leave a problem area on the saddle region.

The condition is related to the proximity to cattle, in which the condition is much more common and is largely subject to statutory control. It is now extremely rare in most countries where statutory control measures are in force for cattle.

Insect bites or stings

Profile

Many variations occur associated with single or multiple stings or bites from individual or swarms of insects. Marked geographical variations reflect the prevalence of the insects. It is not often possible to be certain of the cause unless the episode is observed, but bee stings are identifiable with careful examination of the sites for the remains of the stings.

The role of biting flies in the epidemiology of the equine sarcoid is uncertain, but it seems likely that they have some role in the transmission of the condition.

There is a distinct difference in the clinical implications of contact with venomous insects and non-venomous insects and between a bite and a sting (Tables 9.2 and 9.3).

- *A sting* involves the injection of a toxin of some sort that directly results in tissue damage, although this may be delayed in some circumstances.
- *A bite* usually does not inject any toxin although some insects do 'inject' saliva or an anticoagulant that enables them to feed effectively. This may cause some local irritation but more serious effects arise from hypersensitivity reactions.

The common biting and stinging insects include: *Tabanus* spp. (horse fly), *Stomoxys* spp. (stable fly), *Culicoides* spp. (midge, sand fly), *Haematobia* spp. (buffalo fly), *Simulium* spp. (black-flies), *Hippobosca* spp. (louse fly), *Phlebotomus* spp. (sandfly) and various varieties of wasps, mosquitoes, spiders and bees (see parasite and fly recognition, p. 49).

Horse flies (*Tabanus* spp.) (see p. 51) The life cycle varies from 3–4 months to 2 years. They are mechanical vectors of important viral and other infectious diseases (including equine infectious anaemia). They may also transmit

Table 9.2 Venomous and non-venomous insects

Venomous insects
Wasps
Hornets
Bees
Spiders
Non-venomous insects
Culicoides
Mosquitoes
Mites (all)
Fleas
Lice
Ticks

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Table 9.3 The common characteristics of stings and bites

	Itching	Pain	Allergic reactions	Swelling/inflammation
Venomous (stings)	Sometimes	Yes	Common	Intense Widespread locally
Non-venomous (bites)	Often	Rare	Occasional	Mild/absent Local only

Key points: Insect bites or stings

5 9 14/15 17/18 22 24

1 Insect bites are extremely common. Some are single and some are multiple and some involve swarm attacks (e.g. bees and wasps). Some species of fly lay their eggs in dead or inflamed tissues and maggot infestation can occur in wounds, etc. This is sometimes used medically under controlled conditions. Flies are more important as vectors of significant diseases including some skin conditions such as dermatophytosis, sarcoid, epizootic lymphangitis (histoplasmosis) and some systemic diseases such as trypanosomiasis. The larger flies also cause worry to horses either because of the sheer numbers or because they cause significant bite reactions.
2 Biting patterns and circumstantial exposure are usually well recognized. Each species has its own disease implication but most do induce a local nodular reaction and some degree of inflammation. Usually the bite site can be identified by close examination. Swarm attacks by *Simulium* spp., bees and wasps can cause severe and even fatal systemic reactions/anaphylaxis in some cases.
3 Diagnosis will depend on the circumstances – direct observation is helpful!
4 Treatment will vary from nothing to administration of full anaphylactic measures. Local bites by bees require different attention to wasps and black-flies so it is important to try to establish the cause. Local application and systemic corticosteroids are probably the mainstay of treatment.
5 Prevention may or may not be feasible using fly repellents, etc. However, they seldom last long enough and so some bites are inevitable. Fly fringes, face masks and clothing impregnated with insecticides or repellents are helpful in some circumstances.

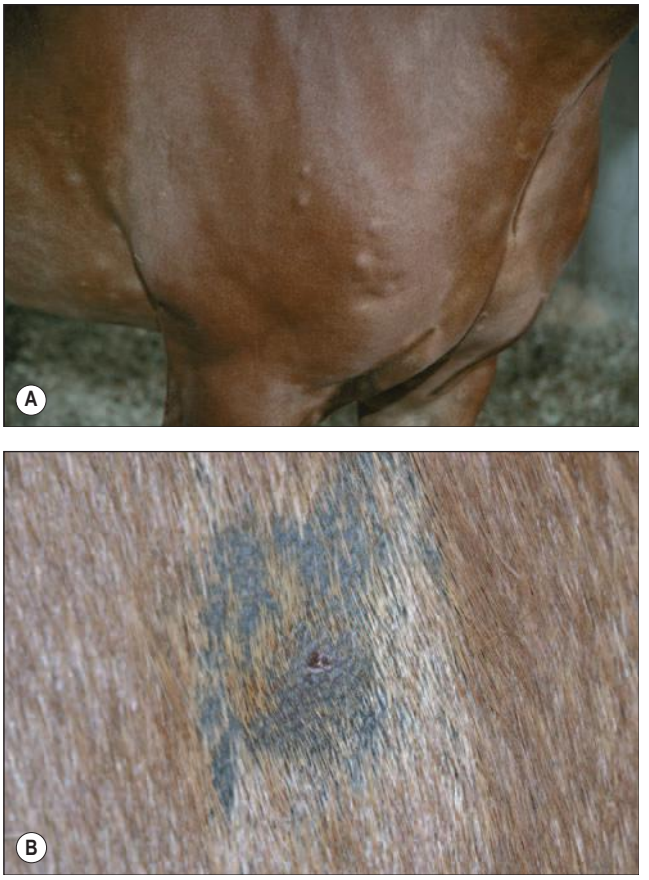


Figure 9.18 This horse was known to have been bitten some 30 minutes previously by several *Tabanus* flies and had been very disturbed by the small swarm of flies that attacked it while it was in an open covered yard. Significant oedematous skin nodules developed quickly **(A)**. The bite site was visible as a small haemorrhagic spot **(B)**. This will usually develop into a small focal crust and the swelling usually subsides within a day or two.

papillomaviruses, fungal infections (dermatophytosis due to *Microsporum gypseum* in particular) and possibly sarcoid. The bites produce a marked painful, pruritic skin response showing as wheals, with a central bite mark often with a blood spot (Fig. 9.18).

Stable fly (*Stomoxys calcitrans*) (see p. 50) Adult flies resemble the common house fly (*Musca domestica*), the bush fly (*M. vetustissima*) and the face fly (*M. autumnalis*). Large biting mouthparts cause considerable skin trauma and leave a relatively large lesion, often in a defined pattern of three or four bites in a chain or group (Fig. 9.19) when feeding is commonly disturbed by the panniculus reflex disturbances **(Fig. CD9 • 6)**. They are capable of mechanical transmission of diseases, including dermatophytosis (ringworm) (Pascoe & Connole 1974) (see Fig. 7.9).

Buffalo and horn fly (*Haematobia exigua, H. irritans*) (see p. 51) This occurs in subtropical/tropical areas. These flies are small (4 mm in length). Their identification on the horse is recognized by their head-down body position and they tend to feed in small or large groups, aggregating around the shoulders, neck, withers (Fig. 9.20), flanks and abdomen of the horse. Less commonly, they bite the legs (Fig. 9.21). Both male and female flies are blood-feeders, taking up to 20–30 meals daily. After feeding, the site often has a small drop of blood **(Fig. CD9 • 7)**.

Sandfly (*Phlebotomus* spp.) (see p. 53) The sandfly is a species that inhabits open sandy places near the sea such as dunes, and swarm attacks are common. They are very prolific breeders and can transmit some important human diseases including leishmaniasis and sandfly fever. However, there is little evidence that they transmit any equine diseases specifically. They do, however, cause major problems when a horse is attacked by swarm attack. Usually

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Figure 9.19 Stable fly (*Stomoxys calcitrans*) bites. Notice considerable skin reaction leaving a relatively large lesion, and distinctive patterning of bites.



Figure 9.20 Buffalo and horn fly (*Haematobia exigua*, *H. irritans*) aggregating around the neck and withers. Each bite site often has a small drop of blood.

myriad bites occur all over the body – the legs tend to be less affected. The bites produce cutaneous nodules which become coalescent simply by force of numbers (Fig. 9.22 and **Fig. CD9 • 8A and B**).

Note

The name 'sandfly' is erroneously given also to any biting fly that is found on or near a beach including *Culicoides* spp., mosquitoes and other flies.

Black-flies (*Simulium* spp.) (see p. 53) Larvae breed in running or still water in spring and summer and they can reach plague proportions following long rainy periods. They cause extreme irritation and inflict painful bites, particularly





Figure 9.21 (A) Persistent attack by *Haematobium* flies resulted in this extensive papular reaction. Each fresh bite has a small drop of fresh blood which dried to a crust. The reactions persisted for several days at each fresh site. Somewhat unusually the flies also bit the legs **(B)** and this disturbed the horse more than the body bites – rubbing and biting at the sites developed within 30 minutes.



Figure 9.22 This horse was taken to the seaside for a day's riding and was attacked voraciously for 3 hours by sandflies. The bites subsided over 48 hours but left areas of alopecia and some thickening of the skin for some months.

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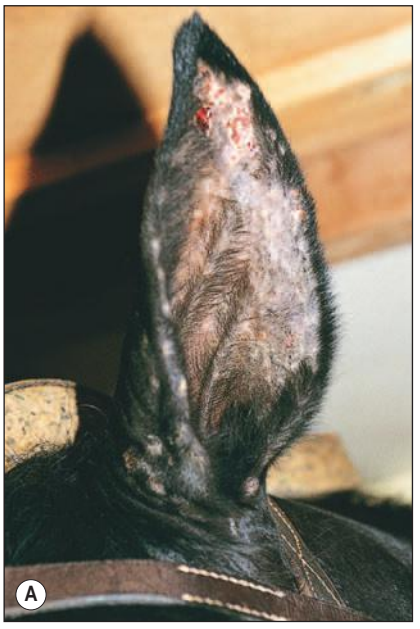




Figure 9.23 **(A)** Severe dermatitis on the pinnae caused by black-fly (*Simulium* spp.) bites. **(B)** Repeated attacks by *Simulium* flies caused skin thickening and pruritus.

on the ear flap and head (Fig. 9.23) and are most active in the morning and evening. There is a putative association with papillary acanthosis of the pinnae (aural plaques) (see Fig. 5.9). Swarm attacks by flies can induce death through release of a toxin capable of causing increased capillary permeability (Barbet et al 1991). Hypersensitivity may also occur with a dramatic generalized systemic and dermal response to a single or few bites.

Louse flies (*Hippobosca equina, H. maculata, H. rupifes*) (see p. 52) These occur in Europe, Africa and South America. The larvae develop in sheltered dry soil or humus. Adult flies are enthusiastic blood-feeders and tend to cluster in the perineum and inguinal regions (Fig. 9.24), causing significant lesions with persistent blood loss. They are not easily disturbed and cause local irritation and



Figure 9.24 *Hippobosca equina* congregating around the perineum. The flies are not dislodged by the tail movement of the horse and may remain in a feeding state for some days at a time. In this case the horse appeared to be unconcerned. Even physical hitting of the flies fails to dislodge them and they are not easy to kill either!



Figure 9.25 Louse flies (*Hippobosca equina*, *H. maculata*, *H. rupifes*) in the perineum. Note the severe hair loss from the tail as a result of persistent rubbing by the horse in an attempt to dislodge them.

fly-worry. They are also extremely difficult to kill by swatting – they almost seem to enjoy it! Affected horses are usually only mildly disturbed by even high numbers but the flies are voracious feeders and can cause local irritation

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Figure 9.26 Muscid flies attacking a fresh wound **(A)** and swarms of flies feeding on a chronic discharging wound **(B)**.

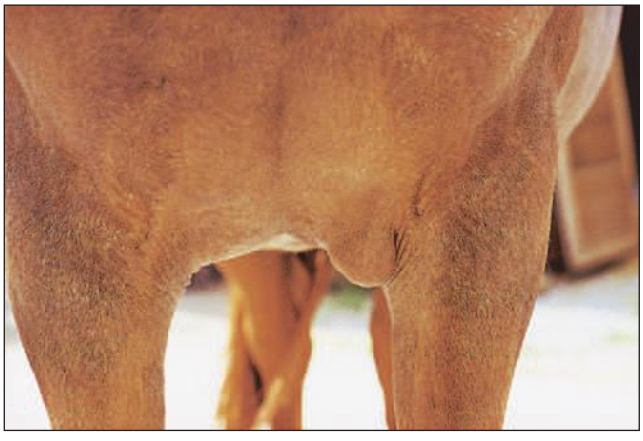


Figure 9.27 Single bee sting in the axilla, with swelling. The sting was identifiable and was removed easily.

manifest as tail-rubbing often to the point of completely denuding the hair on the tail (Fig. 9.25).

Muscid flies (*Musca* spp., *Hydrotea* spp.) (see p. 50) These are the common domestic flies (*M. domestica*, *M. autumnalis*, *M. sorbens*, etc.) that neither sting nor bite. They digest skin debris, exudate and tissue by regurgitating saliva onto the tissue and then ingesting the resulting 'soup'. These flies are a serious cause of worry and irritation to horses rather than causing any painful or inflammatory



Figure 9.28 This donkey was known to have disturbed a bee hive and received his due reward of many stings. The immediate effects were local swelling and generalized depression. Within 24 hours there was extensive local inflammation; moderate pruritus and skin sloughing developed over the area over the next 14 days. The immediate treatment included intravenous corticosteroids and mild topical alkaline solution of bicarbonate of soda and at least limited the damage and probably saved his life.

focus. However, in spite of their rather benign nature they do have a major significance in respect of transfer of bacteria and especially in the eye region and on wounds (Fig. 9.26). *M. domestica* plays a major part in the life cycle of *Habronema musca* (see p. 217).

Bees The honey bee (*Apis mellifera*) is a venomous insect and has been reported to attack horses in the spring and summer, producing oedematous wheals and plaques. Usually single stings are present (Fig. 9.27) although swarm attacks can occur particularly when the hive is disturbed and then the cutaneous and systemic consequences can be very serious (Fig. 9.28). The potency of bee venom varies between individual bees and also between subspecies in Europe, Africa and North America (Schumacher et al 1992, Staempfli & Kaushik 1993).

Wasps Various species of wasp inhabit and nest in shrubs, trees, brick and stone walls, etc. Accidental knocking of a nest usually precipitates a vicious attack by many wasps. Wasps are venomous and each wasp can sting several times and so multiple bite-site reactions can occur even from a single wasp; usually such bites are close to each other or at least on one body region. Swarm stings are correspondingly more extensive (Fig. 9.29) and can result in death.

Spiders Many spiders are highly venomous. Bites from black widow spider (*Ixeuticus robusta*) and white-tailed spider (*Lampona cylindrata*) and others in various parts of the world cause an acute, hot, extremely painful oedematous swelling. Some induce a prolonged necrotizing effect in the bite area, leading to skin sloughing (Fig. 9.30) and some can even kill.

Tick infestation Various ticks are found in many parts of the world; only *Otobius megnini* (the spinose ear tick) is a soft tick (Argasidae), the rest are hard ticks (i.e. they have a scutum) The biting/feeding sites vary with the various species and the bite site is the main dermatological significance. The large majority of the significant ticks are most active in summer and wet weather. The various species of ticks have

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their own feeding patterns – some are spring feeders while others will feed mostly in summer or autumn. Ticks are also classified according to their feeding/host relationship; one-host ticks remain on one host between the egg hatching and the mature egg-laying adult tick. Others require two or three hosts – the larvae and the nymph stages feeding on different individuals after a period of rest off the host. Although ticks do cause significant direct local damage and can be voracious blood-suckers to the point of inducing severe anaemia and hypoproteinaemia (see Fig. 9.32), they also have a major role in the transmission of significant protozoal and rickettsial diseases including babesiosis and *Ehrlichia equi* and can cause tick paralysis (Foil & Foil 1986). The various species also have different preferred feeding sites so that in any geographical area the local ticks are well recognized and can be found for example in the perineum, the ears, groin or distal limb regions. Initially the ticks are not very visible but as they feed they become much larger and the local reaction is more obvious too.

Marked local swellings (Fig. 9.31) and pain develop at the bite site and systemic reactions (fever, depression, central nervous signs, etc.) may be encountered. The presence of larval ticks (often in enormous numbers) can cause



Figure 9.29 Multiple isolated wasp stings. Each sting is very localized and close clipping will reveal a small but distinct central haemorrhagic focus. These bites are alkaline and so diluted vinegar is a good topical dressing in the initial stages.

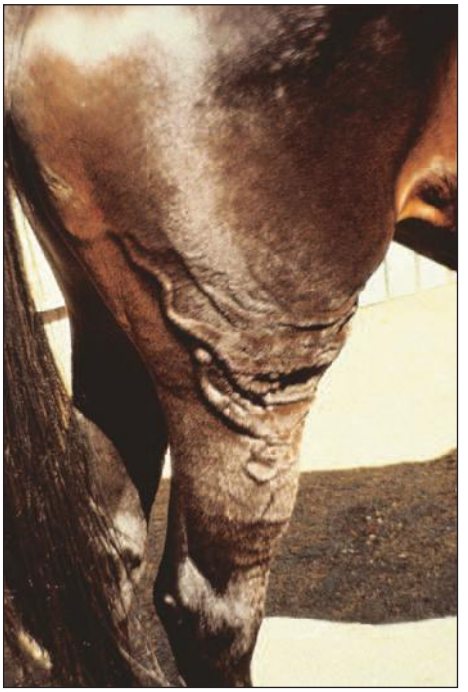


Figure 9.30 Spider bite with extensive local skin necrosis.

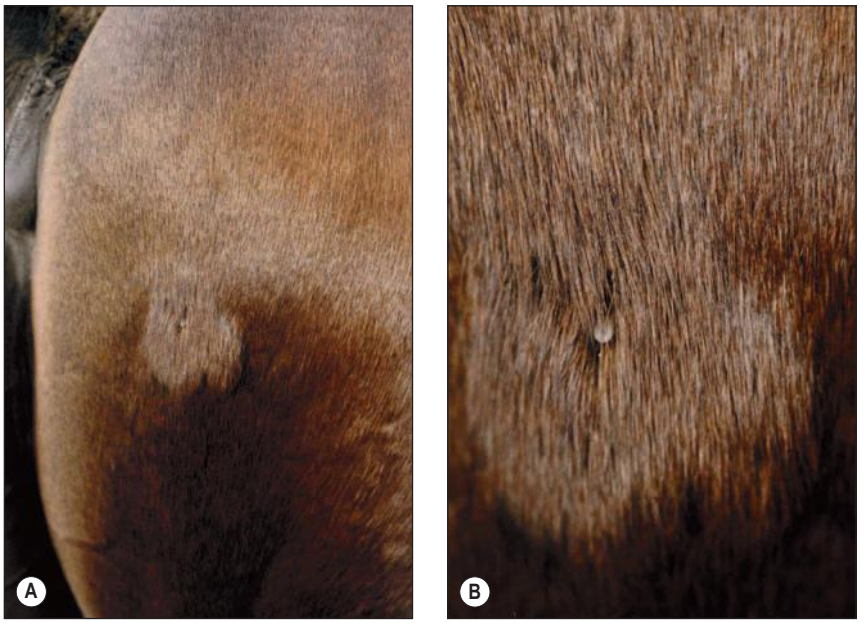


Figure 9.31 (A, B) This single tick bite site shows a typical oedematous response. This *Ixodes ricinus* tick was clearly visible.

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severe pruritus, with stamping and biting at the legs in particular. In spite of the numbers they can be very difficult to find on the horse as they drop off after feeding.

Large numbers of ticks can cause serious anaemia and even death (Fig. 9.32).

Otobius megnini is a soft (argasid) tick that prefers to inhabit the external ear canal and causes ear discomfort and otitis externa. *Boophilus microplus* appears to be liable to induce a hypersensitivity condition with local pruritus, and wheals (at the bite sites on the face and lower leg regions) and some generalized signs including angio-oedema and urticaria.

In countries where the problem is serious, regular insecticidal washes or grease dressings in the ears and perineum in particular are essential.

Note

Whilst amitraz is a common insecticide used in dogs and cattle for tick control this cannot be used on horses.

Clinical signs

The signs relating to the various biting and stinging insects vary enormously from mild worry to death! Direct toxicity, numbers of bites and the possibility of local or systemic hypersensitivity reactions contribute to the signs that can be seen in any individual horse. Both swarm (multiple) bite/stings and some individual bites can be fatal. The location and pattern of the bites can be suggestive of the species responsible. Individual insects such as spiders will cause single lesions for the most part.

Swarms or individual insects can be involved, with a corresponding number of bites. The nature of the bite and the potency of the venom influence the clinical appearance of the lesion(s). Severe attacks can cause severe systemic effects (cardiac and shock signs) or even death as in the case of swarm attack by *Simulium* spp. (black-flies) in eastern regions of Texas, USA.



Figure 9.32 This zebra was severely anaemic as a result of an enormous burden of internal parasites and ticks. The ticks were congregated in the perineum and the ears and most were *Rhipicephalus* spp. It is important to remember that ectoparasites can have serious consequences.

Biting flies and other insects produce a focal bite and/or some degree of worry or agitation in the horse. Most bites and stings have an obvious central bite mark surrounded by a small circular oedematous plaque but this may not be visible for the very small insects. The larger the insect, the larger the puncture and area of oedema, e.g. *Tabanus* spp. are large and may actually draw blood, leaving a visible puncture mark and a small blood spot (see Fig. 9.18), while *Culicoides* and mosquitoes have a microscopic bite-site focus.

Most bites cause local itching as opposed to pain but usually the difference is irrelevant at the outset. Stings involve toxin and so there is much more long-term reaction and more potential for systemic and local complications. Bites that cause pruritus usually do so immediately or within hours. Stings cause a local reaction instantly but then the tissue damage develops or the systemic signs begin.

Irritability from pain and/or annoyance causes foot-stamping, restlessness, nervous agitation and unpredictable movements. Some horses will actively seek out water, smoke or vegetation to avoid being bitten.

Differential diagnosis

- Urticaria: single animals involved with transient almost asymptomatic oedematous wheals of various shapes/sizes; no central focus is identified.
- Collagen granuloma: usually slow onset and long course/development; Biopsy is characteristic. A few cases can have a defined insect-bite like tract.
- Other causes of transient oedematous nodules with pain: the variety and number of bites make it almost impossible to differentiate from other causes of skin nodules.
- Death from swarm attacks may resemble anaphylaxis caused by other factors; skin lesions may not even be obvious if death is extremely rapid.

Diagnostic confirmation

- History can sometimes be helpful but most stings and bites are similar in appearance.
- Individual severity provides no helpful diagnostic information.
- Bee stings are usually recognized by the sting sac being visibly embedded in the skin but in many cases rubbing of the site causes this to deflate and become invisible.
- Biopsy of lesions is helpful and a characteristic inflamed tract can be identified. In some cases this can be found associated with collagen granuloma (see p. 292).

Treatment

The objective of treatment is to counteract the potential toxin in stings and to limit the inflammatory response in stings and bites. Therefore, for example, because bee stings are acidic, application of a mild alkaline solution

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such as 2% bicarbonate of soda can be helpful. Conversely the sting of wasps and spider bites are alkaline and so application of an acidic solution such as diluted vinegar may help.

It is important also to remove the causative agent whether that is a single bee sting stuck into the skin or an insect that is causing a problem.

The second objective is to counter the inflammation – this can be done locally when there is a single or few bites/stings using antihistamine or preferably corticosteroid topical creams or systemically using dexamethasone intravenously if there are severe and extensive reactions. For individual treatment antihistamine injections (tripelennamine and/or acepromazine) can be used.

Analgesia may be required topically using local anaesthetic sprays or creams; in some cases local application or systemic antibiotic is indicated.

Severe reactions to single or multiple stings/bites may warrant emergency adrenaline medication (3–5 mL subcutaneously of a 1:1000 dilution for a 500 kg horse). Intravenous fluid therapy, flunixin meglumine and corticosteroids may be required in addition.

For blood-feeding ectoparasites such as ticks, fleas and some mosquitoes, ivermectin or moxidectin can be useful. It can be administered orally – there is no advantage (and some significant risks) in injecting ivermectin into horses. Doramectin is also possibly useful. There is no established efficacy for this approach in horses, however. Regular dressing with various commercial tick washes and greases can help to reduce the burden of these parasites but again little is established scientifically and ticks remain a scourge in several species of animal in many parts of the world.

Control

Control of insect populations is extremely difficult and typically it is best to avoid the contacts! This is far easier said than done. Many insects cause no problems until they are disturbed and then there may be severe consequences. Others simply create a nuisance. In any case elimination of most of these parasites is impossible.

Manure disposal, slashing of long grass around stable area, removal of rotting vegetation and avoidance of cattle contact will make significant benefits in populations of flies in particular. Spraying stables, walls and yard rails with organophosphates or chlorinated hydrocarbons (where available) is helpful but should be avoided if possible on environmental and safety grounds.

Keeping the horses away from the sources of the insects is an effective measure for many of the worst species but is not generally feasible. Protection with meshed windows and a light insect trap inside stables at night may help. Insect repellents (most are synthetic pyrethrins) have a mixed reputation. They are usually only effective for up to a few hours after application and some are patently useless. The effects are further reduced if the horse is exposed to rain or constant wet periods and then treatment must be repeated after each wetting or it will be even less effective. Impregnated tags can be effective – these are commonly used in cattle where they are very effective. Horses appear to need several tags to be effective and they can be plaited into the forelock mane and tail or attached to head collars, tack or harness.

Note

No single control measure is totally effective for eliminating flies, mosquitoes and midges and therefore a coordinated control strategy involving as many of the feasible ones as possible should be carried out.

Myiasis (calliphorine and screw-worm myiasis)

Profile

The second dermatological implication of insects is infestation of tissue by fly larvae (myiasis).

Blow-fly strike (*Calliphora* spp., *Lucilia* spp., *Phormia* spp.). These are the green or bluebottle flies that are commonly seen feeding on neglected wounds and dead carcasses. They infest infected open wounds, neglected rigid limb casts, dirty bandages and necrotic neoplasms in particular. The flies lay their eggs on open tissue or on exudative inflamed skin, especially if the tissue is necrotic and fetid. Larvae usually hatch quickly (within 72–96 hours depending on species and conditions) and the larvae feed voraciously, growing rapidly for between 20 and 50 days. The larvae pupate on the ground and hatch into adult flies again. The fly population is governed largely by the environmental conditions but some animals seem more liable to the infestation. From a clinical perspective some species are more damaging and tend to be the first to arrive (*Phormia* spp. and *Lucilia* spp) and others tend to infest wounds that already have larvae and so exacerbate the problem (*Chrysomyia* spp. and *Sarcophaga* spp.). Muscid fly larvae may infest the resulting site but they tend to do little further harm.

Note

Several different species including *Lucilia* spp. (the green bottle fly) are being increasingly used as part of wound management systems (maggot therapy). The larvae are potent debriders regardless of the type of infection but do not specifically penetrate and destroy otherwise healthy tissue.

Screw-worm larvae (*Callitroga hominivorax* and *C. macellaria* in North and South America and *Chrysomyia bezziana* in sub-Saharan Africa) cause extensive tissue destruction; they do not digest necrotic tissue only and so the infestation is a significantly more serious disease. They do not breed in dead tissue and carcasses – they require fresh open tissue. Wounds and any inflamed and exudative skin can be infested; even the umbilical remnants and tick bite lesions can be enough to get them started. Eggs are laid at wound margins or at mucocutaneous junctions (including the eyelids, prepuce and vulvar lips). The larvae hatch very quickly (<24 hours) and spend around 7–10 days burrowing through healthy tissue as they mature. The destruction can be severe.

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Key points: Myiasis

5 18 19 21 24

1. Fly strike of two types occur. The first involves the larvae of species of fly that feed on necrotic tissue and exudate (and carcasses) and the second is caused by species that are adapted to survive on only fresh tissue. The larvae of the latter are highly destructive and invasive and destroy live tissue.
2. The infestations are usually obvious to a careful observer but the larvae of the destructive screw-worm group may be deep in the tissues. Usually infested wounds have a characteristic necrotic smell.
3. The diagnosis of the species involved is relatively simple from a few larvae preserved in formalin.
4. The first objective is to remove the invading larvae – this is done by irrigation and physical extraction. Insecticidal solutions and creams are effective also. A dilute solution of ivermectin has been useful – especially if accompanied by dilute hydrogen peroxide (5 vol w/v dilution).
5. Prevention of a new attack is important. Bandages and insecticidal creams can be used effectively. Petroleum jelly is useful. Wound care is critical but even in the best of circumstances there may be a few more maggots found.



Figure 9.33 This mare had an ulcerated sarcoid on her cheek that became infested with maggots – they were easily removed but because of the difficulty in covering the site she was infected several times over some months. Finally a dilute ivermectin solution and hydrogen peroxide resulted in a good improvement that allowed the sarcoid to be treated effectively.

Clinical signs

Sites of wounds (or plaster casts or dressings) show increasing malodour. The sites are irritating and the horse will often chew and lick at the region, as a result of physical irritation. The larvae are usually obvious on thorough exploration of the wound site (Fig. 9.33) but a casual glance may not be enough. All wounds should be thoroughly examined during the fly seasons.

The effects of the screw-worm larvae (*Callitroga hominivorax* and *Chrysomyia bezziana*) are particularly severe and may involve extensive tissue destruction and necrosis. The infestation is accompanied by a very unpleasant fetid odour. Self-trauma by biting at and rubbing of the infected site is common and this makes the site more inflamed and more extensive.

Differential diagnosis

- Exuberant granulation tissue (from any cause): maggot infestation may affect granulating but does not cause granulation tissue to develop.
- Botryomycosis (staphylococcal granuloma): no maggots are visible – lesions are painful and purulent.
- Habronemiasis (*Habronema musca*): this is a less obvious infection – the wounds simply remain open and exudative without obvious maggots.
- Squamous cell carcinoma: can be secondarily maggot infested, especially on the penis.
- Fibroblastic sarcoid transformation at a wound site: this can be infested with maggots but normally is distinctive.

Note

All these conditions are possibly subject to fly strike and, although they are not themselves pruritic, the presence of superimposed fly larvae can change this significantly.

Diagnostic confirmation

- Close examination of the wound. The larvae have an obvious breathing pore at one end. They are sometimes very deep within the granulation tissue and may be difficult to find at first.
- Identification of the species involved is sometimes helpful especially if there is extensive tissue destruction involved; this will help to determine if it is the condition that is destructive or whether screw-worm infestation is involved.

Treatment

The wound must be cleaned thoroughly even if this means removal of a rigid limb cast or dressings. The wound should be explored thoroughly in case any maggots are not visible. The wound should be flushed with high pressure saline to ensure debridement of all non-viable tissue. All visible larvae are lifted out. Insecticidal ointments and lotions (containing gamma benzene hexachloride or organophosphates) are available in some parts of the world and are useful in these cases. Ivermectin applied as a weak solution should be flushed into the wound alternating with 5 volumes hydrogen peroxide solution. It should not be necessary to repeat the procedure unless reinfestation occurs. Mixing a small amount of ivermectin in petroleum jelly or using a conventional oral worming paste is also a useful measure for both treatment and prevention. The wound should be kept covered and inspected regularly.

Oral ivermectin has little proven efficacy.

Pediculosis (louse infestation)

Profile

Lice are found worldwide and are host-specific obligate parasites and fall into two major groups:

- *Biting lice. Werneckiella* (*Damalinia*) *equi* is a small (<2 mm), not easily visible, pale yellowish scale-chewing louse with a broad flattened head (see p. 51).

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Key points: Pediculosis

5 6 10 12 16 18 21 24

1. This is a very common skin infestation of horses worldwide, mainly in winter months. Spread is by direct contact and malnourished or immunocompromised (e.g. pituitary pars intermedia dysfunction/equine Cushing's disease cases) or stressed horses are very liable to heavy infestations.
2. The main sign is persistent usually mild pruritus and irritability. The hair coat can become moth-eaten and damaged by self-trauma. Usually the lice and their eggs (nits) can be seen.
3. The eggs of both main species can be seen on the hair and the lice are visible to the naked eye. However the lice tend to hide under the hair when exposed to cooler conditions. Sometimes a rug is a better place to find them. Grooming almost always produce some recognizable lice.
4. Treatment involves anti-parasitic powders and washes and significant improvements in nutrition and management. It is important to treat all the in-contact horses at the same time and repeat any treatments at 21-day intervals to ensure a good kill-out. Oral ivermectin or moxidectin may help but this is a better approach for the blood-feeders than the scale-eaters.
5. Prevention is by persistent hygiene. Carry-over from year to year does occur so once it is present in a stable the condition can be difficult to eliminate completely.

- *Sucking lice. Haematopinus asini* is about 1–2 mm long and is a dark-coloured, blood-sucking louse with a narrow head (see p. 57).

The equine louse species are fairly host-specific but they can cause transient itchiness in human handlers and in dogs. Lice are particularly prevalent in winter and spring months when horses are congregated together and when the hair coat is dense and long. Shedding of hair reduces the population of lice significantly and sunshine is a strong inhibitor of replication. Spread is by direct contact or indirectly through grooming brushes, rugs and even via the stable environment, where they can survive for some weeks off the host animal.

The eggs are laid on the hair and can be easily recognized as 'nits'.

Clinical signs

The biting lice (*Werneckiella* (*Damalinia*) spp.) prefer the dorsum of the back and the sides of the neck while the blood-sucking lice can be found predominately on the neck, tail and limbs (Fig. 9.34).

Lice of all types cause pruritus, which produces irritation, rubbing and biting, dermatitis and unthriftiness with secondary patchy alopecia leading to a 'moth-eaten' appearance of the coat (Fig. 9.35).

Severe irritation and self-inflicted trauma can produce areas of dermal excoriation and serum exudation. Most obvious sites for the parasites are the side of the neck, the dorsum and the base of the mane and tail, where they can usually be found. These sites are the prime locations for rubbing and biting (Fig. 9.36).

Loss of condition is common in severely affected horses. Clinically significant anaemia can arise from even marginal burdens of the blood-sucking species. Animals that are stressed, immunocompromised or in poor condition (or combinations of these) seem more liable to developing heavy louse burdens.



Figure 9.34 The overall debilitated, anaemic and moth-eaten appearance of a donkey infested with *Haematopinus asini* lice. Note that this animal also had a discharging abscess that was thought to be a result of rubbing and biting at the skin. Close examination shows the extent of hair damage, and the small dark brown parasites are easily visible. Collection and microscopic examination will confirm the diagnosis.



Figure 9.35 Pediculosis. 'Moth-eaten' appearance of the coat of a horse infested with lice.

Differential diagnosis

- Chorioptic and psoroptic mange: parasites are not visible but distinctive under the microscope and tend to affect the legs and head rather than the body.
- Insect bite hypersensitivity (*Culicoides* spp. irritation/sweet itch): summer seasonal pruritus associated with exposure to *Culicoides* and other outdoor insects; affects the mane and tail predominantly.
- Dermatophilosis (*Dermatophilus congolensis*): not pruritic and skin lesions are more obvious; the bacterium can be identified in pus and scrapings.
- Trombiculosis (*Trombicula autumnalis*): geographically restricted and mainly the head/distal limb regions; obvious orange-coloured mites when present.

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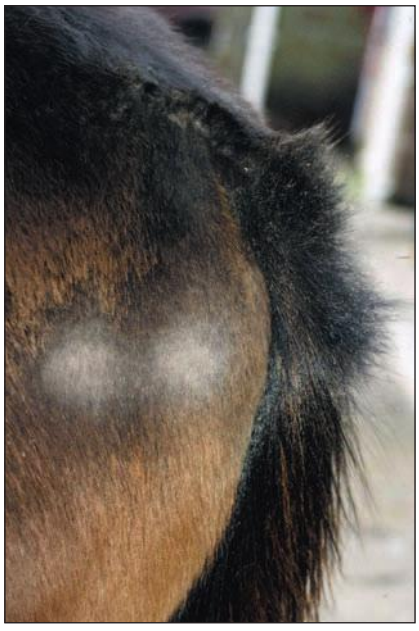


Figure 9.36 Pediculosis. Tail-rubbing caused by lice infestation. Note that the tail itself is less severely affected than the skin of the hind quarters.

- *Oxyuris equi* infestation and intermammary intertrigo (tail-rubbing): tail only affected and no parasites.
- *Dermanyssus gallinae/Tyroglyphus* spp. poultry mite infestation: exposure to poultry – may coexist.
- Trichophytosis and microsporosis (ringworm): not pruritic and self-limiting. Characteristic epidemiology and clinical features.
- Stickfast fleas, spinose ear tick (*Otobius megnini*): visible and few bites in isolated regions mainly on legs and head.
- Scalding (diarrhoea/urine/wound fluids, etc.): not pruritic.

Diagnostic confirmation

- *W.* (*D.*) *equi* is pale cream in colour and their eggs are easily visible. *Haematopinus asini* adults are dark brown in colour and somewhat smaller than *W.* (*D.*) *equi* and the eggs ('nits') can just be seen with the naked eye **(Fig. CD9 • 9)**. Warming of the skin with a hair dryer or light bulb will usually bring them to the surface and the horse will show a marked irritation with prominent scratch reflexes. Small numbers of lice and the smaller species of biting louse are more difficult to see.
- Groomings can be examined on a black or dark blue tile when the dark brown–red parasites will be seen to move. Microscopic examination will identify the species, which are noticeably different in appearance; *Werneckiella* (*Damalinia*) *equi* have a relatively broad body with a squarish head (see p. 57) while *Haematopinus* spp. have a longer, narrower body with a sharp, conical head (see p. 57).

Treatment

Whole-body washes or powdering with organophosphate or chlorinated hydrocarbon washes (where available and approved) are very effective. Aerosol insecticides containing organophosphates are still sometimes available for dogs and cats and have been used effectively, but some horses may resent the sound or may show dermal reactions. Fipronil has been used effectively but it is expensive for horses.

Pyrethroid louse powders and 'spot' treatments have an unpredictable and often disappointing efficacy. Treatments must be applied to all contact horses simultaneously and must be repeated at 10-day intervals. Oral ivermectin has a limited effect but 'pour-on' ivermectin preparations may be useful. All sprays and pour-on solutions must be applied strictly according to the manufacturer's instructions relating to dosage, concentration, repeat doses and concurrent drug therapy if chemical-induced irritations are to be avoided.

Pruritus usually ceases in 24–36 hours following successful treatment. Occasional horses show transient urticaria within 12 hours of pour-on medications.

Coat colour change and loss of hair has been reported after severe louse infestations (Arundel 1978).

Habronemiasis (summer sores/bursatti/kunkurs/swamp cancer)

Profile

Habronemiasis is a common, sporadic cause of ulcerating, cutaneous granulating nodules or wounds in geographical areas where the stomach nematodes *Habronema muscae*, *H. majus* and *Drachia* (*Habronema*) *megastoma* occur. It is

Key points: Habronemiasis

1 5 11 14 21/22 24

1 A common but highly seasonal cutaneous, conjunctival and wound-related helminth, *Habronema musca*, that is transmitted into open sites by surface-feeding flies. The condition affects the skin, conjunctiva and open skin wound sites during warm, wet periods. It is common in some parts of the world and less so in others. There is strong evidence that individual animals are more susceptible and that these will be affected year after year. Others in close proximity may not be affected at all.

2 *Conjunctival forms* result in chronic granulomatous conjunctivitis – often yellowish granules are seen either embedded in the conjunctiva of the nictitans or the palpebral conjunctiva or lying free in the conjunctival sac. *Wounds* affected with the parasite may not heal and are termed summer sores and remain open for many months until the fly season ends and then usually the resulting wounds heal rapidly. The *cutaneous form* characteristically affects the skin rostral to the medial canthus of the eye and the lacrimal duct. *Penile* forms occur on and around the glans penis and the urethral fossa.

3 Diagnosis is easily made in endemic areas but can be more difficult when it is an occasional disorder. Weather conditions and a non-healing wound region should alert the clinician. Conjunctival and lacrimal forms are not easily mistaken for anything else but histological examination of the nodules ('kunkers') may suggest mast cell 'tumour'. Diagnosis can be confirmed by low-power microscopic examination of washings taken from the surface of an infected site.

4 Treatment involves application of a very dilute ivermectin solution and then a steroid-based cream for a day or two. The conjunctival forms require more careful treatment.

5 Prevention is the primary objective for susceptible horses and donkeys; face protectors, fly repellent tags and brow bands and careful wound management are important. The facial form can be difficult because lacrimation wets the face and this attracts further flies.

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associated with increased fly population, poor manure collection or disposal and moist patches of long grass. The life cycle of the helminth is described on p. 58. The condition has no apparent breed, sex, colour or age predilection.

The condition is strongly seasonal, usually with spontaneous remission in autumn/winter; there is no evidence for chronic or subclinical infestations during winter months. Affected horses are often repeatedly infected in succeeding years, indicating a possible genetic susceptibility. Larval worms are capable of penetrating intact skin. Conjunctival, cutaneous and wound site infestations are recognized.

Clinical signs

The disease occurs in several different forms:

- *Cutaneous habronemiasis* occurs in three significant and recognizable forms.
  - a *Facial form*. Lacrimation and epiphora from any cause wets the facial skin and the resulting excoriated area is colonized by the parasite, causing a rapidly enlarging superficial, granulating skin ulcer (Fig. 9.37). Facial and ocular irritation results in rubbing and twitching. Repeated episodes cause facial scarring and secondary distortion of the eyelids is common **(Fig. CD9 • 10A–C)**.
  - b *Wound form* (associated with wounds). Wounds which become infested with the parasite fail to heal and may expand with production of unhealthy, exuberant granulation tissue (Fig. 9.38). Quite often there is a sanguineous exudate from the wound (this is not a common event in normal wounds). Whilst many induce proliferation of granulation tissue (often with accompanying necrosis and secondary infection) others appear to remain more indolent **(Fig. CD9 • 11)**. The wound may present as a static, saucer-shaped, shallow wound bed with little or no contraction or epithelialization (Fig. 9.39).

Chewing, biting and rubbing of wounds is very variable – this may not be genuine pruritus; some show severe pruritus and others are non-pruritic. A stringy serosanguineous exudate may be seen from some sores. Cheesy, yellow granules may be present on the surface of the wound and in the exudate but most are remarkably free of infection and exudate. Yellowish cream-coloured single or multiple merging granules may be seen within the wound bed; these are composed of necrotic cellular and sometimes calcified debris surrounding a dead larva (this may be an important diagnostic aid).

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Figure 9.37 Habronemiasis of the facial skin below the medial canthus of the eye of a horse **(A)** and a donkey **(B)**.



Figure 9.38 Habronemiasis. *Habronema* infestation of a wound on the cannon resulting in failure of healing. The parasites were visible in washings from the wound surface.



Figure 9.39 This pony suffered from a persistent, indolent shallow non-healing wound on the shoulder for 4 months during the wet season. *Habronema* larvae were easily harvested from a lavage fluid. It was managed simply by application of a mild steroid cream and daily lavage for 5 days.

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- *Penile form* (affecting the glans penis and the urethral fossa in particular). The urethral process can be affected, showing a granulating, ulcerated lesion on the glans penis (Fig. 9.40). Preputial and/or urinary bleeding may be seen or bleeding may occur during service, causing haemospermia which may reduce fertility. Discomfort can be shown during urination and the urine stream may be disturbed and spray over the legs (providing another potential site for infestation). Sometimes frank haemorrhage can be seen dripping from the prepuce.

- *Ophthalmic/conjunctival habronemiasis* includes a conjunctival form and a lacrimal form. Conjunctival involvement shows as yellow-cream-coloured, cheesy, gritty, granules (soft 'kunkers') or plaques on the palpebral conjunctiva (Fig. 9.41). On occasion there may be granular expansion of the tarsal glands. Involvement of the nasolacrimal duct/sac results in deep circular, granulating ulceration just below the medial canthus of the eye (see Fig. 9.37). Both forms cause epiphora (tear overflow) and lacrimation (excessive tear production) which may be bloodstained.



Figure 9.40 This gelding developed a severe *Habronema musca* infestation in the urethral fossa resulting in granulating, ulcerative posthitis. (Reproduced from Knottenbelt D K, Pascoe R R (1994) *Color Atlas of Diseases and Disorders of the Horse*, Mosby, Edinburgh; with permission.)



Figure 9.41 Conjunctival habronemiasis showing the typical yellow 'granules'.

Note

Habronema may be present secondarily to ocular forms of squamous cell carcinoma or to cutaneous pythiosis or other causes of non-healing wounds and so the combined alternatives should be carefully explored before treatment.

Habronemiasis can affect many other wound sites and so a secondary condition cannot be ruled out.

The effects of the adult worms in the stomach are negligible but some cases of gastric carcinoma are found to have significant infestations.

Differential diagnosis

- Exuberant granulation tissue/non-healing wounds (for any reason): granulation tissue may be very difficult to differentiate – the only difference might be that the *Habronema* site is flatter and more indolent and remains superficial; no larval involvement; biopsy is helpful.
- Squamous cell carcinoma (facial, penile, palpebral and nictitans forms): this tends to be more destructive and biopsy is characteristic; no larval involvement.
- Sarcoid (fibroblastic and mixed), particularly on the face and at wound sites on the limbs: biopsy is characteristic even though biopsy of sarcoid is generally viewed as potentially hazardous; no larval involvement.
- Phycomycosis/pythiosis (*Pythium* spp.): characteristic epidemiology and only at sites of wounds; no larval involvement.
- Botryomycosis (staphylococcal granuloma): biopsy is characteristic and is a single event; no larval involvement.
- Foreign body reactions: single localized event with no larval involvement.
- Mast cell tumour (conjunctival): this can be very similar and the two conditions are often confused.

Diagnostic confirmation

- Characteristic seasonal appearance in geographically endemic area (the parasite will occur in temperate climates under suitable conditions).
- Impression smears and fluid/washes from wounds examined under dissecting microscope or a simple magnifying glass will reveal the parasites. False negative smears are common.
- Biopsy may not be very helpful – non-specific eosinophilia with granulation tissue and intense vascularization is all that is found usually. Penile forms may have a more infected and granulating/necrotic mass.
- Biopsy of conjunctival forms may be very similar to mast cell tumours.

Treatment

Most lesions resolve spontaneously at the end of the summer months. Surgical removal or curettage is an effective method of treatment on granulating wounds. Oral

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or topical ivermectin suspended in an aqueous cream base is reportedly effective. Occasionally a second dose is required 3–4 weeks after the initial dose. Topical corticosteroids are sometimes helpful and this might imply that a degree of hypersensitivity is involved. Where a horse rubs and scratches the sites, topical local anaesthetic creams and sprays can be helpful in limiting the extent of the self-trauma.

Some *Habronema* lesions seem to respond well to cryotherapy, with a single or double freeze–thaw cycle usually being sufficient to kill the larvae, but this may be an oversevere approach.

Ophthalmic forms Oral ivermectin and/or corticosteroid eye drops. Lesions on the membrana nictitans (third eyelid) should be curetted and then treated with local application of cortisone drops plus a solution made up with 50% injectable form of ivermectin in artificial tears.

Control

Slashing of long grass in the vicinity of the stable and improved manure disposal will help to control muscid flies and therefore limit the number of cases. Careful attention to horses with a history of the condition is wise as repeated episodes are common and there is some suggestion that there are familial susceptibilities.

Parasitic/verminous dermatitis

Profile

These nematodes are facultative pathogens, being able to live freely in the environment. Larval forms of the nematodes *Pelodera strongyloides* and *Strongyloides westeri* may penetrate the skin (particularly of the distal limbs of young horses in wet, poor conditions) and have been associated with the dermatitis (Scott 1988a, Dewes & Townsend 1990).

Key points: Parasitic/verminous dermatitis

4 5 14 18 21 24

1. A rare dermatitis arising as result of larval penetration of the skin, particularly of the distal limbs (and the neck and sides if the animals are lying down) in young horses under poor wet conditions. Adult horses are only exceptionally affected.
2. Acute onset of moderate to severe pruritus can develop if there is exposure to large numbers of parasites. Adults may show mild pruritic pastern dermatitis. Penetration sites develop as papules and pustules but usually self-trauma confuses the clinical and histological findings.
3. Diagnosis is usually made on epidemiology and the fact that whole groups of foals develop it in the same way at the same time. Lack of hygiene and lack of anthelmintic treatments (in mares and foals) are usually strong supporting features. Washings and faecal egg counts may help.
4. The condition is self-limiting but anthelmintic treatment and improvements in hygiene are also required.
5. Prevention is relatively simple but the required measures are not always possible.

Clinical signs

A diffuse dermatitis presents particularly involving the distal limbs of young foals. Usually the condition becomes significant where foals are held in old, well-used horse yards, or housed on contaminated bedding or in poor hygienic conditions with mares and foals congregated in contaminated, wet or boggy yards (Dewes & Townsend 1990).

There is a sudden onset of skin irritation and pruritus with stamping, irritability and biting at the legs (Fig. 9.42). Signs that can easily be mistaken for colic such as rolling and biting at legs and flanks can be seen. Interestingly, foals seem to suffer only single or few episodes, seemingly becoming insensitive or resistant to the parasite. Diffuse skin thickening and dermatitis with limb oedema may be present in repeatedly challenged foals. It is reported to cause severe papules, pustules, ulcers, crusts, alopecia and erythema of the limbs, ventral thorax and abdomen, but these represent unusually severe responses.

Some adult cases have been encountered as diffuse pruritic pastern dermatitis but in these cases bacterial infections are usually the main factor involved.

Differential diagnosis

- Ventral midline dermatitis (biting flies, *Culicoides* spp. irritation): usually adults or older horses under defined conditions; usually single sporadic cases.
- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch): sporadic seasonal, moderate to severe pruritus focused on the mane and tail or ventral midline.
- Chorioptic/psoroptic or sarcoptic mange: usually dorsal pruritus and mites are easily found; seldom affects young foals.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): obvious parasites in brushings or even visible on the horse as adults or eggs (nits) affecting mainly the body trunk.
- Forage mites (*Trombicula* spp.) or poultry (red) mite (*Dermanyssus gallinae*) infestation: adult horses mainly under defined contact conditions; characteristic parasites identified in brushings.



Figure 9.42 This foal was one of several that developed a severe pruritus involving all their distal limb regions. Movement of the foals to cleaner environment and warm water washes resolved the problem quickly.

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Diagnostic confirmation

Diagnostic confirmation is difficult.

- History of management deficits.
- Clinical signs may be suggestive but signs are often not apparently related to skin disease.
- Skin scrapings or washings. Parasite larvae may be seen on or may be recovered from mud washed off the limb into a plastic bag. Positive or negative results may be misleading.
- Skin biopsy shows perifolliculitis, folliculitis and furunculosis. Nematode segments may be present in hair follicles (Scott 1988b).
- Treatment or removal from the source of infection with subsequent resolution is probably diagnostic.

Treatment

Where severe cases occur, the animals should be washed (preferably in warm soapy water) and removed from the affected yards. Improvement may be rapid but full resolution may take over 3–4 weeks. Improvements in hygiene are clearly essential. Topical antibiotic and corticosteroid creams can be used in severe cases if necessary. The population of *Strongyloides westeri* and *Pelodera strongyloides* should be controlled as far as possible by anthelmintic treatments but as the parasite can be free-living, hygiene is the main issue.

Onchocercal dermatitis (onchocerciasis/microfilariasis)

Profile

There are several potential pathogens in the genus *Onchocerca* but only *Onchocerca cervicalis* is commonly recognized. This is an important pathogenic nematode of horses in many parts of the world. The life cycle of the parasite is described in Chapter 3 (p. 58).

The adult nematode remains in the ligamentum nuchae, the tendons of the limbs and, significantly, in the skin and bursae around the withers. The adults produce microfilariae that circulate in the blood. The microfilariae are sequestered into the cutaneous capillary beds, particularly in the ventral midline and the head and neck. The larvae can be found in the capillaries of the skin of normal horses but type 1 and type 3 hypersensitivity reactions appear in some horses affected with microfilariae of *Onchocerca* spp. (Schmidt et al 1985). The parasite is transmitted by *Culicoides* spp. insects and other biting flies. *Culicoides* spp. midges are the main vector – transferring the microfilariae from horse to horse.

There is no material correlation between the number of microfilariae present (or detected in skin specimens) and the severity of the signs that are shown.

Clinical disease is rare in horses under 2–3 years old. Cutaneous and ophthalmic signs are recognized and are usually long-standing and non-seasonal. This differentiates it from many other similar cutaneous conditions. Most of the signs are a result of the microfilariae but the adults may also cause significant pathology. *Onchocerca cervicalis* is also a potential case of fistulous withers and chronic neck pain associated with nuchal ligament inflammation. Some cases of uveitis have been ascribed to *Onchocerca cervicalis* microfilarial migration.

Key points: Onchocercal dermatitis

1 A worldwide cutaneous, ophthalmic and orthopaedic problem disease caused by either the adult *Onchocerca cervicalis* or the circulating microfilaria. The parasite is transmitted by *Culicoides* and possibly by other biting flies. Main signs are attributable to microfilarial death and hypersensitivity reactions.
2 Signs vary from acute necrotizing dermatitis to chronic hyperkeratotic lesions that are roughly circular in shape but may be more superficial and less well defined. Abscessation and fistulation of the withers bursa is a recognized problem associated with the adult parasites.
3 Diagnosis is by identification of the parasites on biopsy or other techniques such as blood smear examination. Response to avermectin wormers is an important aid – the condition does not occur when regular avermectin anthelmintic treatments are used.
4 Treatment is by avermectin administration. The death of larvae as result of treatment can cause significant local or systemic allergic reactions.
5 Prevention is by regular avermectin medication.

Clinical signs

Apart from the ophthalmic and orthopaedic signs, several important different cutaneous syndromes are recognized depending on the stage of the parasite that is involved.

Signs attributable to adult *O. cervicalis* Long-standing, slowly expanding, hot, painful cutaneous swellings particularly at or around the withers that may involve the withers bursa and fistulate onto the skin are the main presenting sign. This may represent an aberrant migration of the adult parasite. The condition is relatively common in some tropical and semitropical countries but little is known about this very common and commonly devastating condition. The adult worm can be identified within the necrotic area (Fig. 9.43). The lesion either fistulates eventually or more commonly is surgically opened to release a clear serosanguineous sterile fluid. If the adult worms are not removed the site remains a discharging, secondarily infected fistula **(Fig. CD9 • 12)**.

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Microfilarial effects Deposition of microfilariae in cutaneous capillary beds can result in acute or chronic skin changes. The live microfilariae appear to cause little problem. However, dead microfilariae, whether as a result of avermectin medication or from spontaneous death, may cause significant local changes (Mazzotti reaction) including local, almost circular inflamed areas of skin (Fig. 9.44) or even an acute necrotizing thrombotic lesion (Fig. 9.45) or the more classically described chronic 'bull's eye lesions' in which there is a localized area of alopecia, epidermal hyperplasia and flaking and scaling. These usually have a prominent raised margin.

Diffuse, seasonal (summer), dermatitis affecting the ventral midline, chest, withers, face and neck may be due to *Onchocerca cervicalis* microfilariae. More diffuse forms are commonly found on the face (Fig. 9.46) and lower limb

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Figure 9.43 This donkey presented with a long-standing hot and painful swelling of the withers bursa which fistulated and discharged for some weeks **(A)**. Careful examination identified the adult *Onchocerca* nematode **(B, C)**. Topical treatment with a dilute ivermectin solution and 2.5 vol hydrogen peroxide can result in healing over some weeks.



Figure 9.44 This acute inflamed, roughly circular area was one of several that developed within 48 hours of an ivermectin anthelmintic treatment. Microfilariae may be identified in biopsies taken from such sites.



regions (Fig. 9.47). Patchy alopecia with small coalescing papules and thickened, dry scaly skin on the forehead (and less usually the sides of the face, pectoral region and ventral midline (Fig. 9.48)) is the most common presentation. The ventral midline shows more consistent alopecia and poor hair regrowth with some leukotrichia (or melanotrichia) and leukoderma (or melanoderma) (Fig. 9.48).

The condition is commonly associated with significant pruritus; severe cases show marked itching and excoriation, leading to scab formation, dermal hyperplasia and hyperkeratosis (the so-called 'bull's eye' lesions on the head). Loss/thinning of mane hair can be encountered. Tail-rubbing is rarely shown.

Differential diagnosis

- Ventral midline dermatitis (biting flies *Culicoides* spp. irritation).
- Insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch): seasonal sporadic and characteristic signs associated with turn out/exposure to insects.

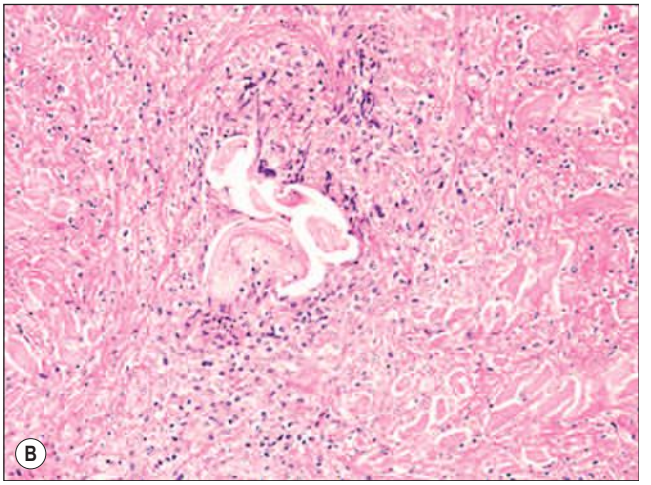


Figure 9.45 (A) This roughly circular necrotic plaque developed on the dorsal muzzle of a 5-year-old gelding following administration of its first avermectin wormer. **(B)** The biopsy specimen taken from the margin of the lesion shows a typical onchocercal microfilaria. This can be typed accurately by parasitological laboratories.

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- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): easily recognized parasites and eggs (nits) on skin and in skin brushings.
- Chorioptic/psoroptic/sarcoptic mange: parasites recognized in brushings and scrapings.
- Trombiculidiasis (*Trombicula* spp.): geographically restricted and easily recognized parasites mainly on distal limbs and head/face regions.
- *Boophilus microplus* infestation (cattle tick larvae): restricted geographically and site-related pruritus in perineum, ears.
- Nematode dermatitis (*Strongyloides westeri*, *Pelodera strongyloides*): foals only in restricted and defined circumstances.
- *Simulium* spp. (black-fly) irritation: ears and ventral midline, mainly seasonal attacks with obvious cutaneous bite marks.
- Unilateral papular dermatosis: restricted condition with spontaneous resolution – almost symptomless.
- Bacterial folliculitis (*Staphylococcal* spp.): painful dermatitis with exudate and identifiable bacteria.
- Occult sarcoid: biopsy may be necessary in some places; no response to avermectin treatments.



Figure 9.46 Onchocerciasis. Typical alopecic, hyperkeratotic, roughly circular facial and limb lesions of *Onchocerca cervicalis* infection.

Note

As both insect bite hypersensitivity (sweet itch) and onchocerciasis are involved with *Culicoides*, both conditions can exist at the same time.



Figure 9.48 Onchocerciasis. Chronic *Onchocerca cervicalis* lesions resulting from irritation and local reaction to the parasites in cutaneous capillaries in the ventral midline.





Figure 9.47 (A, B) These circular alopecic ('bull's eye') areas developed on the fetlock regions over 5 weeks following an acute local response that followed ivermectin administration. Biopsy confirmed the presence of necrotic microfilariae.

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Diagnostic confirmation

- Clinical features in regions where the parasite exists. Regresses in winter and returns in summer.
- History of poor worming regimens (routine use of ivermectin virtually precludes diagnosis).
- Response to ivermectin therapy over several weeks (exacerbation of clinical signs and development of focal suppurative or inflamed areas 24–96 hours after dosing is also supportive of the diagnosis).
- Jugular blood samples can contain microfilariae but this is very rarely so. Most are sequestered into the capillary beds and so a peripheral smear or collection of blood into a heparinized microcapillary tube from pin pricks on the ear flap, lip or eyelid or leg has a much better chance of harvesting a microfilaria. Spinning the sample in a centrifuge may concentrate the microfilaria in the buffy coat zone. Definitive confirmation of *O. cervicalis* microfilariae requires sophisticated laboratory testing.
- Biopsy can reveal microfilariae in sections (see Fig. 9.45B) but may be incidental.
- Concentration of larvae using (minced) biopsy material may also help (but can be misleading also).

Treatment

Oral ivermectin or moxidectin is extremely effective against the microfilariae but not probably against the adult forms in tissues (Herd & Donham 1983). Daily dosage for 5 days with fenbendazole or mebendazole at twice the normal dose rate followed by single monthly 'maintenance' treatment is beneficial. Following administration of avermectin there may be a more florid cutaneous response for a few days and then a gradual subsidence of signs occurs over 2–4 months. This post-treatment reaction occurs in a high proportion of cases and is characterized by oedema and pruritus. A single dose may be able to prevent reinfection for up to 5 months (French et al 1988).

Levamisole at 10 mg/kg per os for 7–10 days may be a cheap and effective way of killing the microfilariae but may be accompanied by a more severe local reaction and possibly even anaphylactic reactions. Diethylcarbamazine at 1 mg/kg in feed daily for 21 days can be used but is not totally or invariably effective.

Note

Neither levamisole nor ivermectin should be administered by injection.

Therapy with ivermectin in clinically unaffected and affected horses may cause death of the microfilariae in the capillary beds of the skin. This can cause an acute exudative dermatitis with characteristic circular patterns over the whole body but most severe in the ventral midline and head regions.

Severe anaphylactic reactions are also possible.

Oxyuriasis (pinworm) infestation

Profile

Infestation with *Oxyuris equi* (pinworm) is relatively common in some regions. The prevalence of the worm has reduced markedly since the advent of the avermectin wormers.

Key points: Oxyuriasis

14 18 21 24

1. An increasingly rare cause of perianal pruritus due to colonic *Oxyuris equi* infestation.
2. Severe tail and anal rubbing with excoriation, breakage and matting of tail hair and distress are the main signs.
3. Diagnosis can be confirmed by identification of the typical oval operculate eggs harvested onto adhesive tape from the perianal skin.
4. Treatment with ivermectin or moxidectin is very effective.
5. Prevention is best through regular worming programmes – avermectins are not the only compounds that will control the worm.

The adult *Oxyuris equi* worms inhabit the caecum, colon and small colon. Female worms migrate down the small colon to lay eggs on the perineal skin. The main symptom of infestation is perianal pruritus in stabled horses and is attributable either to the activity of the female worms or to the sticky substance that the worm uses to attach the eggs to the skin.

Clinical signs

The adult worms cause moderate to severe anal and perianal irritation. Tail-rubbing causes an acute, excoriated, self-inflicted trauma to the perianal skin and tail-base.

Long-term infestation results in excoriation of the base of tail, with broken hairs giving a rat-tailed appearance (Fig. 9.49).

The horses are often restless and irritable and may lose weight as a result of the persistent worry.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch): seasonal pruritus of neck and tail-base mainly.
- Chorioptic or psoroptic mange: characteristic mites identifiable and pruritus is not restricted to the tail-base and perineum.
- Intermammary intertrigo: this can be remarkably similar clinically; easily eliminated by cleaning.
- Lice (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): easily seen lice and eggs – pruritus affects wider areas also.
- Dermatophilosis (*Dermatophilus congolensis*): not pruritic in most cases.
- Atopy: idiopathic pruritus over whole body or varying regions without any identifiable cause in young horses.

Diagnostic confirmation

- Poorly wormed, stabled horses most often affected.
- Examination of perineal area and identification of characteristic triangular operculate eggs from perineal skin using adhesive tape technique (see p. 59).

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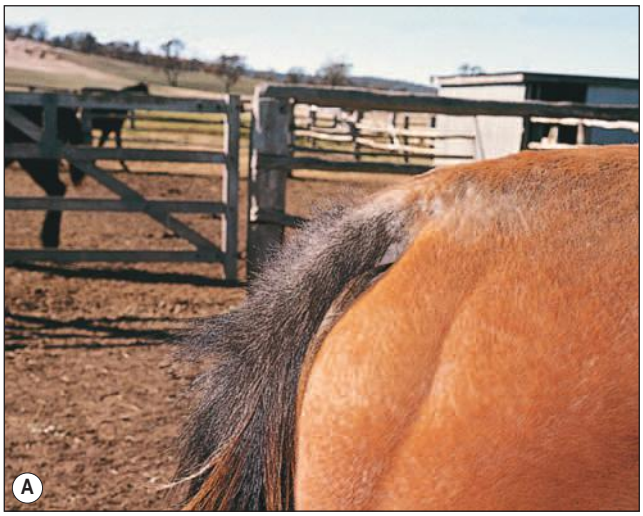




Figure 9.49 (A, B) Oxyuriasis. Tail-rubbing causing breakage, thinning and distortion of tail hairs due to *Oxyuris equi* infestation.

Treatment

Most modern anthelmintics are effective and any worming programme aimed at ascarids, strongyles and cyathostomes will kill the adult worms. The condition can probably be ignored in horses receiving good anthelmintic regimens involving avermectin anthelmintics in particular. Improved stable hygiene with clean bedding and regular cleaning of water troughs is important.

Local treatment consists of washing the perineum with soap and warm water to remove the eggs. The itch may be controlled with tranquillizers until anthelmintics have been given.

Snake envenomation (zootoxicosis)

Profile

Snake bite is a regional occasional event and most bites occur in summer months when the snakes are active. The bites of the various snakes vary according to the specific toxin(s) produced. Usually the spectrum of venomous snakes that are found in any particular geographic region is known. Local veterinarians should acquaint themselves with the types and the clinical signs of each particular species because one of the pillars of management is the use of specific (or more rarely polyvalent) antisera (antivenin).

A species-related panel of neurotoxins, haemotoxins (including coagulants and haemolysins), necrotizing toxins and other substances that increase the local penetration of the toxins such as hyaluronidase are involved. Whilst some are extremely dangerous others are less so. Not every bite injects significant toxin – a significant proportion may be 'dry' or 'nearly dry', i.e. there is no envenomation or only a limited amount.

The signs can be divided further into the immediate signs and those that develop more slowly. The local (usually rapid) responses are possibly a result of the type of toxin rather than the amount but the systemic signs are related more to the relative dose of the venom introduced. The cutaneous signs that develop immediately include local swelling, inflammation and oedema which may obscure the actual bite site. Later there is a variable amount of tissue necrosis around the bite site.

Large horses can be killed by snakes but this is very rare because of the sheer size of the horse. However, some snakes (such as the African black mamba and the Australian diamond back snake) are so venomous that even a large horse can be killed quickly if the volume of venom is high. The bigger risks for horses are the delayed effects such as shock, tissue necrosis, and organ damage; these could result in death some days or weeks later.

The effects of envenomation depend on several factors:

1. The snake type (venoms vary between families and species – cytolytic/haemolytic, coagulative or anti-coagulative/neurotoxic venoms are possible)
2. The specific type of venom (the relative concentration of the venom varies with season and the snake's activity; the age of the snake may also be significant)
3. The volume of venom injected relative to the size of the horse (not all bites inject the same amount of venom; some bites are dry, i.e. have no or very little venom)

Key points: Snake envenomation

1 5 14 17 19 24

1. Various venomous snakes occur in different countries. Usually the species found are known to the local inhabitants. Bites are uncommon but most often occur on the face and legs when the snake is disturbed by the horse. Not every bite injects toxin and there are some bites from non-venomous snakes that are simply infected. Venoms contain various necrotizing toxins, haemolysin, coagulants/anticoagulants, neurotoxins and cardiorespiratory toxins. It is important to know the species involved if possible.
2. Local signs include those at the site of the bite such as pain, swelling and necrosis with bleeding. General signs such as pyrexia, shock, cardiorespiratory or neurological disorders can be encountered.
3. Diagnosis is usually circumstantial – the bite site may be visible but is usually masked by the rapid swelling.
4. Treatment involves local and intravenous injection of specific or polyvalent antiserum (if one exists and is available) and symptomatic treatment for the various signs that develop. Facial swelling can cause death so tracheostomy can be life-saving.
5. Prevention is difficult but knowledge of the local species and their behaviour is helpful. In areas where bites are common antiserum should be available.

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Table 9.4 Snakes and their toxic effects

Species	Examples	Toxins	Clinical effects
Viperidae (folding fang snakes)	Adders Puff adder Gaboon viper Rattlesnake Copperhead Diamondback	Cytolytic/proteolytic enzymes Minor neurotoxins	Local tissue destruction Local pain/rapid oedema Haemorrhagic exudate from bite wounds Generalized neurological signs
Elapidae (fixed fang snakes)	Coral snake Cobra Mamba	Neurotoxins	Slower onset Progressive paralysis (spinal and cranial nerves) Respiratory/cardiac failure
Back fixed fangs	Boomslang Vine snake Herald snake	Blood coagulants Haemorrhagic signs	Coagulopathy similar to disseminated intravascular coagulopathy Bruising, intestinal and respiratory bleeding
Solid teeth snakes	Python Boa	Non-venomous	Bites rapidly become infected Abscess and local necrosis

4 The location of the bite (bites on the face can cause death through swelling-induced asphyxia while bites on the limb may be relatively 'isolated')
5 The time scale between envenomation and local or systemic treatment (delays allow toxin dissemination whilst early antiserum and symptomatic treatment may limit the spread and the toxicity).

There are several groups of snake and they have specific toxic effects, as shown in Table 9.4.

Clinical signs

Most snake bites occur on the face (nose) and on the limbs – correlating with the likely contact areas. Bites around the face and nose may be extremely dangerous – rapidly inducing asphyxia.

The signs of snake bite can be divided into the local signs occurring at and around the bite site and the systemic signs. Provided that the bite is not 'dry' (i.e. no envenomation takes place) there is usually a noticeable mild to extreme local cutaneous swelling and inflammation at the site of the bite (Fig. 9.50).

Profuse sweating, pyrexia, dyspnoea, cardiac irregularities and ventral nervous signs can develop rapidly.

Epistaxis can be seen with some types of snake bite and sometimes haemorrhage from body orifices is present.

Differential diagnosis

- Spider and scorpion stings/bites: can be very similar.
- Vasculitis: usually either generalized or at least affecting multiple roughly circular sites of skin necrosis.
- Skin trauma: usually not as rapid nor as aggressive responses.
- Anthrax: rare septicaemic, highly febrile condition with only limited skin pathology – a hot, painful, small focus may be present.

Diagnostic confirmation

- Known venomous snakes in the environment of the horse (usually their presence is recognized by the local

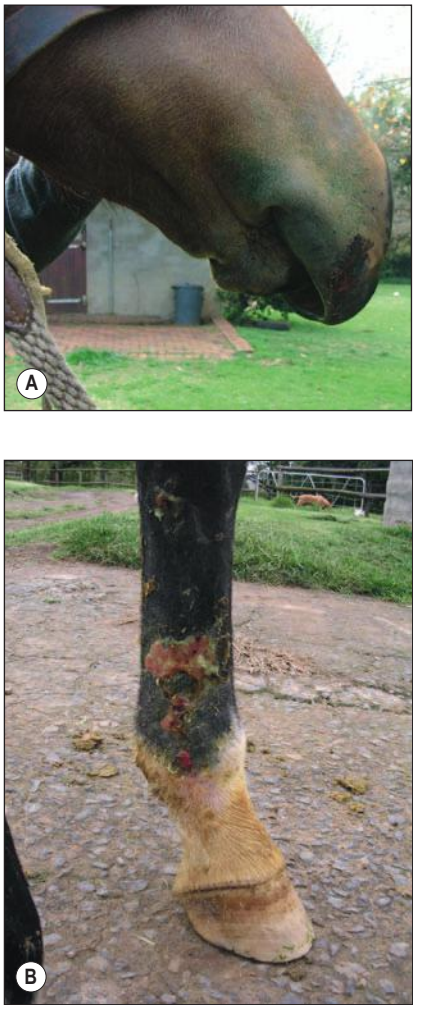


Figure 9.50 (A) This pony received a bite on the lip from a red-lipped herald snake (*Crotaphopeltis hotamboeia*) in South Africa. Note the extensive local swelling that can threaten breathing. Bites around the muzzle are common due to the inquisitive nature of the horse! **(B)** Extensive tissue necrosis on the leg from a bite by a puff adder (*Bitis arietens*). Bites on the limbs usually result from direct tramping or disturbance of the snake. Healing in all cases can be slow and delayed systemic effects can be encountered. (Figures courtesy of Dr Dave Mullins.)

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populations). It is important, however, to recognize the species of snake responsible for the bite because antiserum is available for many but is specific.
- Immediate signs are not usually found easily. Swelling associated with obvious bite marks in a pattern consistent with the fangs of a snake is highly suggestive but the marks may quickly be obliterated by the swelling.
- Snake bite (along with the most venomous spiders and scorpions) is one of the few causes of localized peracute inflammatory swelling and subsequent necrosis of the skin.
- Secondary signs of shock, cardiorespiratory and neurological problems, epistaxis and severe depression are suggestive.

Treatment

The management of snake bite is bound to vary with the species of snake involved. Antisera have been produced for most of the venomous snakes (and incidentally some of the spiders, scorpions and other venomous creatures) but their use depends on their availability and the accurate identification of the snake involved. Usually this is not known but can be surmised from local circumstances. The benefits of antivenin (antiserum) are greatest when it is given soon after the bite has occurred (<2–3 hours) and when it is divided between local infiltration around the bite site and intravenous injection.

Local tourniquets applied may be feasible in some anatomic sites but these are sometimes more distressing for the horse and whatever else happens the horse needs to be kept quiet. Excitement and panic serve only to increase the dissemination of the venom. If possible the horse should be moved by vehicle rather than walking.

Where the bite marks are clearly visible within an hour of the bite, it is likely that the bite was not venomous (or the snake was not a venomous variety or that the bite was dry). In these circumstances careful monitoring and local management of the bite site with antiseptics and systemic antibiotics (and possibly fast-acting corticosteroids) should help.

Moderate reactions probably require a more aggressive response with non-steroidal anti-inflammatory drugs, corticosteroids and fluid therapy support. These animals will probably benefit from a specific antiserum (antivenin). The dose of antivenin may depend more on the dose of venom than the size of the horse so a maximal dose should be given immediately and thereafter there should be little need to repeat it.

Extreme bites require emergency medical procedures including tracheostomy, oxygen administration, aggressive fluid therapy and the full range of sedative and supportive drugs including corticosteroids, non-steroidal anti-inflammatory drugs, analgesics and antibiotics. Multiple doses of antivenin may be justified both locally and intravenously.

Recovery from the bites (either local or systemic) can be very slow.

Tetanus prophylaxis should always be checked.

References

Arundel J H 1978 Parasites of the horse. Veterinary Review, no. 18 Post Graduate Foundation for Veterinary Science, Sydney, Australia

Barbet J L, Baxter G M, McMullen W C 1991 Diseases of the skin. In: Colahan P T, Mayhew I G, Merritt A M, Moore J N (eds) Equine medicine and surgery, 4th edn. American Veterinary Publications, Goleta, CA, pp 1569–1736

Dewes H F, Townsend K G 1990 Further observations on *Strongyloides westeri* dermatitis. Recovery of larvae from soil and bedding and survival in treated sites. New Zealand Veterinary Journal 38:34–37

Fadok V A, Greiner E C 1990 Equine insect hypersensitivity: skin test and biopsy results correlated with clinical data. Equine Veterinary Journal 22:236–240

Foil L, Foil C 1986 Parasitic skin diseases. Veterinary Clinics of North America. Equine Practice 2:403–437

French D D, et al 1988 Efficacy of ivermectin in paste and injectable formulations against microfilariae of *Onchocerca cervicalis* and resolution of associated dermatitis in horses. American Journal of Veterinary Research 49:1550–1554

Herd R P, Donham J C 1983 Efficacy of ivermectin against *Onchocerca cervicalis* microfilarial dermatitis in horses. American Journal of Veterinary Research 44:1102

Littlewood J D, Rose J F, Paterson S 1995 Oral ivermectin paste for the treatment of chorioptic mange in horses. Veterinary Record 137:661–663

Pascoe R R 1973 The nature and treatment of skin conditions observed in horses in Queensland. Australian Veterinary Journal 49:35–45

Pascoe R R, Connole M D 1974 Dermatomycosis due to *Microsporum gypseum* in horses. Australian Veterinary Journal 50:380–383

Schmidt G M, Coley S C, Leid R W 1985 *Onchocerca cervicalis* in horses: dermal histopathology. Acta Tropica 42:55–61

Schumacher M J, Schmidt J O, Egan N B, Dillon K A 1992 Biochemical variability of venoms from individual European and Africanized honey bees (*Apis mellifera*). Journal of Allergy and Clinical Immunology 90:59–65

Scott D W 1988a Dermatohistopathology. In: Large animal dermatology. WB Saunders, Philadelphia, p 49

Scott D W 1988b Large animal dermatology. WB Saunders, Philadelphia

Staempfli H R, Kaushik A 1993 Clinical reactions of horses to venoms from winged stinging insects. Equine Veterinary Education 5:259–261

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10

Congenital/developmental diseases

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Skin lesions are a relatively common complaint in newborn and young foals and these can be congenital or acquired.

Congenital skin diseases are, by definition, those that are present (though not necessarily visible) at birth. As some are not detectable until much later, there is a developmental component of some of the conditions. The fact that they occur congenitally does not mean that they are hereditary – although some are, little is known about most of them. For the few recognized hereditary skin disorders, the breeding implications will have a significant bearing on the advice that is given to an owner. It is easy to assume that a mare or stallion involved in the breeding should be removed from the breeding stock but so little is known about the majority of these conditions that it is probably unfair to make categorical statements. Some breed societies simply accept a low rate of affected foals. For example, the lethal white syndrome in Overo-Paint horses is an accepted very rare congenital condition. Dermoid choristoma can be located on the conjunctiva and be obvious at or shortly after birth. Furthermore, some of the most important congenital conditions are only manifest later in life. Dermoid cysts are not usually detectable in the young foal but are in any case both trivial and treatable. Hereditary equine regional dermal asthenia (HERDA) may only be apparent when a saddle is first applied to the horse but usually careful examination will establish that some pathological events have taken place prior to the first detectable episode.

Most of the congenital disorders are untreatable and some are incompatible with normal life either because they do not resolve or because they are associated with other potentially or actually fatal systemic abnormalities. For example, the lethal white syndrome of Overo-Paint horses is associated with profound neurological abnormalities and intestinal atresia. The skin appearance may be of no material significance but it may act as a marker for the diagnosis of other more serious abnormalities. There are dangers in making this association though and an albino (white) foal presented for neonatal colic may simply have an impacted small colon. However, it may be hard to convince an owner that the foal warrants euthanasia when the extent of cutaneous agenesis seem trivial.

A significant number of congenital skin diseases do not materially affect the welfare of the horse and so can either be tolerated or managed later in life when they may become more significant.

The breeding implications always need to be considered when a foal is presented with serious congenital abnormalities. Where breeding programmes are monitored closely it may be possible to draw conclusions about the merit or otherwise of rebreeding either the mare or the stallion or both. The Fell pony immunodeficiency syndrome has prominent primary cutaneous signs but the inherent immunocompromise means that secondary cutaneous infections are often present. Since the disease is an established autosomal recessive, the mare and the stallion should be removed from the breeding population immediately on the grounds that they must both be heterozygous recessive carriers. However, where a homozygous dominant stallion can be identified, it is sometimes justifiable to allow breeding to carrier mares. Whilst this clearly has implications for the

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dissemination of the disease gene, no foals will be affected by the syndrome and gradually the population will be biased towards dominant (non-carrier) animals. This therefore can be viewed as a justifiable first step in a control programme. However, the reality is that little is known about most of the conditions and so little advice can be given with confidence.

The acquired conditions are those that occur as result of injury, infection or secondary to underlying disease. Examples of these include traumatic injuries as a result of savaging, predation or trauma. Systemic infections may also manifest with skin diseases and some skin lesions are encountered with immunological disorders or primary skin infections. Decubitus ulcers, for example, may occur over the bony prominences in foals that are recumbent or those that struggle to stand or those that are seizuring. Sometimes the skin lesions can be a strong indicator of other perhaps more serious disease that might otherwise pass unnoticed until it is too late. The attending clinician should establish quickly if the skin lesions are in fact secondary to some deeper problem; owners may, for example, blame a skin deficit on savaging or predation but if that was not plausible it may be that the foal has a primary congenital condition.

When a neonatal foal is presented with skin disease, the clinician needs to take account of all the available possibilities because some are cutaneous manifestations of systemic disease. For example, a young foal with a severe cutaneous infection with *Dermatophilus congolensis* may be immunocompromised and the earliest sign of the Fell pony syndrome is a russet-coloured, long coarse coat.

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PRIMARY SKIN DISORDERS IN NEWBORN FOALS

Epitheliogenesis imperfecta (aplasia cutis; cutaneous agenesis)

Profile

This is a rare congenital, inherited cutaneous defect of foals. Possibly a single autosomal recessive gene is responsible, at least in American Saddlebred horses (Scott 1988a, Stannard 1994a, Lieto & Cothran 2003).

There is a complete or partial absence of one or more of the dermal components including the epidermis, dermis, subcutis and fat (Scott 1988b). Although defects on the limbs are probably more common, the body trunk and hooves, and even the tongue and proximal oesophagus may also be affected.

Many of the previous reports of this condition can be attributed to epidermolysis bullosa (see p. 232) rather than cutaneous agenesis (Scott & Miller 2003a).

Clinical signs

Clearly demarcated solitary or regional areas with a complete absence of epidermis and skin appendages are present at birth (Fig. 10.1). The extent of the skin defect may be less severe than the subcutaneous adhesion problems. The commonest and most obvious defects occur distal to carpus and tarsus but it can be over much larger areas. Dermal adhesion in the adjacent tissues can be severely affected and in some cases the defect can be extensive. The margins of the defect are noticeably free of inflammation **(Fig. CD10 • 1)**.

The lesions bleed easily with minor trauma and quickly become infected, possibly leading to septicaemia and death. Foals with limited defects have a reasonable chance of growing normally, but closure of the fault is difficult and requires repeated procedures.

The tongue and the proximal oesophagus can be affected also. Severely affected foals may be aborted.

Differential diagnosis

- Epidermolysis bullosa: a very rare vesico-bullous condition associated with a dermo-epidermal fragility so that the skin can be rubbed off the affected areas; largely restricted to the Belgian breed but can occur in other breeds and donkeys.

Key points: Epitheliogenesis imperfecta

2 3 4 11 14 15 20 24

1. Rare condition with a genetic but not necessarily heritable basis.
2. Absence of variable areas of the skin is the most common syndrome but sometimes it is complicated by concurrent defects in the subcutis resulting in loss of adhesion between the skin and the subcutaneous tissues. Involvement of the oesophagus or tongue can be present.
3. Diagnosis is based on signs and the unexplained skin deficits.
4. Treatment options are limited. Repeated reconstructive surgical procedures can be attempted but are seldom effective; wound breakdown and severe infections are common.
5. Prognosis is very poor but foals with limited defects can survive provided that the affected region can be managed.



Figure 10.1 Epitheliogenesis imperfecta affecting large areas of the skin of a stillborn foal.

- Hereditary equine regional dermal asthenia (HERDA): cutaneous adhesion disorder that is restricted largely to Quarterhorses and manifest by skin fragility and lack of elasticity. Signs are usually delayed until the skin is subjected to trauma from harness and weight-bearing. Wounds that result from skin necrosis are very slow to heal and may not do so or may result in severe disfiguring scars.
- Obstetric trauma/trauma/savaging or predation: history of dystocia or overt evidence of trauma.

CD

Diagnostic confirmation

- Clinical appearance of complete lack of epidermis and skin appendages over a variable area from birth. There are no other diseases which induce this appearance at birth.
- Negative Nikolsky's sign (transverse finger pressure does not cause the epidermis to 'slip' off the underlying dermis).

Treatment

No treatment is consistently effective – even skin grafting of the affected area usually fails to close it. Surgical debridement of the margin of the defect may permit some closure but the loss of adhesion in the adjacent subcutis is a major obstacle to a resolution. In this case extensive debridement and reconstructive surgery must be performed if there is to be any chance at all. Very small defects can sometimes be closed with repeated surgical reconstructive measures and very small defects can be managed in the longer term. Some very small localized defects are said to heal adequately with scarring but the protracted non-healing wounds are usually refractory to any form of treatment. Euthanasia is the only course of action when extensive lesions and hoof, mouth or oesophageal defects are present. The possibility of septicaemia must be considered when these cases are presented.

Neither the mare nor the stallion should be used for breeding in view of the suggestion that it may be an autosomal recessive gene.

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Epidermolysis bullosa

Profile

This is a very rare complex group of heritable junctional mechanobullous diseases occurring principally in the Belgian and Saddlebred breed of both sexes, characterized by fragile 'blister' formation on the skin and oral mucosa, following mild trauma (Kohn et al 1985, Scott 1988a, Stannard 1994a). Other breeds and donkeys are sporadically affected. Although the human condition carries the same name and there are some similarities, there is limited information on how this relates to the equine condition (Linder 2000).

The junctional form of the condition is the most common form and probably results from a lamina lucida defect (Berthelsen & Eriksson 1935, Lieto et al 2002). Variations in location of the clefting within the epidermis are theoretically interesting but have no material significance for the management of the condition. It has been suggested that some of the previous reports of cutaneous agenesis in foals may in fact have been epidermolysis bullosa and so the condition may be slightly more common.

Clinical signs

The condition is recognized by severe blistering of the skin on the newborn foal. The primary lesions consisting of intracutanous bullae are seldom seen. Most rupture quickly with minor trauma or even movement.

Lesions at mucocutaneous junctions and oral mucosa and elsewhere are visible at birth (Fig. 10.2). Further lesions develop shortly afterwards. Collapsed bullae may be found in the mouth, and separation of hooves at the coronary band is sometimes present and foals have sloughed the hoof capsule within hours or days of birth (Frame et al 1988).

Concurrent dystrophic teeth commonly occur.

Secondary local and systemic infections are common and often result in progressive depression, illness and death.

Differential diagnosis

- Epitheliogenesis imperfecta: overt deficits of skin cover present at birth over various localized or more general regions in the complete absence of any traumatic cause.

Key points: Epidermolysis bullosa



1. A rare inherited condition primarily (but not exclusively) restricted to the Belgian breed, affecting the dermo-epidermal junctional regions of limited but sometimes extensive areas of the skin.
2. Minor trauma results in bulla (blister) formation and when these rupture they leave an open ulcerated area that bleeds and is easily traumatized and infected. The limbs are more often affected than the body trunk and head. The mouth and coronary band may be severely affected.
3. Diagnosis is made on the clinical signs and history. The Belgian breed is over-represented.
4. Treatment is not possible and euthanasia is the only effective approach for most cases.
5. Breeding from both parents should probably be stopped.



Figure 10.2 Epidermolysis bullosa affecting the medial forelimb region in a Belgian foal.

- Hereditary equine regional dermal asthenia: usually manifest much later in life.
- Bullous pemphigoid.
- Pemphigus foliaceus.
- Systemic lupus erythematosus-like syndrome.
- Scalding and burning injuries: known/suspected history.

Diagnosis

- Few other conditions are realistically similar.
- Some breed relationships (Belgian/Standardbred).
- Histopathology shows characteristic cleavage and bulla formation at the epidermal–dermal junction.
- Positive Nikolsky's sign (transverse finger pressure *does* cause the epidermis to 'slip' off the underlying dermis).

Treatment

No treatment is possible. The mechanisms of treatment for the human condition, in which genetically modified stem cells are used, should not be attempted in horses.

Breeding from both mare and stallion should probably be stopped although currently the evidence for heredity is tenuous.

Generalized primary seborrhoea

Profile

A very rare condition most noticeable in younger horses (<2 years of age).

Clinical signs

At birth the foal is usually normal but in the first 6–12 months of life, the coat becomes greasy and staring. The normal patina of horse skin is lost (Fig. 10.3).

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Figure 10.3 This foal was diagnosed with primary seborrhoea from birth. (Figure courtesy of Dr Marianne Sloet.)

Key points: Generalized primary seborrhoea

2 3 4 12 20 24

1. A rare congenital disorder of epidermal cell hyper-proliferation with inappropriate sebum production.
2. Generalized greasy scale and flake occur over the whole body from a young age. Secondary bacterial and *Malassezia* spp. infection is common.
3. Diagnosis is only made when the condition has been present from birth or shortly afterwards and when it affects the whole body and causes of secondary seborrhoea have been eliminated. Biopsy is largely unrewarding with non-specific epidermal hyperplasia and hypertrophy and excessive scale.
4. Treatment is palliative/symptomatic with degreasing and descaling shampoos.
5. The prognosis for the animal is good but the skin requires long-term management.

No significant systemic clinical symptoms are associated and there is no pruritus or pain unless secondary bacterial and fungal infections develop.

Differential diagnosis

- Secondary seborrhoea (see p. 459): usually a result of other chronic skin infections/insults.
- Hypothyroidism: extremely rare and associated with hair and skin thinning with goitre development.
- Neonatal pemphigus foliaceus: very rare, generalized, immune-mediated vesico-bullous, seborrhoeic condition with coronary band and chestnut margin involvement; histology may be typical (see p. 266).

Diagnostic confirmation

- Elimination of causes of secondary seborrhoea through exhaustive testing and biopsy.
- Histological evidence of epidermal hyper-proliferation and excessive scale formation.
- Bacterial cultures can identify some secondary bacterial and fungal infections.

Treatment

Treatment is restricted to degreasing and anti-scale shampoos and if necessary intermittent antibacterial and antifungal shampoos. Advice on breeding cannot be given as the condition is very rare.

Congenital curly coat syndrome

Profile

A congenital 'curly coat' syndrome has been recently described occurring mainly in Paint horses. A similar syndrome has been recognized in Quarterhorse, Percheron, Arabian, Appaloosa, Missouri Fox Trotter, Tennessee Walking Horses, Morgan and Paso Fino breeds.

Defined genetic lines are possibly more susceptible and the trait appears to be a dominant gene. Currently there is a move towards recognition of the curly-coated horse as a specific breed (Scott 2004).

Clinical signs

Foals are born with an abnormally long, curly hair coat that remains until the adult hair grows through and in some cases the condition remains (Fig. 10.4). The main and tail are also affected. The extent of curliness is very variable. The mane and tail can be affected alone ('wavy-mane syndrome') but it can also affect the whole body surface. The winter coat is often a more tightly curled 'ringlet-like' pattern while the summer coat is smoother but with prominent 'waves'.

There may be variable degrees of alopecia with coat and mane and tail thinning (hypotrichosis) especially in the summer coat. The tail is often badly affected and may lose almost

Key points: Congenital curly coat syndrome

2 4 16 20 24

1. A congenital disorder mainly affecting Paint horses. This is sometimes viewed as a desirable trait and so active steps are taken to maintain the condition in some breeds and lines within breeds.
2. A prominent curly, dense coat is present from birth and develops into a dense, wavy coat quality. The condition may be restricted to the mane and tail (wavy mane syndrome) but can affect the whole body. Some hair loss is also often present including the tail and mane.
3. Diagnosis is based on the characteristic features and the breeding history.
4. Treatment is not usually requested and in any case there is none!
5. The condition has no apparent deleterious effect on the horse and neither is it associated with any other congenital defect and so the prognosis is irrelevant.

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Figure 10.4 A young Paint horse with a congenital curly coat syndrome.

all its hair, giving the horse a rat-tailed appearance (termed 'string tail').

No significant clinical symptoms associated with the condition but some owners are unaware of the condition until a foal is born with it.

Differential diagnosis

- Normal winter coat: this is seldom very thick and curly but in any case sheds normally, leaving a natural smooth, short summer coat.
- Pituitary pars intermedia dysfunction (PPID/equine Cushing's disease): this occurs in old horses > 15 years of age.

Diagnostic confirmation

- Breed line related (owners will usually recognize it immediately). The signs appear to be the same for winter and summer coats.
- Histological evidence can be derived from body and mane/tail biopsy (Scott 2004) but the histological signs are subtle (Scott & Miller 2003b). Follicular dysplasia appears to be the most characteristic histological feature.

Treatment

Treatment is neither warranted nor possible. Breeding from 'curly horses' has become desirable in some circumstances and a special set of Breed Societies have been set up to study and promote the trait.

Albinism/lethal white foal syndrome

Profile

Albinism is a condition in which there is a congenital lack of pigment (melanin) in the skin, hair and iris. Normal melanocytes that have a biochemical defect preventing them from synthesizing melanin are present. Cream-coloured horses lack pigment but are otherwise normal. True albinos have white hair, pink skins and non-pigmented (pink or light blue) irides (and usually have no pigment in the fundus of the eyes). General albinism is an autosomal *dominant* trait and is only viable in the heterozygous state (Ww). While being white-haired, they may have some coloration of the iris (blue or blue/white streaks). When these horses are mated, non-viable embryos (WW) occur in 25% of conceptions and are resorbed or aborted early in gestation. This is best regarded as one form of lethal white foal syndrome.

Lethal white foal syndrome occurs with the mating of Overo and Paint horses of the pinto breed and is an autosomal *recessive* trait. This is an inherited condition. Cases have been encountered in other breeds also (Lightbody 2002). Affected foals are white (albinos) but frequently have concurrent, highly fatal defects of the gastrointestinal tracts such as atresia ani, atresia coli and in particular ileocolonic aganglionosis (Knottenbelt & Pascoe 1994, Stannard 1994a). The pathogenesis of the condition appears to be linked to the gene that controls endothelin metabolism.

Key points: Albinism/lethal white foal syndrome



1. Albinism is an incidental condition that affects ponies more than horses. Affected animals have a white or pink skin (cf. grey horses which have a black or dark skin). The lethal white syndrome is an important and highly fatal disorder mainly affecting foals with Overo-Paint breeding.
2. Albinism does not, in itself, have any disease implication for the horse although the lack of pigment may make the skin more sensitive to sunlight and so actinic dermatitis and squamous cell carcinoma may be more prevalent.
3. The lethal white syndrome is characterized by a severe non-responsive neonatal colic associated with either an atretic (incomplete) gastrointestinal tract or ileus resulting from aganglionosis or both.
4. Diagnosis of albinism is simple. Diagnosis of lethal white syndrome is based on the very typical clinical signs and breeding history.
5. There is no treatment for either condition. Albinotic horses probably need to be given extra sun-protection (mask for face and eyes and light rugs) but most seem to cope well anyway. Lethal white foals must be destroyed immediately; any attempts to restore normal gut function will fail.

Clinical signs

Partial albino horses are relatively common, showing pink skin and a white hair coat (Fig. 10.5A). The retina is invariably partially or completely albinotic and the iris may be variously pale blue or white in colour (either completely or partially). Possibly the greatest implication for these animals is their liability to actinic dermatitis (sunburn) and squamous cell carcinoma. Many also have degrees of photophobia and light-related retinal damage can occur.

Lethal white foals are a startlingly white colour at birth (Fig. 10.5B); a few have small faint brownish tinges particularly on their ears. Although they may be apparently normal for the first 1–2 hours after birth, they rapidly develop severe intractable and fatal colic as a result of an incomplete intestinal tract or ileus as a result of intestinal aganglionosis (or both). Concurrent abnormalities of the mouth, pharynx, heart or other structures are also sometimes present.

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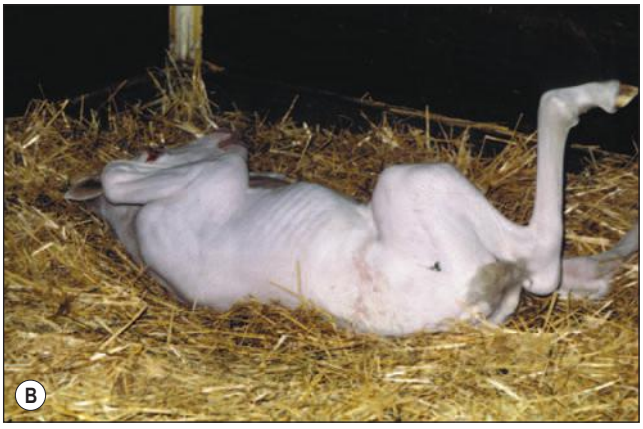


Figure 10.5 (A) Albino foal and albino mare. Both had albinotic retinas and non-pigmented (blue/yellow) irides. Both lived normal lives. **(B)** The lethal white syndrome in Overo-Paint-bred horses.

Differential diagnosis (lethal white syndrome only)

- Neonatal ischaemic hypoxic syndrome (neonatal maladjustment)/cerebral trauma at or during birth: these foals are not usually showing colic signs but are genuinely seizuring.
- Idiopathic/benign epilepsy: genuine seizures without colic and not related to colour or breeding.
- Cerebellar/cerebral hypoplasia syndromes: cerebellar ataxia and hypermetria with intention tremors.
- Extensive blood loss at or during birth (visible or invisible) including early cord separation: obvious and usually profound anaemia.

Diagnostic confirmation (lethal white syndrome only)

- Characteristic colour and abrupt onset of associated clinical syndromes of colic and/or intractable seizures.
- Arterial oxygen normal and no evidence of parturient hypoxia.
- Normal haematology/biochemistry.
- Cerebrospinal fluid and electroencephalograms are normal.
- Post-mortem examination will identify concurrent developmental abnormalities and intestinal aganglionosis.

Treatment

Albino foals are often normal and survive normally (cream horses are a form of this), although many are mildly photophobic. Limiting exposure to bright sunlight is often sensible and appreciated by the animal. Early recognition and treatment of actinic dermatitis and squamous cell carcinoma are important.

Note

Some cremello or albino horses can develop isolated melanocytic tumours in the skin (see p. 422).

There is no treatment for lethal white foals; attempts to control colic and seizures are fruitless. General anaesthesia gives temporary relief. Attempts to anastomose atretic intestine or enterectomy of affected intestine are a waste of time and effort. This condition should presently be regarded as 100% fatal within 24–72 hours.

Lavender foal syndrome

Profile

Lavender foal syndrome is a rare, poorly characterized, (supposedly) hereditary disorder of Arabian foals derived from a particular Egyptian line. The name derives from the unusual hair coat colour, which can be tan to brown but which has a slightly purplish, 'lavender' appearance. Most cases have been reported in the United States but case series have also been reported in South Africa.

The syndrome is invariably fatal and has a highly complex pathogenesis that involves critical neurological signs.

It may be coupled with other congenital/genetic defects such as cardiac deformities, wry nose or cleft palate and possibly intestinal aganglionosis.

Key points: Lavender foal syndrome

2 3 4 16 19 20 24

1. A recently described, genetically based coat colour dilution syndrome mainly seen in Arabian horses and their crosses.
2. Prominent neurological symptoms are present from birth and the affected animal usually fails to stand. Convulsions, blindness and opisthotonus are present.
3. Diagnosis is based on clinical signs alone – laboratory features are non-specific and rather disappointing.
4. There is no treatment – the condition is fatal.
5. Parents of the affected foal should probably not be used for breeding but little is known about the syndrome as yet.

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Clinical signs

'Lavender foals' have a characteristic lavender (bluish/purple) coat colour at birth (Fig. 10.6). The skin is actually normal and there is no dermatological implication. The colour is simply a visible indicator of a critical (lethal) neurological disorder. Affected foals are neurologically abnormal from birth, showing neonatal seizures and convulsions (often even occurring during or immediately after birth). Opisthotonus, paddling, aimless galloping while recumbent and apparent blindness with a light-hungry and startled expression may be present. No periods of remission or sleep occur. Affected foals fail to rise and have no (or reduced/abnormal) suckle reflexes. Death is inevitable.

Differential diagnosis

- Neonatal-hypoxic-ischaemic encephalopathy/cerebral trauma at or during birth: convulsions are similar but biochemical tests and blood gas analysis can be used to confirm the conditions; no coat colour relationship.
- Idiopathic/benign epilepsy: no colour relationship and seizures are intermittent and predictable with prodromal and post-ictic signs.
- Cerebellar/cerebral hypoplasia/abiotrophy syndromes: coarse movements and intention tremors but otherwise bright and alert; mainly Arabian foals.
- Extensive blood loss at or during birth (visible or invisible) including early cord separation: overt anaemia and history of blood loss.

Diagnostic confirmation

- Characteristic colour and abrupt intractable seizures.
- Most cases of lavender foal syndrome show no detectable lesions in any organ, suggesting that it is a functional/physiological neurological disorder with skin colour alterations.
- Arterial oxygen is normal and there is usually no evidence of parturient hypoxia.
- Normal haematology/biochemistry.
- Cerebrospinal fluid and electroencephalograms are normal.
- Post-mortem examination will identify concurrent developmental abnormalities and intestinal aganglionosis.

Treatment

There is no treatment. Attempts to control seizures are fruitless. General anaesthesia gives temporary relief. The condition should presently be regarded as 100% fatal within 24–72 hours (Page et al 2006).

Fell pony immunocompromise disorder

Profile

This condition is a recently described disorder affecting pure bred Fell pony foals under 16 weeks of age (Scholes et al 1988, Richards et al 2000, Butler et al 2006). Severe immunocompromise as a result of a T-cell malfunction with a simple recessive gene is involved. The condition is not similar to any of the other major immunocompromising disorders of foals.

Cutaneous signs are relatively minor but provide a good (if non-specific) early warning system. The panel of signs suggests that other genes may be involved also. There is a very high prevalence of the carrier status in the Fell pony population. Specific breeding lines do carry a major risk of the disease.

Clinical signs

The foal is usually born relatively normal but frequently the coat is rough, russet in colour and there is an abnormal length of many hairs, particularly on the head and neck, giving it a halo-like appearance.

Diarrhoea and failure to thrive as well as increasing lethargy are common. A progressive microcytic, normochromic anaemia is a universal feature (Fig. 10.7).

Immunocompromise results in opportunistic infections including those on the skin and mucosal surfaces. Secondary *Candida* spp. infection of the oral cavity and intestinal tract, and *Dermatophilus congolensis* and *Staphylococcus* spp. infections of the skin are common.

The condition is invariably fatal before 12 weeks of age.



Figure 10.6 A typical lavender foal. The foal showed all the characteristic neurological signs and was destroyed immediately upon diagnosis. (Figure courtesy of Dr Johann Neser.)



Figure 10.7 This 5-week-old Fell pony foal had a russet-coloured coat and was presented with all the classic signs of the Fell pony syndrome including anaemia and respiratory and alimentary tract infections. *Dermatophilus congolensis* skin infection and *Candida* spp. infections are common in these foals and it is easy to be misled into thinking they are primary conditions.

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Key points: Fell pony immunocompromise disorder

2 4 15 16 19 20 23 24

1. A specific inherited autosomal recessive immunocompromising disorder affecting Fell pony foals under 12–14 weeks of age.
2. Cardinal early signs include a russet halo of long coarse hairs and progressive non-regenerative anaemia. Opportunistic infections including those of the skin are frequently encountered. Other signs include diarrhoea, poor growth and weight gain and oral, respiratory and intestinal infections.
3. Diagnosis is based on breeding history and on the typical signs – usually anaemia is taken as an early indicator.
4. Treatment attempts are fruitless – the condition is 100% fatal.
5. Neither parent of an affected foal should be used for breeding.

Differential diagnosis

- Haemorrhagic conditions or hereditary/congenital haemophilia: haemorrhages are obvious with prolonged clotting parameters; regenerative anaemia.
- Severe combined immunodeficiency syndrome (only recorded in Arabian foals): Arabian foals only; usually fairly well until secondary infections develop after 3–6 weeks. Well-recognized hereditary features.

Diagnostic confirmation

- Characteristic clinical combination of opportunistic infections, anaemia and a russet, coarse coat with a halo of visible long hairs over the head and neck in particular make a pathognomonic panel of signs.
- T-cell function tests are definitive.
- Genetic identification of affected cases is not necessary.
- Haematology is consistent with a profound, progressive non-regenerative anaemia and biochemical evidence of immunocompromise.
- Post-mortem examination will identify generalized lymphoid hypoplasia including the thyroid, spleen and lymph nodes. Bone marrow is severely affected with almost no active marrow.

Treatment

There is no treatment. Fell pony foals that have the combined signs that are characteristic of the syndrome have a hopeless prognosis. All affected foals so far have died at less than 6 months of age and most well before 12 weeks.

Congenital equine cutaneous mastocytosis

Profile

A rare form of mast cell tumour that is present at or shortly after birth.

Clinical signs

The skin shows multiple small, focal nodules especially along the body trunk and the lateral aspects of the hind limbs. The size seldom exceeds 3 cm diameter. The larger nodules are characterized by a soft fluctuant centre and superficial skin ulceration. The nodules do not vary with time but new nodules may develop and regress spontaneously (Johnson 1998).

Key points: Congenital equine cutaneous mastocytosis

3 4 9 14 15 23 24

1. Congenital form of mast cell 'tumour'-like condition.
2. Multiple small raised circular/ovoid nodules (3–25 mm diameter) in the skin at or shortly after birth along the body trunk and lateral aspects of the hind limbs, in particular. The lesions may ulcerate or become fluctuant.
3. Characteristic histopathology.
4. Treatment is not required in most cases because the lesions regress spontaneously over a variable period. Individual lesions can be safely removed surgically.
5. There is no evidence of any long-term harm from this very rare condition.

Differential diagnosis

- Cavernous haemangioma/vascular hamartoma: usually obvious and highly vascular expanding tumour.
- Congenital melanoma: focal dark brown or black nodular lesion.

Diagnostic confirmation

- Surgical excision or
- Biopsy and histopathology are definitive.

Treatment

Total surgical removal or radiation are the only effective options.

Prognosis

Guarded. Some are very difficult to remove fully without damaging underlying tissues.

Congenital vascular hamartoma/cavernous haemangioma

Profile

This is an uncommon congenital solitary, spontaneous, cutaneous tumour of vascular endothelial cells (commonly referred to as a congenital vascular hamartoma) and has been recorded on the distal limbs, and elsewhere, of newborn Arabian and Thoroughbred foals in particular (see p. 412). Heritability is not established.

Note

A hamartoma is an abnormal accumulation of a normal tissue type and most often affects muscle (muscular hamartoma), blood vessels (vascular hamartoma) and dermal melanocytes. Although it is often tumorous it is not a neoplasm.

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Figure 10.8 A localized congenital vascular (cavernous) haemangioma on the hind pastern of a 1-week-old Arabian foal.

Key points: Congenital vascular hamartoma (cavernous haemangioma)

3 4 9 11 14 20 23 24

1. Uncommon skin tumour of newborn foals that progresses slowly or occasionally more rapidly.
2. Commonest sites include the distal limbs and the umbilical area.
3. Diagnosis is fairly straightforward as there are few alternatives (naevus and congenital viral papilloma) but biopsy may be helpful – biopsy does result in significant bleeding.
4. Treatment is limited to surgical excision (assuming this is possible) with or without grafting or radiation. Topical anti-tumour compounds are largely ineffective.
5. Prognosis is very guarded as some cases are highly invasive.

Clinical signs

The congenital form appears as a fleshy, proliferative, papilloma-like lesion initially restricted to the skin surface which bleeds readily with minor trauma and is very difficult to remove surgically without recurrence (Fig. 10.8).

More invasive (aggressive) forms may develop, particularly on the belly wall where progression of the tumour ultimately leads to euthanasia **(Fig. CD10 • 2A–C)**.

Where the aggressive form occurs in the distal limb there is a critical and impossible invasion of the underlying structures including the tendon sheath **(Fig. CD10 • 3)**.

Single lesions are commoner than multiple ones.

Differential diagnosis

- Viral papilloma (including the congenital form): localized, benign and persistent naevus condition. No progression and easily amenable to surgical excision.
- Equine congenital cutaneous mastocytosis (congenital mast cell tumour): very rare, mainly on body trunk and pigmented. Sometimes fluctuant and ulcerated.
- Epidermal naevus: benign, usually flat hyperkeratotic papular condition.

Diagnosis

Biopsy (sectional or excisional where feasible) – histopathology is pathognomonic.

Treatment

Complete surgical ablation under general anaesthesia followed by mesh, split- or full-thickness skin grafting of the resultant defect can be used but a high failure rate is common. They may sometimes respond to radiation teletherapy or brachytherapy.

Prognosis

These tumours are particularly difficult to resolve. Recurrences are common unless surgical excision is meticulous and the lesions occurring at the commonest sites on the limb make this problematic. Many are progressive so early diagnosis and treatment gives the best chance.

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SECONDARY SKIN DISORDERS IN NEWBORN FOALS

Congenital hypothyroidism

Profile

This generalized disorder results from a paradoxical hypothyroidism due to excessive iodine. The condition occurs most commonly in foals born to mares receiving abnormally high dietary iodine (including seaweed) and other mineral supplements with high iodine concentrations during the early/mid stages of gestation. Thyroid hormone deficiencies will affect almost every developing organ but the main visible effects are related to cutaneous and musculoskeletal deformities.

Hyperiodic hypothyroidism is a well-recognized state in newborn foals presented with multiple skeletal abnormalities, poor growth (dysmaturity), skin abnormalities and prominent goitre. Skin abnormalities are seldom the most prominent signs. Cutaneous manifestations are recognized as epidermal atrophy and abnormal keratinization and hair formation.

Clinical signs

The foal is dysmature, often with severe musculoskeletal deformities (including fused joints, brachygnathism, and tendon laxity). The skin is often abnormally soft, silky and thinner than normal foals and the hair coat is thin and sparse. Some cases may have extensive alopecia and severe seborrhoea but it is difficult to attribute this to hypothyroidism alone.

A prominent goitre is invariably present. This sometimes leads the stud owner to believe that the mare is iodine deficient and so further supplements are prescribed the next year – making matters worse, not better!

Differential diagnosis

- Prematurity/dysmaturity: no thyroid enlargement. Gestational age and size for age are major factors. Hoof wall immaturity, and mentation are possible aids but differentiation can be difficult.
- Multiple congenital defect syndromes: obvious physical and mental defects.
- Neonatal septicaemia: febrile, systemic inflammatory response syndrome (SIRS), severe illness and possible joint and umbilical involvement.

Diagnosis

- Signs of musculoskeletal deformities associated with an obvious thyroid enlargement (goitre) present at birth are almost pathognomonic.
- Thyroid function in the mare is invariably normal and within hours of birth single and dynamic thyroid function tests in the foal are usually normal. Thyroid function tests are therefore not helpful.
- Diagnosis is best based on the clinical evidence and the history of iodine supplementation.
- Iodine estimations are seldom diagnostic (and are very difficult to perform adequately).
- Careful examination of the mare and assessment of her diet through pregnancy is usually sufficient to identify the cause.

Key points: Congenital hypothyroidism



1. An important cutaneous manifestation of an internal disorder. Hyperiodic hypothyroidism results in multiple congenital musculoskeletal abnormalities and a prominent goitre is usually visible at birth.
2. The skin signs are non-specific and of minor consequence. The hair shafts are thin and hair density is low. Some cases are almost alopecic and some scaling and flaking may be seen.
3. Diagnosis is simple and based on the other evidence of thyroid disease. Biochemical evidence of hypothyroidism is seldom obtained if the tests are performed after 24 hours of age.
4. Treatment is impossible – even thyroid supplementation has no beneficial effect.
5. The prognosis for affected foals in poor – few survive usefully to be effective athletes.

Treatment

No treatment is effective. A few mildly affected foals will mature but most have significant musculoskeletal abnormalities and seldom grow well. The skin signs are largely irrelevant to the prognosis for the foal.

Prevention

Careful dietary control of iodine intake is vital. Mares will seldom have any possibility of clinically significant iodine deficiency if the diet is even only reasonably normal. Dietary supplements containing iodine or seaweed extracts should *not* be fed to pregnant mares.

Ulcerative dermatitis, thrombocytopenia and neutropenia syndrome

Profile

This is a recently described transient but alarming syndrome affecting neonatal foals of no single breed, sex or colour. Increasing numbers of cases of this very interesting condition are being recognized in foals less than 4 days of age. The aetiopathogenesis is uncertain but an alloantibody-related subepidermal clefting is consistent with the signs. Cases have been reported in foals delivered to mares suffering from pemphigus foliaceus (Schott & Petersen 2005). The source of the antibody is likely to be colostral ingestion (Perkins et al 2005). The panel of signs includes neutropenia and thrombocytopenia but as yet it is unclear what is the primary target for the alloantibodies.

Clinical signs

The foal is usually born normally at full term and has a normal adaptive period. The first signs present at 48–72 hours of age. Skin crusting around the muzzle, eyes and axilla and inguinal regions develops and even minor handling of the skin causes significant trauma. Oral ulceration

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Key points: Ulcerative dermatitis, thrombocytopenia and neutropenia syndrome



1. A rare but increasingly important syndrome affecting newborn foals (<4 days of age) possibly related to colostral IgG intake.
2. Oral and possibly skin bullae, crusting, decreased nursing and haemoptyalism ('slobbering with blood') are the primary presenting signs. Less obvious bulla formation occurs on the head, body and limb skin. Extensive cutaneous and mucosal petechial and ecchymotic haemorrhages occur (with nodular haemorrhages in the oral mucosa in particular). Usually the foals remain reasonably alert.
3. Diagnosis is based on the definitive clinical appearance and the severe bulla and 'nodular'/'papular' mucosal haemorrhages.
4. The condition is transient with spontaneous recovery occurring. Supportive corticosteroids, antibiotics and anti-ulcer treatment are helpful measures in hastening recovery but may not be required.
5. The prognosis for affected foals is excellent in spite of the apparent severity of the early signs – most cases are significantly improved within 2–5 days.

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and bulla formation are major features resulting in salivation with blood **(Fig. CD10 • 4A–D)**. The foals go off suck but may remain alert.

Differential diagnosis

- Prematurity/dysmaturity: depression and lack of skeletal, neurological and cutaneous maturity. No bullae and few petechial haemorrhages unless the foal is also septic.
- Epidermolysis bullosa: very rare, persistent bullous skin disease affecting prescribed breeds.
- Neonatal septicaemia: febrile, systemic inflammatory response syndrome (SIRS), severe illness and possible joint and umbilical involvement.

Diagnosis

- Diagnosis is best based on the clinical evidence and the history of rapid-onset cutaneous and mucosal bulla formation.
- Biochemistry reveals a normal or excellent colostral passive IgG status with a mild to severe neutropenia and thrombocytopenia.

Treatment

Supportive antibiotics and anti-ulcer treatment seems to be sensible but septicaemia is not a reported complication possibly because of the relationship to good colostral immunity. Corticosteroids are thought to be helpful. Most cases are self-limiting within 14 days.

Prevention

No definitive advice can be given at present but it does seem sensible to use a different stallion at subsequent breedings and/or to use an alternative source of colostrum for the foal. However, by the time the foal shows signs it is usually too late to make these adjustments.

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PRIMARY CONGENITAL/HEREDITARY CONDITIONS IN OLDER FOALS AND ADULTS

Linear keratosis/linear alopecia

Profile

Linear keratosis is an idiopathic, possibly inherited dermatosis characterized by unilateral, linear, vertical bands of hyperkeratosis on neck, thorax and upper hind leg. The disorder is rare and its pathogenesis is unclear. There may be some heritable tendency but it is seen in many breeds to varying extents (Moriello et al 1998). Most cases occur in Quarterhorses between 1 and 5 years of age but Thoroughbred and other breeds are occasionally affected. A heritable form is encountered in Belgian and Quarterhorse breeds.

This is a relatively common disorder but it is often overlooked. The precise nature of the disorder is unknown but linear keratosis is otherwise very similar to the condition known as linear epidermal naevus. A particular form also occurs on the palmar aspect of the hind cannons from the fetlock upwards (see p. 242).

Linear alopecia is a similar disorder in which alopecia is the main sign. It is seen most often in Quarterhorses and, again, an inherited aspect has been proposed (Fadok 1995).

Although both disorders are probably present from an early age, they are usually only noted when the horse is clipped out or is being groomed during summer months. Clinical signs are obvious in most cases and they usually remain constant for many years (if not for life).

Clinical signs

The condition should be identifiable at some stage in the first 5 years of life but the lesions vary markedly in their severity and milder cases may pass unnoticed for longer.

Signs include one or more vertically arranged bands of persistent alopecia and/or hyperkeratotic papules and plaques associated with scaling/crusting most often occurring on the neck (Fig. 10.9) or the buttock (Fig. 10.10).



Figure 10.9 Linear keratosis in a single well-defined line down the side of the neck.



Figure 10.10 Linear keratosis showing multiple linear areas on the buttock (note that this was only obvious after the hair coat had been clipped away).

Key points: Linear keratosis/linear alopecia



1. A common but easily overlooked and poorly recognized disorder of keratinization. Thoroughbreds and Quarterhorses are most often affected on the sides of the neck, shoulder and buttock regions.
2. Usually only a few linear bands of alopecia and/or hyperkeratinized skin are identified. Neither pruritus nor pain are features but some early cases show mild discomfort. The lesions are non-inflammatory and may progress slowly or remain static.
3. Diagnosis is often delayed until later in life and is most often reported after clipping of the hair. Diagnosis can be made on biopsy but the benign, static and non-inflammatory nature of the disease is usually enough to support the diagnosis.
4. Treatment is neither indicated nor possible at the current time. Keratolytic shampoos and synthetic retinoids may be helpful but often serve little benefit. They may in fact simply widen the affected area and make it painful!
5. Prognosis is excellent – affected horses are not affected at all and it is best regarded as a cosmetic blemish.

Some cases start as small, focal, papular areas of hyperkeratosis and alopecia but this stage is seldom noticed. These gradually coalesce into the more obvious and characteristic linear lesions. The bands of abnormal skin vary in width and in length but are most obvious on the sides of the neck, shoulder and the lateral trunk and thorax. Lesions may be present on the buttock region and occasionally on the upper limb regions.

The lesions are asymptomatic with no pruritus or pain.

Many cases are only detected incidentally after clipping out and may give the impression of a healed traumatic lesion/scratch, etc. They seldom progress or improve over time.

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Differential diagnosis

- Chronic hyperkeratosis from irritant drugs, e.g. mercurial blister or repeated trauma: history of chemical application is usually significant.
- Dermatophilosis (*Dermatophilus congolensis*): more extensive and affects horses maintained in wet conditions. Resolves with treatment.
- Clipper rash/recent trauma (e.g. scratch): history of occurrence after clipping.
- Scar: history of wounding and more cutaneous involvement.
- Verrucose sarcoid: other lesions present and seldom linear in nature.

Diagnostic confirmation

- Clinical appearance is very typical.
- Biopsy shows variably regular, papillated epidermal hyperplasia with marked compact orthokeratotic hyperkeratosis. Primary change is a lymphocytic mural folliculitis (Stannard 1994b). The condition is usually non-inflammatory.

Treatment

No curative treatment is possible (and in any case it is seldom warranted given the trivial nature of the disease). Some cases benefit from retinoids (e.g. tazarotene) (Rosencrantz 1995) or keratolytic/anti-seborrhoeic shampoos and topical creams (such as icthymol or coal tar extracts) applied locally. The extent of hyperkeratosis can be reduced with application of retinoids such as tazarotene but as these are difficult to apply carefully enough and as they cause localized (temporary) hair loss, there is little to be gained. Any improvement is in any case temporary. These treatment may reduce the obvious keratosis and thin the hyperplastic epidermis but hair growth will not be normalized and indeed many of the treatment options may result in further alopecia. Probably the best treatment is benign neglect!

The condition is persistent but as it causes no difficulty for the horse the main problem is the cosmesis. The prognosis for a cure is therefore poor but many horses simply live with the condition without any obvious harm.

Epidermal naevus

Profile

An epidermal naevus is a form of hamartoma and is a well-defined developmental hyperplasia of the epidermis. Many different naevus types can occur and some of the conditions are given special status with their own name and description.

There is no obvious breed, colour or sex predisposition but feathered breeds are possibly more often affected. Often they remain static for life but as they are frequently subjected to unnecessary treatments, gradual exacerbation and extension occur in many cases.

Clinical signs

Most often these are found on the plantar aspects of the hind cannons as a linear tapering area of thickened hyperkeratotic skin (Fig. 10.11). There may be some hair thinning. Some horses have concurrent mite infestation (see *Chorioptes equi*, p. 191) and although the horse may then show some pruritus at the affected site, the mites are not congregated here specifically. Most cases have little or no pruritus and no pain associated with the condition unless there is skin cracking, secondary infection and inflammation. Often then there is also some serum exudation and consequent crusting and hair matting.

Differential diagnosis

- Congenital vascular hamartoma/cavernous haemangioma: localized, fleshy, highly vascular hamartoma of blood vessel tissues most often on the distal limb but also on the body trunk of newborn foals.
- Congenital (viral) papilloma: isolated, single (or few) papillomatous scaly mass on the skin of newborn foals supposedly caused by an equine papillomavirus.

Key points: Epidermal naevus



1. An unusual keratinizing skin disorder usually restricted to the plantar metatarsal region.
2. An area of partial alopecia and thickened skin with prominent scaling and flaking is usually the main sign. The condition is non-inflammatory and there is no pain or pruritus unless the area is secondarily infested with mites and is then subjected to self-trauma.
3. Diagnosis is based on clinical features alone and biopsy is disappointingly non-specific.
4. Treatment is not indicated but keratolytic shampoos and synthetic retinoids may be used if the condition becomes severe. However, repeated treatments will usually result in progressive deterioration and in some cases the treatment may be a painful process.
5. The condition is best regarded as a cosmetic blemish.



Figure 10.11 A typical linear epidermal naevus on the plantar metatarsal region of a middle-aged mare. She had been treated over some years for various skin infections without any material change.

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- Linear keratosis: this is probably a particular form of naevus with a linear keratinized or alopecic nature. The condition is not inflammatory and is symptomless.
- Cannon keratosis: non-progressive, circumscribed plaques of scaling, crusting and hyperkeratosis on the dorsal aspect of (hind) cannons. Not associated with stallions in particular in spite of its name 'stud crud'.
- Idiopathic carpal/tarsal hyperkeratosis (mallenders and sallenders): a common naevus-like condition restricted to the palmar carpal and dorsal tarsal skin.
- Chronic rubbing/callus (induced epidermal hyperplasia) most often due to parasite infestations: repeated rubbing from contact with harness or through self-trauma (pruritus) in focal areas may be very similar but prevention of trauma resolves the condition.
- Pemphigus complex: other sites will invariably be affected by scaling and alopecia; characteristic histology.
- Generalized granulomatous disease (sarcoidosis): generalized scaling and systemic signs.

Diagnosis

- The chronic nature of the condition and its variable skin inflammation are characteristic.
- Biopsy reveals a non-specific compact orthokeratotic (anuclear) and parakeratotic (nucleated) epidermal hyperplasia.

Treatment

Treatment is palliative and recurrence is invariable. Controlling parasitic infestations and any secondary bacterial infections seems an important aspect in limiting the severity of the signs. Topical retinoids can be used but the effects are temporary and they may cause marked skin inflammation in any case. Selenium-based shampoos will loosen and remove much of the superficial flaking and scaling and so this can be a helpful long-term management measure.

Hereditary equine regional dermal asthenia (HERDA; hyperelastosis cutis/cutaneous asthenia/Ehlers–Danlos syndrome/dermatosparaxis)

Profile

This group of autosomal recessive, inherited, connective tissue diseases occurs principally in young Quarterhorses and appears at birth or shortly afterwards (Bridges & McMullen 1984, Brounts et al 2001). Thoroughbreds and Arabian horses have been diagnosed with this condition also (Rashmir Raven et al 2004).

At least nine different subtypes occur in humans and it has been classified on clinical, genetic and biochemical differences. The precise nature of the genetic problem is not yet known. Alterations and disorientation of the dermal collagen fibrils are thought to be the main pathological basis for the condition (White et al 2004). There is decreased collagen as well as fragmentation and disorientation of collagen fibres (Stannard 1994a).

A second recognizable syndrome may occur in newborn foals in which an unexplained wound draws attention to a region of abnormal skin attachment. This syndrome is not yet widely recognized and it is uncertain if it falls into this category rather than epitheliogenesis imperfecta (congenital cutaneous aplasia) – see above.

Clinical signs

Clinical signs develop before 1 year of age with no sex or colour predilection. The severity of the condition is very variable in individual horses. Many cases are only diagnosed around 2 years of age – presumably due to the fact that this is the first time saddles are placed on the animal.

Loose, wrinkled, hyperextensible skin over limited areas of the body or more generalized, giving a wrinkle-skin appearance to the whole body (Fig. 10.12). Most cases show several, well-defined roughly circular areas of damaged skin, between 5 and 30 cm in diameter, that first become evident over the back, sacrum or sides of the body trunk (Fig. 10.13). Some horses may simply appear to have an excess of loose skin, which can be picked up and tented easily, with no severe clinical consequences. There is a remarkable lack

Key points: Hereditary equine regional dermal asthenia

1. A rare but significant congenital and hereditary disorder of the skin of Thoroughbreds and Quarterhorses characterized by recurrent skin trauma resulting from trivial skin insults. Other breeds are less commonly affected.
2. Clinical signs are usually delayed until the skin is subjected to significant trauma such as saddle placement, etc. Large wounds develop that either heal very slowly with severe scar formation, or do not heal at all.
3. Diagnosis is made on the characteristic history and breeding. Biopsy can be helpful but the condition is not yet well characterized pathologically.
4. Treatment is impossible. Attempts to graft the defects or remove the affected skin are fruitless.
5. The prognosis is hopeless although a few cases may be managed provided the horse is not ridden. Wound care is a long-term challenge.



Figure 10.12 Hereditary equine regional dermal asthenia (HERDA) (hyperelastosis cutis/cutaneous asthenia/Ehlers–Danlos syndrome/dermatosparaxis) showing the thin, folded and fragile skin.

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of pain unless the margins of the lesion are stretched and pruritus is not a feature.

Repeated subcutaneous separations and bruising or even tearing of the skin occur even with minor trauma. Saddle pressure/movement or girth tightening may create relative movement differences (shear forces) between the skin and the subcutis. Subcutaneous haematoma and abscesses occur. Limbs may be affected and then the complaint is usually of frequent wounds that seem inordinately severe for the extent of the cause and then fail to heal as expected.

Damaged or torn skin repairs very slowly with a weak, fragile scar formation (and sometimes completely fails to heal) (Fig. 10.14). Sutures do not 'hold' well and frequently tear out as they are tightened. Granulation tissue at the affected sites is usually sparse and of poor quality. Epithelialization is slow and contraction is largely absent.

Recurrent skin and other infections also occur in some affected cases, suggesting a degree of immunocompromise although this may be due to cutaneous immune problems.



Figure 10.13 Hereditary equine regional dermal asthenia (HERDA) (hyperelastosis cutis/cutaneous asthenia/Ehlers–Danlos syndrome/dermatosparaxis) with extensive skin damage due to minor trauma.



Figure 10.14 Hereditary equine regional dermal asthenia (HERDA) (hyperelastosis cutis/cutaneous asthenia/Ehlers–Danlos syndrome/dermatosparaxis) showing extensive scarring and residual poor skin quality after trauma (same horse as Fig. 11.4, some 3 months later).

At present the condition encountered in neonatal foals (see p. 231) is pathologically undefined; it is entirely possible that this condition reflects a variation on the HERDA syndrome.

Differential diagnosis

- Epitheliogenesis imperfecta/cutaneous agenesis: obvious skin deficits and ulcers from birth or shortly afterwards.
- Persistent irritation: scarring and self-trauma results in skin thickening and folding rather than overt necrosis.
- Dehydration and rapid weight loss (loss of subcutaneous fluid/fat resulting in increased skin movement): this does not cause necrosis although skin 'tenting' may be an indicator.
- Selenium poisoning: sloughing of hooves and generalized hair loss including mane and tail hairs.

Diagnostic confirmation

- Clinical appearance of loose skin with hyper-fragility in a Quarterhorse.
- Biopsy may indicate collagen changes but histopathology has not been well documented in the horse. It is important to take deep biopsies because sometimes the superficial skin is normal. Thinning of dermis and fragmentation of collagen are the most common reported findings. Collagen distribution patterns revealed by special stains and electron microscopy are reported to be abnormally wide and random.
- Foals can be tested for the disease from a urine sample.

Treatment

Apart from minimizing trauma and careful, sympathetic wound management, there is no treatment. Debridement of necrotic skin is tempting but closure by suture seldom helps with rapid dehiscence; it is possibly better to leave the necrotic skin in situ to act as biological dressing. The sites most often affected mean that dressings are difficult. Wounds in affected areas do, however, benefit from collagen-based dressings and a moist wound healing environment. Healing may be delayed for long periods in spite of the best possible wound care. Skin grafting is theoretically possible but often fails to take over the affected area.

Ultimately the disease is best regarded as a fatal condition if it is either extensive or severe (or both). Some milder affected animals may survive without serious problems; these are a potential danger for breeding programmes.

Prevention

The recessive nature of the genetic abnormality means that breeding from two carrier horses has a one in four chance of producing an affected foal. Breeding from a non-carrier stallion and a carrier mare will not result in an affected offspring and will bias the population towards a reduction in the number of carriers. Under no circumstances should an affected horse be used for breeding. Neither parent of an affected foal should be used for breeding – both have to be carriers if the foal is affected. It is not yet possible to identify carrier horses and so breeding programmes are still liable to produce affected animals from time to time.

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Hypotrichosis/mane and tail dystrophy (follicular dysplasia)

Profile

Hypotrichosis is a rare hereditary disease found most often in some lines of Arabian horses in which there is less than the normal amount/density of hair. It does not appear to be linked to hair colour. It may affect other breeds, and lesser forms in which there is simply a reduction in hair density over variable areas is possibly more common than is appreciated. In some cases the changes can result in severe hair loss and bizarre hair growth patterns.

A commoner manifestation of hair dystrophy/thinning is well recognized in the Appaloosa breed, where the long tail hairs in particular are sparse and brittle. This is regarded as a normal trait in some lines of the Appaloosa horse. Mane and tail dystrophy occurring simultaneously have been recorded in a variety of breeds.

Clinical signs

Hypotrichosis Permanent thinning or almost complete lack of hair occurring particularly around the eyes and extending ventrally towards the muzzle is characteristic (Fig. 10.15). The skin is sometimes slightly scaly and may feel thinner than normal. It seldom affects body hair to any extent. Many cases have no obvious skin abnormality apart from the absence of hair.

Mane and tail dystrophy (follicular dysplasia) Idiopathic, non-inflammatory loss of mane and tail hairs with residual shorter and sparse hairs. Eventually most cases lose most if not all the mane and tail hairs (Figs 10.16 and 10.17).

Differential diagnosis

- Selenium poisoning: only affects mane and tail initially. Access to excessive selenium in feed supplements or seleniferous plants. Sloughing of hoof capsule.



Figure 10.15 Hypotrichosis – poor hair density and quality. This had been present from birth but became more noticeable with age.

Key points: Hypotrichosis/mane and tail dystrophy

2 3 10 15 20 24

1. Generalized hypotrichosis is possibly commoner than is appreciated but the severe forms are rare. The 'mane and tail' disorders are more common and affect the Appaloosa breed most often.
2. Clinical signs are variable depending on the severity and extent but generalized hair loss in a very young foal, or mane and tail loss in older Appaloosa horses is typical.
3. Diagnosis is usually simple. Biopsy can help to confirm the scarcity of functioning hair follicles but usually they are themselves normal. In the 'mane and tail' form characteristic follicular pathology may be recognized.
4. No treatments are possible.
5. The conditions do not affect the horse and so it is best viewed as a cosmetic nuisance.





Figure 10.16 Mane and tail dystrophy shows as a sparse, thin hair density on either the mane **(A)** or the tail **(B)** or both with the tail having a rat-tail-like appearance with few, shorter than normal hairs. In some cases the tail is completely bald.

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- Mimosa poisoning: access related.
- Zinc, copper and iodine deficiencies: rare cases of hair loss and affect body hair more than mane and tail.
- Pruritic damage (e.g. insect bite hypersensitivity syndromes)/chronic/recurrent mild abrasion (e.g. from hoods or other tack): seasonal or circumstantial changes with return to normal hair growth when cause is avoided/removed.
- Chronic anhidrosis: sporadic and affects body hair more than mane and tail. Circumstantial factors relating to movement.
- Chronic ectoparasite infestation: pruritic damage from lice/mites seldom causes mane and tail hair loss.
- Tail injury (e.g. bandaging pressure): history and evidence of scarring.
- Tail pulling/chewing by other horse: hairs that are pulled show noticeable regrowth. Broken hairs can be detected microscopically. Visible irrefutable evidence of chewing!
- Alopecia areata: mane and tail can be affected but mostly it affects patches or larger areas of skin on the body and head. Distinctive pathology.

Diagnostic confirmation

- Clinical appearance and history of sparse hair coat since birth with very little alteration over time.
- Appaloosa or part-bred Appaloosa breeding.
- Biopsy shows reduced numbers of hair follicles but normal skin structure otherwise.

Treatment

None is available. Apart from appearance, there appear to be no detrimental systemic effects associated with this condition. Breeding programmes have failed to identify this trait and it is now accepted in the Appaloosa breed in particular as being normal.

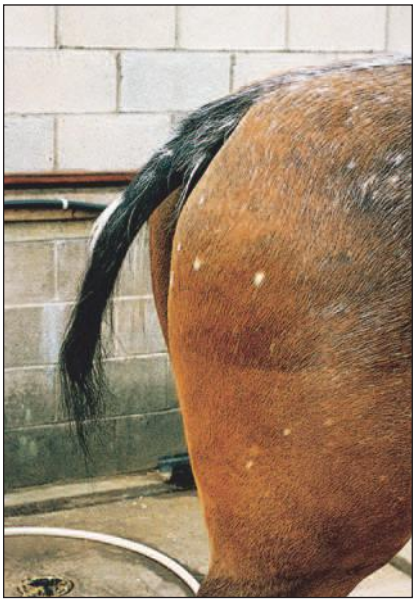


Figure 10.17 A severe case of follicular dysplasia in a 14-year-old Appaloosa gelding in which progressive hair loss developed over 10–12 years.

Appaloosa parentage syndrome

Profile

Appaloosa cross-bred foals often do not have full colour change until 3–4 years of age and then a fading of the spots is sometimes seen so that the horse has a progressive fading of the hair colour without any associated changes in hair density.

Clinical signs

A previously whole-coloured horse develops white hairs and sometimes overt spots over the rump in particular. Patchy progressive depigmentation of muzzle, eyelids, vulva and prepuce occurs. Hair and skin colour fades progressively with age. The hooves may show banded depigmentation particularly around the hoof wall.

Mane and tail hairs may thin out (see mane and tail dystrophy above) but colour changes in these are not prominent.

Differential diagnosis

- Systemic lupus erythematosus-like syndrome: generalized alopecia with pigment loss and some systemic signs. Affected horses are unwell.
- Vitiligo/Arabian fading syndrome: facial and perineal pigment loss and hair loss most obviously over the sides of the face. Otherwise asymptomatic.
- Age-related greying of hair: hair density is unaffected.
- Trauma or heat/cold related leukoderma/leukotrichia: scarring and exposure to factors that damage hair follicles and melanocytes.

Diagnostic confirmation

- Clinical signs coupled with Appaloosa parentage (up to three or more generations away).
- Biopsies are unrewarding.

Treatment

There is no treatment known.

It is widely regarded as a normal trait in Appaloosa cross horses.

Key points: Appaloosa parentage syndrome

2 10 13 16 20 24

1. A breed-related disorder confined to the Appaloosa breed in which genetic aspects are involved in the slow development of the definitive colour pattern and a later progressive dilution of the hair coat colour.
2. The clinical features are a gradual development of the spotted pattern over 3–4 years and then progressive fading of the colour with age.
3. Diagnosis is simple and based on the breeding and known genetic tendency in specific lines.
4. Treatment is not possible.
5. The trait is sometimes regarded as desirable but in other cases it is viewed as normal for the breed.

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Arabian fading syndrome (pinky Arab syndrome)

Profile

Occurs spontaneously most often in Arabian horses at any age (2–23 years) but has also been observed in Welsh mountain ponies and Clydesdale horses. The condition is largely symptom-free apart from the loss of pigmentation and hair in some cases and may be similar or identical to vitiligo. The latter condition is usually reserved for the more localized periocular condition that occurs in a larger number of breeds and is most often facial only.

The face and perineum are more often affected. Grey horses are over-represented.

There is no known link to copper deficiency or any other pathology but a relationship to endocrine changes in pregnancy has been suggested (Thomsett 1991). This does not correspond with the occurrence of the condition in an almost equal number of males (geldings and stallions).

Clinical signs

Anular or irregular areas of depigmentation of muzzle, lips, periorbital regions (Fig. 10.18) and occasionally around perineum, sheath and hooves. Smaller areas may coalesce into extensive loss of pigment (and possibly hair also). Affected horses occasionally show body patches of depigmentation which may wax and wane in intensity but are usually permanent. The pink skin may be very liable to sunburn and it has an increased susceptibility to squamous cell carcinoma.

Differential diagnosis

- Copper deficiency: extremely rare but often mentioned as a potential cause of pigment loss.
- Systemic (cutaneous) lupus erythematosus-like syndrome: generalized scaling, pigmentary incontinence and multi-systemic involvement.



Figure 10.18 Arabian fading syndrome (pinky Arab syndrome). Note the normality of the skin apart from the hair loss and the pigment changes.

Key points: Arabian fading syndrome



1. A common, genetically linked condition affecting primarily grey Arabian horses. Clydesdale and other breeds may be affected also. There is no link to copper deficiency or any other known potential aetiology.
2. The condition is mainly seen around the face and in the perineal region. It is symptom-free – the skin simply loses its pigment and sometimes the hair also.
3. Diagnosis is not usually difficult but biopsy will confirm the loss of melanocytes and in some cases hair follicle density.
4. Treatment is impossible. Tattooing and skin protection with masks or rugs/sheets are the only options.
5. The main implications lie in the cosmetic aspects and the increased tendency to actinic dermatitis and squamous cell carcinoma in the affected skin.

- Vitiligo (may be a variant of the same disorder): idiopathic symptomless loss of pigmentation on the face, periorbital skin and perineum in particular.
- Appaloosa parentage syndrome: breed related and seldom marked changes – usually much more gradual.
- Leukoderma/leukotrichia following trauma or freezing or radiation: history of trauma or treatment.
- Discoid lupus: photo-exacerbated condition with discoid epidermal lesions on face and limbs.

Diagnostic confirmation

- Clinical appearance in Arabian or Welsh mountain pony breeds.
- Wetting the horse with water allows depigmentation to become more obvious.
- Skin biopsy shows loss of melanin from epidermal basal cells.

Treatment

No effective treatment is suggested. Copper supplements must be administered with great care and in any case seldom have any benefit, although some reports have suggested an improvement may be gained with this and vitamin supplements (Thomsett 1991).

Tattoo or hair dyes can be used to mask the true effects but its progressive nature makes it almost impossible to hide permanently. Some horses have shown partial spontaneous recovery. The condition has no material implication for the horse apart from the increased sensitivity to sunlight and therefore some possibility that it could be more susceptible to squamous cell carcinoma.

Dentigerous cyst

Profile

Congenital or developmental swellings occurring between the base of the ear and eye (temporal region) arising from tooth germ tissue. Occasionally they are also found on the cranial vault or maxillary sinus and contain enamel-forming

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tissue. They are lined with stratified squamous epithelium and may contain one or more teeth. Drainage may occur near the ear or through the skin adjacent to the cyst.

Clinical signs

The most topical sign is a discharging sinus tract on the anterior margin of the ear flap producing a sticky, clear or slightly milky fluid (Fig. 10.19). In some cases there is nothing else to find although a probe will pass some distance down the tract.

In some cases a fixed firm/hard nodule can be palpated between the eye and the ear in the temporal region with an associated fluctuant cyst or a persistent discharging sinus (Fig. 10.20). There may be a radiographically obvious dental remnant either associated with the cranium or lying in the adjacent tissues. The former are often fixed firmly while the latter are more mobile.



Figure 10.19 Dentigerous cyst with a typical fistula and discharge on the margin of the pinna.



Figure 10.20 The scissors point to an abnormal solid structure which was identified radiographically as an aberrant tooth in a pseudo-alveolus attached to the cranium.

Key points: Dentigerous cyst



1. Rare congenital abnormality resulting from ectopic dental tissue.
2. A discharging sinus on the leading margin of the pinna is typical and a few cases have a detectable, dental-like, firm swelling lying against the cranium.
3. Diagnosis is simple but a radiograph and a positive and negative contrast study may be helpful.
4. Removal of the cyst is relatively simple but a dental remnant and a pseudo-alveolus on the cranium can be much more problematic.
5. The prognosis is excellent and some cases can simply be left with the minor 'cosmetic' discharge.

Differential diagnosis

- Other cystic structures: rare and none are as typical as the dentigerous cysts.
- Neoplasia: there is no other condition that produces these signs but sinus cysts and infected sarcoids could be mistaken for it.
- Abscess (usually *Streptococcus equi equi*): *S. equi* pus discharges and is usually associated with lymph node abscessation.

Diagnostic confirmation

- Characteristic appearance.
- Radiography and ultrasonography. There may be obvious dental tissue and even an alveolus attached to the cranium.

Treatment

Cautious surgical removal is required (fracture of the cranial vault can occur during attempts to remove the aberrant tooth).

Dermoid cyst

Profile

Congenital or hereditary cutaneous cysts thought to be due to embryonic displacement of ectoderm into the subcutis. Occurs singly and most commonly along dorsal midline. No breed predilection has been established (Wellington & Scott 1991). Paso Fino horses may have a slightly higher prevalence. Usually young horses (<18 months old) are affected.

A particular form of dermoid also occurs in and around the eye. Dermoids may occur on the conjunctiva (especially at the medial canthus and caruncular areas) or on the limbal conjunctiva or the cornea itself. They can be hairless or haired but show variable skin structures including sweat glands, hair follicles, sebaceous glands and fat.

Clinical signs

Single or multiple, firm to fluctuant, smooth, dermal nodules (10–15 mm diameter) with normal overlying haired skin. Most are on the dorsal midline between withers and

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rump (Fig. 10.21 and **Fig. CD10 • 5**). Lesions contain a soft cheese-like grey material and sometimes coiled hairs.

Differential diagnosis

- Epidermoid cysts: localized, fluid-filled fluctuant but sometimes tense, spherical cysts usually around the nasal region.
- Dentigerous cysts: discharging sinus tracts on the anterior margin of the pinna and in the event of dental remnant a discrete swelling somewhere below the ear on the side of the calvarium.
- Hypodermiasis (*Hypoderma bovis*): isolated seasonal nodule, usually painful at first and possibly developing a central pore.
- Eosinophilic granuloma (with/without collagen necrosis): very common condition of adult horses showing isolated or confluent, myriad or few firm cutaneous nodules often but not exclusively restricted to the skin of the body trunk and neck.

Diagnostic confirmation

- Characteristic location and clinical appearance.
- Excisional biopsy shows a cystic wall lined with stratified squamous epithelium and often containing adnexal structures (see **Fig. CD10 • 5**).

Treatment

Total surgical ablation of cyst wall and contents is effective.

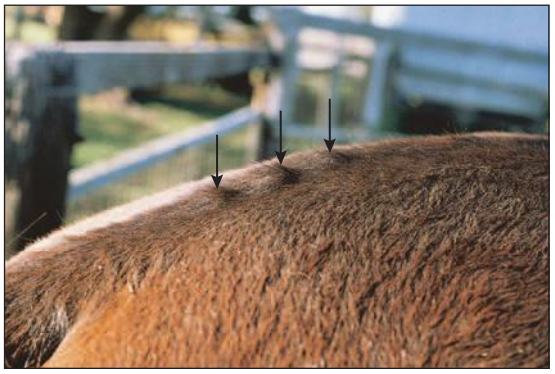


Figure 10.21 Three dermoid cysts in the skin of the croup.

Key points: Dermoid cyst

3 14 15 20 23 24

1 Unusual congenital cystic deformity of the skin usually restricted to single or a few nodular lesions in the dorsal midline. A particular form also occurs on the cornea or conjunctiva where they may be haired or non-haired.
2 The clinical features are usually distinctive – a few (or single), isolated, slow-growing nodules in the dorsal midline of young horses is characteristic. The conjunctival forms are easily recognized.
3 Diagnosis is usually simple but may be confirmed on biopsy or excisional biopsy.
4 Surgical excision is the only alternative to leaving them alone.
5 Prognosis is excellent; in the case of corneal dermoids the surgical scar may be significant.

Epidermoid cyst (atheroma)

CD

Profile

Developmental cystic structure of the epidermis. The commonest site is just above the caudal limit of the false nostril, where it is known as an atheroma. Other sites that are much more rarely affected include the limbs and the hwead.

Clinical signs

Freely movable, well-circumscribed, firm to fluctuant, (usually) solitary nodules (7–30mm diameter) occurring on head (the area dorsal to the false nostril is by far the commonest site) (Fig. 10.22) and legs. They contain a yellow to grey mucoid or porridge-like fluid and no hairs and often remain static for years. A few expand continually but slowly over years. Only very rarely do they reach a size sufficient to cause a problem with air flow in the ipsilateral nostril. It is usually a cosmetic problem only.

Key points: Epidermoid cyst

3 9 14 20 23 24

1 Common congenital and developmental condition of horses usually restricted to the region of the false nostril.
2 Soft, non-painful, non-inflammatory, fluctuant nodule lying subcutaneously in the region of the false nostril. A few can become large enough to interfere with air flow but this is extremely unusual.
3 Diagnosis is simple – there is nothing really that looks like this with a slowly developing cyst-like structure at this site.
4 Most can/should be left alone but surgical removal is possible – it is more difficult than it looks and may leave an equally unacceptable scar.
5 The prognosis is excellent.

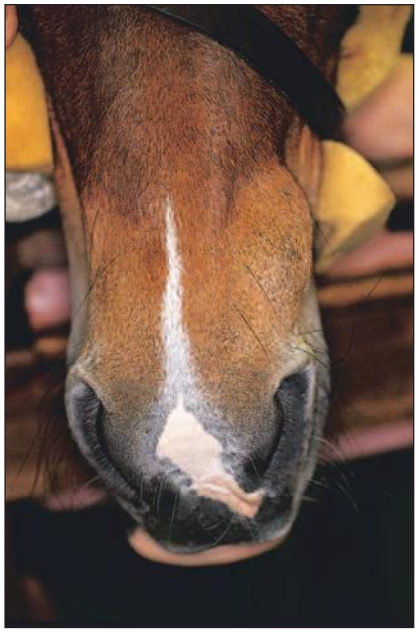


Figure 10.22 Atheroma (nasal inclusion cyst) in a typical location in the outer wall of the false nostril.

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Differential diagnosis

- Dentigerous and conchal cyst: located below ear and often with a discharging sinus on the leading margin of the pinna.
- Dermoid cyst: usually restricted to dorsal midline and associated with coiled hair, sebum and mild inflammation in a few cases.
- Foreign body reaction: inflammatory, usually solid swelling with a history of some traumatic episode.
- Nodular sarcoid: solid and sometimes involving the overlying skin (type B).

Diagnostic confirmation

- Non-painful, non-inflammatory fluctuant or firm swelling above false nostril is typical.
- Aspiration produces a grey or white sterile creamy liquid.

Histopathology shows an epithelial lining showing maturation and keratinization typical of epidermis *but* no adnexal structures.

Treatment

Surgical removal is effective.

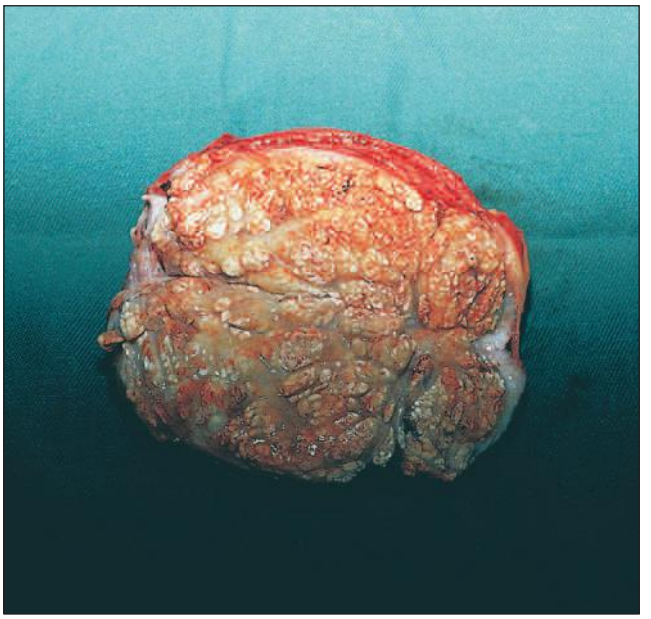


Figure 10.23 The typical appearance of a calcinosis circumscripta nodule surgically removed from the lateral aspect of the stifle.

Calcinosis circumscripta

Profile

Developmental/congenital condition of young horses (1–4 years) typically occurring over the lateral stifle area and less commonly over other joints such as the carpus and tarsus.

Clinical signs

This condition can be recognized by a firm, non-painful, non-pruritic, subcutaneous swelling up to 80 mm (or more) in diameter located over the lateral aspect of the stifle or gaskin (or other regions). The circumscribed mass, which comprises an aggregation of calcified granules in a milky-white crumbly base (Fig. 10.23), is sometimes clearly attached to the underlying ligament, muscle or joint capsule.

Differential diagnosis

- Acquired bursa: fluid-filled and ultrasonographically distinctive – aspiration reveals typical bursal fluid.
- Foreign body: can be calcified also and so can be difficult to differentiate – the site of the lesion and its history may be suggestive of a foreign body.
- Dermoid cyst: usually restricted to the dorsal midline or eye region.
- Calcification following trauma/haematoma: this can produce a 'plate' of calcified tissue, usually with relatively sharp margins and a history of a traumatic episode.

Diagnostic confirmation

- Radiographic and ultrasonographic examinations are characteristic.
- Biopsy shows multi-nodular deposits of minerals within fibrous and granulomatous tissue.

Treatment

Complete surgical removal under general anaesthetic is essential to prevent recurrence but is sometimes not as easy as it might seem initially. There is a high incidence of suture dehiscence (Stone 1990).

Key points: Calcinosis circumscripta

3 9 14 20 23 24

1. A rare developmental abnormality of unknown aetiology; possibly associated with trauma.
2. A firm swelling, sometimes mobile over the underlying tissues, often located over the lateral gaskin/stifle region is the most common presentation. Some masses are bound down to underlying facial tissues. Other sites can be involved also. Usually it is not associated with lameness.
3. Diagnosis is based on the typical appearance and the radiographic and ultrasonographic appearance.
4. Surgical removal is sometimes required but can be far more difficult than it first appears.
5. The prognosis is good whether it is left or removed.

Distichiasis/trichiasis

Profile

Distichiasis A row of eyelashes (cilia) originating from the tarsal glands abnormally positioned along the eyelid margin resulting in abnormal contact with the cornea.
Trichiasis Rubbing of facial hairs on the cornea/facial hair growing in a manner that results in corneal contact.

Both of these conditions are singularly rare in horses and more so as a primary congenital abnormality, being largely restricted to miniature pony breeds.

Note that the commonest condition of corneal inflammation caused by hair contact is congenital or acquired entropion.

Key points: Distichiasis/trichiasis

3 14 20 24

1. Rare congenital disorder largely restricted to the miniature pony breeds.
2. Clinical signs are often restricted to corneal damage and the pain associated with this.
3. Diagnosis is made by close inspection – a magnifying glass may reveal very fine hairs that may be almost invisible to the naked eye. The condition can be mistaken for entropion.
4. Treatment is usually by repeated plucking of the affected hairs but a more permanent result can be achieved by individual follicle cryoepilation or by surgical removal of the line of abnormal follicles in the affected lid.
5. Prognosis is guarded unless total ablation of the hair follicles can be achieved and most cases will require some ongoing treatments.

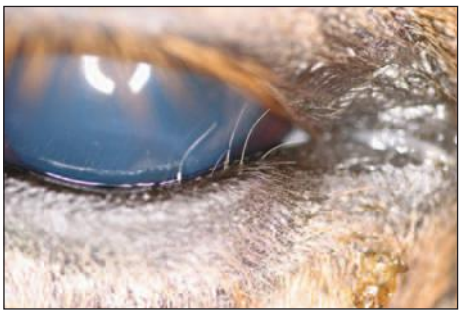


Figure 10.24 This miniature Shetland pony had a long history of ocular discharge with facial excoriation. Close examination revealed a row of abnormal eyelashes along the bottom lid; some were very robust and were visible directly while others could only be seen with magnification and outward turning of the eyelid. Surgical removal of the entire row cured the problem completely.

Clinical signs

The abnormal contact of hair with the cornea either as a result of abnormally located or directed eyelashes or abnormal facial hair direction is relatively easy to recognize in most cases. The main signs are associated with corneal discomfort or pathology. Corneal discomfort, lacrimation and possibly corneal ulceration are seen. Close examination (possibly with the aid of a magnifying glass) will reveal the hairs impacting the cornea (Fig. 10.24).

Distichiasis and trichiasis are clinically quite distinctive. Trichiasis usually has much thicker hairs and they are usually found emanating from the middle of the eyelid margin. Distichiasis usually has much finer hairs but sometimes they are more robust. The hairs emanate from abnormally located hair follicles inside the middle of the eyelid. The lower lid is far more often involved but the upper lid can be involved and so should always be examined carefully.

Differential diagnosis

- Entropion (primary or secondary): easily recognized and resolved.
- Meibomianitis: a discrete swelling or series of swellings in the margin of the eyelid, not usually associated with abnormal eyelashes.
- Scarring/distortion of the eyelid by scar or thickening/masses: history of eyelid trauma or obvious scarring is usually detected.

Diagnostic confirmation

Careful examination under a magnifying glass is usually diagnostic. Some of the hairs can be very fine and sparse in number so careful examination is essential. Holding the eyelids open during examination may mask the presence of the hairs as the lid is turned outwards; it is far better to examine the eyelid margins in their natural position as far as possible – a magnifying glass or loupe is very helpful.

Treatment

Conservative temporary treatment involves plucking out the abnormal hairs with 'eyebrow' tweezers (normal forceps are usually useless!). The hairs may be slow to grow back or may reappear quickly. The horse may resent repeated plucking.

Surgical treatment involves removal of the affected line of hair follicles by microsurgical excision of a small 'V'-shaped portion of the affected lid.

Cryoepilation involves focal microcryosurgical ablation of individual follicles and can be effective for isolated lashes. Many regrow over variable time so the owner will need to be advised that repeated procedures may be required.

Note

Additional skin disorders that can be encountered in neonatal foals include:

- neonatal/congenital viral papilloma (see p. 134)
- parturition trauma (spontaneous/mare savaging or predation/iatrogenic skin trauma due to dystocia)
- urine and faecal scalding.

References

Berthelsen H, Eriksson K 1935 Epitheliogenesis imperfecta neonatorum in a foal possibly of hereditary nature. Journal of Comparative Pathology and Therapeutics 48:285–286

Bridges C H, McMullen W C 1984 Dermatosparaxis in quarter horses. Proceedings of the American College of Veterinary Pathology 35:12–14

Brounts S H, Rashmir-Raven A M, Black S S 2001 Zonal dermal separation: a distinctive histopathological lesion associated with hyperelastosis cutis in a quarter horse. Veterinary Dermatology 12:219–224

Butler C M, Westermann C M, Koeman J P, Sloet van Oldruitenborgh-Oosterbaan M M 2006 The Fell Pony Immunodeficiency Syndrome also occurs in the Netherlands: A review and six cases. Tijdschrift voor Diergeneeskunde 131:114–118

Fadok V A 1995 Update on four unusual equine dermatoses. Veterinary Clinics of North America. Equine Practice 11:105–115

Frame S R, Harrington D D, Fessler J, Frame P F 1988 Hereditary junctional mechanobullous disease in a foal. Journal of the American Veterinary Medical Association 193:1420–1424

Johnson P J 1998 Dermatologic tumors (excluding using sarcoids) Veterinary Clinics of North America. Equine Practice 14:633–634

Knottenbelt D C, Pascoe R R 1994 A colour atlas of diseases and disorders of the horse. Mosby-Wolfe, London

Kohn C W, Johnson G C, Garry F et al 1989 Mechanobullous disease in a 2 month-old Belgian foal. Equine Veterinary Journal 21:297–301

Lieto L D, Cothran E G 2003 The epitheliogenesis imperfecta locus maps to equine chromosome 8 in American Saddlebred horses. Cytogenetics and Genome Research 102:207–210

Lieto L D, Swerczek T W, Cothran E G 2002 Equine epitheliogenesis imperfecta in two American Saddlebred foals is a lamina lucida defect. Veterinary Pathology 39:576–580

Lightbody T 2002 Foal with overo lethal white syndrome born to registered quarter horse mare. Canadian Veterinary Journal 43:715–717

Linder K E 2000 Mechanobullous disease of Belgian foals resembles lethal (Herlitz) junctional epidermolysis bullosa of humans and is associated with failure of laminin-5 assembly. Veterinary Dermatology 11(supplement):24–30

Moriello K A et al 1998 Diseases of the skin. In: Reed S M, Bayley W M (eds) Equine internal medicine. WB Saunders, Philadelphia

Page P, Parker R, Harper C et al 2006 Clinical, clinicopathologic, post-mortem examination findings and familial history of 3 Arabians with lavender foal syndrome. Journal of Veterinary Internal Medicine 20:1491–1494

Perkins G A, Miller W H, Divers T J 2005 Ulcerative dermatitis, thrombocytopenia and neutropenia in neonatal foals. Journal of Veterinary Internal Medicine 19:211–216

Rashmir Raven A M, Winand N J, Read R W et al 2004 Equine hyperelastosis cutis update. In: Proceedings of 50th Annual Convention of the American Association of Equine Practitioners, Denver, USA, p 1409

Richards A F, Kelly D F, Knottenbelt D C et al 2000 Anaemia, diarrhoea and opportunistic infection in Fell ponies. Equine Veterinary Journal 32:386–391

Rosencrantz W S 1995 Systemic/topical therapy. Veterinary Clinics of North America Equine Practice 11:127–144

Scholes S F, Holliman A, May P D, Holmes M A 1988 A syndrome of anaemia, immunodeficiency, and peripheral ganglionopathy in Fell Pony foals. Veterinary Record 142:128–134

Schott H C II, Petersen A D 2005 Cutaneous markers of disorders affecting young horses. Clinical Techniques in Equine Practice 4:314–323

Scott D W 1988a Dermatohistopathology. In: Large animal dermatology. WB Saunders, Philadelphia, pp 29–49

Scott D W 1988b Structure and function of the skin. In: Large animal dermatology. WB Saunders, Philadelphia, pp 1–4

Scott D W 2004 Skin of the neck, mane and tail of the curly horse. Equine Veterinary Education 16:201–206

Scott D W, Miller W M 2003a Congenital and hereditary skin diseases. In: Equine dermatology. WB Saunders, Philadelphia, p 628

Scott D W, Miller W H 2003b Structure and function, immune mediated disorders, congenital and hereditary skin disorders. In: Equine dermatology. WB Saunders, Philadelphia

Stannard A A 1994a Erosive and ulcerative diseases. In: Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatology, Newmarket, UK, pp 77–83

Stannard A A 1994b Linear keratosis. In: Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Stone W C 1990 The pathologic mineralization of soft tissue calcinosis circumscripta in horses. Compendium of Continuing Education for Practising Veterinarians 12:1643–1648

Thomsett L R 1991 Pigmentation and pigmentary disorders of the equine skin. Equine Veterinary Education 3:131–134

Wellington J R, Scott D W 1991 Equine keratinizing cutaneous cysts. Equine Practice 13:8–17

White S D, Affolter V K, Bannasch D L et al 2004 Hereditary equine regional dermal aesthenia ('hyperelastosis cutis') in 50 horses. Clinical histological, immunological and ultrastructural findings. Veterinary Dermatology 15:207–217

11

Immune-mediated/allergic diseases

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Inflammatory changes occur in the skin whenever it is injured. These changes are initially related to the specific cause and, secondarily, may relate to autologous changes which develop later. Regardless of the cause, be it heat, cold, infection due to bacterial, fungal, viral or parasitic organisms, radiation, trauma, or allergens, the skin response will be characterized by responses that involve blood vessels, tissue fluids and cells. These changes will be identifiable clinically as the cardinal signs of inflammation, namely heat, pain, redness, swelling and variable loss of function.

Protection of the host is by means of a structured immune response involving antigens, lymphocytes, antibodies, complement, and other mediators and effector cells. Their normal action is to restore natural body function. Tissues themselves have an immune privilege which severely curtails the extent of self-destruction. Under- or over-activity may lead to serious problems for the host. Under-response leads to failure to control infection and poor elimination of toxic by-products. Over-response causes a loss of, or a reduction in, the immune privilege of tissues (which is responsible for preventing the body destroying itself) and therefore an excessive inflammatory response develops which results in the production of autoimmune disease. Both of these responses are common features of skin disease.

The various ways in which the body can respond have been broadly grouped into four types of hypersensitivity (Gell & Coombs 1964) and these have been described in Chapter 1, p. 14). While they are fairly well defined, many reactions are complexes of these and so may show broad variations in clinical features. Types 1, 2 and 3 all rely on antigen–antibody reactions and therefore induce clinical effects following introduction of the antigen to a previously sensitized animal (in the case of type 1 hypersensitivity usually in less than 4 hours and possibly within minutes). Type 4, however, is cell-mediated and has no immediate antibody–antigen component. Therefore, the responses are delayed for a variable period of time (24–72 hours). The clinical situation, however, is frequently complicated by secondary effects such as self-inflicted trauma and the clinician is only able to examine a single moment in the continuing/continuous process. Although crusting and scaling (dry dermatoses) are prominent features of many allergic or autoimmune conditions, other signs, including nodules and moist dermatosis, may be present.

The diagnosis of allergic or autoimmune disease is not simple. Indeed, apart from insect bite hypersensitivity and some forms of urticaria (hives), the immune-mediated diseases are not common and are often very difficult to diagnose. Diagnosis may rely on meticulous clinical and historical findings and the elimination of the other more common causes of crusting and scaling in particular.

A very careful examination of the horse's history, a carefully considered series of biopsies of recent lesions, and the assistance of a specialist veterinary dermatopathologist are significant aids to reaching a diagnosis.

Knowledge of immune-mediated diseases is being expanded and the similarity to corresponding human disease is evident. However, the horse may present significant or minor variations to the general pattern seen in other species. Similar names are therefore given to some diseases,

but it is important to realize that species differences may not entirely justify the name. Extrapolation from the name of the disease to possible diagnostic features and treatment may also therefore not be justified. Further knowledge and research into these diseases may well reclassify them. Table 11.1 demonstrates the major clinical features of the allergic and autoimmune diseases of the horse, dividing these broadly into those associated with hair loss or no hair loss.

Urticaria and angio-oedema ('hives')

Profile

Of all the species of domestic animals, horses probably show the greatest incidence of urticaria. However, urticaria is a symptom or sign of disease rather than a specific disease entity; it is simply one of the cutaneous manifestations of a number of diseases. In the clinical situation it is usually taken as a single entity even though its clinical manifestations vary from a minor transitory cosmetic blemish to a major, systemic and possibly even life-threatening problem. Horses between 1 and 10 years of age appear most liable and there is no gender predisposition. Thoroughbreds and Arabians are possibly predisposed to urticaria from inhaled allergens.

The aetiopathogenesis of urticaria in horses is complex; a wide variety of factors have been identified as potential causes (Tables 11.2 and 11.3); this can possibly be explained by the fact that the horse seems to be more liable to the condition than many other species and that the clinical signs of 'urticaria' are best viewed as a final manifestation common to many related and apparently unrelated conditions (Stannard 1994a).

The aetiopathogenesis is not clear and although the condition does resemble that in other species there are significant differences in the horse. However, the basic aspects are likely to be similar. Degranulation of mast cells (and to a lesser extent perhaps, basophils) is presumed to be the basic pathogenesis involving the liberation of chemical mediators which cause increased vascular permeability, inflammation and protein leakage with consequent wheal formation (Fig. 11.1).

- Immunological (type 1 and type 2 hypersensitivity responses) and non-immunological (heat, cold, pressure, exercise, some drugs) causes have been identified but in some cases no aetiology is established – usually termed

Table 11.1A General clinical signs of allergic and autoimmune disorders of the horse in which hair loss *does* occur

Diseases in which hair loss occurs	Scabs and crusts	Erosions and ulceration	Pruritus
Contact hypersensitivity	++	+	+
Drug eruptions	++	+	+++
Systemic lupus erythematosus	+++	+	–
Alopecia areata	–	–	–
Granulomatous enteritis	++	+	++?
Insect hypersensitivity	+++	+++	+++
Linear alopecia	++	–	–
Pemphigus foliaceus	+++	–	+
Pemphigus vulgaris/bullous pemphigoid bulla	+++	+/–	?
Discoid lupus	++	+	–
Sarcoidosis	+++	+	–
Onchocercal dermatitis	+++	++	++
Vasculitis	++	++	pain
Pastern leukocytoclastic vasculitis	+++	++	pain

Table 11.1B General clinical signs of allergic/immunological disorders in which hair loss *does not* (initially) occur

	Scabs and crusts	Erosions and ulceration	Pruritus
Erythema multiforme	+	+	–
Panniculitis (nodular)	–	–	–
Urticaria	+/–	–	+/–
Amyloidosis	–	–	–
Eosinophilic granulosa	–	+	–

idiopathic urticaria. The latter does not mean that there is no cause, it simply indicates that we have no real understanding of the possible cause(s).

- Immunological or hypersensitivity reactions in which injected (drugs), ingested (chemical, feeds) or inhaled (pollen, dust, chemicals, moulds) antigens/allergens are delivered to the skin systemically rather than by local contact. Penicillin is the most commonly implicated drug responsible for urticaria.
- Allergic urticaria arises from drugs, food, inhaled allergens (pollens, moulds) (Evans 1987) or more rarely by direct skin or mucous membrane contact. These should be carefully distinguished from allergic contact dermatitis.
- Physical urticaria arises without any immunological component. Dermatographism arises when a wheal develops from a blunt 'scratch' on skin (Fig. 11.2). 'Cold' or temperature-related urticaria can arise from either cold, heat or even light under some conditions. Exercise-induced urticaria may also be encountered following heavy exercise (particularly in hot or very cold conditions).

Table 11.2 The potential causes of urticarial lesions and urticaria-like skin signs

Drugs
• Foods and food additives • Inhalant or contact allergens • Transfusion reactions
Physical urticaria
• Hot/cold urticaria • Exercise induced • Pressure • Dermatographism
Infections
• Viral • Bacterial • Fungal (dermatophytosis) • Parasitic • Insect bites/stings
Stress-related urticaria
Ingestion of vasoactive chemicals
Chronic idiopathic urticaria

After Fadok V A (1990) Of horses and men: urticaria. *Veterinary Dermatology* 1:103–111.

Table 11.3 Urticaria: aetiology and characteristic types

Aetiology	Characteristic type of urticaria	Recurrence	Comments
Food	Conventional Giant	+++	Multiple generalized
Contacts	Conventional Papular Giant	+/−	Localized to contact regions Some generalized
Insect bites	Papular	+/−	Single localized
Plant contact	Papular Giant	+/−	Localized (direct exposure) (Generalized)
Drugs	Conventional Serpiginate/ Gyrate	+/−	Regional Generalized
Antiserum	Papular Giant	Exposure related	Generalized

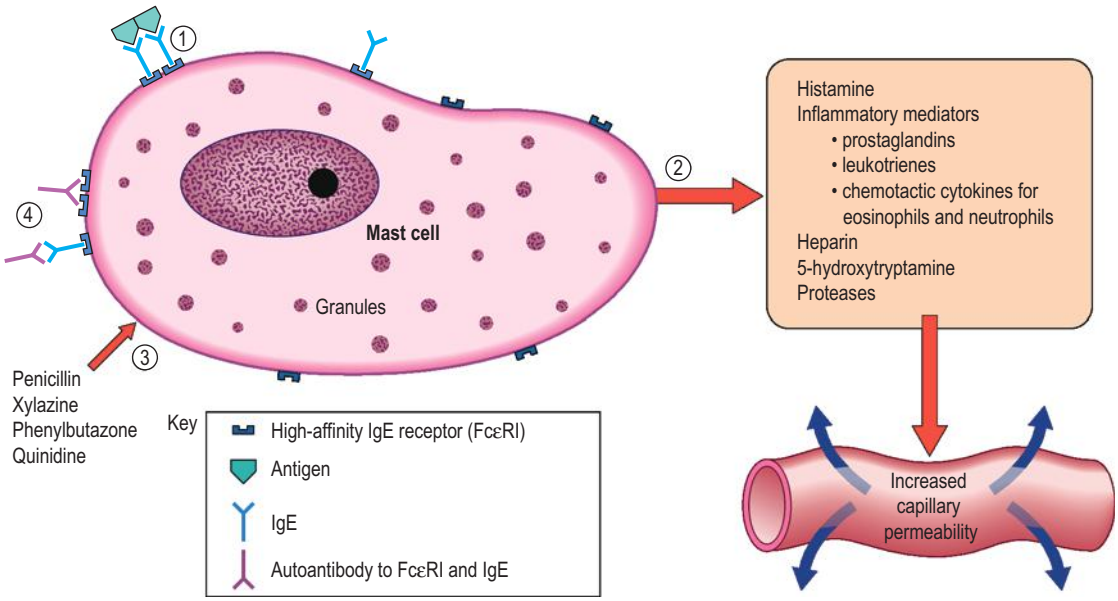


Figure 11.1 The suggested role of the mast cell in urticaria. Mast cell degranulation occurs in several different ways. (1) Type 1 hypersensitivity causes massive degranulation and consequent risks of anaphylaxis as a result of the high histamine release into the general circulation. (2) Spontaneous degranulation occurs in recurrent or chronic urticaria and (3) chemically induced degranulation such as follows drug administration (morphine or aspirin for example). (4) Autoimmune or allergic processes are possibly involved in some cases in the horse. (Adapted from Boon N et al, 2006, *Davidson's Principles and Practice of Medicine*, 20th edn. Churchill Livingstone, Edinburgh; with permission.)

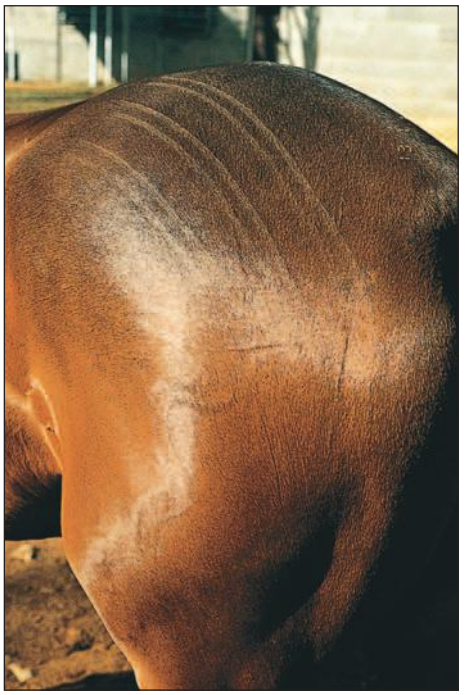


Figure 11.2 Dermatographism arising from light finger scratching over the buttocks.

Key points: Urticaria and angio-oedema

9 15 16 22 24

1. Very common complex of conditions that are poorly understood in spite of the high incidence. Immunological and non-immunological causes have been identified.
2. Clinical signs include abrupt appearance of wheals of varying sizes and sometimes linear, circular and serpiginous oedematous skin changes. Angio-oedema, in which there is massive intracellular protein loss and plasma exudation, is very rare in horses.
3. Biopsy is remarkably disappointing compared to the clinical lesions. Diagnosis can be suggested by feed/withdrawal trials in which suspected causative feeds are withheld for 2 weeks before being reintroduced to the diet. Radioallergosorbent and ELISA tests and intradermal allergy tests can be used to help identify putative allergens. In many cases a careful history will help considerably. Known exposure to drugs, possible contact allergens and some infections can be helpful.
4. Treatment is usually simple in simple cases – intravenous dexamethasone even at lower doses usually quickly resolves the early cases – even some severe cases respond rapidly. Repeated episodes often become more refractory to treatment. Removal of the causative agent is curative.
5. Recurrent episodes become more difficult to treat effectively and may require long-term management and repeated corticosteroid drugs by injection or by mouth.

Note

'Pitting-on-pressure' test: When steady and firm digital pressure is applied to an oedematous swelling or wheal for 10–20 seconds, the imprint/indentation of the finger will remain obvious at the site for several minutes (or longer in some cases) (see Fig. 11.43A, B). Inflammatory or solid/tumour-like swellings do not compress in this way, while fluid-filled swellings are compressible but do not retain the shape of the finger.

In spite of the frequency with which this condition occurs in clinical practice, little is understood about its aetiology/pathogenesis. Certain 'allergens' are 'known' circumstantially at least to be involved including some drugs such as penicillin and some sedative compounds including romifidine and morphine, for example, but other cases are induced by stress, exercise, cold exposure, and very commonly when certain diets are fed. There remains much controversy over the existence of 'food allergy' in horses. Many dermatologists consider the urticaria that occurs in racehorses and others on a high cereal diet to be an 'inappropriate food response' or a food idiosyncrasy or food intolerance, but it is hard to see what this is other than a hypersensitivity condition, given that withdrawal of the feed resolves the issue and reintroduction restores the problem! It is also true, however, that in some cases simple reduction in the volume of cereal may help to reduce the signs and so a true hypersensitivity may be less convincing in these circumstances. Food hypersensitivity seems, however, a logical possibility given the similar conditions in other species and the fact that removal of a causative food material will often result in disappearance of the condition. Plant sugars may be more involved given the high correlation between cereal feeding and recurrent episodes in racehorses in particular. It also seems that endogenous allergens can be produced possibly within the gut so that recurrent episodes may persist for some time after the primary causative agent has been withdrawn. Exogenous or endogenously produced vasoactive chemicals may be involved in some of these cases but if they were present then all horses should react in the same way to them.

Urticaria is often reported to occur under stress, cold, warmth, exercise, etc. and the diversity of suggested causes makes the condition difficult to investigate without an exhaustive history and careful clinical examination.

Contact with irritant plants such as nettles will also induce an urticarial reaction – this, however, is recognizably different by the systemic signs of pruritus and severe discomfort that they cause. Affected horses are often thought to have colic.

Insect bites cause a localized 'wheal' but this is a quite different circumstance to true urticaria (see p. 207).

Recurrent episodes at random intervals are relatively common (especially when these are due to inhaled or ingested allergens).

Individual horses may have regional urticaria, i.e. the lesions remain localized and recurrences often seem to be similarly restricted to the 'susceptible areas'. Persistent episodes may be a result of repeated exposure to the causative agent or to the failure of the body to rid itself of the causative process. However, some persistent cases are more a reflection of serious underlying disease and so whilst the large majority of cases are benign and have few serious implications, a few cases are very serious. In these the urticaria-like plaques may be the only and certainly the most obvious clinical indicator of pathology. Inflammation-related oedema due to local vasculitis produces a very similar plaque but of course carries a very different implication.

Clinical signs

The onset can be acute or peracute with signs developing within minutes up to a few hours following exposure to the instigating factor. In some cases the onset is far slower



Figure 11.3 Urticaria. Wheals showing the steep-sided, sharply demarcated nature of the typical lesions.



Figure 11.4 Urticaria. Conventional wheals. Lesions are 2–3 mm up to 3–5 mm in diameter.

and these often are more chronic in nature. Often a careful historical investigation can identify an associated substance such as a feed or a drug.

Characteristically the signs are oedematous lesions of the skin and/or mucous membranes called 'wheals' which are flat-topped, papules/nodules with steep-walled sides (Fig. 11.3).

The typical urticarial lesions develop quickly and often fade away equally quickly. Typically the individual lesion is a steep-sided papular dermal swelling – usually there are many and some are confined to specific locations. Single lesions can vary in size from 0.5 cm to 15 cm or more and these can coalesce to produce much larger areas in which individual urticarial plaques are not obvious. Usually, however, under the circumstances the skin takes on a 'bumpy' appearance. Anular, arciform and serpiginate urticarial eruptions are common in horses (see p. 258).

Typically the lesions themselves will 'pit on pressure', but the surrounding skin will usually be normal and will not pit on pressure. This test may be difficult to apply effectively to small urticarial lesions but they will compress away only to return a few minutes later – testing after clipping gives the best information. Some lesions have slightly depressed centres, but in true urticaria there is no central focus of inflammation (in contrast to insect bites; see p. 207).

In contrast to inflammation-related oedematous plaques there is seldom any pain on palpation or any palpable heat in the lesions. Pruritus may or may not be present. Some severely affected cases show no pruritus while mild cases with a few lesions sometimes show severe pruritus with extensive self-inflicted trauma suggestive of insect bite hypersensitivity.

Hair loss is not usually present unless individual lesions are exudative (angio-oedema) and long-standing.

Individual urticarial lesions vary in size and shape and, quite arbitrarily, may be divided into:

- *Conventional wheals*: 2–5 mm in diameter (Fig. 11.4).
- *Papular wheals*: multiple, small, uniform 3–6 mm diameter wheals (similar to the appearance of insect bites but without a central focus). Insect bites can induce a very similar localized wheal at the site (Fig. 11.5).
- *Giant wheals*: either single or coalesced, multiple wheals 20–30 cm in diameter or more (Fig. 11.6).

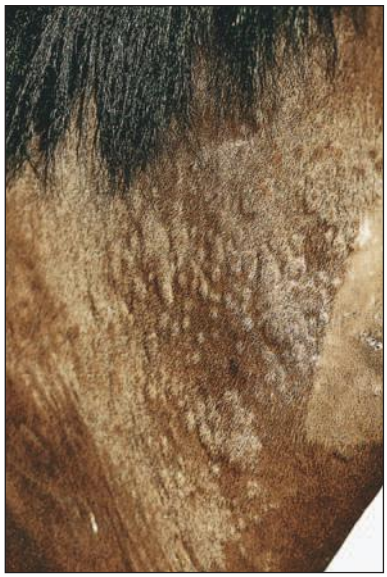


Figure 11.5 Urticaria. Papular wheals: multiple, small, uniform 3–6 mm diameter wheals. These lesions developed following intravenous sedation with an alpha-2 agonist.

- *Linear wheals*: usually multiple 'lines' of urticarial oedema most often occurring along the sides of the body (Fig. 11.7). This form tends to be more obvious in warmer weather and may be pruritic.
- *Anular wheals*: 'doughnut'-like lesions with a regular or irregular ring of oedema surrounding a depressed, non-oedematous or less oedematous centre (Fig. 11.8).
- *Gyrate/serpiginate urticaria*: the urticarial lesions take on a variety of shapes with some straighter linear, some curvilinear lesions and some serpiginous lesions (Fig. 11.9). This has been referred to as the dermal form of 'erythema multiforme' but it should be stressed that this syndrome is not related to 'true' erythema multiforme, which is a primary epidermal disease (see Fig. 11.37). Drug reactions appear to be the most common cause of this form of urticaria.
- *Exudative urticaria:* poorly defined or diffuse exudative oedema of the skin over localized areas or regions (Fig. 11.10). Possibly best regarded as angio-oedema.



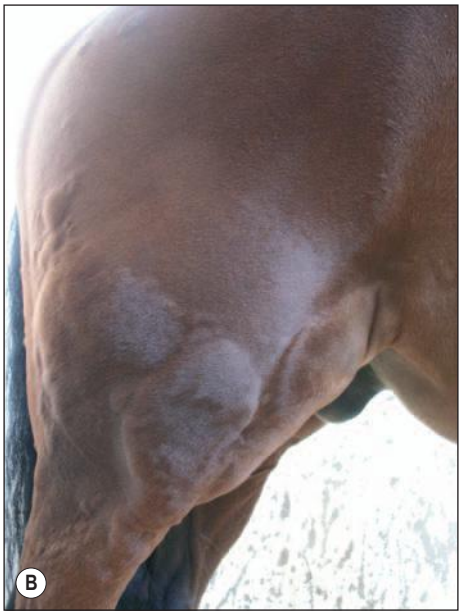


Figure 11.6 (A, B) Urticaria. Giant wheals. Single and coalesced, multiple wheals up to 20–30 cm diameter. Note the variety of sizes on this case.



Figure 11.7 Linear urticaria that occurred repeatedly when the animal was exposed to cereal foods even in small amounts and this response was markedly exacerbated by warm weather and rugging. Note the biopsy sites. Biopsies showed very little significant pathology.



Figure 11.8 Anular urticarial wheals. 'Doughnut'-like lesions with a regular or irregular ring of oedema surrounding a depressed, non- (or less) oedematous centre.



Figure 11.9 Urticaria. Gyrate and serpiginous wheals. Note the open linear nature of these lesions. In this case the lesions were persistent and required high doses of corticosteroids to resolve the problem No recurrences were encountered thereafter.



Figure 11.10 Exudative urticaria. Note the ill-defined lesions and the marked exudation indicative of protein leakage from blood vessels. This may be best termed angio-oedema.

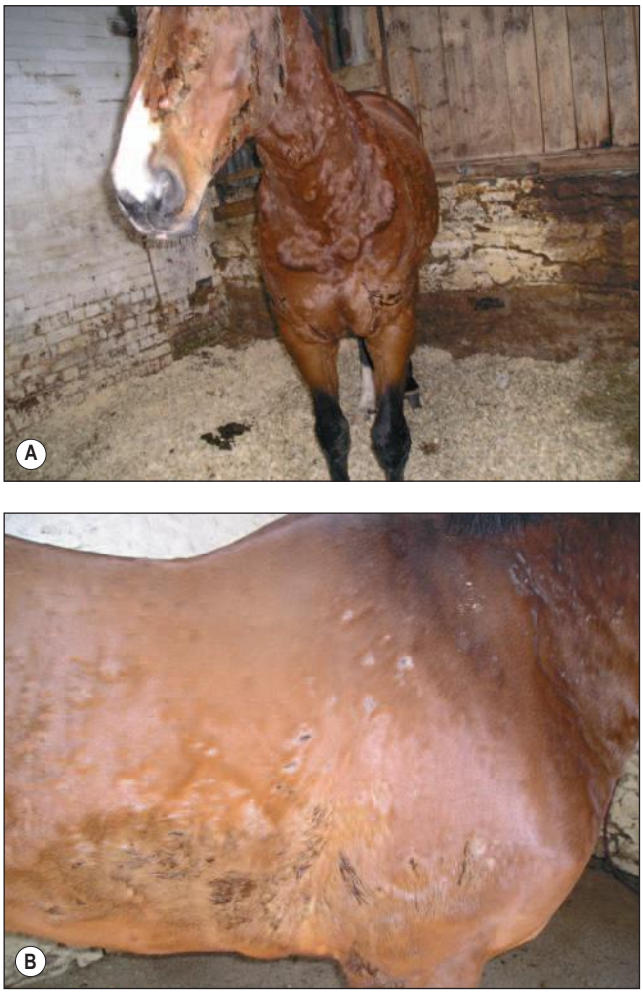


Figure 11.11 **(A)** Focal angio-oedema with severe exudation occurring at each affected lesion. **(B)** Diffuse angio-oedema with serum exudation. Note that the oedema is diffuse and ill-defined but it will still pit on pressure.

The clinical appearance of the lesions may also present in several different ways depending on the precise location of the response in the skin. The classification is a convention but there is little merit in it simply because the type of urticaria is seldom pathognomonic for any potential aetiology and the treatment will probably not vary a great deal.

Oozing urticaria Dermal oedema is severe, resulting in oozing of serum from the skin surface and possibly cracking and superficial sloughing of the hair and skin (Fig. 11.10). In the early stages the oedematous lesions will still 'pit on pressure' in spite of their apparent severity. Care should be taken to distinguish this from a bullous condition, or an erosive/ulcerative process or a pyoderma or vasculitis.

Angio-oedema (angioneurotic oedema) This is a subcutaneous form of urticaria which tends to be more diffuse owing to lack of restraint of the spread of the fluid through the subcutis (Fig. 11.11). In some cases the condition is more reflective of vasculitis (see p. 27). It usually involves the head and extremities and is more indicative of systemic and/or serious disease than 'simple' urticaria. Pruritus may or may not be present. It can easily be mistaken for vasculitis and the skin manifestations of the two conditions may in some cases be indistinguishable. Fortunately in horses angio-oedema is rare.

Individual affected horses showing any of the forms of urticaria may have associated systemic signs also. Dullness, diarrhoea, congested mucous membranes and an ocular/nasal discharge may occur concurrently in some cases and in every case the clinician should establish whether the urticaria is part of the primary condition or secondary as a result of other primary conditions.

It is a particularly common event in horses being fed high cereal rations. Many higher performance horses perform less effectively during acute episodes and often also during periods of remission, and so the condition can have serious implications. Often recurrences coincide with stress or changes in management, for example when the horse is expected to perform well at race meetings. The occasional systemic effects suggest that the condition can affect more body systems than is appreciated. The skin signs may, however, be all that is seen.

Differential diagnosis

Papular, conventional and giant urticaria:

- Insect (mosquito/bee/wasp/fly) bites (*Stomoxys* and *Culicoides* are the commonest causes): the signs may be limited to the single bite area but a few cases show a more generalized response (often with systemic/generalized organ symptoms such as respiratory obstructions).
- Nettle rash: transient signs arising immediately after skin contact with nettles or other plants.
- Dermatophytosis (*Trichophyton equinum*, *Microsporum* spp.): specific epidemiology and microbiological results. Some dermatophyte lesions can be oedematous.
- Dermatophilosis (*Dermatophilus congolensis*): inflammatory and seldom focal oedema.
- Folliculitis (sterile and bacterial/fungal): variously painful and exudative lesions often associated with marked pain and heat; often marked lymphatic and vascular engorgement).

Gyrate urticaria:

- Erythema multiforme: benign, long-standing, slowly progressive skin patterning associated with allergic response. This can be classified in some cases with gyrate or serpiginate urticaria but has a more inflammatory nature on biopsy.

Angio-oedema:

- Purpura haemorrhagica: typical history of prior respiratory tract infection. Diffuse cutaneous and subcutaneous oedema, petechiation of mucous membranes and protein leakage.
- Vasculitis (immune-mediated and photoactivated/actinic): various forms associated with inflammatory vascular endothelial damage as a result of direct endothelial damage or immune-mediated damage.

Other differentials:

- Haematoma (±haemophilia): fluid-filled, usually non-inflammatory swelling. Usually single or few and often large.
- Lymphangitis: diffuse subcutaneous swelling – sometimes with exudation, associated with diffuse inflammation.



Figure 11.12 This horse developed recurrent episodes of urticaria over varying locations. Note the presence of conventional, papular, anular and serpiginate lesions even in this limited photograph.

- Abscess/pyoderma: painful swelling with purulent exudate.
- Cellulitis: diffuse, very painful infectious disorder resulting from diffuse interstitial inflammation.

Diagnostic confirmation

A diagnosis of urticaria is probably unjustified because it is a sign of underlying reactions and it is important to establish the true cause and correct this. Nevertheless because of the inherent difficulties in identification of a cause (whether ingested/food allergy/'inappropriate response to feeding' or inhaled or contacted allergens) the name is applied in clinical equine practice. Specific challenges, seasonal variations and so on can sometimes become obvious when details are carefully recorded.

- Clinical signs and history are typical. Recurrence or repeated isolated episodes in response to any possible aetiology (see above) is very helpful diagnostically provided that the appropriate information is obtainable. A written daily record of work/activity, feed, management changes and drug administrations is very helpful; many owners are unable to recall enough detail about the previous period when an episode occurs. Most urticarial lesions erupt between 1 and 24 hours after exposure to the causative agent/circumstance.
- There are no 'typical' lesion types for specific disease states or specific allergens and many cases have multiple wheals of different types and distributions (Fig. 11.12).
- The distribution of lesions can be helpful. For example, are the lesions restricted to areas where blankets and tack are in contact or are they outside these areas? Some cases show exacerbation when the region is warmed such as under warm lights or with blankets applied to the horse. It is assumed that this reflects a cutaneous response to increased blood supply when greater amounts of the causative agent are delivered faster but this is conjecture rather than fact.
- The response to corticosteroid treatment is helpful. Commonly a single (test) dose results in a dramatic resolution within a few hours at most. However, long-standing cases often respond less rapidly and less completely so the history is very important.
- Biopsy is often unrewarding and can even be misleading. The detection of oedema in a biopsy is complicated by fluid leakage from the biopsy during collection and fixing. In the absence of any cellular responses there may be nothing to report. The section may show non-specific lymphocyte and eosinophil infiltration. However, eosinophils are not an invariable finding in all cases.
  - If there is concurrent/associated pruritus the extent of self-inflicted or secondary trauma can further complicate the problem of diagnosis by biopsy.
- A dermatographism test can be made by scratching the skin with the blunt end of a ballpoint pen and observing for a reaction within 10–15 minutes (a positive reaction is reflected by a marked oedematous wheal which follows the path of the scratch).
- 'Cold' urticaria can be induced by applying an ice cube to the skin for a few minutes, and an urticarial wheal developing in <15 minutes is a positive reaction.
- Other allergens can be 'detected' by enzyme-linked immunosorbent assay (ELISA) or radioallergosorbent test (RAST) on a blood (serum) sample. Tests are designed to detect specific IgE proteins which reflect allergic responses. The tests are not widely standardized and in many ways are too sensitive for practical use. They commonly identify groups of proteins, etc., to which the horse is variously 'allergic' (see p. 80). Often the test panel is not specific for the horse and so many of the allergens tested have little or no relevance even if they are positive. Furthermore the range of allergens tested is limited – the finding of a positive result does not of course mean that the signs are necessarily associated with it at that time – it could equally be a substance that has not been tested for!
- Intradermal allergy testing can also be used but in the case of urticaria the results are highly variable and this method suffers from the same limitations as the serum IgE/RAST tests (see above).
- Suspected food allergy can only be traced by feeding a 'hypoallergenic diet' (such as alfalfa hay only with distilled or boiled water) for 3 weeks and then challenging the horse with each specific single component of the diet for 3 weeks until a positive is identified. Confirmation of this will require a second challenge after withdrawal for 3 weeks. Positive reactions can develop quickly but can also take 2–3 weeks to appear, so it can be a very time-consuming process. Horses undergoing feed exclusion trials often lose weight because it is hard to keep the horse fit on a limited diet. Supplementary oils (especially sunflower or rapeseed oils) can be administered to boost energy requirements. Alfalfa hay is not always available and so a single species (Timothy or rye grass) early cut haylage can be used either alone or with alfalfa to make a more practical forage ration. Many commercial forms of alfalfa have varying supplementary materials such as molasses, minerals, vitamins, oils and often other fibre feeds and so their use is not advised during the trial period.
- 'Contact' causes are rare in horses but during diagnostic tests the horse should be kept in a clean stable on paper bedding and all contact with possible sources of insects or other allergens should be avoided. Urticaria arising

from known contact with either plants or other materials (tack or chemicals) is easier to investigate and treat – often owners are unaware of the potential significance of short-term exposures.

Treatment

From a therapeutic perspective the best approach is to eliminate the cause! However, this is far easier to say than do and even exhaustive investigations often fail to identify the cause – often this is termed idiopathic urticaria.

Treatment is often frustrating, with recurrences occurring at regular or irregular intervals. Usually the first episodes respond rapidly to a single intravenous dexamethasone injection. Recurrent episodes are often reported to become more severe and the plaques become more persistent with time. With repeated episodes they often also respond to corticosteroid less effectively and less rapidly over time.

1 Initial attack:
   - a If possible an investigation should be carried out to try to establish changes in feeding/management and environment.
   - b All drugs and supplements should be withdrawn.
   - c A short (usually single dose) course of systemic corticosteroids such as dexamethasone (0.01–0.04 mg/kg) administered intravenously often has a dramatic benefit. It can be repeated 2–3 times over several days if necessary. There are, however, some ill-defined risks with corticosteroid administration relating to laminitis but these are probably far overstated.
2 Recurrences:
   - a After each recurrence a full further investigation *must* be undertaken. Every effort must be made to establish the likely cause by dietary and management manipulation. The early episodes are often the clearest and least complicated; by the time a case has been in existence for some days or a horse has had many episodes the signs may be masked by self-inflicted trauma or secondary infection of exudative lesions.
   - b All drugs and supplements should be withdrawn and if the clinical evidence suggests that feeds are involved the horse should be restricted to a hypoallergenic diet.
3 Persistence: if the condition persists after 4–6 weeks, it is important to reassess the whole case again and possibly evaluate by intradermal (injection or patch) testing (Chapter 3, p. 81) for atopic disease (IgE) or specific ELISA or RAST tests for IgE. It may be possible in this way to identify possible/probable causes from the panel of positive results and attempt to avoid them. However, the results from these tests are very unreliable and repeated samples from the same horse can give very different results over very short intervals.

Hyposensitization and neutralization may be attempted but this is not generally available and carries risks. It may hold some promise for inhaled allergic urticaria and atopy (see p. 262). The specific dose of the allergen used and the required frequency of challenge are variable according to the individual response but usually the injections have to be maintained throughout life. Recent reports have questioned the relevance and therapeutic benefits of these methods but opinions are divided. Many practitioners feel that they are an effective approach whilst others have less faith in them.

Long-term management

Corticosteroid Where single doses of dexamethasone fail to resolve the issue for long enough or where the lesions persist, it is possibly unwise to continue with increasing doses of dexamethasone. Daily oral doses of prednisolone (0.2–0.4 mg/kg q 24 h) or dexamethasone (0.01–0.06 mg/kg q 24–48 h) administered in feed in the morning should be manipulated so as to obtain an acceptable effect.

Note

Prednisone has no material value in horses and should not be used.

The dose of corticosteroid should then be reduced to reach a minimal effective (therapeutic) single daily dose (MESDD). Double this daily dose should then be administered using twice the interval (48 hours for prednisolone and 48–96 hours for dexamethasone) in the mornings only (see p. 96). Provided that this dose is effective it is then gradually reduced to establish a minimum effective alternate-day dose (MEADD). The objective is to use the least possible drug consistent with a complete absence of clinical signs. Concurrently every effort should be made to remove any possible causative agent.

Antihistamines Antihistamines including oral hydroxyzine hydrochloride, chlorphenhydramine and promethazine are largely disappointing in the horse. If effective, a remission in 3–4 days will be achieved and the drug can then be reduced to a minimum effective daily dose. Side-effects include sedation and/or hyperactivity. The antihistamines may be more effective in preventing episodes than treating them – they will seldom stop episodes from developing, however, but may reduce the amount of corticosteroid required to bring an episode under control. The drugs should probably not be used in pregnant mares as there is a suggestion of teratogenicity.

Prognosis/prevention

Provided that the cause can be eliminated the outlook is excellent because most animals are little affected by it. However, from an owner's and veterinarian's perspective the condition can be extremely frustrating. Some cases are clinically affected and in any case the animals are unsightly. In some cases no explanation can be found for a single or few episodes and then many such cases are not materially affected. Some may show one or two episodes and then remain free of the condition for life. There is little that is predictable about the condition.

It is the recurrent and deteriorating cases that create the long-term problem. Some badly affected horses are never free of the condition or the threat of it. Animals that have concurrent signs such as diarrhoea or respiratory tract obstructions are clearly much more difficult to manage.

Cereal foods are commonly implicated and it is difficult to maintain a high performance horse on a hypoallergenic diet free of all cereal products.

Atopy

Profile

Atopy is a rare, multifactorial, (probably) genetically determined predisposition to allergic responses to environmental antigens that results in formation of excessive IgE, which leads to a generalized and prolonged (Coombs type 1, immediate and late phase) hypersensitivity to common environmental antigens including pollens, grasses, weeds and trees, moulds, dust, feathers, cotton, wool and other fibres. These antigens usually cause no difficulties in non-atopic animals. A type 4 hypersensitivity is also involved in some cases. The skin is the major target organ for the changes.

Little is known about the condition in horses but it is widely recognized as a definitive clinical entity (Scott & Miller 2003a). It is not known whether it is heritable but Arabians and Thoroughbreds in early adulthood appear to be predisposed to the condition (Degler 1997).

The condition is classically attributed to IgE responses to the allergens (often inhaled or contacted but sometimes ingested) but it is likely that the condition is a complicated immune disorder involving IgG, eosinophils, Langerhans cells and other components of the immune system. It is usually characterized by pruritus associated with exposure to specific allergens that provoke allergen-specific IgE production. Repeated exposures result in increasing amounts of allergen-specific IgE that may bind to Langerhans and possibly less significantly cutaneous mast cells, and so lead to increasing disease. Once the exposure is re-established, the clinical signs (moderate to severe pruritus) are triggered locally.

There is debate whether the classical disease exists in the horse. Although it is considered to be a rare disease, it may in fact be under-diagnosed.

Clinical signs

The clinical signs are dependent upon exposure to the allergen and so may be seasonal or non-seasonal; seasonal cases may become non-seasonal over the years, especially if exposure to the allergen is not restricted or measures are not taken to treat the disease (see below).

The primary sign is pruritus developing at a young age (Table 11.4). Some cases have urticaria and some are less pruritic than others. There is recurrent pruritus unrelated to season without obvious or histological primary lesions other than self-mutilation. The affected horse may bite aggressively at a localized site until it is significantly damaged and then turn its attention to another site. Horses often bite unrelated regions in subsequent attacks (Fig. 11.13). Some have more general signs but most seem to have body pruritus as opposed to limb or head pruritus but no site is exempt. Rubbing of the skin (even at sites that are not apparently affected at the time of examination) often induces a 'pleasure response' at the lips and muzzle.

There is some suggestion that the common eosinophilic (collagenolytic) granuloma is a form of this condition but this is far from convincing; other explanations are possibly more plausible. Contrary to some other species, respiratory tract allergic disease is unusual in atopic horses.

Secondary lesions are invariably related to self-inflicted damage and include excoriation, alopecia, lichenification and hyperpigmentation (melanoderma) or scarring (Fig. 11.14).

Differential diagnosis

- Ectoparasites (pathogens and incidentals): identifiable parasites in skin brushings; usually site related such as legs for *Chorioptes equi* and forage mites and *Dermanyssus gallinae* on the head and legs.
- Food hypersensitivity: often with urticarial wheals and always exposure related; possibly concurrent diarrhoea.
- Insect bite hypersensitivity: seasonally related and worse when outside rather than inside/stabled; avoidance of contact resolves the problem.
- Contact dermatitis: exposure related.
- Drug eruptions: history of drugs, less pruritus and more generalized seborrhoea.

Table 11.4 Criteria for diagnosis of atopic dermatitis in horses

(a)	Pruritus without other defined cause at unpredictable and variable sites
(b)	Dry seborrhoeic skin
(c)	Onset within 2 years of birth with progressive deterioration

Key points: Atopy

2 4 5 15 16 18 21 22 24

1. Rare, genetically predisposed inflammatory and allergic skin disease that manifests, initially at least, in younger horses
2. The predominate sign is pruritus. Seborrhoea and occasionally concurrent respiratory tract and other evidence of allergic disease including urticaria may be present.
3. Diagnosis is based on exclusion of other causes of pruritus, serum ELISA and intradermal or patch skin testing.
4. Treatment is limited in horses to corticosteroids and avoidance strategies of proven or putative trigger factors. Hyposensitization procedures involving injection of 'specific' antigen concentrations have been reported to be effective in a few cases.
5. The prognosis is guarded – if the allergens can be avoided then the signs will be minimal but will seldom clear completely.



Figure 11.13 Atopy. A young horse with recurrent self-mutilation for which no other cause could be established.

Note

Some atopic horses have concurrent hypersensitivities to insects, drugs, contacts or food, and other 'allergic' conditions such as recurrent airway obstruction, allergic rhinitis.

This can confuse the diagnosis.

Diagnostic confirmation

- Absolute elimination of all other causes of pruritus.
- Early elimination of insect and food hypersensitivity by exclusion.
- Intradermal testing (limited use, related to availability of antigens) using locally important antigen possibilities (moulds, pollen and dusts). This will identify the specific reactivity of the animal to the allergens.
- Serum testing (RAST/ELISA) gives particularly poor sensitivity and specificity, with false positives and false negatives and poor repeatability.
- Skin biopsy shows only non-specific superficial to deep perivascular dermatitis with mild eosinophilia.

Note

Atopy should be one of the last resorts for diagnosis (see pruritus syndrome, p. 442).

Treatment

If the allergen can be determined, it may be possible to avoid contact.

Hyposensitization is possible in some cases using injections of increasing strength but these are not without hazard and need to be maintained for years. Total resolutions are rare – usually the accepted good result is a 50% reduction in clinical signs with a 50% reduction in drug use to control the condition provided that at the same time the allergen is avoided.

Systemic glucocorticoids are useful and form the basis of treatments. Prednisolone is probably the best option but doses may need to be maintained at a reasonably high level (see Chapter 4, p. 96).

In other species ciclosporin A has been found to be helpful in some cases but the drug is not available in an equine formulation and is inordinately expensive even then. No doses are established or suggested.



Figure 11.14 This 3-year-old pony had a 2-year history of generalized, variable intensity pruritus. The flank regions were the most consistently affected zone and presented with lichenified, damaged and hyperpigmented skin.

Food hypersensitivity/allergy

Profile

There is much debate as to whether this condition exists in horses at all (Littlewood 1999). Food hypersensitivity in horses is often suspected and circumstantial evidence often supports the diagnosis. Also, many texts and scientific articles have referred to it for many years (Huytra & Marek 1922, Volland-Francquille & Sabbah 2004). Food hypersensitivity, food intolerance, adverse reactions to food components or allergic skin reactions to food components are commonly used synonymously with 'food allergy' (Fig. 11.15). Food allergy itself is a form of adverse reaction to some food component where an abnormal immunological reaction occurs (Halliwell 1992). In some cases the term food allergy is used even when there is no immunological basis confirmed and this is better termed food intolerance.

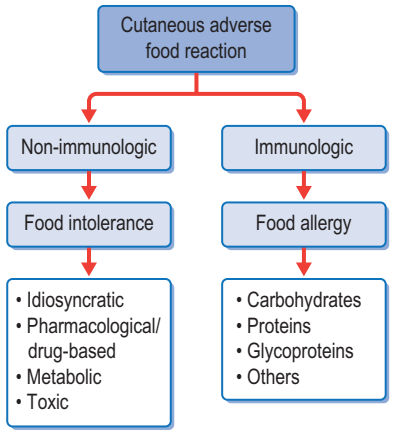


Figure 11.15 The classification of food intolerance/cutaneous adverse food reaction. (After Wagner, R, Knottenbelt D C, Hunsinger B, 2007, *Food Allergy in Horses*. Proceedings of ENUCO, Vienna, July 2007.)

Key points: Food hypersensitivity/allergy

1 Rare and controversial clinical condition related to exposure to a defined food type/component.
2 Clinical signs vary from mild to severe urticarial rashes and diffuse or localized seborrhoea. Concurrent diarrhoea and weight loss may be encountered. Often the horse is not apparently concerned by the condition.
3 Diagnosis is dependent on response to withdrawal of the food responsible for the condition. Confirmation of the diagnosis can be made by test dosing with the suspected allergen. Assay of blood for specific IgE and IgG molecules and skin testing can be performed but currently have a poor sensitivity and specificity.
4 Treatment is inevitably based on avoidance strategies where the allergen is known. Long-term corticosteroid therapy is possible but the side-effects may be worse than the condition. Owners often have to live with the condition.
5 The prognosis remains guarded but is rather better if a definitive allergen is identified and can be avoided easily. Some cases have multiple allergens and then control becomes increasingly problematic. A few cases resolve spontaneously after a while.

Idiosyncratic responses are recognizably abnormal and closely resemble an immunological reaction grossly similar to an allergy. However, this is supposedly due to a non-specific mast cell degranulation that arises because of specific food-related histamine-releasing substances or by ingestion of histaminic foods or by the formation of vasoactive metabolites within the large bowel.

It is suggested that the immunological forms of the condition are a complex type 1, 2 and 3 hypersensitivity reaction in response to individual and groups of foodstuffs. It is certainly true that there are horses that develop urticaria in response to individual food components including proteins, carbohydrates and also preservatives and antioxidants. Plant sugars may be a commoner cause of this response than is appreciated. In some reports protein components are suggested as being more liable to induce the response, but grains and cereals (plant sugars) are commonly involved whether through sugars or through protein or oil components. A condition known amongst horse owners and trainers as 'protein bumps' has been assumed to be an allergic response but in spite of its regular occurrence little or nothing is known about this.

There is no apparent sex, breed or age predilection but individuals seem to be very liable and remain so for much of their lives. The characteristic reactions include non-seasonal, food-related, urticaria with or without pruritus and can lead to discomfort and in a few cases secondary infections.

Clinical signs

Recurrent generalized pruritus, perineal pruritus and/or urticaria related to ingestion of the allergen (feed type) are the common presentations.

Urticarial responses to specific feed components are probably the commonest manifestations of food allergies (see p. 263 and Figs 11.4 to 11.8). Barley and oats are particularly reported as being a cause but this may reflect the higher number of horses receiving these. Some cases are overtly painful when touched and actively resent skin handling; in these cases signs may be limited to resentment, bucking and rearing when tacked up.

Note

The concept of food allergy being a cause of urticaria in horses is controversial and as yet the relationship between the two is poorly understood (see p. 263).

Secondary lesions such as papules and plaques with crusting and exudative excoriation are invariably related to self-inflicted damage.

Skin thickening and extensive flaking can arise with long-standing cases. Repeated episodes can result in skin scarring and altered pigment of both skin and hair.

Differential diagnosis

- Ectoparasites (pathogens and incidentals) – lice and mites in particular: pruritus is usually mild and the parasites can be easily identified in brushings.
- Cutaneous onchocerciasis (*Onchocerca cervicalis*): limited locations involved and a rare condition in temperate climates and where avermectin anthelmintics are used routinely.
- Oxyuriasis (*Oxyuris equi*): perineal pruritus only and history of no avermectin wormer. Eggs can be identified using adhesive tape tests on the perianal skin.
- Atopy: rare pruritic disorder first presented in horses less than 2 years of age for which no other explanation can be found.
- Insect hypersensitivity: strong seasonal relationship and worse pruritus when outside rather than inside. Increasingly severe localized/regional pruritus each succeeding year.
- Intermammary accumulation-related perineal pruritus syndrome: easily checked and cured by removal of the accumulation.

Diagnostic confirmation

- Early clinical elimination of insect/parasite infestation.
- Food exclusion trials.
- Intradermal testing (limited use, related to availability of antigens) using locally important antigen possibilities (moulds, pollen and dusts). This will identify the specific reactivity of the animal to the allergens (see p. 79). The method has poor sensitivity and specificity.
- Serum testing (RAST) gives particularly poor sensitivity and specificity with false positives and false negatives and poor repeatability.
- Skin biopsy shows only non-specific superficial to deep perivascular dermatitis sometimes with eosinophilia.

Treatment

Feed exclusion trials should start with the feeding of good quality hay or haylage (preferably of a single species grass) to which the horse has had no previous exposure. This is fed alone for 3 weeks. If an improvement is produced by this alone, a tentative diagnosis can be made. Reintroduction of the original diet can then be made and a diagnosis confirmed if the condition returns. Not many owners are willing to undergo this, however, preferring to maintain a diet on which there is no problem. If the allergen can be determined, it may be possible to avoid it completely.

Systemic corticosteroids can be administered to control the condition but response is typically poor and affected horses gradually become less responsive, requiring either higher doses, greater frequency of administration or altered steroid.

Elimination of contact with the specific allergen must be the prime objective. Hyposensitization is possible in some cases using injections of increasing strength but these are not without hazard and need to be maintained for years. The accepted 'successful desensitization' is a 50% reduction in signs with a 50% reduction in drug use provided that the allergen is avoided also.

A few cases resolve spontaneously after a while.

The pemphigus complex

Pemphigus is a rare blistering epidermal and mucosal disorder arising from an autoimmune-mediated disruption of the intercellular attachments, characterized by transient vesicles and bullae with histologically detectable characteristic acantholysis.

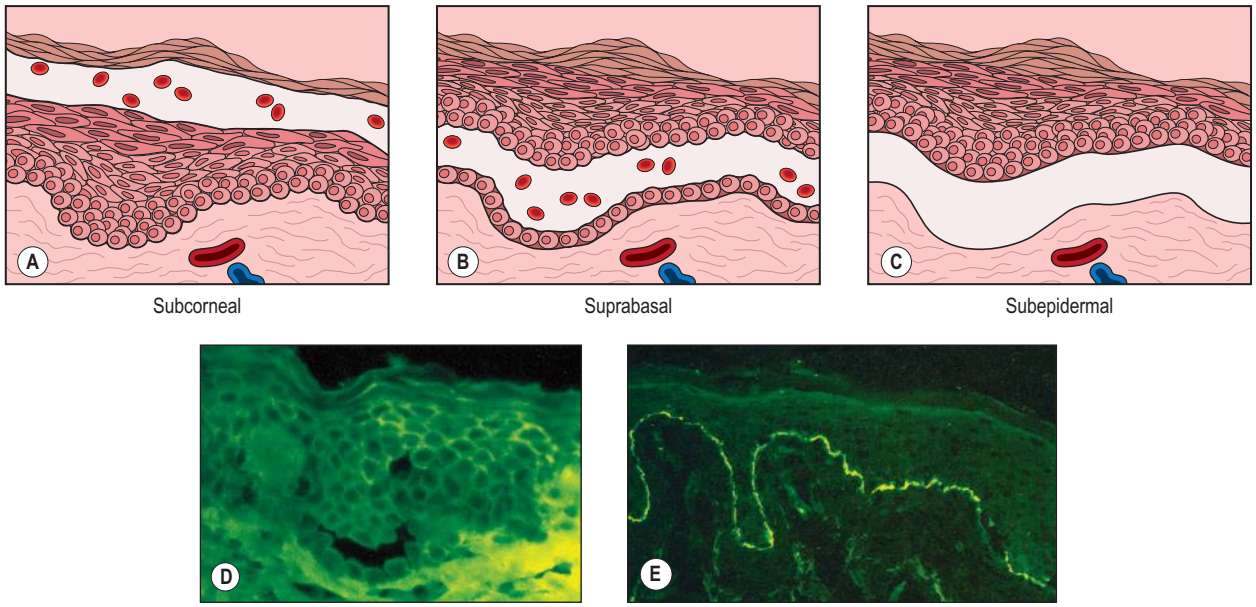


Figure 11.16 These drawings depict the sites where intracutaneous bullae develop. **(A)** Subcorneal blister within the stratum corneum in pemphigus foliaceus (see **(D)**). **(B)** Suprabasilar bulla formation occurs just above the basement membrane of the epidermis – the stratum corneum forms the roof of the blister. This is typical of pemphigus vulgaris (see **(E)**). **(C)** Subepidermal bulla formation involves the entire separation of the epidermis from the dermis and is typical of the most severe forms of bullous pemphigoid. **(D)** Immunostaining for equine IgG shows the distribution of the antibody to be within the epidermis and so bulla formation will occur there as the bonds between the cells are disrupted. **(E)** In pemphigus vulgaris the deposition of immune globulins occurs at the basement membrane and this picture shows its clearly limited distribution; it is understandable that the separation would be more extensive and more serious. (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn. WB Saunders, Philadelphia; with permission.)

The pemphigus complex includes several different, related, immune-mediated vesico-bullous/pustular disorders that differ in the precise location of the disruption within the skin. The various described forms have been recognized in horses. Although the underlying aetiopathogenesis in horses is uncertain, it may be triggered by drugs, insect bites, viruses and other insults. It is generally assumed that the various conditions that fall into this group are similar to those occurring in other species including the dog and cat and man. The exact location of the resulting immune-mediated cellular disruption within the skin explains the variations in the several types of pemphigus recognized in horses; this reflects the precise components of the desmosomes that are targeted by the autoimmune process (Fig. 11.16). The differentiation of these autoimmune conditions is heavily dependent on the recognition of the underlying pathology (Fig. 11.17).

The characteristic pathology in all the disorders results from autoantibodies to the desmosomal (intercellular cementing) proteins in the skin, which are responsible for squamous cell adhesion. The antigen–antibody complex triggers the release of proteases and the cells are 'freed' from their attachments and so bullae develop. Numerous free nucleated (acantholytic) keratinocytes are easily recognized histologically in the bullae and the resulting crusts that develop when the bullae rupture so skin biopsy is usually strongly suggestive of pemphigus complex. Ideally an intact bulla should be biopsied but the chances of this are low and so several (up to 10) biopsies are usually required to confirm the diagnosis. Immunohistochemistry can be useful but the tests are expensive and lack specificity. Some specialist laboratories offer the diagnostic service routinely but others do not.

Most of the equine cases reported in the literature are pemphigus foliaceus. In this the immune responses occur in a more superficial epidermal location and so scaling and limited exudation are features. In other species such as the dog, a milder (or possibly earlier) stage of this condition is sometimes termed pemphigus erythematosus and the lesions are very similar to those of systemic lupus erythematosus. This form is not reported in horses.

The most severe form of the condition is pemphigus vulgaris – although the term 'vulgaris' implies commonality this is in fact an extremely rare disease. In this case the immune disruption occurs adjacent to the basement membrane and so there is a full epidermal disruption with vesicles and blisters developing mostly at mucocutaneous junctions (eyes, lips, nose, tongue, ear, prepuce and anus).

Bullous pemphigoid is probably the rarest form of the condition and here the immune reaction occurs under the basement membrane.

A group of so far unexplained coronary band disorders are recognized in horses. At the University of Liverpool, cases of coronary band dermatosis that affect one or more usually all the coronary bands to a variable extent are seen relatively frequently. The cases are divisible into three main categories and at least one of these is probably related to pemphigus foliaceus; the coronary band hyperplasia and exfoliation is part of a wider skin disease that can be diagnosed histologically as pemphigus foliaceus. The other two disorders are localized to either the coronary band alone (although it may include the sole also) or the coronary bands and ergots and chestnuts. There is, however, little research that has been carried out to establish the true nature of the immune-mediated coronary band changes that occur with some frequency in clinical practice. However, preliminary studies at Liverpool University suggest that some have an autoimmune or a immune-mediated pathogenesis. In other species a condition

recognized as pemphigus vegetans also occurs very rarely. In this case there is a very mild change rather similar to pemphigus vulgaris. The clinical effects are much less obvious and at least one of the forms of immune mediated coronitis could possibly be attributed to this condition.

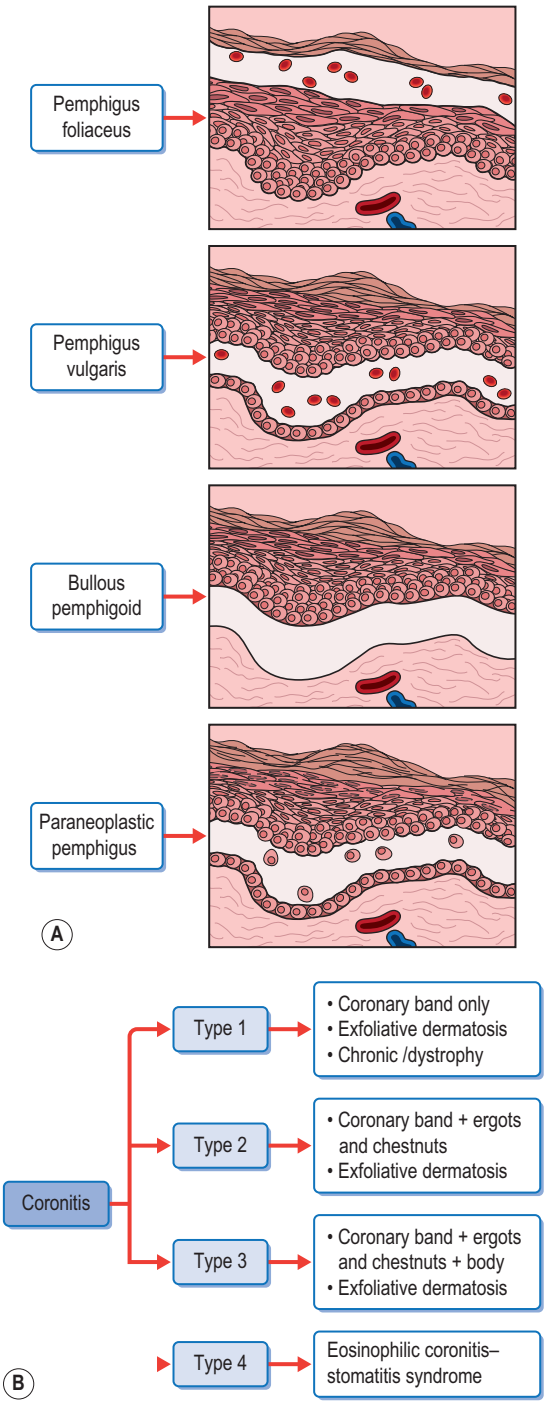


Figure 11.17 (A) Disorders that fall into the pemphigus complex in horses and the location of their respective pathologies (where known). (Adapted from Kumar V, Abbas A, Fuasto N, 1999, *Robbins and Cotran Pathologic Basis of Disease*, 7th edn. WB Saunders, Philadelphia; and from McGavin M D, Zachary J F, 2006, *The Pathologic Basis of Veterinary Disease*. Mosby, St Louis, Fig. 17.12, p. 1211; with permission.) **(B)** Coronitis includes a broad group of diagnoses with four different types being recognized. Most fall into the pemphigus group of diseases.

There are of course other coronary band disorders that are easily mistaken for this group of conditions (see p. 478).

The pemphigus group of disorders has an ill-defined aetiopathogenesis. Whilst the condition is certainly autoimmune in origin, the onset may be triggered by other factors. Stressors, skin disease, allergic conditions and neoplastic disease may be involved and so it is always worth carrying out an extensive clinical examination at the outset. For example, the paraneoplastic pemphigus condition can be typical in dermatological presentation but the underlying neoplastic disease may be far more important. However, it is also true that the pemphigus can in fact be the most critical component of the disease complex.

As a general rule the pemphigus conditions will require immunosuppressive treatment. Conventionally this involves immunosuppressive doses of corticosteroids including oral prednisolone and oral or parenteral dexamethasone. Prednisone administered orally to horses has no material value.

Pemphigus foliaceus

Profile

This is an autoimmune disease characterized by an exfoliative dermatitis due to a type 2 hypersensitivity arising from autoantibodies directed against the cell membrane of the epidermal cells.

The condition occurs in horses of all ages including foals less than 6 months of age – young horses may carry a somewhat better prognosis (Zabel et al 2005). Appaloosas are possibly predisposed but most breeds can be affected. Although a genetic aspect is likely the heritability of this is totally unknown. Trigger factors that may be involved in the aetiopathogenesis include drug administration (no defined drug is reported to be more liable to induce the condition), internal neoplasia (no particular type of neoplastic disease

Key points: Pemphigus foliaceus

10 12 16 19 20 23 24

1. A rare disease but probably the commonest autoimmune skin disease of the horse. Associated with autoantibodies to cell membranes of the epidermis. Bullae are created within the epidermis.
2. Localized or more often generalized exfoliative dermatopathy – often with skin thickening and some superficial erosions. Alopecia reflects the areas where the condition is present. Systemic signs such as leg filling and lethargy can be seen.
3. Diagnosis is based on clinical presentation and characteristic histopathology although up to 10–12 biopsies may be required before the definitive histological features can be found. Biopsy technique is critical.
4. Treatment centres on immunosuppression usually with prednisolone or dexamethasone at high doses. Other immunosuppressives such as sodium aurothiamolate, pentoxifylline and azathioprine can be used but are seldom curative on their own.
5. The prognosis is poor although some cases can be managed long term. Younger horses seem to have a better prognosis and some may even resolve completely.

seems more likely than others to trigger pemphigus), ill-defined stressors, and food and inhaled allergens.

Clinical signs

Early or mild cases These cases show transient vesicles and pustules with superficial erosions forming epidermal collarettes (Fig. 11.18). In some cases the first signs may be a persistent urticaria-like eruption over variable areas of the body. Variable erythematous scaling and crusting commonly begins around the face and/or limbs; some alopecia or thinning of hair may be evident. Inflammation is particularly severe on the skin of the limbs (Fig. 11.19). The skin may 'feel' hot and thickened. Acute inflammation of the coronary band, chestnut and ergot is a common but not consistent finding resulting in variable lameness. Gradual extension to the whole body over some days or weeks is common, with marked alopecia but only mild pruritus. The acute skin lesions may be painful or pruritic (as manifest by persistent rubbing) and secondary excoriations may then be misleading.



Figure 11.18 Pemphigus foliaceus showing circular erythematous and exudative lesions which developed into epidermal collarettes.



Figure 11.19 Pemphigus foliaceus. Severe and extensive lesions on the skin of the limbs.

Advanced/chronic cases Severe, diffuse crusting and scaling with extensive alopecia develop, often over large areas of the body. The exfoliating epidermis can peel off in leaf-like sheets (hence the name 'foliaceus') (Fig. 11.20).

Neonatal/juvenile pemphigus The condition is clinically indistinguishable from the adult form except that it occurs in very young foals – often within weeks of birth **(Fig. CD11 • 1A–C)**. The outlook for affected foals is reported to be better than for older horses.

CD

Paraneoplastic pemphigus This is an aggressive form that is reportedly associated with solid neoplastic disorders. The pathology in these cases is not well reported and in a few cases seen by the author there have been features of pemphigus foliaceus and pemphigus vulgaris with a mix of epidermal bullous locations (Fig. 11.21 and **Fig. CD11 • 2A–D**). Some of these cases are also severely pruritic.

CD



Figure 11.20 Extensive exfoliating epidermis in a confirmed case of pemphigus foliaceus. Note the absence of any apparent underlying ulceration or inflammation. However, the skin felt warm and somewhat thickened in the worst affected areas such as on the face and the lateral neck.



Figure 11.21 This 12-year-old gelding developed severe pemphigus foliaceus over a 5-week period. At autopsy he was found to have a renal carcinoma.

Systemic signs including lethargy, anorexia, ventral/limb oedema, fever, depression and chronic weight loss can be present in all cases but are usually much more evident in the advanced cases.

Differential diagnosis

- Dermatophilosis (*Dermatophilus congolensis*): characteristic distribution of lesions on back and lower limb regions in wet conditions; organism is easily recognized in direct smears taken from crust and pus. Usually resolves spontaneously when appropriate treatment is instituted.
- Dermatophytosis (*Trichophyton* spp., *Microsporum* spp.): characteristic epidemiology and defined microbiology. Spontaneous resolution after 5–9 weeks.
- Staphylococcal folliculitis: painful pustular and focal areas involved. Bacteria can be identified but can be a complication of the pemphigus complex.
- Onchocerciasis (*Onchocerca cervicalis*): epidemiology and distribution of lesions with microfilariasis in some cases; history of poor anthelmintic treatments.
- *Culicoides* spp. dermatitis (non-allergic and allergic forms (sweet itch)): moderate to severe seasonal and management-related pruritus in defined areas.
- (Generalized) granulomatous enteritis syndrome and sarcoidosis: exfoliative dermatitis with no bulla formation in the skin; multiple organ involvement.
- Systemic lupus erythematosus-like syndrome: pigmentary loss and multiple organ involvement and seldom focal.
- Bullous pemphigoid/pemphigus vulgaris: deeper bulla formation results in a more aggressive-looking lesion with bleeding and exudate. Often involves mucocutaneous junctions and the oral and nasal mucosae.
- Exfoliative eosinophilic dermatitis: mildly pruritic without bulla formation.
- Papular dermatitis: focal areas only with long-standing static lesions that may resolve spontaneously after week to months.
- Epitheliogenesis imperfecta: only in newborn foals.
- Epidermolysis bullosa: evident in very young foals.
- Coronary band dystrophy/coronitis syndrome: some forms can be difficult and coronary band involvement in pemphigus foliaceus is relatively common.
- Drug eruptions: history of drug administration and absence of bulla formation in skin biopsies.
- Chemical dermatoses: history of applications.
- Seborrhoea: uncomplicated primary seborrhoea is very rare and is asymptomatic apart from the seborrhoeic state.

Diagnostic confirmation

- History, clinical appearance and chronic course are characteristic.
- Haematology and biochemistry are usually normal or at least non-specific.
- Direct impression smears from fresh lesions show only acanthocytes and neutrophils.
- Biopsy is characteristic (Evan 1990).

Note

To protect intact vesicles or surface scabs the biopsy sites should be carefully chosen and should NOT be wiped, clipped scrubbed or shaved prior to biopsy because these may remove the only evidence there is for the condition – acanthocytes in the superficial crusts and scale. The harvesting of an intact vesicle or pustule is the best outcome.

Multiple sites should be sampled (at least 6–8 biopsies should be taken), with the surface scabs being included as far as possible.

- Direct immunofluorescence testing shows intracellular deposits of immunoglobulin in epidermis. Lesion selection for histopathology is very important. Intact vesicles or pustules are preferred. Perilesional skin should be sampled if vesicles or pustules are not obviously present (Scott 1988). Michel's fixative (buffered ammonium sulphate solution) provides good preservation of immunoglobulins and complement (Rosser et al 1983). In some cases acanthotic epidermal cells can be identified within the fluid aspirated from intact blisters.

Note

Many ordinary laboratories are unable to perform these tests so the best quality labs should be identified and the samples submitted directly with the request for immunocytochemistry and immunofluorescence.

It is useful to contact the lab first to get advice.

Treatment

Treatment has to be directed at immunosuppression and is likely to be long term at best or most likely permanent. Prednisolone is the most useful readily available immunosuppressive drug. Although there are some potential problems related to its use these are probably overstated (see p. 97). High (immunosuppressive) doses of prednisolone are used at 2.2–4.4 mg/kg q 24 h (administered in the mornings) for one week to start with and then maintained at around 1 mg/kg q 24 h for a further 2 weeks before reducing the doses to achieve a minimum effective single daily dose. Attempts should be made to establish alternate-day therapy (see principles of therapeutics, Chapter 4, p. 98). Dose reductions to anti-inflammatory rates are usually unrewarding with rapid recurrence of symptoms. It is likely that treatment will be required for the rest of the animal's life.

Dexamethasone can be used also (both orally and by intravenous or intramuscular injection). Doses of 0.2 mg/kg q 24 h are usually roughly equivalent to the high doses of prednisolone (see p. 96). This drug is less sparing of the pituitary adrenal axis and also carries an ill-defined increase in risk.

With both prednisolone and dexamethasone there may be significant generalized immunosuppression and so secondary skin and other infections can develop.

If the horse fails to respond to corticosteroids, consideration should be given to using gold injections such as sodium aurothiomylate (Myocrisine, JHC) (Manning 1983) at 1 mg/kg once weekly for 4–6 weeks by deep intramuscular injection. Once remission is achieved the interval can sometimes be extended to 4–5 weeks. The clinical benefit may only become obvious after 4–6 weeks. Whilst this material is unlikely to resolve the condition it does limit the severity of

the disease and reduces the dose of corticosteroids required to control it. There are anecdotal reports of full resolution also. Side-effects are potentially severe (bone marrow suppression, anaemia, and renal failure) though there are no evidence-based reports of their occurrence in horses.

Azathioprine or pentoxifylline have also been suggested as potential treatments.

The prognosis is poor, with full recovery only occurring very rarely; younger horses may respond better and carry a marginally better prognosis (Schlipf 1997).

Pemphigus vulgaris (EPV) and bullous pemphigoid (BP)

Profile

These are very rare vesico-bullous, ulcerative diseases which may affect the oral cavity, mucocutaneous junctions and skin (or combinations of these). In these the autoantibodies are directed against the basement membrane so the pathological changes are much deeper than in pemphigus foliaceus. Subepidermal clefting and bulla formation results in a more erosive/ulcerative pathology. The two clinical diseases (pemphigus vulgaris and bullous pemphigoid) have many similarities (George & White 1984) and are often described together.

Although the condition has an autoimmune basis, trigger factors such as stress, infection, skin disease and neoplastic disease may be involved in the development of the typical condition (Williams et al 1995).

Clinical signs

Initial signs are transient, fragile vesicles and bullae in skin and mucocutaneous junctions. These rapidly burst and leave crusts and ulcers with epidermal collarettes. They are very rarely identified.

The most obvious lesions occur around the oral and vulval mucous membrane–skin junctions (Fig. 11.22). Lesions may also occur at junctions between skin and keratinized areas such as the coronet and around the ergots and chestnuts. Whilst the early lesions are most severe at mucocutaneous junctions, the cutaneous changes can extend into the adjacent skin and so the face **(Fig. CD11 • 3)** and perineum can be severely affected. Diffuse lesions may be present in the groin and axillae.

CD

The lesions may be very extensive with serum exudation and gross oedema (particularly of the limbs) but are seldom either painful or pruritic.

Systemic signs are present in severely affected horses including anorexia, depression and intermittent or persistent pyrexia.

Differential diagnosis

- Viral skin diseases such as equine poxvirus and coital exanthema (EHV_3): transient vesicular states with rapid resolution.
- Systemic lupus erythematosus-like syndrome of horses: multi-organ system involvement and pigmentary loss with seborrhoea. Little ulcerative tendency.
- Drug eruptions: unpredictable signs but seldom erosive/ulcerative oral lesions. Local drug-induced oral ulcerations also have known history of exposure.
- (Generalized) granulomatous enteritis syndrome: seldom ulcerative and seldom involves the mucocutaneous junctions; more seborrhoeic.
- Pemphigus foliaceus: seborrhoea is the main sign and localized exudation and some superficial erosions may occur; seldom affects the oral mucosa and mucocutaneous junctions.
- Vesicular stomatitis: transient viral bullous disease that can affect the mouth, nasal and coronary band regions; spontaneous resolution in a few days.
- Chronic dermatophilosis (*Dermatophilus congolensis*): characteristic microbiology and epidemiology with signs mainly restricted to matting and tessellating of the hair on the dorsum and legs of badly managed horses; contagion means that several cases may be encountered.
- Chronic chorioptic mange (*Chorioptes equi*): pruritic disease mainly affecting the legs and head – self-trauma can cause localized ulcerative lesions with crusting but these are not related to mucocutaneous junctions. The mites are easily identified in brushings.

Key points: Pemphigus vulgaris and bullous pemphigoid

10 11 16 17 19 20 23 24

1. A very rare autoimmune vesico-bullous disease occurring in horses of all ages and breeds.
2. Signs usually develop slowly and progressively over some weeks. Mild to severe or even extreme erosions and ulcers with haemorrhage and pain (and sometimes secondary infections) develop particularly on oral and nasal mucosae and mucocutaneous junctions. Systemic signs include weight loss, anorexia and persistent or undulant pyrexia.
3. Diagnosis can be made from carefully collected biopsies. Intact vesicles are most useful but in any case the biopsy site must not be cleaned or clipped/scrubbed. The crusts must be harvested with the biopsy. Immunofluorescent diagnosis is offered by high quality laboratories.
4. Treatment is seldom useful but very high immunosuppressive doses of corticosteroids may be effective at least in the short term. Biopsies MUST be collected before steroid therapy starts.
5. The prognosis is best regarded as hopeless.

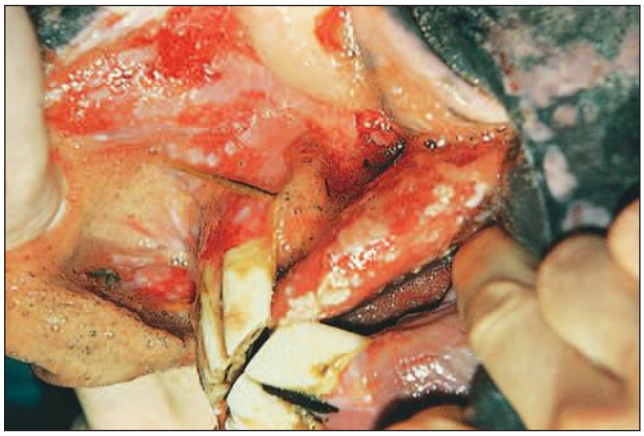


Figure 11.22 Pemphigus vulgaris ulceration and bulla formation in the oral mucosa. Note the lesions also at the mucocutaneous junction of the commissure of the mouth.

- Oral irritations and toxic burns: exposure-related local toxicity with rapid onset of severe damage in most cases.
- Renal failure (oral ulceration): polydipsia, weight loss and poor growth – seldom results in overt oral ulceration and in any case the ulcers are not localized at mucocutaneous junctions.

Diagnostic confirmation

- History and clinical examination are suggestive but diagnosis is usually made after exclusion of all other possible disorders.
- Skin biopsy is very useful. If possible a whole, intact vesicle or bulla should be obtained. Tissue can be examined directly or by immunohistochemistry (with immunofluorescence).
  - In bullous pemphigoid multiple biopsies of fresh lesions must be taken. They will show characteristic subepidermal vacuolar alteration and subepidermal clefting with vesicle formation.
  - Direct immunoglobulin and fluorescent antibody testing of biopsy sections shows linear deposition of immunoglobulins and complement at the basement membrane of skin or mucosa.
- Serum samples can be tested for anti-horse globulins (Coombs test) but results are variable. The test is not commonly available.

Note

The value of biopsy can be significantly affected by pre-administered corticosteroids and so the best results are obtained on first presentation when the lesions are at their youngest and most obvious and before any treatment has been given that might alter the pathology.

Treatment

Very high (immunosuppressive) doses of systemic corticosteroids such as prednisolone may have to be maintained for life. Once an improvement has been obtained using high doses, progressive reduction may establish a minimal effective therapeutic dose. Gold salts, azathioprine and pentoxifylline can be considered (see above in Pemphigus foliaceus section).

CD

The prognosis is very poor and most cases are euthanized either upon diagnosis or when therapy fails to control the disease usefully. A few cases can be managed effectively, however, and there are anecdotal reports of recovery from confirmed pemphigus vulgaris and these may form a different subset of the cases where a primary underlying trigger factor is involved and then resolves.

CD

Equine cutaneous (discoid) lupus erythematosus

Profile

Little is known about this putative autoimmune condition in horses as so few cases have been reported (Scott 1990, Scott & Miller 2003b). The condition is thought to be similar to the human and canine condition and may be a benign form of systemic lupus erythematosus-like syndrome (see p. 276). It is thought the condition represents a photo-exacerbated cutaneous autoimmune disorder, similar in pathogenesis to systemic lupus erythematosus-like syndrome, in which the lesions are limited to the skin (or at least in which systemic signs are very rare).

It is a rather benign but very chronic disorder in the horse. Triggering factors such as sudden, strong sun exposure (especially ultraviolet light), extreme cold, drugs and some stress circumstances may be reported.

Key points: Equine cutaneous (discoid) lupus erythematosus

5 10 11 16 21 22 23

1 A rare, benign cutaneous immune-mediated condition. Photo-exacerbation is a major factor. Stress and changes in climatic conditions can be trigger factors also.
2 Signs are limited to the skin and usually the face and eyes. Roughly circular, oedematous, erythematous and alopecic lesions occur and merge to produce coalescent areas of thickened exudative skin. Scaling and crusting are common. Conjunctival and corneal inflammation can occur. Systemic signs are very unusual.
3 Diagnosis is based on biopsy of an early lesion. There is pigmentary incontinence, and immune depositions in the hair follicles and immunohistochemical stains will reveal immunoglobulin depositions in the basement membrane. Antinuclear antibody (ANA) and lupus erythematosus (LE) tests are usually negative.
4 Treatment is supportive only – avoidance of sunshine is possibly the most important management procedure. Topical or systemic corticosteroids may be used to control periods of exacerbation.
5 The prognosis is good because there is little or no systemic implication in the horse.

Clinical signs

This is best regarded as a photo-exacerbated dermatitis with characteristic roughly symmetrical, discoid, raised, erythematous, hairless, exudative lesions. The more rostral regions of the face and the periorbital region are commonly the earliest sites involved. The conjunctiva and even the cornea can be affected also (Fig. 11.23 and **CD11 • 4**). Extensive areas of the neck, body and perineum can be similarly affected in occasional cases. The limbs are seldom if ever involved.

The skin plaques show varying degrees of oedema, erythema, scaling and crusting. Pigmentary changes (leukoderma/leukotrichia) are features (Fig. 11.23 and **Fig. CD11 • 4**). The lesions are usually neither painful nor pruritic. The roughly circular nature of the lesions may not always be obvious.

The affected horse seldom shows systemic involvement even though the skin changes can be quite extensive.

Differential diagnosis

- (Generalized) granulomatous enteritis syndrome: more obvious systemic signs and prominent seborrhoea; sometimes some pruritus.

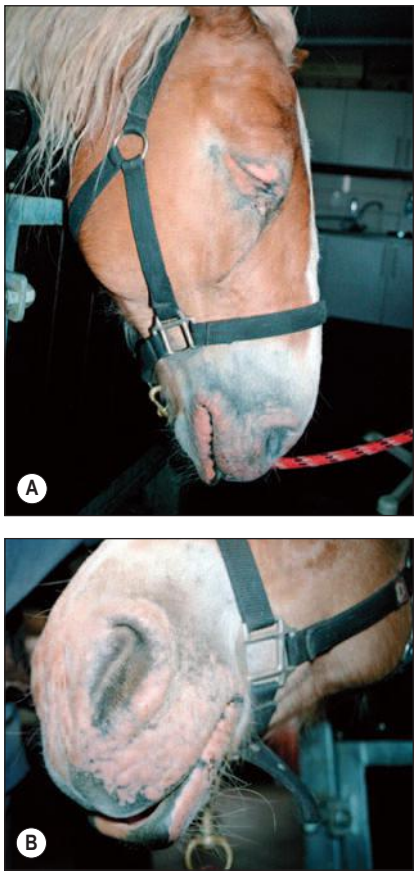


Figure 11.23 (A, B) Discoid lupus in a Haflinger pony. Note the conjunctival involvement and the distribution around the face and eyes with loss of pigment and prominent epidermal hypoplasia. (Figures courtesy of Dr Eva Krudewagen.)

- Pemphigus foliaceus: localized or extensive areas of hair loss, dermal hyperaemia with some exudation and severe scaling of the skin; very obvious pathology when early lesions can be biopsied. Some systemic signs.
- Photosensitization: localized severe necrotizing dermal inflammation in non-pigmented skin.
- Vesicular stomatitis: localized ulcerative condition that is transient and associated with fever and rapid recovery.
- Chronic dermatophilosis (*Dermatophilus congolensis*): crusting and typical paintbrush lesions are most obvious. Characteristic filamentous bacteria can be identified on smears taken from the crusts and pus.
- Chronic chorioptic mange (*Chorioptes equi*): more pruritus and seldom affects the face as badly as the legs.
- Oral irritations and toxic burns: history of exposure.
- Conjunctivitis: bacterial inflammation of the conjunctiva only, with no primary dermal implications – some horses may cause blepharitis if they rub the eyes.
- Conjunctival/corneal carcinoma: limited to the conjunctiva, margin of the eyelid or cornea with proliferative and destructive tumorous masses. Very typical histology.
- Occult/verrucose sarcoid: usually other types of sarcoid are present and they seldom result in depigmentation; typical histopathology.

Diagnostic confirmation

- History and clinical examination are suggestive but diagnosis is usually made after exclusion of all other possible disorders.
- Skin biopsy is very useful. However, antinuclear antibodies are not well categorized in the equine condition. Deposition of immunoglobulin and complement 3 (C3) may be detected at the dermo-epidermal junction. There is a diagnostically useful pigmentary incontinence.

Treatment

Avoidance of the trigger factors including exposure to strong sunlight is a major aspect of management; high factor sunscreens can be helpful but the areas involved often preclude this and avoidance is a better strategy. Topical treatment with corticosteroids (dexamethasone or hydrocortisone) can be used for localized areas. However, this is not always possible and so systemic corticosteroid treatment during periods of exacerbation can help. A one-month course of oral prednisolone (at 1–2 mg/kg q 24 h in the morning) is instituted and then reduced progressively once a clinical remission is achieved. Topical corticosteroids may be useful in limiting the amount of systemic drug that is required to manage the condition. Some cases are reported to benefit from high dietary omega-3 fatty acid supplements but there are too few cases to be sure that this is an important aspect.

The prognosis is good and there are no material systemic effects reported, but recurrences may occur. If a trigger factor can be established and this can be avoided then the condition may go into long-term remission.

The depigmented skin is more liable to actinic dermatitis and so there are several reasons for keeping affected horses out of strong sunlight as much as possible.

Immune-mediated (cutaneous) vasculitis

Profile

This is an uncommon clinical entity characterized by purpura, oedema, necrosis and ulceration particularly affecting the lower limbs and oral mucosa due to combined type 1 and type 3 hypersensitivity reactions. Most cases arise from intravascular reactions between antibodies and protein or bacterial proteins. The most established form of the condition is purpura haemorrhagica. This condition is usually described separately because of its very well-defined clinical and pathological features (Reef 1987). In many equine cases the disorder is related to antigens such as drugs (penicillin) and to streptococcal bacteria. It is important to realize though that tumour antigens, other drugs and other infections can be responsible. Autoantibodies can also be involved such as in some forms of systemic lupus erythematosus-like syndrome of horses.

Immune complex deposition on the vascular endothelium results in inflammation and microvascular thrombosis. This necrotizing vasculitis usually affects small vessels

Key points: Immune-mediated (cutaneous) vasculitis

9 11 15 17 19 20 21 24

1. Rare, small-vessel immune-mediated vasculitis caused most often by drugs (penicillin) and streptococcal bacterial proteins.
2. Palpable purpura with prominent skin lesions and in some cases multi-organ involvement. Petechial haemorrhages and necrotizing cutaneous vasculitis plaques are the main signs; most of the cutaneous lesions are roughly circular at least in the early stages. Concurrent haematuria, colic and protein loss may reflect non-cutaneous organ involvement.
3. Diagnosis is based on clinical signs and the historical exposure to the panel of causative 'allergens'. Biopsy is definitive but immunohistochemistry and fluorescent antibody confirmation is only available at some laboratories.
4. Treatment must focus on removal of the causative antigen. Inevitably treatment will rely on the use of systemic corticosteroids (usually immunosuppressive doses of oral prednisolone) – it is wise to remove all other drugs in case one of these is responsible for the condition. Failure to respond should trigger a reassessment of the case and then the use of pentoxifylline or azathioprine can be considered. Antibiotics should only be used if the 'trigger' factor is an infectious agent.
5. Remission is possible and some cases are cured permanently but others are recurrent and some are persistent in spite of the treatment. A guarded prognosis is always warranted.

only (arterioles, capillaries and small venules) and although the pathology is usually generalized the most obvious signs are found in the skin and exposed mucous membranes. It has been suggested that immune-mediated small vessel vasculitis is involved in both the well-recognized verrucose pastern dermatitis syndrome (Ferraro 2001) (greasy heel, see p. 354) and the interesting condition called (photoactivated) pastern and cannon leukocytoclastic vasculitis (Sloet van Oldruitenborgh-Oosterbaan et al 2000) (see p. 273).

Clinical signs

Signs occur characteristically (but not exclusively) 2–4 weeks after exposure to the causative agent. A history of respiratory infection due to *Streptococcus equi* or *Salmonella*, for example, or exposure to a drug such as penicillin or erythromycin are a major aspect of most cases. Foals with septicaemia (and in particular those showing systemic inflammatory response syndromes (SIRS)) may be affected by extensive cutaneous necrotizing immune-mediated vasculitis.

Focal, roughly circular or more diffuse areas of oedema, erythema, necrosis and ulceration develop. Initially the lesions are thickened, exudative and painful on palpation. There is no pruritus and while pain is shown only in the earliest stages, in some horses it remains a problem for many weeks. Cutaneous necrosis results in a hard black eschar at the site that may take some weeks to slough. The ears, tail and hooves may slough.

The most prominent signs occur on the coronet, pastern and fetlock (Fig. 11.24). The lips and periorbital tissues are also affected in many cases.

Systemic signs of pyrexia, weight loss, depression and anorexia are often present.

It may become chronic or recurrent and some cases completely fail to resolve.



Figure 11.24 Immune-mediated vasculitis lesions on the distal limb (note that the lesions in this case are not restricted to non-pigmented skin).

Differential diagnosis

- Dermatophilosis (*Dermatophilus congolensis*): typical focal, mildly painful cutaneous pustules and crusting with no necrosis. Typical bacterial identification from direct smears and cultures.
- Dermatophytosis (*Trichophyton* spp.): superficial mycosis lesions with no evidence of cutaneous necrosis. Typical mycology and epidemiology.
- Staphylococcal or streptococcal pastern folliculitis: seldom necrotizing and usually very painful, mildly exudative; sometimes more limited in area and producing pustules; bacterial culture is definitive.
- Purpura haemorrhagica: a particular form of vasculitis with the same aetiopathogenesis following streptococcal or viral respiratory infections.
- Pemphigus foliaceus: generalized exfoliative dermatosis with focal or widespread alopecia but no necrosis; definitive pathology of early lesions.
- Bullous pemphigoid/pemphigus vulgaris: severe bullous conditions resulting in erosions and secondary infection; definitive histopathology.
- Drug eruption: often similar to vasculitis but a vague and ill-defined complex of cutaneous and sometimes systemic responses; definitive exposure but can be months previously.
- (Generalized) granulomatous enteritis syndrome: generalized systemic condition without cutaneous necrosis; more generalized exfoliative seborrhoeic dermatosis.
- Leukocytoclastic vasculitis: a particular form of necrotizing vasculitis restricted to the distal limb regions – usually of white hind limbs.
- 'Greasy heel' syndrome: a particular form of vasculitis as part of the greasy heel syndrome/verrucose pastern dermatitis.

Diagnostic confirmation

- Clinical appearance may be typical in later stages.
- Clinical pathology (haematology and biochemistry) shows normal platelet counts, with mild anaemia and panleukopenia. Serum proteins are often abnormal, reflecting sustained inflammatory responses.
- Biopsy shows neutrophilic, eosinophilic, lymphocytic infiltration and mixed vasculitis. Diagnostic biopsies are best taken within 24 hours of a fresh lesion occurring. By the time necrosis occurs the diagnosis can be complicated by infection and sloughing.

Treatment

The main approach has to be directed at removal of any suggested possible cause. This may mean withdrawal of all drugs and medications. If infection is the likely cause then appropriate antibiotics can be maintained; penicillin and erythromycin may be particularly involved in some cases so alternative antibiotics may be preferred.

Provided a diagnosis can be made early, oral prednisolone (with a concurrent long course of antibiotics) should be given twice daily until regression occurs and then reduced to a minimum effective alternate-day dose. After full remission the dose can be gradually reduced. Some cases show a poor response to corticosteroids and then azathioprine or pentoxifylline can be used.

The prognosis is always guarded – provided that early treatment is effected the results are quite good. Some cases are cured by the most direct approaches but others are slower. Recurrences are common and some cases fail to respond to any treatment and even eventually succumb. The worst cases are those that show serious internal organ involvement; cutaneous vasculitis alone usually heals eventually provided that the cause is removed.

Pastern (and cannon) leukocytoclastic vasculitis

Profile

This is a specific sporadic disease of equine skin, which is probably more common than realized. Mature horses are usually affected without sex predilection. The non-pigmented extremities are most frequently affected and the disease usually occurs in summer in regions with strong sunlight exposure (Stannard 1987, 2000a).

The aetiopathogenesis is uncertain, but IgG deposition and/or C3 portion of complement have been detected in the earliest stages by direct immunofluorescence of affected vessel walls. Percutaneous absorption has not been ruled out, but non-pigmented skin involvement suggests a role of ultraviolet light. However, the disease is not a true photosensitization. Records of affected horses also indicate no known contact with photosensitizing compounds and liver function is usually normal (Sloet van Oldruitenborgh-Oosterbaan et al 2000).

Interestingly, individual horses are usually involved and the most characteristic lesions occur on the lateral (and less commonly on the medial) aspects of the pasterns and cannons of non-pigmented hind limbs. It is entirely possible for the animal to have only one of the non-pigmented limbs affected. The distribution of the cutaneous damage is a feature of the disease.

Key points: Pastern (and cannon) leukocytoclastic vasculitis

5 11 15 17 22 24

1 Relatively common disorder, increasing in prevalence, that is often misdiagnosed. Photo-exacerbation is a feature of the disease – it only occurs in pastured horses in summer months or in circumstances of strong sunlight exposure.
2 Early lesions are painful, erythematous and exudative inflammation of limited (usually lateral) areas of the pastern and cannon regions of one or more pigmented (usually hind) limbs occurs. The lesions often have a roughly circular appearance; this may only be apparent when the hair is clipped out. Secondary infection is common and the lesions can 'spread' to involve the dorsal and plantar (palmar) pastern and coronet.
3 Diagnosis is based on the characteristic early lesions but sometimes cases are not presented at this stage. Histologically the disease is non-specific.
4 Treatment depends on removal from sunlight, close clipping of the affected region and application of soothing emollients and corticosteroids topically.
5 Treatment is not easy and can be complicated by secondary bacterial and fungal infections. Recurrences are common and failure to control the progression can result in severe long-standing pastern dermatitis. The suggestion that pasture exposure is involved means that even night-time grazing can be a potential hazard.

Clinical signs

There is an acute onset of erythema, oozing and crusting that is clearly demarcated and restricted to the white areas of the distal limb (and particularly the lateral aspect of the hind pasterns) (Fig. 11.25 and **Fig. CD11 • 5A and B**). One pigmented limb may be affected while others show no signs at all. Pigmented limbs are seldom, if ever, affected. In a few cases the muzzle may be involved but this is more usually genuine actinic dermatitis/sunburn (see p. 303).

The condition is more painful than pruritic. Erosions, ulceration and oedema of affected limbs are more extensive than might be expected for the size and extent of the lesion. The exudate takes on a warty appearance and character, and removal of the scabs and crusts is quite difficult. Secondary infection with *Staphylococcus* spp., *Streptococcus* spp. and *Dermatophilus congolensis* bacteria is common. An accurate history is an important aspect of the case and its management. Bacterial swabs may be important if the condition has been prolonged or if initial treatments fail to cure it. Swabs should always be taken at the outset before any medication is undertaken but the significance of the cultures should be interpreted with care.

The outward appearance of the disorder may belie the severity and extent of the inflamed skin, which can only be truly appreciated by close clipping of the hair. The oedema can sometimes be quite severe. Removal of the crust and the matted hair reveals an inflamed area of skin (often this is roughly circular) restricted to limited areas of the pastern and cannon skin.

The remarkable distribution of the dermatitis is a feature of the disorder. Most often the lateral aspects of a hind leg are most affected and quite often there is an area just under the fetlock that is remarkably free of any pathology

CD

(Fig. CD11 • 6A and B). Other cases have a more generalized lateral and medial inflammatory dermatitis (Fig. 11.26 and **Fig. CD11 • 7**). The plantar (palmar) and dorsal aspects are much less often involved.

Differential diagnosis

- Pastern folliculitis (mixed *Staphylococcus* spp.): highly painful disorder, often worse or restricted to white limbs but very painful and diffuse. Usually involves the plantar/palmar aspects more than the lateral.
- Dermatophilosis (*Dermatophilus congolensis*): mildly painful, mildly exudative condition associated with unhygienic conditions.
- Photosensitization – especially due to plants: careful history to eliminate plant aetiology.
- Actinic dermatitis: liver function tests should always be performed on any case that shows dermatitis restricted to the non-pigmented skin.
- Pemphigus foliaceus: seldom if ever restricted to white limbs and definitive histopathology.

Note

All horses showing dermatitic lesions restricted to non-pigmented skin *must* be tested for liver function.

Diagnostic confirmation

- Clinical appearance on the lateral aspect of a non-pigmented pastern is typical.
- Biopsy is usually non-specific unless the earliest lesions can be identified. Acute changes show leukocytoclastic vasculitis with vessel wall necrosis and thrombosis. Epidermal changes include hyperplastic and degenerative changes and, in chronic cases, papillomatosis has been reported.

Treatment

Close clipping and a single antibacterial wash with a chlorhexidine shampoo is advised with strict avoidance thereafter of exposure to sunlight and the pasture involved.

High (immunosuppressive) doses of corticosteroids (see Chapter 4, p. 96) daily for up to 2 weeks with a gradual withdrawal over 4 weeks and repeated use of emollient cream and keratolytics such as salicylic acid can be helpful.

There are occasional recurrences during or soon after treatment, requiring further courses of corticosteroids.





Figure 11.25 (A, B) Photo-exacerbated, pastern and cannon leukocytoclastic vasculitis. Note the involvement of only the right hind leg although the other is also non-pigmented. Both medial and lateral aspects were involved. Further pictures of this and another case are shown in **Figures CD11 • 5, CD11 • 6 and CD11 • 7**.



Figure 11.26 Pastern and cannon leukocytoclastic vasculitis characteristically affecting only the lateral aspect of the pigmented hind limb. The left leg is obviously unaffected and there is no abnormality on the medial aspect of the white leg. The full extent of the disorder can be appreciated after close clipping.

Soothing creams containing antiseptics, antibiotics and steroids and factor 20+ sunblock lotions can be helpful.

Protection of affected areas from direct or reflected ultraviolet light by stabling in daylight, high factor sunscreens and using leg wraps is helpful.

Purpura haemorrhagica (immune-mediated vasculitis)

Profile

This classically described, rare, sporadic syndrome of an immune-mediated vasculitis is commonly associated with the recovery phase of an upper respiratory infection (most often *Streptococcus equi* var. *equi* or *Streptococcus equi* var. *zooepidemicus*) (Bryans et al 1964). Young and stressed horses are possibly more susceptible but any horse can be affected and virus infections, vaccination for respiratory tract viruses and strangles (*Streptococcus equi*) have been implicated in some cases.

Immune complexes are probably deposited on the walls of peripheral blood vessels, causing localized damage and increased vascular permeability. Leakage of proteins into the extravascular space causes severe oedema (which may initially present as discrete urticarial wheals).

The condition is characterized by oedema and purpura (haemorrhage into tissues), thrombosis of small vessels and necrosis (particularly of the skin). The changes are often generalized so the early skin and mucosal signs are important early signals of the disease.

Clinical signs

Typically there is an acute onset of roughly symmetrical subcutaneous oedema of the face, muzzle, limbs and ventral abdomen that develops 2 days to 4 weeks after a respiratory tract infection or vaccination. Initially urticarial wheals may be seen to develop (mostly over the abdomen and thorax) and then severe diffuse oedema develops in the limbs, characteristically with a clear demarcation at the elbow and stifle (i.e. level with the bottom of the heart) (Fig. 11.27). Oedema is often severe (angio-oedema) with serum exudation from the skin.

Petechial haemorrhages may be detected in the visible mucous membranes; sometimes these are very faint while in other cases there are extensive ecchymoses. Haemorrhages can also been detected in the skin (especially in thin-skinned areas such as the muzzle and pinnae).

Extensive skin erosions, necrosis and exudation are sometimes encountered (Fig. 11.28).

Depression, anorexia and limb stiffness can be present. Severe pulmonary oedema and central nervous signs arising from cerebral oedema may be life-threatening.

Differential diagnosis

- Bullous pemphigoid/pemphigus vulgaris: little oedema and not related to prior respiratory tract infection. Generalized bulla formation with crusting, alopecia and ulceration particularly involving mucocutaneous junctions and the mouth.

Key points: Purpura haemorrhagica

9 11 16 19 20 24

1. A rare classical syndrome associated with recovery from respiratory tract infections (particularly *Streptococcus equi* var. *equi*) or after vaccination.
2. Acute onset of petechial haemorrhages on nasal and oral mucosae and extensive limb oedema – often with a very typical cut-off pattern and sharp demarcation of oedema at the lower thorax–stifle regions. More severe skin changes such as necrosis, sloughing, exudation and ulceration develop later in the course. Systemic signs include pulmonary oedema and central nervous symptoms.
3. Diagnosis is not usually difficult with a defined history of respiratory tract infections. Clinical pathology shows a non-thrombocytopenic purpura and a normal plasma albumin. Histological evidence can be found in some cases.
4. Treatment involves removal of the cause (if that is defined/possible) and the administration of high doses of prednisolone or dexamethasone. Antibiotic treatment is often required. Bandaging of limbs should be undertaken with great care to avoid further skin damage.
5. The prognosis is guarded. Some respond well and fully, others do not. Many cases are euthanized.



Figure 11.27 Purpura haemorrhagica showing the typical oedema pattern involving the limbs with a sharp demarcation level with the bottom of the thoracic cavity.



Figure 11.28 Purpura haemorrhagica showing extensive skin erosions with exudation on the distal limb.

- Systemic lupus erythematosus-like syndrome: generalized exfoliative alopecic condition with scale and depigmentation.
- Drug eruptions: can be difficult to define the possible changes but tend to be more diffuse, scaling and possibly some crusting with alopecia; oedema is not a common feature.
- Actinic dermatoses: sunlight exposure and changes restricted to white-skinned regions.
- Dermatophilosis (*Dermatophilus congolensis*): dorsum and limb inflammatory disorder; bacteria can be identified.
- Infectious vasculitis (e.g. equine viral arteritis): febrile and related to exposure to viruses.
- Disseminated intravascular coagulopathy: evidence of haemorrhages and much prolonged clotting times.
- Lymphosarcoma: nodular disease with systemic signs associated with neoplastic disease.

Diagnostic confirmation

- History of respiratory disease some 2 days to 4 weeks previously.
- Characteristic clinical signs.
- Non-thrombocytopenic purpura (normal platelet counts).

Treatment

Treatment is restricted to high doses of systemic corticosteroids with aggressive antibiosis (high doses of penicillin and/or aminoglycosides). It is important to eliminate drug eruptions before embarking on these treatments. Some cases of drug eruptions which look very similar may be the direct result of these drugs administered some weeks previously for the respiratory tract infection.

It is tempting to attempt to apply supportive limb bandages but sometimes these can be very dangerous – they simply cause more circulatory obstruction and greater necrosis.

The prognosis is very guarded. Some cases respond rapidly and fully whilst others may respond initially only to relapse after some weeks. A few are progressive in spite of therapy, and secondary skin and respiratory tract infections and ongoing plasma loss may lead to early euthanasia. Recurrence carries a very poor prognosis.

Equine nodular eosinophilic vasculitis and arteritis

Profile

This rare nodular disorder predominately affects the skin of the distal limb regions. Few cases have been reported (Scott & Miller 2003c).

Clinical signs

Non-pruritic nodules appear in the skin of the limbs, sometimes with (interlinking) firm cords. The nodules are mildly painful to palpation and may ulcerate and discharge. They may appear suddenly then regress and slowly reappear. The nodules may become chronic and progressive. A seropurulent discharge and crusting are reported. Systemic signs are absent.

Key points: Equine nodular eosinophilic vasculitis and arteritis

1. Rare, poorly documented nodular disease that mainly appears to affect the distal limbs.
2. Solid, occasionally ulcerated, painful cutaneous nodules and interconnecting firm cords of eosinophilic inflammation.
3. Diagnosis is based on biopsy alone.
4. Treatment is not defined but corticosteroids may be logical.
5. The outlook is guarded. Too few cases have been reported to warrant any firm statement on prognosis.

Differential diagnosis

- Eosinophilic granuloma: seldom larger than 1 cm diameter and mainly distributed on dorsum, buttock and neck.
- Mastocytoma and other non-specific granulomas: typical histology.
- Cutaneous histiocytic lymphosarcoma: multiple nodules isolated in the skin over any area and often involving the mouth and pharynx.
- Ethmoid haematoma/other neoplasia: respiratory signs.
- Squamous cell carcinoma: very rare on the limbs and more destructive than nodular vasculitis/arteritis.

Diagnostic confirmation

- Nodules in skin of the limb, some interlinking and isolated cords of similar tissue and nodular thickening of nasal mucosa and occasional nasal bleeding.
- Biopsy shows a prominent eosinophilic vasculitis and arteritis in the deep dermis.

Treatment

There are too few cases to define specific treatment. Corticosteroids are probably the only likely approach but the nodules may remain for years.

Systemic/cutaneous lupus erythematosus-like syndrome (SLE-like syndrome)

Profile

This rare, incompletely defined, multisystemic autoimmune disease of horses differs in several ways from classical forms encountered in humans and dogs. The syndrome has some features of both discoid lupus erythematosus (DLE) and systemic lupus erythematosus (SLE). Multiple organ involvement including the skin is characteristic. A variable number of cases have significant anti-nuclear antibody (ANA) (Stannard 1994b). True lupus cells have not been detected in horses. In some equine cases the disease appears as a more benign form.

The pathogenesis is probably multifactorial. 'Autoimmune disease' results from the interaction of genetic, immunological, environmental and possible infective relationships (particularly viral) as well as iatrogenic causes involving drug administrations. These would seem to be a reasonable basis

Key points: Systemic/cutaneous lupus erythematosus-like syndrome

1. Rare, multisystemic autoimmune disorder with ill-defined aetiopathogenesis.
2. Multiple organs are invariably involved but the most obvious signs are cutaneous. However, these are non-specific and include symmetrical alopecia, seborrhoea, oedema of extremities and loss of cutaneous pigmentation.
3. Diagnosis requires the identification of multisystemic involvement. Positive tests for antinuclear antibody (ANA) are probably definitive but the test is unreliable. Histological findings are usually non-specific but in combination can assist diagnosis.
4. Treatment with high-dose prednisolone is suggested, with reductions to a minimum effective daily or preferably alternate-day dose.
5. The prognosis is very guarded – most cases are euthanized sooner or later.

for induction of the disease. The aetiopathogenesis might include the following:

- Immunological aspects involving antibodies (IgG and IgM) and complement C3 portion have been observed at the epidermal–dermal skin junction and have also been seen in small superficial blood vessels. Circulating antinuclear antibody (ANA) may be present (but may be positive in horses not showing the clinical signs also).
- Trigger factors including (viral) infections, changes in environment, extremes of temperature, drugs, stressful events such as pregnancy, heavy work, etc. are usually reported.
- Underlying systemic disease such as lymphosarcoma (or other internal neoplasm) or other chronic inflammatory processes may also be active trigger factors.
- Virus-like particles have been seen in electron microscopic studies of both SLE and DLE in other species but are not described in the horse.

Clinical signs

SLE is classically acute or insidious in onset and has a characteristic remitting, chronic course with periods of improvement and relapse. Febrile responses may be detected and the condition affects the skin, joints, kidney and serosal surfaces. The dermatological signs are often the only indication in the early stages.

Erythema, scaling and exudation occur early (Fig. 11.29) and the skin of long-standing cases has a wrinkled, leathery appearance with extensive scaling (Fig. 11.30). Haired areas may show various degrees of alopecia. There is often a sharp demarcation between pigmented and depigmented areas.

Apparent photosensitization can be present involving the depigmented areas and this makes the diagnosis difficult. Rapid or insidious depigmentation occurs around the eyes, lips, nostrils (Fig. 11.31), vulva, anal ring and prepuce.

The more severe SLE-like cases show various multiple organ involvement including prominent lameness (polyarthritis), uveitis and pyrexia, and weight loss is



Figure 11.29 Systemic lupus erythematosus-like syndrome showing diffuse, severe, exudative, dermatitis affecting the distal limb region. All four legs showed similar signs (including two which were pigmented).



Figure 11.30 Systemic/cutaneous lupus erythematosus-like syndrome showing chronic skin involvement with roughening and thickening of the skin and a leathery appearance.



Figure 11.31 Systemic lupus erythematosus-like syndrome. Typical focal areas of haemorrhage and necrosis in the skin of the muzzle.

common. Petechiation of mucous membranes, skin, hypopyon (inflammatory debris in the anterior chamber of the eye; Fig. 11.32) and hyphaema (blood in the anterior chamber of the eye) can be present in severe cases. Significantly, the intraocular changes can fluctuate over a relatively short time.

Involvement of several organ systems simultaneously is a strongly suggestive feature of the disorder, but some cases have limited overt signs. The systemic signs may be more serious than the cutaneous signs.

Differential diagnosis

- Drug eruptions: history of administration but signs can be similar although seldom marked systemic signs.
- Occult polysystemic infections (including borreliosis or Lyme disease) or neoplasia (including internal forms of lymphosarcoma): these may be involved in the pathogenesis of SLE-like syndrome and so symptoms can be similar.
- Chronic *Culicoides* spp. hypersensitivity (sweet itch): severely pruritic disorder affecting defined sites with typical historical seasonal and management aspects.
- Chronic/recurrent dermatophilosis (*Dermatophilus congolensis*): localized bacterial infection on dorsum and limbs mainly.
- Other depigmentation diseases:
  - Vitiligo: asymptomatic depigmentation in face and perineal regions mainly.
  - Arabian fading syndrome: genetic predisposition and largely asymptomatic apart from actinic dermatitis superimposition.
  - Appaloosa parentage: breed-related depigmentation and some hair loss but otherwise asymptomatic.
- Sarcoidosis and (generalized) granulomatous enteritis syndrome: very similar but tends towards more seborrhoea and less depigmentation.
- Leukoderma, scarring or chronic irritation: history and obvious scarring in limited areas.
- Scalding (urine/faeces/wound fluids): obvious aetiology and localized signs.



Figure 11.32 Systemic lupus erythematosus-like syndrome showing severe hypopyon (accumulation of inflammatory debris in the anterior chamber of the eye). This showed a remarkable improvement over 3–4 days when it was only just visible, but it returned overnight some 3 days later.

Diagnostic confirmation

- Diagnosis is extremely difficult. No single test or sign should be taken as pathognomonic. A full range of diagnostic aids should be employed as some may show false positive or false negative results from time to time.
- Multiple organ involvement is typical.
- Biopsy of skin (taken into formalin for conventional histopathology and possibly Michel's medium for immunofluorescent studies) is characteristic.
- Serum can be tested for antinuclear antibody (ANA) with variable results. The Coombs test may be positive in some cases.
- Blood analysis is usually non-specific. High plasma fibrinogen, hypergammaglobulinaemia and hypoalbuminaemia with elevated white cell counts with a left-shifted neutrophilia are possible.

Treatment

Attempts should be made to diagnose and then treat or remove any possible trigger factors or underlying disease.

Treatment with antibiotics should always precede and run concurrently with corticosteroid treatment. Prolonged corticosteroid treatment courses are needed.

False cases may recover, while confirmed positive cases seldom undergo remission for anything other than short periods. Acute and severe remissions and deterioration are common.

Alopecia areata

Profile

This is a rare, cell-mediated, (presumed) autoimmune skin disease which affects horses of all breeds and ages. The increased awareness of the disease has resulted in more identified cases. (Stannard 1994c).

The aetiopathogenesis seems to involve the presence of T lymphocytes directed against the hair matrix and root sheath and the epithelium of hair follicles. There is some suggestion of a hereditary factor as some 20% of cases are genetically related.

Key points: Alopecia areata

10 15 16 20 23 24

1. Occasional presumptive autoimmune condition localized to regional areas of the skin or sometimes more generalized.
2. Asymptomatic, non-inflammatory alopecia in small circular areas (most often) or larger areas. The body, face and neck are most affected. Mane and tail hair can be involved with other forms or on its own. The remarkable feature is the complete absence of any overt clinical alteration in the skin apart from the alopecia.
3. Diagnosis can be confirmed by biopsy.
4. No treatments, including high-dose steroids, are effective.
5. Some cases are progressive, some improve with time and others remain static. A few are reported to vary from season to season.

Clinical signs

There is insidious or rapid onset of an otherwise asymptomatic (non-pruritic, non-painful) diffuse thinning of the mane and tail and/or development of one or more reasonably circumscribed areas of partial or complete alopecia (Fig. 11.33). Affected areas may coalesce to produce extensive alopecia without any overt signs of inflammation.

Defective hoof growth may occur.

Spotted leukotrichia (see p. 335) or melanotrichia may be associated with hair loss in affected areas but in most cases the skin colour is what is revealed by the hair loss.

Differential diagnosis

- Dermatophytosis (*Trichophyton* spp., *Microsporum* spp.): expanding, mildly inflammatory scaling lesions showing characteristic mycology; spontaneous resolution over 6–12 weeks or response to specific treatment.
- Dermatophilosis (*Dermatophilus congolensis*): much more inflammatory and exudative – alopecia is not a feature.
- Occult sarcoid: sometimes difficult to differentiate but distribution of lesions helps. Biopsy and the presence of other forms of sarcoid are supportive aspects.

Diagnostic confirmation

- Non-specific alopecia with mane and tail dystrophy.
- Biopsy shows lymphocytic bulbitis with T-cell accumulations at hair bulbs and 'miniature' hair follicles. In early cases it occurs in anagen phase, whereas in chronic cases it is in telogen phase.
- May show defective keratinization in old lesions. Inflammation may be minimal and section may reveal only small telogen follicles lacking hair.

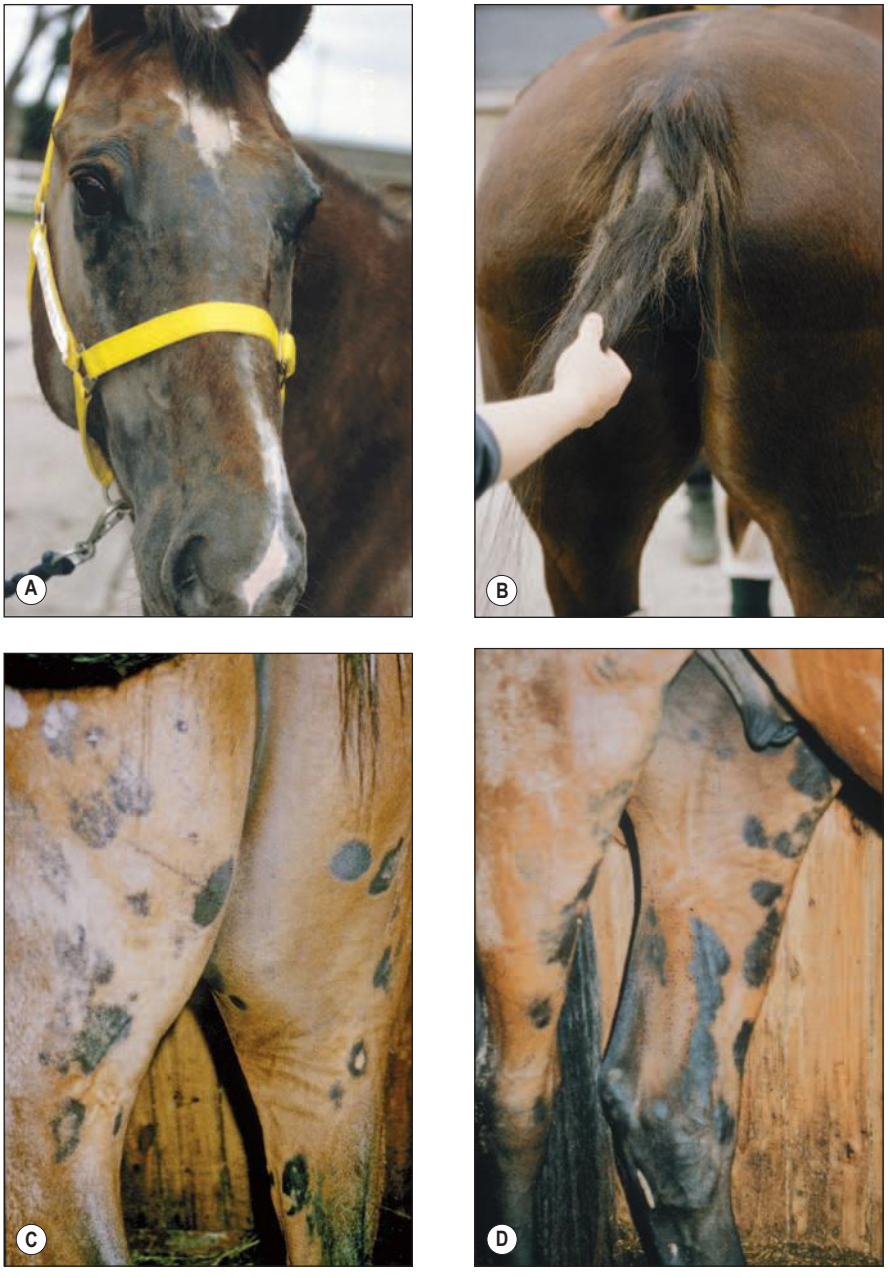


Figure 11.33 This series of pictures shows the variations that occur in alopecia areata. The face **(A)** and tail **(B)** can be diffusely and extensively affected. **(C, D)** The more common body forms are circular areas of non-inflammatory alopecia. These reveal the normal skin colour beneath (in this case a dark skin).

Treatment

There is no effective treatment. A few cases resolve spontaneously but most remain static and symptom-free apart from the alopecia. Some progress and others fluctuate from season to season. Spontaneous remission has been recorded but the chance of complete and permanent remission is poor.

CD

Sarcoidosis (generalized granulomatous disease)

Profile

This is a rare systemic idiopathic granulomatous disease. No age, breed or sex predilections are known. The cause and the pathogenesis are unknown (Stannard 1994d) and no infectious agent has been identified consistently although a viral aetiology has been proposed; it is assumed, however, that a multifactorial aetiology is involved (Stannard 2000b). The aetiopathogenesis is assumed to be broadly similar to the human condition having the same name, i.e. an abnormal immunological response to an ill-defined single or multiple antigens. Plant antigens, bacteria and viruses as well as drugs may be involved.

All described cases have so far (initially at least) been presented for investigation of skin disease. The condition is not related to the equine sarcoid but may be a part of the generalized granulomatous enteritis/dermatitis syndrome (Cotran et al 1994) with exfoliative dermatitis, (severe) wasting and sarcoidal granulomatous involvement of multiple organ systems (Spiegal et al 2006).

Clinical signs

Cutaneous signs are usually the first presentation – most affected horses are first presented for investigation of an exfoliative seborrhoeic dermatosis. The commoner skin lesions include generalized, extensive scaling and crusting of skin (Fig. 11.34) with variable alopecia of the face and limbs (in particular). These signs may more rarely be focal (limited areas) or multifocal (many areas, but discretely demarcated).

A second, even rarer form has skin lesions consisting of nodules or large tumour-like masses. Both skin forms may be present at the same time. Generalized wasting disease and ill thrift are common concurrent signs. Poor appetite and persistent or undulant low-grade fever can be identified in some cases **(Fig. CD11 • 8A–C)**. Peripheral lymphadenopathy and variable organic signs (including mediastinal and pulmonary granulomatous masses) may be detected. The presence of these may account for the poor exercise tolerance and various degrees of dyspnoea.

Liver and intestinal involvement is relatively common, giving rise to icterus and diarrhoea, respectively. Other organs are only rarely involved and so far no ocular, cardiac, splenic or central nervous lesions have been described.

Differential diagnosis

- Dermatophilosis (*Dermatophilus congolensis*): regionally restricted with typical crusting lesions from which the organism can be identified with relative ease. Short course provided that treatment is effective.
- Dermatophytosis (*Trichophyton* spp.): typical expanding alopecic areas and typical epidemiology. Spontaneous resolution within 6–12 weeks and characteristic microbiology.
- Pemphigus foliaceus: severe exfoliative dermatosis that can be rather similar – multisystemic signs are less prominent, however.
- Primary seborrhoea: extremely rare, non progressive seborrhoeic signs without any systemic implications.

Key points: Sarcoidosis

10 12 15 16 19 20 23 24

1. Rare, idiopathic condition assumed to be immunologically based.
2. Signs include acute or insidious onset of cutaneous signs including scaling, crusting and alopecia, usually over wide areas coupled with multi-organ involvement. Smaller, more focal areas and skin nodules can be found in some cases. Concurrent weight loss, undulant fever, thoracic and abdominal (but not peripheral) lymphadenopathy, anaemia and various organ degenerations can be present.
3. Diagnosis is very difficult – the cutaneous and haematological changes are non-specific. Exclusion of other alternatives is required. If possible, biopsy of internal lymph nodes is the best procedure.
4. Treatment is problematic – identification of the aetiological agent or process might help. Systemic corticosteroids are usually prescribed.
5. Some cases improve or even resolve spontaneously after some months or years while others remain a serious and deteriorating problem.



Figure 11.34 Sarcoidosis showing the typically extensive scaling and crusting of large areas of the skin surface.

- Exfoliative eosinophilic dermatitis: cutaneous and internal nodular lesions are recognizable by the eosinophilic and lymphocytic infiltrations.
- Immune-mediated diseases including systemic lupus erythematosus-like syndrome: differentiation from these may be difficult clinically and histologically.
- Erythema multiforme: localized skin patterning and characteristic histology.
- Food sensitivity/allergy: usually more pruritic and responsive to exclusion/avoidance diets.
- 'Hairy vetch' disease in the USA: some cases may be related directly to ingestion of and contact with hairy vetch.
- Verrucose sarcoid: static or slowly progressive with other types present at other sites. Occult lesions will have small nodules within the affected skin.

Diagnostic confirmation

- History and clinical examination. The diagnosis is most often made by exclusion of all other options.
- Haematology provides non-specific support including leukocytosis, increased fibrinogen and hyperglobulinaemia. Mild or more severe anaemia. Abnormal kidney and liver function tests.
- Skin biopsy shows dermatitis with frequent multinucleated histiocytic giant cells. Perifollicular and mid- or superficial dermal sarcoidal granulomas are characteristic.
- Organ biopsy from mesenteric and thoracic lymph nodes, lungs, gastrointestinal tract and liver may be affected with sarcoidal granuloma. These are very characteristic non-suppurative, non-caseating granulomas which may be microscopic. They consist of aggregations of multinucleated giant cells, neutrophils (in low numbers), lymphocytes and plasma cells.

Treatment

Dietary changes can be helpful. Removal of all artificial supplements, avoidance of any toxic plants or other substances and restoration of natural feeding patterns over some months or even years can be helpful – a similar or identical syndrome has been caused by hairy vetch (*Vincia velosa*) in field cases and in some cases of feed allergy. The former has not, however, been reproduced experimentally.

No effective treatment is described, but a few horses have experienced spontaneous remission for no apparent reason. Prolonged oral prednisolone may be helpful and immune-stimulating drugs such as levamisole have been used with variable (largely unsubstantiated) results.

Multisystemic eosinophilic epitheliotropic disease/generalized eosinophilic disorder (hyper-eosininophilic syndrome)

Profile

This is a rare but significant clinical condition that typically affects young horses (3–6 years). Older horses up to 12–15 years can be affected. The condition has been reported widely (Wilkie et al 1985).

The aetiopathogenesis is poorly understood but the multisystemic nature of the condition suggests a widespread organ disorder resulting possibly from a persistent immediate hypersensitivity reaction to toxins, concurrent immunological disease/neoplasia, viruses, parasitic or allergic causes (La Perle et al 1988). Food allergies have been suggested as a major aetiology. It is possible that the condition is similar to that in humans in which the eosinophilia arises from abnormal eosinophilopoietins including interleukin-5, secreted by abnormal clonal T lymphocytes.

Eosinophilic and lymphoplasmacytic infiltration of many organs creates generalized eosinophilic granulomas but in spite of the massive overall number of eosinophils involved there is usually no circulating eosinophilia. Cutaneous signs are the commonest reason for seeking veterinary attention.

The disease has similar clinical presentations to some cases of generalized granulomatous disease (see p. 280) and both of these disorders illustrate the fact that cutaneous signs are sometimes part of a major life-threatening systemic disease.

Clinical signs

The main presenting cutaneous signs include early widespread exfoliative dermatosis with intracutaneous nodules (similar to collagen granulomas) or linear cutaneous thickenings (similar to linear urticaria and some erythema multiforme). Ulceration and exudation of cutaneous nodules and larger areas of acanthotic epidermis are detected both clinically and histologically. Oral ulcerations can be present also – usually along the gingival margins. There may be moderate or severe pruritus but secondary bacterial skin infections are common and complicate the clinical syndrome (Fig. 11.35 and **Fig. CD11 • 9**).

CD

Salivary gland swelling can be found but this is a non-specific sign. Concurrent weight loss, diarrhoea, colic and abdominal distension as a result of an intensely eosinophilic effusion can be encountered (Fig. 11.36).

Key points: Multisystemic eosinophilic epitheliotropic disease/generalized eosinophilic disorder

11 16 17 18 19 20 23 24

1. Rare, but important disorder affecting the skin and internal organs characterized by eosinophilic and lymphoplasmacytic organ infiltration and granuloma formation.
2. Chronic progressive exfoliative dermatitis is the main external symptom. Cutaneous spherical or linear nodules may be detected. Diarrhoea, chronic weight loss, colic, recurrent pyrexia and multi-organ failure result in rapid deterioration over 1–12 months.
3. Diagnosis can be confirmed by biopsy. Rectal and peripheral lymph node biopsy can also be supportive. Often the cutaneous signs have been largely ignored by owners even though they are the most obvious early signs. Diarrhoea and evidence of organ infiltration from biopsy can help diagnosis.
4. Treatment options are limited to the use of immunosuppressive doses of corticosteroids (usually prednisolone) and palliative care. Hypoallergenic diets may help.
5. The outlook for affected horses is poor, particularly if there is obvious wasting. Early diagnosis and aggressive treatment may be successful but relapse follows quickly when steroids are withdrawn or even when the dose is reduced.

Differential diagnosis

- Chronic granulomatous disease (sarcoidosis): very similar disease but with notably different histological findings.
- Disseminated (malignant) melanoma: grey horses in older age groups. Very characteristic lesions although some are non-pigmented either completely or in part.
- (Multicentric) lymphosarcoma: weight loss and diarrhoea are major signs. Leukaemia is rare and so lymph node biopsy is usually required.
- Eosinophilic granuloma: no systemic implications and a chronic benign course even if more develop with time. Distinctive histology.
- Eosinophilic coronitis–stomatitis syndrome: ulceration and inflammation is usually restricted to the coronary band, ergot, chestnut and oral mucosa. This may be a similar condition but lacks such severe systemic involvement.

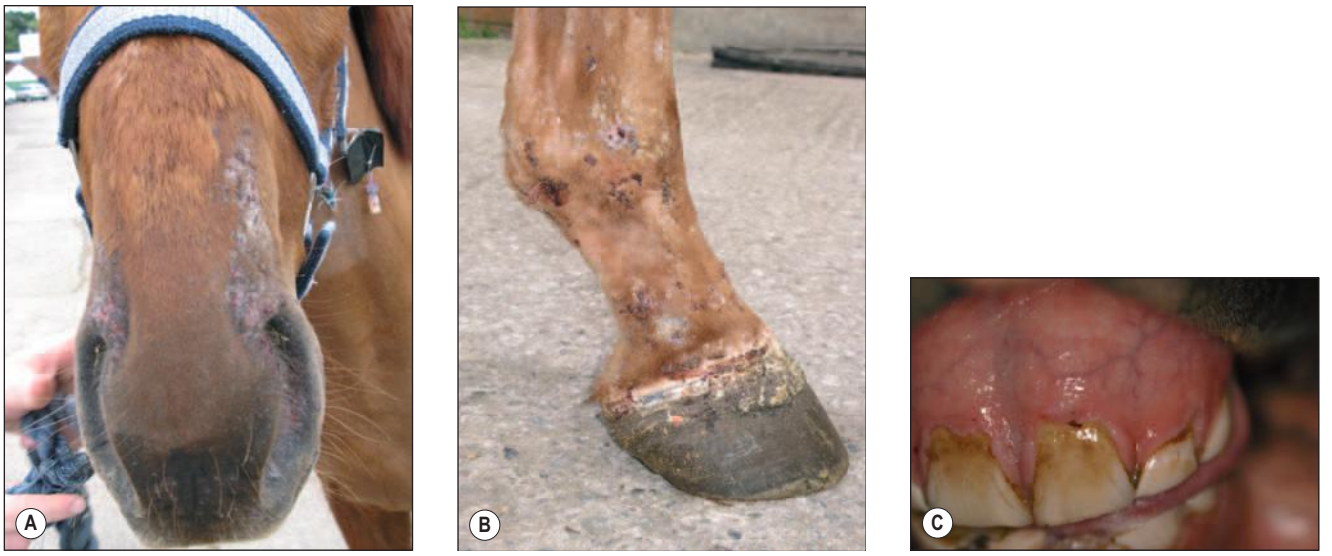
Diagnostic confirmation

- Characteristic clinical signs.
- Skin or lymph node or rectal biopsy can be supportive.
  - If the condition is suspected the pathologist should be informed so that special stains can be applied.
  - Skin biopsies reveal marked hyperkeratosis and acanthosis with a marked perivascular eosinophilic infiltration. Eosinophilic sub-corneal pustules can be a feature in some cases.
- Possible infectious or neoplastic conditions could be involved at the same time.

Treatment

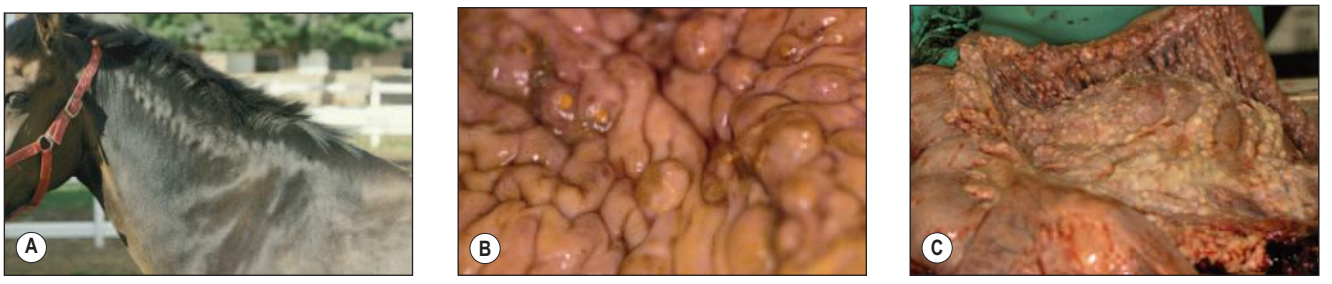
High dose (immunosuppressive) corticosteroids such as prednisolone at 2mg/kg q 12h can be helpful and if maintained for an extended period a useful remission can be achieved. Daily dexamethasone sodium phosphate at 0.2mg/kg can be used initially for 5 days before the prednisolone at 1mg/kg for 7 days orally. Thereafter it is suggested that an alternate-day dosing regimen can be used effectively (Sanford 1989). However, relapses are common if the dose falls below the effective rate and then deterioration is rapid. Reduction in any possible primary immune-stimulating process may be possible and this may help also.

The prognosis is very poor.



CD

Figure 11.35 Multisystem eosinophilic epitheliotropic disease. This 2-year-old Warmblood presented with progressive weight loss and generalized pruritus, developing a dermatitis which affected the head **(A)**, ears, legs **(B)** and ventrum (see **Fig. CD11 • 9**). The mucosa of the mouth also showed obvious (histologically pathognomonic) lesions **(C)**.



CD

Figure 11.36 **(A)** This 4-year-old pony presented with colic. Mesenteric and intestinal nodules were palpable per rectum. Diarrhoea and weight loss and a severe exfoliative dermatitis over large areas of its body, which had been progressively deteriorating over some 3 months, was noticed. It was mildly pruritic and firm, non-painful nodules and linear 'runs' were palpable in the skin. Abdominocentesis revealed a profuse volume of a turbid orange fluid laden with vast numbers of eosinophils. **(B, C)** At laparotomy the extent of the nodular eosinophilic granulomas in the mesentery, intestinal wall, peritoneum and liver was obvious. The pony was euthanized. **Figure CD11 • 9** shows further aspects of this case.

Equine erythema multiforme

Profile

This is a rare, acute, possibly self-limiting, urticarial, maculopapular or vesico-bullous/mucocutaneous dermatosis (Scott et al 1984, Scott & Miller 2003d). Although pathogenically the disease does resemble the human and dog disease with the same name, the aetiopathogenesis is very uncertain in horses. In most cases no aetiology can be established (Affolter & Von Tscharner 1993). Some cases have a more obvious syndrome with pain, pruritus and alopecia in the typical multiform dermal lesions (van Pouke et al 2005).

The complex arises secondarily to other triggering influences such as drugs (potentiated sulphonamides in particular), infections (especially herpesvirus and herpesvirus vaccinations; Fadok 1995) and tumours (particularly reticular neoplasms) (Fritsch & Elias 1993). Some cases are mistakenly identified as gyrate urticaria (and vice versa). It should be considered to be a type of allergic response, being very similar to graft-versus-host reaction, but many cases are classified as idiopathic when no underlying cause can be found. Most cases in horses are attributed to drug reactions (trimethoprim sulphur combinations and ceftiofur, in particular) (Scott 1997) but in many cases no explanation can be found (Marshall 1991).

Clinical signs

Two forms are recognized under the same name.

1. Largely asymptomatic, cutaneous, more or less symmetrically distributed skin eruptions with a characteristic 'wheal-like' lesion similar to an urticarial plaque develop rapidly from an initial, more typical urticarial lesion. Macules, vesicles or bullae can be encountered but usually the presentation is crusting and dermal patterning.
2. An acute, sometimes recurrent generalized alopecic and pruritic dermatosis with multi-form skin patterns – circular, serpiginate and polycyclic erythematous skin inflammation with exudate and erosions. Mucous membranes of mouth and nasal cavity, vulva and conjunctiva may be involved (Dunham 1979). It is likely that this form represents secondary infection of the affected skin.

In both forms lesions show a variable appearance with serpiginous or circular doughnut-like rings (Fig. 11.37). The lesions frequently persist for weeks without showing any scaling, crusting or alopecia in many horses. Some cases appear to undergo some remission of signs over some months or years, while others become progressively worse. No case can be considered to be in full remission or cured as recurrences are likely.

Differential diagnosis

- Urticaria (particularly the gyrate form): largely asymptomatic/transient or recurrent depending on challenge by allergen.
- Amyloidosis: rare, persistent stimulation of reticuloendothelial system.
- Bacterial folliculitis (saddle rash): very painful and positive cultures; typical history/circumstances.
- Dermatophilosis (rain scald/mud fever): transient and circumstantial occurrences.
- Drug eruptions/reaction: drug administration in history.
- Vasculitis (immune-mediated and infectious): localized necrotizing foci on skin and mucous membranes; oedema.
- Systemic lupus erythematosus-like syndrome: polysystemic disorder.
- Insect bite hypersensitivity (IBHS/sweet itch): self-traumatizing dermatosis restricted to defined areas of challenge.
- Pemphigus foliaceus: focal or generalized hair loss/hyperkeratosis and flake and scale production.

Diagnostic confirmation

- Characteristic self-limiting lesions over the whole body.
- Skin biopsy: hydropic interface dermatitis with migration of lymphocytes into epidermis leading to necrosis of keratinocytes. The major dermal changes include oedema of superficial dermis and presence of leaked red cells in superficial dermis with necrosis of keratinocytes.

Key points: Equine erythema multiforme

9 15 21 22 23 24

1. Rare acute immune-mediated disorder supposedly triggered by any one of many infectious and non-infectious challenges.
2. Two forms are recognized. (1) Extensive more or less well-defined circular and polycyclic erythematous skin lesions with erosions, exudate and alopecia are presented. The affected horse may be febrile and pruritic. Secondary bacterial folliculitis may be a complicating factor. (2) Asymptomatic multiform (circular, doughnut and serpiginate) dermatosis.
3. Diagnosis is made on biopsy but the changes are largely non-specific. Bacteriological cultures may be helpful with selecting the therapy.
4. Treatment with immunosuppressive doses of corticosteroids may be effective if pruritus is present, otherwise the condition is more often self-limiting. Antibiotics may be indicated.
5. Recurrences may occur but avoidance of the largely unknown trigger factors is difficult!

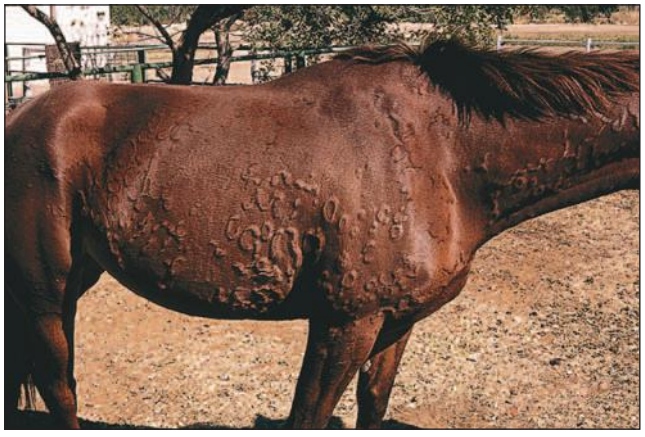


Figure 11.37 Erythema multiforme. Note the serpiginous (expanding) lesions over the chest wall in particular.

Treatment

Treatment is not usually warranted as the disease is more often self-limiting than progressive. The condition may run a benign natural course over 2–3 months or more. Systemic corticosteroids have little benefit unless in high doses but could be helpful if secondary pruritus is present.

While some cases recover spontaneously others have persistent lesions. As any pruritus is likely a secondary sign, its control is best regarded as adjunctive therapy.

Recurrence can occur at indeterminate intervals/frequency.

Equine insect bite hypersensitivity/allergy (summer itch/insect dermatitis, sweet itch, Queensland itch)

Profile

This is by far the most common skin hypersensitivity (allergy) of horses. It is widely regarded as a hypersensitivity to the bites of *Culicoides* spp. midges (Wilson et al 2001). No sex or hair/skin colour predilection is reported. Cases in foals and young horses are rare. Most horses become affected after 4–5 years of age and the condition worsens with advancing age. Certain breeds seem particularly sensitive (e.g. Shire, Icelandic and Welsh ponies), suggesting a genetic basis to the hypersensitivity. The Icelandic pony is a particularly interesting breed – in Iceland there are no *Culicoides* midges so the condition is not a clinical problem but as soon as the ponies are moved to *Culicoides* areas, they develop extreme – often highly distressing – symptoms.

The neck/tail form is the commonest presentation, and is probably the consequence of neck-and-tail-biting *Culicoides* spp. midges. The ventral abdomen form is due to ventral-biting *Culicoides* spp. midges but can be due to other insects. Hypersensitivity is commoner due to the dorsal biting insect.

Similar syndromes can be seen as a result of hypersensitivity to other biting flies (such as *Stomoxys* spp. and *Tabanus* spp.) and mosquitoes.

Note

Swarm attack by *Culicoides* spp. midges or mosquitoes also cause a primary bite irritation in all horses but not all horses develop an allergy/hypersensitivity (see p. 203). Swarm attacks can cause severe irritation in all the horses in the site whereas the hypersensitivity syndromes usually occur in one or at least a few in a group. Whilst swarm attacks can cause virtually identical signs these settle relatively quickly and usually have no long-term implications. Furthermore, there are common antigens amongst several different insect species and so midges may not always be involved even though the signs may be similar/indistinguishable.

Key points: Equine insect bite hypersensitivity/allergy



1. This condition is by far the commonest non-parasitic cause of seasonal pruritus in horses. Breed and family susceptibility and seasonality of clinical signs are characteristic. Most cases begin in the age range 3–5 years and the condition advances in each sequential year. Most cases (but not all) are due to *Culicoides* spp. hypersensitivity.
2. Mild–severe pruritus restricted to defined regions (tail-base, neck/mane and ventral midline are most often involved). Severe self-trauma results in broken hair, excoriation, exudation and skin thickening/lichenification and the development of rugae. Secondary bacterial infections can develop. Pruritus is characteristically worse when the horse is exposed to the appropriate *Culicoides* species (i.e. outside in appropriate wind-free, warm conditions). The anatomic region affected relates to the biting habits of the responsible midge.
3. Diagnosis is best derived from the history and clinical assessment. Skin testing and IgE/IgG estimation in blood are unreliable and usually unhelpful. Elimination of ectoparasites and other less common causes of pruritus should not be difficult. Response to specific avoidance strategies of management is highly supportive.
4. Avoidance of exposure through management, 'clothing' or insect repellents is the best strategy. Corticosteroids (oral prednisolone or dexamethasone) are used only in extreme circumstances when management is unable to eliminate exposure. Corticosteroids must NOT be used in place of management but rather as an adjunct to them for limited periods. Long-acting steroids are not helpful.
5. The prognosis is guarded. Prevention is the best policy but desensitizing vaccines have become available. They are generally poor with little clinical benefit – they probably do nothing that good management control will not do.

Clinical signs

Clinical signs are characteristically seasonal and gradually become worse over succeeding years; the severity and occurrence will depend heavily on exposure to the appropriate *Culicoides* spp. There is also usually a progressive deterioration over the summer months when exposure is at its greatest. The first signs may develop at any age but most cases first show some effects from 4 to 6 years.

The signs are invariably worse when the horse is outside and exposed to the midges. Midges do not tend to move indoors and do not become active in high winds or cold or very sunny, hot weather. They breed in shady, warm damp conditions such as along river courses and marshes (see p. 27) and are most active in the evening and early morning.

General signs Pruritus is always present and is worse in the early evening (dusk) and early morning, corresponding with evidence of *Culicoides* spp. midges; large numbers of midges are not required although obviously more will create more problems. Tail swishing, rubbing and restlessness are always shown. Loss of weight due to constant irritation can occur.

Acute form Pruritus with aggressive rubbing of the tail-base, neck (mane-base), head and back are typical. Papules develop along the back of the horse from ears (Figs 11.38 and 39) to tail. Self-inflicted trauma causes exfoliation, exudation of serum and patchy alopecia. Crusting and melanotrichia are common effects.

The species of midge largely dictates the location of the clinical features but most show a sparse, severely thinned tail and mane hairs that are often broken and distorted (see Fig. 11.39). Ventral-biting midges may cause a similar

response on the ventral abdomen (ventral midline dermatitis; see p. 203).

Chronic form This is characterized by variously thickened (lichenified) skin with rugae on withers, neck and tailhead in particular (Fig. 11.40 and **Fig. CD11 • 10A–D**). There is chronic hair loss and coarsening of hair coat quality in affected areas. The tail may develop a rat-tailed appearance. Although the skin recovers remarkably well once the condition is fully controlled (usually over winter months), some thickening of the skin can sometimes still be appreciated. This may have implications for veterinarians examining horses in winter months on behalf of prospective purchasers.

Acute episodes superimposed on the chronic effects are commonly encountered and of course almost all cases are affected increasingly badly in succeeding years.

Differential diagnosis

- Lice infestation (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): obvious parasites, not seasonal nor circumstantial.
- Chorioptic and/or psoroptic mange (tail forms): limbs and head mainly; mites identifiable in brushings.
- *Oxyuris equi* infestation: perineal pruritus, eggs on adhesive tape preparations, response to deworming.
- Fly bites (*Stomoxys* spp., *Simulium* spp., *Haematobia exigua*) : mass worry/bite by any insect can be very similar but this is not persistent and resolves once the swarm effect has subsided.
- 'Swarm worry'/multiple bites by mosquitoes and *Culicoides* midges, flies and other insects: exposure circumstances and self-resolving when past.
- Dermatophilosis (*Dermatophilus congolensis*): not (obviously) pruritic.
- Dermatophytosis (*Microsporum gypseum*): mildly pruritic associated with characteristic lesions, self-limiting and not resolvable simply by management avoidance of being outside – often better in sunlight.
- Onchocercal dermatitis (*Onchocerca cervicalis*): face and neck most often, mildly pruritic only. Focal lesions with alopecia, loss of pigmentation, epidermal hyperplasia and hyperkeratosis. For many years this has been the main differential but it is very rare and hardly similar at all!
- Unilateral papular dermatosis: seldom pruritic.
- 'Stickfast' flea, spinose ear tick, cattle tick larvae, bee stings: historical exposure, single events – not pruritic.
- Chemical irritation: history of exposure and signs restricted to location.
- Besnoitiosis: very rare; little pruritus and chronic/persistent nature.
- Mane and tail (follicular) dystrophy: not pruritic, not seasonal; non-symptomatic loss of mane and tail hairs.
- Anhidrosis: not pruritic; sporadic with characteristic history of significant move to warm humid climate.

CD



Figure 11.38 Acute insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch/Queensland itch) showing papules on the ears and crest under the mane.



Figure 11.39 Acute insect bite hypersensitivity (*Culicoides* hypersensitivity/sweet itch/Queensland itch) showing papules along the upper part of the neck and sparse, broken and damaged mane hairs.

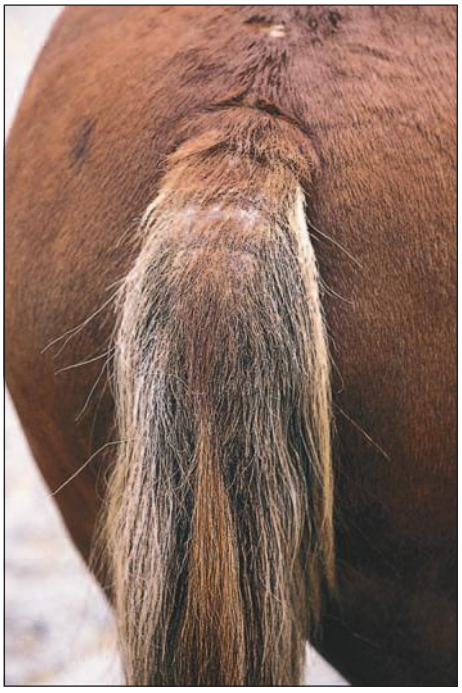


Figure 11.40 Chronic insect bite hypersensitivity (sweet itch/Queensland itch) showing the result of chronic, repeated tail-rubbing resulting in hair loss and skin thickening over the tail-base. This photograph shows the early recovery stage occurring in the winter months.

Diagnostic confirmation

- Characteristic clinical signs are well recognized but there are some difficulties with confirmation.
- Seasonality (summer months and improving in winter or colder periods).
- Elimination of all other ectoparasitic possibilities.
- Biopsy shows mild to severe eosinophilic folliculitis and dermatitis (but is usually unsatisfactory due to secondary changes and non-specific pathological responses).
- Intradermal skin testing using aqueous whole-insect antigen is available. Problems with antigen specificity, sensitivity and reliability have been identified. There is little correlation and repeatability between the skin tests, the serum ELISA and the histological responses seen at the site. Currently the tests have no convincing value (Sloet van Oldruitenborgh-Oosterbaan 2004).
- Identification of biting insects is important as not all *Culicoides* bite dorsal areas; some species attack ventral areas only and cause ventral dermatitis. Not all hypersensitivity is due to *Culicoides* spp. alone and it can be complicated by sensitivity to other flies, e.g. *Tabanus* spp. (horse flies), *Stomoxys* spp. (stable flies) and *Haematobia* spp. (buffalo flies).

Treatment

There are two main goals in the management of insect bite hypersensitivity cases:

1 prevention of contact with the insects
2 limiting the clinical severity of the response.

The only effective 'treatment' is avoidance of exposure. Management strategies must reflect total avoidance if the condition is to be controlled; even a single or few bites may set up a severe response.

Fly repellents (see p. 101) Treatment of unrugged, grazing horses is extremely difficult but application of light-oil dressing to the dorsum of the horse gives some relief. Success has been claimed with 0.5% fenvalerate (200 g/L) at the rate of 200 mL per horse to the horse's backline, or alternatively 0.1% fenvalerate (200 g/L) is applied as a backline and body spray at the rate of 1 litre per horse every 7 days. Up to four applications are claimed to be curative, with hair regrowth and cessation of pruritus (Dunham 1979). Insect repellents, such as dibutyl phthalate and benzyl benzoate, applied to the rugs and/or the head, back and base of the tail of individual horses have been marginally effective in some cases. Permethrin fly repellents are sometimes useful.

Fly traps using octanol and carbon dioxide can significantly reduce the numbers of bites an animal receives but even when the traps collect millions of midges, this has very limited effects on their overall population. Smudge fires built upwind from the horses and removal of midge breeding grounds may be feasible options in some circumstances.

Exposure management The most important control measure is the protection of the horse against further contact with *Culicoides*. Unless this can be accomplished, all other measures are likely to be unsuccessful. Stabling in a protected environment between dawn and mid-morning and mid-afternoon and dusk combined with clothing/rugging with sheets and hoods may prevent serious skin damage. However, *Culicoides* species differ in their habits and so this may not help in every case.

Insect-proof stables and working environment are certainly the ultimate methods of prevention, but they are rarely achievable; a very fine screen mesh must be used even though *Culicoides* midges do not move into stables. Insecticidal spays applied to the meshes over windows and doors is a useful adjunct.

Culicoides spp. midges seem less able to cope with air speeds over 10 km/h so moving the affected horse to a windy area away from river courses, woodlands and standing water is sometimes effective. Placing a fan overhead in the stable with a strong down and outward air flow is very effective and most affected horses will actively seek out the air flow. Individual animals may be particularly sensitive to individual species of *Culicoides* and so may be moved away from the area.

Reduction of clinical severity Daily oral prednisolone during the high-risk season can reduce pruritus to a minimum, but secondary effects from prolonged corticosteroid medication must be constantly considered.

For longer-lasting effect in seriously affected horses, methylprednisolone acetate or betamethasone can be administered intramuscularly at 3–4-week (or sometimes lesser) intervals. Prolonged usage is not advisable as they have significant but small risks. Although long-term administration of depot forms of methylprednisolone is often used in practice, the pharmacokinetics suggest that it is not a logical method and the benefits are much more likely to arise from concurrent avoidance strategies.

Individual horses can be treated in the short term with daily antihistamines such as chlorpheniramine or hydroxyzine or even phenothiazine drugs which have antihistamine effects and sedation effects. However, results are very disappointing.

Desensitization is also an option but so far has failed to improve many cases. Desensitization relies upon repeated challenges with the proven antigen using repeated intramuscular injections. The manufacturers are at pains to point out that an 'acceptable' result is a 50% reduction in signs with a 50% reduction in drug use provided that all other avoidance strategies are followed. Currently there is no proper evidence for efficacy (Barbet et al 1990) but the approach does have a theoretical attraction.

Several homeopathic methods and 'magic' treatment of serum or skin/hair are regrettably widely practised but the advice that comes with these approaches includes the clear necessity to avoid contact so that the benefit from these expensive approaches is negligible at best.

Owners are far better advised honestly about the need to prevent contact with the *Culicoides* flies.

Contact hypersensitivity

Profile

Specific and proven contact hypersensitivities are very rare in horses and have not been subjected to any evidence-based trials.

In horses it may be clinically difficult to separate contact allergic responses from primary irritant contact dermatitis

Key points: Contact hypersensitivity

11 14 17 18 21 24

1. Rare, variable and poorly defined set of cutaneous responses to contact with various chemicals, substances and vegetations. A type 4 hypersensitivity reaction is the likely pathogenesis.
2. Clinical signs are usually restricted to the contact areas. Acute onset of a florid inflammation with variable pruritus are the main signs. Secondary bacterial infection may complicate the signs.
3. Diagnosis can be made by supposition based on the history of contact with some unusual material. Removal from the source results in rapid recovery and re-exposure causes recurrence. Where the causative agent is not known to be irritant and the response is severe, hypersensitivity can be suggested.
4. Treatment of contact allergies relies totally on avoidance. Short-term therapy with corticosteroids (either systemically, topically or both) can be used initially.
5. Genuine hypersensitivities are usually repeatable by repeat exposure and this may remain a life-long problem.

responses (Rietschel 1997). However, soaps, blankets and rugs, bedding, feed components, medication, insect repellents and pasture plants have been implicated with a type 4 hypersensitivity reaction.

Strong circumstantial evidence is usually attainable with patience and care.

Clinical signs

An acute onset of a florid, localized inflammatory response with transient vesicles and papules over the contact area (seldom generalized) is likely (Fig. 11.41). Contact areas of suspect material show greatest reaction.

Vesicles rupture leaving an erythematous oozing and crusting on the skin surface. There is variable pruritus but little pain and secondary bacterial or fungal infections can occur.

Differential diagnosis

- Ectoparasites: pruritus related to parasite distribution and signs largely restricted to local urticaria. The parasites may be detected.
- Insect bite hypersensitivity (including sweet itch): regionally restricted to typical bite areas. Exposure related and seasonal.
- Pastern folliculitis: localized inflammation, more painful than pruritic.
- Other hypersensitivity (including food): urticarial lesions are more likely.
- Drug eruptions: generalized mild pruritus with alopecia and exfoliative seborrhoeic dermatosis.

Diagnostic confirmation

- Careful historical questioning usually identifies something that is 'new' or unusual.
- Removal of all suspected material usually results in a rapid resolution but full normality may take up to 7–10 days. Re-exposure can be tried but owners are often reluctant to allow this.





Figure 11.41 (A) This 7-year-old gelding suffered from an acute onset, mildly pruritic contact allergy to the pollen from *Ranunculus* spp. plants (buttercups) on the muzzle and pastern regions. The condition could be repeatedly induced by allowing exposure for less than 5 minutes, although the signs only developed some 72 hours later **(B)**. The horse was not allowed to graze when these plants were in flower but otherwise had no problems at all. Simply removing the contact resulted in significant improvement within 48 hours without any drugs.

- Patch testing involving the application of suspected substance(s) placed on cloth and fastened to the skin with adhesive tape (beware of possible reaction to the tape itself) for 48 hours. This is difficult owing to the difficulty of adhesion to skin, but a marked reaction is strongly suggestive. Using a blank as a negative control is a wise precaution.
- Secondary bacterial or fungal infections can confuse the clinical progression and investigation.

Treatment

The affected area should be washed carefully to remove any residual irritant/allergen and the horse moved to a different environment away from the suggested 'allergen'.

Note

Avoidance of contact is not an alternative to corticosteroid therapy.

Corticosteroids should only be used when there is no possible avoidance stratagem. It should be restricted to the first day or two after which the condition should resolve. However, the full remission of signs may take up to 14 days or more. Suitable long-term avoidance strategies should be discussed with the owner.

Drug eruption (cutaneous adverse drug reaction)

Profile

Drug eruptions are regularly referred to in the literature but are consistently difficult to both prove and to investigate; dermatologists agree that proving the cause and effect is an enormous challenge.

Delayed drug reactions may not easily be attributable to the drug. No drug is more or less liable to induce the changes. It is usually accepted that drugs, including most antibiotics (especially trimethoprim sulphur combinations and penicillin), anthelmintics, non-steroidal anti-inflammatories, sedatives and tranquillizers, topical applications, and many vaccines, given by any route, may cause significant skin eruptions. The reaction to a drug can be delayed for weeks or months and supposedly produces a type 4 hypersensitivity reaction.

A few drugs induce an immediate hypersensitivity reaction. For example, penicillin and some non-steroidal anti-inflammatory drugs sometimes cause urticaria but this is not really a drug eruption.

The vague nature of the historical exposure, the variable time scale to the appearance of signs and the disparate range of unrelated drugs that are supposedly involved mean that this is often a diagnosis of last resort.

Key points: Drug eruption

10 11 12 15 17 18 21 24

1. An unusual, ill-defined disorder that is putatively linked to exposure to a drug of any sort over an ill-defined period.
2. The signs are totally non-specific and can mimic almost any other infectious or non-infectious dermatosis. The signs range from urticaria and mild pruritus to severe erosive bullous disease. Alopecia, seborrhoea and crusting and possibly focal vasculitis-like lesions can be seen. The commonest signs are generalized exfoliative dermatitis and alopecia with easily depilated hair.
3. There are no pathognomonic histological signs and no tests that can be reliably applied to investigate suspected cases.
4. Where these reactions are suspected, the retrospective information regarding exposure is often unhelpful. Advice to avoid any drugs at all is also not helpful.
5. The prognosis is fair – many cases gradually recover, sometimes over months or years.

Clinical signs

The commonest signs include exfoliative dermatitis and scaling with alopecia and easily depilated hair over wide areas of the body (Fig. 11.42). Non-specific skin eruption(s) may show variable urticarial plaques, papules and/or generalized pruritus. Mucocutaneous junctions may exhibit the earliest signs including vesicles, ulceration and scaling and so a history of initial involvement of these sites may be suggestive of a drug eruption. Long delays have no material influence on the severity of clinical signs but clearly make diagnosis more difficult.

Differential diagnosis

- Urticaria (particularly the gyrate or oozing forms): transient typical wheal lesions with rapid resolution following single dose of corticosteroid. More difficulty





Figure 11.42 (A, B) This pony developed an extensive exfoliative dermatitis with alopecia 4 weeks after receiving an avermectin wormer. It was very difficult to ascribe the signs to that event but no other explanation could be identified. The hair grew back slowly and after a year the pony looked normal. An identical response occurred 14 months later when it received another dose inadvertently. It could be classified as an adverse drug response.

with some generalized angio-oedema states but these tend to be more exudative, and dependent filling oedema is common.
- Systemic lupus erythematosus-like syndrome: very rare multisystem disorders with internal lymphadenopathy in some but not all cases, uveitic changes and secondary cutaneous infections. Moderate exfoliation, focal or extensive alopecic areas and some cutaneous depigmentation.
- Food allergy: exposure-related, mildly pruritic, urticarial condition.
- Drug allergy: post-administration urticarial reactions – usually transient and definitely exposure related.
- Erythema multiforme: serpiginate or circular erythematous intradermal lesions – often chronic and sometimes exudative and alopecic but only related to lesion locations.
- Insect bite hypersensitivity (*Culicoides* spp./sweet itch): moderate to severe, seasonally and environmentally related pruritus. Sites reflect the bite locations of specific species of insect.
- Pemphigus foliaceus: localized or generalized exfoliative dermatosis with alopecia, flake and scale. Definitive biopsy findings.
- Bullous pemphigoid/pemphigus vulgaris: severe vesicobullous disease particularly affecting the mucocutaneous junctions; characteristic histopathology.

Diagnostic confirmation

- Very difficult unless the onset occurs immediately after the drug has been given. There are probably no pathognomonic signs associated with drug eruptions and any unexplained cutaneous lesions could justifiably be considered as a 'drug eruption'. There is always a history of drug administration (even ivermectin anthelmintics have been implicated in the aetiology of variegated hyperaesthetic leukotrichia). Confirmed diagnosis of acute cases can be made if the drug(s) is withdrawn and signs disappear, and then reappear with reintroduction of the suspect drug. Even this may only provide a presumptive diagnosis since the drug is usually administered because the animal has an illness. Carriers and preservatives in drugs can also be responsible, so changing the specific brand may help.

Treatment

Discontinue suspected drugs immediately if any are being administered. It may be possible to change the specific drugs being used if the reason for its use is significant. If a severe, acute reaction is present, high doses of systemic corticosteroids can be administered. However, the response may be poor.

Since so little is known and since the signs and the pathogenesis are vague, the prognosis should be guarded at best, but many do recover with time.

Lymphoedema

Profile

Diffuse oedematous swelling of the limbs and the preputial area may be primary due to a lymphatic system disorder or may be secondary to injury, infection or neoplasia. Significant oedema can also arise from lack of natural movement of one or more limbs or general body movement. The condition is best regarded as a sign of disease rather than a diagnosis in itself so investigation of a primary problem should always be undertaken.

Key points: Lymphoedema

1. Common secondary finding in many local inflammatory or circulatory obstructive lesions. Also arises in conditions associated with hypoalbuminaemia. Lack of movement is a common cause of milder accumulations of oedema in dependent parts.
2. The area 'pits on pressure' when it is due to oedema as opposed to inflammatory states. Oedema may run forwards to the sternal area from sheath or scrotal oedema or inflammation.
3. Diagnosis should be directed at the primary cause. Physiological extravascular fluid accumulation as a result of lack of movement will resolve quickly on even limited exercise.
4. Treatment will depend on the cause. Diuretics are not usually helpful.
5. Prognosis depends on the primary disorder.

Clinical signs

Primary form Localized painless nodules and enlarged lymphatics commonly involving the prepuce which show little 'pitting on pressure'. This condition is often of concern to owners and many cases are notably fat. This may mean that circulatory obstructions are in fact involved.

Secondary form Severe accumulation of interstitial fluid – often associated with vascular protein leakage into the tissues. It is often painful and there is obvious pitting on pressure (Fig. 11.43).

Differential diagnosis

- Oedema due to lack of movement (for whatever reason): history is usually obvious.
- Causes of lymphatic obstruction (haemangioma, lymphangioma, lymphosarcoma, other tumours or inflammatory lymphadenopathy, etc.): evidence is usually found and the distribution of oedema is an indicator of the probable location for the obstruction.
- Low blood protein:
  - primary due to starvation: rare and easily identified from basic haematology
  - secondary due to protein loss (via skin, gut or kidney), failure of absorption, hepatic failure or physiological or pathological consumption of protein: basic haematology plus specific organ profiles are helpful.
- Injury, post-castration swelling (preputial and scrotal forms): signs and history are obvious.
- Lymphangitis and cellulitis: both are inflammatory and will not pit on pressure to the same extent; pain and heat are usually present.
- Preputial fat accumulation (possibly as part of metabolic syndrome): other signs and no pitting on pressure; some discomfort can be present.

- Infection including 'scirrhous cord': staphylococcal granuloma of the spermatic cord and scrotum.

Diagnostic confirmation

There is characteristic lymphatic/oedematous disorder of prepuce, inguinal region and limbs in particular.

Treatment

Treatment options vary with primary cause. Diuretics are not usually helpful and should be avoided if at all possible. Any interference with oedematous skin may result in poor healing.

Cold irrigation/compresses and movement are the best options.

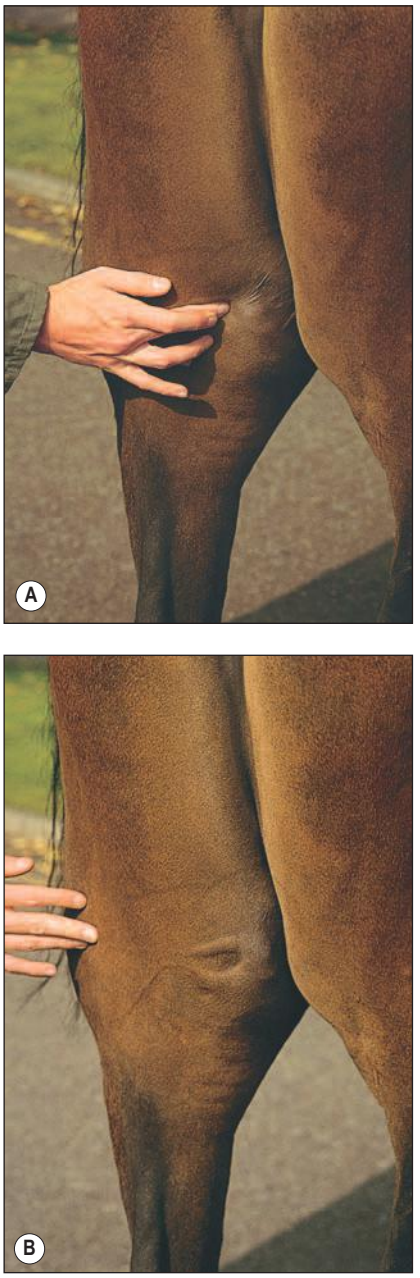


Figure 11.43 (A, B) Lymphoedema. The oedema is diffuse and showed no evidence of inflammation. The pictures illustrate the 'pitting-on-pressure' test for oedema of any kind.

CD

Sterile, nodular panniculitis/steatitis

Profile

This is a rare, multifactorial inflammatory condition of the subcutaneous fat of horses.

The aetiology is obscure but a complex of vitamin E and selenium deficiency has been suspected in some cases and particularly in foals and to a lesser extent adults of the Fell pony breed.

A particular form occurs on the brisket possibly as result of traumatic damage to the brisket fat pad.

Clinical signs

Young foals develop panniculitis as part of a selenium/vitamin E deficiency syndrome – the skin signs are relatively trivial. Muscular weakness, stiffness, recumbency and sometimes myoglobinuria can be found. Nodules can be found mainly in the upper crest area associated with the funicular portion of the nuchal ligament.

The adult horse nodular form is the most significant from a dermatological point of view. Multiple, variably sized deep-seated nodules are found in the subcutis, particularly in the upper neck where they are associated with the nuchal ligament, the lateral thoracic wall and the dorsum (Fig. 11.44). In time, the subcutaneous nature is lost and the nodules become fixed to the skin. Nodules may or may not be painful on palpation (Scott 1988) and may be firm or soft. In some there is exudate and ulceration. Some nodules may be large and may be palpable from both sides of the neck **(Fig. CD11 • 11A and B)**. Cystic lesions lead to ulceration and draining tracts which discharge an oily yellow/brown or bloody discharge. Healing lesions have a fibrotic scar and may remain painful on palpation.

Key points: Sterile nodular panniculitis/steatitis

9 15 17 21 23 24

1. Rare, idiopathic disorder affecting the subcutaneous fat resulting in sterile inflammation and nodule formation. Two forms are recognized. One affects young foals and may be associated with vitamin E/selenium status and may be related to the white muscle disease complex. A second form occurs in adults and is less well defined.
2. Plaque-like swellings of the skin and subcutis over focal or more general areas are typical. The dorsum and upper neck are commonly involved. Concurrent, undulant fever, depression, anorexia and some stiffness/movement restrictions may occur. The adult form shows limited numbers of fluctuating nodules that are painful on palpation. A few ulcerate and discharge a brown–yellow fatty exudate.
3. Diagnosis is difficult – biopsy usually fails to harvest meaningful tissues but neutrophils, giant cells and macrophages can be found – degenerative fat is usually the most obvious finding. Ultrasonographic examination and biochemical testing is useful. Excisional biopsy of an intact nodule is the best approach.
4. Treatment is directed at the suggested cause. In foals this involves systemic and dietary vitamin E and selenium. However, these are seldom helpful. Oral corticosteroids may help in some cases.
5. The prognosis is fair but in the nodular form scarring and some residual pain are common.

Concurrent systemic effects include diarrhoea, anaemia, weakness, variable lameness and failure to thrive in young foals. Where selenium and vitamin E deficiencies are involved there may be concurrent muscular signs (as in the foal syndromes) and some immunocompromise with secondary skin, enteric and respiratory infections.

Differential diagnosis

- Collagenolytic granulomas: these remain firmly within the skin itself and seldom cause any sign apart from a mild discomfort in the early stages – the lesions commonly have a small external pore with a crust. They can become calcified.
- Neoplasia including cutaneous (histiocytic) lymphosarcoma and lymphoma: definitive clinical signs and biopsy.
- Cysts: usually distinctive, chronic and all have definitive histology.

Diagnostic confirmation

- History.
- Clinical examination.
- High-frequency ultrasonography can confirm the extent of the nodules but the echo features are not pathognomonic.
- Wedge biopsy, or excisional biopsy in the case of smaller, more accessible nodules, is helpful. The biopsy itself may be strongly suggestive of the condition as the overlying skin is usually normal and the nodule has a dense texture and a brownish colour. Although it is stated that excisional biopsy is the only method that is suitable (Scott & Miller 2003e), this is far from practical in all circumstances. However, small punch biopsies and fine needle aspiration are seldom diagnostic – a large, deep wedge biopsy may be far better (see **Fig. CD11 • 11B**).

Treatment

Surgical excision of isolated and convenient nodules such as those on the chest walls and breast can be curative. In some cases the extent and location preclude excision. Good results have also been reported with high levels of systemic prednisolone or dexamethasone, but overall results are variable (Karcher et al 1990). Correction of vitamin E/selenium status is helpful but may be disappointing. It is important to minimize trauma to the affected areas so repeated palpation and heavy grooming should be avoided as far as possible.

Long-term scarring at sites of particularly bad damage is common and sometimes these sites remain painful.



Figure 11.44 This 7-year-old pony developed a series of interlinking painful plaques along the dorsum and upper neck. Excisional biopsy confirmed nodular panniculitis.

Axillary nodular necrosis

Profile

Rare, nodular skin condition of unknown aetiology. Girth trauma has been suggested as a possible cause but the nodules also occur spontaneously in non-working (and even wild) horses. No breed, sex or age predisposition is recognized.

The focal nature of the changes and the histological changes, which involve a definite arteritis/vasculopathy, suggest that there may be some immunological aspects to it.

Clinical signs

One or two (occasionally many) firm, painless, well-circumscribed subcutaneous nodules (10–40 mm in diameter) near the girth or axilla (Fig. 11.45) appear. Chains of interlinking nodules may be detected. No pruritus or pain is associated with the nodules. Most show no overlying hair loss but in the largest nodules hair may be rubbed away from the surface. Some nodules may ulcerate but these are probably a different condition.

The nodules are of no material consequence but may resemble some other more significant diseases. Affected horses are otherwise healthy.

Differential diagnosis

CD

- Traumatic damage/scarring: history of wounding and invariably some evidence of scar formation.
- Mastocytoma: rare and definitive histology – seldom found in the girth area.
- Epidermoid and dermoid cysts: not found in the girth region.

Key points: Axillary nodular necrosis

9 14 20 23 24

1. Rare, sporadic nodular skin disorder affecting the axillary skin. Some similarities to the eosinophilic dermatitis with collagenolytic granuloma disorder.
2. Single or few localized firm, roughly spherical nodules varying in size from 2 to 10 cm diameter are found in the skin of the axilla and girth regions ventrally and behind the elbow. Lesions are painless and have no clinically obvious inflammatory nature.
3. Diagnosis requires biopsy – usually excisional biopsy is used. The changes are ill defined but eosinophilic granulomatous inflammation is detected histologically.
4. Surgical excision can be curative but the lesions themselves usually cause no problem so benign neglect is a justifiable option. The problem is in the differential diagnosis with the type A (1 and 2) nodular sarcoid in particular.
5. The prognosis is excellent with or without treatment. Lesions may regress over years but many remain static. Biopsy does not result in exacerbation.

- (Eosinophilic) collagen necrosis: some cases are similar but most cases do not involve the girth region; most affect the dorsum, neck and body trunk.
- Nodular sarcoid: very similar in some forms but definitive fibroblastic nature on biopsy or excisional biopsy.
- Lymphosarcoma/lymphoma: very rare and occasionally found in deep axillary tissues; definitive histology.
- Insect bites: can be similar clinically, especially if the animal has had a marked reaction to the bites; history of acute onset of painful or pruritic lesion.
- Hypodermiasis (*Hypoderma bovis*): extremely rare and most affect the dorsum.
- Amyloidosis: rare condition with definitive histology that seldom affects the axilla.
- Nodular panniculitis/steatitis: painful inflammatory changes in the panniculus or subcutaneous fat – quite commonly affects the girth and lower chest walls where girth trauma may be involved.



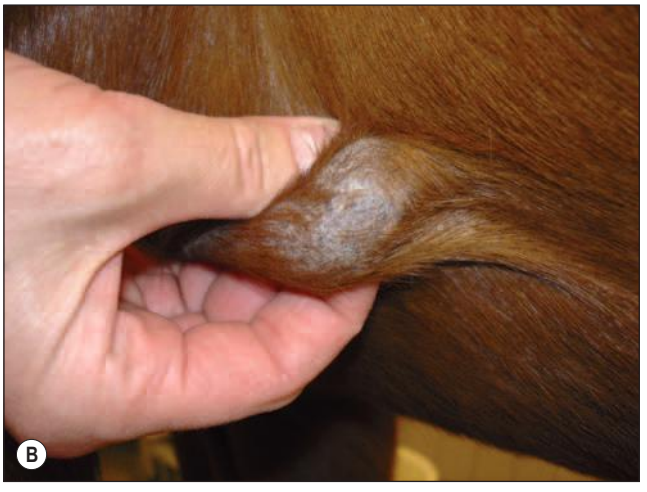


Figure 11.45 (A) Axillary nodular necrosis. A single, non-painful solid nodule in the typical position for axillary nodular necrosis. **(B)** The lesion had no palpable root. This example is somewhat larger than is usual and hair loss has occurred – probably from rubbing on the girth, Surgical removal was uneventful.

- Fungal/bacterial granuloma and foreign body reactions: history and usually painful, at least at first.

Diagnostic confirmation

- Clinical signs are almost characteristic but the condition is very rare.
- Biopsy is distinctive (focal dermal necrosis without collagen degeneration).
- Cultures for bacteria and fungi are negative.

Treatment

Most cases undergo spontaneous resolution over some months but a few are persistent. Benign neglect can be justified. Surgical removal is possible and usually successful. Sub-lesional injection of small doses of methylprednisolone (5–10 mg/lesion) may also be effective (Mathison 1995).

Eosinophilic dermatitis with collagen necrosis/eosinophilic granuloma/collagenolytic granuloma

Profile

This is probably the most common non-neoplastic nodular skin condition in the horse. It is characterized by isolated single or multiple, or groups of isolated firm dermal nodules associated with eosinophilic granulomatous necrosis and degenerative collagen. The role of collagen degeneration is controversial. It may be a product of the pathology or it may be part of the primary process.

Key points: Eosinophilic dermatitis with collagen necrosis/eosinophilic granuloma/collagenolytic granuloma

1. Very common, rather benign nodular disorder of the skin of horses worldwide. Most develop in summer so insects are suspected of being involved. Usually associated with an eosinophilic granuloma with localized dermal collagen degeneration and mineralization.
2. Variable numbers (few to many) of small, firm, non-painful dermal nodules are palpable in the skin with a few showing erect hairs – otherwise better felt than seen especially in winter-coated horses. Early lesions may be mildly inflamed and often are slightly larger than the later ones and these may be mildly painful on palpation. Close examination shows a small crusting surface overlying a 'pore-like' opening with caseous/granular or gritty material in the nodule.
3. Diagnosis is easy and biopsy is diagnostic.
4. The lesions are probably best left alone. Surgical excision is a simple procedure but does not prevent more lesions from developing. Intralesional triamcinolone may help early lesions but may increase calcification. Oral prednisolone may be helpful in early cases if many lesions are developing or if they are larger than normal.
5. The prognosis is excellent – many will settle with time but some remain as hard nodules. A few are large enough to cause long-term problems.

The eosinophilic nature has been used as a reason to propose the cause as insect bites (Mathison 1995, Yu 2006). To support this hypothesis most cases develop in summer and early autumn. Other cases begin in winter months. Remarkably for an insect aetiology, the lesions almost never occur on glabrous skin of the medial thigh and groin regions. It is possibly the result of local hypersensitivity reactions to insect bites. However, even early lesions seldom show any visible or histological evidence of an insect bite. This may be a result of a slowly developing lesion – the earliest stages are seldom recognized. Furthermore many have no possible insect contact history.

Clinical signs

There are one or more (occasionally many) firm, painless, well-circumscribed subcutaneous nodules 5–20 mm in diameter, usually but not always situated along the back, buttock, neck and sides of the chest (Fig. 11.46). Chains and groups can occur, suggesting an insect-related aetiology in some cases. In some cases the distribution is widespread (Fig. 11.47). The lesions are better felt than seen but some have erect hairs that may prove a cosmetic problem.



Figure 11.46 Eosinophilic collagen necrosis/granuloma lesions on the buttock region. Note the slightly erect hairs that make the nodules more visible.



Figure 11.47 Large numbers of isolated eosinophilic collagen necrosis/granuloma lesions. The horse has been clipped out to demonstrate the extent and random distribution in this case.

There is probably a 'typical form' but there are plenty of variations on this. In some cases the granulomas can be large (even up to 3–5 cm in diameter; Fig. 11.48) and they can occur at unusual sites including the limb skin, mouth and eyelid **(Fig. CD11 • 12A and B)**.

Early lesions may be sensitive to palpation and slightly inflamed with a less defined margin. The more mature lesions have a calcified, granular core which can be expressed from the open surface (Fig. 11.49).

The condition is not usually pruritic and there is no overlying alopecia except at the centre of a degenerating lesion.

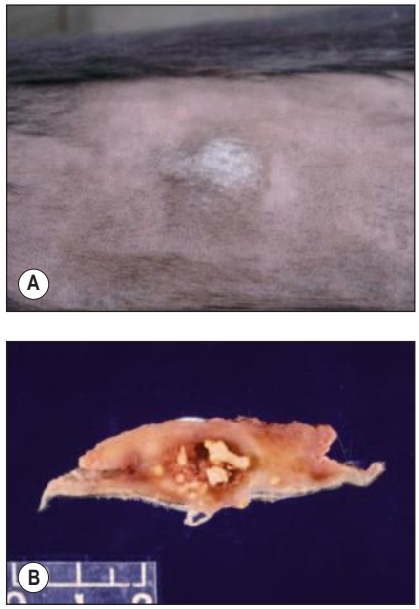


Figure 11.48 **(A)** This single, larger dermal nodule was identified on the back of a young horse. **(B)** The gross appearance of a similar lesion following surgical excision. It illustrates the overall nodular and degenerative nature of the condition.

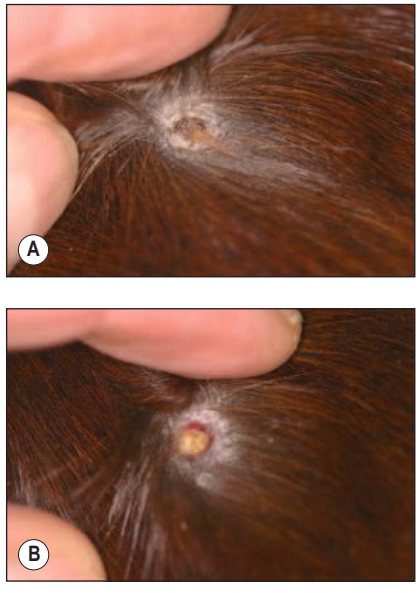


Figure 11.49 **(A)** Close-up picture of a single eosinophilic collagen necrosis/granuloma lesion showing the circular crusting **(B)**. Removal of this crust reveals a calcified core of amorphous material. This can be 'picked out' with a needle or a blunt probe.

Differential diagnosis

- Axillary nodular necrosis: a single or few intracutaneous or subcutaneous, non-painful nodules located in the axilla/girth regions; histologically they can be similar.
- Epidermoid and dermoid cysts: larger, midline, slowly expanding spherical cysts containing hair and sebum.
- Insect bites: transient, acute and obviously inflammatory with a central purple spot; insect bites may be a cause of some collagenolytic granuloma lesions.
- Hypodermiasis (*Hypoderma bovis*): very rare, single, large, painful and expanding nodules usually restricted to the dorsal midline in the withers region; ultimately they may develop a viable moving larva and a breathing pore.
- Cutaneous lymphosarcoma: larger, solid lesions with typical histology; concurrent systemic signs.
- Sarcoid (nodular): usually larger, expanding nodules and often with cutaneous involvement. Concurrent other sarcoid types elsewhere or adjacent lesions at the site.
- Amyloidosis: single plaque-like cutaneous and subcutaneous swellings/nodule associated in some (but not all) cases with immune stimulation.
- Phaeohyphomycosis.
- Panniculitis: subcutaneous softer swelling with an ill defined margin and pain on palpation. Not really similar at all!

Diagnostic confirmation

- Clinical signs are almost characteristic.
- Biopsy is distinctive (foci of necrobiosis of collagen fibres associated with heavy eosinophilic infiltration). Older lesions may mineralize.

Treatment

The condition is sufficiently benign to warrant no treatment in most cases. The large majority will gradually settle over 3–12 months, becoming progressively less obvious and less palpable, but some remain static for years.

A few early lesions may be painful but other than those the condition is of no concern to the horse. Treatment then becomes a matter of cosmesis.

Medical therapy Where there are large numbers of early lesions systemic corticosteroids can be helpful, at least in limiting the numbers and the ultimate size of the lesions. Oral prednisolone or dexamethasone at standard anti-inflammatory rates is used (0.5 mg/kg and 0.05–0.1 mg/kg, respectively). Intralesional triamcinolone can be helpful (1–2 mg/lesion using a dose-limiting pressure syringe to ensure intralesional deposition). Intralesional methylprednisolone acetate is probably contraindicated because in many cases it does not help at all and in others simply increases the persistence and extent of calcification.

Many nodules are dense and may require the use of a (Luer-lock) pressure syringe and a 25G needle. Systemic corticosteroids (prednisolone) can be helpful if many lesions are present, especially in the early stages.

Surgical treatment Surgical removal is possible and usually successful, especially in older calcified lesions. However, some occur in difficult sites where surgery is difficult or may lead to behavioural problems (e.g. saddle area). Surgical excision can be performed easily in most cases using an 8 mm biopsy punch. Standard approaches to skin biopsy can also be used to treat collagenolytic granulomas. Large numbers may require general anaesthesia but otherwise small blebs of local anaesthetic can be effective. Where a biopsy punch ≤ 8 mm is used there is no need to suture the resulting wound.

Primary seborrhoea

Profile

Primary sebaceous gland dysfunction is an extremely rare congenital disorder in horses (see p. 232). The disease seems more likely to be related to abnormal cornification resulting in scaling and crusting than to sebaceous gland dysfunction. Secondary seborrhoea is usually related to a skin response from some other injury, e.g. 'greasy heel' in heavy horses (Thomsett 1984).

A similar clinical appearance can be caused by secondary skin irritants (topical chemical or chronic urine scalding) or *Chorioptes equi* (primary dermatitis of limbs of heavy horses) and/or dermatophilosis (*Dermatophilus congolensis*).

Clinical signs

A matted, greasy hair coat with moderate to heavy scaling of mane, tail, limbs and occasionally body skin is detected. Often the 'greasiness' can be best detected by squeezing the skin debris between the fingers (Fig. 11.50). Patchy alopecia and hyperpigmentation (melanoderma) may occur. Pruritus and pain are absent except when the condition arises secondarily to a painful or pruritic condition. Seborrhoea can be caused by pruritus also.

Note

Many such cases encountered in adult horses are not primary seborrhoea but the greasiness develops secondary to other skin conditions including parasitic infestations, *Dermatophilus congolensis* infection and some more generalized conditions affecting the skin such as pemphigus foliaceus and systemic lupus erythematosus-like syndrome.

Key points: Primary seborrhoea



1. This is a very rare, probably congenital, disorder of the skin. The large majority of seborrhoeic horses have secondary seborrhoea associated with other diseases.
2. Clinical signs are predominately scaling and flaking dermatosis but the coat may be excessively greasy and may have an unpleasant odour. Secondary skin infections are common.
3. Diagnosis is largely by its early age of onset and exclusion of all alternative causes of the seborrhoea.
4. Treatment of primary seborrhoea is not possible. Reduction in coat sebum can be achieved by avoiding dietary fats and by regular washing of the skin with degreasing shampoos.
5. The prognosis is poor for recovery but most cases survive into old age with proper care.

Differential diagnosis

- Pemphigus foliaceus: rare, sporadic bullous disease with localized or generalized alopecia and heavy scaling. Typical histology.
- Other autoimmune complexes including systemic lupus erythematosus-like syndrome: generalized scaling and crusting with variable alopecia and pigmentary changes. More difficult to diagnose but usually systemic signs are either detectable with further tests or clinically obvious.
- Cushing's disease: clinically obvious hirsutism, polydipsia and recurrent laminitis and restricted to old horses.
- Sarcoidosis: generalized scaling and crusting dermatosis with concurrent multisystemic signs.
- Chorioptic mange (*Chorioptes equi*): parasites are easily detected and pruritus is a prominent feature.
- Dermatophilosis (chronic) (*Dermatophilus congolensis*): crusting and characteristic paint-brush lesions with detectable typical bacteria in both crusts and purulent exudates from lesions. Usually restricted to dorsum and limbs but can be generalized.
- Dermatophytosis (*Trichophyton* spp.): characteristic epidemiology and microbiology. Spontaneous recovery within 4–12 weeks or response to specific therapy.
- Iodine poisoning (iodism): exposure related, usually via feed supplements or seaweed access.
- Matted hair from heavy sweating with/without chorioptic mange in long-haired draught horses: history and often complicated by secondary bacterial infections.
- Anhidrosis: characteristic history of a move to a different climate and obvious absence of sweating or abnormal patterns of sweating.

Diagnostic confirmation

- Clinical appearance of hair full of 'dandruff' scales, particularly of the mane and tail.
- Biopsy is most often unrewarding.
- Every effort must be directed at establishing a primary underlying cause.

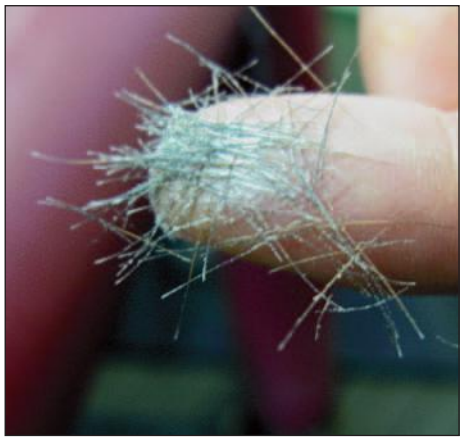


Figure 11.50 This skin debris was removed from the dorsum of a 4-year-old horse that had obvious long-standing generalized matting and greasiness of the hair coat. The debris had an obviously greasy feel typical of seborrhoea oleosa. No primary underlying condition could be found.

Treatment

Close clipping of affected area(s) is helpful but can be difficult (clippers will 'clog' quickly) and it is important to perform this very sympathetically so that the skin is not further damaged. The value of anti-seborrhoeic shampoos such as selenium sulphide if the condition is not greasy can be significant. In greasy cases a soapy antiseptic shampoo such as a surgical scrub antiseptic can be helpful, at least to remove the greasiness.

Antibacterial shampoos and parasiticides can be used if necessary. The hair coat should be kept short and regularly (but not necessarily frequently) shampooed to remove accumulated scale and debris.

Underlying primary disorders may be identifiable and will then need specific treatment.

Amyloidosis

Profile

The term amyloidosis is used to refer to any abnormal accumulation of amyloid in the tissues. It is a rare disorder of the immune system with poorly categorized pathogenesis in which an amorphous eosinophilic hyaline extracellular material (immunocytic or reactive amyloid) is deposited (or develops) in tissues.

The pathogenesis of the abnormal depositions is not well established. The material (amyloid L in immunocytic forms and amyloid A in reactive types) is a metabolic breakdown product of circulating imunoglobulins. Persistent stimulation of the reticuloendothelial system may be involved in some cases but in others no such state can be identified.

Amyloid deposition in the skin may be linked to mononuclear phagocytes, plasma cells and keratinocyte reactions. Deposition of amyloid in any tissue will necessarily lead to altered architecture and possibly altered organ function. The accumulations can be slow to develop or in a few cases a shorter course is noted.

Key points: Amyloidosis

9 11 15 19 20 24

1. Rare nodular disease of unknown aetiopathogenesis, affecting the subcutaneous regions, the respiratory tract mucosa and the conjunctiva. Systemic (reactive) forms seldom affect the skin.
2. Palpable, non-painful, non-pruritic subcutaneous nodules, papules and plaques. Conjunctival and respiratory forms also occur. The latter is associated with various degrees of respiratory tract compromise and nasal lesions are easily traumatized and then bleed heavily. The conjunctival form usually shows as a small chain of linked, pale yellow nodules under the lower lid conjunctiva.
3. Diagnosis can be confirmed by biopsy or excisional biopsy.
4. There is no treatment apart from surgical excision. Spontaneous remissions and improvements following corticosteroid administration are usually temporary.
5. The condition seldom affects the horse itself but there may be an underlying chronic inflammatory process and so that may be therapeutically and prognostically more important.

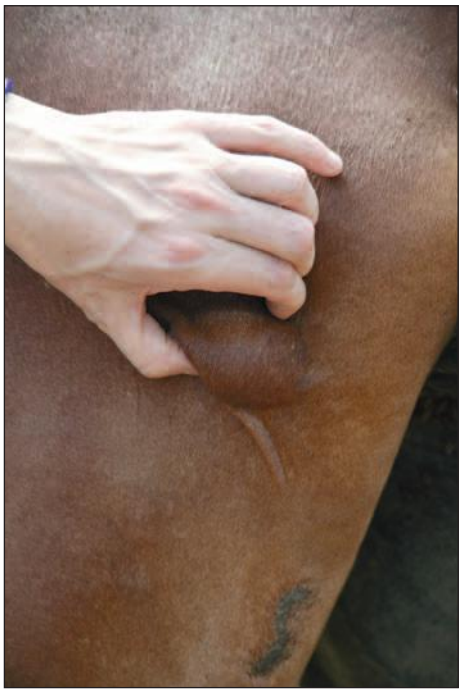


Figure 11.51 This pony had several firm nodular dermal lesions that were identified as equine cutaneous amyloidosis on biopsy. All the nodules sampled showed an identical pathology.



Figure 11.52 This horse shows the typical immune-mediated amyloidosis within the nasal mucosae. Even minor trauma caused significant bleeding.

Two forms of amyloidosis are recognized in the horse.

1 Primary immunocytic, organ-limited amyloidosis: It most often affects the skin and respiratory tract mucosa and seldom involves internal organs.
2 Secondary reactive, systemic amyloidosis. This rarely affects the skin but it may do so in some cases.

Clinical signs

Tumour-like amyloid nodules and subcutaneous amyloid accumulations are identified as multiple nodules, papules or plaques in the skin without affecting overlying hair and other adnexae. The head, neck and breast areas are more commonly involved but it can affect any site (Fig. 11.51) including the eyelids and external genitalia. Extensive chains and coalescing masses can occur. The lesions are firm, non-painful on palpation and non-pruritic.

Concurrent or independent lesions and fragile plaques may be identified on the nasal mucosa (Fig. 11.52). These sites bleed extremely easily with mild trauma – even handling or touching of these mucosal lesions results in prolonged bleeding. The more rostral areas of medial nasal septum are the sites most often reported. More severe cases can have much more extensive respiratory mucosal involvement – nasal respiratory obstructions and stridor and snoring are possible. The conjunctiva can also be affected – pale yellowish chains of nodules are seen in the palpebral conjunctiva of the lower lid most often.

Note

The systemic form seldom involves the skin but can do so in rare cases. Therefore where the diagnosis is made from diagnostic tests (see below) it is vital to eliminate underlying disease – attempts to manage the lesions will be fruitless if there is a serious underlying condition.

Differential diagnosis

- Sarcoid (nodular forms): typically multiple, often interlinking and many will have cutaneous involvement with overlying alopecia and scaling with dermal thinning or thickening. Biopsy is characteristic.
- Melanoma: typically black and some defined anatomic locations. Grey horses mostly affected and some ulcerate making the diagnosis simple.
- Lymphosarcoma: multiple or single solid lesions in the skin and concurrent evidence of serious internal disease.
- Eosinophilic granuloma: benign, typically small and multiple nodules on the back, buttock, neck regions; typical histology.
- (Ulcerative) lymphangitis (*Corynebacterium pseudotuberculosis*): ulceration and cording of lymphatics with honey-coloured exudate.
- Epizootic lymphangitis (*Histoplasma farciminosum*): limited geographical distribution and multiple ulcerative nodules. Organism can be identified from pus.

Diagnostic confirmation

- Characteristic clinical signs (especially if nasal/conjunctival lesions are present also).
- Biopsy or surgical excisional biopsy:
  - If the condition is suspected the pathologist should be informed so that special stains can be applied.
  - A simple test for amyloid involves painting the surface of the cut lesion with Lugol's iodine and then rinsing it off with a 1% sulphuric acid solution. Amyloid stains a deep brown colour.
- Possible infectious or neoplastic conditions could be involved.

Treatment

Amyloid deposits are extremely insoluble and almost totally resistant to any naturally occurring proteases and reparative processes. This makes removal by surgery the only really viable option. This is often complicated and repeated procedures are usually required. However, some cases appear to resolve spontaneously and others benefit from glucocorticoid administration. Whether these effects are a result of reduced immune stimulation is uncertain.

In the event that resolution is apparently achieved, most cases recur and become more difficult to manage in time. Reduction in any primary immune-stimulating process may be possible and then deposition may be slowed.

References

Affolter V K, Von Tscharner C 1993 Cutaneous drug reactions: a retrospective study of histopathological changes and their correlation with the clinical disease. Veterinary Dermatology 4:79–86

Barbet J, Bevier D, Greiner E C 1990 Specific immunotherapy in the treatment of *Culicoides* hypersensitive horses: a double blind study. Equine Veterinary Journal 22:232–235

Bryans J T, Doll E R, Shephard B P 1964 The etiology of strangles. Cornell Veterinarian 54:198–205

Cotran R S, Kumar V, Robbins S L (eds) 1994 Robbins pathological basis of disease, 5th edn. WB Saunders, Philadelphia

Degler J M 1997 Pruritic dermatoses. In: Robinson N E (ed) Current therapy in equine medicine, 4. WB Saunders, Philadelphia, pp 377–381

Dunham R J 1979 The evaluation of 'Sumifly' for the control of Queensland itch (*Culicoides brevitarsus*) on horses. Shell Research Report, Melbourne, Australia

Evans A G 1987 Recurrent urticaria due to inhaled allergens. In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, p 619

Evans A G 1990 Diseases of skin. In: Smith B (ed) Large animal medicine. CV Mosby, Philadelphia, pp 1258–1260

Fadok V 1995 Update on four unusual equine dermatoses. Veterinary Clinics of North America. Equine Practice 11:105–110

Ferraro S L 2001 Pastern dermatitis in Shires and Clydesdales. Journal of Equine Science 21:524–525

Fritsch P O, Elias P M 1993 Erythema multiforme and toxic epidermal necrolysis. In: Fitzpatrick T B, Eisen A Z, Wolff K et al (eds) Dermatology in general medicine, 4th edn. McGraw-Hill, New York, pp 585–600

Gell P G H, Coombs R R A 1964 Clinical aspects of immunology. FA Davis, Philadelphia

George L W, White S L 1984 Autoimmune skin diseases of large animals. Veterinary Clinics of North America. Large Animal Practice 6:79–86

Halliwell R E W 1992 Comparative aspects of food intolerance. Veterinary Medicine 87:893–899

Huytra F, Marek P 1922 Particular pathology and therapy of the pets, 36th edn. Fishers, Jena, Austria, p 534

Karcher L F, Scott D W, Paradis M, Anderson W I 1990 Sterile nodular panniculitis in five horses. Journal of the American Veterinary Medical Association 196:1823–1826

La Perle K M, Peircy R J, Long J F, Blomme A G 1998 Multisystemic, eosinophilic, epitheliotropic disease with intestinal lymphosarcoma in a horse. Veterinary Pathology 35:144–146

Littlewood J D 1999 Food allergy and urticaria: fact or fiction. Proceedings of the BEVA specialist days on Behaviour and Nutrition. BEVA Publications, Newmarket, Harrogate, UK, pp 73–75

Manning T O 1983 Pemphigus foliaceus. In: Robinson N E (ed) Current therapy in equine medicine, 1. WB Saunders, Philadelphia, pp 541–542

Marshall C 1991 Erythema multiforme in two horses. Journal of the South African Veterinary Association 62:133

Mathison P T 1995 Eosinophilic nodular dermatoses. Veterinary Clinics of North America. Equine Practice 11:75–83

Reef V B 1987 Vasculitis. In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, pp 312–314

Rietschel R L 1997 Irritant contact dermatitis. Mechanisms in irritant contact dermatitis. Clinical Dermatology 15:557–559

Rosser E J, Ihrke P J, White S D et al 1983 Equine pemphigus foliaceus. Equine Veterinary Science 3:14

Sanford S E 1989 Multisystemic eosinophilic epitheliotropic disease in a horse. Canadian Veterinary Journal 30:253–254

Schlipf J W 1997 Dermatologic conditions associated with crusts and scales. In: Robinson N E (ed) Current therapy in equine medicine, 4. WB Saunders, Philadelphia, pp 381–386

Scott D W 1988 Dermatohistopathology. In: Large animal dermatology. WB Saunders, Philadelphia, pp 29–49

Scott D W 1990 Le lupus discoide equin: description de trois cas. Point Vet 22:7–9

Scott D W 1997 Erythema multiforme in dogs, cats and horses: literature review and case material from Cornell. Proceedings of the British Veterinary Dermatology Study Group, Stratford upon Avon, 1997

Scott D W, Miller W H 2003a Skin immune systems and allergic skin disease. In: Equine dermatology. WB Saunders, Philadelphia, pp 427–448

Scott D W, Miller W H 2003b Discoid lupus erythematosus. In: Equine dermatology. WB Saunders, Philadelphia, pp 501–504

Scott D W, Miller W H 2003c Immune-mediated disorders. In: Equine dermatology. WB Saunders, Philadelphia, pp 522–534

Scott D W, Miller W H 2003d Immune mediated disease. In: Equine dermatology. WB Saunders, Philadelphia, pp 515–521

Scott D W, Miller W H 2003e Miscellaneous skin diseases. In: Equine dermatology. WB Saunders, Philadelphia, pp 668–675

Scott D W, Walton D K, Blue M G 1984 Erythema multiforme in a horse. Equine Practice 6:26–30

Sloet van Oldruitenborgh-Oosterbaan M M 2004 Equine allergic disease. Proceedings of the Fifth World Congress of Veterinary Dermatology, Vienna, Austria. Blackwell Publishing, pp 349–359

Sloet van Oldruitenborgh-Oosterbaan M M, Enzerink E, Koeman J P, Knaap J H 2000 Pasture dermatosis in the non-pigmented lower limb in the horse. Veterinary Dermatology 11(supplement):21–31

Spiegal I B, White S D, Foley J E et al 2006 A retrospective study of cutaneous equine sarcoidosis and its potential infectious aetiological agents. Veterinary Dermatology 17:51–62

Stannard A A 1987 Photoactivated vasculitis. In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, pp 645–646

Stannard A A 1994a Urticaria. Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Stannard A A 1994b Systemic lupus erythematosus. In: Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Stannard A A 1994c Alopecia areata. In: Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Stannard A A 1994d Sarcoidosis. In: Proceedings of Workshop on Equine Dermatology, European Society of Veterinary Dermatologists, Newmarket, UK

Stannard A A 2000a Pastern leukocytoclastic vasculitis. Veterinary Dermatology 11(supplement):217–220

Stannard A A 2000b Immunologic diseases (systemic granulomatous disease). Veterinary Dermatology 11(supplement):170–172

Thomsett L R 1984 Non-infectious skin diseases of horses. Veterinary Clinics of North America. Large Animal Practice 6:59–78

Van Pouke S, van Hauwermeiren H, Hunt L et al 2005 erythema multiforme in a pony. Vlaams Diergeneeskundig Tijdschrift 74:355–358

Volland-Francquille M, Sabbah A 2004 Recurrent or chronic urticaria in Thoroughbred racehorses: clinical surveillance. Immunology 36:9–12

Wilkie J S N, Yager J A, Nation P N et al 1985 Chronic eosinophilic epitheliotropic disease in five horses. Veterinary Pathology 22:297–305

Williams M A, Dowling P M, Angarano D W et al 1995 Paraneoplastic bullous stomatitis in a horse. Journal of the American Veterinary Medical Association 207:331–334

Wilson A D, Harwood L J, Bjornsdottir S et al 2001 Detection of IgG and IgE serum antibodies to *Culicoides* salivary gland antigens in horses with insect dermal hypersensitivity (sweet itch). Equine Veterinary Journal 33:707–713

Yu A A 2006 Eosinophilic granuloma (nodular necrobiosis). Proceedings of the 52nd Conference of the American Association of Equine Practitioners.

Zabel S, Mueller R S, Fieseler K V et al 2005 Review of 15 cases of pemphigus foliaceus in horses and a survey of the literature. Veterinary Record 157:505–509

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Chemical, toxic and physical dermatoses

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Skin trauma can be primary (caused by direct physical insult, chemical applications) or secondary when the insult itself would normally have no material effect. The damage is a combined result of an insult and a predisposing pathology. Primary injury can be a result of obvious traumatic damage, or application of or accidental contact with damaging/caustic chemicals. Perhaps the best example of secondary damage is the photosensitization that is a result of liver failure and the ingestion of photodynamic plants. Without the sunlight exposure the damage would not occur even though the underlying pathology/toxin is present.

Chemical and toxic dermatoses are rare in horses but arise from systemic poisoning or from direct application of toxic chemicals to the skin. The occurrence of systemic toxicity is singularly rare but recognition of the disorders is important if only because of the strong tendency that owners have to blame some form of 'outward' interference in the health of the horse.

The disorders that result from some external application or in particular circumstances when the toxicity is either driven by some external factor such as sunlight or is exacerbated by management procedures are more common.

Owners may not readily appreciate the risks of plant or chemical poisonings or may be inclined to blame agents that in fact have no material implication. Where poisonings are suspected the clinician really needs to be very careful before making a confirming statement.

Traumatic damage to the skin also occurs from direct injuries and there are some important dermatological states that have a physical aetiology. Some of these such as burns and scalds have obvious causes but their management is a dermatological concern. However, it is important to realize that skin wound healing is a dermatological matter in the final analysis. Many of the problems faced in wound healing are surmountable if the clinician has a detailed knowledge of the structure and function of skin.

Chemical trauma

Profile

History of application of 'pour-on' or topical medications such as antiseptics, insecticides or fungicides (or other materials) is usually obtained. Application of over-strength medication or of normal-strength medication on an oversensitive skin is a common feature. Application of blisters,

Key points: Chemical trauma

10 11 12 15 19 24

1. Common result of inappropriate applications of chemicals to the skin. Mistakes made with calculating concentrations of chemicals or application of misguided 'cures'. Damage can be severe and is often permanent.
2. Exudative or necrotizing dermatitis following known (or suspected) applications of skin chemicals. Usually lesions are confined to the area of application but systemic effects can be induced. Ingestion by licking of the damaged skin can result in oral 'burns', oesophageal ulceration or necrosis and gastrointestinal damage. Strong chemicals such as arsenicals and oils can cause skin necrosis and severe systemic effects.
3. Diagnosis may be more difficult than it seems because many owners will not readily admit to having caused the damage. Histology is sometimes helpful
4. Treatment depends on removing the chemical as soon as possible using suitable solvents and washes without further skin injury and application of wound management procedures. Systemic effects will require more supportive management.
5. The prognosis is variable depending on the type and extent of the damage and the implications for the anatomical region. Many cases have persistent problems with hyperaesthesia and recurrent infections.



Figure 12.1 Accidental chemical burn causing matting and exudation of the skin.



Figure 12.3 Application of a strong arsenic powder caused cutaneous coagulative necrosis. The large eschar sloughed slowly, leaving a large cicatrized scar.



CD

Figure 12.2 Extensive hair loss and generalized dermatitis arising from the ill-advised application of hot engine oil in a misguided attempt to treat louse infestation.

counterirritants and vesicants is a common iatrogenic cause of this response, where it is regarded as a good therapeutic effect (see Fig. 12.12). Prolonged skin contact with surgical spirit or other chemicals may arise during general anaesthesia (recumbency) and can cause serious problems which are actually 'chemical burns'. Historically, old/used engine oil has been used to treat lice and insect bite (*Culicoides*) hypersensitivity (sweet itch) and this can cause extensive and severe damage to skin and has serious potential systemic toxicity.

Exposure to inappropriate washing materials such as detergent washing powders, bleach and other materials can leave a serious long-term skin change. Often the consequences are detected some weeks after the exposure.

Clinical signs

Acute cases show marked moist, exudative dermatitis with matting of hair (Fig. 12.1). Extensive generalized dermatitis such as is caused by application of engine oil causes severe hair loss and a fragile skin which is easily traumatized. Hair loss (Fig. 12.2) and sometimes even open wounds are common consequences.

Chronic or mild recurrent skin insult causes fine to coarse scaling and (temporary) loss of hair (alopecia) over the affected area, often well demarcated over the area of application (e.g. following therapeutic blisters).

Alopecia, pigmentary changes and overt scarring are often induced when deeper damage has been caused by prolonged contact with chemical substances. Pruritus and self-inflicted trauma may be marked. Longer-term effects include skin thickening with lichenification and rugae formation.

Focal application of strong chemicals, focal burns or physical cautery to the skin may result in a dense, coagulated slough (eschar) which may remain in situ for prolonged periods (a 'sitfast') (Fig. 12.3) and inevitably leaves a scar.

The long-term results of chemical damage to the skin can be marked. Inappropriate washing materials such as detergent powders and engine cleaners, etc. can cause an immediate problem, of course, but the long-term damage to the skin is often much more important. Skin pain, sensitivity to sunlight, or even inflammatory damage from basic grooming are all possible **(Fig. CD12 • 1A–E)**. Affected horses usually have varying periods when they are better and worse but may never be normal again.

Differential diagnosis

- *Culicoides* spp. or other insect hypersensitivity (sweet itch, etc.): seasonally related pruritic condition that depends heavily on exposure to causative insects – usually *Culicoides* species.
- Louse infestation (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): parasites are easily detected by visual inspection and by brushings. Mild pruritus and a moth-eaten appearance may be detected.
- Insect bites with secondary self-inflicted trauma: focal bites following exposure to biting/venomous insects; focus of the bite/sting may be detectable.
- Dermatophytosis (*Trichophyton* spp.): typical non-pruritic, scaling disorder with characteristic epidemiology and self-resolution within 6–12 weeks; specific therapy resolves the condition and mycological examination is simple.
- Dermatophilosis (*Dermatophilus congolensis*): distinctive, mildly painful dermatitis localized to wetting areas and exposure to persistent wetting of the coat. Bacteria are easily identified in smears from crusts and pus from the lesions.

- Chorioptic/psoroptic/poultry (red) mite infestation: pruritic disorders mainly restricted to legs and parasites are easily identified. However, they may be present in low numbers so brushings are the ideal specimen to examine.
- Linear keratosis: asymptomatic hair loss in linear patterns often over the neck and lateral chest wall.
- Scars: injury related.
- Wounds: obvious crusting at site of wounds unrelated to chemical exposure.
- Scalding: accidental exposure to scalding liquids is very rare in horses.

Diagnostic confirmation

- History of application of pour-on insecticides, blisters or other over-strength medication/applications. Owners, however, may deny having used anything at all. The dermatological evidence may be delayed for days or even weeks after the event.
- The lesions usually have a 'run-off' pattern and distribution that corresponds to application of chemicals.

Treatment

Acute cases should be washed with warm, bland soapy water and dried. An emollient cream can be applied but the over-riding objective is to remove the original insult and cause no further damage. The value of topical and systemic corticosteroids is debatable but some cases do benefit from a single dose or short course of oral prednisolone.

Lesions often resolutely fail to heal and/or leave extensive unsightly scars which may require corrective surgery with skin grafting.

In many cases it is simply a matter of tolerating the disfigurement and managing the pain, pruritus or wound sites.

Skin scalding (diarrhoea/urine or wound exudate)

Profile

The skin does not tolerate persistent soaking in body fluids such as urine, milk, plasma, serum, lacrimal secretions, synovial fluid or saliva. These body fluids and prolonged or irritant diarrhoea can lead to marked hair matting, scalding and hair loss in the affected skin. These conditions are therefore usually secondary to other pathological disorders. Secondary infections are common.

Wound fluids (whether infected or not) may cause serious excoriation and loss of hair.

Diarrhoea in foals causes severe perineal dermatitis – the prolonged exposure of the dermis to infective agents can lead to significant infection but this is remarkably rare. In adult horses with diarrhoea or those subjected to repeated rectal examinations the perineal region can become tender and mildly inflamed. Often this passes unnoticed.

Urine scalding can occur quickly depending on whether the flow is intermittent/splashing or soaking. Geldings with neurogenic urinary tract disorders including fractures of the sacrum, polyneuritis equi (cauda equina syndrome) or poliomyeloencephalopathy (such as occurs in the neurogenic form of equine herpesvirus infection) and those with idiopathic bladder paralysis syndrome may persistently drip urine onto the hind legs. Usually this causes severe persistent dermatitis (often with secondary infections) and crystallization on the hair which smells strongly of urine. In mares the dermatitis caused by the same group of conditions results in perineal skin damage – usually in an obvious flow pattern from the ventrum commissure of the vulva. Crystallization on the hair indicates chronicity and severity. Milder splashing may cause some localized transient inflammation but seldom extensive dermatitis.

One of the most damaging materials for the skin is wound exudate. Blood and fresh plasma are not significant irritants to the skin but wound exudate is. Often this has altered acidity and may be laden with bacteria.

Tear overflow can also cause superficial dermatitis and this is a primary aspect of facial *Habronema* infection (see p. 217).

Clinical signs

Diarrhoeic animals (particularly foals) may have mild-to-moderate perineal skin inflammation and temporary loss of hair (Fig. 12.4). The cause of the problem is usually obvious and although it can be significant, controlling the primary cause is the most important aspect of this. Repeated rectal examinations also causes some local irritation and inflammation.

Urine scalding usually occurs around caudal area of hind legs of mares or the lower regions of the hind legs in geldings with urinary incontinence (Fig. 12.5). Where urine is responsible an obvious stale, ammoniacal, urinary smell on legs can be detected. Urine and faeces may contain blood and this can be obvious. Skin may be extensively damaged and exudative.

Hair matting is more obvious with wound fluids; serum exudate appears to induce a moderate or severe dermatitis and this also seems to encourage the development of skin infections. Secondary *Habronema musca* infestation can occur with wound exudate or lacrimal secretions on the face (see p. 217).

Key points: Skin scalding

10 11 14 17 24

1. The skin does not tolerate persistent bathing in body fluids such as lacrimal secretion, blood, serum/plasma, urine or milk. Diarrhoea also causes skin damage, particularly in foals.
2. Signs are associated with bathing in obvious body fluids. Hair matting commonly occurs first. Mild to moderate dermatitis occurs with hair loss and secondary infection.
3. Diagnosis is usually simple. Urine has a characteristic odour and crystallized calcium carbonate is attached to the hair.
4. Treatment is dependent on stopping the contact between the skin and the fluid either by correcting the cause or by protecting the skin in some way. Initially the skin should be cleansed carefully with minimal extra trauma. Dead hair and skin should be removed and the infection controlled appropriately. Thereafter the area should be protected with emollient and soothing water repellent creams or petroleum jelly to allow healing.
5. The prognosis for the skin is good (provided that the primary cause can be managed) but in long-standing cases there may be some persistent changes in hair coat and skin thickness and flexibility.



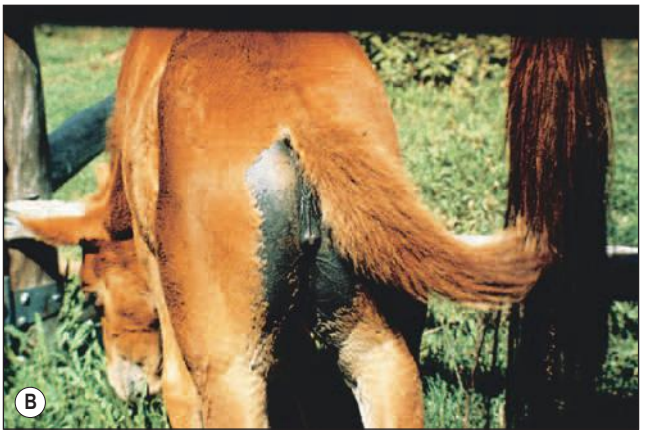


Figure 12.4 **(A)** Mild early perineal dermal inflammation caused by diarrhoea. **(B)** Later on the hair falls out and the bald skin heals eventfully with full hair growth.

A similar diffuse excoriation of the perineum with diffuse dermatitis can be caused by repeated rectal examinations and other investigative procedures (such as ultrasound examinations using alcohol as opposed to coupling gel) involving repeated superficial trauma to delicate skin areas, especially when water-soluble lubricants are not used and when any lubricant is left on the skin after the procedure.

Differential diagnosis

- Dermatophilosis: characteristic distribution and epidemiology affecting horses in poor conditions and subjected to persistent wetting. *D. congolensis* can be detected in scrapings and smears of exudate and crust.
- Dermatophytosis: localized crusting and scaling dermatosis with characteristic epidemiology and mycology. Most cases spontaneously resolve or respond rapidly to specific treatment.



Figure 12.5 Urine scalding dermatitis affecting the dorsal aspects of the hind cannons in a gelding with urinary incontinence. Similar effects are encountered in the perineal region of mares with neurological problems in the bladder and vagina.

- Chemical irritants (e.g. strong iodine/disinfectants, mercurial blister): history of application is usually available; characteristic pattern and anatomic distribution corresponding to applications.
- Allergic dermatitis: exudative and no obvious instigatory factors – signs usually restricted to contact regions.

Diagnostic confirmation

- Almost all cases are secondary to other pathology which is usually obvious. A full history and clinical assessment may establish the primary cause.
- If urinary problems are suspected, examine the entire urinary tract to determine whether retention with overflow or cystic calculi is the primary cause.
  - Urinary incontinence can be due to sorghum feed poisoning, cauda equina neuritis, rabies, sacral or spinal fracture or the neurological form of equine herpesvirus 1 (EHV1) infection.
- History of foaling accident, trauma to hindquarters, e.g. rearing over backwards and landing on sacrum or spontaneous sacral fractures in racing horses.
- Primary diarrhoea from any cause (especially in foals).

Treatment

The primary condition should be managed to prevent deterioration but the local effects of skin inflammation and excoriation must also be managed. Early cases with transient primary signs relating to diarrhoea, lacrimation and wound exudate are not usually a major clinical problem. Protective jellies such as petroleum jelly (Vaseline) can be applied to limit the damage. Antiseptic creams such as human baby nappy cream can be very helpful. It is important to wash off all faecal material before applying anything to the skin.

Where treatment of the primary problem is likely to be ineffective, persistent skin care is required, e.g. if there is neurogenic damage or persistent/chronic diarrhoea or wound

exudate. The anatomic location of the dermatitis is also an issue. The skin of the distal limb is easily damaged and is liable to infection so this can result in a long-term management problem. Neurological disorders may be permanent and strategies have to be formulated to minimize the secondary consequences. Repeated severe washing with cold water and antiseptic shampoos is not ideal. In some cases repeated washing can in itself be harmful to equine skin. Even surface application of petroleum jelly can eventually be detrimental to skin health.

When the primary cause is controlled, hair and skin return to normal rapidly. If treatment is ineffective, local protectants are helpful, e.g. petroleum jelly or waterproof barrier creams with antiseptic and soothing properties (emollient creams, zinc oxide cream). Corticosteroid and local anaesthetic creams can be helpful but should not be overused in case they impair local cutaneous immunity.

Actinic dermatoses

Profile

Sunlight (and light derived from other sources) in the ultraviolet part of the spectrum combines short, medium and long wavelengths (UVc, UVb and UVa, respectively). Damage can be caused directly by simple actinic exposure (sunburn) or the various wavelengths of light can react with abnormal (exogenous or endogenous) photosensitizing proteins deposited in the skin (Fig. 12.6). The abnormal response in the latter case is usually much more severe. Melanin protects pigmented skin from the damaging effects of ultraviolet light by absorbing and scattering the ultraviolet light and where skin has little or no melanin the cells can be significantly damaged. All the significant light-related dermatoses are exaggerated in regions lacking in pigment and hair.

Actinic dermatitis may be acute or chronic and falls into two distinct categories:

1 Sunburn (excessive exposure to UV light) with an *expected* (*understandable*) *outcome*. The severity of the damage is dependent upon the strength of the radiation and the sensitivity of the skin to the damage. Pale-skinned horses are far more liable to sunburn than black-skinned horses.
2 Photosensitization (normal exposure to UV light) with *unexpected* or *disproportionate response*. In this case even mild exposure causes severe (disproportionate) damage.

Sunburn is a primary event that follows exposure to strong sunlight. Some individual horses are more liable to the condition and usually these are horses with little pigment in the skin. Susceptible areas that are not protected by hair are even more liable and that is why many cremello/albino and white-faced horses are affected by sunburn on the muzzle. Repeated exposure over the short term and repeated exposure year on year result in chronic skin changes recognized as solar keratosis. This change can be recognized as an early pre-carcinomatous change particularly in the intraepithelial forms of squamous cell carcinoma (in-situ carcinoma) that are found on the cornea and conjunctiva and the face/eyelids in particular.

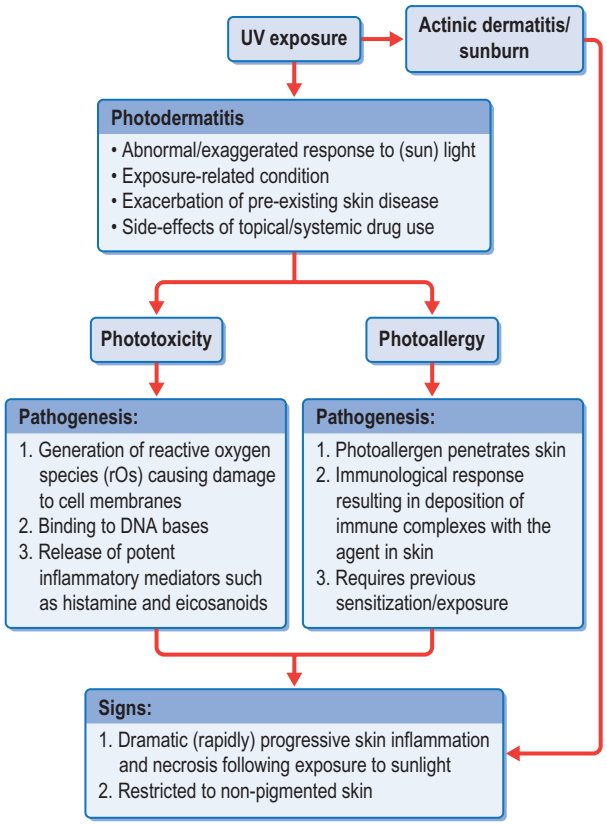


Figure 12.6 Light-related skin disorders. Note the aetiopathogenesis of the various types of light-related dermatosis.

Photosensitization requires three factors:

- the presence of a photodynamic agent in the skin (Table 12.1)
- exposure to sunlight or certain wavelengths of ultraviolet light
- cutaneous absorption of ultraviolet light.

Photosensitization occurs in three forms:

1 *Type 1 (primary) photosensitization*. This form is encountered as a result of the ingestion of preformed photodynamic substances such as those found in certain plants (e.g. St John's wort (*Hypericum perforatum*); Table 12.1) and some chemical 'poisonings' (e.g. phenothiazine anthelmintic). The ingested photodynamic agent is absorbed directly from the digestive tract and is delivered to the skin without any hepatic detoxification or alteration. The result is predictable in most horses and is seldom severe.

Primary photosensitization may be involved in some variants of photoactivated pastern and cannon leukocytoclastic vasculitis. In this condition photodynamic complexes may be formed through combination between tissue proteins and plant proteins on the surface of the skin. In these cases the effects can be more severe but remain localized to the exposed regions (see p. 305).

2 *Type 2* (*endogenous*) *photosensitization*. In these cases endogenously derived metabolites act as photodynamic agents. Examples include porphyrins. This form has not been reported in horses.

Table 12.1 Agents associated with photodermatoses in horses

Origin	Name	Photodynamic agent(s)	Route
Plants	Buckwheat *Polygonum fagopyrum*	Fagopyrin	Ingested
	Dutchman's breeches *Thamnosma texana*	Psoralen	Ingested
	St John's wort *Hypericum perforatum*	Hyperacin	Ingested
	Perennial ryegrass *Lolium perenne*	Perloline	Ingested
	Whiteheads, ranger's buttons *(Sphenociadium capitellatum)*		Ingested
	Giant hogweed *Heracleum mantegazzianum*	Furanocoumarin	Contact
	(Alsike/red) clover *Trifolium hybridum* *Trifolium pretense*	Phenolic glycosides (including trifoliin), isoflavonoids	Ingested Possibly contact
Fungi	*Pithomyces chartarum*	Phytoallexins	Ingested
Toxic chemicals/drugs	Carbon tetrachloride		
	Retinoids Therapeutic photoagents	Tazarotene	Surface application
			Surface application/systemic
Endogenous metabolites*	Porphyrins	Genetic abnormality	Porphyrin
	Phylloerythrin Bilirubin	Hepatic failure	Ingested (secondary)
		Liver/blood disorders	Endogenous

*Endogenous metabolites are formed either within the gut directly or via bacterial activity or are formed as a result of abnormal metabolic pathways.

3 *Type 3* (*secondary*) *photosensitization*. Digestion of chlorophyll produces a potent photodynamic agent, phylloerythrin, which is normally detoxified and excreted by the liver. Severe/advanced liver failure allows the substance to pass into the bloodstream unchanged. It accumulates in the skin where it becomes a highly reactive material when exposed to UV light. Severe/extreme actinic damage that is disproportionate to the amount of sunlight applied to the skin is the common presentation. The aetiopathogenesis is related to concurrent hepatic failure and is therefore unpredictable – failure to recognize the primary hepatic disease leads to recurrent episodes and continued suffering.

Most of the primary photosensitizing plants and the hepatotoxic plants are only marginally palatable, but signs can be seen during periods of ingestion of the plant, i.e. during grazing in summer months. Significant ingestion of hepatotoxic plants such as *Senecio* spp. or *Lantana* spp. is unusual from grazing situations but horses are unable to identify and reject the plants in preserved forage (hay or silage). Thus, most cases develop in late winter and spring and manifest when the ultraviolet light levels increase in early summer.

Specific primary and hepatotoxic plants are probably well known to veterinarians practising in the areas concerned. Almost every country has examples of both types of plant.

There are circumstances when the tissue-toxic photoactivity results when a combination of antibody and a photodynamic agent is deposited in dermal and subcutaneous blood vessel walls and the dermis. In this case the response is complicated by autoimmune or other immunological responses that, in horses at least, are poorly understood. An example of this response may be seen in pastern and cannon leukocytoclastic vasculitis (see p. 273).

In addition to the primary and secondary photosensitization conditions that are described below, there are several skin disorders that are exacerbated by exposure to sunshine/UV light. It is suggested that cytokines released by keratinocytes act as the photodynamic agents. Interestingly, sun exposure is usually necessary for the conditions to develop initially; most cases develop in summer months at grass. Once instigated, removal from the sun may have little initial benefit and the condition may persist thereafter without sunlight exposure. Even minor sunlight will trigger exacerbation. These conditions include systemic lupus

Key points: Actinic dermatoses

1 Dermatitis of varying severity is caused either directly by the action of ultraviolet light on skin (sunburn) or through the effects of a photodynamic chemical or metabolite deposition in the skin deposited in or on the skin. Endogenous photosensitization as a result of porphyrins etc. has not been reported in horses but some drugs such as phenothiazine can cause the problem.
2 Mild to extreme necrotizing dermal inflammation is characteristic. The proportionality of the damage to the degree of sunlight provides the best indicator of the likely type of photosensitization present. White-skinned areas and pale glabrous (hairless) skin are most liable to the condition.
3 Diagnosis is relatively easy when the dermatitis is restricted to non-pigmented skin in summer months. Histologically the changes are not pathognomonic.
4 Primary sunburn is easily managed by avoidance or sunscreen creams, but the risks associated with repeated exposure should be considered. Type 2 primary photosensitization will resolve if the causative plant/agent is eliminated. For secondary photosensitization as a result of hepatic failure, treatment is usually supportive. Antibiotic treatment and avoidance of sunlight exposure are always sensible. High factor sun creams are not really the answer because they may be used as an excuse to avoid management strategies which are far more effective. Avoidance of any defined causative plant/toxin is essential.
5 The prognosis for primary photosensitization is good. For advanced hepatic failure the prognosis is hopeless.



Figure 12.7 Sunburn on an unpigmented nose. No other areas were affected and the animal was otherwise very healthy.

erythematosus-like syndrome (see p. 276), and discoid lupus (see p. 270).

Ultraviolet light is also potentially mutagenic and carcinogenic but it can also suppress skin inflammation. Therefore sunlight can be both harmful and beneficial. Certain skin tumours, in particular squamous cell carcinoma, are thought to be precipitated by exposure to ultraviolet light and certainly the cutaneous and conjunctival forms of this neoplasm (see p. 427) are much more common in non-pigmented tissues and in regions where more sunlight exposure occurs.

The tissue toxicity effects of light and photodynamic compounds can be used therapeutically in photodynamic therapy (see p. 126).

Clinical signs

Cutaneous lesions vary widely in both severity and extent but are usually restricted to light skin or hairless areas.

1 Simple sunburn (Fig. 12.7) can be difficult to differentiate from the more serious forms of actinic dermatitis but the horse is usually clinically normal and often has very pink skin with minimal hair cover over the muzzle and face. The extent of the damage is usually much less severe than in true photosensitization but repeated exposure can cause significant exacerbation. Most prominent signs are found on the face, commonly around lips, nose and eyelids. If there is significant exudation there may be a complicating superficial infection with *Dermatophilus congolensis* (see Fig. 6.22). A history of previous problems during summer months is often given. Most (if not all) cases improve quickly when removed from exposure and the skin rapidly returns to normal.
2 The more severe, systemically mediated condition (either from direct photodynamic agents or hepatic failure) is usually much more serious and has a much more destructive nature. Again, the lesions are typically restricted to the white areas on the face and elsewhere (Fig. 12.8) and are always sharply restricted to the non-pigmented skin (Fig. 12.9). Severe cases may have lesions which just overlap into dark-skinned, well-haired areas. Conjunctivitis, skin oedema, erythema, pruritus and pain are usually present as the skin becomes necrotic. The perineum and coronary band region may be severely affected (Fig. 12.8). Extensive sloughing of skin occurs in most severe cases. Concurrent systemic signs of hepatic disease may be present (ventral oedema, hepatoencephalopathy, icterus and weight loss) with biochemical evidence of hepatic failure.
3 Immune-mediated photosensitizations are usually associated with concurrent signs in other body systems. The damage to non-pigmented skin may be more severe than at other sites but usually these conditions have features that make it recognizably different from the damage caused by endogenous or exogenous photodynamic agents. The one unusual form in this group is pastern and cannon leukocytoclastic vasculitis (see p. 273).

The role of plant or toxic chemicals and ultraviolet light in the pastern-related dermatoses (and pastern leukocytoclastic vasculitis in particular) is uncertain but the distribution



Figure 12.8 Actinic dermatitis (photosensitization) affecting the white facial stripe and the white digits in a mare with liver failure. The foot lesions affect the lateral aspects far more than the medial.



Figure 12.9 Actinic dermatitis (photosensitization) (severe) affecting the white areas of the skin in a piebald pony. Notice the sharp 'cut-off' of the severe dermatitis at the margins of the white areas.

of the lesions suggests in some cases that sunlight is involved (see Fig. 11.26).

- Generalized lesions are suggestive of liver involvement.
- Localized lesions on lip and distal limb are suggestive of primary photosensitization (pasture plants, sprays or drugs).
- Involvement of the lateral aspects of pigmented digits only may suggest pastern leukocytoclastic vasculitis.
- Involvement of the face only in cream horses or those with lightly pigmented faces may suggest sunburn alone. Sunburnt horses usually have a particular distribution of the skin lesions of the dorsal area of the nose rather than on the upper lip region.

Note

All horses showing any evidence of photosensitization *must* be investigated for possible hepatic damage using the full range of liver function tests (including bile acids, hepatic enzymes and possibly liver biopsy).

The detection of pyrrolizidine alkaloids on erythrocytes has been described and is probably the definitive test for current ingestion of the plants (Mattocks & Jukes 1991, 1992). Feeds can be closely examined for the plants themselves but this is not always easy and it may be better to analyse the feeds for the alkaloid.

Differential diagnosis

- Sunburn: primary actinic dermatitis arises when there is strong sunlight and is restricted to non-pigmented skin.
- Dermatophilosis (*Dermatophilus congolensis*): mildly painful, exudative and crusting dermatosis distributed along back and limbs mainly. Organism can be identified in smears from pus and crust.
- Dermatophytosis (*Trichophyton* spp.): characteristic epidemiology and self-cure within 6–12 weeks.
- Pastern and cannon leukocytoclastic dermatitis: probably a form of photoactivated dermatitis relating to pasture exposure with very restricted anatomic distribution on the lateral pastern/cannon regions.
- Greasy heel syndrome: proliferative verrucose pastern dermatitis condition resulting in massive swelling and nodule formation most often in feathered horses and largely restricted to white limbs.
- Photo-exacerbated skin disorders such as discoid lupus and systemic lupus erythematosus-like syndrome: rare and much less aggressive skin responses but with systemic signs in some cases.

Diagnostic confirmation

- The history is critical. Many affected horses have significant features of the history that help with the diagnosis. Weight loss, exposure to primary photosensitizing plants or hepatotoxic substances (including pyrrolizidine alkaloids) or to dietary supplements, antibiotics, or other drugs can be a significant aspect of the history.
- Physical examination with localized damage in non-pigmented or lightly pigmented skin.
- Blood sample for detection of alkaloid on red cells (lithium heparin anticoagulant) only effective during ingestion stages.
- Biopsy of skin is usually non-specific with extensive necrosis and severe inflammation.

Treatment

Elimination of all possible sources of photodynamic agents is essential.

The affected areas must be protected from direct or reflected sunlight by immediate stabling, use of hoods, rugs, bandages, etc. Some protection can be provided by sunblock creams with factors greater than 20. Emollient/soothing and local anaesthetic creams can be applied. Some of the conditions, however, continue after the horse is brought indoors.

Glucocorticoids and non-steroidal anti-inflammatory drugs may be of assistance in reducing inflammation but may exacerbate hepatic failure and concurrent hepatoencephalopathy. There are some conditions and individual horses where simple avoidance of the sunlight is not necessarily 'curative' and then steroids and non-steroidal drugs are an advantage.

Symptomatic treatment of liver disease or other disorders should be instituted, but chronic severe liver failure is not usually treatable. Therefore, cases of true hepatic-related photosensitization carry a very poor prognosis. It is vital to consider hepatic disease in all cases where the dermatitis is restricted to white/non-pigmented skin.

Plant poisoning (leucaenosis/mimosine toxicity)

Profile

The ingestion of plants such as *Leucaena leucocephala*, which is used as forage and shade trees, and mimosa (*Mimosa pudica*), which is the common 'sensitive' bush, can be associated with hair loss. The active principle in both these plants is mimosine, a toxic, non-protein amino acid with potent depilatory effects. The plants are geographically restricted to humid tropical regions. Horses will only eat the plants in small amounts unless normal forage is in short supply.

Clinical signs

The toxins cause gradual loss of the longer hairs on the mane and tail (Fig. 12.10). Alopecia around fetlock and coronary band can be present also. Severe cases may show chronic laminitis-like ringing to walls of all feet. Loss of appetite and weight loss are common.

Differential diagnosis

- Insect bite hypersensitivity (sweet itch): pruritic, seasonal dermatitis affecting the base of the mane and tail dependent on exposure to *Culicoides* spp. and other insects. The hair loss is a result of breakage not shedding.
- Selenosis (selenium poisoning): similar hair loss pattern and prominent hoof wall and laminitic changes. Known access to selenium supplements or seleniferous plants.
- Iodine poisoning (iodism): generalized seborrhoea, ocular and nasal discharges. Known exposure.
- Mercury poisoning: very rare, exposure-related alopecic changes over whole body and mane/tail.

Key points: Plant poisoning

1 5 10 15 19 24

1. Geographically restricted plant poisoning relating to exposure to specific plants including those producing mimosine.
2. Mane and tail hair shedding occurs. Some coronet hair thinning can also be present.
3. Diagnosis is by deduction from known exposure.
4. Treatment is limited to removal of exposure.
5. The hair may return slowly to normal but some remain thinned.

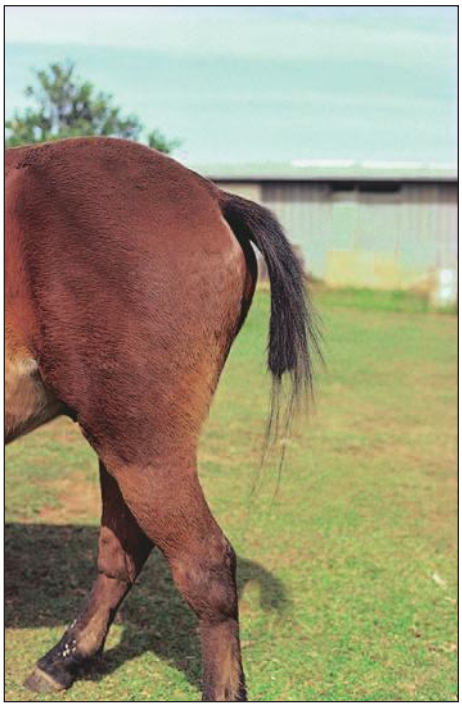


Figure 12.10 Specific *Leucaena* spp./*Mimosa* spp. poisoning causing loss of tail (and mane) hairs but with no effect on body hair.

- Arsenic poisoning: access to organic or inorganic arsenic compounds. Seborrhoea and intestinal and major organ failure. Acute poisoning does not affect skin significantly.
- Coronitis/coronary band dystrophy: restricted changes to coronary band. Usually non-inflammatory.
- Anagen defluxion: symptomless shedding of large amounts of body hair – mane and tail seldom affected.
- Malnutrition: seldom affects mane and tail hairs.
- Mane and tail dystrophy (Appaloosa tail syndrome): breed-related loss of mane and tail hair.
- Traumatic loss of mane/tail hair either from pressure necrosis (such as bandaging injuries of the tail), pruritus (such as insect bite hypersensitivity) or by direct pulling by other horses/other animals: the circumstances are usually obvious and once the instigatory factor is removed most cases return to normal. Scarring damage may however, be permanent.

Diagnostic confirmation

- Access to *Leucaena* spp. or *Mimosa pudica*.
- Elimination of other possibilities.

Treatment

Remove source of plant material. Supportive multivitamin supplementation may help. Other remedies are anecdotal and unreliable.

Selenium poisoning ('bob-tail' disease)

Profile

Chronic excessive selenium ingestion, either through ill-advised feed supplementation or through ingestion of seleniferous plants, results in significant dermatological disease.

Key points: Selenium poisoning

1. Chronic ingestion of excessive selenium (either in seleniferous plants or by direct feed supplementations or in drinking water) results in skin and hoof wall changes. Acute toxicity has no cutaneous implications.
2. Clinical signs include loss of mane and tail hairs and hoof wall defects. Body hairs are less affected but can be thinned and sparse. Coronary band dystrophy is a possible sign. Lameness and severe laminitis can result in hoof shedding. Weight loss is a possible additional finding.
3. Diagnosis is difficult but is most often based on historical ingestion and the geographical availability of seleniferous plants and soils with high selenium.
4. Treatment is largely unrewarding but removal of dietary selenium is critical.
5. Slow recovery can occur but most cases are not treatable.



Figure 12.11 Selenium poisoning. Note the poor coat quality with generalized hair loss and a particularly severe depletion of the mane and tail hairs. The laminitic stance arose from severe 'slippering' of the hooves of all four feet.

Selenium in plants is derived from soil, and some plants such as *Astragulus* spp. and *Oonopsis* spp. are capable of accumulating significant concentrations of selenium if the soil has an inherently high concentration. Examples of plants which actively concentrate/accumulate selenium are *Morinda reticulata*, *Neptunia amplexicaulis*, woody asters and *Senecio* spp. (McKenzie 1994).

Diseases associated with ingestion of seleniferous plants are geographically restricted to areas where the plants occur or where local conditions favour high selenium absorption into plants – concentration ability is often up to 10 000-fold in some circumstances. Unnecessary or excessive administration of selenium supplements may also be responsible for serious toxicity and is possibly much more common in general practice than plant-derived toxicity.

Excess selenium predominately affects keratinocytes that produce hard keratin such as hoof and mane and tail hair. The general loss of primary hairs is likely to be roughly symmetrical and is the result of follicular atrophy. Secondary hairs are less affected or unaffected (Raisbeck 2003). Signs can develop 3 weeks to 12 weeks after exposure.

Clinical signs

The acute form of selenium poisoning shows no dermatological signs.

Chronic poisoning causes dramatic loss of mane and tail hairs in particular (Fig. 12.11). Body hair may become sparse and the skin thin and fragile.

The hoof capsule becomes noticeably abnormal, with weak keratin and hoof tubule structure, and is then flaky, non-waterproof and inclined to horizontal and vertical cracks. Hoof slippering/sloughing may occur. Lameness may be severe with acute and/or chronic laminitis.

Differential diagnosis

- Mimosine toxicity: affects mane and tail hair and is exposure related.
- Coronary band dystrophy: seldom any severe signs and no mane and tail problems.
- Pemphigus foliaceus: generalized or focal alopecia and seborrhoea. Seldom results in hoof and foot problems.
- Other causes of laminitis: restricted circumstances.
- Other causes of mane and tail dystrophy: breed related (e.g. Appaloosa tail syndrome), or sporadic changes for traumatic or other reasons.
- Iodism: generalized seborrhoea and naso-ocular discharges; known exposure.
- Traumatic loss of mane/tail hair either from pressure necrosis (such as bandaging injuries of the tail), pruritus (such as insect bite hypersensitivity) or by direct pulling by other horses/other animals: the circumstances are usually obvious and once the instigatory factor is removed most cases return to normal. Scarring damage may, however, be permanent.

Diagnostic confirmation

- Skin biopsy will usually reveal abnormal keratinocytes in the stratum spinosum of the epidermis and atrophic follicles without hairs.
- Changes in the hoof wall are rather non-specific but parallel bands of atrophic hoof wall of poor quality with coronary band changes are typical. This is one of the possible causes of coronary band changes (see p. 479).
- Assay of selenium in mane and tail hairs, hooves, liver and kidney.
- Affected animals usually have access to seleniferous plants.
- Heavy dietary supplementation with selenium in animals which do not require it (iatrogenic selenosis).

Treatment

Removal of the source of selenium is essential. The diet should be adjusted with a high protein supplement. Additional *d*,1-methionine (2–3 g per day) should be given orally. Feed additives such as sodium arsenate, arsenilic acid and copper supplements have been reported as treatments.

In theory at least any selenium incorporated into the hoof or hair is metabolically inert but the prognosis for an affected horse is extremely poor. Feed supplements containing selenium should only be administered when there is a proven deficiency and when routine and regular monitoring can be established. Pasture management can be an important long-term aspect – areas with high soil selenium

(resulting in abnormal selenium in normal plants) and areas containing active concentrating (seleniferous) plants should be isolated from grazing animals.

Arsenic poisoning (chronic)

Profile

Arsenic compounds in minute amounts have historically been used as skin tonics and most cases are the result of excessive use of these. However, there is little doubt that arsenic can be beneficial in producing a shiny coat and so the use of the tonics is unfortunately still practised. Chronic poisoning can also be due to repeated contact with arsenical insecticidal dips (used in some parts for cattle and sheep) or from industrial pollution. Acute arsenic poisoning has no significant cutaneous signs. It is likely that arsenic poisoning will become less prevalent as compounds are removed from medications and industrial pollution.

Clinical signs

Chronic ingestion of arsenic compounds is associated with poor body condition and long hair growth, heavy scaling and scurf. Appetite may be near normal. There is loss of body condition when rested on basic rations. Mane and tail hair of poor density and irregular poor quality is a feature. Application of arsenic salts (particularly white arsenic trioxide) was historically commonly practised in treatment of the equine sarcoid. It is extremely caustic and indiscriminate in its destruction of tissue, causing extensive necrosis and sloughing. However, it is seldom used in sufficient quantity to cause systemic or chronic cutaneous signs.

Differential diagnosis

- Cushing's disease/syndrome (pituitary adenoma): laminitis is the only common sign. The syndrome results in hirsutism and is restricted to old horses.
- Selenosis (selenium poisoning): hair loss restricted to mane and tail and some coronary band hair loss.
- Pediculosis (lice infestation): mildly pruritic, patchy hair loss giving a moth-eaten appearance; no laminitis and hoof wall implications.
- Plant poisoning (*Mimosa* spp., *Leucaena* spp.): mane and tail hair loss under exposure conditions.
- Mane and tail dystrophy: Appaloosa breed related.

Diagnostic confirmation

- History of feeding or ingestion of organic and inorganic arsenic compounds over long periods or in high dosage.
- Acute poisoning in contact horses or other species.
- Arsenic assay of hair.

Treatment

Removal of the toxic substance should be sufficient provided that the signs are cutaneous alone. Low and reducing (tapering) doses of an organic arsenical may quite rapidly reverse the horse's condition. Treatment doses must be tapered off gradually and it may take a considerable time (10–16 weeks) before the horse starts to return to normal bodily condition.

Key points: Arsenic poisoning

10 12 15 19 20 24

1. Exposure-related chronic poisoning produces progressive alopecia and a non-inflammatory exfoliative dermatosis. There are few sources of significant arsenic but fungicide-treated corn seed and environmental pollution from mining and industrial factories are possible sources.
2. Acute poisoning has no dermatological implications. Chronic poisoning results in generalized hair thinning and marked seborrhoea.
3. Diagnosis can be made from analysis of blood, hair or hoof clippings – specialized laboratories are required.
4. Treatment depends on removal of the source and then provided that the signs are limited to dermatological changes the horse will recover in due course. Specific antidotes can be used.
5. The prognosis in uncomplicated cases is fair.

Mercury poisoning

Profile

This relates mainly to application of mercurial salts to the skin as counterirritants/therapeutic blisters (e.g. red mercuric chloride) or ingestion of dressed grain containing organic mercury compounds as antifungal agents. The latter are no longer used in most parts of the world, so systemic poisoning has largely disappeared.

Clinical signs

Acute mercurial poisoning from ingested organic or inorganic mercury compounds has no significant dermatological signs. Surface application of inorganic mercury salts (used as therapeutic blisters, etc.) causes vesicle formation with ulceration and inflammation (Fig. 12.12). Scabbing and scaling follow. The effects are localized to the treated areas of the limbs.

Prolonged overzealous topical applications (or ingestion) may produce systemic effects including loss of hair from head, neck, body and finally, legs. Mane, tail, forelock and fetlock hairs are least affected. Oral ulceration and corneal opacity with ocular pain may be present.

Key points: Mercury poisoning

10 12 15 19 24

1. Rare, exposure-related heavy metal poisoning. Often organic antifungal chemicals are responsible. Accidental ingestion of topical mercurial blisters/medications can be responsible but usually there is not enough mercury to cause a serious problem. Acute (severe) poisoning has no dermatological implication.
2. Gradual loss of body hairs and then loss of the long hairs on the mane, tail and lower limb regions. Oral ulcers may follow direct contact.
3. Diagnosis is related to exposure. Confirmation can be made from hair and hoof analysis and sometimes from blood.
4. Treatment is limited in practical terms to avoidance of further exposure and symptomatic support.
5. Prognosis is fair provided there is no systemic organ failure.



Figure 12.12 Mercuric blister, applied to the skin in expectation of a therapeutic benefit on the tendons below, resulted in severe dermatitis and extensive scaling over many months.

Differential diagnosis

- Dermatophytosis (*Trichophyton* spp.): typical epidemiology and defined, roughly circular alopecic lesions that resolve spontaneously or which respond to specific treatment.
- Dermatophilosis (*Dermatophilus congolensis*): seasonally and circumstantially related crusting dermatosis over areas of skin exposed to continued wetting).
- Pediculosis (lice) (*Werneckiella* (*Damalinia*) *equi*, *Haematopinus asini*): mildly pruritic disease with patchy alopecia; parasites are easily visible in groomings.
- Trombiculidosis (*Trombicula* spp. mites).
- Occult sarcoid: long-standing alopecic hyperkeratotic skin and usually nodules palpable within the affected skin. Other types present on the horse.
- Anagen defluxion: extensive hair loss without any defined dermatological symptoms.
- Plant poisoning (*Leucaena* spp., *Mimosa* spp.): exposure related.

Diagnostic confirmation

- History of application or ingestion of mercury salts (usually some weeks previously).
- Severe/acute toxicity is unlikely to concern the dermatologist.
- Analysis of hair and hoof wall and possibly blood using a specialist forensic/toxicology laboratory.

Treatment

Affected areas should be washed with warm soapy water or shampoo. The treated areas are rinsed carefully and emollient creams and lotions containing corticosteroids, antibiotics and possibly local anaesthetics can be applied.

Ergot (ergopeptine) alkaloid poisoning

Profile

This is an extremely rare disorder resulting from the ingestion of mycotoxins (ergotoxin) derived from a fungus *Claviceps purpurea*. The toxins are largely derived from fungally contaminated feed sources such as hay and most reports emanate from the USA and involve fescue grasses (Evans et al 2003). The toxins have a wide range of clinical/dermatological effects mainly derived from their potent vasoconstrictive effects.

Key points: Ergot alkaloid poisoning

1 5 11 15 17 20 24

1. Rare, geographically restricted (USA) condition with occasional episodes in other circumstances due to contaminated feeds.
2. Clinical signs include peripheral vasoconstriction and dry gangrene. Ears, tail and feet are most affected. Laminitis can be a major early sign. The main signs are associated with abortion and inappropriate lactation.
3. Diagnosis is usually reliant on circumstantial evidence – several animals similarly affected suggest toxicity.
4. Treatment is usually too late anyway but removal of the toxic source will at least limit the effects.
5. The prognosis is guarded but provided that the structures involved are not severely damaged, the outlook should be reasonably optimistic.

Clinical signs

Localized dry (ischaemic) gangrene is the main dermatological sign. The tips of the ears, the tail and the feet are the main outward cutaneous evidence – early signs include cold extremities with poor pulses. Early ischaemic laminitis can be presented – the feet may be cold and the peripheral pulses weak rather than strong.

Abortion and premature/inappropriate lactation are also encountered.

Differential diagnosis

- Selenium/arsenic or mercury toxicity: exposure related to loss of body and mane hairs in particular. Note that some of these toxic elements are used in seed corn antifungal compounds and so the same circumstances could apply.
- Plant poisoning (*Leucaena* spp., *Mimosa* spp.): exposure related with mane and tail hair loss.
- Disseminated intravascular coagulopathy: focal cutaneous haemorrhage and skin necrosis.

Diagnostic confirmation

Circumstantial evidence – usually several horses under the same conditions are affected. Skin signs may be seen after the earlier, more dramatic signs of abortion, lactation, etc.

Treatment

Removal of the source of the toxin is critical. Usually the damage to peripheral vasculature is not recoverable by the time it is recognized.

Traumatic skin injuries

Profile

Skin injuries of all sorts are relatively common in horses. The investigation of a wound (whether superficial or

Key points: Traumatic skin injuries

10 11 12 13 15 17 24

1 Traumatic injuries and complicated healing processes are common in horse practice.
2 Clinical signs are very variable depending on the wound type, location and complications.
3 Factors that inhibit healing should be explored and eliminated. Most wounds will heal when a truly physiological wound environment is set up.
4 Diagnosis is usually reliant on circumstantial evidence.
5 The prognosis varies markedly depending on the injury, location, complications and the care that can be provided.

complicated) requires very careful investigation to allow effective and rapid healing. Aspects of wound healing are not covered in this book and the reader is guided to specific wound management texts. However, a brief summary is pertinent to the dermatologist because the skin has its own healing and repair mechanisms that can be affected by many metabolic and local factors. Similarly there are often implications deriving from wound exudates that affect adjacent skin.

Clinical signs

Bruising shows as swelling and red/purple-brown discoloration of white skin. Pigmented skin, if shaved, has a shining appearance due to the turgidity of surface layers caused by extravasation of either blood or tissue fluid.

Haematoma is initially a firm subcutaneous swelling. As the blood clots and the clot retracts, the swelling becomes more fluid/fluctuant. Fibrin can be felt to 'crackle' if the haematoma is deeply palpated and squeezed gently. There is seldom marked increase in local skin temperature, and apart from the initial insult a haematoma is not often painful on palpation.

Laceration and other damage to the skin is usually obvious, but some very small injuries have potential to be extremely serious and no wound should be treated lightly.

Cellulitis is a painful oedematous swelling of the subcutaneous and connective tissues. The area becomes hot and obviously inflamed. Serum exudate may be present, often with secondary scalding dermatitis and lymphangitis. The skin may rarely 'pit on pressure' but then quickly regains its previous outline.

Differential diagnosis

- Urticaria: pressure pits remain longer and it is usually not painful or inflamed.
- Hot abscess: more painful, localized swelling.
- Cold abscess: does not pit on pressure. Aspiration is helpful.
- Snake, spider or insect bite: severe local reaction with swelling and subsequent necrosis.

Diagnostic confirmation

- Visual appearance of skin colour and its texture are characteristic.
- Ultrasonographic examination.
- Needle aspiration.

Treatment

All skin injuries should be very carefully assessed for complicating factors which might adversely influence the healing process.

Bruising can be reduced by using cold compresses, cold hosing or ice packs. Restriction of inflammatory response in this way limits the extent of secondary damage (to nerves or muscles, etc.) or the long-term complications and scarring.

Generally, a fresh haematoma should be left to clot. Drainage is probably not advisable as there is a risk of introducing infection, but in some cases it is necessary for cosmetic as well as medical reasons. Drainage should be by wide incision to ensure that all the clotted blood can be removed. Antibiotic therapy is important.

Cellulitis is particularly difficult to treat effectively (especially if on the limbs) and requires the use of hot and cold compresses and intensive antibiotic therapy with non-steroidal anti-inflammatory medications to ease pain and encourage some movement. Some cases progress to chronic lymphangitis.

Exuberant granulation tissue (proud flesh)

Profile

'Proud flesh' is an overgrowth of normal granulation tissue which occurs particularly (but not exclusively) on lower limbs. Fresh cases may be crusted but are usually pink and moist and bleed easily if abraded. Chronic tissue is very firm, often dark red in colour and bleeds less easily than fresh granulation tissue.

Clinical signs

There is overgrowth of normal granulation tissue above skin edges and failure of tissue to epithelialize (Fig. 12.13). Infection of the surface is common, with purulent exudate which may cause scalding below the wound site. The area is seldom painful but occasional horses will bite or chew at the site (particularly if there are complicating factors such as foreign bodies and *Habronema musca* larvae) (see Fig. 9.38).

Key points: Exuberant granulation tissue

9 11 14 20 24

1 Common occurrence on horses and in particular in wounds that fail to heal appropriately.
2 Fleshy, exophytic ulcerated tissue occurring at a wound site. Often haemorrhagic with secondary infection or infestation. May develop a sero-cellular crust. Some cases have a flatter indolent nature. The epithelial margin is inhibited.
3 Diagnosis is usually uncomplicated but biopsy(or submission of all tissue excised during treatments) should be submitted to differentiate it from sarcoid and haemangiosarcoma.
4 Treatment of uncomplicated granulation tissue excess or indolence is a standard wound management procedure involving excision, grafting and topical anti-infective, anti-inflammatory and moisture-donating dressings. Before embarking on any treatment all primary factors that inhibit healing must be addressed.
5 The prognosis for wound is usually good.

Exuberant granulation may reach enormous proportions. In some cases an explanation can be found (see above for factors influencing wound healing).

Some wounds, however, fail to produce healthy granulation tissue and these usually show a hypertrophic fibrous scarring margin with a poorly vascularized and poor-quality granulation tissue (Fig. 12.13). These will fail to epithelialize until a suitable wound bed is created. Indolent wounds are most often encountered on the dorsum or plantar regions of the hock (Fig. 12.14). Here the wound fails to produce any significant granulation tissue and is most often covered by a flaking thin layer of fibrin and purulent exudate. There is usually no evidence of epithelialization at all or it is very poor and has a hypertrophic appearance, giving the margin of the wound a piled-up appearance.



Figure 12.13 This wound shows an indolent non-healing margin with no proper attempt at healing the wound.



Figure 12.14 An indolent wound which failed to show any significant granulation tissue or any epithelialization for several months following an innocuous-looking cut.

Differential diagnosis

- Fibroblastic and malevolent sarcoid: can be very similar but usually some other sarcoid types are present; biopsy is diagnostic.
- Botryomycosis (fibrogranuloma): detectable staphylococci and typical microabscessation detected in biopsy.
- Squamous cell carcinoma: ulcerative/destructive neoplasm with typical histology.
- Foreign body: history and ultrasonographic/radiographic detection.
- Habronemiasis: seasonally and geographically related parasitic condition that responds to topical anti-parasitic treatment. Self-resolving at end of fly season.
- Pythiosis: geographically restricted fungal wound complication.

Diagnostic confirmation

- Clinical appearance is characteristic.
- Biopsy of several sites should be taken to ensure differentiation from fibroblastic sarcoids, botryomycosis and other proliferative masses arising secondarily to parasitic infestations in particular.
- Radiographic, ultrasonographic and vascular perfusion studies with laser flow Doppler may be performed to establish possible aetiological factors.

Treatment

Management of exuberant granulation tissue *must* always be preceded by a full investigation of the possible causes: without addressing the underlying problem, treatment is likely to fail. Under no circumstances should caustic materials (including copper sulphate, sulphuric acid, nitric acid, mercuric chloride, etc.) be applied to the tissues. While the initial effects may appear to be beneficial in reducing the bulk of the tissue, the long-term consequences are very harmful.

Medical management of exuberant granulation tissue should be directed towards reducing the extent of chronic inflammation. Thus, application of corticosteroid creams and non-steroidal anti-inflammatory drugs is logical. However, they will also retard epithelialization and so may ultimately be of less value. They are indicated in the early management of granulation tissue prior to grafting but should not be applied for at least 6–7 days prior to grafting.

Surgical trimming of the exuberant tissue and careful management to produce a healthy granulating bed of well-vascularized tissue level with the epithelial margin should be followed as soon as practicable by suitable grafting techniques. The best granulation tissue quality is obtained by using a full moist wound-healing technique with application of hydrogels and hydrocolloids or collagen-based wound dressings. The best cosmetic effects are obtained with the use of split skin thickness grafts over the whole wound surface (with meshing techniques to enhance adhesion and graft take and minimize the donor site deficit), but good effects can also be obtained by the simpler pinch, punch or tunnel grafting methods which may not require general anaesthesia and sophisticated skin harvesting apparatus.

Burn/thermal/scald injuries

Profile

Burns include thermal, friction, freeze and chemical damage to skin.

Where escape is not possible, extensive body burns are encountered and life-threatening burns are common in stable fires. Burns are classified in terms of the depth of the burn and the extent of skin affected. The depth of burns is usually classified in three degrees.

- *First-degree burns:* erythema, pain and localized, transient oedema.
- *Second-degree burns:* vesicle formation, necrosis and sloughing of the epidermis with exposure of deeper dermal tissues.
- *Third-degree burns:* destruction of cells and blood vessels in the skin and hair follicles. Loss of cutaneous sensation. Sloughing and separation of all layers of the skin.

As the skin of horses has a much reduced tendency to blister (in comparison to the human), this classification is somewhat artificial. In horses, first- and second-degree burns are probably best grouped together as partial-thickness burns and the third-degree burns as full-thickness burns.

The extent of the burn can also be described in terms of the percentage of total body surface which has been involved. Burns over greater than 10–15% of the body surface carry a particularly poor prognosis.

Accidental or deliberate application of heat to the skin causes burns. The severity of clinical signs varies with the length of contact time and the temperature. Hot branding and 'pin' and 'line firing' are the commonest iatrogenic causes of this type of burn.

Friction burns occur from rope and other materials and in these the clinical signs may take some days/weeks to appear.

Freeze burns are used to produce identification freeze brands which are visible in grey or cream horses (an identifiable scar is produced). Lesser skin injury is used in horses with brown or chestnut coats and here the 'burn' induces a change in melanocytes without scarring. Surprisingly, frost bite is particularly unusual in horses even under very adverse cold conditions.

Clinical signs

The initial injury almost always results in skin blistering and a progressive vasculitis, bruising, oedema and exudation. The latter can be very severe even from limited areas of skin damage. Clinically significant protein loss is common. Superficial blistering with hair loss (singeing) is the mildest consequence. More serious burns cause loss of hair and sloughing of the superficial layers of the dermis (or deeper in case of more severe burns) occurs, leaving large skin deficits (Fig. 12.15). Scalding injury may initially show various degrees of blistering without hair loss. The full extent of a burn (however it is caused) is not always easy to establish at the first examination. Although a great deal of heat has to be applied to cause damage, once it is present, the heat dissipates slowly and so the extent of damage can be much greater than is first suspected. Progressive loss of hair, vasculitis, bruising and oedema of the area precede exudation (Fig. 12.15). Death of the epidermis and dermis leads to drying of the skin and/or skin sloughing or the formation of an eschar (an extensive dry, leathery plaque of necrotic skin).

Sloughing of the skin leaves a raw, bleeding, ulcerated area with extensive exudation of plasma. Pathologically significant (and detectable) protein loss can occur with even minor burn injuries. Full-depth burns heal very slowly with extensive scarring and local tissue distortions.

Second- and third-degree burns covering over 10–15% of the body surface are almost impossible to manage. Burns to the limbs (as occurs in grass fires) are also very difficult and commonly fail to heal acceptably within a reasonable time.

A potentially dangerous skin burn can arise in foals which are recumbent on electrically heated pads. Foals in

Key points: Burn/thermal/scald injuries

10 11 12 13 14 17 19 24

1. A variety of burn types occurs ranging from localized 'rope burns' to extensive areas of thermally damaged skin and other structures such as the eyes, hoof capsule and limb tendons, ligaments and joints. The degree of burn (i.e. the depth of the damage) and the extent are major factors. Thermal friction, freeze and chemical (including acid/alkali) burns occur.
2. Local and widespread burns are always serious and warrant very intensive management to remove all necrotic tissue and encourage healing. Replacement of plasma losses may be involved. Smoke inhalation and airway burns can be life-threatening within hours.
3. Diagnosis is usually simple.
4. Treatment of burns is a specialized matter and may involve intensive care and specialized procedures such as grafting.
5. The prognosis depends largely on the location, degree and extent of the burn.



Figure 12.15 Thermal burn showing extensive blistering and sloughing of the skin. Plasma loss can be very significant and such wounds can take a long time to resolve. Skin grafting is often needed.

intensive care often have complications of compromised blood pressure and the skin in contact with the pad can easily be burned even if the pad is thermostatically controlled. The true extent of the damage can take days or even weeks to develop (or become apparent).

Iatrogenic burns such as 'pin' and 'line' firing (Fig. 12.16) and hot branding follow a predictable course with initial acute inflammation accompanied by pain and gradual healing with scar formation. The 'firing' of horses for musculoskeletal disease has disputed therapeutic benefits but the chronological course of the cutaneous repair mechanism is predictable. The scar is permanent but its ultimate extent often belies the extent of the initial insult as contraction of the wound site makes the scar smaller than might be expected.

Differential diagnosis

- Chemical burns (from over-strength therapeutics or errors of application): history of application and localization to area concerned, possibly with run-off patterns.
- Iatrogenic spirit or antiseptic contact (especially in anaesthetized horses and those with compromised blood pressure): history of circumstances.
- Decubitus (pressure) sores (recumbent horses): history of recumbency.
- Pemphigus vulgaris (bullous pemphigoid): diffuse hair loss and epidermal scaling. Insidious in onset and chronic progressive course. Little/no skin necrosis.
- Generalized dermatophilosis (*Dermatophilus congolensis*): seasonally related, characteristic distribution of localized or areas of dermatitis.
- Arsenic or mercury poisoning/caustic effects: areas of necrosed skin and subcutaneous tissue corresponding to applications of known chemical materials.



Figure 12.16 Iatrogenic thermal burns caused by a line firing iron used in an attempt to treat a sprained tendon. The eventual scars were insignificant.

Diagnostic confirmation

A history of burn or scalding contact is usually available.

Treatment

Emergency measures should be taken to save the life first and to relieve pain. Immediate cold water lavage for 20–30 minutes after the burn helps to reduce the extent of scarring and damage. This has a marked effect on the future healing and scarring. Wrapping or covering the burnt area in domestic cling-film is helpful in reducing the immediate extent of plasma/serum loss and controlling shock. Immediate fluid therapy and plasma should be given if indicated. Protection of the burn site can also be provided by silver sulfadiazine cream.

Note

Wiping the site with gauze swabs, etc. and application of grease-based emollient creams or lotions should NOT be applied – they retain heat and may exacerbate the problem.

Partial-thickness burns usually heal well, but deeper burns require intensive therapy to debride all dead tissue and encourage healing. Sustained non-steroidal anti-inflammatory drugs such as phenylbutazone and antibiotics may be required. Self-trauma can be severe and so cross-ties and cradles may be needed.

Scarring is often extensive and the wounds may require cosmetic surgery to speed closure and limit the physical problems associated with prolonged healing.

Pressure necrosis, rope/wire burns/bandage necrosis

Profile

Rope burns and pressure necrosis are common consequences of tethering or other methods of restraint. Pressure

Key points: Pressure necrosis, rope/wire burns/bandage necrosis

10 11 14 17 24

1. Pressure necrosis of skin results in an area of dead skin known as an eschar. Overtight bandaging of the legs and tail is regrettably a relatively common event resulting in severe skin necrosis as a result of ischaemic necrosis. Decubitus ulcers result from prolonged contact with a hard surface in recumbent horses and are particularly bad when the blood pressure is low.
2. Clinical signs may be slow to develop but localized pain is usually significant. – the first signs on the legs may be lameness and hair loss or even just easily dislodged hair. Often the skin necrosis becomes an eschar – a hard black cardboard-like plaque of dead skin.
3. Diagnosis is sometimes difficult in the early stages.
4. Treatment involves protecting the area from further damage and managing the eschar until it sloughs naturally. Surgical removal of necrotic skin can be helpful in some circumstances.
5. Prognosis is variable.

necrosis (decubitus ulcers) results from prolonged recumbency or from destruction of the skin blood supply from applied pressure (bandages, plaster casts and other over-restrictive equipment). Pressure wounds (decubitus) can be particularly severe in horses with low blood pressure and sometimes severe damage can occur even with chemical contact such as surgical skin preparations. In recumbent hypothermic foals with low blood pressure even water bottles and electric blankets can cause severe skin necrosis. Foals are particularly liable to skin injury from pressure because of the relatively thin, delicate skin and concurrent debilitating diseases.

Clinical signs

Rope burns are commonly encountered on the distal limb and initially cause hair loss and exudation. The extent of skin damage is variable with the type of rope, diameter and extent of rub/contact as well as the pressure applied to it. Recurrent injury tends to induce chronic thickening and extensive scar formation with extensive, often movement-limiting scarring.

Pressure sores (decubitus ulcers) arise from interference to blood supply and subsequent necrosis of the overlying skin during prolonged recumbency. Some decubitus/pressure wounds are slow to appear. They may show an area of pain, heat and swelling some days or weeks before the skin is obviously identifiable as dead.

Bandaging injuries are common on the distal limb and vary widely in extent from mild surface epidermal damage and erythema to extensive necrosis involving the underlying structures (Fig. 12.17). Commonly the dead overlying skin forms an eschar (sitfast) of dry, leathery, coagulated skin (Fig. 12.18). In some cases the underlying tissue is severely compromised and may serve to delay healing still further. The secondary consequences of this type of injury are often even more serious than the prolonged healing and extensive scarring which are characteristic of the condition. Healing by granulation and eventual epithelialization is particularly slow. Scarring in either case can be severe and may be limiting to function.

Differential diagnosis

- Dermatophilosis: seasonally and environmentally related, generalized or focal skin disease relating to skin wetting; usually affects the back, lower limbs and head. *D. congolensis* is easily identified.
- Greasy heel syndrome: restricted to the limbs and usually to white-skinned legs. Diffuse verrucose dermatitis with chronic nodular dermal hyperplasia in long-standing cases.
- Mercurial blisters/counterirritants: history of application.
- Pemphigus foliaceus: diffuse or focal superficial, exfoliative dermatosis with characteristic histology.
- Vasculitis: focal ischaemia or generalized thrombosing dermatosis, often with severe oedema and possibly petechiation.
- Thrombosis (particularly if arterial)/thrombophlebitis/gangrene: loss of arterial pulse distally; cold tissues.
- Pastern and cannon leukocytoclastic vasculitis: regionally restricted and usually in pastured horses. Lateral aspects of white-skinned limbs affected.
- Chemical burns: history of contact/application.

Diagnostic confirmation

- Clinical examination and history are almost always clear.
- Overtight bandages are commonly involved in limb pressure necrosis wounds.

Treatment

Restoration of blood supply by hot and cold compresses and local application of soothing emollient creams is required. Normal wound management procedures should be followed to encourage rapid healing, but such wounds



Figure 12.17 Dermal ischaemia caused by bandaging of this difficult site. The exposed fibres of the Achilles tendon were necrotic and the wound only healed after a prolonged nursing period during which no further bandages were used.



Figure 12.18 Cutaneous ischaemia caused by overtight bandages applied for an unrelated problem resulted in the formation of an eschar. The black necrotic area of dried leathery skin can be seen on the wound.

are often very refractory to healing and become a chronic problem. Surgical debridement and grafting may speed the repair and limit the scarring.

Scar

Profile

Scarring is the inevitable result of full skin thickness injury. The character of the scar can vary widely. Scarring is minimal in wounds which heal by primary intention (e.g. surgical wounds), but prolonged healing results in larger scars. Hypertrophic or cheloid scars are the result of abnormal cell behaviour in the healing process.

The importance of scarring lies in its similarity to some significant dermatological disorders and the functional restrictions that they may cause. Although scars often cause concern to owners, they are of course of no cosmetic consequence to the horse.

Key points: Scar

10 12 13 14 20 24

1. Scarring is very common and an inevitable consequence of injury but is seldom more than a cosmetic nuisance to an owner. Scarring results whenever the dermis is injured and obviously varies in extent. Scarring is worse when the skin is abnormal and when a chronic wound is present.
2. Signs of scarring vary from mild hair loss and changes in colour to extensive skin and local tissue destruction. Functional deficits are possible especially if contraction is severe in regions with a need for flexibility such as the eyelid, over joints or around the mouth and nostrils.
3. Diagnosis is usually easy but some cases can be mistaken for sarcoid or other conditions that result in hair loss and skin distortion.
4. Treatment of scars is theoretically possible but often the results of interference are worse than the original scar. However, silicone hydrating sheets and moisturizing creams are useful in some cases. Scarring should be minimized by careful wound management and encouragement of rapid wound healing.
5. The prognosis is variable depending on the secondary effects. Functional limitation can be serious.

Clinical signs

Scarring is minimal in surgical wounds which heal by first intention – the site of a Mackay–Marks laryngeal prosthesis or a digital neurectomy, for example, may be impossible to feel and may be visible only with difficulty in the clipped/shaved skin (Fig. 12.19). Some injuries which heal by secondary intention may also leave a very small scar and may be difficult to identify, e.g. the site of a lateral ventriculectomy (Hobday) incision. The latter is usually the case where wound contraction is most effective, i.e. on the body and head/neck regions. Scarring on the limbs associated with wounds which heal by secondary intention in particular is often severe and reflects the early transition of the wound to a state of chronic inflammation. Prolonged healing on the distal limb is frequently associated with exuberant granulation tissue or an indolent non-granulating, non-healing wound (see Fig. 12.14).

Most scars show loss of hair (absence of hair follicles) and presence of modified skin. The area is fibrosed and dense with limited elasticity and often with an altered hair growth pattern (Fig. 12.20).

Some scars induce leukoderma and/or leukotrichia (e.g. saddle marks and girth marks) (see Fig. 16.7). Freeze branding causes alteration in hair follicle structure and loss of melanocytes without significant scar, while hot branding or repeated freeze brands cause hair loss and scarring. Normal scar tissue contracts markedly over years.

Hypertrophic scarring is an uncommon potential problem of wound healing in the horse. Here the ultimate size and depth of the scar are inappropriate to the original size and severity of the wound (Fig. 12.21). A relatively small



Figure 12.19 A surgical scar at a palmar digital neurectomy site showing minimal scar formation (arrow) typical of first-intention healing. Such scars are often very difficult to detect.



Figure 12.20 Dense fibrotic scarring typical of a slow healing wound.

injury can sometimes result in an extensive scar which can have serious effects on the adjacent tissue. There are few circumstances when such a response can be predicted.

Cheloid scar occurs when the character of the scar is irregular and the surface is roughened, giving a reptilian look to the wound site (Fig. 12.22). Again there are no predictable types or location of a wound which predispose to the problem.



Figure 12.21 Hypertrophic scar developing at the site of a small surgical wound some 12 months previously which appeared at first to heal normally.



Figure 12.22 Cheloid scar with a typical reptilian appearance.

Differential diagnosis

- Sarcoid (verrucose, fibroblastic and mixed forms).
- Recurrent abrasions from harness or other conditions.
- Self-inflicted bites.
- Thermal injury/burns and scalds.
- Frost bite.
- Chemical injury.

Diagnostic confirmation

- Clinical history of wounds is almost always present. Some result from application of strong chemicals, blisters, insecticides, hot or cold thermal injuries.
- Biopsy shows absence of hair follicles and structureless hyaline connective tissue. Hypertrophic scars are densely packed with fibrocytes which have little or no contractility and so these scars do not alter with time. Cheloid scars have dense granulation tissue with a rough irregular epithelial surface which produces excessive and irregular hyperkeratosis.

Treatment

Scarring can be minimized by careful wound management directed at reducing the extent of chronic inflammation. The longer a wound takes to heal, the more extensive is likely to be the scarring. Scars may be surgically removed, but there is a risk of further (sometimes worse) scarring after surgery. Daily application of keratolytic agents followed by vitamin E cream may soften the skin and allow contraction of the scar. Where scarring causes limiting distortion of tissue such as the eyelids or over joints, skin grafting and other reconstructive measures may be applicable.

Bursitis

Profile

Bursitis is an inflammation of an internal or external bursa with a detectable change in size and character. Bursae are natural cushions that overlie bony prominences, joints or tendons. False or acquired bursae can develop at some sites

Key points: Bursitis

9 11 14 17 20 24

1. Inflammation of a natural bursa either by infection or by direct trauma is rare in horses. Open wounds involving the calcanean bursa or the bicipital bursa are very serious. Poll evil, fistulous withers associated with *Brucella* spp. Infections and with 'shoe boil' (elbow bursal trauma/infection) are historically important conditions that seldom occur now. *Onchocerca cervicalis* infection in donkeys in Saharan Africa is a common cause of withers fistulas.
2. Signs of lameness, heat, pain and, in open bursae, a discharge or swelling localized to the bursa can be demonstrated ultrasonographically.
3. Diagnosis is made on clinical features and with ultrasonography.
4. Treatment involves drainage and control of infection. Bursae seldom heal on their own and the whole healing process can be very prolonged. Total ablation of a bursa may be possible.
5. The prognosis is very guarded.

of persistent blunt trauma such as the point of the elbow. Inflammation is usually caused by acute or chronic/recurrent trauma. Chronic trauma over bony prominences or even a single acute event may result in an acquired bursa such as a 'capped' elbow or hock. Although many cases are not infected ('capped hock' is a traumatic bursitis of the calcanean bursa), infection is a serious problem in some circumstances. Historically poll evil (bursitis of the poll bursa) and fistulous withers (bursitis of the withers bursa) were regarded as being caused by *Brucella* spp. bacteria. However, other causes have been identified including *Onchocerca cervicalis* (see p. 221). Excessive (abnormal) secretion of fluid of a non-inflammatory or inflammatory nature occurs within the bursa and the expansion shows as a cutaneous nodule or a diffuse painful subcutaneous swelling.

Clinical signs

Enlarged soft (or occasionally firm) fluctuant swelling in an anatomical area where natural or acquired bursae occur such as the hock (Fig. 12.23) or the withers (Fig. 12.24) and the poll. They are usually distinctive, being of body warmth and non-painful. Once they become infected and can discharge onto the body surface they create serious problems both for the surgeon (in treatment of the disorder) and for the dermatologist (in the management of the secondary scalding dermatitis which is the consequence of profuse discharges running over the adjacent skin) (see p. 301).

Differential diagnosis

- Inclusion cysts (atheroma): non-inflammatory firm but fluid swelling above the false nostril. Slow expansion or static.
- Haematoma: acute onset of a fluid-filled swelling in areas without bursae; aspiration reveals blood or organizing blood clots. Ultrasonography is very distinctive.
- Calcinosis circumscripta: localized, very firm, non-painful, non-inflammatory calcified swelling usually on the lateral aspect of the stifle/gaskin.
- Enlarged joint capsule: synovial fluid accumulation in an anatomically distinctive location.
- Abscess: hot painful progressive swelling with a focus 'pointing', containing pus or sterile inflammatory debris. Ultrasonography and aspiration are characteristic.
- Melanoma: distinctive firm black nodules in grey horses with characteristic black tarry aspirate.
- Nodular sarcoid: not associated with bursal sites and usually multiple forms are present. Nodules are firm/very firm and often there is dermal involvement.
- Tendon sheath fibrous enlargement.

Diagnostic confirmation

- Needle aspiration for analysis of content leads to local collapse unassociated with joint.
- Ultrasonography is a useful way of establishing the true nature of the structure, its contents and its anatomical relations.

Treatment

Treatment of non-infected enlarged bursae is often unrewarding/disappointing, with rapid recurrences and possibility of infection following attempts to aspirate contents and injection of corticosteroids. If the bursa is not infected and sterility can be assured, an attempt can be made to drain the bursa followed by intrabursal injection of corticosteroids and pressure bandaging. Recently there has been some suggestion that very small doses of atropine injected with or without corticosteroids have some benefit. This has not yet been critically evaluated. Surgical removal of the whole structure is very difficult and often leaves a defect larger than the original. Most acquired, non-infected bursae are therefore best left as cosmetic blemishes.

Infected bursae (such as 'poll evil' and 'fistulous withers') create serious problems and extensive surgical interference is usually required. Many of the cases presented as fistulous withers do not, however, involve the bursa and are

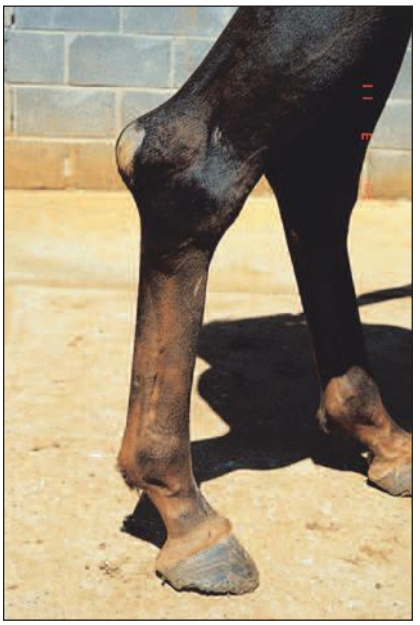


Figure 12.23 Bursitis of the hock bursa ('capped hock').



Figure 12.24 Infected bursitis of the withers bursa ('fistulous withers').

the result of extensive subcutaneous tracking of infection from injury to underlying bone or ligaments. The clinical problem can still be serious and similar extensive surgical debridement is required.

References

Evans T J, Rottinghaus G E, Casteel S W 2003 Ergopeptine alkaloid toxicosis in horses. In: Robinson N E (ed) Current therapy in equine medicine. WB Saunders, Philadelphia, pp 796–798

McKenzie R A 1994 Plant poisoning in horses in Australia. Proceedings of the 16th Bain Fallon Memorial Lectures. Australian Veterinary Association, pp 24–29

Mattocks A R, Jukes R 1991 The identification of hepatotoxic pyrrolizidine alkaloid exposure in horses by the demonstration of sulphur-bound pyrrolic metabolites on their haemoglobin. Veterinary and Human Toxicology 33:286–287

Mattocks A R, Jukes R 1992 Detection of sulphur-conjugated pyrrolic metabolites in blood and fresh or fixed liver tissue from rats given a variety of toxic pyrrolizidine alkaloids. Toxicology Letters 63:47–55

Raisbeck M 2003 Selenium toxicity. In: Robinson N E (ed) Current therapy in equine medicine. WB Saunders, Philadelphia, pp 801–803

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13 Endocrine disorders

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Hypothyroidism

Profile

Confirmed cases of hypothyroidism are very rare. Although thyroid dysfunction has been commonly referred to in texts and there is a copious literature on equine hypothyroidism, the evidence for its existence in adult horses is tenuous. The clinical syndromes attributed to it are based only on experimental cases in which both thyroid glands have been removed (Hillyer & Taylor 1997).

Many of the horses in which hypothyroidism is diagnosed have a metabolic syndrome that is now well recognized. There is no convincing evidence that thyroid deficiencies cause laminitis, fertility problems, reduced performance, anaemia or anhidrosis (see p. 344). There are, likewise, few convincing cases of skin disease associated with adult onset hypothyroidism; only a few cases have been reported in adult horses (Hillyer & Taylor 1992). Bilaterally symmetrical alopecia occurred in one case (Shearer 1988).

Foals born in areas where iodine deficiency occurs have been reported to have signs that can be directly attributed to hyperiodic hypothyroidism and this syndrome is well recognized (Irvine & Evans 1977).

Key points: Hypothyroidism

3 10 15 19 20 24

1. Many metabolic derangements are blamed on thyroid gland dysfunction. In *adult* horses hypothyroidism is extremely rare and any cutaneous signs are vague and ill defined. In foals born to mares with an abnormally high iodine intake, severe musculoskeletal deformities occur. These may also show skin signs.
2. In foals severe musculoskeletal deformities and goitre and sparse, silky soft (almost premature) velvet coat are present. The hoof may be abnormally soft and rubbery. There may be some seborrhoea. The dermatological signs are usually irrelevant. Adult horses could present as bilaterally symmetrical alopecia that responds to thyroid supplementation.
3. Diagnosis in foals is based on the presence of a visible/palpable goitre and musculoskeletal deformities. By the time the foal is 24 hours old dynamic or static thyroid hormone tests are probably of no value. In adult horses dynamic thyroid function tests can be used but interpretation is highly problematic.
4. Treatment of foals is ineffective – supplemental thyroid hormones do nothing to help. In adults the effects of even high doses of thyroid replacement therapy are almost imperceptible.
5. The prognosis for foals is hopeless although mildly affected foals may survive. In adults the outlook is guarded simply because nothing much is known about the condition.

Note

Hyperthyroidism is probably even less common and no skin signs are likely in any case.

Clinical signs

Foals Foals are born with major musculoskeletal deformities and an obviously enlarged thyroid (goitre) may or may not occur. The hair is often sparse and soft, giving it a velvet feel. A more abnormal hair coat develops as the foal hair is shed. The skin may be thickened by myxoedema and there may be seborrhoea. Leg deformities (angular and flexural as well as arthrogryposis) may also occur in foals.

Adults Roughly symmetrical, patchy or diffuse hair loss may occur over the head, neck, forelimbs and hindquarters. The underlying skin is pigmented and the hair may be white in places. No pruritus or ulceration is reported.

Diagnostic confirmation

- Abnormally low levels of plasma T_3 and T_4.
- Confirmation is difficult as histopathology usually shows normal skin structure, but increased hair follicles and decreased sebaceous glands may be identified in the affected area. Hair shafts may be filled with irregular keratinized material.

Treatment

Oral L-triiodothyronine (4 mg/kg bodyweight) is given daily for at least 10 days. Treatment course may require months or years or may show no response at all.

Pituitary pars intermedia dysfunction (PPID; equine Cushing's disease)

Profile

Equine pituitary pars intermedia dysfunction (PPID/Cushing's disease) is almost always due to the very common hyperplasia of the pars intermedia of the neurohypophysis. It occurs commonly in horses and ponies over 12–15 years of age. The condition is very rare in animals under 7–10 years of age.

It is likely that the pathophysiology relates to a brainstem degeneration and a consequent loss of inhibitory control on the pars intermedia. There is a reduction in dopamine secretion which normally inhibits the brainstem melanotropes. The pars intermedia gradually undergoes functional hypertrophy with increasing release of adrenocorticotropin. This in turn results in hypersecretion from the adrenal cortex. Excessive endogenous cortisol has profound and widespread effects on vascular endothelia, glucose and fat metabolism, immune responses, wound healing and endocrine secretions. These effects are used to explain the laminitis, fat redistribution, weight loss, immunosuppression, hyperglycaemia and polydipsia. This is a plausible (if oversimplified) course of events to explain the corticosteroid-related abnormalities but it does not really explain all the clinical signs and the behavioural changes, blindness, seizures and hirsutism in particular. These may be attributable to brainstem alterations and to the space-occupying nature of the hypertrophic pituitary gland in the brainstem and hypothalamus in particular.

No breed, sex or colour predisposition has been established, but ponies and geldings may be over-represented (possibly because they are commonly kept to older ages than larger horses). Tumours may be very small (almost invisible) but functional while others may be large enough to cause physical effects but have little physiological effect (Hillyer et al 1992).

Clinical signs (Table 13.1)

The early signs of polyuria and polydipsia may be difficult to detect and they are often missed by owners unless they are asked specifically about them.

Often the earliest reported evidence is a failure of hair coat shedding which is commonly ascribed to 'old age'.

One or more episodes of laminitis which has characteristic features that are somewhat different from the 'normal' pony laminitis are also commonly reported in the early stages. The laminitis is characterized by a failure to respond to non-steroidal drugs and may have a lesser digital pulse and lesser hoof inflammation than might be expected from the severity of the condition.

Cutaneous signs include:

- Hirsutism (failure to shed hair, rapid regrowth of curly, longer than normal hair (8–10 cm long) with a greatly increased density and patchy areas of longer hair (Fig. 13.1). The mane and tail are almost never affected.
- Sweating (hyperhidrosis) giving the coat a clammy feel and soaked appearance (Fig. 13.2).
- Secondary bacterial cutaneous infections with *Staphylococcus* spp. and *Dermatophilus congolensis* are common.
- Parasitism (including ectoparasites such as lice or mites) is common.
- Oral ulceration which shows no evidence of healing (Fig. 13.3) and oral and cutaneous abscessation.

Other body system signs include:

- Periodontal disease/loose teeth.
- Laminitis (recurrent, mild to severe) with lesser digital inflammatory response than might be expected from the severity of signs.

Key points: Pituitary pars intermedia dysfunction

4 15+ 15 19 24

1. A very common geriatric condition of horses over the age of 15 years resulting from hyperplasia of the pars intermedia of the pituitary gland.
2. Clinical signs reflect a wide range of neurological and endocrine abnormalities and include hirsutism, weight loss, sweating, laminitis and fat redistribution. Impaired immunity results in opportunistic skin infections including bacterial dermatitis and abscessation, fungal infections and ectoparasites (mites and lice) and endoparasites.
3. Diagnosis can be confirmed using dynamic endocrine tests or cortisol/insulin concentrations and urinary excretion ratios. Laboratory tests confirm hyperglycaemia, hypercortisolaemia and often mild increases in tissue/organ enzymes.
4. Treatment is palliative and centred around anti-serotonin or dopaminergic compounds administered orally. Direct suppression of the adrenal gland is not usually effective. Nursing measures such as careful dental care, clipping of the coat and routine farriery and anthelmintic treatment are essential.
5. The prognosis is guarded but for those horses with few major complications such as laminitis and severe weight loss, the outlook is quite good. Survival for some years can be expected if medication can be sustained.

Table 13.1 The relative frequencies of the major clinical signs described in studies involving animals with a confirmed diagnosis of equine Cushing's disease (Schott 1992)

Clinical sign	Frequency (%)
Hair coat changes	94
Weight loss	88
Lethargy	82
Laminitis	82
Polydipsia/polyuria	76
Sweating	59
Tachypnoea	41
Tachycardia	29
Skin infections	24
Sinusitis	18
Oral ulceration	18
Bulging supraorbital fat	18
Neurological signs	6



Figure 13.1 Pituitary hirsutism. Pituitary pars intermedia dysfunction (PPID) (pituitary adenoma/Cushing's disease) is the only cause of significant acquired hirsutism in the horse.

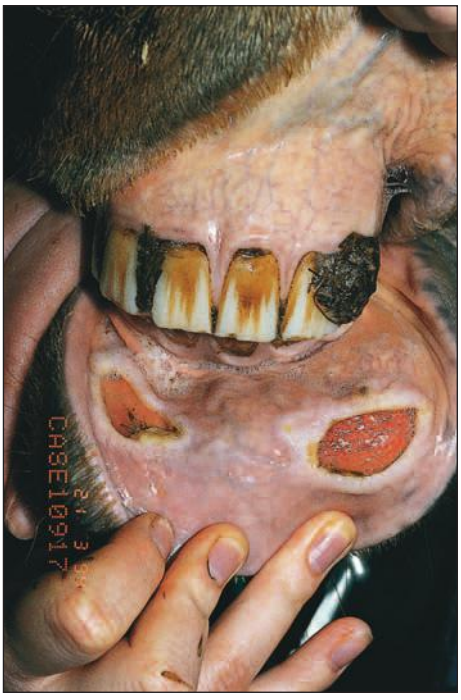


Figure 13.3 Pituitary adenoma/Cushing's disease. Oral ulceration showing a singular lack of healing is a common sign. Other dental and oral lesions may be present also.

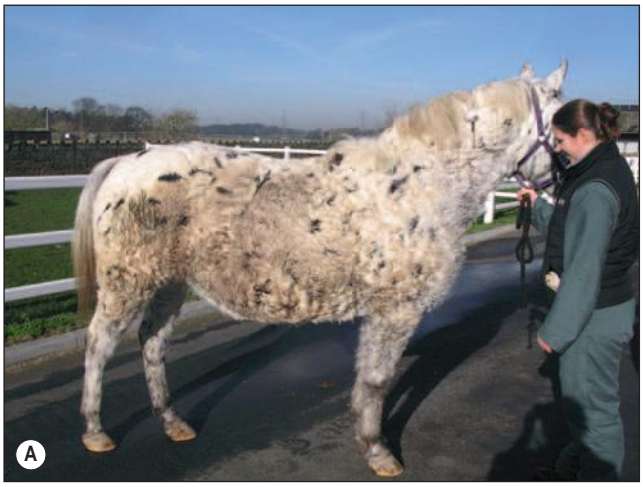




Figure 13.2 Pituitary adenoma/Cushing's disease showing hirsutism and sweating giving the coat a soaked appearance **(A)**. Secondary skin infections and infestations are common. Note the dense curly coat also **(B)**.

- Intestinal parasitism (even ascarids can be present in large numbers).
- Cataract and/or central blindness (amaurosis) (resulting from direct pressure on the optic nerves).
- Supraorbital swellings and a pendulous abdomen (possibly the result of fat redistribution).

Systemic signs include:

- Polydipsia and polyuria (PD/PU) associated with glycosuria/hyperglycaemia.
- Weight loss and loss of muscle tone ('sway back' appearance).
- Immunosuppression with recurrent internal and external bacterial, fungal and parasitic infections.
- Intermittent or persistent pyrexia.

Differential diagnosis

- Congenital curly coat syndromes: horses with curly (sometimes longer) coats from birth; specific breeds.
- Seborrhoea: scaling and flaking disorders.
- Diabetes mellitus: extremely rare and not accompanied by hirsutism or cortisol alterations in the blood biochemistry.
- Diabetes insipidus: renal or central polydipsia without hair coat changes or hyperglycaemia/glycosuria.
- Renal failure: weight loss, azotaemia and uraemia and hypoproteinaemia.
- Other causes of laminitis: food intoxication, pasture-related laminitis, trauma-related laminitis, weight-bearing laminitis/metabolic syndromes/insulin resistance.
- Other causes of hyperhidrosis (including phaeochromocytoma – functional tumour of the adrenal medulla).

- Other causes of immunosuppression (lymphosarcoma complex usually): no hirsutism and not the preserve of older horses.

Note

True pancreatic islet-related diabetes mellitus is extremely rare; most cases showing significant hyperglycaemia/glycosuria and polyuria/ polydipsia can be safely assumed to have PPID, and particularly when at least one other characteristic sign is present.

Diagnostic confirmation

- Classical clinical signs.
  - Pituitary pars intermedia dysfunction (equine Cushing's disease/pituitary adenoma) is the only clinical condition known to cause acquired hirsutism in the horse.
- Hyperglycaemia (fasting blood concentrations of 8–16.75 mmol/L are consistent with the presence of the disorder) (Beech & Garcia 1991) but can also be associated with insulin resistance in non-cushingoid horses.
- Diagnosis can be confirmed by:
  - Adrenocorticotropic hormone (ACTH) stimulation test (Hillyer et al 1992).
  - Dexamethasone suppression test (Beech 1987) (or a combination of this with ACTH stimulation test; Hillyer et al 1992, Knottenbelt 1997) (Fig. 13.4A, B).
  - Thyrotropin-releasing hormone response (Fig. 13.4C) (Beech & Garcia 1985).
  - Blood cortisol concentrations; normal blood cortisol concentrations will vary from 55 nmol/L to 125 nmol/L with a strong diurnal rhythm. PPID horses will often have gross elevations of resting levels and a significant loss of diurnal rhythm.
  - Blood/urinary cortisol excretion ratios. Urinary excretion ratio is calculated from the concentrations of cortisol in the blood and urine obtained at the same time. PPID horses commonly show an inordinately high excretion ratio, i.e. they are excreting significant amounts of cortisol in the urine (this should be minimal in normal horses).
  - Insulin assay. Normal insulin concentrations around 5–36 μIU/mL. In PPID horses' concentrations are usually elevated to around 5–120 μIU/mL. This parameter can be affected also by insulin resistance (equine metabolic syndrome).

Table 13.2 Clinicopathological findings in horses with Cushing's syndrome due to pituitary adenoma (Hillyer et al 1992)

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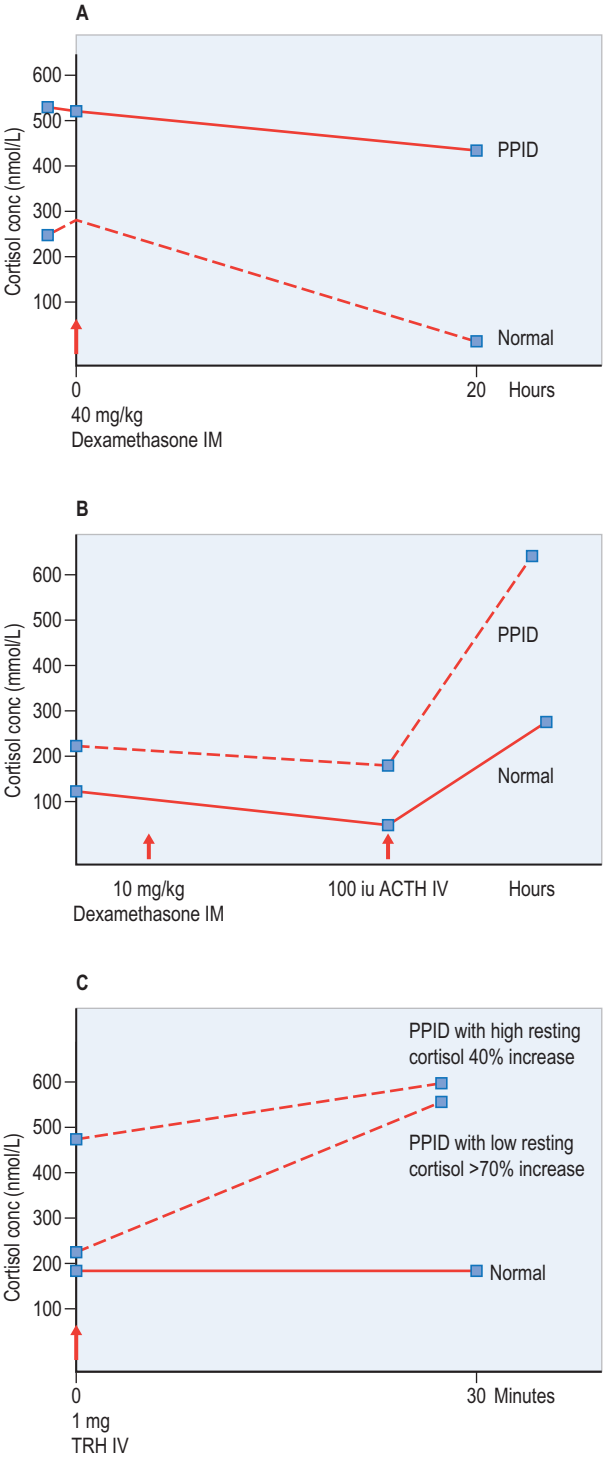


Figure 13.4 Pituitary pars intermedia dysfunction (PPID): diagnosis. **(A)** Dexamethasone suppression test. **(B)** Dexamethasone suppression test/ACTH stimulation test. **(C)** Thyrotropin-releasing hormone (TRH) response test.

Note

The technicalities of the diagnostic tests are to be found in laboratory and other medical texts.

Currently there is no practical method of imaging the pituitary gland in a live horse and so definitive confirmation relies on diagnostic tests.

A diagnosis can be made based on the presence of one or more of the major clinical signs with glycosuria/hyperglycaemia. The more signs that are present the more certain is the clinical diagnosis and it may be entirely possible in some very obvious cases to avoid any expensive testing.

Treatment

Treatment is focused on palliative and supportive care and management of the major symptoms of the condition (Murphy & Love 1996). No current treatment is available to 'cure' PPID. There is a strong temptation to rely on drugs alone but as these are generally poor and only support the patient, the nursing measures are probably equally if not more important.

Horses with significant hirsutism will often benefit from regular clipping. Management of intercurrent infections (using appropriate antibiotics and anthelmintics), weight loss (through careful feeding management and regular dental care) and control of laminitis (through routine foot care, possibly supplemented by use of corrective farriery) is very important. Most cases will also benefit from sustained ectoparasite and endoparasite control measures.

Drug treatments

The responses to drug therapy are largely unpredictable with some individuals responding positively or negatively to each of the available compounds. This problem is compounded by the variability of doses required and the usually slow responses that make dose adjustments difficult. In some cases combinations can be attempted but there are significant numbers of cases that fail to respond to any of the suggested drugs below and these are a real problem for the clinician. Drug therapy should not therefore be regarded as a definite treatment in all cases and the nursing care is likely to be a major aspect of the management.

Bromocriptine mesylate (Parlodel, Sandoz Pharmaceuticals, UK) has anti-serotonin and antihistamine effects. At a daily oral dose of 10 mg total dose daily for 3–12 weeks a significant number of mildly affected horses will show an improvement. Daily oral dosing of 0.04 mg/kg in the morning and 0.02 mg/kg in the evening has been advocated (van de Kolk 1997). The dose can be gradually reduced once a significant therapeutic benefit is achieved but it may take some weeks, with hair loss being the first sign of a beneficial effect. If no improvement is detected after 2 weeks an alternative drug should be used. The injectable formulations of bromocriptine can cause painful swellings and this approach is probably not necessary anyway.

Cyproheptadine hydrochloride (Periactin, MSD, UK) Cyproheptadine is a serotonin antagonist that suppresses the pituitary–adrenal axis. It is administered orally at 0.6 mg/kg q 12 h for the first week. The dose is increased to 0.8, 1.0, 1.2 mg/kg each succeeding week up to a maximum of 1.2 mg/kg until a benefit is gained. Initial doses of 0.25 mg/kg twice daily can be used with progressive increases to effect.

Early improvement is reflected in reducing thirst. Hair normalization may be seen within 1–3 weeks. Serum cortisol concentrations may take longer to fall (and may never reach normal concentrations); therefore laminitis may not improve in the earliest stages. Horses which fail to respond to this drug will often benefit from pergolide treatment (see below).

Pergolide (Celance, Ciba Ltd, UK) This is a dopamine agonist drug which can safely be administered orally at 1–3 mg total daily dose depending of the size of the horse; some cases, however, require up to 5 mg/day and some respond to lower doses. Alternatively, an exact dose can be calculated at 0.014 mg/kg orally. It has been reported to be very effective in some cases but others do not respond. There are few recorded long-term effects and many of those horses with few concurrent complications, will survive usefully for many years.

Trilostane This drug directly suppresses the adrenal cortex and therefore reduces the corticosteroid-related signs associated with PPID. Dose rates of 0.4 mg/kg q 24 h for 30 days are suggested. The results are as might be expected only limited and very temporary. The drug is very expensive and appears to have no significant advantage over the others. However, where no effect is achieved with pergolide or periactin this can be tried.

Prognosis

Therapy can prolong life by 2–3 years or more but it is expensive and needs to be sustained or at least administered intermittently. Effective treatment results in changes in hair quality and quantity, a reduction in blood glucose and a reduction in the polyuria/polydipsia. Changes in plasma cortisol are often slow or disappointing. Not every case shows all the improvements possible and some are single clinical changes, e.g. hair coat stabilization and no reduction in glycosuria or polydipsia.

Complications such as extreme weight loss and weakness, severe recurrent laminitis, central blindness and serious buccal and intestinal infections and infestations are indicators of a poor/reduced prognosis.

References

Beech J 1987 Evaluation of thyroid, adrenal and pituitary function. Veterinary Clinics of North America 3:649–660

Beech J, Garcia M 1985 Tumors of the pituitary gland (pars intermedia). In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, pp 182–185

Beech J, Garcia M C 1991 Disease of the endocrine system. In: Colohan P T, Mayhew I G, Merritt A M, Moore J N (eds) Equine medicine and surgery, 4th edn, vol. II. American Veterinary Publications, Goleta, CA, pp 1737–1751

Hillyer M H, Taylor F G R 1992 Cutaneous manifestations of suspected hypothyroidism in a horse. Equine Veterinary Education 4:116–118

Hillyer H M, Taylor F G R 1997 Cutaneous manifestations of suspected hypothyroidism in a horse. Equine Veterinary Education 4:116–118

Hillyer M H, Taylor F G R, Mair T S et al 1992 Diagnosis of hyperadrenocorticism in the horse. Equine Veterinary Education 4:131–134

Irvine C H G, Evans M J 1977 Hypothyroidism in foals. New Zealand Veterinary Journal 25:354

Knottenbelt D C 1997 Formulary of equine medicine. Liverpool University Press, Liverpool, p 43

Murphy D J, Love S 1996 Diagnosis and treatment of equine pituitary adenoma (Cushing's disease). In: Veterinary Annual, vol. 36. Blackwell Scientific, Oxford, pp 302–308

Schott H C 2002 Pituitary pars intermedia dysfunction: equine Cushing's disease. Veterinary Clinics of North America 18:237–270

Shearer D 1988 Bilateral alopecia in a Welsh pony associated with hypothyroidism. Proceedings of the Annual Congress of the European Society of Veterinary Dermatology 5:25–26

van de Kolk J H 1997 Equine Cushing's disease. Equine Veterinary Education 9:209–214

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14 Nutritional disorders

Iodine poisoning (iodism) 328

It is easy to forget that the skin, like other organs, requires the normal nutrients if it is to behave in a normal fashion. Almost any inappropriate food material and any gross deficiency of the basic requirements can affect the skin to some extent (Hintz 1993). However, even prolonged malnutrition may have surprisingly little material effect on the skin and hair.

Energy deficits (usually the result of starvation, malabsorption, parasites or concurrent disease) commonly induce a dry, scurfy, moth-eaten coat with poor skin elasticity. Normal hair shedding and regrowth can be impaired significantly with long coats and patchy moulting. It is remarkable again that even in the face of severe deprivation the skin is often normal. However, concurrent ectoparasites and other bacterial and fungal skin infections often complicate these cases.

Protein deficits cause failure of hair growth and shaft weaknesses. Alopecia or poor coat density can develop but the status of the horse in other respects will probably command more attention.

Dietary fats and oils have been and are still commonly added to feeds for horses (in particular linseed) and the use of these materials (including evening primrose oil) can have a dramatic improving effect on hair quality. Late winter coats are often improved significantly by dietary supplementation of a vegetable oil over 20–60 days. There are no known harmful effects of these on coat quality, but they may have some effects on fat-soluble vitamin absorption and some have significant metabolic/systemic effects when fed in excess (e.g. linseed).

Dietary minerals are known to affect coat and skin health as well as skin healing ability.

1 *Zinc* in particular appears to have a major influence on skin health and an adult horse requires a regular daily intake of around 500 mg of zinc. Deficiency of zinc develops within a short time as reserves are low and manifests as generalized alopecia with extensive surface scaling and flaking giving an appearance of severe dandruff (Harrington et al 1973). Initially the lesions may appear on the thighs and ventral abdominal wall. Severe and prolonged deficiencies result in generalized exudation and flaking with extensive and severe loss of hair (Fig. 14.1). Any remaining hair is easily pulled out by light grooming. The histological appearance is characteristic (hyperkeratosis, acanthosis and parakeratosis). Daily dietary supplementation with zinc methionine or other zinc salts has a curative effect over some weeks.
2 *Iodine* deficiency during pregnancy has obvious effects on the hair coat of the foal at birth. Affected foals have a sparse coat and an obvious goitre is often found at birth. Other deformities such as contracted tendons and fused joints (arthrogryposis) may be associated with iodine deficiency. Chronic excessive iodine fed to adult horses (often in the form of seaweed powder supplements, etc.) may be responsible for a sparse, short hair coat (Fadok & Wild 1983). Iodism can also develop during treatment of fungal (and other) infections with potassium or sodium iodide. The earliest signs include lacrimation and a scurfy, dry coat quality (see below).



Figure 14.1 Zinc deficiency with severe exfoliative non-inflammatory shedding of the hair and hyperkeratosis.



Figure 14.2 Vitamin A deficiency-related coronitis.



Figure 14.3 Biotin (and methionine) deficiency. The feeding regimen had changed drastically some 4–5 months previously and the deficiency is reflected in the poor quality of hoof periople. Correction resulted in a slow improvement with the damaged horn eventually growing out.

3 *Copper* deficiency causes a loss of black pigment because it is essential for the production of melanin (McLean & Jones 1983). Affected horses develop a coarse, harsh hair quality and a russet-brown hue to the darker areas of the coat. Alopecia and browning of the hair around the eyes gives the animal a 'spectacled' appearance, but these signs are not as prominent in the horse as in cattle and may easily be missed. Confirmation of the diagnosis can be made from blood and liver assays for copper, but response to careful supplementation may be as useful. A more serious tendency to arterial rupture and chronic anaemia is possibly associated with copper deficiency. Horses may be more resistant to copper deficiency than other domestic animals but there have been few studies on the status of horses grazed on known copper-deficient land.

Vitamin A dietary deficiency is reported to cause a rough, coarse and dull hair coat and a moderate to severe ulcerative coronitis. Deficiencies of vitamin A are particularly rare in horses being maintained on normal feeding regimens, but prolonged deficiency may cause a mild or more severe inflammatory reaction at the coronary band (Fig. 14.2). Prolonged deficiency causes marked hyperkeratosis but this is very rare except in young growing horses maintained under prolonged, extremely poor nutritional conditions particularly if grains, which have a poor vitamin A concentration, are fed with very poor quality hay or straw and the animals have no access to green forage.

Other nutritional elements are unlikely to cause dermatological problems but many which induce these signs in other species have not been documented in the horse. Deficiencies of B and C vitamins are extremely unlikely in normal horses on normal feeding regimens or indeed even under conditions of moderate or severe deprivation/starvation. However, methionine and biotin (together or independently) have been used to improve hoof quality. Therefore, by inference, poor hoof quality may be associated with hoof walls which lack a normal periople (Fig. 14.3) or are subject to severe cracking and loss of normal hoof pliability. It is not always easy, however, to differentiate this from dryness and harsh underfoot conditions which cause break-back and alteration in hoof quality.

Iodine poisoning (iodism)

Profile

This may occur as result of ingestion of abnormal amounts of iodine (in seaweed and supplements derived from this) or goitrogenous plants such as *Brassica* spp. Far more common, however, is prolonged medication with oral potassium iodide or sodium iodide (Fadok & Wild 1983). Significant iodism is very unlikely from ingestion of seaweed/kelp or

Key points: Iodine poisoning

12 15 16 20 24

1 Rare, rather trivial condition associated with abnormal iodine ingestion (either through natural intake or through the medicinal use of iodides systemically or topically. It is imperative that pregnant mares do not receive excessive amounts of iodine.
2 Clinical signs are restricted to a generalized seborrhoea. Where very high amounts are used medicinally there may be lacrimation, a greenish sticky naso-ocular discharge and some hoof wall defects ('laminitic' rings and a loss of periople quality).
3 Diagnosis is usually simple and based on known ingestion of goitrogenous plants or long-term medication with iodides. Goitres are not a common diagnostic feature.
4 Treatment by removing the source is the solution.
5 It is important to ensure that iodine intake is regulated in pregnant mares.



Figure. 14.4 Iodism. Heavy scaling and flaking of the skin arising from persistent dietary supplementation with seaweed salts.

iodine supplement, but foals delivered to mares being fed high levels may have obvious goitre with or without concurrent signs of hypothyroidism.

Clinical signs

Excessive ingestion or systemic administration results first in lacrimation and a soft cough. The nasal and lacrimal secretions become progressively stickier and greener in colour. Heavy scurf/seborrhoea develops mostly on the mane and tail base and on the neck and body (Fig. 14.4). Alopecia has been reported.

Differential diagnosis

- Seborrhoea: primary seborrhoea is rare and affects younger horses mainly; secondary seborrhoea usually has an obvious primary cause.
- Chorioptic mange (*Chorioptes equi*): pruritic condition mainly of the legs and head in predominately feathered legs; mites are easily identified.
- Selenium toxicity: laminitis and separation of the coronary band, and mane and tail hair loss.
- Mane and tail seborrhoea: long-standing flaking restricted to the mane and tail.
- Mallenders and sallenders (epidermal naevus): long-standing scaling with intermittent localized pruritus on the front of the hock and behind the knee.
- Other causes of lacrimation.

Diagnostic confirmation

There is history of medication (usually with sodium or potassium iodide) or supplementation (often with sea-salt/seaweed supplements).

Treatment

Reduce or stop intake of supplements or medication.

References

Fadok V A, Wild S 1983 Suspected cutaneous iodism in a horse. Journal of the American Veterinary Medical Association 183:1104–1106

Harrington D D, Walsh J, White V 1973 Clinical and pathological findings on horses fed zinc deficient diets. In: Proceedings of the Third Equine Nutrition and Physiology Symposium, USA

Hintz H F 1993 Nutrition and skin disease. In: Robinson N E (ed) Current therapy in equine medicine 3. WB Saunders, Philadelphia

McLean J, Jones W E 1983 Depigmentation: copper supplement therapy, a case report. Equine Veterinary Science 3:208

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15 Neurological disorders

Psychogenic self-mutilation 331
Neurological self-mutilation and secondary dermatitis 332
Sweating 333

It is easy to forget that the skin is a major sensory organ and that its neurological components are also subject to disease and injury. Whilst direct trauma is usually obvious and elicits the normal pain responses, alterations in a peripheral sensory nerve or within the central nervous system can have significant cutaneous signs. Psychosomatic self-trauma, pruritus and inappropriate sweating patterns are the main presentations of neurological skin disease.

There are some important conditions where the skin is the cause of concern to the owners but the underlying disorder is either psychosomatic or a result of peripheral or central nervous system disease. Indeed there are major diseases that are characterized by cutaneous responses and in some cases these are the earliest and therefore most easily overlooked signs. For example, rabies and Aujesky's disease have central origins but pruritus is an important early sign. Early recognition of the cutaneous signs may enable proactive measures to be taken that will limit the dangers both to handlers and to other animals and may reduce the overall suffering of the affected animals.

In addition, non-infectious damage to nerves such as a spinal fracture or damage to the cauda equina or the autonomic supply to large areas can result in hyperaesthesia, hypalgesia or localized sweating.

Sweating is a major thermal control mechanism in horses and has been the focus of much attention both from the perspective of anhidrosis and as an indicator of autonomic neurological disturbances (Jenkinson et al 2006).

Psychogenic self-mutilation

Profile

This rare condition mainly affects stallions and is considered to be a psychosis arising as a result of boredom or frustration. Occasional cases are found in all classes of horse, where it can be classified as a vice or at least a psychosomatic disorder. There is no known cause and treatment is notoriously difficult.

Clinical signs

Affected animals usually bite at their flanks – most often forward of the stifle joint (Fig. 15.1). The bites can be quite vicious and may even break the skin.

Longer-standing cases have extensive thickening, scarring, alopecia and in some cases leukotrichia/leukoderma at the sites. The condition is not really a genuine pruritus, of course, but it can look remarkably like it!

Key points: Psychogenic self-mutilation

1. Rare condition mostly affecting stallions. Rarely affects other classes of horse but they can be severely affected and are usually presented as pruritus cases.
2. Signs relate to self-trauma – often at one site and usually the flank regions cranial to the stifle. Considerable skin damage can be done and long-standing cases often have gross thickening of the skin with variable degrees of hair loss and scarring. The horses can be distracted easily but return to the vice once the stimulus is removed.
3. Diagnosis is simply by observation and distraction. No specific cause can be identified.
4. Treatment involves changing the environment and reducing boredom but some cases continue regardless.
5. The prognosis for the animal is good but the condition is sometimes a persistent vice.



Figure 15.1 This stallion developed a self-mutilation vice and consistently chewed and bit at its right stifle, causing chronic thickening and hair loss. Scarring resulted in a marked thickening of the skin at the site. Although he could be distracted from the vice it was impossible to control.

Differential diagnosis

- Ectoparasite infestations: seldom affect this region specifically. More often generalized pruritus. Easily diagnosed from brushings.
- Other causes of pruritus (see p. 442).
- Atopy: young horses with genuine pruritus that shifts from site to site in an unpredictable way.
- Cauda equina neuritis (polyneuritis equi): pruritus is not a common feature apart from in the early stages when perineal irritation can be present. There is usually perineal hypalgesia and tail and bladder paralysis.
- Trigeminal neuralgia/head-shaking syndromes: rare, involuntary hyper-reflexic/hyperaesthetic disorder affecting the facial skin only.
- Viral neurological pruritus: usually a single focus and the signs are rapidly progressive. The animal cannot be distracted from the site.
- Dermatophytosis (ringworm): characteristic epidemiology and only mildly pruritic in a few circumstances. Seldom affects the flank area. Size and shape of lesions and epidemiology are characteristic, self-resolving or responds to specific treatments; diagnostic mycology.
- Dermatophilosis: not pruritic.
- Sarcoid (verrucose): not pruritic.

Diagnostic confirmation

- Clinical signs are characteristic (secondary bacterial and fungal infection can occur at the site of the worry).
- Biopsy is unremarkable with evidence of mild ongoing trauma-induced inflammation and bruising.

Treatment

The only treatment is distraction – stable toys, a view of other horses and a reasonable distance away from stallions actually performing stud duties are helpful. Topical local anaesthetics do not help at all. Sedation or behaviour-altering drugs are a severe and unnecessary way to manage the problem (Dodman et al 1994).

Some continue with the vice for life and then the damage to the skin is correspondingly more severe. As time progresses, the bites may become less damaging.

Secondary infections and wound care may be needed as appropriate.

Neurological self-mutilation and secondary dermatitis

Profile

Neurological self-mutilation develops when there is a neurological cause of an apparently pruritic state. Neuritis or neuropathy resulting in inappropriate impulses transmitted down sensory nerves (or in some cases the total obliteration of the sensory pathway resulting in numbness) can cause a horse to rub at a particular skin site. The cause of the damage to the nerve can be infectious (viruses such as rabies and Aujesky's disease are well-recognized examples) or traumatic (such as might occur following a cervical vertebral fracture) or immunological (such as follows equine herpesvirus myeloencephalopathy syndromes in which ischaemic vasculitis of the cauda equina can lead to hyperaesthetic or hypalgesic states around the perineal region. In all these cases the dermatological evidence is secondary to an underlying neurological problem. Equine dermatologists should always remain aware of these conditions and consider them in cases that do not appear to have any dermatological explanation for the signs.

Where neurological function is impaired to the bladder and perineum, the first clinical evidence is usually dermatological – excoriation and scalding from urine or faeces may be the condition that faces the primary clinician.

Clinical signs

In these conditions the dermatological signs are not the first events – the cutaneous lesions develop after the onset of the apparent pruritus, not before. This is a very important question to ask the owner during a dermatological examination. The nature and cause of the 'pruritus' are such that an affected animal cannot usually be distracted from the sign although in some conditions it is either intermittent or variable in severity from time to time.

The various disease/disorders that are associated with apparent pruritus have their own recognizable signs which can be found in medical texts. The major causes of neurological pruritus include:

1. rabies and other encephalitides
2. trigeminal neuritis/neuropathy
3. peripheral neuropathy (neurotmesis/axonopathy)
4. cervical/thoracic or sacral fractures affecting sensory pathways
5. immunological disorders including cauda equina neuritis (polyneuritis equi)
6. head-shaking/trigeminal neuropathy syndromes.

Rabid horses bite and chew at the site of the original bite, and an accurate history is very helpful. Clearly the horse

Key points: Neurological self-mutilation and secondary dermatitis



1. Where there is a significant neuritis/neuropathy in a peripheral sensory nerve pathway or centrally within the brainstem or cortex, an apparent pruritus can be shown.
2. The dermatological signs are secondary and develop after the onset of apparent pruritus. Skin excoriation, thickening and infection occur as result of the self-trauma. The signs of the underlying condition are the primary state and so there will usually be other evidence at the same time.
3. Diagnosis requires an exhaustive clinical examination and elimination of the alternative primary causes of a dermatological disorder.
4. Treatment will depend on the underlying cause but usually topical local analgesics have some effect if the problem is in the peripheral pathway but not if it is central in origin.
5. The prognosis varies with the various diseases and the prospect of restored neurological function. For example, rabies and cauda equina neuritis have a zero prognosis because there is no chance of a restoration of nerve function whereas in some traumatic cases the nerves can and do repair.

would have to have an exposure possibility whether the bite had been recognized at the time or not (Fig. 15.2).

Horses with trigeminal neuropathy-related head-shaking frequently rub their faces, mouths and muzzles on hard objects as if the regions were genuinely pruritic (Fig. 15.3).

Horses with trauma- or inflammation-induced neuropathy usually itch at a specific locus relating to the pathway of the relevant sensory nerve. Often there is detectable pathology even if this takes a considerable effort to locate **(Figs CD15 • 1 and CD15 • 2)**. Clinical signs may include hypalgesia or hyperaesthesia at a defined site.



Figure 15.2 This donkey was found biting incessantly at its foot where a previous dog bite had occurred. A diagnosis of rabies was confirmed post mortem. It is important to consider all the available differential diagnoses in every such case.



Figure 15.3 This horse was presented with an acute onset of self-trauma limited to the side of the face and muzzle of one side. Over a 12-hour period the horse showed distress and rubbed incessantly. Topical steroids and local anaesthesia using an infraorbital nerve block failed to help and 4 days later the horse developed more typical signs of head-shaking and was diagnosed with trigeminal neuralgia.

Horses with cauda equina neuritis or sacral vertebral fractures may rub the perineum as if affected by any of the other causes of perineal pruritus. Where the condition affects anal and bladder function, the clinician may be presented simply with a case having cutaneous excoriation. A similar state could derive from bladder paralysis causing scalding dermatitis of the hind legs of geldings and the perineum of mares.

CD

Differential diagnosis

- Ectoparasite infestations: seldom affect this region specifically. More often generalized pruritus. Easily diagnosed from brushings.
- Other causes of pruritus (see p. 442).
- Atopy: young horses with genuine pruritus that shifts from site to site in an unpredictable way.
- Idiopathic self-mutilation syndrome: stallions usually – easily distracted.
- Perineal pruritus due to *Oxyuris equi* or insect bite hypersensitivity (see p. 224).
- Facial pruritus due to ectoparasites or contact hypersensitivity: intermittency and exposure-related signs – often parasites can be identified.
- Dermatophytosis (ringworm): characteristic epidemiology and only mildly pruritic and seldom affects this particular area. Size and shape of lesions are characteristic, self-resolving or respond to specific treatments; diagnostic mycology.
- Dermatophilosis: not pruritic.
- Sarcoid (verrucose): not pruritic.

Diagnostic confirmation

- Clinical signs of the primary disease are often characteristic.
- There may be secondary bacterial and fungal infection at the site of the worry.
- Biopsy is unremarkable with evidence of mild, ongoing, trauma-induced inflammation and bruising.

Treatment

Treatments are totally dependent on the primary disorder. Topical local anaesthetic pads, gels, injections and so on, are usually ineffective unless the condition relates to a very peripheral neurological stimulus.

Sweating

Profile

Horses have a well-developed sweating mechanism and will sweat in response to excitement, exercise and high ambient temperatures. Sweating is an important sign in several disease states – the dermatological signs may be early indicators of a serious internal disorder.

Abnormal patterns of sweating are manifest as dermatological changes such as diffuse sweating and patchy sweating.

Clinical signs

Abnormal (physiologically unexplained) diffuse sweating can be a result of abnormal catecholamine release such as

Key points: Sweating

1 Abnormal sweating patterns may indicate serious underlying neurological and endocrine disease. The first clinician called to see these cases is often the dermatologist because the overt signs are in the skin.
2 Autonomic diseases such as equine dysautonomia (grass sickness) and Horner's syndrome have characteristic sweating patterns (the signs of the conditions are recognizable in most cases). Spinal and orthopaedic disease can result in segmental interruption of the sympathetic outflow leading to localized vasodilation and sweating.
3 Diagnosis can be established by elimination of other explanations and the findings from a detailed neurological examination. The value of an alpha-2-adrenoreceptor agonist sedative as a diagnostic aid to Horner's syndrome should not be forgotten.
4 Treatment depends on the cause but most often there is nothing that can be done specifically.
5 The prognosis depends on the cause.



Figure 15.4 A defined area of sweating developed in this hunter following a fall. Radiography showed a small fracture of the sixth cervical vertebra. Sweating continued and resulted in long-term hair loss and changes in skin texture and pigmentation.

occurs in cases with phaeochromocytoma (functional tumour of the adrenal medulla). The condition is very rare, of course, but owners may not have detected the concurrent tachycardia and pupillary dilation. Horner's syndrome arising from a thoracic, neck or cranial sympathetic trunk pathology (including guttural pouch mycosis or bacterial diverticulitis lesions) is frequently overlooked by owners who may present their horses with unilateral facial or neck and face sweating. The prolonged sweating often results in secondary infection and matting and greasiness of the hair.

Conditions that result in more localized sweating usually arise as a result of autonomic nerve dysfunction. Interruption of specific segmental sympathetic outflows from the spinal cord may result in defined patches of warmer skin with prominent sweating (Fig. 15.4). Where this is long-standing the sweating may be less obvious but warmth, alopecia that cannot be explained by other reasons, and sometimes pigmentary changes can be recognized.

Sweating over the face/neck is a cardinal sign of Horner's syndrome. Again this is an autonomic nervous system disorder with a secondary dermatological sign.

CD

More generalized autonomic pathology such as equine dysautonomia (grass sickness) results in typical patchy sweating over some areas of the body **(Fig. CD15 • 3)**. In this case the cutaneous signs are helpful diagnostically but have little long-term relevance – the few cases that recover from equine dysautonomia have no obvious sweating tendency and no related clinical skin disease.

Differential diagnosis

Primary neurological diseases responsible for the syndrome should be identified.

Diagnostic confirmation

Clinical signs are characteristic (secondary bacterial and fungal infection can occur at the site if sufficiently prolonged).

Treatment

The primary condition should be identified and where possible treated.

References

Dodman N H, Normile J A, Shuster L, Rand W 1994 Equine self mutilation syndrome (57 cases). Journal of the American Veterinary Medical Association 204:1219–1224

Jenkinson D M, Elder H Y, Bovell D L 2006 Equine sweating and anhidrosis. Part 1: equine sweating. Veterinary Dermatology 17:361–392

16 Iatrogenic and idiopathic disorders

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Transient trauma-related pigmentary loss	339	Idiopathic regional pigment-related follicular dysplasia syndrome	349
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There are several circumstances when iatrogenic skin disease develops.

1 Attempts to treat other diseases with topical applications or skin dressings, for example the application of 'blisters' or counterirritants, and firing of tendons for 'treatment' of orthopaedic disorders (Fig. 16.1). Application of bandages may also cause problems – especially where the dressings are non-elastic or when they are left too long or when they are not of an appropriate type (Fig. 16.1C).
2 Intentional or unintentional over-medication for skin disease. In some circumstances owners fail to read the instructions provided or feel that a 'bit extra' or 'an extra application or two' is bound to make the medication more efficacious. This is an unfortunately common event since owners often feel some degree of frustration when medications fail to improve the condition. Increasing the concentration of medications is often a dangerous approach – sometimes the skin can be significantly damaged and even permanently affected.
3 An unexpected (inappropriate) reaction to a medication or procedure whether for the skin or not; this is usually either idiosyncratic or is sometimes a result of a failure of technique. This includes such things as injection abscesses, allergic or hypersensitive reactions to applied or injected medications. It is difficult to apportion blame in circumstances when the 'correct' procedures and material were chosen!
4 An expected but undesirable change in the skin as a consequence of treatment applied for other purposes. Whilst some of these are temporary or transient



Figure 16.1 Iatrogenic damage can be obvious and intentionally caused such as branding marks **(A)**, or can cause more damage than intended as in firing necrosis **(B)**, or can be unintentional such as bandage necrosis **(C)**.

responses others are permanent. For example, application of an antimitotic cream to a small penile squamous cell carcinoma might cause considerable transient balanoposthitis. The trade-off is of course that the tumour would resolve. A good example of a persistent change would be the changes that follow radiation exposure in the treatment of cutaneous or subcutaneous tumours such as sarcoid or lymphoma. A scar at a wound site would be an expected result of the injury but scars at the sites of the sutures might be a disappointing outcome **(Fig. CD16 • 1A and B)**.

A full history of the skin disease will usually help enormously but understandably in some circumstances owners might be reluctant to provide all the information.

It is almost inevitable that there will be some isolated cases of singularly rare and therefore very poorly understood skin diseases in all species and the horse is no exception.

When faced with a very unusual state that fails to adequately fit into any known syndrome or condition, the clinician should be honest to the client and explain that the condition has no obvious diagnosis. The investigation of such a case should be exhaustive but may justifiably include periods of delay to see if the clinical syndrome becomes more recognizable. Biopsy and a photographic and epidemiological record will add to the base knowledge on skin disease in horses and donkeys and thereby the number of idiopathic skin disorders will eventually be reduced. It is possible that other clinicians will have seen similar conditions and may even have a plausible answer to the condition but without sharing the problem and the solutions all clinicians will find themselves isolated far too often.

In some cases it can be helpful to consult texts and scientific articles relating to other species but there are also obvious dangers in this. The horse is unique in many ways and its skin has very limited ways of 'showing' pathology and so extrapolation is not usually wise.

A good example of this is provided by an outbreak of coronary band disease in young donkeys in Mexico in which the hoof capsule sloughed off leaving somewhat surprisingly a painful but recoverable situation. There were no other obvious clinical signs associated with the condition. Affected animals had no recurrences and no long-term difficulties with the hooves. The affected animals showed little pain during the development of signs. Apart from the age restriction and the seasonality there was no obvious cause and all the major differentials including infections, toxicities such as heavy metal and selenium poisoning and ergotism, etc. were eliminated **(Fig. CD16 • 2A–C)**.

However, there are several well-recognized conditions that have no convincing explanation and these are described here.

Vitiligo

Profile

This is an acquired depigmentation that possibly has an autoimmune basis relating to anti-melanocyte antibodies (Naughton 1986). However, the localized distribution of the condition is somewhat at odds with this theory. An auto-toxicity alternative has been proposed. Neural alterations and virus infections have also been proposed.

Depigmented spots and larger, poorly defined areas appear on the skin. They may be idiopathic or may follow primary damage to melanocytes (Meijer 1962, Pascoe 1973). They are usually restricted to horses over 4 years old. The condition may be heritable and is probably commonest in Shire and Arabian horses.

Clinical signs

Depigmented circular or irregular spots and bigger less well-defined areas up to (or over) 1 cm diameter which increase in number rather than size (Fig. 16.2) are typical. The lesions may be more or less symmetrical in distribution.

Key points: Vitiligo

13 15 20 23 24

1. An acquired depigmenting disorder of limited areas of the skin, possibly related to an autoimmune condition and frequently encountered around the eyes and face in particular.
2. Arabians, Thoroughbreds and Shire horses are possibly more prone to the condition. Grey horses may be more affected but this is possibly because the changes are more obvious in these. Horses of all ages can be affected. The condition is remarkably free of concurrent symptoms but some have associated hair loss.
3. Anular, more or less symmetrical, permanent, areas of depigmentation of varying extent develop without apparent symptoms of inflammation or apparent causative insult.
4. The features are typical but biopsy can be used to confirm the condition. There is a distinct lack of melanocytes but otherwise the skin is normal.
5. No treatment is possible although some reports suggest that copper and vitamin A are helpful.

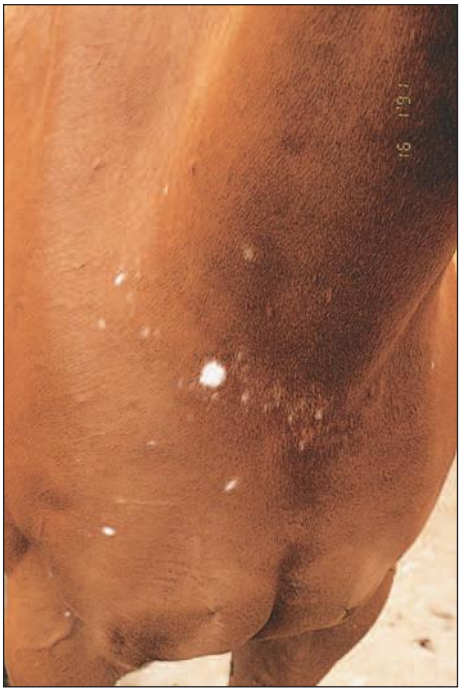


Figure 16.2 Vitiligo (idiopathic depigmentation): spotted, body form (spotted leukotrichia) which caused changes in the hair colour.

On the body these seldom if ever cause alopecia, but around the eye where the hair coat is sparse in any case, hair loss is sometimes pronounced (Fig. 16.3). Leukoderma may or may not copy the associated leukotrichia.

Owners often become very concerned but can be reassured of the benign nature of the condition, although the progressive nature of some cases makes certain uses of the horse unrewarding. There is no associated pain, pruritus or any evidence of inflammatory responses (Figs 16.4 and 16.5 and **Fig. CD16 • 3A and B**).

The depigmented areas may be more prone to actinic dermatitis (sunburn) and ultimately to squamous cell carcinoma.

Differential diagnosis

- Acquired vitiligo (leukoderma/leukotrichia from skin injury such as cold, trauma, radiation. etc.): history of trauma/damage.
- Arabian fading syndrome: Arabian breed; characteristic depigmentation on face and perineum.



Figure 16.3 Vitiligo (idiopathic depigmentation) around the eye.



Diagnostic confirmation

- Absence of injury.
- Circular white spots which appear without reason and do not increase in size.

Treatment

There is no remedy. Surgical removal of 3–5 mm spots where only 2–3 areas occur may be feasible but may cause more extensive depigmentation. It is best left alone. Cosmetic coverage is possible in show horses by dyes or stains.

CD

Acquired persistent leukoderma/leukotrichia

Profile

Loss of pigment in hair and skin related to various factors such as pressure injury, cryosurgery, surgery, radiation

Key points: Acquired persistent leukoderma/leukotrichia

10 13 14 15 20 24

1. An acquired, depigmenting disorder arising from skin damage or insult. In some cases the insult may be trivial. Trauma, radiation or freeze or thermal burns are common instigating factors.
2. The distribution of the white hair (and possibly the associated white skin) changes follow the pattern of the insult. Freeze branding illustrates clearly how the melanocytes are more sensitive to insult than the skin itself.
3. Diagnosis is usually simple and the pattern of leukoderma/leukotrichia is typically associated with other skin damage. Usually by the time the hair colour changes are detected the original insult will have disappeared. Biopsy shows evidence of scarring and loss of melanocytes.
4. Treatment is not usually possible; cosmetic dyes can be used to mask the changes. More invasive 'treatments' such as excision or tattoo are probably contraindicated.
5. The prognosis is excellent – it is best regarded as a cosmetic problem (unless the extent of scarring and tissue damage is limiting).



Figure 16.4 This gelding developed bilateral extensive areas of vitiligo on its face **(A)** and around the eyelids **(B)** that progressed slowly for some years and then stabilized.

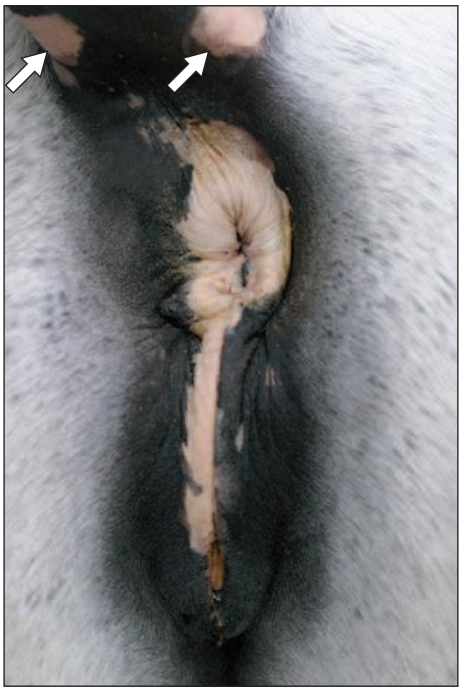


Figure 16.5 Vitiligo can also affect the perineal skin. The perineal skin of this grey horse had been completely black for many years and then over a period of about 6 months the pigment was lost. Note the two patches of vitiligo also on the lateral aspects of the tail base (arrows).



Figure 16.6 This horse was subjected to line and pin firing for a tendon disorder. The treatment has no merit but the scars remain obvious – usually as a combination of an alopecic scar and leukotrichia. Note also that the bandages applied to the limb at the time of the 'treatment' also resulted in leukotrichia on the more dorsal region of the cannon.

or other skin disorders. The pathological consequence is exploited in freeze marking. Even minor insults to the melanocytes cause significant loss or in some cases malfunction. This means that even minor skin damage and some trivial infections such as coital exanthema (see p. 138) can result in depigmented scars. In some disease states the melanocyte loss is less easy to explain. For example, the condition known as pinnal acanthosis (see p. 136) often causes proliferation of the epidermis with loss of pigmentation.

Clinical signs

Ill-defined, irregular white patches of hair and skin appear corresponding to areas of skin damage by many factors such as skin trauma (Fig. 16.6) and freeze branding (Fig. 16.7). Even tight bandages can cause melanocyte damage and white hairs appear and these are particularly obvious on distal limbs – often the owners will deny any knowledge of any episode of over-tight bandages or any obvious trauma.

CD

Cryosurgery or normal sharp surgery, irradiation (X-ray or gamma) (see **Fig. CD16 • 1A**), etc. can also cause changes in the melanocytes with local development of white hair. Freeze branding, which is used as a permanent means of identification, exploits this response. Where the freeze is superficial and transient only the hair becomes white; this implies that the follicular melanocytes are perhaps the most sensitive or that the process of pigmentary uptake in hair shafts is also affected. More extensive freezing causes more damage to the melanocytes in the treated area and so the skin becomes hairless and white. Even more extensive/severe freezing results in a scar formation and this may be darker or lighter in colour than the surrounding skin.

CD

Melanocytes can be destroyed by contact with irritants, harness, rubber, bits, etc. Common sites include the saddle region and withers and girth where they arise from repeated minor or more severe trauma (Fig. 16.7). The changes that follow rubber contact in particular arise without any obvious inflammatory response at all. There may be an abrupt loss of pigment in the affected skin and hair in particular and the contact area is closely related to the pattern of leukoderma/leukotrichia (Fig. 16.8 and **Fig. CD16 • 4A and B**). These changes are poorly understood but alopecia is seldom encountered in these conditions.



Figure 16.7 Leukoderma/leukotrichia (acquired vitiligo). Two forms of the condition are shown. Irregular 'saddle (and girth) marks' are usually accepted as being of traumatic origin. Freeze branding should result in a change in pigment without any other scarring, but in this case the 'burn' was less well judged and only limited pigment changes arose at the margins of the scars.

There is often no apparent thickening or other cutaneous changes, but with injuries and surgical sites

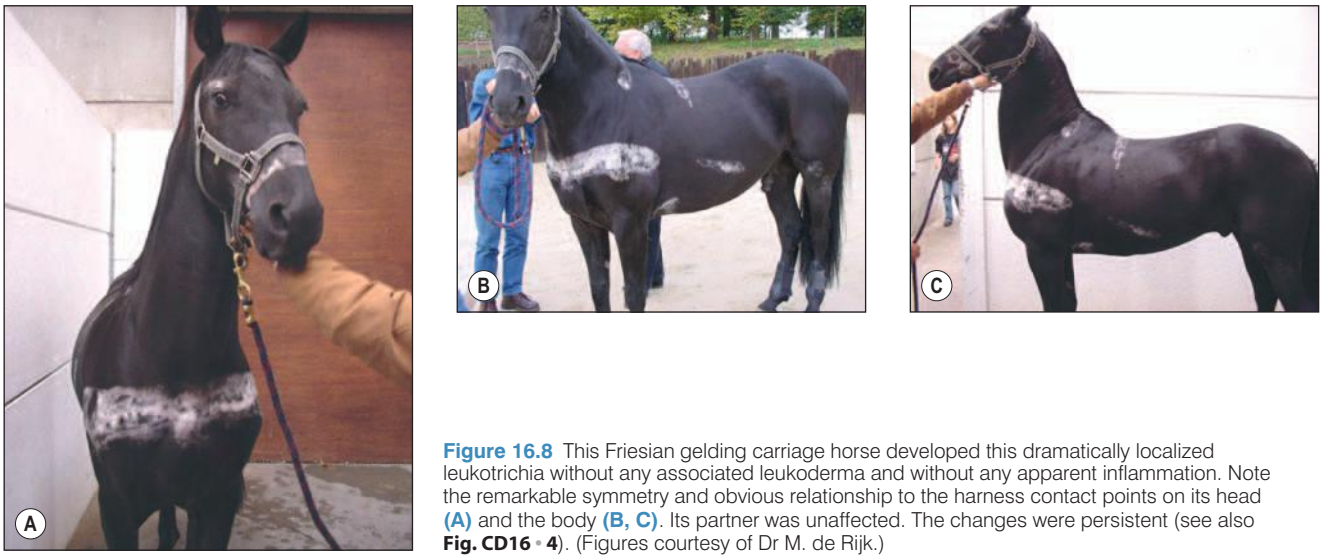


Figure 16.8 This Friesian gelding carriage horse developed this dramatically localized leukotrichia without any associated leukoderma and without any apparent inflammation. Note the remarkable symmetry and obvious relationship to the harness contact points on its head **(A)** and the body **(B, C)**. Its partner was unaffected. The changes were persistent (see also **Fig. CD16 • 4**). (Figures courtesy of Dr M. de Rijk.)

CD

the skin is sometimes patently scarred and thickened. It is important to realize that the hair cannot change colour overnight! The change has to appear with the growth of the hair and so it may be possible to identify roughly when the insult took place by the position of the change in the hair shafts. Often, however, the changes are fastest when the new hair coat (either summer or winter) is being produced and the old coat is being shed. This may give a misleading impression of an abrupt/acute change.

Differential diagnosis

- Vitiligo (idiopathic): no obvious instigatory event and usually round eyes/face and perineum.
- Spotted leukotrichia/leukoderma: isolated spontaneous white spots that may come and go.
- Coital exanthema: localized small and coalescent foci around genitalia only.
- Arabian fading syndrome: extensive areas of depigmentation of face and perineum.
- (Reticulated) Leukotrichia: patterned leukotrichia characteristically along the back.
- Systemic lupus erythematosus-like syndrome: multisystemic disorder.
- Discoid lupus: photo-exacerbated superficial facial disorder.
- Transient trauma-related 'depigmentation': history of trauma, temporary.
- Cutaneous onchocercosis: usually alopecic and thickened skin, face and neck are most often involved.

Diagnostic confirmation

- History and physical appearance are characteristic.
- Biopsy shows an abnormal lack of melanocytes in damaged/scarred skin – the extent of the damage can be very limited or even invisible in some cases.

Treatment

There is no known treatment. Attempts to surgically remove the affected area usually creates another similar (often bigger) problem area. By far the best approach is to leave the area alone and to apply suitably coloured dyes to mask the changes if that is what is necessary! It is important to emphasize to owners how the damage arises and ensure that the potential insults are not repeated. The prognosis is excellent for the horse even if the owner is annoyed/disappointed by the changes. Saddle marks and to a lesser extent girth marks are very common and viewed by most as a cosmetic nuisance.

The erasure or alteration of freeze branding scars is virtually impossible but can be attempted with laser surgery. However, it invariably leaves more white hairs and skin, but the number may be hidden.

Transient trauma-related pigmentary loss

Profile

The loss of pigment in traumatic injuries to the skin occurs when the pigment-containing cells of the epidermis are lost.

Key points: Transient trauma-related pigmentary loss

10 11 12 13 14 24

1. A common transient pigmentary loss with only occasional permanent changes.
2. Area of transient depigmentation that follows bruising, erosion or ulceration of the skin. Signs of trauma may be obvious or may be less so. Sometimes the changes can become permanent and then there may be leukoderma and leukotrichia (see p. 341).
3. History of superficial cutaneous bruising or grazes. The transient nature of the changes differentiates this from other persistent forms of pigmentary change such as vitiligo and the changes that follow more severe skin trauma and selective destruction of melanocytes (e.g. freeze band, saddle marks).
4. Treatment is limited to nursing care of the injured skin but recovery of pigment can take some weeks.
5. The prognosis is excellent.

The most obvious cases relate to grazes and superficial skin erosions and ulcerations. There is usually no long term scar but some injuries may have deeper wounds where scarring and long-term leukoderma/leukotrichia can develop.

Clinical signs

Following obvious trauma, the loss of the epidermis will inevitably result in exposure of the basal and germinal layers and possibly the dermis (Fig. 16.9). The episode of trauma is usually mild but the area involved will vary. A similar condition occurs when the skin is sunburnt or is excoriated with faeces/diarrhoea/urine or other discharges. It can also occur simply following swelling or localized inflammation with epidermal collarette formation. The pigment is gradually restored over some weeks, usually fully but sometimes to a lesser extent.

Hair loss as a result of the trauma or excoriation may occur – this is not really alopecia.

Differential diagnosis

- Vitiligo: non-painful, non-pruritic, spontaneous depigmentation around face and perineum.
- Pinky Arab syndrome: Arabian breeding and face and perineum mainly involved.
- Deep skin trauma: history of trauma.
- Radiation burns/freeze branding: known exposure.
- Reticulated leukotrichia: painful and non-painful forms restricted to patterns ('tiger stripes') on dorsum.
- Photosensitization: changes restricted to exposed non-pigmented skin.
- Sunburn: changes restricted to non-pigmented skin exposed to strong sunlight.
- Chemical burn/irritation: known exposure or suggestive pattern of lesions.
- Contact allergy: only the area exposed to allergen is affected.

CD

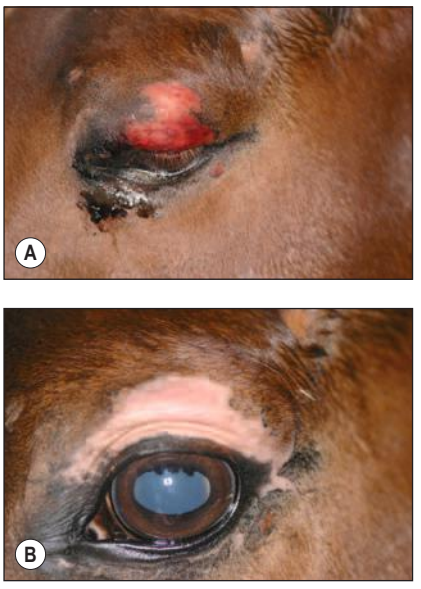


Figure 16.9 This horse suffered a graze to its upper eyelid **(A)** that caused a transient loss of skin pigmentation **(B)**. Note the normal dark brown skin. The hair grew back normally after some weeks.

Diagnostic confirmation

- Clinical appearance and history.
- Biopsy is not necessary but the traumatic inflammation and oedema will be evident.

Treatment

Treatment is not necessary for the pigmentary problem but of course the quicker the skin is restored to normal the quicker the pigmentation will be restored. Often the hair loss that may have occurred takes longer to regrow so the site may remain 'noticeable' for some time. A few white hairs may develop in a few cases.

The prognosis is excellent.

Clipper rash

Profile

Owners often report a variety of clinical signs on the skin of horses that have recently been clipped. Where actual trauma occurs from poor clipper technique or poor adjustment of the blades, the lesions are often obvious traumatic skin injuries. Other cases show nodules or areas of inflammation. It is thought that this might relate either to heat in the blades or to inflammatory responses to the oils and sprays used for lubrication.

Clinical signs

Following clipping, injury may be obvious with bleeding or serum exudation. Nodules or areas of inflammation following clipper tracks and strictly restricted to the clipped area are significant. The signs are usually more severe on the thin skin of the neck and withers regions.

Nodules often take some days to develop **(Fig. CD16 • 5A and B)**.

Differential diagnosis

- Urticaria: oedematous raised plaques with an acute or peracute onset and usually a rapid resolution either spontaneously or following a single injection of dexamethasone.

Key points: Clipper rash

9 10 11 15 17 24

1. A common transient inflammatory skin condition occurring within hours or days of clipping sometimes as a result of heat or from oils or from sharp clipper trauma.
2. Skin trauma, multiple small focal non-painful nodules or more diffuse areas of mild inflammation following the pattern of clipping and strictly restricted to the clipped area are typical.
3. The transient nature of the changes differentiates this from other more persistent conditions. The history is typical.
4. Treatment is limited to nursing care of the injured skin but recovery can take some weeks. If oil is suspected as being involved this should be washed away carefully.
5. The prognosis is excellent but advice regarding clipping techniques and care of the clippers may need to be given.

- Eosinophilic dermatitis with collagen necrosis: usually slower to appear and seldom resolves. Not related to clipping but could be revealed by clipping.
- Dermatophilosis: usually related more to hair loss and characteristic bacteriology.
- Staphylococcal folliculitis/furunculosis: painful and exudative localized lesions with positive cultures. Could be related to clipping trauma also.
- Chemical burn/irritation: known exposure or suggestive pattern of lesions.
- Contact allergy: only the area exposed to allergen is affected.

Diagnostic confirmation

- Clinical appearance and history.
- Biopsy is not necessary but the traumatic inflammation and oedema will be evident.

Treatment

Treatment is not necessary but extra care should be taken with hygiene, and a single antibacterial wash may be helpful. Usually the nodules and any associated skin trauma will heal in time. Some may persist, however, so the sites may remain 'noticeable' for some time. A few white hairs may develop in a few cases.

The prognosis is excellent but care must be taken to avoid repeating such episodes, and advice regarding the use of appropriate lubricants and avoidance of direct trauma and heat should be given.

Leukotrichia (tiger-stripe, variegated leukotrichia, reticulated leukotrichia)

Profile

Dorsal, bilateral, reticulated, white hair-striping is characteristic. Although the condition is reported to affect yearling Quarterhorses in particular in the USA, the condition also affects Thoroughbred and Standardbred horses in Australia.

Key points: Leukotrichia

2 4 13 15 20 24

1. A rare, sporadic pigmentary disorder mainly affecting the dorsum of young (1–3-year-old) horses of different breeds under different conditions. 'Brindle' Quarterhorses may be overrepresented. Several suggestions have been made concerning its possible genetic basis and it may represent a particular form of linear keratosis; some pain initially and scaling along the lines of pigment changes are common.
2. Linear crusting lesions develop along the back with limited pain at first. Some long-term discomfort may be present. The permanent 'tiger striping'/brindling develops along the lines of the crusting some weeks later.
3. The underlying skin is normal histologically but the early stages have an ill-defined oedema and pigmentary incontinence.
4. There is no treatment for the condition.
5. Suggestions that it is sufficiently heritable to exclude breeding from affected horses are ill-founded.

Other breeds and older animals can be affected in other countries also, but it is most often reported in older horses with an unknown earlier history. It has been suggested that it is a form of erythema multiforme. There is a suggested correlation between rhinopneumonitis (EHV) vaccine and the onset of the disease but cases occur in unvaccinated horses and in circumstances when exposure to herpesvirus is very unlikely. Hereditary aspects have been suggested (see **Fig. CD16 • 6A–D**) but this is not established.

CD

Clinical signs

There is a sudden development of painless small vesicles and crusts arranged in a cross-hatched pattern along the dorsum between the tail and the withers. In some cases this stage passes unnoticed. In a few cases extreme pain can be encountered with resentment to both palpation and tack/harness placement; this may then be more akin to the hyperaesthetic leukotrichia disorder (described below). Temporary alopecia follows shedding of crusts. New hair is white but the skin retains its original pigmentation (Fig. 16.10).

The condition has a course of about 3 months. Repeated episodes have also been reported (Yu 1997).

Differential diagnosis

- Vitiligo (idiopathic): usually around eyes, face and perineum.
- Acquired (persistent/transient) leukoderma/leukotrichia: usually trauma related).
- Spotted leukotrichia/leukoderma: isolated spontaneous white spots that may come and go.
- Coital exanthema: localized small and coalescent foci around genitalia only.
- Arabian fading syndrome: extensive areas of depigmentation of face and perineum.



Figure 16.10 Reticulated leukotrichia in a Quarterhorse. Note the obvious dorsal transverse striping.

- (Reticulated) leukotrichia: patterned leukotrichia characteristically along the back.
- Systemic lupus erythematosus-like syndrome: multisystemic disorder.
- Discoid lupus: photo-exacerbated superficial facial disorder.
- Transient trauma-related 'depigmentation': history of trauma, temporary.
- Cutaneous onchocercosis: usually alopecic and thickened skin, face and neck are most often involved.

Diagnostic confirmation

- Absence of injury in clinical history.
- Clinical examination to eliminate any other causes: i.e. should check possibility of drug- or vaccine-related trigger mechanisms.
- Biopsy of affected skin shows pronounced dermo-epidermal inflammation (interface dermatitis).

Treatment

There is no known treatment; it is probably best left alone to settle with time. Attempts at local treatment may simply exaggerate the resentment of the horse until it becomes a serious management problem. Corticosteroids probably will not help, even at high doses. Treatment with aciclovir has been suggested followed by administration of the post-herpes-zoster neuralgia drug amitriptyline. There are no reports of the results of this approach.

The prognosis is good – most cases will eventually settle down and become a cosmetic curiosity.

Hyperaesthetic leuko(melano)trichia

Profile

This is a rare disease with highly characteristic signs restricted to the dorsum of mature horses and, as yet, only reported in some areas of the USA (Stannard 1987). It is possibly a variant of the reticulated leukotrichia condition (see above) but is associated with far more pain and obvious inflammation. Some cases are reported to follow the use of live or killed EHV (rhinopneumonitis/abortion) vaccine (Stannard 1987). The geographical restriction suggests that other factors are in fact involved and the vaccine relationship is likely to be coincidental/irrelevant.

Clinical signs

Single or multiple, very *painful*, non-pruritic, crusting dermatosis with small focal lesions, 1–4 mm in diameter, occur along the dorsum of the back and over the saddle area. Obvious (sometimes extreme) pain is usually manifest – horses react violently if the lesions are handled. (Note: in some cases the pain may precede the development of overt lesions and the horse may be presented for a 'cold back' or saddle resentment.)

Lesions remain for 1–3 months followed by spontaneous regression. Permanent white hair changes and sometimes limited alopecia are the norm (Fig. 16.11). In a few cases a similar clinical picture is seen but the skin darkens and the alopecia is more pronounced. This may or may not be a variation of the more regular but still rare hyperaesthetic leukotrichia (Fig. 16.12).



Figure 16.11 Hyperaesthetic reticulated leukotrichia in a cross-breed working horse in Ethiopia. This horse had severely resented palpation of the back for around 6 months and once the crusts were shed, the white hair appeared in its characteristic pattern and the pain subsided.

Key points: Hyperaesthetic leuko(melano)trichia

1. A rare pigmentary disorder without known aetiology. Immunological changes are suspected but little is known about it. It may reflect an early or unusual form of reticulated leukotrichia in which cutaneous sensory nerve fibres are involved.
2. Multiple linear and focal, crusting, very painful lesions along the back predominately. Pain may precede the development of obvious skin lesions. The consequent pigmentary changes are persistent.
3. Biopsy is non-specific with variable evidence of perivascular superficial and deep inflammatory responses.
4. Treatment is largely symptomatic. Analgesia with non-steroidal anti-inflammatory drugs and possibly corticosteroids may help but there are too few reported cases to be definitive.
5. The condition may last up to around 3 months and may recur.



Figure 16.12 Hyperaesthetic melanotrichia. The sparsely haired pigmented areas were acutely painful and the hair colour changed to white over the following years.

Some cases will resolve completely (although some scarring usually remains), while in others it may recur or be persistent with variable degrees of hypersensitivity.

Differential diagnosis

- Dermatophilosis (*Dermatophilus congolensis*): usually much less painful and with characteristic paint-brush hair loss with pustular bases and a reddened area of smooth underlying skin. The organism can be identified.
- Staphylococcal furunculosis (*Staphylococcus aureus/intermedius*): also very painful but tends to be more diffuse and exudative. The organism can be identified in culture and smears.
- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch): usually very seasonal and not painful – pruritus is the cardinal sign in defined regions. Recurrent seasonal condition.
- Dermoid cysts (single or few, non-painful): few, well-defined, long-standing nodules.
- Reticulated leukotrichia: non-painful similar condition.
- Idiopathic and trauma-induced leukotrichia: history of trauma and changes restricted to region of skin damage.
- Photosensitization: restricted to white-skinned regions.
- Sunburn: white-skinned regions on dorsum and face primarily.
- Chemical burn/irritation: history of exposure.
- Contact allergy: recurrence – more often pruritic than painful unless secondarily infected; affected region corresponds to contact region.

Diagnostic confirmation

- Clinical appearance is suggestive.
- Biopsy shows a marked, chronic subepidermal and intra-epidermal oedema.

Treatment

There is no known effective treatment. Even high doses of corticosteroids and non-steroidal anti-inflammatory drugs may have no material effect. The pigmentary changes can be masked with cosmetic hair dyes.

The course is usually some weeks with gradual remission, but some horses remain 'cold-backed' or 'saddle-shy'.

Unilateral papular dermatosis

Profile

This uncommon but distinctive papular disorder with an unknown aetiology affects yearlings and 2-year-old Quarterhorses (and some Thoroughbreds); other breeds and other ages may also be affected. The aetiopathogenesis is unknown (Scott 1988a). Most lesions develop in spring/summer, suggesting an insect-related aetiology (Williams 1995) and a viral cause has been suggested also (see p. 133). The characteristic restriction of the lesions to one side of the body possibly relates to insect feeding access but the condition is not seasonal and recurrences are rarely reported.

Clinical signs

Acute appearance of non-ulcerative, non-painful, multiple papules and nodules, usually in a loose group, 30 to >200 in number in a roughly circular arrangement limited to one side of the body trunk (Fig. 16.13). The groin and face can be affected also.

Older lesions may be obvious, even-sized, firm, round, well-circumscribed, non-ulcerative and non-alopecic nodules. Fresh nodules may occur in almost concentric rings around the original lesions. There is no associated pruritus and horses are otherwise unaffected.

Differential diagnosis

- Dermatophytosis (*Trichophyton* spp.) (before hair is lost): transient and distinctive epidemiology; organism can be identified histologically, directly and on culture.
- *Stomoxys calcitrans* or other insect bites/reactions: transient localized swellings that resolve quickly; clipping will reveal a central purple or red spot at the site of the bite/sting.
- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity/sweet itch): highly seasonal recurrent

Key points: Unilateral papular dermatosis

5 12 13 15 22 24

1. Unusual idiopathic condition in which localized papules develop (usually) on one side of the body, usually during summer.
2. Papules are often visible in small groups and are non-painful and non-pruritic. There are no pigmentary changes that accompany the lesions either temporarily or permanently.
3. Diagnosis can be made by biopsy but can be confused with eosinophilic dermatitis with collagen necrosis (collagenolytic granuloma).
4. Treatment is not necessary as most cases resolve spontaneously over some months but a resolution may be hastened with oral corticosteroid administration.
5. Most cases resolve spontaneously but a few have recurrences every year.



Figure 16.13 Unilateral papular dermatosis. About 30 non-ulcerative, non-painful papules and nodules in a loose group in a circular arrangement on one side of the body trunk.

and deteriorating condition. Prominent pruritus in defined predilection sites such as mane-base, tail-head and neck.
- Nodular sarcoid: usually distinctive, slow growing and often with others of a similar type and other types. No restriction to one side usually.
- Collagen necrosis/eosinophilic granuloma: characteristic pathology but can be difficult to differentiate clinically. Usually many and seldom restricted anatomically to one side.
- Axillary nodular necrosis: single or few localized non-painful nodules in the skin in the girth region only.
- *Onchocerca cervicalis* filariasis: not restricted in distribution to one side; usually affects the head, neck and withers.

Diagnostic confirmation

- History and clinical appearance is distinctive.
- Skin biopsy shows eosinophilic folliculitis/furunculosis without any consistently recognizable microorganisms.

Treatment

Oral prednisolone at up to 2 mg/kg q 24 h (administered in the morning) (Scott 1988b) or dexamethasone at 0.1 mg/kg q 24 h (mornings) may produce a detectable and sometimes rapid benefit, but even prolonged courses of high doses do not always do so (Rothwell & Birch 1991). The nodules may persist and some become calcified.

Recurrences can be seen in subsequent years sometimes on the same side and sometimes on the other side.

Anhidrosis

Profile

Characteristic failure of sweating occurs in exercising horses over 2–3 years of age of any sex and colour which are subjected to movement from temperate to hot and humid tropical climates, although not all cases have this history. Cases can develop spontaneously in hot humid conditions but cases in temperate climates are very uncommon. Thoroughbreds may be predisposed but there is no strong evidence for its heredity. The condition can cause serious financial loss to racing owners/trainers.

Sweating is a major physiological function for thermoregulation in horses, contributing around 65% of the thermal heat loss during exercise. Failure to adapt to the requirements for extra sweating appears to be the underlying problem. In effect the affected horse suffers from heat exhaustion. Signs can develop suddenly for no apparent reason. The underlying pathology includes a transient abnormality of the sweat gland cells during episodes but this may be more of an effect rather than a cause. It seems likely that the problem lies in the adrenoreceptor function in the sweat gland cells (Warner & Mayhew 1983). The basis to the condition is probably a gradual failure of the secretory mechanisms of sweat glands that involves desensitization and suppression of regulation of cell receptors following continued adrenaline drive due to climatic stress (Jenkinson et al 2007).

There is no evidence to suggest involvement of thyroid hormones or electrolytic disorders.

Horses receiving β_2-adrenoreceptor agonists such as clenbuterol or terbutaline for treatment of airway disorders may be liable to the condition.

Key points: Anhidrosis

1 5 12 15 19 24

1. A significant metabolic disorder of sweating and thermoregulation of unknown aetiology characteristically (but not exclusively) involving horses that have moved to hot, dry or humid conditions from a temperate region. Cutaneous signs may be the most obvious outward evidence but these only develop fairly late.
2. Signs include an inability to sweat and to therefore control body temperature during exercise; body temperature and respiratory rate rise rapidly and remain high after even minimal exercise. The skin becomes persistently dry and the hair coat rough and greasy.
3. Diagnosis is usually suggested by history and the clinical signs. A subcutaneous adrenaline (epinephrine) response test can be diagnostic.
4. Treatment is very difficult and involves returning the horse to a temperate climate or stabling in an air-conditioned stable. A very long period of rest or gradually increasing adrenaline challenges can help. No effective medical treatment is available.
5. The prognosis is poor for return to effective work in the same environment.

Clinical signs

Loss of performance and/or exercise intolerance with an inordinately high respiratory rate after relatively minor exertion are the first evidence of a problem. Rectal temperature of over 41°C may be maintained for more than 30 minutes post exercise. Occasionally the horse may collapse (Hubert & Beadle 1998).

Inability to sweat or sweat patterns that are restricted to the medial thigh and flanks and the axillae may be seen. The skin feels dry and hot during and immediately after work. Prolonged recovery after exercise including prolonged elevations of heart and respiratory rates are common (normal horses will have established normal temperatures and heart and respiratory rates by 30 minutes post exercise).

Longer-term cases show patchy alopecia with a harsh, dry coat (often with a tessellated appearance and a seborrhoeic/greasy feel) (Fig. 16.14). Facial alopecia possibly as result of altered dermal blood perfusion or sustained high dermal temperature may be seen in more prolonged cases and this may be the only outward sign **(Fig. CD16 • 7)**.

Loss of appetite and reduced thirst may be noted.

Differential diagnosis

- Insect bite hypersensitivity (*Culicoides* spp. hypersensitivity, sweet itch): seasonal pruritic condition

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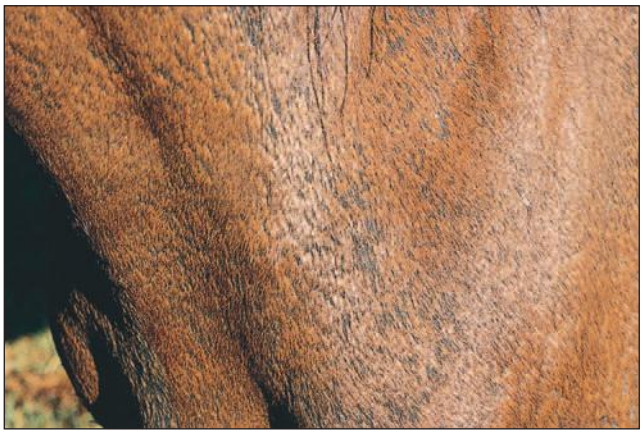


Figure 16.14 Anhidrosis. The coat is dull, dry, harsh and greasy.

with anatomically restricted signs according to biting site of a sensitizing *Culicoides*/fly.

- Dermatophilosis (*Dermatophilus congolensis*) (particularly the generalized, summer form): dry small scabby lesions with paint-brush hair pluckings featuring a crusted neck matting hairs together. Organism is characteristic.
- Dermatophytosis (*Trichophyton* spp.): characteristic epidemiology and clinical course; organisms can be identified on skin sections, hairs and on culture.
- Alopecia areata: localized, otherwise asymptomatic hair loss over small circular or larger diffuse areas; usually permanent but can be variable.
- Sarcoidosis: multisystemic generalized granulomatous condition with scaling seborrhoea and weight loss.
- Pemphigus foliaceus: distinctive clinical history featuring scaling and some ulceration over limited circular areas or larger areas or even the entire surface of the body; pathology is usually very typical and diagnostic.
- Other systemic illnesses with poor or restrictive performance and high respiratory rates.

Diagnostic confirmation

- History of a move from temperate to tropical climate and loss of performance.
- Diagnostic confirmation by intradermal tests usually using adrenaline injections of several dilutions.
  - Usually 0.5 mL of dilutions of $1:10^3$, $1:10^4$, $1:10^5$, $1:10^6$ are injected some distance from each other on the side of the neck.
  - A negative control using saline provides the baseline response.
  - Intradermal β_2-adrenoreceptor agonists such as clenbuterol or terbutaline or salbutamol may be a more specific test.
  - Anhidrotic horses have reduced sensitivity and prolonged response time (some may not sweat at all), while normal horses sweat noticeably at all dilutions. Partially affected horses will sweat only at the higher concentrations.

Treatment

Some cases fail to respond to anything other than a return to a temperate climate. Moving to a colder climate for the summer months may not be a practical option and then air conditioning in the stables over summer can be useful (especially during the acclimatization period) (Peter et al 1981). Careful environmental management appears to be the best approach (Hubert & Norwood 2002) and specific drug treatments including vitamin E supplements, antidiuretics and electrolytes are probably adjunctive rather than curative (Warner & Mayhew 1982).

Increased fitness, reduced stress and rest for an extended period sometimes help but may not do so. Very careful exercise regimens that keep the horse under the threshold of thermal stress may help. Adrenocorticotropic hormone (ACTH) in long-acting gel form by daily intramuscular injection may give some relief in very early cases.

Monitoring fluid intake and urine output (i.e. reducing subclinical dehydration) can also be important and the use of succulent feeds may help to encourage water intake. Low protein diets can help to limit the amount of metabolic heat the horse has to cope with. High protein and high starch diets should be avoided in favour of good quality forage diets supplemented with high quality oils. Oral electrolyte supplements in block form may be helpful (especially sodium chloride and potassium chloride (Lite-Salt)).

The prognosis for affected horses is very guarded – many do not return to normal if they remain in the warmer climate. Long-term performance compromises are common in affected horses.

Prevention

Prevention is clearly much better than attempts to resolve the disorder. Careful acclimatization for up to 3 weeks or more is essential when a move involves major climatic changes. The first 5–10 days appear to be most critical and during this time the core temperature of the horse may drop and there may be decreased sweating during exercise. Long-term support may be required for affected horses – some are never normal. The thermal index (TI) obtained by adding the relative humidity (RH) of the atmosphere and the ambient temperature (in degrees Celsius) provides a rough estimate of the extent of the stress. Values over 100 are viewed as thermally stressful. For example, a climate with a relative humidity of 90% and a temperature of 30°C produces a TI of 120. A cooler ambient temperature may be a more important factor than relative humidity.

Mane and tail seborrhoea

Profile

This condition occurs sporadically and is probably the only primary form of seborrhoea that occurs spontaneously in adult horses (Fadok 1995). There is no breed, age or sex predilection. Secondary bacterial and fungal infection (*Malassezia* spp.) is likely.

The cause is not known.

Key points: Mane and tail seborrhoea

3 12 14 20 24

1. A spontaneous and persistent seborrhoeic condition affecting either the mane or tail-head or both.
2. Prominent flakes and scales are found at the roots of the mane and tail hairs. A few areas of alopecia can develop also.
3. Secondary bacterial and fungal infection (especially *Malassezia* spp.) is identified in many cases and can divert the diagnosis.
4. Diagnosis is relatively straightforward but only the distribution and persistence support the diagnosis of the primary mane and tail condition.
5. Treatment is symptomatic using anti-greasing shampoos and antibacterial/antifungal shampoos when secondary infections are thought to be present. Such treatment can improve the condition but may ultimately exacerbate it.
6. Most cases are persistent and require treatment from time to time. Even persistent and repeated treatments seldom clear the condition for any significant time.



Figure 16.15 A 4-year-old horse developed a progressive seborrhoea affecting only the mane and tail base. There was no evidence of clinically significant seborrhoea at any other body site. Attempts to treat it with a variety of topical washes and even systemic corticosteroids consistently failed to have any beneficial long-term effect.

Clinical signs

The condition is characterized by otherwise asymptomatic moderate to severe scaling at the base of the mane and tail-head (Fig. 16.15). Cases may have a dry scale (seborrhoea sicca) or a greasy scale (seborrhoea oleosa).

There may be some crusting and secondary bacterial infections may cause focal irritations and some rubbing. Secondary *Malassezia* spp. infection is common and then the skin usually has a characteristic unpleasant odour and greasy texture.

Differential diagnosis

- Dermatophilosis (*Dermatophilus congolensis*) (generalized): obvious inflammatory condition seldom restricted to mane and tail base.
- Dermatophytosis (*Trichophyton* spp.) (generalized): characteristic epidemiology and appearance of the skin in affected regions and seldom restricted to the mane and tail.
- Generalized granulomatous enteritis syndrome (sarcoidosis): associated with systemic signs but can be a possible trigger for these conditions).
- Pemphigus foliaceus: localized or generalized shedding with prominent scale and flake. Some systemic signs may be present and biopsy is diagnostic.
- Anhidrosis: characteristic history.
- Mane and tail dystrophy: breed-related mane and tail hair thinning/loss but seldom any flake or scale.
- Mercurial poisoning: extremely rare – access to toxins.
- Selenium and seleniferous plant (*Leucaena* spp.) and mimosine (*Mimosa* spp.) poisoning: tail/mane and hooves affected.
- Trauma relating to repeated tight bandaging of the tail and plaiting of the mane may cause a long-term cutaneous hypertrophic response. Similarly repeated washing in strong chemicals without rinsing effectively can cause a seborrhoeic change.
- Drug eruption: known administration of drugs in previous 3–6 months; transient changes that resolve slowly with time; seldom restricted to mane and tail.

Diagnostic confirmation

- Close examination of the skin under magnifying glass reveals accumulated scale at the base of the mane and at the tail-head. Usually this is present at both sites.
- Biopsy is not usually helpful but can show a non-specific superficial perivascular dermatitis with orthokeratotic and parakeratotic hyperkeratosis.
- Secondary bacterial and fungal infections including various staphylococcal and streptococcal species and *Malassezia* spp. can often be detected.

Treatment

Primary seborrhoea such as this condition is untreatable but can be managed to some extent. Treatment is empirical and symptomatic and requires long-term repetition.

Degreasing shampoos such as selenium sulphide and benzoyl peroxide can be helpful. Some dermatologists prescribe a wash with mild dishwashing detergents to remove the bulk of the greasiness prior to use of more expensive anti-seborrhoeic shampoos. However, over-washing with any shampoo (and the anti-seborrhoeic ones in particular) can result in long-term exacerbation, so timing of washes must be considered carefully.

Control of any secondary infections is essential – persistent infection will exacerbate the condition and such changes are likely to be persistent. Again timing of antifungal and antibacterial washes should be considered carefully.

Attempts to remove the scale by over-grooming can also exacerbate the problem.

Anagen defluxion and telogen effluvium

Profile

Anagen defluxion is characterized by dramatic hair loss deriving from interference with the anagen stage of hair growth. The simultaneous inappropriate shedding of the hair is triggered by a variety of factors including stress, fever or nutritional changes. Two forms are recognized:

(a) Telogen effluvium In this condition there is an abrupt and premature cessation of the growth phase of the hair that is triggered in many follicles simultaneously. This

is very similar in nature to the natural shedding of hairs during annual hair growth cycling. The pathological state is recognized when it occurs at an abnormal time in the annual hair growth cycle. In this circumstance there is a simultaneous shedding of otherwise normal hairs that have entered the telogen phase in a synchronous fashion at an unusual time of the year. The new growing (anagen) hairs are responsible for dislodging the old (telogen) hairs and therefore there is a considerable delay between the trigger factor and the shedding – clinicians will need to be aware that the trigger factor could have taken place up to several weeks or even months before the shedding takes place. The new hairs can usually be seen at the time or very soon after the shedding occurs. Trigger factors include periods of high fever (however short or prolonged), generalized systemic disease, 'stress' and acute severe malnutrition; however, the stress need not be severe. Telogen effluvium is less common than anagen defluxion, or perhaps at least it is less recognized.

(b) Anagen effluvium (defluxion) In this case some factor inhibits hair shaft growth/quality, resulting in a shedding of the more distal portion of the hair shaft which breaks off. Affected hairs break easily during the (growth) anagen phase and so clinical signs of dramatic hair shedding can occur within a few days of the stressor/aetiological agent. A typical example of this is the effect of antimitotic drugs such as cyclophosphamide. Other trigger factors include very high fever and other serious disease/stressors such as surgery. The defluxion takes place soon after the insult – the delays can be as little as days or a few weeks at most. Areas where there is extra pressure on the skin such as pressure points and bony prominences are affected first and this may be an important part of the history.

Key points: Anagen defluxion and telogen effluvium

10 15 16 21 24

1 Ill-defined but dramatic hair loss that follows periods of stress, fever, systemic illness or acute malnourishment. The mechanism for the conditions is unknown.
2 Anagen defluxion occurs shortly after the stressors (within days) and results from hair shaft damage while telogen effluvium develops some months later as result of synchronous (inappropriate) hair bulb cessation. In both cases there is dramatic hair loss over localized or more usually wide areas. The mane and tail hairs are spared.
3 Diagnosis is based on trichogram examination of hairs plucked from the skin without any other interference. In anagen defluxion the shafts are broken/constricted and there are no hair bulbs visible. In telogen effluvium bulbed hairs are easily epilated and are found in profusion – rather like a normal shedding process. There is usually a history of significant illness/stress or fever but sometimes this is not reported. The relative timing between the onset of the condition and the possible trigger factor is critical to the diagnosis.
4 Treatment is palliative. The new hair growth is invariably normal or near normal in both conditions but it may take a full shedding cycle to restore total normality.
5 The prognosis is good but some cases may have areas of thinner hair cover and some pigmentary changes. Prolonged trigger factor contact (e.g. continued antimitotic medication) can result in prolonged hair shaft damage in anagen defluxion.

Clinical signs

Acute onset of excessive, often extreme, hair shedding over the body following systemic disease with an elevated body temperature and/or severe malnutrition over a short time span (Fig. 16.16 and **Fig. CD16 • 8A–D**). The mane and tail hairs are usually spared because their hair cycle is asynchronous. The anagen phase is continuous and so new hair growth becomes obvious soon afterwards when the skin is examined under a magnifying glass. In anagen defluxion hair regrowth occurs soon after the insult is removed with normal cyclicity occurring at an expected time of year. In the telogen effluvium condition, the inappropriate timing of the shedding is typical and hair growth can be slower. In both conditions hair growth may already be detectable when the shedding is at its most dramatic.

Pain and pruritus are characteristically absent in both states but some horses may rub mildly to dislodge the dead hair – seemingly preferring the hair coat to be absent than dead and present.

In some cases the regrowth is of poor quality and density and there may be some scaling/seborrhoea for the following hair growth cycle. Sometimes there is a subtle change in the hair colour after such an episode.

Administration of some drugs can cause similar dramatic moulting, usually with prominent seborrhoea, and then it is usually classified as a form of drug eruption (see p. 288).

Differential diagnosis

- Seasonal coat shedding: this occurs at predictable times in the absence of any trigger factors.
- Dermatophilosis (*Dermatophilus congolensis*) (generalized): obvious inflammatory condition.



Figure 16.16 Anagen defluxion following an acute transient febrile episode some days previously. The new hair growth became obvious within a few weeks.

- Dermatophytosis (*Trichophyton* spp.) (generalized): characteristic epidemiology and appearance of the skin in affected regions; self-resolving, and fungal cultures are diagnostic.
- Generalized granulomatous enteritis syndrome (sarcoidosis): associated with systemic signs but can be a possible trigger for these conditions; tends to be more seborrhoeic.
- Pemphigus foliaceus: localized or generalized shedding with prominent scale and flake. Some systemic signs may be present and biopsy is diagnostic.
- Anhidrosis: characteristic history.
- Mane and tail dystrophy: breed related; mane and tail hairs only.
- Mercurial poisoning: extremely rare – access to toxins.
- Selenium and seleniferous plant (*Leucaena* spp.) and mimosine (*Mimosa* spp.) poisoning: tail/mane and hooves affected mainly with little effect on body hairs; access to appropriate plants.
- Fulminating abscesses: localized, painful and hot.
- Drug eruption: known administration of drugs in previous 3–6 months; transient changes that resolve slowly with time; can be very similar and difficult to differentiate because most sick horses receive some form of medication.

Diagnostic confirmation

- Close examination of the skin reveals the new growth in both cases and the history will help to establish the timing of possible trigger conditions.
- Changes to hairs detectable microscopically as irregularities of shaft diameter with narrowing and deformity indicating anagen defluxion. In anagen defluxion the hairs are all found to be irregular and dysplastic with bent shafts and distorted bulbs with dysplastic changes such as ragged points in weakened areas. Plucked anagen hairs are usually even and regular with a prominent hair bulb.
- Telogen defluxion shows hairs with a uniform shaft diameter and slightly clubbed root with non-pigmented root ends and lack of root sheath – these hairs are usually very difficult to distinguish from normal shedding telogen hairs.
- Biopsy is not usually helpful.

Treatment

Spontaneous resolution follows removal of the exciting cause of fever, etc. No medications are helpful but any underlying disease must of course be managed accordingly. Most cases receive medications and so it is difficult to know if the hair loss is a drug eruption or an effluvium/defluxion.

In cold weather conditions extra blanket protection may be advisable until the coat has restored its thermal capacity.

Idiopathic seasonal alopecia

Profile

A widely recognized but poorly documented set of seasonal hair coat variations in the hair character and density (usually alopecia) affecting middle-aged horses. Progressive annual deterioration resulting in large areas of alopecia more often following shedding of the winter coat and sometimes the summer coat. There is no evidence of a thyroid abnormality and male and female horses can be affected. The condition may reflect a delayed onset or asynchrony of the growth phase of the hair but usually shedding is driven by the new growth anyway so that explanation may not be valid. Little is known about the condition and its possible relationship to oestrogen receptors; the role of endocrinological changes and sex hormones in particular in the hair growth cycles in horses is poorly understood.

Occasional cases show an inappropriate or reversed hair growth pattern in the affected areas.

Key points: Idiopathic seasonal alopecia

5 10 16 22 24

1. A variety of ill-defined idiopathic changes in the hair coat growth patterns associated with shedding of winter or summer coats. Some are regarded as asynchrony of shedding rather than a genuine alopecia.
2. Variable extent and severity of alopecia/hypotrichosis. The changes may be alarming but usually settle after a delay. The skin is usually normal apart from the hair loss.
3. Diagnosis is made on clinical signs and history. Biopsy is not diagnostically helpful.
4. Treatment is neither established nor necessary in any case. Empirical hormonal treatment is irrational.
5. The condition may recur seasonally and may become better or worse with age.

Clinical signs

Variable areas of alopecia develop during the transitional growth phase of the winter or summer coat affecting smaller or larger areas of skin and associated with non-symptomatic failure of hair growth. The few cases encountered appear to have a progressive deterioration with sequential annual moults and whilst it can affect either the summer coat or the winter coat the latter is less commonly involved. Usually it only affects one coat type (most often the emerging summer coat) and manifests as areas of alopecia or inappropriate hair coat density over the body trunk, head and neck in spring (or autumn) (Fig. 16.17). The mane and tail and limbs are largely spared but can be thinner than expected.

Differential diagnosis

- Alopecia areata: signs are usually restricted to localized areas and usually remain static.
- Anagen defluxion: known or suspected stressor, fever or illness some days or months previously.
- Arsenic or mercury poisoning: access to toxins or 'medications' such as mercurial blisters containing these metals.
- Photosensitivity/actinic dermatitis: affects the non-pigmented skin only in horses exposed to sunlight.
- Hypothyroidism: no known/established pathognomonic signs are reported for this.
- Pemphigus foliaceus: persistent focal or generalized junctional disorder with characteristic scaling and flaking and hair loss. Typical histopathology.



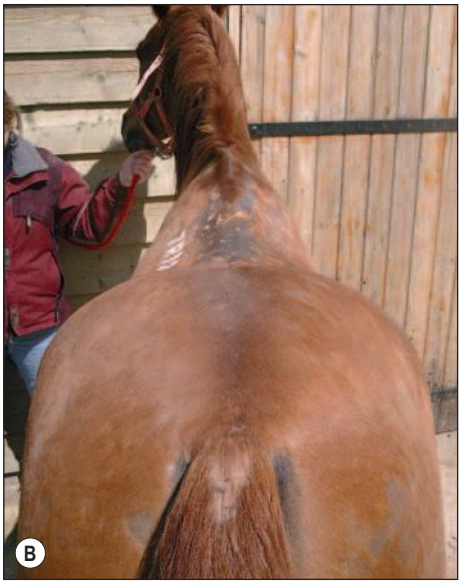


Figure 16.17 This 7-year-old mare developed a progressively severe, spring onset, alopecia affecting the body trunk only **(A)**. The condition was remarkably symmetrical **(B)**. The summer coat grew slowly but remained thin until the winter coat appeared. (Figures courtesy of John Brentnall MRCVS.)

- Generalized/cutaneous granulomatous disease (sarcoidosis): (persistent generalized debilitating multisystemic disorder featuring scaling and flaking with hair loss.
- Systemic lupus erythematosus-like syndrome of horses: persistent disorder with involvement of other organs also.
- Generalized ectoparasitic infections (lice/mites): pruritic disorders of otherwise healthy horses; parasites are easily identified.
- Drug eruptions: known administration of drugs, pruritus and scaling are commonly also present.

Diagnostic confirmation

- History is usually characteristic – photographic records are helpful as the condition is often missed in the early years.
- Biopsy of skin in affected and non-affected areas should be compared with biopsies taken from the apparently normal skin during the 'unaffected season'.

Treatment

No treatment is reported to be effective. Logical supplementation of essential fatty acids and a good mixed diet is possibly helpful. The affected horse should be kept out of possibly dangerous sunlight until the hair coat has recovered.

Idiopathic regional pigment-related follicular dysplasia syndrome

Profile

This is a rare and poorly defined alopecia disorder that affects only black-haired regions. The condition may affect localized areas but the main feature is a sharp demarcation between affected dark-skinned areas and a normal non-pigmented region. There is no information on the overall prevalence nor on breed, colour, sex and age predilections.

Histologically there is no evidence of inflammatory change and the number and character of the hair follicles is normal – there are just no hair shafts.

The condition may be related to some forms of defluxion but the restricted areas affected are most unusual.

Clinical signs

Hair loss is noticed that is restricted to black-haired areas with a sharp demarcation between the affected black regions and the unaffected white regions. Not all the black areas are affected by any means (Fig. 16.18). The skin is palpably normal and shows no pain or pruritus. Hair regrowth may become apparent after some months.

Differential diagnosis

- Dermatophytosis (*Trichophyton* spp.) before hair is lost: transient and distinctive epidemiology; organism can be identified histologically, directly and on culture.
- Alopecia areata: this is a not colour restricted and has a characteristic histopathology.

Key points: Idiopathic regional pigment-related follicular dysplasia

10 14 21 24

1. Rare alopecic disorder affecting localized areas of black skin/hair without any overt gross or microscopic pathology.
2. Temporary or permanent hair loss is restricted to black areas of variable size and number.
3. Diagnosis is made by exclusion and the absence of any diagnostic histological abnormality.
4. There is no recognized treatment – time may bring an improvement but some cases are persistent.
5. There are too few cases to make any predictions on prognosis but the horses themselves seem unaffected.



Figure 16.18 This horse developed several areas of localized black-hair-related alopecia on the face. Note the sharp demarcation between the affected black area and the normal white area. There was no history of any inflammatory change and histopathology was unremarkable apart from the absence of hair shafts.

- Contact-related alopecia (rubber/plastic/chemical): these usually have a known history of exposure and although pigmentary changes may be colour related the alopecia is not. Overt inflammation may be detected clinically or pathologically.
- Occult sarcoid: usually distinctive, slow growing and often with others of a similar type and other types. Usually some hyperkeratosis at least and palpable shot-like nodules.
- Parasitic infestation: these are invariably pruritic and not usually restricted regionally to a single hair colour type.
- Selenium/heavy metal toxicosis: not restricted to single body colour and usually affects mane and tail hairs more than body hairs.
- Idiopathic seasonal alopecia: seasonally related hypotrichosis is not colour related.

Diagnostic confirmation

- History and clinical appearance is distinctive.
- Skin biopsy shows no evidence of inflammatory changes of any sort – the skin is normal but there are no hairs.

Treatment

No treatment is warranted – time may result in normal regrowth and then the condition may recur or may never occur again.

There are too few cases reported to make any suggestions on prognosis.

Idiopathic pastern dermatitis

Profile

The diagnosis of idiopathic pastern dermatitis is difficult to justify unless and until every other alternative has been explored, although this 'condition' was recognized when the full range of conditions causing pastern dermatitis was not clear. The disorder is a chronic, long-standing and largely non-responsive pastern dermatitis. There is no age, breed, colour or sex predilection.

Key points: Idiopathic pastern dermatitis

11 14 17 21 22 24

1. A chronic, usually superficial dermatitis of the pastern and distal parts of the cannon regions. Shires and Clydesdale horses appear to be more liable to the condition and a dysregulation of cutaneous immunological responses, autoimmune reactions, and secondary vasculitis are blamed.
2. Signs are typical of chronic long-standing pastern dermatitis. Pain and pruritus are variable – usually pain is the more dominate sign. Exudation and cracking of the skin with scab formation and verrucose hyperplasia of the epidermis lead to development of multiple tumour-like masses ('grapes') and verrucose pastern dermatitis (see p. 354).
3. Diagnosis is difficult – most cases are complicated by secondary infections, self-trauma and local immunological changes. Many cases with 'grapes' are misdiagnosed as sarcoid or exuberant granulation tissue.
4. Treatment is directed at restoring the skin to a healthy state to allow it to repair. Clipping, avoidance of skin wetting and maceration, and meticulous skin and stable hygiene are essential measures. Medications must be minimized and focused. Secondary infections must be controlled and self-trauma prevented.
5. The prognosis is guarded because most cases have ill-defined causes and secondary complications.

In most circumstances these cases are presented to the veterinarian after many other attempts at treatment have failed – some of these may have been logical at the time but most will have been a panic reaction by owners or other 'advisers'. This results in chronic over-treatment and inappropriate treatment and once the changes have occurred in the skin the condition is extremely problematic. Usually the primary cause cannot be found but the term idiopathic is used to define the fact that there is no detectable primary reason.

Clinical signs

The condition is an 'unexplained' diffuse inflammatory dermatitis of the pastern and sometimes of the cannon region. White-skinned limbs are more liable and feathered legs are more severely affected in most cases. However, it can affect coloured skin and fine-skinned horses in addition.

Initially lesions are mainly found on the back of the pastern (Fig. 16.19) but this stage may not be seen. The dermatitis, which may be mildly or severely painful depending on the secondary bacteria involved or the underlying cause, extends to wider areas and can eventually involve the whole region.

One or more legs may be involved with papular lesions which rapidly coalesce and produce large areas of ulceration and suppuration (Fig. 16.20).

Long-standing cases develop a verrucose hyperplasia of the pastern skin with focal, roughly spherical nodules all over the surface (Fig. 16.21). These are sometimes termed 'grapes'. Initially they may be small and masked by the feathering on the limb but it is common for blood staining to appear on the hairs. Chronic exudative dermatitis with superficial ulceration almost invariably develops



Figure 16.19 Idiopathic pastern dermatitis affecting the heels and distal pastern.



Figure 16.20 Idiopathic pastern dermatitis (chronic form) with adherent scabs and ulcerated areas.

and then the hairs become darkly stained, greasy and matted in 'strings'. Quite commonly, affected horses rub, bite and chew at the affected areas and this results in increasingly severe ulceration, skin thickening and inflammation. Secondary bacterial infection, myiasis and self-trauma (either through rubbing or biting and chewing) causes progressively increasing inflammation and thickening of the skin until ultimately the limb is functionally disabled and secondary effects of blood loss, anaemia of chronic inflammation and plasma protein losses result in debility. Most severely affected horses have significant weight loss. Fortunately the condition is very rare but it was an important issue in poorly managed stables in the mid eighteenth century.



Figure 16.21 Severe long-standing pastern dermatitis for which a primary cause was not established. The case illustrates the hyperplastic verrucose dermatitis that represents the end stage of the condition; note the generalized skin thickening, the localized ulcerated tumour-like nodules and the exudate.

Differential diagnosis

- Pastern folliculitis (mixed *Staphylococcus* spp. infection): usually a more acute, highly painful ulcerative condition; this can be a primary state for the subsequent 'idiopathic' condition.
- Photosensitization: restricted to sun-exposed white skin of limbs and white areas of the body and head. Rapid resolution follows when sunlight is avoided. Usually hepatic in origin or primary photosensitizing toxin.
- Pastern and cannon leukocytoclastic vasculitis: sporadic disorder with characteristic distribution of inflammatory changes – lateral aspects of the pasterns and cannons of white hind legs are most often affected.
- Dermatophilosis (*Dermatophilus congolensis*): characteristic mildly exudative, mildly painful dermatitis known as 'mud fever' occurring in unhygienic conditions. Also more liable to occur in white limbs.
- Dermatophytosis (*Trichophyton* spp.): seldom affects the pastern region on its own; characteristic mycology.
- Pemphigus foliaceus: this seldom if ever affects only the cannon/pastern regions.
- Pemphigus vulgaris/bullous pemphigoid: does not affect the pastern skin on its own – invariably other sites involved and not restricted to white limb.
- Coronary band dystrophy/coronitis complex: coronary band thickening and hyperplasia with hoof wall changes.
- Verrucose pastern dermatitis/stomatitis syndrome: an acute, recurrent or persistent inflammatory disorder restricted to the coronary bands, margins of the ergots

and chestnuts and the mouth. Occasionally the pastern region becomes secondarily infected and then the signs may be more extensive.
- Contact with strong chemical irritant (disinfectants, etc.): known contact – not restricted to white limbs; can be secondary if strong chemicals are applied to an early idiopathic case.

Diagnostic confirmation

- Complete history is important (though seldom obtained); including liver function tests if lesions are limited to non-pigmented skin.
- Biopsy with immunofluorescent studies. Biopsy of the nodules of the chronic verrucose form can be misleading – features of sarcoid and infected granulation tissue can sometimes be found.
- Bacterial and fungal culture. It is important to check for dermatophytes such as *Trichophyton* spp. and bacteria such as *Dermatophilus congolensis* and staphylococcal and streptococcal organisms. These may be primary causes or secondary especially in unhygienic conditions. *Malassezia* spp. infection is a common secondary complication.

Treatment

As is always the case with pastern dermatitis of whatever origin, the first approach is to remove any adherent scabs, crusts and exudate-matted hair. The area can be soaked with a mild antiseptic solution prior to clipping and removal of scabs because sometimes the process of removal is very painful. Where matted scabs and crusts are present, these may be softened by using a hydrogel-based dressing for 24 hours under a light bandage. Modern silver hydrofibre dressings (a good example is Aquacell Ag (Convatec, UK)) applied moist are useful for both the softening of the scabs and control of bacterial infections.

Once the area has been cleaned and clipped closely, it should be gently washed with warm water and an antiseptic surgical scrub such as chlorhexidine. If *Malassezia* spp. infection is suspected, washes with added miconazole (such as Malaseb, Leo Laboratories, UK) can be used.

Probably the most important aspects of the treatment are:

1. To ensure that all scabs/crusts and hair are removed.
2. To ensure that the skin is washed with a suitable antiseptic in *warm* water and then rinsed thoroughly again with *warm* water.
3. The area should be blotted dry with paper towels – not rubbed dry.
4. The treated skin should be left open and kept dry unless deep infection is suspected and then a silver dressing can be applied.

Note

It is very important to protect the area from unhygienic conditions or contact with irritant chemicals and high ultraviolet light conditions, and to maintain the skin in a dry state.

Emollients containing antifungal, anti-inflammatory and antibacterial lotions (triple anti-infective compounds) are commercially available and are sometimes useful if applied for a short period. However, systemic antibiotics are not generally useful.

Corticosteroid creams are commonly used but do little to help the long-term health of the skin. Short-term application can be helpful but it may be better to use systemic prednisolone if the condition warrants anti-inflammatory treatments. High doses of corticosteroids may be indicated where immune complexes and immune-mediated vasculitis are involved such as in the long-standing verrucose pastern dermatitis syndrome of Shire and Clydesdale horses.

Harsh remedies are harmful and often exacerbate the condition and they often encourage the development of verrucose pastern dermatitis, which probably represents the end stage of the condition but which is usually classified as a separate entity (see below).

The prognosis is good if the condition is early but poor if there is any verrucose hyperplasia (see below). In the latter cases the best that can be achieved is management. Harsh surgical treatments and cryosurgery, etc. are contraindicated.

Prevention is far better than cure and whenever the condition is seen, stable management must be addressed. The management of feathered limbs is a difficult but essential part of the care of susceptible horses. Prevention of mite infestations and even hair matting in the absence of any overt infection is essential.

Idiopathic dorsal hock and palmar carpal dermatosis ('mallenders and sallenders')

Profile

This is a common but poorly understood disorder characterized by scaling and hyperkeratosis restricted to the dorsal hock and palmar carpal regions. It is particularly prevalent in feathered breeds and especially the Shire, Clydesdale and Welsh Cob breeds, and has been recognized historically for many years. The historical name is 'mallenders' (the condition on the back of the carpus) and 'sallenders' (the condition on the dorsal aspect of the tarsus). Horses of all ages are affected to varying degrees.

Key points: Idiopathic dorsal hock and palmar carpal dermatosis



1. A common but poorly understood disorder affecting adult horses of heavy and feathered breeds.
2. Signs include prominent flaking and scaling restricted to the dorsal tarsal and palmar carpal regions. Alopecia, pruritus and inflammation are usually not encountered but may indicate secondary parasitic or bacterial infection. Pain is not a feature but exudative dermatitis can develop.
3. Diagnosis is made on the signs and signalment of the horse alone. Biopsy is largely non-specific.
4. Treatment with keratolytic shampoos and synthetic retinoids may help temporarily but may exacerbate the condition in the longer term. Ectoparasite control is essential.
5. The condition is best left alone if possible and then does not usually worry the horse.

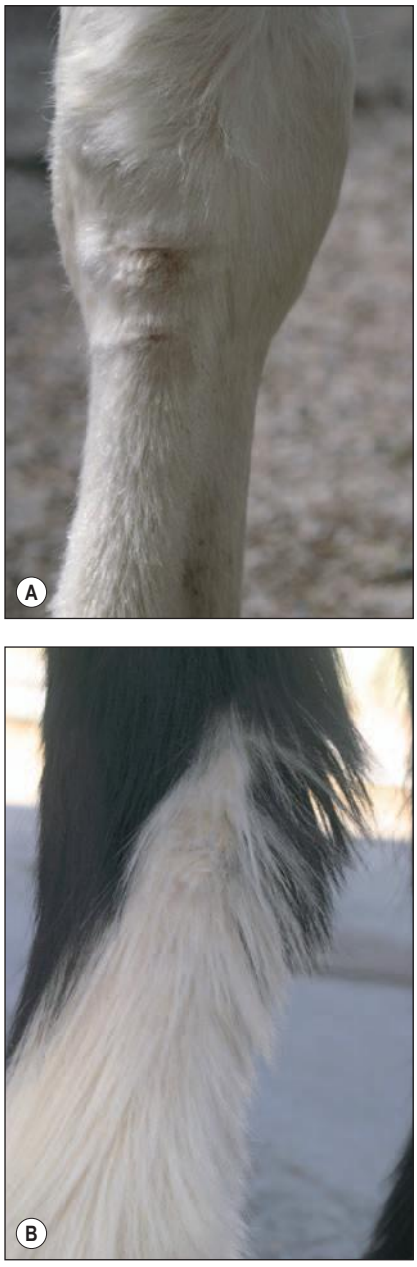


Figure 16.22 Early mallenders lesions on the back of the carpus of two cases. **(A)** A case showing the typical non-inflammatory, hyperkeratotic scaling. **(B)** A case in a heavy horse with more alopecia and skin thickening but less scale. This was hardly noticeable.



Figure 16.23 This 8-year-old Shire mare had a 6-year history of scaling and some pruritus on the dorsal tarsus **(A)** and palmar carpal **(B)** regions. From time to time the lesions became more inflamed and exudative. No treatment was effective but the condition was managed effectively by close clipping and careful washing with keratolytic shampoos. The owner was relieved to know that little could be done and accepted the condition, which did not have any adverse effects on the horse itself.

It is suggested that it is a reactive or secondary disorder to recurrent episodes of pruritus attributed to mite infestations. However, even severely infested horses seldom rub these regions specifically and it seems more likely that it is a form of naevus and that the extra scale attracts mites to the region.

Clinical signs

Characteristic scaling and flaking without pruritus or pain in the dorsal hock and palmar carpal regions. The affected area can be small or large and the edges are poorly defined. Early or mild lesions are often best felt rather than seen but even in these cases the animal may rub or chew at the site and so the hair becomes damaged and distorted (Figs 16.22 and 16.23).

Horses that show pruritus may be secondarily affected with chorioptic mange (see p. 191). The extra scale may provide a focus for the mites so they are a common complication.

A few cases become ulcerated and crusting can develop (Fig. 16.22) with secondary infection (often

with *Dermatophilus congolensis* or various opportunistic pathogens).

Differential diagnosis

- Linear naevus (this term could be applied to this condition): benign usually non-progressive scaling or thickening of the epidermis.
- Linear keratosis/Linear alopecia: localized to single defined linear lesions of hyperkeratosis/alopecia on the sides of the neck or body trunk.
- Occult/verrucose sarcoid: invariably other lesions and in any case this site is a rare focus for occult or verrucose sarcoid. Sarcoid is progressive in most cases and there are usually other types within the affected skin.
- Zinc-responsive dermatosis: extensive exfoliative alopecic disorder that has few other symptoms. Mainly the limbs and ventral abdomen involved.
- Pemphigus complex: bullous disorders resulting in focal or extensive/generalized scaling and crusting dermatosis; diagnostic histology.
- Dermatophilosis: infectious, inflammatory disease affecting the back and limbs in poor (wet) conditions. Characteristic organism identifiable in smears from pus or crusts.
- Rubs/self-trauma: hair breakage is a feature.
- Over-strength or inappropriate medicated washes: history of applications, usually not restricted to one localized area.

Diagnosis

- Clinical features and history are typical but can be masked by secondary signs (associated with pruritus and infection) and can be easily overlooked.
- Biopsy is rather disappointing with mild epidermal hyperplasia and an orthokeratotic (anuclear) hyperkeratosis. A few cases have evidence of a cellular response such as lymphocyte or histiocyte accumulations.

Treatment

Treatment is neither possible nor warranted even when owners are concerned about the condition. Attempts to treat it specifically may lead to further scaling and a more aggressive epidermal change. Clipping of the overlying hair may help. Control/elimination and prevention of secondary parasitic infestations, which may lead to pruritus and exacerbation, are useful and sensible approaches. Secondary bacterial infection is uncommon unless the area is self-traumatized.

Keratolytic shampoos such as selenium sulphide or coal tar shampoos, etc. may reduce the evident scaling and flaking and reduce the thickness of the skin in the affected area.

Topical retinoids and photodynamic therapy can be useful but usually cause overt inflammation and so the skin responses may result in exacerbation. Repeated applications of retinoids may help in the short term but the treatment also causes hair loss and localized inflammation that may be painful.

Verrucose pastern and cannon dermatopathy ('greasy heel' syndrome)

Profile

This is a loose term applied to a well-recognized chronic pastern dermatitis which has been reported historically for many hundreds of years. The syndrome is a chronic hyperplastic inflammatory dermatopathy and, although it is regarded as a distinct clinical condition within the broad description 'pastern dermatitis', it is probably not a specific disease entity. Rather it is probably the end stage of many inflammatory diseases which have similar clinical features including scaling, crusting, erosion, exudation and pyoderma of the pastern and heels of horses. Remarkably it affects white-skinned legs far more frequently than coloured legs but there is no known explanation for this. As it is a common end stage disease it is hard to define a single specific cause. Heredity, management, infection and infestations causing inflammatory skin conditions have been implied. It is a major entity in the pastern dermatitis group of diseases (see p. 471).

The Shire, Clydesdale and Welsh Cob breeds are overrepresented and there is a broad agreement that the condition in these breeds at least is probably immunologically instigated, exacerbated or prolonged. In fact these breeds and other susceptible ones such as the Irish Draught are also liable to several other causes of pastern dermatitis as are the 'feathered' breeds and so there may be a complex aetiology.

There is no apparent sex predilection but older horses are more liable to be presented with the condition in an advanced state. However, the disease occurs almost exclusively in cold-blooded and heavy horses and, in particular, it is probably more prevalent in long-haired (feathered) breeds and draught horses. Horses with a high pastern circumference and tufted fetlocks are reported to be most liable and most severely affected (Geburek et al 2005a).

One pastern, frequently a hind leg, is often involved and may also be 'white socked', but it can affect all four legs and pigmented skin in some cases. The condition has been divided into four clinically recognizable categories (Geburek et al 2005b):

- Group 1: scaling dermatosis – the earliest stage of the condition.
- Group 2: hyperkeratotic, hyperplastic plaque-like dermatosis. This arises as a result of self-trauma or infection.
- Group 3: tumorous skin thickening; initially thickening is generalized and there may be a degree of skin folding.
- Group 4: verrucose lesions with nodules of hyperplastic, ulcerated and exudative thickened skin and scar tissue often with extensive skin folding and deep clefting. Secondary infection and infestations are common.

Many cases have early signs such as scaling, pruritus and plaques resulting from vasculitis, self-trauma and infection that are either overlooked or ignored or treated badly/inappropriately.

Key points: Verrucose pastern and cannon dermatopathy



1. A historically well-recognized disorder of the distal limb regions of heavy horses and others with feathered, white-skinned legs in particular. The condition tends to be progressive with time and can be exacerbated by self-mutilation. Immunological, genetic and persistent self-trauma from pruritus are suggested as the main features of the pathogenesis.
2. Clinical signs are variable thickening of the skin, hyperplastic nodular formations and varying exudative matting of the hair coat. There may be pre-existing pruritus or the pruritus may follow the onset of the condition. Often, the affected leg(s) will become massively thickened and then secondary bacterial infection and fly maggot infestations can occur. The horse may or may not be lame. Debility and weight loss may become significant.
3. Diagnosis is usually simple but some aggressive sarcoids and pyogranuloma are possible differentials.
4. Treatment is extremely difficult because most cases are presented late in the course. Skin hygiene and close clipping of the hair are absolutely essential even in early milder cases. Systemic and topical antibacterial treatment, control of ectoparasites and fly contact are helpful measures. The skin must be kept as dry as possible and maceration and continuous wetting must be avoided. Corticosteroids may be indicated but the heavy horse is not a good candidate for long-term high-dose corticosteroid treatment because of the concurrent risks of laminitis (although these may be grossly overstated). Prevention of ongoing trauma (self-inflicted or otherwise) is critical.
5. The prognosis is very poor for restoration of a normal leg in anything other than the mildest cases but with care some are manageable and can live useful lives. Secondary complications reduce the outlook considerably. Stable management and hygiene must be addressed.

Clinical signs

The clinical signs are common to the many aetiological factors suggested. The condition usually starts at the posterior aspect of one (often white-skinned) pastern but, with time and inadequate therapy, may extend up to the fetlock and around the front of the leg – often to involve the fetlock region and the cannon. Usually white legs are more affected than black and sometimes the black legs are totally unaffected while the white-haired regions are severely affected (Fig. 16.24).

Initially there may be a focus of inflammation either as a direct consequence of skin infection (see above) or due to self-trauma resulting from pruritus. Although there may be some scaling and even some superficial excoriation of the skin with limited crusting, this stage is seldom presented for investigation.

Erythema, oozing, crusting, alopecia, with vasculitis develop quickly and lead to erosion and ulceration of the skin with progressively more serious exudation.

In chronic (long-standing) cases the skin becomes thickened and may develop significant (often extreme) cutaneous hypertrophy and deep fissures/clefts (Fig. 16.24C, D). Often there is a fetid exudative dermatitis with localized or more generalized areas of wart-like exophytic outgrowths and calluses. Severe chronic lesions may lead to chronic granulation tissue (so-called 'grapes') (Fig. 16.24E). Lesions are characteristically painful when secondary infection or maggot fly infestation takes place and chronic thickening and inflammation result in lameness and limb filling.

Secondary bacterial infections and even myiasis are common and some cases are pruritic in addition, which adds further to the trauma of the skin. Some affected horses will be distressed by the condition (whether overtly infected or not) and may bite or chew at the affected legs.

Metabolic effects can occur including hypoproteinaemia/hypoalbuminaemia (as a result of plasma exudation) and anaemia of chronic inflammation. Weight loss is common. The disease is fortunately rare but can be a significant welfare issue.

Differential diagnosis

- Contact dermatitis: primary irritant or allergic contact dermatitis; irritant substances on pastern; contact allergy (plants and other substances); chronic exposure to mud, unhygienic conditions (usually all four limbs are affected).
- Pastern folliculitis/pyoderma infections (staphylococcal infections/botryomycosis; dermatophilosis; fusiformis dermatitis): usually painful, exudative and ulcerative pastern skin inflammation – can develop into the verrucose form if neglected.
- Chorioptic mange: very pruritic and contagious; can be a primary cause of verrucose pastern dermatopathy.
- Photosensitization: systemic (phylloerythrin); primary or hepatic related photosensitization involves white pasterns and other white areas on the face, body and limbs etc.).
- Idiopathic pastern dermatitis: possibly an early stage of this condition.
- Vasculitis (pastern leukocytoclastic vasculitis/immune-complex vasculitis/purpura haemorrhagica): usually restricted to the outer aspects of the white-skinned limbs – thin-skinned breeds are more liable.
- Mycetoma: deep fungal infection of the pastern skin is rare but could be a primary cause of this condition.
- Lymphangitis/cellulitis: these arise as result of disseminated extending infection in the distal limb regions in particular. Severe/moderate pain and obvious inflammation occur acutely. Chronic long-standing cases can be similar but for the most part the skin is normal.
- Fibroblastic or verrucose sarcoid: defined sarcoid types – rare to have verrucose sarcoids on the limbs and the hyperplastic nodules (grapes) have a different appearance and histological features.

Diagnostic confirmation

Diagnosis is difficult as secondary changes are often severe. The tests outlined below are all rather non-specific and may not provide a positive diagnosis. Nevertheless negative results can also be useful.

- Swabs for culture to define infectious types and secondary bacterial contaminants.
- Scrapings for parasites (may be found but may be extensive secondary changes). It is also useful to look at other predilection sites.



Figure 16.24 This series of pictures illustrates the progression of the disease in heavy horses. The disease may take many years to develop into the end stage but in some cases progression can be very rapid. **(A)** An apparently unaffected feathered leg. **(B)** Close examination, however, revealed some thickening and hyperkeratosis with seborrhoeic changes. **(C)** Pruritus resulted in some skin ulceration and bleeding. **(D)** Chronic skin thickening revealed by clipping of the legs. Note that in this case the legs are black. **(E)** A severe case of verrucose pastern dermatitis in a mature Shire horse. All three white feet were affected and the single black limb was mildly affected. (Fig. 16.24E courtesy of World Horse Welfare.)

- Biopsy (usually identifies non-specific chronic dermatitis). Biopsy of the nodules ('grapes') may reveal features which may on casual examination look like sarcoid or granulation tissue. There is no reason why both of these conditions should not coexist.
- Ultrasonographic and radiographic examination of the limb(s) may be useful if extensive thickening of the skin is present to eliminate other causes of limb thickening (e.g. lymphangitis/cellulitis).
- Investigate systemic involvement by haematology and biochemistry.

Treatment

All identifiable causes and exacerbating factors (such as infection, infestation, trauma, maceration, etc.) should be eliminated as far as possible. Although the underlying primary cause should be treated, it is often impossible to establish this. The almost invariable bacterial infection and the occasional myiasis will require specific treatments. Treatment of myiasis may be problematic because the condition can develop rapidly and repeatedly and because the maggots can penetrate deep clefts and so may be difficult to identify and remove.

Note

The main initial objective is to reduce the exacerbating secondary factors and to render the skin inhospitable to the opportunistic pathogens that may complicate the condition.

Topical antiseptic washes (e.g. chlorhexidine possibly with miconazole) are essential, initially with careful removal of all hair and necrotic tissue (this may require general anaesthesia as it can be very painful and access to four

feet may be difficult in the conscious horse). It is vital that all washes and rinses are undertaken with warm water – cold water washes are far less effective.

Once washed, the limb(s) should be blotted (not rubbed or towelled) dry and kept dry thereafter as far as possible. Reduction in the overall degree of inflammation is the best overall approach but this can be difficult to sustain.

Aluminium oxide powder and zinc sulphate and lead acetate solution BPC (White Lotion) have strong drying properties and reduce the overall maceration of the skin which arises from the exudate and the poor superficial immunological defences. Strong desiccating chemicals such as alcohol and astringents may cause cracking and further exudation so they are probably contraindicated.

Non-steroidal anti-inflammatory drugs and antibiotics (topical and systemic) may be indicated. Thick creams and lotions should not be applied if avoidable. Water-soluble-base steroid creams have been used but at least initially should probably be avoided.

Many cases take years to recover fully and some have permanent skin thickening and a tendency to recurrence.

Cases presented in the advanced stages of the condition are genuine welfare concerns – the condition has been recognized for centuries by stable hands, army cavalry stables, etc. as a preventable disorder provided that exemplary care is provided from a young age in susceptible breeds. The failure to provide proper hygiene and the failure to treat the early signs should be viewed as neglect.



Figure 16.25 A dermal choristoma which developed over some months. Surgical excision resolved the cosmetic problem.

Choristoma/hamartoma

Profile

These are rare causes of cutaneous and subcutaneous nodule formation. A choristoma is a tumour-like accumulation of normal tissue cells with a normal histological appearance at an abnormal location. A hamartoma is a focal malformation that resembles a neoplasm but which results from faulty development in an organ such as the skin or muscle, in which there is an improper mixture of tissues usually with excess of one component. An example of a hamartoma is the epidermal naevus and vascular hamartoma in young foals. Some may be present at birth or develop in young animals while the choristoma group tend to develop in older horses.

Key points: Choristoma/hamartoma

3 9 14 20 23 24

1 A focal, benign, non-inflammatory, neoplasm-like accumulation of either a normal tissue type in an abnormal location (choristoma) or abnormal tissue mix in a normal tissue location (hamartoma).
2 Various possibilities exist depending on the tissues involved. Epidermal naevus and cavernous vascular haemangioma/hamartoma represent the hamartoma group whilst choristomas tend to produce subcutaneous nodules.
3 Biopsy will reveal either an abnormal mixture of tissues in an organ with predominance of one type usually or an abnormal tissue type for the location. In both cases there is no evidence of neoplastic changes.
4 Treatment will depend on location and pathology. In many cases, however, the conditions are totally benign and create cosmetic problems rather than a medical or surgical one.
5 The prognosis is excellent as the animal usually copes well and many are good surgical candidates.

Clinical signs

The signs will of course vary with the tissues involved and the type of response but both groups tend to cause tumour-like accumulations in a particular location (Fig. 16.25). The signs may progress with time but may be static or very slowly progressive. They should not resolve spontaneously.

Differential diagnosis

- Sarcoid (nodular and verrucose forms in particular): usually not single and typical static or slowly expanding nature with transition to other types; typical histology.
- Nodular skin conditions (see p. 455).
- Seborrhoeic skin disorders (see p. 459).

Diagnostic confirmation

The tissue type involved in both cases is usually very obvious and the absence of any neoplastic changes supports the diagnosis. The pathologists will usually confirm the basic tissue type and so may express the diagnosis as vascular hamartoma or muscular choristoma, etc.

Ultrasonographic examination may be useful if extensive thickening of the skin is present.

Treatment

Surgical removal may be feasible in some cases but superficial hamartoma on the skin usually occurs in locations where excision is impractical. The specific requirements of the case and the diagnosis will dictate the type of treatment employed or the benign neglect of the condition.

Intermammary intertrigo/irritation

Profile

Intertrigo is defined as a dermatitis that develops as result of intimate contact friction between two skin surfaces. In the horse the condition is rare but can develop when there is abnormal swelling or exudation, for example in the groin or elbow regions. Non-lactating mares commonly develop accumulations of skin debris between the mammary glands and a few such cases are irritated by this whether or not there is any overt inflammatory skin disease. The accumulation may take months or even years to develop.

Areas affected with ulcerative intertrigo or simple accumulation are liable to develop secondary bacterial and *Malassezia* spp. infection, especially where sebum, desquamated skin cells, and glandular secretions such as milk or exudates accumulate. As the skin becomes inflamed and thickened the pressure between the two opposing surfaces increases and so the condition becomes progressively worse.

Clinical signs

The commonest syndrome is one of intermammary accumulation and consequent perineal irritation manifest by perineal pruritus (Fig. 16.26; see also Fig. 7.12).

Where the skin is patently inflamed and eroded the signs are more obvious and exudate and infection are more significant.

Differential diagnosis

- *Oxyuris equi* infestation: usually eggs can be identified; the condition does not affect horses that have received regular avermectin wormers.

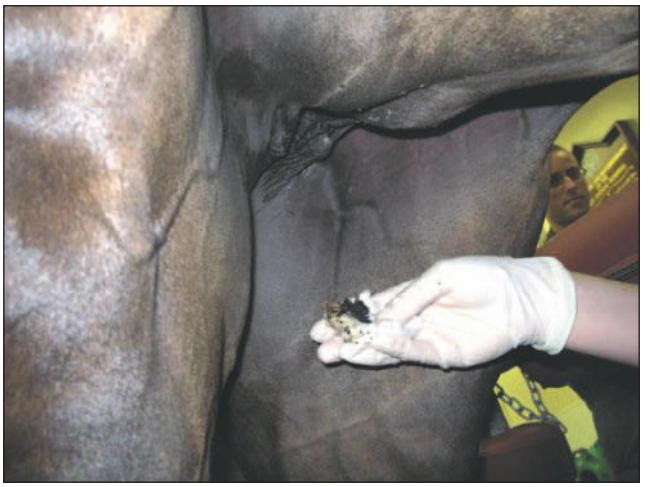


Figure 16.26 This mare had shown prolonged perineal pruritus – never sufficient to cause any significant self-trauma but very irritating for both horse and owner nonetheless! Following removal of the accumulated debris and a gentle clean of the area, the mare showed no evidence of any perineal irritation (see Fig. 7.12).

- Lice and surface mite infestations: usually other regions are affected also and the parasites can be identified easily.
- Insect bite hypersensitivity: characteristic seasonal condition affecting the mane and tail and sometimes the ventral midline.

Diagnostic confirmation

- Physical examination: all mares with perineal/hindquarter pruritus should be examined for this condition. Removal followed by resolution of the signs is diagnostic!

Treatment

Spontaneous resolution follows removal of the material.

Key points: Intermammary intertrigo/irritation

14 18 22 24

1. A common dermatological state with a rare but important clinical manifestation.
2. Affected mares are irritated and show perineal pruritus/itch. Mares usually itch under the tail rather than at the tail base and may produce an over wound. A few cases develop a more overt dermatitis (true intertrigo) and secondary bacterial and fungal infection. Some geldings may show similar signs as a result of a sheath infection or a smegma bean in the urethral fossa.
3. Diagnosis is usually simple. In all mares that present with perineal irritation, it is always worth checking the intermammary region for detritus and accumulated skin debris.
4. Treatment is equally simple where there is no overt skin erosion. Removal and a single wash with an antiseptic solution followed by careful rinsing and drying is usually enough. Where there is overt erosion the problem can be much more persistent and difficult. Topical corticosteroids and soothing creams can be very helpful. Movement should be restricted and the skin problem treated.
5. The prognosis is good but some cases may have some pigmentary changes.

References

Fadok V A 1995 An overview of equine dermatoses characterised by scaling and crusting. Veterinary Clinics of North America. Equine Practice 11:43–53

Geburek F, Deegan E, Hewicker-Trautwein M, Ohnesorge B 2005a The development of verrucose pastern dermatitis syndrome in heavy draught horses. Part 1: Review of the literature. Deutsche Tierarztliche Wochenschrift 112:211–214

Geburek F, Deegan E, Hewicker-Trautwein M, Ohnesorge B 2005b Verrucose pastern dermatitis syndrome in heavy draught horses. Part 2: Clinical findings. Deutsche Tierarztliche Wochenschrift 112:211–214

Hubert J D, Beadle R E 1998 Equine anhidrosis. Compendium for Continuing Education 20:846–849

Hubert J D, Norwood G 2002 Equine anhidrosis. Veterinary Clinics of North America. Equine Practice 18:355–369

Jenkinson D M, Elder H Y, Bovell D L 2007 Equine sweating and anhidrosis. Part 2: anhidrosis. Veterinary Dermatology 18:2–11

Meijer W C P 1962 Vitiligo in seven horses and five cattle. Tijdschrift voor Geneeskunde 87:411–425

Naughton G K 1986 Antibodies to surface antigens of pigmented cells in animals with vitiligo. Proceedings of the Society of Experimental Biological Medicine 181:423–427

Pascoe R R 1973 The nature and treatment of skin conditions observed in horses in Queensland. Australian Veterinary Journal 49:35–40

Peter J E, Boge P, Morris P G et al 1981 Anhidrosis in a thoroughbred. Veterinary Medicine for the Small Animal Clinician 76:730–732

Rothwell T L, Birch C B 1991 Unilateral papular dermatitis in a horse. Australian Veterinary Journal 68:122–123

Scott D W 1988a Unilateral papular dermatosis In: Large animal dermatology. WB Saunders, Philadelphia

Scott D W 1988b Dermatohistopathology. In: Large animal dermatology. WB Saunders, Philadelphia, pp 29–49
Stannard A A 1987 Hyperaesthetic leukotrichia. In: Robinson N E (ed.) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, pp 164–169
Warner A E, Mayhew I G 1982 Equine anhidrosis – a survey of affected horses in Florida. Journal of the American Veterinary Medical Association 180:627–629
Warner A E, Mayhew I G 1983 Equine anhidrosis; a review of pathological mechanisms. Veterinary Research Communications 6:249–252
Williams P D 1995 Lumps, bumps and swellings – a clinical approach to diagnosis. Equine Veterinary Education 7:300–303
Yu A A 1997 Dermatologic conditions associated with abnormal pigment. In: Robinson N E (ed.) Current therapy in equine medicine, 4. WB Saunders, Philadelphia, pp 391–393

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17

Injuries and diseases of the hoof

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The hoof and its components can be regarded as special appendages of the skin. Various conditions caused by environment, injury, neglect or poor farriery practices may cause changes to the wall, sole, frog and/or coronary tissues. These changes may also be related to physical, infectious, neoplastic or genetic factors. It is difficult to categorize the various conditions as they often coexist and many have a common aetiology. For example, a sole crack may lead to an abscess with an under-run sole or wall and then break out at the coronet, thereby involving at least three different hoof structures in different ways.

Clinical examination

The horse may be presented for lameness or for an actual foot condition, so it is necessary, as for any other skin condition, to carry out a full clinical examination of the patient before a detailed examination of the foot. Before the foot can be examined it must be thoroughly cleaned and the shoe may have to be removed. A full set of sharp hoof knives, a hoof pick and a wire or stiff bristle brush capable of penetrating the cleft of the frog should always be available. In any event, the wall, sole and frog should be scrubbed clean and all dead, discoloured and damaged horn and frog material should be removed from the sole in particular. However, if the tissue is patently normal it is unwise to remove excessive sole, wall or frog. The periople or outer waterproof surface of the wall should be interfered with as little as possible as it may result in drying and cracking.

The sole should be palpated using digital pressure and hoof testers, starting at the toe and working laterally and medially. Pressure can be applied to the frog and across the heels from frog to wall. Squeezing gently across the heels (medial and lateral) from the back of the foot applies pressure to the plantar/palmar structures. The wall should be palpated carefully and its temperature assessed in comparison with the contralateral limb. Gentle tapping with a small round hammer is a good test of wall pain, particularly over shoe nails which might be causing problems. Evidence of bruising, haemorrhage or abnormal tissues (both in appearance and texture) in the sole wall or frog should be noted. The white line should be carefully examined for breadth, normality of horn tubules and bruising/haemorrhage.

Genetic defects of the hoof

Thin walls and soles

These are characteristic of certain breeds and genetic lines of horse. They usually appear together and create difficulty in shoeing. Thin walls often lead to pricking, nail binds and wall-breakout during shoeing which make successive shoeing increasingly difficult. Thin soles are more liable to bruising and concussive injuries such as haematoma formation. Repeated bruising of the soles increases the opportunities for foot infections and corns. This has been blamed for some cases of pedal osteitis. While horses are in training, great care has to be given to the method of shoeing and the placement of the nails.

Treatment

Whilst resting the horses in rocky, rough hill country may produce a harder foot, it seldom corrects the original defect – indeed it may sometimes cause serious break-back of the wall. Thin-soled horses benefit from polypropylene pads or a thin 2–3 mm layer of acrylic filler applied over the whole solar surface. Chrome-treated leather has also been glued with contact adhesive to the soles under the shoes. Glue-on plastic shoes help considerably by avoiding the need for any nails at all but they are not as durable and the methacrylate adhesive often loses its strength if allowed to get wet for long periods. Acrylic hoof support bandages applied around the wall before shoeing assist some horses and can be used to provide extra hoof strength. Daily supplementation with biotin and methionine may improve the

quality of the hoof structure but it is not usually curative on its own.

Flat soles

Horses which do not have normal concavity of the sole without any pathological reason are classified as having flat feet. This can be described as a developmental/genetic condition. It occurs more often in some lines of Thoroughbred horses in particular.

Treatment

There is no effective medical option and therapy is limited to placement of well-seated shoes and avoidance of hard or rough going. Polypropylene or leather anti-concussion pads can be applied to help cushion foot impact.

Coronary band (dysplasia) dystrophy

Profile

An idiopathic defect in cornification of the coronary band which affects all four hooves more or less simultaneously and equally. Mature horses of draught breeds seem most susceptible and most cases are reported to be affected from a young age; the signs may not be severe and may easily be overlooked in the growing horse.

Key points: Coronary band (dysplasia) dystrophy

2 12 14 20 23 24

1. A sporadic rare disorder of the coronary band largely restricted to heavy draught breeds. Possibly therefore some genetic aspect.
2. Proliferative, scaling condition of the coronary band only; the pastern skin is usually unaffected. Possibly some consequent alterations in the hoof quality, particularly with loss of periople and a rough, crumbly hoof wall quality.
3. Diagnosis is made by eliminating other causes of coronary band disease and inflammation.
4. Treatment is difficult. Restoration of normal keratinocyte function may be possible with retinoids.
5. The prognosis for resolution is poor – ongoing management and hoof difficulties are almost inevitable. Some horses cope well for many years.

Clinical signs

Onset is usually insidious and often the first sign of a problem is the poor hoof wall quality. All four coronary bands show progressive proliferation and hyperkeratotic changes with extensive scaling and have a greasy feel. There are usually obvious hoof wall changes in severe or prolonged cases (Fig. 17.1).

There is no pain or pruritus unless secondary infection develops.

Open lesions are often present with cracks and fissures which can bleed or ooze serum (Fig. 17.2). The ergot and chestnut are seldom affected similarly.

Differential diagnosis

- Laminitis (acute forms)
- Pemphigus foliaceus
- Dermatophilosis (*Dermatophilus congolensis*)
- Generalized granulomatous enteritis syndrome
- Sarcoidosis
- Selenium toxicosis
- Verrucose sarcoid
- Pastern folliculitis.

Diagnostic confirmation

- Clinical signs, elimination of all other causes
- Non-specific poorly defined histopathology.

Treatment

Only palliative measures are possible; removal of excessive horn and application of emollient creams to affected tissues at coronet. Topical retinoids such as tazarotene may usefully but temporarily restore normal keratinocyte function. Systemic or topical corticosteroids have no material effect.

Mild counterirritants applied to the coronet may be helpful in encouraging healthy coronary tissues but equally may make the condition worse. Dietary supplementation with evening primrose oil, biotin and methionine may be useful.

The owner must be apprised of the chronic nature of the disease. Regular attention to the feet and early intervention if secondary infection develops are important in the long and short term.

Coronitis

See page 473.



Figure 17.1 This middle-aged part Irish Draught gelding had a long history of poor hoof quality **(A)** and showed the typical signs of coronary band dystrophy on all four feet. Note the scaling and thickening of the coronary band **(B)** and the generally poor hoof wall quality with flaking and horizontal lines of abnormal horn growth. Secondary inflammatory changes may be found at the coronet or over the heels **(C)**.

Wall/toe/quarter/heel/vertical or transverse hoof wall cracks

Profile

These are characterized by fissures in the hoof capsule. Coronary band injuries are a common cause of clinically significant secondary cracks and horn defects. Wall, toe and quarter cracks have a defect which is parallel to the laminae, while transverse cracks are at right angles to the laminae. Most cracks of the former type are superficial and due to prolonged desiccation or to trauma/defects of the coronary band. Deep cracks may extend into the sensitive laminae (Fig. 17.3).

Toe and quarter cracks These are mostly due to neglect from allowing the hoof to dry out, or improper dressing to reduce 'winging' of the quarters and heels. They may also arise from sudden turns on rough ground or injuries to the coronary band (Fig. 17.4).



Figure 17.2 Coronary band dystrophy.



Figure 17.3 Deep toe crack extending from solar margin to coronary band and down to sensitive tissue.

Transverse cracks Coronary band injuries or infections spreading up the white line from sole abscesses are usually responsible for these. They only become serious if they are extensive and when they have nearly grown out at the solar margin (Fig. 17.5).

An interesting condition occurs where there is repeated localized bulla formation usually at the dorsal aspect of the coronary band. The condition is pain-free and unexplained. It results in a localized hoof wall deficit that grows down some way before another focus develops. The syndrome is not usually recognized except by farriers (Fig. 17.6).

Sole cracks These are caused by a combination of excessive drying of the sole and standing on rocks or other hard objects in paddock/grazing horses (Fig. 17.7). They are not commonly observed in shod horses.

Heel cracks Cracks in the wall of the heel usually have two fairly distinct causes: (a) coronet injury leading to a persistent defect in the coronary band and so to a wall deficit; (b) bearing-surface injuries which seem to occur more frequently from poor heel support. This is encountered where the angle of the heel becomes more and more acute, leading to tearing of the laminae (Fig. 17.8).



Figure 17.4 Quarter crack extending from solar margin to coronary band.



Figure 17.5 Transverse crack.

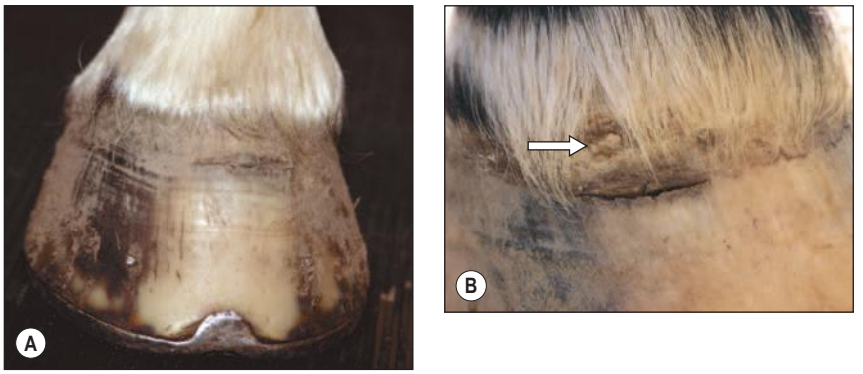


Figure 17.6 This Warmblood gelding had a regular focal blistering of the dorsal coronary band without obvious pain or discomfort. The consequent horn defect gradually grew down the hoof **(A)** but other episodes occurred at irregular intervals. **(B)** Note the rather poor quality hoof more proximal to the hoof wall defect (arrow) indicating that the primary condition actually caused more extensive damage. The syndrome has not been investigated fully.



Figure 17.7 Sole crack (arrows).

Clinical features

Superficial cracks are not often associated with lameness. Deep cracks with pressure on the sensitive laminae may cause lameness. Haemorrhage or purulent discharging sinuses and foci with movement of the wall have serious implications. Working the horse may cause bleeding from the area and, in paddock/grazing horses, pus is often associated with neglected hoof injuries.

Heel cracks are often painful and contact with the ground is too far forward compared with the horn at the coronary band. The cracks are often deep and easily bleed and/or become infected.

Diagnostic confirmation

- Clinical appearance is typical. The extent of crack and/or depth may be determined with a hoof tester applied before and after work.
- Diagnosis of sole cracks is sometimes more demanding. Discoloured horn and the actual line of the crack may only be visible after careful paring of the entire sole surface.



Figure 17.8 Heel crack due to poor hoof conformation and lack of corrective farriery. The true extent may be visible only after careful dressing.

- Long-standing dorsopalmar or mediolateral imbalances which lead to heel cracks are usually easily appreciated.

Treatment

The first objective is to address the underlying cause. Brittle horn and weak horn have both been improved by prolonged feeding of gelatin at 30 g per day or by continuous feeding of biotin at 15–20 mg per day. Foot balance and regular farriery are very important at any stage in the development of a crack. It is important to ensure that the crack is not a secondary manifestation of a seedy toe area.

Uncomplicated superficial cracks These should be treated early by careful debridement and providing as much support as possible by using clips and by dressing the feet with 50% Neatsfoot (or horse foot) oil to maintain a proper hydrated periople. Avoidance of very dry harsh conditions will help – desiccation and trauma can lead to deterioration of a simple superficial crack.

Deeper cracks on the toe or quarter are treated by several methods depending on the availability of shoes, farrier and severity of the injury. All damaged horn should be removed, the crack sterilized with hypochlorite and supported with suitable supporting shoes with clips. In some cases support with a rigid hoof cast will provide enough stability for the crack to repair but it does take a long time and persistence is a major requirement.

Wall, quarter or heel cracks Treatment of cracks which involve sensitive laminae often requires regional anaesthesia.

The wall of the fissure should be gently debrided using an electric burr (Dremel) and very sharp, fine hoof knives. Tight wire sutures can be placed through the hoof wall to limit movement and the whole area can then be filled with synthetic resin hoof-filling compounds.

Alternatively, the debrided fissure can be treated with undiluted metacresolsulphonic acid and formaldehyde solution and dried before a synthetic filler is placed in the groove and allowed to set. The surface is then rasped to give a rough 'key' level with the wall. Self-tapping screws (4–5 mm or shorter) are set in the wall through several layers of fibreglass cloth and this is then covered completely with a synthetic filler or fibreglass car-body material. The fibreglass acts as a stable bridge between the self-tapping screws. It is important that any haemorrhage occurring in the fissure is cauterized or allowed to seal over for 24–48 hours before filling and sealing is attempted.

Heel cracks The damaged horn can be stripped off with shoeing pincers. The loosened wall is torn upwards, outwards and backwards. Tranquillizer and digital nerve blocks can be used but are often unnecessary; a twitch is usually the only restraint needed.

Antibiotic powder is applied to any area showing excessive haemorrhage and the hoof is bandaged. With larger cracks, or with horses in training, a bar shoe may be used to protect the remaining wall from further injury after removal of the quarter. Alternatively, the stripped area can be repaired with synthetic filler once it has dried out and is free of infection or necrotic tissue.

Transverse cracks The weakened wall should be removed and a shoe with side clips each side of the area of horn removal is prepared. An old piece of X-ray film is placed between the foot and lightly nailed shoe, to prevent the filler adhering to the shoe. The defect is then repaired using synthetic resin filler. Once the filler has hardened, the shoe can then be removed to allow resetting and final attachment.

Sole cracks All discoloured horn must be carefully pared out until pin-point haemorrhage appears. The region should be dressed with undiluted metacresolsulphonic acid and 5% formalin solution, or 5% phenol. The defect is covered with a steel or polypropylene treatment plate bolted onto the shoe. Modern strong plastic over-boots make repeated treatments simpler. Leather pads/soles can be used in stabled horses and, if a shoe is unavailable, leather can be glued onto the sole and wall of the foot. Leather is not suitable under wet, boggy conditions or if exercise is allowed on a hard surface.

More severely infected cases can be difficult to treat. The entire sole area may have to be stripped. Where this has occurred, the foot should be examined and dressed daily with antibiotic and metronidazole (or 4% povidone-iodine-soaked swabs) to control infection.

Coronary band injuries (wire and overreach wounds)

Profile

Wire cuts or overreaching injuries are very common and are probably the commonest traumatic injury to the hoof and coronary band (Fig. 17.9). Horses paw at fences, hook the heel over wire, and either pull back or saw the leg sideways, often causing extensive damage to the skin, lateral cartilage, tendons and coronary band (Fig. 17.10).

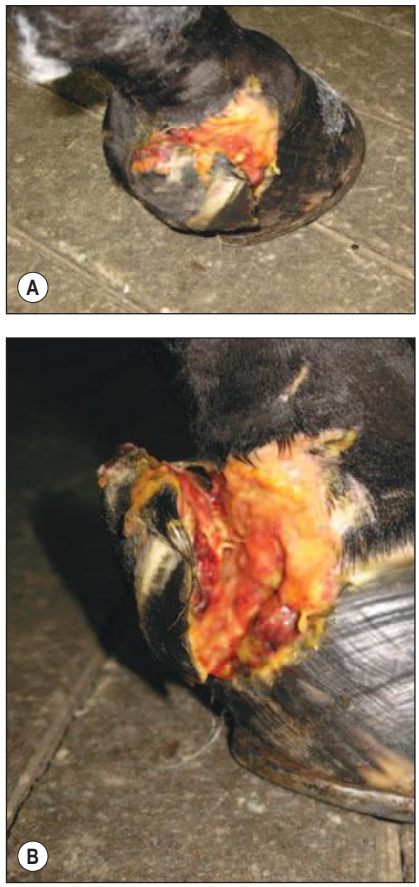


Figure 17.9 This severe wire wound is typical of those injuries that involve the heels and the coronet. In this case the large flap incorporated the coronary band **(A)**. In spite of the gross displacement and almost certain vascular compromise **(B)** it healed very well within 4 weeks following repair using delayed primary healing under a rigid half-limb limb cast. There was a persistent defect in the coronary band which created more problems for the farrier than the horse. The prognosis for such injuries is remarkably good except of course if they involve synovial or neurovascular structures or when the injury is poorly managed. Rapid attention is vital to the prognosis.

All injuries that physically damage or disrupt the continuity of the corium result in persistent hoof growth abnormalities (Fig. 17.11). In many cases the horse will manage provided that appropriate foot care is given. Complications involving synovial structures, tendons or neurovascular tissues carry a much worse prognosis and in all cases of acute trauma these aspects must be investigated fully.

Clinical signs

Injuries involving the coronary band or abnormality of the coronary band caused by previous injury are usually very obvious. Acute injuries *must* be assessed carefully to establish extent of damage to vascular and nervous supply to foot and damage to heel tissues and lateral cartilage.

Secondary defects in horn growth are very variable and depend largely on the extent of treatment applied at the acute stage. Cracks in the hoof wall commencing at the



Figure 17.10 Extensive wire injuries involving the hoof, coronary band and deeper tissues of the foot and the pastern and fetlock.



Figure 17.11 A permanent hoof defect as a result of a coronary band injury.

coronary band defect and extending distally are common outcomes.

Instability of the two sides of the resulting defect/crack may cause repeated lameness and even bleeding from the site. Secondary infection is common and can be very painful.

Diagnostic confirmation

- Visual inspection is simple but establishing the extent of the original injury may not be simple.
- Radiography and if possible MRI should be used to establish the extent of underlying damage to structures of the foot.

Treatment

Very careful assessment of the wound is essential: most are heavily contaminated and/or infected. Prompt, careful debridement using a scalpel (not scissors) of all dead or compromised or contaminated tissues is an essential first step. Hydrosurgical debridement is a useful technique for these injuries because it debrides very efficiently without removing any attached/viable tissue (Knottenbelt 2007).

It is very rare that wounds of this type can be sutured at the time of injury and most attempts to do so result in failure or worse. The choice should be made between allowing the wound to heal by delayed union or using a delayed primary union technique. The choice will depend on the facilities available and the type of injury but the value of rigid limb casting cannot be overstated.

Initially the debrided and cleaned wound should be irrigated carefully and packed with a hydrogel wound dressing material and a cavity absorbent dressing should be inserted into any defect to minimize dead space and to remove wound exudate. This dressing should be replaced at 48-hour intervals (or more frequently if exudate is excessive or patently infected) until granulation tissue is obvious and the wound bed is healthy. At this point (usually 2–5 days) the wound can often be closed surgically following scrupulous debridement.

Application of rigid limb casts over a suitable dressing is a rewarding procedure which has been made simpler and safer by synthetic polypropylene or polyurethane resin bandage casting materials. These are light, strong and easy to apply but the cast should always be applied (and removed) strictly according to the manufacturer's instructions. Cast management is vitally important. Unless complications develop, the cast can be left for 3–4 weeks. In the event of any complication (pain is often the earliest evidence of something going wrong) it must be removed immediately and if necessary replaced. Casts should be used until healing is well advanced. Early removal of support can lead to the wound splitting open over the original wound site.

Note

Casting often reduces healing time by up to 50% or more.

Suitable antibiotic and tetanus prophylaxis should always be given. With complicated wounds, appropriate other measures will be required.

Chronic secondary defects can be treated by several different methods:

- The horn can be grooved out to create a dovetail-type groove (as for the toe crack) extending from the coronary band to the solar surface or the most distal extent of the crack. The two sides can be wired or laced and reconstructed with acrylic filler using screws or lacing methods in which holes are drilled in the insensitive horn on either side of the defect. When this is done it is imperative that the defect is very clean. Repeated sterilization with hypochlorite solution is advisable.
- A groove can be cut down either side of the crack using a vibrating plaster saw or air drill circular saw at low speed. The whole defect is then stripped out

and haemorrhage is controlled by pressure bandages for 10 minutes. A bar shoe using wall clips either side of the defect is then applied to restrict spreading and movement of the healing site. When infection and necrotic tissue are absent the defect can be reconstructed using acrylic filler compounds.

The prognosis for hoof injuries is far better than it may seem at the outset. Even large tissue defects can still heal and the horse remain useful and pain-free. In all cases, however, an outstanding farrier should be used to ensure that the defect(s)/deficits are supported properly.

Puncture wounds of the sole

Profile

Puncture wounds of the sole are common injuries in horses. They result from penetration of the solar surface of the hoof by foreign bodies such as sharp stones, sticks, glass, nails, etc. Punctures in the middle third of the sole are likely to be the most serious. However, serious damage can be done both to the sole itself and to the other structures of the hoof from any solar injury.

Deep punctures (street nail) can be catastrophic when they involve synovial structures or when they cause osteitis of the navicular bone or the distal phalanx. These conditions are outside the scope of this book.

Clinical signs

Acute onset of a progressively severe lameness is characteristic. Toe-pointing, reluctance to bear weight on the leg and swelling of the back of the pastern and fetlock are present in some cases. Swelling as high as the knee may develop.

Where the penetration involves no 'vital structures' the initial lameness may subside for a day or two until infection develops and then a hot painful foot is presented. If the foot is examined carefully it is usually possible to identify the site of the penetration and hoof testers will confirm the focal nature of the pain.

Penetration of the synovial structures (navicular bursa, distal interphalangeal joint or deep digital flexor tendon sheath) gives extreme acute lameness with swelling of the flexor sheath behind the pastern and fetlock.

Diagnostic confirmation

- Thorough cleaning of the foot with a hoof knife to remove all loose or scaly sole or dirt should be carried out immediately. Hoof testers are then used to carefully test the entire white line, the sole and the seat of the corn, the entire frog and bars area and, if shod, each of the nails.
- If the testers locate a painful area in the foot, the shoe should be removed. Careful examination of the nails should be made to see whether any are blackened or discoloured or set higher than normal in the wall. Retesting with testers over the white line and nail holes is then carried out.
- Localized painful area(s) should be pared with a hoof knife until the solar horn is shown to be free of cracks or punctures. The hard, horny layer of the frog is carefully pared with a very sharp knife or scalpel and the sulci are likewise carefully investigated with a fine seeker knife.

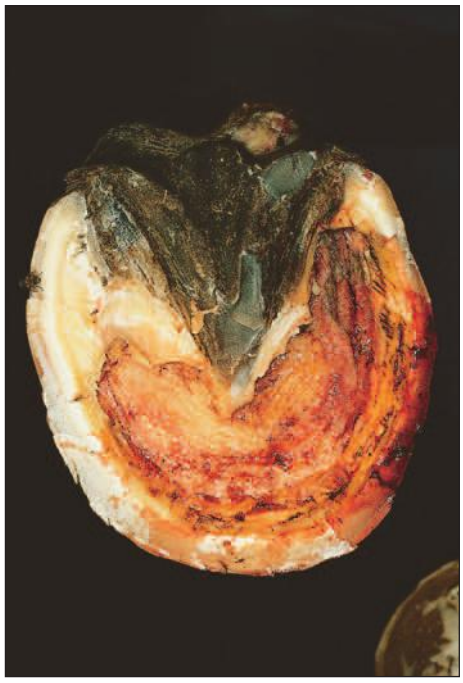


Figure 17.12 Extensive debridement of the sole following a puncture wound and sepsis with under-running.

- All black, cracked puncture marked or necrotic areas should be very carefully, lightly pared away until normal horn shows underneath or the actual damaged or infected area has been found (Fig. 17.12). The foot should be tested again for pain response to the hoof tester. Should this fail to locate an infection or injury site, careful deep palpation of the coronary band may reveal a small painful area corresponding to a very small 'pipe' of infection leading up a single lamina interface from an infection of the white line in the sole – so-called 'gravel' infection.
- If a lesion cannot be found with paring, nerve blocks should be used to ensure the lameness is in fact in the foot. A posterior digital nerve block will desensitize the posterior third of the foot and a low '4/6-point' block will desensitize the foot.
- Radiological examination may be necessary for differential diagnosis of fracture of the pedal bone, pedal osteitis, navicular disease and septic laminitis. Contrast medium can be introduced into the penetration tract to establish its true extent.
- Synoviocentesis of the various synovial structures of the foot can be used to establish their involvement. Lavage from these structures may identify that the injury has penetrated them (lavage fluid will be seen issuing from the tract during pressure irrigation of the relevant synovial structure).

Treatment

Nail pricks, and nail-binds of recent origin, are treated by cleaning out the nail hole and packing with povidone-iodine or 5% phenol solution. Antibiotics maintained daily for 4 days and analgesics (such as phenylbutazone) are also commonly beneficial. Some cases recover without further development of infection. Infected nail pricks, nail holes and puncture wounds of medium duration require that a

1 cm diameter hole with sloping sides be created around the wound to relieve pressure on the sole. This defect can be packed with an antiseptic-soaked swab or cotton wool and a shoe with treatment plate or a plastic boot can be applied to facilitate treatment and provide protection for the injury. The dressings should be replaced daily until a pad can be fastened under the shoe for more permanent protection.

Under-run frogs are treated in a similar manner. In severe case, the entire frog may have to be removed.

Deep punctures of the middle third of the sole in the frog region often penetrate the deep flexor tendon and/or navicular bursa. These injuries require specialist attention under general anaesthesia and should be referred accordingly as a matter of extreme urgency. They all carry a grave prognosis.

Haematoma of heels

Profile

Subcutaneous blood is present in the bulb of the heel(s). In the front feet, it is usually due to over-reaching by the hind foot; in the hind feet, it may be due to being galloped on, or kicking at walls or rails. Over-tired horses at the end of a race are more likely to receive this type of injury. The injury can also be due to badly balanced feet and occasionally it occurs after a rough journey in a transporter.

Clinical signs

Obvious acute onset of foot pain with pointing of the toe in the absence of other more obvious causes for this. Discoloration (bruising) of the heels may be obvious in white-heeled legs (Fig. 17.13) but may be very difficult to confirm in brown- and black-haired heels.

Diagnostic confirmation

- Clinical examination.
- Needle puncture over the heel area reveals dark blood.

Treatment

Blood can be drained from the haematoma with a 16G needle. A pressure bandage is applied to prevent refilling. Analgesics such as phenylbutazone for 3–4 days are advisable. Correction of foot balance and particularly the provision of adequate heel support is important in cases where the dorsopalmar balance is incorrect (overlong toes with broken-back hoof pastern angles and forward-located shoes).

Sole bruising and haematoma

Profile

This is most often due to badly seated or too small shoes, or use of the horse on stony ground without conditioning or protection of the sole with leather or polypropylene protective pads. The focal bruising of the medial seat of corn in the fore limbs is a particular recognized disorder (see p. 374). Other causes include fracture of the pedal bone, short or brittle broken back walls (which forces the horse to walk on the soles). Mild chronic laminitis with dropped soles will also contribute to solar bruising.



Figure 17.13 Heel haematoma with obvious discoloration over the heel bulb. In a pigmented foot it may be very difficult to see.

Clinical signs

The horse is usually lame and blood-streaked horn and, rarely, free blood under the sole of foot can be seen. Typical foot lameness occurs with pain on percussion or pressure with hoof testers over the affected sole.

Differential diagnosis

Laminitic bruising: evidence of laminitis will usually be found in more than one foot and the bruising is associated with the wall and the sole.

Diagnostic confirmation

- Visual appearance of the sole after paring away the outer layers of the sole. Pare away scaly layers of sole and blood-streaked horn will be found over areas of sole. In most cases it is confined to the toe.
- Widespread haemorrhage with a very lame horse could indicate pedal bone fracture. Radiography must be used at an early stage to eliminate this and other causes of bruising.

Treatment

Corrective farriery is vital using correctly fitted shoes and a protective pad of polypropylene under the shoe. Bar shoes with several opposing side clips to support the entire hoof wall are helpful. Specific causes of the bruising require specific treatment measures.

Wall break-back

Profile

Repeated shoeing, brittle walls and incorrect shoe removal are contributing factors. Excessive dryness or wetting of the hoof wall and rough surfaces with unshod feet are relatively common aetiological aspects. Poor management is usually the principal cause although some horses have congenitally brittle hoof



Figure 17.14 Wall break-back to expose the sensitive laminae.

keratin. The role of biotin and rnethionine in the aetiology of hoof wall defects is uncertain, but it does seem that deficiencies can cause obvious abnormalities of the periople (see Fig. 14.3) and histological evidence of keratin abnormalities.

Clinical signs

Portions of the hoof wall break out and expose the sensitive laminae (Fig. 17.14). The condition is not always painful, but reluctance to walk and pain on digital pressure over the damaged hoof can be encountered.

Diagnostic confirmation

- Clinical appearance is typical.
- Radiographs may confirm underlying bone demineralization.

Treatment

A synthetic resin-bonded bandage is a useful supportive material. A protective leather undersole/pad should be fixed under the shoe; the pad may have to be glued over the sole and up the walls before applying the shoe if nail retention is difficult. Glue-on plastic shoes can be very useful as they minimize the damage to the horn. Resolution may take up to 3–6 months in an otherwise normal foot and much longer if there is underlying pathology. Addition of methionine and biotin to the diet can help restore hoof quality.

Hoof wall rings/laminitis

Profile

These are most often due to dramatic feed and work changes, equine metabolic disease, laminitic episodes or other causes of foot inflammation or prolonged periods of systemic illness. They can also follow 'therapeutic' or accidental blistering with strong iodine or even liquid mercurial blisters which are applied in an attempt to promote horn growth. Growth from such treatment is often wavy and irregular. Excessive stress or severe exercise can induce rings in the horn in endurance horses.



Figure 17.15 Typical rings and abnormal hoof growth of chronic laminitis which arises from abnormal and irregular horn growth.

Key points: Laminitis

14 17 19 20 21 22 24

1. Laminitis is a common and serious foot disorder. It usually affects all four feet but can affect even a single foot – although the front feet are more often seriously affected.
2. A history of lameness and foot sensitivity is common but hoof wall 'rings' and milder sensitivity to hoof testers or to mild percussion is common.
3. Diagnosis is relatively simple. Radiographic changes are characteristic but not all cases show the changes, especially the forms associated with equine metabolic disease.
4. Treatment is difficult. Suitable foot care and farriery can be very helpful. Affected horses do not benefit from forced exercise.
5. The prognosis for resolution is poor and the effects are probably permanent – ongoing management and hoof difficulties are almost inevitable. However, some horses cope well for many years provided that acute episodes are avoided and farriery is maintained at a high level.

Laminitis produces the most marked changes and in the chronic form the hoof will have a series of rings plus a change in wall angle due to white line separation.

Chronic laminitis is characterized by lameness, 'laminitic' rings on the hoof, change in the hoof angle, and rotation of the third phalanx and presence of a laminar wedge – usually, bilateral lameness when due to metabolic changes, but a single weight-bearing foot can be affected where another foot or leg has been non-weight-bearing for a long period (Fig. 17.15). It also occurs in selenium poisoning (see p. 307) and pituitary pars intermedia dysfunction (see p. 322).

Clinical signs

Severe, often bilateral lameness with an exaggerated gait having an obvious, characteristic heel–toe foot placement pattern. Heavy 'ring-formation' of the hoof with excessive flaky horn growth is typical (Fig. 17.15). The hoof wall grows faster at the heels than the toe, which leads to dishing or slippering of the wall. Dramatic widening of the white line due to damage to the laminae is common.

A laminar wedge develops with a dropped sole where rotation of the third phalanx has occurred. Bruising of the

sole just behind the white line on the anterior portion of the sole is common.

In severe cases, suppuration of the dropped sole and prolapse of the pedal bone through the sole may occur.

Diagnostic confirmation

Characteristic disease is apparent which has distinctive clinical and radiographic findings.

Treatment

Very careful farriery is required to correct the underlying disorder and the foot/hoof problems. Correction is a slow, costly procedure and the owner should be fully apprised of the likely prognosis. No specific treatment is applicable to the laminitic rings, although careful oiling and reshaping can help. Care must be taken not to damage the outer protective layers of the hoof as this may cause drying and cracking. Where reshaping of the hoof wall is required, hoof dressing on exposed keratin is advisable.

Canker

Profile

This is a destructive (keratolytic) hypertrophic pododermatitis of the frog, hoof wall and adjacent structures seen predominantly in draught horses kept under unhygienic wet conditions. It has, however, been reported in all breeds and all ages of horse. Fortunately it is not common but is overrepresented in heavy horses and this may reflect a lesser foot care provision or some form of inherent defect of the horn.

The cause is not established but it remains a major cause of loss amongst affected horses. It is likely that infection and an underlying defect in horn production are involved. A wide range of bacteria (including spirochaetes, *Fusobacterium* spp. and *Pseudomonas* spp.) have been isolated and this suggests that there is an underlying defect of keratin and hoof tubule formation.

Key points: Canker

2 11 14 17 21 22 24

1 A sporadic, uncommon chronic disorder affecting heavy horses in particular. Possibly some genetic aspect. One limb only may be involved. Hind feet worse than fore in most cases.
2 Destructive decay and suppuration of the frog and horn of the heels at first but extension to involve the sole and walls occurs in neglected cases. Foul-smelling, crumbly horn with exudate is characteristic. Pain is usually mild only but secondary infections can increase this.
3 Diagnosis is relatively simple. The destructive nature of te condition, the odour and the fragile granulating tissue that bleeds easily are cardinal diagnostic features. Bacterial cultures are usually unhelpful.
4 Treatment is difficult. All necrotic tissue must be removed and the resulting haemorrhagic defect supported in a dry and uninfected state. Application of strong caustic chemicals is usually counterproductive.
5 The prognosis for resolution is poor – ongoing management and hoof difficulties are almost inevitable. However, some horses cope well for many years.

Clinical signs

Most cases begin as an area of necrotic foul-smelling horn at the heel or posterior frog area (Fig. 17.16). The abnormal hoof and frog can be pared away easily and the area bleeds readily. Often only one foot is affected and hind feet may be more often involved with the worst forms of the condition.

As the condition progresses abnormal finger-like papillae of hypertrophic horn, which also bleed easily, grow from frog and adjacent sole (Fig. 17.17). A creamy exudate is common especially when some inappropriate treatment has been applied by the owner – usually this involves caustics and tissue-destructive materials.

It can reach considerable proportions and can remain infected and exudative. Gross hoof defects can occur as the horn crumbles away.

Pain is usually mild unless infection becomes significant but some cases can be lame.



Figure 17.16 **(A)** Mild 'canker'. **(B)** This mature Shire horse developed a mildly painful, highly destructive necrotizing canker in the lateral heel of the right hind leg. Note the black necrotic hoof wall; the horn quality was so bad that hand rubbing removed large amounts of damaged horn and frog. The other legs were unaffected. **(C)** A form of 'canker' affecting the frog.

Differential diagnosis

- Hoof trauma: usually obvious episode of injury.
- Thrush: restricted to the clefts of the frog and central sulcus. Characteristic black appearance and odour.
- Foot sepsis including quittor: single focus with acute onset and identifiable hot-spot with pain.

Diagnostic confirmation

- Clinical appearance is characteristic.
- Characteristic histology showing chronic pododermatitis (histological examinations are not often performed).
- Bacterial cultures reveal mixed colonies of destructive bacteria and sometimes even anaerobic species.

Treatment

Treatment is usually prolonged – often up to several months or more. Owners must be advised at the outset of the poor prognosis unless the treatment is persistent and meticulous. Recurrences and setbacks are common.

Early application of a hospital/treatment plate is very helpful – treatment will require very aggressive avoidance of maceration/soaking and repeated applications of topical medicaments so a treatment plate is a good investment. Surgical removal of all affected tissue is essential. This can be done under regional local anaesthesia. Soaking and poulticing of the foot in a povidone-iodine soapy solution for 2–3 days before surgery is helpful. Debridement results in heavy bleeding initially but as the condition is controlled it does reduce. It is important *not* to necessarily remove all damaged tissue on the first occasion – often it is better to take time and remove as much as possible before bleeding becomes marked and then dress the foot carefully using metronidazole-soaked swabs. Repeated treatments at 2-day intervals will bring considerable benefits. Fucidin and metronidazole mixtures in paste or cream forms may also have a significant benefit.

Note

Application of strong chemicals and formalin solutions are counterproductive – they simply destroy more healthy tissue. *Debridement*, *dryness* and *cleanliness* are the main objectives.



Figure 17.17 Canker showing abnormal finger-like papillae of hypertrophic horn which bleed easily, growing from frog and adjacent sole.

Surgical debridement procedures should continue until healthy tissue is exposed over the whole area. Once the initial bleeding and exudation have been controlled, the area must be kept dry using antibiotic powders or spirit-based aerosols. Benzoyl peroxide (2%) has been successfully used as a dressing, but the major requirement is to keep horse's feet clean and dry with daily dressings.

Oral antibiotics can be used but are not generally very helpful especially after the first stages have been passed.

Treatment over many months is always required and repeated debridement needed to control chronic hypertrophic growth.

The prognosis is poor for severely affected horses

Thrush

Profile

Thrush is the name given to a degenerative, exudative, bacterial dermatitis of the central and collateral sulci of the frog characterized by a granular, thick or sticky black discharge and a characteristic foul smell.

Local factors such as poor foot conformation (deep cleft with narrow and occluding margins) and general factors of lack of foot care (e.g. stabled horses in wet, unhygienic conditions or persistently wet underfoot conditions) are significant factors in the aetiology. Sheared heels or overgrown shod feet also predispose to the condition. Although most cases are related to foot care/hygiene, some cases do occur in well-managed feet.

Infection with a variety of microorganisms including *Fusobacterium necrophorus* is commonly present.

Clinical signs

A foul-smelling, necrotic black discharge from the clefts and sulcus of the frog is characteristic (Fig. 17.18). Often several or all of the feet are affected though this may be to varying degrees. Sometimes it is only detectable when a hoof pick or fine hoof knife is used deep in the clefts.

There may be local or more extensive under-running of sulci of the frog and in severe cases sepsis of the deeper

Key points: Thrush

5 14 17 21 22 24

1. A sporadic, common, mildly destructive bacterial disease of the central sulcus and lateral frog clefts. Usually associated with poor conformation and poor foot hygiene and irregular/inadequate farriery.
2. Foul-smelling black discharge from the depths of the clefts and central sulcus of the frog. Mild lameness and possibly on occasion some deeper infection can arise.
3. Diagnosis is simple but care must be taken to explore the depths of the clefts in case the condition is less obvious.
4. Treatment is simply a matter of removal of all necrotic horn and opening the clefts and central sulcus to allow proper air circulation. Diluted formalin solution, alcohol sprays and other foot care products are useful. The treated feet must be kept very dry and very clean.
5. The prognosis is excellent but recurrences do occur. Horses with poor conformation require ongoing preventive farriery.



Figure 17.18 Thrush in the cleft of the frog – the clefts are filled with a foul-smelling black material.

structures of the foot. Swelling of leg and lameness occurs in some horses.

Differential diagnosis

- Hoof trauma: usually obvious and seldom only involves the clefts of the frog.
- Focal hoof sepsis: very painful and centred around a focus. This can develop directly from thrush also.
- Canker: highly destructive condition with a particularly foul smell and a yellowish discharge, primarily affecting heavy horses.

Diagnostic confirmation

Appearance and features are typical.

Treatment

All necrotic frog and under-run infected areas must be debrided down to healthy horn. Thereafter hygiene and regular careful daily treatment of the damaged area with 5% formalin solution worked into the cleft of the frog with a firm paintbrush is continued until all evidence of infection has cleared.

Metronidazole paste applied twice daily for up to 3 weeks is often effective. Weekly foot bathing with 10% magnesium sulphate ($MgSO_4$) solution can be prophylactic. The horse must be placed in clean, dry surroundings and contact with wet, unsanitary conditions must be prevented. Repeated debridement may be necessary.

Intravenous or oral potentiated sulphonamide compounds will assist in clearing the infection, particularly in cases with deep-seated infection but this is seldom more than an adjunctive treatment – it will not remove the need for careful debridement.

The prognosis is excellent provided that regular foot care is given and underfoot conditions are managed properly.

Lateral cartilage necrosis (quittor)

Profile

Quittor is the name given to a lameness associated with swelling and intermittent or more often persistent infection involving one of the lateral cartilages of the foot. It is characterized by a purulent discharge from a sinus tract over the area of the lateral cartilage. It is usually associated with trauma (e.g. wire cuts or overreach injuries) to the lateral or medial heel areas with subsequent infection, inflammation and necrosis of the lateral cartilage.

Key points: Lateral cartilage necrosis

11 14 17 20 24

1. A sporadic, now rare disorder involving necrosis of the lateral cartilages, arising most often as a result of traumatic injury followed by infection.
2. A chronic purulent discharging sinus above the lateral cartilage with a variety of mixed bacterial organisms. Sometimes the foot is mildly painful and sometimes there is wider pathology that complicates the condition still further.
3. Diagnosis is based on the clinical features and the absence of any other cause. Where available magnetic resonance imaging or computed tomography can be helpful
4. Treatment is difficult and always requires surgical intervention. All necrotic tissue (including any necrotic cartilage) has to be removed and once this is done healing may still take some time. Antibiotics are not effective on their own.
5. The prognosis for resolution is good but some cases are refractory.



Figure 17.19 Quittor (necrosis of the lateral cartilages) resulting in a chronic purulent discharge from above the coronary band.

Clinical signs

In the early stages a painful swelling over the area of the lateral cartilage develops. A persistent or intermittent, usually scanty, purulent discharge develops, typically above the coronary band (Fig. 17.19). Periods of apparent healing are followed by repeated, though seldom increasingly severe, discharge. Local pain is not often marked during periods of discharge. In some cases the extent of the discharge can be minimal and show as a slight matting of the hair over the site. Lameness may or may not be obvious.

Differential diagnosis

- Focal foot sepsis: usually hot, painful and rapidly progressive.
- White line disease/seedy toe: chronic recurrent condition associated with separation of the white

line and localized infection that can be evident at the coronary band.

Diagnostic confirmation

- Characteristic history of injury to the coronary band over the lateral cartilage.
- Typical appearance of a chronic discharging sinus tract just above the coronary band.
- Plain and contrast radiography (using a catheter and contrast medium or a sterile blunt probe placed in the tract) will assist in assessing the extent of the problem and differentiate it from other causes of discharging coronary band sinus tracts such as 'seedy toe', white line disease, laminitis and solar sepsis.
- MRI/CT imaging as well as ultrasonography can help to confirm the diagnosis but in the end the diagnosis is usually confirmed during exploratory surgery.

Treatment

Surgical removal of all damaged cartilage under general anaesthetic is essential. Antibiotics and careful dressing of the site with silver dressings will minimize the delay in healing.

Early lesions treated aggressively carry a good prognosis, but the outlook for extensive old lesions, especially when they involve the coronary band or coffin joint capsule, is much worse. Lameness often persists after surgery in the latter cases. Unilateral digital neurectomy can be considered as a palliative treatment if the surgical wound has healed satisfactorily.

Seedy toe/white line disease

Profile

This is a separation of the hoof wall from its laminar attachments that results in a crack developing within the white line at the level of the sole at first. This allows bacteria and or fungal infections to gain entry into the sensitive laminae. Necrosis of the sensitive laminae, usually of the anterior wall of the hoof, is the main feature but it may affect all distal areas of the laminae, extending proximally towards the coronary band.

Cracks in the white line leading to infection and chronic laminitis with separation of the laminae in the area of the white line (usually at the toe) will cause this condition. Anaerobic fungi have been incriminated in some cases.

Many cases are associated with poor hoof care or poor conditions that result in maceration and softening of the hoof. Excessively dry conditions with rough underfoot going can cause splits in the white line also. The name seedy toe is also applied to the widening of the white line that is a feature of laminitis. This is best regarded as a secondary state. No matter how it is caused the outcome is the same.

Clinical signs

Mild or sometimes moderate lameness is usual but not invariable. Often there are hoof wall rings suggestive of this and an obvious widening of the white line (usually in more than one foot).

Examination of the foot will identify a chalky, crumbly, white line region which can be easily gouged out with a foot knife. The adjacent hoof wall has a hollow behind it into which grit and other foreign matter is forced. A quantity of grit/soil or impacted faeces/bedding can often be removed. The affected area is usually easily appreciated radiographically (Fig. 17.20).

Occasionally overt sepsis is present with a discharging tract at the coronary band above the region affected. It is unusual to have pus present in the solar defect but there may be a necrotic smell.

Some affected horses have had recent or historical episodes of laminitis.

Key points: Seedy toe/white line disease

14 17 20 21 22 24

1. A common hoof and foot condition of laminitic or neglected horses. Excessive wet or dry conditions can be responsible and a particular secondary form occurs in chronic laminitic horses. Late presentation is the common state mainly because the lameness is mild in the early stages.
2. A painful focus can be identified associated with white line separation (usually at the toe) and radiographic detection of wall separation. Possible deep-seated infection may result in a discharging sinus above or at the coronary band proximal to the affected site.
3. Clinical diagnosis is relatively simple and can be aided by radiography, MRI or CT imaging.
4. Treatment involves removal of all necrotic horn and infection within the affected area of hoof and preventing further foreign matter and infection from gaining access while the hoof regrows. Farrier attention is the mainstay of treatment and stabilization of the hoof capsule is important.
5. The prognosis is usually good but depends on the intensity of clinical treatment and the future prophylactic measures to ensure good hoof care. Some cases have a weak hoof wall that constantly breaks away and allows secondary white line disease to develop.



Figure 17.20 Seedy toe. Separation at the white line and foreign bodies impacted into the cleft are typical (arrow).

Differential diagnosis

- Hoof sepsis: usually focal and acute onset of lameness associated with a hot, painful focus.
- Quittor: usually lateral rather than the toe and a chronic discharging sinus tract above the coronary band.

Diagnostic confirmation

- Characteristic clinical features are well recognized by owners, farriers and veterinarians.
- Lateral radiographic projections clearly show gas and foreign matter wedged into the area between the wall and the pedal bone dorsally.

Treatment

Small areas of seedy toe can be pared back to healthy tissue with a hoof knife. If the defect is spotlessly clean the defect can be packed with synthetic filler. If it is not clean, the cavity should be packed tightly with a swab soaked in povidone-iodine or metronidazole or hypochlorite and replaced daily until all evidence of infection is eliminated. More severe cases will need to have the wall over the 'seedy' portion completely removed and a small area of wall removed back to sound laminar tissue.

The defect should not be filled permanently until all risk of haemorrhage is past and the area is certainly clean and infection-free.

Extensive defects (some are very wide and as high as the coronet) should be extensively debrided *after* a shoe with supportive side clips has been applied to the foot to stabilize the distal margin. The use of an electric burr (Dremel) allows a distal bridge of the hoof wall to be retained while removing all the damaged horn proximally and along the solar margin. Once all necrotic and infected tissue has been removed, the defect can be filled with synthetic acrylic filler. The shoe is then reset and the horse rested in a stable with only light walking for 2 weeks. The shoe should be regularly reset every 4 weeks at least and a close check kept on the repair patch to ensure that it remains firm and uninfected.

Corns

Profile

Inflammation of the area between the wall and the bars of the foot known as the 'seat of corn'; usually the medial heels of the front feet are worse affected. This is caused through improper shoeing either because shoes are left on the foot too long or by use of poorly sized/undersized shoes. The heels of the shoe lie inside the wall and therefore cause excessive pressure on the seat of corn. However, it can also occur when the heels are allowed to drop by poor shoeing techniques causing tearing of the laminae and consequent bruising without direct shoe pressure. Pressure at this sensitive site causes bruising and necrosis of the horn, allowing infection to gain access to the bruised tissue.

Key points: Corns

1. Corns are the result of pressure or impact bruising (or both) at the seat of corn. Often poorly placed shoes (the heel is too narrow and presses on the seat of corn) or overgrown feet where the shoe is drawn forward so that the heels are displaced medially relative to the wall.
2. Affected horses are painful and positive to hoof tester pressure over the affected heel(s). Removal of the shoe and paring at the seat of corn will reveal bruising (at best) and often sepsis with a purulent discharge.
3. Diagnosis is simple; this is a well-recognized hoof problem.
4. Treatment involves debridement (if necessary to release of pus) and careful restorative farriery to ensure that the seat of the corn is not subjected to pressure possibly by unloading the heel. The site of the corn may need protection with the help of packing until normal foot health is restored.
5. Recurrences may suggest that farriery or management is not of an acceptable standard. The prognosis is, however, very good even when infection is involved.

Clinical signs

Acute lameness developing in a horse with poorly fitted shoes or overgrown feet is typical. The bulb of the heel may be warm and a painful focus may be detectable with hoof testers at the seat of corn.

A focal red or black bruised area or blood-streaked horn or even pus and under-running of the heel may be found when the shoe is removed and the foot is pared back (Fig. 17.21). A very small black focus may be all that can be seen on first glance.

Careful examination of the hoof wall at the heels may reveal a curled-under wall which causes pressure on the seat of corn and lamina problems with the urgent need for corrective farriery.

Differential diagnosis

- Solar penetration wound: usually faster and much more severe with rapid progressive lameness and pain.
- Laminitic bruising: usually more associated with the distal margin of the pedal bone so identified in the middle third of the sole and across the whole foot.
- Thrush: foul-smelling black discharge in the depths of the lateral or central clefts of the frog; mild or no lameness.

Diagnostic confirmation

- Clinical signs and examination of the seat of the corn.
- Palmar digital nerve block removes the lameness.

Treatment

All the damaged and bruised solar tissue must be removed and, if infected, the region should be opened and drained.



Figure 17.21 Corn (arrow). A foul-smelling purulent discharge (often but not always black in colour) can be released from many of these cases by careful paring and following of even the smallest black tract.

A shoe with either a leather or polypropylene pad (possibly with a treatment plate) is applied.

The defect should be packed with an antiseptic-soaked cotton wool plug. Antibiotics (such as penicillin) are often useful once the tract is draining freely. The tetanus status is very important for infections of this type.

Where horses have thin-walled feet, leading to the heel wall turning under, heart-bar shoes can be used to prevent further bruising. It is essential to make sure shoes are fitted properly to prevent shoe pressure on the white line or the seat of corn. The heel is often cut down to sound tissue and the horse shod with raised, graduated heels to balance the foot and regain correct alignment. Plastic heel wedges are often used to retain foot level and balance *but* these frequently apply even more pressure to heels and may actually aggravate the problem. Alternatively, the heels can be reconstructed with synthetic acrylic filler and fibreglass mesh to restore normal conformation.

Tumours of the foot

Sarcoids are probably the commonest tumour located at the coronet and the treatment of these is extremely difficult. Such cases should be referred to a specialist centre where radiation or other means can be attempted. Nevertheless, there is still a high failure rate with the more aggressive types of fibroblastic tumours at this site. The more benign verrucose or occult lesions are far less common and can remain static for years (often until any insult, either accidental or iatrogenic, is applied). They are probably best left alone unless full specialist facilities are available.

Squamous cell carcinoma and *melanoma* have also been reported at the coronary band. The prognosis for these is poor.

Two specific tumour types occur in and on the foot. These are the *sole fibroma* and the *keratoma* and are described in Chapter 18.

Reference

Knottenbelt D C 2007 Hydrosurgical debridement in management of contaminated wounds. Proceedings of the 21st NAVC Conference, Orlando, USA, pp 133–134

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18

Neoplastic conditions

Equine sarcoid	387	Cutaneous histiocytic lymphosarcoma/ lymphoma	418
Fibroma and fibrosarcoma	407	Mycosis fungoides (malignant cutaneous lymphosarcoma)	420
Neurofibroma (schwannoma, neurolemmoma, neurinoma, nerve sheath tumour/perineural fibroblastoma)	408	Melanoma and melanosarcoma	421
Basal cell carcinoma	409	Ossifying fibroma (equine juvenile ossifying fibroma)	426
Mastocytoma (equine cutaneous mastocytosis/mast cell tumour)	410	Squamous cell carcinoma	427
Haemangioma and haemangiosarcoma	412	Sebaceous gland tumour/carcinoma	433
Malignant fibrous histiocytoma (extraskeletal giant cell tumour)	414	Sweat gland tumour	434
Leiomyoma-sarcoma	415	Trichoepithelioma	434
Lipoma (liposarcoma)	415	Fibroma of frog and sole	435
Lymphangioma	416	Keratoma	436
Cutaneous lymphoma	417	Generalized (non-epitheliotropic/multicentric) lymphosarcoma	437

A neoplasm is an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissue and persists in the same excessive manner after cessation of the stimuli which evoked the change.

(Sir Rupert Willis 1952)

See also:

- Congenital skin disorders (congenital melanoma, p. 229; congenital vascular hamartoma/cavernous haemangioma, p. 237)
- Viral skin disorders (equine viral papilloma, p. 135)
- Endocrine disorders (pituitary pars intermedia dysfunction/pituitary adenoma, p. 322).

Literally the term 'neoplasia' means 'new growth'. The resulting mass of cells is termed a neoplasm and this term should be used for any non-inflammatory tissue swelling. The term tumour is often used synonymously although it should probably be reserved for swellings associated with the signs of inflammation (swelling, pain, heat and redness). The term cancer is probably derived from the Latin for 'crab' and is commonly applied to all malignant neoplasms. The term is therefore usually reserved for tissues with a purposeless malignant behaviour that parasitize the host body through their demanding nutritional competition with the normal tissues. Neoplastic tissues are often viewed as virtually autonomous because the neoplasm may flourish while the host loses weight and vitality but ultimately of course the tumour does rely totally on the host to supply nutrients and oxygen.

Neoplastic disease is a major cause of morbidity and mortality in horses and it is certainly true that the skin is by far the most common organ affected by neoplastic disease in horses worldwide (Jackson 1936, British Equine Veterinary Association 1965, Kerr & Alden 1974, Baker & Leyland 1975, Cotchin 1977). The prevalence of skin tumours is relatively high in the horse and most tumour types have particular features which make early diagnosis important. In contrast to most other species, in equine cutaneous oncology there is little correlation between the prevalence of skin tumours and advancing age.

By the time a solid tumour is clinically detectable it has already completed a major portion of its life cycle – cell replication is established with a 'clone' of abnormal cells whose clinical and pathological behaviour is determined by specific genetic changes that have taken place. The latent period before which a tumour becomes clinically detectable is highly unpredictable and will depend upon a number of characteristics such as the rate of replication versus cell death (the growth fraction), the inherent replication rate of the cells themselves (reflected crudely as the mitotic index or the proportion of cells undergoing mitosis at any one time) and the absolute number of tumour cells present. Some tumours are very slow growing while others are rapid. This means that equine tumours are usually only diagnosed when they are in a relatively advanced state. An added complication is the generally casual acceptance of skin tumours by both owners and veterinarians. Although there are some well-recognized basic principles in cutaneous oncology of horses, there is little that is predictable about cutaneous neoplasia.

The pathophysiology of neoplasia centres on cells that lose their normal growth controls, replicate more rapidly and in some cases migrate locally and/or to remote sites by either haematogenous spread, lymphatic dissemination or direct contiguous invasion of adjacent structures and tissues.

The underlying cause for this change in behaviour is invariably genetically driven resulting from spontaneous (innate) or induced (carcinogenic) mutations in their genetic structure, i.e. the acquisition or establishment of an oncogene (a gene that has been mutated in a way that supports neoplastic growth). The genetic alterations that culminate in cell transformation are those that control cell cycle check points, DNA repair and DNA damage recognition, apoptosis (programmed natural/planned cell death) and growth/replication signalling mechanisms. As a result of the changes the cells may lose their 'mortality' (planned cell death/apoptosis) although they may retain some or even all of their normal functions. The loss of normal apoptosis leads to tumour formation and the clonal expansion of the population of these cells culminates in the development of a visible tumour.

Not every cell that has a genetic mutation will result in neoplastic growth! Indeed spontaneous or carcinogen-induced changes may occur far more frequently than we suspect but many of the mutations will either be repaired or will result in immediate cell death, i.e. the cell is no longer viable. In either of these cases normal function will usually be maintained. The tumour will influence the host and in many cases the host will instigate natural defensive mechanisms that may limit or even eliminate the mutated cells. The two main neoplastic changes that occur within the genetic structure are:

1 over-activity of a proto-oncogene
2 loss of inherent tumour suppressor function.

The specific diagnosis of a tumour is very important, particularly with respect to the prognosis and possible treatment options. In some cases, such as the equine sarcoid, biopsy may be specifically contraindicated and a diagnosis may have to rely on other means. Some tumours are easily recognized clinically, e.g. melanoma, but others are much more difficult. Interference with a malignant tumour or incomplete surgical removal may result in a significant reduction in prognosis. Fortunately, the incidence of highly malignant tumours of equine skin is generally very low. Over the last 50 years there has been a dramatic increase in our understanding of neoplastic disease – unfortunately this has not been reflected in similar advances in equine oncology. However, it is certainly true that more effective therapy is being delivered and that there are advances being made to the benefit of horses.

One of the most important major characters of neoplastic masses is their clinical and pathological behaviour. Tumours are broadly divided into benign and malignant neoplasms. Benign tumours tend to grow slowly and remain localized. They are usually well differentiated (i.e. they closely resemble the normal cell type) and exert few remote clinical effects. Malignant tumours in contrast grow rapidly and tend to be invasive. They are usually less differentiated (i.e. the cells are more representative of the earliest developing cells of that specific type or have little or no resemblance to the normal cells) and may spread either locally by invasion of tissues (giving the tumour a bound-down character) or through haematogenous or lymphatic dissemination to other organs such as the lungs, spleen, kidney of bowel.

All neoplastic skin disease, whether benign or malignant, may be expected to have some implication for the horse in terms of either survival or well-being or cosmesis. Both benign and malignant tumours cause significant problems. Although naturally the malignant tumours might have a wider implication by virtue of their pathological behaviour, both benign and malignant tumours can cause both morbidity and mortality.

Tumours exert their effects in many ways. Functional tumours can produce active hormones while others have space-occupying effects. Others are debilitating by virtue of their 'parasitic' nature and others have widespread effects from abnormal, metabolically active secretions.

Both malignant and benign tumours have two basic components:

1 proliferating cells that are the basis of the tumour cell type
2 supportive structures including connective tissue and blood supply.

Where the stromal support is poorly developed, the tumour tends to take on a fleshy, soft nature while those tumours that have a firm, well-structured support tend to be hard/firm.

Benign tumours are usually designated by the suffix -oma to the cell of origin, e.g. fibroma, chondroma, lipoma, melanoma. Benign epithelial tumours with histologically or visually recognizable warty epithelial projections are termed papillomas. Benign tumours that project above the surface of the surrounding epithelium are called polyps.

The nomenclature of malignant tumours follows a well-defined pattern. Malignant tumours of epithelial cell origin (derived from any of the three germinal layers) are termed 'carcinoma' and prefixed by the cell type, e.g. squamous or basal cell carcinoma. Carcinomas with a microscopically identifiable glandular structure are termed adenocarcinoma, e.g. sebaceous adenocarcinoma. Malignant tumours derived from mesenchymal cells are termed '-sarcoma', a term derived from the Greek 'sar' meaning fleshy. The generally fleshy clinical appearance of these tumours arises because they have little connective tissue stromal support. Examples are the fibrosarcoma, melanosarcoma and haemangiosarcoma.

Although in most cases it is possible to differentiate between benign and malignant neoplasia morphologically, in some cases the tumour cannot be certainly categorized. It is important to realize that any particular tumour does not have to fall into one or other category; sometimes there are characteristics of both broad groups in the same mass. Also, the morphological appearance may belie the clinical behaviour of the tumour; histologically it may look benign/innocent but clinically it may behave aggressively. However, there are recognizable features that can be used in combination to try to define the tumour type more accurately (Table 18.1). These are:

Differentiation of the cells (anaplasia) Tumours are the result of clonal expansion and therefore the cells are usually uniform although there may be recognizable differences in their stage of maturation. The terms differentiation and anaplasia are used to describe the parenchymal cells of the tumour. Differentiation refers to the extent the cells resemble or differ from the normal cells of the tissue involved, both morphologically and functionally. The extent of cellular differentiation probably reflects the degree to which the mutation(s) alter the functions of the cell. Well-differentiated tumours are made up of cells that closely resemble the parent cell type in both morphology and function; this may make the tumour difficult to distinguish from simple hyperplasia or a

Table 18.1 The basic differentiating features of benign and malignant tumours

	Benign	Malignant
Extent of cell differentiation/anaplasia	Limited/absent Well-differentiated cells (recognizably similar to normal tissue type)	Marked Lack of differentiation with anaplasia; atypical cell structure/form
Local invasion	Limited Usually cohesive with well-demarcated margins that do not invade local normal tissues. This does not necessarily equate with encapsulation	Extensive Locally invasive with ill-defined margins. Some are more structured and expand locally
Rate of growth/expansion	Slow progression Periods of rapid/slow expansion or static, and some periods of apparent regression	Rapid (or variable/slow) Erratic growth patterns
Mitotic index	Low Few mitotic figures and these are normal in appearance	High Numerous mitotic figures and possibly abnormal/bizarre mitoses
Blood supply	Variable	Variable (usually high)
Metastasis	No	Yes (frequently present) The more undifferentiated the tumour the more likely are metastases

hamartoma – sometimes it is only the abnormal accumulation that is recognizable. Thus, a well-differentiated squamous cell carcinoma will have apparently normal keratinocytes that produce normal keratin although of course the increased cell numbers may mean that the volume of keratin is increased abnormally. This means that a simple morphological diagnosis of malignancy in a well-differentiated tumour can be very difficult. Sometimes it is reliant upon special staining and even the use of immunostaining.

Malignant neoplasms, by contrast, have a wide range of differentiation. Extensive mutation(s) that are not fatal to the cell would probably result in severe alteration of the cell function and therefore a clonal expansion of abnormal cells, often with a high rate of replication (mitotic index (relative number of mitotic figures in the tumour)/doubling time (time taken to double the size of the tumour)), will likely result in a poorly recognizable histological appearance. Malignant cells that are very poorly differentiated are termed anaplastic. The extent of differentiation may reflect the stage at which the cell is transformed into a neoplastic state. Transformation of an early (stem) cell will result in a very poorly differentiated tumour while a mutation of a more mature tissue cell may result in a more differentiated tumour. Typically undifferentiated (anaplastic) (malignant) cells manifest atypical degrees of pleomorphism – variation in cell size and shape – and have bizarre mitotic figures (often with multiple polar spindles). Abnormally large or small cells may be present. The nuclei of the cells contain abnormal amounts of DNA, often clumped in abnormal fashion that stain strongly (hyperchromasia). The nucleus may occupy an abnormally large proportion of the cell – often occupying more than 50% of the cytoplasm (normal nuclei occupy between 12% and 25%).

Anaplastic cells usually also vary in their structural arrangements; the tissue has a disorganized appearance with sheets or clumps of cells with a scanty vascular supply. Often, the rate of growth results in central necrosis – the tumour outgrows its own blood supply. Indeed the doubling time may be reduced if the central or larger parts of the tumour become necrotic. If all the cells survived the tumour would double its size in a predictable fashion depending on the rate of cell replication but most cells produced in a tumour do die as a consequence of pressure necrosis, ischaemia, anoxia, nutritive competition or abnormal mitosis. Also, many tumours replicate at a lower rate than the parent tissue so replication rate or even doubling time is not, on its own, always an accurate measure of the potential danger of the tumour. The absolute size of the tumour is also not a definitive indicator of its clinical significance (Fig. 18.1).

The two extremes of differentiation represent the breadth of cell differentiation that is possible in both benign and malignant tumours. It is certainly true that most tumours fall somewhere in between the two extremes.

A further complication is created by the 'precancerous' changes that are recognizable in some tumour types. Cell dysplasia is an important aspect in the pathology of skin tumours; the term is used to describe the disorderly but non-neoplastic cell replication in which there is a loss of cell uniformity and a disruption of cell architecture with an abnormally high replication rate. In the skin this is a relatively well-recognized pathological change in the horse – mitoses and a chaotic architectural arrangement are present in all the layers of the stratified squamous epithelium. When all the layers are affected this is usually regarded as a pre-invasive neoplasm or carcinoma in situ (intra-epithelial carcinoma). Although there is evidence to suggest that the changes do in fact represent a (pre)carcinomatous change, full development of a malignancy is still not inevitable. Where the changes do not involve all the layers of the epithelium, the changes may be reversible by removal of the carcinogen or removal/sloughing of the affected cells down to a normally replicating population. Topical corticosteroids, retinoids such as tazarotene and other pro-apoptotic compounds,



Figure 18.1 Size is not always the most important aspect of any dermatological tumour. **(A)** This is a large tumorous mass that looks very dangerous. In fact it was benign fibrous tissue choristoma and although removal was time-consuming, the result was excellent. **(B, C)** These two pictures were taken 2 weeks apart. Initially the horse presented with a small swelling below the eye and haemolacrimation **(B)**. Two weeks later the severity of the tumour had become apparent **(C)** and a biopsy showed it to be a carcinoma. By 2 months later the facial and maxillary bones were destroyed by the highly malignant, undifferentiated carcinoma with secondary tumours within the guttural pouch, necessitating euthanasia.

and simple removal of the carcinogen can still be totally curative. A good example is the precarcinomatous dysplasia that occurs on the penile and preputial skin of aged geldings (Fig. 18.2); if left, carcinoma is a regular development while if the area is cleaned of smegma and topical steroid is used, the earliest lesions are usually resolved completely.

Rate of cell replication (mitotic index) Undifferentiated malignant tumours often have an abnormally high number of dividing cells reflecting the high rate of replication. It is important, however, to realize that the presence of mitotic cells alone does not necessarily equate with certain malignancy nor indeed does it necessarily confirm that the tissue is neoplastic. Many normal tissues have a high cell turnover/replication rate and there are circumstances when this is very high, e.g. bone marrow activity in anaemia or healing epithelial cells in a skin wound. However, it is the anatomic character of the cells that is the critical issue.

Giant cells, with a single massive polymorphic nucleus or several nuclei within the same large cell, are also indicative of a basic neoplastic change. Again there may be some difficulty in differentiating this cell type from some reactive cells in the skin in particular (e.g. Langerhans cells or foreign body giant cells). Skilled pathologists can often (but not always) differentiate these.

Cell function As might be expected, well-differentiated tumour cells retain at least some of their function. For example, a well-differentiated squamous cell carcinoma will produce 'normal' keratin. This may of course be produced in abnormal locations and accumulate in the dermis and epidermis as visible or histologically visible keratin 'pearls'. Sebaceous adenoma may produce sebum that is indistinguishable from normal except that it may accumulate in an abnormal site in abnormal amount and then may induce an inflammatory response, implicating a malignancy. Undifferentiated neoplasms by contrast do not function normally – they may produce overtly abnormal proteins,



Figure 18.2 This picture illustrates the major differences between an overt carcinoma (white arrow) and patches of (precarcinomatous) dysplasia (leukoplakia) that occur relatively frequently on the penile skin of middle-aged to older geldings in particular (open arrows).

antigens and even endocrinologically active chemicals with remote effects (see paraneoplastic syndrome, p. 383).

In general it is accepted that the more rapidly growing and the more anaplastic tumours have less (normal) functional activity. The cells of benign tumours are usually well differentiated and so they resemble their normal counterparts, both morphologically and functionally. However, in all true neoplastic disorders there is a degree of alteration of function – sometimes more and sometimes less, sometimes obvious and sometimes subtle.

Rate of tumour growth Most benign tumours grow slowly (often over a period of years) whereas malignant tumours expand rapidly (often over periods of days or weeks) and the latter also may show periodic variation in growth (slow and fast at variable intervals). However, some benign tumours do expand rapidly and some malignant ones can be affected by other metabolic events such as variations in endocrine hormones and adequacy of blood supply (see below) and may then vary in growth rate. Some tumours also require endocrine support and others are simply enhanced through normal endocrine mechanisms. The use of anabolic steroids to 'treat' weight loss in cancer cases can cause dramatic exacerbation of all growth.

The blood supply Tumour growth is very dependent on blood supply. The blood supply to any tumour is a balance between the needs of the tumour tissue and the angiogenic capacity of the vasculature supporting it. In the absence of a dedicated blood supply, tumours may only expand to around 1–3 mm in size (Folkman 1993); once a blood supply is established, both benign and malignant tumours have a considerable ability to expand.

Angiogenesis is supported and enhanced by tumour-derived angiogenic (growth) factors expressed by proto-oncogenes, and by relative hypoxia in the expanding mass. For the most part, rapidly advancing tumours (high cell replication rates and short doubling times) will have a very high metabolic demand but this may exceed the ability of the blood vessels to invade the depths of the tumour. The blood supply to the developing tumour is initially a limitation to its growth and in many cases the central core of a rapidly expanding tumour may become necrotic – the cells outgrow their blood supply and this explains why in many cases aspiration from the centre of a tumour fails to reveal diagnostically valuable cells (see fine needle aspiration, p. 72). The total reliance of tumours on blood supply also provides a major opportunity for controlling tumour growth but adds to the difficulty of surgical management in some circumstances.

The extent of local tissue invasion Benign tumours have a cohesive, well-defined character in general. The edges can be recognized clinically in most cases but in any case histologically the edge is well demarcated. Most malignant tumours have an ill-defined margin giving the mass a 'bound-down' or diffuse character. This is not necessarily an invariable indicator of malignancy, although it is an indicator of *possible* malignancy. For example, some forms of the nodular sarcoid are bound down either to the overlying skin (type B nodules) or to the tissues deep and lateral to the tumour mass (type A2 and type B2 sarcoids) (see p. 388). The invasion of a tumour into the surrounding and deeper tissues is thought to be a result of the alteration of cell adhesion molecules (cadherins). One of the most feared tumour properties is the ability to metastasize to remote sites. This occurs only rarely in skin tumours in horses, but where it does, it is an indicator of malignancy. Haematogenous, lymphatic and direct extensions to adjacent organs occur.

The ability to metastasize to remote sites Although some tumours produce mediators that are directly angiogenic, the mild hypoxia that is induced by a slower-growing tumour can be enough to encourage angiogenesis into the mass. Therefore some tumours acquire large blood supplies (Fig. 18.3) and as a result become progressively more metabolically demanding; this does not necessarily equate directly with malignancy but rapidly growing tumours have a higher chance of being malignant.



Figure 18.3 This superficial sarcoid lesion developed rapidly over a few weeks from a small single focus that had developed at the site of a small wound. Note the prominent venous drainage from the tumour, suggesting that it had a significant arterial blood supply. The growth of a tumour is heavily dependent upon the blood supply it develops.

Assessing tumour activity and pathological behaviour (tumour grading/staging)

The classification of the severity and clinical nature of a neoplasm can be made through the process of grading/staging. This expresses the level of differentiation (grade) and the extent of spread of the tumour (stage). However, attempts to establish this system in horses have commonly been frustrated by the lack of correlation between the clinical and histological features. Grading is therefore not yet widely employed and instead a morphological description is commonly used. It is inevitable, however, that equine tumour medicine will need this kind of classification.

Grading Grading attempts to quantify the degree of cell differentiation and the number of mitoses within the tumour (expressed as a proportion of the cells present) and so correlates quite well with the extent of tumour aggression. Grades of I–IV are used where I is the mildest form of tumour (recognizable differentiation of the cell type involved and low numbers of mitotic cells) and IV is the most undifferentiated and rapidly dividing tumour behaviour. Criteria for the various grades vary for each tumour type, but the general principle is to attempt to quantify the extent to which the tumour cells differ from cells of the appropriate normal tissue. Although the grade is a useful pathological parameter, it is not necessarily an accurate index of the pathological behaviour because the histological appearance and clinical behaviour may not be closely correlated. In an attempt to avoid spurious 'opinion', tumours are also graded according to the histological description of the degree of differentiation from normal using terms such as 'well/moderately differentiated', 'highly undifferentiated', etc. Grading is probably less useful than staging.

Staging This is, potentially at least, a more useful measure of the tumour's nature than the grading system. The

size of the primary lesion, the extent of spread into the adjacent tissues and local lymph node(s) and the presence of remote metastatic dissemination are incorporated into the system.

Although there are two recognized systems in human medicine, neither one is ideal for the equine species. The preferred UICC (Union Internationale Contre le Cancer) staging system is called the TNM system. It uses the tumour size (T), the involvement of regional lymph nodes (N) and the extent of metastases (M) to numerically express some concept of the severity of the tumour. Thus, a large aggressive tumour might be described as T4/L3/M2 while a benign local tumour might be shown as T1/N0/M0. The system is more flexible than the other classifications and allows a clearer description to be made.

This system is not used widely at present in horses, possibly because the overall prevalence of tumours is low (or at least because they are poorly described in the literature) and because few are fully understood and completely characterized. The system does, however, enable better comparisons between treatments and prognosis and so there is merit in its development in equine oncology.

In-vitro cell culture assessment There are two main methods that are being developed in veterinary oncology from similar techniques used in human medicine.

1 Tissue collected by biopsy from a tumour can often be cultured and the cells examined closely for morphological variations and cell replication rate variations. Replication rates are quantified by measuring the rate of uptake of tritiated thymidine or 5′-bromodeoxyuridine. Of course the tests rely heavily on the availability of suitable cultural techniques for the individual cell types. Currently fibroblasts, squamous cells (keratinocytes) and vascular endothelial cells can be cultured easily but melanocytes and others are more difficult at present.
2 Monoclonal antibody technology enables similar information to be gleaned from standard formalin-fixed biopsy specimens. In these procedures antibodies directed at proliferating intracellular proteins are used to identify them. Simplistically the amount of antibody uptake is a direct indicator of the rate of replication. The tests are rapid and have been used for equine squamous cell carcinoma assessment in particular (Theon et al 1994).

Whether these assessments influence the clinical approach and therefore improve the prognosis is not yet certain but any extra information that can be derived that improves our understanding of oncological disease has to be a step forward.

Non-paraneoplastic consequences of cutaneous tumour development

The direct consequences of skin neoplasms can be significant and functionally limiting and debilitating effects can result in major complications with management. Even benign tumours can have profound effects on the animal. The effects can include:

1 Secretion of normal endocrine and paracrine metabolites which occurs when the neoplastic cells of a glandular or secretory structure are fully functional, i.e. well differentiated. This is not a major aspect of dermatological tumours.
2 Functional disability when the tumour interferes with the normal function of any adjacent organ. Usually this is a result of space-occupying properties. For example, a large perineal melanoma can obstruct the rectum and result in tenesmus, obstructive constipation (obstipation) and colic as a result of non-strangulating small colon obstruction (Fig. 18.4). Although penile carcinoma is singularly rare in (working) stallions (as opposed to geldings), when it does occur it can be very aggressive with devastating effects on the animal's breeding capacity (Fig. 18.5). The location of the tumour and its nature will clearly influence the effects it has on the



Figure 18.4 A large perineal melanoma, which had extended into the pelvis resulting in an extramural small colon obstruction. This is turn resulted in distress and finally in colic signs relating to a complete obstruction. Options for treatment in such cases are very limited and in this case colostomy was considered but ultrasonographic examination revealed many intra-abdominal and thoracic tumours and so the pony was destroyed.



Figure 18.5 This 17-year-old working stallion developed a highly malignant, aggressive, proliferative squamous cell carcinoma. This is a very rare condition in entire stallions but precluded his use for stud purposes and he was destroyed. Post-mortem examination showed inguinal and iliac lymph node involvement.

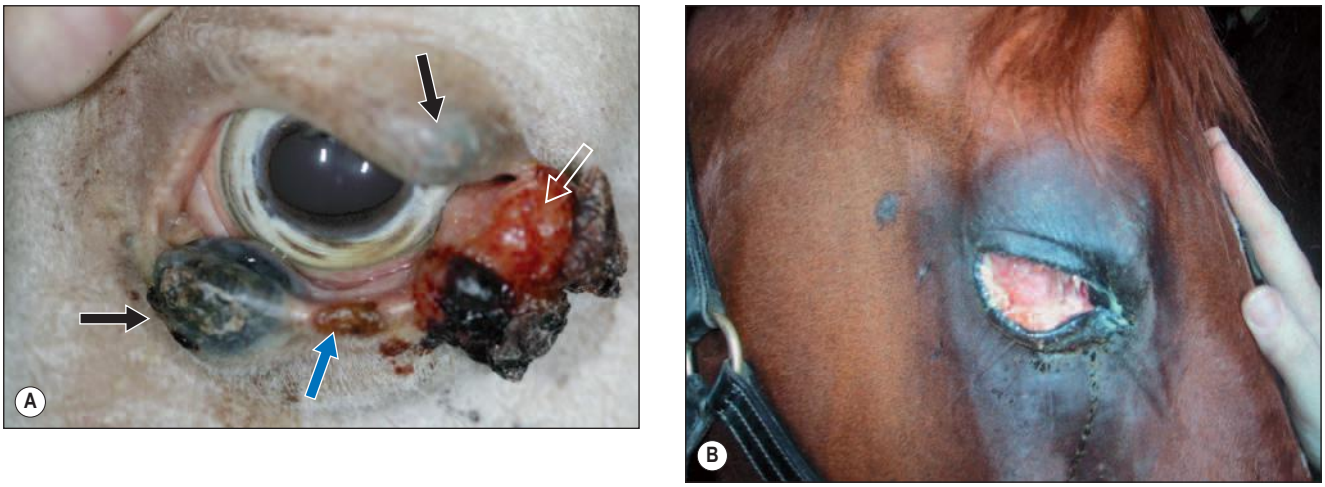


Figure 18.6 **(A)** This 14-year-old cream pony was presented rather later than it should have been with multiple tumours on its eyelids. The melanomas were obvious clinically (black arrows) and the ulcerative squamous cell carcinoma on the lower lid (blue arrow) was confirmed by biopsy. The larger proliferative mass was thought to be a component of one or other of these but was in fact a sarcoid (open arrow). There is no reason why animals cannot have more than one condition at the same time! In spite of the severity and complexity the pony was not seriously disabled by them. **(B)** This highly destructive carcinoma resulted in significant disability and secondary eye complications. Metastatic tumours were present in the parotid lymph nodes and the floor of the guttural pouch.

adjacent structures. For example, a melanoma in the jugular groove can seriously affect the vago-sympathetic trunk and produce Horner's syndrome and laryngeal paralysis. Even head oedema and swallowing difficulties can arise. Tumours that involve the eyelid may have serious effects on the eye itself (Fig. 18.6). This is the reason why a very careful and exhaustive clinical assessment is essential in all cases where neoplastic disease is suspected. The presenting sign may be dramatic but secondary to a less obvious tumour state. Functional loss can present in a misleading way. For example, the main (most obvious) external sign in a horse with multicentric lymphosarcoma may be diarrhoea. A careful clinical examination might identify significant cutaneous nodules. In the same way a single cutaneous haemangioma/haemangiosarcoma lesion may be missed in a horse with unilateral hind limb lameness, which could be due to disseminated tumours. It is always worth establishing the whole range of presenting signs before making a tentative diagnosis.

3 Metabolic deficiencies such as anaemia and hypoproteinaemia as a result of continued plasma and blood loss. Ulcerated and/or exposed tumours that can easily be traumatized can cause major blood and protein losses (Fig. 18.7).

4 Secondary infection/infestation: open ulcerated tumours are very liable to infection and myiasis in particular. Whilst the blood supply can be considerable it does appear that local immunity can be severely compromised by the tumour's existence.

The paraneoplastic syndrome

This is a syndrome that has a complex and poorly understood pathogenesis characterized by clinical and metabolic events that cannot be directly explained by the presence of the tumour (either locally or by metastatic spread) or by the formation of naturally occurring bioactive mediators and hormones in the cell type concerned. Although in horses these syndromes are



Figure 18.7 This large type 2 (sessile) fibroblastic sarcoid on this donkey's chin bled and exuded serum incessantly and resulted in clinically significant hypoproteinaemia and anaemia. Metabolic signs caused by tumours can be primary or secondary as in this case.

less common and in skin tumours they are even rarer, they are important. Quite often it seems that they are overlooked in the process of diagnosis of tumours in general but they may be the very first evidence of a serious neoplastic disease. Also the syndromes themselves may be fatal and then the death may be attributed to causes other than cancer. For the most part the syndromes are very complex and difficult to treat without removing the primary cause, i.e. the neoplastic disease.

There is also a clinical risk that animals showing signs consistent with the paraneoplastic syndrome (weight loss, anaemia, pica, etc.) may have other non-neoplastic reasons for these signs.

Cancer should be regarded as a parasitic state but in the case of the paraneoplastic syndrome it is even more than this. It is generally considered to be a complex multi-endocrinological effect from the secretion of false endocrinologically active metabolites of the tumour. Some tumours have a much higher propensity for inducing these changes. From a clinical perspective it appears as generalized metabolic



Figure 18.8 (A, B) This 5-year-old Shetland pony stallion developed this large exophytic mass on its tail with extensive hair loss and exudation. Initial reports from biopsies suggested that it was a neoplastic state but a review of the histology, and several new biopsies, confirmed that it was a reactive state to free keratin in the dermis, probably as a result of rupture of follicular cysts. Advice on treatment was limited because no previous reports of a similar state could be found.

effects resulting in disproportionate weight loss, inappetence, recurrent fever and sometimes anaemia. There are few primary skin tumour types that have well-recognized paraneoplastic consequences but, for example, the common equine sarcoid can be associated with debilitating effects and with a disproportionate metabolic effect reducing performance and possibly resulting in weight loss. Furthermore there are dermatological consequences of other tumour types such as haemangiosarcoma and lymphosarcoma that are included in the paraneoplastic syndromes. Various forms of immune-mediated vasculitis including paraneoplastic pemphigus (see p. 267) and paraneoplastic pruritus are recognized (Williams et al 1995, Anhalt 1997). Often the clinical effects are most obvious in the oral mucosa but changes can occur in the skin. Histologically subepidermal clefting, and vesicle formation with accumulation of lymphocytes, plasma cells and neutrophils were reported. These changes are dependent on the tumour and so removal of a causative mass can result in resolution of the dermal problem.

The approach to a suspected skin tumour

Clinicians tend to underestimate the contribution they make in the diagnosis of neoplastic disease, often preferring to rely almost totally on the pathologist. However, if the clinician provides a really careful dossier of information for the pathologists the likelihood of an early diagnosis is far greater. The role of the pathologist is critical, if only to make a clear differentiation between abnormal accumulations of normal cells and the various stages of tumour development. Clinically, early tumours can often easily be mistaken for hyperplastic tissue responses and vice versa (Fig. 18.8). Careful review of both the clinical and pathological findings may change the diagnosis.

There is nothing special about the diagnostic approach to a cutaneous mass! The normal clinical investigative procedure should always be carried out – sometimes an aggressive looking lesion turns out to be simple and benign and possibly even not neoplastic, while another may look innocuous and actually be highly sinister. An instant diagnosis may sometimes be available through the recognition of definitive clinical features (such as melanoma) but an intuitive 'guess' can lead to the wrong interpretation even if the tumour is 'recognizable'. For example, does a single, localized, darkly coloured mass on a grey horse have to be a melanoma? Where the mass is not sufficiently recognizable as to allow a definitive diagnosis to be made immediately, biopsy may be suggested. Biopsy is usually definitive provided that a suitably diagnostic specimen is obtained, because most tumour types are now recognized pathologically. The various techniques for biopsy/tissue sampling are described in Chapter 3 and typically some methods are more or less applicable to particular tumour types. However, the potential dangers of interference with any skin tumour must be considered carefully before performing any biopsy procedure. The least traumatic methods are fine needle aspiration and impression smears of ulcerated tumours. In both cases the ease and safety are somewhat offset by their variable clinical 'return'. In many cases a reasoned judgement has to be made on the likely nature of the mass. Frozen sections examined immediately after removal may provide a rapid diagnosis, but this is often not feasible. In the case of very small tumours, a total excisional biopsy (surgical removal) may be undertaken with some degree of safety. Where biopsy is considered unavoidable, suitable therapeutic options should be available for treatment as soon as the results are known. There is no point in taking a biopsy if its results will not influence the clinical approach.

Pathologists are usually highly skilled in recognizing the features of tumours. Laboratory confirmation should always be undertaken if there is doubt and then a positive diagnosis can usually be achieved. Where surgical removal is performed, all tissue removed should be submitted to the pathologist; a pathologist will be better able to decide how and where to cut the tissue blocks to obtain the best specimens. Special stains and immunohistochemistry can give improved diagnostic information. It is sometimes very helpful to supply a photograph of the lesion and the site of the biopsy and it may be even better to consult with the pathologist before taking the samples.

Aspects of malignancy are clearly important (even if only retrospectively). Skilled pathologists are usually able to establish whether there is an adequate margin or whether excision

has not been complete. A report of a 'safe margin of excision' is reassuring for the surgeon. Many tumours have local variations within the mass of tissue and examination of a relatively small piece may be misleading. For example, benign granulation tissue can be interspersed amongst a fibroblastic sarcoid; there may be an area of occult sarcoid around a smaller nodular or fibroblastic sarcoid; and there may be precancerous changes surrounding a squamous cell carcinoma lesion. A limited biopsy may provide the wrong information but extensive biopsy may be undesirable. The clinical examination and the experience of the clinician are therefore important.

Haematological responses including white cell and protein responses are generally not helpful in cutaneous neoplasia of any type. Cancer cachexia, characterized by weight loss, depression, anorexia, metabolic disturbances and general malaise which resolve after tumour treatment, is only rarely encountered in primary cutaneous neoplasia. However, where it does exist, it suggests that neoplastic lesions have significant secondary effects and sometimes these can be identified from blood. It is also important to remember that there are some cutaneous manifestations of internal tumours such as cutaneous metastases, immune-mediated vasculitis, and paraneoplastic pemphigus and in the case of pituitary pars intermedia dysfunction (PPID/Cushing's disease/pituitary adenoma) even hirsutism, polydipsia-polyuria and laminitis.

Note

Pituitary adenoma (adenoma of the pars intermedia) is an internal neoplastic tumour with skin manifestations recognized as pituitary pars intermedia dysfunction (Cushing's disease). However, there remains considerable controversy as to whether this is a true neoplasm or a secondary hyperplasia of the pars intermedia following loss of dopaminergic inhibition. Pathological enlargement of the pituitary gland (and the pars intermedia in particular) is a frequent finding during necropsy examinations of old horses. The pathological features suggest that this is a hyperplastic condition rather than a true neoplasm; the mitotic index is low, cells are well differentiated and often fully functional. The cutaneous manifestations (hirsutism, sweating, abnormal shedding patterns, ectoparasitism and cutaneous abscessation) are not in themselves strongly suggestive of a tumour origin but are typical and easily recognized in most cases, especially when considered with other characteristic signs such as polydipsia/polyuria, supraorbital bulging, weight loss, laminitis and neurological signs or central blindness (amaurosis).

Table 18.2 lists common and unusual skin tumours.

Table 18.2 Common and rare skin tumours

Common skin tumours
Dermal melanoma/melanosarcoma
Fibroma/fibrosarcoma
Lymphosarcoma/lymphoma
Mast cell tumour (mastocytoma/equine cutaneous mastocytosis)
Neurofibroma (schwannoma)
Ossifying fibroma
Sarcoid
Squamous cell carcinoma
Rare/unusual skin tumours
Basal cell carcinoma
Epitheliotropic lymphoma(mycosis fungoides)
Giant cell tumour
Haemangioma/haemangiosarcoma
Histiocytoma (malignant/fibrous)
Keratoma/keratoacanthoma
Sebaceous gland tumour
Sweat gland tumour
Trichoepithelioma
Lipoma/liposarcoma
Lymphangioma

Diagnostic procedures for neoplasia

The majority of skin tumours fall into the nodular diseases category. There are some important features of diagnosis and treatment which should be appreciated before any interference is undertaken. These include aspects of malignancy where interference could result in tumour dissemination, and the tendency to recurrence at the site.

Important diagnostic features include the following:

- elimination of other (non-neoplastic) (nodular) disease(s) such as allergy, infection, trauma and haematoma, preferably by clinical examination
- anatomical location(s) of the tumour(s)
- number and similarity of tumours (multiple or single masses)
- status of the draining local and regional lymph nodes
- status of the surrounding tissue and any secondary effects (e.g. oedema, heat, pain, swelling)
- presence of any systemic effects suggestive of paraneoplastic syndromes, weight loss, diarrhoea, physical deformities/abnormalities, and haematological changes such as hypoglycaemia or hypercalcaemia.

Physical examination

In all cases of cutaneous neoplasia a physical examination must be undertaken. This must include:

1. A detailed, systematic clinical examination (which will often include a rectal examination, endoscopic examination of the airways, radiology and ultrasonographic investigations).
2. Specific close examination of (all) the lesion(s) to establish individual extent and character.
3. Examination of as much of the surrounding tissue as possible and the appropriate draining lymph node(s).

All three of these have a strong influence on the diagnostic process as well as on treatment and prognosis. For example, a horse with a single, slowly expanding subcutaneous mass in the medial thigh might have gross enlargement

of the ipsilateral subiliac lymph node – this might support a preliminary diagnosis of lymphoma but in any case would alter the outlook considerably. Whilst an exhaustive clinical examination may seem in many cases to be an unnecessary complication, there is little worse than making a totally wrong diagnosis that seriously affects both the patient and its owner. Time spent in diagnostic processes is never wasted and there are few genuine dermatological or oncological emergencies!

The body condition of the horse and the health (or normality) of its system/organ functions may be seriously affected by some neoplastic disorders but much less so by others. Local invasion of tumour tissue may result in local distortions, physical alterations or functional problems, e.g. palpebral tumours may cause eyelid problems with secondary corneal ulceration or nasolacrimal obstructions with epiphora. Diffuse, ill-defined tumours may suggest malignancy or at least the prospect of treatment limitations. Few lymph nodes are readily palpable in the horse and further procedures such as endoscopy, radiography, ultrasonography and rectal examination may be required.

The laboratory-based diagnosis of neoplastic conditions has become much more complex as more is understood about the various cell behaviour patterns. Normally diagnostic tests include:

1. histological examination of aspirates/cytology
2. partial or total excisional biopsy material
3. direct (impression smears)
4. haematological examinations (some tumour conditions do cause detectable, if largely non-specific, changes in plasma proteins, red and white cell counts and electrolytes and enzyme concentrations).

Cytological examination (see Chapter 3)

The histological diagnosis of most advanced equine tumours is not problematic. Where a tumour has a very benign, differentiated form and where the changes are so severe that cellular structure cannot easily be recognized, there are few difficulties. The intermediate stages are far more of a problem and specific stains or immunocytochemistry, molecular methods, flow cytometry and the detection of specific tumour markers will often help. Possibly the biggest help is the clinical description and the correct and appropriate sampling of tissues.

The cell abnormalities encountered in aspirates are often subtle and difficult to interpret alongside artefactual alterations from sampling techniques. It is easy to overinterpret artefactual changes and indeed to overlook subtle but important diagnostic features. Incorrect sampling can easily occur and lead at best to a difficult interpretation and at worst to an error of diagnosis that could have been avoided (see p. 71). All smears/aspirates obtained from tumours *must* therefore be examined by an experienced cytopathologist. It is probably unacceptable to make a diagnosis without the help of such a specialist. Even recognizing 'black' cells in a suspect melanoma is not enough to establish useful information on pathological behaviour.

Malignant cells may be reported to exhibit some or all of the following:

- irregular/bizarre cell size/shape with uneven/unusual staining
- large obvious nucleoli within prominent/obvious nucleus with uneven staining and thickened nucleolar membranes
- high mitotic index (high proportion of cells in mitotic state).

Biopsy and histopathological examination (see Chapter 3)

There are several available sampling methods for use in equine oncology.

Biopsy of suspicious lesions is for the most part a sensible procedure. However, if the results of the procedure are not going to influence the management/treatment then there is little point in taking the biopsy at all. In some cases biopsy does not provide any more useful information – the common equine 'melanoma' probably does not warrant a biopsy because the clinical appearance is so 'typical'. However, regular submissions even from such cases might add eventually to our understanding of the condition. Also, some tumours may be exacerbated by the process of biopsy – the equine sarcoid, for example, is probably best not subjected to biopsy unless an immediate therapeutic regimen can be applied because of the risk of exacerbation (Brostrom 1995). Therefore, presumptive diagnoses are often made and acted upon. It is only when there are complications or uncertainties that further testing is really required,

Biopsy of skin lesions is usually feasible under sedation and local analgesia alone. The interpretation of a biopsy relies heavily upon:

- correct collection of a representative (diagnostic) specimen
- correct processing (including fixation, section and staining)
- careful examination of the tissues by a qualified pathologist.

Histopathology is probably compulsory for all excised tissues. There is little or no excuse for not submitting such specimens. These specimens will provide important information including the true (undistorted) characteristics of the tissue and the presence or absence of a clear line of demarcation to normal tissue. Unless every single tumour cell is removed, regrowth is likely and so, if only as an assessment of prognosis, surgical pathology is vital. If excision was not complete the owner can be warned that recurrence is likely; it is not then a disappointing surprise when the tumour recurs. Most owners will accept this more readily if communication is maintained.

Haematology and serum biochemistry

These are almost invariably disappointing in skin neoplasia but may be significantly altered if:

1. There is extensive organ involvement (either primary or secondary).
2. There is significant secondary infection (either locally or systemically) as a result of impaired immune processes.
3. There are paraneoplastic syndromes.

There may be significant findings if the cutaneous condition is secondary to wider neoplastic processes and when the skin changes are the result of paraneoplastic changes due

to internal neoplasia. The important principle is that when haematological changes are detected, they are interpreted with care.

Where specific tumour markers are available this is clearly a major help but so far there are no well-established markers in any tumour condition in horses. Only the melanoma appears to have any specific identifiable protein characteristics. The identification of bovine papillomavirus (BPV1/4) DNA genomic material by PCR in fibroblast cells taken from a suspected sarcoid can be good confirmatory evidence.

Radiology

This is sometimes useful in the horse but the anatomical difficulties make the benefit variable. Sinus and nasal involvement can be detected in some cases. Oblique and ventrodorsal projections are often required to establish the true extent but these are more difficult and in practice conditions may not be feasible. Thoracic radiography is sometimes indicated for highly malignant tumours but these are particularly unusual in equine skin neoplasia apart from the lymphosarcoma complex.

Ultrasonography

A detailed ultrasonographic study of the region and the mass itself is often useful and is an under-utilized aid. Some masses such as the equine melanoma have a very characteristic ultrasonographic character **(Fig. CD18 • 1B)**. It may also be possible to use ultrasonography to guide biopsy instruments into typical or uncomplicated locations and so improve the outcome of the procedure. Space-occupying masses, fluid-filled cavities and displacements and distortions of the normal anatomy are easily recognized with experience. Ultrasonography can also provide a surgeon with useful information about the existence and/or the extent of a surrounding capsule **(Fig. CD18 • 1C)** as well as the proximity to other vital structures such as blood vessels and synovial structures (see **Fig. CD18 • 21B**).

Equine sarcoid

Profile

This is probably the most common cutaneous tumour of Equidae worldwide. All equids are liable to the condition including horses, mules, donkeys and zebras.

There is controversy over the aetiological role of papillomaviruses. A genome which closely resembles that of the bovine papillomavirus (BPV1/BPV2) has been identified consistently in sarcoid tissue from donkeys and horses (Reid & Smith 1992), but no vegetative virus particle has yet been conclusively demonstrated. Furthermore, the genome is apparently restricted to transformed fibroblasts. Keratinocytes do not appear to be involved directly. There are several examples where species-specific papillomaviruses are closely associated with non-papillomatosis neoplastic disease in that species, e.g. human papillomavirus (HPV) and cervical cancer and bovine papillomavirus (BPV) and bladder carcinoma. Nevertheless this is possibly the only circumstance when a papillomavirus from one species has any implication for another species.

The distribution of the lesions and the epidemiology strongly suggest that flies are an important part of the pathogenesis.

Clinically and pathologically, sarcoids present most of the features of a true neoplasm. The predominant cell type is a malignant/transformed fibroblast with characteristic in-vitro appearance and characteristics.

Sarcoids generally have a high capacity for local tissue invasion into the dermis and subcutis. However, true metastatic dissemination does not occur. Tumours can appear in freshly healing wounds in previously normal horses (Knottenbelt 2009), or reoccur at the same site following apparent complete surgical removal even up to 10 or more years later (Brostrom 1995).

Individual horses may be genetically susceptible and a familial tendency to sarcoid has been identified. An apparent predilection for Appaloosa and Arabians in America has led to the suggestion that this is a result of close contact with cattle (presumably infected with the bovine papillomavirus) but these breeds are not over-represented in any other populations of horse whether they are in contact with cattle or not. However, many horses may carry the genetic susceptibility to a varying extent, possibly explaining the variations in numbers and types seen on individuals. A familial tendency has also been identified, heavily supporting a genetic component. This susceptibility is almost certainly genetically orientated and is a property of certain MHC/ELA alleles. The presence of a W13 gene in the MHC complex appears to impart susceptibility; Lipizzaner horses do not appear to have a W13 gene and so the breed appears to be totally (genetically) resistant. The American Quarterhorse is apparently less affected than many other breeds. This is a complex issue with no definitive research progress.

CD

Sex (geldings more commonly) and age (1–6 years) predilections have been proposed, but no horse can be considered to be totally exempt from the condition on the grounds of sex, breed, colour or age. Sarcoids are undoubtedly rare in very young horses but can occur from a few weeks of age.

CD

Sarcoid is a locally aggressive, fibroblastic tumour occurring in six clinically recognizable forms (Fig. 18.9), all of which have a high propensity for recurrence (Knottenbelt & Pascoe 1994, Knottenbelt et al 1995, Knottenbelt 2005). An individual horse may have more than one sarcoid type and for the purposes of clinical description, each lesion is classified individually. There are, however, some individual horses that have a predominant type – sometimes exclusively. Although each of these forms is commonly identifiable, it is important to recognize that the 'less severe' forms can rapidly progress to the more aggressive types, particularly if they are traumatized. Furthermore, the specific types may not be clearly identifiable in every case and then the' mixed' category is applied. It is, however, usually obvious that even the mildest forms are indeed sarcoid – in-vitro fibroblast cell cultures derived from these are typical and indistinguishable from those taken from the more aggressive lesions. These factors suggest that both cell and host factors are responsible in combination for the variety of forms.

CD

Classification of the lesion type is important because the different types have different therapeutic demands and different prognostic implications (Fig. 18.9). What may suit a small occult lesion may not be applicable to an aggressive fibroblastic lesion. This classification is supported by pathological descriptions and is important because treatment options and prognosis vary for the different types of sarcoid. Furthermore, there is a general tendency towards progression from the milder superficial types to the more aggressive forms either

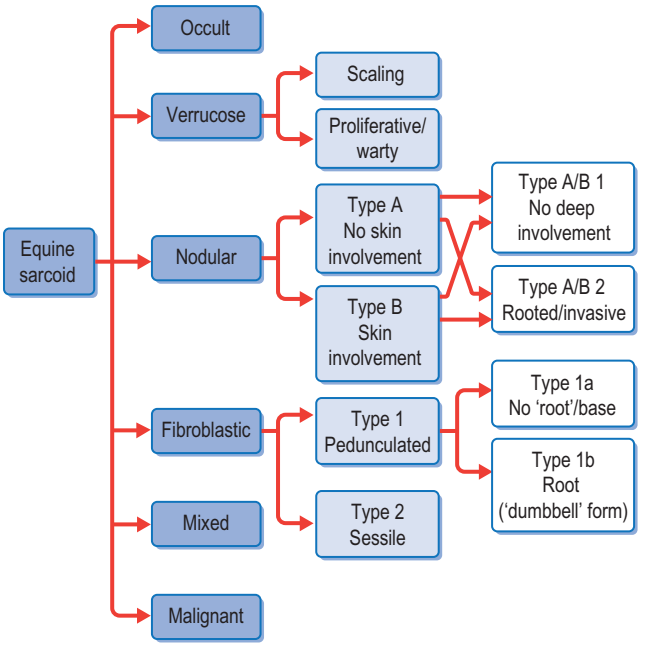


Figure 18.9 The classification of the sarcoid matters because the treatment for each type is somewhat different in both modality and application. This figure shows a useful schematic for the clinical classification which is based upon the predominate type in any particular lesion. The mixed type is only used when no dominant form can be applied.

Key points: Equine sarcoid

2 6 9 10 11 12 14 15 19 20 23 24

1 Commonest (skin) tumour of horses worldwide occurring in horses of all sexes, ages, breeds and colours and in donkeys, mules and other equids. Some indications of transmissibility across the horse and between horses suggestive of infectious aetiology and (fly) vector transmission. Genetic predisposition occurs in a significant number of horses. Nevertheless, some are not affected. Familial tendencies are recognized.
2 Six different types are recognized – each with its own therapeutic limitations and prognostic differences. Sarcoids range from flat hairless areas of skin (occult sarcoid) through verrucose, nodular and fibroblastic forms. A locally malignant form occurs rarely but this does not metastasize. Each sarcoid type has its own set of differential diagnoses and can coexist, making diagnosis of each individual lesion important.
3 The variety of lesions is both a help and a hindrance to diagnosis. There is a tendency for exacerbation following biopsy (or accidental trauma or intentional surgery). Every single lesion should be individually assessed before embarking on any treatment.
4 Treatment is always likely to be difficult and recurrences and new lesions on treated cases are common. There is no consistently effective treatment (save perhaps for radiation) and each type of sarcoid warrants individual assessment and adjustment of the treatment selection. The greater the effort in selection of treatment and the more accurately it is applied, the better the result.
5 The prognosis is always guarded since all treatments have failures and exacerbations and new lesions occur regularly. Irresponsible treatment methods will invariably result in a significant worsening of the prognosis.

as result of natural development or as a result of accidental or iatrogenic interference. The latter tends to cause a more aggressive and rapid exacerbation. The malignant form is rare but as a form of the disease it is a serious complication given that there are no effective treatments for this form and that the nature of the condition is likely to have a significant systemic and local effect on the horse. However, it is important to recognize that the sarcoid does not have defined boundaries between the various types. Usually the lesions have some aspects of other types and many progress from one type (usually occult or verrucose) to the more aggressive types (fibroblastic and malignant). There is, however, no predictable progression except to emphasize the dramatic changes that can follow interference or injury of a sarcoid.

The number of sarcoids affecting individual horses is very variable. Single lesions are encountered both spontaneously and associated with wound sites. Multiple lesions (often even thousands) also do occur. Sarcoids commonly multiply on the individual horse, sometimes very rapidly. Some lesions remain relatively, or even completely, static for years and then may suddenly expand without any apparent reason.

A few individuals show spontaneous full and permanent self-cure and a very few disappear spontaneously after being accidentally or intentionally (by biopsy) traumatized but both of these circumstances are exceptions rather than the rule. Interestingly, spontaneous remission usually means that the horse will not develop further lesions. However, the course of the condition is entirely unpredictable and it is unwise to make any firm predictions on the course or the likelihood of a successful treatment.

The clinical implications of sarcoid are, potentially at least, serious. Whilst a few superficial sarcoids may have no immediate effect on the horse or its performance, there is the real potential for both individual lesion exacerbation and an increase in the number of sarcoids on the horse. Fortunately sarcoid is a non-metastatic tumour but it can be very aggressive locally. The value of an affected horse is certainly less than the same horse would be without sarcoids. This reflects the general awareness amongst owners and veterinarians that:

1 The individual lesions have a tendency to exacerbation (although this may not be invariable and it can occur over a very variable time).
2 More sarcoids are likely to develop over time and these may not necessarily be of the same type as the original lesion(s).
3 The lesions will probably require some form of treatment and that this is likely to be problematic and expensive.
4 Failure of any chosen treatment method represents a major setback and subsequent treatments are likely to be even more difficult.
5 The condition may directly affect the performance of the horse or at least there will be periods of time during treatments or during fly seasons when the horse will not be usable.
6 The likely sale value of an affected horse will be lower than it would have been had it not had any sarcoids and when the horse is sold on at a later date the sarcoids may be a significant deterrent to purchase.

For these reasons the condition has become a major aspect of pre-purchase examination and insurance complications;

it is unlikely that an insurance company will provide insurance cover for neoplastic skin diseases if at the time of proposal there is any evidence at all of sarcoid.

In many neglected cases the disease becomes a significant welfare issue. As flies pester the horse, secondary infection and fly strike occur and serum and blood loss can be significant. Apart from these aspects, the cosmetic aspects are significant for many owners. Finally severe sarcoid has been a common cause of legal charge on the grounds of cruelty and neglect.

Clinical signs

The clinical recognition of the equine sarcoid is considerably complicated by the spectrum of variously recognizable skin lesions that are included in the pathological classification. Each of the recognized types has several variations and individual horses can have several different types at the same time. There is nothing prescriptive about what types of sarcoid occur in particular types of animal and in particular locations. However, there are broad principles that can be useful in some cases. The size of lesions is not related to the type and it is entirely possible to have small or large lesions of each recognized type, and there may be a wide variety of lesions and sizes on a particular animal.

The basic classification of the sarcoid is based upon the predominate type for any particular lesion and this matters because of the differences in differential diagnosis, treatment options and prognosis. Each type described in the following text has its own set of different differential diagnoses because of the spectrum of clinical presentations that are included in the diagnosis of the equine sarcoid. The presence of sarcoids of different types makes the diagnosis simpler but this can also trap the unwary clinician.

Occult sarcoid

Occult sarcoids manifest as hairless areas, often roughly circular and with a thinned skin in the affected area (Fig. 18.10 and **Fig. CD18 • 2A and B**). They usually contain one or more small cutaneous nodules (2–5 mm diameter) or roughened areas with a mild hyperkeratotic appearance (Fig. 18.11 and **Fig. CD18 • 3**) but these may or may not be present or obvious in every case. Careful palpation of the skin between thumb and forefinger will, however, usually confirm their presence even when very small.

Some occult lesions are extensive, covering very large areas of skin. The typical appearance is still the same but invariably nodules or verrucose areas can be identified within the affected skin (Fig. 18.12). In some cases the skin will feel diffusely thickened and in others the thickened areas are distributed almost randomly within the changed area. An area of changed/altered, slightly thickened skin with thin hair coat and slight changes in hair pigment may be all that is seen (Fig. 18.12C); these changes may be difficult to identify in winter-coated animals and create some difficulty with pre-purchase examinations.

The lesions are characteristically slow growing but they may progress to 'warty' verrucose growths (see p. 390) or if injured (or subjected to biopsy or ill-advised treatment attempts) may develop rapidly into fibroblastic lesions (see p. 394).

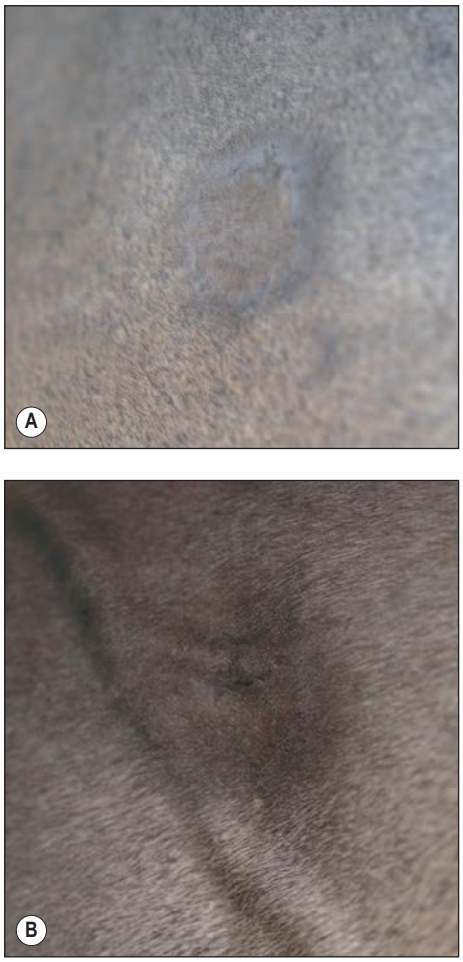


Figure 18.10 These two cases illustrate examples of the earliest and possibly the most difficult type – the occult sarcoid. Note the roughly circular nature of the lesions and the variable extent of alopecia; both of these signs can easily be overlooked. Where they develop in haired skin, the quality, density and sometimes even the colour of the hair coat in the vicinity can be significantly altered (see also Fig. 18.12) It is important to realize that in occult (and other types of) sarcoid, the term *early* is not synonymous with *recent*; some very early lesions remain static or slowly progressive for years. Careful palpation will usually but not always identify small shot-like granules within the boundaries of the lesion itself. **(A)** A very faint, alopecic circular area in the medial thigh in which two or three minute granular foci could be palpated. **(B)** A slightly more obvious area overlying a large cutaneous vein in the thigh region; this is a relatively common finding at this site.

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Figure 18.11 This example of an occult sarcoid is on the lateral belly wall and shows a more keratinized central zone surrounded by an obvious area of partial alopecia. This area shows characteristic histological changes relating to the epidermis and the hair follicles. The fibroblastic foci are usually very limited and subtle.

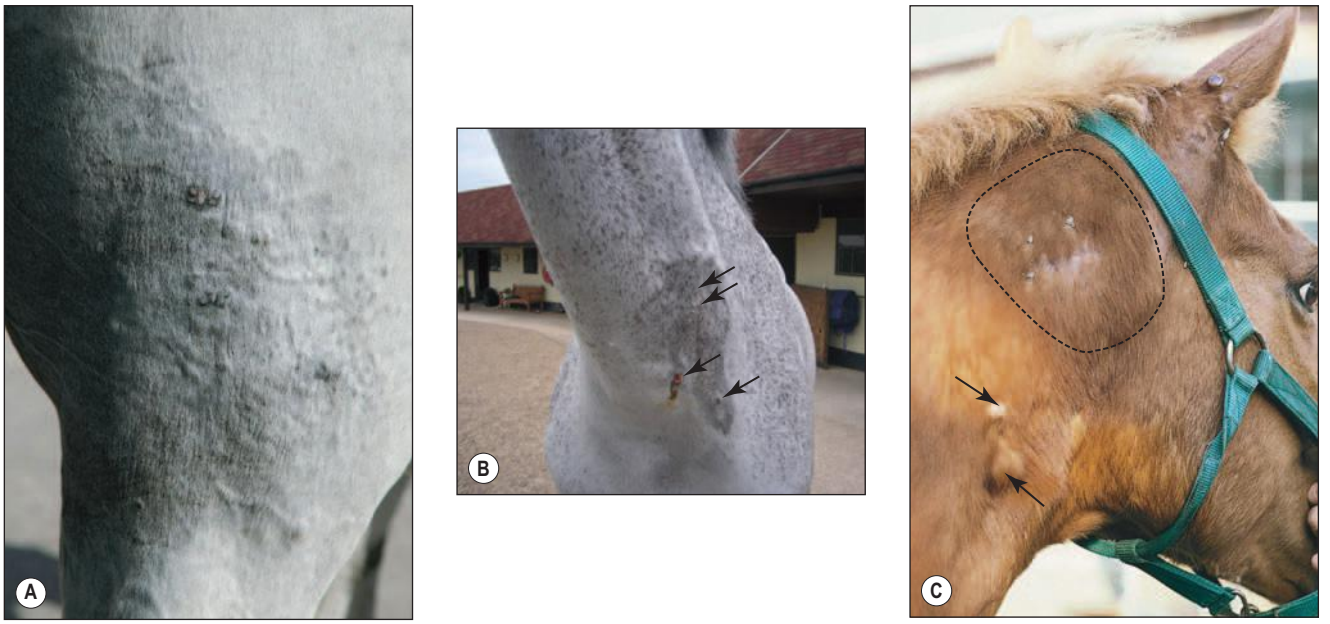


Figure 18.12 Wide areas of occult sarcoid are relatively common in the medial thigh, face and parotid/ear regions. The hair becomes sparse and of poor quality. **(A)** This grey gelding had a very extensive occult lesion occupying the whole medial and caudal thigh. Biopsy had been performed some weeks previously (the sites are visible) and the biopsy sites showed a much more aggressive fibroblastic nature histologically. (See also p. 70, Fig. 3.27.) **(B)** An extensive occult sarcoid in the left lower jugular groove. Small nodules are visible within the area (arrows) but other much smaller ones could be palpated rather than seen. One of these was biopsied to confirm the diagnosis. **(C)** This chestnut 23-year-old pony had a long history of sarcoid (a few of the more obvious lesions can be seen). The hair colour and density over the right upper neck and parotid regions changed when small nodules developed throughout this region.

Pain and pruritus are not features of occult sarcoids and there is no discernible systemic effect regardless of the extent or number of lesions.

Predilection sites include the skin around the mouth and eyes, the neck and other relatively hairless areas of the body including the medial aspects of the forearm and thigh. It is rare on the limbs and main body trunk but they can be found at any site in individual cases.

Cases have existed for over 15 years or more without alteration; however, extensive development of verrucose sarcoid or conversion into fibroblastic type sarcoid usually demands immediate attention. This can occur at any time with or without apparent insult.

Note

While the lesion remains as a static/quiescent hairless patch showing no evidence of progression in either size or number of nodules, it may be wise not to interfere. However, this represents the earliest stage of the condition and as such may be the best time to institute treatment.

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Differential diagnosis

- Idiopathic alopecia/hypotrichosis: very rare congenital disorder but can be acquired in later life. Rarely circular in nature. Nodules are not a feature.
- Dermatophytosis (ringworm): characteristic short-lived course over 6–12 weeks. Expansion of individual lesions with regrowing hair within them; dermatophytes can be identified in hair pluckings and cultures.

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- Bullous disorders including pemphigus foliaceus, vasculitis (immune-mediated and autoimmune forms): persistent affected areas or transient vesicles with hair loss and ulceration; characteristic histology.
- Burns: history of traumatic event.
- Rub marks, chronic skin rubbing and scarring: self-resolving once cause is removed – area affected usually corresponds to obvious cause such as rug or harness contact.
- Alopecia areata: benign alopecia often in circular or less defined areas and also can have large areas involved. No progression and no palpable nodules or hyperkeratosis/skin thickening.

Verrucose (warty) sarcoid

Verrucose sarcoids have a characteristic wart-like appearance in and on the skin (Fig. 18.13) and this appearance is the main reason for referring to sarcoids as 'warts'. The lesions are invariably alopecic when they occur on normal haired skin. The surface is scaly and it is often easy to dislodge the superficial accumulated keratinized material, without pain or haemorrhage.

In many individual lesions the area involved is relatively localized and in some there is an occult margin extending around the affected area (Fig. 18.14 and **Fig. CD18 • 4A–C**). There may, however, be many lesions each with subtle variations in the amount of occult rim and the amount of 'piled up keratin' and even some obvious lesions may be missed under long hair cover (Fig. 18.15).

Extensive areas can be affected and are often surrounded by an area of slightly thickened/changed skin with an altered, thin hair-growth pattern possibly reflecting a surrounding area of early occult sarcoid (Fig. 18.16 and **Fig. CD18 • 5A–D**).

When examined closely, individual lesions may be sessile (flat-based) or pedunculated (with a narrow neck) but coalescing lesions are more common than isolated small ones

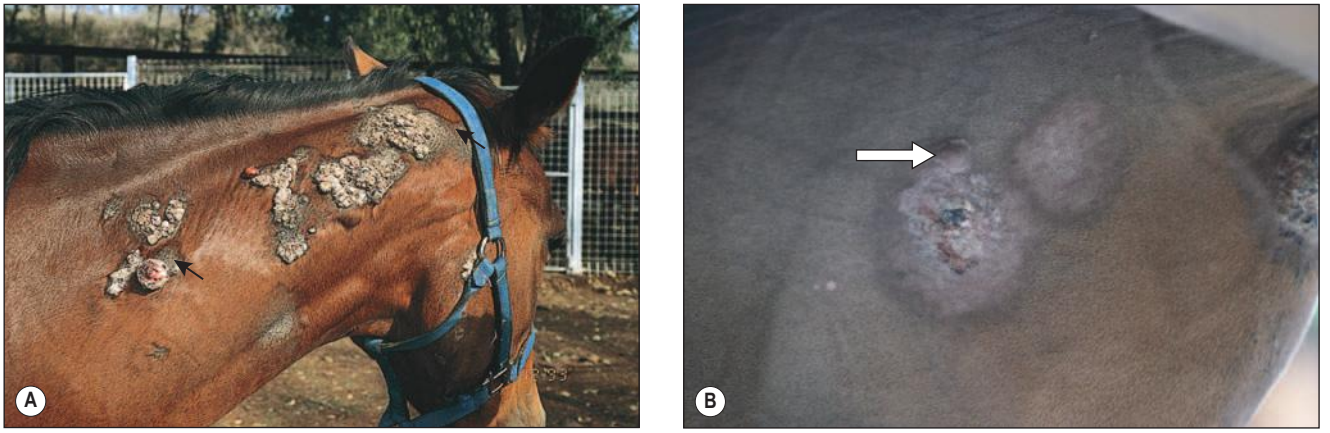


Figure 18.13 **(A)** Extensive and obvious verrucose (warty sarcoid) over the right neck of a middle-aged gelding. Within these areas several small ulcerated fibroblastic sarcoids could be identified. These were previous biopsy sites. Note also the occult circular region on the right jugular vein which developed at a site of venepuncture. **(B)** A verrucose lesion with a faint occult rim, adjacent to an obvious occult lesion in the thigh region with a more obvious verrucose lesion more anteriorly over the coxo-femoral joint. Note that verrucose lesions can also be roughly circular. Note also the more obvious nodule (arrow) and the relationship to cutaneous veins.

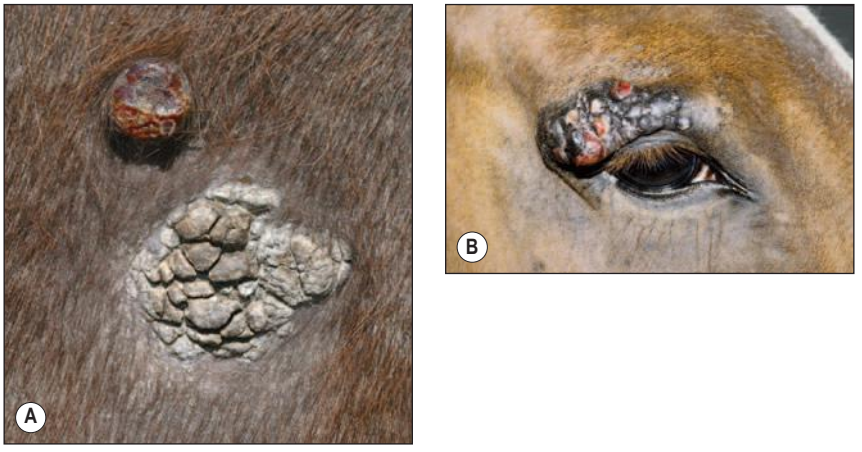


Figure 18.14 **(A)** A localized thick verrucose sarcoid on the medial thigh. The dense keratin could be rubbed off the surface with some ease. The lesion is still roughly circular. Note the adjacent ulcerated nodular sarcoid. Both have a faint occult rim. **(B)** A more fleshy proliferative verrucose sarcoid on the upper eyelid – this could be regarded as a subtype of the verrucose sarcoid and possibly shows some of the features of a fibroblastic lesion.



Figure 18.15 Verrucose sarcoids can be obvious but can also be much less easy to identify. This has implications for pre-purchase examinations in particular. This 9-year-old bay Shire gelding was presented because of the lesions on its ears **(A)** (see also **Fig. CD18 • 13B**) and its breast **(B)**. Close examination showed that under the long hairs of the ventral belly and groin there were many other lesions of a similar type **(C)**. Note again the occult margins which are obvious on the ear and breast verrucose lesions.

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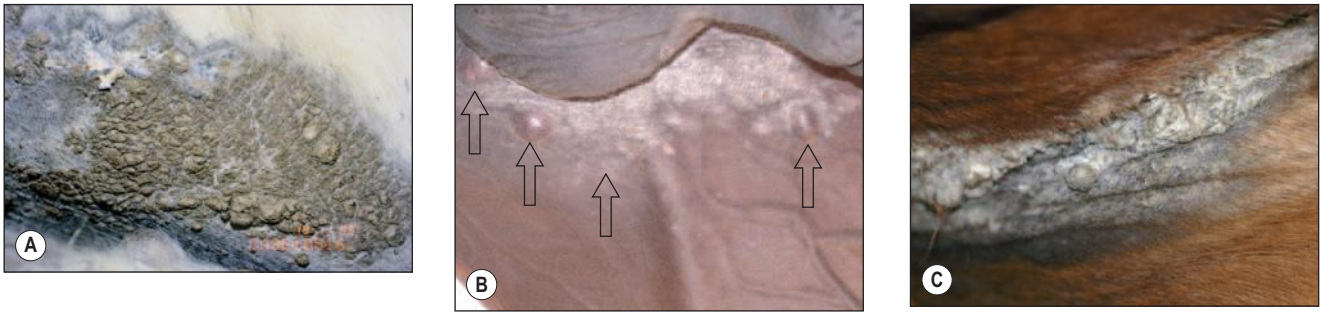


Figure 18.16 In some cases the verrucose changes can be very extensive and again within the affected area the hair coat can be absent, sparse or of very poor quality/colour. **(A)** This verrucose sarcoid on the side of face shows the reptile-like changes typical of this form. **(B)** The whole of the upper medial thigh including the groin fold and the adjacent belly wall was affected in this 14-year-old breeding stallion. This has implications for the ethics of breeding from the animal and of course there were major therapeutic problems. Note the obvious nodules also (arrows). **(C)** This thickened, inelastic, hyperkeratotic verrucose sarcoid developed in the axillary folds. Note the nodules. (See also **Fig. CD18 • 5A–D**.)

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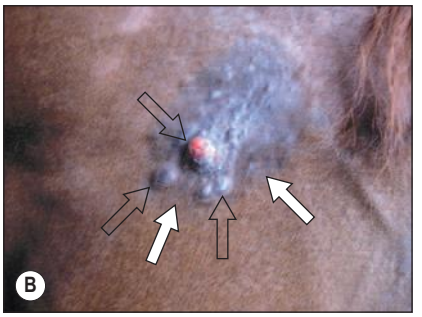


Figure 18.17 **(A)** Isolated verrucose lesions do occur with some regularity and are common on the face (as in this case) and the medial thighs. **(B)** A lesion in the medial thigh with a dominant verrucose form with an attached occult zone (arrows) and several obvious nodules within it (open arrows). This form serves as a reminder of the variable nature and the fact that the equine sarcoid is a progressive tumour that matures through several ill-defined stages.

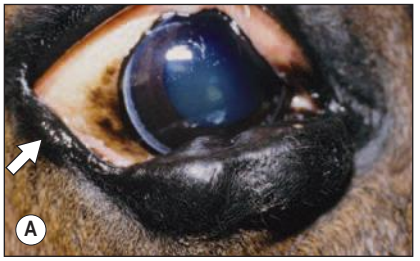




Figure 18.18 **(A)** Type A nodular sarcoid in the lower eyelid. Note that although the skin overlying the complex of two nodules was thinner than normal it could be moved independently of them. Note also the smaller type A nodule laterally (arrow). **(B)** A single type A1 nodule in the thigh. Although the skin was hypoplastic and thinned, the skin moved freely over the nodule and the nodule moved freely over the muscles beneath it.

(Fig. 18.17A). Small nodules may appear at any stage or over any area of the affected skin (Fig. 18.17B). Some areas may become ulcerated and crusting can be somewhat misleading when areas take on a more fibroblastic appearance.

The lesions are most often slow growing and not usually very aggressive until injured/insulted. Rubbing, biopsy, partial excision or minor or major trauma to the surface commonly result in a dramatic exacerbation to a true fibroblastic character over variable areas of the lesion. These may in fact develop whether or not they are insulted or traumatized. The history and clinical examination will rapidly establish the natural progression but the changes may be limited to smaller or larger areas of the verrucose sarcoid.

Pain and pruritus are not features of the verrucose sarcoid and secondary infections are uncommon even when superficial ulceration occurs.

Predilection sites include the face (and particularly the eyelids and periorbital skin), body, axillae, and groin/sheath areas. This form is rare on the limbs.

Differential diagnosis

- Papillomatosis (warts): self-resolving papular disorder sometimes merging to involve larger areas. Nodular expansions in skin may be palpated but hair loss is not a feature; juvenile forms are self-resolving.
- Linear keratosis/epidermal naevus: persistent linear alopecic and/or hyperkeratotic areas usually on neck, buttock or shoulder region. Non-progressive.
- Dematophytosis (particularly when associated with *Trichophyton verrucosum*): circular lesions that expand and spontaneously resolve over 6–12 weeks. Characteristic epidemiology and mycology.
- Chronic blistering: history of application of counterirritant chemicals.
- Hyperkeratosis (e.g. chronic sweet itch), skin irritation/rubbing (responsive hyperkeratosis): restricted to tack/harness contact locations. Changes may be permanent.
- Equine sarcoidosis (chronic granulomatous disease): systemic signs and less defined cutaneous lesions.
- Equine (cutaneous) systemic lupus erythematosus-like syndrome (dermal form): diffuse seborrhoeic disease with multisystemic involvement.
- Squamous cell carcinoma (particularly of face): usually more destructive, ulcerated and haemorrhagic).

Nodular sarcoid

The nodular lesions are easily recognizable as firm, well-defined subcutaneous, usually spherical nodules of 10–75 mm diameter but individual nodules can be much larger or smaller and there can be many nodules of varying size, complexity and location (Fig. 18.18).

Nodules fall into two distinct categories based upon the relationship/involvement of the overlying skin. Each of these is further divided into two based upon the extent of involvement with surrounding tissues (Table 18.3).

Type A nodules Type A nodules have no cutaneous involvement – they fall into the historically recognized fibroma description. The skin can be moved freely over the nodule and there are no changes in the overlying skin (except in a few very long-standing cases in sites where there is very thin skin such as the eyelid when the overlying skin can become atrophied and very thin).

These are then usefully divided into two subtypes. In type A1 the nodule is enclosed in a loose fibro-cellular capsule; the nodule can be moved independently of the skin *and* the underlying structures. In some cases the weight of the nodule creates a skin pedicle – this can only occur with a type A1 nodule because binding into subcutaneous tissue will preclude the weight from hanging on the skin alone **(Fig. CD18 • 6A and B)**. Type A2 nodules have no cutaneous involvement but are locally invasive with a 'bound-down' character. This precludes independent movement of the nodule relative to the deeper structures in the subcutis and beneath. This is a very common type around the eye (Fig. 18.19).

Type B nodules These have visible or palpable cutaneous involvement that means the overlying skin cannot be moved over the nodule. Usually the overlying skin is obviously abnormal (either hyperkeratotic or thinned and alopecic) and

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Table 18.3 The clinical and pathological features of nodular sarcoids

Nodular sarcoid		Clinical and gross pathological features
Type	**Subtype**	
A **No cutaneous involvement** Single or multiple Individual or multilobular No occult changes directly associated with nodules	1	No deep tissue involvement Loose fibro-cellular capsule Defined margins
	2	Deep tissue involvement Invasive 'bound-down' character Ill-defined margins
B **Cutaneous involvement** Single or multiple Individual or multilobular Possible occult and occasionally verrucose margin on skin	1	No deep tissue involvement Loose fibro-cellular capsule Defined margins
	2	Deep tissue involvement 'Bound-down' nature Ill-defined margins



Figure 18.19 Extensive type A2 nodules under the skin of the lower eyelid (and a single large type A1 nodule under the nasal third of the upper eyelid). The skin could be moved over the lesions but they were firmly bound down to the eyelid muscles beneath. This arises through deep extensions into the eyelid muscles. This type also occurs at many other sites including the thigh.



Figure 18.20 This complex of type B nodules shows the variety of cutaneous involvement that is possible. In some cases the skin is ulcerated while in others it is intact but changed. In all cases the skin cannot be moved independently of the nodule(s) but the cardinal property of B1 nodules is that they do move freely over the deeper structures.

occasionally ulcerated. In some cases the skin is ulcerated completely and then a fibroblastic lesion can develop (Fig. 18.20 and **Fig. CD18 • 7**). This type is also divided into two subtypes according to the deeper pathological nature – type



Figure 18.21 This extensive complex of nodules shows the aggressive nature that type B2 nodules can adopt around the eye. In this case the skin is clearly involved and 'attached' to the lesions and it was impossible to move the lesions over the deeper structures. This very bound-down characteristic has a major influence on choice of treatment and (deleterious) prognosis. This type is common in many sites including the face, ear, eyelid, thigh and axilla.

B1 nodules have no deep tissue invasion while type B2 are 'bound-down', seemingly attached to the surrounding and deeper tissues. However, the firm involvement of the skin may preclude a proper test of its deeper invasive nature. All type B nodules are potentially dangerous and this is particularly so around the eye and the side of the mouth where invasion can be extensive (Fig. 18.21 and **Fig. CD18 • 8A–C**) (Knottenbelt & Kelly 2000). Multiple type A and B nodules are commonly found on an individual horse (Fig. 18.22).

The overlying skin may become thin/hypoplastic or atrophied over larger nodules of both A and B types. Type B nodules may have some adjacent occult changes in the skin but this does not occur with type A1 or A2 nodules. When the skin ulcerates the masses quickly take on a more aggressive fibroblastic character. A similar aggressive fibroblastic response commonly follows iatrogenic or accidental damage.

The number of nodules varies widely – single, few, several or even hundreds are common. Many cases of both



Figure 18.22 This case illustrates the extent of nodular involvement that commonly occurs in the medial thigh regions. It is also common on the face and the axillae. Note that there are types A1 and A2 and types B1 and B2 nodules here. The type B nodules have very variable skin involvement.

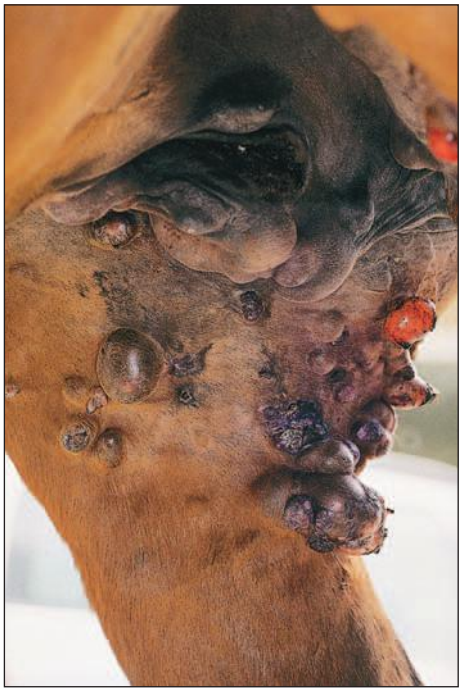


Figure 18.23 This multiple nodular complex case shows the variety of interlinking nodules that are common in certain individuals. Interlinking nodules occur in many sites and implies a degree of extension into adjacent skin and subcutis with clones of expanding tumour cells developing.

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type A and type B nodules have multiple interlinking nodules (Figs 18.22 and 18.23).

Pain and pruritus are not common features – the condition is often very benign in this form but some can become extremely complex and invasive.

Predilection sites include the groin, sheath or eyelid regions. Nodular sarcoids are very rarely found on the distal limbs possibly because they are rarely associated with wounds.

Treatment options vary for the various clinicopathological types so an accurate examination/and description is important.

Differential diagnosis

- Fibroma/fibrosarcoma: true fibroma as opposed to sarcoid is very rare and it may be that these fall squarely into the spectrum of sarcoid. (Fibroma, fibrosarcoma and neurofibroma are commonly separated by some pathologists but others view these as variations of sarcoid.)
- Neurofibroma (schwannoma): well-defined but unencapsulated mass composed of fibroblasts/spindle cells with a highly collagenased stroma with little myxoid material said to be derived from nerve sheath cells. Most often reported around the eye. This may be a variant of the sarcoid also.
- Equine eosinophilic granuloma/collagenolytic granuloma: benign, usually multiple, small static nodules with a characteristic distribution on the back, neck and buttock regions. Distinctive clinical and histological appearance.
- Melanoma: isolated or multiple tumours in grey horses. Other forms occur in non-grey horses but most are solid and black.
- Equine cutaneous mastocytosis/equine malignant cutaneous mastocytosis/congenital equine mastocytoma: rare, single nodules in or under the skin. Possibly pruritic.
- Lymphosarcoma/lymphoma/cutaneous histiocytic lymphoma: multiple cutaneous nodules with diagnostic histological nature.
- Dermoid cyst: dorsal midline mainly, present since birth and definitive excisional biopsy.
- *Hypoderma* spp. (warble fly) cysts: seasonal, single or few dorsal inflamed nodules that develop a pore centrally and sometimes a live grub can be identified.
- Foreign body/fly bite reaction: isolated, transient oedematous reactions.

Fibroblastic sarcoid

These tumours have a characteristic aggressive, fleshy, ulcerated appearance, commonly with a fibro-cellular scab over the surface and secondary infection (Fig. 18.24). There is commonly superficial infection, serum exudation and the lesions are easily traumatized causing haemorrhage.

Fibroblastic sarcoid can be conveniently divided into two broad, clinically recognizable groups (Table 18.4).

Type 1 (pedunculated) fibroblastic sarcoid These have a narrow skin 'pedicle' with a fleshy ulcerated (often bleeding) crown **(Fig. CD18 • 9)**. Quite often it is possible to palpate normal, thin skin adjoining the lesion but sometimes the pedicle is very short **(Fig. CD18 • 10)** and then the true nature of the lesion can only be defined by ultrasonography or after surgical or other procedure to debulk the external



Figure 18.24 A typical fibroblastic sarcoid showing the characteristic fleshy haemorrhagic surface which is inclined to crust formation, foreign body adherence and infection. Some lesions will bleed easily while others may exude clinically significant amounts of plasma. These lesions are somewhat liable to myiasis (see p. 214) but this is probably less common than with wounds and other chronic skin infections.

Table 18.4 The clinical and pathological features of fibroblastic sarcoids

Fibroblastic		Features
Type	**Subtype**	
1 Pedunculated	a	Distinct pedicle No deep tissue involvement
	b	Distinct pedicle Wide/palpable 'root' Invasive 'bound-down' character Ill-defined margins of root
2 Sessile/broad based	'Bound-down' nature Ill-defined margins	

mass. This group has two clinically significant and recognizable subgroups.

- *Type 1a* (*pedunculated*) sarcoids have no discernible root – the base feels free of any thickening or palpable tumour and there are no histologically detectable deep extensions of the sarcoid tissue (Fig. 18.25A). The detection of deep extensions is clearly difficult in early lesions but usually it is possible to get an impression of normality both within and deep to the pedicle.

Note

Where these lesions occur on the distal limb regions in particular they are a major problem and are often associated with a previous wound. As the clinical appearance can be very similar to granulation tissue it is important to establish when and if a wound occurred and to biopsy the tissue.



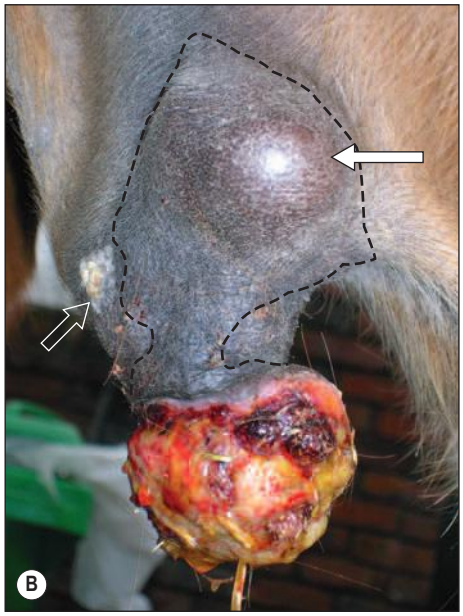


Figure 18.25 **(A)** A typical type 1a (pedunculated, non-rooted) fibroblastic sarcoid. Note the very fleshy and aggressive-looking mass but the relatively narrow stalk. Of course from a clinical perspective alone it is sometimes impossible to be sure that there is no deep extension beyond the skin level. **(B)** This fibroblastic sarcoid fulfils the criteria for classification as a type 1b (pedunculated but rooted) or dumbbell form. The main lesion is the ulcerated mass whilst the narrow stalk expanded proximally to a large nodular and invasive mass in the subcutis above (arrow). The palpable form of the sarcoid is shown in the dotted lines. This has involved the skin surface and could on its own be classified as a type B2 nodule (see above). Note also the adjacent small verrucose lesion which was incidental (open arrow). The horse had many such verrucose lesions but only one fibroblastic sarcoid.

- *Type 1b* (*pedunculated and rooted*) sarcoids have a distinctive root that is palpable beneath the narrow pedicle; it may not be extensive on palpation but the significance of any deep thickening either within or below the pedicle can be used to confirm the type 1b fibroblastic sarcoid (Fig. 18.25B). The difference between these and the type 1a (non-rooted pedunculated sarcoid) can be visible but sometimes can be better appreciated by palpation (Fig. 18.26). In some cases the pedicle can be very short (particularly where there is little loose skin such as on the distal limb and face) The term 'dumbbell' form gives a good impression of the clinical and pathological nature of the type 1b (pedunculated, rooted) fibroblastic sarcoid.



Figure 18.26 This fibroblastic sarcoid on the medial thigh bled heavily and although there were no reports of any previous treatment, palpation and examination revealed a futile attempt to ligate the base with a piece of string! The ligation had been far from effective, probably because this non-rooted (type 1a) fibroblastic sarcoid had not been palpated properly and the presence of the pedicle had not been identified.



Figure 18.27 This type 2 (sessile) sarcoid showed the characteristic broad and invasive base typical of this type. It was possible to palpate the mass deep in the axilla. Significantly, this horse had a previous small verrucose lesion at the site for some years before this dramatic expansion occurred over 9 weeks.

Type 2 (sessile) fibroblastic sarcoid In this case the lesions are sessile; they have a broad base without any detectable narrowing at the skin level. This type invariably has extensive involvement beneath the visible lesion and this may extend some distance from the apparent skin margin. Deep and lateral cutaneous involvement occurs and so the sarcoid may be bound down to the deep tissues with a diffuse ill-defined margin in the dermis/epidermis. Frequently these lesions are exudative and bleed easily (Fig. 18.27 and **Fig. CD18 • 11A and B**).



Figure 18.28 The history of this aggressive type 2 sessile fibroblastic sarcoid included several vain attempts to remove the expanding lesion by surgical excision. After each attempt healing was disrupted and the sarcoid returned with increasing aggression. Treatment was very problematic and prolonged. (Figure courtesy of Dr Martin Stokli.)

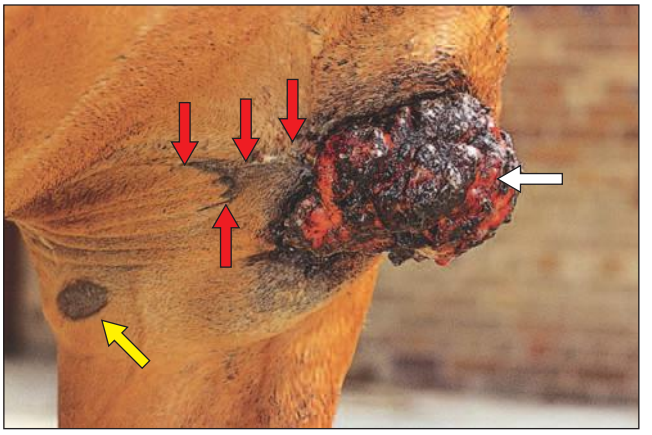


Figure 18.29 This pony developed a highly aggressive, sessile, rooted fibroblastic sarcoid (white arrow) at the site of a relatively trivial wound that appeared to heal well until 6 weeks post injury (red arrows shows the 'healed scar'). Several smaller focal sarcoid lesions were present along the wound. Clinical examination revealed an occult sarcoid in the axilla (yellow arrow) that had been present for 5 years without apparent change.

One of the common features of this type of sarcoid is the history of interference either intentionally or accidentally. Even minor trauma can trigger an expansion of the lesion and repeated insults can result in a challenging clinical position (Fig. 18.28 and **Fig. CD18 • 12)**.

This form is particularly attractive to surface-feeding flies and in summer months they can cause considerable extra surface ulceration and infection. In spite of their open nature, myiasis seldom seems to occur in this type of sarcoid (in contrast to the pedunculated form where this is a much more frequent occurrence).

Fibroblastic sarcoids of both broad types are common at sites of wounds (Fig. 18.29), and particularly on distal limb wounds, especially if other sarcoids are present elsewhere. Concurrent (excessive) granulation tissue growth serves to confuse the diagnosis. Accidental wounds which fail to heal may contain significant sarcoid components in the wound



Figure 18.30 This aggressive, sessile, fibroblastic sarcoid developed following repeated unsuccessful attempts at surgical removal. The site and the circumstances precluded any other treatment method. Initially the tumour had been diagnosed as granulation tissue.

margins and irregularly and unpredictably mixed with granulation tissue.

Both pedunculated and extensive sessile tumours with prominent ulceration and serum exudation are commonly encountered. The latter may reflect single or repeated insults to the 'lesser' forms but may develop spontaneously.

Even when the lesions are highly ulcerated and aggressive, pruritus and pain are rarely present. Very large pendulous sarcoids may irritate the horse and repeated trauma can cause heavy bleeding. Secondary infection and even fly strike (myiasis) occur and so swabs taken from the surface for cell identification often reveal opportunistic pathogens that may confuse the diagnostic process. Infection is seldom a significant issue, however.

Surgical wounds are also liable to sarcoid development. Dehiscence of the suture line within 2–7 days (or up to 5 years afterwards) for no apparent/obvious reason may be suggestive of sarcoid development.

Predilection sites include the groin, eyelid, lower limbs and coronet, sites of skin wounds at any location and sites of any other types of sarcoid subjected to trauma or insult. At some anatomic sites this type of sarcoid can carry a very poor prognosis (Fig. 18.29).

In spite of their aggressive appearance, and in common with other types of sarcoid, they do not metastasize. However, they can spread locally in the dermis by local invasion/extension and then they may justifiably be classified as malignant.

Repeated insult (accidental or iatrogenic) appears to encourage local subdermal and dermal invasion. Therefore, tumours which have had previous unsuccessful treatment attempts can be extremely difficult to manage and become progressively more sessile and malignant in nature (Fig. 18.30).

Differential diagnosis

- (Exuberant) granulation tissue: difficult to differentiate in some cases. Biopsy can be very helpful but false results can occur. Responds positively to steroid and grafting techniques.
- Habronemiasis: seasonal, wound related with typical sites such as the face, penis and wounds; tends to be more ulcerated and parasitic larvae can be found easily in washings.
- Neurofibroma (schwannoma)/neurofibrosarcoma (ulcerated): ulcerated lesions may in fact be a form of sarcoid but can be specific and less localized than many sarcoids.
- Fibrosarcoma: rare, usually regarded as a form of sarcoid very similar to fibroblastic forms.
- Squamous cell carcinoma: highly destructive. Typical locations and relationship to non-pigmented skin and sunlight exposure in some cases. Typical histology.
- Sweat gland tumour: very rare, definitive histology.
- Giant cell sarcoma: often associated with foreign bodies or necrotic tissue, slow expansion at first and remain localized. Often necrotic centrally.
- Haemangioma/haemangiosarcoma: typical histology and less fleshy appearance; bleed easily.
- Cavernous haemangioma/vascular hamartoma: young foals mainly; highly vascular and easily damaged.
- Botryomycosis/pyogranuloma/pseudomycetoma: definitive pathology and microbiology to identify microabscessation; history of non-healing wound.
- Pythiosis ('swamp cancer'): restricted geographically and in warmer, damp conditions *Pythium* spp. can be identified directly from exudate and scrapes from the surface.
- Mycosis fungoides: multiple fungating areas associated with destructive/ulcerated forms of multicentric lymphosarcoma.

Mixed (verrucose, nodular and fibroblastic) sarcoid

In clinical circumstances almost every lesion could be classified as mixed in that most if not all sarcoids have several different types within the affected site. The mixed classification is, however, reserved for those lesions where there is no predominate type.

This type of sarcoid probably represents a progressive/transient state between the verrucose/occult types and fibroblastic/nodular types and in many cases there are obvious sarcoid types visible. It is entirely possible that a single area actually contains all the available types to varying extents (Fig. 18.31 and **Fig. CD18 • 13A–D)**.

CD

Variations in proportion of the types is infinite and complex mixtures of any or all of the above types (containing both verrucose and fibroblastic elements) are common in long-standing lesions or those subjected to repeated minor trauma (such as rubbing by tack or harness or those that are subjected to partial or irrational treatment attempts). They become progressively more aggressive as more fibroblastic transformation takes place. This is also a common consequence of biopsy or injury.

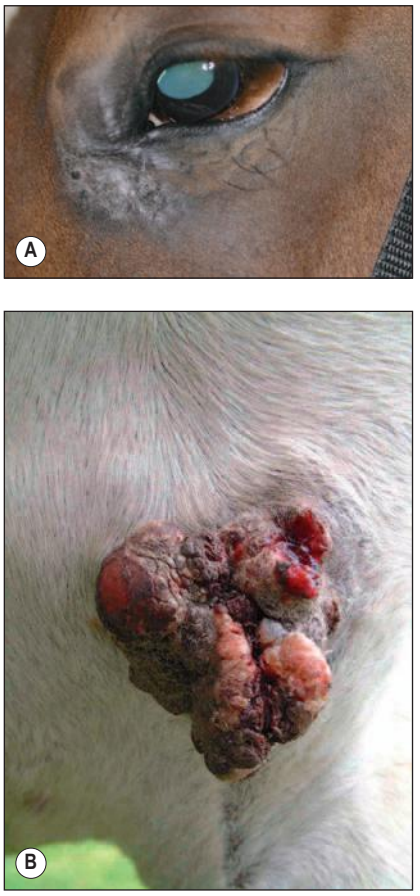


Figure 18.31 Many sarcoids are in fact mixed but usually it is possible to identify a predominate type. The mixed classification is restricted to those lesions in which a predominate type is not definable. **(A)** This mixed periocular sarcoid contains approximately equal elements of verrucose and nodular sarcoid. **(B)** A mixed lesion containing elements of fibroblastic and verrucose as well as nodular sarcoid in the axilla of a young Welsh pony. **(C)** A mixed nodular and verrucose sarcoid in the throat region. **(D)** This whole lesion is not a mixed sarcoid even though it appears to have multiple elements. This complex in the medial thigh includes a large type A1 nodule which was an independent mass. The surface verrucose nodular superficial sarcoid complex is the mixed component.

Predilection sites include the face, eyelid, groin and medial thigh but the reality is that most lesions at most sites could justifiably be termed mixed sarcoid.

Differential diagnosis

- There is little problem with recognition of the mixed sarcoid – the presence of more than one form of sarcoid in a single lesion is virtually pathognomonic.
- Complex mixtures of granulation tissue in verrucose and/or fibroblastic sarcoid: the fibroblastic forms of sarcoid can contain granulation tissue in various proportions and so biopsy should be taken carefully.
- Habronemiasis (particularly if affecting ulcerated verrucose sarcoid): seasonally related, geographically restricted, superficial cutaneous ulceration and non-healing wounds. Larvae can be identified in washings. Rapid response to specific therapy but often recurrent in succeeding years.
- Pemphigus complex: these lesions are seldom proliferative and are usually generalized or restricted to the mucosal junctions and specific skin areas. The pathology is definitive.

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Malignant sarcoid

This is a recently described rare variation (Knottenbelt et al 1995) of the equine sarcoid. A history of repeated trauma to other types of sarcoid, e.g. repeated surgical interference or applications of inappropriate medications, is commonly involved. However, some cases have no such history and the clinical presentation varies. The malignant classification can justifiably be given to a development of typical multiple, locally invasive sarcoids of nodular and fibroblastic character (Fig. 18.32); they do not always have to be widespread but can be reasonably localized.

Whilst visually extensive and distressing, sarcoid such as those that develop on the cheek and face are obvious candidates for this classification; cords of nodules and ulcerated lesions over a wide area could be simply large numbers of unconnected nodules or fibroblastic lesions but where connections occur subcutaneously the classification of malignant sarcoid should be used. Individual horses may have a single area involved such as around the eye or in the groin (Fig. 18.32) but others have multiple severely affected areas at some or all of the characteristic locations (Fig. 18.33).

Locally malignant sarcoid is a possible sequel to surgical excision and to wounds – particularly where the latter are subjected to repeated trauma. Whilst most of these remain reasonably local, some track down lymphatic vessels (Fig. 18.34).

Others show extensive infiltration of lymphatics (cords of tumour are commonly palpable) with numerous ulcerative nodules and surface involvement **(Fig. CD18 • 14A–D)**. Although the local lymph nodes can be enlarged there are no reports of sarcoid tissue within them. No disseminated metastases have been reported for any sarcoid.

Predilection sites include the jaw, face, elbow and medial thigh areas in particular.







Figure 18.32 The malignant form of sarcoid has particularly aggressive clinical behaviour, although it does not necessarily have to be extensive. Common sites for this form are the side of the face **(A, B)** and the elbow region, particularly in the region of the olecranon **(C)**. (Further pictures of the case shown in part C are shown in **Fig. CD18 • 14**.)

CD





Figure 18.33 **(A)** This highly invasive malignant sarcoid lesion in the medial thigh grew rapidly from a relatively localized ulcerating nodule over a period of 7 weeks. The whole medial thigh was infiltrated by sarcoid. **(B)** The malignant form can occur at any site although the foot is a singularly rare location. This sarcoid expanded to involve the hoof wall, the coronary band and the pastern skin. Treatment was impossible.



Figure 18.34 A small verrucose lesion was removed surgically from the skin overlying the left jugular groove. Over the following 4 months an aggressive and invasive malignant sarcoid developed at the site. Histology showed that the tumour was tracking along veins and lymphatics.

Differential diagnosis

- Squamous cell carcinoma: localized, definitive histology.
- Subcutaneous mycosis: chronic, localized masses, geographically restricted.
- Lymphangitis: diffuse swelling.
- Glanders: geographically restricted.
- Epizootic lymphangitis/cutaneous histoplasmosis: geographically restricted.
- Scar and partially healed wounds, especially if complicated by foreign bodies, infection or cheloid/hypertrophic scarring: history of wounding.
- Lymphoma/lymphosarcoma (generalized or cutaneous histiocytic lymphosarcoma): systemic involvement, definitive histology.

Diagnostic confirmation of sarcoid

The tendency to progression of superficial (occult or verrucose) and nodular sarcoid to the more aggressive fibroblastic forms makes it important to obtain an early diagnosis and consider prompt and appropriate treatment. An accurate history is therefore one of the best (and easiest) available clinical features. Many cases have several different types of sarcoid and that can help with the diagnosis but it can also be misleading.

- The clinical features are usually clearly recognizable but confirmation of the diagnosis is not always straightforward.
- Complications can arise when any of the differential diagnoses for the individual types are present simultaneously. Single lesions or single types on an individual horse are more difficult to diagnose definitively on clinical grounds alone. Multiple tumours with the characteristic features of more than one type of sarcoid on an individual horse make the diagnosis



Figure 18.35 Surgical removal of a localized 'wart' from this site was thought to be successful when the wound appeared to be healing well. The original location of the surgical incision is shown by the dotted line. However, the wound broke down distally at 8 days – 1 day after suture removal, and this massive sarcoid developed over the following 2 months. Biopsy confirmed the diagnosis. Note the smaller lesions in the more proximal parts of the wound.

relatively simple – there is no other disease with the same range of clinical features and types. However, it is also easy to assume that all the lesions are in fact variants of sarcoid when in fact they may be other coexisting conditions. It is always wise to asses each suspicious area and consider the full range of differential diagnoses for that single lesion before moving on to the next one.

- Partial or excisional biopsy will almost always provide a definitive diagnosis but both carry attendant risks. There is a recognized risk of exacerbation following surgical biopsy. The attendant risks are probably minimal if a fine needle aspirate is taken from the margin of a suspected lesion (see Chapter 3) but the interpretation of these is much more difficult and so in exchange for the safety there is a risk of a false positive or negative diagnosis. A total excisional biopsy rather than a sample biopsy should therefore be performed if this option is to be used and is feasible (see Chapter 3). It is very important, however, that a proper therapeutic plan should be prepared to cope with either the positive or the negative diagnosis. Biopsy with no plan is a dangerous state and this is even worse when the plan is benign neglect – the risks are too high and if no definitive therapeutic plan can be made then a biopsy should probably *not* be taken at all.
- A diagnosis may be made retrospectively when the tumour either becomes progressively more aggressive or the surgical site fails to heal normally (Fig. 18.35 and **Fig. CD18 • 15**). Wound breakdown and proliferation of fibroblastic tissue from the margins and depth of an excisional surgical site in a horse with other suspicious lesions are strong indicators that the lesion is a sarcoid. Early biopsy of this new tissue is indicated to confirm the presence of sarcoid and to eliminate other causes of wound breakdown (such as foreign body reactions, exuberant granulation tissue).
- Complications can arise particularly when sarcoid is diffusely mixed with granulation tissue. Biopsy may be very misleading if only one of the two distinctive tissue types is recognized in a particular specimen of the tissue. A shave biopsy may be grossly misleading – it may only reveal granulation tissue or may only reveal sarcoid and so an inappropriate therapeutic choice can be made.
- Hair pluckings and skin scrapings can be used to eliminate dermatophytosis (ringworm) and other ectoparasites in the case of occult and minor verrucose lesions. This procedure carries only minor risks of exacerbating a sarcoid.
- Superficial swabs taken from the surface of a suspected lesion can be subjected to PCR testing. Identification of the BPV genome in these swabs is reported to be highly significant (Martens et al 2001). However, the genome is not found in keratinocytes and so a deeper swab may be required if the skin is intact and hyperkeratotic.

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Pathology

Since the sarcoid is a fibroblastic tumour the main cell involved is the transformed fibroblast and recognizable by fibroblast proliferation either diffusely or focally. There are characteristic whorling patterns of fibroblasts with a variable collagen matrix and randomized vascular patterns. Epithelial changes that are recognized both clinically (hyperkeratosis, alopecia and hair colour and hair shaft thickness alterations) and pathologically (epidermal hyperplasia, dermo-epidermal activity) are probably secondary changes arising from biologically active mediators and cytokines produced as a result of the genetic changes within the transformed fibroblast. There are very few sarcoids in which a fibroblast aggregation cannot be identified by careful examination. The correct sampling is clearly critical to the diagnosis and so when deciding which part of a suspected sarcoid lesion to sample it should be examined very closely to maximize the possibility of the correct diagnosis.

Treatment

Treatment should follow as soon after diagnosis as possible. Suspicious lesions can justifiably be treated immediately after biopsy using an appropriate regimen (see below). In most cases diagnosis does not require a biopsy and so treatment can be instituted immediately.

General treatment considerations

The prognosis for all cases is very guarded and owners must be made aware of the possible serious complications which can arise from this disease. Owners must be apprised of the limitations, cost and likely/possible outcome of the various treatment options. No case of sarcoid can be considered to be free of the disease, even following apparently successful treatment. Remission periods of 5 years are usually considered reasonable in many neoplastic conditions but the sarcoid is complicated by known tendency for longer periods of dormancy and by the genetic predisposition to the disease.

Note

A horse (donkey, mule or zebra) that has even one sarcoid lesion is, ipso facto, liable to the disease and remains so for life. New lesions and recurrence at sites of previous lesions should be expected throughout the life of the affected animal.

There is a strong likelihood therefore that prolonged or repeated treatments will be required. However, complete, spontaneous regression of tumours is occasionally seen (Brostrom 1995) and owners can then be reasonably assured that the condition is *unlikely* to recur. This does *not*, however, apply when every single lesion on an individual is treated 'successfully' – in these cases there is probably no immunological response and so they are in fact still liable to develop more lesions and recurrences at the site of previously treated lesions.

Factors affecting choice of treatment method

1. *Individual patient variations.* There is no universally accepted treatment. What works in one case may not work in another – even when similar lesions are treated on similar horses in the same way, different responses can be expected.
2. *The location of the sarcoid.* In some sites treatment options are limited. Functional anatomical problems are very important and collateral damage can be more serious than the original condition. For example, there are limited options for treatment of lesions in the eyelids – the tendency to local invasion will usually preclude surgery (Knottenbelt & Kelly 2000) and any caustic material that gets into the eye may cause catastrophic damage. There a major risk if cytotoxic topical agents are used but also any consequent scarring, deformity or tissue defects will possibly result in loss of the eye. Similarly, lesions inside the prepuce or on the penile skin, coronary band, over major limb joints and those around the mouth and ears have severe restrictions on therapeutic choices.
3. *The type of sarcoid.* The type of sarcoid will inevitably influence the choice of treatment and it is important to have a broad classification of the sarcoid types. Localized, defined sarcoids in suitable places are probably the best ones to treat but the clinician will never have the luxury of dictating what type of lesion a particular horse will develop.
4. *Previous treatments.* The prognosis for treatment is significantly worse if an unsuccessful attempt has been made previously (Knottenbelt & Walker 1994). Repeated failures make the prognosis correspondingly poorer. Therefore, the best available option with the highest chance of success should always be used at the first attempt at treatment (Genetsky et al 1983).
5. *Prognosis (likelihood of a successful outcome to the selected treatment method).* In some cases the lesion is presented in an impossible state by virtue of its location, type, severity or extent. The previous history may also have a major influence on the prognosis. Delays to diagnosis and between biopsy and treatment are an unfortunate consequence of the perception that the disease is a benign, cosmetic skin blemish. Many cases reach an untreatable extent or severity between biopsy and the decision to treat the lesion.
6. *Results of biopsy.* Whilst biopsy is sometimes required, this can be harmful, especially when there is a delay between biopsy and treatment. There is, however, much debate about the tendency to exacerbation following biopsy. Prolonged delay may make treatment difficult or impossible or at the very least makes the prognosis worse.

   Incorrect results, particularly when sarcoid is not diagnosed, are potentially disastrous. False positives are rare as the histological features of the condition are easily recognized from biopsy specimens. There are, however, some occult or verrucose lesions and some mixtures of granulation tissue and sarcoid which are particularly difficult and require the opinion of an experienced pathologist. Some pathologists are reluctant to make the diagnosis unless there are characteristic epithelial changes but this cannot be achieved when the lesions are severely ulcerated and very large. Given that the epithelial changes are probably secondary it should be possible to define the condition in terms of the primary fibroblastic changes – even when there are few transformed cells present; this will rely of course on a suitable biopsy specimen.
7. *Complication* through coexistence of other factors such as granulation tissue, infection, myiasis or other neoplasia may alter both the histological and clinical appearance and may be misleading.
8. *Facilities and practicality* of the available treatment option. The best option may be economically or practically impossible. For example, radiation carries a generally excellent prognosis for the treated lesion but the size of lesion is a significant limitation and the process also requires special facilities and a considerable financial investment. Extensive cryosurgery of multiple lesions may require prolonged general anaesthesia; use of a full three-cycle controlled 'freeze' will involve major time input where many lesions are present. The size of the lesion will also be significant here. Cisplatin is not currently widely available and AW4-LUDES cream is not generally available outside the United Kingdom (and is severely restricted even there). Some treatments have important limitations to use, for example, potentially dangerous drugs such as cisplatin and radiation will necessarily require specialist facilities.
9. *Owner compliance.* The complications of all the sensible treatment options mean that the treatment is likely to be expensive and during treatment the horse may have to be rested or may require special management, e.g. fly control, housing and regular veterinary attention.
10. *Patient compliance.* All treatments will require some degree of patient compliance also – horses that are impossible to handle cannot be expected to respond normally to treatments requiring repeated applications. The temperament of the horse will make a real difference to the possible treatment choices. Repeated topical applications to an unbroken and potentially dangerous horse will inevitably mean that the accuracy and repeatability will be adversely affected. A horse that does not accept confinement will therefore probably not tolerate radiation brachytherapy. Surgical options will often require some form of anaesthesia and this again may prove to be difficult, dangerous or impractical. An old horse may not be a suitable candidate for a general anaesthetic and so surgery either might be precluded or might be restricted by having to operate in the standing horse.

11 *The value of the horse.* The relative value of the horse and the selected treatment is a common limitation. The purely commercial value of a horse may mean that it is more expensive to treat it than buy another horse but in most circumstances this is not a major consideration. Some owners will wish the horse to be rid of sarcoids so that it can be sold as 'unaffected' but almost all treatments result in some detectable changes and early lesions may be identified by an astute and careful clinician.

12 *The possibility of a contagious nature.* There is little doubt that the condition can spread on the individual horse; the mechanism by which this occurs is unknown but flies appear to play a role. Furthermore, the possibility of transmission to other horses in the group is important when considering treatment. The means for this are also uncertain but flies are possibly involved again, possibly by the same general process. The concept of contagion is often used as a trigger to 'treat' a particular horse – owners of other in-contact horses are often aware of the risks of co-grazing, especially in summer months, and so pressure is brought to bear on the owner to 'treat or remove' the horse.

Treatment methods

Many treatment methods including conventional and non-conventional approaches have been used with varying reported success (Table 18.5). Where there is a multitude of

Table 18.5 Relative value of treatment methods in the author's experience for the various forms of equine sarcoid

Treatment	Type of sarcoid				
	Occult	Verrucose	Nodular	Fibroblastic	Mixed
Surgical					
Ligation	N/A	N/A	***	#	#
Excision	***	*	***	*	*
Cryosurgery	**	**	N/A	*	*
Hyperthermia	**	*	N/A	*	N/A
Electrocautery	**	**	***	*	*
CO_2-YAG laser	***	**	***	*	*
Photodynamic	**	**	NA	*	*
Topical					
(AW4-LUDES)	****	***	**	***	**
Podophyllin	*	#	N/A	#	#
5-Fluorouracil	***	***	*	*	NA
Tazarotene	***	**	N/A	NA	NA
Imiquimod	**	**	N/A	*	*
Sanguinaria/ZnCl[a]	**	**	N/A	*	*
Intralesional injection/cytotoxic/antimitotic					
5-Fluorouracil	NA	NA	**	**	*
Cisplatin	N/A	N/A	***	***	N/A
Immune methods					
Autogenous vaccines	#	#	#	#	#
BCG	N/A	N/A	****[b]	*** #	#
Radiation					
Brachytherapy	N/A	N/A	*****	*****	***
Teletherapy	*****	*****	*****	*****	*****

*****, Expected results over 80–90% success; ****, expected 60–80% success; ***, 40–60% success; **, 20–40% success; *, <20% success.
N/A, not appropriate modality.
#, liable to be worse.
[a]*Sanguinaria canadensis* and zinc chloride marketed as XXTERRA and SARC-OFF.
[b]BCG therapy has a good reputation in the treatment of nodular lesions around the eye but a poor one elsewhere. The results are particularly poor on limb or body lesions.

different treatments, the implication is clear – either they have severe restrictions or none is universally effective. In the case of the sarcoid both of these factors are important. Only those treatments that are documented and reported to have a benefit and those that the author has personal experience of are considered here. In fact, there are few large case series that involve properly conducted clinical trials and so the concept of evidence-based medicine cannot really be applied. Instead, there are many small series testing single treatment methods without control procedures. In addition, there are individual hospitals or practices where large numbers of cases have been treated and where experience is available for either treatment or advice.

Although isolated small lesions may respond well to surgical removal, incomplete excision (or even the use of biopsy punches) can precipitate rapid fibroblastic proliferation which can be difficult if not impossible to treat successfully. The general principles of the available treatment methods are described in Chapter 4.

The various treatment methods described below may be more or less applicable to specific types of sarcoid. Currently there is no effective treatment regimen for the malevolent form of the disease – treatment is invariably palliative only.

Benign neglect

Benign neglect is a realistic option in some cases where either the lesions are so small as to make the cost and procedure of treatment seem disproportionate or so extensive as to make any method impossible. For the former cases this option should always be viewed with some caution as many cases become progressively worse. The option of benign neglect may be suitable for the affected horse(s) but it does of course mean that the existing sarcoids could be a problem in terms of spread to other sites on the horse or (to a far lesser degree admittedly) to other horses.

Regular professional reassessments with the aid of a diagrammatic 'sketch map' of the horse with accurate recording of type, size/extent and location is advisable because the examination intervals may be too long to accurately recall the true extent and therefore give a false idea of the progression. A photographic record is very useful and of course, very accurate. The record should enable another veterinarian who might attend the case in the future to make a sensible judgement on the rate of progression.

Careful owner counselling is also an important aspect of this option; in the event that the owner recognizes deterioration, then of course immediate veterinary attention should be sought. Owners will also benefit from careful advice on fly control, wound management and the need for regular reassessments.

For the most part the option of benign neglect should be carefully considered and used only when the case can be monitored for progression or when a proposed treatment method/interference cannot expect to resolve the problem at all. Palliative treatment of the equine sarcoid is usually counterproductive.

Surgical methods

Ligation This is limited to small nodular lesions (type A1 and type B1) or fibroblastic lesions with a well-defined 'neck' (type 1a, pedunculated, see p. 119).

A convenient method for this is the use of lamb castration/elastration bands. These are cheap, effective and convenient but are limited in size **(Fig. CD18 • 16)**.

Ligation with plastic 'ties' or with suture material is far less effective and tends to cut rather than squeeze and so recurrence rates are far higher. Quite often it is helpful to place several of the bands around the base of the lesion – each being applied closer to the body of the horse than the preceding one. In this way, extra tissue is incorporated and the risks of recurrence are correspondingly less.

Occult, verrucose, sessile fibroblastic (type 2), invasive nodular (types A2 and B2), mixed sarcoids and malignant sarcoids are usually not amenable to this treatment. Results with type A1 and type B1 nodular lesions in the groin in particular are satisfactory.

Partial ligation of a large lesion is a high-risk strategy unless there is a definite plan for treatment of the remaining sarcoid tissue immediately following the sloughing of the necrotic part. This is more likely, however, to result in exacerbation and a reduced prognosis unless the second phase is instigated immediately and treatment is persistent thereafter (see Fig. 4.19).

Surgical excision Although this is a frequently practised option, there is a high rate of recurrence in all except the most confined and defined lesions (Jackson 1936). Recurrence rates can be over 70% within 6 months (Diehl et al 1987, Goodrich et al 1998) and often the recurrences are more aggressive and in larger numbers.

In many circumstance this is, however, the only practical option available and then it is important that the best possible technique is applied to maximize the benefit. Wide excision carries the best prognosis where surgery is used. Nonetheless, it is wise always to give a very guarded prognosis when surgery is used.

Superficial (occult and verrucose) lesions can be effectively treated by wide excision provided that the wound can be closed and then protected during complete healing. Any delay in healing may be due to sarcoid regrowth. Complete or partial failure of the wound to heal is early evidence that there is residual tissue in the wound site. Wound breakdown within days of surgery is a common immediate indicator of failure to excise all the sarcoid tissue (Ragland et al 1970) but sarcoid regrowth can take up to 5 or more years to recur at the site. Rapid development of fibroblastic sarcoid commonly follows and indeed removal of a single lesion may be followed by development of several new sarcoids at the site – this possibly reflects that the tumour was not completely excised and/or that the wound site was seeded with desquamated transformed (tumorous) fibroblasts (Brostrom 1995) (Fig. 18.36). It is wise to protect the sarcoid lesion with plastic adhesive dressings or a swab over the main lesion prior to starting the surgery.

Notwithstanding the limitations of surgery, excision of nodular lesions carries a somewhat better prognosis provided that the lesion is not attached to the skin and provided that the operative site is not contaminated with sarcoid tissue released by cutting into the lesion itself. Where these criteria can be met, the prognosis is usually quite good. Nodular lesions in the eyelids, however, are potentially very dangerous as they commonly have extensive ramifications through adjacent tissues and often extend into the muscles of the eyelids (Knottenbelt & Kelly 2000). Surgery almost always fails in these sooner or later **(Fig. CD18 • 17A and B)** but successes are obtained.

Type 1b (pedunculated/dumbbell) and type 2 (sessile) fibroblastic sarcoids, and mixed and malevolent sarcoids are not generally suitable for surgical excision alone. The



Figure 18.36 A type B sarcoid was removed from this caudal thigh some 14 weeks previously. The wound healed well initially but then it became obvious that several new lesions were developing in the site. Surgical pathology of the original mass and biopsy of the new lesions confirmed both were sarcoid. The reason for recurrence of this type may be the seeding of the operative site with cells from the original lesion or may be due to incomplete excision.

prognosis following surgery can be improved somewhat by combining it with other modalities such as cryosurgery, cautery, hyperthermia, topical cytotoxic compounds, intralesional cisplatin injections or radiation.

Should recurrence occur, an alternative method should be used as quickly as possible. Combination methods are usually the best option if there is recurrence.

Cryosurgery Cryosurgery is commonly employed (Fretz & Barber 1987) but suffers from the same limitations as sharp excision. On its own, it has a relatively poor success rate except in the smallest and most defined lesions which carry a reasonable success rate provided that the procedure is controlled and correct. The margins of the lesion need to be defined and thermocouples placed so that the whole lesion is destroyed without unnecessary damage to underlying or adjacent tissues. Furthermore, it is particularly difficult to apply in a controlled fashion to large areas and infiltrative lesions (see p. 123).

The selection of the site is also important as cryosurgery will inevitably result in extensive tissue necrosis. Local scarring may then have important effects on function. It is not an appropriate method for tumours in and on the eyelid in particular.

Aggressive cryosurgery involving the crude application of liquid nitrogen is not an acceptable method. There is uncontrolled tissue destruction and often this fails to resolve the larger lesions in any case.

The reported benefit from cryoantigens released into the bloodstream, which results in a resolution of lesions remote from the treated one (Lane 1977, Steiner 1988), has not subsequently been substantiated. However, reports of up to 100% success have been made, but this is likely to be the result of a combination of careful case selection and meticulous procedure.

Combination therapy (usually with surgical debulking prior to cryonecrosis) may carry a better prognosis but the freezing process following surgical debulking may do little more than destroy seeded cells in the wound bed. Heavy bleeding complicates cryo-destruction with a general loss of efficacy.

In some parts of the world where there are limited resources, combinations of sharp surgical excision and hyperthermia or actual cautery of the resulting wound bed appears to carry a better prognosis that simple surgical excision. This is likely due to the destruction of 'seeded cells' in the wound bed in a similar fashion to the use of cryosurgery following surgical removal.

Hyperthermia/radiofrequency hyperthermia (See p. 121.) Hyperthermia relies upon the extra sensitivity of tumour cells to temperature rises and the method is reported to produce some good results, albeit in a very few cases (Hoffman et al 1983). Repeated procedures are required and the extent of the tumour has to be defined so that every sarcoid cell can be effectively treated. Tissue is heated to 50°C for 30 seconds and the procedure repeated weekly for up to 5 weeks. The method has not gained general acceptance. High-frequency therapeutic ultrasound has a similar effect in raising the temperature of tumour masses but there are no reports of its application in sarcoids.

Electrocautery Surgical electrocautery has advantages in that the margin of the lesion is effectively cauterized and so is extended beyond the incisional line. Furthermore, bleeding from the margins of the wound is minimal. However, its efficiency depends on the extent of the tumour and its locality. Scarring can be limited if primary closure of the wound is possible. It is one of the few possibilities for treatment of pinnal sarcoids (especially those on the skin of the inside of the ear). In this site there are particular difficulties with treatment such as the lack of any subcutaneous or supportive connective tissue or muscle and the close proximity of the lesions to major blood vessels in the ear flap. The practical options are extremely limited and this method does seem to work in a reasonable number of cases.

Laser surgery (CO_2-YAG laser/diode laser excision) (See p. 121.) Laser excision is reported to have a higher success rate than sharp surgery; up to 80% of treated cases may be successful (Diehl et al 1987). Again, selection of the most appropriate lesions is very important. Laser surgery may be even more effective in donkeys and mules than in horses but it is likely that the nature of the lesion and the location are major factors in the prognosis. Typically the prognosis will depend on careful case selection **(Fig. CD18 • 18A–D)**.

The major benefit appears to lie in the bloodless field of surgery and the low rate of recurrence may reflect the destruction of marginal cells which might be left viable by surgery or cryosurgery. Postoperative swelling and pain are minimal and primary closure may be possible. Where a larger defect results the wound site can be covered using a biological matrix material such as ACell (ACell Ltd, USA), or BioSys vet (VetBioSys, UK). Wound contraction, epithelialization and granulation are all slower following laser surgery than sharp surgical excision.

Laser surgery is often the only effective alternative to diathermy for treating localized lesions on the pinna – especially where the lesion is on the medial wall. Non-rooted, pedunculated (type 1a) fibroblastic eyelid tumours can also be successfully treated in this way.

CO_2 laser surgical equipment can be expensive initially and to maintain, but the development of diode lasers has made this less of a consideration. Special safety precautions are required when lasers are used – these include eye

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protection, shielding all reflective surfaces and careful use of the laser pulses.

Local/topical medication with cytotoxic or antimitotic compounds/chemotherapy

Topical chemotherapeutic agents including cytotoxic and antimitotic compounds such as 5-fluorouracil or cisplatin can be used effectively. They are limited, however, by their overall efficacy and the need to sustain medication over prolonged periods. However, when topically applied or injected into lesions they seem to have little or no untoward systemic effects.

Systemic cytotoxic or antimitotic compounds are probably irrelevant at this time.

Heavy-metal compounds (inorganic arsenic/antimony/mercury salts) These induce extensive tissue necrosis and scarring. Combinations of these compounds can limit the toxicity of each individual one but still maintain the efficacy. They are easy to apply and some complex mixtures of these with antimitotic, corticosteroid and cytotoxic drugs have a reasonable reputation (e.g. AW4-LUDES – see below) (Knottenbelt & Walker 1994).

5-Fluorouracil This can be applied as a 5% ointment under a bandage (Roberts 1970) or topically on a twice daily basis for some weeks. It can be successful on single, small occult or verrucose sarcoids and is also a useful material for controlling large areas of verrucose or occult sarcoid which cannot be effectively treated by any other means. Scarring is usually minimal, although the treated area can show marked reaction as cells are destroyed over a wide area of the surface of the skin.

AW4-LUDES The experimental substance AW4-LUDES also contains 5-fluorouracil with heavy metals and natural oils and the combination is an aggressive treatment for certain types of sarcoid, in defined locations under specific conditions (Fig. 18.37). Several different 'strengths' are used in a planned protocol based on the author's experience of many thousands of cases. The results are encouraging overall given the simplicity and economics of the material (Knottenbelt & Walker 1994, Knottenbelt 2006) **(Fig. CD18 • 19)** (see also Chapter 4, p. 111). It is, however, strictly limited in availability.

Podophyllin There are few reports of the use of this material, which is used to treat human viral papilloma. Disappointing and unreliable results are reported anecdotally, with a few good reports. The material needs to be applied at least daily and some cases may not respond at all while others may be exacerbated.

Imiquimod (Aldara 3M) This material is used to treat human genital warts and is said to be an immune response modifier with potent antiviral and anti-tumour properties. A preliminary study involving 19 cases resulted in a detectable and worthwhile benefit. Eighty per cent of lesions showed a 60–75% improvement (Nogueira et al 2006). The material is applied to the surface of the tumour three times a week for between 16 and 32 weeks. The therapeutic benefit was slow to develop and there was alopecia, inflammation and depigmentation in the treatment site. It is not clear which of the major properties brings the benefit. There are reports of serious human side-effects but none of these has so far been reported in horses.



Figure 18.37 **(A)** This highly invasive type 2 (sessile) fibroblastic axillary lesion was treated with repeated courses of AW4-LUDES over a period of 12 months. **(B)** The outcome was very encouraging although there was a small recurrence after 2 years. This site is always problematic no matter what treatment is selected.

Silver nitrate (caustic pencil) application This is a rather old-fashioned method that relies upon the direct tissue destructive effects of silver nitrate. It is applied by means of a 'styptic/caustic' pencil to very small localized lesions only (see p. 111).

Formalin-based gels (Lotagen, Hoescht) This is a derivative of the human wart treatment and is only applicable to very few small, superficial sarcoids. There are no reports of clinical trials but some anecdotal reports of successes.

***Sanguinaria canadensis* (blood root) extract (with zinc chloride) (XXTERRA/Indian mud/SARC-OFF)** The advertising literature for this material (available in the USA as a prescription-only material) states that it alters local immunity and enables the immunological recognition of tumour cells. There is no reported control study for it, but there are reports of successes (and failures).

Intralesional antimitotic/cytotoxic chemicals

Bleomycin Intralesional injection of 1.5 mg bleomycin (Blenoxane, Bristol Meyers Oncology, USA) repeated every 5 to 7 weeks has been reported to be an effective regimen for small localized tumours (Theon 1998). However, other attempts to use the same regimen have failed to show any material advantage over other intralesional and topical compounds (Theon 1998).

Cisplatin (*cis*-diaminedichloro-platinum; DDP) (Platinol, Bristol Myers Oncology, USA; Cisplatin Injection, David

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Bull Laboratories, Warwick, UK and Mulgrave, Victoria, Australia). Good results are reported for small fibroblastic and nodular lesions in particular but it requires repeated injection into the site of the lesion (Theon et al 1993). In some cases it can be used in conjunction with surgical debulking to improve the overall outcome. Cisplatin has a broad spectrum effect against epithelial and fibroblastic (mesenchymal) tumours and this is used to treat sarcoid tumours (see p. 108).

Emulsions made up from equal volumes of an aqueous solution of cisplatin/mL and either sesame or almond oil may be used to provide a longer effect from each injection. A dose of 1 mg cisplatin per cubic centimetre of tumour is suggested but this can be very difficult to inject into the denser forms of sarcoid. The best results are achieved when a more concentrated form of cisplatin is used in a stable emulsion or wax – in this way the volume of injectate is reduced dramatically and so the procedure becomes far easier. In all cases the injections are repeated at 2-week intervals for four or five procedures. Slow-release beads and sponges provide controlled local release and it is likely that these systems will mean that better results are obtained in a safer and more controlled fashion using less chemical. During the treatment period all faeces and urine should be disposed of safely.

Reported results are very encouraging in spite of the logistic difficulties. In the reported protocols parallel 'columns' of the emulsion are injected through several planes of the sarcoid. This is extremely difficult to control in solid tumours and leakage of uncontrolled volumes usually occurs.

Results indicate that up to 93% of sarcoid tumours less than 5 cm diameter are resolved by this method. Tumours larger than 5 cm may require surgical debulking prior to injection and when this is performed the results are reported to be better than simple wide surgical excision – indeed results in difficult sites are said to be up to 100% successful (Theon 1998).

Intralesional cisplatin infiltration can be significantly enhanced by the procedure of electro-chemotherapy (see p. 125); this method relies upon the increased tissue permeability arising from direct application across the site of an electrical current. Although there are no reports of other chemicals being used in this way, it is quite likely that some improvement will be achieved. It is logistically more difficult in requiring general anaesthesia and specialized equipment.

These methods are certainly worth exploring, although they are probably not applicable to verrucose or occult lesions. The logistics of the procedure are probably the biggest limitation to its use but there appear to be no significant contraindications in horses – even pregnant horses are said to be unaffected. However, the material is not universally available and it certainly carries significant dangers for the operators and the handlers of the horse following the procedures. It is likely to be restricted to specialist oncology treatment centres.

5-Fluorouracil injections A solution containing 25 mg/mL of 5-fluorouracil (Oncotaine, DBL, Mayne Pharma Plc, UK) is used for intravenous injection in some serious human neoplastic diseases. Repeated intralesional infiltration of a sarcoid lesion with this material can have significant benefit. However, there are no reported control studies. The best doses and frequency of injections are not yet clear. Slow-release biodegradable sponges and beads are being developed and these systems will provide better controlled release of the chemicals so less drug is needed and better results can be expected. Again there are no large series involving these materials (see p. 112).

Photodynamic therapy

This method relies upon defined wavelength light to an area that has been pretreated with a photodynamic agent. The agent can be applied topically and then the light source is applied to that area or the agents can be administered parenterally and the lesion then exposed to the required wavelength of light. Results are not widely reported but the technique is being used with increasing frequency in other species for other tumour types. The only study described so far is the use of hypericin. Ultimately it is likely that the agent can be delivered to the tissues in an antibody-bound form and so the procedure would specifically target the tumour cells (see p. 126).

Immunological methods

Treatment attempts with 'vaccinations' have been undertaken by several research workers and are still performed by some practitioners. Bovine 'wart vaccine' has been used and was found valueless. It may indeed make the condition worse (Knottenbelt et al 1995). Pox vaccines administered intralesionally are also unsuccessful. The studies involving 'vaccination' have been consistently disappointing and the general consensus is that they cannot be supported at the present time (Scott & Miller 2003). Vaccination with bovine or equine papillomavirus vaccines has been ineffective (Ragland 1970, Roberts 1970). New vaccine technology using chimeric virus-like particles is under development.

Vaccine (autogenous) The use of autogenous vaccines in treatment of the equine sarcoid is probably contraindicated. The method is based on the assumption that there is a 'viral' aetiology and that seroconversion to the virus will induce a curative immune response. The results of this simple treatment suggest that this is not the case and if it worked it would surely have become universal practice. Many sarcoid cases which are treated with an autogenous vaccine are worse afterwards, with some cases developing several thousand lesions. However, the few cases that do resolve spontaneously suggest that there is some 'immunological' response available but autogenous vaccines do not appear to be the most appropriate way of inducing this.

Immunomodulation Immunomodulating proteins including various types of protein cell-wall fractions derived from bacillus Calmette–Guérin (BCG) or other mycobacteria, or live, freeze-dried (lyophilized) BCG have been used widely (Rebhun 1987). Other proteins and 'stimulating' materials have also been used.

BCG (and related protein derivatives) The reconstituted protein material or lyophilized live bacteria is injected intralesionally at repeated intervals (see Chapter 4, p. 104). The technique carries a fair to good prognosis for treatment of nodular and some fibroblastic lesions in and around the eyelids in particular (Knottenbelt & Kelly 2000). The prognosis seems to be worse away from this area and some fibroblastic lesions on the limbs appear to be aggravated rather than treated and in fact become worse. The method is probably not appropriate for mixed, verrucose or occult lesions because the diffuse nature of the tumours precludes effective intralesional injection. There is an ill-defined (slight but definite) risk of an anaphylactic reaction that is significantly

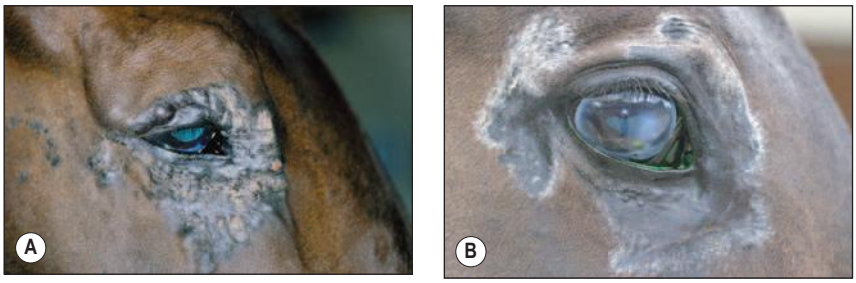


Figure 18.38 (A) This particularly aggressive (almost malignant) mixed sarcoid was treated using interstitial radiation (iridium-192) brachytherapy. **(B)** The results were very pleasing but there were secondary effects on the cornea and a small medial cortical cataract developed after 8 years.

higher with repeated injections and highest if a repeat course is carried out after some months/years.

Non-specific immunostimulation A crude extract of *Propionibacterium acnes* (Eqstim, Neogen, USA) is marketed in the USA as a safe but potent stimulator of cell-mediated immunity. Although there is no data sheet indication for sarcoid treatment, it has been used both by intramuscular injection and by intralesional injection. There are anecdotal reports of successes and failures but it is suggested that this is an inappropriate method for treatment of equine sarcoids (Studer et al 1997). A similar, non-specific oral product known as Equimune is advertised in the USA as a means of stimulating immunity. The suggestion is that this results in an immunological 'restoration' and the ability to specifically treat a wide variety of medical disorders. However, until there are proper controlled, double-blinded clinical trials suggesting genuine evidence of efficacy, these products probably have no indication in the management of sarcoid tumours.

Interferon and interleukin Trials with high-dose systemic and intralesional interferon and interleukins in the treatment of sarcoid are being carried out in several centres. The preliminary results are disappointing but the approach may be found to be helpful in some particular early cases.

Radiation methods

Radiation is by far the most effective method of treatment but has severe limitations of availability and cost. Radiation (brachy- or tele-) therapy using gamma radiation carries a very good prognosis within the limits of the technology (Turrel et al 1985). It is usually limited by cost and availability as well as the need to consider operator safety. It is therefore usually restricted to smaller lesions in areas for which no other method is suitable, such as the eyelids and over joints (see Fig. 4.17) The value of beta radiation delivered to superficial lesions is doubtful in view of its poor penetration through tissues. It is therefore not applicable to larger tumours with extensive tissue involvement (see Chapter 4, p. 117).

Typically for any radiation treatment the beneficial responses are slow to appear and it may take up to 14 months, for example, in cases treated with iridium-192 linear interstitial brachytherapy. In the interim the treated site often develops purulent discharging sinus tracts or moderate to severe inflammation. Some cases, however, respond faster and without significant superficial necrosis. The outcome is cosmetically excellent in most cases but as might be expected there is a degree of scarring, some pigmentary loss and frequently alopecia **(Fig. CD18 • 20A and B)**. Radiation does not discriminate between neoplastic cells and normal cells so some collateral damage is likely and this can be significant; for example, the cornea may become cloudy, cataract may develop and even underlying bone may sequestrate (Fig. 18.38).

General comments

Particular problems are encountered when treating sarcoids at difficult sites such as the eyelids, face, lips, coronary band and over joints and other synovial structures. Notwithstanding the risks of surgery, it is sometimes the only viable option. For example, topical applications on the medial thigh can be highly problematic with possible contact with the unaffected adjacent limb and belly wall. Axillary lesions are often particularly difficult with slow healing and a high rate of recurrence. There is also a significantly increased difficulty with sarcoid transformation at wound sites.

In all cases, whether at a difficult site or not and whether complicated by the extent and type of the lesion(s), the best possible treatment *must* be applied on the first occasion. Failures make the likelihood of future success much less and progressive difficulty is likely with repeated failures of treatment. The choice of treatment remains a clinical decision that should be taken only after all the aspects of the condition are considered. Specialist advice is often available from referral centres and equine dermatologists.

Prognosis

The prognosis for any sarcoid must be guarded. There is a strong tendency both from the veterinary profession and owners to belittle the disease through the terms 'wart' and 'angleberry'. These imply some sort of benign nature akin to normal viral papillomas on cattle and horses but there is little predictable about the equine sarcoid and it is unwise to ignore it.

The prognosis is significantly reduced with each sequential failure to resolve the lesions and this applies to other lesions on the horse that are not subjected to treatment at the time.

It is highly likely that recurrences will occur at treatment sites at any time between days and 20–30 years after treatment and that new lesions will develop in any (genetically) susceptible horse. The only cases that appear to be unlikely to develop any more lesions are those that spontaneously resolve without any treatment. The same does not, however, apply to horses that are treated to the point of having no apparent lesions at all.

Fibroma and fibrosarcoma

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Profile

An uncommon tumour arising from dermal or subcutaneous fibroblasts. Older horses are more liable to be affected with predilection sites on the head, legs, neck and flanks.

Key points: Fibroma and fibrosarcoma

4 9 14 19 20 23 24

1. A rare tumour occurring on the body or the foot in particular that is clinically and pathologically very similar to sarcoid although some pathologists are certain that the two can be meaningfully differentiated.
2. Solar fibroma is more distinctively 'fibroma' but can be confused with chronic inflammatory reactions in the tissues of the frog and sole as a result of injury or foreign body penetration. A fleshy, ulcerated, exophytic, fibroblastic mass on the body trunk is the commonest presentation.
3. Diagnosis is correspondingly difficult – most, including spindle cell sarcoma, myxofibroma, neurofibroma and fibrosarcoma, will in fact be 'sarcoids'.
4. Treatment options are similar to sarcoid although, in theory at least, surgery probably has a better chance of success than in sarcoid. Radiation still carries the best prognosis.
5. Prognosis for genuine fibromas is probably somewhat better than for sarcoid. Metastases are seldom reported.



Figure 18.39 A genuine fibrosarcoma in the girth region with a typically ulcerated, cauliflower appearance.

Lesions are usually solitary well-defined nodular subcutaneous masses that range in size from 3 to 25 cm in diameter.

Fibroma of the sole is one area where the diagnosis can probably be justified in most cases. It is one of the commonest neoplasms encountered in the frog of the horse's foot (see Fig. 18.67). However, it is also possible to confuse this form with dense, granulating frog fibrous tissue at the site of a wound or chronic foreign body reactions.

The terms myxofibroma and fibromyxoma are also applied to some tumours with focal areas of mucinous degeneration but again they are clinically indistinguishable from sarcoid.

Fibrosarcoma is very rare and represents the malignant form of the fibroma – a high proportion of these cases will in fact be aggressive fibroblastic or malignant sarcoid.

Clinical signs

Often single, firm or soft, well-circumscribed, dermal or subcutaneous nodules present which may ulcerate or develop into a flattened, verrucose or fleshy, cauliflower-like lesion especially when the fibrosarcoma form develops (Fig. 18.39). The surface may be hyperkeratotic in the early stages.

The large majority of tumours that are classified as 'fibroma' or 'fibroma durum' are, in fact clinically typical of nodular sarcoids and the differentiation of the two is probably unjustified on clinical or therapeutic grounds.

A particular form of fibroma occurs on the sole of the foot (see p. 435).

Differential diagnosis

- Sarcoid (nodular, verrucose, fibroblastic or mixed depending on type and stage): most are indistinguishable from sarcoid; pathological differentiation may be justified in a few cases.
- Exuberant granulation tissue: history of a non-healing wound and chronic low-grade inflammation. Typical histology.
- Foreign body reaction: ultrasonographic identification of a foreign body and histological evidence of foreign body reactions such as giant cells.
- Lymphoma/lymphosarcoma: multiple nodular masses possibly with multicentric involvement and even leukaemia in a few cases. Single lymphoma nodules are much less aggressive and may progress very slowly. Typical histology.
- Other tumours (including keratoma of the foot): typical clinical features and histology.

Diagnostic confirmation

- Histological section of biopsy or whole tumour is essential for diagnostic confirmation.

Treatment

'Total' surgical excision or cryotherapy of the invasive forms of fibrosarcoma carries a poor success rate – possibly because most of these cases are in fact sarcoids. Excision of fibroma durum lesions is feasible with the same caveats as for nodular sarcoid. The best results are obtained by radiation (teletherapy) but this is not widely available as an option. Repeated surgical procedures or laser surgery can improve the success rates.

Prognosis

Most genuine fibromas are benign and remain localized. Fibrosarcomas may be multiple and are locally invasive but normally have little tendency to metastasize.

Neurofibroma (schwannoma, neurolemmoma, neurinoma, nerve sheath tumour/perineural fibroblastoma)

Profile

These are rare fibrous tumours arising directly from nerve sheaths which can occur anywhere associated with nerve tissue in horses of any age and type. The most common sites recorded include the upper and lower eyelids. Corded nodules extending from the parent tumour have been described (Pascoe 1988).

The relationship between these masses and nodular sarcoids is uncertain, but it seems likely that although the two tumours are clinically similar in many respects they are

Key points: Neurofibroma

1. Rare, fibrous tumour derived from nerve sheaths occurring in horses of all ages and types.
2. Clinically very similar to nodular forms of sarcoid (possibly coexist) and occur predominately around the eye as large or smaller multilobulated subcutaneous masses. Some cases ulcerate.
3. Slow growth rate and multilobulated nature make surgery an attractive option but this can be very dangerous and recurrences are more the norm than the exception.
4. Treatment options are similar to sarcoid with radiation and BCG immunotherapy being the best options.
5. Prognosis is very guarded; unless all the seeded tumours can be removed it will recur. It does not metastasize.

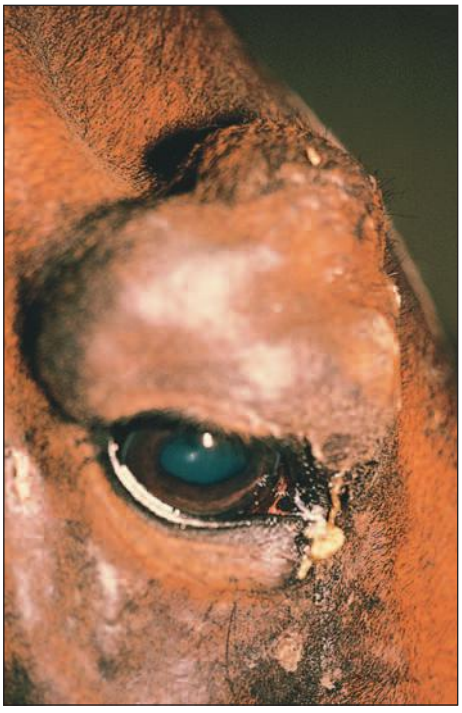


Figure 18.40 Neurofibroma of the upper eyelid with multiple nodules of varying size. Eyelid distortion resulted in a purulent ocular discharge and intermittent corneal ulceration.

pathologically distinct. It does seem that sarcoids may have some neurofibroma-like inclusions that serve to confuse the diagnosis. It may be that these tumours have less effect on the overlying keratinocytes, so tend to mimic type A (1 or 2) nodular sarcoids.

Clinical signs

Small 1–10 mm hard *subcutaneous* nodules and subsequently very much larger multiloculated masses (>25 mm diameter) develop (Fig. 18.40). Multiple and cording lesions may be seen and larger lesions in particular cause erosion through overlying skin and develop into granulomatous-type lesions very similar in appearance and behaviour to fibroblastic sarcoid. However, they rarely show overlying hair loss until ulceration occurs.

Differential diagnosis

- Sarcoid (nodular and malevolent types): some pathologists consider the neurofibroma to be a variant of nodular sarcoid found in the eyelids; tend to involve the skin more often with verrucose or occult changes and alopecia.
- Equine cutaneous mastocytoma (mast cell tumour): single nodules without multilobulation is usual; distinctive histology.
- Cutaneous histiocytic and generalized lymphosarcoma: single or multiple nodules in the skin possibly with systemic signs; characteristic histology.
- Cutaneous nodules caused by insect bites and allergic collagen necrosis: seldom conflict with the location of neurofibroma; multiple, small focal lesions often on the back or neck.

Diagnostic confirmation

- Biopsy or excisional histopathology.
- Fine needle aspirates are usually very difficult to obtain owing to the extreme density of the tissues and the paucity of intercellular matrix and seldom provide any definitive help.
- Recurrence is common with almost identical nodular masses in chains.

Treatment

Complete surgical removal carries a good prognosis but is extremely hard to achieve. Seeding nodules are easily overlooked and tumours recur in a different site on the eyelid in approximately 25–50% of cases where surgical excision is attempted.

Intralesional injection of BCG (or similar immunomodulating proteins, see Chapter 4, p. 104) over an extended period can be effective but is far less effective than against nodular sarcoid in the same site.

Repeated intralesional cisplatin or 5-fluorouracil may be effective. Injections of 1 mg cisplatin or 3–5 mg 5-fluorouracil per mL of tumour are required every 1–2 weeks for four to six injections. Resolution may take a considerable time and there are no specific results published for neurofibroma (as opposed to sarcoid).

Gamma radiation brachytherapy using iridium-192 linear brachytherapy sources, or radio-gold-198 pellets or teletherapy is likely to be effective but is very limited in availability.

Prognosis

A very guarded prognosis is justified because complete resolution is almost impossible to achieve with a single surgical intervention. BCG therapy carries about 40% success for periorbital lesions. Radiation is very effective; iridium-192 linear brachytherapy or gold-198 (Wyn-Jones 1979) treatments carry a good prognosis.

Basal cell carcinoma

Profile

This very rare, sporadic, slow-growing, isolated ulcerative tumour is most often found on the face, neck, pectoral regions and trunk of horses of all ages (4–26 years) of both

sexes and all breeds (Slovis et al 2001). Several different histopathological forms are recognized but the rarity of the tumour means that the classifications derived from other species are hard to justify and in any case the variants have no special aspects either clinically, therapeutically or prognostically. The tumour has a very benign nature and there is no established carcinogen.

Key points: Basal cell carcinoma

1. Rare, benign, localized, usually solitary tumour on the face, limbs and neck, derived from the basal epidermal cells.
2. Affects older horses (12–26 years) and is not restricted to non-pigmented skin, so UV light may not be involved as a carcinogen.
3. Lesions are nodular possibly with a roughly circular, ulcerated centre and a piled-up hyperpigmented margin.
4. Sharp or laser surgical excision or cryosurgery is likely to be curative and topical 5-fluorouracil may be a simple and effective approach.
5. Recurrences are not reported.

Clinical signs

This is a solitary, well-demarcated, firm, freely-movable, nodular dermal mass which frequently ulcerates. Tumours vary from 0.5 to 4–5 cm in diameter. The margins of the lesion commonly develop a 'piled-up' raised appearance giving the impression of a crater (Fig. 18.41). Non-ulcerating lesions may be alopecic and have a prominent blood supply.



Figure 18.41 Basal cell carcinoma showing typical 'piled-up' margins and a central ulcerated area.

Differential diagnosis

Non-tumorous conditions

- Foreign body granuloma: single isolated lesions with a slow progression and usually a purulent discharging sinus tract.
- Botryomycosis/pseudomycetoma/pyogranuloma: histologically distinctive and usually at sites of skin injury.
- Habronemiasis: seasonally and geographically restricted; causes skin ulceration and conjunctival and wound site granular accumulations. Larvae can be identified in washings and rapid response to treatment with spontaneous resolution in autumn is significant.

Tumours

- Sarcoid (occult, nodular type B or fibroblastic ulcerated types): usually multiple; prominent hair loss and circular superficial scaling; usually other types present on the horse.
- Fibroma (fibrosarcoma)/neurofibroma: nodular, firm, slow-growing masses with characteristic histological findings.
- Squamous cell carcinoma: destructive or proliferative neoplasm; seldom static and often highly destructive. Commonly restricted to white-skinned areas.
- Melanoma: characteristically black nodular lesion often in multiple forms. Usually restricted to grey horses but not exclusively.
- Other ulcerating tumours.

Diagnostic confirmation

- Biopsy (excisional) is usually justified in small tumours, particularly in view of the similarity with sarcoid and some melanomas (Slovis et al 2001).
- Histopathology will usually reveal a well-defined proliferation of basal cells arranged in an orderly/symmetrical pattern. Cytokeratin staining is positive.

Treatment

Surgical ablation by excision, cryosurgery or laser excision should be curative. Intralesional cisplatin, topical application of 5% 5-fluorouracil or radiation could also be justified in larger tumours and where surgery is difficult.

Prognosis

Good. Seldom if ever metastasizes or recurs at the site of full surgical excision.

Mastocytoma (equine cutaneous mastocytosis/mast cell tumour)

Profile

This rare tumour occurs more frequently in young or middle-aged male horses than in females or older horses. Compared to many other veterinary species mast cell tumours of any type are less common in horses (Tan et al 2007). There is debate about whether the condition is a true neoplasm, and little is known about the condition or its aetiopathogenesis.

Three forms are recognized:

1. *Nodular equine cutaneous mastocytosis*. Solitary cutaneous nodules that are histologically more representative of an accumulation of hyperplastic mast cells than a true neoplasm. Alternatively the masses are referred to as benign mast cell tumours or focal mast cell dysplasia. Some opinions refer to the condition as being a reactive nodular disease with accumulations of mast cells rather than a true neoplasm.
2. *Malignant equine mastocytosis*. Malignant mastocytosis has been reported very rarely in horses (McEntee 1991, Riley et al 1991, Malikides et al 1996). When this

form occurs the cutaneous lesions may be clinically insignificant but provide a good opportunity for diagnosis provided that a careful clinical examination is performed.

3 *Congenital equine cutaneous mastocytosis*. This is possibly the rarest form of the condition and is detected at or soon after birth (Cheville et al 1972) (see p. 237).

Clinical signs

Cutaneous nodular equine cutaneous mastocytoma These arise typically in young to middle-aged horses (1–18 years, mean around 7 years) as solitary, discrete, firm or slightly fluctuant, nodules (2–20 mm diameter) in the skin. The skin surface may be normal, hairless or ulcerated. Multiple lesions can occur and appear to be less responsive to treatment.

Key points: Mastocytoma

1 A rare tumour-like condition of middle-aged horses with a greater prevalence in males and geldings than females.
2 Several different forms occur with the commonest being a nodular cutaneous or subcutaneous mass. Conjunctival and ulcerated forms also occur. A congenital form also occurs and is reasonably distinctive.
3 Diagnosis is usually achieved by biopsy but the definitive description is not well established in horses and some may be reactive tissues rather than neoplasms. Mast cells are attracted to eosinophilic foci so often both coexist.
4 Treatment is usually restricted to surgical excision but the location may make this problematic in some anatomic sites.
5 Prognosis is good provided that the tumour is completely excised.

Males are more often affected than females. The head is more often affected than the body and limbs but all body sites can be affected (Fig. 18.42). A particular form appears to affect the conjunctiva (Fig. 18.42D).

On the limb the condition is more common in close proximity to joints (especially the carpus) and on the limbs the nodules are often firmly bound down **(Fig. CD18 • 21)**.

Most mast cell tumours are between 2 and 20 mm in diameter and usually involve dermal or subcutaneous layers. They rarely attach to underlying tissues but some do ulcerate and reveal the characteristic 'kunkers' (granular, firm, gritty masses in bunches) (Fig. 18.42B).

In some circumstances there may be secondary space-occupying effects such as corneal ulceration from a conjunctival mast cell tumour or restricted movement from a distal limb mastocytoma.

A few cases are reported to be pruritic but seldom painful. A partial or complete capsule can be recognized ultrasonographically (and histopathologically). There is seldom any hair loss or pigmentary changes in the skin but some are ulcerated or alopecic. They seldom if ever metastasize.

Malignant equine cutaneous mastocytosis In the reported cases clinical signs include pruritus at the site or generalized pruritus and lameness/pain associated with various joint and synovial structures (see **Fig. CD18 • 21A–D**).

Pathologically elevated circulating eosinophil counts and hyperfibrinogenaemia with metastasis to local lymph nodes have been reported.

Congenital equine mastocytosis (See p. 237.) There are widespread/disseminated raised ellipsoidal cutaneous nodules along the back and trunk, varying in size from 2–3 mm to 3–4 cm. The larger nodules may have a fluctuant feel and there may be overlying skin ulceration. These may resolve spontaneously but there are too few cases to allow any sensible interpretation of the prognosis.

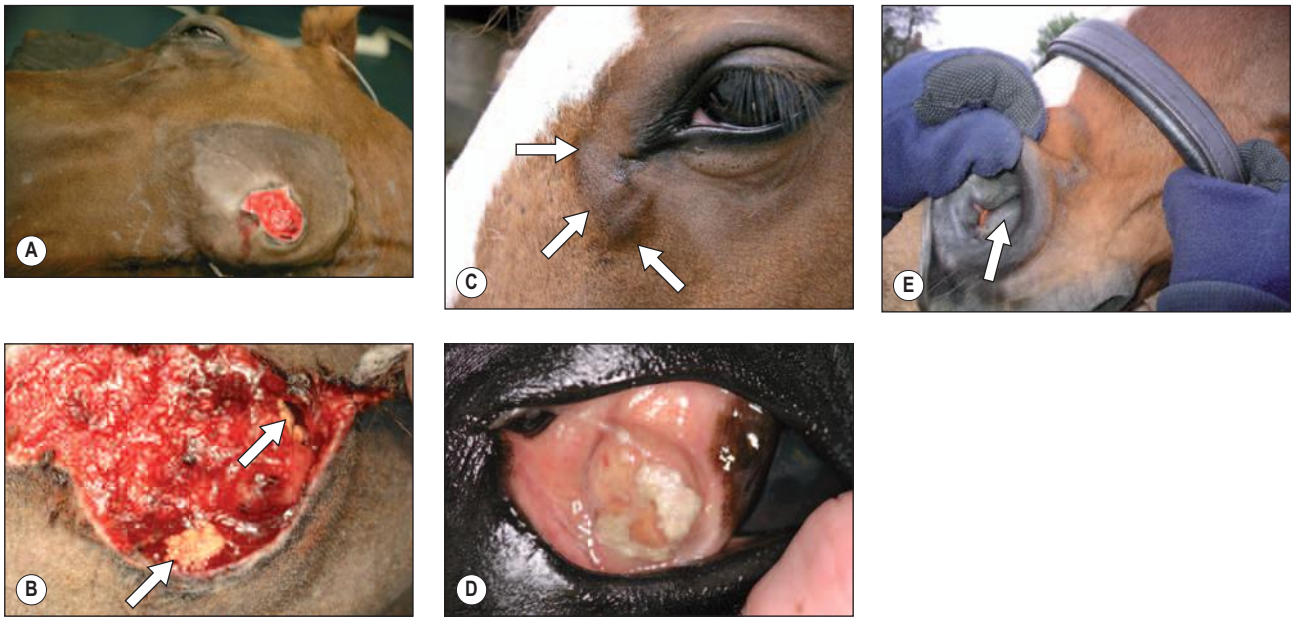


Figure 18.42 Mast cell tumours. These are more often found on the head but can occur almost anywhere on the skin. **(A, B)** This slowly expanding ulcerated mast cell tumour shows the characteristic yellow granules (arrows) **(B)**. **(C)** This mast cell tumour was totally subcutaneous (arrows). **(D)** The conjunctiva of the third eyelid, as in this case, or the bulbar conjunctiva can also be affected. Again there is a characteristic yellow granular material associated with the tumours. **(E)** Mast cell tumours also occur in other sites such as the nostril (arrow).

Differential diagnosis

- Dermoid: single or few, slowly expanding non-inflammatory nodules usually in the dorsal midline of young horses.
- Sarcoid (nodular form especially if centre is necrotic – when it may be softer): can be very similar to mast cell tumours but usually there are several different types and type B nodules affect the overlying skin with alopecia and hyperkeratosis.
- Squamous cell carcinoma: seldom in nodular form, destructive forms are commonest and often associated with non-pigmented skin.
- Habronemiasis (conjunctival): similar yellow kunkers are produced and many conjunctival 'mast cell tumours' may be reactive changes to *Habronema* or other lacrimal parasites.
- Bacterial infection of non-healing wound/granulation tissue.
- Botryomycosis/pseudomycetoma: invariably larger and more inflammatory in nature.
- Fungal granuloma/deep dermal mycosis: definitive histology; these can be very similar but tend to occur in different places and remain small in most cases.
- Eosinophilic/allergic collagenolytic granuloma complex: multiple small shot-like dermal nodules developing in the skin of the dorsum, buttock and neck in particular; some can become very large and tumour-like.
- Insect bite/sting: transient localized swelling at site of a bite; the focus can be seen in the fresh lesion.
- Cutaneous amyloidosis: very rare nodular condition of the skin and subcutis; definitive histologically.

Diagnostic confirmation

- Fine needle aspirate/biopsy or impression smears can help.
  - Giemsa stain reveals heavy eosinophil and mast cell infiltration with metachromic granules.
  - Three distinct forms are recognized and these can make diagnosis more difficult due to their similarity with some other cutaneous nodules.
    - **a** *Initiation and early growth phase*. Proliferation of mast cells in distinctive nests and nodules. This may give an impression of an early reactive mast cell response rather than a true tumour.
    - **b** *Inflammatory phase*. Eosinophilic infiltration with concurrent diffuse collagen necrosis and mineralization (creating aggregates of yellow, gritty material known as 'kunkers'). This response can closely resemble the inflammatory changes accompanying clinical habronemiasis and the very common cutaneous collagenolytic granuloma.
    - **c** *Regression phase.* Fibroplasia and granulomatous inflammation are recognized. This may resemble a post-traumatic scarring state.
- Surgical excision (excisional biopsy) or biopsy and histopathology: focal aggregations of active mast cells, eosinophils, fibrinoid necrosis of collagen and, occasionally, mineralization.

Treatment

The low numbers of cases make the treatments difficult to compare clinically.

Where possible, total, wide surgical removal can be an effective approach (McEntee 1991). There is apparently little risk of any immediate untoward response from handling the tumours or from residual cells within the operative site.

There are anecdotal reports of the use of intralesional sterile water injections – the mast cells are suggested to be highly susceptible to variations in local osmotic pressure and this approach may at least be tried safely. Intralesional injection of distilled water may require two to four treatments at 4–6-day intervals. The author has attempted this on a few occasions with some improvement but no resolution.

Intralesional injection of triamcinolone or methylprednisolone acetate can also be useful.

Oral cimetidine (5 mg/kg q 24 h for 3–6 weeks) has been suggested as a treatment. This has still to be substantiated.

The value of cisplatin is uncertain.

Prognosis

A good prognosis is usual for the benign form of the condition, although where they are situated in difficult sites, some are very difficult to remove fully without damaging underlying tissues. Some lesions undergo spontaneous resolution without apparent reason.

The theoretical dangers associated with surgery are not reported in the horse.

The prognosis for the malignant form is very guarded but there are too few cases to be confident about either the best choice of treatment or the likely outcome from either medical or surgical interventions.

Haemangioma and haemangiosarcoma

Profile

These are uncommon, solitary, spontaneous, cutaneous tumours of vascular endothelial cells. The vascular tumours of the skin of horses include haemangioma, cavernous haemangioma, haemangiosarcoma and capillary haemangioma. Often they are of a mixed pathological character and so the specific diagnosis is more difficult. Most are either congenital or occur in relatively young horses. In the former case they may be presented late, having been overlooked at birth.

The congenital surface form (commonly referred to as a congenital vascular hamartoma) has been recorded in distal limbs of Arabian foals in particular (see p. 237). Most cases are regarded as vascular hamartoma. Lesions that are encountered in older horses, also without breed or sex predilection, may in fact be variations of the congenital haemangioma/vascular hamartoma.

Note

A hamartoma is an abnormal, disorganized but mature accumulation of a well-differentiated (normal) tissue type in a normal location. Hamartoma has been reported most often to affect muscle (muscular hamartoma), blood vessels (vascular hamartoma) and dermal melanocytes (equine cutaneous melanosis). Their behaviour is completely benign and they are probably a result of aberrant growth patterns rather than true neoplasia with a loss of cellular differentiation. Where the abnormal accumulation of a normal tissue type occurs at an ectopic site, the term choristoma is used.

Key points: Haemangioma and haemangiosarcoma

3 9 11 14 20 23 24

1 Uncommon skin tumour of younger horses of all colours and types (congenital form is well recognized (see p. 237). Older horses may be affected. Predilection sites include the distal limbs, axilla, conjunctiva and nasal cavity.
2 Clinical presentation as a highly vascular, easily traumatized, haemorrhagic proliferative mass. Most are single but can be part of disseminated haemangiosarcoma. A more diffuse form (verrucose haemangioma) has a plaque-like appearance with a more diffuse, ill-defined, superficial nature. These are also dark blue-black in colour.
3 Biopsy is distinctive (note that the site will often bleed profusely).
4 Treatment is limited to sharp or laser surgical excision; recurrences are common because the margins are difficult to define and they occur in difficult sites that preclude a wide enough margin.
5 The prognosis is guarded. Provided that the tumour is an isolated one and that it can be removed entirely the outlook should be reasonable but most are very difficult to remove and so in general the prognosis is poor.

Vascular hamartoma and haemangioma (and secondary haemangiosarcoma) can be found in the skin and subcutaneous tissues. The limbs appear to be a predilection site but it can affect the axilla, groin, thorax and rarely eyelids/conjunctiva. The lesions themselves are usually quite distinctive, comprising numerous tortuous, enlarged blood vessels.

A verrucose form (verrucose haemangioma) also occurs from time to time. These have a much less vascular outward appearance and are more often plaque-like; however, they are usually dark blue/black in colour and so they can usually be recognized clinically.

Some cases are secondary to disseminated haemangiosarcoma but here the skin lesions will be largely irrelevant apart from their accessibility and consequent diagnostic value. The clinical behaviour of the tumours is variable – most are usually regarded as benign but this is by no means invariable.

Clinical signs

Large (5–45 cm diameter), well-defined, firm, dark (blue-black) nodules may be seen, often on the limbs. They may involve the overlying skin, resulting in a very fleshy tumour that bleeds easily with minor trauma. The tumours can be rather more diffuse. Some may be ulcerated and others have an apparently verrucose component (see below) (Fig. 18.43). Spontaneous bleeds also occur in the faster expanding tumours. Single lesions are commoner than multiple ones. The fetlock and coronet can also be involved and in some the lesions are entirely subcutaneous (Fig. 18.44).

Large, invasive, more aggressive and malignant haemangiosarcoma also occurs occasionally. These may have an ulcerated surface and are highly vascular; bleeding occurs easily with even minor handling **(Fig. CD18 • 22A and B)**.

A verrucose form (verrucose haemangioma) with a more diffuse, ill-defined, superficial nature is also encountered where there is far less overt evidence of vascular involvement apart from a prominent cutaneous blood supply (Fig. 18.45), and then the condition can closely resemble basal cell carcinoma (see p. 409) and verrucose sarcoid.



Figure 18.43 This mixed type haemangioma was identified on the plantar tarsal region and had a predominately verrucose nature. It had an ulcerated central portion. (Figure courtesy of Dr Petr Jahn.)

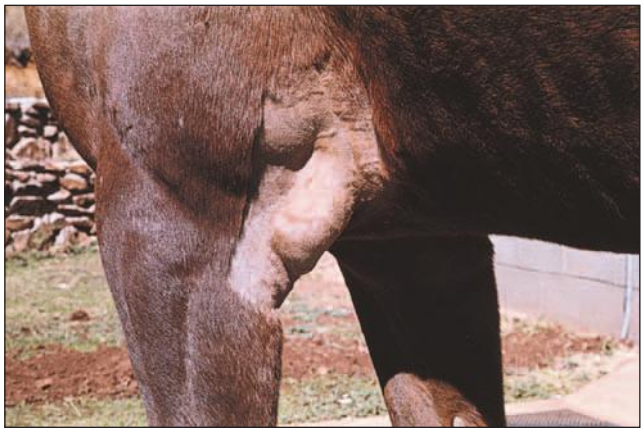


Figure 18.44 A diffuse subcutaneous haemangioma in the elbow region.



Figure 18.45 A verrucose (cutaneous) haemangioma on the caudal thigh of a middle-aged gelding. Note the prominent blood vessels involved in the apparently 'trivial' mass. This should always alert the clinician to a major problem even when the tumour is visibly small.

CD

The haemangioma is, however, dark blue-black in colour and has limited keratin alteration. In some cases they ulcerate and then become more overtly vascular in appearance and may then have an obvious plasma exudate.

Both forms have prominent blood supplies and both (including the plaque-like form) are prone to traumatic damage and heavy bleeding; they may bleed spontaneously or following minor trauma.

Differential diagnosis

- Mixed, verrucose, fibroblastic, malevolent, sarcoid: other sarcoid types are usually present and have a prolonged progressive course. Definitive histology.
- Basal cell carcinoma: very rare tumours with local cutaneous involvement and commonly an ulcerated surface with a piled-up margin; definitive histology.
- Melanoma: characteristic black, firm, usually spherical single or groups of nodules. Fine needle aspirate will reveal black or dark brown pigmented cells; largely restricted to grey horses but other forms occur in other horses.
- Equine cutaneous mastocytosis: slower growing, isolated masses with typical histology.
- Exuberant granulation tissue/wound: history of wounding and prolonged healing.

Diagnostic confirmation

- Biopsy (section or surgical removal) and histopathology.
- Obvious vascular components of varying differentiation are recognized. Some larger cavernous structures and abnormal, thrombosed vessels can be seen.
- Electron microscopy and special staining may help to establish the diagnosis.

Treatment

A few cases are reported to resolve spontaneously.

Complete surgical ablation under general anaesthesia followed by mesh, split- or full-thickness skin grafting of the resultant defect can be used but a high failure rate is common in the congenital form. Removal of all tumour tissue is also a major surgical challenge and closure of the resulting defect is likely to be problematic. Radiation teletherapy or brachytherapy may be an effective option but there are far too few cases reported to offer any definitive advice on this treatment.

Haemangiosarcoma is far more difficult to treat than the more benign haemangioma forms.

Prognosis

Recurrences are common unless surgical excision is meticulous and the common sites make this problematic. Of course where the lesion is a primary haemangiosarcoma or secondary (metastatic) haemangioma from a wider disseminated condition, the outlook is bleak.

Malignant fibrous histiocytoma (extraskeletal giant cell tumour)

Profile

Very rare malignant tumours found in the neck and proximal regions of limbs. There is no reported breed, sex or age predilection. Donkeys may also be affected. There is much debate about the true nature of these tumours. Many are associated with foreign bodies, necrotic tissue and repeated trauma.

Clinical signs

The lesions are characteristically solitary, firm and poorly circumscribed but multilobulated nodules may occur. They are locally invasive but very slow to metastasize. The overlying skin may ulcerate and leave a large, exposed, rapidly expanding and haemorrhagic tumour (Fig. 18.46). In many cases the tumour tissue is soft and easily damaged, causing moderate to severe haemorrhage.

Differential diagnosis

- Sarcoid (nodular, mixed and malevolent forms): rarely very rapid growth rate and usually multiple types in different locations. Definitive histology.
- Neurofibroma: isolated multinodular mass often around the eyes.
- Equine cutaneous mastocytoma: isolated, slow-growing, solid or slightly fluctuant mass that seldom ulcerates. Most have characteristic 'kunkers' and definitive pathology.
- Melanoma: characteristic black, solid, usually spherical masses that may ulcerate, discharging dark brown or

Key points: Malignant fibrous histiocytoma



1. A very rare, poorly defined malignant tumour of histiocytes and multinucleated giant cells occurring in older horses. Lesions are usually solitary and commonest on neck, dorsal carpus, thigh, and femorotibial regions.
2. Individual masses are nodular, firm and poorly demarcated. Many ulcerate early and become necrotic. The rate of growth can be alarming.
3. Diagnosis is usually complicated by the possibility of foreign body related giant cells. A definitive diagnosis can be reached with careful staining and elimination of other causes of giant cell proliferation.
4. Rapid growth is reported and so early surgical excision is the only option.
5. There is a high rate of recurrence.

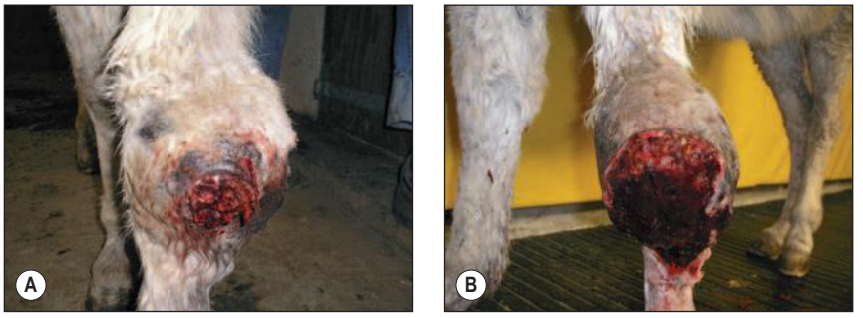


Figure 18.46 A giant cell tumour on the carpus of a donkey. The tumour appeared rapidly over 2 weeks. Picture **(A)** was taken 2 weeks before picture **(B)**. Treatment was not attempted. No secondary tumours were found at necropsy. (Figure courtesy of Alex Thieman.)

black haemorrhagic material. Very common in grey horses.
- Collagenolytic granuloma: multiple small dense nodules in the skin of the back and neck. They can reach considerable size.
- Foreign body reactions: rarely as aggressive but some 'giant cell tumours' are associated with foreign bodies.

Diagnostic confirmation

Biopsy shows characteristic histopathology.

Treatment

Surgical excision can be difficult owing to the invasive nature of the tumour, but if performed meticulously can result in resolution. Cryosurgery and laser excision are, theoretically at least, possible treatment methods.

Prognosis

Recurrence is common owing to the locally invasive nature of the tumour and the consequent difficulty in removal. Metastasis is not reported.

Leiomyoma-sarcoma

Profile

The cutaneous leiomyoma (-sarcoma) is a very rare muscle tumour deriving from the smooth arrector pili muscles. Very few cases have been reported.

Clinical signs

A single, discrete, cutaneous nodule may be the only sign. Early ulceration with minimal bleeding may occur. The tumours grow moderately rapidly but have little local invasion.

Differential diagnosis

- Sarcoid: multiple nodules and other types usually present. Far more common and distinctive in most forms.
- Equine cutaneous mastocytosis: single, slowly expanding cutaneous or subcutaneous nodule that can be pruritic in some forms.
- Squamous cell carcinoma: usually highly destructive and associated with non-pigmented skin.
- Deep dermal mycosis/fungal granuloma: single or few solid nodules with definitive histology.
- Bacterial granuloma: tends to be purulent or inflamed or both.

Key points: Leiomyoma-sarcoma



1 An extremely rare tumour of cutaneous smooth muscle.
2 A non-specific discrete cutaneous nodule is reported.
3 Diagnosis is solely by biopsy.
4 Surgical excision is possibly the treatment of choice but only one cutaneous case has been reported.
5 The prognosis is uncertain but complete excision should be curative and metastases are probably unlikely.

Diagnostic confirmation

This is by surgical excision or biopsy and histopathology.

Treatment

Total surgical removal or radiation are the only effective options.

Prognosis

A guarded prognosis is warranted because complete removal without damaging underlying tissues may prove problematic.

Lipoma (liposarcoma)

Profile

Cutaneous lipoma is relatively uncommon when compared to other species and liposarcomas are very rare. The cutaneous tumours are formed from subcutaneous lipoid tissue primarily in aged horses. Fat/obese animals are most affected, but significant lipoma can also be found in some thin horses.

Mesenteric lipomas are, however, very common in older horses, where they can cause serious colic; there is no apparent correlation between cutaneous/subcutaneous lipoma and internal lipoma formation.

Clinical signs

An entirely subcutaneous, soft but well-defined, lobulated or smooth mass occurs which may be mobile under the skin (Fig. 18.47).

The tumours grow slowly and may fluctuate in size with body condition to some extent – even starvation does not, however, result in complete disappearance.

Differential diagnosis

- Sarcoid (nodular, mixed nodular and malevolent): firmer and more definite nodular structure. Other types also present and some skin involvement in type B nodules. Predilection sites.
- Foreign body reactions: usually with discharging sinus tract and rapid resolution on removal of the foreign body. Necrotic tissue acts in the same way.
- Haematoma (organizing): history of acute onset of a fluid-filled cyst-like structure.

Key points: Lipoma



1 Rare benign tumour of fat cells, predominately encountered on the body trunk. Subcutaneous forms are far rarer than those in the mesenteric tissue where they cause major intestinal problems. Affects adult horses, with no sex predisposition. Ponies may be more affected. They are not necessarily restricted to fat horses/ponies.
2 Solitary, slow-growing, well-demarcated, soft-fluctuant subcutaneous swellings that can be large (5–50 cm diameter).
3 Biopsy or fine needle aspiration is diagnostic.
4 Surgical excision is usually uncomplicated in the benign forms but recurrence can occur.
5 The prognosis is good in most cases.

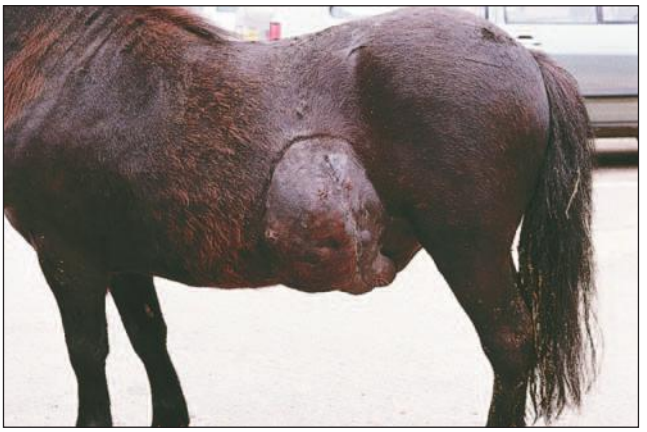


Figure 18.47 Subcutaneous lipoma in a Shetland pony mare. A surgical biopsy taken 2 weeks previously confirmed the nature of the multilobulated, soft, subcutaneous mass. It was removed uneventfully and did not recur for at least 6 years.

- Hernia: abdominal rupture/herniation with abdominal contents either subcutaneously or within a peritoneal lining.
- Muscular, vascular or connective tissue hamartoma: firm, slowly expanding mass with definitive histology. Ultrasonography shows 'typical' tissue features.

Diagnostic confirmation

- Ultrasonographic examination is very useful; there is a characteristic echo pattern typical of fat tissue.
- Histopathology (aspiration or biopsy).

Treatment

Surgical removal is effective and it may help to limit dietary intake for some weeks before surgery to reduce the tumour as far as possible. As the skin is not involved wound closure is not usually a problem. However, if the tumour is disrupted during surgery, the fat released may slow healing significantly.

Prognosis

Good, but other lipomas may develop locally or at other sites (internal and external). There is no recorded tendency to recurrence, but multiple tumours are possible.

Lymphangioma

Profile

This is a rare, benign tumour of lymphatic vessels located in axillary or inguinal regions of older horses.

Clinical signs

Multinodular or fluctuant subcutaneous masses occur singly or in groups (Fig. 18.48). There is seldom any ulceration and tumours remain entirely subcutaneous.

Differential diagnosis

- Sarcoid (nodular and malevolent): other sarcoid types are usually present and many have distinctive features.
- Lymphoma: solid, single, well or poorly demarcated mass below the skin. May ulcerate. Definitive pathology.

Key points: Lymphangioma

4 9 11 14 15 19 20 23 24

1. Rare, usually solitary but sometimes multinodular condition occurring in older horses.
2. Masses are solid on palpation and entirely subcutaneous. Ulceration is not a feature.
3. Diagnosis is achieved by biopsy but it is sometimes difficult to differentiate the pathology from forms of lymphoma.
4. Treatment options are limited to surgical excision but the slow-growing nature possibly makes interference unwarranted anyway.
5. The masses may enlarge significantly but there is little evidence of a malignant behaviour pattern.



Figure 18.48 Lymphangioma on the sternum of an anaesthetized horse. Note the multinodular group of interconnected subcutaneous lesions.

- Lymphosarcoma (generalized or cutaneous histiocytic type): multiple solid, sometimes ulcerated nodules in and below the skin. Often concurrent systemic changes if part of a multicentric syndrome. Cutaneous forms are slower and have fewer systemic effects.

Diagnostic confirmation

- Surgical biopsy (or excision) and histopathology.
- Ultrasonography is useful.

Treatment

Surgical removal may be feasible and effective. Radiation by teletherapy may be used in cases where the site precludes surgery.

Prognosis

The prognosis is guarded; some will recur and multiple tumours can be present.

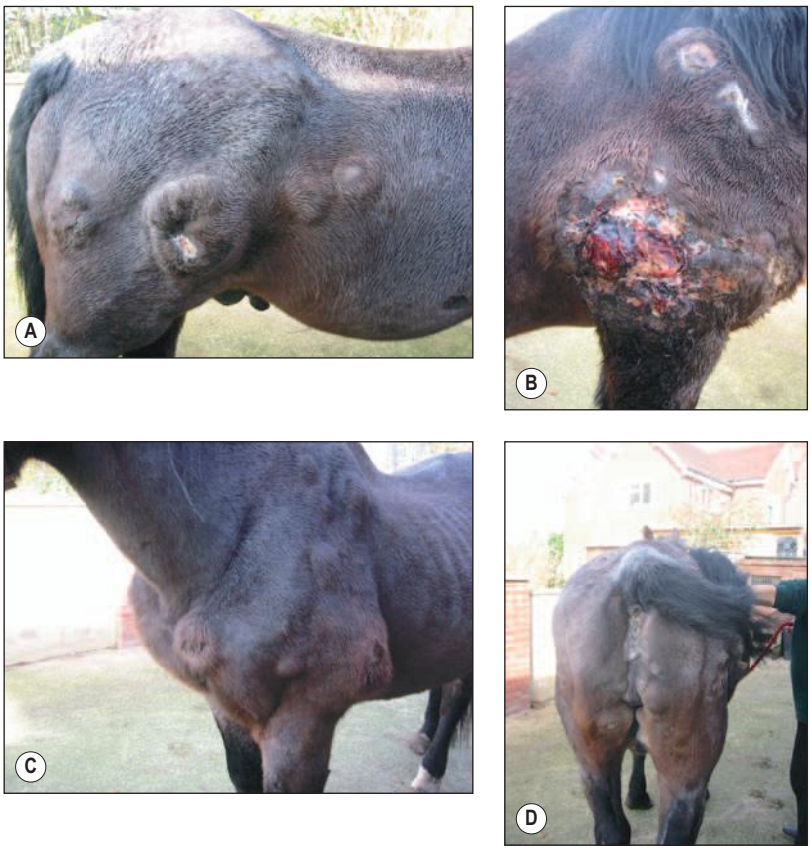


Figure 18.49 (A–D) This aged gelding developed many firm, expanding nodules over much of his body. Some ulcerated. A diagnosis of epitheliotropic lymphoma was based on histology and immunohistochemistry.

Cutaneous lymphoma

Profile

Any tumour involving the lymphoid tissue can be termed a lymphoma.

The cutaneous forms of lymphoma are the commonest malignant neoplasm of horses. Clinical presentations include epitheliotropic and non-epitheliotropic forms.

1 Isolated lymphoma
2 Cutaneous histiocytic lymphoma/lymphosarcoma
3 Multicentric lymphoma.

Lymphoma may rarely present as a skin disease with or without internal organ involvement. Diagnosis is made on biopsy and should be followed by attempts to define the extent of disease (if present) in other body systems. Lymphoma restricted to the skin may present in two forms:

1 Epitheliotropic lymphoma (mycosis fungoides) is suspected of being mainly of T-cell origin and may cause a widespread scaling and crusting with alopecia and perhaps ulceration. Affected horses have a poor prognosis.
2 In marked contrast, epitheliotropic (histiocytic) lymphoma can be restricted to the skin as a multifocal nodular skin disease and is often surprisingly benign. Nodules and plaques may wax and wane over many months or years. Many cases are responsive to glucocorticoids and many horses survive for several years following diagnosis.

Any age of horse can be affected but middle-aged horses are most common (mean age range 5–14 years). There is no sex, breed or colour predisposition.

Key points: Cutaneous lymphoma

1 A rare invariably malignant disease, typically of T-lymphocyte origin,
2 The tumour is not restricted to older horses – the mean age range is around 5–12 years. No sex, breed or colour predisposition is recorded.
3 Diagnosis is achieved by biopsy and immunohistochemical methods to confirm the T-cell nature of the tumour.
4 Treatment is possible only in isolated lesions – radiation or wide surgical excision can be effective.
5 Prognosis is poor in all cases but localized single lesions may be treated.

Isolated lesions occur in the region of the jaw and distal limb but they can occur at any site. A significant number of cases are tentatively linked to the presence of ovarian granulosa cell tumour in mares where a hormone dependency may be a factor.

Multiple nodules representing cutaneous accumulations of multicentric lymphosarcoma occur with some frequency.

Clinical signs

Two major clinico-pathological types can be recognized:

1 *Epitheliotropic form* (mainly of T-lymphocyte origin): this form predominately affects the skin and the misleadingly named mycosis fungoides is an example of this form (see p. 420) (Fig. 18.49). Single isolated or multiple lesions occur. Depressed, ulcerated lesions

involving subcutaneous infiltration give a diffuse, ill-defined character. A slight discharge with some scab formation over non-healing, ulcerated skin is usual. A conjunctival form is also recognized (Fig. 18.50).

2 *Non-epitheliotropic form*: no cutaneous involvement occurs.

Differential diagnosis

- Sarcoid – fibroma (fibrosarcoma): often multiple and usually complex features involving thinning or hyperkeratotic skin and hair loss; definitive histopathology.
- Mast cell tumours (nodular and ulcerated forms): nodular forms are more common than ulcerated ones. Definitive histology and some may be pruritic. Characteristic yellow 'kunkers' in superficial forms.
- Foreign body reaction/fibrosis: slowly expanding, discharging sinus tract with history of wound or foreign body penetration.
- Scarring/cicatrization of skin (particularly after a wound which has been very slow to heal): history of wounding and scar formation.

Diagnostic confirmation

- Clinical features.
- Biopsy or surgical ablation and histopathology.
- Ultrasonography and radiography may help.
- Careful rectal and ultrasonographic (or assay of hormones) should establish the presence of a primary granulosa cell tumour in mares.

Treatment

Surgical removal or radiation teletherapy (or brachytherapy) are the only options likely to be effective. The possible value of cisplatin is uncertain at this time. Removal of a primary granulosa cell tumour may reduce the severity of some cases.

Prognosis

Very guarded; the prognosis depends heavily on treatment efficiency. The prognosis is significantly worse for lymphomas not associated with operable granulosa cell tumours.



Figure 18.50 A conjunctival lymphoma. A single 0.5 cm diameter firm nodule was identified at preliminary clinical examination but during surgical removal three more similar nodules were identified on the conjunctival mucosa.

Cutaneous histiocytic lymphosarcoma/lymphoma

Profile

This is an infrequent tumour affecting the skin only. Middle-aged to older horses are most often affected and present with generalized lesions (trunk, limbs and neck). Some cases are possibly related to a primary ovarian granulosa cell tumour. A very similar appearance with a very much worse prognosis is presented by the cutaneous component of generalized/multicentric lymphosarcoma or cutaneous lymphoma.

Clinical signs

Multiple, subcutaneous nodules (1–35 mm diameter) are typical. The lesions may appear suddenly and then may regress slightly and reappear at irregular intervals. Later the nodules become persistent and slowly enlarge. Lesions are also found in nasal and oral mucosa and the pharynx. There may be an associated dyspnoea or possible dysphagia. Cutaneous lesions may ulcerate and become infected, producing a thin, purulent or seropurulent discharge (Fig. 18.51).

Concurrent systemic signs include depression, weight loss, generalized lymphadenopathy, ventral oedema, undulating fever, respiratory distress, reduced exercise tolerance, colic and intestinal malabsorption with or without diarrhoea. Anaemia and intermittent hypercalcaemia are also present in some cases but all these signs are far more prominent in the more rapid course characteristic of generalized lymphosarcoma with cutaneous involvement.

Differential diagnosis

- Intestinal, thoracic or generalized lymphosarcoma with cutaneous metastases: systemic involvement is rapid with a short progressive deterioration. Concurrent signs including colic, weight loss, ventral and forelimb oedema may occur.
- Cutaneous or subcutaneous lymphoma/lymphangioma: larger single or multiple ulcerated lesions with typical histological findings.

Key points: Cutaneous histiocytic lymphosarcoma/lymphoma

1 Slow-growing, multifocal cutaneous disorder of middle-aged horses. There is no breed, age or colour predilection. A relationship to concurrent ovarian granulosa cell tumour has been suggested in mares.
2 Multiple firm cutaneous nodules varying in size from 0.5 to 5 cm diameter occur. Some may be ulcerated and exudative with a plasma-like exudate.
3 Diagnosis is by biopsy and immunohistochemical staining methods.
4 Treatment is not possible but removal of a concurrent ovarian granulosa tumour may limit the rate of growth and extend the life expectancy.
5 Many cases live for 3–10 years following diagnosis. This form of 'lymphoma ' carries the best prognosis. However, it is important to differentiate it from the cutaneous manifestations of multicentric lymphoma and cutaneous lymphoma.

- Sarcoid (malevolent or nodular forms): slower and restricted to skin and subcutis. Often other forms are present.
- Cutaneous histoplasmosis (epizootic lymphangitis): ulcerative chains of discharging nodules following lymphatic vessels. *H. farciminosum* is easily identified in smears from the exudate. Geographically restricted.
- Ulcerative lymphangitis (*Corynebacterium pseudotuberculosis*): localized chains of nodules with ulceration and discharge from which the organism can be isolated with ease.
- Farcy (equine cutaneous glanders): geographically restricted and usually associated with respiratory tract signs. Ulcerated nodules with honey-coloured discharges and positive mallein test.
- Multiple insect bite reactions: transient cutaneous nodules; history of attack and focus of bite can be identified.
- Idiopathic collagen necrosis/nodular panniculitis/eosinophilic collagen necrosis: benign, multinodular disorders with small solid nodules restricted to the skin and particularly on the body, girth, neck and buttock regions.
- Urticaria: acute onset of transient, oedematous nodules that pit on pressure and resolve rapidly following removal of the cause and/or corticosteroid.

Diagnostic confirmation

- Biopsy of nodules and/or enlarged lymph nodes.
- Fine needle aspiration and direct cytology (impression smears of ulcerated lesions are often disappointing, with heavy inflammatory infiltration and infection).
- Endoscopy of nasal cavities and pharynx.
- Serum chemistry and haematology
  - elevation of liver enzymes indicates liver involvement
  - intermittent elevation of serum calcium
  - elevated serum globulin and plasma fibrinogen with reduced serum albumin concentration
  - occasionally lymphopenia or leukaemia
  - paracentesis of thorax or peritoneal cavity for cytological examination of exfoliating cells. Note, however, that many lymphosarcoma lesions occurring in the body cavities are not exfoliative and so cytology of fluid aspirates may be disappointing.
- Evaluation of bone marrow (requires skilled pathologist).

Treatment

Surgical removal of ulcerating masses is palliative only. Single ulcerating lesions can be effectively removed if the horse is only affected by the cutaneous form of the condition.

In all cases of confirmed histiocytic lymphosarcoma in mares, a detailed clinical examination must be directed to establish the presence or otherwise of a granulosa cell tumour. Removal of a concurrent ovarian granulosa cell tumour may be helpful and may delay the deterioration.

Supportive therapy is important. Iron deficiency anaemia and recurrent infections and infestations with ectoparasites and gastrointestinal parasites are common due to impaired immune status.

Prognosis

True histiocytic cutaneous forms carry a reasonable prognosis with some cases living for up to 5 or more years following diagnosis. The prognosis will depend heavily on the extent of involvement and any secondary complications such as intractable limb oedema, weight loss and parasitism. Primary granulosa cell tumours should be investigated thoroughly in all cases. Generalized (internal) lymphosarcoma cases seldom live for more than a few months from diagnosis and the progression is so rapid that differentiation from the cutaneous disease is relatively simple.



Figure 18.51 Cutaneous histiocytic lymphoma nodules over the face and muzzle **(A)**, body and neck **(B)** were found on this 11-year-old pony gelding. Ulcerated nodules were present on the limbs **(C)**. Further masses were present on the nasal and pharyngeal mucosa **(D)**.

Mycosis fungoides (malignant cutaneous lymphosarcoma)

Profile

Mycosis fungoides is a pathologically distinct, progressive epidermotropic manifestation of a T-cell lymphosarcoma (Potter & Anez 1998). It is very rare but has marked similarities to some other cutaneous conditions, most of which carry a somewhat better prognosis and therefore its clinical features are sufficient to warrant a separate description (Hilbe et al 1997).

Clinical signs

This is a generalized cutaneous condition, accompanied by extensive but patchy alopecia, lichenification with extensive scaling and flaking and localized areas of exudative dermatitis. Pruritus and erythema can be present – these signs may possibly be part of or independent of the paraneoplastic syndrome.

Concurrent signs of weight loss, lethargy and anaemia may occur also. Occasionally distinct nodules may be identified in the skin.

Differential diagnosis

- Intestinal, thoracic or generalized lymphosarcoma with cutaneous metastases: concurrent signs depending on locations and implications of the masses.
- Cutaneous or subcutaneous lymphoma/lymphangioma: may be part of the same set of clinical conditions.
- Sarcoid (extensive occult or verrucose forms): other forms are invariably present. Slowly expanding sarcoid lesions can be misleading, however, especially in the groin region.
- Cutaneous histoplasmosis: geographically restricted ulcerative, debilitating condition with *H. farciminosum* being identified easily in exudates.
- Pemphigus foliaceus: prominent scaling and limited ulceration and crusting. Chronic course and possibly some mild systemic signs. Definitive histology.

Key points: Mycosis fungoides



1. A very rare but distinctive manifestation of a lymphoblastic (T-cell) tumour condition largely restricted to the skin of older horses.
2. Signs are probably restricted to the skin and manifest as multifocal or generalized exfoliative dermatitis, with scaling and alopecia with localized nodules and areas of exudative dermatitis found in some circumstances. Pruritus and generalized signs such as weight loss, lethargy and anaemia may occur.
3. The condition is very similar to several other generalized scaling conditions and so biopsy is important.
4. Treatment is not established but systemic antimitotic and cytotoxic drugs can be attempted.
5. The prognosis is not as bad as for other forms of multicentric lymphosarcoma and the affected horse may live a useful life for some years before secondary changes become unacceptable.

- Sarcoidosis (generalized granulomatous disease): multisystemic seborrhoeic disease with a long course of ill-defined malaise. Prominent skin scaling with some alopecia.
- Generalized eosinophilic epitheliotropic disease: generalized, multisystemic condition with prominent eosinophilic infiltration of the skin but often no circulating eosinophilia.
- Systemic lupus erythematosus-like syndrome of horses: multisystemic disorder with skin changes reflected by scaling and flaking and loss of pigmentation.
- Urticaria (recurrent/persistent): transient oedematous wheals in various patterns – some are more persistent. Rapid response to single-dose corticosteroids at least in early stages.
- Zinc-responsive dermatosis: no systemic involvement – severe exfoliative dermatitis that responds (slowly) to zinc supplementation.
- Alopecia areata: no cutaneous or systemic implication apart from hair loss in defined areas (often circular at least initially) or more extensive; typical histology.

Diagnostic confirmation

Cutaneous biopsy shows severe and extensive diffuse infiltration with abnormal lymphocyte cells. This is a distinct pathological finding that enables differentiation from the cutaneous lesions of multicentric lymphosarcoma.

Treatment

In some cases the condition may be better left alone as it is restricted to the skin and apart from the cosmetics of the condition, the signs may be tolerable.

The basis of therapeutic management is similar to those used for other lymphoproliferative neoplasms and is centred on anti-neoplastic drugs (Couto 1994); this is likely to be very expensive, logistically difficult and there may be unacceptable secondary effects. Single drugs are seldom effective on their own and a combination of agents is suggested as being often effective (Johnson 1998), but there are probably too few controlled studies to provide definitive guidance. However, several treatment regimens may be attempted.

1. Prednisolone (orally, 2 mg/kg q 12 h × 14 days) or dexamethasone (orally or parenterally) at 0.1–0.2 mg/kg q 24 h, either alone or in combination with methotrexate (empirical dose of 100 mg/day for 4 days).
2. Combined regimen of prednisolone (at 2 mg/kg q 24–48 h for some weeks or months) with a single subcutaneous injection of 200–300 mg/m^2 of cytosine arabinoside every 2 weeks and chlorambucil (20 mg/m^2) administered orally every 14 days.
3. Vincristine at 0.5 mg/m^2 administered intravenously once weekly can be added to the combined protocol above if no remission is detected.
4. Cyclophosphamide (200–300 mg/m^2) intravenously once every 10–14 days.

All these treatments may be accompanied by variable side-effects that may prove unacceptable.

Prognosis

The prognosis for mycosis fungoides is probably somewhat better than for other forms of cutaneous lymphosarcoma

but the severity and persistence of the exfoliative dermatitis and repeated episodes of eczematous dermatitis and ulcerations may reduce the outlook for the horse.

Melanoma and melanosarcoma

Profile

Melanoma is the term used to describe a spectrum of skin and deeper or generalized tumours that are readily recognized in clinical practice as affecting grey or colour-diluted horses ranging from newborn to aged. The pathological classification is confused by the wide variety of names given to the clinical condition.

The benign congenital (present at birth) and juvenile (acquired at less than 1 year of age) form could justifiably be termed melanocytoma (benign melanoma) and the term 'melanoma' could be used to encompass all the other acquired tumours (including the malignant melanosarcoma) because they have a tendency to malignancy. However, the term melanoma is the one that is most understood. Whilst some aspects of the condition are well recognized clinically, some are unpredictable and do not fall into any recognizable category. Thus a congenital form has been identified as malignant melanoma in foals.

A retrospective study of cutaneous melanocytic tumours in 53 horses (Valentine 1995) revealed four identifiable clinical syndromes (Fig. 18.52):

1 Melanocytic naevus (melanocytoma): this form is histologically distinctive.
2 Dermal melanoma.
3 Dermal melanomatosis. Histologically similar to dermal melanoma but having distinctly different clinical behaviour.
4 Anaplastic malignant melanoma (melanosarcoma): this form is histologically distinct.

All the common variations are largely (but not exclusively) limited to grey horses and to a lesser extent to non-pigmented (white/abino or cremello) horses. Other colours can be affected with typical melanomas but they are rarer by comparison with grey horses.

At least 80% of grey horses over 12 years of age will have one or more melanomas at some location. Some families of grey horses have a higher incidence (possibly due to some genetic predisposition). There has been a suggestion that melanomas are increasing in prevalence in parallel with human melanoma possibly as a result of increasing solar radiation (Goetz & Long 1993), but this seems an extremely unlikely relationship – the human condition is distinctly different clinically, pathologically and histologically. Also many historical reports confirm the very high prevalence in grey horses in particular. Attempts to draw similarities between the human melanoma and the common melanoma of horses (equine melanoma) have proven largely fruitless (Seltenhammer et al 2004).

Over 95% of cases are benign, at least at the time of diagnosis (Sunberg et al 1977) but a significant proportion (up to 30%) become malignant and may metastasize (Dye et al 1993). It seems that in time there is an increasing risk that the early benign hyperplastic melanoblasts will undergo malignant transformation (this is suggested to be possible over a period of 20 years or more). This implies that a high proportion of the early 'melanotic masses' have at least the potential to adopt an aggressive pathological behaviour.

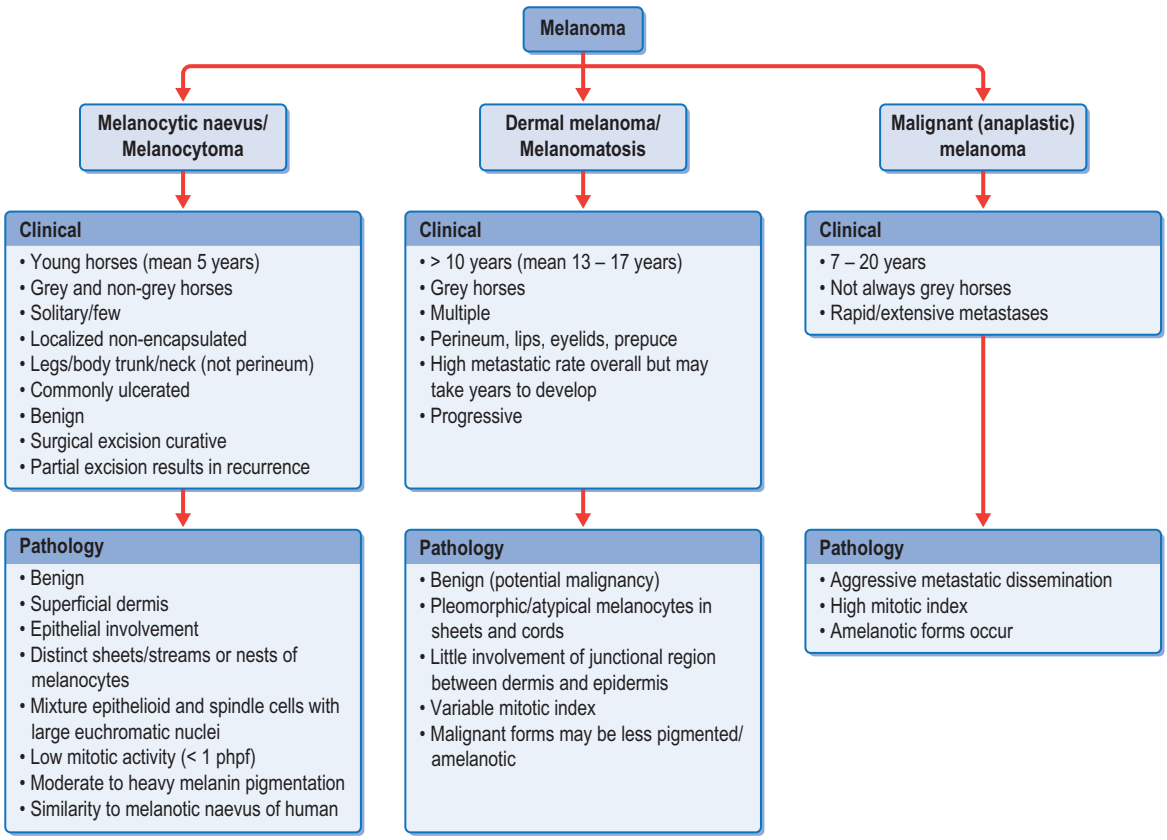


Figure 18.52 Classification and features of equine melanoma and melanoma-like conditions.

Melanomas in non-grey horses may be benign or malignant.

Many may be melanotic hamartoma ('depots' of melanin-containing cells) as a result of deranged melanin metabolism involving either an increase in the volume of melanin within cells and/or an increase in the number of melanoblasts.

They are usually non-invasive but can be coalescent. The clinical effects of the benign forms are largely due to their space-occupying bulk or to the cosmetic and debilitating effects of ulceration of the surface. A few cases are highly malignant and rapidly disseminate through the body via the blood and lymphatic circulations. In these conditions the clinical effects will of course be considerably different.

The common cutaneous sites include the underside of the tail and the skin of the tail itself, the perianal skin, the vulva and the prepuce. The eyelids and lips are probably less affected but these sites are still common. Any cutaneous site can be affected. It also occurs internally in the linings of the guttural pouches (eustachian diverticuli), particularly overlying major blood vessels in the lateral compartment of the pouches, and somewhat less commonly in the parotid salivary glands and lymph nodes, and sporadically anywhere on or in the body (iris, mammary gland, paralumbar and neck musculature, legs, major internal organs or the foot (Honnas et al 1990)). At least some of the latter are assumed to be metastatic.

There is no evidence that interference with the early benign forms will increase the likelihood of developing an aggressive malignant form. Similarly superficial ulceration is not a known precursor of, nor indicator for transformation to malignancy.

Key points: Melanoma and melanosarcoma

3 9 15 19 20 23 24

1. A very common, usually initially benign, nodular cutaneous and internal tumour of grey horses over the age of 10 years without breed or sex predisposition. Younger horses and other colours may be affected more rarely by variants of the disease and usually have far fewer tumours. There may be an increased tendency to malignancy in non-grey horses.
2. Isolated or many, single or coalescent or smaller or larger nodules are recognized – usually in or under the skin. Some other sites such as in the guttural pouches, parotid salivary glands and parotid lymph nodes or any of the internal organs can be affected.
3. Diagnosis is not usually a difficulty but there is little known about the condition pathologically.
4. Treatment options are limited. As cutaneous tumours are likely to be the only ones that owners will become 'concerned' about and because the tumours invariably grow and become more extensive and ulcerated, and have an increasing tendency to metastatic spread, surgical removal of as many as possible as soon as possible is a sensible option. It is clearly better to remove very small tumours when complete removal is feasible, than to wait until they are a surgical challenge.
5. The prognosis is guarded for the horse since new lesions will continue to develop and many will ultimately become malignant. Widely disseminated melanosarcoma carries a hopeless prognosis.

Clinical signs

The clinical signs depend largely on the location, size and clinical behaviour of the lesion(s).

Melanocytic naevus/melanocytoma Isolated, heavily pigmented, non-encapsulated cutaneous nodules varying in size from 0.5 to 2–3 cm in diameter, in the skin of grey or non-grey horses occurring in younger horses (birth to approximately 6–8 years of age) are typical. Variable number but usually relatively few lesions are present. The legs, neck, facial skin and body trunk are more often involved; characteristically this type does not affect the common perineal, penile, mouth and eyelid sites typical of dermal melanoma (see below). Individual nodules are often raised and commonly ulcerated to reveal the black/purple tumour tissue (Fig. 18.53).

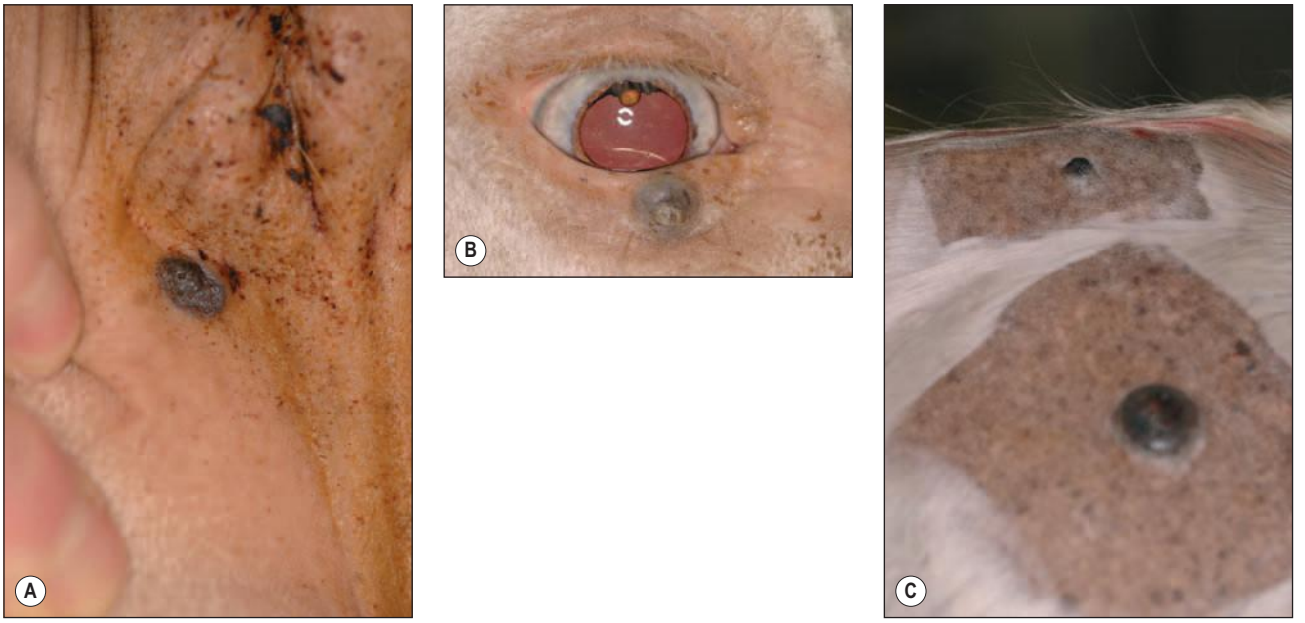


Figure 18.53 This young cremello pony had a variety of black masses at various sites including the perianal skin **(A)** from a very young age. Several stayed static but the tumours on the eyelid **(B)** and shoulder **(C)** changed dramatically after 4 years. All visible lesions were removed with a diode laser.

Dermal melanoma/melanocytosis This is the commonest form of melanoma and affects (older) grey horses almost exclusively. Single, multiple or coalescent cutaneous, often spherical nodules characteristically most prevalent in the perineal skin. Primary tumours range widely in size from a few millimetres to over 20 cm (Fig. 18.54; **Figs. CD18 • 23A–C and CD18 • 24A–D**).

Ulceration is common and then a tarry, black exudate (sometimes with haemorrhage) can be present. Single discrete nodules may have pedicles or may be coalescent with many lesions (often hundreds or more of variable sizes). Growth is characteristically slow. Some lesions are exudative and, where there are many of these, protein loss and debility can occur. Chronic blood loss can lead to mild regenerative anaemia.

Dermal melanomas can occur at almost any site and can reach considerable size. Those developing in the parotid lymph nodes and salivary glands and subcutaneous lesions are most obvious (Fig. 18.54E). Secondary signs are possible, including interference with any other organ such as the anal sphincter **(Fig. CD18 • 25A–C)**, eye (blindness or visual field deficits), nerve fibre ('stringhalt'), spinal cord (ataxia and weakness), trachea (dyspnoea/stertor), jugular groove and guttural pouch (Horner's syndrome), heart muscle (arrhythmias).

Melanomas can be restricted to the subcutaneous tissues and then there is no particular dermal implication. Sometimes sweating and hair loss can be attributable to melanoma; for example, masses in the thoracic inlet or within the jugular groove may interfere with the cranial

CD

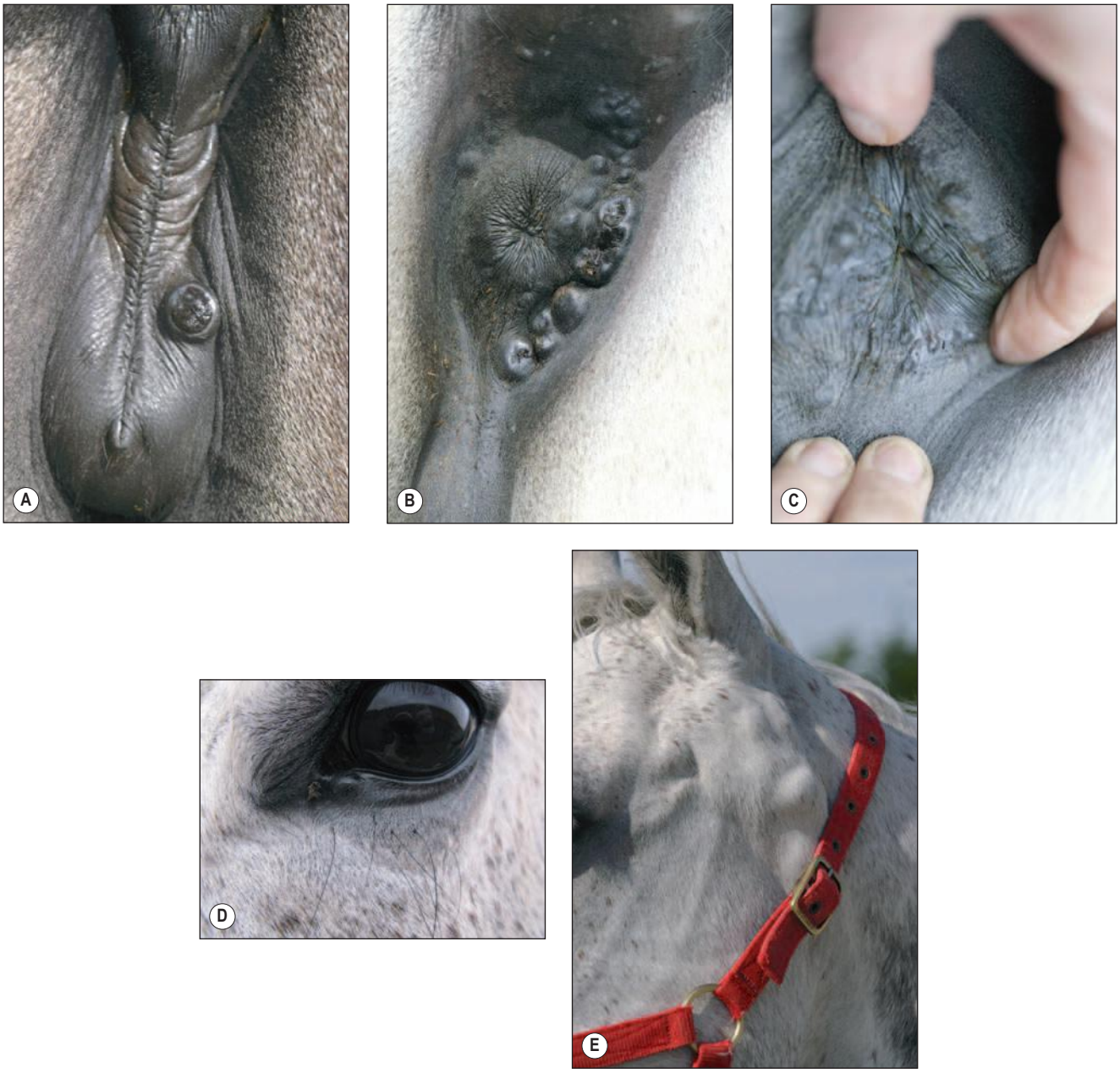


Figure 18.54 (A–E) Single or multiple cutaneous melanomas are very common in middle-aged grey horses. Some ulcerate but others do not. The perineum, penile skin, vulva and eyelid are common sites. In addition, parotid lymph node and salivary gland involvement is a common occurrence that corresponds to guttural pouch involvement.

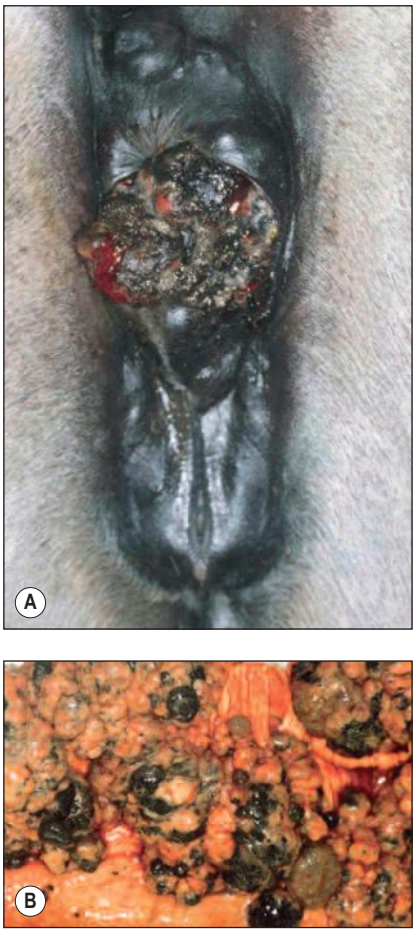


Figure 18.55 **(A)** This 9-year-old grey mare was presented for investigation of recurrent colic. During clinical examination the ulcerated heterogeneous melanoma was identified. Note the pink, grey and cream areas suggestive of anaplastic behaviour. Peritoneocentesis produced copious fluid with bizarre large cells and profuse melanocytic cells with a high mitotic index. **(B)** At laparotomy the whole abdomen and the abdominal organs were infiltrated with tumours, some of which were black and some grey/pink or cream. At necropsy a similar state existed in the chest, and most major organs were involved in this extremely aggressive malignant melanosarcoma.

sympathetic trunk and induce Horner's syndrome. Chronic cutaneous vasodilation causes sweating and gradual hair loss. The presence of isolated or even several secondary signs does not necessarily (nor even usually) mean that the condition has a certain malignancy. It is more likely that the simple space-occupying nature results in organ dysfunction. Any grey horse with a single or many defined cutaneous or subcutaneous nodules should be viewed as a potential melanoma case.

Malignant melanoma/melanosarcoma This represents the most aggressive form of the disease. The malignant forms can be recognized by the topical features of a malignant tumour. The tumours grow rapidly, ulcerate and have a diffuse, bound-down nature. Often there is a grey or grey-pink heterogeneous nature to the surface of the tumour (Fig. 18.55). Malignant dissemination has the potential to affect any internal organ and results in some paraneoplastic syndrome signs. Ultrasonographic features are reasonably well defined and the malignant forms have a softer echogenicity often with necrotic central portions, where the tumour has outgrown its blood supply. These cases will usually have other concurrent signs such as colic, weight loss, cardiac arrhythmias, respiratory or central nervous symptoms. Where any clinical syndrome is presented in a grey horse the possibility of malignant melanoma should be considered even if the cutaneous evidence is limited. It is very important to examine the surface of ulcerated melanomas and to perform guttural pouch endoscopy in all melanoma cases.

Differential diagnosis

- Sarcoid (nodular form): sarcoids are never black in colour!
- Lymphoma/lymphosarcoma: these are not black and are not restricted so much to grey horses.
- Haemangioma/haemangiosarcoma: haemorrhagic, highly vascular, non-pigmented tumours. Verrucose skin forms can have a dark purplish colour.
- Mast cell tumour: isolated single masses that may ulcerate. Not pigmented.
- Fibroma/fibrosarcoma: not pigmented but can be ulcerated.
- Parotid salivary gland pathology: parotid melanoma occurring in the salivary gland or parotid lymph node are usually extensions from the guttural pouch; other parotid swellings may be associated with empyema or tympany.
- Warble fly (*Hypoderma bovis*): not black and usually restricted to the dorsum where melanomas are very rare; geographically and seasonally restricted.
- *Gasterophilus* spp. induced ulcerated lip granuloma: not pigmented.
- Cutaneous collagen necrosis/allergic collagenolytic granuloma: myriad isolated or chains of small firm nodules in the skin. Definitive histology.

Diagnostic confirmation

- Appearance and location are characteristic.
- Biopsy by fine needle aspirate is a very simple and very positive test – the back pigmented material is obvious and pathological examination is usually not required except to establish the true character of the cells themselves.
- Biopsy and excisional biopsy reveals a wide range of pathological possibilities that all fall into the classification of equine melanotic skin disease. Bleaching of the cells is a useful histological technique to aid the detection of mitotic figures. Commonly the tumours contain large polygonal melanin-laden cells. In many primary tumours populations of pleomorphic melanin-laden cells infiltrate dermal connective tissue and extend into adjacent musculature. Only rarely is there evidence of any junctional aggregation of malignant melanocytes appearing to arise from the stratum germanitivum. Others contain spindle cells and giant cells. Non-pigmented (amelanotioc) cells are evidence of anaplastic behaviour. Mitotic indices vary widely from low to very high and reflect the potential or actual aggressiveness of the tumour.
- Complete removal and histopathology.
- It is possibly unwise to partially excise a melanoma.
- Malignant tumours may be recognized by the presence of pale grey or pink-creamy coloured areas within the black mass. Blotting the ulcerated surface with a gauze

swab or absorbent paper towel may reveal the mottled appearance (Fig. 18.55).

Treatment

Melanocytic naevus/melanocytoma

Surgical excision is curative. Partial excision results in localized recurrence. There is no convincing published evidence that residual melanocytic tissue resulting from failure to remove all the 'tumour tissue' increases the risk of dissemination or increases the risk of malignant transformation. The lack of any effective alternative means that the decision for surgical removal should be taken early and all visible lesions should be removed when they are at an easily manageable size. This approach will limit the later numbers that become problematic but it is accepted that this approach has its limitations. Any safe reduction in the number of tumours on an individual horse will reduce or delay the onset of unacceptable ulceration and tissue distortions and will limit the development of malignancy.

Dermal melanoma/melanomatosis

Some tumours are reported to regress spontaneously but this is an extremely rare event. The large majority will expand and cutaneous lesions will eventually ulcerate. There is a significant risk of development of more aggressive forms and so treatment options should be considered carefully.

Benign neglect has long been the 'treatment of choice' based on the premise that the tumours are benign and slow growing. This approach can justifiably be questioned – the tumours will only get bigger and less treatable with time and once they are large it becomes virtually impossible to remove them safely and entirely by any known means. The owner will only be concerned for the visible lesions and so it is sensible to consider early removal of all accessible melanoma lesions by one means or another. Furthermore, there is an increasing risk of malignancy the longer the early benign lesions remain. At this late stage interference is a major surgical challenge and most cases will be untreatable. It is difficult to answer an owner who asks 'Why weren't the tumours removed when they were small and benign before they became untreatable and troublesome later in life?'

Surgical removal Surgical removal of troublesome lesions is possible **(Fig. CD18 • 26A and B)**. Single small tumours (<3–4 cm diameter) can usually be treated successfully by wide surgical excision or cryonecrosis therapy. Even larger areas of complex and coalescent melanomas can be removed surgically and so improve the quality of life for the affected horse (Rowe & Sullins 2004). Ligation of isolated pedunculated lesions may be effective where this is feasible. Much larger tumours will require reconstructive surgery but even some severe cases can be helped significantly although there is an obvious (but completely unproven) risk of both recurrence and possibly even metastatic spread.

Note

Partial surgical excision alone can stimulate rapid tumour regrowth and possibly metastatic spread through stimulation to abnormal melanoblasts in proximity to the surgical site (Goetz & Long 1993). Therefore it seems entirely logical to remove the tumours as soon as they are visible and at a stage when total excision can be safely achieved.

Cryosurgery Cryonecrosis using a double or triple freeze–thaw cycle is effective in treating some localized small, ulcerated and discharging lesions and is useful for tumours which are inaccessible to total surgical excision, e.g. in the anal sphincter area. Better results are achieved when the tumour is surgically debulked as far as possible prior to cryonecrosis of the remaining inaccessible area. This rarely 'cures' the problem but it allows a further useful life span for the treated horse. There is also a serious risk of exacerbation if the tumour is not eliminated completely.

Cisplatin by intralesional injection This treatment is reported by some centres to be highly effective – many tumours appear to resolve following a single treatment. However, others have had little or no success. Tumours greater than 3 cm diameter should be debulked first and a vegetable oil-based emulsion of cisplatin injected into the base of the tumour at the rate of 1 mg/cm^3 of tissue every second week for four treatments. The drug is irritant and the use of 22–25 G needles is advisable to stop leakage following injection. Injection is continued as the needle is withdrawn to deposit the drug along the needle track. Injections should be spaced 5–8 mm apart (Theon et al 1993). Treatment is limited to small tumours and many of these will be improved or resolve. The relative risks of the tumour and the treatment do not support the use of this method in general practice. Recurrence may occur around 7–8 months following therapy. Repeat treatments may follow the same procedure.

Note

Cisplatin powder has to be freshly prepared and when reconstituted is only stable at room temperature for approximately 15 hours. It is difficult to obtain in many parts of the world.

Systemic toxicity may be a problem but the treatment has been used on stallions and pregnant brood mares without ill effects.

Post-treatment therapy with penicillin or potentiated sulphur drugs, plus an anti-inflammatory such as phenylbutazone, is advisable to minimize post-injection swelling and discomfort.

Cisplatin is a very dangerous compound and human contact and environmental contamination must be avoided completely.

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Cimetidine Cimetidine is administered orally at 2.5 mg/kg three times daily, 3.5 mg/kg twice daily or (less satisfactorily) 7.5 mg/kg given once or twice daily for 6–12 weeks. The role of histamine (H_2) and growth of tumours may be related to activation of T-suppresser cells (see p. 108). As cimetidine is a potent H_2-blocker, it has been used experimentally on melanomas. Its successful use appears to be more applicable to tumours which are showing 'active growth'. Where growth has been minimal or even static, the response is reportedly poor. The reports of successful treatment (a 50–90% reduction in tumour size; Goetz et al 1990) are based on a small number of cases and varying doses and intervals and so there is little that can be concluded as yet from the current information. Subsequent studies have failed to show any convincing evidence of a benefit in the large majority of cases. An occasional tumour does, however, seem to respond. The clinical effects are hard to quantify in most cases and so

it is useful to measure at least two dimensions of a single accessible lesion using skin calipers and then re-measure some weeks later. A response is indicated by a slow decline by 50–90% of the pre-treatment size and this response occurs during the first 6 weeks of treatment. If the drug is seen to have a beneficial effect then it should be continued daily until there is no further improvement. If no significant effect is seen by 3–4 weeks, it is probably not worth continuing.

The progression of the tumours is reported to be halted for months to years following cessation of treatment. Regrowth on cessation of treatment is also reported.

Immunological methods

1 *BCG immunostimulation/immunomodulation*. The use of BCG immunotherapy (see p. 104) has not proven successful – a localized inflammatory response occurs but this is seldom helpful in stimulating tumour rejection in any meaningful way.
2 *Autogenous vaccination*. Injection of a crude autogenous whole melanoma cell 'vaccine' has been used successfully in one case (Jeglum 1997). Vaccination with irradiated autogenous vaccines has also been attempted following some successful reports in the human literature. However, in horses the results are unconvincing at present at least. This emphasizes that extrapolation from the human circumstance is possibly unwise as the two conditions are very different in almost every respect.

Prognosis

Although the majority of 'black' lumps are benign and almost every grey horse will eventually develop them, no melanoma should be belittled. Severe aggressive melanosarcomas may be very dangerous with widespread dissemination. Individual tumours on other than grey horses can be surgically removed with little risk but slightly more caution should be exercised with grey horses. It is clinically sensible to perform all the surgeries at as early a stage as possible – there is little point in waiting for the tumours to get bigger and increasingly more difficult. Delays probably increase the possibility of developing malignancy.

Prevention is said to be possible by keeping susceptible horses out of the sunshine or applying copious high factor sunscreen (Goetz & Long 1993). However, not only is this usually highly impractical but sunshine is most unlikely to be a significant causative factor for equine melanoma so this is not helpful. Again this suggestion is largely extrapolated from the human circumstance but the equine melanoma is very different.

Ossifying fibroma (equine juvenile ossifying fibroma)

Profile

A fibro-osseous tumour mass growing from mandible, maxilla or premaxilla characteristically occurs in young horses between birth and 12 months of age. Many horses are born with the mass, which grows slowly over the following months to create a physical disability and visible abnormality. It is often only observed when the mass distorts the mouth or protrudes between the lips or interferes with eating or respiration.

Key points: Ossifying fibroma

1 Rare congenital tumour of the oral mucosa and rostral mandible.
2 Often large, firm to solid, slow-growing, roughly spherical mass with an ulcerated surface leading to lip distortion but seldom any marked disability.
3 Diagnosis is based on characteristic appearance and signalment. Biopsy can be helpful but relies on a deep sample being taken. The clinical appearance is virtually pathognomonic.
4 Treatment is by radical surgical excision often involving hemimandibulectomy.
5 Prognosis is reasonable in most cases but some local and dental deformities are possible if the mandible and teeth are involved. Horses cope well with the surgery.



Figure 18.56 Ossifying fibroma (equine juvenile ossifying fibroma).

Clinical signs

Smooth, mucosa-covered, solid (bony) mass extruding from the maxilla, mandible or premaxilla. There may be displacement or destruction of the roots of the associated incisor teeth. Rapid growth and a locally aggressive nature may create early ulceration (Fig. 18.56). The ulcerated mass rapidly becomes impacted with food material and infected. A malodorous smell is often present.

Differential diagnosis

- Adamantinoma: older horses. A rapidly growing, aggressive destructive tumour mainly affecting the mandible.
- Odontoma: rarely located in the rostral mandible. Slow growing, older horses. Radiographically distinctive.
- Squamous cell and undifferentiated oral carcinoma: highly destructive and affects the oral mucosa or non-pigmented skin around the mouth and face. Histologically distinctive.
- Dental maleruption/malformations: obvious dental deformity.
- Sarcoid: seldom on gingiva but can affect the cheek. Distinctive histology.

Diagnostic confirmation

- Biopsy.
- Ultrasonography.
- Radiography (lateral and occlusal projections) show bony proliferation of bone usually with teeth displacement; may show as dense bone or as highly invasive destructive bone lesion.

Treatment

Wide surgical excision is the only practical option for most cases. Recurrences are common unless the mass is excised effectively, and the resultant defect in dentition and structure of the bony components makes the outlook poorer. Radiation therapy is effective but very limited in availability and application. In any case it is best applied to the surgical site following surgical debulking/removal.

Prognosis

A guarded prognosis is warranted – many recur in spite of aggressive surgical excision involving extensive mandibular or maxillary bone removal.

Squamous cell carcinoma

Profile

This is a common neoplasm in horses with defined predilection sites. Squamous cell carcinoma is the second most prevalent skin tumour of horses. It is an invasive tumour of squamous cells of the skin and squamous mucosae (Table 18.6). The condition does affect other squamous cell tissues including the hoof wall and respiratory, reproductive and alimentary mucosae.

Although the tumour can develop at any site it is certainly more common in non-pigmented skin and at mucocutaneous junctions such as the eyelids, mouth, vulva, prepuce, and facial skin around the nose. Penile forms are particularly common. Studies have confirmed that the commonest site for squamous cell carcinoma is the eyelid and conjunctiva (40–50% of cases) while around 25–40% affect the male genitalia and around 10% affect the female external genitalia (Johnson 1998). In one study (Silvassy et al 1972) squamous cell carcinoma in the horse predominantly affected the external genitalia but these results are possibly unusual. The prevalence in any geographical location may depend on many environmental and management factors as well as the specific type of horses and any individual susceptibility. The skin is involved in around 20% of the cases. There is a suggestion that if the condition is present on the eyelids/conjunctiva/cornea then there is a 10% probability that it will be found at other cutaneous sites also (Dugan et al 1991).

Horses of all ages can be affected but for each anatomic location there are different possibilities for the aetiology and different epidemiological consequences. Squamous cell papilloma caused by papillomavirus infections is often suggested as being a precursor because squamous dysplasia (a precancerous change) can be recognized in some of these cases. A similar dysplastic (precancerous) change and a recognized squamous papilloma state are quite characteristic in some penile and preputial cases – here there is the possibility that smegma may be a causative carcinogen and non-pigmented skin may also be involved in some fashion. The role of papillomaviruses is uncertain.

The condition is generally considered to be rare in adult working stallions and the suggestion is that this is because smegma accumulations are far less likely. However, again there are cases that have occurred in even young working stallions. Most such cases are highly aggressive and have a devastating effect on the animal and its breeding potential.

Chronic exposure to high UV light challenge is suggested as being a significant aetiological factor in the skin and eye forms. Although it is certainly true that a high proportion of cases involve non-pigmented skin/mucosae this is not an invariable feature and serious tumours can develop in unexposed black skin. Similarly, although sunshine is clearly a strong correlating factor, the condition can occur at skin sites where sunshine simply does not reach (e.g. around the anus). There is probably at least a significant genetic predisposition to the condition and this is most noticeable in Clydesdale horses. Squamous cell carcinomas of the third eyelid, corneo-scleral junction and eyelids are probably equally important, particularly in specific breeds; for example, Clydesdale horses in Australia more frequently have tumours of the lips and eyes than of the prepuce and penis while in the United Kingdom it is somewhat the reverse.

The clinical behaviour of the tumours is very variable. The skin forms vary widely in their clinical behaviour from highly malignant to very benign, slow-growing, mildly invasive tumours. Penile forms in younger horses tend to be much more aggressive and commonly metastasize to regional lymph nodes and possibly remote organs such as the lungs and vertebral bodies. Extension to involve preputial ring or primary preputial carcinoma is a serious complication. Most of the other cutaneous/superficial forms have little tendency to metastasize.

Eyelid forms vary widely but are more often static or slowly progressive and are usually fairly well localized. Some do, however, metastasize to the local (parotid and pharyngeal) lymph nodes and may then disseminate further. Certain forms such as the gastric and respiratory mucosal forms are highly dangerous and may metastasize early.

It is important to realize that the clinical behaviour of different lesions on the same horse can be very different – each tumour needs to be assessed in its own right but fortunately tumours occurring at multiple locations are rare.

Techniques of tumour grading/staging (see p. 381) can be used to guide the clinician. Useful prognostic information on cell behaviour can also be derived from new techniques using indicators of nuclear mitotic activity measured from in-vitro cell cultures from small biopsies, and monoclonal antibodies directed at nuclear proteins associated with replicating cells are being increasingly used.

In mares, the clitoral proliferative forms are commoner than ulcerative labial forms.

The skin of the perianal region can develop an ulcerative form in horses with non-pigmented skin. The facial forms are probably commonest in the Clydesdale breed, especially where these are kept in tropical and subtropical areas, but all breeds can be affected – the restriction to non-pigmented skin may be a useful early indicator but later destruction can involve pigmented skin also.

Table 18.6 The main forms of squamous cell carcinoma in the horse and their common features

Form	Location	Features	Assumed carcinogen	Type of horse affected	Age (years)	Progression	Features	
							Clinical behaviour	Notes
Ocular	Palpebral	Proliferative	UV light	Pale skin/coloured/ cremello/albino/vitiligo	All	Slow	Benign/locally aggressive	Can be slow at first and become destructive
		Destructive						
	Caruncular			All				
	Nictitans	Proliferative		Pale exposed mucosa (no pigmented margin)			Benign but can be aggressive if on inner conjunctiva	Usually localized
		Destructive						
	Conjunctival	Proliferative		Pale non-pigmented limbal conjunctiva			Benign	Can invade cornea
	Corneal[a]	Proliferative Superficial spread mostly remaining intra-epithelial	Not known	All			Benign	Can be deeper and involve calcium deposition
Cutaneous	Skin Hoof		UV light/uncertain	Non- pigmented skin	>10	Slow	Usually highly destructive	Hoof forms are very rare
	Preputial	Invasive/destructive	Not known	All	>7	Rapid	Malignant	Can be very benign in appearance but this form is very dangerous
Penile		Proliferative	Smegma Pale skin/mucosa	Geldings All colours/breeds	10+	Slow	Benign in older horses Younger horses have more aggressive/malignant forms	Very common Precarcinomatous changes are common Older horses tend to have more proliferative forms
		Destructive			5+	Rapid		
Vulval	Labial	Destructive	Unknown	Pigmented skin can be affected	>8	Slow	Benign	Locally destructive
	Clitoral	Proliferative/destructive	Smegma			Slow	Benign	Converts to destructive
	Vaginal	Proliferative	Not known	All		Slow	Benign	Can be invasive
Oral	Mouth/tongue, pharynx	Destructive	Not known	All	>12	Rapid	Malignant	
Respiratory/ nasal	Sinus/nasal cavity/pharynx	Proliferative, invasive, destructive	Not known	All	>6	Slow	Space-occupying mass or destruction	
Gastric	Squamous epithelium/ margo plicatus	Invasive	Not known, possible bacteria/ parasites	All	>12	Rapid	Highly malignant	

[a]Intra-epithelial carcinoma (in-situ carcinoma).
Although there are some typical characteristics, as with all neoplastic disease the clinician should not be surprised to find different clinical appearances and behaviour.

Key points: Squamous cell carcinoma

2 9 11 14 15 20 23

1. The second most prevalent skin tumour of horses with many clinical variations. Aetiological factors include exposure to sunlight, non-pigmented skin, papillomavirus infections, smegma and other unknown carcinogens. There are breed predispositions. Precarcinomatous changes are recognized in some locations. Some tumours are very benign but others are extremely aggressive.
2. Three major forms are recognized: (a) proliferative form, (b) ulcerative/destructive form and (c) mixed form. Common sites include the eyelids, conjunctivae, cornea and the external genitalia of geldings in particular. Female external genitalia, normal body skin and the hooves are also liable to the condition.
3. Diagnosis is generally straightforward and biopsy is definitive. The degree of differentiation is a key factor in the choice and extent of treatment so this should be identified from biopsy.
4. Treatment is usually either surgical excision where feasible or cryosurgery or radiation but topical treatments using antimitotic compounds such as cisplatin, 5-fluorouracil or retinoids can be helpful/curative.
5. The prognosis is guarded – not many are malignant but some are and a full investigation is a wise procedure. Preventive measures including avoidance of sunshine (by management or masking) and genital hygiene in geldings are helpful and will reduce the incidence.



Figure 18.57 This 10-year-old gelding developed several proliferative lesions on the penile shaft and glans penis. Biopsy confirmed them to be squamous cell carcinoma.



Figure 18.58 Squamous cell carcinoma with extensive tissue destruction on the face. The affected area was white skinned. (Figure courtesy of Dr Ellen Singer.)

Ocular forms include benign, proliferative and aggressive ulcerative palpebral and nictitans forms. The benign types at all sites in the eye are usually slow-growing and proliferative. The ulcerative forms which are the preserve of the eyelids and the nictitans are faster and locally destructive. Proliferative conjunctival squamous cell carcinomas are relatively common at the lateral limbus (corneo-scleral junction). The corneal form ('carcinoma in situ') is slowly invasive but usually remains very superficial. All types may be slow-growing or highly invasive but remain largely restricted to superficial tissues of the palpebral skin or conjunctival/corneal epithelium. In contrast to the bovine condition, ocular squamous cell carcinoma in the horse is only rarely malignant. The nictitans (third eyelid) form is usually slow-growing and proliferative but sometimes aggressive and ulcerative.

Nasal, paranasal sinus and pharyngeal forms are less common. They are usually fast-growing and exert their main effects by occupying space within the respiratory tract with nasal, facial and pharyngeal distortions, dyspnoea and dysphagia. They are occasionally highly malignant. The gastric form is restricted to older horses with a possible connection to recurrent gastric ulceration (however caused) and *Habronema musca* or *Draschia* spp. lesions in the stomach. The tumour is extremely malignant, with direct transperitoneal spread and metastatic dissemination to liver, lungs and other organs.

Clinical signs

Precancerous changes which may be detected include thickening and mild exfoliation and ulceration of the skin; these are usually very obvious on the preputial and penile skin of older geldings (see p. 34). Initially these early signs are usually not detected and a small, granulating 'sore' on the skin which may be depressed below skin level is often the first report. In some cases a precarcinomatous squamous papilloma can develop (Fig. 18.57).

The skin at any site can be affected but non-pigmented nasal and facial skin (Fig. 18.58) and perineal skin (Fig. 18.59) are probably commonest. Invasive forms are aggressively ulcerative and rapidly erode into normal tissue (Fig. 18.60).

Nasal and oral lesions are often missed in the early stages and malodorous breath and blood staining of the saliva and nasal mucus may be reported first. Lesions affecting the pharynx may pass unnoticed until dysphagia and stertor develop. Secondary infection is common within the respiratory and alimentary tracts.

Eyelid lesions often start as white, raised plaques (recognizable histologically as squamous papilloma or precarcinomatous squamous dysplasia) at the edge of the eyelid and proceed to extensive ulceration and destruction of the eyelid, rapidly and progressively invading the periorbital tissues (Fig. 18.61). Nictitans and conjunctival forms are very common and here the earliest stages may be noted as blood staining of the tears. They may progress rapidly as appearance changes to a granulomatous and ulcerated lesion.

The penile forms may vary from single or multiple pedunculated or sessile 'cauliflower' type to smooth or



Figure 18.59 Squamous cell carcinoma of the perianal skin. Note the tissue destruction and shallow ulcer developing on the non-pigmented skin.



Figure 18.60 This 14-year-old Arabian brood mare developed a small clitoral carcinoma which was left untreated for over 14 months. By that stage the tumour had become highly aggressive and extensive destruction of the perineal skin and vulvar lips was present. There was gross enlargement of the iliac lymph nodes and the mare was losing weight significantly. No treatment was possible.

CD

ulcerated and highly invasive masses giving a wooden feel to the free end of the penis **(Fig. CD18 • 27A–D)**.

Vulvar tumours are often slow-growing and proliferative in the clitoral form but are more often ulcerative in labial forms (Fig. 18.62).

All types have varying degrees of malodour, even with early lesions.

Systemic signs may be relatively minor unless secondary metastatic spread has occurred. Weight loss, colic and chronic intestinal bleeding are signs of the gastric form, which carries a very poor prognosis and a short fulminating course. Secondary tumours can develop at any site



Figure 18.61 Squamous cell carcinoma. Destructive, proliferative palpebral form in a Clydesdale gelding.

(including the spinal cord, vertebral bodies, lungs, liver and spleen) from any form but, apart from the severe, malignant penile form and gastric squamous cell carcinoma, most are not malignant and even local lymph node involvement is unusual.

Differential diagnosis

- Sarcoids (fibroblastic and verrucose/mixed lesions): varying forms of the condition are usually present and are seldom destructive. Histology is diagnostic.
- Melanoma: highly pigmented spherical, single or multiple, sometimes ulcerated masses in typical sites in predominately grey horses over 10 years of age.
- Papilloma (especially nictitans and vulvar proliferative forms): papilloma can be a precursor of carcinoma but the benign viral fibropapilloma is usually very slow-growing in adult horses and self-resolving in younger ones.
- Mast cell tumour: isolated single nodular lesions with little destructive behaviour.
- Lymphosarcoma (cutaneous and generalized): usually non-destructive nodules but some lymphoma lesions can be ulcerated. Definitive histological features.
- Exuberant granulation tissue: history of wounding and failure to heal.
- Botryomycosis/pyogranuloma: history of wounding or focal inflamed pustule.
- Habronemiasis: geographically and seasonally restricted. Facial and wound sites mainly but can affect the glans penis where it causes a severe proliferative ulcerative inflammation. Larvae can be identified from washings. Rapid resolution with specific treatment.
- Phycomycosis and other fungal tumours: geographically restricted and associated with previous wounds.
- Other causes of colic/weight loss/anaemia and protein-losing enteropathy: secondary effects.

Diagnostic confirmation

- Surgical biopsy or even scrape cytology can be definitive.
- Impression smears are possible for eye lesions.
- Total excisional biopsy for histopathological examination usually can confirm both the diagnosis and the degree of differentiation.



Figure 18.62 **(A)** A proliferative clitoral squamous cell carcinoma. **(B)** A clitoral carcinoma that has extended into the ventral commissure of the vulva and has become destructive. **(C)** A perineal carcinoma that is affecting the vulvar lips and the perianal skin. **(D)** A vulvar labial carcinoma that affects only one side of the vulva. Note that in many of these cases the skin is pigmented.

Treatment

The main criteria for selection of treatment focus on the nature of the tumour (specifically its extent of invasion/localization) and its accessibility. A further major aspect is whether the tumour has already metastasized – if so treatment will be palliative only. Prior to the application of any treatment method, a full clinical appraisal must be performed to assess the extent of local and generalized spread of carcinoma. Attention must be paid to the likely secondary effects of treatment such as scarring and failure to remove all the tumour tissue.

Wide surgical excision, cryotherapy, irradiation, immunomodulation and chemotherapy are used with degrees of success related to the invasiveness and accessibility of the tumour. Penile forms are often resolved completely by penile amputation but in younger horses with a 'wooden' penile shaft (see **Fig. CD18 • 27D**) the prognosis is poor or hopeless. In all cases secondary spread to regional lymph nodes (including the iliac nodes which can be palpated per rectum) supports a hopeless prognosis and there is little point in subjecting the animal to surgery.

Early tumours are more successfully removed surgically with complete remission in most cases. Intralesional injection of cisplatin has been found to be useful in some cases and this is simple and relatively non-invasive but the method has implications and special precautions for the clinician.

Immunomodulation with BCG derivatives (see Chapter 4) has disappointing effects in the horse (in contrast to cattle).

Carcinomas are usually highly susceptible to gamma or beta radiation and where this is practical it can be applied effectively. Systemic chemotherapy is not feasible in the horse and in any case carcinoma is probably less responsive than other tumours.

Specific treatment methods (Table 18.7)

Surgical ablation Where wide excision can be performed, the results are usually good, e.g. amputation of tail, enucleation/exenteration with complete removal of eyelids, amputation of penis.

Very good results can usually be predicted on proliferative lesions on the third eyelid. Less successful results are obtained with long-standing invasive tumours on eyelids, prepuce, mouth and, to a lesser extent, the vulva in the mare.

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Table 18.7 Relative value of treatment modalities for treatment of common forms of cutaneous squamous cell carcinoma from the author's experience

	Surgical ablation	Cytotoxic/ antimitotic	Cryosurgery	Immunomodulation	Radiation
Cutaneous	**	**	*	**	****
					(β) (γ)
Labial	*	**	N/A	***	****
					(γ)
Clitoral	***	**	N/A	**	****
					(γ)
Penile					
<8–10 years	*	N/A	N/A	N/A	N/A
>10 years	****	N/A	N/A	N/A	N/A
Palpebral	N/A	**	*	*	****
					(γ)
Nictitans	****	***	*	N/A	N/A
Corneolimbal	**	**	N/A	N/A	****
					(β)
Carcinoma in situ	**	*	N/A	N/A	****
					(β)

*, Unlikely to be effective; **, effective in some cases or partially effective; ***, effective or partially effective in most cases; ****, likely to be effective in almost all cases (the best approach).
N/A, not applicable or invariably fails to help.

Due consideration should be given to the secondary effects of the surgery including scarring and skin/tissue deficits.

Cryotherapy (See Chapter 4 for general principles.) This can be performed on squamous cell carcinoma lesions either directly (facial, anal, nictitans, eyelid and conjunctival forms) or after surgical debulking. It is more likely to be successful in very early cases and is unsuccessful in rapidly or widely invasive tumours. The use of two or three freeze–thaw cycles is recommended. Cryosurgery gives only moderate results and is particularly poor around the eyes and mouth; while it can be very effective on limbal conjunctival and corneal lesions (Lavach 1989), in these circumstances the control of the freezing process has to be meticulous.

Cytotoxic chemicals and antimitotic drugs (See Chapter 4.) Topical application of antimitotic drugs, e.g. 5-fluorouracil and prednisolone (or other corticosteroids), may help but they have a poor reputation in most cases. The best of the topical applications is 5% 5-fluorouracil cream applied twice daily for up to 21 days. There are reports of effective management of a variety of carcinomas in this way (Fortier & MacHarg 1994, Patterson 1997). The successful treatments have been largely restricted to superficial forms in accessible locations and repeated applications over 20–30 days are required. This is not always feasible but the results are impressive. Penile forms have considerable problems with topical treatments. Retinoids such as tazarotene may also be effective. The intralesional injection of cisplatin has been found to be effective in some cases (Theon et al 1997).

Immunomodulation (See Chapter 4.) BCG cell wall extracts (or live BCG bacillus) have been used successfully in some forms of bovine ocular squamous cell carcinoma. The equine condition (at all sites) does not seem to be nearly as responsive, with very disappointing results. This treatment cannot be supported.

Radiation therapy (See Chapter 4.) This is probably the most reliably effective of all treatments, with good results expected in all cutaneous and ocular forms of the condition. Radioactive implants with gold-198 and iridium-192 isotopes offer increased chances of resolution (Wyn-Jones 1983). Interstitial implants of cobalt-60, caesium-137, iridium-192 or gold-198 have all been successfully applied. Where the tumour mass is thin, e.g. corneal carcinoma in situ, or where debulking can be employed to reduce the mass of tumour tissue to < 1–3 mm, strontium-90 probes/plaques (which are potent beta radiation emitters) can also be successfully used.

Corneal squamous cell carcinoma presents a serious challenge. Such tumours can be treated by means of sharp-excision lamellar keratectomy to clear the cornea and conjunctivectomy if the conjunctiva is also invaded, followed by 8000–10 000 rads of beta radiation fractionated over 5 days. Over 80% of such lesions may respond, with little recurrence (Rebhun 1990).

The results of radiation brachytherapy are usually very satisfactory, with a good cosmetic effect and a low recurrence rate, although the full effects may not be obvious for some months. Teletherapy is a very effective treatment for most forms of the disease which have no systemic dissemination but is unlikely to be available except under very particular and specialized circumstances.

As with all tumours, the radiation dose should be accurately calculated and careful placement of the implants can

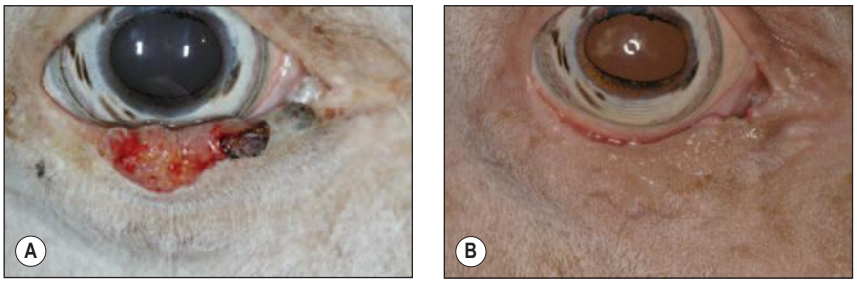


Figure 18.63 (A, B) This early destructive carcinoma was treated by surgical debulking and iridium brachytherapy. Note the small melanoma also present. The fact that the lower eyelid was involved meant that a minor cosmetic and functional problem was not important. There was no recurrence up to 4 years.

maximize the effect (Fig. 18.63). Nevertheless, some tumours will recur in time. These irradiation procedures usually require referral to specialist centres with qualified radiologists and specialized facilities for radiation protection.

Prognosis

Fortunately most (but by no means all) cutaneous forms of the condition tend to remain fairly local. Extensive local spread is rare in any case but is much more common than wide metastatic dissemination. The major exception to this is the penile form which occurs in younger geldings (<8 years of age). In these, the presence of a hard, wooden feel to the free end of the penis is a bad prognostic sign. Palpably enlarged inguinal and subiliac lymph nodes usually indicate a very poor prognosis. Preputial skin forms also carry a higher rate of malignancy than other forms.

Gastric squamous cell carcinoma and to a lesser extent nasal and pharyngeal forms are often highly malignant.

Any secondary involvement of the local lymph nodes should be viewed with suspicion and it may be justifiable to biopsy this to check for metastatic tumour. If this is present then the prognosis is very poor and it is not justifiable to subject the animal to surgery or other treatments.

Prevention

Penile forms can be prevented by regular sheath inspections and washing of suspected cases showing precancerous changes or abnormal accumulations of smegma. The greater incidence of squamous cell carcinoma of the penis occurs in older geldings, usually ponies or work horses which are less likely to receive regular cleaning of the prepuce and penis (Howarth et al 1991). Consequently, therapy should also be aimed at the regular cleansing of the prepuce and penis of this group of horses. This also particularly applies to the 'colour' type horses (Appaloosas, Piebald and Skewbald, Palominos, and to a lesser extent, grey horses). Regular washing of the clitoral region of mares may help to prevent occurrence. Avoidance of other predisposing factors can be helpful but is usually difficult (avoidance of UV light in pale-skinned horses). Regular anthelmintic worming regimens and minimization of gastric ulceration may be useful but the gastric condition is very rare and so these measures directed at prevention are probably disproportionate to the value.

Sebaceous gland tumour/carcinoma

Profile

These are rare tumours arising from sebaceous gland cells in adult to aged horses. They may occur at any cutaneous site and the causative factors are unknown.

Key points: Sebaceous gland tumour/carcinoma

1 Rare, benign tumour of cutaneous sebaceous cells found in adult horses.
2 Solitary or few, isolated, pain-free skin nodules that may ulcerate and become a fleshy exophytic mass that bleeds easily.
3 Diagnosis is usually made from excisional biopsy or a punch biopsy taken from the margin to avoid necrotic areas.
4 Treatment is not usually necessary but total excision is effective. In some sites such as the tail total excision can be problematic.
5 Prognosis is good in spite of the histological appearance of malignancy.



Figure 18.64 An aggressive sebaceous gland carcinoma on the tail. The tumour had remained small for some months and then following ulceration rapidly deteriorated.

Clinical signs

Solitary masses occur which vary from nodules to alopecic lobulated growths. They are usually slow-growing and benign in nature, but ulcerate early and become a fleshy, exophytic tumour mass (Fig. 18.64). Haemorrhage and a non-healing margin are major problems.

Differential diagnosis

- Dermoid cyst: dorsal, midline, static or slow-growing nodules in young horses.
- Squamous cell carcinoma: cutaneous squamous cell carcinoma tends to be more destructive and has very characteristic histology.

- Sarcoid (nodular or fibroblastic): usually other lesions are present and the secondary involvement of the perilesional skin (occult forms) is very characteristic.
- Neurofibroma: single, multilobulated, slow-growing masses with close resemblance to sarcoid but which tend to have less skin involvement.
- Nodular collagenolytic granuloma/eosinophilic collagen granuloma: small, focal, firm nodules in the skin in variable numbers; usually dorsal midline, buttock and neck are mostly involved. Distinctive histology.
- Insect and insect bite reactions: transient oedematous skin swellings with central bite site visible in most cases.
- Foreign body reactions: static or discharging inflammatory skin nodules with no neoplastic tendency.

Diagnostic confirmation

This is by biopsy or surgical excision and histopathology.

Treatment

Surgical excision or cryotherapy are effective with little tendency to recurrence.

Sweat gland tumour

Profile

This uncommon tumour of apocrine sweat glands occurs in older horses, most commonly around ears and vulva. These tumour-like masses are usually very benign in character.

Clinical signs

The single firm or cystic dermal nodules are non-painful, slow-growing and non-alopecic (Fig. 18.65). The lesions have a striking clinical resemblance to nodular sarcoid and neurofibroma but are histologically distinctive.

Differential diagnosis

- Nodular sarcoid/neurofibroma: usually solid with other forms present on the horse (in the case of sarcoid). Definitive histology.
- Foreign body reaction: discharging sinus tract and inflammation.
- Insect bites/stings and migrations (e.g. *Hypoderma bovis*, warble fly): transient oedematous swelling with central focus.
- Collagenolytic granuloma (allergic/eosinophilic collagen necrosis): multiple small, solid, well-defined spherical nodules in the skin. Some resolve spontaneously, others remain. Definitive histology.

Diagnostic confirmation

This is by biopsy/surgical excision and histopathology.

Treatment

Complete surgical ablation is curative.

Prognosis

A good prognosis can be given.

Key points: Sweat gland tumour

1 Rare, very benign tumour of apocrine sweat glands occurring in middle-aged to older horses.
2 Variable sized (1–3 cm) nodules in the skin. Pain-free and slow-growing.
3 Diagnosis is by biopsy/excisional biopsy.
4 Treatment is not really warranted but surgical excision is curative.
5 Prognosis is excellent.



Figure 18.65 Sweat gland tumour on the ear.

Trichoepithelioma

Profile

This uncommon tumour of hair bulb cells occurs in middle-aged horses at any skin site. It is usually very benign in character, remaining local, but may be invasive. The tumour is found most in areas where repeated mild skin trauma occurs such as the girth region so this may be an aetiological factor.

Clinical signs

A firm, single or multilobular, dermal and subcutaneous mass presents. The lesion is non-painful, slow-growing and may have an alopecic, thinned (atrophied) skin overlying it (Fig. 18.66). It has a striking clinical resemblance to nodular sarcoid and to axillary nodular necrosis. The multinodular form does, however, have an 'unusual' nature and experience helps to differentiate them. The trichoepithelioma is histologically distinctive.

Key points: Trichoepithelioma

9 10 11 14 20 23 24

1. Rare, slow-growing, nodular or multinodular cutaneous tumour of horses of any colour, sex and breed.
2. Usually subcutaneous expansions with a bound-down character to the overlying skin. Some hair loss in the overlying skin is likely.
3. Biopsy is important because of the similarity with some forms of sarcoid in particular.
4. Surgical removal is curative.
5. There is no evidence of malignancy so prognosis is excellent.



Figure 18.66 Multiple cystic trichoepitheliomata in the axillary/girth region. The masses were slow to expand and non-painful. A tentative diagnosis of nodular sarcoid was contradicted by excisional biopsy. The site healed without complication and no regrowth occurred.

Differential diagnosis

- Nodular sarcoid/fibroma: other forms usually present and typical occult changes surrounding the lesion are a good indicator. Definitive histology.
- Dermal melanoma: single or multiple firm, possibly ulcerated black-purple nodules predominately in grey horses over 10 years of age and often most visible in the perineal skin, mouth, eyelids and parotid regions.
- Foreign body reaction: single isolated nodules with an inflammatory nature and often a discharging sinus tract.
- Insect bites/stings and migrations (e.g. *Hypoderma bovis*, warble fly): transient oedematous swelling with central focus.
- Collagenolytic granuloma/girth gall/axillary nodular necrosis (allergic/eosinophilic collagen necrosis): multiple small, solid, well-defined spherical nodules in the skin. Some resolve spontaneously, others remain. Definitive histology.

Diagnostic confirmation

Biopsy/surgical excision and histopathology confirm the diagnosis.

Treatment

Complete surgical removal is possible and should be curative.

Prognosis

A good prognosis can be given.

Fibroma of frog and sole

Profile

This soft, fleshy tumour, usually in the frog region, affects horses of all breeds. It rarely causes lameness and is most often found incidentally during dressing of the foot. The cause is not certain and indeed the true neoplastic nature is not established. Potential causative factors include foreign body reactions, continued blunt trauma with sub-solar bruising and injury.

Clinical signs

A well-circumscribed, moist/haemorrhagic tumour resembling granulation tissue is often found in the frog region. The margins of the tumour are well defined (Fig. 18.67). The tumour is easily traumatized and can bleed and become infected.

Differential diagnosis

- Canker of the foot: a destructive condition of horn resulting in softening and necrosis. Well-recognized condition in heavy horse breeds in particular.
- Thrush: a transient but sometimes serious solar infection predominately affecting the clefts of the frog. A characteristic fetid black discharge is present.
- Sarcoid: extremely unusual in the solar surface of the foot.
- Keratoma: a defined tumour-like condition affecting the solar or wall keratin.
- Granulation tissue: softer, haemorrhagic, less well defined and often associated with previous injury.
- Foreign body granuloma: similar to a solar keratoma, being dense, fibrous and sometimes infected with a sinus tract.
- Foot sepsis: acutely painful with foot heat. Possibly a break-out tract on the sole or at the coronary band/heel.

Diagnostic confirmation

- Clinical appearance.
- Characteristic histology from biopsy.

Treatment

Complete surgical removal is usually very effective. Careful deep incision may be required to ensure complete removal

Key points: Fibroma of frog and sole

9 11 14 17 20 23 24

1. Rare, proliferative/exophytic fleshy tumour of the frog or sole of the foot.
2. May represent a fibrous reaction to injury or foreign body.
3. Diagnosis is made on biopsy and is mainly centred on differentiating it from sarcoid and granulation tissue.
4. Treatment is limited to total surgical excision.
5. Prognosis is guarded due to the surgical and healing difficulties and possible recurrences when all tumour tissue is not removed. Some occur in conjunction with keratoma and this will alter the prognosis.

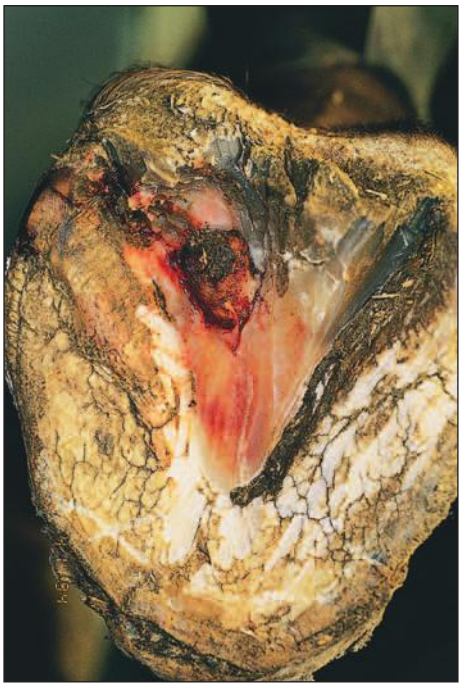


Figure 18.67 Fibroma of the frog/sole. Note the well-defined and dense, fleshy appearance.

but the defined margins of the mass make this relatively simple.

During the procedure, the margins of the mass should be carefully preserved down to the base of the tumour.

- If it is on the sole, the tract can be curetted down to the pedal bone.
- If it is situated in the frog, the dissection should be very careful to avoid opening into the deep flexor tendon sheath.

Application of a silver dressing packed into the cavity is a useful measure. The dressing should not be disturbed for 2–4 days at least. The surgical wound should be packed with antiseptic swabs and once the wound is healing, drying applications of alcohol or other astringents can encourage restoration of horn.

The foot should be kept scrupulously clean during healing with the help of a plastic boot or treatment plate. Recovery is slow, but repeat surgery is not usually required.

Keratoma

Profile

This is a tumour of keratin-producing cells in the inner surface of the hoof wall or sole resulting in a keratin-containing 'core' between the wall and the pedal bone. Most commonly lesions are found at the toe or quarter (Honnas 1997). It may result from injury or chronic irritation (Lloyd et al 1998) so that it may not be true neoplasm, or may occur spontaneously. Lameness is a consequence of pressure on adjacent sensitive tissue. If the tumour impinges on the pedal bone itself, lysis can occur.

Key points: Keratoma

1. Common foot problem affecting all types of horse. Uncertain pathogenesis but widely viewed as a tumour of horn-producing cells developing possibly as result of foreign body reactions.
2. Hoof or white line deformities accompanied by variable degrees of lameness.
3. Diagnosis is made by excisional biopsy and radiography. The clinical appearance of hoof deformity is often recognizable.
4. Surgical removal is the only option but can be problematic if the tumour extends up to the coronary band.
5. Prognosis is variable according to the extent and the degree of secondary foot involvement. Larger complicated tumours will carry a poor prognosis.



Figure 18.68 Keratoma of the foot extending from the solar margin to the coronet.

Clinical signs

Insidious onset of mild and intermittent but progressive lameness (Chaffin et al 1989), probably arising from intracapsular pressure, is often the first presenting sign. Later some change in the outline of the wall and sole contour can occur. Sometimes they are identified incidentally during foot paring and shoeing, having shown no evidence of any problems. Keratoma on the sole is usually circular, 0.5–2cm in diameter. The wall keratoma may cause considerable distortion or bulging of the toe region of the hoof (Fig. 18.68).

The tumour often extends from the coronary band to the distal margin of the hoof (Fig. 18.69). The white line is patently abnormal with a circular distortion of dense, rubbery white tissue. A few cases are patently infected with submural abscess.

Diagnostic confirmation

- Characteristic changes in contour of the wall and sole.
- Radiological examination will often identify the mass and any underlying destruction or distortion of the pedal bone.



Figure 18.69 Keratoma of the foot – surgical specimen.

Differential diagnosis

Cysts in the pedal bone usually have no concurrent wall or sole distortion.

Treatment

Surgical removal of overlying wall with careful and complete resection and curettage of tumour from underlying bone is necessary (Honnas et al 1994). Complete removal of a hoof wall keratoma usually results in an extensive wall defect at the toe. It is particularly important to dissect out the proximal limit of the mass, which may be at or just below the coronary band. It is helpful to leave a bridge of wall at the distal margin of the hoof and possibly in the central portion of the hoof, to provide support. Alternatively, a plate can be screwed to the wall of the hoof to provide a stable hoof capsule. A shoe can be applied before surgery to ensure that the defect is not subjected to excessive movement after surgery and to provide additional support for the hoof bridge. The defect can be packed with a silver antibacterial wound dressing (Aquacel Ag Tape dressing, Convatec, UK/3M, USA) until healing is underway and then filled with acrylic resin filler for a more permanent repair, which will often allow the horse to be used earlier in the course of recovery.

Note

The defect should not be filled with acrylic unless and until the tissue is completely healthy and its surface is keratinized.

This tumour has a relatively high rate of recurrence, but it is not certain whether recurrences are the result of failure to remove the entire mass or genuine recurrence. Most authors report an excellent overall prognosis provided that the whole lesion is removed and any possible causative factors are eliminated (Wagner et al 1986). There may be a persistent hoof wall deficit, particularly if the coronet was involved and damaged during surgery.

Generalized (non-epitheliotropic/multicentric) lymphosarcoma

Profile

This is a rare condition affecting adult horses (most are between 5 and 14 years of age) of all breeds, sex and colour. Widespread malignant proliferation of lymphoid cells in multiple organs occurs in generalized forms of lymphosarcoma. Although the disease affects internal organs in an aggressive way, skin is not often involved. Systemic signs and rapid progression are common.

Clinical signs

Cutaneous nodules of variable size and number which may ulcerate represent the cutaneous manifestation. Significant generalized lymphadenopathy is commonly encountered but may be absent. There are no predictable features of the disease apart from its rapidly progressive course and highly fatal prognosis.

Most cases show vague clinical signs of weight loss, inappetence, ventral oedema, and intermittent or fluctuating pyrexia **(Fig. CD18 • 28A–F)**. The course of the disease is rapid with death (or euthanasia) usually occurring within 6 months.

CD

Various secondary signs include recurrent and undulating pyrexia of unknown origin, recurrent opportunistic infections, paraneoplastic syndrome signs including pruritus (see p. 384), pemphigus, and unexplained urticaria. Significant signs of internal organ involvement such as diarrhoea, renal failure, hepatic failure and cardiorespiratory signs (including forelimb oedema, often one limb at first, ventral oedema, jugular engorgement and pulsation, pleural or abdominal fluid accumulations) are related to the organs affected by the disease and are very variable. Undulant fever is reported.

Skin bruising or an unexpected degree of surface damage and haemorrhage from relatively trivial surface trauma

Key points: Generalized (non-epitheliotropic/multicentric) lymphosarcoma



1. A rare generalized lymphohistiocytic tumour that affects all lymphoid tissues of individuals of all breeds and colours. Horses between 5 and 14 years are most commonly involved.
2. Generalized signs associated with neoplasia and immunosuppression are common. The course is rapid with deterioration within months. Skin signs occur in a low proportion of cases. Skin nodules and subcutaneous lumps over the body trunk, neck and head (including the orbit and conjunctiva) with generalized lymphadenopathy may be distinctive signs. Paraneoplastic pruritus or pemphigus may be present and some cases develop inappropriate hypersensitivity responses resulting in widespread urticarial plaques and autoimmune haemolytic anaemia and thrombocytopenia.
3. Diagnosis is made on the widespread lymphoid involvement and the presence of disseminated multicentric signs including anaemia, gammopathy and even leukaemia.
4. There is no realistic treatment.
5. The prognosis is hopeless and early euthanasia is indicated.

to mucous membranes (such as nasal or tracheal endoscopy) can be significant signs of high capillary fragility. This is sometimes encountered in generalized or splenic forms of lymphosarcoma in particular.

Differential diagnosis

- Other nodular diseases: definitive, much less significant systemic signs and definitive histology.
- Cutaneous histiocytic lymphosarcoma: much more benign disease with a very long course and minimal systemic involvement.
- Nodular and malevolent sarcoid: typical appearance with multiple lesions often with different forms and definitive histology.
- Cutaneous collagen necrosis: benign, multiple, small, hard spherical nodules in the skin of the back and neck predominately.
- Multisystemic eosinophilic epitheliotropic disease: protracted course with possible pruritus and coronitis. Nodules are composed of eosinophils and systemic signs are less common – but still occur with some frequency.

Diagnostic confirmation

- Clinical signs.
- Haematological findings (including hypercalcaemia and low globulin and albumin concentrations). A very low proportion of affected horses are overtly leukaemic but lymphocyte counts of over 100×10^9/L are recorded.

Treatment

No treatment is effective for generalized lymphosarcoma. The course is usually rapid with most cases being destroyed within 6 months of diagnosis. Non-cutaneous signs may be much more significant with respect to the prognosis.

Cutaneous histiocytic lymphosarcoma has a much slower course with individuals surviving for up to 4–6 years or more in some cases. It is therefore very important to establish the diagnosis at an early stage. Usually a skilled pathologist is required. However, if the course is rapid and inexorable then a diagnosis of generalized lymphosarcoma is probably justified.

Note

Cutaneous lumps showing histopathological evidence of cutaneous lymphoma may develop in mares secondary to an ovarian granulosa cell tumour. Typical stallion-like behaviour (or benign anoestrus) accompanied by skin nodules having the histopathological features of (T-cell-rich) B-cell lymphoma is a characteristic history. The cutaneous nodules may regress when an oral progestogen (such as allyltrenbolone) is administered for the infertility, but usually recur when therapy is stopped. Removal of the granulosa cell tumour may cause full regression of the skin problem. The role of oestrogen receptors is uncertain.

References

Anhalt G J 1997 Paraneoplastic pemphigus. Advances in Dermatology 12:77–79

Baker J R, Leyland A 1975 A histological survey of tumours of the horse with particular reference to those of the skin. Veterinary Record 96:419–424

British Equine Veterinary Association 1965 Survey of equine diseases. Veterinary Record 77:528–531

Brostrom H 1995 Equine sarcoids: a clinical, epidemiological and immunological study. Department of Medicine and Surgery, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, Uppsala, Sweden, pp 37–39

Chaffin M K, Carter G K, Sustaire D 1989 Management of a keratoma in a horse; a case report. Equine Veterinary Science 9:323

Cheville N F, Prasse K, van der Maaten M et al 1972 Generalised equine mastocytosis. Veterinary Pathology 9:394–399

Cotchin E 1977 A general survey of tumours in the horse. Equine Veterinary Journal 9:16–19

Couto C G 1994 Lymphoma in the horse. Proceedings of the 12th Annual Forum of the American College of Veterinary Internal Medicine, San Francisco, USA, pp 865–874

Diehl M, Vingerhoets M, Stornetta D 1987 Spezifische Methoden zur Enterfernung des Equinen Sarkoides. Tierarztliche Praxis 7:14–17

Dugan S J, Curtis C R, Roberts S M et al 1991 Epidemiologic study of ocular/adnexal squamous cell carcinoma in horses. Journal of the American Veterinary Medical Association 198:251–255

Dye V, Robinson J L, Aclund H, Hong C B 1993 Morphologic characteristics of equine melanomas and relationship to biologic behaviour. (Quoted in Equine pathology, (eds) J R Rooney and J L Robertson, Iowa State University, p 304)

Folkman J 1993 Tumour angiogenesis. In: Holland J F (ed) et al Cancer medicine, 3rd edn. Lea & Febiger, Philadelphia, pp 153–155

Fortier L A, MacHarg M A 1994 Topical use of 5-fluorouracil for treatment of squamous cell carcinoma of the external genitalia of horses: 11 cases (1988–1992). Journal of the American Veterinary Medical Association 205:1183–1188

Fretz P B, Barber S M 1987 Prospective analysis of cryosurgery as the sole treatment for equine sarcoids. Veterinary Clinics of North America 10:847–850

Genetsky R M, Biwer R D, Myers R K 1983 Equine sarcoid: causes, diagnosis and treatment. Compendium of Continuing Education 5:416–420

Goetz T E, Long M T 1993 Treatment of melanoma in horses. Compendium of Continuing Education 15:608–610

Goetz T E, Ogilvie G K, Keegan K G, Johnson P J 1990 Cimetidine for the treatment of melanoma in 3 horses. Journal of the American Veterinary Medical Association 196:449–452

Goodrich L, Gerber H, Marti E et al 1998 Equine sarcoids. Veterinary Clinics of North America. Equine Practice 14:607–623

Hilbe M, Meier D, Feige K 1997 A case of cutaneous epitheliotropic malignant lymphoma (mycosis fungoides) in a horse. Berliner und Munchen Tierartzliche Wochenschrift 110:86–90

Hoffman K D, Kainer R A, Shideler R K 1983 Radiofrequency current-induced hyperthermia for the treatment of equine sarcoid. Equine Practice 5:24–28

Honnas C M 1997 Keratomas of the equine digit. Equine Veterinary Education 9:203–207

Honnas C M, Liskey C C, Meagher D M et al 1990 Malignant melanoma in the foot of a horse. Journal of the American Veterinary Medical Association 197:756–758

Honnas C M, Pelsos J G, Carter G K, Moyer W 1994 Surgical management of incomplete avulsion of the coronary band and keratomas in horses. Veterinary Medicine 89:984–988

Howarth S, Lucke V M, Pearson H 1991 Squamous cell carcinoma of the equine external genitalia: a review and assessment of penile amputation and urethrostomy as a surgical treatment. Equine Veterinary Journal 23:53–58

Jackson C 1936 The incidence and pathology of tumours of domesticated animals in South Africa. Onderstepoort. Journal of Veterinary Research 6:241–248

Jeglum K A 1997 Melanomas. In: Robinson N E (ed) Current therapy in equine medicine. WB Saunders, Philadelphia, pp 399–401

Johnson P J 1998 Dermatologic tumors (excluding sarcoids). Veterinary Clinics of North America. Equine Practice 14:625–658

Kerr K M, Alden C L 1974 Equine neoplasia – a ten year survey. Proceedings of the American Association of Veterinary Laboratory Diagnosticians 17:183

Knottenbelt D C 2005 A suggested clinical classification for the equine sarcoid. Clinical Techniques in Equine Practice 4:277–295

Knottenbelt D C 2006 Treatment and prognosis for equine sarcoids. Proceedings of the 37th Congress of the British Equine Veterinary Association, Birmingham, UK

Knottenbelt D C 2009 Sarcoid transformation at wound sites in the horse (in press). In: Stashak T S, Theoret C (eds) Equine wound management, 2nd edn. Lea & Febiger, Philadelphia

Knottenbelt D C, Pascoe R R 1994 A color atlas of diseases and disorders of the horse. Mosby-Wolfe, London

Knottenbelt D C, Walker J A 1994 Topical treatment of the equine sarcoid. Equine Veterinary Education 6:72–76

Knottenbelt D C, Kelly D F 2000 The diagnosis and treatment of periorbital sarcoid in the horse: 445 cases from 1974–1999. Veterinary Ophthalmology 3:169–191

Knottenbelt D C, Edwards S E R, Daniel E A 1995 The diagnosis and treatment of the equine sarcoid. In Practice (supplement to Veterinary Record) 17:123–129

Lane J G 1977 The treatment of equine sarcoids by cryosurgery. Equine Veterinary Journal 9:127–133

Lavach D 1989 Large animal ophthalmology. Lea and Febiger, Philadelphia, p 273

Lloyd K C K, Peterson P R, Wheat J D et al 1988 Keratomas in horses: seven cases (1975–1986). Journal of the American Veterinary Medical Association 193:967–970

McEntee M F 1991 Equine cutaneous mastocytoma: morphology, biological behavior and evolution of the lesion. Journal of Comparative Pathology 104:171–178

Malikides N, Reppas G, Hodgson J L et al 1996 Mast cell tumors in the horse – 4 case reports. Equine Practice 18:12–16

Martens A, de Moor A, Ducatelle R 2001 PCR detection of bovine papilloma virus DNA in superficial swabs and scrapings from equine sarcoids. Veterinary Journal 161:280–286

Nogueira S A F, Torres S M F, Malone E D et al 2006 Efficacy of imiquimod 5% cream in the treatment of equine sarcoids: a pilot study. Veterinary Dermatology 17:259–265

Pascoe R R 1988 Colour atlas of equine dermatology. Mosby, London
Patterson S 1997 Treatment of superficial ulcerative squamous cell carcinoma in three horses with topical 5-fluorouracil. Veterinary Record 141:626–628
Potter K, Anez D 1998 Mycosis fungoides in a horse. Journal of the American Veterinary Medical Association 212:550–559
Ragland W L 1970 Equine sarcoid. Equine Veterinary Journal 2:168–172
Ragland W L, Keown G H, Spencer G R 1970 Equine sarcoid. Equine Veterinary Journal 2:2–11
Rebhun W C 1987 Immunotherapy for sarcoids. In: Robinson N E (ed) Current therapy in equine medicine, 2. WB Saunders, Philadelphia, pp 637–639
Rebhun W C 1990 Treatment of advanced squamous cell carcinoma involving the equine cornea. Veterinary Surgery 19:297–302
Reid S W, Smith K T 1992 The equine sarcoid: detection of papillomaviral DNA in sarcoid tumours by use of consensus primers and the polymerase chain reaction. Proceedings of the Sixth International Conference on Equine Infectious Diseases, Cambridge, UK, pp 297–300
Riley C B, Yovich J V, Howell J M 1991 Malignant mast cell tumours in horses. Australian Veterinary Journal 68:346–349
Roberts D 1970 Experimental treatment of equine sarcoid. Veterinary Medicine for the Small Animal Clinician 65:67–69
Rowe E L, Sullins K E 2004 Excision as treatment of dermal melanomatosis in horses: 11 cases (1994–2000). Journal of the American Veterinary Medical Association 225:94–96
Scott D W, Miller W H 2003 Neoplastic and non-neoplastic tumours. In: Equine dermatology. WB Saunders, St Louis, MO, pp 730–731
Seltenhammer M H, Heere-Ress E, Brandt S et al 2004 Comparative histopathology of grey-horse-melanoma and human malignant melanoma. Pigmented Cell Research 17:647–681
Silvassy I P, Brown J W, Dalgreen R R 1972 Prepucial carcinoma in a horse. Veterinary Medicine for the Small Animal Clinician 67:1329–1330
Slovis N M, McEntee M C, Fairley R A et al 2001 Equine basal cell tumors: 6 cases (1985–1999). Journal of Veterinary Internal Medicine 15:43–46
Steiner A 1988 Prufung des Immunetherapeutikums Nomagen zur Behandlung des equinen Sarcoids im Vergleich zur kryochirugischen Therapy. PhD Thesis, University of Zurich, Switzerland
Studer U, Marti E, Stornetta D et al 1997 The therapy of equine sarcoid with a non-specific immunostimulator; the epidemiology and spontaneous regression of sarcoids. Schweizer Archives Tierheildkunde 139:385–390
Sundberg J P, Burnstein T, Page E H et al 1977 Neoplasms of Equidae. Journal of the American Veterinary Medical Association 170:150–158
Tan R H H, Crisman M V, Clark S P et al 2007 Multicentric mastocytoma in a horse. Journal of Veterinary Internal Medicine 21:340–343
Theon A P 1998 Intralesional and topical chemotherapy and immunotherapy. Veterinary Clinics of North America. Equine Practice 14:659–671
Theon A P, Pascoe J R, Carlson G P, Krag D N 1993 Intratumoral chemotherapy with cisplatin in oily emulsions in horses. Journal of the American Veterinary Association 202:261–267
Theon A P, Metzger L, Griffin S 1994 In situ analysis of cellular proliferation in canine, feline and equine tumours by immunohistochemistry: a comparison of bromodeoxyuridine, proliferating cell nuclear antigen, and interchromatin-associated antigen immunostaining techniques. Journal of Veterinary Diagnostic Investigation 6:453–560
Theon A P, Pascoe J R, Madigan J E et al 1997 Comparison of intratumoral administration of cisplatin versus bleomycin for treatment of periocular squamous cell carcinoma in horses. American Journal of Veterinary Research 58:431–436
Turrel J M, Stover S M, Gyorgyfalvy J 1985 Iridium-192 interstitial brachytherapy of equine sarcoid. Veterinary Radiology 26:20–24
Valentine B A 1995 Equine melanocytic tumors: a retrospective study of 53 horses (1988–1991). Journal of Veterinary Internal Medicine 9:291–297
Wagner P C, Balch-Burnett O, Merrit F 1986 Surgical management of keratomas in the foot of the horse. Equine Practice 8:11
Williams M A, Dowling P M, Angarano D W et al 1995 Paraneoplastic bullous stomatitis in a horse. Journal of the American Veterinary Medical Association 207:331–333
Willis R A 1952 The spread of tumors in the human body. Butterworth, London
Wyn-Jones G 1979 Treatment of periocular tumours of horses using radioactive gold grains. Equine Veterinary Journal 11:3–6
Wyn-Jones G 1983 Treatment of equine cutaneous neoplasia by radiotherapy using iridium-192 linear sources. Equine Veterinary Journal 15:361–365

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Syndromes in equine dermatology

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I have six honest serving men,
(they taught me all I knew);
*their names are **What** and **Why** and **When***
*and **How** and **Where** and **Who**.*

From: ***The Just So Stories*** (1902) by Rudyard Kipling (1865–1936)

Note

The coloured tables shown in this section are provided as an aid to the differential diagnosis of the various clinical possibilities. It is important to realize, however, that in tables of this nature it is impossible to describe all the features, and so a page number has been included to allow rapid cross-reference to a more complete description of the condition being considered. The lists are not exhaustive but illustrate the main diagnostic possibilities. Further information can be gained in the differential diagnosis section for each disease, where the salient differentiating features are given.

Disorders shown in bold type are common; normal font is used for rarer disorders (remember that the prevalence and incidence of a particular condition can vary widely in different locations and under different circumstances).

Letters in parentheses indicate that the sign is occasionally present.

Key to coloured tables

Severity codes

○ = very mild
● = mild
● = moderate
● = severe

Presence/absence of feature

Y = sign present
y = sign present but not prominent
N = not a feature of the disease
(n) = sign less often presented

Persistence/recurrence/transience of feature

P = persistent sign (can be fluctuating)
(p) = sometimes persistent
R = recurrent sign occurring at regular or irregular intervals
(r) = sometimes recurrent
T = transient sign (lasting days/weeks)
(t) = sometimes transient

Focal nature of feature

S = single (or few) focus of disease
M = multiple focus or generalized sign

This section contains information and aspects of differential diagnosis that the busy practitioner might consider when faced with an equine dermatology case. The significant aspects of the various syndromes are described in tables that show infectious diseases in a block and non-infectious disorders in an adjacent block.

The commoner conditions are shown in bold text while the less common are shown in normal font. To make the learning experience easier the various diseases are ordered in the standard format rather than simply in their prevalence because there is a wide variation in prevalence of many of the diseases in the various geographical locations. This should allow rapid cross-reference to the main text for each condition. Thus, infections are listed in the order:

1 virus
2 bacteria
3 fungus
4 protozoal
5 parasitic conditions.

Non-infectious conditions are listed in the order:

1 genetic and developmental disorders
2 allergic and immunological diseases
3 chemical and toxic damage
4 nutritional conditions
5 cardiovascular diseases
6 neurological conditions
7 endocrine abnormality
8 traumatic skin diseases

9 neoplastic diseases
10 iatrogenic/idiopathic diseases.

Where the clinical sign is shown in different sites it may be helpful to consider where a disorder occurs at both sites. This is a more likely differential than the concept of a separate disease that presents with the same sign at two different sites. However, the interpretation must always include consideration of the 'codes' for each condition and the specific descriptions and diagnosis of the various diseases in full in the main part of the text.

Cutaneous pain

The presence or absence of cutaneous pain is a helpful diagnostic feature of skin disease. Almost all texts describe disease as being either 'painful' or 'non-painful' in the same way as they are described as being pruritic or non-pruritic. Pain needs to be differentiated from discomfort such as might occur with an over-tight bandage or ill-fitting rigid limb cast or irritation from myiasis, for example. In the latter the horse may bite at or chew at the cast, indicating possible discomfort. Pruritus is noticeably different in its clinical appearance but in a few cases horses will bite and chew at a painful focus as well and so careful clinical examination is essential. The major objective is to assess whether the existing condition is painful either on palpation or without palpation.

Pain can usually best be recognized by the response to palpation. The extent of avoidance that a horse shows when a suspected lesion is palpated is usually proportional to the pain it experiences. However, some ponies and most donkeys in particular may be stoical to the point of appearing to feel no pain. Mild resentment and reflex avoidance behaviour is usually a strong indicator of mild pain. Severe avoidance behaviour either by withdrawing from the threat of contact or direct aggression may be encountered in more severely painful conditions. The accepted overt expressions of pain in horses such as increased heart rate and hyperventilation are not common features of dermatological pain alone.

Pruritus (the itchy horse)

Pruritus can be defined as the (unpleasant) sensation that encourages the animal to itch and is one of the commonest dermatological clinical presentations. It is vitally important to recognize that pruritus is a phenomenon attributable to both central and peripheral neural mechanisms (Blood & Studdert 1988). Nerve endings in the region of the dermo-epidermal junction transmit the sensation of 'itchiness' via slow-conducting C-fibres to a central region where the itch sensation is modulated. 'Scratching' at the site of itch is transmitted via fast conducting fibres and results in a temporary suppression of the sensation by down-regulating the peripheral input (the so-called Wall's gate phenomenon). Pruritus is a common symptom of local peripheral and central pathology and can be triggered by many factors. In addition pruritus such as that associated with insect bites in particular may continue until there is a marked local inflammatory response arising from self-trauma. Only then does the pruritus abate – preventing self-trauma can therefore prolong the 'itchy' state. A diagnostic complication can therefore arise when the pruritic state results in masking of the original signs and this aspect should not be forgotten.

The extent or severity of the pruritus exhibited by an individual animal is very variable – there is no consistent degree of pruritus for any particular condition. Some horses will show mild signs with severe disease and others will show marked pruritus with mild disease of the same type. This is possibly the main reason why grading pruritus has not gained any credibility in horses at least; however, a scale of 0 (no itch) to 10 (extreme itch) has been suggested and it can sometimes be helpful in assessing progression and the effects (positive or negative) of therapeutic interventions in a particular individual (Scott & Miller 2003). This can be difficult to apply when there is variability of signs at different sites and under different conditions such as warmth, cold, etc. Often the severity of the itching can be determined – sometimes simply by asking the owner and at other times by assessing the severity of the secondary changes. Thus, an animal with a severe localized allergic response to the bite of an insect may cause severe self-trauma at the site – often the induced skin damage and inflammation is more extensive than the original bite/sting or insult. Pruritus can therefore be self-perpetuating and triggered by a relatively minor episode of intense itchiness. This can be followed by rubbing, biting and self-mutilation which, in turn, cause further local inflammation and irritation. The instigating factor may not therefore be present at the time of the examination or may be significantly masked by the changes. Most pathologists do not relish the assessment of a chronically rubbed/itched and self-traumatized skin. The more prolonged the effects the more difficult it is for the clinician to be sure of the original cause and the more damaged the skin the less the pathologists will be able to glean from biopsies (Fig. 19.1).

The location of the 'itching' can, however, be a marker for the location of the pathology but there are some circumstances such as rabies, for example (see Fig. 15.2), when the clinical response can be referred, i.e. the pathology is in a different place to the itch. Similarly, head-shaker horses often rub the side of the face suggesting pruritus but this may



Figure 19.1 This horse suffered a prolonged, severe self-trauma to the breast and anterior shoulder region of both sides. The location is unusual for any of the 'common' causes of pruritus but might be consistent with a contact irritation of some sort. It was impossible to get any meaningful information from biopsy of this case and only symptomatic treatment could be applied. (Figure courtesy of Dr Andrea Bertuglia.)

be a response to pain or irritation within the nasal cavity, or to numbness or to 'tingling' sensations (see Fig. 15.3). If the horse could just tell us where and what it is feeling, the diagnosis of pruritus in horses would be far simpler!

It is helpful also to establish whether itching initially occurred in the absence of a perceived lesion (as might be indicated by an allergic response) or whether the itchiness followed the appearance of a distinct lesion. For example, a rabid horse may itch at the site of an original bite wound on the carpus while the pathology that results in this is within the central nervous system. In the same way many head-shaking syndrome cases rub their noses – often to the point of moderate to severe skin damage – it is difficult of course to differentiate between the response to a genuine pruritus (true itch) and nerve 'tingling' or numbness both of which can induce rubbing (false pruritus).

Pruritus is not commonly associated with deep ulceration of the skin. In a few cases pain will result in 'directed attention' at the site (licking and chewing responses) but clinically this is recognizably different from pruritus. However, ulcers may be painful and may induce self-inflicted trauma *suggestive* of pruritus.

Often the pruritic horse will show a 'pleasure response' when the affected area is gently rubbed while the palpation/touching of a painful area will be resented and the animal will show aversion behaviour. The horse that exhibits the rub-pleasure response in the absence of any defined cause usually has an underlying hypersensitivity problem.

Pruritus is a particular feature of parasitic infestations and allergic skin inflammation. It can also arise from hypersensitivity reactions of immediate or delayed responses. It may be due to a primary skin disease, usually related to insect bites, It may be related to immune-mediated diseases such as pemphigus foliaceus or to a wide variety of bacterial, fungal or parasitic diseases. It may also be due to direct irritation by chemicals or to photoactivated dermatoses. More rarely in the horse it may be a manifestation of a systemic disease – the paraneoplastic syndrome, for example, often includes episodes or persistence of pruritus. In the case of hepatocutaneous disease the itch may be less easy to explain – it may have a central or local origin.

Pruritus arises as a result of either physical skin irritation (such as occurs with ectoparasitic infestation, e.g. *Werneckiella* (*Damalinia*) *equi* spp. lice) or superficial skin irritation (such as occurs with mange mites, e.g. *Chorioptes* spp./*Sarcoptes* spp.), or as a result of an inflammatory response. The causes of an inflammatory response may be local or generalized, and in the former the responses can be local (restricted to the direct vicinity of the inciting cause) or can have a more widespread clinical effect. Thus an animal with a louse infestation may have no skin inflammation but severe pruritus can arise from the irritating effects of the movement of the parasites in the hair coat. Horses with chorioptic mange also itch and stamp (cardinal signs of limb pruritus in the horse) because of the irritation effects of parasite movement, but in this case the movement is much closer to the skin itself and the extent of movement of an individual parasite can be much more limited. Burrowing mites such as the sarcoptic mange mites have a dermal irritation effect and, while they do cause local inflammation, their movement is also responsible for the clinical responses. A single insect bite or sting may cause a prominent local inflammatory response but not all such responses are liable to induce pruritus (as opposed to pain). Even a small amount of venom may cause a serious systemic release of inflammatory mediators with systemic as well as (or without) dermatological signs including urticaria and possibly generalized pruritus. In the horse this response is somewhat less prominent than in other species.

Cutaneous infections may also cause self-itching – it is important to differentiate this from a pain response; some horses will bite or chew at a painful locus. Bacterial infections do rarely cause pruritus but the fungal infections (superficial dermatophytosis) or deep (mycetoma) are somewhat more likely to be pruritic. In the former case itch is not one of the main signs but gentle (and carefully protected) rubbing of the lesion may elicit the 'pleasure' response manifest by muzzle twitching, grimacing and arching of the back towards the site. Deep fungal conditions such as pythiosis (see p. 183) can cause enough irritation to cause severe self-trauma.

Although equine urticaria and most hypersensitivity reactions only rarely cause pruritus, insect bite hypersensitivity (IBHS) (most frequently due to *Culicoides* spp. hypersensitivity) is probably the commonest single pruritic disease. Horses suffering from even a single (or few) bites show a very significant extreme pruritic response. The response to masses of bites from the same insects in a non-sensitized horse can be very similar but is usually sporadic, more transient and more restricted anatomically to the preferred regions of insect activity.

The site of the itching is usually a really good indicator of the location of the problem. A horse affected by the spinose ear tick (*Otobius megnini*) or aural (*Otodectes* spp.) mites or the bites of the *Simulium* fly (see p. 210) or certain species of *Culicoides* may itch and worry at the ear and indeed an affected horse may develop a non-neurological head-shaking syndrome with rotatory movements of the head and 'ear shaking'. A common symptom in equine practice is perineal itching. Here the differential diagnosis may include the insect bite hypersensitivity ('sweet itch') syndrome, lice (the tail base being one of the preferred sites for both the major species of louse), or oxyuriasis (due to the combined effects of adult worm activity in and around the anus and perineal skin as well as the presence of the eggs). There are several species of fly, such as *Hippobosca equinum* (see p. 210) and *Haematobius* spp., and some mosquitoes that prefer to feed at this site also. The physical presence of the insects may be enough to cause localized worry but there may also be some direct localized hypersensitivity responses.

Another quite common perineal or rump rubbing/'pruritic' condition is associated with the accumulation of skin debris and grease in the intermammary region of mares. It is always therefore worth checking for this condition in a mare that rubs her rump region (Fig. 19.2).

There are also a few conditions that are characterized by pruritus with minimal or at least easily overlooked reasons. For example, there are horses that have a psychogenic itch – these cases may be distracted from the 'neurosis' (see p. 331). A well-recognized form of this occurs in bored stallions where they develop a so-called 'self-mutilation syndrome'; they will actively bite and chew at a defined location (often on the flank or stifle region). It is very important to investigate such cases very thoroughly because there are significant differences in management and treatment between the various diagnostic possibilities.



Figure 19.2 This mare had been presented for investigation of perineal pruritus. A biopsy had been taken 3 days before referral presentation (left buttock site). The mare showed no other signs at other sites. Simply cleaning out her intermammary detritus resolved the issue. This pattern of pruritus is significantly different from that caused by insect bite hypersensitivity but is similar to that caused by *Oxyuris equi* infestation. However, in the investigation of pruritus no possibility should be ignored.

Investigation of the pruritic horse

The investigation will always involve a detailed history of the case and the specific history of the pruritus. As many horses will be presented in a chronic state, often having received all manner of medications and manipulations, obtaining an honest and complete history is sometimes very difficult. Usually the clinician will be able to consider the veracity of statements suggesting and reporting 'acute onset' when the skin is really thickened and lichenified! It is easy to be critical of this but it is far better to gradually extract the truth by careful questioning in a variety of ways than to directly confront the client.

A full general history should be obtained during which the presence of systemic disease or other chronic conditions and any concurrent medications that may mask or alter the clinical signs may be identified. For example, it might be possible to establish that the horse had been losing significant weight recently; the non-dermatological signs may be due to a second disease but they may be part of a wider syndrome. In the latter case the skin may be the best and possibly the only visible indication of a systemic disorder. A suggested list of preliminary questions is shown in Table 19.1.

Possibly the most important historical step is to establish how long the horse has been itching and when, where and, if possible, why it started. An acute onset of generalized pruritus associated with a change in feed will clearly have a different implication from a recurrent summer seasonal onset closely related to turn-out. Aspects of contagion can be established from enquiry as to whether other horses in contact (directly or indirectly) are affected similarly.

A further fundamental question is related to the presence of a defined lesion *prior* to the onset of itching or whether any visible lesion actually developed after the pruritus. The latter may be due then to self-trauma and subsequent biopsy of this region may be very misleading (see Fig. 19.2). This can be very difficult to establish simply because the owner may not have examined the horse closely enough to detect a lesion before or soon after the horse started to rub or scratch the site. It is possibly unfair to expect owners to be able to answer this question but if it can be answered it is a big help.

The course of a pruritic lesion is unpredictable. It is well recognized in all species that a single focus of pruritus usually abates if the skin becomes inflamed – once the skin surface is broken and inflammation is induced the itch may either settle or at least reduce. Of course this may lead to superficial or in some cases deep infection and then the outward evidence may be masked or very different from the original or primary state.

As might be expected, a full dermatological examination must then be performed (see p. 25). This is particularly important because of the possibility of helpful signs in other body systems. An old horse affected, for example,

Table 19.1 The significant aspects of the clinical investigation of a pruritic horse

1. Was pruritus the first sign?
   - a Where was the first site affected?
   - b Were lesions present before the itch or was the itch present before the lesion(s)?
   - c Was the pruritus localized to one site or several defined sites or more general?
2. When did the pruritus first begin and how has it progressed since then?
   - a Sudden/insidious onset
   - b Gradual/rapid progression or static extent/severity
   - c Alterations associated with management procedures/manipulations
     - Worse/better with different feeds or changes in management
   - d Changes occurring naturally (seasonal/non-seasonal)
   - e Changes occurring as a result of applied therapeutic measures
3. Are any exacerbating factors obvious?
   - a Heat/cold
   - b Exercise
   - c Medications/feeds
4. What alleviating factors can be identified? (What makes it better?)
   - a Management manipulations
     - Worse/better inside or outside
   - b Medications
5. Are any other horses similarly affected?
   - a Did they develop the signs at the same time or another time (before/after)?
   - b Are any similarly affected horses in contact with each other or subjected to the same management?
6. Has the condition adversely affected the horse or not?
   - a Affected prior to onset of pruritus
   - b Affected after onset of pruritus

by pituitary pars intermedia dysfunction (PPID/equine Cushing's disease) may show characteristic features and this condition may suppress immune processes sufficiently for ectoparasites and bacterial skin diseases to develop. Failure to recognize the underlying primary condition may waste time, effort and money. It is often very helpful for the veterinarian to observe the responses that the horse shows; if this cannot be achieved within the normal time scale of a clinical visit a video recording taken by the owner may help enormously; there are 'secret scratchers' who only show the signs when they alone and others that only show the signs at night! Of course such a manifestation may be a strong indicator of possible causes also. A horse that rubs or scratches its legs and face when it is only in a stable may be exposed to biting or worrying parasites in the bedding.

As pruritus is simply a sign of many possible diagnostic options, further samples and diagnostic tests are commonly undertaken. The commonest overall cause of pruritus in the absence of a defined pre-existing lesion in the general population are the ectoparasitic conditions and so a common early test is the collection of brushings from both affected zones and from the animal generally (see p. 60).

Biopsy of pruritic skin lesions or areas affected by pruritus where no overt lesions are apparent is frequently disappointing. In the former case there may be too much secondary trauma and in the later the pathological evidence may be unremarkable or non-specific.

No diagnostic technique can be completely precluded from an investigation of the pruritic horse because some cases can arise from highly unusual disorders such as a cervical vertebral fracture (see p. 332) or a serious internal neoplasm. In some cases even rectal examination, ultrasonographic examinations, or radiography (conventional or computed tomography) may be justified.

Fortunately in some cases, such as insect bite hypersensitivity (IBHS/sweet itch) and atopy, a diagnosis can often be suggested by the detailed history alone. In the former the condition is highly seasonal and anatomically restricted (tail, mane and ventral midline most often), begins around 3–6 years, improves when the horse is brought inside and deteriorates each sequential year during the challenge season. In the latter, the onset is usually between 1 and 3 years, is much less anatomically restricted, is non-seasonal and is usually not improved by altered environmental management. In others, for example in *Chorioptes* spp. infestation, the clinical examination and further tests may help to establish the true cause. Sometimes the diagnostic process can be much more problematic and involved; for example, confirmation of an allergy may involve challenge/deprivation tests, skin or IgE-related blood tests.

There are, however, a few cases for which a working diagnosis cannot be achieved and these are clearly a problem. In these, test therapy may help to identify the type of pathology present and then some extrapolations can be drawn. For example, administration of an antihistamine or an analgesic may provide useful information whether they are effective at eliminating the itch or not.

A good working knowledge of the main and less common disorders associated with pruritus can help enormously. Table 19.2 and Fig. 19.3 outline the main diagnostic possibilities for the pruritic horse; there will, however, inevitably be some rare conditions that are not shown here.

Alterations in hair quality and quantity (hair follicle disorders and alopecia)

Normal shedding of the body hair coat is governed by seasonal photoperiod factors. It is also true that other factors will alter the physical appearance and the length and density of the hair coat. Horses in very hot climates and those that are heavily rugged during winter months in cold climates do not usually grow a long winter coat. They will shed normally of course but the process is down-regulated by body and ambient temperature and the extent of photoperiodicity stimulus.

Alopecia is defined as the absolute loss of hairs or the reduction in hair length or diameter when the number of hairs per unit areas may be normal (Fig. 19.4). Alopecia can be congenital (and sometimes also hereditary), when it is termed hypotrichosis, or acquired. The name alopecia does not apply to the simple rubbing away of hairs to the extent of an apparently bald patch such as occurs when rugs or other tack/harness rub hairs away. In these cases the skin is normal and the hairs are themselves normal in number, density and quality (Fig. 19.5).

Alopecia can arise from a variety of primary and secondary diseases and is defined as a 'deficiency of the hair coat'. It may be due to hair growth failure or to loss of hair. There is a significant difference between hair loss involving fracture of the shaft (stumps of hair fibres remain) and hair which is shed from the follicle, i.e. when the whole hair is shed.

Congenital hypotrichosis is very rare in horses but it does occur – of course there are some disorders that are hereditary and congenital where the alopecia becomes evident at a later age. There are some circumstances such as neonatal hypothyroidism and prematurity when an apparent hypotrichosis/alopecia can be detected at birth but although these are therefore congenital they have no genetic foundation and are classified as acquired congenital disorders. Perhaps the best example of a true hereditary/genetic alopecia is the relatively common mane and tail dystrophy of the Appaloosa horse (see p. 245). Another syndrome of adult-onset facial hypotrichosis (often to the extent of complete hair loss) is recognized by clinicians but is poorly described **(Fig. CD19 • 1A and B)**. There is no known aetiology and biopsies of the skin simply identify atrophic non-functional follicles.

CD

Linear keratosis is another relatively well-recognized congenital (possibly heritable) cause of persistent alopecia. In disorders where there is a genetic reason for the alopecia the condition is most unlikely to improve and owners will need to be apprised of this.

Loss of hair without any other accompanying clinical signs such as pruritus, scaling, crusting, erosion or ulceration occurs in relatively few conditions (see Fig. 11.33). However, as these other clinical signs very frequently occur with loss of hair, they should always be considered as part of the hair loss syndrome. Acquired forms of alopecia have been divided into two groups depending on the cause and whether the hair will grow back once the condition has resolved (Stannard 2000):

Cicatricial alopecia results when the hair follicles are completely destroyed by a pathological process such as physical, chemical or thermal injuries, infections or tumours and results in a complete and permanent alopecia. A typical example is a scar from a wound or chemical burn (Fig. 19.6 and **Fig. CD19 • 2**). This same effect is sometimes used to create identification

CD

Table 19.2 Differential diagnosis of pruritus

19.2A Head & neck

	Severity	Seasonality	Alopecia	Crusting (C/c)/scaling (S/s)	Recurrent (R/r) /persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Rabies	🔴	N	N	C	N	Y	S	N(Y)	N		333
Chorioptes equi	🔴	N(Y)	N	S	R	N	M	Y	N	Housed	191
Psoroptes equi	🟢	N	N	S	P	N	M	Y	N	Ear margin hyperkeratosis	193
Trombicula autumnalis	🔵	Y	N(y)	C(s)	R	Y	M	N	N	Chalky soils/autumn	197
Dermanyssus gallinae	🟢	N	N(y)	S(c)	R	N	M	N	N	Poultry contact/stabled	199
Onchocerca cervicalis	🟢	Y(N)	N(Y)	S(C)	P	Y	M	N	Adult	No avermectin wormer	221
Werneckiella equi	⚪	N(y)	N(y)	S	P(r)	N	M	Y	N	Winter/parasites visible	215
Haematopinus asini	⚪	N(Y)	N	N	P(r)	N	M	Y	N	Winter/parasites visible	215
Habronema musca	⚪	Y	N	C	R	Y	S	N	Adult	Summer/wet season, facial skin	217
Insect worry (fleas/ticks/flies)	⚪	Y(n)	N	N	R/p	N	M	N			207
Myiasis	⚪	Y	N	N		N	S	N	N	Summer/maggots visible/odour	214
Otobius megnini (spinose ear tick)	⚪	Y	N	N	P	Y	S	N	N	Ear worry only/head-shaking	213
Non-infectious diseases											
Atopy	🔴	N(Y)	N(y)	N(Y)	P(r)	n/a	M	n/a	<2 years	Young horse (<2 years at onset)	262
(Multisystemic) eosinophilic dermatitis	🔴	N	N(Y)	C	P	n/a	M	n/a	Adult	Coronary bands affected Generalized severe	281
Insect bite hypersensitivity	🔴	Y	Y	S/C	R	n/a	M	n/a	3–7	Summer/outdoor access	284
Drug eruptions	🟢	N	Y(N)	S	R(p)	n/a	M	n/a	N	Exposure to drug/medication	288
Contact allergy/irritation	⚪	N	N	Y	?	N		n/a	N		286
Food/contact hypersensitivity	🟢	N(Y)	N	(S)	(R)	n/a	M	n/a	Adult	Food change recently	263
Paraneoplastic pruritus	🔵	N	N(Y)	C	P	n/a	M	n/a	Adult	Concurrent neoplastic disease	377
Trigeminal neuralgia/neuritis	🔵	N(Y)	N	N	P(r)	n/a	S		Adult	Face and muzzle only/ head-shaker syndrome	442
Psychogenic pruritus (self-mutilation)	🔵	N	N	N	P	N	S	n/a	Adult/stallions	Rare/idiopathic condition	442

19.2B Limbs

	Severity	Seasonality	Alopecia	Crusting (C)/ scaling (S)	Recurrent (R/r)/ persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Rabies	🔴	N	N	N	N	Y	S	Y	N	Danger	333
Chorioptes equi	🟢	N(Y)	N	S	R-P	N	M	Y	N	Housed	191
Trombicula autumnalis	🔵	Y	N	N	R	Y	M	N	N	Chalky soils	197
Dermanyssus gallinae	🟢	N	N	C	R	N	M	N	N	Poultry contact	199
Haematopinus asini	⚪	N	N	N	R	N	M	Y	N	Mild/parasites visible	215
Insect worry (fleas/ticks/flies etc.)	🟢	Y	N	N	R	Y	M	N(Y)	N	Variable causes/circumstances	215
Myiasis	⚪	Y	N(Y)	N	R	N	S	N	N	Wound or skin damage mostly	214
Strongyloides westeri/Pelodera sp.	🟢	Y	N	N	N	N	M	N	Y	Poor pasture hygiene	220
Non-infectious diseases											
Atopy	🔴	N	N(Y)	C S	R P	N	M	n/a	Y	Young horse (<2 years at onset)	262
(Multisystemic) eosinophilic dermatitis	🔴	N	N(Y)	C	P	N	M	n/a	N	Chestnuts and coronary bands affected by inflammatory changes	281
Drug eruptions	⚪	N	N(Y)	C S	P	N	M	n/a	N	Exposure to drug/medication	288
Food/contact hypersensitivity	🟢	Y	N	S	R	N	M	n/a	N	Food change recently	263
Paraneoplastic pruritus	🔵	N	N(Y)	C	P	N	M	n/a	N	Concurrent neoplastic disease	445

19.2C Body

	Severity	Seasonality	Alopecia	Crusting (C)/ scaling (S)	Recurrent (R/r)/ persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Chorioptes equi	🔵	N(Y)	N(Y)	S	P	N	M	Y	N	Housed	191
Psoroptes equi	🟢	N	N	S	P	N	M	N(Y)	N	Ear margin hyperkeratosis	193
Trombicula autumnalis	🟢	Y	N(Y)	(C)	R	Y	M	N	N	Chalky soils	197
Sarcoptes scabiei	🔴	N	N(Y)	(C)	P	N(Y)	M	N(Y)	N	Concurrent immunocompromise	195
Dermanyssus gallinae	🟢	N	N(Y)	(C)	R	N	S	N	N	Poultry contact	199
Onchocerca cervicalis	⚪	Y	Y	C	P/R	Y	M	N	Adult	No avermectin wormer	221
Werneckiella equi	⚪	N(Y)	N(y)	S	P	N	M	Y	N	Winter	215
Haematopinus asini	⚪	N(Y)	N(y)	(S)	P	N		Y	N	Winter	215
Habronema musca	⚪	Y	(Y)	C	R	Y	S	N	Adult	Summer wet seasons/fly exposure	217

(continued over)

19.2C Body (continued)

	Severity	Seasonality	Alopecia	Crusting (C)/ scaling (S)	Recurrent (R/r)/ persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Oxyuris equi	🔵	N	N	N	P	N	S	N	Adult	Perianal/perineal pruritus	224
Insect worry (fleas/ticks/flies/midges)	🟢	Y	N	N	R	N	M	N	N	Usually mild worry only	207
Myiasis	⚪	Y	N	N	n/a	N	S	N	N	Summer	214
Non-infectious diseases											
Atopy	🔴	N(Y)	N(y)	(S/C)	P/R	n/a	M	n/a	Young	Young horse (<2 years at onset)	262
(Multisystemic) eosinophilic dermatitis	🔴	N	N(Y)	(S/C)	P	n/a	M	n/a	Adult	Chestnuts and coronary bands affected by inflammatory changes	281
Insect bite hypersensitivity	🔴	Y	Y	(S/C)	R	n/a	M	n/a	>3–4 years	Progressive each year	284
Drug eruptions	🟢	N	Y(N)	(S/C)	P/R	n/a	M	n/a	N		288
Ventral midline dermatitis	🔴	Y	Y	(S/C)	R	n/a	S	n/a	Adult	Summer months/insect bite related	203
Food/contact hypersensitivity	🟢	N(Y)	N(y)	(S/C)	R	n/a	M	n/a	Adult	Known exposure to specific feed	263
Paraneoplastic pruritus	🔴	N	N(Y)	(S/C)	P	n/a	M	n/a	Adult	Concurrent signs of neoplastic disease	378
Intermammary 'intertrigo'	🔵	N	N	N	P	n/a	S	n/a	Adult	Perineal region	358
Wound inflammation/neuritis	🔵	N	N	N	P	n/a	S	n/a	N	Obvious wound/dressing	—
Psychogenic pruritus (self-mutilation)	🔵	N	N	N	P	N	S	n/a	Adult/stallions	Rare/idiopathic condition	331

19.2D No defined areas or whole body involvement

	Severity	Seasonality	Alopecia	Crusting (C/c)/scaling (S/s)	Recurrent (R/r)/ persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Chorioptes equi	🔵	Y/N	N	N	P/R	N	M	Y	N	Housed horses	191
Sarcoptes scabiei	🔴	N	Y/N	N	P	N	M	Y	Y/N	Concurrent immunocompromise	195
Dermanyssus gallinae	⚪	N	N	Y	R	N	S	N	N	Poultry contact	199
Werneckiella equi	⚪	Y	N Y)	N	P	N	M	Y	N	Winter mainly	215
Haematopinus asini	⚪	Y	N	N	P	N	M	Y	N	Winter mainly	215
Fly worry/insect bites	🟢	Y	N	N	R	N	S	N	N	Summer/outside mainly	207

19.2D No defined areas or whole body involvement

	Severity	Seasonality	Alopecia	Crusting (C/c)/scaling (S/s)	Recurrent (R/r)/ persistent (P/p)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Non-infectious diseases											
Atopy	●	N(Y)	N(y)	(c)	R	n/a	M	n/a	Y	Young horse (<2 years at onset)	262
(Multisystemic) eosinophilic dermatitis (various forms)	●	N	N(Y)	(C)	P	n/a	M	n/a	Adult	Chestnuts and coronary bands affected by inflammatory changes	281
Drug eruptions	●	N	Y(n)	S	R/P	n/a	M	n/a	Adult		288
Hepatocutaneous syndrome	●	N	N(Y)	C	P	n/a		n/a	Adult	Concurrent hepatic/neurological signs	—
Food/contact hypersensitivity	●	N(Y)	N(y)	S(c)	R	n/a	M	n/a	N	Known exposure to specific feed	263
Paraneoplastic pruritus	●	N	N(Y)	(C)	P	n/a	M	n/a	Adult	Concurrent signs of neoplastic disease	397
Psychogenic pruritus (self-mutilation)	●	N	N	N(c)	P	n/a	S	n/a	Adult/	Rare/idiopathic condition/stallions	331

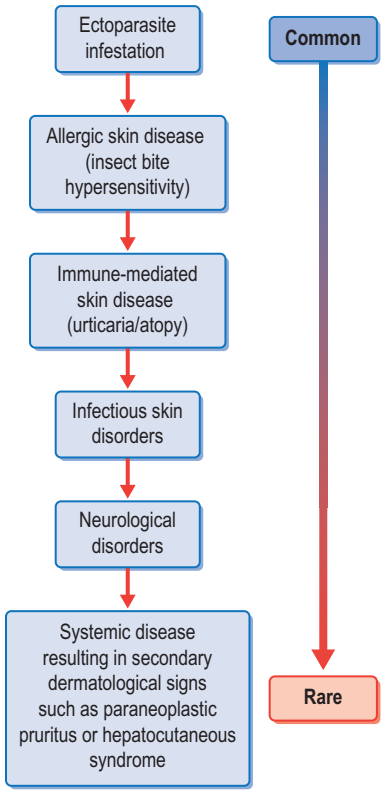


Figure 19.3 The relative prevalence of the various types of disease as a cause of pruritus in horses.

marks on horses using thermal branding. The resulting alopecia and scarring enables firm identification to take place (Fig. 19.7 and **Fig. CD19 • 3**; see also Fig. 16.1A).

Severe viral (see p. 129) or bacterial skin infections (see p. 141) and some tumour conditions such as the equine sarcoid (see p. 387) also cause localized alopecia in which there is seldom any recovery of hair growth.

In non-cicatricial alopecia the hair is not permanently destroyed and will regrow once the instigating factors have been removed. So long as the hair follicles are uninjured or at least only mildly injured hair will regrow. There are many causes of this type of hair loss in horses including:

1 Traumatic injuries that result in epidermal and/or superficial dermal damage (as a result of either direct trauma, chemical or thermal injuries or self-inflicted trauma including the effects of pruritus and cutaneous 'worry'). Even pressure applied to the skin under bandages, etc. can cause significant hair loss – sometimes with more overt epidermal or dermal damage but sometimes without these. Often there is a dramatic shedding of hair as a result of contact with urine, diarrhoeic faeces, saliva, wound exudate, etc.) (see p. 299).
2 Alterations in the function of hair follicles including several dystrophic conditions that incorporate the dramatic effluvium or gross shedding of hairs at defined stages in the growth cycle (see p. 346). Alopecia areata is also a good example of a sterile folliculitis resulting in localized or sometimes generalized alopecia (see p. 278).
3 Bacterial and fungal infections resulting in hair follicle suppression or hair shaft damage are important causes

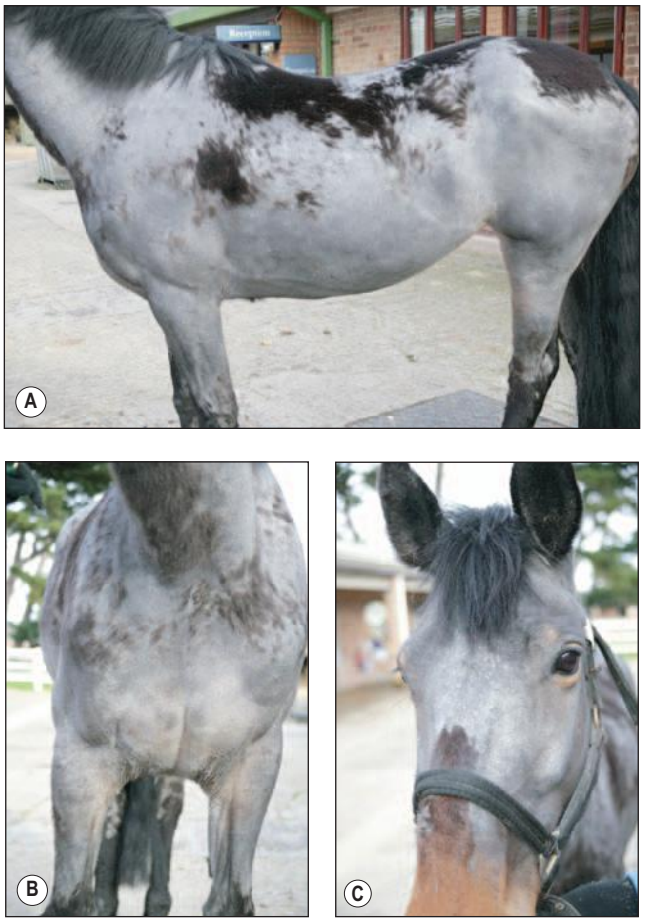


Figure 19.4 True alopecia. The hair density is very low and the hairs that are present are thin and shorter than the normal hair adjacent. **(A)** The mane and tail are largely spared. The apparently spared areas over the back and rostral areas of the face were easily depilated. Notice in this case that the hair loss is accompanied by scaling (seborrhoea) over the breast **(B)** and face **(C)** in particular – other changes are not always present. This was a case of pemphigus foliaceus.



Figure 19.5 This apparently bald patch over the breast and shoulders was easily explained by simple rubbing of a tightly fitted, heavy winter rug. This is *not* alopecia.

of alopecia – dermatophilosis, staphylococcal and streptococcal dermatitis and dermatophytosis are good examples of this type of response.

Abnormally long/dense hair coat

The hair coat quality and quantity varies widely between breeds and individuals. Most horses have a 'typical' pattern



Figure 19.6 Scarring inevitably causes adnexal damage. Where a large wound occurs on the body trunk contraction limits the size of the scar and therefore the extent of the alopecia. This scar was the result of a chemical 'burn' during preparation for surgery.



Figure 19.7 This thermal burn resulted in a scar and a patterned alopecia that allowed permanent firm identification of this animal. Note that the alopecia is variable over the damaged skin.

of hair growth with isolated whorls and increases and reductions in coat density at different sites. For example, the hair density on the side of the face is usually much less and the hairs are much shorter here than on the main body. Some horses have 'unusual' patterning of hair growth that can at first glance be misinterpreted as an abnormality. A good example is shown in Fig. 19.8, in which the lie of the hair creates an impression of abnormality. A brief examination will show that this is a normal healthy coat that has been present from birth. This is a relatively common occurrence but is very variable in extent.

There are individual horses that have hair length variations and some breeds that have longer hair coats than others. Some have isolated 'tresses' of longer hair in summer and these same sites usually have a normal length winter coat but sometimes a summer coat hair growth can be identified at these sites (Fig. 19.9).





Figure 19.8 **(A)** This Welsh pony gelding exhibited a very prominent patterning of a hair alignment over the neck and shoulder regions in particular. **(B)** Close examination showed the hair and skin were, as might be expected, totally normal.





Figure 19.9 **(A)** This pony regularly grew large numbers of isolated long 'winter-type' 'tresses' in its summer coat. **(B)** The hair was not noticeably abnormal in any way apart from its length. Although these areas were more obvious there was also a rather more diffuse, isolated distribution of long hairs over the body trunk and neck. The summer coat therefore lacked sheen/patina. In winter there was no detectable difference at these sites.

There are also some individuals that may have a very dense wavy, curly coat in winter and more normal hair coat in summer – the important point about all these animals is that they shed normally and usually have shown the same pattern of hair growth throughout their lives without any concurrent primary or secondary pathology. An interesting 'curly coat' genetic variation is being encouraged by some breed societies (see p. 233).

Sites of skin grafting can result in an abnormal hair growth pattern. The recipient site will adopt the characteristics of the donor site in terms of hair shedding and colour. The history and clinical features of these sites is easily recognized.

For all practical purposes the only disease that is characterized by *acquired* generalized hirsutism (overgrowth of hair) is pituitary pars intermedia dysfunction (PPID) (pituitary adenoma/equine Cushing's disease) (see p. 322). This symptom is almost pathognomonic for the disease in older horses and occurs in a high proportion of cases.

Investigation of equine skin with either altered density or length of hair

As is usually the case, a full history will be very helpful. Indeed, in most of the major syndromes much of the differential diagnosis can be achieved directly from this. The full range of possible aetiological states also exists for cases showing hair loss and alteration in hair density and structure







Figure 19.10 (A) This horse suffered from severe tail pulling by cattle in the same field. Once the cattle were removed the tail hair would regrow normally. This is false alopecia. **(B)** An over-tight tail bandage caused necrosis of the skin of the tail and much of its tail hair was shed. Regrowing hair was white but otherwise the individual hairs appeared normal. This is true cicatricial alopecia. **(C)** This Appaloosa gelding developed progressive thinning of his tail and mane. This is a congenital/hereditary condition and has no implication for the horse.

so it is in fact always wise to explore the history to the fullest possible extent. For example, diagnosis of a genetically based disorder occurring in a known specific breed, such as the mane and tail dystrophy condition in Appaloosa horses, can usually be assumed from the history and the physical appearance (Fig. 19.10). Of course such intuitive supposition can also be misleading. The loss of mane and tail hairs may simply be a matter of hair pulling by either another horse or even cattle (see Fig. 19.10A). Chronic ingestion of selenium (either in inorganic feed-supplement form, or in seleniferous plants and grains) may result in significant more-or-less symmetrical alopecia and hoof wall growth alterations (see Fig. 12.11). If the history includes dietary supplementation or the environmental conditions suggest that plant feeds might be involved, the process of diagnosis can be shortened dramatically. This also emphasizes the importance of understanding the local environmental conditions – are potentially toxic plants prevalent in the area and available to the horse? Has the horse received any abnormal nutrients? It is possible to draw effective diagnostic conclusions from the historical features and clinical appearance alone in most cases.

Having completed the history, a full clinical examination is important. The objective is to try to establish whether the hair loss is primary (true alopecia) or secondary (false alopecia). Skin rubbing by tack or harness or contact with walls and other solid objects can induce changes that closely resemble alopecia. Viewing the skin with an illuminated magnifying glass can usually confirm the nature of the problem. Furthermore, the correlation of the hair loss with obvious trauma is a key factor. Careful questioning can be very helpful (Table 19.3).

A few horses have a congenital (genetic/hereditary) form of alopecia – in some this is a genuine primary hypotrichosis but in others it may be a secondary result of hypothyroidism. Establishing the true primary condition may involve further diagnostic tests such as biopsy or laboratory tests such as dynamic thyroid function tests in the case of hypothyroidism.

Many skin diseases are accompanied by varying changes in the hair coat quality and where these take place the condition is secondary and so identification of the primary disease is a major objective. There are many causes of secondary alopecia relating to infections, for example – exudate, swelling and even local heat associated with inflammatory changes can cause significant alopecia. Most infectious disorders cause secondary alopecia – viral and bacterial skin infections are typical examples. For example, an abscess that develops slowly will often become 'hairless' over the region and further, more extensive alopecia may accompany the exudate when the abscess bursts or is surgically lanced. Fungal infections such as dermatophytosis (ringworm) due to *Microsporum* or *Trichophyton* spp. are somewhat different – the infections have a direct and damaging effect on the hair shaft itself.

Ectoparasites such as *Chorioptes equi* and other surface-feeding mites may chew the hair shafts and so break them off. The appearance of hair shafts in these various conditions will be notably different. If the pathogen can be seen then it is probably an academic exercise to undertake a trichogram examination but in some cases it is very helpful because there may be more than one condition present. For example, chorioptic mites often prefer sites with scaly/seborrhoeic skin and so might expect to be found at these sites in greater numbers. However, in some circumstances the pruritic nature of the disease causes plasma exudation and secondary bacterial infection and then the mites may be less detectable.

The localized loss of hair around a sarcoid (occult sarcoid – see p. 390) is another good example of the way in which the hair character can be altered by a primary disorder (Fig. 19.11).

Figure 19.12 shows the typical presentation of false alopecia.

In equine dermatology there are many alopecic disorders that are either poorly understood or not understood



Figure 19.11 This area of alopecia on the medial stifle area was an occult sarcoid. Note the characteristic and pathognomonic nodules within the alopecic circular area.

at all. Often these are the extensive hair loss syndromes. In some cases horses can be almost completely bald and still no definable aetiology can be established (see Fig. 9.13). These conditions include the hair loss that follows fever, infections or stress – the so-called telogen and anagen defluxion/effluvium are good examples of this (see p. 346). The names given to the diseases are more descriptive of the signs rather than defining the aetiology – mostly because they have no known aetiology. These names often satisfy owners but are rather unsatisfactory for scientific clinicians.

The pattern of hair loss is also an important aspect. Where extensive body hair loss occurs and the mane and tail hairs

Table 19.3 Possible questions for the investigation of hair loss in horses

1 ***Has the alopecia been present from birth?*** If so was the foal otherwise normal? Congenital alopecia is usually associated with prematurity/dysmaturity, hypothyroidism or in rare cases congenital hypotrichosis. The possibility of congenital junctional disorders such as cutaneous agenesis, epidermolysis bullosa and heritable equine regional dermal asthenia can be considered where signs are obvious.

2 ***Has the horse been ill (febrile or non-febrile systemic disease) or had access to any possible toxic materials (such as selenium, mimosa, etc.)?*** Information on previous disease states is often very important. However, often there is no known/recognized/declared episode and so the answer can be a bit misleading.

3 ***Is there any correlation between the areas affected and the previous existence of any inflammatory or infectious process?*** Horses may develop alopecia at sites that have extensive oedema or localized infections – this applies particularly to the limbs but also occurs over developing abscesses and in areas that have body fluids such as plasma, pus, urine, diarrhoea, saliva in contact with the skin.

4 ***Was the hair loss preceded by pruritus?*** Hair loss preceded by pruritus might suggest that it is a secondary state and it is worth investigating causes of pruritus and establishing the nature of the hairs by using a trichogram (see p. 62).

5 ***Did pruritus develop after the hair loss or at the same time?*** Where hair loss precedes the development of pruritus, it is often safe to assume that the hair loss is the primary event.

6 ***Has any chemical wash or unusual skin dressing been used?*** Often it is possible to detect patterns of hair loss that are associated with applications of chemicals and in some cases simply wetting the skin can encourage infection such as *Dermatophilus congolensis* (see p. 156).

7 ***Did the alopecia start at one place and spread or did it develop simultaneously over several areas?*** Centrifugal 'spread' of alopecia is highly suggestive of infection – often fungal such as dermatophytosis (see p. 168). Simultaneous development of alopecia at several sites could of course be infectious also but in the absence of any other signs this may, for example, be typical of alopecia areata (see p. 278).

8 ***Is the alopecia restricted to any defined area?*** Loss of mane and tail hair has very different implications from loss of body hair because of the significant difference in growth patterns. Many alopecic disorders spare the mane and tail but some important ones such as selenium or heavy metal toxicity have a disproportionate effect on the mane and tail and possibly the hoof also.

9 ***Is the alopecia associated with normal skin or is the skin abnormally flaky (seborrhoeic, exudative) or are there palpable variations in the skin itself?*** In the former case alopecia areata might be considered – it is the most typical and symptomless defined primary cause of focal alopecia. Exudate and other body fluids are usually obvious but the alopecia can develop well after the event so that history is important. Variations in the skin (seborrhoea, micro-nodules or obvious thickening) are invariably detected clinically simply by palpation. Equine occult or verrucose sarcoid, for example (see p. 390), and the pemphigus conditions (see p. 264) are often associated with alopecia but the concurrent evidence is usually diagnostically very significant.

10 ***Is any other in-contact animal similarly affected?*** Where more than one animal has a similar state, contagious infection or common exposure to a toxic or contact insult should be considered.

11 ***Is there any obvious explanation for the hair loss?*** Rubbing of harness, rugs and contact with walls and trailer sides are often overlooked! Similarly chewing or licking of the skin by other in-contact animals occurs with some frequency. These are clearly false (secondary) hair loss conditions and should not be classified as alopecia (Fig. 19.12). Traumatic hair loss is usually obvious. Grazes and wounds, for example, are obvious; again this is not really alopecia.

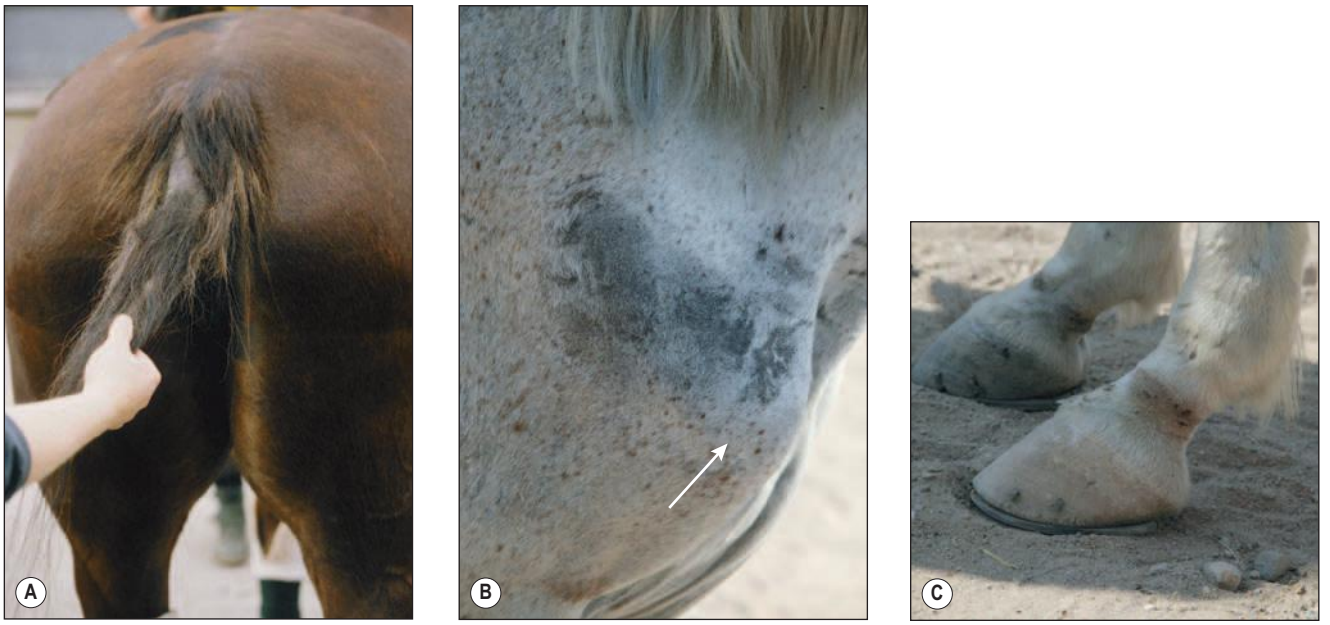


Figure 19.12 These two cases show the typical appearance of false alopecia. **(A)** The bay horse lived with 14 goats who regularly relished a few tail hairs! **(B)** The grey gelding developed an extensive rug-rub from a tightly fitting rug with a canvas underlayer that wore the hair out over most of the strong contact areas. Note that there is also an area of scarring within the area (arrow). **(C)** This horse had also been hobbled by ropes and had chronic scarring and both false and true alopecia on its pasterns.

are spared it is reasonable to suppose that there is an alteration of the growth pattern. Given that little is understood about the controlling physiological influences, endocrinopathies of varying types are often suggested even though there is no supporting haematological evidence in most of them. The commonest defined alopecic condition that has a multifocal (sometimes restricted, sometimes roughly circular and sometimes extensive) clinical appearance is alopecia areata – in this case the condition has a well-defined pathology but little or nothing is understood about its likely aetiology.

It is said that nutrition can play a major part in hair quality and quantity but this is far less obvious in horses than in some other species – even severely deprived horses often have remarkably healthy coats. However, in some cases the nutritional deficits result in significant immunological and physical compromises. Secondary infections and cutaneous injuries such as decubitus respectively may develop. Some nutritional deficiencies such as zinc deficiency may have a significant influence and the evidence for the condition of zinc-responsive dermatosis is based on the largely anecdotal reports of positive responses to zinc supplementation (see p. 327). This may in fact be far more complicated than it seems and unless a better aetiopathogenesis can be established, there will be little hard evidence for many of the disorders.

Toxicities have a better defined aetiopathogenesis than deficiencies! Selenium, heavy metals such as mercury and arsenic, and some plant poisonings (such as mimosa toxicity) cause quite severe generalized alopecia and sometimes the mane and tail are spared. Careful historical enquiry and clinical examinations may help significantly. Criteria for confirming a poisoning requires that the poison be accessible and known to be ingested. The signs should be consistent with the toxicity and the removal of the toxin should result in no further signs (although some may or may not resolve thereafter). The toxin should be demonstrable in the tissues of the animal (assuming of course that an assay is available). In some cases samples of hoof wall are the best specimens to identify previous exposure to toxic substances but this will of course depend on whether analysis facilities are available at all and if any reference data are available to identify abnormal concentrations.

Little is known about the changes that occur in hair shaft structure with the various disorders of hair quality and quantity. Electron microscopy has some potential to provide accurate information but so far no reports have been made on its value. Direct microscopic examination of plucked hairs (trichogram) could be helpful. In a few cases such as anagen defluxion/effluvium patterns of hair bulb changes can be helpful. Self-trauma can be detected by examination of hair shafts for 'broken' hairs.

Bizarre patterns of hair loss can sometimes be found. Individual horses have shown hair loss on one forelimb without any obvious cause or detectable plausible pathology. In some cases these can be attributed to some form of prior inflammatory response (Fig. 19.13 and **Fig. CD19 • 4A and B**) but in others there is no history of any swelling, inflammation, trauma or other insult. To an owner this can be very worrying and often there is a general lack of understanding of the difficulties of diagnosis. Hair loss, whether explicable or not, is often one of the most intolerable states for an owner and they will often seek all manner of (usually expensive) unqualified advice on nutritional supplements, herbs and 'remedies in bottles'. It is easy to understand their frustration but it is also an indicator that we need to know much more about hair loss and hair growth patterns in the horse.

In the final analysis, however, the owner will wish to know both the likely prognosis and whether any treatments are possible. Clearly both require evidence-based records (which unfortunately do not exist for most conditions). In spite of the clinical importance of hair loss in many defined

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Figure 19.13 This horse developed a single limb alopecia some weeks after a relatively benign cellulitis. Note the normal left fore limb. The hair took many months to regrow and was never the same as the contralateral limb.

conditions, remarkably little relevant information is available for clinical use.

The differential diagnosis of alopecia (Table 19.4)

Table 19.4 should be used to narrow down the differential diagnosis for a case presented to the clinician. For example, where pain is a feature, diseases with that option displayed positively should be considered. Where there is geographical restriction or localization of lesions on the horse, the options will narrow considerably.

Cutaneous nodules (the 'lumpy' horse)

A nodule is a solid skin swelling greater than 1 cm diameter, which exceeds the dimensions of a papule. However, there are some papular lesions that fall comfortably into the category of nodular skin disease. Nodules may be a result of hyperplasia of the epidermal or dermal tissues, amyloid or lipid deposition, inflammation or neoplastic proliferation.

Single or multiple nodules are commonly presented in equine practice and so their differentiation is important (Fig. 19.14 and **Fig. CD19 • 5A–E**). Some may originate as macules or papules but progressively increase in size, either singly or in numbers, and are then justifiably included in this category.

Nodules are usually elevated above the skin surface but may be dermal or subcutaneous; in the latter circumstances they are often easier to 'feel' than see. Some have a pathognomonic appearance whilst others are largely non-specific. Experience is an important aspect of the diagnosis because most of the neoplastic nodules have characteristic features that enable a presumptive diagnosis to be made based on clinical examination and a full history. For example, a localized single (or multiple) spherical, 0.3–1 cm diameter, black nodule under the tail of a middle-aged grey horse can reasonably be 'assumed' to be an equine cutaneous melanoma. However, even when the diagnosis is 'probable' the clinician should always remember that unusual lesions do occur from time to time.

From a pathological perspective nodules are possibly the best understood clinical entity in horses and so it is usually possible to obtain a definitive diagnosis, provided that the correct sample is taken and handled in the correct way. As might be expected, however, cutaneous neoplasms form only a few the possible diagnoses. Others are far more difficult to differentiate both clinically and pathologically. However, because most nodules are by definition reasonably well defined diagnostic biopsy is a very helpful procedure in many cases.

Tumours, nodules and swellings tend to be primary diseases in horses and are only rarely manifestations of systemic disorders. The major disease considerations in establishing a differential diagnosis are:

- infectious nodules (i.e. a result of primary infective processes, whether viral bacterial, fungal or parasitic)
- hypersensitivity/immune-mediated reactions
- sterile inflammatory disorders
- neoplasia.

Nodules can also be subdivided broadly into:

- inflammatory nodules
- non-inflammatory swellings
- neoplasia.

Inflammatory nodules

Inflammation may be due to immune/allergic responses, injury or infection and the inflammatory infiltrate will reflect the different aetiologies. The response will vary from blood and tissue fluid to more definite inflammatory exudate containing inflammatory cells. Visible nodules can then develop as the infiltrating cells attempt to demarcate the injury or the inflammatory process. As the lesion enlarges, the dermis and subcutis become completely infiltrated by cells and the overlying epidermis becomes atrophic. This leads to erosion, ulceration and possible exudation. The inflammatory nodules include urticarial wheals and abscesses (Fig. 19.15).

Abscesses are localized lesions with a fluid content of variable consistency. They are the result of inflammatory changes, usually related to trauma and/or infection, and consist of dead cells, debris and liquefied local tissue components arising through proteolytic and histolytic enzymes. While most abscesses are related to infection, they can also be sterile.

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Non-inflammatory swellings

Body swellings such as hernias and cysts can also fall into the nodular category, although strictly they do not fully meet the dermatological criteria. Some of them do however and these are relatively common in horses. Good examples are the nasal atheroma and dermoid cysts. Both of these are congenital abnormalities and both are very benign.

Some inflammatory nodules culminate in a non-inflammatory residual nodule so it is always worth making a detailed enquiry about the history and also examining for possible more recent lesions.

Table 19.4 Alopecia and hair growth alterations: differential diagnosis

19.4A Focal alopecia

	Severity	Crusting (C/c)/scaling (S/s)	Pruritus	Pain	Recurrent	Transient (T)/ persistent (P/p)	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Horse pox	○	S C	N(Y)	N(Y)	N	T	M		N	Very rare	129
Coital exanthema	○	C	(Y)	Y	N	T	M	Y	N	Genitalia/perineum	138
Viral papilloma	○	S	N	N	Y	T	M	Y	Young	Transient in young horses	135
Dermatophilosis	●	S C	N(y)	Y	(y)	T(p)	M	Y	N	Common	156
Cellulitis/abscess	●	C	N	Y	(y)	T	S(m)	N	N	Single inflammatory focus	—
Dermatophytosis	●	S C	N(Y)	N	Y(n)	T	M	Y	N	Expanding areas	168
Fungal/bacterial granuloma	●	S	N(Y)	N	N(y)	P	S(m)	N	N	Solid/solitary/progressive	176
Besnoitiosis	○	S	N	N	–	P	M	N	N	Geographically restricted	187
Onchocercosis	●	S	N	N	Y	P	M	N	N	Geographically restricted	221
Mite infestations	○	S (C)	Y	N	Y	T	M	Y	N	Identified in brushings	191
Pediculosis	○	S	Y/N	N	Y	T	M	Y	N	Visible	215
Non-infectious diseases											
Hypotrichosis	Var	N	N	N	N	P	–	n/a	Young	Diffuse hair loss/thinning	245
Epidermolysis bullosa	Var	N	N	N(Y)	N	P	S(m)	n/a	Neonate	Vesiculo-bullous disease	232
Alopecia areata	Var	N	N	N	Y	P	M	n/a	Adult	Symptom-free	278
Follicular dysgenesis	Var	N	N	N	N(y)	P	M	n/a	N	Variable regions/extent	245
Mane and tail dystrophy	○	N	N	N	N	P	S	n/a	Adult	Appaloosa parentage	245
Hereditary regional asthenia	Var	C	N	N(Y)	N(y)	P	M	n/a	Young	Quarterhorse	243
Pemphigus foliaceus	●	+++	N	N	N(y)	P	M	n/a	N	Severe/progressive	266
Sarcoidosis/granulomatous disease	●	S(C)	N(Y)	N	Y	P	M	n/a	Adult	Multisystemic	280
Multisystemic eosinophilic disease	●	C	Y	N	Y	P(T)	M	n/a	N	Pruritus is common	281
SLE-like syndrome (equine)	●	S/C	N	N	Y	P	M	n/a	Adult	Depigmentation also	276
Discoid lupus (equine)	●	C	N	Y	Y	P(t)	M	n/a	Adult	Photo-exacerbated	270
Selenosis	●	N	N	N	(Y)	T	M	n/a	N	Evidence of access/plants	307
Immune-mediated vasculitis	●	Y	N(y)	Y	(Y)	T	M	n/a	Adult	Purpura haemorrhagica	275
Leucaenosis/mimosa	●	N	N	N	(Y)	T(p)	M	n/a	N	Exposure dependent	307
Sarcoid	●	S (c)	N	N	N	P	M(S)	(Y)	N	Occult and verrucose forms	387
Melanoma	○	N	N	N	N	P	M	n/a	>10	Older grey horses *usually*	421

19.4B Generalized alopecia

	Severity	Crusting (C/c)/scaling (S/s)	Pruritus	Pain	Recurrent	Transient (T)/ persistent (P/p)	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Dermatophilosis	🟢	C	N	(Y)	R	T	M	Y	N	Purulent/crusting	156
Dermatophytosis	🔵	S	N(Y)	N	R	T	M	Y	N	Expanding areas	168
Besnoitiosis	⚪	S	N	N	P	P	M	N	N	Geographically restricted	187
Onchocercosis	🟢	S	N	N	P	P(t)	M	N	Adult	Geographically restricted	221
Mite infestations	⚪	S	Y	N	R	P	M	Y	N	Mainly winter months	191
Pediculosis (louse infestation)	🟢	S	Y/N	N	R	P	M	Y	N	Mainly winter months	215
Non-infectious diseases											
Hypotrichosis	Var	N	N	N	n/a	P	M	n/a	Young		245
Epidermolysis bullosa	Var	N(C)	N	N(Y)	n/a	P	M/S	n/a	Neonatal	Vesiculo-bullous disease	232
Alopecia areata	Var	N	N	N	N	P	M	n/a	Adult	Symptom-free	278
Follicular dysgenesis	Var	N	N	N	n/a	P	M	n/a	Young	Variable regions/extent	245
Anagen/telogen defluxion	⚪	N	N	N	N	T	M	n/a	N	Appaloosa parentage	346
Zinc-responsive dermatosis	Var	C	N	N(Y)	N	T	M	n/a	Adult	Quarterhorse	327
Pemphigus foliaceus	🔴	S	N	N	N	P	M	n/a	N	Severe/progressive	266
Sarcoidosis/granulomatous disease	🔵	S	N(y)	N	N	P	M	n/a	Adult	Multi-organ involvement	280
Multisystemic eosinophilic disease	🟢	C(s)	Y	N(y)	N	P	M	n/a	Adult	Severely pruritic	281
SLE-like syndrome (equine)	⚪	S	N(y)	N(y)	Y(N)	P	M	n/a	Adult	Multi-organ involvement	276
Immune-mediated vasculitis	⚪	C	N(y)	(Y)	N(Y)	T	M/S	n/a	N	Purpura haemorrhagica	275
Anhidrosis	🔵	S	N	N	N	P(T)	M	n/a	N	History of move	344
Hepatocutaneous syndrome	🟢	C	Y	N(y)	N	P	M	n/a	N	Evidence of liver failure	—

Table 19.4C Hirsutism*

	Severity	Seasonality	Alopecia	Crusting (C/c)/scaling (S/s)	Recurrent (R)/ persistent (P)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Non-infectious diseases											
Congenital 'curly coat'	🔵	n/a	(y)	N	P	n/a	M	n/a	Y(n)	Congenital	233
Idiosyncratic patchy coat growth	🔵	n/a	N	N	P	n/a	M	n/a	N	Variable	—
Natural winter coat patterns	🟢	Y	N(y)	N	R	n/a	n/a	n/a	N	Repeated annual changes	—
Pituitary pars intermedia dysfunction	🔴	N	N	N	P	n/a	M	n/a	Y	Older horses only (>15 years)	322

*There are no significant infectious causes of hirsutism.

Neoplastic nodules

Most neoplasms of the skin and subcutaneous tissue, at least initially, appear as nodules. Enlargement and secondary inflammatory changes impart the typical tumour appearance of an obvious enlarged nodule. The overlying dermis and epidermis may undergo significant changes including verrucose and ulcerative changes, hair loss, atrophy and necrosis. Thus the overlying surface may be normal, altered or ulcerated and the critical points to consider are the changes that were seen first and how the condition has developed. For example, a type B nodular sarcoid may present with a focus of hair loss and verrucose changes in the hair coat that closely resembles dermatophytosis. Careful palpation will, however, usually confirm the presence of the dermal nodule.

Note

The wide variety of possible diagnoses for cutaneous nodules makes the clinical investigation an important aspect of the diagnostic process.



Figure 19.14 These are typical examples of nodules on a horse's skin. The differential diagnosis is a major challenge but fortunately many of the commoner types are easily recognized pathologically. These were eosinophilic granulous with collagen necrosis.

The investigation of the horse presented with nodular skin disease

On presentation many nodules are clinically distinctive and it is common to use intuitive supposition to make a diagnosis. However, mistakes are still liable to occur unless a full history and a detailed clinical examination is made. There are many cases that have several different nodules at the same time. For example, a grey horse may have classical cutaneous melanomas whilst also having sarcoids and even cutaneous immunocytic amyloidosis. This means that the history of each lesion should be considered before a diagnosis is suggested. The history might identify that a single nodule appeared first and that thereafter many others may have developed at different sites. In the event that the signalment and clinical features were supportive, a diagnosis of sarcoid or melanoma could be made on clinical supposition alone in many cases.

As the prognosis for individual nodular skin conditions is very variable it is important to try to break down the diagnostic categories that are involved. The first major differentiation is the division between neoplastic and non-neoplastic nodules, because the implications for neoplastic disease are significantly different from those for inflammatory nodules. The clinical objective has to be to establish the

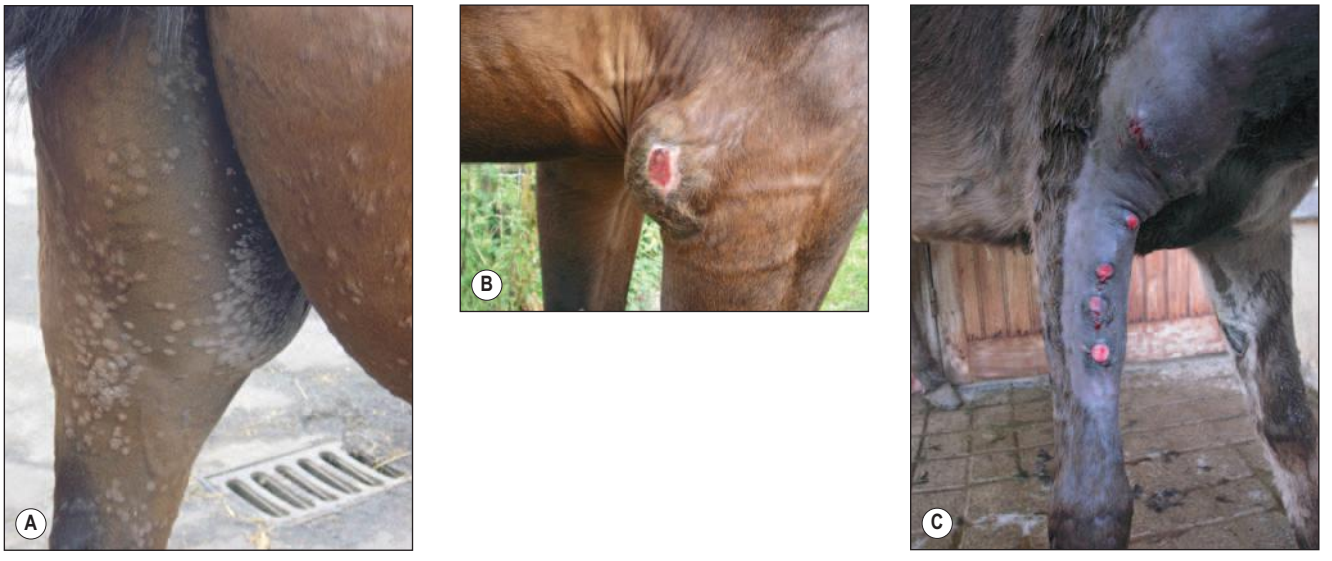


Figure 19.15 Examples of inflammatory nodules. **(A)** Papular dermatosis. **(B)** Bursitis (shoe boil). **(C)** Epizootic lymphangitis.

cell type involved and whether the nodule is a neoplasm (cancer) or an inflammatory nodule resulting from an infection of some other inflammatory process such as trauma or allergic or immunological cutaneous disorders. A diagnosis of neoplasia does not necessarily (nor even usually) mean that an aggressive cancerous condition is present – many of the cutaneous neoplastic conditions of horses have few non-cutaneous implications. However, almost all neoplastic conditions have some limitations and difficulties with treatment, even though the diagnosis is usually straightforward.

The non-neoplastic nodules can then be further subdivided into the inflammatory nodules and the non-inflammatory ones. The inflammatory nodules in turn can be subdivided into the infectious ones (viral, bacterial, fungal and parasitic lesions) and those arising from either allergic or immunological responses of which urticarial conditions are an example, and the traumatic nodules. Insect bites and stings are a common cause of transient skin nodules and in these cases a diagnosis can be achieved by careful examination of the site (usually a haemorrhagic focus can be seen in the middle of the nodule) and by the history of exposure/attack. Haematoma, abscess, false bursae and so on can all be classified as nodular skin disease.

Inevitably there are some nodules that occur in skin that have no known explanation and so this simply becomes a group of poorly understood disorders. Fortunately as more knowledge is gained, fewer cutaneous nodules fall into this category and whilst the reason for them is far from clear some are well-recognized clinical entities. An example of this is the condition known as calcinosis circumscripta (tumoral calcinosis) – this condition is a largely asymptomatic disorder of young horses, characterized by a firm plaque of dense mineralized deposits and fibrosis most often over the lateral aspects of the stifle or tarsal regions. Repeated trauma may be involved but usually this is not reported.

The physical appearance of the skin is a major clinical feature – where the skin is unaffected the diagnostic options are significantly different from those conditions in which there are concurrent skin changes or direct skin involvement. Alopecia over and around a skin nodule can be a result of pressure atrophy or of active inflammatory or neoplastic-mediator-induced changes and so the information gained by simple clinical observation and palpation can be very helpful. For example, some type B sarcoid nodules have epidermal and adnexal changes (see p. 393) while subcutaneous forms and cutaneous amyloidosis (see p. 295) have no discernible effect on the overlying skin unless and until there is an induced atrophy through pressure or vascular compromise.

The presence or absence of concurrent signs such as overt inflammation (heat, pain, swelling and if visible, redness) or pruritus will indicate particular localized responses. Inflammation can in turn be due to infection (either primary or secondary) or to other inflammatory responses such as necrosis and allergic/immunological reactions. A good example of an inflammatory nodule is an abscess – the area is swollen, hot and painful – there may be hair loss but it is rare for the region to be pruritic. Mast cell tumours may be pruritic but are seldom painful – the horse may rub or bite at the site but palpation is not resented.

Nodules that are part of a more generalized systemic disorder have a completely different implication from those that are restricted to the skin. Furthermore there may be systemic consequences of relatively benign cutaneous nodular disease. Whilst sarcoids are confined to skin, melanomas can become widely disseminated and so it is wrong to simply consider the skin condition on its own.

A thorough clinical examination and further tests, even including peritoneocentesis and rectal examination, blood samples and even radiography and ultrasonography can be useful. There is little point in attempting to treat an apparently isolated penile carcinoma when the associated iliac and inguinal lymph nodes are heavily affected by metastatic spread. Similarly a horse with melanomas may be found to have a cardiac arrhythmia if a melanoma is located in the interventricular septum – failure to examine the whole horse could lead to both an error of diagnosis and an irrelevant or unnecessary treatment process.

Close examination of an ulcerated melanoma might reveal non-pigmented foci within the lesion and this might be consistent with a more aggressive potentially or actually malignant lesion. In that circumstance a more detailed clinical examination can be justified. Grey horses with melanotic masses should probably be subjected to guttural pouch endoscopy because this is one site where the true nature of the lesions can be appreciated without any confounding external factors such as trauma.

Biopsies are commonly obtained in the investigation of nodular skin disease – most of the common nodules are well described in the literature (see **Fig. CD19 • 5A–E**) but some are rare or very rare. In the former case clinical decisions and/or treatment methods are usually available but in the latter, it is often impossible to obtain evidence-based information on prognosis and treatment methods. This also emphasizes the need for publication of case series and even individual cases of the rarer cutaneous disorders.

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As the need for treatment is very variable no presumptive statements should be made unless and until a proper assessment has been made of the whole horse. For example, a decision might be made to leave a few collagenolytic granulomas alone on the back of a horse if no others were found and they had a history of slow or no progression. Even this can be a hazard in some cases – the difficulty of predicting the lesion that will change is often not appreciated by owners (Fig. 19.16).

Leaving sarcoids alone is probably unwise as exacerbation is the norm. However, where the available therapeutic choices are limited there is no point in undertaking treatment that is contraindicated simply so that something is seen to be done. Currently melanoma is not treatable except by surgical excision but little is known about the risks of incomplete surgical excision in this condition. The application of evidence-based medicine is always the objective – in some cases the best approach cannot be taken for justifiable reasons and then the 'next-best' treatment may be used.

As in all clinical situations, it is far better to know definitely what the problem is before attempting treatment. However, many of the skin nodules are not neoplastic and differentiation of these from the cancerous conditions is possibly the first investigative objective.

Table 19.5 lists nodules likely to be found in different age groups, Table 19.6 presents a classification by clinical character, and Table 19.7 shows the differential diagnosis.

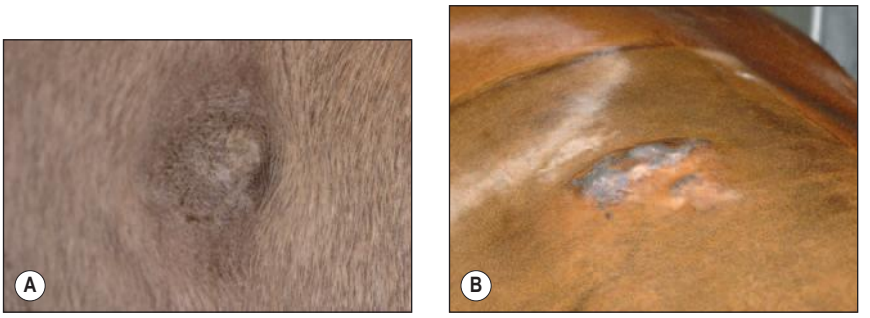


Figure 19.16 **(A)** A small collagen granuloma (around 0.5 cm diameter) had been present for some weeks before enlarging into this ill-defined, painful mass around 1.5 cm diameter. This illustrates that not all remain static or resolve. **(B)** This very extensive and complex collagen granuloma developed over a 3-week period from a lesion that had been present on the saddle region for some years without any apparent change. The possibility of a collagen granuloma was initially considered but the veterinarian decided to leave it alone on the grounds that he expected it to resolve or remain static. The owner felt that the advice given previously to leave it alone had been wrong.

Table 19.5 Nodules appearing in the various age groups

	Foals (<6 months)	Young horses (6 months to 2 years)	Mature horses (2–10 years)	Old horses (>10 years)
Congenital and developmental	Epidermal naevus Dermoid Vascular hamartoma/ (cavernous haemangioma)	Dermoid cysts Temporal odontoma Dentigerous cysts	Atheroma	
Infectious nodules	Congenital papilloma Viral papilloma Abscess	Viral papilloma Molluscum contagiosum Unilateral papular dermatosis Abscess	Viral papilloma Pinnal acanthosis Abscess Fungal/bacterial granuloma Parasitic dermal nodules (*Onchocerca cervicalis*/*Hypoderma bovis*)	
Non-infectious non-neoplastic nodules	Hernia Contact urticaria Haematoma/trauma	Urticaria Nodular panniculitis Haematoma/trauma	Urticaria Cutaneous collagenolytic granuloma, axillary nodular granuloma Erythema multiforme Haematoma/bruise – trauma	
Neoplastic nodules	Cavernous haemangioma Mastocytoma	Melanocytoma Sarcoid	Melanosarcoma Squamous papilloma Sarcoid Squamous cell carcinoma Mast cell tumour Fibroma/fibrosarcoma	Cutaneous melanoma Squamous papilloma Sarcoid Squamous cell carcinoma

Scaling and flaking (dry dermatoses/ seborrhoea) syndrome (the 'scaly' horse)

Scaling and flaking is a very common end result of skin disease of many types and aetiologies in horses; virtually all skin conditions can have degrees of flaking and scaling. Any disease which induces an excessive sloughing or an excessive production of epithelial cells is characterized by scaling. Scale is the result of desquamated keratinocyte cells and debris from hair shafts. It is not a condition of the sebaceous glands in spite of its name! Seborrhoeic conditions produce significant scale because of the higher rate of keratinocyte production/turnover or from an excessive production of squames or from a pathological lifting of the superficial layers of the epidermis.

Scale varies in appearance from fine, almost invisible, dust-like particles to extensive large, flat flakes of separated epidermis (Fig. 19.17). The consistency also varies depending upon the amount of sebum included with the scale and so can be greasy in texture or very dry and dusty. The colour of the scales varies from yellow-brown to grey-white.

Epidermal collarettes are not well recognized in horses but represent a circular accumulation of scale around a central focus. It usually reflects the historical presence of a vesicle, bulla or pustule in the superficial layers of the skin. However, many equine skin diseases have transient vesicles or other superficial dermal inflammation which result in scale formation and so scaling is a finding common to many diseases.

Scaling should be differentiated from the accumulation of dust and other environmental particles such as from hay and bedding. Microscopic examination is not always conclusive but this examination should always be performed.

Table 19.6 Nodules classified by clinical character

	Foals (<6 months)	Young horses (6 months to 2 years)	Mature horses (2–10 years)	Old horses (>10 years)
Persistent	Naevus Congenital papilloma Dermoid cysts Cavernous haemangioma	Sarcoid	Melanoma Sarcoid Mast cell tumour Collagen granuloma Melanoma	
Transient	Viral papilloma	Urticaria	Urticaria Erythema multiforme	Urticaria
Firm	Mastocytoma Melanocytoma	Sarcoid Cutaneous/generalized lymphosarcoma		
Soft/fluctuant	Atheroma Bursa	Abscess Haematoma		

The investigation of the scaling/flaking seborrhoeic dermatosis case

A full history of the horse and specifically of the condition is important. Many seborrhoeic conditions have an insidious onset and some have a defined inflammatory condition that precedes the scaling and flaking. The large majority of scaling is secondary – primary seborrhoea is very rare. In the latter cases the condition is a genetic problem and so usually manifests early in life – possibly within the first year of life. Usually the diagnosis of primary seborrhoea is made when all other possibilities have been exhausted, but it can occur, especially around the base of the mane and tail.

As many infectious, allergic and immunological disorders are characterized by flaking and scaling, it is important to establish the primary pathology and where this was located. Dermatophytosis, for example, commonly expands and spreads across the horse from a few localized primary lesions – this is entirely understandable given that grooming will spread the spores and damage the epidermis and so inoculate the infection. Contagion is a significant aspect also – a condition that spreads from horse to horse is unlikely to be an allergic or immunologically based disorder. Contagion therefore almost always means an infection (viral, bacterial, fungal or parasitic) but the impression of contagion can be given when several horses are subjected to the same insult – for example common exposure to a toxic spray or topical application.

Another important aspect of the history is the previous application of topical medications – often the extent of flaking and scaling can be dramatically increased by the repeated applications of strong (over-strength) medications whether these are designed to control infection (antiseptic washes), keratolytic shampoos or other irrational materials. The skin can respond by epidermal hyperplasia and increased flaking so that a previously trivial disorder becomes a serious one. Diagnosis can be complicated significantly and so it is vital to obtain an honest history. In addition, repeated applications of some keratolytic shampoos may reduce the diagnostic value of a biopsy; washing (and incidentally surgical scrubbing of a potential biopsy site) may remove the best evidence of disease. This is best illustrated by pemphigus foliaceus where the most (and sometimes the only) diagnostic information is to be found in the crusts and exfoliating scale. Even over-grooming can significantly increase scaling and reduce the value of biopsy. Sometimes owners are ashamed of their animals and make strenuous attempts to remove all the signs prior to a clinical examination being carried out.

Generalized conditions will have a different implication from the local scaling and flaking. For example, *Chorioptes* infestation accompanied by pruritus can result in prominent flaking and scaling in areas where the mites prefer to feed, i.e. on the distal limb regions. Generalized pemphigus foliaceus or sarcoidosis causes extensive whole body flaking, while focal infections such as dermatophytosis cause a localized inflammation-based flaking of the epidermis.

The character of the scale is possibly helpful in achieving a diagnosis. Where the scale has an excessive greasy texture (seborrhoea oleosa) it is usually indicative of bacterial or fungal skin infection (whether primary or secondary) (see Fig. 11.50). *Staphylococcus* spp. and *Malassezia* spp. infections seem more prone to inducing this change. It can be recognized by the greasy feel of the skin and a characteristic unpleasant odour. There is usually detectable diffuse inflammation. In addition, because serum exudate is often present a degree of crusting (see p. 464) is usually detectable – this is often termed seborrhoeic dermatitis. Seborrhoea sicca is characterized by a light, very dry scaling. Sometimes this can be extensive and thick but the flakes feel dry and break up into a fine dusty powder if handled – as a result the coat has a dry, dusty appearance. The scale can sometimes be seen blowing on the hairs in even a light wind.

Table 19.8 outlines the main causes of seborrhoea in horses.

Laboratory examination of the flake and scale is useful to confirm its general nature and to detect any crusting that might indicate inflammatory changes (Table 19.9). Brushings are far more likely to reveal typical changes than skin scrapings. However, a diagnosis cannot usually be achieved by this alone because the seborrhoeic changes are non-specific and common to many primary and secondary disorders of

Table 19.7 Nodules: differential diagnosis

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R/r)/ persistent (P/p)/ transient (T/t)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Viral papilloma	🔵	Y	N	N	T	N	M	Y	Y	Common in young but occasionally in adults	135
(Pinnal) viral acanthosis	🟢	N(y)	N(y)	N	P	N	S/M	N	N	Fly-transmitted papillomavirus. Also ventral midline/thighs	136
Coital exanthema	🟢	(Y)	N	N(y)	T	N	S/M	Y	N	Venereal infection	138
Abscess	🔴	N	N(y)	N	T	N	S	N	N	Painful inflammatory swelling	142
Bacterial folliculitis/granuloma	🔵	N	N(y)	N	T/P	N	S/M	N	N	Common condition	145
Botryomycosis	🔵	N	N(y)	N	T/P	N	S	N	N	Common skin nodules	148
Corynebacterium pseudotuberculosis abscess (pigeon breast)	🔵	Y	N	N	P	Y	S/M	Y	N	Fly bites are significant	142
Glanders	🔴	N	N(y)	N	P	Y	M	Y	Y	Respiratory tract signs also	161
Epizootic lymphangitis	🔴	N(y)	N(y)	N	P	Y	M	Y	N	Multiple chains of discharging nodules	179
Sporotrichosis (*Sporothrix schenckii*), streptothricosis	🔵	N(y)	N(y)	N	P	Y	M	Y	Y	Multiple discharging nodules	178
Fungal granuloma	🟢	N	N	N(y)	P	N(y)	M	N	N	Chronic – can be extensive	183
Dourine	🔵	Y	N	N	P	Y	M	Y	Y	Venereal infection	189
Microfilariasis/onchocercosis	⚪	Y	Y(n)	N	P	Y	M	N	Y	Fistulous withers	221
Insect bites/stings	🟢	Y	N	N(y)	T	N	S/M	N	N	Very common worldwide	207
Non-infectious diseases											
Naevus	🔴	N	N	N	P	N	S	n/a	Y	Congenital	242
Vascular hamartoma	🔴	N	N	N	P	N	S	n/a	Y	Congenital	237
Dermoid cyst	🔴	N	N	N	P	N	S/M	n/a	(y)	Congenital	248
Urticaria	🔵	N(Y)	N	N(y)	R/T	N	M	n/a	N	Very common/various forms; exposure dependent	254
Eosinophilic dermatitis with collagen necrosis	🟢	N(y)	N	N	P	N	M	n/a	N	Possibly insect bite related	292
Amyloidosis	🟢	N	N	N	P/r	n/a	S/M	n/a	n/a	Immunological disorder	295
Erythema multiforme	🔵	N	N(y)	N	P(T)	n/a	S	n/a	n/a		283
Shoe boil/poll evil/fistulous withers	🟢	N	N	N	T	n/a	S	n/a	n/a	*Brucella* spp./*Onchocerca* spp.	317
Haematoma	🟢	N	N	N	T	n/a	S	n/a	n/a	Trauma related	310
Foreign body reaction	⚪	N	N	(y)	T(p)	n/a	S	n/a	n/a	Variable severity/cause	—

Girth gall	○	N	N	N	P	n/a	S	n/a	n/a	Isolated at girth	291
Choristoma/hamartoma	○	N	N	N	P	n/a	S	n/a	n/a	Rare	357
Vascular haemangioma	●	N	N	N	P	n/a	S	n/a	n/a	Rare in adults	237
Melanoma	●	N	N	N	P	n/a	S/M	n/a	n/a	Grey horses very common	421
Mast cell tumour	●	N	N	N(y)	P	n/a	S	n/a	n/a	Isolated/face and limbs mainly	410
Sarcoid	●	N	Y(n)	N	P	n/a	S/M	n/a	n/a	*Very* common; various types influence treatment options	387
Neurofibroma	●	N	Y(n)	N	P	n/a	S	n/a	n/a	Eyelid mainly	408
Fibrosarcoma	●	N	N	N	P	n/a	S	n/a	n/a	Rare; sometimes malignant	407
Lymphoma	●	N	N	N	P	n/a	M	n/a	n/a	Malignancy	407
Lipoma	●	N	N	N	P	n/a	S	n/a	n/a	Rare subcutaneously	417
Haemangiosarcoma	●	N	N	N	P	n/a	S/M	n/a	n/a	Rare/malignant possibility	412
Keratoacanthoma	○	N	Y(N)	N	P	n/a	S	n/a	n/a	Very slow/benign	—
Squamous cell carcinoma	●	N	y/n	N	P	n/a	S/M	n/a	n/a	Variable malignancy/white skin	427
Basal cell carcinoma	●	N	N(Y)	N	P	n/a	S	n/a	n/a	Rare/face	409
Epithelioma	●	N	Y(n)	N	P	n/a	S	n/a	n/a	Rare isolated lesions	434

the skin; any disorder that affects cellular proliferation, and desquamation will show seborrhoeic changes. Of course if bacteria, fungi or parasites are identified the diagnosis of the primary condition *may* be established.

A skin biopsy is frequently required in addition to superficial swabs and cultures from collected debris from the skin. Biopsies are particularly helpful in some of the more serious conditions such as pemphigus foliaceus and pemphigus vulgaris because a definitive diagnosis can be established when acanthocytes and clefting of the epidermis can be detected. Of course the reliability of the biopsy is dependent on the care with which it is taken. It is particularly easy to over-prepare the skin in these cases and in so doing all the diagnostic epidermal cells are removed. The pathologist may then quite rightly report that nothing abnormal is seen. Correct biopsy technique is critical to the diagnosis of most of the scaling dermatoses (see p. 60).

Similarly bacteriological swabs can be severely affected by incorrect technique – failure to sample the right type of lesion and chemical wiping beforehand can lead to false positives and false negative cultures, respectively.

The diagnosis of seborrhoeic dermatosis is not difficult – this is simply a matter of recognizing the cutaneous evidence and making a few preliminary tests. The problem is establishing the aetiology (Table 19.10). This may take considerable diagnostic acumen and determination.

Weeping and seeping/crusting (wet) dermatoses (the 'eczematous' horse)

Weeping and seeping is the result of exudation of inflammatory or other fluids onto the skin surface. This can be a primary event but is also commonly the result of repeated wetting and maceration of the skin and surface damage, which result in a secondary exudative skin inflammation. 'Eczema' is a human clinical term that embraces a wide range of exudative dermatoses with distinctive pathological differences – it is a term that should not really be applied to equine skin disease.

Crusting is dried exudate on the surface of the skin. It will therefore vary with the character of the underlying exudate. Blood-stained exudate results in a brown or dark red crusting, while serum/plasma exudation produces yellow or pale crusts (Fig. 19.18). Crusts have a tendency to aggregate around hair shafts and so can be difficult to remove without pulling the hair out. Furthermore, attempts to remove the crusts are often painful and resented by the animal because of both the underlying condition and the removal of intact growing hairs. Gross accumulations of crusts are called 'vegetations'.

One of the commonest and most troublesome dermatological syndromes characterized by moist dermatosis is pastern dermatitis. This is recognized as a distinct clinical entity with its own differential diagnosis but it is an important clinical sign of some very significant skin and systemic diseases (see p. 471) (Fig. 19.19).

Cases of exudative dermatitis may fail to respond to any measures taken to control infection and this condition is possibly one of the best examples of how complicated a skin condition can be. A relatively small, localized area of superficial dermatitis arising from minor trauma, contact

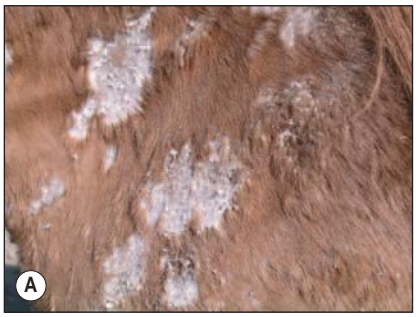




Figure 19.17 **(A)** An example of skin flakes and scale on a horse with a drug eruption. **(B)** Typical scale harvested from the same case as seen via a low power magnifying glass.

Table 19.8 The main causes of seborrhoea in horses

	Primary/secondary	Localized/generalized	Crusting	Examples
Genetic	Primary	Generalized	No	Primary seborrhoea
Congenital	Primary/secondary	Localized/generalized	Possible	Congenital papilloma Pemphigus foliaceus Hypothyroidism
Immunological	Secondary	Generalized	Yes	Pemphigus complex SLE-like syndrome Sarcoidosis Multisystemic epitheliotropic disease Anhidrosis
Allergic	Secondary	Localized/generalized	Yes/no	Drug eruptions
Nutritional	Secondary	Generalized	No	Iodism Zinc-responsive dermatosis
Chemical/toxic insults	Secondary	Localized	Yes/no	Arsenic/mercurial blisters
Neoplastic	Secondary	Localized	Yes/no	Sarcoid
Infections	Secondary	Localized	Yes	Virus papilloma Bacterial dermatitis Dermatophilosis Dermatophytosis

Table 19.9 The major clinical and microscopic differences between flake/scale and crusting

	Appearance	Texture	Microscopic
Scaling	Grey Flaky	Dry Greasy	Keratin cells
Crusting	Yellow-red/ brownish	Soft/hard and gritty	Amorphous (blood cells sometimes seen)

dermatitis, irritation or self-trauma, or continued wetting/maceration of the epidermis, may rapidly expand as the exudate and environmental conditions macerate and soften the skin, allowing bacteria to multiply. If the hair becomes crusted, ideal conditions may exist under the crusts and scab for opportunistic pathogens and primary bacterial and fungal pathogens to gain a foothold. These then establish a cycle of infection, inflammation, exudation, maceration and further inflammation and more complex infection continues. Once the skin is severely compromised it becomes extremely difficult to control the infections and limit the inflammation (Fig. 19.20).

The investigation of the wet dermatosis case

As might be expected, the clinical history is a major factor in establishing the primary disorder. Investigation of such a case requires a very careful history taking to establish the possible primary factors (Table 19.11). Detection of the primary condition might be very difficult because the presence of serum or more particularly infected serum and inflammatory exudate also causes skin inflammation and further exudation. The first clinical signs are usually the

Table 19.10 Seborrhoea (scaling and flaking/dry dermatosis): differential diagnosis

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R)/ persistent (P)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Viral papilloma	🔵	Y	Y	N	N	N	M	(Y)	Y(n)	Very common (grass/milk warts)	135
Pinnal (viral) acanthosis	🔵	N	N(y)	N	P	N	S/M	N	Adult	Common: internal pinna but also ventral midline/sheath/thighs	136
Molluscum contagiosum	🔵	N	Y	N	P	Y	M	Y	N	South Africa mainly	130
(Unilateral) papular dermatosis	🔵	N	Y	N	P	N	M	(Y)	Y	Adult horses, rare	133
Dermatophytosis	🔴	Y(n)	Y	N(y)	N	N	M	Y	N	Very common, scaling prominent	168
Malassezia	🔵	N	N	(Y)	P	N	S	N	N	Intermammary site most common	175
Besnoitiosis	🔵	N	N	N	P	Y	M	N(y)	N	Extremely rare, donkeys	187
Chorioptes equi	🟢	Y	N(y)	Y	P/R	N	M	Y	N	Housed horses mostly; carriers	191
Psoroptes equi	🟢	N	Y	Y	P	N	M	(y)	N	Ear margin hyperkeratosis	193
Trombicula autumnalis	🟢	Y	Y	Y	P	Y	M	N	N	Chalky soils; exudation	197
Sarcoptes scabiei	🔴	N	Y	Y	P	N	M	N	N	Concurrent immunocompromise; severe signs with exudate also	195
Dermanyssus gallinae	🟢	N	N	Y	R	N	M	N	N	Poultry contact	199
Werneckiella (Damalinia) equi	🟢	Y(n)	Y	Y	P/R	N	M	Y	N	Winter worse; poor management	215
Haematopinus asini	🟢	N	N	(y)	P	N	M	Y	N	Winter worse; anaemia; poor management	215
Onchocerca cervicalis	🟢	Y	Y	(n)	(R/P)	Y	M	N	Y	Adults; No avermectin wormer	221
Habronema musca	🟢	Y	Y	(y/n)	R	Y	S (M)	N	(y)	Summer wet seasons/fly exposure; adults: wounds and exudate; face/eye/ wounds	217
Insect worry (fleas/ticks/flies/midges)	🟢	Y	N	N(y)	R/P	N	M	N	N	Seasonally variable/stings and bites	207
Non-infectious diseases											
Epidermal naevus/mallenders and sallenders	🔴	N	Y(n)	N	P	n/a		n/a	Y(n)	Congenital keratinization disorder	242
Atopy	🔴	N	N	Y	P/R	n/a	M	n/a	Y	Young horse (<2 years at onset)	262
Primary seborrhoea	🔴	N	N	N	P	n/a	M	n/a	N(y)	Very rare, greasy hair/skin	232
Linear keratosis/alopecia	🔴	N	Y	N	P	n/a	S/M	n/a	Y(n)	More obvious in summer coat and after clipping	241

(continued over)

Table 19.10 (continued)

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R)/ persistent (P)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Hypothyroidism	🔵	N	Y	N	P	n/a	n/a		n/a	Congenital forms only	321
Anhidrosis	🔵									History of move to warm climate conditions	344
(Multisystemic) eosinophilic Dermatitis	🔴	N	N(y)	Y	n/a	n/a	M	n/a	N	Chestnuts and coronary bands affected by inflammatory changes	281
Insect bite hypersensitivity	🔴	Y	(Y)	Y	R	n/a	M	n/a	Y	Onset usually 4–6 years; progressive annual deterioration	284
Drug eruptions	🟢	N	Y(N)	Y(N)	P/R	n/a	M	n/a	N	Can mimic almost any skin disease!	288
Iodism	🟢	N	Y	N	P	n/a	n/a	n/a	n/a	Usually iatrogenic intake/seaweed supplements/access	328
Ventral midline dermatitis	🔴	Y	Y	Y	R	n/a	M	n/a	N	Summer months/insect bite related	284
Food/contact hypersensitivity	🟢	N(y)	Y	Y(N)	R	n/a	M	n/a	N	Known exposure to specific feed	263
Discoid lupus	🔵	N	Y	N	P	n/a	M	n/a	N	Photo-exacerbated	270
Cutaneous/SLE-like syndromes	🔵	N	Y	N	P	n/a	M	n/a	N	Severe scaling; systemic effects usually	276
Pemphigus foliaceus	🔵	N	Y	N	P	n/a	M	n/a	N	Severe scaling; systemic effects possible	266
Bullous pemphigoid	🔵	N	Y	N	P	n/a	M	n/a	N	Bulla formation/ulcers/bleeding	269
Pemphigus vulgaris	🔵	N	Y	N	P	n/a	M	n/a	N	Severe oral/cutaneous ulcers	269
Sarcoid	🔵	n/a	n/a	n/a	P	n/a	M	(Y)	N		387
Mycosis fungoides	🔵	N	Y	N	P	n/a	M	n/a	N	Lymphosarcoma related	418
Paraneoplastic pemphigus	🔵	N	Y	N	P	n/a	M	n/a	N	Serious internal neoplasia	420
Heavy metal toxicity	🔵	n/a	Y	N	P	n/a	M	n/a	n/a	Exposure related (arsenic/mercury)	309
Traumatic scar/hypertrophic scar	🔵	n/a	Y	N	P	n/a	n/a	n/a	n/a	History of injury	—
Burns/blistering	🔵	n/a	Y	N	P	n/a	n/a	n/a	n/a	Thermal burns	313
Iatrogenic chemical applications	🔵	n/a	Y	N	P	n/a	n/a	n/a	n/a	History of applications	299

most relevant – by the time the condition has been in existence for some days or weeks and the secondary changes have become established it may be impossible to identify the primary condition. In addition, the owner may well have applied some form of medication/treatment and in many cases these are ill-advised and directly harmful. Unfortunately applications that make matters worse are seldom admitted by owners – if a benefit is gained then they are usually far more willing to provide the information! This is why the history is particularly critical for wet dermatoses (eczema). Because few moist dermatoses are pruritic, enquiry as to whether the horse has shown signs of 'being itchy' is important. If the primary sign is pruritus then it is entirely possible that the eczematous dermatitis is secondary. This is a common outcome, for example, in horses affected with severe insect bite hypersensitivity – the itch precedes the excoriation dermatitis and subsequent biopsy can be grossly misleading. Some cases will rub at a primary eczematous area of skin and this applies to the head and the limbs in particular; in this circumstance the owner may justifiably have not recognized the primary focus.

Failure to recognize the primary cause will usually result in failure of treatment. The distribution of the abnormal skin is important. For example, a wet, exudative dermatosis over the dorsum of the horse might suggest that the instigating factors were related to sunlight or wetting of the skin. Dermatitis that was restricted to a blanket or harness contact sites only may suggest a primary contact allergy/reaction or an infection related to sweating maceration and lack of hygiene. Exudation from white areas only might be suggestive of actinic dermatoses, or if the lesions were restricted to the pastern of a white foot the diagnoses of dermatophilosis, pastern (and cannon) leukocytoclastic vasculitis or pastern folliculitis might be suggested (see p. 273).

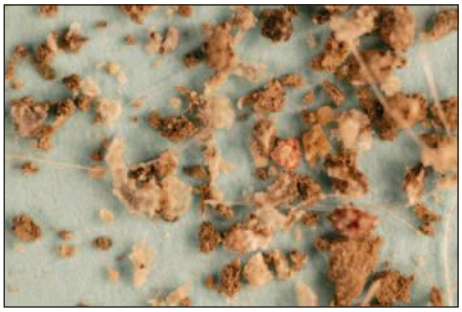


Figure 19.18 Typical crusts harvested with brushings from a horse with a severe, generalized, exudative pruritic dermatosis. Note the hard granular appearance and reddish-brown colour.

Exudation on the inside and dorsum of both hind legs of a gelding or the perineum of a mare might arise from urine scalding – the cause may be abnormal flows of urine or bladder paralysis conditions. Careful examination of the hair might reveal the accumulated crystalline deposits of calcium carbonate and the area will usually have a strong urine smell. In the same way diarrhoea commonly causes degrees of perineal inflammation in young foals and the dermatitis is accompanied by shedding of the hair in the adjacent skin. A history of diarrhoea could easily explain an exfoliative superficial perineal dermatitis in a foal or an adult horse.

The presence of pain is an important aspect of the investigation of a wet dermatosis. Few viral skin infections are painful even when they are extensive – coital exanthema is an uncomfortable condition for both mares and stallions but palpation of the lesions is not markedly resented. Often the most painful conditions are those associated with bacterial infection. Even within the bacterial dermatitis conditions there are important and helpful differences in the severity of the pain. Staphylococcal infections are notoriously painful – a horse with a staphylococcal dermatitis of the saddle (saddle



Figure 19.20 This picture illustrates the severity of the underlying dermatitis in a severe case of bacterial pastern dermatitis. The true severity can usually only be judged by the full exposure of the area of skin involved.

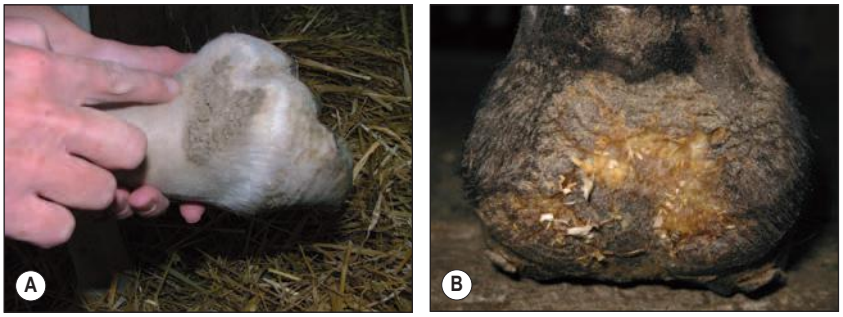


Figure 19.19 **(A)** Pastern dermatitis with adherent dry crusting. It is easy to visualize that removal of the crust might be painful and problematic. **(B)** Exudative dermatitis also results in crusting and tight matting of the hair. These two cases show pastern dermatitis but a broadly similar clinical appearance will occur at any other site to produce the same therapeutic challenges.

Table 19.11 Exudative dermatosis (eczematous/moist dermatosis): differential diagnosis

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R)/ persistent (P)/ transient (T)	Pain	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Vesicular stomatitis	🔵	N	N	N	T	Y	S	Y	N	Oral and coronet ulcers	131
Bacterial folliculitis/furunculosis	🔵	N	N	N	P/T	Y	S/M	(y)	N	Mainly *Staphylococcus* and *Streptococcus* spp.	145
Dermatophilus congolensis	🔵	Y(n)	N(y)	N	T	(y)	M	Y	N	Poor management/immunocompromise	156
Fusiformis spp. Folliculitis	🔵	N	N	N	T	Y	S/M	N	N	Distal limbs and frog regions mainly; moist conditions	—
Ulcerative lymphangitis (*C. pseudotuberculosis*)	🟢	Y	N	N	P	(Y)	M	Y	N	Fly bites may be a major factor	155
Sporotrichosis (*Sporothrix schenckii*)	🟢	y	N	N	P	N	S/M	(Y)	N	Geographical restriction	178
Pythiosis *(Pythium* spp.*)*	🔵	Y	N(y)	N/Y	P	Y	S/M	N(y)	N	Warm climates in USA	183
Chorioptes equi	🔵	Y(n)	N	Y	P	N	M	(y)	N	Housed; secondary exudation	191
Psoroptes equi	🟢	N	N	(y)	P	N	M	N(y)	N	Ear margin hyperkeratosis; secondary exudate	193
Trombicula autumnalis	🟢	Y	N(y)	Y(n)	R	N	M	N	N	Chalky soils; secondary exudate	197
Sarcoptes scabiei	🔴	N	(Y)	Y	P	N	M	N	(y)	Concurrent immunocompromise	195
Dermanyssus gallinae	🟢	N	N(y)	Y	R	N	M	N	N	Poultry contact	199
Onchocerca cervicalis	🟢	N(y)	(Y)	N(y)	P	N(y)	M	N	N	No avermectin wormer	221
Werneckiella (Damalinia) equi	🟢	N(y)	N(y)	(y)	P	N	M	Y	N	Winter	215
Haematopinus asini	🟢	N	N(y)	(y)	P	N	M	(y)	N	Winter; anaemia	215
Habronema musca	🟢	Y	Y	(y)	P/R	N(y)	S/M	N	N	Summer wet seasons/fly exposure; geographically restricted	217
Insect worry (fleas/ticks/flies/midges)	🟢	N(Y)	N	(Y/N)	R	N(y)	S/M	N	N	Fly/insect seasons	207
Myiasis	🟢	Y	N	N(y)	P/R	N(y)	S/M	N	N	Summer	214
Non-infectious diseases											
Pastern and cannon leukocytoclastic vasculitis	🔴	(Y)	Y	N	R/P	(Y)	M	n/a	Adult	White limbs; lateral aspect and hind worse than fore	273
Immune-mediated vasculitis (purpura haemorrhagica)	🔴	N	(y)	N	P	Y	M	n/a	N	History of bacterial/viral infection; ventral/limb oedema	275
(Multisystemic) eosinophilic dermatitis	🔴	N	(y)	Y	P	N/Y	M	n/a	N	Chestnuts and coronary bands affected by inflammatory changes	281

Drug eruptions	●	N	Y/N	Y/N	P/T	Y/N	M	n/a	N	Can mimic almost every skin condition; history important	288
Insect bite hypersensitivity	●	Y	Y	Y	R/T	N	M	n/a		Mane, head and tail mostly; worse when outside	284
Ventral midline dermatitis	●	Y	Y	Y	R/T	N	S	n/a	N	Summer months/insect bite related; allergic condition; worse when outside	284
Food/contact hypersensitivity	●	N(y)	N	Y/N	R/T	N	M	n/a	N	Known exposure to specific feed	263
Exudate dermatitis	●	N	N	N	T	N(y)	S	n/a	N	Urine/diarrhoea/wound exudate/saliva etc.	301
Paraneoplastic pruritus	●	N	N(y)	Y/N	P	N(y)	M	n/a	N	Concurrent signs of neoplastic disease	377
Intertrigo	●	N	N	Y	R/T/P	N	S	n/a	N	Skin rub/contact zones	358
Wound inflammation/neuritis	●							n/a		Obvious wound/dressing	—

rash) or pastern region will actively avoid attempts to palpate the area – even to the point of becoming dangerous to handle. Streptococcal infection is usually less painful and *Dermatophilus congolensis* (mud fever) infection is uncomfortable but handling is not usually resented markedly. Of course these broad statements are not invariable and there may be occasions when different responses are encountered. Furthermore many skin infections are complicated by multiple infections.

Bacterial dermatitis is seldom if ever pruritic but some horses will lick, bite or chew at the site – it is important to remember that a history of biting or chewing at a site could actually cause the breaks in the skin that result in an exudative dermatitic change and secondary infection – the critical issue then is whether the lesion existed before the pruritus or developed as result of self-trauma.

Fungal infection is seldom painful. Some forms of deep fungal infection such as pythiosis (due to *Pythium* spp. fungal organisms, see p. 177) can cause self-trauma but the circumstances where this occurs are usually well known – a preceding wound site and a progressive, expanding exudative and erosive/ulcerative dermatitis is characteristic.

One of the complications of an exudative dermatitis is the fact that the exudate is itself irritant to the surrounding skin – exudate from wounds also has the same effect. This might lead to a much more extensive area of dermatitis than occurred at the outset but again the history should enable this to be identified. Of course applied medications could be a further complication. Where and when did the first lesion appear and how has it progressed? What organisms are involved and where did they come from?

The diagnostic aids commonly used in the investigation include direct smears for qualitative microbiology, and swabs for culture and sensitivity. The presence of a particular pathogen may or may not be primary and many commensals may be involved. It is very important to realize that bacteriological culture depends totally on the absence of any applied antiseptic or antibiotic applied or delivered systemically. Even if systemic antibiotics do not reach their minimum inhibitory concentration in the site, cultural results will be altered significantly. Swabs must always be taken before any interference from typical sites within the area but this may also be misleading if the whole area is covered with commensal or opportunistic bacteria. It is often helpful to take repeated swabs during the clipping and cleaning process to maximize the reliability of the results. In some cases the culture swabs can be taken from squeezed skin – this will possibly yield a more meaningful result if only one set of swabs can be taken.

Biopsy of acutely inflamed skin can help significantly where there are pathognomonic signs such as in pemphigus vulgaris, but for the most part the secondary complications arise quickly and so even this can be confusing. Swabs taken from biopsy sites or from the deeper layers of the biopsy specimen may be significant because they are possibly less likely to be contaminated by secondary bacteria or fungal infections.

Alteration in pigmentation

Cutaneous pigmentation is the result of interaction between melanocytes and the keratinocytes and hair bulb

cells. Melanocytes are present in many tissues but the highest concentrations are found in the skin (dermis, hair follicles), mucous membranes, eye and meninges. Epidermal melanocytes are usually found in the basal cell layer and supply melanin to the keratinocytes. Melanin pigment is produced from tyrosine under the influence of the enzyme tyrosinase in the presence of copper (this being one possible explanation for poor pigmentation of skin and hair). This interaction occurs within melanosomes within the melanocyte and once fully laden with melanin the pigment is transferred to the keratinocytes in the dermis and epidermis.

CD

Two possibilities exist for acquired variations in colour of the skin and the hair.

CD

1 *Hyperpigmentation* occurs when the skin (melanoderma) or hair (melanotrichia) becomes darker as a result primarily of increased melanin incorporation within the keratinocytes. Hyperpigmentation is caused by an increased amount of melanin in the epidermis and/or the dermis. It is uncommon in horses as for the most part they already have darkly pigmented skin. However, where stimuli from excessive friction or trauma from harness occurs, hyperpigmentation or melanotrichia (conversion of natural body/hair colour to black) can occur (see Fig. 16.12). This change is commonly seen in other species also but is far less recognized in horses. There are also a few conditions characterized by darkening of the skin including some mild inflammatory conditions such as hyperaesthetic melanotrichia and scarring. Hot/thermal branding results in hair loss, scarring and commonly darker pigmentation of the damaged skin area.

CD

2 The other possibility is the loss of melanin/melanocytes and a lightening of the skin (leukoderma) or hair (leukotrichia) colour.

Both of these possibilities can occur in defined smaller areas or more extensively. Both can arise spontaneously but pigmentary changes involving changes from body colour to non-pigmented (white) are far more common than those featuring darkening/blackening of the skin. Furthermore, hyperpigmentation is more often the result of obvious insult than loss of pigmentation, which can arise spontaneously for a number of reasons. However, abnormal darkening of the skin without reason does occur in a macular form, particularly around the groin, mouth and lips of ageing horses, when it is known as lentigo.

Melanocytes in the epidermis and in the hair bulbs are frequently affected independently of each other. Where the melanocytes in the epidermis are affected, the skin loses pigment and the condition is referred to as leukoderma. Where pigment is lost from the hair bulb, it leads to white hairs or leukotrichia. The independent relationship between hair and skin is easily demonstrated by shaving the area and noting the discrepancy between the location of the original white hairs and underlying white-skinned area.

The appearance of white hairs or pink skin patches/spots on a previously whole-coloured horse often causes owners serious distress, and while in some cases an explanation such as a focus of trauma (or even an injection site) can be offered and a diagnosis easily reached, there are others which are difficult to identify and many are impossible to explain with our present state of knowledge. Some conditions such as spotted leukotrichia (see p. 337) are well recognized but others are less easily understood.

Some forms of hair pigment alteration are natural/non-pathological. For example, an old horse may 'turn grey' on the face in particular; this is an almost inevitable change in older dun or brown horses **(Fig. CD19 • 6A and B)**.

It is also well known that grey horses become 'whiter' with age. In these animals the hair becomes less pigmented but the skin remains black. A 25-year-old Lipizzaner horse is a dramatic, startling 'white' colour whereas at birth the animal was probably black in colour and during its younger years was probably 'iron grey'. This phenomenon is accepted as normal but is still poorly understood – the fact that it is an almost inevitable event in all grey horses almost certainly means that it is a genetically based growth pattern change **(Fig. CD19 • 7A and B)**. The other circumstance when a normal change occurs is when a foal is born brown or chestnut and turns grey over the first 12 months of its life. At birth there is often no indication of the potential changes and so descriptions made for identification purposes can seem to be ludicrously wrong! This change does not affect regions that are white-skinned – these will have white hairs no matter whether the body colour is grey or some other colour **(Fig. CD19 • 8A and B)**.

Loss of pigmentation in the hair and/or skin over varying areas and in varying locations can occur without apparent reason. There are some well-recognized 'idiopathic' changes that develop without any apparent insult – for example a condition known as spotted leukotrichia (see p. 337) and the vitiligo disorders appear for no apparent reason. The loss of hair and loss of pigment in the skin of the face and perineum are well recognized changes in Arabian horses – so much so that the condition is given its own name 'pinky Arab syndrome' (see p. 247).

Changes can also occur with skin insults of all types. Viral and bacterial skin infections such as coital exanthema (EHV-3 infection), viral papilloma or pyoderma, and other non-infectious inflammatory conditions such as equine lupus erythematosus-like syndrome of horses or discoid lupus can cause a reduction in numbers and function of the melanocytes and so the sites of the skin changes are marked by white skin or hair or both. Exposure to chemicals or even just skin contact with rubber can cause loss of melanocyte function (see p. 339). This type of change is very poorly understood – the extent of the insult seems trivial and yet the consequences can be dramatic and persistent. Even short-duration application of over-tight bandaging of the forelimbs or pressure from a girth or saddle can cause pigmentary changes, often without any obvious wound (see Fig. 16.8); changes are more understandable when there is concurrent cutaneous damage. This might mean that melanocytes are really quite a sensitive cell type, reacting adversely to a number of trivial insults. The loss of pigmentation can arise from either a loss of melanocyte numbers (destructive conditions) or a reduction in their ability to synthesize or release melanin.

Patterns of white hairs can be the result of iatrogenic ('therapeutic') thermal burns ('firing' procedures). As melanocytes are known to be far more sensitive to the effects of gamma and beta radiation and respond far more to laser beams, pigment loss in radiated or laser surgical sites is entirely understandable.

The sensitivity of melanocytes to cold is exploited during freeze branding of horses for identification purposes. If the

branding is undertaken lightly the hair colour is changed but there is no apparent effect on the structure and function of the skin as a whole; the hair remains normal in density and character apart from the change in pigment. On grey horses such a change would be 'invisible' and so in these cases, the freeze is stronger and longer and then there is hair loss and whitening of the skin so that the markings are obvious.

Usually when any changes to white occur they are permanent – restoration of hair colour is not currently possible in any of the known conditions. Supplementation with copper in feed and various herbs are also advocated but all these have no material benefit.

The investigation of the horse with pigmentary changes

As always a thorough history of the horse is essential. This will include aspects of its genetic status and breeding because there are significant and common disorders that are genetically based. Known or suspected genetic causes of 'abnormal' pigment loss (as opposed to the normal colour variations in the skin/hair coat colours responsible for the markings of an individual horse) include pinky Arab syndrome (see p. 247), lethal white syndrome (see p. 234), lavender foal syndrome (see p. 238) and albinism. Apart from the pinky Arab syndrome these conditions are usually obvious from birth and in some cases the skin colour is a good marker for the more serious internal dysfunctions that characterize the conditions.

There are several important and relatively common conditions in which pigmented hair and/or skin becomes white for no apparent reason, although a genetic aspect has to be more than a possibility. The conditions known as 'spotted leukotrichia' and 'vitiligo' have no known cause but the distribution of the lesions and their benign, almost asymptomatic nature makes the diagnosis relatively simple (Fig. 19.21). Of course an owner of an affected horse may not be convinced by the idiopathic nature of the condition because the cosmetic changes are often dramatic.

Aspects of the clinical distribution of the pigmentary changes are possibly the most important of all when taken with the presence or absence of any obvious inciting cause. Thus, an animal that had a history of radiation treatment for a sarcoid around its eye may expect to have white hairs at the site. Patterns of change may correspond with obvious contacts – for example, contact with some rubbers and some other chemicals used on the harness may result in a 'harness pattern' of hair colour change (often without concurrent alopecia) (see Fig. 16.8; p. 339). Where this occurs of course the cause can be proposed with some confidence although the mechanisms behind the change in melanocyte function is often far less obvious.

The signalment of the horse will matter because some of the conditions are the preserve of older horses or younger animals while others have no age predilection. The colour of the horse may be significant for some defined conditions but generally not for pathological or physiological pigmentary changes. As little is known about the relationships between colour and many of the changes that occur, this is simply of interest at this time. The Arabian breed has a known pigmentary disorder (pinky Arab syndrome) so horses with either pure Arabian blood or their crosses may be affected. The features of the disease mean that a diagnosis can usually be assumed when there are focal/coalescing areas of facial and/or perineal pigmentary loss with or without alopecia.

The conditions known as hyperaesthetic leukotrichia and reticulated leukotrichia (see p. 342) are possibly the most pathological – the signs include pain and a progressive change in hair colour. The patterns of the disorder are quite distinctive but again little is understood about the conditions.

The implication of pigmentary changes on the face and around the eyelids in particular is that the affected skin is probably more liable to sunburn/actinic dermatitis and, anecdotally at least, to squamous cell carcinoma.

Biopsy of the affected regions may reveal significant pathology but it may be very subtle also; the changes can simply reflect a reduction in the number and density of melanocytes in the skin or the hair follicles. Most of the conditions have remarkably little pathology and so a skilled dermatopathologist is helpful. In some cases the initial biopsy findings can be missed and so when this occurs the histological findings should be reviewed.

Treatment of hair coat and skin colour change is not usually possible – changes tend to be persistent. Restoring the colour with tattoo and with protective skin cosmetics is palliative and temporary. Fortunately the prognosis for most of the conditions is excellent and apart from the cosmetic aspects and the extra tendency to sunburn on pale skin the horse has little problem.

See Table 19.12 for the differential diagnosis of pigmentary changes.

Pastern dermatitis syndrome

The syndrome of pastern dermatitis is one of the commonest skin disorders in clinical practice and possibly one of the most frustrating. It includes primary diseases of the coronet and pastern (and sometimes the cannon region) alone but may be related to or linked to disease processes elsewhere on the horse. Combined, they represent a set of

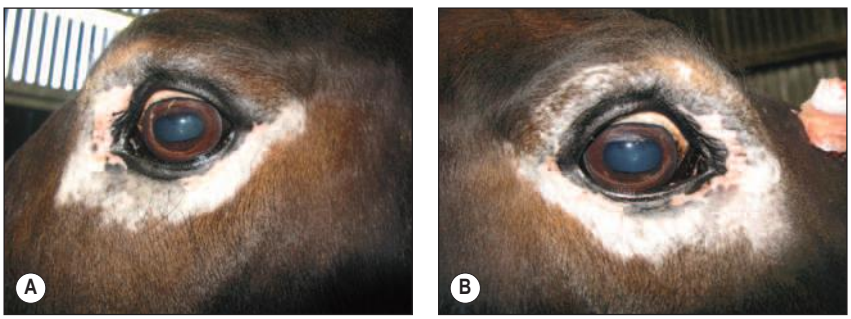


Figure 19.21 (A, B) Bilateral vitiligo in a 14-year-old Shire mare. The owner learned to 'hide' the changes when 'on show'!

Table 19.12 Pigmentary changes: differential diagnosis

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R/r)/ persistent (P)/ transient (T)	Primary (P)/ secondary (S)	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Virus papilloma	○	Y	Y	N	T	P	M	Y	Y	Adults sometimes	135
Coital exanthema	●	Y	N	(y)	T	P	S	Y	N	Venereal mainly	138
Pinnal acanthosis	○	N(y)	Y(n)	N	P	P	M	N	Adult	Common; also ventral midline	136
Onchocerca cervicalis	○	N(y)	Y(n)	y(n)	P/T	P	S/M	N	Adult	Geographically restricted	221
Non-infectious diseases											
Vitiligo	●	N	N(y)	N	P	P	S/M	n/a	N	Thoroughbred/Arabian/Shire	336
Pinky Arab syndrome	●	N	N(y)	N	P	P		n/a	N	Arabian/Arabian cross horses	247
Fading Appaloosa syndrome	●	N	N	N	P	P		n/a	N		246
Atopy	○	N(y)	N(y)	Y	R(P)	S	M	n/a	Y(n)	Young horse (<2 years at onset)	262
(Multisystemic) eosinophilic dermatitis	○	N	N(Y)	Y	P	S	M	n/a	N	Chestnuts and coronary bands affected by inflammatory changes	281
Insect bite hypersensitivity	○	Y	N(Y)	Y	R	S	M	n/a	Adult	Common; summer months	284
Drug eruptions	●	N	Y/N	N/Y	R/T	P	M	n/a	Adult	Can mimic almost all skin diseases	288
(Cutaneous) SLE-like syndrome	○	N	N(y)	N(y)	P	P	M	n/a	Adult	Summer months/insect bite related	276
Discoid lupus	○	N	Y	N	R/P	P	M	n/a	Adult	Photo-exacerbated	270
Food/contact hypersensitivity	●	N(y)	N(y)	N	T	P	M	n/a	Adult	Known exposure to specific feed	337
Paraneoplastic pruritus	○	N	N(y)	Y	P		M	n/a	N	Concurrent signs of neoplastic disease	377
(Hyperaesthetic) leukotrichia	●	N	N	Y	P	P	M/S	n/a	Adult	Vaccine related? *Pain*	342
Reticulated leukotrichia	●	N	N	N	P	P	M	n/a	Adult	Genetic origin possible	341
Spotted leukotrichia	●	N	N	N	P(r)	P	M	n/a	Adult	Spots may vary seasonally	337
Scar/pressure marks	●	N	Y(n)	N	P	S	S	n/a	N	Used in brand/freeze marking also	337
Radiation	●	N	N	N	P	S	S	n/a	N	Site exposure (therapy)	337
Age-related changes	○	N	N	N	P	P	S/M	n/a	Y	Old age greying is common	337

diseases which are both difficult to manage and difficult to explain to owners when they fail to respond to medication. Neglected cases are a common cause of cruelty investigations and prosecutions for cruelty.

Although there is no apparent (detectable or reported) reason for it, the condition is far more prevalent in white-skinned legs than in coloured ones. This fact is widely known by owners who therefore, somewhat perversely, assume that the condition is always the same. Inflammation of the pastern skin is clearly only a cutaneous reaction pattern in many skin conditions, both infectious and non-infectious. It is also true that many milder conditions become progressively more serious with secondary infection, exudation and skin maceration (derived either from exudate, poor conditions or from inappropriate care).

Possibly the most serious form of the disease occurs in heavy horse breeds such as the Clydesdale and Shire (Ferraro 2001). In these this complex condition is thought to include degrees of an immune-mediated vasculitis possibly with some genetic predisposition to local immunological dysregulation (English & Pollen 1995). However, as an example of how difficult the condition can be to understand, these two breeds are also far more prone to chorioptic mange and the long feathered legs mean that chronic self-trauma and lack of skin hygiene are common. By the time the condition is recognized, there is usually significant secondary self-trauma, infection and skin exudation with maceration. Often the secondary skin changes are so chronic and so severe that the original cause cannot be established. The resulting proliferative 'masses' can easily be mistaken for tumours and granulation tissue **(Fig. CD19 • 9A–C)**.

Proximity to the ground adds significant challenges to the skin of the lower limb due to changing conditions (from dry stables to unhygienic stables, from normal paddock to wet boggy conditions, from dry, sandy, solid to clay etc.). The environmental conditions do seem to play a major part in the overall syndrome. Most cases (but certainly not all) are presented in winter months in out-grazed horses.

Almost every case will have had a great array of 'ointments', 'salves' and 'cures' applied before veterinary attention is sought in response to irrational and often grossly harmful medications that are advertised in the lay press. It is little wonder that some horses develop serious lameness and unremitting chronic dermatoses in which a primary caused has long since been lost in the complexity of mismanagement and neglect.

It is often difficult to identify the primary problem and treat it satisfactorily. Some lower limb problems are found as part of a general disease pattern such as dermatophilosis, occasionally dermatophytosis and less commonly vasculitis. Interestingly these are not often restricted to white-haired limbs (Fig. 19.22). Others such as pastern and cannon leukocytoclastic vasculitis (see p. 273) have, as yet, unidentified causes and require further investigation. Many of the latter diseases can justifiably be included in the 'greasy heel syndrome'.

Investigation of a horse with pastern dermatitis

The signalment of the horse is a critical issue here because there are some breed-related conditions but the management is possibly the most significant single factor. Even the genetic tendency of the Shire and Clydesdale to chronic verrucose pastern dermatitis can be much influenced by management. Recognition of the tendency to the condition in the individual horse enables the carer to take suitable preventive measures at an early stage. By the time the skin is hyperplastic, thickened, exudative and has exophytic 'grapes', treatment is usually palliative (see p. 354). This difficult condition was, for example, a major scourge of draught horses in military units until the importance of early attention was recognized. Thereafter the condition virtually disappeared from army horses; it still remains in neglected draught horses maintained under poor hygiene conditions. Of course the reluctance of owners to clip out the feathered limbs is a major factor in the pathogenesis and progression of the disease.

One of the major problems faced by the clinician is the willingness of many owners to attempt ill-advised (often over-strength and harsh) treatment at the early and most diagnostic stage. By the time inappropriate medications have been applied for some weeks the original cause is almost certainly either irrelevant or is masked by other changes. The other problem arises because of the concept that all cases of pastern dermatitis are caused by *Dermatophilus congolensis*. Whilst this infection is certainly common, it is by no means the only cause of pastern dermatitis. Many cases appear to be secondary *D. congolensis* infections and many of the other conditions are clinically similar (Fig. 19.23). Lay advice often assumes the condition 'mud fever' is a defined entity but it is in reality a highly complex condition with several possible primary causes and many secondary complicating factors including other infections (Table 19.13).

CD

Having obtained a full history, clinical examination should be carried out carefully. The distribution of the inflammatory changes is important. Restriction to white skin is a well-recognized phenomenon in many conditions despite the lack of any known predisposing anatomic or physiological reason for this. The extent of pain associated with the condition is also a significant factor. Staphylococcal infection in particular causes a very painful dermatitis. Other infections and non-infectious conditions tend to be much less painful.

It is often impossible to examine the skin properly without clipping it out – sometimes this alone can be very difficult and sedation may be required. It is important to take superficial bacterial swabs and impression smears before any attempt to clean the leg because the significant crusting and scale could be removed by this.

Often clipping is impossible because of the matted and crusted hairs and ulcerated painful cracks in the skin. Under these conditions the affected limb can be covered with a debriding antibacterial cream with a hydrogel added under a light bandage for 24 hours. On removal the crusts will usually be softened and are then far easier to remove. Clipping is usually much less resented under these conditions. Soaking the legs in an antibacterial shampoo or surgical scrub can be useful but this is not nearly so effective.

Once the skin is cleaned and clipped the full extent of the lesion can be appreciated.

See Table 19.14 for the differential diagnosis of pastern dermatitis.

Coronary band disease (coronitis syndrome)

The coronary band is the main generative tissue for the hoof capsule. Its connections to the pedal bone involve the delicate and exquisitely constructed laminal attachments.



Figure 19.22 This Welsh cross pony mare presented with generalized dermatophytosis – the condition extended to the pastern and cannon regions. Note that the colour of the skin is not a significant factor in this condition. **(A, B)** The limbs before clipping and cleaning. **(C–E)** The severity of the infection. However, it resolved rapidly.

The growth of the hoof wall from the coronary band downwards takes place at roughly 1 cm per month. The whole weight of the horse is suspended by the laminal attachments during all stages of weight-bearing – during equal foot load the pressure will be minimal but will still be significant. This weight tends to drive the pedal bone into the foot and so the laminal attachments act as a strong suspension bridge between the wall and the pedal bone. At no stage in the normal foot during weight-bearing is this load removed. During this process the laminal attachments have to maintain their attachment; in order to allow natural growth there have to be exquisitely controlled release and reattaching processes of the hoof to the laminae. As soon as there are any inflammatory changes (usually termed laminitis) there will be a degree of scarring of the attachments and this then either fails to release (and therefore reattachment is not relevant) or only releases partially. This is reflected in alteration in the appearance of the hoof capsule. Rings, distortions and growth retardation are common features of laminitic diseases.

The coronary band is a particularly interesting anatomical feature in the horse. It sometimes provides important information on underlying systemic or generalized disease processes. Thus several autoimmune disorders are manifest by inflammation or changes in the function of the coronary band. In some cases the condition may then only become



Figure 19.23 This series of pictures is included to illustrate some of the possible conditions that can manifest with pastern dermatitis. The name pastern dermatitis is not a disease in itself – it is a symptom of another condition even when the pastern is the only structure involved. Important aspects of the investigation relate to the localization in the pastern regions, the restriction to white-haired regions and the presence or absence of pain or other signs and whether there are systemic signs associated with the disease. **(A)** Part of the multisystemic eosinophilic epitheliotropic disease syndrome (see p. 281). **(B)** Staphylococcal dermatitis/folliculitis (see p. 150). **(C)** Dermatophilosis (see p. 156). **(D)** Cutaneous/systemic lupus erythematosus-like syndrome (see p. 276). **(E)** Pemphigus foliaceus (see p. 266). **(F)** (Photo-exacerbated) pastern and cannon leukocytoclastic vasculitis (see p. 273). **(G)** Verrucose pastern dermatitis (chronic '*Chorioptes*') syndrome (see p. 191). **(H)** Photosensitization (hepatic failure related) (see p. 303). **(I)** Idiopathic pastern dermatitis (see p. 350). **(J)** Streptococcal folliculitis (see p. 145).

Table 19.13 Primary and secondary causes of pastern dermatitis[a]

Type	Basis	Disease	Primary[a]	Secondary
Infections	Viral	Viral papilloma	✓	
		Horse pox	✓	
		Vaccinia	✓	
	Bacterial	*Dermatophilus congolensis*	✓	✓
		Staphylococcus spp.	✓	✓
		Streptococcus spp.	✓	✓
		Fusobacterium necrophorum	✓	✓
	Fungal	Dermatophytosis	✓	✓
		Malassezia spp.		✓
	Parasitic	Chorioptic mange mites	✓	
		Poultry mites	✓	
		Forage/harvest mites	✓	
		Myiasis		✓
		Strongyloides westeri	✓	
		Pelodera spp. helminths	✓	
Immune/allergic disorders		Pemphigus/coronitis	✓	
		Systemic/cutaneous lupus erythematosus-like syndrome	✓	
		Vasculitis	✓	✓
		Eosinophilic exfoliative dermatitis/stomatitis	✓	
		Multisystemic eosinophilic epitheliotropic syndrome	✓	
		Eosinophilic coronitis stomatitis syndrome	✓	
		Verrucose pastern dermatitis	(✓)[b]	✓
Traumatic disorders		External trauma	✓	
		Self-trauma		✓
Contact (hypersensitivity)		Accidental/incidental	✓	✓
		Iatrogenic	✓	✓
Chemical/contact		Urine/exudates scalding	✓	✓
		Iatrogenic 'medication'	✓	
Actinic dermatitis		Plant/chemical toxicity	✓	✓
		Hepatic failure		✓

[a]The primary conditions are the main target for diagnostic investigation and curative therapy. While the secondary conditions can be very significant, the underlying primary disease should be identified.
[b]Verrucose pastern dermatitis of heavy/draught or feathered horses is possibly a primary disease but very little is known about it.

manifest when the hoof wall shows abnormal growth patterns. Traumatic damage to the coronary band almost always affects the hoof growth in that region. Distorted or altered hoof growth or in some cases a complete failure of hoof wall production may have serious implications for the horse. It is important to realize that similar tissues are present around the ergots and chestnuts and so whenever coronary band disease is suspected these structures should also be examined carefully.

The syndrome of coronary band inflammation is often one of the most frustrating conditions in equine dermatology (Fig. 19.24). This arises for several important reasons. Firstly the coronary band is subjected to considerable challenge from the environment and secondly the hoof quality depends heavily on a normal functioning coronary band and when the hoof quality is severely abnormal a secondary changes can be encountered in the coronary band tissues. The coronet is also a very obvious structure to most

Table 19.14 Pastern dermatitis: differential diagnosis

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R)/ persistent (P)/ transient (T)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Viral papilloma	○	N(y)	N(y)	N	T	N	S/M	N	N(y)	Skin trauma/other sites also	135
Vesicular stomatitis	○	Y	N	N	T	Y	M		N(y)	Oral/lip vesicles	131
Dermatophilosis	●	Y(n)	Y	N(y)	T/R	N	S/M	Y	N	Scabbing/exudate; mildly painful	156
Pyogranuloma/botryomycosis	●	N	N(y)	N	P	N	S	N	N	Proliferative/granulation	148
Pastern folliculitis – *Staphylococcus* spp., etc.	●	N	N(y)	N	P/R/T	N	S/M	N	N	Severe pain/adherent scabs	153
Fusiformis coronitis	●	N(y)	N	N	T	N	S/M	N(y)	N	Dorsal hind limb regions mainly	362
Dermatophytosis/microsporosis/trichophytosis	●	N	Y	N(y)	T	N	S/M	Y	N	Typical expanding lesions; other sites involved also	168
Mycetoma	●	N	N	N(y)	P	N(y)	S	N	N	Chronic non-healing ulcerated site	178
Pythiosis	●	N(Y)	N(y)	N/Y	P(T)	Y	S/M	N(y)	N	USA only	177
Chorioptic mange	○	Y(n)	N	Y	P/R/T	N	M	Y	N	Worse in winter/housed	191
Sarcoptic mange	●	N	N(Y)	Y	P	N	M	N(Y)	N	Immunocompromise	195
Strongyloides westeri/*Pelodera* spp.	○	Y(n)	N	Y	T	N	M	N	Y	Foals/unhygienic conditions	220
Trombiculidiasis	●	Y	N(y)	Y	T	Y	M	N	N	Autumn/chalky soils	197
Non-infectious diseases											
Coronary band dystrophy	○	N	N	N	P	n/a	S	n/a	Y(n)	Chronic proliferative changes; may develop late	362
Scalding (urinary/faecal/exudate)	●	N	N(y)	N	T	n/a	S	n/a	n/a	Variable severity	313
Quittor	●	N	N	N	T(P)	n/a	S	n/a	n/a	Focal discharging sinus tract	372
Wire injury/rope gall	●	N	N(y)	N	T	n/a	S	n/a	n/a		—
Over-treatment with harsh medication	●	N	Y(n)	N(y)	T	n/a	S	n/a	n/a		—
Sarcoid	●	N	Y(n)	N	P	n/a	S/M	n/a	n/a	Fibroblastic/wound site related	387
Squamous cell carcinoma	●	N	N(y)	N	P	n/a	S	n/a	n/a	Rarely affects skin of distal limb	427
Coronitis complex	●	N	N	N(y)	P	n/a	S	n/a	n/a	Idiopathic coronitis	362
Vasculitis (purpura haemorrhagica)	●	N	Y(n)	N	T(P)	n/a	M	n/a	n/a	Previous respiratory tract signs	275
Pastern leukocytoclastic vasculitis	●	Y	Y(n)	N	T(P/R)	n/a	S	n/a	n/a	Lateral aspects of white hind limbs worse affected/photo-exacerbated	273

(continued over)

Table 19.14 (continued)

	Severity	Seasonality	Alopecia	Pruritus	Recurrent (R)/ persistent (P)/ transient (T)	Geographic restriction	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Pemphigus foliaceus	●	N	Y	N	P	n/a	M	n/a	n/a	Generalized condition	266
Eosinophilic (epitheliotropic; exfoliative) dermatitis/ stomatitis syndrome	●	N(y)	Y(n)	Y	P	n/a	M	n/a	n/a	Severe pruritus is cardinal sign; oral lesions also	281
Selenosis	●	N	N	N	T/P	n/a	M	n/a	n/a	Access to selenium source (coronitis)	307
Actinic dermatosis/photosensitization	●	Y	Y(n)	N(y)	T/P/R	n/a	M	n/a	n/a	Sunlight exposure related; hepatic failure/plant poisoning	303
Verrucose pastern dermatitis 'greasy heel' syndrome)	●	Y	N	N	P(T)	n/a	M	n/a	n/a	Winter months worse; Possibly immune-related; grapes formed on skin; secondary infection/myiasis	354
Idiopathic pastern dermatitis	●	N	N(y)	N(y)	P/T	n/a	S	n/a	n/a	Unexplained and difficult	350

owners and so abnormalities should be noticed quickly. However, this is often not the case and cases are presented most often in a chronic state that might even not have been noted by the owner at all.

When the hoof wall is patently abnormal there are several diagnostic aspects to consider.

1 The coronary band may be inherently (genetically) abnormal such as occurs in some of the larger breeds of horse such as the Shire and the Clydesdale. In these cases there is a progressive thickening of the coronet and a progressive deterioration of the hoof wall quality resulting in cracking (vertical and horizontal), a lack of a healthy periople and deformed hoof wall formation. Usually no amount of medication or local management will have a material effect on the progression in these cases but most manage adequately for many years. The major difficulty here is with farriery and shoe retention; nails may be unstable and quickly come loose. Separation of the white line and flaring can be serious secondary effects.
2 The condition could arise as a result of a metabolic disorder resulting in a generalized failure of keratin production. In this case the changes in all the hooves will be similar and the whole hoof wall will be equally affected on each foot. Syndromes that result in poor hoof wall quality include those that involve hoof wall formation – such as biotin, methionine and sulphur deficiencies. The rationale for assumption that the primary disorder is a vitamin/mineral deficiency is not based on science but the fact that many cases will benefit from supplementation of these materials. Quite commonly a definite demarcation is seen that corresponds to some definitive change in management and where this is recognized it is worth obtaining a really thorough history.
3 The coronary band is a very active dermatological tissue with a significant blood supply and is subject to both specific and generalized disease conditions. These include the autoimmune, neoplastic (Fig. 19.25) and some inflammatory conditions.
4 The coronary band is subject to trauma and to secondary damage when the laminal attachments are disrupted within the foot in laminitic cases. Some of the primary and secondary causes of laminitis can also affect the coronary band directly (e.g. selenium toxicosis).

Clinical investigation of coronary band disorders

Typically with any case involving a skin disorder a full history and a detailed clinical examination are essential. This is the more important because several of the major coronary band disorders can be eliminated simply by establishing whether there are other tissues involved as well. Thus, the possibility of a diagnosis of the rare eosinophilic exfoliative dermatitis/stomatitis syndrome can be supported (but not necessarily proved) by examining the mucosae of the mouth in particular. This condition can affect young horses and if there are dental eruption processes going on simultaneously the diagnosis can be a bit misleading (Fig. 19.26).

It is important to get some indication of the progression or otherwise, and the duration of the condition is an important

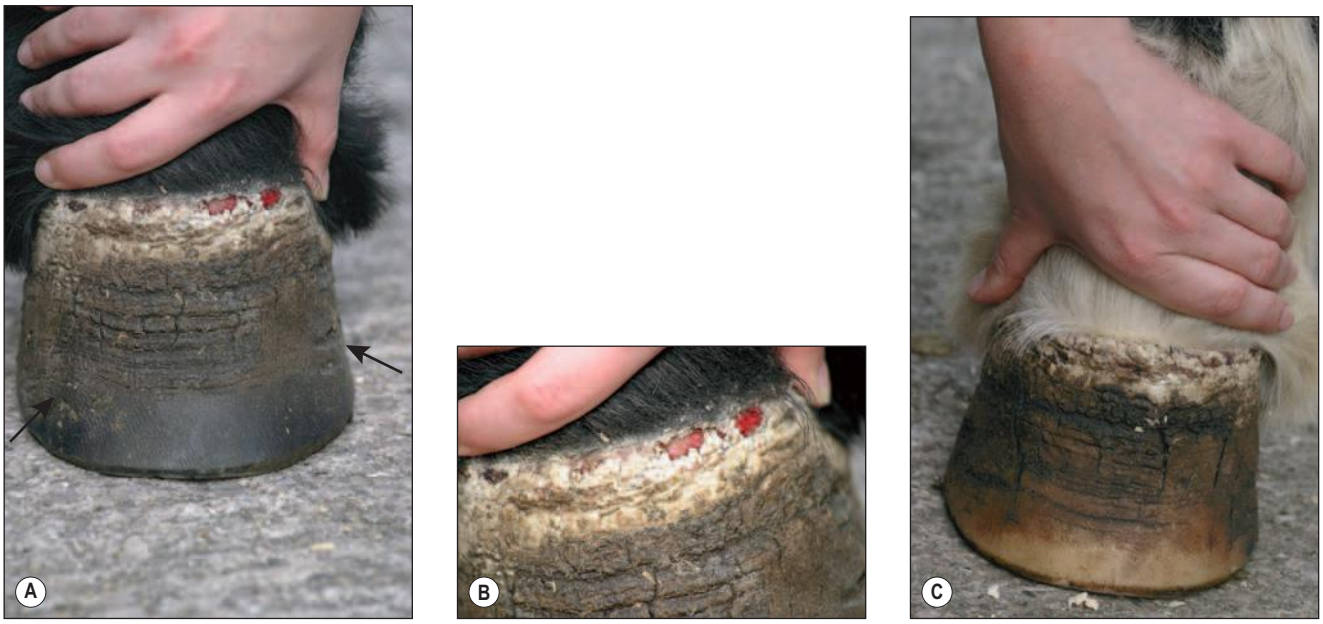


Figure 19.24 (A, B) These pictures show the variations in immune-mediated coronitis due to pemphigus foliaceus. This horse had major symptoms over its whole body and the coronary band lesions were obvious but rather insignificant. Note the extent of coronary band disruption and the consequent alteration in the hoof quality. Although the rate of hoof growth is variable, it is possible to correlate the onset of the condition with the hoof growth by identifying where the hoof was normally produced (arrows). In this case there was an acute onset of signs some 8 months previously. Note also that the toe tends to be more severely affected than the quarters. **(C)** This case of immune-mediated coronitis involved *only* the coronary bands and the ergots and chestnuts (see p. 362). The hoof changes are very similar, as might be expected, but there is no generalized skin symptom.



Figure 19.25 A diffuse mixed sarcoid involving the pastern skin and the coronary band. Notice the deforming effect this has on the hoof wall formation. The periople and the hoof wall structure are both severely affected. The successful treatment of this case resulted in resolution of the hoof wall problem but it did take some 12 months.

aspect of diagnosis. It is possible to get some idea of the duration from examination of the hoof capsule. Long-standing coronary band inflammatory and non-inflammatory disease will almost inevitably have a detectable effect on horn and periople production. In this way it is possible to make some estimate of duration, progression and severity.

Biopsy of the coronary band is highly problematic; any major disruption can quickly result in a defect of the hoof wall formation and instability of the hoof capsule with possibly permanent hoof cracks. Therefore it is better to biopsy the heel region when a full thickness biopsy is required. Where the condition only involves the more dorsal or quarter coronet, shave biopsies may give useful information; these are always likely to be less satisfactory, however. Given the chronic and intractable nature of most of the conditions, a proper diagnostic biopsy will be far better in most cases than repeated inadequate attempts. Nevertheless, little is known about the pathology of the coronet in horses and so even then, a meaningful diagnosis may not be achieved.

Conditions that affect the equine coronary band are shown in Table 19.15. The differential diagnosis of coronitis is shown in Table 19.16.

Hoof syndromes

The outer structures of the foot are of dermal origin. Although they are commonly grouped together and then may be regarded as a unique structure, the conditions that affect them are often of dermatological relevance. The hoof can be divided for practical dermatological purposes into:

- the coronet (see above)
- the hoof wall
- the sole (including the frog and digital cushion).

In spite of the common origin of the tissues, the pathological conditions are significantly different for each of these structural parts. However, their health or otherwise is

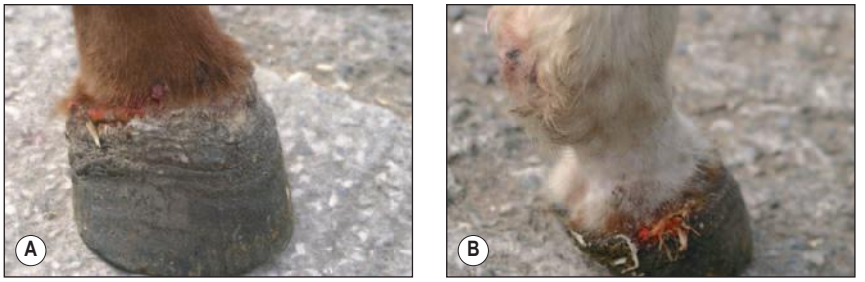


Figure 19.26 This 3-year-old horse was presented with an inflamed, painful coronitis of all four feet. **(A)** The margins of the chestnuts and ergots **(B)** were similarly affected and the chestnuts were notably thin and severely affected. Small gingival and buccal ulcers were recognized in the mouth but initially these were attributed to 'teething' changes. There were no mucocutaneous junctional changes and on biopsy neither bullae nor apoptotic keratinocytes (acanthocytes) were identified. Histopathology confirmed the animal was suffering from the eosinophilic exfoliative dermatitis/stomatitis syndrome. This is possibly a variant of multicentric eosinophilic epitheliotropic disease (see p. 281).

Table 19.15 Primary and secondary causes of coronary band disease

Type	Basis	Disease	Primary	Secondary	CB only
Infections	Viral	Viral papilloma	✓		No
		Vesicular stomatitis	✓		
	Bacterial	*Dermatophilus congolensis*		✓	
		Staphylococcus spp.		✓	
		Streptococcus spp.		✓	
	Fungal	Dermatophytosis	✓	✓	
		Malassezia spp.		✓	
	Parasitic	Chorioptic mange mites	✓		No
		Poultry mites	✓		
		Forage/harvest mites	✓		
		Myiasis		✓	
		Strongyloides westeri	✓		
		Pelodera spp. helminths	✓		
Genetic		Coronary band dystrophy	✓		Yes
Immune/allergic disorders		Pemphigus/coronitis	✓		No/yes
		Systemic lupus erythematosus-like syndrome	✓		No
		Vasculitis	✓	✓	No
		Eosinophilic exfoliative dermatitis/stomatitis	✓		No
		Sarcoidosis			No
Traumatic disorders		External trauma	✓		Yes
		Self-trauma		✓	No
Contact hypersensitivity			✓	✓	No
Toxicity		Ergotism	✓		No
		Selenium toxicosis	✓	✓	No
		Blister beetle toxicosis	✓		No
		Fescue toxicosis	✓		No
Iatrogenic damage		Blister (counterirritant application	✓	✓	No
		Bandage/boot trauma	✓	✓	Yes/no
Neoplastic disorders		Sarcoid	✓		No
		Squamous cell carcinoma	✓		No
		Keratoma	✓		No

Table 19.16 Coronitis: differential diagnosis

	Severity	Pain	Pruritus	Alopecia	Recurrent (T)/ persistent (P)/ transient (T)	Hoof wall involvement	Single (S)/ multiple (M) focus	Contagion	Age restriction	Special features	Page
Infections											
Viral papilloma (+pastern + cannon)	○	N	N	N(y)	T	N	M	N	Y	Young grazing horses, skin trauma	135
Dermatophilosis (+pastern + cannon)	●	N(y)	N	Y	T/R	N	M	Y	N	Multiple focus/poor hygiene/winter 'mud fever'	156
Staphylococcal dermatitis (+pastern + cannon)	●	Y	N	N(y)	T	N	S/M	N	N	*Very* painful Adherent crusting	150
Dermatophytosis (+pastern + cannon)	●	N	N(y)	Y	T	N	S/M	Y	N	Circular alopecia; lesions elsewhere also	168
Chorioptes equi	●	N	Y	N	R/T	N	S/M	Y	N	Winter worse; easily identified	191
Dermanyssus gallinae	●	N	Y	N(y)	T	N	S/M	N	N	Poultry contact	199
Trombicula autumnalis	●	N	Y	N(y)	T	N	S/M	N	N	Chalky soils/autumn	197
Strongyloides westeri (+pastern + cannon)	○	N	Y	N	T	N	S/M	N	Y	Young horses, poor hygiene conditions	220
Pelodera spp. (+pastern + cannon)	○	N	Y	N	T	N	S/M	N	Y		220
Non-infectious diseases											
Coronary band dystrophy of heavy/draught breeds	●	N	N	N	P	(y)	M	n/a	Adult	Thickened/scaling	362
Over-treatment ('greasy heel')	●	N(y)	Y/N	N/Y	T	N	S	n/a	N	History of applications	—
Acquired fibrous dystrophy of the peri-coronary tissues	●	N	N	N	P	Y	S	n/a	N	Idiopathic change/possibly trauma related	362
Vitamin A deficiency	●	Y	N	N	N/Y	Y/N	M	n/a	N	Extremely rare – almost impossible!	362
Pemphigus foliaceus Pemphigus vulgaris	●	N	N	Y	P	N/Y	M	n/a	N	Multifocal lesions	266 269
Verrucose pastern dermatitis (+ pastern + cannon)	●	N(y)	N(y)	N	P	N	M	n/a	Adult	Chronic 'grapes'/exudate	354
Generalized granulomatous disease (sarcoidosis)	●	N	N	N/Y	P	N(y)	M	n/a	Adult	Generalized disease	280
Eosinophilic exfoliative dermatitis/stomatitis (+chestnuts/mouth)	●	N(y)	N(y)	N/Y	P	N(y)	M	n/a	Adult	Multifocal Pruritus commonly present	281
Sarcoid verrucose fibroblastic	●	N	N	Y	P	Y/N	M	n/a	N	Other similar lesions	387
Keratoma (+hoof/sole)	●	N(y)	N/Y	N	P	Y	S	n/a	Adult	More related to hoof wall	436

intimately interrelated. For example, conditions affecting the coronet such as coronary band dysplasia or pemphigus foliaceus can cause significant pathology in the hoof wall. Wall abnormalities can result directly in sole disorders; for example, a keratoma originating high in the wall will cause significant distortion of the wall and the sole. A solar penetration can manifest as a discharging sinus at the coronary band but this could easily be mistaken for lateral cartilage necrosis (quittor).

CD

The general health of the horse and its management can have a significant bearing on the condition of the hoof. Certain cracks in the hoof wall may be due to management factors or genetic factors or both **(Fig. CD19 • 10)**. The health of the keratin-producing tissues and the periople (the thin waterproof outer layer of the hoof wall) is strongly dependent on the health of the animal and the health of the foot. Errors or changes in nutritional status or the health of the horn-producing tissue will soon reflect in obvious changes in the hoof wall. Laminitic episodes provide perhaps the most obvious changes to hoof growth; the 'laminitic rings' are characteristic but not pathognomonic (see Fig. 17.15). Accurate diagnosis of the primary underlying problem is essential if effective therapeutic measures are to be taken.

The formation and maintenance of the sole is somewhat different from that of the wall and so repair mechanisms are also different. The sole has a complex arrangement of tissues and is subjected to direct concussive axial forces that are not duplicated in any other tissue. There are few tissues that have to withstand the focal forces created, for example, when a horse stands on a sharp stone. Furthermore the sole is in contact with much organic matter – often heavily contaminated with bacteria – and so damage is often accompanied by infection. The natural elastic nature of the frog and digital cushion also adds some complicating factors – tracts left by foreign bodies such as nails may close tightly after removal of the object and the tract may be almost invisible. The consequences of this can be very serious. Treatment should take account of the need to protect the integrity of the sole to avoid the introduction of foreign matter and infection. Trauma, including penetration of the sole, is common and the interrelationship with the underlying synovial and tendon structures is vitally important. Even minor sole disorders can have life-threatening implications.

Damage to the wall of the hoof can arise from failure of normal production of horn or from direct trauma. The close proximity of the horn to the sensitive laminae of the hoof means that damage is likely to cause lameness. Healing relies heavily on repair originating in the coronary band; growth is usually slow even in the healthy foot and so repair of hoof disorders is likely to be protracted and suitable supportive measures may need to be taken to permit the tissues to heal at all. Both the sole and the wall are subjected to significant pressure forces at every weight-bearing step. The frog and underlying digital cushion are compressed and the heels are forced outward. Under normal conditions the natural flexibility of the structures permits this without any damage or stress. Outward flexion of the heels is a major natural shock-absorbing mechanism. When the hoof capsule is damaged by trauma or disease this movement can be detrimental to the healing process. The slow repair of hoof wall disorders means that special attention must be paid both to the primary aetiology of the condition and to any secondary consequences on the hoof itself. Treatment of wall disease therefore has an important temporal aspect and so early diagnosis is essential.

The cutaneous hoof structures are also liable to some specific neoplastic changes. Some of these are significant challenges to the veterinary surgeon but most are benign and if correctly diagnosed and managed carry a good or reasonable prognosis. A good outcome following treatment of these conditions is very satisfying but a good understanding of the anatomical relationships and appropriate aftercare is vital if this is to be achieved.

Inevitably some disorders of the hoof have profound implications for the welfare and health of the animal itself. The use of the horse may be severely restricted if damage or disease of the hoof affects its ability to work safely and soundly. The structures underneath the hoof capsule are critical. The interrelationship between the coronary band, the hoof capsule and the sole, on the one hand, and the soft tissues and the synovial structures and tendons of the foot, on the other, make an accurate and early diagnosis particularly important. Failure to address the primary problem can lead to frustrating failures and possibly even a fatal outcome. The value of a skilled farrier when dealing with hoof disorders cannot be overstated.

Investigating the hoof wall disease case

A thorough history of the horse and a detailed history of the presenting hoof wall changes are vital. Variations in hoof wall quality may reflect changes at the coronet but also may be associated with congenitally poor feet or with environmental insults and trauma. Certain breeds of horse such as the Thoroughbred have a greater reputation for soft hoof wall and soles and these animals may be difficult to shoe correctly and may cast their shoes regularly. Break-back and deformity is of course a major issue in these. Others such as the Shire have a reputation for brittle feet, and cracks and roughening of the outer surface of the hoof are common. These aspects are seriously confused and complicated by management and environmental aspects. Horses maintained on a firm rocky surface in dry hot conditions such as the ponies in Saharan Africa or the wild horses of the USA often have very hard feet whilst those in wetter, cooler and less harsh underfoot conditions often have softer feet. Remarkably, the more natural the conditions, the more normal and strong the feet tend to become. Many of the pathological conditions can be improved significantly by restoring a natural way of life.

In some cases some impression of the time scale can be gained by clinical assessment but it is important in any case to correlate these with known periods of foot problems. Because the hoof wall grows slowly and is very visible, its character can reflect generalized disease as well as localized ones.

Where all four feet are presented with very similar changes it is safe to assume either that the condition was generalized (including metabolic and inflammatory conditions) or that the feet have been subjected to an identical local insult. However, the problem is in fact slightly complicated by the differences in weight-bearing between the front and back feet. In the standing horse the front feet together bear around 66% of the weight of the horse. The hind legs bear significantly less weight and the changes may be less obvious here especially when the condition originates in the laminal tissues.

CD

Normal growth of the hoof wall that is not addressed by regular and correct farrier care can result in physical damage **(Fig. CD19 • 10A)**. Even minor nutritional changes often reflect in a hoof wall band pattern **(Fig. CD19 • 10B)**. This can be a normal event but in some cases horizontal cracks can occur **(Fig. CD19 • 10C)**. Careful questioning and detailed examination can be very helpful.

Laminitis has a profound effect on the hoof wall quality and the rate and pattern of growth (Fig. 19.27). The natural growth of the horn from the coronet downwards is an extremely delicate process requiring coordinated release and attachment of the laminae. Focal damage or more widespread disruption of these attachments results in scarring and an abnormal hoof wall growth and this is reflected in distortion of the hoof wall and particularly in the presence of diverging 'laminitic' rings and differential growth between the heel regions and the toes so that 'Turkish slippering' occurs.

The solar structures are often overlooked but they are clearly critical to the normal function of the horse. It is very important to properly examine all the feet from all angles when investigating hoof wall problems. Further investigations such as radiography or even magnetic resonance or computed tomography imaging can help significantly. The primary objective is to establish whether the hoof problems are primary or secondary.

Careful cooperative effort with a good farrier is usually a significant aid to the diagnosis of hoof wall problems. It is usually possible to establish the progression of any particular problem because the farrier will usually have prior knowledge of the horse's feet.

Biopsy of the hoof wall is extremely difficult and so little is known about the structures in normality and in disease that meaningful results are seldom achieved. However, without continued encouragement the situation is likely to remain and so specimens of abnormal hoof should be submitted for pathology.

See Table 19.17 for differential diagnosis of the various hoof disorders.





Figure 19.27 **(A)** The changes following laminitic episodes (no matter how caused) are usually detectable in the early stages by careful examination alone. **(B)** In extreme cases it becomes very difficult to establish whether the trigger for abnormal growth was laminitis or simply neglect.

Table 19.17 Hoof disorders: differential diagnosis

Table 19.17A Coronary band

Infections			Non-infectious conditions		
Disease	**Lameness severity**	**Confirmatory test**	**Disease**	**Lameness severity**	**Confirmatory test**
Viral papilloma	Nil	Biopsy	Coronary band dystrophy	Nil	Biopsy at heel
Vesicular stomatitis	Mild	Biopsy/viral isolation	Idiopathic coronitis	Mild	Biopsy (non-specific)
Dermatophilosis	Mild	Culture/smear	Wire cuts/trauma	Variable	History
Staphylococcal infection	Mild	Culture/biopsy	Over-reach	Variable	History
Fusiformis coronitis	Moderate	Culture/treatment	Heel haematoma	Moderate	Examination
Dermatophytosis	Nil	Rarely affects coronet	Quittor – lateral cartilage	Mild	Surgical exploration
Mycetoma	Mild	Biopsy	Pemphigus complex	Nil	Biopsy other sites
Chorioptic mange	Nil	Brushings	Eosinophilic coronitis/stomatitis syndrome	Mild	Biopsy
Dermanyssus gallinae	Nil		Sarcoidosis	Nil	Biopsy other sites
Trombicula autumnalis	Nil		Biotin/methionine deficiency	Nil	Supplementation
			Vitamin A deficiency	Nil	Supplementation
			Sarcoid(verrucose/fibroblastic)	Nil	Biopsy

Table 19.17B Hoof wall

Infections			Non-infectious conditions		
Disease	**Lameness severity**	**Confirmatory test**	**Disease**	**Lameness severity**	**Confirmatory test**
Vesicular stomatitis	Mild	Serology/virus isolation	Genetically thin walls	Mild	Persistent difficulty
Wall abscess	Severe	Paring of site			
Canker	Moderate	Concurrent hoof wall defects Culture/biopsy	White line disease/seedy toe	Moderate	Obvious deformity/ hollowing in white line at toe/radiography/ MRI
			Permanent coronary band injury – wall defect	Mild	History and obvious defect
			Cracks (quarter/heel/toe)	Variable	Visual
			Nail bind, bad shoeing	Moderate	History
			Low heels – crushed or torn laminae (low heels)	Mild	Visual and radiography
			Rolled under wall at heels	Nil	Visual
			Coronet injury	Variable	Visual
			Wall break-back	Variable	Visual
			Biotin/methionine deficiency	Nil	
			Nutritional changes	Nil	Wall rings
			Laminitis	Severe	Stance, laminitis, heat and digital pulses etc. Radiography/MRI
			Selenium poisoning	Severe	History
			Mimosine, selenosis poisoning	Moderate	History of access
			Changes to work or feeding	Mild	History
			Cushing's disease	Moderate	Hirsutism/PU/PD
			Keratoma	Mild	Radiography/MRI

Table 19.17C Sole

Infections			Non-infectious conditions		
Disease	**Lameness severity**	**Confirmatory test**	**Disease**	**Lameness severity**	**Confirmatory test**
Gravel/white line disease	Moderate	Discharge from coronet	Bruise	Mild	Obvious solar bruising
			Corns	Mild	Bruising at seat of corn
			Sole crack	Mild	Visible
			Penetrating wound – street nail	Severe	History/radiography/ MRI
Sole abscess	Severe	Foot paring/ radiography	Laminitis	Severe	Heat/pain/reluctance to move/stance and radiography
Penetrating wound – street nail	Severe	History	Thin sole, flat feet	Mild	Direct examination

(continued over)

Table 19.17C (continued)

Infections			Non-infectious conditions		
Disease	**Lameness severity**	**Confirmatory test**	**Disease**	**Lameness severity**	**Confirmatory test**
Canker	Moderate	Foul smell and destruction	Keratoma	Mild	Examination/ radiography/MRI
Thrush	Mild	Black tarry fetid discharge	Fibroma	Mild	Biopsy
Under-run sole	Moderate	Paring sole	Dropped sole, bruising	Mild	Visible changes

Table 19.17D Frog/digital cushion

Infections			Non-infectious conditions		
Disease	**Lameness severity**	**Confirmatory test**	**Disease**	**Lameness severity**	**Confirmatory test**
Penetrating wound – street nail Penetration of navicular complex	Severe	Radiography/MRI/CT	Physical injury Tearing/street nail	Variable	History/radiography/ synoviocentesis/MRI
Thrush	Mild	Black tarry fetid material in cleft of frog	Fibroma	Mild	Biopsy
Canker	Moderate	Destructive infection			

References

Blood D C, Studdert V P 1988 Baillière's comprehensive veterinary dictionary. Baillière Tindall, London

English M, Pollen S 1995 Pastern dermatitis and unguilysis in two draft horses. Equine Practice 17:25–27

Ferraro G L 2001 Pastern dermatitis in Shires and Clydesdales. Journal of Equine Veterinary Science 21:524–526

Scott D W, Miller W H 2003 Diagnostic methods. In: Equine dermatology. WB Saunders, Philadelphia, pp 64–66

Stannard A A 2000 Alopecia in the horse – an overview. Veterinary Dermatology 11:191–203

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