COURSE CODE: **VCM 501**

COURSE TITLE: **Food Animal Medicine**

NUMBER OF UNITS:

COURSE DURATION:

COURSE DETAILS:

COURSE DETAILS:

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COURSE CONTENT:

Diagnosis and management of diseases of cattle, sheep , goats and pigs which are of economic importance in Nigeria including *Rinderpest, Peste des petit Ruminants, foot- and – mouth Disease, Lumpy skin disease, Poxes, African Swine Fever, caseous lymphadenitis, streptothricosis, Contagious Bovine Pleuropneumonia, Contagious Caprine Pleuropneumonia, Anthrax, Haemorrhagic Septicaemia, Tuberculosis, Black Quarter, Brucellosis, Enterotoxaemias, Ectoparasitism, Parasitic Gastroenteritis, Fasciolosis, Trypanosomosis, Babesisosis, Cowdriosis, Nutritional Diseases, Metabolic/Production diseases, Poisonings.*

COURSE REQUIREMENTS:

READING LIST:

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LECTURE NOTES

**Viral Diseases of Food animals**

**PESTE DES PETITS RUMINANTS (PPR)**

This is a highly contagious and infectious viral disease of domestic and wild small ruminants.

**Aetiology**: The virus which causes PPR is called Peste des petits ruminants Virus (PPRV) which belong to the morbilli virus group of the paramyxovirus family. Other members of this group which are closely related to PPR include: Rinderpest virus of cattle and buffaloes, Measles virus of humans, Distemper virus of dogs and some wild carnivores and Morbilli virus of aquatic mammals. Genetic characterization of PPR virus strains has recognised 4 groups (3 groups from Africa and 1 from Asia. The epidemiological significance of these grouping is not clear at present.

**Geographical distribution**: PPR infection has been recognised in many African countries especially those that lie between the Atlantic Ocean and the red sea. The affected areas extend North of Egypt and South to Kenya in the East and Gabon in the West. The disease is not recognised in most Northern and Southern African countries. Recently the disease has been reported in the near East and the Arabian peninsula in countries that include; Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirate and Yemen. Outbreaks are also common in India, Nepal, Bangladesh and Pakistan.

**Clinical signs**: Clinical signs appear 2-6 days post natural infection with the virus (incubation period). This is followed by sudden onset of fever of between 40 and 410C. There is marked depression and the animal appears sleepy. Hair stands erect giving a bloated appearance especially in short haired breeds. After which clear watery discharges from the eyes, nose and mouth later becomes thick and yellow (mucopurulent) due to secondary Bacterial infections. This discharge causes wet chin and hairs below the eyes which becomes dry causing matting together of the eyelid, obstruction of the nose and difficulty in breathing.

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| FIGURE 1:  PPR in a goat: purulent eye and nose discharges Discharges from the nose and eyes in advanced PPR infection; the hair below the eyes is wet and there is matting together of the eyelids as well as partial blockage of the nostrils by dried-up purulent discharges. |  |

One or two days post fever, the mucous membrane of the mouth and the eye becomes very reddened and congested.

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| FIGURE 2: PPR in a goat: inflamed (reddened) eye membranes Reddening of the mucous membranes of the eye (the conjunctiva) in the early stages of infection. Note the purulent eye discharges. |  |

The oral cavity epithelial necrosis causes small pin point greyish areas to appear on the gum, dental pad, palate, lips, inner aspect of the cheek and the upper surface of the tongue. These areas increase in number and size and join together. The lining of the mouth becomes pale and coated with dead cells. Underneath this dead surface cells are shallow erosions. Gentle rubbing across the gum and palate with a finger may yield a foul smelling material containing shreds of epithelial tissues. Similar lesions may be seen in the mucous membrane of the nose, vulva and vagina.

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| FIGURE 3: PPR in a goat: early mouth lesions showing areas of dead cells Early pale, grey areas of dead cells on the gums. |  |

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| FIGURE 4: PPR in a goat: later mouth lesions The membrane lining the mouth is completely obscured by a thick cheesy material; shallow erosions are found underneath the dead surface cells. |  |

Affected animals resist attempt to open their mouth because of the lesion and associated pains. This results in refusal of food and water.

Diarrhoea appears 2 to 3 days after fever, although in early or mild cases, it may not be obvious. Faeces initially are soft, watery, foul smelling and contain blood streaks and pieces of dead gut tissues.

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| FIGURE 5: PPR in a goat: signs of diarrhoea The hindquarters are soiled with liquid faeces. |  |

Affected sheep and goats breathe fast, thus exhibiting rocking movements with both the chest and the abdominal wall moving as the animal breaths.

Severely affected animal show difficulty and noisy breathing marked by extension of the head and neck, dilation of the nostrils, protrusion of the tongue and soft painful cough. Up to 100% of the animals in a flock may be affected in a PPR outbreak with between 20 and 90% mortality.

**Differential diagnosis:** Contagious Ecthyma (ORF): No diarrhoea in ORF, whereas in PPR, there is diarrhoea and ocular discharges. Pasteurellosis caused by *Mannhemia haemolytica*: No diarrhoea, no oral lesions and the number of goats affected are very low. Coccidiosis: No oral lesions, no coughing and mortality is very low. *Contagious caprine pleuropneumonia* (CCPP): No oral lesions and no diarrhoea.

**DIAGNOSIS**

A tentative diagnosis of PPR can be made on epidemiological and clinical features. In the event of history, oral discharges, diarrhoea, deaths with prominent breathing problems in a sheep and goat flock, no history of contact with cattle and most affected animals in the flock are adolescents; a suspicion of PPR may be made.

**LABORATORY CONFIRMATION**

Viral isolation from blood, lymph nodes around the lungs (mediastinal lymph nodes), spleen and alimentary tract lymph node (mesenteric lymph nodes). Detection of viral antigen by Agar gel immune-diffusion test (AGIDT) is a simple test but this test does not discriminate between PPR and Rinderpest; therefore a further test is required. Viral antigen can be detected by ELISA technique very rapidly. This is sensitive and differentiates between PPR and Rinderpest.

**POST MORTEM LESIONS**

Erosions on the gum, soft palate, tongue, cheek, and oesophagus. Lips showed erosion with possibly scabs and nodules in later cases. Lungs shows dark red purple areas, firm to touch mainly in the anterior and caudal lobes of the lungs (evidence of pneumonia).

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| FIGURE 6: PPR in a goat: the early lesions of pneumonia Note the small, red, solid areas of lung tissue caused directly by PPR virus infection. |  |

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| FIGURE 7: PPR in a sheep: advanced pneumonia Note the extensive, dark red/purple areas, firm to the touch, in the anterior and cardiac lobes of the lungs. Although such pneumonia is commonly seen in PPR, it is caused by secondary bacterial infection, most commonly *Mannheimia* haemolytica. These lesions are typical of pneumonic pasteurellosis. |  |

Small intestine congested with reddened lining haemorrhages and some erosions.

Large intestines (caecum, colon, and rectum) small red haemorrhages along the folds of the lining, joining together over time and becoming darker termed “Zebra Stripes”

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| FIGURE 8: PPR in a goat: "zebra striping" in the large intestine Note the lines of haemorrhage along the tips of the folds of the lining of the caecum and colon. Later, the individual haemorrhages join up and, after death, turn black. |  |

**Treatment**: Antibiotic treatment to prevent secondary bacterial infection: Penicillin -Streptomycin, Enrofloxacin

**Control**: CONTROL. Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Until recently, the most practical vaccination against PPR made use of tissue culture Rinderpest vaccine. Recently, a homologous PPR vaccine has been developed and the vaccine is available in NVRI Vom.

The appearance of clinical PPR may be associated with any of the following:

1. History of recent movement or gathering together of sheep and/or goats of different ages with or without associated changes in housing and feeding.

2. Introduction of recently purchased animals; contact in a closed/village flock with sheep and/or goats that had been sent to market but returned unsold.

3. Change in weather such as the onset of the rainy season (hot and humid) or dry, cold periods (for example the harmattan season in West Africa); contact with trade or nomadic animals through shared grazing, water and/or housing.

4. A change in husbandry (e.g. towards increased intensification) and trading practices.

5. In endemic areas, most of the sick and severely affected animals are over four months and up to 18 to 24 months of age.

**NECESSARY PRECAUTIONARY MEASURES TO TAKE TO PREVENT OUTBREAK OF PPR BY A FARMER ESTABLISHING A GOAT OR SHEEP FARM:**

1. Prior to acquisition of animals, a suitable house must be provided to prevent exposure of these animals to adverse weather conditions especially extreme cold / Harmattan.

2. Avoid buying of new stock of sheep/ goats from open market where there are congregation of different animals both the sick and healthy ones. Close contact in the market aid easy transfer of disease from the sick to healthy ones.

3. It is recommended to get new stock from small holding goat farmers who have adequate information and history of their animals.

4. Gradual change of feed from old to new feed.

5. Acquisition of animals should be done in batches.

6. From the first day of arrival on the farm, animals should be placed on Antibiotic and multivitamins to reduce the stress of change of environment, after which PPR homologous vaccine should be administered to all apparently healthy animals.

**RINDERPEST**

Cattle plague, also known as Rinderpest, is a contagious disease that principally affects cattle, but occasionally can also affect sheep, goats, camels, certain wild ruminants and pigs. The disease is characterized by severe inflammation and necrosis of the mucous membrane of the digestive tract. The disease has been associated with high mortality and it is an OIE (Office International des Epizooties also known as World Organization for Animal Health) Class A disease reflecting its serious economic impact.

**AETIOLOGY**: The rinderpest virus (RPV) is a RNA Morbilli virus, closely related to the PPR, Measles and Canine distemper viruses. Despite its extreme virulence, the virus is particularly fragile and is quickly inactivated by heat, desiccation and sunlight.

**CLINICAL SIGNS**: Mortality rates during outbreaks are usually extremely high, approaching 100% in immunologically naive populations. Initial symptoms include fever, loss of appetite, and nasal and eye discharges. Subsequently, irregular erosions appear in the mouth, the lining of the nose and the genital tract. Acute diarrhoea, preceded by constipation, is a common feature as well. Most animals die 6 to 12 days after the onset of these clinical signs.

**TRANSMISSION**: The disease is mainly spread by direct contact and by drinking contaminated water, although it can also be transmitted by air.

**Clinical Signs**: The temperature rises in the early stages. The animal is off its food, dull and the coat is starry. Sometimes shivering is noticed. The breathing is quick: a watery or mucous discharge flows from the eyes and nostrils; in the latter case there may be a slight amount of blood in the discharge. In milking cows, the secretion of milk is diminished or arrested. The membrane of the nostrils reddens, and an eruption, like grains of bran, appears in the nostrils and inside the lips and cheeks. This eruption is often followed by distinct ulceration. The animal is at first constipated, but in the later stages diarrhoea often sets in. In this case the faeces has a foul smell and is often tinged with blood. The animal rapidly loses condition and the disease usually terminates fatally in from 6 to 10 days. Cattle plague does not attack single animals in a herd, but spreads rapidly from one to another.

Rinderpest: Photos of clinical signs

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|  | Clinical signs of Rinderpest: Purulent ocular discharge and conjunctivitis |
|  | Clinical signs of Rinderpest: Erosions of buccal mucosa and gingival |
|  | Clinical signs of Rinderpest: Erosions on ventral surface of tongue. |

**DIAGNOSIS**

This is based tentatively on the clinical signs and post mortem lesions. Rising antibody titre in paired sera, serology neutralization, CFT, Agar gel diffusion tests and isolation of virus from body discharges and excretion have been used to confirm the disease.

**POST MORTEM LESIONS**: The principal lesions in Rinderpest are in the alimentary tract. Small necrotic areas, which later develop into sharply defined deep ulcers, occur in the mouth, pharynx and oesophagus. These ulcers later coalesce to form large erosions. Similar lesions and also numerous small haemorrhages occur in the mucosa of the abomasum. Zones of intense inflammation are found in the large intestine. Typically, these are arranged transversely giving a striped appearance “Zebra stripes”. All lymph nodes are severely congested and dark red in colour.

**TREATMENT AND CONTROL**: No specific treatment but supportive therapy with antibiotics and fluids may reduce mortality. Annual vaccination with the Tissue culture rinderpest vaccine (TCRV). Control of outbreaks by quarantine and ring vaccination.

**FOOT AND MOUTH DISEASE**

This is an acute, highly contagious viral disease of cloven footed animals but most important in cattle and pigs. The disease is enzootic in NIGERIA. However it has been endemic in EUROPE in the last 1 to 3 years.

**AETIOLOGY**: Apthovirus of the Picona virus group. Seven serotypes of the virus has been identified and this include the following: A, O, C, SAT 1, SAT 2, SAT 3 as well as ASIA type 1 serotype. However these serotypes further comprises of over 50 sub types. Recently six new strains have been discovered in recent outbreaks in Europe.

**TRANSMISSION**: The major route of transmission of FMD is through aerosol. Other routes include contact with infective material or discharges, through carrier animals, by ectoparasites, experimentally by artificial insemination and use of FMD infected carcases as meat scraps in pig farming.

**CLINICAL SIGNS**: Initial lesions are seen on the lingual mucosa which later develops into vesicles. Dullness, anorexia and pyrexia may precede the appearance of the vesicles. Vesicles extends to the nares, buccal cavity and between the hooves which result in lameness especially in pigs. Hoof deformities may cause a persistent lameness even after remission of other signs. Drooling of saliva as a result of lesions in the oral cavity may be seen. Pregnant animals may abort and mortality is often high among calves and piglets. Mortality may be up to 100% especially in calves but rarely exceeds 1% in adults. Mammary gland involvement may lead to mastitis. In young calves, there is involvement of the heart leading to abnormal heart sounds.

**DIAGNOSIS**: History, clinical signs and PM lesions will give a tentative diagnosis. Virus neutralization, Agar gel precipitation and ELISA will have to be undertaken to confirm the tentative diagnosis.

**POST MORTEM:** Heavy presence of blisters or vesicles in the oral cavity which extends to the lips, nostrils, muzzles, teats, snout, hooves and inter digital spaces. In calves there are degenerative changes in the heart muscles.

**MANAGEMENT AND CONTROL**: In FMD free counties, infected animals are slaughtered and carcasses burnt or buried with Calcium oxide and whole premises decontaminated. Antibiotic therapy both systemic and topical for local lesions on the legs and oral cavity to minimize secondary complications. Quarantine, vaccination and subsequent release of in contact animal are undertaken, however such animal serve as carrier for the disease.

**Bovine Viral Diarrhoea and Mucosal Disease Complex**

Bovine viral diarrhoea (BVD) is most common in young cattle (6-24 mo old) and generally is accompanied by typical mucosal lesions; it must be distinguished from other viral diseases that produce diarrhoea and mucosal lesions. These include malignant catarrhal fever and Rinderpest

**ETIOLOGY**

Bovine viral diarrhea virus (BVDV), the causal agent of BVD and mucosal disease complex, is classified in the genus Pestivirus in the family Flaviviridae. Although cattle are the primary host for BVDV, several reports suggest most even-toed ungulates are also susceptible. Isolates of BVDV are separated into noncytopathic and cytopathic biotypes based on cytopathic effects observed in infected cell cultures. Noncytopathic BVDV are the predominant viral biotype in nature. Cytopathic BVDV are relatively rare and arise in cattle that are persistently infected with noncytopathic BVDV

Cattle that are persistently infected with noncytopathic BVDV serve as a natural reservoir for virus. Persistent infection develops when noncytopathic BVDV is transmitted transplacentally during the first 4 month of fetal development. The calf is born infected with virus, remains infected for life, and usually is immune-tolerant to the resident noncytopathic virus. Transplacental infection that occurs later in gestation results in abortion, congenital malformations, or birth of normal calves that have antibody against BVDV. The prevalence of persistent infection varies among countries and between regions within a country. In some areas, the prevalence of persistent infection in calves may be as high as 1-2% of cattle <1 yr of age.

On a given farm, persistently infected cattle are often found in cohorts of animals that are approximately the same age. Persistently infected cattle can shed large amounts of BVDV in their secretions and excretions and readily transmit virus to susceptible herd mates. Clinical disease and reproductive failure often are seen after healthy cattle come in contact with a persistently infected animal. Biting insects, fomites, semen, biologic products, and possibly wild ruminants also can spread BVDV. Disease induced by BVDV varies in severity, duration, and organ systems involved. Acute disease results from infection of susceptible cattle with either noncytopathic or cytopathic BVDV.

**CLINCAL SIGNS**

Acute BVD, also termed transient BVD, often is an inapparent to mild disease of high morbidity and low mortality. Biphasic fever (~104°F [40°C]), depression, decreased milk production, transient inappetence, rapid respiration, excessive nasal secretion, excessive lacrimation, and diarrhea are typical signs of acute BVD. Clinical signs of disease usually are seen 6-12 days after infection and last 1-3 days. Transient leukopenia may be seen with onset of signs of disease. Recovery is rapid and coincides with production of viral neutralizing antibody. Gross lesions seldom are seen in cases of mild disease. Lymphoid tissue is a primary target for replication of BVDV, which may lead to immunosuppression and enhanced severity of intercurrent infections.

Some isolates of BVDV induce clinically severe disease that manifests as high fever (~107°F [41-42°C]), oral ulcerations, eruptive lesions of the coronary band and interdigital cleft, diarrhea, dehydration, leukopenia, and thrombocytopenia. In thrombocytopenic cattle, petechial haemorrhages may be seen in the conjunctiva, sclera, nictitating membrane of the eyes; and on mucosal surfaces of the mouth and vulva. Prolonged bleeding from injection sites also occurs. Swollen lymph nodes, erosions and ulcerations of the GI tract, petechial and ecchymotic haemorrhages on the serosa surfaces of the viscera, and extensive lymphoid depletion are associated with severe forms of acute BVD. The duration of overt disease may be 3-7 days. High morbidity with moderate mortality is common. Severity of acute BVD is related to the virulence of the viral strain infecting the animal and does not depend on viral biotype or genotype.

In pregnant cattle, BVDV may cross the placental barrier and infect the fetus. The consequences of fetal infection usually are seen several weeks to months after infection of the dam and depend on the stage of fetal development and on the strain of BVDV. Infection of the dam near the time of fertilization may result in reduced conception rates. Infection during the first 4 months of fetal development may lead to embryonic resorption, abortion, growth retardation, or persistent infection. Congenital malformations of the eye and CNS result from fetal infections that occur between months 4-6 of development. Fetal mummification, premature birth, stillbirth, and birth of weak calves also are seen after fetal infection.

**DIAGNOSIS**

BVD is diagnosed tentatively from disease history, clinical signs, and gross and microscopic lesions. Diagnostic laboratory support is required when clinical signs and gross lesions are minimal.

Laboratory tests for BVDV include virus isolation and assays that detect antibody in serum or detect viral RNA or viral antigen in clinical specimens and tissues. Because antibody against BVDV is prevalent in most cattle populations, a single serologic test is seldom sufficient for diagnosis. A >4-fold increase in antibody titer in paired serum samples obtained 2 more weeks apart is necessary to verify recent infection. Isolation of BVDV from blood, nasal swab specimens, or tissues confirms active infection. Identification of persistent infection requires detection of virus in clinical specimens obtained at least 3 wk apart. At necropsy, tissues of choice for viral isolation include spleen, lymph node, and ulcerated segments of the GI tract.

**DDX**

Laboratory support also is required in some outbreaks of mucosal disease or clinically severe acute BVD because either disease may appear similar to rinderpest, FMD and malignant catarrhal fever. These include malignant catarrhal fever which is sporadic disease in more matured cattle and rinderpest can be seen in outbreaks form but is exotic in most countries

**TREATMENT**

Treatment of BVD is limited primarily to supportive therapy. Control is based on sound management practices that include use of biosecurity measures, elimination of persistently infected cattle, and vaccination. Replacement cattle should be tested for persistent infection before entry into the herd. Quarantine or physical separation of replacement cattle from the resident herd for 2-4 wk should be considered, and vaccination of replacement cattle for BVD should be done before commingling with the resident herd. Embryo donors and recipients also should be tested for persistent infection. If vaccination of embryo donors or recipients is warranted, it should be done at least 1 estrous cycle before embryo transfer is performed. Because BVDV is shed into semen, breeding bulls should be tested for persistent infection before use. Artificial insemination should be done only with semen obtained from bulls free of persistent infection.

**SHEEP POX AND GOAT POX**

**Definition**

Sheep and goat pox are contagious viral diseases of small ruminants. These diseases may be mild in indigenous breeds living in endemic areas, but are often fatal in newly introduced animals.

**Importance**

Economic losses result from decreased milk production, damage to the quality of hides and wool, and other production losses. Sheep and goat pox can limit trade and prevent the development of intensive livestock production. They may also prevent new breeds of sheep or goats from being imported into endemic regions.

**Etiology**

Sheep pox and goat pox result from infection by sheep-pox virus (SPV) or goat-pox virus (GPV), closely related members of the *Capripox* genus in the family Pox-viridae. Most isolates are host specific, with SPV mainly causing disease in sheep and GPV predominantly affecting goats. However, some isolates can cause serious disease in both species. SPV and GPV are closely related to the virus that causes lumpy skin disease in cattle (LSDV).

**Species Affected**

Sheep and goat capripox viruses cause disease only in these two species. Many SPV isolates are specific for sheep, and many GPV strains are specific for goats, but some strains of these viruses readily affect both species. Infections have not been reported in wild ungulates.

**Geographic Distribution**

Sheep pox and goat pox are found in parts of Africa and Asia, the Middle East, and most of the Indian subcontinent.

**Transmission**

SPV and GPV are often transmitted by aerosol route, close contact and flies. These viruses can remain infectious for up to six months in shaded sheep pens. They may also be found on the wool or hair for as long as three months after infection, and possibly longer in scabs.

**Clinical Signs**

The incubation period varies from four to 21 days, but it is usually 1 to 2 weeks. Clinical signs generally appear sooner when the virus is inoculated by insects than when it is transmitted in aerosols. The clinical signs vary from mild to severe, depending on the animal’s age, breed, immunity and other factors. Inapparent infections also occur. Clinical signs in sheep and goats are similar but generally less severe in goats. Fever, swollen eyelids, mucopurulent nasal discharges, widespread skin lesions especially on the muzzle, ears, areas free of wool or long hair. Lesions start as erythematous areas on the skin and progress rapidly to raised, circular plaques with congested borders caused by local inflammation, edema and epithelial hyperplasia. When scabs are removed, a star shaped scar, freeof hair or wool remains. In severe cases, lesions can develop in the lungs. All superficial lymph nodes usually become enlarged within a day of the appearance of generalized papules; the prescapular lymph nodes are particularly noticeable.

Nodules in the intestines can cause diarrhea. Depression and emaciation may be seen in some animals. Abortions can occur but are not common.

Capripox lesions can take several weeks to heal, and may leave permanent scars on the skin. During healing, they are susceptible to fly strike. Secondary bacterial infections, including pneumonia, are common, and death can occur at any stage of the disease. Recovery can be slow if the animal was severely affected. Infection results in solid and enduring immunity.

Capripox lesions have a predilection for areas of sparsely wooled/ haired skin such as the axillae, muzzle, eyelids, ears, mammary gland and inguinal area, but in more severe cases, they may cover the body. In animals with heavy wool, the lesions can be easier to find by palpation than

**Morbidity and Mortality**

Morbidity and mortality vary with the breed of the animal, its immunity to capripox viruses, and the strain of the virus. Mild infections are common among indigenous breeds in endemic areas, but more severe disease can be seen in young or stressed animals, animals with concur-rent infections, or animals from areas where pox has not occurred for some time. Reported morbidity rates in indigenous breeds range from 1% to 75% or higher. Although the mortality rate is often less than 10%, case fatality rates of nearly 100% have been reported in some young animals.

Imported breeds of sheep and goats usually develop severe disease when they are moved into an endemic area. The morbidity and mortality rates can approach 100% in newly imported, highly susceptible flocks.

**Diagnosis**

**Clinical**

Sheep or goat pox should be suspected in febrile animals with the characteristic full–thickness skin lesions and enlarged lymph nodes. Dyspnea, conjunctivitis, nasal discharges and other signs may also be seen. The mortality rate is usually high in naïve animals. Although sheep pox and goat pox are usually distinctive in fully susceptible animals, these diseases can be subtler and more difficult to diagnose in indigenous animals.

**Laboratory tests**

* Electron microscopy: because the morphology of the virus particle is characteristic, capripox viruses can be differentiated from most poxviruses that cause lesions in small ruminants.
* Histopathology can also be helpful.
* Serological tests include virus neutralization, AGID, the indirect fluorescent antibody test (IFA), ELISAs and immunoblotting (Western blotting).
* Polymerase chain reaction (PCR) assays: this can detect capripox-virus genomes in tissue samples or cultures, but cannot identify whether the virus is SPV or GPV.

**Differential diagnosis**

The differential diagnoses include contagious ecthyma (contagious pustular dermatitis), dermatophilosis/ streptothricosis, mange (e.g., psoroptic mange/sheep scab), photosensitization or urticaria, peste des petits ruminants, parasitic pneumonia, multiple insect bites and caseous lymphadenitis.

**Samples to collect**

In live animals, biopsies of skin lesions should be taken for virus isolation and antigen detection. SPV and GPV can also be found in vesicular fluid, scabs and scrapings of skin lesions, as well as lymph node aspirates and blood (collected into heparin or EDTA). At necropsy, samples should be collected from skin lesions, lymph nodes and lung lesions. An additional set of samples should be taken for histology; these samples should include a wide range of lesions from the skin, as well as spleen, rumen, trachea, lungs and other affected tissues. PCR can detect capripox-viruses in blood, nasal or oral swabs, scabs, skin lesions and tissue samples. Neutraliz-ing antibodies can interfere with virus isolation and some antigen-detection tests; samples for these tests must be collected during the first week of illness. Samples for PCR can be taken after neutralizing antibodies have developed. Paired serum samples should be collected for serology.

Samples for virus isolation must be sent to the laboratory as soon as possible. They should be kept cold and shipped on wet ice or gel packs. If these samples must be shipped long distances without refrigeration, glycerol (10%) can be added; the tissue samples must be large enough that the medium does not penetrate into the center of the tissue and destroy the viruses there.

**Treatment**

No specific treatment is advised, but palliative treatment may be necessary in severely affected animals.

**Control**

* Prohibition of importation from infected areas (for countries that are free)
* In outbreaks, control is by quarantines, movement restriction, and depopulation of infected and exposed animals, followed by stringent cleaning and disinfection of farms and equipment. Proper disposal of infected carcasses is important; burning or burial is often used.
* Live, attenuated virus vaccine and live, attenuated Lumpy skin disease virus can be used.

**Public Health**

SPV and GPV do not infect humans.

**LUMPY SKIN DISEASE**

**IMPORTANCE**

Lumpy skin disease is a pox-viral disease with significant morbidity in cattle. Although the mortality rate is generally low, losses occur from decreased milk production, abortion, infertility, loss of condition and damaged hides. Lumpy skin disease is endemic in parts of Africa, where outbreaks may be widespread. This disease has the potential to become established in other parts of the world.

**AETIOLOGY**

LSD is caused by a virus in the genus *Capripoxvirus* of the family *Poxviridae*. Lumpy Skin Disease Virus (LSDV) IS closely related antigenically to sheep and goat poxviruses. Although these three viruses are distinct, they cannot be differentiated with routine serological tests.

**EPIDEMIOLOGY**

• Morbidity rate varies between 5 and 45%

• Mortality rate up to 10%.

**Hosts**

• LSD is primarily a disease of cattle. *Bos Taurus* breeds, particularly Jersey, are more susceptible to clinical disease than zebus cattle (*Bos indicus)*.

**Transmission**

• The principal method of transmission is mechanical by arthropod vector. Though no specific vector has been identified to date, mosquitoes (e.g. *Culex mirificens* and *Aedes*

*natrionus*) and flies (e.g. *Stomoxys calcitrans* and *Biomyia fasciata*) could play a major role.

• Direct contact could be a minor source of infection.

• Transmission may also occur by ingestion of feed and water contaminated with infected saliva.

• Animals can be infected experimentally by inoculation with material from cutaneous nodules or blood.

**Sources of virus**

• Skin; cutaneous lesions and crusts. Virus can be isolated for up to 35 days and viral nucleic acid can be demonstrated by PCR for up to 3 months.

• Saliva, ocular and nasal discharge, milk, and semen. All secretions contain LSD virus when nodules on the mucous membranes of the eyes, nose, mouth, rectum, udder and genitalia ulcerate. Shedding in semen may be prolonged; viral DNA has been found in the semen of some bulls for at least 5 months after infection. In experimentally infected cattle LSD virus was demonstrated in saliva for 11 days, semen for 22 days and in skin nodules for 33 days, but not in urine or faeces. Viraemia lasts approximately 1–2 weeks.

• Lung tissue

• Spleen

• Lymph nodes

• No carrier state

**Occurrence**

In the past LSD was restricted to sub-Saharan Africa but currently it occurs in most African countries. The most recent outbreaks outside Africa occurred in the Middle East 2006 and 2007 and in Mauritius 2008.

**CLINICAL SIGNS**

The incubation period under field conditions has not been reported. Following inoculation the onset of fever is in 6–9 days, and first skin lesions appear at the inoculation site in 4–20 days.

LSD signs range from inapparent to severe disease.

• Pyrexia which may exceed 41°C and persist for 1 week.

• Rhinitis, conjunctivitis and excessive salivation.

• Marked reduction in milk yield in lactating cattle.

• Painful nodules of 2–5 cm in diameter develop over the entire body, particularly on the head, neck, udder and perineum between 7 and 19 days after virus inoculation.

Although the nodules may exude serum initially, they develop a characteristic inverted conical zone of necrosis, which penetrates the epidermis and dermis, subcutaneous tissue, and sometimes the underlying muscle. Over the following 2 weeks they may become necrotic plugs that penetrate the full thickness of the hide/skin and are called “sit-fasts”.

• Pox lesions may develop in the mucous membranes of the mouth and alimentary tract and, in trachea and lungs, resulting in primary and secondary pneumonia.

• Depression, anorexia, agalactia and emaciation.

• All the superficial lymph nodes are enlarged and edematous

• Limbs may be oedematous and the animal is reluctant to move.

• Nodules on the mucous membranes of the eyes, nose, mouth, rectum, udder and genitalia quickly ulcerate, and all secretions contain LSD virus.

• Discharge from the eyes and nose becomes mucopurulent, and keratitis may develop.

• Pregnant cattle may abort.

• Bulls may become permanently or temporarily infertile from orchitis and testicular atrophy, and the virus can be excreted in the semen for prolonged periods.

Temporary sterility in cows may also occur.

• Recovery from severe infection is slow due to emaciation, pneumonia, mastitis, and necrotic skin plugs, which are subject to fly strike and shed leaving deep holes in the hide.

**Lesions**

• Nodules involving all layers of skin, subcutaneous tissue, and often adjacent musculature, with congestion, haemorrhage, oedema, vasculitis and necrosis

• Enlargement of lymph nodes draining affected areas with lymphoid proliferation, oedema, congestion and haemorrhage

• Pox lesions of mucous membrane of the mouth, the pharynx, epiglottis, tongue and throughout the digestive tract

• Pox lesions of the mucous membranes of the nasal cavity, trachea and lungs

• Oedema and areas of focal lobular atelectasis in lungs

• Pleuritis with enlargement of the mediastinal lymph nodes in severe cases

• Synovitis and tendosynovitis with fibrin in the synovial fluid

• Pox lesions may be present in the testicles and urinary bladder.

**Differential diagnosis**

• Severe LSD is highly characteristic, but milder forms can be confused with those below.

• Pseudo lumpy skin disease/ Bovine herpes mammillitis (Bovine Herpesvirus 2)

• Bovine papular stomatitis (Parapoxvirus)

• Pseudocowpox (Parapoxvirus)

• Vaccinia virus and Cowpox virus (Orthopoxviruses) – uncommon and not generalized infections

• Dermatophilosis

• Insect or tick bites

• Besnoitiosis

• Demodicosis

• *Hypoderma bovis* infection

• Photosensitisation

• Urticaria

• Cutaneous tuberculosis

**Laboratory diagnosis**

Virus isolation and identification (using samples of lesions, including tissues from surrounding areas) can be carried out by

* ELISA
* PCR assays
* Histopathology

Serological tests (using frozen sera from both acute and convalescent animals) can also be carried out:

• Virus neutralisation

• Indirect fluorescent antibody test

• Capripox antibody ELISA.

• Western blot: highly sensitive and specific but expensive and difficult to perform.

**TREATMENT**

No specific treatment. Strong antibiotic therapy may avoid secondary infection.

**PREVENTION AND CONTROL**

In free countries: import restrictions on livestock, carcasses, hides, skins and semen.

In infected countries:

* strict quarantine to avoid introduction of infected animals into safe herds
* in cases of outbreaks, isolation and prohibition of animal movements
* slaughtering of all sick and infected animals (as far as possible)
* proper disposal of dead animals (e.g. incineration)
* cleaning and disinfection of premises and implements
* vector control in premises and on animals
* vaccination:

1. Homologous live attenuated virus vaccine:

Neethling strain: immunity conferred lasts up to 3 years

2. Heterologous live attenuated virus vaccine:

Sheep or goat pox vaccine, but may cause local, sometimes severe, reactions

Note: not advised in countries free from sheep and goat pox.

With the exception of vaccination, control measures are usually not effective.

Vector control in ships and aircraft is highly recommended.

**INFECTIOUS KERATITIS, INFECTIOUS BOVINE KERATOCONJUNCTIVITIS (IBK) OR (PINK EYE)**

**Introduction**

Infectious keratitis (pinkeye) is a highly contagious disease causing inflammation of the cornea (the clear outer layer) and conjunctiva (the pink membrane lining the eyelids) of the eye. It is also associated with ulceration of the cornea and potential loss of an eye.

**Aetiology**: *Moraxella bovis* causes IBK, an important ocular disease of cattle,

**Epidemiology**: It occurs worldwide and affects cattle of all ages and breeds. The disease is seasonal in nature.

**Transmission**: The disease is transmitted by direct contact, aerosols, and fomites. Flies may serve as mechanical vectors of the bacteria. . Carrier animals are animals that show no signs of clinical disease but shed the bacteria in their secretions. Carrier animals may shed the organism for long periods of time so they are an important factor in the spread of the disease and its survival over winter.

**Economic importance**: The disease causes economic losses arising from decreased weight gain in beef breeds, loss of milk production, short-term disruption of breeding programs, and treatment costs. Affected animals may also bring significantly discounted prices when sold. Animals which are blind in both eyes are at risk of death through accidents or starvation and they are also a significant animal welfare concern.

**Pathogenesis:** The primary infectious agent for pinkeye is the bacterium *Moraxella bovis*. This bacterium is found in the eyes of many recovered and apparently normal cattle. Pinkeye is a multifactorial disease, which means there are many factors that predispose and contribute to the development of the disease.

Eye irritation is necessary for the development of the disease. Face flies feed around the eyes and nostrils of cattle, causing a mechanical irritation to the eye and spreading the disease from one animal to another. The bacteria can survive on the flies for up to four days, allowing that fly to infect numerous animals.

Other sources of eye irritation which predispose the animal to disease are tall weeds and grasses; feed and dust,. Dust on windy days, and exposure to excessive UV sunlight also increase the chances of disease development.

**Clinical signs**: include excessive watery lacrimation, blepharospasm, photophobia, corneal ulceration, opacity, and in some cases, a slight to moderate fever with fall in milk yield and depression of appetite.

There are four stages of pinkeye. The disease may resolve at any stage while animals that receive no treatment will often progress through all four stages.

* **Stage I:** Cattle have excessive tearing and increased sensitivity to light. They will blink frequently and there is redness along the eyelids.
  + **Stage II:** The clinical signs described in Stage I continue, but an ulcer spreads across the cornea.
  + **Stage III:** The ulcer covers most of the cornea and the inflammation continues to spread into the inner parts of the eye.
  + **Stage IV:** The ulcer extends completely through the cornea, and the iris. These eyes may be permanently damaged and the animal rendered blind.

**Treatment**:

* Pinkeye is frequently a self limiting disease with mild to severe clinical signs and blindness in approximately 2 percent of the cases.
* Early treatment of cattle with pinkeye is important, first for a successful outcome for the affected individual animal and then to stop the shedding of the bacteria, decreasing the risk of transmission to other cattle.
* It is based on antibiotics effective against moraxella. Antimicrobial therapy should be administered both parentally (oxytet LA) and subconjunctivally or topically early in the disease course.

**Prevention:**

* Fly control: examples are fly tags, fly traps, insecticide pour-on and knock-down sprays with insecticides. Face flies can develop resistance to pesticides over time, so switching the drug class of the pesticides used every year is important. For example, if pyrethrins are used one year, then organophosphates should be used the following year.
* Isolation and prompt treatment of affected animals. It is important to remove animals suffering from pinkeye from the herd because they serve as a reservoir for the organism.
* Appropriate grazing and pasture clipping to prevent seed-head development helps to decrease irritation to the eyes of cattle, and reduces resting areas for flies.

**ATROPHIC RHINITIS**

**Rhinitis** is an inflammation of the upper respiratory tract and is present to some degree in almost every commercial swine herd. This kind of inflammation can be caused by bacteria, viruses, chemicals (manure gas), dust, pollen, temperature fluctuations, and other irritants in the environment, and can have a negative impact on the affected pig’s feed-conversion efficiency and rate of gain.

**Atrophic rhinitis (AR)** is the term commonly used to refer to the condition of a sneezing pig with a crooked, bleeding snout and tear-stained face. The term atrophy indicates that the turbinate bones inside the snout are shrunken and distorted (as a result the tissues inside the nose become infected or damaged). These bones are lined with mucous membranes and filter the air the pig inhales, and so they are vulnerable to irritation and infection by changes and contaminants in the environment. Atrophy of the turbinate bones without external signs, like a crooked nose, is called “turbinate atrophy”.

**Aetiology**

Toxigenic strain of *Bordetella bronchioseptica* and *Pasteurella multocida* type D and type A.

**Epidemiology**

AR occurs worldwide where pigs are reared under intensive conditions. It has, however, become much less important with the onset of vaccination and hygienic farrowing house. Disease is more common in young herds particularly those containing large numbers of gilts.

**Transmission**

Direct contact (carrier sow to piglets) and droplet infection are the routes of transmission.

**Forms of AR**

There are two forms of the disease:

a) **Mild and non-progressive** where the infection or irritation occurs over a period of 2 to 3 weeks. The inflammation does not progress and structures in the nose called turbinate bones repair and return to normality. The term non-progressive atrophic rhinitis is used for the slight to severe rhinitis and usually transient turbinate atrophy in which no toxigenic P. multocida are found, where there are no clinical signs and no obvious growth retardation. Organisms such as *Bordetella bronchioseptica*, non toxigenic *Pasteurella multocida*, other environmental organisms and dust or gases can produce this type of rhinitis in the nose. The mild form is very common.

b)  **Progressive atrophic rhinitis** (PAR) where toxin producing strains of the bacterium *Pasteurella multocida*, cause a continual and progressive inflammation and atrophy of the tissues and nose distortion. For a herd however, to have PAR toxigenic pasteurella must be present. They are carried in the nose and tonsils of the adult pig and there is always the risk therefore of buying them into the herd. This is the most common method of entry. Progressive atrophic rhinitis (PAR) is a serious condition both in sucking and growing pigs.

**Economic Importance**

The economic effects of PAR may be reduced growth rate and worsened feed-conversion efficiency.

**Clinical signs**

They include sneezing, runny eyes, discharges from the nose sometimes containing blood and early signs of distortion of the face, with shortening or twisting of the upper jaw becoming evident at weaning time. There may be dyspnea in severe cases. It is important also to appreciate that sneezing is a common occurrence in the sucking pig and need not necessarily be associated with PAR. PAR affects most of the piglets present. However, individual piglets may also develop distortion of the nose from trauma or some other cause but this is not PAR.

**Diagnosis**

This is carried out by:

* The clinical signs in the sucking piglets and nasal distortion in growing pigs.
* Sectioning the snout of pigs at slaughter and examining the degree of turbinate damage in the nose.
* Isolating the organism from sucking or rhinitic pigs by swabbing the nostrils and submitting for bacteria culture.

**Differential Diagnoses**

The most common would be non progressive rhinitis and sneezing caused by cytomegalo virus, bordetella and haemophilus organisms or environmental irritants. A significant differentiating feature here is that if these organisms are causing sneezing in the sucking pigs then by 4 weeks after weaning sneezing will have disappeared with no facial distortions.

**Treatment**

* Once toxigenic pasteurella have been identified the complete breeding herd should be immediately vaccinated six weeks apart using a vaccine made from toxigenic pasteurella. It takes approximately four months for a total herd immunity to develop and it may be nine months or more before the disease is brought completely under control. In the early stages of a herd breakdown the following could be recommended:
* In-feed medicate sows with trimethoprim/sulpha or sulphadimidine from point of entry into the farrowing house through to weaning. (500g/tonne)
* Inject all piglets with 0.25 to 0.5ml of long-acting oxytetracycline or amoxycillin on days 3, 10 and 15 during sucking.
* Inject pigs similarly at weaning time with 0.5 to 1ml of long-acting antibiotic. This treatment programme should continue for a period of at least 2 months after all sows have been fully vaccinated.
* Medicate the creep rations with oxytetracycline 800g/tonne or trimethoprim/sulpha combinations for 4 weeks post-weaning.
* Sows should be given a booster dose of vaccine 2 to 3 weeks prior to each subsequent farrowing.

**Management control and prevention**

Many approaches to treatment and prevention of AR are available, but proper management of the pig’s environment is paramount to the success of any approach. Control can be attempted in at least 4 ways:

* Total eradication
* Reduction of infection pressure
* Mass medication with antimicrobials to reduce the severity and adverse effects of infection
* Vaccination.

**Eradication**

Total eradication can only be achieved with confidence by complete depopulation for a 4-week period and repopulation with primary or purchased specific-pathogen-free stock.

**Vaccination**

Vaccines containing killed *B. bronchioseptica*, *P. multocida* type D and type A, plus toxoids to *P. multocida* type D toxin or *P. multocida* types D and A toxins may be used. Vaccines containing killed bacteria and toxoids are called bacterin/ toxoids.



**DIARRHEA (SCOURS) IN SMALL RUMINANTS**

Diarrhea is defined as an increased frequency, fluidity, or volume of fecal excretion. The feaces may contain blood or mucous and be smelly. The color of the feaces may be abnormal. However, it is not possible to definitively determine the infectious organism by looking at the color, consistency, or odor of the feaces. A definitive identification requires a sample for microbiological analysis.

In livestock, diarrhea is called scours. There are many causes of diarrhea: bacterial, viral, parasites, and diet.

**DIARRHEA IN YOUNG (NEONATAL) LAMBS AND KIDS**

The four major infectious causes of diarrhea in lambs and kids during the first month of life are ***E. Coli*, rotavirus, *Cryposporidum* sp. and *Salmonella* sp.** *E. coli* scours are most common.

**Nutritional**  
Infectious agents are not the only cause of diarrhea in neonates. Nutritional problems can result in diarrhea. Nutritional diarrhea is most common in orphaned animals as a result of poor quality milk replacers, mixing errors, and overfeeding. Consumption of lush pasture or high-energy diets can also result in diarrhea in young lambs and kids.

**Treatment**

Whatever the microbial cause of scours, the most effective treatment for a scouring lamb or kid is rehydration by administering fluids.

**DIARRHEA IN OLDER LAMBS AND KIDS**

The most common causes of diarrhea in older lambs and kids are coccidiosis and gastro-intestinal parasites (worms). Other major causes of diarrhea in older lambs and kids are *clostridium perfringins*, rumen acidosis, and nutritional.

**Coccidiosis**  
Coccidosis is a protozoan parasitic disease that is a common cause of diarrhea in lambs and kids. It may also cause subclinical production losses. Lambs and kids are most suceptible to the problem at 1 to 4 months of age, although younger animals may be affected. Lambs are resistant to the disease in their first few weeks of life. Exposure to the protozoa during this time confers immunity and resistance to later infections. Clinical disease is common after the stress of weaning, feed changes, or shipping. The diarrhea of lambs and kids is usually not bloody, but it may contain blood or mucous and be very watery. Treatment of affected animals includes supportive care and adminstration of [coccidiostats](http://www.sheepandgoat.com/articles/coccidtable.html). All animals in a group should be treated during an outbreak. Prevention involves improved sanitation and the use of coccidiostats.

**Gastro-intestinal worms**  
Heavy loads of other gastro-intestinal worms can cause diarrhea in sheep and goats: *Ostertagia circumcincta* (medium or brown stomach worm), *Trichostrongylus* (bankrupt or hair worm), *Coopera sp*. (small intestinal worm), and *Nematodirus sp*. (threadneck worm).

Control of gastro-intestinal parasites is best achieved via good pasture, grazing, and animal management, and strategic and/or selective deworming of affected individuals with effective [anthelmintics](http://www.sheepandgoat.com/articles/antheltable.html).

***Clostridium perfringins*** *Clostridium perfringins* types A, B, C, and D can all cause diarrhea in lambs and kids, though type D is the most common agent. With type D, the onset of neurologic signs followed by sudden death is more common in sheep, whereas goats are more likely to show signs of diarrhea before death. Treatment is rarely effective but consists of aggressive supportive care and administration of the antitoxin.

*Clostridium perfringens* type C tends to affect very young lambs (<2 weeks of age) and presents itself as bloody diarrhea, hemorrhagic enteritis, and bloody scours. Clostridial diseases are easily prevented in the young by vaccinating pregnant dams about three weeks prior to delivery and subsequent vaccination of offspring. Consumption of adequate, high quality colostrum is important.

**Rumen Acidosis**  
Acidosis is caused by too much grain or concentrate, which causes a change in rumen acidity and bacteria population. The increase in acid causes an inflammation of the rumen wall and a reduction in the bacteria needed to digest fiber. Symptoms may include depression, off feed, bloat, founder, scours, and occasionally death. Treatment includes drenching with mineral oil or antacids. Acidosis is prevented by proper feeding management. Concentrates (grain) should be introduced to the diet slowly and increased incrementally to give time for the rumen to adjust.

**Nutritional**  
Nutritional scours can be caused by anything that disrupts normal habits. It can also be the result of low intake of dry matter to fluid ratio. A lamb needs to consume at least 2.5 percent of its body weight in dry matter daily. Young or fast growing lambs turned out to pasture must eat large quantities of grass to satisfy their nutritional needs. Green grass is high in moisture. They may develop diarrhea if they aren't getting enough dry matter in their diet.

**DIARRHEA IN ADULT SHEEP AND GOATS**

Adult-onset diarrhea is less common than in lambs and kids, but nevertheless is possible. Parasitism can cause diarrhea in adult sheep and goats. Coccidiosis can occur in adults under extreme stress or due to lack of immunity. The ingestion of toxins, of which the list is long, can also cause diarrhea. It is not uncommon for sheep or goats to scour when they are grazing lush or wet pasture.

**Treatment Strategies**

Diarrhea should not be considered an illness in and of itself but rather a symptom of other more serious health problems in sheep and goats. It can be the symptom of many different illnesses, e.g. bloat, acidosis, enterotoxemia, and polio. Diarrhea is not always the result of an infectious disease. It can be induced by stress, poor management, and nutrition.

Before treating an animal for diarrhea, it is essential to determine why the animal is scouring. Take the animal's temperature using a rectal thermometer. If body temperature is above the normal range (102-103°F), fever medications and antibiotics can be used to control the infection.

Many of the common causes of diarrhea are self-limiting, and the major goals of treatment are to keep the animal physiologically intact while the diarrhea runs its course.

A variety of oral antidiarrheal medications have been used in sheep and goats. Pepto-Bismol (Bismuth Subsalicylate, Bismusal) is commonly used to treat livestock with diarrhea. Pepto Bismol contains bismuth which coats, soothes, and relieves the irritated lining of the stomach. Kaopectate (Kaolin-Pectin) can be used to treat non-infectious causes of diarrhea. Treatment with antibiotics is usually not useful when animals are infected with viruses or protozoa. However, antibiotics are useful when bacterial infections are the primary infective agent or where the risk of secondary bacterial infections is high. Sulfa-antibiotics or amprolium should be used in the case of [](http://www.flickr.com/photos/baalands/1368749965/)coccidia. [](http://www.flickr.com/photos/baalands/4204304023/)

**DIAGNOSIS & MANAGEMENT OF BACTERIAL DISEASES**

**1. MASTITIS**

Mastitis means inflammation of the parenchyma of the mammary gland regardless of the cause. It is characterized by physical, chemical and usually bacteriological changes in the milk and by pathological changes in the glandular tissue.

The most important changes in the milk include discoloration, the presence of clots and the presence of large number of leukocytes.

Factors that predispose to infection within the gland are poor milking hygiene, milking machine faults, faulty milking management, teat injuries, teat sores and environmental population of pathogens.

**Aetiology:** Bacterial pathogens including *Staphylococcus aureus*, *Streptococcus agalactiae*, *Streptococci spp* and coliform organisms; *Corynebacterium pyogenes*, *Pseudomonas aeruginosa*, *Nocardia asteroids*, *Clostridium perfringens*, *Mycobacterium spp*, *Mycoplasma spp* and *Pasteurella spp*.

Fungal infections include *Trichosporon spp*, *Aspergillus fumigatus* and *A. nidulans*

Yeast infections include *Candida spp, Cryptococcus neoformans* and *Saccharomyces spp*

Algal infections include *Prototheca spp*.

**Clinical Findings:** The four clinical types of mastitis are:

**Per acute**: in which swelling, heat, pain and abnormal secretion in the gland are accompanied by fever and other signs of a systemic disturbance such as marked depression, rapid weak pulse, sunken eyes, weakness and complete anorexia.

**Acute**: in which changes in the gland are similar to those above, but fever and depression are slight to moderate.

**Sub acute**: in which there are no systemic changes and the changes in the gland and secretion are less marked.

**Sub clinical**: in which the inflammatory reaction within the gland is detectable only by tests.

Changes in the secretion can vary from a slight wateriness with a few flecks (as in sub-acute Staphylococcal mastitis), through watery or serous with large yellow clots (as in acute or per acute Streptococcal, Staphylococcal or Mycoplasmal mastitis), to watery and brownish with fine mealy, flakes (as in coliform or Mycoplasmal mastitis). With severe chronic mastitis, the affected gland gradually loses its productive capacity and may either atrophy or slowly develop firm nodular abscesses or granuloma-like masses within the parenchyma.

**Diagnosis:** A diagnosis of mastitis is based on clinical findings, on culture and identification of the pathogen from a sample of milk collected aseptically and on results of tests designed to detect increases in the WBC content of the milk. In clinical cases, a tentative diagnosis usually is based on signs and knowledge of the predominant pathogens in the herd, but it should be confirmed by culture and sensitivity tests.

**Treatment:** Penicillin, tetracyclines, cephalosporin, novobiocin, sodium cloxacillin, penicillin-streptomycin, neomycin, erythromycin, streptomycin, lincomycin or ampicillin by systemic treatment or intra-mammary infusion.

Yeasts grow well in the presence of penicillin and some other antibiotics. If mastitis due to yeast is suspected, antibiotic therapy should cease immediately (Signs may be severe with a high temperature, followed by a spontaneous recovery in about 2 weeks or by a chronic destructive mastitis).

**2. MASTITIS – METRITIS – AGALACTIA (MMA) SYNDROME**

The MMA syndrome occurs in sows between 12 and 48 hours after farrowing and is characterised clinically by anorexia, lethargy, disinterest in the piglets, fever, swelling of the mammary glands and agalactia.

**Aetiology:** Infectious mastitis due to *Escherichia coli* and *Klebsiella pneumoniae*. Predisposing factors include overfeeding during pregnancy, nutritional deficiencies, constipation, endocrine dysfunction and the stress of farrowing in total confinement in a crate.

**Clinical findings:** The sow is usually normal with a normal milk flow for the first 12 to 18 hours after farrowing. There is failure of the sow to suckle her piglets. The sow lies in sternal recumbency, disinterested in the piglets and unresponsive to their squealing and sucking demands (in any situation in which 2 to 3 day-old piglets are showing signs of starvation, MMA must be considered). Many piglets may die from starvation and hypoglycaemia.

Affected sows do not eat, drink very little and are generally lethargic. There is fever, varying degrees of swelling and inflammation of the mammary glands. The teas are usually empty and may be slightly oedematous. In severe cases, the milk contains flakes and pus or is watery.

A vaginal discharge is normal following parturition and normal sows frequently expel up to 50ml of viscid non-odourous clear mucus which contains variable amounts of white material within the first 3 days following farrowing.

**Diagnosis:** This is based on clinical findings, with care being taken to differentiate the condition from other specific entities that might also lead to lactation failure (such as TGE, mycotoxicosis, erysipelas, mastitis and metritis) and causes of hypoglycaemia in piglets.

**Treatment:** Most affected sows will recover within 24 to 48 hours if treated with a combination of Antimicrobials, oxytocin and corticosteroids. Broad spectrum antimicrobials daily for at least 3 days.

Use of oxytocin (30-40 units i/m or 20-30 units i/v) at 3 to 4 hour intervals helps with milk let down; and warm water massaging of the affected mammary glands with towels and hand milking for 10 to 15 minutes every few hours may assist in reducing the swelling and inflammation.

Dexamethasone at the rate of 20mg i/m daily for 3 days for sows weighing 150-200kg.

The piglets must be given a supply of milk and or balanced electrolytes and dextrose until the milk flow of the sow is resumed, which may take 2-4 days. Piglets should received 300 to 500ml of milk per day divided into hourly doses of 40-50ml through a stomach tube.

Piglets may be cross-fostered to a normal sow.

**3. NEONATAL STREPTOCOCCAL INFECTION**

Streptococcal infection of the new born is characterised by bacteriaemia, and septicaemia resulting in localisation of the infection in other organs, particularly joints, meninges and endocardium.

**Aetiology:** *Streptococcus pyogenes* (calves); *S. suis* (piglets); *S. faecalis* (lambs and kids).

The source of the infection is usually the environment which may be contaminated by uterine discharges from infected dams or by discharges from lesions in other animals.

The portal of infection in most instances appears to be the umbilicus. In older animals, the incidence of clinical disease appears to depend on environmental factors such as inadequate ventilation, high population density and other stressors.

**Clinical Findings:** Arthritis (Polyarthritis), meningitis, omphalophlebitis and ophthalmitis may occur alone or together in neonates less than 6 weeks of age. There may also be fever, anorexia and depression. The gait is stiff, piglets may stand on their toes and there is swaying of the hindquarters. Pus accumulates and the joint capsule often ruptures.

**Diagnosis:** Streptococcal infections are delayed in their onset and usually produce a polyarthritis. Arthritis due to *Mycoplasma spp.* is less suppurative but may require cultural differentiation. Glasser’s disease occurs usually in older pigs and is accompanied by pleurisy, pericarditis and peritonitis. Erysipelas in very young pigs is usually manifested by septicaemia. Meningitis in young calves may be caused by *Pasteurella multocida*. Polyarthritis in calves, lambs and piglet may also be caused by infection with *Actinomyces* (*Corynebacterium*) *pyogenes* and *Fusobacterium necrophorum*. *Streptococcus suis* can also be the cause of meningitis in older pigs of 10 to 14 weeks of age.

**Treatment:** Penicillin.

**4. EXUDATIVE EPIDERMITIS = GREASY PIG DISEASE**

Exudative epidermitis of sucking pigs is caused by *Staphylococcus spp.* and characterised by the appearance of an acute, generalised seborrhoeic dermatitis.

**Aetiology:** *Staphylococcus hyos*; *S. hyicus*

Most cases occur in animals under 6 weeks of age.

**Clinical Findings:** In per acute form which occurs most commonly in few days old piglets, there is a sudden onset of marked cutaneous erythema, with severe pain on palpation evidenced by squealing. Anorexia, severe dehydration and weakness are present and death occurs in 24 to 48 hours. The entire skin coat appears wrinkled and reddened and is covered with greasy, gray-brown exudates which accumulate in thick clumps around the eyes, behind the ears and over the abdominal wall. In the less acute form, seen in older pigs of 3 to 10 weeks of age, the greasy exudates become thickened and brown and peel off in scabs, leaving a deep pink-coloured to normal skin surface. There is no irritation or pruritus. In the sub acute form, the exudate dries into brown scales which are most prominent on the face, around the eyes and behind the ears.

**Diagnosis:** Exudative epidermitis may resemble several skin diseases of pigs of all age groups including swine pox, skin necrosis, parakeratosis, ring worm, facial dermatitis, ulcerative granuloma, sarcoptic mange, allergic dermatoses and erysipelas.

**Treatment:** Cloxacillin, penicillin, novobiocin, neomycin.

**5. CASEOUS LYMPHADENITIS OF SHEEP AND GOATS**

Caseous lymphadenitis is a chronic disease of sheep and goats characterised by the formation of abscesses in lymph nodes and exerting little effect on the general health unless the disease becomes generalised.

**Aetiology:** *Corynebacterium pseudotuberculosis*

Infection of an animal is facilitated by the presence of skin wounds but the organism can invade through intact skin.

**Clinical Findings:** There is palpable enlargement of one or more of the superficial lymph nodes: submaxillary, prescapular, prefemoral, supramammary and popliteal nodes. The abscesses commonly rupture and thick, green pus is discharged.

In cattle, ulcerative lymphangitis of the lower limbs may occur mildly.

**Diagnosis:** Palpable enlargements of peripheral lymph nodes as a flock problem in sheep are usually due to this disease. The caseous greenish pus is diagnostic.

**Treatment:** Treatment is not usually attempted. If animal value warrants, abscesses may be surgically removed or drained and cauterized using copper sulphate or iodine. Long term procaine penicillin may be beneficial.

Class work: Write briefly on **Ulcerative Lymphangitis of Cattle**

**6. LISTERIOSIS**

Listeriosis is an infectious disease characterised by encephalitis, abortion and septicaemia.

**Aetiology:** *Listeria monocytogenes* primarily in ruminants particularly sheep. Many animals exposed to this organism carry it in their faeces as a normal bowel inhabitant, but small proportions of animals develop clinical disease. Predisposing factors include poor nutritional state, sudden changes of weather to very cold and wet, the stress of late pregnancy and parturition, overcrowding and unsanitary conditions with poor access to feed supplies.

**Clinical Findings:** The most readily recognized form of Listeriosis in ruminants is encephalitis, affecting all ages of both sexes. The course in sheep and goats is rapid and death may occur 4 to 48 hours after the appearance of signs. Infected animal becomes solitary, leans against stationary objects as if unable to stand. When walking, it often moves in a circle. Marked elevation of temperature, anorexia, conjunctivitis and blindness may be present. Marked depression, in-coordination and paralysis of the muscles of the jaw, eye and ear as well as stringy salivation and nasal discharge are conspicuous signs. Terminally, involuntary and aimless running movements are common. *Listeria* *monocytogenes* has been associated with late gestation abortion and perinatal death in ruminants. Usually, the dam shows no signs of illness or residual damage from listeric abortion. Encephalitic signs and abortion do not usually occur simultaneously on the same premises.

**Diagnosis:** Listerial meningoencephalitis may be confused with the nervous form of acetonaemia in cattle and with early cases of pregnancy toxaemia in sheep. Both of these metabolic diseases are accompanied by marked ketonuria. Listerial abortion must be differentiated from the other causes of abortion.

**Treatment:** Chlortetracycline.

**7. ERYSIPELAS IN SWINE**

Erysipelas is an infectious disease of pigs and appears in an acute, septicaemic form often accompanied by diamond-shaped skin lesions, and a chronic form manifested by a non-suppurative arthritis and a vegetative endocarditis.

**Aetiology:** *Erysipelothrix rhusiopathie* (*insidiosa*) through skin abrasions and the alimentary tract mucosa by ingestion of contaminated feed. Young pigs in contact with carrier sows rapidly acquire the status of carriers and shedders.

**Clinical Findings:** The several forms of the disease (acute septicaemia, skin form, chronic arthritis and vegetative endocarditis) may occur together, in sequence, or separately. Swine (suckling pigs) with acute septicaemia may die suddenly without previous manifestation of illness. Most acutely infected animals have high temperatures (40 to 420C), walk stiffly on their toes, lie on their sternums, or lie about separately rather than piling in groups. They squeal readily when handled or when submitted to any type of body pressure, and shift weight from foot to foot when standing.

Skin discolouration may vary from erythema and purplish discolouration of the ears, snout and abdomen, to urticaria (diamond-skin lesions) over all areas of the body. The lesions may occur as variably sized pink or light purple areas that become raised and firm to the touch within 2 to 3 days of illness. Later, they may disappear or progress to a more chronic type of lesion, such as diamond-skin disease or even necrosis and separation of large areas of skin. Mortality may vary from 0 to 100% and death may occur up to 6 days after the first sign of illness.

Untreated animals may develop chronic arthritis or vegetative valvular endocarditis. These conditions may also occur in pigs that have shown no previous signs of septicaemia. Mortality in chronic cases is low.

**Diagnosis:** The acute disease may be confused with the other septicaemias affecting pigs, but pigs with erysipelas usually show the characteristic skin lesions and are less depressed than pigs with hog cholera or salmonellosis.

**Treatment:** Penicillin

**8. ANTHRAX**

Anthrax is a peracute disease characterised by septicaemia and sudden death accompanied by the exudation of tarry blood from the body orifices of the cadaver. Failure of the blood to clot, absence of rigor mortis and the presence of splenomegaly are the most important necropsy findings.

**Aetiology:** *Bacillus anthracis*

**Clinical findings:** The incubation period is typically 3 to 7 days. The clinical course ranges from peracute to chronic. The **peracute form** is characterised by sudden onset and a rapidly fatal course. There may be staggering, difficult breathing, trembling, collapse, a few convulsive movements and death in ruminants without any previous evidence of illness.

In **acute anthrax**, there is first an abrupt rise in body temperature and a period of excitement followed by depression, stupor, respiratory or cardiac distress, staggering, convulsion and death. There may be bloody discharges from the natural body openings.

**Chronic** infections most frequent in swine are characterised by localised, subcutaneous oedematous swelling, which can be quite extensive.

**Diagnosis:** A diagnosis based on clinical findings may be difficult, especially when the disease occurs in a new area. Therefore, a confirmatory laboratory examination should be done. A small amount of blood collected aseptically from a superficial vessel such as the jugular vein is the preferred specimen.

Anthrax must be differentiated from other conditions that cause sudden death such as Clostridia infections, bloat, lighting stroke, acute Leptospirosis, bacillary haemoglobinuria, anaplasmosis, hog cholera, African swine fever and acute poisoning.

**Treatment:** Penicillin, tetracyclines, erythromycin or sulphonamides.

Immunise all apparently healthy animals in the herd and on surrounding premises.

**9. TETANUS**

Tetanus is a highly fatal, infectious disease of all species of domestic animals, characterised clinically by hyperesthesia, tetany and convulsions.

**Aetiology:** Toxins of *Clostridium tetani*. *Clostridium tetani* forms spores which are capable of persisting in soil for many years. The portal of entry is usually through deep puncture wounds.

**Clinical findings:** The incubation period varies between 1 and 3 weeks with occasional cases occurring as long as several months after the infection is introduced. A general increase in muscle stiffness is observed first and is accompanied by muscle tremor. There is trismus with restriction of jaw movements, prolapse of the third eyelid, stiffness of the hind limbs causing an unsteady, straddling gait and the tail is held out stiffly especially when backing or turning. Additional signs include an anxious and alert expression, erect carriage of the ears, retraction of the eyelids and dilation of the nostrils, and exaggerated responses to normal stimuli. The animal may continue to eat and drink in the early stages but mastication is soon prevented by tetany of the masseter muscles, and saliva may drool from the mouth. If food or water is taken, attempts at swallowing are followed by regurgitation from the nose. The temperature and pulse rate are within the normal range in the early stages but may rise later when muscular tone and activity are further increased.

As the disease progresses, muscular tetany increases and the animal adopts a ‘sawhorse’ posture. There is great difficulty in walking and the animal is inclined to fall. Once down, it is almost impossible to get a large animal to its feet again.

**Diagnosis:** Fully developed tetanus is so distinct clinically.

**Treatment:** The prognosis is guarded. Average treatment course is 27 days. The main principles in the treatment of tetanus are to eliminate the causative bacteria, neutralize residual toxin, relax the muscle tetany to avoid asphyxia and maintain the relaxation until the toxin is eliminated or destroyed. This can be achieved by:

Large doses of parenteral penicillin.

Administer antitoxin - 300,000 units, 12 hourly for 3 injections.

Debridement of infection site and irrigation with hydrogen peroxide.

Chlorpromazine (0.4mg/kg i/v or 1.0mg/kg i/m) and acetyl promazine (0.05mg/kg) twice daily for 8 to 10 days until severe signs subside.

Intravenous or stomach tube feeding during the critical stages when the animal cannot eat or drink.

Keep animal as quiet as possible in dark, well-bedded pen.

**10. BOTULISM**

Botulism is a rapidly fatal disease characterised motor paralysis. It is most common in birds but cattle, sheep and horses are susceptible.

**Aetiology:** Toxins of *Clostridium botulinum*, which proliferates in decomposing animal or plant materials. Botulism is intoxication, not an infection and results from ingestion of toxin in food. Toxicoinfectious botulism is the name given to the disease in which *Clostridium botulinum* is present in tissues of a living animal and produces toxins there.

**Clinical findings:** Signs usually appear 3 to 17 days after the animals gain access to the toxic materials, the incubation period being shorter as the amount of toxin available is increased.

**Peracute** cases die without prior signs of illness although a few fail to take food or water for a day before hand. The disease is not accompanied by fever and the characteristic clinical picture is one of progressive muscular paralysis affecting particularly the limb muscles and the muscles of the jaw and throat. Muscle weakness and paralysis commence in the hindquarters and progress to the forequarters, the head and the neck. The onset is marked by very obvious muscle tremor and fasciculation, often sufficient to make the whole limb tremble.

In the **sub acute form**, restlessness, pica, in-coordination, stumbling, knuckling and ataxia are followed by inability to rise or to lift the head. Skin sensation is retained. Affected animal lie in sternal recumbency with the head on the ground or turned into the flank, like the posture of a cow with parturient paresis. Paralysis of the chest muscles results in a terminal abdominal-type respiration. Sensation and consciousness are retained until the end which usually occurs quietly and with the animal in lateral recumbency, 1 to 4 days after the commencement of illness. Death is usually due to respiratory or cardiac paralysis.

**Diagnosis:** Demonstrating the presence of toxin in the suspected food, or filtrates of the stomach and intestinal contents.

Differentiate from parturient paresis, hypocalcaemia, plant poisonings, louping-ill, scrapie and paralytic rabies.

**Treatment:** Prognosis is poor. Hence vaccination with type-specific or combined toxoid is practiced in enzootic areas. A single dose, gives good immunity after 2 weeks and the immunity is solid for about 24 months.

**11. BLACKLEG**

Blackleg is an acute infectious disease of cattle and sheep characterised by inflammation of skeletal and cardiac muscles, severe toxaemia and a high mortality.

**Aetiology:** *Clostridium chauvoei* ingested in contaminated feed or by wound infection. Animals in excellent health, gaining weight and usually the best are affected.

**Clinical findings:** Many animals die without signs having been observed. Initially, there is fever but, by the time clinical signs are obvious, the temperature may be normal or subnormal. There is acute lameness, marked depression and characteristic oedematous and crepitant swellings in the hip, shoulder, chest, back, neck or elsewhere. At first, the swelling is small, hot and painful. As the disease rapidly progresses, the swelling enlarges, there is crepitation on palpation, and the skin becomes cold and insensitive as the blood supply to the area diminishes. General signs include prostration and tremors. Death occurs in 12 to 48 hours.

**Diagnosis:** The occurrence of a rapidly fatal febrile disease in well nourished young ruminants with crepitant swellings of the heavy muscles suggests black leg. The affected muscle is dark red to black, dry and spongy, has a sweetish odour, and is infiltrated with small bubbles, but with little oedema.

**Treatment:** Parenteral and multiple local injections of penicillin. Prognosis is guarded.

**12. MALIGNANT OEDEMA**

Malignant oedema is an acute wound infection of food animals characterised by acute inflammation at the site of infection and a profound systemic toxaemia.

**Aetiology:** *Clostridium septicum*, *Cl. chauvoei*, *Cl. perfringens*, *Cl. sordelli* and *Cl. novyi* have all been isolated from lesions typical of malignant oedema. The natural habitats of these organisms are the soil and intestinal tract of animals. The portal of entry in most cases is a wound and all ages are affected.

**Clinical findings:** Clinical signs appear within 12 to 48 hours of infection. There is always a local lesion at the site of infection consisting of a soft, doughy swelling with marked local erythema accompanied by severe pain on palpation. At a later stage, the swelling becomes tense and the skin dark. Extensive frothy exudation (emphysema) may be present. A high fever (41 to 420C) is always present and affected animals are depressed, weak, show muscle tremor and usually stiffness or lameness. The illness is of short duration and affected animals die within 24 to 48 hours of the first appearance of signs.

**Diagnosis:** The association of profound toxaemia with local inflammation and emphysema is characteristic. The disease is differentiated from blackleg by the presence of wounds and absence of typical muscle involvement.

**Treatment:** Heavy doses of crystalline penicillin i/v, 6-hourly with non-steroidal anti-inflammatory drug twice daily.

Surgical incision to provide drainage, irrigation with hydrogen peroxide and local injection of penicillin into and around the periphery of the lesions.

**13. INFECTIOUS NECROTIC HEPATITIS**

Infectious necrotic hepatitis is an acute toxaemia of ruminants especially sheep associated with fasciolosis. Pigs may be affected.

**Aetiology:** Toxins of *Clostridium novyi*. This organism can be isolated from the livers of normal animals but under local anaerobic conditions, such as when migrating flukes causes severe tissue destruction of the liver, the organisms already present in the liver, proliferate, liberating toxins which cause local liver necrosis and more diffuse damage to the vascular system.

**Clinical findings:** Usually death is sudden with no well-defined signs. Affected animals tend to lag behind the flock, assume sternal recumbency and die within a few hours. Most cases occur when liver fluke infection is at its height.

Clinical course is longer in cattle (1 to 2 days). There is sudden severe depression, reluctance to move, coldness of the skin, absence of rumen sounds and weakness and muffling of the heart sounds. There is abdominal pain on deep palpation of the liver.

**Diagnosis:** At necropsy, there is massive liver destruction which has a dark, gray-brown appearance and exhibits characteristic areas of necrosis. Blood stained serous fluid is always present in abnormally large amounts in the pericardial, pleural and peritoneal cavities. In addition, clinically, the course of fasciolosis is longer (2 to 3 days in sheep).

**Treatment:** No effective treatment

**14. BACILLARY HAEMOGLOBINURIA**

This is an acute, infectious, highly fatal toxaemia of ruminants especially cattle characterised by high fever, haemoglobinuria, jaundice and the presence of necrotic infarcts in the liver.

**Aetiology:** *Clostridium haemolyticum*

**Clinical findings:** The illness is of short duration and cattle at pasture may be found dead without signs having been observed. More often, there is a sudden onset, with complete cessation of rumination, feeding, lactation and defaecation. Abdominal pain is evidenced by disinclination to move and an arched-back posture. Respiration is shallow and laboured and the pulse is weak and rapid. Fever (39.5 to 410C) is evident in the early stages but subsides to subnormal before death. The faeces are dark brown; there may be diarrhoea with much mucus and some blood. The urine is dark red while jaundice is present. The duration of the illness varies from 12 hours to 4 days before death.

**Diagnosis:** Differentiate from acute Leptospirosis, post parturient haemoglobinuria, haemolytic anaemia caused by plant poison, babesiosis, anaplasmosis, anthrax, blackleg and infectious necrotic hepatitis.

**Treatment:** Penicillin or tetracycline combined with antitoxic serum, blood transfusion, parenteral electrolytes, adequate feeds and provision of mineral supplements containing iron, copper and cobalt.

**15. *Clostridium perfringens***

*Clostridium perfringens* is widely distributed in the soil and the alimentary tract of animals, and is characterised by its ability to produce potent exotoxins, some of which are responsible for specific enterotoxaemias. Six types: A, B, C, D, E and F – have been identified on the basis of the toxins produced, but of these, only three, that is, B, C and D are important.

**(a) ENTEROTOXAEMIA CAUSED BY *Clostridium perfringens* TYPES B & C** Infection with *Clostridium perfringens* Types B and C causes severe enteritis, dysentery, toxaemia and high mortality in young ruminants and pigs. Type C also causes enterotoxaemia in adult ruminants.

**Aetiology:**

**Lamb dysentery:** *Clostridium perfringens* Type B in lambs up to 3weeks of age.

**Calf enterotoxaemia**: *Clostridium perfringens* Types B & C in well fed calves up to 1 month.

**Pig enterotoxaemia**: *Clostridium perfringens* Type C in piglets during first few days of life.

**Struck:** *Clostridium perfringens* Type C in adult sheep.

**Goat enterotoxaemia**: *Clostridium perfringens* Type C in adult goats.

**Clinical Findings:** Lamb dysentery is an acute disease of lambs less than 3 weeks of age. Many may die before signs are observed, but some stop nursing, become listless and remain recumbent. Foetid, blood-tinged diarrhoea is common and death usually occurs within a few days.

In calves, there is acute diarrhoea, dysentery, abdominal pain, convulsions and opisthotonos. Death may occur in a few days and recovery over a period of several days is possible.

Piglets become acutely ill within a few days of birth and there is diarrhoea, dysentery, reddening of the anus and a high fatality rate. Most affected piglets die within 12 hours.

**Necropsy:** Haemorrhagic enteritis with ulceration of the mucosa is the major lesion in all species. Grossly, the affected portion of the intestine is deep blue-purple and appears at first glance to be an infarction associated mesenteric torsion. Smears of intestinal contents can be examined for large numbers of Clostridia and filtrates made, for the recovery of the specific type toxins.

**Diagnosis:** The early age at which this disease occurs, the rapid course and typical necropsy findings suggest the diagnosis, which can be readily confirmed by laboratory examination. Differentiate from enteritis and septicaemia caused by *Escherichia coli* and *Salmonella spp* and porcine transmissible gastroenteritis.

**Treatment:** Treatment is usually ineffective because of severity of the disease, but if available, specific hyper immune serum is indicated and oral administration of antibiotics may be helpful.

**(b) TYPE D ENTEROTOXAEMIA = PULPY-KIDNEY DISEASE**

This is an acute toxaemia of ruminants caused by the proliferation of *Clostridium perfringens* Type D in the intestines and the liberation of toxins. Clinically, the disease is characterised by diarrhoea, convulsions, paralysis and rapid death. The commonest predisposing factor is the ingestion of excessive amounts of feed or milk in the very young, and grain in feedlots.

**Clinical findings:** In lambs, the course of illness is very short, often less than 2 hours and never more than 12 hours, and many are found dead without previously manifesting signs. In closely observed flocks, the first signs may be dullness, depression, yawning, facial movements, loss of interest in feed, severe clonic convulsions with frothing at the mouth and rapid death. Cases which survive for a few hours show a green, pasty diarrhoea, staggering, recumbency, opisthotonos and severe clonic convulsions. The temperature is usually normal but may be elevated if convulsions are severe. Death occurs during a convulsion or after a short period of coma.

Adult sheep usually survive for longer periods, up to 24 hours. They lag behind the flock, show staggering and knuckling, clamping of the jaws, salivation and rapid shallow, irregular respiration.

In calves, the syndrome is similar to that seen in adult sheep, with nervous signs predominating. Per acute cases are found dead without having shown premonitory signs of illness and with no evidence of struggling. The more common acute cases show a sudden onset of bellowing, mania and convulsions, the convulsions persisting until death occurs 1 to 2 hours later. Subacute cases, many of which recover, do not drink, are quiet and docile and appear to be blind, although the eyes preservation reflex persists. They may continue in this state for 2 to 3 days and then recover quickly and completely.

Diarrhoea is a prominent sign in affected goats especially in those which survive for more than a few days. In per acute cases, there are convulsions after an initial attack of fever (40.50C) with severe abdominal pain and dysentery, and death occurs in 4 to 36 hours. In acute cases, there is usually no fever. Abdominal pain and diarrhoea are prominent with death or recovery within 2 to 4 days. In chronic cases, the goats may be ill for several weeks and show anorexia, intermittent severe diarrhoea, dysentery, presence of epithelial shreds in the faeces, chronic wasting and anaemia.

**Diagnosis:** The history, clinical and necropsy findings are diagnostic. Differentiate from other causes of sudden death such as acute Pasteurellosis, hypocalcaemia and septicaemia due to *Haemophilus sp*. In the adult, rabies, acute lead poisoning, louping-ill, pregnancy toxaemia and hypomagnesaemic tetany.

**Treatment:** Prognosis is poor. The clinical course of the disease is too acute for effective treatment. Use of hyper immune serum, oral sulphadimidine and antitoxin may be beneficial.

**16. ACUTE UNDIFFERENTIATED DIARRHOEA OF NEWBORN FARM ANIMALS**

Diarrhoea in newborn food animals (ruminants under 30 days of age and piglets in the first week of life) is one of the most common disease complexes which the large animal clinician is faced with in practice. It is characterised clinically by acute profuse watery diarrhoea, progressive dehydration, acidosis and death in a few days or earlier after the onset.

**Aetiology:** Field and laboratory investigations have indicated that there is not a single aetiology but rather, the cause is complex and usually involves interplay between entero-pathogenic bacteria, viruses, protozoa, the immunity of the animal and the effects of the environment.

That is, enterotoxigenic *Escherichia coli*, Rotavirus, Corona virus, *Cryptosporidium spp*, dietary abnormalities, *Salmonella spp*, *Chlamydia sp*, adenovirus, transmissible gastroenteritis, infectious bovine rhinotracheitis, bovine viral diarrhoea, and *Clostridium perfringens* types B and C. The predisposing factors include overcrowding, inadequate sanitation and hygiene, failure to acquire sufficient colostral immunity, adverse climatic conditions especially cold, wet and windy weather and the care provided by the calf attendant.

**Clinical management:** When a clinician is faced with an outbreak of acute diarrhoea in the newborn in which there is profuse watery diarrhoea, progressive dehydration and death in a few days or earlier, the following steps are recommended:

1. Visit the herd and conduct an epidemiological investigation to identify the risk factors responsible for the outbreak. That is, overcrowding, recent changes in climate, recent stress, the quality of milk replacer diet, recent introductions of replacement calves and failure of calves to ingest an adequate quantity of colostrum within hours after birth (by the measurement of serum immunoglobulin at about 3 days of age).

2. Clinically examine affected newborns, dead ones examined by necropsy and determine if diarrhoea is the major problem.

3. All affected newborns should be identified, isolated and treated immediately with oral and parenteral electrolyte fluid therapy as indicated.

4. Antibacterial may be given orally and parenterally for 3 days. This is to avoid elimination of too many species of drug-sensitive intestinal flora and their replacement by pathogenic fungi or bacteria such as *Candida spp*, *Proteus spp* and *Pseudomonas spp*.

5. Collect 30 to 50g of faecal samples from diarrhoeic and normal newborns to screen for aetiological agents. Also, blood samples from affected and normal newborns and colostral samples are useful for immunoglobulin and antibody studies.

6. Move pregnant dams to new uncontaminated area. Ensure that neonates take adequate colostrum as soon as practicable after birth. Passive transfer of maternal immunoglobulin to newborns depends on three successive processes: (i) formation of colostrum with a high concentration of immunoglobulin by the dam (ii) ingestion of an adequate volume of colostrum by the newborn and (iii) efficient absorption of colostral immunoglobulin by the newborn. Colostral immunoglobulins are absorbed for up to 24 hours after birth in calves and up to 48 hours in piglets. However in calves, maximum efficiency of absorption occurs during the first 6 to 12 hours after birth and decreases rapidly from 12 to 24 hours after birth.

7. Vaccinations

8. Submit a report to the owner outlining specific recommendations for clinical management and for control of the disease in future.

**17. COLIBACILLOSIS OF THE NEWBORN**

One of the most common diseases of newborn farm animals is colibacillosis caused by pathogenic *Escherichia coli*. There are at least two different types of the diseases: enteric colibacillosis manifested by varying degrees of diarrhoea, dehydration and death in a few days if not treated and septicaemic colibacillosis manifested by septicaemia and rapid death in several hours after onset.

**Clinical findings:**

**Septicaemic colibacillosis of calves:** This is common in calves during the first 4 days of life. The illness is acute, the course varying from 24 to 96 hours. There are no diagnostic clinical signs. Affected animals are depressed and weak, anorexia is complete, there is marked tachycardia and although the temperature may be high initially, it falls rapidly to subnormal levels when the calf becomes weak and moribund. The suck reflex is weak or absent and the oral mucous membranes are dry. Diarrhoea and dysentery may occur but are uncommon. If the calf survives the septicaemia state, clinical evidence of post-septicaemic localisation may appear in about 1 week. This includes arthritis, meningitis, pan-ophthalmitis and less commonly, pneumonia.

**Enterotoxigenic colibacillosis of calves:** This is common in calves from 3 to 5 days of age and only rarely up to 3 weeks. There is diarrhoea in which the faeces are profuse and watery to pasty, usually pale yellow to white in colour and occasionally streaked with blood flecks and very foul-smelling. Defaecation is frequent and effortless and the tail and buttocks are soiled. The temperature is usually normal in the initial stages but becomes subnormal as the disease worsens. Affected calves may or may not suck or drink depending on the degrees of acidosis, dehydration and weakness. In the early stages of the disease, the abdomen may be slightly distended due to distension of fluid-filled intestines which can be detected by succussion and auscultation of the abdomen. Death usually occurs in 3 to 5 days. Affected calves can lose 10 to 16% of their original body weight during the first 24 to 48 hours of diarrhoea. Mild to moderately affected calves may be diarrhoeic for a few days and recover spontaneously with or without treatment.

The degree of dehydration is best assessed by ‘tenting’ the skin of the upper eyelid or the neck and measuring the time required for the skin-fold to return to normal. In calves with 8% dehydration, 5 to 10 seconds will be required for the skin-fold to return to normal; in 10 to 12% dehydration, up to 30 seconds.

**In lambs**, colibacillosis is almost always septicaemic and peracute. Two age groups appear to be susceptible, lambs of 1 to 2 days of age and lambs 3 to 8 weeks old. Peracute cases are found dead without premonitory signs. Acute cases show collapse and occasionally signs of acute meningitis manifested by a stiff gait in the early stages, followed by recumbency with hyperaesthesia and titanic convulsions. Chronic cases are usually manifested by arthritis.

**In piglets**, enterotoxic colibacillosis is the most common form and occurs as from 12 hours of age up to several days of age with a peak incidence at 3 days of age. Usually, more than one piglet or the entire litter is affected. The first noticed sign is the faecal puddles on the floor. Affected piglets may still nurse in the early stages but gradually lose their appetite as the disease progresses. The faeces vary from a pasty to watery consistency and are usually yellow to brown in colour. When the diarrhoea is profuse and watery, there will be no obvious staining of the buttocks with faeces but the tails of the piglets will be straight and wet. The temperature is usually normal to subnormal. As diarrhoea and dehydration continues, the piglets become very weak and lie in lateral recumbency and make weak paddling movements. Within several hours, they appear very dehydrated and shrunken and die within 24 hours after the onset of signs. In severe outbreaks, the entire litter may be affected and die within a few hours of birth.

**Diagnosis:** The septicaemia of the newborn cannot be distinguished from each other clinically. Mixed infections are more common than single infections. Every economically possible effort should be made to obtain an aetiological diagnosis.

**Treatment:** The prognosis is favourable if treatment is started early before significant dehydration and acidosis occur. The prognosis is however unfavourable if the level of immunoglobulin is low, regardless of intensive fluid and antimicrobial therapy.

Withhold milk. It has been common practice in hand fed calves to use oral fluids and electrolytes as milk replacement during the period of diarrhoea up to 24 hours or until there is clinical evidence of improvement.

Diarrhoeic neonates are usually treated with antimicrobials and oral fluids and electrolytes and left with the dam.

Parenteral fluid therapy may be indicated.

**18 POST-WEANING DIARRHOEA OF PIGS=COLIFORM GASTROENTERITIS**

Post weaning diarrhoea of pigs occurs commonly within several days to 2 weeks after weaning and is characterised by reduced growth rate, sudden death or severe diarrhoea, dehydration and toxaemia.

**Aetiology:** *Escherichia coli*. Predisposing factors include stress from loss of maternal contact, early weaning (at 3 weeks) and introduction of different batch or formulation of the creep feed.

**Clinical findings:** Diarrhoea is the cardinal sign; the faeces are very watery and yellow in colour but may be passed without staining of the buttocks and tail. Several weaners show mild depression, moderate pyrexia, reduction in growth rate, reduction in feed intake (affected pigs will still drink), gaunt abdomens, lusterless hair coats, and a pink discolouration of the skin of the ears, ventral neck and belly in the terminal stages. The course of an outbreak within a group is generally 7 to 10 days and the majority of weaners that die do so within the initial 5 days. Survivors show poor growth rate for a further 2 to 3 weeks and some individuals show permanent retardation in growth.

**Diagnosis:** Coliform gastroenteritis must always be the prime consideration in pigs that are scouring or dying with a 3 to 10 day period of some feed or management change. Differentiate from Swine dysentery, Salmonellosis, Swine fever, Erysipelas, Pasteurellosis, and *Haemophilus parahaemolyticus* infection.

**Treatment:** Treat all weaners within the group with oral or parenteral antimicrobials such as neomycin, nitrofurans, ampicillin, tetracyclines, sulphonamides or trimethoprim-potentiated sulphonamides for 5 to 7 days. Water medication is preferable and fluid and electrolytes for severally dehydrated ones.

**19. SALMONELLOSIS = PARATYPHOID**

Salmonellosis is a disease of all animals caused by many species of salmonellae and characterised clinically by one of three major syndromes: peracute septicaemia, acute enteritis and chronic enteritis. Young ruminants and piglets are susceptible and usually develop the septicaemic form; adult ruminants commonly develop acute enteritis and chronic enteritis may occur in growing pigs and occasionally in cattle.

Salmonellosis is a zoonotic disease and animals especially pigs and poultry have been incriminated as the principal reservoir. Transmission to man occurs via contaminated drinking water, milk, meat and eggs.

**Aetiology:**

Cattle: *Salmonella typhimurium*, *S. dublin*, *S. newport*

Sheep/Goat: *S. typhimurium*, *S. dublin*, *S. anatum*

Pig: *S. typhimurium*, *S. choleraesuis*

There are no significant differences between infections caused by the different Salmonella species. Stressors that precipitate clinical disease include deprivation of feed and water, long transportation, recent calving and mixing and crowding of feedlots.

**Clinical findings:** Septicaemia is the usual syndrome in newborn ruminants, piglets and pigs up to 6 months of age. Illness is acute, depression is marked, fever (40.5 to 41.50C) in usual and death occurs in 24 to 48 hours. In pigs, a dark red to purple discolouration of the skin is common, especially at the ears and ventral abdomen. Nervous signs may occur in calves and pigs. Pig may also suffer from pneumonia.

**Acute enteritis** is the common form in adults and it also may occur in calves 3 to 6 weeks of age. Initially there is a fever (40.5 to 41.50C), followed by severe watery diarrhoea, sometimes dysentery and often tenesmus. In a herd outbreak, several hours may lapse before the onset of diarrhoea, at which time the fever may disappear. The faeces vary considerably: they may have a putrid smell and contain mucus, fibrinous casts, and even shreds of mucous membrane; in some cases, large blood clots are passed. Rectal examination causes severe discomfort, tenesmus and commonly dysentery.

**Subacute enteritis** may occur in adult ruminants on farms where the disease is endemic. The signs include mild fever (39 to 400C), soft faeces, inappetence and some dehydration. There may be a high incidence of abortion in cows and ewes, some deaths in ewes after abortion, and a high mortality rate due to enteritis in lambs under a few weeks of age. In cattle, the first signs may be fever and abortion followed several days later by diarrhoea.

**Chronic enteritis** is a common form in pigs and adult cattle. There is persistent diarrhoea, severe emaciation, intermittent fever and poor response to treatment. The faeces are scant and may be normal or contain mucus, casts or blood. In growing pigs, rectal stricture may be a sequela if the terminal part of the rectum is involved. Affected pigs are anorexic, lose weight and their abdomen becomes grossly distended.

**Diagnosis:** Clinical signs and laboratory examination of the faeces and tissues from affected animals will aid diagnosis. Differentiate from enteric colibacillosis, coccidiosis, infectious bovine rhinotracheitis, bovine viral diarrhoea, haemorrhagic enteritis due to *Clostridium perfringens* types B and C, helminthosis, arsenic poisoning, paratuberculosis and dietetic diarrhoea in cattle.

In swine, differentiate from Enteric colibacillosis of newborn pigs and weaners, Swine dysentery, Campylobacteriosis, Erysipelas, Hog cholera and Pasteurellosis.

In sheep, enteric colibacillosis, septicaemia due to *Haemophilus sp*. or *Pasteurella sp*. and coccidiosis.

**Treatment:** Broad spectrum antibiotics are used parenterally to treat the septicaemia. Trimethoprim-sulphadoxine, ampicillin, amoxicillin, trimethoprim-suphadiazine, daily for up to 6 days.

Oral medication should be given in drinking water since affected animals are thirsty due to dehydration and their appetite is generally poor.

Oral dosing is satisfactory in pre-ruminant calves, but it much less effective when given to grazing ruminants. Fluid therapy, oral and parental may be beneficial.

**20. BOVINE RESPIRATORY DISEASE**

A major problem which large animal clinicians commonly encounter is a group of cattle which are affected with an acute respiratory disease of uncertain diagnosis. The clinical findings may include some unexpected deaths, dyspnoea, coughing, nasal discharge, varying degrees of depression, inappetence or anorexia, a fever ranging from 40 to 41.50C, evidence of pneumonia on auscultation of the lungs and a variable response to treatment.

In some instances, even after intensive clinical and laboratory investigation, the specific aetiology will not be determined and the veterinarian may be left with a diagnosis of ‘acute undifferentiated respiratory disease of cattle’ or bovine respiratory disease.

The infectious diseases caused by either viruses or bacteria alone or in combination are often difficult to distinguish from each other on a clinical basis and create the most difficult problems.

Determine if the animals have pneumonia, rhinitis, laryngitis, tracheitis, bronchitis or combinations of these abnormalities. Attempt to make a clinical diagnosis by closely examining several typically affected animals and determine if the lesions are in the lower or upper respiratory tract.

The presence of toxaemia, depression, fever, anorexia, agalactia in lactating dairy cattle indicates a primary or secondary bacterial infection. The presence of increased breath sounds and abnormal lung sounds indicates the presence of pneumonia.

Diseases of the upper respiratory tract are characterised by inspiratory dyspnoea, stridor, loud coughing, sneezing, wheezing and lesions of the nasal mucosa.

An important factor which contributes to the difficulty of unraveling the aetiologies in field outbreaks of respiratory diseases is that practicing veterinarians are limited in most situations to correlating clinical, epidemiological and necropsy findings in making a diagnosis.

Diagnostic laboratories may not be readily available and their resources for sophisticated microbiological and serological investigations may be much less than is needed for an accurate determination of causes.

**Clinical management:**

1. Visit the herd to conduct clinical and epidemiological investigations. For example, check for recent purchases, weather changes, inadequate ventilation in housed cattle, movement to and fro markets.

2. Parenteral antimicrobial therapy for at least 3 days is of prime importance. Administer as soon as detected.

3. Treated animals should be suitably identified and a record kept of the initial body temperature and the treatment administered.

4. A beneficial response to therapy should be apparent within 12 to 24 hours. The body temperature should decline significantly and the appearance of the animal and its appetite should be improved.

5. Animals which do not respond to treatment and die should be submitted to intensive necropsy examination and culture of affected lungs.

6. There are single-antigen or multiple-antigen vaccines, modified live virus or inactivated virus vaccines containing one or more of the following antigens: *Pasteurella haemolytica*, *Haemophilus somnus*, Infectious bovine rhinotracheitis, Para influenza -3, Bovine respiratory syncytial virus and Bovine viral diarrhoea.

**21. PNEUMONIC PASTEURELLOSIS**

Pneumonic Pasteurellosis is a respiratory disease of ruminants and swine characterised clinically by acute bronchopneumonia with toxaemia and pathologically by lobar, anteroventrally distributed, exudative pneumonia in which fibrin is usually a prominent part of the exudate.

**Aetiology:** *Pasteurella haemolytica*, *P. multocida*. All age are susceptible, however, young growing cattle from 6 months to 2 years of age, lambs and kids during the first few months of life, ewes at lambing and grower-finisher pigs are more susceptible. Transmission occurs by the inhalation of infected droplets. When animals are closely confined in inadequately ventilated barns (pens), or when overcrowded in trucks, or held for long periods in holding pens, the disease may spread very quickly thereby affecting a high proportion of the herd within 48 hours. The rate is much slower in animals at pasture.

**Clinical findings:**

**Cattle**: the disease usually develops 10 to 14 days after they have been stressed or within 1 day of arrival if they have been incubating the disease prior to arrival. The first sign of an outbreak may be sudden death. When viewed from a distance, affected cattle are usually depressed and the respirations are shallow and rapid. There may be a weak protective cough which becomes more pronounced and frequent if they are urged to walk. Animals which have been ill for a few days will appear gaunt in the abdomen because of anorexia; they may continue to drink maintenance amounts of water. A mucopurulent nasal discharge, a crusty nose and an ocular discharge are common. There is fever of 40 to 410C and evidence of bronchopneumonia. In the early stages, there are loud breath sounds (bronchial tones) audible over the anterior and ventral parts of the lungs. As the disease progresses, the loud breath sounds become louder and extend over a greater area and crackles become audible followed by squeaky and musical wheezes in a few days, especially in chronic cases which are ill for several days. The course of the disease is usually short, 2 to 4 days. If treated early, affected cattle recover in 24 to 48 hours but severe cases and those which have been ill for a few days before being treated may die or become chronically affected in spite of prolonged therapy.

**Lambs and kids**: outbreaks of pneumonic Pasteurellosis often commence with sudden deaths. Older animals show signs of respiratory involvement which can be accentuated by driving. There is fever (40.4 to 420C), depression, anorexia, dyspnoea, slight frothing at the mouth, cough and nasal discharge. In more chronic cases, air flow sounds is heard over the area of the bronchial hilus often with fluid sounds. Death may occur as soon as 12 hours after the first signs of illness but the course in most cases is about 3 days. Animals that recover have evidence of chronic pneumonia and are often culled because of persisting ill-health and poor thrift.

Affected **pigs** have fever up to 410C; there is anorexia, disinclination to move, and show significant respiratory distress with laboured respiration, often breathing through the mouth. Without treatment, death is common after a clinical course of 4 to 7 days. There is a marked tendency for the disease to become chronic, resulting in reduced weight gains and frequent relapses. Although pneumonic Pasteurellosis of swine is secondary to other underlying respiratory disease (caused by *Mycoplasma hyopneumoniae*), *Pasteurella multocida* infection can occur as an outbreak, spreading to affect several pigs within a group. The first indication of disease may be the finding of a pig dead with a peracute infection.

**Diagnosis:** Differentiate from other caused of pneumonia.

**Treatment:** Oxytetracycline, Trimethoprim/sulphadoxine, Penicillin, Erythromycin.

**22. CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP)**

CBPP is a highly infectious septicaemia characterised by localisation in the lungs and pleura.

**Aetiology:** *Mycoplasma mycoides* subspecies *mycoides* (small colony type). It is not communicable to other species. The principal method of spread of CBPP is by inhalation of infective droplets from active or carrier cases of the disease. Because of this method of spread, outbreaks tend to be more extensive in housed animals and in those in transits by train or foot. Morbidity up to 90% while case mortality up to 50%.

In goat, CCPP is due to *Mycoplasma capricolum* subspecies *capripneumoniae*.

**Clinical findings:** After an incubation period of 3 to 6 weeks, there is a sudden onset of high fever (400C), a fall in milk yield, anorexia and cessation of rumination. There is severe depression and the animals stand apart or lag behind a traveling group. Coughing, at first only on exercise, and chest pain are evident, affected animals being disinclined to move, standing with the elbows out, the back arched and the head extended. Respirations are shallow, rapid and accompanied by expiratory grunting. Pain is evidenced on percussion of the chest. Auscultation reveals pleuritic friction sounds in the early stages of acute inflammation, and dullness, fluid sounds and moist gurgling crackles in the later stages of effusion. Dullness of areas of the lung may be detected on percussion. Inconstantly, oedematous swellings of the throat and dewlap occur.

Recovered animals may be clinically normal but in some, an inactive sequestrum forms in the lung, with a necrotic centre of sufficient size to produce a toxaemia causing unthriftiness, a chronic cough, and mild respiratory distress on exercise. These sequestra commonly break down when the animal is exposed to environmental stress and cause an acute attack of the disease. In fatal cases, death occurs after a variable course of from several days to 3 weeks.

**Diagnosis:** Diagnosis is based on a history of contact with infected animals, clinical findings, complement fixation test and cultural examination. Differentiate from pneumonic Pasteurellosis and parasitic pneumonia.

**Treatment:** No therapeutic treatment is effective. Antibiotics have no role in the eradication of CBPP either at the farm level or more importantly, nationally and internationally. Antibiotics can alleviate the clinical course of the disease enabling some improvement in condition. For a Nigerian farmer, this prevents the loss of his form of income and livelihood.

Effective antibiotics include tilmicosin, danafloxacin, florofenicol, tylosin and tetracycline.

**23. PLEUROPNEUMONIA OF PIGS**

This is highly contagious pleuropneumonia of growing pigs (2 to 6 months old) characterised by a rapid onset and a short course with severe dyspnoea, the passage of blood-stained foam from the mouth and nose and a high case fatality rate.

**Aetiology:** *Actinobacillus pleuropneumoniae* (formerly known as *Haemophilus pleuropneumoniae*). Transmission is by the respiratory route and overcrowding and inadequate ventilation may facilitate spread.

**Clinical findings:** The onset is sudden. Several pigs which were not seen ill may be found dead and others show severe respiratory distress. Affected pigs are disinclined to move and are anorexic. Fever (up to 410C) is common, laboured respirations, cyanosis and blood stained frothy discharge from the nose and mouth. In peracute cases, the clinical course may be as short as a few hours, but in the majority of pigs, it is 1 to 2 days. Chronic cases are febrile and anorexic initially but respiratory distress is less severe and a persistent cough may develop with an unthrifty appearance. If affected pigs are not treated, there will be a high case fatality rate. Abortions and sudden deaths may occur in adult pigs.

**Diagnosis:** The rapidity of onset and spread with fever, anorexia, severe dyspnoea and high mortality differentiate this disease. Differentiate from Enzootic pneumonia, Pasteurellosis, swine influenza and Glasser’s disease.

**Treatment:** Treat affected and in-contact pigs with tetracycline or penicillin as soon as practicable.

**24. ENZOOTIC PNEUMONIA OF PIGS**

This is a highly contagious disease of pigs manifested clinically by pneumonia of moderate severity and failure to grow at a normal rate.

**Aetiology:** *Mycoplasma hyopneumoniae* is the primary causative agent while *Pasteurella multocida* is commonly a secondary invader. These organisms are inhabitants of the respiratory tract of pigs and transmission occurs by direct pig-to-pig contact. Air borne transmission has been demonstrated.

**Clinical findings:** After an incubation period of 10 to 1 6 days, an acute or chronic farm of the disease may occur.

In the **acute form**, pigs of all ages are susceptible and a morbidity of 100% may be experienced. Acute respiratory distress with or without fever is characteristic and mortality may occur. The usual course of this form of the disease within a herd is usually about 3 months after which it subsides to the chronic form.

The **chronic form** of the disease is much more common and is the pattern seen in endemically infected herd. Young piglets are usually infected when they are 3 to 10 weeks of age. The onset of clinical abnormality is insidious and coughing is the major manifestation. It may disappear in 2 to 3 weeks or persist throughout the growing period. In affected herds, individual pigs may be heard to cough at any time but coughing is most obvious at initial activity in the morning, at feeding time and in the period immediately following exercise. A dry or crackling, hacking cough, which is usually repetitive, is characteristic. Respiratory embarrassment is rare and there is no fever or obvious in appetence. Subsequently, there is retardation of growth which varies in severity between individuals so that uneven group size is common. Some pigs affected with the chronic form of the disease may later develop acute pneumonia due to secondary invasion of *Pasteurella multocida*.

**Diagnosis:** Differentiate from swine influenza, pleuropneumonia of growing pigs, lungworm and ascaris infestations.

**Treatment:** There is no effective treatment to eliminate infection with *Mycoplasma hyopneumoniae*, although the severity of the clinical disease may be reduced by Lincomycin-spectinomycin, Tylosin, Doxycycline, Oxytetracycline and Ciprofloxacin.

**25. BRUCELLOSIS**

Brucellosis is a disease of ruminants and swine characterised by abortion in late pregnancy, high rate of infertility in the female and varying degrees of sterility in the male. It is an important zoonosis, causing undulating fever in humans.

**Aetiology:** *Brucella abortus* in cattle, *B. ovis* in sheep, *B. suis* in pigs, *B. melitensis* in goat, sheep and cattle.

Transmission is by ingestion, penetration of the intact skin and conjunctiva and contamination of the udder during milking. Also contact with aborted foetuses and infected newborn calves.

**Clinical findings:** In highly susceptible non-vaccinated pregnant cattle, abortion after the 5th month of pregnancy is the cardinal feature of the disease in cows. In subsequent pregnancies, the foetus is usually carried to full term although second or even third abortions may occur in the same cow. Retention of the placenta and metritis are common sequels to abortion. In the bull, orchitis and epididymitis may occur; one or both scrotal sacs may be affected with acute, painful swelling to twice normal size, although the testes may not be grossly enlarged. Affected bulls are usually sterile when the orchitis is acute but may regain normal fertility if one testicle is undamaged. Such bulls are potential spreaders of the disease if they are used for artificial insemination.

Abortion during late pregnancy is the most obvious sign in goats and sheep (abortion most common in the last 2 months of pregnancy), or the birth of weak or stillborn lambs. Infections in males may be followed by orchitis, lameness and hygroma.

In sows, infertility, which may be temporary, irregular oestrus, small litters and abortion occur. Sows abort usually once and they breed normally thereafter. Although abortion is most common during the third month, it may occur earlier. In boars, orchitis with swelling and necrosis of one or both testicles is followed by sterility.

**Diagnosis:** Isolation of the causative organism and serology.

Differentiate from Trichomoniasis, Vibriosis, Listeriosis, Leptospirosis, dietary factors and Infectious bovine rhinotracheitis in cattle.

In sheep and goat, Vibriosis, Listeriosis, Salmonellosis, Toxoplasmosis.

In pigs, Vitamin deficiencies, Leptospirosis, Swine fever, Erysipelas.

**Treatment:** Treatment of brucellosis is generally unsuccessful because of the intracellular sequestration of the organisms in lymph nodes, the mammary gland and reproductive organs. However, the use of long acting oxytetracycline at 20mg/kg i/m at 3 to 4 days intervals for five treatments in combination with streptomycin at 25mg/kg i/m or i/v for 7 consecutive days was partially successful.

**26. INFECTIOUS POLYARTHRITIS=GLASSER’S DISEASE**

This is a fibrinous, occasionally fatal, polyserositis, pleurisy and polyarthritis of young pigs.

**Aetiology:** *Haemophilus parasuis*, *H. suis*.

Predisposing factors include stressors such as weaning and transportation.

**Clinical findings:** The onset is sudden with a moderate to high fever (40 to 420C), complete anorexia, an unusual, rapid, shallow dyspnoea with an anxious expression, extension of the head and mouth breathing. Coughing may occur. The animals are very lame, stand up on their toes and move with a short shuffling gait. All the joints are swollen and painful on palpation and fluid swelling of the tendon sheaths may also be clinically evident. A red to blue discolouration of the skin appears near death. Most cases die 2 to 5 days after the onset of illness. Animals which survive the acute stage of the disease may develop chronic arthritis and some cases of intestinal obstruction caused by peritoneal adhesions occur. Also, meningitis manifested by muscle tremor, paralysis and convulsions occur.

**Diagnosis:** The unusual combination of arthritis, fibrinous serositis and meningitis is sufficient to make a diagnosis. Differentiate from Erysipelas, Mycoplasma arthritis and Streptococcal arthritis.

**Treatment:** Penicillin, oxytetracycline, trimethoprim-sulphadoxine.

**27. TUBERCULOSIS**

This is an infectious disease of all animal species including cattle, goats and pigs characterised by the progressive development of tubercles in any of the organs. It is a zoonotic disease. Infection is effected by the inhalation or ingestion of the causative organisms which are excreted in the exhaled air, in sputum, faeces, milk, urine, vaginal and uterine discharges and discharges from open peripheral lymph nodes.

**Aetiology:**

*Mycobacterium bovis*. The closer the animals are packed together, the greater is the chance that the disease will be transmitted. Hence, housing and zero grazing predisposes to the disease.

**Clinical findings:**

Signs exhibited depend upon the extent and location of lesions. Enlarged superficial lymph nodes provide a useful diagnostic sign. The general signs are weakness, anorexia, dyspnoea, emaciation and low-grade fluctuating fever. The lungs are usually involved, with an intermittent, hacking cough. Also, there is chronic wasting or emaciation that occurs despite good nutrition and care. Affected animals tend to become more docile and sluggish but the eyes remain bright and alert.

Tuberculous mastitis is of major importance because of the danger to public health, spread of the disease to calves and the difficulty of differentiating it from other forms of mastitis. Its characteristic feature is a marked induration and hypertrophy which usually develops first in the upper part of the rear quarters. Also, there is enlargement of the supramammary lymph nodes.

**Diagnosis:** Difficult clinically. Most cases are found at necropsy. Tuberculin test. Differentiate from Lung abscess due to Aspiration pneumonia, Pleurisy and Pericarditis following Traumatic reticulitis, and Chronic Contagious Bovine Pleuropneumonia.

**Treatment:** Not advisable even though isonicotinic acid hydrazide (INH) in combination with streptomycin and para-aminosalicylic acid is effective.

**28. PARATUBERCULOSIS = JOHNE’S DISEASE**

Johne’s disease is incurable, chronic, infectious, granulomatous enteritis of ruminants characterised clinically by chronic diarrhoea and progressive emaciation. There is no known zoonotic risk, but chronic synovitis and tendonitis have occurred in man after accidental self-inoculation of the vaccine.

**Aetiology:** *Mycobacterium paratuberculosis*. Transmission is principally by the ingestion of feed and water contaminated by the faeces of infected animals. Usually infected animals may excrete organisms in the faeces for 15 to 18 months before clinical signs appear.

**Clinical findings:** In cattle, clinical signs do not appear before 2 years of age, and are commonest in 2 to 6 year age group. Emaciation is the most obvious abnormality and is usually accompanied by submandibular oedema which has a tendency to disappear as diarrhoea develops. There is marked absence of blood, epithelial debris, mucus and offensive odour in the faeces. The course of the disease varies from weeks to months but always terminates in severe dehydration, emaciation and weakness necessitating destruction.

**Diagnosis:** Chronic diarrhoea of Johne’s disease does not respond to therapy. Differentiate from Salmonellosis, Coccidiosis, Parasitism, Malnutrition, Chronic reticulo-peritonitis, Hepatic abscess and Lymphosarcoma.

**Treatment:** No suitable treatment.

**29. ACTINOMYCOSIS = LUMPY JAW**

This is a chronic disease of the mandible, maxilla and other bony tissues of the head characterised by swelling, abscesses, fistulous tracts, extensive fibrosis, osteitis and granuloma in cattle.

**Aetiology:** *Actinomyces bovis*. This organism is a common inhabitant of the bovine mouth and infection may occur through wounds of the buccal mucosa or dental alveoli.

**Clinical findings:** Actinomycosis of the jaw commences as a painless, bony swelling which appears on the mandible or maxilla, usually at the level of the central molar teeth. The swellings are very hard, are immovable and in the later stages, painful to the touch. They usually break through the skin and discharge through one or more openings. The discharge of pus is small in amount and consists of sticky, honey-like fluid containing minute, hard, yellow-white granules. There is a tendency for the sinuses to heal and for fresh ones to develop periodically. Teeth embedded in the affected bone become misaligned and painful and cause difficult mastication with consequent loss of condition. In severe cases, spread to contiguous soft tissues may be extensive and involve the muscles and fascia of the throat. Excessive swelling of the maxilla may cause dyspnoea. The course of infection varies from several months to a year or more.

**Diagnosis:** Differentiate from abscess of cheek muscle, foreign bodies or accumulations of dry feed between the teeth.

**Treatment:** Sulphonamides, penicillin, streptomycin, tetracycline, chloramphenicol and erythromycin are effective. Cases of Actinomycosis may be treated surgically by opening of the lesion to provide drainage, and packing with gauze soaked in tincture of iodine.

**30. ACTINOBACILLOSIS = WOODEN TONGUE**

This is a disease similar to Actinomycosis but most often affecting soft tissues especially the tongue and lymph nodes.

**Aetiology:** *Actinobacillus lignieresii*. This organism is a normal inhabitant of the oral cavity and rumen of ruminants. Infection of cattle occurs most commonly through ulcerating or penetrating lesions of the tongue.

**Clinical findings:** The onset of glossal Actinobacillosis is usually acute, the affected animal being unable to eat for a period of about 48 hours. There is excessive salivation and gentle chewing of the tongue as though foreign bodies were present in the mouth. The tongue is swollen and hard, particularly at the base; nodules and ulcers are present on the side of the tongue.

In the later stages, the tongue becomes shrunken and immobile and there is considerable interference with prehension in addition to lymphadenitis which may be palpable as the enlargement of the submaxillary and parotid nodes.

In sheep, the tongue is not usually affected. Lesions up to 8cm in diameter occur on the lower jaw, face and nose or in the skin-folds from the lower jaw to the sternum. They may be superficial or deep and usually extend to the cranial or cervical lymph nodes. Viscid, yellow-green pus containing granules is discharge through a number of small openings. Prehension or respiration may be impeded and affected sheep may die of starvation.

**Diagnosis:** Differentiate from early Rabies, Foreign bodies in the mouth, Tuberculosis and Lymphomatosis.

**Treatment:** Sulphonamides, penicillin, streptomycin, tetracycline, chloramphenicol and erythromycin are effective.

**31. NOCARDIOSIS = BOVINE FARCY**

Nocardiosis is a chronic disease of cattle characterised by generalised, purulent lymphangitis, lymphadenitis, granulomatous nodular lesions and the common occurrence of lesions on the lower limbs suggests that the causative organisms are soil-borne and gain entry through minor injuries.

**Aetiology:** *Nocardia asteroides* (*Mycobacterium farcinogens*)

**Clinical findings:** Initially, there is chronic, painless, localised subcutaneous cellulitis which spreads along lymphatics to involve local lymph nodes. Further spread to the lungs may occur. Typical lesions include chronic indurated, subcutaneous swellings and enlargement and thickening of local lymphatics and lymph nodes. Discrete swellings develop along the affected lymphatic vessels and these may rupture and discharge pus through sinuses. The general health of affected animals is not impaired unless the lesions are extensive or pulmonary involvement occurs. Farcy nodules may also be present at favoured points of tick (Amblyomma) attachment. The lesions may rupture to discharge odourless thick gray or yellow pus which is often granular or cheesy.

**Diagnosis:** Differentiate from Ulcerative lymphangitis and Tuberculosis.

**Treatment:** As for Actinobacillosis

**32. CUTANEOUS STREPTOTRICHOSIS = DERMATOPHILOSIS**

This is an epidermal infection of ruminants, more prevalent in the tropics, characterised by an exudative dermatitis with scab formation. In Africa, the disease is often combined with Demodicosis to produce ‘Senkobo disease’, a more severe and often fatal combination.

**Aetiology:** *Dermatophilus congolensis*. Factors such as prolonged wetting by rain, high humidity, high temperature and various ectoparasites that reduce or permeate the natural barriers of the integument influence the development, prevalence, seasonal incidence and transmission of Dermatophilosis.

**Clinical findings:** In cattle, lesions occur on the neck, body or back of the udder and may extend over the sides and down the legs and the ventral surface of the body. Skin lesions are usually in different stages of progression. The hairs may be matted together as ‘paint brush’ lesions; crust or scab formation and accumulations of cutaneous keratinized material forming ‘wart like’ lesions varying in colour from cream to brown. In the early stages, the crusts are very tenacious and attempts to lift them cause pain. Beneath the crusts, there is a granulation tissue and some pus. In the later stages, the dermatitis heals and the crusts separate from the skin but are held in place by penetrating hairs and are easily removed. The thick scabs of dermatophilosis are characteristic.

In goats, lesions appear first on the lips and muzzle and then spread. In sheep, the distribution of lesions is chiefly over the dorsal parts of the body.

**Diagnosis:** Differentiate from Photosensitization.

**Treatment:** Better results are obtained during dry hot weather and in dry climates.

Tetracycline repeated weekly as required, Penicillin streptomycin, Dipping or spraying with 0.2 to 0.5% zinc sulphate, Quaternary ammonium compounds as hand application to individual lesions. The removal of scabs and exudates prior to topical treatment is recommended when practicable

**33. FOOT ROT**

This is an infectious disease of ruminants and swine characterised by inflammation of the sensitive tissues of the feet and severe lameness.

**Aetiology:**

Cattle: *Fusobacterium necrophorum*

Sheep: *Bacteroides nodosus*, *Fusobacterium necrophorum*

Pigs: *Fusobacterium necrophorum*, *Corynebacterium pyogenes* (Secondary infections after trauma)

All ages are susceptible. Transmission is highest where conditions are wet underfoot and in wet humid seasons.

**Clinical findings:** Sudden onset of moderate fever, lameness (usually in one limb) and drop in milk production. The typical lesion occurs in the skin at the top of the interdigital cleft and takes the form of a fissure. Pus is never present in large amount but the edges of the fissure are covered with necrotic material and the lesion has a characteristic odour.

**Diagnosis:** Bacteriological examination is not usually necessary for diagnosis but direct smears of the lesion will usually reveal the causative organisms. The characteristic site, nature and smell of the lesion, the pattern of the disease in the group and the season and climate are usually sufficient to indicate the presence of the disease.

**Treatment:** Parenteral administration of antibiotics or sulphonamides and local treatment of the foot lesion are necessary for best results. Procaine penicillin, oxytetracycline, long-acting tetracycline and sodium sulphadimidine are effective. Scrub the foot, remove all necrotic tissue and apply a local dressing (antibacterial and astringent) under a pad or bandage. A wet pack of 5% copper sulphate solution is effective control.

Control by: Prevention of foot injuries and provision of a footbath containing 5 to 10% solution of formaldehyde or copper sulphate so that animals can walk through it twice daily.

**34. SWINE DYSENTERY**

This is a highly fatal disease of pigs characterised by mucohaemorrhagic diarrhoea and death if untreated for a few days.

**Aetiology:** *Treponema hyodysenteriae*, a large strongly beta-haemolytic Spirochaete, is present in faeces of affected pigs. It is most common in the 7 to 16 week old age group. Morbidity rate within a group of pigs can range from 10 to 75% and if untreated, the case fatality rate can be as high as 50%.

**Clinical findings:** Most commonly, the disease initially affects only a few pigs within the group but spreads over a period of a few days to 2 weeks to involve the majority. Affected pigs show moderate fever, slight depression and some reduction in appetite. The faeces are characteristic. They are only partially formed, usually of a porridge-like consistency and are passed without apparent conscious effort and splatter on contact with the pen floor. Affected pigs commonly defaecate almost anywhere and on anything in the pen. The faeces are light gray to black and on close inspection, much mucus is present and flecks of blood and epithelial casts may be seen. The occurrence of blood in the faeces generally occurs 2 to 3 days after the initial onset of diarrhoea. Affected pigs become progressively dehydrated and their abdomens appear gaunt and sunken. Death usually occurs some days to weeks after the initial onset of signs and results primarily from dehydration and toxaemia. In untreated pigs, the disease may persist for 3 to 4 weeks before clinical recovery.

**Diagnosis:** Swine dysentery must be differentiated from other diseases in which there is diarrhoea in growing pigs. These include Coliform gastroenteritis, Salmonellosis and Hog cholera (The onset and spread within a group in these diseases is much more sudden and rapid than with Swine dysentery and death occurs earlier).

**Treatment:** Treat all pigs within the group by water medication. Severely affected pigs should be treated initially by parenteral injection. Tiamulin through i/m, in drinking or in the feed. Other antimicrobials include Tylosin, carbadox, dimetridazole and ronidazole.

Effective control of swine dysentery is dependent on elimination of the source of the organism from affected pigs, the prevention of re-infection and avoidance of the introduction of carrier animals into herds considered free of the disease. The reduction of the stress of transportation and overcrowding and prevention of a build up of faecal wastes.

**DIAGNOSIS OF BACTERIAL DISEASES**

The clinical and laboratory diagnosis of bacterial diseases is possible with the appropriate laboratory support and suitable samples. For each disease certain samples must be submitted to the laboratory for isolation or demonstration of the specific pathogen.

Clinical and epidemiological findings will usually result in a tentative diagnosis and a rule-out list of possible diagnoses. Particular attention should be given to the following: age of affected animals, distribution in age, length of the course of the disease, morbidity and case fatality rates, seasonal incidence, relationship to other species, recent changes in management, vaccination history, nutritional history, disease in previous years, source of imported animals, known risk factors and the treatments used and the success rate. Clinical, haematological, clinical chemistry and immunological examinations, as appropriate, should be done on as many clinically affected animals as possible. Tissue samples from necropsies and from live animals (biopsies) and discharges, faeces and urine should be submitted for isolation or demonstration of the suspected pathogen. Every precaution must be taken to prevent the spread of the disease to nearby herds or other geographical areas.

**CONTROL OF BACTERIAL DISEASES**

The control of bacterial diseases is dependent on the knowledge of the aetiology and epidemiological characteristics of the disease. Any one or a combination of the following may be effective for the control of a specific bacterial disease.

1. Ensure adequate colostral immunity in the newborn.

2. Identify affected animals, isolate them from the normal animals and treat or dispose of them as indicated, or return them to the herd if they are considered to be safe.

3 Prevent the introduction of infected animals into herds previously considered free of disease.

4. Quarantine all newly acquired animals into a herd for a period of 30 to 60 days. Serological testing may be done on the imported animals.

5. Determine the source of the infection and remove it if possible. Sources include infected animals, feds and water supplies, wildlife, and contaminated environments.

6. Control by the use of mass medication of fed and water supplies may be appropriate with some diseases.

7. Clean and disinfect animal house and grounds regularly. This also prevents the buildup of faecal wastes.

8. Provide an optimal environment for housed animals. This includes adequate ventilation, the prevention of overcrowding and effective removal of manure.

9. Regionally establish primary breeding stock.

10. Vaccinate susceptible animals against endemic diseases. This should be part of a regularly scheduled herd health programme that includes vaccination of the pregnant dam for the enhancement of colostral immunity in the newborn.

11. Avoid stress associated with long transportation, inclement weather and under nutrition.

12. General management and hygiene to include the regular drenching and dipping to control ticks, flies and internal parasites.

**DIAGNOSIS & MANAGEMENT OF RICKETTSIAL DISEASES**

**1. ANAPLASMOSIS**

This is an infectious, haemoparasitic disease of ruminants characterised by severe debility, emaciation, anaemia and jaundice. The disease is usually subclinical in sheep and goats.

**Aetiology:**

*Anaplasma spp*. are obligate intra-erythrocytic parasite.

*Anaplasma marginale* in cattle and wild ruminants.

*A. ovis* in sheep and goats.

*A. centrale* cause mild anaplasmosis in cattle.

The source of infection is always the blood of an infected animal. Transmission is biologically by ticks (*Boophilus spp* in the tropics) but can also be effected mechanically by biting files (the Tabanidae) or blood contaminated fomites.

**Clinical findings:** In cattle, the disease is sub acute especially in young animals after an incubation period of 3 to 4 weeks. Rectal temperature rises slowly and rarely to above 40.50C. It may remain elevated or fluctuate with irregular periods of fever and normal temperature alternating for several days to 2 weeks. The mucous membranes are jaundiced and show marked pallor (due to anaemia), severe debility, emaciation and fertility is impaired (pregnant cows usually abort, depressed testicular function for several months), but there is no haemoglobinuria.

**Diagnosis:** Demonstration of the causative agent in Giemsa or Romanowsky stained blood smear. Differentiate from other causes of haemolytic anaemia.

**Treatment:** Tetracyclines, imidocarb

Blood transfusions are indicated in animals with a PCV less than 15%.

**2. TICK-BORNE FEVER**

This is a disease of ruminants and deer characterised by a high fever, leucopenia and a short clinical course.

**Aetiology:** *Anaplasma phagocytophilia* (*Ehrlichia phagocytophilia*, *Cytoecetes phagocytophilia*). Tick-borne fever is also used for infection with related organism such as *Anaplasma bovis* (formerly *Ehrlichia bovis*). These organisms are transmitted by *Rhipicephalus appendiculatus*, *Amblyomma variegatum* and *Hyalomma truncatum*.

**Clinical findings:** In cattle there is an incubation period of 5 to 9 days followed by a rise in temperature to about 40.50C which persists for 2 to 12 days and for a longer period in late pregnant cows than in lactating cows. The temperature falls gradually and is followed by a secondary febrile period and, in some cases, yet further episodes of pyrexia. During each febrile period, there is a marked fall in milk yield, lethargy and polypnoea, although feed intake is not reduced. Pregnant cattle, in the last two months of pregnancy, and placed on tick-infested pastures for the first time, commonly abort and occasionally animal dies suddenly. Some calves are born alive but they are weak and die.

**Diagnosis:** Demonstration of Anaplasmae in the neutrophils and monocytes as intracytoplasmic inclusion bodies. (There is thrombocytopenia, neutropenia, lymphocytopenia.)

**Treatment:** Tetracyclines, sulphadimidine, trimethoprim sulphadimidine.

**3. HEARTWATER = COWDRIOSIS**

This is a tick-borne disease of domestic and wild ruminants characterised by fever, nervous signs, oedema of the body cavities and diarrhoea.

**Aetiology:** *Ehrlichia ruminantium* (formerly *Cowdria ruminantium*). Transmission is by ticks of the genus Amblyomma.

**Clinical Findings:** The incubation period is 1 to 3 weeks after transmission in tick saliva. Depending on the susceptibility of individual animals and the virulence of the infecting organism, the resulting disease may be peracute, acute, sub acute or mild and in apparent.

**Peracute** cases show only high fever, prostration and death with terminal convulsion in 1 to 2 days. **Acute** cases are more common and have a course of about 6 days. A sudden febrile reaction is followed by in appetence, listlessness and rapid breathing followed by the classical nervous syndrome which is characteristics of heart water. It comprises ataxia, chewing movements, twitching of the eyelids, circling, aggression, apparent blindness, recumbency, convulsions and death. Profuse foetid diarrhoea is frequent.

**Subacute** cases are less severe but may terminate in death in 2 weeks or the animal may gradually recover. The **mild** form is often subclinical and is seen mainly in indigenous animals and wild ruminants with high natural or induced resistance.

The case mortality rate in peracute cases is 100%, in acute cases 50 to 90% and in calves below 4 weeks of age it is 5 to 10%, most animals recover in mild cases.

**Diagnosis:** In enzootic areas, a diagnosis of heart water is the first choice for susceptible animals infected with the tick vector and having a fever of unknown origin. Diagnosis must be based on detection of the rickettsial organism.

Differentiate from anthrax, rabies, sporadic bovine encephalomyelitis, tetanus, cerebral forms of babesiosis, theileriosis, trypanosomosis, hypomagnesaemia and poisoning

**Treatment:** Tetracycline, oxytetracycline, Doxycycline

**CONTROL OF RICKETTSIAL DISEASES**

Arthropod control especially ticks.

Keep flock off tick-infested pastures.

**DIAGNOSIS & MANAGEMENT OF FUNGAL DISEASES**

**1. ASPERGILLOSIS, CANDIDIASIS AND ZYGOMYCOSIS**

Mycoses including Aspergillosis, Candidiasis and Zygomycosis in ruminants and pigs are usually sporadic infections and cause non-specific syndromes because of variation in the organs in which they localize.

**Aetiology:** *Aspergillus spp*., *Candida spp*., *Absidia sp*., *Rhizopus sp*., *Rhizomucor* (*Mucor*) *sp*. all cause systemic mycotic disease. They are not contagious. Each infection arises from the fungal habitat as a saprophyte in organic matter, commonly mouldy hay or straw or moist feeds. A high prevalence can be expected in animals kept indoors in intensive housing units.

**Clinical findings:** Clinical syndromes include pneumonia, encephalopathy, pharyngitis, gastroenteritis, abortion, mastitis and dermatitis.

**Diagnosis:** PCR, ELISA-sophisticated laboratory techniques.

**Treatment:** Amphoterin and Nystatin. Newer drugs include Enilconazole, Fluconazole, Itraconazole and Ketoconazole.

**2. RINGWORM**

This is an infection of the superficial layers of the skin (keratinized epithelial cells) and the hair fibres with \one of a group of dermatophytic fungi.

**Aetiology:** *Trichophyton sp*. and *Microsporum sp*. They occur in all animal species but more commonly where animals are accommodated in dense groups, especially indoors. Direct contact with infected animal is the common method of spread of ringworm, but indirect contact with inanimate objects is probably more important.

**Clinical findings:** In cattle, the typical lesion is a heavy, gray white crust raised above the skin. The lesions are roughly circular and about 3cm in diameter. In the early stages, the surface below the crust is moist, in older lesions the scab becomes detached and pityriasis and alopecia may be the only obvious abnormalities. Itching does not occur and secondary acne is unusual.

Regular ringworm lesions in pigs develop as a centrifugally progressing ring of inflammation surrounding a scabby, alopecic centre.

**Diagnosis:** Demonstration of spores and mycelia in skin scrapings and in culture. Differentiate from sarcoptic and psoroptic mange.

**Treatment:** Remove the crusts by scraping or brushing. Suitable topical applications include Whitfield’s ointment, 10% ammoniated mercury ointment, and solutions of quaternary ammonium compounds.

**CONTROL OF FUNGAL DISEASES**

Hygiene and Prevention of mycotic build-up in pens

**DIAGNOSIS & MANAGEMENT OF PROTOZOAN DISEASES**

**1. BABESIOSIS = TICK FEVER**

This is a disease of ruminants and pigs transmitted by blood sucking ticks and characterised by fever and intravascular haemolysis causing a syndrome of anaemia, haemoglobinaemia and haemoglobinuria.

**Aetiology:**

Cattle: *Babesia bovis*, *B. bigemina*, *B. divergens* and *B. major*

Sheep and Goat: *B. motasi*, *B. ovis*

Pigs: *B. trautmanni*

These babesial parasites are intra-erythrocytic, the distribution of which is governed by the geographical and seasonal distribution of the vectors that transmit them. Tick vectors include *Boophilus sp*., *Rhipicephalus sp*., *Dermacentor sp*., *Hyalomma sp*. and *Ixodes sp*.

**Clinical findings:**

The acute disease generally runs a course of 3 to 7 days and a fever of more than 400C is usually present for several days before other signs become obvious. This is followed by inappetence, depression, polypnoea, weakness and a reluctance to move. Haemoglobinuria is often present; urine is dark-red to brown in colour and produces a very stable froth. Anaemia and jaundice develop especially in more prolonged and severe cases. Muscle wasting, tremors and recumbency develop in advanced cases followed terminally by coma and death. During the fever stage, pregnant cattle may abort and bulls may become sterile for 6 to 8 weeks. The disease is milder in young animals. Cerebral babesiosis is manifested by incoordination followed by posterior paralysis or by mania, convulsions and coma.

**Diagnosis:**

Demonstration of Babesia in a Giemsa-stained smear of capillary blood. Differentiate from Theileriosis, Post parturient haemoglobinuria, Bacillary haemoglobinuria, Leptospirosis and Chronic copper poisoning. Haematology will reveal low RBC, Hb and Platelet levels.

**Treatment:**

Effective drugs include Diminazene aceturate, Imidocarb dipropionate, Amicarbalide diisethionate and Phenamidine. Recent drugs include Parvaquone, Buparvaquone and Alovaquone. Supportive treatment includes blood transfusion and haematinics.

**Control:**

Control of ticks

**2. COCCIDIOSIS**

This is contagious enteritis in all domestic animals characterised by diarrhoea, dysentery, anaemia and inferior growth rates and production.

**Aetiology:**

*Eimeria spp*, *Isospora sp*.

Infection is commonly in young animals and those housed or confirmed in small areas contaminated with oocysts. The source of infection is the faeces of clinically affected or carrier animals, and infection is acquired by ingestion of contaminated feed and water or by licking the hair coat contaminated with infected faeces. Oocysts passed in the faeces require suitable environmental conditions to sporulate. Moist, temperature or cool conditions favour sporulation, whereas high temperatures and dryness impede it.

**Clinical findings:**

The first sign of clinical coccidiosis is the sudden onset of diarrhoea with foul-smelling, fluid faeces containing mucus and blood. The perineum and tail are soiled with blood stained faeces. Severe straining is characteristic, often accompanied by the passage of faeces and rectal prolapse may occur. Anaemia, dehydration or haemoconcentration and reduced growth rate may be seen. Severely affected calves do not quickly regain the body weight losses which occurred during the clinical phase of the disease.

Nervous signs consisting of muscular tremors, hyperaesthesia, clonic-tonic convulsions with ventroflexion of the head and neck and nystagmus, and high mortality rate (80 to 90%) occurs in calves with acute clinical coccidiosis.

**Diagnosis:**

A count of over 5000 oocysts programme of faeces of ruminants is considered significant.

**Treatment:**

Chemotherapeutic agents include Sulphadimidine, Amprolium, Monensin and Lasalocid.

**Control:**

Avoid overcrowding, pens should be kept dry, feed and water troughs should be high enough to avoid heavy faecal contamination. Pasture rotation, proper cleaning and disinfection.

**3. EAST COAST FEVER**

This is an acute, tick-borne disease of cattle characterised by high fever, lymph node enlargement, dyspnoea, weakness, emaciation and a high mortality rate in susceptible breeds.

**Aetiology:**

*Theileria parva* which is found in circulating lymphocytes and in erythrocytes. Its occurrence is related to the distribution of the vector tick, the brown ear tick, *Rhipicephalus appendiculatus*. All susceptible cattle in endemic areas are at the risk of contracting the disease. The morbidity and case fatality rates are very high, approaching 90 to 100% in recently introduced exotic (*Bos taurus*) breeds and in previously unexposed or naïve indigenous cattle. However, indigenous zebu cattle (*Bos indicus*) and African buffalo in endemic areas have a strong resistance to the disease and calf hood mortality is around 5%.

**Clinical findings:**

The basic syndrome associated with *Theileria parva* infection lasts for a few weeks. The incubation period is 1 to 3 weeks, depending on the virulence of the strain and the size of the infecting dose. The first clinical sign is enlargement of lymph nodes in the area draining the site of tick attachment. Later, there is fever, depression, anorexia and a drop in milk production. In inter stages; there may be nasal and ocular discharges, dyspnoea, generalised lymph node enlargement and splenomegaly. In severe cases, diarrhoea occurs sometimes with dysentery. Emaciation, weakness and recumbency lead to death from asphyxia in 7 to 10 days. Terminally, there is often a frothy nasal discharge. Occasional cases of brain involvement occur and are characterised by circling.

Young animals are less susceptible and indigenous breeds and buffaloes are less clinically affected than exotic breeds, but buffaloes are the carriers of the disease.

**Diagnosis:**

Demonstration of *Theileria parva* in lymphocytes and erythrocytes of Giemsa stained blood smears. *T. parva* are difficult to differentiate from other piroplasms, hence the necessity to find schizonts. Blood count will reveal a panleukopenia and thrombocytopenia with little or no anaemia. Differentiate from Babesiosis, Anaplasmosis, Trypanosomosis, Heart water and (Rinderpest, Bovine virus diarrhoea, Malignant catarrhal fever).

**Treatment:**

Tetracycline, Halofuginone, Parvaquone and Buparvaquone.

**Control:**

Control of ticks.

**4. AFRICAN TRYPANOSOMOSIS = NAGANA**

This is a group of diseases in virtually all species of domestic animals caused by the salivarian trypanosomes and characterised by an acute, subacute or chronic course, fever, anaemia, emaciation and a heavy mortality rate.

**Occurrence:** African animal trypanosomosis occur where the tsetse fly vector exists in Africa, between latitude 150N and 290S. *Trypanosoma vivax* can also be transmitted mechanically by biting flies, and thus is also found in parts of Africa free or cleared of tsetse, and parts of Central and South America. Tsetse flies infest 10 million square kilometres of Africa, involving 37 countries. Hence, nagana is today the most important disease of livestock in the continent. The added risk of human infections has greatly affected social, economic and agricultural development of rural communities.

**Aetiology:** Trypanosomes are flagellated protozoan parasites that live in the blood, lymph and various tissues of their vertebrate hosts. *Trypanosoma congolense*, *T. vivax*, and to a lesser extent *T. brucei brucei*, *T. uniforme* and *T. simiae* are other, less common tsetse-transmitted species.

*T. congolense* and *T. vivax* are mainly intravascular parasites while *T. brucei* has an affinity for tissues. Several types of *T. congolense* can be distinguished by molecular biology; the most common and pathogenic one in cattle is the type “savannah”, the other ones (type ‘forest’ and ‘Kilifi’) are less pathogenic and have different host affinity. Mixed trypanosome infections with two or three species are common.

**Sources of infection:** Blood, lymph and other fluids of infected animals.

**Disinfectants/chemicals:** Controlling arthropod vectors and preventing access to host species is important in preventing new infections. Disinfection does not prevent spread of disease (blood-borne parasite).

**Survival:** These agents can only survive in blood, body fluids and tissues of animal hosts and within tsetse flies. Mechanically transmitted *T. vivax* cannot survive long outside the host. Agents disappear within a few hours after death of the vertebrate host.

**Transmission:** Cyclical transmission: trypanosomes are transmitted through the bite of an infected tsetse fly. Tsetse flies get the infection when feeding on an infected animal; after implementation of the parasitic cycle in the fly (15–21 days) it becomes infective and may remain infective for the rest of its life. Transmission occurs in the early stage of the blood feeding, when the fly inject some saliva before sucking the blood of its host.

Mechanical transmission: Biting flies, especially tabanids and stomoxys, but possibly other biting insects (including tsetse flies) are the mechanical vectors of *T. vivax*. Mechanical transmission can occur when interrupted feeding is re-started on a new host; thus it is efficient inside a group of animals but has little chance to occur at distance. Trypanosomosis due to *T. vivax* has thus spread to some areas of Africa free or cleared of tsetse, and also in Central America and South America.

Vertical transmission can occur intra-utero and during partum.

For *T. brucei*, per-oral transmission can occur after birth, when contaminant blood or other fluids can be ingested by the calf. Per-oral transmission is also common for carnivore when eating fresh infected prey.

**Hosts:** Wild animals are the natural hosts. At least 30 species, including greater kudu (*Tragelaphus strepsiceros*), warthog (*Phacohoerus aethiopicus*), bushbuck (*Tragelaphus scriptus*), bush pig (*Potamochoerus porcus*), African buffalo (*Syncerus caffer*), African elephant (*Loxodonta africana*), white rhinoceros (*Ceratotherium simum*), black rhinoceros (*Diceros bicornis*), wild Equidae, lion (*Panthera leo*) and leopard (*Panthera pardus*). These wild animals usually show no clinical signs since host and parasite are in equilibrium. They are enormous reservoir of trypanosomes.

**Biological vector:** Tsetse flies are biological vector. 23 species in sub-Saharan Africa between latitudes 140N and 290S are competent, but primarily *Glossina morsitans*, *G. palpalis* and *G. fusca*. Tsetse flies are grouped according to preferred habitat: savannah, riverine and forest. They remain infected by trypanosomes for life. Trypanosome life cycle involves cyclical development in the tsetse fly, taking up to 3 or more weeks depending on trypanosome species and ambient temperature.

**Domestic animals:** Cattle is the most economically important incidental host. *T. congolense* in cattle, pigs, goats, sheep, horses, and dogs. *T. vivax* in cattle, horse, sheep, and goats. *T. brucei brucei* in cattle, horses, dogs, cats, camels, sheep, goats, and pigs.

**Trypanotolerant breeds:** West African indigenous taurine breeds: N’dama, Baoule, Muturu, Laguna, Somba and Dahomey. East African zebu breeds: Orma Boran and Maasai zebu. Indigenous breeds of small ruminants: West African dwarf sheep and goats, and East African goats.

**Reservoirs:** Many wild animals, trypanotolerant animals, chronically infected animals, tsetse flies.

Laboratory rodents, especially rats and mice are used for revealing subpatent infections of *T. brucei brucei* (and *T. evansi*) infections, but does not work for some *T. congolense* strains and *T. vivax* rarely infects them.

Humans: Sleeping Sickness is caused by *T. brucei gambiense* and *T. brucei rhodesiense*. The animal trypanosomes very rarely cause human infection, but they do share animal reservoirs (wild and domestic) and tsetse vectors.

**Clinical findings:** The basic clinical syndrome appears after an incubation period of generally 8-20 days. *T. congolense* usually becomes apparent in 4–24 days, *T. vivax* in 4–40 days, and *T. brucei brucei* highly variable.

There are variations in the general clinical picture depending on the level of tsetse challenge, the species and strain of the trypanosome, and the breed and management of the host. Nagana in most species is a progressive, but not always fatal disease and the main features are anaemia, tissue damage and immunosuppression. Disease is classically acute or chronic, and is affected by poor nutrition, concurrent diseases, and other stressors. Trypanosomosis in cattle is usually chronic – some may slowly recover but usually relapse when stressed. The most important clinical sign is non-regenerative anaemia, and the most common reason animals are unable to function normally. The major clinical signs are: intermittent fever, anaemia, oedema of the throat and underline, lacrimation, enlarged lymph nodes, abortion, decreased fertility, semen quality progressive deteriorates, loss of appetite, body condition and productivity, early death in acute forms, emaciation and eventual death in chronic forms often after digestive and/or nervous signs. When tsetse challenge is high, morbidity is usually also high. All three species of trypanosomes will eventually cause death in their hosts within 2 to 4 months or longer unless treated.

Mixed infections are common and are usually more severe. Furthermore, intercurrent bacterial, viral or other parasitic infections may mask or complicate the basic clinical syndrome. Immune response to bacterial and some viral vaccines is also depressed.

**Lesions:** Post-mortem lesions are nonspecific and are usually related to anaemia and the prolonged antigen-antibody response: emaciation and serous atrophy of fat, enlarged lymph nodes, liver and spleen, excessive fluid in the body cavities and subcutaneous oedema, petechial haemorrhages, lymphoid tissue may be atrophic in the terminal phases as the animal is too debilitated to mount an immune response, and severe myocarditis is common. *T. brucei brucei* tends to invade tissues to cause inflammation and/or degeneration of multiple tissues, in addition to anaemia.

**Differential diagnosis:** Differentiate acute trypanosomosis with fever from: Babesiosis, Anaplasmosis, Theileriosis (East Coast Fever), Haemorrhagic septicaemia and Anthrax. Differentiate chronic trypanosomosis with anaemia and emaciation from: Helminthosis, Malnutrition, other haemoparasitoses.

**Confirmatory diagnosis:** Direct examination of fresh blood or buffy coat between slide and cover slide can sometimes lead to species identification, based on the epidemiological situation and on typical size, shape and movements of the parasites but fixation and staining is required for a reliable species identification. Direct identification of the parasite in stained thick or thin blood films or wet mounts. Diagnostic sensitivity is increased significantly by concentrating the parasites in the buffy coat layer of a heparinised microhaematocrit tube. The buffy coat is then examined directly at low power (Woo’s method) or in a wet preparation with phase-contrast or dark-ground microscopy (Murray’s method). Buffy coat can also be smeared and stained. Parasitaemia are highly variable during the course of infection: high during early infection, low during chronic infection, and almost nil in healthy carriers.

Other methods include Mini-anion exchange centrifugation technique: simplified method for detecting low parasitaemia by separating salivarian trypanosomes from host red blood cells. This is widely used in human medicine but not suitable for large scale screening of animal samples.

Polymerase chain reaction (PCR): Highly specific and more sensitive test than direct identification. Can identify parasites at subgenus, species or subspecies level. False negatives can occur when parasitaemia are very low (<1 trypanosome per ml), which occurs frequently with chronic infections, or when primers do not recognise all isolates of a particular trypanosome species

Serological tests include:

1. Antibody detection ELISA: very useful for large-scale surveys

2. Indirect fluorescent antibody test

Both tests have high sensitivity and genus specificity, but their species specificity is generally low. At present they can only be used for presumptive diagnosis of trypanosomosis. Antibodies persist on average 3-4 months after curative treatment or self-cure, but may last up to 13 months.

**Treatment:** Drugs such as isometamidium chloride and quinapyramine sulphate and chloride can be used prophylactically during transhumance or high seasonal parasitic pressure.

Curative drugs are diminazene aceturate and quinapyramine methylsulfate which can be used as curative and sanative. Chemoresistance may occur and care must be taken due to the presence of fake drugs on some markets.

No vaccines are available nor likely in the near future because of the ability of trypanosomes to rapidly change variable surface glycoproteins (VSG) in their coats to avoid an effective immune response (antigenic variation). This also leads to establishment of prolonged infections with intermittent parasitaemia. There are estimated to be about 1,000 VSGs, in the trypanosomal coat, which switch genetically as antibodies are produced by the host.

**Control:** Trypanosomosis is a major constraint to ruminant livestock production in many areas of Africa. An integrated approach to control by: Reduce exposure to the vectors by large scale tsetse trapping and pour-on applications; strategic treatment of exposed animals by chemotherapy and chemoprophylaxis.

1. Methods involving land spray of insecticide, bush clearing and elimination of game animals destroy valuable animal resources and also leads to soil erosion; they have been abandoned.

2. Control and eradication of tsetse vector by insecticides: synthetic pyrethroids applied directly on the animal as a spray or pour-on offers great promise; insecticide foot bath are also under evaluation.

3. Sterile male technique: potentially valuable since females mate only once in a lifetime but production facilities are expensive and can only be applied at the end of the eradication campaign, when the density of remaining flies is very low.

4. Pheromone baited tsetse traps that attract and catch tsetse flies: simple, cheap, non-polluting, and readily accepted by local communities.

5. Good husbandry of animals at risk and avoid contact with tsetse flies as much as possible.

6. Introduction and development (selective cross breeding) of trypanotolerant animals. Cattle breeds, like the N’dama and West African Shorthorn, have been in West Africa for centuries and have developed innate resistance to trypanosomes. They are infected by tsetse flies but do not show clinical disease. However, these breeds have not been readily accepted because they are small in size and low in milk producing. Cross breeding is however a common practice.

**DIAGNOSIS & MANAGEMENT OF HELMINTH DISEASES**

**1. PARASITIC GASTROENTERITIS IN RUMINANTS**

This is infestation with the nematodes that commonly occur together in the abomasum and small intestine of ruminants characterised by persistent diarrhoea and wasting.

A combination of infestation by *Trichostrongylus sp*., *Haemonchus sp*. *Ostertagia sp*., *Cooperia sp*., *Nematodirus sp*., *Oesophagostomun sp.* (nodule worm) and *Bunostomum sp.* (hookworm). Each of these helminths can individually cause a separate disease.

**Clinical findings:**

Young animals are more often affected, but adults not previously exposed to infection frequently show signs and succumb. There may be poor weight gain, profuse watery diarrhoea, variable degree of anaemia, hypoproteinaemia, oedema particularly under the lower jaw (bottle jaw), progressive weight loss, weakness, rough hair coat, anorexia, dehydration, sometimes blood and or mucus in the faeces, nodules due to *Oesophagostomun sp*. may be palpated per rectum. Heavy infestations can result in death before clinical signs appear.

**Diagnosis:**

Demonstration of nematode eggs in the faeces.

EPG counts can be low or negative in the presence of large number of immature worms. Also, nematode egg production may be suppressed by immune reaction or previous anthelmintic treatment. The advent of safe and effective broad-spectrum anthelmintics has largely reduced the need for differentiating the genera and species of these parasites.

Differentiate from Malnutrition, Vitamin-mineral deficiency, Coccidiosis, Paratuberculosis, and Chronic fascioliosis.

**Treatment:**

Anthelmintics including Albendazole, Febantel, Fenbendazole, Netobimin, Oxfendazole, Levamisole, Ivermectin, Doramectin, Moxidectin.

**Control:**

Check faecal egg count regularly to confirm that anthelmintic used is effective.

Do not under dose; do not deworm too often (every 2months), rotate anthelmintics annually, quarantine all new stock, practice pasture rotation and do not graze goat on sheep pastures (AR is likely in goats than in sheep).

**2. PARASITIC GASTRITIS IN PIGS**

**Aetiology:**

*Hyostrongylus rubidus*, *Ascarops sp.* and *Physocephalus sexalatus* in the stomach of pigs. These nematodes are almost exclusively confined to outdoor management systems. Ascarops and Physocephalus have lifecycles requiring the dung beetles in which hatching and development to the infective larvae occur.

*H. rubidus* is unlikely to persist in pig houses practicing a reasonable standard of hygiene.

**Clinical findings:**

Heavy infestations may be associated with anaemia, unthriftiness, poor growth, diarrhoea and depraved appetite.

**Diagnosis:**

Demonstration of nematode eggs in faeces.

**Treatment:**

Anthelmintics including Doramectin, Ivermectin, Fenbendazole, Flubendazole, Febantel and Oxibendazole.

**Control:**

Standard hygienic precautions, frequent removal of manure, provision of drainage in outside pens and pasture rotation, control of dung beetle is impracticable in outdoor management and deworm pigs before farrowing.

**3. ASCARID INFESTATION IN RUMINANTS AND PIGS**

**Aetiology:**

*Ascaris suum* in pigs; *Toxocara vitulorum* in cattle; no specific ascarid for sheep and goats.

**Clinical findings:**

Heavy infestations of the intestine with adult ascarid nematodes can cause digestive disturbances, poor growth in young animals, afebrile diarrhoea, intestinal obstruction and lowered resistance to other diseases. Migration of the immature stages through the lungs may results in coughing and dyspnoea.

**Diagnosis:**

Characteristic eggs are usually present in large numbers in the faeces of clinically affected animals.

**Treatment:**

Anthelmintics including Ivermectin, Doramectin, Flubendazole, Fenbendazole, Febantel, Oxibendazole, Thiophanate, Pyrantel tartrate, Levamisole, and Piperazine.

**4. FASCIOLOSIS IN RUMINANTS**

**Aetiology:**

*Fasciola gigantica* in warmer regions of Africa and Asia. Lymnaeid mud snails are intermediate hosts.

**Clinical findings:**

**Acute** fasciolosis in sheep most often occurs as sudden death. If the disease is observed clinically in sheep, it is manifested by dullness, weakness, lack of appetite, pallor of mucosae and conjunctivae, pain when pressure is exerted over the area of the liver and death which may be accompanied by the passage of blood-stained discharges from the nostrils and anus. Acute fasciolosis rarely occurs in cattle.

**Chronic** fasciolosis is apparent when small numbers of metacercariae are ingested over a long period. Affected ruminants lose weight; develop submandibular oedema (bottle jaw) and pallor of the mucosae.

**Diagnosis:**

In acute cases, fasciola eggs will not be present in the faeces as the fluke are still juvenile. There is severe normochromic anaemia, eosinophilia and a severe hypoalbuminaemia. The acute disease can only be confirmed at necropsy.

Differentiate Acute fasciolosis from Haemonchosis, Infectious necrotic hepatitis, Anthrax and Enterotoxaemia.

Differentiate Chronic fasciolosis from Vitamin-mineral deficiencies, Internal parasitism and Paratuberculosis.

**Treatment:**

Triclabenzazole, Albendazole, Netobimin, Closantel, Clorsulon, Nitroxynil, and Oxyclozanide

**Control:**

Control of snails and strategic use of anthelmintics.

**END OF CLASS ASSIGNMENT**

Oesophagostomosis = nodule worm disease in ruminants and pigs; Strongyloides infestation; Trichuris infestation; Lungworm (*Dictyocaulus sp*.); Thelezia (Eyeworm);

Kidney worm disease in pigs (*Stephanurus dentatus*);

Stomach fluke disease (*Paramphistomum sp*.);

Tapeworm infestation (Moniezia, Thysaniezia, Avitellina, Stilesia, Thysanosoma)

**DIAGNOSIS & MANAGEMENT OF VIRAL DISEASES**

**1. CONTAGIOUS ECTHYMA (ORF, SORE MOUTH)**

Contagious ecthyma also known as orf, scabby mouth or contagious pustular dermatitis, is a specific dermatitis of sheep and goat.

**Aetiology:** Orf is caused by the Orf virus which is a poxvirus in the genus Parapoxvirus (family Poxviridae). Infection can be from environmental persistence of the virus or from contact with infected animals and inanimate objects. Morbidity rates in outbreaks may approach 100% and case fatality rate from 5 to 15%. The deaths that occur are due to the extension of lesions in the respiratory tract and if severely affected animals are not provided with adequate care and support.

**Clinical Findings:** The disease causes unthriftiness, varying degrees of pain and some economic losses. The first lesions develop at the oral mucocutaneous junction, usually at the oral commissures and are accompanied by swelling of the lips. Lesions develop initially as papules and pustules, the stages which are not usually initially observed, and progress to raised moderately proliferative area of granulation and inflammation covered with a thick, tenacious scab. From here, they spread on to the muzzle and nostrils, the surrounding haired skin and, to a lesser extent, on to the buccal mucosa. They may appear as discrete, thick scabs or coalesce and be packed close together as a continuous plaque. Fissuring occurs and the scabs are sore to the touch. They crumble easily but are difficult to remove from the underlying granulation. Affected animals suffer a severe setback because of restricted sucking and grazing.

**Diagnosis:** A tentative diagnosis may be made based on the characteristic clinical findings of epidermal proliferation, encrustation, multifocal lesions in the mouth and nostril and weight loss.

**Treatment:** There is no treatment. In the absence of a specific treatment, the animal involved must be isolated, scabs removed and ointments or astringent lotions applied. In addition, provision of soft and palatable food as well as warmth is recommended.

**Control:** Contagious Ecthyma can be controlled by vaccination at 6 to 8 weeks of age, although this vaccine is currently not available in Nigeria. However, sheep and goat can be effectively immunized with a live virus vaccine derived from scab material administered to ewe/doe at about two months before lambing/kidding by scarification on the thigh.

**2. CLASSICAL SWINE FEVER = HOG CHOLERA**

Classical Swine Fever, also known as Hog Cholera, is a highly infectious pestivirus infection of pigs characterised clinically by an acute highly fatal disease and pathologically by lesions of a severe viraemia. The chronic or in apparent disease also occur, including persistent congenital infection in newborn pigs infected during foetal life. The disease has been responsible for large economic losses in the swine industry worldwide

**Aetiology:** The virus of the family Flaviviridae, belongs to the genus Pestiviridae and related to the bovine diarrhoea virus. There is only one antigenic type but a number of strains of variable virulence. All breeds and ages are susceptible and adults are more likely to survive an acute infection. The disease usually occurs in epidemics, often with a morbidity of 100% and a case fatality rate approaching 100% when a virulent strain of the virus infects a susceptible population. The source of virus is always an infected pig or its products and the infection is usually acquired by ingestion but inhalation is also a possible portal of entry. Direct animal to animal contact is the most important method of spread. Infected pigs shed a large amount of the virus in all normal secretions- nasal, salivary, urinary and faecal.

**Clinical Findings:** In the peracute and acute disease, clinical signs usually appear 5 to 10 days after infection. At the beginning of an outbreak, young pigs may die peracutely without evidence of clinical signs.

Acute cases are most common. Affected pigs are depressed, do not eat and stand in a drooped position with their tails hanging. They are disinclined to move and when forced, do so with a swaying movement of the hindquarters. They tend to lie down and burrow into the bedding, often piled one on top of the other. Prior to the appearance of other signs, a high temperature (40.5 - 41.50C) is usual. Constipation followed by diarrhoea and vomiting also occur. Later, a diffuse purplish discoloration of the abdominal skin occurs. Small areas of necrosis are sometimes seen on the edges of the ears, on the tail and lips of the vulva. A degree of conjunctivitis is usual and in some pigs the eyelids are stuck together by dried, purulent exudate. Nervous signs often occur in the early stages of illness and include circling, incoordination, muscle tremor and convulsions. Death can be expected 5 to 7 days after the commencement of illness. Reproductive failure including high incidence of abortion, low litter size, mummification, stillbirth and anomalies of piglets may occur.

The chronic disease is as a result of low virulence strains of the virus. A chronic form occurs in field outbreaks and occasionally after serum-virus simultaneous vaccination. The incubation period is longer than normal and there is depression, anorexia, persistent mild fever, unthriftiness, the appearance of characteristic skin lesions including alopecia, dermatitis, blotching of the ears and a terminal, deep purple coloration of the abdominal skin. Pigs may apparently recover following a short period of illness but subsequently relapse and die if stressed.

**Diagnosis:** A tentative diagnosis may be made based on clinical and post mortem findings. A highly infectious, fatal disease of pigs with a course of 5 to 7 days in a group of unvaccinated animal should arouse suspicion.

Differentiate from Salmonellosis (accompanied by enteritis and dyspnoea), Erysipelas (diamond skin lesions and ecchymotic subserous haemorrhages), Pasteurellosis (respiratory signs and pleuropneumonia) and African swine fever (greater severity). Confirmation is by PCR, ELISA, virus isolation, fluorescent antibody techniques and agar gel precipitation test.

**Treatment:** Hyper-immune serum

**Control:** By eradication and vaccination. In areas where effective barriers to re-introduction of the disease can be established, eradication of the disease by slaughter methods is feasible and usually desirable. In contrast, in areas where the structure and economics of the pig industry require considerable within-country and across border movement of pigs, it may not be practical or economical feasible to institute a slaughter eradication programme. In these areas, control by vaccination is the approach of choice.

**3. AFRICAN SWINE FEVER**

African Swine Fever is a peracute, highly infectious and fatal viral infection of pigs indistinguishable on the field from Classical Swine Fever.

**Aetiology:** The virus is a DNA virus of the genus Asfariviridae. It is a major threat to pig-producing countries especially in African, western European and Caribbean countries. Transmission may be by direct contact with infected pigs or by argasid tick from wild to domestic pigs.

**Clinical Findings:** In the acute form, there is high morbidity and high case fatality rate and the animal dies in an acute state of shock characterised by a disseminated intravascular coagulation with multiple haemorrhages in all tissues. The incubation period varies from 5 to 15 days. A high fever (40.50C) appears abruptly and persists without other apparent signs for about 4 days. The fever then subsides and the pig show marked cyanotic blotching of the skin (purplish skin), depression, anorexia, huddling together, disinclination to move, weakness and incoordination. Other signs may include nasal and ocular discharges, diarrhoea, vomiting, abortions and death in a few days.

The subacute and chronic forms are characterised by fever, depression and lethargy. Animals recover in few weeks but remain persistently infected. Chronic cases are intermittently pyrexic and become emaciated with soft oedematous swelling over joints and mandible.

**Diagnosis:** This is based on clinical signs (which are not distinguishable in the field from classical swine fever) and post mortem examination. Confirmation is by fluorescent antibody techniques, PCR and ELISA.

**Treatment:** There is no treatment.

**Control:** ASF can be eradicated by:

1. Slaughter affected pigs and their ticks as quickly as possible.

2. Serological surveillance of all sows and boars in every herd.

3. Improvement of sanitary conditions of housing.

4. Improved hygiene (safe disposal of manure, vehicle disinfection).

5. Veterinary control of all swine and livestock transfers.

6. Health certification of every animal used for herd herd replacement.

7. Destruction of every seropositive animal.

The control and eradication of ASF is difficult in Nigeria because:

1. Lack of effective vaccine.

2. Transmission of the virus in fresh meat and offals.

3. Persistent infection in warthogs and bush pigs.

4. Clinical similarity of ASF and classical swine fever.

5. Effective barriers to re-introduction of the disease cannot be established.

**4. SWINE INFLUENZA**

Swine influenza is an important cause of broncho-interstitial pneumonia throughout all pig keeping areas of the world. Real problems are associated with the changing viruses that cause disease.

**Aetiology:** Classical swine influenza is associated with Influenza A virus subtypes H1N1, H1N2 and H3N2 belonging to the genus Orthomyxovirus (family Orthomyxoviridae). The natural reservoir of influenza A virus is aquatic birds. Various subtypes have been established in other species, such as influenza A H1N1 viruses which infect human and different animal species. The influenza viruses may be transmitted between humans and pigs. Swine are the sole animals known to be susceptible to influenza A viruses of human, swine and avian origin. The primary route of infection is through pig to pig contact via the nasopharyngeal route.

**Zoonotic Implications:** Swine influenza pose a significant health risks to humans.

**Clinical Findings:** Swine influenza is essentially a herd disease. After an incubation period of 1 to 7 days, the disease appears suddenly, with a high proportion of the herd showing fever (up to 41.50C), anorexia and severe prostration. The animal is disinclined to move or rise because of muscle stiffness and pain. Laboured, jerky breathing is accompanied by sneezing and a deep, painful cough which often occurs in paroxysms. There is congestion of the conjunctivae with a watery ocular and nasal discharge. Sometimes, there is open mouthed breathing and dyspnoea especially if the pigs are forced to move. Morbidity is usually 100% but mortality is rarely above 1%. After a course of 4 to 6 days, signs disappear rapidly depending partly on the level of colostral antibody. However, there is much loss of weight, which is slowly gained.

**Diagnosis:** This is based on clinical signs. Confirmation is by haemagglutination inhibition test, ELISA, viral detection and PCR. Differentiate from Enzootic pneumonia (more insidious and chronic course), Hog cholera (less respiratory involvement and high mortality rate), Inclusion body rhinitis (in piglets) and Atrophic rhinitis (longer course accompanied by distortion of facial bones).

**Treatment:** There is no treatment. In the absence of a specific treatment, administer antimicrobials for secondary infections; provide comfortable, well-bedded quarters, free from dust and good nursing.

**Control:** Vaccination and biosecurity.

**5. PESTE DES PETITS RUMINANTS (PPR)**

This is a highly contagious and infectious viral disease of domestic and wild small ruminants.

**Aetiology:** The virus which causes PPR is called Peste des petits ruminants Virus (PPRV) which belong to the morbili virus group of the paramyxovirus family. Other members of this group which are closely related to PPRV include: Rinderpest virus of cattle and buffaloes, Measles virus of humans, Distemper virus of dogs and some wild carnivores, and Morbillivirus of aquatic mammals.

Genetic characterization of PPR virus strains has recognised 4 groups (3 groups from Africa and 1 from Asia. The epidemiological significance of these grouping is not clear at present.

**Geographical Distribution of PPR:** PPR infection has been recognised in many African countries especially those that lie between the Atlantic Ocean and the red sea. The affected areas extend North of Egypt and South to Kenya in the East and Gabon in the West. The disease is not recognised in most Northern and Southern African countries. Recently the disease has been reported in the near East and the Arabian peninsula in countries that include; Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirate and Yemen. Outbreaks also common in India, Nepal, Bangladesh and Pakistan.

**Clinical Findings:** Clinical signs appear 2-6 days post natural infection with the virus (incubation period). This is followed by sudden onset of fever of between 40 and 410C. There is marked depression and the animal appears sleepy. Hair stands erect giving a bloated appearance especially in short haired breeds. After which clear watery discharges from the eyes, nose and mouth later becomes thick and yellow (mucopurulent) due to secondary Bacterial infections. This discharge causes wet chin and hairs below the eyes which becomes dry causing matting together of the eyelid, obstruction of the nose and difficulty in breathing.

One or two days post fever, the mucous membrane of the mouth and the eye becomes very reddened and congested. The oral cavity epithelial necrosis causes small pin point greyish areas to appear on the gum, dental pad, palate, lips, inner aspect of the cheek and the upper surface of the tongue. These areas increase in number and size and join together. The lining of the mouth becomes pale and coated with dead cells. Underneath this dead surface cells are shallow erosions. Gentle rubbing across the gum and palate with a finger may yield a foul smelling material containing shreds of epithelial tissues. Similar lesions may be seen in the mucous membrane of the nose, vulva and vagina.

Affected animals resist attempt to open their mouth because of the lesion and associated pains. This results in refusal of food and water.

Diarrhoea appears 2 to 3 days after fever, although in early or mild cases, it may not be obvious. Faeces initially are soft, watery, foul smelling and contain blood streaks and pieces of dead gut tissues.

Affected sheep and goats breathe fast, thus exhibiting rocking movements with both the chest and the abdominal wall moving as the animal breaths.

Severely affected animal show difficulty and noisy breathing marked by extension of the head and neck, dilation of the nostrils, protrusion of the tongue and soft painful cough. Up to 100% of the animals in a flock may be affected in a PPR outbreak with between 20 and 90% mortality.

**Diagnosis:** A tentative diagnosis of PPR can be made on epidemiological and clinical features. In the event of history, oral discharges, diarrhoea, deaths with prominent breathing problems in a sheep and goat flock, no history of contact with cattle and most affected animals in the flock are adolescents; a suspicion of PPR may be made. Differentiate from: Contagious Ecthyma (ORF) (no diarrhoea in ORF, whereas in PPR, there is diarrhoea and occular discharges). Pasteurellosis caused by *Mannheimia haemolytica* (no diarrhoea, no oral lesions and the number of goats affected are very low). Coccidiosis (no oral lesions, no coughing and mortality is very low). *Contagious caprine pleuropneumonia* (CCPP) (no oral lesions and no diarrhoea).

**Laboratory Confirmation:** Viral isolation from blood, lymph nodes around the lungs (mediastinal lymph nodes), spleen and alimentary tract lymph node (mesenteric lymph nodes).

Detection of viral antigen by Agar gel immunodiffusion test (AGIDT) is a simple test but this test does not discriminate between PPR and Rinderpest; therefore a further test is required.

Viral antigen can be detected by ELISA technique very rapidly. This is sensitive and differentiates between PPR and Rinderpest.

**Treatment:** Antibiotic treatment to prevent secondary bacterial infection: Penicillin -Streptomycin, Enrofloxacin

CONTROL

Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Until recently, the most practical vaccination against PPR made use of tissue culture rinderpest vaccine. Recently, a homologous PPR vaccine has been developed and the vaccine is available in NVRI Vom.

The appearance of clinical PPR may be associated with any of the following:

1. History of recent movement or gathering together of sheep and/or goats of different ages with or without associated changes in housing and feeding.

2. Introduction of recently purchased animals; contact in a closed/village flock with sheep and/or goats that had been sent to market but returned unsold.

3. Change in weather such as the onset of the rainy season (hot and humid) or dry, cold periods (for example the harmattan season in West Africa); contact with trade or nomadic animals through shared grazing, water and/or housing.

4. A change in husbandry (e.g. towards increased intensification) and trading practices.

5. In endemic areas, most of the sick and severely affected animals are over four months and up to 18 to 24 months of age.

**NECESSARY PRECAUTIONARY MEASURES TO PREVENT OUTBREAK OF PPR BY A FARMER WHO WANTS TO ESTABLISH A GOAT OR SHEEP FARM**

1. Prior to acquisition of animals, a suitable house must be provided to prevent exposure of these animal to adverse weather conditions especially extreme cold / Harmattan.

2. Avoid buying of new stock of sheep/ goats from open market where there are congregation of different animals both the sick and healthy ones. Close contact in the market aid easy transfer of disease from the sick to healthy ones.

3. It is recommended to get new stock from a small holding goat farmers who has adequate information and history of their animals.

4. Gradual change of feed from old to new feed.

5. Acquisition of animals should be done in batches.

6. From the first day of arrival on the farm, animals should be placed on Antibiotic and multivitamins to reduce the stress of change of environment, after which PPR homologous vaccine should be administered to all apparently healthy animals.

**6. RINDERPEST**

Cattle plague, also known as Rinderpest, is a contagious disease that principally affects cattle, but occasionally can also affect sheep, goats, camels, certain wild ruminants and pigs. The disease is characterized by severe inflammation and necrosis of the mucous membrane of the digestive tract. The disease has been associated with high mortality and it is an OIE (Office International des Epizooties also known as World Organization for Animal Health) Class A disease reflecting its serious economic impact.

**Aetiology:** The Rinderpest virus (RPV) is a RNA Morbillivirus, closely related to the PPR, Measles and Canine distemper viruses. Despite its extreme virulence, the virus is particularly fragile and is quickly inactivated by heat, desiccation and sunlight. The disease is mainly spread by direct contact and by drinking contaminated water, although it can also be transmitted by air.

**Clinical Findings:** Mortality rates during outbreaks are usually extremely high, approaching 100% in immunologically naive populations. Initial symptoms include fever, loss of appetite, and nasal and eye discharges. Subsequently, irregular erosions appear in the mouth, the lining of the nose and the genital tract. Acute diarrhoea, preceded by constipation, is a common feature as well. Most animals die 6 to 12 days after the onset of these clinical signs.

The temperature rises in the early stages. The animal is off its food, dull and the coat is starry. Sometimes shivering is noticed. The breathing is quick: a watery or mucous discharge flows from the eyes and nostrils; in the latter case there may be a slight amount of blood in the discharge. In milking cows, the secretion of milk is diminished or arrested. The membrane of the nostrils reddens, and an eruption, like grains of bran, appears in the nostrils and inside the lips and cheeks. This eruption is often followed by distinct ulceration. The animal is at first constipated, but in the later stages diarrhoea often sets in. In this case the faeces has a foul smell and is often tinged with blood. The animal rapidly loses condition and the disease usually terminates fatally in from 6 to 10 days. Cattle plague does not attack single animals in a herd, but spreads rapidly from one to another.

**Diagnosis:** This is based tentatively on the clinical signs and post mortem lesions. Rising antibody titre in paired sera, serology neutralization, CFT, Agar gel diffusion tests and isolation of virus from body discharges and excretion have been used to confirm the disease.

**Treatment:** No specific treatment but supportive therapy with antibiotics and fluids may reduce mortality. Annual vaccination with the Tissue culture Rinderpest vaccine (TCRV).

Control of outbreaks by quarantine and ring vaccination.

**7. FOOT AND MOUTH DISEASE**

This is an acute, highly contagious viral disease of cloven footed animals but most important in cattle and pigs.

**Aetiology:** Apthovirus of the Picona virus group. Seven serotypes of the virus have been identified and these include the following: A, O, C, SAT 1, SAT 2, SAT 3 as well as ASIA type 1 serotype. However these serotypes further comprises of over 50 sub types. Recently six new strains have been discovered in recent outbreaks in Europe. The major route of transmission of FMD is through aerosol. Other routes include contact with infective material or discharges, through carrier animals, by ectoparasites, experimentally by artificial insemination and use of FMD infected carcasses as meat scraps in pig farming.

**Clinical Findings:** Initial lesions are seen on the lingual mucosa which later develops into vesicles. Dullness, anorexia and pyrexia may precede the appearance of the vesicles. Vesicles extend to the nares, buccal cavity and between the hooves which result in lameness especially in pigs. Hoof deformities may cause a persistent lameness even after remission of other signs. Drooling of saliva as a result of lesions in the oral cavity may be seen.

Pregnant animals may abort and mortality is often high among calves and piglets.

Mortality may be up to 100% especially in calves but rarely exceeds 1% in adults.

Mammary gland involvement may lead to mastitis.

In young calves, there is involvement of the heart leading to abnormal heart sounds.

**Diagnosis:** History, clinical signs and PM lesions will give a tentative diagnosis. Virus neutralization, Agar gel precipitation and ELISA will have to be undertaken to confirm the tentative diagnosis.

**Management and Control:** In FMD free counties, infected animals are slaughtered and carcasses burnt or buried with Calcium oxide and whole premises decontaminated. Antibiotic therapy both systemic and topical for local lesions on the legs and oral cavity to minimize secondary complications. Quarantine, vaccination and subsequent release of in contact animal are undertaken, however such animal serve as carrier for the disease.

**AFRICAN ANIMAL TRYPANOSOMOSIS**

**(Nagana, Tsetse Disease, Tsetse Fly Disease)**

**Definition**

African animal trypanosomosis (AAT) is a disease complex caused by tsetse-fly-transmitted *Trypanosoma congolense*, *T. vivax*, or *T. brucei brucei*, or simultaneous infection with one or more of these trypanosomes. African animal trypanosomosis is most important in cattle but can cause serious losses in pigs, camels, goats, and sheep. Infection of cattle by one or more of the three African animal trypanosomes results in subacute, acute, or chronic disease characterized by intermittent fever, anemia, occasional diarrhea, and rapid loss of condition and often terminates in death. In southern Africa the disease is widely known as nagana, which is derived from a Zulu term meaning "to be in low or depressed spirits"— a very apt description of the disease.

**Etiology**

African animal trypanosomosis is caused by protozoa in the family Trypanosomatidae genus *Trypanosoma*. *T. congolense* resides in the subgenus *Nannomonas*, a group of small trypanosomes with medium-sized marginal kinetoplasts, no free flagella, and poorly developed undulating membranes. In east Africa, *T. congolense* is considered to be the single most important cause of AAT. This trypanosome is also a major cause of the disease in cattle in West Africa. Sheep, goats, horses, and pigs may also be seriously affected. In domestic dogs, chronic infection often results in a carrier state.

*T. vivax* is a member of the subgenus *Duttonella*, a group of trypanosomes with large terminal kinetoplasts, distinct free flagella, and inconspicuous undulating membranes. *T. vivax* is a large (18-26 μm long) monomorphic organism that is very active in wet-mount blood smears. Cattle, sheep, and goats are primarily affected. Although this organism is considered to be less pathogenic for cattle than *T. congolense*, it is nevertheless the most important cause of AAT in West African cattle. This trypanosome readily persists in areas free of tsetse flies (for example, in Central and South America and in the Caribbean), where it is transmitted mechanically by biting flies or contaminated needles, syringes, and surgical instruments.

*T. brucei brucei* resides in the subgenus *Trypanozoon. T. b. brucei* is an extremely polymorphic typanosome occurring as short, stumpy organisms without flagella, long slender organisms with distinct flagella, and intermediate forms that are usually flagellated. Horses, dogs, cats, camels and pigs are very susceptible to *T.* *b. brucei* infection. Infection of cattle, sheep, goats and sometimes pigs results in mild or chronic infection.

**Host Range**

Cattle, sheep, goats, pigs, horses, camels, dogs, cats, and monkeys are susceptible to AAT and may suffer syndromes ranging from subclinical mild or chronic infection to acute fatal disease. Rats, mice, guinea pigs, and rabbits are useful laboratory species.

More than 30 species of wild animals can be infected with pathogenic trypanosomes, and many of these remain carriers of the organisms. Ruminants are widely known to be active reservoirs of the trypanosomes. Wild Equidae, lions, leopards, and wild pigs are all susceptible and can also serve as carriers of trypanosomes.

**Geographic Distribution**

The tsetse-fly-infested area of Africa extends from the southern edge of the Sahara desert to Angola, Zimbabwe, and Mozambique.

**Transmission**

In Africa, the primary vector for *T. congolense*, *T. vivax*, and *T. b. brucei* is the tsetse fly. These trypanosomes replicate in the tsetse fly and are transmitted through tsetse fly saliva when the fly feeds on an animal. The three main species of tsetse flies for transmission of trypanosomes are *Glossina morsitans*, *G. palpalis*, and *G. fusca*.

Trypanosomosis is also mechanically transmitted by tsetse and other biting flies through the transfer of blood from one animal to another. The most important mechanical vectors are flies of the genus *Tabanus*, but *Haematopota, Liperosia, Stomoxys*, and *Chrysops* flies have also been implicated. In Africa, both *T. vivax* and *T. b. brucei* have spread beyond the "tsetse fly belts", where transmission is principally by tabanid and hippoboscid flies.

The vector for *T. vivax* in the Western Hemisphere remains unknown, but several species of hematophagous (especially tabanid and hippoboscid) flies are believed to serve as mechanical vectors.

**Incubation Period**

The incubation period for *T. congolense* varies from 4 to 24 days; for *T. vivax*, from 4 to 40 days; and for *T. b. brucei*, from 5 to 10 days.

**Pathogenesis**

Initial replication of trypanosomes is at the site of inoculation in the skin; this causes a swelling and a sore (chancre). Trypanosomes then spread to the lymph nodes and blood and continue to replicate. *T. congolense* localizes in the endothelial cells of small blood vessels and capillaries. *T. b. brucei* and *T. vivax* localize in tissue. Antibody developed to the glycoprotein coat of the trypanosome kills the trypanosome and results in the development of immune complexes. Antibody, however, does not clear the infection, for the trypanosome has genes that can code for many different surface-coat glycoproteins and change its surface glycoprotein to evade the antibody. Thus, there is a persistent infection that results in a continuing cycle of trypanosome replication, antibody production, immune complex development, and changing surface-coat glycoproteins.

Immunologic lesions are significant in trypanosomosis, and it has been suggested that many of the lesions (e.g., anemia and glomerulonephritis) in these diseases may be the result of the deposition of immune complexes that interfere with, or prevent, normal organ function. The most significant and complicating factor in the pathogenesis of trypanosomosis is the profound immunosuppression that occurs following infection by these parasites. This marked immunosuppression lowers the host's resistance to other infections and thus results in secondary disease, which greatly complicates both the clinical and pathological features of

trypanosomosis.

**Clinical Sign*s***

Because simultaneous infections with more than one trypanosome species are very common, and simultaneous infection with trypanosomes and other hemoparasites (*Babesia* spp., *Theileria* spp., *Anaplasma* spp., and *Ehrlichia* spp.) frequently occurs, it is difficult to conclude which clinical signs are attributable to a given parasite.

The cardinal clinical sign observed in AAT is anemia. Within a week of infection with the haematic trypanosomes (*T. congolense* and *T. vivax*) there is usually a pronounced decrease in packed cell volume, hemoglobin, red blood cell, and white blood cell levels, and within 2 months these may drop to below 50 percent of their pre-infection values. Also invariably present are intermittent fever, edema, lethargy and loss of condition. Abortion may be seen, and infertility of males and females may be a sequel. The severity of the clinical response is dependent on the species and the breed of affected animal and the dose and virulence of the infecting trypanosome. Stress, such as poor nutrition or concurrent disease, plays a prominent role in the disease process, and under experimental conditions, where stress may be markedly reduced; it is difficult to elicit clinical disease.

The marked immunosuppression resulting from trypanosome infection lowers the host's resistance to other infections and causes in secondary disease, which greatly complicates both the clinical and pathological features of trypanosomosis.

**Gross Lesions**

No pathognomonic change is seen in AAT. Anemia, edema, and serous atrophy of fat are commonly observed. Subcutaneous edema is particularly prominent and is usually accompanied by ascites, hydropericardium, and hydrothorax. The liver may be enlarged, and edema of lymph nodes is often seen in the acute disease, but they may be reduced in size in the chronic disease. The spleen and lymph nodes may be swollen, normal, or atrophic. Necrosis of the kidneys and heart muscle and subserous petechial hemorrhages commonly occur. Gastroenteritis is common, and focal polioencephalomalacia may be seen. A localized lesion (chancre) may be noted at the site of fly bite, especially in goats.

The lesions caused by the trypanosomes in susceptible host species vary considerably, depending on the species and strain of trypanosome and the species and breed of host animal affected. The haematic trypanosomes (*T. congolense* and *T. vivax*) cause injury to the host mainly by the production of severe anemia, which is accompanied in the early stages of the disease by leukopenia and thrombocytopenia. In the terminal stages of the disease caused by the haematic trypanosomes, focal polioencephalomalacia probably results from ischemia due to massive accumulation of the parasites in the terminal capillaries of the brain.

The lesions resulting from *T. b. brucei* (a tissue parasite) are remarkably different from those seen with the haematic trypanosomes. Anemia is an important lesion, but much more dramatic are the inflammation, degeneration, and necrosis resulting from cellular invasion of various organs. Marked proliferative changes reflecting immunologic response are observed in most body tissues.

**Diagnosis**

**Field Diagnosis**

Trypanosomiasis should be suspected when an animal in an endemic area is anaemic and in poor condition. Confirmation depends on the demonstration of the organism in blood or lymph node smears.

In the early phases of infection, especially with *T. vivax* and *T. congolense*, the parasite can readily be observed by microscopic examination of a wet-mount of blood slides. Thick blood films and stained with Giemsa are also a good technique, but in thin fixed blood films, which are favored for species identification, the parasites may be hard to demonstrate. When parasitemia is low, smears of buffy coat (obtained by microhematocrit centrifugation) can be useful for demonstration of the parasites. Because *T. congolense* tends to associate with the erythrocytes, it is essential that buffy coat and adjacent erythrocytes be included in the smear to ensure demonstration of the parasite.

Stained lymph node smears are a very good method for diagnosis, especially for *T. vivax* and *T. b. brucei*. In chronic *T. congolense* infection, the parasites localize in the microcirculation of the lymph nodes and in other capillary beds, allowing diagnosis by examination of lymph node smears or smears made with blood collected from the ear. Early in infection, blood smears are optimal for the demonstration of *T. congolense*.

These conventional techniques of microscopic examination for the presence of trypanosomes are still widely used, but newer and far more sensitive methods are beginning to supplant them. The antigen-detecting enzyme-linked immunosorbent assay is extremely sensitive for the detection of trypanosomosis in cattle and goats, and species-specific DNA probes have been shown to detect simultaneous infection of cattle with *T. vivax*, *T. b. brucei*, and *T. congolense* when conventional methods revealed only single infections.

**Specimens for the Laboratory**

To perform the preceding and more sensitive procedures, the following specimens should be submitted to the laboratory from several animals: serum, blood with the anticoagulant EDTA, dried thin and thick blood smears, and smears of needle lymph node biopsies.

**Control and Eradication**

**Vector Control**

Fly eradication and drug prophylaxis are the only effective trypanosomosis control methods now available. The recent introduction of odor-baited targets impregnated with insecticides is proving promising as a means of reducing the tsetse fly.

**Chemotherapy and Chemoprophylaxis**

Chemoprophylactic drugs: Isometamidium chloride, which comes under the trade names Samorin, Trypamidium, and M&B 4180A, is excellent for the prophylaxis of all three African animal trypanosomes, and gives protection for 3-6 months. The development of resistance to this drug has been reported in both east and West Africa. Homidium bromide has also been found to be an effective chemophrophylactic drug in Kenya, and the newly introduced arsenical Cymelarsan is effective in treatment of *T. b. brucei* infection.

A very widely used chemotherapeutic drug is diminazine aceturate (Berenil), which is effective against all three African animal trypanosomes. The isometamidium drugs are also excellent chemotherapeutic agents as are the quaternary ammonium trypanocides Antrycide, Ethidium and Prothidium.

**Immunization**

No vaccine is currently available for African animal trypanosomosis.

**Trypanotolerance**

It has long been recognized that certain breeds of African cattle are considerably more resistant to African trypanosomosis that others. This is especially true of the

West African short-horned cattle (Muturu, Baoule, Laguna, Samba, and Dahomey) and the N'Dama, which is also of West Africa.

**Public Health**

The three AAT trypanosomes are considered to be nonpathogenic for humans. *T. b. brucei*, although not causing human disease, is closely related to *T. b. gambiense* and *T. b. rhodesiense*. The latter is the cause of human sleepingsickness, a very debilitating and often fatal disease considered to be of majorpublic health significance in 36 sub-Saharan countries of west, central, and eastAfrica.