THE UNITED REPUBLIC OF TANZANIA

MINISTRY OF LIVESTOCK AND FISHERIES

STANDARD TREATMENT GUIDELINES FOR ANIMAL DISEASES IN TANZANIA

Food and Agriculture Organization of the United Nations

FEBRUARY 2022

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FOREWORD

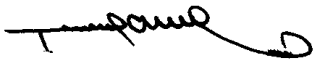
The Standard Treatment Guidelines for animal diseases are prepared as a tool to assist and guide veterinarians, paraprofessionals, and other animal healthcare workers who prescribe at facilities in providing quality treatments to animals. They are designed to guide treatment choices and as a reference book to help in the overall management of ruminants and poultry and will be used at all levels within the animal health system, both public and private. Furthermore, they list the preferred treatments for common animal health problems experienced by people in animal health particularly in ruminants and poultry.

The potential benefits of introducing standard treatments guidelines in animal health include consistency among prescribers, reduced confusion, and increased compliance and for policy makers, it provides a method to control animal diseases more efficiently and serve as a basis to assess and compare quality of care.

Standard treatments guidelines focus on critical aspects of therapeutic processes like careful identification of signs and symptoms, correct diagnosis and proper use of drugs or non-drug treatments that will truly benefit the farmer and the animal.

The existence of these treatments will have a major impact on the consistency, effectiveness and economy of prescribing. These guidelines will have to undergo periodic reviews in view of dynamics that occurs in animal health systems globally.

The Guidelines are intended to be used by all veterinary professionals and paraprofessional in Tanzania. The system rests on the foundation that, all veterinary service providers will commit themselves to meeting the requirement for diagnosis.



Tixon T. Nzunda
Permanent Secretary (Livestock)

ACKNOWLEDGEMENT

The Veterinary Council of Tanzania is pleased to accomplish this important milestone of formulating Standard Treatment Guidelines (STG) for Animal Diseases. This will enable veterinarians and paraprofessionals to effectively treat ruminants and poultry diseases aiming at contributing to the control of diseases and the misuse of medicines.

Standard Treatment Guidelines offer a number of potential advantages for animal health providers, inputs supply managers and animal health policy makers. The potential benefits of introducing the standard treatments guidelines in animal health include:

For Animals:

- Consistency among prescribers, reduced confusion and increased compliance.
- Most effective treatments prescribed.
- Improved supply of medicines, if they are prescribed only when needed.

For Providers:

- Give expert consensus on most effective, economical treatment for a specific setting.
- Provider can concentrate on correct diagnosis.
- Provide a standard to assess quality of care.
- Provide a simple basis for monitoring and supervision.

For Supply Management Staff:

- Performance standard for medicine supply, and therefore, need for sufficient quantities of medicines available for the most commonly treated problems at different levels of the animal health system.
- Facilitate pre-packaging of therapy quantities of commonly prescribed items for common conditions.
- Medicine demand more predictable and hence, forecasting more reliable.

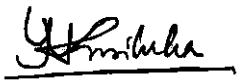
For Animal Health Policy Makers:

- Provide methods to effectively control animal diseases.
- Serve as a basis to assess and compare quality of animal health services.

- Development and implementation of a single set of standard treatments can be a vehicle for integrating special programmes at various veterinary facilities.

These treatments are initially developed for animals by the most eminent veterinarians. The first choice of treatment of an animal depends on the patients' diagnosis and condition. Veterinarians and other animal health care providers can use the same standard treatment. Updates that include a change or alteration in the therapeutic preferences will be made regularly to reflect upcoming recommendations.

On behalf of the Veterinary Council of Tanzania, I would like to thank all the people and offices that have provided technical advice and support leading to the accomplishment of this important task. Specifically, we are grateful to the Tanzania Veterinary Laboratory Agency; Directorate of Veterinary Services; College of Veterinary Medicine and Biomedical Sciences, Sokoine University of Agriculture; Tanzania Veterinary Association, and other stakeholders in the public and private sectors for supporting and facilitating the development of these standards. The production of this document was made possible by support from the Food and Agriculture Organization of the United Nations (FAO) through funding from the United States Agency for International Development (USAID), which is very highly acknowledged.



Prof. Lughano J.M. Kusiluka

Chairman of the Veterinary Council of Tanzania

ACRONYMS

ACRONYMS

AMR	Antimicrobial Resistance
BW	Body Weight
CAP	Chapter
CNS	Central Nervous System
CVMBS	College of Veterinary Medicine and Biomedical Sciences
DNA	Deoxyribonucleic Acid
DVS	Department of Veterinary Services
EDTA	Ethylene-diamine-tetra acetic acid
ELISA	Enzyme-linked Immunosorbent Assay
FAO	Food and Agriculture Organization of the United Nations
FMD	Foot and Mouth Disease
GI	Gastrointestinal
IM	Intramuscular
IMI	Intramammary Infection
IV	Intravenous
WOAH	World Organization for Animal Health
PCR	Polymerase Chain Reaction
PO	Per oral
RNA	Ribonucleic Acid
SC	Subcutaneous
STG	Standard Treatment Guidelines
SUA	Sokoine University of Agriculture
TCU	Tanzania Commission for Universities
TVA	Tanzania Veterinary Association
VCT	Veterinary Council of Tanzania

SECTION 1

STANDARD TREATMENT GUIDELINES FOR ANIMAL DISEASES IN TANZANIA

1.0 INTRODUCTION

1.1 Background

Standard Treatment Guidelines (STG) in animal health is defined as systematically developed statements designed to assist veterinarians, paraprofessionals and other animal health care workers in making decisions about appropriate animal health care for specific clinical circumstances. The STGs offer several potential advantages for animal health providers, supply managers and animal health policy makers.

In the interest of therapeutic and economic efficiency, STGs have been prepared to address conditions that contribute most in terms of morbidity and mortality patterns, targeted priority conditions based on local disease factors and use fewest medicines necessary, cost-effective treatments, and have considered farmers perspective.

1.2 General and specific objectives

These Standard Treatment Guidelines are primarily for Ruminants and Poultry Diseases but, they may be applicable to other animal species. The Guidelines list preferred treatments for common animal health problems in Tanzania. They are designed to be used as a tool to prioritize treatment choices and as a reference to help in the overall management of ruminants and poultry diseases. They will be used at all levels within the public and private animal health systems. The existence of these Guidelines will have a major impact on the consistency, effectiveness and economy of prescribing.

Standard treatments focus the thinking on critical aspects of the therapeutic process like:

- Careful identification of signs and symptoms.
- Correct diagnosis.
- Effective and proper use of drugs or non-drug treatments that will truly benefit the animal and the farmer.

1.3 Key features of the Guidelines

- Standard treatment exists for common illnesses of ruminants and poultry.

- Selected numbers of important animal diseases are listed with their key clinical diagnostic criteria.
- Medicine and dosage information are indicated clearly and concisely.
- Standard treatments are developed by eminent veterinarians and revisions will be based on the field experience and evolving scenarios.
- The first choice of treatment of a disease depends on the diagnosis and condition.
- Veterinarians and other animal health care providers can use similar standard treatment.
- These Guidelines are published as small, durable pocket size manuals, which make them convenient to carry and use.
- This document should be used in relation to other documents with standard case definitions of animal diseases.

SECTION 2

GUIDELINES FOR RUMINANT DISEASES

2.1 Preamble

These Standard Treatment Guidelines for large and small ruminants have been developed based on the comprehensive category so as to help in rationalizing veterinary practice. The Guidelines are aimed to protect the respective animal population from irrational therapy and hazardous consequences. Educating farmers and animal health professionals about the hazards of irrational curative care is another necessity and is being advocated. Rationalizing clinical animal health care will reduce costs for animal health system and makes the system more effective for the same level of expenditure.

The Guidelines are arranged based on the causative agent that include viruses, bacteria and parasites.

2.2 Ruminant Viral Diseases

2.2.1 Foot and Mouth Disease (FMD)

Definition and causative agent

It is an acute, contagious, febrile disease of cattle, sheep, goats, swine, and other cloven-hoofed ruminants characterized by vesicular eruption in the mouth, hooves, teats and udder. The FMD virus is highly contagious, and outbreaks of the disease can have severe economic consequences. FMD is caused by an *Aphthovirus* of the family *Picornaviridae* and there are seven different serotypes (A, O, C, SAT1, SAT2, SAT3 and Asia1). Each serotype requires a specific vaccine to provide immunity, which may not be effective against all the strains within each serotype. Six out of the seven existing FMDV serotypes have occurred in Africa (O, A, C, SAT 1, SAT 2, SAT 3) and more importantly, all six of the seven serotypes have been detected in East Africa. This makes East Africa one of the regions which has had the highest diversity of FMDV serotypes which consequently, complicate FMD epidemiology and control.

Transmission

An infection may occur via the respiratory or oral routes, or through contact with contaminated infrastructure and equipment. Animals recovered from the disease may still carry the virus and are considered as potential sources of infection for new outbreaks.

Clinical signs:

The clinical signs of FMD depend on many different factors such as the strain of the virus; age and species of the animal and the exposure dose.

Typically, the affected animal presents with ulcers and wounds in the mouth and feet, causing hyper-salivation and lameness. Other frequent clinical signs are fever, depression, loss of appetite and weight, growth retardation and heavy losses in milk production. In young animals, sudden death can be observed.

The FMD morbidity levels are very high, reaching 100% in naive susceptible populations. Mortality is generally low in adult animals but may be significantly high in calves.

Lesions:

Specifically, blisters can appear on the nose, lips, tongue, oral mucosa, between the toes, above the hooves, on the teats and at pressure points on the skin.

Diagnosis:

The disease may be suspected based on epidemiology and clinical signs.
Confirmation of any suspected FMD case through laboratory tests is, therefore, a matter of urgency. Laboratory diagnosis is usually performed using antigen-capture ELISA or serotyping ELISA. This is the preferred method for countries with endemic FMD for viral antigen detection and serotyping. Concurrent virus isolation may be performed, preferably in primary bovine thyroid cell cultures.

Differential diagnosis:

FMD should be differentiated from other vesicular diseases such as vesicular stomatitis and vesicular exanthema.

Treatment and control:

Treatment:

There is no treatment for FMD. However, the external application of antiseptics contributes to the healing of the ulcers and guards off attacks by flies. Rational treatment with dihydrostreptomycin 10 mg/kg body weight (IM) or Ooxtetracyline 10 mg/kg (IM) for 3-5 days to prevent secondary infection is recommended. For localized treatment, it is recommended to rinse the ulcerated vesicles using normal saline or acidic citric 1% or potassium permanganate 1%. After cleaning the teats and limbs then apply antiseptic, ointment and bandage.

Control:

Depending on the epidemiological situation, vaccination strategies can be designed to achieve mass coverage or be targeted to specific animal sub-populations or zones. Vaccination programmes carried out in a target population should meet several critical criteria, mainly the following:

- coverage should be at least 80%;
- campaigns should be completed in the shortest possible time;
- vaccination should be scheduled not to allow for interference from maternal immunity;

Vaccination must be done using a vaccine which has a specific serotype for the circulating virus. It is important to use inactivated virus vaccines because the inactivated virus does not have the ability to multiply in vaccinated animals. The use of live virus vaccines is not acceptable due to the danger of reversion to virulence. It is advisable to carry out two to three vaccinations at an interval of six months.

Biosecurity measures:

Outbreak of FMD must be reported to the competent veterinary authority, movement of animals be restricted and control measures employed to avoid rapid spread of the disease. Warning systems, early detection and effective surveillance are essential.

Best practice for livestock owners and producers in case of an FMD outbreak is to carry out regular cleaning and disinfection of equipment, buildings and vehicles, maintaining biosecurity practices and monitoring.

2.2.2 Lumpy Skin Disease (LSD)

Definition and causative agent:

Lumpy skin disease is an infectious, eruptive, occasionally fatal disease of cattle characterized by nodules on the skin and other parts of the body. It is a poxvirus disease of cattle caused by the Lumpy Skin Disease virus from the family Poxviridae, genus *Capripoxvirus.*

Transmission:

There is strong evidence that the virus is spread by biting insects (vectors) such as biting flies (e.g. *Stomoxys calcitrans* and *Biomyia fasciata*) and mosquitoes (e.g. *Culex mirificens* and *Aedes natrionus*). Direct transmission between animals is believed to occur but the relative importance of this method of spread compared to vector transmission is unknown. There is no evidence of virus multiplication in vectors, but it may not be excluded.

Clinical signs:

Morbidity level is 5-50%; mortality is usually low and ranges between 1% and 3%. Susceptibility of the host depends on the immune status, age, and breed.

The incubation period is usually 6-9 days until the onset of fever. In acute infection there is an initial pyrexia (may exceed 41°C) that is persistent for 1 week, enlargement of superficial lymph nodes, reduced milk production in lactating animals. Depression, anorexia, excessive salivation, ocular and nasal discharge, and emaciation are also observed.

Lesions:

Development of large, firm nodules that are up to 5 cm in diameter on the skin. These can be found all over the body, but particularly on the head, neck, udder, scrotum and perineum. The nodules may become necrotic and ulcerate, leading to an increased risk of fly strike.

In severely affected animals, necrotic lesions can also develop in the respiratory and gastro-intestinal (GI) tract.

Diagnosis:

Samples identification:

Polymerase chain reaction (PCR) is the least expensive and quickest method for detection of LSD virus. Skin nodules and scabs, saliva, nasal secretions, and blood are suitable samples for PCR detection of the LSD virus.

Virus isolation (VI) followed by PCR to confirm the virus identity takes longer and is more expensive but has the advantage of demonstrating the presence of live virus in the sample.

Virus neutralization is currently the gold standard test for the detection of antibodies raised against Capri pox viruses.

Differential diagnosis:

Severe LSD is highly characteristic, but milder forms can be confused with the following diseases: Bovine Herpes Mammillitis (sometimes known as pseudo-lumpy skin disease), bovine papular stomatitis (Parapoxvirus), Pseudo cowpox (Parapoxvirus), Dermatophilosis, Demodicosis and Photosensitization.

Treatment and control:

Treatment:

There is no treatment for the disease.
Non-specific treatment of antibiotics especially oxytetracyline 10 mg/kg. (IM) or dihydrostreptomycin 10 mg/kg for 5 days, anti-inflammatory drugs (e.g. dexamethasone at a dose of 0.2 mg/kg IM for 3 days) and vitamins injections are used for treating secondary bacterial infections, and suppressing inflammation and fever.

Control:

Attenuated vaccines against LSD and some Neethling virus based LSD vaccine are available. These vaccines are >80% effective. Reconstitute each vial with a corresponding sterile diluent. Keep reconstituted vaccine cool and away from sunlight. Administer the vaccine SC at a dose of 2 ml per animal of all ages. Annual revaccination is recommended.

Successful control and elimination of LSD depend on early detection of the index case and initiation of vaccination ahead of virus entry. Annual vaccination is recommended.

Biosecurity measures:

Employ restriction of movement of cattle in infected areas, proper disposal of dead animals by incineration, and cleaning and disinfection of premises and implements.

2.2.3 Peste des Petits Ruminants (PPR)

Definition and causative agent:

Peste des petits ruminants (PPR), also known as 'goat plague', is a viral disease of goats and sheep characterized by fever, sores in the mouth, diarrhea, pneumonia, and sometimes death. It is caused by a virus of the genus *Morbillivirus* in the family Paramyxoviridae. PPRV is closely related to rinderpest virus. Heavy losses can be observed, especially in naïve herds/ flocks (up to 80% mortality may occur).

Transmission:

The virus is secreted in tears, nasal discharge, secretions from coughing, and in the faeces of infected animals. Therefore, close contact between animals, especially through inhalation of fine droplets that are released into the air when affected animals cough and sneeze will spread the disease. Water, feed troughs, and bedding can also be contaminated with secretions and become additional sources of infection; however, the virus does not survive for a long time outside the body of a host animal. Since animals excrete the virus before showing signs of the disease, it can spread by movement of infected animals.

Clinical signs:

In acute form, there is sudden onset of fever with rectal temperature of at least 40°- 41°C. The affected goats/sheep show dullness, sneezing, and serous discharge from the eyes. During this stage, profound halitosis (foul smell) is easily appreciable and the animal is unable to eat due to sore mouth and swollen lips.

Later ocular discharge becomes mucopurulent and the exudate dries up, matting the eyelids and partially occluding the nostrils. Diarrhea develops 3-4 days after the onset of fever and is profuse, and faeces may be mucoid or bloody depending upon the damage of the gastrointestinal tract.

Dyspnea and coughing occur later due to secondary pneumonia. Death occurs within one week of the onset of the illness.

Lesions:

Discrete lesions develop in the mouth and extend over the entire oral mucosa, forming diphtheritic plaques.

Diagnosis:

The disease may be suspected when there is sudden onset of fever, nasal discharges, diarrhoea in sheep and goats, while cattle are uninvolved. Tests such as immunocapture/sandwich-ELISA, competitive-ELISA, immunochromatographic techniques, and Reverse Transcription (RT)-PCR can be efficiently applied on very large sample sizes for routine diagnosis of PPR. However, during the later stages of PPR eradication, critical screening of suspected samples from PPR-like diseases as well as samples from wild animals might require the use of highly sensitive, multiple diagnostic tools. Under these situations, sensitive and reliable diagnostics such as RT-PCR-ELISA could also be applied, in spite of the expenses and time involved.

Differential diagnosis:

PPR is frequently confused with other diseases that present fever and grossly similar clinical signs, especially when it is newly introduced. Differential diagnosis should consider, FMD, Bluetongue, Contagious ecthyma (orf or "sore mouth"). Others include Pneumonic pasteurellosis and Contagious Caprine Pleuropneumonia (CCPP).

Treatment and control:

Treatment:

Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections at a dose of 10 mg/kg B.W.

Lesions around the eyes, nostrils and mouth should be cleaned twice daily with sterile cotton swab. Animal health workers should inspect first the unaffected goats followed by treatment of affected sheep and goats. Immediate isolation of affected animals from clinically healthy ones is most important measure in controlling the spread of infection. Nutritious soft, moist, palatable diet should be given to the affected animals.

Control:

Control in endemic areas is by vaccination. Current PPR virus (PPRV) attenuated vaccines are thermo labile and to avoid their thermal inactivation, they require uninterrupted maintenance of the cold chain until their application to the animal.

The currently commercially available vaccines are in freeze-dried form, and they are stable for at least two years at 2°C to 8°C and for several years at –20°C. Once the vaccine is reconstituted it should be kept under ice and it needs to be utilized as soon as possible, at a dose of rate 1 ml (SC) for goats and sheep. Use vaccine as early as 4 months of age or older. Animals that recover from disease develop good immunity, which persists for at least four years.

Biosecurity measures:

Immediate measures should be taken for notification of disease to nearest government veterinary authority. Carcasses of affected sheep and goats should be burned or buried, and proper disposal of contact fomites, decontamination is a must.

2.2.4 Rift Valley Fever (RVF)

Definition and causative agent:

It is a zoonotic insect-borne viral disease that affects a variety of species, including ruminants and camels. The disease is caused by the Rift Valley Fever Virus (RVFV), an RNA virus in the genus *Phlebovirus* and family Bunyaviridae. Outbreaks of RVF often cause substantial socio-economic and public health impacts. RVF strikes in periodic epidemics, which typically occur after heavy rainfalls.

Transmission:

Rift Valley fever virus is transmitted by 8 species of mosquitoes from the *Aedes* and *Culex* family. which act as biological vectors. The virus can also be transmitted vertically through the placenta. Humans can acquire RVFV by direct contact with infected animal tissues/products, by aerosol containing viruses generated in laboratories and during slaughter, or from infected mosquitoes. The incubation period is thought to be one to three days.

Clinical signs:

In endemic regions, epidemics of RVF are characterized by high mortality in young animals and abortions in adults. Foetuses may be aborted at all stages of pregnancy. Nonspecific signs of fever, anorexia, weakness, lymphadenopathy, respiratory signs and hemorrhagic diarrhoea can be seen in young animals.

Lesion:

The signs associated with viraemia, such as widespread petechial and ecchymotic haemorrhages on serous surfaces and organs are seen, with extravasation of blood tinged fluids in the body cavities.

Diagnosis:

Whole blood (in EDTA anticoagulant) is collected from febrile animals for virus isolation. Tissue specimens such as liver and spleen from animals at post-mortem and aborted foetuses, both fresh and preserved (in 10% neutral buffered formalin and in glycerol) samples should be collected. Identification of RVFV can be achieved by virus isolation, antigen-ELISA or immunopathology. Viral RNA can be detected by reverse-transcription polymerase chain reaction.

Differential diagnosis:

Differentiation has to be made with Nairobi Sheep Disease, Bluetongue, other viral and bacterial causes of abortion.

Treatment and control:

Treatment:
There is no specific treatment for RVF.

Control:

Conventional and novel vaccines are available. Attenuated virus vaccine (Smithburn strain) is used. The diluent is used to re-hydrate the freeze-dried component during reconstitution. Vaccines are given SC, at a dose rate of 2ml for cattle and 1ml for sheep and goats injected, but they may cause abortions in pregnant animals. Annual revaccination is recommended. Control of mosquitos by application of pyrethroids acaricides on ruminants is recommended.

Biosecurity measures:

Immediate notification of clinical cases upon detection and reporting and implementation of sanitary measures to prevent spread are recommended.

2.2.5 Sheep and Goat Pox

Definition and causative agent:

Sheep pox and goat pox (SGP) are highly contagious diseases of sheep and goats characterized by fever, ocular and nasal discharges. Pox lesions appear on the skin and on the respiratory and GI mucosa. Mortality can be high.

The diseases are caused by *capripox* viruses. This group also includes the Lumpy skin disease of cattle.

Transmission:

Sheep Pox Virus (SPV) and Goat Pox Virus (GPV) are transmitted by close contact through mucous membranes and abraded skin. Viruses are shed in milk, urine, faeces, skin lesions and their scabs, saliva, nasal and conjunctival secretions. SPV and GPV can also be spread on fomites or transmitted mechanically by insects such as stable flies. These viruses can remain infectious for up to six months in shaded stables. The incubation period is four to 21 days.

Clinical signs:

In affected animals, an initial fever is usually followed by the characteristic skin lesions, which develop into hard papules with scabs. The lesions can develop on mucous membranes and internal organs, causing respiratory signs, diarrhea and sometimes abortion.

Lesions:

In severe cases, lesions can develop in the lungs. In some sheep and in certain breeds, the disease may be mild or the infection in apparent. Papules could be seen at necropsy especially in the lungs, the upper respiratory tract, rumen, the upper digestive tract, kidney, and reproductive organs.

Diagnosis:

Sheep and Goat Pox should be suspected when an acute disease of sheep or goats is accompanied by fever, pox-like skin lesions and high mortality rate.

For serological examination, paired blood samples from animals with fever should be collected. For virus identification, samples can be serum, vesicular fluid, scabs and skin scrapings of lesions and lesions in the respiratory and GI tract. Samples should be taken within the first week of apparition of the symptom and be kept fresh.

World Organization for Animal Health (WOAH) recommends identification methods which could be cell inoculation and identification by immunofluorescence, staining of intracytoplasmic inclusion bodies and inhibition of cytopathic effect using positive serum or ELISA.

Differential diagnosis:

Differentiation should be made with contagious pustular dermatitis (scabby mouth), Bluetongue, Mycotic dermatitis, sheep scab and mange

Treatment and control:

Treatment:

Treatment is directed at preventing secondary bacterial infection. Administration of Oxytetracycline at a dose of 10mg/kg b.w. to control secondary infection and good nursing care are recommended.

Control:

Vaccines are available to control sheep and goat pox in endemic areas. Live attenuated freeze-dried Sheep and Goat Pox Vaccine is available. Vaccination is done, after reconstitution with a corresponding sterile diluent, at a dose of 1 ml per animal (SC). Goats and Sheep should be vaccinated at the age of over 5 weeks. Annual vaccination is recommended. In the face of an outbreak, animals as young as 3 weeks may be vaccinated.

Biosecurity measures:

Movement controls for both animals and vehicles should be applied.

2.2.6 Bovine Ephemeral Fever

Definition and causative agent:

Is a disease of cattle and occasionally buffaloes, marked by short duration of fever, shivering, lameness and muscular stiffness, commonly known as three-day sickness, The virus is classified as a member of the genus Ephemerovirus in the family Rhabdoviridae (single-stranded RNA).

Transmission:

It is an insect-transmitted, noncontagious, viral disease of cattle and water buffaloes that is observed in Africa, the Middle East, Australia, and Asia. During epidemics, onset is rapid; many animals are affected within days or 2–3 weeks.

Clinical signs:

In the tropics, Bovine Ephemeral Fever (BEF) is most prevalent in the wet season (when conditions favor multiplication of biting insects). Morbidity may be as high as 80%; overall mortality is usually 1%–2%, although it can be higher in lactating cows, bulls in good condition, and fat steers (10%–30%). Clinical signs occur suddenly and vary in severity, include biphasic to polyphasic fever (40°–42°C, shivering, in appetence, lacrimation, serous nasal discharge, drooling, increased heart rate, tachypnea or dyspnea, atony of fore stomachs, depression, limb stiffness and lameness, and a sudden decrease in milk yield.

Affected cattle may become recumbent and paralyzed for 8 hours to >1 week. After recovery, milk production often fails to return to normal levels until the next lactation. Abortion, with total loss of the season's lactation, occurs in about 5% of cows pregnant for 8–9 months.

Lesions:

The most common lesions include polyserositis affecting pleural, pericardial, and peritoneal surfaces; serofibrinous polysynovitis, polyarthritis, polytendinitis, and cellulitis; and focal necrosis of skeletal muscles. Generalized edema of lymph nodes and lungs, as well as atelectasis, also may be present.

Diagnosis:

The diagnosis during epidemics is made through clinical signs including lameness, muscular stiffness, pain, rapid spread of the disease through herds and short fever.

Diagnosis is based almost entirely on clinical signs in an epidemic. Whole blood should be collected from sick and apparently healthy cattle in affected herds and must be sufficient to provide two air-dried blood smears, 5 ml of whole blood in anticoagulant (not EDTA), and ~10 ml of serum.

Timely laboratory confirmation is mostly performed by PCR and rarely by virus isolation. Serum neutralization is diagnostic in retrospect. A 4-fold rise in antibody titer between paired sera collected 2–3 weeks apart confirms infection.

Differential diagnosis:

BEF can be suspected on the basis of its transient nature and rapid spread. In individuals it may resemble conditions such as Traumatic reticulitis, acute laminitis and Parturient paresis.

Treatment and control:

Treatment:

Complete rest is the most effective treatment, and recovering animals should not be stressed because relapse is likely.

Animal generally recover quickly without treatment. Most animals will recover if provided with water and shade. However, the disease respond well to anti-inflammatory drugs (Dexamethasone 0.2mg/kg b.w. for three days) and calcium injections may aid animals that are down.

Control:

A modified live vaccine of two-part vaccine, with freeze-dried and chilled liquid diluent. The initial vaccine is administered twice 2 to 4 weeks apart under the skin of the neck are necessary for long-lasting protection. Annual boosters are recommended.

2.2.7 Infectious Bovine Rhinotracheitis (IBR)

Definition and causative agent:

Is a highly contagious, infectious respiratory disease that is caused by Bovine Herpes Virus-1 (BHV-1) affecting young and older cattle.

Transmission:

It is highly contagious, resulting in rapid spread among cattle in close confinement through aerosol route.

Clinical signs:

The disease is characterized by acute inflammation of the upper respiratory tract. The virus can also cause conjunctivitis, abortions, encephalitis, and generalized systemic infections.

Lesions:

In uncomplicated IBR infections, most lesions are restricted to the upper respiratory tract and trachea. Petechial to ecchymotic hemorrhages may be found in the mucous membranes of the nasal cavity and the paranasal sinuses. Focal areas of necrosis develop in the nose, pharynx, larynx, and trachea. The lesions may coalesce to form plaques.

The sinuses are often filled with a serous or serofibrinous exudate. As the disease progresses, the pharynx becomes covered with a serofibrinous exudate, and blood-tinged fluid may be found in the trachea. The pharyngeal and pulmonary lymph nodes may be acutely swollen and hemorrhagic.

The tracheitis may extend into the bronchi and bronchioles; when this occurs, epithelium is sloughed in the airways. In young animals with generalized BHV-1 infection, erosions and ulcers overlaid with debris may be found in the nose, esophagus, and fore stomachs.

Diagnosis:

Uncomplicated BHV-1 infections can be diagnosed based on the characteristic signs and lesions. Samples should be taken early in the course of the disease, and a diagnosis should be possible in 2–3 days. A rise in serum antibody titer also can be used to confirm a diagnosis.

BHV-1 abortion can be diagnosed by identifying characteristic lesions and demonstrating the virus in fetal tissues by PCR, virus isolation, immunoperoxidase, or fluorescent antibody staining.

Differential diagnosis:

For the respiratory form: Enzootic bronchopneumonia, Bovine Virus Diarrhoea/ Mucosal Disease and Theileriosis should be considered. For the abortion form: Bovine Virus Diarrhoea/Mucosal Disease, Brucellosis and Leptospirosis should be differentiated.

Treatment and control:

Treatment:

Antimicrobial therapy (use of broad-spectrum long-acting antibiotics such as oxytetracyline 10mg/kg b.w. is indicated to prevent or treat secondary bacterial pneumonia.

Additionally the use of non-steroid anti-inflammatory drug (such as Meloxicam, 20mg/ml) at a dose of 2.5ml/100kg SC or IV single dose may help relieve respiratory symptoms and pyrexia.

Control:

mmunization with modified-live or inactivated virus vaccines generally provides adequate protection against clinical disease. Both IM and intranasal modified-live vaccines are available, but the IM types may cause abortion in pregnant cattle.

The intranasal vaccines can be used in pregnant cattle. The IM vaccines are easier to use and often are the vaccines of choice in feedlots. Vaccination is done after reconstitution of a freeze-dried vaccine that is injected SC or IM at a dose of 2ml per animal that are in good health, and non-pregnant. To avoid maternal antibody interference, calves vaccinated before the age of 6 months should be revaccinated after 6 months of age. Annual revaccination is recommended.

Biosecurity measures:

Importation of infected animals and semen should be avoided

2.2.8 Bovine Viral Diarrhoea (BVD) and Mucosal Disease Complex

Definition and causative agent:

Bovine Viral Diarrhea (BVD) and Mucosal Disease Complex is caused by Bovine Viral Diarrhea Virus (BVDV), classified in the genus *Pestivirus* and family Flaviviridae. Although cattle of all ages are susceptible, most cases of overt clinical disease are seen in cattle between 6 months and 2 years old.

Transmission:

BVDV is transmitted in a number of ways, either congenitally or after birth. Congenital infections may cause resorption, abortion or stillbirth. Congenitally infected fetuses that survive in-utero infection (i.e. the live births) may be born as BVDV-infected calves. The BVDV infection in these calves will persist during the entire life of the calf, and they will shed BVDV continuously in the farm environment. The virus may also be spread by biting insects, fomites, semen, biologic products, and other animals, including swine, sheep, goats, camelids, and possibly wild ruminants.

Clinical signs:

Clinical signs of disease usually are seen 6–12 days after infection and last 1–3 days. In adults, clinical signs are highly variable. Signs of acute infection include fever, lethargy, loss of appetite, ocular and nasal discharges, oral lesions, diarrhea and decreasing milk production. Chronic infection may lead to signs of mucosal disease.

In calves, the most commonly recognized birth defect is cerebellar hypoplasia. The signs of this are ataxia/lack of voluntary coordination of muscle movements, tremors, wide stance; stumbling and failure to nurse.

In severe cases the calf may die.

Transient infections include diarrhea, calf pneumonia, decreased milk production, reproductive disorders, increased occurrence of other diseases, and death.

The losses from fetal infection include abortions, congenital defects, weak and abnormally small calves, unthrifty, persistently infected (PI) animals; and death among PI animals.

Mucosal disease is an uncommon but highly fatal form of BVD occurring in persistently infected cattle and can have an acute or chronic presentation. Mucosal disease is induced when persistently infected cattle become superinfected with cytopathic BVDV. Clinical signs of chronic mucosal disease may last several weeks to months and are less severe than those of acute mucosal disease. Intermittent diarrhea and gradual wasting are common. Coronitis and eruptive lesions on the skin of the interdigital cleft cause lameness in some cattle.

Lesions:

Lesions are seldom 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.

Lesions found at necropsy are less pronounced than, but similar to, those seen in acute mucosal disease. Often, the only gross lesions seen are focal ulcerations in the mucosa of the cecum, proximal colon, or rectum, and the mucosa over Peyer's patches of the small intestine may appear sunken.

Diagnosis:

Laboratory tests for BVDV include isolation of virus or viral antigen in clinical specimens and tissues, and assays that detect anti-BVDV antibody in serum or milk. Isolation of BVDV from blood, nasal swab specimens, or tissues confirms active infection.

Alternatives to viral isolation include antigen-capture ELISA to detect virus in blood, serum, or tissue biopsies; immunohistochemistry to detect viral protein in frozen or fixed tissues; and PCR to detect viral RNA in clinical specimens.

Differential diagnosis:

BVD must be distinguished from other viral diseases that produce diarrhea and mucosal lesions. These include Malignant Catarrhal Fever, which usually is a sporadic disease in more mature cattle; Bluetongue and Rinderpest, which is currently considered to be eradicated worldwide.

Treatment and control:

Treatment of BVD remains limited primarily to supportive therapy.

Control:

Inactivated and modified-live virus vaccines are available. They contain a variety of strains of BVDV representing both viral biotypes and viral genotypes 1 and 2. A booster dose of vaccine is often administered before first breeding, and additional booster doses of vaccine may be administered in subsequent years before breeding.

Rehydrate the vaccine for reconstitution and administer 2ml of the vaccine (IM) per animal. Annual booster for breeding stock is recommended. Do not vaccinate pregnant cows to avoid abortion.

Colostral antibody confers partial to complete protection against disease in most calves for 3–6 months after birth. Vaccination of neonatal cattle that have acquired colostral antibody may not stimulate a protective immune response, and revaccination at 5–9 months of age may be necessary.

Biosecurity measures:

Control is based on sound management practices that include use of biosecurity measures, Quarantine or physical separation of replacement cattle from the resident herd for 2–4 week should be considered.

2.2.9 Blue tongue

Definition and causative agent:

It is a noncontagious, insect-borne viral disease of ruminants mainly sheep, and less frequently cattle, goats, yaks, deer, dromedaries and antelopes. The causative agent is a Blue Tongue Virus (BTV) of the family Reoviridae and genus *Orbivirus*.

Transmission:

The disease is transmitted by midges of Cullicoides species, which infects domestic and wild ruminants and also camelids. Midges are the only significant natural transmitters and thus distribution and prevalence of the disease is governed by ecological factors (i.e. high rainfall, temperature, humidity and soil characteristics). Hence, in many parts of the world infection has a seasonal occurrence.

The likelihood of mechanical transmission between herds and flocks, or indeed within a herd or flock, by unhygienic practices (use of contaminated surgical equipment or hypodermic needles) may be a possibility.

Clinical signs:

Signs range from in apparent to acute form (sheep and some species of deer), resulting in excessive salivation, initially clear nasal discharge that becomes mucopurulent and upon drying, may form a crust around the nares. Incubation period is usually 5–10 days. Sub-clinically infected cattle can become viraemic 4 days post-infection. Morbidity in sheep can reach 100% with mortality between 30 and 70% in more susceptible breeds. Ulceration and necrosis of the mucosae of the mouth may appear. Tongue may become hyperaemic and oedematous, later cyanotic and protrude from the mouth.

Extension of hyperaemia to coronary band of the hoof, the groin, axilla and perineum may be observed, as well as lameness due to coronitis or pododermatitis and myositis. Torticolis in severe cases, abortion or birth of malformed lambs, death may occur within 8–10 days or recovery with alopecia, sterility and stunted growth may be observed. In-apparent infection is frequent in cattle and other species.

Note: a blue colored tongue is rarely a clinical sign of infection.

Lesions:

Sheep: hemorrhages into or under skin, inflammation of the coronary band, respiratory problems, fever and lethargy.

Diagnosis:

Samples for laboratory diagnosis include, in live animals: blood samples collected in heparin. In freshly dead animals: spleen, liver, red bone marrow, heart blood, lymph nodes can be sampled. Aborted and congenitally infected newborn animals: pre-colostrum serum plus same samples as for freshly dead animals. All samples have to be preserved at 4°C, and not frozen. A prescribed test for international trade, Real-time RT-PCT is available. Agar gel immunodiffusion (an alternative test for international trade) is also available. ELISA-based procedures for the specific detection of anti-BTV antibodies have been developed. Indirect ELISA has been shown to be reliable and useful for surveillance purposes for bulk milk samples.

Differential diagnosis:

Differential diagnosis include FMD , Vesicular stomatitis and PPR.

Treatment and control:

No efficient treatment is available.

Control:

Both live attenuated and killed BTV vaccines are currently available. Attenuated vaccines are serotype specific. Vaccine serotype must be same as those causing infection. Attenuated vaccines can be transmitted to unvaccinated animals and could reassert with field strains; resulting in new viral strains. A freeze-dried vaccine is reconstituted using a sterile diluent supplied with the vaccine. The reconstituted vaccine should be kept cool and away from sunlight and it is administered SC, at a dose of 1 ml to sheep over 3 months of age, . Revaccination should be carried out annually.

Biosecurity measures:

If bluetongue is suspected, it must be reported within 24 hours to the veterinary authority. Animal movement control, quarantines and vector control should be applied.

2.2.10 Calf scour

Definition and causative agent:

It is a broad descriptive term referring to diarrhea in calves under one month of age with the majority occurring between 3 and 16 days of life. Calf scour is not a specific disease with a specific cause, but it is actually a clinical sign of a disease complex with many causes such as bacterial or viral infections. The primary causes of scours include: Rotavirus, Coronavirus, *Cryptosporidium parvum*, Salmonella spp., and *Escherichia coli*. Rotavirus, Coronavirus or Cryptosporidium cause 95% of calf scours in calves under 3 weeks of age.

Transmission:

Calf scours is common in dairy calves and almost all calves are exposed to these pathogens and the deciding factor whether or not a calf gets sick is often dose-dependent

Clinical signs:

Watery stools, that may be brown, grey, green, yellow in colour.

Calves are often weak, depressed and may lose their desire to nurse, and may develop a sunken eye appearance as a result of dehydration. Death may occur 12-48 hours after onset of the clinical signs.

Diagnosis:

Determine if the calf is looking listless. Calves that are lethargic or not participating much in the playful activities with other calves are a red flag to pay attention to. Calves that are losing condition are also cause for alarm.

Bacteriology and fecal smear examination to determine the cause has to be done. The virus isolation test is still considered the 'gold standard' for detecting viral pathogens in specimens, although new methods such as an ELISA and PCR-based tests have been developed. PCR testing is especially useful for detecting viruses that are difficult to isolate in cell culture or bacteria that require a long time to grow.

Fecal bacteria culturing is a commonly used laboratory method for isolating and identifying bacterial pathogens in feces and intestinal contents. *Salmonella* spp., *E. coli* K99^{+}, and *C. perfringens* are primary bovine enteric pathogens.

Treatment and Control:

Treatment:
Determine if treatment is required. Calves that are moving around in the pasture, with their tails up, and if the faeces is normal in appearance in this case, treatment is probably not needed. Administer fluids using your veterinarian-approved electrolyte solution**.** You may need to inject the fluids via (IV) or orally.

Control:
Control is through feeding calves adequate colostrum during the first two hours of life. Ensure calving pens are clean. Clean individual calf pen and feeding equipment using chlorine dioxide.
Cows should be fed adequately before calving.

Biosecurity measures:

Employ measures such as separating the sick calf or calves from the healthy to avoid further spreading of the disease. Do not use same fields for calving/ lambing and change fields every year or when clinical cases occur in that season.

2.2.10 Rabies

Definition and causative agent:

Rabies is a viral disease that affects the central nervous system (CNS) of mammals, including humans caused by a *Lyssavirus*.

Transmission:

Rabies virus is transmitted when infected saliva of an infected animal, most often a dog, is passed to an uninfected animal, mostly through a bite.

Clinical signs

The incubation period varies from several days to several months. The time taken depends on the distance of the bite from the brain. If the bite is on the face or head, the bitten animal or human will quickly show signs, but if the bite is on the leg it will take much longer for signs to develop.

Other susceptible animals include, foxes, wolves, hyenas and bats which feed on blood in some places. All rabid animals show similar signs in the beginning.

- They change their normal behaviour and behave very strangely.
- They stop eating or drinking.
- Male animal will try to mate (mount) other animals.
- There is no change in the body temperature.
- These signs will continue for 3 to 5 days. Then, before it dies, the animal will develop one or the other of two types of the disease:
    a. The furious (mad) type of the disease which makes the animal aggressive and it will bite anything.
    b. The quiet (dumb) type when the animal is quiet and does not move.

Rabies in sheep, goats and cattle

Rabies is characterized by the animals becoming restless and excited. They may bite themselves and saliva drips from the mouth. The most important sign in cattle is that the animal bellows (calls) very frequently and with strange sound. Eventually, the animals will become paralyzed and die.

Diagnosis:

Reference method for diagnosing Rabies is by Fluorescent Antibody Test (FAT).
The diagnosis can also be made from saliva, urine, and cerebrospinal fluid samples, but this is not as sensitive or reliable as brain samples. Cerebral inclusion bodies called Negri bodies are 100% diagnostic for rabies infection but are found in only about 80% of cases.

Differential diagnosis:

Any suspected mammalian encephalitis and neurological disorder must be considered in the differential diagnosis.

Control:

Avoid handling wild animals or strays.
Contact a veterinarian upon observing a wild animal or a stray, especially if the animal is acting strangely.

Vaccinate dogs against Rabies. Puppies at the age of 12 weeks (3 months) to be a given a single dose 1 ml of a killed vaccine (SC or IM). Adult dogs with an unknown history of vaccination be given a single dose 1ml of a killed vaccine (SC or IM), a booster given a year later and thereafter, vaccination performed every three years by the vaccine approved for three year administration. Cattle and sheep at the age of above 12 months are vaccinated at a dose of 2ml IM. Annual revaccination is recommended

2.2.11 Malignant Catarrhal Fever (MCF)

Definition and causative agent:

It is an infectious systemic disease of domestic cattle, and water buffaloes and rarely swine. With occasional exceptions, the disease in cattle normally is seen sporadically and affects single animals. It is caused by one of several members of a group of closely related ruminant gamma herpesviruses of the genus *Rhadinovirus*

The principal carriers and their viruses are sheep (ovine herpesvirus-2), wildebeest (alcelaphine herpesvirus-1), and goats (caprine herpesvirus-2). Virtually all clinical cases are caused by the sheep or wildebeest viruses.

Transmission:

Inhalation is thought to be the primary means of transmission for all MCF viruses, although susceptible animals may be exposed to parturient wildebeest or young calves through ingestion of contaminated pasture.

MCF is transmitted only between carriers and clinically susceptible animals and the latter do not transmit MCF to their cohorts.

Clinical signs:

Acute MCF cases caused by ovine herpesvirus-2 and alcelaphine herpesvirus-1 are similar clinically and pathologically. Disease course may

range from per-acute to chronic. High fever [41°–41.5°C]) and depression are common. Other possible signs include catarrhal inflammation; erosions and mucopurulent exudation affecting the upper respiratory, ocular, and oral mucosa; swollen lymph nodes; lameness; and CNS signs (depression, trembling, hypo responsiveness, stupor, aggressiveness, convulsions). In cattle, swollen lymph nodes and severe eye lesions (panophthalmitis, hypopyon, and corneal opacity) are more frequent and hemorrhagic enteritis and cystitis. Peripheral (centripetal) corneal opacity is an important clinical sign suggestive of MCF in cattle.

Skin lesions (erythema, exudation, cracking, crust formation) are common in animals that do not succumb quickly. As many as 25% of cattle experience chronic disease, and sometimes the disease dies out. Mortality rates in clinically affected animals generally approach 95%. However, in limited circumstances, survival in cattle can be higher, although survivors can rarely return to normal production.

Lesions:

The disease is systemic, and lesions may be found in any organ, although severity and frequency varies greatly. The principal lesions are inflammation and necrosis of respiratory, alimentary, or urinary mucosal epithelium; sub epithelial lymphoid infiltration; generalized lymphoid proliferation and necrosis; and widespread vasculitis. Mucosal ulcerations and hemorrhage are common. Hemorrhages may be present in many parenchymatous organs, particularly lymph nodes. A classic but not pathognomonic histologic lesion is fibrinoid necrosis of small muscular arteries. MCF is acute and highly lethal, capable of affecting large numbers of animals. It also occasionally presents as chronic alopecia and weight loss.

Diagnosis:

Diagnosis of MCF is based on clinical signs, gross and histologic lesions, and laboratory confirmation.

The test of choice for clinical diagnosis is PCR to detect viral DNA. Preferred tissues for testing are anticoagulated blood, kidney, intestinal wall, lymph node, and brain.

Serology is used to survey healthy animals and is indicative only of infection. Latent infection among susceptible animals may render serology alone an inconclusive evidence of current disease. Several sero-assays are available, including viral neutralization, immune peroxidase, immunofluorescence, and ELISA.

Differential diagnosis:

Include Bovine Viral Diarrhea/Mucosal Disease, Infectious Bovine Rhinotracheitis, and East Coast Fever.

Treatment and control:

Treatment:
The prognosis is grave. No treatment has been found to provide any consistent benefit. Stress reduction of subclinical or mildly affected animals is indicated.

Control:
No vaccine is currently available. The only other effective control strategy is separation of carriers from susceptible species.

Biosecurity Measures:

Prevent contact between carriers and clinically susceptible species, Separate susceptible animals from sheep, goats, wildebeest or other suspected reservoir hosts. Cattle should not graze pastures where wildebeest have grazed and given birth.

2.3 Ruminant Bacterial Diseases

2.3.1 Contagious bovine pleuropneumonia (CBPP)

Definition and causative agent:

CBPP is a highly contagious disease of cattle and water buffaloes caused by *Mycoplasma mycoides* subsp. *Mycoides* (*M. mycoides*). The disease affects lungs and the membranes that line the thoracic cavity (the pleura).

Transmission:

Transmission occurs through direct contact between an infected and a susceptible animal by inhaling droplets disseminated through coughing. Since some animals can carry the disease without showing signs of illness, controlling the spread is more difficult. There is no evidence of transmission through fomites (inanimate objects such as clothing, implements or vehicles) as the organism does not persist in the environment.

Clinical signs:

CBPP is manifested by loss of appetite, fever and respiratory signs, such as rapid respiratory rate, cough, nasal discharges and painful, difficult breathing.

In hot climates, an affected animal often stands by itself in the shade, its head lowered and extended, its back slightly arched, and its limbs turned out. In many cases, the disease progresses rapidly, animals lose condition, and breathing becomes very laboured, with a grunt at expiration.

The animals become recumbent (lie down) and in severe cases die within 1-3 weeks. The mortality rate may be as high as 50% in the absence of antibiotic treatment. However, clinical signs are not always evident in sub-acute or asymptomatic forms and affected animals partially recover after a period of 3 to 4 weeks. These cattle may be capable of spreading the disease, acting as in apparent carriers.

Lesions:

Yellow fluid in the chest cavity, lungs covered with yellowish material adhering to the chest wall and those that do not collapse and are solid or marbled.

Sequestra are seen in lungs of chronic case.

Diagnosis:

The presence of the disease can be detected in two ways: detection of the causal organism in affected tissues and detection of serum antibodies to the organism. The causal organism, *M. mycoides*, can be demonstrated in the fluid present in the chest and in diseased lung by culture, by antigen detection tests (interface precipitin test or agar gel immunodiffusion test) and by PCR.

The rapid slide agglutination test using whole blood or serum for antibody detection can be a useful test to detect infected herds. It can be used in the field to give rapid results. It is performed by mixing a drop of a suspension of killed and stained *M. mycoides* organisms with a drop of serum or blood on a glass slide. In a positive result aggregates form within one minute.

At present, the laboratory test of choice for detecting serum antibodies is the complement fixation test (CFT) or C-ELISA. Great care is needed in collecting and storing sera to be used for this test which is complex to perform. Histopathology of affected lung fixed in formalin can also help in confirming the diagnosis.

Samples for laboratory confirmation

Infected tissue is used to demonstrate the presence of the *Mycoplasma* organisms. Usually the samples required will be chest fluid and diseased lung kept on ice during transport to the laboratory. Additional samples may be fixed in 10% formalin solution for histopathology.

Serum, used for antibody tests, is obtained by allowing blood to clot at room temperature and then collecting the clear serum which is produced when the clot contracts. Separated sera should be kept on ice, not frozen, and transported quickly to a laboratory.

Differential diagnosis:

In the acute form, pleuropneumonia and bronchopneumonia from mixed infections, East Coast Fever and Traumatic pericarditis must be considered. In the chronic form hydatid cyst, Actinobacillosis, Tuberculosis, and Bovine farcy.

Treatment and control:

Treatment:

Treatment of affected animals with a macrolide antibacterial Tylosin at a dose of 10 mg/kg b.w. can result in healthy looking animals that are still infected and able to spread the disease, so it is not recommended.

Control:

Surveillance of the disease through slaughterhouse inspection is a very efficient method of detecting clinical cases.

Vaccination campaigns are useful in controlling the disease. Reconstitute the vaccine with corresponding diluent and keep the reconstituted vaccine cool and away from sunlight. Administer via SC 0.5ml to cattle of at least 6 months of age and above. Annual vaccination with an attenuated strain of the bacteria is used to reduce the level of infection. Attenuated virulent strains stimulate the best immunity, but also induce the most severe and undesirable local and systemic reactions. Two strains are used for preparing CBPP vaccines: strain T1/44, a naturally mild strain isolated in 1951 by Sheriff & Piercy in Tanzania, and strain T1SR; which is completely a virulent but has shorter immunity than T1/44, which may induce an unpredictable number of animals with post-vaccinal reactions requiring treatment with antibiotics 2 to 3 weeks after vaccination.

Biosecurity measures:

Outbreaks can be eliminated through quarantines, movement controls, slaughter of infected and exposed animals, and cleaning and disinfection of the premises.

2.3.2 Contagious Caprine pleuropneumonia (CCPP)

Definition and causative agent:

It is highly contagious disease of goats caused by a bacterium *Mycoplasma capricolum* subspecies *capripneumoniae*.

Transmission:

This disease is transmitted during close contact and by the inhalation of respiratory droplets. The incubation period is commonly six to ten days but may be prolonged (3–4 weeks).

Clinical signs:

Per acutely affected goats can die within 1 to 3 days with minimal clinical signs. Acute symptoms include fever, lethargy, violent coughing, extended necks, laboured breathing, loss of appetite and abortions. In naive herds, the morbidity rate may reach 100% and the mortality rate can be as high as 80%. CCPP causes major economic losses in endemic areas and should be suspected in the field when a highly contagious disease occurs in goats characterized by pyrexia of 41°C or higher, severe respiratory distress, high morbidity and mortality,

Lesions:

Post-mortem examination reveals fibrinous pleuropneumonia with massive lung hepatisation and pleurisy, accompanied by accumulation of straw-colored pleural fluid. CCPP is strictly a respiratory disease.

Diagnosis:

Definitive diagnosis can be made by isolating *M. capripneumoniae* from lung tissue and/or pleural fluid at necropsy. This organism has a branching, filamentous morphology in exudates, impression smears or tissue sections examined under the microscope. Other caprine mycoplasmas usually appear as short filamentous organisms or coccobacilli. Biochemical, immunological and molecular tests can be used for identification of the culture. PCR is the preferred assay for the identification of *M. capripneumonia* through cultures.

Differential diagnosis:

Differential diagnosis must consider bacterial pneumonia (e.g. pasteurellosis), other mycoplasma pneumonia and caseous lymphadenitis

Treatment and control

Treatment:

Early treatment with Tylosin, Oxytetracycline or Enrofloxacin at a dose rate of 10, 10 and 5 mg per kg b.w. respectively, IM, 48 hourly for three times, in a period of 6 days or single dose of long acting antibiotics is effective. However, macrolides especially Tylosin is considered to be the drug of choice against *M. capripneumoniae*.

Antibiotic-based treatment provides temporary kind of relief but for better therapeutic exploration and preventing threat of antibiotic resistance, minimum use of antibiotics should be practiced. If the use of antibiotics is unavoidable, for example, remote livestock rearing areas, alternating effective antibiotics can be adopted. However, better prevention is by vaccination so as to avoid frequent and large-scale use of antibiotics with consequent antimicrobial resistance (AMR) and involvement of huge therapeutic costs.

Control:

Vaccines are of use in preventing the disease. The current CCPP vaccine contains inactivated *M. capripneumoniae* suspended in saponin, has a shelf life of at least 14 months, and provides protection for over 1 year. Administer to animals over 3 months of age at a dose of 1ml per animal. Revaccination should be carried out every 6 months.

Biosecurity measures:

Outbreaks can be eliminated through quarantines, movement controls, slaughter of infected and exposed animals, and cleaning and disinfection of the premises.

2.3.3 Brucellosis

Definition and causative agent:

It is a zoonotic disease that affects humans, cattle, small ruminants, pigs and dogs caused by *Brucella* bacterium spp. a small, Gram-negative, coccobacillus. They are facultative intracellular, capable of growing and reproducing inside of host cells, specifically phagocytic cells. The most important species are *B. melitensis* (in goats, occasionally sheep), *B. abortus* (in cattle, bisons, buffaloes), *B. suis* (in pigs), and *B. canis* (in dogs)..

Transmission:

Infection occurs by ingestion, through mucous membranes, or through broken skin. The bacteria are shed from an infected animal at the time of calving or abortion.

It is present in milk and in both male and female reproductive tracts. *Brucella* spp. can survive in manure, hay, dust, and soil for several months. Humans can be infected by ingestion of contaminated milk or dairy products and by contact with aborted material or blood. Brucellosis in humans is usually a result of occupational exposure to infected animals, but infections can also occur from ingesting contaminated dairy products

Clinical signs:

Brucella spp. causes chronic disease that, if not treated, persists for life. Abortion is the most obvious manifestation. Infections may also cause stillborn or weak calves, retained placentas, and reduced milk yield. Usually, general health is not impaired in uncomplicated abortions. Seminal vesicles, ampullae, testicles, and epididymides may be infected in bulls; therefore, pathogens being present in the semen.

Lesions:

Testicular abscesses may occur. Long standing infections may result in arthritic joints in some cattle.

Diagnosis:

Diagnosis is based on bacteriology or serology. *B. abortus* can be recovered from the placenta but more conveniently in pure culture from the stomach and lungs of an aborted fetus. Most cows cease shedding organisms from the genital tract when uterine involution is complete. Foci of infection remain in some parts of the reticuloendothelial system, especially supramammary lymph nodes, and in the udder. Udder secretions are the preferred specimens for culture from a live cow.

Serum agglutination tests have been the standard diagnostic method. Agglutination tests may also detect antibodies in milk, whey, and semen. An ELISA has been developed to detect antibodies in milk and serum. When the standard plate or tube serum agglutination test is used, complete agglutination at dilutions of 1:100 or more in serum samples of non-vaccinated animals, and of 1:200 of animals vaccinated at 4–12 months of age, are considered positive, and the animals are classified as reactors. Other tests that may be used are complement fixation, rivanol precipitation, and acidified antigen procedures.

Screening Tests:

In official eradication programs on an area basis, the *Brucella* spp. milk ring test (BRT) has effectively located infected dairy herds, but there are many false-positive tests. The brucellosis status of dairy herds in any area can be monitored by implementing the BRT at 3- to 4-months intervals. Cows in herds with a positive BRT are individually blood tested, and seropositive cows are slaughtered to determine herd status.

Nondairy and dairy herds in an area may also be screened for brucellosis by testing serum samples collected from cattle destined for slaughter or replacements through intermediate and terminal markets, or at abattoirs.

Differential diagnosis:

The disease should be diffentiated from Trichomoniasis, Vibriosis, Leptospirosis and IBR.

Treatment and control:

Treatment:
No practical treatment is available.

Control:

Vaccination of calves with *B. abortus* Strain 19 or RB51 increases resistance to infection. With S19 vaccine vaccinate calves between 3 and 6 months of age with 2ml of the vaccine SC after reconstitution with respective diluent. Resistance may not be complete, and some vaccinated calves may become infected, depending on severity of exposure. A small percentage of vaccinated calves develop antibodies to S19 that may persist for years and can confuse diagnostic test results. To minimize this problem, calves may be vaccinated with a vaccine of Strain RB51. Vaccination with Strain RB-51 after reconstitution is by SC to female cattle at the age of 4-12 months of age. Do not vaccinate pregnant animals and those within 3 weeks before slaughter.

Biosecurity measures:

Eventual eradication depends on testing and eliminating reactors. The disease has been eradicated from many individual herds and areas by this method. Herds must be tested at regular intervals until two or three successive tests are negative.

2.3.4 Chlamydiosis or enzootic abortion

Definition and causative agent:

This is a zoonotic disease caused by the bacterium *Chlamydia abortus* and is one the most common infectious causes of abortion and the birth of weak lambs.

Transmission:

Infection usually occurs via the oral-nasal route through the ingestion of chlamydia present in contaminated water or food, or through the licking and ingestion of placental residues. It occurs in the last 2 to 3 weeks of pregnancy.

Clinical signs:

Abortion is the most evident clinical sign of infection by *C. abortus*. Abortion rates can reach 40% of pregnant animals. This high percentage of abortions in ewes are accompanied by a high mortality rate of lambs and a reduction in milk production. In addition, *C. abortus* can also be a cause of infertility and affect early lamb development. Animals that have been infected before pregnancy show no clinical signs of infection. Infected ewes remain latent carriers of the infection, until the microorganism reactivates in the next pregnancy.

Lesions:

Acute pulmonary lesions include bronchiolitis and pulmonary edema may occur.

Diagnosis:

Chlamydia infections require collection of swab nasal, ocular, rectal or vaginal samples, tracheal washings or broncho-alveolar lavage fluid. Newer diagnostic methods based on the detection of chlamydial nucleic acid, either by direct hybridization or preferably by nucleic acid amplification are being used. The latter uses a variety of amplification reactions, including the PCR, ligase chain reaction (LCR), and strand displacement amplification or transcription-mediated.

Differential diagnosis:

The disease must be differentiated from other causes of abortion in small ruminants that include Toxoplasmosis, Leptospirosis, Brucellosis, and Q fever.

Treatment and control:

Treatment:

The antibiotics of choice in veterinary medicine for the treatment of *C. abortus* infections are doxycycline (10mg/kg)(IM), or tetracycline(10mg/kg) (IM) and the fluoroquinolone enrofloxacin administered orally (feed/drinking water) or parenterally (IM or SC).

Control:

Vaccination can help to control the disease, but variable efficacy values have been described, possibly associated with factors related to both the host and the vaccine. Currently, two types of vaccine (inactivated and attenuated live vaccines) are available commercially.

A single administration of a live vaccine (2ml SC or IM) to the females at the age of 5 months or two to three weeks prior to breeding has been proven to reduce abortion and shedding for up to 3 years.

Biosecurity measures:

Importation of infected small ruminants should be avoided.

2.3.5 Anthrax

Definition and causative agent:

It is a highly infectious and fatal disease of humans and all worm-blooded animals caused by a relatively large spore-forming rectangular shaped bacterium called *Bacillus anthracis*. The bacteria produce extremely potent toxins which are responsible for the ill effects. Animals such as deers, cattle, goats, and sheep, are highly affected by this disease.

Transmission:

Infection can occur through ingestion of anthrax spores, inhalation (breathing in) the spores, and direct contact with contaminated materials.

Clinical Signs:

Signs of the illness usually appear 3 to 7 days after the spores are swallowed or inhaled. Sudden death (often within 2 or 3 hours of being apparently normal) is by far the most common sign.

Very occasionally, some animals may show trembling, and a high temperature. Difficulty breathing, collapse and convulsions before death. This usually occurs over a period of 24 hours.

Lesions:

Oozing black blood, which may not clot, resulting in a small amount of bloody discharge from the nose, mouth and other openings, and lack of rigor mortis may be observed.

Diagnosis:

The optimal sample is a blood swab taken for laboratory analysis and blood smear. This results in sporulation of *B. anthracis* which overwhelm the growth of other bacteria and contaminants. Swabs should remain closed until they get into the laboratory for processing.

Specific diagnostic tests include bacteria culture, PCR and fluorescent antibody stains to demonstrate the agent in blood films or tissues.

Differential diagnosis:

Anthrax may be difficult to differentiate from other causes of sudden death such as lightning strikes, per acute Blackleg, acute Leptospirosis, Bacilliary haemaglobinuria, per acute lead poisoning, Hypomagnesaemic tetany and acute bloat.

Treatment and control:

Treatment:

Due to the acute nature of the disease resulting in sudden death, treatment is usually not possible in animals.

Control:

Vaccination may be carried out at least a month prior to expected disease occurrence in endemic areas. A freeze-dried live bacterial vaccine is used, and is reconstituted in100ml of sterile saline water before use at a dose of 1ml in cattle above 3 months age and at 0.5ml SC in sheep and goats above 3 months of age. Blanthrax, a combined vaccine for Anthrax an Black quarter is available.

Biosecurity measures:

Never open a carcass of an animal suspected to have died from anthrax. Contact the competent veterinary authority immediately if anthrax is suspected and seek advice on measures to be adopted.

2.3.6 Black quarter (Black-leg)

Definition and causative agent:

Black quarter is an acute infectious disease of cattle and sheep manifested by severe inflammation of the muscle with high mortality. It is caused by *Clostridium chauvoei*. In cattle, blackleg infection is endogenous and lesions may develop without any history of wounds. The case fatality rate approaches 100%.

Transmission:

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

Clinical signs:

Usually, onset is sudden, and a few cattle may be found dead without premonitory signs. Acute, severe lameness and marked depression are common. Initially, there is a fever but, by the time clinical signs are obvious, body temperature may be normal or subnormal. General signs include prostration and tremors. Death occurs within 12–48 hrs. Characteristic edematous and crepitant swellings develop 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 with decreased blood supply to affected areas. Most cases are seen in cattle from 6–24 months old, but thrifty calves as young as 6 weeks and cattle as old as 10–12 years may be affected.

Lesions:

In some cattle, the lesions are restricted to the myocardium and the diaphragm. The affected muscles are dark red to black and dry and spongy, have a sweetish odor, and are infiltrated with small bubbles but little edema. The lesions may be seen in any muscle, even in the tongue or diaphragm. In sheep, because the lesions of the spontaneously occurring type are often small and deep, they may be overlooked.

Diagnosis:

At times, both *C. septicum* and *C. chauvoei* may be isolated from blackleg lesions, particularly when the carcass is examined ≥24 hrs after death, which allows time for postmortem invasion of the tissues by *C septicum*.

Field diagnoses are confirmed by laboratory demonstration of *C. chauvoei* in affected muscle (standard methods: culture and biochemical identification). The samples of muscle should be taken as soon after death as possible. The fluorescent antibody test for *C. chauvoei* is rapid and reliable. A PCR is available and reported to be very good for clinical samples but not for environmental samples.

Differential diagnosis:

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

Treatment and control:

A multivalent vaccine containing *C. chauvoei*, *C. septicum* and, where needed, *C. novyi* antigens are safe and reliable for cattle and sheep. Calves 3–6 months of age should be vaccinated twice, 4 weeks apart, followed by annual boosters before the anticipated danger period.

Blanthrax containing a virulent strain of *B. anthracis* and *C. chauvoei* is available at a dose of 2ml to cattle, sheep and goats at 6 months of age and annually thereafter. Administration of antibiotics should be avoided until 2 weeks after vaccination

Naive ewes should be vaccinated twice, 1 month before lambing and then with annual boosters.

Biosecurity measures:

Carcasses should be destroyed by burning or buried deeply in a fenced-off area to limit heavy spore contamination of the soil.

2.3.7 Tetanus

Definition and causative agent:

This is an infectious, non-febrile disease of animals and man caused by a bacterium *Clostridium tetanii*, and is characterized by spasmodic tetany and hyperaesthesia. This disease is prevalent all over the world. Tetanus affects many species of domesticated animals but occurs particularly in horses and lambs; less frequently in adult sheep, goats, cattle, pigs, dogs and cats; and rarely in poultry.

Transmission:

Deep punctured wounds provide favorable conditions for the spores to germinate, multiply and produce toxin, which is subsequently absorbed in the animal body.

Clinical signs:

The incubation period is generally 1-2 weeks but it may be as short as 3 days. The initial symptoms are mild stiffness and an unwillingness to move in all the affected animals. More severe symptoms develop after 12-24 hours, which are stiffness of limbs, neck, head, tail and twitching of muscles. The spasms develop in response to noise. In terminal stages ears are erect, nostrils dilated, and nictitating membrane protruded. Mastication becomes very difficult because the mouth cannot be opened, hence the name lockjaw.

Diagnosis:

Involves isolation and identification of *C. tetani* by enrichment of culture from tissue or exudate of suspected wound and detection of toxigenicity in the isolate by mouse toxicity testing.

A sensitive method of *C. tetani* detection depends on PCR based on tent containing clostridia.

Differential diagnosis:

Can be confused with intoxications due to strychnine or meningitis. A differential diagnosis should be made with cervical vertebral fracture, cervical osteomyelitis, colic, pleuritis, laminitis, meningitis or myopathies.

Treatment and control:

Treatment:

Antitoxin is of very little use unless given in the very early stages of infection. Sedatives and muscular relaxants can aid recovery. Cattle with developed tetanus it is not worth treating.

Control:

Proper hygiene and cleanliness at castration and other surgical procedures should be observed. A vaccine (Tetanus Toxoid) for horses, cattle, sheep and goats is available and is administered at a dose of 1ml IM and a second injection of 1ml IM at an interval of 8 weeks after the first dose. Revaccinate annually.

Biosecurity measures:

Correct hygiene practices are necessary; and avoid contact of wounds with potentially contaminated material.

2.3.8 Campylobacteriosis (Vibriosis)

Definition and causative agent:

It is a venereal disease of cattle caused by *Campylobacter* spp. and transmission occurs by coitus, ingestion and other means. *C. fetus* subspp. *venerealis* is the main cause venereal disease of cattle.

C. jejuni also known as *C. fetus* subspp. *jejuni* is a normal inhabitant of the intestinal tract of cattle, sheep, goats and many spp. of birds. *C. jejuni* is also recognized as a cause of stillbirth and late abortion in goats and sheep and may cause mastitis in cattle.

Transmission:

The affected bulls carry the organisms in preputial cavity indefinitely remaining as asymptomatic carriers. Mature cows and heifers also carry the infection for long periods. Infected semen from an infected bull is the important means of the disease transmission.

Clinical signs:

The disease is insidious and often unrecognized in herds causing production losses such as abortions, poor conception rates, long calving intervals, uterine infections, and gastroenteritis. Abortions usually occur between fifth and sixth month of pregnancy. Infected bulls show no symptoms and their semen is normal.

Healthy bulls become infected during coitus with diseased cows. Among sheep the disease is characterized by abortion occurring towards the end of gestation. Usually abortion is preceded by vaginal discharge for several days.

Lesions:

The aborted foetus is edematous with petechial hemorrhages on serous surfaces and necrotic foci in the liver.

Diagnosis:

The disease is confirmed by measuring antibodies against *C. fetus* in the cervicovaginal mucus of an infected cow or heifer by using an agglutination test or ELISA.

Treatment and control:

Treatment:

Treatment of bulls has not been perfected.

Control:

For practical reasons, cows are not usually treated for genital campylobacteriosis. When practical, artificial insemination is an excellent way to prevent or control genital campylobacteriosis. Because *C. fetus* has been isolated from cows for >6 months after the end of pregnancy, it has been suggested that artificial insemination should continue until all the cows in a herd have been through at least two pregnancies.

In routine use, the vaccine in an oily adjuvant is available (vibrobrax) for use in bulls given two 5ml doses a minimum of 4 weeks apart before breeding starts; and in heifers given a single dose of 5ml after 18 months of age (SC). An annual booster dose of 2ml or convenience of 5ml dose every two years will provide ongoing immunity.

Biosecurity measures:

Biosecurity screening of bulls bought in can help identify the disease. Use of borrowed bulls should be avoided.

2.3.9 Mastitis

Definition and causative agent:

Mastitis is an infectious disease condition resulting in an inflammatory reaction in the mammary gland and it may be accompanied by signs of inflammation in the mammary gland including swelling, redness, and painfulness. Although, stress and physical injuries may cause inflammation of the gland, infection by invading bacteria or other microorganisms (fungi, yeasts and possibly viruses) are the primary cause of mastitis. Mastitis can be categorized as subclinical, clinical, and severe clinical and mild clinical.

Transmission:

Infections begin when microorganisms penetrate the teat canal and multiply in the mammary gland. Transmission occurs at milking with either milkers' hands or milking equipment acting as fomites.

2.3.9.1 Subclinical mastitis

Subclinical mastitis is the presence of an infection without apparent signs of local inflammation or systemic involvement.

Clinical signs:

Although transient episodes of abnormal milk may appear, subclinical mastitis is, for the most part, asymptomatic. If the infection persists for at least two months, the infection is termed chronic. Once established, many of these infections persist for entire lactations or the life of the cow, although this varies with the causative pathogen.

Although any number of quarters can be infected simultaneously in subclinical mastitis, typically only one quarter will display clinical mastitis. However, it is not uncommon for clinical episodes caused by *Mycoplasma* to affect multiple quarters. Gangrenous mastitis can also occur, particularly when subclinical, chronic infections of *Staphylococcus aureus* become severe at times of immune dysfunction (e.g., at parturition).

Lesions:

The udder has no obvious lesions.

Diagnosis:

Milk samples should be collected from affected quarters and tested for somatic cell count (SCC) in milk using the California Mastitis Test (CMT). Culture of milk samples collected from affected quarters is the only reliable method to determine the etiology of clinical cases.

Treatment and control:

Treatment:

Therapy for subclinical mastitis is given on the premise that treatment costs will be outweighed by production gains after elimination of infection and that prevention is always preferred.

Drug distribution after intra-mammary administration may not be adequate because of extensive fibrosis and micro abscess formation in the gland. Cows with a long duration of infection (>3 months), more than one quarter infected, are likely to be refractive to therapy.

An unusual opportunity for successful therapy of subclinical intramammary infection (IMI) is possible with *Staphylococcus. agalactiae*. Prevalence of IMI caused by this pathogen can be rapidly reduced by treating all the infected cows in a herd with antimicrobials. Cure rates often range from 75% to 90%. Labeled use of commercial intra-mammary products that contain amoxicillin, penicillin, or cephalosporin at a dose of 10ml with concentration of (600mg/ml) for infusion in the mammary gland per quarter for 7 to10 days is preferred.

Use of drugs originating from multiple-dose vials (labeled for systemic therapy) should not be used for intra-mammary therapy, because commercial intra-mammary preparations have superior quality control standards for sterility and better reliability to predict withholding periods for milk and meat after treatment.

Dry Cows

The dry period of the lactation cycle is a critical time for the udder health of dairy cows. The mammary gland undergoes marked biochemical, cellular, and immunologic changes. Consequently, the dry period is an ideal time to attain synergy between antimicrobial therapy and immune function, without incurring the extensive costs typical of lactating cow therapy.

Numerous commercial products are available for dry cow treatment and include penicillin, cloxacillin, cephapirin, ceftiofur, or novobiocin. One tube with 10mls per quarter is sufficient and should be administered immediately after the last milking of lactation.

Internal teat sealants, as a supplemental infusion after antimicrobial infusions at dry off, serve as a physical barrier to help reduce new infections.

Blanket dry cow therapy (BDCT; treating all quarters of all cows at dry off) has been a foundation of mastitis control for more than 50 years.

Heifers

Most IMI in calving heifers are caused by staphylococcal species other than *S. aureus*, which have a high rate of spontaneous cure. However, under some herd conditions, a substantial portion of heifers have more intractable infections, including those caused by *S. aureus*.

Intramammary infusions of beta-lactam antibacterial drugs (penicillin or cephalosporins) (10mls tube) 7–14 days before expected calving dates have been reported to reduce the prevalence of IMI at calving.
Control:

Control of new infections by focusing management efforts on reducing the presence of pathogens on the teat end by ensuring clean and dry bedding, clean and dry udders at the time of milking is recommended.

For contagious pathogens, the single most important management practice to prevent transmission of new infections is the use of an effective germicide as a post milking teat dip. These products should be applied as a dip (rather than a spray) immediately after milking. Other practices that augment teat hygiene include use of individual towels to dry teats, gloves for milkers' hands, use of a pre milking germicide (spray or dip).

Practices that have a positive impact on environmental mastitis control include:

- regular cleaning or changing of bedding
- reducing heat stress
- removing udder hair
- preventing teat trauma
- avoidance of areas that accumulate water
- maintenance of stalls for proper lying behavior
- preventing frost bite and fly exposure

2.3.9.2 Clinical Mastitis

Except for outbreaks of *Mycoplasma*, clinical mastitis in most dairy herds is caused by environmental pathogens. In addition, many clinical mastitis cases are transient, especially those that are initial episodes for a cow and quarter. Thus, assessment of clinical mastitis is based on incidence and not prevalence.

The balance between host defenses and invading pathogens causes a marked inflammatory response, and clinical signs become apparent. Infections from any pathogen can be clinical or subclinical, depending on the duration of infection, host immune status, and pathogen virulence.

Transmission:

Infections begin when microorganisms penetrate the teat canal and multiply in the mammary gland; transmission occurs at milking with either milkers' hands or milking equipment acting as fomites

Lesions:

The most obvious symptoms of clinical mastitis are abnormalities in the udder such as swelling, heat, hardness, redness, or pain; and milk may have a watery appearance, flakes, clots, or pus.

Diagnosis:

Cow history from each case (eg, season, age, stage of lactation, and previous episodes) should be recorded to help determine risk factors. Milk samples should be collected from affected quarters and, when feasible, antimicrobial susceptibility testing performed.

Typically, 30%–40% of milk samples collected from clinical mastitis cases yield no organisms on culture. However, of the samples that do yield organisms, 90%–95% of the isolated bacteria include a wide variety of streptococci, staphylococci, or coliforms. If this is not the case, especially if non-coliform, Gram-negative rods, mycotic, or algal (*Prototheca* spp.) pathogens predominate, a point source of infection should be considered.

Treatment and control:

Treatment and control of clinical mastitis is similar to control of subclinical mastitis.

2.3.9.3 Severe Clinical Mastitis

Coliforms (lactose-fermenting Gram-negative rods of the family Enterobacteriaceae) are the most common cause of this form of mastitis. Most coliform infections are cleared from the gland with few or mild clinical signs. However, severe mastitis (systemic signs) occurs when bacterial concentrations in milk increase enough to stimulate a marked immune response.

Clinical signs:

Severe mastitis caused by coliforms results in a higher incidence of cow death or agalactia-related culling (30%–50% of cases) than mastitis caused by other pathogens (5%–10% of cases). Prognosis for cases of *Klebsiella* infection is particularly guarded, because affected cows are twice more likely to be culled or die than those infected by other coliforms.

After infection, coliform numbers in milk increase rapidly, often attaining peak concentrations within a few hours. Inflammatory and systemic changes that result during severe coliform mastitis are caused by release of lipopolysaccharide (LPS) endotoxin from the bacteria and subsequent activation of cytokine and arachidonic acid-derived mediators of inflammation; this causes the acute phase response (sepsis).

Diagnosis:

Milk samples should be collected for culture and confirmation is by isolation of the bacteria.

Treatment and control:

Primary therapy for severe clinical mastitis should be directed against coliform organisms, although secondary consideration must be given for other causative agents. Supportive care, including fluids, is likely the most beneficial component of the therapeutic regimen.

Antimicrobial therapy is ideally based on identification of the causative pathogen. However, this is not attainable for some hours after initial case recognition. In addition, systemically administered antimicrobial regimens administered for severe clinical mastitis are not labeled.

By the time therapy is initiated, much of the LPS exposure has occurred. Thus, the primary therapeutic concern is the treatment of LPS-induced shock with fluids, electrolytes, and anti-inflammatory drugs. The IV route is preferred as the initial method of fluid administration.

Isotonic saline is administered, 30–40 L are necessary throughout a 4-hour period, which can be difficult under farm conditions. A practical alternative is 2 L of 7% NaCl (hypersaline) administered IV. This induces rapid fluid uptake from the body compartment into the circulation. Cows should then be offered free-choice water to drink, and if at least 40 to 50 litres is not consumed, 25–35 litres should be pumped into the rumen. Many cows with endotoxic shock are marginally hypocalcemic; thus, 500 ml of calcium borogluconate should be administered SC (to avoid potential complications of IV administration).

Alternatively, rapid absorption calcium gels, designed for periparturient hypocalcemia, can be given. If the cow remains in shock, continued fluid therapy should be administered per os (PO) or IV as isotonic, not hypertonic, fluids.

Administration of dexamethasone (30 mg/kg B.W, IM) to dairy cows immediately after introduction of *E coli* into the mammary gland has been reported to reduce mammary gland swelling and improve rumen motility. Care should be exercised in administering these drugs to pregnant animals; however, severe clinical mastitis in and of itself may cause pregnancy loss in cattle.

Flunixin meglumine is labeled for beef and dairy cattle and is therefore the most logical choice to treat severe clinical mastitis at a dosage of 1.1 mg/kg (IV) slowly once a day or twice a day at an interval of 12 hours for three days or 2.2 mg/kg (I.V) once a day. It reduces the severity of clinical signs such as fever, depression, heart and respiratory rates, and udder pain. Withdrawal intervals are 4 days for slaughter and 36 hours for milk when used as labeled by IV administration. Because of extensive and unpredictable withdrawal periods, this drug should *not* be administered by IM injection.

Antimicrobial therapy may be of secondary importance relative to immediate supportive treatment of endotoxic shock, but it remains an integral part of a therapeutic regimen. Intramammary infusion of antimicrobials should be administered to cows with severe clinical mastitis. This treatment may not affect the outcome of coliform cases but will likely improve outcomes for cases caused by Gram-positive cocci.

2.3.9.4 Mild Clinical Mastitis

As previously stated, microorganisms are not isolated from 30%–40% of bacteriologic cultures of milk samples collected from cows with clinical mastitis. Many mild mastitis cases that fail to yield bacteria on culture are coliform IMI that resolve before treatment is necessary. In addition, numerous mild clinical mastitis cases are temporary setbacks in the balance between pathogen and host defenses that occurs in chronic IMI.

Common sense and individual herd history should determine the course of therapy for mild clinical mastitis cases in dairy herds.

Diagnosis:

Simple milk bacteriology that identifies Gram-positive from Gram-negative pathogens, as well as no isolation, can be performed in veterinary clinics, or with proper training, on the farm.

Treatment and control:

Use of approved commercial intramammary infusions is the best option as in subclinical mastitis. The foundation of success is bacteriologic cure but will be more practically based on return to normal milk. If mastitis recurs regularly in affected quarters in the absence of systemic signs, repeated treatment of what now has become a chronic IMI is not warranted.

If standard regimens achieve less than desired results, it would be better to extend initial therapy for a prolonged period rather than to change to other antimicrobial drugs or increase the amount of each dose. Studies recommend intramammary infusions for up to 8 days as compared with 2 days. The most efficient use of antimicrobial therapy, and the best option to reduce unnecessary use, is to apply "culture-based therapy" Therapy is withheld from an affected quarter (mild clinical mastitis) until results from a bacteriologic culture of a milk sample are obtained, usually within 24–48 hours.

2.3.10 Q fever

Definition and causative agent:

Also called Query Fever, Coxiellosis, Abattoir Fever is a zoonotic disease caused by an obligate intracellular polymorphic bacillus, *Coxiella burnetii*, which has a cell membrane similar to Gram-negative bacteria. The main reservoir for the pathogen is domestic animals such as cattle, sheep and goats.

Transmission:

The main route of transmission of the disease is inhalation of the bacteria from the infected environment and ingestion of contaminated food or water with discharge of infected animals. Ticks act both as vectors and as reservoirs of the causative agent of Q fever, but this is not the most important route of infection for livestock. Infected animals usually shed the agent intermittently in milk, feces and urine with no outward signs of disease and should be regarded as possible sources of human infection.

Clinical signs:

In cows, ewes and goats, Q fever has been associated with late abortion and reproductive disorders such as premature birth and weak offspring and with infertility and mastitis in cattle. Although mortality is a rare outcome of the acute form of the disease, the major clinical manifestation of chronic form of Q fever is endocarditis with case fatality in untreated cases exceeding 10%.

Q fever is a worldwide zoonosis, which may occur in sporadic as well as epidemic forms. However, it may be an emerging disease, probably related to climate change.

This disease causes significant reproductive losses in both cattle and small ruminants, due to losses from abortion in small ruminants and infertility in cattle.

Lesions:

Gross lesions are none specific.

Diagnosis:

Culture, immunohistochemical and PCR tests may be used to identify the *C. burnetti* in tissues.

Differential diagnosis:

In animals, differential diagnosis include other causes of abortion and infertility that are Leptospirosis, Brucellosis, IBR, Campylobacteriosis, BVD, Listeriosis, Salmonellosis, Chlamydiosis, Mycotic abortion and severe deficiency in vitamins A, E or selenium.

Treatment and control:

It is not advised to treat the disease in cattle.
The aim of vaccination against *C. burnetii* is to reduce shedding and the risk of abortion. There is no licensed vaccine to prevent Q-fever in livestock

2.4 Ruminant Parasitic Diseases

2.4.1 Animal African Trypanosomosis (AAT)

AAT is a protozoan parasitic disease of cattle, water buffaloes, sheep, goats, horses, pigs, dogs and other species, also known as *nagana pest*. Trypanosomoses affecting cattle are the most important economically since they are a major cause for reduced meat and milk production and draught power for agricultural production. ATT is caused by trypanosomes from the genus *Trypanosoma* predominantly by species *T. congolense* and *T. vivax*, and to a lesser extent by *T. brucei brucei*.

Transmission:

The trypanosomes are mainly transmitted by tsetse flies. Most trypanosomes develop for one to a few weeks in tsetse flies (*Glossina* spp.), which act as biological vectors. The parasites are transmitted by saliva when the fly bites the animal. Trypanosomes can also be spread by fomites and mechanical vectors including surgical instruments and various biting flies including horse flies (especially *T. vivax*).

Clinical signs:

The incubation period is four days to approximately eight weeks. Although acute cases can be seen, trypanosomosis is often a chronic disease in susceptible animals. The trypanosomes infect the blood of the host causing fever, weakness, lethargy and anaemia, which lead to weight loss as well as reduced fertility and milk production. In cattle, the mortality rate can reach 50-100% within months after exposure, particularly when poor nutrition or other factors contribute to debilitation.

Lesions:

Necropsy findings vary and are nonspecific. In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen. In chronic cases, swollen lymph nodes, serous atrophy of fat, and anemia are seen.

Diagnosis:

A presumptive diagnosis is based on finding an anemic animal in poor condition in an endemic area. Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. The most sensitive rapid method is to examine a wet mount of the buffy coat area of a PCV tube after centrifugation, looking for motile parasites.

Various serologic tests measure antibody to trypanosomes, but their use is more suitable for herd and area screening than for individual diagnosis.

Rapid agglutination tests to detect circulating trypanosome species-specific antigens in peripheral blood are available for both individual and herd diagnosis, although their reliability remains varied. Molecular techniques for trypanosome detection and differentiation have been developed, but they are not generally available for routine field use.

Differential diagnosis:

Other infections that cause anemia and weight loss, such as Babesiosis, Anaplasmosis, Theileriosis, and Haemonchosis, should be differentiated from AAT.

Treatment and control:

Treatment:

Several drugs can be used for treatment. Most have a narrow therapeutic index, which makes administration of the correct dose essential. Drug resistance occurs and should be considered in refractory cases. The Drugs for treatment and prophylaxis are Isometamidium chloride at a dose rate of 0.25 to 0.5 mg/kg b.w. (IM) for treatment and 0.5 to 1 mg/kg b.w. (IM) for propylaxis. Diminazene diaceturate at 3.5 mg/kg b.w. (IM) is administered for treatment purposes.

Control:

AAT can be controlled by reducing tsetse fly populations through dipping by using pyrethroid formulation, traps and insecticides.

Animals can be given antiparasitic drugs prophylactically in areas with a high population of trypanosome-infected tsetse.

No vaccine is yet to be available for this parasitic disease. Control is ideally achieved by combining methods to reduce the tsetse challenge and by enhancing host resistance with prophylactic drugs.

2.4.2 Bovine Babesiosis (Tick fever)

Definition and causative agent:

It is an acute disease of cattle characterized by fever (frequently 41°C), which persists throughout, and is accompanied later by in appetence, increased respiratory rate, muscle tremors, anemia, jaundice, and weight loss. Hemoglobinemia and hemoglobinuria occur in the final stages. It is a tick-borne disease of cattle caused by the protozoan parasites of the genus *Babesia*, order *Piroplasmida*, phylum *Apicomplexa*. The principal species of *Babesia* that cause Babesiosis are *Babesia bovis*, *Babesia bigemina* and *Babesia divergens*.

Transmission:

Transmission is by ticks with *Rhipicephalus* ticks being the major vector, when feeding on another animal. In endemic areas, cattle become infected at a young age and develop a long-term immunity. However, outbreaks can occur in these endemic areas if exposure to ticks by young animals is interrupted or immuno-naïve cattle are introduced.

Clinical signs:

There is high fever, neurologic signs such as incoordination, teeth grinding and mania. Some cattle may be found on the ground with the involuntary movements of the legs. When the nervous symptoms of cerebral babesiosis develop, the outcome is almost always fatal.

Morbidity and mortality vary greatly and are influenced by prevailing treatments employed in an area, previous exposure to a species/strain of parasite, and vaccination status. Dark colored urine is seen, anorexia and animals likely to separate from herd, be weak, depressed and reluctant to move. Animals that recover from the acute disease remain infected for a number of years with *B. bovis* and for a few months in the case of *B. bigemina*. No clinical signs are apparent during this carrier state.

Lesions:

Lesions include an enlarged and friable spleen, a swollen liver with enlarged gallbladder containing thick granular bile. Pale or icteric mucous membranes and blood may appear thin and watery. Subcutaneous tissues, abdominal fat and omentum may appear icteric. Kidneys appear darker than normal with possible petechial haemorrhages. Bladder may contain dark red or brown-colored urine.

Diagnosis:

Microscopic examination of Giemsa-stained thick and thin blood films.
PCR-based techniques are reported to be at least 1000 times more sensitive than thin blood smears for detection of *B. bovis*. A competitive ELISA is apparently in routine use.

Differential diagnosis:

Other infections that cause anemia such as Anaplasmosis, AAT, Theileriosis, and Haemonchosis, should be differentiated from Babesiosis.

Treatment and control:

Treatment:

Mild cases may recover without treatment. Sick animals can be treated with an antiparasitic drug Diminazene diaceturate at 3.5mg/kg b.w. (IM). Treatment is most likely to be successful if the disease is diagnosed early; it may fail if the animal has been weakened by anemia.

Imidocarb dipropionate at a dose of 3.0mg/kg B.W (SC or IM) once has been reported to protect animals from disease but immunity can develop. There are also concerns with regard to residues in milk and meat and withdraw period should be adhered to.

Control:

Effective control of tick fevers has been achieved by a combination of measures directed at both the disease and the tick vector.

Tick control by acaracide dipping is widely used in endemic areas.
Dipping may be done as frequently as every 4-6 weeks in heavily infested areas.

Babesiosis vaccines are available in the market, but have been not in use in Tanzania and are highly effective.

2.4.3 East Coast Fever (ECF)

Definition and causative agent:

Is an acute disease of cattle, usually characterized by high fever, swelling of the lymph nodes, dyspnea, and high mortality caused by a protozoa *Theileria parva*.

Transmission:

Transmission is by the tick vector *Rhipicephalus appendiculatus*. The African buffalo (*Syncerus caffer*) is an important wildlife reservoir of *T. parva,* but infection is asymptomatic in buffaloes.

Clinical signs:

Signs vary according to the level of challenge, and they range from in apparent or mild to severe and fatal. Typically, fever occurs 7–10 days after parasites are introduced by feeding ticks, continues throughout the course of infection, and may reach 41°C.

Lymph node swelling becomes pronounced and generalized. Anorexia develops, and the animal rapidly loses condition. Lacrimation and nasal discharge may occur. Terminally, dyspnea is common; just before death, a sharp drop in body temperature is usual, and pulmonary exudate pours from the nostrils. Death usually occurs 18–24 days after infection.

Animals that recover are immune to subsequent challenge with the same strains but may be susceptible to some heterologous strains. Most recovered or immunized animals remain carriers of the infection.

Lesions:

The most striking postmortem lesions are lymph node enlargement and massive pulmonary edema and hyperemia. Hemorrhages are common on the serosal and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Anemia is not a major diagnostic sign (as it is in Babesiosis) because there is minimal division of the parasites in red blood cells, and thus no massive destruction of them.

Diagnosis:

Giemsa–stained blood films from infected cattle reveal the presence of macroshizonts as Koch's Blue Bodies in lymph node aspiration. Pathology includes anorexia, dyspnea, corneal opacity, nasal discharge, frothy nasal discharge, diarrhea, pulmonary edema, leukopenia, and anemia.

Differential diagnosis:

The disease should be diffentiated from Anaplasmosis and Babesiosis.

Treatment and control:

Treatment:

Since the early 1990s, Buparvaquone as a second-generation hydroxynaphthoquinone is used in bovine theileriosis with remarkable results of 90 to 98% recovery rate, twenty times more active than parvoquone. Treatment at a dose 2.5 mg/kg b.w. deep IM injection into neck muscles. A single treatment is usually sufficient, but may be repeated after 48-72 hours interval if necessary and in severe cases. Milk and meat withholding should be observed as per manufactures instruction.

Other chemotherapeutic options are the Parvaquones that have shown to have an 80.5% efficacy against *T. parva parva* infections.

Treatment with parvaquone at a dose of 10 mg/kg b.w. IM injection into neck muscles and repeated at 48 hours interval. Milk withholding is 14 days and for meat 28 days.

Control:

Immunization of cattle against *T. parva* using an infection-and-treatment procedure is practical and continues to gain acceptance. Cattle should be immunized 3–4 weeks before being allowed on infected pasture. Incidence of ECF can be reduced by rigid tick control and immunization.

2.4.5 Bovine Anaplasmosis

Definition and causative agent:

Clinical bovine anaplasmosis is usually caused by *Anaplasma marginale*. Cattle are also infected with *A. centrale*, which generally results in mild disease. Bovine anaplasmosis is of economic significance in the cattle industry.

Transmission:

Up to 17 different tick vector species (including *Dermacentor*, *Rhipicephalus*, *Ixodes*, *Hyalomma*, and *Argas*) have been reported to transmit *Anaplasma* spp. Anaplasmosis may also be spread through the use of contaminated needles or dehorning or other surgical instruments.

Clinical signs:

The urine may be brown but, in contrast to Babesiosis, hemoglobinuria does not occur. A transient febrile response, with the body temperature rarely exceeding 41°C occurs at about the time of peak rickettsemia. Mucous membranes appear pale and then yellow. Pregnant cows may abort. Surviving cattle convalesce over several weeks, during which hematologic parameters gradually return to normal.

Lesions:

Lesions are typical of those found in animals with anemia due to erythrophagocytosis. The carcasses of cattle that die from Anaplasmosis are generally markedly anemic and jaundiced. Blood is thin and watery. The spleen is characteristically enlarged and soft, with prominent follicles. The liver may be mottled and yellow-orange.

The gallbladder is often distended and contains thick brown or green bile. Hepatic and mediastinal lymph nodes appear brown. There are serous effusions in body cavities, pulmonary edema, petechial hemorrhages in the epi- and endocardium, and often evidence of severe GI stasis. Widespread phagocytosis of erythrocytes is evident on microscopic examination of the reticuloendothelial organs. A significant proportion of erythrocytes are usually found to be parasitized after death due to acute infection. At necropsy, thin blood films of liver, kidney, spleen, lungs, and peripheral blood should be prepared for microscopic examination.

Diagnosis:

In Giemsa-stained thin blood films, *Anaplasma* spp. appear as dense, homogeneously staining blue-purple inclusions 0.3–1 µm in diameter. *A. marginale* inclusions are usually located toward the margin of the infected erythrocyte, whereas *A. centrale* inclusion bodies are located more centrally. *A. caudatum* cannot be distinguished from *A. marginale* using Giemsa-stained blood films.

Chronically infected carriers may be identified with a fair degree of accuracy by serologic testing using the msp5 ELISA, complement fixation, or card agglutination tests. Nucleic acid–based detection methods are most useful, because species and strain differentiation tests may not detect carrier levels.

Differential diagnosis:

The disease should be differentiated from other infections such as AAT, Theileriosis, Haemonchosis, and Babesiosis.

Treatment and control:

Treatment:

Tetracycline antibiotics and imidocarb are currently used for treatment. Cattle may be sterilized by treatment with these drugs and remain immune to severe Anaplasmosis subsequently for at least 8 months.

Prompt administration of tetracycline drugs (tetracycline, chlortetracycline, oxytetracycline, rolitetracycline, doxycycline, minocycline) in the early stages of acute disease (eg, PCV >15%) usually ensures survival. A commonly used treatment consists of a single (IM) injection of long-acting oxytetracycline at a dosage of 20 mg/kg b.w.

The carrier state may be eliminated by administration of the same drug at the similar dose at least two injections with a 1 week interval). Withholding periods for tetracyclines apply in most countries. Injection into the neck muscle rather than the rump is preferred.

Imidocarb is also highly efficacious against *A. marginale* as a single injection (as the dihydrochloride salt at 1.5 mg/kg, b.w.(SC), or as imidocarb dipropionate at 3 mg/kg b.w. Elimination of the carrier state requires the use of higher repeated doses of imidocarb (eg. 5 mg/kg, b.w. (IM or SC), two injections of the dihydrochloride salt 2 weeks apart). imidocarb is a suspected carcinogen with long withholding periods and is not approved for use.

Control:

In South Africa, Australia, Israel, and South America, infection with live *A. centrale* (originating from South Africa) is used as a vaccine to provide cattle with partial protection against the disease caused by *A. marginale*. *A. centrale* (single dose) vaccine produces severe reactions in a small proportion of cattle.

Immunity generated by using multidose killed vaccine protects cattle from severe disease on subsequent infection, but cattle can still be susceptible to challenge with heterorogous strains of *A. marginale*.

2.4.6 Heart water

Definition and causative agent:

Heartwater is an infectious, noncontagious, tickborne rickettsial disease of ruminants. The disease is seen only in areas infested by ticks of the genus *Amblyomma*. Cattle, sheep, goats, and some antelope species are susceptible to Heartwater. In endemic areas, indigenous African cattle breeds (*Bos indicus*), especially those with years of natural selection, appear more resistant to clinical Heartwater than exotic breeds (*B. taurus*).

The causative organism was previously known as *Cowdria ruminantium*, but through molecular evidence has now been classified as *Ehrlichia ruminantium*.

Transmission:

Under natural conditions, *E. ruminantium* is transmitted by *Amblyomma* ticks. These three-host ticks become infected during either the larval or nymphal stages and transmit the infection during one of the subsequent stages (transstadial transmission).

Clinical signs:

The clinical signs are dramatic in the peracute and acute forms. In peracute cases, animals may drop dead within a few hours of developing a fever, sometimes without any apparent clinical signs; others display an exaggerated respiratory distress and/or paroxysmal convulsions.

In the acute form, animals often show anorexia and depression along with congested and friable mucous membranes. Respiratory distress slowly develops along with nervous signs such as a hyperaesthesia, a high-stepping stiff gait, exaggerated blinking, and chewing movements. Terminally, prostration with bouts of opisthotonus; "pedaling," "thrashing," or stiffening of the limbs; and convulsions are seen. Diarrhea is seen occasionally.

In subacute cases, the signs are less marked and CNS involvement is inconsistent.

Lesions:

Lesions are associated with functional injury to the vascular endothelium, resulting in increased vascular permeability without recognizable histopathologic or even ultrastructural pathology. The concomitant fluid effusion into tissues and body cavities precipitates a fall in arterial pressure and general circulatory failure. The lesions in peracute and acute cases are hydrothorax, hydropericardium, edema and congestion of the lungs and brain, splenomegaly, petechiae and ecchymoses on mucosal and serosal surfaces, and occasionally hemorrhage into the GI tract, particularly the abomasum.

Diagnosis:

Demonstration of colonies of organisms in the cytoplasm of capillary endothelial cells is necessary for a definitive diagnosis. Traditionally, this is done with "squash" smears of cerebral or cerebellar gray matter stained with Romanowsky-type stains.

For the "brain squash smear," a piece of gray matter (~3 × 3 mm) is macerated between two microscope slides; the softened material is then spread like a blood smear with the material pushed rather than pulled along. A slight lifting of the spreader slide about every 5–10 mm creates several thick ridges across the slide, from which capillaries are arranged straight and parallel in the thin sections of the smear for easier examination.

The endothelial cells of all the capillaries on a smear should be carefully scrutinized for presence of the dark purple colonies made up of clusters of individual organisms (granules) of *E. ruminantium*.

Using immunoperoxidase staining methods, a definitive diagnosis can be made on any formalin-fixed tissue samples, even from autolyzed carcasses. The contrasting color makes the search for and identification of the rickettsial colonies much quicker, although the substructure of the colonies should be identified before the diagnosis is confirmed.

DNA probes, available at research institutions, can be used together with PCR technology. A combination of a pCS20 probe and probes to 16S ribosomal RNA of several of the stocks are used routinely to examine samples from animals when permits for movement of animals from endemic to nonendemic areas are required. Real-time PCR has also come into use.

Differential diagnosis:

In clinical cases, Heartwater must be differentiated from a wide range of infectious such as Babesiosis, Theileriosis, Listeriosis, Tetanus, Rabies and noninfectious diseases, especially plant poisonings that manifest with CNS signs.

Treatment and control:

Treatment:

Oxytetracycline at 10 mg/kg b.w. /day, (IM), or doxycycline at 2 mg/kg b.w/day (IM) will usually effect a cure if administered early in the course of Heartwater infection. A higher dosage of oxytetracycline (20 mg/kg/B.W) (IM) is usually required if treatment begins late during the febrile reaction or when clinical signs are evident. In such cases, the first treatment should preferably be given slowly by IV route. A minimum of three daily doses should be given regardless of temperature; if fever persists, oxytetracycline treatment should continue for a fourth and fifth day. If the fever still does not abate, a potentiated sulfonamide at 15 mg/kg/day, b.w. (IM), has been successful.

Withdrawal times for milk and meat after treatment with doxycycline, short- or long-acting oxytetracycline, and sulfonamides must be observed based on local regulations.

Corticosteroids have been used as supportive therapy (prednisolone 1 mg/kg, IM), although there is debate as to the effectiveness and rationale for their use.

Diazepam may be required to control convulsions.
Affected animals must be kept quiet in a cool area with soft bedding and be totally undisturbed; any stimulation can preempt a convulsive episode and subsequent death.

Control:

The "infection and treatment method" for immunization is in use in South Africa, where infected sheep blood containing fully virulent organisms of the Ball 3 stock is used for infection, followed by monitoring of rectal temperature and antibiotic therapy after fever develops.Control of tick infestation is a useful preventive measure in some instances but may be difficult and expensive to maintain in others.

2.4.7 Parasitic Diseases of small ruminants

Definition and causative agent:

Parasitic diseases of small ruminants impair health, reproduction, growth, and productivity. In severe cases, parasitic diseases may even cause death. These diseases are caused by internal helminths (nematodes [roundworms], tapeworms and flukes) as well as external arthropods (mites, lice, ticks, and flies).

Transmission:

Transmission of helminths is through oral ingestion or direct skin penetration by larval parasites on pasture. Transmission of arthropods is through direct contact (mites and lice) or exposure to larval stages on pasture (ticks) or fly-in by adult flies or skin penetration by larval flies (grubs).

2.4.7.1 Gastrointestinal nematodes

The epidemiology of GI nematode infections is influenced by climatic factors (particularly rainfall and temperature), management systems used for the animals, host factors and parasite factors. Clinical signs caused *Heamonchus*, *Trichostrongyle*, *Bunostomum* and *Dictyocaulus* are described:

Clinical signs:

General symptoms of worm infections are rough hair coat, diarrhea, emaciation, weight loss, and/or blood loss.

The major parasites associated with parasitic gastro-enteritis in small ruminants are *H. contortus*, *T. colubriformis* and *Oesophagostomum columbianum*. Due to the differences in their predilection sites and pathogenetic mechanisms, GI nematodes present with differing clinical and pathological features.

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

Acute haemonchosis signs include weakness, pallor of the mucous membrane, lethargy or agalactia which may lead to starvation and death of kids. Dark colored faeces are often observed. Chronic haemonchosis is the common form of field infection and it is a chronic gastritis with chronic blood loss and abomasal dysfunction leading to weakness, progressive weight loss, rough hair coat and stunted growth.

Trichostrongylosis is commonly a disease of young animals an acute enteritis is characterized by dark-coloured diarrhoea and foul smelling faeces. There may be sudden death without evidence of anaemia or emaciation but weakness of the legs is a frequent feature.

Most commonly, trichostrongylosis is a chronic wasting disease characterized by loss of appetite, emaciation, loss of weight, dry skin, diarrhoea, oedema and atrophy of skeletal muscles or mycocardium. There is mucoid diarrhoea or sometimes constipation, emaciation, general weakness, dry skin, prostration and death. The diarrhoea often coincides with the emergence of larvae from the nodules.

Bunostomosis is characterised by progressive anaemia, emaciation, weakness or paresis, submandibular oedema, dark coloured faeces, prostration and death. Parasitic bronchitis (verminous pneumonia) caused by *Dictyocaulus filaria* and *Muellerius capillaris* is more common in kids or lambs under 6 months than in other age groups. Adult *D. filaria* cause alveolar and bronchiolar irritation leading to coughing, dyspnoea and loss of body condition. Secondary bacterial infection may lead to toxaemia.

Lesions:

Hyperacute haemonchosis has limited gross pathological features due to sudden death, although a large number of immature or young adults may be found in the abomasal mucosa. However, hyperacute haemonchosis is not very common in field infections.

Acute haemonchosis is characterised by a pale and watery carcass. The abomasal mucosa is petechiated and oedematous with many parasites on its surface and contents.

At necropsy, the acute trichostrongylosis is characterised by a swollen and haemorrhagic or catarrhal intestinal mucosa and, worms may be found in the mucosal scrapings. The chronic disease is characterised by an emaciated carcass, fatty degeneration and, a thickened, inflamed and ulcerated intestinal mucosa. At post mortem examination in bunostomosis there is hydrothorax, hydropericardium and pin-point haemorrhages in the small intestine or blood in its content.

Necropsy in trichostrongylosis is related to the migration of larvae in muscularis mucosa of the large intestine resulting in a fibroblastic response around the larvae forming fibrous nodules At necropsy, in *D. filaria* there is pulmonary oedema and emphysema with consolidation of some parts of the lung. The bronchioles are filled and may be blocked with exudate. Bronchoectasis frequently accompanies secondary bacterial infections.

Diagnosis:

Internal roundworm parasites are diagnosed by fecal egg or larval counts from live animals. Post mortem, worms can be counted in the abomasum, intestine, lungs and liver. External parasites are diagnosed by visual observation, hair loss, or skin scrapings.

Roundworm fecal egg counts are only reliable for approximately 4 months after exposure to a parasite species. As an animal is continuously exposed to a worm species, its worm counts and fecal egg counts rise for about 3-4 months. In most herds, there are three groups of animals:

- A group (10-15%) of animals which never get high worm or egg counts.
- A group (60-70%) of animals that get moderate worm numbers and egg counts.
- A group (10-15%) of animals that get high worm numbers and high egg counts.

After 4 months, the egg counts drop in the second group, but their worm numbers don't decline for another 4-6 months. In the third group, egg counts don't decline for an additional 4-6 months, and the worm counts decline another 4 months later.

Treatment and control:

Treatment:

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

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

Control:

The control of helminthosis should be designed to eliminate or reduce the prevalence of helminths and improve the productivity of the livestock industry. The eradication of helminthosis in animals is difficult and the aim of control is therefore to limit the infection by minimising the challenge to an economically justifiable level.

Best practices include sheep and goats being treated at the beginning of a roundworm/fly or lice infection period with a long acting product to eliminate existing roundworm or arthropod parasites and prevent reinfection if the animals are in an environment where there is infection challenge.

In southern Tanzania, a single anthelmintic treatment at the end of the dry season has been found to minimize worm burdens during the rainy season while in the northern part of the country treatment at the beginning and end of the dry season has been recommended.

Other studies conducted in Tanzania, have shown that treatment of goats two weeks after the onset of rains and at a four-weekly interval during the rainy season minimizes pasture contamination with helminth eggs thereby reducing the number of infective larvae on pastures during the dry season.

In small-holder farming systems where worm burdens are generally low, treatment of clinical cases may be more practical, economical and acceptable by farmers than mass treatment. Rotational grazing, separation of animals according to age groups, alternate grazing by different species of hosts, adjustment of stocking rates, improvement of nutrition and better housing systems are the common management manoeuvres employed in the control of helminthosis in most countries.

Alternate grazing by different host species depends on the degree of cross-transmission of parasites between host species. Although this practice may reduce the overall burden of the species in question, the reduction may not be sufficient for efficient parasite control.

2.4.7.2 Fasciolosis

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

Transmission:

Eggs are passed in the feces, and miracidia develop within as little as 9–10 days (at 22°–26°C. Hatching only occurs in water, and miracidia are short-lived (~3 hours). Miracidia infect lymnaeid snails, in which asexual development and multiplication occur through the stages of sporocysts, rediae, daughter rediae, and cercariae. After 6–7 weeks, cercariae emerge from snails, encyst on aquatic vegetation, and become metacercariae. Metacercariae may remain viable for many months unless they become desiccated.

After ingestion by the host, usually with herbage, young flukes excyst in the duodenum, penetrate the intestinal wall, and enter the peritoneal cavity, where they migrate to the liver.

Clinical signs:

Acute fascioliasis occurs when there is an acute traumatic hepatitis caused by the migration of larvae through the parenchyma leading to extensive destruction and marked haemorrhage. The haemolytic crisis results in progressive weakness, pallor of the mucous membranes, enlargement of the liver and abomasal distension. Anorexia, paresis prior to death and anasarca are observed in terminal stages of the acute disease in goats.

Subacute fascioliasis is associated with ingestion of a large number of metacercariae over a long period of time. The syndrome is characterized by anorexia, rough hair coat, slight abdominal distension, pallor of mucous membranes, disinclination to move and emaciation. Chronic fascioliasis is a persistent wasting disease characterized by emaciation, anaemia and submandibular oedema.

Lesions:

At necropsy, acute fascioliasis is characterized by the presence of a blood-tinged fluid in the peritoneal cavity, fibrinous exudate covering the liver surface, hepatomegaly and numerous haemorrhagic and friable tracts in the liver parenchyma. Subacute infection is accompanied by fibrosis and thickening of the bile ducts resulting from cholangitis is evident.

Diagnosis:

Fasciolosis diagnosis is by demonstration of oval, operculated, golden brown eggs (130–150 × 65–90 µm) which must be distinguished from those of paramphistomes (rumen flukes), which are larger and clear. Eggs of *F. hepatica* cannot be demonstrated in feces during acute fasciolosis. Diagnosis can be aided. At necropsy, the nature of the liver damage is diagnostic. Adult flukes are readily seen in the bile ducts, and immature stages may be squeezed or teased from the cut surface.

Treatment and control:

Treatment:

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

Albendazole and oxfendazole are also effective against *Fasciola* spp. Niclosamide (90 mg/kg), brotianide (15 mg/kg) and closantel have been shown to be effective in treating paramphistomosis.

Control:

Control measures for *F. hepatica* ideally should involve removal of flukes in affected animals, reduction of the intermediate host snail population, and prevention of livestock access to snail-infested pasture.

2.4.7.3 Cestode infections

The pathogenic effects of *Moniezia* spp. are limited and the parasite is considered to be non-pathogenic. However, heavy infections in young animals may cause anorexia, weight loss, and moderate anaemia, inflammation of the intestinal mucosa and sometimes obstruction of the intestines.

Migration of *Coenurus cerebralis* in the brain may cause meningo-encephalitis while massive numbers of cysts of *Echinococcus granulosus* in the lungs may cause respiratory problems. Other cestodes have limited clinical significance in small ruminants.

Transmission:

The intermediate host for *Moniezia*, is a mite which lives in damp grass. The mite ingests the eggs and then is accidentally swallowed by the calf, kid or lamb. Since these young animals will start licking grass at an early age, they can be infected very early in life. It is possible for a 6 week old calf to be infected by an adult tapeworm and already passing eggs. The passed segments can be seen in the dung looking like grains of rice.

Clinical signs:

The symptoms of infection are potbelly, dullness, poor growth and diarrhoea.

Treatment and control:

Treatment:

Niclosamide (80 mg/kg), resorantel (75 mg/kg), praziquantel (15 mg/kg), bunamidine (25-50 mg/kg), cambendazole (25-35mg/kg), albendazole (5

mg/kg), mebendazole (10 mg/kg) and fenbendazole (10 mg/kg) have been in use against cestodes. Benzimidazoles are particularly effective against anoplocephalid tapeworms.

Control:

Prevention is difficult, but young animals should be kept away from areas where the mite lives, on wet grass. Calf houses should be regularly cleared and disinfected.

2.4.8 Common Cattle Parasites

Common important internal parasites of cattle are hairworms, lungworms, liver flukes and coccidia. Common external parasites include horn flies, lice and grubs.

2.4.8.1 Internal parasites

Hairworms

The GI tract of cattle is often infected with hairworms, also called stomach worms and intestinal worms.

Transmission:

These worms are transmitted when infected cattle pass eggs in manure onto the ground eggs hatch in the manure; rain washes the larvae from the manure; and cattle swallow larvae on wet grass in moderate temperatures.

Normally the disease (wormy cattle) is secondary to inadequate nutrition. Poor nutritional management practices such as over crowdedness and overgrazing create inadequate nutrition and allow cattle to be re-infected continuously. The primary malnutrition condition, a protein deficiency, worsens because the larvae interfere with digestion.

Clinical signs:

Calves have low immunity and usually infested during their exposures. Heavy exposures cause disease while light exposures produce immunity. Adult cattle and young cattle have immunity from previous exposures, but often become infested.

Signs of cattle infested with worms include pale mucous membranes, bottle jaw, potbelly, diarrhea, drawed, not grazing, not chewing cud, rough and dry hair coat, thinness, weakness and inability to stand. These signs are similar to those caused by malnutrition and liver flukes.

Diagnosis:

Examine feces each month to check fluctuations of worm eggs per gram of feces, which will help you time the drug administration properly and monitor the effectiveness of your control measures.

Treatment and control:

Treatment:

Use strategic de-worming whereby feacal egg count is established and parasites present is determined to better select the de-wormer.

Control:

The most important way to control hairworms is to maintain good nutrition by rotating pastures, preventing overcrowding and overgrazing and providing good quality pasture, hay and supplements. When cattle have a diet with enough protein, vitamins and minerals, fewer worms are normally established and the cattle are more able to withstand their effects.

Additional control measures include proper drainage and sanitation, separating age groups.

Lungworms

Lungworms cause a lung disease in cattle with clinical signs similar to those caused by viruses, bacteria and allergies. Lungworm disease occurs in previously unexposed cattle, such as in calves or moved cattle.

Transmission:

Transmission is similar as for hairworms.

Treatment and control:

De-wormers are administered to cattle not only as a treatment to kill internal parasites and to stop damage caused by parasites, but also to prevent pasture contamination and reinfection of the cattle. For lungworms and other GI worms,

administer albendazole as an oral suspension at a dose rate of 10 mg/kg b.w. in cattle and in goats at dose of 7.5 mg/kg b.w.

Drugs to control internal parasites should supplement but not replace management practices to improve sanitation and nutrition.

Liver flukes

Cattle living in wet areas with alkaline soils may develop liver fluke infections. Liver flukes are transmitted when infected cattle, deer and rabbits pass eggs in manure and drop the manure in water; eggs hatch in water and larvae develop in snails; and cattle swallow cysts on grass or hay.

Clinical signs:

Signs of digestive inefficiency are evident in young cattle with acute liver disease and in older cattle with chronic liver disease. Fluky cattle show signs similar to those with malnutrition and hairworms.

Diagnosis:

Examine feces each month to check fluctuations of worm eggs per gram of feces, which will help you, time the drug administration properly and monitor the effectiveness of your control measures.

Treatment and control:

Strategic deworming.

For liver flukes in cattle use nitroxynil at a dose of 12.5-13mg/kg (SC) and for goats a dose of 10-15 mg/kg (SC) and for parafilaria use a dose rate of 20 mg/kg (SC). Drugs to control internal parasites should supplement but not replace management practices to improve sanitation and nutrition.

Coccidia

Coccidia cause an intestinal disease of young cattle, usually 3 weeks to 6 months old, but can affect cattle up to 2 years old.

Transmission:

They are transmitted when infected cattle pass cysts in manure onto the ground; rain washes the cysts from the manure; the cysts develop under moist and moderate temperature conditions; and cattle swallow cysts on moist ground.

Clinical signs:

As with hairworms and lung worms, transmission is common during rainy times The diarrhea caused by coccidia may be confused with the diarrhea caused by hairworms, bacteria and viruses.

Diagnosis:

Diagnosis of coccidiosis is by finding oocysts on feacal floatation or direct smear of by the McMaster technique.

Treatment and control:

Effective drugs are amprolium at a dose of 10mg/kg b.w. for 5 days

2.4.8.2 External parasites

Horn flies

Thousands of flies may infest a single animal, causing extreme nervousness and energy loss. Horn flies suck blood, irritate and annoy, reduce weight gains and cause weight losses. The annoyance and irritation interfere with cattle's feeding and resting.

Transmission:

Horn flies reproduce in fresh cattle manure during the rainy season. Hot, dry conditions may naturally reduce horn fly numbers.

Treatment and control:

Treatment is economically justified when horn fly populations reach 250 per head. To control them satisfactorily throughout the season, use self-treatment insecticides or routinely apply spray, pour-on, spot-on or dust chemicals.
Used properly, self-treatment devices are more effective than hand application in controlling horn flies and lice.

Pyrethroid ear tags (permethrin, fenvalerate) have induced widespread horn fly resistance. Vary the types of ear tag insecticides rather than using the same kind year after year. Remove tags as soon as possible once they have lost their effectiveness in killing horn flies. Tags used 4 to 5 months emit too little insecticide to control fly populations adequately. Tags emitting reduced doses seem to add to the resistance problem by prolonging fly exposure, thus making the surviving population more resistant to the insecticide.

Lice

Lice cause a condition called lousy, an itching skin disease with possible anemia.

Transmission:

Blood-sucking lice are transmitted between cattle by contact.

Clinical signs:

Include dry, scaly skin, hair loss and itching exhibited by biting, rubbing and scratching. Lice bites and allergies to lice cause the itching. The allergic dermatitis may persist after the lice are gone. These signs may be confused with malnutrition and allergies caused by horn flies, mosquitoes and gnats.

Treatment and control:

Although chemicals do not harm lice eggs, cattle can be treated effectively by administering an anti-parasitic pour-on containing ivermectin on animals twice at a 2-week interval or once with ivermectin containing 1% (10 mg/ml) (SCor IM) at a dose of 1 ml/50 kg b.w. Injections are effective against suckling lice, but have low efficacy against biting lice.

2.4.9 Ormilo (Coenurosis)

Definition and causative agent:

It is neurological disease of sheep and goats known locally as *'ormilo'* as the leading animal disease concern amongst Maasai pastoralist livestock-keepers in northern Tanzania. The condition is ultimately fatal, leading to substantial losses for pastoralist households who are highly dependent on livestock for household income and food security.

The study, which was carried out in four villages in Arusha region, identified *Taenia multiceps*, a tapeworm parasite carried by domestic dogs, as a major cause of '*ormilo*' with the parasite detected in >80% of cases reported by farmers.

Transmission:

Sheep and goats become infected after consuming food or water that is contaminated with tapeworm eggs. Following infection, larval stages of the parasite form large cysts in the brain (cerebral coenurosis) leading to severe untreatable disease that is known as 'gid' in the UK.

Clinical signs:

The neurologic signs range from changes in awareness to walking in circles, unsteadiness and collapse, ultimately into death.

Lesions:

Appearance of one or more cysts of larva stages of the dog tapeworm in brain of sheep or goat.

Diagnosis

Cysts located in cerebral hemisphere (80-90%) and in cerebellum (5-10%).

Treatment and Control:

Treatment is based on surgical removal of the cyst after general anesthesia of the animal.
The best control is to prevent dogs from having access to sheep and goat carcass and not to feed them uncooked meat.

2.5 Metabolic Conditions

2.5.1 Milk fever

Definition and causative agent:

Milk fever, also known as parturient hypocalcaemia and parturient paresis, is a disease, which has assumed considerable importance with the development of heavy milking cows. Decrease in the levels of ionized calcium in tissue

fluids is the cause of the disease. In all adult cows, there is a fall in serum-calcium level with the onset of lactation at calving. The disease usually occurs in 5 to 10-year-old cows and is chiefly caused by a sudden decrease in blood-calcium level, generally within 48 hours after calving.

Clinical signs:

In classical cases, hypocalcaemia is the cause of clinical symptoms. Hypophosphataemia and variations in the concentration of serum-magnesium may play some subsidiary role. The clinical symptoms develop usually in one to three days after calving. They are characterized by loss of appetite, rumen atony, downer cow, cold extremities and lethargy but there is no rise in temperature.

Control and Treatments:

Treatment:

Treat with 500 ml of calcium borogluconate (IV) followed by oral administration of two oral calcium boluses 12 hrs apart.

Control:

Effective nutritional management during the dry period and early lactation decrease clinical cases.

SECTION 3

GUIDELINES FOR POULTRY DISEASES

3.1 Preamble

Poultry plays a key role in the livelihood of millions of poor rural communities associated with poultry industry in Tanzania and the World over. Food and Agriculture Organization of the United Nations (FAO) has estimated that with increased urbanization, demand in consumption of chicken meat, eggs and animal protein will increase and therefore the need to promote poultry production. Poultry eggs are ranked second after cow milk in terms of nutritive value.

Poultry are susceptible to several types of infectious and/or non-infectious diseases. The need to provide a guidebook on poultry diseases, diagnostic techniques and their effective treatment to avoid the production losses facing the Tanzanian poultry industry is timely. These Standard Treatment Guidelines will provide useful information of diagnostic techniques of diseases at their initial stages which can reduce the disease risk and improve the immunity status of birds by using disease specific vaccination.

Biosecurity measures

The fact that prevention of diseases will likely yield better results goes without saying. Biosecurity is among measures of disease control. Biosecurity measures involve bio containment which includes quarantine and other measures designated to keep the virus on the infected farm or area; and exclusion biosecurity which keeps the virus out of the disease free farm or area.

Exclusion biosecurity includes isolation which is keeping the poultry protected from source of infection including unauthorized access and carriers of disease and separating group of animals to minimize spread of infection across the population.

Rigorous disinfection of contaminated farms after depopulation may help to decontaminate the premises but has achieved limited success.

3.2 Poultry Viral Diseases

3.2.1 Newcastle Disease

Definition and causative agents:

Newcastle disease (ND) is a highly contagious and often severe disease found worldwide that affects birds including domestic poultry. It usually presents as a respiratory disease, but depression, nervous manifestations, or diarrhoea may be the predominant clinical form.

It is caused by an RNA avian paramyxovirus serotype 1 (PMV-1) which can be categorized into three groups: The original classification of Newcastle disease virus (NDV) include the virulent (velogenic), moderately virulent (mesogenic), or of low virulence (lentogenic) strains. Velogens and mesogens are now classified as virulent NDV (vNDV), the cause of Newcastle disease and reportable infection, whereas infections with lentogens, the low virulence NDV (loNDV) widely used as live vaccines, are not reportable.

Transmission:

Chickens are readily infected by aerosols and by ingesting contaminated water or food. Infected chickens and other domestic and wild birds may be sources of NDV. Movement of infected birds and transfer of virus, especially in infective feces, by the movement of people and contaminated equipment or litter are the main methods of virus spread between poultry flocks. Spread is slower if the fecal-oral route is the primary means of transmission, particularly for caged birds.

Clinical signs:

Signs appear simultaneously throughout the flock (2-15 days) after exposure. Young and naïve chickens are more susceptible and show signs sooner than older one. Respiratory or nervous signs or both are the most common clinical signs.

Respiratory signs like gasping, coughing, sneezing, and rales predominate in infections with loNDV. Nervous signs of tremors, paralyzed wings and legs, twisted necks, circling, clonic spasms, and complete paralysis may accompany, but usually follow the respiratory signs in neurotropic velogenic disease.

Respiratory signs with depression, watery greenish diarrhea, and swelling of the tissues of the head and neck are typical of the most virulent form of the disease termed as viscerotropic velogenic ND. Varying degrees of depression and in appetence are seen. Partial or complete cessation of egg production may occur.

Eggs may be abnormal in color, shape, or surface and have watery albumen. Mortality is variable but can be as high as 100% with vNDV infections.

Lesions:

Remarkable gross lesions are usually seen only with viscerotropic velogenic ND. Petechiae may be seen on the serous membranes; hemorrhages of the proventricular mucosa and intestinal serosa. In contrast, lesions in birds infected with loNDV strains may be limited to congestion and mucoid exudates seen in the respiratory tract with opacity and thickening of the air sacs.

Diagnosis:

Clinical signs and lesions are suggestive of the disease.

Rapid tests (such as lateral flow immunochromatographic assay) can be used for virus identification and hence, differentiation. NDV can be isolated from oropharyngeal or cloacal swabs or tissues from infected birds by inoculation of the allantois cavity of 9- to 11-day-old specific pathogen free (SPF) embryonated chicken eggs. To confirm diagnosis, identification of an isolate such as vNDV is established by the rapidity of killing day-old SPF chicks inoculated by the intracerebral route, the intracerebral pathogenicity index, or by the presence of a specified amino acid motif at the cleavage site of the fusion protein (F) precursor (FO).

Infection is confirmed by recovery of a hemagglutinating virus that is inhibited with NDV antiserum or by detection of NDV RNA by RT-PCR.

Differential diagnosis

ND should be differentiated from Fowl cholera, Highly pathogenic avian influenza, Infectious Laryngotracheitis, Fowl pox (diphtheritic form) and Infectious bronchitis

Treatment and control:

Treatment:
No treatment is available.

Control:

Vaccines are available for chickens, turkeys, and pigeons and are used to induce an antibody response. Healthy chicks are vaccinated as early as day 1–4 of life. However, delaying vaccination until the second or third week avoids maternal antibody interference with an active immune response. Mass vaccination methods must ensure that >85% of the flock is immunized, which is needed for herd immunity.

Unfortunately, ND vaccines do not provide sterile immunity, and in many areas of the World, vaccines are used to prevent losses from sickness and death.

I-2 thermostable ND vaccine is available in Tanzania and it is administered as an eye drop at an interval of 4 months and is effective in control of NCD.

Live lentogenic vaccines, chiefly B1 and LaSota strains, are widely used and typically administered to poultry by mass application in drinking water or by spray. For drinking water vaccination do not open and mix the vaccine until ready to vaccinate. Remove all medication, saniters and disinfectants from drinking water 72 hours before vaccination. Withhold water for 2 to 4 hours prior to vaccination to stimulate thirsty.

At age of 2-4 weeks mix 1000 dose vaccine into 10 litres drinking water, at age at 4-8 weeks in 20 litres and over 8 weeks of age in 40 litres of water; and then distribute vaccine solution among the waterers. Do not provide any drinking water until the solution has been consumed.

Oil-adjuvanted inactivated vaccines are also used after live vaccine in breeders and layers and may be used alone in situations where use of live virus may be contraindicated (eg, in pigeons). Administering inactivated vaccines is more labor intensive, because each bird has to be handled individually.

The frequency of revaccination to protect chickens throughout life largely depends on the risk of exposure and virulence of the field virus challenge. In Tanzania, it is recommended to vaccinate every three months after the initial vaccination.

Fowl pox or turkey herpesvirus–vectored ND vaccines are commercially available for chickens and have the advantage of being able to be administered i*n ovo* at the hatchery. These vaccines must be reconstituted as directed by the manufacturer, and they take 3–4 week to produce a protective level of immunity.

Biosecurity measures

Biosecurity measures should be employed as described in the preamble to control the disease.

3.2.2 Avian Encephalomyelitis

Definition and causative agents:

Avian Encephalomyelitis (AE) a viral disease of the CNS of young chickens, turkeys, Japanese quail, pheasants, and pigeons caused by an RNA virus, *Tremovirus A* in the family Picornaviridae.

Transmission:

Infection occurs via vertical and horizontal transmission. If a breeder flock becomes infected during egg production, the virus is vertically transmitted to the offspring, and a major outbreak occurs. Natural field strains of the virus are enterotropic and multiply in the intestine. Infected birds shed the virus in their feces for a few days to a few weeks, which serves to spread the infection to hatch mates through faecal-oral route. There is no convincing evidence that the virus persists in infected birds. AE virus is resistant to environmental conditions and may remain infectious for long periods.

Clinical Signs:

Vertically infected chicks commonly show clinical signs of avian encephalomyelitis during the first week after hatching, although signs may be present in a few birds at hatching. Clinical signs appear later in hatch mates that are horizontally infected by the fecal-oral route. Vertical infection followed by horizontal infection causes a characteristic biphasic mortality pattern.

The main clinical signs are ataxia and leg weakness that varies from sitting on hocks to paresis that progresses to paralysis and recumbence. Fine tremors of the head and neck are evident in some birds and are characteristic of the disease. Cupping the bird in one's hands often results in a buzzing feeling because of rapid, fine tremors. Severely affected birds lay on their side and exhibit intermittent fine tremors of the head, neck, and legs.

Horizontally infected chicks usually show clinical signs at 2–4 weeks of age. Morbidity and mortality rates vary and depend on the level of egg transmission and degree of immunity in the flock. In severe outbreaks, both morbidity and mortality may exceed 50%.

After 4 weeks of age, chickens are resistant to disease but not to infection. In laying chickens, there is a sudden 5%–10% drop in egg production, which usually lasts for <2 weeks, followed by a return to normal production. There is no deterioration in egg shell quality.

Lesions:

No gross lesions are seen in the brain of birds infected with avian encephalomyelitis. Gray to white foci may be visible on cut surfaces of the muscle of the gizzard. Weeks after infection, opacity of eye lenses (cataracts) may occur in a small percentage of chickens that survive the infection.

Diagnosis:

Tissues collected for virus isolation must include the brain and duodenum with the pancreas. Demonstration of AE virus antigen in the brain, spinal cord, and other tissues by immunofluorescent and immunohistochemically staining is a reliable method of diagnosis.

Differential diagnosis:

The major differential diagnosis for neurologic signs in very young chicks is bacterial or mycotic encephalitis. Rickets and nutritional encephalomalacia are next in the list of differential diagnoses, although the clinical manifestations of these diseases differ from those in AE.

Treatment and control:

Treatment:

No treatment is available.

Control:

Vaccination at least 4 weeks before start of laying by administering the vaccine through wing web route using a double needle applicator. Insert the double needle into the vaccine bottle after preparation for use for wetting before piercing the wing web.

Immunization of broiler breeder pullets with a commercial chick-embryo-propagated live vaccine prevents vertical transmission of the AE virus and provides progeny with maternal immunity. AE vaccine is usually combined with fowl pox vaccine and given to chickens by wing-web stab. Also available is a live fowl pox-vectored infectious laryngotracheitis and AE combination vaccine. Chicks and turkey poults with neurologic signs are ordinarily euthanized because they rarely recover.

Biosecurity measures:

Rigorous disinfection of contaminated farms after depopulation may help to decontaminate the premises but has achieved limited success.

3.2.3 Infectious Bursal Disease (Gumboro disease)

Definition and causative agent:

Infectious Bursal Disease (IBD) is seen in young domestic chickens worldwide and is caused by a birnavirus (IBD virus-IBDV) that is most readily isolated from the bursa of Fabricius, but may be isolated from other organs.

Transmission:

It is shed in the feces and transferred from house to house by fomites and by a faecal-oral route. It is very stable and difficult to eradicate from premises.

Clinical signs:

IBD is highly contagious. Results of infection depend on age and breed of chicken and virulence of the virus. Infections may be subclinical or clinical. Infections before 3 weeks of age are usually subclinical. Chickens are most susceptible to clinical disease at 3–6 weeks of age when immature B cells populate the bursa and maternal immunity has waned, but severe infections have occurred in Leghorn chickens up to 18 weeks of age.

Early subclinical infections are the most important form of the disease because of economic losses. They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen.

Some strains of IBDV can cause subclinical infections in older birds (3–6 weeks old), which leads to losses from poor feed efficiency and longer times to market. In clinical infections, onset of the disease occurs after an incubation of 3–4 days. Chickens may exhibit severe prostration, incoordination, watery diarrhea, soiled vent feathers, vent picking, and inflammation of the cloaca. Flock morbidity is typically 100%, and mortality can range from 5% to greater than 60% depending on the strain of virus and breed of chicken.

Lesions:

At necropsy, the lesions seen will depend on the strain of IBDV. For strains that cause a clinical disease, the cloacal bursa is swollen, edematous, yellowish, and occasionally hemorrhagic, especially in birds that died of the disease.

Strains of IBDV cause similar cloacal bursa lesions, and congestion and hemorrhage of the pectoral and leg muscles can also occur. Chickens that have recovered from IBDV infections have small, atrophied, bursas of Fabricious due to the destruction and lack of regeneration of the bursal follicles.

Diagnosis:

Initial diagnosis of IBD is accomplished by the observation of gross lesions in the bursas of Fabricious, followed by microscopic analysis of the bursa for lymphocyte depletion in the follicles.

The RT-PCR assay is used to identify the viral genome in bursa tissue.
Serology can be used to detect the presence of antibodies to IBDV in convalescent chicks. Commercially available ELISA kits are most often used to quantitate IBDV antibodies. The presence of IBDV antibodies in chicks is not always an indication of infection because most young chicks have maternal antibodies.

Differential diagnosis:

Other diseases that need to be differentiated from hyper-virulent IBD on clinical or pathological grounds include: Marek's disease, Mycotoxicosis, Coccidiosis, Haemorrhagic syndrome, Avian adenovirus infection and Infectious bronchitis.

Treatment and Control:

Treatment:

No treatment is available.

Control:

Live vaccines of chicken embryo or cell-culture origin and of varying low pathogenicity can be administered by eye drop, drinking water, or SC routes at 1–21 days of age.

Replication of these vaccines and thus the immune response can be altered by maternal antibody, although the more virulent vaccine strains can override higher levels of maternal antibody. Vectored vaccines that express the IBDV VP2 protein in herpesvirus of turkeys (HVT) can be used *in ovo* or at hatch. These HVT-IBD vaccines are not affected by maternal antibodies.

Vaccines that use live-attenuated viruses bound to antibodies (immune-complex vaccines) are also available for *in ovo* or at hatch administration. Breeder flocks should be vaccinated one or more times during the growing period, first with a live vaccine and again just before egg production with an oil-adjuvanted, inactivated vaccine.

The goal of any vaccination program for IBD should be to use vaccines that most closely match the antigenic profile of the field viruses.

Biosecurity measures:

Rigorous disinfection of contaminated farms after depopulation may help to decontaminate the premises but has achieved limited success.

3.2.4 Marek's disease

Definition and causative agent:

Marek's disease is a highly contagious viral disease of poultry characterized by T-cell lymphomas and peripheral nerve enlargement caused by an alphaherpes virus member of the genus *Mardivirus* within the subfamily Alphaherpesvirinae.

Transmission

The disease is readily transmitted among chickens. The virus matures into a fully infective, enveloped form in the epithelium of the feather follicle, from which it is released into the environment. Infected chickens continue to be carriers for long periods and act as sources of infectious virus. The virus is mainly transmitted through an aerogenous route.

Clinical signs:

More common in younger birds that are usually under the age of 20 weeks. Paralysis is sometimes noted, but more typically affected birds show only depression before death. A transient paralysis syndrome has been associated with the disease. Chickens become ataxic for several days, and then recover. The syndrome is rare in immunized birds.

Lesions:

Enlarged nerves are one of the most consistent gross lesions in affected birds and has a diagnostic significance. Various peripheral nerves, particularly the vagus, brachial, and sciatic, become enlarged and lose their striations. Diffuse or nodular lymphoid tumors may be seen in various organs, particularly the liver, spleen, gonads, heart, lung, kidney, muscle, and proventriculus. Enlarged feather follicles (commonly termed skin leukosis) may be noted in broilers after defeathering during processing and are a cause for condemnation. The bursa is only rarely tumorous and more frequently is atrophic.

Diagnosis:

Standard criteria: history and clinical signs, gross pathology, and histopathology. Advanced criteria: immunohistochemistry, standard and quantitative PCR, virus isolation, and serology

Differential diagnosis:

Lymphoid leucosis.

Treatment and control:

Treatment:
There is no effective treatment for Marek's disease.

Control:

Prevention methods include vaccination, biosecurity and adherence to strict zoosanitary measures. Vaccination is the central strategy for the prevention and control of Marek disease, along with strict sanitation.

The most widely used vaccines include:

- Turkey herpesvirus (HVT, naturally avirulent *Meleagrid alphaherpesvirus* 1) (SC 0.2ml/dose)
- SB-1 or 301B/1 (naturally avirulent *Gallid alphaherpesvirus* 3) (SC 0.2ml/dose).
- CVI988/Rispens (attenuated *Gallid alphaherpesvirus* 2) (SC 0.2ml/dose).

Vaccines are administered at hatch or *in ovo* to embryos at the 18th day of incubation. *In ovo* vaccination is now performed by an automated technology.

Biosecurity measures:

Rigorous disinfection of contaminated farms after depopulation may help to decontaminate the premises but has achieved limited success.

3.2.5 Infectious Bronchitis (IB)

Definition and causative agent:

It is an acute, highly contagious upper respiratory tract disease in chickens caused by an avian gamma coronavirus IB virus.

Transmission:

Virus is shed by infected chickens in respiratory discharges and feces, and it can be spread by aerosols, ingestion of contaminated feed and water, and contact with contaminated equipment and clothing. Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks after infection. The incubation period is generally 24–48 hours, with the peak in excretion of virus from the respiratory tract lasting 3–5 days after infection.

Clinical signs:

Morbidity for flocks affected by IB is typically 100%. Chicks may cough, sneeze, and have tracheal rales for 10–14 days. Conjunctivitis and dyspnea may be seen, and sometimes facial swelling, particularly with concurrent bacterial infection of the sinuses.

Chicks may appear depressed and huddle under heat lamps. Feed consumption and weight gain are reduced. Infection with nephropathogenic strains can cause initial respiratory signs, then later depression, ruffled feathers, wet droppings, greater water intake, and death.

In layers, egg production may drop by as much as 70%, and eggs are often misshapen, with thin, soft, wrinkled, rough, and/or pale shells, and can be smaller and have watery albumen. Egg production and egg quality can return to normal, but this may take up to 8 weeks.

In most outbreaks, mortality is approximately 5%, although mortality rates can be as high as 60% when disease is complicated by concurrent bacterial infection or when nephropathogenic strains induce interstitial nephritis in chicks.

Lesions:

In the respiratory tract, the trachea, sinuses, and nasal passages may contain serous, catarrhal, or caseous exudates, and the air sacs a foamy exudate initially, progressing to cloudy thickening. If complicated by infection with *E coli*, there may be caseous airsacculitis, perihepatitis, and pericarditis.

Birds infected when very young may have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and regression of the ovaries. Infection with nephropathogenic strains results in swollen, pale kidneys, with the tubules and ureters distended with urates. In birds with urolithiasis, the ureters may be distended with urates and contain uroliths, and the kidneys may be atrophied.

Diagnosis:

Laboratory confirmation is required for diagnosis of respiratory forms of IB because of similarities to mild forms of disease caused by agents such as NDV, avian metapneumovirus, infectious laryngotracheitis virus, mycoplasmas, *Avibacterium paragallinarum*, and *Ornithobacterium rhinotracheale.*

Definitive diagnosis is generally based on virus detection and identification. Diagnosis is commonly achieved using ELISA, hemagluttination inhibition test, RT-PCR assays to detect viral RNA in nucleic acid extracts of tracheal, cecal, tonsil, or kidney tissue.

Differential diagnosis:

ND, ILT and Infectious Coryza.

Treatment and control:

Treatment:

No medication alters the course of IBV infection, although antimicrobial therapy may reduce mortalities caused by complicating bacterial infections.

Control:

The ability of the virus to quickly mutate requires constant surveillance to identify IBV types circulating in a specific region. Attenuated live and killed vaccines are used to control the disease. Live-attenuated vaccines are administered by coarse spray, aerosol or drinking water depending on the vaccine which is presented 10 ml vial containing (500-10,000 doses). These vaccines are initially given to 1- to 14-day-old chicks. Inactivated vaccines are intended for use in layers and breeders and are administered (SC) at 13-18 weeks of age to pullets previously primed with attenuated live vaccines.

Biosecurity measures:

Rigorous disinfection of contaminated farms after depopulation may help to decontaminate the premises but has achieved limited success.

3.2.6 Infectious Laryngotracheitis (ILT)

Definition and causative agent:

An economically important acute respiratory disease of poultry, highly contagious, caused by Gallid alpha herpesvirus 1 (GAHV-1) commonly known as infectious laryngotracheitis virus (ILTV).

Transmission:

The virus can be easily transmitted by infected birds and fomites. Lax biosecurity, transportation of infected birds, and spread of contaminated litter facilitates spread of the virus. After recovery, birds remain carriers for life and become a source of infection for susceptible birds. The latent virus can be reactivated under stressful conditions. Infection may also be spread mechanically.

Clinical signs:

In the acute form of ILT, gasping, coughing bloody mucoid exudate, rattling, and extension of the neck during inspiration are seen 5–12 days after natural exposure. Reduced productivity is a varying factor in laying flocks. Affected birds are anorectic and inactive. Mortality varies but may reach 50% in adults and is usually due to occlusion of the trachea by hemorrhage or exudate. Signs usually subside after ~2 weeks, although some birds may show signs for longer periods. Strains of low virulence produce little or no mortality, with mild respiratory signs and a slight decrease in egg production.

In a subacute disease there is nasal and ocular discharge, tracheitis, conjunctivitis, and mild rales.

Lesions:

The acute form of ILT is characterized by the presence of blood, mucus, yellow caseous exudates, or a hollow caseous cast in the trachea. Microscopically, it is characterized by a desquamative, necrotizing tracheitis and conjunctivitis. The mild forms of the disease are characterized by discrete hemorrhagic areas in the upper trachea and larynx and mild conjunctivitis.

Diagnosis:

Rapid diagnosis of the disease can be achieved by histopathologic examination and the detection of lesions that are pathognomonic of the infection, such as syncytial formation and intranuclear inclusion bodies in the trachea and conjunctiva mucosal epithelium.

Diagnosis can be rapidly confirmed by detection of viral DNA using virus-specific PCR assays.

Differential diagnosis:

IB, ND, mycoplasmosis, and Avian coryza.

Treatment and control:

Treatment:
There is no effective treatment.

Control:

In endemic areas and on farms where a specific diagnosis is made, ILTV is controlled by implementation of biosecurity measures and vaccination. Vaccination is done with live attenuated vaccines and viral vector recombinant vaccines. These are applied via eye drop or through mass vaccination by water or spray.

Viral vector recombinant vaccines in Fowlpox and Herpesvirus of turkeys have been designed to express ILTV immunogenic proteins and are administered to individual birds by *in ovo*, subcutaneous, or wing-web vaccination.

3.2.7 Fowlpox

Definition and causative agent:

A worldwide viral infection of chickens and turkeys. The large DNA virus (an avipoxvirus in the Poxviridae family) that is resistant and may survive in the environment for extended periods in dried scabs cause the disease.

Transmission:

The virus is present in large numbers in the lesions and is usually transmitted by contact through abrasions of the skin. Skin lesions (scabs) shed from recovering birds in poultry houses can become a source of aerosol infection. Mosquitoes and other biting insects may serve as mechanical vectors. Transmission within a susceptible flock is rapid when mosquitoes are plentiful. The disease tends to persist for extended periods in multiple-age poultry complexes because of slow spread.

Clinical signs:

Infected chicken are usually depressed and anorexic. Nasal discharge may be seen due to localization around the nostrils, and may be accompanied with dyspnea. Complete closure of one or both eyes also occur due to cutaneous lesions on the eyelids or both eyes. Extensive infection in a layer flock results in decreased egg production. Cutaneous infections alone ordinarily cause low or moderate mortality and these flocks generally return to normal production after recovery. Mortality is usually high in diphtheritic or systemic infections.

Lesions:

The cutaneous form of fowl pox is characterized by nodular lesions on various parts of the un-feathered skin of chickens and on the head and upper neck of turkeys. Generalized lesions of feathered skin may also be seen. In some cases, lesions are limited chiefly to the feet and legs. Localization around the nostrils may cause nasal discharge. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes. Only a few birds develop cutaneous lesions at one time.

In the diphtheritic form of fowl pox, lesions develop on the mucous membranes of the mouth, esophagus, pharynx, larynx, and trachea (wet pox or fowl diphtheria). Occasionally, lesions are seen almost exclusively in one or more of these sites. In cases of systemic infection caused by virulent fowl pox virus strains, lesions may be seen in internal organs. More than one form of the disease, ie, cutaneous, diphtheritic, and/or systemic, may be seen in a single bird.

Diagnosis:

Microscopic examination of affected tissues stained with Haematoxylin and Eosin (H&E) reveals eosinophilic cytoplasmic inclusion bodies. The most commonly used method in diagnostic laboratories is by observing characteristic gross and microscopic lesions and PCR. PCR can be used to amplify genomic DNA sequences of various sizes using specific primers. This procedure is useful when an extremely small amount of viral DNA is present in the sample.

Differential diagnosis:

Symptoms of the cutaneous form are easy to recognize. For the diphteric form, differential diagnosis may include vitamin A deficiency and respiratory diseases such as ILT.

Treatment and control:

Treatment:
There is no effective treatment.

Control:

The most widely used vaccines are attenuated fowl pox virus and pigeon pox virus isolates of high immunogenicity and low pathogenicity. Where fowl pox is prevalent, chickens and turkeys should be vaccinated with a live-embryo or cell-culture-propagated virus vaccine. In high-risk areas, vaccination with an attenuated vaccine of cell-culture origin in the first few weeks of life and revaccination at 12–16 weeks is often sufficient.

Because the infection spreads slowly, vaccination is often useful to limit spread in affected flocks if administered when <20% of the birds have lesions. Vaccinated birds should be examined 1 week later for swelling and scab formation ("take") at the site of vaccination. Absence of "take" indicates lack of potency of vaccine, passive or acquired immunity, or improper vaccination. Revaccination with another serial lot of vaccine may be indicated.

3.2.8 Lymphoid Leucosis

Definition and causative agent:

Lymphoid leucosis occurs naturally only in chickens and is characterized by B-cell lymphoma, occurring in chickens approximately 16 weeks of age and older. It is caused by certain group of avian retroviruses. Is a clonal malignancy of the bursal-dependent lymphoid system.

Transmission:

Avian leucosis virus is shed by the hen into the albumen or yolk, or both. Infection occurs after onset of incubation. Congenitally infected chickens remain viremic for life. Horizontal infection after hatching is also important when chicks are exposed immediately following hatching to high doses of the virus.

Clinical signs:

Chickens with lymphoid leukosis have few typical clinical signs. These may include in appetence, weakness, diarrhea, dehydration, and emaciation.

Infected chickens become depressed before death. Palpation often reveals an enlarged bursa and sometimes an enlarged liver and infected chickens lay fewer eggs.

Lesions:

Diffuse or nodular lymphoid tumors on liver surface, spleen and bursa and occasionally on the kidney. Involvement of the bursa is pathognomonic.

Diagnosis:

Tumorous involvement of the liver, spleen or bursa in absence of peripheral nerve lesion suggest the disease. The tumors occurs in ≥14 weeks old chicken. Detection of the major antigen present in the core of leukosis/ sarcoma viruses forms the basis of several diagnostic tests.

Differential diagnosis:

Marek's disease should be considered.

Treatment and control:

Treatment:
There is no treatment or vaccine available, so eradication of the virus from breeding flocks through culling of infected chickens is the most effective control method.

Control:

The virus is readily inactivated by disinfectants and transmission can be reduced or eliminated by strict sanitation.

3.2.9 Avian influenza (AI)

Definition and causative agent:

Avian influenza (AI) caused by a type A orthomyxoviruses (*Influenzavirus A*) is a viral infection of domestic poultry, pets, zoo and wild birds. In domestic poultry, AI viruses are typically of low pathogenicity (LP), causing subclinical infections, respiratory disease, or drops in egg production, but a few AI viruses are highly pathogenic (HP), causing severe systemic disease with multiple organ failure and high mortality.

Transmission:

Transmission between individual birds is by ingestion or inhalation of contaminated substances. Spread between farms is the result of breaches in biosecurity practices, principally by movement of infected poultry or contaminated feces and respiratory secretions on fomites such as equipment or clothing. Airborne dissemination between farms may be important over limited distances. The incubation period is highly variable and ranges from a few days in individual birds to 2 weeks in the flock.

Clinical signs:

Low Pathogenicity Avian Influenza (LPAI) viruses typically produce respiratory signs such as sneezing, coughing, ocular and nasal discharge, and swollen infraorbital sinuses in poultry. In layers, there is decrease egg production and infertility. In severely affected birds, greenish diarrhea is common.

Highly Pathogenicity Avian Influenza (HPAI) viruses cause severe, systemic disease with high mortality in chickens, turkeys, and other gallinaceous poultry. Mortality can be as high as 100% in a few days. In per acute cases, clinical signs or gross lesions may be lacking before death. Greenish diarrhoea is also common. Birds that survive the per acute infection may develop CNS signs such as torticollis, opisthotonos, incoordination, paralysis, and drooping wings.

Lesions:

Lesions associated with LPAI include congestion and inflammation in the trachea and lungs. Free egg yolk in the body (egg yolk break brown and mix in the abdomen) especially in H9 case all the flock should be buried to avoid spreading of the disease. Petechial hemorrhages on visceral organs and in muscles; and blood-tinged oral and nasal discharges. There is also visceral urate deposition.

In HPAI there is hemorrhage of the skin on the feet and shanks. Petechial hemorrhages on visceral organs and in muscles; and blood-tinged oral and nasal discharges. However, in acute cases, lesions may include cyanosis and edema of the head, comb, wattle, and snood (turkey); edema and red discoloration of the shanks and feet due to subcutaneous ecchymotic hemorrhages.

Diagnosis:

Diagnosis is made by AI virus isolation; detection of AI viral RNA (PCR) or by detection of AI-specific antibodies (ELISA).

Differential diagnosis:

LPAI must be differentiated from other respiratory diseases or causes of decreased egg production, including:

- Acute to subacute viral diseases such as IB, ILT, low virulent ND, and infections by other paramyxoviruses.
- Bacterial diseases such as mycoplasmosis, Infectious Coryza, and the respiratory form of Fowl Cholera.
- Fungal diseases such as aspergillosis.

HPAI must be differentiated from other causes of high mortality such as virulent ND, the per acute septicemic form of Fowl Cholera, heat exhaustion, and severe water deprivation.

Treatment and control:

Treatment:
There is no treatment.

Control:

There is no vaccine and the chickens infected will always be carriers.

Biosecurity measures:

You need to sanitize all areas where the birds were residing, before introducing a new flock. Practice of exclusion biosecurity strategies to prevent introduction of AI into poultry is the best preventive measure. Suspected outbreaks should be reported to appropriate regulatory authorities.

3.3 Poultry Bacteria Diseases

3.3.1 Infectious Coryza

Definition and causative agent:

It is an acute respiratory disease of chickens characterized by nasal discharge, sneezing, and swelling of the face under the eyes caused by a bacterium *A. paragallinarum*, a gram-negative, pleomorphic, and non-motile, catalase-negative, microaerophilic rods.

Transmission:

Chronically ill or healthy carrier birds are the reservoir of infection. Chickens of all ages are susceptible, but susceptibility increases with age. Transmission is by direct contact, airborne droplets and by contamination of drinking water. The incubation period is 1–3 days, and the disease duration is usually 2–3 weeks.

Clinical signs:

In the mildest form of the disease, the only signs may be depression, a serous nasal discharge, and occasionally slight facial swelling.

In the more severe form, there is severe swelling of one or both infraorbital sinuses with edema of the surrounding tissue, which may close one or both eyes. In adult birds, especially males, the edema may extend to the intermandibular space and wattles. The swelling usually abates in 10–14 days. There may be varying degrees of rales depending on the extent of infection. Egg production may be delayed in young pullets and severely reduced in producing hens. Birds may have diarrhea, and feed and water consumption usually is decreased during acute stages of the disease.

Lesions:

In acute cases, lesions may be limited to the infraorbital sinuses. There is a copious, tenacious, grayish, semifluid exudate. As the disease becomes chronic or other pathogens become involved, the sinus exudate may become consolidated and turn yellowish. Other lesions may include conjunctivitis, tracheitis, bronchitis, and airsacculitis, particularly if other pathogens are involved.

Diagnosis:

A smear of nasal exudates or sinus should be made and Gram stained and should reveal Gram-negative bipolar staining rods. Isolation of a Gram-negative, satellitic, catalase-negative organism from chickens in a flock with a history of a rapidly spreading coryza is diagnostic. The catalase test is essential, because nonpathogenic hemophilic organisms, which are catalase-positive, are present in both healthy and diseased chickens. A PCR test that can be used on the live chicken and that has proved superior to culture, has been developed. A real-time version of the PCR is also available.

Differential diagnosis:

Swelling of the face and wattles must be differentiated from that seen in Fowl Cholera. Other diseases that must be considered are mycoplasmosis, ILT, ND, IB, AI, swollen head syndrome (Ornithobacterosis), and vitamin A deficiency.

Treatment and control:

Treatment:

Sulfathiazole at a dose of 0.5% in feed for 5 days or 1-1.5 gm/liter in drinking water for 5-7 days is a drug of choice in alleviating the severity of Infectious Coryza.

Sulfadiazine-trimethoprim 1 gm/liter for 5 days. Never use sulfa drugs in chickens older than 14 weeks for commercial layer hens.

Second generation quinolone derivatives such as Enrofloxacin 10 mg/kg b.w. for 3-5 days, Norfloxacin 12 mg/kg b.w. for 3-5 days or Danofloxacin 5 mg/kg b.w. for 3 days in water may be used.

Control:

Vaccination (coryza vaccine) is applied in endemic areas SC or IM, at a dose of ½ ml per injection between 10-12 weeks of age. Two injections are given at an interval of 3-4 weeks before 20 weeks. Immunity period is 9 months.

Biosecurity measures:

Prevention is the only sound method of control. "All-in/all-out" farm programs with sound management and isolation methods are the best way to avoid Infectious Coryza. Replacements should be obtained from clean flocks. If there is an outbreak, incinerate the dead birds, clean and disinfect houses.

3.4.2 Fowl Cholera

Definition and causative agent:

A serious, highly contagious disease caused by the bacterium *Pasteurella multocida* in a range of avian species including chickens, turkeys, and water fowls, (increasing order of susceptibility). The disease can range from acute septicaemia to chronic and localized infections and the morbidity and mortality may be up to 100%.

Transmission:

The route of infection is oral or nasal with transmission via nasal exudate, faeces, contaminated soil, equipment, and people. The incubation period is usually 5-8 days.

Clinical signs:

In acute disease, there is high mortality without any sign. Clinical signs include depression, ruffled feathers, loss of appetite, diarrhea, increased respiratory rate, coughing and nasal, ocular and oral discharge. In chronic form, there is localized infection which include swollen sternal bursa, wattles, joints, tendon sheaths and foot pads; which lead to lameness. Sudden death may also occur. There is also pharyngitis and conjunctivitis.

Lesions:

In per acute and acute forms, there is hyperemia and congestion throughout the carcass which is accompanied by enlargement of the liver and spleen. Petechial and ecchymotic hemorrhages on the subepicardial and serosal locations may also be seen. There is also increased pericardial and peritoneal fluids.

In subacute cases, there is multiple small necrotic foci disseminated throughout the liver and spleen. In chronic form, there is fibrinonecrotic dermatitis that includes caudal parts of the dorsum, abdomen and breast and involve cutis, sub cutis and underlying muscles. There are also sequestered necrotic lung lesions.

Diagnosis:

History, clinical signs and gross lesions may help in diagnosis. High mortality without any sign may be the first sign of the disease. Impression smears, isolation (aerobic culture on trypticase soy or blood agar yields colonies up to 3 mm in 24 hours - no growth on MacConkey), confirmed with biochemical tests. Immunofluorescent microscopy can be used to detect *P. multocida* in tissues and exudates. PCR may also be used successfully to detect carrier birds.

Differential diagnosis:

Consider salmonellosis, colibaccillosis, and listeriosis.

Treatment and control:

Treatment:
Treatment is by sulphonamides, tetracycline, erythromycin, streptomycin and penicillin. The disease often recurs after medication is stopped, necessitating long-term or periodic medication. Antibiotics may reduce mortality but won't eliminate *P. multocida* from a flock.

When antibiotics are used, early treatment and adequate dosages are important. Sensitivity testing often aids in drug selection and is important because of the emergence of multi-resistant strains. Sulfamethazine, sulfamerazine, sulfaquinoxaline or sulfadimethoxine in water at a dose of (0.04 %) solution for 2 – 3 days. If recurs, repeat. Sulfas should be used with caution in breeders because of potential toxicity and cannot be used in hens laying eggs for human consumption. Withdraw period in chickens for slaughter is 14 days. High levels of tetracycline antibiotics in the feed (0.04 %), drinking water, or administered parenterally may be useful.

Control:

Vaccines are available but give variable results.

Biosecurity measures:

The disease is best controlled by eradication and cleaning and disinfection of buildings and equipment. The premise should then be kept free of poultry for a few weeks. Biosecurity, good rodent control, hygiene, and separation of birds by age with thorough cleanout between flocks.

3.4.3 Fowl Typhoid

Definition and causative agent:

Fowl typhoid may be acute or chronic and is caused by *Salmonella gallinarum*; and produces lesions in chicks and poults similar to those produced by *S. pullorum*.

Transmission:

It is egg-transmitted (vertical transmission) or by faecal-oral contamination by egg eating (horizontal transmission).

Clinical signs:

Fowl typhoid may be acute or chronic. Clinical signs and lesions in young birds are similar to those seen with *S. enterica* Pullorum infection. Clinical signs in chicks and poults include anorexia, diarrhoea, dehydration weakness and high mortality. Older birds may be pale, dehydrated, and have yellowish diarrhea. Mortality in young birds is similar to that seen in S. *pullorum* infection but may be higher in older birds.

Lesions:

In older birds may include a swollen, friable, and often bile-stained liver, with or without necrotic foci, an enlarged spleen and kidneys, anemia and enteritis.

Diagnosis:

Tentative diagnosis can be made based on flock history, clinical signs, mortality and lesion. Positive serology can also be of great value in detecting infection. Confirmation is by isolation, identification, and serotyping of *S. gallinarum*

Differential diagnosis:

Consideration should be given to Pullorum diseases, Fowl Cholera and Erysipelas.

Treatment and Control

Treatment:
Treatment is never recommended as no drug has been successful in eliminating the disease.

Control:

Vaccines (killed or modified live) made from a rough strain of *S. gallinarum* (9R) had variable results in controlling mortality. More recently, vaccines derived from outer membrane proteins, mutant strains, and a virulence-plasmid-cured derivative of *S. enterica* Gallinarum have shown promise in protecting birds against challenge. Vaccines for Salmonella are not capable of eradicating infection from flocks but can increase the threshold for infection, reduce the level of shedding of the organism and reduce vertical transmission in poultry that results in contamination of hatching or table eggs. Vaccination is therefore an aid to other eradication and control measures such as culling, all-in-all out production, biosecurity and farm hygiene.

Biosecurity measures:

Chicks and poults should be obtained from disease-free sources. Disease-free poults should not be mixed with diseased ones. Sound biosecurity program should be in place to minimize introduction of the disease from outside sources. Rodents, flying birds and insects should be limited into poultry houses. Another method is detection and elimination of carriers.

3.4.4 Colibacillosis

Definition and causative agent:

It is one of the most commonly occurring and economically devastating bacterial diseases of poultry worldwide caused by pathogenic strain of *Escherichia coli*. *E. coli* is a Gram-negative, rod-shaped bacterium normally found in the intestine of poultry and other vertebrates.

Transmission:

Usually the main transmission route is fecal-oral route.

Clinical signs:

Signs vary and can include acute fatal septicemia, airsacculitis, pericarditis, perihepatitis, and lymphocytic depletion of the bursa and thymus. It manifests in diverse ways, including as acute fatal septicemia, subacute pericarditis, airsacculitis, salpingitis, peritonitis, and cellulitis.

Lesion:

Young birds dying of acute septicemia have few lesions except for an enlarged, hyperemic liver and spleen with increased fluid in body cavities.

Birds that survive septicemia develop subacute fibrinopurulent airsacculitis, pericarditis, perihepatitis, and lymphocytic depletion of the bursa and thymus.

Airsacculitis is a classic lesion of colibacillosis, Sporadic lesions include pneumonia, arthritis, osteomyelitis, peritonitis, and salpingitis.

Diagnosis:

Is made by isolation of a pure culture of *E. coli*.

Differential diagnoses:

It includes Chronic Respiratory Disease, Fowl Cholera and Pullorum disease.

Treatment and Control:

Treatment:
Most isolates are resistant to tetracycline, streptomycin, and sulfa drugs; and due to widespread resistance, antibiotics are not recommended.

Fluoroquinolone use is now banned. Widespread resistance to disinfectants, including certain heavy metal compounds, further complicates control of colibacillosis.

Drug sensitivity testing is necessary before institution of any treatment.

Control:

Prevention of exposure through good management is recommended.

Biosecurity measures:

Proper biosecurity and biosafety practices should be instituted.

3.4.5 Pullorum Disease/Bacillary white diarrhoea

Definition and Causative agent:

Pullorum disease is a septicemic disease, primarily of chickens and turkeys, caused by Gram-negative bacteria, *Salmonella pullorum*.

Transmission:

The disease is primarily egg transmitted, but transmission may occur by other means such as fecal-oral route. It can be contracted through contaminated surfaces and other birds that have become carriers of the disease.

Clinical Signs:

The effect of the disease on chicks and older birds are different. The chicks huddle near source of heat, do not eat, appear sleepy and have a white paste all over their backsides. Some will die with no signs at all. Survivors, frequently become asymptomatic carriers with localized infection of the ovary. In older birds, however, you will see sneezing and coughing besides poor laying. Infection transmitted via hatchery or egg usually results in mortality during first few days and up to 2-3 weeks of age. White thick and sticky droppings adhering to feathers around vent i.e. copious white diarrhoea.

Lesions:

In chicks

- Acute: Septicemic lesions: ompholitis with persistent yolk sac (contain creamy or caseated materials).
- Hepatomegally, friable and hemorrhagic liver with necrotic foci.
- Catarrhal enteritis with white diarrhoea.
- Peritonitis, pericarditis, pneumonia, splenitis, splenomegally, synovitis of hock joints.
- The lungs may be congested.
- The caeca are inflamed, enlarged and distended with hard, dry, necrotic material.
- Chronic: Pale yellow nodules in myocardium, intestines and gizzard.

In adults

- Characteristic lesion is an abnormal ovary. The ova are irregular, cystic, deformed and pedunculated (attached) with prominent thickened stalks.
- Oophoritis (hemorrhagic/atrophied), salpingitis, peritonitis, ascitis.
- In some infected adult hens the ovary is inactive, the ova being small, pale and undeveloped.
- Enlarged liver with bronzy color (hemosiderosis) with necrotic foci (as in typhoid).
- Enteritis (ulcerative duodenitis), intestinal or caecal inflammation.
- Caeca are enlarged & distended with casts of hard, dry necrotic material.
- Grayish necrotic foci on the lungs, liver, intestines, heart, gizzard and spleen.
- Urate crystals in ureters.

Diagnosis:

Tissue and faecal samples can be submitted for bacteria identification through culture or genetic techniques.

Serological tests are satisfactory for establishing the presence and estimating the prevalence of infection within a flock.

Differential diagnosis:

Consideration should be given to Fowl typhoid, Fowl Cholera and Erysipelas.

Prevention and Treatment:

Treatment of Pullorum disease is not feasible. Recovered birds have a tendency to become carriers of the bacteria. It is best to depopulate a flock that tests positive for *S. pullorum* There is no vaccine for this disease.

3.4.6 Chronic Respiratory Disease (CRD)

Definition and causative agent:

This infection is caused by *Mycoplasma gallisepticum* when they are stressed and is associated with slow onset, chronic respiratory disease in chickens and other birds.

Transmission:

M. gallisepticum spreads via eggs, airborne transmission and indirect or mechanical routes such as introducing infected birds to an existing flock, or via bird transport containers. *M. gallisepticum* can reside in a flock with few indications of its presence until the flock or individuals in it are stressed sufficiently to show signs of respiratory disease.

Clinical signs:

In chickens, uncomplicated infections may be in apparent. Affected birds may have varying degrees of respiratory distress, coughing, difficulty breathing and sneezing. Morbidly is high and mortality is low.

Lesions:

Uncomplicated infections in chickens result in mild sinusitis, tracheitis and airsacculitis (cloudy air sac).

Diagnosis:

Lesions, serology, isolation and identification of organism, demonstration of specific DNA (commercial PCR kit available).

Culture requires inoculation in mycoplasma-free embryos or, more commonly in *Mycoplasma* Broth followed by plating out on Mycoplasma Agar. Suspect colonies may be identified by immuno-fluorescence.

Serology: serum agglutination is the standard screening test, suspect reactions are examined further by heat inactivation and/or dilution. ELISA is accepted as the primary screening test

Differential diagnosis:
Differentiate from Infectious Coryza, Aspergillosis, viral respiratory diseases, vitamin A deficiency, other *Mycoplasma* infections such as *M. synoviae* and *M. meleagridis* (turkeys).

Treatment and control:

Treatment:

Sensitive to antibiotics such as chlortetracycline, oxtetracyline and enrofloxacin.
Bio-tilmiconsin is very effective to CRD in poultry. Dose: 1 ml/12.5 kg b.w. or 0.3 ml/liter of water, continuously in 3 days. If suspect CRD combined with *E. coli*, use special medication Bio-tylodoxplus, strongly effective with CRD combined with vitamin E. Other treatment include, Tylosin, spiramycin, tetracycline, and fluoroquinolones.

Effort should be made to reduce dust and secondary infections.
Purchase of uninfected chicks, all-in/all-out production,

Control:

Live attenuated or naturally mild strains are used in some countries and may be helpful in gradually displacing field strains on multi-age sites.

Biosecurity measures:

Management issues must be addressed before the birds arrive. Ensure birds are free of *M. Gallisepticum* on introduction. Thorough cleaning down between batches helps. Housing that are difficult to clean and thus accumulate manure, dust and vermin should be avoided.

3.4.7 *Salmonella enteritidis* and *typhimurium* infection

Definition and causative agent:

It is a zoonotic disease of poultry caused by caused by Gram-negative bacteria - salmonella (*S. enteritidis* and *S. typhimurium*).

Transmission:

Vertical transmission through shell contamination or internal transovarial contamination of yolk. The disease is also transmitted through a fecal-oral route through feces, fomites or egg shells.

Clinical sign:

Symptoms include lethargy, diarrhea and conjunctivitis. Other signs include growth retardation, twisted necks and lameness.

Mortality rates in different flocks range from 1.7% to 10%.

Lesions:

Lesions include panophthalmitis, hepatomegaly with necrotic foci, enlarged spleen and kidney, pericarditis, coagulated and unabsorbed hyperemic yolk, abnormal ovum and purulent arthritis and enteritis.

Diagnosis:

Isolation and identification. In clinical cases direct plating on Brilliant Green and McConkey agar may be adequate. Enrichment media such as buffered peptone followed by selective broth or semi-solid media (e.g. Rappaport-Vassiliadis) followed by plating on two selective media will greatly increase sensitivity. However, this has the potential to reveal the presence of *salmonellae* that are irrelevant to the clinical problem under investigation. It is possible to detect reactions with specific antigens in agglutination tests but competitive and direct Elisa tests are more commonly used today.

Differential Diagnosis:

Differentiate from Pullorum disease, Fowl Typhoid, and Colibacillosis.

Treatment and control:

Treatment

Gentamycin: Dosage: 0.01 mg/kg b.w. IM once daily; Or 25 mg in 120 ml of drinking water. Trimethoprim/Sulfamethoxazole suspension: Dosage 0.002 ml/kg b.w. orally twice daily. Limitations associated with use of sulfa drugs should be considered.

Control

Vaccines are increasingly being used for *S. enteritidis* and *S. typhimurium* infection; both inactivated (bacterins) and attenuated live organisms.

Biosecurity measures:

Measures to clean nests, fumigate eggs, all-in/all-out production, good feed, competitive exclusion and uninfected breeders must be ensured.

3.5 Poultry Parasitic Diseases

3.5.1 Coccidiosis

Definition and Causative agent:

The disease is characterized by enteritis, diarrhoea and mortality. The bird develops reduced ability to absorb nutrients, which results in weight loss and eventually death. Sub clinically, it is manifested by poor performance, impaired feed conversion, poor flock uniformity and poor growth.

Transmission:

Infection is via the fecal-oral route. Disease occurs after ingestion of a relatively large number of sporulated oocysts by susceptible birds. Oocysts may be transmitted by mechanical carriers (equipment, insects, clothing, farm workers and other animals)

Clinical signs:

Signs range from decreased growth rate or weight gain to a high percentage of visibly sick birds which show mild loss of appetite, ruffled plumage, severe diarrhea, which may be bloody and may lead to dehydration, anemia, and high mortality rates. Mild infections of intestinal species, which would otherwise be classified as subclinical, may cause depigmentation and potentially lead to secondary infection, particularly *Clostridium* spp. infection. Survivors of severe infections recover in 10–14 days but may never recover lost performance.

Lesions:

E. tenella infections are found only in the ceca and can be recognized by accumulation of blood in the ceca and by bloody droppings.

E. necatrix produces major lesions in the anterior and middle portions of the small intestine. Small white spots, usually intermingled with rounded, bright- or dull-red spots of various sizes, can be seen on the serosal surface. This appearance is sometimes described as "salt and pepper." In severe cases, the intestinal wall is thickened, and the infected area dilated to 2–2.5 times the normal diameter. The lumen may be filled with blood, mucus, and fluid.

*E. acervulina*is associated with numerous whitish, oval or transverse patches in the upper half of the small intestine, which may be easily distinguished on gross examination.

E. brunetti is found in the lower small intestine, rectum, ceca, and cloaca. In moderate infections, the mucosa is pale and disrupted but lacking in discrete foci, and may be thickened. In severe infections, coagulative necrosis and sloughing of the mucosa occurs throughout most of the small intestine.

E. maxima develops in the small intestine, where it causes dilatation and thickening of the wall; petechial hemorrhage; and a reddish, orange, or pink viscous mucous exudate and fluid.

E. mitis is recognized as pathogenic in the lower small intestine. Lesions are indistinct but may resemble moderate infections of *E. brunetti*. *E. mitis* can be distinguished from *E. brunetti* by finding small, round oocysts associated with the lesion.

Diagnosis:

History, clinical signs and gross lesions may help in diagnosis. Coccidial infections are readily confirmed by demonstration of oocysts in feces or intestinal scrapings. However, the number of oocysts present has little relationship to the extent of clinical disease. Classic lesions of *E tenella* and *E necatrix* are pathognomonic. Mixed coccidial infections are common.

The white spots are diagnostic for *E necatrix* if clumps of large schizonts can be demonstrated microscopically.

Differential Diagnosis:

Differentiation from non-coccidial diseases is crucial, and common differential diagnoses include:

Chickens:

Differentiate from blackhead (caecum), *salmonella* (caecum), necrotic enteritis (*Clostridium perfringens*) (small intestine/ileum), capillarisis (small intestine) salt poisoning (small intestine), mycotoxicoses (small intestine) and cannibalism (blood in feces).

Turkeys:

Differentiate from *salmonella*, blackhead (caecum), necrotic enteritis (*Clostridium perfringes*) and hemorrhagic enteritis.

Treatment and control:

Treatment

Most anticoccidials currently used in poultry production are coccidiocidal and include:
Amprolium with a composition of 300 mg/g is given per 1.25 to 2.5 litres in drinking water for 5-7 days for curative purposes. For prevention, administer 1 g/5 litres in drinking water for 1-2 weeks.

Clopidol and **quinolones** (eg, pyridinols at a dose of 125 ppm) and (enrofloxacin at a dose of 5-20 mg/kg per oral).

Diclazuril and **toltrazuril** are highly effective against a broad spectrum of coccidia. Diclazuril is used mostly for prevention at 1 ppm in the feed, whereas toltrazuril is used primarily for treatment in the water (2.5% oral liquid) at a dose of 5 ml/5 litre of drinking water (25 ppm) for continuous medication over 48 hours or 15 ml/5 litre drinking water (75 ppm) for 8 hours per day for 2 consecutive days.

Control

Control moisture by management of watering systems. If possible periodically move location of your chickens.

Commercial vaccines consist of live, sporulated oocysts of the various coccidial species administered at low doses. The self-limiting nature of coccidiosis is used as a form of attenuation for some vaccines, rather than biologic attenuation.

Modern anticoccidial vaccines are given to day-old chicks, either at the hatchery or on the farm. Because the vaccine serves only to introduce infection, chickens are re-infected by progeny of the vaccine strain on the farm.

Anticoccidials are commonly withdrawn from broilers 3–7 days before slaughter to meet regulatory requirements and to reduce production costs.

There is now a solution, a benchmark product that is competitively priced which functions not only as a replacement for coccidiostats and antibiotic growth promoters but which can also provide many other benefits. Orego-Stim is a natural feed additive used in poultry production worldwide. It has been extensively researched and tested, and is able to increase the performance of poultry production by improving the feed conversion ratio (FCR) as well as increasing the body weight gain of broilers. It also helps to reduce mortality caused by GI diseases by preventing the occurrences of GI pathogen invasion. Its active components effectively kill these microorganisms, which include both Gram-positive and Gram-negative bacteria upon contact within the gut of the animals.

Biosecurity measures:

Good biosecurity measures should be adhered to.

3.5.2 Gapeworm Infection

Definition and causative agent:

Also known as syngamiasis, red worm, or forked worm, is characterized by gaping for air caused by *Syngamus trachea*, a parasitic nematode of the superfamily Strongyloidea, found in the trachea of domestic and wild birds worldwide where they feed on blood. They are tiny, bright red (caused by ingestion of the host's blood), worms that have a 'y'-shaped appearance (which are actually two worms, the male and female that are joined together, with the male acting as an anchor for the female).

Transmission:

Female *S. trachea* lay eggs in the bird's trachea, which hatch and are either coughed up or swallowed by the bird, later defecated out into the environment.

Chickens can also become infected indirectly, by eating earthworms, snails or slugs that are infected. Regardless of how, once chickens ingest the larvae, they will migrate through the GI system until they reach the trachea, where they reproduce, lay eggs, feed on blood, and live.

Clinical signs:

The parasite causes laboured breathing, and 'gaping': affected birds stretch out their necks, open their mouths and gasp for air producing a hissing noise as they do so. Severe infestation may obstruct the tracheal lumen resulting in suffocation.

Other clinical signs include: coughing, weakness, emaciation and shaking of the head. Adult birds are usually less affected and may only show an occasional cough or even no obvious clinical signs.

Lesions:

This results in the development of lymphoid nodules, catarrhal tracheitis and occasional secondary lobar pneumonia. If enough worms are present, they can cause partial to complete obstruction of the trachea.

Diagnosis:

It is usually made on the basis of clinical signs. On post mortem: small nodules and adult worms can be found in the trachea of infected birds. Faecal smears can also be performed, which may reveal characteristic bioperculate egg.

Treatment and control:

Treatment:

Albendazole is a benzimidazole anthelmintic which is used off-label in poultry. The drug is given to each bird orally. Measure out ¼ mL (per bantam) or ½ mL (per regular-sized breed). Repeat in 2 weeks.

Control

Tilling the soil in the pens at the end of the growing season helps to reduce the residual infection.

3.5.3 Helminthiasis

Definition and causative agent:

It is an infection by members of the phylum *Nematoda* (roundworms) or the class *Cestoda* (tapeworms, flatworms). In rare cases, infected birds develop clinical signs such as apathy or diarrhea, and the influence on technical parameters is usually negligible. *Ascaridia galli* is by far the most common roundworm.

Transmission:

Nematodes have either a species-specific, direct life cycle with bird-to-bird transmission by ingestion of infective eggs or larvae or have an indirect cycle that requires an intermediate host (e.g. insects, snails, or slugs).

Cestodes require an intermediate host (eg. insects, crustaceans, earthworms, or snails).

Clinical signs:

Ascaridia, *Heterakis*, and *Capillaria* spp. are widely distributed and cause such nonspecific as general unthriftiness, inactivity, depressed appetite, and suppressed growth; in severe cases, death may result.

H. gallinarum, a mild pathogen, in large numbers may cause thickening, inflammation, or nodulation in the cecal walls. *H. gallinarum* carries *Histomonas meleagridis*, the protozoan that causes h.

Lesions:

Most pathogenic tapeworms are found in the small intestine; the scolex, usually buried in the mucosa, generally causes mild lesions.

Diagnosis:

A reliable diagnosis of helminthiasis can be made by accurate identification of the individually recovered parasites by their morphology or increasingly by molecular biological methods.

To determine the species by morphology, worms detected during necropsy should be carefully removed, put into a saline solution, and examined under a microscope.

Detection of worm eggs by fecal flotation allows for the reliable confirmation of the presence of worms.

Treatment and Control:

There are decreasing numbers of medications approved for treatment of helminthiasis in poultry.

There are also reports of resistance developing against the remaining drugs. Treatment can be by piperazine citrate 10 gm in 6-8 litres of water or 10 kg powder in 3.5 kg of feed for 2 consecutive days.

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