CHAPTER 1: INTRODUCTION

1.1 Definitions

Health: The state of health implies much more than freedom from disease and good health may be defined as the attainment and maintenance of the highest state of mental and bodily vigor of which any given individual is capable.

Animal Disease:
- Any abnormality of animal bodily structure or function.
- Any deviation from normal physical & physiological condition
- Can also be defined as "a disturbance of all or part of the biological functioning of the animal"

1.2 Livestock disease

❖ Importance of livestock
1) As food elements such as milk, meat, eggs etc
2) To generate cash for owners following sell of animal & animal products
3) To get dung to use as source of fertilizer & fuel for fire.
4) To generate draught power
5) To fulfill cultural obligations such as paying dowry
6) To serve as bank
To top of these, livestock contribute 16% of GDP and 14% of the Ethiopia foreign exchange earnings through export.

❖ Constraints of livestock productivity
- Inadequate feeds and water (quality & quantity)
- Inadequate management
- Prevalent of livestock diseases and poor veterinary service
- Low genetic potential with respect to productivity

❖ General impact of livestock disease in Ethiopia
Impacts of livestock diseases
a) Loss of productivity through reduced feed intake & reduced efficiency of feed utilization.
b) Loss livestock through mortality of breeding and productive animals
c) Treatment and professional service cost.
d) Reduce/lack of draught power, thereby influence crop production  
e) Public health hazards (zoonotic diseases)  
➤ Those elicits that livestock diseases cause larger economic and health importance. Thus, economic effective prevention & control measures should to be implemented in livestock production so as to minimize their impacts.

1.3 General concept of animal disease

❖ Classification of animal diseases & some terminologies  
• According to mode of ORIGIN/GENESIS  
a) Hereditary disease: those diseases which are transmitted to the offspring through sire or dam eg. Hypotrichosis (hairlessness), haemophilia, syndactyly (mulefoot) etc  
b) Congenital disease: Diseases which are acquired during intra-uterine life of an individual and are appreciable at birth. Eg.  
c) Acquired disease: are neither hereditary nor congenital but are contracted during entire life span of an individual.

• According to system involved  
a) Localized disease: diseases confined to a particular spot or organ/part  
   eg. Glossitis, localized abscess  
b) Generalized disease: diseases, which affect most or all parts of a body  
   eg. Toxaemia, septicemia

• According to SPECIFIC CAUSE  
a) Specific diseases: Caused by specific pathogen or factor.  
   ➤ Infectious causes eg. bacterial, viral, fungal, parasitic diseases  
   ➤ Non-infectious causes Eg. Milk fever, ketosis  
b) Non-Specific disease: those diseases whose causes are indefinite or multiple.  
   Eg. Diarrhea, septicemia

• According to CLINICAL MANIFESTATION  
a) Acute disease: Such type of disease is characterized by a sudden onset & comparatively short courses with sever manifestation and that runs its course quickly  
   Generally illness may prevail for 3-14 days. E.g. FMD, anthrax  
b) Per-acute disease: duration of disease is shorter than acute disease illness lasts for few hours to 48 hrs. eg. Per-acute mastitis  
c) Sub-acute disease: diseases whose onset & severity is lesser than acute on. Usually it has a course of 2-4 weeks  
   eg. Sub-acute mastitis  
d) Chronic disease: Diseases, which has got a protracted course illness which lasts 1 or two months or even more.eg. Tuberculosis, paratuberculosis  
e) Carrier: it means a form of interrelation ship between the micro organism& the animal body without manifesting an obvious disease.

• According to the INTENSITY & SPREAD OF DISEASE  
a) Sporadic disease: those diseases occur occasionally in animal population. Eg. FMD outbreaks in United Kingdom  
b) Enzootic disease: donates an outbreak of disease among animals in a definite area or particular district. Eg. Enzootic hematuria in cattle  
c) Epizootic disease: diseases which affect large population of animals in large area. The disease spreads quickly & is capable of covering a wide area. Eg.  
d) Endemic disease: is a special form of spread of infectious disease in which disease is retained for a long time in some locality affecting a large number of animals of particular species. Eg. Anthrax, blackleg  
e) Panzootic disease: When the epidemic reaches usually large size in some country or spreads over many countries or even continents. eg. Influenza, Rinder pest.
1.4 Routes of disease transmission

The main routes of disease transmission are:

1) **Ingestion**: Ingestion of feed & water contaminated with the discharged organism from infected animal eg. FMD, Anthrax, Blackleg
2) **Inhalation**: inhalation of infectious organism that has been discharged into air by infected animal. Eg. CCPP, CBPP, Newcastle disease
3) **Infection through skin** usually by contamination of cuts, abrasions, etc. Eg. Rabies
4) **Infection from fomites**: fomites are any objects (inanimate) which can convey infectious organisms. eg. Sheep & goat pox, FMD
5) **Venereal & congenital infection** by coitus/mating. Eg. Trichomoniasis, Dourine, Brucellosis
6) **Arthropod borne diseases**: Biting arthropods spreads many important infectious diseases from animal to animal eg. Trypanosomosis, Rift valley fever, Lumpy skin disease

1.5. Terminologies

1. **Infection**: invasion of living organism (host) by another organism (agent)
   → **Infectious disease**: is one in which an animal is invaded by a foreign organism such as virus bacterium, fungi etc. from another infected animal.
2. **Contagious disease**: transmitted by direct contact or indirect contact in which the infectious organism can survive outside of the animal & be picked up from the environment. eg. External parasites = unable to fly Eg. lice & mite, most fungal infections Eg. ringworm.
3. **Diagnosis**: is an art of determination of the cause of disease.
4. **Etiology**: refers to study of cause of disease
5. **Symptom**: outward manifestation of the disease that can be recognized the layman.
6. **Clinical sign**: Outward manifestation that can be recognized by the professional only.
7. **Lesion**: is the structural change observed in organ or part of it.
8. **Prognosis**: it is the forecast of the probable outcome (course & termination) of the disease process.
9. **Treatment**: Management & care of a patient or combating of diseases or disorders.
   - Use of medicine to try to cure/make better

**Reading assignment**: Read about the following terminologies: *Hyperthermia, Fever, Hypothermia, Toxaemia, Septicemia*

1.6 Period and course of disease

![Diagram of disease timeline](image_url)

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Incubation period: lasts from beginning of entrance of infective agent to the manifestation of detectable symptoms.
- Ranges from several minutes to several months.

1. **Prodromal period:** lasts from discovery of the first sign of the disease to its complete manifestations.
   ✓ Manifestations such as depression, in appetite, rise in temperature etc (general clinical signs).

2. **Period of manifestation:** usually follows "2" prodromal period
   - It is a period of marked development of Clinical manifestations.
   - The manifestation helps the clinician to diagnose the disease (specific clinical signs).

3. **Recovery:** restoration of body function which can be either:
   - Complete recovery
   - Incomplete

4. **Relapse:** means recurrence of the disease after clinical recovery during period of convalescence.
   - It may be due to the break in the immune competency of the host as a result of exposure, exertion & errors in the diet.

5. **Complications:** in some cases, primary disease causes a weakness of the body, which then becomes susceptible to other diseases called secondary complication.

6. **Convalescence:** a state through which a patient passes after a prolonged illness till it regains normal health & optimum power of production.

7. **Death:** if the animal is unable to adjust the altered conditions of existence, its adaptation mechanism become exhausted and unable to continue life.

   Types:
   - local death: death of part of the body only (necrosis)
   - Somatic death: death of entire body

**CHAPTER 2: PRINCIPLES OF DISEASE TREATMENT, CONTROL, PREVENTION & ERADICATION**

**2.1. Principles of Treatment /Therapeutic Measure**

Treatment depends on general care & nursing which are very important for rapid recovery of diseased animal. Treatment may be classified as:

I. **General treatment**
   - Include measures to be taken to combat certain complications during the course of disease & to resuscitate the vitality of the animal.
   - It includes:
     a) Fluid and electrolyte therapy- for dehydration
        eg. Dextrose with NaCl
     b) Mechanical treatment
        eg. Exercise, massage, traction etc
     c) Physical treatment
        eg. Use of heat, electricity, x-rays etc
     d) Dietetic treatment: Therapeutic nutrition

II. **Specific treatment**
   - Means administration of specific curative drug against certain diseases.
   - Only possible when the specific etiology of disease is diagnosed.
   eg. - Ca-therapy for milk fever
        - Specific antihelmintics for helminthiasis
        - Antibacterial for bacterial infection

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Control: is the reduction of mortality & morbidity from disease by interfering with the unrestricted occurrence of the disease whatever its cause. It is an ongoing process.

- Reduce the disease prevalence
- Involves both treatment & prevention measures

→ Effective control of infectious diseases is dependent on good knowledge of etiology & epidemiological characteristic (distribution in age, mortality & morbidity, rate, seasonality of occurrence etc)

Prevention: refers to the act undertaken so as to stop the disease occurrence or reduce its impacts if it occurs

Eradication: is reduction of the infectious diseases prevalence in a specified area to a level at which transmission does not occur

Disease control methods

1. Quarantine
   - Segregation of apparently healthy animals (brought into the herd for 1st time) which have been exposed to risk of infection from healthy & unexposed animals.
   - Quarantine period depends on incubation period of the disease (usually 30-60 days)
   - It could be up to 6months sometimes.
   - Objective of quarantine: to prevent the introduction of new diseases into the farm together with the new entrant animals.

2. Isolation
   - Means segregation of infected /suspected to be affected a contagious disease from the apparently healthy animals (why?).
   - Attendants working on sick animals & equipments used for them should not be used for healthy stock

3. Sanitation
   → The causative organism of some disease can be found in all parts of the carcasses of animals that have died from those diseases. Therefore, carcasses should be disposed off as quickly by burying deeply and burning to avoid further spreading. The place where animal die must be disinfected with appropriate chemicals or should be burned.

   → Abundant straw bedding is provided to dispose urine & introduce air in deep litter
   → Drainage must also be adequate to drain liquids away from the heap.
   → Dung must be cleaned regularly
   → Supply clean, fresh water

4. Disinfection and sterilization
   - Disinfection: destruction of pathogenic micro organism from place/equipments

METHODS

- Heat (burning for bedding, litter, brushes etc)
- Radiation
- Chemical substances
  Eg. 10% formalin, boric acid, phenols acids, alcohols etc

Sterilization: refers to complete destruction of all forms of life especially microorganisms by some chemical or physical means.

For using chemical disinfectants manufacturer’s instructions should be followed especially:
1) Many disinfectants have to be diluted in water & the correct concentration is essential.
2) Mixing disinfectants should be avoided as they may make some disinfectors ineffective.
3) Some disinfectants become ineffective if they contain a buildup of dirt & organic matter.
Disinfection of animal house

a) Under ordinary conditions:
- Daily scrubbing & washing of house & action of sunlight falling in house is sufficient to keep it germ free.
b) When disease outbreak has occurred: disinfection is a must for this
a) dung, litter etc should first be disinfected in situ by sprinkling of disinfectant
b) If the floor is of earth the top 10cm earth should be removed & disposed off along with litter.
- After that the place should be scrubbed & washed 4% hot washing soda solution (i.e. 4 kg washing soda in 100 lit boiling water)
- Disinfectant solution should then be sprayed & left so to act for 24 hrs.
→ The house is washed with clean water & left to dry by wind & sunlight.
⇒ The house is then fit for housing healthy animals.

5. Vaccination
- Means artificially building up animal body immunity against specific infectious diseases by injecting biological agents called vaccine.
- Vaccine: is a suspension or freeze dried live, killed or attenuated micro-organisms (bacteria, virus) or product derived from them (Toxin, toxoid) which when injected stimulate the production of antibodies against a disease.
- Types of Vaccines:
  a) Inactivated (killed vaccine): these are made by irradiation or other method
     - Are generally safest but they are liable to stimulate a relatively weak immune response and may have to be repeated at regular interval to produce a useful immunity (disadvantage)
  b) Attenuated vaccine/live vaccine:
     - These are based on live organisms which have been altered so that if inoculated into an animal they produce an immune response but cause no disease.
     - Attenuation can be achieved by growing the organism in a laboratory culture, by transferring the organism many times (passaging) through a series of animals.
     - Such vaccines are generally more effective than killed vaccine but they generally require more careful handling (disadvantage)
       ✓ They have to be kept at refrigerator to or even deep frozen "Cold chain"
  c) Toxoids:
     - Some pathogenic bacteria produce their harmful effect by secretion of exotoxins
     - Vaccines called toxoids have been developed against the toxin rather than the causative organism.
     - Toxoids are toxins, which have been rendered non-toxic & usually produced by growing organism in laboratory culture & treating the toxin produced with a chemical Eg. Formaldehyde
  d) Mass prophylactic treatment
    - Use of drugs as a means of disease prevention is used to combat diseases for which vaccine is not available.
      Eg. Pasteurellosis, blackleg (but vaccine is available for these disease), trypanosomosis

6. Selective breeding for resistance:
- Genetic improvement of susceptible animal breeds by cross breeding program with resistant breeds of animals.

7. Elimination of carriers
Generally, convalescent animals are carrier for a short duration. In some diseases, however, the carrier state may remain for years & the animal become a potential danger to susceptible animals. Eg. Tuberculosis, brucellosis

8. Depopulation (if economy allows): total elimination of animals from the herd whenever the disease is chronic, not treatable & fast spreading.

9. Treatment of sick animals: to prevent suffering, death & economic losses

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CHAPTER 3: INFECTIOUS DISEASES

3.1 Cause of disease

According to the cause disease is broadly divided in to two: infectious and non-infectious causes. Infectious diseases are those that are caused by living agents these include microbial cause. Microbes (micro-organisms) are unicellular organisms that cause disease which comprise bacteria, virus, protozoa, fungus etc. Non-infectious causes include agents which are non-living such as those related with feed (nutrient), chemicals agents, physical agents, metabolic disturbance etc. Eg. milk fever (calcium deficiency), ketosis (glucose deficiency), magnesium deficiency, protein deficiency, lead poisoning, etc.

The infectious diseases are of major importance in agricultural animals. Accordingly the bacterial, viral, fungal, protozoal and parasitic diseases account for a major portion of loss in livestock. The infectious diseases are capable of affecting many animals in a short period of time and the case fatality rate in some diseases can be very high and similarly the economic loss. Certain infectious diseases, especially the viral diseases, are endemic in some countries and pose a threat to other countries considered to be free of them.

3.2 Infectious diseases caused by bacteria

3.2.1. Anthrax

Anthrax is a per-acute or acute infectious septicemic disease affecting all animals, but specially cattle, sheep, goats, pigs, horses and man. Carnivores are more resistant and birds rarely affected. The disease is characterized by sudden death and black tarry exudates from the natural orifices.

Etiology: Bacillus anthracis causes the disease. Bacillus anthracis is gram positive spore forming bacteria that can persist in the soil in warm moist climate for long periods. The spores are resistant to most external influences, normal environmental temperatures and standard disinfectants. The spore withstands 165 °C dry heat for 1 hour and 100 °C moist heat for 5 minutes.

Distribution: Anthrax occurs all over the world, particularly in warm countries, where the ready production of highly resistant spores favors the persistence of contamination in soil and water. Anthrax outbreak occurs irregularly and is commonly associated with neutral or alkaline soils where the spores revert to the vegetative form and multiply to infectious levels if environmental conditions of soil, moisture, temperature, and nutrition are optimal.

Transmission: Animals are usually infected by ingestion of material containing spores or virulent bacilli. but biting flies can infect horses and carnivores feeding on infected carcasses readily spread the disease to other areas. The soil around an open infected carcass become heavily contaminated with spores, and may remain a source of infection for up to 20 years. Where carcass remains unopened, however, the bacilli, being protected from exposures to air, don’t so readily form spores and the ensuing putrefaction rapidly destroy them.

Importance: Anthrax is important to the livestock industry, in certain populations of wildlife and humans, especially those who are occupationally exposed.

- Vernacular/Local name: - “Abasenga”, Afrit

Man may contract the disease in three ways.

- By inoculation, through cuts or abrasions while slaughtering an infected animal or conducting a postmortem examination.
- By inhalation of dry spores. This gives rise to the so-called ‘wool sorters’ disease.

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• By ingestion: Consumption of raw or insufficiently cooked meat of an animal infected with Anthrax, usually after an emergency slaughter.

**Pathogenesis:** Ingestion of spore → local lymph nodes→ proliferation → lymphatic vessels→ blood stream → septicemia → lethal toxin → edema and tissue damage → death due to shock, renal failure and anoxia.

**Clinical signs:** The incubation period range from 1-2 weeks. The clinical course ranges from peracute to chronic. Peracute form is characterized by sudden onset and a rapidly fatal course. Acute form is mainly seen in cattle and sheep. There is rise in body temperature, excitement, depression, staggering, and rumination ceases, milk production is reduced, pregnant animals may abort and there is bloody discharge from natural body orifices. Chronic form is seen in horses and pigs, and is characterized by localized, sub-cutaneous edematous swelling around the throat, neck and thorax.

The carcass of an animal dead of anthrax should not be opened because the anthrax bacilli form spores when exposed to free oxygen. A carcass opened by mistake show the following changes:-
- Rigor mortis is frequently absent.
- Dark blood oozing from the mouth, nostrils and anus with bloating.
- Rapid body decomposition.

In endemic areas, sudden death, oozing of blood from body orifices and rapid distension of the carcass should arouse suspicion of anthrax.

**Diagnosis:** Anthrax should be suspected if an animal dies suddenly, dark bloody exudates come out through the natural body orifices and rapid distension of the carcass. Laboratory examination of stained blood smear from the ear vein usually confirms the presence of anthrax provided the smears are taken immediately after death.

Visualization of the capsulated bacilli, usually in large numbers, in a blood smear stained with polychrome methylene blue (M’Fadyean reaction) is fully diagnostic. Isolation of the bacteria (culture) from blood or tissues of a recently dead animal, using nutrient agar incubated aerobically at 37 °C is essential.

**Treatment:** Severely ill animals are unlikely to recover but in the early stages, recovery can be anticipated if the correct treatment is provided. Penicillin has had considerable vogue, but streptomycin is much more effective. Oxytetracycline has also good in the treatment of clinical cases after vaccination in cattle and sheep. Antiserum, if available, should also be administered for at least 5 days in doses of 100-250 mL daily but it is expensive.

**Control:** Vaccination. Anthrax of livestock can be controlled largely by prophylactic vaccination of all grazing animals in the endemic area. The live Sterne-strain spore vaccine is now used universally. It is safe to use in all species, can readily be prepared in the laboratory (many tropical countries including Ethiopia now produce their own stocks. Vaccination should be done 2-4 weeks prior to the season when outbreaks may be expected.

Control procedures, beside therapy and immunization (to contain the disease and prevent its spread) include:
- Notification to the appropriate regulatory official.
- Rigid enforcement of quarantine.
- Prompt disposal of dead animal and contaminated material by burning or deep burial.
- Isolation of sick animal and removal of well animals from the contaminated areas.
- Disinfection of tables, pens, milking barns, and equipments used on infected livestock.
- Control of scavengers that feed on animals dead of the disease.
- Observation of general sanitary procedures for persons who contact diseased animals, for their own safety and to prevent spread of the disease.

3.2.2. Blackleg

**Blackleg** is an acute, infectious disease of cattle and sheep characterized by inflammation of muscle accompanied by emphysematous swelling usually in the heavy muscles, severe toxemia and high mortality.

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**Etiology:** *Clostridium chauveoi* (spore forming anaerobic bacteria). The spore being highly resistant to heat (10 minutes at 120 °C) and standard disinfectants can survive in soil.

**Epidemiology:** The majority of cases of blackleg occur in cattle between the age of 6 months and 2 years. Blackleg is common in Ethiopia during dry periods of the year and is locally known as ‘abagorba’ or ‘koreba’. The mode of infection is by ingestion

**Pathogenesis:** The bacteria, which cause the disease, produce toxins, which lead to necrotizing myositis, systemic toxemia and death.

**Clinical signs:** the period of incubation period is 2-5 days. Affected animals are frequently found dead with swelling of the quarters. Observations done at the onset of the disease will reveal the following signs:
- The animal become dull, stand apart from the others with the head depressed.
- Cease to feed and ruminate.
- Increased body temperature and respiration.
- Lameness due to affection of muscle.
- Hot swelling of the upper portion of the legs. The swelling is painful with crepitating sound on palpation.
- The course of the disease is fast; death may be preceded by blood stained discharge from the bowel and nose.

**Diagnosis:** The occurrence of a rapidly fatal febrile disease in well-nourished young cattle, particularly of the beef breeds, with crepitant swelling of the heavy muscles suggests Blackleg. The affected muscle is dark red to black, dry and spongy, has sweetish odor, and is infiltrated with small bubbles. Confirmation of a field diagnosis can be made by laboratory examination of tissue specimens taken as soon as after death.

**Treatment:** Good results are obtained with antibiotics (penicillin, oxytetracycline, chlortetracycline) if rapid treatment follows early diagnosis.

**Control:** control is by vaccination. On farms where the disease is enzootic, annual vaccination of all cattle is essential. In an outbreak all cattle in the remainder of the herd should be vaccinated immediately and injected with penicillin. If antibiotics are not given, new cases of blackleg may occur for up to 14 days until immunity develops. Hygiene i.e., cleaning and treatment of all traumatic wounds with antiseptics (alcohol, iodine etc.) until the wounds are healed in case of sheep is important. Carcass of animals dying of blackleg must be destroyed by burning or deep burial to limit soil contamination.

### 3.2.3. Enterotoxemia

*Clostridium perfringens* resides in the soil and intestinal tract of domestic animals and can produce a number of toxins that result in enteric and histotoxic disease. *C. perfringens* isolates are classified into one of five types these are A, B, C, D and E (of these only type B, C, and D are important) depending on their ability to produce the four major lethal toxins: the alpha, beta, epsilon, and iota toxins. The activities of these major lethal toxins are the basis of the pathogenesis of the classical enterotoxemias attributed to this organism and described below.

*Clostridium perfringens* type B enterotoxaemia causes lamb dysentery, scour and lamb diarrhea. *Clostridium perfringens* type C causes struck, hemorrhagic enterotoxaemia, necrotic enteritis in sheep, hemorrhagic enteritis or enterotoxaemia in calves, necrohaemorrhagic enterocolitis in foals and bloody scour in piglets. *Clostridium perfringens* type D causes pulpy kidney disease, enterotoxaemia, and overeating disease.

**Occurrence:** Enterotoxaemia is triggered by mistakes in feed management and the influences of weather and may occur as well in both intensive and extensive animal production systems of temperate and tropical climate.

**Clinical features:** The course of enterotoxaemia is mostly peracute. The animals die suddenly on the pasture without having shown previous symptoms of a disease. Diarrhea and symptoms of abdominal pain are only occasionally observed. Signs of nervous disorders, like excitation, ataxia, blindness, tooth grinding and convulsions may appear;
they are typical indication for lesions in the central nervous system. The animals may walk in circle. The temperature may rise or be reduced to subnormal. The prognosis is unfavorable, and the animals die with violent abdominal pain and tonic-clonic convulsion.

**Diagnosis:** Clostridial enterotoxaemia has to be suspected whenever there is a connection between a change in feeding and sudden death. The laboratory diagnosis has to be accomplished by demonstrating the toxins in the intestinal contents of dead animals and serum of recovered animals.

**Treatment:** If enterotoxaemia is recognized on time, it can be treated. High doses of tetracyclines (oxytetracycline 10 mg/kg body weight) are effective intravenously. In valuable animals, administration of homologous hyper immune sera is recommended.

**Control:** Preventing sudden changes of pasture, the provision of feed containing structurally crude fiber and the gradual adaptation of the animals to new feed which is rich in nutrients, and/or changing the rotating pasture system to open pasturing are efficient means of disease control. Enterotoxaemia in lambs can be prevented by booster vaccination of the ewes. Use of site-and type-specific toxoids

### 3.2.4. Tetanus (*lock-jaw*)

Tetanus is an infectious, highly fatal, disease affecting all animals and man and characterized clinically by spasmodic tetany and hyperaesthesia. Horse and man are the most susceptible hosts. In Ethiopia the incidence of tetanus in horses and donkeys is more common than in any other species.

**Etiology:** *Clostridium tetani*, anaerobic bacteria with terminal and spherical spores that are found in the soil and intestinal tract.

**Transmission:** The portal of entry is usually through deep puncture wounds but the spores may lie dormant in the tissues for some time and produce clinical illness only when tissue conditions favor their proliferation. The bacteria can be introduced into the tissue through wounds following docking, castration etc. In horse is commonly associated with injuries, often accidental, causing deep penetrating wounds or during shoeing. The wounds, if they are deep, provide a good oxygen free (anaerobic) environment for germination of the contaminating tetanus spores. The disease in humans is usually associated with a history of personal injury.

**Pathogenesis:** The tetanus bacilli remain localized at their sites of introduction and don’t invade surrounding tissues. Local trauma → lowering of local oxygen tension → proliferation → production of neurotoxin → peripheral nerves → CNS → death (fixation of muscle of respiration).

**Clinical signs:** The incubation period varies between 1 and 3 weeks but occasionally as long as several months is possible. The clinical signs include: increased stiffness, developing to rigidity of the muscles, at first those of the head, so that there is inability to masticate. The ears become stiff, erect and immobile. Third eyelid is protruded, nostrils are dilated, and the jaw muscle becomes rigid (*lock-jaw*). Walking is difficult and turning is carried out without flexing of the body, the horse remaining rigid. A characteristic is the temporary recovery of the animal for a short period before there is a final severe spasm and death.

**Diagnosis:** The clinical signs and history of recent trauma are usually adequate for a diagnosis of tetanus. The diagnosis is confirmed by demonstrating the presence of tetanus toxin in the serum and Gram-stained smears and anaerobic culture from suspected wounds.

**Treatment:** The response to treatment in horses and sheep is poor but cattle frequently recover. The main principles in the treatment of tetanus are to eliminate the causative bacteria, neutralize residual toxin, relax the muscle tetany to avoid asphyxia, and maintain the relaxation until the toxin is eliminated or destroyed. Elimination of the organism is

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usually attempted by the **parenteral penicillin** in large doses and of toxin is by **antitoxin**. If the infection site is found, it should be treated locally but preferably only after antitoxin has been administered, because debridement, irrigation with hydrogen peroxide and the local application of penicillin may facilitate the absorption of the toxin. Keeping animals in dark and quiet places may be helpful.

**Control**

- Proper hygiene and cleanliness at castration and other surgical procedures.
- Surgical wounds should be well drained and disinfected, and kept free of contamination by dirt.
- Nails and wires should not be allowed to lie around premises where animals are kept.
- Injection of TAT (give protection for few weeks)
- Vaccination toxoid vaccine of high immunogenecity.

### 3.2.5. Tuberculosis

Tuberculosis is a chronic infectious disease characterized by the slow progressive development of "tubercles" in almost any organs of the body except the skeletal muscles. It is one of the major diseases of domestic animals and man.

**Etiology:** Tuberculosis is caused by *Mycobacterium bovis* (cattle), *Mycobacterium avium* (poultry) and *Mycobacterium tuberculosis* (man) species which is an acid fast bacterium.

**Epidemiology:** It is mainly through inhalation of infected droplets (housed animals), ingestion of infected discharges from open lesions in lymph nodes, faeces, milk, or urine (in open fields). Housing; high density, poor ventilation, stressors, immunosuppressor diseases are the factor increasing incidence of the tuberculosis. Animals may retain the microorganism for many years in the capsule, which may break down and release microorganisms into the surroundings, thus setting off an outbreak in a susceptible herd.

**Clinical signs:** The signs are weakness, anorexia, dyspnea, and emaciation and low-grade fluctuating fever. In mammals the organs of thoracic cavity (lung) is commonly involved, and there is an intermittent cough. The principal sign of tuberculosis commonly is chronic wasting or emaciation that occurs despite good nutrition and care. Involvement of reproductive tract leads to abortion and generalized tuberculosis in a full-term calf. Involvement of intestinal tract leads to chronic diarrhoea. Tuberculosis of the udder is important from a public health point of view, as milk from infected udders is a common cause of tuberculosis in human beings.

**Diagnosis:** Single or comparative intradermal tuberculin test; confirmation of diagnosis requires isolation and identification of the organism, which may take 4 to 8 weeks. Sputum or discharges may be examined by inoculation into guinea-pigs but improved cultural techniques are available.

**Treatment:** Treatment of human tuberculosis is with combination of isoniazid, streptomycin and para-aminosalicyli, Treatment of bovine tuberculosis is not recommended because it is not economical (DACA, 2006) & some treated animals remain carrier after recovery endangering the public health. However, in valuable animals’ isonicotinic acid hydrazine / isoniazid (INH) 20 mg/kg per os every 24 hrs for 8 weeks.

**Control**

- Tuberculin testing and slaughter of positive animals and their replacement by a build up from non- reactors (for rich countries).
- Tuberculin testing and the separation of reactors and non-reactors into infected and non-infected herds. Repeat testing every 6-month and the reactor herd is gradually reduced in size by slaughter as economics allow.

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Prevention of spread by strict isolation and hygiene, and prevention of further introduction of the disease into clean herd.

3.2.6. Paratuberculosis (Johne’s disease)

Paratuberculosis is chronic enteritis of ruminants caused by *Mycobacterium paratuberculosis*. *Mycobacterium paratuberculosis*, an acid-fast bacillus, causes enteritis in ruminants.

**Epidemiology:** Under natural conditions, the disease in cattle spreads by ingestion of *Mycobacterium paratuberculosis* from the contaminated environment. The disease persists in breeding stocks after the introduction of infected animals. A potential source of infection in calves is milk from infected cows or milk that is contaminated with the feces of diseased cattle. Paratuberculosis represents a significant economic loss to the dairy industry where the incidence is high due to culling of clinical animals from the herd and loss from subclinical paratuberculosis through an increased incidence of mastitis, decreased milk production, and increased calving intervals.

**Pathogenesis:** Infection usually is acquired relatively early in life. There follows a long incubation period during which intermittent fecal excretion of small numbers of organisms occurs. Some animals recover at this stage; in others, the organisms multiply and extensive intestinal lesions develop and cause overt clinical signs.

**Clinical signs:** The clinical signs of paratuberculosis are a slowly progressive wasting and diarrhea, which is intermittent at first, becoming progressively more severe until it is constantly present. Diarrhea is less common in small ruminants. Clinical paratuberculosis is characterized by profuse, non-treatable diarrhea, emaciation, and eventual death. During the course of the disease, the intestine becomes thickened and corrugated, disallowing proper absorption of nutrients. Although cattle usually become infected as calves by ingestion of feces and milk contaminated with *M. paratuberculosis* or by in utero transmission from infected dams, clinical signs of the disease may not manifest themselves until the animals reach 3 to 5 yr of age. Indeed, not all infected cattle will develop clinical disease. Stressors such as parturition, lactation, dietary deficiencies, or parasitemia may precipitate clinical disease.

**Diagnosis:** The diagnosis of paratuberculosis is made on clinical ground confirmed by the demonstration of *Mycobacterium paratuberculosis* in the feces by microscopy, culture, or by the use of DNA probe and Polymerase Chain Reaction (PCR). The detection of subclinical infection depends on the demonstration of delayed-type hypersensitivity to Johnin or to avian tuberculin, detection of specific antibodies by serology, or culture of *Mycobacterium paratuberculosis* from feces.

*Mycobacterium paratuberculosis* can be isolated from feces, mesenteric and ileo-cecal lymph nodes, thickened intestinal wall and, less frequently, the udder and the reproductive tracts of both sexes.

**Prevention and control:** No satisfactory treatment is known. The lack of accurate tests and the long incubation period of the diseased combine to make Johne’s disease difficult to control or impossible to eradicate the disease, other than by completely clearing a farm and then restocking, and to prevent the subsequent reintroduction of infected animals. Because intrauterine infection can occur, calves from dams that have or develop the disease should not be reared. Sanitary measures, including manure removal and pasture rotation is essential.

3.2.7. Pasteurelloses

The groups of microorganisms known as *pasteurellae* are responsible for a number of different diseases in cattle, sheep, goats, pigs and poultry generally termed as pasteurelloses. The most important ones are Hemorrhagic septicemia (cattle and other animals), pneumonic pasteurellosis or shipping fever (sheep and cattle) and avian pasteurellosis or Fowl cholera.
In cattle the disease named septicemic pasteurellosis (hemorrhagic septicemia), is commonly associated with infection by P. multocida, and is the classical disease with high mortality rate. Pneumonic pasteurellosis of cattle, commonly associated with infection by Mannheimia (formerly Pasteurella) haemolytica and also P. multocida type A, is also a common disease. Pasteurellosis of pigs this is usually associated with infection by P. multocida and is mainly pneumatic in form. Pasteurellosis of sheep and goats is usually associated with infection by M. haemolytica and, although it is often pneumatic in form, a septicemic form of the disease is not unusual, especially in lambs.

Epidemiology

Hemorrhagic septicemia occurs in cattle, yaks, camels, and water buffalo and to lesser extent, pigs and horses. It is considered economically important throughout the world including Africa. Animals of all ages are susceptible but the most susceptible age group is 6 months to 2 years of age. There is no difference in susceptibility between breeds. The incidence of disease is reduced significantly in areas where the vaccine is used. Both morbidity and case-fatality rates vary between 50% and 100%, and animals that recover require a long convalescence.

Pneumonic pasteurellosis of cattle, sheep and goat is a disease that occurs in ewes and young lambs in late spring and early summer and feeder lambs late summer. In pastured sheep outbreaks often associated with changes in climate or management. Outbreaks in housed sheep associated with poor ventilation. In cattle young growing cattle, especially recently weaned beef calves placed in feedlot. Can occur in nursing calves and mature cows. Stressors include transportation, mixing animals from many different sources, ineffective ventilation of housed animals. Commonly there is a history of stress in all groups of animals.

Risk factors

Animal risk factors: The disease occurs most commonly in young growing cattle from 6 months to 2 years of age but all age groups are susceptible. Calves and lambs that are non-immune to M. haemolytica are considered to be more susceptible to the disease than those that have serum neutralizing antibodies.

Environmental and management risk factors: The mixing of cattle from different sources is an important risk factor. Mixing of recently weaned beef calves from different sources at auction markets was associated with an increased risk of fatal fibrinous pneumonia in calves moved to feedlots. Confinement in drafty or humid and poorly ventilated barns, exposure to inclement weather, transport, fatigue and deprivation from feed and water are commonly followed by outbreaks of the disease in cattle.

Transmission: The disease is carried from one season to another by carrier animals. Pasteurella exist in nasopharynx region of healthy animals and from this spread to susceptible in-contact animals by droplet inhalation or by ingestion. Stress factors play a role in changing healthy carrier to disease state.

Clinical signs: High fever, anorexia, depression, increased respiration and profuse salivation (characteristics) are seen. Subcutaneous swelling at the throat, dewlap, and brisket and around the parotid region of the head is frequent manifestations. This leads to respiratory distress and sometimes even suffocation and protrusion of tongue. Hemorrhagic diarrhea, coughing and 80 - 90 % mortality are other manifestations of the disease.

In pneumonic pasteurellosis: Sudden deaths, acute bacterial bronchopneumonia, fever, toxemia, anorexia, abnormal lung sounds are characteristic.

Diagnosis: The season of the year, rapid course, and high herd incidence, with fever and edematous swellings indicate typical hemorrhagic septicemia. Identification of the organism by culture from specimen such as nasopharyngeal swabs and bronchoalveolar lavage reveal moderate agreement. Hemogram indicates severe infection and increase in fibrinogen. Lesion such as acute fibrinohemorrhagic pneumonia with pleuritis is also indicative.

Treatment: Antimicrobials

Control: Satisfactory economical control of the disease depends on the successful integration of management and perhaps the use of vaccines and antimicrobials. Thus the control program has to include:

- Management strategies to reduce stressors.
- Mass medication with antimicrobials of individual animals on arrival in the feedlot.
- Vaccines containing antigens of M. haemolyticaperior to stress period.

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3.2.8. Brucellosis (Bang’s disease)

Brucellosis is a contagious disease primarily affecting cattle, swine, sheep, goats and dogs, caused by bacteria of the genus *Brucella*. *Brucella* organisms primarily infect organs of reproduction. It is characterized by abortion in the female and to a lesser extent, orchitis and infection of the accessory sex glands in the males, and infertility in both sexes.

**Etiology:** The bacteria called Brucella and their principal farm animal hosts are *Brucella abortus* (cattle), *Brucella melitensis* (goats), *Brucella suis* (pigs), and *Brucella ovis* (sheep).

**Public health significance:** Brucellosis is more ignored in humans and most cases go undiagnosed and untreated, leading to considerable suffering for those affected. *B. abortus, B. melitensis* and *B. suis* infections can cause serious disease in human beings. The disease can be transmitted readily from animals to man and represents a real occupational hazard for veterinarians, slaughter man, and farmers. In man, infection due to *Brucella abortus* is contracted by drinking of raw infected cows' milk and by direct contact with infected fetuses, membranes, and discharges of aborting cows. The disease in man is known as "undulant fever" or "Malta fever."

**Epidemiology:** Aborting cattle discharge large numbers of *B. abortus* bacteria in the placenta, fetus and vaginal discharges. Outside the host, the organism can survive for up to several weeks and other cattle become infected by ingestion and sniffing of contaminated material, aborted fetuses and fetal membranes or licking vaginal discharges. Once an animal has aborted, it may remain infected and discharge large numbers of organisms at subsequent parturitions which can be normal. Calves can be infected in the womb before birth from infected dams. The organism can be excreted in milk (a common source of infection for human beings) and calves can also be infected by suckling infected dams. Call-hood infections usually disappear before puberty.

The occurrence and epidemiology of brucellosis are best understood for bovine brucellosis and to a lesser extent for ovine and caprine brucellosis but in pigs it is poorly understood.

**Pathogenesis:** *B. abortus* has a predilection for the pregnant uterus, udder, testicle and accessory male sex glands, lymph nodes, joint capsules, and bursae. After the initial invasion of the body, localization occurs initially in the lymph nodes draining the area and spreads to other lymphoid tissues, including the spleen and the mammary, iliac lymph nodes and finally the uterus. Erythritol is a substance produced by the fetus and capable of stimulating the growth of *B. abortus*, occurs naturally in greatest concentration in the placental and fetal fluids and is responsible for localization of the infection in these tissues. Invasion of the gravid uterus results in a severe ulcerative endometritis of the intercotyledonary spaces.

**Clinical signs:** Cattle of all ages and either sex can be infected with *B. abortus*. Infection is usually symptomless (Hunter, 1994); however, in pregnant females it causes abortion after the fifth month of pregnancy is the cardinal feature of the disease in cows. Retained placenta and metritis with the following period of genital discharge and infertility is common. Subcutaneous swellings containing infected fluid (hygroma) are quite common on the legs in African cattle. Brucellosis is a very serious disease in intensive cattle production system while it is less serious in extensive pastoral systems, where the chances of contact with an aborting or infected calving cow are reduced.

**Diagnosis:** Laboratory tests used in the diagnosis of brucellosis include isolation of the organism, PCR and serological tests for the presence of antibodies in blood, milk, whey, vaginal mucus, and seminal plasma.

Most infected animals are identifiable using the standard serological test (RBPT, CFT) but vaccination may confuse. For isolation of the bacteria organs and lymph nodes of the fetus, the placenta, milk, vaginal mucus, or uterine exudates is used.

**Control:** *Brucella* infections are usually persistent and even protracted intensive courses of antibiotic treatment may not eliminate the organism from infected tissues as it is intracellular.

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Vaccination of young stock age 4-8 months using *Brucella abortus* strain 19 vaccine and adults using K 45/20 strain vaccine. Hygienic measures, which include isolation of cows to abort, disposal of aborted fetus and discharges and disinfection of the area, separate and last milking of infected animals, boiling or pasteurization of milk and control or movement of animals of unknown disease status. During farm establishment introduce animals that are found to be negative for Brucellosis.

If the economic situation allows testing and slaughtering of infected animals is the best. This approach is costly and requires a high level of organization and administration but it has been applied effectively to eradicate *B. abortus* in cattle and *B. suis* in pigs from certain regions. In eradication programs, the level of disease may be first reduced by use of vaccines followed by detection and slaughter.

### 3.2.9. Mastitis

Mastitis is an inflammation of the cow's udder caused mainly by bacteria such as streptococci or staphylococci. Mastitis results in a sharp reduction in milk yield. If untreated, mastitis can result in the loss of the infected quarter or even death.

**Etiology:** Mastitis is mainly caused by infection with bacteria. Although over 135 microorganisms have been reported to cause the disease, *Staphylococcus aureus*, *Streptococcus agalactiae*, *Streptococcus ubris*, *Streptococcus dysgalactiae*, other *Streptococci*, *Aracanobacterium pyogenes*, *Mycoplasma* spp, *Nocardiaasteroides* and Coliforms are the most common agents.

**Risk factors:** The infection is usually caused by poor management practices such as incomplete milking of the udder, unwashed and dirty udders and teats, dirty milking area, milkers dirty hands and untreated cracks and sores on teats. Mammary infections are caused with a few exceptions by the penetration of microorganisms through the teat canal. From there the microorganisms extend into and infect the mammary tissue and cause mastitis. Following invasion of the teat canal, the severity of the mastitis depends on the species of the microorganisms involved, the stage of lactation and the physical condition of the mammary gland.

Mastitis can occur in adult cows and pregnant heifers at any time but animals are particularly susceptible at the beginning of lactation just after calving, and at the end of lactation when drying off.

Mastitis can be classified as contagious and environmental based on the source of the causative agent or peracute, acute or chronic based on course of infection and clinical or subclinical based on its manifestation.

**Clinical signs:** The clinical signs include: thick or clotted milk, reduced yield, swollen or inflammed udder or quarter, painful udder when touched and losses of appetite. The clinical signs of mastitis depend on the organisms involved. Systemic signs could also be observed. Subclinical mastitis is the most common and economically important.

**Diagnosis:** Diagnosis of mastitis is based on clinical sings, and identification of the pathogen from milk samples. Tests to detect sub-clinical mastitis include California Mastitis Test, or direct somatic cell count.

**Treatment:** Clinical cases are treated by infusion of antibiotic ointment into the affected quarter, which must be first stripped out by hand, and the outside thoroughly washed and dried. The end of the teat should be disinfected; the nozzle of the antibiotic tube gently inserted into the teat canal and the tube content is squeezed into the quarter. The end of the teat is then gently closed by hand and the udder massaged to ensure diffusion of the antibiotic. This is repeated for 3 days.

**Control:** Mastitis problems can be reduced by observing a few simple rules on milking practices and hygiene measures such as:-

- Wash hands before milking.
- Wash the cow’s udder thoroughly.
- Always use clean milking utensils.
Keep the milking area clean.
• Squeeze the teat don’t pull when handling for milking.
• Milk the first draw of milk into a strip cup, this will ensure early detection of mastitis.
• Teat disinfections by dipping the whole teat in an appropriate solution immediately after milking.
• Systematic treatment of quarter at drying off with effective long acting antibiotics.
• Culling of animals that don’t respond to treatment.

3.2.10. Salmonellosis

Salmonellosis is a disease of all animals caused by many species of salmonellae and characterized clinically by one or more of three major syndromes - septicemia, acute and chronic enteritis. The clinically normal carrier animal is a serious problem in all host species. Septicemic form is usually seen in calves, piglets, lambs and foals. Acute enteritis is common in adult cattle, sheep and horses and chronic enteritis is usually seen in growing pigs and occasionally in cattle.

**Etiology:** There are host specific and non-host specific salmonella serotypes. The host specific salmonellae include Salmonella Typhi (man), Salmonella Paratyphi (man), Salmonella Gallinarum (fowl), Salmonella Pullorum (fowl), Salmonella Abortusequi (horse), Salmonella Abortusovis (sheep), Salmonella Abortussuis (pig) and Salmonella Dublin (mainly in cattle). Non-host specific salmonella is Salmonella Typhimurium (man, cattle, sheep, pig, horse, fowl and rodents).

**Epidemiology:** In most instances of salmonellosis stress is a predisposing factor. Transmission is mainly through ingestion of contaminated feed and drinking materials.

**Clinical signs:** In septicemic form (in neonates) the symptoms are high fever, incoordination of gait and recumbency. Faeces may contain blood streaks. Tarsocarpal joints in calves may also be swollen (arthritis). Enteric form is seen commonly in adults and symptoms are high fever, severe watery diarrhoea with blood clots, later on temperature falls to normal or subnormal. In prolonged cases dehydration, severe emaciation and abortion is seen. Recovered animals frequently become carrier for life.

**Lesions:** Septicemic hemorrhages, fibrinohemorrhagic necrotic enteritis; enlarged mesenteric lymph nodes. Kidney petechiation in pigs. Foci of necrosis and thickened intestinal wall in chronic enteritis.

**Diagnosis:** Culture organism from feces blood, spleen, liver, lymph nodes; detect organism with special tests (biochemical tests); hematology for changes in leukocyte and clinical chemistry electrolyte changes

**Chicken salmonellosis**

Salmonella Gallinarum and Salmonella Pullorum cause avian salmonellosis, i.e., fowl typhoid and pullorum disease, respectively. Fowl suffering from these diseases shed the bacteria in their droppings and contamination of water and feed soon occurs. Eggs from carriers may often contain the bacteria and transmission through the egg (vertical) is common. Pullorum disease can be transmitted by infected (carrier) breeder hens through their eggs (vertical transmission). Chicks that hatch from such infected eggs will have typical pullorum disease (white diarrhoea) and high mortality. Infected chicks can also infect other chicks via droppings. Fowl typhoid is more a disease of adult chickens, with high mortality and morbidity. Horizontal transmission is important with fowl typhoid through infected droppings, dead bird carcasses, and infected clothing, shoes, utensils and other fomites. Species affected: Chickens, pheasants, ducks, geese and guinea fowl can contract both pullorum and fowl typhoid.
Clinical signs: Pullorum in chicks causes typical white bacillary diarrhoea, with pasted cloaca and high mortality. Infected adult breeders do not have clinical signs of the disease but have internal lesions in the ovary (mis-shaped, dark colored follicles).
Fowl typhoid in adult chickens causes listlessness and sulphur-coloured diarrhoea. The birds have generalized infection with swollen livers, spleens, and kidneys and haemorrhages in such tissues. Mortality is usually high, 25 to 60%.

Treatment and control: Treatment of pullorum disease will not bring about a cure and is undesirable from a standpoint of eradication. It is far more practical to control the disease by elimination of infected carrier breeder hens.

Blood testing of breeder chickens by the serum plate or tube agglutination test with suitable Salmonella Pullorum antigen will detect infected carrier birds, which can then be culled. Such control measures will stop the incidence of egg-transmitted pullorum disease. Treatment of fowl typhoid with drugs like sulphonamides, tetracyclines, or furazolidone, has been more or less successful however, infected carriers may remain after treatment. The best control method is eradication of infected birds. Vaccination for fowl typhoid with a special Salmonella gallinarum (GR strain) has been practiced in several countries but it should be discouraged in breeders when an eradication program is in operation.

Salmonellosis in humans is readily acquired by contact with animals. Stockmen and animal husbandry professionals become infected either by having close contact with diseased animals or, in the case of rural families, by drinking raw milk from infected cows. In stressed individuals and human societies of lower socioeconomic group clinical infections are featured by diarrhoea, vomiting and low-grade fever. Sometimes it progresses to dehydration, prostration, and death, especially in very young or old age.

3.2.11. Colibacillosis/ Escherichia coli infection/ Colisepticemia

Etiology: Only the species Escherichia coli (E. coli) belongs to the genus Escherichia that is a natural inhabitant of both the human and animal caecum and becomes pathogenic only under specific conditions. E. coli infections can become important especially in intensive production systems of the tropics in particular where the hygienic conditions of the premises of the animals are inadequate.

Epidemiology, Pathogenesis and Symptoms: E. coli causes diseases in young animals, especially in calves, lambs and piglets, which manifest themselves as septicaemia or enteritis. Inadequate keeping and feeding are usually the cause for the outbreaks of the disease. Badly ventilated dirty sheds as well as overfeeding may stimulate E. coli as a normal inhabitant of the intestine to become pathogenic. E. coli infection is often associated with other facultative pathogenic bacteria (Clostridia, Pasteurella) or viral infections. Different serovars may cause different symptoms. In adult animals, E. coli may produce localized infections of organs e.g. the urogenital tract and the udder. With the enteric forms (oedema disease in piglets, colidiarhhea in calves), the pathogens multiply mainly in the jejunum and bacteriemia does not occur. The enterotoxin produced by the pathogens causes dehydration of the body and enteritis through the release of liquid into the intestine.

Diagnosis: Whether the infection with E. coli has been the relevant cause of the disease of the animal is difficult to know because of the clinical and pathological features of the disease, neither is the cultural isolation of E. coli from specimens of the infected animal alone relevant. Serological diagnosis of the pathogenic serovars is possible.

Treatment: In principle, the E. coli infection can be treated with the same antimicrobial agents as are effective against salmonelloses. Chloramphenicol, streptomycin and tetracycline as well as most of sulphonamides are especially effective.

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Control: Diseases of young animals with the causal participation of *E. coli*, even if only opportunistic, can be prevented efficiently by organizing appropriate keeping, hygiene and balanced feeding. Attention should be paid in feeding colostrums to new-borne animals.

3.2.12. Dermatophilosis (*Streptothricosis*)

Dermatophilosis is an epidermal infection of cattle, horses, sheep, goats, wild ruminants and occasionally of human being, worldwide in distribution, but more prevalent in tropics. The lesions are characterized by an exudative dermatitis with scab formation. The disease is of considerable economic importance because of the damage of the hide subsequently derived from the animal, reduced performance of animals chronically affected, costs of control and the occasional mortality. Dermatophilosis is known to be endemic in cattle in Ethiopia and has recently been reported to be a threat to livestock production in the country. It is one of the causes of rejection of hide and skin due to poor quality.

Etiology: *Dermatophilus congolensis*. *Dermatophilus congolensis* is fungus like dimorphic organism that grows as branched filamentous mycelia containing dormant zoospores which are transformed by moisture to the infective stage of motile isolated cocci.

Transmission: The zoospores cannot penetrate into the intact epidermis with the normal protective layer of fatty acids. Factors such as prolonged wetting by rain, high humidity, high temperature and various ectoparasites that reduced the natural barrier of the integument influence the development, seasonal incidence and transmission of the disease. The disease is common during rainy seasons. Contact with infected animal leads to the spread of the disease. Ticks (particularly *Amblyomma* spp.) and biting flies (mainly *Musca* and *stomoxys* spp.) may act as mechanical vectors. Furthermore ticks prepare the necessary micro lesions as places of entry for the agent. Thus, the main distribution of the epizootic in the rainy seasons is explained by the fact that, on the one hand, infective zoospore are released from the moist skin and, on the other, insect populations multiply excessively during rainy periods.

Clinical signs: Skin lesions on sites where flies, ticks and thorn scrub damage are more likely to occur, i.e., the back, round the ears, on the shoulder or flank, the inner aspect of hind legs, the base of tail and the scrotum. The skin lesions are non-itching. Animals seriously affected lose condition, become weak and being unable to forage may die from starvation. The termination of humid and hot conditions often results in spontaneous recovery, the microorganism remaining quiescent on the skin until the following wet season when the disease progress once more. Clinically, the disease is characterized by erythema that becomes scaly and progresses to an exudative crusty lesion. When the crust is removed, a raw bleeding area is left, resembling a strawberry surface.

Diagnosis: is through Gram stain smears of the exudate; branched filaments forming packets up to eight coccoid cells wide are seen.

Treatment: High dose of oxytetracycline (20 mg/kg, IM, if required repeated after 3 to 5 days) or strepto-penicillin for five consecutive days. Wash the surface of the animal body with any of the following bactericidal substances not irritating the skin (2 -5% lime sulfur, 0.5% zinc sulfate, 0.03 % copper sulphate, 1.0 % peracetic acid, and 1% potassium aluminum sulfate ( alum)) for 3 to 5 days, then weekly until the lesion heals.

Control: There is no effective vaccine and control depends on prevention of skin damage through:-

- Proper control of tick damage by regular spraying or dipping with anti tick preparation.
- Housing of valuable and well-cared animals during rainy season.
- Isolation of infected animals and avoidance of contact with infected materials.
- Animals showing extensive lesions are best sent for slaughter to reduce infection of others.


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Leptospirosis is a contagious disease of animals and humans caused by infection with the Spirochete *Leptospira*. The disease is characterized by haemolytic crisis, nephritis, mastitis, abortions, stillbirths and reproductive failure in cattle and pigs, agalactia in sheep and goats, and ophthalmia in horses. Leptospirosis is an occupational hazard (zoonosis) of persons in close contact with animals, such as farmers, slaughterhouse workers, veterinarians etc usually associated with exposure to urine of infected animals.

**Etiology:** The pathogenic leptospires are classified into one species of *Leptospira interrogans* containing over 212 serovars arranged into 23 serogroups and *Leptospira interrogans* serovar Pomona is the commonest. *Leptospira* are long, very thin filamentous organisms varying in length. The degree of pathogenicity varies considerably with the host and the virulence of strains within a serovar. Serological evidence shows wide distribution of infection in irrigated areas of Ethiopia.

**Transmission:** Infection is commonly acquired from skin or mucus membrane contact with urine, and to a lesser extent by intake of urine – contaminated feed or water.

**Pathogenesis:** Leptospires are carried by the blood from the portals of entry to the liver where primary multiplication occurs. Liver lesions include focal necrosis, mononuclear cell infiltration in animals with marked icterus.

Bacteremia → *Leptospira* in: Brain → encephalitis
Kidney → acute edema hemorrhage in glomeruli
Chronic glomerular atrophy
Uterus → abortion
Mammary glands
Testicles

Secondary multiplication in kidney → and brain cause septicemia → hemoglobinuria due to extensive intravascular hemolysis. Localization in kidney → interstitial nephritis & prolonged leptospiuria → uremia → death

**Clinical findings:** In all animals the incubation period is from 3 – 7 days. Clinical signs may be severe, mild, or in apparent. Clinically fever, weakness, anorexia, conjunctivitis and anemia and in most severe cases, jaundice, pneumonia are frequent.

Acute leptospirosis should be suspected in the following cases: sudden onset of agalactia (in adult milking cattle and sheep); icterus and hemoglobinuria, especially in young animals; meningitis; and nephritis and hepatitis in dogs. Recovery from acute signs is generally associated with the appearances of circulating antibodies and the disappearance of leptospires from the blood). Chronic leptospirosis should be considered in the following cases: abortion (abortion occurs most frequently during the middle or last third of gestation), stillbirth, birth of weak offspring (may be premature); infertility; and cases of periodic ophthalmia in horses.

**Diagnosis:** Diagnosis of leptospiral infection poses considerable problems. Serological testing is the most widely used method, and the microscopic agglutination test (MAT) is the standard serological test. In the acute disease, during the febrile response, leptospires may be isolated from the blood during the first few days either in appropriate liquid or semisolid media or by inoculation of laboratory animals (weanling hamster). Urine samples are generally the most appropriate source of leptospires for either culturing or animal inoculation. Clinical diagnosis also may be confirmed by demonstrating the organism in sections of kidney and liver stained by the silver impregnation method (histopathology). Characteristic motility must be observed to provide positive identification under dark field microscope.

**Treatment:** Usually, initiation of antibiotic therapy as soon as possible to exposed animals in a herd will significantly reduce clinical signs and mortality. Administration of either dihydrostreptomycin at a dosage of 25 mg/kg or tetracyclines at 200 mg/lb of body weight daily for 3 to 5 days is preferable. With increasing severity response to antibiotic therapy decreases.

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Prevention and control: An understanding of the host-agent-environment interaction in domestic animals and wildlife of the region is imperative in developing a valid control program. In the tropics, extensive distribution of leptospires in domestic animals and wildlife provide many opportunities for transmission of a variety of leptospiral serovars in cattle. Complete confinement is the only procedure, which can assure reasonable protection from exposure for a herd of cattle. All ponds and streams are potential sources of leptospiral exposure. Rodent control is very important due to the high incidence of leptospires in rats and mice. All cattle introduced into a cattle herd should be serologically tested for all potential serovars prevalent in the region where the replacements originate. Clinically normal carrier cattle remain potentially the most likely source of a new infection.

Vaccination: As the protective antibodies induced by leptospiral bacterins are serovar specific, it is important to determine the serovars prevalent in the region. Bacterins should be administered to calves after 3 months of age and annually thereafter.

Damp areas should be drained or fenced and pens disinfected after use by infected animals. Because of the development of serological methods of diagnosis, of vaccination, and pharmaceutical elimination of the carrier state, it is now reasonable to attempt eradication of the disease from individual herds, and possibly from areas.

3.1.14. Actinomycosis (Lumpy-jaw)
Actinomycosis is a sporadic chronic infectious disease, mainly of cattle, characterized by swelling of mandible and maxilla, formation of abscesses and fistulous tracts.

**Cause:** Actinomyces bovis.

**Transmission:** Infection is believed to occur through small wounds in the mouth, which are caused by penetrating splinters of dry feed such as straw or foreign bodies. It may be carried from the primary lesion via the blood or lymph to internal organs. Infection may occur in young cattle by penetration of damaged buccal mucous membrane when the teeth are erupting.

**Clinical signs:** Initially there is a gradual development of large, hard swelling over the bones of the jaw. Later on an eruption occurs at one or more points on the surface of the skin covering and pus oozes out. Distortion of the bone and teeth make it difficult for the animal to masticate. There is usually no increase in temperature.

**Diagnosis:** Purulent discharges commonly contain ‘sulfur’ bodies which are granular in nature and, on microscopic examination, consist of club-like rosettes with a central mass of gram negative bacteria.

**Treatment:** Surgical removal, local injection of antibiotics (streptomycin)

3.2.15. Actinobacillosis (Wooden tongue)
Actinobacillosis is an infectious, usually sporadic disease, mainly of cattle and sheep, which is characterized by inflammation of soft tissues, notably the tongue in cattle, and the formation of granulomatous pus in the affected tissue. Small abscesses with a diffuse, extensive connective tissue proliferation are prominent features. The spread via lymphatics and invasion of local lymph nodes helps to distinguish *A. lignieresii* infection from that of actinomycosis.

**Etiology:** The disease is caused by a bacteria called *Actinobacillus lignieresii*, which is an extremely pleomorphic microorganism varying from a coccoid form to long filaments. It has a tendency to form in clumps in infected tissue. These clumps may be seen as very small brownish-white granules in pus collected from a typical abscess. The granules examined in pressed smears under the microscope are seen as an arrangement of club-like radiating bodies, similar to and easily confused with *Actinomyces bovis*. When these granules are broken down and stained by Gram’s Method it is seen that the microorganism is a uniform gram-negative rod compared to the filamentous gram-positive appearance of *Actinomyces bovis*.

**Epidemiology:** The disease affects mainly young adult cattle with most cases arising in cattle between one and three years of age. Cases are usually sporadic but multiple cases may arise locally. The age of cattle with the disease strongly suggests that the eruption of permanent teeth often creates the condition that are necessary for the organism to penetrate sub-epithelial tissues.

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Pathogenesis: *Actinobacillus lignieresii* is a common inhabitant of the normal alimentary tract and it is generally accepted that it gains access to tissues through breeches in the epithelial lining such as occur following trauma, ulceration or the eruption of permanent teeth.

Clinical signs: The tongue is the favorite site, although sometimes the cheek or esophagus may be affected. As the infection progresses, the tongue becomes hard and swollen. The animal is reluctant to eat, it drools and loses weight and often there is a secondary swelling in the throat. Frequently, the intermandibular space is swollen and hard and the sub-mandibular lymph nodes are enlarged.

Treatment: Circumscribed lesions may be treated by complete excision. The response to iodides is usually dramatic. Sulfonamides and a number of broad-spectrum antibiotics are effective as well. Streptomycin is possibly the drug of choice.

Control: Restriction of the spread of the disease is best implemented by quick treatment of affected animals and the prevention of contamination of pasture and feed troughs. Isolation or disposal of animals with discharging lesions is essential, although the disease does not spread readily unless predisposing environmental factors cause a high incidence of oral lacerations.

3.1.16. Foot rot
Foot rot is a disease of the feet of sheep, and sometimes goats, caused by infection with two bacteria, *Fusobacterium necrophorum* and *Bacteroides nodosus*. It occurs worldwide.

Transmission: Both bacteria are required to cause Foot rot, which spreads via contamination of pasture. *Fusobacterium necrophorum* occurs wherever there are domestic livestock and can survive for long periods in the feet of sheep, goats and other domestic livestock, as well as on pasture, soil, etc. *Fusobacterium necrophorum* infection causes "scald" which itself may cause lameness. True Foot rot occurs when the second bacterium *Bacteroides nodosus* infects feet already inflamed due to *Fusobacterium necrophorum* infection. *Bacteroides nodosus* on its own cannot cause foot rot. Foot rot can be introduced into a previously clean flock by animals infected with *Bacteroides nodosus* and then spread throughout the flock via contamination of the pasture and general environment.

Although foot rot can occur any time, the chances of spread increase in warm weather with wet conditions underfoot. These conditions favor the survival of the organisms in the environment, cause softening of the skin of the feet and increase the chances of infections with the bacteria. Thus outbreaks of foot rot in the tropics tend to occur during the rains and are worst when large numbers of animals are congregated together.

Clinical signs: Foot rot causes lameness of varying degrees depending on the severity of the lesions and the number of feet affected. In the worst cases, especially if more than one foot is affected, sheep are reluctant to walk, tending to lie or crouch, and graze in a kneeling position. The first sign of the disease is moist reddening of the skin between the digits, often called "scald". This is followed by separation and under running of the hoof at the bulb of the heel; in severe cases the separation and under-running may involve the entire hoof. Healing eventually takes place in the form of growth of new hoof-horn, but in severe cases there may be permanent distortion of the foot.

Treatment: Cut away under-run horn to expose the diseased tissues to the air, which improves healing. Because Foot rot is an infectious disease, cutting knives and shears should be disinfected between each sheep (10% zinc sulphate). Once trimmed, affected feet should be treated by standing infected animal in a footbath containing 5% formalin or 5% copper sulphate. Instead of foot bathing, infected feet may be sprayed with an antibiotic aerosol. The easiest, but most expensive method of treatment is injection with appropriate antibiotics (streptopenicillin, oxytetracycline).

Control: If a flock is free of the disease, steps should be taken to ensure infection is not introduced (quarantine). In endemic areas every animal should be examined and infected ones segregated. Diseased animals must be treated until they are cured before joining the rest of the flock. Those that don't respond should be culled.

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3.2. Major Viral Diseases

3.2.1. Foot and Mouth Disease
Foot and Mouth Disease (FMD) is an acute viral infection of cattle, sheep, pigs, goats’ buffalo, and many species of cloven-hoofed wildlife. It is one of the most contagious of animal diseases characterized by the appearance of vesicles in the mouth, on the feet, udder, teat and pillars of rumen. The disease is not highly fatal, but the lack of production resulting from the disease and the prohibition on the free movement of livestock, livestock products and livestock by-products makes it one of the most serious animal diseases in the world. FMD is occasionally seen in man. It gains admittance through a local lesion where it produces a vesicle.

**Cause:** FMD viruses belong to the genus *Aphthovirus* in the family *Picornaviridae*. A number of FMD virus types are responsible to bring the disease. They are known as O, A, C, SAT1, SAT2, SAT3 (South African territory) and Asia-1 types. Subtypes exist in each type. The types and even subtypes are immunologically distinct. In Ethiopia, type O, A, SAT 1, SAT 2 and C have been identified of which type C becomes disappear.

**Transmission:** The respiratory tract is the usual route of infection for FMD virus, and the viraemia which follows results in distribution of the virus to many tissues of the body. Further replication occurs in many of these tissues, giving rise to characteristic lesions of FMD. Significant excretion of the virus in infected animals may occur for 4 or more days before development of clinical signs; milk and semen are of particular importance in this regard. Saliva contains highest quantities of virus in acutely infected animals. High levels of the virus may be found without lesions in many tissues; however the pregnant uterus appears not to be involved. Abortion is rare, and transplacental infection is not reported. Transmission is by contact between animals, through the air in cool, humid conditions, ingestion of contaminated feed and through contaminated clothing, feed bags etc.

**Pathogenesis:** Entry of virus (infection) → bloodstream → dissemination to epidermal sites (lesions develop on epithelium of mouth, feet and teats after incubation period of 3 – 8 days in most species). At the end of viremia, the animal recovers, but the virus may persist in the pharyngeal area of convalescent ruminants for months and occasionally years. Bacterial complication aggravates the lesion leading to lameness and mastitis. The virus causes necrotizing myocarditis.

**Symptoms:** Rise in temperature, development of vesicles in the mouth (leads to difficulty in chewing, profuse salivation) on the feet (leads to lameness). Loss of appetite, sudden drop in milk yield, abortion and loss of condition, low mortality on adults and high death rate in young. Generally FMD is less severe in endemic zebu cattle than in exotic stock. Secondary bacterial infection prolongs the recovery.

Recovered cattle are generally immune to re-infection with homologous virus for 1-3 years. Maternally derived antibody disappears before 6 months of age in calves and piglets.

Treatment and control: There is no specific treatment. Antibiotics can be used to combat secondary bacterial infection.

**Vaccination:** The first step is to know the type of virus involved in the outbreak, then prepare the vaccine out of the type responsible for the outbreak and finally give the vaccine to the outbreak area. Vaccination results in protection against experimental challenge for 3-6 months. The duration of immunity depends on the vaccine and type of adjuvant. However, although fully immune animals may be protected against clinical disease, they may still be infected and become carriers. The carcass must be buried or burned.

Movement of infected animals, farm personals, and community people should be controlled. Farm machineries, vehicles, personnel should pass over a material soaked with 10% caustic soda in getting in and out of the infected area i.e., disinfections of animate and inanimate objects.

3.2.2. Rabies
Rabies is an acute severe viral infection of the central nervous system (CNS). It is caused by a virus, which is present in the saliva of infected animals and is commonly transmitted by bites. All warm blooded animals are susceptible but it is predominantly a disease affecting carnivores and its danger to man is mainly through the bite of rabid dog. Rabies is endemic to Ethiopia.

Although the domestic dog is the chief transmitter of rabies to man, domestic cats, wild and feral carnivores (including jackals, foxes and wolves) and bats can be infected and be direct danger to man.

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In dogs the usual incubation period is 21 to 60 days, but it may, at times, be much longer. There is a direct relationship between the severity and location of the bite and the length of the incubation period. Thus, if the head or face is severely bitten symptoms may appear in as few as ten days, whereas under some circumstances illness may not develop for a year or more.

Chronic rabies, clinically inapparent infections, and recovery from clinical disease with persistent shedding are extremely rare. Strains of rabies virus have been identified in Ethiopia that has been associated with nonfatal clinical disease or symptomless infection, both with excretion of virus. In countries where rabies is endemic, serologic surveys of dogs for antibodies indicative of a non-fatal response to infection have been documented, as have clinical recoveries from laboratory-induced rabies, but unless further evidence comes to light it can be assumed that the carrier state does not exist.

Rabies in human beings

Rabies in man is suspected if, weeks or months after exposure the individual experiences head ache, fever, nausea, sore throat or loss of appetite. Other early symptoms include unusual sensitivity to sound, light and change of temperature, muscle stiffness, dilation of the pupil and increased salivation. Later irrational excitement alternating with periods of alert calm develops. Most dramatic of all are the severe and extremely painful throat spasms suffered by the victim on attempting to swallow or even upon viewing liquids. This leads to Hydrophobia, or fear of water, which is characteristic of rabies victims and gives the disease its common name. No specific treatment for rabies is available once symptoms appear.

Transmission: The virus may be recovered from the Central Nervous System (CNS) and salivary glands. In nature, it is transmitted from animal to animal by means of a bite that introduces the virus bearing saliva. Transmission through ingestion and inhalation is possible rarely. Bats are symptom less carriers.

The virus can be found in the salivary glands for 5 days before clinical signs appear in the infected animal, a factor that is of vital importance for control.

Clinical signs: There are two syndromes.

1. Furious form. Furious rabies represents the classical "mad-dog syndrome" in which the animal becomes irrational and viciously aggressive. The facial expression is one of alertness and anxiety, with pupil dilated, noises invites attack. Such animals lose all caution and fear of natural enemies. Dogs of this form of rabies frequently roam streets and highways, biting other animals, people and any moving objects. They commonly swallow foreign objects, faeces, straw, sticks and stones. Rabid dog will chew the wire and frame of their cages, breaking their teeth, and will follow a hand moved in front of the cage, attempting to bite. Rabies in cattle follows the same pattern. Lactation ceases abruptly, the animal become alert, and the eyes and ears follow sound and movement. The most typical clinical sign in cattle is bellowing of a character that can hardly be mistaken once encountered.

2. Paralytic form. This is characterized by early paralysis of the throat and masseter muscles, usually with profuse salivation and inability to swallow. The paralysis progresses rapidly to all parts of the body, and coma and death follow in a few hours.

Procedures after bite

As a first aid measure, bite wounds should always be cleaned immediately and thoroughly with soap and water to remove the saliva from the area. The wound then may be squeezed to promote bleeding, since this will also help to clean it. Antiseptics (like 70% alcohol) should be used alone or after cleaning.

If at all possible, any dog or cat inflicting a bite should be captured alive and kept under surveillance. This may make it possible for the bitten individual to avoid undergoing rabies vaccination unnecessarily.

Diagnosis: Rabies is a major zoonosis for which diagnostic techniques have been standardized internationally. For the laboratory diagnosis of rabies central nervous tissues i.e., hippocampus, cerebellum and medulla oblongata are the tissues of choice.

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**Control:** Vaccination is a very important control measure. A range of highly effective, safe, inactivated veterinary vaccines is available, producing a protective immunity which lasts from one to three years depending on the antigen content of the vaccine. Some of the vaccines may be used in all domestic carnivores and herbivores, while others may be designed for use in specific species.

- Elimination of stray dogs and cats.
- Unvaccinated dogs, cats and other pets bitten by a known rabid animal should be killed immediately. If the owner is unwilling to do this, the animal should be placed in a strict isolation for a minimum of 4 months. If the exposed animal is dog and has been vaccinated within 3 years with vaccine providing 3-year immunity, it should receive a booster within 5 days of the exposure.

Comments: Human vaccinations were administered in Ethiopia, all because of dog bite. Furthermore of the 488 deaths reported by the same author over 97% had been bitten by rabid dogs. The National Research Institute for health has also recorded 9812 human vaccinations during 1983 - 1989 of which over 90% were bitten by dogs and 194 fatal human cases with over 97% caused by dog bite. The fact that certain Ethiopian rabies virus isolates continue to be shed after dogs have recovered from illness is an area of public concern.

### 3.2.3. Newcastle disease

NCD is an infectious, highly contagious and destructive disease of domestic poultry and other birds caused by *Paramyxovirus*. Three strains exist: the velogenic strains, which are highly pathogenic and easily transmitted; the mesogenic strains, which show intermediate pathogenicity; and the lentogenic strains, which show low pathogenicity in chickens. The disease was reported in Ethiopia in 1974 from exotic chicken. Since then, NCD has become cosmopolitan (DACA, 2006).

Although Newcastle disease virus can produce a transitory conjunctivitis in human beings, the condition has been limited primarily to laboratory workers and vaccination teams exposed to large quantities of virus and, before vaccination were widely practiced, to crews eviscerating poultry in processing plants. The disease has not been reported in individuals who rear poultry or consume poultry products (Frazer et al., 1986).

Transmission: The virus is present in exhaled air, in respiratory discharges, in faeces, in eggs laid during clinical disease and in all part of the carcass during acute infection and at death. Chickens are readily infected by aerosol, and by ingesting water or feed contaminated by the virus. While the primary source of virus is the chicken, other domestic birds and certain wild birds are susceptible and may spread the virus.

**Clinical signs:** Respiratory, nervous and digestive signs are seen. Respiratory signs are gasping and coughing. Nervous signs include drooping wings, dragging legs, twisting of head and neck (torticollis), circling, walking backwards (particularly after drinking water), depression, loss of appetite and complete paralysis. Laying flocks may have partial or complete cessation of production and fail to recover. Watery and greenish diarrhea and swelling of the tissues around the eyes and in the neck region are common. Morbidity may be up to 100%. The mortality rate may be anything from 5-100%, being highest in young chickens.

![Figure 7: Nervous form of Newcastle disease](image)

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Diagnosis: Tentative diagnosis of a rapidly spreading, respiratory-nervous disease may be confirmed by isolation of the hemagglutinating virus identified by inhibition with Newcastle disease antiserum. A rise in hemagglutination-inhibition antibodies in paired serum samples is confirmatory.

Control: The best control is a combination of sanitation and production of immunity by vaccination, but this must be carried out on an area wide basis. In areas where NCD is reported non-existent, definite precautions should be taken to prevent its introduction. Live virus vaccines are widely used for vaccination. This vaccine is administered through drinking water, in spray or dust, nose or eye drops. Healthy chicks are vaccinated as early as the fourth or even the first day of life. However, delay until two or three weeks avoids partial blockage of the active immune response by maternal antibody. Other than vaccination, standard sanitation practice is essential with strict isolation and disinfection procedures.

Comments: The disease was imported to Ethiopia in day old chicken brought from abroad. The first cases were seen in 1970 on a poultry farm near Asmara, Eritrea, from where it spread all over the Ethiopia. Among humans, particularly poultry workers and those handling the virus in the laboratory are affected. The clinical signs in humans usually consist of conjunctivitis.

3.2.4. Rinderpest (cattle plague)
Rinderpest is a highly contagious and most serious epidemic disease of cattle characterized by fever, ulceration in the mouth, gastroenteritis with diarrhea and high mortality. It is primarily a disease of cattle and buffaloes, and high mortalities are seen in these animals. Infections may also occur in sheep, goats, pigs, and many wild cloven-hoofed animals, without always producing clinical disease.

Rinderpest, a viral disease of mainly cattle and buffaloes, has been associated with significant numbers of deaths of cattle which have affected the rural economy and contributed to the general poverty, mass starvation and subsequent famine in Ethiopia. The first epidemics of the disease entered the coastal region of Ethiopia, now Eritrea, in 1887 during the major outbreaks that affected most of Africa. The disease swept across the northern provinces of Tigray and Shewa southwards, resulting in an estimated 90% mortality of the country's cattle population and wildlife, including buffaloes and antelopes.

In the 1960s, the International Rinderpest Joint Project (JP-15) campaign came close to eradicating rinderpest from Africa. However, endemic pockets remained in the Ethiopian lowlands and the disease occasionally spread into adjacent highland sedentary cattle populations where it caused minor epidemics.

In 1979, the disease reappeared in many parts of the country because of disruption of the annual vaccination campaign due to famine and civil unrest, which caused large scale migration of people and animals searching for water and food. In the 1980s a wave of outbreaks with serious animal loss was experienced as a result of the continued movement of animals from lowlands to adjacent highlands during the drought. Animal markets also played a significant role in dissemination of the disease, as herdsmen and other traders sold sick animals for cheaper prices. In 1989, the Pan African Rinderpest Campaign (PARC) program started in Ethiopia and undertook mass vaccination of animals for almost one and half decade (the last vaccination campaigns being in Afar region) and eventually able to eradicate the disease.

The long persistence of rinderpest in Afar is primarily a result of the lack of accessibility of the herds for conventional vaccination, which could be explained by the difficult terrain, insecurity and inadequate animal health service delivery. Ethiopia is declared free of infection since 2005.

Cause: The disease is caused by a *Morbillivirus* in the family *Paramyxoviridae* (Murphy et al., 1995) of which there are many strains, but which are immunologically the same, a factor of considerable importance in control. The virus is fragile and rapidly inactivated (destroyed) by environmental factors such as heat and light, but remain viable for long periods in chilled or frozen tissues. There is no carrier state and the virus must maintain itself by continuous transmission among susceptible animals (domestic and wild ruminants and pigs).
**Transmission**: Transmission of rinderpest requires close contact between infected and susceptible animals; animals, which are infected but often before clinical signs appear, introduce the disease. The virus is present in all the discharges of the sick animals. Healthy cattle contract the infection through eating and drinking contaminated materials and occasionally by inhalation.

The underlying requirement for the maintenance of the transmission cycle of rinderpest is a sufficiently large population of animals to provide a regular supply of susceptible hosts, and sufficient animal movement to allow animal mixing. Therefore, in Africa and Asia outbreaks of rinderpest are now rarely seen except in areas of civil unrest. However, there is also a pocket of infection in Southern India affecting buffalo, cattle, goats, sheep, pigs and wildlife, and the disease is endemic in the Landhi cattle colony in Karachi, Pakistan.

**Pathogenesis**: Inhaled virus → penetrate upper respiratory epithelium → multiply in tonsils and regional lymph nodes → blood stream → affinity for lymphoid organs and alimentary mucosal → destruction of lymphocytes (leukopenia) → death (dehydration and immunosuppression).

**Clinical signs**: Rinderpest virus has an affinity for lymphoid tissue. In blood the virus is found in mononuclear leukocytes which transport the virus to epithelial tissues, especially those of the alimentary tract. This accounts for the characteristic clinical signs of disease, which include necrotic stomatitis and gastroenteritis. Pregnant animals frequently abort, sometimes weeks or months after the clinical stage of the disease, but transplacental infection of the foetus does not occur.

Clinical signs vary from peracute, with collapse and death within a few days, to inapparent subclinical infection. Incubation period of 6-15 days is followed by high fever. In a few days the appetite is reduce. Nose and eye discharges are at first thin and watery but later on becomes purulent. The breath is offensive and respiration shallow and fast. Later breathing is labored and painful with grunting expiration. 3-5 days after the onset of fever mouth lesions appear as pinpoint ulcers, which rapidly enlarge and join together to form large areas of erosion. Opening the mouth releases a foul smell. The common sites for the lesion are the inside of the lower lip and gum, the check papillae, the edge of the upper lip and dental pad, the ridges of the hard palate, the ventral tip of tongue and inside the vulva. Diarrhea begins 1-2 days after the appearance of mouth lesions, at this time the temperature falls to normal or subnormal. The diarrhea is fetid in odor, may contain blood, mucus and pieces of tissues. The animal become dehydrated and dies.

Recovery from the disease results in lifelong immunity. Surviving animals generally shed the virus only for about 3 weeks post infection. It is generally accepted that recovered cattle are free from infection and that there is no carrier state. The situation in other cloven-hoofed animals is less clear, as small ruminants and pigs have been suggested as reservoir. This has shown to be the case on the Indian subcontinent, where there are strains of rinderpest virus which cause subclinical infections in sheep and goats, from which the disease can be passed to cattle.

Where the disease is endemic often the only susceptible animals are young weaned stock. The adults are immune through recovery from earlier natural infection or vaccination and suckling young are protected by their immune mothers.

The appearance of the lesions of rinderpest is almost identical to that occurring in the Bovine virus diarrhea mucosal disease complex of cattle. This is a sporadic disease complex occurring most commonly in animals up to 18 months old. The lesions of malignant catarrhal fever are also very similar.

A related virus causes a disease of sheep and goats called "peste des petits ruminantis" (PPR) in other parts of Africa and Middle East. The disease is similar to that seen in cattle being characterized by fever, erosions in the mouth, diarrhea and often pneumonia followed by death.

**Treatment and control**: There is no specific treatment however, in valuable animals' injection of broad-spectrum antibiotics and good nursing is useful to fight secondary invaders and facilitate recovery.

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Vaccination: Many rinderpest vaccines have been produced. A live attenuated cell culture vaccine is available which confers an immunity that lasts at least 5 years after a single inoculation. Revaccination produces a lifelong immunity. Immediate vaccination of all in contact cattle and neighboring herds aged 6 months and above and annual vaccination of growing stocks.

Ethiopia should try to maintain disease free status by importation of animals from rinderpest free countries only; ring vaccination if reintroduced and proper disposal and hygiene of infected animals is crucial.

Comments: Previously of the infectious diseases found in Ethiopia, rinderpest and CBPP constitute the two most serious disease problems; for they create a heavy economic loss in some regions in certain years. It has been because of the widespread nature of these diseases, their high incidence and the serious economic loss caused by their presence in the country that special attention was given to their control on a countrywide basis. Thanks to the Pan African Rinderpest Campaign (PARC) conducting mass vaccination against rinderpest and CBPP starting from the early 1980's E.C. (the vaccine for the two diseases is given mixed) throughout Ethiopia rinderpest is on the verge of eradication and CBPP is reasonably controlled.

3.2.5. Lumpy skin disease (LSD)

LSD (synonym - Bovine Nodular Dermatitis) is an acute, subacute or inapparent infectious viral disease of cattle caused by a virus which belongs to the genus *Capripoxvirus* in the family. LSD is characterized by a formation of nodules on the skin and visible mucous membranes, generalized lymphadenitis, with a high body temperature, lameness and edema of the facial, subcutaneous tissues and legs The severity of clinical signs depends on the strain of the virus, but these may include fever, skin nodules, necrotic plaques in the mucous membranes, and swelling of peripheral lymph nodes. The disease was first diagnosed in Zambia in 1929. Since then it has occurred in many African countries and in Madagascar, with considerable variation in mortality rate. In outbreaks over the past 20 years the mortality rate has been less than 5%. In 1989 the disease occurred for the first time outside Africa, in southern Israel. The disease is widely distributed in Ethiopia imposing severe economic loss due to damage of hides (DACA, 2006). According the authors information the virus strain causing outbreak of LSD in Ambo and its surrounding during September and October 2008 appears to be more virulent and has caused serious economic losses through morbidity and mortality, particularly in exotic and cross bred dairy animals..

A virus known as neethling virus, which is a poxvirus, related to the virus causing sheep pox disease, causes true LSD and cattle can be protected against true LSD by prior inoculation with virulent sheep pox virus. Transmission: LSD is not particularly contagious, and direct transmission by contact between animals is inefficient. Biting flies have been incriminated in most epidemics, which have been well-defined and have occurred at regular intervals. Outbreaks are more common during the wet summer and autumn months, particularly in low-lying areas and along water courses. The virus has been recovered from *Bionyiafasciata* and *Stomoxys calcitrans* caught while feeding on infected cattle. Because the disease can be transmitted by means of infected saliva, contact infection must be accepted as method of spread.

**Clinical signs:** Incubation period of 4-14 days is followed by rise in body temperature, anorexia, increased salivation, lacrimation and clear nasal discharge, which later becomes mucopurulent. This is followed by the sudden appearance of the characteristics nodules in almost all parts of the skin of affected animals. A primary nodule appears at the site of inoculation about 7 days later, and it seems that the virus multiplies in the dermis at the site of insect bites.
Hairs over the nodules stand erect, thereby clearly delineating the affected area. Often the skin dies and falls off in patches leaving open wounds, which on healing leaves scars. Nodules may also develop on the oral and respiratory mucous membranes. Superficial lymph nodes are enlarged and easily palpable. Although the mortality rate is low (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.

The incidence of secondary infection in association of LSD probably accounts for more loss than from the actual virus. During the course of disease the virus is present in saliva for 11 days, semen for 22 days, and skin and nodules for 33 days, but not in urine or faeces. Although the saliva contains large amounts of virus, infection by direct contact or by fomites is not of importance, and the period of infectivity will largely depend on the accessibility of the virus to biting flies. It appears that the animal will be most infective during the short viraemic period 2-3 days before and after the appearance of lesions. Since the virus is present in skin nodules for 5 weeks, infected cattle are probably a source of infection during this period.

Based on South African field experience it is generally accepted that recovered cattle are not virus carriers (Woods, 1990).

**Diagnosis:** Complete diagnosis is usually made in the laboratory by viral isolation and identification or detection of specific antibodies. However, the interpretation of a positive serological test in a clinically normal animal would depend on the test used. A positive reaction could result from a cross reaction with other pox viruses, such as the parapoxvirus which causes contagious ecthyma (scabby mouth), or it could indicate an animal that was immune through either vaccination or recovery from infection with capripox virus.

**Treatment and control:** There is no specific treatment. Antibiotics can be used to control secondary infections in valuable animals. Animals which have recovered from disease develop neutralising antibodies which persist for at least 5 years (Woods, 1990). The immunity to reinfection is predominantly cell mediated. Cattle vaccinated with attenuated cattle capripox strains or with strains from sheep and goats develop neutralising antibody in 10 days and this persists for at least 3 years. Calves of immune cows acquire maternal antibody via colostrum and are able to resist serious clinical disease for at least 6 months. Immunity in recovered or vaccinated animals is lifelong. The prophylactic use of live sheep pox vaccine is obviously limited to countries where sheep pox disease already occurs. Comments: LSD has not been regarded as a disease of great importance in Ethiopia, but recent evidences suggests that it is probably more prevalent than was previously believed. From personal observation most cases of LSD are seen from September to December in western Shoa region including Ambo college of Agriculture’s livestock farms.

### 3.2.6. Sheep pox and Goat pox

Sheep pox and goat pox are acute or subacute contagious and often fatal diseases of sheep and goats, caused by a member of the genus *Capripoxvirus* in the family *Poxviridae*. Both forms are widespread in Ethiopia. Most strains of *Fundamentals of animal disease by Dr. Fayera Gemeda*
the virus are specific to the host species in which they are isolated, but in some countries strains exist that can infect both sheep and goats (Munz and Dumbell, 1994). The clinical signs vary considerably with the strain of the virus and the species and breed of host (Kitching and Carn, 1996). The morbidity rate in sheep may be as high as 70%; mortality varies from 5 to 50% in adult animals and it may be even higher in lambs. Both morbidity and mortality rates are generally lower in goats. Abortion is rare, except in severe cases which are usually fatal. Mild and inapparent infections can also occur.

Pox viruses are epitheliotropic, and the effects of disease are therefore seen especially in the skin and in the lungs. Infected animals shed virus in all excretions and secretion).

Sheep pox is a serious, often fatal disease of native sheep and goats characterized by widespread skin eruptions. Young sheep are more susceptible than older ones.

Transmission: Transmission may be through inhalation of virus in contaminated water droplets, dust or dry skin scabs or through wounds or scratches on the skin. Infection by contact with lesions or infected milk is of minor importance. Mechanical transmission is possible by the stable fly Stomoxys calcitrans, which is widespread in New Zealand the virus may survive on flies for up to 4 days.

The disease is regarded as being endemic in most African countries north of the equator, as well as in the Middle East, Turkey, Iran, Afghanistan and the Indian subcontinent. In these countries transmission is facilitated by sheep and goats being herded into crowded enclosures at night, and environmental contamination leads to introduction of the virus into small skin lesions. During outbreaks the virus is probably transmitted between animals by aerosols.

Occurrence: The disease occurs throughout the year, but severe outbreaks usually occur during the winter or during wet and cold weather and in animals weakened by parasites or other infections (Munz and Dumbell, 1994).

Clinical signs: Infection is introduced into a healthy flock by a sick sheep. The incubation period is up to 8 days, and then a typical pox eruption develops. Fever develops; eyelids become swollen and mucopurulent discharge crusts the nostrils. Widespread skin lesions develop that are most readily seen on the muzzle, ears and areas free from wool or long hair.

Nodules usually scab and persist for several weeks, healing to form a permanent, depressed scar. Lesions within the mouth ulcerate and constitute an important source of virus for infection of other animals. Recovery and healing of skin lesions may take 5-6 weeks. High concentrations of virus occur in lesion material. The quantity and duration of virus excretion, especially in the conjunctival and nasal discharges, seems to depend on the capacity of the particular virus strain to produce well-developed pox lesions. The rate of transmission is therefore probably related to the severity of lesions which develop in clinical cases. Peracute cases usually die before significant amounts of virus are excreted. Recovered animals are immune to reinfection for years.

The duration of shedding of the sheep- and goatpox viruses by recovered animals has not been subjected to detailed studies. As with other pox viruses, infectivity is destroyed by exposure to direct sunlight, but it is retained in dark stables for long periods, particularly in scabs shed by infected animals.

Infectivity may also be present in the wool or hair of recovering animals. It is generally considered that skin scabs are the main source of shed virus and that infectivity may survive in scab material for at least 3 months.

Control and prophylaxis: Effective cell culture-derived vaccines containing attenuated capripoxviruses provide immunity which lasts over a year, and will probably provide lifelong protection against lethal challenge. Prevent secondary bacterial infection by application of antibiotic ointment. Give vaccination every two years.
3.2.7. Fowl pox
Fowl pox is virus disease of poultry characterized by pox lesions on the head. In Ethiopia fowl pox is common in commercial and backyard chicken. It takes two forms both of which can occur in the same bird. In the form most easily recognized, wart like pox is present on the comb, wattles, eyelids and corners of the beak. In severe cases the legs and feet and the skin of the body may also be affected. In the other form, a cheesy membrane forms in the mouth, throat and there may be pus in the facial sinuses, conjunctival sac and palate cleft. In severe outbreak affected birds' loss appetite, are depressed, loss weight and die.

There is no specific treatment for the disease; however, it can be controlled by vaccination, isolation and disinfection.

3.2.8. Orf/ Contagious ecthyma (orf) of sheep & goats, Bovine Papular Stomatitis
Orf is an exanthemous disease caused by a parapox virus and occurring primarily in sheep and goats. It is also known as contagious pustular dermatitis, infectious labial dermatitis, ecthyma contagiosum, and sheep pox, thistle disease and scabby mouth.

Orf has been recorded since the late 1800s and has been reported from most sheep- or goat-raising areas including those in Europe, the Middle East, the United States, Africa, Asia, Alaska, South America, Canada, New Zealand and Australia. It is spread by fomites and direct contact. In some environments infection is injected by scratches from thistles of both growing and felled plants.

**Etiology:** genus, parapoxviruses and family poxviridae

**Epidemiology:** The disease occurs in sheep and goats and causes unthrifitness, varying degree of pain and some economic loss.

It occurs most commonly in lambs (3-6 months of age).

- Morbidity and case fatality: outbreaks may occur in sheep and goats, with morbidity approaching 100 % and case fatality rates from 5-15 %.
- Death is due to the extension of lesions in the respiratory tract.
- Recovered animals are immune for 2-3 years

**Transmission:** Spread in a flock is very rapid and occurs by contact with other affected animals or inanimate object, such as ear-tagging pliers and emasculators.

**Pathogenesis:** Damage to skin is essential for the establishment of infection and the development of typical lesions.

→Virulinal challenge of mildly abraded skin →scarification of skin → infection spread laterally and uniformly from the new epidermis

**Clinical findings:** Symptoms include papules and pustules on the lips and muzzle, and less commonly in the mouth of young lambs and on the eyelids, feet, and teats of ewes. The lesions progress to thick crusts which may bleed. Orf in the mouths of lambs may prevent suckling and cause weight loss, and can infect the udder of the mother ewe, thus potentially leading to mastitis. Sheep are prone to re-infection. Occasionally the infection can be extensive and persistent if the animal does not produce an immune response.

Sheep

- Lesions develop initially as papules and then pustules (stage which are not usually observed)
- Then a thick tenacious scabs covering a raised area of ulceration, granulation and inflammation
- The first lesions develop at the oral mucocutaneous junction, usually at oral commissures
- From here the lesions spread on to the muzzle and nostril, the surrounding haired skin
- The affected lambs suffer a severe setback because of restricted suckling and grazing
- There is severe systemic reaction
- Extension to GIT—gastroenteritis
- Extension to trachea—bronchopneumonia
- Lesion in the mouth may occur on tongue, gums, dental pad etc

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Goats

- Multifocal lesions over the head, neck, thorax and flanks
- The lesions began as plaques, followed by epidermal proliferation and severe encrustation
- The skin crusts gradually dried and fell off, leaving area of alopecia and depigmented skin
- Recovery occurs within 3-6 weeks without treatment.

Samples for confirmation of diagnosis
- Virology—vesicle fluid, scraping from lesion

Treatment: there is no specific treatment. More typically sheep will cure themselves of orf within a week or so as the disease runs its course. Sheep custodians can assist by applying antibiotic sprays on to large scabs, ensuring infected lambs receive sufficient milk and separating out the infected stock to slow down cross-transmission to healthy animals. It is advisable for those handling infected animals to wear disposable gloves to prevent cross-infection and self-infection.

Control:
- Isolation of affected animals and vaccination of the remainder
- Lambs should be vaccinated at 6-8 weeks of age.

3.2.9. Marek’s disease (MD)
Marek's disease (MD) is a highly contagious viral neoplastic disease in chickens. MD is a lymphomatous and neuropathic disease of domestic fowl caused by a herpesvirus and characterized by infiltration of the peripheral nerve, brain, and other organs by lymphoid cells. Marek's disease is one of the most ubiquitous avian infections found worldwide.

Transmission: Marek's disease is highly contagious and is readily transmitted among chickens. Young chicken are mainly affected. The virus matures into a fully infectious enveloped form in the epithelium of the feather follicle, from which it is released into the environment. Infection usually occurs via aerosol exposure through the respiratory tract. Once infected, chickens continue to be carriers for long periods and act as a source of infectious virus.

Clinical signs: Infected birds show weight loss, or may exhibit some form of paralysis. Mortality varies from 5 to 50% in unvaccinated birds. The classical form (paralysis) with leg nerve involvement causes a bird to lie on its side with one leg stretched forward and the other backward.
Lesion: Enlarged nerves are one of the most consistent gross lesions in affected birds and, when present, have diagnostic significance. Diffuse or nodular lymphoid tumors may be seen in various organs, particularly liver, spleen, heart, lung, kidney, muscle, proventriculus and gonads.

Diagnosis: Diagnosis is made on clinical signs and gross or microscopic lesions. Chickens may become persistently infected with MD virus without developing clinical disease. Infection by MD virus is detected by virus isolation and demonstration of viral antigen or antibodies.

Control: Vaccination is the principal method of control. It has been demonstrated that MD vaccine only prevents the appearance of Marek’s disease tumors and paralysis; it does not prevent the birds from becoming infected with MD-virus. It is therefore of major importance to maintain high hygienic and sanitary measures by good management to avoid early exposure of young chickens.

Comments: Lobago and Woldemeskel in their study conducted on an outbreak of MD in commercial poultry farm containing 8500 chickens in central Ethiopia reported 46 % mortality rate during the first 14 weeks of outbreak in chicken 12 – 22 weeks old. They also indicated that youngs are highly susceptible to acute disease and MD causes considerable economic loss and is a major threat to poultry production in Ethiopia.

3.3. Rickettsial and Protozoan Diseases

3.3.1. Heart water or Cowdriosis

Heartwater (cowdriosis) is a rickettsial disease of domestic and wild ruminants caused by *Ehrlichia ruminantium* (formerly *Cowdria ruminantium*) and transmitted by *Amblyomma* ticks. *Ehrlichia ruminantium* is classified in the order Rickettsiales and in the family Anaplasmataceae, together with the genera *Anaplasma*. Although ruminants remain the primary target of the pathogen, in South Africa a possible canine *E. ruminantium* infection has been reported, and, more recently, *E. ruminantium* has been strongly suspected in several cases of rapidly fatal encephalitis in humans. However, in all cases, evidence of *E. ruminantium* infection was based on molecular detection. Isolation and characterisation of the infectious agent is necessary before *E. ruminantium* can be considered an emerging pathogen in species other than ruminants and especially in humans.

Animals affected: cattle, sheep, goat and wild ruminants. Temperate climate breeds of cattle, sheep, and goats are much more susceptible than indigenous breeds have been constantly in contact with the micro-organism. Young animals are resistant. Recovered animals appear to be immune and apparently do not act as a reservoir of infection.

Etiology: The causative organism *Cowdriaruminantium* is a minute intracellular parasite transmitted under natural condition by ticks belonging to the genus *Amblyomma*.

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**Pathogenesis:** Tick inoculation—local lymph node—blood—endothelial cells—vascular permeability—clinical signs (edema in lungs, body cavities and brain) and lesions.

**Clinical signs:** The average natural incubation period is 2–3 weeks, but can vary from 10 days to 1 month. In most cases, heartwater is an acute febrile disease, with a sudden rise in body temperature, which may exceed 41°C within 1–2 days after the onset of fever. It remains high for 4–5 weeks with small fluctuations and drops shortly before death. Fever is followed by inappetence, sometimes listlessness, diarrhoea, particularly in cattle, and dyspnæa indicative of lung oedema. Nervous signs develop gradually. The animal is restless, walks in circles, makes sucking movements and stands rigidly with tremors of the superficial muscles. Cattle may push their heads against a wall or present aggressive or anxious behaviour. Finally, the animal falls to the ground, pedalling and exhibiting opisthotonos, nystagmus and chewing movements. The animal usually dies during or following such an attack. Subacute heartwater with less pronounced signs, and peracute heartwater with sudden death, can also occur, according to the breed of ruminant and the strain of *E. ruminantium* involved. The most common macroscopic lesions are hydropericardium, hydrothorax, pulmonary oedema, intestinal congestion, oedema of the mediastinal and bronchial lymph nodes, petechiae on the epicardium and endocardium, congestion of the brain, and moderate splenomegaly.

**Diagnosis:** A tentative diagnosis of heartwater is based on the presence of *Amblyomma* vectors, nervous signs, and presence of transudates in the pericardium and thorax on post-mortem examination. When making a diagnosis based on clinical signs, the following other diseases should be considered: bovine cerebral babesiosis and theileriosis, anaplasmosis, botulism, haemonchosis in small ruminants, rabies and poisoning.

**Treatment and control**

- Control is by specific therapy, vaccination, tick control and prophylaxis.
- Tetracyclines at 10 mg / kg body weight will usually effectively cure if administered early.
- Reduction of ticks’ numbers may interfere with the maintenance of an adequate immunity through regular challenge and may result in heavy losses.

### 3.3.2. Anaplasmosis

Anaplasmosis is a disease of cattle, sheep and goats caused by infection with *Anaplasma* spp.

**Etiology:** *Anaplasma marginale* in cattle and wild ruminants and *A. ovis* in sheep and goat. *A. centrale* causes mild anaplasmosis in cattle.

**Epidemiology:** common in tropical and sub-tropical regions. Carrier animals are the source of infection. Many ticks have been found to be vectors, the most important in Ethiopia being *Boophilus decoloratus*, *Rhipicephalus simussimus*, *Rhipicephalus bursa*. Mechanical transmission can also take place via biting flies, but for transmission to be successful in this case the fly must move very quickly between two animals. Similarly the organism can be carried on surgical instruments (knife, needle, etc) between animals.

**Pathogenesis:** *Anaplasma* are the Rickettsial organism causing the disease (*Anaplasma* spp) obligate intracellular organism resides in or on the erythrocytes or free in the plasma. They infect and reproduce inside mature red blood cells. Parasitized erythrocytes are removed by phagocytosis and there will be a release of acute inflammatory reactants and the consequent development of fever. Continued destruction of erythrocytes results in anemia.

**Clinical signs:** In cattle anemia and jaundice are the main characteristics, because the *Anaplasma* attacks and destroys the red cells. The onset of the symptoms occurs 3–4 weeks after first infection and is accompanied by fever. Acutely infected animals may die. During the acute stage animals become aggressive; abortion is common. There is anorexia, and later a reluctance or inability to move, with general weakness, debility and progressive emaciation. Recovered animals remain carrier.

Symptoms may be complicated or obscured by concurrent tick borne infections, principally Babesiosis. The disease is usually sub-clinical in sheep and goats. Exotic breeds of cattle are more susceptible than indigenous zebu cattle. Calves are more resistant than adults.

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Diagnosis: Confirmative diagnosis may be obtained by detection of the organism in the blood smear using Giemsa stain.

Treatment: Injection of tetracycline (chlortetracycline or oxytetracycline) or imidocarb.

Control: Vector control and vaccination

3.3.3. Trypanosomosis

Trypanosomosis are the term applied to the group of diseases of man and animals caused by trypanosome, belonging to the flagellate protozoa. The local name of the disease in animals is called “Gendi”. In Ethiopia trypanosomiasis is a disease of considerable economic importance. The direct economic losses from the disease are death of breeding, trade and working animals. Indirect losses are reduced breeding efficiency, abortions, and slow growth rate in growing stocks and poor weight gain in mature animals.

<table>
<thead>
<tr>
<th>Trypanosome spp.</th>
<th>Host</th>
<th>Name of disease</th>
<th>Transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trypanosoma vivax</td>
<td>Ruminant, camels, horses, Nagana</td>
<td>Nagana</td>
<td>Biting flies and tse tse fly (Glossina spp)</td>
</tr>
<tr>
<td>Trypanosoma congense</td>
<td>All domestic and wild Nagana game animals</td>
<td>Nagana</td>
<td>Glossina spp, biting flies</td>
</tr>
<tr>
<td>Trypanosoma suis</td>
<td>pig</td>
<td>-</td>
<td>Glossina spp</td>
</tr>
<tr>
<td>Trypanosoma Brucei</td>
<td>ruminant, carnivores, equine and pig</td>
<td>Nagana</td>
<td>Glossina spp</td>
</tr>
<tr>
<td>Trypanosoma gambiense</td>
<td>man</td>
<td>sleeping sickness</td>
<td>Glossina (tse tse fly)</td>
</tr>
<tr>
<td>Trypanosoma Rhodesiense</td>
<td>man</td>
<td>Sleeping sickness</td>
<td>Glossina (tse tse fly)</td>
</tr>
<tr>
<td>Trypanosoma evansi</td>
<td>camel, dog, horse</td>
<td>surra</td>
<td>tabanus, stomoxys flies</td>
</tr>
<tr>
<td>Trypanosoma equiperdeum</td>
<td>equine</td>
<td>Dourine</td>
<td>Coitus</td>
</tr>
<tr>
<td>Trypanosoma Cruzi</td>
<td>man, pig, dog, cat</td>
<td>Chaga</td>
<td>triatomid bugs (blood sucking)</td>
</tr>
</tbody>
</table>

Life cycle

- The main infection sources are latently infected wild and domestic animals.
- Trypanosome spp are transmitted cyclically by Tsetse flies and acyclically- alimentarily by other blood sucking flies (Tabanus spp., Stomoxys spp.)
- In the mammalian host:
  - Trypanosome get into the tissue and blood system of the mammal host by becoming injected along with the saliva of an infected tsetse fly as the fly takes its blood meal.

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In the Tsetse fly:
- When a clean Tsetse fly takes a blood meal from trypanosome infected animal, there is a chance that it may become infected itself.
- The trypanosome go through a cycles of development in the fly, lasting several days or weeks before the infection is mature and the fly becomes infective to new host.
- After that period the fly remains infective for the rest of its life.
- Wild animals, particularly antelopes are reservoir host of trypanosomiases and tsetse flies are responsible for transmission to domestic animals.

**Clinical signs:** Regardless of the species of trypanosome and the species of host, the principal clinical signs are intermittent fever, progressive anemia and loss of condition. The symptoms are very variable, and there is no specific syndrome characteristic of the disease. Except Trypanosomavivax infection in cattle, in which case the disease may be peracute, the course is usually chronic and the mortality is high if no treatment is given, but the nutritional status of animals and the amount of stress they may undergo (lack of water, being overdriven etc.) are important factors in determining the course.

The incubation period is between 1 and 3 weeks. There is harsh coat, listlessness, intermittent fever and anorexia. The pulse and respiration are rapid and shallow. The mucous membrane is pale. Abortion is common in females. In acute cases death occur in one week.

As the condition become chronic there is emaciation and extreme anemia. Lymph nodes are moderately enlarged. Generally speaking native breeds are more resistant

**Diagnosis:** The essential feature is to determine whether the animal is or has been in or near a known trypanosomiases area. In tropics and sub-tropics, any livestock showing anemia, fever and progressive loss of condition should be suspected of trypanosomosis. Confirmatory examination, for the flagellated protozoa in between red blood cells of Giemsa stained thick and thin blood smear taken from ear vein.

**Control**
- No vaccine is available but the disease can be prevented by careful use of prophylactic drugs. Animals can be given drugs (trypamedium) prophylactically in areas with a high population of trypanosome infected with Tsetse flies. The problem of drug resistance must be carefully monitored by frequent blood examinations for trypanosome in prophylactically treated animals (commonly every 2-3 months interval). Drugs like trypanidium, berenil etc are used for curative purpose.
- Control of the insect vector
  - Elimination of the fly itself by direct application of insecticides to the habitat of Tsetse fly and on the back of the animal (deltamethrin, cypermethrin). The principle in using these insecticides on the animal is simply that Tsetse coming to feed on cattle or other treated domestic livestock will either be killed by picking up a lethal deposit of insecticide whilst feeding, or will be repelled by the insecticide and will therefore not attempt to feed
  - Changing the habitat of the vector by bush clearing, to render the habitat unsuitable for the fly
  - Interfere with the reproduction of the vector by releasing sterile males. This method is used to eradicate the fly after the fly population has been reduced to high level by other methods.
  - Use of tsetse traps: In East Africa there are different types of traps used to catch the flies. Traps work more effectively when used in conjunction with odour attractants
- Use of trypanotolerant breeds Example N'dama breeds of Western African cattle belonging to *BosTaurus* cattle

Note:Tsetse fly control is very costly and requires a high degree of management, organization and specialist expertise.
3.3.4. Coccidiosis

Coccidiosis is a protozoal disease of all domestic mammals and poultry. It is a major disease in poultry and ruminants. The protozoan, *Eimeria* spp, attacks the epithelial cells of the intestine and the disease is characterized by diarrhea often bloody, emaciation and anemia.

**Etiology:** the pathogenic coccidial spp are as follows:
- Cattle: *Eimeria zuemii*, *E. bovis*, and *E. ellipsoidalis*,
- Sheep: *E. arloingi*, *E. crandallis*, *E. ahsata*
- Goats: *E. arloingi*, *E. faurei*, *E. caprovina*,
- Pig: *E. debliecki*, *E. scabra*
- Horses and donkey: *E. leudarti*

**Epidemiology**

- young calves, lambs, piglets, kids and rarely foals are more affected
- Occurs most commonly in crowded conditions both in barns and on pasture
- Transmitted by fecal-oral route; oocysts shed from infected animals
- Clinical disease occurs rarely in adult cattle
- Under extensive systems of management the clinical disease is seldom seen but it is of importance where animals are housed or confined in small areas.

**Life cycle**

The source of infection is the faeces of clinically affected or carrier animals. Infection is acquired by ingestion of contaminated feed and water containing oocysts or by licking the hair contaminated with infected feces. Moist conditions, temperature of 12-32 °C and oxygen are essential for sporulation of the oocyst in the external environment. Very high temperature and dry conditions are not conducive for sporulation. The coccidial life cycle is self limiting. Sporozoites released from the ingested oocyst and invade the epithelial cells of intestine to develop into asexual schizont.

After the schizont matures, the merozoites are released by rupture of the epithelial cells. New epithelial cells are again invaded and second generation schizogony occurs.

Release of another generation of merozoites which invade epithelial cells and produce the sexual stages, the macrogametocytes and microgametocytes.

As the second generation schizont mature, the cells containing them slough from the basement membrane and cause hemorrhage and destruction of intestine.

- The oocysts are the result of fertilization of the gametocyte and are discharged at the time of rapture of the cells which usually coincides with the onset of clinical signs of dysentery.
- The severity of the disease depends upon the number of intestinal cells invaded and the amount of tissue adversely affected.
- There can be no absorption through severely affected tissue.
- Retarded growth and loss of weight in affected animals are the result of decreased feed intake, reduced absorption of nutrients and loss of water, blood and tissues through diarrhea

**CLINICAL FINDINGS**

- The first sign of clinical coccidiosis is usually the sudden onset of severe foul smelling diarrhea containing mucus and blood.
- Severe straining/tenesmus is characteristic and rectal prolapsed
Anemia with pale mucosa, weakness, staggering, decreased appetite, loss of weight and dehydration are evident. Mild abdominal pain in lambs.

**Diagnosis:** The presence of large number of oocyst in faeces with characteristic blood stained diarrhea and sloughing of the superficial mucus membrane of the intestine may be taken as reasonable diagnostic.

**Treatment:** Coccidiosis is a self-limiting disease, and spontaneous recovery without specific treatment occurs commonly when the multiplication stage of the coccidia has passed. Sulphonamide particularly sulphadimidines are successful in treatment as well as prevention. Treatment in late stage of the disease is ineffective because damage to the intestine has already been done.

**Control**

- The most effective means of controlling coccidiosis is based on the normal hygiene requirement for proper livestock management.
- Young animals should not be kept for prolonged periods in humid and shaded enclosures, covered with manure.
- Likewise, concentrating animals in one place for long period should be avoided.
- Good hygiene and avoidance of overcrowding are very important.
- Animals should be kept away from wet areas and should be given dry, well drained standings.
- Concrete enclosures should be thoroughly cleaned and drained.
- Maintaining poultry at all times on wire floors to separate birds from droppings is used as prevention.

### 3.3.5. Babesiosis (tick fever, red water, piroplasmosis)

Babesiosis includes those diseases caused by *Babesia* spp. in cattle, sheep, pigs and horses. They are all characterized by fever and intravascular haemolysis causing a syndrome of anemia, haemoglobinuria and sometimes jaundice. In non-resistant animals they can be highly fatal.

**Etiology:** *Babesia bovis* and *Babesia bigemina* found exclusively in cattle and their distribution coincides with that of their major tick vectors, Boophilus spp., certain other ticks can act as vectors, and mechanical transmission by biting flies can occur. The protozoal parasites invade and multiply in the red blood cells.

**Clinical Findings**

- Temperature rises
- In milking cows there is a fall in yield
- Anorexia, weakness and a staring coat
- In peracute cases death may occur at this stage
- At about third day haemoglobinuria (dark brown urine)
- Increased heart rate and respiration
- Pallor
- In protracted cases jaundice and constipation follow.

**Diagnosis:** Definitive diagnosis of Babesiosis depends on demonstrating the causative organism in giemsa stained, thin blood smear.

**Treatment:** Berenil (diaminazine aceturate), IM

**Control:** Vector tick control

### 3.4. Fungal Infection

**General properties of fungi**

Fungus is classified in one of the five kingdoms called kingdom fungi. The five kingdoms are monera, protista, fungi, plantae and animalia.

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1. They are **eukaryotic**; cells contain membrane bound cell organelles including nuclei, mitochondria, golgi apparatus, endoplasmic reticulum, lysosomes etc. They also exhibit mitosis.

2. Have a rigid cell wall and are therefore **non-motile**, a feature that separates them from animals. All fungi possess cell wall made of chitin.

3. Are **chemoheterotrophs** (require organic compounds for both carbon and energy sources) and fungi lack chlorophyll and are therefore not autotrophic.

4. Fungi are osmiotrophic; they obtain their nutrients by absorption.

5. They obtain nutrients as **saprophytes** (live off of decaying matter) or as **parasites** (live on living matter).

6. All fungi require water and oxygen and there are no obligate anaerobes.

7. Typically reproduce **asexually and/or sexually** by producing spores.

8. They grow either reproductively by budding or non-reproductively by hyphal tip elongation.

9. Food storage is generally in the form of **lipids and glycogen**.

**Based on Morphology**

Fungi exist in two fundamental forms; the filamentous (hyphal) and single celled budding forms (yeast). But, for the classification sake they are studied as moulds, yeasts and dimorphic fungi.

1. **Moulds (Molds):** Are filamentous fungi. The thallus of mould is made of hyphae, which are cylindrical tube like structures that elongates by growth at tips. A mass of hyphae is known as mycelium. It is the hypha that is responsible for the filamentous nature of mould. Eg: Aspergillus sps, Trichophyton rubrum

2. **Yeasts:** Yeasts are unicellular spherical to ellipsoid cells. They reproduce by budding, which result in blastospore (blastoconidia) formation. Eg: Cryptococcus neoformans, Saccharomyces cerviciae

3. **Dimorphic:** Fungi existing in two different morphological forms at two different environmental conditions. They exist as yeasts in tissue and in vitro at 37°C and as moulds in their natural habitat and in vitro at room temperature. Eg: Histoplasma capsulatum, Blastomyces dermatidis, Paracoccidiodes brasiliensis, Coccidioides immitis

**Factors predisposing to fungal infections**

- Prolonged antibiotic therapy, Immunosuppressive drugs and Drug addiction
- Underlying disease (HIV infection, cancer, diabetes, etc.) commonly immunosuppressive
- Age
- Surgical procedures, Irradiation therapy, indwelling catheters and Transplants
- Occupation

**Laboratory diagnosis of mycoses:**

Specimen collection: specimen collection depends on the site affected. Different specimens include hair, skin scrapings, nail clippings, sputum, blood, CSF, urine, corneal scraping, discharge or pus from lesions, vaginal swab and biopsy. Hairs may be collected in sterilized paper envelopes

- All specimens must be transported to the laboratory without any delay to prevent bacterial overgrowth. Biopsy specimens must be transported in saline.

**Microscopy:** Microscopy is used to observe clinical specimens for the presence of fungal elements or to identify the fungus following culture. In the latter case, lactophenol cotton blue is stain of choice, which stains the fungal elements blue. Direct examination of clinical specimens could be stained or unstained.

- **Wet mount:** Candida may be collected in urine wet mounts
- **10-20% KOH mount:** The material is mixed with 20% KOH on a slide and a cover slip is placed. The slide is then gently heated by passing through the flame 2-3 times.
- **India Ink:** Capsules of Cryptococcus neoformans can be demonstrated by this negative staining technique.
- **Periodic Acid-Schiff (PAS) stain:** On staining by this stain, fungal elements appear bright magenta coloured while the background stains green. It is useful in staining tissue specimens.
• **Giemsa's stain**: It is particularly useful in the detection of Histoplamsa capsulatum in the bone marrow smears.

• **Haematoxylin and Eosin (H&E) stain**: Useful for staining tissue sections.

• **Gram stain**: Candida is best demonstrated in clinical specimen by Gram stain.

• **Immunofluorescence**: Monoclonal antibody labelled with fluorescent dyes can be used to detect several fungi in the clinical specimens.

**Culture**: One of the most common media used to culture fungi in laboratory is:

**Sabouraud's Dextrose Agar (SDA)**. It consists of peptone, dextrose and agar. High concentration of sugar and a low pH (4.5-5.5) prevents growth of most bacteria and makes it selective for fungi. Gentamicin or Streptomycin to SDA serves to inhibit bacterial multiplication.

Other basal media **Potato Dextrose Agar, Malt Extract Agar** etc. Most fungi are able to grow at room temperature while few pathogenic fungi (e.g, Cryptococcus, dimorphic fungi) can grow at 37oC. Saprophytic fungi grow much quickly than pathogenic fungi (e.g, dermatophytes). In such situations the saprophytic fungi can be inhibited by the addition of cycloheximide (actidione) to the SDA. Addition of antibiotics such as Chloramphenicol,

**Other specialized media used for different fungi include**:

• **Brain Heart Infusion Agar** general isolation of fungi and conversion of dimorphic fungi.

**Serology**: Detection of anti-fungal antibody is helpful in diagnosis of sub-cutaneous and systemic mycoses, prognosis and response to anti-fungal drugs. Different serologic techniques that are used include agglutination, immunodiffusion, complement fixation test, immunofluorescence, RIA and ELISA.

**Molecular techniques**: Newer techniques such as DNA hybridization, PCR are useful in diagnosis of mycoses in a shorter period as well as detect those fungi that are difficult or dangerous to cultivate in

**Mycoses are of growing importance for the following reasons**

1. They are produced by fungi that are widely distributed in the environment and, therefore, very difficult to eradicate. Most funguses are saprophytes.

2. The clinical manifestation of disease caused by fungal infection can be highly variable. For example, in the case of aspergillosis, with effects on very diverse organs, there is a variety of responses, such as local (aspergilloma), systemic (renal, lung, nervous central system, etc.) or even allergic (allergic bronchopulmonary aspergillosis in human).

3. Diagnosis of these diseases can be problematic because of the difficulty of interpreting the very different clinical pictures in individuals in the presence of colonization, infection and/or disease.

4. There are a few varieties of vaccines available against these diseases, which are therefore difficult to prevent. At this time, vaccines are limited to a few animal species, to only a few processes and with variable effectiveness.

5. Treatment is problematic: compared to the antibacterials, the number of antifungal drugs available at present is very small, with much greater difficulty in production, with many side-effects, and with the possibility of the appearance of resistance.

**Fungal Disease (Mycosis)**

**Aspergillosis (Brooder Pneumonia, Mycotic Pneumonia)**

Aspergillosis is a fungus disease occurring mainly in poultry but also in mammals and man. It is characterized by infection of the respiratory tract affecting chickens, turkeys, and less frequently ducklings, pigeons, canaries, geese, and many other wild and pet birds.

**Etiology and Epidemiology**: Aspergillus fumigatus is a common cause of the disease. Chicks and poults may become infected during hatching as a result of inhaling large numbers of spores in heavily contaminated hatching machines and eggs or from contaminated litter. In older birds, infection is caused primarily by inhalation of spore-laden dust from contaminated litter or feed or dusty range areas. The fungus grow particularly in hay or straw which has been balled or stacked too tightly while still with a water content higher than 25%.

**Clinical Findings and Lesions**: Dyspnea, hyperpnea, somnolence, and other signs of nervous system involvement, inappetence, emaciation, and increased thirst may be seen. Pulmonary lesions are characterized by cream-colored
plaques a few mm to several cm in diameter; occasionally, mycelial masses may be seen within the air passages on gross examination. Miliary nodules (Aspergillus granulomas) up to 3 cm across are found in the lung of chicken.

**Diagnosis:** The fungus can be demonstrated by culture or by microscopic examination of fresh preparations. Histopathologic examination using a special fungus stain reveals granulomas containing mycelia.

**Differential diagnoses** include infectious bronchitis, Newcastle disease and infectious laryngotracheitis.

**Treatment and Control:** Treatment of affected birds is considered useless. Strict adherence to sanitation procedures in the hatchery minimizes early outbreaks. Grossly contaminated eggs should not be set for incubation. Contaminated hatchers should be fumigated with formaldehyde or thiabendazole (120-360 g/m³). Avoiding moldy litter or ranges serves to prevent outbreaks in older birds. Pens should be sprayed with antifungals like nistatin, and all equipment cleaned and disinfected. Infected chicken should be destroyed and disposed by burning.

**Epizootic Lymphangitis**

Epizootic lymphangitis is a chronic granulomatous disease of the skin, lymph vessels, and lymph nodes of the limbs and neck of equines mainly horses.

**Etiology:** Epizootic lymphangitis is caused by the dimorphic fungus *Histoplasma farciminosum*. The fungus forms mycelia in nature and yeast forms in tissues and has a saprophytic phase in soil. Infection probably is acquired by wound infection or transmission by bloodsucking insects.

**CLINICAL FINDINGS AND LESIONS**

Clinically, the disease is characterized by freely movable cutaneous nodules, which originate from infected superficial lymph vessels and nodes and tend to ulcerate and undergo alternating periods of discharge and closure. Affected lymph nodes are enlarged and hard. Lesions also may be present in the lungs, conjunctiva, cornea, nasal mucosa, and other organs. The nodules are pyogranulomas with a thick, fibrous capsule and contain thick, creamy exudate and the causative organisms.

**Diagnosis:** The clinical signs are highly suggestive and can be confirmed by microscopic examination of exudates and biopsy specimens. The yeast forms of the organisms distend the cytoplasm of macrophages and appear in hematoxilin eosin stained sections as oval bodies (3-4 µm) with a central basophilic body surrounded by an unstained zone.

**Treatment and control:** No completely satisfactory treatment is known. But in early cases:

- Surgical excision of lesions combined with antiseptic dressing and antifungal drugs (early case)
- Parenteral iodides (Sodium iodide, IV)
- Isolation of affected animals while undergoing treatment
- Chronic cases don’t usually recover and it is better to advice destruction of such animals.
- Thorough disinfection of equipment that have come in contact with sick animals.

**Dermatophytosis (Ringworm)**

Ringworm is a contagious fungus infection of keratinized tissue (skin, hair and nails) by dermatophytes. It is found worldwide, and all domestic animals and humans are susceptible especially children and young animals, but it may affect people of all ages, as well as animals.

**Etiology:** Dermatophytosis is caused by a group of fungi collectively called dermatophytes: - *Epidermophyton* (human pathogen), *Microsporum*, and *Trichophyton*. The most important animal pathogens worldwide are *M. canis, M. gypseum, T. mentagrophytes, T. equinum, T. verrucosum*, and *M. nanum*.

**Transmission:** Contact with infected individuals and contaminated fomites by the lesion are responsible for the spread of the disease. The disease is commonly seen in calves in poor condition kept in unhygienic houses during wet season. Human beings can become infected by handling sick animals (zoonotic).

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**Clinical signs:** The **circular** (ring shaped) **lesions** from which the hair is lost (alopecia) are usually 2 to 3 cm in diameter with a grey scaly centre and red periphery, they usually occur first on the head. In bad cases many lesions may be present and join up to cover much of the body.

**Diagnosis:** **Fungal culture** is the most accurate means of diagnosis. **Direct microscopic examination** of hairs and scrapings from the periphery of lesions for fungal elements in a wet preparation of 20% potassium hydroxide that has been gently warmed or incubated in a humidity chamber overnight is also possible.

**Treatment** requires both systemic and oral treatment with most of the same drugs used in humans—terbinafine, fluconazole, or itraconazole as well as topical “dip” therapy.

**NON INFECTIOUS DISEASES**

**Bloat**

**Definition:** Bloat is the abnormal accumulation of gas in the ruminants’ forestomachs (rumen and reticulum).

**Etiology:** There are two categories of bloat (1) **primary** (frothy) bloat by diets that lead to the formation of a stable froth or foam in the rumen, (2) **secondary** (free gas) bloat caused by failure to eructate from extraruminal causes of gas accumulation such as esophageal obstruction.

1) **Primary or frothy bloat:** Is a bloat associated with lush green legume (such as fresh cut forage, winter wheat pasture) and concentrate finishing diet. Legume causing bloats are clover and alfalfa. In these plants the plant materials are easily fermented and the inner leaf cells are available to the bacterial activity. **Foaming or frothiness** which leads to high viscosity or surface tension of the fluid of the ruminal contents is the vital factor in causing primary pasture bloat. The rumen viscosity wills increase due to a number of plant components particularly soluble leaf proteins, saponins, pectins and hemicelluloses. The other factors which play a role in the persistence of the viscosity are the low pH, the activity and composition of rumen flora (an increase in slime-producing bacteria), rate and composition of saliva (salivary mucin is antifoaming agent and also buffers the pH of the rumen contents). Frothiness causes retention of gas within the mass of ingesta contained within the rumen and animal is unable to belch because no gas pocket is developed in the dorsal compartment of the rumen.

2) **Free gas bloat:** Ruminal tympany or free gas bloat usually occurs when fermentation within the rumen by the rumen bacteria is beyond the rate of eructation due to some highly fermentable feed. The major cause is physical obstruction to eructation in oesophageal obstruction caused by a foreign body (eg, potatoes, apples...), stenosis or by pressure from enlargements outside the oesophagus or by obstruction of the cardia from the interior that prevent eructation.

**CLINICAL FINDINGS**

- Sudden death of cattle on pasture and feedlot
- Distension of the rumen in the upper left flank and tympanic resonance up on percussion
- Abdominal pain and the animal get up and lie down frequently and kick at the belly
- Dyspnea accompanied with mouth breathing, protrusion of the tongue, salivation and extension of the head in severe cases and the respiratory rate may reach up to 60/minute

**Diagnosis:** Clinical diagnosis of frothy bloat is by history, clinical sign and passage of stomach tube to ascertain the cause of the failure of eructation. Bloat has to be differentiated from rupture of the urinary bladder, advanced pregnancy, left or right abomasal displacement and ascites.

**Treatment and Prognosis**

- **Stomach tube** to relief gaseous bloat
- Surgical intervention utilizing a rumen **trochar and canula.**
- **Emergency rumenotomy** may be necessary to evacuate the frothy contents.

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• Treatment of frothy bloat should include providing via the stomach tube one of the following: *mineral or vegetable oil*, or a surfactant such as *detergents* to break down the froth then the animal can eructate.

**Control and Prevention**

• Management practices like feeding hay before turning cattle on pasture
• To move animals to a new strip in the afternoon and
• Continual administration of an antifoaming agent during the risk period on the pasture
• Rations should contain ≥10–15% roughage mixed into the complete feed to prevent feedlot bloat preferably cereal, grain straw, grass hay, or equivalent.

**Simple Indigestion (Mild Dietary Indigestion)**

Simple indigestion is a minor disturbance in ruminant GI function that is seen in cattle and rarely in sheep.

**Etiology:**
• Sudden change in feed of beef cattle and high level of concentrate like grain ration
• Large quantity of poor quality straw and low protein intake and water deprivation

**Clinical Findings:**
• Reduced appetite, mild depression and dullness, absence of rumination and ruminal motility, rumen is full firm and doughy
• Temperature, pulse and respiration are normal and recovery usually is spontaneous within 24-48 hr.
• Simple indigestion due to excessive feeding of grain results in anorexia and ruminal stasis.

**Diagnosis:** History of a change in the nature or amount of the diet. As a differential diagnosis traumatic reticuloperitonitis, ketosis, displaced abomasum and vagal indigestion are considered.

**Treatment**

• Administration of **20-40 L of warm water or saline** via a stomach tube
• Magnesium hydroxide (laxative) to facilitate removal of the content through feces

**Grain Overload (Lactic Acidosis, Carbohydrate Engorgement)**

Grain overload is an acute disease of ruminants that is characterized by indigestion, rumen stasis, dehydration, acidosis, toxemia, incoordination, collapse and frequently death.

**Etiology:** Accidentally access to large quantities of readily digestible carbohydrates, particularly grain wheat, barley, and corn and less commonly bread, batter’s dough, and sugar beets. It also is common in feedlot cattle when they are introduced to heavy grain diets too quickly.

**Pathogenesis:** Ingestion of toxic amounts of highly fermentable carbohydrates is followed within 2–6 hr by a change in the microbial population in the rumen ► gram-positive bacteria (Streptococcus bovis) increases markedly ► large quantities of lactic acid (pH≤5) ► destroys protozoa, cellulolytic organisms ► impairs rumen motility► lactobacilli produce excessive quantities of lactic acid ► causes osmosis► excessive quantities of fluid into the rumen ► causing dehydration► hemoconcentration ► cardiovascular collapse ► shock ► death. The lactic acid ► chemical rumenitis, and its absorption ► lactic acidosis

**Clinical Findings:** At early stage-an enlarged rumen and abdominal pain (belly kicking).
• In severe grain overload, recumbency, staggering, and anorexia.
• Body temperature is subnormal; respiration and heart rate is increased in severe acidosis
• The rumen, as palpated feel firm and doughy.
• Recumbent animals with the head turned into the flank and response to any stimulus is much decreased
• Death may occur in 24-72 hr.

**Diagnosis:** If history is available it can be confirmed by the clinical findings and laboratory:
• **A low ruminal pH (<5), Normal pH 5.5-7**
• Microscopically; 5-7 protozoa in the fluid are normal but in acidosis no protozoa seen

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• A Gram's stain reveals a change from predominantly gram-negative bacteria (normal) to predominantly gram-positive bacteria in acidosis.

In differential diagnosis milk fever and peracute coliform mastitis can resemble acidosis Treatment: Restriction of water intake for the first 18-24 hr has to be the first measure.
• In serious cases, slaughter for salvage should be considered in feedlot animals.
• Emergency ruminotomy in severe cases and replacement of ruminal flora
• Rumen lavage and rigorous fluid therapy to correct the acidosis and dehydration
• 5% sodium bicarbonate solution should be given IV (5 L/450 kg) to correct the acidosis.

Prevention: Accidental access to concentrates for which the cattle are unaccustomed, should be avoided and feedlot cattle should be introduced gradually to concentrate over a period time.

PRODUCTION DISEASES

The term ‘production disease’ includes those diseases previously known as metabolic diseases’, such as parturient paresis (milk fever), hypomagnesemia, acetonemia and perhaps some other conditions, all of which are attributable to an imbalance between the rates of ‘input’ of dietary nutrients and the ‘output’ of production. In these diseases, output is greater than input either because of the selection of cattle which produce so heavily that no naturally occurring diet can maintain the cow in nutritional balance or because the diet is insufficient in nutrient density or unevenly balanced.

Hypocalcaemia in Cattle (Milk Fever)

A disease of cattle, sheep and goat occurring around the time of parturition and caused by hypocalcemia and characterized by weakness, recounence, reduced consciousness, circulatory collapse and ultimately shock and death. It occurs in older and highly lactating animals 12hrs before, during and maximum 48hrs after parturition. In sheep usually it occurs before parturition.

Major risk factors include increasing age of cow (intestinal calcium absorption decrease with age), production (high-producing dairy cattle) and dry period nutrition (high calcium).

Calcium homeostasis: There are three factors affect calcium homeostasis
• Excessive loss of calcium in the colostrum beyond the capacity of absorption from the intestines and mobilization from the bones to replace.
• Impairment of absorption of calcium from the intestine at parturition
• Mobilization of calcium from storage in the skeleton may not be sufficiently rapid to maintain normal serum levels.

Stages of milk fever and clinical findings

Stage I milk fever has short duration (< 1 hour). Signs observed during this stage include loss of appetite, excitability, nervousness, hypersensitivity and weakness
Stage II milk fever can last from 1 to 12 hours. The head turns to flank or may extend. The animal appears dull, listless, cold ears and a dry nose; she exhibits incoordination when walking; and muscles trembling are evident. A sub normal body temperature ranging from 35.6°C to 37.8°C and the heart rate will be 100 beats per minute.
Stage III milk fever is characterized by the animal's inability to stand and a progressive loss of consciousness leading to a coma. Heart rate exceeds 120 beats/ min. Unless treatment cows in stage III will not survive. Diagnosis
• Clinical sign and response to treatment with parenteral injections of calcium solutions
• By blood calcium level (hypocalcemia 4.2- <2.0 mg/dl serum). Normally it ranges from 4.3-5.1 mg/dl.

In ewes, the history usually contains some reference to recent physical stress and the disease is more common in the period preceding lambing.

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Differential diagnosis

- Other metabolic diseases like ketosis…
- Diseases associated with toxemia and shock
- Injuries to the pelvis and pelvic limbs

Treatment

Calcium borogluconate at 100 -200 g is the treatment of choice. For cattle, 400-800 ml of a 25% solution is the usual dose administered in sc or IV route depending up on the concentration.

Control:

- administration of calcium gels orally at the time of parturition
- Administration of vitamin D immediately before parturition to enhance intestinal absorption.
- Diets low in calcium during the prepartum period will reduce the incidence of milk fever in dairy cows.

Hypomagnesemic Tetany (Lactation Tetany, Grass Tetany, Wheat Pasture Poisoning)

Hypomagnesemic tetany is a disease of ruminants caused by deficiency of magnesium characterized by hypomagnesiumia, hypocalcaemia, muscular tetany or convulsion and death.

Etiology: Low magnesium concentration in the diet and the presence of competing cations such as potassium and sodium that affect either herbage magnesium status or magnesium absorption in the intestine.

Pathogenesis: Magnesium is the major intracellular divalent cation, and is an essential element in a large number of enzymatic activities in the body. Absorption, and the serum magnesium concentration, is influenced by the Na:K ratio in the rumen with maximum absorption occur at 5:1 ratio of Na: K. Young rapidly growing grass is low in sodium and high in potassium which falls Na:K ratio causing impairment of Mg absorption. Magnesium has many influences on impulse transmission at the neuromuscular system, including effects on the release of acetylcholine, on the sensitivity of the motor end plate, on the threshold of the muscle membrane and on activation of the cholinesterase system. These offer an attractive hypothesis for the muscular irritability seen with the disease.

Epidemiology: Disease of all classes of ruminants but reaches its highest incidence in older lactating cows grazing green cereal crops or lush grass-dominant pasture. Desorption of Mg from bone will decrease with age.

Clinical findings: In coordination, hyperesthesia and tetany, tonic-clonic muscular spasms and convulsions. High case fatality without treatment is common.

Diagnosis: Low serum, urine or cerebrospinal fluid (CSF) magnesium concentrations and response to treatment is confirmatory. As a differential diagnosis in cattle acute lead poisoning, Rabies and Nervous ketosis and in sheep hypocalcaemia has to be considered.

Treatment: Mg or combined calcium/magnesium solutions administered IV and/or SC. (e.g. 500 ml of a solution containing 25% calcium borogluconate and 5% magnesium hypophosphite for cattle, 50 ml for sheep).

Control: Magnesium supplementation orally and applied to pastures.

Ketosis or Hypoglycemia (Acetonemia in Cattle, Pregnancy Toxemia in Sheep)

Ketosis is a disease of ruminants caused by deficiency of glucose 10 to 60 days after calving in high-producing cows characterized hypoglycemia, acetonemia, acetonuria and nervous signs in cattle and toxemia in sheep.

Etiology and Pathogenesis: The diseases in cattle and sheep occur in different parts of the pregnancy-lactation cycle. The proposing factors are:-

- Primary ketosis (production ketosis) - need of excessive glucose due to heavy production
- Secondary ketosis- hypoglycemia due to less feed appetite following some abnormalities
- Alimentary ketosis-excessive butyrate in silage which is highly ketogenic
- Starvation ketosis-deficiency of propionate and protein from the diet for gluconeogenesis
- Ketosis due to deficiency of cobalt and possibly phosphorus. Cobalt is essential to metabolize propionic acid into the tricarboxylic acid (TCA) cycle.

The ruminant absorbs very little dietary carbohydrate as glucose (hexose sugar) because dietary carbohydrates are fermented in the rumen to short chain fatty acids, principally acetate (70%) converted to long chain fatty acid and

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stored as lipid, **propionate** (20%) precursor of glucose and **butyrate** (10%) secreted in milk as milk fat. Consequently glucose need in ruminants must largely be met by gluconeogenesis. Propionate and amino acids are the major precursors for gluconeogenesis with glycerol and lactate of lesser importance.

The most important etiological factor in pregnancy toxemia in sheep is a decline in the plane of nutrition during the last 2 months of pregnancy, particularly in ewes carrying twins or triplets and that have been well fed in early and mid-pregnancy.

**Pathogenesis:** Ruminants are particularly vulnerable to ketosis particularly in the cow in the period between calving and peak lactation because the demand for glucose is increased and cannot be completely restrained. Cows will reduce milk production in response to a reduction of energy intake, but due to hormonal stimuli for milk production overcome the effects of reduced food intake. Under these circumstances lowered blood glucose levels result in lowered blood insulin. Long chain fatty acids are released from fat stores this leads to increased production of acetoacetate (ketones) ketogenesis. A large proportion of butyrate produced by rumen fermentation of the diet like silage is converted to **beta-hydroxybutyrate** (BHBA) ketone.

**Clinical findings:** Cattle show **wasting** with decrease in appetite, **loss of body condition and milk production**. Some have short periods of neurological and behavioral abnormality such as walking in circles, head pushing and apparent blindness. Sheep have encephalopathy with blindness, muscle tremor, convulsions, metabolic acidosis and a clinical course of 2-8 day, usually terminating fatally unless treated early.

**Clinical pathology:** Hypoglycemia, ketonemia, ketonuria or elevated ketones in milk. Blood glucose levels are reduced from the normal of approximately 50 mg/dl to 20-40 mg/dl in cattle and sheep. Blood ketone levels are elevated from a normal of up to 10 mg/dl to 10-100 mg/dl.

The **differential diagnosis** for the nervous form includes rabies, hypomagnesaemia and bovine spongiform encephalopathy.

**Treatment:** In cattle, parenteral glucose with corticosterone and oral glucose precursors such as propylene glycol, occasionally insulin. In cattle, the disease responds readily to treatment and is self-limiting, but in sheep the disease is highly fatal.

**Control:** Correction of energy imbalance. Herd and flock biochemical monitoring coupled with condition scoring. Cows should neither have been starved nor be over fat at calving and dry cows with condition scores of 4 on a 1-5 scale.

**MINERAL DEFICIENCY**

**Cobalt Defficiency (Enzootic Marasmus) or Wasting Disease**

It is a metabolic defect arising from cobalt deficiency characterized clinically by clinically anorexia and wasting in ruminants.

**Etiology:** Deficiency of cobalt in the soil, plant and animal body. It is common disease in the hot costal parts of the country like south eastern, eastern low land areas following the east African rift valley. Ruminants are the only susceptible groups among which sheep is the most and young are the most susceptible. Other species are less susceptible (pig and pets) as their diet is rich in vitamin B₁₂. In general soil which is sandy and volcanic is rich in calcium carbonate and manganese which is low in cobalt because they decrease is availability in the soil.

**Pathogenesis:** The process of change of propionic acid to vitamin B₁₂ enzymes requires cobalt and these enzymes are important for energy production and assimilation of nitrogen. In the absence or low cobalt in the diet these process fails and that end up with loss of appetite and death

**Clinical sign**

- Gradual loss of appetite and anorexia
- Loss of body weight and emaciation (marasmus)
- Poor growth and increase mortality in young

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Laboratory findings

- Low cobalt in soil, plant and animal tissue
- Low serum vitamin B12 level
- Anemia

**Differential diagnosis:** Fasciolosis, GIT parasitism, Starvation and John’s disease but Co deficiency is a herd problem

**Treatment:** Cobalt as salt lick, Oral cobalt solution and IM injection of vitamin B12

**Copper Deficiency (Hypocuprosis)**

It is disease of grazing ruminants caused by low level of copper in plasma characterized by bone deformity, anemia and change in hair and wool color and development.

**Etiology:** Deficiency of copper in diet (primary deficiency)

- Problem in tissue utilization (secondary deficiency) Mb and so₄ in the GIT reduce copper absorption and also

\[
\text{Cu + Se} = \text{copper deficiency}
\]

Coastal areas which are sandy and bleached are usually deficient in copper and cobalt.

Copper is a component of enzymes like cytochrome oxidase which are important in energy production at cell level.

**Pathogenesis**

- Anemia: hemoglobin synthesis require copper
- Loss of body weight: due to reduced appetite
- Wool and hair: conversion of thylosine to melanin will not occur in the absence of copper thus pigmented animal will be white around the eye (spectacled eye)
- Bone disorder: conversion of osteoblasts to osteocyte will not occur thus in copper deficiency the bone remain large and fractures
- Nerve disorder and blindness: synthesis of phospholipid require copper dependent enzymes
- Reproductive failure: fertilization and implantation do not occur in copper deficient animal

**Clinical sign:** untriftiness in young and loss of body condition in adults, anemia, change in coat color (spectacled eye), diarrhea, enzootic ataxia (in coordination of movement) and sway back (congenital copper deficiency = hind limb paralysis)

**Diagnosis**

- History feed and area location
- Clinical sign and response to treatment

**Treatment:** Oral copper administration and copper in the form of mineral lick

**Other important deficiencies**

**Delayed puberty** is largely attributable to diets greatly decreased in energy fed to young, growing animals. **Growth retardation** can also develop as a result of many nutritional factors, most commonly deprivation of energy. Additional factors may include salt (sodium chloride), cobalt, copper, iron, zinc and some B vitamins in calves. **Hair coat roughness** is a rather subjective evaluation but is related to deficiency of energy and can also be seen in deficiency of phosphorus and salt.

**Deficiency of calcium, Vitamin D and phosphorous: Osteomalacia**, which is characterized by weak, brittle bones that may fracture when stressed, can develop after demineralization of the bones of aged animals. Feeding a diet low
in calcium to lactating cows over a long period of time may cause a depletion of calcium and phosphorus, resulting in fragile, easily fractured bones plus decreased milk production, without affecting calcium level in the milk produced. 

**Rickets** is characterized by improper calcification of the organic matrix of bone, which results in weak, soft bones that lack density. Signs include swollen, tender joints; enlarged bone ends; an arched back; stiffness of the legs; and development of beads on the ribs. Rickets is a disease of young animals and may be caused by deficiencies of calcium, phosphorus, or vitamin D. **Bone calcification retardation** is generally attributed to a vitamin D deficiency but is rare because sunlight is so effective in converting the provitamin D (7-dehydrocholesterol) of the skin to active vitamin D.

The nutritional causes of **retained placenta** appear to be rather complex and include deficiencies of selenium, copper, and iodine. The incidence increases with parturient hypocalcemia and appears to be related to fat cow syndrome. Prepartum injection of selenium has reduced the incidence of retained placenta. There is a genetic implication, and such cows should be considered strong candidates for culling.

**White muscle disease** normally is seen in young calves (or lambs) and is associated with deficiencies of selenium or vitamin E, or both. Affected animals have chalky white striations, degeneration, and necrosis of cardiac and skeletal muscle. In addition, paralysis of the hind limbs and a dystrophic tongue may be evident. Heart failure is also associated with a selenium deficiency.

**Vitamin A deficiency**: **Xerophthalmia** (degenerative condition of the eye), **corneal lesions**, **hair coat roughness**, **retained placenta** and **growth retardation** are all usually associated with advanced vitamin A deficiency.

**Iodine deficiency**: **Thyroid gland enlargement** (goiter) is the first sign. Iodine deficiency may be seen in cattle that are consuming an "adequate" level of iodine if they are also consuming fairly large quantities of crops of the cruciferae family, such as turnips or cabbage. Affected cows may give birth to hairless calves.

**Cardiac arrhythmia** is usually associated with a long and severe deficiency of sodium in the diet.

**Zinc deficiency**: **Dermatitis** can be seen in both calves and mature cows due to zink deficiency, usually in the range of <10 ppm of dietary zinc. Generally, it is most severe on the legs, neck, and head and around the nostrils. Wounds are slow to heal. Additional signs associated with zinc deficiency include decreased testicular growth, listlessness, development of swollen feet with open scaly lesions, and alopecia.