DEBRE MARKOS UNIVERSITY
COLLEGE OF AGRICULTURE AND NATURAL RESOURCE
PROGRAM: *ANIMAL SCIENCE*

Course Title: ***Animal Health and Disease Control (Vetm 3103)*** **3 credit hours**

Chapter one

1. General Concept of disease (infection) and its importance

Disease is defined as an alteration of the state of the body, or of some of its organs, which interrupts the proper performance of the body functions. According to the causative agent diseases can be classified in to: -

I. Infectious disease: The word "infection" is derived from the Latin "inficere", meaning "to put into" or "to dip into, to stain". In the most exact sense, infection implies the presence of microorganisms in or on the body of the host. Therefore an infectious disease is one that is caused by the presence in or on an animal body of a foreign living organism, which creates a disturbance leading to the development of signs of illness.

II. Non-infectious disease: It is a disease, which is caused by non-living agents or factors like injury, plant or animal poison, cold, excessive heat or faulty nutrition.

1.1. Basic terminologies used to study Infectious Diseases.

Infectious diseases are those diseases caused by organisms (such as bacteria, viruses, rickettsiae, fungi and protozoa) and which are capable of passing from infected to healthy one under favourable conditions. In other words, infectious diseases are the result of the invasion of a host by a pathogenic organism.

Contagious disease is a disease that is caused by an organism, which is readily transmitted, from one individual to another by direct or indirect contact.

Non-contagious disease is one that does not transmitted by contact.

The term infectious and contagious diseases are used interchangeable. It is best, however, to limit the terms. Infectious defines the cause of the disease and contagious indicates the method of

disease transmission. All contagious diseases are infectious but all infectious diseases are not contagious.

Infection: invasion of pathogenic microorganisms into body tissues so that the tissues are affected and altered.

- **Inapparent (Silent)/ Sub-clinical infection** - This is infection of a susceptible host without showing clinical signs. The inapparently infected animal poses a considerable problem to the disease controller because it is impossible to detect without auxiliary diagnostic aids. And also causes great loss of productivity.
- **Clinical Infection**: - This type of infection produces clinical sign or disease.

1.2. Host and agent interaction/ relationships

The relationships between infection and disease are frequently dynamic in nature. They centre on the "balance" that can be achieved between the resistance mechanism of the host and the infectivity and virulence of the agent. Disease outbreaks caused by the introduction of an agent into a susceptible host population which has not been previously exposed to that agent normally result in a disease of high pathogenicity with commensurate severe losses in the host population. Such a process is actually detrimental to the agent's survival, since by killing off the host population it adversely affects both its ability to reproduce and its chances of gaining access to new susceptible hosts. An agent can therefore improve its chances of survival by increasing its infectivity and decreasing its pathogenicity, and some agents have a natural tendency to do this under certain circumstances. Since a commensal or parasitic relationship confers no benefits to the hosts, they tend to develop means of resisting infection by disease agents. While the agents, in order to survive, develop methods of circumventing the hosts' defences. Disease agents normally have much shorter generation intervals and can multiply much more rapidly than their hosts, and therefore tend to evolve much quicker. This rapid evolution usually enables the agents to keep comfortably ahead of the hosts' defence mechanisms.

There are many mechanisms by which infectious agents can avoid or overcome the defences of the host. The two mechanisms whose consequences are of particular importance in the field of livestock disease control are the carrier state and antigenic variation.

Creation of the carrier state:- The term "carrier" is used to describe an individual that is infected by a disease agent and is capable of disseminating that disease agent but shows no sign of clinical disease. Three types of carrier state are recognized: **The true carrier**, which is an infected individual capable of disseminating the infectious agent but which never exhibits clinical signs of disease. True carriers occur in various diseases, including salmonellosis. **The incubatory carrier**, which is an infected individual capable of disseminating the infectious agent while the disease is still in the incubatory stage. In foot-and-mouth disease, for instance, infected animals are most infectious 12 to 24 hours before the clinical signs of the disease appear. **The convalescent carrier**, which is an individual that continues to disseminate the infectious agent after the clinical signs of the disease have disappeared. Convalescent carriers occur in such diseases as contagious bovine pleuropneumonia.

Other terms used to further define host/agent relationships include: **Incubation period**, which is the period of time that elapses from the infection of the host by the agent to the appearance of clinical symptoms. **Prepatent period**, which is the period between the infection of the host by the agent and the detection of the agent in the tissues or secretions of the host. **Period of communicability**, which is the period of time during which an infected host remains capable of transmitting the infective agent.

1.3. Factors influencing the occurrence of diseases (disease determinates)

A determinant is any factor or variable that can affect the frequency with which a disease occurs in a population. Determinants can be broadly classified as being either intrinsic or extrinsic in nature. Intrinsic determinants are physical or physiological characteristics of the host or disease agent (or intermediate host or vector, if present) which are generally determined genetically. Extrinsic determinants are normally associated with some form of environmental influence on the host or disease agent (or intermediate host or vector, if present). They may also include interventions made by man into the disease process by the use of drugs, vaccines, dips, movement controls and quarantines. For the establishment of infectious diseases the interaction of host, agent and Environment plays a vital role. For example environmental mastitis involves the interaction between Escherichia coli or streptococcus uberis (the agent), milking machine

faults, and poor hygiene resulting from inadequate bedding, drainage and cleaning of passage ways (the Environment). In addition cows (the hosts) are most susceptible in early lactation.

Factors associated with the host (Intrinsic factors)

The main intrinsic determinants in the host are species, breed, age and sex.

Species susceptibilities and natural reservoirs: Most disease agents are capable of infecting a range of animal species, both vertebrate and invertebrate. The severity of the disease resulting from such infections may, however, vary between the species concerned. While certain host species may be refractory to infection with certain disease agents, e.g. equines to the foot-and-mouth disease virus, very few disease agents are in fact restricted to one host species. The multi-species susceptibility to disease agents is particularly important if the species concerned are able to maintain the disease agent within their populations i.e. to function as natural reservoirs of infection. The failure of programmes aimed at controlling a certain disease in one species has often been blamed on the presence of natural reservoir species, because they can reintroduce the infectious agent. When investigating the potential of a certain species to act as a natural reservoir of a particular disease agent, and the implications on disease control policy, the following considerations need to be borne in mind:

Infection with the disease agent:- Although it may be possible to infect a certain host species with a disease agent under laboratory conditions, this may only be achievable by using a method of transmission that does not occur naturally. If this is the case, that particular host species is unlikely to play a significant role in the epidemiology of the disease.

Ability of a host species to maintain a disease agent:- It may prove possible to demonstrate that a particular host species can be infected by a certain disease agent and that infection can be accomplished by a natural means of transmission. A further question then needs to be asked, namely, is that species capable of maintaining the agent within its populations for significant periods of time? If this is not the case, then although that particular species may be involved in

the localized spread of the disease agent during an outbreak, it will not serve as a continuous source of infection.

Transmission from the natural reservoir:- Even if a species can function as a natural reservoir for a particular disease agent, transmission from that reservoir to domestic livestock may only occur rarely and in certain, clearly defined circumstances. If this is the case, the reservoir species is unlikely to cause a major problem in the initial control of the disease in question. However, when the frequency of occurrence of the disease has been reduced to a low level, and eradication of the disease becomes a possibility, the implications of the presence of reservoir host species for the success of the proposed eradication programme may have to be re-assessed.

Breed susceptibilities: Within a host species, wide ranges of susceptibility to a particular disease are often observed between different breeds. In Africa, for example, certain breeds of cattle, horses, sheep and goats are more tolerant of trypanosomiasis than others. Bostaurus breeds of cattle are generally more susceptible to ticks and tick-borne diseases than Bosindicus. It is important, however, to distinguish between the differences in susceptibility that are genuinely related to breed or species and the differences that may arise as a result of previous exposure to infection. Breeding for disease resistance is probably most applicable as a disease control option in instances where particular disease agents are ubiquitous in the environment, or of non-infectious diseases caused by multi-causal determinants, or where other methods of control have proved unsatisfactory. Differences in species or breed susceptibility to disease must be taken into account when introducing new breeds or species into new environments. The new breed or species may be exposed to disease agents to which the new breed or species is highly susceptible. Conversely, the imported breed or species may itself introduce a new disease agent to which it is resistant but to which local breeds or species are susceptible. This factor has become the cause for much concern in recent years given the rapid development of international transport facilities whereby livestock and their products can easily be conveyed from one part of the world to another.

Age susceptibilities: Differences in susceptibility to disease are often seen between different age groups. For example, young animals are generally less susceptible to tick-borne diseases than older animals. There is, however, often a problem in distinguishing between true age resistance

in young animals and passive resistance occasioned by the transfer of maternal antibodies via the placenta or in the colostrum.

Sex associations in disease: In these associations the clinical signs of disease are associated with sexual attributes, as in the case of diseases of the reproductive tract, rather than with the fact that males may be more susceptible than females or vice versa.

Factors associated with disease causing agents (Extrinsic factors)

Agents associated with disease can be categorized into two broad groups:"Living" agents, such as viruses, bacteria, rickettsia, protozoa, helminths, arthropods etc. "Non-living" agents, such as heat and cold, water, nutrients, toxic substances etc. In instances of infectious disease, the presence or absence of the etiological agent is the main determining factor in the epidemiology of the disease. Obviously, disease cannot occur in the absence of the agent, but, conversely, disease need not always result from the presence of the agent. This leads us to the important epidemiological distinction between infection and disease

Infectivity, virulence and pathogenicity

Whether infection takes place or not may depend on a whole range of determinants, both intrinsic and extrinsic, which affect the host and the agent (and the intermediate host or the vector, if present). **Infectivity** is a measure of the ability of a disease agent to establish itself in the host. This term can be used qualitatively, when an agent is referred to as being of low, medium or high infectivity, or quantitatively. Having become infected, the host may or may not become diseased, and this is again determined by a range of intrinsic and extrinsic determinants affecting the agent and the host. **Two terms - virulence and pathogenicity** - are often used to describe the ability of the agent to cause disease. **Virulence** can be defined as a measure of the severity of a disease caused by a specified agent. In its strict sense, virulence is a laboratory term and is used to measure the varying ability of disease agents to produce disease under controlled conditions. It is often quantified by a statistic known as LD_{50}. **Pathogenicity** is an epidemiological term used to describe the ability of a particular disease agent of known virulence to produce disease in a range of hosts under a range of environmental conditions.

Environmental Factors (Extrinsic factors)

Extrinsic determinants of disease are important in epidemiology in that they can have effects on the host, on the agent, and on the interactions between the host and the agent. They can also affect any intermediate hosts or vectors involved in the transmission of a disease, and thus determine the type and extent of the disease transmission taking place. There are three major extrinsic determinants. The first two are climate and soils, which, by interacting in a variety of ways, affect the environment of the host, the agent, and the intermediate host or vector, if they are present. The third major factor is man, who, uniquely among animals, has the ability to modify both the environment in which he lives and the environment in which he keeps his livestock.

Physical factors: Climate (macroclimate: terrestrial and microclimate: biological)

Climate: When considering climate as a determinant of disease, a distinction is normally made between the macroclimate or weather, and the microclimate. The term microclimate refers to the actual climatic conditions prevailing in the specific, restricted environment where the host, agent, vector or intermediate host actually lives. While man is as yet largely incapable of deliberately manipulating macroclimates, he can control and manipulate microclimates to some extent.

Macroclimates: A large number of different factors combine to make up the microclimate. Some of these factors (heat, cold, rainfall, wind, humidity etc) can act as disease agents in their own right, either individually or in combinations. As such they can cause disease in young and newborn animals which are particularly sensitive to heat, cold and dehydration. In older animals they tend to act more as indirect determinants of disease in that they can produce either alone or in combinations with other managemental and nutritional determinants - "stress" conditions in the host, which may lower its resistance both to infection and, if infection takes place, to disease. Microclimates. While macroclimates can have a direct effect on microclimates, the study of macroclimates alone can frequently be misleading in achieving an understanding of the epidemiology of a disease. Regions where existing macroclimatic conditions might be thought unsuitable for the transmission of a disease may, in fact, contain limited areas where the microclimatic conditions are suitable for the survival of the disease agent and its vector or intermediate host. (An example may be a water hole or an irrigated pasture in an arid

environment). Such areas often provide enhanced conditions for disease transmission, since the host and the agent (and the vector or intermediate host, if they exist) are in close contact, the transmission of disease can be effected rapidly and easily.

Soils: By interacting with climate, soils determine vegetation and the environment in which the livestock are kept. The main effect of vegetation is on nutrition. Soils therefore act indirectly as determinants of disease by causing starvation, if there is little or no vegetation, or nutritional imbalances such as protein, energy, vitamin or mineral deficiencies. Malnutrition can be the direct cause of disease, or it can stress the host and thus increase its susceptibility to infection and disease from other sources. Soils can also have an effect on the ability of the agent to survive in the environment, through such factors as water logging, pH etc.

Man: Man is often able to create favourable, artificial microclimates for livestock rearing by providing such inputs as housing, water supplies, irrigation etc. Unfortunately, this often results in the creation of conditions favourable for the survival of disease agents and their intermediate hosts or vectors. This means that, by altering the environment, man can alter the determinants of the diseases present in that environment. The changes in determinants will favour some diseases and be detrimental to others. Man is also able to interfere directly in the disease process through the use of drugs, vaccines, movement controls, quarantines etc.

Husbandry: Housing, Diet and Management (reading assignment)

1.4. Patterns of Disease Occurrence

Endemic Occurrence - It is a special form of infectious disease occurrence in which disease is retained for a long time in some locality and affecting a large number of animals of a particular species or breeds. So, ' endemic' is used to describe a constant presence of a disease in a population.

Epidemic Occurrence- It is the occurrence of an infectious or non- infectious disease to a level in excess of the expected (i.e. endemic) level

Pandemic Occurrence - It is a widespread epidemic that usually affects a large proportion of the population. Many countries can be affected.

Sporadic Occurrence - It occurs irregularly and haphazardly. This implies that appropriate circumstances have occurred locally producing small, localized outbreaks.

1.5. Methods of disease transmission

Ascertaining the means by which disease agents are transmitted is a major objective since once the mechanisms by which a particular disease is transmitted are understood, it may become possible to introduce measures to prevent transmission from taking place. There are three main ways by which disease agents are transmitted from infected to susceptible hosts. An agent may be transmitted through contact between infected and susceptible individuals, or it may be conveyed between these individuals by means of an inanimate object or via another animal serving as a vector or intermediate host. These methods of transmission are not mutually exclusive; the same disease agent may be transmitted by more than one of the following ways.

Contact transmission:- In contact transmissions the agent is conveyed between hosts through direct physical contact, as in the case of venereally transmitted diseases such as vibriosis or trichomoniasis, or through indirect contact. In cases of indirect contact the agent is normally contained in the excretions, secretions or exhalations of the infected host i.e. in the faeces, urine, milk, saliva, placenta and placental fluids, or as aerosols or droplets in the breath. Susceptible hosts contract the infection either by direct exposure to these or through exposure to substances contaminated by them. Diseases spread in this fashion include FMD, Newcastle disease, and CBPP.

Contact transmissions can be further distinguished according to whether they occur horizontally between individuals of the same generation or vertically between individuals of different generations. In vertical transmissions the infectious agent is usually passed from dam to offspring either in the uterus or through the colostrum. Main factors determining whether or not transmission takes place in contact-transmitted diseases are:

- The ability of the agent to survive in the environment. Foot-and-mouth disease can spread between widely separated stocks.
- The extent of the contact that occurs between infected and susceptible individuals of the host populations and their mobility within these populations. The control of livestock movements is, therefore, a vital factor in the control of contact-transmitted diseases

which, in Africa, normally occur more frequently during the dry season when livestock movements are at their highest.

Vehicular transmission:- In vehicular transmission the agent is transferred between infected and susceptible hosts by means of an inanimate substance or object (sometimes called fomite), such as water, foodstuffs, bedding materials, veterinary equipment and pharmaceuticals, or on the skin, hair or mouthparts of animals. In contrast to indirect transmission, the survival time of the agent in or on the vehicle is usually prolonged. This means, in effect, that vehicular transmission can take place over greater distances and over longer time periods. Hygiene, disinfection and control over the distribution of likely vehicles of transmission are important factors in the control of vehically transmitted diseases. Certain agents may take the opportunity to reproduce themselves during vehicular transmission. This occurs in the transmission of food-borne bacteria, such as salmonella and coliforms, and underlines the importance of strict hygiene in the handling of foodstuffs and livestock feeds, since a small initial contamination may eventually result in the gross contamination of a whole batch of food or feed.

Vectors and intermediate hosts:- Confusion frequently arises between the terms "vector", "intermediate host" and "definitive host". The latter two terms are essentially parasitological terms and describe the different types of hosts that are biologically necessary in the lives of agents with relatively complicated life cycles. A vector is an invertebrate animal that actively transmits an infectious agent between infected and susceptible vertebrates. Essentially, vectors can transmit infectious agents in two ways. They can serve as a vehicle whereby the infectious agent is conveyed from one host to another without undergoing a stage of development or multiplication. This is known as mechanical transmission. In mechanical transmission the agent is carried on the skin or mouthparts of the vector from an infected to a susceptible host. The survival time of the agent in or on the vector is usually short, and as a result the transmission of the agent has to be accomplished rapidly. Alternatively, the infectious agent can undergo some stage of development or multiplication in the vector - this is known as biological transmission - and in this case the vector is serving either as an intermediate or definitive host, depending on which stage of the development cycle of the agent takes place within it. Vertebrate intermediate hosts play the same role in the transmission of their disease agents as biological vectors. In biological transmission, since the agent develops in the vector, a period of time elapses between

the acquisition of the infectious agent by the vector and its becoming infective. Once it has become infective, the vector may remain so, normally for a considerable period if not the rest of its life. This provides more than a single opportunity for disease transmission.

In addition, vectors may be able to pass the agent on to their own offspring transovarially. Transovarial transmission enables an infectious agent to be maintained in a vector population through many generations without that population having to be reinfected, and, as such, the vector population remains a continuous source of risk. Arthropod vectors that undergo metamorphosis have the capacity to pass an agent from one developmental stage to the next. This is known as transtadial transmission. Usually in transtadial transmission, one developmental stage becomes infected with the disease agent and the following stage transmits it. If different developmental stages feed on different host species, transtadial transmission can provide a mechanism for an inter-species transmission of disease agents.

1.6. Disease Control Measures

Control: is the reduction of the morbidity and mortality from diseases, and is a general term embracing all measures intended to interfere with the unrestrained occurrence of disease, whatever its cause.

Methods of Control

1.**Quarantine** - It is the isolation of animals that are either infected or suspected of being or non-infected animals that are at risk. Quarantine is used to isolate animals when they are imported from countries where exotic diseases are endemic. In this case suspected animals are isolated until infection is either confirmed or discounted.

2. **Creation of unfavourable environment:** this may be related to the livestock or the environment. For instance tolerance- only animals that are resistant to the organism may be kept in the area. E.g. N`dama cattle tolerant to trypanosomosis.

3. **Disinfection-** it involves the destruction of pathogenic organisms on inanimate objects, usually by physical or chemical means. All disinfectants are effective against the vegetative forms of organisms but not necessarily against the spore form of the organisms.

4. **Immunization** - It is the process by which antibody is produced or administered for the prevention or treatment of disease. Generally, there are two types of immunity.

a) Natural immunity - It is attributed to antibodies present or appearing without obvious external stimulus.
b) Acquired immunity- is that which an animal develops or receives at any time after birth.

- Active immunization-follows actual injection and also artificial stimulation with living or attenuated microorganisms, dead organisms or their components or products.
- Passive immunization- is the result of natural transfer of antibodies from the mother to the fetus or newborn animal or the injection of antitoxins and other antisera.

5. **Stamping out** – It is killing animals that are affected, suspected of being affected in the herd and comes in a direct or indirect contact with affected animals. All susceptible animals, vaccinated or unvaccinated, on an infected premises should be killed and the carcasses destroyed by burning or burial, or by any other method which will eliminate the spread of infection through the carcasses or products of the animals killed.

6. **Control of vectors:** vectors are contributory factors for disease occurrence. They are prevented from invading or eliminating by the application of suitable control measures. For vector control you can use insecticide or acaricide.

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.

CHAPTER THREE

3. POULTRY DISEASES

I-BACTERIAL DISEASES OF POULTRY

AVIAN PASTEURELOSIS; Synonyms: fowl cholera

It is a peracute infectious disease that affect all poultry species and wild birds.

Etiology: caused by *pasteurella multocida*. It is a gram-negative bacteria.

Occurrence: it has worldwide distribution and notifiable in many countries. Adult birds are more frequently affected than younger stock.

Species affected: all species of bird are susceptible

Transmission: source of infection include carrier birds, clinically diseased poultry and their excretion and carcass of birds, which have died of the infection. It transmitted by inhalation and ingestion of contaminated feed and water. Infection also occur through conjunctival and wound

Clinical sign: the disease may occur in many forms: Per acute, acute, chronic and localized disease.

In per acute form*:* no premonitory sign, large numbers of birds in the flock found dead but in a good body condition.

In acute form*:* marked depression, anorexia mucus discharge from the orifices and fetid diarrhoea may be seen.

In chronic form depression, conjunctivitis, dyspnoea as well as lameness, torticolis and / or swelling of the wattles.

Post mortem finding: carcass congestion, petechation throughout the viscera, Necrotic foci in the liver and Oedema.

Diagnosis: based on history of the disease, clinical sign and gross lesion are helpful. Isolation of the organisms

Treatment: trimethoprim or sulphadiazine preparation in the feed and water Injection of affected individual with long acting oxy tetracycline.

Control: to eradicate infection, it is necessary to depopulate, to cleanse and disinfect building and equipments.

-Vaccination of animal also important.

SALMONELLOSIS

Salmonella pullorum and *salmonella galinarum* are highly host adapted to the chicken. Salmonella have major public health significance because contaminated food can be a source of infection to man.

FOWL TYFOID

Caused by *S. gallinarum*. It is egg-transmitted and produce lesion in chicks. Mortality at all age usually is high.

Transmission: by ingestion of contaminated food & water. Recovered birds remain carrier for long period of time.

Clinical sign: In acute cases, anorexia, drop egg production, depression, ruffled feather and their eyes closed, is a common finding. Respiratory distress with rapid breathing can occur but the most characteristic sign is a watery to mucoid yellow diarrhoea. In chronic progressive loss of condition and intense anaemia develops which produce shrunken, pale combs and wattle

Post mortem finding: have a swollen, friable and often bill-stained liver with or without necrotic foci. Enlargement of spleen and kidney may also occur.

Diagnosis: by isolation and identification of the organism

Treatment: furazolidone continuously in the feed for 10 days. Recently, Enrofloxacin has been used

Control: vaccination.

PULLORUM DISEASE: Synonym: bacillary white diarrhoea

Caused by *salmonella pulorum*. It usually cause high mortality in young chicken and occasionally in adult chicken

Transmission: is chiefly directly through the egg but also occurs by direct or indirect contact. Infection transmited via egg or hatchery usually result in death during the firist few days of life and up to 2-3 weeks of age.

Clinical finding: predominantly seen in chicken under 3 weeks of age. And the first indication is excessive number of dead-in- shell chicks and death shortly after hatching. Affected bird shows depression, respiratory distress, anorexia and white viscous dropping that adhere to the feather around the vent. A subacute form with lameness and swollen hock joints may be seen in growing birds and result in poor growth rate. Reduced egg production with lowered fertility and hatchability.

Post-mortem finding: in young birds, unabsorbed yolk sac; focal necrosis of the liver and spleen; and greyish nodules in the lung, heart and gizzard.In adult pericarditis, peritonitis or distorted ovarian follicles.

Diagnosis: based on post mortem finding, and isolation of the organism.

Treatment: no antibacterial are currently approved for the treatment of infected flock

Control: routine testing of breeding flock assure freedom from infection.

II-VIRAL DISEASES OF POULTRY

Marek's Disease

It is economically important neoplasms in poultry.

Etiology: caused by a *herpes virus*. Three genotypes are recognized & virus serotype 1 is the virulent chicken isolate.

Occurrence: Marek's disease is one of the most ubiquitous avian infections (worldwide)

Species Affected: Chickens are the only important natural host

Transmission: Marek's disease is highly contagious and is readily transmitted among chickens. The virus is released into environment from the epithelium of the feather follicle. It may survive for months or years in poultry house letter or dust. Infection usually occurs through aerosol exposure. Once infected chickens continues to be carriers for long periods and act as a source of infection.

Clinical Findings: Depression is noted prior to death. Sometimes transient paralysis is also noted. Chicken becomes ataxic for periods of several days, followed by recovery. This syndrome (transient paralysis syndrome) is rare in immunized birds,

Necropsy Finding: peripheral virus particularly the vagus, brachial and isciatic nerve become enlarged and lose their striations.

- ⇨ Diffuse or nodular lymphoid tumors may be seen in various organs, particularly in the liver, spleen, heart, lung, kidney, muscle, proventriculur and gonad.
- ⇨ The bursa is frequently atrophic.
- ⇨ Enlarged feather follicles (skin leukosis) may be noted in broilers.

Diagnosis: the disease occurs at any age over 3 weeks. Confirmation of a diagnosis may be made histologicaly (a mixture of tumor cells and reactive inflammatory cells) or by demonstrating the tumor-associated surface antigen on some of the individual cells by immuno fluorescence.

Control: Vaccination is the principal method of control.

NEWCASTLE DISEASE

An acute, rapidly spreading, viral disease of poultry in which the respiratory signs (coughing, sneezing and rales) are often accompanied or followed by nervous manifestations and infection with some strain, by diarrhoea and swelling of the head.

Etiology: caused by *paramyxo virus*. It has three strains: high virulent (Velogenic), intermediate (Mesogenic) and less virulent (Lentogenic).

Occurrence: The disease occurs worldwide in a variety of domestic and wild birds.

Transmission: Virus is shad during incubation, during the clinical stage and limited period during convalescence. Virus is present in exhaled air, in respiratory discharges, in faces, in eggs layed during clinical disease and in all parts of the carcass during acute infection. Chickens are readily infected by aerosols and by ingesting water or food contaminated with the virus.

Clinical Findings: Respiratory or nervous signs or both occur in the most widespread forms of the disease. Signs appear almost simultaneously throughout the flock 2-15 days after exposure.

- Respiratory signs are gasping and coughing.
- Nervous signs include dropping wings, dragging legs, twisting of head and neck, circling, walking back ward (particularly after drinking water), depression, in appetence and complete paralysis. Laying flacks may have partial or complete cessation of production. Eggs become abnormal in colour, shape or surface and with watery albumen are produced.
- Viscerotropic sign are predominate in the peracute disease includes watery and greenish diarrhoea and swelling of the tissue around the eyes and in the neck.

Necropsy Findings: Petechation may be seen on the serous membranes.

- ⇨ Haemorrhages of the proventricular mucosa and the intestinal serosa occur and are accompanied by necrotic areas on the mucosal surface.
- ⇨ Congestion and mucoid exudates may be seen in the respiratory tract, with opacity and thickening of the air sacs.

Diagnosis: Tentative diagnosis is based on history of rapidly spreading disease and signs associated with involvement of both the respiratory and nervous system. Confirmation by serology

Differential Diagnosis: Avian influenza.

Control: Live virus vaccine are widely used. Lentogenic strains, chiefly B1 and Lasota are administered in drinking water, nose or eye drops. Healthy chicks are vaccinated as early as the 4^{th} or even the first day of life.

CHRONIC RESPIRATORY DISEASE (CRD); Synonyms*: infectious sinusitis*

CRD affects all parts of the respiratory tract and is characterized by being extremely responsive to stress factors.

Etiology: *Mycoplasma gallisepticum.*

Occurrence: It is prevalent in almost every tropical country.

Species Affected: The disease is economically important in chickens and turkeys.

Transmission: The major route is via the egg and the disease continues from generation to generation by embryonic transmission.. Air born transmission is also common and can occur rapidly among pen mats or between pens. The infection may be dormant in the infected chick for days to months, but when it is stressed, aerosol transmission occurs rapidly and infection spreads through the flock.

Live virus vaccination, natural virus infection, cold weather or crowding may initiate the spread. In addition, the infection may be carried by person, by fomites or by infected birds introduced into a clean flock.

Clinical Findings: Affected birds have varying degrees of respiratory distress, slight to marked rales, difficulty in breathing, coughing and sneezing. Morbidity is high and mortality low in uncomplicated cases.

Feed efficiency and weight gain are reduced. In laying flocks, birds fail to reach peak egg production and the production rate is lower than normal.

Necropsy Findings: uncomplicated CRD infection in chicken result in relatively mild sinusitis, trachitis and air saculitis. E. Coli infection is concurrent and results in severe air sac thickening and turbidity, with exudative accumulations, fibrino-purulent pericarditis and per hepatitis.

Diagnosis: Because of many poultry diseases that produce lesions in the respiratory tract, accurate diagnosis of CRD from lesions alone cannot be made. It can be confirmed by isolation and identification of the organism.

Differential Diagnosis: Infectious coryza, laryngotrachitis, etc…

Treatment: In the field many cases of CRD infection are complicated by other pathogenic bacteria, thus, effective treatment must also attack the secondary invader. Most strains of the organism are sensitive to a number of antibiotics (such as oxytetracycline, streptomycin, etc) and tylosin. Twenty two

Control: Avoid stress of any kind

- Introduction of birds from endemic farms or areas has to be prevented.
- Workers and other personnel do not move from flock to flock on the same day.
- Vaccination is used to control the disease but not fully reliable.

Chapter four

4. Non infectious disease

4.1. Disturbance of the Digestive Tract

4.1.1. Abnormalities of prehension, mastication and swallowing

a. **Prehension** is the act of taking feed and water into the mouth; it varies in different species of animals. Tongue, lip and teeth are the main organs of prehension. The abnormal act is mostly due to painful conditions of the organs of prehension, e.g. stomatitis, gingivitis, broken teeth, glossitis, paralysis of lips, malposition of jaws, etc.
b. **Mastication** is the function of crushing ability of teeth, which needs the unique mobility of tongue and massive action of the masseter muscles of the cheeks. It is impaired due to painful conditions of the various organs of the oral cavity, e.g. stomatitis, gingivitis, glossitis, or the presence of foreign body. Dropping of food from the mouth clinically manifests due to occlusion or painful reaction in the oral cavity and this condition is called "**cud dropping**".
c. **Swallowing (deglutition)** is the process of transferring materials from the mouth to the stomach through the passage of oropharynx and esophagus. The structures involved in swallowing are floor of the mouth, tongue, hard and soft palate, esophagus, and laryngeal muscles. Causes of inability to swallow includes foreign body, tumor or inflammatory swelling in pharynx or esophagus, painful condition of pharynx or esophagus, esophageal dilatation due to paralysis, esophageal diverticulum, or esophageal spasm at site of mucosal erosion.
d. **Polyphagia** is excessive or increased feed intake. This can be seen in case of starvation, chronic pancreatitis and metabolic disease like diabetes mellitus (chronic hyperglycemia).
e. **Anophagia or Aphagia** is decreased feed intake. It may be due to painful conditions of the mouth and pharynx or to any bacterial or viral infection producing toxemia or septicemia. Vitamin deficiency like thiamin and cyanocobalamin may also produce inappetance.
f. **Pica or Allotriophagia** refers to ingestion of materials other than the normal feed. It is often due to deficiency in the diet such as lack of salt or inadequate trace elements such as phosphorus or copper.
g. **Starvation and Thirst**

Starvation is complete deprivation of feed as in drought, flood, bushfire or human intervention. Starvation may lead to hypoglycemia; acidosis, & ketosis.

Clinical manifestations include: hunger, weight loss, dramatic fall in milk yield, faecal output decline, weakness, recumbency, staggery gait.

Treatment -provide small amounts of highly digestible carbohydrate and proteins orally; avoid lipids, which may exacerbate ketosis.

Thirst: - the subjective sensation of the need to drink fluid which is manifested in farm animals by polydipsia, or allotriophagia in the form of drinking abnormal fluids, especially urine.
Etiology: • in dehydrated animals due to diarrhoea, vomiting, sweating,
• Polyuria, excessive salt intake in the feed, water deprivation or diabetes insipidus.

Clinical findings: partial water deprivation: restlessness, wondering around water supply, aggressiveness, sunken eyes, hollow abdomen, skin tenting; tremor, excitement, frothing at mouth, stiff gait, terminally very excited, urine specific gravity increased.
Treatment: replacement therapy with fluids and electrolyte, slowly in small amounts to avoid water intoxication.

4.1.2. Syndromes associated to digestive tract disturbances and their treatment

1. Diarrhea and Constipation

Diarrhea

Definition: frequent defecation of fluid or semi fluid faces

Etiology: Enteritis; Malabsorption, e.g. due to villous atrophy; Functional diarrhea as in excitement

Clinical findings: increased frequency of defecation; increased faecal water content and volume; thinner consistency of faces; decreased alimentary tract transit time; dehydration.

Diagnosis: History of feed ; change in frequency, color, consistency & elements of faces

Treatment: -fluid therapy & dietary adjustment and removal of the cause of the GIT diseases

Constipation: the decreased frequency of defecation accompanied by dried faces.

Etiology:• Severe debility as in old age; Deficiency of fibres /bulk in the dietary; Chronic dehydration; Partial obstruction of large intestine; Painful conditions of the anus; Paralytic ileus &Terminal stages of pregnancy in cows.

Diagnosis: - retention of faces, unfruitful or ineffective attempt to defecate

Treatment: - correction of predisposing factors, mineral oil preparation, e.g. liquid paraffin by mouth or as enema, fibrous diets, calcium, vit A, &vit B complex therapy

2. Vomiting and Tenesmus

***Vomiting*:** the act of forceful emptying of stomach contents by vigorous contractions of diaphragm and abdominal muscles. It is the most protective disturbance of digestive tract. It is frequently observed in dog and cat, sometimes in pig, seldom in cattle, horse, sheep or goat.

Etiology: Gastric irritation and ulceration; Gastric dilatation, emotional conditions

- Foreign body in oesophagus or pharynx
- Most toxins and some drugs

Vomition *may be central or peripheral*

Central: - occurs when there is stimulation of the vomiting centre in the medulla. It is induced by drugs and toxaemic conditions, e.g. arsenic poisoning, poisonous plants, acute chemical poisoning (phosphorous) or fear and tension

Peripheral- irritation of mucosa of the alimentary tract, e.g. gastritis, gastric dilatation, foreign body in the pharynx, oesophagus, overloading of the stomach,

Clinical findings: restlessness extends head and neck, contraction of abdominal wall, retching, protrudes tongue and large volume of digesta pours from mouth, stomach rupture (a terminal event in horse).

Treatment: elimination of the primary cause of vomition, fluid therapy and antiemetic drugs like chlorpromazine

***Tenesmus*:** it is a condition in which defecation is accompanied by signs of pain like groaning, grunting, with repeated tensing of the abdominal musculature.

Etiology: problem in pelvic cavity (inflammation of lower bowel or lower reproductive tract, e.g. coccidiosis, vaginitis, retained placenta, dystocia spinal cord lesions like abscess, or due to rabies), constipation, or old age.

Clinical findings: frequent straining at defecation

Treatment: the primary cause should be treated.

- Supportive treatment with fluid and electrolyte
- Epidural anaesthesia-to avoid straining

3. Bloat (ruminal tympany)

Definition: it is over distension of the rumen and reticulum with the gases of fermentation, either in the form of persistent foam mixed with the rumen contents or in the form of free gas separated from the ingesta. Thus, there are two classes of bloat**:** ***Frothy or primary bloat*** is dietary in origin and occurs in cattle on legume pasture and in feedlot cattle on highly fermentable grain diets; ***Free gas or secondary bloat or chronic bloat*** is usually due to failure of eructation of free gas because of a physical interference with eructation.

Etiology:

A. Frothy bloat: there are two major factors:

1. Dietary factor-- it is the major cause of frothy bloat. It has a primary and secondary factor

Primary factors include grazing on very green succulent lush pastures or immature (pre-blooming) rapidly growing legumes, excessive intake of cereal crops, peas, beans, cabbage, potato, etc. this is known as pasture bloat.

Secondary factor seen in feedlot, due to feeding finely ground grains that produces enormous quantity of gas under the influence of microbial fermentation. This bloat is known as feedlot bloat. The froth causes physical obstruction to the cardiac and impedes eructation.

2. Animal factors—some are susceptible while others are not. This is due to certain inherited characters. There is also age factor (young animal is more susceptible).

B. Free gas bloat: physical or functional defects in the eructation of the normally produced gas. This is caused by the mechanical obstruction of the esophagus (choke), stenosis of esophagus, interference with esophageal groove function, vagus damage (vagus indigestion), reticulitis, gastritis, tumor mass, etc.

Clinical findings: sudden onset of abdomen distension; Inappetence to anorexia, marked dyspnoea with mouth breathing (extension of the head and neck, protrusion of tongue, abducted elbow),

- Respiratory rate increased, tachycardia, depression, and salivation
- Death usually commence 3-4 hours later.

Note: The cause of death in case of bloat is dyspnoea due to the increased intraruminal pressure by the distended rumen on diaphragm to such an extent that it depresses the cardiovascular and respiratory system. The outcome of this condition is hypoxia and ultimately asphyxia and death.
Diagnosis: Based on the clinical signs **;** Percussion revealing tympanic sound on the left Para lumbar fossa **;** History of access to lush pasture or finely ground grain**;** Season of the year.

Treatment:

- Keep the animal where the anterior portion is elevated over posterior part to reduce pressure on diaphragm
- Trocarization (puncture of the rumen at Para lumbar fossa with trocar and canula) or introduction of stomach tube in case of free gas bloat
- Use of antifoaming agents like mineral or vegetable oil or liquid paraffin (dose500mg-1liter) in case of frothy bloat
- Correction of the predisposing factors.

4. Simple indigestion

Definition: it is a minor disturbance in ruminant GIT function related to a change in the quality and quantity of the diet. It is a milder form of CHO engorgement.

Etiology: or predisposing factors include

- Sudden change of feed

• Poor feed quality, e.g. mould, spoiled and indigestible roughage
• Prolonged oral antibiotic therapy
• Animal fatigue or stress e.g. shipping

Clinical findings: reduced appetite, depression, and absence of rumination, firm and doughy rumen without abnormal distension, normal physical clinical parameters.

Treatment: • Correction of the suspected predisposing factors;

• Correction of the rumen environment by cud transfer from a healthy or slaughtered animal;
• Administration of rumen stimulants like neostigmine or physiostigmine;
• Use of Vitamin B-complex and mineral oils.

5. Rumen over load /CHO engorgement / lactic acidosis

Definition: it is a condition in which large amounts of highly fermentable carbohydrate rich feeds causes an acute disease due to the excessive production of lactic acid in the rumen. It is characterize by ruminal stasis, severe toxaemia, diarrhoea, weakness and recumbency, and dehydration

Etiology: sudden ingestion of toxic doses of carbohydrate rich feeds especially grain. A sudden ingestion of toxic doses of carbohydrate rich feeds results in production of lactic acid that affect the microbial population of the rumen (marked increase in the number of gram positive bacteria) that further increases production of large quantity of lactic acid. This lactic acid will be absorbed in to the blood streams and causes rumen stasis, chemical rumenitis, diarrhea, dehydration, and muscular weakness.

Clinical findings: ruminal distension, abdominal pain (belly kicking) and ruminal stasis, subnormal body temperature, shallow and rapid respiration, diarrhoea, the faeces contain undigested fragments of the feed, dehydration, dullness and anorexia, etc.

Diagnosis: • history of sudden access to toxic doses of grain; Characteristic clinical signs especially anorexia, rumen stasis and depression, rumen fluid PH, complete absence of ruminal protozoa.

Treatment: - correction of ruminal and systemic acidosis by using alkalinizing agents through stomach tube and /or IV by sodium bicarbonates.

- Balanced electrolyte solutions.
- Cud transfer (10-20lit) from healthy animals;
- Restore ruminal motility by using rumen stimulants.
- Prevent animals from further access to high quality feeds, e.g. grain.

6. Colic

Definition: Colic is a complex symptom exhibited by animals when there is painful condition of any abdominal organs. It is common in equine specie, however, it may occur in other animals as well.

Etiology: depending on its cause, colic is classified into the following classes even though they are not mutually exclusive.

a. Spasmodic (spastic) colic: It is characterized by a periodic spastic contraction of the intestinal muscles or visceral pain. It is the most common type in equine species. This may occur due to irritation of GI mucosa by unsuitable foodstuff, excitement, or drinking of cold water following work.

b. Tympanic (flatulent) colic: it results from excessive gas accumulation in the intestinal tract where over distension of viscera stimulates pain and pressure receptors causing mild to severe colic.

c. Impactive colic/ obstructive colic: It may result due to obstruction or impaction of GIT with impacted food mass, stone, sand, foreign body. It can be seen in stomach, cecum, colon, etc.

Causes: physical agents like:

1. Feed related: -coarse roughage may predispose the horse to improper digestion of feedstuffs with a resultant impaction.
2. Water related-insufficient amounts of water create dry ingesta prone to impaction;
3. Poor teeth- similar to poorly digestible feeds, improper mastication causes some impactions.
4. Parasitic agents: migrating larval forms of S.vulgaris interfere with circulation and innervations of various parts of the large intestine, which affects gut motility leads to impaction.

d. Extra-luminal colic: due to mechanical distortion or obstruction of intestine with a consequence of interference with blood supply, e.g. volvulus, strangulation, intussusceptions, etc.

Clinical findings: the cardinal sign of colic is due to abdominal pain manifested by pawing, rolling, sweating, kicking at the abdomen, getting up and down frequently, flank-watching, hyper motility of intestine, increased respiratory and heart rate, absence of faeces.

Diagnosis: clinical findings and history of the animal related to management of feed.

Treatment: treatment should aim to relief pain targeting to the predisposing factors.

- Use of analgesics like flunixinmeglumine, xylazine, phenaylbutazole
- Use of laxatives in case of impactive colic for softening the mass
- Surgical exploration or laparotomy in case of extra-luminal colic
- Regular deworming of horses and improvement of husbandry.

7. Oesophageal obstruction (Choke)

Definition: it is an obstruction or occlusion of esophageal lumen or tube.

Etiology: Inruminants by solid objects, such as apples, pears, potatoes, etc. In horses most frequently due to greedily eating behaviours, by dry grains or a bolus of hay, less often by corn, potatoes, and occasionally on medical boluses.

•Tumors, swollen lymph nodes, abscesses of the surrounding tissue etc.

Clinical findings: The site of obstruction in large animals most frequently occurs in cervical and less often in the thoracic region.

- Sudden stoppage of eating & forceful attempts to swallow;
- Profuse salivation and food and saliva are regurgitated through nostrils; coughing and continuous chewing movements; in cattle bloat and salivation are typical signs.

Diagnosis: from history and prominent signs.

- Cervical oesophageal choke may be located by external palpation or passage of a stomach tube.
- Diagnosis of thoracic obstruction may be confirmed by the careful passage of a stomach tube.

Note: in any animal, signs of difficulty of swallowing and salivation should be considered as a case of rabies

Treatment: a) Conservative treatment:

- Sedate animals to control pain that is caused by obstruction and allow muscles relaxation
- Passage of stomach tube to the obstructed site with repeated pushing with great care and softens with water to loosen the obstructing material and drop down to stomach
- Relief the bloat by trocarization.

b. Radical treatment (esophagotomy): surgical removal of the obstructing materials if conservative measures failed.

8. Intestinal obstruction

Definition: it is the occlusion of the intestinal lumen either from the external pressure or from other physical occlusions or intestinal displacement. It is characterized by abdominal pain,

absence of defecation or often the passage of blood and mucus tinged faces. It occurs in all large animals but is most common in horses.

Etiology: physical obstructions due to intestinal accidents like:

Intussusception is a form of obstruction in which a part of intestine turns in (invaginate) on itself..

Volvulus is a form of obstruction produced by the twisting of a loop of intestine round its mesenteric axis.

Torsion is obstruction caused by the bowel twisting on its own long axis.

Strangulation is an obstruction caused by volvulus or intussusception in which blood supply to a loop of intestine trapped in a hernia form; its development causes tissue necrosis and consequently toxemia and death

Incarceration is a condition in which some part of intestine passes through a torn peritoneum and lie under the skin just like hernia.

Clinical findings: Signs associated with abdominal pain**;** Increased respiratory and pulse rate**;** Dehydration, gastric dilatation**;** Absence of defecation.

Diagnosis: based on clinical signs, especially complete absence of faces and passage of blood and mucus are typical signs. Rectal palpation reveals distended loop of intestine, empty rectum.

Treatment: some impaction may be treated with mineral oil. However, most true obstructions usually require surgery (laparotomy), use of analgesics.

9. Rectal prolapse

Definition: is the protrusion of one or more layers of the rectum through the anus due to persistent tenesmus and associated with intestinal or urogenital diseases. Prolapse may be classified as either incomplete (where only rectal mucosa is everted) or complete (where all rectal layers are protruded).

Etiology: severe enteritis, parasites, lacerations, neoplasia of the rectum or distal colon, urolithiasis, retained foetal membrane, dystocia, etc.

Clinical findings: an elongated, cylindrical mass protruding through the anal orifice

Diagnosis is by typical clinical signs.

Treatment: identify and eliminate the cause of the rectal prolapse

replacement of viable prolapsed tissue to its proper anatomic location or amputation of the segment if necrotized & caudal epidural anaesthesia to reduce straining , facilitate repositioning of the prolapse and to permit surgical manipulation.

4.2.Metabolic disease

Amongst domestic farm animals' metabolic diseases are very importance in dairy cows and pregnant ewes. In dairy cows, the incidence of metabolic diseases is highest in the period commencing at calving and extending until the peak of lactation is reached, and their susceptibility appears to be related to extremely high turnover of fluids, salts and soluble organic materials during the early part of lactation. The term "production disease" encompasses those diseases previously known as "metabolic diseases".

4.2.1. Parturient paresis/milk fever/hypocalcaemia

Definition: it is a metabolic disease occurring most commonly about the time of parturition in adult females and is characterized by hypocalcaemia, general muscular weakness, circulatory collapse and depression of consciousness.

Etiology: sudden loss of calcium through milk at or near the time of parturition. Serum phosphorus is decreased but Serum magnesium is increased. Hypocalcaemia causes general muscle weakness, circulatory collapse, depression and unconsciousness.

Clinical findings: there are three different stages of milk fever:

Stage1 is a brief excitement and tetany with hypersensitiveness and muscle tremor of the head, flank and limbs. Animal is able to stand but restlessnes, shuffling feet, bellowing, and open-mouth breathing with tongue extension.

Stage2 is sternal recumbency.

In this case the patient is unable to stand, depression, anorexia, subnormal temperature, loss of defecation and urination, GIT atony (i.e. mild bloat, constipation), decreased heart sound, retained foetal membrane, lies on her brisket with the head characteristically turned into the flank over one shoulder or rested on the ground &eye blinking.

Stage3, lateral recumbency.

Lose consciousness progressively to the point of coma, unresponsive to stimuli, severe bloat because of lateral recumbency and GIT atony, heart sound becomes inaudible and pulse may be undetectable.

Diagnosis: • clinical signs and history of animals (calving, lambing)

- Laboratory tests: total serum Ca, P and Mg determination (hypophosphatemia, hypermagnesemia)

• Favorable response to therapy of IV injections of calcium solutions.

Treatment: IV administration of calcium borogluconate slowly, within 30 minutes of treatment animal will stand and relief from bloat.

Prevention: -feeding diets low in Ca and normal to high phosphorus during late pregnancy

• Administration of Vit D_3 and Ca in the form of Ca gel at calving.

Ketosis in cattle/Acetonemia of cattle

Definition: it is a disease of lactating cows characterized Clinically by loss of body weight, reduced milk yield, nervous signs (e.g. walking in circles, straddling or crossing of the legs, head-pushing, apparent blindness, aimless movements and wandering, vigorous licking of the skin and inanimate objects, depraved appetite and chewing movements with salivation), Laboratoricaly by ketonemia, ketonuria, hypoglycaemia, & ketone bodies in milk.

Etiology: ketosis is basically the result of a negative energy balance early in lactation. High milk production causes energy (glucose) drain and the need for energy exceeds the capacity. A defecit in digestion or metabolism may result in inadequate amounts of glucose (hypoglycemia) available at the cellular level. In an effort to correct this condition, body fat and limited protein stores are mobilized for gluconeogenesis in the liver with a parallel rise in ketone body formation. Theseketone bodies have a toxic effect to the CNS if the level rises beyond the physiological limit. It occurs most commonly during the first month of lactation, less commonly in the second month, and only occasionally in late pregnancy. The highest frequency is between 20-30 days after calving. It can occur in cows of any age, rarely at first lactation, reach its peak at fourth.

The risk factors include low energy intake, high protein intake, excessively fat cows, inter current diseases e.g. milk fever, foot rot.etc…

Clinical findings:

Wasting syndrome: - gradual decrease in milk yield and appetite, weight loss, depression, disinclination to move, ketone odor on breath or milk, ruminal movements decreased in amplitude & number.

Nervous syndrome:- circling, leg-crossing posture, head-pressing, blindness, aimless wandering, vigorous licking of self/fixed objects, depraved appetite, chewing movements with excessive salivation, incoordination, bellowing etc.

Diagnosis: based on clinical findings and history of lactation.

Treatment: IV injection of 500ml 50% glucose solution result in rapid improvement, however, relapse is common; IM administration of glucocorticoids; supportive treatment by

oral administration of propylene glycol for 2 days or other glucose precursors to overcome relapse conditions must be included.

Hypomagnesaemia in cow / Lactating Tetany /Grass Tetany /Grass Staggers/ Wheat pasture poisoning

Definition: It is a metabolic disease of cattle, horse, buffalo, sheep and goat characterized by hypomagnesaemia, and usually hypocalcaemia, and clinically by hyper aesthesia, in coordination, tonic-clonic muscular spasms, convulsions, and death due to respiratory failure.

Etiology: a deficiency of magnesium in blood stream is the principal cause of hypomagnesictetany.

Factors affecting magnesium homeostasis:

a. Tetanogenic pasture. Young, green grass has a lower content of available Mg than mature grass, heavy applications of potassium-rich and nitrogen-rich fertilizers reduce the availability of soil Mg.

a. Ammonia formation. Excessive production of ammonia in the rumen from protein rich diet prevents, by a process of chelating the absorption of magnesium.
b. Starvation: during bad weather, transport, and movement to unaccustomed pasture may bring about such a period of partial starvation.
c. Lactation. Considerable quantity of magnesium is excreted through milk during lactation resulting to hypomagnesaemia if not replenished through diet.
d. Scour. Scour causes decreased absorption of magnesium.

Clinical findings:

Acute: • Suddenly ceases to graze ; Unusual alertness and appear uncomfortable

- Staggering gait and fall down with obvious tetany of the limbs that is rapidly followed by clonic convulsions lasting for about a minute
- Opisthotonus, nystagmus, Champing of the jaws , Frothing at the mouth
- Temperature rises to 40-40.5^{o}C, high pulse and respiratory rates

Sub acute: -The onset is more gradual over a period of 3-4 days

- Slight inappetence; Wildness of the facial expression ; Exaggerated limb movements
- Spasmodic urination and frequent defecation are characteristic
- The appetite and milk yield are diminished and ruminal movements decrease
- Muscle tremor and mild tetany of the hind legs and tail with trembling
- Straddling gait may be accompanied by retraction of the head

Chronic tetany: -May not show clinical signs although serum Mg level is low. May show vague syndrome like dullness, unthrift ness, and indifferent appetite. Chronic tetany may turn to sub acute form.

Diagnosis: in coordination, hyperesthesia and tetany are the major clinical abnormalities especially if they occur in ruminants exposed to grazing green cereal crops or lush grass-dominant pasture. Lactating animals are likely to be affected first.

Treatment: combined calcium-magnesium preparation (e.g. 500ml of a solution containing 25% Caborogluconate and 5% Mg hypophosphite for cattle, 50ml for sheep IV followed by a subcutaneous injection of a concentrated solution of a magnesium salt. If signs of respiratory distress or increase in heart rate are noticed the injection should be stopped immediately and, if necessary, a calcium solution injected.

Control: • Feeding of magnesium supplements, 120g magnesium oxide per day.

- Spraying with a 2% solution of magnesium sulfate at fortnightly intervals or by application of very finely ground magnesium oxide to the pasture (30kg/ha) before grazing commences.

- Feeding on hay and unimproved pasture.

Ovine ketosis /Pregnancy toxemia of ewes/ Twin lamb disease/

Definition: it is a highly metabolic disease of sheep and goat that occurs in late pregnancy and characterized by hypoglycemia, ketonaemiaand low liver glycogen content.

Etiology: Negative energy balance and multiple fetuses predispose the disease. A deficiency of blood glucose level is the main cause. This deficiency may be related to low intake of grains or for carrying twin or triplets

Clinical findings:

- Anorexia is the first signs. This is followed by depression of consciousness,
- Twitching of muscles, rapid respiration and ataxia,
- Blind (no corneal reflex) and stand with their head pressed against some object, Walk in circle,
- Spasms of the head and neck muscles, spasticity of the limb muscles, Recumbency, paralysis and death.

Diagnosis: based on the history of pregnancy and inadequate amount of grain feeding. When female goat or sheep is sick in advanced pregnancy, suspect for this disease. • By the typical clinical signs.

Treatment: once the advanced signs have developed, no treatment is effective.

- •Cesarean section or induced abortion early in the course of the disease usually leads to recovery.
- •Gluco-corticoids are probably only effective through increasing blood sugar.
- • Provide palatable feed and water and protection from extreme weather
- • Twice daily force-feeding with finely ground-dried grass given by stomach tube may be good practice with especially valuable animals

Prophylaxis-obesity should be avoided in early pregnancy and adequate good feed supplied during the last 6 weeks of pregnancy.

- • Minimum changes of feeds should be made during advanced pregnancy,
- • Avoid over stress, arrange adequate exercise in pregnancy period, and rule out parasitic burden.

4.3. Mineral and Vitamin Deficiencies/ imbalance

Deficiency of Minerals

At least 15 mineral elements are nutritionally essential for animals. The macrominerals are calcium, phosphorus, potassium, chlorine, magnesium, and sulfur. The trace elements, or microminerals, are copper, selenium, zinc, cobalt, iron, iodine, manganese and molybdenum. The trace elements are involved as component parts of many tissues and one or more enzyme activities and their deficiency leads to a wide variety of pathological consequences and metabolic defects.

Copper Deficiency

Copper deficiency occurs primarily in young ruminants in a range of clinical manifestations including unthriftiness, diarrhea, lameness, demyelination of the central nervous system in neonates, anemia in the later stages of deficiency and falling disease.

Etiology:

1. Primary copper deficiency is due to inadequate intake of copper in the diet when forage is grown on deficient soils or on soils in which copper in unavailable.

2. Secondary (conditioned) copper deficiency is due to impairment in the utilization of the copper by tissues even though blood copper levels are high in response to the physiological needs of the tissues for the element. In this case the dietary intake is sufficient. It is conditioned by the presence of other materials in the diet e.g. molybdenum, sulfate, zinc, iron, and lead, which impede the utilization of copper by body tissue. Molybdenum and sulfate alone or in combination can affect copper metabolism through the formation of insoluble complex of copper-thiomolybdates in the rumen that are not absorbed from the intestine. This effect also operates in the fetus and interferes with copper storage in the fetal liver.

Clinical findings:

•Anemia →. Copper is essential for the synthesis of hemoglobin along with iron.

- Diarrhea (scouring) → high molybdenum content in the diet is usually responsible for this disorder. Diarrhea is usually seen in cattle. This diarrhea is persistent in nature with the passage of watery, yellow green to black feces with inoffensive odor. The feces are released without effort, often without lifting the tail. It is defined as "peat scours" and "teart".
- Bone deformities → copper deficiency reduces the strength and stamina of bones due to loss of collagen. Bones become porous (osteoporosis) and there is tendency of spontaneous fracture.
- Nervous disorder → copper deficiency produces demyelination of central nervous system. Usually seen in lamb and kids. There is necrosis and neuronal degeneration of spinal cord and brain stem. Such animal shows nervous manifestations known as "neonatal ataxia", "enzootic ataxia" and "sway back". Paralysis is the ultimate outcome of this problem.
- Pigmentary disorder → copper deficiency causes depigmentation of hairs and wools known as "achromotrichia". Copper interferes with the formation of melanin from tyrosine as such black hairs turn grey or brown (bleached). Wools lose crimp, become straight, rough and staring. There are abnormalities in the growth of hairs and wools. Hair color around the eyes is strikingly altered and this is ascribed as "spectacle disease".
- Myocardial degeneration → copper deficiency produces fibrosis characterized by sudden death. This condition is described as "falling disease". Acute heart failure is the cause of death.
- Infertility → copper deficiency has been considered as the cause of delayed estrus in cattle. Copper deficient animals may yield dead fetus and abortion.

Diagnosis: based on history of diet, clinical findings and estimation of copper levels on the soil, diet, blood and liver. Low level of copper reduces hemoglobin level and RBC number.

Treatment: supplementation of copper sulphate through oral route is recommended in clinical cases. Dose: 4g of copper sulfate for calves from 2-6 months of age and 8-10gfor mature cattle given weekly for 3-5 weeks. The requirement of copper is about 5 ppm of the dry diet. 0.5% copper can be added in mineral mixture. Supplementary selenium may enhance copper absorption.

Cobalt Deficiency

Cobalt plays a vital role in the rumen of ruminants for the synthesis of vitamin B_{12} (Cyanocobalamin). Vitamin B_{12} plays a crucial role in the production of red cells. Cattle and sheep are similarly affected and the signs are identical in both species. Cattle are slightly less susceptible than sheep; lambs and calves are more seriously affected than adults. Goat is less vulnerable than sheep.

Etiology: the disease is caused by a deficiency of cobalt in the diet and characterized by anorexia and wasting.

Clinical findings: No specific signs are characteristic of cobalt deficiency. When sheep, cattle or goat are confined to cobalt-deficient diets, there is a gradual decrease in appetite, failure of growth or weight loss, rapid muscular wasting (marasmus), pica is likely to occur especially in cattle, severe anemia (pallor of mucus membrane), lactation and wool production are severely retarded. In late stage there are infertility, diarrhea and lacrimation.

Diagnosis: - is based on treatment response to oral dosing with cobalt &parentral administration of vitamin B_{12} and determination of vitamin B_{12} content of the feces. On oral dosing with cobalt the signs disappear and vitamin B_{12} in the feces return to normal.
Treatment: - Oral dosing with cobalt or IM injection of vit. B_{12} or oral dosing with large amount of vitamin B_{12}.
Prevention: - the recommended safe level of cobalt in the diet for sheep and cattle is 0.11mg cobalt/ kg dry matter diet, or supplementation of the diet with cobalt is necessary.

Iodine Deficiency

Goiter (thyroid hyperplasia) is the cardinal sign of iodine deficiency. The major clinical manifestation is neonatal mortality, with alopecia and visible and palpable enlargement of the thyroid gland.

Etiology: primarily due to deficient iodine intake or secondarily conditioned by a high calcium intake.

Iodine deficiency reduces the ability of thyroid gland to produce thyroxin hormone. It stimulates the secretion of TSH by pituitary gland. It causes hyperplasia of the thyroid gland and goiter.

Clinical findings: thyroid hyperplasia, loss of libido in the bull, failure to express estrus in cow, hair or wool loss (alopecia), high incidence of abortion, stillbirth and weak newborn animals.

Diagnosis: easily diagnosed if goiter is present but the occurrence of stillbirths without goiter may be confusing. Estimation of iodine levels in the blood and milk is needed.

Treatment: potassium iodine 200mg/kg dry feed or provision of iodine salt preparations. Recommended intake of iodine for lactating and pregnant cows is 0.8-1mg/kg dry feeds; for dry cow are 0.1-0.3mg/kg dry matters. Treatment must be undertaken with care, as over dosage will cause toxicity.

Iron Deficiency

Functionally iron is necessary for hemoglobin formation. A deficiency of iron in the diet causes anemia and failure to thrive. It is the most common problem of piglets kept under artificial conditions.

Etiology:

•**Primary:** is most likely to occur in newborn animals whose sole source of iron is the milk of the dam, milk being a poor source of iron. Deposits of iron in the liver of the newborn are insufficient to maintain normal hemopoiesis for more than 2-3 weeks, and are particularly low in piglets.Continued blood loss by hemorrhage in any animal may bring about a sub clinical anemia and an associated iron deficiency.

•**Secondary**: due to chronic bleeding, high infestation of cattle with sucking lice, high burden of blood-sucking strongylid parasites in horses.

Clinical findings: the highest incidence occurs at 3 weeks of age although the disease can occur in pigs up to 10 weeks of age. Significantly lower growth rate of anemic pigs than normal pigs. Obviously reduced food intake.

• Diarrhea is very common, but the feces are usually normal in color. Severe dyspnea, lethargy, and marked increase of the apex beat with exercise. The mucosa and skin are pale; Edema of the head and forequarters.

• Death usually occurs suddenly.

Diagnosis: is based onclinical findings, age (neonates are highly affected), laboratory test to determine Hb level and treatment response (IM injection of iron-dextran preparations or other iron preparations).

Treatment: the treatment is usually parenteral and consists of organic iron preparations such as iron-dextran, iron-sorbitol-citric acid complex, iron sacccharate or gluconate. The dose rate is 0.5-1g elemental iron in one injection once each week. Vitamin B_{12} is often used in the same injection at a dose rate of 5000 mg per week in a single dose.

Calcium, Phosphorus and Vit D Deficiency

A dietary deficiency or disturbance in the metabolism of calcium, phosphorus or vitamin D, including imbalance of the calcium: phosphorus ratio, is the principal cause of the osteodystrophies. The interrelation of these various factors is often very difficult to define.

Calcium Deficiency (hypocalcicosis)

Calcium deficiency may be primary or secondary, but in both cases the end result is an osteodystrophy, the specific disease depending largely on the species and age of the animals affected.

Etiology: Primary calcium deficiency is due to an absolute deficiency in the diet. This occurs rarely.

Secondary calcium deficiency is due to marginal calcium intake aggravated by high phosphorus in the diet. Such a diet depresses intestinal absorption and retention of calcium in the body, and the absorption of calcium from bones is increased. This can also occurs accompanied by a vitamin D deficiency in animals confined indoors.

Clinical findings: the Clinical signs, apart from the specific syndromes, are less marked in adults than they are in young animals.

Non-specific syndrome includes: - decreased rate or cessation of growth

- Dental mal-development which is characterized by deformity of the gums, poor development of the incisors, failure of permanent teeth to erupt for periods of up to 27 months and abnormal wear of the permanent teeth due to defective development of dentine and enamel, occurring principally in sheep.

- Soft bones, bendable ribs, easly fracture.
- Reduced fertility, difficult parturition.
- Tetany in pigs and young cattle.

Specific syndrome includes rickets, osteomalacia, osteodystrophiafibrosa, bovine degenerative arthropathy, and ovine hypocalcemic paresis.

Diagnosis: clinical signs, and diagnostic response to diet supplementation with calcium.

Treatment: -calcium borogluconate solution injection (as in milk fever) for tetany

- Supplement diet with calcium (ground limestone, bone meal)
- Reduce excessive phosphorus intake
- Aim at calcium: phosphorus ratio of 2:1 (optimum) up to 1:1; if urolithiasis is a problem ratio should be 2.5:1.

Phosphorus Deficiency (hypophosphatosis)

Phosphorus deficiency is usually primary and is characterized by pica, poor growth, infertility and, in the later stages, osteodystrophy.

Etiology: Phosphorus deficiency is usually primary under field conditions but may be exacerbated by a deficiency of vitamin D and possibly by an excess of calcium. Unlike calcium, a dietary deficiency of phosphorus is widespread under natural conditions.

Clinical findings: primary phosphorus deficiency is common only in cattle.

Young animals grow slowly and develop rickets. In adults there is an initial sub clinical stage followed by osteomalacia. Retarded growth, low milk yield and reduced fertility are the earliest signs of phosphorus deficiency. Osteophagia is common and may be accompanied by a high incidence of botulism. Cows in late pregnancy often become recumbent and although they continue to eat are unable to rise.

Diagnosis: based on clinical findings

Treatment & control: urgent treatment is 30g sodium dihydrogen phosphate in 300ml water IV for cattle. Daily supplementation for cattle of 15g phosphorus minimal, 40-50g optimal, as bone meal, rock phosphate, soft (colloidal clay) phosphate in form of free-access lick, mineral mix fed in concentrate or hay, pasture, in drinking water.

Vitamin D Deficiency (hypovitaminosis D)

Vitamin D deficiency is usually caused by insufficient solar irradiation of animals or their feed and is manifested by poor appetite and growth, and in advance cases by osteodystrophy.

Etiology: a lack of ultraviolet solar irradiation of the skin, coupled with a deficiency of calcium and phosphorous. Fish liver oil is the richest source of vitamin D.

Clinical findings: reduced productivity, poor weight gains, inappetence, and infertility. In late stages there is rickets in young and osteomalacia in adults.

Diagnosis: based on history of feeding vitamin D deficient diets and less exposure to sunlight, clinical signs, response to vitamin D therapy.

Treatment and control: Arrange exposure to solar irradiation; Include sun-dried hay in diet

Deficiencies of Vitamins

Vitamin A Deficiency (hypovitaminosis A)

Vitamin A is essential for the regeneration of the visual purple necessary for dim-light vision, for normal bone growth and for maintenance of normal epithelial tissues. Deprivation of the vitamin produces effects largely attributable to disturbance of these functions.

Etiology: Vitamin A deficiency occurs either as a primary disease, due to inadequate intake of this vitamin or its carotenoid precursors in the diet, or as a secondary disease in which the dietary supply of the vitamin or its precursor is adequate, but their digestion, absorption or metabolism is interfered with to produce a deficiency at tissue level. Major sources of this vitamin are preformed vitamin A from animal sources (fish oil, egg yolk, liver) and provitamin carotene from plants. Dark green leafy vegetables are the good sources of carotenes.

Clinical findings:

• Poor appetite and emaciation in growing animals
• Impairment of vision (*night blindness*)
• Skin thickening and keratinization (xerodermia)
• Conjunctivalxerosis: dryness, loss of transparency, thickening, wrinkling
• Corneal xerosis and ulceration,
• Xeropthalmia (dry eye)
• Susceptible to infection, so this vitamin is considered as anti-infection vitamin.

- In the urinary system, there is degeneration and cornification of epithelium. These favor the formation of renal stones. Low vitamin A and high calcium diet accentuates the formation of calcium phosphate calculi in the bladder and kidney.

Diagnosis: based on history of diet, clinical accounts, estimation of vitamin A level and respond to vitamin A supplementation.

Treatment: High dose of vitamin parenterally, even ten times of it may be warranted in some vulnerable sick animals. Doses: Calf 1,000,000 to 1,500,000iu, cow 250,000 to 2,500,000iu, lamb 125,000 to 250,000iu, horse 200,000 to 300,000iu.

Vitamin K Deficiency

This vitamin is adequately available in the plants and synthesized by herbivorous animals in the gut. Therefore, deficiency of it is rarely seen in animals.

Etiology: - prolonged use of oral antibiotics can retard the synthesis of this vitamin due to reduction or destruction of beneficial bacteria. High dosing with vitamin A can also interfere with bacterial synthesis of this vitamin.

This vitamin helps the formation of prothrombin and takes part in the normal coagulation process. Thus deficiency of vitamin K causes prothrombinemia and hemorrhage. Bile salt helps its absorption.

This vitamin should be used when there is bleeding, e.g. bleeding from nostril or other orifices, hepatitis and hemorrhagic gastroenteritis, bleeding due to warfarin and salicylate poisoning.

Treatment: in horse and cattle 80-250mg or 20mg/kg Bwt IM at 12-hour interval till bleeding ceases.

Vitamin B Complex Deficiency

Vitamin B complex deficiency seldom occurs in ruminants as they can synthesize adequate amount of these vitamins in the rumen through the bacteria. But, prolonged use of antibiotics and sulphonamides through oral route cause reduction of bacterial population and thus impede the synthesis of this vitamin. High carbohydrate diet may also reduce the bacterial population and synthesis of this vitamin.

Ascorbic acid (Vitamin C)

It regulates oxidation-reduction potential. It acts as co-enzyme in the oxidation of tyrosine and phenylalanine. It helps wound healing. It maintains capillary integrity.

Deficiency signs: scurvy in human beings. Infertility, slow healing and susceptibility to infection in animals.

Selenium And/ Or Vitamin E Deficiency

Selenium is an essential nutrient for animals. It is as a component of the enzyme glutathione peroxidase, which protects cellular membranes and lipid containing organelles from peroxidative damage acting in conjunction with vitamin E to maintain integrity of these membranes.

Etiology: selenium deficiency occurs in animals fed plant material grown on soils poor in the element. Vitamin E deficiency may be caused by a primary deficiency in the feed, or by the presence in the diet of polyunsaturated fatty acids that destroy the vitamin.

Clinical findings: The clinical effects are manifested principally in skeletal muscles but occur also in most other organs.

A. Acute enzootic muscular dystrophy

Sudden death, without prior illness, often after exercise, excitement, sudden onset of dullness, dyspnea, frothy, sometimes blood-stained nasal discharge in some.

• Many cases laterally recumbent

•Heart rate 150-200/minut, often grossly irregular

• In spite of treatment, death may occur after 6-12-hour course.

B. Sub acute enzootic muscular dystrophy (white muscle or stiff lamb disease)

• Sternal recumbency; Patient anxious to stand but most unable to do so
• Standing patients have rigidity, tremor, stumbling gait, easy falling
• Weakness and stiffness followed by paralysis
• Large, upper limb muscle masses symmetrically swollen, firm
• Myopathy as muscular degeneration or "white muscle disease"
• Retention of placenta in cattle
• Dyspnea, transient fever in some
• Heart rate moderately elevated

Diagnosis: history of diets, clinical signs, lesions in muscles and estimation of vitamin E. Respond to vitamin E therapy may confirm diagnosis.

Treatment: administration of vitamin E and selenium-containing medicine.

4.4. Diseases Caused By Chemical Agents and Poisonous Plants

A. Lead poisoning (plumbism)

Lead is one of the commonest causes of poisoning in dogs and farm animals, particularly cattle, sheep and horses especially young cattle.

Etiology: - accidental ingestion of sources of lead compounds or ingestion of feed, usually forage, containing lead from pollution of the environment, e.g. licking lead-bearing paint and metallic lead, lubrication grease, car battery, grazing beside rubbish dumps, discarded paint cans Small proportion is absorbed usually in the form of lead acetate or carbonate and deposited in the

liver and kidneys in acute poisoning and in the bones in chronic poisoning, a small amount also in the brain.

Clinical findings: -

Acute form: - is common in young animals.

• Staggery gait, muscle tremor, salivation, eye rolling, bellowing, blindness, hyperesthesia (oversensitive), convulsion, head pressing and death.

Sub acute form: - common in adult**.**

- Extreme depression, immobility, complete anorexia, blindness, staggery gait, tremor, hyperesthesia, salivation, teeth grinding, kicking at belly, complete ruminalatony, initial constipation followed by fetid, black diarrhea, recumbency, quiet death after 3-4-day course.

Treatment: If there is extensive tissue damage, particularly of the nervous system, treatment may not be successful.

The following treatments are used singly or in combination:

• Immediate relief of acute nervous signs in calves by IV pentobarbital sodium
• Emptying of rumen and reticulum completely, supplemented by wash-out
• Calcium versenate 150mg/kgBW
• IV or Sc (110mg/kgWB) Calcium disodium edetate (CaEDTA)
• Parenteral thiamin hydrochloride (2mg/kgWB) reduces deposition of lead in tissues.

B. Common salt poisoning (sodium chloride poisoning)

Definition: the ingestion of excessive quantities of sodium chloride causes inflammation of the alimentary tract with the production of gastroenteritis and diarrhea. It is called "water deprivation sodium ion toxicosis" because it can be resulted when excessive quantities of salt are ingested and intake of potable water is limited.

Lactating females are most susceptible.

Etiology: •is directly related to water consumption.

- Thirsty cattle get access to saline bore water
- Animals on low salt diet allowed access to ad lib salt
- Animals on normal salt intake (2% of ration) temporarily deprived of water
- Prepared feeds to housed cattle contain excess salt

Clinical findings: • In cattle and sheep signs of acute poisoning include: -

Salivation, increased thirst, vomiting (regurgitation), abdominal pain & diarrhea, opisthotonus, nystagmus, tremor, blindness, paresis, fetlock knuckling, recumbency, convulsions, death after course of 24 hours.

Treatment: • the toxic feed or water must be removed immediately

In advanced cases animals may be unable to drink and water may have to be administered by stomach tube

Symptomatic treatment includes alimentary tract sedatives when gastroenteritis is present and the provision of isotonic fluids when dehydration has occurred. When there is evidence of cerebral

edema it may be necessary to administer a sedative and cerebral decompression may be attempted by use of diuretics or hypertonic solutions injected parenterally.

C. Nitrite poisoning: Nitrate is a source of nitrite; nitrite poisoning causes a syndrome of respiratory distress because of the formation of methemoglobin (nitrite ion in contact with RBC reacts with hemoglobin to form a stable methemoglobin), which results in anemic anoxia.

Etiology:the nitrate may be reduced to nitrite in the plant before ingestion often hay in the stack (if hot and wet), overheated in the sun or damp for some time before feeding, by bacterial action or heating. The formation of methemoglobin results in anemic anoxia.

Clinical findings: dyspnea, tremor, stumbling gait, and mucosal cyanosis, fast, weak pulse.

Treatment: • methylene blue IV 1-2mg/kg as 1% solution

• Feeding chlortetracycline or sodium tungstate reduces nitrate to nitrite conversion.

D. Bracken fern poisoning

In ruminants the effect is depression of bone marrow activity with pancytopenia expressed primarily as ecchymotic hemorrhages and often followed by bacterial invasion of tissues. There is also a relation between bracken and the disease enzootic hematuria. Carcinomas of the intestine and urinary bladder in sheep and cattle have also been attributed to the ingestion of bracken for long periods. It is a retinal degenerant that causes bright blindness.

Clinical findings:

a. Pancytopenic disease of cattle: there is an access to bracken for from 2 to 8 weeks. Initially there is loss of condition and dryness and slackness of the skin. Clinical signs occur suddenly and include high fever (40.5-43c), dysentery or melena, bleeding from the nose, eyes and vagina, and drooling of saliva, nasolabial ulcers, petechiae in mucosae and skin, death after 1-3 days in most.
b. Enzootic hematuria: high incidence of bladder carcinomas, intermittent high-volume hematuria, hemorrhagicanemia, discrete, firm lesions palpable rectally in bladder wall, entire wall thickened in advanced casts, cystitis a common complication.
c. Bright blindness of sheep: in sheep on bracken for years, blind but bright, alert, pupils dilate, poor light, menace reflexes, retinal degeneration, leukopenia.
d. Alimentary tract neoplasia: carcinomas in sheep on long-term grazing bracken pasture, lesions in small intestine, liver, mandible, pharynx, esophagus of cattle.

Diagnosis: based on pasture, feeding, season, clinical findings, especially high fever, sudden onset in acute form, large hemorrhage.

Treatment: thiamine 50-100mg once a day for 6 days, batyl alcohol (to stimulate bone marrow function) plus antibiotics (to control secondary complication), vitamin B complex IM as a supportive treatment to stimulate appetite.

E. Fluorine poisoning

Fluorosis is a chronic disease caused by the continued ingestion of small but toxic amount of fluorine in the diet (drinking water). It is characterized by mottling and excessive wear of developing teeth and osteoporosis. Acute fluorine poisoning usually occurs as a result of the inhalation of fluorine-containing gases or accidental administration of large amount of fluoride

and is manifested by gastroenteritis. Irrespective of treatment used, no improvement in dental or osseous lesions can be anticipated but amelioration of the other clinical signs may occur.

4.5. Allergy and anaphylaxis

Trauma/wound, stress

4.3.1 Diseases Due To Physical Agents:

A. Heat (sun) stroke (Hyperthermia or heat stress): It is a disturbance of the heat regulating mechanism of the body due to high environmental temperature, high humidity and inadequate ventilation.

Etiology: • prolonged exposure to high environmental temperature

- Predisposing factors: physical effort (muscle exertion), obesity and stagnation of the air.
- Animals like dog, suffer from hyperthermia due to their inability to sweat. In addition sheep with fleece and large dense cattle are especially prone to heat stroke.

Clinical findings: elevation of temperature

- Increased heart rate and respiratory rate
- Sweating and restlessness
- Weakness, depression, panting,, frothy discharge from mouth and nostrils, tachycardia, protrusion of tongue, congestion of mucus membrane of conjunctiva, cardiac arrhythmia, muscle tremor, coma and death.
- In pregnant animals hyperthermia may lead to abortion

Treatment: •cold applications like immersion in water or spraying of water and rectal enema.

- Provision of adequate drinking water, shelter and ventilation.
- IV administration of isotonic saline solutions

B. Cold (over-cooling, hypothermia)

It is a disturbance in thermo regulation accompanied by drop in body temperature below the normal level.

Etiology: •increase in heat loss due to exposure to cold stress; wet skin exposed to high air current

• Decrease in heat production or combination of both factors.

-Newborn animals born in cold weather and their mother is incapable to lick it dry.

- Emaciated and malnourished animals are more susceptible to over-cooling**.**

Treatment: •Adequate covering with sack or blanket

- Dextrose 5-10% solution IV in severe cases
- Corticosteroid preparation to prevent shock.

C. Burn

Definition: the destruction of epithelium or deeper tissues by direct heat, radiant heat, flames, electricity or corrosive chemicals. Destruction of skin causes fluid loss, entry of bacteria, dehydration, toxemia, and shock.

Clinical findings: Reddening of the skin, formation of vesicles and blister, diffuse edema of the skin and subcutaneous tissue, charring and devitalization of tissue, sloughing of the

skin, constant exudation or effusion of serum, considerable loss of protein and fluid, reluctant to move, shock, and implications including infection

Treatment: •if burns are severe and involve more than 50% of the body, euthanasia should be considered.

- In less severe and small area burns, initially the application of cold packs may reduce pain and edema, clipping of the hair over and around the burn, remove the sloughing tissues and debris and clean with iodine or saline solutions, local application of antibacterial creams e.g. silver sulfadizine.

D. Trauma

It is a condition in which part of the body is damaged by wound or injury. Depending on its nature trauma can be classified in to: -

1. Blunt trauma: - is commonly associated with thoracic and abdominal bleeding, organ rapture, fractures and neurological injuries.
2. Penetrating trauma: - is localized to the path of penetrating object, e.g. animal bite, gunshot etc.

Etiology: sharp objects, car accident, gunshot, animal bite, etc.

Diagnosis and treatment: -The traumatized animal should be approached as if multiple injuries are present. The neck and spine should be immobilized until a thorough examination for spinal fracture, thoracic auscultation and abdominal palpation is done.

- Active hemorrhage should be stopped with pressure.
- Bandages must support fractures of the extremities.
- Keep careful monitoring in the 12-24 hours after the trauma occurred, which would allow early detection of potentially life threatening complications.