Ovine Meat Inspection - 2nd Edition

Anatomy, physiology and disease conditions

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A Grist

Nottingham
University Press

Nottingham University Press
Manor Farm, Main Street, Thrumpton
Nottingham NG11 0AX, United Kingdom
www.nup.com

NOTTINGHAM

First published 2010

British Library Cataloguing in Publication Data
Ovine Meat Inspection - 2nd Edition
Anatomy, physiology and disease conditions:
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ISBN 978-1-907284-76-2

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Typeset by Nottingham University Press, Nottingham
Printed and bound by Latimer Trend & Co Ltd, Plymouth

INTRODUCTION

This book forms part of a series that has been designed to provide a study aid for students undertaking the Royal Society for the Promotion of Health Red Meat Inspection Certificate and other courses where knowledge of ovine meat inspection is required such as those for Environmental Health Officers and Official Veterinary Surgeons and students undertaking Veterinary Public Health courses.

It is hoped that it may also provide information in a 'user friendly' format for abattoir owners and producers as to the nature of rejections recorded by inspectors.

Wherever possible, photographs illustrating anatomical features and conditions have been included, the latter generally depicting severe examples of conditions. This is a conscious decision, arrived at after giving numerous lectures on meat inspection where I learnt that no two people would describe something the same way.

The section covering anatomy and physiology does not go into great detail; numerous other texts fulfil this function. The order that the bodily systems are introduced should not be construed as an order of importance.

The section covering diseases loosely follows the format of name, synonym, aetiology, pathogenesis, clinical lesions, gross lesions and judgement as to the fitness for human consumption. The diseases are listed in alphabetical order for ease of reference.

The Affections of Specific Parts and Conditions Encountered at Post-Mortem Inspection in Abattoirs is hoped to provide a tool for day-to-day inspection decisions in the abattoir environment, the former now updated to provide a guide to conditions encountered at ante mortem inspection.

AUTHOR DISCLAIMER

I must add that the judgements as to the fitness for human consumption given in this book are my own views, gained through experience and consultation with others.

A.Grist

ACKNOWLEDGEMENTS

This book in the series of meat inspection guides would not have been possible without the patience and assistance given by the following people:

Thanks to William (Bill) Boxall for his friendship, encouragement and initially persuading me to train as a Meat inspector.

I cannot express the gratitude I have for Dave Barrah for his friendship and boundless enthusiasm for every aspect of meat inspection. I have progressed from being his student 12 years ago to flecturfing wfith hfim at the , Schoofl of
Veterinary Science, and have always been treated by Dave as a colleague, despite the awe that I and his other students have for his knowledge. One of the greatest compliments I have received has been referred to as 'Dave junior'.

So many people from different organisations with whom I have dealt have helped with this edition so it seems easier to list them under their organization.

, Department of Cfllfinficafl Veterfinary Scfience, Langford

Alison Small BVM&S DVPH(MH) MRCVS formally of the Division of Food Animal Science, Bristol University for supplying the foreword to the first edition, encouragement, photographs and advice; Professor Geoff Pearson for supplying photographs and advice on other conditions. The post mortem room staff Bob Brafield, Donna Harraway and Andy Phillips for their help and patience; Dr Ross Harley, Islwyn Thomas, Steve Wotton MBE and my assistant Christine Rowlings.

Thanks are due to the following Meat Hygiene Inspectors of the **Food Standards Agency Operations Group**, Colin Walters, for his friendship and for teaching me how to take photographs and going out of his way to collect and provide samples, Julian Ponting, Eric Harvey, Manuel Sarnago MRCVS (Official Veterinarian) and Martin Evans. Special thanks are due to Mark Golden who collected samples for me despite the extra work this created.

The Red Meat Industry

F. Drury and Sons, Tockenham, Wiltshire allowed photographs of anatomy to be taken, despite the time and occasional delays my presence caused. Thanks go to the management and productfion staff of thfis estabflfishment; The
abattoir staff, Richard Ley, Tony Kelly and Colin Walters. Gavin Morris MRCVS of Dunbia without whom I would have lost enthusiasm.

Other Agencies

Dr Sophia Rizvi MRCVS of DEFRA, Nigel Durnford, Principal Animal Health Officer of Gloucestershire County Council, for supplying photographs and advice. Alex Barlow of the Veterinary Laboratories Agency for his constant support and advice.

My children, George, Elizabeth and Henry, who once again gave encouragement and assisted in preparing samples and also edited and labelled pictures, the latter being mostly undertaken by Elizabeth. Since embarking on this series of books they have become young adults who make me proud.

Once again, Sarah Keeling and Ros Webb of Nottingham University Press have displayed a level of calmness and patience I so obviously lacked during the production of this second edition.

My wife Grace deserves so much credit for the production of this volume. As with the others books she was always on hand to provide guidance, copious mugs of coffee, tolerance and editorial support in addition to being a sounding board for any ideas. I have always felt that her name should be on the front of the book in addition to mine as none of them would have been possible without her.

DEDICATION

I have always held the opinion, during my time as a Meat Hygiene Inspector, abattoir manager and lecturer that the food that I inspect should be fit for my own children to eat. If you would not feed it to your own children, you should not expect the consumer to feed it to theirs.

To that end I dedicate this to my wife Grace and our children George, Elizabeth, Henry and Harriette.

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[1] Author's own photographs
[2] Courtesy of Mr David Barrah
[3] Courtesy of Mr Eric Harvey
[4] Courtesy of Miss Alison Small BVM&S DVPH(MH) MRCVS
[5] Courtesy of Mr Manuel Sarnago MRCVS, Eville and Jones, Leeds
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[8] Courtesy of Dr Sophia Rizvi MRCVS, State Veterinary Service
[9] Courtesy of Nigel Durnford, Principal Animal Health Officer, Gloucestershire Council
[10] Courtesy of Prof Geoff Pearson

FOREWORD TO THE FIRST EDITION

For years now, those of us studying and working in the meat hygiene and inspection field have pieced together the information we need to formulate judgements on the status of carcase meat presented for human consumption from a variety of sources. Our teachers during our studies imparted their knowledge and experience, gained in much the same manner as we then did; textbooks and atlases of anatomy, pathology and pathogenesis, physiology, parasitology, animal health and meat inspection were consulted; questions were asked of our peers and mentors, and gradually, block by block, we built our understanding of the subject.

Finally, somebody came to the conclusion that study of the topic might be much easier if an integrated textbook was available. Furthermore, that somebody was possessed with the enthusiasm and perseverance to produce such a tome – in fact series of tomes.

This somebody was none other than Andy Grist, whom I first encountered when he was a student Meat Inspector, at Bristol University. And I was one of the teachers imparting my building blocks of information. Andy's enthusiasm for the subject has remained outstanding, and in developing this series of textbooks, he has been relentless in researching, asking and photographing. The thoroughness of his work is matched by its accessibility – Andy writes in a manner that encourages further reading, and further consideration of the topic.

In the following pages, book three enfolds, offering readers a sound working knowledge of applied ovine pathology. It, as its companions in the series, will prove a valuable addition to the personal reference libraries of Agriculturalists, Public Health Professionals, Veterinarians and students in these fields.

I wish you all success in your careers, and Andy – when you finish the series, take time to reflect on a job well done.

Alison Small
B.V.M.&S., D.V.P.H.(M.H.), Dip.E.C.V.P.H., M.R.C.V.S., M.A.M.I.

FOREWORD

Meat Inspection is both a science and an art, and skilled inspectors are able to make judgements on the fitness of carcases using all of the senses available to them. As we move further into the twenty first century, we see the general health of the UK sheep flock improving, and we see the risks of food borne disease from meat taking on a more microbiological basis, but we must not forget the fundaments of carcase post mortem inspection, and the valuable role undertaken by meat hygiene inspectors up and down the country.

This welcome new edition of Ovine Meat Inspection should serve as a valuable tool for veterinary students, official veterinarians and meat inspectors alike, but I would hope that breeders and stockmen would avail themselves of the vast array of information available, as we develop systems to report back the findings of post mortem inspection back to the holdings of origin. As such, developments can be made flock health generally, and everyone involved in the meat chain, from farm to fork, can play their role in ensuring that the meat we eat comes from healthy, well cared for sheep.

Craig Kirby
BSc, BVM&S, MRCVS

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ANATOMY/ PHYSIOLOGY

SKELETAL SYSTEM

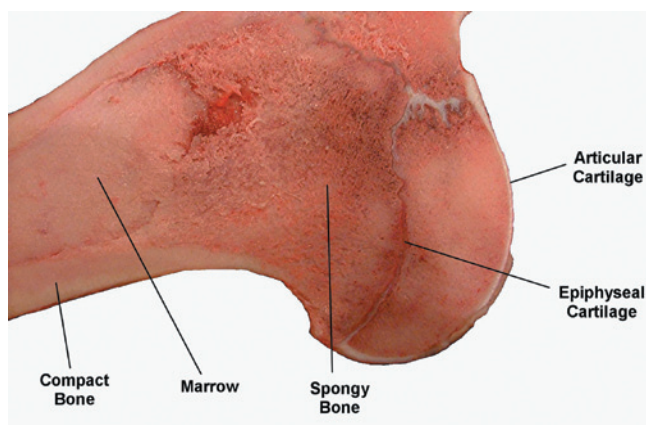
INTRODUCTION

The skeletal system provides structure, protection and a means of locomotion. Muscles are attached to the bones by ligaments or tendons and move the animal by contracting around a pivot or joint. In this section the basic structure, position and function of the bones is described. For this purpose the bones of the skeleton are divided into the skull, the axial skeleton and the appendicular skeleton (forelegs and hindlegs).

THE STRUCTURE OF BONES

Bone is a collagen matrix containing mineral salts, chiefly calcium phosphate, and various cells including osteoclasts and osteoblasts. The deposition of mineral salts within the matrix is controlled by osteoblasts. Mineral reabsorbtion and release of the minerals into the blood is attributed to the large, mononucleated osteoclasts. These cells work in balance, their activity being controlled by parathyroid hormone (PTH) secreted by the parathyroid gland in response to fluctuation in the serum- calcium level of the blood. If this level decreases, more hormone is released which has the effect of increasing the activity of the osteoclasts whilst decreasing the osteoblast activity and hence subsequently increasing the calcium in the blood. This deposition and reabsorbtion of mineral salts of the bone is a continual process.

Bones have a connective tissue membranous covering, the periosteum, which has bone-forming properties and, through fusion with muscular connective tissue, anchors the muscle to the bone. Under the periosteum is the dense, or compact bone, which in the long bones forms a hollow shaft containing marrow and spongy bone. Marrow occurs in two forms, red and yellow, and is a combination of blood vessels and connective tissue containing fat and blood producing cells. Red marrow produces blood cells such as erythrocytes and leukocytes; yellow marrow is formed mainly from fatty tissue. Spongy or cancellous bone is usually found at the extremities of long bones and is composed of thin intersecting layers of bone. The



Section through a humerus

articular cartilage has a bluish white colour and is also known as hyaline cartilage due to its glassy appearance. The epiphyseal cartilage represents the site at which bone growth increases the length of the long bones.

BONE TYPES

There are several classification systems used to describe bone types, the most common being the subdivision of bones into shapes, therefore the types encountered can be considered to be:

- Long bones, such as those of the humerus and femur.
- Short bones, such as the carpus and tarsus, roughly cuboid in shape.
- Flat bones, consisting of two layers of compact bone separated by a thin layer of spongy bone, as occurs in cranial bones and the scapula.
- Irregular bones, for example the vertebrae.

THE STRUCTURE OF JOINTS

The separate bones of the skeletal system are jointed together by systems that either fix the bone in relation to its neighbour or allow partial or free movement (articulation). Fibrous and cartilaginous joints between bones form fused or limited mobility joints, and synovial joints allow free movement between articulating bones.

Cartilaginous joints are found in areas such as between the vertebrae, at the pelvic symphysis and between the two halves of the mandible. Their structures vary and allow a small range of movement between the bones.

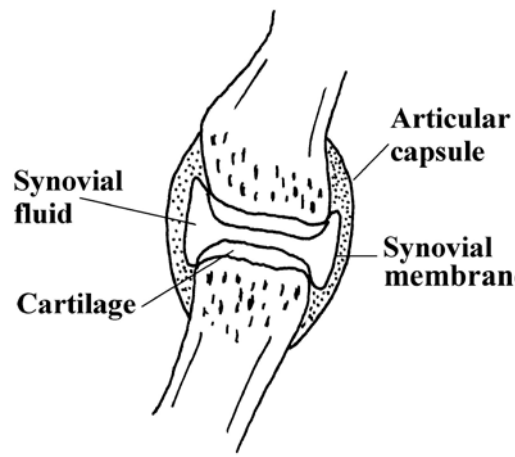
Fibrous joints immobily connect bones forming joints such as those fusing the flat bones of the skull at joints known as sutures.

The cartilaginous joints between the vertebrae

Skull sutures

Synovial joints occur wherever free movement is required. The direction of movement (be it rotational or in one or two planes) is dependant on the structure of the articulating surface of the bones connected and the direction of the action of muscular activity. Synovial joints are mostly associated with the appendicular skeleton. The area of the joint is surrounded by a capsule (articular capsule) the inner surface of which consists of a membrane (synovial membrane). The articular capsule contains synovial fluid, a plasma-like fluid secreted by the synovial membrane that lubricates moving parts and nourishes the cartilage.

The pivoting plane of the joint is determined by the shape of the bones of the joint and the arrangement of the surrounding ligaments. This plane has been used to classify the various types of synovial joint into groups, two of which include the hinge joint of the elbow which provides for pendulum movement in one plane, and the spheroidal joint (ball and socket) of the hip which allows both pendulum movement and a degree of rotation.



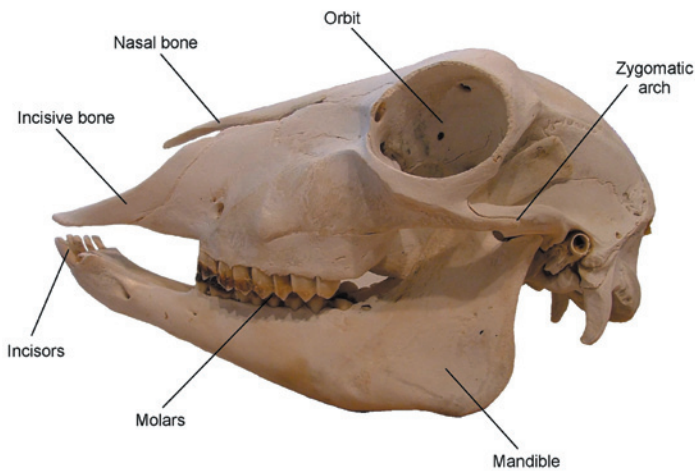
THE SKULL

The skull is formed by the cranium, facial bones and the lower jaw (mandible).

Fused joints called sutures, forming a dome within the cavity of which the brain is protected, join the cranial bones.

The facial bones, including the incisive and nasal bones, are again joined by sutures and house the organs of sight and smell. The nasal or turbinate bones form passageways or sinuses within the facial bones.

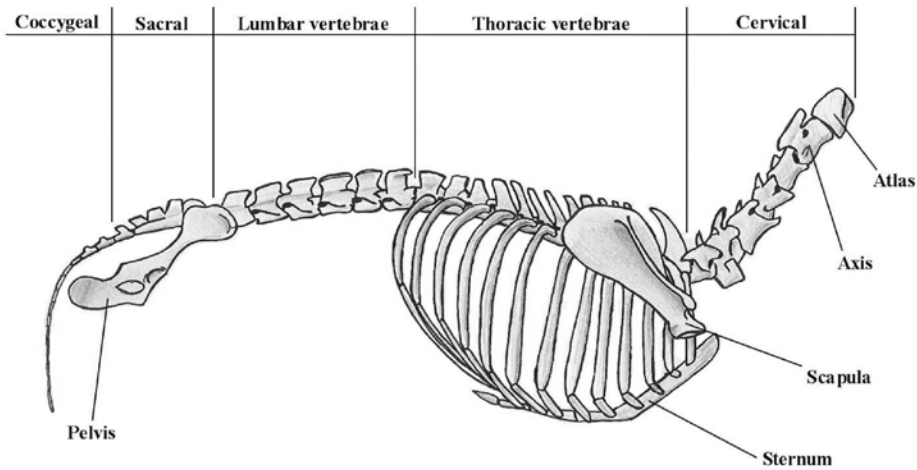
The lower jaw, the mandible, is U-shaped and very strong, capable of resisting the tremendous force exerted on it during the process of chewing. The entire mandible is made up of two halves, jointed at the chin by a mandibular symphysis, allowing a flexibility of rotation. The jaw is chiefly raised and lowered by the masseter muscles that are fused to the periosteum at the facial tuberosity.



THE AXIAL SKELETON

This term denotes the bones of the spine, the ribs and the pelvic girdle. The bones that comprise the spine are grouped together according to their position and function. The vertebrae are examples of irregular bones; they have small and irregular processes (projections) emanating from their dorsal and ventral surfaces. They have a central tunnel or canal which, when grouped with the other vertebrae, form the spinal channel through which the spinal cord runs. The articulation between the vertebrae is of a semi mobile cartilaginous symphysis, allowing a small range of movement per vertebrae but, when taken as a whole, allowing strength and flexibility of the spine.

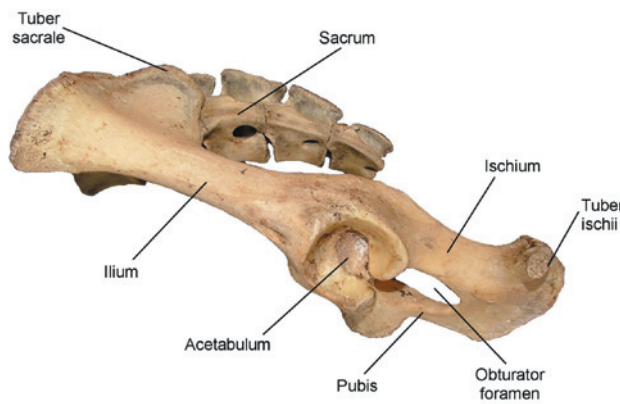
There are seven cervical vertebrae, (the skull articulating with the 1st cervical vertebrae, the axis bone), twelve to thirteen thoracic vertebrae (articulating with the ribs), six lumbar vertebrae (forming the roof of the abdominal cavity), five sacral vertebrae (fused together to form the synsacrum) and eighteen to twenty coccygeal or tail vertebrae.



THE RIBS

The ovine skeleton contains thirteen pairs of ribs articulating dorsally with the thoracic vertebrae. The ribs are classified by their relationship to the sternum, those that articulate ventrally with the sternum are known as true ribs, and those that do not are termed false ribs. The cavity formed by the thoracic vertebrae, ribs and sternum is the thorax.

THE PELVIS

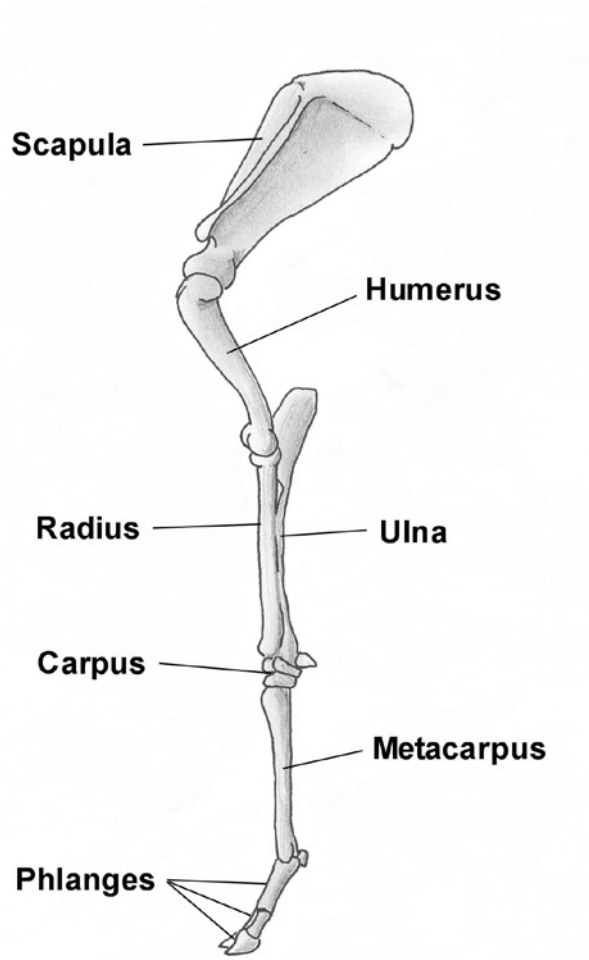


The pelvis forms a cavity within the body. The roof of the cavity is formed by the fused sacral vertebrae, the sides and base by the right and left *os coxae* (the pelvic bones, a fusion of the ilium, ischium and pubis). The pelvic bones are joined posterially by the pelvic symphysis. The articular surface of the femur joins the pelvis at the acetabulum forming a ball and socket joint.

THE APPENDICULAR SKELETON

FORELEG

The foreleg is similar in nomenclature to the human arm. The scapula (shoulder blade) is held in position by muscle and connective tissue, and as such does not articulate with the axial skeleton proper. It is classed as a flat bone, triangular in shape with a surface spine. This bone terminates at a ball and socket joint at which point it articulates with the humerus.

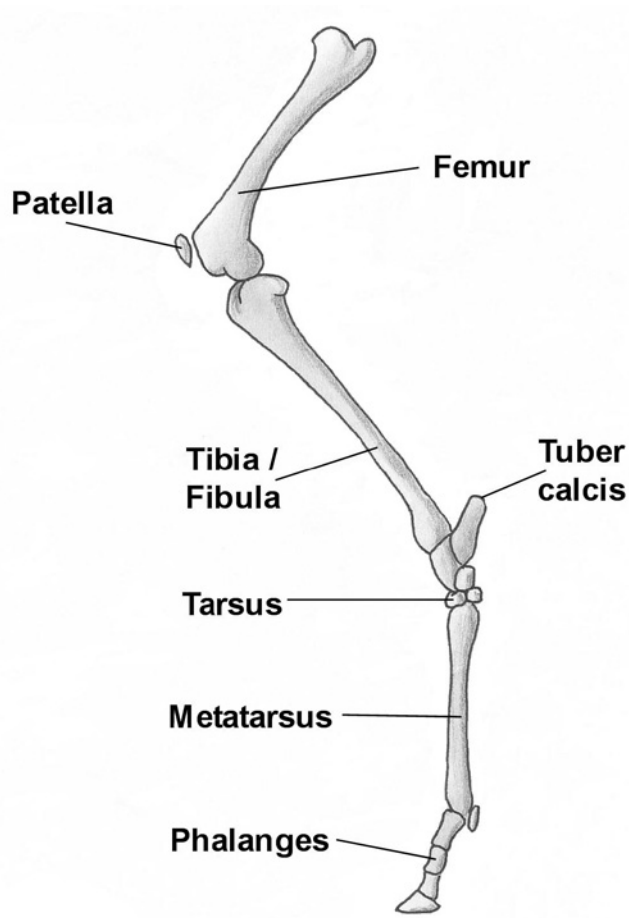


The humerus is an example of a long bone, basically consisting of a hollow cylinder of dense bone surrounding marrow, as such it possesses great load bearing ability in the vertical plane. The proximal humerus has several projections of bone (tuberosities) that represent the point of attachment of musculature, including the greater tuberosity that forms the point of the shoulder.

Distally the humerus articulates with a hinge type synovial joint with the radius and ulna. The ulna is fused to the larger radius and extends to form the olecranon or elbow.

The carpus (wrist) is made up of six bones, articulating dorsally with the radius and ventrally with the metacarpus. The bones of the foot (phalanges) form two digits separated by an interdigital cleft that is prevented from splaying by interdigital ligaments in the live animal. It is worth noting that only the hooves proper are separated by the cleft.

HINDLEG



As with the foreleg, the hind leg can be considered to be anomalous with the human leg. The large femur articulates proximally with the acetabulum of the pelvis in a ball and socket synovial joint, it is further secured in this joint by the presence of a round ligament (*lig. capitis*). The femur articulates distally with the tibia and rudimentary fibula at the stifle joint. Cranial to this joint, articulating with the femur is the patella (kneecap), held in position by three ligaments which also connect the patella to the tibia and fibula the latter of which is rudimentary and partially fused to the tibia, but is important as the distal end forms part of the hock joint.

The tibia articulates with two rows of tarsal bones, two in the upper row, three in the lower, the arrangement of which allows for flexion and extension of the hock. The rear of the hock is formed by the *Os calcis* situated at the rear or the top row which represents the point of attachment of the biceps and gastrocnemius muscles. The metatarsus is longer than the corresponding metacarpus of the forelimb and articulates distally with the two digits each formed by three phalanges.

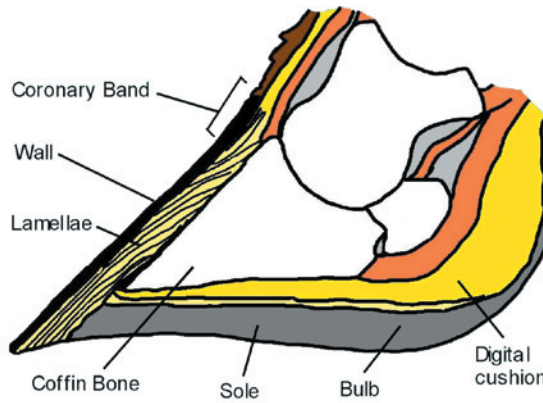
ANATOMY OF THE FOOT

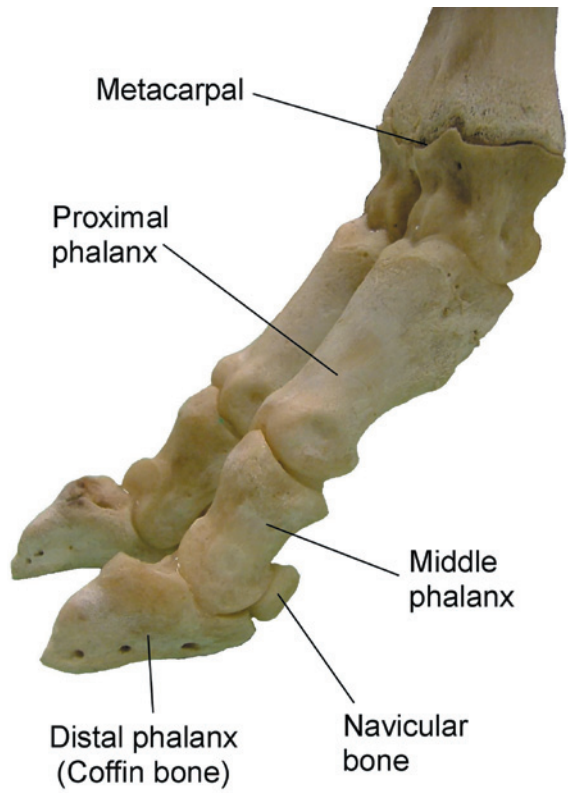
The hooves support the entire weight of the animal and can also be diagnostic of certain disease and welfare conditions in addition to being an obvious entry portal to inoculated pathogens present in the environment.

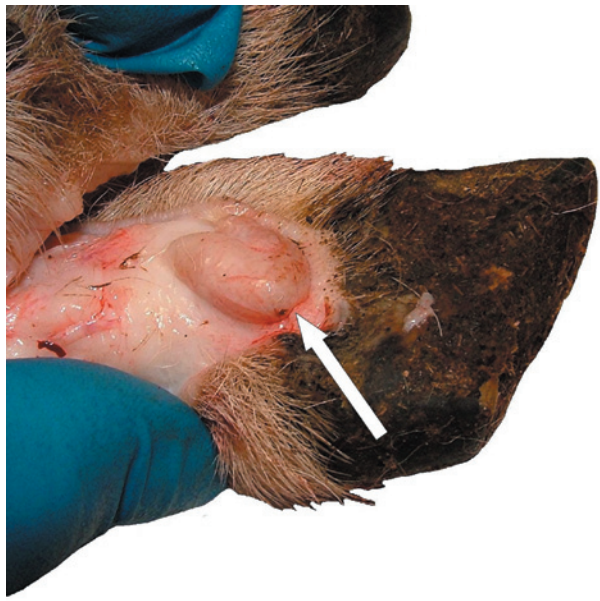
The heel of the hoof is formed by the bulb and digital cushion, the former being composed of soft rubbery horn material and the internal digital cushion acting as an elastic shock absorber.

Attached to the coffin bone, the terminal phalange, are laminae, formed by horn growth. In normal situations wear of the wall and laminae is equal to their growth, however sheep kept on soft ground will frequently display extended growth of the horn which eventually leads to lameness as debris compacts within the cavity formed by the overgrowth. This compaction applies pressure to the underside of the hoof and also promotes bacterial infection.

In sheep both the fore- and hind-legs contain an interdigital pouch (sinus interdigitalis) which opens just above the hoof. The glands situated in the walls of this pouch secrete a waxy fluid that is used as a scent marker for trails.







Incision through interdigital cleft, exposing interdigital pouch (arrowed)

Rear of hoof

MUSCLE

INTRODUCTION

Muscle is the reason that the meat animals are reared and slaughtered; the post mortem changes that occur in muscle converting it to meat. In the live animal muscle is found anchored to the skeletal frame providing posture, an ability to move, and protection for the internal organs. It also occurs internally to provide movement in the viscera and to provide valves to regulate flow, achieving this by converting the chemical energy of ATP (Adenosine triphosphate) to mechanical energy. There are three types of muscle; skeletal muscle, cardiac muscle, and smooth muscle, each possessing different characteristics. Cardiac muscle will be dealt with in the cardiovascular system later.

SKELETAL MUSCLE

This is also known as striated muscle and is under semi voluntary control of the nervous system and generally contracts around a fulcrum to facilitate movement about a joint.

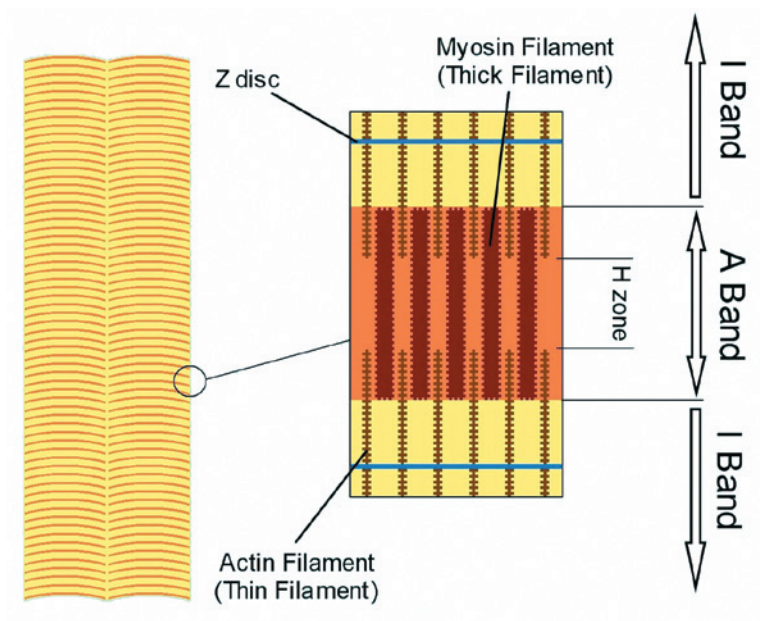
ANATOMY OF MUSCLE

Muscle is composed of individual long muscle fibres enclosed by a collagenous membrane, the endomysium, grouped into bundles surrounded by a second membrane called the perimysium. These bundles are further enclosed by the epimysium to form the muscle proper. The epimysium membrane extends past the termination of the muscle fibres to form the tendons that attach the muscles to the bone via the periosteum.

Muscle 'bundles'

FIBRES

Fibres are formed by the fusion of muscle cells (myoblasts) and hence each fibre contains numerous nuclei and mitochondria. Each fibre contains filaments of protein, the interaction of which in the presence of ATP coverts chemical energy into mechanical energy. Thick filaments are formed by the protein myosin, a protein that contains areas that have an affinity for bonding with ATP, and also the protein forming the thinner filaments, actin. The term striated muscle is given as, microscopically, bands are visible within the muscle fibre which corresponds to the degree of overlap of these protein filaments in a given area.



The banding noted in the muscle is given nomenclature; the dark bands are due to the thick filaments of myosin and are termed the A-band, and conversely the I-band is due to the presence of the thinner actin filaments. The area where the two types of filaments do not overlap is called the H-zone. The I-band is bisected by the Z-disc which is a band of non-contractile protein to which the myofibrils are attached; the area between two Z-discs forms the sarcolemma, the functional contractile unit of muscle.

ATP- ADENOSINE TRIPHOSPHATE

ATP is a compound formed by the hydrolysis of nucleic acids known as a nucleotide. It exists in all cells of the body and provides the main energy storage unit, a type of battery that stores energy in the form of high-energy phosphate bonds on the molecule. During muscular contraction the ATP molecule breaks the bond between the actin and myosin filaments but loses a phosphate group in the process releasing energy. The ATP now converts to adenosine diphosphate (ADP).

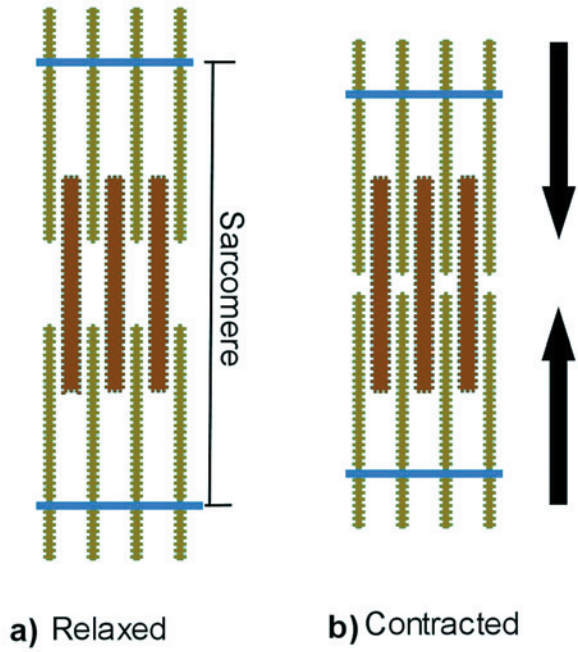
A small amount of ATP is stored in muscle cells; once this has been exhausted the muscle fibre will fatigue as the lactic acid level raises and will cease to function. The replacement of ATP is achieved in three ways, each method producing a phosphate bond to convert ADP to ATP; cellular respiration, conversion of creatine phosphate, and conversion of glycogen. When these methods are exhausted the myosin/actin bonds become permanent.

Cellular respiration is the main method of producing enough ATP to meet the demands of the muscle during prolonged activity and for the conversion of lactic acid to glycogen.

Glycogen is stored in muscle fibres and through the glycolytic pathway reaction converts ADP to ATP by breakdown of lactic acid. Creatine phosphate reacts with ADP to produce ATP and creatine. These latter reactions are finite; once they have been exhausted cellular respiration provides the energy for ATP production in the muscle.

CONTRACTION

On receiving stimulus from the nerve the myosin head attracts the actin toward it and binds with it, drawing the unfixed thin filament along the fixed thick filament. ATP then breaks this bond and the actin rebinds at the next available myosin head further down the thick filament and the process repeats having the effect of shortening the sarcomere in a gear toothed or ratchet-like manner. During contraction the A-band (the darker band) remains the same size, the I-band shortens as the Z-discs are pulled toward each other.



FIBRE TYPES

Two types of fibre are found in a muscle group, Type I (red fibres) and Type II (white fibres) that have different characteristics as listed below. The combination of these fibres within a muscle determines that muscles efficiency for the tasks required of it.

*Type I Fibre	Type II Fibre
Red fibre (Slow Twitch)	White Fibre (Fast Twitch)
Large number of mitochondria	Low number of mitochondria
Dependent on cellular respiration ATP	Dependent on Glycolytic ATP
Rich in myoglobin	Low in myoglobin
Activated by small diameter (slow conducting) neurons	Activated by large diameter (fast conducting) neurons
Fatigue resistant	Used for rapid contraction
More prevalent in muscles of posture	Fatigue easily
	More dominant in muscles used for rapid movement.

RIGOR MORTIS

Rigor mortis, the 'death stiffening' that occurs after death is the permanent bonding of the myosin/actin filaments once the available ATP is exhausted due to the lack of cellular respiration. The process of rigor mortis has three phases: A **delay phase,** during which time available ATP is used within the muscle fibre to prevent myosin/actin bonding. An **onset phase,** during which time the cellular respiration ATP is exhausted and the pool of ATP from creatine phosphate and glycolysis are used up and permanent rigor myosin/actin bonds are formed, and a final **completion phase,** where the regeneration of ATP ceases and the myosin/actin filaments become fully bonded. These bonds eventually break due to the lower pH conditions that occur in the carcase. It is worth mentioning that the hardening of fat is not due to rigor but to the lowering of temperature.

NERVE SUPPLY

Each individual muscle fibre is served by an axon that branches from a motor neuron (see nerves types and function in Nervous System section). Stimulation of the motor neuron creates contraction within every fibre served by the braches of its axon, therefore muscles requiring precise control will be served by many neurons with few branches, and large muscles can be controlled by few motor neurons with many branches.

SMOOTH MUSCLE

Smooth muscle, also known as visceral muscle is found within the walls of the hollow structures of the body with the notable exception of the heart. Contraction of smooth

muscle alters the dimension of the structures including the diameter of arteries, the peristaltic motion of the intestines, the diameter of the bronchi, contraction of the urinary bladder during urination, and contraction of the uterus during parturition.

Unlike cardiac and skeletal muscle there are no visible strata within smooth muscle, although it still contains the sliding filaments of actin and myosin but fixed to the cell membrane rather than a Z-disc. Smooth muscle comprises single cells that possess contractile properties. The contraction of smooth muscle is partially involuntary as is cardiac muscle, but can also be stimulated by motor neurons and the presence of hormones. For example the presence of bile in the small intestine has a triggering effect on the peristaltic movement of the intestinal smooth muscle, and oxytocin released into the bloodstream stimulates contraction of the uterus at parturition.

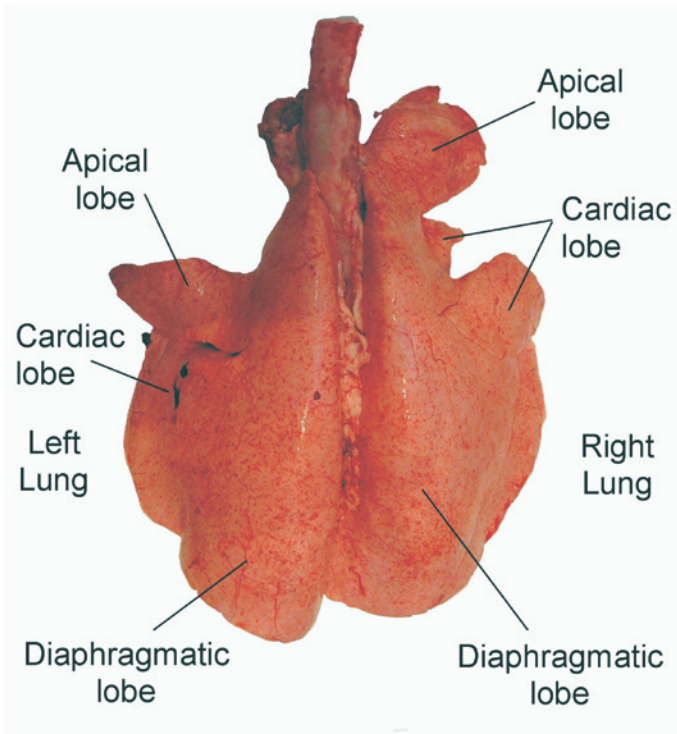
THE RESPIRATORY SYSTEM

INTRODUCTION

The tissue cells of the body require oxygen to function; they also require the removal of the waste products of their metabolism, including carbon dioxide. The cardiovascular system is the transport vector by which this is achieved, the respiratory system being the method of gaseous exchange of oxygen for carbon dioxide, voiding the latter from the body. For the purposes of description the respiratory system will be considered as the nostrils, nasal passages, larynx, trachea, bronchi and the lungs.

DISTINCTIVE FEATURES

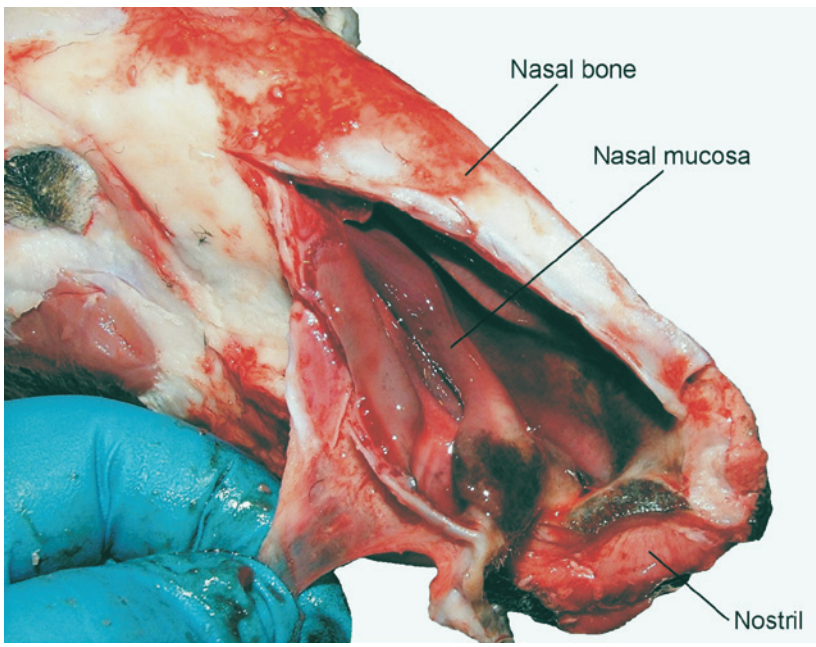
The ovine trachea is long, made up of 50+ cartilaginous rings and possesses a dorsal ridge. There are two main bronchi and a single accessory bronchus leading to the right apical lobe. The lung tissue is soft, spongy and less distinctly lobulated than those of bovines. The left lung consists of three lobes; the right consists of four or five lobes.



Ovine Lungs

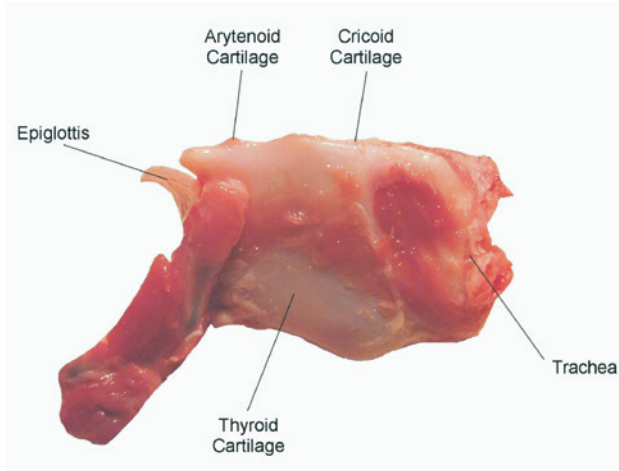
NOSTRILS AND NASAL PASSAGES

Air is drawn into the body through the nostrils, the size of which can be varied by contraction or relaxation of the surrounding cartilage-reinforced muscles. Inside the nostrils begins a mucous lining which covers the scroll-like turbinate bones that lie ventral to the nasal bone, the shape of these bones forces the inspired air against the mucous lining. The presence of mucous on hairs inside the nostrils serves as a trap for foreign particulate matter above 10μm in diameter, and also, in conjunction with the heavily vascularised nasal tissue, serves to moisten and warm the inspired air before it continues to the larynx.



Nasal mucosa – incision along nasal bone after head has been flayed

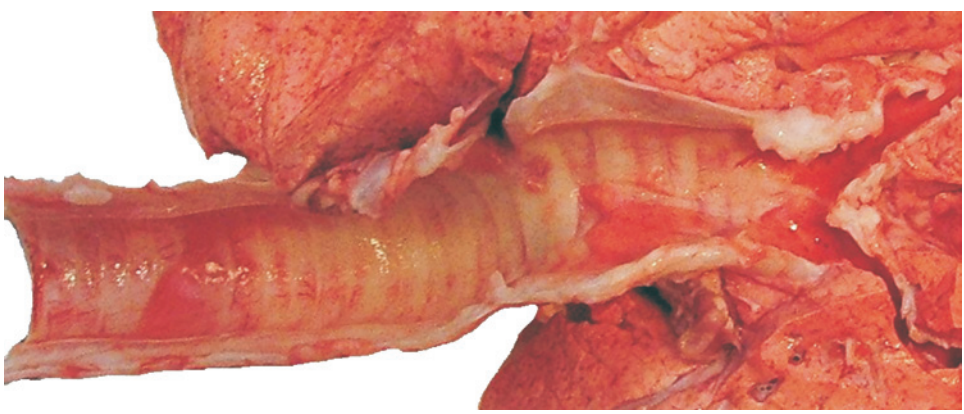
LARYNX



The larynx is formed from five cartilaginous structures that articulate with each other. The main body of the larynx is formed by the cricoid and thyroid cartilages. Within this body are two arytenoid cartilages that articulate with the thyroid cartilage and form the mechanism of vocalisation of the animal. Cranial to the paired arytenoids cartilages is the epiglottis, which also articulates with the thyroid cartilage; this structure moves to cover the opening of the larynx during swallowing to prevent the aspiration of foodstuffs into the trachea.

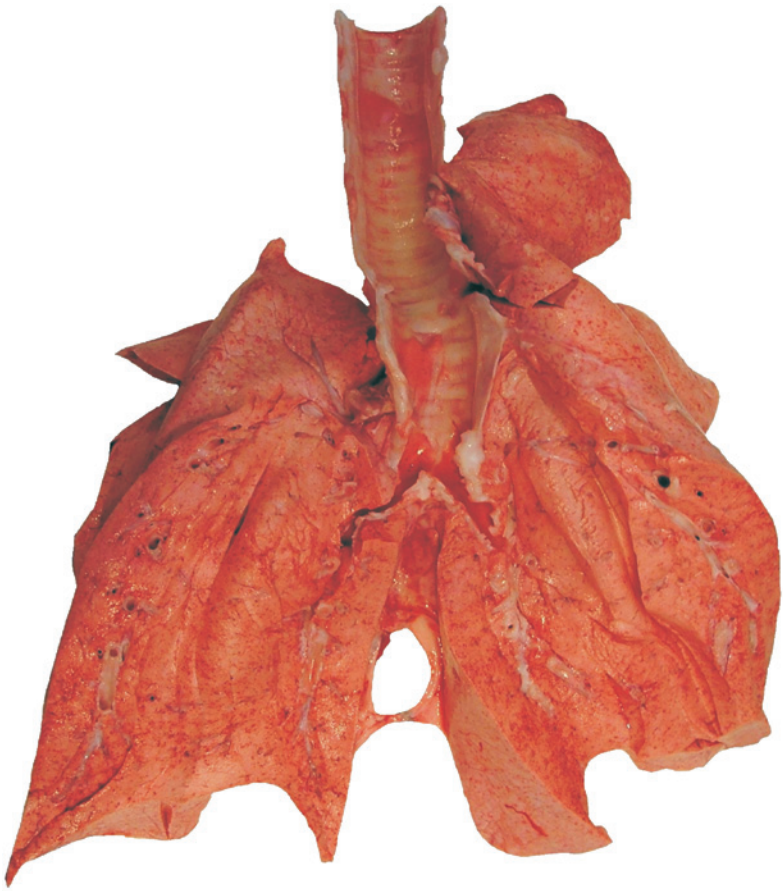
TRACHEA

The trachea (windpipe) is given structure by approximately 50 cartilaginous, incomplete, rings that prevent the collapse of this tube during the low pressure encountered during respiration. These incomplete rings meet behind the trachea forming a prominent dorsal ridge that can be used as an aid when identifying specimens. The internal structure of the trachea includes ciliated cells and mucosal cells forming part of the bodies' immune response, catching particles larger than 2µm and removing them by the escalatory peristaltic motion of the cilia to the larynx and pharynx where expectoration expels them from the body.



BRONCHI

The trachea branches off into the smaller diameter bronchi on entrance to the lung tissue. In sheep a cranial accessory bronchus branches off the trachea to the right apical lobe before the two main bronchi divide the trachea at the point known as the bifurcation. The bronchi then further subdivide within the lung tissue forming the air passageway known as the bronchial tree, the branches of which continue to subdivide until they reach microscopic terminal sacs, the alveoli, also known as air sacs.



The alveoli, which are surrounded by a web of interjoining capillaries, are formed by alveolar cells attached to a thin gas permeable basement membrane which separates them from the capillaries. The alveolar cells are constantly dividing and have the ability to move from their position to undertake their role as part of the bodies' immune system. The alveolar cells have phagocytic properties, and can absorb particles less than 2μm in diameter and remove them by entering lymphatic vessels in the surrounding tissue.

THE ACT OF RESPIRATION

During inspiration the muscles of the diaphragm contract pulling it flat and tight. This, combined with the simultaneous contraction of the intercostal muscles that increases the diameter of the thorax, creates a reduction in the internal air pressure which leads to atmospheric air being drawn in to equalize the pressure. Exhalation involves a relaxation of the muscles that increases the internal pressure and air is drawn out of the body in another attempt to equalize pressure.

The air is drawn through the nostrils, down the trachea and to the alveoli via the bronchial tree where it diffuses through the alveolal wall into the blood. Haemoglobin in the erythrocytes binds oxygen to form oxyhaemoglobin in this circumstance, where the partial oxygen pressure is high, and releases carbon dioxide which moves into the alveoli for expulsion. The oxygen is then carried to the tissue cells of the body where the reduced partial oxygen pressure causes disassociation and release of the oxygen, converting the oxyhaemoglobin back to haemoglobin in which state, in conjunction with the reduced partial oxygen pressure, carbon dioxide is absorbed.

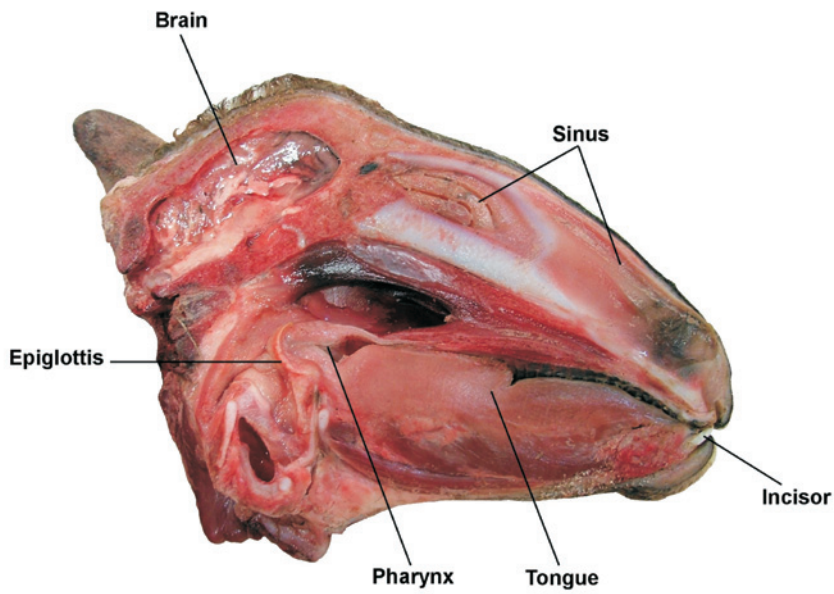
DIGESTIVE SYSTEM

INTRODUCTION

The purpose of the digestive system is the conversion of in-taken fuel (food) into basic components that are either water-soluble or to particles small enough to be held in suspension in solution. In this form these components can be removed from the digestive system, and transported through the body for synthesis into complex chemicals, storage etc. The digestive system achieves this conversion by means of mechanical and biochemical mechanisms aided by the length of the intestines and immense surface area available for absorption of these nutrients which are then transported through the body by both the blood circulation and the lymphatic system. The digestive system can be considered as a tube of varying diameter stretching from the mouth to the anus.

THE MOUTH

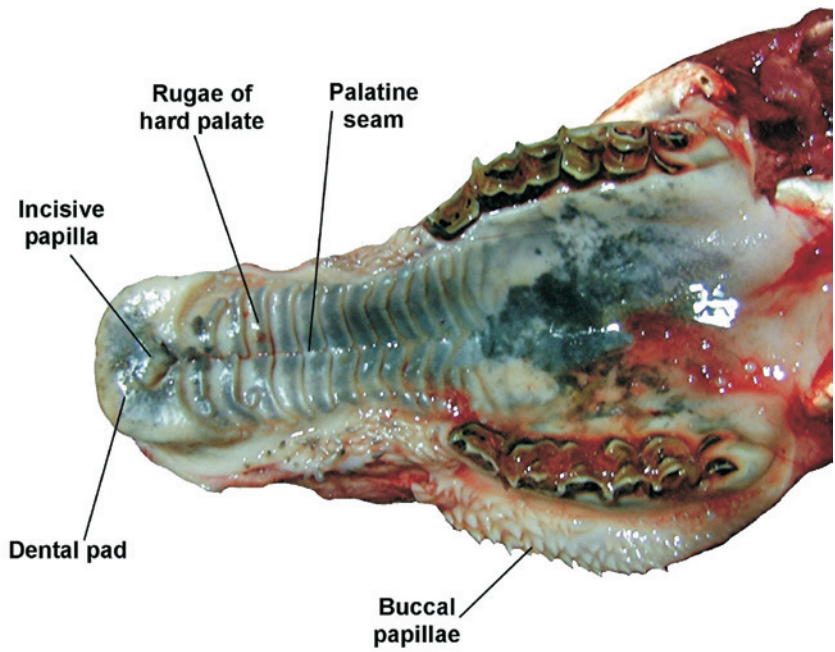
The mouth, or buccal cavity, provides the initial stages of digestion, mechanical and chemical. The upper hard palate, the mandible, the soft tissue pharynx at the rear, and the lips at the front, form this cavity. The sides of the buccal cavity are formed by the cheeks, the purpose of which is to expand to allow more food into the cavity but possess enough elastic capability to move food back into the central cavity for further processing by chewing (mastication). The inner surfaces of the cheeks of sheep are protected from penetrative damage by hard or sharp items of herbage by conical buccal papillae.



Median section through an ovine head

There are three pairs of salivary glands secreting a fluid containing mucin to moisten the food as it is chewed and reduce froth in the rumen, a cause of bloat. In addition saliva contains sodium and potassium bicarbonate to buffer the acidity of the rumen, and mineral salts to aid bacterial growth. These glands are situated at the rear of the mouth near the base of the jaw (submaxillary), in front of the insertion of the ear (parotid) and in a sub mucosal layer lateral to the tongue (sublingual).

The ovine upper palate possesses no incisors, food being sheared by the action of the lower incisors against the hard, keratinised dental pad. The hard upper palate extends dorsally joining the soft palate that in turn joins the pharynx. The soft palate is bordered by the upper molars that move in a grinding action against those of the lower jaw.

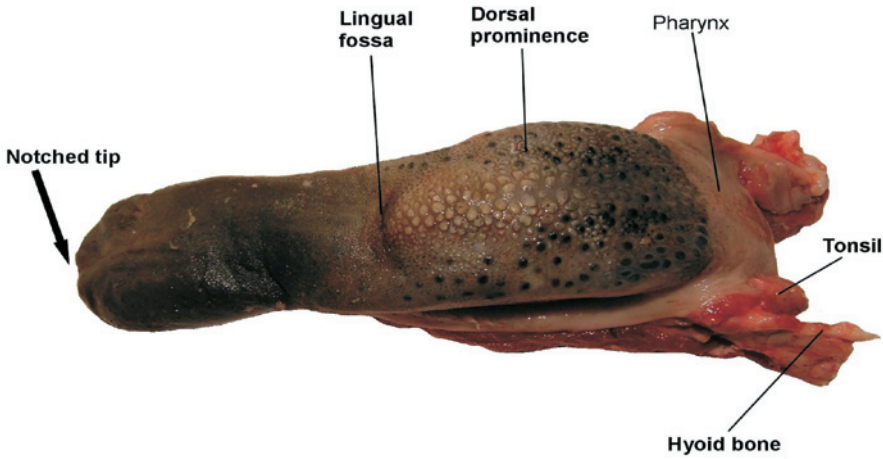


Internal view of upper buccal cavity - Ovine

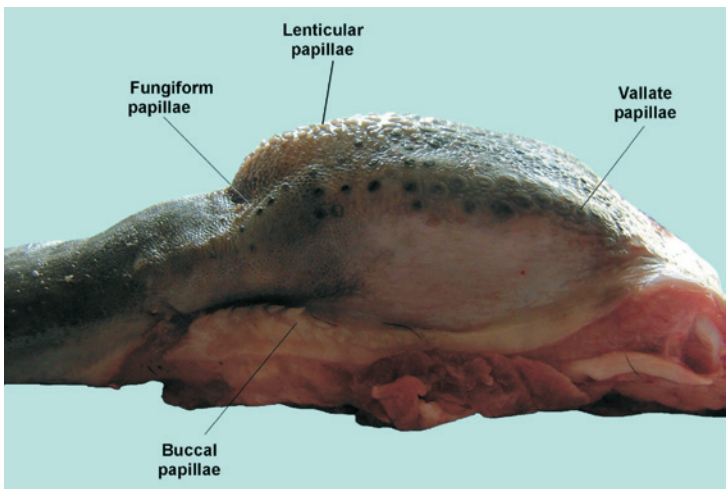
Movement of the mandible is mainly achieved through contraction of the external masseter muscles and the internal (pterygoid) muscles. At the front of the mandible are the incisors. Initial temporary (deciduous) incisors are replaced by permanent counterparts over time, the eruption of this permanent dentition being (given the vagaries of genetics and chance) both systematic and predictable and therefore used as a method of approximating the age of sheep, the first permanent incisor erupting at approximately one year of age.

THE TONGUE

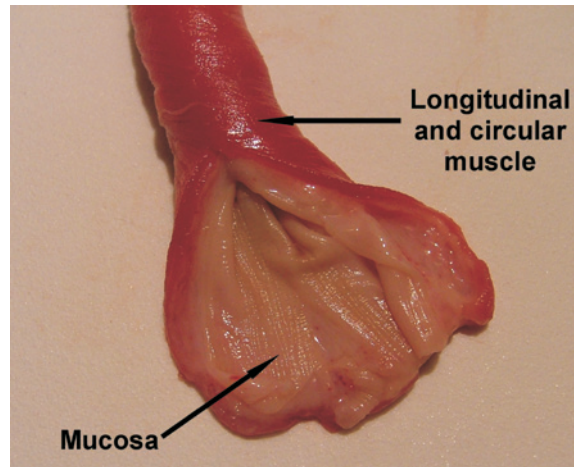
This is a muscular, occasionally pigmented, semi-prehensile organ used to draw food into the buccal cavity where it is severed by the action of the incisors against the upper dental pad. The shape of the tongue conforms to the basic shape of the lower jaw and is rooted at the base to the hyoid bone at the rear of the mandible.



The dorsal surface of the tongue is distinguished by the presence of various characteristic lingual papillae. The tip of the tongue is roughened by horny, rearward facing, filiform (threadlike) papillae; scattered amongst these are fungiform (mushroom like) papillae. Approximately half way along the dorsal surface of the tongue is a transverse depression in front of a dorsal prominence, the latter being covered in lenticular (lens like) papillae. Toward the rear of this prominence, either side of the midline, are groups of vallate (rim like) papillae, which in conjunction with the fungiform papillae constitute the taste buds.



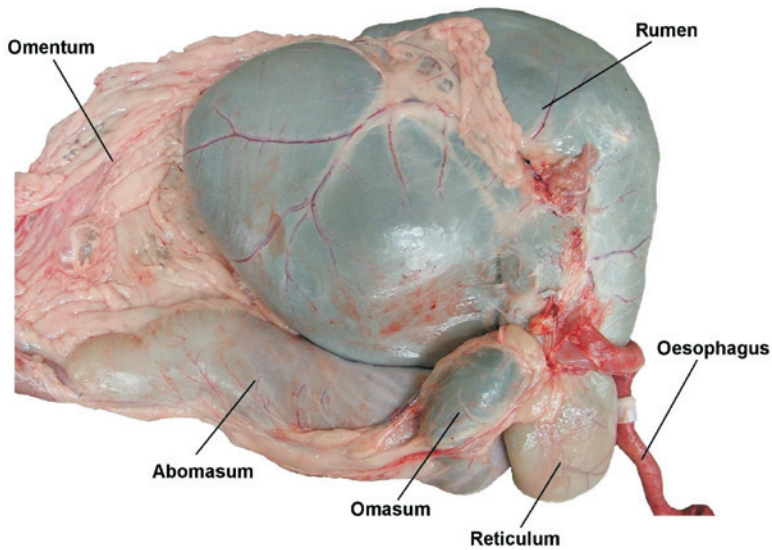
OESOPHAGUS



This runs from the pharynx to the rumen, the fore stomach. The oesophagus is a mucosal-lined tube, surrounded by an inner layer of circular muscle, encapsulated in a sheath of longitudinal muscle. When these muscles contract they produce a wave like motion (peristalsis) squeezing food material down the length of the oesophagus. In ruminant animals, such as sheep, this action is partly under conscious control, allowing material to be moved back up the oesophagus from the fore stomach to the mouth for further mastication.

FORESTOMACHS

In the ruminant animal, the digestion of rough complex carbohydrates requires a degree of fermentation to occur by bacterial action. This is accomplished in the forestomachs, comprising the three chambers of the rumen, reticulum and omasum; the abomasum being the true, or gastric, stomach. The walls of the forestomachs comprise a smooth peritoneal covering, a thick muscular layer (involuntary muscle arranged in circular, oblique and longitudinal layers to provide a churning of the contents), a submucosal layer of connective tissue, and a thick mucous membrane.



RUMEN

This chamber communicates directly with the reticulum. An internal groove (reticular or oesophageal groove) directs food from the oesophagus into both chambers. The rumen occupies a large percentage of the abdominal cavity mostly on the left hand side, accounting for approximately 80% of the volume of the stomachs. The interior mucosa of the rumen is covered with keratinised papillae that range from a leaf form to barely visible conical papillae at the roof of the rumen.

Detail of the leaf-like papillae of the rumen

The contents of the rumen tend to form layers, the lower layers being made up of remasticated fermenting material, the upper layers of less dense material awaiting regurgitation up the oesophagus for further chewing, with a final layer of gas.

RETICULUM

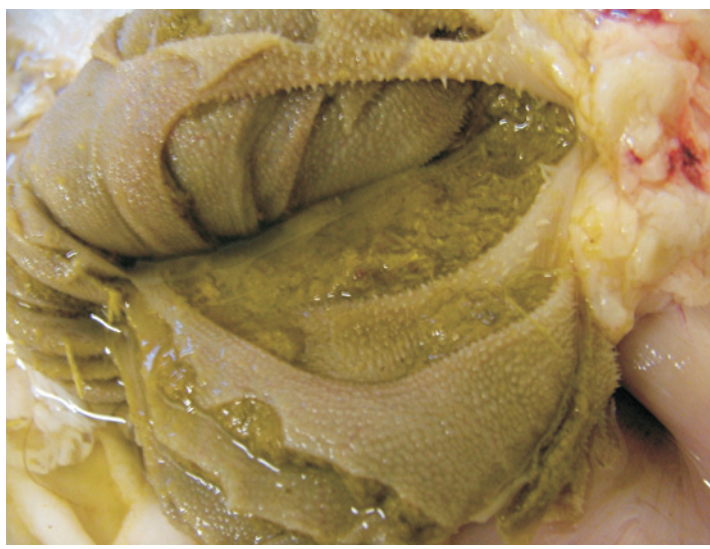
Also known as the honeycomb due to the ridge enclosed shape of areas of the mucosa. The reticulum is situated cranial to the rumen and receives a percentage of the oesophageal discharge, generally the remasticated denser material. It is thought that a portion of the volatile fatty acids produced during the bacterial fermentation of the complex carbohydrates are absorbed through the mucosa of the rumen and reticulum and that the structure of the internal mucosa of the reticulum provides an increased surface area for this to occur.

The 'honeycomb' structure of the reticular mucosa

The uneven surface of the reticulum also allows penetration of sharp or hard objects through the mucosal wall.

OMASUM

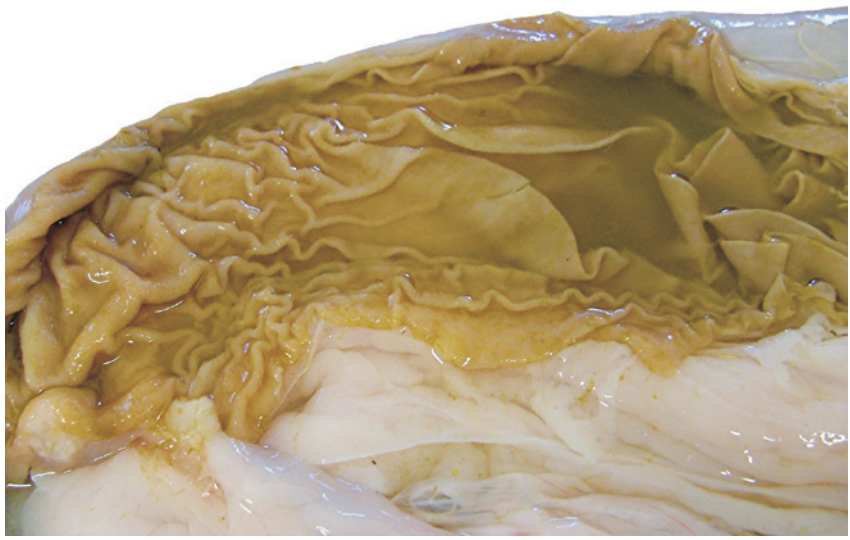
The omasum (colloquially known as 'manyplies' or 'bible') is the third, smallest, and final forestomach, of ovines. Internally the mucosal surface is heavily folded into laminae, hence the synonyms, these laminae being covered in lenticular and conical papillae. Ingesta enters via the reticulo-omasal orifice and exits into the abomasum through an omasal-abomasal orifice. Contractions of the omasum force ingesta between the laminae and further into the abomasum.



A portion of omasum, demonstrating laminae and lenticular papillae

ABOMASUM

The abomasum represents the true gastric stomach of the ruminant animal. Once fermented, the ingesta passes from the omasum into the abomasum, the internal mucosal surface of which is generally pink and glandular as opposed to the greenish discolouration of the previous forestomachs.



Opened and cleaned abomasum

RUMINATION

Once the in-taken food has undergone the initial process of mastication and mixing with saliva it is swallowed, moves down the oesophagus by peristaltic motion, and enters the rumen. Here the ingesta is subjected to the churning motion of the stomach and the presence of cellulose-splitting bacteria aided by the alkalinity of the saliva. After a period of time in the rumen a bolus of the ingesta is regurgitated by contraction of the rumen and reverse peristalsis of the oesophagus back to the mouth where it is re-chewed with the addition of more saliva, to begin the process of converting starches to sugars; the food is then re-swallowed.

In the new born lamb, the rumen accounts for approximately 30% of the stomach volume, the reticulum and omasum being vestigial, and the animal almost behaves as a monogast. Once weaned the bacterial loading of the rumen increases until the rumen accounts for 80% of the volume of the forestomachs. The rumen contains bacteria and

protozoa that produce surface enzymes to break down food and synthesise the by-products of fermentation, such as volatile fatty acids (VFAs) and vitamins B and K, the VFAs being absorbed directly through the rumen. The gaseous by-products of the fermentation process, carbon dioxide and methane, are voided from the rumen by eructation (belching).

Which compartment of the stomach the ingesta then enters is dependant upon its state of maceration and specific gravity; if still requiring fermentation and maceration it is heavy and re-enters the rumen, if highly fluid and well macerated it passes into the omasum. The omasum has the sole function of grinding the ingesta into a paste before it enters the abomasum. The food is forced through the leaves of the omasum and is subjected to grinding by the keratinised papillae and the muscular action of the omasal walls. Finally the food paste enters the abomasum. Throughout the passage and storage of the ingesta in the forestomachs, cellulose-splitting bacteria break down the fibres of the herbage.

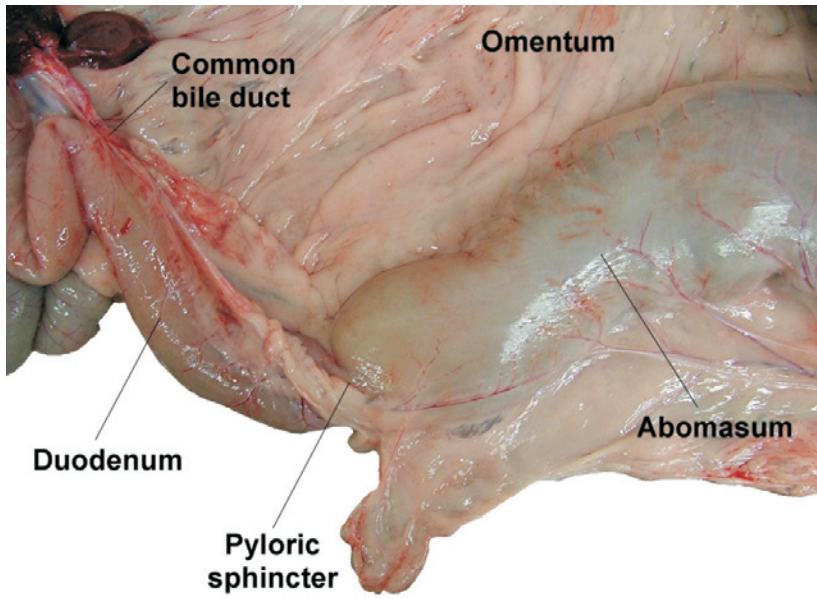
The mucous membrane of the abomasum secretes gastric juice which contains enzymes such as pepsin and rennin in addition to hydrochloric acid. In this acidic environment fat and peptones are released, lysozyme enzyme is also released which breaks down bacterial cell walls destroying bacteria before they enter the small intestines The food then enters the intestines via the pyloric sphincter.

THE INTESTINES

The intestines are generally divided into the small intestine consisting of the duodenum, jejunum and ileum, and the large intestines of the caecum, colon and rectum.

SMALL INTESTINE

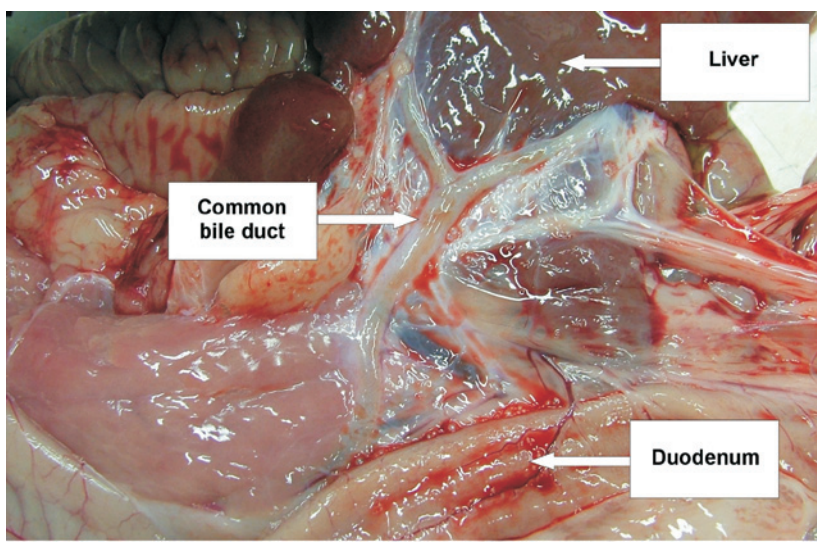
The small intestine runs from the pyloric sphincter of the abomasum to the ileo-caecal valve. Although the small intestines do secrete a nominal amount of enzymes, their primary function is the absorption of released nutrients. The walls of the intestines are formed, like the forestomachs, by four layers; a thin peritoneal surface, two muscular layers, longitudinal and lateral, and an inner mucosal layer. In addition to mucosal folds (rugae) the internal mucosa of the intestines is lined with microvilli, finger-like projections into the lumen that greatly increase the surface area of the intestines but also act as the main points through which nutrients are absorbed. The villi encapsulate a central lymph channel surrounded by blood capillaries. Situated in the mucosa and submucosa are raised patches of lymphoid follicles collectively known as Peyer's patches.



Transition from abomasum to duodenum via pyloric sphincter

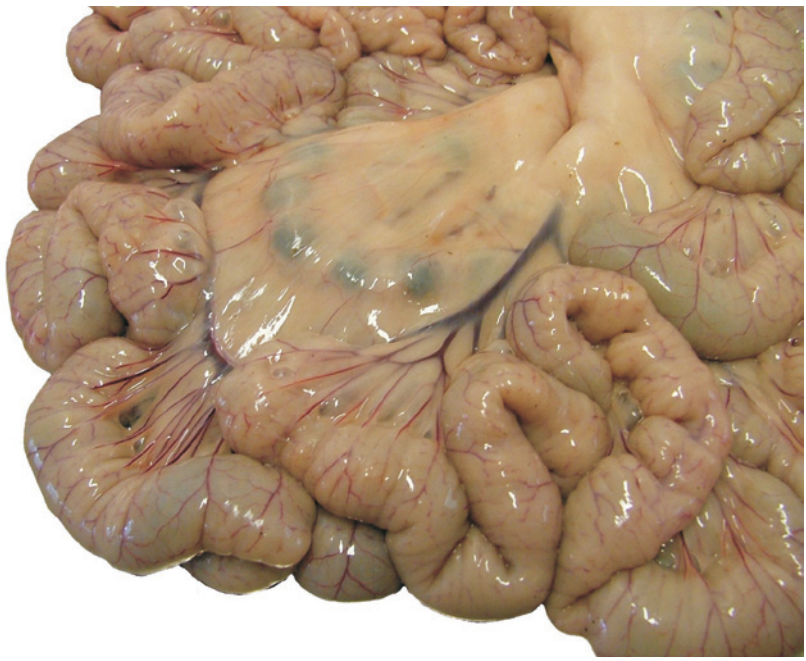
DUODENUM

The duodenum is divided into two ascending and a single descending portion. The ascending portion starts at the pyloric sphincter and follows a vertical path to the visceral surface of the liver to which it is attached by a portion of the omentum; followed by a descending portion running toward the pelvic region before ascending again to a point of attachment with the mesentery. The pancreatic duct joins the descending duodenum, and the bile duct enters at a point where the duodenum is held to the visceral hepatic surface.



JEJUNUM

The jejunum continues from the point at which the duodenum enters the periphery of the mesentery and follows the extremities of this to join the ileum. The jejunum is shadowed by a parallel line of mesenteric lymph nodes situated further within the substance of the mesentery, embedded in fat.



The jejunum running along periphery of the mesentery

ILEUM

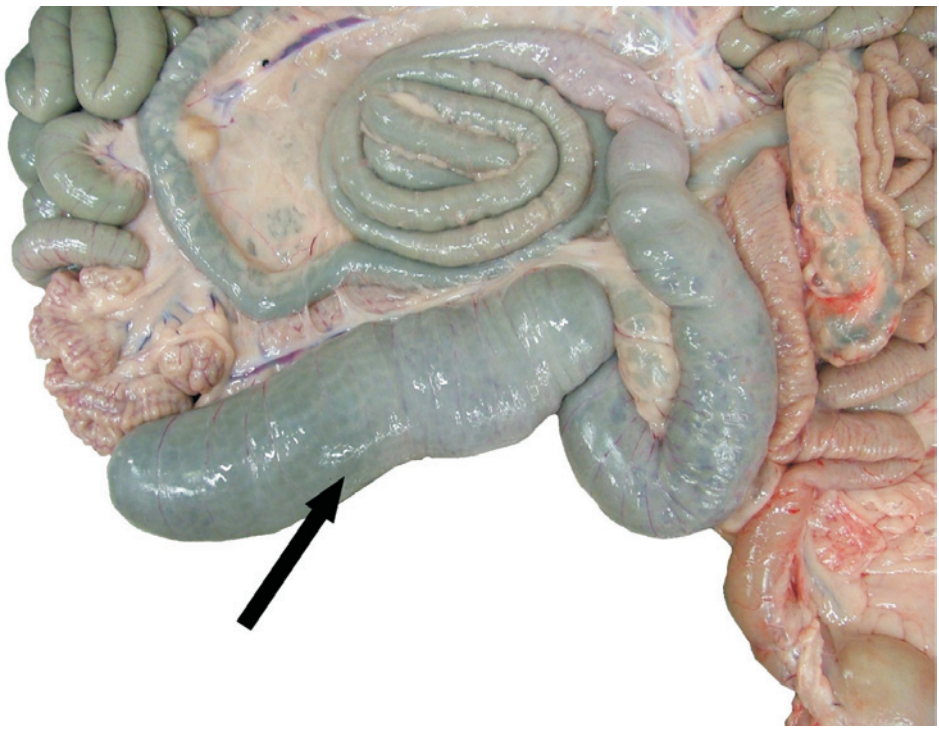
The ileum is short and terminates at the ileo-caecal valve.

LARGE INTESTINE

The large intestine is mainly involved in water balance and the absorption of minerals and volatile fatty acids. The caecum also acts as a secondary fermentation vessel, bacterial activity providing further break down of the food. The large intestine primarily secretes mucous to aid movement of material and terminates at the rectum, or anus, where the waste matter is voided from the body.

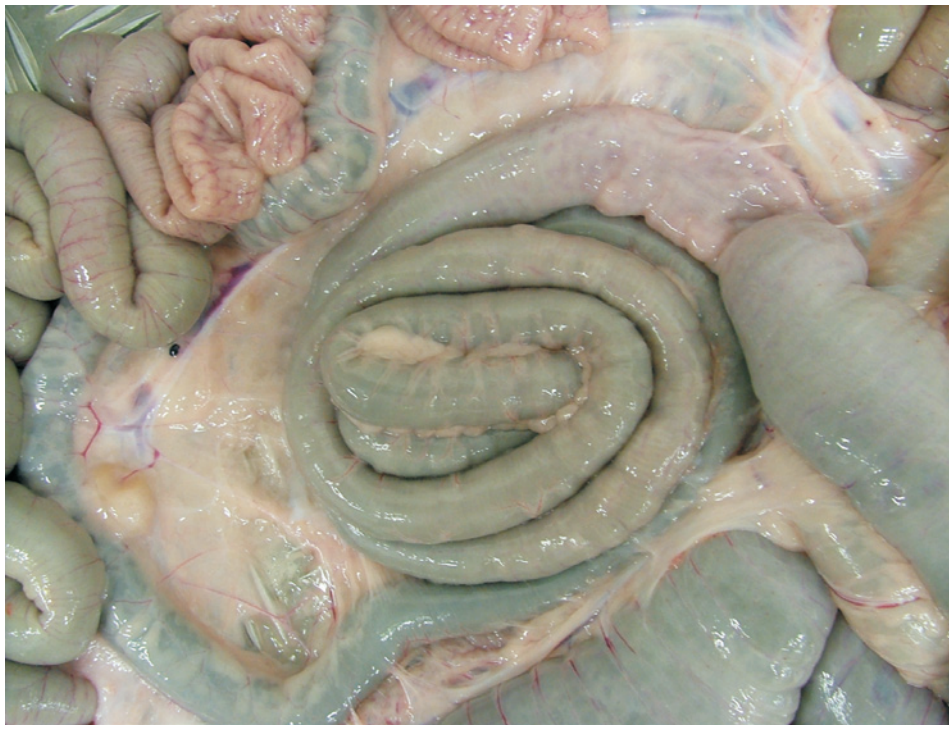
CAECUM

Starting at the ileo-caecal valve (preventing the contents of the caecum from extruding back into the ileum during muscular contraction) the caecum is a blind-ended diverticula of the large intestine. The end of the caecum is loosely attached allowing vertical movement of the sac dependant on the volume of gas it contains.



COLON

The colon is of a similar diameter to the undistended caecum and is positioned within the mesentery where it narrows in diameter and follows a winding clockwise diminishing spiral pattern for 1½-2 turns before returning in an anticlockwise spiral and joining the free transverse section of colon that leads to the rectum. The main function of the colon is maintenance of the water balance, the removal of water from the faecal matter before entering the rectum. The mucosa of the colon secretes mucous to lubricate the faecal matter.



The spiral of the colon, embedded in mesenteric fat

RECTUM

This area stores faecal matter prior to expulsion through the anus. The rectum has a larger calibre than the colon with muscular walls. It leads to the anus which comprises an orifice controlled by an internal and external sphincter, the latter of which is comprised of skeletal muscle and hence is under conscious control.

INTESTINAL FUNCTION AND DIGESTION

After the foodstuff has undergone initial enzyme degradation and been subjected to the acid conditions of the abomasum (pH>2) the resultant fluid paste enters the duodenum through the pyloric sphincter. This involuntary muscle prevents the reflux of the ingesta back to the abomasum. The muscular layers of the intestinal wall then move the food by peristaltic motion; this propels the food through the intestine, has a 'mixing' affect and also forces the food against the absorptive lining of the mucosa.

The bile received in the cranial duodenum stimulates peristaltic contraction of the intestinal muscles, and in conjunction with the pancreatic juice reduces fats to free fatty acids and emulsifies them to enable their removal through the lymphatic channels of the

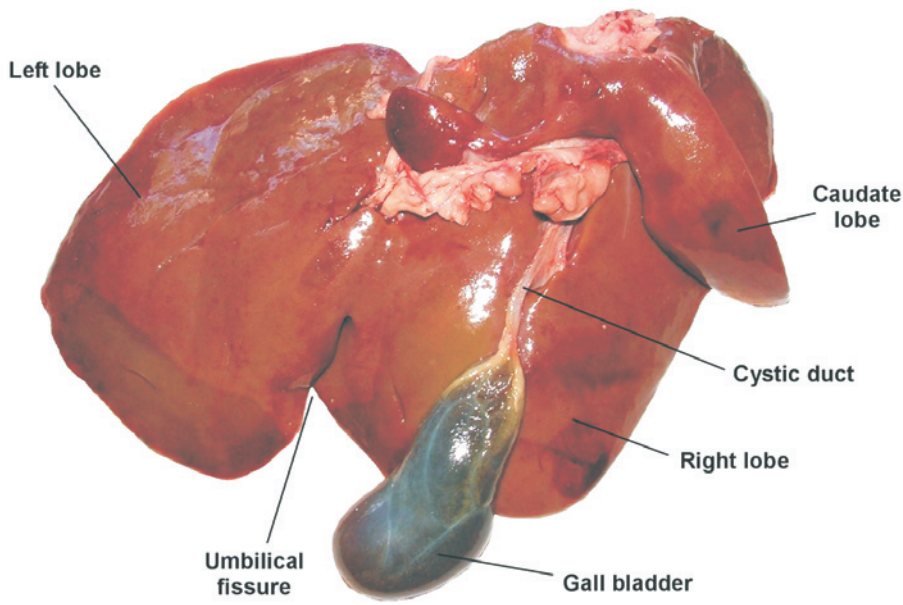
villi. This fat removal generally takes place in the jejunum giving the lymph channels leading from the intestines a milky appearance known as 'chyle'. This fluid collects in the cisterna chyle and then enters the blood circulation at the thoracic duct.

In the presence of pancreatic enzymes, proteins are degraded to amino acids and peptides, starches are reduced to glucose and other sugars and absorbed directly into the capillary beds surrounding the villi and thence to the liver via the hepatic portal vein.

THE LIVER

APPEARANCE

The ovine liver is a dark brown colour, with a smooth convex outer surface and consists of three lobes; a left and right lobe indistinctly separated at an umbilical fissure, and a caudate lobe (a cigar shaped gall bladder partially imbedded in the liver substance). The liver possesses several depressions where it is in contact with other organs. The left lobe is thinner than the right, bearing the impression of the reticulum against which it rests in the live animal. This lobe is bordered by the umbilical fissure (which occasionally bears a remnant of the umbilicus) and the oesophageal notch. The thicker right lobe is compressed by the presence of the omasum. At the centre of this lobe, normally covered by the pancreas, is situated the entry point of the portal vein and artery into the liver surrounded by the hepatic lymph nodes, and also indicates the point at which the cystic duct leaves the liver for the gall bladder.



Ovine liver – visceral surface

Joined to the right lobe of the liver is the square ended caudate lobe which blends with the right lobe at a depression formed by the presence of the right kidney, the renal impression.

When the liver is removed from the carcase a segment of the posterior vena cava remains attached.

LIVER STRUCTURE

The liver is mainly composed of small filtration and secretion units called lobules. Each lobule is enclosed in a thin connective tissue and comprises specialist liver cells, including the main liver cells (hepatocytes) surrounding a central capillary of the hepatic portal vein. The hepatocytes are segregated from the blood vessels by a basement membrane covered with basic epithelial cells and the phagocytic Kupfer cells. The hepatocytes are closely packed together but do have small canals between them that collect the produced bile and then connect together to form a network that eventually produces bile ducts. The primary cystic duct exits the liver at the portal notch and transports the bile to the gall bladder where it is stored and concentrated.

The blood supply of the liver is provided by the hepatic artery and the hepatic portal vein from the intestines. The blood from each of these mixes within the lobules before exiting the liver via hepatic veins directly into the caudal vena cava embedded in the margin of the liver.

BILE

This is produced by the hepatocytes and is a complex formula of water, pigments (biliverdin and bilirubin), fats, minerals and bile salts, the latter being produced from cholesterol by organelles within the hepatocytes. The bile salts are saponification agents; they react with the fat in food allowing it to be further broken down by lipase. Bile salts are reabsorbed in the area of the ileum, their pigments colour the faeces brown after conversion to urobilinogen by microbial action in the intestines.

FUNCTIONS OF THE LIVER

In addition to the production of bile, the liver can be considered the chemical and filtration organ of the body:

- It receives absorbed food for further processing or storage via the hepatic portal circulation where it converts short chain fatty acids into longer chain fatty acids either to be used immediately or stored within the liver. Glycogen is stored after conversion

of glucose for recombination and release into the bloodstream. In addition lactic acid formed during muscular activity is taken to the liver for recombination to glucose for immediate use or glycogen for storage.

- Essential amino acids from the body are combined within the hepatocytes to produce proteins.
- Most plasma proteins are produced within the liver, including those that assist in the regulation of blood volume.
- Essential blood clotting factors including fibrinogen are produced in the liver.
- Urea is produced in the liver after protein deamination and enters the bloodstream to be removed by the kidneys.
- The phagocytic properties of hepatic cells (Kupfer cells) remove bacteria and other invasive organisms from the blood, including that of the hepatic portal circulation. This filtration system is important, the liver acts as a line of defence against infection borne through the digestive system ideally purifying the blood before it is returned to the circulation proper.

CARDIOVASCULAR SYSTEM

INTRODUCTION

The cardiovascular system refers to the heart, blood, and blood vessels. This represents a sealed system of tubes through which a fluid media (blood) is pumped by the heart. The cardiovascular system can be considered the transportation system of the body, carrying items to the tissue cells of the body including food, messages and protection, and removing the waste products of their existence.

BLOOD

This is a high viscosity red fluid consisting of a fluid part (plasma) and cellular elements that clot rapidly on exposure to air. Plasma is a straw coloured, protein rich fluid containing water, salts, nutrients, waste products of cellular metabolism and hormones. Suspended within this fluid is the cellular portion, red and white corpuscles and platelets.

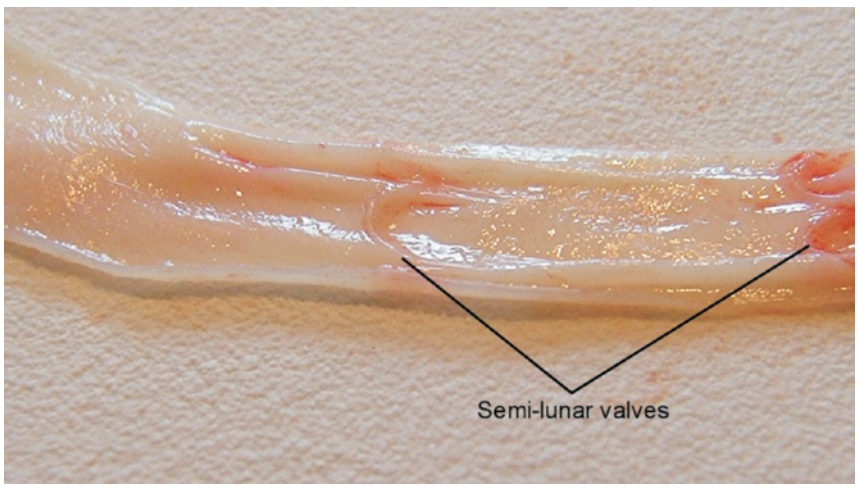
The red corpuscles, erythrocytes, are biconcave discs with a thin capsular membrane with extensions that enter the cell forming a fine internal lattice, and are distinctive in that they possess no nucleus. The membranous lattice enmeshes haemoglobin, a protein that readily converts to oxyhaemoglobin in the presence of oxygen in the lungs and transports this oxygen to the cell tissues where it is released in return for the carbon dioxide waste of the cells which binds to the haemoglobin until returned to the lungs.

The white corpuscles, leucocytes, form part of the immune system recognising and digesting foreign material including bacteria, neutralising toxin and removing dead or damaged tissue. The platelets possess no nucleus and form part of the clotting mechanism.

BLOOD VESSELS

The blood vessels carry the blood throughout the body, arteries carrying blood under pressure from the heart, veins carrying blood back to the heart. The arteries have a three layered wall; an inner wall (*tunica interna*) of endothelial cells on an elastic connective tissue membrane, a central layer (*tunica media*) comprised of smooth muscle and elastic tissue, and a fibrous outer layer (*tunica adventitia*) that provides a defence against overexpansion of the arterial wall and possible rupture. This layering allows for expansion of the artery during the pump phase of the hearts movement (systole) followed by elastic relaxation and return to normal size during the rest phase, an action which also serves to further propel the blood along the artery. The arteries

branch to smaller diameter arterioles which in turn branch into capillaries from which they are differentiated by retaining a proportion of muscle in their walls to regulate blood flow. The capillaries are thin tubes of endothelial cells on a thin connective tissue support, allowing fluid to leave the blood at the arterial end of the capillary bed to 'bathe' the tissue cells before a percentage of this fluid re-enters toward the venous portion of the capillary bed. The capillaries then reform into venuoles and then veins for return to the heart. The veins are thinner walled than arteries as they do not suffer the effects of the systolic 'pump', the muscular *tunica media* being confined to the larger veins. The *tunica interna* is thinner but forms one way valves within the vein to prevent back flow of the low pressure blood.

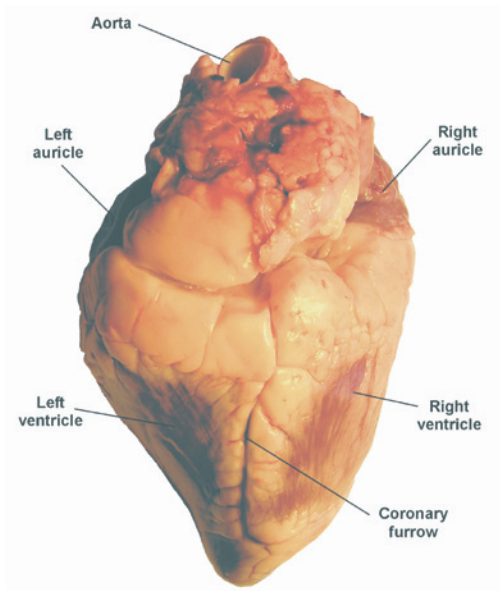


Semi-lunar valves in a dissected femoral vein

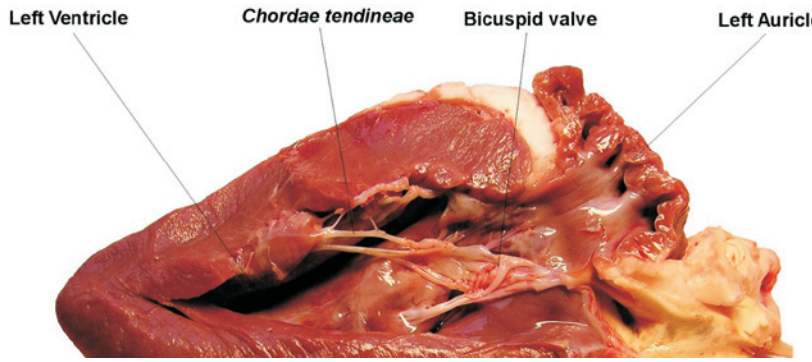
THE HEART

APPEARANCE

The ovine heart is a muscular organ, roughly conical in appearance consisting of four chambers - the right atrium and ventricle, and the left atrium and ventricle, separated by a septum. The left side of the heart has thicker muscular walls and supplies arterial blood to the body under pressure. The ventricles are divided from the atria by valves (bicuspid between the left



ventricle and atrium, tricuspid between the right ventricle and atrium); these valves are prevented from inverting under pressure by tendons, *Chordae tendineae*, that originate from the endocardium. The heart is enclosed in a serous sac, the pericardium, which is an invagination of the parietal pleura that secretes fluid to both nourish and lubricate the heart as it beats. The pericardium itself has a visceral and parietal layer, the visceral pericardium being attached to the surface of the heart forming the epicardium, the parietal pericardium containing more fibrin.



Cross section of the left side of the heart

CARDIAC MUSCLE

The heart muscle, cardiac muscle, is a specialised form of musculature. As with skeletal muscle it appears striated microscopically and contains the sarcomeres associated with skeletal muscle. Cardiac muscle is differentiated from skeletal muscle tissue in unique ways; the two main differences are by-products of the method of function of the heart muscle. Firstly the cardiac muscle is formed by single cells containing a single nucleus as opposed to the multi-nucleated fusion of myoblasts that form skeletal muscle. The second notable difference is that the myofibrils of actin and myosin are branched in cardiac muscle and interlock with those of adjacent fibres to form immensely strong junctions that prevent the fibres separating during the forceful contractions associated with a heart beat.

The contraction of the heart muscle is set up by the fibres themselves; the contraction of one sarcomere initialising contraction of its neighbour via gap junctions, thereby creating a wave effect of contraction starting at the atrium and proceeding down the ventricle forcing blood from the heart. The nerve supply to the heart simply offers only modulation of this beat, either speed or strength. The table below illustrates the basic difference between cardiac and skeletal muscle.

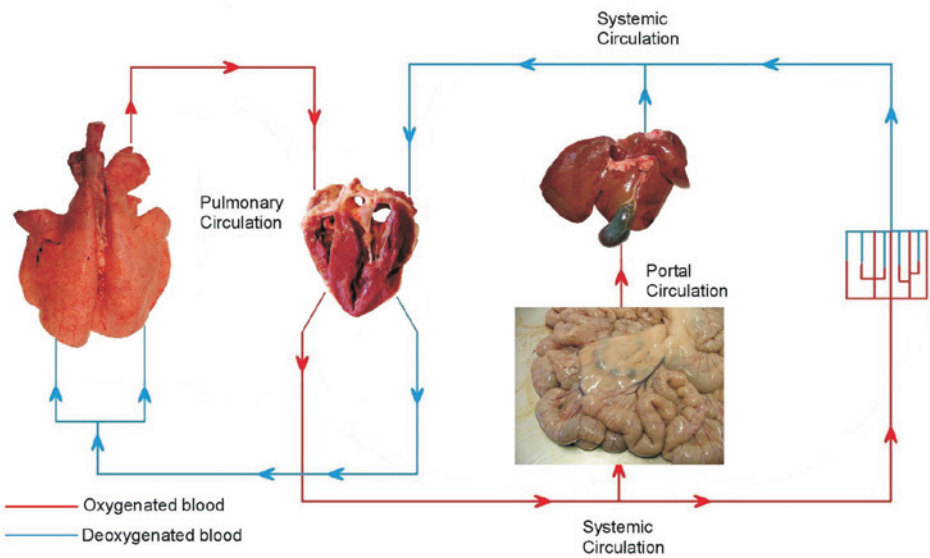
Skeletal Muscle	*Cardiac Muscle*
Multi-nucleated, fusion of myoblasts	Single celled fibres, single nucleus
ATP derived from three sources, creatine phosphate, glycogen, and cellular respiration	ATP almost exclusively via cellular respiration, thus more mitochondria
Myofibrils separated	Myofibrils branched and interlocked
Motor nerve stimulates contraction	Motor nerve modulates contraction
Glycogen stores ten times larger than quantity of ATP stored	Small amount of glycogen stored

BLOOD CIRCULATION

The circulation of the blood throughout the body can be subdivided into three stages or circuits; the pulmonary circulation, the systemic circulation and the portal circulation.

PULMONARY CIRCULATION

This system provides for the reoxygenation of the blood. Blood is pumped from the right ventricle via the pulmonary artery which bifurcates to enter each side of the lungs where further subdivision of the arteries to arterioles and capillaries occurs. Once gaseous exchange takes place the pulmonary capillaries unite to form veins to take the purified blood back to the left auricle of the heart.



SYSTEMIC CIRCULATION

The oxygenated blood then passes from the left auricle to the left ventricle via the bicuspid valve and is pumped through the aorta from which arteries branch off followed by further subdivision into arterioles and capillaries conveying the blood to all the tissues of the body. These capillaries then reunite to form venuoles and veins which carry the impure deoxygenated blood back to the heart via the anterior and posterior vena cava.

PORTAL CIRCULATION

The portal circulation carries the blood from the intestines and stomachs to the liver via the portal vein. Once the blood has passed through the liver, the nutrients removed for conversion or added to the blood by the liver, it passes into the normal circulation by being discharged into the posterior vena cava or via the hepatic vein back to the heart.

BLOOD CLOTTING

The clotting of blood is achieved by the action of platelets in conjunction with the protein fibrinogen which is carried in the plasma. On exposure to air platelets bind together and release thrombokinase. Thrombokinase reacts with prothrombin, produced by the liver, and plasma calcium to produce the enzyme thrombin which then converts fibrinogen to fibrin. The filamentous fibrin then forms a lattice of insoluble material that enmeshes corpuscles to form a clot. Blood serum is plasma from which fibrinogen has been removed during clotting.

THE LYMPHATIC SYSTEM

LYMPH VESSELS

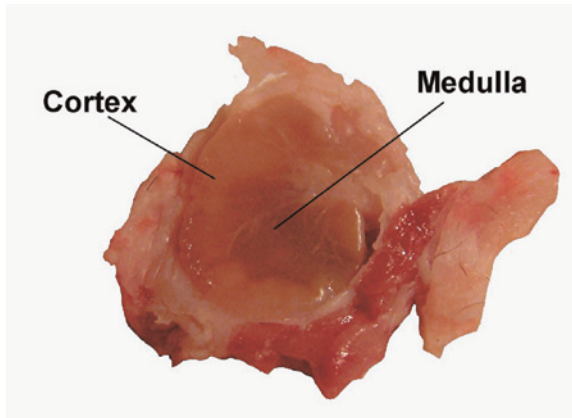
These are thin walled, blind-ended, tubes that operate in association with the blood circulatory system of blood vessels and capillary beds. The fluid they contain performs the function of bathing the tissue cells, providing them with nutrients, hormones and oxygen before removing carbon dioxide and waste material back to the blood stream. The blood capillaries of the area remove the majority of the carbon dioxide; particulate matter is taken up by the tissue fluid and removed to the lymphatic circulation.

LYMPH

This is a fluid, similar to blood plasma, which enters the lymphatic system. It contains significantly less protein than blood plasma and as a consequence is less viscous and able to move in and out of the vessels easily.

LYMPH NODES

The ovine body has lymph nodes situated at specific points, each one acting as a filter for lymph fluid drained from a particular area, and as such the change of state of a node is an indicator of infection in that drainage area. Therefore knowledge of the position and normal appearance of these nodes and their respective drainage areas is of the utmost importance to the meat inspector.

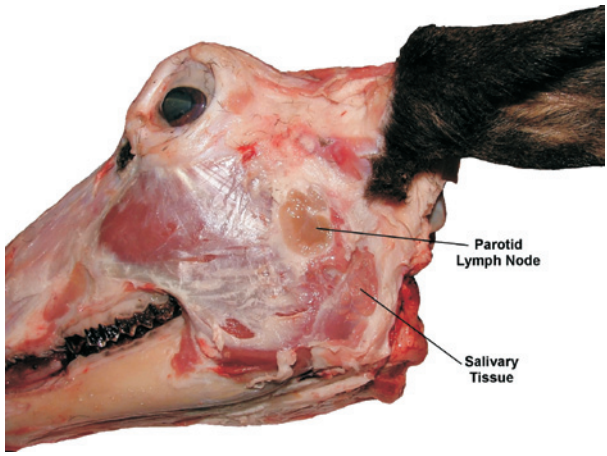


Before returning to the blood circulation lymph fluid passes through at least one lymph node. These are accumulations of lymphoid material encapsulated within a connective tissue shell, forming a firm oval, or more generally bean shaped node. The lymph entering a node is termed afferent lymph; that leaving the node is termed the efferent lymph. Lymph nodes consist of two regions; the medulla and cortex, each of these regions has a specific function. The cortex contains germinal centres in which continual lymphocyte production takes place, and the medulla contains a lattice containing phagocytic cells. The lymph fluid enters the capsule through afferent vessels entering the surface of the node and into the sub capsular sinus, the fluid then percolates through the cortical region collecting more lymphocytes and then through the medulla where any foreign particular matter, including bacteria, is removed by phagocytosis before the lymph leaves the node through the efferent vessels situated at the hilus of the node.

POSITION OF NODES AND DRAINAGE AREAS

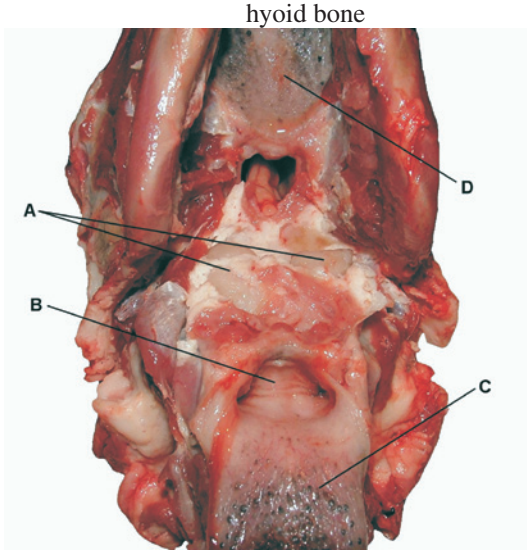
Head

Node	*Position*	*Afferent*	*Efferent*
Parotid	Ventral to mandibular joint, overlying masseter muscle	Dorsal half of the head	Lateral retropharyngeal



Position of parotid lymph node

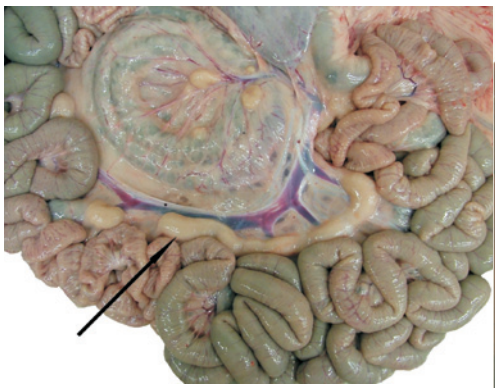
Submaxillary	Medial to mandible	Nasal mucosa and ventral area of the head including larynx	Lateral retropharyngeal
Retropharyngeal	Dorsal to pharynx medial to long horn of hyoid bone	Deep tissues of the head	Lateral retropharyngeal



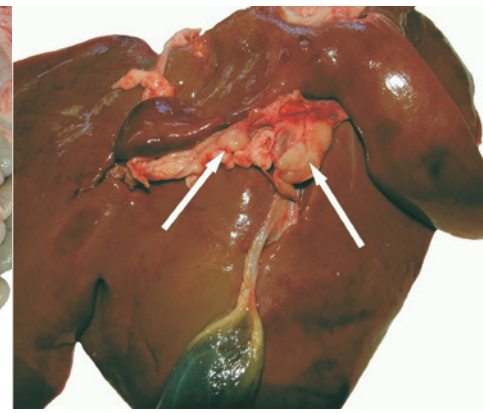
Interior of ovine buccal cavity, incised through rear of pharynx. A, retropharyngeal lymph nodes. B, Epiglottis. C, tongue. D, Roof of buccal cavity (soft palate).

Abdominal Viscera

Node	*Position*	*Afferent*	*Efferent*
Mesenteric nodes	Chain from duodenum to caecum in fat of mesentery	Jejunum and ileum	Cisterna chyli or local lymph trunks
Renal	In fat at hilus of kidney	Kidneys	Cisterna chyli
Hepatic portal	On visceral surface of liver in area of portal vein and hepatic artery.	Liver, pancreas and duodenum	Intestinal lymph trunks and mediastinal nodes



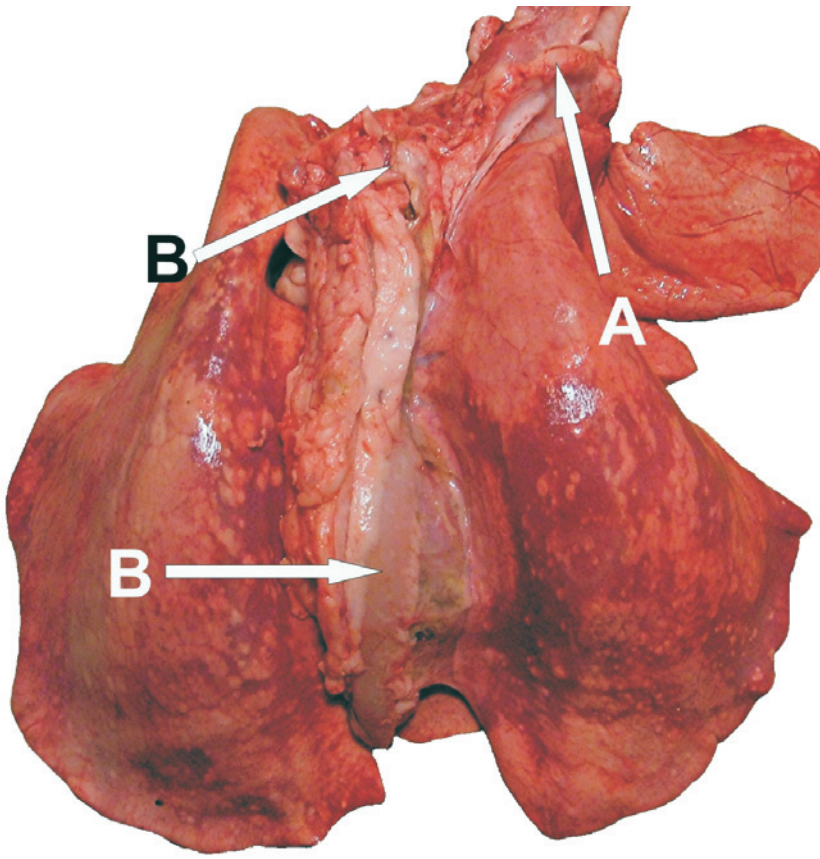
Mesenteric lymph nodes



Hepatic Lymph nodes

Lungs

Node	*Position*	*Afferent*	*Efferent*
Bronchial	Caudal to the bifurcation of the trachea	Trachea, heart, pulmonary and thoracic aortic nodes	Mediastinal nodes or thoracic duct
Mediastinal	Chain of nodes running, in fat, from cranial to heart down the oesophagus to caudal to the heart	Mediastinal tissue, diaphragm, spleen, liver, parietal pleura and intercostals, bronchial and aortic nodes	Cervical nodes



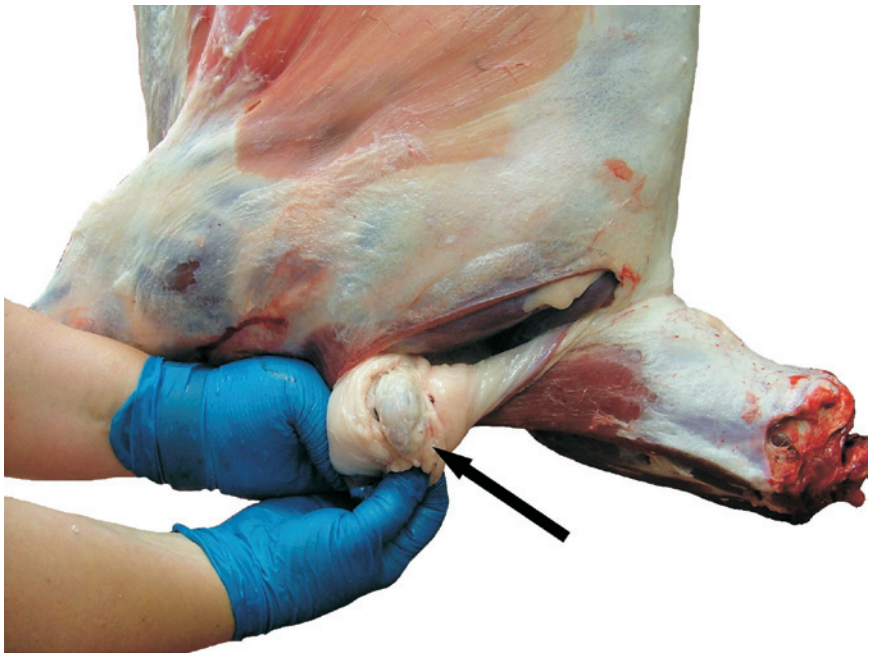
Lung lymph nodes. A- Bronchial. B- Mediastinal

Neck

Node	*Position*	*Afferent*	*Efferent*
Lateral retropharyngeal	Ventral to wing of the atlas bone	Caudal head, cranial neck, parotid and submaxillary nodes	Forms the tracheal duct
Deep cervical	Close to trachea and oesophagus	Local tissues	Tracheal trunk

Forelimb

Node	*Position*	*Afferent*	*Efferent*
Prescapular	On cranial border of the supraspinatus muscle.	Local tissues, head nodes, forelimb and cranial mammary glands	Cervical nodes

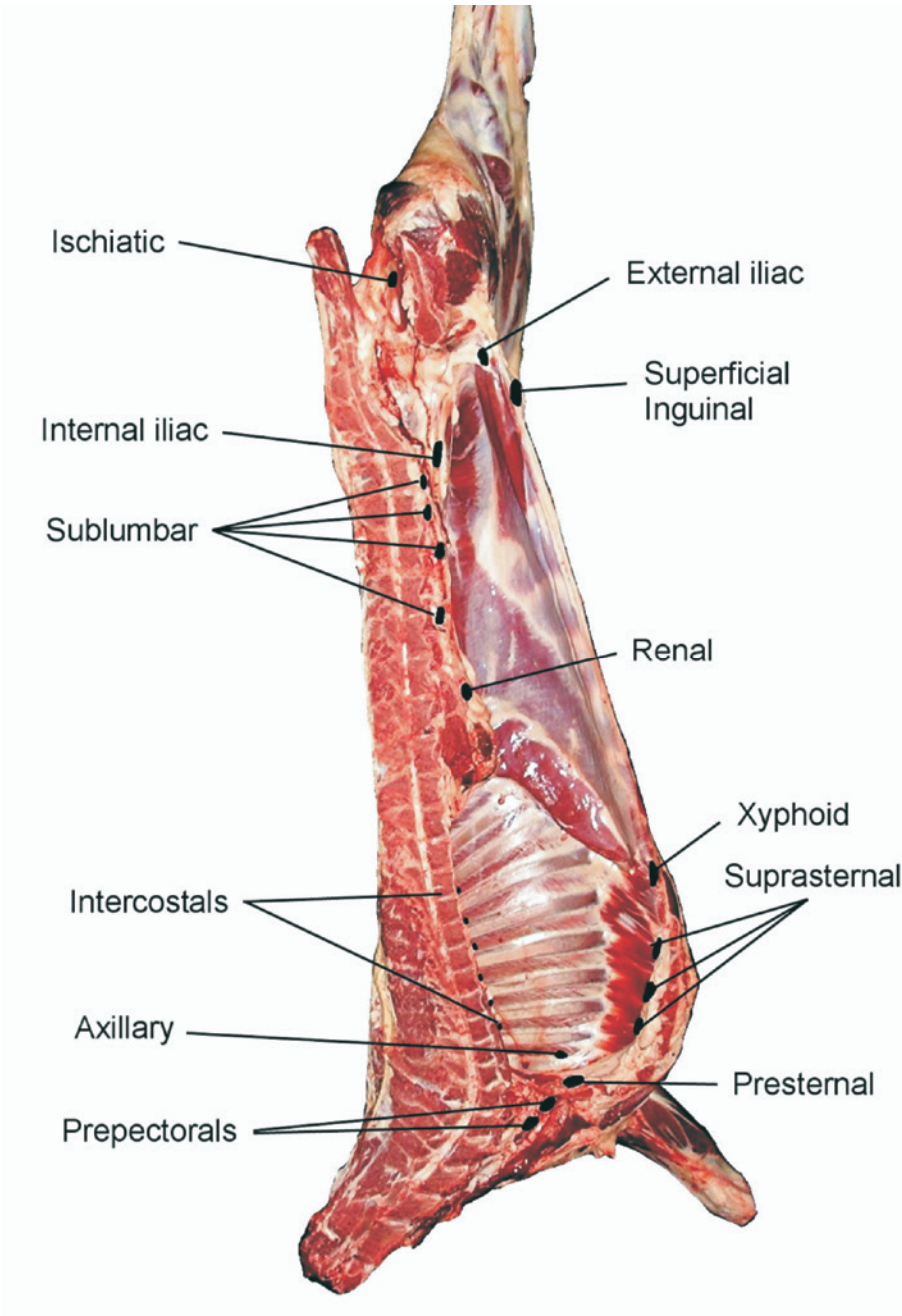


Prescapular lymph node (arrowed)

Axillary	In the second intercostals space 6-10cm caudal to the shoulder joint.	Local tissues of the shoulder and foreleg	Deep cervical

Thorax

Node	*Position*	*Afferent*	*Efferent*
Intercostal	Intercostal spaces at the level of the rib heads.	Chest wall, diaphragm and vertebrae	Mediastinal and thoracic aortic nodes
Sub dorsal (thoracic)	Dorsal border of aorta and T5-T13 vertebrae	Shoulder region, chest wall, diaphragm, heart, spleen, pleura and peritoneum, mediastinum, and intercostals and mediastinal nodes	Thoracic duct



Lymph nodes of the ovine carcase – Internal view

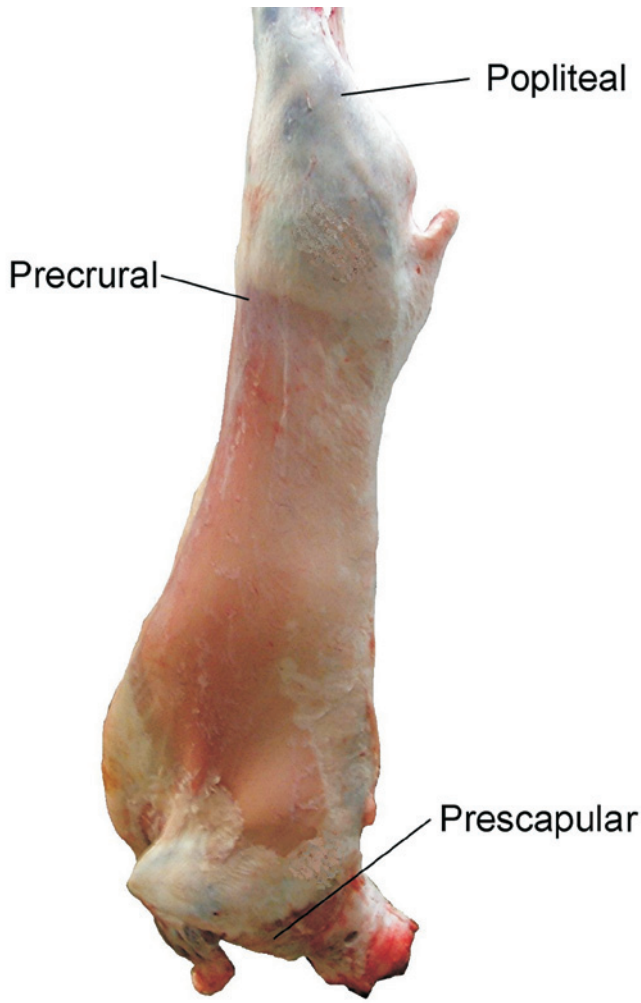
Presternal	Ventral border of internal thoracic vessels level with first costal cartilage	Ventral chest wall, mediastinum, diaphragm,	Thoracic and tracheal trunk.
Suprasternal & Xiphoid	Along course of internal thoracic vessels	Diaphragm, pericardium, chest wall, liver, pectoral and abdominal muscles	Cranial sternal nodes

Abdomen

Node	*Position*	*Afferent*	*Efferent*
Sublumbar	Chain of small nodes each side of aorta and caudal vena cava	Lumbar region, abdominal wall, iliac nodes	Lumbar trunks and cisterna chyli
Internal iliac	Ventral to psoas minor muscles.	Caudal half of abdominal wall, pelvic limb, urogenitalia, and superficial inguinal, popliteal and lateral iliac nodes	Lumbar trunks to cisterna chyli
External iliac	Either side of the bifurcation of the deep circumflex iliac vessels	Abdomen, deep gluteal and tensor fasciae latae muscles, peritoneum and sub iliac nodes	Lumbar aortic and internal iliac nodes
Superficial inguinal (male) Supramammary (female)	Male: Scrotal nodes between cod fat and abdominal wall. Female: Usually in a straight line with the cut pubic tubercle	External genitalia, abdominal wall, skin of hind limb and the udder in females	Internal iliac nodes
Ischiatic	Near sciatic nerve. Exposed by a deep incision through the middle gluteal muscle on a vertical line midway between the sacrum and the caudal part of the ischium	Pelvic and hip regions and popliteal node	Internal iliac nodes

Hindlimb

Node	*Position*	*Afferent*	*Efferent*
Precrural	Midway between patella and tuber coxae of ilium	Thigh, abdominal wall, umbilicus and caudal part of thorax.	Iliac nodes
Popliteal	Caudal to stifle joint, found by separating biceps and semitendonosus muscles.	Distal limb	Medial iliac and ischiatic nodes



Lymph nodes of the ovine carcase – External view

LYMPHOID TISSUE

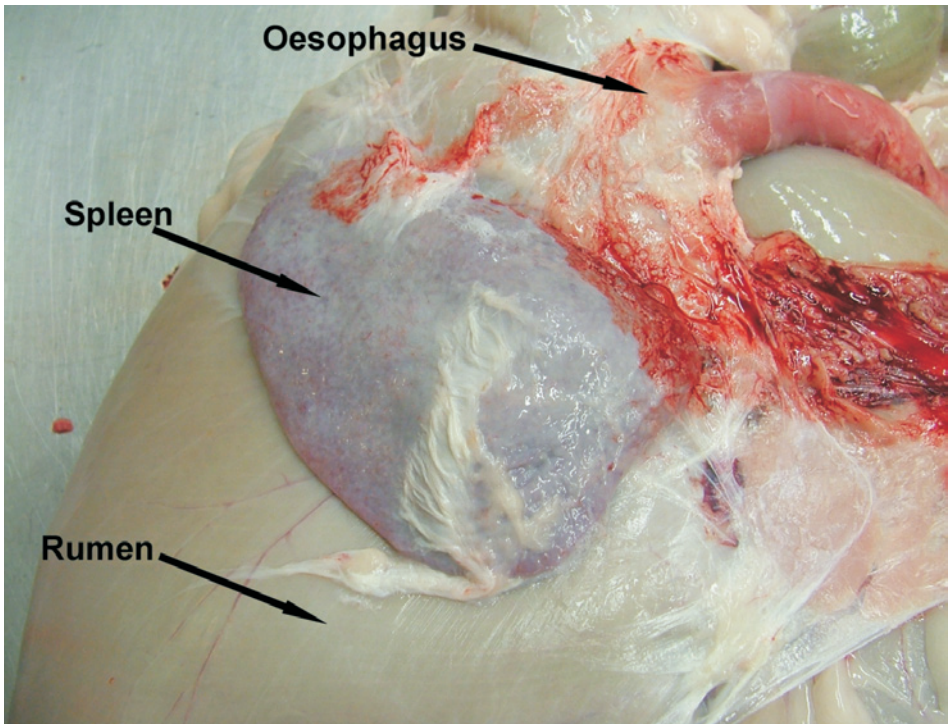
Lymphoid tissue also forms part of the lymphoid system; however it is not as formalised and discrete as lymph nodes. Lymphoid tissue is found throughout the body such as in the liver and in the bronchi of the lungs and represents a method of localised response to pathogenic particles. Lymphoid tissue is primarily a latticework of tissue containing interspatial lymphocytes. The accumulation of the tissue tends to be in areas where infectious organisms may gain entry into the body; most noticeable amongst these is the lymphoid tissue of the digestive system: the gut associated lymphoid tissue (GALT). This includes the tonsils, the connective tissue layer of the mucous membrane of the intestines and the Peyer's patches, the latter of which are recognised as raised oval shaped areas in the mucosal and sub mucosal layers of the lower intestine.

THE SPLEEN

The spleen, also known as the 'melt', is a mottled greyish-blue organ that is described as oyster shaped. In life it is positioned in the upper left hand side of the abdominal cavity attached to the rumen by the omentum. It consists of a firm capsule enclosing red and white pulp into which the capsule extends forming a supporting network called trabeculae.

Incision through spleen exposing 'pulp'

The spleen is supplied by the splenic artery which branches from the celiac artery. The white pulp can be observed when the spleen is incised and represents areas of lymphoid tissue aggregated near the arteries that pass through the spleen. These areas of white pulp (Malphigian corpuscles) possess lymphogenic and phagocytic properties, removing old and diseased red blood cells and particulate matter from the circulation in addition to adding lymphocytes to the blood leaving the spleen via the splenic vein to the portal vein.



Position of spleen on exterior of rumen

The red pulp acts as a blood storage reservoir in addition to the production of erythrocytes, the latter function utilising the iron stored in the spleen after the destruction of damaged erythrocytes by the white pulp.

The splenic capsule and substance is innervated by nerves entering with the artery, and possesses the ability to contract forcing stored blood into the circulation in times of need.

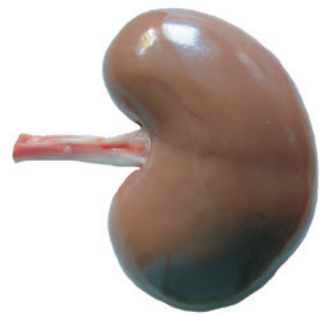
UROGENITAL SYSTEM

URINARY TRACT

INTRODUCTION

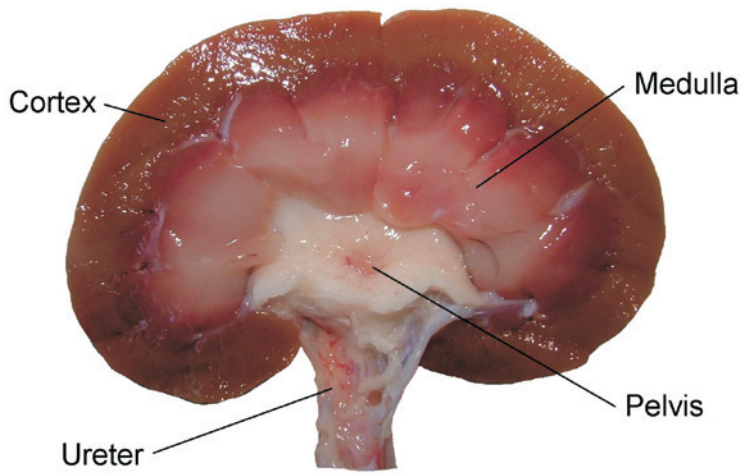
The deamination of proteins in the liver releases toxic nitrogenous compounds that are subsequently converted to urea and released into the blood circulation. The main function of the urinary system is the filtration of toxic substances, including urea, from the blood and then excreting them from the body. This filtration and excretion system consists of paired kidneys, paired ureters, a bladder and a single urethra.

KIDNEY STRUCTURE



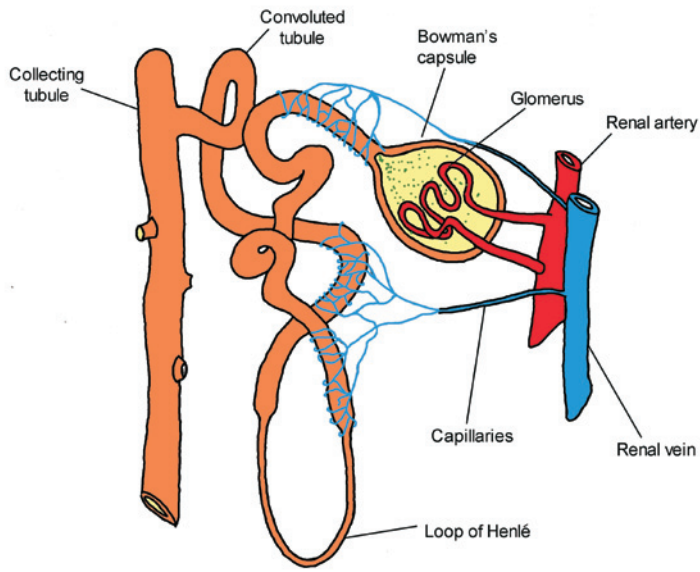
Ovine kidneys are brown coloured bean shaped organs; both kidneys are embedded in thick renal fat (cod fat) in a position lateral to the vertebrae. The right kidney is held tight against the body, the left has a looser attachment enabling movement of the organ in relation to the expansion of the rumen, thereby preventing the kidney from being crushed. The left kidney is also characterised by a slight rotation about its axis. The kidneys are further covered by a membranous renal capsule that can be easily peeled in the healthy animal.

In cross section each lobe of the kidney is found to contain a cortex, medulla, calyx and renal pelvis, the latter of which forms the ureter.



Gross structure of an ovine kidney

The renal cortex contains nephrons, each comprising a glomerus and tubule in units known as Malphigian bodies. The glomerus is basically a knot of blood capillaries surrounded by a capsule (Bowman's capsule). One side of the capsule joins a collecting tubule that is surrounded by blood capillaries. The tubule, at first convoluted, extends down into the medulla before returning back to the cortex, the loop of the tubule in the medulla being the loop of Henlé.



Schematic of a nephron. Bowman's capsule peeled to show glomerus

FUNCTION OF THE KIDNEY

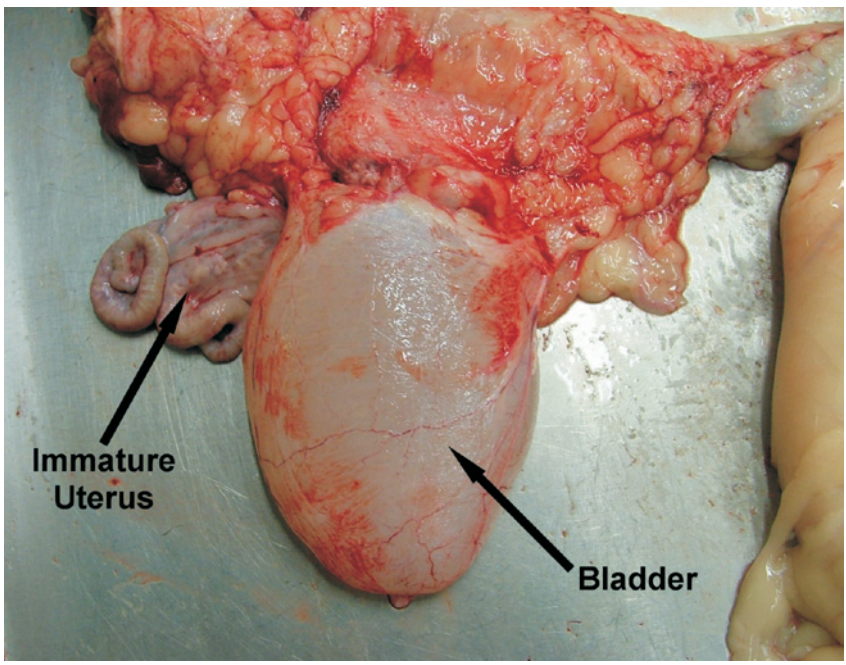
Blood at arterial pressure enters the glomerus which forces the fluid portion of the blood into the Bowman's capsule. This fluid then moves down the tubule during which time the majority is reabsorbed into the capillaries that surround it. Approximately 20% of the fluid and the urea, etc, remains to enter the main collecting tubule to trickle into the renal pelvis.

URETERS

The renal pelvis is formed by the expanded ureter, down which the urine trickles into the bladder. The ureters are thin walled tubes with a coat of fibrous tissue surrounding a layer of involuntary muscle and an internal mucous membrane. The ureters pass along the body of the bladder before discharging near its neck.

BLADDER

The bladder is a pear shaped distensible sac that possesses a similar wall structure to the ureters and is situated on the pubic bone but, when full, extends into the abdominal cavity under the peritoneum. The control of urine entering the bladder is physical; as the bladder expands it exerts pressure on the ureters as they pass over its body and reduce the flow of urine into the bladder. This action also has the effect of preventing reflux of urine from the bladder into the ureters. The neck of the bladder contains a ring of involuntary sphincter muscle which, in conjunction with an external skeletal sphincter muscle under conscious control of the animal, regulates the flow of urine into the urethra.



Ovine urinary bladder

URETHRA

A single urethra emanates from the neck of the bladder and follows the floor of the pelvis to which it is held by fat and connective tissue. In the female the urethra is short; the dorsal surface is also attached to the outer wall of the vagina into which it discharges in the vestibule ventral to the hymen. In males the urethra consists of a pelvic and a penile area, continuing to the tip of the penis.

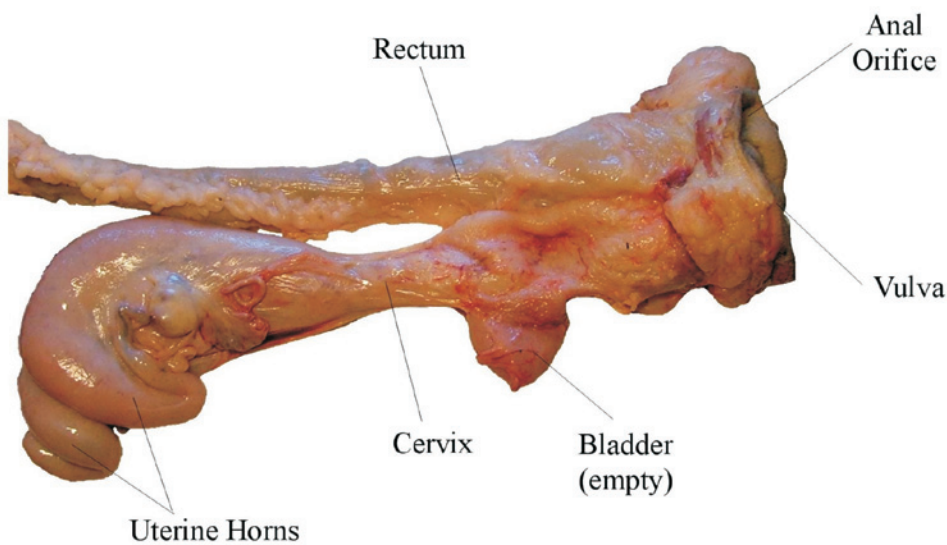
REPRODUCTIVE SYSTEM

FEMALE REPRODUCTIVE SYSTEM

INTRODUCTION

The reproductive system of the female ovine has specific functions; to produce the macrogametocyte (the ova, or eggs, to provide 50% of the genetic material of the lamb), to provide an environment and opportunity for fertilisation of the egg, to provide a safe environment for the development and nutritional needs of the growing foetus, and to provide a means of expelling the lamb by parturition (birth) once developed.

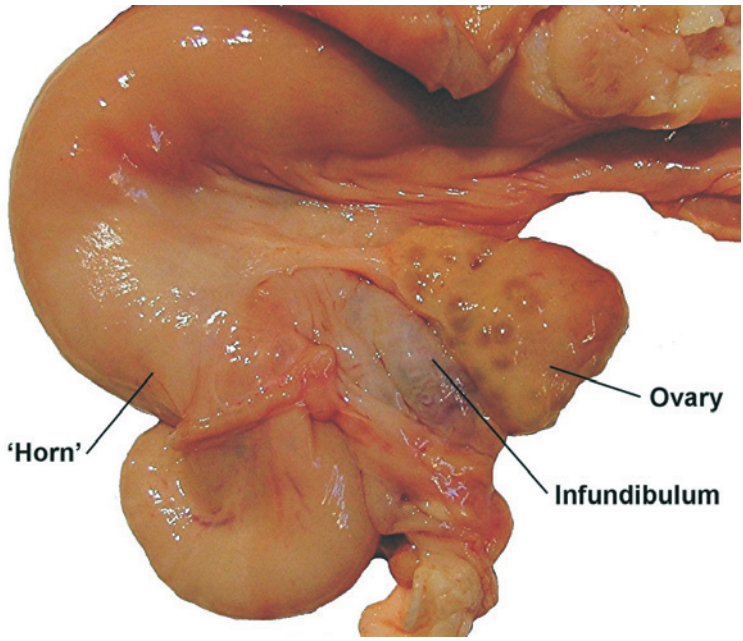
The female reproductive system, consisting of paired ovaries, oviducts, single uterus, single cervix and single vagina, lies parallel and ventral to the rectum, being attached to this by membrane and fat. The reproductive system terminates externally at the vulva.



Female reproductive tract in relation to the terminal large intestine. Fat and supportive ligament removed

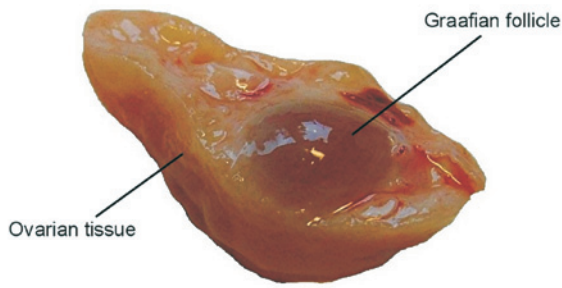
OVARIES

There are two ovaries (left and right) whose functions include the formation of eggs and the secretion of the hormones oestrogen and progesterone. Eggs are produced and matured in primordial follicles; water filled cavities within the ovary consisting of an outer layer of oestrogen producing cells.



Detail of ovary

The formation of these cyst-like bodies (follicles), and the subsequent maturation of the eggs, is stimulated by Follicle Stimulating Hormone (FSH) produced within the pituitary gland. The level of a second hormone also produced in the pituitary gland, Lutenizing Hormone (LH), stimulates the secretion of oestrogen by the follicle cells and finally prompts the release of the ovum from the follicle (ovulation) after the follicle ripens into a mature cyst known as a Graafian follicle.



Graafian follicle

Once the ovum has left the ruptured follicle, the latter degenerates into a yellowish glandular mass, the *corpus luteum*, which secretes progesterone, a hormone that aids implantation of a fertilised ovum within the uterus. If conception does not occur, the *corpus luteum* regresses and a new follicle forms. If conception occurs, the *corpus luteum* regresses in the latter stages of the pregnancy to be replaced by white fibrous tissue at which point it is termed the *corpus albicans*.

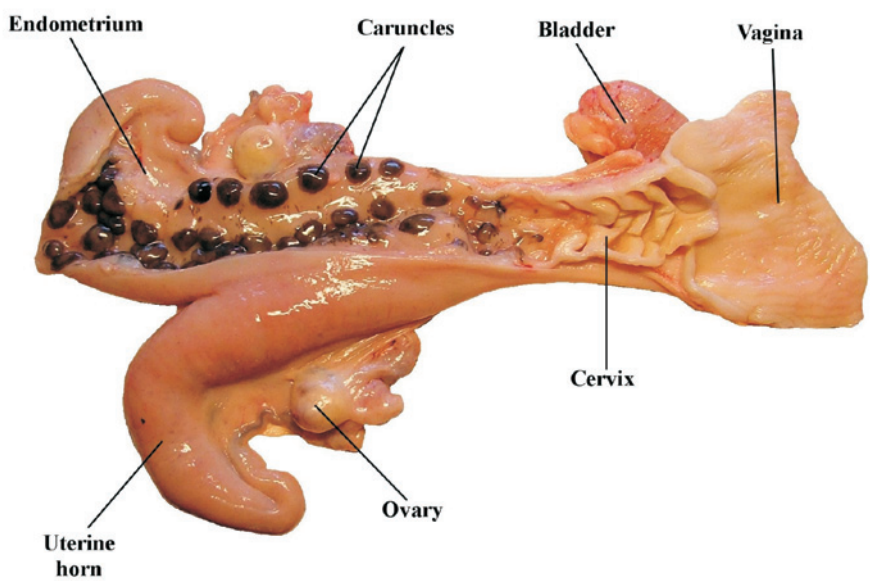
UTERINE HORNS

These paired structures are embedded in a uterine ligament which holds them in position against the ileum. The ovaries are surrounded by finger-like projections of the funnel-shaped infundibulum designed to guide released ova into the body of the uterine horns which join to form a single uterine body. The uterus and uterine horns are comprised of an internal mucosal membrane(endometrium) containing glands whose size vary with the stage of the reproductive cycle, a muscular wall (myometrium) of both inner circular and outer longitudinal muscles the contractions of which at parturition expel the lamb, and a final outer membrane (perimetrium) that is formed by the uterine ligament.

The endometrium also serves as the site of attachment of the maternal side of the placenta, the position of which is determined by the presence of up to 100 cotyledons (caruncles), circular button-like structures approximately 5-10mm diameter in the non-pregnant ewe but expanding during pregnancy.

CERVIX

The cervix represents the terminus of the uterus, a cervical canal surrounded by five muscular rings that, in conjunction with a mucous plug, form an impervious sphincter that relaxes during oestrus to allow sperm to pass through and dilates at parturition to allow the lamb to be born.



A dissected uterus, illustrating the banding of the cervix and the presence of cotyledons

THE VAGINA

The vagina starts at the cervical canal and terminates at the vulva. It has a highly elastic structure and lies in the pelvis and is attached dorsally to the ventral surface of the rectum. In addition to being the site at which the urethra terminates at the external urethral orifice on the floor of the vagina, the vagina accepts the penis and sperm from the male during copulation.

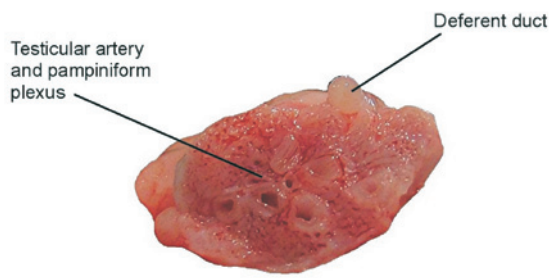
MALE REPRODUCTIVE SYSTEM

INTRODUCTION

The male reproductive system consists of paired testicles suspended in a scrotal sac, the penis, and glands such as the bulbourethral gland and the seminal vesicles. The function of these being to produce, mature, and deliver half of the genetic material (spermatozoa) required to produce a foetus in conjunction with the female ova.

TESTES

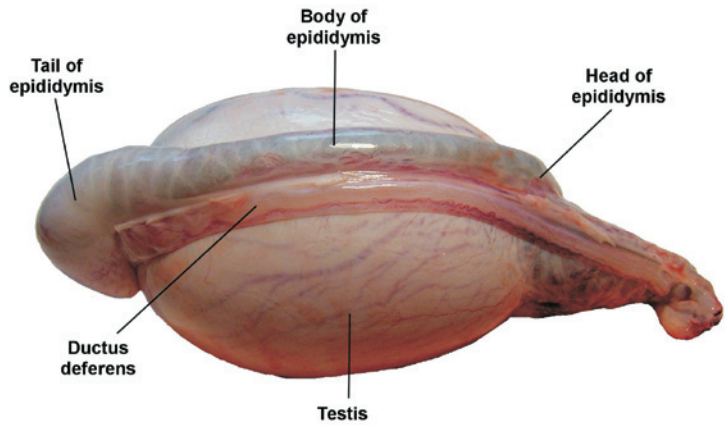
A single rams' testicle weighs approximately 300g and is oval, the length generally being twice the breadth. These glandular organs hang in a scrotal sac formed by a fold in the abdomen between the hind legs of the ram. The internal surface of the scrotum is continuous with the peritoneum and is smooth and serosal. The paired testes are suspended in the scrotum by the spermatic cord, which contains the red cremaster muscle enabling the testes to be individually raised or lowered in the scrotum in response to external temperatures (as the production of sperm is highly temperature dependant), in addition to the testicular artery and the pampiniform plexus (a convoluted series of the testicular veins that spiral around the artery). The *ductus deferens* that carries the spermatozoa to the urethra is attached to the visceral vaginal tunic that surrounds the spermatic cord.



Transection of the spermatic cord

On the median line of each testicle is the epididymis, folded back on itself at each pole forming a semi-pyramidal extension to the base of the testicle as it hangs in the scrotal sac. The epididymis is recognised as having three zones - the head leaving the testicle, the body, and the tail - and provides storage for the maturation of sperm prior to ejaculation.

The *ductus deferens,* also known as the *vas deferens,* is the tubular continuation of the epididymis and provides the linkage between the epididymis and the urethra for the transportation of the sperm.



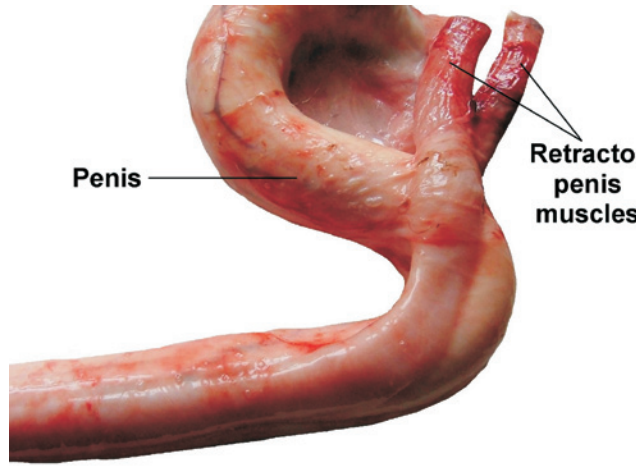
Ovine testicle

On dissection, the testes are found to comprise a yellowish coloured lobulated substance formed by tightly coiled tubes called seminiferous tubules in which the sperm are produced, and leydig cells which secrete the hormone testosterone.

URETHRA AND PENIS

The penis has a dual role, as a delivery system for semen during copulation and the expulsion of urine from the body, both functions utilising the urethra that extends from the neck of the urinary bladder to the tip of the penis. The *ductus deferens* from each testicle passes dorsally over the bladder, at which point the mucosa of the duct is thickened by glandular cells to form the cylindrical ampullae which lie next to the lobulated vesicular glands, whereupon the *ductus deferens* enter the urethra.

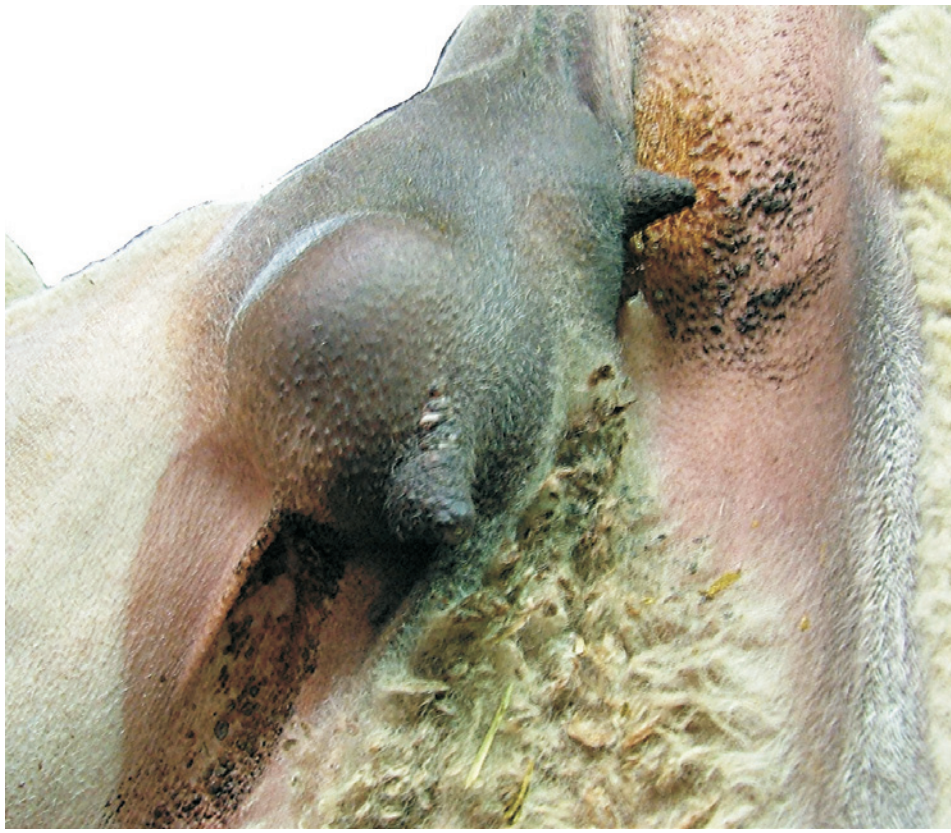
The pelvic portion of the urethra continues from the urinary bladder to the bulbourethral gland which adjoins the urethra via a small diverticulum. It is at this point that the origin is situated of the *corpus cavernosum*, the column of erectile tissue that forms the main body of the penis. This origin, termed the crus penis is usually left near the ischial arch on the dressed carcase and can be used as a method of gender identification.



The penis proper is composed of three columns of tissue, with a rich arterial blood supply, that surround the hollow urethra. Contained within a fibrous tunic and roughly circular in section, the dorsal aspect of the penis is formed by the two fused *corpus cavernosae* with the urethra surrounded by the *corpus spongiosum* carried in a ventral groove. The columns of *corpus cavernosae* terminate at the glans of the penis which is formed by the *corpus spongiosum*. In addition to the columns of erectile tissue the penis also contains a large proportion of connective tissue, and is characterised by the presence of a sigmoid flexure (an 'S' shaped portion) that straightens out when the penis becomes erect and is retracted, post copulation, by the retractor penis muscles following disgorgement of blood from the erectile tissue.

MAMMARY GLANDS

The mammary glands are modified sweat glands containing connective tissue, blood vessels, lymphatic vessels and glandular tissue. Alveoli within the gland tissue secrete and store milk which is then collected in ducts and delivered to the teats. Sheep possess two mammary glands; each with a teat, combined to form the udder. The udder is visibly separated into two halves along the median plane by the presence of suspensory sheets; the division between the two glands on each side of the separation is indistinct as the glandular tissue of one gland tends to fuse with that of the other. The milk produced collects in interconnecting ducts before entering the teat cistern. The teat contains a single duct, usually held closed by a combination of the naturally elasticity of the connective tissue of the teat, and an encircling layer of smooth muscle.



Ovine mammary glands

NERVOUS SYSTEM

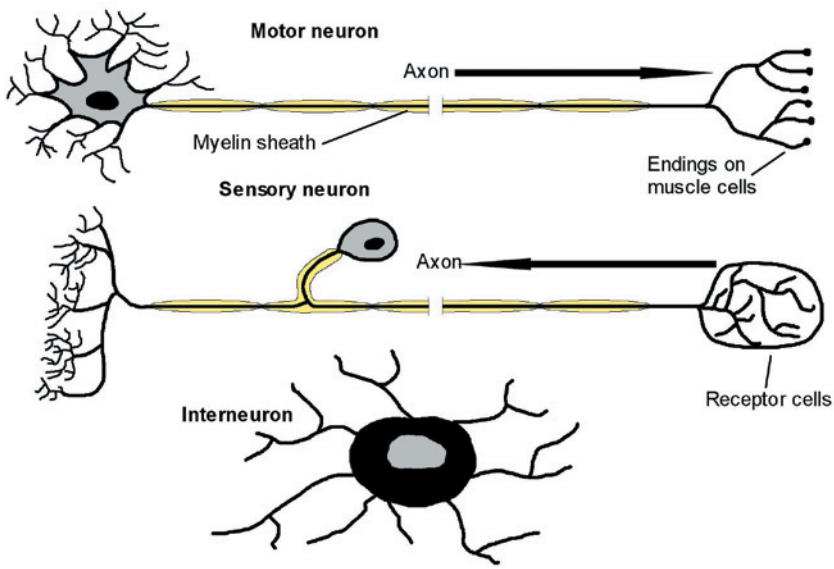
INTRODUCTION

The nervous system is the body's messenger system, nerves prompting activities such as muscular action, organ activity and hormone secretion, as well as informing the brain of the condition of the body systems and the environment. The nervous system is divided into two regions, the Central Nervous System (the brain and spinal cord) and the Peripheral Nervous System (organs, muscles, skin etc.) These systems themselves function on two levels, the somatic nervous system, co-coordinating impulses that are under conscious control, and the autonomic system, controlling involuntary action such as heart beat, organ function and hormone secretion by glands.

NERVES

Nerves are non-microscopic, string like structures formed by bundled nerve cells that pass impulses between the central nervous system and parts, or organs, of the body. There are three basic forms of nerve, categorized by their function and the direction of the impulses; these nerve types being efferent, sensory, and intermediate. Efferent nerves (also known as motor nerves) transmit impulses from the central nervous system usually to groups of muscles, sensory nerves convey impulses such as temperature or pain to the central nervous system, and intermediate nerves transmit impulses both ways.

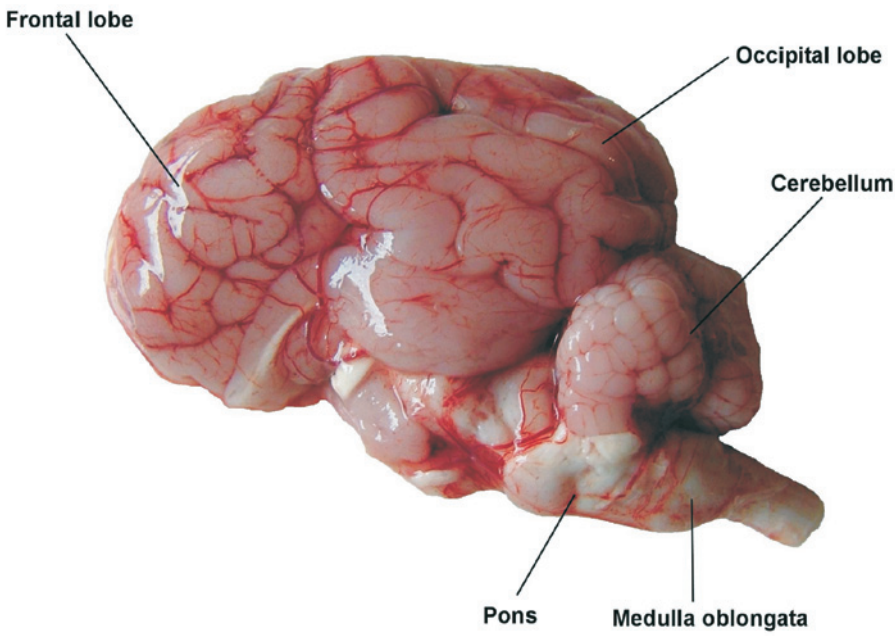
NERVE CELLS



The nerve cell, or neuron, is the basis of the nervous system. The cell body is surrounded by dendrites, extensions of the cell wall, of which one is elongated to form the axon. During neuron development the axon is surrounded by specialist cells (Schwann cells) that spiral around the axon; this envelope of cells then secretes a layer of myelin forming an insulating sheath. Bundles of axons form the nerves; the sciatic nerve serving the hind leg contains axons of over a metre in length. The nerve chain forms circuits throughout the body, the cellular electrical impulse travelling along the axon from one nerve cell to the dendrite of another. Each nerve cell is separated from the next by a junction gap (synapse) across which chemical transmitters cross causing cellular excitement and a continuation of the impulse. Certain drugs, including alcohol, interfere with this system.

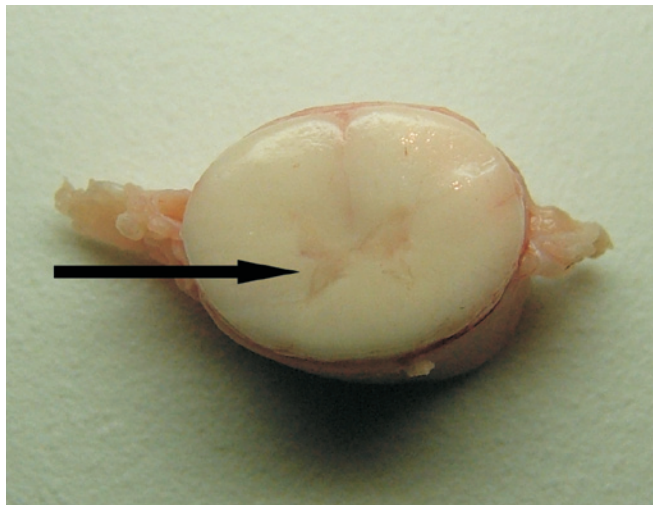
CENTRAL NERVOUS SYSTEM

The brain is situated in the cranial cavity of the skull and consists of two pear shaped hemispheres comprising the cerebrum and cerebellum, the former consisting of the frontal and occipital lobes. The base of the brain forms the medulla oblongata.

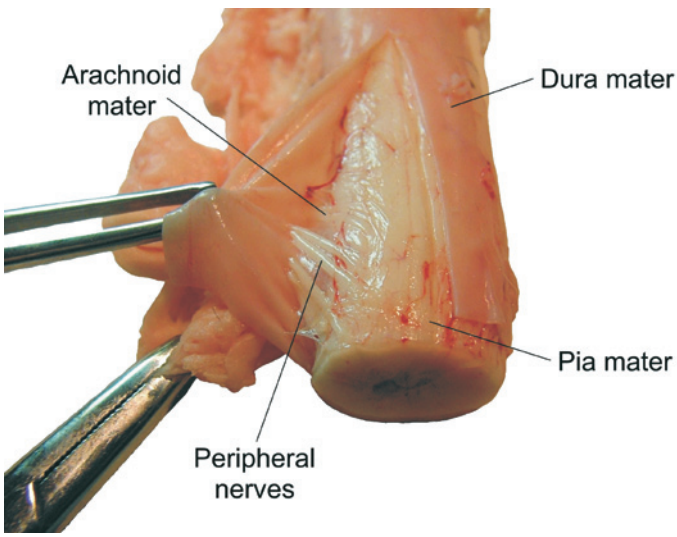


OVINE BRAIN

The spinal cord originates from the medulla oblongata and runs through a protective channel formed by the hollow mid-section of the spinal vertebrae down to the last free vertebrae of the tail.



A cross section of the spinal cord (Grey matter arrowed)



Dissection of the spinal cord meninges

In cross section a 'butterfly' shaped area of grey matter can be seen surrounded by white matter. The grey matter is formed by interneurons; the white matter is created by the myelin sheath of the axons passing down the length of the spinal cord.

The spinal cord is covered by three layers of protective tissue; the meninges, consisting of the dura mater, a strong layer of connective tissue not attached to the periosteum of the spinal canal; the arachnoid mater, a fibrous layer with spaces containing the cerebrospinal fluid; and the pia mater which provides nutrient and structural support to the neurons.

PERIPHERAL NERVOUS SYSTEM

The peripheral nerves emanate symmetrically from each side of the spinal cord and pass through the body of the vertebrae. The nerves can be single or grouped

together (plexus or ganglia). Two major groups of nerves are the brachial plexus, with nerves branching out to the forelimbs, which originate from the last four cervical and first thoracic vertebrae, and the sciatic or ischiadic nerve, the longest nerve in the body, which branches from the lumbrosacral plexus and runs down the thigh.

ENDOCRINE GLANDS

INTRODUCTION

Defined as the ductless glands, organs of the endocrine system possess specialist cells that secrete hormones - chemical transmitters used for communication and control within the body. These glands include the pituitary, thyroid, parathyroid, thymus, adrenals, pancreas and gonads.

THYROID GLAND

This is a brownish gland situated on either side of the thyroid and cricoid cartilages of the larynx and joined on the dorsal surface by a narrow isthmus so that when removed it has been described as 'butterfly shaped.' It is composed of densely packed sacs containing a clear viscose fluid.

PARATHYROID GLAND

These are two to four pea sized glands lighter in colour to the thyroid glands in which they are embedded.

PITUITARY GLAND (HYPOPHYSIS)

This small yellowish-brown bi-lobed gland is situated in a protective hollow in the cranium on the midline of the ventral brain. The action of the anterior and posterior lobes is controlled by the hypothalamus from which the pituitary is separated by a small portal system of blood vessels by which communication between the two organs is achieved.

THYMUS

The thymus is made up of two yellowish lobulated parts situated either side of the trachea. The thymus is largest in the young animal where the left thymus (anterior)

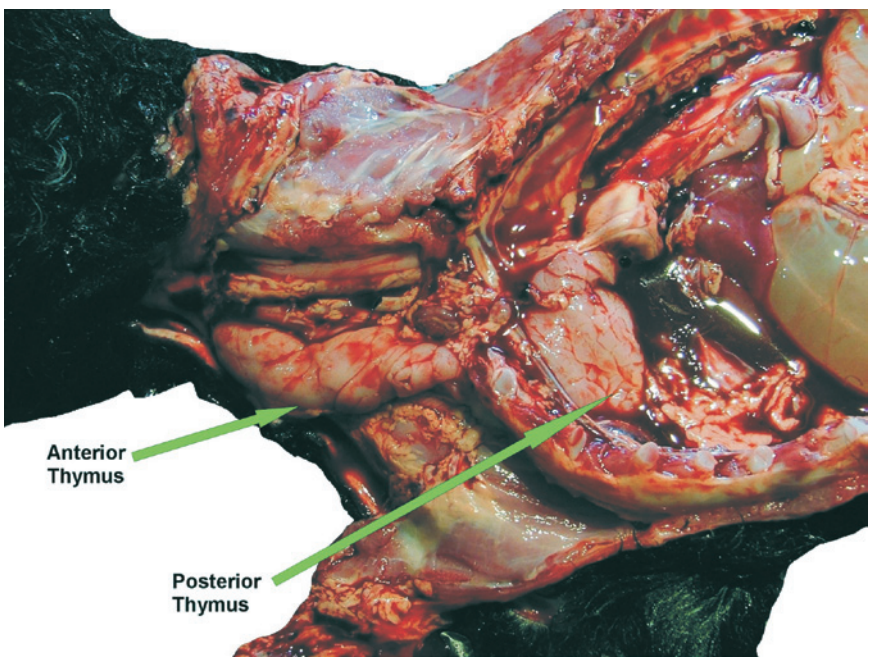
lies in the throat region and the right (posterior) extends down into the thoracic cavity and terminates at the anterior pericardium. As the animal matures the thymus undergoes atrophic change and the separation between the two parts increases as the thymus tissue is gradually replaced by adipose (fat) cells to the extent where the anterior thymus becomes vestigial and the posterior thymus is found only above the cardiac sac. (The thymus is also known as the sweetbreads).

ADRENAL GLANDS

These are paired, dark brown, bean shaped, glands approximately 3cm in length found in fat anterior to the kidneys. Each adrenal gland is composed of two differing tissues with separate functions and can been seen in cross section as a dark brown cortex surrounding a lighter medulla.

PANCREAS

In addition to the exocrine function of the pancreas in the digestive process a second endocrine function, described in the following table, is undertaken by foci of specialist cells (Islets of Langerhans) found throughout the substance of the pancreas.



Position of anterior and posterior thymus in a dissected foetal lamb

Endocrine glands - summary of function

Thymus	Development and maturation of T-lymphocytes. Also sexual development.
Adrenal Cortex	Produces multiple hormones (corticoids) which maintain blood volume, have an anti-inflammatory action, maintenance of blood glucose levels and fat deposition. Secretion controlled by anterior pituitary gland.
Adrenal Medulla	Secretes adrenalin and noradrenalin for stimulation of cardio-respiratory system, increase in blood sugar levels and increased metabolic rate. Also raises blood pressure by vaso-constriction.
Pancreas	Insulin and glucagon work antagonistically to maintain blood sugar levels, insulin prompting conversion and storage of glucose in liver and muscles, glucagon prompting release.
Thyroid	Regulates metabolic rate through production of thyroxin prompted by hormone released by anterior pituitary in response to monitoring by the hypothalamus. Also produces calcitonin to maintain constant blood calcium levels.
Parathyroid	Secretes parathyroid hormone influencing calcium to be absorbed into bone from blood. Works antagonistically with calcitonin.
Pituitary Posterior	Produces oxytocin and antidiuretic hormone (ADH). Oxytocin produced in females only, has a calming and relaxing effect during parturition and also prompts release of milk from mammary gland to the teats.
Pituitary Anterior	Produces many hormones, primarily TSH (Thyroid stimulating hormone) in response to messages from the hypothalamus.

2

DISEASES OF SHEEP

INTRODUCTION

The survival of an animal depends on the symbiotic function of all the bodily systems. Disease can be considered to be an abnormality in the structure or function of these systems, and even an inability of the animal to perform as expected in relation to its peers. The traditional concept of disease is that there is a causal factor that produces recognisable macroscopic and microscopic lesions that can lead to identification of the causal factor. In this section infectious diseases are considered, including those due to viral, bacterial and fungal pathogens, be they of communicable origin (passed from one animal to another) or commensal organisms (organisms that form part of the normal microflora associated with the animal). The role of the Meat Inspector is to determine whether the carcase or part carcase is fit for human consumption, given the macroscopic evidence of lesions presented. Of greatest importance are zoonotic diseases, those that are naturally transmissible between vertebrate animals and man, such as anthrax and brucellosis.

ROUTES OF INFECTION

There are five routes by which infectious organisms gain entry into the host animal, these being:

- **Inhalation** - The causative agents are drawn into the body during the act of inspiration. Tuberculosis lesions usually have primary foci within the lung tissue.
- **Ingestion**- Infection and subsequent spread is achieved by entry of the infectious agent via the digestive tract, initial lesions in this tract are usually followed by transference of the infection to the liver via the hepatic portal blood circulation. Examples include endoparasites such as liver fluke and the larval stages of *Echinococcus granulosus.*
- **Inoculation** - Penetration of the physical barrier to infection, provided by the skin and mucous membranes, allows entry of micro organisms, either through exposure of subcutaneous tissue to environmental contamination or by the injection of these agents that may be present on or in the object penetrating the barrier as occurs in wounds and insect bites. Examples include the tick transmitted infection of red water fever, Louping Ill and Tick Borne fever.
- **Congenital**- Vertical transmission from the dam to the offspring; if this infection occurs before the foetus has developed its own immune system (before third trimester) any infectious organism can be included in the phase where 'recognition of self' occurs so that it may not be countered as it is not recognised as foreign material.

- **Sexual**- Infection can be passed during copulation. Brucellosis and other forms of infectious abortion are known to be transmitted in this manner.

The initial site of microscopic or macroscopic infection is known as the primary focus. The description of condition being a peracute infection indicates rapid onset (few hours), an acute infection generally denotes one of short duration (1-2 days) as opposed to a chronic infection (1 week plus.)

IMMUNITY

Immunity can be described as non-susceptibility to a particular disease, pathogenic effects of micro organisms, or chemical toxins. Immunity is the ability of the body to distinguish foreign material and to neutralize or eliminate it. This is achieved through the body's immune response. Foreign material is recognised by the presence of substances called antigens, which can be toxins, foreign proteins and even parts of the polysaccharide cell walls of bacteria and other tissue. The immunity of a body to infection, either exogenous (where external organisms breach the body defences) or endogenous (where natural flora become pathogenic), is dependant on the inter-reaction of three lines of defence. The first two lines of defence, the skin/mucous membranes and the cellular/humoral factors are innate and non-specific; they are a normal part of the body and react to the presence of any foreign body. The third line of defence, the acquired immune response, is specific to each individual type of invading micro organism and takes longer to mobilise. The first lines of defence form the initial immune reaction and initiate the specific response.

SKIN/ MUCOUS MEMBRANES

These form the initial defence to infection in three ways. Firstly they provide a physical barrier, which produces antimicrobial factors such as the fatty acids present in sweat, the lysosomes in mucous, and surfactant in the lungs. Secondly, the mucous membranes provide a mechanical defence; wave-like movement of hair-like projections (cilia) of the cells in conjunction with mucous remove foreign particles. Thirdly, the presence of normal flora (commensal organisms) on the skin and mucous membranes compete with pathogens for space and nutrients, this competition normally involving the production of short chain fatty acids that act as microbial agents against the invading organisms.

CELLULAR FACTORS

Specialised cells (collectively known as phagocytes) free within the body systems are capable of **phagocytosis**, literally the eating of cells; these include neutrophils which primarily attack extracellular bacteria, eosinophils which attack parasites, and macrophages that attack intracellular bacteria, protozoa and viruses. The process of phagocytosis is four staged, beginning with **chemotaxis**, whereby the phagocyte is attracted to the invader by chemicals produced by the invader itself as well as the body due to the presence of the invader. The second stage is the **adhesion** of the phagocyte to the invader. This is usually accomplished by the phagocyte extending its membrane wall into a 'false limb' (pseudopodium) that reaches out and attaches to the invader. The invader is then **ingested**; the pseudopodium retracts, drawing the invader into the phagocyte, which then extends further pseudopodia around the organism trapping it in a space called a vacuole within the phagocyte. The final stage is the **killing** of the invader, this is achieved either though an oxidative burst by chemicals such as hydrogen peroxide, or through the damaging of the bacterial membrane by lysosomes present in the phagocyte. Enzymes then degrade the dead bacterium.

HUMORAL FACTORS

These assist the process of phagocytosis and are a chain of chemical reactions of serum proteins, the product of one reaction being the catalyst or initiator of the next. The products of these reactions induce chemotaxis, aid adhesion of the phagocyte, lyse (kill) organisms by breaking down the cell wall and increase the vascular permeability allowing the increased supply of phagocytes to the infected area.

ACQUIRED IMMUNE RESPONSE

This specific immune defence is acquired after exposure to infection, and forms the basis of the theory of vaccination. It relies on **antibodies**, molecules of immunoglobulins that comprise three areas; two constant that activate phagocytes and humoral responses and a third that 'keys' onto the specific antigen of the invading organism. Each microbe is therefore recognised by a specific antibody that attaches to it, attracts phagocytes, and triggers the humoral factors. The acquired immune response is dependant on cells known as **lymphocytes**, of which there are two groups, characterised by their function and site of maturity. Lymphocytes are derived from stem cells that travel to areas of the body to mature. **B-cells** are antibody producing cells that are matured in the bone marrow in mammals, **T-cells** are matured and released from the thymus and are involved in cell mediated immunity and assisting the production of antibodies in B-cells.

Each B-cell produces one specific type of antibody and carries large numbers of these on its surface to act as receptors. When confronted with the infectious agent that displays the specific antigen which allows the receptors to lock, the B-cell is activated, multiplies, and stimulates other plasma cells to produce copies of the antibody, producing large numbers of cloned B-cells called **affector cells**. Once the infection has been overcome, some of these B-cells remain viable as '**memory cells'** and remain present should the infectious agent be encountered in the future.

T-cells have various functions, but are primarily aimed at intracellular microbes. **Cytotoxic** T-cells recognise the antigen in virus-infected cells and kill the cell before the virus is released and also produces chemicals that create resistance to infection in neighbouring cells. **Helper/inducer** T-cells assist other T-cells to become cytotoxic, help B-cells produce antibodies, recognise microbial antigens in phagocytes and produce a chemical to activate the macrophage to kill the microbe. **Suppressor** T-cells decrease the activity of other T and B-cells.

Inflammation is a localised response to tissue damage or the presence of antigens. The local capillaries enlarge and increase permeability allowing large numbers of lymphocytes and phagocytes to enter the area, which then proceed to localise and neutralise the cause of the inflammatory response. The classic signs of inflammation are side effects of these changes and include heat, pain, swelling, redness and disturbance of function. Once the causal factor has been overcome, the result to the affected tissue can be regeneration, scarring, or cell death (necrosis).

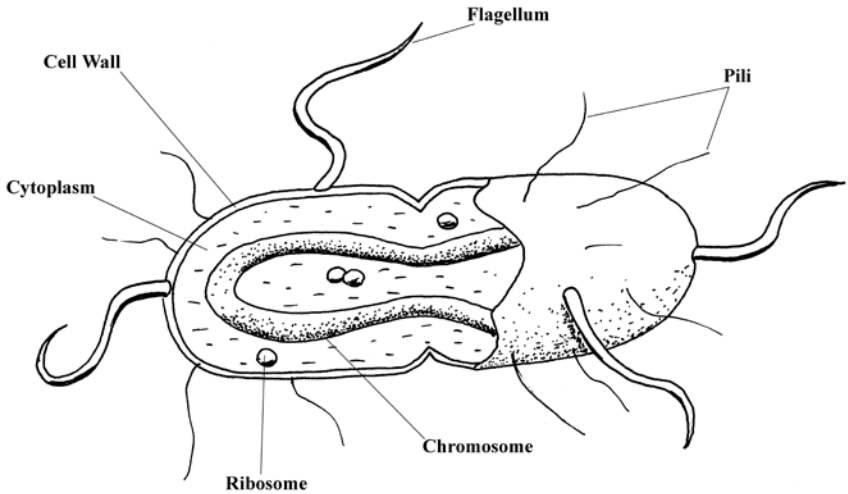
There are various forms of inflammation including those leading to the formation of fibrous tissue (fibrinous inflammation), promoting adhesion of adjacent organs or membranes (adhesive inflammation) and diphtheric inflammation, where fibrinous exudates are formed attached to underlying tissue.

BACTERIA

SIZE AND STRUCTURE

Bacteria are single celled organisms classed as prokaryotic organisms, with varying shapes (morphologies) including spherical, rod shaped, comma shaped, spiral and ranging in size from approximately 0.5-6.0μm.

Prokaryotic organisms (bacteria and green celled algae) are distinguished from other living organisms by containing DNA in a double stranded loop that is unenclosed by a nuclear membrane, small ribosomes (granules containing RNA) and no endoplasmic reticulum (a network of membranes within a cell involved in protein, lipid and glycogen synthesis). They are also characterised by the absence of mitochondria or other membrane enclosed organelles, and the possession of a complex protein cell wall, (with the exception of *Mycoplasma spp*.)



Representative bacterium

The bacterial cell structure includes a rigid cell wall and in many bacteria a mucoid polysaccharide layer complements this. This capsule resists absorption and destruction (phagocytosis) by white blood cells and also aids adherence to tissues.

The pili (fimbrae) are hair-like in appearance, can aid attachment to host cells, are anti-phagocytic and can avoid host antibody response by rapidly altering their antigenic protein, pilin.

Flagella are much longer than pili and provide mobility for the bacterium. The bacteria can have one flagellum at one or both ends (monotrichous), many flagella at one or both ends (lopotrichous), or be 'covered with hair' (peritrichous). Mobility is achieved by clockwise or counter clockwise rotation of the flagella.

Certain bacteria have the ability to sporulate (e.g. *Clostridium* and *Bacillus spp*.), where an extremely tough protective coat surrounds concentrated bacterial DNA. The cell becomes metabolically inert and can survive dehydration, heat and most chemical agents for years, until favourable conditions for growth reactivate the bacterium.

ENERGY, NUTRITION AND GROWTH

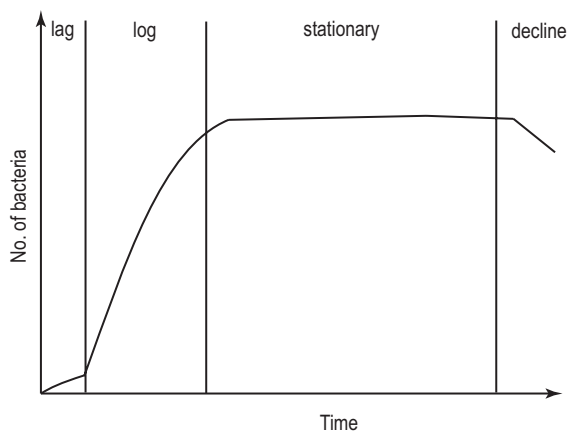
Bacteria use three sources for their energy requirements, light (phototrophy), chemical reactions (chemotrophy) or a host cell (paratrophy).

Nutritionally bacteria can be divided into two types; autotrophs, which can synthesise nutrients from inorganic raw materials; and heterotrophs, which depend on preformed organic molecules from the environment as the source of nutrients. As with some parasitic and pathogenic bacteria, that can only survive intracellularly, heterotrophs possess DNA and RNA but only have a certain amount of metabolic activity.

Bacterial growth is by binary fission, where the internal contents of the bacterium halve, split and then reform into two matching bacterium, the doubling time varying from 20 minutes (*E.coli*) to 24 hours (tubercle bacilli). The process of binary fission means that one bacterium becomes two, two become four, four become eight, and eight become sixteen, and so on. Given balanced growth, where all required nutrients are available, the growth curve of bacteria follows distinct phases. After an initial lag phase, high-virulence exponential growth occurs during the log phase, followed by a stationary phase where the number of bacteria produced is equal to the number dying, which is in turn followed by the decline phase where the number dying is greater than the number produced as the available nutrients are used up.

Each bacterial species has varying requirements for optimum growth, including available nutrients, temperature, available moisture in the form of water activity (Aw), and the pH or hydrogen ion concentration of their growth medium.

Taking these factors in order, bacterial growth requires water-soluble nutrients, and high protein foods are favoured such as eggs, fish and meat. Food was historically salted to preserve it, which reduces moisture as well as preventing the osmotic diffusion of nutrients into bacterial cells.



Bacterial growth curve

In terms of temperature, the lower the temperature, the slower bacteria grow. Most disease causing bacteria (pathogens) cannot grow below 4ºC and none can grow below 0ºC, spoilage bacteria can grow at temperatures down to -5ºC. As these are the only two groups of bacteria present on meat, at temperatures below -5ºC all bacterial growth ceases.

Each bacterial species has an optimum temperature for growth, as well as a temperature range in which they grow, it is generally accepted that there are four groups:

	Optimum	*Range*	
Psychrophiles	Below 20ºC	-5 to 25ºC	Includes spoilage bacteria prevalent in cold stores and refrigerators.
Psychrotrophs	Above 20ºC	-5 to 40ºC	
Mesophiles	20 to 45ºC	10 to 56ºC	Includes most common pathogens such as *E.coli*, *Staphylococcus aureus* and *Campylobacter jejuni*.
Thermophiles	Above 45ºC	35 to 80ºC	Important in canning.

The moisture content, or available water level (Aw), is vital to the growth of bacteria. Pure water has an Aw value of 1.0. Bacteria have a preferred optimum available water value of around 0.99, however, *Staphylococcus aureus* will grow at 0.89 and some bacteria will grow as low as 0.75.

The hydrogen ion concentration, or pH, of the medium also affects bacterial growth. Water has a neutral ph of 7, pH values from 1 to 7 are decreasingly acidic (decreasing concentration of +H ions) and those from 7 to 14 are increasingly alkaline, (increasing concentration of –OH ions). Bacteria prefer mediums with a pH of around 7; most bacteria will not grow in food with a pH below 4.5.

TOXINS

Bacteria possess the ability to form toxins, chemicals that alter the characteristic of cells and tissue, both for the purpose of defence against attack, and to increase the invasive potential of the bacteria. There are two types of toxin, endotoxin and exotoxin.

Endotoxins are formed from polyliposaccharides that form part of the cell wall and released into surrounding tissue only when the bacterium breaks down. Exotoxins are formed from proteins and are excreted from the cell into surrounding tissues. Bacterial exotoxins, such as that produced by *Clostridium botulinum* (botulism) are the most potent poisons known to man.

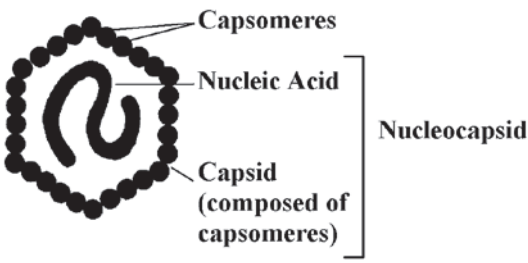
BACTERIAL IDENTIFICATION

In general, owing to secondary infection and the fact that various bacteria can manifest themselves in similar lesions in the affected organism, identification of the infectious agent is conducted in laboratories by culturing samples.

Bacteria are classified in various ways including their shape/size, motility, resistance to antibiotics, oxygen requirements, culture media etc. One of the identifying tests conducted is Gram's staining in which the bacteria are initially stained with crystal

violet, treated with iodine solution and then decolourised with alcohol. When observed under a microscope the bacterial cells either retain a deep purple colouration and are said to be Gram positive, or their complex cell wall chemical structure resists iodine and the walls are decolourised by the alcohol and are said to be Gram negative.

VIRUSES

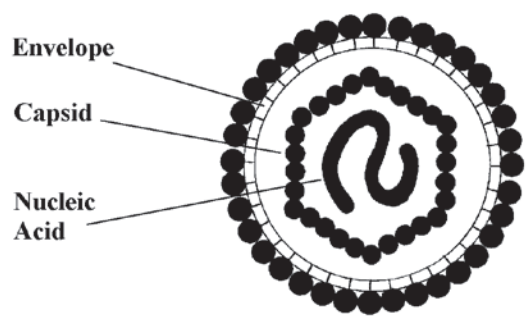


Viruses are obligate parasites, they do not contain the biochemical mechanisms for their own replication and are unable to replicate outside of a living cell.

The basic viral particle (nucleocapsid) consists of linear genetic material (DNA or RNA) surrounded by a protein coat (capsid) composed of capsomeres. Some viruses are also enclosed in a membranous envelope of lipoprotein; in the case of the influenza virus this coat is formed from the host's cellular tissue and inhibits the body's auto immune response.

A virion is the complete viral particle, found extracellularly and capable of surviving in a metabolically inert form and possessing the ability to infect living cells. Virion range in size from 0.003 to 0.05 μm.

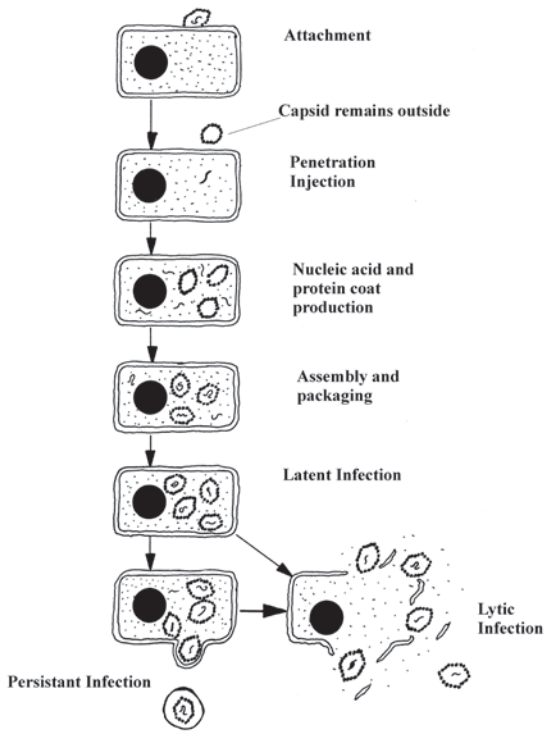
Viruses replicate by using the biochemical mechanisms of a host cell to synthesize and assemble their separate components. When attached to a host cell, only the viral nucleic acid and in some cases a few enzymes, are injected into the cell. The nucleic acid is then replicated within the cell, followed by the synthesis of the capsid.



Enveloped Virus

VIRUS TYPES

Adenovirus	Unenveloped, linear, double stranded nucleic acid.
Birnavirus	Unenveloped. (Bi=two) double stranded RNA present in two segments.
Coronavirus	Enveloped. Corona refers to the crown-like appearance of spikes on the envelope. A cytocidal, organ specific virus leading to progressive degeneration.
Herpesvirus	Enveloped. Large, double stranded, linear DNA, icosahedral capsid.
Orthomyxovirus	Enveloped. Influenza virus. Type A affects humans and animals, Types B&C affect only humans. RNA genome divided into eight RNA molecules.
Paramyxovirus	Enveloped. Infective mechanism relies on fusion of the envelope with the host cell membrane. RNA protected by helical nucleocapsid.
Parvovirus	Unenveloped. Smallest and simplest viral particle. Icosahedral capsule containing one molecule of single stranded DNA.
Picornavirus	Unenveloped. Pico-RNA literally means small RNA that is single stranded.
Pox virus	Large ovoid or brick shaped viruses, replicates in the cytoplasm of cells. First virus to be seen microscopically.



Replication cycle of a virus

After infection by a virus, there are four possible effects on the host cell.

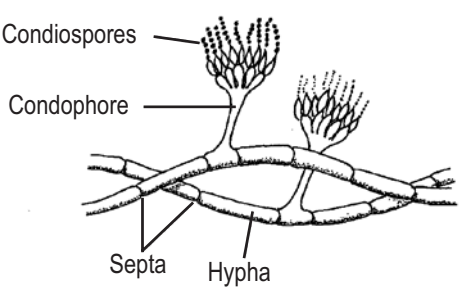
- Transformation of normal cells to tumour cells. Followed by division and the production of a tumour.
- Lytic infection. After viral multiplication the cell dies releasing the virus.
- Persistent Infection. After viral multiplication there is a slow release of the virus without cell death.
- Latent Infection. After viral multiplication the virus is present in the cell but not causing harm. Later emerges in lytic infection.

FUNGI

Fungi (yeasts, moulds etc) are eukaryotic organisms, characterised by the absence of chlorophyll, the presence of a rigid cell wall in some stages of their life cycle, and their reproduction by means of spores. Their cell size ranges between 10-30 μm, containing organelles such as mitochondria, Golgi apparatus, lysosomes and endoplasmic reticulum, and linear DNA contained in a nuclear membrane. The cells are non-motile and heterotrophic. Their reproduction is complex (asexual and sexual).

There are two major morphological forms of fungi, small round yeasts and long filaments called hyphae. Yeasts are round, unicellular, and multiply by budding or by fission. Filamentous fungi form hyphae; long tubes containing protoplasm which may have cross walls, called septa, which possess a central pore allowing the through flow of protoplasm; or the hyphae may simply be multinucleate. A collection of hyphae is known as a mycelium, which may be vegetative, growing on a nutrient surface, or can extend upward as an aerial mycelium producing 'spores' (conidia) which spread very easily.

Dimorphic fungi exist in both forms. Many pathogenic fungi are dimorphic, usually with the yeast variety forming inside tissues and the filamentous (mould) form in the environment or surface.



Structure of a filamentous mould

In terms of meat quality, fungi can survive on medium with a lower Aw than bacteria, and are less affected by pH. Different fungal groups produce varying physical appearances on meat, white 'whiskers' are attributable to *Mucor spp*., green patches to *Penicillium spp*., white spots to *Sporotrichum spp*., and black spots to *Cladosporium spp*. They do, however, take

longer to grow than bacteria, and meat spoilage is more likely to be attributable to the latter.

Various fungi produce chemicals during their growth such as antibiotics and toxins, known as mycotoxins. These secondary products of metabolism are not necessary for the growth of the fungi, but are felt to form a system to prevent competition for available space and nutrient from bacteria and other fungal species by inhibiting the growth of these other microorganisms. The antibiotics disrupt the growth of bacteria, the use of *Penicillium spp*. as an antibiotic is well known. Mycotoxins, when produced by fungi on foodstuffs such as grain kept in a humid environment can cause toxaemia in animals. The effects of which vary according to the level of toxin in the feedstuff, the duration of intake, and the age, sex and nutritional status of the host. Fungal toxins generally affect the liver.

PRION PROTEINS

Prion proteins are pathogenic variants of proteins that are naturally produced mainly by nerve cells. These are an infectious agent, despite not containing nucleic acid and have the same chemical composition as the normal proteins produced but have a different molecular shape and the ability to force the normal protein to alter its shape and become pathogenic. Prion proteins cause a group of diseases known as Transmissible Spongiform Encephalopathies (TSE's) that include scrapie in sheep, new variant CJD and 'Kuru' in humans and Bovine Spongiform Encephalopathy (BSE, Mad Cow Disease) in cattle. Much research has been undertaken as to the nature and pathogenesis of prion proteins, and legal requirements for the slaughter and dressing of sheep alter in line with the emergence of new scientific advice.

When material that may contain prions is ingested (pasture contaminated by infected placental tissue from infected animals, mammalian foodstuffs etc), the prions congregate and are isolated at the Peyer's patches within the small intestine. They are then transported by lymphocytes to lymph nodes and other lymphoid material such as the tonsils, spleen and thymus. Here, replication of the prion takes place. Normal proteins are produced within cells and move via the Golgi bodies to the cell membrane. Initial encounters with rogue prion proteins forces the normal proteins to change shape and characteristic to become a prion protein, this action is the initial transmission of infection. Once altered the new rogue prion protein then infects its neighbours and so on. If the prion proteins accumulate within a cell there is also the possibility that the infection can spread during the normal protein production within the cell.

It is felt that the prion proteins may then move along nerve axons to the spinal cord and then to the brain, the exact mechanism for this movement is unclear, it may be due to the movement of vesicles within and between nerve cells or may be

just a matter of spread of infection between contacting surfaces. The replication of the prions in the brain tissue leads to the formation of non-inflammatory vacuoles and brain dysfunction, the clinical signs of which are atypical of this dysfunction; loss of motor control, paralysis and loss of control of the autonomic nervous system (dysautonomia) and finally death.

At present the accumulation of prion proteins in infected animals in certain lymphoid and nervous tissue has led to that tissue being removed from the carcase under special precautions and treated in such a way as to prevent it from entering the human and animal food chain. In lambs with at least one permanent incisor erupted this material, termed Specified Risk Material currently includes the head (excluding the tongue), spleen, ileum, spinal cord and brain. In young lambs (no permanent incisors) this material includes the spleen and ileum.

DISEASES OF SHEEP

ACTINOBACILLOSIS

Synonyms	Leather lips, 'Cruels'
Type	Bacterial
Aetiology	*Actinobacillus lignieresi* Gram –ve, pleomorphic rod
Pathogenesis	A commensal organism of the oropharynx. Infection derived by inoculation of pathogen into soft tissues through buccal cavity damage by rough herbage or tooth eruption, for example.
Clinical Signs	Inflammation of the soft cutaneous tissues of the head and neck. In cases of nasal actinobacillosis there is discharge, snoring, and breathing sounds associated with nasal obstruction of the respiratory system.
Gross Lesions	Ulceration and nodules in subcutaneous tissue. Subcutaneous abscesses. Local lymph nodes can contain granular yellowish-green pus.
Judgement	Reject affected parts

ACTINOMYCOSIS

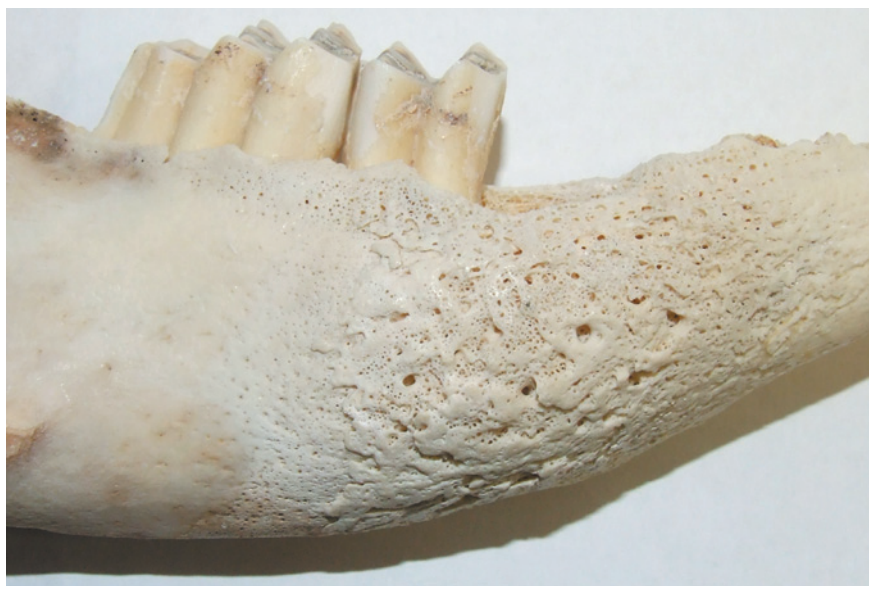
Synonyms	Lumpy Jaw, Ray Fungus
Type	Bacterial
Aetiology	*Actinomyces bovis*. Gram +ve facultative anaerobe.
Pathogenesis	Considered to be a commensal organism within the buccal cavity, the route of infection is due to inoculation and entry through wounds, especially through the gum. Produces an osteomyelitis usually within the mandible. The abscessation that follows can fistulate to the surface and discharge. The term 'ray fungus' derives from the formation of a mycelium of branching filaments.

Clinical Signs Swollen painful bones, especially the mandible and maxilla. Evidence of fistulae (scarring, depression within the skin of the cheek), flow of sticky granulomatous pus.

Gross Lesions Honeycombed appearance of the bone due to rarefying osteomyelitis, the cavities produced normally containing thick green granulomatous pus. Some cases may have local lymph node involvement.

Judgement The affected part should be rejected.

Actinomycosis of the lower mandible



Actinomycosis – a rarefying osteomyelitis

ANTHRAX

Synonyms	Wool sorters disease, Splenic fever, Charbon, Malignant pustule
Type	Bacterial
Aetiology	*Bacillus anthracis*. Large Gram +ve rod. Forms stable spores in the presence of air and remains viable in the soil for decades
Pathogenesis	Spores germinate in mucosa of throat or intestine. Rapid invasion of the bloodstream, lymphatics and spleen produces acute septicaemia. Toxin produced by the bacterium damages and kills phagocytes, increases blood vessel permeability and damages the clotting mechanism.
Clinical Signs	Death. Exudation of tarry dark blood from the natural orifices of the body. Absence of rigor mortis. Rapid decomposition of the body. Oedema. Cutaneous form – Blackened raised pustule surrounded by oedema. Most common form in humans due to presence of the bacterium in open wounds.
Gross Lesions	A peracute septicaemia. Congestion throughout carcase, dark red lymph nodes and severe splenomegaly. Petechial haemorrhaging of mucous membranes, fatty change of liver and kidneys. Blood is thick, tar-like and does not clot.
Judgement	Suspect cases must not be opened for post mortem examination. *Bacillus anthracis* will sporulate in the presence of air making decontamination problematic. **Notifiable**. Unfit for human consumption. Zoonotic disease. Human pulmonary form known as woolsorters disease. Cutaneous form malignant pustules.

BACILLARY HAEMOGLOBULINURIA

Synonyms	Red Water Disease
Type	Bacterial
Aetiology	*Clostridium haemolyticum.* Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	Generally follows liver damage caused by migration of immature fluke or any event that damages the liver changing the micro-environment to an anaerobic state. Toxins formed by bacteria are necrotizing and haemolytic.
Clinical Signs	High fever and cessation of rumination, rapid breathing. Dark red urine, death.
Gross Lesions	Rapid rigor mortis, red urine in bladder. Petechial haemorrhages in kidneys and occasionally the tracheal mucosa. Haemorrhagic enteritis (small intestine). Raised pale hepatic infarcts surrounded by an area of congestion. Blood stained hydrothorax and ascites.
Judgement	Systemic toxaemia. Reject carcase and associated offal.

BLACK DISEASE

Synonyms	Infectious necrotic hepatitis
Type	Bacterial
Aetiology	*Clostridium novyi* type B. Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	Generally follows liver damage caused by migration of immature fluke or any event that damages the liver changing the micro-environment to an anaerobic state. Toxins formed by bacteria are necrotizing and haemolytic.
Clinical Signs	Rapid onset, fever, dullness, unsteady gait, an inability to move, followed by collapse and quiet death within a few hours of onset.
Gross Lesions	Hepatitis associated with darkening, necrotic patches. Skin appears darker due to vascular engorgement, occasionally straw coloured ascites, hydrothorax or hydropericardium.
Judgement	Systemic toxaemia. Reject carcase and associated offal.

BLACKLEG

Synonyms	Black quarter
Type	Bacterial
Aetiology	*Clostridium chauvoei*. Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	Spores remain viable in the soil for years. Infection acquired through ingestion, movement through compromised digestive membrane to the body. The spores then lodge in various organs and tissues, especially muscles. Here they lie dormant until stimulated to multiply, by a reduction in oxygen usually by injury to the animal. The injury reduces blood flow to the area, thereby reducing the supply of oxygen to the tissues. In the absence of oxygen, the spores germinate and multiply. As they grow, the bacteria produce toxins which destroy surrounding tissues. The toxins are absorbed into the animal's bloodstream which makes the animal acutely sick and causes rapid death. A fatal disease of young lambs, especially those rapidly gaining weight between 6 months and 2 years of age. Produces myonecrotising toxins that kill individual muscle fibres.
Clinical Signs	High fever, toxaemia, lameness, loss of appetite. Swellings develop in the hip, shoulder, chest and back. Starting small, hot and painful becoming swollen and gaseous. Subcutaneous emphysematous crepitation can be felt. Mortality high, usually within 12 to 48 hours due to toxaemia.
Gross Lesions	Carcases undergo rapid putrefaction. Skin over swellings can develop dry gangrene in the centre. On incision the swellings usually contain gas and discoloured serum, affected musculature is dry, sweet smelling, contains gas bubbles and becomes black or darker than normal, and swollen.
Judgement	Reject carcase and associated offal

BLOAT

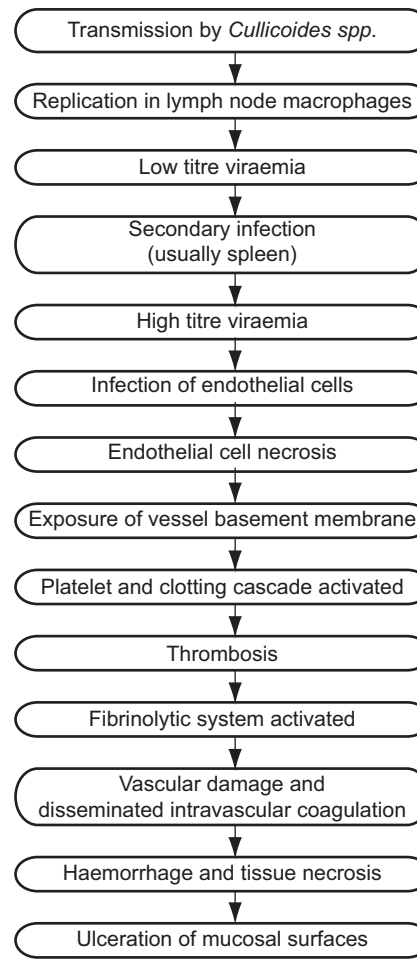
Type	Miscellaneous
Aetiology	Physical blockage, bacterial infection (*Streptococci bovis* a commensal organism of the digestive tract), feed type.
Pathogenesis	Two forms - frothy bloat and gaseous bloat. Frothy bloat is due to surfactants in feed, such as legumes, forcing fermentation gas to form in frothy bubbles that cannot be eructated. Gaseous bloat can follow a rapid change in feeding leading to improper fermentation, particularly of grain. It can also occur if there is physical blockage of the oesophageal orifice from the rumen.
Clinical Signs	Dyspnoea (laboured breathing) due to pressure on the diaphragm when the size of the rumen is increased. Loss of rumination, the animal literally stops 'chewing the cud'.
Gross Lesions	Grossly extended rumen. Can be a hazard of contamination, increased gas pressure can increase chances of bursting the stomachs releasing their contents.
Judgement	Fit. Animals found with bloat in the lairage should be slaughtered immediately on welfare grounds.

Bloat – distended abdomen due to gas build up in the rumen

BLUE TONGUE

Synonyms	Ovine catarrhal fever, 'sore muzzle' disease
Type	Viral
Aetiology	Orbivirus

Pathogenesis Transmission of the virus is via biting midges such as *Culicoides spp.* Now found in the UK as the warmer climate predisposes to transfer from the continent as does the import of cattle which can act as reservoirs for the virus. After infection, viral replication occurs in regional lymph nodes and the spleen. Viraemia follows with the virus destroying the endothelium of the blood vessels, increasing permeability and leading to development of thrombosis.



Clinical Signs Fever. Laboured breathing and panting, hyperaemia and oedema of muzzle, lips and tongue. Hair loss, and lameness due to laminitis and inflammation of the coronary band may also occur. Nasal discharge that becomes muco-purulent or bloody, and excess tear production (lacrimation). Oedema of the face and jaw common.
Death is generally due to secondary bacterial or inspiration pneumonia.

Gross Lesions Cyanosis of the tongue, glossitis and vesicles/ ulceration/ erosion of the buccal mucosa especially the dental pad.
If suffering an acute viraemia, haemorrhaging in the endocardium and enlarged haemorrhagic lymph nodes may be found, also petechial haemorrhaging of the base of the aorta. Other signs that may be seen include; enlarged spleen, generalised lymphadenitis, pale necrotic areas within musculature (cardiac and skeletal), and haemothorax.

Judgement **Notifiable** in the UK. Has been confused with foot and mouth lesions. Carcase and associated offal unfit for human consumption.

BRAXY

Synonyms	Bradshot
Type	Bacterial
Aetiology	*Clostridium septicum.* Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	The ingestion of frosted grass produces favourable conditions for the bacteria to invade tissues of the abomasum and multiply.
Clinical Signs	Fever and depression, accumulation of abdominal gas, cessation of rumination followed by coma and death.
Gross Lesions	Inflammation of the wall of the abomasum, purple congestion and occasional ulceration. Toxaemia
Judgement	Reject carcase and associated offal.

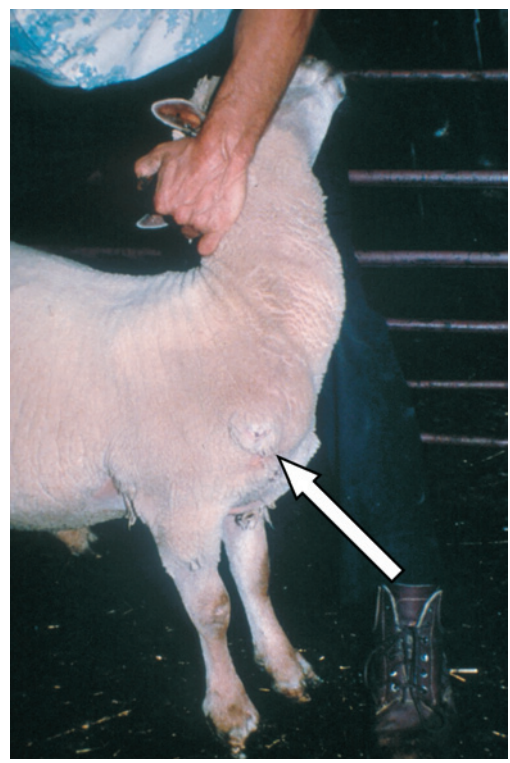
Frosted grass, ideal conditions for cases of braxy

BRUCELLOSIS

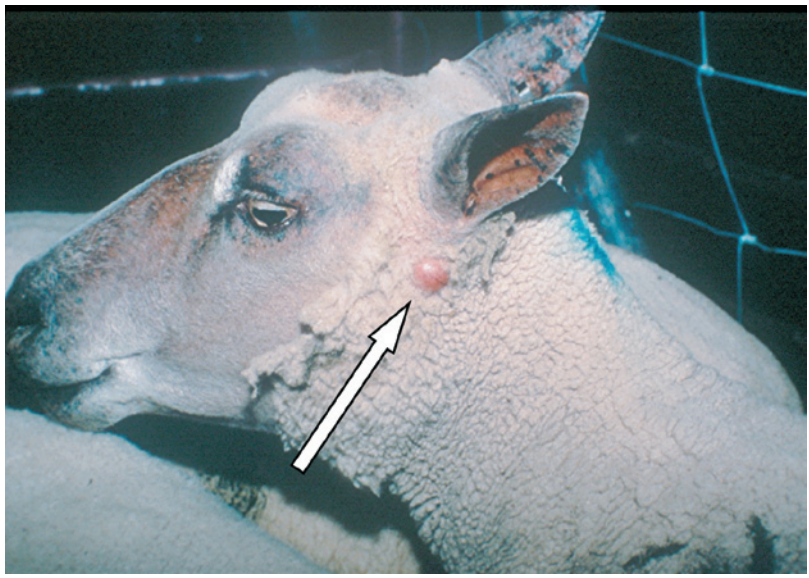
Synonyms	Ovine brucellosis, OB
Type	Bacterial
Aetiology	*Brucella melitensis*. Gram –ve short rod.
Pathogenesis	Transmission via inhalation or inoculation through skin wounds.
Clinical Signs	Abortion. Fever. Weight loss, mastitis, lameness, orchitis
Gross Lesions	No specific lesions
Judgement	Carcase and offal unfit for human consumption**. Notifiable in UK**
Other	**Zoonotic**. Causes Malta fever in humans. Transmission via infected milk, post-birth discharge, and handling of infected uteri.

CASEOUS LYMPHADENITIS

Synonyms	CLA. CL. Cheesy Gland. Pseudotuberculosis
Type	Bacterial
Aetiology	*Corynebacterium pseudotuberculosis* Gram +ve rod. Pyogenic due to outer lipid layer. Produces exotoxin
Pathogenesis	Inoculation spread. Shearing wounds, castration and docking implicated in spread. Can survive for many months in purulent material. After inoculation of bacteria into superficial wounds, exotoxin production induces localised inflammation and necrosis in addition to increased vascular permeability. The bacterium is carried to regional lymph nodes by means of phagocytes in which multiplication takes place followed by death of the phagocyte, protection to phagocytosis being afforded by the outer lipid layer of the bacterium. *C.pseudotuberculosis* behaves as an intracellular parasite, multiplying in host phagocytes until cell death disseminates more bacteria.
Clinical Signs	Enlargement of the peripheral lymph nodes (5-15cm diameter) including submaxillary, parotid, prescapular, prefemoral and popliteal. These infected nodes may fistulate with thick cheesy non-odorous pus. If it becomes an internal infection the clinical signs are dependent on system affected.
Gross Lesions	Grossly enlarged lymph nodes containing concentric laminated pus and necrotic material, resembling an onion; can become calcified. Kidney abscesses. Common sites for the formation of visceral lesions are the mediastinal and bronchial lymph nodes which can lead to lung abscesses.
Judgement	Reject affected parts. If generalised or associated with emaciation, reject carcase and associated offal. A zoonotic condition



***Caseous lymphadenitis* – Enlargement of the Prescapular lymph node evident at ante-mortem inspection.**



***Caseous lymphadenitis* – enlargement of parotid lymph node.**

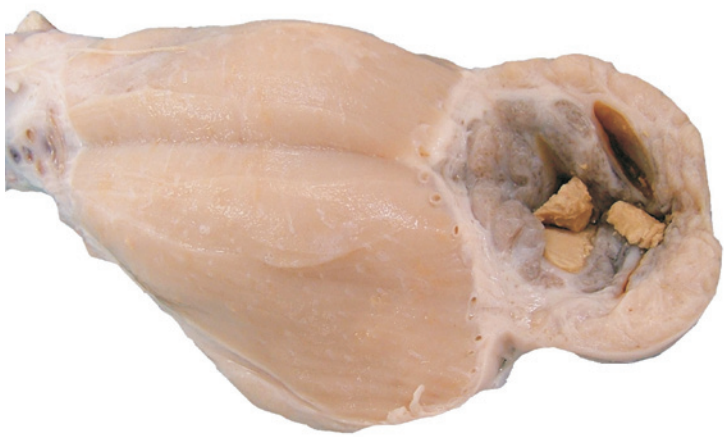
CONTAGIOUS AGALACTIAE

Synonyms	Mycoplasmosis
Type	Bacterial
Aetiology	*Mycoplasma agalactiae.* Gram –ve.
Pathogenesis	Often a seasonal infection, more prevalent in late spring / early summer characterised by fever followed by mastitis, polyarthritis and keratoconjunctivitis. Spread via mechanical vectors such as flies, contaminated bedding or contaminated feed etc. *M.agalactiae* can survive in the supramammary lymph nodes, re-emerging at the next lactation.
Clinical Signs	Abortion is common. Fever during a septicaemic phase, mastitis leading to atrophy of the udder and a reduction in milk quantity (agalactia). Milk produced contains clots and has a greenish tinge. Lameness can follow, the carpal and tarsal joints of the feet being especially prone to swelling and discharge of exudates. Unilateral or bilateral eye-infection can be an associated lesion, leading to impairment of vision and blindness if corneal damage occurs. Arthritis can develop in association with conjunctivitis; the latter can lead to temporary blindness. Males can suffer from orchitis in addition to arthritis.
Gross Lesions	Acute mastitis followed by fibrosis and atrophy, inflammation of the eyeball (initially mucopurulent but can lead to corneal ulceration), fibrinous polyarthritis. In acute septicaemic stages congestion of liver, musculature and spleen is common, as is a generalised peritonitis. Lymphadenitis common.
Judgement	**Notifiable** in UK. Unfit for human consumption.

CONTAGIOUS EPIDIDYMITIS

Synonyms	Ram Epididymitis,
Type	Bacterial
Aetiology	*Brucella ovis*.
Pathogenesis	A venereal disease of rams. Transmission ram to ram directly or via infected ewes
Clinical Signs	Orchitis, which can be unilateral. Affected testes can be grossly enlarged
Gross Lesions	Varying degrees of inflammation of the epididymis including abscessation.
Judgement	**Notifiable** in UK. Unfit for human consumption.

Orchitis



Abscess formation in an epididymis. (Incised sample fixed in formal saline)

ENTEROTOXAEMIA

Synonyms	Pulpy kidney, Overeating disease
Type	Bacterial
Aetiology	*Clostridium perfringens* Type D. Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	Occurs most commonly in fattening stock between 6 months to 1 year old when changing to lush pasture. Rapid change of diet is not immediately compensated for by ruminal digestive bacteria, this leads to changes in the biochemistry of the intestines allowing proliferation of *Cl. perfringens*. The bacteria produce a non-lethal toxin which is acted upon by the enzyme trypsin to form a lethal toxin that is absorbed through the intestinal wall
Clinical Signs	Sudden death with no previous sign of disease is common. Other signs include: green pasty diarrhoea, muscular contraction, teeth grinding, dullness and depression. Collapse and rapid kicking, abdominal pain, and coma precede death.
Gross Lesions	Straw coloured hydropericardium, occasionally blood stained. Petechial haemorrhaging of cardiac muscle, congestion of abomasum, liver and intestinal mucosa, soft pulpy kidneys and rapid decomposition of the carcase.
Judgement	Carcase and offal unfit for human consumption.

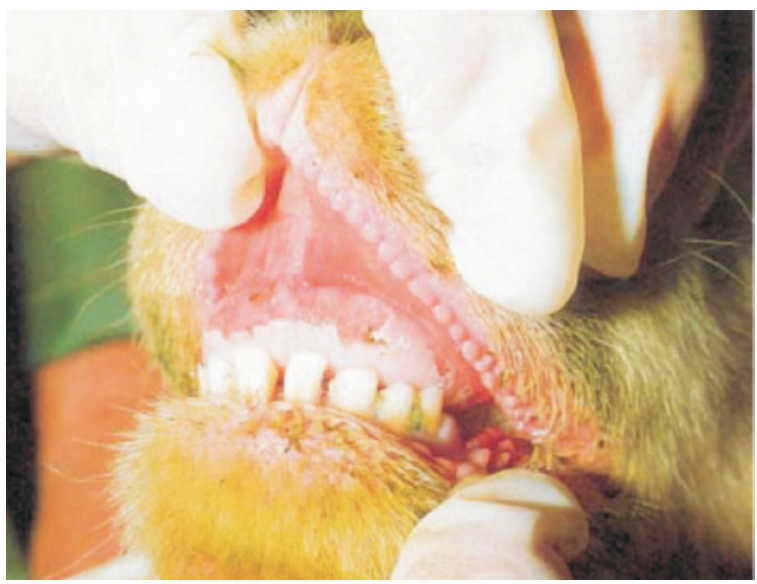
ENZOOTIC ABORTION

Synonyms	Chlamydiosis
Type	Bacterial
Aetiology	*Chlamydia psittaci*
Pathogenesis	*C.psittaci* is an intercellular parasite that exists in two forms; an infective stage and replication stage. The infective stage enters cells; being drawn into the cell after adhering to the membrane. A vacuole is formed within which the infective stage alters to the replication stage, reproducing by binary fission. Before the cell ruptures the replication stage reverts to the infective stage and the process repeats.
Clinical Signs	Vulval discharge followed by abortion of stillborn lamb up to two weeks before due date
Gross Lesions	Metritis with yellowish-grey thickened areas surrounding dark red cotyledons. Can also be associated with pneumonia in young lambs.
Judgement	**Zoonotic**. Carcase and associated offal unfit for human consumption.

FOOT AND MOUTH DISEASE

Synonyms	FMD, Apthous fever, Contagious aptha
Type	Viral

Aetiology	Apthovirus. Seven strains; A,O,C, South African Territories (SAT) 1,2,3, ASIA1
Pathogenesis	Primarily aerosol spread. Although not resistant to sunlight or high temperatures, evidence suggests that the virus may be spread by the wind over distances of 30 miles. Initial infection occurs in the mucous membranes of the throat, followed by replication and subsequent entry into the blood circulation and secondary infection of susceptible cells within the body. Type C apthovirus has a predilection for cardiac muscle.
Clinical Signs	Fever, followed by vesicle formation on the nostrils and in the buccal cavity, especially the dental pad in sheep, and on the coronary band and interdigital cleft of the hoof. Excessive drooling and lip smacking can be observed as can foot shaking followed by lameness. Young lambs can die due to cardiac failure.
Gross Lesions	Initial rupture of vesicles leaves red raw denuded areas of epithelium with sharp edges, over the following four days the deposition of fibrin and regrowth of epithelium occurs which can be left as scar tissue. In young lambs (especially with Type C infection) there may be pale areas of multifocal necrosis of the myocardium.
Judgement	**Notifiable** Disease in UK. Carcase and associated offal unfit for human consumption.
Other Information	The outbreak of FMD in the UK in 2001 preceded an unprecedented level of feet and buccal cavity inspection in ovines at the point of slaughter. The differential diagnosis of FMD following these examinations include Orf, Idiopathic Oral Ulceration, Bluetongue, Foot Rot, Post-dipping lameness and buccal cavity injury due to rough grazing.



Ruptured vesicles on dental pad (Crown Copyright)

Vesicles on coronary band (Crown Copyright)

FOOT ROT

Synonyms	Malignant foot rot. Foot lure, foot foul, foul of the foot, Contagious ovine interdigital dermatitis, COID,
Type	Bacterial
Aetiology	A synergistic infection of *Dichelobacter nodosus* which only survives in infected feet and *Fusobacterium necrophorum* an environmental anaerobic bacteria.
Pathogenesis	*F.necrophorum* alone produces dermatitis of the interdigital cleft but is uninvasive. *D.nodosus* has invasive potential and leads to an inflammation of the skin-horn junction progressing to an underunning of the horn to the laminae.
Clinical Signs	Lameness, can affect all four feet. Laminitis and associated foul odour.
Gross Lesions	Infection can lead to separation of the hoof at the skin-horn junction.
Judgement	Reject affected parts.

GREEN WOOL DISEASE

Synonyms	Fleece rot
Type	Bacterial
Aetiology	*Pseudomonas aeruginosa*. Gram –ve Anaerobe
Pathogenesis	Prolonged moisture of the fleece and skin can lead to exudates and crust formation which mats together the wool fibres. *P.aeruginosa* can then colonise this anaerobic micro-environment.

Virulent foot rot, leading to separation of the hoof from the underlying laminae.

Clinical Signs	Matted wet fleece with a greenish tinge due to the colour producing characteristics of *P.aeruginosa*
Gross Lesions	Possible enlargement of local lymph nodes draining skin.
Judgement	Fit for consumption if there is no evidence of secondary bacterial infection.

HAEMORRHAGIC ENTERITIS

Synonyms	Bloody scours, Romney Marsh Disease, Struck
Type	Bacterial
Aetiology	*Clostridium perfringens* Type C. Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	An acute enterotoxaemia, affected animals may die without any gross lesions being present
Clinical Signs	Diarrhoea, straining, abdominal pain, death.
Gross Lesions	Haemorrhagic enteritis, deep blue/purple discolouration of the intestines. Peritonitis and myositis.
Judgement	Carcase and associated offal unfit for human consumption

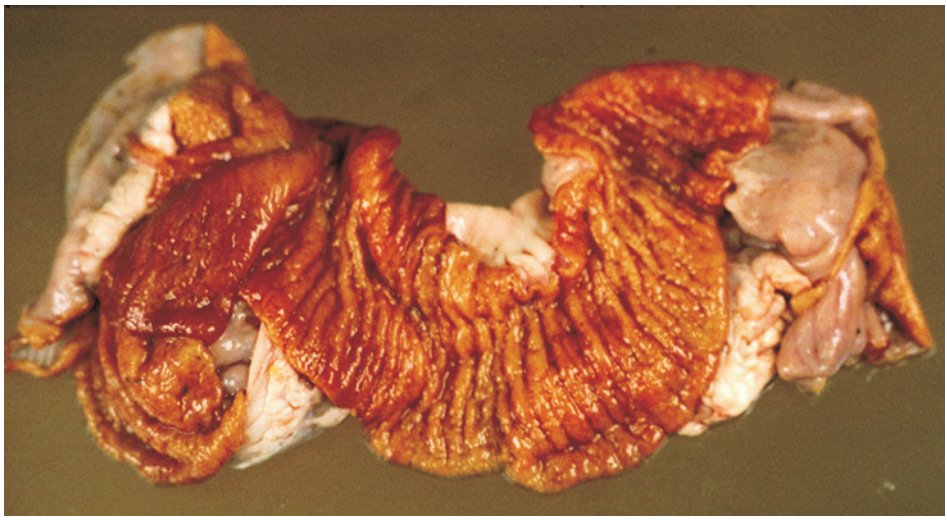
JAAGSIEKTE

Synonyms	Ovine pulmonary adenomatosis, Ovine pulmonary carcinoma, Driving sickness.

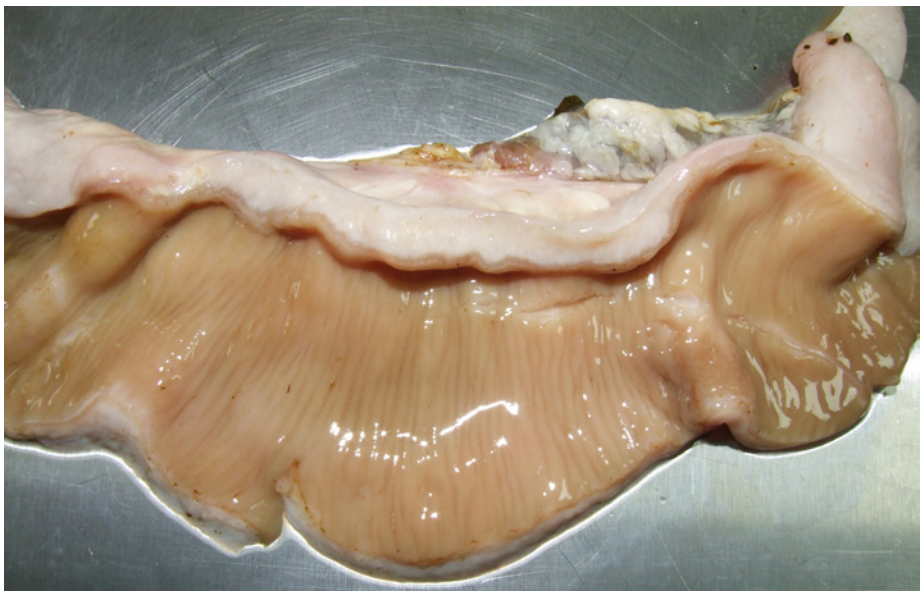
Type	Viral
Aetiology	Retrovirus
Pathogenesis	Transmission via respiratory route by contaminated aerosol or droplet. Incubation period from 9 months to three years. Most cases occur in animals over 2 years of age, death can occur due to secondary bacterial pneumonia after 2-6 months of infection.
Clinical Signs	Progressive emaciation, severe dyspnoea. A 'wheelbarrow' test, whereby the head is lowered can result in flow of frothy fluid accumulated in the respiratory tract from the nostrils, a characteristic sign of this infection.
Gross Lesions	Lung enlargement and in chronic cases these do not collapse on opening of the thoracic cavity. Neoplasia ranging from nodular to solid masses is found in lung tissue. These are sharply demarcated, firm and range from a grey to pinkish-grey colour. Generally on incision the tumours are glistening and slightly granular and a frothy fluid can be expressed from the cut surface. Frothy fluid is found in the trachea and bronchi. Secondary bacterial pneumonia and pleurisy common.
Judgement	Due to lack of metastatic spread, reject affected parts. If condition associated with emaciation of the carcase, reject carcase and associated offal.

JÖHNE'S DISEASE

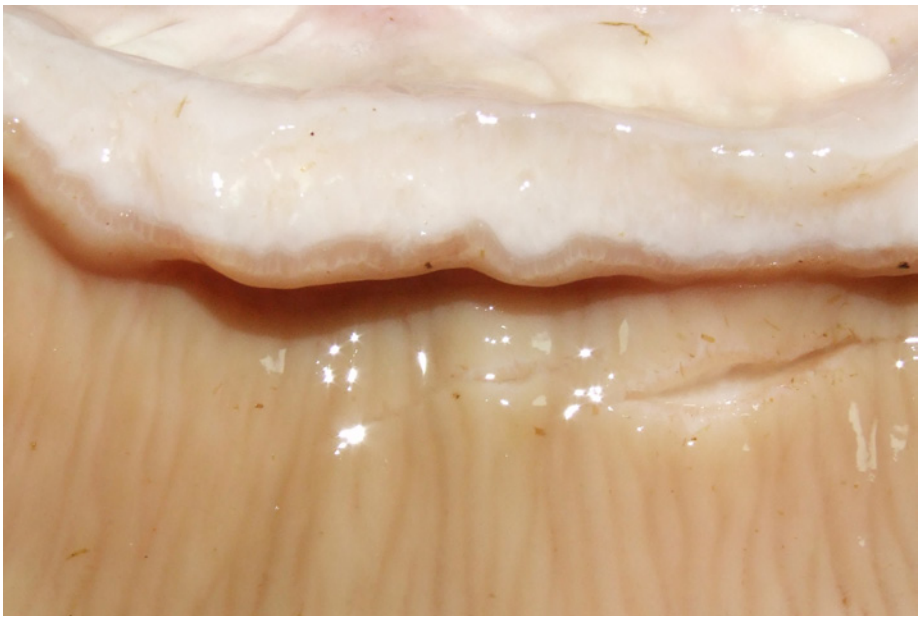
Synonyms	Paratuberculosis
Type	Bacterial
Aetiology	*Mycobacterium johnei* Aerobic acid fast rod
Pathogenesis	Chronic enteritis. Transmitted via faecal-oral route. Possibly by contamination of the dams' teats with infected feaces. After ingestion, the lymphatic tissue of the intestinal mucosa is invaded, followed by spread to the mesenteric lymph nodes after 2-3 months of multiplication. If unchecked by the animals' immune response the intestinal lesions spread until interfering with the normal function.
Clinical Signs	Tends to affect animals 2 years of age onward. Progressive emaciation. Anaemia can occur. Faecal pellets softer than usual, frequently soiling of thighs. Wool becomes brittle and is easily pulled out.
Gross Lesions	Thickening of the mucosa of the jejunum, ileum and caecum, most noticeable at the terminal ileum. Occasional yellow pigmentation of the bowel mucosa. Enlargement and oedema of the mesenteric lymph nodes which may contain focal necrosis of the cortex. Mesenteric lymph channels may become enlarged (up to 3mm diameter) and corded. Granulomas may be found in afferent lymph vessels.
Judgement	Reject carcase and associated offal if emaciated. If localised reject intestines.



Jöhne's Disease - Yellow pigmentation and corrugation of intestinal mucosa



Onset of Jöhnes disease



Thickening of intestinal mucosa

LEPTOSPIRIDIOSIS

Type	Bacterial
Aetiology	*Leptospira species*, in particular *Leptospira hardjo*. Small coiled motile bacterium.
Pathogenesis	Transmission via urine or contaminated water and inoculation of damaged skin.
Clinical Signs	Adults – Fever, abortion, mastitis, undulating fever, jaundice and anaemia
Gross Lesions	Lambs – Interstitial nephritis, jaundice, acute haemolytic anaemia Adults - Ranges from anaemia, interstitial nephritis, septicaemia.
Judgement	If systemic, reject carcase and offal. If localised reject affected parts. **Zoonotic**.

LISTERIOSIS

Synonyms	Circling disease
Type	Bacterial
Aetiology	*Listeria monocytogenes*. Gram +ve. Non-sporing rod.
Pathogenesis	*L.monocytogenes* is a common environmental bacterium found in soil, feedstuffs and faeces. Infection via inhalation or ingestion leads to localisation in predilection sites of intestinal wall, medulla oblongata and placenta. Most prone to infection are animals with erupting cheek teeth (approximately 2 years plus) and those fed silage from compromised storage conditions that allow multiplication of the bacterium.

Clinical Signs	Those associated with cranial nerve dysfunction. Unilateral facial paralysis and circling toward the affected side common, as is drooling of saliva, and recumbency. Abortion if localised in placenta.
Gross Lesions	Encephalitis and micro-abscessation of the brainstem. Focal hepatic necrosis in young lambs whose rumen is not fully functional. Placentitis with yellow necrotic foci, metritis. Haemorrhagic gastroenteritis
Judgement	**Zoonotic**. Carcase and associated offal unfit for human consumption.

LOUPING ILL

Synonyms	Ovine encephalomyelitis, Trembling
Type	Viral
Aetiology	Flavivirus
Pathogenesis	Transmission is via the tick *Ixodes ricinus* or by direct droplet infection. After multiplication in lymphoid tissue a viraemia develops followed by the virus invading the tissues of the central nervous system (CNS). Whilst in the CNS the virus appears not to be susceptible to the immune response.
Clinical Signs	Initial fever during viraemic phase, followed by clinical signs attributable to CNS infection including moving with a 'louping gait' (both fore and hind legs jumping forward in pairs), muscular tremors most notable in neck region, nervous nibbling, rear limb paralysis.
Gross Lesions	No macroscopic lesions. A secondary pneumonia may develop.
Judgement	Reject carcase and offal if associated with emaciation / oedema.
Other	**Zoonotic** through tick bite.

MAEDI-VISNA

Synonyms	Maedi, Ovine Progressive Interstitial Pneumonia, Lunger disease, OPP
Type	Viral
Aetiology	Lentivirus
Pathogenesis	Two forms Maedi (respiratory) and Visna (nervous). Long incubation period of 3-4 years. Generally affected are the lungs and udder.
Clinical Signs	Loss of body condition and respiratory difficulties. Chronic mastitis. In advanced nervous form circling, ataxia, and paralysis of the hind quarter.
Gross Lesions	Enlarged and oedematous mediastinal lymph nodes. Greyish-yellow non-collapsing lungs that are dry and 2-4 times heavier than the norm. Meat-like appearance of incised lung.
Judgement	If localised, reject affected parts. If associated with emaciation the carcase and offal are unfit for human consumption.

MALIGNANT OEDEMA

Synonyms	Big head,
Type	Bacterial
Aetiology	*Clostridium septicum.* Soil borne, Gram +ve spore forming anaerobic rod
Pathogenesis	An acute toxaemia. Infection route is through contamination of wounds.
Clinical Signs	Fever and lameness. Extensive tissue swelling that pits on pressure. Rams may show 'big head' where traumatic injury from butting allows organisms to enter subcutaneous tissues of head producing oedema of head and neck.
Gross Lesions	Dark brown to black musculature due to extensive infiltration of exudates in subcutaneous and intramuscular connective tissue.
Judgement	If localised reject affected parts. If systemic reject carcase and associated offal.

MELIOIDOSIS

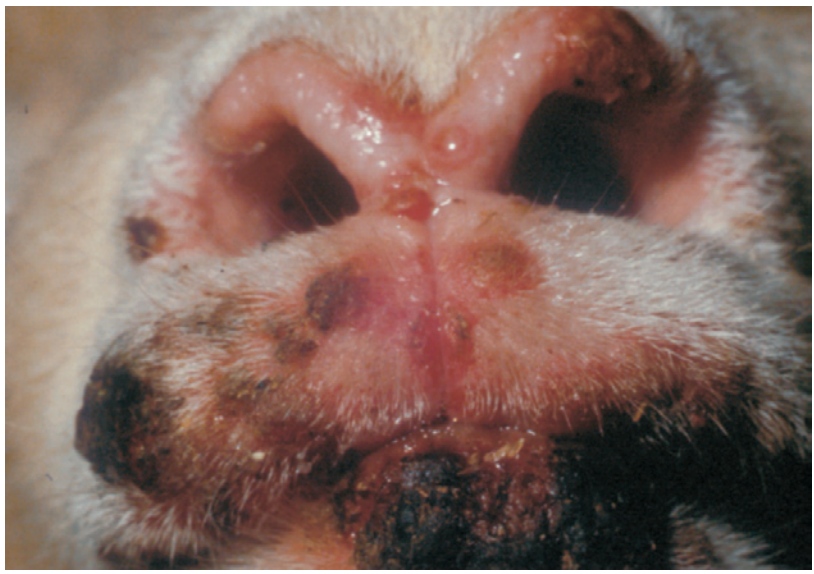
Type	Bacterial
Aetiology	*Pseudomonas pseudomallei* . Gram –ve anaerob*e*
Pathogenesis	Basically a disease of rodents that excrete the pathogen during the course of the disease. Ovine infection occurs through ingestion of faecally contaminated material, insect bites, abrasions and inhalation
Clinical Signs	Lameness, respiratory distress, weakness and recumbency and death within a week.
Gross Lesions	Multiple abscessation in most organs, especially lungs, spleen, liver, sub-cutaneous tissues and lymph nodes, particularly nodes of the thoracic cavity. The pus in the abscesses tends to be thick, greenish and of a cheese-like consistency.
Judgement	**Zoonotic**. Carcase and associated offal unfit for human consumption.

ORF

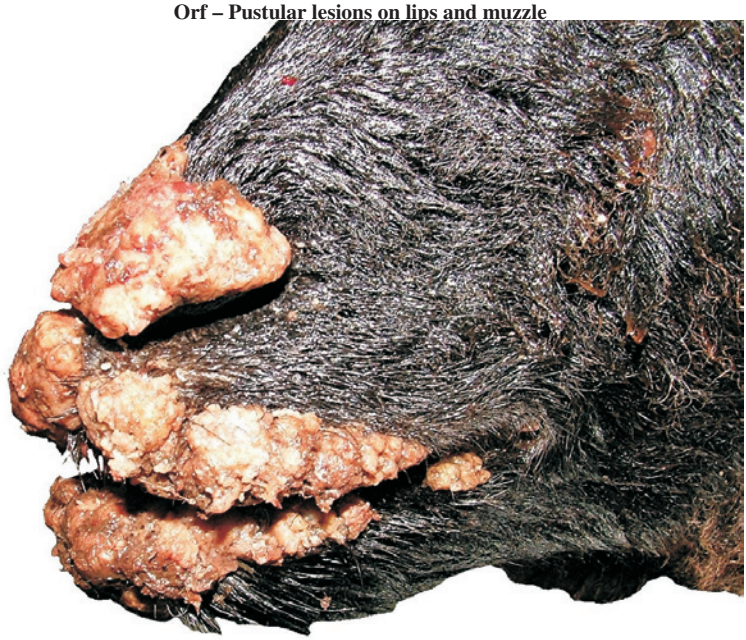
Synonyms	Contagious ecthyma, contagious pustular dermatitis, 'sore mouth'
Type	Viral
Aetiology	Parapoxvirus
Pathogenesis	Transmission is mainly via direct contact, although contaminated fomites are also implicated. Incubation ranges from 2-3 days. Fluid from ruptured pustules contains virus. Virus resistant to dessication and is found in dried crusts.
Clinical Signs	Initial small red nodules appear, usually at the lip junction, progressing to become pustular and scabby lesions of the muzzle, buccal mucosa and tongue occasionally of the udder in feeding ewes due to transmission from suckling lambs. The painful lesions around the lips of lambs reduces desire to suckle or eat leading to emaciation. Strawberry foot rot has also been attributed to orf.

Gross Lesions Lesions on internal mucosa of the mouth, especially dental palate and tongue have been confused with those of foot and mouth disease.
Necrotic lesions in lungs have been reported as has erosion of the mucosa of the upper respiratory tract.
Can lead to inflammation of the fore-stomachs and enteritis.

Judgement Reject affected parts.
If gastritis and enteritis is present the entire carcase and associated offal should be rejected. **Zoonotic**.



Orf – Pustular lesions on lips and muzzle



Orf –pustules on lips and muzzle

OVINE INFECTIOUS KERATOCONJUNCTIVITIS

Synonyms	Pinkeye, Contagious ophthalmia
Type	Bacterial
Aetiology	*Mycoplasma conjunctivae*
Pathogenesis	Transmission is via insect vectors.
Clinical Signs	Conjunctivitis, lacrimation and staining of the face. Chronic infection can lead to corneal ulceration and blindness.
Gross Lesions	None
Judgement	Reject head.

Opacity of the cornea, which can be associated with trauma or keratoconjunctivitis

Blindness due to trauma, a ewe that had been laterally recumbent

PASTEURELLOSIS

Synonyms	Enzootic pneumonia
Type	Bacterial
Aetiology	*Pasteurella haemolytica* Type A (Pasteurella pneumonia) and type T (Systemic Pasteurellosis). Aerobic, Gram –ve coccus.
Pathogenesis	A commensal organism of the upper respiratory tract and tonsils, stressors including normal husbandry procedures such as docking, dipping, shearing, climatic conditions and other diseases promote rapid multiplication, movement from tonsular material, and invasion of the lungs. Initial infections with adenoviruses and *Mycoplasma ovipneumoniae* can lower host resistance to invasion by *Pasteurella spp*., indeed the latter organism is frequently isolated from lesions in association with the others. If Type T *P.haemolytica* then passes to the blood circulation, either septicaemia or localisation of infection in areas such as the joints, meninges or udder can follow.
Clinical Signs	Depression, dyspnoea and fever
Gross Lesions	Type T: Septicaemia; hepatic and splenic haemorrhages, fatty infiltration of the liver. In older sheep acute pneumonia. Generalised congestion of the lungs and sub-pleural haemorrhages. Congestion of laryngeal mucosa. Type A: Heavy, non-collapsing lungs with purple-reddish patches. On incision the affected areas exude blood stained froth. Occasionally in the more chronic form these areas undergo necrosis and alter to a greenish-brown. Hepatisation of lung tissue is common in the cardiac lobes, as is the presence of multiple abscesses. Bronchial lymph nodes are enlarged with petechial haemorrhages.
Judgement	Systemic pasteurellosis – reject carcase and associated offal if septicaemia present. Pasteurella pneumonia – reject affected parts.

PESTE DES PETITS RUMINANTS

Synonyms	Stomatitis-pneumoenteritis complex, PPR, Contagious pustular stomatitis
Type	Viral
Aetiology	Morbillivirus
Pathogenesis	Not vary contagious. Inhalation of aerosol from coughing or sneezing appears main vector although virus is shed in faeces and ocular secretions
Clinical Signs	Acute: Fever. Dry muzzle. Dyspnoea. Serous nasal discharge. Congestive conjunctivitis. Bad breath and stomatitis. Coughing, severe diarrhoea, emaciation. Death can occur within 10 days.
Gross Lesions	Erosive stomatitis. 'Zebra stripes' in posterior colon. Congestion and enlargement of spleen. Oedematous lymphadenitis. Broncho-pneumonia. Haemorrhagic streaks in upper duodenum.
Judgement	**Notifiable in UK**. Carcase and offal unfit for human consumption.

POST-DIPPING LAMENESS

Type	Bacterial
Aetiology	*Erysipelothrix rhusiopathiae*. A Gram +ve rod. A common soil borne bacterium and commensal organism of the lower intestines.
Pathogenesis	Contamination of sheep dips with this bacterium is followed by growth. *E.rhusiopathiae* then enters into foot wounds during the dipping process.
Clinical Signs	Severe unilateral lameness affecting the lower limb and foot which feels hot. Symptoms appear within 2-3 days of dipping.
Gross Lesions	Cellulitis and laminitis. Occasionally can lead to arthritis.
Judgement	Reject affected part.

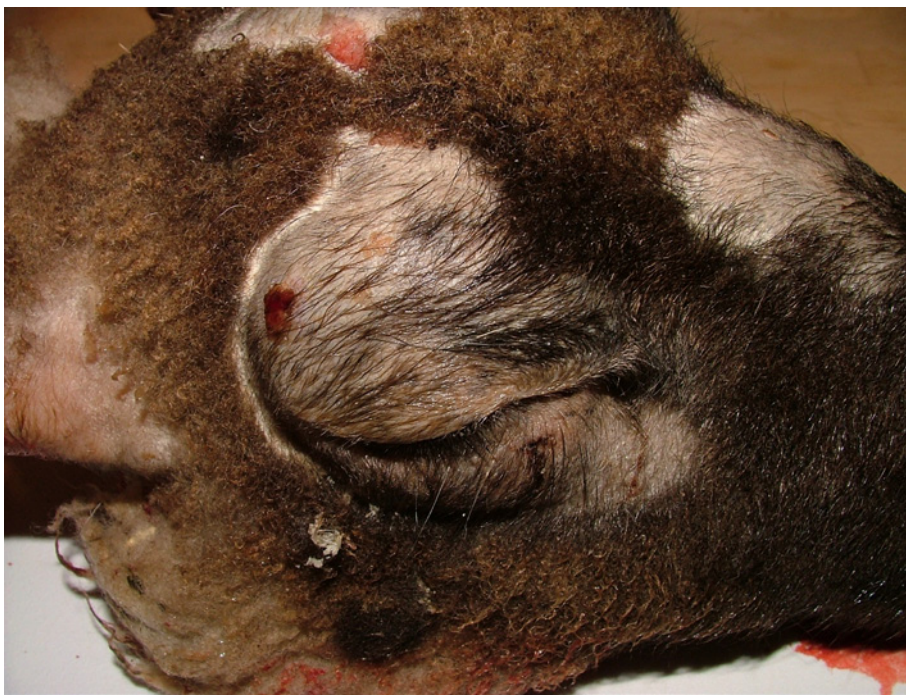
PREGNANCY TOXAEMIA

Synonyms	Twin Lamb Disease, hypoglycaemia, hyperketonaemia
Type	Metabolic Disorder
Pathogenesis	Tends to affect ewes carrying twin lambs, hence the synonym. The condition results from the increased nutrient requirement of the ewe during the latter stages of pregnancy not being met, with the developing foetuses requiring nearly half the glucose output of the liver. Extra energy is then produced by fat breakdown to ketones, some of which is reconverted into fat and stored in the liver, but unoxidised ketones may enter the blood resulting in hyperketonaemia. The decrease in energy intake can be due to insufficient pasture and also other factors such as stress, parasitic infection and diseases.
Clinical Signs	Depression, apparent blindness (sometimes referred to as 'snow blindness'), blundering into objects, reluctance to move. Spasms of head, face, and neck, the former of which may be held to one side.
Gross Lesions	Common signs include fatty infiltration of kidneys and liver. Acetonaemia, with the carcases tissue smelling strongly of 'nail varnish'
Judgement	Reject carcase and associated offal.

RINGWORM

Synonyms	Club lamb fungus, Wool rot, Lumpy wool, Ovine dermatophytosis, Ovine Ringworm
Type	Fungal
Aetiology	*Trichophyton verrucosum*
Pathogenesis	Infection occurs when the fungus invades the skin and wool follicles. Fungal spores invade wool follicles and the skin through abrasions. Fomites, such as clippers and bedding, transfer spores. The lanolin exuded by the sheep affords protection from the fungus; this protection can be lowered with excessive shearing or by washing with detergents. Most commonly encountered in show lambs that have been routinely shampooed (hence 'club lamb fungus').

Clinical Signs	Initial ulceration leads to lesions that are normally round, thick, scaly pustules on woollen areas especially the head, neck and back.
Gross Lesions	Lymphadenitis of nodes draining affected area can occur.
Judgement	Fit for human consumption unless secondary systemic bacterial infection.
Other	**Zoonotic**.



Ringworm

SALMONELLOSIS

Type	Bacterial
Aetiology	*Salmonella typhimurium*
Pathogenesis	Can occur sporadically on farms, however the most common cause of disease onset is associated with food withdrawal and stress such as occurs during marketing and prolonged lairaging.
Clinical Signs	Some affected animals die within hours of the onset of signs, most die within 1 to 5 days. Fever, depression, rapid loss of condition, dehydration, severe foul smelling, very fluid diarrhoea, sometimes with shreds of mucosa and blood.
Gross Lesions	Severe enteritis especially of the small intestine and colon. Gut contents are fluid, with occasional blood, mucus and fibrin. Congestion of the abomasum is a common finding as is hydropericardium and congestion of the liver and spleen, often accompanied by cholecystitis.
Judgement	Unfit for human consumption.

SCALD

Synonyms	Foot Scald, Necrobacillosis of the foot.
Type	Bacterial
Aetiology	*Fusobacterium necrophorum.* Gram -ve
Pathogenesis	Damage to the skin of the interdigital cleft allows *F. necrophorum* entry.
Clinical Signs	Lameness
Gross Lesions	The epidermis of the interdigital cleft becomes inflamed and oedematous.
Judgement	Reject affected parts.

SCRAPIE

Synonyms	Rida, Tremblante du mouton
Type	Prion
Aetiology	Proteinaceous Infectious Particle. An abnormal form of a normal cellular protein.
Pathogenesis	Transmission felt to be via ewe to offspring and also via contact with affected placenta and placental fluid. Long incubation period with clinical signs being displayed between 2-5 years of age.
Clinical Signs	Symptoms associated with neurological dysfunction; including dry skin / wool loss and behavioural changes such as aggression, restlessness and itching/rubbing/nibbling (hence scrapie). Teeth grinding common, as is muscular twitching.
Gross Lesions	No gross lesions. Microscopically vacuoles are found in the neurons and bilobal spongiform encephalopathy.
Judgement	**Notifiable** in UK. Total rejection.

A ewe with scrapie.The animal shook and constantly chewed at itself.

Self inflicted trauma in scrapie case above

SHEEP POX

Type	Viral
Aetiology	Capripoxvirus
Pathogenesis	Originally entered UK in an imported flock in 1847. Transmission: Direct transmission via infected animals, aerosol. Indirect transmission via fomites. Two forms encountered; skin and generalised which can be concurrent.
Clinical Signs	Skin: Pocks, scabs or pustules on unwoollen parts of skin especially the muzzle and lips. Peri-anal areas a common site as are the mammary glands. Generalised: Laboured breathing, hypersalivation and excess tear production.
Gross Lesions	Fevered carcase. Inflammation of the respiratory and digestive tract. Reddish / white firm nodules in lungs and in mucosa of trachea and pharynx.
Judgement	Total rejection
Other	**Notifiable** Disease in UK. Has not occurred in UK since 1866, however the disease was found in Greece in 2000

STRAWBERRY FOOT ROT

Synonyms	Proliferative dermatitis
Type	Bacterial

Aetiology	*Dermatophilus congolensis.* Gram +ve bacterium with fungus characteristics (branching filament production)
Pathogenesis	Lesions tend to develop after prolonged periods of wet weather
Clinical Signs	Lameness. Characteristic strawberry-like lesions on foot that haemorrhage easily.
Gross Lesions	Proliferative scab formation on coronary band of foot. The lesions produce fissures that expose raw bleeding material.
Judgement	Reject affected parts. **Zoonotic**.

Strawberry foot rot

SWAYBACK

Synonyms	Enzootic ataxia
Type	Nutritional
Aetiology	Copper deficiency
Pathogenesis	The level of available copper can be low in the pasture, but more commonly the deficiency is due to chemical induced blocking of available copper ion absorption in the rumen. Characteristically the deficiency of copper in young lambs is evidenced by a lack of myelin in the central nervous system.

Clinical Signs	Initially copper deficiency is evidenced by a band of 'uncrimped' wool which breaks easily. Hindquarter weakness ranging from a slight swaying when the lamb is moved to complete collapse of the hind legs.
Gross Lesions	Damage to brain tissue
Judgement	Fit for human consumption as long as no signs of emaciation are present.

TETANUS

Synonyms	Lock-jaw,
Type	Bacterial Toxaemia
Aetiology	*Clostridium tetani.* Soil borne, Gram +ve spore forming anaerobic rod.
Pathogenesis	Enters deep penetrating wounds, through the umbilicus at birth or through the docking/castration process. Produces neurotoxins (tetanospasmin, haemolysin) that bind to nerve membranes causing nerve excitement and continual spasmodic contraction of muscles. Incubation period averages approximately 10 days.
Clinical Signs	High temperature, stiffness and reluctance to move. Chewing ceases as masseter muscles spasm (lockjaw) accompanied by excessive salivation. Paralysis and tetanic spasms, characteristic is extension of the front legs and cocking of the tail. Death usually due to respiratory failure.
Gross Lesions	No gross lesions.
Judgement	This disease condition highlights the need for ante mortem inspection of animals, although characteristic before death, there are no post mortem indicators. Total rejection.

TICK BORNE FEVER

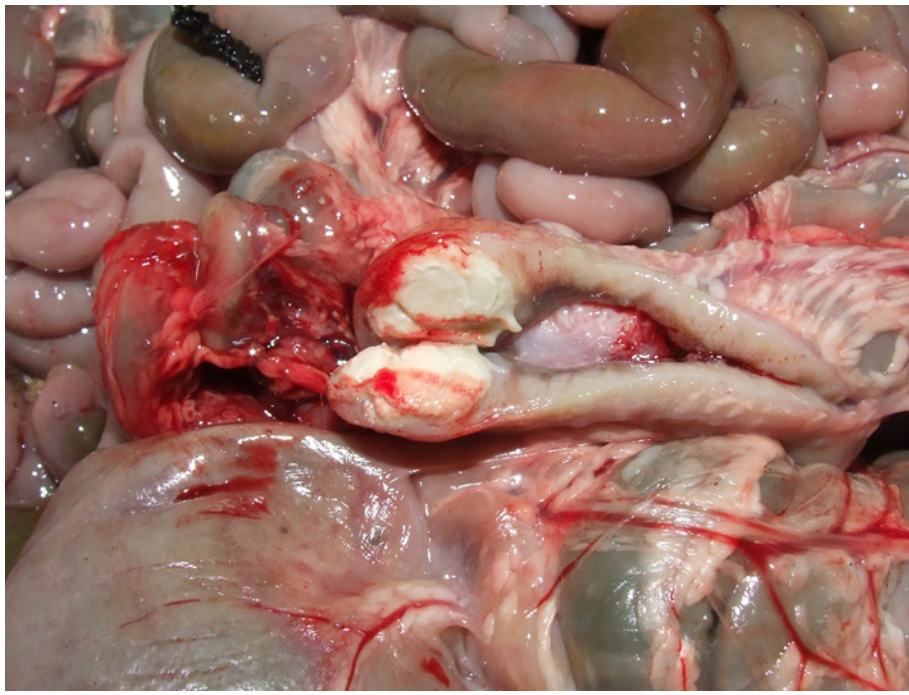
Synonyms	TBF
Type	Bacterial. Rickettsia. Intracellular parasite of white blood cells.
Aetiology	*Ehrlichia phagocytophilia*.
Pathogenesis	Inoculated via infected ticks. Incubation period is between 5 days to 2 weeks. Peaks of disease coincide with peaks of tick activity (spring and early autumn). *E. phagocytophilia* affects white blood cells (eosinophils, neutrophils and monocytes) forming colonies inside membrane lined vacuoles within the host cell cytoplasm. The infection ultimately impairs the animals' humeral and cellular defence mechanism leading to secondary infection especially with other tick associated infections such as Tick Pyaemia, Louping ill, in addition to pasteurellosis and listeriosis.
Clinical Signs	Sudden high fever lasting 4-10 days. Concurrent breathing and heartbeat rate increase. Abortions within the final stages of pregnancy are common 2 days to one week after febrile onset.
Gross Lesions	Bacteraemia
Judgement	Systemic infection. Carcase and associated offal unfit for human consumption

TICK PYAEMIA

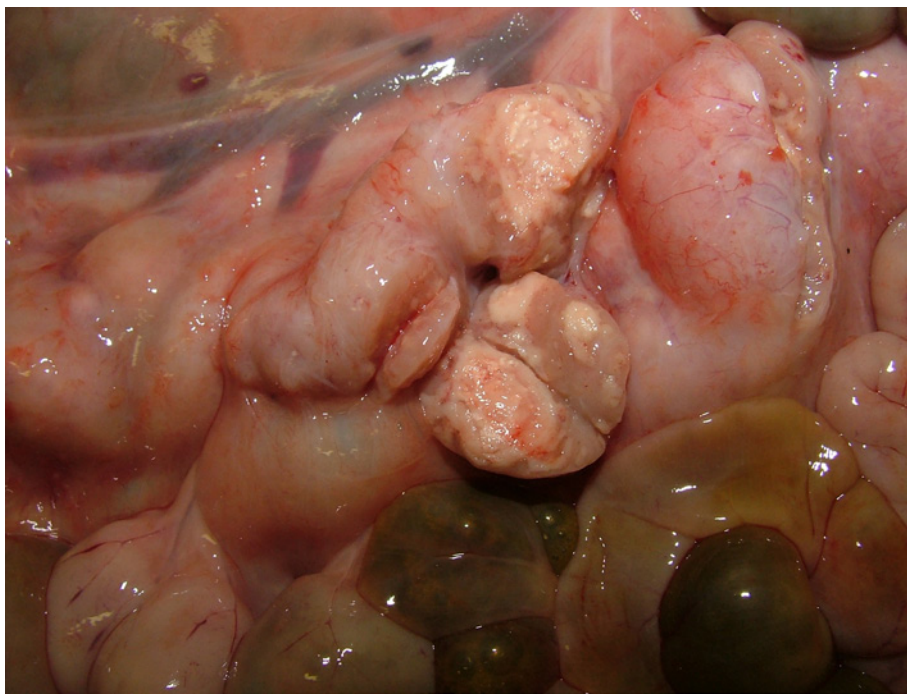
Synonyms	Staphylococcal pyaemia, Enzootic staphylococcosis
Type	Bacterial
Aetiology	*Staphylococcus aureus.* Gram +ve spheroidal
Pathogenesis	*S. aureus* is inoculated under the skin by the biting action of the tick *Ixodes ricinus*. Two forms; acute and chronic.
Clinical Signs	Acute: Tick infestation. Dullness, fever, reluctance to move and occasional partial paralysis. Chronic: Lameness, swollen joints. Superficial subcutaneous abscesses
Gross Lesions	Multiple abscesses in muscles, tendons and occasionally spinal cord. Abscesses may be found in lungs and liver. Splenomegaly. Joints may contain pus.
Judgement	Reject affected parts. If generalised or systemic reject carcase and associated offal.

TUBERCULOSIS

Synonyms	TB
Type	Bacterial. Gram +ve rods.
Aetiology	*Mycobacterium bovis*.
Pathogenesis	Slow developing chronic wasting disease. Various forms depending on route of infection and secondary spread. Bacterium can grow inside phagocytes; the internal spread of infection being mainly through the lymphatics, a primary focus normally develops in the afferent node for the area. Escape of bacilli into the blood circulation can lead to a generalised condition. The bacilli grow and a tubercle is formed around them, the connective tissue capsule enveloping the bacilli and macrophages and a central area of necrotic material.
Clinical signs	In advanced tuberculosis coughing and progressive emaciation are the main ante mortem signs
Gross lesions	Gross enlargement of afferent lymph nodes containing yellowish white granulation tissue. Formation of tubercles at primary foci, spread can be metastatic or through contact; respiratory TB lesions found in the lungs (apical and cardiac lobes most common) can spread to the surrounding parietal pleura forming visceral and parietal pleurisy and further tubercles which hang in grape-like clusters. Digestive TB usually follows from respiratory TB (ingestion of expectorated material) but can occur as the primary foci if infection if gained maternally. Initial sites of infection found in mesenteric lymph nodes followed by hepatic nodes then the liver and spleen.
Judgement	Reject affected parts, if systemic or generalised reject entire carcase and associated offal. **Notifiable** in UK
Other	Tuberculosis is a rare condition in sheep, however it can and does occur in areas where tuberculosis is endemic, especially in the wild deer population.



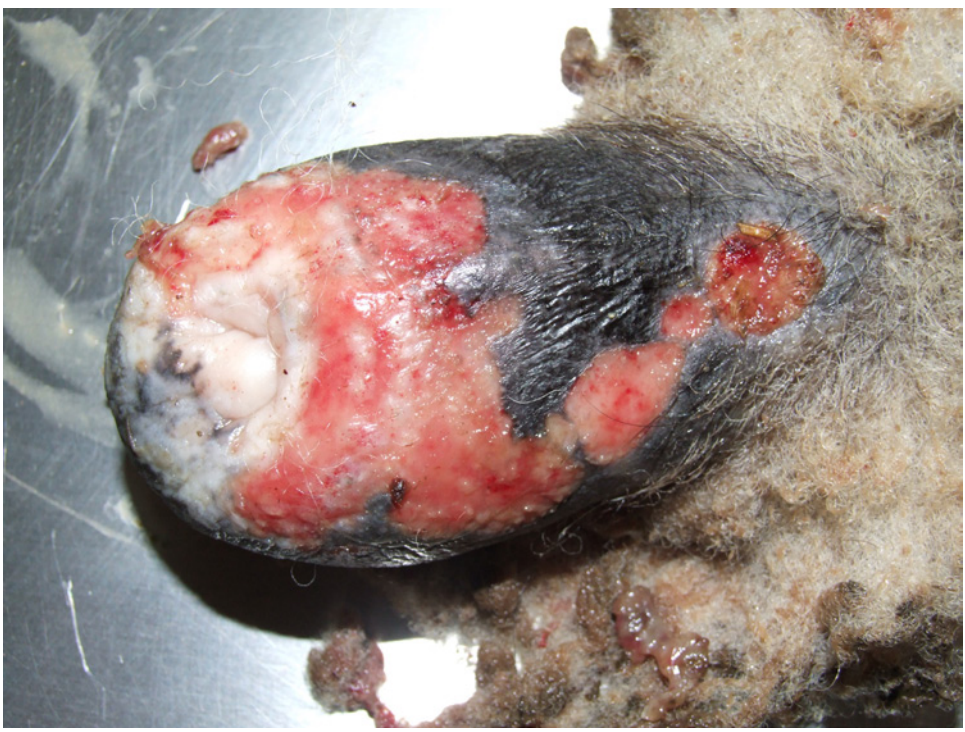
Tuberculosis in mesenteric lymph nodes



Tuberculosis in mesenteric lymph nodes

ULCERATIVE DERMATOSIS

Synonyms	Venereal balanoposthitis, lip and leg ulceration
Type	Viral
Aetiology	Paravaccinia
Pathogenesis	Two forms encountered, venereal and lip and leg ulceration.
Clinical Signs	Venereal: Ulceration of the vulva and prepuce/penis Lip and leg: Ulcers around the mouth and nose and on legs.
Gross Lesions	Formation of scabs covering crateriform ulcers on dermis that in the chronic form may perforate the dermis. Craters haemorrhage easily and occasionally contain creamy, non-odorous pus. Leg lesions tend to form above the hoof up to the tarsal joint. Venereal lesions in ewes can lead to oedema and scab formation of the lips of the vulva. In males complete covering of the preputial orifice may lead to an inability for it to withdraw over the glans penis, thereby affecting the reproductive process.
Judgement	Reject affected parts. Check carcase for signs of secondary bacterial infection.



Ulcerative Dermatosis – ram prepuce

WHITE LIVER DISEASE

Type	Nutritional
Aetiology	Vitamin B_{12} / Cobalt deficiency
Pathogenesis	Dysfunction of the ruminal microbes that produce vitamin B12 using available cobalt, leading to a reduction in levels of two coenzymes that influence both DNA synthesis and the production of a constituent of glucose.
Clinical Signs	Emaciation, watery ocular discharge, debility.
Gross Lesions	Livers become pale, swollen and friable due to fat accumulation
Judgement	Reject affected livers. Reject carcase and associated offal if emaciated.

WHITE MUSCLE DISEASE

Synonyms	Stiff lamb disease, nutritional muscular dystrophy, WMD
Type	Nutritional Deficiency
Aetiology	Selenium and / or vitamin E deficiency
Pathogenesis	Affects musculature
Clinical Signs	Signs dependant on muscles affected. Stiffness of hind legs, arched back. Exacerbated by exercise. Acute and rapid wasting of skeletal muscles. Dyspnoea in cases of cardiac type.
Gross Lesions	Oedema due to deficiency of the walls of the vascular system is common in cases of selenium / Vitamin E deficiency. Skeletal muscle: Groups of muscles either side of the body tend to be bilaterally affected. The muscle is paler with a chalky-white colouration or striations caused by the deposition of calcium. Cardiac muscle: The right ventricle tends to be most commonly affected with the presence of whitish plaques below the endocardium.
Judgement	Reject affected parts. If carcase is emaciated or oedematous reject carcase and associated offal.

YERSINIOSIS

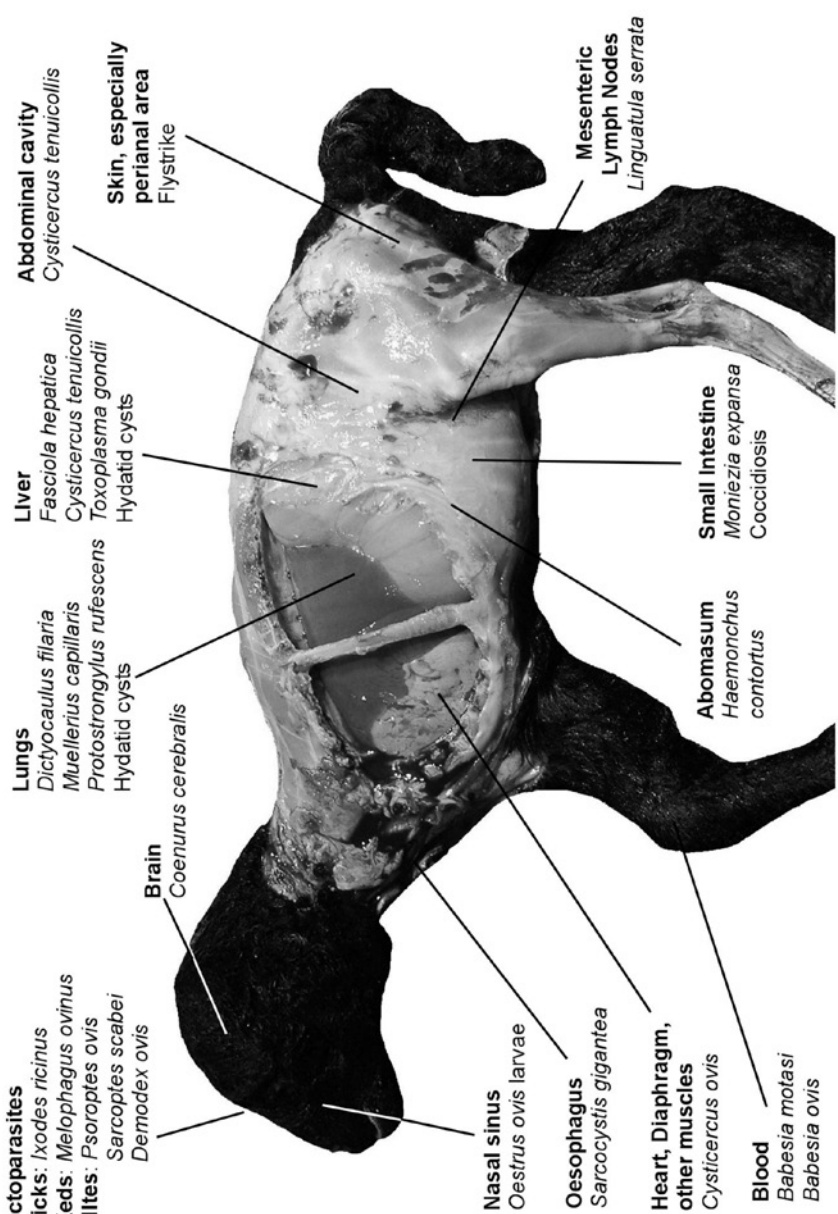
Type	Bacterial
Aetiology	*Yersinia pseudotuberculosis* and *Yersinia enterocolitica*
Pathogenesis	Infection is acquired by the oral route. The organism appears to be a commensal of sheep. Conditions that increase stress (cold weather in association with increased handling) appear to increase the shedding of the organism by carrier sheep. Wild birds and rodents have also been implicated in the transmission of the disease. On ingestion the bacteria attach to, and subsequently invade, epithelial cells of the intestinal mucosa. Surface proteins present on the bacterium confer a level of resistance to phagocytosis; an exotoxin also produces focal hypersecretion that enhances the virulence of the pathogen.

Clinical Signs	Dark green to black diarrhoea, which tends to be mild and chronic and which leads to dagging around the perineum and hocks. Mild dehydration and poorness also noted.
Gross Lesions	Abnormally fluid intestinal contents. Congestion and oedema of the wall of the small intestine, thickening of the wall of the large intestine, small haemorrhagic foci and erosions may be found. Congestion and oedema of mesenteric lymph nodes. Multi-focal abscessation of the liver can occur.
Judgement	Reject affected parts. If associated with emaciation and oedema, reject affected carcase and associated offal.

3

PARASITES

MAIN PARASITES OF SHEEP



PARASITISM

Parasites can be defined as plants or animals that live on or within another living organism at whose expense it gains some advantage whilst giving nothing in return. The host/parasite association can be complicated. The type of parasite encountered ranges from viruses (intracellular parasites) that can only reproduce in a living cell, to protozoa (single celled organisms) to intestinal worms and insects. Although they are parasitic, viruses are generally treated as a separate group, and we will only be considering parasites that are internal (endoparasites) and external (ectoparasites) that affect sheep.

During post mortem inspection of the ovine species, the presence of parasites and their affects on the host tends to be the most common condition encountered.

Parasites may have a direct or indirect lifecycle. A direct lifecycle means that the parasite can only complete the lifecycle by parasitizing the host. An indirect lifestyle involves at least one secondary (or intermediate) host. In the case of sheep, the ancestral association with canines both as control and predator has led to a number of parasites relying on the ingestion of parts of infected ovine carcases by canines to complete their lifecycle. To this end the infective parasite stage in sheep forms infective hibernation stages, usually in the form of cysts, awaiting digestion by canids and their subsequent activation to evolve to their adult form.

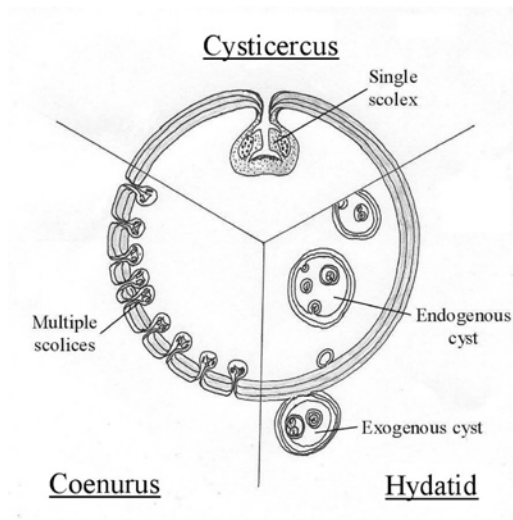
These cysts, or metacestodes, present in differing forms in preferred parts of the body known as predilection sites, some larvae having an affinity for musculature with a good blood supply such as the heart, diaphragm, others only maturing within the central nervous system. Once the larvae have reached this predilection site it develops into one of three metacestodes in sheep, a *cysticercus*, a *coenurus* or a hydatid.

CYSTICERCUS

A cysticercus is a fluid-filled cyst containing a single protoscolex (the scolex is the attachment organ of the tapeworm, in this case infolded within the structure of the cysticercus).

COENURUS

A fluid filled cyst containing numerous protoscolices.



HYDATID

A hydatid cyst contains a germinal layer from which multiple invaginated scolices are formed. These scolices can be found within the fluid or grouped together within miniature cysts known as 'brood cysts'. Hydatid cysts also have the ability to produce 'daughter cysts' which also have the ability to produce scolices. If established internally they are known as endogenous cysts, if found external to the main cyst they are termed exogenous cysts.

ENDOPARASITES

ENDOPARASITE GROUPS

The endoparasites of sheep are divided into four categories depending on their shape and structure: Protozoa, Nematodes (Roundworms), Trematodes (flukes) and Cestodes (Tapeworms).

HOST / PARASITE RELATIONSHIP

Endoparasites undergo a three-step association with the host animal. Firstly, the parasite must infect the host via the intestines, respiratory system or the skin. Secondly the individual parasite must be maintained within the host, this includes feeding, growth and migration within the host. Thirdly the species must be maintained, which means reproduction and the dispersal of the infective agents.

Each of these stages of association present different hurdles for the parasite.

INFECTION

Obviously the initial stage of parasitic development is gaining entry to the host animal. The parasite has to survive in some form in the atmosphere, whilst remaining available to enter the host. The lifecycle of most endoparasites includes a hibernation stage, for example as a cyst or egg (the infective agent) where the immature parasite lies dormant until suitable conditions trigger the release of the agent. The availability of the infective agent to host entry is achieved in various ways; some stages of a lifecycle may include the infective agent existing in a secondary or intermediate host such as worms, beetles or ants that are ingested by the primary host. The single overriding factor in the availability of the infective agent to host entry is mathematical. Infection of the host is a matter of chance; the vast numbers of infective agents produced by the adult parasite increases the odds.

INDIVIDUAL MAINTENANCE

Once the agent has entered the hosts' body, by whatever means, it has to be able to migrate to its preferred site of habitation (predilection site), where it can mature. This can involve migration through body tissue, or at the very least, passage through the digestive system. Some parasites use the digestive process to activate the infective agent, others produce secretions to neutralise the effects of the gastric juices. The migration and settling of the immature parasite will also prompt the host animals' immune response to a foreign agent. This is overcome by some parasites by protective secretions, and actively encouraged by others as part of their lifecycle, using the immune response to isolate them and encase them in fibrous material where they form cysts.

PARASITE MAINTENANCE

On reaching their predilection site the parasite takes nutrients from the host to mature. A successful parasite can be considered one that infects, lives, reproduces, and infects other hosts without killing the primary host.

SPECIES MAINTENANCE

The role of any organism, from bacterium to human, is propagation of the species. Eggs are passed by mature parasites in the faeces, and some become infective agents, and the cystic stages of other parasites gain entry into the host through ingestion.

EFFECTS ON THE HOST

The effects on the host of parasitism vary according to the parasite type and the level of infestation However the main effects can be considered as being mechanical, depletive, anaemic, vector and destructive. The mechanical effects range from cell/tissue destruction by the parasite and migratory larvae, to obstruction caused both by the presence of the parasite (for example *Moniezia expansa* blocking the intestines and *Dictyocaulus filaria* producing localised blockage of bronchi) or pressure exerted in and upon other organs by the cystic stage (Hydatid cysts in abdominal cavity and *Coenurus multiceps* in brain tissue).

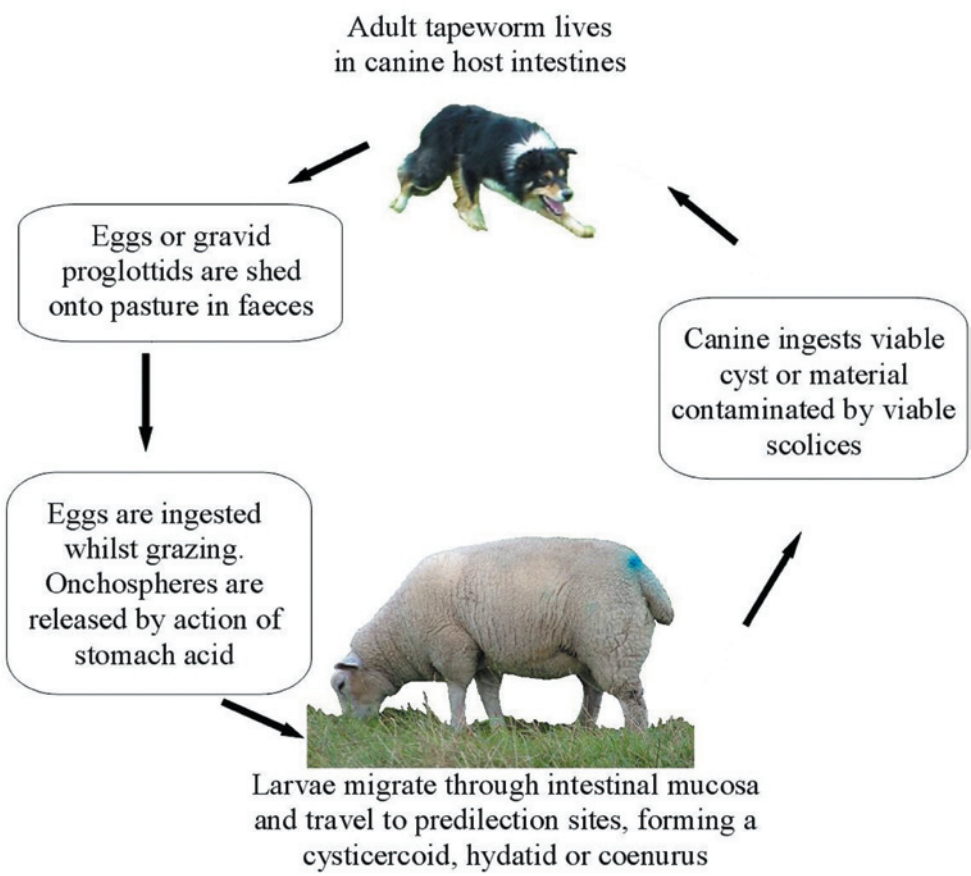
The depletive affects are ostensibly due to the parasite stealing nutrient from the host or preventing normal digestive function by, for example, affecting the porosity of the intestinal mucosa.

The anaemic effects parasites can have on the host are best illustrated by a severe *Haemonchus contortus* infection, the adult worms feeding on blood through the abomasal mucosa can create a severe blood loss which can be noted by pallor in the mucous membranes. The most obvious parasitic vector is the hard tick *Ixodes ricinus*, culpable in the transmission of various diseases and zoonoses including Babesiosis, Tick Pyaemia and tick borne fever.

The destructive effects of parasitic infestation are most apparent in the damage to brain tissue by Coenuriosis, abortions due to *Toxoplasma gondii* and the cellular destruction of the intestinal mucosa by the intracellular parasites of the Eimeria species.

DOG TAPEWORM PARASITES

CANINE TAPEWORMS - GENERIC LIFECYCLE



CYSTICERCUS OVIS
Sheep measles, Muscular Cysticercosis

This is the name given to the metacestodes formed by the embryos of the canine cestode *Taenia ovis* in the muscles of sheep. The cysts are oval and 3-8mm in size and when viable contain a visible single scolex.

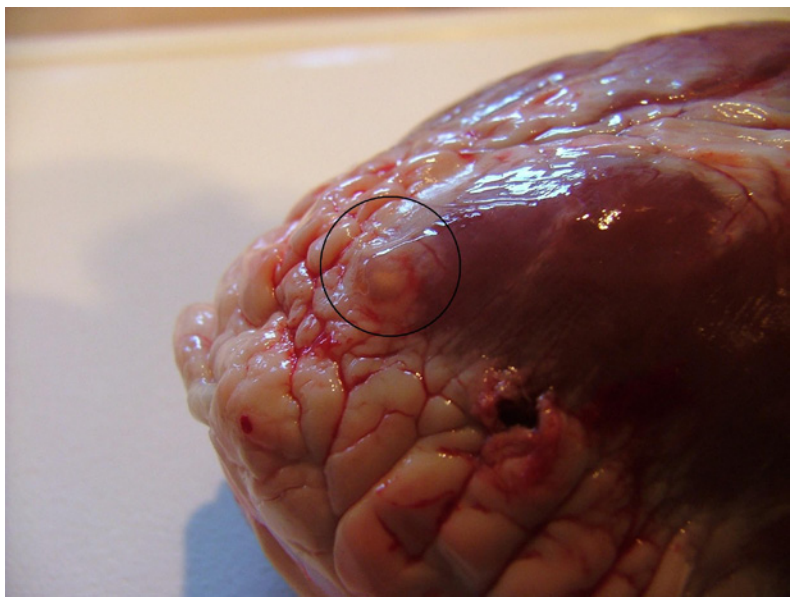
In the dog the mature tapeworm is approximately 0.5 - 1 metre in length and lives in the small intestine. It is made up of a head with 24-36 hooks that hold it in place, a neck, and segments known as proglottids. These proglottids bud from the neck and sexually mature as they move away from the head. The dog sheds mature proglottids containing eggs in its faeces.

When these eggs are deposited on pasture they are ingested by sheep, where the embryos hatch out in the intestine. They then penetrate the wall of the ileum by means of oral hooks and are carried in the bloodstream or lymphatic circulation to skeletal or cardiac muscles where they form a cysticercus containing a single invaginated scolex. The life cycle is completed when a dog eats infected muscle containing a viable cyst which dissolves allowing the liberated scolex to attach to the small intestine and mature.

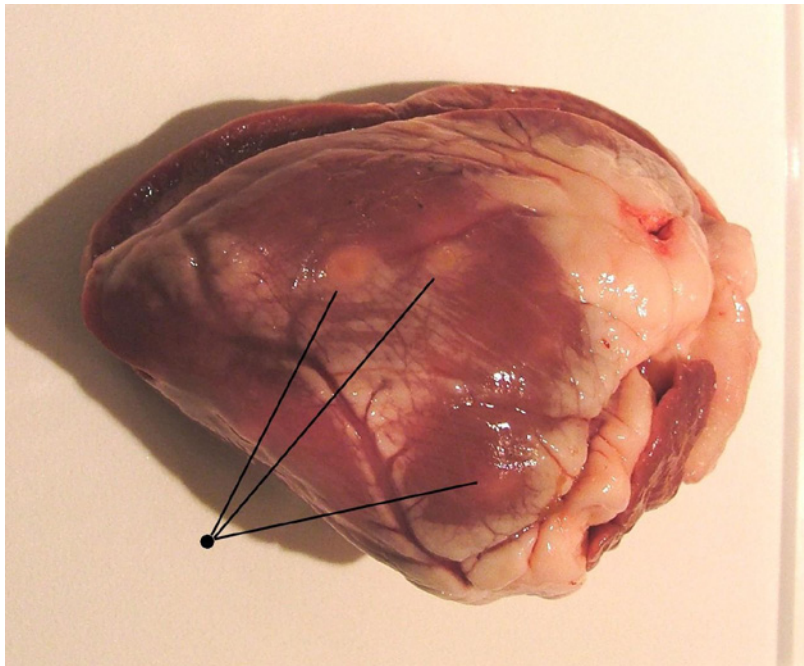
The cysts are generally found in muscle with a good blood supply such as the heart, the diaphragm, tongue, cheek muscles and skeletal muscles. Sheep appear to tolerate a large number of metacestodes without displaying clinical signs unless there is heavy infestation of the heart that may lead to cardiac failure.

If localised, the affected area is rejected, for example the heart or diaphragm. If the cysts are generalised i.e. they are found throughout the body, the carcase and offal are rejected.

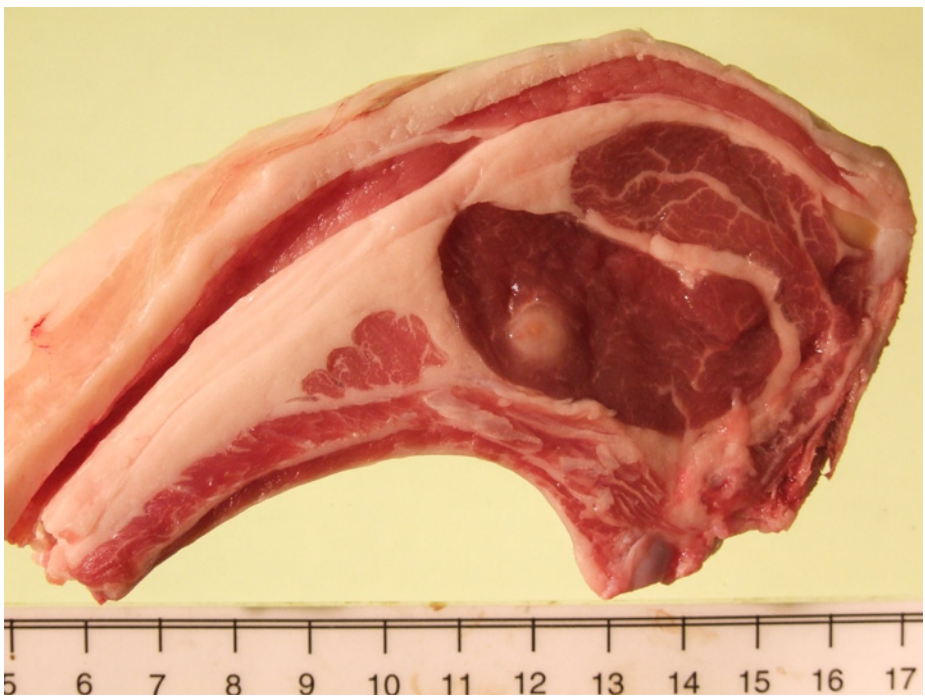
In cases of doubt it may be worth asking for the carcase to be cut down into primal joints that then provides more cut surfaces to examine for cysts.



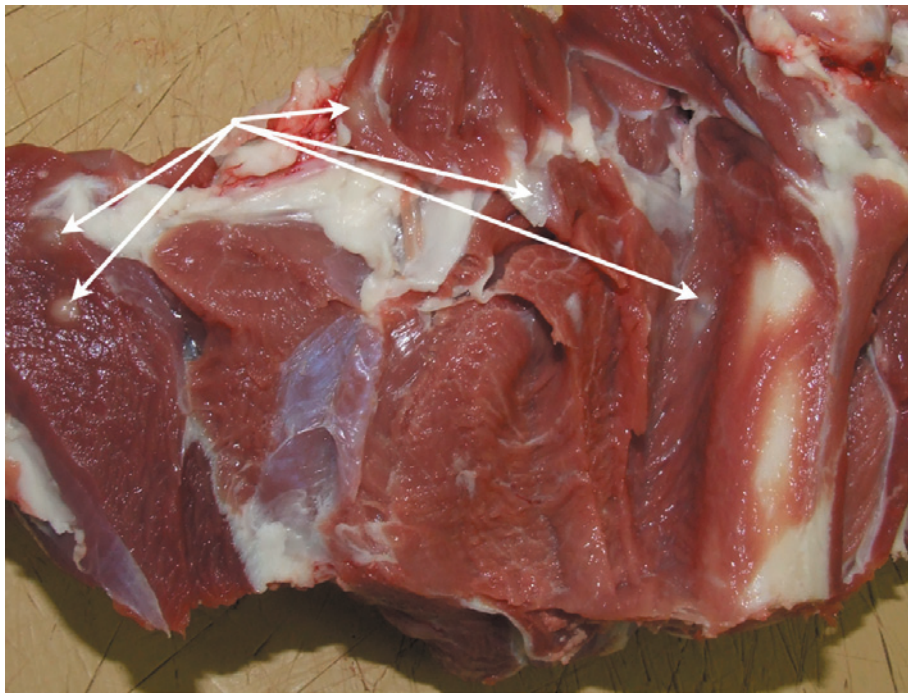
A viable Cysticercus ovis with visible scolex (circled)



Three cysts clearly visible in an ovine heart



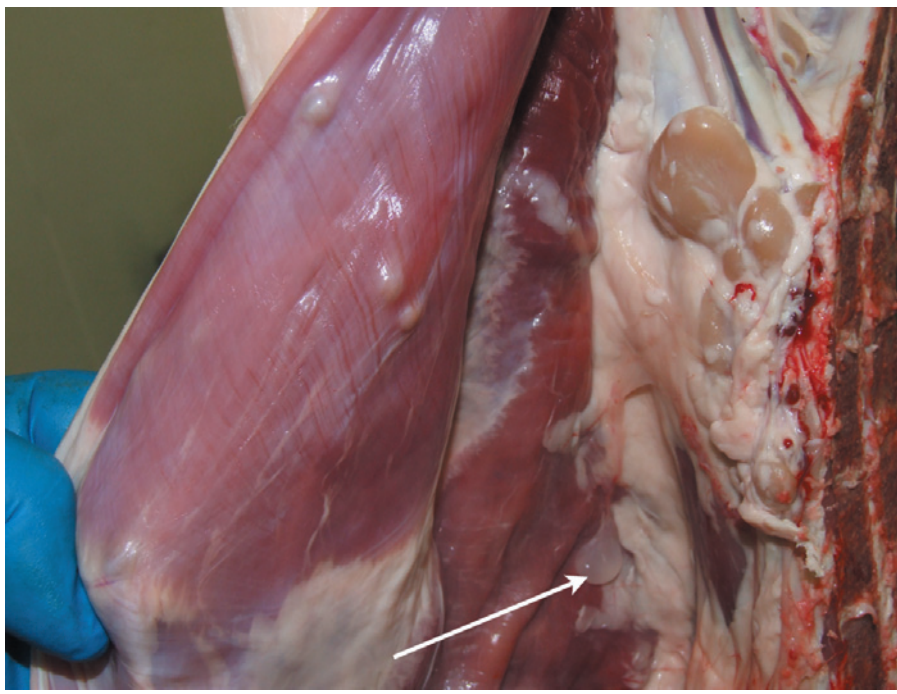
A degenerating cyst in a lamb chop



Cysts evident (arrowed) in an opened leg joint

Multiple cysts in diaphragm

Degenerated cysts present in tenderloin muscle



Multiple *C. ovis* in the flank and a *C. tenuicollis* cyst (arrowed)

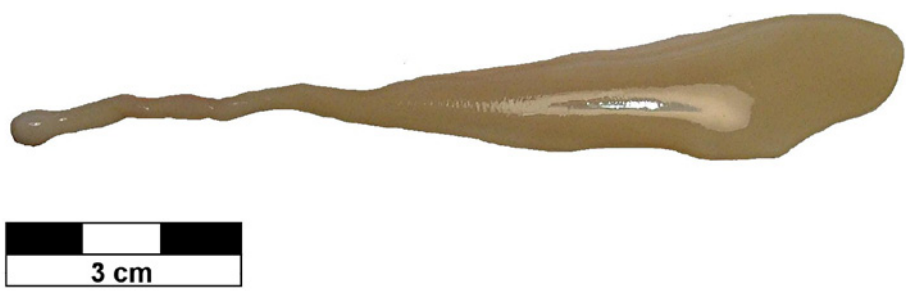
CYSTICERCUS TENUICOLLIS
Visceral cysticercosis, VC, Sheep bladder worm

This is the name given to the fluid filled cysts formed by the embryo of the canine tapeworm *Taenia hydatigena*. The adult tapeworm can be up to 5 metres in length made up of a scolex and segments and lives in the intestine of the host animal. The triangular eggs and segments are passed in the faeces of the host as they mature

When ingested by the sheep (the intermediate host) the intestinal secretions digest the outer membrane (the embryophore) of the egg and activate the embryo (onchosphere). Using its hooks the onchosphere tears through the lining of the intestine to reach the bloodstream whereby it is carried to the liver. The onchospheres migrate through the liver substance for about 4 weeks before emerging at the surface, perforating the liver capsule and entering the abdominal cavity. At post mortem inspection, the migration of the onchospheres through the liver substance is indicated by 'serpentine tracts' throughout the liver. These tracts are initially blood filled 10-20 days post infection but become yellowish when filled with leucocytes from 25-35 days. If these are evident the liver is rejected as unfit for human consumption. Mechanically transported bacteria may also produce a parasitic peritonitis.

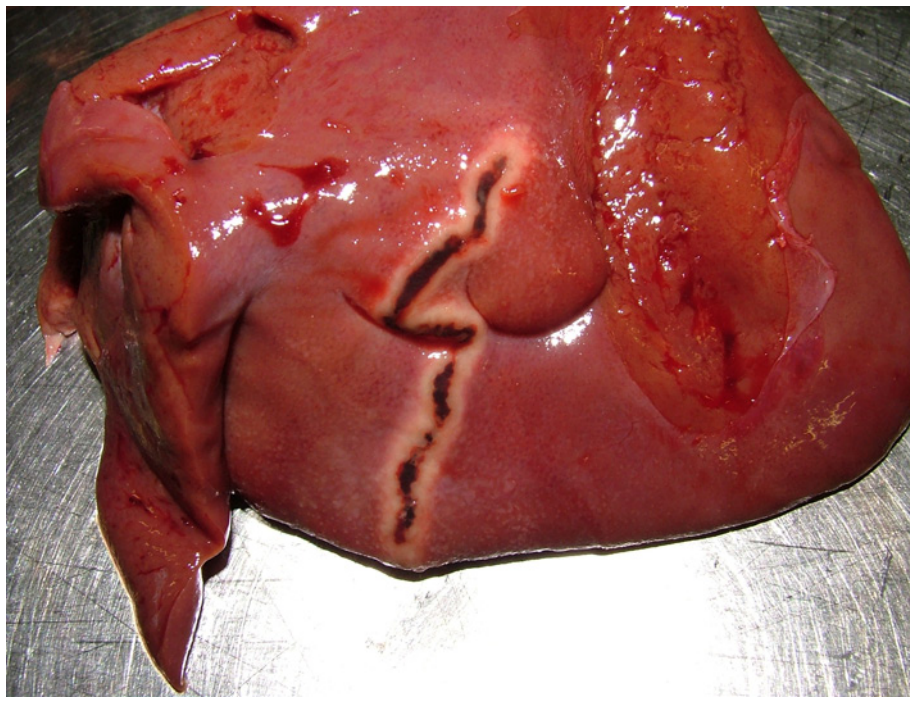
The embryos attach to the surface of the abdominal viscera, especially the omentum fat, lose their hooks and develop into a large fluid filled sac containing a single head or scolex. Not all the embryos develop into cysts, some degenerate into caseous lesions on the liver surface. A common area for cyst development is the umbilical fissure of the liver. The life cycle is completed when a dog eats the viable cyst.

Unlike *C.ovis*, the infestation of sheep with *C.tenuicollis* can have severe clinical signs including hepatitis and peritonitis. The carcase and associated offal should be rejected if the infestation is severe and leads to emaciation and oedema.

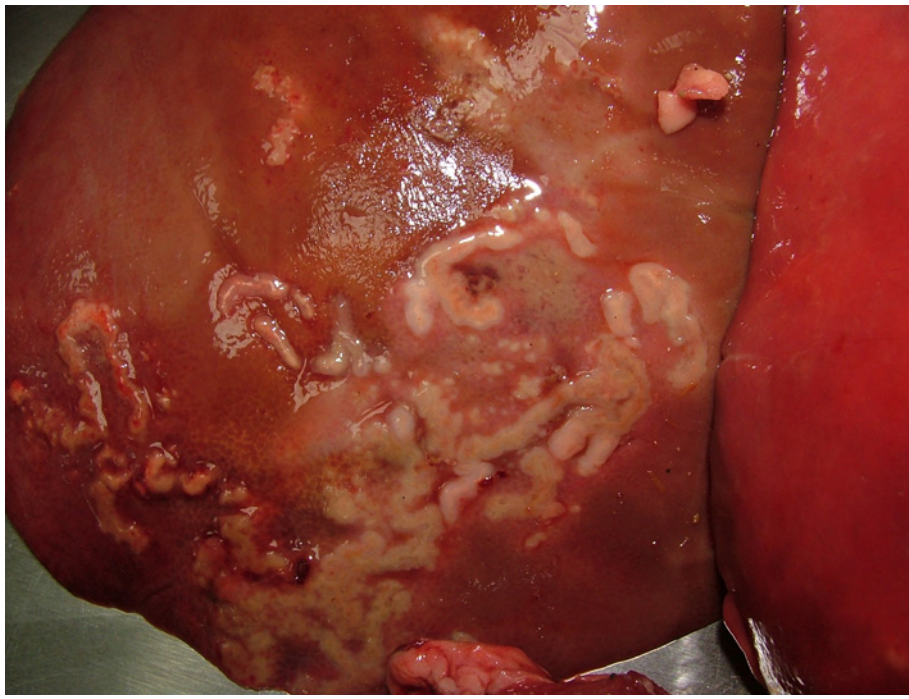


***C. tenuicollis* 'bladder worm' removed from a cyst**

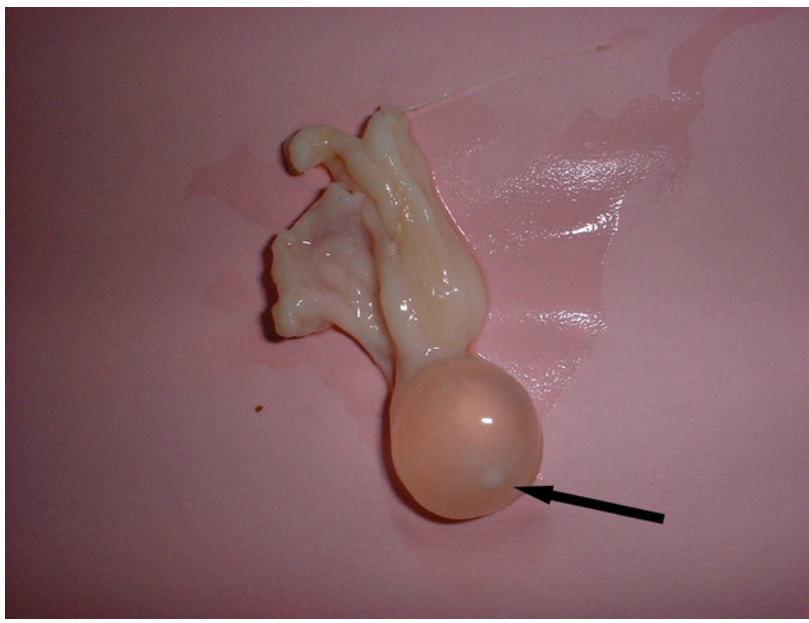
Typical 'serpentine tracts', scar tissue left by migrating parasite larvae



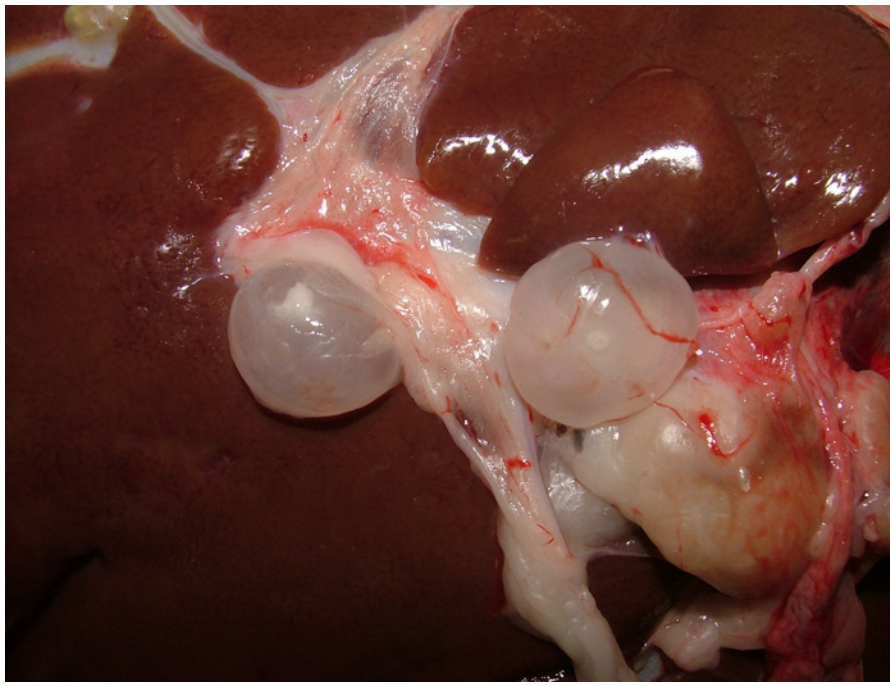
Larval migration scar approximately 20 days post infection. Haemorrhagic tracts being replaced by leucocytes



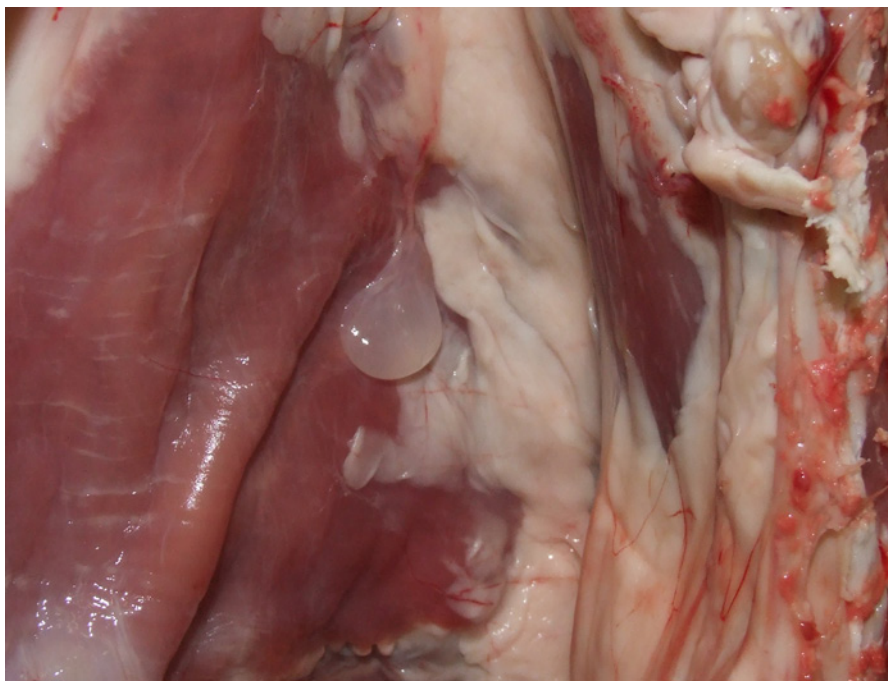
Scarred tracking in a liver



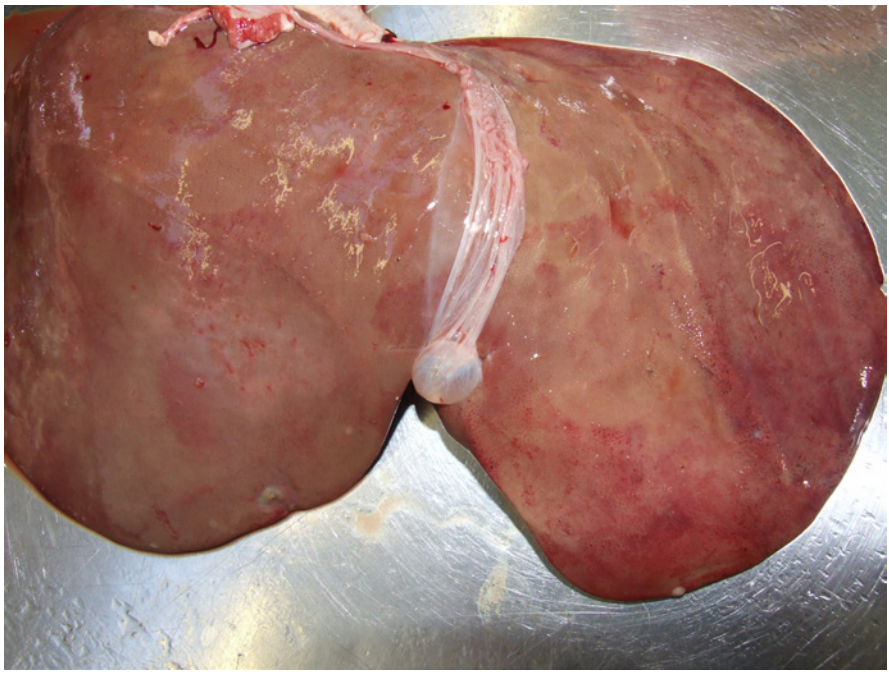
The single scolex is clearly visible in this cyst.



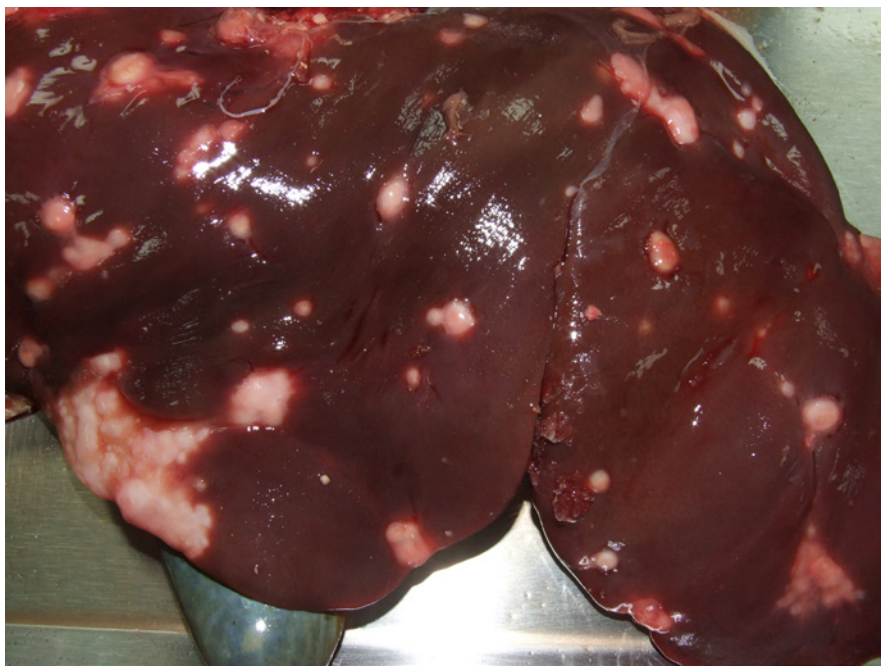
Two cysts near hepatic lymph node



***C. tenuicollis* cyst adherent to abdominal wall**



***C. tenuicollis* cyst visible in umbilical fissure, a common attachment area**

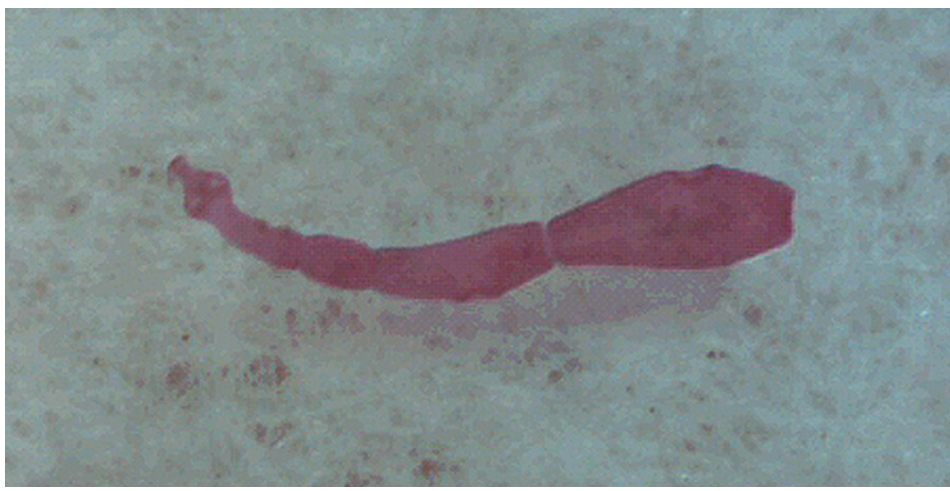


Arrested development of larvae in a sensitised older sheep with an active immunity. The larvae provoke an immune response whilst in the liver resulting in scarring and abscessation

HYDATIDOSIS

Hydatidosis is the condition where fluid filled cysts of the intermediate or metacestode stage of the canine tapeworm *Echinococcus granulosus* are found in the parenchyma of internal organs, normally the liver or lungs. *E.granulosus* has the ability to produce millions of scolices from one ingested onchosphere due to the germinal epithelium of the hydatid cyst.

Echinococcus granulosus (also known as the dwarf dog tapeworm) is found in the canine intestine and is approximately 5-6mm in length. It consists of a head or scolex and three segments. The eggs (embryophores) contain one embryo (onchosphere), which has six oral hooks, and are passed in the faeces at a rate of one per week. The onchosphere can remain viable outside the host on the ground for up to two years.



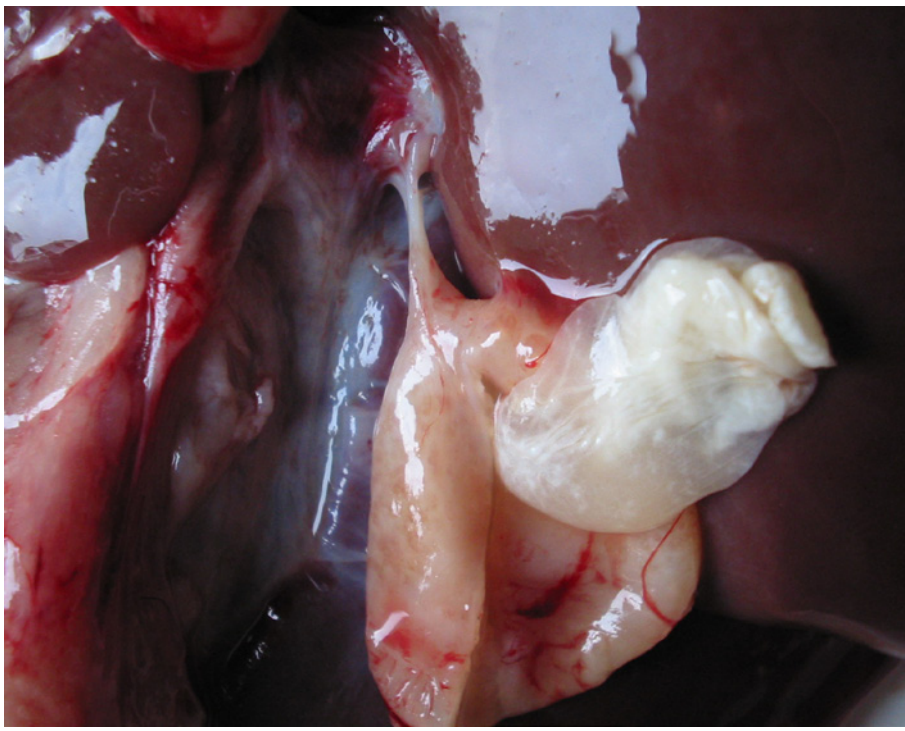
***Echinococcus granulosus* adult**

When the intermediate host ingests the embryophore, the onchosphere penetrates the gut wall and travels in the blood to the liver or in the lymphatic system to the lungs. Occasionally onchospheres escape into the general circulation and cysts are formed elsewhere in the body, including the CNS and kidneys.

Humans can also be intermediate hosts, when onchospheres are ingested accidentally from the coats of dogs, or from eating vegetables or other foodstuffs contaminated by dog faeces containing onchospheres. As such, hydatidosis is of Veterinary Public Health concern as there is no treatment except surgical excision of the cysts dependent on location, and cyst rupture can lead to anaphylaxis or further cyst development due to metastatic spread of daughter cysts.

The cysts (hydatid) are slow growing, reaching maturity in 6-12 months. In the liver the cysts can reach a diameter of 20cm. A viable cyst is filled with a sterile fluid produced by interaction of parasite molecules and host serum components.

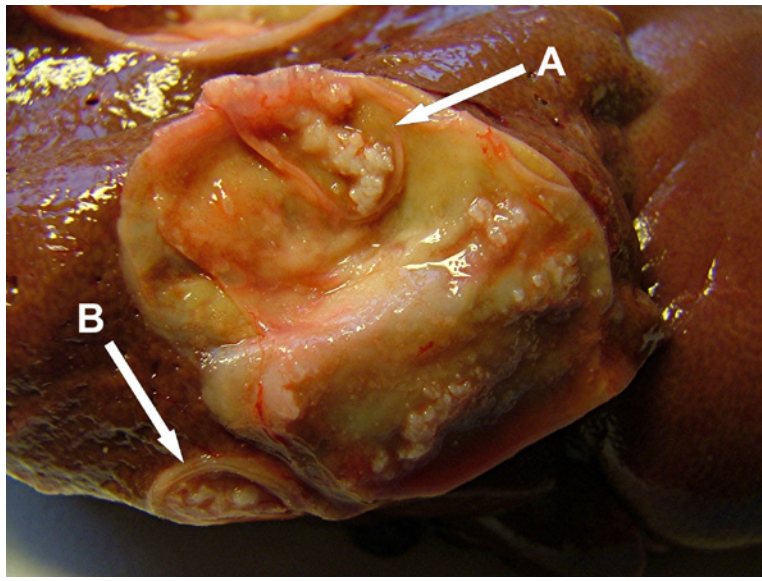
The cyst wall has three layers; an outer wall produced by the host in response to the parasitic presence; a middle laminated layer that provides the cyst with stability and protection against bacterial infection but allows nutrient transfer; and a thin internal germinal wall, the latter layer producing the scolices. which occasionally become detached from the cyst wall and float freely in the fluid, giving rise to the term 'hydatid sand.'



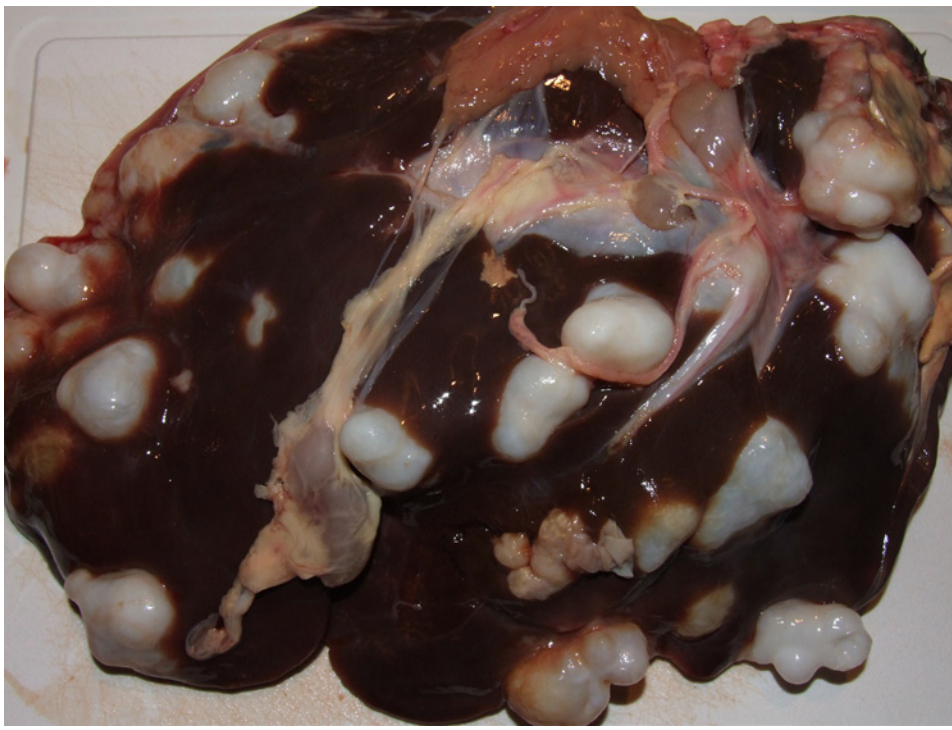
A hydatid cyst

The lifecycle is completed when a dog ingests the viable cysts.

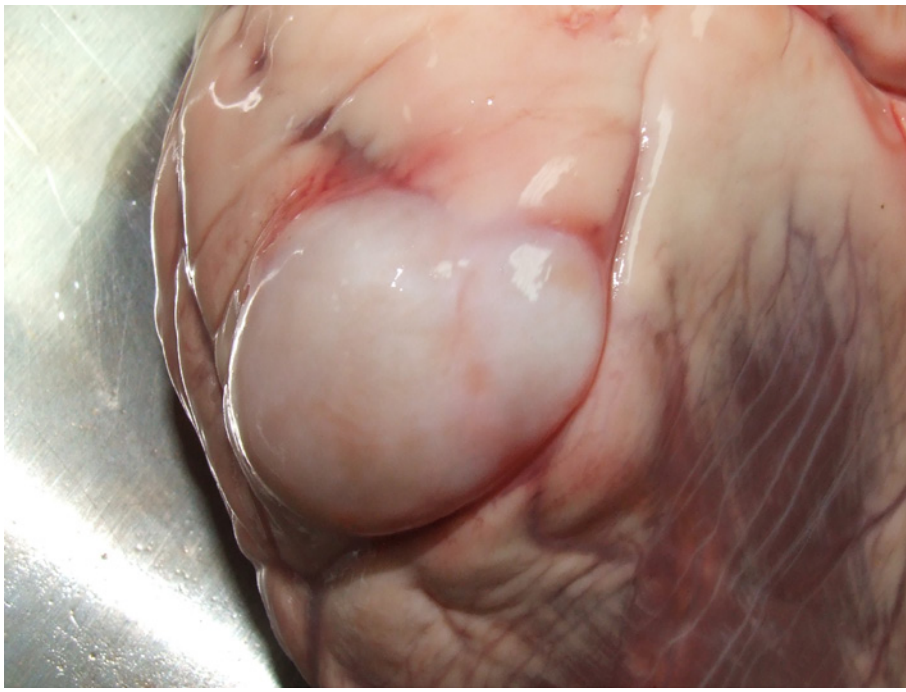
Offal containing hydatid cysts are rejected as unfit for human consumption and care must be taken to prevent feeding of affected offal to dogs. In certain areas of the United Kingdom there are measures being put in place to reduce the incidence of hydatidosis due to the risk to human health.



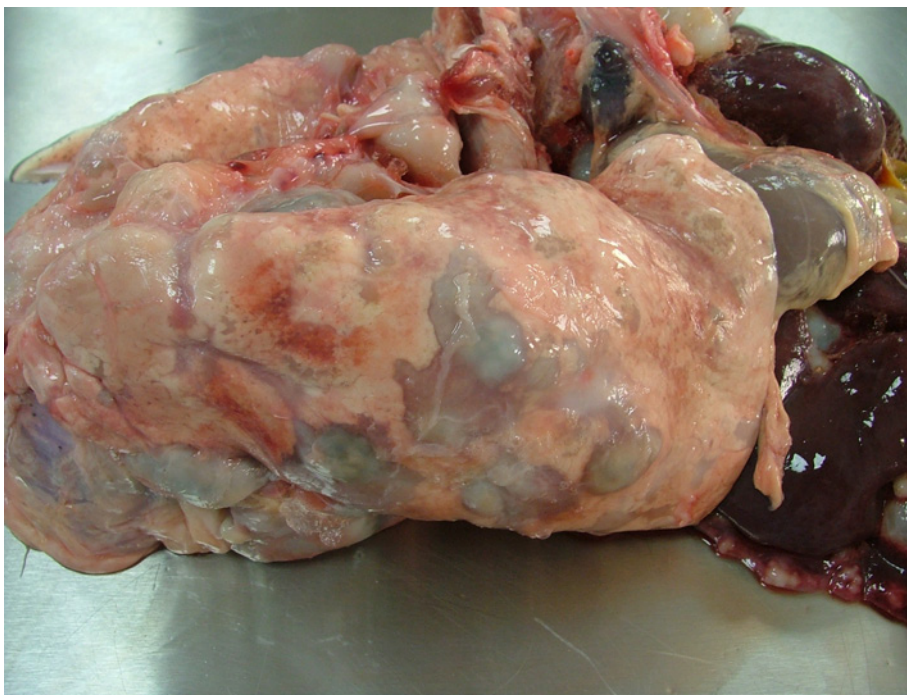
An incised hydatid cyst illustrating an endogenous 'daughter' cyst (A) and an exogenous daughter cyst (B)



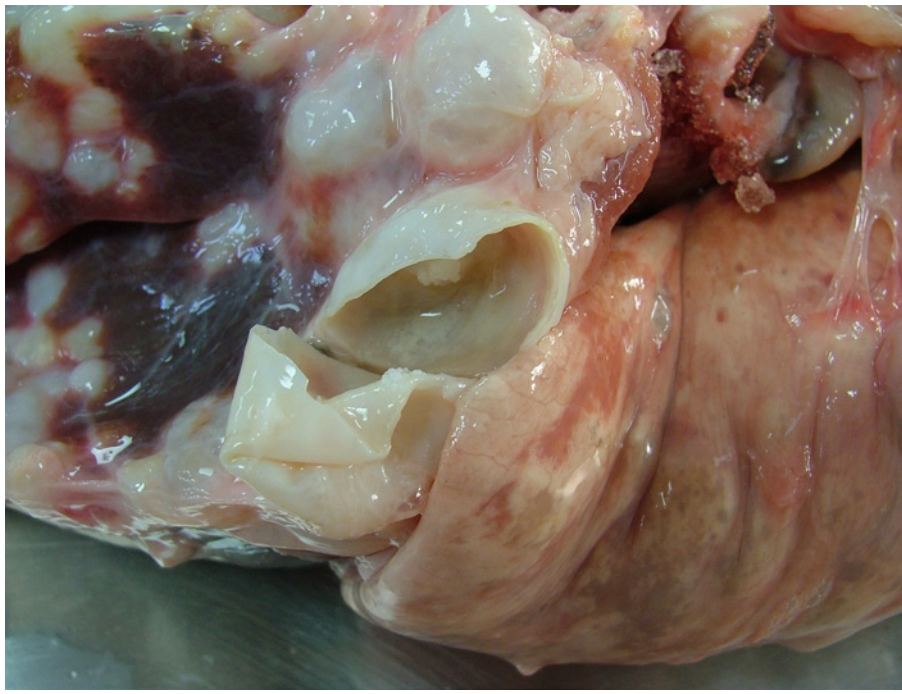
Multiple hydatid cysts in an ovine liver



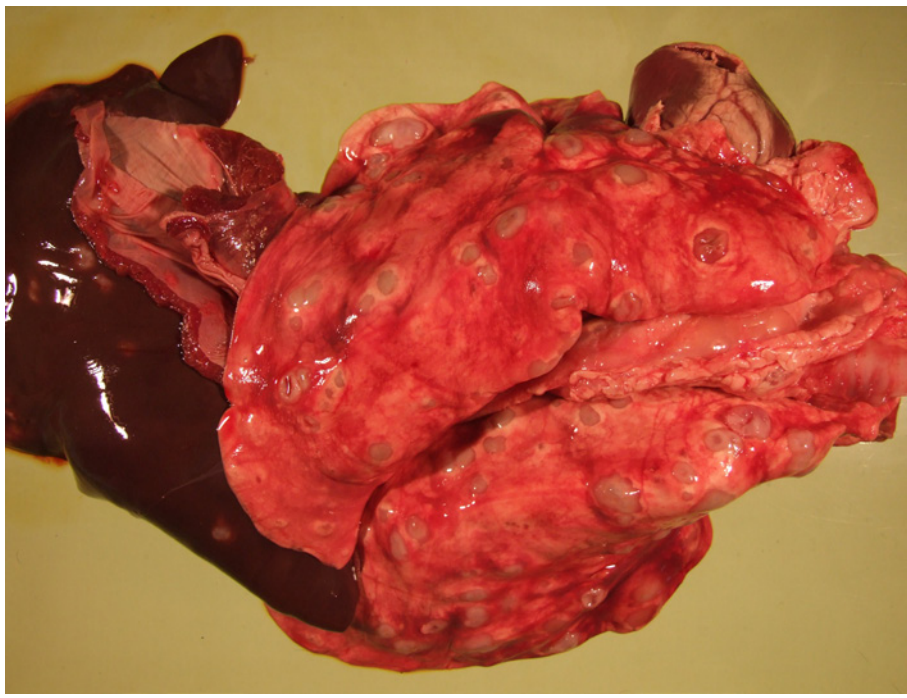
A hydatid cyst in the myocardium



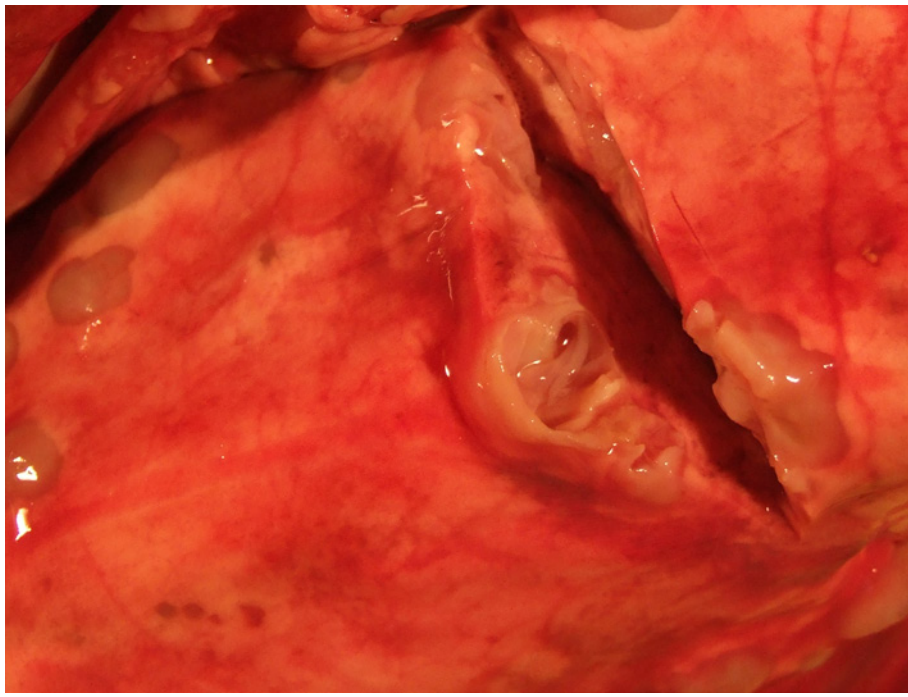
Multiple hydatid cysts within lung tissue



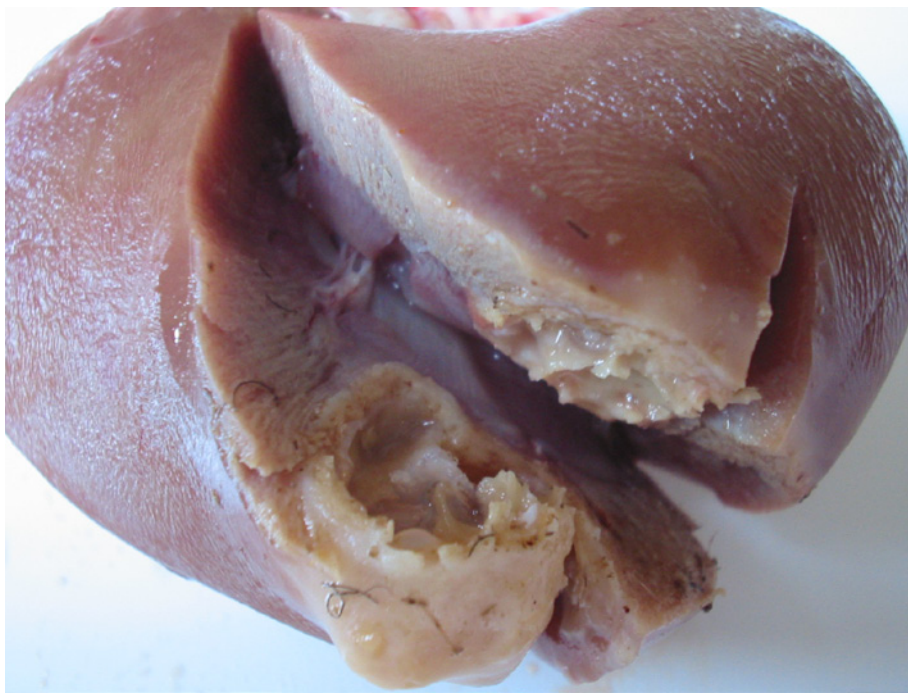
An incised hydatid cyst at the periphery of a heavily infected liver



Hydatid cysts in liver and lungs



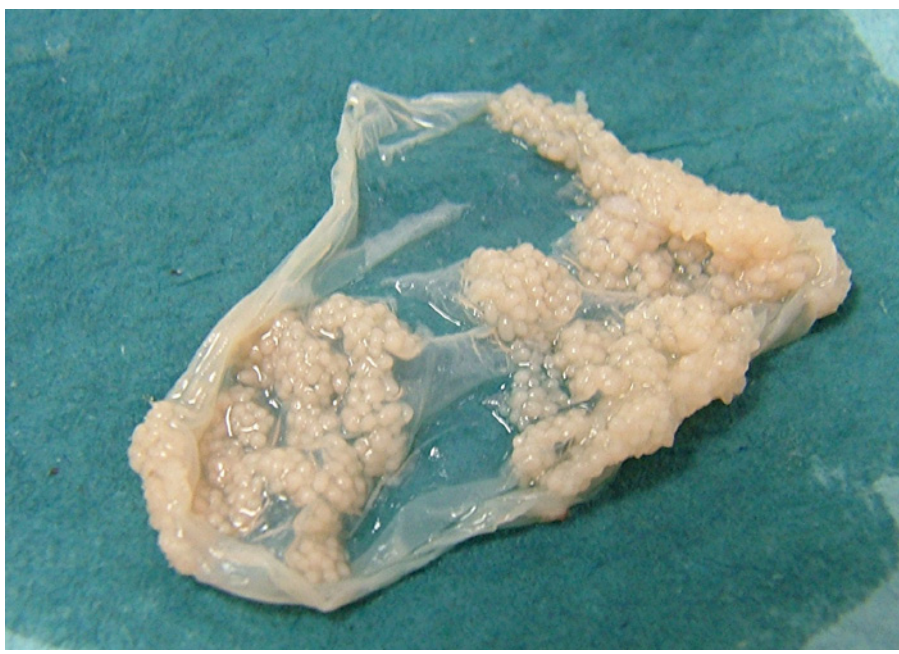
Incised pulmonary hydatid cyst



A hydatid cyst in a kidney, due to metastatic spread

COENURIOSIS

Also known as 'gid' and 'sturdy', coenuriosis is a condition of sheep caused by the presence of the migrating larvae and metacestode stage of the canine tapeworm *Taenia multiceps* in the central nervous system. In canines the adult tapeworm is 40-100cm in length and like other Taeniid worms consists of a head, neck and stroblia, the latter being formed by proglottids that bud from the neck and sexually mature as they move down the stroblia. The gravid proglottids (approximately 10mm wide by 5mm long) are shed in the faeces of the host. When the ova are ingested by a sheep they hatch releasing the hexacanth embryos that penetrate the intestinal wall, enter the blood circulation and travel to the central nervous system. The principal predilection site is brain tissue however the lumbar region of the spinal cord can also be affected. The developing larvae migrate through the nervous tissue, eventually forming a coenurus which matures after 7-8 months. The lifecycle is completed when a canine ingests material containing a coenurus or contaminated by the protoscolices of one.

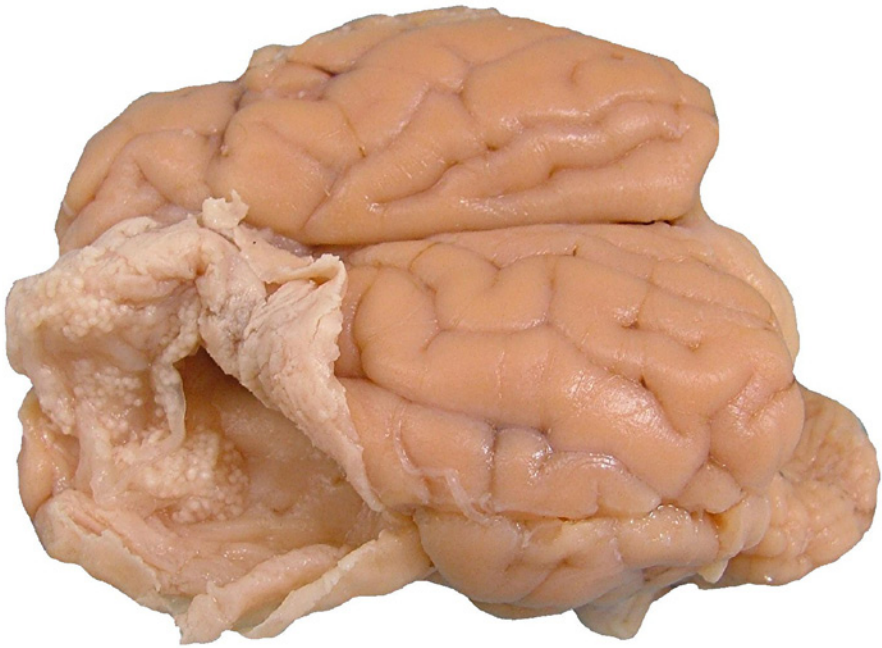


Multiple protoscolices visible in an excised coenurus

Clinically, there tend to be three phases to infection. The initial migratory stage occurs as the larvae migrate through the central nervous system creating focal haemorrhages, focal necrosis and scarring and is clinically associated with head tilting, blindness, muscular tremor and incoordination, excitability and collapse, the severity of the lesions being dependant on the number of migrating larvae and the area of the central nervous system affected.

This phase is usually followed by a period during which the animal shows signs of improvement, the so-termed 'healing phase.'

The final phase is caused by the development of the coenurus within the brain producing pressure necrosis and increased intercranial pressure. Ante-mortem signs during development and maturation of the cyst include head pressing, partial paralysis, a high stepping gait and holding the head to one side.



Damage produced by a coenurus in a lambs brain.
(Sample fixed in formal-saline)

LUNGWORM
VP, Verminous Pneumonia, Threadworms

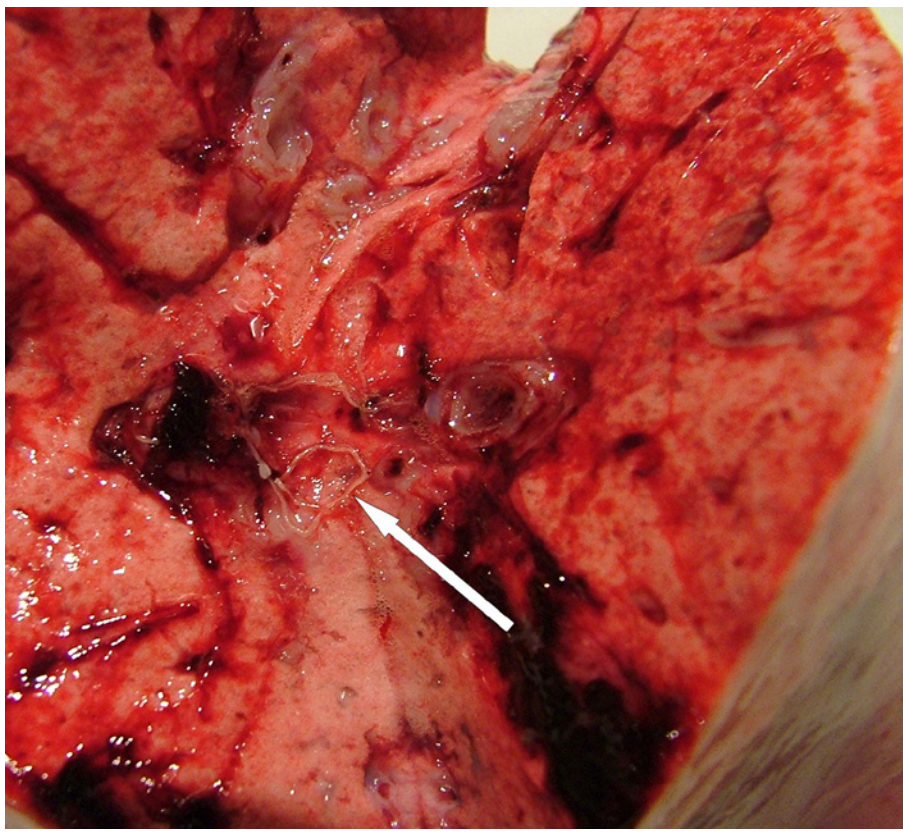
There are three lungworm of sheep normally encountered at inspection, *Dictyocaulus filaria, Muellerius capillaris* and *Protostrongylus rufescens*.

Dictyocaulus filaria and *Protostrongylus rufescens* infection produces a condition known as husk or hoose. It causes chronic coughing and unthriftyness and severe cases can lead to lung oedema and emphysema. The adult worms live in the bronchi (generally bronchioles in the case of *Protostrongylus rufescens)* where they lay eggs. These hatch and the larvae travel up the trachea, where they are swallowed and pass out with the faeces. They then move to grass where they are ingested by other sheep. The larvae then penetrate the intestinal wall, pass to the mesenteric lymph nodes where they moult.

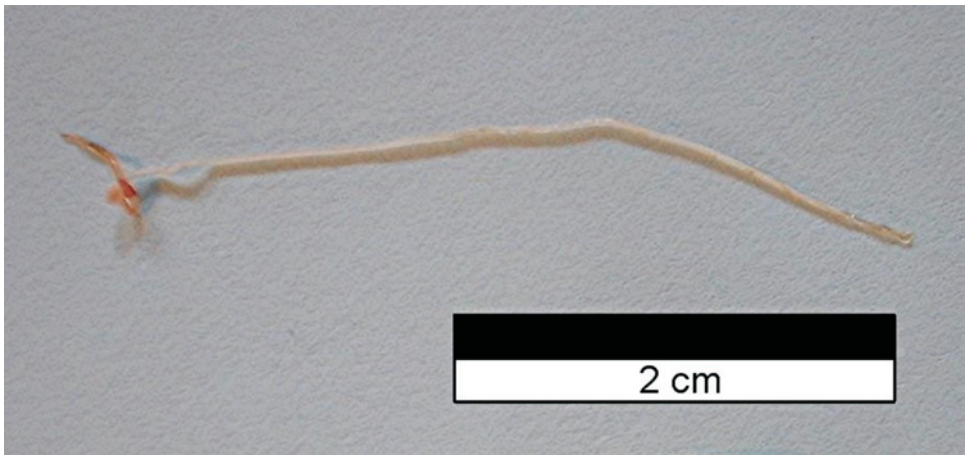
The larvae travel through the lymph ducts and blood capillaries to the lungs where they break into the alveoli (the blind ends of the bronchioles.) The final moult occurs in the bronchioles from where they move into the bronchi and mature. The presence of the adult worms in the bronchi promotes a localised inflammatory reaction and subsequent blocking of the bronchiole, leading to obstructive atelectasis and pneumonia. This inflammatory reaction is not dependent on the number of worms present.

One of the characteristic lesions of *Dictyocaulus filaria* infection in ovine lungs is pneumonia in the posterior third of the lung or at the margins of the lower lobe.

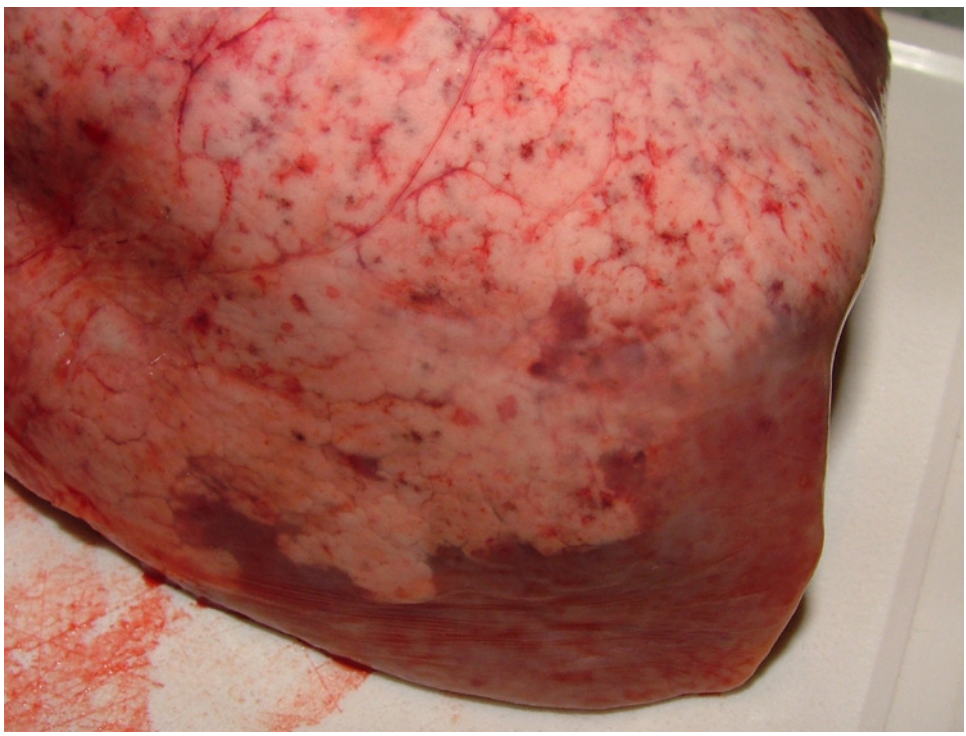
Lungs infected with *Dictyocaulus filaria* are rejected as unfit for consumption. Secondary infection by bacteria may warrant rejection of the carcase if the infection becomes systemic. An incision into the lung tissue, cutting through the bronchi reveals thread like worms in severe infestation.



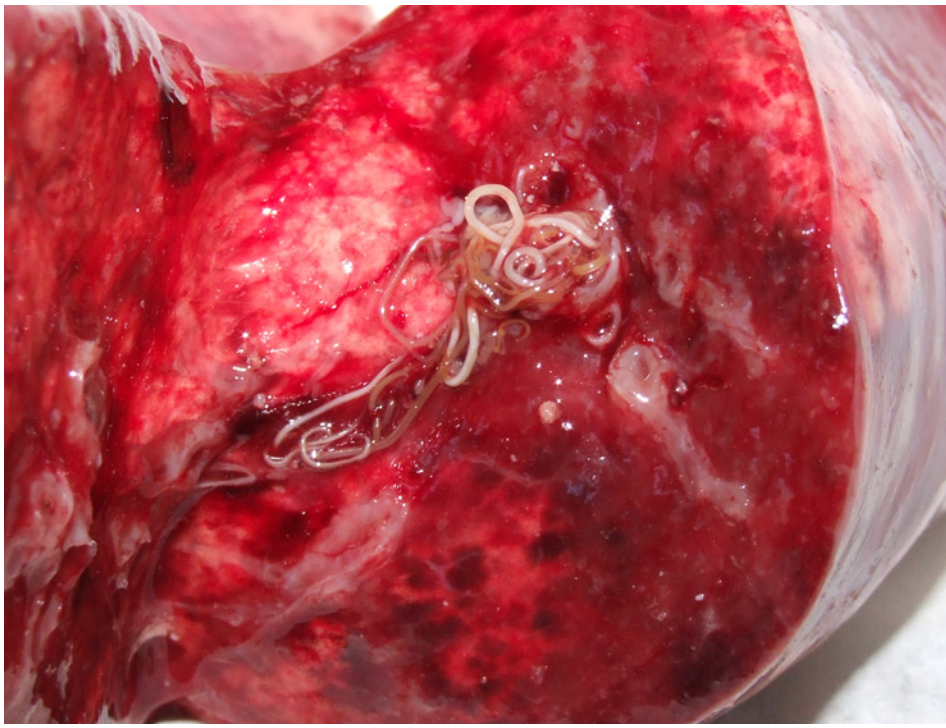
***Dictyocaulus filaria* worm (arrowed) exiting incised bronchi**



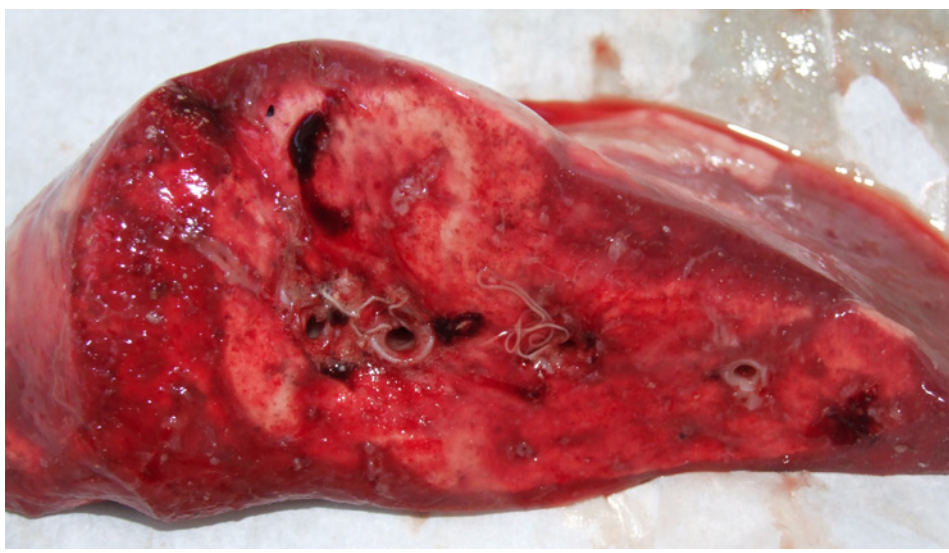
***Dictyocaulus filaria* – sheep 'threadworm'**



Pneumonic patches on margins of posterior third of a lung – obstructive atelectasis due to the presence of *D. filaria*

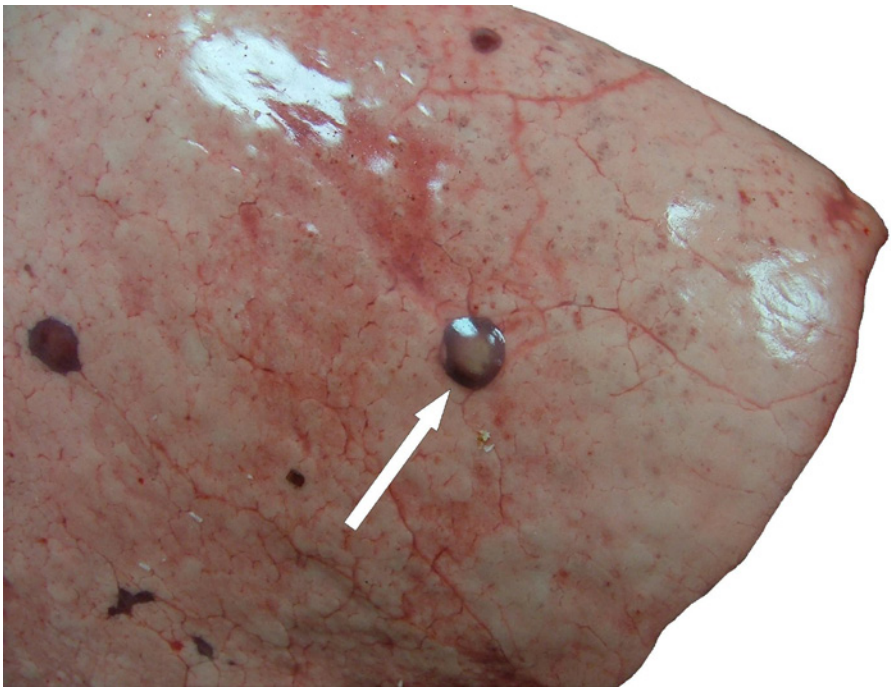


***D. filaria* exiting incised bronchi**



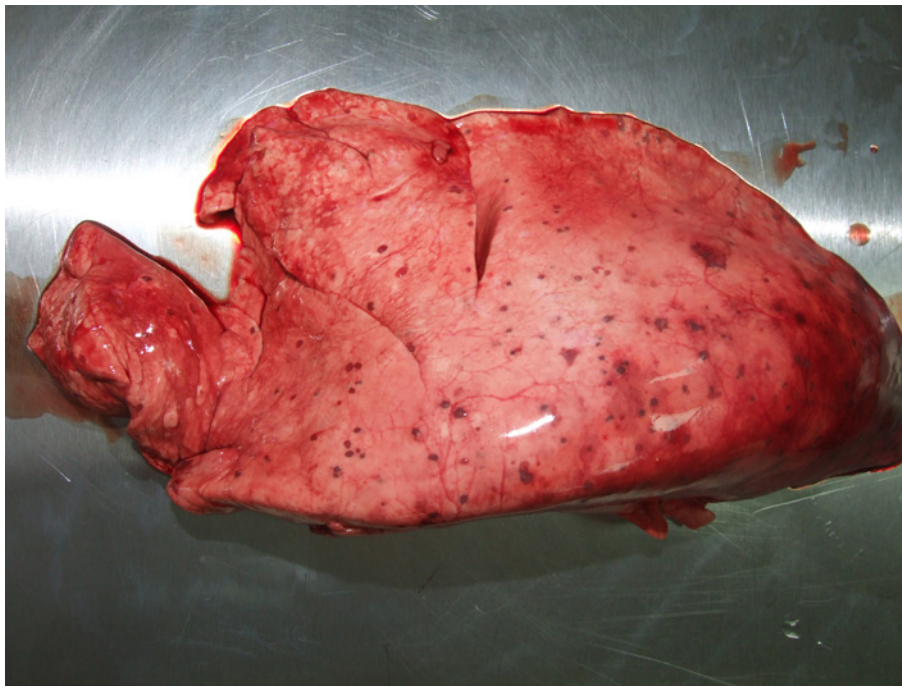
Threadworms in posterior third of lung, note obstructive atelectasis

Muellerius capillaris and *Protostrongylus rufescens* have similar life cycles to each other except that the intermediate host of *Muellerius capillaris* can be snails as well as slugs. *Muellerius capillaris* are brown hair-like worms, difficult to discern with the naked eye. *Muellerius capillaris* is associated with nodules within the lung parenchyma which are described as "lead shot'. These nodules contain several worms as well as eggs and larvae. Initial infection with *M.capillaris* occurs in the small bronchioles.

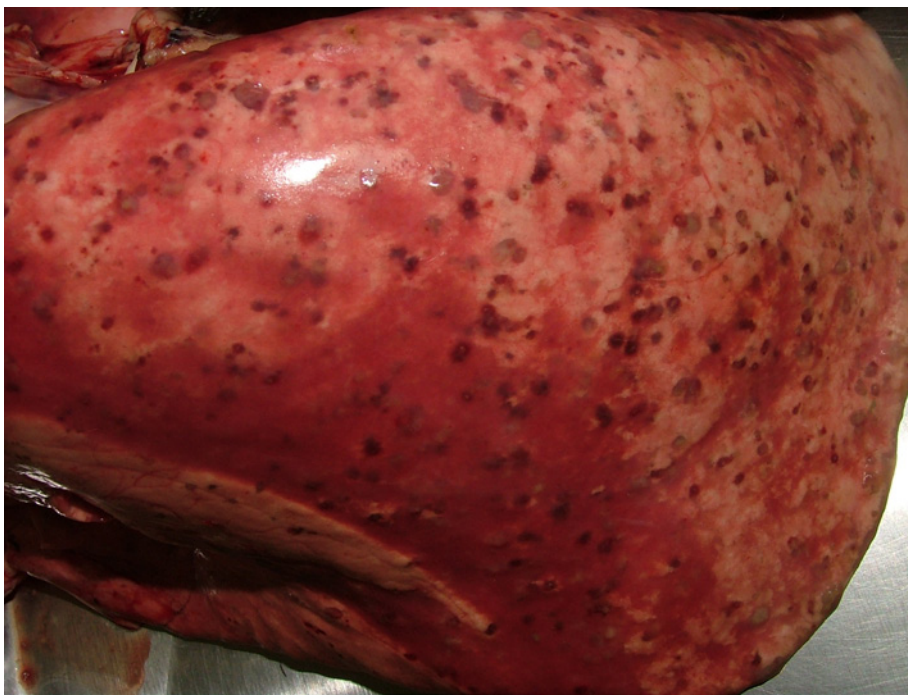


***Muellerius capillaris* – 'shot like' nodules within lung parenchyma**

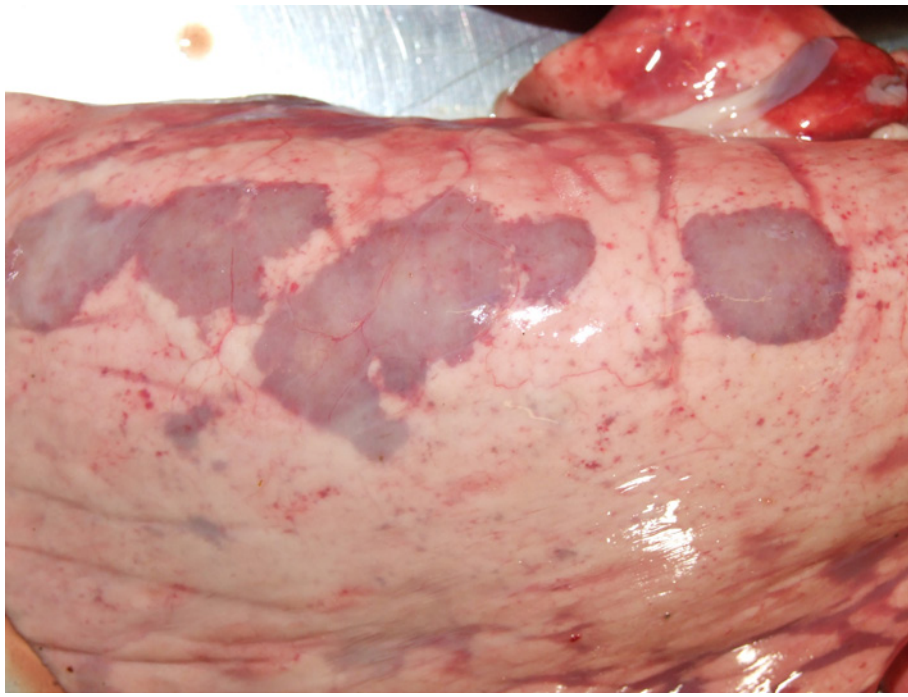
All lungs infested with lungworm are rejected as unfit for human consumption.



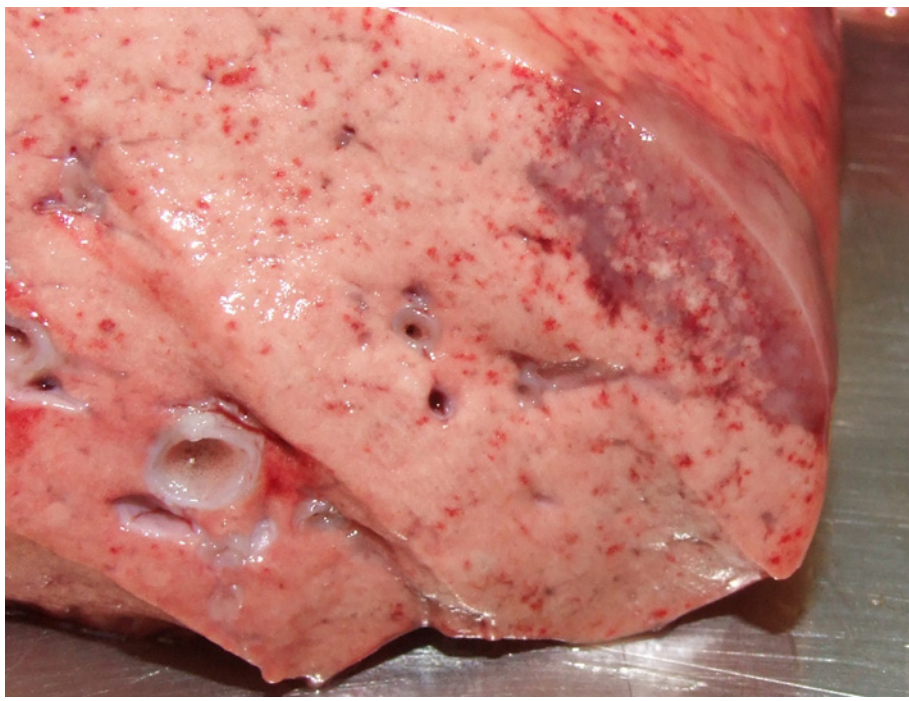
Ovine lung with multiple 'shot-like' nodules throughout parenchyma



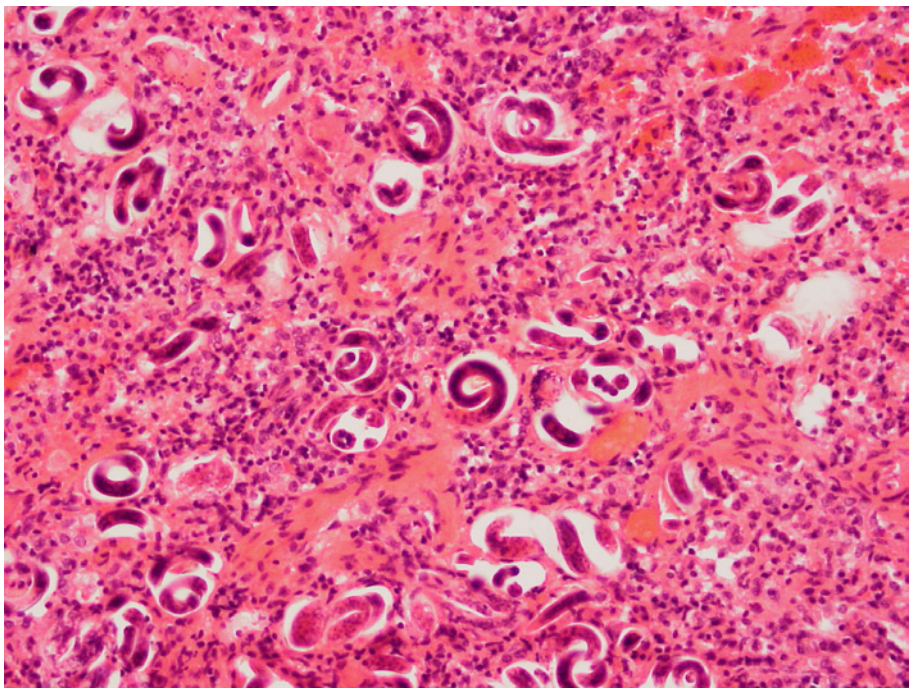
A generalised case, warranting rejection of the lungs



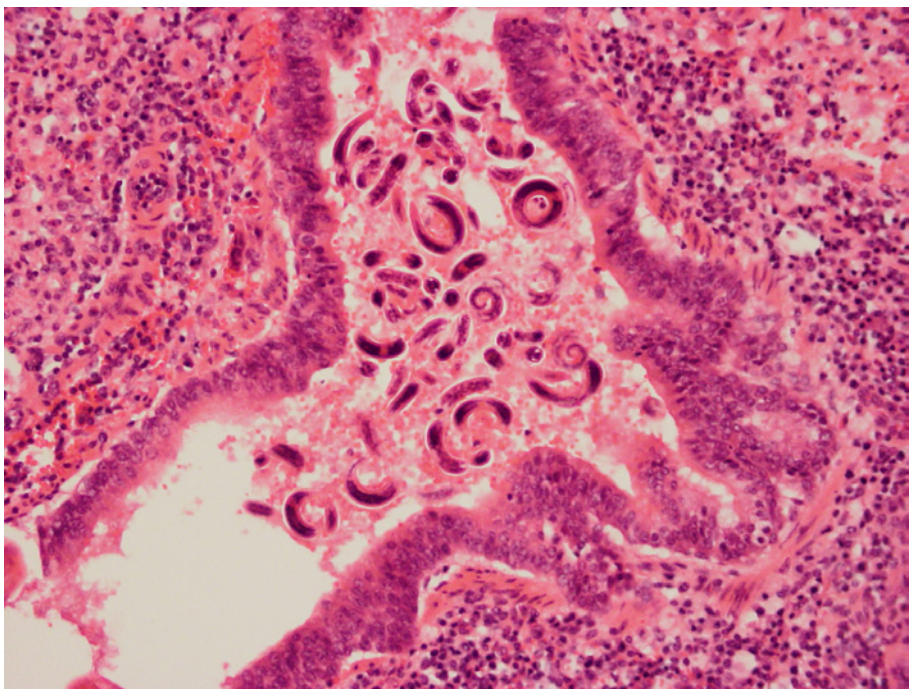
The early stage of *M. capillaris* infection, pearlescent plaques



Incision into lung tissue illustrating depth of immune response



Adult *Muellerius capillaris* in lesion from previous sample



***M. capillaris* in alveoli – note debris build up and inflammatory reaction**

MONIEZIA EXPANSA

Moniezia expansa is a tapeworm of sheep and cattle, found in the intestines. The mature adult can be up to 6 metres in length. In common with tapeworms of other species it consists of a head (scolex) from which bud proglottids that mature as they are moved from the head by the development of the following proglottid. Eggs and whole proglottids are passed in the faeces of the host infecting the pasture; the proglottids resembling cooked rice grains in the faecal matter. The triangular shaped eggs are prone to desiccation in the environment and only remain infective for approximately 24 hours.

If the infective egg is ingested by small forage mites present on the pasture, the hexacanth embryos hatch in the intestine and penetrates the internal cavity of the mite and form a cysticercus which matures after approximately 15 weeks.

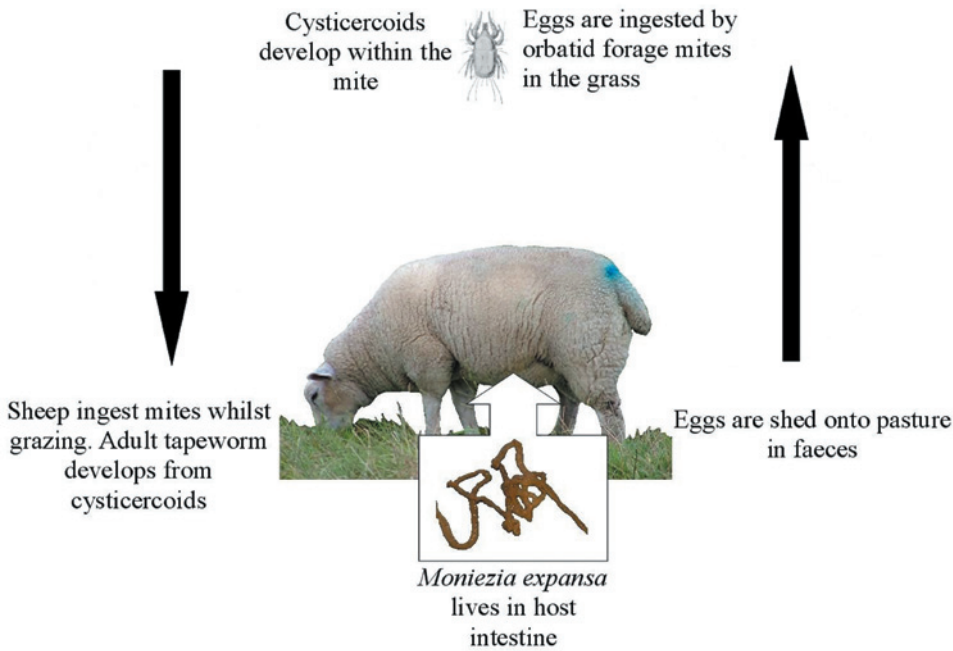
When an infected mite is ingested by a sheep during grazing the cysticercus is digested releasing the invaginated scolex which attaches to the intestinal mucosa by means of oral suckers, and matures after 40 days.

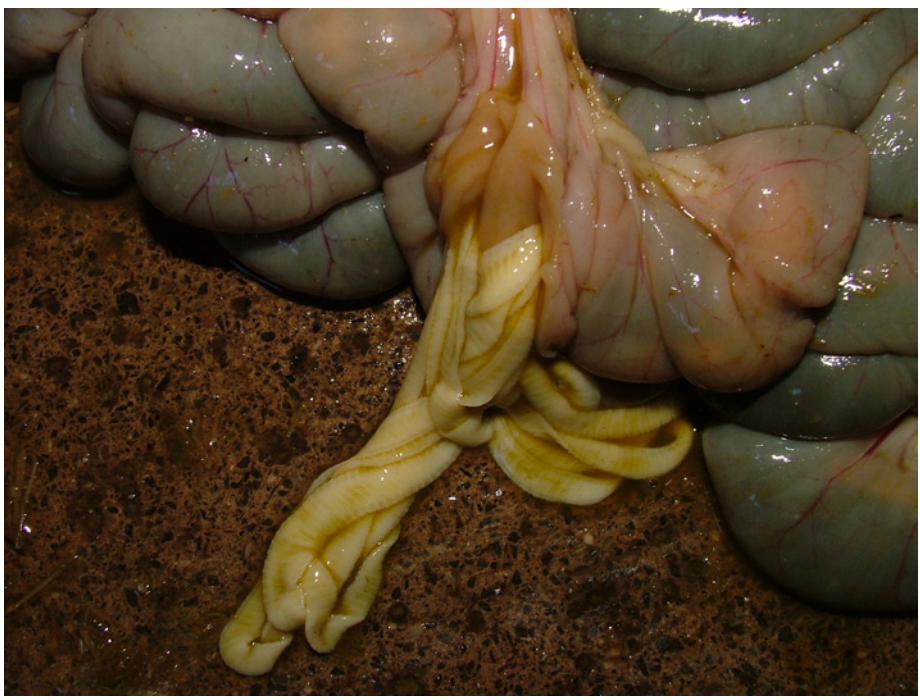
The prevalence of *Moniezia expansa* infection in sheep is attributable to the vast numbers of eggs shed and the large numbers of forage mites that can be found in pasture (some estimates suggest up to 7000 mites may be present per kilogramme of ingested grass.)

A section of an adult Moniezia expansa

The affects of a severe infestation of *Moniezia expansa* include the 'obstructive', due to the presence of large numbers of adult tapeworms within the intestine, and the 'nutritional', the worms depriving the host of food, although the presence of this parasite is considered to be only mildly pathogenic.

LIFECYCLE OF *MONIEZIA EXPANSA*





***M. expansa* exiting incised intestine**

HAEMONCHUS CONTORTUS
Barber's Pole Worm, Stomach worm, Wireworm

Haemonchus contortus is a nematode worm with a direct lifecycle and male and female forms. The adult female is larger than the male and can range from 1 to 3cm in length. The term Barber's pole refers mainly to the female which has white ovaries that spiral around the blood congested intestines.

The direct lifecycle begins with the ingestion of eggs on the pasture, which can remain infective for 6 months. On ingestion the larvae hatch and attach to the abomasal wall, and mature. Eggs are passed in the faeces and become infective after 4-7 days in optimal conditions.

The main effectsof the presence of this parasite is anaemia, which can cause the death of otherwise healthy animals. The final stage larvae and adults feed on blood accessed by puncturing vessels within the abomasal mucosa; chronic infestations can produce sub-mandibular oedema, 'bottle jaw'.

If associated with anaemia, emaciation and oedema, the carcase should be considered unfit for human consumption. Milder infestations warrant rejection of the abomasum.

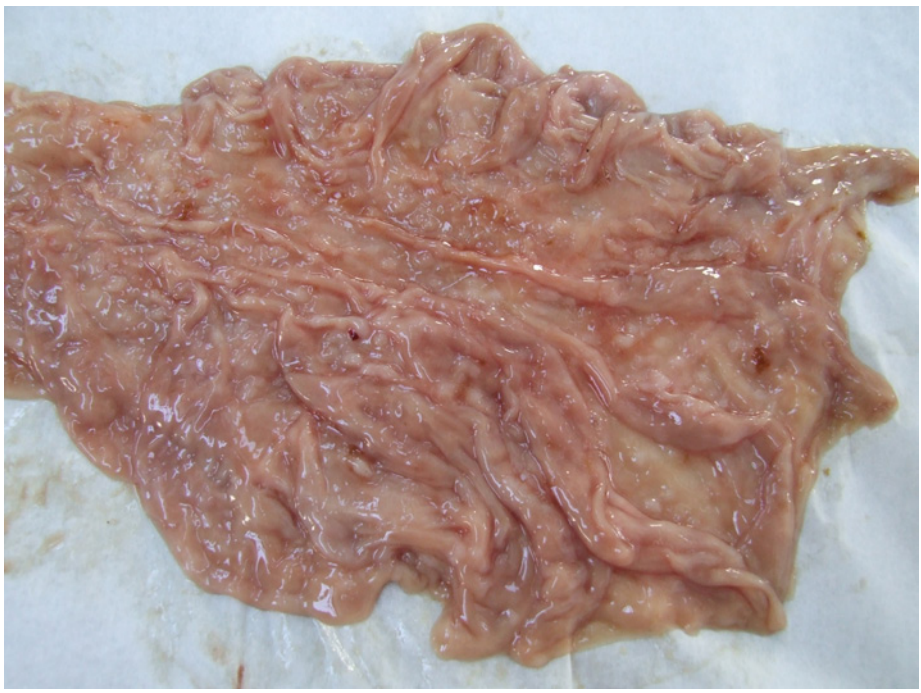
OSTERTAGIOSIS
Parasitic Gastroenteritis. Brown Stomach Worms

Ostertagiosis (also known as parasitic gastritis) is caused by the nematode *Ostertagia circumcinta*, a reddish brown worm, up to 1cm in length as an adult that affects the abomasum in sheep. *O. circumcinta* is part of the same superfamily of nematodes as *Dictyocaulus viviparus* that causes parasitic bronchitis.

Ostertagia circumcinta has a direct lifecycle. Eggs are passed in the faeces and mature in the faecal matter, the third stage larvae migrate to the surrounding soil and grass within two weeks under optimum conditions of temperature and moisture. Infection is then acquired by ingestion. The larvae then enters the gastric glands of the abomasum and moults a further two times before emerging and maturing on the surface of the abomasum. This double moult can occur approximately three weeks after infection but can also be arrested for up to 6 months depending on the time of year and environmental conditions that prevail on infection. Clinical ostertagiosis is usually manifested in two forms, Type I that effects lambs during their first year on pasture and a more serious Type II form in yearlings due to the eruption of immature worms that have had arrested development from the previous year.

The effects of a heavy infestation can be severe. The kidnap and destruction of large numbers of gastric glands and the pressure the growing larvae exert on

neighbouring acid- producing cells dramatically disrupts the ability of the abomasum to produce gastric juice. This cell to cell disruption can lead to an abnormal increase in the formation of new cells (hyperplasia) of the gastric surface, oedema, and excess blood (hyperaemia) in the abomasal folds, and eventually sloughing off of an almost diphtheric mucous membrane. The emergence of the immature worms is accompanied by the formation of characteristic nodules on surface of the mucosa.



Nodules on the internal surface of the abomasum, characteristic of ostertagiosis

In conjunction with the presence of adult worms on the surface of the abomasum the effects on the animal are those initially associated with failure of the digestive system including an inability to deaminate proteins due to the loss of acidity of the gastric juice, this loss also inhibiting the antimicrobial properties of the abomasum.

The damage to the epithelium of the abomasum also permits larger molecules to move directly from the abomasum into the circulation. This reduction in the efficiency of the digestive system can lead to a 10% reduction in feed conversion with the mild Type I form.

In the live animal a slight Type I infection will reduce the expected weight gains, in heavy or Type II infestations there is a reluctance to eat, profuse green diarrhoea, submandibular oedema and marked weight loss.

PROTOZOA

Protozoa are single-celled organisms that, unlike bacteria, possess a nucleus and other organelles that enable them to lead an independent existence and include Eimeria, Sarcocystis, Babesiosis and Toxoplasma species. Protozoa are mobile organisms, using methods of propulsion such as flagella, cilia and undulating membranes. They feed by enveloping particles and digesting them, followed by the extrusion of waste material from the cell.

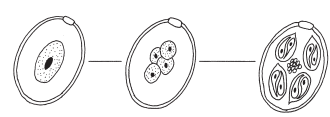
COCCIDIOSIS

Coccidiosis is a disease condition caused by the actions of host specific coccidian protozoan parasites of the Eimeria species, including *Eimeria arloingi*, *E. crandallis* and *E.ovinoidalis*

Eimeria is an intercellular parasite of the cells that line the internal surface of the intestines (epithelial cells) and lacteal channels and cause lesions by destroying the cells as part of their lifecycle. The severity of the disease is dependant on the number of infective agents ingested, but an infected individual can pass several hundred million of these agents in faeces during the course of the disease.

LIFECYCLE

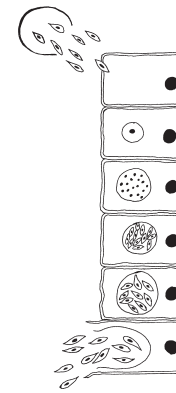
There are three basic stages in the lifecycle of *Eimeria* species, **Sporulation** occurring outside the host, **Infection** and **Shizogony**, and **Gametogony** and oocyst formation.



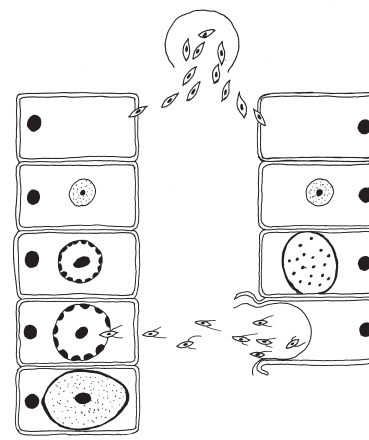
Sporulation – eggs, or oocysts (resistant shell surrounding protoplasm and a nucleus) are passed in the faeces. Under optimum conditions, within 2-4 days the nucleus divides to produce four sporoblasts, each containing two sporozoites (the infective agent). The sporoblasts secrete a covering wall to form sporocysts. This make up, four sporocysts containing eight sporozoites, is indicative of *Eimeria* species and is used as a diagnostic method.

Infection and Shizogony. (Asexual reproduction.) – When ingested by the host, either through eating vector organisms such as earthworms that have ingested oocysts, or by ingestion of oocysts during feeding, the sporocysts are liberated from the oocyst by the digestive processes of the host animal. The sporozoites are activated by the

presence of bile and trypsin in the small intestine and leave the sporocyst, and are now known as trophozoites. The trophozoites enter epithelial cells in the intestine and forms schizonts, in which division of the trophozoites nucleus forms merozoites. When the shizont containing merozoites is mature, the shizont and host cell rupture, allowing the merozoites to enter other epithelial cells where the process is repeated.



Gametogony (Sexual reproduction.)- Eventually the merozoites enter a host cell and develop into either a male form (microgametocyte) containing microgametes (small, actively mobile, flagellated organisms, similar in function to sperm), or develop into the female form, the macrogametocyte, which are single celled and expand to fill the host cell. When the microgametocyte is mature, it ruptures, together with the host cell and releases the microgametes. One microgamete then enters the host cell containing the macrogametocyte, penetrates the latter, and fusion of the two nuclei takes place. A cyst wall then develops forming a zygote that is later released as an unsporulated oocyst in the faeces when the host cell rupture



EFFECTS ON THE HOST

Eimeria species are host-specific, which is to say that a species that affects cattle will not infect sheep and vice versa. Sheep, like other animals affected by species of Eimeria can develop certain immunity to infection from exposure to previous infections However, this acquired immunity is again species specific, only relevant to the species of Eimeria encountered. The susceptibility to infection is also increased by the presence of other disease conditions that may lower the immune response.

BABESIOSIS
Haemosporidiosis, Piroplasmosis, Red Water Fever

Babesia ovis and *Babesia motasi* are intraerythrocitic protozoal parasites transmitted by the tick *Ixodes ricinus*.

The organism lays singly or in pairs in sheep red blood cells and eventually divides by binary fission and ruptures the cell, releasing the organism to infect other red blood cells. When infected blood is ingested by a tick the organisms undergo their sexual phase in the gut of the tick followed by shchizogony which in turn produces motile vermicules (worm-like). These vermicules multiply in the tissues of the tick and eventually invade the eggs to continue reproduction in the larvae when it hatches. The vermicules eventually migrate to the salivary areas of the larvae and form the infective sporozoites which are injected into the ovine host when the larvae feed.

The clinical signs of babesiosis are due to the wholesale destruction of the erythrocytes and release of haemoglobin and include jaundice, anaemia, splenomegaly and haemoglobinuria (the presence of free haemoglobin in the urine – 'red water').

The judgement in these cases is dependent on the condition of the animal and carcase, such as rejection for icterus and anaemia.

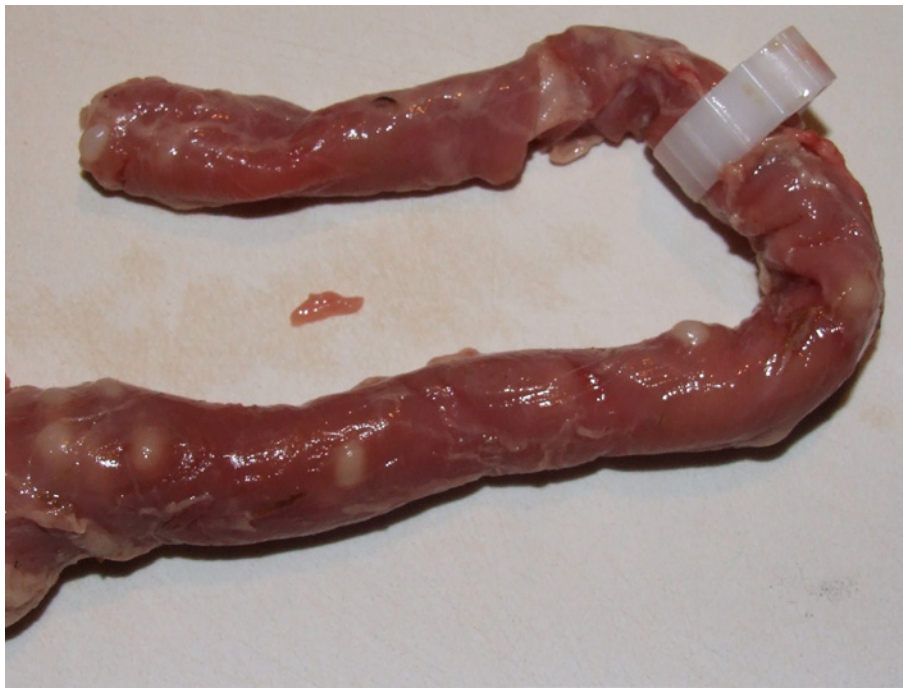
SARCOCYSTS

Sarcocysts are a sub-group of protozoa.

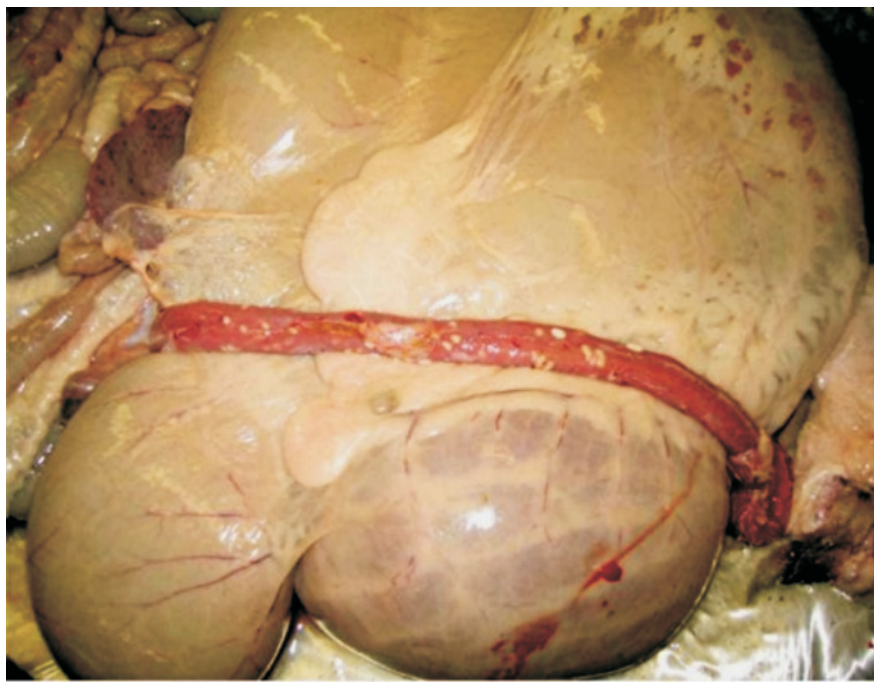
They have a two-host lifecycle, the main species found in sheep, *Sarcocystis tenella* (canine – sheep) and *Sarcocystis gigantea* (cat – sheep) being transmitted respectively in the faeces of canines and felines. Studies have shown that approximately 35% of sheepdogs and up to 75% of hunting dogs are affected, possibly due to being fed undercooked meat. Another species *Sarcocystis hominis* is transmitted in human faeces.

Sarcocysts are the cystic stage of the lifecycle, which can be discerned with the naked eye, they are found embedded in muscle. These cysts are known as Meischer's tubes, and are oval, up to 10 mm in length and are an off white/green colour. The earlier stage, sporocysts, develop in and destroy cells in the lining of blood vessels, and can cause anorexia, fever, anaemia, a reluctance of the animal to move and abortions.

The judgement depends on the severity of the infection, localised areas can be trimmed and rejected, if the infestation is generalised the entire carcase and associated offal is rejected as unfit for human consumption.



Oesophageal sarcocystosis – note rodding clip



***Sarcocystis gigantea* – Meischer's tubes similar to rice grains visible on the oesophagus**

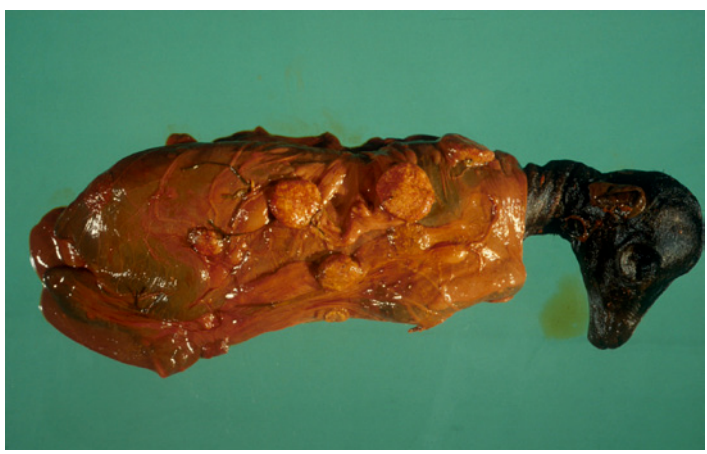
TOXOPLASMOSIS

Toxoplasma gondii is an obligate intracellular coccidian parasite of all warm blooded animals including man, the definitive host being the cat. The lifecycle of *T. gondii* has two stages, a sexual lifecycle in the definitive host and an asexual lifecycle in the intermediate host. During infection, the definitive feline host passes oocysts in faeces which become infectious (sporulate), a state that can continue for up to 1 year in a warm, moist environment. When ingested by the intermediate host the sporoblasts alter to tachyzoites (rapidly forming stages), invade cells and form vacuoles within the cytoplasm. Within this vacuole rapid binary fission of the tachyzoite takes place until the host cell ruptures releasing the parasite to invade other cells.

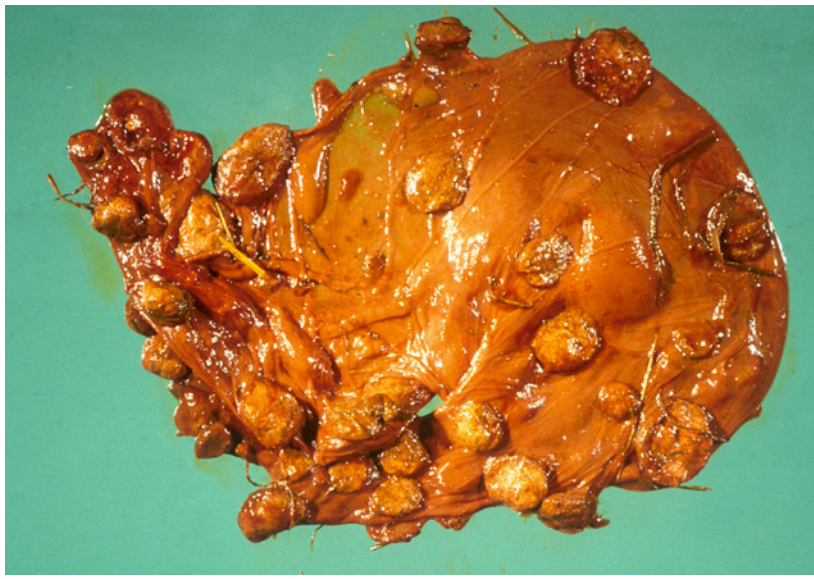
In addition to tachyzoites, a slower replication form can develop, called a bradyzoite, a more quiescent stage that can produce cysts in which the bradyzoites slowly multiply. This latter slow growing form within host cells appears not to produce an antibody response, whereas the tachyzoite form does.

When tissue containing cysts is ingested by cats, the bradyzoites revert to the tachyzoite stage, enter the epithelial cells of the intestinal mucosa and begin the sexual stage and oocyst formation in a manner similar to that of *Eimera spp*. In addition *T.gondii* can be distributed throughout the body via the blood circulation and in sheep the predilection sites include the placenta.

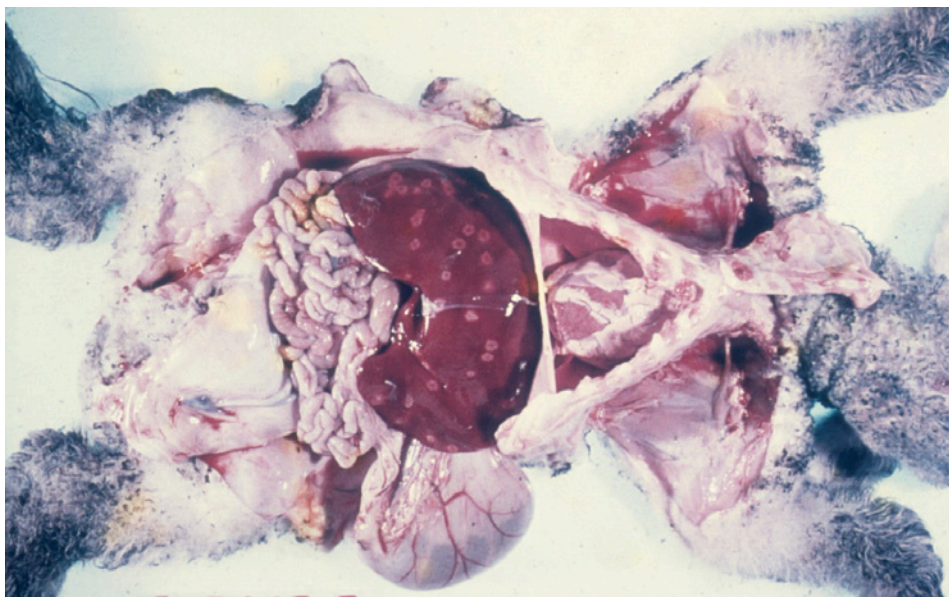
The clinical findings are of abortion and stillbirths, fever, muscular tremor and dysponea. Post mortem lesions include the retention of a mummified foetus and placentitis accompanied by necrosis, granulomatous lesions in lungs, encephalitis and associated oedema. Toxoplasmosis is a zoonotic condition, with bradyzoites forming in brain tissue of humans in addition to causing abortion. Carcase and associated offal is unfit for human consumption.



Toxoplasmosis – mummified foetus



Toxoplasmosis - Affected placenta



Toxoplasmosis – liver lesions in aborted lamb

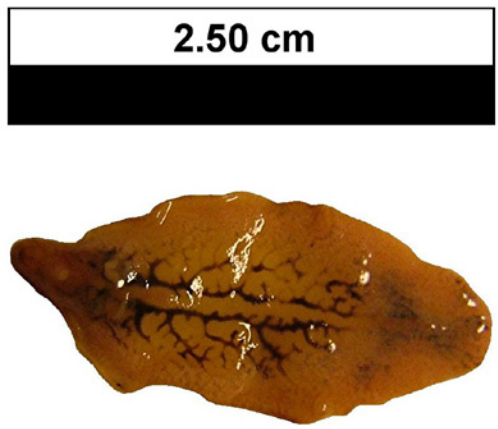
FASCIOLIASIS

Fascioliasis is the term given to the change of state of sheep due to the actions of the liver fluke, a parasitic trematode called *Fasciola hepatica*.

Fasciola hepatica has a complicated life cycle, involving the mud snail *Lymnaea truncatula*. Sheep pastured in damp fields between May to June and August to October being particularly susceptible as this is when the temperature, conditions, and snail population, favour the parasite.

Fascioliasis may be acute, sub- acute or chronic.

Acute fascioliasis occurs 2-6 weeks after the sheep ingests huge numbers of metacercaria (2000+), and their subsequent migration through the liver causes massive haemorrhage and damage to the liver. Acute fascioliasis can cause sudden deaths in a flock during autumn and early winter.



An adult *Fasciola hepatica*.

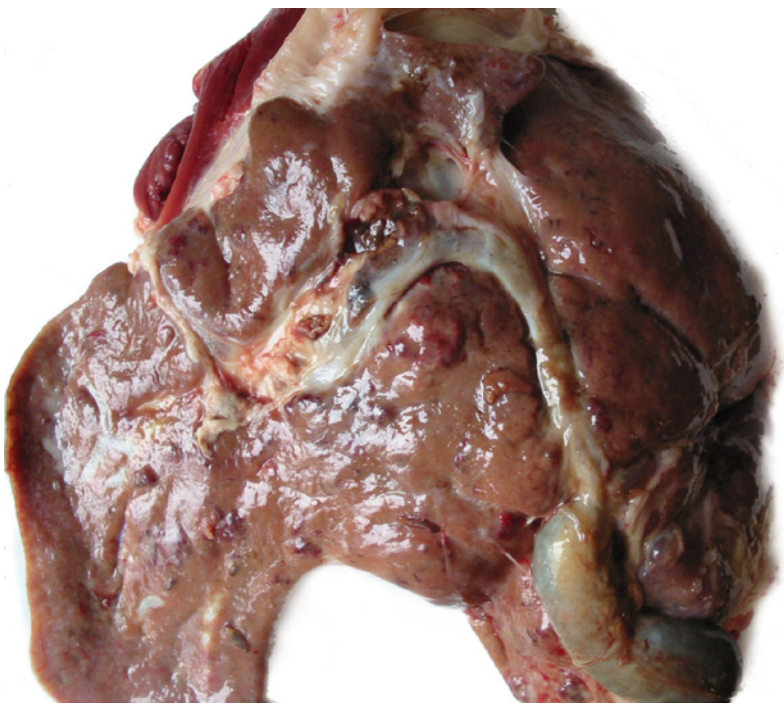
Sub acute fascioliasis occurs, again, in Autumn / early winter, when metacercaria are ingested over a longer period and the immature fluke are at varying stages of development, some have reached the bile ducts and are causing inflammation, others are still migrating through the liver substance causing haemorrhages. It is not so rapidly fatal as the acute form, sheep show signs of loss of condition, and occasionally the presence of fluid around the jaw and head, ('bottle jaw'), and can display these conditions for 1-2 weeks before death.

Chronic fascioliasis occurs normally in early spring, 4-5 months after 200-500 metacercaria are ingested. This form is the most common. The sheep suffer loss of condition, emaciation due to lack of appetite and the effect of the fluke on the

metabolism of food. Each fluke can cause the leakage of 0.5ml of blood into the bile ducts per day, as well as plasma proteins. The liver is fibrous and the bile ducts inflamed (cholangitis.) This thickening and subsequent calcification of bile ducts eventually produces an inhospitable environment for the fluke.

Livers affected by adult fluke, or the migrations of immature fluke are rejected as unfit for human consumption. The presence of fluke can also lower the animal's resistance to other disease, which could warrant the rejection of the entire carcase as well as reducing the yield of that carcase.

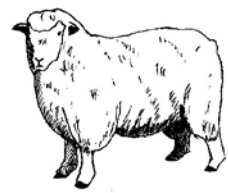
The presence of fluke can lead to cirrhosis and subsequent anaerobic conditions that favour the development of conditions such as Blacks disease. Fluke can also act as transport vectors for other bacterial agents such as *Fusobacterium necrophorum* and *A.pyogenes* leading to hepatic abscessation.



Thickening of the bile ducts (cholangitis), the so called 'pipey liver' is a characteristic indication of the presence of fluke.

Life cycle of the liver fluke *Fasciola hepatica.*

Minimum 17-18 weeks at optimum temperatures.



Mature liver fluke live in the bile ducts of the liver, digesting secretions caused by their presence. Eggs pass through the bile ducts to the intestines.

Egg.
Egg passes in faeces, develops and hatch releasing miracidia. Takes 9 days at temp between 22-26 degrees centigrade.

Development on grass and in sheep.

Miracidium.
Mobile larva which must find a suitable snail within three hours as it has a short lifespan. It penetrates the snail using enzymes.

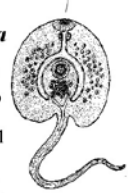
Development in mud snail *Lymnaea truncatula*

(Intermediate host)

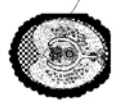
Sporocyst.
Approximately 30 minutes after entering the snail the miracidium develops into a sporocyst in which the larval redia are produced.

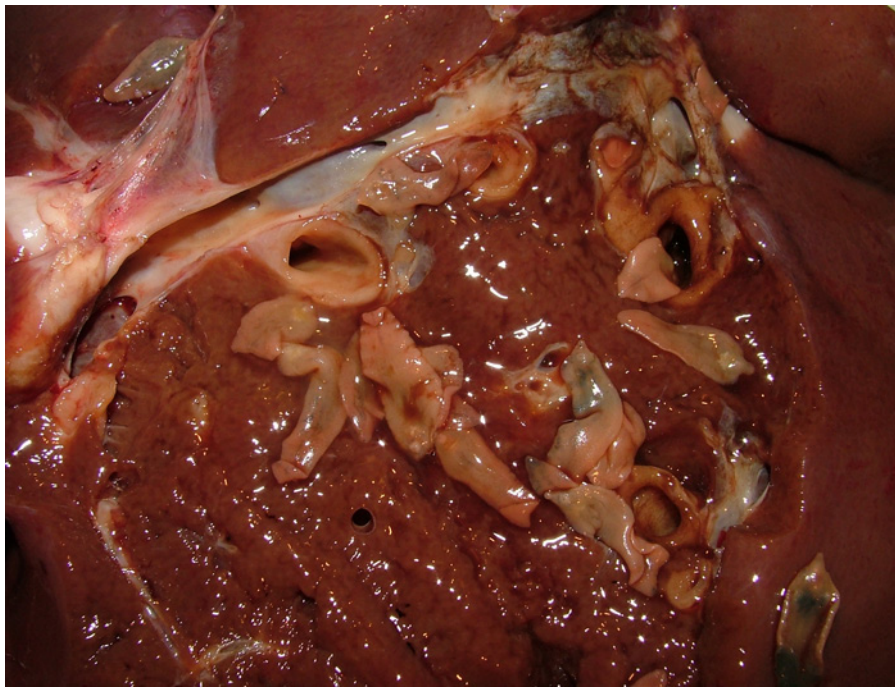
Redia.
This larval stage leaves the sporocyst and slowly migrates through the tissues of the snail to reach the digestive gland. Here the cercaria are formed within the redia.

Cercaria
When mature, the cercaria pass out of the redia, migrate to the pulmonary chamber of the snail and then to the outside. These then swim through water film to firm surfaces such as grass and encyst becoming an infective metacercaria.

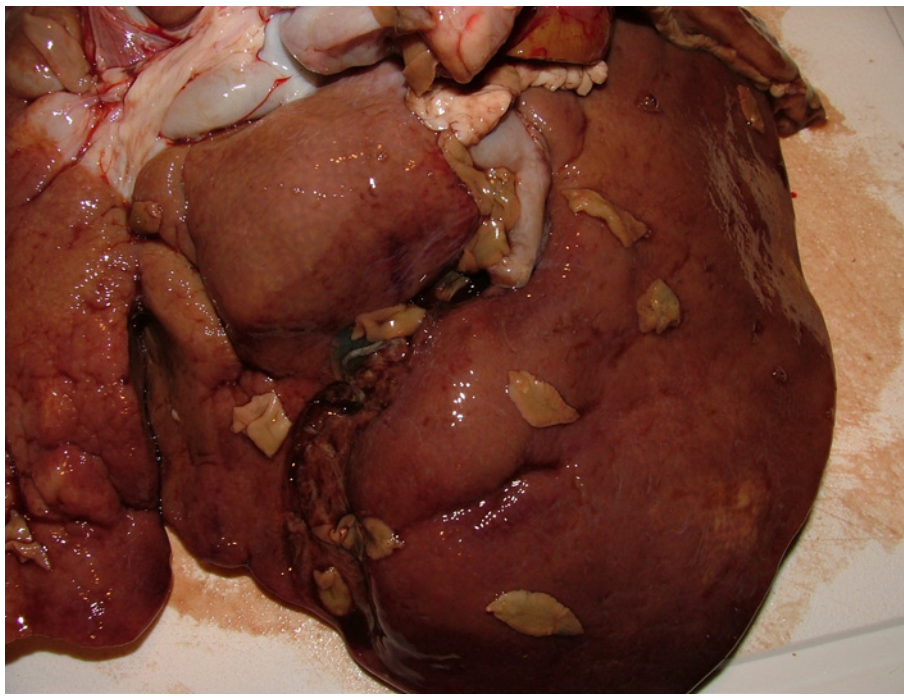


Metacercaria.
When eaten by a sheep, the cyst walls are digested, the young flukes which emerge bore through the walls of the gut and enter the body cavity. After 1-2 days they bore through the liver surface and feed on liver cells, finally entering the bile ducts and grow to maturity.

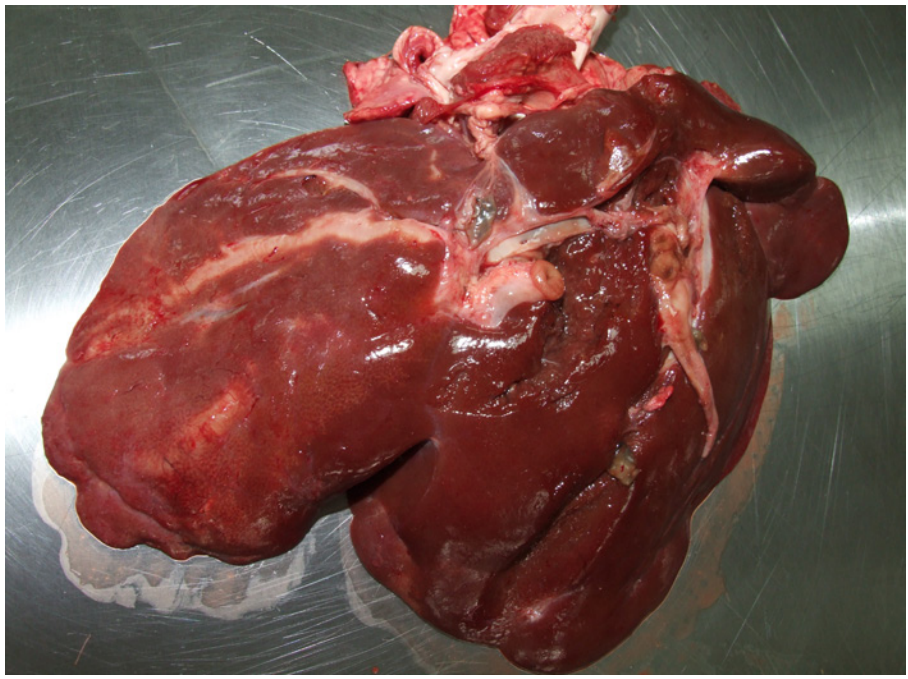




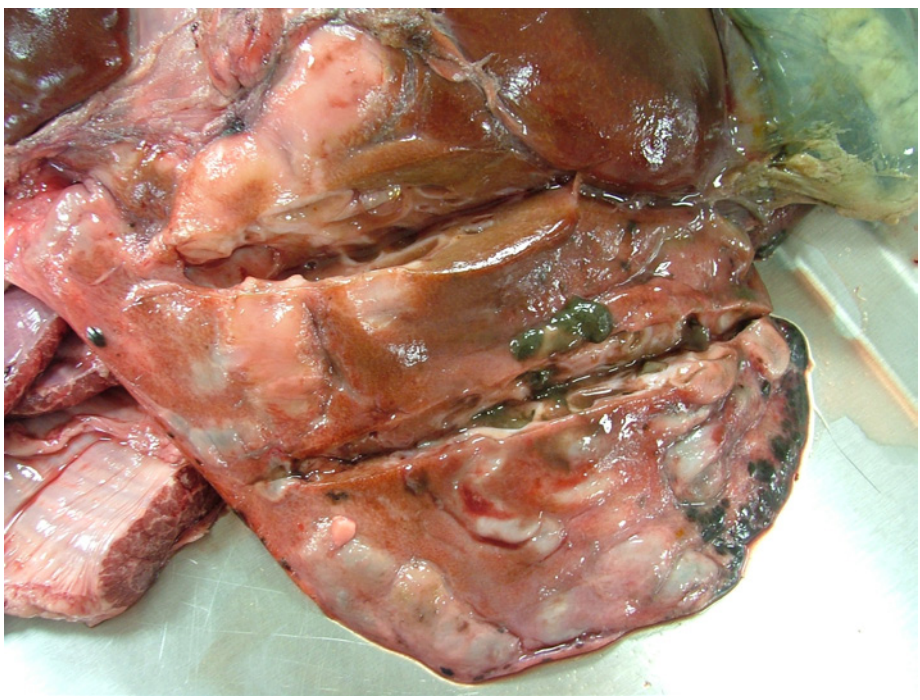
Adult *Fasciola hepatica* emerging from thickened bile ducts



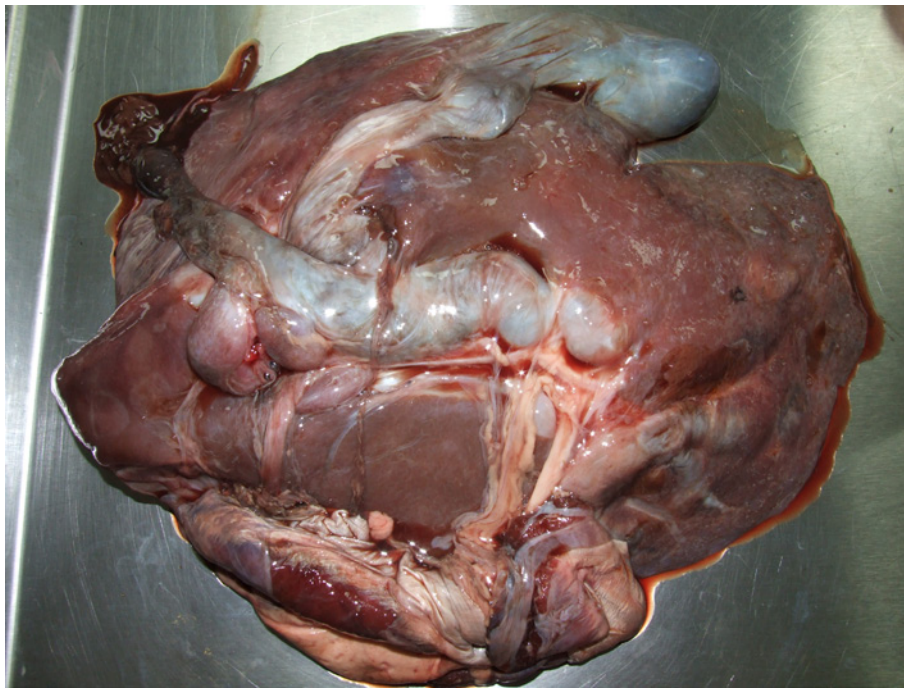
Removal of the gall bladder allowing more *F. hepatica* egress



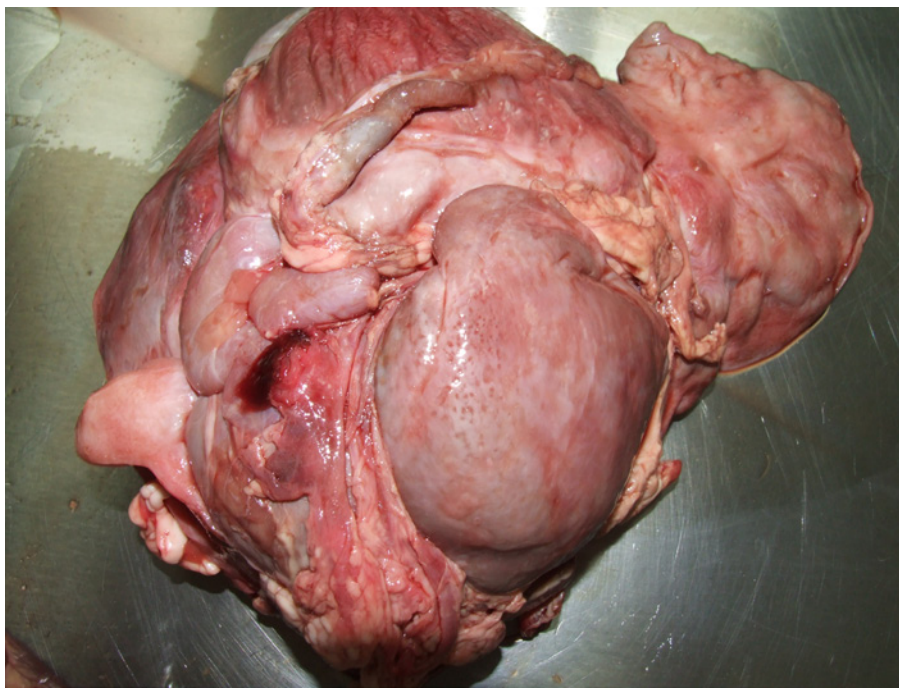
Readily discernable bile ducts due to fasciola cholangitis



Cirrhosis at the periphery of the hepatic lobe and degenerating fluke

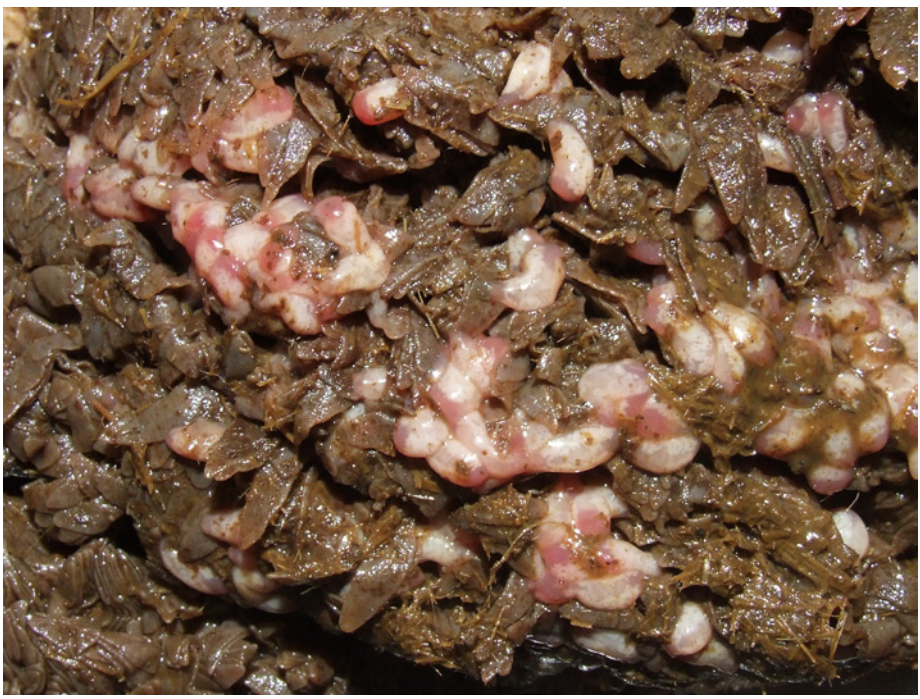


Above and below: Distortion of the liver due to massive fluke infestation. The hepatitis that results from the larval migration is so acute that the liver expands quicker than hepatic parietal peritoneum leading to bossellation and deformation



PARAMPHISTOMES
Rumen fluke, Cone fluke, Paramphistomosis

Paramphistomum spp are trematode parasites of ruminants. The most common form encountered is *P.cervi* in the rumen and reticulum. Unlike the leaf shape usually associated with trematode flukes the adult *P.cervi* are between 0.5 and 1cm in length and have a conical, maggot like appearance with a visible sucker at their anterior and posterior, the name Paramphistomum literally meaning 'two sided mouth'.



Adult *Paramphistomum cervi* in rumen

The lifecycle is similar to *Fasciola hepatica*, however the intermediate hosts are water snails (*Planorbis* and *Bilinus*), and tends to be associated with herbage on land that has flooded. On ingestion by the ruminant, excystment of the metacercariae and development of the larvae occurs in the duodenum (occasionally the proximal jejunum) where they attach to the intestinal mucosa and feed for approximately 5-6 weeks. The larvae then migrate to the forestomachs where they mature whilst attached to the ruminal/reticular epithelium on which they feed. Occasionally adult fluke may be found in the oesophagus. Sheep appear to tolerate large numbers of adult flukes in the forestomachs. Clinical disease tends to be due to the attachment of the larval stage to the duodenum and results in diarrhoea, anorexia and thirst.

Reject affected parts.

ECTOPARASITES

PARASITE TYPES

The external parasites (ectoparasites) of sheep are classed into two main groups; insects and arachnids. Insects, (flies, mosquitoes, keds and lice) posses three pairs of legs, a head, thorax and abdomen, arachnids (ticks and lice) possess four pairs of legs, a head fused with the thorax, an abdomen and no wings or antennae.

EFFECTS ON HOST

The effects of ectoparasites on the host can be dramatic, ranging from annoyance and fear, to skin infections, anaemia and the transmission of disease. Annoyance is caused in avoiding the parasite; flies such as *Oestrus ovis* can have an alarming affect on sheep when present, and can also be due to the intense itching and irritation the presence of feeding parasites can have. Indeed a large amount of the damage to skin in cases of mange is not solely due to the mite, it can be due to the sheep rubbing itself to relieve the itching. This damage to the skin can lead to secondary bacterial infection in addition to attracting flies. Keds and ticks when present in large numbers can result in anaemia, the constant drawing of blood eventually depleting the host animals iron reserves. Finally all the ectoparasites, especially multiple host feeders such as the ticks have the capability to act as vectors for disease, ticks also being implicated in the inoculation of toxin into the sheep during feeding leading to a condition known as tick paralysis.

FLY STRIKE
Blowfly myiasis

Myiasis is defined as the infestation of living animals with the larvae of dipteran flies.

Sheep with open wounds, but more especially soiled fleece (normally around the hindquarters) attract female blowflies such as greenbottles (*Lucilia sericata*), blackbottles (*Phormia terrae-novae*) and bluebottles (*Calliphora vomitoria*).

These females lay clusters of yellow/cream eggs in areas of decomposing matter. When a sheep has had eggs laid on it, it is said to be 'Fly blown'. Of these, *Lucilia sericata* tends to be the 'first strike' species.

***Lucilia sericata* – the 'greenbottle'**

Normally during the summer, the eggs hatch into larvae within 12 hours, the larvae feed on the infected area and grow into maggots within 3-10 days after moulting twice. The maggots then fall to the ground and pupate. When the eggs hatch the sheep is said to be 'fly struck'.

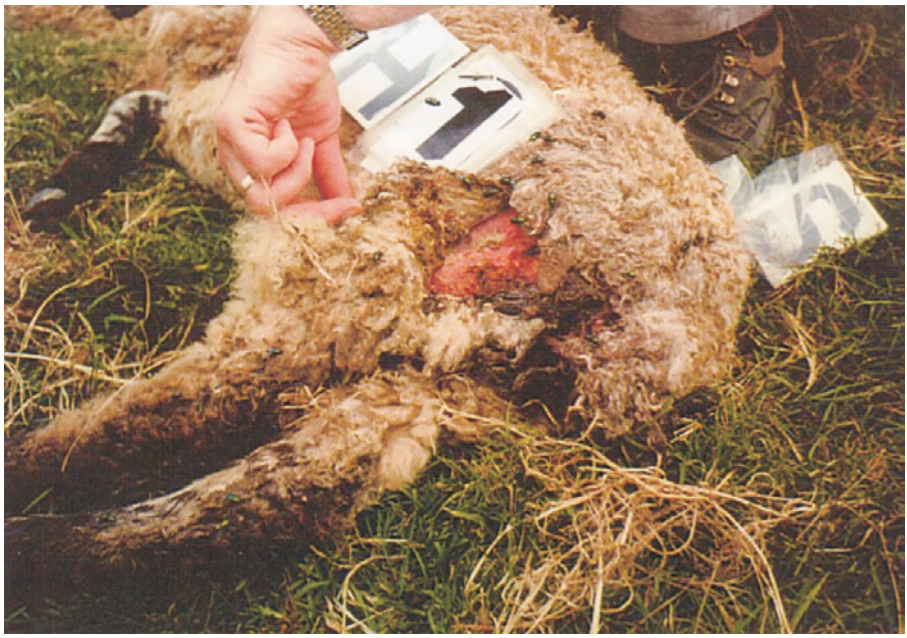
Blowfly myiasis

The larvae feed on the skin, which they lacerate with hooks on their mouthparts; then secrete enzymes, which digest and liquefy the tissues. The smell of decomposition can also attract more flies leading to secondary attacks. The wound caused by the feeding larvae can also allow infection by bacteria.

Affected sheep lose condition due to the irritation, can appear anorexic and generally stand away from the rest of the flock. In advanced cases the wool around the affected area can appear darker, moist and have a pungent odour. Where death occurs it is usually due to secondary septicaemia.

There are five basic forms of strike: Breech, Body, Poll, Pizzle and Wound strike. Breech strike occurs in the areas affected by urine soiling, diarrhoea and those perianal areas with loose, wrinkled fleece. Body strike affects the areas around the shoulders. Poll strike tends to affect rams especially in head wounds and areas at the base of the horns where sweat may accumulate. Pizzle strike can occur in this area due to urine staining especially if in association with matted under fleece. Wound strike occurs when flies are attracted to fistulating abscesses or infected wounds that occur in the shearing operation for example.

Conditions that favour the incidence of fly strike include temperature, (strike usually occurring during late spring and summer,) moisture, and host susceptibility, where the odour produced by bacterial decomposition of organic matter attracts the adults. The commonest causes of this are the soiling of hindquarters due to urine or faecal contamination, (breeds with folds of skin in the rear being most affected) and injuries.



Fly blown carcase

Damaged areas are removed at the inspection point, leading to reduction in the carcase quality. Secondary infection of the area can also warrant rejection of the carcase and offal as unfit for human consumption.

A flystuck ewe, inapparent until shearing

TICKS

An *Ixodes ricinus* nymph

Ticks are blood sucking arachnid parasites with four pairs of legs. These are important parasites in terms of inspection not only due to the blood loss a major infestation can cause, but because they represent a transport vector for various diseases including Lyme's Disease and Babesiosis, as well as biting humans.

Although there are various types of tick in this country, the genera *Ixodes ricinus* is the most important in terms of disease. *Ixodes ricinus* is a hard tick, known as the 'castor bean tick', a three-host tick, and a temporary parasite, each stage of the development taking place on a different host. The females are larger than the males and can reach a length of 1cm.

Each tick feeds only three times during its three-year lifespan, once as a larva, once as a nymph and once as an adult. After mating on the host the female engorges with blood over a period of approximately a fortnight, falls from the host and lays thousands of eggs before dying. The larvae hatch and feed from a second host before again falling to the ground and moulting to the nymphal stage which feeds from a further host before dropping off and becoming an adult. In total a tick will only parasitize hosts and feed for three to four weeks during its lifespan.

Generally considered to be a parasite associated with rough herbage, infestation occurs seasonally in the British Isles, where each stage of the tick travels to the tips of plants and awaits passing animals, usually in two periods March-June and August-November where the relative humidity and temperature favours the survival of the tick.

The affects of tick infestation can be anaemia in heavy infestations but is mostly noted for secondary infection of the bite wounds by bacteria such as *Staphylococcus aureus*, also blowfly myiasis (attraction to the wounds caused by the biting), but most predominant is the transfer of rickettsial infection such as red water fever, and spirochete infection such as lymes disease.

An immature *Ixodes ricinus* tick, note piercing mouthparts that anchor the tick into position (x200)

Engorged adult (ventral side) and immature tick

SHEEP SCAB

MANGE

Mites are insects from the same class as spiders and ticks. There are both parasitic and non-parasitic forms with the parasitic forms being further subdivided into burrowing and non-burrowing mites. There are four main parasitic species of mite encountered on sheep in the UK, all of which collectively cause a condition known as mange, these being; *Sarcoptes scabei*, *Demodex ovis*, *Chorioptes bovis* and *Psoroptes ovis*. These cause the respective conditions of sarcoptic mange, demodectic mange, chorioptic mange and psoroptic mange the latter of which is known as sheep scab in ovines.

Sheep scab

PSOROPTIC MANGE
Common scabies

The non-burrowing mite *Psoroptes ovis* causes sheep scab. This mite is oval in shape, approximately 0.75mm in size. The female lays about 90 eggs in her 4-6 week lifespan. The lifecycle of *Psoroptes ovis* from egg to adult (through larval and nymphal stages) takes 10 days. *Psoroptes ovis* has piercing and chewing mouthparts that can severely damage the skin. Infection begins as a zone of inflammation with small blisters and fluid, as the lesion spreads the centre becomes dry and covered with a yellow scab while the borders, where the mites are multiplying, remain moist.

The activity of these mites causes severe itching, and patchy wool loss will be seen where the sheep will scratch against posts and other objects in an attempt to relieve this. The sheep can cease to feed where they are preoccupied with scratching, and weight loss can be seen in adults, and a reduction in weight gains of up to 20% in growing animals may occur.

At the inspection point the external lymph nodes of the carcase may be enlarged and oedematous, if the internal lymph nodes are also affected, the carcase may be rejected as unfit for human consumption, as the enlargement of these nodes can be a sign of various other diseases and the balance of the decision to reject must lean toward the consumer.

The action of the mites can also lead to the introduction of bacterial organisms that can lead to septicaemia, pyaemia and toxaemia, which also warrant rejection of the carcase and offal.

Sarcoptes scabei (head scabies) is a roundish mite approximately 0.5mm in diameter. The fertilised female tunnels through the upper layers of the epidermis feeding on the liquid from the tissues it damages. It is within these tunnels that eggs are laid which produce six legged larvae that crawl to the surface of the skin. These larvae burrow into the skin forming pockets in which they mature and leave to seek a mate.

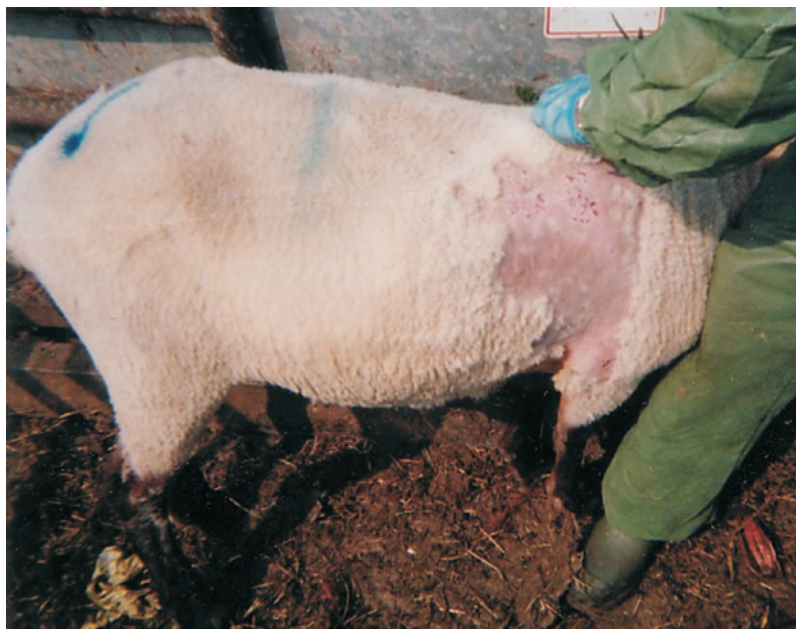
The complete lifecycle takes between 17-21 days from egg to adult. In a mild infection scaly skin is observed with little hair loss, however in a chronic infection there is a thickening of the skin, hair loss, the formation of crusts and intense pruritus.

Infection appears to be acquired by direct contact with the skin so presumably the larval stage is the most likely to be infective.

Demodex ovis (follicular mange) is a second burrowing mite although with less pathogenic properties than *Sarcoptes scabei* above. It has an elongated tapered body approximately 0.2mm in length as an adult. This mite has a predilection for sebaceous glands and hair follicles in which it completes its lifespan causing raised pea sized nodules containing caseous material and several thousand mites. It does however tend to burrow deeper into the dermis than *Sarcoptes scabei* and infection is

thought to be acquired during feeding of the lamb, limiting infection to the muzzle, head and back.

Chorioptes bovis feeds on surface debris and tends to only produce a mild flaky dermatitis near the tail in affected animals and has little pathogenic potential.



Sheep scab

ZOONOTIC POTENTIAL

Sarcoptes scabei produces scabies in humans with itching, circular elevated skin lesions in areas that have contact with the animal such as the hands and wrists. Although the mites tend not to burrow in cases where infection is derived from an animal, the condition can lead to hypersensitivity in humans once affected.

SHEEP KED

Melophagus ovinus

The sheep ked*, Melophagus ovinus*, is a wingless dipteran fly approximately 6-7mm in length as an adult. This parasite spends its entire life on the sheep, only surviving less than a week off a host. During its 100-120 day life a female ked will produce around 10 larvae which are born live and cemented to hairs of the fleece where they pupate and emerge after 20-23 days depending on conditions.

Ked infestations peak during the winter and early spring especially in housed sheep as the close quartering of the animals allows contact transfer.

The main effect of the presence of these parasites is due to their feeding action.

The ked pierces the skin with its pointed mouthparts and sucks blood. The preferred areas of feeding are the neck, breast, shoulders and flanks. Initially ked infestation is manifested by staining of the fleece with ked faeces, their presence also causes intense itching leading to rubbing and chewing of the fleece by the host.

Melophagus ovinus

Severe infestations can lead to anaemia and unthriftyness in the sheep, especially nursing lambs and pregnant ewes, and a concurrent reduction in immunity to other infections.

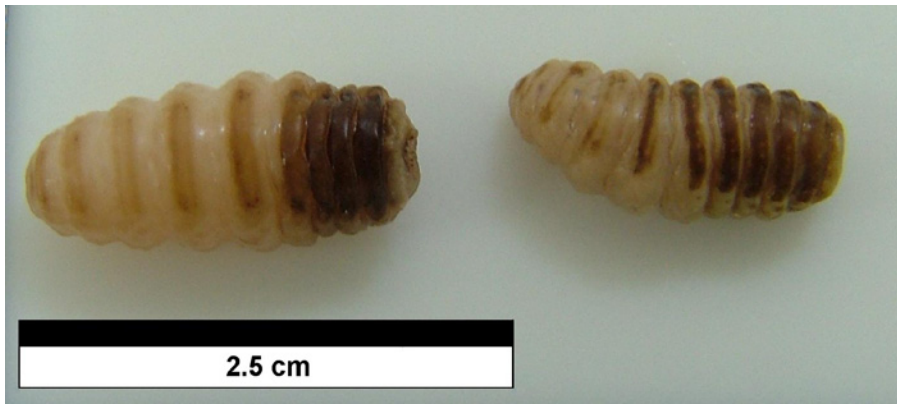
OESTRUS OVIS

Oestrus ovis, also known as the sheep nasal bot, is a fly approximately 10mm in length whose larval stage is parasitic in the nasal passages of sheep. The female adult fly is viviparus, the young hatching within the adult who then squirts fluid containing approximately 15-20 first stage larvae (L_1) at the muzzle of the animal.

The L_1 larvae, approximately 1mm in length then migrates through the nostrils to the frontal sinuses where they feed on the mucoid secretion stimulated by their presence. After the first moult the L_2 larvae crawl into the frontal passages where the second moult to the L_3 larvae occurs.

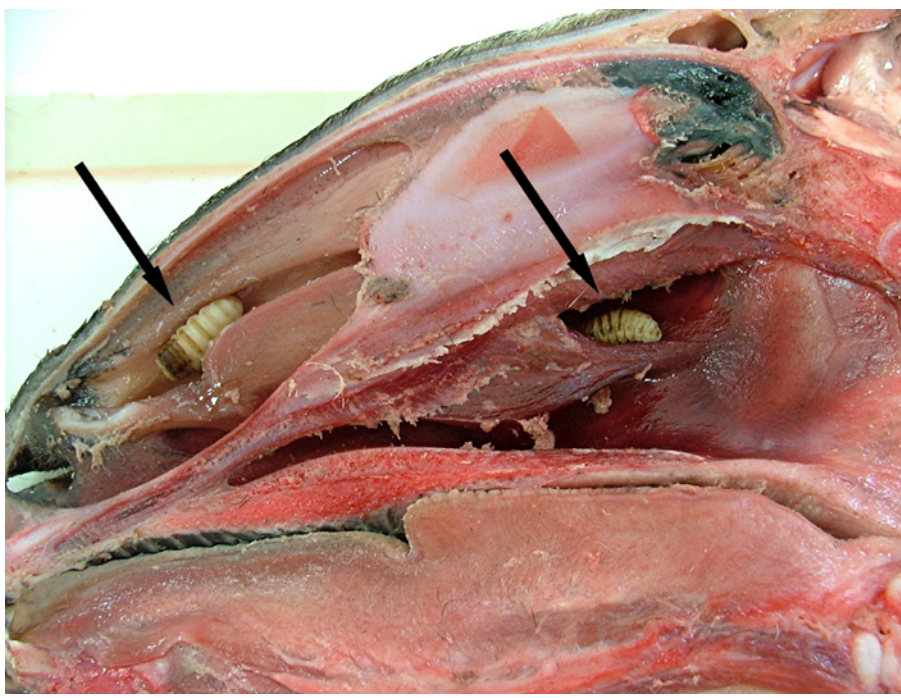
The L_3 larvae then move to the nostrils at which point they either crawl out or are sneezed out onto the ground where they burrow just below the surface and pupate.

The L_1 and L_2 larvae have the capability to remain dormant throughout the winter months and moult during the warmer spring months.



***Oestrus ovis* larvae removed from the sinuses of an infected ewe.**

The presence of adult flies has an unnerving effect on a flock that will tend to huddle close together with their faces toward the centre and facing down.



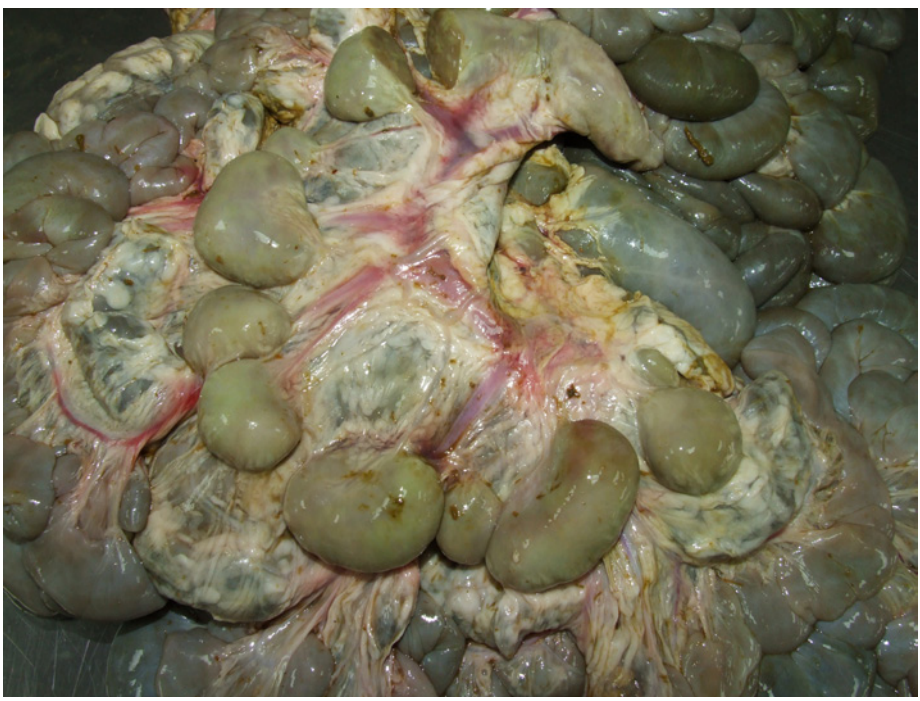
***Oestrus ovis* larvae in the sinuses of a ewe. Head sectioned medially**

LINGUATULA SERRATA
Canine tongue worm

Linguatula serrata is a pentastome arthropod worm of the nasal and respiratory passages of canines, felines and occasionally humans. Colloquially known as the tongue worm due to the shape of the adult which possesses two pairs of retractable hooks by means of which it anchors to the mucosal lining of the definitive hosts respiratory passages.

The adult male varies from 1.5-2cm in length compared to the 8-13cm of the female. Eggs are passed in nasal discharge or faeces and when ingested by sheep they hatch in the intestines, the larvae burrow through the intestinal wall, migrate to the mesenteric lymph nodes where they moult 6-9 times to produce a nymph approximately 5mm in length that resembles the adult.

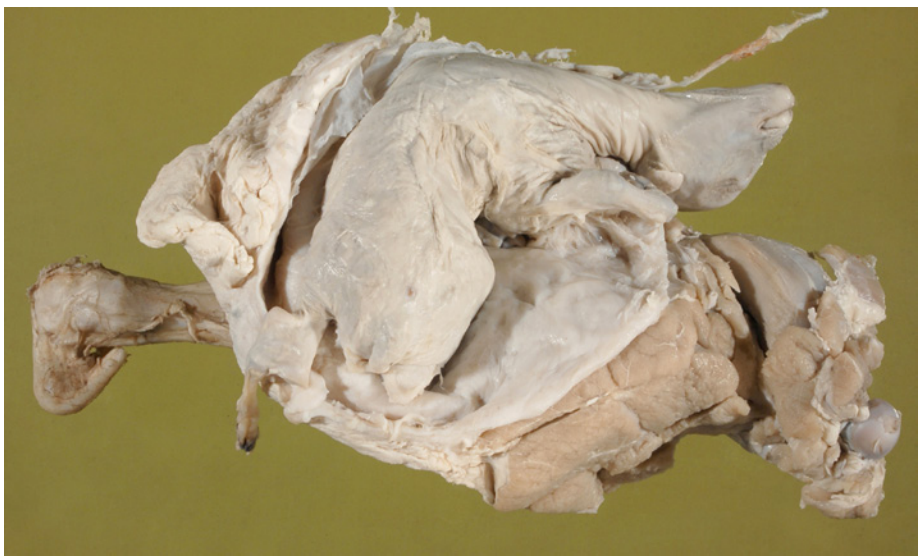
The mesenteric lymph nodes become enlarged and contain cysts within which is usually a viscous fluid surrounding the nymph. The lifecycle is completed when the definitive predatory host ingests the viable nymph.



Enlarged, oedematous and green mesenteric lymph nodes due to L.serrata - an eosinophillic reaction to parasite presence

AN ANOMALY – A 'PARASITIC' FOETUS

An incidental finding during post mortem inspection in a ewe plant was a large neoplastic growth on the hind leg. On incision to reach a differential diagnosis a partially formed lamb was found within the encapsulated area.



An encapsulated parasitic foetus in hind leg (sample fixed in formalin)

4

NEOPLASIA

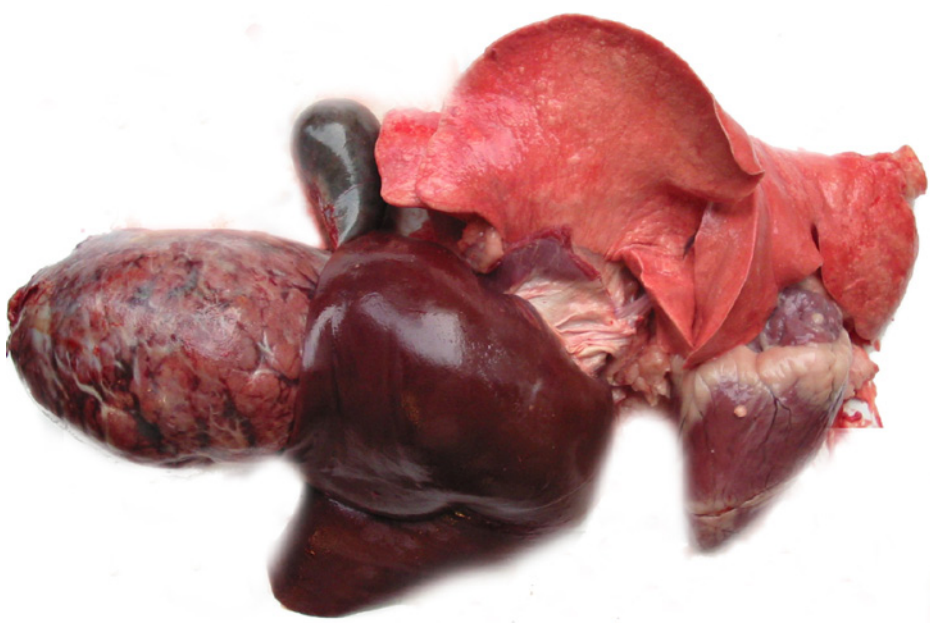
TUMOURS

INTRODUCTION

The term tumour literally applies to any abnormal swelling, but nowadays it tends to refer exclusively to neoplasms, which are abnormal new growth of tissue, in which cell multiplication is uncontrolled and progressive in addition to serving no purpose and growing faster than normal tissue. These neoplastic formations are classified as being either benign or malignant, the gross appearance of neoplasia is variable being dependant on their origin and type. Both malignant and benign tumours are served by increased blood vascularisation, any tumour requiring nutrients to continue growth. In the case of the rapidly growing malignant tumours the blood vessels tend to be thin, poorly formed and prone to rupture creating the appearance of haemorrhagic areas within the tumour. Masses require histological examination for a definitive diagnosis, however there are characteristics of benign and malignant tumours.

BENIGN TUMOURS

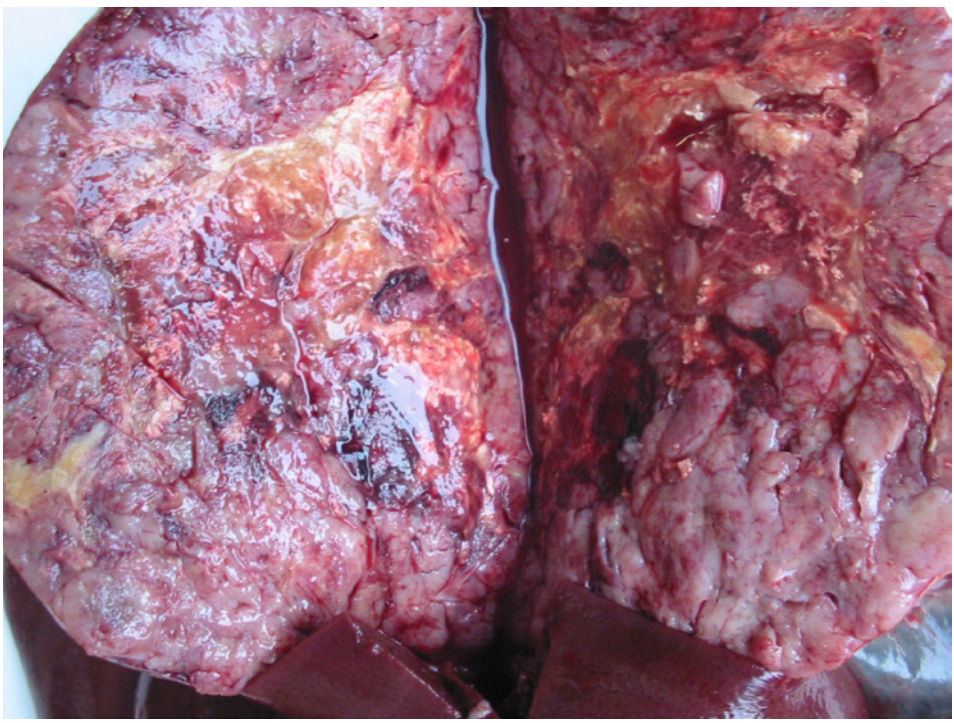
These grow slowly, pushing aside normal tissue without invading it. They are usually encapsulated and do not produce secondary tumours within the body. Although some benign tumours are caused by viruses, on the whole they do not appear to be infectious. The effect of their presence tends to be physical, blockage of systems in the body and pressure applied to organs being obvious examples.



A large tumour (hepatic tumour) attached to a liver

Malignant tumours grow in an irregular shape and so quickly that the cell nutrition can become affected leading to cell death with areas of necrosis. Malignant tumours invade surrounding tissue and can produce secondary tumours within the body through a process of metastasis (cells transported through the blood stream or lymphatic system.)

Tumours are further classified based upon the type of tissue cell from which the tumour cells originated. For example: Fibromas – composed of neoplastic fibrous cells; Lipomas – fat cells; Chondromas – cartilaginous cells; Osteomas – bone cells; Myelomas – blood cells; Adenomas – glandular cells; etc. Tumours with the suffix sarcoma or carcinoma are malignant e.g. lymphosarcomas.



An incised malignant neoplasm, showing lobulation necrosis and haemorrhagic areas.

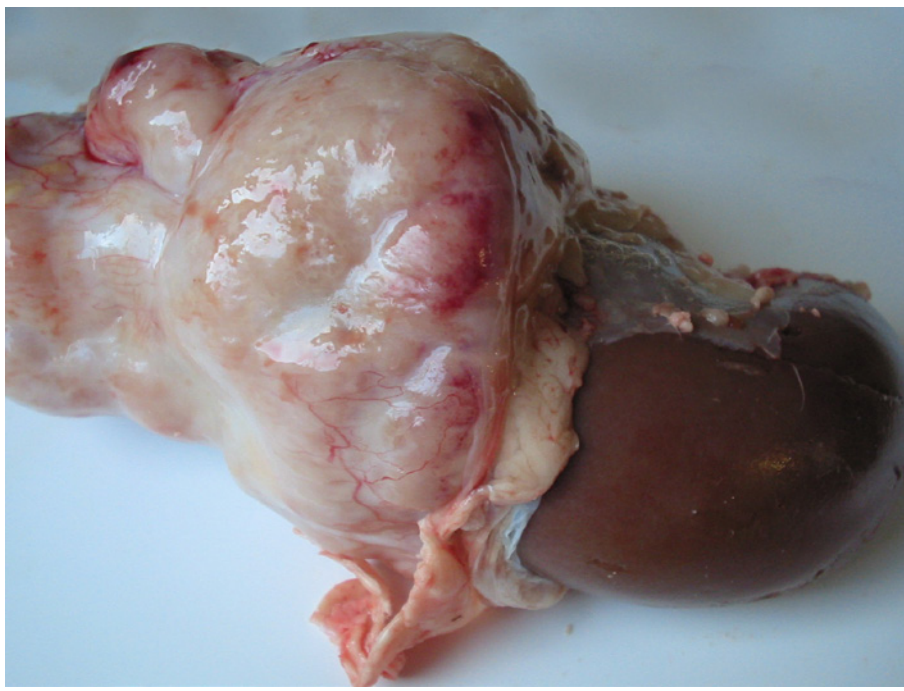
METASTASIS

This is the method of dissemination of malignant neoplasia throughout the body forming secondary tumours, the transfer of tumour cells tending to be either through direct contact between adjacent surfaces (contact spread) or via the blood or lymphatic circulation. Metastasis occurs as a product of the invasive abilities of malignant neoplasia. Detachment of tumour cells from the unencapsulated mass is followed by

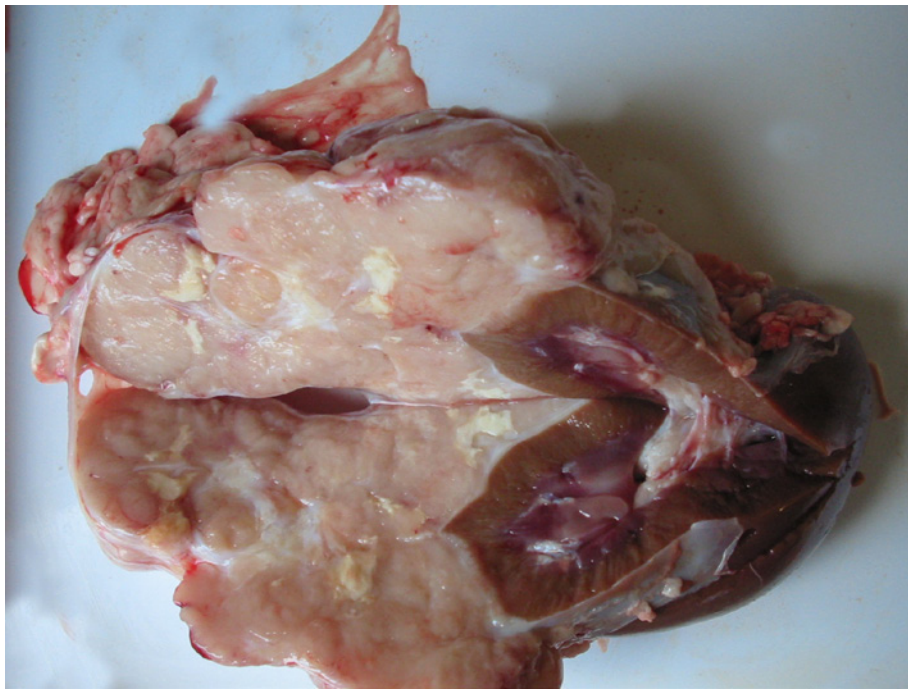
infiltration of cells into surrounding tissue (contact), or erosion of the epithelial cells lining the blood and lymph vessels allowing ingress into the circulation. Studies have shown, however, that less than 1 in 10,000 neoplastic cells that enter these circulations will produce metastatic growth. As a general rule of thumb sarcomas are spread via the blood circulation and carcinomas via the lymphatics.

CHARACTERISTICS OF NEOPLASIA

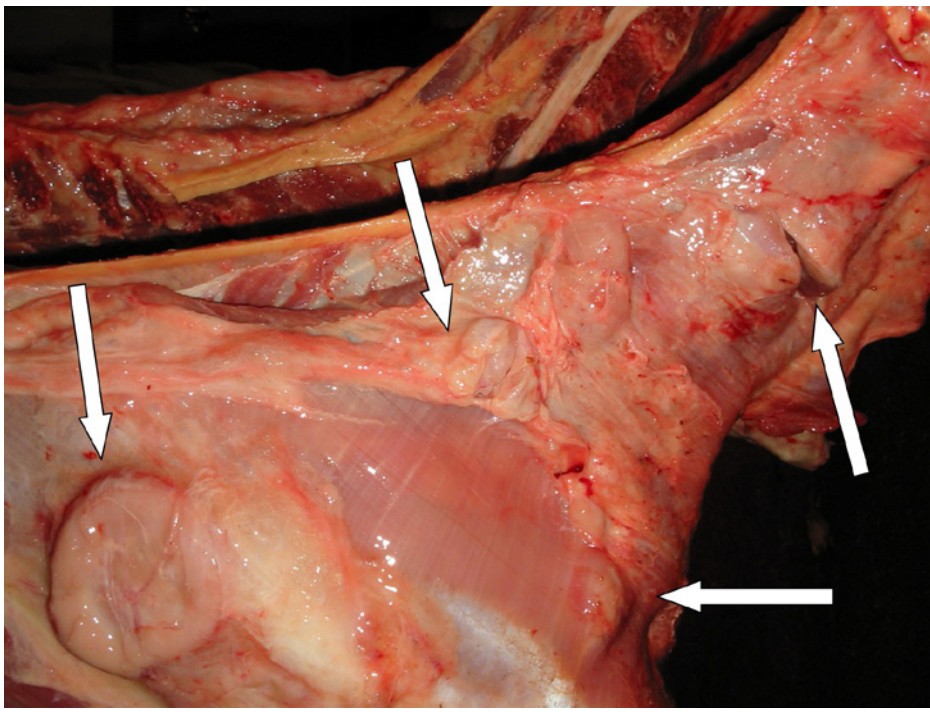
Benign Tumours	*Malignant Tumours*
Grow slowly	Rapid growth
Usually encapsulated	Unencapsulated
Do not invade local tissue	Highly invasive
No metastatic spread	Metastatic spread
Cut surface often and homogenous	Often variegated (haemorrhage and necrosis)



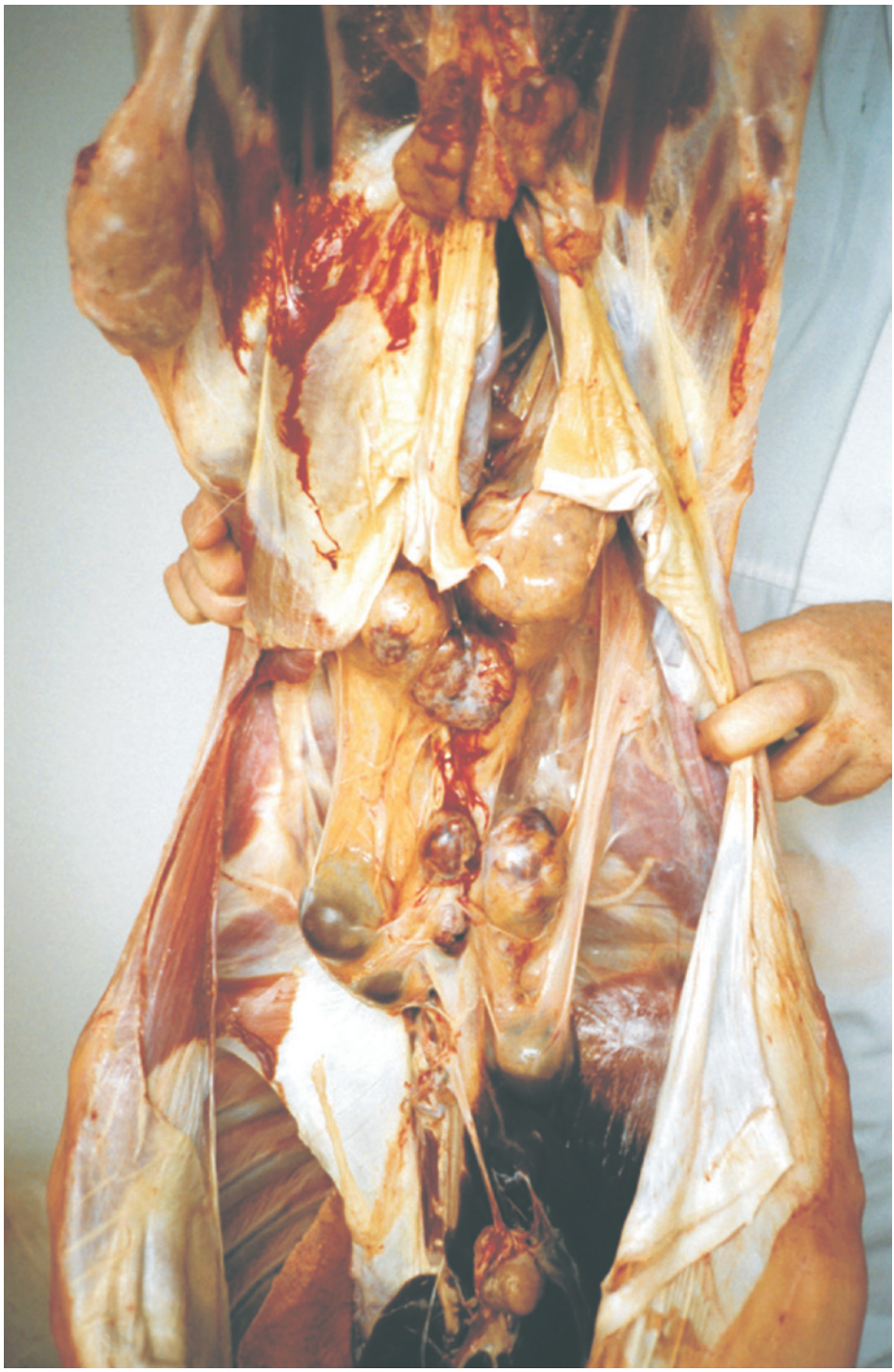
Lymphosarcoma; kidney



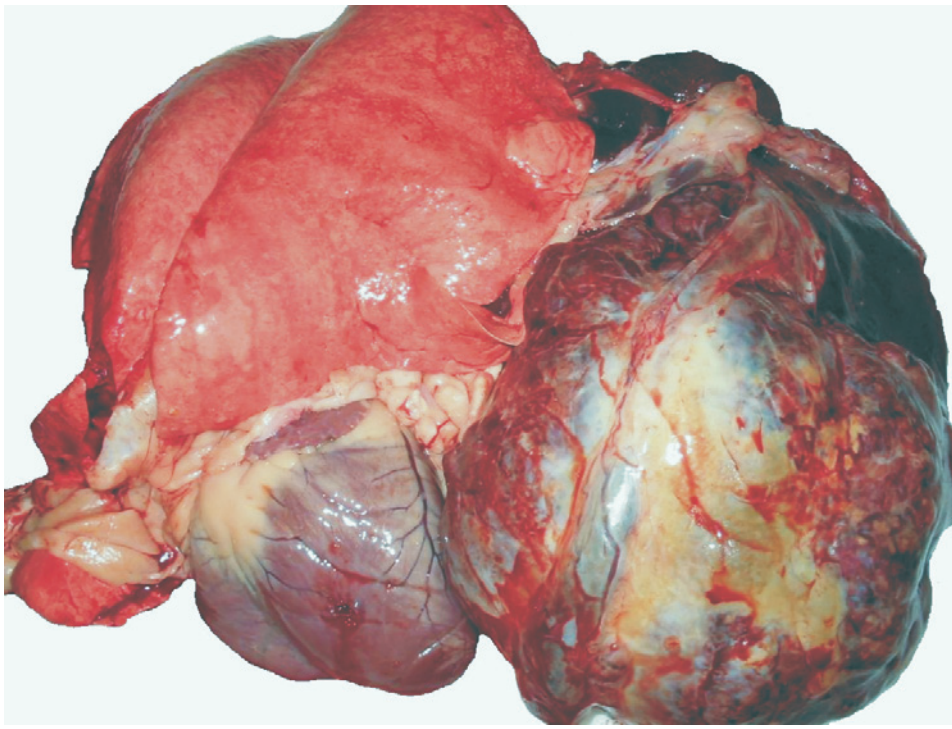
Incision through neoplastic growth



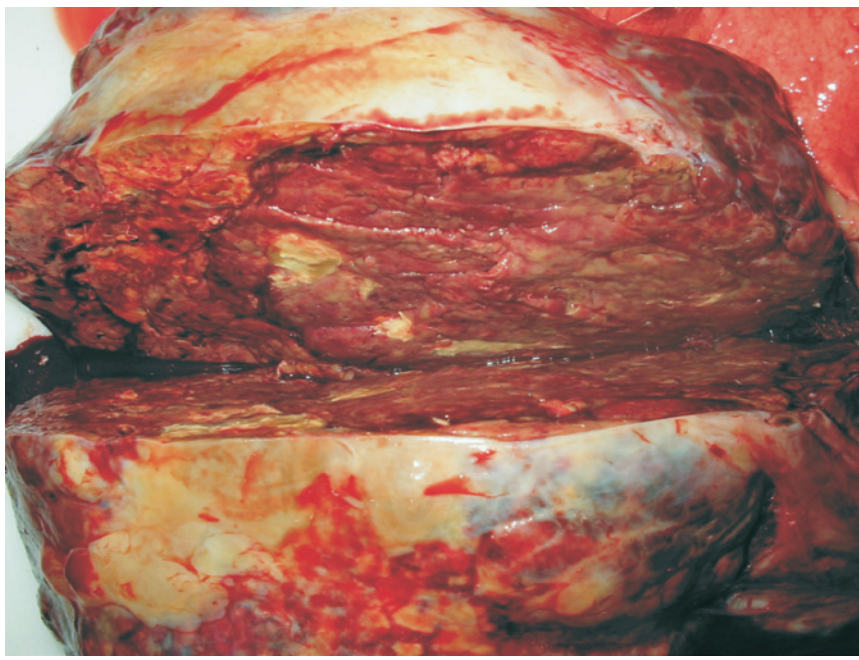
Multiple nodules in the shoulder and neck region



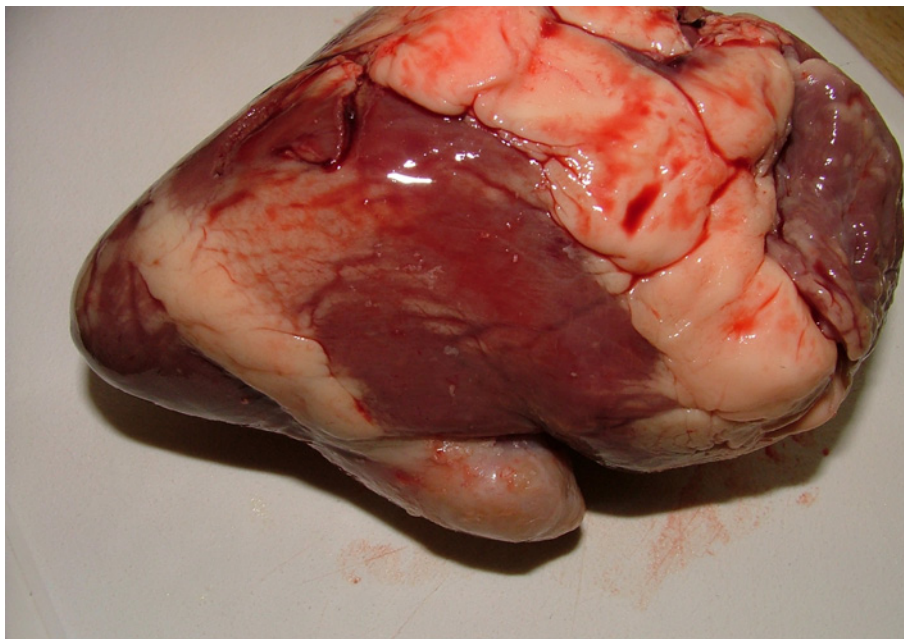
Multiple lymphosarcomas in a ewe carcase



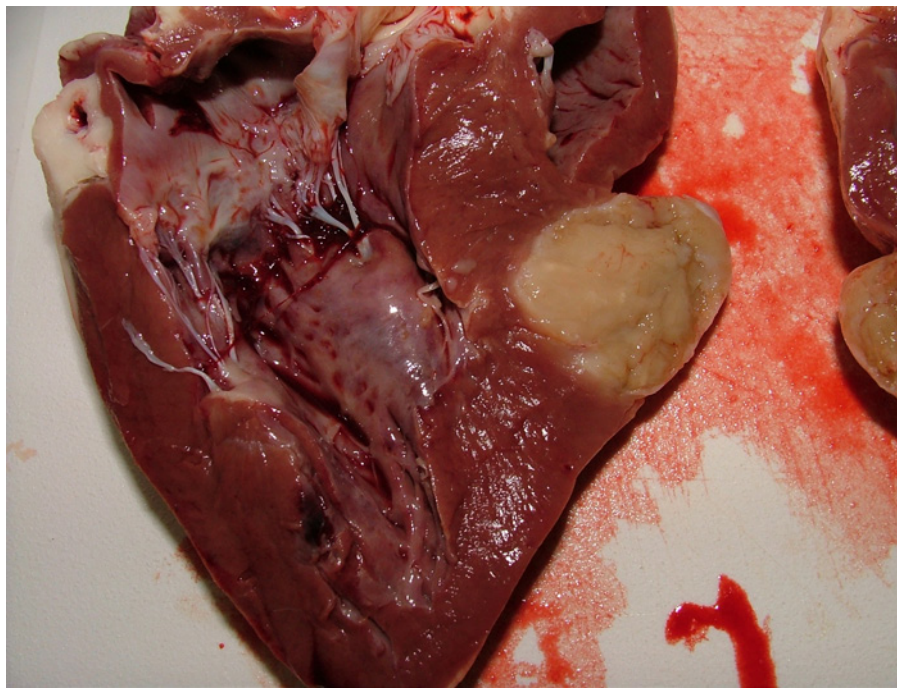
Cardiac tumour compared with normal heart on left



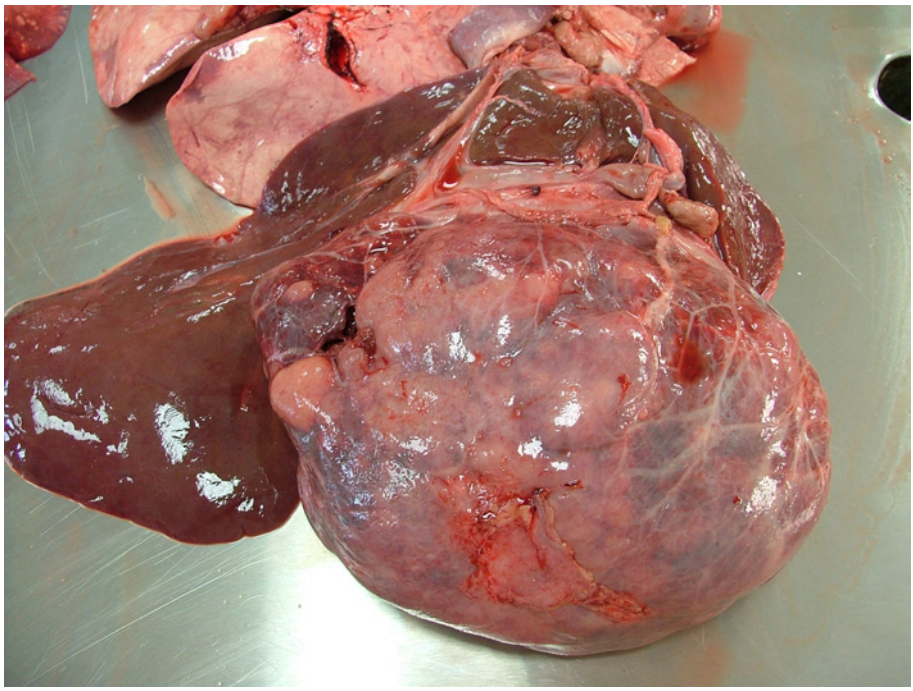
Incision through affected heart



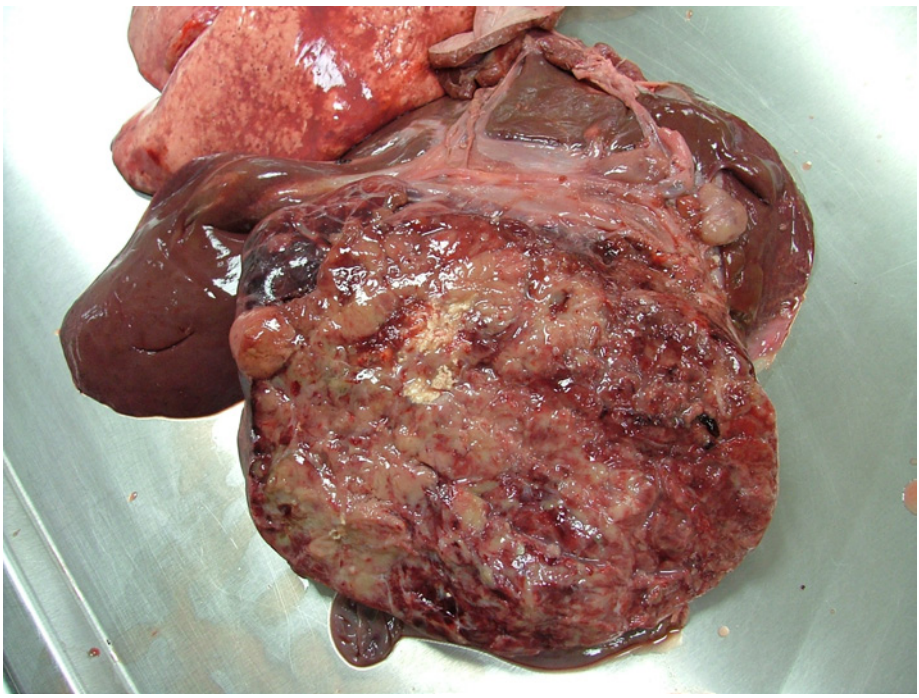
Cardiac tumour



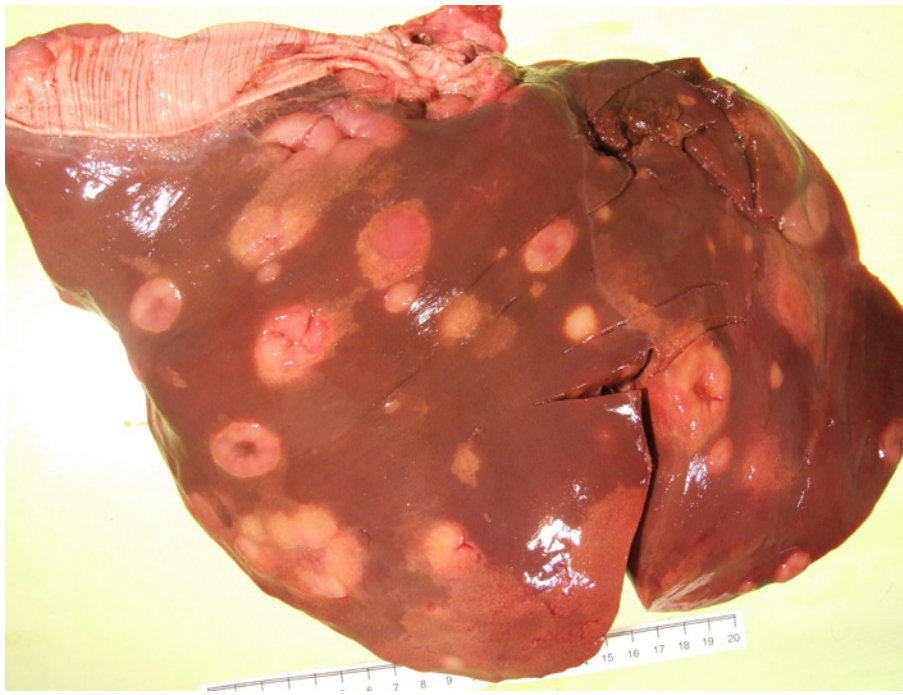
Incised lesion



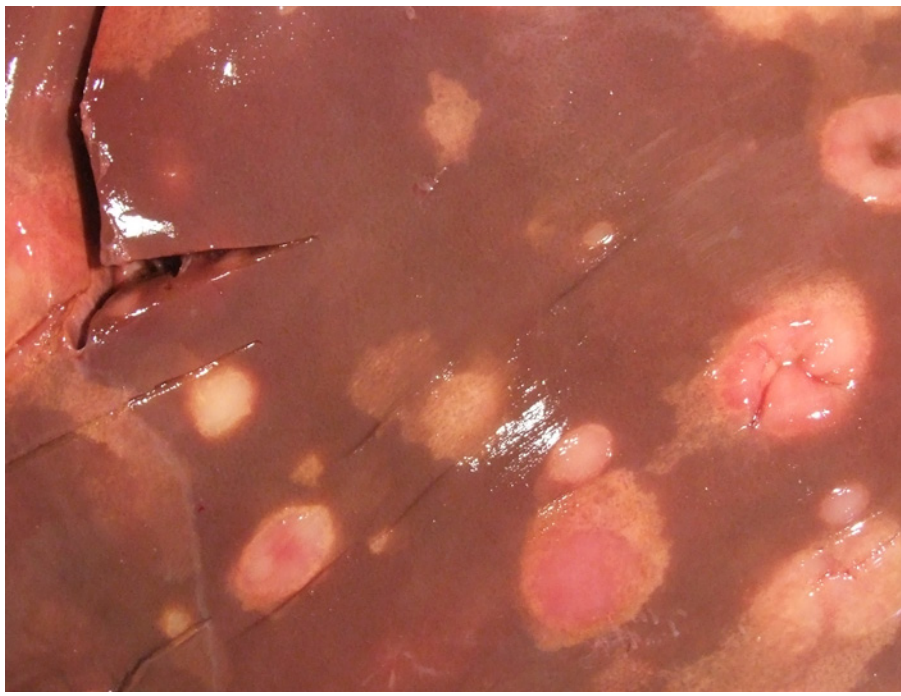
Large hepatic malignant tumour



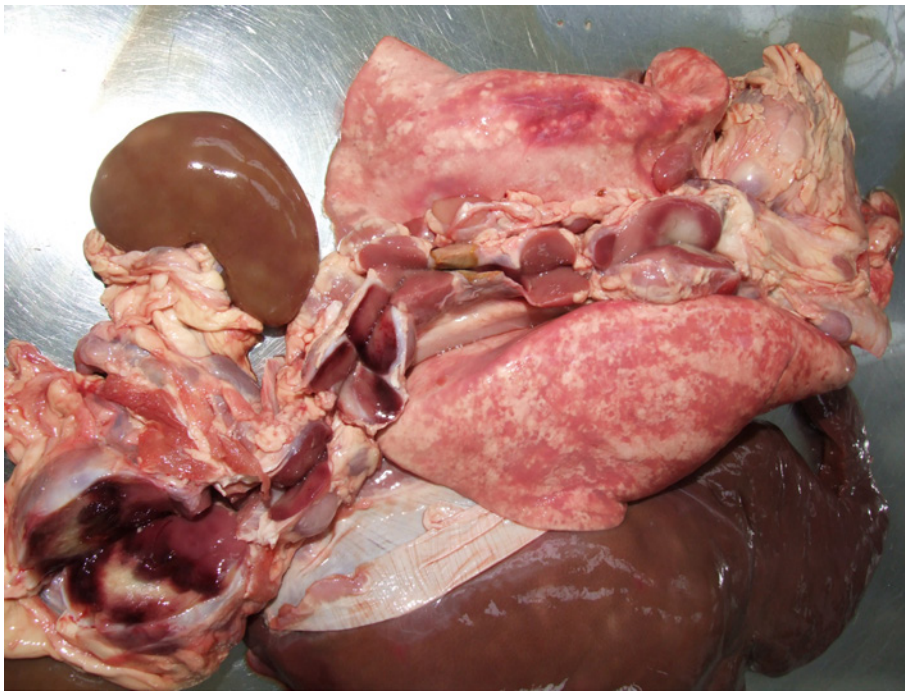
Variegated cut surface with areas of necrosis



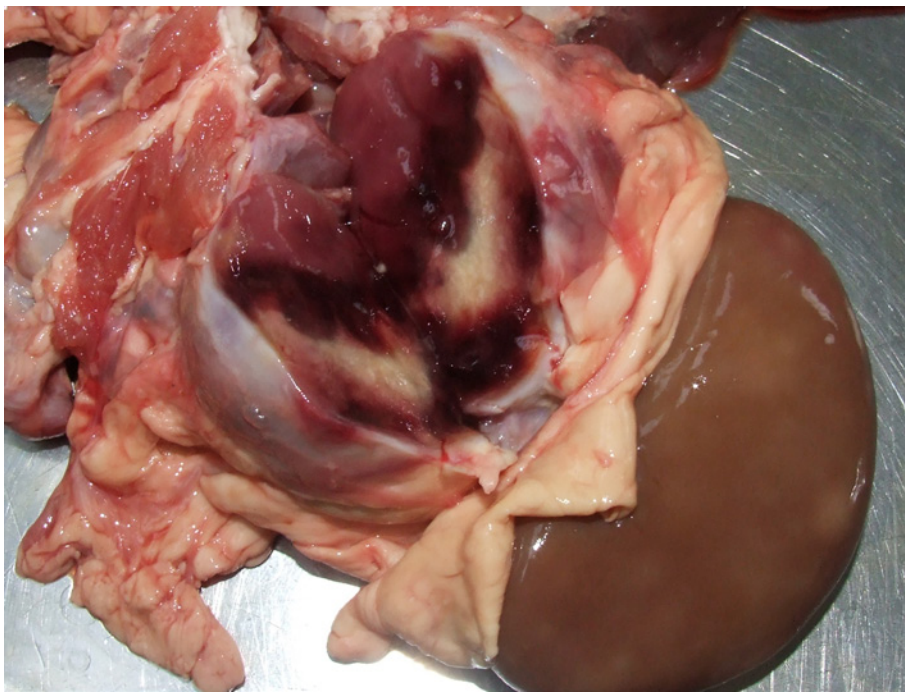
Multiple neoplastic nodules in liver (possibly biliary carcinoma, hepatic tumours, lymphosarcoma or metastatic lesion)



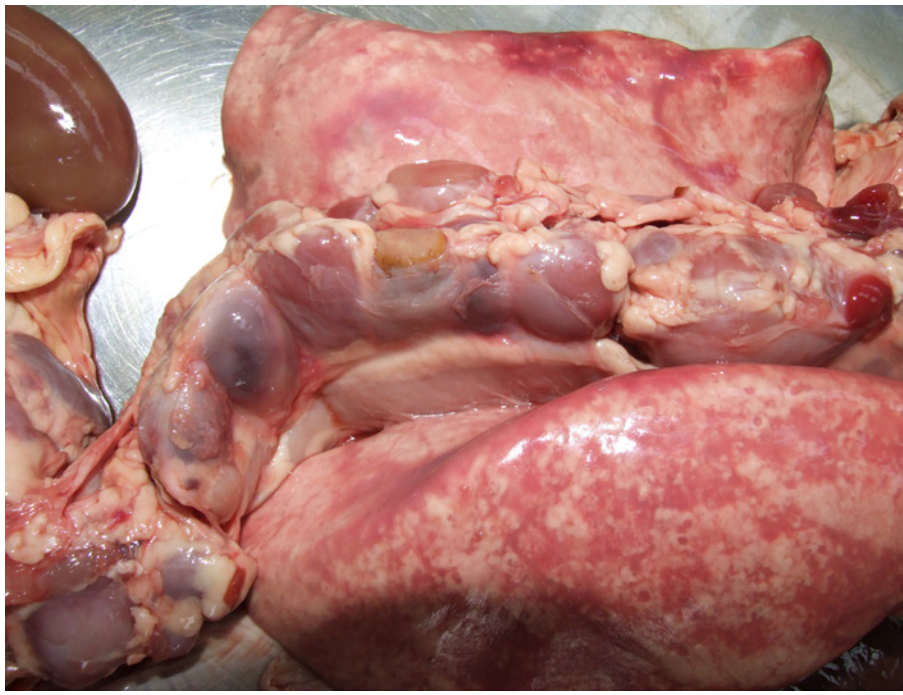
Lesions have a depressed centre



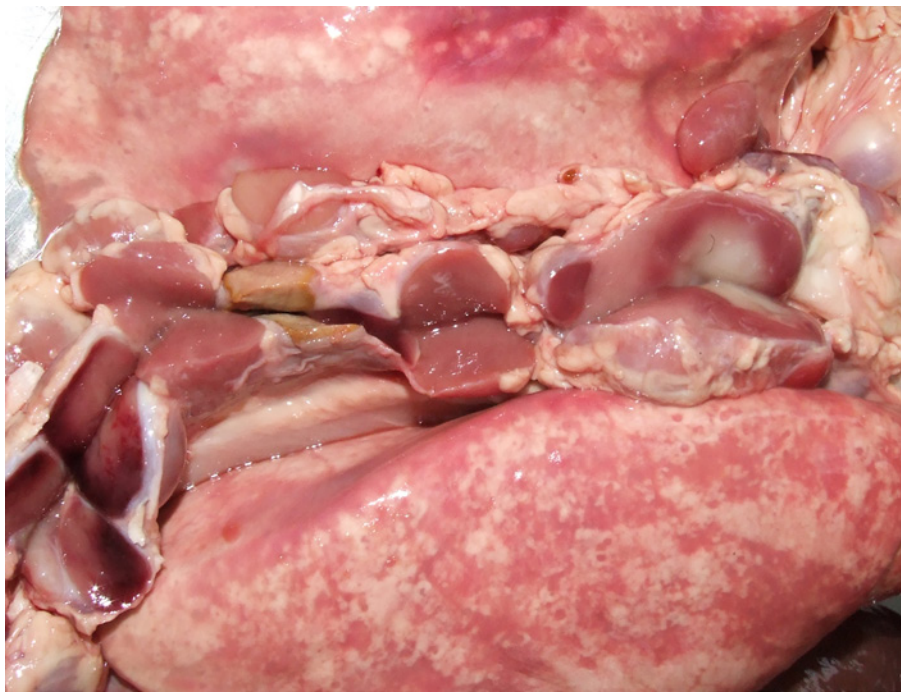
Ovine lymphosarcoma (diagnosed on microscopy)



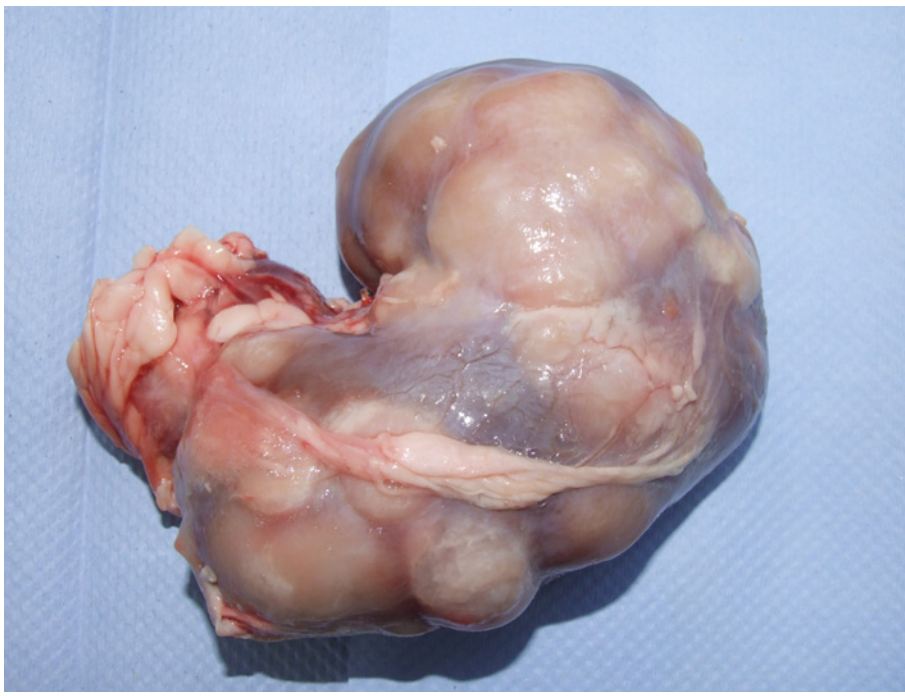
Detail of neoplastic formation in renal area



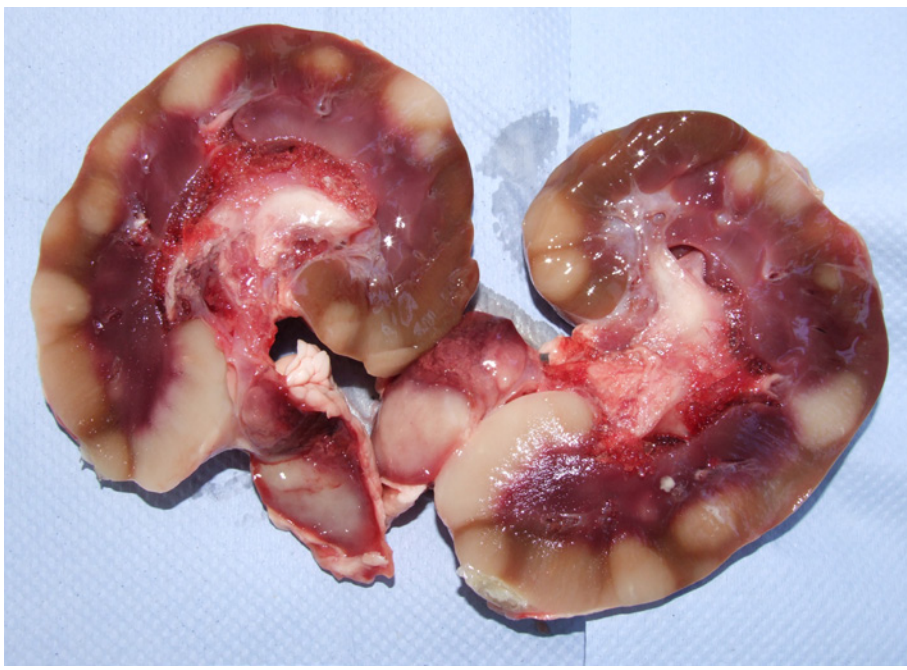
Lymphosarcoma – detail of mediastinal lesions



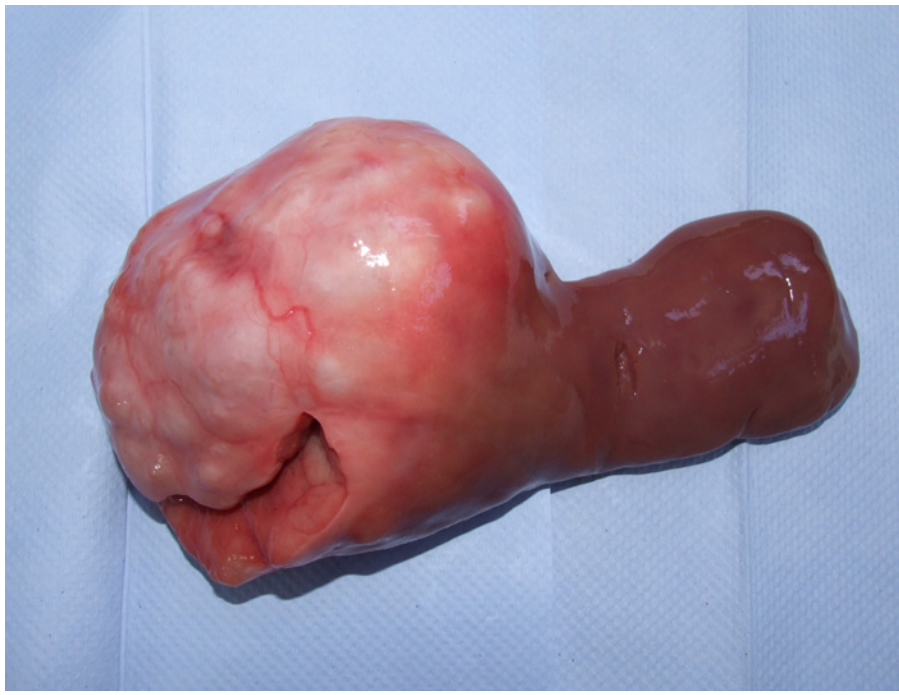
Incised neoplastic formations



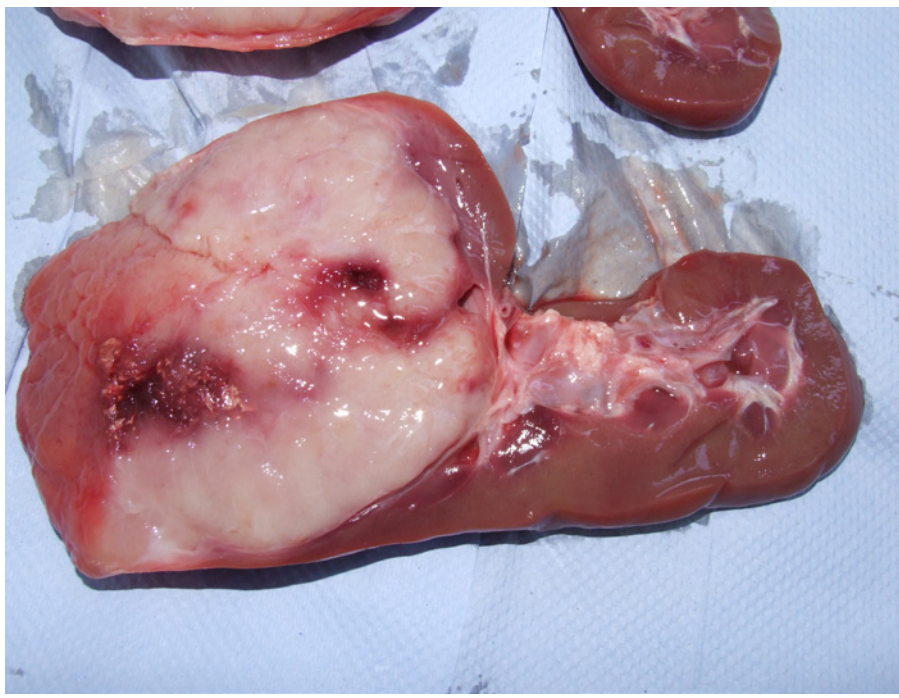
Multiple neoplastic formations in a kidney



Incision through above kidney (possibilities include renal tumour, lymphosarcoma or metastatic lesion)



Distortion of the kidney due to neoplastic formation



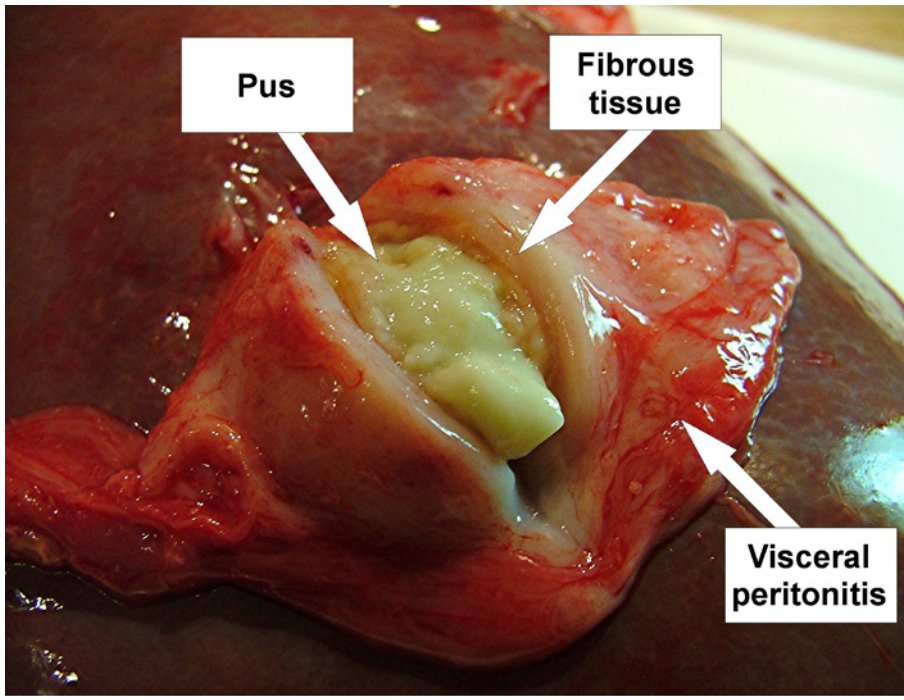
Incision through above kidney

5

CONDITIONS ENCOUNTERED AT OVINE POST MORTEM INSPECTION

ABSCESSES

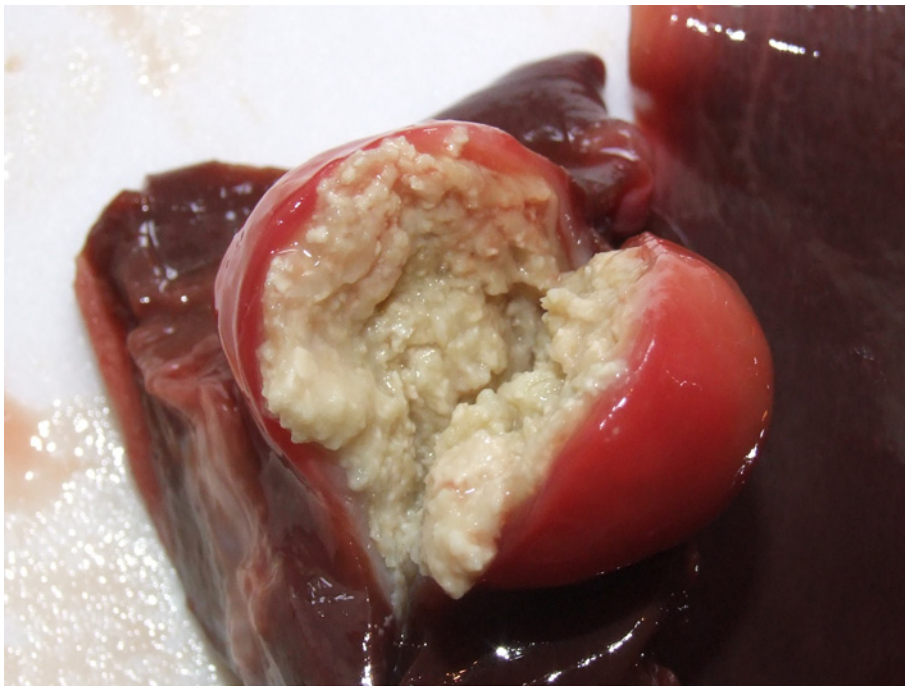
An abscess is a collection of pus surrounded by fibrous tissue; it is part of the bodies' defence mechanism normally triggered by the presence of pus forming bacteria. This fibrous tissue is a normal healthy reaction and consists of newly formed connective tissue and blood vessels. Pus may consist of dead or dying bacteria, dead tissue cells that formerly occupied the area, dead or dying white blood cells, debris and a certain amount of fluid exuded from the blood vessels in the vicinity.



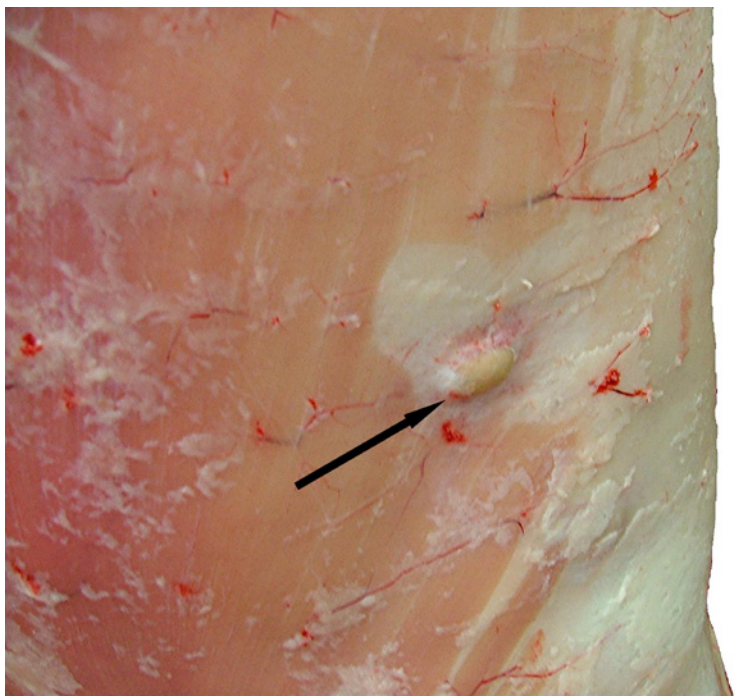
Anatomy of a hepatic abscess

If the organisms multiply, the abscess will continue to increase in size until the pus escapes. If the abscess is near the surface of the tissue part of the wall becomes necrotic and is removed allowing the pus to discharge.

When the abscess is deeper within tissue and the pus is under tension it can burrow through less resistant tissue and reach the surface through a canal known as a sinus. If the bodies' immune response is sufficient to destroy the organisms contained in the pus the abscess may remain localised and be eventually absorbed, caseated or calcified.

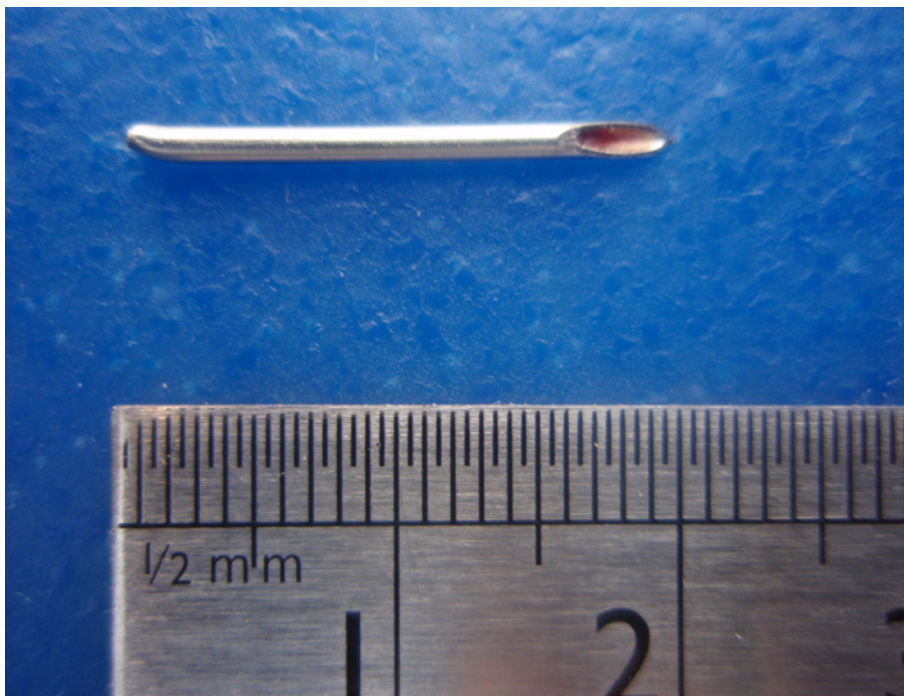


Thick capsular wall and granulomatous pus in a hepatic abscess

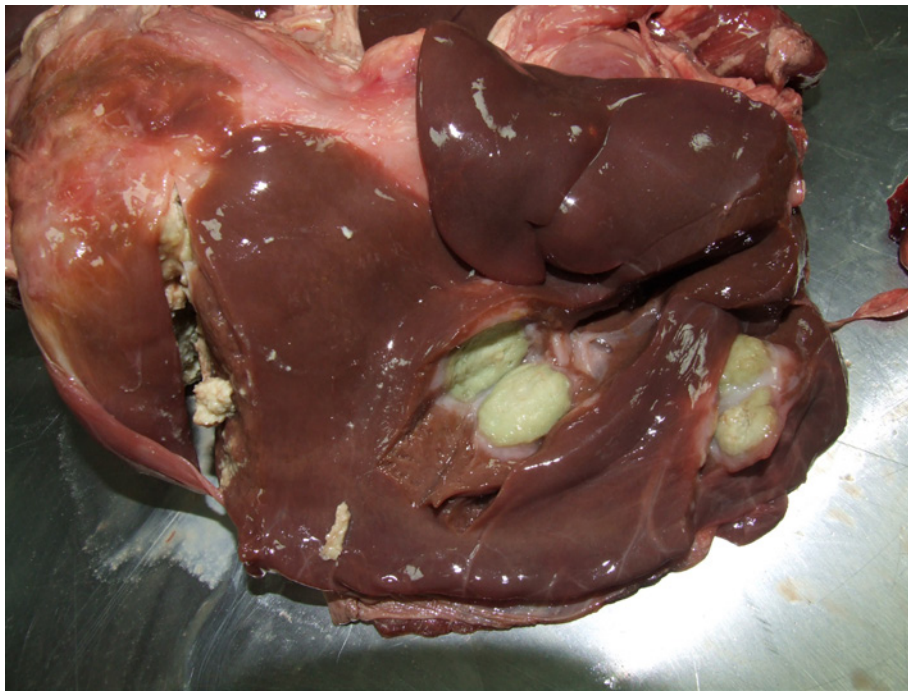


An injection abscess on the flank

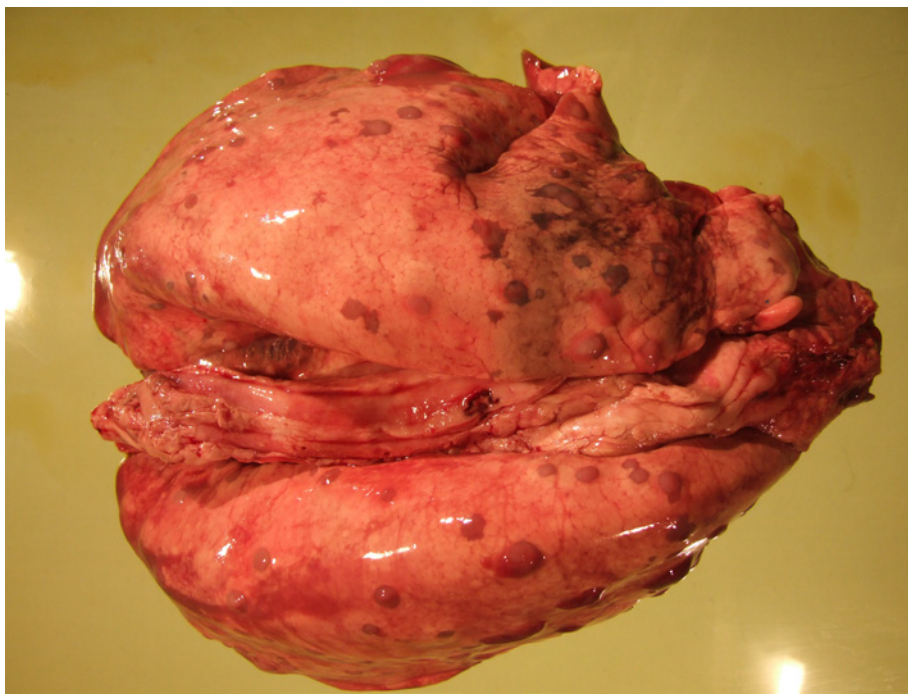
Intramuscular abscesses are generally caused by injection, the use of unsterilised needles introducing infection through the skin. As well as injection abscesses in the neck and flank, superficial abscesses are common in the inner thigh area due to the administration of the Orf vaccine. If localised the affected area is trimmed and rejected as unfit for human consumption which affects the carcase quality, especially when removed from commercially higher value cuts of meat such as the legs. If the infection is not contained, it can lead to pyaemia, which warrants the rejection of the whole carcase and associated offal. Hepatic abscesses present a high risk of subsequent pyaemia if they vent into the vena cava.



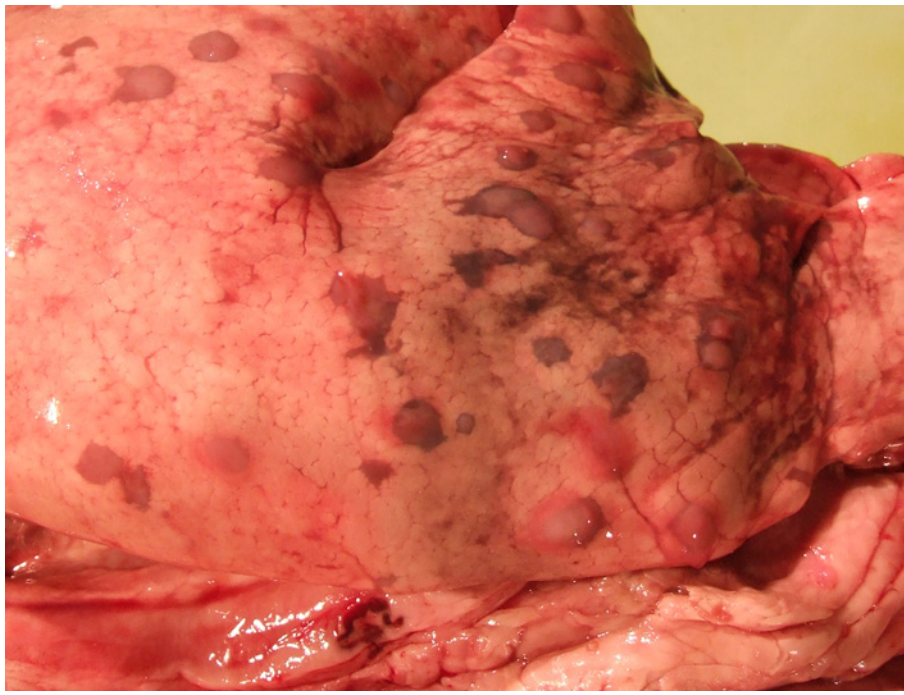
A broken needle removed from a large shoulder abscess



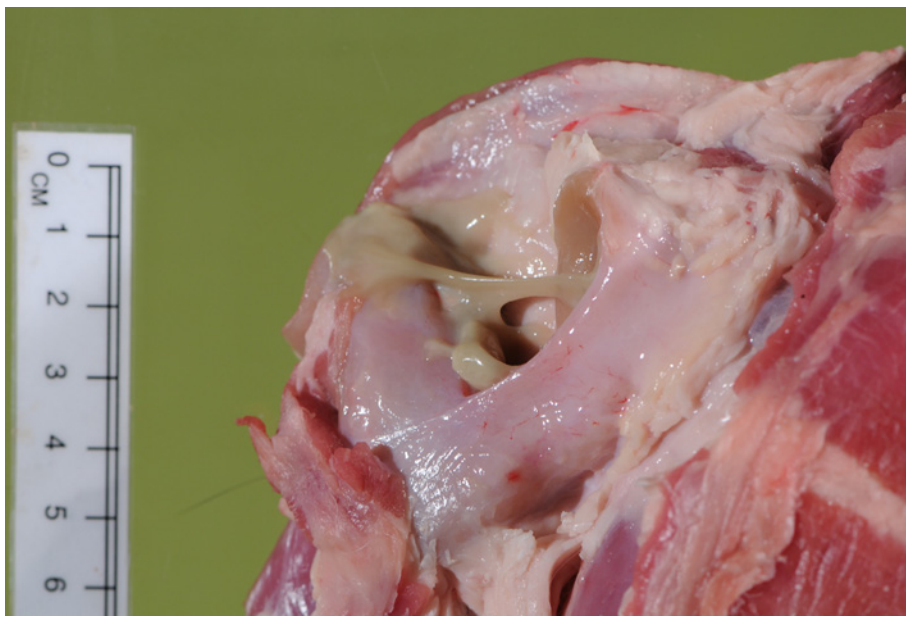
Hepatic abscesses possibly due to ruminal acidosis



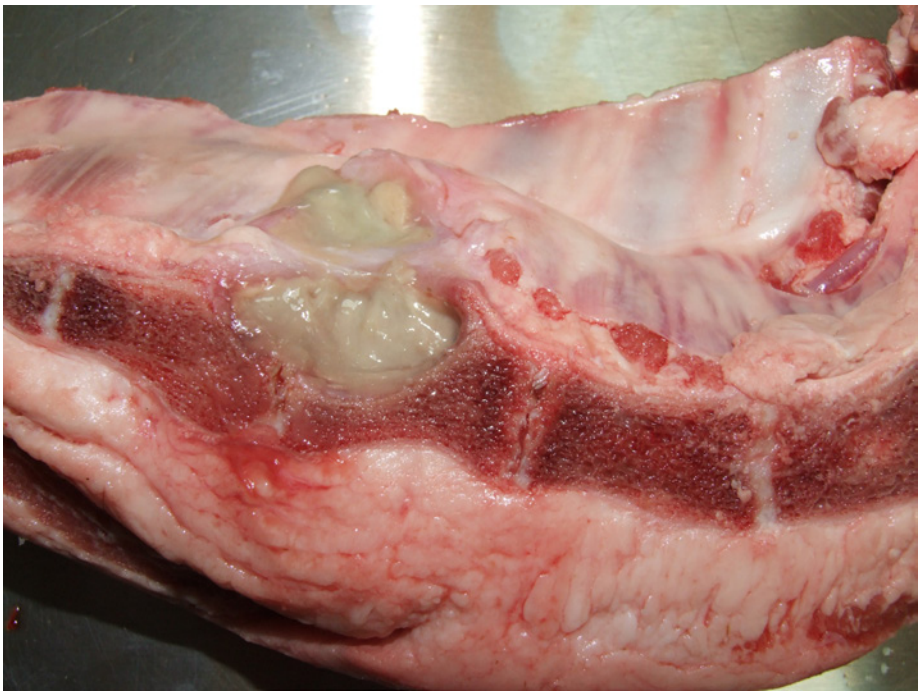
***A. pyogenes* abscessation of lungs**



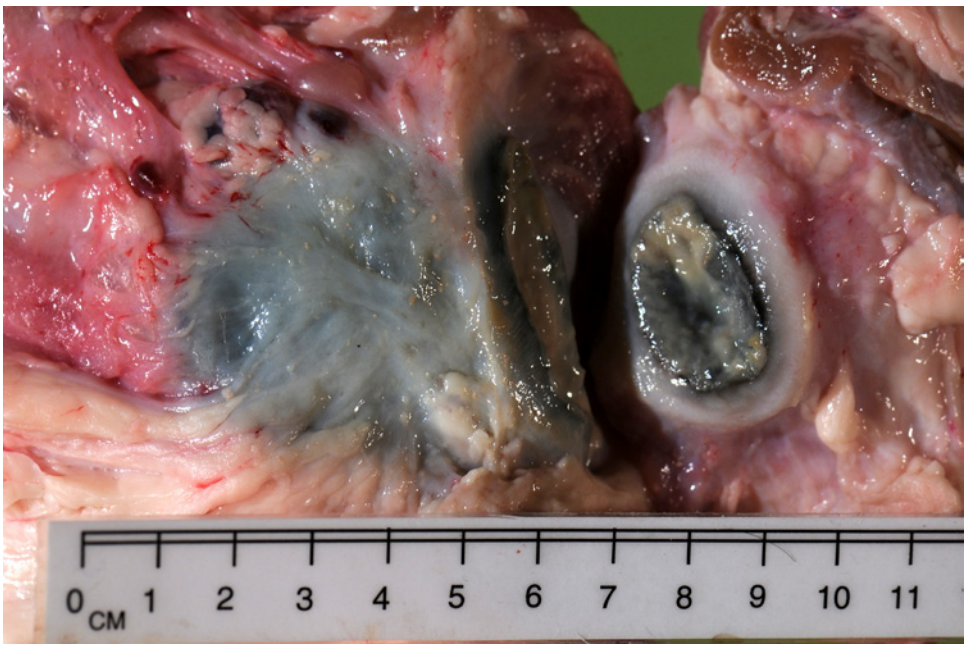
Detail of abscesses



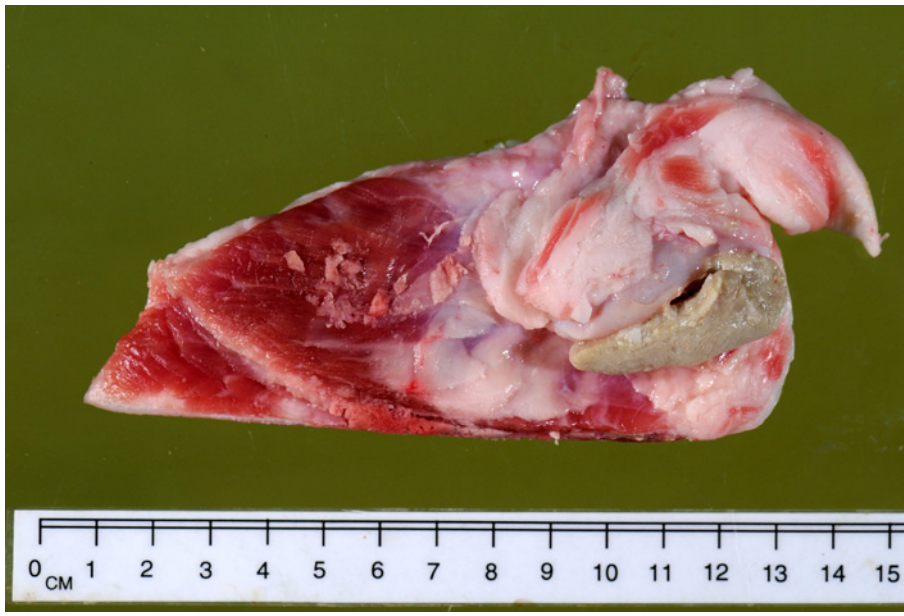
A large abscess ventral to the scapula, most probably due to injection. Injection abscesses are common in sheep; the large number held in a flock can preclude sufficient needle sterilisation between animals



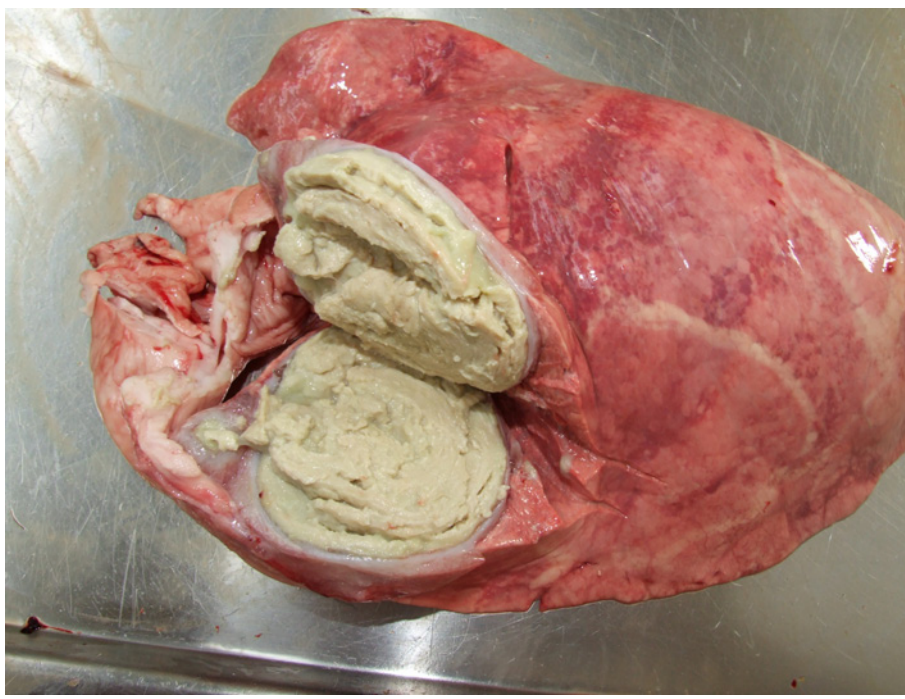
Ventral sternum, osteomyelitis and abscessation



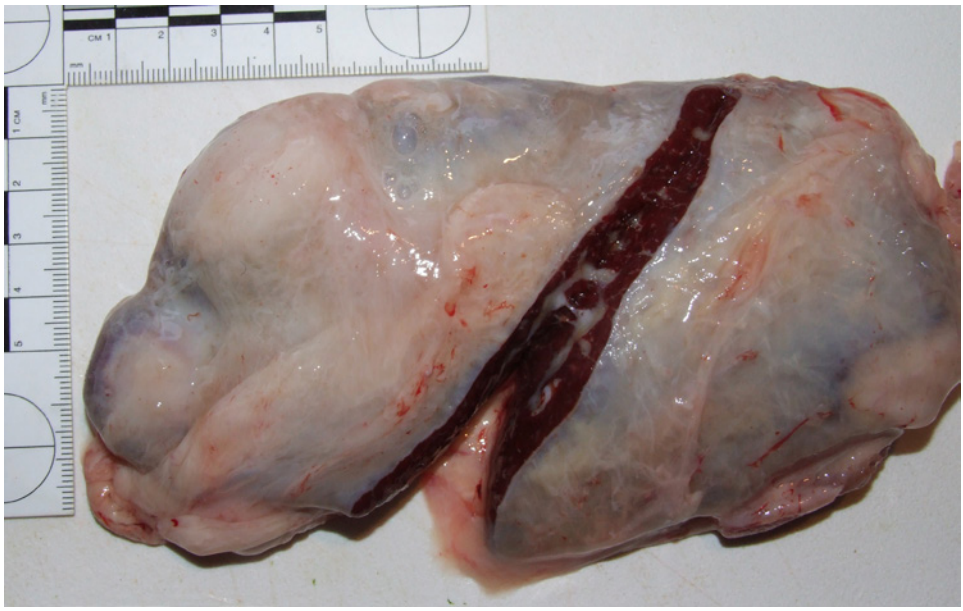
Intramuscular injection – note thick capsule and necrosis



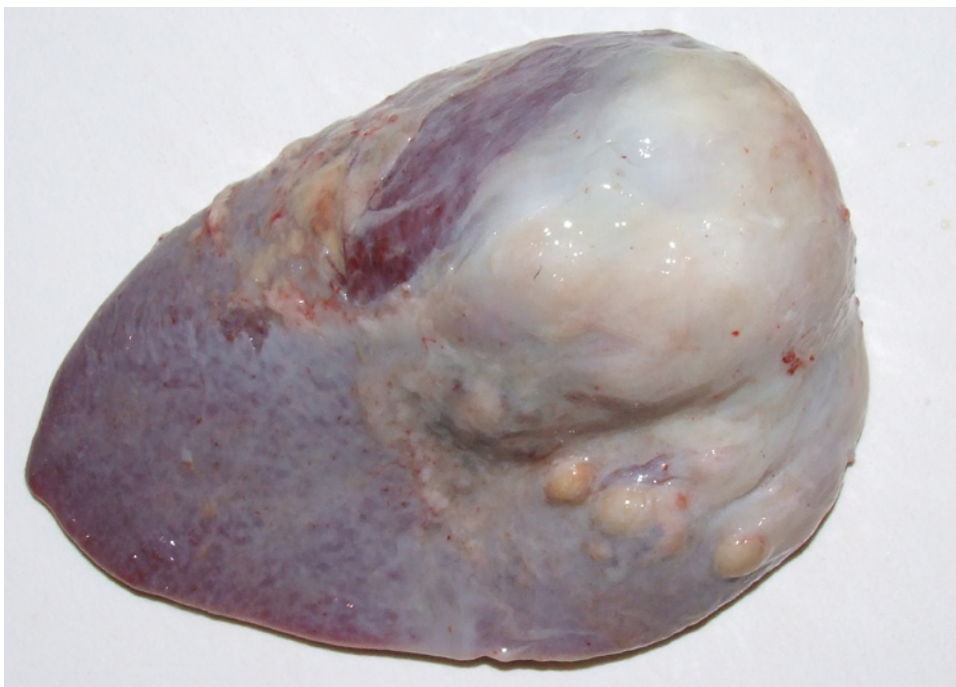
Abscess formation ventral scapula



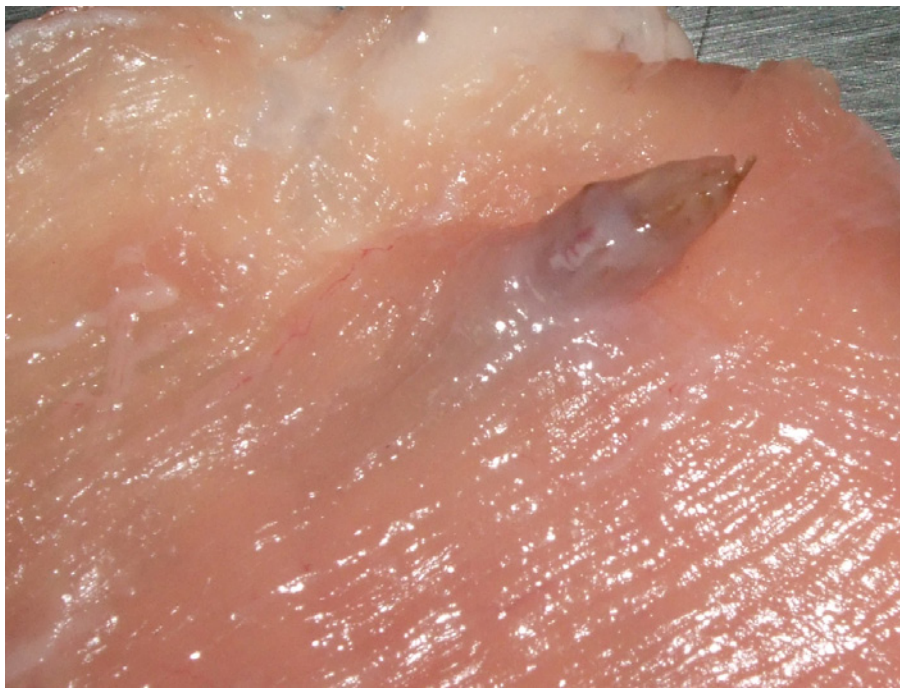
A large (5cm diameter) encapsulated abscess in apical lobe of lung



Multiple Splenic abscesses

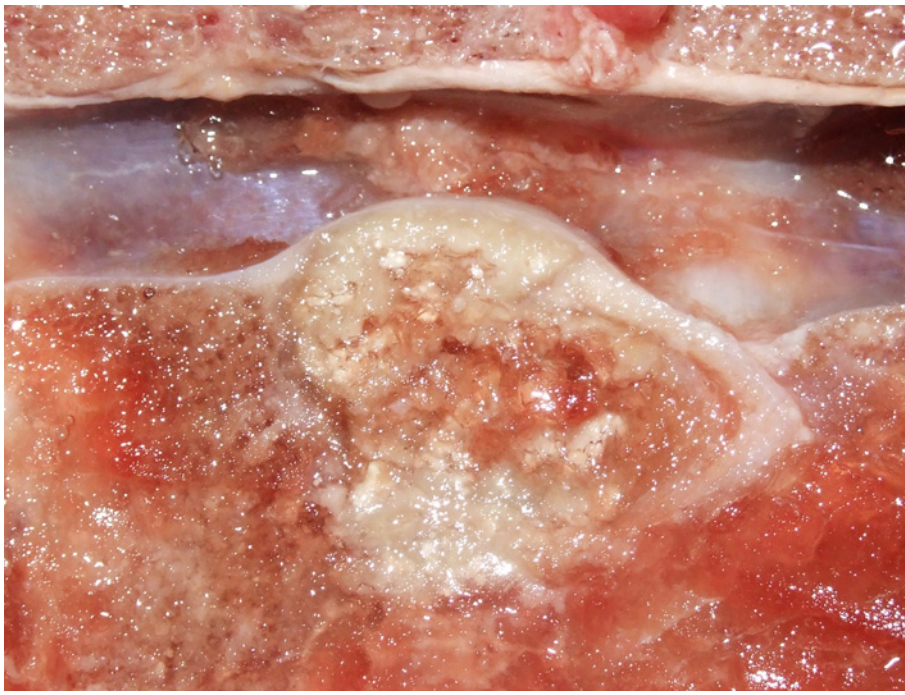


Multiple Splenic abscesses – because of the lymphatic properties of the spleen it must be determined whether the presence of abscesses is from contact spread or via the bloodstream



A grass awn penetrating into subcutaneous fascia can lead to an abscess

Osteomyelitis – vertebral column



Detail of abscess – impinging on spinal cord at this point

ANAEMIA

This is defined as a deficiency in the blood, the number and size of the red blood cells, or the haemoglobin in the blood. This may be due to haemorrhage, the presence of internal or external parasites, chronic illness, iron deficiency, bracken poisoning or in the case of aplastic anaemia, where the body ceases production of red blood cells; it may be drug induced.

Anaemia is difficult to detect in the live animal, which may show signs of breathlessness, loss of energy and appetite and have pale mucous membranes. Flocks that have a history of *Haemonchus contortus* are checked for anaemia by comparing the colour of the eyelid mucosa against a colour chart.

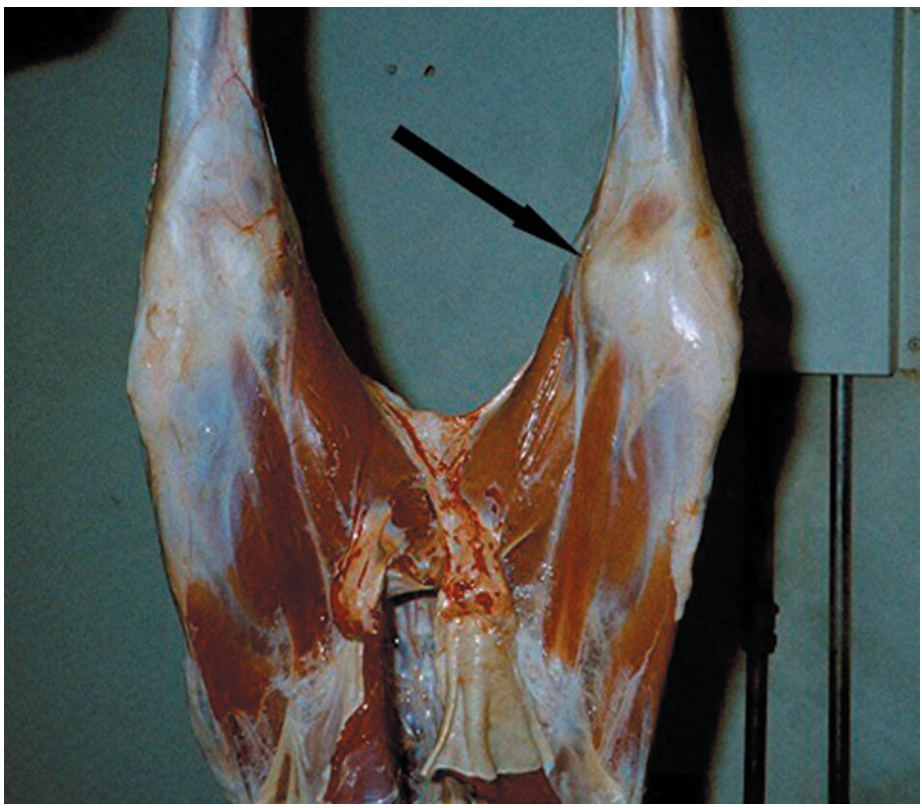
An anaemic carcase is generally pale in colour, and is often emaciated

ARTHRITIS

Arthritis is inflammation of the structures of joints between bones and their associated tissues. Arthritis may be infectious, traumatic or non-infectious.

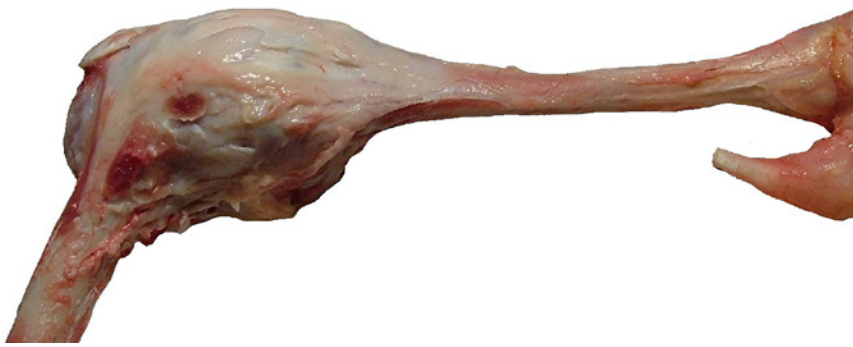
The synovial joints, for example knees, knuckles and shoulders, provide a nutrient rich fluid surrounded by a good blood supply that offers ideal conditions for bacterial growth. Microorganisms can enter the joint through a penetrative wound, via the blood stream or from a nearby area of infection, foot rot for example. The most common method of acquired infection is through the umbilicus, however studies have also demonstrated that *Streptococcus dysgalactiae* infection of joints can be achieved via the digestive route from the mother during feeding.

When infected the joint swells as fluid increases, the synovial membrane becomes hyperaemic and thicker (fibrinous). If the infection entered via, or gains access to, the blood stream it can lead to polyarthritis, where most joints are infected, especially the large joints of the limbs.

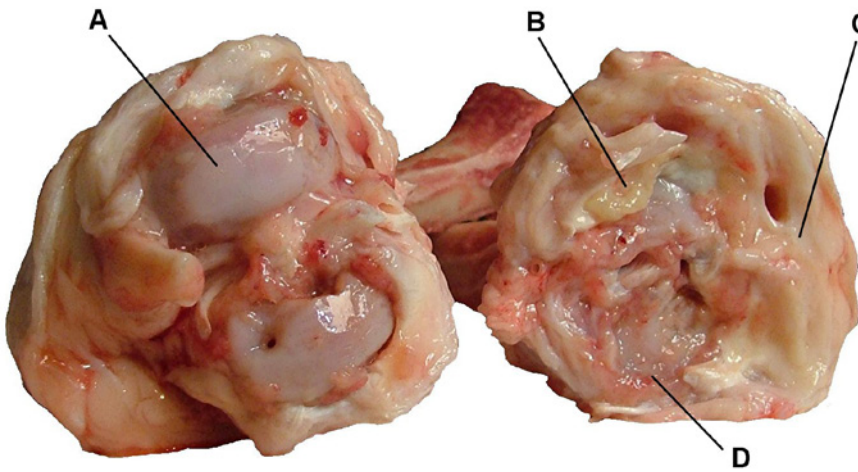


Arthritic joint (arrowed)

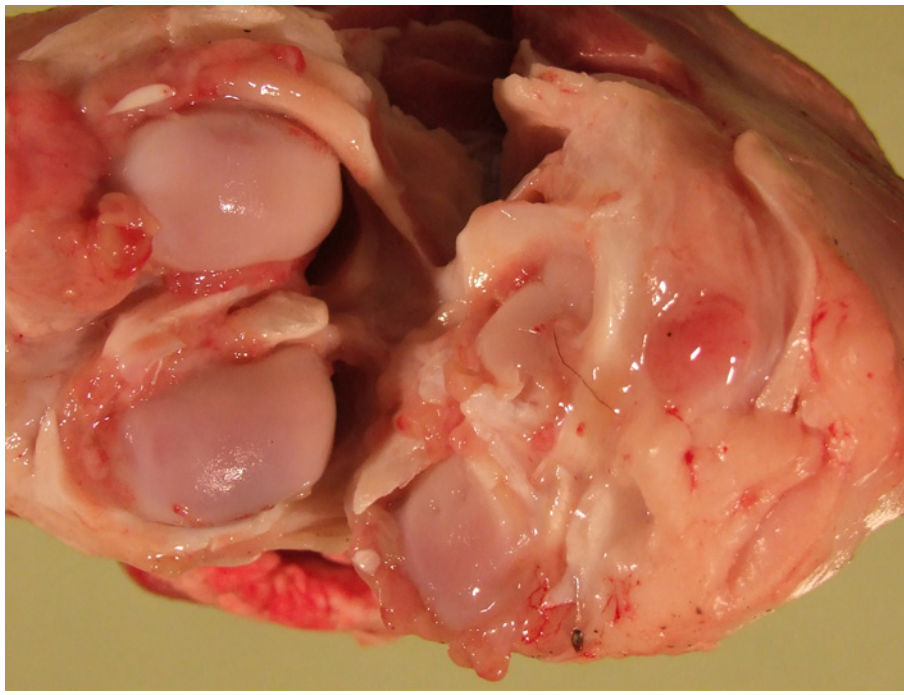
One of the most commonly isolated pathogens in cases of lamb arthritis is *Streptococcus dysgalactiae*; other implicated bacteria include *E.coli*, *Erysipelothrix rhusiopathiae*, *Actinomyces pyogenes*, *Pasteurella haemolytica* and *Streptococcus spp.*



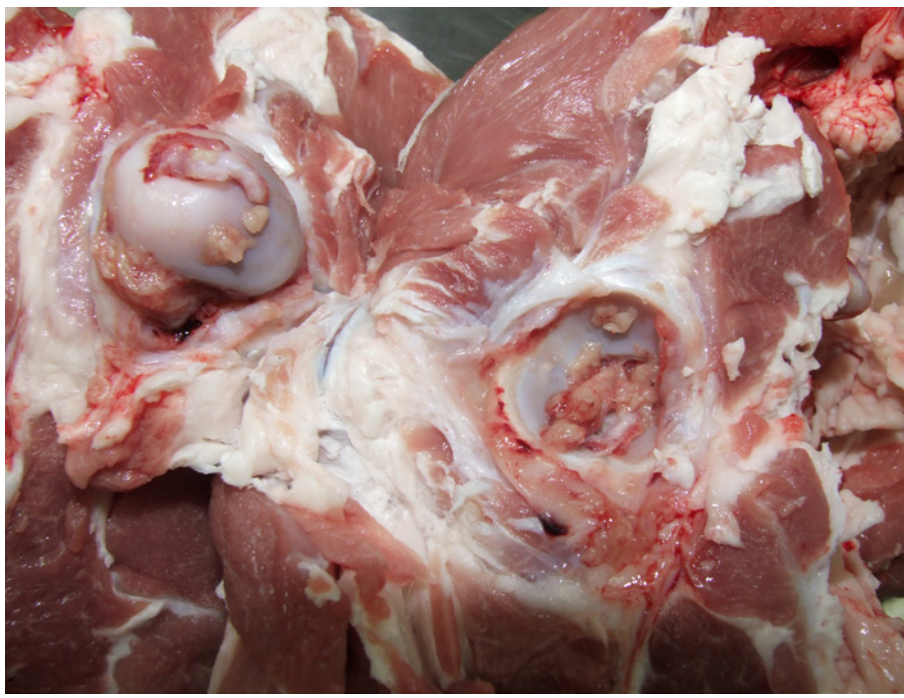
Arthritic joint with muscle removed



An incised arthritic joint, opened to expose pivotal surfaces. ***A*** *Breakdown of hyaline cartilage.* ***B*** *Pus development around tendon sheath.* ***C*** *Fibrinous deposit and swelling of synovial membrane.* ***D*** *Inflammatory response on pivotal joint*



Incised joint displaying inflammatory processes



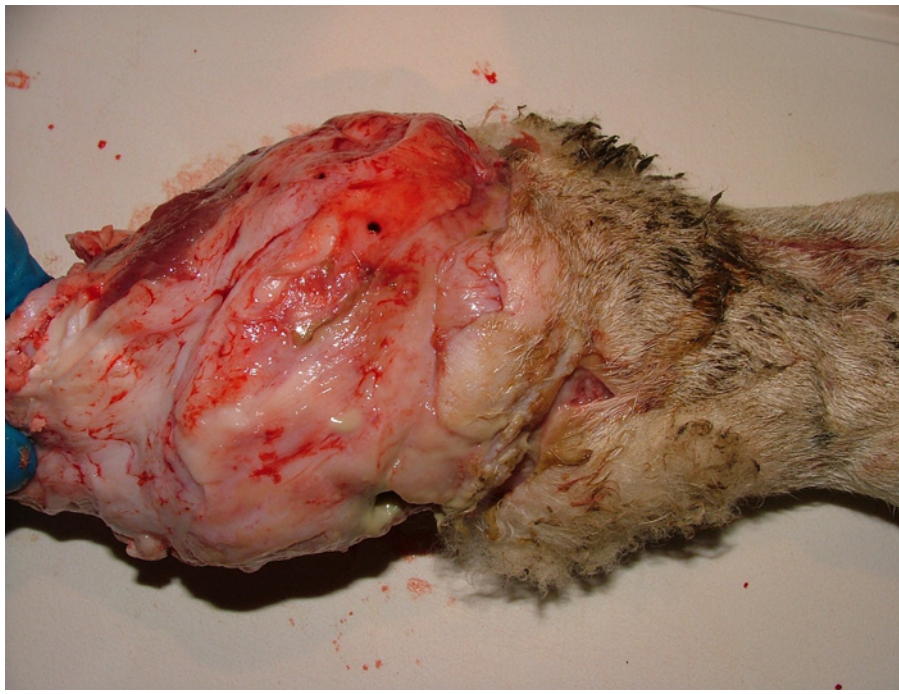
Inflammatory material in pelvic acetabulum

At post mortem inspection enlarged joints are opened and examined.

All cases of septic polyarthritis warrants rejection of the entire carcase and offal, as the infection is spread by the bloodstream and is said to be systemic. The synovial fluid resembles a 'beaten egg' and contains pus, blood and other turbid matter, and is associated with septicaemia, pyaemia or emaciation.

A lamb with an ankylosed joint. This occurs when the animal alleviates the pain from the joint by holding a leg position that offers the least discomfort. This can lead to either fibrous deposits or ossification of the joint that ultimately leads to permanent immobility.

In other forms of arthritis the rest of the carcase including non-suppurative polyarthritis due to *Erysipelothrix rhusiopathiae*, organs and lymph nodes are inspected for evidence of a generalised infection. Lambs affected by *Erysipelothrix rhusiopathiae* polyarthritis may develop vegetative endocarditis in later life. The affected area of inflammation should be trimmed if there are no signs of systemic involvement. The articular surface of the bone is commonly damaged.



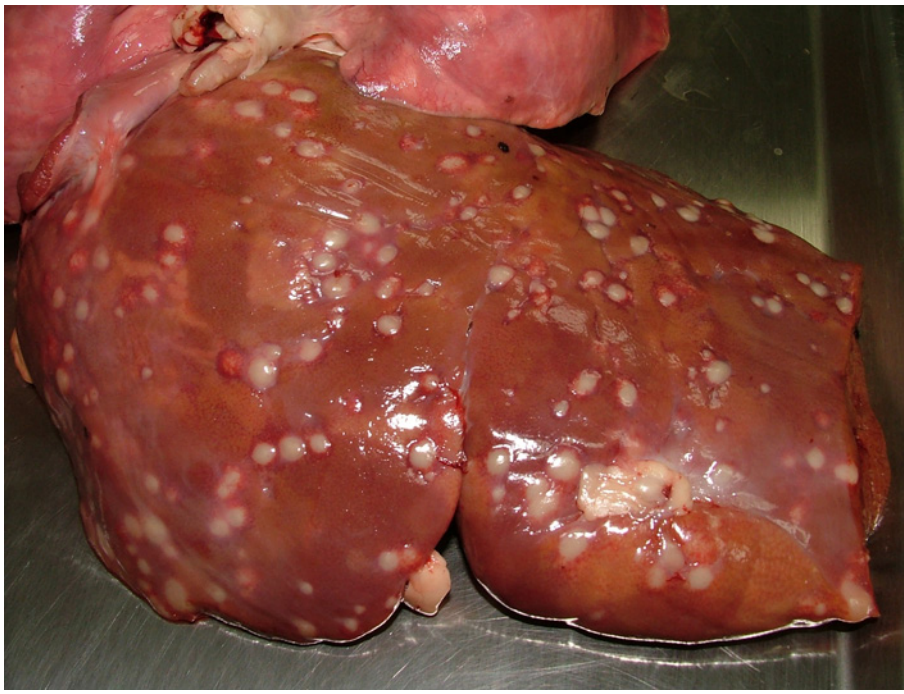
Purulent arthritis – venting to surface (vent at base)

BACTERIAL NECROSIS

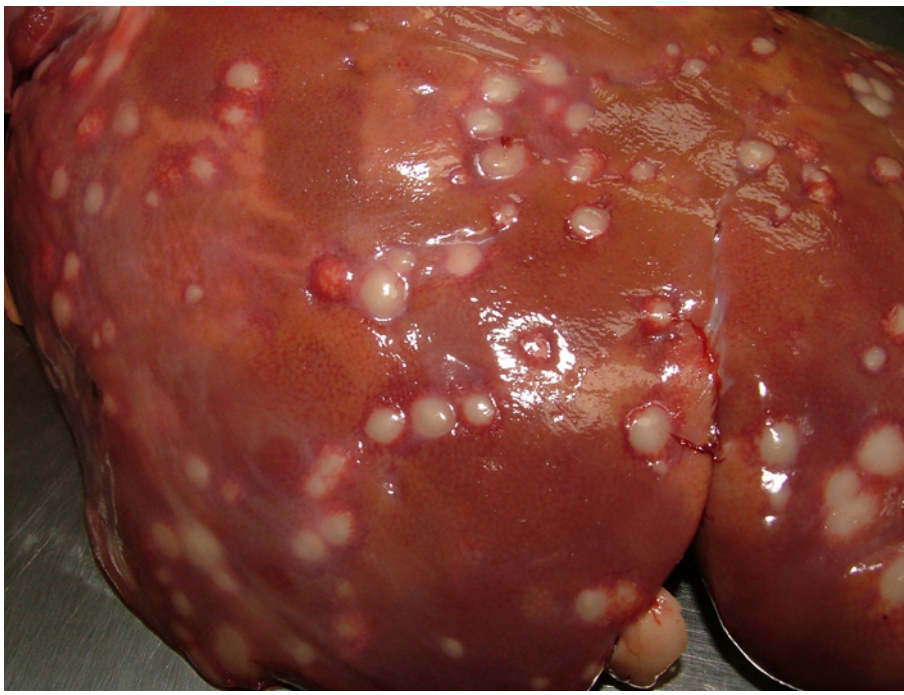
A term applied to micro-abscessation of the liver. Small pale, circumscribed necrotic lesions are found in the liver parenchyma, in an active infection these lesions are usually surrounded by an area of hyperaemia.

Although most likely due to an ascending infection from the intestines via the portal vein, the remainder of the carcase and offal should be checked for evidence of pyaemic spread. Ruminal acidosis can contribute to pathogenic bacterial growth that can utilise areas of acidic ulceration in the rumen to enter the bloodstream.

The liver should be rejected as unfit for human consumption.

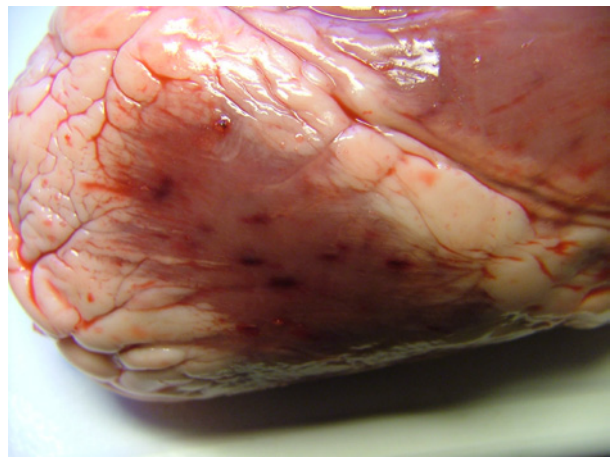


Bacterial necrosis

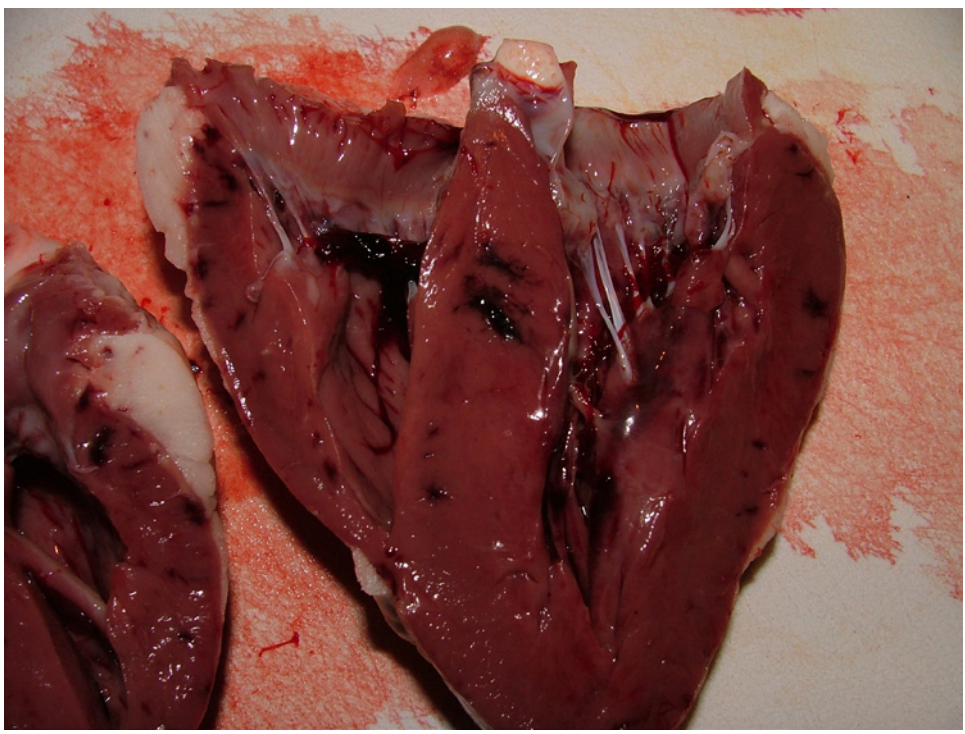


Bacterial necrosis – hepatic lesions circumscribed by a hyperaemic zone

BLOODSPLASH



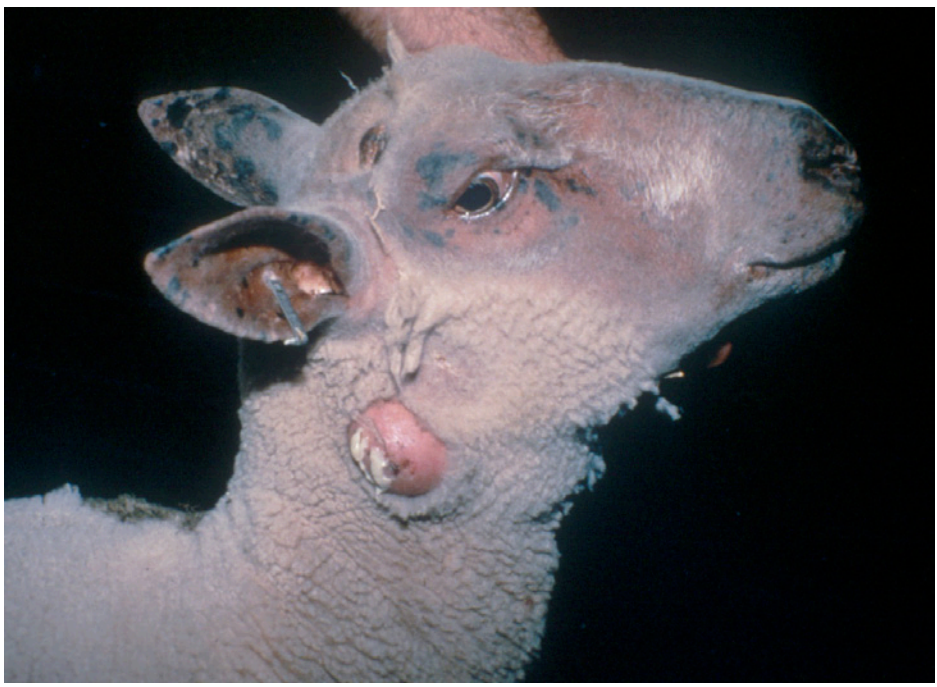
Small areas of haemorrhage throughout the organs, tissues and muscles of the body also known as blood speckle and spotting. It occurs during the slaughtering process. The application of an electric current, especially at 50Hz causes the muscles to contract antagonistically causing tearing of the capillary beds; blood vessel fragility and severe external pressure of the venous and arterial systems during electric stunning have all been mooted as causal factors. Most commonly affected are the heart and diaphragm, although the abdominal muscles may also be affected. Affected parts are trimmed and rejected.



Subepicardial and myocardial haemorrhage, indicative of bloodsplash, or more serious conditions such as septicaemic pasteurellosis

CASEOUS LYMPHADENITIS

This is a condition produced by *Corynebacterium pseudotuberculosis (ovis).* This is a pus forming bacterium that gains access to the body through superficial wounds such as those caused by shearing, docking and castration.

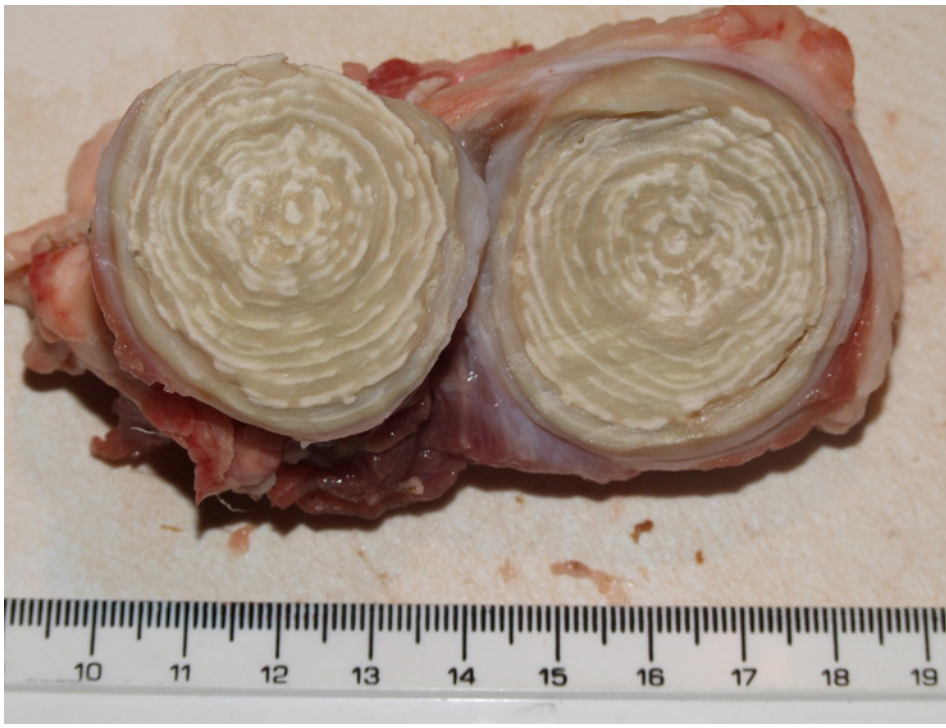


Caseous lymphadenitis – discharging parotid lymph node

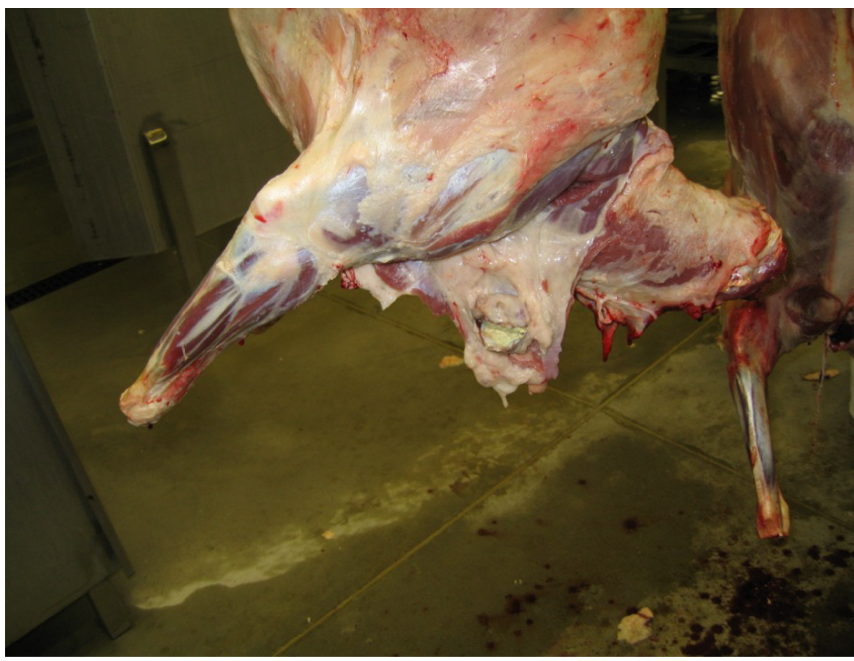
Unlike other bacteria *Corynebacterium pseudotuberculosis* has fatty substance on its cell surface that is toxic to the white blood cells (phagocytes) of the body's immune system. This enables the organism to survive inside the phagocyte as it is carried to the nearest lymph node. Here it kills the phagocyte and multiplies to form an abscess. More phagocytes are then infected, carrying the infection further down the lymphatic chain where further abscesses are formed.

At post mortem inspection the lymph nodes are enlarged and become filled with greenish, non-odorous pus. This pus quickly thickens, becomes caseous and sets in concentric rings like the layers of an onion. It is this laminated layering that is so characteristic of this condition.

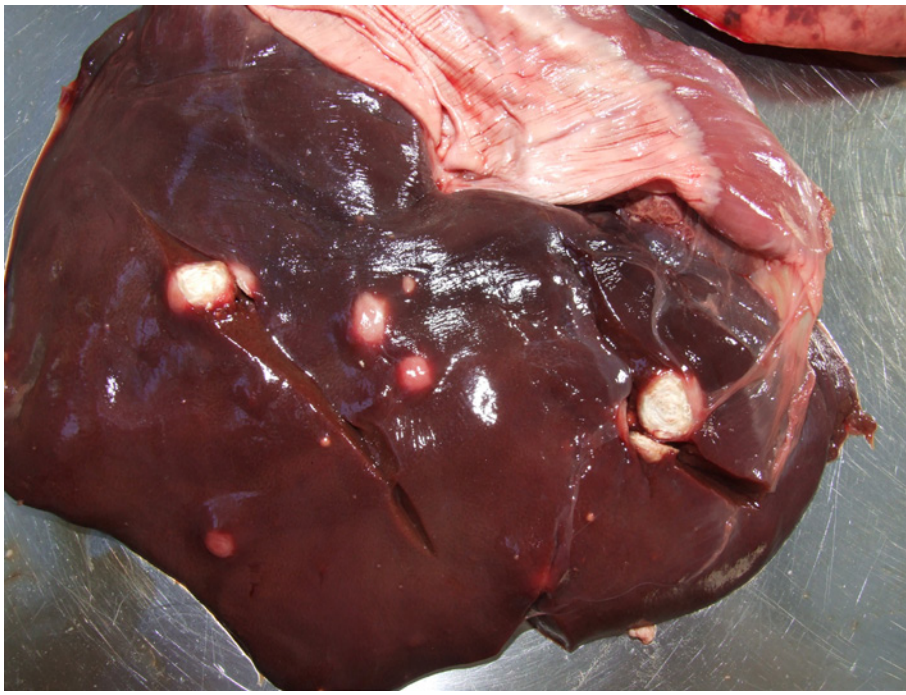
The abscesses can spread to the internal organs affecting lymph nodes in the lungs and stomach. Carcases with caseous lymphadenitis are rejected as unfit for human consumption. *Corynebacterium pseudotuberculosis* has zoonotic potential.



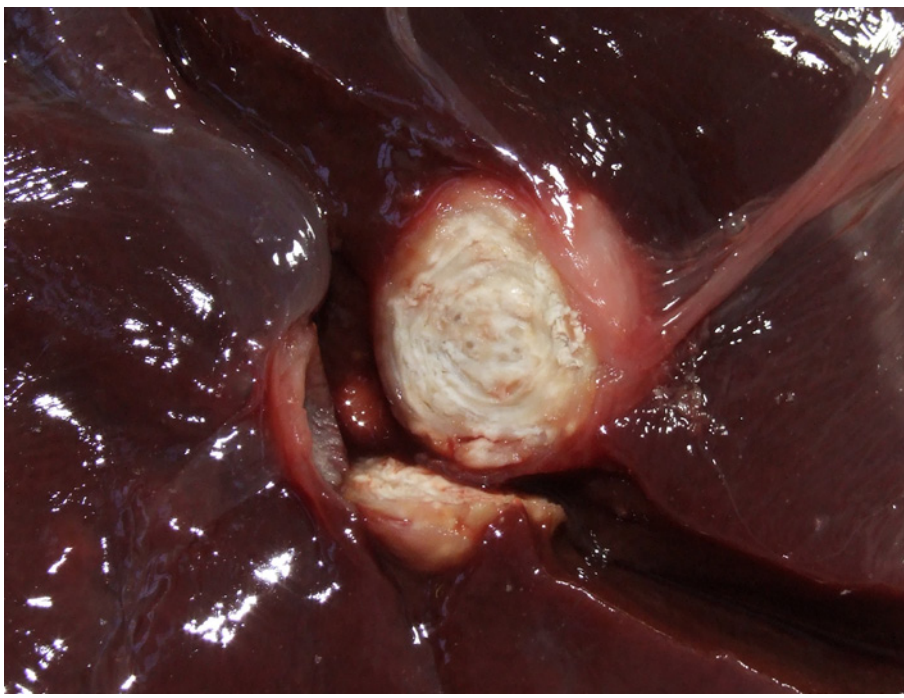
Laminar pus formation – a characteristic lesion of caseous lymphadenitis



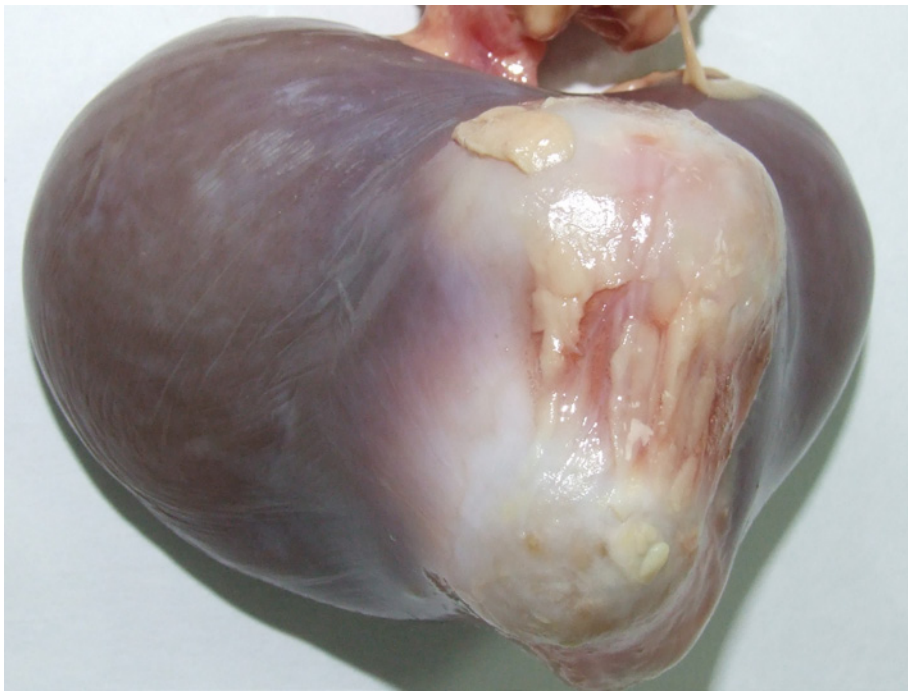
Caseous lymphadenitis – abscessation and enlargement of the prescapular lymph node



Caseous lymphadenitis – hepatic formations



Incised lesion – displaying characteristic lamination of *C. pseudotuberculosis*



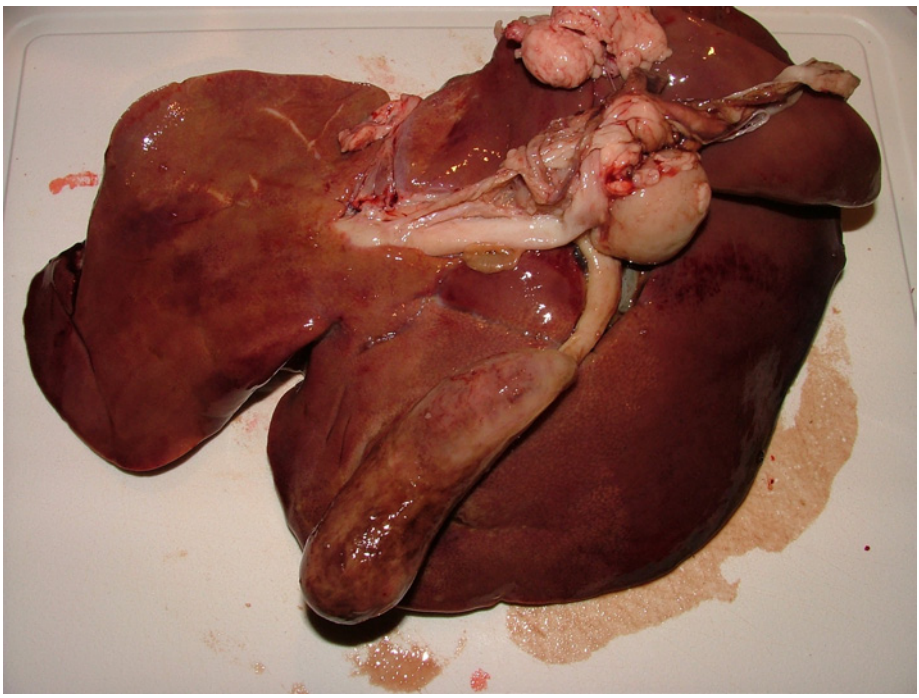
Caseous lymphadenitis lesions in a kidney

Incised lesion illustrating laminar pus

CHOLECYSTITIS

Cholecystitis is inflammation of the gall bladder. The most common cause brought to the attention of inspectors is the presence of *Fasciola hepatica*, that can produce the inflammatory response by their presence, by occluding the larger bile ducts leading to biliary stasis, or acting as transport vectors for bacterial pathogens.

However the most common causes of cholecystitis are biliary calculi (gall stones) and ascending infecting from the duodenum via the bile duct. Unfortunately the gall bladder is usually removed during the carcase dressing procedure and is not required to be inspected.



Cholecystitis due to Fasciola hepatica – note enlarged cystic duct and hepatic lymph node

Blockage of the common bile duct to the duodenum can lead to post hepatic jaundice and/or emaciation if bile is excluded from the digestive process. The liver should be judged on its' merits, if the bile ducts are affected the liver should be rejected which is also the case if the liver is affected with fluke.

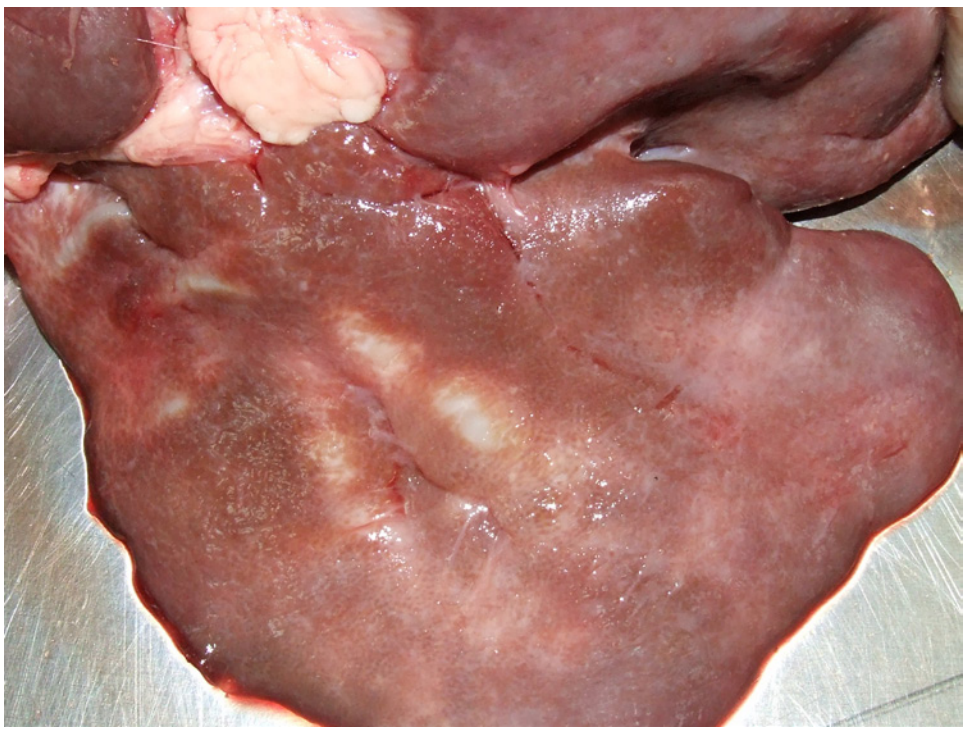
CIRRHOSIS

In cases of cirrhosis the normal hepatic parenchyma is replaced by fibrous connective tissue following an inflammatory process. Most commonly affected is the edge of the left lobe although any part of the liver may be affected in cases of chronic infection (including abscessation), the action of plant toxins, or parasitism. The presence of the fibrous connective tissue can produce lobulation.

Fasciola hepatica infection can produce pathogenic and physical cirrhosis and is considered a precursor to Black disease in which *Clostridium novyi* proliferates in the anaeorobic conditions within the cirrhotic areas of the liver producing an infectious necrotic hepatitis.

Cirrhosis of the liver can also lead to jaundice.

Affected livers are unfit for human consumption, although partial liver rejection may be possible if there are no signs of fluke, secondary infection or toxaemia.



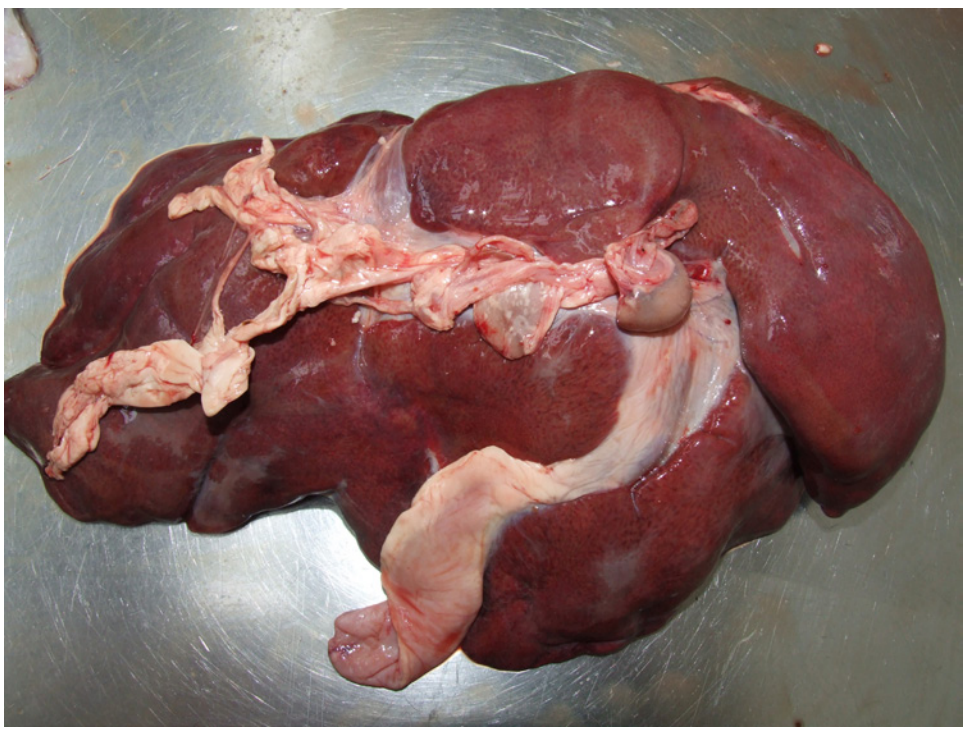
Cirrhosis of the left hepatic lobe due to acute fascioliasis

CONGESTIVE HAEMORRHAGIC HEPATITIS

A form of liver inflammation in which extensive haemorrhagic areas and congestion produces a dark red substance. The associated swelling of the liver can lead to a lobulated appearance and visceral peritonitis. In the United Kingdom this condition is commonly associated with extended ingestion of bracken, ragwort poisoning, or mycotoxicosis. Ragwort (*Senecio jacoboea*) is a plant that contains pyrrolizidene alkaloids that affect the liver and may also affect the abomasum. Mycotoxicosis can also occur due to ingestion of mould in stored feedstuffs.

If no other lesions are found only the liver need be rejected.

It is important in these cases to check the heart for signs of vegetative endocarditis; if this is present the congestive inflammation may be due to passive congestion and may warrant rejection of the carcase and offal as unfit for human consumption.

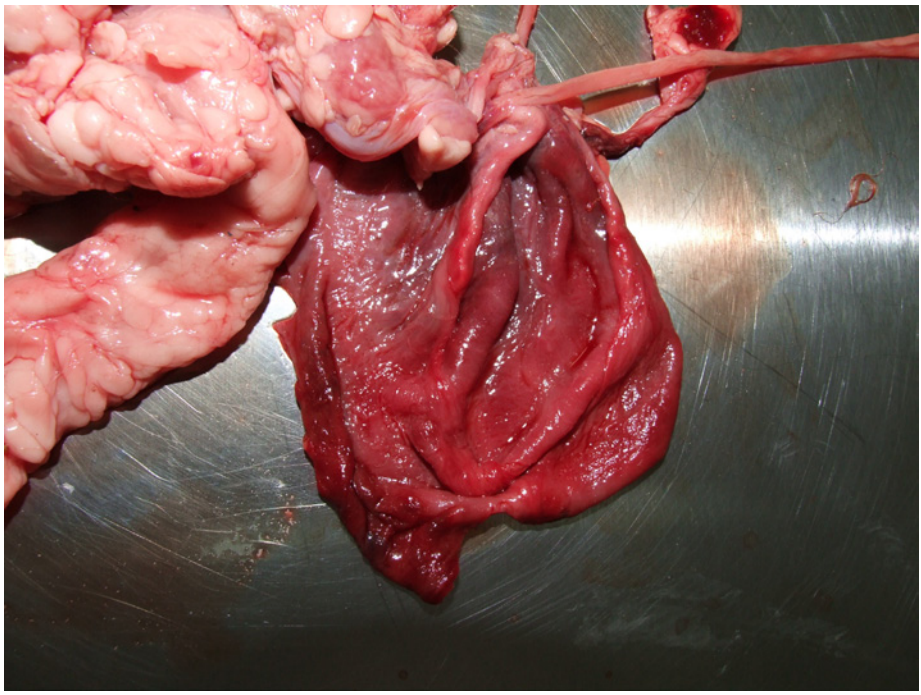


A congested, swollen liver

CYSTITIS

Inflammation of the urinary bladder can have a pathological or traumatic cause. Pathological infection can be either an ascending infection via the urethra or a

descending infection from the kidneys via the ureters such as *Corynebacterium renale*. Traumatic causes include renal and urethral calculi. Affected parts are rejected and the carcase checked for signs of the cause of the cystitis, as it is rarely an isolated condition.



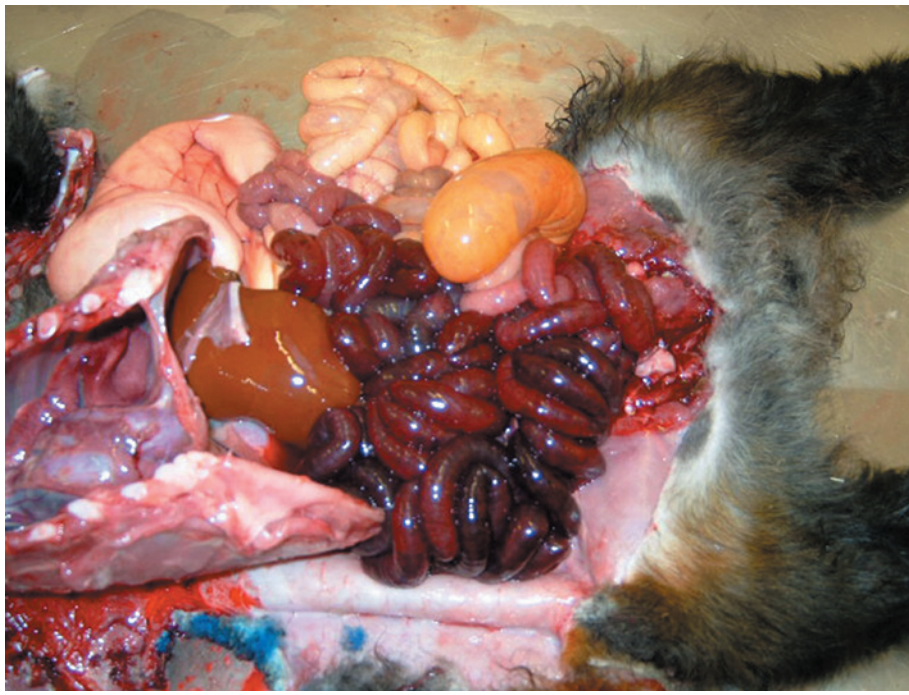
Haemorrhagic cystitis following urethral calculi in a ram

ENTERITIS

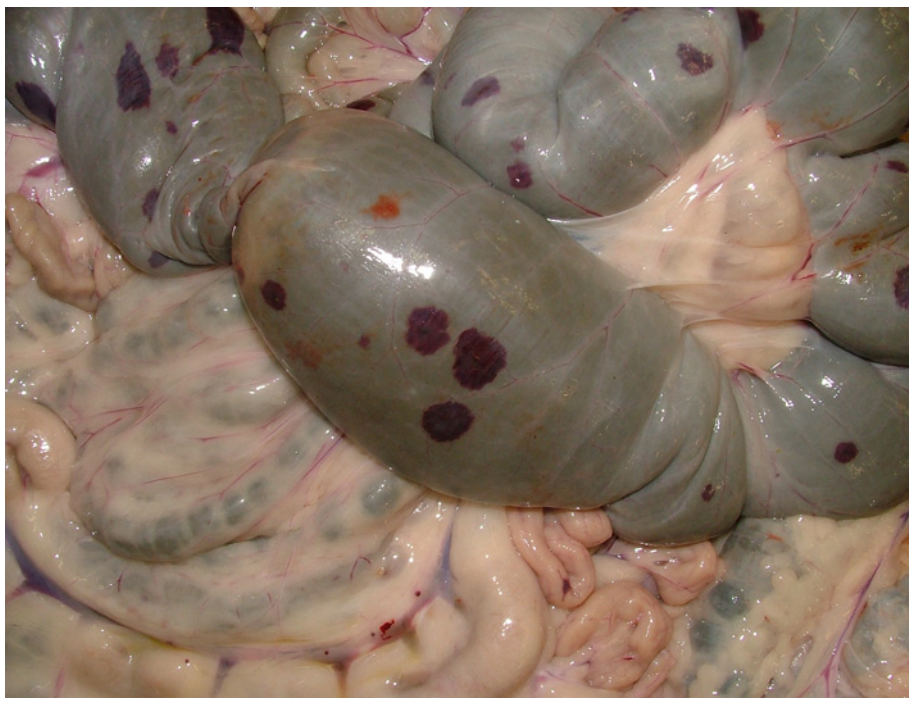
Enteritis is inflammation of the intestines and stomach. It can be either acute or chronic and occurs during the course of some diseases, as a result of the action of parasites or toxic agent.

In the live animal enteritis is characterised by diarrhoea.

At post mortem inspection, with acute enteritis, the stomach and intestinal contents are often blood stained. The mesenteric lymph nodes are enlarged and haemorrhagic. The liver shows signs of pathological change (fatty degeneration) and the gall bladder is often thickened. The lungs can be hepatised, the lung substance altering to give the appearance of liver, and petechiae are found in the kidneys.

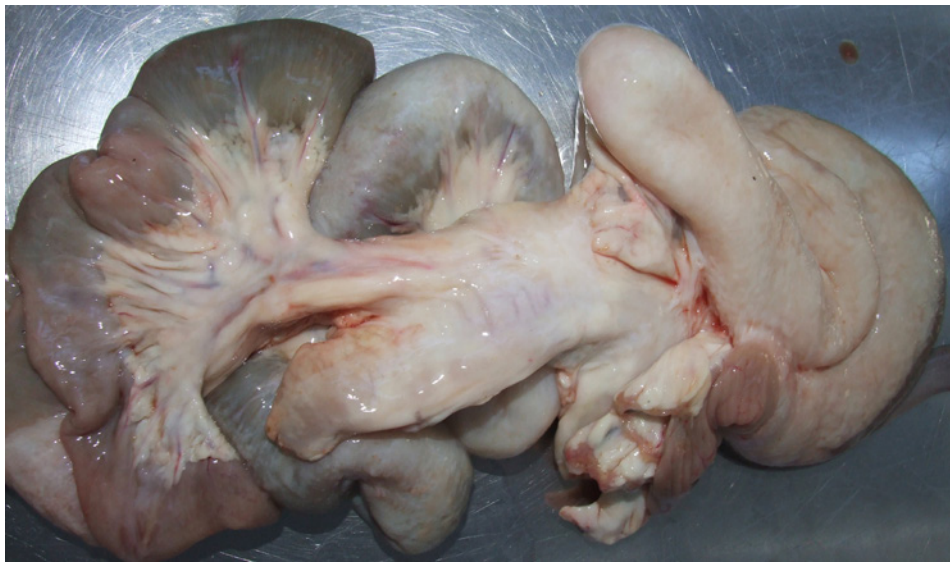


Acute enteritis



Focal haemorrhaging due to parasitic migration

With chronic enteritis there are areas of tissue death (necrosis), especially in the intestines at the caecum and colon. The bowel wall can be thickened with greenish yellow necrotic material. In cases of chronic enteritis the carcase is examined for signs of systemic infection.



Thickening of intestinal mucosa – onset of Jöhne's disease

If the carcase shows signs of acute enteritis, both the entire carcase and associated offal are rejected as unfit for human consumption.

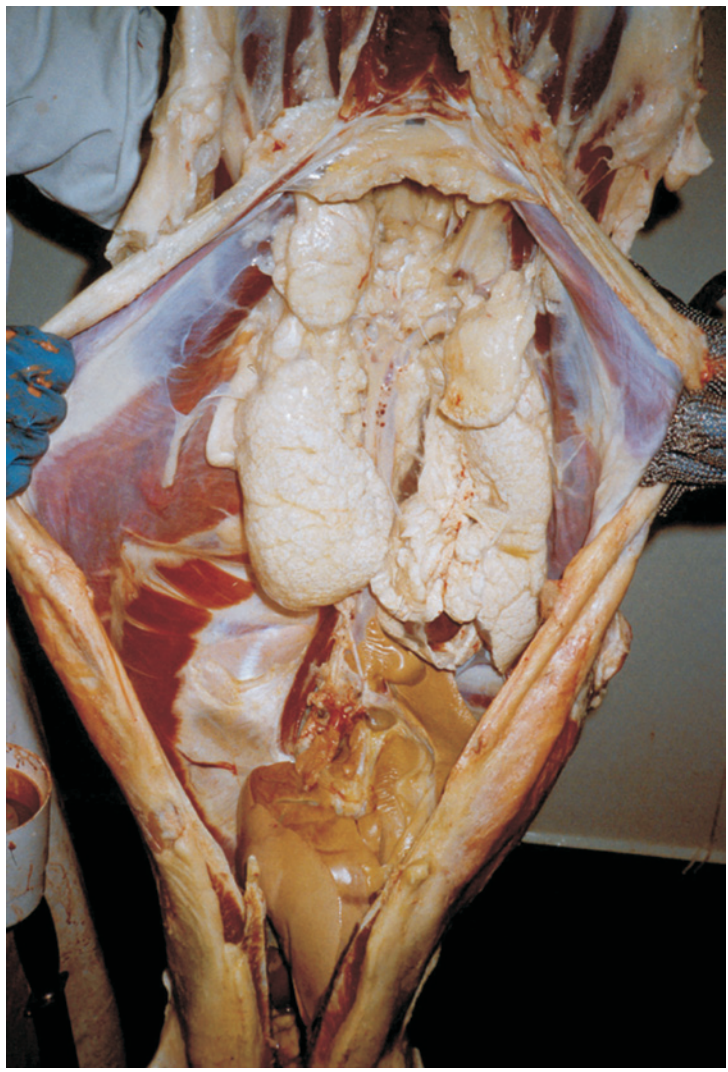
If the carcase displays lesions that indicate chronic enteritis the affected parts are rejected, unless evidence of systemic infection warrants rejection of the entire carcase and offal as unfit for human consumption.

FAT NECROSIS (LIPOMATOSIS)

Diffuse, hard, necrotic fat deposits may be found in the abdominal cavity. These can be due to physical trauma or the release of pancreatic enzymes either from an infected pancreas or from the gut itself. The enzymes release free fatty acids in the fat cells which combines with calcium. The destruction and necrosis of these fat cells can be accompanied by an inflammatory response. Clinically no indication may be seen unless the amount of firm necrotic tissue becomes obstructive either on the intestines or other organs of the pelvic region.

Affected areas are trimmed and affected fat rejected as unfit for human consumption. Conditions that produce rapid mobilisation of fat deposits due to acute energy deficit may also lead to lipomatosis including pregnancy toxaemia and are

usually associated with fatty degeneration of the liver, If generalised throughout the carcase, possibly due to a chronic pancreatitis, the entire carcase and associated offal should be considered unfit for human consumption.



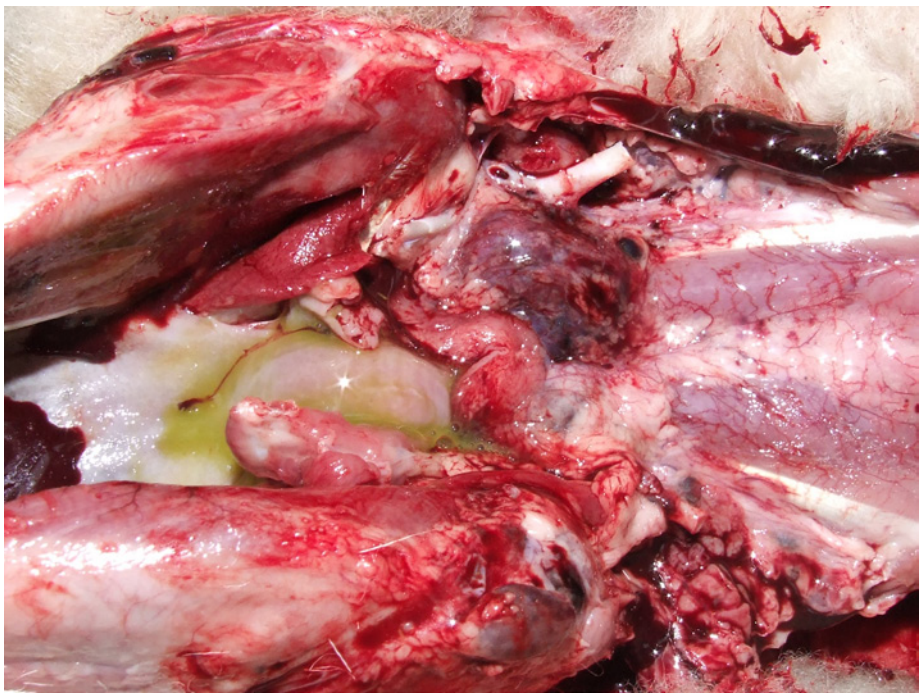
Fat necrosis in the abdominal cavity. Note also fatty, friable liver.

FEVER

This encompasses fever, toxaemia and septicaemia. The carcase is fiery in appearance, has usually bled badly, the carcase lymph nodes are enlarged and have undergone change.

TOXAEMIA

This is the presence of bacterial toxins in the bloodstream of the animal. If these toxins have produced generalised and systemic changes within the carcase it is considered unfit for human consumption and is rejected. These changes may take the form of cloudy swelling or fatty change in the liver, kidneys or heart muscles.



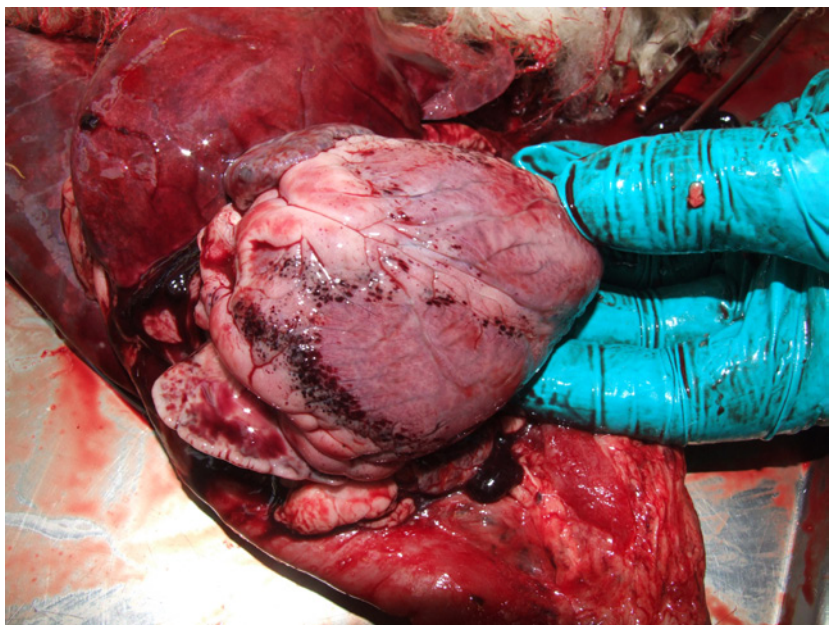
Petechial haemorrhages in the retropharyngeal lymph nodes – pasteurella septicaemia

SEPTICAEMIA

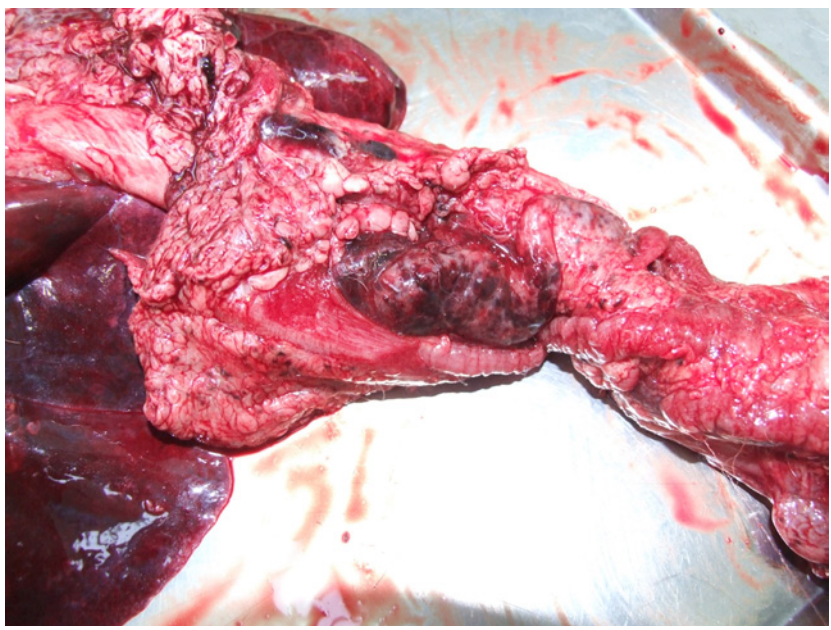
This is the presence and multiplication of bacteria in the bloodstream of the animal. This differs from the condition bacteraemia in that the organisms are widely distributed as is the subsequent tissue damage, and the organisms are not only present in the blood but are actively multiplying therein. The affected carcase is congested, bleeds badly and rigor mortis is slight or absent. Small haemorrhages known as petechial haemorrhages are present in organs such as the kidneys. The lymph nodes are enlarged; the liver and kidneys can undergo pathological change. Vascular damage leads to petechial haemorrhages throughout the carcase. Fevered, septicaemic and toxaemic carcases are rejected as unfit for human consumption. The congestion of the carcase affects the keeping quality of the meat, rendering it unmarketable, but more importantly the causal organisms may be pathogenic, which would render the consumption of the carcase dangerous to the consumer. Therefore in cases where

the carcase displays signs of the above conditions the benefit of doubt must always be given to the consumer.

Studies have shown that flesh from animals suffering from generalised septic conditions, especially those of the intestinal tract, are most likely to give rise to gastro-intestinal disturbances in man.



Pasteurella septicaemia - Sub-epicardial haemorrhages



Pasteurella septicaemia - Petechial haemorrhages in the thymus

GANGRENE

In meat inspection, gangrene denotes the invasion of dead (necrotic) tissue by pus forming bacteria. It can occur by means such as bacterial infection of a wound, or as a secondary infection. Gangrenous tissue is soft, swollen, foul smelling and dark or greenish in colour. The bacteria cause damage both by direct invasion and rapid multiplication and by the actions of their toxins.

Braxy is caused by the bacterium *Clostridium septicum*. Well-nourished sheep die suddenly after eating frosted grass. The abdomen distends rapidly and starts to decompose. The walls of part of the stomach (abomasum) shows dark patches of black haemorrhage that can be seen without opening the organ. Straw coloured fluid can be found in the chest and abdominal cavities. Other organs may show signs of toxaemia.

Malignant oedema is caused by the same bacterium and is associated with wound infection. The damaged area becomes rapidly swollen and pits on pressure, the muscles and tissues are oedematous and gas may be produced.

Black disease affects the liver (infectious necrotic hepatitis) and is caused by secondary infection of the liver after immature liver fluke have migrated through the tissue. As well as areas of necrosis in the liver, the skin of the animal is normally bluish black and the underlying tissues are oedematous and congested. Normally caused by *Clostridium novyi*.

Black-leg, black-quarter, is caused by *Clostridium chauvoei* and is characterised by swellings containing gas that increase rapidly in size and crackle if pressed.

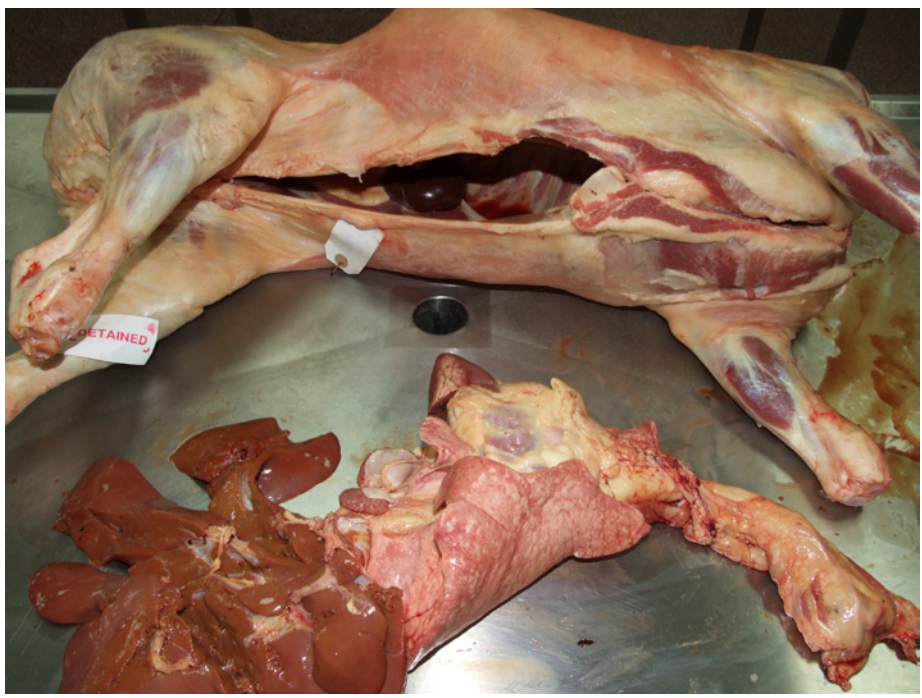
At inspection any affected organ is rejected as unfit for human consumption. If evidence of toxaemia or septicaemia is present the carcase and associated offal are rejected. Owing to the rapid nature of these diseases most animals will die within hours of the onset of infection.

JAUNDICE

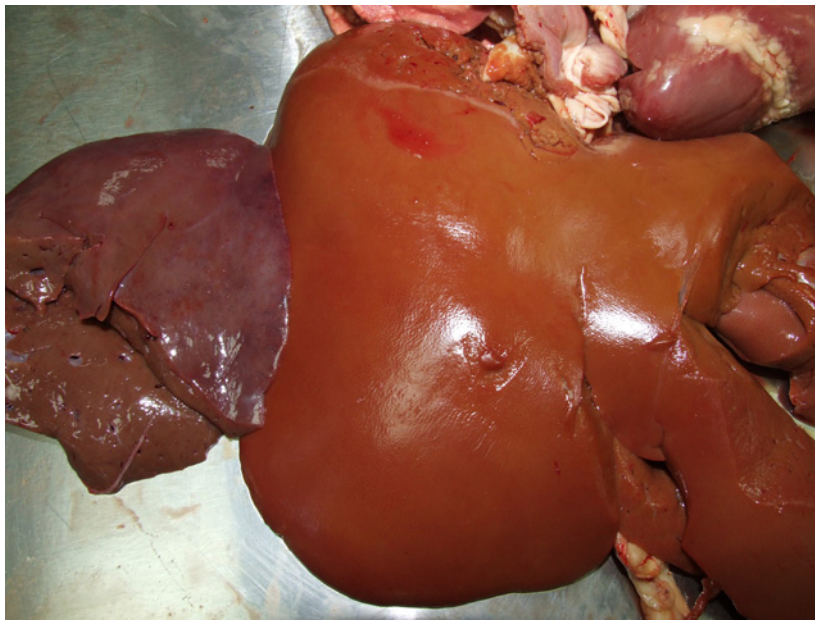
ICTERUS

Jaundice (or icterus) is displayed at post mortem inspection as a yellow discolouration of carcase tissues. It is caused when the pigmentary constituents of bile gain access to the blood circulation and are then deposited throughout the body. This can be caused by cirrhosis of the liver, through blockage of the bile ducts of the liver by fluke; calculi (stones); or tumours and specific diseases. As such, icterus may be hepatic or obstructive. In hepatic icterus the cause is disruption of hepatic cells through disease or poisoning. Obstructive jaundice occurs when the flow of bile into the duodenum is disrupted by factors such as fascioliasis of the liver, gallstones and cholangitis (inflammation of the bile ducts). The live animal is out of condition, the eyes, inside of the mouth and other mucous membranes are tinged yellow.

At inspection jaundice is differentiated from 'yellow fatted sheep' (where discolouration occurs as a result of an inability to break down components in grass). This differentiation can be achieved using the Rimmington and Fowrie test but in practice examination of cartilage can assist in the decision. In jaundice the cartilage and tendon sheaths will also be coloured yellow. A cut made across the xiphoid cartilage at the end of the sternum will be white in yellow fatted lambs, yellow in cases of jaundice. A jaundiced carcase will be rejected with all associated offal as unfit for human consumption.



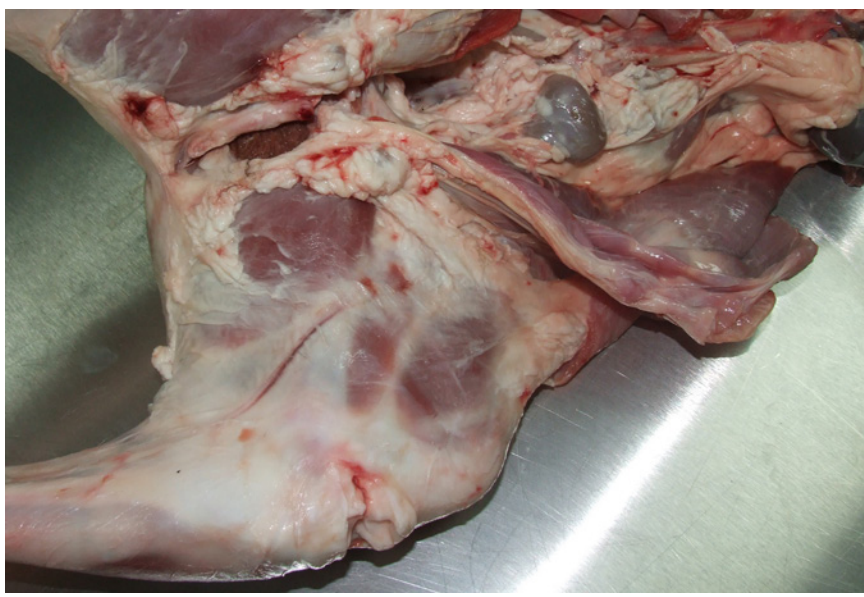
Detained jaundiced carcase and offal



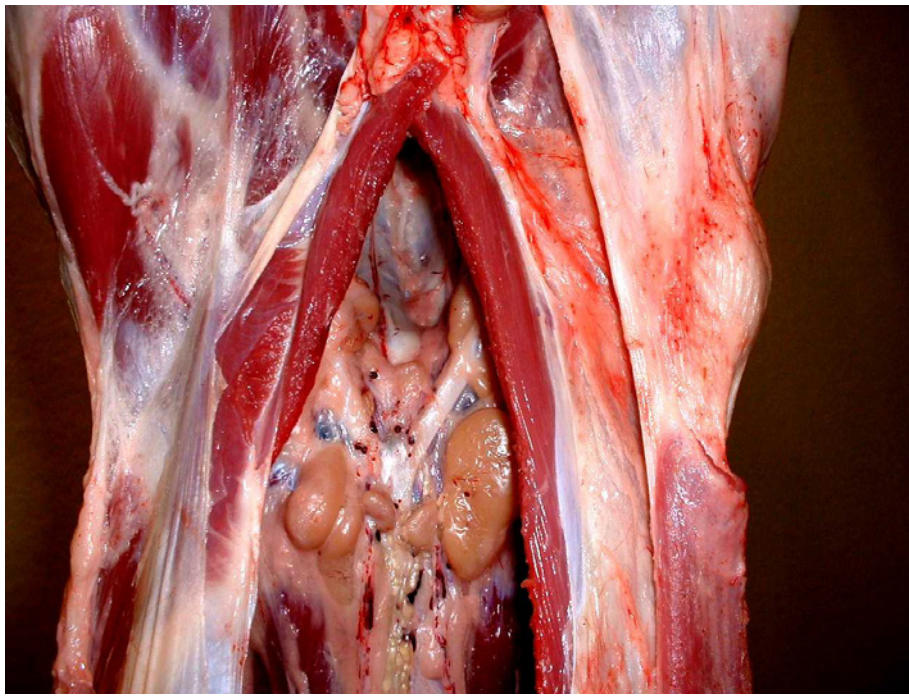
Liver discoloration compared to normal portion

LYMPHADENITIS

Inflammation of the lymph nodes, part of the bodies' immune system, sometimes mistakenly referred to as glands. Enlarged lymph nodes are indicative of infection in the area they drain. Lymph nodes can become locally enlarged in cases of arthritis; external lymph nodes can become enlarged in cases of sheep scab. Carcases in which all the lymph nodes are enlarged, congested and oedematous are rejected as evident of a systemic infection.



An enlarged iliac lymph node due to arthritis in rear leg

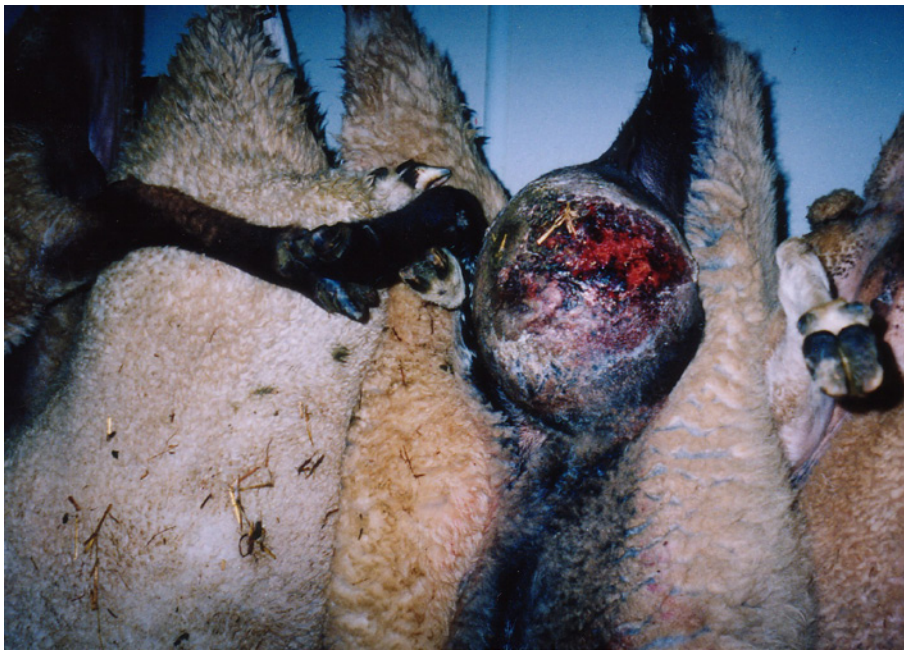


Inflammation of the internal and external lymph nodes

MASTITIS
Blue bag, Hard bag, Udder clap

Mastitis is inflammation of the udder. If encountered the udder is rejected. The main importance of mastitis is that the normal causative organisms of peracute gangrenous mastitis, *Pasteurella haemolytica* and *Staphylococcus aureus*, are pus-forming bacteria. If these bacteria are not successfully contained within the udder, access can be gained to the rest of the animal; a scenario made more likely by the high vascularisation of the organ, resulting in secondary abscess formation, pyaemia and septicaemia all of which warrant rejection of the entire carcase and offal as unfit for human consumption.

The formation of granulomatous lesions containing pus in the mammary gland tends to be due to the action of *Staphylococcus aureus*. Another common finding in sheep is superficial lesions of orf (contagious ecthyma or contagious pustular dermatitis) spread by direct contact with the mouths of affected lambs.



Acute mastitis evident on a carcase suspended after slaughter

Septic mastitis can lead to gangrene.

MELANOSIS

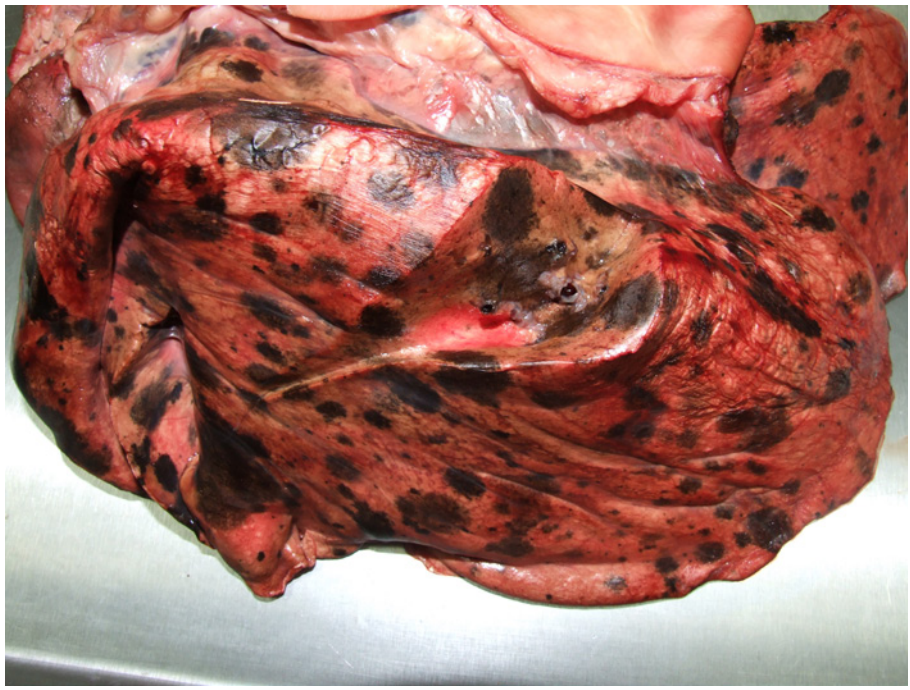
Melanin is a normal protein based pigment of the body, which colours the horns, hair, palate etc of sheep.

It can occur in abnormal amounts within the body, especially tissues such as the lungs, kidneys and spinal cord. These deposits are jet black, varying in size and shape and normally resemble ink splashes.

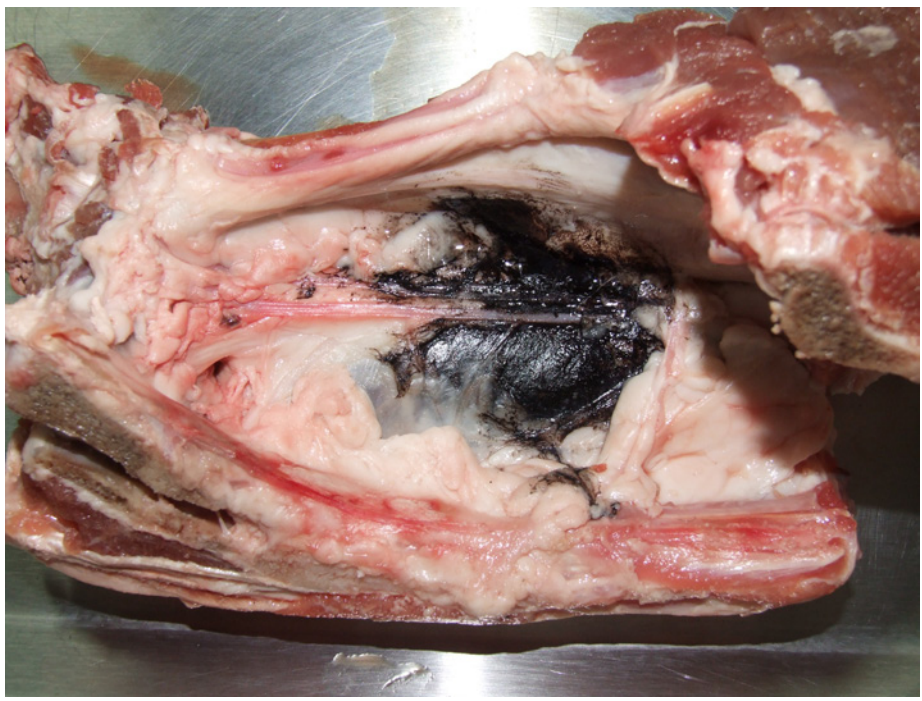
Melanosis is congenital and the deposits are laid down early in the development of the foetus.

Affected parts are rejected for aesthetic reasons.

If the pigmentation is generalized throughout the muscular tissue the entire carcase is rejected.



Melanosis of the lung



Melanosis – deposition in pelvic channel

METRITIS

Metritis is inflammation of the uterus, and generally occurs as one of three types:

Septic metritis is a bacterial infection and normally results from problems during birthing, such as retention of the placenta, rupture or injury to the uterine mucous membrane, and, very rarely, due to the decomposition of the foetus before birth.

The live animal suffers high fever, increased pulse rate, muscular weakness and frequently the inability to rise.

At inspection the uterus is enlarged up to three times normal size and is full of foul smelling chocolate or greyish fluid. The internal membrane of the uterus is thickened and discoloured, and the external surface may exhibit peritonitis. Toxaemia can be produced within the carcase, with enlargement of the lymph nodes.

Carcases showing lesions indicating acute septic metritis are rejected as unfit for human consumption.

Chronic endometritis is characterised by a purulent vaginal discharge but no signs of fever or physiological disturbance. The uterus is rejected.

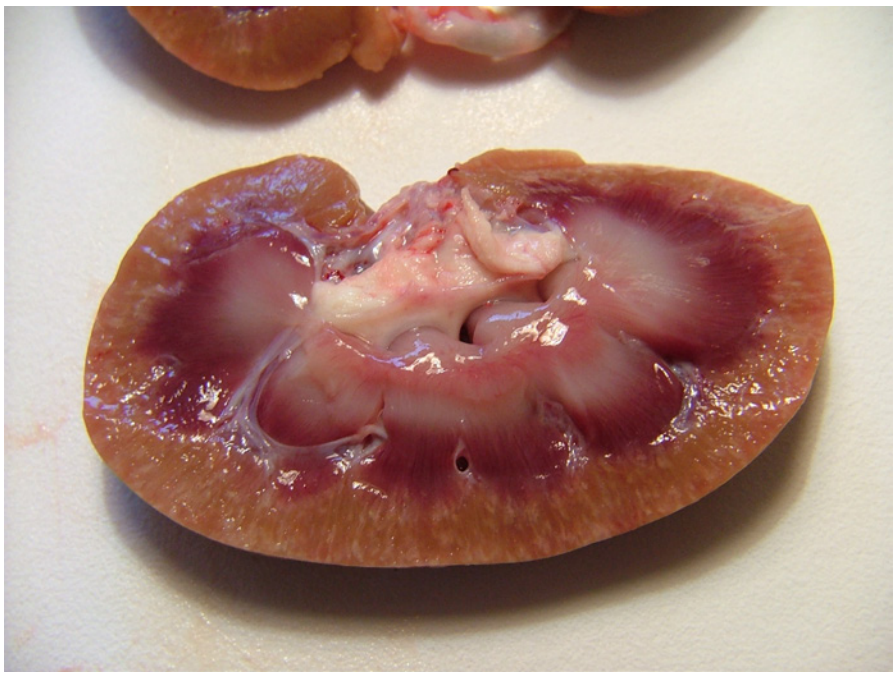
Pyometra (pus in the uterus) occurs without systemic infection of the carcase. The uterus is rejected, although in chronic form this can lead to emaciation of the carcase that could warrant the rejection of the entire carcase and associated offal.

NEPHRITIS / NEPHROSIS

Nephritis denotes inflammation of the kidney. Nephrosis denotes the change of state of the kidney.

In sheep the kidneys are bean shaped and smooth. Their basic function is the excretion of waste materials from the blood stream and the maintenance of the blood water proportions. Urine is passed through two ureters to the bladder. As the structure of the kidney is basically made up of filtration systems (nephrons) the kidneys are susceptible to infection from various sources and are examined closely at inspection.

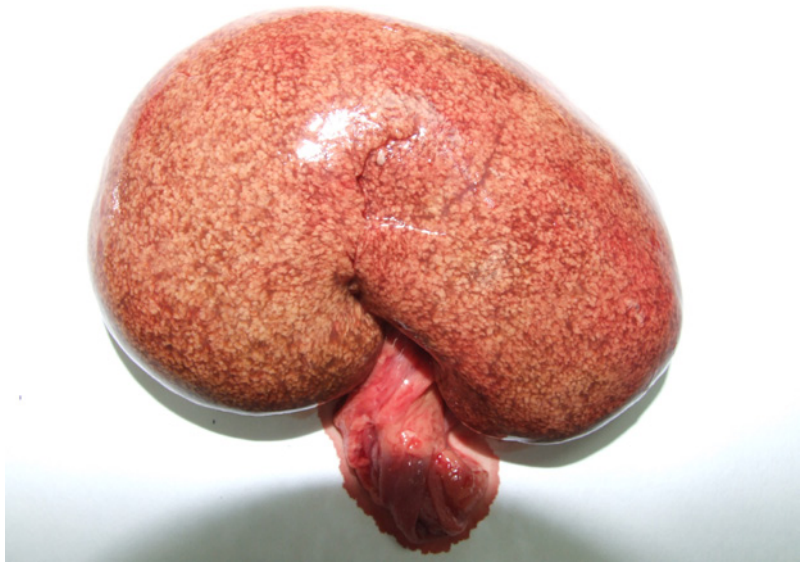
Interstitial nephritis



Interstitial nephritis – kidney incised

GLOMERULONEPHRITIS
Glomerulonephropathy

Glomerulonephritis denotes inflammation of the capillary 'ball' encased in the malpighian capsule of the kidney that forms the individual nephrons, or filtration units of the kidney. Lobar glomerulonephritis affects the entire kidney and if bilateral can lead to uraemia. Affected kidneys should be rejected.

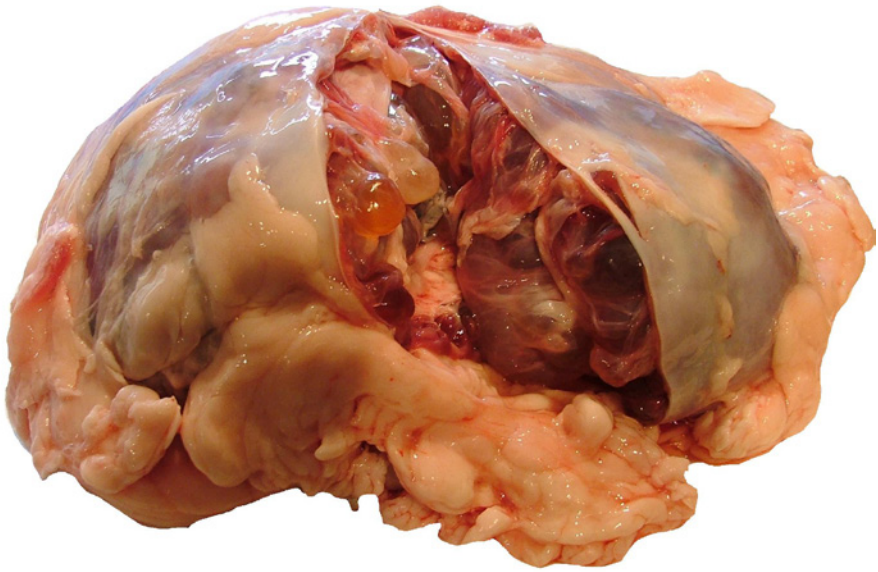


Lobar glomerulonephritis

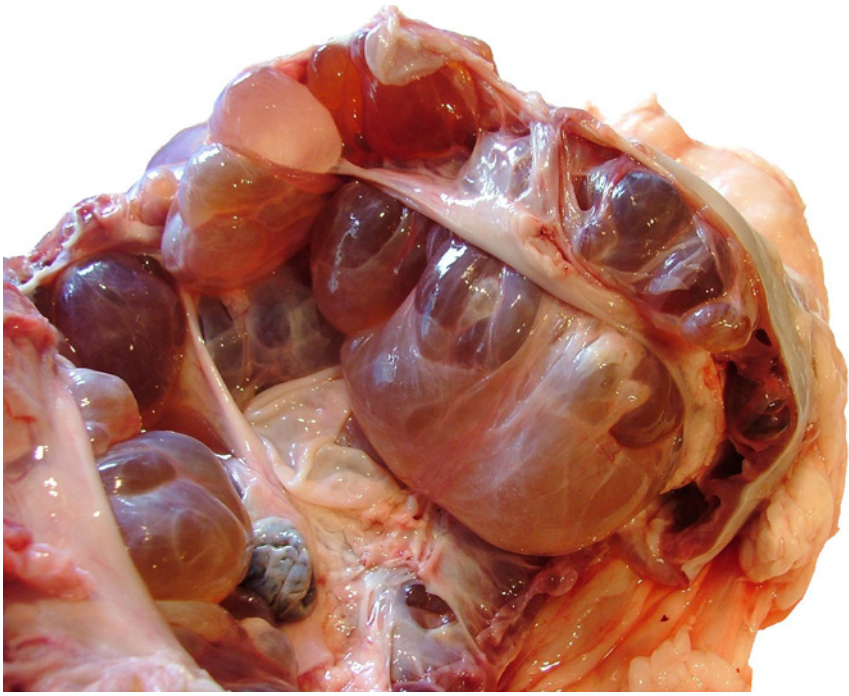
HYDRONEPHROSIS
Obstructive Uropathy

Occurs as a physical or infectious blockage of one or both ureters preventing urine from draining to the bladder. The retained fluid and increased hydrostatic pressure leads to destruction of the kidney tissue, which can be further exacerbated by ischaemic necrosis as a sequel to pressure occlusion of blood vessels within the kidney. Pressure atrophy of the kidney tissue begins in the renal pelvis, followed by destruction of the medullary papillae and finally the renal cortex. Generally when encountered at post mortem the condition is unilateral as bilateral hydronephrosis causes the rapid death of the animal.

If unilateral the affected kidney is rejected, the unaffected kidney may undergo compensatory hypertrophy. If bilateral the carcase is examined for uraemia and may be rejected as unfit for human consumption. The affected kidney is rejected.



Hydronephrosis in an ovine kidney



Detail of incised kidney. Note cyst formation and destruction of kidney tissue

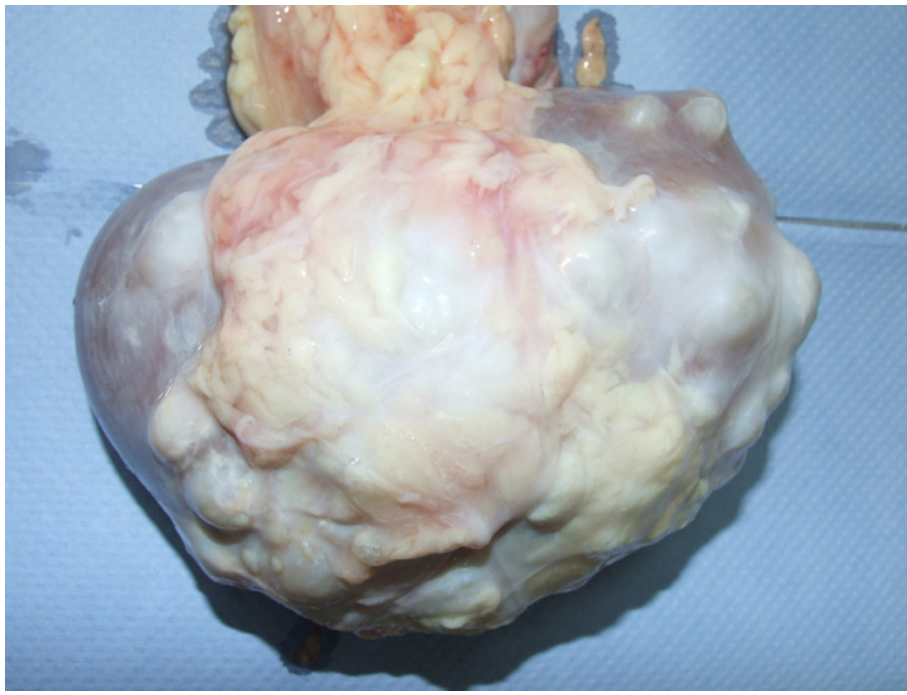
HAEMOGLOBINURIC NEPHROSIS

This condition occurs when the kidneys are pigmented following release of haemoglobin from blood cells. This haemolysis can occur in cases of babesiosis, enterotoxaemia and most commonly in cases of copper poisoning. The kidneys should be rejected and the carcase examined for signs of systemic involvement, including haemolytic jaundice.

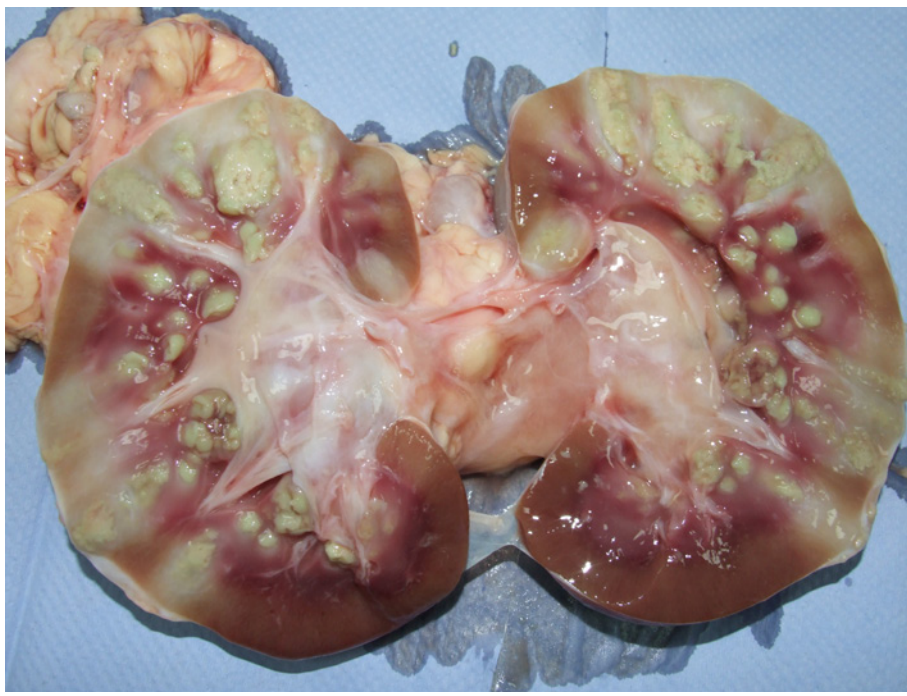
Haemoglobinuric Nephrosis

NEPHROPYELITIS
Pyelonephritis

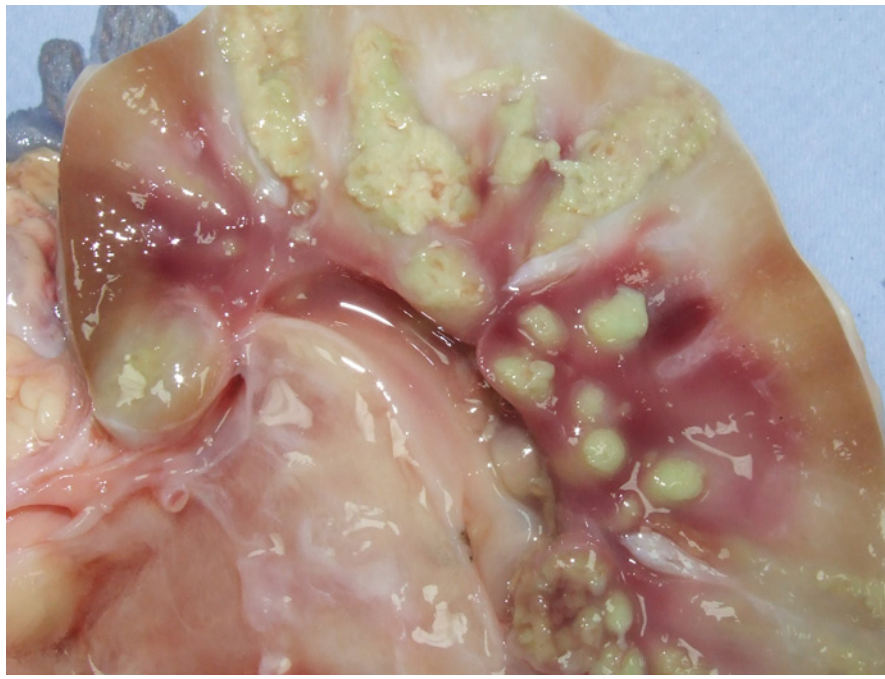
Abscess or pus formation occurs when pyogenic organisms localise in the kidneys either via the bloodstream (Pyaemic nephritis) or from a rising infection from the bladder (Pyelonephritis); in the latter case *Corynebacterium renale* is the most common cause. If one side is affected (unilateral) the affected organ is rejected after examination of the rest of the carcase (in cases of pyelonephritis the urinary system should also be rejected and the carcase checked for signs of uraemia). If both kidneys are affected (bilateral infection) the carcase may be rejected depending on evidence of a systemic infection (abscesses in other organs indicating metastatic spread).



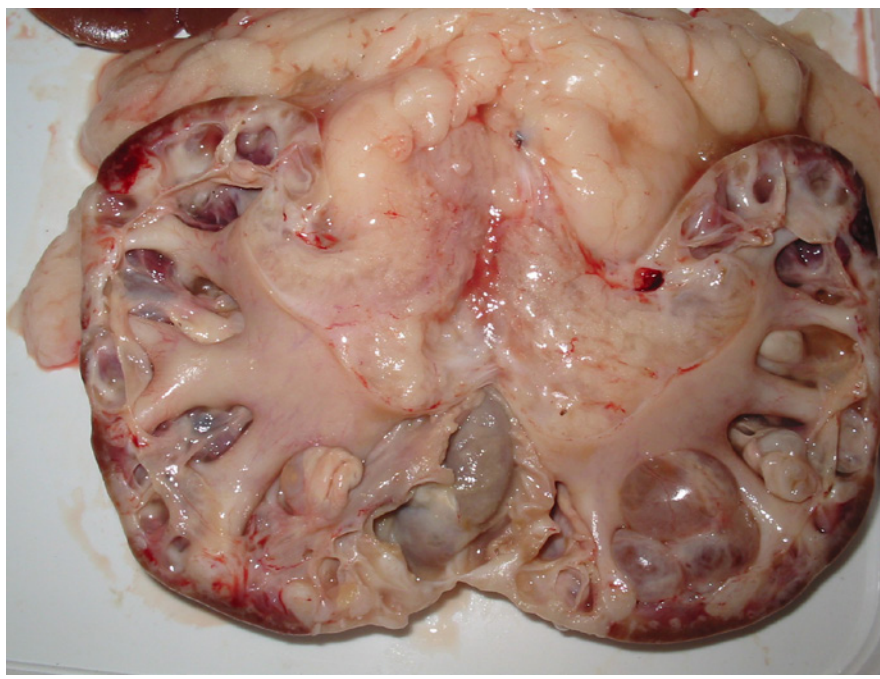
Multiple abscesses evident through the kidney capsule



Incision through kidney above, multiple abscesses, carcase was rejected for pyaemia as it displayed purulent pneumonia, pyaemic nephritis and polyarthritis



Detail of infection, displaying inflammatory response and blockage of capillary beds leading to infarction of the cortical region



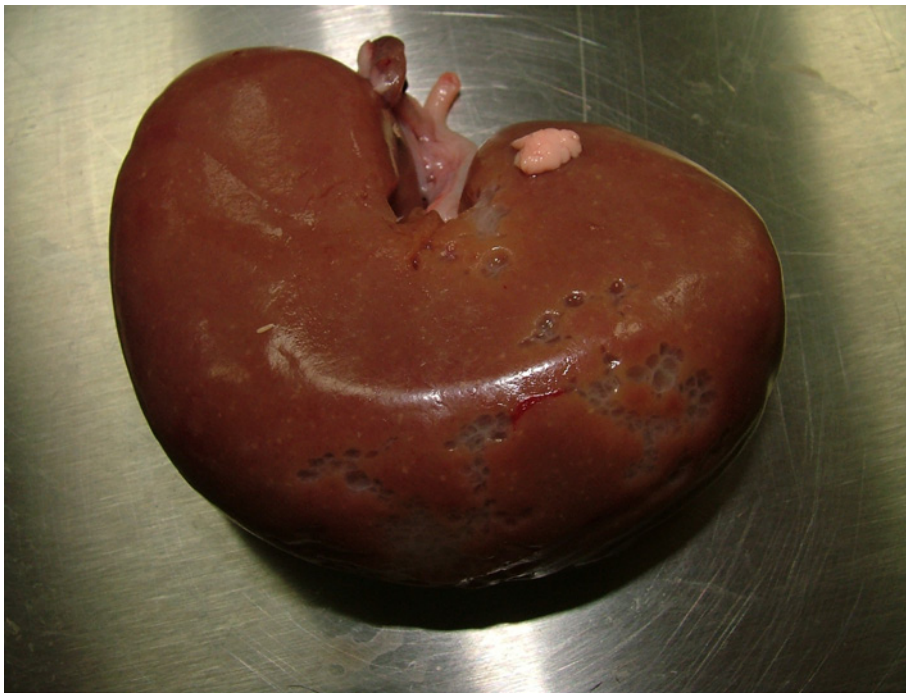
An incised kidney displaying pyelitis; a distended renal pelvis and cortical destruction produced from an ascending urinary tract infection

RENAL RETENTION CYSTS

GENETIC CYSTS

These cysts tend to affect only one kidney. They are not felt to be due to a pathological cause but an autosomal recessive congenital defect in the tubule supplying the affected area. The kidneys have a huge functional reserve and clinical signs of renal failure only become apparent when 75% of all kidney tissue has been damaged. As such the unaffected areas are considered fit for human consumption. These cysts are often accompanied by hypertrophic growth in the remaining kidney tissue.

A polycystic kidney –a congenital condition. In this case with over 70% of the kidney affected it is unlikely to be a functional unit. The other kidney had undergone compensatory hypertrophy



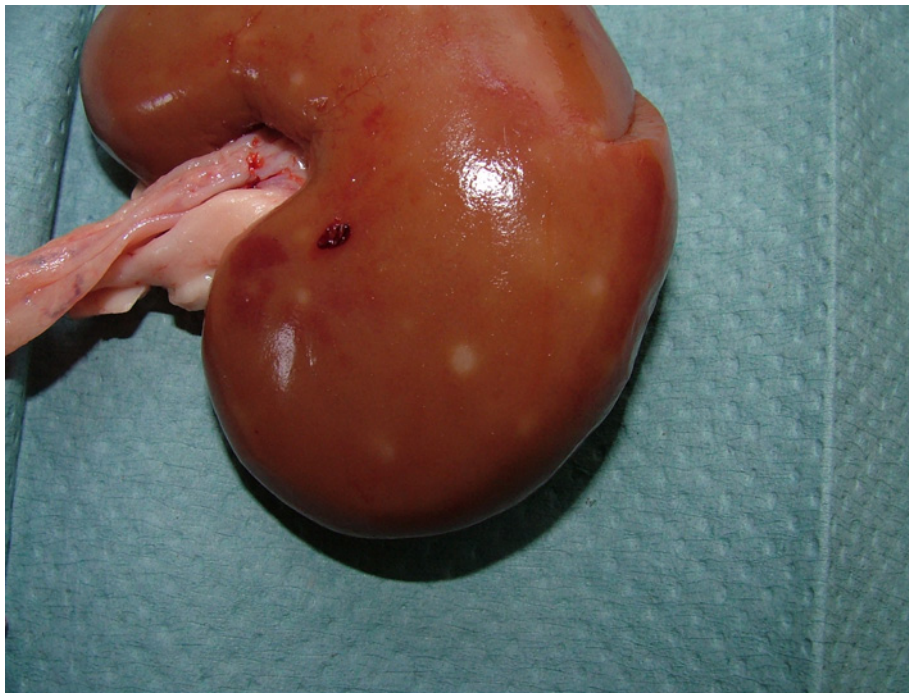
Polycystic kidney

RENAL INFARCTION
White spotted kidney, Tubulonephrosis

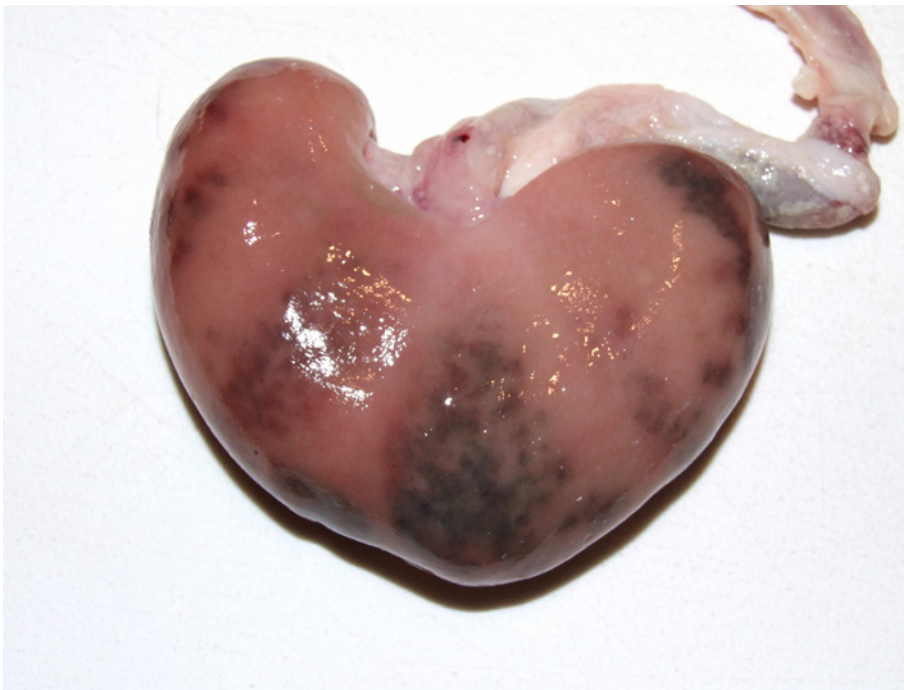
The kidneys are prone to infarction, whereby the arterial supply, or venous drainage to the cortex, is obstructed by an embolus or thrombus resulting in the death (necrosis) of the cells supplied by that blood vessel.

In the kidney these appear as pale spots on the surface and have a characteristic cone shape when the kidney is incised. Occasionally the infarcts may become haemorrhagic due to leaching of blood cells into the area. These kidneys are rejected as unfit for human consumption.

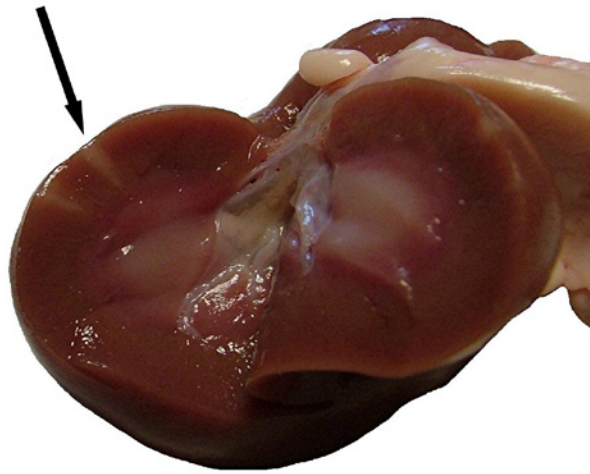
If the emboli are due to pyogenic material the infarction can develop into abscesses or nephritis.



Renal infarction



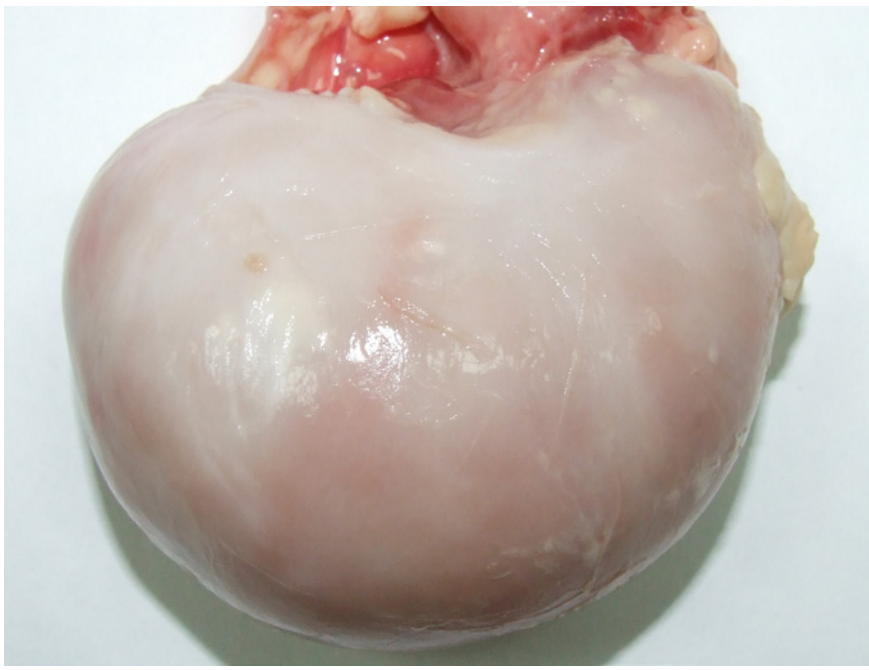
Haemorrhagic infarction in an ovine kidney



Atypical infarction (arrowed), a cone shaped area of ischaemia due to embolic capillary obstruction

TOXIC SHOCK

Pale kidneys are common in cases of bacterial septicaemia. The kidneys should be rejected and the remainder of the carcase checked for signs of systemic infection.



A pale kidney due to toxic shock

OEDEMA AND EMACIATION

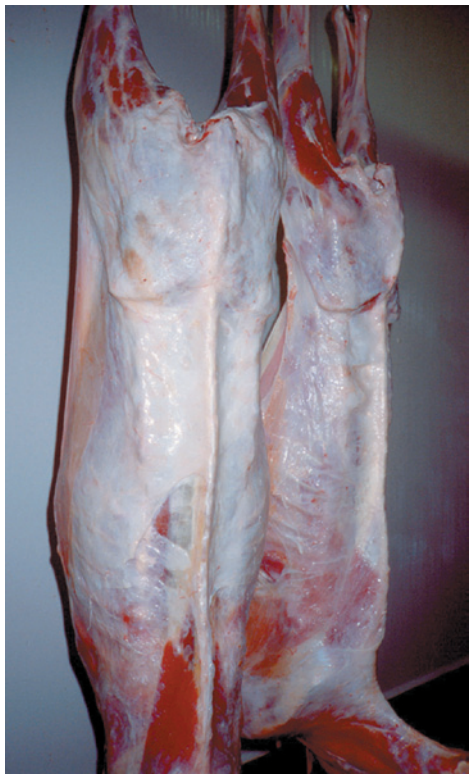
Emaciation is generally due to a pathological condition. The muscle wastes and the remaining fat becomes jelly like. The fat is wet and does not set. In contrast a similar condition, poorness, is due to a change in the functioning of the animal, such as shortage of food etc. Emaciated carcases that are considered borderline cases are detained overnight. Those that are emaciated do not set, and generally become wetter.

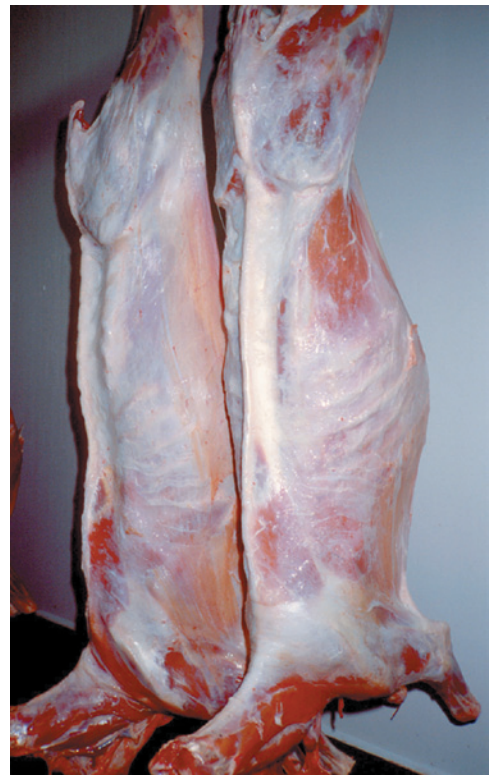
Emaciated carcases are rejected as unfit for human consumption.

Oedema is the abnormal pathological accumulation of tissue fluid and is a symptom associated with disease or insufficiency rather than a disease in its own right. This accumulation can occur in interstitial spaces, beneath the skin (anasarca) and within cavities including the thoracic (hydrothorax), abdominal (ascites) and pericardial (hydropericardium) potential spaces. The presence of oedema may be localised or generalised dependent on cause. Infections or conditions that increase the permeability of blood vessels (such as Vitamin E or selenium deficiency), a reduction in the protein levels of the blood plasma decreasing osmotic pressure, or simple vascular pressure increase (due to physical blockage of the system), promotes the leaking of excess fluid from the blood vessels.

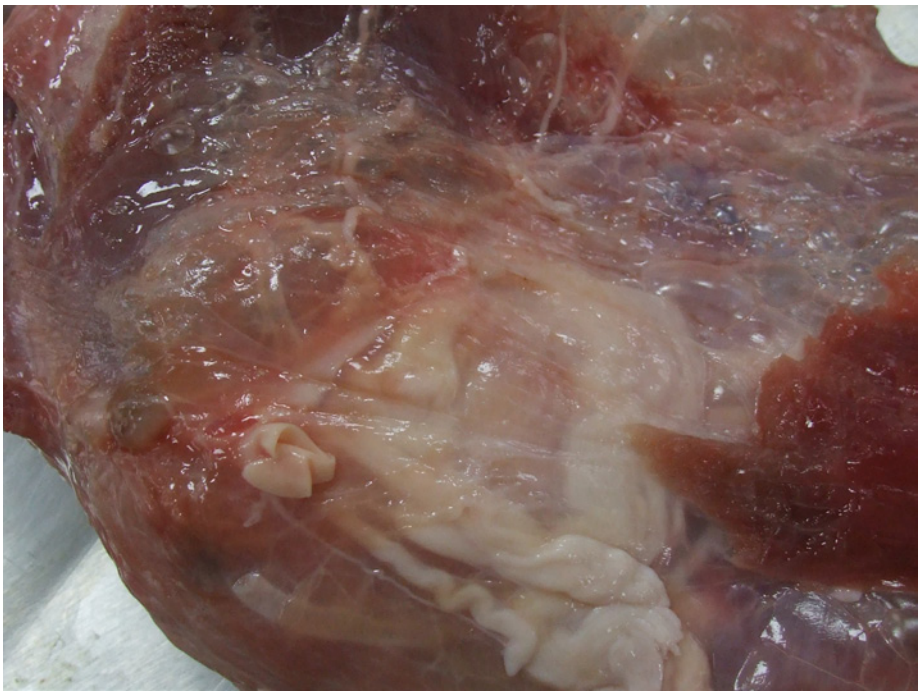
Localised oedema, especially anasarca, can be produced by trauma, prolonged recumbancy interfering with venous flow, reduced cardiac efficiency, or localised infection. Some forms of generalised oedema are indicative of certain conditions; *Fasciola hepatica* infection of the liver can produce a submandibular oedema known as 'bottle jaw' in addition to anasarca in the area of the brisket. Liver disease is a common cause of ascites either through a reduction in the ability to produce plasma proteins or through an increase in pressure in the portal circulation. Generalised oedema is often associated with malnutrition and emaciation and can also be associated with toxaemic and septicaemic conditions that damage the vascular epithelium.

In cases of localised oedema the affected part should be rejected and the carcase checked for other lesions. In cases of generalised oedema, where the fat becomes sparse and gelatinous, the carcase and associated offal should be rejected. In cases of doubt the carcase should be detained overnight and will become wetter in cases of generalised oedema especially in the areas of the pleura and peritoneum. In carcases with generalised oedema a common finding is the oozing of jelly-like exudates from between the vertebral discs in a split carcase.

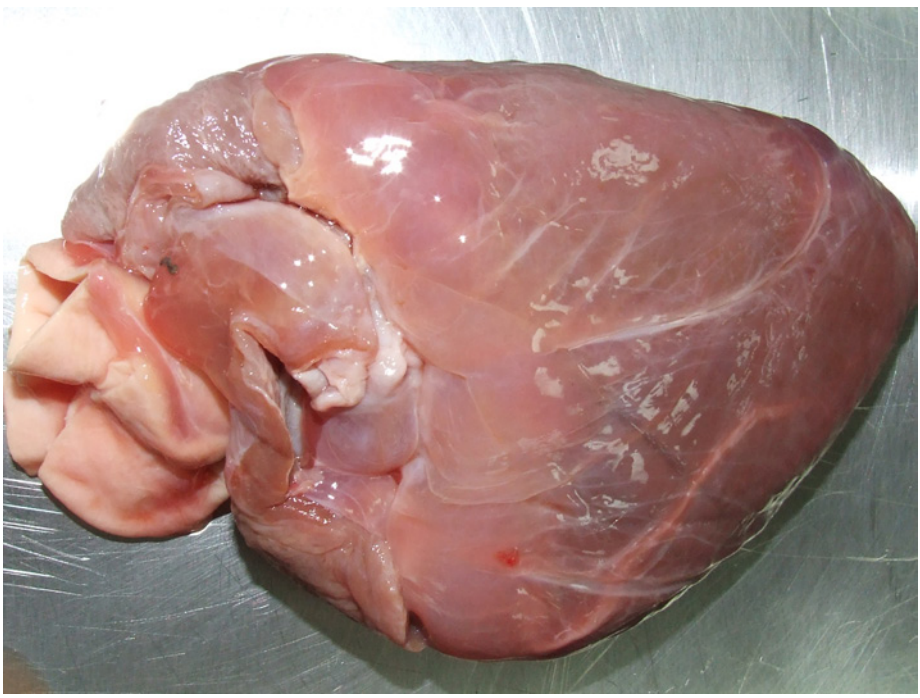




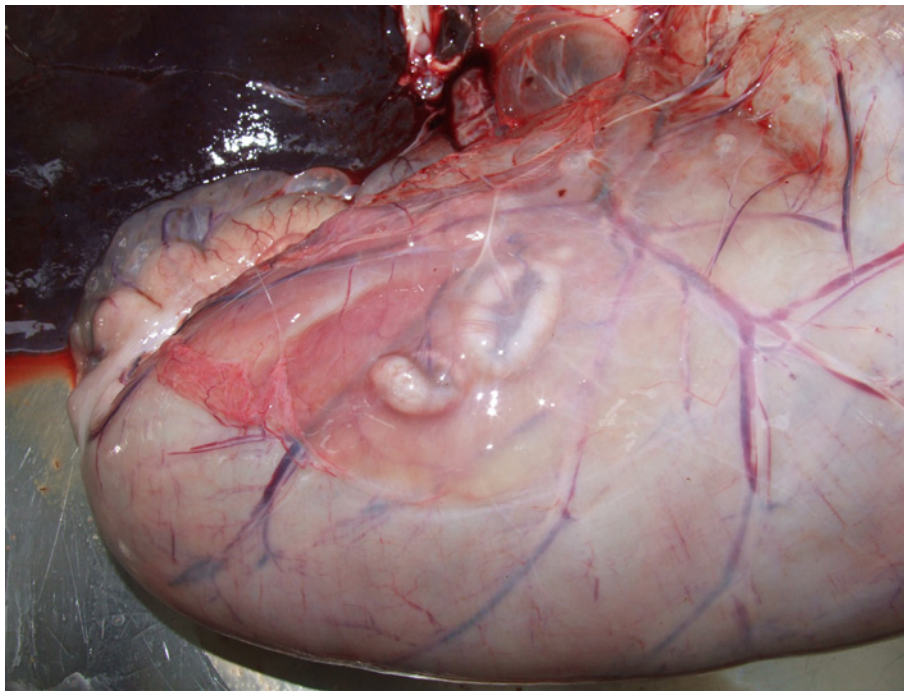
Two ewes graded as emaciated at ante-mortem inspection. Both were too weak to stand



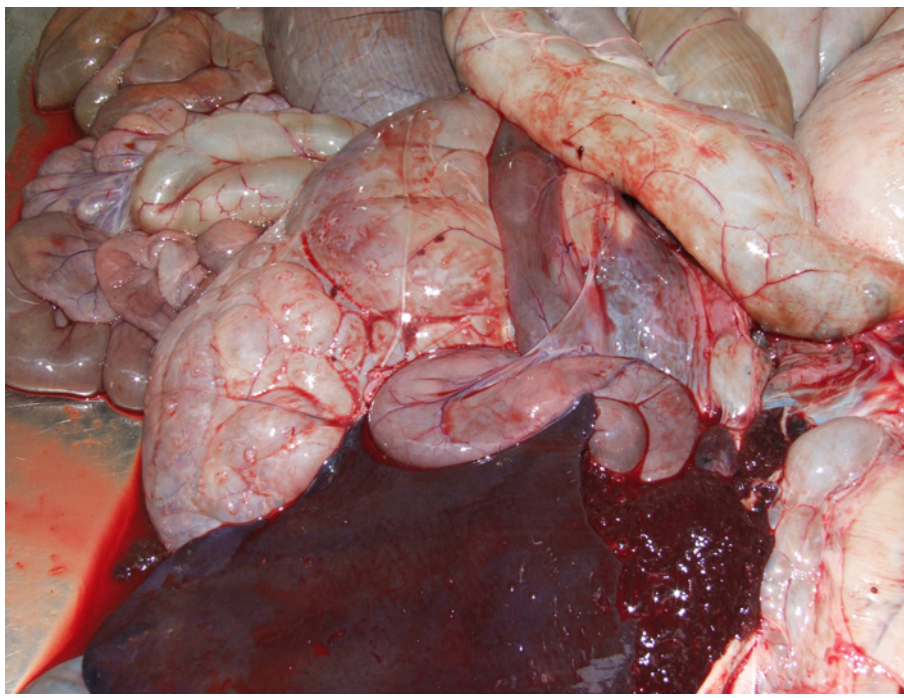
Gelatinous fat and exudation of fluid – excised shoulder, oedematous ewe



Gelatinous cardiac fat and flaccid musculature – Oedematous ewe



Oedematous forestomach – note lack of fat

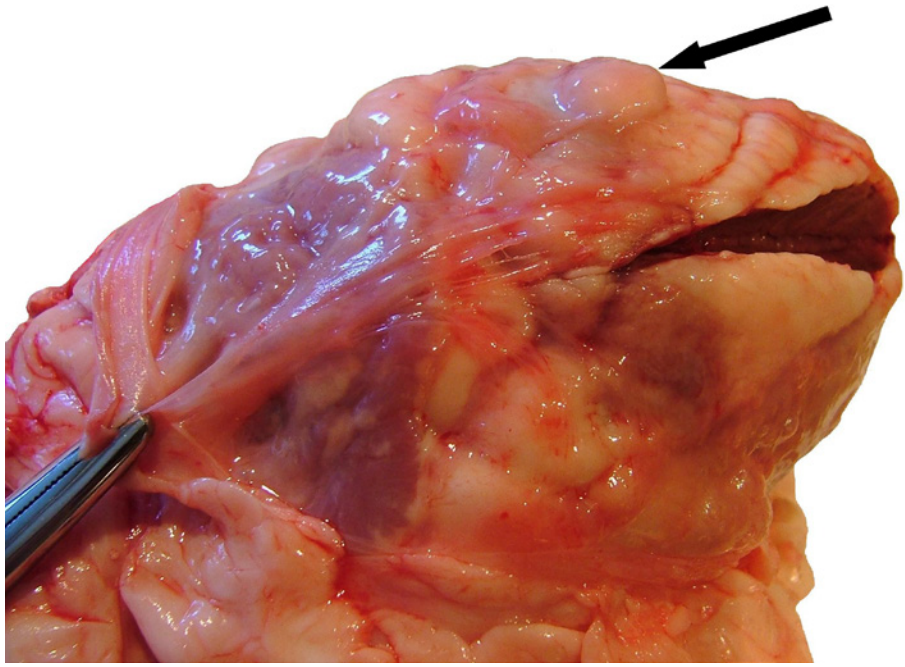


Oedematous green offal from an affected ewe

An emaciated carcase

PERICARDITIS

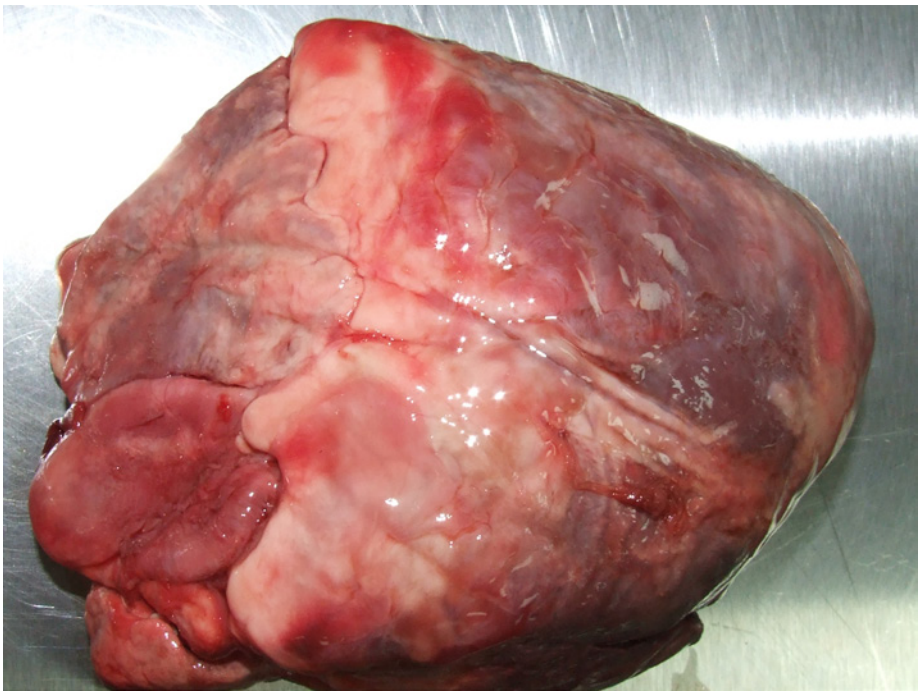
Inflammation of the pericardium, the membrane surrounding the heart containing lubricant. Severe pericarditis can cause adhesion between the heart and this sac. The heart is rejected if no signs of pyaemia or other systemic infection are present in the carcase.



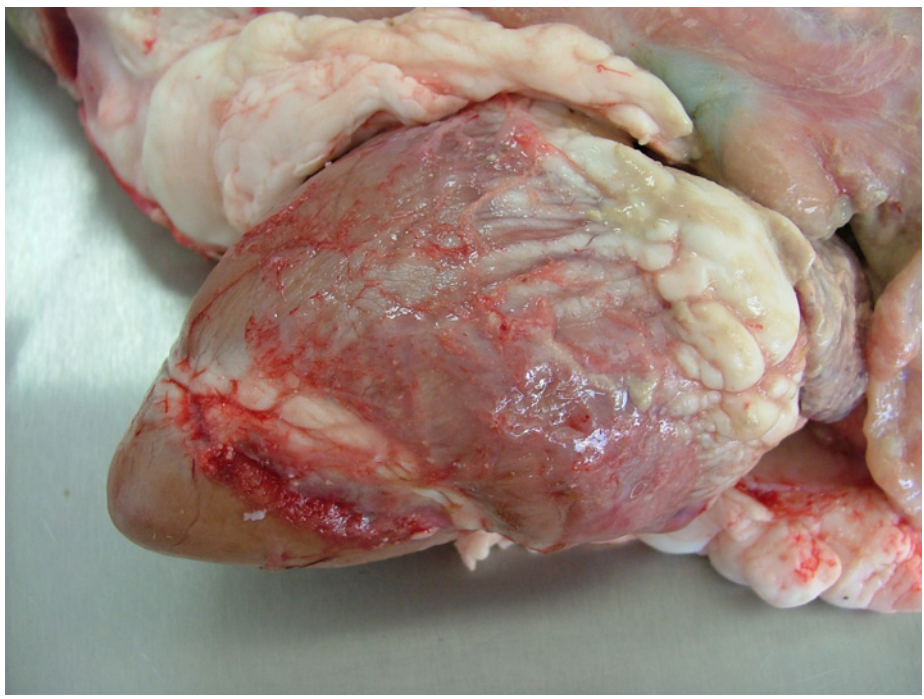
Epicarditis – initially set up by the presence of a *Cysticercus ovis* cyst (arrowed)

Experimentally, the stages of pericardial fibrosis and adhesion following infection consist of inflammatory response, including increased vascular permeability, and exudation of fluid containing fibrin and white blood cells between 3-24 hours after infection.

Pericardial fibrosis and adhesion between the epicardium and pericardium occurs as focal lesions after two weeks with more complete adhesion taking a month to develop.



Pericarditis – an acute inflammatory response



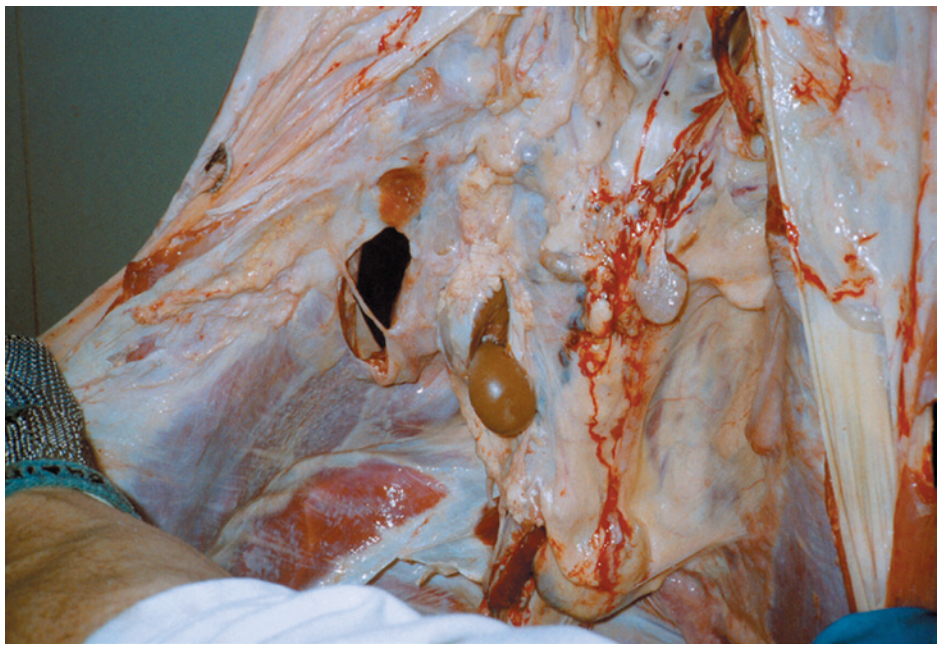
Fibrinous epicarditis – approximately 2-3 weeks post infection

PERITONITIS

Peritonitis is inflammation of the peritoneum. The peritoneum is the serosal membrane that lines the abdominal cavity and also covers the organs within that cavity. The membrane lining the cavity is called the parietal membrane, that part covering the organs is called the visceral membrane.

Peritonitis can be localised or diffuse.

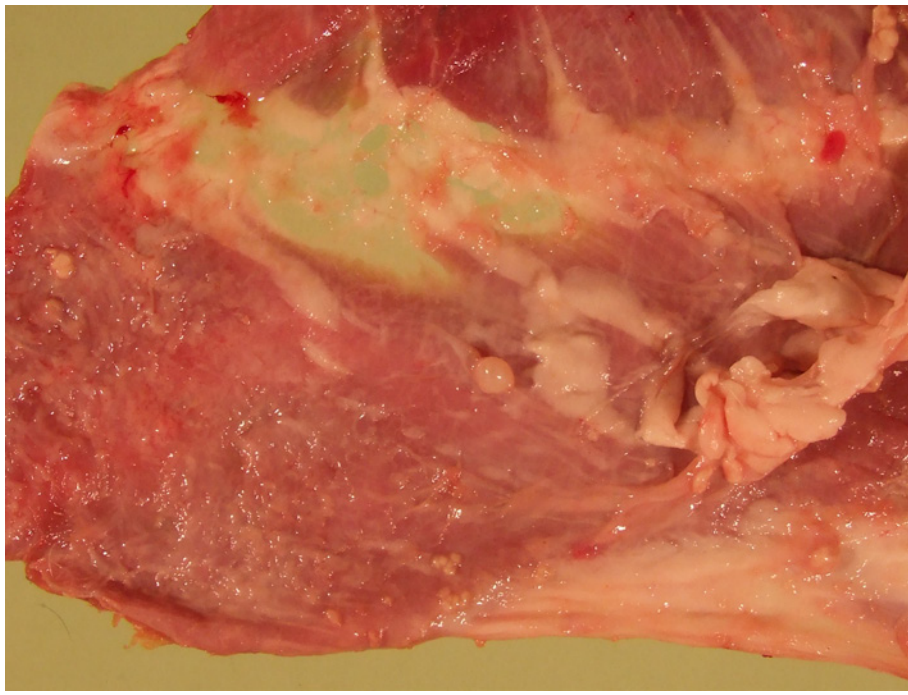
Chronic peritonitis can be produced by factors such as abscesses in the liver, and the affected parts are rejected.



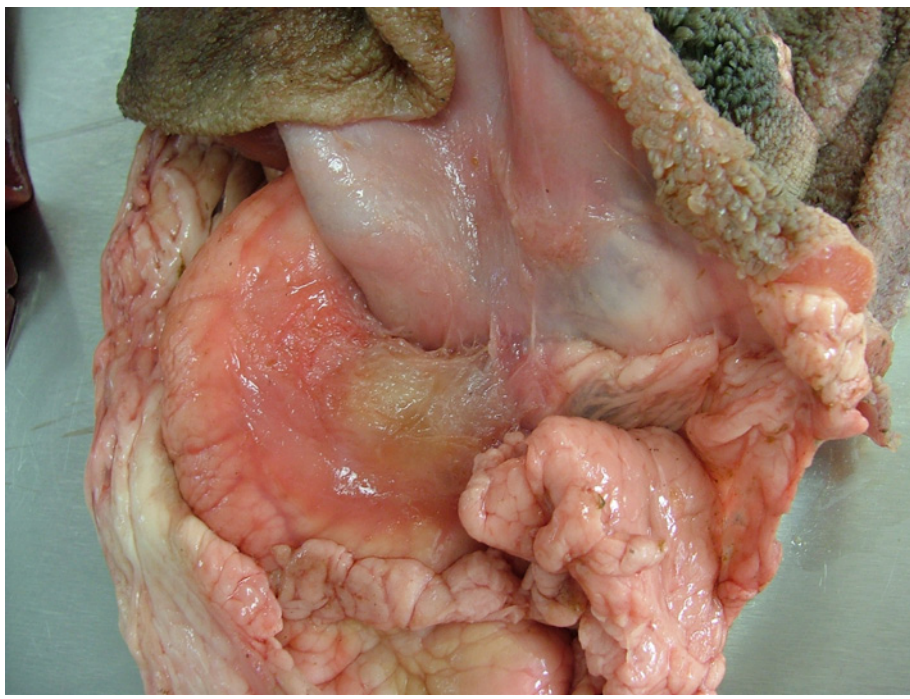
Fibrinous peritonitis

Acute peritonitis is normally produced after bacterial infection of the membrane, either through a wound or as an infection from other areas, such as the stomach. It can lead to ascites. Affected parts are rejected.

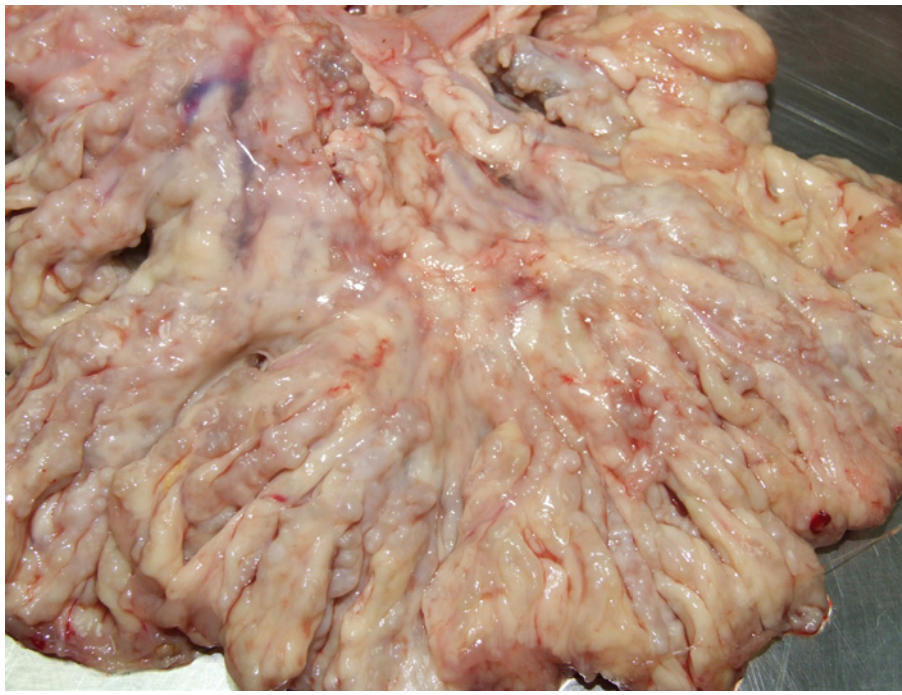
If the peritonitis becomes diffuse and septic the entire carcase and offal is rejected



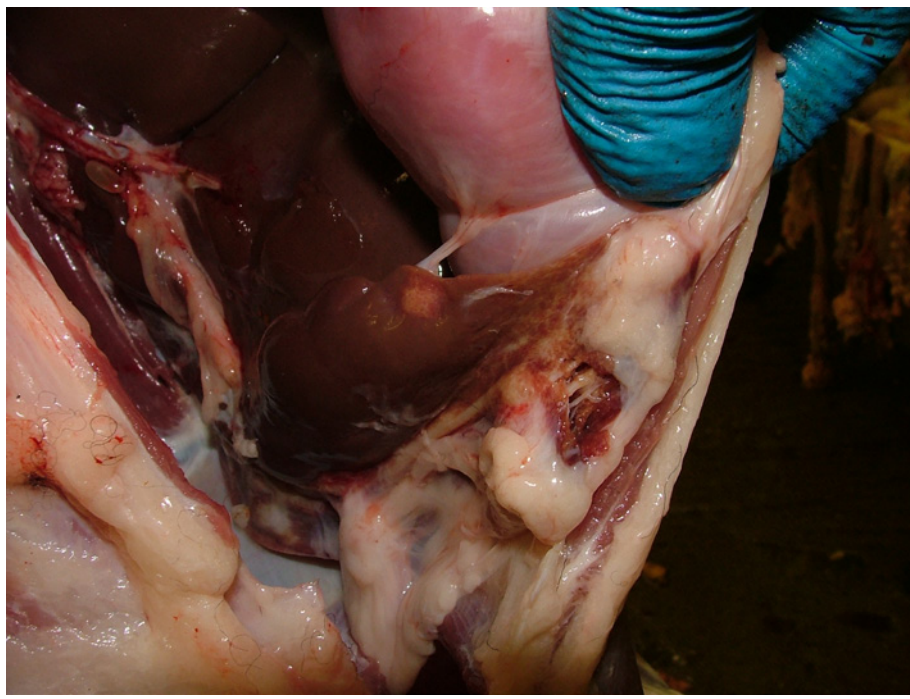
Parasitic granulomatous peritonitis, possibly due to rupture or leakage of a hepatic hydatid cyst



Traumatic peritonitis due to a wire protruding from the reticulum



Extensive parasitic granulomatous peritonitis



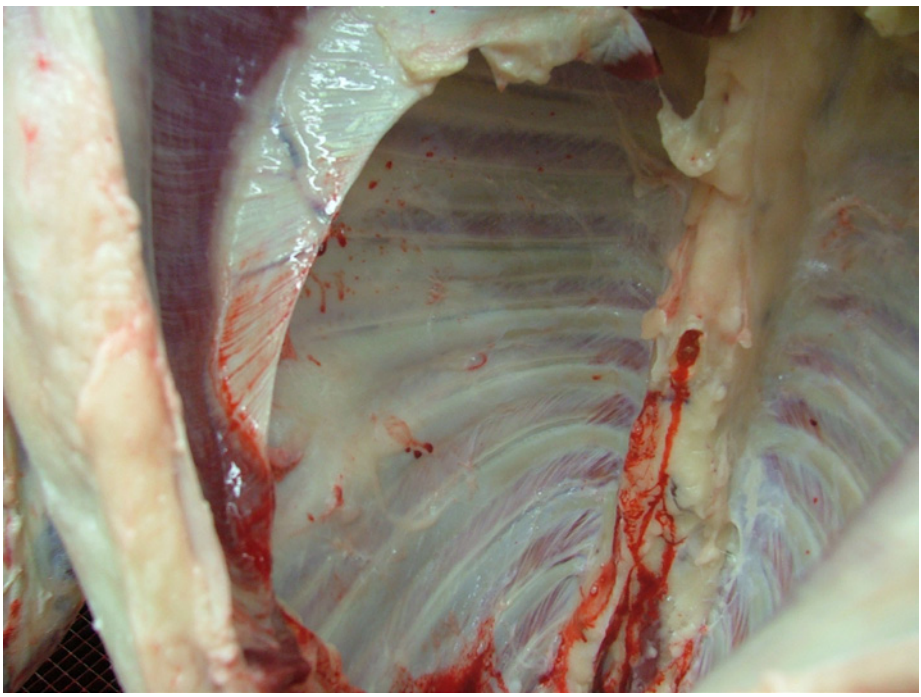
Localised peritonitis due to contact spread of visceral peritonitis as a result of a hepatic abscess

PLEURISY
Pleuritis

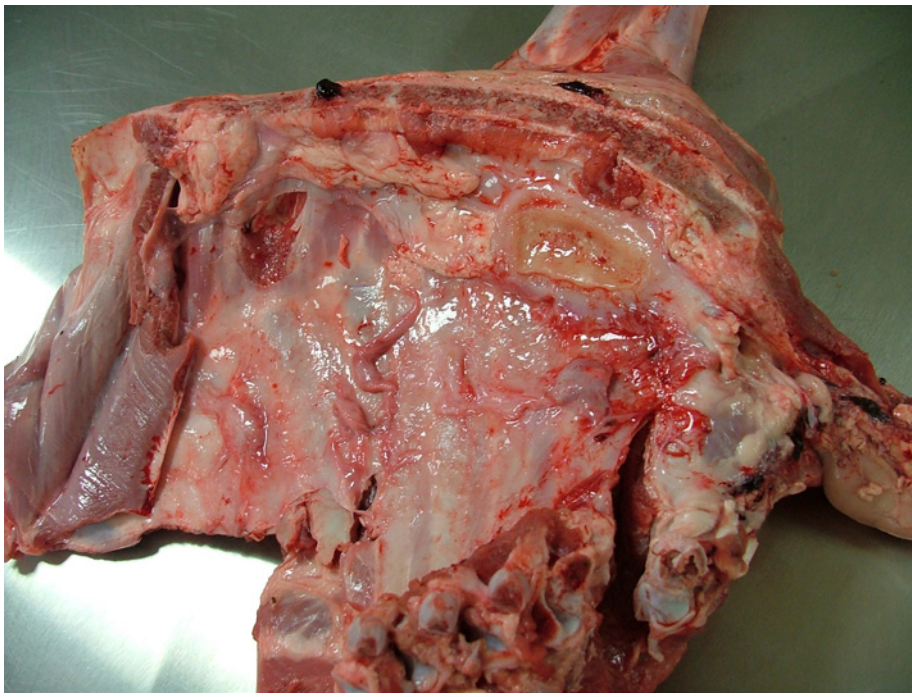
Pleurisy is the inflammation of the pleura, the serous membrane that covers the lungs, heart and walls of the thoracic cavity. This can lead to part of the lung adhering to the ribs following initial fibrin deposition, at which point it is termed constrictive pleurisy. If the infection continues the area of attachment can spread. In the live animal breathing is painful, and the animal will tend to breathe using abdominal muscles rather than its' ribcage. Visceral pleurisy tends to be a complication of pneumonia.

If the pleurisy becomes diffuse and septic consideration should be given to rejecting the entire carcase and associated offal, mild cases of parietal pleurisy can be stripped from the thoracic cavity and rejected

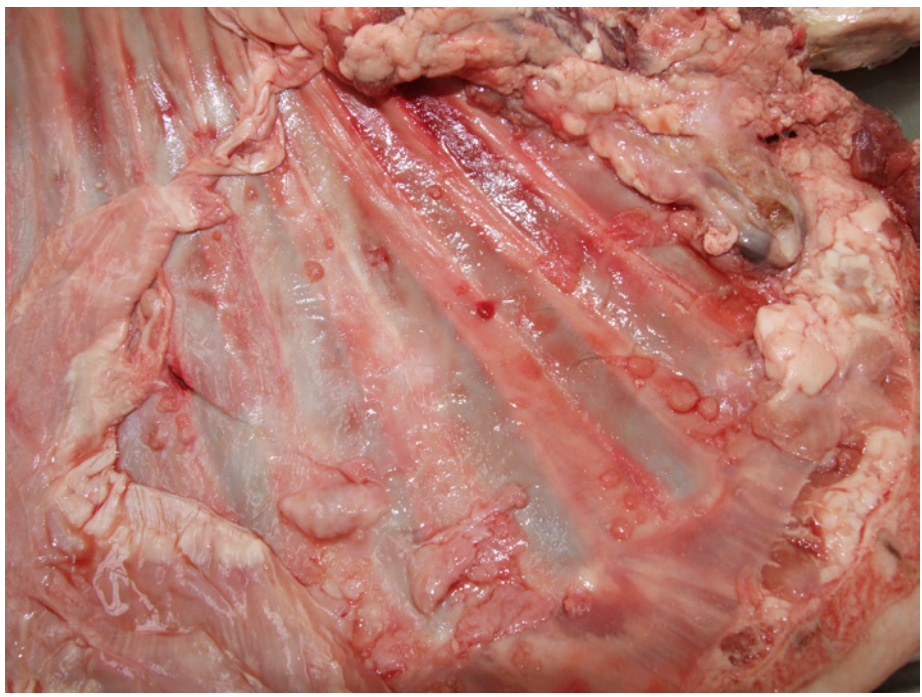
If infected the pleura can be stripped from the ribs and rejected.



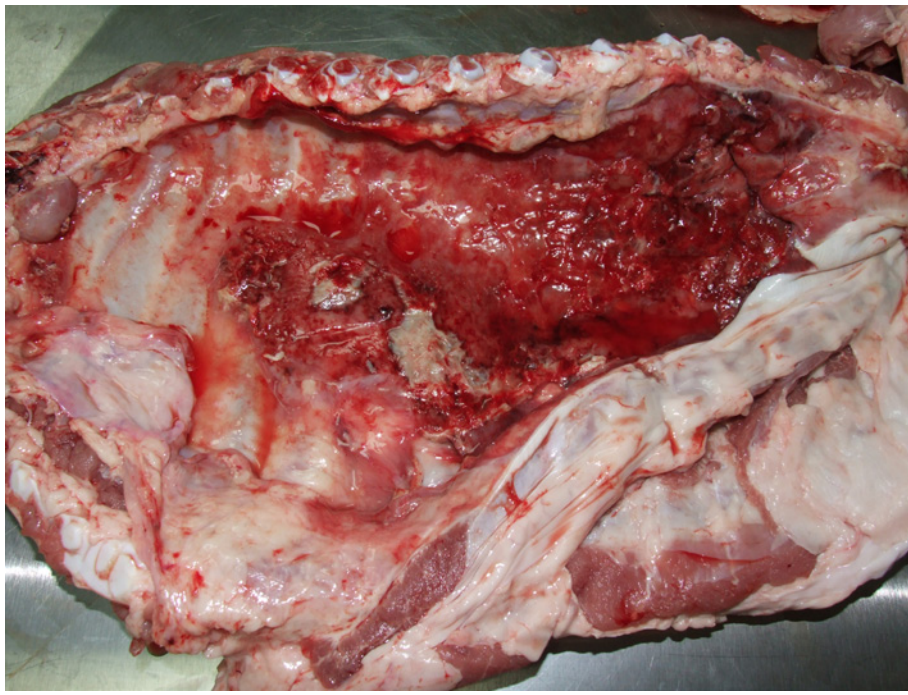
Mild parietal pleurisy can be stripped from the thoracic cavity



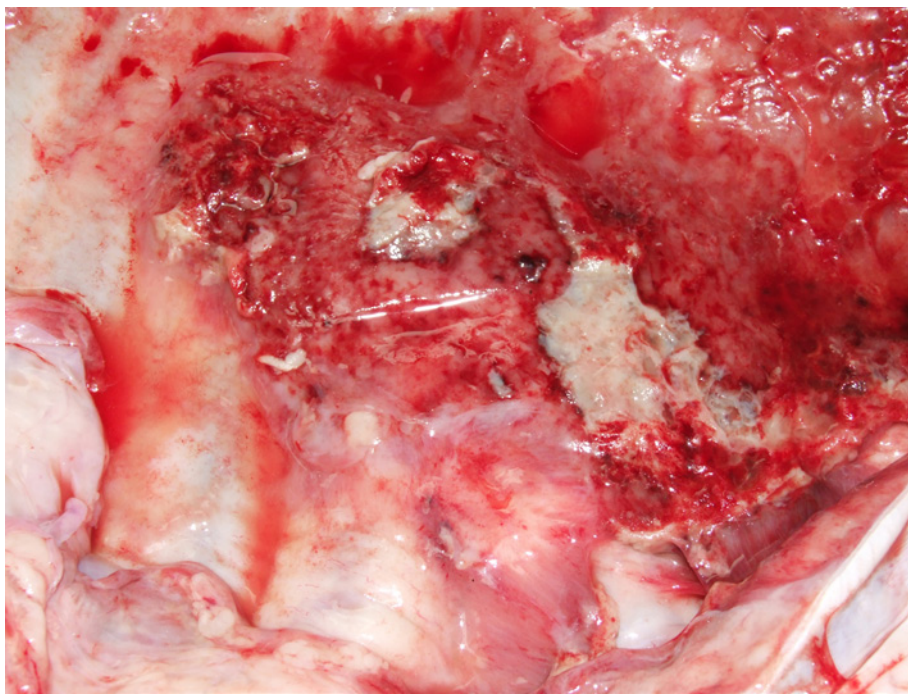
Fibrinous pleurisy due to a pulmonary abscess



Pleurisy and microabscessation



Septic, purulent pleurisy

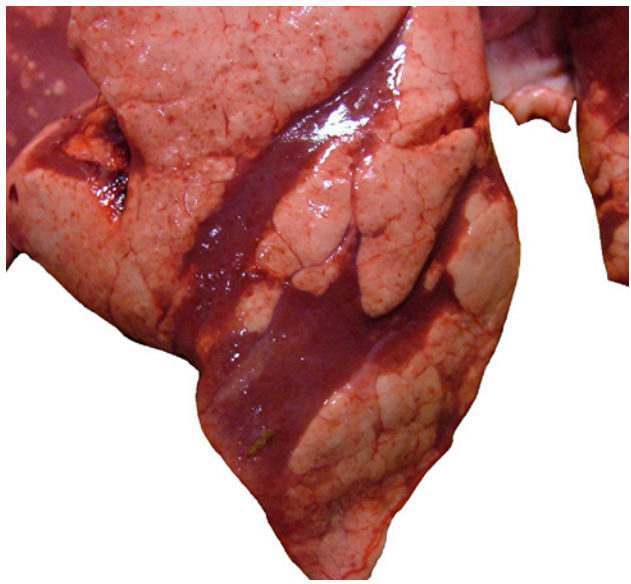


Detail of lesion, which could not be stripped from the ribcage

PNEUMONIA

Pneumonia is inflammation of the lung parenchyma and can be caused by viruses, bacteria, fungus, parasites etc. The main forms encountered are enzootic pneumonia, fibrinous pneumonia, pulmonary adenomatosis (Jaagsiekte) and parasitic pneumonia.

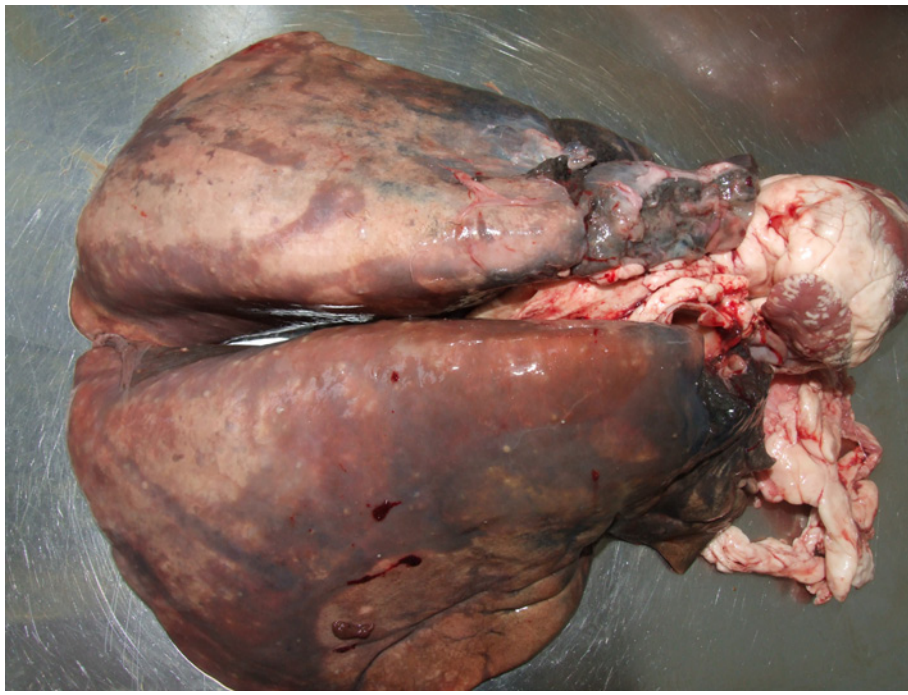
Enzootic pneumonia tends to affect the apical (cranial) lobes and is characterised by collapse and subsequent consolidation of lung tissue producing areas of atelectasis, which do not float in water. The most common primary cause is a viral one in conjunction with, or secondarily bacterially infected by, species such as Mycoplasma. Reinflation can occur and the pneumonia can be reversible. If saprophytic bacteria colonise the affected areas a gangrenous pneumonia can ensue.



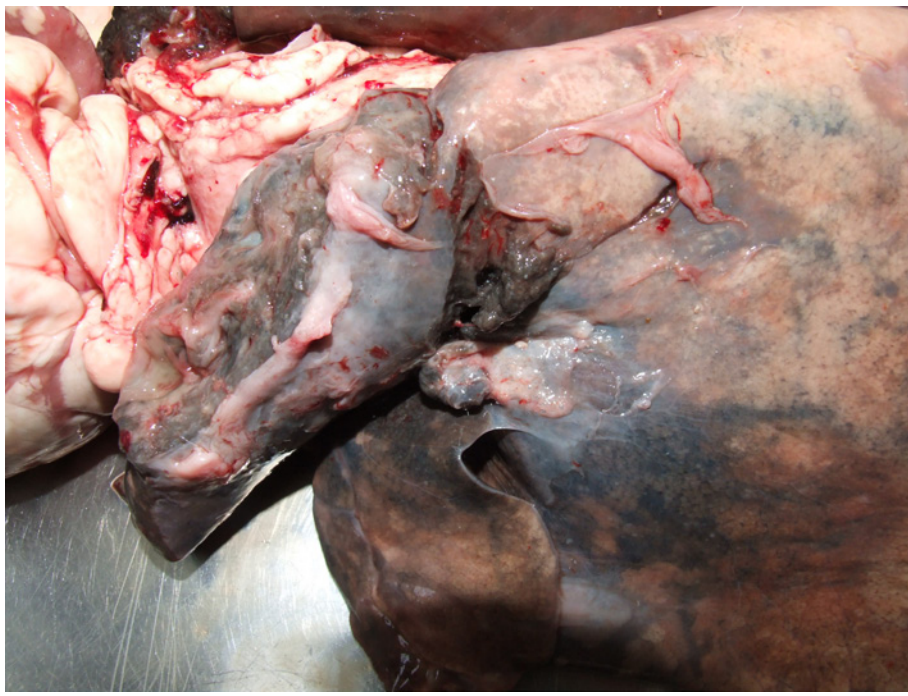
Hepatisised lung tissue atypical of pneumonia. The fissure-like formation of the atelectic tissue indicates that the pneumonia arose from an early infection that is now undergoing reparatory process.

Pasteurella spp are most commonly associated with the development of fibrinous pneumonia that generally only affects one lung. Abscess formation is common in these cases but as a sequel to the infection. Environmental stressors can lead to the pulmonary form of pasteurellosis following previous exposure to the organism in earlier life. Fibrinous pneumonia is characterised by extensive fibrinous exudation and a gelatinous pleurisy which can become adherent to the parietal pleura**.**

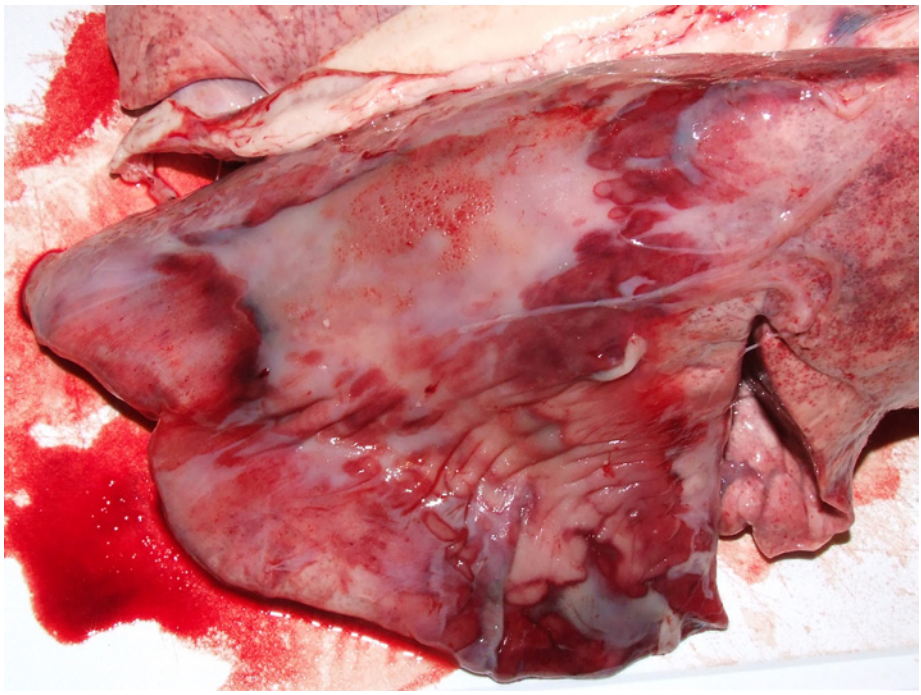
Jaagsiekte and parasitic pneumonia are detailed in Diseases and Parasites respectively.



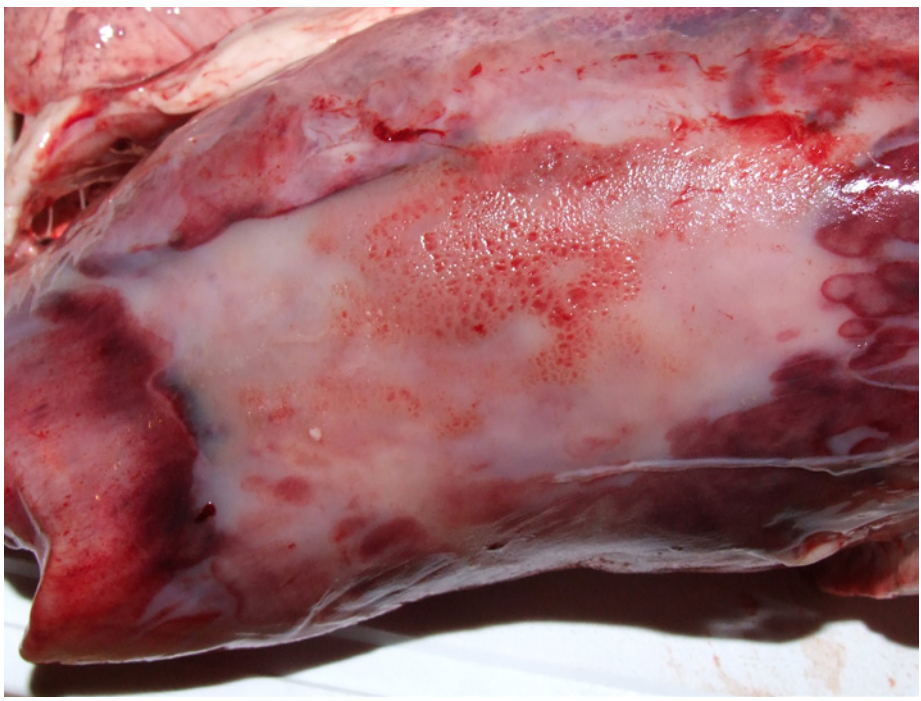
Gangrenous pneumonia



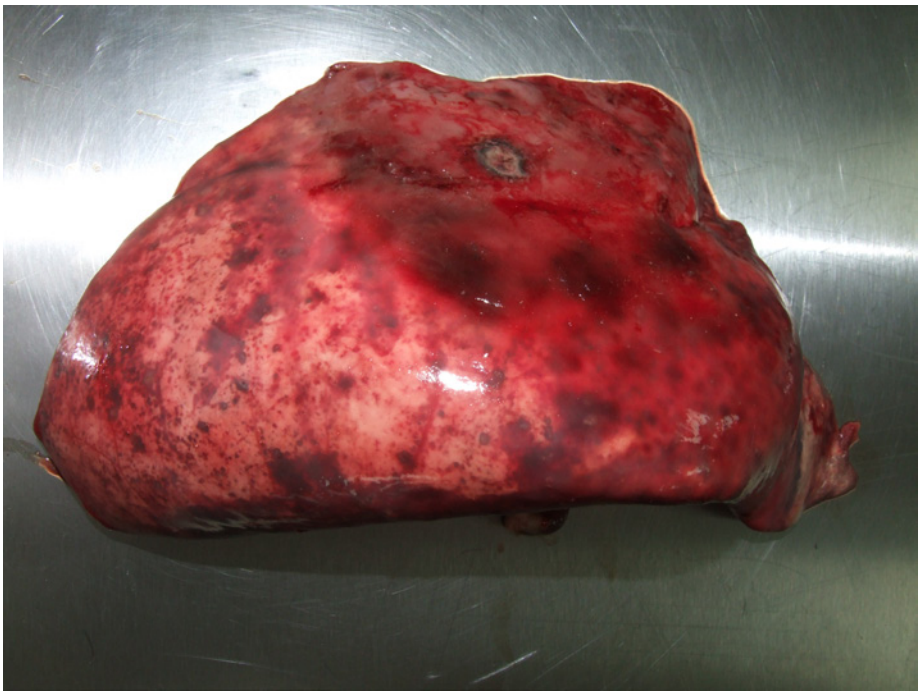
Gangrenous pneumonia - detail



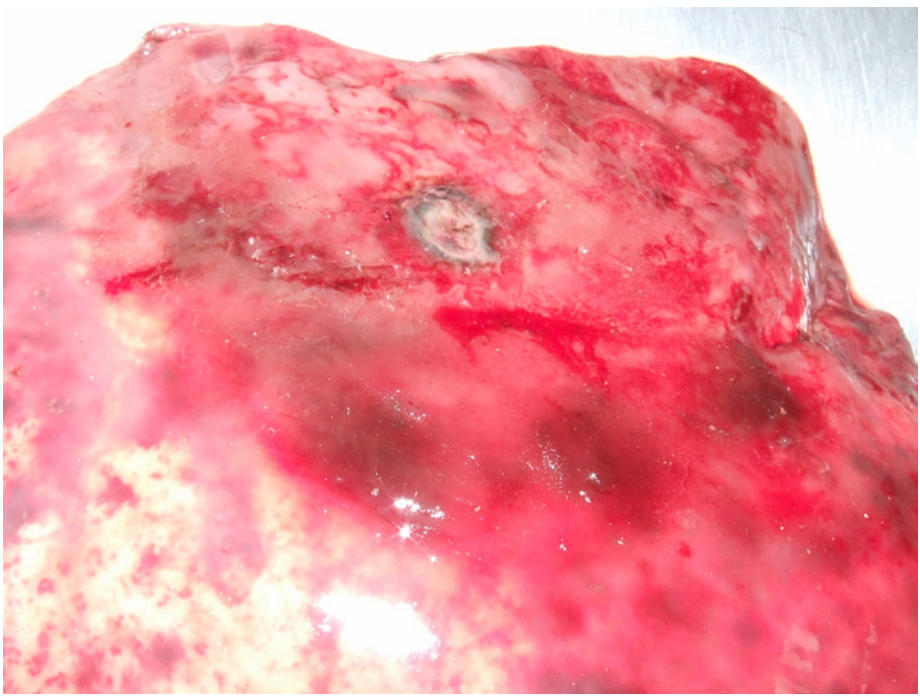
Fibrinous pneumonia



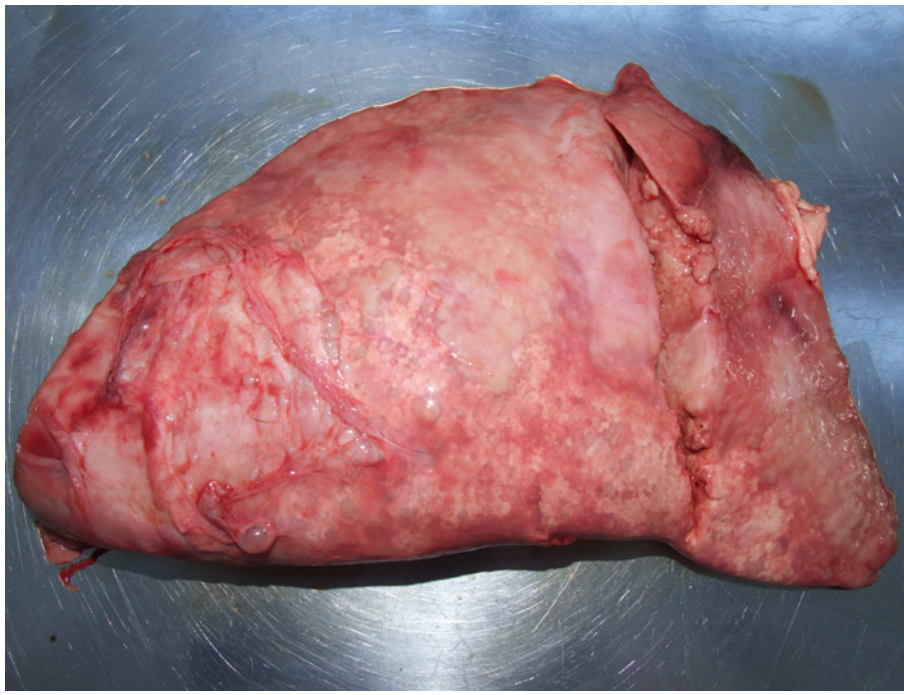
Fibrinous pneumonia – detail of lesion



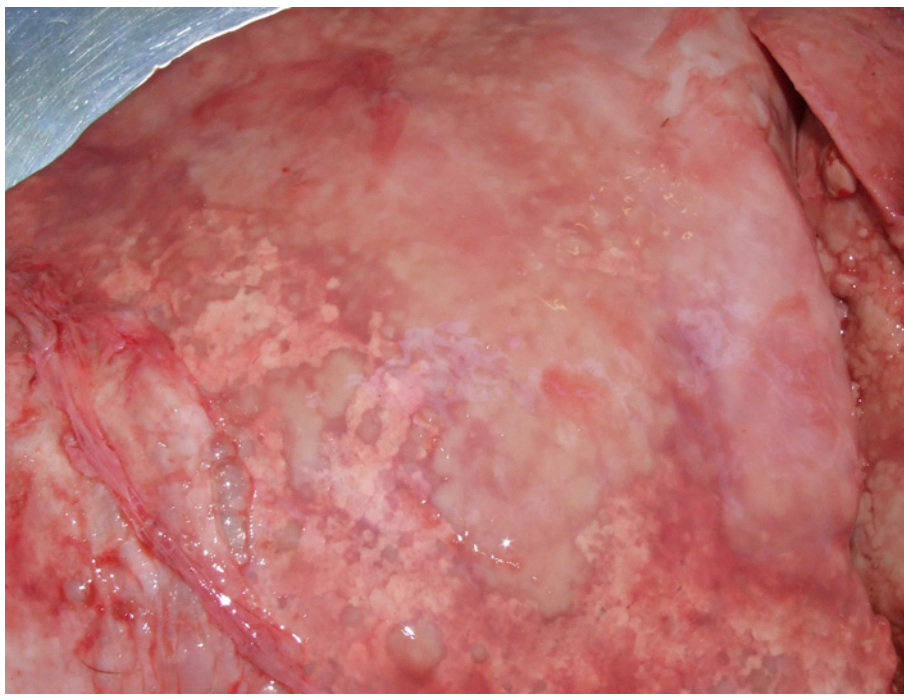
Fibrinous pneumonia and abscess formation



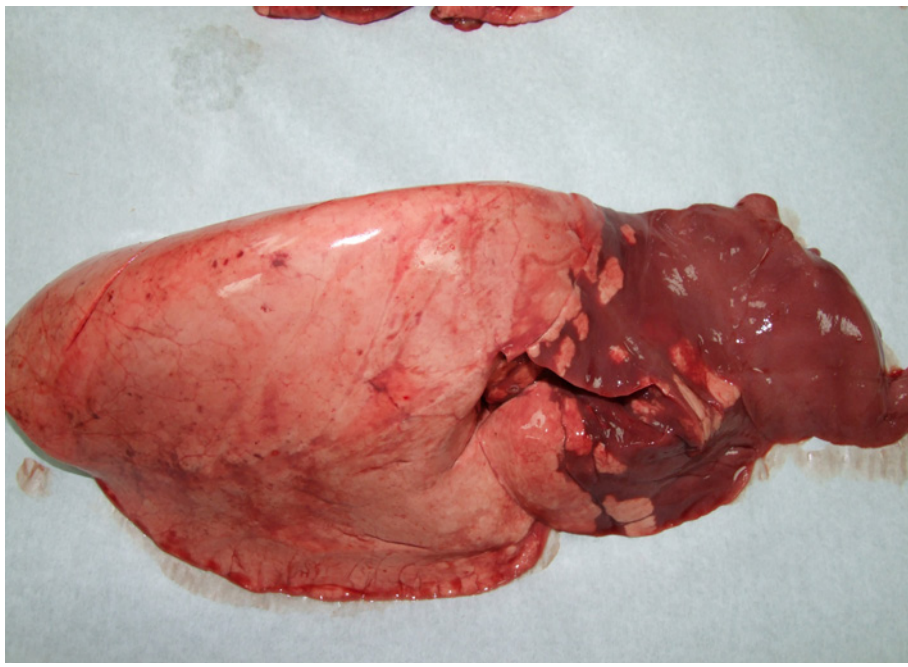
Detail of abscess formation



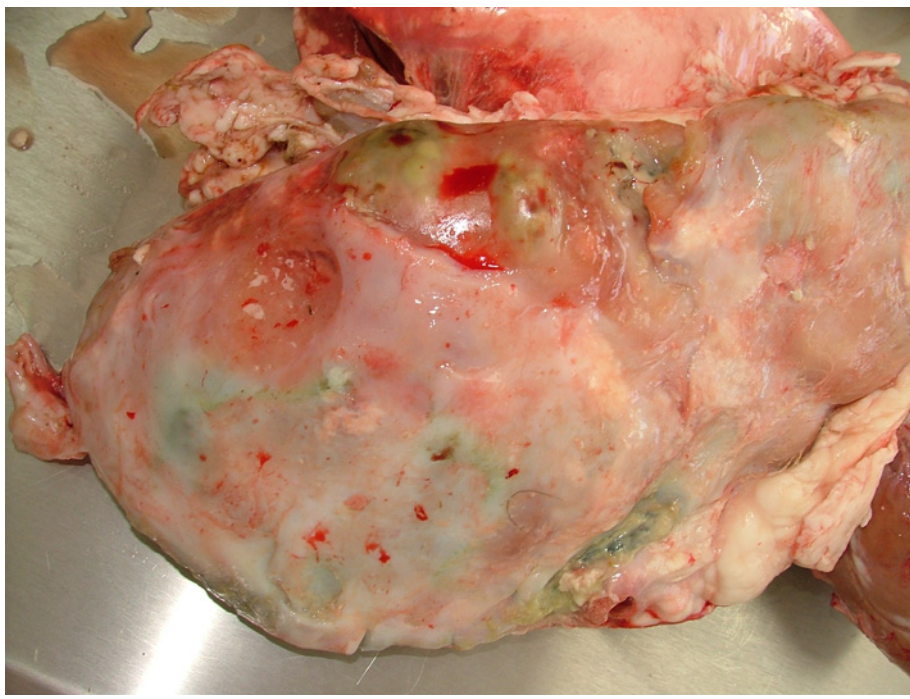
Pulmonary adenomatosis



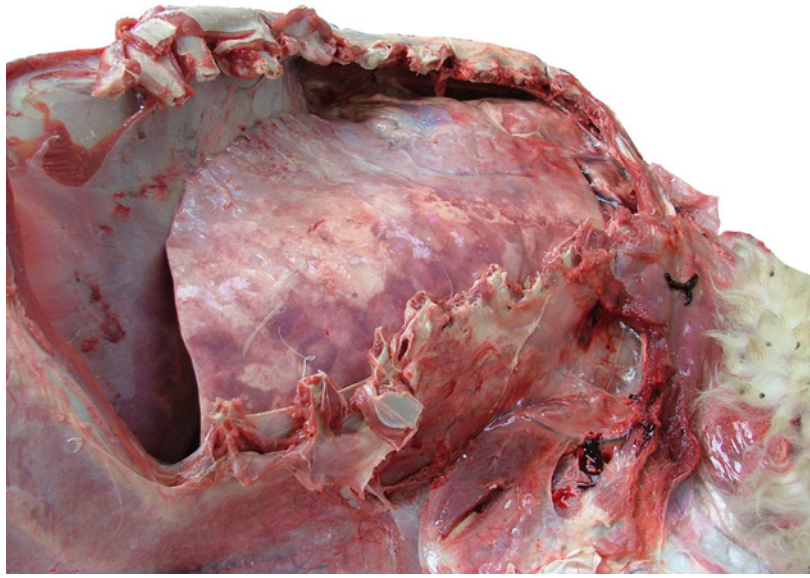
Detail of adenoma



Enzootic pneumonia – affecting apical lobes

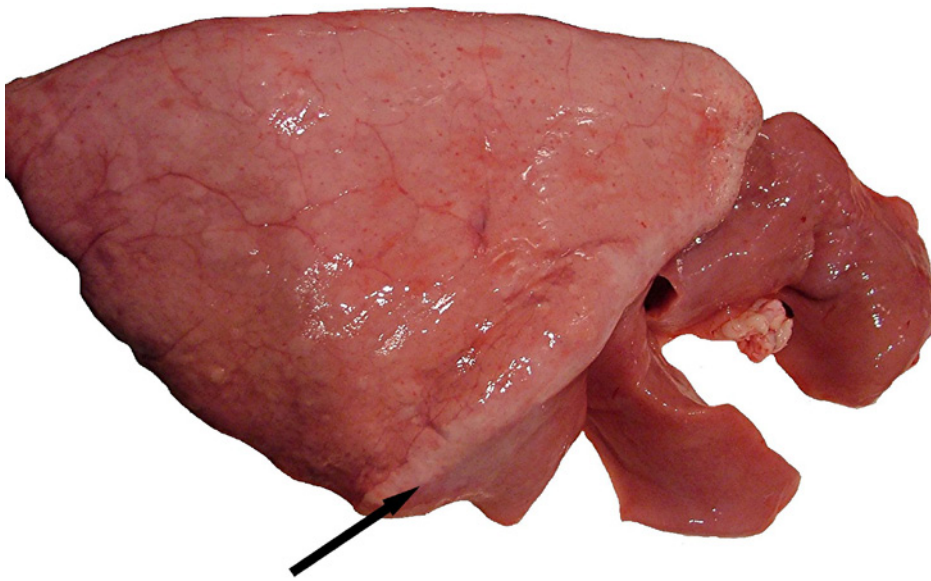


Abscess formation as sequel to fibrinous pneumonia, commonly a unilateral condition



Non-collapsing lungs, adhering to the ribcage

All forms of pneumonia can become septic or gangrenous and may spread to the pleura. Septic pneumonia is fairly common in sheep, and the carcase is judged on the evidence of systemic infection, if the evidence indicates a systemic infection the carcase and offal are rejected as unfit for human consumption. Lungs with pneumonia or pleurisy are rejected as unfit for human consumption.



Atypical ovine broncho-pneumonia. Hepatisized apical and cardiac lobes

PYAEMIA / GENERALISED ABSCESSATION

This condition is produced when pyogenic (pus forming) bacteria gain access to the blood stream and form abscesses throughout the body. Access to the bloodstream is normally from a suppurative focus such as foot rot, injection abscesses, injury, etc.

In the live animal high fever and the presence of an area of local infection may be apparent. If the infection is particularly virulent it can cause the animals' death. In young animals the route of infection can be through the infection of the umbilicus.

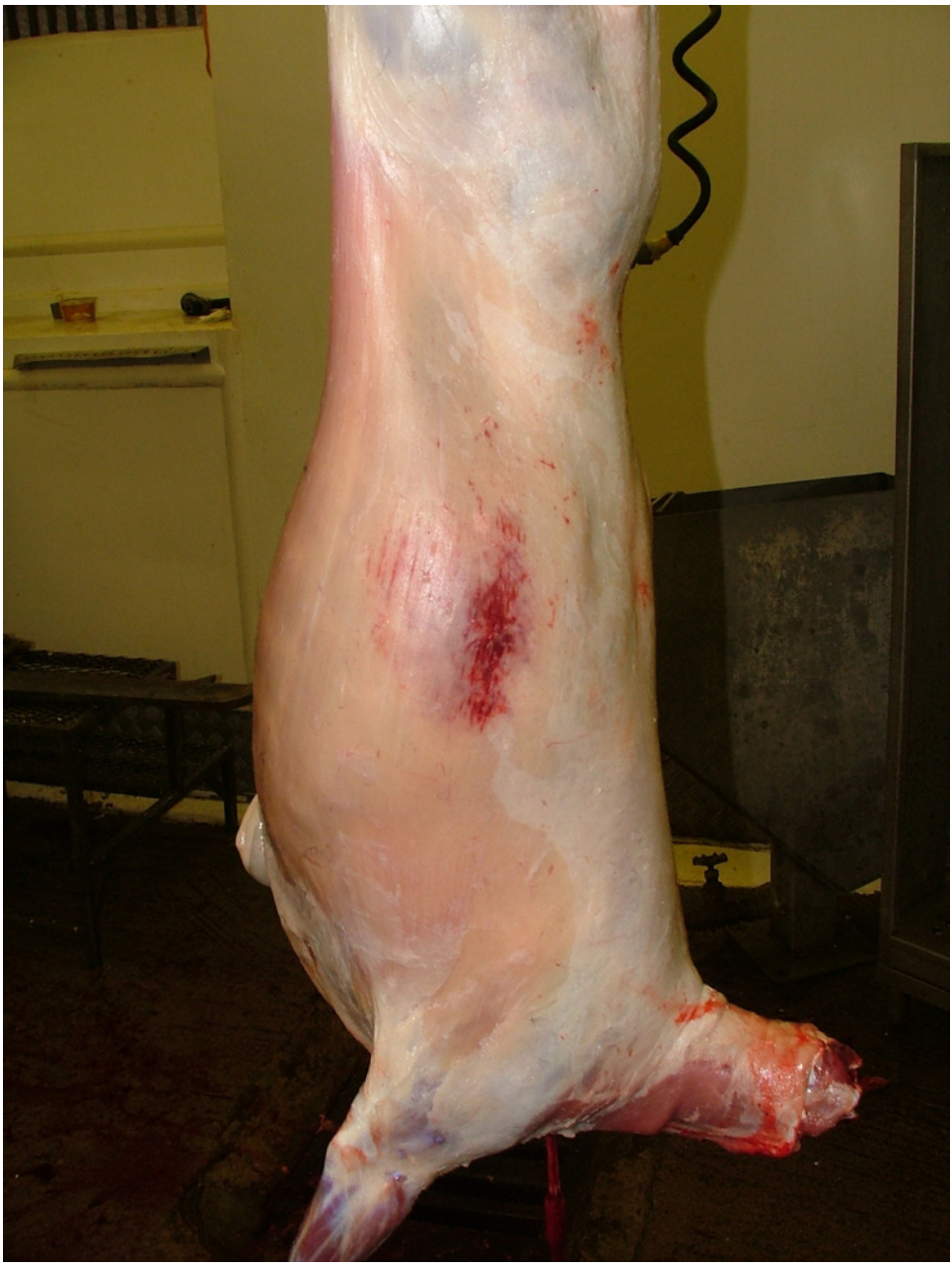
Acute pyaemia exhibits lesions in the carcase similar to those of septicaemia, with the addition of abscesses. The presence of abscesses throughout the carcase especially in conjunction with a localised infection warrants complete rejection of the carcase and offal.

TRAUMA

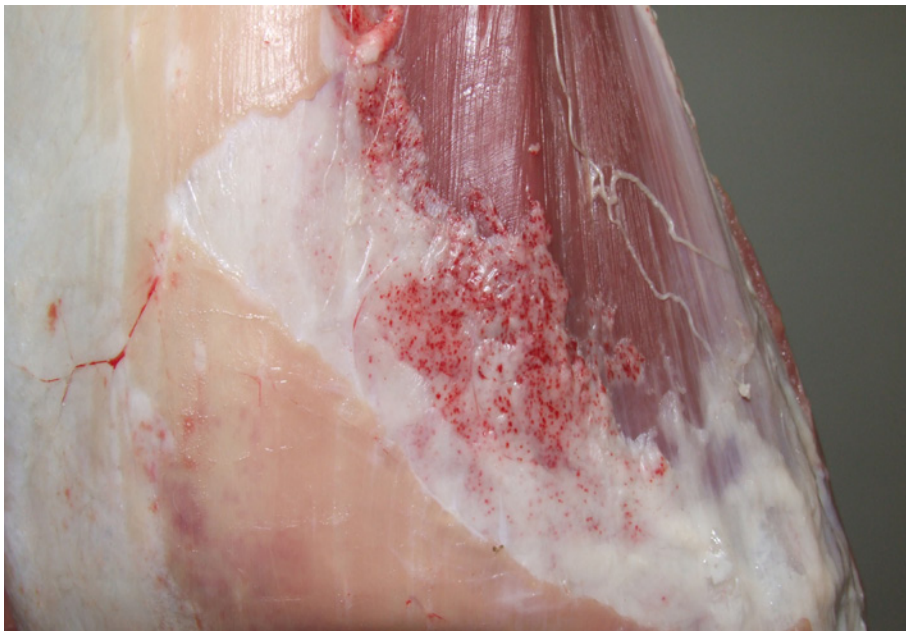
The title trauma denotes any injury to the animal that warrants rejection of part or the entire carcase as unfit for human consumption. Any trauma to the animal pre-slaughter, i.e. before the blood is drained, will produce associated bruising (haemorrhage); all cases of pre slaughter trauma are examined with a view to their welfare implications. These injuries can include:

Bone fractures – the local area is trimmed and rejected, due to both haemorrhage and the possibility of bone fragments. Depending on the age and condition of the fracture, the transportation of the affected animal should raise welfare concerns. Affected parts are trimmed and rejected as unfit for human consumption.

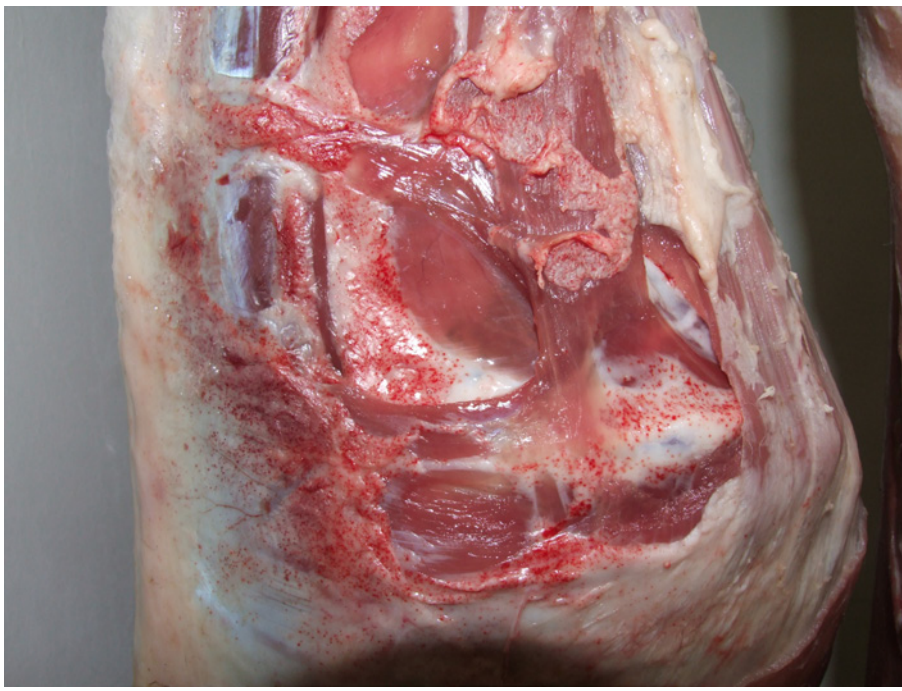
Wool pull – Lifting or catching sheep by grasping the fleece can lead to sub-epidermal haemorrhaging and is illegal in the UK. Affected parts should be trimmed and rejected. The incidence of this condition at post mortem inspection should be monitored, as it is a welfare issue. It is especially prevalent in spring lambs.



Cutaneous haemorrhaging caused by pulling the fleece

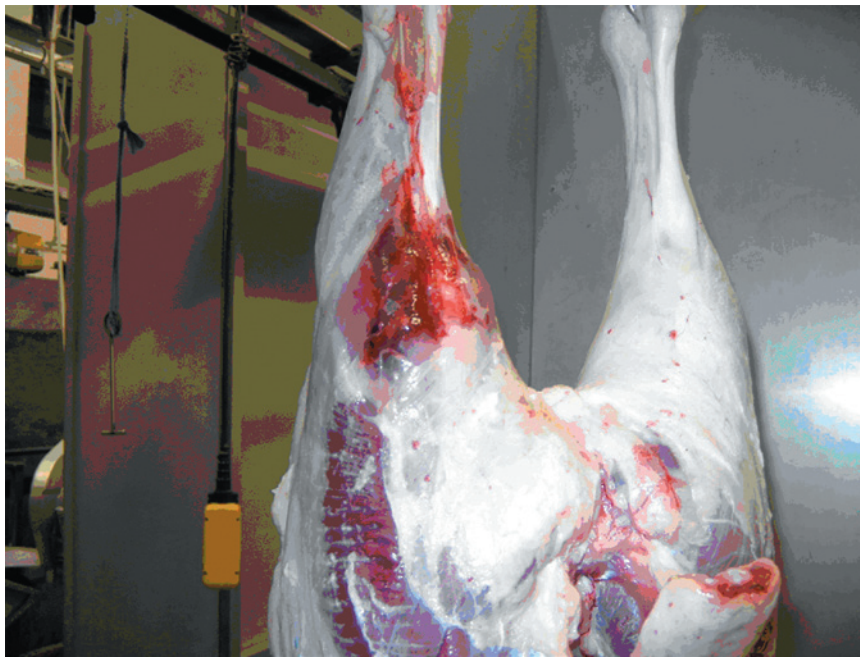


Extensive Petechial haemorrhaging produced by wool pull extends into subcutaneous fat deposits



Trimming of a wool pull lesion shows the extent of the damage that can occur

Dog bites – are trimmed, although large numbers of dog bites to a batch, or reoccurring bites may require report. These present at post-mortem inspection as puncture wounds associated with haemorrhage. Generally the rear legs are affected due to 'nipping' of the legs when sheep are chased. Old wounds may be a route of infection.

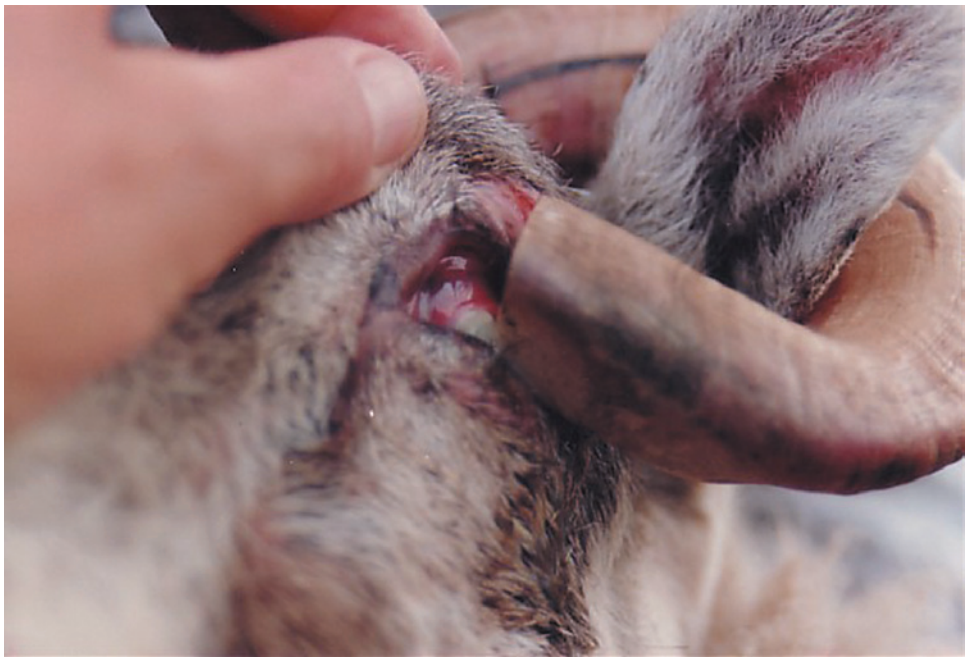


Dog bite

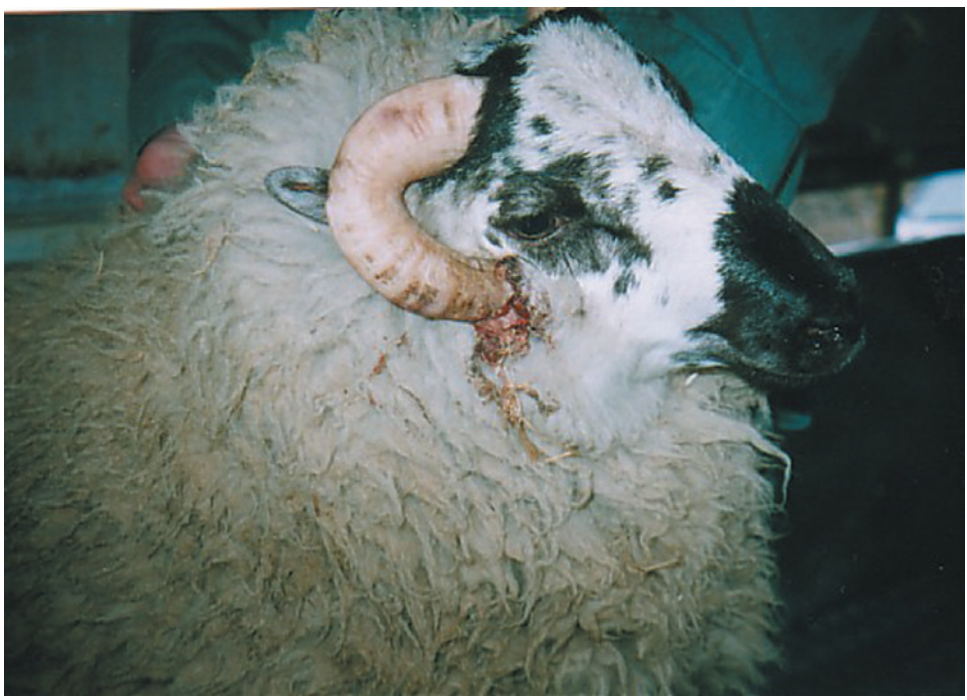
Bruising – small areas of bruising, for example fleece pull, are trimmed and rejected.

Ingrowing Horn – Most commonly found in hill or mountain sheep, this condition is a result of the unchecked growth of the horn that can eventually spiral back into the head of the animal. This level of growth takes months to achieve and its occurrence is therefore considered an indication of poor stockmanship and is a welfare concern.

The lesions associated with this condition are dependant on where the horn comes into contact with the head; traumatic blindness is common as is abscessation at the site, which in turn can lead to secondary bacterial infection and blowfly myiasis.



Ingrowing horn – Corneal ulceration and laceration of the eyelid



Ingrowing horn – note exudates on fleece

An infected eartag

TRICHOBEZOARS
'Hairballs', Enteroliths

Trichobezoars are an incidental finding at post mortem inspection. They can be found in the rumen and reticulum, particularly in sheep that have been overwintered outside. A primary focus of ingested wool builds up in concentric layers due to the churning motion of the rumen, forming a ball. This material can become densely packed producing a felt-like core. Mineralisation of the external surface can occur producing a hard outer shell.

Pathogenic conditions causing pruritis and excessive grooming (trichophagia) such as scrapie and mange can also lead to trichobezoar formation. The ingested wool collects on the surface of the liquid layer in the rumen, sinking when the collected mass becomes dense.

A trichobezoar, split medially illustrating shell and concentric formation rings

Large trichobezoars remain in the forestomach and appear to cause no ill effects to the animal. Small boluses can block the oesophagus, the pyloric sphincter, and occasionally cause intestinal blockage.

URAEMIA

Uraemia is an affection where urine is absorbed into the circulating blood, normally as a result of obstruction of the normal flow of urine from the body due to renal failure.

Urine is a fluid containing water and the soluble waste products of the body. It is produced within the kidneys and flows down two ureters to the bladder, where it is stored until it is voided through the urethra. The waste products include urea and other nitrogen type products that are produced as a result of proteins and amino acid reactions in the body; these are filtered out in the kidneys.

Kidney (or renal) failure obstructs the normal flow of urine, as can occur in animals suffering from hydronephrosis or pyelonephritis. Other conditions such as urethritis (inflammation of the urethra), urethral calculi, ureteroliths (stones in the ureters) and renal calculi (kidney stones) can also obstruct the flow of urine. The reabsorbed waste, which is carried by the blood to all the organs and tissues of the body, gives the flesh a pronounced smell of urine, affecting the odour of the entire carcase and offal.

A quick test for uraemia in a carcase makes use of the fact that the smell of urine increases when the tissues are heated, a fact that also partially explains why uraemic carcases are unfit for human consumption. A piece of muscle is removed from the carcase after 24 hours and boiled or fried. In cases of uraemia the smell increases. This is useful

in determining whether the smell of urine is due to uraemia or the incision of the bladder during evisceration.

Carcases and their associated offal are rejected as unfit for human consumption in confirmed cases of uraemia, as the offensive smell of urine increases in intensity on cooking. It is also an indication that the blood urea nitrogen content and creatinine levels in the body tissues has risen to levels that are toxic to the animal, in addition to the presence of other nitrogenous compound products of metabolism that are normally voided from the body.

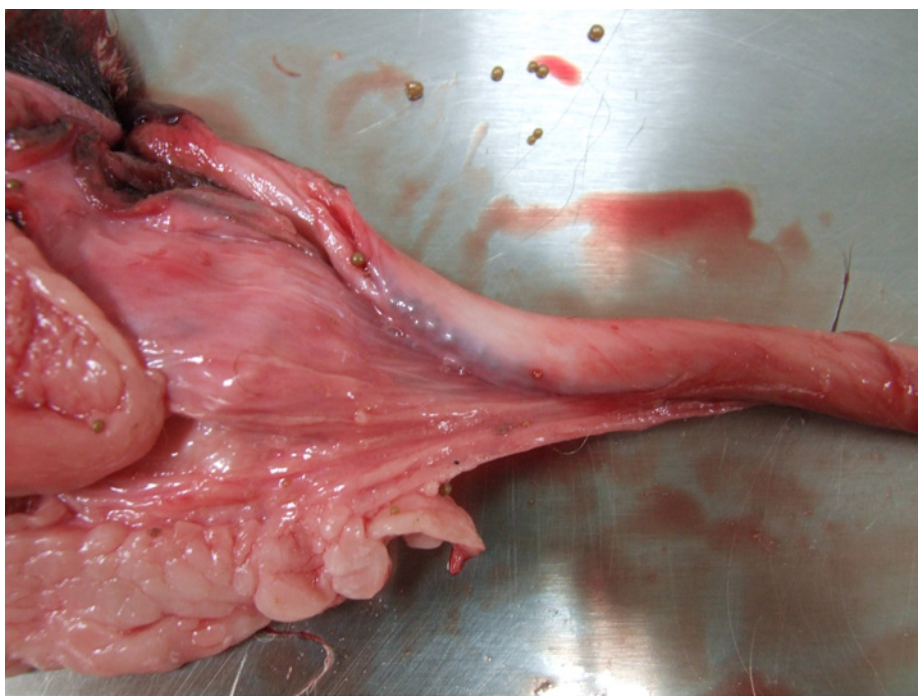
URETHRAL CALCULI

Calculosis, Urolithiasis, Urinary calculi, Waterbelly

This condition affects males and has a metabolic aetiology resulting in the formation of concretions within the urinary tract. This formation involves the deposition of magnesium and phosphate salts from urine following infections that increase the urine pH, and/or animals on high grain diets.

At ante mortem affected animals may have a protruding penis, abdominal pain and tail twitching.

Urethral calculi may lead to cystitis and rupture of the bladder and possibly to urethral perforation and uraemia.



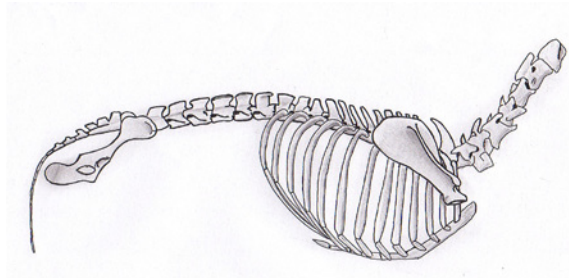
Urethral calculi visible as spherical masses along the line if the urethra

6

AIDE MEMOIRE - ANATOMY

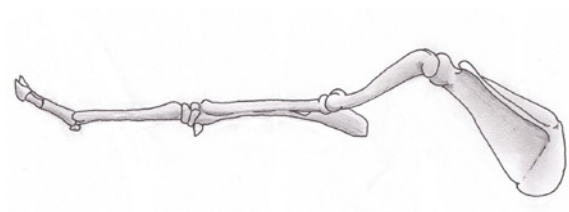
Vertebral Column

- 7 cervical vertebrae
- 13 thoracic vertebrae
- 6 lumbar vertebrae
- 5 sacral vertebrae
- 18-20 coccygeal vertebrae



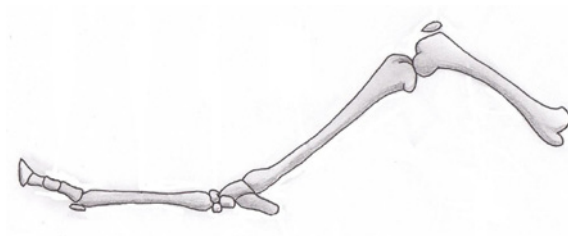
Foreleg

- Scapula
- Humerus
- Radius
- Ulna
- Carpus
- Metacarpus
- Phalanges



Hind Leg

- Femur
- Patella
- Tibia
- Fibula
- Tarsus
- Metatarsus
- Phalanges
- Head of femur articulates with acetabulum of pelvis



Pelvis

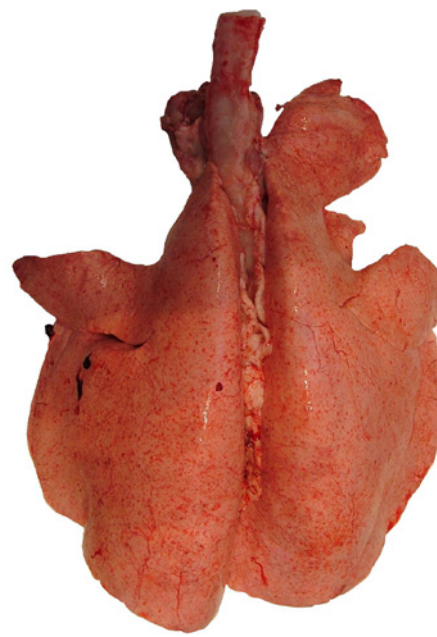
- 3 bones, ilium, ischium and pubis.
- Top of cavity formed by sacral vertebrae
- Paired pubis joined by pubic symphysis

Tongue

- Occasionally pigmented
- Dorsal prominence on upper surface
- Conical papillae on tip
- Fungiform papillae scattered on surface
- Lenticular papillae especially on dorsal prominence
- Vallate papillae grouped either side of dorsal prominence
- Weight 250-300g

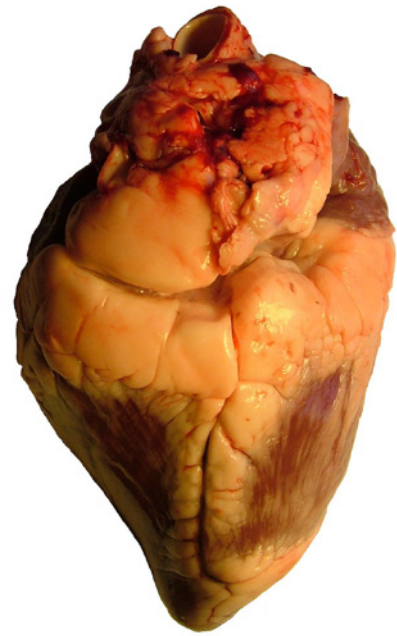
Trachea / Lungs

- Approximately 50-55 cartilage rings
- Overlap of rings creates dorsal ridge
- 3 main bronchi, one to each lung and 1 accessory bronchus to right apical lobe
- Right lung has 4 lobes
- 1 apical
- 2 cardiac
- 1 diaphragmatic
- Left lung has 3 lobes
- 1 apical
- 1 cardiac
- 1 diaphragmatic
- Lobulation indistinct
- Lungs covered by visceral pleura
- Weight 400g-1Kg



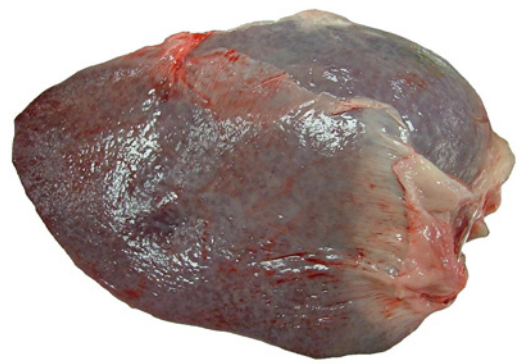
Heart

- Fat is crispy due to stearin
- 2 bones present at aortic origin in older animals
- 3 ventricular furrows
- 4 chambers, left atrium and ventricle, right atrium and ventricle.
- Average heartbeat 70-120 bpm at rest.
- Weight 100-150g



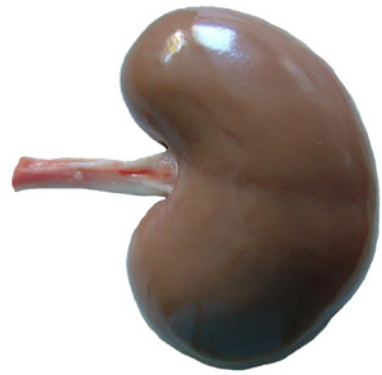
Spleen

- Flat and oval in outline
- Bluish/red in colour
- White Malphigian corpuscles visible in red pulp when incised
- Attached to rumen
- Weight 50-100g



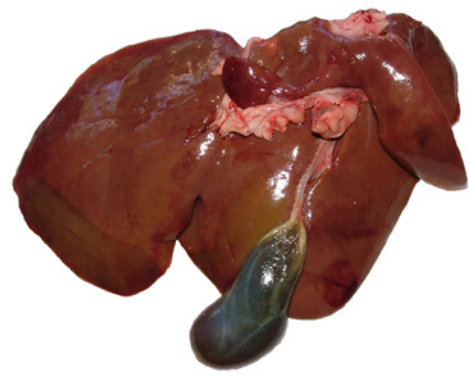
Kidneys

- Paired
- Right kidney held tight to body
- Left loose, more mobile
- Reddish brown in colour
- Weight 60-90g



Liver

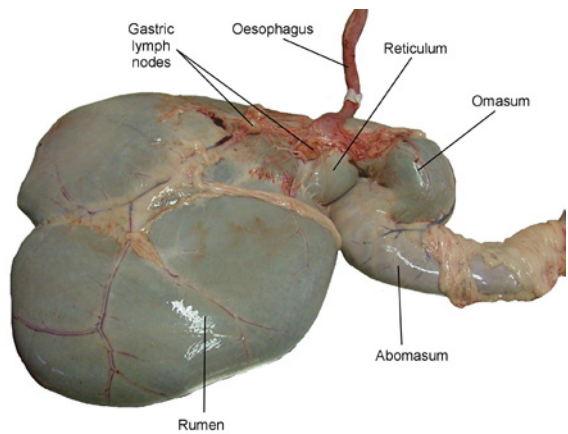
- 3 lobes, left, right and caudate lobe
- Cigar shaped gall bladder
- Renal impression at base of caudate lobe.
- Umbilical groove on outer edge of left lobe.
- Hepatic lymph nodes surround portal opening
- Weight 0.5-1Kg



Forestomachs

Consists of:

- Rumen
- Reticulum
- Omasum
- Abomasum



Rumen

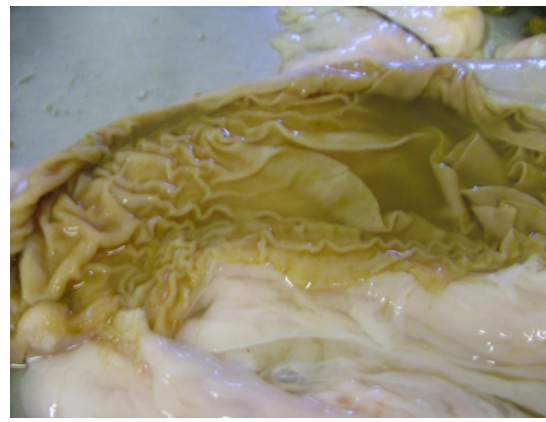
- First stomach compartment
- Fermentation vessel, food moved in churning motion.
- Leaf-like papillae
- Protozoa and bacterial colonies present that act on ingesta
- Food bolus regurgitated and remasticated providing further mechanical breakdown and addition of saliva before re-entering rumen

Reticulum

- Second stomach compartment
- Also known as the 'honeycomb'

Omasum

- Third stomach compartment
- Also known as 'manyplies', 'bible', 'psalterium'. Refers to tightly packed laminae.
- Ingesta forced between laminae and ground, smaller particles pass into abomasum

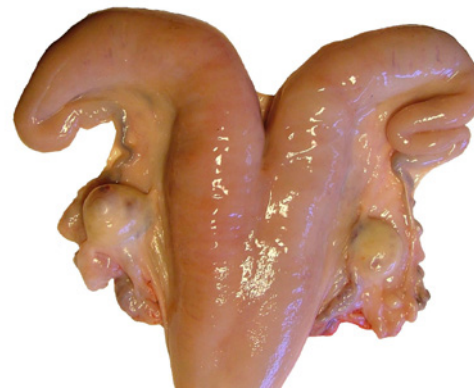


Abomasum

- True gastric stomach
- Glandular cells in mucosa secrete gastric juice

Uterus

- Y-shaped muscular organ
- Size varies with age
- Two horns (cornua) lead to ovaries
- When horns are separated a single fold of the intercornual ligament is visible
- Ovaries appear cystic.
- Yellowish formation on ovaries follows release of ova into cornua.
- Muscular cervix separates uterus from vagina
- Uterus situated ventrally to rectum



Testicle

- Heavily veined and oval in shape.
- Prominent conical swelling at base formed by tail of epididymis.
- Epididymis leads to ductus deferens (vas deferens) that carries sperm to urethra.
- Internal surfaces yellowish and lobulated.
- Weight 250-300g

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CONDITION AND CAUSE

Disease	*Aetiology*	*Type*
Actinobacillosis (Leather lips, 'Cruels')	*Actinobacillus ligniersi*	Bacterial
Actinomycosis (Lumpy Jaw, Ray Fungus)	*Actinomyces bovis*	Bacterial
Anthrax (Wool sorters disease, Splenic fever, Charbon, Malignant pustule)	*Bacillus anthracis*	Bacterial
Babesiosis (Haemosporidiosis, Piroplasmosis, Red Water Fever)	*Babesia motasi* *Babesia ovis*	Parasitic
Bacillary Haemoglobulinuria (Red Water Disease)	*Clostridium haemolyticum*	Bacterial
Black Disease (Infectious necrotic hepatitis)	*Clostridium novyi*	Bacterial
Blackleg (Black quarter)	*Clostridium chauvoei*	Bacterial
Bloat	Miscellaneous	
Bluetongue (Ovine catarrhal fever, 'sore muzzle' disease)	Orbivirus	Viral
Braxy	*Clostridium septicum*	Bacterial
Brucellosis (Ovine brucellosis, OB)	*Brucella melitensis*	Bacterial
Campylobacteriosis (Ovine Genital Campylobacteriosis)	*Campylobacter fetus* *Campylobacter jejuni*	Bacterial
Caseous Lymphadenitis (CLA. CL. Cheesy Gland. Pseudotuberculosis)	*Corynebacterium pseudotuberculosis*	Bacterial
Contagious Epididymitis (Ram Epididymitis)	*Brucella ovis*	Bacterial
Coccidiosis	*Eimeria arloingi* *Eimeria crandallis* *Eimeria ovinoidalis*	Parasitic

Disease	*Aetiology*	*Type*
Coenuriosis (Gid, Sturdy)	**Cystic***: Coenurus cerebralis* **Adult***: Taenia multiceps* **Definitive Host**: Canine	Parasitic
Contagious Agalactia (Mycoplasmosis)	*Mycoplasma agalactiae*	Bacterial
C. ovis	**Cystic***: Cysticercus ovis* **Adult***: Taenia ovis* **Definitive Host**: Canine	Parasitic
Enterotoxaemia (Pulpy kidney, Overeating disease)	*Clostridium perfringens* Type D	Bacterial
Enzootic Abortion (Chlamydiosis)	*Chlamydia psittaci*	Bacterial
Fascioliasis (Fluke, Liver Fluke)	*Fasciola hepatica*	Parasitic
Foot and Mouth Disease (FMD, Apthous fever)	Apthovirus	Viral
Foot Rot (Malignant foot rot. Foot lure, foot foul, foul of the foot, Contagious ovine interdigital dermatitis, COID)	*Dichelobacter nodosus* *Fusobacterium necrophorum*	Bacterial
Grass Staggers (Grass tetany, hypomagnesaemia)	Magnesium deficiency	Nutritional
Green Wool Disease (Fleece rot)	*Pseudomonas aeruginosa*	Bacterial
Hydatidosis	**Cystic***:* Hydatid **Adult***: Echinococcus granulosus* **Definitive Host**: Canine	Parasitic
Haemorrhagic Enteritis (Bloody scours, Struck, Romney Marsh Disease)	*Clostridium perfringens* Type C	Bacterial
Jaagsiekte (Ovine pulmonary adenomatosis, Ovine pulmonary carcinoma, Driving sickness)	Retrovirus	Viral

Disease	*Aetiology*	*Type*
Jöhne's Disease (Paratuberculosis)	*Mycobacterium johnei*	Bacterial
Listeriosis (Circling disease)	*Listeria monocytogenes*	Bacterial
Louping Ill (Ovine encephalomyelitis)	Flavivirus	Bacterial
Lungworm (Husk, Hoose)	*Dictyocaulus filaria* *Muelleris capillaris* *Protostrongylus rufescens*	Parasitic
Maedi-Visna *(Maedi, Ovine Progressive Interstitial Pneumonia)*	Lentivirus	Viral
Malignant Oedema (Big head)	*Clostridium septicum*	Bacterial
Melioidosis	*Pseudomonas pseudomallei*	Bacterial
Nasal Bot	*Oestrus ovis* **Fly larvae**	Parasitic
Orf (Contagious ecthyma, contagious pustular dermatitis, 'sore mouth')	Pox virus	Viral
Ovine Infectious Keratoconjunctivitis (Pinkeye, Contagious ophthalmia)	*Mycoplasma conjuntivae*	Bacterial
Pasterurellosis (Enzootic pneumonia)	*Pasteurella haemolytica*	Bacterial
Peste de petits ruminants (Stomatitis-pneumoenteritis complex, PPR)	Morbillivirus	Viral
Post Dipping Lameness	*Erysipelothrix rhusiopathiae*	Bacterial
Pregnancy Toxaemia (Twin Lamb Disease, hypoglycaemia, hyperketonaemia)	Metabolic Disorder	Nutritional
Ringworm (Club lamb fungus, Wool rot, Lumpy wool)	*Trichophyton verrucosum*	Fungal

Disease	*Aetiology*	*Type*
Rumen Fluke	*Paramphistomum cervi*	Parasitic
Salmonellosis	**Abortion**: *Salmonella abortus ovis* *Salmonella montevideo* **Enteritis / Septicaemia:** *Salmonella typhimurium* *Salmonella dublin*	Bacterial
Sarcocystosis	*Sarcocystis gigantea*	Parasitic
Scald (Foot Scald, Necrobacillosis of the foot)	*Fusobacterium necrophorum*	Bacterial
Scrapie (Rida, Tremblante du mouton)	Prion protein	Prion
Sheep Pox	Pox virus	Viral
Strawberry Foot Rot (Proliferative dermatitis)	*Dermatophilus congolensis*	Bacterial
Swayback (Enzootic ataxia)	Copper deficiency	Nutritional
Tenuicollis	**Cystic***: Cysticercus tenuicollis* **Adult***: Taenia hydatigena* **Definitive Host**: Canine	Parasitic
Tetanus (Lock-jaw)	*Clostridium tetani*	Bacterial
Tick Borne Fever (TBF)	*Ehrlichia phagocytophilia*	Bacterial
Tick Pyaemia (Staphylococcal pyaemia, Enzootic staphylococcosis)	*Staphylococcus aureus*	Bacterial
Toxoplasmosis	*Toxoplasma gondii* **Definitive Host**: Feline	Parasitic
Ulcerative Dermatosis (Venereal balanoposthitis, lip and leg ulceration)	Paravaccinia	Viral
White Liver Disease	Cobalt deficiency	Nutritional

Disease	*Aetiology*	*Type*
White Muscle Disease (Stiff lamb disease, nutritional muscular dystrophy, WMD)	Vitamin E / Selenium deficiency	Nutritional
Yersiniosis	*Yersinia pseudotuberculosis* and *Yersinia enterocolitica*	Bacterial

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AFFECTIONS OF SPECIFIC PARTS

ANTE MORTEM INSPECTION OF SHEEP

Many things can hamper ante mortem inspection of sheep but one factor predominates: sheep are almost the ultimate prey species and as such will attempt to appear fit and healthy in the presence of a predator (humans), disguising clinical signs of disease or weakness due to injury.

Given the time constraints of ante mortem inspection, the reaction of the animals to the preceding transportation, strange lairage environment, and the factors above, the clinical signs of disease, trauma or other animal health and welfare issues can be barely noticeable; a fact that leads to sheep being the forgotten species in terms of welfare. The greatest tool available to those conducting ante mortem inspection of sheep is experience, especially of normality in the species and as such this section gives a guide to ante mortem conditions based on deviation from the norm for sheep in the setting of the lairage environment under the following headings: Abnormality of behaviour, Abnormality of breathing, Abnormal discharges, Abnormality of movement/posture, Abnormality of shape / condition and Abnormality of skin/eyes/mucous membranes.

The process of ante mortem inspection should ideally be performed with the animals in movement and at rest, and preferably with the opportunity to view the animal from both sides in cases of conditions that may be unilateral. Movement obviously allows for checking gait, locomotion and central nervous system lesions; monitoring resting behaviour can demonstrate signs of conditions such as pruritus and laboured breathing.

PHYSIOLOGICAL DATA

Rectal temperature	39.5°C ± 0.5°C
Heart rate	60-120 beats per minute, average 75 bpm
Respiratory rate	19 breaths per minute

DEAD ON ARRIVAL/IN LAIRAGE

Anthrax, Black Disease, Bluetongue, Braxy, Enterotoxaemia, Haemorrhagic enteritis, Salmonellosis, Tetanus

ELEVATED TEMPERATURE

Bacillary Haemoglobulinuria, Black Disease, Blackleg, Bluetongue, Braxy, Foot and mouth disease, Leptospiridiosis, Louping Ill, Malignant oedema, Pasteurellosis, Peste Des Petits Ruminants, Salmonellosis, Tetanus, Tick borne fever, Sarcocysts, Toxoplasmosis

ABNORMALITY OF BEHAVIOUR

Clinical sign	*Possible diagnosis*
Aggression	Scrapie
Apparent blindness	Pregnancy toxaemia
Cessation of rumination	Bacillary Haemoglobulinuria, Bloat, Braxy,
Circling	Listeriosis, Meningitis, Coenurus cerebralis, Chronic Maedi-Visna
Collapse and rapid kicking	Enterotoxaemia, Tetanus
Dullness	Black Disease, Braxy, Enterotoxaemia, Pasteurellosis, Pregnancy toxaemia, Tick pyaemia
Loss of appetite	Blackleg, Ostertagiosis,
Nervous nibbling	Louping ill, Sheep ked
Straining	Haemorrhagic enteritis
Teeth grinding	Enterotoxaemia, Scrapie

ABNORMALITY OF BREATHING

Clinical sign	*Possible diagnosis*
Coughing	Peste Des Petits Ruminants, Tuberculosis, *Dictyocaulus filaria*
Discharge	Actinobacillosis, *Oestrus ovis,* Peste Des Petits Ruminants
Discharge - frothy	Jaagsiekte
Laboured breathing	Pleurisy, Bloat, Jaagsiekte, Maedi-Visna, Meliodosis, pasteurellosis, pneumonia, Sheep pox, Peste Des Petits Ruminants, *Dictyocaulus filaria, Toxoplasmosis*
Rapid breathing	Bacillary Haemoglobulinuria, Tick borne fever, Pleurisy, Pneumonia,
Snoring, breathing sounds associated with nasal obstruction	Actinobacillosis, *Oestrus ovis,* Orf

ABNORMAL DISCHARGES

Clinical sign	*Possible diagnosis*
Dark green/black diarrhoea	Yersiniosis
Dark red urine	Bacillary Haemoglobulinuria, babesiosis, Haemorrhagic cystitis
Diarrhoea	Stress, Haemorrhagic enteritis, Jöhne's Disease**,** Peste Des Petits Ruminants, Paramphistomosis
Excessive salivation	FMD, Listeriosis
Green,pasty diarrhoea	Enterotoxaemia, Ostertagiosis

Muco-purulent or bloody nasal discharge	Bluetongue, *Oestrus ovis*
Severe, foul smelling, very fluid diarrhoea	Salmonellosis
Sticky granulatamous pus from facial fistula	Actinomycosis
Vulval discharge	Metritis, Pyometra, Enzootic abortion

ABNORMALITY OF MOVEMENT/POSTURE

Clinical sign	*Possible diagnosis*
Facial paralysis	Listeriosis, Tetanus
Fore and hind legs jumping forward in pairs	Louping ill, polyarthritis,
Inability to move	Black Disease
Lameness	Blackleg, Bluetongue, Contagious agalactiae, FMD, Trauma, Joint ill, Arthritis, Foot Rot, Scald, Malignant oedema, Meliodosis, Post-Dipping Lameness, Tick pyaemia
Muscular tremors	Louping ill, Scrapie, *Coenurus cerebralis*, Toxoplasmosis
Rear end paralysis	Louping ill, Chronic Maedi-Visna, Swayback
Stiffness	White muscle disease
Unsteady gait	Black Disease, *Coenurus cerebralis*

ABNORMALITY OF SHAPE / CONDITION

Clinical sign	*Possible diagnosis*
Emaciation	Jöhne's Disease, Haemorrhagic enteritis, Maedi-Visna, Peste Des Petits Ruminants, Salmonellosis, Tuberculosis, Caseous lymphadenitis, Ostertagiosis, Paramphistomosis
Enlargement of peripheral lymph nodes	Caseous lymphadenitis,
Oedema of face and jaw	Bluetongue
Subcutaneous emphysematous crepitation	Blackleg
Submandibular oedema	Hepatitis, Fascioliasis, Ostertagiosis
Swellings in the hip, shoulder, chest and back	Blackleg, Malignant oedema,
Swollen abdomen	Bloat. Pregnancy
Swollen painful bones, especially the mandible and maxilla	Actinomycosis, Dental caries

ABNORMALITY OF SKIN/EYES/MUCOUS MEMBRANES

Clinical sign	*Possible diagnosis*
Anaemia	Jöhne's Disease, Haemorrhagic enteritis, Leptospiridiosis, *Haemonchus contortus*, Sarcocysts, Coccidiosis

Blackened raised pustule surrounded by oedema	Cutaneous anthrax
Conjunctivitis, corneal ulceration	Trauma, Contagious agalactiae, Infectious Keratoconjunctivitis, Peste Des Petits Ruminants
Erosion of buccal mucosa	Bluetongue, Orf, FMD
Greenish matted wool	Green wool disease,
Hyperaemia and oedema of muzzle	Bluetongue
Pocks, scabs or pustules on un-woollen parts of skin especially the muzzle, lip and perianal areas	Sheep pox, Orf
Proliferative scab on coronary band	Strawberry footrot, Orf
Thick, scaly pustules on the head, neck or back	Mange, Ringworm
Ulceration of penis/prepuce or vulva	Ulcerative Dermatosis
Vesicle formation	FMD, Orf, Sheep pox, Ulcerative Dermatosis
Wool brittle and easily removed	Jöhne's Disease,
Wool loss	Scrapie, Mange, Ringworm

POST MORTEM

LESION	POSSIBLE DIAGNOSIS
ABDOMEN	
Inflammation of peritoneum	Peritonitis
Peritonitis	Haemorrhagic enteritis, trauma, metritis, ascending mastitis, contact spread of visceral peritonitis, trauma, parasites
Gross distension	Bloat
Fluid filled pendulous cysts	*Cysticercus tenuicollis*
Fat becomes hard, granular and 'soapy'	Fat necrosis
BONES	
Inflammation of	Osteitis
Purulent inflammation of	Osteomyelitis
Softening, swelling and curvature	Rickets (vitamin D deficiency)
Callous formation	Resolving fracture, neoplasia
BLADDER	
Inflammation of	Cystitis
Dark red urine	Bacillary haemoglobinuria, Babesiosis, Uroliths, Haemorrhagic cystitis
FEET	
Inflammation of laminae	Laminitis
Inflammation of interdigital cleft	Scald
Laminitis	Post-dipping lameness, foot rot
Strawberry-like proliferative growths	Strawberry foot rot, orf
Ulceration	Foot and mouth lesions, orf, Ulcerative dermatosis
Foul odour	Foot rot, white line abscess, impaction
HEAD	
Bones	
Swelling of mandible/ other bones	Actinomycosis, abscessation, traumatic infection
Buccal cavity	
Inflammation of	Stomatitis
Erosion of cheek papillae	Bluetongue
Erosion of mucous membrane	Peste des petits ruminants
Hyperaemia of muzzle and lips	Bluetongue
Necrotic areas on pharynx	Trauma, (drenching gun injury), sheep pox

LESION	POSSIBLE DIAGNOSIS
Vesicles on dental pad	Foot and mouth disease, Orf
Eyes	
Ulceration of cornea	Ovine Infectious keratoconjunctivitis, trauma, grass awns, Contagious agalactia
Conjunctivitis	Ovine infectious keratoconjunctivitis, trauma, fly irritation
Lymph Nodes	
Necrotic/caseous/calcified lesions	Actinobacillosis, tuberculosis, Pyogenic bacteria
Abscessation (laminated or cheese like)	Caseous lymphadenitis
Abscessation (Yellow-green granular pus)	Actinobacillosis, tuberculosis, Pyogenic bacteria, Tick pyaemia
Muscle	
Pea sized cyst (calcified/caseated) single scolex	*Cysticercus ovis*
Black 'ink splashes'	Melanosis
Skin	
Scabby, scaly, loss of hair	Ringworm, mange, scrapie, rubbing injury
Scabby pustules	Orf, Sheep pox, Ulcerative dermatosis
Nasal passages / sinuses	
Nodules	Actinobacillosis
Larvae	*Oestrus ovis*

HEART	
Inflammation of heart muscle	Myocarditis
Inflammation of heart surface	Epicarditis
Inflammation of internal structures	Endocarditis
Inflammation of pericardium	Pericarditis
Black 'ink splashes'	Melanosis
Excess blood within pericardium	Haemopericardium
Excess fluid within pericardium	Hydropericardium
Fluid filled cysts containing 'sand'	Hydatid cyst
Haemopericardium	Enterotoxaemia
Haemorrhage	Blood splash, Pasteurellosis (systemic), trauma, Septicaemia, toxaemia, Salmonellosis
Hydropericardium	Black disease, Enterotoxaemia, Salmonellosis
Iridescent Greenish/yellow areas of discolouration	Eosinophilic myositis
Necrotic areas	Listeriosis

LESION	POSSIBLE DIAGNOSIS
Opaque serous membrane	Pericarditis (traumatic, fibrinous or septic)
Pale areas of necrosis	Blue tongue, foot and mouth disease
Pea sized cyst (calcified/caseated) single scolex	*Cysticercus ovis*
Petechial haemorrhages	Enterotoxaemia
Petechial haemorrhages at base of aorta	Blue tongue, toxaemia
Tumours	Lymphosarcoma
Whitish plaques in endocardium	White muscle disease
Yellow fat	Jaundice, Babesiosis, carotene pigmentation, xanthrophyll pigmentation
Yellowish/white areas around coronary furrows	White muscle disease

INTESTINES

Inflammation of	Enteritis
Congested and oedematous small intestine	Yersiniosis, non-specific enteritis, Coccidiosis, Bacilliary haemoglobinuria
Dark blue/purple discolouration	Haemorrhagic enteritis
Haemorrhages	Coccidiosis, Haemorrhagic enteritis, listeriosis, Salmonellosis
Haemorrhagic streaks in upper duodenum	Peste des petits ruminants
Striped appearance of caecum and colon	Peste des petits ruminants
Sub mucosal Haemorrhage	Coccidiosis
Thickened and corrugated mucosa	Jöhne's Disease
Ulcerative enteritis	Yersiniosis
Yellow pigmentation of bowel mucosa	Jöhne's Disease

JOINTS

Inflammation of	Arthritis
Arthritis	Tick pyaemia, Erysipelas, non-suppurative polyarthritis, 'joint-ill', Maedi-Visna
Fluid filled cysts becoming fibrous over limb joints	Trauma, pressure swellings, bursitis
Polyarthritis (fibrinous)	Contagious agalactia, joint ill, post-dipping lameness

KIDNEYS

Inflammation of	Nephritis

LESION	POSSIBLE DIAGNOSIS
Abscesses	Caseous lymphadenitis, pyelonephritis
Change of state	Nephrosis
Crystalline 'balls' or stones	Renal calculi
Enlarged and fluid filled	Hydronephrosis, retention cysts, blockage of ureter
Fatty degeneration	Enterotoxaemia, septicaemia, anthrax, Pregnancy toxaemia
Grain sized cysts	Congenital cysts
Interstitial nephritis	Leptospiridiosis, toxins
Petechial haemorrhages	Bacillary haemoglobinuria, septicaemia, toxaemia
Slimy fluid in renal pelvis	Pyelonephritis
White patches, pyramidal in section	Infarcts, embolic infarction
White/yellow raised foci	Abscessation, caseous lymphadenitis, infarction

LIVER

Inflammation of	Hepatitis
Abscesses	Meliodosis, Tick pyaemia, bacterial necrosis, arrested parasites
Adhesion between liver and diaphragm, or alimentary tract	Peritonitis, traumatic peritonitis
Black 'ink splashes'	Melanosis
Darkened, with necrotic patches	Black disease
Distended bile ducts	Fascioliasis, cholecystitis
Enlargement	Hepatitis, babesiosis
Fluid filled cyst(s) containing 'sand' in liver tissue	Hydatidosis
Fluid filled sac on liver surface containing single white scolex	*C.tenuicollis*
Fluke,	Fascioliasis.
Focal depressed bluish black spots throughout liver substance	Telangiectasis
Haemorrhages	Pasteurellosis
Infarcts with surrounding hyperaemia	Bacillary haemoglobinuria, Black disease
Liver is greasy and friable to the touch	Fatty change, septicaemia/toxaemia, anthrax, Pasteurellosis, Pregnancy toxaemia, White liver disease, Plant toxin
Liver tissue replaced by fibrous tissue	Cirrhosis
Lymphoid growths in liver	Tumours,
Multifocal fibrosis	Migratory fluke
Focal necrosis	Listeriosis, bacterial necrosis

LESION	POSSIBLE DIAGNOSIS
Reddish patches throughout substance	Haemangioma
Serpentine tracts on surface	Scarring produced by migrating larvae of *Taenia hydatigena*
Small pale necrotic foci in liver tissue	Bacterial necrosis, toxoplasmosis

LUNGS

Inflammation of lung surface	Visceral pleurisy, pleuritis
Inflammation of lung tissue	Pneumonia
Abscessation of lung tissue	Melioidosis, Tick pyaemia, Pasteurella pneumonia
Abscessation of bronchial lymph nodes	Caseous lymphadenitis
Acute inflammation of bronchi	Bronchitis, sheep pox
Air bubbles visible between lung septa	Emphysema
Black 'ink splashes' in substance	Melanosis
Firm masses in lung tissue	Abscesses, tuberculosis, hydatid cysts, Jaagsiekte, errant fluke
Fluid filled cysts	Hydatid cysts
Frothy fluid in bronchi	Jaagsiekte, suffocation, fluid aspiration
Hepatisation of cardiac lobes	Pasteurella pneumonia, non-specific pneumonia, aspiration pneumonia
Large area of lung or lobe hepatised or consolidated, well demarcated	Lobar pneumonia, pasteurellosis, lungworm, aspiration pneumonia, foreign body,
Lungs do not collapse	Jaagsiekte, Maedi-Visna, Pasteurella pneumonia
Necrotic foci	Orf
Reddish/white nodules in tracheal mucosa	Sheep pox
Shot-like lesions in substance	*Muellerius capillaris*
Small areas of pneumonia	Broncho-pneumonia, aspiration pneumonia
Thread-like pieces in bronchi	*Dictyocaulus filaria, Protostrongylus rufescens*
Tumours	Jaagsiekte, lymphosarcoma

LYMPH NODES

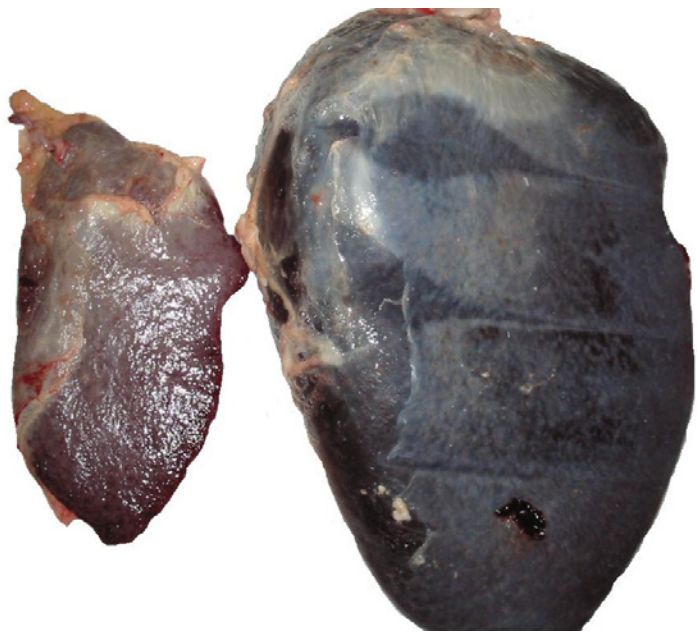
Inflammation of	Lymphadenitis
Abscessation	Pyogenic spread, actinobacillosis, tick pyaemia
Abscessation (cheesy, non odorous)	Caseous lymphadenitis, Melioidosis
Enlarged peripheral nodes	Caseous lymphadenitis, Mange, Sheep scab, Sheep pox, Green wool disease, Fly strike, Tick pyaemia
Greenish granular flecks in mesenteric lymph nodes	*Linguatula serrata*
Haemorrhagic / dark red	Anthrax, Septicaemia, Toxaemia, Blue tongue
Oedematous lymphadenitis	Peste des petits ruminants, Jöhne's Disease

LESION	POSSIBLE DIAGNOSIS
MAMMARY GLANDS	
Inflammation of	Mastitis
Lymphoid masses	Neoplasia
Udder swollen and hot, oedematous L.N.	Septic mastitis, Contagious Agalactia
Vesicles on teats	Foot and Mouth disease, trauma, Orf, Sheep pox
Violet red to greenish black tissue	Gangrene
MUSCLE	
Chalky white patches or striations	White muscle disease
Dark brown to black discolouration	Malignant oedema
Fatty change	Steatosis, resolving traumatic injury
Greenish foci in muscle fibres	Eosinophilic myositis
Greenish white oval cysts resembling cooked rice	Sarcocysts
Iridescent greenish/yellow areas of discolouration	Eosinophilic myositis
Jelly-like blood tinged infiltration	Resolving bruise, blackleg
Localised fibrosis	Interstitial fibrosis, muscular fibrosis, post traumatic scarring
Pale areas of necrosis	Blue tongue
Pea sized cysts (calcified/viable)	*Cysticercus ovis*
Swellings containing gas, discoloured serum	Blackleg, malignant oedema
White, hyaline areas	White muscle disease
OESOPHAGUS	
Longitudinal shallow erosion	FMD, Orf
Greenish white oval cysts resembling cooked rice	Sarcocysts
Granulomatous lesions in lower portion	Tuberculosis
Reddish-white nodules in mucosa	Sheep pox
SKIN	
Inflammation of	Dermatitis
Abscesses	Injection abscesses, Orf vaccine (between thighs), Tick pyaemia, fly strike
Cauliflower-like outgrowths	Papillomas

LESION	POSSIBLE DIAGNOSIS
Circumscribed elevated patches, scaly or scabby	Rubbing injury, ringworm
Dark colour due to vascular engorgement	Black disease
Denuded areas	Rubbing injury, ringworm, mange, scrapie, Green wool disease, Fly strike,
Ulceration of breast skin (Ram)	Raddle injury
Vesicles/ulceration of coronary band of foot	Foot and Mouth Disease, Foot rot, Orf

SPLEEN

Inflammation of	Splenitis, splenomegaly
Congestion	Salmonellosis
Cysts containing 'sand'	Hydatid cysts
Granular nodules	Tuberculosis, abscesses, Melioidosis
Gross enlargement	Anthrax, mounting damage, babesiosis, septicaemia, 'slaughter spleen', Peste des petits ruminants, trauma, Tick pyaemia
Haemorrhage	Pasteurellosis, Trauma
Necrotic foci	Listeriosis



Splenomegaly of the spleen on the right compared to normal spleen on left

LESION	POSSIBLE DIAGNOSIS
STOMACHS	
Inflammation of	Gastritis
Congestion of abomasum	Braxy, Salmonellosis
Grossly distended rumen	Bloat, Braxy, overfeeding
Haemorrhagic mucosa of abomasum	Trauma, gastritis, Braxy, Leptospiridiosis, toxins, Salmonellosis, Bluetongue
Raised plaques in mucosa - reticulum	Actinobacillosis
Ulceration of abomasal mucosa	Trauma, Leptospiridiosis

A ruminal bolus, a ceramic cylinder containing a transmitter by means of which animal identification is achieved

TESTICLES	
Inflammation of	Orchitis
Abscessation of testes, spermatic cord	Brucellosis, traumatic infection, caseous lymphadenitis
Orchitis	Injury, Contagious agalactia, Contagious Epididymitis
THORAX	
Inflammation of pleura	Pleurisy, pleuritis
Adhesion	Parietal pleurisy
Blood tinged hydrothorax	Bacillary haemoglobinuria
Excess blood in thoracic cavity	Haemothorax
Excess fluid in thoracic cavity	Hydrothorax

LESION	POSSIBLE DIAGNOSIS
Haemothorax	Blue tongue
Hydrothorax, straw coloured	Pasteurellosis, Black disease
Lumps on ribs	Resolving fractures, lymphosarcomas
TONGUE	
Inflammation of	Glossitis
Cyanosis of tongue	Bluetongue
Glossitis	Bluetongue
Nodules in substance	Orf, Actinobacillosis, injury, abscessation
Oedema	Bluetongue
Solid areas in tongue	Traumatic scar tissue
Ulceration of mucosa	Foot and Mouth Disease, Orf, actinobacillosis, trauma
Vesicles	Foot and Mouth Disease, Orf, Bluetongue
TRACHEA	
Inflammation of	Tracheitis
Acute inflammation	Non-specific tracheitis, injury, Drenching gun injury
Erosion of mucosa	Orf
Frothy fluid	Jaagsiekte
Red frothy fluid	Bleed out aspiration, suffocation
Petechial haemorrhages	Bacillary haemoglobinuria, tracheitis
Reddish-white nodules in mucosa	Sheep pox
UTERUS	
Inflammation of	Metritis
Grey necrotic foci	Campylobacteriosis
Gross enlargement, foul odour, brownish pus	Septic metritis
Pus in uterus	Pyometra
Serous yellow/red to brown discharge	Septic metritis, brucellosis, retained placenta
Yellow necrotic foci	Listeriosis
Yellowish grey thickened areas surrounding dark red cotyledons	Enzootic abortion

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