SECTION 6

Ante-mortem inspection

Ante-mortem inspectior

Ante-mortem inspection systems required by the competent authority should include the following:

- all relevant information from the level of primary production should be taken into account on an ongoing basis, e.g. declarations from the primary producers relating to the use of veterinary drugs, information from official hazard control programmes;
- animals suspected as being unsafe or unsuitable for human consumption should be identified as such and handled separately from normal animals;
- results of ante-mortem inspection should be made available to the competent person undertaking post-mortem inspection before animals are examined at the post-mortem stations so as to augment final judgement. This is particularly important when a competent person undertaking ante-mortem inspection judges that a suspect animal can proceed to slaughter under special hygiene conditions;
- in more equivocal situations, the competent person undertaking ante-mortem inspection may hold the animal (or lot) in special facilities for more detailed inspection, diagnostic tests and/or treatment;
- animals condemned as unsafe or unsuitable for human consumption should be immediately identified as such and handled in a manner that does not result in cross-contamination of other animals with food-borne hazards; and
- the reason for condemnation should be recorded, with confirmatory laboratory tests being carried out if deemed necessary. Feedback of this information to the primary producer should take place.



Ante-mortem judgement categories include:

- passed for slaughter;
- passed for slaughter subject to a second ante-mortem inspection after an additional holding period, e.g. when animals are insufficiently rested, or are temporarily affected by a physiological or metabolic condition;
- passed for slaughter under special conditions, i.e. deferred slaughter as "suspects", where the competent person undertaking antemortem inspection suspects that post-mortem inspection findings could result in partial or total condemnation;
- condemned for public health reasons, i.e. due to meat-borne hazards, occupational health hazards or likelihood of unacceptable contamination of the slaughter and dressing environment following slaughter;
- · condemned for meat suitability reasons;
- emergency slaughter, when an animal eligible for being passed under special conditions could deteriorate if there were a delay in slaughter; and
- condemned for animal health reasons, as specified in relevant national legislation, and disposed of accordingly.

Source: FAO/WHO, 2004.

Diseases covered in this section

GENERAL PATHOLOGICAL CONDITIONS

Fever (pyrexia)	10
Inflammation in viral diseases	10
Septicaemia	10
Toxaemia	11
Bruises	11
Abscesses	12
Emaciation	12
Oedema	13
Immaturity	14
Plant poisoning	14
Chemical poisoning	14

SPECIFIC DISEASES

DISEASES CAUSED BY VIRUSES

Foot-and-mouth disease (FMD)	15
Rinderpest (RP)	17
Vesicular stomatitis (VS)	18
Malignant catarrhal fever (MCF)	19
Rabies	20
Lumpy skin disease	20
Bovine herpes dermophatic disease (BHD)	21
Infectious bovine rhinotracheitis (IBR)	22
Bovine viral diarrhoea (BVD)	23
Bovine leukosis	23

DISEASE CAUSED BY PRIONS

Bovine spongiform encephalopathy (BSE, "mad cow disease")	24
DISEASES CAUSED BY RICKETTSIA AND MYCOPLASMA SPP.	
Heartwater (hydropericardium) Q fever (Queensland fever, Nine mile fever, American Q fever, Australian Q fever) Contagious bovine pleuropneumonia (CBPP)	25 25 26

DISEASES CAUSED BY BACTERIA

Blackquarter (blackleg) Botulism	27 28
Malignant oedema	29
Tuberculosis	30
Johne's disease (bovine paratuberculosis)	31
Leptospirosis	32
Brucellosis (contagious abortion, Bang's disease)	33
Anthrax	34

Salmonellosis in bovines	35
Haemorrhagic septicaemia	36
Calf diphtheria	36
Actinobacillosis ("wooden tongue")	37
Actinomycosis ("lumpy jaw")	37
Pyelonephritis (contagious bovine pyelonephritis)	37
Metritis	38
Mastitis	38
Endocarditis	38
Traumatic reticuloperitonitis (TRP, hardware disease,	
traumatic gastritis, traumatic reticulitis)	39

PARASITIC DISEASES

Diseases caused by helminths	
Lungworms	40
Fascioliasis	41
Dicrocelium dendriticum infestation	42
Oesophagostomiasis (pimply gut, nodular worms)	42
Cysticercosis	43
Hydatid disease (hydatidosis, echinococcosis)	44
Onchocercosis	44
Diseases caused by protozoa	
Trypanosomiasis	45
Theileriosis (East Coast fever)	46
Besnoitiosis	46
Anaplasmosis (gall sickness)	47
Babesiosis (piroplasmosis, Texas fever, red water fever, tick fever)	47
Sarcocystosis (sarcosporidiosis)	48
Dispasos causod by arthropod parasitos	

Diseases caused by arthropod parasites	
Hypoderma bovis infestation	50
Screwworm myiasis	51

INTRODUCTION

Inspection of the live animal prior to slaughter is an important step in the production of wholesome meat for human consumption. Only in the live animal can abnormalities of posture, movement and behaviour be detected. Antemortem inspection can improve the efficiency of the operation by screening out a number of animals that would be unfit for consumption. This section outlines the process of ante-mortem inspection and indicates the actions that can be taken to protect human and animal health.

Meat inspection is commonly perceived as the sanitary control of slaughter animals and meat. The aim of meat inspection is to provide safe and wholesome meat for human consumption. Meat inspection covers both ante- and postmortem inspection.

The responsibility lies primarily with the relevant public health authorities who are represented by veterinarians and meat inspectors at the abattoir stage.

In many developing regions and in particular in rural abattoirs, meat inspectors often lack the necessary information and guidelines to assess the sanitary status of carcasses, meat and organs from slaughter animals. Therefore this section and Section 8 on post-mortem inspection provide concise guidelines on the subject, together with colour illustrations demonstrating the pathological lesions that may occur in bovines, small ruminants, pigs, game, poultry and rabbits. The statements made on the judgement of diseased carcasses or parts of the carcasses are recommendations that are also influenced by the need to salvage as much meat as possible for human consumption. These recommendations are not meant to interfere with any existing regulations on the subject in individual countries.

AIMS OF ANTE-MORTEM INSPECTION

The primary aim in all inspections throughout the meat-processing operation is the protection of the consumer from zoonotic or meat-borne disease. Ante-mortem inspection also increases the protection of slaughter personnel from disease, these people being the first in the chain to have direct contact with the animal and its products. A further aim is to protect animal health. The slaughterhouse receives animals from many origins, and is an ideal place to monitor the health of the livestock in the local area. Animal diseases that have severe consequences for national animal health, trade and the economy are often notifiable, and the ante-mortem inspection at the slaughterhouse can be a significant earlydetection point for such diseases. The third aim of ante-mortem inspection is to monitor and improve animal welfare, by the detection of existing or potential problems, and the implementation of appropriate control measures.

THE PROCESS OF ANTE-MORTEM INSPECTION

Ante-mortem inspection should ideally be carried out at the time of the animals' arrival at the slaughterhouse. There needs to be sufficient natural or artificial light to allow observation of the animals in motion and also at rest. At the time of arrival, the condition of the transport vehicle can also be assessed, and if an animal has suffered injury during transport, action can be taken to prevent further suffering.

If it is not possible to carry out inspection at the time of the animals' arrival, an inspection should be carried out within 24 hours after arrival, again to prevent further suffering in the event of a welfare problem.

The ante-mortem inspection should also be carried out within the 24-hour period prior to slaughter, as signs of disease may become manifest as time progresses. Where animals remain in the lairage for longer periods of time, they may be inspected more than once.

In the ideal situation, the ante-mortem inspection will be accompanied by examination of information relating to the animal's life and health history (Photos 6.1 and 6.2). This "chain information" can give useful indications of the overall health status of the herd or flock of origin, the possibility of there being chronic lesions in the carcass produced, and a guarantee that there will be no chemical residues in the meat as a result of medications administered or pesticides used.



PHOTO 6.1 A veterinary inspector examines information relating to the animal's life and health history

Ante-mortem inspection has two components:

- the screening of animals and segregation of animals suspected of being diseased or in an unsatisfactory condition;
- veterinary examination and diagnosis of the screened-out animals.

In general, any animal that deviates from normal should be segregated during the initial screening process. There are some exceptions of minor significance, such as cows with one horn or with an extra teat, minor cuts, etc.

Some suggested abnormalities to look out for in the initial screening process are listed below.

Abnormalities in breathing

Usually this refers to frequency of respiration, but there are also other abnormalities, such as frequent coughing and difficulty in breathing. The main point to remember is that if the breathing pattern differs from normal, the animal should be screened out.

Abnormalities in behaviour

Abnormalities in behaviour can be significant in some very serious diseases, such as rabies, bovine spongiform encephalopathy (BSE) and lead poisoning.

Examples of abnormal behaviour are:

- an animal pushing its head against the wall;
- · an animal walking in circles;
- · an animal charging at various objects;
- an animal with an anxious expression in its eyes;
- · an animal with a dull expression in its eyes;
- an animal that is acting very aggressively. Animals that behave in an abnormal way

should be screened out at the time of ante-

mortem inspection. Special attention should be given to ensuring that the animal will not pose a danger to other animals or to humans.

Abnormalities in gait

When an animal has an abnormal gait or is reluctant to move, it usually indicates that there is pain somewhere. The animal may be suffering from abnormalities anywhere in its legs or may have pain in the chest or abdomen. This may also indicate nervous disorders.

Abnormalities in posture

An animal with abnormal posture:

- may stand with the abdomen tucked in;
- may lie with its head turned and along its side;
- may stand with its feet stretched out in front;
- may stand with its head and neck extended;
- may be unable to rise.

Normal animals may sometimes temporarily assume postures that may be mistaken for abnormal postures, e.g. a cow that has rested for a long time may stretch and stand with its legs out in front as in some disease conditions; also, resting cattle sometimes have their heads turned along their sides. In normal animals this posture disappears when the animal is stimulated.

The most frequently observed abnormal posture is of course the "downer". Downers are any animals that cannot stand or can only stand for short periods. Such animals must be handled without causing undue suffering and are usually segregated on initial ante-mortem inspection. If they cannot be segregated, operations should cease so that they may be dealt with. After veterinary inspection, downers must be stunned in the yard if moving them causes undue pain, and sent directly to the appropriate bleeding area.

Abnormal discharges or protrusions from body openings

The normal animal has no discharges or protrusions from its body openings. Examples of abnormal discharges or protrusions from the body are:

- discharge from the nose;
- bloody diarrhoea;
- excessive saliva coming out of the mouth;
- afterbirth hanging out of the vulva;
- calf leg protruding from the vulva;

PHOTO 6.2 A veterinary inspector examines information relating to the animal's identification



EICH, SWITZERLAND

- · intestine protruding from the rectum;
- uterus protruding from the vulva;
- growth protruding from the eye.

Abnormal colour

Abnormal colour is generally not as important as the other abnormalities, but the inspector

- should be on the lookout for this. Examples are:
- black areas on the skins of pigs;
- red areas in light-coloured skin (inflammation);
- dark blue areas, e.g. gangrenous udder;
- yellow coloration of the sclera of the eye or skin (jaundice).

Abnormalities in appearance (conformation)

Inspectors will see many of these. Whenever there is a change in the normal conformation of an animal, a disease process should be suspected. Examples are:

- swelling of the skin (abscesses);
- enlarged joints;
- swelling of the umbilicus;
- greatly enlarged udder;
- bloated abdomen;
- swollen legs;
- enlarged jaws ("lumpy jaw");
- pendulous lower abdomen (hanging down);
- swelling of subcutaneous lymph nodes.
- In some instances it is helpful to compare both sides of the animal to find discrepancies. Any

animal affected with the above abnormalities or other abnormalities of conformation should be segregated for veterinary inspection.

Abnormal odour

This is often difficult to detect on ante-mortem inspection. Examples of odours found at antemortem are stinkweed, medicinal or punctured abscess odours. The inspector should hold the animals for veterinary inspection whenever there is suspicion that an animal is affected with an abnormal odour.

The initial inspection will allow normal animals to proceed to slaughter while those showing any abnormality should be separated into an isolation facility and kept for further inspection and, if necessary, a full examination by an appropriate competent person.

OUTCOMES OF ANTE-MORTEM INSPECTION

For the isolated animals, a detailed inspection follows the initial separation. The animals could be judged into any one of the following categories that have been proposed in the *Draft code of hygienic practice for meat* (FAO/WHO, 2004):

- Passed for slaughter. These are animals that have been judged normal and so can proceed to slaughter without undue delay.
- Passed for slaughter subject to a second antemortem inspection. A second ante-mortem inspection should be carried out after an additional holding period. Animals that would fall in this category include those that have been insufficiently rested, or are temporarily affected by a physiological or metabolic condition.
- Passed for slaughter under special conditions. Where the competent person undertaking ante-mortem inspection suspects that postmortem inspection findings could result in partial or total condemnation, the animals are treated as "suspects". Their slaughter is deferred, preferably to the end of normal slaughter.
- **Condemned**. Animals could be condemned for:
 - public health reasons due to the presence of meat-borne hazards, occupational health hazards or the likelihood of

unacceptable contamination of the slaughter and dressing environment following slaughter;

- meat suitability reasons;
- animal health reasons animals in this category are treated as specified in relevant national legislation, and disposed of accordingly.
- Emergency slaughter. This judgement is made when an animal eligible for being passed under special conditions could deteriorate if there were a delay in slaughter.

ANIMALS THAT DO NOT FALL INTO THE "NORMAL" CATEGORY

An animal condemned for signs of serious animal disease should be removed from the slaughter process immediately. In the event of this disease being a notifiable disease, it must be reported immediately to the competent authority, and the carcass destroyed and disposed of in accordance with legislative requirements. If the animal has a disease that can be transmitted to other animals, other animals must be protected through hygiene and biosecurity measures.

An animal showing signs of zoonotic disease, or other disorders that would make the meat unfit for human consumption, must be removed from the slaughter process. This animal could be slaughtered in a separate facility, or at the end of the processing line, after which its products should be disposed of hygienically, and the slaughter facility thoroughly cleaned and disinfected to prevent contamination being carried over to the next processing batch.

Behavioural or postural abnormalities may be seen when animals are fatigued, injured or suffering from neurological disease. Animals that are fatigued or stressed may be rested for 24 hours or more prior to slaughter, although severe cases may need to be slaughtered on welfare grounds. Animals that are to be rested require adequate space and bedding, water, food and tranguillity. Animals showing signs of neurological disease must be separated from the normal animals and examined carefully, as these diseases may be of great public health or animal health significance. Ruminants may carry transmissible spongiform encephalopathies (TSEs), while listeriosis can manifest as neurological disease in animals (Box 6.1).

When animals are injured, it is important to ensure that they do not undergo unnecessary suffering. Other animals should be removed from the immediate area, and the injured animal slaughtered without delay. Processing of the resultant carcass should be carried out in a separate area, as there may be hygiene implications relating to the specific nature of the injury, and the animal is often dirty if it was recumbent.

Animals that are dirty can pose a severe risk to meat hygiene. Many of the causes of food-borne disease in humans are carried in the intestinal content and on the skins of animals. When the animal is visually dirty, there is an increased likelihood of these organisms being transferred to the meat during processing of the carcass. Dirty animals should be identified at antemortem inspection and removed from the slaughter process (Box 6.2). These animals could be cleaned prior to being re-presented for antemortem inspection. Cleaning could involve shaving or clipping the hair coat, or washing the animal. When an animal is washed, it is important that the coat is then allowed to dry before processing begins; otherwise contaminated fluid will be expressed onto the meat during skinning in the manner of a cloth being squeezed. It may be possible in some slaughter facilities to process the animals at the end of the line, using careful dressing procedures to limit the risk of contamination. For example, dirty hair may be clipped off and removed after slaughter and bleeding, but before skinning begins.

Animals found dead should be recorded and sent for rendering. Condemned animals or their carcasses, and animals found dead, should never be permitted to pass through the slaughter floor or other areas of the establishment that are used for handling edible parts of the carcass.

Box 6.1 Neurological disease – a disease of the nervous system or brain

TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY (TSE)

- For example, bovine spongiform encephalopathy (BSE) in cattle, scrapie in sheep.
- Linked to variant Creutzfeldt-Jakob Disease (vCJD), a fatal degeneration of the brain in humans.
- Animals with clinical signs should not be used for human or animal feed production.
- There may be a requirement to remove certain tissues of ruminants from the food chain.
- Some animals may be tested prior to their carcass entering the food chain.

LISTERIOSIS

- Bacterial meningitis, classically presenting as circling behaviour.
- Zoonotic disease.
- · Infected animals are unfit for consumption.

Box 6.2 Livestock cleanliness categories of the Irish Department of Agriculture and Food

In 1998 the Irish Department of Agriculture and Food introduced regulations to address the issue of excessively dirty cattle at the abattoir. These regulations allow the ante-mortem veterinary inspector to reject animals for slaughter or to permit slaughter under special conditions.

On the basis of pictorial and written guidelines, cattle are placed in categories from 1 (very clean) to 5 (very dirty). Normal dressing procedures are employed for categories 1 and 2, with extra care being taken for animals with wet hides. Slowing of the line speed, reduced numbers of carcasses on the line and improved workstation hygiene are considered for category 3 animals. Category 4 animals are slaughtered under similar special conditions at the end of the day and category 5 animals are rejected for slaughter.

Cleanliness categories:

CATEGORY 1

No evidence of adherent faecal material and very limited amounts of loose straw/bedding.

CATEGORY 2

A light covering of dried faecal material and limited amounts of loosely adherent straw/bedding.

CATEGORY 3

Animals with significant amounts of straw/bedding/dirt over large areas of predilection sites.

CATEGORY 4

Animals with heavy amounts of adherent dirt/faeces on fore and hind legs and/or on predilection sites and/or significant amounts of straw/bedding attached to the hide or between areas of dried faecal material. Hide clipping may be practised before animals are permitted for slaughter.

CATEGORY 5

Animals with very heavy amounts of adherent dirt/faeces on predilection sites. Balling of adherent dirt/faeces may be evident on the underside of the abdomen. Animals are rejected for slaughter and returned to the premises of origin or subjected to hide clipping.

Source: Doherty, 1999

GENERAL PATHOLOGICAL CONDITIONS

Fever (pyrexia)

Fever is an abnormal elevated body temperature. It may be classified as septic or aseptic according to the presence or nonpresence of an infection. In septic fever the infection is caused by viruses, bacteria, bacterial toxins, protozoa or fungi. Aseptic fever may be caused by:

- tissue necrosis, as seen in muscle degradation due to intermuscular injection of necrotizing substances, in rapidly growing tumours undergoing necrosis or lysis of burned tissue;
- chemicals or surgery in the former by an administration of drugs and in the latter by breakdown of tissue and blood;
- anaphylactic reaction of antibodies to foreign antigens.

Ante-mortem findings:

- · chills and sweating;
- · dehydration;
- · elevated body temperature;
- increased pulse and respiration;
- depression and dullness;
- anorexia and constipation.
 In septic fever, other signs may include:
- · diarrhoea and vomiting;
- urinous or phenolic odour of breath;
- shock, convulsions and coma.

Differential diagnosis:

Hyperthermia and septicaemia. In hyperthermia the elevation of body temperature is caused by physical factors such as high environmental temperature or prolonged muscular exertion, particularly in humid weather.

Inflammation in viral diseases

Inflammation associated with viral diseases is usually secondary to primary cellular change. Secondary bacterial infections frequently accompany and complicate viral diseases, particularly respiratory and skin diseases. Viral infection associated with fever, malaise, anorexia or incoordination is attributed to absorption of injured cell products, viral toxicity and viral abnormalities that cause circulatory disturbances. Vascular shock, together with viral toxicity and failure of one or more vital organs, is thought to be associated with death in viral diseases.

Septicaemia

Septicaemia is a morbid condition caused by the presence of pathogenic bacteria and their associated toxins in the blood. The positive diagnosis of septicaemia can only be made by isolation of the causative organism from the bloodstream. This is not practised on routine ante-mortem inspection of animals in abattoirs; however, the evidence of septicaemia is determined by the ante-mortem and postmortem findings.

Ante-mortem findings:

- · depression;
- changes in body temperature the temperature is usually elevated, but it can also be normal and subnormal during the terminal phases;
- · difficult and rapid breathing;
- shivering and muscle tremors;
- congestion or petechial haemorrhages of conjunctivae, mouth and vulvar mucosae.

Toxaemia

The identification of toxaemia presents some difficulties on routine ante-mortem and postmortem inspection. The gross lesions differ depending on the specific organisms and toxins involved. Also the clinical signs of toxaemia simulate a variety of other pathologic conditions. Toxaemia is defined as the presence and rapid proliferation of exotoxin and endotoxin derived from micro-organisms or produced by body cells in the bloodstream. Clinical signs and post-mortem findings are similar to those of septicaemia.

Ante-mortem findings:

- normal or subnormal temperature fever may be present if toxaemia is due to microorganisms;
- confusion and convulsions;
- · abnormal changes in locomotion;
- moribund animal or evidence of pain (noted by grinding of teeth);
- animal is not able to rise or rises with great difficulty;
- dehydration may also be present.

Toxaemia is frequently associated with:

- gangrenous mastitis;
- metritis;
- aspiration pneumonia;
- old wounds and injuries;
- diffuse peritonitis due to perforation of the reticulum or uterus.

All these signs may not be seen in every animal affected with toxaemia.

Judgement:

The primary lesions causing septicaemia or toxaemia, including metritis, mastitis, pericarditis, enteritis and others, should be observed and recorded as causes of condemnation. Comatose or moribund animals should be condemned on ante-mortem inspection.

Bruises

Bruises are frequently found on ante-mortem and post-mortem inspection in food-producing animals and poultry. In cattle, bruises caused by transportation or handling are commonly found in the hip, chest and shoulder areas; in pigs, within the ham and in sheep, in the hind leg. Bruises and haemorrhage in the hip joint are caused by rough handling of animals during shackling. Bruises in poultry can be localized or generalized and are frequently associated with bone fractures or ruptured ligament tendons.

Judgement:

Bruised animals should be treated as "suspects" on ante-mortem inspection.

Abscesses

An abscess is a localized collection of pus separated from the surrounding tissue by a fibrous capsule.

Judgement:

The judgement of animals affected with abscesses depends on findings of primary or secondary abscesses in the animal. The portal of entry of pyogenic organisms into the system is also of importance. The primary abscess is usually situated in tissue that has contact with the digestive tract, respiratory tract, subcutaneous tissue, liver, etc. The secondary abscess is found in tissue where contact with these body systems and organs is via the bloodstream. Inspectors should differentiate between abscesses in the active and growing state and older calcified or healed abscesses. In domestic animals, the primary sites of purulent infections are post-partum uterus, umbilicus or reticulum in hardware disease. Secondary abscesses are frequently observed in distant organs.

Animals affected with abscesses spread through the bloodstream (pyemia) are condemned on ante-mortem if the findings of abscesses are over most areas of the body and systemic involvement is evident, as shown in elevated temperature and cachexia.

Emaciation

Emaciation is a common condition of food animals and is characterized by a loss of fat and flesh following loss of appetite, starvation and cachexia. It is associated with gradual diminution in the size of organs and muscular tissue, as well as oedema in many cases. The organs and muscular tissue appear thinner, moist and glossy. Cachexia is a clinical term for a chronic debilitating condition or general physical wasting caused by chronic disease.

Emaciation may be associated with chronic diseases and parasitic conditions such as roundworms in pigs and fascioliasis in cattle and sheep, swine erysipelas, neoplasms, tuberculosis, Johne's disease, caseous lymphadenitis, poor teeth and lack of nutrition. Emaciation is a postmortem descriptive term that should be differentiated from thinness.

Ante-mortem findings:

- wrinkled, dry leathery skin;
- · rough hair coat;
- prominent bones and sunken eyes.

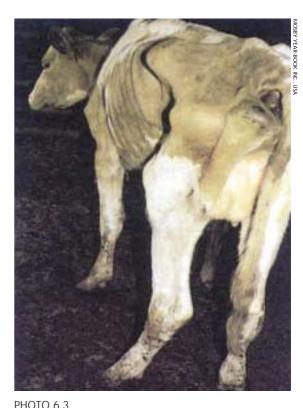
Judgement:

Animals affected with emaciation should be treated as "suspects" on ante-mortem inspection.

Differential diagnosis:

Thinness/leanness, oedema and uraemia.

Leanness (poorness) is often observed in range bulls on poor-quality pasture, high-milking cows and young growing animals that have had a protein-deficient diet.



Emaciated cow showing marked reduction of muscle mass

Oedema

Oedema is the accumulation of excess fluid in the intercellular (interstitial) tissue compartments, including body cavities.

There are two types of oedema:

- inflammatory oedema (exudate);
- non-inflammatory oedema (transudate).
 Inflammatory oedema shows yellow, white or

greenish, clear or cloudy fluid in the area of inflammation. Non-inflammatory oedema is an accumulation of fluid in subcutaneous tissue, submucosae, lungs and brain.

Localized oedema is noted after:

- the swelling of a cow's leg in prolonged decubitus – this swelling is caused by obstruction of the venous outflow;
- interference with the lymph circulation of an organ or area by proliferation of tumours in or around bile ducts;
- inflammation or an allergic reaction.
 Systemic or generalized oedema may occur secondary to congestive heart failure or be caused by low protein levels in the blood.

The latter may be associated with:

severe malnutrition;

EAR-BOOK INC.

· severe amyloidosis of the kidney;

- gastrointestinal parasitic infestation;
- · chronic liver disease;
- damage to the vascular endothelium by toxins and infectious agents.

Anasarca is a form of oedema of the subcutaneous tissues. Ascites is an accumulation of fluid in the peritoneal cavity. Hydrothorax is an accumulation of fluid in the pleural cavity. Hydrothorax may accompany traumatic pericarditis, ascites, cirrhosis of the liver and roundworm infestation in sheep. Anasarca may be caused by toxaemic infection.

Ante-mortem findings:

- depressed and drowsy;
- swelling of the mandible, dewlap, legs, shoulder, brisket and abdomen;
- oedematous tissue is cool upon touch and is of a firm, doughy consistency.

Judgement:

Animals affected with generalized oedema may be condemned on ante-mortem inspection. In less severe non-generalized cases, animals are treated as "suspects".

PHOTO 6.4 Abdominal oedema caused by liver disease

Immaturity

Immaturity occurs mainly in calves. In many countries, the slaughter of calves younger than two weeks of age is prohibited.

Ante-mortem findings:

· presence of the umbilical cord;

• bluish and not completely retracted gums. (In addition, greyish muscles that are flabby, tear easily and are not well developed, and dark red kidney and oedematous kidney capsule, are found at post-mortem.)

Plant poisoning

In developing countries, slaughter animals, and particularly cattle, are often trekked some hundreds of kilometres on the hoof to the abattoirs. During this journey, animals may suffer from various types of plant poisoning. In addition, cattle living in areas where pasture has poisonous plants may suffer from the effects of chronic plant poisoning. Different body systems may be affected and various lesions may be seen at meat inspection.

Clinical signs and gross lesions observed in animals that have ingested certain poisonous plants are listed below:

- tulip (*Tulipa* spp.) causes diarrhoea, bloated abdomen and heart failure;
- Lantana camara causes photosensitization;
- Senecio spp. cause necrosis and cirrhosis of liver;
- · Crotalaria spp. cause laminitis;
- *Dichapetalum cymosum* causes heart failure and sudden death.

Judgement:

Judgement of the animal will depend on the clinical signs and the extent and severity of the lesions.

Chemical poisoning

Dipping of cattle in acaricide on a regular basis is practised in many parts in order to control tick-borne diseases. Chemicals used for this purpose include arsenic, chlorinated hydrocarbons and organophosphates. Dipping may lead to clinical cases of poisoning, which may be manifested with the following clinical signs: nervous system disturbances, acute abdominal pain, diarrhoea and skin lesions.

Judgement:

The carcass, offal and intestine should be condemned if clinical signs of poisoning are associated with post-mortem lesions.

SPECIFIC DISEASES

DISEASES CAUSED BY VIRUSES

Foot-and-mouth disease (FMD)

FMD is an acute viral and extremely contagious disease of cloven-footed animals such as cattle, sheep, goats, pigs and antelopes. It is manifested by vesicles and erosions in the muzzle, nares, mouth, feet, teats, udder and pillar of the rumen. There are three main strains of viruses causing FMD, namely A, O and C. Three additional strains, SAT 1, SAT 2 and SAT 3 have been isolated from Africa and a further strain ASIA-1 from Southeast Asia.

Transmission:

Direct and indirect contact with infected animals and their secretions, including saliva, blood, urine, faeces, milk and semen, aerosol droplet dispersion, infected animal by-products, swill containing scraps of meat or other animal tissue and fomites.

PHOTO 6.5 Excessive salivation in a cow affected with FMD



Ante-mortem findings:

Before vesicle formation:

- incubation: one to five days or longer;
- morbidity: nearly 100 percent;
- mortality: variable depending on the strain of virus and its virulence and susceptibility of host; 50 percent in young animals, 5 percent in adults;
- fever up to 42 °C;
- dullness;
- lack of appetite;
- drastic drop in milk production;
- uneasiness and muscle tremors.

Vesicle formation:

- smacking and quivering of lips;
- extensive salivation and drooling;
- shaking of feet and lameness.

The vesicles and later erosions are commonly found on the muzzle, tongue, oral cavity and teat, and on the skin between and above the hooves of the feet. In more chronic cases in cattle the hoof becomes loose and the animal may walk with a characteristic "clicking" sound (slippering).

Some strains of FMD, particularly in swine, sheep and goats, cause erosions instead of vesicles.

Judgement:

In countries or in zones within a country free or nearly free of FMD, diseased or suspect animals are prohibited from admission to an abattoir or slaughtered. In countries where this disease is present, the judgement should be in accordance with the current animal health requirements, and consistent with effective public health protection. Particular attention should be paid to secondary bacterial infections and general findings. Sanitary measures should be taken to comply with national animal health policy.

Remarks:

Latent infections with *Salmonella* organisms have been reported in animals affected with FMD.

Differential diagnosis in bovine and ovine species:

Vesicular stomatitis, allergic stomatitis, feedlot glossitis, photosensitization, bluetongue, rinderpest, infectious bovine rhinotracheitis, malignant catarrhal fever, bovine papular stomatitis, bovine viral diarrhoea, pseudocowpox, ovine pox, contagious ecthyma, foot-rot, mycotoxicosis and increased salt content in feed.

Discussion:

In order to prevent the spread of the virus in the abattoir, the equipment and room should be disinfected with 2% NaOH (caustic soda). In some countries sodium carbonate (Na_2CO_3) is used. The vehicle conveying diseased animals should also be disinfected and abattoir personnel leaving the abattoir should pass through a footbath with 1% solution of NaOH.

The virus of FMD can survive in meat and meat products for a considerable length of time. Outside the pH range of 6–9, viral infectivity is destroyed. A bovine carcass matured at above +2 °C produces a drop in the pH of muscle tissue to between 5.3 and 5.7 within 24 hours of slaughter. This is caused by the formation of sarcolactic acid. Quick freezing of the meat arrests acid production and consequently the virus remains infective for about six months. In salted meat at 4 °C, the virus is still infective in bone marrow and lymph nodes for six months. In blood clots in large vessels of cattle and swine, the virus is infective for two months. The virus is inactivated by ultraviolet rays, acetic



PHOTO 6.6 FMD: extensive areas of eroded epithelium on a bovine tongue

acid, 2% lye and ethylene oxide. At high temperatures, the virus is only active for a short period. A solution 2% NaOH of inactivates the virus in one to two minutes. In dry refuse in stalls, the virus remains infective for 14 days, for three days on soil surfaces in summer, compared to 39 days in fall. It is also infective for 39 days in urine and for 20 weeks on hay dried at 22 °C. The virus can be destroyed with 0.5% citric or lactic acid, by cooking meat to an internal temperature of 69 °C and by pasteurization processes of milk.

ISDA APHI

Rinderpest (RP)

RP is an acute, highly contagious, fatal viral disease of cattle, buffalo and wild ruminants manifested by inflammation, haemorrhage, erosions of the digestive tract, wasting and often bloody diarrhoea. Some swine species are also susceptible. Humans are not susceptible to RP virus.

Transmission:

Direct contact with infected animals or their excretions and secretions and fomites. The virus appears in the blood and in secretions before the onset of clinical signs and this may cause infection in abattoirs and stockyards.

Ante-mortem findings:

- incubation: 3–10 days or longer;
- morbidity: up to 100 percent in a susceptible herd;
- mortality: 50 percent and may reach 90–95 percent;
- high fever (41-42 °C);
- nasal discharge and excessive salivation;
- punched-out erosions in the mouth;
- loss of appetite and depression;
- abdominal pain (grunting, arched back);

- constipation followed by bloody diarrhoea and straining;
- dehydration and rough hair coat;
- marked debility;
- abortion;
- the classical "milk fever position" in cattle.

Judgement:

In areas free of RP and in zones where the final stages of eradication exist, the animals are condemned.

Remarks:

RP virus is sensitive to environmental changes and is destroyed by heat, drying and a wide range of disinfectants.

Differential diagnosis:

Bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, bluetongue, coccidiosis, FMD, vesicular and necrotic stomatitis and bovine papular stomatitis. Vesicular diseases do not have accompanying haemorrhage, and blisters should be differentiated from erosions (ulcers) seen in RP.



PHOTO 6.7 **RP** erosions on the dental pad and the hard palate that resemble FMD

Vesicular stomatitis (VS)

This is a viral disease of ruminants, horses and swine characterized by vesicular lesions of the mouth, feet and teats. VS virus has two immunologically distinct serotypes: Indiana and New Jersey.

Transmission:

In susceptible animals, contamination of preexisting abrasions with saliva or lesion material, by ingestion of contaminated pasture or during milking within dairy herds. Mechanical transmission by biting arthropods is also a possibility. The virus is isolated from mites, tropical sandflies and mosquitoes.

Ante-mortem findings:

- · fever;
- mouth lesions in cattle and horses;
- vesicles tend to disappear quickly and only papules may be seen in cattle outbreaks;

- marked weight loss and cessation of lactation in dairy cows;
- chewing movements and profuse salivation;
- refuses food but eagerly acceptes water;
- · horses rub lips on edges of mangers;
- foot lesions occur in about 50 percent of cases in cattle;
- lameness;
- teat lesion may occur in all species.

Differential diagnosis:

FMD, swine vesicular exanthema, vesicular disease, bovine papular stomatitis.

Mouth and muzzle lesions: bovine viral diarrhoea, rinderpest, mycotic stomatitis, photosensitization and Potomac Valley fever in horses

Teat lesions: cowpox, pseudo-cowpox, pseudo-lumpy skin disease and bovine herpes mammillitis.



Malignant catarrhal fever (MCF)

An acute viral disease of cattle, deer, bison and buffalo characterized by inflammation of the mucous membranes of the nose and eyes, corneal opacity, profuse nasal discharge and enlargement of lymph nodes. MCF is arbitrarily divided into peracute, intestinal, head-eye and mild forms according to ante-mortem findings. It is not communicable to humans.

Transmission:

Close contact between cattle and wildebeest (gnu, antelope), by common use of drinking troughs or by direct contact between cattle and newborn wildebeest and placenta of parturient dams. In American or European cases of MCF, cattle are infected from sheep.

Ante-mortem findings:

- incubation: 9-44 days;
- morbidity is low and mortality is high;
- increased temperature;

- bilateral ocular and nasal discharges;
- dyspnoea and cyanosis;
- · loss of appetite;
- encrustation of muzzle and eczema of the perineum, scrotum and udder;
- erosions on the lips, tongue, gums, soft and hard palate;
- swollen reddened eyelids, corneal opacity and conjunctivitis (Photo 6.9);
- photophobia associated with corneal opacity and blindness;
- reluctance to swallow because of oesophageal erosions and drooling;
- · enlarged body lymph nodes;
- rarely, uncoordinated movements and shivering.

Differential diagnosis:

Bluetongue, RP, bovine viral diarrhoea/mucosal disease, FMD, VS.



PHOTO 6.9 *MCF: early* stages of corneal opacity, conjunctivitis and reddening of the eyelids

Rabies

This is an acute infectious viral disease of the central nervous system in mammals.

Transmission:

It is usually transmitted through the saliva by a bite from a rabid animal, commonly the dog or jackal. Humans are infected in the same way.

Ante-mortem findings:

Furious form:

- incubation: from two weeks to six months or longer;
- restlessness;
- aggressive, may attack other animals;
- sexual excitement;
- · bellowing;
- paralysis and death;
- paralytic form;
- sagging and swaying of the hindquarters;
- drooling and salivation;
- the tail is held to one side;
- tenesmus or paralysis of the anus;
- · paralysis;
- the animal falls to the ground;
- death after 48 hours of decubitus.

Differential diagnosis:

Indigestion, milk fever or acetonaemia when first seen, foreign body in the mouth, early infectious disease, poisoning.

Discussion:

In a diseased animal, the virus is found in saliva, salivary gland and nervous tissue. Extreme caution should be instituted in abattoirs in order to prevent occupational hazards. Abattoir personnel can contract the disease through surface contact with infected tissue. Infection does not occur by consumption of meat from a rabid animal.

Slaughter may be prohibited during a quarantine period of eight months following exposure to the disease. An animal suspected of having rabies should be placed under a "Held tag". The warning sign should read "The animal is not to be handled". Any person who was in touch with the animal should thoroughly wash his/her hands with strong soap and/or disinfectant. If possible, the wound should be opened to encourage bleeding in order to flush out the virus and expose the deeper area of the wound. Tincture of iodine (up to 0.001% aqueous solution of iodine or ethanol 43.70%) should be applied.

Lumpy skin disease

Acute pox viral disease of cattle manifested by sudden appearance of nodules on the skin.

Transmission:

Insect vectors by direct and indirect transmission. Seasonal and geographic distribution.



Ante-mortem findings:

- incubation: 4-14 days;
- · fluctuating fever;
- diarrhoea;
- nasal discharge and salivation;
- the first lesions appear in the perineum;

PHOTO 6.10 Lumpy skin disease: cutaneous nodules of various sizes in a severe case of lumpy skin disease

- cutaneous nodules of various sizes may occur throughout the body;
- skin lesions may show scab formation;
- swelling of superficial lymph nodes and limbs, and lameness;
- infertility and abortion;
- secondary infection may lead to joint and tendon inflammation.

Judgement:

The carcass of an animal showing, on antemortem inspection, generalized acute infection accompanied with fever, is condemned.

Differential diagnosis:

Differential diagnosis:

enlarged lymph nodes.

Dermatophilis infection, cowpox and

pseudocowpox, VS and lumpy skin disease. The latter is differentiated from BHD by

Allergies, screwworm myiasis, urticaria, dermatophilosis (streptothricosis), bovine herpes dermophatic infection, cattle grubs, vesicular disease, bovine ephemeral fever, photosensitization, besnoitiosis (elephant skin disease), sweating weakness of calves, bovine farcy and skin form of sporadic bovine lymphomatosis.

Bovine herpes dermophatic disease (BHD)

A herpes virus infection of cattle and sometimes sheep and goats manifested by cutaneous lesions and fever.

Transmission:

Biting insects, mechanical milking.

Ante-mortem findings:

- incubation: 3–7 days;
- morbidity: high in primary infections;
- fever;
- cutaneous nodules: at first these are round, then later become flattened and covered with dry scabs;
- hairless skin is normal after the scab falls off;
- ulcerative lesions of the teats and udder;
- erosions between the digits.

Judgement:

The carcass of an animal affected with BHD is condemned.



PHOTO 6.11 BHD: dried scabs on the skin of the neck

PHOTO 6.12 BHD: ulcerative lesions of the teats and udder

Infectious bovine rhinotracheitis (IBR)

IBR is a highly infectious viral respiratory disease of cattle, goats and pigs manifested by inflammation of respiratory passages and pustular lesions on the male and female genital organs. Generally four forms of the disease are recognized; the respiratory form, the genital form, the enteric form and the encephalitic form.

Transmission:

Respiratory droplet and nasal exudate in the respiratory form of IBR. Obstetrical operations, coitus and licking of genitalia of affected animals in the genital form of disease.

Ante-mortem findings:

Respiratory form:

- incubation: 5–14 days;
- fever;
- nasal and ocular discharge and red, swollen conjunctiva;
- · drop in milk yield;
- · breathing through the mouth and salivation;
- hyperaemia of the nasal mucosa and necrotic areas on the nasal septum;
- secondary bronchopneumonia;
- abortion.

Genital form:

- frequent urination and tail elevation;
- oedematous swelling of the vulva and pustule formation on reddened vaginal mucosa;
- mucoid or mucopurulent exudate in the vagina.
- Enteric form:
- severe oral and stomach necrosis in newborn animals;
- high mortality.

The encephalitic form in calves:

- · depression;
- excitement;
- high mortality.

Judgement:

The carcass of an animal affected with IBR is approved if signs of acute infection are not present and the animal is in good body condition.

Differential diagnosis:

Pneumonic pasteurellosis, bovine viral diarrhoea, MCF and calf diphtheria.

PHOTO 6.13 Breathing through the mouth and salivation in a bovine affected with IBR



Bovine viral diarrhoea (BVD)

This is an infectious viral disease of cattle manifested by an active erosive stomatitis, gastroenteritis and diarrhoea.

Transmission:

Direct contact with clinically sick or carrier animals, indirect contact with feedstuffs or fomites contaminated with urine, nasal and oral secretions or faeces, and contact with aborted foetuses. Transmission through aerosol droplet dispersion or by insect vector may also be a possibility. Virus may persist in recovered and chronically ill cattle, which are considered a potential source of infection.

Ante-mortem findings:

- incubation: 1-3 days;
- fever;
- congestion and erosions in the mucous membranes of the oral cavity;
- · depression and anorexia;
- · cough, polypnea and salivation;
- dehydration and debilitation;

- · foul-smelling diarrhoea;
- cessation of rumination;
- reduced milk supply;
- abortion in pregnant cows;
- laminitis;
- congenital anomalies of the brain (cerebellar ataxia) and arthritis in young calves.

Judgement:

The carcass and viscera of an animal that on ante-mortem inspection shows generalized signs of acute infection accompanied with fever and/or emaciation, are condemned. Chronic cases of BVD with no systemic involvement have a favourable judgement of carcass, viscera and organs.

Differential diagnosis:

MCF, RP, bluetongue and vesicular diseases. The latter produce vesicles that are not present in BVD. Diseases with no oral lesion or diarrhoea include salmonellosis, Johne's disease and parasitism.

Bovine leukosis

Bovine leukosis is a persistent and malignant viral disease of the lymphoreticular system. It occurs in all breeds and in both sexes.

Bovine leukosis is observed in two forms: a) the sporadic and b) the enzootic form. The sporadic form is rare and occurs in cattle under three years of age. The enzootic form is most commonly found in adult cattle, particularly in cull cows.

Transmission:

By small amounts of infected blood (e.g. infected needles, dehorning), vertical transmission from the dam to the calf (3–20 percent of calves may become infected) and by colostrum or milk (less than 2 percent). Insect transmission is also a possibility; higher rates of infection are reported in the summer.

Ante-mortem findings:

- · laboured breathing due to heart involvement;
- persistent diarrhoea following infiltration of the abomasum wall by neoplastic cells;
- marked enlargement of several superficial lymph nodes;
- oedema of the brisket and the intermandibular region;
- paralysis of the hind legs due to tumour compression of the spinal cord;
- protrusion of the eye as a result of tumour invasion of the orbital cavity;
- debilitation or emaciation;
- pale mucosal surface;
- · bloated animal;
- swelling of the neck when thymus is involved;
- cutaneous nodules in the terminal stage.

Differential diagnosis:

Lymphadenitis, lymphoid hyperplasia, hyperplastic haemolymph nodes, pericarditis, enlarged spleen in septicaemic conditions, other neoplasms and parasitism.

DISEASE CAUSED BY PRIONS

Bovine spongiform encephalopathy (BSE, "mad cow disease")

BSE is a progressive and fatal disease of adult cattle characterized by degeneration of the central nervous system (CNS). The causative agent is thought to be composed of an abnormal form of the host-encoded prion protein (PrPc), termed PrPsc. It is believed that the infective prion (PrPsc) induces a conformational change of the host's natural prion (PrPc), which leads to degeneration of the CNS.

Transmission:

The main transmission pathway is the ingestion of BSE-contaminated feeds, which are in most cases protein feed supplements made from BSEcontaminated meat and bone meal.

Ante-mortem findings:

Cattle with BSE tend to have subtle signs of disease. Signs are progressive, variable in type and severity, and may include depression, abnormal behaviour, weight loss, sensitivity to stimuli (light, sound, touch), and gait or movement abnormalities. Other signs that have been noted in some BSE cases include reduced milk yield, bradycardia, and reduced ruminal contractions. None of these signs are specific (pathognomonic) for the disease.

The following clinical signs may occur:

- 1. Disturbances in behaviour
- 2. Disturbances in locomotion
- 3. Disturbances in sensitivity
- 4. Slow weight loss

No diagnostic test for the BSE agent in the live animal is presently available. Current postmortem diagnostic methods are histopathological and detection of the infectious Prion PrPsc.

Judgement:

The carcass is condemned.

Differential diagnosis:

Listeriosis, viral encephalitis (sporadic bovine encephalitis, Borna disease), bacterial encephalitis, brain oedema, tumors, cerebrocortical necrosis, cerebellar atrophy (Purkinje cells), metabolic diseases and others.

Discussion:

BSE was first diagnosed in cattle in the United Kingdom in 1986. It is now been recognized in many other countries in and outside Europe. BSE belongs to a group of human and animal diseases classified as transmissible spongiform encephalopathies (TSE). Significant human diseases of this group are variant Creutzfeldt-Jakob disease (vCJD), Creutzfeldt-Jakob disease (CJD), Kuru and Gerstmann-Sträussler-Scheinker syndrome. Of these, only vCJD has been associated with BSE. TSE-affected animals are bovines (BSE), sheep and goats (scrapie), cervids (chronic wasting disease [CWD]), minks (transmissible mink encephalopathy [TME]) and felines (feline spongiform encephalopathy [FSE]). Cattle testing positive for BSE have ranged from 20 months to 19 years of age, though most of the cases are between 4 and 6 years of age. A breed or genetic predisposition has not been found. Most cases of BSE have come from dairy herds, probably due to differences in feeding systems compared to beef cattle.

Two levels of control and prevention measures must be considered:

- those that block the cycle of amplification in the feed chain;
- 2. those that prevent infective material from entering human food.

Measures to protect animal and human health:

- Introduction of feed bans
- Utilization of appropriate rendering parameters
- Removal from the food chain of specified risk material (SRM)
- Introduction of measures to avoid crosscontamination of meat with SRM
- Ban of mechanically recovered meat for food
- Import control
- Introduction of surveillance systems (active and passive)
- On-farm measures
- Identification and elimination of clinically affected animals before slaughter.

DISEASES CAUSED BY RICKETTSIA AND MYCOPLASMA SPP.

Heartwater (hydropericardium)

"Black dung" when affecting African cattle and buffalo; "Sheep fever" when seen in sheep.

Heartwater is an acute, non-contagious disease of cattle, sheep, goats, antelopes and wild ruminants. It is caused by the rickettsial organism *Cowdria (Rickettsia) ruminantium*.

Transmission:

Heartwater is transmitted by various species of *Amblyomma* ticks. Transstadial transmission of the organism occurs in vector ticks.

Ante-mortem findings:

Peracute form:

- incubation: 14–28 days;
- fever;
- · diarrhoea;
- · convulsions and death.
- Acute form:
- fever up to 42 °C;

· rapid breathing;

• lack of appetite, depression and listlessness. Nervous signs include:

- twitching of the eyelids;
- protrusion of the tongue;
- champing of the jaw;
- · walking in circles;
- paddling with legs in recumbent animals; opisthotonos (arched back) and convulsions.

Differential diagnosis:

Peracute form of heartwater should be differentiated from anthrax. The acute nervous form of the disease is differentiated from tetanus, rabies, cerebral trypanosomiasis, strychnine poisoning, piroplasmosis, theileriosis, lead and organophosphate poisoning, parasitism, arsenical poisoning and poisoning with certain plants.

Q fever (Queensland fever, Nine mile fever, American Q fever, Australian Q fever)

Q fever is a disease of cattle, sheep, goats, donkeys, camels, fowl, dogs, cats, pigeons and humans. It is caused by *Coxiella burnetii*. Q fever is an occupational disease of livestock personnel, farmers and laboratory personnel.

Transmission:

Ticks spread infection to cattle, which develop mild disease. The faeces deposited on animal hide by ticks may be the source of infection for humans. Q fever is also transmitted by inhalation of dust contaminated with infected animal secreta or excreta. Healthy animals may serve as carriers and shed the organism in milk, urine, faeces, placenta and foetal fluids. They harbour the infection and no clinical signs are observed. Contaminated meat and water are further means of infection spread.

In field cases, there are no clinical signs of this disease. In the disease produced by the inoculation of cows via the udder the clinical signs may include:

acute mastitis;

- loss of appetite and depression;
- serous nasal and lacrimal discharge;
- · difficult breathing;
- atony of the rumen;
- · abortion in pregnant cows;
- no gross lesions are reported in cattle.

Discussion:

Coxiella burnetii is highly resistant and has been isolated from farm soil six months after the removal of animals. It may persist in the udder for up to three years. The temperatures of milk pasteurization (in bulk at 63 °C for 30 minutes or the common method at 72 °C for 15 seconds) kill this agent in milk. Vaccination will reduce shedding of organisms in milk.

This disease in humans has a sudden onset and is characterized by loss of appetite, weakness and generalized malaise lasting from one to two weeks. Pneumonia may also be present. Death may be caused by endocarditis in older people. More severe symptoms of Q fever are noticed. This is an acute, subacute or chronic highly infectious disease of cattle caused by *Mycoplasma mycoides* var. *mycoides*.

Transmission:

Aerosol and droplet infection from the infected animals. Recovered animals, called "lungers", may act as carriers and shedders, especially under stress.

Ante-mortem findings:

- incubation: acute: 10–14 days, chronic: 3–6 months;
- morbidity: 90 percent in susceptible cattle;
- mortality: 10–50 percent;
- fever;
- · depression;
- lack of appetite and loss of weight;
- · coughing on exercise;
- shallow rapid respiration, grunting and gurgling;

- extended neck, lowered head and open mouth;
- arched back and outward rotated elbow;
- arthritis in young animals.

Judgement:

The carcass of an animal affected with CBPP is condemned if the disease is associated with fever, inadequate bleeding of carcass, serous infiltration of the brisket and emaciation. Recovered animals showing no generalized signs of the disease are approved and the affected organs are condemned.

Differential diagnosis:

Shipping fever (pasteurellosis), East Coast fever, foreign body pneumonia, IBR, tuberculosis, chlamydial infections and lungworms.

DISEASES CAUSED BY BACTERIA

Blackquarter (blackleg)

Blackquarter is an acute infectious disease of cattle and sheep manifested by severe inflammation of the muscle with high mortality. It is caused by *Clostridium chauvoei*.

Transmission:

The organisms of blackleg are found in the soil. During grazing, organisms may enter the digestive tract of a susceptible animal. *C. chauvoei* is also found in the digestive tract of healthy animals. In sheep, the agent is transmitted through wounds at shearing, docking and castration, and during lambing in ewes.

Ante-mortem findings:

- high fever (41 °C);
- lameness;
- loss of appetite;
- discoloured, dry or cracked skin;
- stiff gait and reluctance to move;
- crepitating swellings often on the hips and shoulders;
- in sheep, gaseous crepitation cannot be felt before death.

Judgement:

Carcasses of animals affected with blackleg should be condemned. It is prohibited to slaughter and dress an animal diagnosed with this disease at ante-mortem inspection.

Differential diagnosis:

Other acute clostridial infections, lightning strike, anthrax, bacillary haemoglobinuria, lactation tetany, extensive haemorrhage and acute lead poisoning.

Discussion:

Blackleg is worldwide in distribution. Well nourished animals are more frequently affected. It is also more commonly seen in grass-fed animals than in stall-fed animals. Clostridia are soil-borne organisms that cause disease by releasing toxins. Specific antitoxins and antibiotics are rarely effective in the treatment of this disease. An adequate preventive vaccination programme may be the most effective method in protecting animals from blackleg.

Botulism

Botulism is a disease manifested by progressive muscular paralysis. It is seen in humans, animals, birds and fish and is caused by various strains of *Clostridium botulinum*.

Transmission:

Decomposed flesh and bones are the source of infection for animals. The incubation period is usually 12–24 hours, but incubation periods of anything between two hours and 14 days have been recorded.

Ante-mortem findings:

- In cattle and horses:
- restlessness;
- · knuckling and incoordination;
- paralysed tongue and drooling of saliva;
- sternal recumbency;
- progressive muscular paralysis from hindquarters to frontquarters, head and neck.

In sheep:

- serous nasal discharge and salivation;
- abdominal respiration;
- stiffness upon walking and incoordination;
- switching of the tail on the side;
- limb paralysis and death.
- In pigs:
- lack of appetite, refusal to drink and vomiting;
- pupillary dilatation;
- muscular paralysis.

Judgement:

Total condemnation of the carcass because of human hazards.

Differential diagnosis:

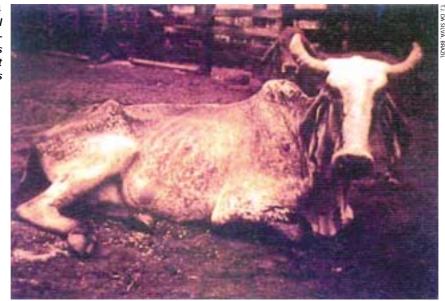
Parturient paresis, paralytic rabies, equine encephalomyelitis, ragwort poisoning in horses, miscellaneous plant poisoning. In sheep: louping ill, hypocalcaemia and some cases of scrapie.

Discussion:

C. botulinum is found in the digestive tract of herbivores. Soil and water contamination occurs from faeces and decomposing carcasses. The proliferation of *C. botulinum* organisms may also occur in decaying vegetable material. Sporadic outbreaks of botulism are reported in most countries. Outbreaks of botulism in cattle and sheep in Australia, southern Africa and the Gulf Coast area of the United States of America are associated with phosphorus-deficient diets and ingestion of carrion. Cattle, sheep and rarely swine are susceptible to this disease. Dogs and cats are resistant.

C. botulinum produces a neurotoxin which causes functional paralysis. Seven strains of this organism ("A through G") are distinguished according to immunological differences. The diseases caused by various strains of this agent are frequently regarded as a separate entity

PHOTO 6.14 Botulism: sternal recumbency – muscular paralysis of hind and front quarters



owing to some of their prominent signs. Names such as "Bulbar paralysis in cattle", "Lamziekte in sheep" in South Africa (meaning lame sickness), and "Limberneck in poultry" are often used. *C. botulinum* is often found in anaerobic conditions of deep wounds. It produces neuroparalytic exotoxins that cause symptoms of the disease. This organism will grow and produce toxins if the temperature is between 10 and 50 °C, pH above 4.6, water activity (AW) is above 0.93 and anaerobic conditions exist. Fresh meats are implicated in less than 10 percent of botulism outbreaks. The major sources of this organism are fish, home-cured meats, homecanned vegetables and fruit. Eggs, milk and their products are rarely the cause of an outbreak. Most frequently, raw, insufficiently cooked foods or foods not fully salted, cured, dried or smoked are implicated. Botulism toxins are heat labile and food suspected of having the organism should be boiled before serving.

In humans, the signs of the disease are weakness, dizziness, blurred or double vision, dilatation of pupils, dry mouth, difficulties in breathing and speech, progressive muscular weakness, respiratory failure and death. Pneumonia may be a complication associated with botulism in humans.

Malignant oedema

Malignant oedema is a bacterial disease of cattle, sheep, goats, swine, horses and poultry. It is caused by *Clostridium septicum* and is manifested by wound infection. The infection is commonly soil-borne. Deep wounds associated with trauma provide ideal conditions for the growth of this agent.

Ante-mortem findings:

- fever 41-42 °C;
- depression and weakness;
- muscle tremor and lameness;

 soft doughy swelling and erythema around the infection site.

Judgement:

The carcasses of animals affected with malignant oedema are condemned.

Differential diagnosis:

Blackleg. In malignant oedema the muscle is not involved and the wound site is noted. Anthrax in pigs. Subcutaneous oedema in the throat region is present.

Tuberculosis

Tuberculosis is a chronic disease of many animal species and poultry caused by bacteria of the genus *Mycobacterium*. It is characterized by development of tubercles in the organs of most species. Bovine tuberculosis is caused by *Mycobacterium bovis*. It is a significant zoonotic disease.

Transmission:

An infected animal is the main source of transmission. The organisms are excreted in the exhaled air and in all secretions and excretions. Inhalation is the chief mode of entry and, for calves, infected milk is an important source of infection. When infection has occurred, tuberculosis may spread: a) by primary complex (lesion at point of entry and the local lymph node) and b) by dissemination from primary complex.

Ante-mortem findings:

- · low-grade fever;
- chronic intermittent hacking cough and associated pneumonia;
- difficult breathing;
- weakness and loss of appetite;
- emaciation;
- swelling of superficial body lymph nodes.

Discussion:

Mycobacteria invade cattle by respiratory (90–95 percent) and oral routes (5–10 percent). Congenital infection in the bovine foetus occurs from an infected dam. Tuberculosis lesions can be classified as acute miliary, nodular lesions and chronic organ tuberculosis. Young calves are infected by ingestion of contaminated milk. The incidence of human tuberculosis caused by *Mycobacterium bovis* has markedly dropped with the pasteurization of milk. It has also dropped in areas where programmes of tuberculosis eradication are in place. Humans are susceptible to the bovine type. In cattle, lesions of tuberculosis caused by the avian type are commonly found in the mesenteric lymph nodes. Tuberculosis in small ruminants is rare. In pigs, the disease may be caused by the bovine and avian types. Superinfection is specific in cattle.

Judgement:

The carcass of an animal affected with tuberculosis requires additional post-mortem inspection of the lymph nodes, joints, bones and meninges. It is suggested that the Codex Alimentarius judgement recommendations for cattle and buffalo carcasses be followed. Carcasses are condemned:

- where an eradication scheme has terminated or in cases of residual infection or reinfection;
- in final stages of eradication where natural prevalence is low;
- during early stages of eradication in highprevalence areas.

The carcass of a reactor animal without lesions may be approved for limited distribution. If the economic situation permits, this carcass should be condemned.

Heat treatment of meat is suggested during early and final stages of an eradication programme: in low- and high-prevalence areas where one or more organs are affected, and where miliary lesions, signs of generalization or recent haematogenous spread are not observed. If the economic situation permits, then the carcass is condemned. In some countries, the carcass is approved if inactive lesions (calcified and/or encapsulated) are observed in organs and without generalization in lymph nodes of carcass.

Differential diagnosis:

Lung and lymph node abscess, pleurisy, pericarditis, chronic contagious bovine pleuropneumonia, actinobacillosis, mycotic and parasitic lesions, tumours, caseous lymphadenitis, Johne's disease, adrenal gland tumour and lymphomatosis.

Johne's disease (bovine paratuberculosis)

Johne's disease is a chronic, infectious bacterial disease of adult wild and domestic ruminants such as cattle, sheep and goats. It is characterized by the thickening and corrugation of the wall of the intestine, gradual weight loss and chronic diarrhoea, and is caused by *Mycobacterium paratuberculosis*.

Transmission:

Ingestion of faeces harbouring *M. paratuberculosis*.

- The agent is persistent in soil, pasture, manure and stagnant water for prolonged periods.
- Carrier animals, so-called "faecal shedders", are the most important source of infection.
- Ingestion of organism causes infection. Calves may become infected from a nursing infected dam.
- Transmission with semen and *in utero* are minor sources of infection.

Ante-mortem findings:

- incubation: 2 to 3 years with range from 6 months to 15 years;
- poorly performing animal that stops eating in the final stages of the disease;
- gradual and chronic weight loss and emaciation;

- rough hair coat and dry skin;
- non-responsive diarrhoea with watery fluid faeces;
- submandibular oedema ("bottle jaw");
- reduced milk production;
- mastitis and infertility;
- debility and death.

Judgement:

The carcass of an animal affected with Johne's disease is approved when generalized systemic signs of disease are not present. A poor, thin and slightly moist carcass should be held in the chiller and assessed after 24 or 48 hours. If the dryness and setting of the carcass improves during this time it can be released. A carcass with associated oedema and emaciation is condemned.

Differential diagnosis:

Other causes of diarrhoea and weight loss, malnutrition, chronic salmonellosis, parasitism (e.g. ostertagiasis), winter dysentery, BVD, hardware disease, coccidiosis, liver abscesses, kidney disease, inflammation of the heart and its sac, toxic inflammation of the intestine caused by arsenic, plants and mycotoxicosis and neoplasm.

Leptospirosis

Leptospirosis is an important and relatively common disease of domestic and wild animals and humans. In cattle, it is manifested by interstitial nephritis, anaemia, mastitis and abortion in most species. *Leptospira* spp. are the causative agents.

Transmission:

Animals contract the disease by eating and drinking *Leptospira*-contaminated urine or water, or by direct contact of broken skin or mucous membranes with mud, vegetation or aborted foetuses of infected or carrier animals. Recovered animals and animals with unapparent (subclinical) leptospirosis frequently excrete billions of *Leptospira* organisms in their urine for several months or years.

Ante-mortem findings:

Acute and subacute forms:

- transient fever;
- · loss of appetite;
- lactating cows may stop milking;
- mastitis;
- milk may be yellow, clotted and frequently blood-stained;
- severely affected animals:
 - jaundice and anaemia
 - pneumonia
 - abortion with frequent retention of the placenta (afterbirth).

Severe illness in young calves may be associated with yellowish discoloration of mucous membranes and reddish-brown urine before death. The chronic form has mild clinical signs and only abortion may be observed. If meningitis occurs, the animal may show incoordination, salivation and muscular rigidity.

Judgement:

The carcass of an animal affected with acute leptospirosis is condemned. A chronic and localized condition may warrant an approval of the carcass.

Differential diagnosis:

Acute and subacute forms to be differentiated from babesiosis, anaplasmosis, rape and kale poisoning, bacillary haemoglobinuria, postparturient haemoglobinuria and acute haemolytic anaemia in calves. The presence of blood in the milk is a characteristic clinical sign which will differentiate leptospirosis from other infectious diseases.

Discussion:

Leptospirosis is a zoonosis and is also an occupational hazard for farmers, veterinarians and butchers. Human infection may occur by contamination with infected urine and urine contents. The bacteria may be also found in milk in acute cases; however, it does not survive for long periods of time in milk. Pasteurization will also kill *Leptospira* organisms. They can survive for months in moist and humid environments, particularly in swamps, ponds and streams or poorly drained pastures.

Brucellosis (contagious abortion, Bang's disease)

Brucellosis of cattle is an infectious, contagious disease caused by *Brucella abortus* and is characterized by abortion in late pregnancy and a high rate of infertility. *B. melitensis* affects mainly goats, *B. ovis* sheep and *B. suis* swine. *B. abortus* may also occur in horses.

Transmission:

An uninfected animal may become infected with *Brucella* organisms by contaminated feed, pasture, water, milk, by an aborted foetus, foetal membranes and uterine fluid and discharges. The disease may also be spread by dogs, rats, flies, boots, vehicles, milking machines and other equipment used in the barn. *Brucella* organisms may be occasionally shed in urine.

Ante-mortem findings:

In cattle:

- abortion in non-vaccinated pregnant cows in the last three to four months of pregnancy;
- occasional inflammation of testes and epididymis;
- swelling of scrotum (one or both sacs);
- · oedematous placenta and foetus;
- hygromas on the knees, stifles, hock and angle of the haunch, and between the nuchal ligament and the primary thoracic spines.

In sheep:

- fever, increased respiration and depression;
- inferior quality of semen in rams;
- oedema and swelling of scrotum;



Brucellosis: hygromas on the knee joints – this condition may be a sequel to Brucella abortus infection

PHOTO 6.15

- in chronic stage, enlarged and hard epididymis, thickened scrotal tunics and frequently atrophic testicles;
- infertility in rams and abortion in ewes.

Judgement:

Cattle and horse carcasses affected with brucellosis are approved (after the removal of affected parts), as *Brucella* bacteria remain viable for only a short period in the muscles after slaughter. In the acute abortive form (after the miscarriage), cattle carcasses are condemned. Pig, sheep, goat and buffalo carcasses require total condemnation. Heat treatment may be recommended in some areas for these species due to economic reasons. Affected parts of the carcass, udder, genital organs and corresponding lymph nodes must be condemned.

Reactor animals should be carefully handled during slaughter and dressing procedures. Gloves and goggles should be worn when known reactors are being slaughtered and hygroma lesions should be sprayed liberally with 1% lactic acid at meat inspection.

Differential diagnosis:

Causes of abortion in cattle, IBR, vibriosis, leptospirosis, trichomoniasis, mycoplasma infections, mycosis, nutritional and physiological causes.

Discussion:

Brucella organisms have only a short life in the muscles of slaughtered animals. They are destroyed by lactic acid. While slaughtering and dressing the reactors, a hook should be used in handling the uterus and udder. Employees in close contact with infected animals should wear gloves and avoid accidental cuts.

In humans, brucellosis is called "undulant fever". The general population is not at risk from this disease if high levels of hygiene and sanitation are practised. Pasteurized milk is *Brucella*-free. Affected humans will suffer from intermittent high fever, headache and generalized malaise.

Brucellosis is an important zoonosis in particular in rural areas in developing countries and is an important occupational hazard for veterinarians, meat inspectors, farmers, animal health inspectors and butchers.

Anthrax

Anthrax is a peracute disease of ruminants manifested with septicaemia, sudden death and tarry blood from the body openings of the cadaver. It is caused by *Bacillus anthracis*.

Transmission:

Humans may contract anthrax by inhalation, ingestion and through a wound in the skin. Biting flies have been shown to be transmitters.

Ante-mortem findings:

The peracute and acute forms in cattle and sheep are without clinical signs. Death may follow in the acute form after one to two hours of illness. The acute form lasts about 48 hours.

In pigs and horses this disease is usually localized and chronic and is often characterized by swelling around the throat and head.

Ante-mortem findings in pigs:

- incubation: 1–2 weeks;
- oedematous swelling of the throat and neck;
- swallowing and breathing difficulties;
- death due to choking or toxaemia;
- · septicaemia is not observed.

Differential diagnosis:

Peracute blackquarter and septicaemic forms of other diseases. In splenic enlargement as seen in babesiosis, anaplasmosis and leucosis, spleen consistency is firm. In anthrax, the spleen is soft and upon incision the pulp exudes like thick blackish-red blood.

Discussion:

If an animal has died from an unknown cause in an abattoir's pen or in the stockyard, a blood smear from the tip of the ear should be examined to eliminate anthrax as a cause of death. All measures should be taken to prevent further contact with the carcass. The orifices of the nose, vulva and anus should be packed with cotton swabs to eliminate further spillage of discharge. The carcass must not be opened. As a result of insufficient oxygen supply in the closed carcass, spores of *B. anthracis* will not be formed and the organism will be killed. The spilled discharge is first removed by drying with sawdust and sand and is then destroyed together with the carcass. The carcass is wrapped in thick plastic sheets and destruction is performed under the supervision of an appropriate government official.

An open carcass facilitates exposure of *B. anthracis* to air and consequently spores are formed within a few hours. Anthrax spores are resistant to heat and disinfectants and may survive in a suitable environment for years.

An abattoir's pen or stockyard area suspected of being in contact with an anthrax animal should be disinfected with 10% NaOH or 5% formaldehyde and cleaned. This cleaning should also include the cattle trucks or cars used for the transportation of infected animals. All personnel that were in contact with anthrax, or that handled contaminated material, are also subjected to decontamination. The arms and hands should be washed with liquid soap and hot water. After they have been rinsed, they should be immersed for about one minute in an organic iodine solution or 1 ppm solution of mercuric perchloride or other acceptable agents. This is followed by a potable water rinse. Clothing of the personnel involved should also be cleaned and thoroughly disinfected by boiling.

If the carcass is discovered on the killing floor, all operations must cease. The carcass and its parts, including hides, hooves, viscera and blood must be condemned and destroyed. Carcasses that have been dressed by the same abattoir employees prior to or after the affected carcass must also be condemned and destroyed. Carcasses that had been dressed before the affected carcass may have a second option of being salvaged with sterilization. They must be boiled for a minimum of three hours if contamination occurred with blood splashes.

Disinfection of equipment used for the dressing of a diseased carcass as well as the infected abattoir area should be done with 5% solution of sodium hydroxide (NaOH). This disinfectant is used because of its action on fat and grease removal. Heat in the form of a blowtorch can be used for disinfecting buildings.

Salmonellosis in bovines

Salmonellosis is a disease that occurs in all animals and humans. In animals, salmonellosis is characterized clinically by one of three syndromes: a) peracute septicaemic form, b) acute enteritis, or c) chronic enteritis.

Young, old, debilitated and stressed animals are at greater risk. More than 200 antigenically different serotypes of Salmonella have been identified and all of these have pathogenic potential. The most frequently identified serotypes of the organisms that cause the disease in cattle are S. typhimurium, S. dublin, S. muenster and S. newport. Salmonellosis in stressed animals is frequently associated with inadequate diet, irregular feeding, water deprivation, overcrowding, parasitism, weather extremes, pregnancy, parturition, intercurrent diseases, etc. The calving complications that may predispose an animal to the disease include abortion or early termination of pregnancy, retained placenta, endometritis and postparturient metabolic conditions.

Transmission:

Ingestion of feed that has been contaminated by the faeces of infected animals, by drinkingwater in stagnant ponds and by contact with carrier animals. In housed animals, transmission is via contaminated feedstuff containing improperly sterilized animal by-products, such as bone-and-meat meal and fishmeal. Casual workers, infected clothing and utensils, transportation trucks and birds may transmit the disease to the farm. Active carrier animals shed *Salmonella* organisms intermittently and without obvious stress factors. Latent carriers with stress factors are also identified in the transmission of salmonellosis.

Human infection is transmitted via contaminated water, raw milk and meat. Compared to bovines, pigs and poultry are more significant sources of infection in humans.

Ante-mortem findings:

Peracute septicaemic form:

- occurs most frequently in colostrum-deficient animals up to four months of age;
- increased temperature 40.4–41.5 °C;
- · depression;
- diarrhoea and dehydration;
- death within 24-48 hours.

Approximately four weeks after the onset of diarrhoea:

- polyarthritis;
- meningoencephalitis;
- necrosis of distal limbs, tail and ears.
- Acute enteritis:
- common form in adult cattle in late pregnancy and early postpartum;
- high temperature of 40-41 °C;
- depression and loss of appetite;
- watery, foul smelling diarrhoea and dehydration;
- · emaciation;
- reduced milk production and abortion;
- death.

Chronic enteritis – preceded by acute enteric form:

- further emaciation (poor doer), diarrhoea and dehydration;
- fluctuating fever (35.5–40 °C).

Differential diagnosis:

Acute diarrhoea in calves: diarrhoea caused by infections (such as rotavirus, corona virus, cryptosporidiosis, *Escherichia coli*), septicaemia, dietetic gastroenteritis, coccidiosis, *Clostridium perfringens* type C enterotoxaemia.

Acute diarrhoea in adult cattle: BVD, coccidiosis, "grain overload", gastrointestinal parasitism, winter dysentery, arsenic and lead poisoning, bracken fern poisoning and intestinal obstruction.

Chronic diarrhoea of adult cattle: Johne's disease, copper deficiency and gastrointestinal parasitism.

Haemorrhagic septicaemia

Haemorrhagic septicaemia is a systemic disease of cattle, buffalo, pigs, yaks and camels. It is caused by *Pasteurella multocida* type B of Carter. Outbreaks of this disease are associated with environmental stresses such as wet chilly weather and overworked, exhausted animals. It is a specific type of pasteurellosis distinct from other forms of *Pasteurella* infections.

Transmission:

By ingestion of contaminated feedstuff.

Ante-mortem findings:

- disease more severe in buffalo than in cattle;
- high fever up to 42 °C;
- salivation and difficulties in swallowing;
- cough, and difficult breathing and associated pneumonia in later stages;

Calf diphtheria

Calf diphtheria is an acute oral infection of calves less than three months old. It is caused by *Fusobacterium necrophorum*. This agent also causes liver abscesses and foot-rot in cattle.

Transmission:

Fusobacterium necrophorum is an inhabitant of the digestive tract of cattle and of the environment. Under unhygienic conditions, infection may be spread on feeding troughs and dirty milk pails. Some of the contributory factors for occurrence of this disease include abrasions in the oral mucosa, animals suffering from poor nutrition and other (intercurrent) diseases present in young calves.

- oedematous swelling of throat, dewlap, brisket and peritoneum;
- diarrhoea.

Judgement:

The carcass of an animal affected with haemorrhagic septicaemia is condemned. If the disease is diagnosed on ante-mortem inspection, an animal should not be allowed to enter the abattoir. Dressing of such a carcass would create potential danger for the spread of infection to other carcasses.

Differential diagnosis:

Anthrax, blackleg, acute leptospirosis, RP, other forms of pasteurellosis, snake bite and lightning stroke.

Ante-mortem findings:

- · high temperature;
- coughing;
- · loss of appetite and depression;
- · difficult breathing, chewing and swallowing;
- swollen pharyngeal region;
- deep ulcers on the tongue, palate, and inside of cheeks;
- pneumonia.

Judgement:

The carcass of an animal affected with local lesions is approved. Generalized diphtheric lesions associated with pneumonia or toxaemia require condemnation of the carcass. The carcass is also condemned if lesions are associated with emaciation.

Differential diagnosis:

Vesicular diseases, neoplasms and abscesses.

Actinobacillosis ("wooden tongue")

Actinobacillosis is a chronic disease of cattle caused by *Actinobacillus lignieresi*. It is manifested by inflammation of the tongue, less frequently of the lymph nodes, and further inflammatory lesions in the head, the viscera and the carcass.

Ante-mortem findings:

- · loss of appetite;
- salivation and chewing;

- swollen tongue;
- · mouth erosions;
- enlarged parotid and retropharyngeal lymph nodes.

Differential diagnosis:

Neoplasms, tuberculosis, abscesses in the lymph nodes, foreign body, salivary cysts, fungal granulomas, chronic pneumonia and parasites.

Actinomycosis ("lumpy jaw")

Actinomycosis is a chronic granulomatous disease of cattle and pigs and rarely of sheep and horses. It is caused by *Actinomyces bovis*, which is an obligatory parasite in the mucous membrane of the mouth and pharynx. Infection occurs following injury with a sharp object or hard feed pieces to the oral mucosa.

Ante-mortem findings:

- painful swelling of the maxilla and mandible ("lumpy jaw") and, rarely, of the feet;
- suppurative tracts in the granulation tissue breaking towards oral cavity or skin;

- ulceration of cheeks and gums and wart-like granulations outward on head;
- difficult breathing and salivation;
- loss of weight;
- · diarrhoea and bloat.

Judgement:

See actinobacillosis.

Differential diagnosis:

Tooth infection, impacted food, bone injury, neoplasms and osteomyelitis due to other causes.

Pyelonephritis (contagious bovine pyelonephritis)

Pyelonephritis is a purulent and inflammatory bacterial disease of the kidney, pelvis and parenchyma caused by *Corynebacterium renale*. This disease is particularly observed in adult cows and sows. A predisposing factor for developing a kidney infection is trauma to the bladder and urethra during parturition.

Transmission:

Infection is spread from clinically normal "carrier cows". The organism enters via the vulva from: a) bedding contaminated with urine; b) tail swishing by "carrier cows"; c) venereal transmission by infected bulls; and d) nonsterilized obstetrical instruments.

Ante-mortem findings:

- persistent increased temperature (39.5 °C);
- loss of appetite and progressive weight loss;
- painful urination and increased frequency of urination;
- ammoniac odour from animal;
- acute abdominal pain (colic);
- ceased rumen contraction;
- · decreased milk production.

Differential diagnosis:

Enzootic haematuria in certain areas, postparturient haemoglobinuria, reticulitis, peritonitis, cystitis, metritis, leptospirosis, Johne's disease, white spotted kidneys of calves, urinary obstruction, infarcts, neoplasms and hydronephrosis.

Metritis

Metritis is inflammation of the uterus. This condition is of bacterial origin. It occurs as a result of calving problems such as retention of the placenta, abortion, twin births, abnormal labour and traumatic lesions of the uterus, cervix and vagina.

Ante-mortem findings:

- high fever and depression;
- muscular weakness;
- placental retention;
- listlessness;
- reddish fetid discharge from the vulva.

Differential diagnosis: Recent calving.

Mastitis

Mastitis is inflammation of the udder caused by bacteria, fungi and yeasts. Depending on the virulence of the agent and the resistance of the udder, mastitis is manifested in acute or chronic forms.

Ante-mortem findings:

- variable temperature depending on stage of condition;
- swollen warm, painful udder or hard enlargement involving one or all quarters;

- depression, loss of appetite and dehydration;
- abnormal gait caused by rubbing of the hind leg against inflamed quarter;
- · animal tends to lie down;
- purulent or bloody exudate from teats or watery pale fluid in chronic cases.

Differential diagnosis:

Oedema, haematoma and rupture of the suspensory ligament.

Endocarditis

Endocarditis is inflammation of the endocardium of the heart. The lesion is most commonly seen in the valves. It may be the result of bacteraemia caused by infection in some remote organs such as the udder, uterus or other sites.

In cattle, the organisms most commonly associated with endocarditis are *Actinomyces pyogenes* and *Streptococcus* spp. Strains of *E. coli* are also frequently found. The lesion is most commonly found on the valves. Portions of atheromatous material may become detached and released into the bloodstream as emboli that may lodge in other organs. They may be septic or aseptic. The latter contain thrombotic material. Emboli brought from the right heart to the lungs by blood vessels may cause pulmonary abscesses, or pulmonary thrombosis, and the emboli brought from the left heart to the spleen and kidneys may cause septic or aseptic infarcts in these organs. Abscesses in the heart may also be observed.

Ante-mortem findings:

- · moderate fever;
- breathing with accompanied grunt;
- pallor of mucosae;
- loss of condition and muscle weakness;
- temporary fall in milk production in lactating animals;
- jaundice and death.

Differential diagnosis:

Pneumonia, pericarditis, pulmonary oedema, emphysema, pleuritis, lymphoma, high altitude disease, congenital heart disease, congenital valvular heart cysts or deformities, especially in calves.

Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis)

TRP is caused by the perforation of the

reticulum by a metallic foreign body. It is mostly seen in adult dairy cattle and can occur in beef cattle.

Ante-mortem findings:

- sudden drop in milk production;
- · depression, loss of appetite and weight loss;
- stretched head and neck;
- reluctance to walk, arched back and tucked up abdomen;
- scant, hard faeces, sometimes, but rarely, covered with mucus;
- mild rumen bloat;

- audible "grunt" in early stages;
- if mild septicaemia develops the animal shows:
 - elevated temperature (40 °C);
 - increased heart rate.

In chronic localized peritonitis, acute signs and pain lessen, temperature falls and stomach reticulo-rumen motility may return.

Differential diagnosis:

Uterine or vaginal trauma, abomasal ulceration with perforation, liver abscessation, pyelonephritis, ketosis, abomasal displacement and volvulus, and "grain overload".

PARASITIC DISEASES

Diseases caused by helminths

Lungworms

Dictyocaulus viviparus is a lungworm in cattle causing verminous pneumonia or bronchitis, husk or hoose. Mature lungworms live in the bronchi. During coughing, the eggs are swallowed by the host. Hatching of eggs takes place in air passages or the digestive tract. Larvae are passed in the faeces. These will survive and develop on the ground if moist, and at moderate temperatures they become invasive in three to seven days. Larvae are resistant to cold, although their maturation will be delayed.

Upon ingestion by the primary host, larvae migrate through the intestinal wall to the mesenteric lymph nodes. From the mesenteric lymph nodes they pass via the lymphatics to the venous circulation and to the heart. From the heart they reach the lung alveoli. Three to six weeks after infection they migrate to bronchi where they mature and lay eggs. They survive seven weeks in bronchi where they terminate their life cycle.

Ante-mortem findings:

- elevated temperature (40-41 °C);
- rapid shallow breathing, which in later stages becomes laboured breathing;
- · nasal discharge;
- grunting;
- cyanosis and recumbency.

Judgement:

The carcass of an animal affected with lungworms is approved if infestation is slight and no secondary changes are observed. The lungs are condemned. The carcass is condemned if lungworm infestation has caused pneumonia that is accompanied by emaciation or anaemia.

Differential diagnosis:

Bacterial bronchopneumonia, abscess, necrobacillosis, tuberculosis, actinobacillosis, hydatid disease and atelectasis.

Fascioliasis

The term "fascioliasis" is commonly used to cover all liver flukes, but in fact there are various species. *Fasciola hepatica* is the most widespread in distribution. *Fasciola gigantica* is found in Africa and some parts of Southeast Asia, and *Fasciola magna* is found mostly in North America including Canada, and in Europe. In Zimbabwe between 30 and 70 percent of cattle slaughtered are infested with flukes. Usually the liver needs to be trimmed or condemned.

Fasciola hepatica (Photo 6.16) is the most common of liver flukes. It is leaf-shaped and measures 2.5–5 cm by 1.3 cm. It lives in the bile ducts of ruminants and other mammals.

Fasciola magna (Photo 6.17) is one of the largest flukes (10 cm by 2.5 cm), noted in the liver and rarely in the lungs of cattle, sheep, deer, moose, elk and other cervidae in Canada. It is found in North America. It may be differentiated from *Fasciola hepatica* by the absence of an anterior cone-like projection.

Fasciola gigantica is two or three times larger then *Fasciola hepatica*. It causes severe economic losses in cattle in Africa.



PHOTO 6.16

Numerous flukes of Fascicola hepatica observed in the bile ducts and liver parenchyma of a cow



PHOTO 6.17 Young fluke of Fascioloides magna taken from a bovine liver

Life cycle:

Each adult is hermaphroditic and produces fertilized eggs that are passed in the bile and faeces on to pasture. In the presence of water or moisture, the eggs hatch into larvae called miracidia. If the miracidia find a suitable intermediate host, which is usually the aquatic snail *Lymnaea truncatula*, they will develop into sporocysts. In different parts of the world different snails act as intermediate hosts.

The sporocysts divide to form rediae. The rediae transform into cercariae, which are the final larval stage of the cycle. They leave the snail and encyst into metacercariae. After ingestion by a herbivorous animal, the cyst wall is digested in the duodenum and the larvae cross the small intestine wall and peritoneal space to the liver. They penetrate the liver and make their way to the bile ducts and mature within a few weeks. The complete cycle of this fluke takes three to four months in favourable conditions.

Ante-mortem findings:

- weight loss and emaciation;
- fall in milk production;
- anaemia;
- · chronic diarrhoea;
- swelling in the mandibular area.

Judgement:

Judgement depends on the extent of the fluke lesions and the condition of the carcass. Severe infestation with associated emaciation or oedema would necessitate total condemnation of the carcass. Mild, moderate and heavy infestation without emaciation may have a favourable judgement. If the parasitic lesions in the liver are clearly circumscribed, the liver may be salvaged after trimming off affected tissue; otherwise it is condemned.

Differential diagnosis:

Melanosis, melanoma, *Dicrocelium dendriticum* and *Gigantocotyle explanatum* infestations in Southeast Asia.

Dicrocoelium dendriticum infestation

Dicrocoelium dendriticum (the lancet fluke) is the smallest of the four mentioned flukes in the liver.

Life cycle:

Two intermediate hosts are required for its complete cycle. The eggs excreted with faeces by the final host are ingested by a land snail. Many species of land snail can act as intermediate hosts where the miracidia develop into sporocysts and cercariae. *Cionella lubrica* is the principal first intermediate host in North America.

The cercariae are expelled by the snail in mucus and are deposited on plants. They are

further ingested by ants of the genus *Formica* where they develop into metacercariae. Several species of this genus can act as second intermediate hosts. In North America *Formica fusca* is the second intermediate host.

Ruminants, while grazing, may ingest these ants. The cyst wall of the metacercariae is digested and larvae then migrate to the bile ducts where they mature. *Dicrocoelium dendriticum* is only slightly pathogenic and does not produce clinical symptoms in the animal.

Judgement and differential diagnosis: See fascioliasis.

Oesophagostomiasis (pimply gut, nodular worms)

Oesophagostomiasis is a parasitic disease of ruminants and swine. *Oesophagostomum radiatum* is found in cattle, *Oesophagostomum columbianum* in sheep and *Oesophagostomum dentatum* in swine. The larvae in these species are found in the intestine, caecum and colon. In some southern African countries the parasite may affect 5–10 percent of cattle, sheep and pigs.

Life cycle:

The larvae develop to the infective stage on pasture. They are sensitive to cold, dryness and temperature changes. The infected larvae penetrate the intestinal mucosa and many of them become encysted. The larvae that penetrate into deeper mucosal layers provoke an inflammatory reaction and nodules of "pimply gut". Further stages of development occur in the intestinal wall. It is believed that many larvae are killed by the reaction they provoke in the intestine. When the larvae leave nodules due to malnutrition or lower resistance of the animal, they reach the colon. In the colon they become adults and attach themselves to the colonic mucosa where they lay eggs. A great number of nodules appear as gross lesions after

the departure of larvae. With repeated parasitic exposure, the host becomes immune and resistant to these larvae and local intestinal reaction becomes granulomatous. The nodules that surround dead larvae and those which calcify after caseation are persistent and they protrude from the intestinal wall. This may explain why nodules are present in adult animals and why no adult worms are observed in the intestinal lumen. In young animals that have no immunity, adult worms are present in the lumen of the intestine and nodules are lacking. There are some adults with both nodules and adult worms in the intestine. O. columbianum in sheep may cause extensive formation of nodules, which may become suppurative and may rupture. This leads to inflammation of the peritoneum and adhesions.

Ante-mortem findings:

- diarrhoea with black-green faeces which may be mixed with mucus and blood;
- · loss of condition and emaciation;
- stiff gait;
- young calves may show loss of appetite, diarrhoea, emaciation and anaemia.

Cysticercosis

Bovine cysticercosis is caused by Cysticercus bovis, which is the cystic form of the human tapeworm Taenia saginata.

Life cycle:

Cysticercus bovis is the larval stage of T. saginata. T. saginata may grow from 3 to 7 m in length and lives in the intestine of humans. It consists of a suckered head called scolex which is attached to the intestine. It also consists of a neck and hundreds of proglotid segments. Mature proglotids are filled with eggs. The proglotids break off and are excreted in the faeces where they fragment and release the ova. Cattle become infected by grazing on ground and by the digestion of foodstuff contaminated with human faeces. The oncosphere liberated in

FIGURE 6.1 The life cycle of Taenia saginata Infected meat eater human beind vaginated Cysticercus Gravid

Source: G.J. Jackson, Division of Microbiology, US FDA, Washington, DC.

diaphragm. In some countries in Africa the cysticerci appear to show uniform distribution in the musculature. If ingested by humans, the final or definite host, the scolex attaches itself to the intestinal wall and tapeworms then develop

Transmission:

and mature (Figure 6.1).

Infection in humans occurs following consumption of raw or undercooked beef containing viable cisticerci. Cattle become infected by ingestion of feedstuff containing ova passed from infected humans. Cattle raised on free range often become infected through contamination of grazing with human faeces. Infected farm workers may contaminate hay, silage, other feeds or sewage effluent. Intrauterine infection of a bovine foetus has also been recorded.

intestinal wall and through the lymphatics and

containing a scolex. The sites of predilection are

bloodstream reaches the skeletal muscles and heart. In the muscles the oncosphere develops

into the intermediate or cysticercus stage

the masseter muscles, tongue, heart and

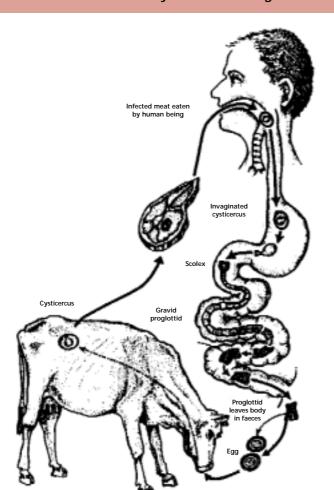
Ante-mortem findings:

Heavy infestation in cattle may show:

- muscle stiffness;
- rarely fever.

Differential diagnosis:

Hypoderma species (migration to heart), nerve sheath tumour, eosinophilic myositis, abscess and granuloma caused by injections.



the intestine from the egg penetrates the

Hydatid disease (hydatidosis, echinococcosis)

Hydatid disease in cattle is caused by the larval stages of the 2–7 mm long tapeworm *Echinococcus granulosus*, which lives in the intestines of dogs and other carnivores. Several strains of *E. granulosus* exist – the cattle/dog strain is primarily responsible for hydatid disease in cattle. In Africa, hydatid disease is reported more commonly in cattle that are communally owned or are raised on free range, and which associate more intimately with domestic dogs. Hydatidosis in domestic ruminants inflicts enormous economic damage because of the condemnation of affected organs and lowering of the meat, milk and wool production.

Life cycle:

The infective eggs containing the oncosphere passed in the faeces are accidentally ingested by cattle, sheep, pigs, other animals or humans, which act as intermediate hosts. After the infective eggs are ingested by these intermediate hosts, the oncospheres in the eggs penetrate the intestine and reach the liver, lungs and other organs, including the brain and muscles, to develop into hydatid cysts after about five months. These cysts commonly measure 5–10 cm and contain fluid. Some may reach up to 50 cm in diameter. Others may produce daughter cysts. The diagnostic features of hydatid cysts are a concentrically laminated thick outer layer within which is a germinal layer. In fertile hydatids, the germinal layer is granular and has brood capsules each containing protoscoleces. When brood capsules become detached and float free in the cysts' fluid they are referred to as hydatid sand. In some animals a fair proportion of hydatids may be sterile. The life cycle is completed when a fertile hydatid cyst is eaten by a definitive host, a dog or appropriate carnivore. Cattle and the majority of intermediate hosts show no clinical evidence of infection. However, in humans hydatid cysts can cause serious disease.

Ante-mortem findings:

None of significance.

Judgement:

A carcass showing emaciation, oedema and muscular involvement is condemned and destroyed. Otherwise the carcass is approved. Affected viscera and any other tissue are also condemned and destroyed. Burying of carcasses is not sufficient, since dogs may retrieve the affected organs.

Differential diagnosis:

Retention cysts in kidneys, cysts in liver, granulomatous lesions, *Cysticercus tenuicolis* and tuberculosis.

Onchocercosis

Onchocercosis in cattle is caused by nematodes of the genus *Onchocerca*. Several species are involved, but the most important species is *Onchocerca gibsoni*, which causes subcutaneous nodules or "worm nests" in cattle in some countries of the Asia–Pacific region and southern Africa.

Life cycle:

The adult worm lives in the nodules and the fertilized females liberate microfilariae into the tissue lymph spaces from where they are taken up by an insect vector that acts as an intermediate host. Midges of the genus *Cullicoides* are common vectors. Other biting flies can act as intermediate hosts. The larvae develop to the infective stage in these insect vectors. Infection of cattle occurs when these biting flies with infective larvae feed on them.

Ante-mortem findings:

Careful palpation reveals subcutaneous nodules in the brisket and buttock regions.

Differential diagnosis:

Abscesses, neurofibromatosis, cysticercosis, eosinophilic myositis.

Diseases caused by protozoa

Trypanosomiasis

This is a protozoan disease of animals and humans caused by parasites of the genus *Trypanosoma*, which are found in blood plasma, various body tissues and fluids.

Transmission:

Trypanosomes (*Trypanosoma* spp.) are transmitted primarily by the *Glossina* spp. (tsetse flies), *Stomoxys* spp. (stable flies), Tabanidae (horseflies) and Reduviidae (assassin bugs), and by venereal contact. Trypanosomes in the insect vector undergo one or two cycles of development.

Ante-mortem findings:

- intermittent fever;
- · anaemia;
- weight loss and weakness;
- oedema, particularly observed in the face and legs;
- · enlarged body lymph nodes;
- haemorrhage;
- opacity of the cornea, keratitis and photophobia;
- chronic form of trypanosomiasis is sometimes manifested by progressive weakness, despite absent parasitaemia, and death.

Judgement:

The carcass affected with trypanosomiasis or any other protozoan diseases is condemned if an acute condition is associated with systemic body changes. Heat treatment may be recommended in some cases if economically feasible. The carcasses of recovered and reactor animals may be approved if generalized lesions are lacking. A carcass showing borderline emaciation or slight oedema should be examined after 24–48 hours in the chiller. A satisfactory setting would lead to a favourable judgement of the carcass. The affected parts of the carcass and organs are condemned.

Differential diagnosis:

Helminthiasis, malnutrition and other chronic wasting diseases, equine infectious anaemia, heartwater, babesiosis and anaplasmosis.



PHOTO 6.18 Trypanosomiasis: this animal shows icteric mucous membranes, weakness in leg muscles and emaciation

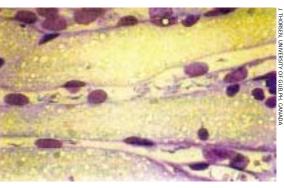


PHOTO 6.19 An impression smear of the

An impression smear of the trypanosomes and the red blood cells in the capillaries

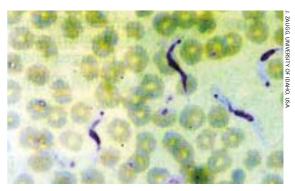


PHOTO 6.20 Trypanosoma vivax in blood smear

Theileriosis (East Coast fever)

East Coast fever is a subacute haemoprotozoan disease of cattle caused by Theileria parva. Theileriosis is characterized by fever, enlarged lymph nodes, dyspnea and death. In chronic cases loss of condition, emaciation, diarrhoea, blindness, etc. can be seen.

Transmission:

Vectors are ixodid ticks of the species Rhipicephalus.

Ante-mortem findings:

- mortality up to 90 percent;
- high temperature (up to 41 °C);
- · difficult breathing and coughing;
- nasal discharge, salivation and watery eyes;
- swelling of the lymph nodes draining the area where the infected tick fed (Photo 6.21);
- · cerebral signs manifested by circling to one side, convulsions and death.

Judgement:

The carcass and viscera of an animal affected with febrile chronic theileriosis and without systemic lesions are approved.

The carcass is condemned, if acute febrile theileriosis is accompanied with fever and generalized lesions. The affected organs are also condemned.

Differential diagnosis:

Haemorrhagic septicaemia, babesiosis, MCF, trypanosomiasis, Rift Valley fever, heartwater and bovine leukosis.



PHOTO 6.21 East Coast fever (theileriosis): enlarged body lymph nodes

Besnoitiosis

Besnoitiosis is a chronic debilitating protozoan disease of cattle and horses. It also occurs in wild animals such as antelope and wildebeest (gnu) in Africa and caribou in Canada. The causative agent in cattle is Besnoitia besnoiti and Besnoitia benetti in horses.

The organism is closely related to the genus Toxoplasma. The mode of transmission is still unknown. It is believed that tabanids are mechanical vectors.

Ante-mortem findings:

- elevated temperature;
- increased respiration;
- nasal discharge and lacrimation;
- diarrhoea;
- · cysts in the skin and subcutaneous tissue and loss of hair;

- swollen body lymph nodes;
- severe generalized oedema of the head, neck, ventral abdomen and legs;
- · chronic skin lesions show in folding and cracking;
- decreased milk production;
- inflammation of the testicles.

Judgement:

The carcass is approved if the lesions are localized with no systemic involvement. The carcass is condemned if disseminated, generalized lesions are accompanied with emaciation.

Differential diagnosis:

Lumpy skin disease, sweating sickness and ectoparasitism (mites, ticks, fungi).

Anaplasmosis (gall sickness)

Anaplasmosis is a disease caused by a rickettsialike organism and characterized by severe debility, emaciation, anaemia and jaundice. It is caused by *Anaplasma* spp. They are obligate intraerythrocytic parasites. *Anaplasma marginale* is the causative agent in cattle and wild ruminants.

Transmission:

Ticks of the genus *Boophilus* transmit anaplasmosis. Mosquitoes and horseflies are mechanical transmitters. Transmission is also possible through injection needles.

Ante-mortem findings:

Acute infection with A. marginale:

- · high fever;
- jaundice and anaemia demonstrated by pale mucous membranes;
- frequent urination and constipation.
- Chronic infection:emaciation.

Judgement:

The carcass of an animal showing acute infection should be condemned. Recovered and "suspect" animals manifesting inconclusive signs of anaplasmosis are approved if otherwise healthy.

A mildly yellow discoloured carcass may be chilled and assessed after setting. If the discoloration has disappeared, the carcass is approved. Animals affected with anaplasmosis could be treated under the supervision of a government official. Guidelines for the withdrawal period for therapeutic agents should be followed if the animals are being shipped for slaughter.

Differential diagnosis:

Icterus and anaemia of different causes, anthrax, leptospirosis, emaciation caused by parasitism and malignant lymphoma, babesiosis.

Remarks:

The access of biting insects to contaminated fresh blood should be prevented. Blood from suspicious carcasses should not be salvaged.

Babesiosis (piroplasmosis, Texas fever, red water fever, tick fever)

Babesiosis of cattle, horses, sheep and swine is a febrile, tick-borne disease caused by various species of the protozoan genus *Babesia*.

Transmission:

Different species of ticks in the family Ixodidae serve as vectors in different locations. The *Babesia* parasites can be transmitted transstadially and transovarially within a tick species.

Ante-mortem findings:

- incubation: 7–10 days;
- mortality up to 50 percent or over depending on age, breed, etc;
- high fever (41.5 °C);
- · dark reddish brown urine in the terminal stage;
- reddened and injected mucous membranes at the early stages and later, anaemic mucous membranes;
- clinical signs may resemble rabies in cerebral form of babesiosis.

Judgement:

The carcass of an animal in the acute form of the disease, with associated icterus, is condemned. An emaciated, jaundiced carcass showing yellow gelatinous fat also requires total condemnation. A mild form of this disease showing yellow or orange coloration of the carcass, not associated with icterus, may be approved. The satisfactory setting of the carcass in the chiller must be considered in this approval.

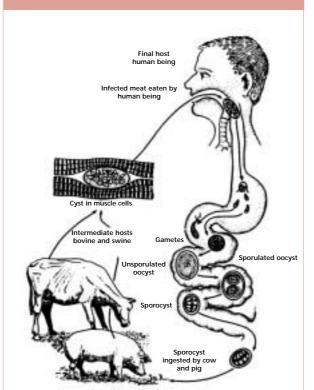
Differential diagnosis:

Anaplasmosis, trypanosomiasis, theileriosis, leptospirosis and bacillary haemoglobinuria.

Sarcocystosis (sarcosporidiosis)

Sarcocystosis is caused by the various species of the protozoan genus *Sarcocystis*. This is one of the most common parasitic conditions in domestic food animals and a high percentage of cattle in various parts of the world are infested

FIGURE 6.2 The life cycle of Sarcocystis hominis (cattle) and Sarcocystis suihominis (pigs) in the final host (humans)



Source: G. J. Jackson, Division of Microbiology, US FDA, Washington, DC.

with these parasites, which are usually hostspecific. In cattle, three species have been recognized. They are listed in Table 6.1. Cattle are the intermediate hosts of *Sarcocystis* spp. All *Sarcocystis* spp. in the intermediate hosts, the food animals, are characterized by the formation of cysts in the muscles.

Life cycle:

All *Sarcocystis* spp. require two hosts and a prepredator cycle to complete their life cycle. A herbivore – the prey – and a carnivore or omnivore – the predator – are involved. Sexual development occurs in the predator, which is therefore a definitive host, and asexual development occurs in the prey, which is the intermediate host. Two species, one in cattle (*S. hominis*) and one in pigs (*S. suihominis*), use humans as definitive hosts and therefore these infections in animals are zoonoses (Figure 6.2). Generally speaking, dog-transmitted *Sarcocystis* are pathogenic, whereas cattransmitted ones are not.

The most important species in cattle is *S. cruzi*, which has worldwide distribution and uses the dog as the definitive host. Sexual development takes place in the dog after which infective sporocysts are passed in the faeces. The details of development in cattle are illustrated in Figure 6.3.

The buffalo is the intermediate host for two species: *S. levinei*, which forms microscopic cysts and uses the dog as the definitive host, and *S. fusiformis*, which forms macroscopic spindle or globular-shaped cysts measuring 32 mm x 8 mm and uses the cat as the definitive host.

Species	Distribution	Definitive host/s	Size of cyst	Pathogenicity
S. cruzi	Worldwide	Dog, coyote, red fox, raccoon and wolf	Microscopic, less than 0.5 mm long	Most pathogenic species; in cattle it can cause fever, anaemia, abortion, neurological signs and even death
S. hirsuta	Probably worldwide	Cat	Macroscopic, up to 8 mm long and 1 mm wide, fusiform in shape	Mildly pathogenic
S. hominis	Europe	Humans and some primates	Microscopic	Mildly pathogenic to cattle

TABLE 6.1 Sarcocystis spp. in cattle

S. fusiformis cysts are seen in the oesophagus and the skeletal musculature and are common parasites of the water buffalo in many parts of the world.

Transmission:

Cattle acquire infection by ingesting sporocysts contaminating feed, pasture or water. After several generations of asexual reproduction by schizogony, they form cysts in muscles. *S. cruzi*, the most pathogenic species for cattle, forms microscopic cysts. The definitive hosts, including humans, acquire the infection when

they eat bovine tissues containing the viable *Sarcocystis* cysts. The data provided below are

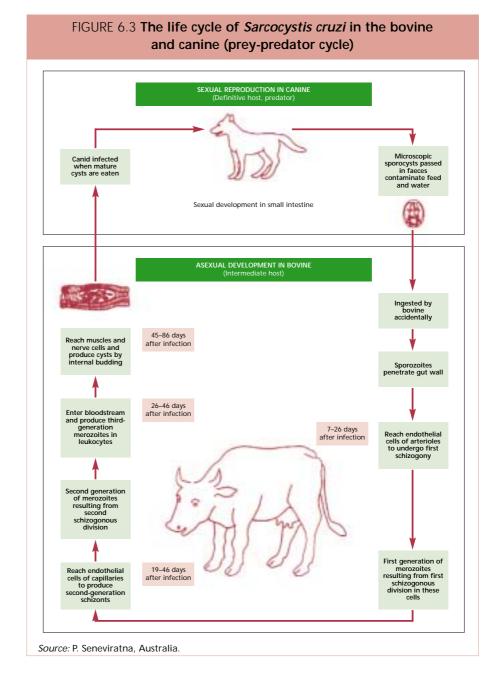
for *S. cruzi* infestations, unless specified otherwise.

Ante-mortem findings:

- incubation: 5–11 weeks;
- fever;
- · loss of appetite;
- excessive salivation;
- anaemia;
- · abortion;
- loss of hair, especially at the tip of the tail.

Differential diagnosis:

Cysticercosis, toxoplasmosis, neurofibromatosis, eosinophilic myositis.



Diseases caused by arthropod parasites

Hypoderma bovis infestation

There are two warble flies in cattle, *Hypoderma bovis* and *Hypoderma lineatum*. They have similar cycles. During the summer, the adult fly lays its eggs on the leg hair and occasionally on the body of cattle. Within a week the larvae hatch and burrow into the skin and, for several months, they travel through the body. *H. bovis* migrates into the thoracic and abdominal cavities towards the spinal canal before moving under the skin of the back. *H. lineatum* migrates to the

oesophageal area before reaching the dorsal area of the animal. In spring (February–May), the larvae reach the area of the back. They burrow a breathing hole and increase in size to approximately 8 mm x 25 mm. They are visible for a month. After this cycle, maggots fall to the ground where they develop into flies and start the whole cycle once again (Figure 6.4).

Ante-mortem findings:

- swelling or eroded skin on the back;
- larvae protruding from the skin of the back (Photo 6.22);
- cattle may rush violently and kick the abdomen with hind legs;
- · erected tail;
- paralysis of the lower body and legs if the spinal cord is involved.

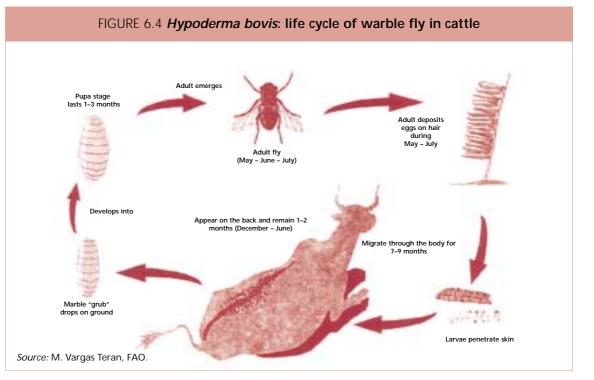
Judgement:

The carcass of an animal affected with *H. bovis* is approved. Subcutaneous lesions are removed.

Differential diagnosis:

Cysticercus bovis cysts in oesophagus.

PHOTO 6.22 Hypoderma bovis: larvae protruding from back in a two-year-old steer





50

Screwworm myiasis

Screwworm myiasis caused by larvae of the flies *Cochlliomyia hominironux* (New World screwworm – NWS) and *Chrysomya bezziana* (Old World screwworm – OWS) is characterized by larvae feeding on living tissues in open wounds of any warm-blooded host, including humans, resulting in weight loss, other signs of morbidity and sometimes death. NWS is found in Central and South America, including the Caribbean region. OWS is located in India, Southeast Asia, tropical Africa and in the Persian Gulf area.

Life cycle:

In the preferred temperature range (20–30 °C) it is about 21 days. The female, which mates only once, lays one or more batches of up to 300 eggs at the edge of any wound or break in the skin in any warm-blooded animal. Skin breaks as small as tick-bites, as well as natural orifices, can be sites of oviposition. The larvae develop within 24 hours, and burrow into the living flesh, creating large, deep, open wounds that attract further egg-laying females. If unattended, these wounds are often fatal, particularly in newborn animals where the oviposition site is usually the navel (Figure 6.5).

Ante-mortem findings:

A serosanguinous discharge often exudes from the infested wounds (Photo 6.23), and a



FIGURE 6.5 The life cycle of the screwworm

distinctive odour may be detected. In some cases, the openings in the skin may be small with extensive pockets of screwworm larvae beneath (Photo 6.24). In dogs, screwworm larvae commonly tunnel under the skin. Screwworm infestations in anal, vaginal and nasal orifices may be difficult to detect, even in the later stages.

Differential diagnosis:

Other blow flies such as *Compsomyia macellaria* and *Sarcophagidae* spp.



PHOTO 6.23 Wound of adult bovine infested by NWS



PHOTO 6.24 Typical pocket-like wound from screwworm larvae

Bibliography

- **Canadian Food Inspection Agency.** 2004. *Meat hygiene manual of procedures* (available at http://www.inspection.gc.ca/english/anima/meavia/mmopmmhv/mane.shtml).
- **Doherty, A.M.** 1999. Cattle cleanliness and its effect on carcass contamination. *Hygiene Review* (available at http://www.sofht.co.uk/isfht/irish_99_cattle.htm).
- **FAO/WHO.** 2004. Draft code of hygienic practice for meat. In *Report of the 10th Session of the Codex Committee on Meat Hygiene*. Alinorm 04/27/16. Rome (available at ftp://ftp.fao.org/codex/Alinorm04/AL04_16e.pdf).