

DISEASES OF SMALL RUMINANTS IN SUB-SAHARAN AFRICA A HANDBOOK

LUGHANO KUSILUKA AND DOMINIC KAMBARAGE

Common Diseases of
Sheep and Goats in
Sub-Saharan Africa



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by

**Lughano Kusiluka
and Dominic Kambarage**

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INTRODUCTION

The main constraints hindering the productivity of the livestock sector in most sub-Saharan countries are diseases; poor nutrition; poor breeding policies and poor management. The common diseases which affect goats and sheep in sub-Saharan countries are helminthosis, peste des petits ruminants, contagious ecthyma, goat/sheep pox, pneumonia, anthrax, blackquarter, footrot, caseous lymphadenitis and brucellosis. Other diseases include heartwater, coccidiosis, trypanosomosis, Nairobi sheep disease, Rift Valley fever, blue tongue, mastitis and tuberculosis. Mange mites, fleas, ticks, lice and *Oestrus ovis* are the major ectoparasites infesting small ruminants in the region. Physical injuries and chemical or plant poisoning are reported to occur in occasional incidences. Malnutrition is the major non-infectious cause of unthriftiness in goats.

The motive behind the publication of this handbook has been the scarcity of easily accessible literature on small ruminant health in the sub-Saharan region particularly to the field veterinarians and animal scientists. Most often, scientific literature published in journals is collected in libraries in universities and research institutions to which workers in the extension service have little access. Therefore, this handbook is an attempt to extend the accumulated knowledge on diseases of goats and sheep in sub-Saharan Africa to field workers. It is also suitable for students undertaking veterinary or small ruminant health-related studies in the region. The handbook is written in a straight forward and easily understandable manner. Emphasis is on the most commonly reported diseases of goats and sheep but some of the less common conditions and those with a great potential to occur have also been mentioned. Since the occurrence of diseases in animals is greatly influenced by the management systems used, an introductory chapter on management systems is included. A list of references is provided at the end of each chapter so that the reader can get more details on aspects which have not been covered in much detail in the handbook. Authors are of the opinion that consultation of literature given under the list of references for further details will greatly complement the handbook.

CHAPTER 1 SYSTEMS OF SMALL RUMINANT MANAGEMENT IN SUB-SAHARAN AFRICA

TRADITIONAL SMALL RUMINANT MANAGEMENT SYSTEMS

Small ruminant management systems in sub-Saharan Africa can be grouped into two broad categories, that is, traditional and modern systems. The traditional management systems can be further subdivided into groups namely, the extensive (pastoral), semi-intensive (agropastoral) and intensive (agricultural or village) systems.

The extensive or pastoral system

This system is common in arid and semi-arid areas receiving rainfall below 700 mm per annum and because of the unreliability of rainfall, crop agriculture is restricted to irrigated areas where only drought resistant crops such as millet, sorghum and cassava can be grown. Forage and water sources for the animals in these areas are scarce and subsistence is mainly derived from livestock use, sale or exchange.

Two sub-systems, nomadism and transhumance are found under the extensive system of management. In the nomadic system, people and animals are constantly moving in search of pasture and water or to avoid disease epidemics and tribal conflicts. Subsistence is based wholly on animals. Examples of nomadic tribes are the Maasai of Kenya and Tanzania and the Iraqwis (Mang'ati) of Tanzania. In the transhumant system, livestock production is associated with rain-fed agriculture. Examples of transhumant tribes are the Wagogo and Wasukuma of Tanzania and the Fulani of West Africa.

Under the extensive system, animals graze on communal land and, animal herds owned by different families or individuals compete for grazing and water. Often, herds of cattle, small ruminants and camels graze together. At night, animals are protected from theft and/or predation by confinement in night enclosures which are constructed using thorn bushes or wooden poles depending on the availability of these materials. The only human input into this system is the unpaid family labour used to look for the animals and the main management objective is to avoid risks of diseases, drought and to maintain herd numbers. There is no controlled breeding, supplementary feeding or veterinary care for the animals except where governments are developing veterinary extension services to encourage settlement of pastoral communities. The marketing infrastructure for animals is also underdeveloped.

Because of shortage of water and forage, malnutrition is the major limiting factor for profitable production of small ruminants particularly during the dry season. Heavy mortalities of animals have been recorded during severe drought periods in Somalia, Ethiopia and Sudan. Intermingling of animals in communal grazing land facilitates spread of infectious diseases such as peste des petits ruminants, contagious caprine pleuropneumonia, contagious ecthyma, goat and sheep-pox, footrot, trypanosomosis, helminthosis and ectoparasitic infestations. Unrestricted movement of animal herds in search food grazing, to avoid disease epidemics or tribal conflicts have resulted in spread of diseases beyond country borders. Outbreaks of diseases such

as Nairobi sheep disease, bluetongue and Rift valley fever usually occur when naive animals are moved into endemic areas. The occurrence of diseases under the extensive system is also precipitated by stress factors such as long distance trekking in search for feed and water, intense heat and sunlight, malnutrition and poor housing systems. For example, it has been reported that, in some parts of Nigeria, high incidences of endoparasites in goats are most prevalent in extensively managed small ruminants compared to the intensively managed ones. Also, a higher incidence of coccidiosis is encountered in goats kept on solid rather than on slatted floors. In Cameroon, higher incidence of accidents has been observed among the extensive compared with intensively managed small ruminants. The high mobility of people and their animals in search of scarce resources, particularly under the nomadic system makes it difficult to devise or institute small ruminant improvement programmes.

Since the extensive system of management is characterised by low inputs, poor husbandry systems, poor nutrition, poor veterinary care and poor marketing systems, productivity under this system is extremely low and losses resulting from mortality and poor growth rates are high. Unless responsible government make efforts to improve the delivery of extension services and veterinary intervention, productivity of small ruminants under the extensive system of management will continue to be low.

The semi-intensive or agropastoral system

This system is common in areas where the annual rainfall is more than 1000 mm and therefore suitable for crop cultivation. Thus, subsistence is gained mainly from crops and, small ruminants are kept as an insurance against crop failures and as a source of income. Under this system, between 10 and 50 percent of household revenue is derived from livestock or livestock products and, in years of drought they may be the only means of subsistence.

Livestock are sedentary, although movement of animals for a short-distance is not uncommon. Most commonly, animals are grazed for a restricted period of time, usually late morning or late evening, depending on the availability of labour and feed. Herding is practised to control grazing, protect animals from raiders and predators. Herders also assist diseased animals and collect newly born kids. Family labour, particularly women and children under school age and, rarely hired labour is used to supervise the animals. Small ruminants complement crop production by utilising crop residues and by-products while their manure fertilises lands. Supplementary feeding of the animals with crop residues and household wastes such as cassava and banana peelings or vegetables is sometimes practised and at night, animals are confined in enclosures close to the owner's house. The night enclosures are similar to those observed under the extensive system. Sometimes animals are just tied to pegs around the owner's house. There is minimal control of breeding and veterinary care.

Under the semi-intensive system, since most of the land is utilised for crop production, land available for animals is limited. As a result, herd sizes are smaller than in the extensive system. Studies conducted in Tanzania, Kenya, Swaziland and Ethiopia have indicated that herd sizes under this system range between 1-20 goats or sheep although some households may have more animals. In Tanzania, the agropastoral system is the most dominant system of small ruminant production. The restriction of feeding and sub-standard husbandry practices associated with this system results in lowered productivity of the animals just as in the extensive system. Supplementary

feeding of animals under this system has a great potential for improving productivity and because animals are sedentary, it is possible to institute small ruminant improvement programmes.

The intensive or village system

This system is common in areas with high population density and intense agricultural activity. Land scarcity and labour shortages are the major limiting factors to small ruminant production. Four feeding systems are found within this system; tethering, stall-feeding, intensive use of cultivated forages/pastures and integration with tree crops.

Tethering

Tethering is a widespread systems of small ruminant management by small-holders in sub-Saharan Africa and, has been adopted in order to prevent animals from destroying crops and to enable farmers to carry out other farm activities. Due to scarcity of land and shortage of labour, fewer goats are kept per household. In this system, goats are secured with a rope and tied to a peg, tree or post close to the owner's house, by the roadside or on nearby uncultivated areas. Supplements such as banana leaves, bean/pea leaves and pods, brewer's or maize grain may provided by some farmers. Houses are similar to those under the agropastoral system, but in some areas, animals are kept in the family house for fear of theft. Animals are individually owned and only family labour is involved in animal management under this system. Flock sizes under the tethering system in Tanzania are in the order of 1-10 goats or sheep per household. Because feeding is restricted, animals have no choice of feed and this results in poor body condition and low weight gains. Tethering has been found to be associated with low weight gains and predisposition of animals to heavy helminth burdens in Tanzania, Kenya, Cameroon, Nigeria and in The Gambia. Studies in Kenya have indicated that mineral deficiencies are common in tethered compared to free range goats. In Tanzania, tethering is common in many parts of Tanzania and it has become more popular as more children attend schools. Indigenous breeds of goats and sheep predominate under extensive, semi-intensive and tethering systems.

Stall-feeding

Stall-feeding (zero-grazing) is commonly practised in the densely populated and intensively cultivated areas of Kenya, Tanzania, Nigeria and Ethiopia. Stall-feeding is mainly identified with small-holder dairy goat keepers and research institutions. The system is also increasingly being adopted in urban and peri-urban areas. Flock sizes are determined by the owner's capital base and land availability. Animals are confined in houses and food is cut and carried to them. Household wastes are used as supplements. Fodder crops are sometimes grown for the goats and concentrates are also provided. In West Africa, strip farming, using a high quality leguminous browse such as *Leucaena leucocephala* and *Gliricidia spp* has greatly increased productivity of small ruminants under the cut- and-carry system. Houses are constructed of concrete or wooden walls, usually with slatted or concrete floors and thatched with grass or sheet iron roofs. Due to the high capital input in acquiring dairy animals better animal husbandry practices are implemented and returns from this system are considerably high compared to other systems. Because of the high nutrient demand of dairy goats and, proper veterinary care which cannot be met under the extensive, semi-intensive and tethering system,

production can be very poor if the dairy goats are kept under such systems. In recent studies in Tanzania, it has been shown that, attempts by small-holders to raise dairy goats under the tethering system without supplementation have been disappointing. Poor hygiene in stall-feeding units may be associated with high incidences of endoparasites (helminths and coccidial), ectoparasites (fleas, lice, mange mites) and pneumonia. In Nigeria, some workers have reported that pneumonia is more prevalent in confined animals than in extensively kept small ruminants. Nevertheless, there is a great potential for increasing milk production under this sub-system especially in peri-urban and urban areas.

Intensive use of cultivated forages/pastures

This is a high capital investment system, with carrying capacity of up to 60 goats or sheep per ha depending on the nutritive value of the grass, level of fertiliser and presence or absence of legumes. The system has a high potential for increased productivity, but it is less commonly practised in the Africa.

Integration with tree crops

Integration with tree crops is common in many West African countries and the eastern coastline of East Africa. This an efficient mixed farming system where animals graze below tree crops and are protected from heat stress by the tree canopy shade. In return, they fertilise the land with their excreta, graze waste herbage and control weeds.

MODERN SMALL RUMINANT PRODUCTION

Modern small ruminant production systems require a high capital investment. This system is not widespread in the sub-Saharan region although small ruminant commercial or stud flocks are important components of ranching systems in South Africa, Kenya, Zimbabwe and Swaziland. In other countries, the systems are mostly identified with government and research institutions. Animals are either stall-fed or grazed on improved pastures during the day and are housed at night. Supplementary feeds such as concentrates are often provided. Most commonly small ruminants are integrated with dairy, beef and sometimes large scale crop farming. Because animals are raised for commercial purposes, modern animal husbandry practices are employed and the productivity of animals under this system is generally high. However, considerable economic losses may be encountered under this system if husbandry practices are poor. Disease such as helminthosis, coccidiosis, clostridial enterotoxaemias and pneumonia are frequently encountered under this system causing tremendous losses. Close confinement is very favourable for cross-transmission of diseases.

It follows from the above section that, the prevalence of various disease problems in small ruminants are influenced by management systems. Therefore, any disease control programmes in an area should be formulated with due consideration of the management systems. Most diseases of livestock can be controlled by proper management.

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CHAPTER 2 DISEASES CAUSED BY HELMINTHS

Helminthosis is considered to be a major cause of mortality and sub-optimal productivity in goats and sheep in traditional farming systems in sub-Saharan countries. Helminths cause direct losses due to deaths and indirect losses due to reduced productivity through reduced feed intake and liveweight gains and, decreased quality of skins, wool or mohair. Furthermore, they render animals more susceptible to other infections.

Aetiology

Helminthosis is a widespread infection of small ruminants in the sub-Saharan region. Nematodes, trematodes and cestodes are the three major classes of parasitic helminths of economic and zoonotic importance affecting goats and sheep in this region.

The most common species of nematodes associated with parasitic gastro-enteritis in small ruminants in most sub-Saharan countries are *Haemonchus contortus*, *Oesophagostomum columbianum* and *Trichostrongylus colubriformis*. *Trichostrongylus axei*, *Bunostomum trigonocephalum*, *Cooperia curticei*, *Trichuris ovis*, *Trichuris globulosus*, *Strongyloides papillosus*, *Gaigeria pachyscelis* and *Chabertia ovina* also contribute to the syndrome. In winter rainfall and cool highland areas, *Ostertagia circumcincta* and *Nematodirus filicollis* are also involved in the pathogenesis of parasitic gastro-enteritis in goats and sheep. Lungworms such as *Dictyocaulus filaria*, *Muellerius capillaris* and *Protostrongylus rufescens* cause parasitic bronchitis particularly in young animals.

Fasciola spp, *Paramphistomum* spp and *Schistosoma* spp are the main trematodes infecting goats and sheep. *Fasciola gigantica* is the commonest species associated with fasciolosis in most sub-Saharan countries. *Fasciola hepatica* has also been shown to be a significant cause of fasciolosis in highland areas of Kenya, north-eastern and south-western Tanzania, Ethiopia, Lesotho and the Republic of South Africa. Clinical paramphistomosis in small ruminants is caused by *Paramphistomum microbothrium*. *Schistosoma bovis* is the main cause of clinical schistosomosis in small ruminants although *Schistosoma mattheei* has also been implicated.

Stilesia hepatica and *Moniezia expansa* are the common parasitic cestodes encountered in goats in the sub-Saharan region. *S. hepatica* causes biliary fibrosis and is an important cause of liver condemnations in abattoirs in Kenya and Tanzania. *M. expansa* infection is very common in kids and heavy infection with the parasite causes unthriftiness. The significance of *Stilesia globipunctata* and *Avitellina centripunctata* infections in small ruminants in sub-Saharan Africa has not been well documented. The presence of other cestodes such as *Echinococcus granulosus*, *Taenia ovis* (metacestode, *Cysticercus ovis*), *Taenia multiceps* (metacestode, *Coenurus cerebralis*) and *Taenia hydatigena* (metacestode, *Cysticercus tenuicollis*) in tissues or organs leads to condemnation of the affected tissues/organs. The migration of *Coenurus cerebralis* through the brain can cause meningo-encephalitis while the presence of many hydatid cysts in the lungs may be associated with respiratory problems.

Epidemiology

Gastrointestinal nematodes

The epidemiology of gastrointestinal nematode infections is influenced by climatic factors (particularly rainfall and temperature), management systems used for the animals, host factors and parasite factors.

Climatic factors: Rainfall or moisture is the most important factor which influences the survival, development, dissemination and availability of free living stages of helminths. Moisture facilitates horizontal and vertical migration of nematode larvae on the environment. Some workers have demonstrated that dung beetles may also transport larvae up and down the herbage. Higher worm burdens and outbreaks of parasitic gastro-enteritis in goats and sheep in this region are encountered during or immediately after the end of the rainy season. Temperature also influences the development of nematode larvae and the optimal temperature for the development of most trichostrongylid larvae is 22-30°C. No development of trichostrongylid larvae occurs below 5°C while temperatures above 40°C are lethal. Some trichostrongylid larvae such as *T. colubriformis* and *O. columbianum* are known to be resistant to desiccation and this ability enables them to survive under extremely low or high temperatures.

Gastrointestinal nematodes can survive harsh conditions by hypobiosis or arrested development of larvae (usually L3 or early L4) within the host. In the absence of hypobiosis nematodes survive in hosts during the hot and dry season as adults. The humid tropical climate is favourable for the survival, development and transmission of gastrointestinal nematodes throughout the year.

Management systems: Management systems for the animals have a strong influence the epidemiology of gastrointestinal nematodes. High stocking density increases the contamination of the environment with nematode eggs or larvae and thus makes the infective stages to be more accessible to susceptible animals. High stocking rates and intensive management with little or minimal rotational grazing, are associated with high pasture contamination and outbreaks of clinical helminthosis. On the other hand, low stocking rates and extensive management systems in the traditional husbandry systems preclude a built-up of high worm burdens. The concentration of animals at watering points particularly during the dry season may also result in massive contamination of pastures with eggs or larvae leading to outbreaks of parasitic gastro-enteritis. Tethering of goats and sheep during the wet season which is common in many agro-pastoral societies has been reported to result in increased environmental contamination with infective larvae and incidence of clinical disease. Outbreaks of parasitic gastro-enteritis in such systems have been reported in Tanzania, Nigeria, Kenya and Cameroon. However, if tethered animals are moved each day to fresh ground and the number of animals in the area is small, the risk of helminthosis is reduced.

Similarly, if animals are totally confined and fed on helminth free diets the risk of helminthosis is reduced.

Anthelmintic treatment reduces the prevalence and severity of gastrointestinal nematode infections and may significantly influence their epidemiology. However, the effectiveness of anthelmintic treatment regimes depends on a thorough knowledge of other factors which influence the epidemiology of nematodosis. Indiscriminate use of anthelmintics may result in the development of resistant nematode strains and this

problem is increasing in importance across the sub-Saharan region.

Host factors: The incidence rate and severity of infection with gastrointestinal nematodes can also be influenced by host factors such as age, breed, nutrition, physiological state and presence or absence of intercurrent infections. For instance, kids and lambs are known to be more susceptible than adults and there is a tendency for the worm burdens in goats and sheep to decrease with increasing age. Some breeds of goats and sheep are known to be genetically resistant to gastrointestinal nematode infections than others. It has been demonstrated in Kenya that the Small East African (SEA) goats are more resistant to *H. contortus* infection than their crosses with the Toggenburg and the Galla goats. The Red Maasai sheep have also been found to be more resistant to *H. contortus* infection than the Merino, Dorper, Corriedale, Romney Marsh and Hampshire sheep. The West African Dwarf goats and sheep are also known to be resistant to gastrointestinal nematodes.

The physiological status of the animal may influence its susceptibility to gastrointestinal nematode infections. Hormonal changes during late pregnancy and lactation lower the resistance of the host to nematodes and consequently result in the establishment of higher worm burdens. Prolactin and glucocorticoids are considered to be modulators of periparturient egg rise in goats and sheep. The increase in the fecundity of adult worms already in the alimentary tract and non-specific immunological loss of resistance by the does and ewes as a result of stress associated with lambing and kidding are also considered to be responsible for the 'post parturient rise' in faecal egg count in does and ewes. On the other hand, oestrogens have been found to be responsible for the resistance of female hosts to gastrointestinal nematodes.

Poor nutrition lowers the resistance of the animal thus enhancing the establishment of worm burdens and increasing the pathogenicity of the parasites. Consequently, worm burdens tend to be higher in poorly-fed than in well-fed animals. Malnutrition during the dry season has been found to lower the resistance of goats and sheep to *H. contortus* infection in Sierra Leone, Nigeria and Kenya resulting in heavy mortalities while restricted feeding due to tethering during the rainy season has been associated with high nematode burdens and mortality of goats in some parts of Tanzania. Intercurrent infections and other stress factors also enhance the establishment of higher worm burdens.

Parasite factors: The intrinsic multiplication rate of the nematode species determines the rate of establishment and size of nematode burden in the host. The multiplication rate is determined by the fecundity of the adult worms, the prepatent period and the survival and development rate of the parasite in the environment. For example, *H. contortus* and *O. columbianum* have a high biotic potential such that establishment of these nematodes occurs very rapidly as long as environmental factors are favourable. *Trichostrongylus* spp has a lower biotic potential and hence its establishment is slower.

Lungworms

The epidemiology of lungworms is similar to that of gastrointestinal nematodes. A damp and cool environment is very suitable for the development of *D. filaria* and the third stage larva (L3) is resistant to cold. The sporangia of a fungus (*Pilobolus* spp) and dung beetles facilitate the spread of *D. filaria* larvae. Under adverse (dry) conditions the larvae may be inhibited in the lungs. Some animals may harbour adult worms in the lungs and act as carriers which continue to contaminate the pastures and maintain the

infection in the environment. *Muellerius capillaris* and *Protostrongylus* spp have indirect life cycles, with land snails and slugs acting as the intermediate hosts. Therefore, factors which influence the epidemiology of the intermediate hosts will determine the epidemiology of the parasite as well. Moisture is considered to be an important factor determining the survival and availability of land snails and slugs.

Trematodes

As in nematode infections, climate, management systems, parasite and host factors influence the epidemiology of trematode infections in goats and sheep. However, unlike gastrointestinal nematodes, trematodes have indirect life cycles and intermediate hosts play an important role in their epidemiology. Therefore, factors determining the availability, development and survival of intermediate hosts in the environment will also influence the level and severity of trematode infections.

The intermediate host for *F. gigantica* is the aquatic snail, *Lymnaea natalensis* although *L. truncatula* has also been found to serve as an intermediate host of *F. gigantica* in East and Central African highlands. The optimum temperature range for the survival and development of *L. natalensis* is 15-26 °C while temperatures below 10 °C inhibit the development and reproductive activity of the snails. *L. natalensis* requires permanent water bodies with abundant vegetation such as lakes, slow-moving rivers, irrigation ditches and water tanks. Clean, clear and unpolluted water is the most favourable habitat for *L. natalensis*.

Light, suitable temperature (20-25 °C) and availability of oxygen are essential for the embryonation and hatching of *Fasciola* spp eggs. No development occurs below 10 °C and higher temperatures (>35 °C) result in death of the larvae. The humid environment of most sub-Saharan countries is favourable for the embryonation and hatching of *Fasciola* spp eggs throughout the year. The development of miracidia in the snail host is also temperature-dependent. Faeces and manure pats can act as reservoirs of the infection. It has been found that the concentration of animals at watering points during the dry season is a favourable factor for the transmission of *F. gigantica* leading to outbreaks of fasciolosis.

The epidemiology of *Paramphistomum microbothrium* is similar to that of *F. gigantica* although other species of snails such as *Planorbis* spp and *Bulinus* spp may serve as intermediate hosts.

The epidemiology of *Schistosoma* spp is similar to that of *F. gigantica* and *Bulinus* spp is the common intermediate host for *S. bovis* and *S. mattheei* in Africa. In Tanzania, *Bulinus africanus* is the main intermediate host for *S. bovis*.

Cestodes

The cestodes, *M. expansa*, *S. hepatica*, *S. globipunctata* and *A. centripunctata* have indirect life cycles and the intermediate host are the soil-inhabiting oribatid mites such as *Oribatula* spp, *Galumna* spp and *Peloribates* spp. Birds may involved in the dissemination of tapeworm eggs.

Transmission

Gastrointestinal nematodes

The adult nematodes inhabit various regions of the gastrointestinal tract. Females lay eggs which are passed out with faeces of the host. The eggs embryonate and hatch into first (L1), second (L2) and third (L3) larvae, the latter being the infective stage. Animals are infected with *Haemonchus* spp, *Trichostrongylus* spp and *Oesophagostomum* spp by ingestion of L3 with pastures. After ingestion, the L3 of *Haemonchus* spp and *T. axei* develop through the fourth (L4) and fifth (L5) stage larvae and mature into adults in the abomasum while the maturation of *T. colubriformis* and *Oesophagostomum* spp occur in the small and large intestines respectively.

The third stage larvae of *Bunostomum* spp and *Strongyloides* spp enter the host mainly by skin penetration although infection through ingestion is also possible. After skin penetration they are carried into the venous circulation through the heart and the lungs. The larvae penetrate the alveoli, are coughed up and then swallowed. They pass to the small intestine where further development and maturation occur. The larva of *Trichuris ovis* is contained within the egg and the infective L1 is released when the egg is ingested by the host. The prepatent period for most gastrointestinal trichostrongyles is about 21 days.

Lungworms

Adult Dictyocaulus spp are found in the trachea or bronchi where eggs are produced. The eggs are coughed up and swallowed. Hatching of eggs occur in the air passages or in the intestines and it is the L1 which is found in host faeces. Under suitable conditions of temperature and moisture, the L1 hatches into L2 and L3. Infection is acquired through ingestion and the L3 migrate through the intestinal wall and enter the mesenteric lymph nodes where they moult into (L4). The L4 passes through the lymphatic and venous circulation to the heart and then through the pulmonary circulation to the lungs where they enter the alveoli. Maturation occurs in the bronchi or trachea and the mature worms start to produce eggs. Adult *M. capillaris* is found in the alveoli and pulmonary parenchyma while *P. rufescens* lives in the bronchi and their life cycles are similar to that of Dictyocaulus spp.

Trematodes

Adult *Fasciola* spp lay eggs in the bile ducts and the eggs are transported to the gall bladder through the bile. When the gall bladder contracts the eggs enter the duodenum and then are expelled from the host with the faeces. Under optimum conditions of temperature and moisture the eggs hatch into miracidia. The latter actively penetrate snail hosts and develop through the sporocyst, redial and cercarial stages. The cercariae leave the snail hosts, encyst onto herbage just below the water level and become metacercariae, which are the infective stage. The metacercariae are ingested by grazing animals with infected herbage or water. They excyst in the duodenum, penetrate the intestinal wall and pass through abdominal cavity (or sometimes through the blood stream) to the liver where they penetrate the liver capsule. The immature flukes migrate in the liver parenchyma and then enter the bile ducts where they mature and start to produce eggs. The prepatent period of *F. gigantica* in goats is about 90-100 days and may be shorter under heavy infections.

The transmission of *Paramphistomum* spp is similar to that of *Fasciola* spp. However, after excystation and attachment in the duodenum, the immature paramphistomes migrate up the alimentary tract and finally attach to the epithelium of the rumen and reticulum. The prepatent period is 4-5 months.

The life cycle of *Schistosoma* spp is similar to that of *Fasciola* spp but when the eggs are passed out in the faeces of the host they already contain a miracidium which hatches shortly if environmental conditions are optimum. There are no rediae or metacercarial stages in the life cycle of *S. bovis*. The cercaria is the infective stage and infection occurs through skin penetration or ingestion. The cercariae are carried through the lymphatic and blood systems to the mesenteric veins where they mature and commence to produce eggs 6-8 weeks after infection.

Gastrointestinal cestodes

The adults worms are found in the small intestine of goats or sheep. Proglottids and eggs are passed out in the faeces of the infected animal. In the environment, the eggs may be ingested by oribatid mites where they develop into cysticercoids. The cysticercoids which are the infective forms are produced in 1-4 months depending on temperature. Ruminants are infected by the ingestion of the infected mites with herbage. The prepatent period is 5-6 weeks.

Clinical and pathological features

Parasitic gastro-enteritis

The major parasites associated with parasitic gastro-enteritis in small ruminants in sub-Saharan Africa are *H. contortus*, *T. colubriformis* and *O. columbianum*. Other nematodes of lesser importance are *T. axei*, *B. trigonocephalum*, *T. ovis*, *Cooperia* spp, and *S. papillosus*. *O. circumcincta* and *N. fillicolis* are important in cooler highland areas. Due to the differences in their predilection sites and pathogenetic mechanisms, gastrointestinal nematodes present with differing clinical and pathological features. However, most commonly in field infections the clinical and pathological features of parasitic gastro-enteritis are due to the additive pathogenetic effects of several nematodes.

Haemonchosis

Haemonchosis is considered to be the most economically important disease of goats and sheep in Africa. The pathogenesis of haemonchosis is related to the blood sucking habit of the parasite. Three syndromes; hyperacute, acute and chronic haemonchosis occur in goats and sheep.

Hyperacute haemonchosis occurs when there is a sudden massive challenge of susceptible animals with infective larvae resulting in severe blood loss due to haemorrhagic gastritis. The syndrome is of short duration and is characterised by sudden death although in some animals dark coloured faeces may be seen before death. Faecal egg count of up to 400,000 may be encountered in the affected animals.

Hyperacute haemonchosis has limited gross pathological features due to sudden death, although a large number of immature or young adults may be found on the

abomasal mucosa at *post mortem* examination. There are also multiple erosions and petechiae of the abomasal mucosa and minimal expansion of the bone marrow. However, hyperacute haemonchosis is not very common in field infections.

Acute haemonchosis occurs when animals are exposed to a continuous challenge from eggs leading to gastritis associated with hypoproteinemia and generalised oedema. Other signs include weakness, pallor of the mucous membrane, lethargy or agalactia which may lead and starvation death of kids. Dark coloured faeces are often observed. Self-cure may occur at any stage of the disease. The syndrome is more common when young susceptible animals become infected.

Pathologically, acute haemonchosis is characterised by a pale and watery carcass. The abomasal mucosa is petechiated and oedematous with many parasites on its surface and contents. There is a marked expansion of the bone marrow throughout the medullary cavity which may extend up to the epiphyses.

Chronic haemonchosis is the common form of field infection. The syndrome is caused by gradual intake of infective larvae and the course of disease may take 2-6 weeks. It is a chronic gastritis with chronic blood loss and abomasal dysfunction leading to weakness, progressive weight loss, rough hair coat and stunted growth. Chronic haemonchosis is aggravated and often confused with malnutrition.

At necropsy, chronic haemonchosis is characterised by pallor carcass, hyperplastic thickening of the abomasal wall, chronic expansion of the bone marrow and resorption of the cancellous and cortical bones. In terminal stages the bone marrow reverts to white due to exhaustion.

The clinical pathology of haemonchosis includes lowered packed cell volume, haemoglobin concentration, erythrocyte counts and serum iron.

Trichostrongylosis

Trichostrongylosis is commonly a disease of young animals. The penetration of larvae and adult worms into the intestinal mucosa results in desquamation of the latter causing a malabsorption syndrome and hence, a protein-losing gastroenteropathy and hypoalbuminemia. Heavy infections cause an acute enteritis which is characterised by dark-coloured diarrhoea and foul smelling faeces. There may be sudden death without evidence of anaemia or emaciation but weakness of the legs is a frequent feature. Most commonly, trichostrongylosis is a chronic wasting disease characterised by loss of appetite, emaciation, loss of weight, dry skin, diarrhoea, oedema and atrophy of skeletal muscles or myocardium.

At necropsy, the acute disease is characterised by a swollen and haemorrhagic or catarrhal intestinal mucosa and, worms may be found in the mucosal scrapings. The chronic disease is characterised by an emaciated carcass, fatty degeneration and, a thickened, inflamed and ulcerated intestinal mucosa. Histopathologically, chronic infection with *T. colubriformis* is characterised by marked villous atrophy, flattening of the intestinal mucosa and osteoporosis. The clinical pathology is characterised by hypoalbuminaemia, hyperglobulinaemia and hypophosphataemia.

Oesophagostomosis

The pathogenicity of *O. columbianum* is related to the migration of larvae in muscularis mucosa of the large intestine resulting in a fibroblastic response around the larvae forming fibrous nodules. The extensive nodular formation interferes with digestion, absorption and bowel movements. Mucoïd diarrhoea or sometimes constipation, emaciation, general weakness, dry skin, prostration and death are the common clinical features. The diarrhoea often coincides with the emergence of larvae from the nodules. The nodules are frequently invaded with pyogenic bacteria which cause suppuration. Rupture of the nodules may cause peritonitis and multiple adhesions.

At *post mortem* examination, the most severe form of the disease is characterised by ulcerative colitis. The adult worms cause thickening of the bowel wall, congestion and production of excessive amount of mucus. In primary infection, adult worms are found in the lumen of the large intestine where they are often covered with mucus and there may be few nodules. In superinfection, there is an extensive nodular formation with marked emaciation and severe fatty degeneration. Abscesses with greenish or yellowish pus may also be present.

Bunostomosis

Bunostomosis is characterised by progressive anaemia, emaciation, weakness or paresis, submandibular oedema, dark coloured faeces, prostration and death. At *post mortem* examination there is hydrothorax, hydropericardium and pin-point haemorrhages in the small intestine or blood in its content.

Other gastrointestinal nematodes

Other gastrointestinal nematodes such as *Cooperia* spp, *T. ovis* and *S. papillosus* have limited pathogenicity. Their clinical and pathological features are often masked with the more pathogenic species. However, heavy challenge with these parasites particularly in young animals may be associated with anorexia, weight loss, moderate anaemia and inflammation of the intestinal mucosa.

Parasitic bronchitis

Parasitic bronchitis (verminous pneumonia) caused by *D. filaria* and *M. capillaris* is more common in kids or lambs under 6 months than in other age groups. Adult *D. filaria* cause alveolar and bronchiolar irritation leading to coughing, dyspnoea and loss of body condition. Secondary bacterial infection may lead to toxæmia.

At necropsy, there is pulmonary oedema and emphysema with consolidation of some parts of the lung. The bronchioles are filled and may be blocked with exudate. Bronchoectasis frequently accompanies secondary bacterial infections. In *M. capillaris* infection, nodular circumscribed and raised grey or greyish white patches are observed especially on the dorsal and lateral aspects of the diaphragmatic lobes. Consolidation of the ventral portion and antero-ventral aspects of the diaphragmatic lobes is frequently observed. There may also be fibrinous strands which are adherent to the thoracic wall and a frothy fluid may be expressed from cut lung surface.

Histopathologically, eggs, larvae or adult worms are found in the air passages and in the interalveolar spaces. Mucopurulent exudate containing inflammatory cells such as lymphocytes, fibrocytes, mononuclear phagocytes, eosinophils and foreign body giant cells fill the bronchial and alveolar lumina. The inflammatory cells may also be found in the blood vessels accompanying bronchioles. Thickening of the interalveolar connective tissue, erosion of bronchiolar epithelium, mucosal hyperplasia, peribronchial lymphoid hyperplasia and epithelialisation of the alveoli are common features.

Trematode Infections

Fasciolosis

F. gigantica infection is associated with a clinical disease in goats and sheep even when the fluke burden is light. It has been found that as few as 42 flukes can cause clinical fasciolosis in goats. The disease is more severe in goats than in sheep. Three syndromes; acute, subacute or chronic fasciolosis may occur.

Acute fasciolosis occurs when there is an acute traumatic hepatitis caused by the migration of larvae through the parenchyma leading to extensive destruction and marked haemorrhage. The haemolytic crisis results in progressive weakness, pallor of the mucous membranes, enlargement of the liver and abomasal distension. Anorexia, paresis prior to death and anasarca are observed in terminal stages of the acute disease in goats. At necropsy, acute fasciolosis is characterised by the presence of a blood-tinged fluid in the peritoneal cavity, fibrinous exudate covering the liver surface, hepatomegaly and numerous haemorrhagic and friable tracts in the liver parenchyma. The gall bladder is also enlarged. Immature flukes can be expressed from the cut liver surface. Adhesion of the liver to the diaphragm or other internal organs may occur.

Subacute fasciolosis is associated with ingestion of a large number of metacercariae over a long period of time. The syndrome is characterised by anorexia, rough hair coat, slight abdominal distension, pallor of mucous membranes, disinclination to move and emaciation.

Chronic fasciolosis is a persistent wasting disease characterised by emaciation, anaemia and submandibular oedema. At *post mortem* examination, fibrosis and thickening of the bile ducts resulting from cholangitis is evident. The bile ducts may be blocked with flukes and desquamated epithelial cells. The damaged parenchyma becomes indurated and flukes may be seen in the bile ducts with granulomata often being observed around fluke remnants. Calcification of the bile duct walls which is commonly observed in cattle is not a feature of fasciolosis in small ruminants. *F. hepatica* produces a disease similar to *F. gigantica*.

The clinical pathology of acute fasciolosis is characterised by a normochromic anaemia, eosinophilia, hypoalbumaemia and elevation of plasma glutamate dehydrogenase and gamma-glutamyl aminotranspeptidase. The two enzymes are very sensitive indicators of the liver damage. Plasma sorbitol dehydrogenase and aspartate aminotransferase are also elevated. The elevation of plasma aspartate aminotransferase is more notable in the fourth week of infection coinciding with the migration of the immature flukes in the liver.

Paramphistomosis

This is an acute enteritis caused by the migration of the immature flukes in the duodenal

mucosa and is characterised by profuse and foetid diarrhoea, dehydration, loss of body condition, weakness, pallor of the mucosae and submaxillary oedema. In some countries, outbreaks of the disease have been reported to occur during the dry season following concentration of animals at watering points and subsequent massive contamination of the environment with fluke eggs.

At *post mortem* there is a marked haemorrhagic enteritis with large numbers of the parasites on the mucosa or contents of the duodenum and upper ileum, subcutaneous oedema, gelatinous fatty degeneration. Extensive catarrhal or haemorrhagic duodenitis or jejunitis with destruction of associated glands and lymph nodes are the main histopathological features. Young flukes may be found embedded in the duodenal mucosa. There is a marked fall in total plasma proteins due to increased leakage of plasma albumin.

Schistosomosis

Intestinal and hepatic forms of the disease are distinguished. The intestinal syndrome is related to the damage caused by passage of large numbers of eggs in the intestinal mucosa and is characterised by diarrhoea or dysentery, dehydration, anorexia, loss of weight and profuse, foetid or intermittent diarrhoea, submandibular oedema and anaemia. Neuralgical signs may be observed in the chronic form of the disease. The hepatic syndrome occurs when eggs are washed back to the liver by portal circulation during penetration of the intestinal wall resulting in hepatic damage. The clinical and pathological features of the hepatic syndrome resemble those of fasciolosis

Pathologically, the intestinal form of the disease is characterised by petechiae or ecchymoses and granulomata in the gastrointestinal mucosa and, oedema and pallor of the carcass. In the hepatic syndrome there is hepatic infarction, portal fibrosis, thrombosis and dead parasites may be expressed from the cut vessels. There may also be hydrothorax, hydropericardium and ascites.

Cestode infections

The pathogenic effects of *Moniezia* spp are limited and the parasite is considered to be non-pathogenic. However, heavy infections in young animals may cause anorexia, weight loss, moderate anaemia, inflammation of the intestinal mucosa and sometimes obstruction of the intestines. *S. hepatica* occurs in the bile ducts of small ruminants and its economic importance is associated with condemnation of the affected livers. Migration of *C. cerebralis* in the brain may cause meningo-encephalitis while massive numbers of cysts of *E. granulosus* in the lungs may cause respiratory problems. Other cestodes have limited clinical significance in small ruminants.

Diagnosis

Diagnosis of helminthosis is based on history, epidemiological, clinical and pathological findings and laboratory analysis of appropriate samples. The most commonly used laboratory methods for diagnosis of gastrointestinal nematodes are faecal egg counts, faecal cultures, determination of infective larvae on herbage and worm counts at *post mortem*.

Nematodes

Faecal egg counting is the most common antemortem means of diagnosis of nematodosis and is based on the assumption that the size of worm burden in animals may be accurately deduced from faecal egg counts. Faecal egg counting is a cheap and easily performed technique. The McMaster technique is a rapid, easy and most commonly used method in quantitative analysis of nematode egg counts in the field and in epidemiological surveys. However, the number of nematode eggs or larvae in faeces is influenced by several factors such as the biotic potential of the nematode species, resistance of the host, developmental stage of the parasite, season of the year, quantity and consistency of the faeces passed and, sensitivity of the diagnostic method used. Therefore, the interpretation of faecal egg counts should take into account these factors.

Faecal cultures are based on the fact that the infective larvae of different genera of nematodes differ morphologically hence their examination permits a specific diagnosis of nematode infections. However, faecal culturing is a slow process and does not take into consideration the differences in fecundities of different nematode species.

Estimation of number of infective nematode larvae on pastures provides an indication of the level of infection to which grazing animals are exposed. The technique is useful in the diagnosis and prognosis of a nematode infection on farm and in epidemiological investigations. However, the larval population in herbage is affected by rainfall, herbage cover and stocking density. The method and time when herbage samples are taken may also affect the number of larvae recovered. These factors should be considered when interpreting the significance of the number of infective nematode larvae recovered from pastures. Tracer or sentinel animals can be used alone or in conjunction with herbage larvae determination to provide an indication of the availability of infective larvae on pastures. The use of tracer animals is more reliable than herbage sampling but it is limited by its cost.

Post mortem worm counting permits identification of adult worms and a direct count of worms present in an animal thereby providing a precise assessment of the worm burden. *Post mortem* worm counting is considered to be the most accurate method of diagnosis of helminthosis, however, it is also expensive.

Other helminths

Patent *Fasciola* spp, *Paramphistomum* spp and *Schistosoma* spp infections can be diagnosed by faecal egg and *post mortem* worm counting. Examination of eggs in faeces is most commonly used in routine diagnosis of chronic fasciolosis but a more precise assessment of the fluke burden of an animal can be made *by post mortem* examination and identification of immature and mature flukes.

Moniezirosis and **stilesiosis** can be diagnosed by demonstration eggs or proglottids in host faeces whereas, the adult worms can be found in the small intestine or bile ducts at *post mortem* examination.

Other methods used in the diagnosis of helminth infections include immunodiagnosis and enzyme assays. However, these methods are used where laboratory facilities are adequate and are of limited use in direct field investigations of helminthoses. Helminthosis especially haemonchosis closely resembles trypanosomosis but the mortality rate is higher in haemonchosis than in trypanosomosis and, in addition, the demonstration of

trypanosome in circulation can rule out haemonchosis. However, it should be borne in mind that concurrent infections of helminthosis and trypanosomosis in the field is very common.

Treatment

Many anthelmintics which are effective against different species of helminths affecting small ruminants have been developed. Benzimidazoles, imidazothiazoles, tetrahydropyrimidines, organophosphates and ivermectins form the major classes of anthelmintics.

The following benzimidazoles are used to treat gastrointestinal nematodes, lungworms and some tapeworms; albendazole (5.0-10 mg/kg), fenbendazole (5.0-7.5 mg/kg), mebendazole (12.5 mg/kg) and oxfendazole (4.5-5.0 mg/kg), oxibendazole (15 mg/kg), parbendazole (20 mg/kg) and thiabendazole (80 mg/kg). Oxibendazole is not effective against *Trichostrongylus* spp while parbendazole is not effective against *Bunostomum* spp. Fenbentel and thiophanate are effective against gastrointestinal nematodes, lungworms and anoplocephalid tapeworms. The imidazothiazoles, tetramisole (15 mg/kg) and levamisole (7.5 mg/kg) and the tetrahydropyrimidines, morantel (7.5 mg/kg) and pyrantel (15 mg/kg) are also used to treat gastrointestinal nematodosis. The organophosphates; coumaphos, haloxon, trichlorfon, naphthalophos and dichlorvos have also been used to treat parasitic gastro-enteritis in small ruminants. Ivermectin 0.2 mg/kg is very effective against gastrointestinal nematodes and immature stages of lungworms in goats.

Triclabendazole (10 mg/kg), rafoxanide (5.0-10.0 mg/kg), brotianide (10-15 mg/kg), nitroxylin (8-15 mg/kg), diamphenetide (80-120 mg/kg) and niclofolan (4-8 mg/kg) are effective against both the immature and mature stages of *Fasciola* spp while oxyclozanide (15 mg/kg) has been found to be effective against the adult stages only. Albendazole and oxfendazole are also effective against *Fasciola* spp.

Niclosamide (90 mg/kg), brotianide (15 mg/kg) and closantel have been shown to be effective in treating paramphistomosis whereas, praziquantel (15 mg/kg) and trichlorfon are the drugs of choice for the treatment of schistosomosis.

Niclosamide (80 mg/kg), resorantel (75 mg/kg), praziquantel (15 mg/kg), bunamidine (25-50 mg/kg), cambendazole (25-35mg/kg), albendazole (5 mg/kg), mebendazole (10 mg/kg) and fenbendazole (10 mg/kg) have been in use against cestodes. Benzimidazoles are particularly effective against anoplocephalid tapeworms.

The routes of administration and the dosage forms of various anthelmintics are documented in detail in standard textbooks of veterinary pharmacology and parasitology. It is worth noting that the pharmaceutical industry in most sub-Saharan countries is underdeveloped and hence, most drugs for use in small ruminants are manufactured in the developed countries and their efficacies have been tested mainly in sheep than in goats. Therefore, it is always important to adhere to the manufacturers' instructions before using them.

Control

The control of helminthosis is designed to eliminate or reduce the prevalence of helminths and improve the productivity of the livestock industry. The eradication of helminthosis in animals is difficult and the aim of control is therefore to limit the infection by minimising the challenge to an economically justifiable level. It is therefore important to accurately assess the cost-benefit effectiveness of any helminth control programme if production from the animals is to be optimised. Effective control of helminthoses can be achieved by judicious use of anthelmintics and good management. The methods of control of helminthosis can be grouped into three main categories;

Control by use of anthelmintics: This method aims at eliminating the parasitic stages of the parasite in the host thus preventing the discharge of eggs and larvae into the environment. Chemoprophylaxis is extensively used in the control of helminthosis throughout the world. Since the species of helminths and epidemiological factors vary from one place to another, the strategies for anthelmintic treatment will vary between different agro-ecological zones within a country. It is therefore recommended that the frequency of treatment should be determined by the epidemiology and biology of the dominant parasite, the stocking rates of the particular area and the benefits accrued from the control regime adopted.

In Nigeria, for example, some workers recommend treatment at the end of the rainy season because the helminth burdens in small ruminants tend to be higher during these times. Others recommend anthelmintic treatment in the early dry season in order to eliminate the declining worm burdens and because there will be no recrudescence of worm population due to resumed development of inhibited larvae. In Cameroon, some workers have found that treatment at the beginning or middle of the rainy season will keep the worm burdens throughout the year at a minimum level. In Kenya, some workers have recommended treatment just before the beginning of rains because helminth burdens are found to be higher during the dry season while others recommend a four weekly treatment interval. In southern Tanzania, a single anthelmintic treatment at the end of the dry season has been found to minimise worm burdens during the rainy season while in the northern part of the country treatment at the beginning and end of the dry season has been recommended. Other studies conducted in Tanzania, have shown that treatment of goats two weeks after the onset of rains and at a four-weekly interval during the rainy season minimises pasture contamination with helminth eggs thereby reducing the number of infective larvae on pastures during the dry season.

In small-holder farming systems where worm burdens are generally low, treatment of clinical cases may be more practical, economical and acceptable by farmers than mass treatment. Furthermore, small farmers unlikely to spend their meagre income to purchase drugs for regular treatment.

It should also be remembered that frequent use of anthelmintics interferes with the development of immunity of animals against helminths and indiscriminate use of anthelmintics can lead to the selection of drug resistant helminths. Anthelmintic resistance has been reported to occur in goats and sheep in Kenya, Tanzania and South Africa and, it is likely to be a serious problem in many other countries.

Control through management: This method aims at reducing the contact of susceptible animals with infective larvae. Control of helminthosis by good management is considered

to be more sustainable than the use of anthelmintics and may be the most practical method for use by small-holder farmers who cannot afford to purchase expensive drugs. Rotational grazing, separation of animals according to age groups, alternate grazing by different species of hosts, adjustment of stocking rates, improvement of nutrition and better housing systems are the common management manoeuvres employed in the control of helminthosis in most countries.

(i) Rotational grazing is commonly used in modern production systems but is of limited use under communal grazing systems. However, a rigid rotational grazing system may delay the development of immunity in animals. Grazing of kids or lambs ahead of adults prevents them from acquiring heavy worm burdens from pastures contaminated with helminth eggs or larvae from the adult animals.

(ii) Alternate grazing by different host species depends on the degree of cross-transmission of parasites between host species. Alternate grazing of cattle and small ruminants is practised in some ranches in South Africa, Kenya and Zimbabwe. Although this practice may reduce the overall burden of the species in question, the reduction may not be sufficient for efficient parasite control.

(iii) Reducing the stocking rates reduces pasture contamination with helminth eggs or larvae thereby reducing the acquisition of heavy worm burdens by animals.

(iv) The use of parasite-free forage, slatted-floor housing systems and raised feed or water troughs has been found to be effective in minimising infections of animals with helminths. Provision of adequate nutrition also lessens the pathogenic effects of helminths on animals.

Control by breeding resistant stocks: This method is based on genetic resistance of different breeds of animals to helminth infections. The resistance of indigenous breeds of goats and sheep to hemonchosis compared to exotic or crossbred ones is well documented. Breeding of resistant stock can be possibly adopted as a long term strategy for the control of helminthosis. Presently, this method of control of helminthosis is not widely used in sub-Saharan countries but it still has a great potential if well pursued.

Control by immunisation: Vaccination for helminth control is based on the ability of some helminths to stimulate an immune response that can persist after infection. It has been demonstrated that vaccination of adult sheep with X-irradiated *H. contortus* larvae confers immunity to subsequent challenge. However, little work has been done regarding the control of helminthosis in small ruminants by immunisation in the sub-Saharan region.

In general, the ideal approach towards an effective control of helminths in small ruminants is the integration of the several methods because each method has its own advantages and disadvantages.

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CHAPTER 3 DISEASES CAUSED BY BACTERIA

The most common bacterial diseases affecting small ruminants in sub-Saharan Africa are pneumonia, brucellosis, footrot, dermatophilosis, caseous lymphadenitis, anthrax and clostridial infections such as blackquarter, tetanus, malignant oedema and enterotoxaemias. Colibacillosis and salmonellosis are also encountered particularly under the intensive production systems.

PNEUMONIA

Pneumonia refers to the inflammation of the pulmonary parenchyma usually accompanied by the inflammation of bronchioles and often pleurisy and, it is characterised by respiratory embarrassment or sometimes toxæmia. Upper respiratory infections are accompanied with respiratory abnormalities and very often they descend to pneumonia.

Aetiology

The aetiological classification of bacterial pneumonia is complicated by the fact that many types of bacteria may be isolated from the same pneumonic lesions. However, *Pasteurella spp* are the most common bacteria isolated from cases of clinical pneumonia in goats and sheep. *P. haemolytica* biotype A is the commonest isolate from pneumonic pasteurellosis although *P. haemolytica* biotype T and *P. multocida* may also be encountered. Other bacteria isolated from pneumonic lungs of goats and sheep in sub-Saharan Africa include *Corynebacterium pyogenes*, *Streptococcus spp*, *Staphylococcus aureus*, *Proteus mirabilis*, *Pseudomonas aeruginosa* and *Escherichia coli*. Bacteria and mycoplasmas are commonly involved together in the pathogenesis of pneumonia in goats and sheep.

Epidemiology

Pneumonia is widespread among goats and sheep in sub-Saharan Africa and it is considered to be one of the most important causes of losses in the small ruminant industry. Pneumonia has been reported to be responsible for heavy losses in goats in Nigeria, Ghana, Mali, Sudan, Kenya, Somalia, Tanzania, Republic of South Africa and many other sub-Saharan countries. Morbidity has been estimated to be 33 % in West Africa and 24 % in East Africa while mortality up to 4 % is commonly encountered. These figures are gross underestimates because they are based on few studies in areas accessible to researchers and on abattoir records while the prevalence and importance of the disease in the traditional sector where many goats and sheep are kept have not yet been well studied.

Some of the bacteria commonly isolated from pneumonic lungs such as *Pasteurella spp*, *Staphylococcus spp*, *Streptococcus spp* and *Corynebacterium spp* are normal flora of the respiratory tract. Predisposing factors such as poor ventilation in animal houses, inclement weather, exhaustion during transport, severe parasitism are important in the epidemiology of pneumonia. Kids and lambs are more susceptible than adults. Some workers in West Africa have found that the incidence of pneumonia is higher in

intensively managed than in semi-intensively or extensively managed goats while the harmattan winds have also been found to be associated higher incidences of pneumonia in small ruminants. In Somalia, malnutrition during the dry season is considered to be the major predisposing factor to caprine and ovine pneumonia while in Tanzania, long distance trekking of goats from rural areas to markets near or in urban centres and overcrowded housing conditions are considered responsible for many cases of pneumonia in small ruminants encountered in slaughter houses and abattoirs. The poor housing systems characteristic of traditional small ruminant management systems in most sub-Saharan countries subject animals to various stresses such as cold, wind, rain and dust which predispose them to pneumonia.

Pathogenesis

The main route of transmission of pneumonia and other respiratory infections is by inhalation of infective aerosols. The pathogenesis of the disease depend on the presence of virulence factors in the bacterium, host immunity and presence or absence and severity of the predisposing factors. *P. haemolytica* possess adhesive fimbriae, secrete proteolytic enzymes and a cytotoxin, all of which enhance its establishment on the respiratory system. The fimbriae facilitates attachment on the mucosa, proteolytic enzymes break down the mucosal barrier and impair the mucocilliary function of the respiratory tract thus facilitating colonisation and, the cytotoxin cause lysis of respiratory tract cells. The presence of other pathogens in the respiratory tract such as para-influenza-3 virus and adenoviruses disrupt the phagocytic mechanisms and lower the host immunity thus favouring proliferation of pasteurellae.

Clinical features

Acute, subacute and chronic pasteurellosis may occur. Acute pneumonic pasteurellosis is characterised by laboured breathing, coughing, nasal discharges, lacrimation, anorexia, depression and sometimes pyrexia if there is a systemic involvement. Exercised animals exhibit tachypnoea and dyspnoea. Some of the acutely affected animals may die without showing any clinical signs. The subacute and chronic diseases are mainly characterised by unthriftiness.

Pathological features

The gross pathological features of pneumonic pasteurellosis include oedema, haemorrhage, congestion, emphysema and red or grey hepatisation of the lung tissue. The lesions are found predominantly on the cardioventral aspects of the lung although other areas may also be affected. Tracheal froth, adhesive pleuritis and enlargement of bronchial and mediastinal lymph nodes are common features. A serofibrinous or fibrinopurulent exudate may be expressed from the bronchioles of the cut lung surface. Other features include presence of gelatinous exudate over the pericardium and straw-coloured exudate in the pleural cavity and in the interlobular spaces resulting in distension of the interlobular septa. Pulmonary abscess and adhesive fibrinous pleuritis and pericarditis are features of the subacute and chronic syndromes. Abscesses may be encountered in regional lymph nodes.

At histopathology, acute pneumonic pasteurellosis is characterised by dilation of the alveolar capillaries which are engorged with blood and mucus. The lumina of the bronchi, bronchioles and alveoli contain a serofibrinous exudate which is mixed with neutrophils,

macrophages and desquamated epithelial cells. There may be diffuse alveolar necrosis, oedema of the interlobular septae and necrosis of the bronchial mucosa. Gram-negative bacteria are abundant around the necrotic foci. Thrombosis of the blood and lymphatic vessels is also evident. Abscessation, organisation and fibroplasia of the affected tissue are evident in the subacute and chronic syndromes.

Diagnosis

A tentative diagnosis of pneumonia can be achieved by consideration of the epidemiological, clinical and pathological findings. In *Pasteurella* spp infections, Gram- or methylene blue-stained smears from the lungs reveal Gram-negative Small coccobacilli which may show bipolar staining. The bacteria can be isolated from the lung lesions, pleural exudates and mediastinal lymph nodes by culturing on sheep or ox blood or MacConkey agar. The cultures are incubated aerobically at 37 °C for 24-48 hours. In MacConkey agar *P. haemolytica* produces pin-point red colonies surrounded by a narrow β -haemolytic zone. *P. multocida* colonies are larger, non-haemolytic and, may be mucoid and produce a characteristic sweetish odour. *P. multocida* does not grow on MacConkey agar. In septicaemic cases a large number of organisms can be isolated from the liver, spleen, kidneys, heart blood, pharyngeal and oesophageal mucosa. Intraperitoneal mice inoculation with suspensions of the organisms from affected tissues elicits a clinical disease with large numbers of the bacteria in tissues.

Acute pneumonic pasteurellosis should be differentiated from contagious caprine pleuropneumonia (CCPP) and other mycoplasmal pleuro-pneumonias, aspiration pneumonia and septicaemic colibacillosis. CCPP does not affect sheep and can be confirmed by isolation and identification of the causative *Mycoplasma* spp. Aspiration pneumonia is commonly associated with gangrenous lesions which are not common features of acute pneumonic pasteurellosis. Chronic pneumonic pasteurellosis can be differentiated from abscesses caused by *C. pseudotuberculosis* and *Actinomyces pyogenes* by isolation and identification of the causative bacteria while verminous pneumonia can be confirmed by demonstration of eggs or larvae in the bronchi, bronchioles and alveoli. Ovine progressive pneumonia can be differentiated from chronic pneumonic pasteurellosis by histopathological examination. Pneumonia caused by other bacteria can be confirmed by bacteriological tests.

Treatment and Control

P. hemolytica is sensitive to oxytetracycline at 20 mg/kg given parenterally. The treatment should be repeated after 4-6 days because relapses may occur. Penicillins are also used although some strains of *P. haemolytica* are not responsive to Penicillins. Ampicillin, sulphadimidine and trimethoprim-sulphonamide combinations have also been found to be effective.

There is no effective vaccine against bacterial pneumonia because of the diversity of aetiologic bacteria and serotypes but in some countries, vaccination of lambs and kids with vaccines prepared from local strains of *P. haemolytica* have been used. Control of pneumonia in a herd can be achieved by isolation and treatment of the affected animals. Avoidance or minimisation of predisposing factors such as overcrowding, long distance trekking and inclement weather can greatly reduce the incidence of pneumonia in a herd or farm.

BRUCELLOSIS

This is a disease caused by infection with bacteria of the genus *Brucella* and it is characterised by abortion in late pregnancy and subsequent high rate of infertility. The disease is zoonotic and occupational causing undulant or Malta fever in man.

Aetiology

Brucellosis in goats and sheep is normally caused by a Gram-negative coccobacillary rod, *Brucella melitensis* although *Brucella abortus* may also cause clinical brucellosis. *Brucella ovis* is a cause of epididymitis of rams but it has also been associated with abortions and infertility. *B. melitensis* infection causes a fulminating disease in man (undulant or Malta fever) which is characterised by intermittent fever, malaise, fatigue, night sweats, muscle and joint pains whereas, *B. abortus* causes a mild disease. Osteomyelitis is a common complication in human brucellosis.

Epidemiology

Brucellosis has been reported to be an important cause of reproductive losses in small ruminants in some sub-Saharan countries. For example, the seropositivity of goats for *B. melitensis* infection in the northern, eastern and western parts of Nigeria has been estimated to be 4.3-12.5 % while 9.4-14.5 % of sheep in the same area have also been found to be *B. melitensis*-seropositive. A 36.8% and 69.6% reactor rate of goats and sheep to *B. ovis* in Northern Nigeria has also been reported. In Central Ethiopia, about 1.5% of goats and 1.5% of sheep have been reported to be brucellosis-seropositive while in Tanzania, the prevalence of brucellosis in goats and sheep has been estimated to be 4.3 % and 2.2 % respectively. *B. melitensis* infection in goats has also been reported to occur in Somalia, Kenya, Zambia, Malawi, Namibia and South Africa. Although correct estimates of the losses associated with brucellosis in most sub-Saharan countries are lacking, the disease may be responsible for many cases of reproductive wastage and infertility in small ruminants. The consumption of raw milk in rural areas may be a potential public health hazard since it is possible for the milk to be infected with *Brucella* spp. Human brucellosis caused by *B. melitensis* has been reported in Kenya.

The source of infection is the infected doe or ewe and *Brucella* spp tend to be abundant in the placenta, placental fluid, uterine exudate and aborted foetuses. The bacteria may persist in the uterus for about 5 months after abortion. Inhalation is the most important route of infection in goats and sheep but infection may also be acquired through ingestion of infected material and by penetration of the bacteria through the conjunctival mucosa. *In utero* transmission may occur. The infective discharges can contaminate the environment very rapidly causing grazing animals to ingest massive numbers of the organisms. *B. melitensis* is known to be the most pathogenic of the *Brucella* spp and is more contagious than *B. abortus*. Overcrowding of animals in houses, communal grazing areas and water sources and, poor hygiene favour the rapid spread of the disease. The unrestricted movement of animals and personnel can facilitate the transmission of brucellosis between herds. Man can be infected through handling of contaminated materials, consumption of infected meat or milk or accidental inoculation with the live attenuated Rev *B. melitensis* vaccine.

Pathogenesis

After infection, *Brucella* spp multiply in macrophages and neutrophils of the regional lymph nodes causing lymphadenitis. This is followed by bacteraemia and subsequent localisation of the bacteria in various organs. The gravid uterus is the primary target organ but the organisms also lodge in the mammary tissue and supramammary lymph nodes. *Brucella* spp produce an exotoxin which enables it to establish and cause lesions. The consequences of the infection are determined by the virulence of the bacteria, resistance and reproductive status of the host. The presence of erythritol, steroid hormones and other substances in the uterus, placenta and foetal fluids favours the proliferation of *B. melitensis*. Spontaneous recovery usually occurs in animals which were infected when not pregnant.

Clinical features

Abortion storm in late pregnancy is the principal manifestation of brucellosis. An abortion storm involving about 60 % of the pregnant does in the farm or herd is common. Other features include reduced milk yield and birth of weak kids or lambs which become asymptomatic carriers.

An acute septicaemic form of brucellosis may occur and is characterised by fever, depression, weight loss and sometimes diarrhoea. The presence of bacteria in the mammary tissue may cause mastitis. Epididymitis, orchitis, synovitis, hygromas, osteoarthritis, lameness and infertility are usually observed in male animals. *B. abortus* infection has been associated with neonatal deaths in lambs in Nigeria.

B. ovis infection in rams causes inflammation of the scrotum which is manifested by oedema, enlarged and hard palpable epididymis and, obliteration of the groove between the testis and epididymis. In the advanced stages of the disease the testis become atrophic. The infection in ewes is characterised by abortion, stillbirths or birth of weak lambs.

Pathological features

The pathological features of *B. melitensis* infection are mainly localised to the genital organs. Greyish-white necrotic areas are observed in the placenta and there is a brownish red exudate between the allantochorion and the endometrium. Acute endometritis is a common feature. Abscesses may also be present in the spleen and other parenchymatous organs. Histopathologically, there are necrotic foci around the placentomes and granulomatous foci may be encountered in the costochondral junction.

Thickening and fibrosis of the tunicae, granulomata or caseation necrosis of the testis and infiltration of the semen with inflammatory cells are the main features associated with *B. ovis* infection in rams. The semen is characterised by reduced total sperm count, morphologically abnormal sperm cells which also have poor motility. In the affected ewes, there is purulent exudate in the uterus, necrosis of the uterine surface, thickening of the placenta and raised yellowish-white or whitish areas in the intercotyledonary area. The aborted foetus is oedematous.

Diagnosis

The clinical history, endemicity of the disease in the area and clinical signs may be

suggestive of the disease. The disease can be confirmed by demonstration of the bacteria in smears made from the vaginal discharges, placenta, colostrum and the abomasum of the aborted foetus using the modified Ziehl-Neelsen stain (MZN) or Koster method. In MZN-stained smears the bacteria appear as red intracellular coccobacilli. Brucellae can be isolated from the abomasal contents and lungs of the foetus; mammary glands; supramammary, retropharyngeal, parotid and mandibular lymph nodes and, seminal vesicles by culturing on 5-10 % blood or selective serum agar. After incubation for about 15 days at 37°C pinpoint, smooth, glistening, bluish and translucent colonies appear. The colonies become opaque as they age. Farrels' medium and Albimi Brucella medium are selective enriched media for isolation of *Brucella* spp.

The serological methods used in the diagnosis of brucellosis include serum agglutination test, Rose Bengal plate test, ELISA, agar gel immunodiffusion and complement fixation test. The complement fixation test is considered to be the most specific and most sensitive method for the diagnosis of brucellosis in small ruminants. The milk ring test, Coomb's test and whey complement fixation test are used to detect the infection in milk. Cross-reaction occur between *B. melitensis* and *B. abortus* but not with *B. ovis*.

Brucellosis should be differentiated from other causes of abortion such as toxoplasmosis, Rift Valley fever, chlamydiosis, campylobacteriosis, listeriosis, salmonellosis and *Coxiella burnetii* infections. Hepatic necrosis is a characteristic feature of RVF and is distinguishable histologically. The other causes of abortion can be confirmed by demonstration of the causal organisms through bacteriological or other microbiological tests.

Treatment and Control

Treatment of the affected animals is usually not undertaken and such should be culled in order to reduce the sources of infection. Regular testing of animals, restriction of movement of animals and personnel between herds and purchase of animals with known health and reproductive records can prevent introduction and reduce the spread of the disease. Pasteurisation of milk is recommended in order to reduce incidence of the disease in man. All the infected materials should be incinerated and the contaminated premises disinfected. A test and slaughter policy can only be effective if it is preceded by a well organised educational programme to the livestock owners and assurance for compensation.

Vaccination with a live attenuated *B. melitensis* Rev 1 strain vaccine confers strong immunity but it causes abortion if used in pregnant does and ewes. It is recommended that kid and lambs should be vaccinated at 3-8 months while adults should be vaccinated 2 months before breeding. A formalin-killed adjuvant vaccine 53 H 38 has been in use in pregnant animals elsewhere.

FOOTROT

Footrot is a contagious infection of the feet characterised by inflammation of the skin-horn junction, under-running of the horn, ulceration and necrosis of the sensitive laminae of the foot and severe lameness. The disease is associated with production losses and sometimes mortality due to starvation.

Aetiology

Footrot in goats and sheep is caused by a large Gram-negative rod-shaped bacterium, *Bacteroides nodosus* which is commonly associated with *Treponema penortha*. *Fusobacterium necrophorum* and other aerobic or anaerobic bacteria may be isolated together with *B. nodosus* from the footrot lesions. Three serotypes A, B and C of *B. nodosus* exist. Serotypes A and B cross-react while serotype C is antigenically distinct.

Epidemiology

Footrot has been reported to be an important cause of morbidity in many countries. For example, the prevalence of footrot in goats in Kenya has been estimated to be 0.3 % while in northern Nigeria the prevalence of footrot in small ruminants has been estimated to be 4.9 %. In Tanzania, a 5.4-21.0 % prevalence of footrot has been reported in goats and sheep causing about 1.8-3.2 % mortality.

Footrot is a contagious infection and discharges or exudates from the affected feet contaminate the pasture or bedding. Infection occurs through contact with infected material and the organism gain entry into the body by penetration through broken skin. Prolonged wetting of the skin, scratches and bruises or surgical wounds facilitate the penetration of the bacteria and are therefore important predisposing factors. In Kenya, housing of goats in stony floors has been found to predispose goats to footrot. Penetration of nematode larvae such as *Bunostomum* spp and *Strongyloides* spp and trombiculid mites through the skin can also facilitate the entry of the causative bacteria.

Wet and warm weather conditions favour the proliferation of the bacteria and soften the animal's skin thus making it easily breakable and penetrable. Dry and hot conditions are unfavourable for proliferation of *B. nodosus* and transmission of footrot and hence, the incidence of footrot tend to rise during the rainy season and drop during the dry season. In Tanzania, it has been noted that footrot is an important disease in areas of high rainfall and relative humidity and in intensively managed herds. The intermingling and congregation of animals in communal grazing areas, poor floor types and poor disposal of urine and faeces favour the spread of the disease. Carrier animals may harbour the organism for 2-3 years.

Pathogenesis

B. nodosus produces a growth factor and extracellular proteolytic enzymes which facilitates its penetration, establishment and growth in the host tissues. The proliferation of the bacteria causes severe tissue destruction leading to interdigital dermatitis and suppuration.

Clinical features

Initially, there is a moist, swollen, hyperaemic and macerated interdigital skin and later on, a foul smelling discharge from the lesion is observed. Fever may or may not occur. Severe lameness occurs and the affected animals become recumbent. Affected animals may be seen to graze on their knees to relieve pain in affected fore feet. There is also reduced feed intake, weight gain and milk yield. Animals may die because of starvation.

Pathological features

There are no characteristic pathological features associated with footrot although grossly

there is always interdigital necrosis. There is almost always some under running of the horn of the wall and usually the sole of the affected claws. A characteristic black, foul smelling material is present due to the bacterial necrosis of the horn. Spread of the infection to joints may result in pyo-arthritis and accumulation of pus in the joint cavity. At histopathology, neutrophils are abundant in pus.

Diagnosis

Clinical signs are highly suggestive of the disease. The disease can be confirmed by demonstration of *B. nodosus* in pus smears and scrapings taken from the edge of the lesions. The smears can be stained by Gram's method or by dilute carol fuchsin. The bacteria stain faintly by Gram's method but in carol fuchsin, they appear as large Gram-negative rods with terminal enlargement at one or both ends. The bacteria can be isolated from pus by culturing on a *B. nodosus* specific medium containing Eugon agar base with 0.2 % yeast extract, 10 % defibrinated horse blood agar and 1 µg lincomycin. Colonies of pathogenic strains of *B. nodosus* appear as beaded or papillate while the less pathogenic strains produce mucoid colonies. Mouse or rabbit inoculation and fluorescent antibody tests are also used in the confirmation of the disease.

Footrot should be differentiated from other causes of lameness such as traumatic injury, necrobacillosis, dermatophilosis (strawberry footrot), bluetongue, parasitic dermatitis, arthritis, foot and mouth disease and vesicular stomatitis. The clinical signs of footrot and necrobacillosis (foot abscess) are very similar but in necrobacillosis the principal bacterial isolate is *F. necrophorum*. The characteristic signs of necrobacillosis also include swelling of the tissues of the pastern, and the development of one or more sinuses at the coronet. The infection often spreads to involve the inter-digital space. Mixed infections with other bacteria is, however, not uncommon. Strawberry footrot is a proliferating dermatitis caused by *Dermatophilus congolensis* and it is characterised by itching and, lesions extending from the coronet to the hock or knee joints. Apart from coronitis which may be accompanied by separation of the hoof, the presence of fever, salivation, severe erosions on the muzzle and buccal cavity can be used to distinguish bluetongue from footrot. Lameness is not a feature of parasitic dermatitis but a foul smelling discharge and separation of the hoof may be confused with footrot. In addition, the demonstration of larvae of *Strongyloides* spp, *Bunostomum* spp and trombiculid mites may be suggestive of parasitic dermatitis.

Treatment and Control

A single heavy dose of penicillin-streptomycin (containing 70,000 IU penicillin and 70 mg/kg streptomycin) given intramuscularly can be effective in the treatment of the disease. A follow-up treatment may be required if the response after the initial injection is not satisfactory. Chloramphenicol, tetracycline, erythromycin, tylosin, clindamycin, nitrofurazole parenteral and topical preparations can also be used in the treatment of the disease. Regular hoof trimming is recommended and has been found to facilitate recovery of the treated animals. Furthermore, hoof trimming can help to reduce the carrier state.

Control is based on the prevention of the spread of the bacteria, maintaining good hygienic conditions in the herds and minimisation of predisposing factors. Foot-baths containing 5 % copper sulphate, 10 % zinc sulphate and 5 - 10 % formalin are used in intensive production systems.

Although vaccines containing *B. nodosus* in an oil adjuvant or pili of *B. nodosus* cells and *Pseudomonas aeruginosa* in incomplete Freund's adjuvants are used in intensive production units, vaccination of small ruminants against footrot in the traditional small ruminant systems in sub-Saharan countries is not common because of the low mortality and seasonal incidence of the disease.

DERMATOPHILOSIS (STREPTOTHRICOSIS)

This is an acute, subacute or chronic and sometimes fatal exudative dermatitis of animals and less frequently man which is characterised by exudation, matting of the hair/wool and formation of crusts and thick scabs. The disease is caused by a dimorphic Gram positive bacterium, *Dermatophilus congolensis*.

Epidemiology

Dermatophilosis causes losses in terms of skin damage, reduced meat and milk production, culling or death of the affected animals and, costs of control and treatment. The disease is common among small ruminants in Nigeria, Ghana, Cameroon, Mali, Somalia, Kenya, Tanzania, Uganda, Malawi, Zimbabwe, Angola, Zaire and Madagascar. Fatal caprine dermatophilosis has been reported in Tanzania.

The source of infection is the sick or carrier animal and the disease spreads by contact. Prolonged wetting and mechanical damage to the skin either by bruises, scratches or surgical wounds are the predisposing factors. Arthropod vectors such as ticks (*Amblyomma* spp), flies (*Stomoxys* spp, *Glossina* spp and *Musca* spp), lice (*Linognathus* spp) and sheep ked (*Melophaga ovinus*) may be involved in the transmission of dermatophilosis. *Amblyomma* spp ticks seem to play the most important role in the transmission of the disease in the field. The incidence of the disease increases with increase in rainfall, humidity and insect activity and hence the prevalence of the disease tends to higher during the rainy season compared to the dry season. Grazing of animals in spiky vegetation types predisposes them to damage of the skin and thus facilitating the penetration of the organisms.

Pathogenesis

After penetration through the skin *D. congolensis* causes an exudative epidermitis. Secondary bacterial infection cause extensive suppuration of the lesions or toxæmia. The lesions begin with the production of a greasy exudate and crusts on the skin which later on turn into yellowish scabs. Tension of the skin caused by adherent scabs at flexion points results in fissures. The yellowish scabs then become hard, horny and confluent resulting into alopecia. Localised lesions are common but a generalised condition has also been observed.

Clinical features

Goats are more susceptible to dermatophilosis than sheep. Clinical signs include papular and scab formation on the muzzle, face, nose, ears, scrotum and feet. The under surface of the scabs is covered with a yellow, creamy or haemorrhagic and hair-matting exudate. Concurrent infection with the contagious ecthyma virus and stress factors such as malnutrition, pregnancy and lactation exacerbate the disease. The case fatality in

untreated goats is high although spontaneous recovery may occur.

In sheep, the lesions start on the dorsal parts of the body and spread laterally and ventrally. Lesions may also occur on the ears, neck, face, muzzle and outer sides of legs. *D. congolensis* may cause strawberry footrot which is a proliferative dermatitis characterised by development of small, raised and dome-shaped crusts on legs especially on the anterior aspect of the pastern. Coalescence of the lesions results in the formation of wart-like masses which may extend from the coronet to the hock or knee regions.

Pathological features

At necropsy, the disease is characterised by ulceration of the skin, extensive dermatitis and secondary bacterial pneumonia. Histopathologically, there is oedema, congestion and infiltration of the epidermis with neutrophils, vacuolation of skin cells and mononuclear cell infiltration. Occasionally, the bacteria may spread to the liver, kidneys and lymph nodes and cause hepatitis, nephritis and hyperplasia of lymph nodes.

Diagnosis

Epidemiological and clinical features are highly suggestive of the disease. The disease is confirmed by demonstration of Gram-positive mycelial organisms in impression smears made from the under surface of the scabs. The smears can be stained with 10 % Giemsa for 30 minutes or 1 % methylene blue for 30 seconds. The bacteria can be isolated by culturing suspensions of scab material which has been ground with sterile sand on blood agar containing 1,000 IU aerosporin per ml of medium. The cultures are incubated in a candle jar at 37 °C for 48-72 hours, after which whitish-yellow raised colonies with an irregular surface and clear zone of haemolysis are observed. The colonies are hard to lift from the medium. Serological methods of diagnosis include fluorescent antibody test, ELISA and counterimmunoelectrophoresis.

The differential diagnosis of dermatophilosis include mange, contagious ecthyma, fungal dermatitis, fleece rot and photosensitisation. The haemorrhagic or yellowish under surface which is evident when scabs are removed and the absence of itching differentiates dermatophilosis from mange. In addition, the mange mites can be demonstrated in skin scrapings. Contagious ecthyma can be differentiated by the presence of large, thick, greyish-black and tenacious scabs which may also distort the lips and muzzle. The causal virus can be demonstrated by virological and serological tests. Fleece rot, which is caused by *Pseudomonas aeruginosa* is characterised by formation of a mat of exudate on the wool which may also be stained green, red, yellow, brown, or blue following proliferation of chromogenic bacteria on the lesions. The restriction of scabs on unpigmented and hairless parts of skin and, a history of grazing on photosensitising plants will be highly suggestive of photosensitisation.

Treatment and Control

Heavy doses of penicillin-streptomycin (containing 70,000 IU/kg penicillin and 70 mg/kg streptomycin) are effective if administered in early stages of the disease. Heavy doses of long acting tetracyclines (20 mg/kg) may be used and a 2.5% chloramphenicol ointment may be applied topically. Cyclophosphamide (25 mg/kg) given orally has been found to be effective in the treatment of the disease in sheep.

Control of ticks and biting insects by dipping or spraying with insecticides may limit transmission of the disease. Zinc sulphate (0.5 %), copper sulphate or magnesium fluosilicate (0.2 %) solutions have been found to be effective in reducing the spread and incidence of the disease. Wherever possible injury of the animal's skin should be avoided.

CASEOUS LYMPHADENITIS

This is a chronic insidious disease affecting small ruminants and it is characterised by caseous abscesses in peripheral lymph nodes although the organism can spread and cause abscessation in other organs. The disease is caused by a Gram-positive facultative anaerobic and pleomorphic bacterium, *Corynebacterium pseudotuberculosis*. The economic importance of caseous lymphadenitis is related to the condemnation of the affected carcasses.

Epidemiology

Caseous lymphadenitis occurs among goat and sheep populations world-wide. The disease is documented to be an important cause of organ and carcass condemnation in goats and sheep in South Africa, Tanzania, Kenya, Ethiopia, Mali and Nigeria. The prevalence of caseous lymphadenitis in Kenya has been estimated to be 7% in goats and 2% in sheep, while a 50% morbidity of caseous lymphadenitis in goats has been reported in Nigeria.

Caseous lymphadenitis affects animals of all ages although it is commonly encountered in adult animals because of cumulative chances of getting infected rather than a true age-related susceptibility. Infection is mainly acquired by contact and, wounds or skin abrasions are the major portal of entry. Occasionally, the disease can be acquired by ingestion. Inhalation of infective material can lead to lung abscesses or pneumonia.

Wet skin can be easily macerated and thus enhance penetration of bacteria. The bacteria can survive in soils which are rich in organic matter or in formites at low temperature for along time. Pastures, animals shed and dips contaminated with pus discharges from ruptured or incised abscesses may be a source of infection. Biting insects or ticks can damage the skin and facilitate transmission. In addition, un-hygienic surgical procedures such as vaccination, ear marking, wool shearing and castration can spread the disease. The use of contaminated hypodermic needles was reported to be responsible for an outbreak of the disease in a goat herd in Nigeria.

Pathogenesis

After penetration through the skin *C. pseudotuberculosis* is carried via the lymphatic and blood vessels either as free or within macrophages to the regional lymph nodes or other parts of the body. The pathogenicity of *C. pseudotuberculosis* is related to its ability to produce a haemolysin and a toxic wall factor. It has been found that the haemolysin has a phospholipase activity and it acts on the sphingomyelin of the erythrocytes and endothelial cell membranes causing haemolysis and increased vascular permeability. This facilitates further invasion of the bacteria in the tissues. The toxic wall factor protects the bacterium from phagocytosis by lysosomes thus enabling it to survive within phagolysosomes. This is considered responsible for the chronicity of the lesions associated with *C. pseudotuberculosis*. The final outcome of the infection is determined by the initial number of bacteria entering the body of the host, the multiplication rate of the organisms and efficiency of the host

defence mechanisms.

Clinical features

The incubation period can extend from 3 weeks to 4-5 months. Caseous lymphadenitis is a mild disease characterised mainly by abscessation of the prescapular, parotid, submandibular and precrucial lymph nodes. Occasionally, abscessation may occur in the lungs, kidneys, spleen, heart, tongue, spinal cord, brain and joints. The general health of the animals is usually not affected although the presence of numerous active abscesses leads to progressive weight loss, weakness, collapse, coughing or respiratory distress. Other non-specific but rare signs may occur when the location of the abscesses interfere with the normal function of a particular organ or system. It has also been observed that toxæmia may occur in kids and lambs leading to arthritis and sometimes death.

Pathological features

The major gross pathological feature is the suppuration of the affected lymph nodes. The incised lymph nodes contain a thick greenish-white or yellowish-white inspissated or semifluid pus surrounded by a fibrous capsule. Inspissation may not occur in goats. On histopathology, there is a necrotic central area surrounded by neutrophils, giant cells, macrophages, plasma and epithelial cells. Gram-negative organisms can be demonstrated in smears made from the edge of the lesion. Infection of the lungs is associated with interstitial fibrosis.

Diagnosis

A provisional diagnosis of the disease can be based on clinical and pathological features. Confirmation of the disease is achieved by the demonstration of *C. pseudotuberculosis* in smears made from pus. In Gram-stained smears, the bacteria appear as pleomorphic Gram-positive rods. Pus cultured on sheep or ox blood agar for 24-48 hours at 37 °C produce small white and dry colonies surrounded by a narrow zone of haemolysis. The colonies become dry, crumbly and creamy in colour with time.

Other bacteria such as *S. aureus*, *C. pyogenes* and *Actinomyces pyogenes* which cause similar abscesses in or close to lymph nodes can be differentiated by isolation and characterisation of the bacteria. Other causes of chronic wasting such as chronic parasitism and malnutrition should also be considered in the differential diagnosis.

Treatment and Control

Treatment of affected animals is considered to be not economically justifiable because of the non-fatal and non-progressive nature of the disease. However, treatment may be needed for valuable stock such as breeding animals. *C. pseudotuberculosis* responds to penicillin although the perfusion of the drug through the capsule of the abscess is poor. Parenteral antibiotics may be used in severe cases. Surgical drainage of the affected lymph nodes is recommended.

The disease can be controlled by elimination of the source of the infection through culling of the affected animals. Surgical procedures such as castration, shearing or mass vaccination should be carried out under aseptic or hygienic conditions and, infected

premises should be disinfected. Vaccination of 2-3 month old kids and lambs is practised in some countries.

ANTHRAX

This is a peracute, acute or subacute and often fatal disease of animals and man and, in small ruminants it is characterised by septicaemia, splenomegaly and gelatinous infiltration of subcutaneous and subserosal tissues. The disease is caused by a large Gram-positive, spore-forming bacterium, *Bacillus anthracis*.

Epidemiology

Anthrax has been reported to affect goats and sheep across the sub-Saharan region and was associated with heavy mortalities in the 1960-70s. Outbreaks of the disease have been reported in Sierra Leone, Ghana, Chad, Ivory Coast, Nigeria, Chad, Uganda, Tanzania, Kenya, Botswana, Namibia and Republic of South Africa. Strict vaccination programmes have reduced the incidence of the disease in most countries in recent years. Nevertheless, sporadic cases are still being reported.

Spores are formed when the vegetative bacteria are exposed to atmospheric oxygen, suitable temperature (20-40 °C) and relative humidity (> 60 %). Spores may remain viable in the soil or water holes for many years. They may be dispersed by wind, predators, fertilisers or effluent from factories processing contaminated animal products. Animals are infected by ingestion of food, water or soil contaminated with spores. Infection may also occur by inhalation or through broken skin. Abrasion of the oral mucosa facilitates the penetration of bacteria. Mechanical transmission by biting insects has been reported. Movements of nomadic flocks of sheep and goats can introduce the disease to non-endemic areas. Outbreaks of the disease may occur following vaccination with inadequately attenuated vaccines. Wild animals can act as carriers of the disease and this makes it difficult to eliminate the disease in areas bordering national parks or game reserves because it not possible to control the movement of wild animals or institute effective vaccination programmes.

Pathogenesis

The pathogenicity of *B. anthracis* is related to the presence of the antigenic capsule and the ability of the organism to produce a leucocidal protein toxin which is antiphagocytic, increase vascular permeability, delays blood clotting and produces capillary thrombosis. Increased capillary permeability causes leakage of body fluids into tissues and body cavities causing oedema and haemonchocentration. Oedema of the lungs interferes with pulmonary perfusion leading to hypoxia, respiratory distress and inadequate supply of oxygen to the central nervous system. Leakage of body fluid into body tissues also results in decreased serum calcium and increased serum potassium leading to hyperirritability and convulsions which are observed in some animals. Presence of the toxin in the circulation causes severe anoxia, hypoglycaemia, alkalosis and shock which terminate into death.

Clinical features

The incubation period is 1-3 weeks. Peracute and acute forms of the disease occur in sheep and goats. The peracute disease is characterised by sudden death without

premonitory signs, although there may be fever, dyspnoea, muscle tremors, congestion of the mucosae and terminal convulsions in few animals. The course of the acute disease takes about 2 hours and it is initially characterised by severe depression and listlessness. Fever (42 °C), anorexia, laboured breathing, congested and haemorrhagic mucosae, increased heart rate, rumenal stasis and reduced milk production are common features. There may be blood discharges from the mouth, nostrils, anus and vulva. Diarrhoea or dysentery and oedema of the tongue, sternum, flanks and perineum have been observed. Pregnant animals abort and blood-stained or reddish-yellow milk is produced. Animals then collapse and die after terminal convulsions.

Pathological features

Post mortem examination of carcasses suspected to have died from anthrax is not recommended because of the risk of exposure of the vegetative organisms to air which triggers the formation of endospores and, hence contamination of the environment. There is also an additional occupational risk. The common gross *post mortem* features of anthrax in goats or sheep include complete absence of rigor mortis and rapid putrefaction and bloating of the cadaver. Non-clotting dark tarry blood oozes from the mouth, ears, nostrils, anus and vulva. The spleen is grossly enlarged with softening and sometimes liquefaction. Severe enteritis, ecchymotic haemorrhages throughout the body tissues and blood-stained fluid in body cavities are frequently observed.

Histopathologically there is widespread necrosis and haemorrhage in tissues and, capillary thrombosis. Large numbers of vegetative *B. anthracis* can be demonstrated in peripheral blood during the terminal stages of the disease.

Diagnosis

Clinical signs are highly suggestive. The disease can be confirmed by demonstration of large square-ended rods in thin blood smears prepared from the ear and tail veins or from the oedema fluid. Smears stained with 1 % polychrome methylene blue (McFadyean reaction) for two minutes reveal square-ended blue rods in chains surrounded by a pink capsule while those stained with 10 % Giemsa for 30 minutes show a red-mauve capsule. Spores can also be demonstrated by the Schaeffer and Fulton malachite green technique.

B. anthracis can be cultured from portions of the spleen and ear or blood by inoculating on sheep or ox blood agar. After aerobic incubation at 37 °C for 24-48 hours flat, dry greyish colonies with a granular 'ground glass' appearance are observed. At low magnification the edges of colonies show curved and curled projections giving rise to a 'medusa head' appearance. Intramuscularly inoculation of guinea pigs with 1 ml broth culture or oedema fluid leads to death in 24-48 hours with marked inflammatory reaction at the site of inoculation and extensive gelatinous oedema in subcutaneous tissues. Blood smears prepared from guinea pigs show typical capsulated organisms. Inoculation of mice also produces a fatal disease. The selective medium for *B. anthracis* is polymyxin-lysozyme-EDTA thallos (PLET) medium. The Ascoli test is also commonly used in the diagnosis of anthrax.

The differential diagnosis of anthrax include lightning stroke, acute bloat, peracute lead poisoning, peracute blackquarter and other clostridial infections. Lightning stroke is associated with singeing of the hair and, in addition there will be a history of an electrical

storm. Peracute blackquarter is mainly restricted to young animals and the crepitating swelling of the affected muscles is not observed in anthrax. Demonstration of *B. anthracis* in tissues of suspected affected animals will help to rule out acute bloat whereas, in acute lead poisoning nervous symptoms dominate.

Treatment and Control

Treatment of peracute cases is usually untimely because of sudden death. An anthrax antiserum may result in recovery if used in early stages of the disease. Oxytetracycline at a dose rate of 5 mg/kg body weight parenterally can be effective if used in early stages of the disease. Large doses of penicillin-streptomycin combinations at 12-hour interval given concurrently with the antiserum for 5 days have also been found to be effective.

Control of anthrax in endemic areas is achieved by annual vaccination with live attenuated vaccines or avirulent spore vaccines. Inadequately attenuated organisms may revert to virulent forms and cause a clinical disease. If an outbreak occurs, affected animals should be isolated and strict quarantine measures should be imposed and followed by vaccination of the unaffected animals. The infected premises should be disinfected using strong disinfectants such as 5 % sodium hydroxide or formalin. Clothes can be disinfected by soaking in 10 % formaldehyde and, where facilities are available hides and skin should be disinfected with gamma irradiation to avoid human infection. Carcasses should be buried in 2-metre deep pits and covered with quicklime to prevent spore formation.

BLACKQUARTER (BLACKLEG)

Blackquarter is an acute infectious disease of ruminants which is characterised by inflammation of muscles, severe toxæmia and high mortality. The disease is caused by *Clostridium chauvoei* which is a Gram-positive, spore-forming and rod-shaped bacterium.

Epidemiology

Blackquarter is associated with significant mortalities in goats in some sub-Saharan countries. The disease has been encountered in small ruminants in Nigeria, Mali, Chad, Cameroon, Uganda, Kenya, Tanzania, Zambia, Malawi, Botswana and Madagascar. In some countries, blackquarter occurs in the same zones as anthrax. Vaccination programmes have greatly reduced the incidence of the disease in East Africa although sporadic outbreaks are not uncommon.

C. chauvoei may be present in the liver, spleen and alimentary tract of apparently healthy animals and the clinical disease occurs when conditions in tissues become favourable for spore formation. Spores are resistant to heat and common disinfectants and can persist in soils which are rich in humus or in water holes for many years. The source of infection contaminate the soil, pasture and water reservoirs. Dead animals may be sources of spores to the environment. Animals are infected by ingestion of contaminated food or water. Spores may also enter the body through broken skin. Unhygienic mass vaccination or surgical procedures such as wool shearing or tail docking may spread the spores and result in outbreaks of the disease. Infection may occur through laceration wounds which occur in the genital tract during parturition. Sheep appear to be more susceptible than goats.

Pathogenesis

Ingested *C. chauvoei* spores pass through the intestinal wall and are carried through the lymphatic channels and blood circulation to muscles and other tissues where they lie dormant. When the muscles are bruised or necrotised the latent spores germinate and elaborate alpha, beta, gamma and delta toxins. The alpha toxin is a necrotising and lethal histotoxin which causes necrotising myositis and absorption of the toxin by muscles lead to toxæmia and death. The beta toxin destroys the nuclei of muscle cells. Exotoxins and other metabolites produced by the multiplying bacteria may cause lesions in the myocardium. Bacteraemia has also been found to develop terminally.

Clinical features

Affected animals exhibit stiff gait and hot painful swelling of the affected muscles. The muscles become oedematous and spongy. There may be crepitation but this not as marked as in cattle. Muscles of the shoulder, loin and buttocks are the most commonly involved. Serous or blood-stained fluid may ooze from the affected areas. Fever, lameness, severe depression are common features. The skin over the affected area becomes dark or black and, in later stages the swellings become cold and painless. Extensive local lesions can occur at the portal of entry.

Pathological features

The carcass rapidly putrefies and bloats. Sometimes, blood stained fluid may ooze from the nostrils and nose. There is excess fluid in body cavities which contain air bubbles, fibrin or blood. A blood-tinged or yellowish subcutaneous oedema fluid which may contain gas is a common feature. The incised affected muscles are dark-red or black with a characteristic rancid odour. Regional lymph nodes may be oedematous and haemorrhagic. Lesions tend to be deeper in sheep than in goats. The liver may decompose and produce gas.

Diagnosis

Clinical and pathological features can aid a tentative diagnosis of blackquarter. The disease is confirmed by the demonstration of large Gram-positive single rods or chains with oval, sub-terminal or central spores in smears made from the affected tissues or exudates. Smears should be made as soon after death as possible from aseptically removed pieces of the affected tissues such as muscles, subcutaneous tissue, liver, kidney and intestinal mucosa in order to avoid invasion with enteric facultative anaerobes such as *C. perfringens* and *Cl. septicum*. *C. chauvoei* is a strict anaerobe and difficult to culture. However, the bacteria can be cultured from affected muscles, liver or kidneys using *Cl. chauvoei* sheep blood agar medium incubated anaerobically at 37 °C for 24-48 hours. Colonies are small (1-2 mm), grey and rough. They are surrounded with a clear zone of haemolysis. Colonies of most clostridia resemble and can be differentiated by Gram stain or fluorescent antibody test. The latter test uses a fluorochrome-labelled *C. chauvoei* antiserum.

The differential diagnosis of blackquarter includes anthrax, lightning strike, snake bites, malignant oedema and other clostridial infections. Anthrax can be differentiated by its characteristic splenic lesion and the demonstration of the large square-ended bacilli in Giemsa-stained or polychrome methylene blue-stained smears (McFadyean reaction)

from the ear or tail veins. It is recommended to rule out anthrax by the McFadyean reaction before carrying out a *post mortem* examination of the suspected cases. A history of an electrical storm and singeing of the hair will help to differentiate blackquarter from lightning strike. Malignant oedema and other clostridial infections may be differentiated by the fluorescent antibody test.

Treatment and Control

In early cases of the disease large doses of penicillin (10,000 IU/kg body weight) given intravenously may result in recovery. Infiltration of the affected tissues with penicillin may also be effective in early stages of the disease. Long acting preparations of Penicillins are usually recommended. Annual vaccination using polyvalent clostridial vaccines is the main method of control of the disease in endemic areas. Vaccines derived from local strains of the bacterium are recommended. Combined blackquarter and anthrax or multi-component clostridial vaccines are commonly used in the field. Vaccination of pregnant ewes or does 2-4 weeks before parturition is useful in order to stimulate the production of antibodies that can passively protect the neonates. Quarantine measures can prevent spread of the disease in the event of an outbreak. Carcasses should be burned and buried in deep pits as for anthrax.

MALIGNANT OEDEMA (GAS GANGRENE)

This is an acute, febrile and fatal soil-borne wound infection of animals characterised by acute gangrenous inflammation at the site of infection, oedema and toxæmia. The disease is caused by clostridial organisms and *Clostridium septicum*, *C. chauvoei*, *C. perfringens*, *C. sordellii* and *C. norvyi* have all been isolated from the malignant oedema lesions. However, *C. septicum* is the most frequent isolate.

Epidemiology

Malignant disease is a sporadic disease which occurs world-wide. *C. septicum* occurs as normal flora in the intestinal tract of animals and faeces from such animals are a source of environmental contamination. The spores of *C. septicum* can persist in the soil and water reservoirs for a long time. All breeds and age groups of domestic animals are affected and infection occurs by contamination of wounds with spores of the bacteria. Deep puncture wound and severe trauma to tissues create anaerobic conditions which are favourable for the proliferation of the organisms. Contamination of wounds which may occur during castration, docking, wool shearing, vaccinations, intramuscular injections and dipping can result in outbreaks. The organism may also gain entry through the umbilical vessels of the new-born animals or lacerations in the genital tract which may occur during parturition.

Pathogenesis

Injury to the skin and mucous membranes facilitates penetration of the organisms into the body. Trauma of tissues is associated haemorrhage and effusions which create anaerobic conditions that are favourable for the proliferation of the bacteria. The bacteria multiply and produce an alpha toxin which is haemolytic, necrotising and lethal. The toxin causes necrosis, gangrenous inflammation and oedema at the site of infection. Absorption of the toxin into blood circulation is associated with toxæmia and shock.

Other toxins which help to amplify the pathogenic effects of the alpha toxin such as beta, gamma and delta are also produced.

Clinical features

Clinical signs may be observed as early as 12-48 hours or 4-5 days after infection. Initially there is a soft swelling, marked erythema and pain of the affected area. The swelling expands rapidly, becomes tense and the skin over it becomes dark. Emphysema and marked frothy exudation from the wound may occur, but it is not observed in *C. norvyi* infections. Fever (41-42 °C), weakness, depression, muscle stiffness and tremors and lameness occur. Death occurs within 24-48 hours after the onset of the clinical disease. In infection acquired through the genital tract clinical signs appear within 12-24 hours and they include swelling of the vulva, perineal region and pelvic tissues. A reddish-brown discharge from the vulva is also observed.

Pathological features

At necropsy, there is gangrene of the skin and oedema of the subcutaneous and intermuscular connective tissue around the site of infection. A serous or blood-stained gelatinous oedema fluid which contain gas accumulates at the lesion but in *C. norvyi* infection, the oedema fluid is clear or gelatinous but contains no gas. When muscles are involved they become congested, pale red or brownish. Haemorrhages are observed in the subserosal tissues and the body cavities contains a serosanguinous fluid. A foul putrid odour is common in *C. perfringens* and *C. sordellii* infection. The uterus becomes atonic, reddish in colour and emphysematous.

Diagnosis

The clinical and necropsy features are quite characteristic but the disease has to be confirmed by isolation of the causative bacteria in smears made from the affected tissues. Blocks of affected tissues for laboratory diagnosis should be collected and chilled as soon as possible after death because *post mortem* invasion of tissues with enteric clostridia may complicate the diagnosis. Malignant oedema should be differentiated from blackquarter, anthrax, snake bite and other histotoxic clostridial infections. The characteristic muscle involvement which is evident in blackquarter is not a frequent feature in malignant oedema. Exudation of dark tarry blood from natural orifice is highly suggestive of anthrax and, the latter can be confirmed by demonstration of capsulated organisms by the McFadyean reaction. Other clostridial organisms can be differentiated by immunofluorescence.

Treatment and Control

Injection of penicillin or cephalosporidine on the periphery of the lesion is commonly practised in suppress replication and production of toxins by the bacteria. Other broad spectrum antibiotics can also be used. Antibiotic therapy should be accompanied with surgical drainage and irrigation of the infected wound with hydrogen peroxide. The infection can be controlled by maintaining asepsis when performing surgical procedures on animals. Prevention of animals from other causes of wounds may reduce transmission and incidence of the disease. Infected premises should be properly disinfected. In endemic areas annual vaccination of animals with specific or polyvalent formalised bacterins is recommended. In high risk areas, animals may be protected from accidental contamination by vaccination prior to anticipated

surgical operations.

TETANUS

This is a highly fatal infectious disease of all domestic animals and man caused by a neurotoxin produced by *Clostridium tetani* and it is characterised clinically by hyperaesthesia, tetany and convulsions. *C. tetani* is a rod-shaped, spore forming Gram-positive bacterium in young cultures but becomes Gram-negative in old cultures.

Epidemiology

Tetanus is a sporadic disease which occurs world-wide. *C. tetani* is normal intestinal flora of mammals which are sources of environmental contamination. The bacterium form resistant spores which can persist in the faeces of herbivores or soil for many years. Spores enter the body through deep puncture wound and they normally lie dormant in tissues until conditions become favourable for the proliferation and production of the toxin. Outbreaks of the disease may occur following mass contamination of animals during vaccination, castration, docking, shearing or other surgical procedures. Grazing on rough and spiky pastures may traumatise the buccal mucosa and facilitate entry of the bacteria. The wound may heal and close leaving the dormant spores in tissues, and they may proliferate later on when conditions become favourable. Although the disease is primarily caused by toxins produced by organisms already in tissues, pre-formed toxins in feeds or toxins produced in the gut by ingested organisms may also cause a clinical disease.

Pathogenesis

Trauma and necrosis of tissues create anaerobic conditions which favours the proliferation of dormant *C. tetani* spores and production of a potent neurotoxin (tetanospasmin). The toxin travels from site of production to the central nervous system through the blood system or through the peripheral nerves. The presence of the tetanospasmin at the inhibitory synapses or motor neurones blocks the release of the gamma aminobutyric acid thus blocking the inhibitory neural impulses. As a result, there is constant potentiation of the sensory stimuli which leads to constant spasticity of muscles and hyperaesthesia. Tetanic spasms of the respiratory muscles cause asphyxia, cardiac arrest and death.

Clinical features

The incubation period is 1-3 weeks but may be longer depending on the pathogenicity of the strain, amount of toxin produced and amount of toxin entering the neural pathways or blood stream. The earliest signs include muscle stiffness, tremors and prolapse of the third eyelid. This is followed by trismus, unsteady gait and inability to move which is caused by stiffness of the limbs and abnormal flexion of joints. Tetany of the masseter muscles causes drooling of saliva from the mouth and regurgitation of food through the nostrils. There is also anxiety, dilatation of the nostrils, retraction of the eyelids and hyperaesthesia. Increased muscular activity may result in increased body temperature (up to 42 °C). Spasms of the alimentary and urinary tract muscles cause constipation and retention of urine. Abnormal muscular contractions may cause opisthotonus, curvature

of the spine and bending of the tail. Startled animals fall down with their fore and hind limbs stretched. The disease is highly fatal and death occurs 3-4 days after the onset of the clinical signs. However, spontaneous recovery may occur in animals which show a mild disease. A transient period of temporary improvement may occur before severe terminal spasms of the respiratory muscles.

Pathological features

No specific pathological lesions are associated with the disease except for the wound or traumatised tissue at the site of entry and toxin production. *C. tetani* may be cultured from such lesions.

Diagnosis

The muscular spasms and prolapse of the third eyelid are characteristic features of tetanus and a history of recent surgical procedures or trauma of tissues can be very supportive in the diagnosis of the disease. The disease can be confirmed by demonstration of the organisms in Gram-stained smears from the wounds in which they appear as Gram-positive single rods or chains with bulging round or spherical spores at the end giving a typical 'drumstick' appearance. On blood agar, small, slightly raised, feathery, semi-translucent or grey colonies which are surrounded with zone of haemolysis appear after 48 hours of incubation. Smears from young cultures reveal Gram-positive rods whereas, those from old cultures show Gram-negative organisms.

The differential diagnosis of tetanus include strychnine poisoning, plant poisoning, heartwater, enterotoxaemia of lambs and cerebral meningitis. The muscular spasms in strychnine poisoning are not as marked as in tetanus and, a history of exposure to strychnine or demonstration of *C. tetani* or the toxin tissues of the suspected animals will help to differentiate the two conditions. In heartwater, the nervous signs are less severe and fever is a frequent feature. Enterotoxaemia can be differentiated by isolation of the causative bacteria while cerebral meningitis is accompanied with depression.

Treatment and Control

A tetanus antitoxin is used to treat affected animals and is effective if given in early stages of the disease. Large doses of penicillin given parenterally or injected locally at the site of infection has been found to reduce further proliferation of the bacteria and toxin production. Local injection of antitoxin near the wound before debridement and irrigation with hydrogen peroxide is recommended to prevent spread of the toxin from the wound. Muscle relaxants such as acepromazine (0.05 mg/kg) should be given intramuscularly twice per day until the signs subside. Affected animals should be kept in a quiet environment and provided with enough space and soft bedding to avoid injury which may occur following muscle spasms. Intravenous or stomach tube feeding may be necessary.

Prevention of wound contamination with the *C. tetani* is the major principle of control of the disease. Surgical or other procedures which may be associated with trauma to the tissues should be carried out under strict hygienic conditions. A tetanus antitoxin should be given before mass surgical operations are carried out to prevent outbreaks should contamination of the wounds occur. In endemic areas, animals should vaccinated to prevent outbreaks. A toxoid produced from an alum precipitated, formalin-treated toxin is available commercially and it provides protection for one year beginning from 2 weeks

post vaccination. A booster vaccination given after 12 months provides life long immunity. Vaccination of ewes 8 weeks and then 2-3 weeks before parturition stimulates antibody production for passive protection of neonatal animals.

INFECTIOUS NECROTIC HEPATITIS

This is an acute septicaemic disease of animals especially sheep which is characterised by sudden death, hyperaesthesia, severe depression and sternal recumbency. It is caused pathogenic strains of *Clostridium norvyi* type B but the occurrence of a clinical disease is associated with necrosis of the liver tissue which makes the environment favourable for the proliferation of the bacterium and production of a lethal alpha toxin.

Epidemiology

C. norvi B is present in the intestinal tract of clinical normal animals which act as carriers. Faeces from carrier animals and cadavers of dead sheep are the source of infection. Faecal contamination by carrion animals is also a source of infection. Water and soils may be contaminated by spores carried away from other areas by floods. Wild animals and birds have also been reported to be involved in the spread of spores. Infection occurs mainly through ingestion of spores and all ages of sheep are affected although the incidence of the disease has been found to be higher in healthy mature sheep of 2-4 years old. Neonatal animals may be infected through the umbilicus and infection through the genital tract can also occur. The occurrence of the disease is related to the distribution of liver flukes (*Fasciola* spp). A seasonal incidence of occurrence of the disease which is related to the seasonal incidence of *Fasciola* spp and their snail hosts has been demonstrated. In communal grazing system, overstocking and the congregation of animals in watering points during the dry season can result in outbreaks of fasciolosis and infectious necrotic hepatitis. Irrigation creates favourable habitats for snail hosts of liver flukes and may be associated with high incidences of both fasciolosis and infectious necrotic hepatitis. Damage to the liver tissue caused by *Dicrocoelium dendriticum* and hepatotoxic chemicals can precipitate the occurrence of the disease.

The disease is said to occur world-wide although specific reports from sub-Saharan countries are lacking. However, the disease has been reported to affect sheep in Mali. It is quite possible that some cases of infectious necrotic hepatitis are misdiagnosed for fasciolosis.

Pathogenesis

After infection, the spores cross the intestinal barrier and are transported through the lymphatics and blood circulation to the liver and the spleen where they remain dormant. Damage of the liver tissue caused by migrating liver flukes create anaerobic conditions which activate latent spores. The bacteria multiply and elaborate alpha and beta toxins which are necrotising, lethal and haemolytic. The toxins cause necrosis of the hepatic tissue and the presence of toxins in blood circulation result in diffuse toxæmia.

Clinical features

Sudden death without evidence of premonitory signs may occur. Animals which survive sudden death become depressed, show disinclination to move and lag behind or separate from the rest of the flock. The neck and head may be extended and the back may

be arched. There is fever (40-42 °C) which fall to subnormal temperature prior to death. Rapid and shallow respiration, ruminal stasis and hyperaesthesia which is manifested by spasmodic twitching of the ears are common features. The animals then falls on sternal recumbency and dies without struggling.

Pathological features

Usually the animal is in good condition but rapid putrefaction occurs. Blood-stained froth at the mouth and nostrils may be observed. There is an extensive haemorrhagic subcutaneous oedema in the sternum, ventral abdomen and inguinal regions. Congestion and cyanosis of the subcutaneous tissue results in blackening of the skin and hence, the name black disease. Thickening and oedema of the abomasal wall and congestion of the duodenal mucosa may be evident. The liver becomes engorged and dark-brown with characteristic 1-4 cm yellowish necrotic areas which are surrounded by a bright red zone of congestion are seen especially under the capsule of the diaphragmatic lobes. These areas may be deeply seated and only evident after careful incision of the liver. Haemorrhagic tracts caused by migrating immature liver flukes are evident but adult flukes may be absent. The serous cavities contain a blood-stained or serous fluid which may lead to ascites, hydrothorax and hydropericardium. Subendocardial and subepicardial haemorrhages are evident and congestion of the parietal surface of the rumen, reticulum and omasum may occur. Large numbers of *C. norvi* B may be demonstrated in impression smears made from the liver sections.

The presence of a central zone of necrosis surrounded by a leucocytic zone containing mainly polymorphonuclear cells and occasionally lymphocytes in histological sections taken from the suspected liver is considered to be pathognomonic. A large number of vegetative or sporulating *C. norvi* are present within the leucocytic zone.

Diagnosis

Epidemiological, clinical and pathological features may be suggestive. The disease is confirmed by demonstration of *C. norvi* type B in impression smears made from the edges of the necrotic lesion. The bacteria may be cultured from pieces of the necrotic liver which have been aseptically removed from the carcass. Pieces of the liver tissue can be preserved in formalin for histological examination. The demonstration of organisms in typical lesions and demonstration of toxins in peritoneal fluid or in the liver is considered to be a positive diagnosis. The fluorescent antibody test is a rapid and simple method of diagnosis of infectious necrotic hepatitis. The clinical pathology of black disease is characterised by elevation of the liver enzymes especially gamma glutamyl dehydrogenase and, eosinophilia.

Infectious necrotic hepatitis has to be differentiated from fasciolosis. In the latter, the course of the disease is longer and the affected animals exhibit depression and anorexia. In fasciolosis, the liver is enlarged, friable and mottled and, immature flukes may be seen through the capsule together with subcapsular haemorrhages which they cause by perforation, but the necrotic foci characteristic of infectious necrotic hepatitis are absent. Other disease characterised by sudden death such as anthrax, blackquarter, malignant oedema, pulpy kidney and other clostridial enterotoxaemias can be differentiated from infectious necrotic hepatitis by identification of the causative bacteria in smears from the affected tissues using the fluorescent antibody test.

Treatment and Control

Sudden death often precludes timely and effective treatment although antibiotic therapy may be helpful in early stages of the disease. In endemic areas, vaccination of animals with an alum precipitated toxoid is commonly practised and may confer life long immunity. The use of multi-component vaccines with other clostridial species is recommended. Vaccination may reduce mortality during outbreaks. Control of fasciolosis and snail hosts can greatly reduce the incidence of the disease. Infected carcasses should be properly destroyed to avoid contamination of the environment with *C. norvyi* type B spores and infected premises should be disinfected.

ENTEROTOXAEMIA CAUSED BY CLOSTRIDIUM PERFRINGES TYPES B AND C.

Clostridium perfringens type B causes lamb dysentery and enterotoxaemia in goats. Few reports of the conditions are available in the sub-Saharan region although lamb dysentery is reported to be considerable losses in intensive sheep production units in South Africa. *C. perfringens* type C also causes acute enteritis in lambs.

Epidemiology

C. perfringens is found in the intestinal tracts of clinically normal or sick animals and pastures or soils are contaminated with faeces from such animals. Resistant spores are formed and can persist in the soil for months. Overcrowding and prolonged confinement have been found to be favourable for the spread and increased severity of the disease. Infection is acquired by ingestion of contaminated food or water. Lambs may also acquire the infection from contaminated udders or teats during suckling.

Pathogenesis

Following ingestion, the organism colonise and proliferate on the intestinal mucosa. *C. perfringens* type B producing alpha, beta and epsilon toxins whereas, *C. perfringens* type C produce alpha and beta toxins only. The beta toxin causes a severe haemorrhagic and ulceration of the intestinal mucosa. In adult animals the toxins produced are inactivated by trypsin but the neonate animals do not produce enough quantities of trypsin to destroy the beta toxin. Hence the disease is restricted to lambs under 1-2 weeks old. Necrosis and desquamation of the mucosa occurs resulting into ulcers. The irritation of the intestinal mucosa caused by presence of the organisms and toxins increases peristaltic movements and disturb the absorptive mechanisms on the mucosa resulting in loss of water and electrolytes from tissues into the gut. This is followed by dehydration, and acidosis. In addition, toxemia which is caused by presence of toxins in the circulation cause shock which progress into death.

Clinical signs

Peracute, acute, subacute and chronic syndromes may occur. The peracute disease is characterised by sudden death without any premonitory signs. The acute syndrome is characterised by depression, failure to suckle, severe abdominal pain, bleating and lagging behind the flock or recumbency. A brownish or bloody diarrhoea is also present. The lambs then become comatose and die within 24 hours from the onset of the disease. The

subacute syndrome is manifested by dullness, sluggish movements and abdominal pains. Tenesmus and a mucoid yellowish or blood-stained diarrhoea are observed. Death follows after prostration and coma. The chronic disease is mainly characterised by unthriftiness and a mucoid or blood-stained diarrhoea may be observed.

Pathological features

The acute and subacute cases are characterised by haemorrhagic enteritis, congestion and ulceration of the mucosa which becomes dark red. The intestinal contents are blood-stained and the peritoneal cavity contains excess serous or serosanguinous fluid. Adhesions of intestinal loops and presence of frank blood in the intestinal lumen have been reported. Perforation of the ulcerated intestine may occur resulting in peritoneal effusion, fibrinous peritonitis and adhesions. Subepicardial and subendocardial haemorrhages and, hepatic and renal degeneration may be evident. In chronic cases, there may be splenomegaly and clotted milk may be found in the abomasum. Large numbers of *C. perfringens* can be demonstrated in smears made from the affected portions of the intestine.

In adult sheep, *C. perfringens* type C causes a condition known as struck which is often characterised by sudden death although the affected animals may exhibit abdominal pain and convulsions. Ulcerative and haemorrhagic enteritis, fibrinous peritonitis, petechiae in serosal tissues and transudation in the peritoneal, pleural and pericardial are observed at *post mortem*. If the examination is delayed, rapid putrefaction of the carcass occurs and the disease may resemble malignant oedema.

Diagnosis

A provisional diagnosis can be made on the basis of clinical and pathological features but the syndromes caused by *C. perfringens* can be confirmed by demonstration of the bacteria in Gram-stained smears from affected portions of the small intestine. The presence of many vegetative Gram-positive bacilli in the smears can support the diagnosis. Isolation and typing of the bacteria and demonstration of the toxins in filtrates of the intestinal contents may be a positive diagnosis. ELISA is used to detect specific antitoxins in serum or beta toxins in the intestinal contents. Enterotoxaemia should be differentiated from salmonellosis and colibacillosis which also cause haemorrhagic enteritis in neonatal lambs or kids by demonstration of the causative organisms in intestinal contents or faeces. In addition, in colibacillosis ulceration of the mucosa is not a common feature.

A hyperimmune serum is effective in the treatment of the disease if given in early. Antibiotic therapy can be effective in treating mild cases of the disease. Penicillins, oxytetracyclines and chloramphenicol can be used. Vaccination of pregnant ewes 2 months and then 2 weeks before parturition using *C. perfringens* type B or C toxoid to stimulate antibody production for passive protection of neonatal lambs is recommended. A booster dose should be given annually. Cross-protection between *C. perfringens* type B and C bacterins has been reported to occur. Polyvalent vaccines are commercially available in South Africa. When outbreaks occur, sick animals should be isolated and treated while animals at risk should be protected using a specific immunoglobulin against *C. perfringens* type B. Maintenance of good hygiene in the herd can greatly reduce transmission of the disease.

PULPY KIDNEY DISEASE

This is a toxæmic of sheep caused by toxin produced by *Clostridium perfringens* type D in the intestines and it is characterised by diarrhoea, paralysis, convulsions or sudden death. Goats are less commonly affected.

Epidemiology

The disease occurs world-wide. *C. perfringens* type D is an obligate parasite of the intestinal tract but under certain conditions it proliferates and produce large quantities of toxins which can be lethal. Heavy grain diet or lush pastures have been found to be favourable for the proliferation of the bacteria and occurrence of the disease. Thus, the disease commonly affects well-fed animals especially in intensive feedlot units. Factors which result in intestinal stasis or slow the of passage of the ingesta through the intestines such as heavy tapeworm infestation favour the accumulation of the toxin and occurrence of the disease. Inclement weather, coccidiosis and deworming can predispose animals to the disease. Lambs and kids of 3-12 weeks and 6-12 months old have been found to be the most susceptible groups.

Pathogenesis

C. perfringens type D organisms are abundant in the ileum and less so in other parts of the small intestine. Sudden change from low to high energy and especially starchy diets favours rapid multiplication of the saccharolytic *C. perfringens* type D which produces an epsilon toxin. The toxin is endotheliotropic and binds to the endothelial cells causing damage. Damage to the capillary endothelium results in increased permeability of the intestinal mucosa and this facilitate further absorption of the epsilon and other toxins. The toxin also causes capillary damage in other tissues such as kidneys, lungs and the brain resulting in oedema. Extracellular oedema in the brain is associated with nervous signs.

Clinical signs

Sudden death is the principal manifestation of the peracute disease in young animals although some of the animals may be dull, depressed and anorexic. In acute cases, there is frothy salivation, green or pasty diarrhoea, staggering, recumbency, opisthotonus, colonic convulsions, coma and death. Colic and bloat may occur. Affected adult sheep often lag behind the rest of the flock and show nervous signs as in young animals which include hypersensitivity, staggering gait, ataxia and knuckling of the fetlock. Champing of the jaws, blindness, salivation, rapid and shallow respiration, atonic rumen and pasty faeces may also be evident. In goats, the acute disease is characterised by diarrhoea or dysentery, abdominal discomfort and convulsions. Death may occur within 24-36 hours of onset of the disease. In both sheep and goats, the subacute syndrome is characterised by anorexia, intermittent diarrhoea/dysentery or presence of epithelial shreds in faeces. The chronic disease is characterised by progressive weight loss, emaciation and anaemia but goats may also be diarrhoeic. The chronic nervous form of the disease is characterised by aimless wandering, incoordination, paralysis of the masseter muscles, inappetence and ruminal atony.

Pathological features

At necropsy, the carcass is in good condition and no gross changes are observed in the peracute disease. The acute syndrome is characterised by presence of a clear straw coloured fluid in the pericardial sac which rapidly clots on exposure to air, patchy congestion of the abomasal and intestinal mucosa and, presence of custard ingesta in the intestines. Diffuse petechial haemorrhages occur on the peritoneal surface of the abomasum and intestines. Petechial or ecchymotic haemorrhages also occur in the muscles of the flank, muscular portion of the diaphragm, epicardium and in the thymus. There is rapid decomposition of the carcass and purple discoloration of hairless areas of the body. The small intestine is distended with gas. A dark congested liver with haemorrhagic spots on its surface and gelatinous or blood-tinged pericardial fluid are observed a few hours after death. The kidney has a mottled appearance, soft consistency and the cortex is jelly-like or semi-fluid (pulpy kidney). Nephrosis, congestion of the renal cortex and rupture of capillaries occur. In young animals terminal rupture of the abomasum has been reported.

In goats, the acute disease is characterised by pulmonary oedema, necrosis of the small and large intestinal walls. The intestinal contents may be green, blood-stained or mucoid and, fibrinous casts or strands may present the lumen of the large intestine. The mesenteric lymph nodes are oedematous.

In histological sections of the brain, the presence of perivascular oedema, haemorrhages and bilateral symmetrical areas of leucoencephalomalacia in the basal ganglia, thalamus, substantia nigra and cerebellar peduncles is characteristic of the nervous form of the disease (also known as focal symmetrical encephalomalacia).

Diagnosis

The diagnosis of the disease is based on the epidemiological features especially the type of diet, clinical and pathological features. Gram-positive *C. perfringens* D rods can be demonstrated from smears of the ingesta or intestinal lesions. Isolation of the bacteria by culturing a sample of faecal material and demonstration of epsilon toxin in intestinal contents can be highly suggestive of the disease. Protection of mice injected with toxin filtrates from the ingesta using a specific antiserum is diagnostic. An agglutination test using specific antiserum for the epsilon toxin is also used to confirm the disease. Other diagnostic tests include ELISA, counterimmunofluorescence, passive haemagglutination and radial immunodiffusion.

The differential diagnosis of pulpy kidney disease in young animals include acute ruminal impaction, polioencephalomalacia, other clostridial enterotoxaemias and acute pasteurellosis. No convulsions are observed in acute ruminal impaction and the course of the disease is longer (1-3 days) and polioencephalomalacia takes a longer course. Acute pasteurellosis can be differentiated by isolation and identification of *Pasteurella* spp from the affected tissues. In adult animals rabies, acute lead poisoning and pregnancy toxaemia should be considered in the differential diagnosis of focal symmetrical encephalomalacia. In rabies, there will be a history of encounter with the rabid animal or bite wounds. The presence of materials containing lead such as paints can be suggestive of lead poisoning. Pregnancy toxaemia occurs during late pregnancy in under inadequate nutrition and ketonuria is common feature at clinical pathology.

Treatment and Control

A hyperimmune serum is used in the treatment of the disease. Combining the hyperimmune serum with sulphadimidine has been found to be effective in goats. Oxytetracycline is effective in treating subacute cases. Chelating agents may be used to neutralise the toxins. Outbreaks of the disease may be prevented by vaccination of animals prior to anticipated changes in diet. Vaccination accompanied with reduction of feed intake has proved to be effective in the control of the disease. An alum precipitated, formalin-killed whole culture toxoid is commercially available. Vaccination of animals using toxins prepared in Freund's adjuvant have been found to provide immunity for up to 2 years. Oral vitamin E preparations stimulate immune response in vaccinated animals. Lambs should be vaccinated at 3 days and 4 weeks old followed by re-vaccination at 6 months. Kids are first vaccinated twice at 4 weeks interval and then re-vaccinated at 6 months. Severe anaphylactic reactions have been reported in Saanen kids following re-vaccination with the toxoid. Vaccination using a multi-component clostridial toxoid may be beneficial.

BOTULISM

This is a highly fatal motor *paralysis* caused by ingestion of a neurotoxin produced by a Gram-positive, spore forming anaerobic bacterium, *Clostridium botulinum*. Four antigenically distinct types of *C. botulinum*, A, B, C and D may cause the disease.

Epidemiology

Although the distribution and economic importance of botulism in sub-Saharan Africa is not well documented, the wide distribution of the causative organisms and food scarcity which commonly occur during the dry season in most of these country merits its consideration. The vegetative form of *C. botulinum* is commonly found in the intestinal tract of herbivores and faeces from these animals contaminate the environment. The spores which are formed are highly resistant to environmental conditions. *C. botulinum* has been found to proliferate only in decomposing animal carcasses or sometimes plant materials. Carrion animals may spread the organisms from one area to another. Pica caused by deficiency of protein in the diet or starvation may force animals to crave on infected carrion or decomposing plant materials and subsequent ingestion of lethal doses of the toxin. Deficiency of phosphorus in the diet lead to osteophagia and if the bones are contaminated with *C. botulinum* toxin animals may ingest lethal doses of the toxin. Botulinum toxin from dead rodents, birds, chicken litter or inadequately sterilised fertilisers may contaminate water sources resulting in outbreak of the disease in animals using such water sources. Warm and wet conditions are favourable for the proliferation of *C. botulinum* and toxin production.

Pathogenesis

Proteolytic enzymes present in the alimentary tract digest the toxin and hence, ingestion of large doses of the toxin is required for a clinical disease to occur. Following ingestion, the toxin crosses the intestinal wall into systemic circulation. Presence of the toxin at the neuromuscular junctions inhibits the secretion of acetylcholine, the neuromuscular transmitter. This inhibition prevents transmission of impulses to the motor endplates resulting in flaccid paralysis. Death

is caused by asphyxiation following paralysis of the respiratory muscles. A toxoinfectious form of botulism caused the neurotoxin produced by organisms already present in body tissues has been reported.

Clinical features

In initial stages, the acute disease is characterised by stiffness of muscles, incoordination and excitability. The affected animal becomes listless and, the head is raised or lowered during walking or may be held on one side. Weakness of the neck muscles may result in torticollis. Lateral bending of the tail, arching of the back, salivation, serous nasal discharges and frequent urination also occur. Abdominal respiration and flaccid paralysis are observed in the terminal stages of the acute disease. The chronic disease is characterised by ruminal and intestinal stasis.

Pathological features

No specific pathological features are associated with the disease although congestion of the intestinal mucosa and serosa, subepicardial and subendocardial haemorrhages may be observed. Presence of foreign bodies such as bones, sand and pieces of wood in the stomach or intestinal contents; catarrhal enteritis; pulmonary oedema and excess pericardial fluid may be observed. Perivascular haemorrhage in the brain and destruction of the Purkinje cells may be evident in histological sections. At clinical pathology the toxin can be demonstrated in the liver.

Diagnosis

A provisional diagnosis can be based on epidemiological features such as starvation, pica or osteophagia and clinical signs. Demonstration of large quantities of the toxin in suspected feed or intestinal tracts of dead animals is diagnostic. Occurrence of the clinical disease in unvaccinated susceptible animals fed on suspected feed and absence of the disease in vaccinated animals is confirmatory. Botulism resembles the paralytic form of rabies, but a history of a bite wound caused by a rabid animal should be highly suggestive of rabies. Plant poisoning, lead poisoning, polioencephalomalacia hypocalcaemia, hypomagnesaemia and louping ill should also be differentiated on the basis of epidemiological features and demonstration of the aetiological agents.

Treatment and Control

A hyperimmune serum given together with the antitoxin for 5 days may result in recovery. Treatment is not effective in late stages of the disease. Good nursing and supportive intravenous or stomach tube feeding is recommended for animals which cannot feed on themselves. Animals should be put on sternal recumbency to prevent bloat. Control of the disease is achieved by removing the infected feed and correcting the dietary deficiencies which predispose animals to the disease. Proper disposal of carcasses to avoid contamination of the pastures and watering points is recommended and in high risk areas, annual vaccination of animals is useful. A polyvalent toxoid containing type A, B, C and D strains is commercially available.

COLIBACILLOSIS

This is a disease of new-born lambs and kids caused by pathogenic strains of *Escherichia coli* and characterised by septicaemia or enteritis depending on the strains of bacteria involved.

Epidemiology

The disease is common under intensive production systems. Faeces of infected animals are the main sources of infection. Sub-clinically infected animals act as reservoirs which contaminate bedding, pens and feed or water troughs making them sources of infection. People working with animals can transmit the infection between herds or pens. Stress factors such as cold, wet or windy weather; poor hygiene in animal houses and overcrowding predispose animals to the disease. Inadequate intake of immunoglobulins through colostrum immediately after birth has also been found to increase the susceptibility of animals to the septicaemic form of the disease (colisepticaemia). Outbreaks may occur when there is mass lambing or kidding.

The losses associated with colibacillosis in small ruminants in sub-Saharan Africa are not well documented. This may probably be because, with the exception of small ruminant intensive production units in few countries such as South Africa, Kenya and Zimbabwe the majority of sheep and goats in the region are kept extensively and hence, the incidence of the disease is low. However, with increasing population pressure and decreasing land available for grazing, intensive small ruminant production systems are being adopted especially in urban or peri-urban areas. This change of management systems may be associated with an increase in incidence of the disease.

Pathogenesis

The occurrence of clinical colibacillosis depends on the type and pathogenicity of the infecting strain of *E. coli*, host susceptibility and presence or absence of predisposing factors. Thus, two syndromes of the disease, that is, septicaemic (colisepticaemia) and enteric colibacillosis can occur.

Colisepticaemia is caused by invasive strains of *E. coli*. Invasion of tissues may occur through the intestinal lumen, the umbilical vessels, nasopharyngeal mucosa and tonsillar crypts. Posses adhesive pili, resistance to antibacterial activity of serum and production of endotoxin are the factors which enhance the pathogenicity of the invasive strains of *E. coli*. Presence of receptors for the pili in the epithelium of intestinal mucosa also facilitates establishment of the bacteria. After successful establishment on the mucosa the bacteria produce endotoxins which pass into systemic circulation and initiate bronchoconstriction, pulmonary hypertension and pulmonary oedema. Invasive bacteria also cause tissue damage.

Enteric colibacillosis is caused by enterotoxigenic strains of *E. coli* which are capable to colonise, proliferate and producing an enterotoxin in the upper small intestine. The bacterial fimbriae attach on the receptor sites on the villous epithelial cells and the bacteria multiply and colonise the villous surface. The colonisation of the villi and production of enterotoxins disrupt the absorptive mechanisms on the intestinal surface resulting in the secretion of fluids and

electrolytes from the systemic circulation into the intestinal lumen. This leads to electrolyte imbalance, dehydration, acidosis, hyperkalemia, circulatory failure and death.

Clinical and pathological features

Septicaemic colibacillosis is common in lambs and kids. Lambs and kids of 1-2 days and 3-8 weeks old have been found to be the most susceptible groups. The syndrome may be peracute in which case sudden death occurs without any premonitory signs. The acute disease is characterised by stiff gait or recumbency, depression, fever, hyperaesthesia and tetanic convulsions. Animals may collapse because of acute meningitis. The chronic form of the disease is characterised by polyarthritis. The bacteria may also cause local reactions in other tissues.

No gross pathological lesions are observed in peracute septicaemic colibacillosis because of sudden death. In the acute form there are widespread subserosal and submucosal petechial haemorrhages. Enteritis and gastritis are common features. Fibrinous exudates are found in joints and in serous cavities. Fibrinopurulent meningitis and peritonitis may also be encountered. Infection through the umbilicus is associated with omphalophlebitis.

Enteric colibacillosis is manifested mainly by a haemorrhagic or mucoid diarrhoea various degrees of diarrhoea and slight fever. Other enteropathogens such as rotaviruses, salmonellae and *Campylobacter* spp may also be involved and complicate the clinical picture. Pathologically there are widespread haemorrhages in the intestinal mucosa and large numbers of the bacteria can be demonstrated in smears from the intestinal mucosa.

Diagnosis

The epidemiology, clinical signs, pathological features and, response to treatment may support a presumptive diagnosis of colibacillosis. Confirmation is achieved by the isolation and characterisation of *E. coli* from suspected animals. Bacterial culture alone is of limited use because of the presence of non-pathogenic strains of *E. coli* and, demonstration of specific toxins may be of great value to support the diagnosis. In the peracute form of the disease the organisms may be isolated from abdominal viscera and heart blood.

The differential diagnosis of colisepticaemia include clostridial enterotoxaemia and salmonellosis. These conditions can be confirmed by isolation and identification of the causative bacteria. The differential diagnosis of enteric colibacillosis include dietetic diarrhoea, coccidiosis and campylobacteriosis. Dietetic diarrhoea is manifested by passage of voluminous and pasty or gelatinous faeces and the animals are usually bright or alert although they may be inappetent. Other enteritides can be differentiated by isolation and identification of their aetiologic agents

Treatment and Control

In view of the diversity of strains of *E. coli* which are involved in the syndrome it is important to carry out drug sensitivity testing before any treatment is instituted. Trimethoprim-sulphonamide combination (15-25 mg/kg) and kanamycin (20 mg/kg) given parenterally and colistin administered at a rate of 1-2 g/kg in drinking water have been found to be effective in the treatment of the disease. Other antibiotics such as oxytetracycline, neomycin, chloramphenicol and sulphadimidine are also used.

Vaccination of dams 2-4 weeks before parturition to stimulate production of specific antibodies is recommended in order to provide passive protection of neonatal lambs and kids through colostral immunoglobulins. Formalin-killed whole-cell vaccines are commercially available. Specific *E. coli* strain vaccines produced using K99+ pili antigens have been found to confer immunity to lambs and kids. Ewes have to be vaccinated twice in their first year of lambing, first at 8-10 weeks and then at 2-4 weeks before lambing. In subsequent years, one vaccination 2-4 weeks before parturition has been found to be satisfactory. Maintenance of good hygiene in the animal environment can reduce transmission and incidence of the disease. Provision of adequate colostrum to newly born kids and lambs will help to protect them from colisepticaemia.

SALMONELLOSIS

This is a disease of animals and man caused by different species of salmonellae and is characterised clinically by three major syndromes: peracute septicaemia, acute enteritis or chronic enteritis. *Salmonella dublin*, *S. typhimurium* and *S. anatum* are the common species associated with the disease in sheep and goats. *S. abortusovis* has been found to cause abortion in sheep.

Epidemiology

Salmonellae are widespread and the disease occurs world-wide. *Salmonella* spp are enteric bacteria and carrier animals shed the organisms in faeces thus contaminating the environment. It has been found that infection with *S. dublin* may result in a clinical disease or an active or passive carrier state. Active carriers constantly shed the organisms in faeces whereas, passive carrier will shed the organisms when stressed and, they may also manifest an overt disease. Recovered animals become subclinical carriers and shed the organisms in faeces. *S. typhimurium* may also originate from man or wild animals. Infection is acquired by ingestion of contaminated material. Animals may acquire the infection through food of animal origin and pastures contaminated with infective slurry or improperly treated fertilisers. Watering points may be contaminated with slurry from infected herds or fertilisers. Intensification of animal management favours spread of the disease from carrier animals. The organisms may be introduced in the herd via contaminated feed stuffs, formites, birds or nematodes. Stresses such as transport, starvation, parturition, overcrowding in communal grazing land, holding yards and dips activate latent infections and favour rapid spread of the disease. Disruption of the intestinal flora by factors such as antibiotic therapy, change of diet and water deprivation increases the susceptibility of the host to infection. Infection in animals occurs mainly by ingestion but in sheep it has been shown that infection may also be acquired by inhalation of infective material. Animal salmonellosis is the principal reservoir for human

salmonellosis.

Pathogenesis

The ability of *Salmonella* spp to produce disease is facilitated by the presence of virulence factors. It has been found that pathogenic salmonellae possess adhesive pili, protective plasmids and, produce an enterotoxin, cytotoxin and lipopolysaccharide. These act together and enable the bacteria to adhere and colonise the intestinal epithelium, survive the phagocytic activity of macrophages and increase the permeability of the intestinal epithelium. The presence of bacteria on the intestinal wall also initiates an inflammatory response. After successful establishment, colonisation and disruption of the integrity of the intestinal wall, the organisms pass through lymphatic system to mesenteric lymph nodes after which a clinical disease may occur depending on the virulence of the organisms, immune status and age of the host and, presence of intercurrent infections or other stress factors. From the mesenteric lymph nodes, the organisms invade the reticuloendothelial cells and then enter the blood stream causing septicaemia, enteritis and localisation in various tissues. Invasion of bacteria in the uterus and placenta causes abortion.

Clinical features

Enteric salmonellosis is the commonest form of the syndrome encountered in sheep and goats. The incubation period is 1-4 weeks. The syndrome is characterised by fever (40-41°C), anorexia, listlessness, severe diarrhoea and sometimes dysentery or tenesmus. Faeces have a putrid smell, mucoid and may contain blood clots or fibrin casts. Fibrin sheets may be found in the intestinal mucosa. Shallow and fast respiration, rapid pulse and congestion of the mucosae are observed. Abortion is a common feature. There may also be dehydration, toxæmia, loss of weight, prostration, recumbency and death. New-born animals that survive the septicaemic disease develop severe enteritis characterised by diarrhoea. Polyarthrititis and pneumonia are a common sequel. Does and ewes often die after abortion and lambs born alive may die subsequently. *S. dublin* is most common cause of the disease in goats. *S. typhimurium* also causes peracute septicaemia or acute enteritis.

Pathological features

Acute enteritis is characterised by muco-haemorrhagic enteritis and submucosal petechiation. In *S. typhimurium* infection, there is necrotic enteritis in the ileum and large intestines. The intestinal contents are putrid, mucoid, blood-tinged or may contain frank blood. The intestinal mucosa may be covered by an extensive diphtheritic membrane. The mesenteric lymph nodes are enlarged, oedematous and haemorrhagic. There is also enlargement and fatty degeneration of the liver; thickening of the gall bladder wall and presence of blood-stained fluid in the serous cavities.

The histopathological picture is characterised by necrosis, oedema, congestion and infiltration of the lamina propria and submucosa of the caecum, colon and small intestine with neutrophils, lymphocytes, plasma cells and macrophages. Focal necrosis in the mesenteric lymph nodes and thrombosis of the submucosa vessels occur. Hepatocellular necrosis and neutrophilic and mononuclear cell infiltration in the portal tracts may be evident. Necrosis and neutrophilic infiltration in the

mesenteric lymph nodes and lymphoid and reticuloendothelial hyperplasia occur in protracted cases.

Diagnosis

A provisional diagnosis can be based on the epidemiological, clinical and pathological features and the disease can be confirmed by bacterial isolation and serotyping. In the acute disease the bacteria are present in heart blood, spleen, liver, bile, mesenteric lymph nodes and intestinal contents while in chronic cases, the bacteria can be isolated from the intestinal lesions or other viscera. Lymph nodes which drain the caecum and lower intestine have been found to be rich in the bacteria. The organisms can be easily demonstrated in a thick smear made from the wall of the gall bladder. Selective media such as MacConkey agar, brilliant green agar, triple sugar iron agar and xylose-lysine deoxycholate medium are used in the isolation of *Salmonella* spp. Species-specific antibodies may be used to diagnose the disease but cross-reaction do occur.

Coccidiosis, campylobacteriosis and parasitic gastroenteritis should be considered in the differential diagnosis of salmonellosis. Unlike the above conditions, salmonellosis is often manifested by a more acute and often fatal enteritis. High faecal oocyst and demonstration of developmental stages of *Eimeria* on the intestinal wall may be highly suggestive of coccidiosis whereas, high faecal egg and worm burdens may be highly suggestive of parasitic gastroenteritis. These features are not observed in salmonellosis except when they occur as intercurrent infections. Campylobacteriosis can be differentiated by demonstration of *Campylobacter* spp in faeces.

Salmonellosis can be treated using chloramphenicol (20 mg/kg) infused intravenously at 6 hours interval for 3 days. Other drugs include trimethoprim-sulphadoxine combination, sulphadimidine, framomycin, ampicillin and amoxicillin. Oral nitrofurazone daily for 5 days mixed in the feed or as a drench is commonly used in mass medication during outbreaks. The recommended treatment regime is to combine oral and parenteral therapy. It is important to remember that oral antimicrobial therapy may disrupt the normal intestinal flora and increase host susceptibility to the disease. Supportive fluid therapy to alleviate the effects of dehydration and electrolyte loss is beneficial. In some countries, treatment of animals against salmonellosis has led to selection for drug resistant strains thus complicating the effectiveness of treatment of human cases of the disease. Salmonellosis can be controlled by avoiding faecal contamination of feed or water and maintaining good hygiene in the animal houses. Animals should be purchased from herds which are known to be free from the disease. Regular testing should be carried out to identify carriers which should be culled. Infected premises should be properly disinfected and the infective materials should be destroyed. Personnel from infected herds should not be allowed to come into contact with disease-free animals. Vaccination of small ruminants against salmonellosis is not widely practised.

MASTITIS

Mastitis refers to the inflammation of the mammary gland and is characterised enlargement of the udder and abnormal milk secretion with or without fever. The disease has a multiple aetiology but *Staphylococcus aureus* and *Streptococcus agalactiae* are

the commonest bacteria isolated from cases of mastitis in small ruminants. Other bacteria encountered include *Corynebacterium pyogenes*, *Klebsiella* spp, *Mycobacterium* spp and *Brucella* spp.

Epidemiology

Reports on clinical mastitis in small ruminants are available from South Africa, Kenya and Nigeria and, information from other countries is limited. This lack of information is probably related to the fact that the indigenous small ruminants are kept primarily for meat and, hence little attention has been paid to the economic significance of mastitis. However, with recent introduction of dairy goats and intensification of management systems, mastitis may become an important disease entity worth attention.

Un-hygienic conditions in animal houses and poor milking hygiene are important predisposing factors. Mechanical or surgical wounds in the teats or udder facilitate penetration of the bacteria. Most often, *Mycobacterium* spp and *Brucella* spp spread systemically and lodge in the mammary tissue causing mastitis.

Pathogenesis

After entry through the teat canal the bacteria colonise and multiply in the mammary tissue. Some bacteria produce enzymes and toxins which cause inflammation and damage to the mammary tissue, Pyogenic bacteria cause abscessation and suppuration. These inflammatory changes are associated with abnormalities in milk. The severity of infection is determined by the virulence of the organism, extent of mammary tissue damage, stage of lactation and efficiency of host defence mechanisms in the mammary tissue.

Clinical features

The clinical signs of acute staphylococcal mastitis in goats include restlessness, elevated pulse (up to 144 per minute) and respiratory rate (up to 80 per minute) rates, hot, painful and enlarged mammary glands. Gangrenous necrosis of the mammary tissue may occur. On palpation there is marked diffuse induration of the mammary glands and enlargement of the supramammary lymph nodes. The milk shows a thick yellowish discoloration or may be blood-stained.

Pathological features

The affected mammary glands are enlarged and hard on palpation. The teat orifices may be blocked. Abscesses may be present in different sections of the mammary tissue.

Diagnosis

A tentative diagnosis is based on the clinical signs especially presence of abnormalities in milk and pathological lesions. Confirmation is achieved by isolation or demonstration of the causative agents in smears prepared from pus or milk secretions. Cell counts and California mastitis test are also used in the diagnosis. Mastitis in goats' may also be caused by *Mycoplasma agalactiae* and *Candida albicans* and these should be considered in the differential diagnosis of the disease.

Other mammary abnormalities such as oedema, passive congestion and haematomata are usually not associated with abnormalities in milk.

Treatment and Control

Bacterial mastitis can be treated by penicillin, streptomycin, oxytetracycline and gentamycin either as intramammary infusions or parenterally in systemic cases. Combination of systemic and intramammary antibiotic therapy is beneficial where there is systemic involvement. Some strains of *S. aureus* are resistant to Penicillins, hence drug sensitivity testing is recommended before the use of these drugs in the treatment of mastitis. Proper herd and milking hygiene is the most effective means of controlling mastitis.

Other bacteria such as *Mycobacterium* spp, *Listeria* spp, *Actinobacillus* spp and *Actinomyces* spp cause disease syndromes in small ruminants, the clinical and pathological features are similar to those observed in cattle.

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CHAPTER 4 DISEASES CAUSED BY MYCOPLASMA

The major syndrome associated with *Mycoplasma* spp infection in small ruminants is pneumonia. However, *Mycoplasma* spp do cause other disease syndromes such as polyarthritis and mastitis.

PNEUMONIA

Many species of *Mycoplasma* are involved in the pathogenesis of pneumonic syndromes in small ruminants. The common species include *Mycoplasma mycoides* subspecies *mycoides* (large colony type), *Mycoplasma mycoides* subspecies *capri*, *Mycoplasma arginini*, *Mycoplasma ovipneumoniae*, *Mycoplasma agalactiae* and *Mycoplasma capricolum*. Contagious caprine pleuropneumonia (CCPP) is a distinct disease entity caused by *Mycoplasma* biotype F 38. *Mycoplasma mycoides* subspecies *capri* is also still being considered as a cause of a mild form of CCPP.

Epidemiology

The sero-prevalence of *Mycoplasma* spp in goats in south-eastern Nigeria has been found to be 92 % and *M. ovipneumoniae*, *M. agalactiae* and *M. arginini* have been isolated from pneumonic lungs in goats and sheep in different parts of Nigeria. *M. arginini*, *M. mycoides* subspecies *capri*, *M. ovipneumoniae* and *Mycoplasma* F strain 38 have been isolated from pneumonic lungs of small ruminants in the Sudan. *Mycoplasma* F strain 38 and *M. mycoides* subspecies *capri* have been isolated from goats in Kenya and Ethiopia while *M. mycoides* subspecies *mycoides* (large colony type), *M. mycoides* subspecies *capri*, *M. ovipneumoniae*, *M. capricolum*, *M. arginini* and *M. agalactiae* have been isolated from pneumonic lungs of goats in Tanzania.

Mycoplasma spp are labile organisms which are easily destroyed by heat, dehydration, sunlight and common disinfectants and therefore they do not survive for a long time outside the body of the animal. Sources of the infection are the clinically sick and carrier animals and infection occurs by inhalation of infective aerosols. Overcrowding in animal sheds, communal grazing land, water points, markets and dips facilitates rapid spread of the infection within and between herds. Stress factors such as heavy rainfall, cold weather and malnutrition which compromise the immune system of the animal can activate latent infection in carrier animals which start to shed the organisms. A long term carrier state may occur. Nomadic practices facilitate the spread of the disease, as so is stock raiding which is common in East African countries.

Clinical features

The pathogenesis of mycoplasmal pneumonia is similar to pneumonic pasteurellosis, as so are the clinical signs and pathological features. The incubation period may be as short as 3-6 days or as long as 3-4 weeks in natural field infections. CCPP is a highly contagious disease of goats and it is characterised by depression, fever (41.6-41.7°C), anorexia, dyspnoea, coughing, abdominal respiration, reluctance to move, bleating, extension of the

neck, frothy or mucopurulent nasal discharges and subcutaneous oedema on the chest and abdomen. Stringy salivation may be observed before death. Death may occur within 2 days of the onset of the clinical signs. Morbidity may reach 100 % and mortality ranges from 60% to 100%. Similar features are manifested by acute pleuropneumonia caused by other *Mycoplasma* spp. *Mycoplasma* biotype F 38 does not cause disease in sheep or cattle. In endemic areas animals may suffer a chronic disease which is characterised by catarrhal nasal discharges, debility, emaciation and sometimes enteritis. Recovered animals serve as carriers. The syndrome produced by *M. mycoides capri* causes a less contagious septicaemic disease characterised by inappetence, respiratory distress, low morbidity and mortality rates. The disease may be acute or subacute and may involve the respiratory, alimentary and reproductive systems.

Pathological features

In early stages, acute CCPP is characterised by formation of yellow nodules and congestion around the nodules. Congestion, consolidation and marbling of the lungs together with fibrinous pleurisy, pleural effusion and distension of the interlobular septae are prominent features. The chronic disease is characterised by thickening of visceral pleura and fibrinous adhesion to the chest wall. No sequestrae are formed as in the case of contagious bovine pleuropneumonia. Histopathologically, *Mycoplasma* F 38 infection is characterised by intralobular oedema while *M. mycoides* subspecies *capri* is characterised by interlobular oedema.

Diagnosis

A provisional diagnosis of CCPP can be based on the epidemiology, clinical and pathological features. The causative organisms can be demonstrated in impression smears or cultures of materials from lung lesions, mediastinal lymph nodes and pleural fluid on *Mycoplasma* media such as Modified Hayflick and Newings's tryptose B. Serological methods of diagnosis include complement fixation, ELISA and agar gel immunodiffusion. Latex agglutination test is very useful in field diagnosis of CCPP. Monoclonal antibodies are also employed in the diagnosis of CCPP. CCPP has to be differentiated from pneumonia caused by *M. mycoides* subspecies *mycoides* (larger colony type), *M. mycoides* subspecies *capri*, *M. capricolum* and pneumonic pasteurellosis. Other diseases to be considered in the differential diagnosis of CCPP include pasteurellosis, verminous pneumonia, PPR and other viral pneumonias.

Treatment and Control

Tylosin (11 mg/kg) is effective in the treatment of CCPP when used in early stages of the disease. Oxytetracycline (15 mg/kg), tiamulin, chloramphenicol and penicillin-streptomycin can also be used. Separation and treatment of the affected animals will minimise the spread of the disease. Susceptible naive animals should be vaccinated before being moved to endemic areas. A saponified inactivated *Mycoplasma* strain F 38 vaccine has been found to be effective in the control of the disease.

CONTAGIOUS AGALACTIAE

M. agalactiae is a cause of contagious agalactia of goats and sheep which is characterised by sudden onset of severe mastitis, arthritis, ophthalmitis and abortion. The

organisms may be cultured from milk and may be demonstrated serologically using complement fixation test and ELISA. The distribution and economic importance of contagious agalactia in goats and sheep in the sub-Saharan region is not well documented.

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CHAPTER 5 DISEASES CAUSED BY VIRUSES

The common viral infections of goats and sheep in sub-Saharan Africa are peste des petits ruminants (PPR), contagious ecthyma, goat/sheep pox and Nairobi sheep disease. Other viral diseases are Rift Valley fever and blue tongue.

Peste Des Petits Ruminants

This is a contagious viral disease of goats and less commonly sheep and it is characterised by fever, erosive stomatitis, enteritis, pneumonia and death. The disease is caused by a PPR virus of the genus *Morbillivirus* and family Paramyxoviridae which closely resembles the rinderpest virus.

Epidemiology

PPR is endemic and is considered to be the most serious infection of small ruminants in West and Central Africa. The disease has recently been encountered in small ruminants in the eastern part of the continent (Ethiopia). An infection rate of 22.0-51.0 % has been estimated in Nigeria and heavy mortalities associated with the disease have been reported. No sero-evidence of PPR so far has been reported in Africa south of the Equator, however, uncontrolled movement of livestock between countries is a potential danger to the spread of the disease.

The source of infection is the sick or sub-clinically infected animal and the virus is discharged in milk, saliva, urine or faeces. The disease spreads primarily by inhalation but the virus can also be acquired by ingestion and penetration through the conjunctival mucosa. Animals may acquire the infection by licking or muzzling each other. Bedding, feed and water troughs can be sources of infection. Goats are highly susceptible to PPR compared to sheep, and kids under one year are most susceptible. In West Africa, the dwarf breeds of goats have been found to be more susceptible than the Sahelian breeds. Presence of other diseases and other stress factors precipitate the occurrence of the disease. Severe outbreaks can occur when naive animals are moved into an endemic area. In West Africa, the incidence of PPR is reported to increase during the rainy season and during the cool harmattan winds.

Pathogenesis

After infection, the PPR virus multiplies in the regional lymph nodes which is followed by viraemia. The virus then infects and causes cytopathic changes in epithelial cells which are associated with the clinical signs and lesions observed.

Clinical features

Acute and subacute forms of PPR occur. The incubation period of acute PPR is about 7 days and the syndrome is characterised by severe depression, pyrexia, sneezing, dyspnoea, coughing, serous or mucopurulent ocular-nasal discharges which lead to matting of eyelids and blockage of nostrils. There may be crackling lung sounds which are clearly audible. Focal necrotic stomatitis, halitosis, anorexia, profuse mucoid diarrhoea or

dysentery and sometimes tenesmus are prominent features. Abortion occurs and there may be superficial erosions on vulva or prepuce. Death of severely affected animals occur in about 7 days after the onset of clinical signs. Mortalities of 71-100 % are not uncommon.

Subacute PPR is the commonest form of the disease in sheep. Clinically, it resembles the acute syndrome of goats except that clinical signs are milder. Intercurrent infections with contagious ecthyma is common and secondary bacterial infection often aggravates the severity of PPR. Solid immunity develops in recovered animals and passive maternal immunity protects kids under 4 months.

Pathological features

The gross pathological picture include an emaciated and dehydrated carcass, soiled hind quarters, matted eyelids and nostrils are blocked with exudate. Focal necrotic lesions in the oral mucosa, pharynx, upper oesophagus, abomasum and small intestines are evident. Zebra striping of the large intestinal mucosa, oedema and congestion of mesenteric lymph nodes occur. The nasal cavity and larynx are filled with mucopurulent exudate and presence of froth in the trachea and pulmonary oedema are common features. Secondary bacterial infection results in bronchitis, tracheitis, atelectasis and interstitial pneumonia.

The histopathological picture includes lymphocyte depletion in lymphoid tissues, picnosis and karyorrhexis of lymphocytes in the cortex of lymph nodes. Multinucleated giant cells, eosinophilic intracytoplasmic and intranuclear inclusion bodies can be demonstrated in epithelial cells which also show hydropic degeneration and necrosis. The lamina propria of the small intestine is infiltrated with lymphocytes, macrophages and eosinophils. There is also necrosis of the epithelium of the abomasal and intestinal glands, villous atrophy and accumulation of necrotic debris in glandular pits. The alveoli and terminal bronchi are infiltrated with giant cells containing intracytoplasmic and intranuclear inclusion bodies. There may be hepatic necrosis and glomerulonephritis. The clinical pathology of PPR is dominated by leucopenia and lymphopenia.

Diagnosis

A tentative diagnosis of PPR is based on the epidemiology, clinical and pathological features. The virus in swabs from tissue excretions, buffy coat or tissue suspensions can be isolated by inoculation in primary kid or goat kidney cells. Chilled pieces of the spleen, lungs, lymph nodes and gut mucosa can transported to the laboratory for virus isolation. Inoculation of tissue suspensions from affected or suspected cases in susceptible animals elicits a severe disease in goats, a mild disease in sheep and a subclinical disease in cattle.

The virus neutralisation test, ELISA, complement fixation test, immune electrophoresis, monoclonal and polyclonal antibodies detection are the common serological methods employed in the diagnosis of PPR. Serum neutralisation test and DNA probes are used to differentiate PPR from rinderpest. Agar gel immunodiffusion, counterimmunodiffusion and DOT-ELISA are used to demonstrate virus antigens from ocular and nasal discharges, pharynx, faeces and lymphoid tissues.

The differential diagnosis of PPR include rinderpest, contagious ecthyma, goat/sheep pox,

Nairobi sheep disease, blue tongue, CCPP, pneumonic pasteurellosis, salmonellosis, colibacillosis and parasitic gastro-enteritis. PPR and rinderpest are differentiated by serum neutralisation test. In contagious ecthyma, lesions in the alimentary tract are not common except in secondary infections. Nairobi sheep disease is not very severe in goats and no oral lesions are observed. Furthermore, NSD is restricted to areas where the vector tick, *R. appendiculatus* is found. CCPP is primarily a disease of the respiratory system affecting goats and no mucosa lesions or diarrhoea are observed in uncomplicated cases. FMD and bluetongue can be differentiated by close examination of the feet lesions and diarrhoea is not a feature of these diseases. Salmonellosis and colibacillosis can be differentiated by isolation of the causative bacteria whereas, in parasitic gastroenteritis, demonstration of high egg or worm burdens precludes PPR.

Control

Treatment of secondary bacterial pneumonia using broad spectrum antibiotics may be effective in the early stages of the disease. A hyperimmune serum produced from cattle hyperimmunised against rinderpest can be used. Fluid therapy is recommended to alleviate the effects of diarrhoea/dysentery. Good nursing of the sick animals may facilitate recovery. Control of the disease can be achieved by preventing the contact between susceptible animals and clinically or sub-clinically affected ones. Affected animals should be isolated and infected premises disinfected. If an outbreak occurs, quarantine measures should be imposed and supported by vaccination of the population at risk.

Vaccination using tissue culture rinderpest vaccine starting at 3-4 months of age is the commonest method of control of PPR in endemic areas. Although the PPR homologous vaccine and attenuated tissue culture vaccines are not widely used they are showing promising results.

RINDERPEST

This is a disease of ruminants characterised by fever, erosive stomatitis, severe diarrhoea or dysentery, dehydration and death. It is caused by a rinderpest virus of the genus *Morbillivinis* and family Paramyxoviridae which closely resembles the PPR virus. The epidemiology and pathogenesis of rinderpest in sheep and goats is similar to that of PPR. Small ruminants may suffer an inapparent, subacute or acute form of rinderpest. However, the mild form is more common in field infections. The acute disease is characterised by fever, depression, inappetence, arched back, serous nasal and lacrimal discharges which may become mucopurulent and may block the nostrils or mat the eyelids. In late stage, a foetid diarrhoea may occur. Death may occur in 6-7 days after the onset of the clinical disease although recovery may occur within 2 weeks. The subacute disease is mainly characterised by transient fever.

Congestion of the crest of the longitudinal folds in the rectal mucosa result in characteristic zebra striping lesions. The diagnostic methods are similar to PPR. The two viruses can be differentiated by virus neutralisation test and monoclonal antibodies.

CONTAGIOUS ECTHYMA

Contagious ecthyma (contagious pustular dermatitis or orf) is a highly infectious disease of goats and sheep characterised by pustular and scabby lesions on the muzzle, commissures

of the lips and nostrils. The disease is caused by a contagious ecthyma virus of the genus *Parapoxvirus* and family Poxviridae and, it also affects man.

Epidemiology

Contagious ecthyma is endemic in most African countries. The sero-prevalence of the disease in goats has been reported to be 3.0-19.0 % in the humid zone of Nigeria, 1.6 % in Mali, 14.0 % in Kenya and 14.0% in Tanzania. The disease has also been encountered in small ruminants in Sudan, Ethiopia, Uganda and Malawi.

All breeds of goats and sheep are affected with contagious ecthyma but the disease is chiefly confined to kids and lambs of 3-6 months old. The source of infection is the affected animal or contaminated inanimate objects and, transmission occurs by contact. The contagious ecthyma virus cannot penetrate the intact skin, hence abrasions of the skin caused by spiky plants, hypodermic needles and surgical operations facilitate the penetration of the virus. The contagious ecthyma virus is highly resistant to desiccation and can survive in dry scabs for years.

Pathogenesis

Initial multiplication of the virus occurs at the site of primary infection. Viraemia develops and is followed by subsequent localisation of the virus in the epithelial cells of the Malphigian layer of epidermis of the target organs especially the head, extremities and udder. Cells of the genital tract, lungs and liver can also be infected. The cytopathic effects of the virus in the infected cells include the development of papules, vesicles, pustules and scabs.

Clinical features

Contagious ecthyma is characterised initially by appearance of erythema which later develop into papules and pustules. When the pustules rupture, the pus forms a thick layer of grey crust and later on result in discrete and thick scabs which are crumbly but adherent to the underlying tissues. Lesions usually begin at the oral commissures and then spread to the lips, muzzle, nostrils, ears and sometimes to the buccal and nasal mucosae. Lesions may also occur on the coronet, interdigital cleft, skin of the udder and teats, vulva, preputial orifice, perineal area, thighs and axillae. Adjoining scabs coalesce and form continuous plaques. Fissures which occur between scabs cause soreness. The lesions may become malignant. Invasion of the virus into respiratory and alimentary tracts may occur leading to pneumonia and gastroenteritis. Morbidity may reach 70-90 % but mortality is rare although it may be considerable if secondary bacterial infection occurs. Spontaneous recovery occurs in 2-3 weeks. Lesions along the alimentary tract interfere with feeding and result in considerable weight loss and emaciation.

Pathological features

At gross pathology, contagious ecthyma is characterised scabby lesions on the affected areas and in malignant cases there are ulcerative lesions in the nasal cavity, trachea, oesophagus, abomasum and small intestine. Inflammation and oedema of the affected dermis is evident in histopathological sections. Parakeratosis, acanthosis, and ballooning degeneration occur in keratinocytes. Nuclear pyknosis and oesinophilic intracytoplasmic inclusion bodies in the affected cells are common features. The virus can be

demonstrated in ultra-thin sections of the affected tissue by examination under electron microscope.

Diagnosis

Clinical signs and lesions of contagious ecthyma are quite characteristic. Confirmation of the disease can be achieved by electron microscopy, tissue culture and transmission experiments. Complement fixation, virus neutralisation and gel diffusion tests are the common serological methods used in the diagnosis of the disease.

Contagious ecthyma has to be differentiated from dermatophilosis, bluetongue, goat/sheep pox and ulcerative dermatosis. Lesions caused by dermatophilosis are small, light and yellowish in colour and can be easily removed unlike the thick greyish black tenacious scabs of contagious ecthyma and, distortion of the lips and muzzle is not observed in dermatophilosis. Dermatophilosis can be confirmed by demonstration of Gram-positive mycelial-like *D. congolensis* in impression smears made from the under surface of the lesions. Bluetongue is commonly associated with the presence of vector midges (*Culicoides* spp) and is characterised by a systemic reaction; excoriation, ulceration and necrosis of the lips and buccal mucosa; lesions in the coronet and high mortality which are not features of contagious ecthyma. The presence of distinctive pox lesions, a febrile reaction and high mortality are features that can be used to distinguish goat/sheeppox from contagious ecthyma. Ulcerative dermatosis is manifested by pyo-ulcerative lesions with thin, brown and bloody scabs. However, isolation and characterisation of the causative viruses is important in order to confirm the above diseases.

Control

Antiseptic ointments such as Lugol's iodine are used to protect secondary bacterial infection on lesions. Parenteral antibiotics are useful in the treatment of systemic secondary bacterial infections. Good nursing such as provision of soft diet to severely affected cases may enhance recovery. Local autogenous live-virus vaccines prepared from vesiculo-pustular material are effective. Attenuated virus vaccines are commercially available. In endemic areas, annual vaccination of 6-8 week kids or lambs is recommended.

GOAT AND SHEEPPOX

This is an acute, febrile and highly contagious disease of goats and sheep which is characterised by fever, cutaneous nodular or visceral eruptions. Goat and sheep pox are caused by the goat and sheep pox viruses of the genus *Capripoxvirus* and family Poxviridae. The viruses closely resemble each other although they are antigenically different. A Kenya sheep and goat pox virus which is closely related to the lumpy skin disease virus also causes the disease in both goats and sheep.

Epidemiology

Goat and sheep pox is endemic in some sub-Saharan countries especially north of the Equator. Morbidity rates of 25 % in Nigeria, 18 % in Mali and 83% in Tanzania have been reported. Information regarding the occurrence of goat/sheeppox in southern

Africa is lacking.

Goat and sheep pox affects animals of all ages although kids and lambs are most susceptible. The disease is highly contagious and transmission occurs mainly by inhalation but animals can also be infected by drinking contaminated milk or by direct contact. Cuts and abrasions on the skin facilitate entry of the virus into the body. The virus can be spread mechanically by insects, birds and personnel. Transplacental transmission has been demonstrated. Congregation of animals in communal grazing lands, markets, dips and the dry and dusty environments are favourable for the transmission of the disease because the virus can be excreted through nasal and conjunctival discharges. Such husbandry conditions were considered to be important epidemiological factors for the outbreak of the disease in northern Tanzania in 1983.

Pathogenesis

Following infection the virus is carried through the lymphatic system to the blood circulation. Viraemia develops and is followed by localisation of the virus in the skin and mucous membranes and appearance of pox lesions.

Clinical features

The incubation period of goat/sheep pox is about 2 weeks. The disease is more severe in sheep than goats and in young than adult animals. In sheep, the disease is characterised by depression, pyrexia (40-41 °C), anorexia, laboured breathing, ocular and nasal discharges. Cutaneous nodules normally appear on hairless areas of the body such as lips, nostrils, udder, vulva, scrotum and under the tail but they may also occur on hairy areas. Severely affected animals may die before pox lesions appear. The nodules progress through vesicles, pustules and finally become scabs. Severe erosive and ulcerative plaques may be found on the buccal, oesophageal and tracheal mucosae. Lesions in the alimentary tract may lead to diarrhoea whereas, those in the genital tract can cause abortion. A 70% morbidity of sheep pox is common and mortality varies from 5% to 50%. Secondary bacterial pneumonia and mastitis are common sequelae.

In goats, lesions occur mainly on lips, eyes, scrotum, udder and medial aspects of the limbs. Severe infections in goats may extend to the neck, thoracic and abdominal organs as hard intracutaneous nodules. A 51% case fatality in goats was reported in an outbreak of goat pox in Nigeria in 1982. Recovered animals become immune for life.

Pathological features

At necropsy, reddish circular nodules or confluent plaques are observed on the skin. Marked greyish-white subpleural nodules are present in the lungs. The superficial lymph nodes are swollen and oedematous. Erosions and ulcers may be present in the mouth pharynx, oesophagus and trachea. There may also be haemorrhagic gastroenteritis.

Histopathologically, there is hyperplasia of epidermal cells, local oedema and cell degeneration or coagulative necrosis. Vasculitis and thrombosis can also be present. Bronchitis and alveolitis may be observed in the lungs. Intracytoplasmic

inclusion bodies are observed in infected cells. The presence of vacuolated nuclei in infected cells is highly diagnostic.

Diagnosis

The pox lesions are characteristic and highly suggestive of the disease. The demonstration of eosinophilic intracytoplasmic inclusion bodies in cells which show a clear vacuole is pathognomonic. In dead animals, the virus can be isolated from the skin and mouth lesions, lungs and liver tissue suspensions by inoculating in lamb kidney cells or other cell lines whereas, in live animals the virus can be isolated in blood collected during the viraemic stage of the disease. Electron microscopy of the negatively stained preparations of skin and mucous membrane sections show typical infected cells. Virus neutralisation and indirect fluorescent antibody tests are the serological methods commonly used in the diagnosis of goat and sheeppox.

Goat and sheeppox has to be differentiated from contagious ecthyma, FMD, blue tongue, peste des petits ruminants and mange. The pox lesions are quite diagnostic and are different from those observed in contagious ecthyma. The diseases can also be differentiated by electron microscopy or serologically. Foot lesions are used to distinguish goat/sheeppox from FMD. PPR and bluetongue can be distinguished by their oral lesions and mange can be confirmed by demonstration of mange mites on skin scrapings.

Control

No treatment is available for the disease. Goat/sheep pox is a notifiable disease and quarantine measures should be imposed immediately if an outbreak occurs to minimise its spread. The affected flock should be destroyed and infected premises properly disinfected. Commercially available single- or mixed-virus cell culture-derived vaccines are used to vaccinate animals in countries where the disease is endemic.

NAIROBI SHEEP DISEASE

Nairobi sheep disease (NSD) is a tick-borne infection of sheep and goats which is characterised by fever, nasal discharges, dyspnoea, haemorrhagic gastroenteritis, abortion and high mortality. It is caused by a Nairobi sheep disease virus of the genus *Nairovirus* and family *Bunyaviridae*.

Epidemiology

The disease is endemic among goat and sheep populations in East and Central Africa. It has also been reported to occur in Ethiopia, Somalia, Zaire and Mozambique. The disease is considered to have a more wider serological distribution in Africa, than the available reports.

All breeds of sheep and goats are affected with Nairobi sheep disease. The disease is transmitted by *R. appendiculatus* ticks. Both transstadial and transovarial modes of transmission occur. Other ticks considered to be involved in the transmission of the disease include *R. pulchellus*, *R. sinus* and *A. variegatum*. Ticks can harbour the virus for a long time. The distribution of Nairobi sheep disease in East Africa is closely linked with the

distribution of *R. appendiculatus* and the incidence of the disease increases with increased tick activity. Animals in endemic areas tend to be immune but naive animals introduced in such areas suffer a severe disease. Outbreaks may also occur when there is a breakdown of tick control measures. Goats are less susceptible than sheep and adult sheep and goats suffer a more severe disease than lambs or kids.

Pathogenesis

The virus is introduced into the host's through bites infected ticks. Viraemia develops and is followed by localisation of the virus in the liver, spleen, lungs and other organs of the reticuloendothelial system. The NSD virus has a particular predilection in the vascular endothelial cells in which the cytopathic effects include endothelial swelling, oedema and necrosis. The necrosis of the vascular endothelial cells of the mucous membranes of the abomasum, small intestine, gall bladder and female genital tract results in congestion, haemorrhagic and catarrhal inflammation and finally desquamation of the necrotic epithelium.

Clinical features

The incubation period for NSD is about 4-6 days. The disease is characterised clinically by fever (41-42 °C), dullness, anorexia, mucopurulent and blood-stained nasal discharges, ocular discharges, conjunctivitis, dyspnoea and groaning. Straining, severe, mucoid and foetid diarrhoea or dysentery and, abortion occur. Deaths may occur 3-9 days after the onset of the acute disease or after 11 days in less acute cases: The clinical signs are less severe in goats. The case fatality rate may reach 90 %.

Pathological features

At necropsy the carcass is dehydrated with soiled hind quarters. Dry crusts are observed on the nostrils and may occlude the nasal passages. The mesenteric lymph nodes are enlarged and oedematous. There may be splenomegaly and swelling of superficial lymph nodes. There is marked haemorrhagic inflammation of the abomasal and intestinal mucosa which present as multiple haemorrhages especially on the longitudinal folds of the abomasum, distal ileum, ileo-caecal valve, caecum and proximal colon. Haemorrhages on the colon and caecum appear as longitudinal striations or lines of ecchymoses extending from the cranial part of the caecum to the rectum. Haemorrhages also occur in the subserosa of the gall bladder, below the kidney capsule and, in the genital and lower respiratory tracts. Pulmonary congestion and alveolar oedema may be evident. The heart becomes pale, flaccid and contains unclotted blood. Epicardial petechiae and endocardial ecchymoses are present. The bone marrow of the long bones become gelatinous and bright red. Dermal haemorrhages are evident in the foetus of dead pregnant ewes.

The histopathological picture is characterised by myocardial and glomerulo-tubular necrosis, degeneration of tubular epithelium, accumulation of hyaline material and cellular casts in the tubuli. Coagulative necrosis of the gall bladder may occur.

Diagnosis

The clinical and pathological features of Nairobi sheep disease are highly suggestive. Recent introduction of animals in an endemic area is an important epidemiological factor to be considered. The liver, lungs, spleen, mesenteric lymph nodes contain high titres of

the virus and the virus may be isolated by inoculation of the affected tissue suspensions in several cell lines and infant mouse brain. The virus can also be isolated from whole blood or serum taken from animals during the febrile stage of the disease. Inoculation of susceptible sheep with suspensions from the affected organs or blood and subsequent development of clinical disease is the most sensitive method of diagnosis. Viral antigens may be detected in the spleen, lung tissue and mesenteric lymph nodes using NSD-virus specific hyperimmune serum. Complement fixation, virus neutralisation, agar gel immunodiffusion, ELISA, indirect haemagglutination and immunofluorescence tests are common employed in the serological diagnosis of the disease.

The differential diagnosis of Nairobi sheep disease include peste des petits ruminants, rinderpest, Rift Valley fever, heartwater and salmonellosis. Virological and serological tests are necessary to differentiate NSD from the other viral diseases. Hepatic necrosis which occurs in RVF is diagnostic. However, inoculation of blood or tissue suspensions from suspected animals into susceptible sheep and subsequent development of a clinical disease is considered to be the most sensitive method for confirmation of NSD. Demonstration of *C. ruminantium* in the cytoplasm of endothelial cells of blood vessels or in Giemsa-stained lymph node or brain biopsy smears will confirm heartwater. Salmonellosis can be confirmed by isolation and characterisation of *Salmonella* spp from the intestinal lesions or contents.

Control

No effective treatment is available for NSD. Fluid therapy is recommended in diarrhoeic or dysenteric cases. Sick animals should be isolated and well nursed. Control of ticks by dipping or spraying of susceptible flocks can reduce the transmission and incidence of the disease. Vaccination of susceptible risk groups such as naive animals before being introduced into endemic area is recommended. Modified live-virus or inactivated-virus vaccines are available. However, vaccination is very not commonly practised in endemic areas because animals tend to be immune and losses sporadic.

BLUETONGUE

This is an arthropod-borne disease of ruminants particularly sheep which is characterised by catarrhal stomatitis, haemorrhages, enteritis, cyanosis of the oronasal cavity, laminitis, oedema of the head and neck and, torticollis. Bluetongue (BT) is caused by a blue tongue virus of the genus *Orbivirus* and family Reoviridae.

Epidemiology

Blue tongue is endemic among sheep and goat populations in Africa. Reports of outbreaks are available from Ghana, Nigeria, Guinea, Kenya and South Africa. Biting midges (*Culicoides* spp) are the principal vectors of the disease and *Culicoides imicola* is considered to be the main species involved in the transmission of the disease in Africa. The infected midges can remain infective for life. Mechanical transmission by the sheep ked (*Melophaga ovinus*), ticks (*A. variegatum* and *Ornithodoros coviaceus*) and blood sucking flies such as *Stomoxys* spp and *Tabanus* spp may also occur. The incidence and distribution of the disease is related to the environmental and climatic factors which affect the survival and distribution of the vectors. Sheep are more susceptible than goats and young animals are more severely affected than adult ones. Introduction of naive

animals into an endemic area may result in an outbreak of a severe disease. Recovered sheep harbour the virus for some months. Cattle and wild animals may act as reservoirs of the virus.

Pathogenesis

Following infection the virus is carried through the lymphatic vessels to the blood circulation and viraemia develops. Replication of the virus also occurs in lymphoid tissues and is followed by localisation of the virus in the vascular endothelial cells resulting in the destruction of the vessel walls. Haemorrhages, exudation, oedema, stasis and occlusion of the blood vessels give rise to hypoxia and other lesions epithelial lesions. It has been observed that exposure of the affected animals to sunlight and other stress factors exacerbates the disease. The presence of the virus in semen causes structural changes in the spermatozoa. The virus can cross the placental barrier/ invade the foetus and cause death because of hepatic necrosis and suppression of the foetal haemopoietic system.

Clinical features

Acute and chronic forms of the disease occur. The acute disease is characterised by fever (41-42 °C); hyperaemia of the buccal mucosa, muzzle, eyes, ears; increased salivation (which may become blood-stained due to excoriations of the buccal mucosa); conjunctivitis with ocular discharges and serous, mucopurulent or blood-stained nasal discharges which form crusts that may interfere with breathing. Oedema of the tongue, lips, face, eyelids, ears and submandibular region occur. Erosions and petechial haemorrhages of the muco-cutaneous junction of the lips, muzzle, buccal, nasal and conjunctival mucosae are evident. There is anorexia, cyanosis of the tongue which may protrude out of the mouth (blue tongue), necrosis of the buccal mucosa, foetid breath, ruminal stasis and occasionally vomiting and terminal diarrhoea or dysentery.

Foot lesions are characterised by hyperaemia of the coronary band, warm and painful feet, reluctance to move or severe lameness which may be manifested by walking on the knees. A dark-red to purple band in the skin above the coronet is diagnostic. Later on the hoof sloughs off. Muscle degeneration and cachexia are common features with degeneration of the neck muscles resulting in torticollis. The clinical signs are less severe in endemic areas.

Pathological features

The post-mortem picture is characterised by mucosal and skin lesions, generalised oedema, hyperaemia, haemorrhage and necrosis of the skeletal and cardiac muscles. Petechial or ecchymotic haemorrhages at the base of the pulmonary artery is a distinctive feature.

Haemorrhages and hyaline degeneration of muscles occur. Copious froth in the trachea, hydrothorax and oedema of the lungs are common features. Aspiration pneumonia may be observed. The nasal, pharyngeal and tracheal mucosae become petechiated and oedematous. Hyperaemia, petechiation, erosion or ulceration on of papillae of the fore stomachs, rumenal pillars, reticular folds, oesophageal groove and abomasal mucosae may be evident. Catarrhal or haemorrhagic enteritis, hyperaemia, oedema and cyanosis of the mucous membranes are features. The inside of the lips, dental pads, cheeks and tongue

become excoriated and ulcerated. Pharyngeal, cervical and thoracic lymph nodes become oedematous and swollen. There may be splenomegaly, epicardial haemorrhages and hydropericardium with fibrin clots. Generalised hyperaemia and malformations are observed in foetuses.

Histopathologically, there is hyaline degeneration, necrosis and mineralisation of muscle fibres. The muscles are infiltrated with neutrophils, macrophages and lymphocytes. Lesions in the foetus include generalised hyperaemia, focal haemorrhages and symmetrical bilateral leucoencephalomalacia.

Diagnosis

Epidemiological, clinical and pathological features may be suggestive of the disease. The virus can be isolated from blood or scarified tissue suspensions by intravascular inoculation in 10-12 days old embryonated hen's egg or in mouse brain after passage of the virus in the hen's egg. The virus can also be isolated in BHK21, Vero and mouse L-cells. Inoculation of blood from suspected animals into susceptible sheep and subsequent development of a clinical disease is diagnostic. Fluorescent antibody test, indirect or competitive ELISA and gel immunodiffusion are the serological methods used in the diagnosis of bluetongue. Virus neutralisation and plaque reduction or plaque inhibition tests are used for serotyping. DNA probes are used for diagnosis in advanced laboratories.

Bluetongue should be differentiated from foot and mouth disease, foot rot, polyarthritis, contagious ecthyma, sheep/goat pox, heartwater and pulpy kidney disease. Unlike FMD, bluetongue has a seasonal pattern of distribution which is closely related to the activity of *Culicoides* spp and, cyanosis of the mucous membranes is not a feature of FMD. Goat/sheeppox has characteristic pox lesions and is highly fatal compared to bluetongue. Contagious ecthyma and ulcerative dermatosis have characteristic lesions. The reproduction of a clinical disease followed by production of neutralising antibodies and resistance to subsequent challenge by susceptible sheep inoculated with blood from suspected cases is diagnostic. The virological and serological methods described above are also useful to differentiate bluetongue from other viral diseases. Heartwater, foot rot, pulpy kidney disease and polyarthritis can be confirmed by demonstration of the causative organisms.

Control

There is no effective treatment for blue tongue. Good nursing of sick animals is recommended. Control can be achieved by restriction of entry of animals from endemic areas. Control of the midges and blood sucking insects will reduce the transmission of the disease. In endemic areas, it is recommended to vaccinate the susceptible stocks one month before the predicted occurrence of the disease. A polyvalent attenuated virus vaccine is produced in South Africa. A modified live virus vaccine is also available. Vaccination of ewes between 4-8 weeks of pregnancy has been associated with deformities in lambs.

RIFT VALLEY FEVER

Rift Valley Fever (RFV) is a mosquito-borne peracute or acute disease of ruminants which

is characterised by fever, necrotic hepatitis, haemorrhages, abortion and high mortality in young animals. The disease, which is also a serious zoonosis is caused by a Rift Valley fever virus of the genus *Phlebovirus* and family Bunyaviridae.

Epidemiology

RVF is widespread in Africa and serious outbreaks have been encountered in both animals and man in Egypt, Sudan, Kenya, South Africa, Zimbabwe, Zambia and Senegal. The disease has also been encountered in Guinea, Nigeria, Burkina Faso, Gabon, Central African Republic, Zaire, Uganda, Tanzania, Mozambique and Angola.

Sick or carrier animals are the sources of infection. The virus is transmitted by mosquitoes and *Aedes mcintoshi* is considered to be the main vector. *Aedes dentatus*, *Culex* spp and *Anopheles* spp can also transmit the disease. *Hyalomma truncatum* ticks have also been incriminated to play part in the transmission of the disease. The incidence of the disease increases with increase in vector activity. The disease is common in areas with dense vegetation and heavy rainfall which are favourable for the multiplication of the mosquitoes. It has been observed that floods may be followed by outbreaks. Body secretions except milk do not contain the virus. The virus may also be present in aborted foetuses. Sheep are more susceptible than goats and lambs and kids are more severely affected than adult animals. Man is infected by penetration of the virus through abrasions on the skin or mucosa when handling infective materials or by inhalation of infective aerosols.

Pathogenesis

After injection of the virus in tissues, initial replication occurs at the site of infection followed by viraemia and localisation of the virus in the target organs especially the liver, spleen and kidneys. Further replication of the virus in these organs amplifies the viraemia. Severe destruction of the hepatic cells which is caused by the cytopathic effects of the virus or immunopathological mechanisms results in hepatic insufficiency. Damage to the blood vessel walls causes vasculitis and widespread haemorrhages in the affected tissues. Abortion is caused by placentitis or foetal death following invasion of virus to the gravid uterus.

Clinical features

The incubation period of RVF in neonatal lambs is 24-36 hours. The disease is characterised by high fever (41°C), listlessness, rapid respiration, abdominal pain, staggering gait and recumbency. Death in severely affected animals can occur 36 hours after the onset of clinical signs. Mortality in neonatal lambs and kids may be over 90%.

Lambs and kids over 2 weeks old and adult sheep and goats are much less susceptible but may develop a peracute, acute or inapparent infection. The acute disease, with an incubation period of 1-3 days is the commonest form of field infection. It is characterised by high fever (up to 42°C) which may last for 1-4 days, anorexia, weakness, listlessness, staggering gait and increased respiration. There may also be melaena or diarrhoea and blood-stained mucopurulent nasal discharges. The ingesta may be regurgitated. Jaundice is commonly observed in adult animals. Abortion of autolysed foetuses at any stage of pregnancy may occur. Salpingitis, purulent metritis and placental retention usually occur and are followed by a high rate of infertility. Congenital anomalies may occur in lambs or

kids born from infected dams. During outbreaks the mortality may reach 50% in goats and 60% in sheep while an abortion rate of 80-100% has been reported.

Pathological features

Peripheral and visceral lymph nodes are enlarged, oedematous and petechiated. The liver shows a bright yellow or yellowish-brown discoloration with congestion and haemorrhages in the parenchyma. Greyish-white necrotic foci occur throughout the liver parenchyma (mottled appearance). Fibrinous peri-hepatitis and haematomas may be evident. The gall bladder wall becomes haemorrhagic and oedematous. Petechial or ecchymotic haemorrhages are observed on the abomasal mucosa and in neonatal lambs chocolate-brown blood is found in the abomasum. There are also widespread subcutaneous, serosal and visceral haemorrhages. The body cavities may contain blood-tinged, fluid and in lambs, haemoperitoneum may be observed. The lungs are congested and oedematous. The hepatic lesions are also present in aborted foetuses.

Severe coagulative centrilobular necrosis with the loss of the hepatic architecture is the most characteristic histopathological feature. The necrosis is diffuse in lambs and multi-focal in adult animals. Some hepatocytes contain acidophilic intracytoplasmic and oval or rod-like intranuclear inclusion bodies. Hyaline material accumulates in the cytoplasm of necrotic hepatocytes. In the new-born lambs or kids there is pyknosis and karyorrhexis of lymphocytes, cloudy swelling and hydropic degeneration of epithelial cells of the convoluted tubules and glomeruli. Multi-focal necrosis and haemorrhage of the adrenal cortex may also be evident. Nephrosis with severe tubular degeneration, hyaline casts in the tubular lumen and fibrin deposits in the glomeruli are features of the disease in adult animals. The clinical pathology is characterised by severe leucopenia.

Diagnosis

The epidemiology, clinical signs and *post mortem* features may be suggestive of the disease. Histopathological features are pathognomonic. The virus can be isolated from blood, plasma or serum collected from the affected live animals during the viraemic phase of the disease or from the liver, kidneys, spleen, lymph nodes and the heart of dead animals by inoculation in Vero cells; BHK 21; mosquito line cells; primary calf, lamb and goat kidney or testis cells. The virus may also be isolated from suckling and weaned hamster inoculated intraperitoneally or intracerebrally. Viral antigens in tissue suspensions may be detected by immunofluorescence, complement fixation, immunodiffusion and in tissue sections by immunoperoxidase staining. ELISA, virus neutralisation and reverse haemagglutination tests are used to detect viral antigens in serum. Antibodies can also be detected by complement fixation, virus neutralisation, immunodiffusion, ELISA, radio-immunoassay and by haemagglutination inhibition tests.

The characteristic hepatic lesions distinguishes RVF from other diseases with similar clinical signs such as Wesselsbron disease which is common in southern Africa, peste des petits ruminants, Nairobi sheep disease, enterotoxaemia and plant poisoning. In addition, abortion and mortality rates observed in RVF are higher than in Wesselsbron disease. Stomatitis and buccal mucosal lesions which are features of PPR are not observed in RVF. The occurrence of NSD is closely associated with distribution and activity of *R. appendiculatus* ticks. A recent history of grazing on poisoning plants will help to differentiate RVF from plant poisoning. Clostridial enterotoxaemia

can be confirmed by isolation and characterisation of the causative bacteria or fluorescent antibody test. Abortion caused by brucellosis and salmonellosis can be confirmed by isolation and identification of the causative bacteria. Other serological and microbiological tests applicable to the different diseases should be carried out to confirm them.

Control

There is no effective treatment for the disease. Control can be achieved by restriction of entry of animals from endemic into non-endemic areas. Vaccination of newly introduced animals is recommended. The modified live Smithburn vaccine is cheap and stimulates a long lasting immunity but, the organisms can revert to virulence and induce abortion or teratology of the foetus. The formalin-inactivated cell culture vaccine is expensive and elicits a short lasting immunity but does not induce abortion or teratology of the foetus. Control of mosquitoes by spraying with insecticides and destroying their breeding habitats can reduce the transmission and incidence of the disease in endemic areas.

OVINE PROGRESSIVE PNEUMONIA (MAEDI/VISNA)

This is an insidious disease of sheep mainly characterised by progressive weight loss and dyspnoea. It is caused by an ovine progressive pneumonia virus of the subfamily Lentivirinae and family Retroviridae.

Epidemiology

The disease has been reported to occur world-wide. It is mainly encountered in adult sheep over 3 years of age. Housing and close confinement facilitate transmission of infective droplets. The virus can also be transmitted mechanically by biting insects, fleas, lice and surgical equipment. Infection through contaminated water may occur.

Pathogenesis

The pathogenesis of the disease is not well elucidated but it has been demonstrated experimentally that after infection the virus multiplies in the regional lymph nodes. Viraemia develops and the virus spreads to the target organs.

Clinical features

The course of the disease takes about 3-8 months but it may be shortened when animals are exposed to stress factors such as inclement weather, poor nutrition and intercurrent diseases especially *Pasteurella* spp pneumonia. The affected animals lag behind the flock, the nostrils are flared and inspiration is associated with rhythmic jerking of the neck. There may be nasal discharges or coughing. The animal progressively loses condition although it remains alert and maintains its appetite. Severe dyspnoea which is exacerbated by exercise develops in arid the animal spends most of the time lying down. Chronic suppurative arthritis of the carpal and tarsal joints and mastitis are often associated with OPP.

A nervous form of the syndrome (visna) may occur. It has a shorter incubation period than the pulmonary syndrome and it is characterised initially by weakness of the hind

limbs, straggling and stumbling or falling without any apparent cause. Progressive weight loss and trembling of the facial muscles may occur. There may also be ataxia, paresis, paraplegia and quadriplegia. There is no fever and the animal remains alert.

Pathological features

At necropsy, OPP is characterised by a marked extensive consolidation of the lungs, multi-focal grey foci on the surface and marked enlargement of the bronchial and mediastinal lymph nodes. At histopathology, there is hyperplasia of the fibrous tissue and muscles of the alveolar septa and, mononuclear cell infiltration.

In visna, there may be myogenic muscle atrophy and minimal lung abnormalities but the histopathological picture is dominated by a de-myelinating leucoencephalomyelitis, mononuclear cell infiltration, perivascular cuffing and neuronal necrosis. Demyelination and the malacic lesions are scattered throughout the central nervous tissue.

Clinical features and pathological features may support a provisional diagnosis of the disease. ELISA, complement fixation, gel diffusion and plaque reduction tests are used for the serological diagnosis of the disease. Monoclonal antibodies are also employed in specialised laboratories. The differential diagnosis of OPP include chronic bacterial, fungal and mycoplasmal pneumonia and, parainfluenza- or adenovirus infections. The neurological form of the disease can be differentiated from hypomagnesaemia, copper, selenium or and vitamin E deficiency by consideration of the feeding history and clinical pathology. *Cl. perfringens* enterotoxaemia, listeriosis and brain abscesses can be confirmed by demonstration of the causative bacteria and, rabies can be differentiated on the basis of history and confirmation by serological tests. In neonatal animals, polyarthritis caused by *Chlamydia* spp and bacterial infection through the umbilicus can be differentiated by isolation of the causative organisms. Mastitis caused by *Staphylococcus* ssp and *Streptococcus* spp can be differentiated by isolation of the bacteria. Serological tests are also used. Control of the disease is achieved by culling of clinically affected animals or positive reactors on serological tests.

Other viral infections of sheep and goats such as pulmonary adenomatosis (jaagsiekte) and caprine arthritis encephalitis are not common in sub-Saharan Africa, but there is a danger for their introduction with exotic breeds of goats and sheep from endemic other area.

RABIES

This is a highly fatal disease of the central nervous system affecting animals and man which is characterised by abnormal behaviour and paralysis. It is caused by a neurotropic rabies virus of the genus *Lyssavirus* and family Rhabdoviridae.

Epidemiology

Rabies is endemic in Africa and reports of fatal human and animal cases of rabies are available from most countries. Different antigenic variants of the rabies virus

have been isolated from different geographical locations. The rabies virus is sensitive to most disinfectants, heat and irradiation. Animals or human beings become infected through bite wounds caused by a rabid animal. For example, stray dogs and cats are the major sources of livestock and human rabies in Tanzania. Infection may also occur by inhalation of infective aerosols. Wild animals such as foxes, wolves, and vampire bats can be reservoirs and source of the disease to livestock or human beings.

Pathogenesis

Infective saliva is deposited in the wound or abrasions during a bite by a rabid animal. Initial replication of the virus occurs in the muscle or subepithelial tissue cells at the site of infection. When a sufficient concentration of the virus is reached it binds to acetylcholine or other receptors at the motor or sensory nerve endings. The virus passively moves centripetally along the peripheral nervous system to the spinal cord and the brain. The presence of the virus in the nervous system results in nervous dysfunction. Replication of the virus in the limbic system is associated with the furious form of the disease. Further replication and spread of the virus to the neocortex causes the dumb form of the disease. After replication in the central nervous system the virus spreads centrifugally to other tissues such as adrenal cortex, pancreas and salivary glands. The furious form of the disease coincides with a high concentration of the virus in the saliva.

Clinical signs

The incubation period is about 14-90 days but sequestration of the virus in striated muscles before ascending through the peripheral nervous to the central nervous system may delay the onset of clinical signs. The clinical signs of rabies in different animal species are similar with only slight variations. Two clinical syndromes; furious and paralytic rabies progressing through the prodromal, excitation and paralytic phase occur. The prodromal phase is characterised by the animal refusing to eat or drink and drop in production.

The furious syndrome is more common in goats and is characterised by aggressiveness and continuous bleating while in sheep, the paralytic form of the disease which is characterised by inability to swallow, excessive salivation, posterior paralysis, convulsions and respiratory arrest. Some animals, however, may show aggressiveness and sexual excitement. In the terminal stages the animal becomes comatose and dies within 5-7 days after the onset of clinical signs.

Pathological features

There are no significant gross features associated with the disease and at histopathology there may be a moderate mononuclear cell infiltration.

Diagnosis

A history of being bitten by a rabid animal and the clinical signs may support a presumptive diagnosis of the disease. A rabies-suspected animal should be confined and the clinical course closely monitored. If the animal survives for more than 10 days then the presence of rabies is ruled out. In live animals the virus may be isolated from saliva or brain biopsies. Sometimes the virus can be demonstrated in impression smears made from the cornea. In dead animals, the presence of intracytoplasmic inclusion bodies (Negri bodies)

in neurones of formalin-fixed and paraffin-embedded brain tissue sections particularly in the cerebellum and brain stem is pathognomonic. The fluorescent antibody test has been used to confirm the disease for many years. Intracerebral inoculation of suckling or weaning mice with 10% brain suspensions from a suspected animals will result in nervous signs and presence of the virus in cells in 14-18 days. The virus can be isolated by inoculation of tissue suspensions onto monolayers or mouse neuroblastoma cells after incubation for 1-4 days, and then confirmed by fluorescent antibody test. ELISA is also a reliable method of diagnosis especially when facilities for fluorescent antibody test are lacking. Other serological tests include radioimmunoassay, passive haemagglutination and indirect fluorescent antibody tests radioimmunoassay. Brain material for laboratory examination should be kept in cold packs and transported in crush resistant containers.

Rabies should be differentiated from other disease manifested by nervous signs such as enterotoxaemia and pregnancy toxaemia and choke. Enterotoxaemia can be differentiated by identification of the causative bacteria and toxins in the intestinal tract, whereas pregnancy toxaemia can be differentiated on the basis of pregnancy status and nutritional status of the animal and, marked ketonemi or ketonuria. Choke can be confirmed by location of a foreign body in the pharyngeal or oesophageal regions.

Control

In endemic areas, all dogs and cats should be vaccinated and all stray dogs and cats should be destroyed. Vaccination of valuable stock at risk such as breeding animals may be necessary. People who are at risk such as veterinarians and animal attendants should be immunised. People who have been bitten with pets or livestock whose vaccination history is unknown should seek medical advice immediately. Tissues or secretions from rabid animals such as meat and milk should not be used for human consumption. Co-operation between the veterinary and medical professionals is of paramount importance in the control of the disease both in livestock and man.

FOOT AND MOUTH DISEASE (FMD)

This is a highly infectious disease affecting cloven-hoofed animals caused by a foot and mouth disease virus of the genus *Aphthovirus* and family Picornaviridae and characterised mainly by fever, stomatitis, lameness and fall in production.

Epidemiology

FMD is enzootic in Africa and other parts of the world and is characterised by considerable losses especially in the dairy cattle industry. Of the seven serotypes which are associated with the disease (A, O, C, SAT-1, SAT-2 and SAT-3), only serotypes SAT-1, SAT-2 and SAT-3 are commonly associated with the disease in the sub-Saharan region. The FMD virus is easily destroyed by sunlight, boiling, autoclaving under pressure and 1-2 % formalin but it is resistant to other disinfectants and environmental conditions. The virus can be present in saliva, milk, blood, urine, faeces and vesicular fluid which become sources of infection to in-contact susceptible animals. Cattle, sheep and goats may harbour the virus for up to 6 months. Camels and buffaloes are said to be asymptomatic carriers.

Inhalation of infective aerosols is the main method of transmission although the virus can also be acquired through ingestion of contaminated food and water, inoculation with contaminated vaccines and insemination with infected semen. The virus can be carried by wind to distant places. Movement of animals and people between herds can transmit the disease. After infection, initial multiplication occurs at the site of entry resulting in the formation of primary vesicles. Viraemia then develops and is followed by spread of the virus to target tissues where further replication occurs resulting in a large number of secondary vesicles. The FMD virus is epitheliotropic and the mucous membranes and the skin are the main target organs. Damage to the mucous membranes or skin facilitates penetration of the virus.

Clinical features

In goats the disease is characterised by formation of vesicles in the coronary band and interdigital cleft. Vesicles may also occur on the lower lip, commissures of the lips and buccal mucosa. Rupture of vesicles causes small fissures. In sheep, small vesicles appear on the feet resulting in lameness, although they may also be encountered on the udder and vulva. Immunity develops after recovery but animals may be infected with other serotypes or subtypes. [Text missing in original] ever, acute stomatitis, anorexia, lameness and marked fall formation of vesicles in the mouth and foot, fall in production and lameness.

Diagnosis

In live animals, the virus can be demonstrated in vesicular fluid, epithelial tissues from the edge of ruptured vesicles, pharyngeal and oesophageal secretions, and blood collected in anticoagulant. The virus can be isolated following inoculation in monolayers of bovine thyroid, suckling mice-, baby hamster, lamb and calf kidney cells. In dead animals, the virus can be isolated from lymph nodes, thyroids, and heart. Samples should be frozen or kept in glycerol buffer (pH 7.6) during transit to the laboratory. The serological tests used for the diagnosis of the disease include ELISA, complement fixation, virus neutralisation and use of monoclonal antibodies. FMD in sheep and goats should be differentiated from other causes of lameness such as bluetongue, polyarthritis and traumatic injury. Bluetongue in sheep is more severe than FMD and cyanosis of the mucous membranes is not observed in FMD. Serological tests can be employed to distinguish these diseases. Polyarthritis and foot rot can be differentiated by isolation of the causative bacteria.

Control

FMD is a notifiable disease. Vaccination of animals is the main method of control of the disease in endemic areas. Restriction entry of animals and animal products from endemic areas has been used to prevent entry of the disease in disease-free countries. If an outbreak occurs strict quarantine measures should be instituted and accompanied with vaccination of animals at risk. Destruction of affected population is used in some countries. Commercial vaccines for FMD are available.

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CHAPTER 6 DISEASES CAUSED BY PROTOZOA AND RICKETTSIA

Coccidiosis and trypanosomosis are the clinically and economically most important protozoan diseases of goats and sheep in sub-Saharan countries. Toxoplasmosis is associated with reproductive wastage in small ruminants but the extent of the problem and the economic significance in the sub-Saharan region is not well documented. *Babesia spp* infection in goats and sheep is an inapparent infection but it may cause a mild or serious disease in exotic or immunosuppressed animals.

COCCIDIOSIS

This is an enteric disease affecting particularly kids and lambs and it is characterised by debility, malaise, inappetance, diarrhoea or sometimes dysentery, dehydration and death in untreated animals.

Aetiology

Coccidiosis in goats and sheep is caused by protozoa of the genus *Eimeria*. The common species of *Eimeria* affecting goats in sub-Saharan countries are *E. alijevei*, *E. arloingi*, *E. ninakohlyakimovae* and *E. christenseni*. Other species are *E. hirci*, *E. caprovina*, *E. jolchijevi* and *E. aspheronica*. *E. arloingi*, *E. christenseni* and *E. ninakohlyakimovae* are considered to be the most pathogenic species of *Eimeria* in goats.

The species affecting sheep include *E. crandallis*, *E. ahsata*, *E. faurei*, *E. intricata* and *E. ovina*. Other species are *E. ovinoidalis*, *E. pallida* and *E. parva*. *E. ovinoidalis*, *E. crandallis*, *E. ovina* and *E. ahsata* are known to be pathogenic in sheep.

Epidemiology

Coccidiosis is widespread among small ruminants and has been reported in all sub-Saharan countries. Outbreaks of clinical Coccidiosis with mortality up to 86% have been reported in Nigeria. Studies carried out in Senegal, Ghana, Kenya, Tanzania, Zimbabwe and Botswana have indicated that Coccidiosis is an important subclinical disease which may be associated with significant economic losses in the small ruminant industry. The prevalence of Coccidiosis among goats and sheep range between 40-90 %.

Animals acquire the infection by ingestion of contaminated feed and water. Non-grazing lambs and kids can acquire infection from infected udders or the wool of their dams. Sub-clinically infected animals continuously shed the oocysts and contaminate the environment. Overstocking and poor hygiene favour rapid transmission and built-up of coccidial infections in animals whereas, stress factors such as weaning, inclement weather, confinement and intercurrent diseases precipitate the occurrence of a clinical disease. Clinical coccidiosis is frequently encountered in intensively managed animals than in extensively managed ones. Coccidiosis is likely to become a more important disease of small ruminants in sub-Saharan countries in future as the increasing land scarcity is forcing people to adopt more intensive management systems.

Temperature, moisture and oxygen tension are the main factors which determine the survival and development of coccidial oocysts to the infective stage. The optimum temperature for the sporulation of most *Eimeria spp* oocysts of sheep and goats is 28-31 °C while temperatures below -40 and above 40 °C are considered to be lethal. Sporulated oocysts are resistant to heat and desiccation and; at 0-5 °C oocysts may remain viable for up to 10 months in faecal sediments and moist pellets. Sunlight and low oxygen tension are detrimental to the oocysts. The climatic conditions of the humid tropics are favourable for the survival and development of coccidial throughout the year.

Transmission

The unsporulated oocysts are voided in faeces of infected hosts and under optimum conditions of temperature, moisture and oxygen tension they sporulate and become infective in 2-5 days. The sporulated oocysts are ingested by goats or sheep followed release of sporozoites in the intestine. Sporozoites penetrate the intestinal wall and become trophozoites. The latter subdivide to form schizonts (meronts). The schizonts rupture and release merozoites which infect new intestinal cells. Asexual (schizogony) or sexual (gametogony) development may occur. During gametogony microgametocytes and macrogametocytes develop into microgametes and macrogametes respectively. Microgametes fertilise intracellular macrogametes and oocysts (zygotes) are produced. When the host cell ruptures, the oocysts are released into the intestinal lumen and are passed out in faeces.

The average prepatent period for *Eimeria spp* of goats and sheep is 2-3 weeks. Variations in prepatent periods between different *Eimeria spp* occur. For example, it has been found that the prepatent periods of *E. ninakohlyakimovae*, *E. alijevi* and *E. christenseni* range between 15-20 days whereas, the prepatent period of *E. intricata*, *E. pvina* and *E. \veybridgensis* range between 20-33 days. *Eimeria spp* are host-specific and cross-transmission between hosts rarely occur.

Pathogenesis

The pathogenesis of the disease is dependent on the effect of developmental stages of the parasite in various regions of the intestine. The number of oocysts ingested, species of *Eimeria* present, age and immune status of the host, location of the parasite in tissues and number of host cells destroyed determine the severity of the disease. Severe damage to the intestinal mucosa is caused by the second generation meronts and sexual stages of *Eimeria*. Destruction of capillaries in the intestinal mucosa may lead to hypoproteinaemia and anaemia. Secondary bacterial infection can occur and cause severe enteritis. The changes in the intestinal mucosa cause increased the rate of peristalsis, malabsorption and diarrhoea. Diarrhoea is followed by dehydration, acidosis, anaemia and terminal shock. Coccidiosis is mainly a disease of kids and lambs up to 4-6 months of age and in adult animals the disease is usually asymptomatic or mild. The clinical disease occurs when young non-immune animals are exposed to massive challenge with sporulated oocysts.

Clinical features

Diarrhoea which may be mucoid or bloody, abdominal pain, tenesmus, inappetence, debility, loss of weight and dehydration are the common features associated with coccidiosis. Anaemia may also be encountered. In the acute disease, there may be fever, ocular and nasal discharges. Subclinical coccidiosis is associated with reduced feed intake, poor weight gains and poor food utilisation. Coccidiosis is self-limiting, however, other enteropathogens can complicate the clinical picture. Exposure to low-grade challenge results in development of strong immunity against the disease. Successive infections in young animals may cause animals to excrete large numbers of oocysts with subsequent heavy contamination of houses, pastures or watering places.

Pathological features

The gross pathological picture includes a thickened, oedematous and sometimes haemorrhagic intestinal wall. Necrosis, greyish-white nodular lesions and polyp-like growths may be seen on the mucosa. The intestinal contents become fluid, dark brown or haemorrhagic.

Denudation of the intestinal epithelium resulting in the shortening or disappearance of the villi occur. Sometimes there may be hyperplasia of the intestinal villi and proliferative lesions on the epithelium. There is hypertrophy and hyperplasia of glandular epithelium are evident. Various developmental stages of *Eimeria* spp can be demonstrated in various sections of the intestinal wall depending on the species of the infecting *Eimeria*. Submucosal oedema and subacute enteritis may also be observed.

Diagnosis

This is based on history, clinical signs, necropsy features and microscopic examination of intestinal mucosa and faeces. The developmental stages of *Eimeria* spp in the intestinal cells can be demonstrated in Giemsa-stained intestinal smears or scrapings and, in haematoxylin eosin stained histological sections. The demonstration of various developmental stages of *Eimeria* spp and the denudation of the intestinal epithelium in dead or sacrificed animals is considered to be a positive diagnosis for coccidiosis.

Faecal oocyst counts can support the diagnosis but they are usually not very reliable because most animals will excrete the oocysts in the absence of the disease and acute coccidiosis may occur before the oocysts are demonstrable in faeces. Furthermore, *Eimeria* species vary in their pathogenicity. However, the presence of very high numbers of oocysts in faeces together with clinical signs may be highly suggestive of the disease. Clinical coccidiosis has been demonstrated in lambs experimentally infected with 100,000-800,000 oocysts of *E. christenseni* and 50,000-500,000 oocysts of *E. ninakohlyakimovae*. Serum antibody quantification is a common serological method for the diagnosis of coccidiosis.

The differential diagnosis of coccidiosis include colibacillosis, salmonellosis, cryptosporidiosis, lamb dysentery and helminthosis. The confirmation of these

conditions can be achieved by isolation and identification of the causative agents from faecal specimens and other affected tissues. Coccidiosis and helminthosis commonly occur together and they can be differentiated on the basis of faecal oocyst or egg counts and demonstration of various developmental stages of coccidial in the intestinal mucosa and/or worms in the mucosa or lumen of the gastrointestinal tract.

Treatment and Control

Coccidiosis can be treated using decoquinate (0.5 mg/kg) and lasolacid at a daily intake of 4.3 mg/kg body weight. Sulphonamides such as sulphadimidine, sulphamerazine, sulphamethazine and sulphaquinoxaline at dosage rates of 50-100 mg/kg for 4 days are effective against coccidiosis in small ruminants. Amprolium in feed is also used to treat the disease in goats (100 mg/kg) and sheep (50 mg/kg). Nitrofurazone given orally (10-20 mg/kg) is also effective. Other drugs include monensin (20 g/ton of feed), toltrazuril and diclazuril.

Coccidiostats in drinking water or feed are commonly employed to control the disease in intensive production systems. Decoquinate (0.3 -4.0 mg/kg) in feed mixtures is a safe and very effective coccidiostat in goats and sheep. Monensin fed prophylactically at 10-30 mg per ton of feed controls shedding of oocysts and increases feed conversion. However, high levels of monensin render the feed unpalatable and toxic.

Proper hygiene in the house and minimisation of predisposing factors are important factors to be considered in the control strategies of coccidiosis. Provision of adequate nutrition enhances the resistance of animals to coccidiosis.

TRYPANOSOMOSIS

Trypanosomosis is a debilitating disease of animals which is characterised by parasitaemia, intermittent fever, anaemia, loss of condition, reduced productivity and mortality.

Aetiology

A haemoflagellate protozoan, *Trypanosoma spp* is the cause of the disease. *T. congolense* and *T. vivax* are the main species associated with clinical trypanosomosis in small ruminants in the sub-Saharan region. *T. brucei* and *T. simiae* are frequently encountered as asymptomatic infections in goats although the latter can cause an acute and fatal disease in sheep.

Epidemiology

The distribution of trypanosomosis in goats and sheep in sub-Saharan Africa is closely related to the ecology and distribution of the vector tsetse flies of the genus *Glossina*. Increased tsetse fly activity particularly during the rainy season is associated with increased incidence of the disease. Clinical caprine trypanosomosis has been reported in Mozambique, Tanzania, Kenya, Uganda, Nigeria and Zambia. Infection rates with *T. congolense* and *T. vivax* in goats varying from 3.5% to 5.0 % have been reported in Nigeria and Kenya. The wide distribution of tsetse flies in the sub-

Saharan region (15°N-30°S) suggests a more extensive distribution of the disease.

Tsetse flies (*Glossina* spp) are the principal vectors of trypanosomosis in sub-Saharan Africa and *G. morsitans* and *G. pallidipes* are the most commonly involved in the transmission of the disease. Other blood sucking flies such as *Stomoxys* spp and *Tabanus* spp may also transmit the disease. Wild animals such as bush pigs, bush bucks, kudus, warthogs and buffaloes act as reservoirs of the infection in endemic areas. Stress favours such as malnutrition, intense heat and intercurrent infections have been shown to render animals more susceptible to the disease. Animals in endemic areas show some trypanotolerance while exotic breeds are much more susceptible.

Pathogenesis

Following introduction into the body by an insect bite, the trypanosomes initially multiply and cause inflammation at the site of infection. The parasites are then carried through the lymphatic channels to the blood circulation. During the parasitaemic stage, trypanosomes release haemolysins and enzymes such as phospholipases, proteases and neuraminidases which cause damage of the cell membranes of erythrocytes. Damage to the red blood cells is followed by disseminated intravascular coagulation. The trypanosomes may also block capillaries causing ischaemia and anaemia.

Clinical features

T. congolense is the most pathogenic species in goats causing an acute, subacute or chronic disease. *T. simiae* can induce a highly acute and fatal disease in sheep. *T. vivax* is less pathogenic while *T. brucei* may affect goats causing an inapparent infection. Thus, the severity of the disease is determined by the pathogenicity of the strain of trypanosome.

The incubation period of acute trypanosomosis caused by *T. congolense* is 5-10 days. The disease is characterised by depression, anorexia, rigidity of the hind limbs, drooping ears, increased heart rate (up to 250 per minute), increased respiratory rate (up to 45 per minute), watery salivation and flaccidity of the tail. The temperature may be subnormal or may reach 41 °C. Mucous nasal discharges, mild conjunctivitis, enlarged superficial lymph nodes, lethargy and recumbency are also evident. Later on, the animal becomes unresponsive to external stimuli and pallor of the mucous membranes become apparent. Death may occur 4-6 weeks post-infection.

The subacute syndrome lasts for 6-12 weeks and is characterised by enlargement and oedema of the superficial lymph nodes (especially the prescapular lymph node), pallor of the mucous membranes and marked jugular pulsation. Other superficial lymph nodes may also be enlarged and oedematous. Animals may recover or die in 10-12 weeks. The course of chronic trypanosomosis in goats takes 12-24 weeks and is characterised by progressive weight loss, rough and dull hair coat, anaemia, weakness and paresis in terminal stages. It has been observed that trypanosomosis grossly impairs the fertility of the affected animals.

Pathological features

Acute trypanosomosis is manifested grossly by a pale and dehydrated carcass; petechial and ecchymotic haemorrhages in the serosal surfaces; enlarged and oedematous lymph nodes and congestion of the liver. The spleen is also enlarged and dark red.

Chronic trypanosomosis is characterised by an oedematous and pale carcass; enlarged lymph nodes; gelatinous fatty degeneration of the heart and kidneys; ascites, hydrothorax and hydropericardium; enlarged and flabby heart and, hepatomegaly. The bone marrow becomes yellowish and gelatinous. Testicular degeneration and, atrophy and, oedema of the choroid plexus may also be observed.

At histopathology, trypanosomes are demonstrable in the lumina of blood vessels in which they may cause thrombosis. The acute disease causes intravascular coagulation. Capillary dilatation and oedema, perivascular mononuclear cell infiltration, .hemosiderosis, and hyperplasia of lymphoid tissues are common features. Macrophages, lymphocytes and plasma cells infiltrate the myocardium, kidneys, thyroids, adrenals and gonads. The clinical pathology of trypanosomosis is characterised by a marked fall in the packed cell volume, normocytic anaemia and leucopenia.

Diagnosis

The epidemiology, particularly the presence of tsetse flies in the area, clinical and pathological features may be useful in a provisional diagnosis of the disease. Wet blood smears are useful in field diagnosis of the disease but they may be unreliable for the detection of light infections. Air-dried thin or thick blood smears stained with 10% Giemsa for 20 minutes are used for specific diagnosis of trypanosomes. In light infections with *T. congolense* and *T. vivax*, the trypanosomes can be concentrated by centrifugation and demonstrated by examination of cells at the leucocyte/plasma interface of heparinised blood by dark ground illumination. This is considered to be the most sensitive method of diagnosis of trypanosomosis. If EDTA is used as anticoagulant, trypanosomes can be demonstrated in Giemsa stained smears of cells from the buffy coat region.

The common serological methods of diagnosis are indirect haemagglutination test and ELISA. Monoclonal antibodies against *T. congolense*, *T. vivax* and *T. brucei* are available. DNA probes are being developed in some specialised laboratories. The differential diagnosis of trypanosomosis includes haemonchosis and malnutrition. Demonstration of trypanosomes in blood circulation is required to rule out helminthosis.

Treatment and Control

Homidium bromide (1 mg/kg), quinapyramine methyl sulphate (5 mg/kg) and diminazene aceturate (3.5 mg/kg) are used for the treatment of *T. congolense* and *T. vivax* infections in small ruminants. Chemoprophylaxis is an important control strategy in endemic areas and isometamidium (0.5 mg/kg) and pyriithidium bromide (2 mg/kg) are the drugs commonly used in small ruminants. Control of the disease depends on the prevention of contact between susceptible animals and vectors particularly tsetse flies. The methods

of control of tsetse flies include bush clearing, spraying of animals and habitats with effective insecticides and the use of tsetse fly traps. The use of trypanotolerant breeds of goats and sheep is also being adopted in some countries. Avoidance of stress factors such as malnutrition, intense heat and intercurrent infections can reduce the incidence of clinical cases in endemic areas.

BABESIOSIS

Babesiosis is an infectious tick-borne disease of livestock characterised by high fever, haemoglobinuria, anaemia and prostration. The disease in goats and sheep is mainly caused by an intra-erythrocytic protozoan, parasite, *Babesia motasi*. *B. ovis* causes inapparent infections.

Epidemiology

Babesia spp infections are widespread among goat and sheep populations in Africa. Babesiosis is transmitted by ticks. *Boophilus*, *Rhipicephalus*, *Hyalomma*, *Ixodes* and *Haemaphysalis* spp are involved in the transmission of different species of *Babesia*. Both transovarial and transstadial transmissions occur. Increased tick activity which is associated with high humidity results in increased incidence of the disease. *Babesia* spp affecting goats and sheep may be maintained in non-susceptible hosts such as wild animals. Presence or absence of intercurrent infections and differences in breed susceptibility determine the incidence and severity of the disease. Movement of naive animals from non-endemic into endemic areas may result in outbreaks.

Pathogenesis

The pathogenesis of babesiosis is related to the damage of erythrocytes and the production of pharmacologically active substances following activation of the kallikrein and complement systems. These substances destroy erythrocytes resulting in intravascular haemolysis. The haemolytic anaemia subsequently result in hypoxia and death.

Clinical features

B. motasi can cause an acute or chronic disease in goats or sheep. The acute disease is manifested by anorexia, fever, fast and audible heart beats, pallor of the mucous membranes, icterus and haemoglobinuria. Other features include abdominal pains, diarrhoea, prostration and death. The chronic disease is manifested mainly by emaciation but coughing and oedema may be features.

Pathological features

The necropsy features include widespread subcutaneous and intramuscular oedema, icteric carcass, thin and watery blood, yellow and gelatinous fat. The urinary bladder contains dark urine. The spleen is enlarged and the splenic pulp is soft. The gall bladder is distended and contains thick and dark bile.

The histopathological picture includes centrilobular necrosis of the liver; haemosiderin deposits in Kupffer cells, congestion of the lungs, heart, spleen and

kidneys. The germinal layers of the spleen and lymph nodes are depleted and the reticular tissue is hyperplastic with large numbers of macrophages containing haemosiderin. Haemosiderosis is also evident in other tissues. The clinical pathology is one of marked reduction in packed cell volume and haemoglobin concentration.

Diagnosis

Clinical features can support a provisional diagnosis of the disease. The demonstration of *Babesia* in thin or thick smears made from peripheral blood and stained with Giemsa is confirmatory. Thick smears are recommended in light infections. *B. motasi* occur singly or in pairs and in the latter form the angle formed between them is acute while *B. ovis* often occur singly. Indirect haemagglutination and complement fixation tests are the serological methods used in the diagnosis of the disease.

Treatment and Control

Babesiosis can be treated using diminazene aceturate (2.0-3.5 mg/kg), imidocarb (2.0 mg/kg) three times at 24 hours interval and amicarbilide (10 mg/kg). Quinuronium is also used. The control of the disease depends on effective control of ticks by dipping or spraying animals at risk with recommended acaricides. In small herding units, ticks can be manually removed from the bodies of infested animals. Rotational grazing is employed to control ticks in some commercial or institutional farms.

HEARTWATER

Heartwater is a tick-borne rickettsial disease of ruminants which is characterised clinically by pyrexia, nervous signs, diarrhoea and death and; at necropsy by hydropericardium, hydrothorax and oedema of the lungs and brain. The disease is caused by a pleomorphic rickettsia, *Cowdria ruminantium*.

Epidemiology

Heartwater is endemic in many parts of Africa affecting both domestic and wild ruminants. The disease has been reported in Madagascar, Nigeria, Kenya, Tanzania, South Africa, Reunion and Sao Tome islands.

The disease is transmitted by *Amblyomma spp* ticks. In most parts of Africa *A. variegatum* is the principal vector. *A. hebraeum* is an important vector of the disease in the Republic of South Africa while *A. gemma* and *A. lepidum* have been reported to be involved in the transmission of heartwater in East Africa. Wild animals maintain the vector ticks and are asymptomatic carriers of the disease. Transmission occurs mainly trans-stadially whereas, transovarial transmission rarely occurs. Increased tick activity which is associated with increased humidity especially during the rainy season may be associated with increased incidence of the disease. Outbreaks occur when susceptible animals from tick-free areas are moved into endemic areas. Different strains of *C. ruminantium* with differences in virulence exist and animals which are immune to one strain may succumb to others. Kids and lambs possess innate resistance in the first week of their life although sometimes they may succumb to a clinical

disease. Goats are more susceptible to heartwater than sheep and the Angora goats are particularly very susceptible. Black Head Persian sheep possess some degree of natural resistance to the disease.

Pathogenesis

The organisms are introduced in the body through the saliva of the infected ticks. Initial multiplication in the regional lymph nodes is followed by colonisation of the endothelial cells of blood vessels in all organs. *C. ruminantium* has a distinct predilection in the endothelial cells of the brain cells. Invasion and colonisation of the endothelial cells causes vascular damage resulting in increased vascular permeability, transudation and effusion of fluids into the body cavities and subsequent development of oedema and hypovolaemia. *C. ruminantium* also produces an endotoxin which, together with increased cerebrospinal fluid pressure are considered to be involved in the pathogenesis of the brain lesions. Progressive pulmonary oedema and hydropericardium results in asphyxia and cardiac insufficiency which terminate into death. Severe renal ischaemia and nephrosis has been reported to occur in goats. The severity of the disease varies depending on the virulence of the infecting organism, breed, age and immune status of the host.

Clinical features

The incubation period of heartwater in goats and sheep is 1-5 weeks and the course of acute disease takes 3-6 days. A peracute, acute, subacute or chronic disease may occur. The peracute syndrome is characterised by sudden death without premonitory signs. Sometimes, animals with a peracute disease may exhibit high fever, prostration and paroxysmal terminal convulsions. The case fatality rate in peracute cases is 100 %. The acute disease is characterised by pyrexia, dullness, anorexia and nervous signs. The nervous signs include unsteady or high-stepping gait, ataxia, circling or galloping movements, chewing movements and aggression. Other signs include dullness, bleating, nystagmus, twitching of the tail, blindness, opisthotonus, droopy ears, lowering of the head, increased urination and forced respiration. Convulsions, prostration and lateral recumbency are observed in terminal stages of the disease. The case fatality rate of the acute disease is 50-90 %.

Subacute heartwater develops in 7-10 days and the clinical signs are less pronounced than in the acute syndrome. They include listlessness, inappetence, loss of weight and hair/wool, recumbency, fall in temperature and ruminal atony. The chronic disease which is common in indigenous breeds of goats and sheep may be characterised by transient fever. Natural recovery and subsequent re-infection may be observed.

Pathological features

Little changes are seen in the peracute disease although petechial haemorrhages may be observed in the endocardium and pericardium. The gross pathological features of the acute disease include ascites, hydrothorax and hydropericardium. The fluid in the pericardial sac is turbid, light yellow and clots on exposure to air. Congestion of the liver and distension of the gall bladder occur. Subserosal haemorrhages, splenomegaly and lymphadenopathy are common features. The trachea and bronchi are filled with a serofibrinous foam, congested and have petechiated and ecchymotic mucosae. The lungs

are oedematous and a frothy fluid exudes from the cut lung surface. The mediastinal and bronchial lymph nodes are oedematous. There is also oedema of the brain and, the meninges are swollen, congested and oedematous. The choroid plexus is dull greyish in colour.

At histopathology there is perivascular infiltration of organs/tissues with macrophages and lymphocytes. Nephrosis, perirenal oedema and petechiation of the renal cortex occur. Lesions in the brain include foci of necrosis and microcavitation in the cerebral cortex, oedema of the axon sheaths, necrotic degeneration and formation of PAS positive granules and globules in the cytoplasm of neurocytes and accumulation of the latter in the perivascular space. Parasitised endothelial cells are distended. *C. ruminantium* colonies can be demonstrated in the cytoplasm of endothelial cells of the brain, lungs and kidneys. At clinical pathology there is oesinophilia, neutrophilia, lymphocytosis, lowered packed cell volume and haemoglobin concentration and, normocytic, normochromic anaemia.

Diagnosis

Presumptive diagnosis can be based on the epidemiological, clinical and pathological features. Demonstration of *C. ruminantium* in the cytoplasm of the endothelial cells of blood vessels or in Giemsa-stained smears of lymph node or brain biopsy samples is confirmatory. In dead animals, the organisms can be demonstrated in brain crush smears prepared from the hippocampus or cerebral cortex. The brain crush smears are air-dried, fixed in methanol for 1 minute and then stained with 10% Giemsa for 30 minutes or 50% Giemsa for 10 minutes. *C. ruminantium* appear as clusters of bluish-purple to reddish-purple cocci in the cytoplasm of vascular endothelial cells. The organisms may also be demonstrated in histological sections of the endothelial cells of renal glomeruli or capillaries of the grey matter of the cerebral cortex.

Other methods of diagnosis include the inoculation of blood from suspected animals in susceptible animals in which reproduction of a clinical disease is a positive diagnosis. Indirect fluorescent antibody test and ELISA are the common serological methods of diagnosis.

Sudden death in peracute heartwater should be differentiated from anthrax and while the nervous signs can also occur in bacterial meningoencephalitis, tetanus, plant and heavy metal poisoning. Demonstration of the causative organisms is necessary in order to differentiate the disease from anthrax, tetanus and bacterial meningoencephalitis. The nervous symptoms observed in tetanus are more severe than those encountered in heartwater. A recent grazing history and presence of poisonous plants or heavy metal contaminants in the animals' environment can be highly suggestive of poisoning. Hydropericardium and hydrothorax may also be observed in bluetongue and pulpy kidney disease. Bluetongue is can be differentiated by the mouth and feet lesions and hydropericardium is not a feature. Virological and serological tests can also be used to differentiate heartwater and bluetongue. Characteristic lesions in the kidney and isolation of *C. perfringens* type D and demonstration of the epsilon toxin will confirm pulpy kidney disease.

Treatment and Control

Treatment of the peracute cases is usually untimely because of sudden death. However, if correctly diagnosed in early stages, the disease can be treated with oxytetracycline (10-20 mg/kg) given parenterally and repeated after 24 hours. Other drugs used include doxycycline (2 mg/kg), rifamycin (0.2 mg/kg) and sulphadimidine. Supportive treatment with diuretics such as furosemide (1 mg/kg) 2-3 times per day for one or two days is recommended to reduce oedema formation.

Control of heartwater depends on effective control of the vector ticks. Live virulent vaccines have been developed but their effectiveness is hampered by the diversity of the strains of *C. ruminantium* in different areas. The infection and treatment method with oxytetracycline is used in some farms in South Africa.

Chemoprophylactic treatment of susceptible animals in endemic areas with oxytetracycline (3 mg/kg) at day 10, 20, 30, 45 and 60 has also been adopted in some commercial production units. When the protocol is adopted, animals are not dipped until day 60. Alternatively, a regime using long acting oxytetracycline (10-20 mg/kg) at day 7, 14 and 21 can be used. The costs involved in chemoprophylactic treatment do not justify adoption of the protocol in the traditional herds.

ANAPLASMOSIS

Anaplasmosis is a mild rickettsial infection of goats and sheep which is characterised in immunosuppressed animals by weakness, anaemia, icterus and respiratory distress. The disease is caused by *Anaplasma ovis*. *A. marginale* can cause latent infections in small ruminants.

Epidemiology

Anaplasmosis is transmitted by ticks. *Rhipicephalus evertsi* and *Hyalomma* spp and *Ornithodoros* spp have been found to be the main vectors of the disease in Africa. Biting insects or inoculation of blood into susceptible animals can also transmit the disease. Recovered animals become carriers. Splenectomy, intercurrent infections such as trypanosomosis, babesiosis, eperythrozoonosis and heavy parasitism lower the resistance of the animals and render them more susceptible to the disease. Other stress factors such as malnutrition and pregnancy also increase the susceptibility of animals to anaplasmosis. Goats have been shown to be more susceptible to *A. ovis* than sheep.

Pathogenesis

The presence of *Anaplasma* spp in erythrocytes induces physical and chemical damage to the cells culminating into erythrocytaphagocytosis. It has also found that the sensitised erythrocytes produce antierythrocytic autoantibodies. Damage to the erythrocytes result in anaemia. The organisms cause a mild disease under natural infections but a clinical and sometimes fatal disease may occur in immuno-compromised animals. Thus, the clinical manifestations of the disease are precipitated by splenectomy, intercurrent infections and other stress factors.

Clinical features

Listlessness, anorexia, weakness, ruminal stasis, respiratory distress, pallor of the mucous membranes, increased heart and respiratory rates are the main clinical signs. Constipation, oedema of the submandibular region and ventral side of the neck and, abortion may occur.

Pathological features

The carcass becomes pale and icteric with thin and watery blood. There is anasarca and presence of straw-coloured fluid in body cavities. The lungs become pale and oedematous; the liver becomes enlarged and yellowish while the gall bladder is filled with yellowish-green bile. Lymphadenopathy and nephrosis may be features.

Histopathological features include erythrophagocytosis, eosinophilia, reticulocytosis, anisocytosis and haemosiderosis of reticuloendothelial cells. Basophilic stippling, centrilobular necrosis of the liver and degenerative changes in the cells of the convoluted tubules occur. The clinical pathology is manifested by a marked fall in the haematocrit.

Diagnosis

A presumptive diagnosis of anaplasmosis is based on clinical features, haematological changes and demonstration of the organisms in erythrocytes. Blood smears prepared on grease-free slides, fixed with methanol and stained with Giemsa show the parasite as a round, uniformly stained black or dark purple body in the cytoplasm of erythrocytes. Reproduction of a clinical disease by inoculation of suspected blood in splenectomised susceptible animals is a diagnostic but it is an expensive and time consuming method. The serological methods of diagnosis include rapid card agglutination, capillary tube agglutination, complement fixation, direct and indirect fluorescent antibody tests.

Anaplasmosis should be differentiated from haemonchosis, trypanosomosis, babesiosis, eperythrozoonosis, copper poisoning and other conditions which are manifested with loss of condition, listlessness, mild fever and anaemia. The demonstration of high nematode egg and worm burdens in anaemic animals can be highly suggestive of haemonchosis. Trypanosomosis can be distinguished from anaplasmosis by the demonstration of *Trypanosoma* spp in blood circulation. *Babesia* spp can be easily demonstrated in erythrocytes of the affected animals while *Eperythrozoon* spp is an extraerythrocytic parasite and much smaller than *Anaplasma* spp. Haemoglobinuria which is common in chronic copper poisoning is not a feature of anaplasmosis.

Treatment and Control

Long acting oxytetracycline (20 mg/kg) repeated one or two times after 7 or 14 days has been shown to be effective in the treatment of anaplasmosis in sheep. Chlortetracycline and imidocarb (5 mg/kg) are also used. Control of the disease depends on effective control of vector ticks and insects. Asepsis during mass vaccination and surgical procedures can prevent mechanical transmission of the disease. Vaccination is not practised and is not justifiable under natural conditions because of the asymptomatic nature of the disease.

ERYTHROZONOSIS

This is an arthropod-borne subclinical disease of sheep and sometimes goats caused by a rickettsial *Eperythrozoon ovis* and is characterised by mild fever, anaemia and icterus in immunosuppressed animals.

Mosquitoes and stable flies are the main vectors of the disease although the disease can also be transmitted by inoculation of blood from affected animals into susceptible animals or infection through surgical procedures such as castration, docking and shearing. Placental infection may also occur. Occurrence of the clinical disease is exacerbated by stress factors. The organism induces erythrophagocytosis resulting in anaemia and icterus.

Clinical signs include anorexia, mild fever, depression, debility, loss of weight, exercise intolerance, pallor of the mucosae and icterus. At necropsy, the carcass is pale or icteric. The kidney becomes reddish-brown and there may also be fatty degeneration of the liver, subcutaneous oedema and hydropericardium. Haemoglobinuria may be encountered. Haemosiderosis in the liver, kidney and spleen are common histopathological features. The clinical pathology is characterised by a fall packed cell volume and haemoglobin concentration, leucopenia and hypoglycemia.

The epi-erythrocytic *E. ovis* can be demonstrated in the Giemsa-stained thick smears as pale-or reddish-purple rings, rods or clusters of organisms around the erythrocytes. ELISA, complement fixation, indirect haemagglutination, direct and indirect immunofluorescent tests are the serological methods used in the diagnosis of the disease. Fluorescent microscopy is also employed. The disease should be differentiated from anaplasmosis, helminthosis, malnutrition and chronic copper poisoning. The mild nature of the disease does not justify treatment, but iminocarb can be effective.

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CHAPTER 7 DISEASES CAUSED BY ARTHROPODS

ECTOPARASITES OF GOATS AND SHEEP

Ectoparasites are not associated with heavy mortalities in small ruminants but they are important causes of unthriftiness and loss of production in affected animals. The common ectoparasites of veterinary importance in sub-Saharan Africa are mange mites, fleas, lice, ticks and *Oestrus ovis*.

MANGE

Mange, a contagious disease of animals caused by parasitic mites, is characterised by a variety of clinical signs depending on the species of mites. The mange mites which are reported to affect small ruminants in sub-Saharan Africa include *Sarcoptes scabiei*, *Psoroptes caprae*, *P. cuniculi*, *P. communis*, *Chorioptes texanus*, *Demodex* spp and rarely *Notoedres cati*.

Epidemiology

Mange mite infestations are widespread among small ruminants across the sub-Saharan region. The prevalence of *S. scabiei*, *P. communis*, *D. folliculorum* in goats in Nigeria have been estimated to be 24-33%, 22% and 11% respectively. In Kenya a 11% prevalence of sarcoptic mange has been reported. Sarcoptic mange is also a common skin condition of goats in Tanzania, Mozambique, Uganda and Mali. Demodectic mange can cause economic loss due to spoiling of hides.

Clinically affected and carrier animals are the source of infestation. Transmission occurs by direct contact and contaminated fomites can be sources of infestation. Overcrowding of animals in houses, markets, dips and communal grazing land facilitates rapid spread of the parasites. Kids and lambs are more severely affected than adult animals. Moist conditions favour the proliferation of the mites while desiccation is detrimental. Poor nutrition and intercurrent infections increase the susceptibility of animals to mange mites.

Clinical features

Sarcoptes spp pierce the skin, suck lymph and feed on young epidermal cells. Their activity causes marked irritation. There is intense itching and the animal rubs on hard surfaces and objects resulting in partial or complete alopecia. Alopecic patches are evident in the medial aspects of the rear limbs, axillae and on the brisket. The infection may also extend to the abdomen, trunk, udder and teats. Dry and bran-like scales are formed on the face, around the nostrils and ears. Later on, the scales transform into hard crusts which extend from the muzzle to the area between the eyes and nostrils; the region between the eyes and horns and, the inner and outer aspects of the ears. Cracks and fissures appear on the skin at the hock joint. The skin is thickened and wrinkled especially that of the scrotum and pinnae of the ears. Heavy dandruff is evident in hairy areas covering the neck and abdominal areas.

Psoroptes spp are which are non-burrowing mites puncture the epidermis, suck lymph and stimulate a local inflammatory reaction. Psoroptic mange in goats is characterised by the accumulation of hyaline material in the external ear canal resulting in occlusion of the canal and deafness. The skin of the pinna becomes wrinkled. Generalised lesions resembling sarcoptic mange may occur. *Psoroptes* spp infestation in sheep causes a highly contagious infection (also known as sheep scab) which is characterised by intense pruritus, restlessness, scratching and rubbing on objects and raised tufts of wool. Reddish or yellowish vesicles, pustules and papules are formed and the exudate from papules results in the formation of crusts and matting of the wool. Later on, tufts of wool fall leaving alopecic areas which may cover the whole body. Thickening and cracking of the skin occur. Severely affected animals become emaciated, anaemic and may die due to exhaustion. Lesions first appear on the lumbar region and spread to other parts of the body.

Chorioptic mange in goats and sheep is characterised by intense erythema, itching, restlessness, scratching and rubbing which results in alopecia. Scales, crusts and wart-like growths, thickening and folding of the skin are common features. In goats, lesions appear on the interdigital clefts, coronet, muzzle, eyelids, udder, scrotum, anal and tail regions whereas, in sheep lesions begin at the fetlock region and spread to the udder and scrotum.

Pathological features

There are no significant pathological features associated with mange mite infestation apart from the skin lesions.

Diagnosis

Tentative diagnosis is based on clinical manifestations and the definitive diagnosis depends on the demonstration of the mange mites in skin scrapings. Skin scrapings are taken using a scalpel from the hairless edge of the lesion. In sarcoptic mange, scrapings are taken where pruritus or pimples are seen and, the skin is scraped until blood oozes. In the laboratory, the samples are boiled in 10% potassium hydroxide for 10 minutes until all hair and crusts are digested. The mixture is then cooled at room temperature and then centrifuged at 576 g for 2 minutes (or 1500 rpm for 15 minutes). The supernatant is decanted and the deposits are microscopically examined at x 100 magnification and identified according to standard keys.

The differential diagnosis of mange include ringworm, dermatophilosis, contagious ecthyma, goat/sheeppox and scrapie. Circumscribed lesions which extend in a ringed fashion are characteristic of ringworm and the infection is confirmed by demonstration of fungal hyphae in scrapings from the lesions. In fleece rot there is no itching whereas, in strawberry foot rot of sheep, a strawberry-like raw surface is evident after removal of the scabs. *D. congolensis* can be demonstrated in impression smears made from the under surface of the scabs in fleece rot and strawberry footrot. Contagious ecthyma is characterised by large, hard and adherent scabs and, alopecia is not a common feature. In scrapie, itching is accompanied by muscle tremors and impaired locomotion which are not observed in mange.

Treatment and Control

Sarcoptic mange can be treated using ivermectin (0.2 mg/kg), 0.05% diazinon, 0.1% phoxim and 0.05% coumaphos. Two applications of phoxim (0.05%) at 10 days interval has been found to be effective against chorioptic mange and, permethrin (4 % w/v) can be used as a pour-on formulation. Propetumphos (0.005%) applied twice or thrice at 10 days interval has been used to treat psoroptic mange. Dipping with other insecticides is useful in the control of the parasites. Hygiene in houses and avoidance of overcrowding will minimise the accumulation and spread of the mites.

FLEA AND LOUSE INFESTATION

Heavy infestation with fleas and lice can cause severe anaemia in young animals in addition to the damage of the skins causing considerable losses in the leather industry. Significant economic losses caused by flea and lice infestation in goats and sheep have been reported in Nigeria, Kenya and Tanzania.

The most common species of fleas reported to infest goats and sheep in sub-Saharan countries are *Ctenocephalides canis* and *C. felis*. *Echidnophaga gallinaea* has also been reported to infest goats. *Linognathus stenopsis*, *L. ovillus*, *Damalinia caprae*, *D. ovis* and *Menacanthus stramineus* are the main species of lice infesting goats and sheep in sub-Saharan countries.

Epidemiology

Infested animals are the sources of infection and they contaminate bedding and farm equipment thus perpetuating the infestation in the herd. Overcrowding and a warm, humid environment favour the built up of fleas and lice burdens. Kids and lambs are more severely affected than adult animals.

Clinical signs

Clinical signs of heavy flea and louse infestation include unthriftiness, restlessness, pruritus, alopecia, body weakness, scratching, rubbing and licking. Heavy infestation with fleas and lice causes anaemia. There is a marked fall in the packed cell volume and haemoglobin concentration which is manifested clinically by pallor of the mucous membranes.

Fleas and lice infestation can be diagnosed by careful examination of the coat. The use of magnifying lens can aid the diagnosis.

Treatment and Control

Ivermectin has been found to be effective in the treatment of flea and lice infestations. Sulphonamide preparations have also been found to be effective in the treatment of flea dermatitis in Nigeria. Dipping or spraying with effective insecticides are commonly used in the control of these parasites. Avoidance of overcrowding, regular cleaning of houses and change of bedding can prevent the built-up of flea and louse burdens.

TICK INFESTATION

Ticks are important vectors of infectious disease of small ruminants as indicated in previous chapters but heavy infestation with ticks can also cause anaemia. Some species of ticks cause tick paralysis while others cause tick toxicosis. Intense lameness has been noted in Goats where ticks are attached around the coronary band.

There is an extensive tick fauna of small ruminants in the sub-Saharan region which cannot be covered within the scope of this handbook. However, the species of ticks which are commonly recovered from small ruminants include *R. evertsi*, *R. appendiculatus*, *R. lunulatus*, *H. truncatum*, *A. variegatum*, *A. hebraeum*, *Ixodes ricinus* and *Boophilus decoloratus*.

High humidity is favourable for the maintenance of tick populations. Wild animals act as reservoirs of ticks which can infest livestock grazing in the same areas. Different species of ticks have different predilection sites which include the ears, perianal area, scrotum and lower parts of limbs. Direct contact is the principal mode of transmission but animals can be infested by various stages of ticks which have dropped on pastures by other livestock or wild animals.

Tick Paralysis

This is a disease of man and animals caused by some species of ticks and it is characterised by an acute ascending flaccid motor paralysis. The disease may terminate fatally especially when there is paralysis of the respiratory muscles. *Ixodes ricinus*, *I. holocyclus*, *I. rubicundus* and *Ornithodoros* spp are the species of ticks commonly associated with the disease in Africa. *I. rubicundus* is a cause of Karoo paralysis, a syndrome which is common among livestock in South Africa.

Ticks secrete a toxin which acts on the motor and sensory nerves inhibiting the release of acetylcholine at the neuromuscular junctions. The released toxin has a strong affinity to peripheral nerve tissue but it also affects the cerebral and spinal cord tissues. The degree of paralysis is related to the number of ticks and the length of time the ticks have been feeding on the animal. It appears that the toxin has short half life because the affected animal recovers following removal of ticks. Recovered animals become immune for up to 8 months.

Tick Toxicosis

Tick toxicosis is a syndrome distinct from tick paralysis and is caused by toxins of some species of ticks. Tick toxicosis is designated as sweating sickness of goats, sheep and cattle in East, Central and South Africa. *H. truncatum* is the main species of ticks responsible for tick toxicosis. *Hyalomma rufipes* has also been incriminated. In South Africa, *R. appendiculatus* has been demonstrated to produce a leucytotropic toxin.

Tick toxicosis is characterised by profuse moist eczema and hyperaemia of the mucous membranes. Young animals are most severely affected and mortality up to 70% has been reported.

Control of ticks

The main method of control of ticks is by dipping animals using recommended acaricides. In small herding units, ticks can be manually removed from the animals. Rotational grazing has also been recommended as a means of controlling tick infestation. Although burning of heavily infested pastures is practised in some countries, it is not widely recommended because of its detrimental effects on the environment. Tillage of the grazing land exposes different stages of ticks in the ground to sunlight and also buries them in deep soil layers thus hindering their development.

Oestrus Ovis Infestation

Infestation of sheep and goats with nasal bots (*Oestrus ovis*) has been reported in some sub-Saharan countries. The adult bots deposit larvae around the nostrils and the larvae migrate to the frontal sinuses and dorsal turbinates. The migrating larvae traumatise the nasal mucosa with their spiny surface inciting a nasal catarrhal inflammation. Clinical signs include sneezing, snoring respiration and mucopurulent nasal discharge. The affected animals become restless, stamp their feet and shake or press their heads against objects. Secondary bacterial infection may cause suppuration and complicate the clinical picture. At necropsy, the larvae are found in the sinuses.

Ivermectin (0.2 mg/kg), rafoxanide (7.5 mg/kg) and nitroxylin (7.5 mg/kg) are used for the treatment of *O. ovis* myiasis. Spraying with insecticides can reduce activity of the nasal adult bots.

FUNGAL INFECTIONS

Trichophyton verrucosum and *T. mentagrophytes* are the common causes of ringworm in goats and sheep in the sub-Saharan region. The importance of the disease is related to the destruction of the skin leading to losses in the leather industry. Ringworm is characterised by round, alopecic lesions which expand in a ringed fashion. The lesions may be only localised to few parts but may also spread and cover the entire body. Spontaneous regression of the lesions occur in 4-5 weeks. Fungal spores or hyphae can be demonstrated in skin scrapings and the organisms can be isolated by culturing in Sabouraud's medium. Treatment is not normally undertaken because spontaneous recovery occurs but commercial anti-fungal preparations may be used when circumstances necessitate treatment.

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CHAPTER 8 MALNUTRITION

The productivity of animals depends on the type and amount of nutrients which are ingested, digested, absorbed and utilised by the body for various physiological and anatomical functions. Carbohydrates, proteins, fats, vitamins, minerals and water are the dietary components required for proper functioning of the animal's body. Inadequate or excessive intake of these nutrients can collectively be termed as malnutrition. However, the commonest form of malnutrition small ruminants and indeed, other livestock in sub-Saharan Africa is inadequate supply or deficiency of nutrients. The majority of small ruminants are under the extensive systems depending entirely on natural forage. The amount of forage available is greatly influenced by soil types and climate and, in most countries, there is a tendency for natural forage to be abundant during the rainy season and scarce during the dry season. As a result, malnutrition is a serious problem in the small-ruminants sector. As indicated elsewhere in the handbook, even in the presence of adequate natural forage during the rainy season, other systems of grazing such as tethering and zero-grazing restrict feeding of animals resulting in poor productivity.

The susceptibility or resistance of animals to infections is influenced by their nutritional status. Undernourished animals tend to be more susceptible to diseases than well fed animals. The occurrence of diseases such as helminthosis, coccidiosis, trypanosomosis, anaplasmosis, babesiosis and ectoparasitic infestations is precipitated by poor nutrition of the animals. Studies have indicated that well-fed animals can withstand heavy helminth burdens than poorly fed animals. Pregnancy toxemia of ewes and does occurs as a result of inadequate nutrition in late pregnancy. It therefore important that, animals should be well-fed if production is to be optimised. Supplementation of feeding especially in the agro-pastoral and agricultural systems where crop by-products are abundant can result in appreciable increase in the productivity of animals and decreased susceptibility to infections.

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