CHAPTER TWO

2. Major Animal Diseases and their management

2.1. Infectious diseases caused by Bacteria

ANTHRAX; Synonyms: Splenic fever; Wool sorter's disease

Anthrax is a widespread infectious disease of livestock that occurs throughout the globe. It is a peracute disease characterized by sudden death with the exudation of tarry blood from the natural body orifices of cadaver. Anthrax is a zoonotic disease

Etiology: ***Bacillus anthracis*** is the specific cause of the disease. The bacilli are aerobic, spore forming, gram-positive organisms. When materials containing anthrax bacilli are exposed to air (oxygen) spores are formed. The spores are never formed when the organisms are in the circulation. The spores are resistant to most external influences including cold, hot, chemicals and drying conditions.

Occurrence- Outbreaks originating from a soil-borne infection always occur after a major climatic change, for example heavy rain after a prolonged drought and always in warm weather when the environmental temperature is over 15ºc.
Species Affected -. The most susceptible animals are cattle and sheep and the disease occurs less frequently in goats and horses. Birds are refractory to natural infection.

Transmission – The spores have got the ability to remain viable in the soil for a considerable period of time and thus remain as a continuous source of infection to the susceptible animal. Infection gains entrance to the body by ingestion, inhalation or through the skin. It is generally considered that animals are infected by the ingestion of contaminated food and water.

Biting flies and other insects have often been found to harbour anthrax organism and their ability to transmit the infection mechanically has been reported. Streams, insects, dogs and other carnivores, and wild birds may accomplish spread of the organism within an area. However, introduction of infection into a new area is usually through contaminated animal products such as bone meal, fertilizers, hides, hair and wool.

Clinical Findings - The clinical sign differs in different species. The incubation period varies from 2 to 10 days. In cattle and sheep only two forms of the disease occur, the peracute and the acuteforms.

- ***Peracute form*** - This form is the most common at the beginning of an outbreak. Animals are usually found dead without premonitory signs, the course being probably only 1-2 hours, but fever, muscle tremor, dyspnoea and congestion of the mucosa may be observed. After death, discharge of tarry blood from the nostrils, mouth, anus and vulva are common.

- **The acute form-** This form runs a course of about 48 hours.
  - Severe depression and listlessness are usually observed first.
  - High body temperature up to 42º c; Rapid and deep respiration. Increased heart rate.
  - Anorexia and ruminal stasis is evident.
  - Pregnant cow may abort. Congested and hemorrhagic mucous membrane. Reduced milk yield and the milk may be stained with blood.
  - Alimentary tract involvement is usual and is characterized by diarrhoea and dysentery.
  - Local edema of the tongue, in the region of the throat, sternum, perineum and flank may occur.

Post-mortem Findings: Absence of rigor mortis and the carcass undergoes decomposition. All natural orifices usually exudate dark tarry blood, which doesn't clot, and putrefaction and bloating are rapid.

☠ **WARNING**: If there is a good reason to suspect the existence of anthrax, the carcass should not be opened. The failure of the blood to clot, the presence of blood stained serous fluid in the body cavities, severe enteritis and gross enlargement of the spleen are almost certain indications of anthrax.

Diagnosis: To confirm the diagnosis on an opened carcass, smears of peripheral blood or local oedema fluid should be collected by needle puncture. Then, the smear stained with Methylene blue or Geimsa stain. Positive blood film stained shows the presence of large square ended blue rods in short chains with pink capsules.

Differential Diagnosis: Anthrax should be differentiated from lightening stroke, black quarter, hemorrhagic septicaemia and snakebite.

Treatment*:* Antibiotics and anti-anthrax serum are most commonly used. Severely ill animals are unlikely to recover but in early stages, particularly when fever is detected before other signs are evident, recovery can be anticipated. Anthrax bacilli are sensitive to Penicillin and other broad -spectrum antibiotics.

- Penicillin - 10,000 units / kg body weight twice daily.
- Streptomycin - 8-10 mg/kg / day in two doses IM for cattle is much more effective.
- Oxytetracycline- 5 mg /kg body weight / day has also proved superior to penicillin in the treatment of clinical cases after vaccination in cattle.
- Anti-anthrax serum- 100-250 ml daily through intravenous route along with a course of antibiotic may be given.

Note: In all cases treatment has to be continued at least for 5-7 days.

Control

- Careful disposal of infected material is most important. Infected carcasses should not be opened but immediately burned or buried, together with beddings and soil contaminated by discharges. Burial should be at least 2m deep with an ample supply of quicklime added.
- Strict quarantine arrangement should be made in anthrax prone areas.
- Adjacent areas of the dead animals should be thoroughly disinfected by applying 10% caustic soda or 10% formaline.
- Fly repellent may be used to control the insect population.
- Immunization- Anthrax vaccine should be given annually one to two months prior to anthrax season. *Sterne* vaccine is an avirulent vaccine that does not have risk of causing anthrax following vaccination and produce a strong immunity.

CLOSTRIDIAL INFECTIONS

Clostridial organisms are all potent producers of *exotoxins* upon which their pathogenecity depends on species. Pathogenic clostridia are commonly present in soils. They are also found in the intestinal contents of normal animals and cause disease only in special circumstances.

I- BLACKLEG*; Synonyms*: Black quarter; symptomatic Anthrax

Blackleg is an acute infectious but not contagious disease of cattle, goat and sheep and characterized by inflammation of the muscles, severe toxaemia and high mortality.

Etiology: caused by *Clostridium chauvoei*, a gram-positive, spore forming and toxin producing anaerobe. The spores are highly resistant to environmental changes and disinfectants and persist in the soil for many years.

Occurrence: The disease is wide spread in most of the tropical countries of the world. Blackleg is a soil-borne infection. In cattle the disease is largely confined to young stock between the ages of 6 months and 2 years and it appears to occur most frequently in rapidly growing cattle and highly nourished animals.

Species Affected: Cattle are the most susceptible animals but infection may spread to sheep and goats following trauma of muscles. Horse and pig can also be infected in a very rare occasion.

Transmission: The disease spread from contaminated soils. The organism gains entry through ingestion of infection feeds or possibly through contamination of wounds.

Clinical Findings: Incubation period is usually 2 to 5 days.

- In cattle the first symptom is fever (41 ºc).

- Anorexia, ruminal stasis and there is lameness in one of the limb.
- Characteristic swelling develops in one of the thick layers of muscles. Most commonly the lesions are located on the thigh, shoulder, neck and lumbar region.
- Swellings are hot and painful in the early stage and become cold and painless later.
- The muscle tissues are swollen, dark in colour and turn dry.
- On palpation swellings emit cracking or crepitation sound due to emphysema.
- There is also labored breathing and abdominal pain.

Necropsy Findings: large crepitating swellings are the most characteristic necropsy Findings.

- Affected muscles are infiltrated with yellowish exudates.
- Affected muscles turn black.
- A rancid odour (butyric odour) emanate from the muscles
- Blood stained discharge may ooze from the nostrils but here the blood rapidly clots
- Putrefaction and bloating occur very quickly.

Diagnosis: Tentative diagnosis is based on history, clinical observation and post-mortem findings. In the laboratory the disease can be diagnosed by microscopic examination of smear made from the affected tissues or fluid of the swellings. Gram-positive rods with sub terminal spores will be seen.

Differential Diagnosis: Anthrax, Malignant oedema and Bacillaryhaemoglobinuria.

Treatment: Satisfactory response has been reported from the use of penicillin and oxytetracycline. The antibiotic may be injected into the affected muscles. Penicillin at the dose of 10,000 units / kg body weight / day is extensively used and considered as drug of choice.

Control: In endemic areas annual vaccination of all cattle between 6 months and 2 years of age should be carried out prior to the anticipated danger period.

- Immunity following vaccination doesn't develop for 14 days and deaths may continue for some days if vaccination is carried out during an outbreak. Therefore, to avoid new cases during this time antibiotics (penicillin at a dose of 6000 unit / kg body weight intramuscularly) should be administered at the time of vaccination.The carcasses of animals dying of blackleg are destroyed by burning or deep burial to limit soil contamination.

II-TETANUS; Synonym: lock jaw

It is a highly fatal, non-contagious, non-febrile infectious disease of mammals caused by toxin and characterized by spasmodic contraction of skeletal muscles.

Etiology: ***Clostridium tetani***.The organism is anaerobic and it is gram-positive bacteria. The bacteria is spore forming and are capable of persisting in the soil for many years. The spores are also resistant to many standard disinfectants.

Occurrence: Tetanus occurs in all parts of the world and in all farm animals as sporadic case. The portal of entry is usually through deep puncture of wounds. A high incidence of tetanus may occur following castration, shearing, docking and vaccination for other diseases in lambs.

Species Affected: There is a considerable variation in susceptibility between species, the horse being the most susceptible and cattle the least. Human beings are extremely susceptible.

Transmission: Organisms may continue to live in the faeces for a long period of time and thus remain as a potential source of infection to manand other animals. The organism gains entrance through deep punctured wound contaminated with bacterial spores. Organisms may gain access during parturition and manual handling of the genitalia with contaminated hands.

Clinical Findings: The clinical picture is similar in all animal species.

- A generalized increase in muscle stiffness is observed first and is accompanied by muscle tremor.
- Prolapse of the third eyelid.
- Restriction of jaw movements - the animal may continue to eat and drink in the early stage but mastication is soon prevented by tetany of the masseter muscle and saliva may drool from the mouth.
- Stiffness of the hind legs
- Anxious and alert expression contributed by an erection of the ears, retraction of the eyelids and dilation of the nostrils and exaggerated responses to normal stimuli.
- The temperature and pulse rate are within the normal range in the early stages but may rise later when muscular tone are further increased.
- As the disease progresses, muscular tetany increases and the animal adopts a '*Sawhorse'*posture.

Post-mortem Findings: There are no satisfactory gross or histological findings by which a diagnosis can be confirmed.

Diagnosis: Fully developed tetanus is so characteristic clinically that it is seldom confused with other diseases. The muscular spasms, prolapse of the third eyelid and a recent history of accidental injury or surgery are characteristic findings.

Differential Diagnosis:strychnine poisoning, grass tetany, milk fever, and rabies may be confused with tetanus.

Treatment - The principle of treatment directs to:

i. Destruction of tetanus organisms - for this penicillin is the drug of choice and it should be given in massive doses. It may be given locally to minimize further multiplication of the bacteria. 1% H_2O_2 may be used to clean the wound.

ii. Neutralization of circulating toxin - Antitoxins is used for this purpose. 3000 to 7000 i.u. of antitoxin may be used depending on the size of the animal at 12hrs interval.

iii. Relaxation of muscle - muscle relaxants like chlorpromazine can be used for this purpose. This is important to prevent asphyxiation

Control

- Proper skin and instrument disinfection during castration, docking and shearing time.
- For short - term prophylaxis, passive immunity can be achieved by the injection of antitoxin subcutaneously.
- In endemic areas all susceptible animals should be actively immunized with ' toxoid 'formalin treated toxin.
- Vaccinating their mothers at late pregnancy can protect young foals and lambs.

TUBERCULOSIS

It is a chronic contagious disease of man & animals caused by certain pathogenic organisms of the genus *Mycobacterium*, characterized by development of tubercles with resultant caseation & calcification in any organs.

Etiology: *Mycobacterium bovis* is the common cause of tuberculosis in cattle. However, avian strain (*M. avium*) and human strain (*M. tuberculosis*) can cause infection in cattle and other species of animals. The organism is acid-fast, non-sporulated and non-motile in nature. The tubercle bacilli are enveloped in a waxy or fatty capsule, which protect and make them resistant to external influences.

Occurrence: The disease has a worldwide distribution.

Species Affected: The disease can occur in all species including man. But of all domestic animals, cattle are most susceptible and bovine strain is most pathogenic over the other strains.

Transmission: Age, malnutrition, concurrent infection, hygiene and management are important factors that affect the transmission as well as establishment of the disease.

Susceptible animals contract infection directly through inhalation of infected droplets and ingestion of contaminated feed and water by sputum, faeces, uterine and vaginal discharges, semen, milk, lymph and wound discharges.

Calf may contract the infection via infected milk or as a result of uterine infection. Congenital infection of calf occurs due to tuberculous metritis of dam. Cattle may be infected while they come in contact with tuberculous people or when exposed to secretion or excretion of infected human sewage. Tuberculous cattle are the major sources of milk-borne disease in human particularly in children.

Clinical Findings: The disease is a chronic insidious in nature and therefore may not be apparent clinically in the initial stage of the disease. .

- The infected animals lose body weight slowly but continuously in spite of provision of optimum nutrition.
- The respiratory system is mostly affected. There is sign of dyspnoea, increased rate of respiration and deep, persistent, painful, dry and hacking cough. Sings of bronchopneumonia is evident. Tuberculous pleurisy may occur.
- Percussion will produce dull sound on the affected consolidated areas.
- The involvement of the digestive tract is characterized by abdominal pain, persistent diarrhoea, chronic bloat and emaciation.
- When the uterus is involved there is purulent caseous discharge tinged with blood. The affected animal may also show irregular oestrus cycle, abortion and sterility.
- Tuberculous mastitis is a major importance because of the danger to public health and spread of the disease to calves.

Necropsy Findings: The organs most commonly affected include lungs, liver, pleura, peritoneum, kidney, spleen and regional lymph nodes. In some cases bones and joints are involved. Male and female genital organs can also affect. Tuberculous lesions (granulomatus lesions) are the main lesions. The lesions may be large and involve the entire lungs or liver or small and numerous; such condition is referred as **milliary tuberculosis**. In bovine tuberculosis lungs and associated lymph nodes are usually affected. Tuberculous lesions may persist in animals' body for the entire lifetime.

Diagnosis: This is based on the following:

a) Clinical signs: characterized by gradual emaciation, stunted growth, enlargement of lymph nodes, diurnal variation of temperature and characteristic coughing.
b) Necropsy finding: clinical lesions are usually recognized during post-mortem examination of a slaughtered animal or the one that has died of the effect of the disease.
c) Laboratory diagnosis: is made by direct microscopic examination (sputum and milk) stained with ziehl - Nielsen may demonstrate acid-fast rods and culture (The samples which can be used to culture acid fast organisms are sputum, faeces, nasal discharge, urine, uterine discharge, milk and discharges from lymph nodes.)
d) Radiology: radiological examination is helpful in small-domesticated animal (pet animals).
e) Single Intradermal tuberculin test: In this case 0.1ml tuberculin or purified protein derivative (PPD) injected intradermaly in the middle portion of neck of bovine, a site preferred due to maximum sensitivity. Prior to tuberculin injection, the skin should be cleaned, shaved and thickness of skin fold measured using calliper. The reaction can be read after 72 hr. of injection; the positive reaction is characterised by a hot, red and painful swelling with an increase of 4mm or more in the skin**;** less than 2mm is negative and between 2-4mm is doubtful.

Treatment: treatment of tuberculosis in animals is generally attempted more or less in the same line as of human treatment. Since treatment takes long duration, in animals chemotherapy is not recommended because of economic reason and transmission of the disease while the animal is under chemotherapy. But if necessary, isoniazid and Streptomycin can be used against tuberculosis in animals.

Control: test and slaughter policy is the only method by which effective eradication of the disease can be achieved.

- Hygienic measures to prevent the spread of infection should be instituted as soon as the first group of reactors is removed.5% phenol can be used to disinfect all feeding and watering troughs.
- It is most important that calves being reared as herd replacements be fed on tuberculosis-free milk, either from known free animals or pasteurised.
- Avoid or prevent communal use of watering facilities or pasture.

PARATUBERCULOSIS: Synonym: Johne's disease

Johne's disease is specific infectious enteritis of cattle, sheep and goats. It is characterized by progressive emaciation in all species affected, and in cattle by chronic diarrhoea and a thickening and corrugation of the wall of the intestine.
Etiology: the disease is caused by *Mycobacterium Paratuberculosis.* The organism is a small, acid-fast bacillus.

Occurrence: The disease occurs throughout the world. Infection occurs in animals at a very early age, usually under 30 days of age, and clinical disease does not occur until 3-5 years of age. However calves reared on infected nurse cows may develops clinical disease at 13-18 months of age. Adult animals exposed for the first time may either develop clinical disease or they may became carriers of the organism without manifesting clinical signs

Species Affected: Johne's disease is essentially a bovine disease but also other ruminants like sheep and goats

Transmission: under field conditions the disease is transmitted principally by ingestion of feed and water contaminated by the faeces of infected animals Calves may acquire the infection in their intra-uterine lives and/or though the way of alimentary tract by consuming milk from cows with clinical Johne's disease.

Cl. Findings: incubation period is long (15-18 month). In cattle, clinical signs donot appear before 2 years of age

- Emaciation, sub-mandibular edema and absence of fever. The edema has a tendency to disappear as diarrhoea develops.

- The animal eats normally but thirst is excessive. The faeces are soft and thin and without offensive odour (no blood, epithelial, debris and mucus)
- Diarrhoea may be continuous or intermittent and the disease always terminates in severe dehydration, emaciation and weakness
- Cases occur only sporadically because of the slow rate of spread of the disease.

In sheep and goats the disease is manifested by emaciation and less severe diarrhoea (faces lose its usual pelleted form). Sheep may shed wool, lose weight and goats may be show depression and dyspnoea.

Necropsy findings: In cattle, lesions are confined to the posterior part of the alimentary tract and its associated lymph node.

- Thickening of the intestinal wall up to three or four times normal thickness, with corrugation of the mucosa, is characteristic. The lesions may reach from the rectum to duodenum.
- In sheep there may be a deep yellow pigmentation of the intestinal wall and although corrugation of the mucosa is not a common finding. The wall may be thickened. .

Diagnosis: The characteristic features of clinical Johne's disease includes the following:

- ⇨ Clinical sings like progressive weight loss and emaciation in single animal and chronic diarrhoea, which does not respond to therapy.
- ⇨ Definitive diagnosis can be obtained by using a combination of serological tastes, faecal culture and biopsy of intestine.
- ⇨ Cutaneous delayed hypersensitivity tests may be used but bovine or avian tuberculosis and animals vaccinated against Johne's disease may give suspicious or positive reaction.
- ⇨ Serological tests: CFT, FAT etc…
- ⇨ Rectal scrapings or rectal pintch biopsy are used. A positive finding is clumps of acid-fast bacilli in epithelial cells or macrophages.

Differential diagnosis: In cattle: Salmonellosis, coccidiosis, parasitism, and malnutrition

In sheep and goats: caseaous lymphedenitis, GI parasitism, Internal abscesses, ovine progressive pneumonia, etc…

Treatment: Not recommended due to adverse course of the disease. However, streptomycin 50mg/kg body weight per day may result transient improvement in clinical signs. .

Control: lack of accurate tests and the long incubation period of the disease make Johne's disease difficult to control.

- On herd basis control depends upon eradication of infected animals
- Hygiene to prevent further spread
- In some instances, vaccination to increase the resistance of the residual population. In cattle vaccination is carried out only in calves less than 1 months of age.

PASTEURELLOSIS

This is an acute septicemic disease occurring most often in cattle, camel, sheep, goat and pig and usually occurs following some form of stress such as transportation, shipping to feedlots or inclement weather.
In cattle there are two types of pasteurellosis: *pneumonic pasteurellosis* and *Septicemicpasteurellosis*.

I-Pneumonic pasteurellosis: Synonyms: Shipping fever pneumonia

It is associated with infection by *pasteurella hemolytica* and occasionally *pasteurella multocida*. It is characterized clinically by acute bronchopneumonia with toxaemia.

Occurrence - In most cases, both of these pasteurella species are part of the normal flora of the upper respiratory tract. Following stress or viral infection these appears to be an increase in the total numbers and virulence of the organism in the nasopharynx, which are then inhaled in to the alveoli. Young growing cattle within the age group of 6 months to 2 years are most often affected. Feedlot cattle may suffer when they are put under stress (transportation, temporary starvation, rapid fluctuations in ambient temperature, mixing of cattle from d/t origins)

Transmission: occurs by the inhalation of infected droplets. This may be from clinical cases or recovered carriers in which the infection persists in the upper respiratory tract. The disease may spread very quickly in closely confined animals but may be much slower in animals at pasture.

Clinical finding: The disease usually develops in cattle within 10 to 14 days after they have been stressed. The affected cattle are usually **depressed**, **anorectic**, have a fever (40 -41º c) and a serous to mucopurulent nasal discharge with rapid, shallow breathing and often coughing if they move. In severe cases, pleurisy may develop, which is characterized by an irregular breathing pattern and grunting on expiration Lung auscultation may reveal moist **rales**, pleuritic frictional rubs, and crackling. As lung consolidation progresses, lung sounds may be decreased.

Post-mortem examination: the anteroventral portions of the lung are dark red, swollen, and hard (marked consolidation) and often are covered with fibrin.

- Adhesions between adjacent pleural surfaces are common.
- Catarrhal bronchitis and bronchiolitis and serofibrinous pleurisy are usually present and may be accompanied by a fibrinous pericarditis.
- The bronchi may contain fibrin, mucus, blood clots and pus.

Diagnosis: is based on history of movement or any stress, clinical findings and necropsy findings. Confirmatory diagnosis made by the isolation and identification of the organism.

Differential diagnosis - Infectious Bovine Rhinotrachitis (IBR), Lungworm (verminus) pneumonia , CBPP

Treatment:

- Oxytetracycline 10 mg/Kg body weight (10%), Iv or IM daily for 3 days
- Trimethoprim-sulfamethoxazole 3-5 ml/45kg body weights IV or IM for 3 days
- Penicillin 20000-30000 IU/kg body wt. IM or SC daily for 3 days
- Sulfamethazine (liquid preparation) 150 mg/kg b. wt IV or orally daily for 3 days.

Control and prevention: Successful control begins with the adoption of good management techniques, when the calves are still on the range, the judicious use of efficacious vaccines and care in handling and transportation of cattle.

- Stressful procedures such as castration, dehorning, branding, deworming should be distributed over a period of time rather than concentrated at once. Vaccination at least 2 weeks before shipment.
- During transportation bedding are necessary and cattle should not be without feed and water for more than 24 -30 hours. Ventilation is very important.
- In dairy calves, vaccination of the dam may be beneficial to provide passive immunity to the calves

Septicemic Pasteurellosis; Synonym: hemorrhagic septicaemia
The disease is an acute septicaemia caused by *pasteurella multocida.*

Occurrence: it occurs in an outbreak during periods of environmental stress, the causative organism in the intervening periods persisting on the tonsilar and nasopharyngeal mucosa of carrier animals.

Transmission: spread occurs by ingestion of contaminated foodstuff, the infection originating from clinically normal carriers or clinical cases. The saliva of affected animals contains large numbers of pasteurella during the early stages of the disease. Although infection occurs by ingestion, the organism does not survive on pasture for more than 24 hours.

Clinical signs: Clinically the disease is characterized by a sudden onset of fever (41-42ºc), profuse salivation, and submucosal petechiation.; Severe depression and death with in 24 hours.

- Localization may occur in subcutaneous tissue, resulting in the development of warm, painful swellings about the throat, dewlap, brisket or perineum,& severe dyspnoea may occur if the respiration is obstructed.

Post mortem findings: generalized patechialhaemorrhages, particularly under the serosaand edema of the lung and lymph nodes. In a few animals hemorrhagic gastroenteritis may be present.

Diagnosis: tentatively, the disease is diagnosed based on history, clinical findings and necropsy findings but confirmatory diagnosis is done by isolation and identification of the causative bacteria.

Differential Diagnoses: anthrax, blackleg, and acute leptospirosis

Treatment: Those drugs listed for pneumonic pasterellosis are also effective for septicemicpasterellosis

Prevention and control: Vaccination of susceptible group is effective to protect for at least 12 months. The effective control lies with institution of adequate management, rational executions of vaccines and care in the transportation of animals.

COLIBACILLOSIS; Synonyms: Calf scour, white diarrhoea

Colibacillosis is one of the diseases of new borne animals (calves, piglets, lambs and foals) caused by *Escherichia coli* and characterized by marked prostrations, profuse diarrhoea and septicaemia.

Etiology: *E. coli* is the causative agent. The agent produces endotoxin.

Occurrence: The disease is common in farm animals under 3 days of age but it may occur as early as 12-18 hours after birth and occasionally occurs in calves up to several days of age when there is mixed infection with viral pathogens. There is high mortality in calves (10-50%) and piglets.

Species affected: Incidence of colibacillosis is higher in dairy herds (calves) but also affect piglets, foals, lambs& kids.

Transmission: The disease is transmitted through contaminated feed and water. In most species the major primary source of infection is the faeces of infected animals. In addition calves obtain the organism from contaminated bedding and calf pans, diarrheic calves, mastiticmilk (coliform mastitis) and from the skin of the perineum and udder of the cow, and from the animal attendant.

Clinical findings: The disease appears as acute, per acute or chronic form.

- In per acute infection there is marked diarrhoea, prostration and death with in 12 hours
- Acute signs appear when the newborn animal is 1-2 days old. The usual signs are depression, in appetence, stiffness, sunken eyes, tucked up abdomen, rough hair coat, elevation of temperature and rapid pulse and respiratory rate. There are profuse loose faeces (yellowish brown to greyish white) with rapid peristalsis. There is straining in evacuation of faeces, which has a fetid odour. There is soiling of the anal, and hindquarter region. There is rapid dehydration and ultimately death.
- Chronic cases show joint ill, navel ill and pneumonia.

Necropsy Findings: inPer-acute cases there will not marked changes in organs, except hypermia of gastro-intestinal mucosa.

Acute cases show edema and haemorrhages of the abomasal folds in calves. The intestinal mucosa will show hemorrhage and wrinkling.

In chronic cases, there is a suppurative change in the joints. Changes may be seen in umbilicus, lungs and kidneys.

Diagnosis: This is based on history, clinical findings, lesions, isolation of organism in culture and serological tests.

Treatment: The consideration for treatment of acute neonatal diarrhoea includes.

- Alteration of the diet**.** i.e.provide readily absorbable substances such as glucose and electrolyte mixtures.
- Fluid & electrolyte replacement to prevent dehydration, acidosis and electrolyte imbalance. Give by parenteral or oral route. Glucose, sodium bicarbonate, sodium chloride; potassium chloride and water.
- Anti microbial therapy
    - Chloroamphinicol25-50 mg/kg day body wt.
    - Ampicillin 7-10 mg/kg for 5-7 days may be given orally or parentally.
    - Streptomycin and tetracycline may also be used.

Control - The following three principles are important in control of colibacillosis

- Reduce the degree of exposure of new borne calves and piglets to the infectious agent.
- Provide maximum non-specific resistance with adequate colostrums and optimum animal husbandry.
- Increase the specific resistance of the newborn by vaccination of the dam or the new born by *E. coli* K^{99} antigen.

NB- When outbreaks of colibacillosis occur, every effort should be made to isolate affected animals from other susceptible calves and piglets. Dieteric diarrhoea should be differentiated in which the faeces are voluminous, pasty to gelatinous in consistency; the animal is bright and alert.

SALMONELLOSIS

It is an acute or chronic contagious disease of all species of animals and human being characterized by gastro-enteritis. The disease has got economic importance as it causes heavy mortality and high morbidity.

Etiology: The disease is caused by different species of salmonella. Some of the important salmonella species are: *S. typhi* (man): *S. typhimarium* (cattle, sheep, goat, pig, and horse):

S. dublin (cattle); and *S. galinarum* (birds). The organisms are gram- negative and rod shaped.

Occurrence: The disease is worldwide in distribution and has been recorded from all the tropical countries.

Species affected: The disease may occur in cattle, sheep, horse, man, fowl and pigs.

Transmission: Contaminated water is the important source of transmission. Cattle may acquire the infection from infected cattle or from infected pig, horse, sheep, dog or poultry. Animals may

contract the infection from human sources. Introduction of the disease into a new farm is through carrier animals.

Clinical Findings: Animals may remain as a symptomatic carrier without exhibiting any clinical manifestations. The disease may appear as acute or chronic one. From clinical standpoint, it has been classified as enteric form and septicemic form.

Enteric Form: this form is most commonly observed in adults characterized by anorexia depression, high temperature and profuse watery diarrhoea. Initially, animals may pass semisolid faeces, which are followed by diarrhoea with blood clot and mucus. Abdominal pain and anaemia may occur following frequent defecation and extensive haemorrhage, respectively. Gradually the initial temperature drops to subnormal level and death occurs. Pregnant cow may abort.

Septicaemic form*:* this form is observed in young animals characterized by high-rise of temperature, dullness, ataxia, and recumbency. The animal may die within 1-2 days, but in some cases there is involvement of musculo-skeletal system resulting to enlargement of joints. Signs of pneumonia may appear (pneumoenteritis). The recovered animals continue to excrete the organisms throughout its life.

Necropsy Findings: Haemorrhage and edema in the gastric mucosa.

- Hyperemia, necrosis and Ulcers in the intestinal mucosa
- Liver becomes enlarged with necrotic foci
- Spleen becomes enlarged and pulpy; Haemorrhage of mesenteric lymph nodes

Diagnosis: It can be made based on history, clinical signs, and necropsy findings. Confirmation is by isolation and identification of the organism by culture

Differential Diagnosis: Coccidiosis, pasteurellosis, colibacillosis, paratuberculosis andliver fluke infestation.

Treatment: early treatment with broad-spectrum antibiotics and with sulphonamides is highlyeffective. Delays in treatment means loss of the integrity of intestinal mucosa to the point were repair couldn't occur. Drugs commonly used for the treatment of salmonellosis are: Chloramphenicol, Trimethoprim and Sulfadoxine mixture, and Nitrofurazone (orally).

Note*:*Parentral or oral treatment alone may be sufficient if cases are detected early, but in all circumstances parentral and oral treatment combined is superior and is recommended.

Control: Calving should be made in a hygienic place.

- Calves should be allowed to have access to colostrums following their birth
- Calves should not be exposed to adverse weather (too hot or too cold environment)
- Animals should not be allowed to drink water from sewage of stagnant pool or pond.
- Affected animals should be brought under immediate treatment.

BRUCELLOSIS: Synonyms: Bang's disease

Brucellosis is an acute or chronic contagious disease of domestic animals that causes placentitis and abortion. It is occupational disease in human being and may be acquired from animals.

Etiology: - caused by *Brucella* spp. i.e. caused by *Br. abortus* (for cattle), *Br. suis* (for swine) and *Br. melitensis* (for sheep and goat) but they can produce infection in other species of animals and human being. It is a gram-negative bacterium

Occurrence: -Brucellosis is widespread and of major economic importance in most countries of the world, particularly amongst dairy cattle. Infection occurs in cattle of all ages but persists most commonly in sexually mature animals. Pregnant cattle are highly susceptible.

Transmission: -the organism has a greatest concentration in the contents of the pregnant uterus, the fetus and the fatal membranes, and these must be considered as major sources of infection. The disease is transmitted through ingestion of contaminated feed and water, penetration of intact skin and conjunctive and contamination of the udder during milking. Artificial insemination by infected semen also spreads the disease. Organisms are shed before and after abortion.

Clinical Findings: abortion occurs after 5th month of pregnancy with retention of placenta and metritis in cow. Metritis may be acute with septicaemia and following death, or chronic leading to sterility.

In the bull, orchits and epididymitis occur occasionally. One or both scrotal sacs may be affected with acute, painfulswellings twice-normal size. The seminal vesicles become enlarged and can be detected through rectal palpation.

Hygromatous swelling, especially on the knees, should be viewed with suspicion.

Necropsy Findings: The placenta is usually edematous and necrosis on the cotyledons.

Diagnosis: -Abortion in last trimester of pregnancy.

- Blood samples for serological tests (Rose Bengal plate test, CFT, ELISA, milk ring test, etc).
- Examine uterine fluids and the contents of the fetalabomasums.

Differential Diagnosis: Trichomonosis, Leptospirosis, Listeriosis, vibriosis, mycotic abortion, etc.

Treatment: Treatment is not usually under taken.

For cow long acting Oxytetracycline at 20-mg/kg b.wt. IM for 5 days treatments in combination with streptomycin at 25mg/kg bwt. IM or IV daily for 7 consecutive days may be partially successful

Control: test and slaughter method will be the most rational approach. All abortion should be suspected of brucellosis, unless otherwise proved.

- ⇨ Hygienic disposal of uterine discharges, foetus, and foetal membranes should be done strictly and clean and disinfect contaminated premises.
- ⇨ Restrict movement of infected animals.
- ⇨ Vaccination with a single 5ml dose of Br. abortus strain 19 live vaccine given subcutaneously for 2 to 6 months of age confess adequate immunity against abortion for 5 or more subsequent lactations. Strain 45/20 vaccines in adjuvant are used for those beyond 8 to 9 months of age. This is a killed (inactivates) vaccine.

STRANGLES

Synonym: Equine Distemper

It is an acute infections disease of equines characterized by catarrhal inflammation of the upper respiratory tract with suppuration and abscessation of the associated lymph nodes.

Etiology: The disease is caused by S*treptococcus equi*. It is a gram-positive coccus**. Occurrence:** The distribution is worldwide. Although, with the decline in horse numbers and improvement in therapy, it has become a minor importance in most countries. Outbreaks can occur at any time of the year but are most likely to happen in cold, wet weather.

Species affected: this is essentially a disease of equines. Horses suffer much more severely than donkeys and mules. Young horses within the age group of 6 months to 3years are most susceptible.

Transmission: nasal discharges of the infected animals are the most important source of transmission. Organisms usually get entry through ingestion of contaminated materials. Inhalation of infected droplet is also a possible source of infection. Transmission may also take place from contaminated fomites.

Clinical Findings: The incubation period ranges from 2 to 5 days. The mortality rate of the disease is 9-10% but the morbidity can reach 30-100%.

- ➢ High-rise of temperature, profound depression, reluctance to move, eat or drink.
- ➢ Nasal discharge which range from serous to mucopurulent and finally purulent.
- ➢ Cough is evident due to pharyngitis and laryngitis.
- ➢ In most cases, there is formation of abscess, which burst out liberating large quantities of thick yellowish or creamy pus.
- ➢ Recovery follows provided that there is no further complication. The complications that may follow are:
    - ⇨ Suppuration of retropharyngeal lymph nodes
    - ⇨ Abscess formation on mediastinal, bronchial and mesenteric lymph nodes
    - ⇨ Bronchopneumonia due to extension of infection from guttural pouch

Necropsy Findings: in a rare fatalities that occur there is extensive suppuration in internal organs, especially the liver, spleen, lungs, pleura, large vessels and the peritoneum.

- ➢ Abscess formation in pharyngeal and sub maxillary lymph nodes.

- Emphyema of the guttural pouch

Diagnosis: based on clinical sing*i.e.*upper respiratory tract infection with purulent nasal discharge and enlargement of the lymph nodes of the throat region are diagnostic of strangles.

- Epidemic nature of the disease in young horse
- Detection of streptococcus equi in supportive material by microscopic and serological tests.

Differential Diagnosis: Glanders

Treatment: Infected horse should be isolated and treatment started early as soon as possible. In the early stage penicillin is quite adequate but at a later stage intravenous injection of tetracycline (10mg/kg body wt) will be more effective but must be continued for 4-5 days.

Control: - The infected animals should be kept in isolation.

- Contaminated premises should be thoroughly disinfected
- In contact animals may be passively immunized by injecting immune serum.
- Freeze dried strangles vaccine may be given to susceptible animals for active immunization.

GLANDERS: Synonym: Farcy

Glanders is a contagious disease of equines, occurring in either acute or chronic forms and characterized by formation of nodules and ulcers involving upper air passage, lungs and cutaneous tissues. The disease is highly fatal and of major importance in any affected horse population. The disease is also transmissible to human beings.

Etiology: caused by P*seudomonas mallei*. It is a gram negative and non-spore forming bacteria. The organisms show very less resistance to the environmental influences. They are readily destroyed by direct sunlight and by most of the commonly used disinfectants.

Occurrence: the disease was prevalent in most of the horse rearing countries. It is still reported in Eastern Europe, Asia and Africa.

Species affected: Horse, mule and donkeys are highly susceptible. Man is susceptible and the prognosis of the disease always ends fatally.

Transmission: Spread mostly occurs by ingestion of contaminated feed and water contaminated by nasal discharge or sputum. Rarely the cutaneous form appears to arise through contamination of skin abrasions by direct contact or from harness or grooming tools. Spread by inhalation can also occur but this mode of infection is rare under natural conditions.

Clinical Findings: In the acute formthere is a high fever, cough and nasal discharge with rapidly spreading ulcers appearing on the nasal mucosa and nodules on the skin of the lower limbs or abdomen. Death due to septicaemia occurs in a few days.

In the chronic form, the signs may be related to the lesions, which occur in one or more of the prediction sites. When the localization is pulmonary, there is a chronic cough, frequent epistaxis

and laboured respiration. Nasal lesions appear on the lower part of the nasal septum. They start as nodules that may ulcerate. In the early stages there is a serous nasal discharge, which may be unilateral and which later becomes purulent and blood stained. Enlargement of the sub maxillary lymph node is a common occurrence.

The affected animal gradually looses its condition with poor hair coat. There is edema of the hind limbs down to the hock accompanied with discharge from lymph nodes.

The skin form is characterized by the appearance of subcutaneous nodules (1-2 cm in diameter), which ulcerate and discharge pus. Lymph nodes draining the area also involved and may discharge to the exterior. The lesions mostly occur in the medial aspect of the hock but they can occur on any part of the body.

Necropsy Findings: Lesions in the form of nodules and ulcers are noted in the nasal septum. Small nodules are observed throughout the legs. Sings of catarrhal bronchopneumonia and purulent lymphadenitis are observed.

Diagnosis: based on clinical signs and post mortem findings.

Differential Diagnosis: it should be differentiated from epizootic lymphangitis, ulcerative lymphangitis, strangles and other pneumonia of horse.

Treatment: Sodium sulfadiazine has been highly effective.

Control: For control and eradication of glanders every animal with clinical evidence of glanders and those that react positively must be regarded as diseased and should be destroyed.

- The carcass of animals must be burned or buried
- Infected premises should be vigorously disinfected
- Manure, beddings, feed residue should be burned and buried.

CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP)

Acute, sub-acute or chronic disease characterized by formation of massive pathological changes in lungs& pleura.

Etiology: *Mycoplasma mycoides Var. Mycoides* (small colony type) is the cause of the disease in cattle. The organism is extremely plemorphic in nature and sensitive to all environmental influences including disinfectants, heat and drying.

Occurrence: The disease is one of the major plagues in cattle causing heavy losses in many parts of the world.

Species Affected: This is primarily a disease of cattle, although the organism can infect sheep and goat.

Transmission: The principal method of spread of this disease is by inhalation of infective droplets from active or carrier cases of the disease. Because of the method of spread, outbreaks tend to be more extensive in housed animals and in those in transit by train.

A recovered cattle act as a carrier. They contain the organism in sequestra or in necrotic areas enclosed by connective tissue capsule of previously affected lungs. Such cattle are called *'Lungers*' .The sequestra may act as potential source of infection for a period as long as three years. Conditions of stress due to starvation, exhaustion or inter current disease can cause the sequestrum to break down and convert the animal into an active case.

Clinical Findings: incubation period of the disease is 3-6 weeks

- Sudden onset of high fever (40ºc), a fall in milk yield, anorexia, and cessation of rumination.
- There is severe depression and the animals stand apart or lag behind a travelling group.
- Coughing, first only on exercise, and chest pains are evident and disinclined to move.
- Standing with the elbows abducted, back arched and head extended,
- Respirations are shallow, rapid and accompanied by expiratory grunting.
- Auscultation reveals pleuritic frictional sounds in early stages of acute inflammation and dullness, fluid sounds and moist gurgling in the later stages of infection.
- Dullness of area of the lung may be detectable on percussion.
- Dilated nostrils, Drooling of saliva, nasal discharge and abortion of pregnant cow are also observed.

Necropsy Findings: Lesions are confined to the chest cavity. There is thickening and inflammation of the pleura often with heavy deposition of fibrin. Affected lung show various stages of gray and red hepatisation, the classical '*marbled*' appearance of the lung in this disease. Adhesion between pleural surfaces is a common finding

Diagnosis: This is based on the following consideration.

- ⇨ History - Prolonged incubation period, history of contact with infected animals.
- ⇨ Clinical Findings-Typical signs of respiratory involvement
- ⇨ Necropsy-Classical marbled appearance of the lung.
- ⇨ Culture the organism in special media. Pleomorphic organisms are seen with predominant filamentous form.

Differential Diagnosis: Pasteurellosis and parasitic pneumonia

Treatment: Treatment is usually undertaken only in areas where the disease is endemic, otherwise, eradication being the more logical practice when the diseases break out in a new area.

- Sulfadimidine and organic arsenicals are used extensively and appears to reduce the mortality rate.
- Streptomycin has some curative effect.
- Oxytetracycline and chloramphenicol have some value.
- Tylosine tartrate (10mg/kg body weight every 12hours for 6 injections IM) is highly effective

Control: Movement of cattle should be restricted.

- Infected animals should be removed from the herd as soon as possible
- Vaccination-It is an effective procedure in the control of CBPP but its application is usually controlled by local legislation. All the vaccines in use are living preparations.
- Carrier animals should be slaughtered

CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)

This is a contagious disease of goats having resemblance with CBPP. CCPP has many similarities clinically and at necropsy to CBPP, but it is not transmissible to cattle.

Etiology: *Mycoplasma mycoides*var. *caprae* is the causative organism.

Occurrence: The disease is wide spread in tropical countries

Transmission: The disease is readily transmitted by inhalation, but the organism does not survive for long time outside the animal body, so that a carrier animal brings infection into the flock.

Clinical Findings: This is a highly fatal disease of sheep and goat. Mortality varies from 60-100%. The incubation period is about 4 days. The symptoms are comprised of anorexia, dullness, depression, nasal discharge, coughing, dyspnoea, fever (40.5-41.5ºc) and in the terminal stages mouth breathing, tongue protrusion and frothy salivation with death in 2 or more days.

Necropsy Findings: The clinical lesions and pattern are almost similar to cattle except that a sequestrum

Diagnosis: More or less similar to CBPP

Treatment: tylosine tartrate (10mg/kg) or oxytetracycline (15mg/kg bwt) is highly successful

Control: It depends on:

- Isolation of affected flocks
- Vaccination

EPIZOOTIC LYMPHANGITIS: Synonym: pseudo glanders

A chronic contagious disease of horses characterized by supportivelymphangitis, lymphadenitis and ulcers of skin.

Etiology: *Histoplasmafarciminosum* is the cause of the disease. It is fungal disease.

Occurrence: The disease occurs chiefly in Asia, Africa and the Mediterranean. It occurs in out breaks rather than as an endemic disease.

Species affected -Horses and rarely cattle and man are the species affected. Horses under 6 years of age are the most susceptible,

Transmission: from infected animals by direct contact or on inanimate objects such as bedding, grooming utensils, horse blankets or harness, and gain entry through cutaneous abrasions.

Clinical Findings: ulcer develops at the portal of entry, lymphatic vessels leaving the ulcer become thickened and enlarged and develop nodules along their course. The nodules erupt and discharging thick creamy pus. Local lymph nodes also enlarge and may rupture. Thickening of the skin in the area and general swelling of the whole limb are common. The lesions are quite painless.

In most cases the lesions develop on the limbs particularly about the hocks but may also be present on the back, sides, neck, vulva and scrotum. Occasionally, lesions appear on the nasal mucosa but are situated inside the nostrils and not involved in the nasal septum.

The disease is chronic, persisting for 3-12 months and affected animals lose body condition and cannot be worked. Spontaneous recovery occurs and immunity is solid after an attack but many animals are destroyed because of the chronic nature of the diseases.

Necropsy Findings: Lesions are usually confined to the skin, subcutaneous tissues and lymph vessels and lymph nodes

Diagnosis: microscopic examination from the discharge reveals Gram-positive yeast cells

- Culturing
- Serological tests like FAT can be used.

Differential Diagnosis: Glanders, ulcerative lymphangitis.

Treatment: Early cases can be cured by extensive excision of affected parts followed by frequent local application of silver nitrate or tincture of iodine

Control - Strict hygienic precautions must be observed.

- In endemic areas severe cases should be destroyed and less severe cases kept in strict quarantine while under going treatment.
- All contaminated bedding, harness and utensils should be destroyed or vigorously disinfected.

DERMATOPHILOSIS

Synonyms: Mycotic dermatitis; cutaneous streptothricosis; senekebo disease of cattle; lumpy wool of sheep; cutaneous actinomycosis.

This is a superficial skin infection of animals and man characterized by exudation and matting of hairs and wools and formation of crusts and scab.

Etiology: *Dermatophiluscongolensis*. The organism is a gram positive. The organism invade hair follicles, sweat glands and other epidermal structures.

Occurrence: The disease is prevalent throughout the tropical and temperate regions of the world. The disease appears to be most common under low moist climatic conditions.

Species Affected: The disease is most commonly encountered in cattle, sheep, and horse. But, it has been also reported in goat, donkey, dog, cat and pigs. Man is readily infected with dermatophilosis.

Transmission: All ages of animals are susceptible and the disease is usually transmitted through direct contact with infected animals or infected materials. Mechanical transmission through contaminated instrument, brush, bedding materials are possible. The disease may be transmitted through arthropod vectors like flies, ticks and sheep kids.

Moisture and cold weather are predisposing factors to transmit the disease and most of the outbreaks have been found to occur during rainy season. Skin abrasion is also another predisposing factor of dermatophilosis.

Clinical Findings: In cattle, lesions occur on the neck, body or back of the udder and may extend over the sides and down the legs and the ventral surface of the body. In adult cattle the characteristic lesions are thick, horny crusts, varying in color from cream to brown and they are 2-5 cm in diameters. In the early stages the crusts are very tenacious and attempts to lift them cause pain. Beneath the crusts there is granulation tissue and some pus. In the later stages, the dermatitis heals and the crusts separate from the skin but are held in place by penetrating hairs or wool fibres and are easily removed.

In young calves crust formation does not occur. There is extensive hair loss with tufting of the fibres, heavy dandruff and thickening and folding of the skin in later stages.

Necropsy Findings: In the occasional animals that die, there is extensive dermatitis sometimes a secondary pneumonia, and often evidence of concurrent disease.

Diagnosis: Microscopic examinations of stained exudates with Giemsa or Gram's stain. The causative organism may be isolated from skin scrapings or a biopsy section and is much easier to isolate from an acute case than a chronic one.

Differential Diagnosis: Photosensitization

Treatment: There is no completely satisfactory treatment for cases that show very extensive involvement. In general terms, better results are obtained during dry hot weather and in dry climate.

- ⇨ Penicillin and streptomycin at a very heavy dose rates (70mg streptomycin and procaine penicillin G 70000 Iu/kg body weight) is recommended as being 100% effective in animals.
- ⇨ Tetracycline (5mg /kg body weight repeated weekly as required is recommended and long acting tetracycline (20mg/kg body weight) in one injection has been reported to give excellent result in cattle.

- ⇨ Topical application of astringent or antibacterial solution as spray or dip has been considered as rational approach. For this, copper sulphate, Zinc sulphate are useful.

Control: Contact with infected animals and grooming materials are to be avoided.

- Control of tick and fly population are to be made with appropriate insecticide application.
- Quarantine of the affected animals may be made to prevent possible spread.

DERMATOPHYTOSIS; Synonym: Ringworm

Ringworm of the skin is caused by the invasion of the keratinized epithelial cells and hair fibers by dermatophytes.

Etiology: caused by fungi that grows on hair or skin or both. Dermatophytes are classified in to three genera; Microsporum, Trichophyton and Epidermophyton.

Occurrence: Dermatophytosis occurs in every parts of the world affecting human and animal population. Young animals are more susceptible to the infection. The disease is more commonly seen in animals housed in close proximity to each other for long periods.

Species Affected: Horses, donkeys, cattle, pig, sheep, goat, dog and cats are affected. Man is also affected.

Transmission: Direct contact with infected animals is a common method of spread of ringworm and licking with the tongue undoubtedly aids spread of the fungus. However, indirect contact with any inanimate objects, particularly bedding, harness, grooming kits and horse blankets, is probably more important.

Clinical Findings: In cattle the lesion is a heavy, grey- white crust raised perceptibly above the skin. The lesions are roughly circular and about 3 cm diameter. In the early stages the surface below the crust is moist.

In older lesions the scab becomes detached and pityriasis and alopecia may be the only obvious abnormalities. Lesions are most commonly found on the neck, head and perineum. Itching does not occur and secondary acne is unusual.

In horses the lesion commence as round patches of raised hair and soreness of the lesion to touch. After about 7 days matting of the hair, which becomes detached leaving a bald, grey, shining area about 3 cm in diameter will be resulted.

Diagnosis - The diagnosis of ringworm depends on evidence of infectivity, the appearance of characteristics lesions and the presence of fungal mycelia and spores (from scrapings and culture).

Treatment: There is spontaneous recovery but treatment greatly reduces contamination of the environment by infected animals. Local or systemic treatments are used, the later when lesion are wide spread.

For local application - weak solutions of iodide (2-5 %), white fields ointment, 10% ammoniated mercury ointment

For systemic treatment - IV injection of NaI (1g/14kg b.wt) as a 10% solution. Griseofulvin 5-7.5 mg/kg for 7 days for cattle and horses, 18/100kg for pig 630-40 days).

Control

- Isolation and treatment of infected animals.
- Provision of separate grooming tools, horse blankets and Feeding utensils and disinfection of the items after use on infected animals.
- Calves should be vaccinated against ringworm with LTF 130 vaccine at 1 month of age. A live vaccine can also be used in horses.

MASTITIS

The term mastitis refers to inflammation of the mammary gland regardless of the cause. It is characterized by physical, chemical and usually bacteriological changes in the milk and by pathological changes in the glandular tissue. The most important changes in the milk include discoloration, the presence of clots and the presence of large numbers of leukocytes.

Etiology: Mastitis can be categorized in to three based on etiology.

- ⇨ Specific mastitis**:** caused by true pathogens that do not require predisposing factors. Such mastitis usually takes place through haematogenous route. Ex-Tuberculosis, Brucellosis, leptospirosis.
- ⇨ Non -specific Mastitis**:** This is caused by agents that are normally found on the surface of the skin and teat. Most commonly staphylococcus and streptococcus are responsible for this type of mastitis. These are not true pathogens and they require predisposing factors to produce mastitis.
- ⇨ Environmental Mastitis**:** This is usually associated with the number of micro organisms in the environment. Ex-E. coli (Coliform mastitis) .

Species Affected: All breeds of dairy cows, goats, sheep, pig, horse are susceptible. High yielding dairy cows are more commonly affected than low yielder. Infection rate is more in successive lactation than the first lactation. Exotic and cross breed cows are more prone to mastitis than our local cattle.

Transmission: There are two sources of infection. These are the udder (organisms that are normal inhabitant in the udder) and contaminated environment. The cutaneous surface of the cow may have many organisms as resident population and form where the organism may have the chance to invade through contamination by handlers. The contamination of milkers' hands, clothes, and milking machine cup by milk from the affected quarter may lead to the spread of the disease to other non-infected teats of cow.

Clinical Findings: Mastitis may clinically be classified as:

- Per acute mastitis**:** This is the most serious form of mastitis, which most often endangers the life of the animal. The affected animal shows a very high temperature, remain off feed and show respiratory distress. The udder is swollen and extremely painful. There is cassation of milk secretion and exudates are often blood stained.
- Acute Mastitis**:** In this case systemic reactions are slight to moderate.The udder becomes swelled and there is change in the milk.
- Sub-acute Mastitis**:** There are variable changes in the milk but practically no changes in the udder tissues. Culture of milk will show presence of pathogenic bacteria.
- Chronic Mastitis**:** This is the terminal stage of the disease. Udder becomes hard due to fibrosis.

Diagnosis: This is based on the following consideration.

- ⇨ Physical examination of the udder; Shape, size and consistency of the udder should be viewed properly. Detailed examination of the teat and teat orifices should be made.
- ⇨ Signs of inflammation (hot, swelling, pain, redness, and loss of function) should be clinically assessed.
- ⇨ Tests for milk abnormalities: there are different testes used to indicate the presence of abnormalities in the milk. Ex- California mastitis test (CMT), White side test etc…
- ⇨ Direct test: Identification and isolation of the organisms from suspected milk.
- ⇨ Cultural examination

Treatment: Isolation of the affected animal should be made from rest of the herd.

- Healthy quarter should be milked first before milking of affected quarter.
- Removal of secretions as much as possible should be attempted before administration of treatment.
- Intra-mammary antibiotic preparations should be used for local treatment.
- Systemic antibiotic therapy may be necessary where systemic reactions are evident.
- Supportive treatment may be required. It includes parentral injection of large quantities of isotonic fluid containing glucose and corticosteroids.
- Drying off Quarters: When the quarter does not respond to treatment the animal is required to be made dried off by using chemical agents. This is done by infusion of 30-60 ml of 3% silver nitrate solution or 20ml of 5% copper sulphate.

Control: A) Reduction of duration of infection. This may be achieved by:

- Treatment of all quarters of all cows at drying off
- Treatment of clinical cases as soon as possible
- Culling of chronic or clinically non-responsive cases

B) Reduction of new infection rate. This may be achieved by:

- Dipping of all teats following milking
- Washing of udder and hand of the milker before and after milking

- Milking order I.e. the healthy, non-infected cows should be milked first and known infected cows should be milkedat least.

COWDRIOSIS; Synonym: heart water

This is a tick-borne disease of cattle, sheep and goats; characterized by fever, nervous signs, edema of the body cavities and diarrhoea.

Etiology: C*awdriaruminantum* is the cause of the disease. It is the rickettisial disease

Occurrence: Heart water affects imported breeds of cattle, sheep and goats and it is limited in its occurrence to Africa, Madagascar and some of the West Indian countries. Generally, young animals have an innate resistance against rickettisial diseases.

Transmission: transmitted by ticks of the genus *Amblyomma*, especially *A. Variegatum*.

Clinical Findings: The incubation period is 1-3 weeks.

- Per acute cases show only high fever, prostration and death with terminal convulsions.
- Acute cases have a course of about 6 days and show the nervous syndrome, which is characteristic of heart water. It comprises ataxia, chewing movements, twitching of the eyelids, circling, aggression, apparent blindness, recumbence, convulsion and death. In less severe cases the principal sign may be diarrhoea. The mortality rate in per acute cases is 100%, in acute cases 50-90% and in mild cases most animals recover.

Necropsy Findings: Standard lesions are ascites, hydrothorax, and hydro pericardium. There may be pulmonary edema and sub-serosal haemorrhages in most cavities and there is spleenomegaly and lymphadenopathy.

Diagnosis: Identification of the rickettisial organisms in a stained, squash preparation of tissue.

- Serological tests like ELISA

Treatment: Tetracycline is the standard treatment and may be used as a prophylactic measure.

Control: eradication of the tick population is not usually a practical option but regular dipping of animals 3-7 days interval is a common practice in enzootic areas.

Vaccination -this is possible through artificial exposure of animals followed by tetracycline administration. Artificial exposure can be effected in several ways, the most common of which is the intravenous, injection of heart water infected blood or ground up infected ticks.

2.2. DISEASES CAUSED BY VIRUSE

Fundamental Characteristics of viruses

Viruses are small, obligate intracellular parasites, which can replicate only in living susceptible cells. They differ fundamentally from other classes of microorganisms in that.

- ⇨ They possess a central core of nucleic acid enclosed within a protein - rich outer coat.
- ⇨ The genome nucleic acid is either DNA or RNA
- ⇨ They lack enzyme systems (no independent growth and multiplication).
- ⇨ not susceptible to antibiotic or other agents that act against the metabolic pathways of microorganisms.

Diagnostic and Control methods of viral Infections

In general, the procedures that are available for the diagnosis of viral diseases depends on Isolation and identification of the causative virus from clinical specimens; Serological tests for the detection and measurement of the specific antibodies and Molecular techniques

Before attempting a diagnosis, a good clinical history is desirable. This should include the nature of infection, clinical signs, lesions and other abnormalities, the stages of the illness, details of the geographical location, breed, age, sex of the animals affected, movements on and off the farm, previous vaccinations and so on. Provisional diagnosis made based on the clinical history and detail of any post-mortem examination, together with the results of microscopical and histological examination. Confirmatory diagnosis can be obtained by isolation of the causal virus (Animal injection, cell and tissue cultures), identification of the virus (cytopathic effects, fluorescent antibody staining, and electron microscopy) and demonstration of a rise in titre of specific antibodies.

Methods of immunization of animals against viral diseases.

There are several methods of immunization animal against viral diseases including the following.

1. Inoculation of live/ non-attenuated virus.
2. Attenuated vaccines and
3. Killed vaccines

Live vaccines stimulate a stronger immunity over a longer period of time but there is possibility of inoculated virus multiplying in the animal body. Inactivated vaccines provide shorter immune reaction but are the safest types of vaccines to use.

PESTE DES PETITIS RUMINANTS (PPR): Synonym: Goat plague

It is an acute or sub-acute viral disease of goat and sheep that characterised by fever, gastroenteritis, necrotic stomatitis, and pneumonia.

Etiology: *Morbilli* virus of the family *paramyxoviridae*

Occurrence: prevalent in Africa and Middle East

Species affected: sheep and goats are highly susceptible to the disease. Sheep are less susceptible than goats; cattle are only subclinically infected .The mortality and morbidity rate is very high in young animals.

Transmission: Secretion and excretions of sick animals are the source of infection. Close contact is important for the spread of the disease. Entry into the animal body occurs through inhalation and ingestion of contaminated feed and water**.**

Clinical sign: fever (40-41^{0}c), dull coat, dry muzzle, congested mucous membrane and depressed appetite. Initially serous nasal discharge latter changed in to mucopurulent nasal discharge. Necrotic stomatitis affects the lower lip, gum, tongue and cheek. Profuse diarrhoea followed by dehydration, hypothermia and death. Broncho pneumonia may also develop.

Post mortem finding: necrotic lesions on the inside part of lower lip and the adjacent gum, cheeks near the commissure and on the ventral surface of the tongue.

Diagnosis: provisional diagnosis based on history of outbreak, clinical findings, and necropsy findings. Confirmatory diagnosis requires isolation and serological identification of the virus.

Differentia diagnosis: CCPP, blue tongue, coccidiosis, mineral poisoning

Treatment: no specific treatment. Administration of antibacterial and anti-parasitic drug to reduce secondary complication.

Control: It is important to apply a national eradication programme, i.e. Mass vaccination

- Complete Prohibition of imports of domestic ruminants; pigs and animal Products from enzootic areas are required.
- If out breaks of disease occurs, it has to be controlled by
    - Slaughter of the infected animals
    - By restriction of stock movements
    - Vaccination of all neighbouring animals

AFRICAN HORSE SICKNESS

It is a highly fatal, infectious disease of horses, mules and donkeys.

Etiology: *orbivirus (*family *reoviridae*). The virus has 9 antigenically distinct serotypes and there is no cross-reaction or protection between the different strains.

Occurrence: The disease is distributed throughout sub-Saharan African countries and Middle East. The incidence of the disease is seasonal because of the seasonal variation in the number of arthropod vector present, particularly *culicoides spp*. Areas most commonly affected are low lying and swampy areas and most cases occur after mid-summer.

Species Affected: natural infection occurs in equine species. The degree of susceptibility are horses> mules > donkeys. Zebras are highly resistant to the disease and serve as reservoir of infection for susceptible hosts.

Transmission: by biting of arthropod insects especially culicoides spp. There is no transmission of the disease through contact with infected animals

Clinical Findings: The incubation period is less than 10 days and the disease has three clinical forms. In all forms of the disease an intermittent fever of 40-41ºc is a characteristic finding.

The three clinical forms are:

***I) Acute (pulmonary) form*:** the most common form in acute outbreaks in susceptible animals. Initially there is fever followed by very laboured breathing and severe paroxysm of coughing. There is a profuse nasal discharge of yellowish serous fluid. Profuse sweating and the horse become very weak, develops a staggery gait and becomes recumbent. Death follows within a few hours

II) Sub acute (Cardiac) form: this most common in horses in endemic areas. This term is characterized by fever but develops more slowly and persists for long period. The most obvious sign is edema in the head region, particularly in the supra orbital fossa, eyelids and the lips, and this may spread to the chest. Auscultation of the heart and lung reveals evidence of hydro pericardium, endocarditis and pulmonary edema. Paralysis of the oesophagus with inability to swallow and regurgitation of food and water through the nose.

III) Horse sickness Fever: this presents no diagnostic signs and may unrecognised except that it usually occurs in endemic areas. Most commonly occur when an existing immunity is partially overcome. The temperature rises to 40.5ºc over a period of 1-3 days but return to normal about three days later.

Necropsy Findings: Acute form- severe hydrothorax, pulmonary edema, moderate ascitis, congested liver. The pharynx, trachea and bronchi are filled with yellowish serous fluid.

In cardiac form-there is marked hydropericardium, endocardial haemorrhage, myocardial degeneration and anasarca, especially of the supra orbital fossa.
Diagnosis: based on Clinical signs, Serological tests: CFT, Serum neutralization, ELISA

Differential diagnosis: Equine infectious pneumonia, Babesiosis, equine infectious anaemia.

Treatment: no treatment; but careful nursing and symptomatic treatment are recommended.

Control: It is very difficult because of the mode of transmission of the disease.

- Restriction of introduction of horses.

- Vaccination**:** Polyvalent vaccine containing all 9 strains or sometimes only 7 strains is effective in the control of the disease. Immunity following vaccination is solid for one year. Therefore, annual vaccination is recommended.

SHEEP POX AND GOAT POX

It is viral infectious disease sheep and goat characterized by fever and generalized pox lesion on the skin.

Etiology: *Capri poxvirus* (family *poxviridae*) affects sheep and goats and animals of all age are susceptible.

Occurrence: These diseases are distributed in different parts of the world including Ethiopia. Sheep pox is the most serious of all the pox diseases in animals, often causing death in 50% of affected animals. Major losses may occur in each new crop of lambs.

Goat pox in sheep is more severe than sheep pox, and lesions occur on the lips and oral mucosa, the teat and udder. The goat poxvirus affords solid protection in sheep against both sheep pox and goat pox but sheep pox vaccine does not protect goats against goat pox.

Species Affected: Sheep pox affects sheep whereas goat pox can affect both sheep and goats. In all cases younger are more susceptible than adults.

Transmission: The usual mode of transmission is contact with the infected animal. The virus may gain entrance through skin wounds and abrasions. The virus is present in the skin lesions and while the affected animals rub their body on other animals, the virus is passed directly to susceptible animals. Aerosol or droplet infection is quite possible.

Clinical Findings: There is an incubation period of 2-14 days. The disease may appear in three clinical forms.

- **Malignant Form:** This form is more common in lambs and kids. There is marked depression and prostration, a high fever and discharge from the eyes and nose. Affected animal may die during this stage before typical pox lesions develop. They lesions commence as papules, then become pustule, vesicular, and finally scabs. Skin lesions appear in unwooled skin and on the buccal, respiratory, digestive and urogenital tract mucosae. The mortality rate in this form may reach 50%.
- **Mild (Benign) form:** This is the common form in adults. Only skin lesionsoccur, particularly under the tail, and there are no systemic signs. The mortality rate is low, usually about 5%.
- **Abortive form:** generalization is rare, mortality is low and affected pregnant animal may abort and the fetus may show pox lesions. Lactating ewe may show the signs of mastitis due to lesions in the udder.

Necropsy Findings: Characteristic papules, pustules, vesicles and scabs are noted on cutaneous surfaces. Lesions may be observed in the mucosa of respiratory and alimentary tract especially on trachea.

Diagnosis: based on the clinical sign (characteristic of a pox disease) and endemic occurrence of the disease as well as Serology like CFT.

Differential Diagnosis: Bluetongue and contagious ecthyma (orf).

Treatment: Antiseptic or antibiotic ointments or lotions may be applied to control secondary bacteria complication.

Control: Strict sanitary measures are to a adopted; Sick animals are to be isolated from healthy one and Vaccination

LUMPY SKIN DISEASE

It is a highly infectious skin disease of cattle that characterized by sudden appearance of nodules on all parts of the skin.

Etiology: *Neethling poxvirus*. The *Neethling virus* has similar cultural characteristics to sheep pox and goat poxviruses. All the three are classified as Capri poxvirus. Moreover, prior to infection with sheep poxvirus confers immunity against the Neethling virus infection.

Species Affected: All ages and breeds of cattle are susceptible to the causative virus except animals recently recovered from an attack, in which case there is a solid immunity lasting for about 3 months.

Occurrence: Different African Countries such as South Africa, Kenya, Sudan, Chad, Niger, Central Africa and Ethiopia are now known to suffer from the disease.

Transmission: The exact mode of transmission is not known, however, the rapid spread of the disease occur byinsect vector.

Clinical Findings: incubation period 2 to 4 weeks.

- In severe cases, an initial rise of temperature with lacrimation, nasal discharge, salivation and lameness.
- Multiple nodules appear suddenly about a week later. They are round and firm, varying from 1 to 4 cm in diameter. They vary in number from a few to hundreds and in most cases are confined to the skin area.
- In severe cases the lesions may also be present in the nostrils, causing respiratory obstruction and snoring.

They may also be in the mouth. Nodules may develop on the conjunctiva, causing severe lacrimation.

- Lymph nodes in the affected area become enlarged and there may be local edema. Pregnant cows may abort.

Necropsy Findings: Granulomatous cutaneous nodules in the affected part of the skin. Similar lesions are present in the mouth, pharynx, trachea, bronchi and stomach and there may be accompanying pneumonia.

Diagnosis: The rapid spread of the disease and the sudden appearance of lumps in the skin after an initial fever make this disease quite unlike any other disease in cattle. Serology - FAT

Treatment: No specific treatment, but prevention of secondary infection is essential. The use of antibiotics or sulphonamides is recommended.

Control: A safe vaccine against the Neethling virus is effective. It is administered to all animals over 6 months of age.

- A freeze-dried, live attenuated virus vaccine is also available.
- Vaccination of cattle with attenuated sheep poxvirus is effective in preventing infection with Neethling virus.

Note: Although the mortality rate is low (less than 10%), the economic loss caused is high due to loss of milk production, damage to hides and loss of body condition during the long course of the disease.

RABIES: Synonyms*:* lyssa; madness

This is an acute viral infection in man and other warm-blooded animals characterized by signs of abnormal behaviour, nervous disturbance, impairment of consciousness, ascending paralysis and death.

Etiology: caused by a virus belonging to the family *Rhabdoviridae* and genus *lyssa virus*. The virus is truly neurotropic and causes lesions only in nervous tissue. It is one of the larger viruses and is relatively fragile.

Occurrence: Rabies occurs in most countries of the world except the island countries that are able to exclude it by rigid quarantine measures or prohibition of the entry of dogs.

Species Affected: All warm-blooded animals are susceptible and there is no difference in susceptibility with relation to age of the animals. Susceptibility varies in different species. Animals like dog, fox, cat wolf, rat and vampire bat are extremely susceptible in the tropical areas of the world. Cattle, goat and sheep are moderately susceptible. Equine are also susceptible for the disease and they are considered as dead end host for the rabies virus. Man and birds are susceptible to the disease.

In general, rabies in animals exist in two epidemiological types:

Urban type: where the transmission takes place through dogs.

Sylvatic type: where the disease is transmitted through wild life ex-fox, wolf, hyena and vampire bat.

Transmission: Transmission of rabies under natural condition is commonly by bite of rabid animals usually carnivorous animals. Rabies virus can penetrate the mucous membrane of eyes, nose and mouth and thus aerosol infection is possible in a rabies laboratory by accident.

The ability of the virus to reach the central nervous system depends on various factors. They are as follows:

- ⇨ Age of the animal
- ⇨ Distance of bite from the CNS is an important factor in reaching the virus in the brain.
- ⇨ Virulence of the virus; Concentration of the virus

Clinical Findings: Rabies has got two types of clinical manifestations:*Furious form* **and** dumb *(paralytic*) form

The incubation period of the disease varies considerably from weeks to several months depending on the site and severity of wound inflicted by rabid animals and the amount of virus deposited at the site.

Furious Form: It refers to syndrome in that excitation is the predominant changes and it can be divided into stage of melancholy and stage of excitation.

Stage of Melancholy: Here, there is change in the behaviours of the animal. The dog may show the tendency to bite either inanimate or animate objects. Dogs those are under restraint bite nearby objects. But, set free dogs show the tendency to bite other animals and human beings that comes across their way. Infected dog remains in unusual alert condition. They respond very sharply to any stimulus. They may show imaginary fly catching stance. They will bark with no cause behind that. There is no deviation in appetite in the initial phase, but later on it becomes changed. They will have the urge to chew or bite or eat non-edible substances like stone, mud, grass or even their own faces. Gradually, signs of excitability increases. The pupils get dilated and there is altered facial expression. Appetite is entirely suppressed but the animal may lick water and attempt to drink water but due to paralysis of pharyngeal and laryngeal muscles does not succeed to drink water. Saliva hangs from mouth in long stand. The period lasts for 1-3 days.

Stage of excitement: In this stage the excitability and irritability increases and dogs become very much aggressive. This period may last for 1-7 days. At the onset dog may hide in dark place due to photophobia. There is development of change in the bark-which is very characteristic. The animal is unable to swallow. There is drooling of saliva. The change in voice is due to paralysis of the vocal cord. If these animals are not confined they will wonder aimlessly and bite animals or persons who will fall in their way. After some time the affected animals will show emaciation and exhaustion. There will be incoordination and muscle tremors.

The bitch may show the sign of heat and accept the male.

The dog will loose its ability to bark; the lower jaw will hang, tongue will protrude and head will drop down. The dog will develop dyspnoea, ascending paralysis, coma and death. The total episode may last as long as 10 days.

Dumb (paralytic) Form: In this form, there is paralysis of the lower jaw, tongue, larynx and hind-quarters. The dogs are not capable to bite but their saliva remains infective, throat muscles are paralysed.

There is hanging of the jaw due to paralysis of the jaw muscle and the dog is unable to close his mouth. In the terminal stage of the disease dogs show progressive weakness and paralysis that causes them to stagger or fall. Ultimately there is coma and death.

Necropsy Findings: There is no gross lesion at necropsy. However, during histopathological preparation of nervous tissue (hippocampus) reveals the presence of cytoplasmic inclusion bodies, which are called **Negri bodies**.

Diagnosis: The diagnosis of rabies is one of the most difficult and important duties that a veterinarian is called upon to perform. Since in most cases there is a probability of human exposure, failure to diagnose the disease may place human life in jeopardy. Confirmation depends on careful laboratory examination of fresh brain.

Differential Diagnosis: Ac lead poisoning in cattle, Listeriosis- in sheep and cattle, Enterotoxaemia- In sheep

Treatment: No specific treatment but immediately after exposure irrigation of the wound with antiseptic solution (soap) may prevent the establishment of infection.

Post-exposure vaccination is unlikely to be of value in animals, as death usually occurs before appreciable immunity had to develop.

Control: reduce the population of dogs by destruction of street dogs, castration and spaying vaccination of all cats and dogs and keeping farm animals indoors.

BLUE TONGUE

Bluetongue is a disease of sheep and occasionally cattle, characterized by catarrhal stomatitis, rhinitis and enteritis and lameness due to inflammation of the coronary bands and sensitive lamina of the feet.

Etiology: caused by *orbi virus* of the family *Reo viridae*. The virus is resistant to decomposition, desiccation and against antiseptic agents. The virus is destroyed by 3% NaOH solution.

Occurrence: Bluetongue is arthropod borne viral disease. The infection is widespread on the African continent and also to many other parts of the world.

Species Affected: It is basically a disease of sheep but amongst sheep susceptibility varies in age; young sheep within the age group of one year are more prone to infection. Suckling lambs are relatively resistant. Goats are relatively resistant

Transmission: spread is through blood sucking midges of the genus culicoides. Ticks and mosquitoes also transmit the virus. The disease is more prevalent in late summer and early autumn that provide conducive environment for the multiplication of the vectors.

Clinical Findings: Incubation period varies from 1-10 days.

The disease is characterized by high fever persisting for 5-6 days with progressive reddening of the buccal mucous membrane. It has acute, sub acute and abortive form.

Acute form: is characterized by fever, nasal discharge, salivation and lacrymation. Swelling of the tongue, gum and lips followed by ulceration is evident. There may be extensive necrosis of the dental pad. Skin of other areas like flank, neck, back, etc may crack.

There is cyanstic and bluish appearance of the tongue. Udder may be swollen and teat may show signs of ulceration. Coronary band may become swollen and it may crack Hoof may become separated leading to lameness,
Rapid shallow respiration, pneumonia & muscular weakness may be seen.

Abortive formis characterized by abortion and deformities. Deformed calves may remain normal after birth.

Sub acute: noted in cattle and generally passed unnoticed.

Necropsy Findings: Characteristic lesions are observed in the mouth and tongue. On the mouth there is hyperaemia, oedema, cyanosis, erosion, haemorrhage and ulceration of the oral mucosa.

The tongue becomes blue and gangrenous. Signs of hyperaemia and necrosis on the skeletal and cardiac muscles and pneumonic changes on the lungs are seen.

Diagnosis: Presumptive diagnose is based on history, clinical findings (oedematous swelling and cyanosis), seasonal occurrence, absence of spread by direct contact.

Confirmative diagnosis is based on animal inoculation, isolation of the virus and serological tests (CFT, AGID...)
Differential Diagnosis: FMD, MCF**,** photosensitization.

Treatment: There is no treatment against the virus. Localized lesions may be treated with topical antibiotic solution to accelerate rate of healing. Affected animals should be housed and protected from hot sun.

Control: Recovered animals (sheep) are immune for 6 months.

- ⇨ Reduce vector population by applying insecticides and good water
- ⇨ Managements required.
- ⇨ Infected animals should be isolated.
- ⇨ Vaccination (attenuated live vaccine)

RIFT VALLEY FEVER (RVF)

It is an acute, febrile disease of cattle, sheep and man characterized by hepatitis and high mortality in lambs and calves, in adult sheep and in cattle abortion and in man influenza like disease.

Etiology: p*hlebo virus* (family *Bunyeviridae*).

Species affected: Cattle, sheep, camels, buffalo, monkeys and man are highly susceptible and goats moderately susceptible.

Occurrence: is conferred to Africa continent but it has great potential for spread to other countries. Losses are due mainly to deaths in young lambs and calves, although there may be a high incidence of abortions, and some deaths, in adult sheep and cattle.

Transmission: is by biting flies, chiefly mosquitoes

Clinical findings: In lambs and calves after an incubation period of about 12 hours there is sudden outset of high fever and incoordination followed by collapse and sudden death within 36 hours.

In adult sheep and cattle, abortion is the common sign. In fatal cases sudden death is preceded by high fever for 1-2 days. Goats show a febrile reaction but few other clinical signs.

Necropsy Findings: Extensive hepatic necrosis is the characteristic lesion. Venous congestion and petechiation in the heart, lymph nodes and alimentary tract.

Diagnosis: The hepatic lesions are characteristic; severe leukopoenia is a common finding. Serological tests like ELISA, hemagglutination, CFT and serum neutralization are used.

Differential Diagnoses: Blue tongue (in sheep), Ephemeral fever (in cattle), Enterotoxaemia and other causes of abortion.

Treatment: no specific treatment.

Control: - Prevent introduction of infected animals and human beings to free areas.

- ➢ In endemic areas mosquito control is important to reduce the spread of disease
- ➢ Both killed and living attenuated virus vaccines are available.

BOVINE VIRAL DIARRHOEA (MUCOSAL DISEASE) (BVD)

It is a sub acute, acute or in apparent contagious disease that characterized by high rise of body temperature, diarrhoea and erosion on the mouth, oesophagus, rumen, abomasums and intestines.
Etiology: caused by a virus belongs to the genus *pest virus* and family *Toga viridae.*

Species Affected: Cattle are the only species that develop mucosal disease but pig and sheep can also be infected

Occurrence: The disease has nearly a worldwide distribution

Transmission: Virus is present in secretions and excretions of affected cattle. The disease often rapidly spread by direct or indirect contact with infected animal. In crowded feedlot and transport vehicles, virus from nasal and oral secretions easily spread from affected cattle to susceptible animals. Contaminated feed and water are the important agents of transmission of virus. Urine and nasal discharges may act as a source of infection.

Clinical Findings: The clinical manifestation may be grouped as

Acute (epidemic) form: This form is serious in nature and characterized by high fever, anorexia, depression, polypnea, tachycardia , and polydypsia. Nasal discharges are mucoid to mucopurulent in nature. Erosion may be noted under the crest. There may be signs of conjunctivitis accompanied with mucopurulent ocular discharges. Profuse foul smelling watery faces and may contains mucus and blood. Oral lesions appear 1 to 3 days following diarrhoea. There is hyperemia and ulceration of oral mucosa, tongue, palate and gum. Perfuse salivation will be noted at this stage. There is abortion. Fetus is abnormal and malformed. Finally, animals may die due to septicaemia and severe dehydration.
Mild form**:** characterized by fever of short duration, temporary loss of milk yield, transient diarrhoea and infrequent mouth lesions. Anorexia, nasal discharge and enlargement of superficial lymph nodes may be the clinical outcome.

Sub acute (chronic) form*:* There is retarded growth, loss of body weight, emaciation, rough skin coat and intermittent diarrhoea. Chronic buccal lesions and chronic bloat may be noted.

Necropsy Findings: Lesions are located in the oral mucosa, oesophagus, rumen, reticulum, omasum and intestines. Lesions are hyperaemia, erosive, ulcerative, and necrosis in nature. There is destruction of payer's patches. There is destruction of lymph nodes.

Diagnosis: made based on typical clinical findings and characteristic gross and microscopic lesions. High morbidity and low mortality rate and leucopoenia are the other aids in diagnosis. Confirmatory diagnosis is by isolation of the virus and serology like ELISA, CFT etc.

Differential Diagnosis: MCF**,** Rinderpest, IBR, Johne's disease

Treatment: There is no treatment against the virus. But supportive therapy is indicated to safeguard the life of the animal.

- ⇨ To correct dehydration balanced electrolyte and fluid therapy should be restored.
- ⇨ Broad-spectrum antibiotics to control secondary bacterial complication

Control: All the affected animals should be isolated during an outbreak. All sorts of hygienic and sanitary measures are to be adopted to inhibit further spread of the disease. Vaccination of animals by using modified live virus vaccine after 6 months of age.

FOOT AND MOUTH DISEASE (FMD): Synonym: Aphthous fever

It is an extremely contagious acute disease of all cloven-footed animals that characterized by fever and vesicular eruption in the epithelium of buccal cavity, tongue, muzzle, feet, teat and udder.

Etiology: caused by *picorna virus* group (genus *Aphtho virus*). At least 7 immunologically distinct serotypes of the virus have been identified. These are A, O, C, SAT-1, SAT-2, SAT-3 and Asia-1. The virus is resistant to various external agents including common disinfectant. NaoH, formalin (1-2%), Na_2Co_3 (4%) have ability to destroy the virus within few minutes.

Occurrence: The disease is endemic in South America, central Europe and central African countries notably in Ethiopia and Tanzania.

Species Affected: Cattle are more susceptible. A wide range of hosts like sheep, goat, pig and camel are susceptible to FMD. Dog, cat and man may occasionally be infected after close contact with infected host.

Transmission: The disease spread at an extremely rapid rate through direct contact with infected animals. In tropical countries, spread is through ingestion of contaminated feed and water and inhalation of infected droplets. All the fomites like clothes, harness, beddings, straws, hay etc may get infected and therefore act as a source of infection. All the secretion and excretions like urine, milk, faces and saliva remain infective. Cattle may remain carrier following recovery.

Clinical Findings: the morbidity is 100% but mortality is less in indigenous cattle and comparatively more in cross and pure breed cattle. The disease has an incubation period of 2-8 days. The signs are drooling and vesicles on the nares, in the buccal cavity and between the claws. Before the visible sign of illness, animals may show dullness, in appetence, fever and shivering followed by smacking of the lips, drooling and shaking or kicking of the feet. After vesicle formation there is pronounced salivation and lameness. Pregnant animals may abort. Hoof deformation may result in permanent lameness. Mammary gland involvement may result in mastitis and permanent impairment of milk production.

Necropsy Findings: The diagnostic lesions are vesicles or blisters. They may be found on the tongue, dental pad, gum, cheek, hard and soft palate, lips nostrils, muzzle, coronary bands, teat and udder, as well as in the myocardium (degenerative change) and in skeletal muscles.

Differential Diagnosis: MCF, BVD**,** Rinderpest, vesicular stomatitis, blue tongue.

Treatment: There is no specific treatment. Symptomatic treatments may be rendered depending on clinical manifestations. Antiseptic solution like potassium permanganate or sodium bicarbonate may be applied over mouth lesions. Similarly antiseptic or antibiotics may be given on feet lesions and lesions on mammary tissue.

Control: restrict animal movements

- ⇨ Vaccination of all animals of an area/village is to be done at one time.
- ⇨ A footbath or truck bath may be made at the entrance of village or farm.
- ⇨ Always purchase fodder from a place where FMD has not been recorded for a period of 6 months or so.
- ⇨ Try to isolate and confine the affected animals immediately after detection.
- ⇨ Calves should not be allowed to suckle affected mothers and they should not be fed with milk from affected animals.