CHAPTER 7

Host resistance to parasitic diseases

Broadly speaking, resistance to parasitic infections falls into two categories. The first of these, often termed innate resistance, includes species resistance, age resistance and in some cases breed resistance, which, by and large, are not immunological in origin. The second category, acquired immunity, is dependent on antigenic stimulation and subsequent humoral and cellular responses. Although, for reasons explained in this chapter, there are few vaccines available against parasitic diseases, natural expression of acquired immunity plays a highly significant role in protecting animals against infections and in modulating the epidemiology of many parasitic diseases.

SPECIES RESISTANCE

For a variety of parasitological, physiological and biochemical reasons, many parasites do not develop at all in other than their natural hosts; this is typified by, for example, the remarkable host specificity of the various species of *Eimeria*. In many instances, however, a limited degree of development occurs, although this is not usually associated with clinical signs; for example, some larvae of the cattle parasite *Ostertagia ostertagi* undergo development in sheep, but very few reach the adult stage. However, in these unnatural or aberrant hosts, and especially with parasites which undergo tissue migration, there are occasionally serious consequences, particularly if the migratory route becomes erratic. An example of this is visceral larva migrans in children due to *Toxocara canis*, which is associated with hepatomegaly and occasionally ocular and cerebral involvement.

Some parasites, of course, have a very wide host range, *Trichinella spiralis*, *Fasciola hepatica*, *Cryptosporidium parvum* and the asexual stages of *Toxoplasma* being four examples.

AGE RESISTANCE

Many animals become more resistant to primary infections with some parasites as they reach maturity. For example, ascarid infections of animals are most likely to develop to patency if the hosts are a few months old. If hosts are infected at an older age, the parasites either fail to develop or are arrested as larval stages in the tissues; likewise, patent *Strongyloides* infections of ruminants and horses are most commonly seen in very young animals. Sheep of more than 3 months of age are relatively resistant to *Nematodirus battus*, and in a similar fashion dogs gradually develop resistance to infection with *Ancylostoma* over their first year of life.

The reasons underlying age resistance are unknown, although it has been suggested that the phenomenon is an indication that the host–parasite relationship has not yet fully evolved. Thus, while the parasite can develop in immature animals, it has not yet completely adapted to the adult.

On the other hand, where age resistance is encountered, most parasitic species seem to have developed an effective countermechanism. Thus, *Ancylostoma caninum*, *Toxocara canis*, *Toxocara mystax*, *Toxocara vitulorum* and *Strongyloides* spp. all survive as larval stages in the tissues of the host, only becoming activated during late pregnancy to infect the young *in utero* or by the transmammary route. In the case of *Nematodirus battus*, the critical hatching requirements for the egg, i.e. prolonged chill followed by a temperature in excess of 10°C, ensure the parasites' survival as a lamb-to-lamb infection from one season to the next.

Oddly enough, with *Babesia* and *Anaplasma* infection of cattle, there is generally thought to be an inverse age resistance, in that young animals are more resistant than older naive animals.

BREED RESISTANCE

In recent years, there has been considerable practical interest in the fact that some breeds of domestic ruminants are more resistant to certain parasitic infections, such as coccidian protozoa, nematodes, ticks and flies, than other breeds.

Probably the best example of this is the phenomenon of trypanotolerance displayed by West African humpless cattle such as the N'dama and the West African Shorthorn cattle, which survive in areas of heavy trypanosome challenge. The mechanism whereby these cattle control their parasitaemias is still not fully known, although it is thought that immunological responses may play a role.

In helminth infections, it has been shown that Red Masai sheep, indigenous to East Africa, are more resistant to *Haemonchus contortus* infection than some imported breeds studied in that area, while in South Africa it has been reported that the Merino is less susceptible to trichostrongylosis than certain other breeds. In the USA, the Florida Native (Gulf Coast), Barbados Blackbelly and the St Croix breeds of sheep are considerably more resistant to *H. contortus* than the Merino or the European breeds.

Within breeds, haemoglobin genotypes have been shown to reflect differences in susceptibility to *H. contortus* infection in that Merino, Scottish Blackface and Finn Dorset sheep, which are all homozygous for haemoglobin A, develop smaller worm burdens after infection than their haemoglobin B homozygous

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or heterozygous counterparts. Unfortunately, these genotypic differences in susceptibility often break down under heavy challenge.

Studies within a single breed have shown in Australia that individual Merino lambs may be divided into responders and non-responders on the basis of their immunological response to infection with *Trichostrongylus colubriformis* and that these differences are genetically transferred to the next generation.

The selection of resistant animals could be of great importance, especially in many developing areas of the world, but in practice would be most easily based on some easily recognisable feature such as coat colour rather than be dependent on laboratory tests.

In Australia resistance to ticks, particularly *Rhipicephalus* (*Boophilus*), has been shown to be influenced by genetics, being high in the humped, *Bos indicus*, Zebu breeds and low in the European, *Bos taurus*, breeds. However, where cattle are 50% Zebu, or greater, in genetic constitution, a high degree of resistance is still possible allowing a limited use of acaricides.

ACQUIRED IMMUNITY TO HELMINTH INFECTIONS

Multicellular parasites such as helminths are very complex genetic organisms and because of their physical size they are unable to be consumed by phagocytic cells or destroyed by classic cytotoxic T cells. The host's immune system has had to devise new approaches to combat invasion by these parasites. These are generally referred to as type 2 immune responses (T-helper 2 or Th2) or the allergy-type immune responses. They are frequently characterised by increases in the concentrations of interleukin (IL)-4 and other Th2-type cytokines, such as IL-5, IL-9, IL-13 and IL-21. These responses are usually characterised by the recruitment and activation of effector cells, such as eosinophils, basophils and mast cells which can produce various type 2 cytokines. There is continual communication between the innate and adaptive components of an active immune response to parasite invasion, with the T cells evoking signals that increase and modify the function of innate effector cells. The specific effector cells and antibody classes that mediate protection in the host immune responses vary considerably between different parasites. Individual effector-cell types may also have multiple functions. Frequently, parasite infestation causes chronic infections and the immune response that develops over a prolonged period can induce pathological changes in tissues. For example, in schistosome infections antigens shed from eggs can induce a marked Th2-type response that stimulates the development of granulomatous lesions in the liver parenchyma. Immune responses to helminths are complex, possibly depending on antigenic stimulation by secretory or excretory products released during the development of the L_3 to the adult. For this reason it has only been possible to develop one or two practical methods of artificial immunisation, of which the radiation-attenuated vaccine against *Dictyocaulus viviparus* is perhaps the best example.

Despite this, there is no doubt that the success of many systems of grazing management depend on the gradual development by cattle and sheep of a naturally acquired degree of immunity to gastrointestinal nematodes. For example, experimental observations have shown that an immune adult sheep may ingest around 50,000 *Teladorsagia* (*Ostertagia*) L_3 daily without showing any clinical signs of parasitic gastritis.

EFFECT OF THE IMMUNE RESPONSE

Dealing first with gastrointestinal and pulmonary nematodes, the effects of the immune response may be grouped under three headings, the sequence reflecting the usual progression of acquired immunity.

1. Initially, the host can attempt to limit reinfection by preventing the migration and establishment of larvae and penetration of the mucosal barrier or, sometimes, by arresting their development at a larval stage. This type of inhibition of development should not be confused with the more common hypobiosis triggered by environmental effects on infective larvae on pasture or, in the present state of knowledge, with the arrested larval development associated with age resistance in, for example, the ascarids.
2. Adults that do develop may be stunted in size or their fecundity may be reduced. The important practical aspect of this mechanism is perhaps not so much the reduced pathogenicity of such worms as the great reduction in pasture contamination with eggs and larvae, which in turn reduces the chance of subsequent reinfection.
3. The development of immunity after a primary infection may be associated with an ability to kill or expel the adult nematodes.

Each of these mechanisms is exemplified in infections of the rat with the trichostrongyloid nematode *Nippostrongylus brasiliensis*, a much-studied laboratory model, which has contributed greatly to our understanding of the mechanisms of host immunity in helminth infection. The infective stage of this parasite is normally a skin penetrator, but in the laboratory is usually injected subcutaneously for convenience. The larvae travel via the bloodstream to the lungs where, having moulted, they pass up the trachea and are swallowed. On reaching the small intestine they undergo a further moult and become adult, the time elapsing between infection and development to egg-laying adults being 5–6 days. The adult population remains static for about another 5 days. After this time the faecal worm egg output drops quickly, and the majority of the worms are rapidly expelled from the gut. This expulsion of adult worms, originally known as the 'self-cure' phenomenon, has been shown to be due to an immune response.

If the rats are reinfected, a smaller proportion of the larval dose arrives in the intestine, i.e. their migration is stopped. The few adult worms which do develop in the gut remain stunted and are relatively infertile, and worm expulsion starts earlier and proceeds at a faster rate.

Under natural grazing conditions larval infections of cattle and sheep are acquired over a period, but an approximately similar series of events occurs. For example, calves exposed to *Dictyocaulus viviparus* quite rapidly acquire patent infections, readily recognisable by the clinical signs. After a period of a few weeks, immunity develops and the adult worm burdens are expelled. On subsequent exposure in succeeding years such animals are highly resistant to challenge, although if this is heavy, clinical signs associated with the reinfection syndrome (i.e. immunological destruction of the invading larvae in the lungs) may be seen. With *Ostertagia* and *Trichostrongylus* infections, the pattern is the same, with the build-up of an infestation of adult worms being followed by their expulsion and subsequent immunity; in later life only small, short-lived, adult infections are established and eventually the infective larvae are expelled without any development at all. However, with gastrointestinal infections in ruminants, the ability to develop good immune

responses is often delayed for some months because of immunological unresponsiveness.

The mechanism of immunity to luminal parasites is still not fully understood despite considerable research. This response includes both innate and adaptive components. However, it is generally agreed that such infections stimulate a Th2-type response and produce a state of gut hypersensitivity associated with an increase in mucosal mast cells in the lamina propria and the production of worm-specific IgE, much of which becomes bound to the surface of the mast cells. The reaction of worm antigen, from an existing infection or from a subsequent challenge, with these sensitised mast cells releases vasoactive amines, which cause an increase in capillary and epithelial permeability and hyperproduction of mucus. Some workers have concluded that these physiological changes simply affect the well-being of the worms by, for example, lowering the oxygen tension of their environment so that they become detached from the mucosa and subsequently expelled. Others have postulated that, in addition, the permeable mucosa allows the 'leakage' of IgG anti-worm antibody from the plasma into the gut lumen, where it has access to the parasites. Although the majority of helminths induce marked mast-cell responses, their role in mediating resistance to infection varies considerably between parasite infections. For example, in rodent models, the presence of mast cells is required for the expulsion of *Trichinella spiralis*, whereas mast cells are not essential for the rejection of *Nippostrongylus brasiliensis*.

Additional factors, such as the secretion of specific anti-worm IgA on the mucosal surface and the significance of sensitised T cells, which are known to promote the differentiation of mast cells, eosinophils and mucus-secreting cells, are also currently under study.

With regard to tissue-invading helminths, the most closely studied have been the schistosomes. Schistosomulae of *Schistosoma mansoni* may be attacked by both eosinophils and macrophages, which attach to the antibody-coated parasite. Eosinophils, especially, attach closely to the parasites where their secretions damage the underlying parasite membrane. Eosinophils release a secondary granule protein and can also produce cytokines (IL-4, IL-13) which can have a regulatory role. Attempts to find if a similar mechanism exists against *Fasciola hepatica* have indicated that although eosinophils do attach to parts of the tegument of the young fluke, the latter seems able to shed its surface layer to evade damage.

In *Schistosoma* infections, there is an initial Th1-type response to the acute infection that is directed against adult parasites, but following patency and egg deposition in tissues the response changes to predominately a Th2-type response. In cases where an effective Th2-type response fails to develop after egg deposition, the outcome is often increased granulomatous inflammation in the tissues and liver parenchyma, induced by the underlying Th1-type response. Thus the protective Th2-type response minimises the pathological consequences of a Th1-type response and the outcome is often mild granulomas composed of macrophages, lymphocytes and eosinophils.

EVASION OF THE HOST'S IMMUNE RESPONSE

Despite the evidence that animals are able to develop vigorous immune responses to many helminth infections, it is now clear that parasites, in the course of evolution, have capitalised on certain defects in this armoury. This aspect of parasitology is still in its infancy, but four examples of immune evasion are described here.

Neonatal immunological unresponsiveness

This is the inability of young animals to develop a proper immune response to some parasitic infections. For example, calves and lambs fail to develop any useful degree of immunity to reinfection with *Ostertagia* spp. until they have been exposed to constant reinfection for an entire grazing season. Similarly, lambs remain susceptible to *H. contortus* infection until they are between 6 and 12 months old. The cause of this unresponsiveness is unknown. However, while calves and lambs ultimately do develop a good immune response to *Ostertagia* or *Teladorsagia* infection, in the sheep/*H. contortus* system the neonatal unresponsiveness is apparently often succeeded by a long period of acquired immunological unresponsiveness, for example Merino sheep reared from birth in a *Haemonchus*-endemic environment remain susceptible to reinfection throughout their entire lives.

Concomitant immunity

This term is used to describe an immunity which acts against invading larval stages, but not against an existing infection. Thus a host may be infected with adult parasites, but has a measure of immunity to further infection. Perhaps the best example is that found with schistosomes, which are covered by a cytoplasmic syncytium that, unlike the chitinous-like cuticle of nematodes, would at first seem to be vulnerable to the action of antibody or cells. However, it has been found that adult schistosomes have the property of being able to incorporate host antigens, such as blood group antigens or host immunoglobulin, on their surface membrane to mask their own foreign antigens.

Concomitant immunity does not appear to operate with *F. hepatica* in sheep, in that they remain susceptible to reinfection. On the other hand, cattle not only expel their primary adult burden of *F. hepatica*, but also develop marked resistance to reinfection. Concomitant immunity also includes the situation where established larval cestodes may survive for years in the tissues of the host, although the latter is completely immune to reinfection. The mechanism is unknown, but it is thought that the established cyst may be masked by host antigen or perhaps secrete an 'anti-complementary' substance which blocks the effect of an immune reaction.

Polyclonal stimulation of immunoglobulin

As well as stimulating the production of specific IgE antibody, helminths 'turn on' the production of large amounts of non-specific IgE. This may help the parasite in two ways. First, if mast cells are coated by non-specific IgE, they are less likely to attract parasite-specific IgE and so will not degranulate when exposed to parasite antigen. Secondly, the fact that the host is producing immunoglobulin in a non-specific fashion means that specific antibody to the helminth is less likely to be produced in adequate quantity.

Parasite immunomodulation

Despite the diversity of the helminth parasites, they show common ways of evading or manipulating the host immune response to their benefit. They suppress immunopathology by modulating the activity of immune effector cells (different B and T cell types) and the expression of a range of cytokines. Thus distantly related

parasites have independently evolved to exploit a range of host immunoregulatory mechanisms to their own advantage, and by invoking generic suppressive pathways can also suppress bystander responses to allergens and self-antigens. *Fasciola hepatica*, the liver fluke, is a common parasite of cattle in much of the world and suppresses interferon (IFN)-γ responsiveness in cattle infected with bovine tuberculosis. The determination of blood IFN-γ levels is an important element of tuberculosis testing programmes and it has been suggested that the presence of *F. hepatica* in infected animals could markedly interfere with tuberculosis eradication programmes where tuberculosis incidence and *F. hepatica* infection are both high.

COST OF THE IMMUNE RESPONSE

Sometimes, immune responses are associated with lesions that are damaging to the host. For example, the pathogenic effects of oesophagostomosis are frequently attributable to the intestinal nodules of *Oesophagostomum columbianum*; similarly, the pathogenic effects of schistosomosis are due to the egg granulomas, the result of cell-mediated reactions, in the liver and bladder.

There is evidence from some studies for a negative genetic interaction between production traits and resistance to parasitism. Sheep which have been selected for their resistance to gastrointestinal nematode infection show a higher incidence of scouring. This may be the result of an increased hypersensitivity to ingested larvae. Indeed, there is now a general consensus that the host's immune responses and immune pathology directly contribute to the impaired productivity observed in parasitised livestock.

Developing and implementing a strong effective immune response against parasite invasion, establishment or subsequent reinfection will utilise essential host resources, in particular protein, since antibodies, cytokines, leukotrienes and effector cellular responses are highly proteinaceous. These effects will be more deleterious in situations where nutrient supply is limited. Thus in high production animals on lower planes of nutrition it may not be desirable to induce a strong immune response. However, these consequences have to be weighed against the need to maintain protection against the potential pathogenic effects of the parasitic infection. One of the consistent features of many gastrointestinal infections of ruminants is a reduction in voluntary food intake (parasite-induced anorexia), although recent data have shown that the appetite of immunosuppressed parasitised lambs is similar to that of their uninfected controls. This observation might imply that the cascade of events involved in mounting an effective immune response against infection may be partly responsible for the parasite-induced reduction of food intake. Thus the cost of mounting an effective immune response may outweigh the benefits gained in some situations, although caution is required as these effects may vary between parasite species, host species and genotype. Longer-term studies would suggest that the lowering of production performance in younger livestock, which often occurs as they are initially acquiring a protective immune response to a gastrointestinal parasite infection, may be offset by the reduction in susceptibility to larval challenge observed in older animals and also the lower infectivity of pastures that results from reduced contamination with nematode eggs. Now that this complex relationship between the parasite, nutrition and the acquisition of immunity is better understood, it is hoped that in the future the trade-off between the nutrient cost of developing an effective immune response and the economic benefit of maintaining an acceptable level of performance can be addressed in order to maintain the nutrient status of the parasitised animal, particularly in situations where nutrient supply is limited.

ACQUIRED IMMUNITY TO PROTOZOAL INFECTIONS

As might be anticipated from their microscopic size and unicellular state, immunological responses against protozoa are similar to those directed against bacteria. However, the subject is exceedingly complex and the following account is essentially a digest of current information on some of the more important pathogens. As with bacterial infections, immune responses are typically humoral or cell-mediated in type and occasionally both are involved. The cell-mediated responses are typically IFN-γ-dominant Th1-type responses which are associated with an increase in the number of cytotoxic CD8+ T cells, Th1 cells, macrophages and neutrophils.

Trypanosomosis is a good example of a protozoal disease to which immunity is primarily humoral. Thus, *in vitro*, both IgG and IgM can be shown to lyse or agglutinate trypanosomes and *in vivo* even a small amount of immune serum will clear trypanosomes from the circulation, apparently by facilitating their uptake, through opsonisation, by phagocytic cells. Unfortunately, the phenomenon of antigenic variation, another method of immune evasion, prevents these infections being completely eliminated and typically allows the disease to run a characteristic course of continuous remissions and exacerbations of parasitaemia. It is also likely that the generalised immunosuppression induced by this disease may, sooner or later, limit the responsiveness of the host.

It is also relevant to note that some of the important lesions of trypanosomosis, such as anaemia, myocarditis and lesions of skeletal muscle, are thought to be attributable to the deposition of trypanosome antigen or immune complexes on these cells and leading to their subsequent destruction by macrophages or lymphocytes, a possible debit effect of the immune response.

Acquired immunity to babesiosis also appears to be mediated by antibody, perhaps acting as an opsonin, and facilitating the uptake of infected red cells by splenic macrophages. Antibody is also transferred in the colostrum of the mother to the newborn animal and confers a period of protection against infection.

Finally, in trichomonosis, antibody, presumably produced by plasma cells in the lamina propria of the uterus and vagina, is present in the mucus secreted by these organs and to a lesser extent in the plasma. This, *in vitro*, kills or agglutinates the trichomonads and is probably the major factor responsible for the self-limiting infections that typically occur in cows.

Of those protozoal infections against which immunity is primarily cell-mediated, leishmaniosis is of particular interest in that the amastigotes invade and proliferate in macrophages whose function, paradoxically, is the phagocytosis and destruction of foreign organisms. How they survive in macrophages is unknown, although it has been suggested that they may release substances which inhibit the enzyme activity of lysosomes or that the amastigote surface coat is refractory to lysosomal enzymes. The immunity that develops seems to be cell-mediated, perhaps by cytotoxic T cells destroying infected macrophages or by the soluble products of sensitised T cells 'activating' macrophages to a point where they are able to destroy their intracellular parasites. Unfortunately, in many cases the efficacy of the immune response and the consequent recovery

is delayed or prevented by a variable degree of immunosuppression of uncertain aetiology.

As noted in the preceding paragraphs, sometimes both humoral and cell-mediated reactions are involved in immunity, and this seems to be the situation with coccidiosis, theileriosis and toxoplasmosis.

In coccidiosis, the protective antigens are associated with the developing asexual stages and the expression of immunity is dependent on T-cell activity. It is thought that these function in two ways: first, as helper cells for the production of neutralising antibody against the extracellular sporozoites and merozoites and, secondly, in a cell-mediated fashion by releasing substances such as lymphokines which inhibit the multiplication of the intracellular stages. The net effect of these two immunological responses is manifested by a reduction in clinical signs, and a decrease in oocyst production.

As described earlier, the proliferative stages of theilerial infections are the merogonous stages, which develop in lymphoblasts and divide synchronously with these cells to produce two infected daughter cells. During the course of infection, and provided it is not rapidly fatal, cell-mediated responses are stimulated in the form of cytotoxic T cells that target the infected lymphoblasts by recognising two antigens on the host surface. One of these is derived from the *Theileria* parasite and the other is a histocompatibility antigen of the host cell. The role of antibodies in protection is less clear, although it has been recently demonstrated, using an *in vitro* test, that an antibody against the sporozoites inoculated by the tick may be highly effective in protection.

In toxoplasmosis also, both humoral and cell-mediated components appear to be involved in the immune response. However, the relative importance of their roles remains to be ascertained, although it is generally believed that antibody formation by the host leads to cessation in the production of tachyzoites and to the development of the latent bradyzoite cyst. It is also believed that recrudescence of tachyzoite activity may occur if the host becomes immunosuppressed as a consequence of therapy or some other disease.

ACQUIRED IMMUNITY TO ARTHROPOD INFECTIONS

It is known that animals exposed to repeated attacks by some insects gradually develop a degree of acquired immunity. For example, at least in humans, over a period of time the skin reactions to the bites of *Culicoides* and mosquitoes usually decrease in severity. Likewise, after several repeated infestations by blowfly larvae, sheep have been known to develop a degree of resistance to further attack, although this response is short-lived.

A similar sequence of events has been observed with many tick and mite infestations. The immune reaction to ticks, dependent on humoral and cell-mediated components to the oral secretions of the ticks, prevents proper engorgement of the parasites and has serious consequences on their subsequent fertility; dogs that have recovered from sarcoptic mange are usually immune to further infection. Some sheep infected with psoroptic mange (sheep scab) and which recover may demonstrate a degree of protective immunity to subsequent reinfection.

Although these immune responses may moderate the clinical significance of some ectoparasitic infections, the immune response to infestation may result in the unfortunate consequences which often occur when an animal becomes sensitised to arthropod antigens. Examples of this are flea dermatitis in dogs and cats, the pruritus and erythema associated with sarcoptic mange in the dog and pig and with psoroptic mange in sheep and cattle, and 'sweet itch' of horses due to skin hypersensitivity to *Culicoides* bites.

THE FUTURE OF PARASITE VACCINES

Early approaches that investigated the use of live radiation-attenuated parasite vaccines, which induced a high level of protection against larval challenge, resulted in commercially available vaccines for the bovine lungworm, *Dictyocaulus viviparus*, and also for *Eimeria* infections in poultry. Apart from *D. viviparus* there are no commercially produced vaccines for the control of helminth infections in ruminants. These encouraging results were experimentally applied to other economically important nematodes of ruminants (notably *T. colubriformis* and *H. contortus*); however, although partially effective in older animals, the vaccines provided an insufficient level of protection or the response was too variable in young animals under field conditions. An irradiated larval vaccine was developed against the dog hookworm *Ancylostoma caninum* that gave a high level of protection in the field but the vaccine was withdrawn from use over concerns with efficacy and storage viability. Early studies to develop a vaccine against liver fluke was hampered by the observation that these parasites do not appear to induce significant immunity in the natural ruminant host, even after repeated exposure to infection. The increased prevalence of parasites resistant to chemotherapy has led to further investment in vaccine development, particularly those based on recombinant parasite components, and considerable progress has been made over the past two decades in identifying candidate antigens for several important parasite species.

Experimental vaccines have already been developed, for example against *Taenia ovis* infection in sheep, *Babesia canis* in dogs, and *Babesia bovis* and *Rhipicephalus* (*Boophilus) microplus* in cattle. The commercial success of current experimental recombinant vaccines will depend not only on their efficacy under field challenge but also on factors such as effective low-cost delivery systems that will confer long-acting protection.

Two main approaches have been adopted for vaccine development: those based on 'natural antigens' on the surface of the parasite or excreted/secreted by the parasite which are recognised by the host during the course of infection, and those based on 'hidden' or 'covert' antigens. The latter strategy ignores the mechanisms of natural immunity and directs responses towards molecules located or secreted internally. There have been encouraging advances towards the aim of producing vaccines for the control of several parasitic diseases in the last decade. Recent research has identified protective antigen fractions which have then been enriched and characterised and the genes encoding the active components of many of these have been cloned.

HELMINTHS

Natural antigens

Natural antigens are those which are recognised by the host immune system following infection. The following information is not intended to represent a comprehensive list but to mainly

highlight some successful natural antigens derived from parasites of veterinary interest.

Surface and somatic antigens

A high level of protective immunity has been demonstrated in sheep vaccinated with fractions derived from the infective larval stage contained within the oncospheres of the cestodes *Taenia* and *Echinococcus* and identification of these protective proteins led initially to effective recombinant antigens for *T. ovis* and *E. granulosus*. Highly effective recombinant vaccines were subsequently available for vaccination of the intermediate hosts against infections with *Echinococcus multilocularis*, *Taenia solium* and *Taenia saginata*. However, the commercial development of vaccines for *T. ovis* and *T. saginata* was not entirely viable as these tapeworm infections are primarily a zoonotic problem in poor developing countries and are generally of minor economic significance. The economic return and sources of funding need further evaluation.

Excretory/secretory antigens

Parasitic helminths produce and secrete a range of proteins that have a variety of functions. Some enable the parasite to penetrate the host's tissue, while others are involved with the digestion of nutrients or in evasion of the host's immune response. It has been demonstrated that the immune response of the host can impair the function of these proteins and thus lead to worm expulsion and so they have received considerable attention as vaccine antigens. Highly protective effects have been reported against *H. contortus* using adult worm excretory/secretory (ES) products comprising two proteins of 15 and 24 kDa. However, recombinant versions of these proteins have not been protective. Research conducted over the last two decades has shown that the ES proteases, particularly cysteine proteases, associated with the intestinal surface of ovine nematodes such as *H. contortus* or bovine *Ostertagia ostertagi* are most effective vaccine components. Other proteases which can mediate vaccine-induced protective immunity in ruminants are the aspartic proteases, metalloproteases, dipeptidylpeptidases and aminopeptidases and thiol-binding fractions. The mode of action is mainly via induction of antibodies which block enzyme activity resulting in worm expulsion or impaired fecundity. However, expression of these promising native protease candidates as recombinant vaccines has been unsuccessful, probably as a result of incorrect folding and or glycosylation of the proteins.

Recently, protective antigens from *Teladorsagia circumcincta*, the major pathogen causing parasitic gastroenteritis in small ruminants in temperate regions, have been identified by studying IgA responses directed at proteins specific to post-infective larvae and also on the basis of their potential immunomodulatory role at the host–parasite interface. Recombinant versions of eight molecules identified by immunoproteomics, homology with vaccine candidates in other nematodes and/or with potential immunoregulatory activities were administered to sheep in a single vaccine formulation with an adjuvant and the animals subsequently subjected to a repeated challenge infection designed to mimic field conditions. The trial was performed on two occasions. In both trials, vaccinates had much lower mean faecal worm egg outputs and adult worm burdens were reduced by as much as 75% compared to the controls at postmortem. These levels of protection indicate that control of parasitic helminths via vaccination with recombinant subunit vaccine cocktails could indeed be an alternative option in the face of multidrug resistance.

Promising vaccines against *F. hepatica* have mostly been based on antigens such as cysteine proteases, leucine aminopeptidase and glutathione *S*-transferase. Cathepsin L cysteine proteases are secretion products from liver flukes that are released throughout the life cycle in the host and facilitate penetration of the parasites through the tissues of the host and are targets for vaccination. High levels of protection (reduced fluke burdens and fewer flukes developing to maturity) have been attained in sheep and cattle against *F. hepatica* using these natural cysteine protease enzymes in vaccine trials. Efficacy was further improved in cattle trials when cathepsin L2 was used in combination with fluke haemoglobin compared with either antigen alone. Recently, field trials of a recombinant cathepsin L1 (rFhCL1) against *F. hepatica* in cattle showed a 48% reduction in fluke burden compared with non-vaccinated controls. Cathepsin B proteases of *Fasciola*, which are predominantly released in the juvenile stage of the life cycle, have also been shown to be promising vaccine targets. A further potential vaccine candidate for fluke are the fatty acid binding proteins (FABPs), which are thought to play an important role in the uptake of fatty acids from the bloodstream of the mammalian host. FABPs from *F. hepatica* exhibit cross-protection and cross-reaction against *Schistosoma mansoni*. A recombinant version of a FABP (Sm14) from *S. mansoni* reduced the number of liver flukes and limited the histopathological damage to the liver in vaccination trials in sheep against a challenge with *F. hepatica*.

Leucine aminopeptidase, a gut-associated protease isolated from detergent soluble-extract of adult flukes, was successfully used as a vaccine against *F. hepatica* in sheep. Given alone with adjuvant, or in combination with the adult stage-specific secreted cathepsin L proteases, it induced high levels of protection in recipient sheep, with vaccinated animals showing an 89% reduction in fluke burden compared with the controls. This success has been duplicated with a bacterially produced recombinant protein version of the vaccine.

Considerable progress has been achieved in the area of defining antigenic targets of potential hookworm vaccines. A recombinant haemoglobinase protein, aspartic protease (Ac-APR-1), from the hookworm *Ancylostoma caninum* induces protection in dogs via antibodies that neutralise enzymic activity and thus disrupt blood-feeding activity, resulting in reduced parasite burdens and blood loss. A recombinant glutathione *S*-transferase (Ac-GT-1) also shows efficacy as a vaccine candidate. A further recombinant product, *Ancylostoma* secreted protein (Ac-ASP-2), gave good protection in dogs. The mechanism of protection appears to be directed against the larval stages of the hookworm. The future approach may be to incorporate several potential vaccine antigens, including the promising candidates discussed here, in order to target both the larval and adults stages of hookworms.

Hidden antigens

This approach primarily uses gut membrane proteins from parasites and these are not normally exposed to the host's natural immune system. Injection of these proteins into a host induces high titres of circulating antibody. When a haematophagous parasite ingests blood these antibodies bind to the surface of the parasite's gut and impair the digestion/absorption of nutrients and the weakened parasite is expelled from the host. This gut membrane

approach formed the basis of the recombinant vaccine against *Rhipicephalus* (*Boophilus*) *microplus*, the Australian cattle tick (see details in the section Ectoparasites). Early experimental studies with *H. contortus* in sheep, using natural fractions of these gut proteins, have shown that the reduction in the number of eggs passed in faeces can be greater than 80% and worm burdens can be lowered by more than 50% in vaccinated animals when compared to unvaccinated controls. Fractionation of these proteins from adult *H. contortus* showed that two main components are involved: H11 (also known as aminopeptidase N), which contains microsomal aminopeptidases, and H-gal-GP, a gut membrane-associated protein complex containing metalloproteases and aspartyl proteases. Numerous trials conducted over the last two decades with penned sheep have confirmed the efficacy of this approach using native gut membrane proteins. There has been partial success in applying this 'hidden' antigen approach to non-haematophagous parasites such as *Ostertagia ostertagi* and *Teladorsagia circumcincta*. Despite these encouraging results using natural proteins and the characterisation of the protective antigens, the testing of recombinant versions of these gut membrane proteins have been unsuccessful in vaccine trials, indicating that conformational epitopes are likely to be important in conferring protection. The situation is similar with liver fluke vaccine trials. A natural gut membrane fraction enriched for glutathione *S*-transferases has been shown to lower fluke egg output in faeces and also reduce fluke burdens in both sheep and cattle, although with variable efficiency. However, attempts to vaccinate animals with recombinant versions of these proteins have been unsuccessful. An important advantage of using 'covert' or hidden antigens in vaccine strategies is that they should be effective in those infections where natural immunity is poorly developed or is ineffective. A possible disadvantage is that immunity is not boosted by infection. However, it has been shown in *H. contortus* infection in lambs that vaccination with hidden gut membrane antigens, which are predominantly proteases and which are not normally recognised by the host during infection, will provide protection and by the time this wanes sufficient natural immunity will have been acquired. Although considerable progress has been made towards the experimental production of some monovalent vaccines, it is likely to be several years before commercially produced recombinant vaccines are available.

In the light of the problems encountered in expressing the natural proteins in a recombinant form that is immunologically active and protective, attention has focused back on the possibility of using the natural proteins as vaccines. One of the perceived problems with this approach was that large amounts of fresh parasite material were required to produce sufficient quantities of the active fractions. However, once it had been established that the dose of natural antigen required to produce a significant level of protection was actually very small (as low as 5 μg), it became feasible, and a commercial possibility, to extract the relevant integral gut membrane proteins from adult worms collected from animals given booster infections. One example was a field trial where weaner sheep were grazed on pastures contaminated with *H. contortus* and were then vaccinated on three occasions at 3-week intervals with the native gut membrane glycoproteins H11 and H-gal-GP in combination. The vaccinated animals showed a significant reduction in their faecal egg counts and also in the severity of anaemia in comparison to that observed in the unvaccinated controls. An experimental vaccine, comprising native integral gut membrane proteins from *H. contortus*, has been shown to confer significant cross-protection in calves against a challenge infection with *H. placei*. Recently, the technique for the rapid mass recovery of worms from booster-infected sheep has been markedly improved and this approach has formed the basis for a commercial vaccine, which uses small amounts of native *H. contortus* proteins, purified from the lining of worm intestines. Barbervax was registered for use in Australia in 2014. This is the first vaccine in the world for a nematode parasite for sheep and it presents a new approach for control of haemonchosis. Currently, trials are being extended into other countries to confirm the commercial potential of this *Haemonchus* vaccine.

It is important to consider that this vaccine strategy will not induce a sterile protection but will lower worm burdens and the faecal egg output sufficiently to be a very useful means of reducing and maintaining a low level of pasture contamination. Mathematical modelling of vaccines that use the hidden antigen approach predict that a level of protection of around 80% efficacy in 80% of the flock or herd would give a higher level of control than that achieved through an anthelmintic approach.

In conclusion to this whole area of vaccination against helminth parasites it is considered that the apparent inefficiency of many vaccines, experimentally tested against helminth infections, may be partly due to the focus on only one or two antigens and this invariably produces a fairly narrow antibody response. The most efficient vaccines will undoubtedly need to induce broad Th2-type responses that include strong humoral and cell-mediated constituents.

PROTOZOA

A number of vaccines have been on the market for several years or decades to ameliorate the impact of protozoal diseases to the livestock and poultry sectors. The majority of these products are based on live organisms, although more recently there has been increased focus on the development of killed and subunit vaccines. Live organism vaccines are more likely to induce T cell-mediated immune responses and induce a more potent and longer-lived protective immunity against a challenge infection. Their disadvantage is that many have a fairly short shelf-life and there can be safety issues using live vaccines. They also often require a cold facility for storage and administration.

Live vaccines

Several approaches have been used to produce a protective immune response, including using the native protozoa, attenuated strains, truncated life cycles and chemically abbreviated infections.

Unattenuated vaccines which involve complete life-cycle infections

An example is the control of coccidiosis in poultry using small doses of *Eimeria* species that are sufficiently low as to cause minimal clinical symptoms yet still able to induce a significant level of protection. The first commercial vaccine (CocciVac) comprised wild-type strains of *Eimeria tenella* oocysts, and over several decades other species of *Eimeria* were introduced to broaden the effectiveness of the vaccine. One disadvantage of this approach was the differences in the levels of pathogenicity induced in inoculated hosts by the live parasites.

Vaccines using virulence-attenuated strains

The safety of the unattenuated approach has been improved through the inclusion of oocysts from natural 'precocious' *Eimeria* strains that exhibit a smaller number of merogenic cycles and hence offer a lower risk of inducing disease. 'Precocious' parasites complete their life cycle more rapidly and exhibit reduced virulence with high immunogenicity and have been developed for the seven species of *Eimeria* in poultry (e.g. Paracox to protect breeding and laying hens and more recently Eimeriavax 4m for egg-laying hens and broilers). These vaccines should be administered simultaneously to all individuals and comprise the major type of live attenuated vaccines in use for the control of coccidiosis in poultry.

A further example is the inoculation of cattle with an attenuated strain of *Theileria annulata* for the control of tropical theileriosis. Repeated *in vitro* passaging of the intracellular macroschizont stage of *T. annulata* in tissue culture cell lines resulted in a live attenuated vaccine that has been used in many countries (China, India, North Africa and the Middle East). Continuous passage attenuates the schizont-infected cells so that their pathogenicity is reduced but their infectivity is retained. Cell-line immunisation has not been as successful with *Theileria parva* due to histo-incompatibility between the cell line and the recipient animal and the fact that *T. parva* and *T. annulata* infect distinct bovine leucocyte populations. Similarly, passage of *Babesia bigemina* and *Babesia bovis* piroplasms in splenectomised calves resulted in an attenuated strain which is used as a frozen vaccine to reduce the pathogenicity of infection in inoculated cattle.

Vaccines using drug-abbreviated infections

This approach (initiated in the 1970s) has been adopted to limit the losses in cattle arising from East Coast fever in East and Central Africa. Cattle are vaccinated with a defined dose of a cryopreserved wild-type *Theileria parva* sporozoite stabilate and concurrently given a long-acting tetracycline treatment. The antibiotic slows the rate of schizogony and allows the immune response time to develop. The immunity induced is very strain-specific. The main disadvantage is that this approach is expensive for resource-poor farmers. However, a recent collaboration between a small private company and Maasai cattle herders in Tanzania has seen the successful vaccination and treatment of around 500,000 cattle against East Coast fever that lowered the mortality in the herds by up to 95% in some cases. Success was partly due to improved quality control of the vaccine stabilate and the production processes.

Vaccines using parasites which produce a truncated life cycle

This approach is particularly relevant to those parasites that produce cysts within the intermediate hosts. One example is *Toxoplasma gondii* in sheep and goats. The live vaccine comprises tachyzoites attenuated by repeated passage in mice. This attenuated strain (S48) does not form tissue cysts (which contain the bradyzoites) in the intermediate host and is not able to establish a persistent infection. It has also lost the potential to form oocysts in the definitive host, the cat. Thus the S48 strain is incomplete and undergoes limited multiplication within the intermediate host but is still able to stimulate protective immune responses. This live vaccine confers long-acting immunity (effective protection even at 18 months after inoculation in the absence of further *T. gondii* challenge) against abortion induced by *Toxoplasma* infection in breeding ewes and is available commercially as Toxovax. This is currently the only commercial vaccine available to help prevent toxoplasmosis.

Killed and subunit vaccines

These vaccines are generally less effective than those using live organisms as they rely mainly on the induction of neutralising antibodies but they can reduce transmission of disease and often can also lower the pathogenic effects of natural infection. The major challenge is to be able to identify and then present relevant parasite antigens to the host's immune system in such a way that they can be processed to induce protective immune responses.

Vaccines using inactivated parasites

Bovilis Neoguard® was a commercial vaccine developed to reduce abortion in pregnant cattle resulting from infection with *Neospora caninum* and was available in the USA, New Zealand and some other countries. This vaccine comprised inactivated whole tachyzoites and inoculation aimed to reduce the transmission of the parasite to the developing fetus. The vaccine has recently been withdrawn from the market by the manufacturer. Although progress has been achieved towards reducing the impact of bovine neosporosis in cattle, a fully effective vaccine needs to prevent disease on primary exposure, reduce vertical transmission and abrogate the clinical signs of infection.

A vaccine is available commercially (GiardiaVax®) to reduce the clinical signs and pathogenesis of *Giardia intestinalis* (syn. *G. duodenalis*) infection in dogs and cats. It also lowers the faecal output of oocysts in young vaccinated animals and the period of diarrhoea is of reduced duration. This vaccine is based on disrupting axenically cultured trophozoites from an ovine isolate.

A vaccine has been developed to reduce the pathogenesis of equine protozoal myeloencephalitis in horses caused primarily by infection with *Sarcocystis neurona*. The vaccine is based on chemically inactivated cultured merozoites and has shown to be promising in ameliorating the neurological effects of infection.

Subunit vaccines

A subunit transmission-blocking vaccine which targets the sexual macrogametocyte stages and thus reduces oocyst output has been developed for the control of coccidial infections in poultry. The vaccine (CoxAbic®) comprises affinity-purified antigens from the gametocyte stages of *Eimeria maxima*. It provides a good level of protection across three species of *Eimeria* (*E. maxima*, *E. tenella*, *E. acervulina*) and is administered to laying hens where protection is passed, via the yolk, to their broiler offspring. Unfortunately, it is an expensive vaccine to manufacture and work is ongoing to test whether recombinant forms of the gametocyte proteins are as effective at producing antigenicity as the natural proteins.

A subunit vaccine is available to reduce the severity of clinical disease resulting from canine babesiosis. It contains soluble surface proteins expressed by cultures of *Babesia canis canis*. The strain-specific immunity has been broadened in a similar vaccine by the inclusion of *Babesia canis rossi* antigens.

A subunit vaccine has been developed in South America for the control of visceral leishmaniosis in dogs caused by *Leishmania donovani infantum*. This vaccine is based on a surface fucose-mannose- ligand antigen complex. CaniLeish® is a parasite lyophilisate vaccine, commercially available in Europe, containing ES proteins of *Leishmania infantum* for the immunisation of dogs.

ECTOPARASITES

Using the hidden antigen approach, the first recombinant vaccine was developed in 1994 against the cattle tick *Rhipicephalus* (*Boophilus*) *microplus*, and introduced commercially in Australia. The vaccine was later available in Cuba and in parts of South America. The active antigen is a membrane-bound protein (BM86) from the gut of the tick. In controlled field trials in Cuba, Brazil, Argentina and Mexico, this vaccine showed 55–100% efficacy in the control of *B. microplus* infestations in grazing cattle 12–36 weeks after the first vaccination. However, in order to maintain high levels of circulating antibody cattle have to be inoculated repeatedly.

CHAPTER 8

Parasites of cattle

ENDOPARASITES

Parasites of the digestive system

OESOPHAGUS

Gongylonema pulchrum

Synonym: *G. scutatum*

Common name: Gullet worm

Predilection site: Oesophagus, rumen

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Description, gross: A long, slender, whitish worm, the males being about 5.0 cm and the females up to about 14.0 cm in length.

Description, microscopic: Worms are easily distinguished microscopically by the presence of longitudinal rows of cuticular bosses in the anterior region of the body. Asymmetrical cervical alae are prominent. The egg is thick-shelled and possesses two opercula. It measures 50–70 by 25–37 μm and contains an L_1 when passed in faeces.

Final hosts: Sheep, goat, cattle, pig, buffalo, horse, donkey, deer, camel, human

Intermediate host: Coprophagous beetles, cockroaches

Geographical distribution: Probably worldwide

For more details see Chapter 9 (Sheep and goats).

Hypoderma bovis

For more details see Parasites of the integument.

Hypoderma lineatum

For more details see Parasites of the integument.

RUMEN AND RETICULUM

Gongylonema verrucosum

Common name: Rumen gullet worm

Predilection site: Rumen, reticulum, omasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Description, gross: Long slender worms, reddish when fresh. The males are about 3.5 cm and the females 7.0–9.5 cm in length.

Description, microscopic: The adult parasites have a festooned cervical ala and cuticular bosses only on the left side of the body. The males' spicules are unequal in length, with the left spicule longer than the right.

Final hosts: Cattle, sheep, goat, deer, zebu

Intermediate hosts: Coprophagous beetles and cockroaches

Geographical distribution: India, South Africa, USA

Pathogenesis: Usually regarded as non-pathogenic.

Clinical signs: Infection is usually asymptomatic.

Diagnosis: Usually an incidental finding on postmortem.

Pathology: Adult worms bury in the epithelium of the forestomachs producing white or red, blood-filled zig-zag tracts in the mucosa.

Epidemiology: Infection is very much dependent on the presence and abundance of the intermediate hosts, principally coprophagous beetles of the genera *Aphodius*, *Onthophagus*, *Blaps* and *Caccobius*.

Treatment: Not reported

Control: Control is neither practical nor necessary.

Paramphistomum and other rumen fluke

Rumen fluke, as their name implies, are mainly parasitic in the forestomachs of ruminants. Their shape is not typical of the trema-

Veterinary Parasitology, Fourth Edition. M.A. Taylor, R.L. Coop and R.L. Wall.

todes, being conical and thick and fleshy rather than flat. All require a water snail as an intermediate host. There are several genera: *Paramphistomum*, *Cotylophoron*, *Bothriophoron*, *Orthocoelium* and *Giganocotyle*, of which *Paramphistomum* is the most common and widespread in ruminants.

The taxonomy of the paramphistomes is complex and unresolved and many of the species described may be synonymous, being differentiated mainly on size and shape of the suckers.

Pathogenesis: The adult parasites in the forestomachs are generally well tolerated, even when many thousands are present and feeding on the wall of the rumen or reticulum (Fig. 8.1). Any pathogenic effect is mainly associated with the intestinal phase of the infection, although the presence of adults in the rumen has been reported to cause effects on rumination leading to weight loss and ill-thrift.

Clinical signs: In heavy duodenal infections, the most obvious sign is diarrhoea accompanied by anorexia and intense thirst. Sometimes in cattle there is rectal haemorrhage following a period of prolonged straining. Mortality in acute outbreaks can be as high as 90%.

Diagnosis: This is based on the clinical signs usually involving young animals in the herd and a history of grazing around snail habitats during a period of dry weather. Faecal examination is of limited value since the acute disease occurs during the prepatent period. However, large numbers of paramphistome eggs can sometimes be present in faeces during acute disease as the intestinal phase may also be accompanied by large numbers of adult flukes in the forestomach. Confirmation can be obtained by postmortem examination and recovery of the small pink-coloured immature flukes from the duodenal musosa and ileal contents.

Pathology: The immature flukes are embedded in the mucosa of the upper ileum and duodenum and are plug feeders, and this can result in severe erosion of the duodenal mucosa. In heavy infections these cause enteritis characterised by oedema, haemorrhage, ulceration and associated anaemia and hypoproteinaemia. At necropsy, the young flukes can be seen as clusters of brownish-pink parasites attached to the duodenal mucosa and occasionally also in the jejunum and abomasum.

Epidemiology: Paramphistomosis often depends for its continuous endemicity on permanent water masses, such as lakes and ponds, from which snails are dispersed into previously dry areas by flooding during heavy rains. Paramphistome eggs deposited by animals grazing these areas hatch and infect snails. Subsequent production of cercariae often coincides with receding water levels, making them accessible to grazing ruminants. In other areas, the situation is complicated by the ability of the snails to aestivate on dry pastures and become reactivated on the return of rainfall. A good immunity develops in cattle, and outbreaks are usually confined to youngstock. However, adults continue to harbour low burdens of adult parasites and are important reservoirs of infection for snails. In contrast, sheep and goats are relatively susceptible throughout their lives.

Treatment: Resorantel and oxyclozanide are considered the anthelmintics of choice against adult rumen flukes in both cattle and sheep. Recents studies have shown that closantel is also effective in cattle at 10 mg/kg.

Control: As in *Fasciola gigantica*, the best control is achieved by providing a piped water supply to troughs and preventing access of animals to natural water. Even then snails may gain access to watering troughs and regular application of a molluscicide at source or manual removal of snails may be necessary.

Paramphistomum cervi

Synonym: *Paramphistomum explanatum*

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Description, gross: The adults are small, conical (pear-shaped), maggot-like flukes about 1.0 cm long and light red in colour when fresh (Fig. 8.2).

Description, microscopic: One sucker is visible at the tip of the cone and the other well-developed sucker is at the base. The tegument has no spines. The larval stages are less than 5.0 mm, fresh specimens having a pink colour. The egg resembles that of *Fasciola hepatica*, being large (about 115–175 by 75–100 μm) and operculate,

Figure 8.1 Adult paramphistomes in the rumen.

Figure 8.2 Adult flukes of *Paramphistomum*.

but is transparent or slightly greenish rather than yellowish-brown and slightly smaller than eggs of *F. hepatica* (see Fig. 9.1). In the early stages of segmentation the egg contains four to eight blastomeres surrounded by yolk cells.

Final hosts: Cattle, sheep, goat, deer, buffalo, antelope

Intermediate hosts: Water snails, principally *Planorbis* and *Bulinus*

Geographical distribution: Worldwide. They are of little veterinary significance in Europe and America, but are occasionally the cause of disease in the tropics and subtropics.

Paramphistomum microbothrium

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Final hosts: Cattle, sheep, goat, deer, buffalo, antelope

Intermediate hosts: Freshwater snails (*Fossaria* spp., *Bulinus* spp.)

Geographical distribution: Africa

Paramphistomum ichikawa

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Final hosts: Sheep, cattle

Intermediate hosts: Planorbid snails (*Gyraulus*, *Helicorbis*, *Segnetilia*)

Geographical distribution: Southeast Asia

Paraphistomum streptocoelium

Synonyms: *Ceylonocotyle streptocoelium, Orthocoelium streptocoelium*

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Final hosts: Cattle, sheep, goat and wild ruminants

Intermediate hosts: Freshwater snails (*Glyptanisus* spp.)

Geographical distribution: Africa

Calicophoron daubneyi

Synonyms: *Paramphistomum daubnei, Paramphistomum daubneyi*

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Description: Resembles *P. cervi*, but there is a genital sucker surrounding the genital pore.

Final hosts: Cattle, goat

Intermediate hosts: Freshwater snails (*Omphiscola* spp.), Galba truncatula

Geographical distribution: Europe (mainly Mediterranean areas but also recorded in the UK and Ireland), parts of Asia

Cotylophoron cotylophorum

Synonym: *Paramphistomum cotylophorum*

Common name: Rumen fluke

Predilection site: Rumen, reticulum

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Description, microscopic: The fluke is very similar to *Paramphistomum cervi* but the genital pore is surrounded by a genital sucker. The egg measures 125–135 by 60–68 μm.

Final hosts: Cattle, sheep and many other ruminants

Intermediate hosts: Freshwater snails (*Bulinus* spp.)

Geographical distribution: Indian subcontinent, Australia and many other countries except northern temperate regions

Calicophoron calicophorum

Synonym: *Paramphistomum calicophorum*

Common name: Rumen fluke

Predilection site: Rumen, reticulum

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Description, microscopic: The body measures 7.5–14.8 by 3–4 mm, and the genital pore is post-bifurcal. Eggs are 110–150 by 60–90 μm.

Final hosts: Cattle, sheep and many other ruminants

Intermediate hosts: Freshwater snails

Geographical distribution: Indian subcontinent, Southeast Asia, Australasia and South Africa

Carmyerius spatiosus

Common name: Rumen fluke

Synonym: *Gastrothylax spatiosus*

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrothylacidae

Description, gross: The flukes measure 8.5–12 by 2.5–3.0 mm.

Description, microscopic: The posterior sucker is quite small and is spherical. The intestinal caeca extend down into the last quarter of the body. The ventral pouch is either circular or slightly triangular with blunt angles and the terminal genitalium lies within the pouch. The testes lie horizontally, one on each side of the median line, which differs from the position in *Fischoederius*. Eggs measure 115–125 by 60–65 μm.

Final hosts: Cattle, zebu, antelope

Intermediate hosts: Water snails

Geographical distribution: Southeast Asia, India, Africa and America

Carmyerius gregarius

Common name: Rumen fluke

Predilection site: Rumen

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrothylacidae

Description, gross: The flukes are 7–10 mm in length.

Description, microscopic: The intestinal caeca extend only a short distance below the middle of the body.

Final hosts: Cattle, buffalo

Intermediate hosts: Water snails

Geographical distribution: India, Africa

Gastrothylax crumenifer

Common name: Rumen fluke

Predilection site: Rumen, reticulum

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrothylacidae

Description, gross: This is an elongated fluke, being circular in transverse section and reddish in colour when fresh. The body measures 10–16 by 5–8 mm.

Description, microscopic: These flukes differ in having an extremely large ventral pouch that opens anteriorly and which covers the ventral surface of the fluke as far as the large ventral sucker. The ventral pouch is normally triangular in cross-section with a dorsally directed apex. The terminal genitalium opens into the ventral pouch about halfway between the intestinal bifurcation and the pharynx. The terminal oval sucker is small. Eggs are 115–135 by 66–70 μm.

Final hosts: Cattle, buffalo, zebu, sheep and many other ruminants

Geographical distribution: Indian subcontinent, China, Middle East, Africa and parts of Asiatic Russia and Europe

Pathogenesis: The fluke mainly causes anaemia.

Fischoederius elongatus

Predilection site: Rumen, duodenum or anterior small intestine

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrothylacidae

Description, gross: The flukes are reddish when fresh. The body measures 10–20 by 3–5 mm.

Description, microscopic: The terminal genitalium is within the ventral pouch. The uterus is situated along the midline. The testes are lobed and one is sited dorsally to the other. Eggs measure 125–150 by 65–75 μm.

Final hosts: Cattle, buffalo, zebu, sheep and many other ruminants. It can accidentally infect humans.

Geographical distribution: Asia

Pathogenesis: Flukes in the rumen usually cause only mild congestion but flukes attached to the duodenum can result in thickening of the mucosa.

Fischoederius cobboldi

Predilection site: Rumen, duodenum or anterior small intestine

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrothylacidae

Description, gross: The flukes are reddish in colour when fresh. The body measures 8–10 mm in length.

Description, microscopic: Eggs measure 110–120 by 60–75 μm.

Final hosts: Cattle, buffalo, zebu, sheep and many other ruminants

Geographical distribution: Asia

Pathogenesis: Similar to *F. elongatus*

Monocercomonas ruminantium

Synonyms: *Trichomonas ruminantium, Tritrichomonas ruminantium*

Predilection site: Rumen

Phylum: Preaxostyla

Class: Tritrichomonadea

Family: Monocercomonadidae

Description: The trophozoite is subspherical, 3–8 by 3–7 μm, with a rounded anterior end. The axostyle is curved and may or may not extend beyond the body. Both a pelta and parabasal body are present. The cytostome and anterior nucleus are anterior. There are three anterior flagella and a trailing one (see Fig. 2.18).

Hosts: Cattle, sheep

Life cycle: The life cycle is simple with trophozoites dividing by binary fission. No sexual stages are known and there are no cysts.

Geographical distribution: Worldwide

Pathogenesis: Not considered pathogenic

Diagnosis: Identification of trophozoites based on morphological examination.

Epidemiology: Transmission presumably occurs by ingestion of trophozoites from faeces or rumen contents.

Treatment and control: Not required

Entamoeba bovis

Predilection site: Rumen

Phylum: Amoebozoa

Class: Archamoebae

Family: Entamoebidae

Description: Trophozoites are 5–20 μm in diameter. The smoothly granular cytoplasm is filled with vacuoles of various sizes. The nucleus is large with a large central endosome made up of compact granules, with a row of chromatin granules of varying sizes around its periphery. The cysts are 4–14 μm in diameter and contain a single nucleus when mature with irregular clumps of chromatin granules. A large glycogen granule may or may not be present.

Hosts: Cattle

Distribution: Worldwide

Pathogenicity: Non-pathogenic

Diagnosis: Identification of trophozoites, or cysts in large intestinal contents or faeces.

Treatment and control: Not required

ABOMASUM

Cattle can be parasitised by over 18 species of gastrointestinal nematodes, infection causing parasitic gastroenteritis. The most economically important gastrointestinal nematode in cattle is *Ostertagia ostertagi* and while the diagnosis, epidemiology, treatment and control are described in detail for this parasite, details are similar for other gastrointestinal nematodes. Although treatment for gastrointestinal nematodes is mainly targeted at susceptible first-year grazing animals, more recently there has been a trend to also treat cattle in their second grazing season and in some circumstances even adult animals, particularly where other helminths such as liver fluke and lungworm are present.

Ostertagia ostertagi

Synonyms: *Ostertagia lyrata, Skrjabinagia lyrata*

Common name: Brown stomach worm

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Descripton, gross: Adults are small, slender, reddish-brown worms with a short buccal cavity which is not very pronounced. Males measure 6–8 mm and females 8–11 mm in length (Fig. 8.3).

Description, microscopic: The cuticle in the anterior region is striated transversely whereas the rest of the body is unstriated and bears around 30 longitudinal ridges. A pair of very tiny cervical papillae is present in both sexes. The spicules are divided in the posterior region, where two thin lateral branches arise from the main stem (see Table 1.4a). The bursa is small and the accessory bursal membrane is supported by two divergent rays (Fig. 8.4; see also Fig. 1.18). In the female, the vulva is sited about 1.5 mm from the posterior and is covered with a flap. Female worms have double ovejectors. The tail tapers gradually and ends in a slender rounded tip which often bears several refractile cuticular annulations. The medium-sized eggs measure about 75–90 by 38–45 μm. They are elliptical and symmetrical with slightly barrel-shaped walls and the thin chitinous shell has a smooth surface. The egg is filled with many blastomeres which are hard to distinguish.

In the morph species, *Ostertagia lyrata*, the spicules are stout and divided into three branches posteriorly. The main branch is solid and ends in a shoe-like expansion. One lateral branch is thick and massive, terminating in a hat-like expansion; the other is is small and pointed. The gubernaculum is spindle-shaped.

Hosts: Cattle, deer and very occasionally goats

Geographical distribution: Worldwide. *Ostertagia* is especially important in temperate climates and in subtropical regions with winter rainfall.

Pathogenesis: Large populations of *O. ostertagi* can induce extensive pathological and biochemical changes and these are maximal

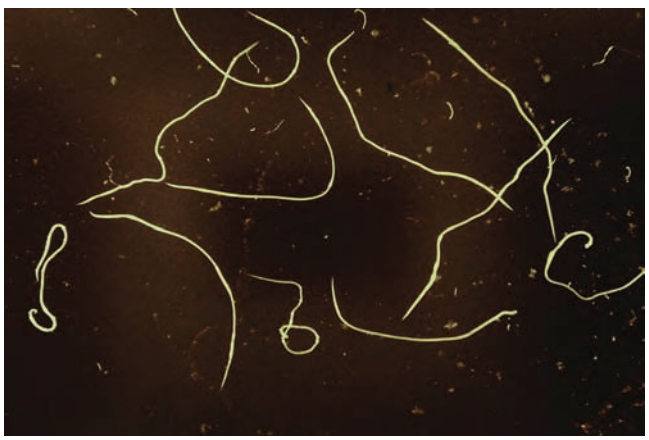

Figure 8.3 Adult *Ostertagia ostertagi.*

Figure 8.4 Bursa of adult male *Ostertagia ostertagi*.

when the parasites are emerging from the gastric glands (about 18 days after infection) but may be delayed for several months when arrested larval development occurs.

In heavy infections of 40,000 or more adult worms, the principal effects of these changes are as follows.

1. A reduction in the acidity of the abomasal fluid, the pH increasing from 2.0 to 7.0. This results in a failure to activate pepsinogen to pepsin. There is also a loss of bacteriostatic effect in the abomasum.
2. There is enhanced permeability of the abomasal epithelium to macromolecules.

The results of these changes are a leakage of pepsinogen into the circulation, leading to elevated plasma concentrations, and the loss of plasma proteins into the gut lumen, eventually leading to hypoalbuminaemia. In addition, in response to the presence of the adult parasites, the zymogen cells secrete increased amounts of pepsin directly into the circulation.

Although reduced feed consumption and diarrhoea affect liveweight gain, they do not wholly account for the loss in production. Current evidence suggests that this is primarily because of substantial leakage of endogenous protein into the gastrointestinal tract. Despite some reabsorption, this leads to a disturbance in postabsorptive nitrogen and energy metabolism due to the increased demands for the synthesis of vital proteins, such as albumin and the immunoglobulins, which occurs at the expense of muscle protein and fat deposition.

Clinical signs: Bovine ostertagiosis occurs in two clinical forms. In temperate climates with cold winters the seasonal occurrence of these is as follows.

- Type I disease is usually seen in calves grazed intensively during their first grazing season, as the result of larvae ingested 3–4 weeks previously; in the northern hemisphere this normally occurs from mid-July onwards. In type I disease, the morbidity is usually high, often exceeding 75%, but mortality is rare provided treatment is instituted early.
- Type II disease occurs in yearlings, usually in late winter or spring following their first grazing season and results from the maturation of larvae ingested during the previous autumn and which subsequently become arrested in their development at the EL_4 stage. Hypoalbuminaemia is more marked, often leading to submandibular oedema. In type II the prevalence of clinical disease is comparatively low and often only a proportion of animals in the group are affected; mortality in such animals can be high unless early treatment with an anthelmintic effective against both arrested and developing larval stages is instituted.

The main clinical sign in both type I and type II disease is a profuse watery diarrhoea; in type I disease, where calves are at grass, this is usually persistent and has a characteristic bright green colour. In contrast, in the majority of animals with type II disease, the diarrhoea is often intermittent and anorexia and thirst are usually present. In both forms of the disease, the loss of body weight is considerable during the clinical phase and may reach 20% in 7–10 days.

Diagnosis: In young animals this is based on the following.

1. The clinical signs of inappetence, weight loss and diarrhoea.
2. The season, for example in Europe type I occurs from July until September and type II from March to May.
3. The grazing history. In type I disease, the calves have usually been set-stocked in one area for several months; in contrast, type II disease often has a typical history of calves being grazed on a field from spring to midsummer, then moved and brought back to the original field in the autumn. Affected farms usually also have a history of ostertagiosis in previous years.
4. Faecal egg counts. In type I disease these are usually more than 1000 eggs per gram (epg) and are a useful aid to diagnosis; in type II the count is highly variable, may even be negative and is of limited value.
5. Plasma pepsinogen levels. In clinically affected animals up to 2 years old these are usually in excess of 3.0 iu tyrosine (normal levels are 1.0 iu in non-parasitised calves). The test is less reliable in older cattle where high values are not necessarily correlated with large adult worm burdens but, instead, may reflect plasma leakage from a hypersensitive mucosa under heavy larval challenge.
6. Postmortem examination. Adult worms can be seen on close inspection of the abomasal surface. Adult worm burdens are typically in excess of 40,000, although lower numbers are often found in animals which have been diarrhoeic for several days prior to necropsy. Species differentiation is based on the structure of the male spicules (see Table 1.4).

In older animals, laboratory diagnosis is more difficult since faecal egg counts and plasma pepsinogen levels are less reliable.

A serum *Ostertagia* enzyme-linked immunosorbent assay (ELISA) has been developed that can detect worm infections in adult milking cattle and this may have a potential effect on milk production. However, the assay can suffer from the disadvantage of cross-reactions with other helminths such as *Dictyocaulus viviparus* and *Fasciola hepatica* where these infections coexist. A milk ELISA has also been developed to monitor *Ostertagia* antibody levels in adult cattle from individual or from bulk-tank milk samples, with a good level of repeatability. However, milk antibody levels can be influenced by factors such as the age of the cow, stage of lactation and milk yield. Evaluation in the field is currently underway in some countries.

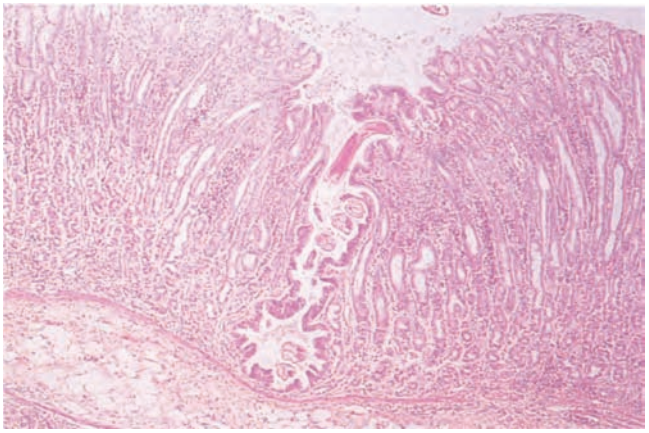

Figure 8.5 *Ostertagia ostertagi* emerging from a gastric gland.

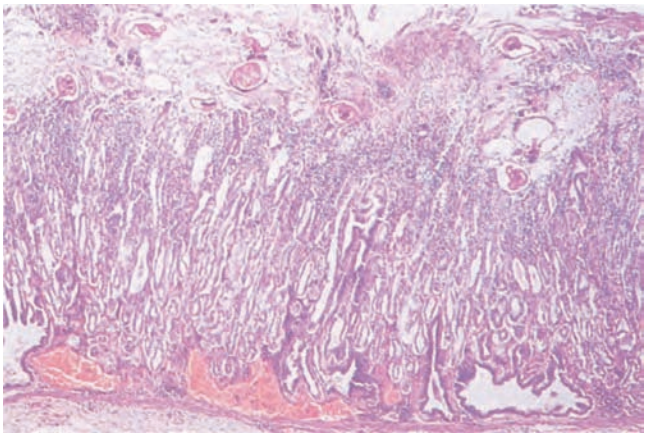

Figure 8.7 Necrosis of mucosa in severe ostertagiosis.

Figure 8.6 Abomasum showing the characteristic nodules produced by the development of *O. ostertagi* larvae in the gastric glands.

Pathology: The developing parasites cause a reduction in the functional gastric gland mass; in particular the parietal cells, which produce hydrochloric acid, are replaced by rapidly dividing, undifferentiated, non-acid-secreting cells. Initially, these cellular changes occur in the parasitised gland (Fig. 8.5), but as it becomes distended by the growing worm these changes spread to the surrounding non-parasitised glands, the end result being a thickened hyperplastic gastric mucosa.

Macroscopically, the lesion is a raised nodule with a visible central orifice; in heavy infections these nodules coalesce to produce an effect reminiscent of morocco leather (Fig. 8.6). The abomasal folds are often very oedematous and hyperaemic and sometimes necrosis and sloughing of the mucosal surface occurs (Fig. 8.7); the regional lymph nodes are enlarged and reactive.

Epidemiology of ostertagiosis in temperate countries of the northern hemisphere

Dairy herds

1 A considerable number of L_3 can survive the winter on pasture and in soil. Sometimes the numbers are sufficient to precipitate type I disease in calves 3–4 weeks after they are turned out to graze in the spring. However, this is unusual and the role of the surviving L_3 is rather to infect calves at a level which produces patent subclinical infection and ensures contamination of the pasture for the rest of the grazing season.
2 A high mortality of overwintered L_3 on the pasture occurs in spring and only negligible numbers can usually be detected by June. This mortality, combined with the dilution effect of the rapidly growing herbage, renders most pastures, not grazed in the spring, safe for grazing after midsummer. However, some L_3 may survive in the soil for at least another year and can subsequently migrate on to the herbage.
3 Eggs deposited in the spring develop slowly to L_3; this rate of development becomes more rapid towards midsummer as temperatures increase and, as a result, the majority of eggs deposited during April to June all reach the infective stage from around mid-July onwards. If sufficient numbers of these L_3 are ingested, the type I disease occurs any time from July until October. Development from egg to L_3 slows during the autumn.
4 As autumn progresses and temperatures decline, an increasing proportion (up to 80%) of the L_3 ingested become inhibited at the early fourth larval stage (EL_4). In late autumn, calves can therefore harbour many thousands of these EL_4 but few developing forms or adults. These infections are generally asymptomatic until maturation of the EL_4 takes place during winter and early spring when type II disease may materialise. Where maturation is not synchronous, clinical signs may not occur but the adult worm burdens which develop can play a significant epidemiological role by contributing to pasture contamination in the spring.

Two factors, one management and one climatic, appear to increase the prevalence of type II ostertagiosis.

1 The practice of grazing calves from May until late July on permanent pasture, then moving these to hay or silage aftermath before returning them to the original grazing in late autumn. Such pasture will still contain many L_3 and when ingested they will become arrested.
2 In dry summers the L_3 are retained within the crusted faecal pat and cannot migrate on to the pasture until sufficient rainfall occurs. If rainfall is delayed until late autumn, many larvae liberated on to pasture will become arrested following ingestion and so increase the chance of type II disease.

Although primarily a disease of young dairy cattle, ostertagiosis can nevertheless affect groups of older cattle in the herd, particularly if these have had little previous exposure to the parasite.

Acquired immunity is slow to develop and calves do not achieve a significant level of immunity until the end of their first grazing season. Housing over the winter allows the immunity to wane by the following spring and yearlings turned out at that time are partially susceptible to reinfection and so contaminate the pasture with small numbers of eggs. However, immunity is rapidly re-established and any clinical signs which occur are usually of a transient nature. By the second and third year of grazing, adult stock in endemic areas are usually highly immune to reinfection and of little significance in the epidemiology. However, around the periparturient period when immunity wanes, particularly in heifers, there are reports of clinical disease following calving. Burdens of adult *Ostertagia* spp. in dairy cows are usually low and routine treatment of herds at calving should not be required.

Beef herds Although the basic epidemiology in beef herds is similar to that in dairy herds, the influence of immune adult animals grazing alongside susceptible calves has to be considered. Thus, in beef herds where calving takes place in the spring, ostertagiosis is uncommon since egg production by immune adults is low, and the spring mortality of the overwintered L_3 occurs prior to the suckling calves ingesting significant quantities of grass. Consequently, only low numbers of L_3 become available on the pasture later in the year. However, where calving takes place in the autumn or winter, ostertagiosis can be a problem in calves during the following grazing season once they are weaned, the epidemiology then being similar to that for dairy calves.

Epidemiology of ostertagiosis in subtropical and temperate countries in the southern hemisphere

In countries with temperate climates, such as New Zealand, the seasonal pattern is similar to that reported for Europe with type I disease occurring in the summer and burdens of arrested larvae accumulating in the autumn. In those countries with subtropical climates and winter rainfall, such as parts of southern Australia, southwest Africa and some regions of Argentina, Chile and Brazil, the increase in L_3 population occurs during the winter and outbreaks of type I disease are seen towards the end of the winter period. Arrested larvae accumulate during the spring and where type II disease has been reported it has occurred in late summer or early autumn. A basically similar pattern of infection is seen in some southern parts of the USA with non-seasonal rainfall, such as Louisiana and Texas. There, larvae accumulate on pasture during winter and arrested development occurs in late winter and early spring with outbreaks of type II disease occurring in late summer or early autumn.

The environmental factors which produce arrested larvae in subtropical zones are not yet fully known.

Treatment: Type I disease responds well to treatment at the standard dosage rates with any of the modern benzimidazoles, the pro-benzimidazoles (febantel, netobimin and thiophanate), levamisole, or the avermectins/milbemycins. All these drugs are effective against developing larvae and adult stages. Following treatment, calves should be moved to pasture which has not been grazed by cattle in the same year.

For the successful treatment of type II disease it is necessary to use drugs which are effective against arrested (hypobiotic) larvae as well as developing larvae and adult stages. Only the modern benzimidazoles (such as albendazole, fenbendazole or oxfendazole) or the avermectins/milbemycins are effective in the treatment of type II disease when used at standard dosage levels, although the pro-benzimidazoles are also effective at higher dose rates.

The field where the outbreak originated may be grazed by sheep or rested until the following June.

In lactating dairy cattle, topical eprinomectin has the advantage that there is no milk withholding period.

Control: Traditionally, ostertagiosis has been prevented by routinely treating young cattle with anthelmintics over the period when pasture larval levels are increasing. However, it has the disadvantage that since the calves are under continuous larval challenge their performance may be impaired. With this system, effective anthelmintic treatment at housing is also necessary using a drug effective against hypobiotic larvae in order to prevent type II disease.

The prevention of ostertagiosis by limiting exposure to infection is a more efficient method of control. This may be achieved by allowing young cattle sufficient exposure to larval infection to stimulate immunity but not sufficient to cause a loss in production. The provision of this 'safe pasture' may be achieved in two ways.

1. Using anthelmintics to limit pasture contamination with eggs during periods when the climate is optimal for development of the free-living larval stages, i.e. spring and summer in temperate climates, or autumn and winter in the subtropics.
2. Alternatively, by resting pasture or grazing it with another host, such as sheep, which are not susceptible to *O. ostertagi*, until most of the existing L_3 on the pasture have died out.

Sometimes a combination of these methods is employed. The timing of events in the systems described in Table 8.1 is applicable to the calendar of the northern hemisphere.

Prophylactic anthelmintic medication Since the crucial period of pasture contamination with *O. ostertagi* eggs is the period up to mid-July, one of the efficient modern anthelmintics may be given on two or three occasions between turnout in the spring and July to minimise the numbers of eggs deposited on the pasture. For calves going to pasture in early May two treatments, 3 and 6 weeks later, are used, whereas calves turned out in April require three treatments at intervals of 3 weeks. Where parenteral or pour-on macrocyclic lactones are used the interval after first

Table 8.1 Risk management of pastures.

	High	Medium	Low
Spring	Grazed by first-year calves in the previous year	Grazed only by adult or yearling cattle the previous year Grazed by beef cows (with or without calves at foot) the previous year	New leys/seeds or forage crops Sheep or conservation only in the previous year
From mid July	Grazed by first-year calves in the spring	Adult cattle or conservation in the spring Pasture clean at the start of the year and grazed by parasite-naive calves	Grazed by sheep or conservation only in the first half of the grazing season Forage crops or arable by-products

treatment may be extended to 5 or 8 weeks (the interval depends on the anthelmintic used) due to residual activity against ingested larvae. A long-acting injectable formulation of moxidectin is available in some countries with persistent activity against *O. ostertagi* for around 120 days.

Several rumen boluses are available which provide either the sustained release of anthelmintic drugs, at a constant level, over periods of 3–5 months or the pulse release of therapeutic doses of an anthelmintic at intervals of 3 weeks throughout the grazing season. These are administered to first-season grazing calves at turnout, although some can be administered later in the season, and effectively prevent pasture contamination and the subsequent accumulation of infective larvae. Although offering a high degree of control of gastrointestinal nematodes, there is evidence to suggest that young cattle protected by these boluses, or other highly effective prophylactic drug regimens, are more susceptible to infection in their second year at grass. Boluses can also be used in the second grazing season. One advantage of using boluses is a reduction in handling and hence labour costs.

Anthelmintic prophylaxis has the advantage that animals can be grazed throughout the year on the same pasture and is particularly advantageous for the small heavily stocked farm where grazing is limited.

Anthelmintic treatment and move to safe pasture in mid-July This system, usually referred to as 'dose and move', is based on the knowledge that the annual increase of L_3 occurs after mid-July. Therefore if calves grazed from early spring are given an anthelmintic treatment in early July and moved immediately to a second pasture such as silage or hay aftermath, the level of infection which develops on the second pasture will be low. The one reservation with this technique is that in certain years the numbers of L_3 that overwinter are sufficient to cause heavy infections in the spring and clinical ostertagiosis can occur in calves in April and May. However, once the dose-and-move system has operated for a few years this problem is unlikely to arise. In some European countries the same effect has been obtained by delaying the turnout of calves until midsummer.

The dose-and-move strategy is considered to select heavily for resistance and the current recommendation, for sheep, is to delay the move after the dose. Practically, however, this is difficult with calves because of the variations in persistence in activity between macrocyclic lactone products and the timing between treatment intervals. The intention of delaying treatment is to allow any treated calves to become 'lightly' reinfected with susceptible worms before allowing them access to the 'low-risk' pasture. This will ensure that soon after the move, contamination of the 'clean' pasture with eggs from susceptible worms will recommence and reduce the reproductive advantage offered to any resistant parasites surviving treatment. However, it should be possible to plan the availability of aftermaths with turnout and the need for strategic early season worming plans as part of farm health planning initiatives.

Alternate grazing of cattle and sheep This system ideally utilises a 3-year rotation of cattle, sheep and crops. Since the effective lifespan of most *O. ostertagi* L_3 is under 1 year and cross-infection between cattle and sheep in temperate areas is largely limited to *O. leptospicularis*, *Trichostrongylus axei* and occasionally *C. oncophora*, good control of bovine ostertagiosis should, in theory, be achieved. It is particularly applicable to farms with a high proportion of land suitable for cropping or grassland conservation. In marginal or upland areas reasonable control has been reported using an annual rotation of beef cattle and sheep. The drawback of alternate grazing systems is that they impose a rigorous and inflexible regimen on the use of land. Furthermore, in warmer climates where *Haemonchus* spp. are prevalent, this system can prove dangerous since this very pathogenic genus establishes in both sheep and cattle.

Rotational grazing of adult and young stock This system involves a continuous rotation of paddocks in which the susceptible younger calves graze ahead of the immune adults and remain long enough in each paddock to remove only the leafy upper herbage. The incoming immune adults then graze the lower more fibrous echelons of the herbage, which contain the majority of the L_3. Since the faeces produced by the immune adults contain few if any *O. ostertagi* eggs, the pasture contamination is greatly reduced. The optimal utilisation of permanent grassland and the control of internal parasitism without resort to therapy make it an option for organic systems of production. In single-suckled beef production systems, the grazing of immune cows with their offspring will lower the pasture infectivity levels for the susceptible calves.

Influence of production systems In northern temperate regions beef cows normally calve in the spring or autumn. Dairy cows may follow a similar pattern of calving, although in many herds calving occurs all year round. Parasite control in dairy herds reflects the management of the calves, which are usually removed from the dam soon after birth and reared indoors until weaning as heifer replacements. Age and timing of turnout will therefore be influenced by month of birth and availability of pasture. Calving typically occurs in late winter/early spring in spring-calving herds and weaned calves may be turned out onto pasture as early as 2 months of age in April or May, acquiring infection from overwintering infective larvae and subsequent pasture contamination. With a longer calving period the calves may be housed until after weaning and then grazed on pasture from midsummer. If the pasture has been grazed by older calves, then they could be exposed to high levels of larval challenge. Calves that are born in the late summer or autumn may be housed and then enter their second year as mainly parasite-naive livestock and be susceptible to infection after turnout.

Anthelmintic resistance There are sporadic reports of gastrointestinal nematodes in cattle showing some resistance to benzimidazoles in several countries and a few isolated reports of resistance to the macrocyclic lactones, particularly in *Cooperia* species. However, anthelmintic resistance is considered to be much less of a problem in cattle compared to the situation in sheep and goats. This may be due to less frequent treatment of cattle and also the persistence of faecal pats, which prolong the survival of the free-living larval stages. Despite the current low level of anthelmintic resistance in cattle nematodes, it is advisable to follow guidelines (such as those listed in Table 8.2) devised to limit the development of resistance in cattle.

Notes: *Ostertagia ostertagi* is perhaps the most common cause of parasitic gastritis in cattle. The disease, often simply known as ostertagiosis, typically affects young cattle during their first grazing season, although herd outbreaks and sporadic individual cases have also been reported in adult cattle. *Ostertagia ostertagi* is considered to be a polymorphic species with *Ostertagia lyrata* (syn. *Skrjabinagia*).

Table 8.2 Guidelines for the control of gastrointestinal nematodes and use of anthelmintics in cattle (UK COWS recommendations).

Guideline	Comment
1 Work out a control strategy with your veterinarian or adviser	Specialist consultation as part of herd health planning is an increasing requirement on farms. Worm control programmes for cattle will require ongoing consultations
2 Use effective quarantine strategies to prevent the importation of resistant worms in introduced cattle	Bought-in cattle can be a potential route of introducing resistance alleles into a non-closed herd
3 Test for anthelmintic efficacy on your farm	While resistance is still rare in cattle nematodes, treatment failures do occur. It is important to monitor continued efficacy as underdosing can select for anthelmintic resistance
4 Administer anthelmintics effectively	Administer the right dose in the correct way by following the manufacturer's instructions
5 Use anthelmintics only when necessary	Understand the trade-off between tolerating some level of parasitism and minimising selection for anthelmintic resistance. FEC monitoring has an important role
6 Select the appropriate anthelmintic for the task	Target treatment according to parasites (and their stages) present, based on time of year
7 Adopt strategies to preserve susceptible worms on the farm	Aim to reduce selection for anthelmintic resistance when treating adult cattle, immune older animals or when dosing on low contamination pastures
8 Reduce dependence on anthelmintics	Alternative control measures include grazing management using sheep or older immune animals

Ostertagia leptospicularis

Synonyms: *Ostertagia crimensis, Skrjabinagia kolchida, Grosspiculagia podjapolskyi*

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: Adults are slender reddish-brown worms with a short buccal cavity. Males measure 6–8 mm and females 8–9 mm in length.

Description, microscopic: Distinguished from other ostertagian species by the length of the oesophagus, which is longer than in other species (0.7 mm compared with approximately 0.6 mm). In cattle, the worms are thinner than *Ostertagia ostertagi* and male worms are differentiated on spicule morphology (see Table 1.4).

Hosts: Deer (roe deer), cattle, sheep, goat

Geographical distribution: Many parts of the world, particularly Europe and New Zealand

Notes: Considered to be a polymorphic species with two male morphs, *Ostertagia leptospicularis* and *Skrjabinagia kolchida* (*Grosspiculagia podjapolskyi*).

Details of the pathogenesis, clinical signs, diagnosis, pathology, epidemiology, treatment and control are as for *O. ostertagi*.

Spiculopteragia spiculoptera

Synonyms: *Apteragia spiculoptera, Rinadia spiculoptera, Mazamostrongylus spiculoptera*

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, microscopic: The spicules are of equal length, bifurcating distally where it contains a cavity and ending distally in a fan-shaped expansion (see Table 1.4g). The gubernaculum is absent.

Hosts: Deer (red deer, fallow deer, roe deer), cattle, sheep, goat

Haemonchus contortus

Synonym: *Haemonchus placei* (see Notes)

Common name: Barber's pole worm

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Notes: Until recently the sheep species was called *H. contortus* and the cattle species *H. placei*. However, there is now increasing evidence that these are the single species *H. contortus* with only strain adaptations for cattle and sheep.

For more details see Chapter 9.

Haemonchus similis

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: The adults are 2.0–3.0 cm long and reddish in colour.

Description, microscopic: The male has an asymmetrical dorsal lobe and barbed spicules differing from *H. contortus* in that the terminal processes of the dorsal ray are longer and the spicules shorter.

Hosts: Cattle, deer

Geographical distribution: North America, Europe

Pathogenesis: As for *H. contortus*

Trichostrongylus axei

Synonym: *Trichostrongylus extenuatus*

Predilection site: Abomasum or stomach

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

For more details, see Chapter 9.

Mecistocirrus digitatus

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: To the naked eye, the worm is indistinguishable from *Haemonchus contortus*, although it is closely related to *Nematodirus*. The white ovary is wrapped around the red blood-filled intestine giving it a 'barber's pole' appearance. The males measure up to around 30 mm and the females 42 mm in length.

Description, microscopic: The male is distinguishable from *Haemonchus* by the presence of long narrow spicules that are fused together for the majority of their length and the tips are enclosed in a spindle-shaped appendage (in *Haemonchus* the spicules are thicker, separate and barbed at the tips). The dorsal ray is symmetrically located in the bursa, whereas in *Haemonchus* the dorsal ray is asymmetrical. The female differs from *Haemonchus* in that the slit-shaped vulva is positioned nearer to the tip of the tail and there is no vulval flap. The cuticle contains many longitudinal ridges and the cervical papillae are readily apparent. The small buccal capsule is armed with a lancet. The eggs are large and, unlike *Nematodirus*, typically strongylate and measure around 100 μm in length.

Hosts: Cattle, buffalo, zebu, sheep and goat; occasionally the stomach of the pig and rarely human

Geographical distribution: Tropical and subtropical regions, particularly Central America and parts of Asia

Pathogenesis: In endemic areas, the pathogenesis of this haematophagous parasite is similar to that of *H. contortus* and it is of similar economic importance.

Clinical signs: Similar to *H. contortus*, inducing anaemia, weight loss and emaciation.

Diagnosis: See the description of the parasite.

Treatment and control: See *H. contortus* for details.

Parabronema skrjabini

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Geographical distribution: Central and East Africa, Asia, and some Mediterranean countries, notably Cyprus

For more details see Chapter 9.

Capillaria bilobata

Predilection site: Abomasum

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichuroidea

Description: Male worms measure 10–16 mm and females 14–21 mm in length. The lemon-shaped eggs measure 33–53 by 14–21 μm and have two slightly protruding polar plugs.

Host: Zebu

Geographical distribution: Indian subcontinent

Cryptosporidium andersoni

Synonym: *Cryptosporidium muris*

Predilection site: Abomasum

Phylum: Apicomplexa

Class: Conoidasida

Family: Cryptosporidiidae

Description: Oocysts, passed fully sporulated, are ellipsoid, 6.0–8.1 by 5.0–6.5 μm (mean 7.4 × 5.5 μm), with a length/width ratio of 1.35.

Host: Cattle

Geographical distribution: Reported in USA, Brazil, UK, Czech Republic, Germany, France, Japan and Iran.

Pathogenesis: Generally considered to be non-pathogenic.

Clinical signs: Usually asymptomatic, although depressed weight gain in calves and milk yields in milking cows have been reported.

Diagnosis: Oocysts may be demonstrated using Ziehl–Neelsen stained faecal smears in which the sporozoites appear as bright red granules. Speciation of *Cryptosporidium* is difficult, if not impossible, using conventional techniques. A range of molecular and immunological techniques has been developed that includes the use of immunofluorescence or ELISA. More recently, DNA-based techniques have been used for the molecular characterisation of *Cryptosporidium* species.

Pathology: The presence of the endogenous stages of the parasite leads to destruction of the microvilli of peptic glands, leading to elevated concentrations of plasma pepsinogen.

Epidemiology: The epidemiology of infection has not been studied, although it is likely to be similar to that of *Cryptosporidium parvum* in cattle. Many calves are likely to become infected without showing clinical signs but become sources of infection for calves that follow. The primary route of infection is direct animal to animal via the faecal–oral route. Thus in calves, for example, overcrowding, stress of early weaning, transport and marketing, together with low levels of hygiene, will increase the risk of heavy infections.

Treatment and control: There is no reported treatment. Good hygiene and management are important in preventing disease from cryptosporidiosis. Feed and water containers should be high

enough to prevent faecal contamination. Young animals should be given colostrum within the first 24 hours of birth and overstocking and overcrowding should be avoided. Dairy calves should be either isolated in individual pens or kept in similar age groups and cleaned out daily.

Notes: Based on oocyst morphology, *C. muris*-like oocysts have been found in cattle in several countries around the world. Recent molecular characterisations have indicated that all bovine isolates are *C. andersoni*.

SMALL INTESTINE

Trichostrongylus colubriformis

Synonym: *Trichostrongylus instabilis*

Common name: Black scour or bankrupt worm

Predilection site: Duodenum and anterior small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

For more details see Chapter 9.

Trichostrongylus longispicularis

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: The adults are similar in size to *T. colubriformis*.

Description, microscopic: The spicules are stout, brown, unbranched, slightly unequal in length and terminate in a tapering blunt tip that has a small semi-transparent protrusion.

Hosts: Cattle, sheep, goat, deer, camel, llama

Geographical distribution: Ruminants in Australia; cattle in America and parts of Europe

Details of the pathogenesis, clinical signs, diagnosis, pathology, epidemiology, treatment and control are as for *T. colubriformis*.

Cooperia oncophora

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: In size *C. oncophora* is similar to *Ostertagia* but with a large bursa. Males measure around 5.5–9 mm and females 6–8 mm in length. When fresh the worms appear pinkish white.

Description, microscopic: The main generic features are the small cephalic vesicle and the transverse cuticular striations in the oesophageal region. The body possesses longitudinal ridges. The spicules have a distinct wing-like expansion in the middle region and often bear ridges (see Table 1.5a); there is no gubernaculum. The females have a long tapering tail. The medium-sized eggs are oval, thin-shelled with a smooth surface and measure 74–95 by 36–44 μm. The egg contains many blastomeres that are not easily distinguished. The small poles are very similar and the side walls are parallel. This feature allows the eggs to be differentiated from those of *Ostertagia* which have wider poles and more spherical walls.

Hosts: Cattle, sheep, goat, deer, camel

Geographical distribution: Worldwide

Pathogenesis: *Cooperia oncophora* is generally considered to be a mild pathogen in calves, although in some studies it has been associated with inappetence and poor weight gains. Immunity to reinfection develops after about 8–12 months of exposure to infective larvae.

Clinical signs: These include loss of appetite and poor weight gains. Occasionally a heavy infection can induce intermittent diarrhoea.

Diagnosis: Eggs of *Cooperia* spp. are all very similar morphologically. Faecal culture will allow identification of infective larvae.

Pathology: Moderate to heavy infections can induce a catarrhal enteritis with localised villous atrophy and oedema of the intestinal mucosa.

Epidemiology: In temperate areas, this is similar to that of *Ostertagia*. Arrested development (hypobiosis) at the EL_4 stage is a regular feature during late autumn and winter in the northern hemisphere, and in spring and summer in the southern hemisphere. Adult animals usually show few signs of infection but act as carriers, shedding low numbers of eggs in their faeces.

In the subtropics, the epidemiology is similar to that of *Haemonchus* though *Cooperia* does not have the same high biotic potential and the L_3 survive rather better under arid conditions. Hypobiosis is also a feature during prolonged dry seasons.

Treatment: The principles are similar to those applied in bovine ostertagiosis. *Cooperia* is one of the dose-limiting species and one should consult the manufacturer's data sheets for efficacy of anthelmintics against adult and L_4 stages.

Control: Similar to that described for *Ostertagia*.

Notes: In temperate areas, members of the genus *Cooperia* usually play a secondary role in the pathogenesis of parasitic gastroenteritis of ruminants, although they may be the most numerous trichostrongyle present. However, in some tropical and subtropical areas, some species are responsible for severe enteritis in calves.

Three further species of *Cooperia* are found in cattle. Details of the diagnosis, epidemiology, treatment and control are as for *C. oncophora*.

Cooperia punctata

Common name: Cattle bankrupt worm

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: Similar to *C. oncophora*. Males measure around 4.5–6.0 mm, and females 6–8 mm in length.

Description, microscopic: See *C. oncophora* and Table 1.5(b) for further details. The medium-sized, thin-shelled oval eggs have a smooth surface and measure about 69–83 by 29–34 μm. The egg contains many blastomeres which are not easily distinguished.

Hosts: Cattle, deer

Geographical distribution: Worldwide

Pathogenesis: *Cooperia punctata* is a pathogenic parasite since it penetrates the epithelial surface of the small intestine and causes a disruption similar to that of other intestinal trichostrongylid species, which leads to villous atrophy and a reduction in the area available for absorption. In heavy infections, diarrhoea has been reported.

Clinical signs: There is loss of appetite, poor weight gain and diarrhoea and there may be submandibular oedema.

Cooperia pectinata

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: Similar to *C. oncophora*. Males measure around 7–8 mm and females 7.5–10 mm in length.

Description, microscopic: See *C. oncophora* and Table 1.5(e) for details.

Hosts: Cattle, deer

Geographical distribution: Worldwide

Pathogenesis and clinical signs: Similar to *C. punctata*. A catarrhal enteritis is often present with loss of appetite, poor weight gain, diarrhoea and, in some cases, submandibular oedema.

Cooperia surnabada

Synonym: *Cooperia mcmasteri*

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: The males measure around 7 mm and the females 8 mm in length.

Description, microscopic: The appearance is very similar to *C. oncophora*, although the bursa is larger and the bursal rays tend to be thinner. The spicules are thinner with a posterior bifurcation and the tips possess a small conical appendage (see Table 1.5c).

Hosts: Cattle, sheep, camel

Geographical distribution: Parts of Europe, North America and Australia

Pathogenesis: Moderate pathogenicity as the worms penetrate the surface of the small intestine and can induce villous atrophy.

Clinical signs: See *C. punctata*

Diagnosis: See *C. oncophora*

Treatment and control: Refer to *C. oncophora*

Nematodirus helvetianus

Common name: Thread-necked worm

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: The adults are slender, males measuring around 11–16 mm and females 17–24 mm in length.

Description, microscopic: A small but distinct cephalic vesicle is present. The male has two sets of parallel rays in each of the main bursal lobes and the long slender spicules end in a fused point with the surrounding membrane being lanceolate (see Table 1.6d). The female has a truncate tail with a small spine. The egg is large (160–233 by 87–121 μm), ovoid with slightly sharp poles and clear, and twice the size of the typical trichostrongyle egg. The chitinous eggshell is thin with a smooth surface and contains two to eight large dark blastomeres, which are separated from the yolk membrane by quite a large fluid-filled cavity.

Hosts: Cattle, occasionally sheep, goat and other ruminants

Geographical distribution: Worldwide

Pathogenesis: Although this is similar to that of *Nematodirus battus*, there is some controversy over the extent of the pathogenic effect. *Nematodirus helvetianus* has been incriminated in outbreaks of bovine parasitic gastroenteritis but experimental attempts to reproduce the disease have been unsuccessful.

Clinical signs: Low to moderate infections may produce no obvious clinical manifestations. In severe infections, diarrhoea can occur during the prepatent period and young animals may become dehydrated.

Diagnosis: Examination of faeces will allow the large colourless eggs to be differentiated from those of *N. spathiger*. At necropsy, the tips of the male spicules will allow diagnosis from other *Nematodirus* species.

Pathology: Increased mucus production and focal compression and stunting of villi may occur in the small intestine.

Epidemiology: The eggs do not usually exhibit delayed hatching. The pattern of infection is similar to that of *Trichostrongylus* species.

Treatment: Several drugs are effective against *Nematodirus* infections: levamisole, an avermectin/milbemycin or one of the modern benzimidazoles. However, *Nematodirus* is one of the dose-limiting

species and manufacturer's data sheets should be consulted as there are differences in efficacy against adults and L_4 stages between oral and parenteral administration for some macrocyclic lactones. The response to treatment is usually rapid and if diarrhoea persists, coccidiosis should be considered as a complicating factor.

Control: Disease due to monospecific *Nematodirus* infections is rarely seen. They are usually part of the worm burden of trichostrongyloid species that are responsible for the syndrome of parasitic gastroenteritis in cattle and as such may be controlled by the measures outlined elsewhere.

Nematodirus battus

Common name: Thread-necked worm

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Notes: *Nematodirus battus* has only rarely been recorded in cattle.

Nematodirus spathiger

Common name: Thread-necked worm

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Notes: *Nematodirus spathiger* is only occasionally reported in cattle. The eggs are similar in appearance and may be confused with those of *N. helvetianus*.

For more details of both these species see Chapter 9.

Bunostomum phlebotomum

Synonym: *Monodontus phlebotomum*

Common name: Cattle hookworm

Predilection site: Small intestine, particularly the anterior jejunum and/or duodenum

Phylum: Nematoda

Class: Secernentea

Superfamily: Ancylostomatoidea

Description, gross: *Bunostomum* is one of the larger nematodes of the small intestine of ruminants (see Fig. 8.8), being 1–3 cm long, stout, greyish-white and characteristically hooked at the anterior end with the buccal capsule opening anterodorsally.

Description, microscopic: The large buccal capsule opens anterodorsally and bears on the ventral margin a pair of chitinous cutting plates and internally a large dorsal cone. Dorsal teeth are absent from the buccal capsule but there are two pairs of small subventral lancets at its base. In the male the bursa is well developed and has an asymmetrical dorsal lobe. The right externo-dorsal ray arises higher up on the dorsal stem and is longer than the left. It arises near the bifurcation of the dorsal ray, which divides into two tridigitate branches. The spicules are very long and slender. In the female the vulva opens a short distance in front of the middle of the body.

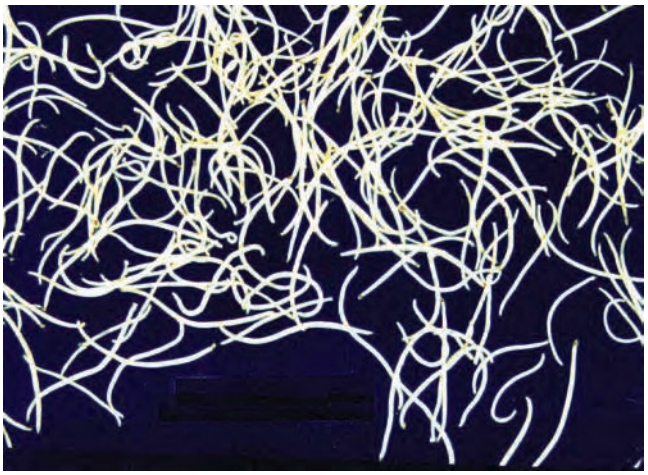

Figure 8.8 Adult *Bunostomum phlebotomum*.

The infective larva is small with 16 gut cells and a short filamentous tail. The egg is a medium-sized (97–106 by 45–55 μm), thin-shelled, irregular broad ellipse with blunt ends and dissimilar side walls, one being flattened. It contains four to eight darkly pigmented blastomeres.

Hosts: Cattle

Geographical distribution: Worldwide

Pathogenesis: The adult worms are blood-suckers and infections of 100–500 worms can produce progressive anaemia, hypoalbuminaemia, loss of weight and occasionally diarrhoea. Worm burdens of around 2000 may lead to death in cattle. In stabled cattle, pruritus of the limbs, probably caused by skin penetration by the larvae, is seen.

Clinical signs: There may be inappetence, diarrhoea and emaciation, more frequently seen in young animals. Severe infection can also induce submandibular oedema ('bottle jaw'). Postmortem examination often reveals hydrothorax and fluid within the pericardium. Older livestock frequently develop sufficient immunity to limit reinfection and in many cases *Bunostomum* is present asymptomatically. In calves, foot stamping and signs of itching may accompany skin penetration by the larvae.

Diagnosis: The clinical signs of anaemia and perhaps diarrhoea in calves are not in themselves pathognomonic of bunostomosis. However, in temperate areas, the epidemiological background may be useful in eliminating the possibility of *Fasciola hepatica* infection. In the tropics, haemonchosis must be considered, possibly originating from hypobiotic larvae. Faecal egg counts are useful in that these are lower than in *Haemonchus* infection while the eggs are more bluntly rounded, with relatively thick sticky shells to which debris is often adhered. For accurate differentiation, larval cultures should be prepared.

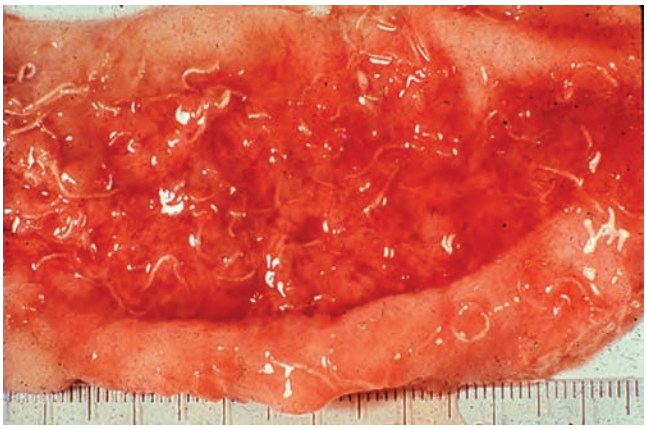

Figure 8.9 Inflamed and haemorrhagic intestinal mucosa due to the presence of feeding worms (*Bunostomum phlebotomum*).

Pathology: The carcass is anaemic and cachexic. Oedema and ascites are seen. The liver is light brown and shows fatty changes. The intestinal contents are haemorrhagic and the mucosa is usually swollen, covered with mucus, and shows numerous lesions resulting from the worms feeding (Fig. 8.9). The parasites may be seen still attached to the mucosa or free in the lumen.

Epidemiology: Pathogenic infections are more common in the tropics and subtropics and in some areas, such as Nigeria, the highest worm burdens are found at the end of the dry season, apparently due to the maturation of hypobiotic larvae. Young livestock are most susceptible. *Bunostomum phlebotomum* is often a serious pathogen in many regions, such as the southern and mid-western USA, Australia and parts of Africa. In temperate countries, high worm burdens are usually uncommon. The prophylactic dosing regimens, adopted for the control of trichostrongyles, have contributed to the low prevalence of *Bunostomum*.

Treatment: Anthelmintics listed for *O. ostertagi* are effective.

Control: A combination of strategic dosing with anthelmintics and pasture management is used in the control of larvae as they are susceptible to desiccation, and the infection is mainly found on permanently or occasionally moist pastures. Avoiding or draining such pastures is an effective control measure. The ground around water troughs should be kept hard and dry, or treated with liberal applications of salt. Stabled cattle should be protected by ensuring the floors and bedding are kept dry and that faeces are removed frequently, and are not allowed to contaminate food and water.

Agriostomum vryburgi

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Ancylostomatoidea

Description, gross: Worms are stout and greyish-white in colour. Males are around 9–11 mm and females 13–16 mm in length. Spicules are equal in length and a gubernaculum is present.

Description, microscopic: The shallow buccal capsule contains four pairs of large teeth on its margin and has a rudimentary leaf-crown. The large oesophageal opening houses two small subventral lancets. Eggs measure about 130–190 by 60–90 μm.

Hosts: Cattle, buffalo, ox and zebu

Geographical distribution: Asia and South America

Pathogenesis: The hookworms attach to the mucosa of the anterior small intestine. The pathogenicity, although unknown, presumably depends on its haematophagic habits, inducing anaemia.

Notes: *Agriostomum vryburgi* is a common hookworm of the large intestine throughout its distribution range. Details on the diagnosis, treatment and control are likely to be similar to those for *B. phlebotomum*.

Strongyloides papillosus

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Rhabditoidea

Description, gross: Slender hair-like worms generally less than 1.0 cm long.

Description, microscopic: Only females are parasitic. The long oesophagus may occupy up to one-third of the body length and the uterus is intertwined with the intestine, giving the appearance of twisted thread. Unlike other intestinal parasites of similar size the tail has a blunt point. *Strongyloides* eggs are oval with blunt poles and slightly barrel-shaped side walls, thin-shelled and small, being half the size of typical strongyle eggs. These colourless eggs have a smooth shell and measure about 43–60 by 20–25 μm and contain an L_1 larva. Infective larvae measure about 600 μm.

Hosts: Cattle, sheep, goat and other ruminants, pig and rabbit

Geographical distribution: Worldwide

Pathogenesis: Skin penetration by infective larvae may cause an erythematous reaction. Passage of larvae through the lungs has been shown experimentally to result in multiple small haemorrhages visible over most of the lung surfaces. Mature parasites are found in the duodenum and proximal jejunum and if present in large numbers may cause inflammation with oedema and erosion of the epithelium. This results in a catarrhal enteritis with impairment of digestion and absorption.

Clinical signs: The common clinical signs, usually seen only in very young animals, are diarrhoea, dehydration, anorexia, dullness, loss of weight or reduced growth rate.

Diagnosis: The clinical signs in very young animals, usually within the first few weeks of life, together with the finding of large numbers of the characteristic eggs or larvae in the faeces, are suggestive of strongyloidosis. However, it should be emphasised that high faecal egg counts may be found in apparently healthy animals.

Pathology: Adult worms establish in tunnels in the epithelium at the base of the villi in the small intestine. In large numbers they may cause villous atrophy, with a mixed mononuclear inflammatory cell

infiltration of the lamina propria. Crypt epithelium is hyperplastic and there is villous clubbing.

Epidemiology: *Strongyloides* infective larvae are not ensheathed and are susceptible to extreme climatic conditions. However, warmth and moisture favour development and allow the accumulation of large numbers of infective stages. For this reason, it can be a major problem in housed calves up to 6 months of age in some Mediterranean countries. A second major source of infection for the very young animal is the reservoir of larvae in the tissues of their dams and this may lead to clinical strongyloidosis in the first few weeks of life. Successive progeny from the same dam often show heavy infections.

Treatment: Specific control measures for *Strongyloides* infection are rarely required. The benzimidazoles and the avermectins/milbemycins may be used for the treatment of clinical cases.

Control: Reduction in numbers of free-living larvae by removal of faeces and provision of dry bedding and areas may limit numbers and transmission. Suckling calves should be kept on clean dry areas to prevent infection by skin penetration.

Toxocara vitulorum

Synonym: *Neoascaris vitulorum*

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Ascaridoidea

Description, gross: This is a very large whitish nematode. The adult male is up to 25 cm and the female 30 cm in length.

Description, microscopic: The cuticle is less thick than other ascarids and somewhat soft and translucent. There are three lips, broad at the base and narrowing anteriorly. The oesophagus is 3–4.5 mm long and has a posterior, granular ventriculus. The tail of the male usually forms a small spike-like appendage. There are about five pairs of post-cloacal papillae; the anterior pair is large and double. Pre-cloacal papillae are variable in number. The vulva is situated about one-eighth of the body length from the anterior end. The medium-sized egg of *T. vitulorum* is subglobular, with a thick finely pitted albuminous shell, and is almost colourless (75–95 by 60–74 μm) (see Fig. 4.3). The egg is unsegmented and the granular contents frequently only occupy part of the internal volume.

Hosts: Cattle, buffalo and zebu, rarely sheep and goat

Geographical distribution: Africa, India, Asia

Pathogenesis: The main effects of *T. vitulorum* infection appear to be caused by the adult worms in the intestines of calves up to 6 months old. Heavy infections are often associated with unthriftiness, catarrhal enteritis and intermittent diarrhoea, and in buffalo calves particularly fatalities may occur. Heavy burdens can be associated with intestinal obstruction and occasionally perforation may occur leading to peritonitis and death.

Clinical signs: Diarrhoea, poor condition

Diagnosis: In some instances heavily infected calves may exhale an acetone-like odour. The subglobular eggs, with thick pitted shells, are characteristic in bovine faeces. Egg output in young calves can be very high (>50,000 epg) but patency is short and by around 4–6 months of age calves have expelled most of their adult worm population.

Pathology: The pathological effects of adult worms in the intestine are poorly defined. Heavy infections may obstruct the gut and lead to gut perforation. Migration up the bile or pancreatic duct may lead to biliary obstruction and cholangitis.

Epidemiology: The most important feature is the reservoir of larvae in the tissues of the cow, with subsequent milk-borne transmission ensuring that calves are exposed to infection from the first day of life. The majority of patent infections occur in calves of less than 6 months of age.

Treatment: The adult worms are susceptible to a wide range of anthelmintics, including piperazine, levamisole, macrocyclic lactones and the benzimidazoles. Many of these drugs are also effective against developing stages in the intestine.

Control: The prevalence of infection can be dramatically reduced by treatment of calves at 3 and 6 weeks of age, preventing developing worms reaching patency.

Capillaria bovis

Synonym: *Capillaria brevipes*

Predilection site: Small intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichuroidea

Description, gross: These are very fine filamentous worms, the narrow stichosome oesophagus occupying about one-third to half the body length. Males measure around 8–9 mm and females up to 12 mm.

Description, microscopic: The males have a long thin single spicule about 0.9 mm long and often possess a primitive bursa-like structure. The small eggs are slightly lemon-shaped (similar to *Trichuris*) but the side walls are almost parallel. They measure 45–50 by 22–25 μm, are colourless and have thick shells that are slightly striated with two protruding transparent bipolar plugs. The contents are granular with no blastomeres.

Hosts: Cattle, sheep, goat

Geographical distribution: Worldwide

Pathogenesis: Considered to be of low pathogenicity and of little veterinary significance.

Clinical signs: No clinical signs have been attributed to infection with this parasite.

Diagnosis: Because of the non-specific nature of the clinical signs and the fact that, in heavy infections, these may appear before eggs are present in the faeces, diagnosis depends on necropsy and careful examination of the small intestine for the presence of the worms. This may be carried out by microscopic examination of mucosal scrapings squeezed between two glass slides; alternatively, the contents should be gently washed through a fine sieve and the retained

material resuspended in water and examined against a black background.

Pathology: No associated pathology

Epidemiology: Infection is by ingestion of the larvated eggs. Infection is common in sheep though not significant.

Treatment: Not usually required

Control: Not required

Moniezia benedeni

Predilection site: Small intestine

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: These long tapeworms (2 m or more) are unarmed and possess prominent suckers.

Description, microscopic: Segments are broader than they are long (up to 2.5 cm wide) and contain two sets of genital organs grossly visible along the lateral margin of each segment. There is a row of inter-proglottid glands at the posterior border of each segment, which may be used in species differentiation; in *M. benedeni* they are confined to a short row close to the middle of the segment (see Table 1.10). The medium-sized irregularly quadrangular eggs have a well-defined pyriform apparatus and vary from 80 to 90 μm in diameter. The egg has a thick smooth shell and contains an embryo.

Final hosts: Cattle, buffalo

Intermediate hosts: Forage mites, mainly of the family Oribatidae

Geographical distribution: Worldwide

Pathogenesis: Generally regarded as of little pathogenic significance.

Clinical signs: No clinical signs have been associated with infection.

Diagnosis: This is based largely on the presence of mature proglottids in the faeces and the characteristic shape of *Moniezia* eggs (triangular, *M. expansa*; quadrangular, *M. benedeni*) that contain the oncosphere. The eggs of *M. benedeni* are slightly larger than those of *M. expansa* in sheep.

Pathology: No reported pathology

Epidemiology: Infection is common in calves during their first year of life and less common in older animals. A seasonal fluctuation in the incidence of *Moniezia* infection can apparently be related to active periods of the forage mite vectors during the summer in temperate areas. The cysticercoids can overwinter in the mites.

Treatment: In many countries a variety of drugs, including niclosamide, praziquantel, bunamidine and a number of broad-spectrum benzimidazole compounds, which have the advantage of also being active against gastrointestinal nematodes, are available for the treatment of *Moniezia* infection. If this is carried out in calves in late spring, in temperate areas, the numbers of newly infected mites on pasture will be reduced.

Control: Ploughing and reseeding, or avoiding the use of the same pastures for young animals in consecutive years, may prove beneficial.

Notes: This genus of cestodes is common in ruminants and resembles, in most respects, *Anoplocephala* of the horse. *Moniezia* spp. are the only tapeworms of ruminants in many countries of western Europe.

Thysaniezia ovilla

Synonyms: *Thysaniezia giardi, Helictometra giardi*

Predilection site: Small intestine

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: Adults reach 200 cm in length, varying in width up to 12 mm.

Description, microscopic: The scolex is small, measuring up to 1 mm in diameter. Segments are short, bulge outwards giving the margin of the worm an irregular appearance, and contain a single set of genital organs, rarely two, with genital pores alternating irregularly (see Table 1.10). The oval eggs (measuring up to 27 by 19 μm) are devoid of a pyriform apparatus and have a thick grey shell and a protruberance at one end. They are found in groups of 10–15 in the numerous elongated paruterine organs (100 μm long) in each proglottid.

Final hosts: Cattle, sheep, goat, camel and wild ruminants

Intermediate hosts: Oribatid mites (*Galuma*, *Scheloribates*) and psocids (bark lice, dust lice)

Life cycle: Mature segments are passed in the faeces of the infected host onto pasture, where forage mites ingest the oncospheres. Cysticercoids develop within the oribatid intermediate hosts and infection of the final host is by ingestion of infected mites during grazing.

Geographical distribution: Southern Africa

Pathogenesis: Not considered pathogenic

Diagnosis: The mature segments found in the faeces are readily distinguishable from those of *Moniezia*.

Epidemiology: Infection is very commonly found in adult cattle in southern Africa.

Treatment and control: As for *Moniezia*

The following species have also been reported in cattle. For more details on these species see Chapter 9.

Moniezia expansa

Predilection site: Small intestine

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: These long tapeworms (2 m or more) are unarmed and possess prominent suckers.

Description, microscopic: Segments are broader than they are long (up to 1.5 cm wide) and contain two sets of genital organs grossly visible along the lateral margin of each segment (see Table 1.10). There is a row of inter-proglottid glands at the posterior border of each segment, which may be used in species differentiation. In *M. expansa* they extend along the full breadth of the segment.

Final hosts: Sheep, goat, occasionally cattle

Intermediate hosts: Forage mites, mainly of the family Oribatidae

Avitellina centripunctata

Predilection site: Small intestine

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: This tapeworm resembles *Moniezia* on gross inspection except that the segmentation is so poorly marked that it appears somewhat ribbon-like. It can reach 3 m in length by about 3–4 mm in width and the posterior end is almost cylindrical in appearance.

Description, microscopic: Single genitalia are present with the pores alternating irregularly (see Table 1.10). Proglottids are short. Eggs lack a pyriform apparatus and measure around 20–45 μm.

Final hosts: Sheep, goat, camel and other ruminants

Intermediate hosts: Thought to be oribatid mites or psocid lice

Geographical distribution: Europe, Africa and Asia

Pathogenesis: Of negligible pathogenicity, similar to that of *Moniezia* spp.

Clinical signs: Usually asymptomatic

Stilesia globipunctata

Predilection site: Small intestine

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: Adults measure around 0.5 m in length by 3–4 mm wide.

Description, microscopic: A single set of genital organs is present.

Final hosts: Sheep, cattle and other ruminants

Intermediate hosts: As for *Avitellina centripunctata*

Thysanosoma actinoides

Common name: Fringed tapeworm

Predilection site: Small intestine, bile and pancreatic ducts

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description, gross: Adult tapeworms measure 15–30 cm in length by around 8 mm wide.

Description, microscopic: The scolex is up to 1.5 mm; segments are short and fringed posteriorly. In the distal regions of the tapeworm the 'fringes' are as long as the proglottid (see Fig. 1.98 and Table 1.10). Each proglottid contains two sets of genital organs with the testes lying medially. Several paruterine organs are present in each proglottid and the eggs have no pyriform apparatus.

Final hosts: Sheep, cattle, deer

Intermediate hosts: As for *Thysaniezia ovilla*

Geographical distribution: North and South America

Cymbiforma indica

Predilection site: Gastrointestinal tract

Phylum: Platyhelminthes

Class: Trematoda

Family: Notocotylidae

Final hosts: Sheep, goat, cattle

Intermediate host: Snails

Description, gross: Adult fluke are pear-shaped, concave ventrally and measure 0.8–2.7 cm by 0.3–0.9 mm wide.

Description, microscopic: There is no ventral sucker and the cuticle is armed with fine spines ventrally and anteriorly. The ovary has four demarcated lobes. The fluke lacks a pharynx and the oesophagus is short.

Geographical distribution: India

Bovine coccidiosis

At least 20 different species of *Eimeria* are known to infect cattle, of which 13 species are more commonly found. Clinical signs of diarrhoea are associated with the presence of *E. zuernii* or *E. bovis*, which occur in the lower small intestine, caecum and colon. *Eimeria alabamensis* has been reported to cause enteritis in yearling calves in some European countries. Affected animals develop watery diarrhoea, shortly after turnout in the spring on to heavily contaminated pastures previously grazed by calves.

The life cycles of the *Eimeria* species are typically coccidian and the general life cycle is described in detail in Chapter 2. Variations in sites of development of meront and gamont stages, and prepatent periods do occur, and where known are described under the respective species.

Prevalence: Most cattle are infected with coccidia during their lives and in the majority of animals the parasites coexist and cause minimal damage. Disease usually only occurs if animals are subjected to heavy infection or if their resistance is lowered through stress, poor nutrition or intercurrent disease. The presence of infection does not necessarily lead to the development of clinical signs of disease and in many situations low levels of challenge can actually be beneficial by stimulating protective immune responses in the host.

Table 8.3 Predilection sites and prepatent periods of *Eimeria* species in cattle.

Species	Predilection site	Prepatent period (days)
E. alabamensis	Small and large intestine	6–11
E. auburnensis	Small intestine	16–24
E. bovis	Small and large intestine	16–21
E. brasiliensis	Unknown	?
E. bukidnonensis	Unknown	?
E. canadensis	Unknown	?
E. cylindrica	Unknown	?
E. ellipsoidalis	Small intestine	8–13
E. pellita	Unknown	?
E. subspherica	Unknown	7–18
E. wyomingensis	Unknown	13–15
E. zuernii	Small and large intestine	15–17

Pathogenesis: The most pathogenic species of coccidia are those that infect and destroy the crypt cells of the large intestinal mucosa (Table 8.3). This is because the ruminant small intestine is very long, providing a large number of host cells and the potential for enormous parasite replication with minimal damage. If the absorption of nutrients is impaired, the large intestine is, to some extent, capable of compensating. Those species that invade the large intestine are more likely to cause pathological changes, particularly if large numbers of oocysts are ingested over a short period of time. Here, the rate of cellular turnover is much lower and there is no compensation effect from other regions of the gut. In calves that become heavily infected, the mucosa becomes completely denuded, resulting in severe haemorrhage and impaired water resorption leading to diarrhoea, dehydration and death. In lighter infections, the effect on the intestinal mucosa is to impair local absorption. Species that develop more superficially in the small intestine cause a change in villous architecture with a reduction in epithelial cell height and a diminution of the brush border, giving the appearance of a 'flat' mucosa. These changes result in a reduction of the surface area available for absorption and consequently reduced feed efficiency.

Clinical and pathological signs: Clinical signs are associated with the presence of the pathogenic species, *E. zuernii* or *E. bovis*, which occur in the lower small intestine, caecum and colon. *Eimeria alabamensis* has been reported to cause enteritis in first-season grazing calves in the first week following turnout in some European countries. Some animals with coccidiosis develop concurrent nervous signs, including tremors, nystagmus, opisthotonus and convulsions. The cause of these symptoms is unknown, although the possibility of the neurological signs being induced by a toxin has been suggested.

Clinical signs of coccidiosis include weight loss, anorexia and diarrhoea, often bloody. On postmortem, there may be little to see beyond thickening and petechiation of the bowel but mucosal scrapings will reveal masses of gamonts and oocysts. Giant meronts may be seen in the mucosa of the small intestine as pin-point white spots, but unless they are present in vast numbers they cause little harm. The most pathogenic stages are the gamonts.

Host resistance: While animals of all ages are susceptible to infection, younger animals are generally more susceptible to disease. Occasionally, however, acute coccidiosis occurs in much older, even adult cattle with impaired cellular immunity or in those which have been subjected to stress, such as transportation, crowding in feedlot areas, extremes of temperature and weather conditions, changes in environment or severe concurrent infection.

Epidemiology: Bovine coccidiosis is primarily a disease of young animals, normally occurring in calves between 3 weeks and 6 months of age but has been reported in cattle aged 1 year or more. The disease is usually associated with a previous stressful situation such as shipping, overcrowding, changes in feed, severe weather or concurrent infection with parvovirus.

Adult cattle, although possibly the original source of infective oocysts in the environment, are not usually responsible for the heavy levels of contamination encountered. The source is often young calves themselves, which following an initial infection often in the first few days of life may produce millions of oocysts within their own environment. Growing animals may then face potentially lethal doses of infective oocysts 3 weeks later when their natural resistance is at its lowest. Later-born calves introduced into the same environment are immediately exposed to heavy oocyst challenge. Under unhygienic overcrowded conditions, the young calves will be exposed to and ingest a large proportion of this infection and will develop severe disease and may even die from the infection. If conditions are less crowded and more hygienic, the infective dose ingested will be lower, they may show moderate, slight or no clinical signs and develop immunity to reinfection, but they will in turn have multiplied the infection a million-fold.

Stress factors, such a poor milk supply, weaning, cold weather and transport, will reduce any acquired resistance and exacerbate the condition. A major problem in milking herds (cattle) is that in an attempt to ensure a constant year-round milk supply, births often take place over an extended period of time. If the same pens are used constantly for successive batches, or if young calves are added to a pen already housing older calves, then the later born are immediately exposed to heavy challenge and can show severe coccidiosis in the first few weeks of life.

Age is therefore one of the main risk factors. During their first weeks of life, young ruminants are normally protected by passive immunity derived from colostrum. Neonatal animals receiving insufficient intake of colostrum and milk or experiencing periods of stress may start to show clinical signs of disease from about 18 days of age onwards.

Adult cattle are usually highly resistant to disease, but not totally resistant to infection. As a result, small numbers of parasites manage to complete their life cycle and usually cause no detectable harm. In the wild or under more natural extensive systems of management, susceptible calves are exposed to only low numbers of oocysts and acquire a protective immunity. Extensive grazing, as occurs under natural conditions in the wild, limits the level of exposure to infective oocysts. Under modern production systems, however, young calves are born into a potentially heavily contaminated environment, and where the numbers of sporulated oocysts are high, disease often occurs. Traditionally, indoor housing is a high-risk period especially where young calves are heavily stocked and in conditions that favour rapid oocyst sporulation and high numbers of oocysts in the environment. Three management factors are associated with the development of high levels of infection and the development of disease: pens not cleaned on a regular basis, overcrowding in the pens, and pens used to house different age groups.

The season of the year can also play a role in the appearance of coccidiosis. Coccidiosis is common in spring when young calves are born and turned out onto permanent pastures close to the farm buildings. Inclement weather at this time may cause stress at this

stage, lowering immunity and precipitating disease. Cold winters favour survival of overwintering oocysts in large enough numbers to represent sufficient disease challenge at turnout in spring; conversely, mild wet springs favour sporulation and rapid accumulation of large numbers of infective oocysts. Autumn-born calves may be born into an already heavily contaminated environment.

The effects of coccidial infections may be exacerbated if different species that affect different parts of the gut are present at the same time. Similarly, concurrent infections with other disease-producing agents such as helminths, bacteria and viruses may affect the severity of disease. Interactions between coccidia and parvovirus infection is thought to aggravate coccidiosis in calves.

Diagnosis: Diagnosis should be based on history, clinical signs (severe diarrhoea in young calves), postmortem findings (inflammation, hyperaemia and thickening of caecum with masses of gamonts and oocysts in scrapings), supported by oocyst counts and speciation to identify pathogenic species. Counts of faecal oocysts identified to species can help to complete the picture, but oocyst numbers may be grossly misleading when considered in isolation. Healthy cattle may pass more than 1 million oocysts per gram of faeces, whereas in animals dying of coccidiosis the count may be less than 10,000 oocysts per gram. For instance, high counts of non-pathogenic species could mask significant numbers of the more pathogenic species and give the impression that the abundant species was the cause. A key to the identification of sporulated oocysts of cattle is provided in Chapter 4 (see Fig. 4.33 and Table 4.7).

Treatment: Outbreaks of clinical coccidiosis can appear suddenly and may prove troublesome to resolve as they often occur on heavily stocked farms, particularly where good husbandry and management are lacking. If deaths are occurring, early confirmation of the diagnosis is vital. Affected cattle should be medicated and moved to uncontaminated pens or pasture as soon as possible.

Normally all cattle in a group should be treated, as even those showing no symptoms are likely to be infected. For calves, this would normally be in the form of a single oral drench with either diclazuril or toltrazuril, in countries where these products are both available and licensed for use. Decoquinate can be administered in feed, bearing in mind that not all animals may consume the feed, especially severely affected animals that may be off their feed and dehydrated. Where these products are not available or licensed, then treatment with a sulphonamide such as sulphadimidine or sulphamethoxypyridazine can be considered.

Severely infected animals that are diarrhoeic and dehydrated may require oral or intravenous rehydration. Coccidiosis-infected animals may also have concurrent bacterial or parasitic infections that need to be diagnosed and treated with either antibacterial or anthelmintic treatments.

Where non-specific symptoms of weight loss or ill-thrift are present, it is important to investigate all potential causes and seek laboratory confirmation. If coccidiosis is considered significant, much can be done through advice on management and instigation of preventive measures. Batch rearing of animals of similar ages limits the build-up and spread of oocysts and allows targeting of treatment to susceptible age groups during the danger periods.

Prevention and control: Coccidial infections can be reduced through avoidance of overcrowding and stress, and attention to hygiene. Raising of food and water troughs, for example, can help avoid contamination and thus reduce levels of infection. Young animals should be kept off heavily contaminated pastures when they are most susceptible.

The control of outbreaks of coccidiosis is a balance between controlling the disease and allowing the development of protective immunity against subsequent oocyst challenge. It should also be remembered that not all species are pathogenic and that immunity is species-specific so that exposure to one coccidial species does not confer resistance to another species. Also, exposure to a single infective challenge may confer strong protective immunity with some species of coccidia, while with others repeated exposure may be required before full protective immunity is acquired.

The timing of any treatment intervention is therefore crucial in both preventing severe disease outbreaks and at the same time allowing protective immunity to develop through adequate parasite exposure. To achieve this it is important to understand the epidemiology of coccidial infections in relation to the different ruminant hosts and the differing systems of production around the world. Using this approach it is possible to identify within management and husbandry systems periods of stress that may precipitate outbreaks of disease. While appropriate disease prevention methods should always be considered and instigated wherever possible, a more strategic approach to anticoccidial treatment should be applied based on an assessment of disease risks.

Eimeria bovis

Predilection site: Small and large intestine

Eimeria zuernii

Predilection site: Small and large intestine

For more details see Parasites of the large intestine.

Eimeria alabamensis

Predilection site: Small and large intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are usually ovoid, 13–24 by 11–16 μm (mean 18.9 by 13.4 μm) with a smooth colourless wall with no micropyle, polar body or residuum. Sporocysts are ellipsoidal, 10–16 by 4–6 μm with a tiny Stieda body and a sporocyst residuum. The sporozoites lie lengthwise head to tail in the sporocysts and have one to three clear globules. First-generation meronts are usually ovoid, 7–9 by 5.5–8.0 μm, containing 8–16 merozoites. Second-generation meronts are 9–12 by 6–9 μm, ovoid or ellipsoidal in shape and contain 18–26 merozoites.

Life cycle: The life cycle is typically coccidian with the developmental stages occurring in the nucleus of epithelial cells. Sporozoites penetrate the intestinal cells as early as day 2 after infection, and meronts are visible in the nucleus 2–8 days post infection. Parasitised cells are usually those at the tips of the villi and multiple invasion of the nucleus may occur. Two generations of meronts have

been found: mature first-generation meronts are seen 2–7 days post infection, and mature second-generation meronts 4–7 days post infection. The gametocytes are found in the posterior third of the small intestine and may also occur in the mucous membrane of the caecum and colon in heavy infections. Oocysts may be seen in the cells of the lower ileum as early as 6 days post infection. The prepatent period is 6–11 days, with a patent period of 1–13 days. Sporulation takes 4–8 days.

Geographical distribution: Presumed worldwide, mainly Europe

Pathogenesis: Particularly pathogenic, attacking the epithelial cells of the jejunum, ileum and, in heavy infections, the caecum and colon.

Pathology: Infection causes catarrhal enteritis in the jejunum, ileum and caecum with petechial haemorrhages. Histologically, there is necrotic inflammation and destruction of epithelial cells. There is an inflammatory response consisting predominantly of mononuclear cells with a few eosinophils and neutrophils. Numerous meronts are seen in the nuclei of villous epithelial cells, with occasional meronts in the upper colon. The mesenteric lymph nodes are enlarged, and parasite stages have been observed in the lymph nodes.

Clinical signs: Diarrhoea in calves recently turned out on to permanent paddocks. The calves become depressed and reluctant to rise. From 8 days after turnout, 850,000 to several million oocysts per gram faeces are excreted. Growth rate of the calves is adversely affected. Morbidity ranges from 5 to 100% (average 64%) but mortality is generally low.

Treatment: Sulphonamides can be used to treat infection. Decoquinate has a prophylactic action against the parasite.

Control: Where infection is suspected to be due to oocysts overwintering on the pasture, the grazing land should be rotated to ensure that calves are not turned out on to potentially heavily infected pasture.

Infection with the following species of coccidia present in the small intestine is not usually associated with clinical signs. Specific treatment and control measures are not usually indicated for these species, although they often present as mixed infections. Differentiation is based on oocyst morphology (see Tables 2.3 and 4.7 and Fig. 4.33).

Eimeria aubernensis

Predilection site: Small intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are elongated, ovoid, 20–46 by 20–25 μm (mean 38.4 × 23.1 μm), yellowish-brown, with a smooth or heavily granulated wall with a micropyle and polar granule, but no oocyst residuum. Sporocysts are elongate ovoid, almost ellipsoidal, 15–23 by 6–11 μm with a Stieda body and a residuum. The sporozoites are elongate, almost comma-shaped, 15–18 by 3–5 μm, lie lengthwise head to tail in the sporocysts, and have a clear globule at the large end, and sometimes one to two small globules arranged randomly.

Life cycle: The first-generation meronts occur throughout the small intestine deep in the lamina propria near the muscularis mucosae. Second-generation meronts and gamonts occur in the subepithelium in the distal part of the villi, in the jejunum and ileum. The macrogamonts are about 18 μm in diameter when mature. The prepatent period is 16–24 days and the patent period is usually 2–8 days. The sporulation time is 2–3 days.

Geographical distribution: Worldwide

Eimeria brasiliensis

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are ellipsoidal, yellowish-brown, 33–44 by 24–30 μm (mean 37 × 27 μm) with a micropyle covered by a distinct polar cap. Polar granules may also be present, but there is no oocyst residuum. Sporocysts are ellipsoidal, 16–22 by 7–10 μm, with a residuum and sometimes a small dark Stieda body. The sporozoites are elongate and lie lengthwise head to tail in the sporocysts and have a large posterior and a small anterior clear globule.

Life cycle: Details of the life cycle are unknown. The sporulation time is 12–14 days.

Geographical distribution: Worldwide

Eimeria bukidnonensis

Predilection site: Small and large intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are pear-shaped or oval, tapering at one pole, 47–50 by 33–38 μm (mean 48.6 × 35.4 μm), yellowish-brown, with a thick radially striated wall and micropyle. A polar granule may be present but there is no oocyst residuum. Sporozoites are elongate and lie lengthwise head to tail in the sporocysts with a clear globule at each end.

Life cycle: Details of the life cycle are unknown. The sporulation time is 4–7 days.

Geographical distribution: Worldwide

Eimeria canadensis

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are ovoid or ellipsoidal, colourless, or pale yellow, 28–37 by 20–22 μm (mean 32.5 × 23.4 μm) with an inconspicuous micropyle, one or more polar granules and an oocyst residuum. Sporocysts are elongate ovoid, 15–22 by 6–10 μm, with an inconspicuous Stieda body and a residuum. The sporozoites are elongate, lie lengthwise head to tail in the sporocysts and have two to three clear globules each.

Life cycle: Details of the life cycle are unknown. The sporulation time is 3–4 days.

Geographical distribution: Worldwide

Eimeria cylindrica

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are elongated, cylindrical, 16–27 by 12–15 μm (mean 23.3 × 12.3 μm) with a colourless smooth wall, no micropyle and no oocyst residuum. Sporocysts are elongate ellipsoidal, 12–16 by 4–6 μm with an inconspicuous or absent Stieda body and a residuum. The sporozoites are elongate, lie lengthwise head to tail in the sporocysts and have one or more rather indistinct clear globules.

Life cycle: Details of the life cycle are unknown. Both the prepatent and patent periods are 10 days. The sporulation time is 2–3 days.

Geographical distribution: Worldwide

Eimeria ellipsoidalis

Predilection site: Small intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are ellipsoidal to slightly ovoid, colourless, 20–26 by 12–17 μm (mean 23.4 × 15.9 μm) with no discernible micropyle, polar granule or oocyst residuum. Sporocysts are ovoid, 11–17 by 5–7 μm and may have a conspicuous Stieda body, each with a residuum. The sporozoites are elongate, 11–14 by 2–3 μm, and lie head to tail in the sporocysts and have two clear globules.

Life cycle: Mature gamonts lie in the terminal section of the ileum, and are seen 10 days after infection in the epithelial cells near the bottom of the crypts. The prepatent period is 8–13 days. The sporulation time is 3 days.

Geographical distribution: Worldwide

Eimeria pellita

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are egg-shaped, with a very thick brown wall with evenly distributed protruberances, 36–41 by 26–30 μm (mean 40 × 28 μm), with a micropyle and polar granule consisting of several rod-like bodies but no oocyst residuum. Sporocysts are ellipsoidal, 17–20 by 7–9 μm (mean 18.5 × 8 μm), each with a small Stieda body and a small sporocyst residuum. The sporozoites are elongate and each has two clear globules.

Life cycle: Details of the life cycle are unknown. The sporulation time is 10–12 days.

Geographical distribution: Presumed worldwide

Eimeria subspherica

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are round or subspherical, colourless, 9–14 by 8–13 μm (mean 11 × 10.4 μm), with no micropyle, polar granule or oocyst residuum. Sporocysts are elongate ovoid, 6–10 by 2–5 μm, each with a small Stieda body, but usually lacking a sporocyst residuum. The sporozoites are elongate and lie lengthwise head to tail in the sporocysts and each has a clear globule at the large end.

Life cycle: Details of the life cycle are unknown. The prepatent period is 7–18 days and the patent period is 4–15 days. The sporulation time is 4–5 days.

Geographical distribution: Worldwide

Eimeria wyomingensis

Predilection site: Unknown

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: The oocysts are ovoid, yellowish-brown, 37–45 by 26–31 μm (mean 40.3 × 28.1 μm), with a thick wall, a wide micropyle but no polar granule or oocyst residuum. Sporocysts are ellipsoidal with narrowed ends (18 × 9 μm) and each has a tiny Stieda body and the residuum is generally small or absent. The sporozoites are elongate, about 7–8 by 5 μm, and lie lengthwise head to tail in the sporocysts, each with a large clear globule.

Life cycle: Details of the life cycle are unknown. The prepatent period is 13–15 days and the patent period 1–7 days. The sporulation time is 5–7 days.

Geographical distribution: Worldwide

Cryptosporidium parvum

Predilection site: Small intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Cryptosporidiidae

Description: Mature oocysts are ovoid or spheroid, 5.0 by 4.5 µm (range 4.6–5.4 × 3.8–4.7 µm); length/width ratio 1.19.

Hosts: Cattle, sheep, goat, horse, deer, human

Geographical distribution: Worldwide

Pathogenesis: Cryptosporidiosis is common in young calves, although the pathogenesis of infection is not clear. It is remarkable in that, unlike other members of the Eimeriidae, *Cryptosporidium* does not enter the cells of the host and lacks host specificity so that cross-infection can occur between domestic animals and humans.

Clinical signs: Clinically the disease is characterised by anorexia and diarrhoea, often intermittent, which may result in poor growth rates.

Diagnosis: Oocysts may be demonstrated using Ziehl–Neelsen stained faecal smears in which the sporozoites appear as bright red granules (Fig. 8.10). Speciation of *Cryptosporidium* is difficult, if not impossible, using conventional techniques. A range of molecular and immunological techniques has been developed that includes the use of immunofluorescence (Fig. 8.11) or ELISA. More recently, DNA-based techniques have been used for the molecular characterisations of *Cryptosporidium* species.

Pathology: The meronts and gamonts develop in a parasitophorous envelope, apparently derived from the microvilli and so the cell disruption seen with other coccidia does not apparently occur. However, mucosal changes are obvious in the ileum where there is stunting, swelling and eventually fusion of the villi. This has a marked effect on the activity of some of the membrane-bound enzymes.

Epidemiology: A variety of mammals act as hosts to *C. parvum* but little is known of the importance of their involvement in transmitting infection to, or maintaining infection in, domestic livestock.

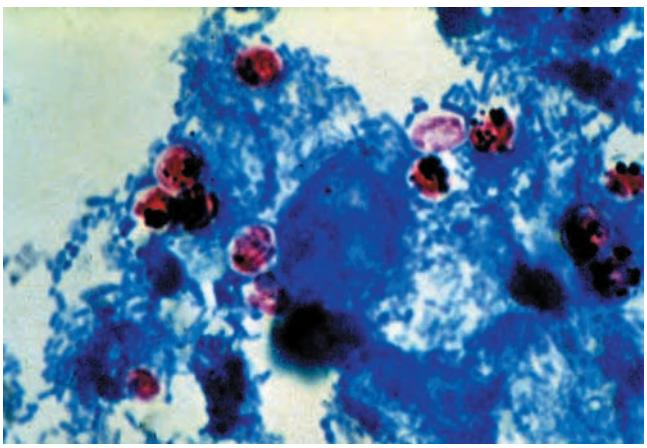

Figure 8.10 Oocysts of *Cryptosporidium parvum* (Ziehl–Neelsen stain).

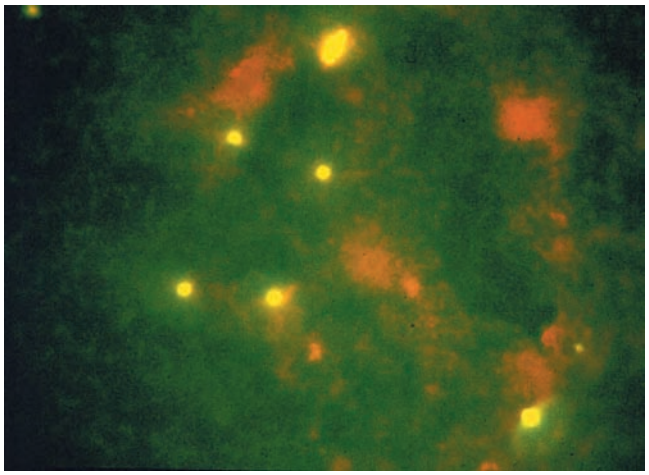

Figure 8.11 Oocysts of *Cryptosporidium parvum* (immunofluorescent antibody test).

In young calves infection appears to be age related, with seasonal peaks of disease reported to coincide with birth peaks in spring and autumn. The first calves to be born often become infected without showing clinical signs but become sources of infection for calves that follow. Infection spreads rapidly, and later-born calves can become so heavily infected that clinical disease results. In many instances where *Cryptosporidium* is diagnosed in animals, it appears that infections usually originate from the same host species. The primary route of infection is mainly direct animal to animal via the faecal–oral route. Thus in calves, for example, overcrowding, stress of early weaning, transport and marketing, together with low levels of hygiene, will increase the risk of clinical infections. In lambs, chilling due to adverse weather conditions in the neonatal period, intercurrent infections or nutritional or mineral deficiencies could exacerbate or increase the likelihood of disease. Infection in these cases is likely to occur via grooming, nuzzling, coprophagy, or faecal soiling by direct contact with infected animals. Infection may also occur indirectly through consumption of contaminated foods or environmental sources, including pasture and water.

Treatment: There is no known treatment, although spiramycin may be of some value. The infection is difficult to control since the oocysts are highly resistant to most disinfectants except formol-saline and ammonia. Halofuginone is available for the prevention of cryptosporidiosis in calves at a dose rate of 1 mg per 10 kg. Symptomatic treatment may be given in the form of antidiarrhoeals and fluid replacement therapy.

Control: Good hygiene and management are important in preventing disease from cryptosporidiosis. Feed and water containers should be high enough to prevent faecal contamination. Young animals should be given colostrum within the first 24 hours of birth and overstocking and overcrowding should be avoided. Dairy calves should be either isolated in individual pens or kept in similar age groups and cleaned out daily. On calf-rearing farms with recurrent problems, the prophylactic use of halofuginone can be considered by treating for 7 days consecutively commencing at 24–48 hours after birth.

Notes: Recent molecular characterisations have shown that there is extensive host adaptation in *Cryptosporidium* evolution, and many mammals or groups of mammals have host-adapted *Cryptosporidium* genotypes, which differ from each other in both DNA sequences

and infectivity. These genotypes are now delineated as distinct species and include, in cattle, *C. parvum*, *C. bovis* (also termed the bovine genotype or genotype 2), *C. ryanae* and *C. ubiquitum*.

Giardia intestinalis

Synonyms: *Giardia duodenalis, Giardia lamblia, Lamblia lamblia*

Predilection site: Small intestine

Phylum: Fornicata

Class: Trepomonadea

Family: Giardiidae

Description: The trophozoite has a pyriform to ellipsoidal, bilaterally symmetrical body, 12–15 µm long by 5–9 µm wide (Fig. 8.12). The dorsal side is convex and there is a large sucking disc on the ventral side. There are two anterior nuclei, two slender axostyles, eight flagellae in four pairs and a pair of darkly staining median bodies. The median bodies are curved bars resembling the claws of a hammer. Cysts are ovoid, 8–12 by 7–10 µm and contain four nuclei (Fig. 8.13).

Hosts: Human, cattle, sheep, goat, pig, horse, alpaca, dog, cat, guinea pig, chinchilla

Geographical distribution: Worldwide

Pathogenesis: Infections in cattle are often asymptomatic but have been reported to cause diarrhoea in young calves.

Clinical signs: When disease does occur, the signs often include chronic pasty diarrhoea, weight loss, lethargy and failure to thrive. The diarrhoea may be continuous or intermittent.

Diagnosis: *Giardia* cysts can be detected in faeces by a number of methods. Traditional methods of identification involve direct examination of faecal smears, or faecal concentration by formalin-ethyl acetate or zinc sulphate methods and subsequent microscopic examination. It is generally recommended that three consecutive samples be examined as cysts are excreted intermittently.

Pathology: There may be villous atrophy, crypt hypertrophy and an increased number of intraepithelial lymphocytes. Trophozoites may be seen between villi, attached by their concave surface to the brush border of epithelial cells.

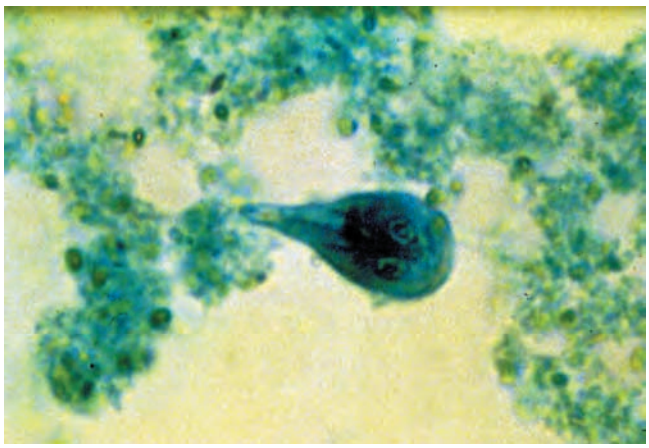

Figure 8.12 *Giardia intestinalis* trophozoite.

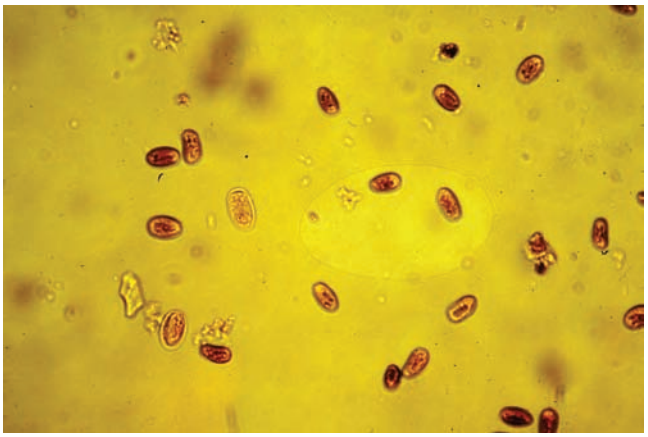

Figure 8.13 *Giardia* cysts.

Epidemiology: Molecular studies have revealed a substantial level of genetic diversity in *G. intestinalis* isolates. Human isolates fall into two major groups (assemblages A and B) with a wide host range in other mammals and some separate species names may be applicable. Other assemblages may also represent distinct species. Limited epidemiological studies suggest that in animal isolates direct animal-to-animal contact and faecal soiling is the most likely method of transmission, although water contamination can also be considered as a possible route. The incidence of these parasites varies but can be assumed to be higher in some species than has been reported. Studies in Canada and the USA indicate levels of infection in cattle of up to 20% in clinically normal animals and 100% infection rates in young diarrhoeic calves.

Treatment: There is no recommended treatment for infection in calves. Several benzimidazole anthelmintics (e.g. albendazole, fenbendazole) are effective and may prove to be of benefit.

Control: As infection is transmitted by the faecal–oral route, good hygiene and prevention of faecal contamination of feed and water is essential.

Notes: The parasite is important because of water-borne outbreaks that have occurred in human populations. Phylogenetic data suggest that *G. intestinalis* is a species complex composed of several species that are host-specific. There is still some controversy over the classification of *Giardia* spp. The current molecular classification places isolates into eight distinct assemblages. Some authors give separate specific names to *Giardia* organisms isolated from cattle, for example *Giardia enterica* (Assemblage B), *Giardia bovis* (Assemblage E), although species specificity of many isolates is unknown.

LARGE INTESTINE

Oesophagostomum radiatum

Common name: Nodular worm

Predilection site: Large intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Strongyloidea

Description, gross: Adults are slender whitish worms, 1–2 cm in size, with males 12–17 mm and females 16–22 mm long.

Description, microscopic: The cuticle forms a rounded mouth collar, and large cephalic vesicle, constricted around the middle by a shallow annular groove (see Fig. 1.43). External leaf crowns are missing and the internal ring consists of 38–40 small triangular denticles. Cervical papillae are present, just posterior to the cervical groove. The male bursa is well developed. The egg is a medium-sized (75–98 × 46–54 μm), regular, broad ellipse with barrel-shaped side walls and rounded poles, and contains 16–32 blastomeres when passed in the faeces. The colourless chitinous shell is thin with a smooth surface. Infective larvae (L_3) have long filamentous tails, 32 gut cells and a rounded head.

Hosts: Cattle, buffalo

Geographical distribution: Worldwide

Pathogenesis: In *O. radiatum* infections in cattle, the pathogenic effect is attributed to the nodules (up to 5.0 mm in diameter) in the intestine and it is one of the most damaging worms to cattle when present in high numbers, with >200 adult worms in calves and >1000 adults in adult cattle sufficient to produce clinical signs. In the later stages of the disease, anaemia and hypoalbuminaemia develop due to the combined effects of protein loss and leakage of blood through the damaged mucosa.

Clinical signs: In acute infections, there is anaemia, oedema and diarrhoea.

Diagnosis: This is based on clinical signs and postmortem examination. The presence of pea-shaped nodules in the intestinal wall on postmortem is indicative of nodular worm infection. In the chronic disease, eggs are present and L_3 can be identified following faecal culture.

Pathology: On postmortem examination, animals may be pale from anaemia, and oedematous from hypoproteinaemia. Colonic lymph nodes are enlarged and the mucosa of the colon is grossly thickened and folded by oedema and increased mixed inflammatory cell infiltrates in the lamina propria. Colonic submucosal lymphoid follicles are large and active. Effusion of tissue fluid and blood cells may be evident through small leaks between cells, or from erosions in glands or on the surface. Although repeated exposure to infective larvae may result in the accumulation of large numbers of fourth-stage worms in nodules, formation of nodules has little pathogenic significance in cattle.

Epidemiology: It is not yet known if hypobiosis occurs in *O. radiatum*. It is also capable of overwintering on pasture as L_3. In tropical and subtropical areas *O. radiatum* in cattle is especially important. Cattle develop a good immunity, partly due to age and partly to previous exposure so that it is primarily a problem in weaned calves.

Treatment: Anthelmintic therapy with broad-spectrum anthelmintics (benzimidazoles, levamisole and avermectins/milbemycins) is highly effective.

Control: While not generally considered highly pathogenic, a combination of strategic dosing with anthelmintics and pasture management, as used in the control of other nematodes, will also help to control *O. radiatum*.

Trichuris globulosa

Synonym: *Trichocephalus globulosa*

Common name: Whipworms

Predilection site: Large intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichuroidea

Description, gross: The adults are long white worms (about 4.0–7.0 cm) with a thick broad posterior end, tapering rapidly to a long filamentous anterior end that is characteristically embedded in the mucosa.

Description, microscopic: The male tail is coiled and possesses a single spicule in a protrusible sheath. The sheath is covered with minute spines and bears a spherical appendage; the female tail is merely curved. The characteristic medium-sized eggs are lemon-shaped, 70–80 by 30–40 μm, with a thick smooth shell and a conspicuous protruding transparent polar plug (operculum) at both ends. The content of the egg is granular, there being no blastomeres. In the faeces these eggs appear yellowish or brown in colour.

Hosts: Cattle, occasionally sheep, goat, camel and other ruminants

Geographical distribution: Worldwide

Pathogenesis: Most infections are light and asymptomatic. Occasionally, when large numbers of worms are present they cause a diphtheritic inflammation of the caecal mucosa.

Clinical signs: Despite the fact that ruminants have a high incidence of light infections, the clinical significance of this genus, especially in ruminants, is generally negligible, although isolated outbreaks have been recorded.

Diagnosis: Since the clinical signs are not pathognomonic, diagnosis may depend on finding numbers of lemon-shaped *Trichuris* eggs in the faeces. Egg output is often low in *Trichuris* infections.

Pathology: In severe cases, the mucosa of the large intestine is inflamed and haemorrhagic with ulceration and formation of diphtheritic membranes.

Epidemiology: The most important feature is the longevity of the eggs, which may survive for 3 or 4 years. On pasture this is less likely since the eggs tend to be washed into the soil.

Treatment: In ruminants the benzimidazoles, the avermectins/milbemycins or levamisole by injection are very effective against adult *Trichuris*, but less so against larval stages.

Control: Prophylaxis is rarely necessary in ruminants.

Notes: The adults are usually found in the caecum but are only occasionally present in sufficient numbers to be clinically significant.

Trichuris discolor

Common name: Whipworms

Predilection site: Large intestine

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichuroidea

Description, gross: Worms are similar to *T. globulosa* but the females are yellow–orange in colour.

Description, microscopic: Eggs measure about 65 by 30 μm.

Hosts: Cattle, buffalo, occasionally sheep, goat

Geographical distribution: Europe, Asia, USA

Details of the life cycle, pathogenesis, clinical signs, diagnosis, pathology, epidemiology, treatment and control are as for *T. globulosa*.

Homalogaster paloniae

Predilection site: Large intestine

Phylum: Platyhelminthes

Class: Trematoda

Family: Gastrodiscidae

Description, gross: The body is divided into two with a large anterior region and small cylindrical posterior region.

Hosts: Buffalo and cattle

Intermediate hosts: Water snails

Geographical distribution: Asia, Australasia

Pathogenesis: Generally considered to be non-pathogenic

Treatment and control: Not required

Eimeria bovis

Predilection site: Small and large intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are ovoid or subspherical, colourless, 23–34 by 17–23 μm (mean 27.7 × 20.3 μm) and have a smooth wall with an inconspicuous micropyle, no polar granule or oocyst residuum (Fig. 8.14). Sporocysts are elongate ovoid, 13–18 by 5–8 μm, and have an inconspicuous Stieda body and a sporocyst residuum. The sporozoites are elongate and lie lengthwise head to tail in the sporocysts and usually have a clear globule at each end.

Life cycle: There are two asexual generations. The first-generation meronts are in the endothelial cells of the lacteals of the villi in the posterior half of the small intestine, mature at 14–18 days after infection and can be seen grossly as whitish specks in the mucosa. Second-generation meronts occur in the epithelial cells of the caecum and colon, but may extend into the last metre of the small intestine in heavy infections. The sexual stages generally occur in the caecum and colon, but may extend into the ileum in heavy infections; they appear 17 days after infection. The prepatent period is 16–21 days and the patent period usually 5–15 days. The sporulation time is 2–3 days.

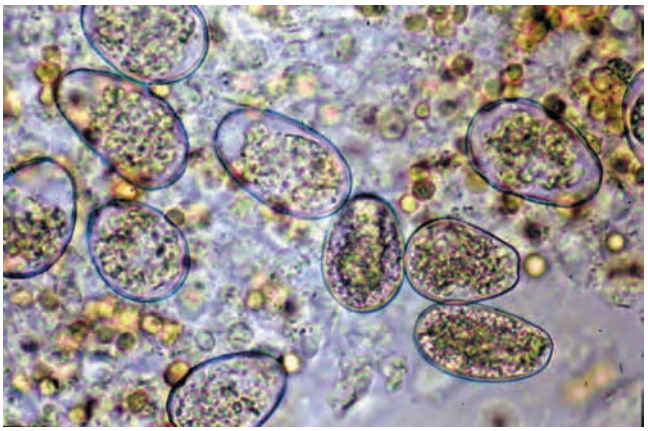

Figure 8.14 Oocysts of *Eimeria bovis*.

Geographical distribution: Worldwide

Pathogenesis: Particularly pathogenic, attacking the caecum and colon, causing mucosal sloughing and haemorrhage.

Pathology: The most severe pathological changes occur in the caecum, colon and terminal 30 cm of the ileum, and are due to the gamonts. The mucosa appears congested, oedematous and thickened with petechiae or diffuse haemorrhages. The gut lumen may contain a large amount of blood. Later in the infection the mucosa is destroyed and sloughs away. The submucosa may also be lost. If the animal survives, both the mucosa and submucosa regenerate.

Clinical signs: Severe enteritis and diarrhoea, or dysentery with tenesmus in heavy infections. The animal may be pyrexic, weak and dehydrated, and if left untreated loses condition and may die.

Epidemiology: Disease is dependent on conditions that precipitate a massive intake of oocysts, such as overcrowding in unhygienic yards or feedlots. It may also occur at pasture where livestock congregate around water troughs.

Eimeria zuernii

Predilection site: Small and large intestine

Phylum: Apicomplexa

Class: Conoidasida

Family: Eimeriidae

Description: Oocysts are subspherical, colourless, 15–22 by 13–18 μm (mean 17.8 × 15.6 μm), with no micropyle or oocyst residuum (Fig. 8.15). Sporocysts are ovoid, 7–14 by 4–8 μm, each with a tiny Stieda body, and a sporocyst residuum is usually absent. The sporozoites are elongate and lie head to tail in the sporocysts; each has a clear globule at the large end.

Life cycle: First-generation meronts are giant meronts and are found in the lamina propria of the lower ileum and mature at 14–16 days after infection, visible as whitish specks in the mucosa; second-generation meronts occur in the epithelial cells of the caecum and proximal colon from about 16 days after infection. The sexual stages generally occur within epithelial cells of the caecum and colon, but may extend into the ileum in heavy infections, appearing 16 days

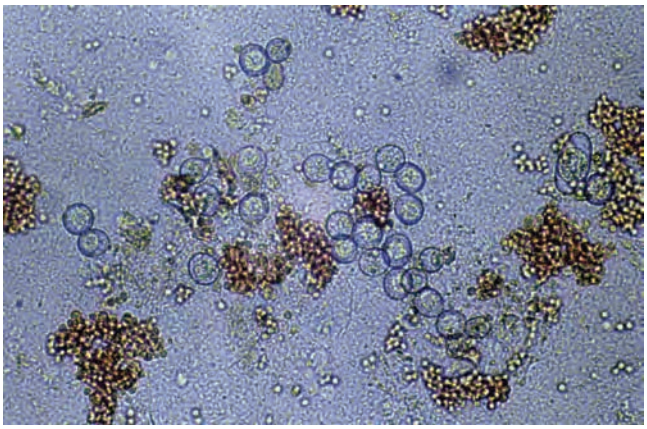

Figure 8.15 Oocysts of *Eimeria zuernii*.

after infection. The prepatent period is 15–17 days and the patent period usually 5–17 days. The sporulation time is 2–10 days.

Geographical distribution: Worldwide

Pathogenesis: This is the most pathogenic species causing haemorrhagic diarrhoea through erosion and destruction of large areas of the intestinal mucosa. *Eimeria zuernii* is the most common cause of 'winter coccidiosis', which occurs primarily in calves during or following cold or stormy weather in the winter months. The exact aetiology of this syndrome is uncertain.

Pathology: Generalised catarrhal enteritis involving both the large and small intestines is present. The lower small intestine, caecum and colon may be filled with semi-fluid haemorrhagic material. Large or small areas of the intestinal mucosa may be eroded and destroyed. The mucous membrane may be thickened with irregular whitish ridges in the large intestine or smooth dull-grey areas in the small intestine or caecum. Diffuse haemorrhages are present in the intestines in acute cases, and petechial haemorrhages are seen in milder cases.

Clinical signs: In acute infections, *E. zuernii* causes haemorrhagic diarrhoea of calves. At first, the faeces are streaked with blood, but as the diarrhoea becomes more severe, bloody fluid, clots of blood and liquid faeces are passed. Tenesmus and coughing can result in the diarrhoea being spurted out up to 2–3 m. The animal's hindquarters are smeared with red diarrhoea. Secondary infections, especially pneumonia, are common. The acute phase may continue for 3–4 days. If the calf does not die in 7–10 days, it will probably recover.

Eimeria zuernii may also cause a more chronic form of disease. Diarrhoea is present, but there is little or no blood in the faeces. The animals are emaciated, dehydrated, weak and listless. Their coats are rough, their eyes sunken and their ears droop.

Treatment: Treatment of both *E. bovis and E. zuernii* is with a sulphonamide, such as sulphadimidine or sulphamethoxypyridazine, given orally or parenterally and repeated at half the initial dose level on each of the next 2 days. Alternatively, decoquinate in feed, or diclazuril and toltrazuril given orally may be used.

Control: Prevention is based on good management; in particular, feed troughs and water containers should be moved regularly and bedding kept dry.

Flagellate protozoa

The life cycle of the following flagellate protozoa is similar for all species found in cattle. The trophozoites reproduce by longitudinal binary fission, no sexual stages are known and there are no cysts. Transmission is thought to occur by ingestion of trophozoites from faeces. All are considered to be non-pathogenic and are generally only identified from smears taken from the large intestine of fresh carcasses.

Tetratrichomonas buttreyi

Synonym: *Trichomonas buttreyi*

Predilection site: Caecum, colon

Phylum: Parabasalia

Class: Trichomonadea

Family: Trichomonadidae

Description: The body is ovoid or ellipsoidal, 4–7 by 2–5 μm (mean 6 × 3 μm) in size. Cytoplasmic inclusions are frequently present. There are three or four anterior flagella, which vary in length from a short stub to more than twice the length of the body, and each ends in a knob or spatulate structure. The undulating membrane runs the full length of the body and has three to five undulations ending in a posterior free flagellum. The accessory filament is prominent, and the costa relatively delicate. The axostyle is relatively narrow, has a spatulate capitulum and extends 3–6 μm beyond the body. There is no chromatic ring at its point of exit. A pelta is present. The nucleus is frequently ovoid (2–3 × 1–2 μm) but is variable in shape and has a small endosome.

Hosts: Cattle, pig

Geographical distribution: Worldwide

Tritrichomonas enteris

Predilection site: Colon

Phylum: Parabasalia

Class: Trichomonadea

Family: Trichomonadidae

Description: The body measures 6–12 by 5–6 μm and there are three anterior flagella of equal length, which arise from a single blepharoplast. The flagellum at the edge of the undulating membrane is single and lacks an accessory filament. The undulating membrane extends three-quarters of the body length and a free flagellum extends beyond the undulating membrane. The axostyle is straight and slender, bending around the nucleus to give a spoon shape and extending at most one-quarter of the body length beyond the body.

Geographical distribution: Worldwide

Tetratrichomonas pavlovi

Synonyms: *Trichomonas bovis, Trichomonas pavlovi*

Predilection site: Caecum

Phylum: Parabasalia

Class: Trichomonadea

Family: Trichomonadidae

Description: The body is pyriform and is usually 11–12 by 6–7 μm. It has four anterior flagella, which are about the same length as the body. The undulating membrane is well developed and has two to four waves that extend almost to the posterior end of the body. There is a posterior free flagellum, an accessory filament and a costa. The nucleus is round or ovoid. The axostyle is slender, broadening to form a capitulum at the anterior end.

Geographical distribution: Unknown

Retortamonas ovis

Predilection site: Large intestine

Phylum: Fornicata

Class: Retortamonadea

Family: Retortamonadorididae

Description: Trophozoites are pyriform and average 5.2 by 3.4 μm. There is a large cytostome near the anterior end containing a cytostomal fibril that extends across the anterior end and posteriorly along each side. An anterior flagellum and a posterior trailing flagellum emerge from the cytostomal groove. Cysts are pyriform and ovoid, containing one or two nuclei and retain the cytostomal fibril.

Geographical distribution: Worldwide

Buxtonella sulcata

Predilection site: Large intestine

Phylum: Ciliophora

Class: Litostomatea

Family: Pycnotrichidae

Description: The body is ovoid, 100 by 72 μm, and uniformly ciliated with a prominent curved groove bordered by two ridges running from end to end with a cyathostome at the anterior end, and an oval or bean-shaped macronucleus, 28 by 14 μm in size.

Geographical distribution: Worldwide

Parasites of the respiratory system

Mammomonogamus laryngeus

Synonym: *Syngamus laryngeus*

Common name: Gapeworm

Predilection site: Larynx

Phylum: Nematoda

Class: Secernentea

Superfamily: Strongyloidea

Description, gross: The worms are reddish in appearance and about 0.5–2 cm long. The females and males are found in permanent copulation. The buccal capsule lacks a cuticular crown.

Description, microscopic: Eggs are ellipsoid, 42–45 by 75–85 μm, with no operculum at either end.

Hosts: Cattle, buffalo, goat, sheep, deer, rarely human

Geographical distribution: Asia, Central Africa, South America and Caribbean islands

Pathogenesis: *Mammomonogamus laryngeus* in not very pathogenic for cattle. Worms are attached to the mucosa of the larynx and may cause laryngitis and bronchitis.

Clinical signs: Infections are usually asymptomatic but affected animals may cough and lose condition. Calves may develop bronchitis and aspiration pneumonia has been recorded.

Diagnosis: This is based on clinical signs and the finding of eggs in the faeces. Disease is probably best confirmed by postmortem examination of selected cases, when reddish worms will be found attached to the tracheal mucosa. The infected trachea often contains an increased amount of mucus.

Pathology: Not described

Epidemiology: Unknown

Treatment: Successful treatment has not been reported. Benzimidazoles and macrocyclic lactones are likely to be effective.

Control: No preventive or control measures have been described.

Notes: This genus, closely related to *Syngamus*, is parasitic in the respiratory passages of mammals. Infection has been reported in humans, causing a laryngo-pharyngeal syndrome.

Mammomonogamus nasicola

Synonyms: *Syngamus nasicola, Syngamus kingi*

Predilection site: Nasal cavities

Phylum: Nematoda

Class: Secernentea

Superfamily: Strongyloidea

Description, gross: The worms are reddish in appearance and about 0.5–2 cm long. Males are 4–6 mm and females 11–23 mm long and found in permanent copulation. The buccal capsule lacks a cuticular crown.

Description, microscopic: Eggs are ellipsoid, 54–98 μm, with no operculum at either end.

Hosts: Sheep, goat, cattle, deer

Geographical distribution: Central and South America, Central Africa, Caribbean islands

For more details on this species see Chapter 9.

Dictyocaulus viviparus

Common names: Bovine lungworm, husk, hoose, verminous pneumonia, parasitic bronchitis

Predilection site: Bronchi, trachea

Phylum: Nematoda

Class: Secernentea

Superfamily: Trichostrongyloidea

Description, gross: The adults are slender thread-like worms; males measure around 4.0–5.5 cm and females 6–8 cm in length.

Description, microscopic: First-stage larvae present in fresh faeces are about 300–450 µm in length and 25 µm in width, the intestinal cells containing numerous brownish chromatin granules (see Fig. 1.36). The head is rounded, there being no protruding anterior knob (cf. *D. filaria* in sheep and goats). The oesophagus is simple strongyloid and the tail terminates in a blunt point.

Hosts: Cattle, buffalo, deer and camel

Geographical distribution: Worldwide, but especially important in temperate climates with a high rainfall.

Pathogenesis: Dictyocaulosis is characterised by bronchitis and pneumonia and typically affects young cattle during their first grazing season on permanent or semi-permanent pastures. Pathogenesis may be divided into three phases.

1 Prepatent phase: around days 8–25. This phase starts with the appearance of larvae within the alveoli where they cause alveolitis. This is followed by bronchiolitis and finally bronchitis as the larvae become immature adults and move up the bronchi. Towards the end of this phase bronchitis develops, characterised by immature lungworms in the airways and by cellular infiltration of the epithelium. Heavily infected animals, whose lungs contain several thousand developing worms, may die from day 15 onwards due to respiratory failure following the development of severe interstitial emphysema and pulmonary oedema.
2 Patent phase: around days 26–60. This is associated with two main lesions: first, a parasitic bronchitis characterised by the presence of hundreds or even thousands of adult worms in the frothy white mucus in the lumina of the bronchi (Fig. 8.16); and second, the presence of dark-red collapsed areas around infected bronchi (Fig. 8.17). This is a parasitic pneumonia caused by the aspiration of eggs and L_1 into the alveoli.
3 Postpatent phase: around days 61–90. In untreated calves, this is normally the recovery phase after the adult lungworms have been expelled. Although the clinical signs are abating, the bronchi are still inflamed and residual lesions such as bronchial and peribronchial fibrosis may persist for several weeks or months. Eventually the bronchopulmonary system becomes completely normal and coughing ceases. However, in about 25% of animals, which have been heavily infected, there can be a flare-up of clinical signs during this phase that is frequently fatal. This is caused by one of two entities. Most commonly, there is a proliferative lesion so that much of the lung is pink and rubbery and does not collapse when the chest is opened. This, often described as 'epithelialisation', is due to the proliferation of type 2 pneumocytes on the alveoli giving the appearance of a gland-like organ. Gaseous exchange at the alveolar surface is markedly impaired and the lesion is often accompanied by interstitial emphysema and pulmonary oedema. The aetiology is unknown, but is thought to be due to the dissolution and aspiration of dead or dying worm material into the alveoli. The clinical syndrome is often termed 'postpatent parasitic bronchitis'. The other cause, usually in animals convalescing indoors, is a superimposed bacterial infection of the imperfectly healed lungs leading to acute interstitial pneumonia.

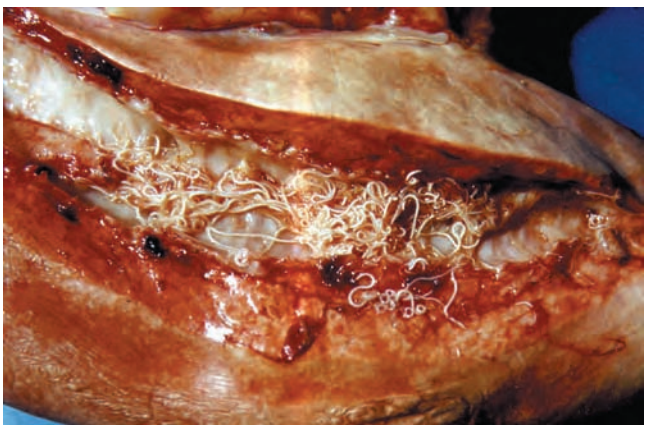

Figure 8.16 *Dictyocaulus viviparus* worms in the opened bronchi of an infected calf.

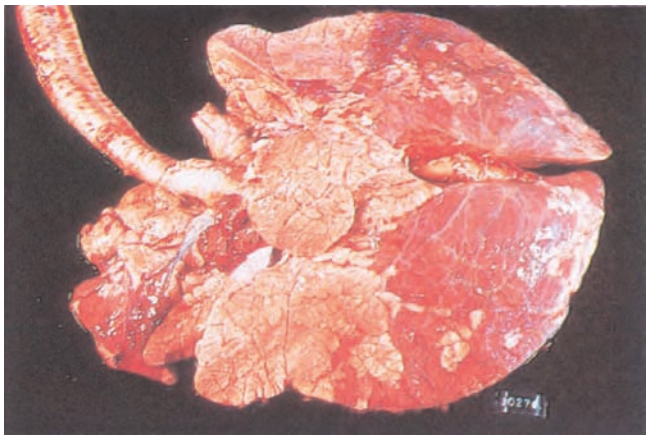

Figure 8.17 Typical distribution of pneumonic lesions of parasitic bronchitis.

Clinical signs: Within any affected group, differing degrees of clinical severity are usually apparent. Mildly affected animals cough intermittently, particularly when exercised. Moderately affected animals have frequent bouts of coughing at rest, tachypnoea (60 respirations per minute) and hyperpnoea. Frequently, squeaks and crackles over the posterior lung lobes are heard on auscultation. Severely affected animals show severe tachypnoea (80 respirations per minute) and dyspnoea and frequently adopt the classic 'air-hunger' position of mouth breathing with the head and neck outstretched. There is usually a deep harsh cough, squeaks and crackles over the posterior lung lobes, salivation, anorexia and sometimes mild pyrexia. Often the smallest calves are most severely affected.

Calves may show clinical signs during the prepatent period and occasionally a massive infection can cause severe dyspnoea of sudden onset often followed by death in 24–48 hours.

Most animals gradually recover, although complete return to normality may take weeks or months. However, a proportion of convalescing calves suddenly develop severe respiratory signs, unassociated with pyrexia, which usually terminates fatally 1–4 days later (postpatent parasitic bronchitis).

Diagnosis: Usually the clinical signs, the time of year and a history of grazing on permanent or semi-permanent pastures are sufficient to enable a diagnosis to be made.

Larvae are found (50–1000/g) only in the faeces of patent cases so that faecal samples should be obtained from the rectum of a number of affected individuals. At necropsy, worms will often be apparent in the opened bronchi and their size is diagnostic. A lungworm ELISA can be used to detect antibodies to *D. viviparus*. Seroconversion takes 4–6 weeks and titres persist for 4–7 months. Serology can be helpful in cases of reinfection husk, as it will often detect larval stages. Cross-reactivity can occur with intestinal nematode species so test sensitivity and specificity requires validation and setting of appropriate optical density (OD) cut-off values when interpreting results.

Pathology: Two phases are recognised.

1 Prepatent phase. Cellular infiltrates of inflammatory cells temporarily plug the lumina of the bronchioles and cause collapse of other groups of alveoli. This lesion is largely responsible for the first clinical signs of tachypnoea and coughing.
2 Patent phase. The bronchial epithelium is hyperplastic and heavily infiltrated by inflammatory cells, particularly eosinophils. Aspirated eggs and larvae quickly provoke dense infiltrates of polymorphs, macrophages and multinucleated giant cells around them (Fig. 8.18). There may be varying degrees of interstitial emphysema and oedema.

Epidemiology: Generally only calves in their first grazing season are clinically affected, since on farms where the disease is endemic older animals have a strong acquired immunity. In endemic areas in the northern hemisphere, infection may persist from year to year in two ways.

1 Overwintered larvae: L_3 may survive on pasture from autumn until late spring in sufficient numbers to initiate infection or occasionally to cause disease.
2 Carrier animals: small numbers of adult worms can persist in the bronchi, particularly in yearlings, until the next grazing season. Chilling of infective larvae before administration to calves will produce arrested L_5; hypobiosis at this stage has also been observed in naturally infected calves in Switzerland, Austria and Canada, although the extent to which this occurs naturally after ingestion of larvae in late autumn and its significance in the transmission of the infection has not yet been fully established.

The dispersal of larvae from the faecal pat appears to be effected by a fungus rather than by simple migration as the infective larvae are relatively inactive. This fungus, *Pilobolus*, is commonly found growing on the surface of bovine faecal pats about 1 week after deposition. The larvae of *D. viviparus* migrate in large numbers up the stalks of the fungi on to, and even inside, the sporangium or seed capsule (Fig. 8.19). When the sporangium is discharged it is projected a distance of up to 3 m in still air to land on the surrounding herbage.

Parasitic bronchitis is predominantly a problem in areas such as northern Europe that have a mild climate, high rainfall and abundant permanent grass. Outbreaks of disease occur from June until November, but are most common from July until September. It is not clear why the disease is usually not apparent until calves, turned out to graze in the spring, have been at grass for 2–5 months. One explanation is that the initial infection, acquired from the ingestion of overwintered larvae in May, involves so few worms that neither clinical signs nor immunity is produced; however, sufficient numbers of larvae are seeded on to the pasture so that by July the numbers of L_3 are sufficient to produce clinical disease. Young calves, added to such a grazing herd in July, may develop clinical disease within 2–3 weeks. An alternative explanation is that L_3 overwinter in the soil and possibly only migrate on to pasture at some point between June and October.

Although dairy or dairy-cross calves are most commonly affected, autumn-born single-suckled beef calves are just as susceptible when

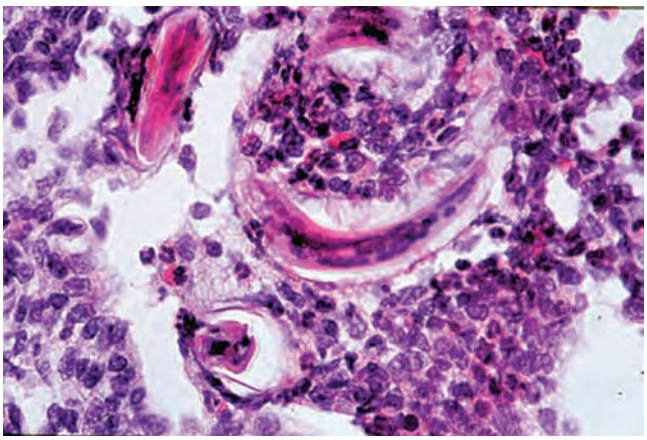

Figure 8.18 Inflammatory response due to the presence of aspirated eggs and larvae in the bronchioles and alveoli.

Figure 8.19 Larvae of *Dictyocaulus viviparus* on the fungus *Pilobolus*.

turned out to grass in early summer. Spring-born suckled beef calves grazed with their dams until housed or sold do not usually develop clinical signs, although coughing due to a mild infection is common. However, the typical disease may occur in weaned calves grazed until late autumn.

Adult cattle can be affected with parasitic bronchitis if they have not had sufficient exposure to lungworm larvae in previous years to develop adequate immunity and are subsequently grazed on heavily contaminated pastures. This is sometimes first seen as a reduction in milk yield with subsequent coughing.

In tropical countries, where disease due to *D. viviparus* may occur intermittently, the epidemiology is presumably quite different and probably depends more on pasture contamination by carrier animals, such as may occur during flooding when cattle congregate on damp high areas, rather than on the prolonged survival of infective larvae.

Treatment: The modern benzimidazoles, levamisole or the avermectin/milbemycins have been shown to be highly effective against all stages of lungworms with consequent amelioration of clinical signs. For maximum efficiency, all these drugs should be used as early as possible in the treatment of the disease. Where the disease is severe and well established in a number of calves one should be aware that anthelmintic treatment, while being the only course available, may exacerbate the clinical signs in one or more animals with a possible fatal termination. Whatever treatment is selected, it is advisable to divide affected calves into two groups, as the prognosis will vary according to the severity of the disease. Those calves which are only coughing and/or tachypnoeic are usually in the prepatent stage of the disease or have a small adult worm burden and treatment of these animals should result in rapid recovery. Calves in this category may not have developed a strong immunity and after treatment should not be returned to grazing which was the source of infection; if this is not possible, parenteral ivermectin, doramectin or moxidectin may be used since their residual effect prevents reinfection for an extended period.

Any calves which are dyspnoeic, anorexic and possibly pyrexic should be kept indoors for treatment and further observation. The prognosis must be guarded as a proportion of these animals may not recover while others may remain permanently stunted. In addition to anthelmintics, severely affected animals may require antibiotics if pyrexic and may be in need of hydration if they are not drinking.

Control: The best method of preventing parasitic bronchitis is to immunise all young calves with lungworm vaccine. This live attenuated vaccine is currently only available in parts of Europe and is given orally to calves aged 8 weeks or more. Two doses of vaccine are given at an interval of 4 weeks and, in order to allow a high level of immunity to develop, vaccinated calves should be protected from challenge until 2 weeks after their second dose. Dairy calves or suckled calves can be vaccinated successfully at grass provided the vaccine is given prior to encountering a significant larval challenge.

Although vaccination is effective in preventing clinical disease, it does not completely prevent the establishment of small numbers of lungworms. Consequently, pastures may remain contaminated, albeit at a very low level. For this reason it is important that all the calves on any farm should be vaccinated whether they go to pasture in the spring or later in the year and a vaccination programme must be continued annually for each calf crop.

Control of parasitic bronchitis in first-year grazing calves has been achieved by the use of prophylactic anthelmintic regimens either by strategic early-season treatments or by the administration of rumen boluses, as recommended in the control of bovine ostertagiosis. The danger of these measures, however, is that through rigorous control in the first grazing season, exposure to lungworm larvae is so curtailed that cattle may remain susceptible to husk during their second season; in such situations it may be advisable to consider vaccination prior to their second year at grass.

Because of the unpredictable epidemiology, the technique commonly used in ostertagiosis of 'dose and move' in midsummer does not prevent parasitic bronchitis.

Parasitic bronchitis in adult cattle Parasitic bronchitis is only seen in adult cattle under two circumstances.

1. As a herd phenomenon, or in a particular age group within a herd, if they have failed to acquire immunity through natural challenge in earlier years. Such animals may develop the disease if exposed to heavy larval challenge, as might occur on pasture recently vacated by calves suffering from clinical husk.
2. Disease is occasionally seen where an individual adult is penned in a heavily contaminated calf paddock.

The disease is most commonly encountered in the patent phase, although the other forms have been recognised. In addition to coughing and tachypnoea, a reduction in milk yield in cows is a common presenting sign.

In selecting an anthelmintic for treatment, one should consider the withdrawal period of milk for human consumption. Eprinomectin has no withdrawal period for milk.

Reinfection syndrome in parasitic bronchitis Normally, the natural challenge of adult cattle, yearlings or calves that have acquired immunity to *D. viviparus*, whether by natural exposure or by vaccination, is not associated with clinical signs. Occasionally, however, clinical signs do occur to produce the 'reinfection syndrome', which is usually mild but sometimes severe. It arises when an immune animal is suddenly exposed to a massive larval challenge that reaches the lungs, and migrates to the bronchioles where the larvae are killed by the immune response. The proliferation of lymphoreticular cells around dead larvae causes bronchiolar obstruction and ultimately the formation of a macroscopically visible, greyish-green, lymphoid nodule about 5.0 mm in diameter. Usually the syndrome is associated with frequent coughing and slight tachypnoea over a period of a few days; less frequently there is marked tachypnoea, hyperpnoea and, in dairy cows, a reduction in milk yield. Deaths rarely occur. It can be difficult to differentiate this syndrome from the early stages of a severe primary infection. The only course of action is treatment with anthelmintics and a change of pasture.

Echinococcus granulosus

For more details see Parasites of the liver.

Pneumocystis carinii

Synonym: *Pneumocystis jiroveci*

Common name: Pneumocystosis

Predilection site: Lung

Kingdom: Fungi

Phylum: Ascomycota

Class: Pneumocystidomycetes

Family: Pneumocystidaceae

Description: Two major forms of *P. carinii* have been consistently identified from histological and ultrastructural analysis of organisms found in human and rat lung. These are a trophic form and a larger cyst stage containing eight intracystic stages.

Hosts: Human, cattle, rat, ferret, mouse, dog, horse, pig and rabbit

Geographical distribution: Worldwide

Pathogenesis: *Pneumocystis* is one of the major causes of opportunistic mycoses in the immunocompromised, including those with congenital immunodeficiencies, retrovirus infections such as AIDS, and cases receiving immunosuppressive therapy.

Clinical signs: Infections in animals are generally asymptomatic. In humans, pneumocystosis is observed in four clinical forms: asymptomatic infections, infantile (interstitial plasma cell) pneumonia, pneumonia in immunocompromised host and extrapulmonary infections.

Diagnosis: Gomori's methenamine silver (GMS) and Giemsa stain may be used for microscopic visualisation of *Pneumocystis*. Toluidine blue (TBO) is the most effective for cyst stages while Giemsa stains are used to show trophozoites. Axenic culture methods have been described; however, *in vitro* cultivation, especially from clinical samples, is not always successful. Fluorescence antibody staining techniques can be used to detect both cyst and trophozoite stages of *P. carinii*. A number of polymerase chain reaction (PCR) tests have been reported which amplify specific regions of DNA from *P. carinii* and are approximately 100 times more sensitive than conventional staining techniques.

Pathology: Lesions are characterised by a massive plasma cell or histiocyte infiltration of the alveoli in which the organisms may be detected by a silver staining procedure. A foamy eosinophilic material is observed in the lungs during infection. This material is composed of masses of the organism, alveolar macrophages, desquamated epithelial alveolar cells, polymorphonuclear leucocytes and other host cells.

Epidemiology: The organism is apparently quite widely distributed in latent form in healthy human individuals, as well as a wide variety of domestic and wild animals. The organism is thought to be transmitted by aerosol, although the natural habitats and modes of transmission of infections in humans are current areas of research. *Pneumocystis* DNA has been detected in air and water, suggesting that the free forms of the organism may survive in the environment long enough to infect a susceptible host. However, little information on the means of transmission exists currently. In humans, infections appear to spread between immunosuppressed patients colonised with *Pneumocystis* and immunocompetent individuals transiently parasitised with the organism. Human and non-human *Pneumocystis* species have been shown to be different and host-specific, suggesting that zoonotic transmission does not occur.

The organism has been reported from a range of animals. In Denmark, examination of lungs from carcasses selected randomly in an abattoir detected *P. carinii* pneumocysts in 3.8% of calves, 3.6% of sheep and 6.7% of pigs. Studies in Japan detected *P. carinii* in cattle and a wide range of other animals. The organism has also been reported to have caused pneumonia in weaning pigs.

Treatment: Trimethroprim–sulphamethoxazole is the drug of choice for treatment and prophylaxis of *Pneumocystis* infections. Pentamidine and atovaquone are the alternative therapeutic agents in humans.

Control: Control is difficult given that details of the routes of transmission are unknown. Infection is generally asymptomatic in animals and is only likely to be detected in immunocompromised individuals.

Notes: Initially reported as a morphological form of *Trypanosoma cruzi*, this microorganism later proved to be a separate genus and was named *Pneumocystis carinii* and classified as a protozoan until the late 1980s. Following further taxonomic revision, *Pneumocystis* is now classified as a fungus, not a protozoan. The taxonomy is still complicated in that *Pneumocystis* from humans and other animals are quite different and there appear to be multiple species in this genus. Genetic variations and DNA sequence polymorphisms are often observed, suggesting the existence of numerous strains even within a single species of *Pneumocystis*.

Parasites of the liver

Fasciola hepatica

Common name: Liver fluke

Predilection site: Liver

Phylum: Platyhelminthes

Class: Trematoda

Family: Fasciolidae

Description, gross: The young fluke at the time of entry into the liver is 1.0–2.0 mm in length and lancet-like. When it has become fully mature in the bile ducts it is leaf-shaped, grey–brown in colour and is around 2.5–3.5 cm in length and 1.0 cm in width. The anterior end is conical and marked off by distinct shoulders from the body (Fig. 8.20; see also Fig. 1.70a).

Description, microscopic: The tegument is covered with backwardly projecting spines. An oral and ventral sucker may be readily seen. The egg is thin-shelled, oval with symmetrical barrel-shaped

Figure 8.20 Adult *Fasciola hepatica* flukes.

side walls, operculate, browny-yellow and large (130–150 × 65–90 μm), and about twice the size of a trichostrongyle egg (see Figs 4.3 and 9.1). The granular contents fill the whole of the egg.

Final hosts: Sheep, cattle, goat, horse, deer, human and other mammals

Intermediate hosts: Snails of the genus *Galba* (*Lymnaea*). The most common, *Galba* (syn. *Lymnaea*) *truncatula*, is an amphibious snail with a wide distribution throughout the world. Other important vectors of *F. hepatica* outside Europe are:

L. tomentosa	Australia, New Zealand
L. columella	Central and North America, Australia, New Zealand
L. bulimoides	Northern and southern USA and the Caribbean
L. humilis	North America
L. viator	South America
L. diaphena	South America
L. cubensis	South America
L. viridis	China, Papua New Guinea

Geographical distribution: Worldwide

Pathogenesis: This varies according to the number of metacercariae ingested, the phase of parasitic development in the liver and the species of host involved. Essentially the pathogenesis is twofold. The first phase occurs during migration in the liver parenchyma and is associated with liver damage and haemorrhage. The second occurs when the parasite is in the bile ducts, and results from the haematophagic activity of the adult flukes and from damage to the biliary mucosa by their cuticular spines. Most studies have been in sheep and the disease in this host is discussed in more detail in Chapter 9. The seasonality of outbreaks described is that which occurs in western Europe.

Although acute and subacute disease may occasionally occur under conditions of heavy challenge, especially in young calves, the chronic form of the disease is by far the most important and, as in sheep, is seen in the late winter/early spring.

The pathogenesis is similar to that in sheep but has the added features of calcification of the bile ducts and enlargement of the gallbladder. The calcified bile ducts often protrude from the liver surface, giving rise to the term 'pipe-stem liver'. Aberrant migration of the flukes is more common in cattle and encapsulated parasites are often seen in the lungs. On reinfection of adult cows, migration to the fetus has been recorded, resulting in prenatal infection. There is some experimental evidence that fasciolosis increases the susceptibility of cattle to infection with *Salmonella dublin*.

Fasciola infections may cause a loss of production in milking cows during winter. Clinically, these are difficult to detect since the fluke burdens are usually low and anaemia is not apparent. The main effects are a reduction in milk yield and quality, particularly of the solids-non-fat component.

Clinical signs: In heavy infections in cattle, where anaemia and hypoalbuminaemia are severe, submandibular oedema frequently occurs (Fig. 8.21). With smaller fluke burdens, the clinical effect is minimal and the loss of productivity is difficult to differentiate from inadequate nutrition. It must be emphasised that diarrhoea is not a feature of bovine fasciolosis unless it is complicated by the presence of *Ostertagia* spp. Combined infection with these two parasites has been referred to as the fasciolosis/ostertagiosis complex.

Diagnosis: This is based primarily on clinical signs, seasonal occurrence, prevailing weather patterns, and a previous history of fasciolosis on the farm or the identification of snail habitats. While diagnosis of ovine fasciolosis should present few problems, especially when a postmortem examination is possible, diagnosis of bovine fasciolosis can sometimes prove difficult. In this context, routine haematological tests and examination of faeces for fluke eggs (note that eggs of *Fasciola* are browny-yellow and eggs of Paramphistomidae are colourless) are useful and may be supplemented by other laboratory tests.

Figure 8.21 Submandibular oedema in a cow infected with *Fasciola hepatica*.

Routine haematology will often show the presence of anaemia (normochromic and normocytic) as a result of haemorrhage resultant from the direct feeding of the flukes. The packed cell volume (PCV) is also reduced. Fluke infection also leads to an eosinophilia (Table 8.4).

Fluke infections lead to a decrease in the albumin/globulin ratio. Hypoalbuminaemia due to protein loss occurs during the parenchymal stage of infection by immature flukes, and also due to the presence of adult fluke in the bile ducts. Globulin levels increase as a result of increased immunoglobulin synthesis.

Serum concentrations of liver-specific enzymes are generally higher in acute liver disease than in chronic liver disease and may be within normal limits in the later stages of subacute or chronic hepatic disease. Glutamate dehydrogenase (GLDH) is released when parenchymal cells are damaged and levels become elevated within the first few weeks of infection. Another enzyme, gamma-glutamyltranspeptidase (GGT), indicates damage to the epithelial cells lining the bile ducts; elevation of this enzyme takes place mainly after

Table 8.4 Haematological/biochemical parameters in normal and fluke-infected cattle.

Parameter	Normal	Fluke infected
PCV (%)	32 (24–40)	≥20
Eosinophils (%) (×10^3/μL)	2–20 0–2.4	>20%
Glutamate dehydrogenase (GLDH) (iu/L)	2–23	5× normal (50–120) Elevated ≥6 weeks post infection
Gamma-glutamyltranspeptidase (GGT) (iu/L)	20–46	Up to 10× normal levels in chronic fluke

the flukes reach the bile ducts and raised levels are maintained for a longer period. Interpretation of raised liver enzyme activity can be difficult and careful analysis of laboratory values in conjunction with clinical findings is essential.

Detection of antibodies against components of flukes in serum or milk samples can also be undertaken, the ELISA and the passive haemagglutination test being the most reliable. Antibodies to liver fluke can be detected in serum 2–4 weeks post infection but levels may rise or fall over time. A positive result does not necessarily indicate a current infection but a history of exposure. Serological testing is not widely available and may vary from country to country as to availability for either cattle or sheep. A bulk-tank milk ELISA for cattle gives a positive result when the prevalence in a herd is more than 25%. Interpretation can be difficult as false positives can occur. A coproantigen test is also available which detects fluke proteins in faeces.

Pathology: In cattle, the pathogenesis is similar to that seen in sheep with the added features of calcification of the bile ducts and enlargement of the gallbladder. The calcified bile ducts often protrude from the liver surface, giving rise to the term 'pipe-stem liver' (Fig. 8.22). Aberrant migration of flukes is more common in cattle and encapsulated parasites are often seen in the lungs.

Epidemiology: For a more detailed description, see entry in Chapter 9.

Treatment: The older drugs such as carbon tetrachloride, hexachloroethane and hexachlorophene may still be used in some countries, but these have been largely replaced by more efficient and less toxic compounds and only the latter are discussed.

At present there is only one drug, triclabendazole, which will remove the early immature (around 2 weeks of age in cattle) parenchymal stages. Apart from triclabendazole, the other drugs most commonly used for subacute or chronic fasciolosis are closantel, nitroxynil and oxyclozanide, and several others, such as clorsulon, rafoxanide and niclofolan, are also marketed in some countries.

Figure 8.22 Gross appearance of the liver in bovine fasciolosis.

Albendazole, ricobendazole and netobimin are also effective against adult fluke at an increased dosage rate. In lactating cows, where the milk is used for human consumption, the above drugs are either banned or have extended withdrawal periods, and are more usually given during the dry period. An exception is oxyclozanide, which is licensed for use in lactating animals in many countries and has a nil or very short milk-withholding time. At times of the season when the fluke burden predominantly comprises adults, the use of narrow-spectrum flukicides with activity against adult stages only will help to reduce the selection pressure on drugs such as triclabendazole. Combination products with activity against fluke and gastrointestinal nematodes should only be used where both helminths are present. Resistance to flukicides has been reported with triclabendazole use in sheep but is not currently considered to be as significant a problem with fluke in cattle.

Control: Control of fasciolosis may be approached in two ways: by reducing populations of the intermediate snail host or by using anthelmintics (for a more detailed description see entry in Chapter 9). The timing of treatments will depend on the spectrum of activity of the flukicide and it is also important to monitor the need for treatment. The use of meteorological forecasting for fasciolosis is described in detail in Chapter 9.

A typical treatment schedule for non-lactating cattle in the northern hemisphere in an average rainfall season would be as follows.

- Dose cattle in autumn with a flukicide that is effective against young immature fluke to reduce liver damage from fluke migration. This is irrespective of whether cattle will be housed or outwintered.
- Dose grazing cattle in winter with a flukicide that is effective against adult fluke and immature stages. Cattle wintered inside need to be treated after housing (timing of the dose varies with the flukicide used).
- Dose outwintered cattle in spring with a flukicide that is effective against adult stages. This will remove fluke burdens and reduce contamination of pastures with fluke eggs and thus reduce the summer infection of snails.

Dairy cows can be treated at drying-off but particular attention needs be paid to contraindications relating to both stage of pregnancy and lactation.

Fasciola gigantica

Common name: Tropical large liver fluke

Predilection site: Liver

Phylum: Platyhelminthes

Class: Trematoda

Family: Fasciolidae

Description, gross: The adult fluke is larger than *F. hepatica*, the body is more transparent, and can reach 7.5 cm in length and 1.5 cm in breadth. The shape is more leaf-like, the conical anterior end is very short and the shoulders, characteristic of *F. hepatica*, are barely perceptible (see Fig. 1.70b).

Description, microscopic: The eggs are larger than those of *F. hepatica*, measuring 170–190 by 90–100 μm.

Final hosts: Cattle, buffalo, sheep, goat, pig, camel, deer, human

Intermediate hosts: Snails of the genus *Lymnaea* (syn. *Galba*); in southern Europe it is *L. auricularia*, which is also the important species in the southern USA, the Middle East and the Pacific Islands. Other important vectors of *F. gigantica* are:

L. natalensis	Africa
L. rufescens	Indian subcontinent
L. acuminata	Indian subcontinent
L. rubiginosa	Southeast Asia
L. viridis	China and Japan

All these snails are primarily aquatic snails and are found in streams, irrigation channels and marshy swamps.

Geographical distribution: Africa, Asia, Europe, USA

Clinical signs: Clinical signs are similar to those of *F. hepatica*.

Diagnosis: This is based primarily on clinical signs, seasonal occurrence, prevailing weather patterns and a previous history of fasciolosis on the farm or the identification of snail habitats. Diagnosis can be confirmed by the identification of the typical operculate eggs in faeces samples.

Pathogenesis: Acute and chronic infection occurs in sheep but only the chronic form predominates in cattle. Like *F. hepatica*, *F. gigantica* is capable of infecting humans.

Pathology: The pathology is similar to that described for *F. hepatica*. In cattle, the pathology is similar to that seen in sheep with the added features of calcification of the bile ducts and enlargement of the gallbladder. The calcified bile ducts often protrude from the liver surface giving rise to the term 'pipe-stem liver'.

Epidemiology: The snails that carry the larval stages of *F. gigantica* are primarily aquatic and as a result the disease is associated with animals grazing on naturally or artificially flooded areas or around permanent water channels or dams. In subtropical or tropical countries with distinct wet and dry seasons, it appears that optimal development of eggs to miracidia occurs at the start of the wet season and development within the snail is complete by the end of the rains. Shedding of cercariae then commences at the start of the dry season when the water level is still high and continues as the water level drops. Under laboratory conditions, a large number of metacercariae simply encyst on the surface of the water rather than on herbage, and under natural conditions this could have a very significant effect on the dissemination of infection. Metacercariae are acquired by animals utilising such areas during the dry season and clinical problems, depending on the rate of infection, occur at the end of that season or at the beginning of the next wet season. Metacercariae encyst on plants under water, such as rice plants, and can survive for up to 4 months on stored plants, such as rice straw.

Treatment: The drugs and dose rates given for the treatment of *F. hepatica* are also generally applicable for the treatment of *F. gigantica*. Triclabendazole is effective against both mature and immature stages of *F. gigantica* in cattle.

Control: The principles are the same as for the control of *F. hepatica* and are based on the routine use of anthelmintics together with measures to reduce populations of the snail intermediate host. However, there is the important difference that the latter are water snails whose control depends on a different approach from that for the mud snail *G.* (*Lymnaea*) *truncatula*.

Routine anthelmintic treatment of animals at seasons when heavy infections of adult flukes accumulate in the host is recommended using a drug effective against adult and immature flukes. This should prevent serious losses in production, but for optimal benefit should be accompanied by snail control.

When watering of stock is from a reservoir or stream, complete control can be achieved by fencing the water source and leading a pipe to troughs. To do this effectively from streams, the water may require to be pumped and in remote areas simple water-driven pumps whose power source depends on the water flow have been found useful. It is important that the water troughs be cleaned out regularly since they can become colonised by snails.

Where grazing depends on the dry-season use of marshy areas around receding lakebeds, snail control is difficult. Molluscicides are usually impractical because of the large body of water involved and their possible effect on fish, which may form an important part of the local food supply. Apart from repeated anthelmintic treatment to prevent patency of acquired infections of *F. gigantica*, there is often little one can do. Ideally, such areas are often best suited to irrigation and the growing of cash crops, the profit from which can be used to improve the dry-season food and water supply to cattle.

Fascioloides magna

Common name: Large American liver fluke

Predilection site: Liver and bile ducts

Phylum: Platyhelminthes

Class: Trematoda

Family: Fasciolidae

Description, gross: Flukes are large and thick and measure up to 10 by 2.5 cm. The flukes are oval, with a rounded posterior end. They possess no anterior cone and when fresh are flesh-coloured (Fig. 8.23).

Figure 8.23 *Fascioloides magna*.

Description, microscopic: Eggs are large, operculate, measure 109–168 by 75–96 µm and have a protoplasmic appendage at the pole opposite the operculum.

Final hosts: Deer, cattle, sheep, goat, pig, horse, llama

Intermediate hosts: A variety of freshwater snails, *Fossaria* spp., *Lymnaea* spp., *Stagnicola* spp.

Geographical distribution: Mainly occurs in North America, central, eastern and southwestern Europe, South Africa and Mexico

Pathogenesis: In deer and cattle, the flukes are frequently encapsulated in thin-walled fibrous cysts in the liver parenchyma and this restricted migration results in low pathogenicity. In cattle and pigs the flukes may become entrapped in a thick-walled fibrous capsule and there is no connection to the bile ducts and consequently it is rare to find fluke eggs in faeces in these livestock. Sometimes flukes can also be found in calcified cysts. Although haemorrhage and fibrosis may be present in the liver, there is often no obvious clinical sign of infection.

Clinical signs: In deer and cattle the parasites can cause hepatic damage on reaching the liver but the flukes rapidly become encapsulated by the host reaction and clinical signs are minimal.

Diagnosis: This is based primarily on clinical signs, and history of contact with grazing deer in known endemic areas. Cysts and the large flukes are usually seen on postmortem examination. Faecal examination for the presence of fluke eggs is a useful aid to diagnosis.

Pathology: In cattle and pigs, thick-walled cysts with fibrous capsules or calcified cysts may be present in the liver.

Epidemiology: The various snail intermediate hosts tend to occur in stagnant semi-permanent water that contains large amounts of dead or dying vegetation, swamp areas, or pools and streams. *Fascioloides magna* is indigenous to North America and is common in Canada and the Great Lake areas where the white-tailed deer and the elk are commonly infected. Domestic cattle and sheep become infected when they graze pasture where parasitised deer occur.

Treatment: For cattle and sheep the commonly used flukicides, such as triclabendazole, closantel, clorsulon and albendazole, are effective. Mature *F. magna* are susceptible to oxyclosanide.

Control: Avoid grazing sheep or cattle on areas which are frequented by deer. Elimination of the snail intermediate hosts is difficult due to their varied habitats. Similarly, removal of Cervidae may not be practical. Because of these factors sheep rearing, particularly, is difficult in areas where the parasite is prevalent.

Notes: *Fascioloides magna* is primarily a parasite of deer (Cervidae) and is commonly found in white-tailed deer, elk and moose. For more details see Chapter 14.

Dicrocoelium dendriticum

Synonym: *Dicrocoelium lanceolatum*

Common name: Small lanceolate fluke

Predilection site: Liver

Phylum: Platyhelminthes

Class: Trematoda

Family: Dicrocoeliidae

Description, gross: There is no possibility of confusion with other flukes in the bile ducts of ruminants as *Dicrocoelium* is 0.6–1.0 cm long and 1.5–2.5 mm wide, distinctly lanceolate and semi-transparent. The oral sucker is smaller than the ventral sucker (see Fig. 1.74).

Description, microscopic: The gut is simple, consisting of two branches and resembles a tuning fork. Behind the ventral sucker the testes lie in tandem with the ovary immediately posterior. There are no spines on the cuticle (cf. *Fasciola*). The thick-shelled egg is small, 35–45 µm in length by 22–30 µm in width, dark brown with small round poles and slightly barrel-shaped walls and operculate, usually with a flattened side. The operculum is often difficult to see. It contains a miracidium that completely fills the egg when passed in the faeces.

Final hosts: Sheep, goat, cattle, deer and rabbit, occasionally horse, dog and pig

Intermediate hosts: Two are required.

1. Land snails of many genera, principally *Cionella lubrica* in North America and *Zebrina detrita* in Europe. Some 29 other species have been reported to serve as first intermediate hosts, including the genera *Abida*, *Theba*, *Helicella* and *Xerophila*.
2. Brown ants of the genus *Formica*, frequently *F. fusca*.

Geographical distribution: Worldwide except for South Africa and Australia. In Europe the prevalence is high but in the British Isles prevalence is low, being confined to small foci throughout the country.

For more information on pathogenesis, epidemiology, treatment and control see Chapter 9.

Dicrocoelium hospes

Predilection site: Liver

Phylum: Platyhelminthes

Class: Trematoda

Family: Dicrocoeliidae

Hosts: Cattle, ox and occasionally sheep, goat

Description: Details are essentially similar to *D. dendriticum* and the flukes are usually found in the liver and gallbladder.

Geographical distribution: Parts of Africa

Gigantocotyle explanatum

Synonyms: *Explanatum explanatum, Paramphistomum explanatum*

Predilection site: Liver, intrahepatic ductules, bile ducts, gallbladder, duodenum

Phylum: Platyhelminthes

Class: Trematoda

Family: Paramphistomatidae

Description, gross: These are conical pinkish flukes when fresh. Adult flukes are 8–10 mm in length by 4.7–5.7 mm in width.

Description, microscopic: The body tapers anteriorly and is curved ventrally with no tegumental papillae. The acetabulum is very large and the genital pore is bifurcal. The oval eggs measure 180–200 by 110–130 µm, are colourless and have an operculum.

Final hosts: Cattle, buffalo and other ruminants

Intermediate hosts: Snails

Geographical distribution: Indian subcontinent, Southeast Asia, tropical and subtropical regions of the Middle East and Africa

Pathogenesis: Large numbers of immature flukes can cause amphistomosis with enteritis that in some cases, particularly young buffaloes, can be fatal to the host. The flukes can cause connective tissue proliferation and haemorrhages at the site of attachment.

Clinical signs: General wasting of body condition, diarrhoea and loss of weight.

Pathology: There is extensive fibrosis and hyperplasia of the bile ducts and multifocal granulomatous nodules occur over their luminal surface.

Echinococcus granulosus, Echinococcus orteleppi (G5)

Common names: Dwarf dog tapeworm, hydatidosis

Predilection site: Mainly liver and lungs (intermediate hosts); small intestine (definitive hosts)

Phylum: Platyhelminthes

Class: Cestoda

Family: Taeniidae

Description, gross: Hydatid cysts are large fluid-filled vesicles, 5–10 cm in diameter, with a thick concentrically laminated cuticle and an internal germinal layer.

Description, microscopic: The germinal layer produces numerous small vesicles or brood capsules each containing up to 40 scolices, invaginated into their neck portions and attached to the wall by stalks. Brood capsules may become detached from the wall of the vesicle and float freely in the vesicular fluid and form 'hydatid sand'.

Final hosts: Dog and many wild canids

Intermediate hosts: Cattle (G5), sheep, camel, pig, buffalo, deer, human

Geographical distribution: Worldwide

Notes: *Echinococcus granulosus* possesses a high degree of genetic divergence and various strains (G1–G10) have been described that show differences in morphology, host range, pathogenicity and geographical distribution. *Echinococcus orteleppi* (the former cattle strain G5) is now recognised as an individual species.

For more details on pathology, treatment and control see Chapter 9.

Stilesia hepatica

Predilection site: Bile ducts

Phylum: Platyhelminthes

Class: Cestoda

Family: Anoplocephalidae

Description: The adult tapeworm measures 20–50 cm long by 2–3 mm wide. The neck is narrow and the scolex is large with prominent suckers. The genital organs are single and the opening pores alternate irregularly. There are 10–12 testes on either side lying dorsal to the ventral canal. The proglottids are short.

Final hosts: Sheep, cattle and other ruminants

Intermediate hosts: The intermediate host is probably an oribatid mite.

Geographical distribution: Africa and Asia

For more details see Chapter 9.

Taenia hydatigena

Synonyms: *Taenia marginata, Cysticercus tenuicollis*

Predilection site: Abdominal cavity, liver (intermediate hosts); small intestine (definitive hosts)

Phylum: Platyhelminthes

Class: Cestoda

Family: Taeniidae

Description: The semi-transparent cysticercus may be up to 5–7 cm in size and contains a watery fluid and invaginated scolex with a long neck.

Final hosts: Dog, fox, weasel, stoat, polecat, wolf, hyena

Intermediate hosts: Sheep, cattle, deer, pig, horse

Geographical distribution: Worldwide

Notes: The correct nomenclature for the intermediate host stage is the 'metacestode stage of *Taenia hydatigena*' rather than '*Cysticercus tenuicollis*'.

For more details see Chapter 9.

Thysanosoma actinioides

For more details see Parasites of the small intestine.

Parasites of the pancreas

Eurytrema pancreaticum

Synonyms: *Distoma pancreaticum, Eurytrema ovis*

Common name: Pancreatic fluke

Predilection site: Pancreatic ducts, rarely the bile ducts

Phylum: Platyhelminthes

Class: Trematoda

Family: Dicrocoeliidae

Description, gross: Oval, leaf-shaped, reddish-brown flukes measuring around 8–16 by 5–8.5 mm.

Description, microscopic: The body is thick and the juvenile flukes are armed with spines, which are often absent by the adult stage. The oral sucker is larger than the ventral sucker and the pharynx and oesophagus are short. The testes are positioned horizontally just behind the ventral sucker. A tubular cirrus sac is present. The uterus completely occupies the posterior body. Eggs measure around 40–50 by 25–35 μm and are similar to those of *Dicrocoelium*.

Final hosts: Cattle, buffalo, sheep, goat, pig, camel and human

Intermediate hosts: Two are required:

1 Land snails, particularly of the genus *Bradybaena*.
2 Grasshoppers of the genus *Conocephalus* or tree crickets (*Oecanthus*).

Geographical distribution: South America, Asia and Europe

Pathogenesis: Low to moderate infections produce little effect on the host. Heavy infections may cause a sporadic wasting syndrome and emaciation.

Clinical signs: No specific signs but general weight loss may occur in heavy infections.

Diagnosis: Usually reported as an incidental finding at necropsy.

Pathology: Large numbers of flukes can cause dilation and thickening of the pancreatic ducts and extensive fibrosis. Flukes may also be embedded in the pancreatic parenchyma causing chronic interstitial pancreatitis and there is sometimes a granulomatous reaction around fluke eggs that have penetrated the walls of the ducts.

Epidemiology: Infection is influenced by the availability of the invertebrate intermediate hosts.

Treatment: There is no specific treatment for eurytrematosis, although praziquantel 20 mg/kg for 2 days or albendazole 7–10 mg/kg have been reported to be effective.

Control: This is not feasible where the intermediate hosts are endemic.

Eurytrema coelomaticum

Synonym: *Distoma coelomaticum*

Common name: Pancreatic fluke

Predilection site: Pancreatic ducts and occasionally the bile ducts and the duodenum

Phylum: Platyhelminthes

Class: Trematoda

Family: Dicrocoeliidae

Description, gross: A leaf-shaped reddish-brown fluke with adults measuring around 8–12 by 6–7 mm.

Geographical distribution: Eastern Asia and South America

Details of the life cycle, host range, pathogenesis, clinical signs, diagnosis, pathology, epidemiology, treatment and control are as for *E. pancreaticum*.

Thysanosoma actinioides

For more details see Parasites of the small intestine.

Parasites of the circulatory system

Elaeophora poeli

Common name: Large aortic filariosis

Predilection site: Blood vessels

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Slender worms, males measuring around 4–7 cm and females up to 30 cm in length.

Description, microscopic: There are no lips and the oesophagus is very long. The tail of the male bears five to seven pairs of papillae, two pairs being pre-cloacal. Microfilariae are 340–360 μm.

Final hosts: Cattle, buffalo, zebu

Intermediate hosts: Not known, possibly tabanid flies

Geographical distribution: Parts of Africa, Asia and the Far East

Pathogenesis: In cattle, nodules, from which the female worms protrude, form on the intima of the vessels but in other animals the adults appear to provoke little reaction.

Clinical signs: Infection is usually asymptomatic.

Diagnosis: This is not normally required. Infection is usually diagnosed as an incidental finding on postmortem examination of thickened blood vessels, or those containing nodules.

Pathology: The main affected area is the thoracic region of the aorta. In light infections, the lesions are found chiefly on the dorsal wall of the aorta, near the openings of the intercostal arteries. In heavy infections, the artery becomes swollen, the wall is thickened and the intima contains fibrous tracts. The raised nodules can measure up to 1 cm in diameter.

Epidemiology: Because of the innocuous nature of the infection in cattle, the distribution of the species in these hosts is not completely known.

Treatment: Treatment is not indicated.

Control: Any reduction in vector numbers will reduce transmission.

Onchocerca armillata

Common name: Aortic filariosis

Predilection site: Aorta

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Slender whitish worms. Male worms are about 7 cm and female worms up to 70 cm long.

Description, microscopic: Microfilariae are unsheathed and measure 346–382 μm.

Final hosts: Cattle, buffalo, sheep, goat, rarely camel

Intermediate hosts: Midges (*Culicoides*), blackflies (*Simulium*)

Geographical distribution: Africa, Middle East, India

Pathogenesis: It is interesting that *O. armillata*, though occurring in a strategically important site in the bovine aorta, is not usually associated with clinical signs. It is usually only discovered at the abattoir, surveys in the Middle East having shown a prevalence as high as 90%.

Clinical signs: Infection is usually inapparent.

Diagnosis: Typical nodular lesions may be found in the wall of the aorta on postmortem examination. Microfilariae may also be found in skin biopsy samples taken from affected areas. The piece of skin is placed in warm saline and teased to allow emergence of the microfilariae, and is then incubated for about 8–12 hours. The microfilariae are readily recognised by their sinuous movements in a centrifuged sample of the saline. Another option is to scarify the skin of a predilection site and examine the fluid exudate for microfilariae.

Pathology: *Onchocerca armillata* is found in grossly visible nodules in the intima, media and adventitia of the aorta (Fig. 8.24), and atheromatous plaques are commonly seen on the intima. In chronic infections, the aortic wall is thickened and the intima shows tortuous tunnels with numerous nodules containing yellow caseous fluid and coiled worms. Aortic aneurysms have been noted in about one-quarter of infections.

Epidemiology: Prevalence is very high; in some regions 80–90% of animals are infected.

Treatment: Rarely indicated. Daily administration of diethylcarbamazine over a period of 21 days acts as a microfilaricide, and a single dose of ivermectin is highly efficient in this respect, although the dying microfilariae may provoke local tissue reactions.

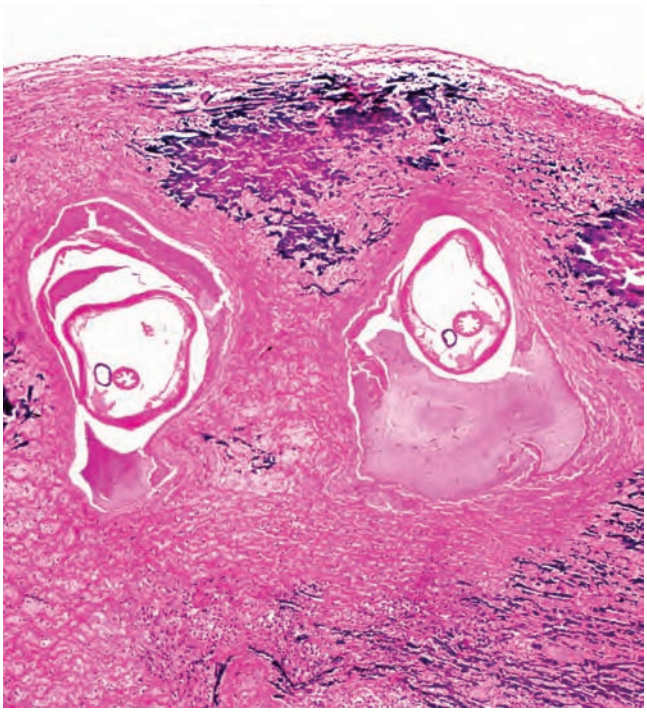

Figure 8.24 *Onchocerca armillata* within the aorta.

Control: With the ubiquity of the insect vectors there is little possibility of efficient control, though the use of microfilaricides will reduce the numbers of infected flies. In any case, with the relatively innocuous nature of the infection there is unlikely to be any demand for control.

Schistosomes

Schistosomes are flukes found in the circulatory system in which the sexes are separate, the small adult female lying permanently in a longitudinal groove, the gynaecophoric canal, in the body of the male. The genus has been divided into four groups – *haematobium*, *indicum*, *mansoni* and *japonicum* – but the genus as currently defined is paraphyletic so revisions are likely.

Haematobium group

Schistosoma bovis

Common name: Blood fluke, bilharziosis

Predilection site: Portal and mesenteric veins, urogenital veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate; the males are 9–22 mm long and 1–2 mm wide, and the female 12–28 mm long. The suckers and the body of the male behind the suckers are armed with minute spines, while the dorsal surface of the male bears small cuticular tubercles. The slender female worm lies permanently in a ventral groove in the broad flat body of the male.

Description, microscopic: The eggs are usually spindle-shaped, but smaller eggs may be oval and have a mean measurement of 187 by 65 μm when passed in the faeces. There is no operculum.

Final hosts: Cattle, sheep, goat, camel

Intermediate hosts: Snails (Bulinus contortus, B. truncates, Physopsis africana, P. nasuta)

Geographical distribution: Africa, Middle East, southern Asia, southern Europe

Pathogenesis: The young flukes cause some damage during migration but most serious damage is caused by the irritation produced by the parasite eggs in the intestine and the blood-sucking habit of the worms. Acute disease is characterised by diarrhoea and anorexia due to the response to deposition of eggs in the mesenteric veins and their subsequent infiltration in the intestinal mucosa. The presence of the worms in veins of the bladder in cattle may cause damage to the bladder wall and haematuria.

Clinical signs: These are diarrhoea, sometimes blood-stained and containing mucus, anorexia, thirst, anaemia and emaciation. In cattle, the presence of the worms in the vesical veins may cause haematuria.

Diagnosis: This is based mainly on the clinicopathological picture of diarrhoea, wasting and anaemia, coupled with a history of access to natural water sources. The relatively persistent diarrhoea, often blood-stained and containing mucus, may help to differentiate this syndrome from fasciolosis.

The demonstration of the characteristic eggs in the faeces or in squash preparations of blood and mucus from the faeces is useful in the period following patency but less useful as egg production drops in the later stages of infection.

In general, when schistosomosis is suspected, diagnosis is best confirmed by a detailed postmortem examination which will reveal the lesions and, if the mesentery is stretched, the presence of numerous schistosomes in the veins. In epidemological surveys, serological tests may be of value.

Pathology: At necropsy during the acute phase of the disease there are marked haemorrhagic lesions in the mucosa of the intestine, but as the disease progresses the wall of the intestine appears greyish, thickened and oedematous due to confluence of the egg granulomas and the associated inflammatory changes. The liver may be larger than normal, depending on the stage of the disease, and may be markedly cirrhotic in long-standing infections. On microscopic examination there is pigmentation of the liver and numerous eggs may be found, surrounded by cellular infiltration and fibrous tissue. The spleen may be slightly swollen and lymph glands are usually pigmented.

Epidemiology: The epidemiology is totally dependent on water as a medium for infection of both the intermediate and final host. Small streams, irrigation canals, wet savannah and marshy or damp areas are the main snail habitats. Eggs, miracidia and cercariae are short-lived with seasonal transmission directly related to rainfall and temperature. The fact that percutaneous infection may occur encourages infection where livestock are obliged to wade in water. In cattle, high prevalence is usually associated with low numbers of worms, although worm burdens increase with age while egg excretion declines markedly in animals above 2 years of age due to the development of partial immunity.

Treatment: For economic reasons, chemotherapy is not suitable for the control of schistosomosis in domestic stock except during severe clinical outbreaks. Care has to be exercised in treating clinical cases of schistosomosis since the dislodgement of the damaged flukes may result in emboli being formed and subsequent occlusion of major mesenteric and portal blood vessels with fatal consequences. Older drugs still used in some areas are the antimonial preparations tartar emetic, antimosan and stibophen, and niridazole and trichlorphon, all of which have to be given over a period of days at high dosage rates. Fatalities associated with the use of these drugs are not uncommon. Praziquantel, which is the drug of choice for treatment of human schistosomosis, is also effective in ruminants at 15–20 mg/kg *per os* but may be cost-prohibitive.

Control: This is similar to that outlined for *F. gigantica* and *Paramphistomum* infections. Since the prevalence of snail populations varies according to temperature, local efforts should be made to identify the months of maximum snail population, and cattle movements planned to avoid their exposure to dangerous stretches of water at these times.

When watering of stock is from a reservoir or stream, fencing the water source and leading a pipe to troughs can achieve control. To do this effectively from streams, the water may require to be pumped and in remote areas simple water-driven pumps whose power source depends on the water flow have been found useful. It is important that the water troughs be cleaned out regularly since they can become colonised by snails.

When grazing depends on the dry-season use of marshy areas around receding lakebeds, snail control is difficult. Molluscicides are usually impractical because of the large body of water involved and their possible effect on fish, which may form an important part of the local food supply. Apart from repeated anthelmintic treatment to prevent patency of acquired infections of *Schistosoma*, there is often little one can do. Ideally, such areas are often best suited to irrigation and the growing of cash crops, the profit from which can be used to improve the dry-season food and water supply to cattle.

Schistosoma mattheei

Predilection site: Portal, mesenteric and bladder veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate; the males are 9–22 mm long and 1–2 mm wide, and the females 12–28 mm long. The suckers and the body of the male behind the suckers are armed with minute spines, while the dorsal surface of the male bears small cuticular tubercles.

Description, microscopic: The eggs passed in faeces are usually spindle-shaped, but smaller ones may be oval. They measure 170–280 by 72–84 μm. There is no operculum.

Final hosts: Cattle, sheep, goat, camel, rodents, human

Intermediate hosts: Snails (*Bulinus* and *Physopsis* spp.)

Geographical distribution: South and Central Africa, Middle East

Notes: Thought to be synonymous with *S. bovis* but differs on morphological and pathological grounds and is restricted to the alimentary canal.

Schistosoma leiperi

Predilection site: Mesenteric veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: Similar to *S. spindale*. The eggs are large and resemble those of *S. spindale*, measuring 240–300 by 40–60 μm.

Final hosts: Antelope, cattle

Intermediate hosts: Snails (*Bulinus*)

Geographical distribution: Africa

Indicum group

Schistosoma indicum

Predilection site: Portal, pancreatic, hepatic and mesenteric veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate; the males measure 5–19 mm and the females 6–22 mm in length.

Description, microscopic: The eggs are oval with a terminal spine and measure 57–140 by 18–72 µm.

Final hosts: Cattle, sheep, goat, horse, donkey, camel, buffalo

Intermediate hosts: Snails (*Indoplanorbis*)

Geographical distribution: India

Schistosoma nasale

Common name: Snoring disease

Predilection site: Veins of nasal mucosa

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate; the male, which is broad and flat and about 6–11 mm long, carries the female (5–11 mm) in the hollow of its inwardly curved body. The flukes closely resemble *S. spindale*.

Description, microscopic: The eggs measure 350–380 by 50–80 µm and are boomerang-shaped, with a terminal spine.

Final hosts: Cattle, goat, sheep, buffalo, horse

Intermediate hosts: Snails *(Lymnaea luteola, L. acuminata, Indoplanorbis exustus)*

Geographical distribution: India, Pakistan, Southeast Asia

Pathogenesis: In heavy infections there is a copious mucopurulent discharge, snoring and dyspnoea. The main pathogenic effects are associated with the eggs, which cause abscess formation in the mucosa. Fibrous granulomatous growths occur which may occlude the nasal passages.

Clinical signs: Coryza, sneezing, dyspnoea and snoring

Diagnosis: Infection is confirmed by the presence of the spindle-shaped eggs in the nasal discharge.

Pathology: The mucosa of the nasal sinuses is studded with small abscesses that contain the eggs of the worms, and later show much fibrous tissue and proliferating epithelium (Fig. 8.25).

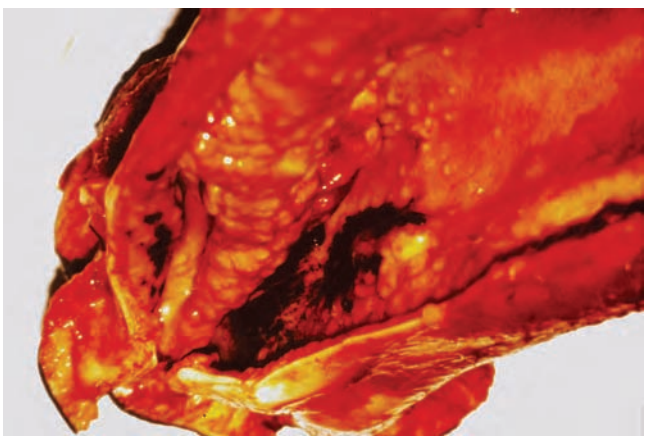

Figure 8.25 *Schistosoma nasale* lesions in nasal mucosa.

Epidemiology: The epidemiology is totally dependent on water as a medium for infection of both the intermediate and final host.

Treatment and control: As for *S. bovis*

Several other *Schistosoma* species have been reported in cattle. Details on the life cycle, pathogenesis, epidemiology, treatment and control are essentially similar to those for *S. bovis*.

Schistosoma spindale

Predilection site: Mesenteric veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate, with the male, which is broad and flat and measures up to about 1.5 cm in length, carrying the female in the hollow of its inwardly curved body.

Description, microscopic: The eggs are spindle-shaped, measure 200–300 by 70–90 µm and have a lateral or terminal spine. There is no operculum.

Hosts: Cattle, buffalo, horse, pig and occasionally dog

Geographical distribution: Parts of Asia and the Far East

Japonicum group

Schistosoma japonicum

Common name: Blood fluke, bilharziosis

Predilection site: Portal and mesenteric veins

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: The sexes are separate; the male, which is broad and flat and 9.5–20 mm long, carries the female (12–26 mm long) in the hollow of its inwardly curved body. The suckers lie close together near the anterior end. The cuticle is spiny on the suckers and in the gynaecophoric canal. This characteristic and the vascular predilection site are sufficient for generic identification.

Description, microscopic: The eggs are short, oval, measuring 70–100 by 50–80 µm, and may have a small lateral subterminal spine. There is no operculum.

Final hosts: Cattle, horse, sheep, goat, dog, cat, rabbit, pig, rodents, human

Intermediate hosts: Snails belonging to the genus *Oncomelania*

Geographical distribution: South and East Asia

Pathogenesis: The penetration of the cercariae through the skin causes dermatitis, which is evident about 24 hours after infection. Passage through the lungs may cause pneumonia in gross infections and abdominal organs such as the liver may become congested during the early stages of the disease due to the arrival of immature

worms in the intrahepatic portal blood vessels. The most serious damage is caused by the adult parasites in the egg-laying stage due to the irritation caused by eggs lodged in the tissues, which are forced to find their way through small venules to the epithelium and lumen of the gut. The masses of eggs become surrounded by inflamed areas and an infiltration of leucocytes, particularly eosinophils, gives rise to a rather characteristic type of abscess. The abscesses in the intestinal wall usually burst, discharging their contents into the lumen of the gut and eventually heal forming scar tissue. In the liver the abscesses become encapsulated and eventually become calcified, a large number of such foci leading to liver enlargement, cirrhosis and ascites.

Acute disease, characterised by diarrhoea and anorexia, occurs 7–8 weeks after heavy infection and is entirely due to the inflammatory and granulomatous response to the deposition of eggs in the mesenteric veins and their subsequent infiltration in the intestinal mucosa. Following massive infection death can occur rapidly, but more usually the clinical signs abate slowly as the infection progresses. As this occurs, there appears to be a partial shift of worms away from the intestinal mucosa and reactions to these migrating parasites and their eggs can occur in the liver.

Schistosomosis is generally considered to be a much more serious and important infection in sheep than in larger ruminants, and even where a high prevalence of the parasite is detected in slaughtered cattle, clinical signs of the disease are seen only rarely. In sheep, anaemia and hypoalbuminaemia have been shown to be prominent during the clinical phase apparently as a result of mucosal haemorrhage, dyshaemopoiesis and an expansion in plasma volume. The significance of low-level infection is not known, but it has been suggested that this may have a considerable effect on productivity.

There is experimental evidence of acquired resistance to reinfection by homologous species and, from natural infections, that resistance may develop as a result of prior exposure to a heterologous species.

Pathology: This is similar to that seen in *S. bovis*. Scar tissue and frequent papillomatous growths may be seen on the intestinal mucosa. On sections of the liver there is also evidence of egg granulomas and of portal fibrosis provoked by eggs which have inadvertently been swept into small portal vessels. The mesentery, mesenteric lymph nodes and spleen are frequently altered due to the presence of abnormal amounts of connective tissue.

Details of the clinical signs, diagnosis, epidemiology, treatment and control are as for *S. bovis*.

Other schistosomes

Schistosoma turkestanica

Synonym: *Orientobilharzia turkestanicum*

Predilection site: Mesenteric veins and small veins of the pancreas and liver

Phylum: Platyhelminthes

Class: Trematoda

Family: Schistosomatidae

Description, gross: This is a small species, the male measuring 4.2–8 mm and the female 3.4–8 mm in length.

Description, microscopic: The spirally coiled ovary is positioned in the anterior part of the body. In the male there are around 70–80 testes. The female uterus is short and contains only one egg at a time, which measures 72–77 by 16–26 µm with a terminal spine and a short appendage at the opposite end.

Final hosts: Cattle, buffalo, sheep, goat, camel, horse, donkey, mule and cat

Intermediate hosts: Snail (*Lymnaea euphratica*)

Geographical distribution: Asia, Middle East and parts of Europe

Pathogenesis: Of little significance in cattle but can produce marked debility in sheep and goats, causing hepatic cirrhosis and nodules in the wall of the intestines. This is often accompanied by loss of body weight in small ruminants.

Trypanosomes

Members of the genus *Trypanosoma* are haemoflagellates of overwhelming importance in cattle in sub-Saharan Africa as a cause of trypanosomosis. See Chapter 2 for general descriptions.

Salivarian trypanosomes

A number of species of *Trypanosoma*, found in domestic and wild animals, are all transmitted cyclically by *Glossina* in much of sub-Saharan Africa. The presence of trypanosomosis precludes the rearing of livestock in many areas, while in others where the vectors are not so numerous trypanosomosis is often a serious problem, particularly in cattle. The disease, sometimes known as nagana, is characterised by lymphadenopathy and anaemia accompanied by progressive emaciation and, often, death.

Pathogenesis: The signs and effects of the various trypanosomes found in domestic animals are more or less similar. The pathogenesis of trypanosomosis may be considered under three headings.

1. **Lymphoid enlargement** and **splenomegaly** develop. This is associated with plasma cell hyperplasia and hypergammaglobulinaemia, which is primarily due to an increase in IgM. Concurrently there is a variable degree of suppression of immune responses to other antigens such as microbial pathogens or vaccines. Ultimately, in infections of long duration, the lymphoid organs and spleen become shrunken due to exhaustion of their cellular elements.
2. **Anaemia** is a cardinal feature of the disease, particularly in cattle, and initially is proportional to the degree of parasitaemia. Anaemia is caused mainly by extravascular haemolysis through erythrophagocytosis in the mononuclear phagocytic systems of the spleen, liver and lungs, but as the disease becomes chronic there may be decreased haemoglobin synthesis. Leucopaenia and thrombocytopaenia are caused by mechanisms that predispose leucocytes and platelets to phagocytosis. Immunological mechanisms in the pathogenesis lead to extensive proliferation of activated macrophages, which engulf or destroy erythrocytes, leucocytes, platelets and haematopoietic cells. Later, in infections of several months' duration, when the parasitaemia often becomes low and intermittent, the anaemia may resolve to a variable degree. However, in some chronic cases it may persist despite chemotherapy.

3 **Cell degeneration** and **inflammatory infiltrates** occur in many organs, such as the skeletal muscle and the central nervous system (CNS), but perhaps most significantly in the myocardium where there is separation and degeneration of the muscle fibres. The mechanisms underlying these changes are still under study.

Clinical signs: In cattle, the major signs are anaemia, generalised enlargement of the superficial lymph glands (Fig. 8.26), lethargy and progressive loss of bodily condition. Fever and loss of appetite occur intermittently during parasitaemic peaks, the latter becoming marked in the terminal stages of the disease. Typically, the disease is chronic, extending over several months, and usually terminates fatally if untreated. As a herd phenomenon, the growth of young animals is stunted while adults show decreased fertility, and if pregnant may abort or give birth to weak offspring. In the terminal stages, animals become extremely weak, the lymph nodes are reduced in size and there is often a jugular pulse. Death is associated with congestive heart failure due to anaemia and myocarditis. Occasionally, the disease is acute, death occurring within 2–3 weeks of infection preceded by fever, anaemia and widespread haemorrhages.

Diagnosis: The clinical signs of the disease, although indicative, are not pathognomonic. Confirmation of clinical diagnosis depends on the demonstration of trypanosomes in the blood. If a herd or flock is involved, a representative number of blood samples should be examined, since in individual animals the parasitaemia may be in remission or in long-standing cases may be extremely scanty. Occasionally, when the parasitaemia is massive it is possible to detect motile trypanosomes in fresh films of blood. More usually, both thick and thin smears of blood are air-dried and examined later. Thick smears, de-haemoglobinised before staining with Giemsa or Leishman's stain, offer a better chance of finding trypanosomes, while the stained thin smears are used for differentiation of the trypanosome species.

More sensitive techniques utilise centrifugation in a microhaematocrit tube followed by microscopic examination of the interface between the buffy coat and the plasma; alternatively, the tube may be snapped, the buffy coat expressed onto a slide, and the contents examined under dark-ground or phase-contrast microscopy for motile trypanosomes. With these techniques, the PCV is also obtained which is of indirect value in diagnosis if one can eliminate other causes of anaemia, especially helminthosis.

Figure 8.26 Enlarged prescapular lymph node of Zebu with trypanosomosis.

A number of serological tests have been described and include the indirect fluorescent antibody test and ELISA and have been partially validated but require further evaluation and standardisation.

Pathology: The carcass is often pale and emaciated and there may be oedematous swellings in the lower part of the abdomen and genital organs with serous atrophy of fat. The liver, lymph nodes and spleen are enlarged and the viscera are congested. Petechiae may appear on lymph nodes, pericardium and intestinal mucosa. The liver is hypertrophic and congested with degeneration and necrosis of the hepatocytes, dilation of blood vessels and parenchymal infiltration of mononuclear cells. A non-suppurative myocarditis, sometimes associated with hydropericarditis, has been reported accompanied by degeneration and necrosis of the myocardial tissue. Other lesions can include glomerulonephritis, renal tubular necrosis, non-suppurative meningo-encephalomyelitis, focal poliomalacia, keratitis, ophthalmitis, orchitis, interstitial pneumonia and bone marrow atrophy. Splenic and lymph node hypertrophy occur during the acute phase but the lymphoid tissues are usually exhausted and fibrotic in the chronic stage.

Epidemiology: The vectors are various species of *Glossina* including *G. morsitans*, *G. palpalis*, *G. longipalpis*, *G. pallidipes* and *G. austeni*. *Trypanosoma congolense* can also be transmitted mechanically by other biting flies in tsetse-free areas, although this is uncommon. Since the life cycle of *T. vivax* is short, it is more readily transmitted than other species and mechanical transmission of *T. vivax* by tabanids allows it to spread outside the tsetse belt. The disease can also be transmitted mechanically through contaminated needles and instruments.

The epidemiology depends on three factors: the distribution of the vectors, virulence of the parasite and the response of the host.

- **The vectors**. Of the three groups of tsetse flies (see *Glossina*), the savannah and riverine are the most important since they inhabit areas suitable for grazing and watering. Although the infection rate of *Glossina* with trypanosomes is usually low, ranging from 1 to 20% of the flies, each is infected for life, and their presence in any number makes the rearing of cattle, pigs and horses extremely difficult. Biting flies may act as mechanical vectors, but their significance in Africa is still undefined.
- **The parasites**. Since parasitaemic animals commonly survive for prolonged periods, there are ample opportunities for fly transmission. Perhaps the most important aspect of trypanosomosis which accounts for the persistent parasitaemia is the way in which the parasite evades the immune response of the host. As noted previously, metacyclic and bloodstream trypanosomes possess a glycoprotein coat which is antigenic and provokes the formation of antibodies that cause opsonisation and lysis of the trypanosomes. Unfortunately, by the time the antibody is produced, a proportion of the trypanosomes have altered the chemical composition of their glycoprotein coat and now, displaying a different antigenic surface, are unaffected by the antibody. Those trypanosomes possessing this new **variant antigen** multiply to produce a second wave of parasitaemia; the host produces a second antibody, but again the glycoprotein coat has altered in a number of trypanosomes so that a third wave of parasitaemia occurs. This process of **antigenic variation** associated with waves and remissions of parasitaemias, often at weekly intervals, may continue for months, usually with a fatal outcome. The repeated switching of the glycoprotein coat is now known to depend on a loosely ordered sequential expression of an undefined number of

Figure 8.27 Trypanotolerant N'Dama breed of West Africa.

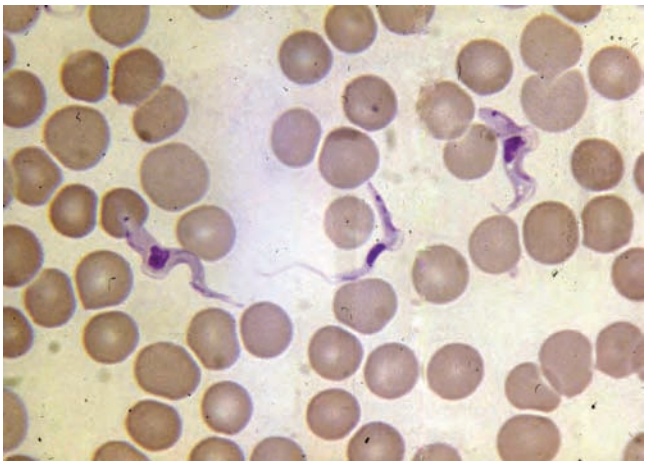

Figure 8.28 Tryptomastigotes of *Trypanosoma brucei*.

genes, each coding for a different glycoprotein coat. This, together with the finding that metacyclic trypanosomes may be a mixture of antigenic types, each expressing a different genetic repertoire, explains why domestic animals, even if treated successfully, are often immediately susceptible to reinfection. The complexity of antigens potentially involved has also defeated attempts at vaccination.

- **The hosts**. Trypanosomosis is basically an infection of wildlife in which, by and large, it has achieved a *modus vivendi* in that the animal hosts are parasitaemic for prolonged periods but generally remain in good health. This situation is known as **trypanotolerance**. In contrast, rearing of domestic livestock in endemic areas has always been associated with excessive morbidity and mortality, although there is evidence that a degree of adaptation or selection has occurred in several breeds. Thus in West Africa, small humpless cattle of the *Bos taurus* type, notably the N'Dama, survive and breed in areas of heavy trypanosome challenge despite the absence of control measures (Fig. 8.27). However, their resistance is not absolute and trypanosomosis exacts a heavy toll, particularly in productivity. In other areas of Africa, indigenous breeds of sheep and goats are known to be trypanotolerant, although this may be partly due to their being relatively unattractive hosts for *Glossina*. Precisely how trypanotolerant animals cope with antigenic variation is unknown. It is thought that the control and gradual elimination of their parasitaemias may depend on the possession of a particularly rapid and effective antibody response, although other factors may also be involved.

Trypanosoma brucei brucei

Common name: Nagana

Predilection site: Blood. *Trypanosoma brucei brucei* is also found extravascularly in, for example, the myocardium, CNS and reproductive tract.

Phylum: Euglenozoa

Class: Kinetoplastea

Family: Trypanosomatidae

Subgenus: *Trypanozoon*

Description: *Trypanosoma brucei brucei* is pleomorphic in form and ranges from long and slender, up to 42 μm (average 29 μm), to short and stumpy, 12–26 μm (mean 18 μm), the two forms often being present in the same blood sample. The undulating membrane is conspicuous, the kinetoplast is small and subterminal and the posterior end is pointed. In the slender form the kinetoplast is up to 4 μm from the posterior end, which is usually drawn out, tapering almost to a point, and has a well-developed free flagellum; in the stumpy form the flagellum is either short or absent and the posterior end is broad and rounded with the kinetoplast almost terminal. Intermediate forms average 23 μm long and have a blunt posterior end and moderately long flagellum (Fig. 8.28; see also Fig. 2.6). A fourth form with a posterior nucleus may be seen in laboratory animals. In fresh unfixed blood films, the organism moves rapidly within small areas of the microscope field.

Hosts: Cattle, horse, donkey, zebu, sheep, goat, camel, pig, dog, cat, wild game species, particularly antelope

Geographical distribution: Approximately 10 million km^2 of sub-Saharan Africa between latitudes 14° N and 29° S.

Pathogenesis: In *T. brucei brucei* infections, the disease is usually more chronic in cattle and animals may survive for several months and may recover.

Treatment: The two drugs in common use are isometamidium and diminazene aceturate. These are usually successful except where trypanosomes have developed resistance to the drug or in some very chronic cases. Treatment should be followed by surveillance since reinfection, followed by clinical signs and parasitaemia, may occur within a week or two. Alternatively, the animal may relapse after chemotherapy, due to a persisting focus of infection in its tissues or because the trypanosomes are drug resistant.

Notes: Antelope are the natural host species and are reservoirs of infection for domestic animals. Horses, mules and donkeys are very susceptible, and the disease is very severe in sheep, goats, camels and dogs (see respective hosts).

Other subspecies of *T. brucei* – *T. brucei evansi* and *T. brucei equiperdum* – are described separately under their respective subspecies and definitive hosts.

Two other subspecies, *T. brucei gambiense* and *T. brucei rhodesiense*, are important causes of 'sleeping sickness' in humans.

Trypanosoma brucei evansi

Synonyms: *Trypanosoma evansi, Trypanosoma equinum*

Common names: Surra, el debab, mbori, murrina, mal de Caderas, doukane, dioufar, thaga

Predilection site: Blood

Phylum: Euglenozoa

Class: Kinetoplastea

Family: Trypanosomatidae

Subgenus: *Trypanozoon*

Hosts: Horse, donkey, camel, cattle, zebu, goat, pig, dog, water buffalo, elephant, capybara, tapir, mongoose, ocelot, deer and other wild animals. Many laboratory and wild animals can be infected experimentally.

Geographical distribution: North Africa, Central and South America, central and southern Russia, parts of Asia (India, Burma, Malaysia, southern China, Indonesia, Philippines)

Pathogenesis: Domestic species such as cattle, buffalo and pigs are commonly infected, but overt disease is uncommon and their main significance is as reservoirs of infection.

Treatment and control: Suramin or quinapyramine (Trypacide) are the drugs of choice for treatment and also confer a short period of prophylaxis. For more prolonged protection a modified quinapyramine known as Trypacide Pro-Salt is also available. Unfortunately, drug resistance, at least to suramin, is not uncommon.

Notes: The original distribution of this parasite coincided with that of the camel, and is often associated with arid desserts and semi-arid steppes.

For more details see Chapter 10.

Trypanosoma congolense

Common names: Nagana, paranagana, Gambia fever, ghindi, gobial

Predilection site: Blood

Phylum: Euglenozoa

Class: Kinetoplastea

Family: Trypanosomatidae

Subgenus: *Nannomonas*

Description: *Trypanosoma congolense* is small, monomorphic in form and 8–20 μm long. The undulating membrane is inconspicuous, the medium-sized kinetoplast is marginal and the posterior end is blunt. There is no free flagellum (Fig. 8.29; see also Fig. 2.5). In fresh blood films the organism moves sluggishly, often apparently attached to red cells.

Hosts: Cattle, sheep, goat, horse, camel, dog, pig. Reservoir hosts include antelope, giraffe, zebra, elephant and warthog.

Geographical distribution: Widely distributed in tropical Africa between latitudes 15° N and 25° S.

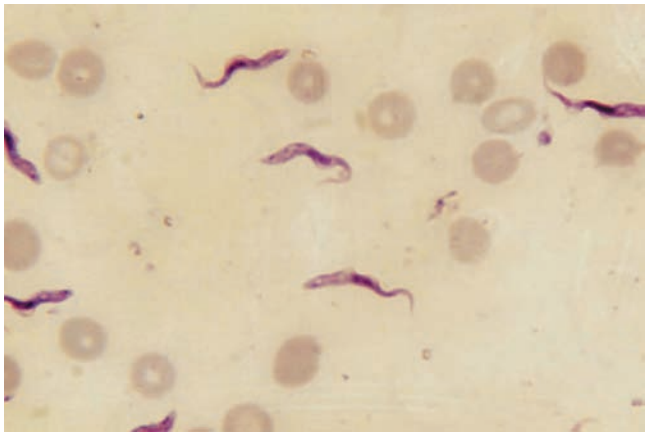

Figure 8.29 Tryptomastigotes of *Trypanosoma congolense*.

Pathogenesis: With *T. congolense*, there are many strains which differ markedly in virulence. In cattle, the parasite can cause an acute fatal disease resulting in death in about 10 weeks, a chronic condition with recovery in about 1 year, or a mild almost asymptomatic condition. The signs caused by this species are similar to those caused by other trypanosomes, but the CNS is not affected.

Treatment and control: In infected cattle, the two drugs in common use are diminazene aceturate (Berenil) and homidium salts (Ethidium and Novidium). As with *T. brucei*, these drugs are usually successful except where trypanosomes have developed resistance to the drug or in some very chronic cases.

Additional comments made for treatment and control of *T. brucei* infections equally apply to *T. congolense*.

Notes: *Trypanosoma congolense congolense* is the most important trypanosome of cattle in tropical Africa. The African disease nagana is caused by *T. congolense*, often in mixed infection with *T. brucei* and *T. vivax*.

Trypanosoma vivax

Common names: Nagana, souma

Predilection site: Blood

Phylum: Euglenozoa

Class: Kinetoplastea

Family: Trypanosomatidae

Subgenus: *Duttonella*

Description: *Trypanosoma vivax* is monomorphic, ranging from 20 to 27 μm. The undulating membrane is inconspicuous, the large kinetoplast is terminal and the posterior end is broad and rounded. A short free flagellum is present (Fig. 8.30; see also Fig. 2.4). In fresh blood films, *T. vivax* moves rapidly across the microscope field.

Hosts: Cattle, sheep, goat, camel, horse; antelope and giraffe are reservoirs.

Geographical distribution: Central Africa, West Indies, Central and South America (Brazil, Venezuela, Bolivia, Colombia, Guyana, French Guiana), Mauritius

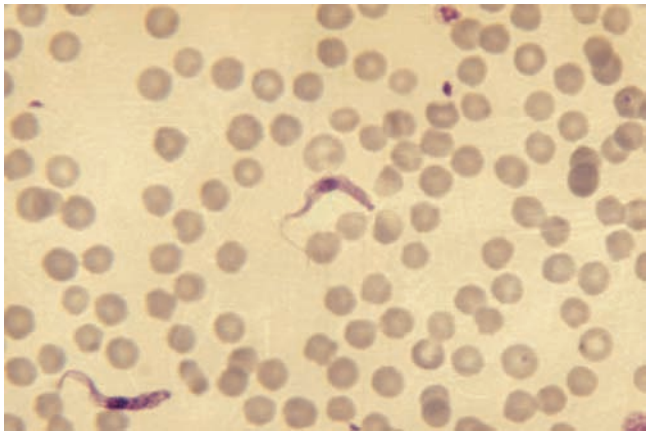

Figure 8.30 Tryptomastigotes of *Trypanosoma vivax*.

Pathogenesis: *Trypanosoma vivax* is most important in cattle. Generally, strains of *T. vivax* in West Africa are more pathogenic than ones in East Africa, except for one strain in East Africa that causes acute haemorrhagic disease which is very pathogenic.

Treatment: As for *T. congolense*

Notes: There are three subspecies.

- *Trypanosoma vivax vivax* causes the disease souma in Africa and is found in mixed infections with *T. congolense* and *T. brucei*.
- *Trypanosoma vivax viennei* occurs in the New World and is transmitted by horse flies. This subspecies occurs in cattle, horses, sheep and goats in northern South America, Central America, West Indies and Mauritius.
- *Trypanosoma vivax uniforme* is similar to *T. vivax vivax* but is smaller, 12–20 μm long (mean 16 μm). It occurs in cattle, sheep, goats and antelopes in Uganda and Zaire, causing a disease similar to that of *T. vivax vivax*.

Salivarian trypanosomosis control This currently depends on the control of tsetse flies, discussed under Tsetse flies (*Glossina* spp.) in Chapter 17, and on the use of drugs (Table 8.5).

In cattle, and if necessary in sheep and goats, isometamidium is the drug of choice since it remains in the tissues and has a prophylactic effect for 2–6 months. Otherwise, diminazene may be used as cases arise, these being selected either by clinical examination or on the haematological detection of anaemic animals. To reduce the possible development of drug resistance it may be advisable periodically to change from one trypanocidal drug to another. To further enhance the effective use of trypanocidal drugs, they may be used as 'sanative' pairs and treatment restricted to individual clinically affected animals.

Table 8.5 Drugs used in the treatment and control of Nagana in cattle.

Drug	Recommended dose	Comments
Diminazene aceturate	3–10 mg/kg i.m.	*T. brucei*, *T. congolense*, *T. vivax*
Isometamidium	0.25–1 mg/kg i.m.	*T. brucei*, *T. congolense*, *T. vivax* Local reaction
Homidium bromide Homidium chloride	1 mg/kg s.c.	*T. congolense*, *T. vivax* Prophylaxis for 6 weeks
Pyrithidium bromide	2–2.5 mg/kg	*T. congolense*, *T. vivax* Prophylaxis for 4 months

Two important aspects of control are:

- the necessity to protect cattle from a tsetse-free zone while being trekked to market through an area of endemic trypanosomosis;
- an awareness of the dangers of stocking a tsetse-free ranch with cattle from areas where trypanosomosis is present, as mechanical transmission may cause an outbreak of disease.

In both cases treatment with a trypanocidal drug at an appropriate time is advisable.

An alternative approach, using trypanotolerant breeds of ruminants, perhaps combined with judicious drug therapy, may in the future offer a realistic solution in many areas where the disease is endemic and this aspect is currently under intensive study.

Stercorarian trypanosomes

These are relatively large trypanosomes found in the blood of cattle, with faecal transmission by tabanid flies (*Tabanus*, *Haematopota*).

Trypanosoma theileri

Predilection site: Blood

Phylum: Euglenozoa

Class: Kinetoplastea

Family: Trypanosomatidae

Subgenus: Megatrypanum

Description: Large trypanosome, 60–70 μm in length, although may be up to 120 μm with posterior end long and pointed (Fig. 8.31; see also Fig. 2.7). There is a medium-sized kinetoplast with a prominent undulating membrane and a free flagellum. Both trypomastigote and epimastigote forms may appear in the blood.

Hosts: Cattle

Geographical distribution: Worldwide

Pathogenesis: Infection produces transient parasitaemias, but is generally considered to be non-pathogenic. Under conditions of stress it may cause abortion and even death.

Clinical signs: Infections are usually asymptomatic.

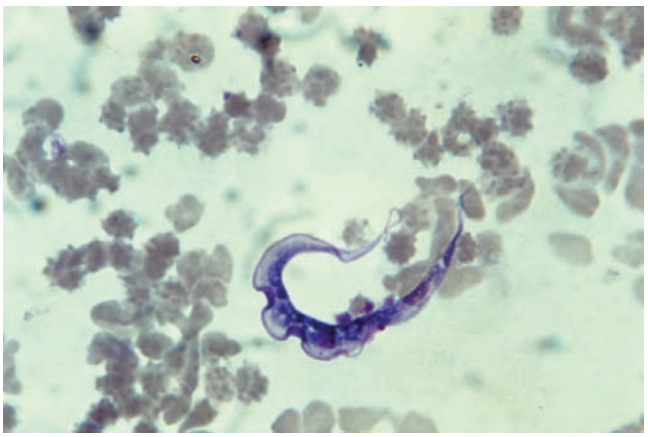

Figure 8.31 Tryptomastigotes of *Trypanosoma theileri*.

Diagnosis: Can only be usually diagnosed by incubating blood in culture medium suitable for the multiplication of trypanosomes.

Epidemiology: *Trypanosoma theileri* is transmitted by tabanid flies (*Tabanus*, *Haematopota*); the worldwide distribution of the trypanosome corresponds to the range and prevalence of its intermediate hosts. The metacyclic tryptomastigotes, present in the faeces of the vector, gain access to the blood of their mammalian host by penetrating abraded skin, by contamination of mucous membranes, or following ingestion of the vector when the liberated trypanosomes penetrate the mucosa. Intrauterine infection has been reported.

Treatment and control: Not usually required, although general fly control measures may help limit potential transmission from tabanid flies.

Notes: Often referred to as a 'non-pathogenic trypanosome'.

Babesiosis

Babesia are intraerythrocytic parasites of domestic animals and are transmitted by ticks. Babesiosis is particularly severe in naive cattle introduced into endemic areas and is a considerable constraint on livestock development in many parts of the world.

Epidemiology: The epidemiology of the bovine *Babesia* species depends on the interplay of a number of factors.

1. The virulence of the particular species of *Babesia*. *Babesia bigemina* and *B. bovis* in tropical and subtropical regions are highly pathogenic, *B. divergens* in northern Europe is relatively pathogenic, while *B. major* produces only mild and transient anaemia.
2. The age of the host. It is frequently stated that there is an inverse age resistance to *Babesia* infection in that young animals are less susceptible to babesiosis than older animals. The reason for this is not known.
3. The immune status of the host. In endemic areas, the young animal first acquires immunity passively, in the colostrum of the dam and, as a result, often suffers only transient infections with mild clinical signs. However, these infections are apparently sufficient to stimulate active immunity, although recovery is followed by a long period during which they are carriers when, although showing no clinical signs, their blood remains infective to ticks for many months. It used to be thought that this active immunity was dependent on the persistence of the carrier state and the phenomenon was termed 'premunity'. However, it seems unlikely that this is the case since it is now known that such animals may lose their infection either naturally or by chemotherapy, but still retain a solid immunity.
4. The level of tick challenge. In endemic areas, where there are many infected ticks, the immunity of the host is maintained at a high level through repeated challenge and overt disease is rare. In contrast, where there are few ticks or when they are confined to limited areas, the immune status of the population is low and the young animals receive little if any colostral protection. If, in these circumstances, the number of ticks suddenly increase due to favourable climatic conditions or to a reduction in dipping frequency, the incidence of clinical cases may rise sharply. This situation is known as enzootic instability.
5. Stress. In endemic areas, the occasional outbreak of clinical disease, particularly in adult animals, is often associated with some form of stress, such as parturition or the presence of another disease, such as tick-borne fever.

Babesia bigemina

Common name: Texas fever

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Babesiidae

Description: *Babesia bigemina* is a large pleomorphic babesia but characteristically is seen and identified by the pear-shaped bodies joined at an acute angle within the mature erythrocyte (Fig. 8.32). Round forms measure 2 μm and the pear-shaped elongated ones 4–5 μm.

Hosts: Cattle, buffalo

Geographical distribution: Australia, Africa, North, Central and South America, Asia and southern Europe

Pathogenesis: The rapidly dividing parasites in the red cells produce rapid destruction of the erythrocytes with accompanying haemoglobinaemia, haemoglobinuria and fever.

Generally, *B. bigemina* infections are not as virulent as those of *B. bovis*, despite the fact that the parasites may infect 40% of the red cells. Otherwise the disease is typically biphasic, the acute haemolytic crisis, if not fatal, being followed by a prolonged period of recovery.

Clinical signs: Calves are relatively resistant to infection and do not usually show clinical disease. In older animals, clinical signs can be very severe; however, differences in pathogenicity may occur with various *B. bigemina* isolates associated with different geographical areas. The first sign is usually a high fever with rectal temperatures reaching 41.5°C (106.7°F). There is anorexia and ruminal atony. Often the first visible appearance of infection is that the animal isolates itself from the herd, becomes uneasy, seeks shade and may lie down. Cattle may stand with an arched back, have a roughened hair coat and show evidence of dyspnoea and tachycardia. The mucous membranes are first inflamed and reddened, but as erythrocytic lysis occurs they become pallid and show signs of anaemia. Anaemia is a contributory factor to the weakness and loss of condition seen in cattle that survive the acute phase of the disease. The anaemia may occur very rapidly, with 75% or more of the erythrocytes being

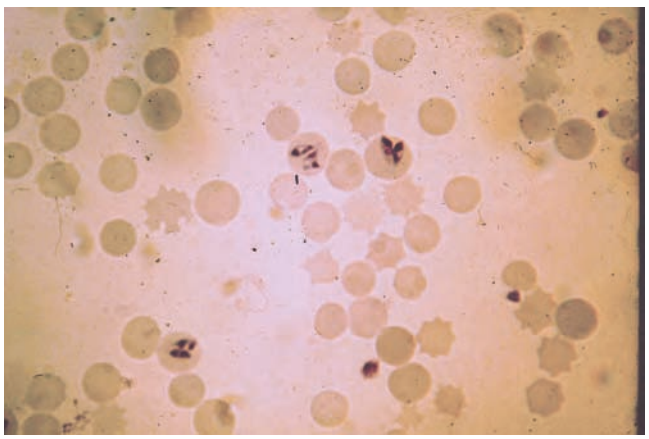

Figure 8.32 Intraerythrocytic stages of *Babesia bigemina*.

destroyed in just a few days. This is usually associated with severe haemoglobinaemia and haemoglobinuria. After onset of fever, the crisis will usually pass within a week, and if the animal survives there is usually severe weight loss, drop in milk production, possible abortion and a protracted recovery. Mortality is extremely variable and may reach 50% or higher, but in the absence of undue stress most animals will survive.

Diagnosis: As for *B. bovis*.

Pathology: Acute infections as for *B. bovis*. In cattle that have suffered a more prolonged illness, acute lesions are much less conspicuous. Subepicardial petechial haemorrhages may be present, the carcass is usually emaciated and icteric, the blood is thin and watery, the intermuscular fascia is oedematous, the liver yellowish-brown, and the bile may contain flakes of semi-solid material. The kidneys are pale and often oedematous, and the bladder may contain normal urine, depending on how long after the haemolytic crisis the necropsy is performed. Although the spleen is enlarged, the pulp is firmer than in acute babesiosis.

Epidemiology: *Rhipicephalus* (*Boophilus*) *annulatus*, *Rhipicephalus* (*Boophilus*) *microplus* and *Rhipicephalus* (*Boophilus*) *decoloratus* are the principal vectors of *B. bigemina*. Mechanical transmission is possible, but it is not efficient enough to maintain infection in the absence of specific tick vectors.

Treatment: As with *B. bovis*, successful treatment of *B. bigemina* depends on early diagnosis and the prompt administration of effective drugs. If medication is administered early, success is the rule, for there are several effective compounds. One of the first successful treatments was trypan blue. This treatment may be used to determine the type of infection present: *B. bigemina* is susceptible to trypan blue treatment, whereas *B. bovis* is not. Generally, the small babesias are more resistant to chemotherapy. The most commonly used compounds for treatment are diminazene diaceturate (3–5 mg/kg), imidocarb (1–3 mg/kg) and amicarbalide (5–10 mg/kg); however, the quinuronium and acridine derivatives are also effective where these are available. Treatment of *B. bigemina* is so effective in some instances that radical cures occur that will eventually leave the animal susceptible to reinfection. For this reason, reduced drug levels are sometimes indicated. Imidocarb has been successfully used as a chemoprophylactic that will prevent clinical infection for as long as 2 months, but will allow mild subclinical infection to occur as the drug level wanes resulting in premunition and immunity.

Control: Specific control measures are not usually necessary for animals born of mothers in endemic areas, since their colostrum-acquired immunity is gradually reinforced by repeated exposure to infection. Indeed, the veterinary importance of babesiosis is chiefly that it acts as a constraint to the introduction of improved livestock from other areas. Areas of enzootic instability also create problems when tick numbers suddenly increase or animals, for some reason, are forced to use an adjacent tick-infested area.

Immunisation, using blood from carrier animals, has been practised for many years in tropical areas, and more recently in Australia; rapidly passaged strains of *Babesia*, which are relatively non-pathogenic, have been widely utilised in live vaccines. In the near future, these may be superseded by adjuvanted vaccines prepared from several recombinant *Babesia* antigens. Otherwise the control of babesiosis in susceptible animals introduced into endemic areas depends on surveillance for the first few months after their arrival and, if necessary, treatment.

Vaccination of cattle against *B. bigemina* infection is commonly practised in many countries by inoculating blood from donor animals. This is usually obtained from a recently recovered case, any untoward reactions in the 'vaccinates' being controlled by babesicidal drugs. In Australia, the procedure is more sophisticated in that the vaccine is produced from acute infections produced in splenectomised donors. For economy, the blood is collected by exchange transfusion rather than by exsanguination. It is interesting that the rapid passage of the parasite by blood inoculation in splenectomised calves has fortuitously had the very desirable effect of decreasing the virulence of the infection in non-splenectomised calves to the extent that post-vaccination surveillance of cattle is frequently not performed. The parasite count of the blood determines the dilution of the latter, which is dispensed in plastic bags, packed in ice and despatched in insulated containers. Each dose of vaccine contains about 10 million parasites. Most of the vaccine is used in cattle under 12 months of age living in conditions of enzootic instability. The degree of protection induced is such that only 1% of vaccinated cattle subsequently develop clinical babesiosis from field challenge, compared with 18% of unvaccinated cattle.

The primary disadvantage of red cell vaccines is their lability and the fact that unless their preparation is carefully supervised, they may spread diseases such as enzootic bovine leucosis. Obviously there will be no such problem with a vaccine based on recombinant antigens.

A regimen of four injections of long-acting oxytetracycline at weekly intervals, administered to naive cattle during their first month of grazing on tick-infested pastures, has been shown to confer prophylaxis against *B. bigemina* during this period, after which the cattle were immune to subsequent challenge.

Notes: *Babesia bigemina*, a large babesia, is of particular interest historically since it was the first protozoan infection of humans or animals demonstrated to have an arthropod intermediate host. This was shown in 1893 by Smith and Kilborne while investigating the cause of the locally known 'Texas fever' in cattle in the USA. The disease has since been eradicated in that country.

Babesia bovis

Synonym: Babesia argentina

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Babesiidae

Description: *Babesia bovis* is a small pleomorphic babesia, typically identified as a single body, as small round bodies, or as paired pear-shaped bodies joined at an obtuse angle within the centre of the mature erythrocyte. The round forms measure 1–1.5 μm and the pear-shaped bodies 1.5 by 2.4 μm in size. Vacuolated signet ring forms are especially common.

Hosts: Cattle, buffalo, deer

Geographical distribution: Australia, Africa, Central and South America, Asia and southern Europe

Pathogenesis: *Babesia bovis* is generally regarded as the most pathogenic of the bovine babesia. Although the classical signs of

fever, anaemia and haemoglobinuria occur, the degree of anaemia is disproportional to the parasitaemia since haematocrit levels below 20% may be associated with infections of less than 1% of the red cells. The reason for this is unknown. In addition, *B. bovis* infection is associated with sludging of the red cells in the small capillaries. In the cerebrum this causes blockage of the vessels by clumps of infected red cells, leading to anoxia and tissue damage. The resulting clinical signs of aggression, incoordination or convulsions and depression are invariably fatal. Finally, recent work has indicated that some of the severity of *B. bovis* infection may be associated with the activation of certain plasma components, leading to circulatory stasis, shock and intravascular coagulation.

Clinical signs: Incoordination, convulsions, depression, death

Diagnosis: The history and clinical signs of fever, anaemia, jaundice and haemoglobinuria in cattle located in enzootic areas where *Rhipicephalus* (*Boophilus*) ticks occur are usually sufficient to justify a diagnosis of babesiosis. For confirmation, the examination of blood films stained with Giemsa will reveal the parasites in the red cells. However, once the acute febrile phase has subsided they are often impossible to find since they are rapidly removed from the circulation. In addition, a technique of brain biopsies has been described that has proven very useful in detecting and diagnosing *B. bovis* infections. The characteristic low parasitaemias in the circulating blood make this technique very useful in improving the chances of seeing the organism. There is a marked concentration of infected erythrocytes in the capillaries of the brain.

From each animal six blood smears should be made, air-dried and fixed in methanol and/or a sample of whole blood in an anticoagulant and serum should be collected. In cases of chronic infection, diagnosis is usually made using a variety of serological tests for the detection of specific antibodies, since the organism disappears or is present in extremely low numbers soon after the acute infection. Presently, immunofluorescence assay is the test of choice in the serologic diagnosis of *B. bovis*.

Other conditions that should be considered and which may resemble babesiosis are anaplasmosis, trypanosomosis, theileriosis, leptospirosis, bacillary haemoglobinuria, haemobartonellosis and eperythrozoonosis.

Pathology: At necropsy, the carcass is pale and jaundiced and the lungs may be oedematous and congested in cattle that have died early in the course of infection. The pericardial sac may contain serosanguineous fluid and subepicardial and subendocardial petechial haemorrhages. The liver is enlarged and icteric, and the gallbladder, which may have haemorrhage on the mucous surface, is distended with thick dark-green bile. The spleen is markedly enlarged, and has a dark pulpy consistency. The abomasal and intestinal mucosa may be icteric with patches of subserosal haemorrhages (Fig. 8.33). The blood is thin and watery. The urinary bladder is frequently distended, with dark reddish-brown urine. Jaundice is commonly distributed in the connective tissue. The lymph nodes are oedematous and often have petechiation.

Epidemiology: *Babesia bovis* is transmitted by the same ticks that transmit *B. bigemina*, i.e. *Rhipicephalus* (*Boophilus*) *annulatus* and *Rhipicephalus* (*Boophilus*) *microplus*. The tick *Rhipicephalus* (*Boophilus*) *decoloratus*, which is widely distributed in Africa, does not appear to transmit *B. bovis* even though it readily transmits *B. bigemina*. There are reports from Europe of *B. bovis*, for which the vector is thought to be *Ixodes ricinus*.

Figure 8.33 Postmortem findings with *Babesia bovis* infections.

Treatment: Successful treatment depends on early diagnosis and the prompt administration of effective drugs. There is less likelihood of success if treatment is delayed until the animal has been weakened by fever and anaemia. Chemotherapy is generally effective, although *B. bovis* is usually somewhat more difficult to treat than other *Babesia* species, and a second treatment, or slightly increased dose rates, may be desirable. The most commonly used compounds for the treatment of babesiosis are diminazene diaceturate (3–5 mg/kg), imidocarb (1–3 mg/kg) and amicarbalide (5–10 mg/kg); however, the quinuronium and acridine derivatives are also effective where these are available. Trypan blue is not effective against *B. bovis*.

Control: The numbers of ticks and therefore the quantum of *Babesia* infection may be reduced by regular spraying or dipping with acaricides. In addition, the selection and breeding of cattle which acquire a high degree of resistance to ticks is practised, particularly in Australia. Widespread use of tick vaccines may also have a significant influence on the incidence of infection in cattle (see control of *B. bigemina*).

Repeated passage of *B. bovis* in splenectomised calves results in the attenuation of the organism and for many years this attenuated vaccine has been produced and successfully used in Australia for the prevention of *B. bovis*. In some cattle (older, and producing dairy cows), chemotherapy may be indicated, but usually the vaccine may be used without treatment.

The development of *in vitro* techniques for the cultivation of *B. bovis* on bovine erythrocytes has led to the isolation of soluble antigens which, when combined with adjuvants, have proven immunogenic. Although they do not prevent infection, these non-infectious vaccines appear to be responsible for moderating the effects of infection. They do not produce as high a level of protection as seen with premunising vaccines but are safe and do not yield carriers. In some instances, these vaccines, although protective against homologous challenge, may not protect against immunological variants. The continuous *in vitro* passage of *B. bovis* has been shown to induce a level of attenuation similar to that seen with passage of the organism in splenectomised calves and infection with this attenuated organism has been reported to prevent clinical infection following a challenge with virulent *B. bovis*. The primary disadvantage of red cell vaccines is their lability and the fact that, unless their preparation is carefully supervised, they may spread diseases such as enzootic bovine leucosis. Obviously there will be no such problem with a vaccine based on recombinant antigens.

Babesia divergens

Common name: Redwater fever

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Babesiidae

Description: Examination of stained blood films shows the organisms to be within red cells, almost always singly or as pairs, often arranged at a characteristic angle with their narrow ends opposed. Typically, they are pyriform, but may be round, elongated or cigar-shaped. *Babesia divergens* is a 'small babesia' and in blood films typically appears as paired, widely divergent organisms, 1.5 by 0.4 µm, lying near the edge of the red cell (see Fig. 2.31). Other forms may be present measuring 2 by 1 µm, some are circular up to 2 µm in diameter and a few may be vacuolated.

Hosts: Cattle

Geographical distribution: Northern Europe

Pathogenesis: The rapidly dividing parasites in the red cells produce rapid destruction of the erythrocytes with accompanying haemoglobinaemia, haemoglobinuria and fever. This may be so acute as to cause death within a few days, during which the PCV falls below 20%. The parasitaemia, which is usually detectable once the clinical signs appear, may involve between 0.2 and 45% of the red cells. Milder forms of the disease, associated with relatively resistant hosts, are characterised by fever, anorexia and perhaps slight jaundice for a period of several days.

Clinical signs: Typically the acute disease occurs 1–2 weeks after the tick commences to feed and is characterised by fever and haemoglobinuria ('redwater'). The mucous membranes, at first congested, become jaundiced, the respiratory and pulse rates are increased, the heartbeat is usually very audible, and in cattle ruminal movements cease and abortion may occur. If untreated, death commonly occurs in this phase. Otherwise, convalescence is prolonged, there is loss of weight and milk production and diarrhoea followed by constipation is common. In animals previously exposed to infection, clinical signs may be mild or even inapparent.

Diagnosis: The history and clinical signs are usually sufficient to justify a diagnosis of babesiosis. For confirmation, the examination of blood films stained with Giemsa will reveal the parasites in the red cells (Fig. 8.34). However, once the acute febrile phase has subsided they are often impossible to find since they are rapidly removed from the circulation.

Pathology: At necropsy, the carcass is pale and jaundiced, the bile is thick and granular and there may be subepicardial and subendocardial haemorrhages.

Epidemiology: *Babesia divergens* is transmitted by *Ixodes ricinus*, and is widespread and pathogenic, with clinical cases occurring during the periods of tick activity, primarily in the spring and autumn. Infection in the tick is transovarially transmitted and the larvae, nymphs and adults of the next generation are all able to transmit infection to cattle.

Treatment: Amicarbalide, diminazene aceturate and imidocarb are the most commonly used drugs. Recently, long-acting preparations of oxytetracycline have been shown to have a prophylactic effect against *B. divergens* infection. Imidocarb, due to its persistence in the tissues, has a prophylactic effect for several weeks. During the convalescent phase of the disease, blood transfusions may be valuable as are drugs designed to stimulate food and water intake.

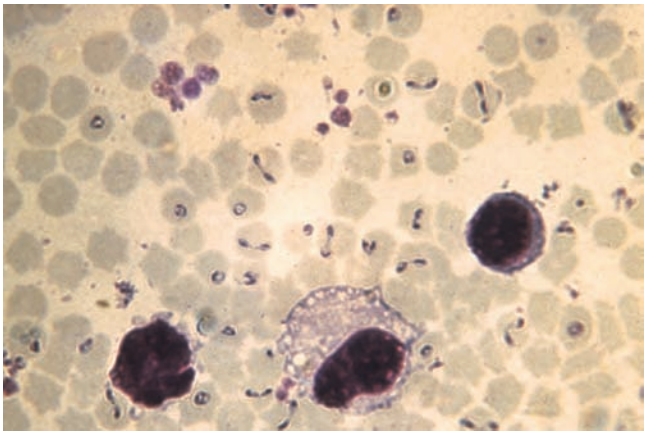

Figure 8.34 Intraerythrocytic stages of *Babesia divergens*.

Control: Normally no effort is made to control this infection in endemic areas, although cattle recently introduced require surveillance for some months since, on average, one in four will develop clinical disease and of these one in six will die if untreated. However, in some parts of mainland Europe, such as the Netherlands, where ticks are confined to rough vegetation on the edge of pastures and on roadsides, it is often possible to take evasive measures. It is thought that red and roe deer are not important reservoir hosts since only mild infections have been experimentally produced in splenectomised deer.

Notes: Since 1957, several cases of fatal babesiosis due to *B. divergens* infection have occurred in humans in the former Yugoslavia, Russia, Ireland and Scotland. In each case, the individual had been splenectomised sometime previously or was currently undergoing immunosuppressive treatment.

Babesia major

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Babesiidae

Description: A 'large babesia', with pyriform bodies 2.6 by 1.5 µm, being characteristically paired at an acute angle less than 90° and found in the centre of the erythrocyte (Fig. 8.35). Round forms about 1.8 µm in diameter may form.

Hosts: Cattle

Geographical distribution: Europe, North Africa, South America

Pathogenesis: *Babesia major* is only mildly pathogenic.

Clinical signs: Clinical signs with *B. major* are usually inapparent but where symptoms do occur these are characterised by a

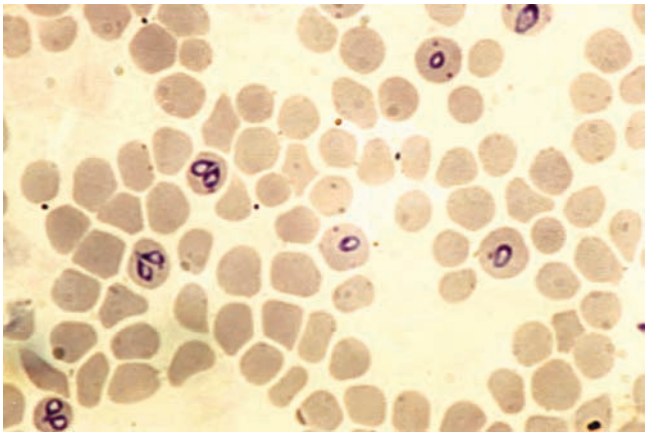

Figure 8.35 Intraerythrocytic stages of *Babesia major*.

haemolytic syndrome with elevated temperature, mild anaemia and haemoglobinuria.

Diagnosis: Examination of blood films stained with Giemsa will reveal the parasites in the red cells.

Epidemiology: *Babesia major* is transmitted by the three-host tick *Haemaphysalis punctata*.

Treatment: Not usually required but amicarbalide, diminazene aceturate and imidocarb are effective.

Control: Specific control measures are not usually necessary for animals born of mothers in endemic areas since, as noted previously, their colostrally acquired immunity is gradually reinforced by repeated exposure to infection. Tick numbers may be reduced by regular spraying or dipping with acaricides. The control of infection in susceptible animals introduced into endemic areas depends on surveillance for the first few months after their arrival and, if necessary, treatment.

Other species of Babesia in cattle

See Table 8.6.

Theileriosis

Diseases caused by several species of *Theileria* (theileriosis) are a serious constraint to livestock production in Africa, Asia and the Middle East. The parasites, which are tick transmitted, undergo repeated merogony in the lymphocytes ultimately releasing small merozoites that invade the red cells to become piroplasms.

Table 8.6 Other species of *Babesia* in cattle.

Species	Hosts	Vectors	Distribution
Babesia jakimovi	Cattle, deer (roe deer, elk, reindeer)	*Ixodes ricinus*	Northern Europe (Siberia)
Babesia ovata	Cattle	*Hyalomma longicornis*	Japan, China
Babesia occultans	Cattle	*Hyalomma marginatum rufipes*	Southern Africa

Theileria are widely distributed in cattle in Africa, Asia, Europe and Australia, have a variety of tick vectors and are associated with infections which range from clinically inapparent to rapidly fatal. Although the speciation of many *Theileria* is still controversial, largely because of their morphological similarity, there are two species of major veterinary importance in cattle. Minor and mildly pathogenic species infecting cattle include *T. velifera* and *T. taurotragi* in Africa, *T. mutans* and the *T. sergenti/orientalis/buffeli* complex.

Theileria parva

Subspecies: *Theileria parva parva, Theileria parva lawrencei*

Common name: East Coast fever, corridor fever

Predilection site: Blood and lymphatics

Phylum: Apicomplexa

Class: Aconoidasida

Family: Theileriidae

Description: Trophozoite forms in the erythrocyte are predominantly rod-shaped (1.5–2.0 × 0.1–1.0 μm), but may also be round, oval and comma-shaped (Fig. 8.36). Koch bodies are present in the lymphocytes and endothelial cells of the spleen or lymph nodes where they are very numerous and average 8 μm but can range up to 12 μm or more. Two types have been described: macroschizonts containing chromatin granules 0.4–2.0 μm in diameter (Fig. 8.37), these dividing further to become microschizonts that contain chromatin granules 0.3–0.8 μm in diameter and produce merozoites 0.7–1 μm in diameter.

Hosts: Cattle, buffalo

Geographical distribution: East and Central Africa

Pathogenesis: The sequence of events in a typical acute and fatal infection progresses through three phases, each spanning about 1 week. The first is the incubation period of about 1 week when neither parasite nor lesions can be detected. This is followed during the second week by marked hyperplasia and expansion of the infected lymphoblast population, initially in the regional lymph node draining the site of the tick bite and ultimately throughout the body. During the third week, there is a phase of lymphoid depletion and disorganisation

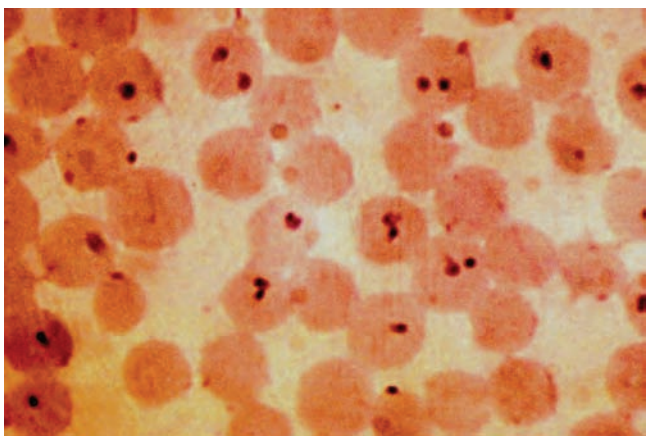

Figure 8.36 Intraerythrocytic stages of *Theileria parva*.

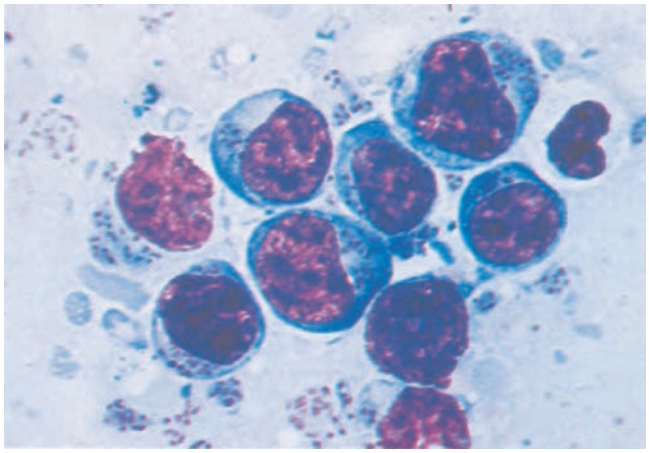

Figure 8.37 Macroschizonts of *Theileria parva* in a smear of a lymph node.

associated with massive lymphocytolysis and depressed leucopoiesis. The cause of the lymphocytolysis is unknown, but is due perhaps to the activation of natural killer cells like macrophages.

Theileria parva lawrencei is transmitted from the African buffalo and becomes indistinguishable in its behaviour from *T. parva parva* following several passages in cattle.

Clinical signs: About 1 week after infection, in a fully susceptible animal, the lymph node draining the area of tick bite, usually the parotid, becomes enlarged and the animal becomes pyrexic (40–41.7°C, 104–107°F). Within a few days there is generalised swelling of the superficial lymph nodes, ears, eyes and submandibular regions. The animal becomes anorexic, shows decreased milk production and rapidly loses condition, ceases rumination, becomes weak with a rapid heartbeat, and petechial haemorrhages may occur under the tongue and on the vulva. Affected animals become emaciated and dyspnoeic and there is terminal diarrhoea, often blood-stained. Recumbency and death almost invariably occur, usually within 3 weeks of infection. Occasionally nervous signs, the so-called 'turning sickness', have been reported and attributed to the presence of meronts in cerebral capillaries.

Milder infections show a mild fever lasting 3–7 days, listlessness and swelling of superficial lymph nodes.

Diagnosis: East Coast fever only occurs where *R. appendiculatus* is present, although occasionally outbreaks outwith such areas have been recorded due to the introduction of tick-infected cattle from an enzootic area. In sick animals, macroschizonts are readily detected in biopsy smears of lymph nodes and in dead animals in impression smears of lymph nodes and spleen. In advanced cases, Giemsa-stained blood smears show piroplasms in the red cells, up to 80% of which may be parasitised.

The indirect fluorescent antibody test is of value in detecting cattle which have recovered from East Coast fever.

Pathology: Necropsy during the terminal phase shows lymph nodes to be swollen, with atrophy of the cellular content of the lymph nodes and variable hyperaemia. The spleen is usually enlarged with soft pulp and prominent Malpighian corpuscles. The liver is enlarged, friable, brownish-yellow, with parenchymatous degeneration. The kidneys are either congested or pale brown, with variable number of infarcts. The meninges may be slightly congested. The heart is flabby, with petechiae on the epicardium and endocardium. The lungs are often congested and oedematous. There may be hydrothorax and hydropericardium, and the kidney capsule may contain a large amount of serous fluid. There may be petechiae in the visceral and parietal pleura, adrenal cortex, urinary bladder and mediastinum. There are characteristic ulcers 2–5 mm or more in diameter in the abomasum and small and large intestines. Peyer's patches are swollen, and the intestinal contents yellowish.

Epidemiology: Since the tick vector, *Rhipicephalus appendiculatus*, is most active following the onset of rain, outbreaks of East Coast fever may be seasonal or, where rainfall is relatively constant, may occur at any time. Fortunately, indigenous cattle reared in endemic areas show a high degree of resistance and, although transient mild infection occurs in early life, mortality is negligible. The mechanism of this resistance is unknown. However, such cattle may remain carriers and act as a reservoir of infection for ticks. Susceptible cattle introduced into such areas suffer high mortality, irrespective of age or breed, unless rigid precautions are observed.

In areas where survival of the tick vector is marginal, challenge is low and indigenous cattle may have little immunity. Such areas, during a prolonged period of rain, may become ecologically suitable for the survival and proliferation of the ticks, ultimately resulting in disastrous outbreaks of East Coast fever. In some parts of East and Central Africa where populations of cattle and wild African buffalo overlap there is an additional epidemiological complication due to the presence of a strain of *T. parva* known as *T. parva lawrencei*. This occurs naturally in African buffalo, many of which remain as carriers. The tick vector is also *R. appendiculatus* and, in cattle, the disease causes high mortality. Since infected ticks may survive for nearly 2 years, physical contact between buffalo and cattle need not be close.

Treatment: Although the tetracyclines have a therapeutic effect if given at the time of infection, they are of no value in the treatment of clinical cases. The drugs of choice in clinical cases of East Coast fever are the naphthaquinone compounds parvaquone and buparvaquone and the anticoccidial drug halofuginone.

Control: Traditionally, the control of East Coast fever in areas where improved cattle are raised has relied on legislation to control the movement of cattle, on fencing to prevent access by nomadic cattle and buffalo and on repeated treatment of cattle with acaricides. In areas of high challenge, such treatments may require to be carried out twice weekly in order to kill the tick before the infective sporozoites develop in the salivary glands. This is not only expensive, but creates a population of fully susceptible cattle; if the acaricide fails, through human error or the acquisition of acaricide resistance by the ticks, the consequences can be disastrous.

Great efforts have been made to develop a suitable vaccine, but these have been thwarted by the complex immunological mechanisms involved in immunity to East Coast fever and by the discovery of immunologically different strains of *T. parva* in the field. However, an 'infection and treatment' regimen that involves the concurrent injection of a virulent stabilate of *T. parva* and long-acting tetracycline has been shown to be successful, although it has not been used on a large scale as yet. Apparently, the tetracycline slows the rate of schizogony, giving the immune response time to develop.

Notes: Because of the wide distribution of its tick vector, *Rhipicephalus*, and the fact that infection in cattle introduced into enzootic areas can be associated with a mortality of 100%, *T. parva* infection is an immense obstacle to livestock improvement.

Theileria annulata

Common names: Mediterranean theileriosis, Mediterranean Coast fever

Predilection site: Blood and lymphatics

Phylum: Apicomplexa

Class: Aconoidasida

Family: Theileriidae

Description: Trophozoite forms in the erythrocyte are predominantly round (0.5–2.7 μm) to oval (2 × 0.6 μm), but may also be rod-shaped or comma-shaped (1.2 × 0.5 μm). Division by binary fission may form two or four daughter cells, the latter in the shape of a cross. Koch bodies are present in the lymphocytes of the spleen or lymph nodes or even free in these organs. They average 8 μm but can be up to 27 μm. Two types have been described: macromeronts containing chromatin granules 0.4–1.9 μm in diameter, these dividing further to become micromeronts that contain chromatin granules 0.3–0.8 μm in diameter and produce merozoites 0.7–1 μm in diameter.

Hosts: Cattle, domestic buffalo

Geographical distribution: Mediterranean countries (Portugal and Spain, the Balkans), the Middle East, Indian subcontinent and China

Pathogenesis: The pathogenesis and clinical signs are initially similar to those of East Coast fever with pyrexia and lymph node enlargement, but in the late stages there is a haemolytic anaemia and often icterus. Convalescence is protracted in those cases that recover.

Clinical signs: In the acute form there is fever (40–41.7°C, 104–107°F), inappetence, cessation of rumination, rapid heartbeat, weakness, decreased milk production, swelling of superficial lymph nodes and eyelids, diarrhoea (containing blood and mucus), jaundice and petechial haemorrhages. Affected animals become emaciated and death can occur. In the more chronic form there is intermittent fever, inappetence, emaciation, anaemia and jaundice.

Diagnosis: Diagnosis depends on the detection of meronts in both lymph node biopsy specimens and, unlike *T. parva*, in blood smears. A low-grade piroplasm parasitaemia, in the absence of schizonts, is usually indicative of a recovered carrier animal.

Pathology: The lymph nodes are often but not always swollen; the spleen is often much enlarged and infarcts are usually present in the kidneys. The lungs are usually oedematous; characteristic ulcers are present in the abomasum and small and large intestines.

Epidemiology: *Theileria annulata* is transmitted transtadially by ticks of the genus *Hyalomma*: *H. detritum* in North Africa; *H. detritum* and *H. excavatum* in the former Soviet states; *H. truncatum* in parts of Africa; *H. dromedarii* in central Asia; *H. excavatum*, *H. turanicum* and *H. marginatum* in Asia Minor; *H. marginatum* in India; and *H. longicornis* in Siberia and the Far East. Like East Coast fever, indigenous cattle in endemic areas are relatively resistant while improved cattle, particularly European breeds, are highly susceptible. However, unlike East Coast fever, the disease in such cattle is not uniformly fatal, although the mortality rate may reach 70%.

Congenital infection can occur occasionally in calves.

Treatment: See under *T. parva*

Control: In many areas, the prevention of *T. annulata* infection in imported dairy stock is based on permanent housing. However, this is expensive and there is always the possibility that infected ticks may be brought in with the fodder to cause disease and colonise crevices in the cattle accommodation. In some countries immunisation with meronts attenuated by prolonged *in vitro* culture has given excellent results.

Theileria orientalis complex

Synonyms: *Theileria mutans, Theileria buffeli, Theileria sergenti*

Common name: Benign theileriosis

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Theileriidae

Description: Trophozoite forms in erythrocytes are round (1–2 μm diameter), oval (1.5 × 0.6 μm), pyriform, or comma-shaped (Fig. 8.38). Binary fission produces two or four daughter cells. There are relatively few Koch bodies (8–20 μm) in the lymphocytes of the spleen and lymph nodes, which contain 1–80 chromatin granules (1–2 μm in diameter).

Hosts: Cattle, buffalo

Geographical distribution: Southern Europe, Middle East, Asia, Australia

Pathogenesis: Mildly pathogenic

Clinical signs: Similar in appearance to the mild form of *T. annulata* causing anaemia, with jaundice and lymphadenopathy occasionally present.

Diagnosis: Giemsa-stained blood smears may show piroplasms in the red cells, or macroschizonts may be detected in biopsy smears of lymph nodes.

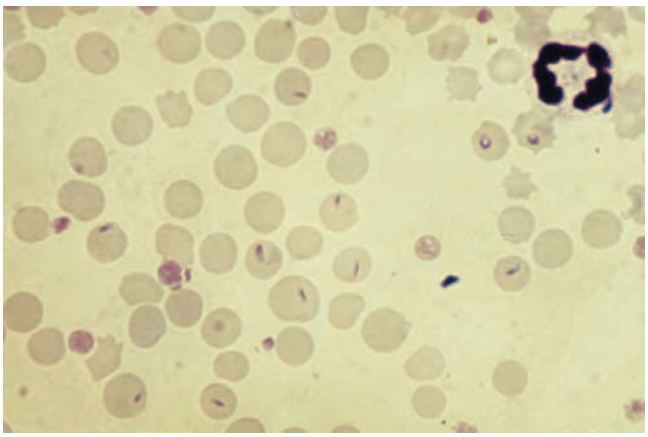

Figure 8.38 Intraerythrocytic stages of *Theileria orientalis* (*mutans*).

Pathology: In acute cases the spleen and liver are swollen, the lungs may be oedematous and there are characteristic ulcers in the abomasum; infarcts may be present in the kidneys. Macroschizonts may also be found in impression smears of lymph nodes and spleen taken from dead animals.

Epidemiology: Vectors are *Amblyomma variegatum, A. cohaerens* and *A. hebraeum*. *Haemaphysalis bispinosa* and *H. bancrofti* are the probable vectors in Australia.

Treatment: Little information is available on treatment, although the drugs of choice in clinical cases are likely to be parvaquone and buparvaquone.

Control: Tick control methods may be considered including fencing and dipping or cleaning cattle of ticks but these are not usually required.

Notes: The taxonomy of benign theileriosis species is complicated and it is now considered that *T. orientalis* is part of a complex with *T. sergenti*, *T. buffeli* and *T. mutans*.

Theileria taurotragi

Synonym: *Cytauxzoon taurotragi*

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Theileriidae

Description: Erythrocytic forms are similar in appearance to *T. parva*. Trophozoite forms in the erythrocyte are predominantly round to oval, but may also be rod-shaped or comma-shaped (1.2 × 0.5 μm).

Hosts: Cattle, antelope, particularly the eland (*Taurotragi oryx*)

Geographical distribution: Africa

Pathogenesis: Mildly pathogenic

Clinical signs: Mild transient fever and anaemia

Diagnosis: Presence of erythrocytic forms in blood smears or meronts in lymph node biopsy specimens. *Theileria taurotragi* is morphologically indistinguishable from more pathogenic forms, but generally differentiated on clinical signs and history.

Pathology: Meront stages have been reported in liver, lung and lymph nodes.

Epidemiology: Vectors are *R. appendiculatus* and *R. pulchellus*.

Treatment and control: Not usually required

Theileria velifera

Synonym: *Haematoxenus veliferus*

Predilection site: Blood

Phylum: Apicomplexa

Class: Aconoidasida

Family: Theileriidae

Description: Trophozoite forms in erythrocytes are pleomorphic and most often appear as small rods 1–2 μm long. The great majority have a rectangular 'veil' 1–3.5 μm extending out from the side.

Hosts: Cattle, zebu

Geographical distribution: Africa

Pathogenesis: Non-pathogenic

Clinical signs: Not reported

Diagnosis: Giemsa-stained blood smears may show the characteristic 'veiled' piroplasms in the red cells.

Pathology: No associated pathology

Epidemiology: Known vectors are *Amblyomma variegatum, A. lepidu and A. hebraeum*.

Treatment and control: Not usually required

Rickettsia

While the Rickettsia are now considered to be in the Kingdom Bacteria, for historical reasons they are included within parasitological texts and for this reason mention is made to some genera and species of importance in cattle.

Anaplasma marginale

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Anaplasmataceae

Description: In Giemsa-stained blood films the organisms of *A. marginale* are seen as small round dark-red 'inclusion bodies' approximately 0.3–1.0 μm within the red cell (Fig. 8.39). Often there is only one organism in a red cell and characteristically this lies at the outer margin; however, these two features are not constant.

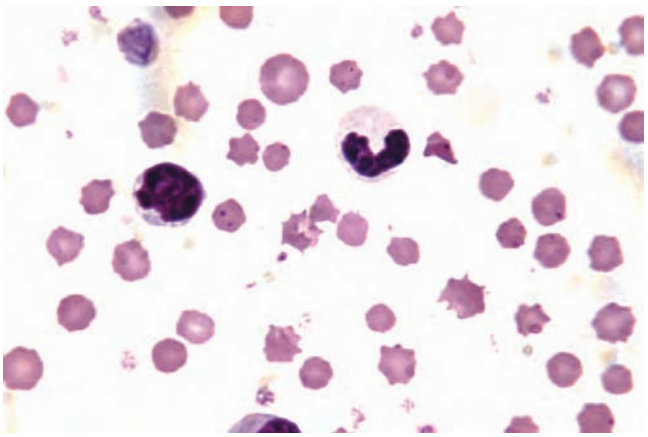

Figure 8.39 Intraerythrocytic stages of *Anaplasma marginale*.

Hosts: Cattle, wild ruminants

Geographical distribution: Africa, southern Europe, Australia, South America, Asia, former Soviet states and USA

Pathogenesis: Typically, the changes are those of an acute febrile reaction accompanied by a severe haemolytic anaemia. After an incubation period of around 4 weeks, fever and parasitaemia appear, and as the latter develops the anaemia becomes more severe so that within a week or so up to 70% of the erythrocytes are destroyed. The clinical signs are usually very mild in naive cattle under 1 year old. Thereafter, susceptibility increases so that cattle aged 2–3 years develop typical and often fatal anaplasmosis, while in cattle over 3 years the disease is often peracute and frequently fatal.

Clinical signs: Clinical signs are attributed to severe anaemia and include depression, weakness, fever, laboured breathing, inappetance, dehydration, constipation and jaundice. The acute stage of the disease is characterised by fever (39.4–41.7°C, 103–107°F) that persists for 3–7 days. During the febrile phase there is decreased rumination, dryness of the muzzle, loss of appetite, dullness and depression. Lactating cows show a depression in milk yield and abortion is a common feature in advanced pregnancy. The severity of the disease increases with age, with animals over 3 years of age showing the peracute and possibly fatal disease.

Diagnosis: The clinical signs, supplemented if possible by haematocrit estimation and the demonstration of *Anaplasma* inclusions in the red cells, are usually sufficient for diagnosis. For the detection of immune carriers, complement fixation and agglutination tests are available; an indirect fluorescent antibody test and DNA probe have also been developed.

Pathology: Gross pathological lesions are those usually associated with anaemia. Mucous membranes are jaundiced and there is pallor of the tissues. The spleen is often greatly enlarged with enlarged splenic follicles. The liver may be enlarged with rounded borders. The gallbladder is enlarged and obstructed with dark thick bile. Petechiae may be observed on the epicardium, pericardium, pleura and diaphragm. The lymph glands are enlarged. Microscopically there is hyperplasia of the bone marrow. The spleen shows a decrease in lymphoblasts and increased vacuolation and degeneration of reticular cells and there is reduction of the white pulp and accumulation of pigment resembling haemosiderin.

Epidemiology: The organism is distributed throughout the tropics corresponding to the distribution of the main tick vectors, *Rhipicephalus* (*Boophilus*) *annulatus*, *Rhipicephalus* (*Boophilus*) *decoloratus* and *Rhipicephalus* (*Boophilus*) *microplus*. In the USA, the main tick vectors are *Dermacentor andersoni*, *D. occidentalis* and *D. variabilis*. Horse flies (Tabanidae), stable flies (*Stomoxys*), deer flies (*Chrysops*), horn flies and mosquitoes have also been incriminated as potential vectors. Reservoirs of infection are maintained in carrier cattle and in wild ruminants such as deer. Cattle, especially adults, introduced into endemic areas are particularly susceptible, the mortality rate being up to 80%. In contrast, cattle reared in endemic areas are much less susceptible, presumably due to previous exposure when young, although their acquired immunity usually coexists with a carrier state. This balance may, on occasions, be disturbed and clinical anaplasmosis supervenes when cattle are stressed by other diseases such as babesiosis.

Treatment: Tetracycline compounds are effective in treatment if given early in the course of the disease and especially before the parasitaemia has reached its peak. More recently, imidocarb has been shown to be effective and may also be used to sterilise carrier animals.

Control: Vaccination of susceptible stock with small quantities of blood containing the mildly pathogenic *A. centrale* or a relatively avirulent strain of *A. marginale* is practised in several countries, any clinical signs in adults being controlled by drugs. In the USA, a killed *A. marginale* vaccine containing erythrocyte stroma is also available. Although all are generally successful in the clinical sense, challenged cattle become carriers and so perpetuate transmission. The killed vaccine has the disadvantage that antibodies produced to the red cell stroma, if transferred in the colostrum, may produce isoerythrolysis in nursing calves. Improved inactivated vaccines are currently under development. Otherwise, control at present depends largely on the reduction of ticks and biting flies.

Anaplasma centrale

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Anaplasmataceae

Description: As for *A. marginale*, except that the organisms are commonly found in the centre of the erythrocyte.

Hosts: Cattle, wild ruminants (and perhaps sheep may act as reservoirs of infection)

Geographical distribution: Worldwide in tropics and subtropics including southern Europe. It is also present in some temperate areas, including parts of the USA.

Pathogenesis: Similar to *A. marginale* but generally considered to be less pathogenic.

Clinical signs: The clinical features include pyrexia, anaemia and often jaundice, anorexia, laboured breathing and, in cows, a severe drop in milk yield or abortion. Occasionally peracute cases occur, which usually die within a day of the onset of clinical signs.

Pathology: Necropsy at this time often reveals a jaundiced carcass, a grossly enlarged gallbladder and, on section, a liver suffused with bile. The spleen and lymph nodes are enlarged and congested and there are petechial haemorrhages in the heart muscle. The urine, unlike that in babesiosis, is normal in colour. In survivors, recovery is prolonged.

Epidemiology: Apart from the various modes of transmission previously described, little information is available. Reservoirs of infection are maintained in carrier cattle and perhaps in wild ruminants or sheep. Cattle, especially adults, introduced into endemic areas are particularly susceptible, the mortality rate being up to 80%. In contrast, cattle reared in endemic areas are much less susceptible, presumably due to previous exposure when young, although their acquired immunity usually coexists with a carrier state. This balance may, on occasion, be disturbed and clinical anaplasmosis supervenes when cattle are stressed by other diseases such as babesiosis.

Details on the life cycle, diagnosis, treatment and control are as for *A. marginale*.

Anaplasma phagocytophilum

Synonyms: *Anoplasma phagocytophila, Ehrlichia phagocytophila, Cytoecetes phagocytophila*

Common names: Tick-borne fever, pasture fever, canine granulocytic ehrlichiosis, human granulocytic ehrlichiosis, equine granulocytic ehrlichiosis

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Anaplasmataceae

Description: Blood smears stained with Giemsa or Wright's stain reveal one or more loose aggregates (morulae or inclusion bodies, 1.5–5 μm in diameter) of blue–grey to dark-blue coccoid, coccobacillary or pleomorphic organisms within the cytoplasm of neutrophils (see Fig. 9.56).

For more detailed descriptions on pathogenesis, epidemiology, treatment and control see Chapter 9.

Ehrlichia bovis

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Anaplasmataceae

Description: Round or irregular-shaped intracytoplasmic organisms (2–10 μm in diameter) present in mononuclear cells, particularly monocytes.

Hosts: Cattle

Geographical distribution: Africa, Middle East (Turkey, Iran), India, Sri Lanka

Pathogenesis: Has been associated with acute and fatal disease in some regions of Africa.

Clinical signs: Affected animals show anorexia, weakness, muscular trembling, drunken gait and bulging eyes.

Diagnosis: The rickettsiae can be demonstrated by staining blood or organ smears with Giemsa.

Pathology: In fatal cases there is hydropericardium, hydrothorax, splenomegaly and swollen lymph nodes. Monocytosis may occur in terminal infections.

Epidemiology: Transmitted by ticks of the genera *Hyalomma, Rhipicephalus and Amblyomma.* Known vectors are *Hyalomma anatolicum, Rhipicephalus appendiculatus, Amblyomma cajennense* and possibly *A. variegatum*.

Treatment: Little information is available although, as with other member of this group, tetracyclines may be effective.

Control: Specific control measures have not been reported but tick control may assist in preventing infection with *E. bovis*.

Ehrlichia ruminantium

Synonym: *Cowdria ruminantium*

Common names: Heartwater, cowdriosis, malkopsiekte (Afrikaans)

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Anaplasmataceae

Description: Organisms are seen as close-packed colonies consisting of less than ten to many hundred cocci. The organism varies in size from 0.2 to greater than 1.5 μm. The diameter of individual organisms in a given cluster is rather uniform but groups are very pleomorphic. The small granules tend to be coccoid, with larger ones looking like rings, horseshoes, rods and irregular masses.

Hosts: Cattle, sheep, goat, buffalo and wild ruminants

Geographical distribution: Africa, south of the Sahara; Caribbean (Guadeloupe, Marie-Galante and Antigua)

Pathogenesis: In the ruminant host the organism is first found in reticuloendothelial cells and then parasitises vascular endothelial cells. Division is by binary fission and it produces morula-like colonies in the cytoplasm of infected cells. The pathogenesis of the disease is far from clear. Hydropericardium may lead to cardiac insufficiency and hydrothorax and pulmonary oedema to respiratory difficulties. Oedema is often so pronounced in peracute heartwater that it is responsible for sudden death by asphyxia. The occasional sudden fall in plasma volume preceding death has been associated with the development of the transudates. Brain lesions are not sufficiently consistent to explain the nervous symptoms.

Clinical signs: The average natural incubation period is 2 weeks, but can vary from 10 days to 1 month. In most cases, heartwater is an acute febrile disease, with a sudden rise in body temperature; temperature may exceed 41°C within 1–2 days. It remains high with small fluctuations and drops shortly before death.

A peracute form occurs in exotic breeds introduced into an endemic region. The animal appears clinically normal, but if examined will have a marked pyrexia. It may then suddenly collapse, go into convulsions and die. Thoracic auscultation will often reveal oedema in the lungs and bronchi.

In the acute form, fever is followed by inappetence, sometimes listlessness, diarrhoea (particularly in cattle) and dyspnoea indicative of lung oedema. The course of infection is 3–6 days and consists of pyrexia (often over 41°C, 106°F). A mild cough may be heard and, on auscultation, hydrothorax, hydropericardium and lung

oedema are noted. A profuse diarrhoea is often present or there may be blood in the faeces. Nervous signs develop gradually. The animal is restless, walks in circles, makes sucking movements and stands rigidly with tremors of the superficial muscles. Cattle may push their head against a wall or present aggressive or anxious behaviour. Finally, the animal falls to the ground, pedalling and exhibiting opisthotonus, nystagmus and chewing movements. The animal usually dies during or following such a nervous attack.

In the subacute form, the signs are like those of the acute form but they are much less severe with a transient fever and sometimes diarrhoea. Disease may last for over a week and the animal usually improves gradually but a few cases progress to collapse and death. This is often the most severe form seen in indigenous cattle and those previously infected. In these stock, symptoms are usually absent.

Diagnosis: There is no specific method for diagnosis in the living animal. A tentative diagnosis of heartwater is based on the presence of *Amblyomma* vectors, of clinical nervous signs and of transudates in the pericardium and thorax at postmortem examination. Provisional indication can be gained from the history and clinical signs. Lymph node material can be aspirated to examine for vacuoles containing organisms in the cytoplasm of the reticular cells. Serum can be examined using a capillary flocculation test. A number of serological tests have been described but all suffer from false-positive reactions due to cross-reactions with other *Ehrlichia* species.

Diagnosis is easier at postmortem as the organism can be discerned in brain tissue capillaries that have been fixed in methyl alcohol and stained with Giemsa. Typical colonies of *E. ruminantium* can be observed in brain smears made after death. Slides are examined for the presence of the characteristic colonies. Experience is required to differentiate from other haemoparasites (*Babesia bovis*), certain blood cells (thrombocytes, granulocytes), normal subcellular structures (mitochondria, mast cell granules) or stain artefacts (stain precipitates). The specificity of the reading can be improved by staining formalin-fixed brain sections using immunoperoxidase techniques. Transmission electron microscopy can be used to demonstrate organisms inside a vacuole-like structure, which is surrounded by a membrane in the endothelial cell's cytoplasm.

Differential clinical diagnosis should be made with anthrax, theileriosis, anaplasmosis, botulism, and, in nervous cases, rabies, tetanus, strychnine poisoning, cerebral theileriosis, cerebral babesiosis and hypomagnesaemia.

Pathology: The lesions present are very variable and not pathognomonic. In the peracute form there are few gross lesions, but in some there is marked lung oedema with tracheal and bronchial fluids. In the acute form the most common macroscopic lesions are hydropericardium, hydrothorax, pulmonary oedema, intestinal congestion, oedema of the mediastinal and bronchial lymph nodes, petechiae on the epicardium and endocardium, congestion of the brain and moderate splenomegaly. The liver is often engorged, with the gallbladder distended. The spleen is occasionally enlarged. There may be congestion of the meningeal blood vessels.

Epidemiology: Distribution of heartwater coincides with that of the *Amblyomma* ticks, which require a warm humid climate and bushy grass. A number of African species of the genus *Amblyomma* (*A. hebraeum*, *A. variegatum*, *A. pomposum*, *A. gemma*, *A. lepidum*, *A. tholloni*, *A. sparsum*, *A. astrion*, *A. cohaerens*, *A. marmoreum*) and American species of *Amblyomma* (*A. maculatum*, *A. cajennense*, *A. dissimile*) are able to transmit infection. Transmission usually appears to be trans-stadial, although transovarian transmission can occur more rarely. The level of infection is often unknown as indigenous domestic and wild animals often show no signs. It is only when susceptible exotic species are introduced that infection becomes apparent. Besides cattle, sheep, goats, Asian buffalo, antelopes and deer are susceptible to infection and disease. Indigenous cattle undergo inapparent infection. Calves under 3 weeks old, even from susceptible stock, are difficult to infect. Heartwater can occur throughout the year, but incidence declines in the dry season due to reduced tick activity. The incubation period is variable, from 7 to 28 days, with fever starting on average after 18 days. Mortality can be up to 60% in exotic breeds, but less than 5% in local cattle.

Treatment: Therapy is most effective when carried out early in disease. Tetracyclines can be used and do not interfere with development of immunity.

Control: Prevention is aimed at controlling the tick vector by dipping cattle at weekly intervals with reliable acaricides. However, ticks of the genus *Amblyomma* are less susceptible than those from other genera. As the tick may transmit infection after 24 hours on the host, better control is obtained by applying acaricide by dipping or spraying every 3 days. Resistance to organophosphates and arsenic has been reported. Care should also be taken not to introduce *Amblyomma* on infected animals or in forage to uninfected cows.

In areas where disease is endemic most cattle are immune. A carrier state develops after infection and remains for several weeks. Non-infected resistance persists a variable time, lasting from a few months to several years. After this time reinfection can occur.

The only method of immunisation is an infection and treatment method using infected blood or homogenised pre-fed infected ticks followed by tetracycline treatment as soon as pyrexia develops.

Notes: Heartwater is one of the main obstacles to the improvement of livestock productivity in sub-Saharan Africa. It was first recognised as a major disease in southern Africa after the introduction of exotic breeds. Its importance depends to a very large extent on the type of livestock present. There are very few reliable figures about its importance in local breeds in endemic areas. However, there is no doubt that in endemic areas indigenous cattle are far more resistant than exotic or crossbred cattle, presumably because of natural selection. In contrast, small ruminants in general, and goats in particular, are not always very resistant.

The name 'heartwater' was used because hydropericardium was regarded as a pathognomonic lesion of the disease. The disease is still also generally known as 'cowdriosis'.

Eperythrozoon wenyonii

Synonym: *Mycoplasma wenyonii*

Predilection site: Blood

Kingdom: Bacteria

Phylum: Firmicutes

Order: Mycoplasmatales

Family: Mycoplasmataceae

Description: Coccoid, ring- or rod-shaped structures on the surface of red cells, blue to purple when stained (see Diagnosis).

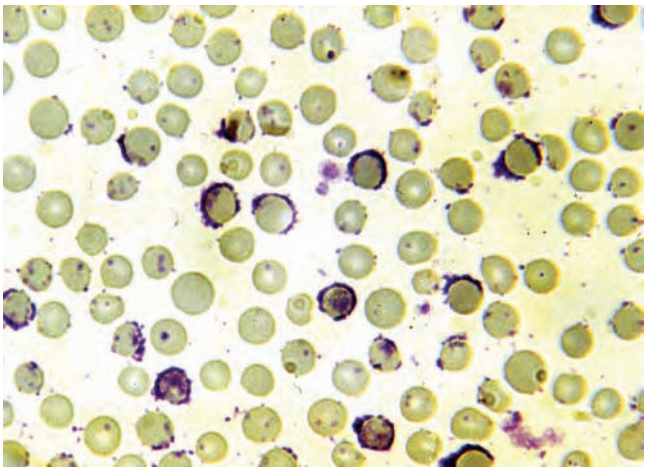

Figure 8.40 *Eperythrozoon wenyonii* on the surface of erythrocytes.

Hosts: Cattle

Geographical distribution: Worldwide

Pathogenesis: Typically present on red cells, it produces mild and clinically inapparent infections in a variety of domestic animals throughout the world.

Clinical signs: *Eperythrozoon wenyonii* is occasionally responsible for fever, anaemia and loss of weight.

Diagnosis: Identification of parasites from staining artefacts requires good blood films and filtered Giemsa stain. They appear as cocci or short rods on the surface of the erythrocytes, often completely surrounding the margin of the red cell (Fig. 8.40). However, the organisms of *Eperythrozoon* are relatively loosely attached to the red cell surface and are often found free in the plasma.

Epidemiology: Vectors are thought to be involved in transmission but precise details are not known.

Treatment: Susceptible to tetracyclines.

Control: Lack of detailed knowledge on the vectors limits any vector control measures.

Notes: The taxonomy of this species is subject to much debate and there is a proposal to reclassify it into the bacterial genus *Mycoplasma* (class Mollicutes) based on 16S rRNA gene sequences and phylogenetic analysis.

Rickettsia conorii

Common names: Boutonneuse fever, Mediterranean spotted fever, Indian tick typhus, East African tick typhus

Predilection site: Blood

Kingdom: Bacteria

Phylum: Proteobacteria

Class: Alphaproteobacteria

Order: Rickettsiales

Family: Rickettsiaceae

Description: Small, pleomorphic, Gram-negative, coccoid, obligatory intracellular organisms infecting endothelial cells of smaller blood vessels.

For more detailed descriptions on pathogenesis, epidemiology, treatment and control see Chapter 12.

Parasites of the nervous system

Taenia multiceps

For more details see Chapter 12.

Thelazia rhodesi

Common name: Cattle eyeworm

Predilection site: Eye, conjunctival sac, lacrimal duct

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Description, gross: Small thin yellowish-white worms, 1.0–2.0 cm long. Males are 8–12 mm and females 12–20 mm.

Description, microscopic: A mouth capsule is present and the cuticle has prominent striations at the anterior end. The male worms have about 14 pairs of pre-cloacal and three pairs of post-cloacal papillae.

Final hosts: Cattle, buffalo, occasionally sheep, goat, camel

Intermediate host: Muscid flies, particularly *Fannia* spp.

Geographical distribution: Worldwide

Pathogenesis: Lesions are caused by the serrated cuticle of the worm and most damage results from movement by the active young adults causing lacrimation, followed by conjunctivitis. In heavy infections the cornea may become cloudy and ulcerated. There is usually complete recovery in about 2 months, although in some cases areas of corneal opacity can persist. Infection may predispose to infectious keratoconjunctivitis ('pink eye') caused by *Moraxella*.

Clinical signs: Lacrimation, conjunctivitis and photophobia. Flies are usually clustered around the eye because of the excessive secretion. In severe cases, the whole cornea can be opaque and, without treatment, progressive keratitis and ulceration of the cornea may occur.

Diagnosis: The presence of a conjunctivitis that is coincident with the season of fly activity is an indication of possible infection. In some cases the *Thelazia* worms may be seen on the surface of the conjunctiva or in the conjunctival sac. Sometimes eggs or larvae can be recovered from lacrimal secretions. It may be necessary to instil a few drops of local anaesthetic to facilitate manipulation of the third eyelid.

Pathology: Invasion of the lacrimal gland and ducts may cause inflammation and necrotic exudation leading to occlusion and reduced tear production. Mechanical irritation of the conjunctiva produces inflammation, while damage to the cornea leads to opacity, keratitis and corneal ulceration.

Epidemiology: *Thelazia* infections occur seasonally and are linked to periods of maximum fly activity. The parasite can survive in the eye for several years, but since it is only the young adult which is pathogenic a reservoir of infection may persist in symptomless carrier cattle. Survival of larvae also occurs in the pupal stages of flies during the winter.

Treatment: Treatment was at one time based on manual removal of the worms under a local anaesthetic, but this is now replaced by administering an effective anthelmintic such as levamisole or an avermectin; the former drug may be applied topically as a 1% aqueous solution.

Control: Prevention is difficult because of the ubiquitous nature of the fly vectors. Fly control measures aimed at protecting the face, such as insecticide-impregnated ear tags, aid in the control of eyeworm infection.

Two other species of eyeworm (*T. gulosa* and *T. skrjabini*) are found in cattle. Details are essentially similar to *T. rhodesii*.

Thelazia gulosa

Synonym: *Thelazia alfortensis*

Common name: Cattle eyeworm

Predilection site: Eye, conjunctival sac and lacrimal duct

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Description: *Thelazia gulosa* are milky-white worms, with thin transverse cuticular striations (less evident in the rear part of the body) and a large, deep, cup-shaped buccal cavity. Males are 4.8–10.9 mm long and have a variable number of pre-cloacal papillae (from 8 to 33 pairs) and three pairs of post-cloacal papillae. There are two asymmetric spicules. The females are 4.8–18.8 mm long with a tapered caudal extremity.

Geographical distribution: Probably worldwide

Thelazia skrjabini

Common name: Cattle eyeworm

Predilection site: Eye, conjunctival sac and lacrimal duct

Phylum: Nematoda

Class: Secernentea

Superfamily: Spiruroidea

Description: Adult worms are whitish in colour, with transverse fine cuticular striations. The buccal cavity is small and shallow. Males are 5–11.5 mm long and curved posteriorly with 16–32 pairs of pre-cloacal and three pairs of post-cloacal papillae. The spicules are unequal in length. The females are 7.5–21 mm long with a truncated caudal extremity.

Geographical distribution: North America, parts of Asia and Europe

Raillietia auris

Predilection site: Ear canal

Class: Arachnida

Subclass: Acari

Order: Mesostigmata

Family: Halarachnidae

Description, gross: The mites are oval and pale yellow, adults measuring approximately 1 mm in length. They have a smooth cuticle with relatively few setae.

Description, microscopic: The holodorsal shield is heavily patterned but with a well-developed tritosternum, longer peritremes and the presence of both a genital and sternal shield in the female. This species has a long dorsal shield (700–800 μm) with 12 pairs of setae.

Final host: Cattle

Clinical signs: Infestations are usually inapparent but the presence of mites in the ear canal may lead to otitis media and otitis interna, with head shaking, head rotation, circling and incoordination.

Hypoderma bovis

For more details see Parasites of the integument.

Toxoplasma gondii

For more details see Parasites of the locomotory system.

Trypanosoma brucei brucei

For more details see Parasites of the circulatory system.

Parasites of the reproductive/urogenital system

Stephanurus dentatus

Common name: Pig kidney worm

Predilection site: Kidney, perirenal fat

Phylum: Nematoda

Class: Secernentea

Superfamily: Strongyloidea

Description: A large stout worm up to 4.5 cm long, with a prominent buccal capsule and transparent cuticle through which the internal organs may be seen. Males are 2–3 cm and females 3–4.5 cm long. The colour is usually pinkish. The size and site are diagnostic. The buccal capsule is cup-shaped with small leaf crowns and six external cuticular thickenings (epaulettes) of which the ventral and dorsal are most prominent, and six cusped teeth at the base. The

male bursa is short and the two spicules of either equal or unequal length.

Hosts: Pig, wild boar, rarely cattle

Pathogenesis: *Stephanurus* may occasionally cause severe liver damage in calves grazing on contaminated ground.

For a more detailed description see Chapter 11.

Tritrichomonas foetus

Synonym: *Trichomonas foetus*

Predilection site: Prepuce, uterus

Phylum: Parabasalia

Class: Trichomonadea

Family: Trichomonadidae

Description: The organism is pear-shaped, approximately 10–25 μm long and 3–15 μm wide, and has a single nucleus and four flagella, each arising from a basal body situated at the anterior rounded end. Three of the flagella are free anteriorly, while the fourth extends backwards to form an undulating membrane along the length of the organism and then continues posteriorly as a free flagellum (Fig. 8.41; see also Fig. 2.10). The axostyle, a hyaline rod with a skeletal function, extends the length of the cell and usually projects posteriorly. The costa is prominent but there is no pelta.

In fresh preparations, the organism is motile and progresses by rolling jerky movements, the flickering flagella and the movements of the undulating membrane being readily seen. Occasionally, rounded immobile forms are observed and these are possibly effete.

Hosts: Cattle

Geographical distribution: Worldwide. However, the prevalence has now decreased dramatically in areas where artificial insemination is widely practised and in Britain, for example, the disease is now probably extinct.

Pathogenesis: In the bull, a preputial discharge associated with small nodules on the preputial and penile membranes may develop shortly after infection. Organisms are present in small numbers in the preputial cavity of bulls, with some concentration in the fornix and around the glans penis. The chronically infected bull shows no gross lesions.

In the cow, the initial lesion is a vaginitis, which can be followed in animals that become pregnant by invasion of the cervix and uterus. Various sequelae can result, including a placentitis leading to early abortion (1–16 weeks), uterine discharge and pyometra. Abortion before the fourth month of pregnancy is the commonest sequela and this is normally followed by recovery. Occasionally the developing fetal membranes are retained leading to a purulent endometritis, a persistent uterine discharge and anoestrus; infrequently the corpus luteum is retained and the cervical seal remains closed, when a massive pyometra develops which visually simulates the appearance of pregnancy. In some cases, despite infection, pregnancy is not terminated by abortion and a normal full-term calf is born.

Clinical signs: In the bull, there are no clinical signs once the infection is established. In the cow, early abortion is a characteristic feature although this is often undetected because of the small size of the fetus and the case may present as one of an irregular oestrous cycle. Other clinical signs are those of purulent endometritis or a closed pyrometra and, in these cases, the cow may become permanently sterile. On a herd basis, cows exhibit irregular oestrous cycles, uterine discharge, pyometra and early abortion. The cow usually recovers and generally becomes immune, at least for that breeding season, after infection or abortion.

Diagnosis: A tentative diagnosis of trichomonosis is based on the clinical history, signs of early abortion, repeated returns to service, or irregular oestrous cycles. Confirmation depends on the demonstration of organisms in placental fluid, stomach contents of the aborted fetus, uterine washings, pyometra discharge or vaginal mucus. Apart from a problem of infertility, which usually follows the purchase of a mature bull, confirmation of diagnosis depends on the demonstration of the organism. Vaginal mucus collected from the anterior end of the vagina by suction into a sterile tube, or preputial washings from the bull, may be examined using a warm-stage microscope for the presence of organisms. The number of organisms varies in different situations. They are numerous in the aborted fetus, in the uterus several days after abortion and, in recently infected cows, they are plentiful in the vaginal mucus 12–20 days after infection. Thereafter the number of organisms varies according to the phase of the oestrous cycle, being highest 3–7 days after ovulation. In the infected bull *T. foetus* organisms are present in highest numbers on the mucosa of the prepuce and penis, apparently not invading the submucosal tissues. It is generally recommended to allow 1 week after the last service before taking a preputial sample. Since the organism is often only present intermittently, the examination may need to be repeated several times. Under phase illumination, the number of flagella observed is an important characteristic as this can help to differentiate *T. foetus* from some bovine flagellates that appear similar. Organisms may be cultured *in vitro*, in Diamond's medium, Clausen's medium or *Trichomonas* medium, which is available commercially. A field culture test that allows for growth of the trichomonads and direct microscopic examination without aspiration of the inoculum has been developed in the USA (InPouch TF).

Alternatively, on a herd basis, samples of vaginal mucus may be examined in the laboratory for the presence of specific agglutinins against laboratory cultures of *T. foetus*.

Figure 8.41 *Tritrichomonas foetus* showing three anterior flagella and trailing posterior flagellum.

Pathology: Infection in females causes cervicitis and endometritis leading to infertility, abortion or pyometra. The inflammatory changes in the endometrium and cervix are relatively mild and non-specific, although there may be a copious mucopurulent discharge. The exudates may be continuous or intermittent in their discharge, and the number and activity of the trichomonads can vary considerably. Abortions may occur at any time but mainly in the first half of pregnancy. There are no specific fetal lesions, but large numbers of protozoa may be found in the fetal fluids and stomach. The placenta may be covered by white or yellowish flocculent exudates in small amounts, and thickening and haemorrhage without necrosis may be evident on the cotyledons. Pyometra, when it develops, may be copious with watery exudates containing floccules which may be brownish and sticky and contain swarms of trichomonads.

Epidemiology: Bulls, once infected, remain so permanently. The organisms inhabit the preputial cavity and transmission to the cow occurs during coitus. From the vagina, the trichomonads reach the uterus via the cervix to produce a low-grade endometritis. Intermittently, organisms are flushed into the vagina, often 2 or 3 days before oestrus. Infection is usually followed by early abortion, the organisms being found in the amniotic and allantoic fluid. Subsequently cows appear to 'self-cure' and, in most cases, appear to develop a sterile immunity.

Treatment: Since the disease is self-limiting in the female only symptomatic treatment and sexual rest for 3 months is normally necessary. In the bull, slaughter is the best policy, although dimetridazole orally or intravenously has been reported to be effective.

Control: Artificial insemination from non-infected donors is the only entirely satisfactory method of control. If a return to natural service is contemplated, recovered cows should be disposed of since some may be carriers.

Notes: Normally one might expect the overall prevalence of trichomonosis to be high, since it is venereally transmitted by bulls, which show no clinical signs. In fact, the advent of supervised schemes of artificial insemination has largely eradicated the disease, and today it is limited to areas where there are many small farms each with their own bulls, or to countries where veterinary supervision is limited.

In a few early studies, three serotypes were recognised based on agglutination: the '*Belfast*' strain, reportedly predominated in Europe, Africa and the USA; the '*Brisbane*' strain in Australia; and the '*Manley*' strain, which has been reported in only a few outbreaks.

A morphologically identical organism (*T. suis*) has been identified in pigs, in which it commonly causes asymptomatic infection of the nasal cavity, stomach and intestine (see Chapter 11). This organism is now considered synonymous with *T. foetus*. The organism has also been reported in cats to be associated with large bowel diarrhoea (see Chapter 12).

Neospora caninum

Predilection site: Blood

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Description: Tachyzoites measure 6 by 2 μm and are usually located in the cytoplasm of cells. Tissue cysts are oval, 107 μm long, have a thick wall (up to 4 μm) and are found only in neural tissue.

Intermediate hosts: Cattle, sheep, goat, deer, horse, dog, fox, chicken, wild birds

Final hosts: Dog, coyote, wolf, dingo

Geographical distribution: Worldwide

Pathogenesis: *Neospora caninum* is a major cause of abortion in both dairy and beef cattle. Cows of any age can abort from 3 months of gestation to full term, although most abortions occur at 5–6 months. Fetuses can be born alive or may die *in utero* and be mummified or reabsorbed. Calves that are infected may be born underweight, weak or with neurological symptoms such as ataxia, decreased reflexes and exophthalmia. Infection is thought to reduce milk production in adult dairy cows through its effects on fertility.

Clinical signs: Abortion, mummification, weak calves with ataxia, exophthalmia

Diagnosis: Diagnosis is based on histological examination of freshly aborted fetuses. The lesions in the heart and CNS are significantly characteristic for diagnosis but can be confirmed by immunocytochemistry. An ELISA is commercially available and can be used to test serum samples for *Neospora*-specific antibodies and several PCR-based tests have been reported. Bulk milk sampling can also be used but is generally only useful in herds where more than 10–20% of cows are infected.

Pathology: Tachyzoites and tissue cysts are found intracellularly in the CNS and retina of affected cattle. Although infection can be found in many organs, the commonest site is the brain. Microscopic lesions of non-suppurative encephalitis and myocarditis may be seen in the brain, spinal cord and heart of aborted fetuses. Hepatitis can also be found in epidemic abortions.

Epidemiology: The dog and other canids are the final host, and can also act as intermediate hosts in prenatal infections. In cattle, infection can be both vertically transmitted from dam to calf *in utero* and lactogenically and naturally by ingestion of food and water contaminated with dog faeces containing *Neospora caninum* oocysts. *Neospora caninum* is one of the most efficiently transplacentally transmitted parasites and in certain herds it has been found that virtually all calves born alive are born infected but without symptoms of infection. Transmission from infected bulls is thought not to occur. The presence of birds on pasture has been correlated with higher infection rates in cattle and birds may be an important link in the transmission of *N. caninum* to other animals.

In some countries, there appears to be an increase in abortion rates associated with mild wet seasons. Infections with other disease agents, such as bovine viral diarrhoea, leptospirosis and *Salmonella*, appear to increase the risk or recrudescence of latent infection and are likely to be associated with a higher risk of abortion in infected cows. It is possible for cattle that have previously aborted due to *Neospora* infection to have a repeat abortion, such that infected cows are more likely to abort than non-infected cows. Offspring born alive are likely to be infected themselves and go on to have a higher risk of abortion.

Treatment: There is no effective treatment in cattle.

Control: Control of *Neospora*-induced abortion in cattle depends on protecting food and water sources from possible contamination

with the faeces of any animal and the disposal of aborted fetuses and placentas by incineration. The lack of complete knowledge of both the life cycle and the range of definitive hosts has limited effective control measures but there is a strong argument for the culling of seropositive animals from a herd. Seropositive animals have been shown to suffer a higher risk of abortion than seronegative animals in the herd. Dogs should not be allowed to eat aborted fetuses or fetal membranes, and their faeces should be prevented from contaminating bovine feedstuffs.

Where *Neospora* has not previously been isolated in a herd, there are several measures which can be taken to reduce the risk of the disease entering the herd.

- Quarantine and testing of all replacements before entry to the herd to ensure freedom from infection.
- Preventing transmission by keeping dogs away from foodstuffs, and ensuring that dogs have no access to either placentas or aborted fetuses.
- Reducing the risk of water-borne transmission by using a mains water supply and avoiding cattle drinking from stagnant water such as ponds.
- Maintaining good rodent control as some studies have implicated rodents in the spread of disease.

In herds where *Neospora* is present, further methods can be used to reduce the risk of animals aborting.

- Testing and culling: *N. caninum*-infected cows should be considered a reservoir of infection with the potential to transmit the infection to other cows. This can occur either by giving birth to live infected offspring or by environmental contamination. Although this method of control is effective, it is not always economically realistic. It can be applied as follows:
  - test and cull either seropositive or seropositive aborting cows;
  - test and inseminate seropositive cows with beef semen only; or
  - test and exclude the progeny of seropositive cows from breeding.

If testing of cows is carried out and cattle are culled on the basis of these results, it is important to ensure that steps are also taken to prevent infection from the environment.

A commercial vaccine (Bovilis Neoguard) was developed to reduce abortion in pregnant cattle resulting from infection with *N. caninum* and was available in the USA, New Zealand and some other countries. This vaccine comprised inactivated whole tachyzoites and inoculation aimed to reduce the transmission of the parasite to the developing fetus. However, the vaccine has been withdrawn from the market by the manufacturer.

Trypanosoma brucei brucei

For more details see Parasites of the circulatory system.

Parasites of the locomotory system

Taenia saginata

Synonyms: *Cysticercus bovis, Taeniarhynchus saginata*

Common names: Beef tapeworm, 'beef measles'

Predilection site: Small intestine (definitive host); muscle, liver, kidney (intermediate host)

Phylum: Platyhelminthes

Class: Cestoda

Family: Taeniidae

Description, gross: The adult tapeworm is found only in humans and ranges from 5 to 15 m in length.

Description, microscopic: The scolex, exceptional among the species of *Taenia*, has neither rostellum nor hooks.

In cattle, the mature cysticercus, *C. bovis*, is greyish-white, oval, about 0.5–1.0 by 0.5 cm long, and filled with fluid in which the scolex is usually clearly visible. As in the adult tapeworm, it has neither rostellum nor hooks.

A subspecies, *Taenia saginata asiatica*, has a rostellum and posterior protruberances on segments and 11–32 uterine buds. The metacestodes are small, about 2 mm, and have a rostellum and two rows of primitive hooks, those of the outer row being numerous and tiny.

Final host: Human

Intermediate host: Cattle, although other ruminants can serve as intermediate hosts

Geographical distribution: Worldwide. Particularly important in Africa and South America.

Pathogenesis: Although *C. bovis* may occur anywhere in the striated muscles, the predilection sites, at least from the viewpoint of routine meat inspection, are the heart, the tongue and the masseter and intercostal muscles (Fig. 8.42). Under natural conditions the presence of cysticerci in the muscles of cattle is not associated with clinical signs although, experimentally, calves given massive infections of *T. saginata* eggs have developed severe myocarditis and heart failure associated with developing cysticerci in the heart.

Clinical signs: In humans, the adult tapeworm may produce diarrhoea and hunger pains, but the infection is usually asymptomatic and is mainly objectionable on aesthetic grounds.

Diagnosis: Individual countries have different regulations regarding the inspection of carcasses, but invariably the masseter muscle, tongue and heart are incised and examined and the intercostal muscles and diaphragm inspected; the triceps muscle is also incised in many countries. The inspection is inevitably a compromise between

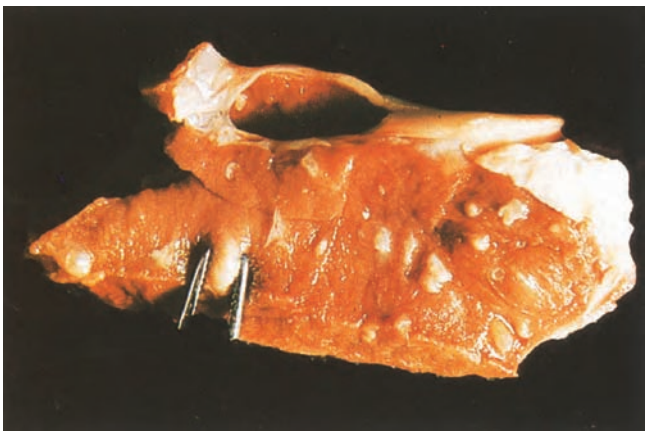

Figure 8.42 *Cysticercus bovis* in skeletal muscle.

detection of cysticerci and preservation of the economic value of the carcass.

Immunoserology has some usefulness for screening infected herds. In humans, the presence of tapeworms is recognised by the passage of proglottids and/or eggs in faeces.

Pathology: Cysticerci commence to degenerate 4–6 months after infection and by 9 months a substantial number may be dead. With light infections cysticerci may remain viable for 2 years or more.

Epidemiology: There are two quite distinct epidemiological patterns found in developing countries and developed countries respectively.

- Developing countries. In many parts of Africa, Asia and Latin America cattle are reared on an extensive scale, human sanitation is poorly developed and cooking fuel is expensive. In these circumstances the incidence of human infection with *T. saginata* is high, in certain areas being well over 20%. Because of this, calves are usually infected in early life, often within the first few days after birth, from infected stockmen whose hands are contaminated with *Taenia* eggs. Prenatal infection of calves may also occur but is rare. Of the cysts which develop, a proportion persist for years even though the host has developed an acquired immunity and is completely resistant to further infection. Based on routine carcass inspection, the infection rate is often around 30–60%, although the real prevalence is considerably higher.
- Developed countries. In areas such as Europe, North America, Australia and New Zealand, the standards of sanitation are high and meat is carefully inspected and generally thoroughly cooked before consumption. In such countries, the prevalence of cysticercosis is low, being less than 1% of carcasses inspected. Occasionally, however, a cysticercosis 'storm', where a high proportion of cattle are infected, has been reported on particular farms. In Britain and in Australia, this has been associated with the use of human sewage on pasture as a fertiliser in the form of sludge, i.e. sedimented or bacterial-digested faeces. Since *T. saginata* eggs may survive for more than 200 days in sludge, the occurrence of these 'storms' is perhaps not surprising. Other causes of a sudden high incidence of infection on particular farms are due to a tapeworm infection in a stockman occurring either as a random event or, as has been reported from feedlots in some of the southern states of the USA, as a result of the use of migrant labour from a country with a high prevalence of infection. As distinct from these 'storms', the cause of the low but persistent prevalence of infection in cattle is obscure, but is thought to be due to the access of cattle to water contaminated with sewage effluents, to the carriage and dispersal of *T. saginata* eggs by birds which frequent sewage works or feed on effluent discharged into rivers or the sea, and to occasional fouling of pasture by itinerant infected individuals. In contrast to the epidemiology in developing countries, cattle of any age are susceptible to infection since they generally possess no acquired immunity. There is also evidence that when cattle are first infected as adults the longevity of the cysticerci is limited, most being dead within 9 months.

Treatment: As yet there is no licensed drug available that will effectively destroy all the cysticerci in the muscle, although praziquantel has shown efficacy in experimental situations.

Control: In developed countries the control of bovine cysticercosis depends on a high standard of human sanitation, on the general practice of cooking meat thoroughly (the thermal death point of cysticerci is 57°C) and on compulsory meat inspection. Regulations usually require that infected carcasses are frozen at –10°C for at least 10 days, which is sufficient to kill the cysticerci although the process reduces the economic value of the meat. Where relatively heavy infections of more than 25 cysticerci are detected, it is usual to destroy the carcass. In agricultural practice the use of human sludge as a fertiliser should be confined to cultivated fields or to those on which cattle will not be grazed for at least 2 years. In developing countries the same measures are necessary, but are not always economically feasible, and at present the most useful step would appear to be the education of communities in both sanitary hygiene and the thorough cooking of meat.

Notes: The intermediate stages of this tapeworm, found in the muscles of cattle, frequently present economic problems to the beef industry and are a public health hazard.

Onchocerca dukei

Predilection site: Muscle connective tissue

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Slender whitish worms; males measure 2–6 cm, while females are up to 60 cm long or more.

Description, microscopic: Microfilariae are 250–265 μm long and unsheathed.

Final host: Cattle

Intermediate hosts: Probably blackflies (*Simulium*)

Geographical distribution: Africa

Pathogenesis: *Onchocerca dukei* is of little clinical or economic importance. Losses may occur by condemnation of localised areas at meat inspection caused by nodular damage.

Clinical signs: Infection in cattle is asymptomatic.

Diagnosis: Diagnosis is often made at meat inspection. Nodules are found particularly in the thorax and abdomen and may need to be differentiated from *Cysticercus bovis*. Microfilariae may be identified after soaking skin biopsy specimens in physiological saline for 12 hours and staining with Giemsa.

Epidemiology: The incidence of infection can be very high in endemic areas, though the parasite is rarely detected.

Treatment and control: Not required

Sarcocystiosis

The previously complex nomenclature for the large number of *Sarcocystis* spp. has largely been discarded by many workers in favour of a new system based on their biology. The new names generally incorporate those of the **intermediate** and **final hosts** in that order. Although unacceptable to systematists, this practice has the virtue of simplicity. The three species of *Sarcocystis* reported in cattle are summarised in Table 8.7 and described below. Further details are given in Chapter 2.

Table 8.7 *Sarcocystis* species found in the muscles of cattle.

Species	Synonym	Definitive host	Pathogenicity (cattle)	Pathogenicity (final host)
Sarcocystis bovicanis	*S. cruzi*, *S. fusiformis*	Dog, coyote, wolf	+++	0
Sarcocystis bovifelis	*S. hirsuto*	Cat	0	0
Sarcocystis bovihominis	*S. hominis*	Human, primates	0	+

0, non-pathogenic; +, mildly pathogenic; +++, severe pathogenicity.

Diagnosis: Most cases of *Sarcocystis* infection are only diagnosed at meat inspection when the grossly visible sarcocysts in the muscle are discovered. However, in heavy infections of cattle, diagnosis is based on the clinical signs and on histological demonstration of meronts in the blood vessels of organs such as kidney or heart and the presence of cysts in the muscles at necropsy or biopsy. An indirect haemagglutination test, using bradyzoites as antigen, is also a useful aid to diagnosis; however, the presence of a titre need not imply active lesions of *Sarcocystis*. Also, animals may die prior to a detectable humoral response. In cattle, the degenerative muscle changes closely resemble those of vitamin E/selenium deficiency, although the latter lacks an inflammatory cellular response. Examination of faeces from dogs or cats on the farm for the presence of sporocysts may be helpful in the diagnosis.

Epidemiology: Little is known of the epidemiology, but from the high prevalence of symptomless infections observed in abattoirs, it is clear that where dogs or cats are kept in close association with farm animals or their feed, then transmission is likely. Sheepdogs are known to play an important part in the transmission of *S. bovicanis* and farm cats in the transmission of *S. bovifelis* so care should be exercised that only cooked meat is fed to dogs or cats. Acute outbreaks are probably most likely when livestock, which have been reared without dog or cat contact, are subsequently exposed to large numbers of the sporocysts from dog or cat faeces. The longevity of the sporocysts shed in the faeces is not known.

Treatment and control: There is no effective treatment for infection, in either the final or the intermediate host. Where an outbreak occurs in cattle, it has been suggested that the introduction of amprolium (100 mg/kg *per os*, daily over 30 days) into the diet of the animals has a prophylactic effect.

The only control measures possible are those of simple hygiene. Farm dogs and cats should not be housed in, or allowed access to, fodder stores nor should they be allowed to defecate in pens where livestock are housed. It is also important that they are not fed uncooked meat.

Sarcocystis bovicanis

Synonyms: *Sarcocystis cruzi, Sarcocystis fusiformis*

Predilection site: Muscle

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Description: In cattle, the meronts found in the endothelial cells are quite small, measuring 2–8 μm in diameter. In contrast, the bradyzoite cysts can be very large and visible to the naked eye as whitish streaks running in the direction of the muscle fibres. They have been reported as reaching several centimetres in length, but more commonly they range from 0.5 to 5.0 mm (Fig. 8.43). The cyst wall is thin and smooth and has a small number of flattened protrusions 0.3–0.6 μm long, without fibrils.

Intermediate hosts: Cattle

Final hosts: Dog, fox, wolf, coyote

Geographical distribution: Worldwide

Pathogenesis: Infection in the final host is normally non-pathogenic, although mild diarrhoea has occasionally been reported. The principal pathogenic effect is attributable to the second stage of merogony in the vascular endothelium. Heavy experimental infections of calves with *S. bovicanis* have resulted in mortality 1 month later, with necropsy showing petechial haemorrhages in almost every organ including the heart, together with generalised lymphadenopathy. Experimental infection of adult cows has resulted in abortion.

A naturally occurring chronic disease of cattle, Dalmeny disease, has been recognised in Canada, the USA and Britain. This is characterised by emaciation, submandibular oedema, recumbency and exophthalmia; at postmortem examination, numerous meronts are found in endothelial cells, and developing sarcocysts in areas of degenerative myositis.

Clinical signs: In heavy infections there is anorexia, fever, anaemia, loss of weight, a disinclination to move and sometimes recumbency. In cattle, there is often a marked loss of hair at the end of the tail. These signs may be accompanied by submandibular oedema, exophthalmia and enlargement of lymph nodes. Abortions may occur in breeding stock.

Pathology: Meronts present in endothelial cells of capillaries in various organs lead to endothelial cell destruction. As the organisms enter muscle, a wide range of change may be encountered.

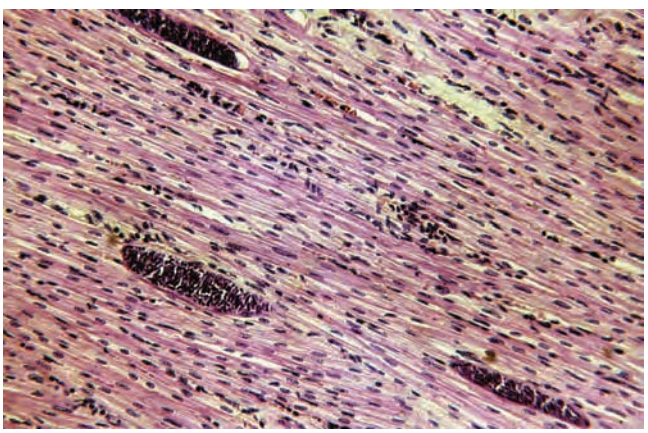

Figure 8.43 Meronts of *Sarcocystis bovicanis* in muscle.

Microscopic inspection of *Sarcocystis*-infected muscle often reveals occasional degenerate parasitic cysts surrounded by variable numbers of inflammatory cells (very few of which are eosinophils) or, at a later stage, macrophages and granulation tissue. Usually there is no muscle fibre degeneration, but there may be thin linear collections of lymphocytes between fibres in the region. The extent of muscle change bears little relationship to the numbers of developing cysts, but generally very low numbers of *Sarcocystis* produce no reaction. As cysts mature, the cyst capsule within the enlarged muscle fibre becomes thicker and more clearly differentiated from the muscle sarcoplasm.

Sarcocystis bovifelis

Synonym: *Sarcocystis hirsuta*

Predilection site: Muscle

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Description: The first-generation meronts measure 37 by 22 μm and contain more than 100 tachyzoites. Second-generation meronts when mature measure 14 by 6.5 μm and contain up to 35 tachyzoites. Sarcocysts are up to 8 mm long with a striated wall, 7 μm thick, and may be visible to the naked eye as whitish streaks running in the direction of the muscle fibres.

Intermediate hosts: Cattle

Final host: Cat

Geographical distribution: Worldwide

Pathogenesis: Infections are generally non-pathogenic; any pathogenic effect is attributable to the second stage of merogony in the vascular endothelium.

Clinical signs: Infections are usually asymptomatic. Heavy infections may occasionally produce anorexia, fever, diarrhoea, anaemia and weight loss.

Pathology: In cattle, the tissue cysts may be visible to the naked eye especially in the oesophagus but are more likely to be detected on histopathology (Fig. 8.44).

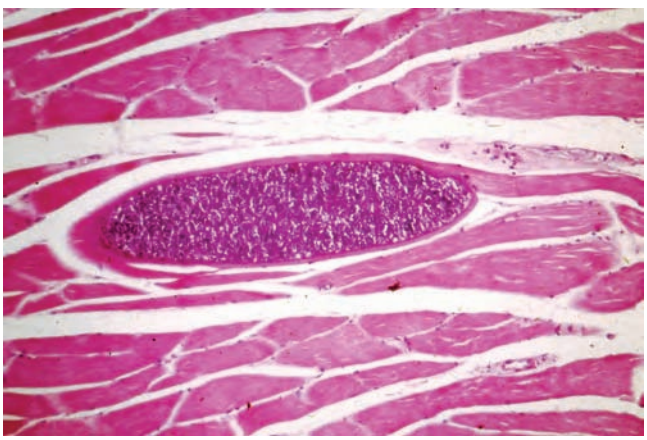

Figure 8.44 *Sarcocystis bovifelis* in oesophageal muscle.

Sarcocystis bovihominis

Synonym: *Sarcocystis hominis*

Predilection site: Muscle

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Description: In the intermediate host, sarcocysts are compartmented with a radially striated wall of about 6 μm in thickness.

Intermediate hosts: Cattle

Final hosts: Human, primates

Geographical distribution: Worldwide

Pathogenesis: The species is slightly if at all pathogenic for calves.

Clinical signs: Infection is usually asymptomatic in calves.

Pathology: Sarcocysts are present in striated muscle. Usually there is no muscle fibre degeneration, but there may be thin linear collections of lymphocytes between fibres in the region.

Toxoplasma gondii

Predilection site: Muscle, lung, liver, reproductive system, CNS

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Intermediate hosts: Any mammal, including human, or birds. Note that the final host, the cat, may also be an intermediate host and harbour extra-intestinal stages.

Final hosts: Cat, other felids

Geographical distribution: Worldwide

Pathogenesis: Most *Toxoplasma* infections in cattle are light and consequently asymptomatic. Infections are usually acquired via the digestive tract, and so organisms are disseminated by the lymphatics and portal system with subsequent invasion of various organs and tissues. Pathogenic effects are always related to the extra-intestinal phase of development. In heavy infections, the multiplying tachyzoites may produce areas of necrosis in vital organs such as the myocardium, lungs, liver and brain, and during this phase the host can become pyrexic and lymphadenopathy occurs. As the disease progresses bradyzoites are formed, with this chronic phase being usually asymptomatic.

Clinical signs: There are only a few reports of clinical toxoplasmosis associated with fever, dyspnoea, nervous signs and abortion in cattle.

Pathology: In heavy infections, the multiplying tachyzoites may produce areas of necrosis in vital organs such as the myocardium, lungs, liver and brain.

Epidemiology: The cat plays a central role in the epidemiology of toxoplasmosis and the disease is virtually absent from areas where cats do not occur. Compared with sheep, toxoplasmosis in cattle is relatively uncommon and rarely causes clinical signs.

Treatment: Not indicated

Control: Control on farms is more difficult, but where possible animal feedstuffs should be covered to exclude access by cats.

For more detailed description see Chapter 9.

Trypanosoma brucei brucei

For more details see Parasites of the circulatory system.

Parasites of the connective tissue

Several species of *Onchocerca* are found in the connective tissue of cattle and are summarised in Table 8.8.

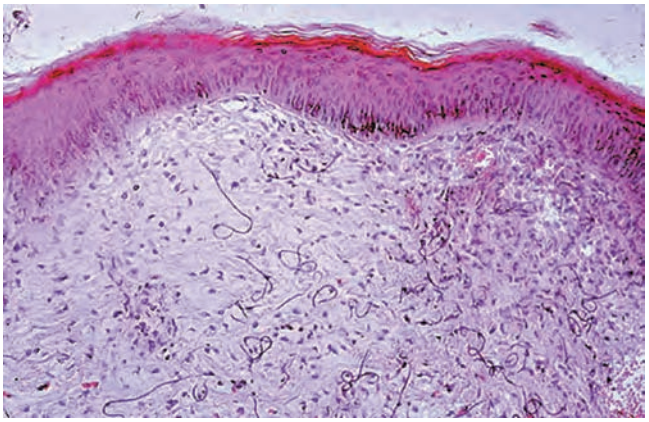

Figure 8.45 Microfilariae of *Onchocerca gutturosa* in subdermal connective tissue of the back.

Onchocerca gutturosa

Synonym: *Onchocerca lienalis*

Common name: Ligamentary onchocercosis

Predilection site: Connective tissue, ligamentum nuchae, gastro-splenic ligament

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Slender whitish worms; males measure 2–6 cm, while females are up to 60 cm long or more.

Description, microscopic: Microfilariae are 250–265 µm long and unsheathed.

Final host: Cattle

Intermediate hosts: Blackflies (*Simulium*)

Geographical distribution: Worldwide. In Australia and North America, the parasite *O. lienalis* (considered to be synonymous) is found in the gastro-splenic ligament.

Pathogenesis: *Onchocerca gutturosa* is of little clinical or economic importance.

Clinical signs: Infection in cattle is asymptomatic.

Diagnosis: Diagnosis is rarely called for and depends on the finding of microfilariae in skin biopsy samples taken from affected areas (Fig. 8.45). The microfilariae are concentrated in the preferred feeding sites of the vectors, which are the back, ears and neck. The piece of skin is placed in warm saline and teased to allow emergence of the microfilariae, and is then incubated for about 8–12 hours. The microfilariae are readily recognised by their sinuous movements in a centrifuged sample of the saline. Another option is to scarify the skin of a predilection site and examine the fluid exudate for microfilariae.

Pathology: Adult worms, which are found in pairs, are most frequently located in the ligamentum nuchae adjacent to the thoracic spines and less frequently in the connective tissue on the scapula, humerus and femur. The worms do not stimulate nodule formation but lie loose in the connective tissue and cause no disease or reaction.

Epidemiology: The incidence of infection can be very high in endemic areas, although the parasite is rarely detected.

Treatment: Not required

Control: With the ubiquity of the insect vectors there is little possibility of efficient control, though the use of microfilaricides will reduce the numbers of infected flies. In any case, with the relatively

Table 8.8 Bovine onchocercosis.

Species	Site	Distribution	Vector	Significance
Onchocerca gutturosa (syn. *O. lienalis*)	Ligamentum nuchae and other parts of the body	Most parts of the world	*Simulium* spp.	No economic significance
	Gastro-splenic ligaments	Many parts of the world	*Simulium* spp.	No economic significance
Onchocerca gibsoni	Subcutaneous and intermuscular nodules	Africa, Asia, Australasia	*Culicoides* spp.	Carcass trimming
Onchocerca ochengi (syn. *O. dermata*)	Scrotum, udder, connective tissue	East and West Africa	Unknown	Blemished hides
Onchocerca armillata	Wall of thoracic aorta	Middle East, Africa, India	*Culicoides*, *Simulium*	No economic significance
Onchocerca dukei	Abdomen, thorax, thighs	West Africa	Unknown *Simulium*?	Confused with *Cysticercus bovis* at meat inspection
Onchocerca cebei (syn. *O. sweetae*)	Abdomen, thorax, thighs	Far East, Australia	*Culicoides* spp.	Blemished hides

innocuous nature of the infection there is unlikely to be any demand for control.

Notes: Some consider this parasite to be synonymous with *O. lienalis*.

Onchocerca gibsoni

Predilection site: Connective tissue

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: These slender worms range from 2 to over 20 cm in length and lie tightly coiled in tissue nodules. Males are 3–5 cm and females 14–20 cm, although there have been reports of worms up to 50 cm in length.

Description, microscopic: The tail of the male is curved and bears lateral alae and six to nine papillae at either side. The spicules are unequal in size. Microfilariae are not sheathed and are 240–280 µm long and are found mainly in the brisket area. The cuticle possesses transverse striations.

Final hosts: Cattle, zebu

Intermediate host: Midges (*Culicoides*)

Geographical distribution: Africa, Asia and Australasia

Pathogenesis: Worms occur in groups ('worm nests') and provoke a fibrous reaction around the coiled worms in muscle tissue (nodules can measure up to 5 cm in diameter). The nodules are often in the brisket and can be responsible for economic loss due to carcass trimming.

Clinical signs: Affected animals are not clinically ill and show no presenting signs other than subcutaneous nodules at the predilections sites.

Diagnosis: In active lesions, the presence of worms is readily established on section of the subcutaneous nodules. Microfilariae may also be found in skin biopsy samples taken from affected areas with subcutaneous lymph spaces. The microfilariae are concentrated in the preferred feeding sites of the vectors, which for *Culicoides* spp. are usually the shaded lower parts of the trunk, and it is usually recommended that samples be taken from the region of the linea alba. The piece of skin is placed in warm saline and teased to allow emergence of the microfilariae, and is then incubated for about 8–12 hours. The microfilariae are readily recognised by their sinuous movements in a centrifuged sample of the saline. Another option is to scarify the skin of a predilection site and examine the fluid exudate for microfilariae.

Pathology: A nodule forms around the worms with the head becoming fixed and surrounded by fibroblasts. Successive portions of the worm are drawn into the nodule, where they eventually lie coiled up and surrounded by a fibrous tissue capsule, which increases in thickness as the lesion grows older. In older nodules, degeneration of the tissues and calcification of the worms frequently takes place. The capsule consists of dense fibrous tissue containing blood vessels, leucocytes and lymph spaces. Microfilariae are common and wander in the lymph spaces. Their presence may lead to thickening of the dermis.

Epidemiology: The incidence of infection can be very high in endemic areas.

Treatment: In the past this has consisted of daily administration of diethylcarbamazine over a period as a microfilaricide, but it now appears that a single dose of ivermectin is highly efficient in this respect, although the dying microfilariae may provoke local tissue reactions. Affected carcasses must be trimmed to remove the nodules.

Control: With the ubiquity of the insect vectors there is little possibility of efficient control, though insect repellents will help reduce insect attack. In any case, with the relatively innocuous nature of the infection there is unlikely to be any demand for control.

Onchocerca ochengi

Synonym: *Onchocerca dermata*

Predilection site: Connective tissue, scrotum and udder

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description: The slender worms range from 2.0 to 6.0 cm in length and lie tightly coiled in tissue nodules. In active lesions the presence of worms is readily established on section of these nodules.

Final host: Cattle

Intermediate hosts: Unknown

Geographical distribution: Parts of East and West Africa

Pathogenesis: *Onchocerca ochengi* in the skin causes some economic loss from blemished hides.

Clinical signs: Affected animals are not clinically ill and show no presenting signs other than subcutaneous nodules at the predilection sites.

Diagnosis: As for *O. gibsoni*

Pathology: Not reported

Epidemiology: The incidence of infection can be very high in endemic areas.

Treatment and control: As for *O. gibsoni*

Parafilaria bovicola

Common names: Summer 'bleeding disease', verminous nodules

Predilection site: Subcutaneous and intermuscular connective tissue

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Slender white worms 3.0–6.0 cm in length. Males are 2–3 cm and females 4–6 cm.

Description, microscopic: Anteriorly, there are numerous papillae and circular ridges in the cuticle. In the female the vulva is situated

anteriorly near the simple mouth opening. Small embryonated eggs, 45 by 30 μm, that have a thin flexible shell are laid on the skin surface where they hatch to release the microfilariae or L_1, which are about 200 μm in length.

Final hosts: Cattle, buffalo

Intermediate hosts: Muscid flies; *Musca autumnalis* in Europe

Geographical distribution: Africa, Asia, southern Europe and Sweden

Pathogenesis: Adult worms in the subcutaneous connective tissue induce small inflammatory lesions and haemorrhagic nodules, usually in the upper body regions. When the gravid female punctures the skin to lay her eggs, there is a haemorrhagic exudate or 'bleeding point' which streaks and mats the surrounding hairs and attracts flies. Individual lesions only bleed for a short time and healing is rapid. There is some evidence that exposure to sunlight is required to initiate bleeding of the nodules.

At the sites of infection, which are predominantly on the shoulders, withers and thoracic areas, there is inflammation and oedema which, at meat inspection, resemble subcutaneous bruising in early lesions and have a gelatinous greenish-yellow appearance with a metallic odour in longer-standing cases. Sometimes the lesions extend into the intermuscular fascia. The affected areas have to be trimmed at marketing and further economic loss is incurred by rejection or downgrading of the hides.

Clinical signs: The signs of parafilariosis, such as 'bleeding points' during the warmer seasons, are pathognomonic. Active bleeding lesions are seen most commonly in warm weather, an apparent adaptation to coincide with the presence of the fly intermediate host. The haemorrhagic exudate often streaks the hair and may lead to focal matting.

Diagnosis: This is normally based on clinical signs, but if laboratory confirmation is required the small embryonated eggs or microfilariae may be found on examination of fresh exudate from bleeding points. The demonstration of eosinophils in smears taken from lesions is also considered a constant diagnostic feature. Serodiagnosis using an ELISA technique has been developed.

Pathology: Nodules formed in the cutaneous and intermuscular connective tissue are 1–2 cm in diameter, enlarge in the summer months, burst open and haemorrhage and heal with scarring.

Epidemiology: In Europe, bovine parafilariosis occurs in spring and summer, disappearing in winter, whereas in tropical areas it is seen mainly after the rainy season. A high prevalence of 36% in cattle has been reported from some endemic areas in South Africa and the disease is now present in Sweden, an area previously free from infection. *Parafilaria* infection may be introduced by the importation of cattle from endemic areas, but its spread will depend on the presence of specific fly vectors. It has been estimated in Sweden that one 'bleeding' cow will act as a source of infection for three other animals.

Treatment: Patent infections in beef and non-lactating dairy cattle may be treated with ivermectin, moxidectin or nitroxynil. The former two drugs are given parenterally as a single dose, whereas two doses of nitroxynil are required at an interval of 3 days. None of these drugs is licensed for use in lactating cattle, when the less effective levamisole may be tried. These drugs produce a marked reduction in bleeding points and, due to resolution of the muscle lesions, a significant reduction in meat condemnation if slaughter is delayed for 70 days after treatment.

Control: This is difficult because of the long prepatent period during which drugs are thought not to be effective. In Sweden, dairy cattle and particularly heifers at pasture are the main source of infection for *M. autumnalis*, which is an outdoor fly, active in spring and summer. However, infections in young beef cattle are the chief cause of economic loss through carcass damage.

Since neither ivermectin nor nitroxynil is effective against immature worms, treatment is only useful for patent infections recognisable by the clinical signs. However, because of restrictions on the use of ivermectin and nitroxynil in lactating cows, these are rarely treated and instead are kept indoors during the period of fly activity.

In endemic areas, young beef cattle may be treated with an anthelmintic some time before slaughter as described above. In Sweden the use of insecticide-impregnated ear tags has been recommended for vector control.

Notes: The adults of this genus of primitive filarioids live under the skin where they produce inflammatory lesions or nodules and, during egg laying, haemorrhagic exudates or 'bleeding points' on the skin surface.

Setaria labiato-papillosa

Synonyms: *Setaria cervi, Setaria altaica, Setaria digitata*

Common name: Bovine abdominal filariosis

Predilection site: Peritoneum, pleural cavity

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: Long slender whitish worms, up to 12.0 cm in length, and in which the posterior end is spirally coiled. The site and gross appearance are sufficient for generic identification (Fig. 8.46). Males are 40–60 mm and females 60–120 mm in length.

Description, microscopic: The tail of the female ends in a marked button, which is divided into a number of papillae. Microfilariae are sheathed and measure 240–260 μm.

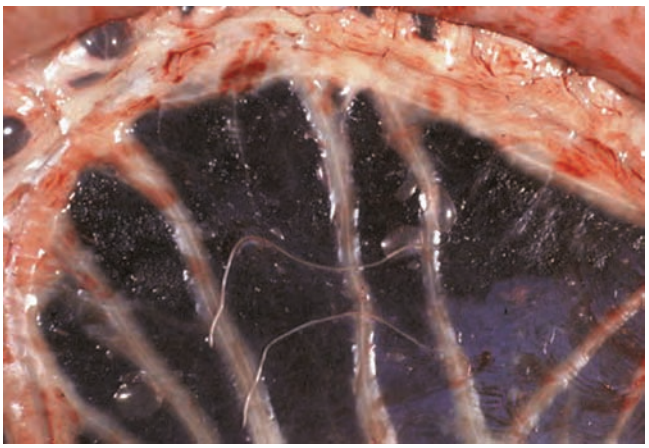

Figure 8.46 Worms of *Setaria* spp. in the mesentery.

Final hosts: Cattle, buffalo, bison, yak, and various deer and antelope, rarely sheep

Intermediate hosts: Mosquitoes (*Aedes, Culex*)

Geographical distribution: Worldwide

Pathogenesis: The worms in their normal site are usually harmless, occasionally inducing a mild fibrinous peritonitis and are only discovered at necropsy. *Setaria labiato-papillosa* may have an erratic migration in sheep and goats and enter the spinal canal causing cerebrospinal setariosis ('lumbar paralysis'), which is irreversible and often fatal; the condition has only been reported in the Middle and Far East.

Clinical signs: There are no clinical signs when the worms are in their normal site, but when nervous tissue is involved there is locomotor disturbance, usually of the hindlimbs, and if the parasites are high in the spinal canal there may be paraplegia.

Diagnosis: Infection with the adult worms is only accidentally discovered in the living animal by the finding of microfilariae in routine blood smears. In cases of cerebrospinal nematodosis, confirmatory diagnosis is only possible by microscopic examination of the spinal cord, since the parasites exist only as larval forms in their aberrant site.

Pathology: A mild fibrinous peritonitis may be found on postmortem. Migrating larvae affecting the CNS may cause areas of damage seen as brown foci or streaks grossly. The lesions show microcavitation and variable haemorrhage. There is loss of myelin and fragmentation of axons locally with eosinophils, neutrophils and macrophages present along with mild meningitis and vascular cuffing.

Epidemiology: Since the worms are usually innocuous, their epidemiology has received little study. The prevalence is higher in warmer countries, where there is longer seasonal activity of the mosquito vectors.

Treatment: There is no treatment for setarial paralysis.

Control: This would depend on control of the mosquito vectors, which is unlikely to be applied specifically for this parasite.

Notes: *Setaria labiato-papillosa* has often been referred to as *S. cervi,* although the latter species is considered a parasite of axis deer (*Cervus axis*). The parasite is also considered to be identical to *S. digitata*, although some consider the latter to be a valid and distinct species.

Setaria digitatus

Common name: Kumri

Predilection site: Peritoneum, pleural cavity

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: As for *S. labiato-papillosa*. The male is 40–50 mm and the female 60–80 mm in length.

Description, microscopic: The tail of the female ends in a simple button.

Final hosts: Cattle, buffalo

Intermediate hosts: Mosquitoes (*Armigeres*, *Aedes*, *Anopheles*, *Culex*)

Geographical distribution: Asia

Pathogenesis: The parasites inhabit the thoracic and peritoneal cavities causing little harm. Immature forms have been reported in the CNS of sheep, goats and horses causing epizootic cerebrospinal nematodosis. Affected animals suffer acute focal encephalo-myelomalacia, which causes acute or subacute tetraplegia or paraplegia of the hindlimbs.

Pathology: In aberrant hosts, migrating larvae affecting the CNS may cause areas of damage seen as brown foci or streaks grossly. Acute malacia occurs along the track of the worm such that the lesions show microcavitation and variable haemorrhage. There is loss of myelin and fragmentation of axons locally with eosinophils, neutrophils and macrophages present along with a mild meningitis and vascular cuffing.

Details on the life cycle, epidemiology, treatment and control are as for *S. labiato-papillosa*.

Parasites of the integument

Stephanofilaria stilesi

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: These are small nematodes, males measuring 2.6–3.7 mm and females 3.7–6.9 mm in length.

Description, microscopic: There are four to five cephalic spines and 18–19 peribuccal spines. The male spicules are unequal and the female worms have no anus. The thin-shelled eggs are 58–72 by 42–55 μm in size. Microfilariae are 45–60 μm in length and are characterised by a peribuccal elevation with a single spine and a short and rounded tail.

Final host: Cattle

Intermediate hosts: Horn fly (*Haematobia irritans*, *H. titillans*)

Geographical distribution: USA, Japan, Commonwealth of Independent States (CIS)

Pathogenesis: Lesions begin to appear within 2 weeks of infection. In this species, the lesions are usually localised to the preferred biting areas of the vectors on the lower abdomen, commonly along the mid-ventral line between the brisket and navel, but also on the udder, scrotum, flanks and ears. The flies feed predominantly along the mid-ventral line of the host and their bites create lesions that permit microfilariae to invade the skin. These lesions are attractive to both species of horn flies as well as non-biting muscids. Adult nematodes occur in the dermis and microfilariae in the dermal papillae of lesions but not in adjacent healthy tissue.

Clinical signs: In endemic regions, granulomatous and ulcerative lesions may be seen on the skin, particularly in the mid-ventral line between the brisket and navel (Fig. 8.47). The dermatitis can be exudative and haemorrhagic.

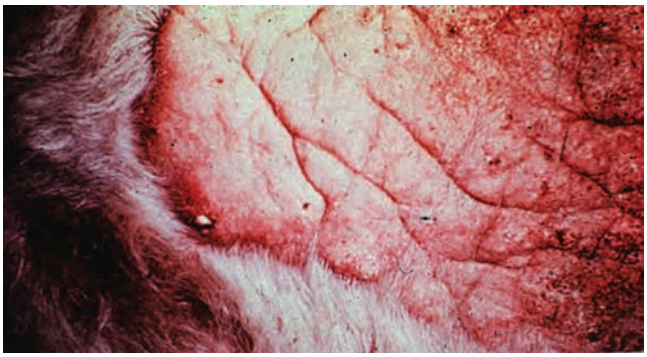

Figure 8.47 Granulomatous skin on the lower abdomen associated with *Stephanofilaria stilesi*.

Diagnosis: Though adult worms and microfilariae are present in the lesions, they are often scarce and many scrapings prove negative. Diagnosis is therefore usually presumptive in endemic areas, and is based on the appearance and site of the lesions. Deep skin scrapings macerated in saline will release microfilariae and adult worms. Biopsy sections readily reveal microfilariae and adults.

Pathology: The skin is at first nodular, but later there is papular eruption with an exudate of blood and pus. In the centre of the lesion there may be sloughing of the skin, but at the margin there is often hyperkeratosis and alopecia. The condition is essentially an exudative, often haemorrhagic, dermatitis that attracts the fly vectors. Sometimes the lesions are exacerbated by secondary bacterial infection.

Epidemiology: In endemic areas the incidence of infection may be as high as 90% and the occurrence is to a great extent influenced by the type of herbage. Succulent grazing produces soft moist faeces, which are more suitable breeding sites for the flies than the hard crumbly faeces deposited on sparse dry grazing. Hence, irrigation of pasture may result in an increase in stephanofilariosis. Although the lesions subside in cooler weather, the damage to the hide is permanent and may result in considerable economic loss. Milk yield may be severely diminished from the pain of the lesions and the irritation of cattle by the flies.

Treatment: Organophosphate compounds, such as trichlorphon, applied topically as an ointment have proved effective. Levamisole at 9–12 mg/kg by injection followed by daily application of zinc oxide ointment has also been reported as effective. Avermectins have reported activity against larval stages but have no appreciable effect against adult stages.

Control: Control of horn flies is feasible by the proper handling of manure and the use of insecticides. Macrocyclic lactones applied topically give reported protection against horn flies for periods of up to 5 weeks.

Stephanofilaria dedoesi

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: These are small nematodes, males measuring 2.3–3.2 mm and females 6.1–8.5 mm in length.

Description, microscopic: The oral aperture is surrounded by a protruding cuticular rim with a denticulate edge. The anterior extremity has a circular thickening, which bears a number of small cuticular spines. The male spicules are unequal and the female worms have no anus.

Geographical distribution: Indonesia

Clinical signs: With *S. dedoesi*, lesions occur mainly on the head, legs and teats of cattle. The dermatitis can be exudative and haemorrhagic.

Other filarial species have been reported in cattle and buffalo in India and parts of Asia. The identification of individual species is beyond the scope of this book and interested readers will need to consult a relevant taxonomic specialist.

Stephanofilaria assamensis

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Geographical distribution: India in *Bos indicus*

Pathogenesis: Infection causes a marked dermatitis ('humpsore'), particularly in the hump, legs and neck.

Stephanofilaria okinawaensis

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Description, gross: The parasites are small, rounded, whitish and slender-bodied. Females are 7.0–8.5 mm and males 2.7–3.5 mm in length

Stephanofilaria zaheeri

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Geographical distribution: India

Final hosts: Cattle, buffalo

Clinical signs: With *S. zaheeri*, lesions occur mainly on the head, legs and teats of cattle and buffalo.

Stephanofilaria kaeli

Predilection site: Skin

Phylum: Nematoda

Class: Secernentea

Superfamily: Filarioidea

Geographical distribution: India

Parafilaria bovicola

For more details see Parasites of connective tissue.

Dracunculus medinensis

Common names: Guinea worm or Medina worm

Predilection site: Subcutaneous connective tissue

Phylum: Nematoda

Class: Secernentea

Family: Dracunculidae

Description, gross: Males measure about 2–3 cm and females up to around 100 cm in length.

Description, microscopic: The female worm has no vulva.

Final hosts: Human and occasionally cattle, horse, dog, cat and other mammal

Intermediate hosts: Copepod crustaceans (*Cyclops* spp.)

Geographical distribution: Africa, the Middle East and parts of Asia

Pathogenesis: Following initial infection there are virtually no signs of disease until the gravid adult female emerges in the subcutaneous tissues of the extremities. Pathogenesis is associated with the cutaneous ulcer formation.

Clinical signs: The migration of the worm to the suface of the skin may induce pruritis and urticaria and a blister on an extremity.

Diagnosis: Symptoms of dracunculosis are pathognomonic.

Pathology: Secondary bacterial infection of the ulcer lesion or degeneration of worms can cause marked abscessation.

Epidemiology: A major global eradication programme has reduced the incidence and importance of *D. medinensis*.

Treatment: The worm may be gradually removed through the lesion by winding it round a small stick at a rate of about 2 cm each day or it may be surgically excised. Treatment with thiabendazole or niridazole, administered over several days, might be effective. Ivermectin or albendazole may be useful but efficacy data are lacking.

Control: This is best achieved through the provision of clean drinking water or water that has been adequately sieved to remove any copepods.

Besnoitia besnoiti

Synonym: *Sarcocystis besnoiti*

Predilection site: Skin, conjunctiva

Phylum: Apicomplexa

Class: Conoidasida

Family: Sarcocystiidae

Description: The pseudocysts are non-septate and about 100–600 μm in diameter, with a thick wall containing thousands of merozoites but no metrocytes.

Intermediate hosts: Cattle, goat, wild ruminants (wildebeest, impala, kudu)

Final hosts: Cat, wild cats (lion, cheetah, leopard)

Geographical distribution: Worldwide, although important in tropical and subtropical countries, especially Africa.

Pathogenesis: Following infection in cattle there is a systemic phase accompanied by lymphadenopathy and oedematous swellings in dependent parts of the body. Subsequently bradyzoites develop in fibroblasts in the dermis, subcutaneous tissues and fascia and in the nasal and laryngeal mucosa. The developing cysts in the skin result in a severe condition characterised by painful subcutaneous swellings and thickenings of the skin, loss of hair and necrosis. Apart from the clinical manifestations, which in severe cases can result in death, there can be considerable economic losses due to condemnation of hides at slaughter.

Clinical signs: Affected animals show skin thickening, swelling, hair loss and skin necrosis. Photophobia, excessive lacrimation and hyperaemia of the sclera are present, and the cornea is studded with whitish elevated specks (pseudocysts).

Diagnosis: Besnoitiosis can be diagnosed by biopsy examination of skin. The spherical encapsulated cysts are pathognomonic. The best method is examination of the scleral conjunctiva where the pseudocysts can be seen macroscopically.

Pathology: This genus differs from other members of the Sarcocystiidae in that the cysts containing bradyzoites are found mainly in fibroblasts in or under the skin. The host cell enlarges and becomes multinucleate as the *Besnoitia* cyst grows within a parasitophorous vacuole, eventually reaching up to 0.6 mm in diameter (see Fig. 2.26).

Epidemiology: Although infection of cattle is thought to be mainly by ingestion of sporulated oocysts from cat faeces, there is a suggestion that mechanical spread by biting flies feeding on skin lesions of cattle may be another route of transmission.

Treatment: There is no known treatment.

Control: Limiting contact of domestic cattle with cats can help reduce the incidence of infection. In countries where the disease is endemic in wildlife populations, control is difficult or impossible to achieve and may be limited to the elimination of infected animals.

Hypoderma spp.

Class: Insecta

Family: Oestridae

(a) (b)

Figure 8.48 (a) *Hypoderma bovis*; (b) *Hypoderma lineatum*.

Description, adults: The adults are large and the abdomen is covered with yellow–orange hairs giving them a bee-like appearance (Fig. 8.48). The adults have no functioning mouthparts.

Description, larvae: The mature larvae are thick and somewhat barrel-shaped, tapering anteriorly. When mature they are 25–30 mm long, and most segments bear short spines. The colour is dirty white when newly emerged from the host, but rapidly turns to dark-brown; the pupa is almost black. The third-stage larvae of the two species of *Hypoderma* that commonly parasitise cattle (*H. bovis* and *H. lineatum*) may be distinguished from other species of *Hypoderma* by examination of the posterior spiracular plate, which is completely surrounded by small spines (Table 8.9). The two *Hypoderma* species in cattle may be distinguished from each other by the fact that in *H. bovis* the posterior spiracular plate surrounding the button has a narrow funnel-like channel, whereas in *H. lineatum* it has a broad channel (see Fig. 3.50).

Hosts: Cattle; the larvae occur erratically in other animals including equines, sheep and, very rarely, humans.

Table 8.9 Summary of differences between the *Hypoderma* species which parasitise cattle.

Feature	Hypoderma bovis	Hypoderma lineatum
Adult length	15 mm	13 mm
Eggs laid	Singly	In batches
Larval morphology	Posterior spiracular plate surrounding the button has a narrow funnel-like channel	Posterior spiracular plate surrounding the button has a broad channel
Migration path	Along nerves	Between the fascial planes of muscles and along connective tissue
Overwintering site	Epidural fat of the spinal cord	Sub mucosa of the oesophagus

Geographical distribution: Northern hemisphere. However, *Hypoderma* is absent from extreme northern latitudes, including Scandinavia, and it has occasionally been found sparsely south of the equator in Argentina, Chile, Peru and southern Africa following accidental introduction in imported cattle.

Pathogenesis: By far the most important feature of this genus is the economic loss caused by downgrading and condemnation of hides perforated by larvae. The L_3 under the skin damage the adjacent flesh and this necessitates trimming from the carcass the greenish gelatinous tissue called 'butcher's jelly', also seen in the infested oesophageal submucosal tissues. In addition the adult flies themselves are responsible for some loss. When they approach animals to lay their eggs, their characteristic buzzing noise, which appears to be instantly recognisable, causes the animals to panic or 'gad', sometimes injuring themselves on posts, barbed wire and other obstacles. Dairy cows show reduced milk yield and beef animals have reduced weight gain as a result of interrupted feeding. This species will pursue animals for some distance making repeated attacks.

Clinical signs: Except for poor growth and decreased milk yield in bad cases, the host animals show no appreciable signs until the larvae appear along the back. The presence of L_3 causes characteristic fluid-filled swellings ('warbles') in the dermis of the back, which can be seen and felt (Fig. 8.49).

Diagnosis: The presence of the larvae under the skin of the back allows diagnosis of warble flies. The eggs may also be found on the hairs of the animals in the summer. Immunodiagnostic tests may be used to detect animals infected with migrating larvae and hence those needing treatment.

Pathology: Warble larvae induce a pronounced tissue inflammation. The cellular reaction is predominantly eosinophilic and lymphocytic. The presence of the larvae also induces the production of

Figure 8.49 'Warble' larvae of *Hypoderma* spp. on the back of a bovine animal.

a thickened connective tissue-lined cavity surrounding the larva, filled with inflammatory cells, particularly eosinophils. If larvae die in the spinal canal, the release of a highly toxic proteolysin may cause paraplegia. Larval death in other regions may, in very rare cases, lead to anaphylaxis in sensitised animals.

Treatment: *Hypoderma* is highly susceptible to systemically active organophosphate insecticides and to the macrocyclic lactones abamectin, ivermectin, doramectin, eprinomectin and moxidectin. The organophosphate preparations are applied as pour-ons to the backs of cattle and are absorbed systemically; macrocyclic lactones can be given by subcutaneous injection or pour-on.

Control: In control schemes in Europe, a single annual treatment is usually recommended, preferably in September, October or November. This is before the larvae of *H. bovis* have reached the spinal canal, so that there is no risk of spinal damage from disintegration of dead larvae. Treatment in the spring when the larvae have left their resting sites and arrived under the skin of the back, although effective in control, is less desirable since the breathing L_3 has then perforated the hide. However, in some countries such as the UK, such treatment is mandatory if warbles are present on the backs of cattle.

Successful eradication schemes supported by legislation, such as restriction of cattle movement on infected farms and compulsory treatment in the autumn, have been undertaken on islands such as the UK and Eire. For example, in the UK the prevalence of infected cattle was reduced from around 40% in the 1970s to virtually zero in the 1990s. However, evidence of infection is still encountered occasionally in animals imported into the UK. Other areas that have practised successful eradication such as Denmark and the Netherlands are clearly at greater risk of reintroduction.

Epidemiology: The flies occur in the summer, particularly from mid June to early September. They are most active on warm days, when they lay their eggs on cattle. The flies are limited in dispersal ability and can travel for more than 5 km.

Hypoderma bovis

Common names: Warble fly, northern cattle grub

Predilection site: Subcutaneous tissues, spinal canal

Description: Adult female *H. bovis* are about 15 mm in length and bee-like in appearance; the abdomen is covered with yellow–orange hairs with a broad band of black hairs around the middle (Fig. 8.48a). The hairs on the head and the anterior part of the thorax are greenish-yellow.

Hypoderma lineatum

Common names: Warble fly, common cattle grub, heel fly

Predilection site: Subcutaneous tissues, oesophagus

Description: Adult female *H. lineatum* are about 13 mm in length and bee-like in appearance; the abdomen is covered with yellow–orange hairs with a broad band of black hairs around the middle. The hairs on the head and the anterior part of the thorax are yellowish-white (Fig. 8.48b).

Pathogenesis: The panic reaction provoked by the approach of adult warble flies is less pronounced with *H. lineatum* than with *H. bovis*, since it reaches the animals by a series of hops along the ground and remains on the lower limb for a time while it lays its row of eggs, so that the animal may be unaware of its presence. Consequently, in parts of the USA this species is appropriately termed the 'heel fly'. If larvae of *H. lineatum* die in the oesophageal wall, they may cause bloat through oesophageal stricture and faulty regurgitation. Larval death in other regions may, in very rare cases, lead to anaphylaxis in sensitised animals.

ECTOPARASITES

FLIES

The larval stages, 'maggots', of a number of species of fly (Diptera) are found in skin wounds on cattle and are listed in the host–parasite checklist at the end of this chapter. More detailed descriptions of these parasites can be found in Chapters 3 and 17.

LICE

Heavy louse infestation is known as pediculosis. Blood-sucking lice have been implicated in the transmission of disease such as those that transmit rickettsial anaplasmosis; however, lice are predominantly of importance because of the direct damage they cause. This effect is usually a function of their density. A small number of lice may be very common and present no problem. However, louse populations can increase dramatically reaching high densities. Transfer of lice from animal to animal or from herd to herd is usually by direct physical contact. Because lice do not survive for long off their host, the potential for animals to pick up infestations from dirty housing is limited, although it cannot be ignored. Occasionally, lice may also be transferred between animals by attachment to flies (phoresy).

Description: Lice have a segmented body divided into a head, thorax and abdomen. They have three pairs of jointed legs and a pair of short antennae. All lice are dorsoventrally flattened and wingless. The sensory organs are poorly developed; the eyes are vestigial or absent.

Geographical distribution: Worldwide, primarily in cooler areas

Pathogenesis: Light infestations are usually only discovered accidentally and should not be considered of any pathogenic importance, lice being almost normal inhabitants of the dermis and coat of many cattle, especially in winter. Moderate infestations are associated only with a mild chronic dermatitis, and are well tolerated. In heavier infestations there is pruritus, with rubbing and licking, but if sucking lice are present in large numbers there may be anaemia and weakness.

Clinical signs: Light infestations are usually only discovered accidentally. In these infections the lice and eggs are easily found by parting the hair, especially along the back, the lice being next to the skin and the eggs scattered like coarse powder throughout the hair. It is important to remember that a heavy louse infestation may itself be merely a symptom of some other underlying condition such as malnutrition or chronic disease, since debilitated animals do not groom themselves and leave the lice undisturbed. In such animals the shedding of the winter coat may be delayed for many weeks, retaining large numbers of lice.

Diagnosis: The lice may be seen on the skin. Removal and examination under a light microscope will allow species identification. The eggs are also visible and appear as white specks attached to the hairs.

Epidemiology: In warm countries there is no marked seasonality of bovine pediculosis, but in cold and temperate regions the heaviest infestations are in late winter and early spring, when the coat is at its thickest, giving a sheltered, bulky and humid habitat for optimal multiplication. The most rapid annual increase in louse populations is seen when cattle are winter-housed, and lice can build up in numbers very quickly. In late spring, there is usually an abrupt fall in the numbers of lice, most of the parasites and eggs being shed with the winter coat. Numbers generally remain low throughout the summer, partly because the thinness of the coat provides a restricted habitat, but partly also because high skin surface temperatures and direct sunlight limit multiplication and may even be lethal.

Treatment: The organophosphate insecticides (e.g. chlorfenvinphos, coumaphos, chlorpyrifos, crotoxyphos, trichlorphon, phosmet and propetamphos), usually applied as pour-on or spot-on applications, are effective in killing all lice. However, most insecticides registered for use on cattle are not very active against louse eggs. This means that after treatment eggs can still hatch and continue the infestation. A second treatment is therefore recommended 2 weeks later to kill newly emerged lice. Pour-on or spot-on synthetic pyrethroids, such as cypermethrin or permethrin, or pour-on avermectins may also be used, although the latter have only limited activity against chewing lice. Essential oils have been shown to be very effective against chewing lice when groomed into the hair.

Control: The timing and frequency of treatments depends very much on individual circumstances. In many cases treatment in late autumn or early winter will give adequate control of cattle lice. In Europe, louse control is usually undertaken when cattle are housed for the winter. Because a wide variety of chemical classes are effective, louse control is not difficult to achieve. Insecticide resistance is widespread in lice, and its rapid spread may be linked to the facultative parthenogenesis seen in many louse species. Hence, in an attempt to reduce the risk of selection for resistance, rotation of chemical classes is strongly advised. Treatment of all stock on farm and subsequent initial quarantine and treatment of all newly introduced animals will allow a good degree of louse control to be maintained.

Bovicola bovis

Synonym: *Damalinia bovis*

Common names: Red louse, cattle chewing louse

Predilection site: Favours the top of the head, especially the curly hair of the poll and forehead, the neck, shoulders, back and rump, and occasionally the tail switch.

Class: Insecta

Order: Phthiraptera

Suborder: Ischnocera

Family: Trichodectidae

Description: *Bovicola bovis* are a reddish-brown in colour with dark transverse bands on the abdomen. Adults measure up to 2 mm in length and 0.35–0.55 mm in width. The head is relatively large, as wide as the body, and is rounded anteriorly (Fig. 8.50; see also Fig. 3.69). The mouthparts are ventral and are adapted for chewing. The legs are slender and are adapted for moving among the hair. The claws, on each leg, are small.

Hosts: Cattle

Pathogenesis: The mouthparts of *B. bovis* are equipped for biting and chewing, and these lice feed on the outer layers of the hair shafts, dermal scales and blood scabs. If infestations increase, the lice may spread down the sides and may cover the rest of the body. This louse feeds by scraping away scurf and skin debris from the base of the hairs, causing considerable irritation to the host animal. The skin reaction can cause the hair to loosen and the cattle react to the irritation by rubbing or scratching, which will result in patches of hair being pulled or rubbed off. Scratching may produce wounds or bruises and a roughness to the skin. This may lead to secondary skin infections and skin trauma such as spot and fleck grain loss in the hide, reducing its value.

Figure 8.50 Chewing louse, *Bovicola*.

Epidemiology: *Bovicola bovis* is one of the commonest cattle parasites in Europe and it is the only chewing louse found on cattle in the USA. Though it causes less individual damage than sucking lice, it is present in larger numbers and so can be extremely damaging. Infested cattle may show disrupted feeding patterns.

Haematopinus eurysternus

Common name: Short-nosed louse

Predilection site: Skin, poll and at the base of the horns, in the ears, and around the eyes and nostrils and even in mild infestations it is found in the tail switch.

Class: Insecta

Order: Phthiraptera

Suborder: Anoplura

Family: Haematopinidae

Description: *Haematopinus eurysternus* is one of the largest lice of domestic mammals, measuring 3.4–4.8 mm in length. The louse is broad in shape, with a short pointed head (Fig. 8.51; see also Fig. 3.53). The head and thorax are yellow or greyish-brown, and the abdomen blue–grey with a dark stripe on each side. The hard-shelled eggs are opaque and white and are pointed at their base.

Hosts: Cattle

Pathogenesis: In severe infestations, the entire region from the base of the horns, over the face (Fig. 8.52) to the base of the tail can be infested.

Notes: This species is more commonly found infesting mature cattle than young animals. In North America, *Haematopinus eurysternus* is more prevalent in the Great Plains and Rocky Mountain regions.

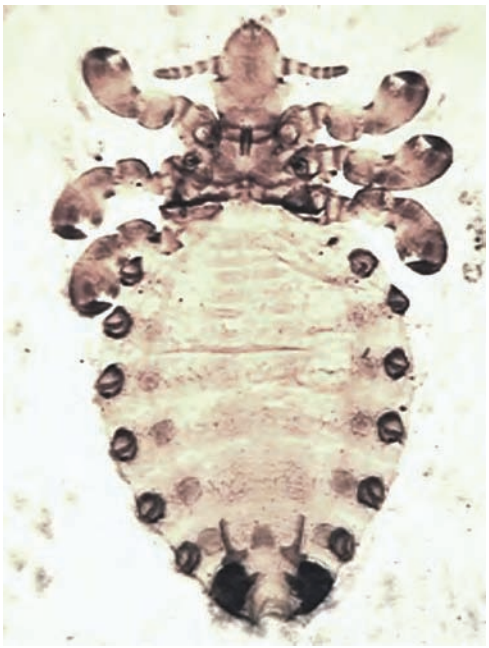

Figure 8.51 Sucking louse, *Haematopinus eurysternus*.

Figure 8.52 Severe bovine pediculosis due to *Haematopinus eurysternus*.

Haematopinus quadripertusus

Common name: Tail louse

Predilection site: Tail and perineum

Class: Insecta

Order: Phthiraptera

Suborder: Anoplura

Family: Haematopinidae

Description: *Haematopinus quadripertusus* is a large eyeless louse about 4–5 mm in length. It has a dark well-developed thoracic sternal plate. Behind the antennae are prominent angular processes, known as ocular points or temporal angles. The legs are of similar sizes, each terminating in a single large claw that opposes the tibial spur. Distinct sclerotised paratergal plates are visible on abdominal segments 2 or 3 to 8.

Hosts: Cattle, commonly zebu cattle (*Bos indicus*)

Pathogenesis: *Haematopinus quadripertusus* feeds on host blood using its piercing mouthparts. In severe infestations, the entire region from the base of the horns to the base of the tail can be infested.

Epidemiology: This species is most commonly found among the long tail hairs at the base of the tail. Unlike other cattle lice, *Haematopinus quadripertusus* is most abundant during the summer and in warmer climates. The lice are transmitted through direct contact between hosts.

Haematopinus tuberculatus

Common name: Buffalo louse

Class: Insecta

Order: Phthiraptera

Suborder: Anoplura

Family: Haematopinidae

Description: A large louse measuring about 5.5 mm in length, with prominent ocular points but without eyes.

Hosts: Cattle, buffalo

Pathogenesis: Populations build up during the winter when the animal's coat is longer and thicker but it is not generally considered of any great clinical importance.

Notes: Known originally to infest buffalo but now found to infest cattle in Africa.

Linognathus vituli

Common name: Long-nosed cattle louse

Predilection site: Skin, preferring the head, neck and dewlap

Class: Insecta

Order: Phthiraptera

Suborder: Anoplura

Family: Linognathidae

Description: Bluish-black medium-sized louse with an elongated pointed head and body, approximately 2.5 mm in length (see Fig. 3.55). There are no eyes or ocular points. Forelegs are small. Midlegs and hindlegs are larger, with a large claw and tibial spur. There are two rows of setae on each segment. The thoracic sternal plate is weakly developed or absent. The eggs may be dark in colour, and are less easy to see on hair. These lice are gregarious in habit, forming dense isolated clusters. While feeding they extend their bodies in an upright position.

Hosts: Cattle

Pathogenesis: This species is capable of transmitting bovine anaplasmosis, dermatomycosis (ringworm) and theileriosis.

Epidemiology: Heaviest infestation occurs in late winter and early spring, commonly on the head and around the eyes (Fig. 8.53).

Solenopotes capillatus

Common name: Little blue cattle louse

Predilection site: Skin of neck, head, shoulders, dewlap, back and tail

Figure 8.53 Heavy louse infestation of *Linognathus vituli*.

Class: Insecta

Order: Phthiraptera

Suborder: Anoplura

Family: Linognathidae

Description: Small bluish lice which tend to occur in clusters on the neck, head, shoulders, dewlap, back and tail. These lice may be distinguished from the genus *Linognathus* by the presence of abdominal spiracles set on slightly sclerotised tubercles, which project slightly from each abdominal segment (see Fig. 3.56). At 1.2–1.5 mm in length, *S. capillatus* is the smallest of the anopluran lice found on cattle. Eyes and ocular points are absent, and the louse has a short rostrum. There are no paratergal plates on the abdomen. The second and third pairs of legs are larger than the first pair and end in stout claws. In contrast to species of *Linognathus*, the thoracic sternal plate is distinct. The eggs of this louse species are small, short and dark blue.

Hosts: Cattle

MITES

The ectoparasitic mites of cattle feed on blood, lymph, skin debris or sebaceous secretions, which they ingest by puncturing the skin, scavenge from the skin surface or imbibe from epidermal lesions. Most ectoparasitic mites spend their entire lives in intimate contact with their host, so that transmission from host to host is primarily by physical contact. Infestation by mites is called acariosis and can result in severe dermatitis, known as mange, which may cause significant welfare problems and economic losses.

Demodex bovis

Predilection site: Hair follicles and sebaceous glands

Class: Arachnida

Subclass: Acari

Order: Prostigmata (Trombidiformes)

Family: Demodicidae

Description: Species of *Demodex* have an elongate tapering body, up to 0.1–0.4 mm in length, with four pairs of stumpy legs ending in small blunt claws in the adult (see Fig. 3.100). Setae are absent from the legs and body. The legs are located at the front of the body, and as such the striated opisthosoma forms at least half the body length.

Hosts: Cattle

Geographical distribution: Worldwide

Pathogenesis: The most important effect of bovine demodicosis is the formation of many pea-sized nodules, each containing caseous material and several thousand mites, which cause hide damage and economic loss. Although these nodules can be easily seen in smooth-coated animals, they are often undetected in rough-coated cattle until the hide has been dressed. Problems caused by demodicosis in cattle are primarily a result of the damage caused to the hides. In some rare cases demodicosis may become generalised and fatal.

Clinical signs: Pea-sized nodules containing caseous material and mites, particularly on the withers, lateral neck, back and flanks. Concurrent pyoderma may occur, leading to furunculosis with ulceration and crust formation.

Diagnosis: For confirmatory diagnosis, deep scrapings are necessary to reach the mites deep in the follicles and glands. This is best achieved by taking a fold of skin, applying a drop of liquid paraffin, and scraping until capillary blood appears.

Pathology: In cattle cutaneous nodules consist of follicular cysts lined with squamous epithelium and filled with waxy keratin squames and mites. Eruption of the cysts on to the skin may form a thick crust; rupture within the dermis may form an abcess or granulomatous reaction.

Epidemiology: Probably because of its location deep in the dermis, it is almost impossible to transmit *Demodex* between animals unless there is prolonged contact. Such contact usually only occurs during suckling, and as such it is thought that most infections are acquired in the early weeks of life. The muzzle, neck, withers and back are all common sites of infestation.

Treatment: In many cases demodicosis spontaneously resolves and treatment is unnecessary. The organophosphate trichlorphon, used on three occasions 2 days apart, and systemic macrocyclic lactones may be effective.

Control: Control is rarely applied since there is little incentive for farmers to treat their animals, as the cost of damage is borne by the hide merchant.

Notes: Species of the genus *Demodex* are highly specialised mites that live in the hair follicles and sebaceous glands of a wide range of wild and domestic animals, including humans. They are believed to form a group of closely related sibling species that are highly specific to particular hosts: *Demodex phylloides* (pig), *Demodex canis* (dog), *Demodex bovis* (cattle), *Demodex equi* (horse), *Demodex musculi* (mouse), *Demodex ratti* (rat), *Demodex caviae* (guinea-pig), *Demodex cati* (cat) and *Demodex folliculorum* and *Demodex brevis* on humans.

In some parts of Australia 95% of hides are damaged, and surveys in the USA have shown one-quarter of the hides to be affected. In Britain 17% of hides have been found to have *Demodex* nodules.

Psorobia bovis

Synonym: *Psorergates bos*

Common name: Cattle itch mite

Predilection site: Skin, all over the body

Class: Arachnida

Subclass: Acari

Order: Prostigmata (Trombidiformes)

Family: Psorergatidae

Description: *Psorobia bovis* is a small mite, roughly circular in form and less than 0.2 mm in diameter. The legs are arranged more or less equidistantly around the body circumference, giving the mite a crude star shape (see Fig. 3.103). Larvae of *P. bovis* have short stubby legs. The legs become progressively longer during the nymphal stages until, in the adult, the legs are well developed and the mites become mobile. Adults are about 190 μm long and 160 μm wide. The tarsal claws are simple and the empodium is pad-like. The femur of each leg bears a large, inwardly directed, curved spine. In the adult female, two pairs of long whip-like setae are present posteriorly; in the male there is only a single pair.

Hosts: Cattle

Geographical distribution: Australia, New Zealand, southern Africa, North and South America. It has not been reported in Europe.

Pathogenesis: Little or no pathogenic effect

Clinical signs: There are few clinical signs associated with infestations of this mite. Mites may occur on apparently normal skin without causing itching of the host animal.

Diagnosis: To obtain mites it is necessary, having clipped away a patch of hair, to apply a drop of mineral oil and scrape the skin down to the blood capillary level. The mites themselves are easily identified.

Pathology: Rarely the mite may cause alopecia and desquamation, but in the majority of cases there appears to be no recognisable lesion associated with the infection.

Epidemiology: This mite is not normally considered to be of clinical significance.

Treatment: *Psorobia* is relatively unsusceptible to most acaricides, although the formamidine amitraz has recently been shown to be of considerable value. Otherwise, the older arsenic–sulphur preparations may be used. Macrocyclic lactones may be effective.

Control: Regular checks of livestock and treatments will keep infection rate under control.

Psoroptes ovis

Synonyms: *Psoroptes communis* var. *ovis, Psoroptes cuniculi, Psoroptes cervinus, Psoroptes bovis, Psoroptes equi*

Predilection site: Skin, particularly the legs, feet, base of tail and upper rear surface of the udder

Class: Arachnida

Subclass: Acari

Order: Astigmata (Sarcoptiformes)

Family: Psoroptidae

Description: *Psoroptes* mites are up to 0.75 mm in length and oval in shape (see Fig. 3.92). All the legs project beyond the body margin. Its most important recognition features are the pointed mouthparts and the three-jointed pretarsi (pedicels) bearing funnel-shaped suckers (pulvilli) (see Fig. 3.87). Adult females have jointed pretarsi and pulvilli on the first, second and fourth pairs of legs and long whip-like setae on the third pair. In contrast, the smaller adult males, which are recognisable by their copulatory suckers and paired posterior lobes, have pulvilli on the first three pairs of legs and setae on the fourth pair. The legs of adult females are approximately the same length, whereas in males the fourth pair is extremely short.

Figure 8.54 Bovine psoroptic mange.

Pathogenesis: In cattle these mites cause intense pruritus, papules, crusts, excoriation and lichenification (Fig. 8.54). Lesions may cover almost the entire body; secondary bacterial infections are common in severe cases. Death in untreated calves, weight loss, decreased milk production and increased susceptibility to other diseases can occur.

Treatment: In cattle, dipping and topical application of non-systemic acaricides, such as the organophosphates (diazinon, coumaphos or phosmet), amitraz or a lime–sulphur dip, may be effective. Dippings should be repeated at 2-week intervals. The topical application of flumethrin is also used in some parts of the world. Most treatments are not licensed for use in dairy cattle. Injectable formulations of avermectins (ivermectin and doramectin) and milbemycins (moxidectin) may be effective, although following treatment with ivermectin the isolation of treated animals for 2–3 weeks after treatment is required to pevent reinfestation. Eprinomectin is available as a pour-on formulation, and is the only macrocyclic lactone that may be used in dairy cattle.

Following diagnosis, the treatment of all animals on infected premises and subsequent treatment of all incoming stock is recommended.

For a more detailed description see Chapter 9.

Psoroptes natalensis

Predilection site: Skin, particularly the legs, feet, base of tail and upper rear surface of the udder

Class: Arachnida

Subclass: Acari

Order: Astigmata (Sarcoptiformes)

Family: Psoroptidae

Description: Very similar to *P. ovis* but it is believed that *P. natalensis* can be distinguished morphologically by the length and spatulate shape of the fourth outer opisthosomal seta of the male. However, the precise species status of *P. natalensis* has yet to be confirmed.

Hosts: Primarily buffalo but it has been reported on cattle.

For treatment and pathogenesis see *P. ovis*.

Chorioptes bovis

Synonyms: *Chorioptes ovis, Chorioptes equi, Chorioptes caprae, Chorioptes cuniculi*

Predilection site: Skin, particularly the legs, feet, base of tail and upper rear surface of the udder

Class: Arachnida

Subclass: Acari

Order: Astigmata (Sarcoptiformes)

Family: Psoroptidae

Description: Adult female *Chorioptes bovis* are about 300 μm in length (see Fig. 3.93), considerably smaller than *Psoroptes ovis*. *Chorioptes* do not have jointed pretarsi; their pretarsi are shorter than in *Psoroptes* and the sucker-like pulvillus is more cup-shaped, as opposed to trumpet-shaped in *Psoroptes* (see Fig. 3.87). In the adult female, tarsi I, II and IV have short-stalked pretarsi and tarsi III have a pair of long, terminal, whip-like setae. The first and second pairs of legs are stronger than the others and the fourth pair has long slender tarsi. In the male, all legs possess short-stalked pretarsi and pulvilli. However, the fourth pair is extremely short, not extending beyond the body margin. Male *C. bovis* have two broad flat setae and three normal setae on well-developed posterior lobes. The mouthparts are distinctly rounder, and the abdominal tubercles of the male are noticeably more truncate than those of *Psoroptes* (see Fig. 3.87).

Hosts: Cattle, sheep, horse, goat, rabbit

Geographical distribution: Worldwide

Pathogenesis: In cattle, chorioptic mange occurs most often in housed animals, particularly dairy animals, affecting mainly the neck, tail head, udder and legs. Usually only a few animals in a group are clinically affected. The mites are found more commonly on the hindleg than on the foreleg. It is a mild condition, and lesions tend to remain localised, with slow spread. Its importance is economic, the pruritus caused by the mites resulting in rubbing and scratching, with damage to the hide. High infestations have been associated with decreased milk production. The treatment is the same as for sarcoptic mange in cattle.

Clinical signs: Hosts can be asymptomatic with low densities of mites present and thus act as carriers that transfer the mite to other animals. Host reactions are normally only induced when the numbers increase to thousands of mites per host. Scabs or scales develop on the skin of the lower parts of the body. There is some exudation and crust formation on the legs and lower body, but in most cases this does not spread over a wide area. Infected animals may stamp and scratch infected areas. The majority of the mites are likely to be found on the lower leg, particularly the pastern and foot. However, in some animals the infestation may become acute and generalised, and closely resemble infestation with *Psoroptes*.

Diagnosis: Skin scrapings from the suspect lesions should be taken for microscopic examination.

Pathology: The pathology is highly variable depending on the intensity and duration of infection; subclinical infections are common. Clinically affected animals may have pustular, crusted, scaly and lichenified lesions and alopecia.

Epidemiology: Mite populations are highest in the winter and may regress over summer. It is the most common type of mange in cattle in the USA.

Treatment: The dips used for psoroptic mange in cattle are also effective against *Chorioptes*. They should be repeated at 2-week intervals. Ivermectin, doramectin, eprinomectin and moxidectin applied topically as a pour-on are also effective against chorioptic mange.

Control: Regular checks of livestock and quarantining of infected animals will help to control the frequency and extent of infestations.

Notes: The names *Chorioptes ovis*, *Chorioptes equi*, *Chorioptes caprae* and *Chorioptes cuniculi* used to describe the chorioptic mites found on sheep, horses, goats and rabbits, respectively, are now all thought to be synonyms of *Chorioptes bovis*.

Figure 8.55 Characteristic lesions of bovine sarcoptic mange.

Sarcoptes scabiei

Common name: Scabies

Predilection site: Skin

Class: Arachnida

Subclass: Acari

Order: Astigmata (Sarcoptiformes)

Family: Sarcoptidae

Description: Adult mites have a round, ventrally flattened, dorsally convex body (see Fig. 3.89). Adult females are 0.3–0.6 mm long and 0.25–0.4 mm wide, while males are smaller, typically up to 0.3 mm long and 0.1–0.2 mm wide. The posterior two pairs of limbs do not extend beyond the body margin. In both sexes, the pretarsi of the first two pairs of legs bear empodial claws and a sucker-like pulvillus, borne on a long stalk-like pretarsus. The sucker-like pulvilli help the mite grip the substrate as it moves. The third and fourth pairs of legs in the female and the third pair of legs in the male end in long setae and lack stalked pulvilli. The mouthparts have a rounded appearance. These mites have no eyes or stigmata. The dorsal surface of the body of *S. scabiei* is covered with transverse ridges, but also bears a central patch of triangular scales. The dorsal setae are strong and spine-like. The anus is terminal and only slightly dorsal. There are a number of host-adapted varieties of *S. scabiei* that differ subtly in their morphology.

Pathogenesis: Sarcoptic mange is potentially the most severe of the cattle manges, although many cases are mild. Nevertheless, it is being increasingly diagnosed in Britain and in some areas, including Canada and parts of the USA, the disease is notifiable and the entry of cattle carrying *Sarcoptes*, whether clinically affected or not, is not permitted. The mite has partial site preferences, which have given it, in the USA, the common name of 'neck and tail mange', but it may occur on any part of the body. Mild infections merely show scaly skin with little hair loss, but in severe cases the skin becomes thickened, there is marked loss of hair and crusts form on the less well haired parts of the body (Fig. 8.55), such as the escutcheon of cows. There is intense pruritus leading to loss of meat and milk production and to hides being downgraded because of damage by scratching and rubbing.

Treatment and control: Treatment has largely depended on the use of repeated washes or sprays usually organochlorine insecticides such as gamma-hexachlorocyclohexane. However, organochlorine insecticides are not now available in most countries. Systemic macrocyclic lactones may give good results. Alternatively, the application of a pour-on organophosphate such as phosmet, on two occasions at an interval of 14 days, is also effective. Neither macrocyclic lactones nor phosmet are licensed for use in lactating animals whose milk is used for human consumption. The formamidine amitraz is effective against sarcoptic mange in cattle and has withdrawal periods of 24 and 48 hours, respectively, for meat and milk.

For further details see Chapter 11.

A number of non-obligate ectoparasites are found on cattle and are listed in the host–parasite checklist at the end of this chapter. More detailed descriptions of these parasites can be found in Chapters 3 and17.

HOST–PARASITE CHECKLIST

In the following checklists, the codes listed below apply:

Helminths

N, nematode; T, trematode; C, cestode; A, acanthocephalan.

Arthropods

F, fly; L, louse; S, flea; M, mite; Mx, maxillopod; Ti, tick.

Protozoa

Co, coccidia; Bs, blood sporozoa; Am, amoeba; Fl, flagellate; Ci, ciliate.

Miscellaneous 'protozoal organisms'

B, blastocyst; Mi, microsporidian; My, Mycoplasma; P, Pneumocystidomycete; R, Rickettsia.

Cattle parasite checklist.

	Helminths		Arthropods		Protozoa	
Section/host system	**Parasite**	**(Super) family**	**Parasite**	**Family**	**Parasite**	**Family**
Digestive						
Oesophagus	*Gongylonema pulchrum*	Spiruroidea (N)	*Hypoderma bovis* *Hypoderma lineatum*	Oestridae (F) Oestridae (F)		
Rumen/reticulum	*Gongylonema verrucosum* *Gongylonema pulchrum* *Paramphistomum cervi* *Calicophoron daubneyi* *Paramphistomum microbothrium* *Paramphistomum ichikawa* *Paramphistomum streptocoelium* *Cotylophoron cotylophorum* *Calicophoron calicophorum* *Gastrothylax crumenifer* *Fischoederius elongatus* *Fischoederius cobboldi* *Carmyerius spatiosus* *Carmyerius gregarius*	Spiruroidea (N) Spiruroidea (N) Paramphistomatidae (T) Paramphistomatidae (T) Paramphistomatidae (T) Paramphistomatidae (T) Paramphistomatidae (T) Paramphistomatidae (T) Paramphistomatidae (T) Gastrothylacidae (T) Gastrothylacidae (T) Gastrothylacidae (T) Gastrothylacidae (T) Gastrothylacidae (T)			*Monocercomonas ruminantium* *Entamoeba bovis*	Monocercomonadidae (Fl) Entamoebidae (AM)
Abomasum	*Ostertagia ostertagi (lyrata)* *Ostertagia leptospicularis* *Spiculopteragia spiculoptera* *Haemonchus contortus* *Haemonchus similis* *Trichostrongylus axei* *Mecistocirrus digitatus* *Parabronema skrjabini* *Capillaria bilobata*	Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Spiruroidea (N) Trichuroidea (N)			*Cryptosporidium andersoni*	Cryptosporidiidae (Co)
Small intestine	*Trichostrongylus colubriformis* *Trichostrongylus longispicularis* *Cooperia oncophora* *Cooperia punctata* *Cooperia pectinata* *Cooperia surnabada* *Nematodirus helvetianus* *Nematodirus battus* *Nematodirus spathiger* *Bunostomum phlebotomum* *Agriostomum vryburgi* *Strongyloides papillosus* *Toxocara vitulorum* *Capillaria bovis* *Moniezia benedeni* *Moniezia expansa* *Thysaniezia ovilla* *Avitellina centripunctata* *Stilesia globipunctata* *Thysaniezia ovilla* *Thysanosoma actinoides* *Cymbiforma indica*	Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Trichostrongyloidea (N) Ancylostomatoidea (N) Ancylostomatoidea (N) Rhabditoidea (N) Ascaridoidea (N) Trichuroidea (N) Anoplocephalidae (C) Anoplocephalidae (C) Anoplocephalidae (C) Anoplocephalidae (C) Anoplocephalidae (C) Anoplocephalidae (C) Anoplocephalidae (C) Notocotylidae (T)			*Eimeria bovis* *Eimeria zuernii* *Eimeria alabamensis* *Eimeria auberensis* *Eimeria brasiliensis* *Eimeria bukidnonensis* *Eimeria canadensis* *Eimeria cylindrica* *Eimeria ellipsoidalis* *Eimeria pellita* *Eimeria subspherica* *Eimeria wyomingensis* *Cryptosporidium parvum* *Cryptosporidium ryanae* *Cryptosporidium ubiquitum* *Giardia intestinalis*	Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Eimeriidae (Co) Cryptosporidiidae (Co) Cryptosporidiidae (Co) Cryptosporidiidae (Co) Giardiidae (Fl)

(Continued)

Cattle parasite checklist. *Continued*

	Helminths		Arthropods		Protozoa	
Section/host system	**Parasite**	**(Super) family**	**Parasite**	**Family**	**Parasite**	**Family**
Caecum	*Oesophagostomum radiatum*	Strongyloidea (N)			*Eimeria zuernii*	Eimeriidae (Co)
Colon	*Trichuris globulosa*	Trichuroidea (N)			*Eimeria bovis*	Eimeriidae (Co)
	Trichuris discolor	Trichuroidea (N)			*Tetratrichomonas buttreyi*	Trichomonadidae (Fl)
	Homalogaster paloniae	Gastrodiscidae (T)			*Tetratrichomonas pavlovi*	Trichomonadidae (Fl)
					Tritrichomonas enteris	Trichomonadidae (Fl)
					Retortamonas ovis	Retortamonadoridae (Fl)
					Buxtonella sulcata	Pycnotrichidae (Ci)
Respiratory						
Nasal cavities	*Mammomonogamus laryngeus*	Strongyloidea (N)				
Trachea/bronchi	*Mammomonogamus nasicola*	Strongyloidea (N)				
	Schistosoma nasale	Schistosomatidae (T)				
	Dictyocaulus viviparus	Trichostrongyloidea (N)				
Lung	*Echinococcus granulosus*	Taeniidae (C)			*Pneumocystis carinii*	Pneumocystidaceae (P)
Liver						
	Fasciola hepatica	Fasciolidae (T)				
	Fasciola gigantica	Fasciolidae (T)				
	Fascioloides magna	Fasciolidae (T)				
	Dicrocoelium dendriticum	Dicrocoeliidae (T)				
	Dicrocoelium hospes	Dicrocoeliidae (T)				
	Gigantocotyle explanatum	Paramphistomatidae (T)				
	Echinococcus granulosus	Taeniidae (C)				
	Echinococcus ortleppi	Taeniidae (C)				
	Stilesia hepatica	Anoplocephalidae (C)				
	Cysticercus tenuicollis (*Taenia hydatigena*)	Taeniidae (C)				
	Thysanosoma actinioides	Anoplocephalidae (C)				
Pancreas						
	Eurytrema pancreaticum	Dicrocoeliidae (T)				
	Eurytrema coelomaticum	Dicrocoeliidae (T)				
	Thysanosoma actinioides	Anoplocephalidae (C)				
Circulatory						
Blood	*Schistosoma bovis*	Schistosomatidae (T)			*Trypanosoma brucei brucei*	Trypanosomatidae (Fl)
	Schistosoma mattheei	Schistosomatidae (T)			*Trypanosoma brucei evansi*	Trypanosomatidae (Fl)
	Schistosoma leiperi	Schistosomatidae (T)			*Trypanosoma congolense*	Trypanosomatidae (Fl)
	Schistosoma indicum	Schistosomatidae (T)			*Trypanosoma vivax*	Trypanosomatidae (Fl)
	Schistosoma nasalis	Schistosomatidae (T)			*Trypansoma theileri*	Trypanosomatidae (Fl)
	Schistosoma spindale	Schistosomatidae (T)			*Babesia bigemina*	Babesiidae (Bs)
	Schistosoma japonicum	Schistosomatidae (T)			*Babesia bovis*	Babesiidae (Bs)
	Schistosoma turkestanica	Schistosomatidae (T)			*Babesia divergens*	Babesiidae (Bs)
					Babesia major	Babesiidae (Bs)
					Babesia occultans	Babesiidae (Bs)
					Babesia ovata	Babesiidae (Bs)
					Babesia jakimovae	Babesiidae (Bs)
					Theileria parva	Theileriidae (Bs)
					Theileria annulata	Theileriidae (Bs)
					Theileria orientalis complex	Theileriidae (Bs)

					Theileria taurotragi *Theileria velifera* *Anaplasma marginale* *Anaplasma centrale* *Anaplasma phagocytophilum* *Ehrlichia bovis* *Ehrlichia ruminantium* *Rickettsia conorii* *Eperythrozoon wenyonii*	Theileriidae (Bs) Theileriidae (Bs) Anaplasmataceae (R) Anaplasmataceae (R) Anaplasmataceae (R) Anaplasmataceae (R) Anaplasmataceae (R) Rickettsiaceae (R) Mycoplasmataceae (My)
Blood vessels	*Elaeophora poeli* *Onchocerca armillata*	Filarioidea (N) Filarioidea (N)				
Nervous						
CNS	*Coenurus cerebralis* *(metacestode: Taenia multiceps)*	Taeniidae (C)	*Hypoderma bovis*	Oestridae (F)	*Toxoplasma gondii* *Trypanosoma brucei brucei*	Sarcocystiidae (Co) Trypanosomatidae (Fl)
Eye	*Thelazia rhodesii* *Thelazia gulosa* *Thelazia skrjabini*	Spiruroidea (N) Spiruroidea (N) Spiruroidea (N)				
Ear			*Raillietia auris*	Halarachnidae (M)		
Reproductive/ urogenital						
	Stepahanurus dentatus	Strongyloidea (N)			*Tritrichomonas foetus* *Neospora caninum* *Trypanosoma brucei*	Trichomonadidae (Fl) Sarcocystiidae (Co) Trypanosomatidae (Fl)
Locomotory						
Muscle	*Cysticercus bovis* (metacestode: *Taenia saginata)*	Taeniidae (C)			*Sarcocystis bovicanis* *Sarcocystis bovifelis* *Sarcocystis bovihominis* *Toxoplasma gondii*	Sarcocystiidae (Co) Sarcocystiidae (Co) Sarcocystiidae (Co) Sarcocystiidae (Co)
Connective tissue						
	Onchocerca gutturosa *Onchocerca gibsoni* *Onchocerca ochengi* *Onchocerca dukei* *Parafilaria bovicola* *Setaria labiato-papillosa* *Setaria digitatus*	Filarioidea (N) Filarioidea (N) Filarioidea (N) Filarioidea (N) Filarioidea (N) Filarioidea (N) Filarioidea (N)				
Subcutaneous	*Parafilaria bovicola* *Dracunculus medinensis*	Filarioidea (N) Dracunculidae (N)	*Hypoderma bovis* *Hypoderma lineatum* *Dermatobia hominis* *Calliphora albifrontis* *Calliphora nociva* *Calliphora stygia* *Calliphora vicina* *Calliphora vomitoria* *Lucilia sericata* *Lucilia cuprina* *Lucilia illustris* *Protophormia terraenovae* *Phormia regina* *Cordylobia anthropophaga*	Oestridae (F) Oestridae (F) Oestridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F) Calliphoridae (F)		

(Continued)

Cattle parasite checklist. *Continued*

Section/host system	Helminths		Arthropods		Protozoa	
	Parasite	**(Super) family**	**Parasite**	**Family**	**Parasite**	**Family**
			Cochliomyia hominivorax	Calliphoridae (F)		
			Cochliomyia macellaria	Calliphoridae (F)		
			Chrysomya bezziana	Calliphoridae (F)		
			Chrysomya megacephala	Calliphoridae (F)		
			Wohlfahrtia magnifica	Sarcophagidae (F)		
			Sarcophaga haemorrhoidalis	Sarcophagidae (F)		
Integument						
Skin	*Stephanofilaria stilesi*	Filarioidea (N)	*Bovicola bovis*	Trichodectidae (L)	*Besnoitia besnoiti*	Sarcocystiidae (Co)
	Stephanofilaria assamensis	Filarioidea (N)	*Haematopinus eurysternus*	Haematopinidae (L)		
	Stephanofilaria zaherii	Filarioidea (N)	*Haematopinus quadripertusus*	Haematopinidae (L)		
	Stephanofilaria kaeli	Filarioidea (N)	*Haematopinus tuberculatus*	Haematopinidae (L)		
	Stephanofilaria dedoesi	Filarioidea (N)	*Linognathus vituli*	Linognathidae (L)		
	Stephanofilaria okinawaensis	Filarioidea (N)	*Solenopotes capillatus*	Linognathidae (L)		
			Demodex bovis	Demodicidae (M)		
			Psorobia bovis	Psorergatidae (M)		
			Psoroptes ovis	Psoroptidae (M)		
			Psoroptes natalensis	Psoroptidae (M)		
			Chorioptes bovis	Psoroptidae (M)		
			Sarcoptes scabiei	Sarcoptidae (M)		

The following species of flies and ticks are found on cattle. More detailed descriptions can be found in Chapter 17.

Flies of veterinary importance on cattle.

Group	Genus	Species	Family
Blackflies Buffalo gnats	*Simulium*	spp.	Simuliidae (F)
Blowflies and screwworms	*Calliphora*	*albifrontis* *nociva* *stygia* *vicina* *vomitoria*	Calliphoridae (F)
	Chrysomya	*albiceps* *bezziana* *megacephala*	
	Cochliomyia	*hominivorax* *macellaria*	
	Cordylobia	*anthropophaga*	
	Lucilia	*cuprina* *illustris* *sericata*	
	Phormia	*regina*	
	Protophormia	*terraenovae*	
Bot flies	*Gedoelstia*	*haessleri*	Oestridae (F)
	Hypoderma	*bovis* *lineatum*	
	Dermatobia	*hominis*	
Flesh flies	*Sarcophaga*	*fusicausa* *haemorrhoidalis*	Sarcophagidae (F)
	Wohlfahrtia	*magnifica* *meigeni* *vigil*	
Hippoboscids	*Hippobosca*	*equina* *rufipes* *maculata* *camelina*	Hippoboscidae (F)
Midges	*Culicoides*	spp.	Ceratopogonidae (F)
Mosquitoes	*Aedes* *Anopheles* *Culex*	spp. spp. spp.	Culicidae (F)
Muscids	*Haematobia*	*irritans* *exigua*	Muscidae (F)
	Musca	*autumnalis* *domestica*	
	Stomoxys	*calcitrans*	
Sandflies	*Phlebotomus*	spp.	Psychodidae (F)
Tabanids	*Chrysops* *Haematopota* *Tabanus*	spp. spp. spp.	Tabanidae (F)
Tsetse flies	*Glossina*	*fusca* *morsitans* *palpalis*	Glossinidae (F)

Tick species found on cattle.

Genus	Species	Common name	Family
Ornithodoros	*moubata*	Eyeless or hut tampan	Argasidae (Ti)
	savignyi	Eyed or sand tampan	
Otobius	*megnini*	Spinose ear tick	Argasidae (Ti)
Amblyomma	*americanum*	Lone star tick	Ixodidae (Ti)
	cajennense	Cayenne tick	
	gemma		
	hebraeum	South African bont tick	
	maculatum	Gulf coast tick	
	pomposum		
	variegatum	Variegated or tropical bont tick	
Dermacentor	*andersoni*	Rocky Mountain wood tick	Ixodidae (Ti)
	marginatus	Ornate sheep tick	
	nutalli		
	reticulatus	Marsh tick, meadow tick	
	occidentalis	Pacific coast tick	
	silvarium		
	variabilis	American dog tick	
Haemaphysalis	*punctata*		Ixodidae (Ti)
	concinna	Bush tick	
	bispinosa	Bush tick	
	longicornis	Scrub tick, New Zealand cattle tick	
Hyalomma	*anatolicum*	Bont-legged tick	Ixodidae (Ti)
	detritum	Bont-legged tick	
	dromedarii	Camel tick	
	excavatum		
	marginatum	Mediterranean tick	
	truncatum	Bont-legged tick	
Ixodes	*ricinus*	Castor bean or European sheep tick	Ixodidae (Ti)
	holocyclus	Paralysis tick	
	rubicundus	Karoo paralysis tick	
	scapularis	Deer tick, black-legged tick	
Rhipicephalus	*appendiculatus*	Brown ear tick	Ixodidae (Ti)
	bursa		
	capensis	Cape brown tick	
	evertsi	Red or red-legged tick	
	sanguineus	Brown dog or kennel tick	
	simus	Glossy tick	
Rhipicephalus (*Boophilus*)	*annulatus*	Texas cattle fever tick	Ixodidae (Ti)
	decoloratus	Blue tick	
	microplus	Pantropical or southern cattle tick	