**UNIT.1   
1.General Concepts of animal health**

**Concept of disease (infection) and its importance**

**Disease** is defined `The sum of the *abnormal phenomena* displayed by a group of living organisms in association with a *common characteristic or set of characteristics* by which they differ from the norm of their species in such a way as to place them at a biological or economic disadvantage'

**Disease** can also defined as an *alteration of the state of the body*, or of some of its organs, which interrupts the proper performance of the body functions.

**Impact of Animal Disease**

* Animal Health

Death, illness, loss of production

* Economics

Loss or disruption of trade

Loss of consumer confidence

Movement restrictions

* Human Health

Zoonoses

Mental health

* Costs would also be generated due to the needed measures for eradication and control of the disease
* The occurrence of any of these diseases may have trade implications, including blockage and import restrictions

**Infectious disease**

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

**Non-infectious disease** it is a disease, which is caused by non-living agents or factors like

Injury plant or animal poison cold excessive heat or faulty nutrition.

**Different stages of diseases**

**Acute disease**

An **acute** condition is one where symptoms appear suddenly and worsen rapidly.

An acute disease lasts for just a short time but can begin rapidly and have intense (strong) symptoms. An acute disease can be mild, severe or even fatal.

The term "acute" does not indicate the severity of the disease.

Instead, it indicates how long the disease lasts and how quickly it develops.

**Chronic disease**

A chronic condition is an animal health condition or disease that is persistent or otherwise long-lasting in its effects.

Chronic condition is one that develops gradually and worsens over an extended period of time.

The term chronic is usually applied when the course of the disease lasts for more than three months. Like an acute disease, a chronic disease can be mild, severe or fatal.

**Subacute Diseases**

Diseases that fall between what normally are considered acute diseases and chronic diseases are referred to as subacute diseases. A disease might be considered acute at first, then subacute after a few days or a few weeks. If the disease continues for several months, it might then be called a chronic disease.

**Modes of transmission and maintenance of infection**

* Infectious disease is the result of the invasion of a host by a pathogenic organism.
* The continued survival of infectious agents, with or without the induction of disease, depends on their successful transmission to a susceptible host, the instigation of an infection therein and replication of the agent to maintain the cycle of infection.

**Horizontal transmission**

Horizontally transmitted infections are those transmitted from any segment of a population to another. Infections can be transmitted horizontally either directly or indirectly.

Direct transmission occurs when a susceptible host contracts an infection, either by physical contact with an infected host or by contact with the infected discharges. Indirect transmission involves an intermediate vehicle, living or inanimate, that transmits infection between infected and susceptible hosts.

**Vertical transmission**

Vertically transmitted infections are transmitted from one generation to the next by infection of the embryo or fetus while in utero (in mammals) or in ovo (in birds, reptiles, amphibians, fish and arthropods).

There are two types of vertical transmission: hereditary and congenital.

*Hereditarily* transmitted diseases are carried within the genome of either parent.

*Congenitally* transmitted diseases are, literally, those present at birth.

**Methods of transmission**

Six main methods of transmission, which bring infectious agents into contact with the sites of infection, can be identified:

1. Ingestion

2. Aerial transmission

3. Contact

4. Inoculation (vaccination)

5. Iatrogenic transmission (treatment)

6. Coitus (sexually)

**Maintenance strategies**

The ways in which infectious agents are maintained can be considered as strategies for maintenance. Five main strategies can be identified:

1. avoidance of a stage in the external environment

2. the development of resistant forms

3. a 'rapidly in, rapidly out' strategy.

4. persistence within the host

5. extension of host range

**UNIT 2  
Factors influencing disease occurrence**

Disease is caused by multiple factors. The factors are called determinants of disease.

A determinant is any characteristic that affects the health of a population. Knowledge of determinants facilitates identification of categories of animal that are at particular risk of developing disease. It is therefore a prerequisite for disease prevention, and is an aid to differential diagnosis

**Classification of determinants**

Determinants commonly are classified into those associated

* with the host,
* the agent and
* the environment

These three groups of factors are sometimes called the triad.

**Host determinants**

*Factors associated with the host (Intrinsic factors)*

**Age**

The occurrence of many diseases shows a distinct association with age. Many bacterial and virus diseases, for instance, are more likely to occur, and to be fatal, in young than in old animals.

Many protozoan and rickettsial infections, in contrast, induce milder responses in the young than in the old.

**Sex**

Sexual differences in disease occurrence may be attributed to hormonal, occupational, social and ethological, and genetic determinants

Susceptibility may be vary between sexes due to anatomical and /or physiological differences between sexes such as in case of metritis

**Species and breed**

Species and breeds vary in their susceptibility and responses to different infectious agents, and therefore in the role they play in disease transmission.

The reasons for species susceptibility are many and not fully understood.

The efficacy of the immune mechanism against an infectious agent may be important

Different species have been shown to have different receptors for infectious agents on the cell surface. Phylogenetically closely related animals are likely to be susceptible to infection by the same agent, although with different signs. Apparently new diseases can develop when a species or breed is placed in a new ecosystem that contains a pathogen that has a well-balanced relationship with local species or breeds.

Resistance to the tick *Boophilus microplus* is greater in indigenous Zebu cattle (Bos indicus) than in European cattle (Bos taurus)

There is also species and breed variation in the occurrence of non-infectious diseases.

***Factors associated with disease causing agents (Intrinsic factors)***

Infectious agents vary in their ability to infect and to induce disease in animal.

The ability to infect is related to the inherent (natural) susceptibility of a host and whether or not the host is immune. The ability to induce disease is expressed in terms of virulence and pathogenicity

***Virulence*** is the ability of an infectious agent to cause disease, in a particular host, in terms of severity.

***Case fatality*** is an indicator of virulence when death is the only outcome.

***Pathogenicity*** is sometimes incorrectly used as a synonym for virulence, with virulence reserved for variations in the disease inducing potential of different strains of the same organism

However, 'pathogenicity' refers to the quality of disease induction (introduction)

**Environmental determinants *(Extrinsic factors)***

The environment includes location, climate and husbandry

***Location***

Local geological formations and vegetation affect the spatial distribution of both animals and disease

***Climate***

Two types of climate can be identified: macroclimate and microclimate.

**Macroclimate**

* + The macroclimate comprises the normal components of weather to which animals are exposed
* Rainfall
* temperature
* solar radiation all of which can affect health
* humidity and
* wind

The macroclimate can also affect the stability of infectious agents. Climatic variation can also induce changes in disease occurrence over vast distances

**Microclimate**

Microclimate is a climate that occurs in a small, defined space.

This may be as small as within a few millimeters of a plant’s or an animal’s surface. In the former, microclimate may be terrestrial (e.g., over the surface of leaves) or biological (e.g., over the surface of a host's body)

The terrestrial microclimate affects the development of arthropods and helminths.

The biological microclimate can change during the course of a disease, assisting in its spread.

***Husbandry***

* + Housing

The importance of well-designed ventilation and the structure of bedding materials and surfaces is also a determinant

Thus, claw lesions are more common and severe in pigs reared on aluminium slats than in pigs reared on steel or concrete slabs, or on soil

* + Diet

Diet has obvious effects in diseases caused by energy, protein, vitamin and mineral deficiencies.

Feeding regimes may be a determinant. Thus, gastric torsion in sows kept in sow stalls has been associated with once-a-day rather than twice-a-day feeding, which may indicate that the ingestion of a relatively large amount of food is a causal factor.

* + Management (including animal use)

Management determines stocking density and production policy.

Increased densities increase the challenge of microbial pathogens.

An internal replacement policy is less likely to introduce pathogens into an enterprise than a policy involving buying in animals from outside the herd.

* + Stress

In veterinary medicine, it is often considered as arising from factors such as weaning, overcrowding, transportation, changes in diet and other environmental factors.

In such case, normal microflora can cause a disease, e.g pasteurellosis.

**Unit 3.   
Major Animal Diseases and their** **management**

***Non-infectious diseases***

***1. Parturient paresis (milk fever)***

A disease of cattle, sheep, and goats occurring around the time of parturition and caused by hypocalcemia and characterized by weakness, recumbency, and ultimately shock and death.

This disease is less common in beef cattle, sheep, and goats. Most commonly within 48 h after calving but also occurs several weeks before or after. Usually, there is a distribution of cases around parturition.

* + **Etiology**

A depression of the levels of calcium in tissue fluids is the basic biochemical defect in milk fever. Most cows adapt within 48 h after calving by increases in plasma concentrations of parathyroid hormone and vitamin D at the onset of the hypocalcemia and mobilize calcium by increasing intestinal absorption and bone resorption.

* + **Clinical Signs**

Three progressively worse stages including the following signs

***Stage I.***

The signs consist of mild *excitement* and *tetany (involuntary muscular contraction)* without recumbency. Anorexia is also a consistent finding. These signs may go unobserved because stage I rapidly progresses to stage II (1-hour progression).

***Stage II***

(a) There is depression, paralysis, and recumbency. The head is characteristically turned into the flank or rested on the ground in an extended position. Fine muscle tremors (shake) may be evident, and the cow may make threatening motions with the head (e.g., head shaking, open-mouth bellowing).

(b) Examination reveals tachycardia (rapid heartbeat) with decreased heart sounds, cool extremities, and a low rectal temperature (35.5"C-37.8"C). Gastrointestinal atony (e.g., mild bloat, constipation), and a slow pupillary light reflex are evident.

Stage II may last from 1 to 12 hours.

***Stage III***

Cattle with stage III milk fever exhibit further weakness and progressive loss of consciousness.

Bloat may be life threatening because of lateral recumbency and gastrointestinal tract (GIT) atony. There is a danger of aspiration pneumonia, the heart sounds become inaudible, and a pulse may be undetectable. This stage may progress to death in 3-4 hours.

* + **Diagnostic plan**

The field diagnosis is based on clinical signs, history, and response to therapy.

At the time of treatment, serum should be drawn and held for calcium analysis.

(***2) Administer calcium.***

(a) Administer 500 ml of 23% calcium gluconate intravenously (calcium borogluconate and glucose, phosphorus, magnesium, and potassium are often found in commercial preparations).

Intravenous injections must be given slowly (over 20 minutes)

Within 30 minutes of treatment, 60% of animals with uncomplicated milk fever will stand.

(b) Subcutaneous calcium. A subcutaneous calcium preparation without glucose (one 500-mi bottle) is administered as well.

***(3) Follow-up***

Full restoration of calcium homeostasis does not occur for 2-3 days.

The owner should request a revisit if there has been no response to therapy within 12 hours of treatment.

**2. Bloat (ruminal tympany)**

Is over distention of abdominal cavity due to impaired or inhibited and the rate of gas production exceeds the animal’s ability to expel the gas.

Bloat occurs more commonly in cattle than other ruminants, and certain individual cattle may be more susceptible than others. Bloat can be divided into two types: frothy bloat and free gas bloat.

**Etiology**

* + ***Frothy bloat*** is associated with legume consumption

(a) It is thought that the fine, thin leaf structure of certain varieties of legumes coupled with tender growth (early or late season) allows for more rapid bacterial degradation and intra ruminal particle suspension.

Chloroplast released from the legume leaf forms monomolecular foams that trap gas bubbles.

These foams have great surface tension and are highly stable.

(b) The result is that small gas bubbles do not coalesce, the cardia or the forestomach cannot be cleared of this foam, and the animal is unable to eructate.

(c) A stable froth can also be formed in feedlot animals consuming a primarily finely ground grain diet. In this case, however, a mucoprotein slime stabilizes the foam. This foam is stable at a low pH created by lactate and VFA production. Salivation is decreased because of the fine grind of the diet, which also lessens intra ruminal buffering.

* + ***Free gas bloat*** may have a variety of causes.

Irregular feed intake may occur as a result of uneven feeding intervals, illness (such as a mild grain overload), changing weather, a change in the diet, Unpalatable(bad) feed resulting from spoilage, or an interruption in the supply of salt or water.

Certain postures or diseases also can produce a functional free gas bloat. Examples include milk fever and tetanus. Moderate free gas bloat also may be a finding in vagal indigestion

Excess acidity in the lower gut can inhibit rumen movement.

If rumen movement is inhibited as a result of grain overload or for any other reason, free-gas bloat might develop. Cattle that have overcome severe respiratory disease often suffer from chronic feedlot bloat as a result of damage to the vagal nerve.

If a foreign object creates a physical obstruction in the esophagus, gas is trapped in the rumen and causes acute free-gas bloat. Blockages can occur in a number of ways, but happen most frequently when an animal swallows a large object that does not pass into the rumen.

Feeding whole potatoes, beets, carrots, or fruit has caused problems.

Pathological growths occurring in the esophagus or rumen that form a blockage can also cause free-gas bloat.

**Clinical findings**

Animals present with severe abdominal distention.

The distention initially is restricted to the left dorsal abdominal quadrant, but in severe cases, the right flank will also distend.

The rumen may be hyper motile or hypo motile.

Animals have excessive salivation and are anxious.

The animal is uncomfortable and may get up and lie down frequently, defecate often, kick at the belly, and roll over in attempting to relieve the discomfort.

Breathing is difficult or labored (a condition known as dyspnea) and occurs through the mouth.

The animal protrudes the tongue, salivates, and extends the head.

**Diagnosis**

Clinical sign

An accurate history and the passage of an orogastric tube rapidly determines whether the condition is one of abdominal distention resulting from the accumulation of ruminal free gas or froth. If the tube cannot be passed, then the likely diagnosis is free gas bloat due to esophageal obstruction. If the tube can be passed but ruminal gas is not readily forthcoming, then frothy bloat is likely.

**Treatment**

Treatment approaches depend on the circumstances in which the bloat occurs, whether the bloat is frothy or free-gas and whether or not it is life-threatening

In mild bloat, the left flank is distended, the animal is not in distress, and skin over the upper flank can be easily grasped and lifted.

In moderate bloat, a more obvious distension of the rumen is evident, particularly of the left flank.

The animal may appear uncomfortable, and the skin over the upper left flank is usually tight, but still can be grasped and tented.

In severe bloat, gross distension of both sides of the rumen is evident, especially on the left side, and the animal may breathe through the mouth and protrude the tongue.

The animal is uncomfortable and may be staggering.

The skin over the left upper flank is tense and cannot be grasped and tented.

All animals should be removed immediately from the source of the bloat-provoking pasture or feed.

In severe cases, in which gross distension, mouth-breathing, protrusion of the tongue, and staggering occur, an emergency surgery called a rumenotomy is necessary to save the animal’s life. The passage of a stomach tube is recommended when mild to moderate distension of the rumen occurs. Even in severe cases, the stomach tube should be used because it is the least traumatic method for the release of rumen gas and pressure.

With frothy bloat, the tube becomes plugged by froth immediately upon entering the rumen.

The operator should clear the froth from the end of the tube by blowing through it and moving it back and forth to locate pockets of gas.

With frothy bloat, it may be impossible to reduce the pressure, and an anti-foaming agent such as oil should be administered while the tube is in place.

Any non-toxic oil, especially a mineral oil that persists in the rumen, is an effective bloat treatment. The rate for treatment is 300 to 500 mL for a 450 kg animal, administered in one dose.

This treatment can be repeated several times within a few hours if necessary.

The anti-foaming agent can be administered with a stomach tube or with a large syringe directly into the rumen through the flank. The anti-foaming agent may also be delivered as a drench, using a standard drenching procedure.

***3. Lactic acidosis (Ruminal acidosis)***

Acidosis is a pathological condition associated with the accumulation of acid or depletion of alkaline reserves in blood and body tissues, and characterized by increased hydrogen ion concentrations. Ruminal acidosis refers to a series of conditions that reflect a decrease in pH in the rumen of cattle.

Rumen lactic acidosis develops in sheep and cattle that have ingested large amounts of unaccustomed feeds rich in ruminally fermentable carbohydrates

The resulting production of large quantities of volatile fatty acids (VFA) and lactic acid decreases rumen pH to non-physiological levels,

Simultaneously weakening the buffering (protecting) capacity of the rumen, and reduces the efficiency of rumen flora (motility) and fermentation.

* + **Etiology**

Acidosis in cattle is caused by excessive ingestion of feeds which are rich in readily available carbohydrates. Thus, factors which contribute to excessive ingestion of high energy diets are predisposing to acidosis.

Glucose is liberated from starch granules by specific strains of microbes that attach to the grain particle. Presence of free glucose in the rumen can have at least **three** adverse effects.

**First**, ruminal bacteria that normally are not competitive can grow very rapidly when provided with high amounts of glucose.

Streptococcus bovis, an inefficient microbe that thrives only when free glucose is available

**Second,** other opportunistic microbes, including coliforms and amino acid decarboxylating microbes, may thrive in the rumen of cattle fed concentrate diets and produce or, during lysis, release endotoxins or amides (e.g., histamine; when glucose is readily available).

**Third,** free glucose released from starch increases the osmolality of ruminal contents.

An increased osmolality exacerbates accumulation of acid within the rumen by inhibiting VFA absorption.

* + **Signs of Acidosis**

When acidosis develops in the animal the following physiological changes have been observed. Anorexia, diarrhea, mucous in feces, in coordination and, sometimes, death.

Increased level of lactic acid in the rumen and blood.

Reduction in rumen pH and blood pH.

Increased osmotic pressure in the rumen.

Destruction of gram-negative bacteria and proliferation of gram-positive bacteria in the rumen.

Reduction in rumen protozoal count.

Rumenitis and sloughing of rumen epithelium.

Rumen stasis.

Reduced urine pH.

Dehydration and hemoconcentration.

* + **Diagnosis**

Clinical sign and detail history of animal

While various diagnostic tools have been employed to detect acidosis, particularly the prevalence of cows with rumino centesis pH <5.8,

A ruminal pH of 5.6 to 5.8 suggests a marginal or developing problem of ruminal acidosis, while a pH of greater than 5.9 is considered ‘normal’

* + The faeces of stock can provide indirect evidence of clinical and sub-clinical acidosis and lack of fiber in the diet

Typically, the faeces of acidotic stock are liquid, contain undigested fiber and grain, are often lighter in color and may contain gas bubbles. Faecal smearing around the perineum can indicate acidosis. The smell of the faeces may be bitter-sweet, rather than the typical herbaceous character found from stock fed on grass.

* + **Treatment**

Treatment of severe cases (dehydration >8%, collapsed and subnormal temperature, static rumen and evidence of scouring) can be unrewarding, time consuming and expensive.

Severe cases should be treated by withholding concentrates, giving intravenous fluids, e.g. hypertonic saline and access to water or balanced electrolyte solutions not containing lactic acid.

Treatment of mild cases of acidosis includes withholding concentrates and feeding hay to stimulate saliva flow.

Additional therapy includes oral antacids such as magnesium hydroxide, magnesium oxide or sodium bicarbonate at 1 g/kg body weight initially to alkalinize the rumen, and oral electrolyte solutions, preferably those containing additional sodium bicarbonate to treat metabolic acidosis.

Antibiotics including penicillins, tylosin, potentiated sulphonamides and tetracycline should be given to reduce the risk of liver abscessation

**Prevention**

Two common management practices that help to prevent acidosis are diluting the diet with roughage or modulating/reducing intake of starch.

Dietary roughage decreases eating rate and meal size. Increasing the concentration of dry roughage increases chewing time and saliva production.

Although an increased extent of mastication will decrease size of grain particles entering the rumen and thereby increase its rate of fermentation, an increased input of buffers from saliva from a longer chewing time or rumination neutralizes and dilutes ruminal acids.

Avoiding allowing of Animals to feed forage-based diets with small particle sizes (i.e. diets containing hays and silages with short chop lengths).

Avoiding allowing of Animals to low pH silages (especially maize, high moisture grains, and whole grain silages).

Change processing of grain from fine to coarse.

Add buffers, neutralizing agents and rumen modifiers to the diet.

**4. Traumatic reticuloperitonitis (TRP)**

Is a relatively common disease in adult cattle caused by the ingestion and migration of foreign bodies in the reticulum. Swallowed metallic objects, such as nails or pieces of wire, fall directly into the reticulum or pass into the rumen and are subsequently carried over the ruminoreticular fold into the cranio ventral part of the reticulum.

Contractions of the reticulum and the pressure of the calf during late pregnancy or the efforts of parturition promote penetration of the wall by the foreign object.

However, the development of severe sequel to penetration of the reticular wall depends on the characteristics of the foreign body, as well as the direction and extent of the penetration

Perforation of the wall of the reticulum allow leakage of ingesta and bacteria, which contaminate the peritoneal cavity, resulting in local or diffuse peritonitis

The swallowed object can also penetrate into the pleura cavity, causing pleuritis and pneumonia and into then pericardial sac, causing pericarditis, myocarditis endocarditis and septicaemia (invasion of blood stream/blood poisoning).

Occasionally, the foreign body may pierce and infect the liver or spleen.

**Host**

Cattle are more susceptible to foreign body syndrome than small ruminants because they do not use their lips for prehension and are more likely to eat chopped feed

Moreover, the honeycomb-like structure of the reticulum provides many sites for fixation of a foreign body.

**Risk factors**

Traumatic reticulo peritonitis is most common in mature dairy cattle, occasionally seen in beef cattle, and rarely reported in other ruminants.

Cattle commonly ingest foreign objects because they do not discriminate against metal materials in feed and do not completely masticate feed before swallowing.

The disease is common when green chop, silage, and hay are made from fields that contain old rusting fences or baling wire, or when pastures are on areas or sites where buildings have recently been constructed, burned, or torn down.

The grain ration may also be a source due to accidental addition of metal.

**The clinical signs**

Cattle with TRP are variable, depending on the severity, duration and involvement of other organs.

Fever, increased heart and respiratory rate, anorexia, dehydration, decreased milk production, weight loss, ruminal stasis, chronic tympani and rigid abdominal pain with grunting.

Cows with TRP also had arched backs, reluctance to move, abduction of the forelimbs and severe dullness and depression, and edema of the brisket and submandibular regions.

Forced sudden movements as well as defecating, urinating, lying down, getting up, and stepping over barriers may be accompanied by groaning.

**Diagnosis**

This can be based on history (when available) and clinical findings if the cow is examined when signs initially appear.

Without an accurate history and when the condition has been present for several days or longer, diagnosis is more difficult.

Ultrasonography of the ventral abdomen is the most accurate means of diagnosing localized peritonitis near the reticulum and characterizing the reticular contraction frequency.

It rarely identifies the presence of a penetrating object.

Ultrasonography of the heart and thorax is very useful in the diagnosis of pleuritis and pericarditis as a sequelae to traumatic reticuloperitonitis.

Lateral radiographs of the cranioventral abdomen can detect metallic material in the reticulum.

Electronic metal detectors can identify metal in the reticulum but do not distinguish between perforating and nonperforating foreign bodies.

**Treatment**

Two treatment procedures for TRP were recommended; conservative therapy and surgery.

If hardware disease is suspected, a magnet should be administered orally through a tube into the rumen. Even if the animal already has one magnet, there is no harm in inserting a second.

A broad-spectrum antimicrobial agent such as oxytetracycline (16 mg/kg, IV, sid) should be used to control infection. The cow should be confined and movement limited in the hopes that the reticulum can repair the hole. Surgery involves rumenotomy with manual removal of the object(s) from the reticulum; if an abscess is adhered to the reticulum, it should be aspirated (to confirm that it is an abscess) and then drained into the reticulum. *Antimicrobials s*hould be administered perioperatively.

**Prevention**

Preventive measures include avoiding the use of baling wire

Keeping cattle away from sites of new construction, and completely removing old buildings and fences. Additionally, bar magnets may be administered PO, preferably after fasting for 18–24 hr.

Usually, the magnet remains in the reticulum and holds any ferromagnetic objects on its surface.

***5. Intestinal obstruction***

It is the occlusion of the intestinal lumen either from the external pressure or from other physical occlusions or intestinal displacement.

It is characterized by abdominal pain, absence of defecation or often the passage of blood and mucus tinged faces.

It occurs in all large animals but is most common in horses.

**Types**

* **Intussusception**
  + - is a form of obstruction in which a part of intestine turns in (invaginate) on itself. Usually results from irregular peristaltic movements, associated with enteritis, intestinal parasites, dietary disorders, and bowel tumours.
  + **Volvulus**
    - is a form of obstruction produced by the twisting of a loop of intestine round its mesenteric axis
* **Torsion**
  + is obstruction caused by the bowel twisting on its own long axis. Volvulus and torsions occur most frequently in horses, caused by strenuous exercise, jumping, rolling, etc. and verminous arthritis.
* **Strangulation**
  + is an obstruction caused by volvulus or intussusception in which blood supply to a loop of intestine trapped in a hernia form; its development causes tissue necrosis and consequently toxemia and death (unless it is corrected).
* **Incarceration**
  + is a condition in which some part of intestine passes through a torn peritoneum and lie under the skin just like hernia.

small intestine torsion

**Clinical findings**

Signs associated with abdominal pain

Increased respiratory and pulse rate

Dehydration, gastric dilatation

Absence of defecation.

**Diagnosis**

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

**Treatment**

Some impaction may be treated with mineral oil. However, most true obstructions usually require surgery (laparotomy)

1. **Anthrax**

**Synonyms:** Charbon, Malignant pustule, Splenic fever, Woolsorter’s disease.

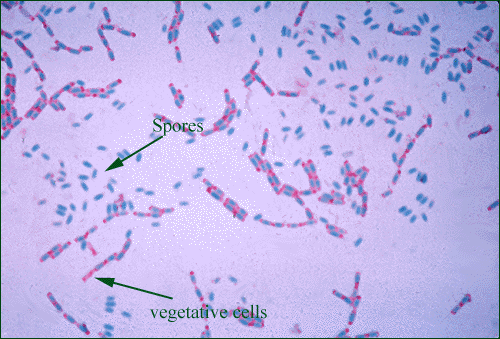
**Definition:** It is a highly infectious and fatal direct bacterial zoonosis. Ancient disease

**Occurrence:** Worldwide. Endemic in Asia, Africa and Middle East. Practically disappeared from Australia and Western Europe. Incidence of disease in some tropical countries is as high as 100000 cases a year.

**Host:** Man, cattle, goat, sheep, antelope, bison, buffalo, camel, cat, deer, dog, elephant, fox gaur, hippopotamus, horse, kangaroo, leopard, lion, mink, primate, rhinoceros, tiger.

**Etiology**

* Anthrax is a bacterial disease caused by the spore forming *Bacillus anthracis, a Gram-positive, rod shaped*, capsulated, non-motile bacterium
* When organisms come out of the host’s body, spores are formed in the presence of air (oxygen). Capsules develop in the body of infected host. Endospores remain viable in soil at least 12 years.
* Spores are markedly resistant to biological extremes of heat, cold, pH, desiccation, chemicals(and thus to disinfection), irradiation and other such adverse conditions.

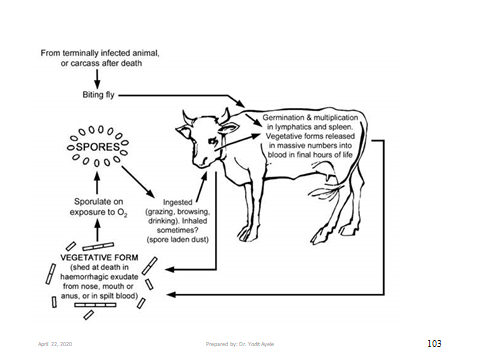


**Cycle of infection and transmission**

* When conditions are not conducive to growth and multiplication of the vegetative forms of *B. anthracis,* they start to form spores.
* The infected host sheds the vegetative bacilli onto the ground and these sporulate on exposure to the air.
* The spores, which can persist in soil for decades, wait to be taken up by another host, when germination and multiplication can again take place upon infection
* It is very largely through the uptake of spores from the environment that anthrax is contracted.
* Ingestion of the spores while grazing is a frequent mode of uptake

**Generally transmission occur by:**

* 1. Direct contact with diseased animal, infected tissue, body discharge and contaminated nonliving object (fomite).
  2. Inhalation of *B. anthracis* spores while working with animal products such as hair, wool, hide, skin, leather, fertilizer, animal protein (blood meal).
  3. Accidental inoculation of organism into skin.
  4. Arthropods may act as a mechanical vector in the spread of infection.



**Seasonality**

* + Anthrax is a seasonal disease.
  + Typically, an outbreak in an enzootic area follows a prolonged hot dry spell, which in turn was preceded by heavy rains or flooding, or with rain ending a period of drought

**Clinical manifestations**

* + The normal incubation period in naturally-infected cattle is stated to be 1–14 days or more
  + During the systemic phase, the animals become distressed, appear to have difficulty breathing and cease eating and drinking.
  + Severe dyspnoea, anorexia, tremor, congestion of mucosa, ruminal statis, diarrhoea, dysentery, oedema of throat, sternum, bloody discharges from natural orifices, staggering gait and abortion.
  + Swellings in the submandibular fossa may be apparent; temperatures may remain normal for most of the period or may rise.
  + In highly susceptible species, the period between onset of visible symptoms and death may be just a few hours.
  + The blood characteristically clots poorly or not at all upon death from anthrax and is dark (sometimes described as “tarry”) and partially haemolysed.

**Diagnosis**

* 1. Clinical symptoms and history of the patient.
  2. Detection of organisms in blood smear by MacFadyean’s reaction.
  3. Isolation of pathogen on blood agar from blood, pus etc…
  4. Inoculation of specimen in guinea pig (0.5ml SC); in case of anthrax, animal will die within 30-40 hours and blood smear/spleen smear will reveal large capsulated bacilli.





**Treatment**

* Erythromycin, Penicillin (4-30 million units/d), Penicillin plus Streptomycin, Oxytetracycline, Tetracycline can be tried.
* However, treatment is not feasible in animals.

**Prevention**

* Control in animals. Annual vaccination protects.
* Disposal of animal carcasses: disinfect with oil, burn, bury deep, covered with quicklime.
* Spores will NOT form inside the carcass, and putrefaction kills the *Bacillus*.
* Flies feeding on incoagulable blood may be a problem.





**2. BLACKLEG**

* **Etiology** 
  + It is associated with *Clostridium chauvoei,* a Gram­postIve, spore-forming, rod-shaped bacterium
  + The spores are highly resistant to environmental changes and disinfectants and persist in soil for many years
* **Host**
* common only in cattle but infection with this organism initiated by trauma occurs occasionally in other animals
* **Epidemiology** 
  + When the disease occurs, it is usual for a number of animals to be affected within the space of a few days.
  + Cattle 6 months to 2 years of age that are rapidly growing and on a high plane of nutrition.
  + Seasonal occurrence in warm wet months.
  + There are often multiple cases in at-risk animals.
  + Sheep of all ages - occurs as outbreaks predisposed by wounds from shearing, docking, castration, dystocia
  + **Source of infection**
  + Blackleg is a soil-borne infection but the portal by which the organism enters the body is still in dispute.
  + It is presumed that the portal of entry is through the alimentary mucosa after ingestion of contaminated feed
  + The bacteria may be found in the spleen, liver, and alimentary tract of normal animals, and
  + contamination of the soil and pasture may occur from infected feces or decomposition of carcasses of animals dying of the disease
* **Transmission** 
  + In cattle the disease usually occurs with­out a history of trauma but in sheep is almost always a wound infection.
  + The disease is not transmitted directly from sick animals to healthy animals

**Risk factors**

***Environment risk factors***

* + Typical blackleg of cattle has a seasonal incidence, with most cases occurring in the warm months of the year
  + occurred following excavation of soil, which suggests that disturbance of the soil may expose and activate latent spores.

*Animal risk factors*

* + In cattle the disease is largely confined to young stock between the ages of 6 months and 2 years, although disease occurs occasionally in younger animals and cattle up to 3 years
  + In the field, risk factors include rapidly growing cattle and a high plane of nutrition
  + In sheep there is no restriction to age group.
  + **Pathogenesis**
  + Toxin formed by the organism produces a severe necrotizing myositis locally in skeletal muscles, and a systemic toxemia that is usually fatal.
  + In cattle and sheep atypical outbreaks of sudden death occur in which the lethal lesion is a clostridial cardiac myositis.

**Clinical findings**

* + If the animal is observed before death there is severe lameness, usually with pronounced swelling of the upper part of the affected leg
  + Characteristics swellings can also develop in the hip, shoulder, chest, back and neck
  + On closer examination the animal will be found to be very depressed and have complete anorexia and ruminal stasis, and a high temperature (41°C)
  + In the early stages the swelling is hot and painful to the touch but soon becomes cold and painless, and edema and emphysema can be felt.
  + The skin is discolored and soon becomes dry and cracked.
  + Occasional cases are seen where the lesions are present in other locations such as the base of the tongue, the heart muscle, the diaphragm and the brisket, and the udder.
  + The condition develops rapidly and the animal dies quietly 12-36 hours after the appearance of signs.
  + Many animals die without signs having been observed
  + Incision of the affected muscle mass reveals dark red to black

**Diagnosis**

* + A diagnosis can be reached on the basis of clinical signs and necropsy findings
  + The swelling is spongy and if pressed it gas can be felt under the skin.
  + *C. chauvoei* can be identified by the staining of lesion impression smears
* Confirmation Fluorescent antibody identification of *C. chauvoei* in lesion
* Cultural examination is likely to be unrewarding unless fresh tissue is available and special techniques used.

**Treatment**

* + Treatment of affected animals with penicillin and surgical debridement of the lesion,
  + Recovery rates are low because of the extensive nature of the lesions.
* **3.TUBERCULOSIS**
* It is an important highly infectious bacterial disease.

**Synonyms**

* Great white plague, Pearl disease, Scorofula.

**Occurrence**

* Worldwide; eradicated in cattle from certain areas (Britain, Canada, Denmark, Finland, Switzerland)

**Etiology**

* *M. bovis*, *M. tuberculosis* and also *M. avium* which are nonsporing, nonmotile, aerobic or microaerophilic, acid-fast, slender bacilli, resistant to many disinfectants, grow slowly.

**Host**

* Cattle, goat, sheep, Man, cat, chimpanzee, cockatoo, deer, dog, elephant, horse, leopard, monkey, parrot, pig, wild animal.
* All species and age groups are susceptible to *M. bovis*, with cattle, goats, and pigs most susceptible.

**Source of infection**

* + Infected cattle are the main source of infection for other cattle.
  + Organisms are excreted in the exhaled air, in sputum, feces, milk, urine, vaginal and uterine discharges, and discharges from open peripheral lymph nodes.
* **Transmission**
  + Commonly entry is effected by inhalation or ingestion.
  + Inhalation is the almost invariable portal of entry in housed cattle, and even in those at pasture it is considered to be the principal mode of transmission.
  + Inhalation of infectious droplets or droplet nuclei from active pulmonary lesion of a tuberculous infected person/animal.
  + Indirect contact with tubercule bacilli contaminated articles or dust.
  + Direct invasion of bacilli through mucous membrane or abraded skin
* **Tuberculosis (*Mycobacterium bovis) mode of transmission***
* **Pathogenesis**
  + The primary complex consists of the lesion at the point of entry and in the local lymph node.
  + A lesion at the point of entry is common when infection is by inhalation.
  + When infection occurs via the alimentary tract, a lesion at the site of entry is unusual.
  + A visible primary focus develops within 8 days of entry being effected by the bacteria.
  + Calcification of the lesions commences about 2 weeks later.
  + The developing necrotic focus is soon surrounded by granulation tissue, monocytes, and plasma cells and the pathognomonic 'tubercle' is established.
  + Bacteria pass from this primary focus, which is in the respiratory tract in 90-95% of cases In cattle.
  + Post-primary dissemination from the primary complex may take the form of acute military tuberculosis, discrete nodular lesions in various organs, or chronic organ tuberculosis caused.
  + In cattle, horses, sheep, and goats, the disease is progressive and, although generalized tuberculosis is not uncommon in pigs
  + **Clinical signs**
  + Chronic cough , noisy breathing and dyspnoea
  + Depression , weight loss and emaciation
  + Fever, lymphadenopathy, dysphagia
  + intestinal ulceration, diarrhoea
  + weakness, osteomyelitis, mastitis , infertility
* **Diagnosis**
  + Direct microscopic examination of sputum, pus, pleural fluid, exudates, lung biopsy etc… for the presence of acid fast bacilli with Ziehl Neelsen staining technique
  + Isolation of organism from sputum, pleural fluid, CSF, urine etc… on Lowenstein Jensen (LJ) medium. The specimen should be properly homogenised and decontaminated by using 2% sodium hydroxide.
  + Guinea pig is also inoculated to recover the tubercle bacilli from samples.
  + Radiography is helpful to detect pulmonary lesion
  + Intradermal tuberculin test- PPD
  + DNA amplification

Treatment

* Bovine TB is extremely difficult to treat and animals are culled rather than having any treatment even attempted.
* If the cow is treated, it can take as long as nine months and involves using various antibiotics at different intervals as a result of its resistant nature.

**Prevention**

Preventative measures are essential in the prevention of this disease. Wildlife such as badgers should have reduced contact with cattle.

Areas storing feed should be protected against contact with wildlife as they could spread the disease.

* New cattle brought in should be tested and proven to not have any TB present in their system. It is also recommended that they be isolated and observed for a period of time prior to being introduced to the herd.
* Regular tests are made to control the incidence in the area. Cattle that have tested positive are culled.
* **4. BRUCELLOSIS**
* Brucellosis is a chronic granulomatous infection caused by intracellular bacteria and requires combined, protracted antibiotic treatment.

**Synonyms**

* + Bang’s disease, Contagious abortion, infectious abortion, epizootic abortion

**Occurrence**

* + Worldwide. The disease can cause an economic loss of up to 20% to a country’s beef industry. 20 -25% loss in milk production..

**Etiology**

* + Disease is caused by *Brucella abortus, Brucella ovis, B. melitensis* and *B. suis* which are small, non-spore-forming, non-motile, gram negative, rod-shaped organisms.
  + Intracellular and localize in the tissues of the reticulo endothelial system (RES) and organs such as the uterus and udder
  + Sensitive to direct sunlight, disinfectants and pasteurization.
  + Survive up to 60 days in damp soil and up to 144 days at 20 0C and 4% relative humidity

**Host**

* + Cattle, goat, sheep, Man, bear, bison, buffalo, camel, deer, dog, elk, horse, pig, poultry, reindeer, yak.
  + Infection occurs in cattle of all ages but is most common in sexually mature animals, particularly dairy cattle.
  + Brucellosis in cattle is prevalent in many countries of Africa.
  + Studies on bovine brucellosis in Ethiopia indicated seroprevalence ranging from 0.2% up to 22%.

**Pathogenesis**

* + The organism 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 and iliac lymph nodes.
  + *B. abortus* is phagocytized by macrophages and neutrophils in an effort by the host to eliminate the organism
  + The phagocyte migrates via the lymphatic system to the draining lymph node, where *Brucella* infection causes cell lysis and eventual lymph node hemorrhage 2-3 weeks following exposure.
  + Because of vascular injury, some of the bacteria enter the bloodstream and subsequent bacteremia occurs, which disseminates the pathogen throughout the body.
  + If the infected animal is pregnant, *B. abortus* will colonize and replicate to high numbers in the placenta of the developing fetus.
  + The resulting tissue necrosis of the fetal membranes allows transmission of the bacteria to the fetus. The net effect of chorionic and fetal colonization is abortion during the last trimester of pregnancy.
  + Erythritol, 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.
  + In the testes there are uni – or bila teral visceral to parietal tunica adhesions.
  + Interstitial lymphocyte orchitis with seminiferous tubular degeneration, necrotizing intratubular orchitis
  + Calcified sperm, epididymitis, seminal vesiculitis

**Transmission**

* + The disease is transmitted by ingestion, penetration of the intact skin and conjunctiva, and contamination of the udder during milking.
  + Grazing on infected pasture, or consuming other feedstuffs and water supplies contaminated by discharges and fetal membranes from infected cows, and contact with aborted fetuses and infected newborn calves are the most common methods of spread
  + Intra herd spread occurs by both vertical and horizontal transmission.
  + Congenital infection may occur in calves born from infected dams but its frequency is low.
  + Bulls do not usually transmit infection from infected to non infected cows mechanically.
  + Susceptibility to infection depends on age, sex, breed and pregnancy status of the animal.

-Younger animals are more resistant to infection than mature animals.

-In females pregnancy has positive contribution to degree of susceptibility.

-In contrast to bull, boars are more likely to be a source for introducing *Brucella* into a swineherd

* **Clinical sign**
  + Abortion
  + Orchitis and epididymitis
    - One or both scrotal sacs may be affected, with acute, painful swelling to twice normal size, although the testes may not be grossly enlarged.
  + Synovitis
    - Progressive and erosive non suppurative arthritis of the stifle joints

**Diagnosis**

* + Clinical signs and history of the case.
  + Serological tests
  + Isolation of bacteria from blood, bone marrow, CSF, urine, synovial fluid, etc…
* Detection of gamma-G-antibody by Coomb’s test particularly in chronic cases.
* Biological test
* Milk ring test
* PCR

**Treatment**

* + Treatment is unsuccessful because of the intracellular sequestration of the organisms in lymph nodes, the mammary gland, and reproductive organs.
  + In severe case Streptomycin (1g, 1M) and Tetracycline (500 mg/6h PO) for 3 weeks; in mild ones Tetracycline (500 mg/12 hr PO) for 2 weeks
  + To reduce Relapses after Rx, combined treatment: doxycycline 6 wks and streptomycin 2-3 wks or rifampin for 6 wks
* **Control and eradication**
  + Test and reduction of reservoir of infection.
    - All breeding cattle in the herd are tested and those that are positive are culled.
    - This removes infected cows from the herd and reduces exposure and transmission within the herd.
  + Quarantine
    - This will prevent inter herd transmission by infected cattle, especially those that are test-negative and incubating the disease.
  + Depopulation
    - Depopulation is slaughter of all cattle in a herd when all animals have been exposed and are capable of becoming infected and acting as a source of new infection.
  + Vaccination
    - Provides increased resistance against field strain infection following natural exposure.
* **5. MASTITIS**
* Is inflammation of the parenchyma of the mammary gland regardless of the cause. **Mammae = breast -itis = Latin suffix for inflammation**
* Mastitis is therefore characterized by a range of physical and chemical changes in the milk and pathological changes in the glandular tissue
* The most important changes in the milk include discoloration, the presence of clots and the presence of large numbers of leukocytes.
* There is swelling, heat, pain and edema in the mammary gland in many clinical cases.

What’s the significance of bovine mastitis ?

* The most costly disease affecting dairy cattle throughout the world
  + Animal health
    - Loss of functional quarter
    - Lowered milk production
    - Death of cow
  + Human health
    - Poor quality milk
    - antibiotic residues in milk
    - Milk born disease
* **Type and causes**
* Based on their epidemiology and patho physiology, these pathogens have been further classified as

- contagious,

- teat skin opportunistic or

- environmental mastitis

**Contagious mastitis pathogens**

* + There are many contagious mastitis pathogens.
  + The most common are *Staphylococcus aureus* and *Streptococcus agalactiae*.
  + The usual source of contagious pathogens is the infected glands of other cows in the herd;
  + However, the hands of milkers can act as a source of *S. aureus*.
  + Udder wash, cloths, residual milk in teat cups and inadequate milking equipment.
  + The predominant method of transmission is from cow to cow by contaminated common house or equipments, Milkers hand
  + *Mycoplasma bovis* and *Corynebacterium bovis* are a less common cause of contagious mastitis
* **Environmental mastitis pathogens** 
  + Environmental pathogens are found in the immediate surroundings of the cow, such as the sawdust and bedding of housed cows, the manure of cattle and the soil.
  + Environmental mastitis is associated with three main groups of pathogens, the coliforms (particularly *E. coli* and Klebsiella spp.), environmental Streptococcus spp. and *Arcanobacterium pyogenes.*
  + The major method of transmission is from the environment to the cow by inadequate management of the environment.

Examples

* + - Include wet bedding
    - Dirty lots
    - Milking wet udders
    - Inadequate pre milking udder and teat preparation,
    - Housing systems that allow teat injuries, and
    - Poor fly control

**Teat skin opportunistic mastitis pathogens**

* The incidence of mild clinical mastitis associated with bacterial pathogens that normally reside on the teat skin
* Teat skin opportunistic pathogens have the ability to create an intra mammary infection via ascending infection through the streak canal.
* Coagulase-negative staphylococci are the most common teat skin opportunistic mastitis pathogens.
* **Pathogenesis**
* Infection of the mammary gland mostly occurs via the teat canal and on first impression the development of inflammation after infection seems a natural sequence.
* However, the development of mastitis is more complex than this and can be most satisfactorily explained in terms of three stages:
  + - **invasion**
    - **infection**
    - **inflammation**

**Invasion**

* + is the stage at which pathogens move from the teat end to the milk inside the teat canal.

**Infection**

* + is the stage in which the pathogens multiply rapidly and invade the mammary tissue.
  + After invasion the pathogen population may be established in the teat canal and, with this as a base, a series of multiplications and extensions into mammary tissue may occur, with infection of mammary tissue occurring frequently or occasionally depending on its susceptibility.
    - Multiplication of certain organisms may result in the release of endotoxins, as in coliform mastitis, which causes profound systemic effects with minimal inflammatory effect .

**Inflammation**

* + Follows infection and represents the stage at which clinical mastitis occurs with varying degrees of clinical abnormalities of the udder and variable systemic effects from mild to per acute; gross and subclinical abnormalities of the milk appear.
  + Abnormalities of the udder include marked swelling, increased warmth and, in acute and peracute stages
* gangrene in some cases and abscess formation and atrophy of glands in chronic stages. The systemic effects are due to the mediators of inflammation.
* Once at the site of infection, neutrophils phagocytose and kill pathogens.
* Neutrophils exert their bactericidal effect
* As they colonize and multiply in the mammary gland, some bacteria release metabolic byproducts or cell-wall components (endotoxin if a Gram-negative bacteria) that serve as chemoattractants for leukocytes.
  + If neutrophils move rapidly from the blood stream and are able to eliminate the inflammatory stimuli (bacteria), then recruitment of neutrophils ceases and the sec returns to normal levels.
  + If bacteria are able to survive this immediate host response, then the inflammation continues
  + Prolonged diapedesis of neutrophils damages mammary tissue, resulting in decreased milk production
* **Clinical findings**
* The clinical findings in mastitis include abnormalities of secretion, abnormalities of the size; consistency and temperature of the mammary glands and, frequently, a systemic reaction.
* In other words, there are three categories of clinical mastitis: abnormal milk, abnormal gland and an abnormal cow (systemic disease)
* Abnormal milk is visibly abnormal (i.e. is not 'drinkable').

- Discoloration may be in the form of blood-staining or wateriness,

* Clots or flakes are usually accompanied by discoloration and they are always significant, indicating a severe degree of inflammation
  + Flakes at the end of milking may be indicative of mammary tuberculosis in cattle.

Abnormal milk

* + Discoloration
    - Blood-staining
    - Wateriness
    - Clots or flakes
    - Pus

**Abnormal gland**

* + Abnormalities of size and consistency of the quarters may be seen and felt.
  + Palpation is of greatest value when the udder has been recently milked, whereas visual examination of both the full and empty udder may be useful.
  + Although in most forms of mastitis the observed abnormalities are mainly in the region of the milk cistern, the whole of the quarter must be palpated.
  + Palpation and inspection of the udder are directed at the detection of fibrosis, inflammatory swelling and atrophy of mammary tissue.
  + Acute inflammatory swelling is always diffuse and is accompanied by heat and pain and marked abnormality of the secretion.
  + In severe cases there may be areas of gangrene, or abscesses may develop in the glandular tissue.
  + The terminal stage of chronic mastitis is atrophy of the gland.

**Abnormal cow (systemic response)**

* + A systemic response including toxemia, fever, tachycardia, ruminal stasis, depression, recumbency and anorexia may or may not be present, depending on the type and severity of the infection.
  + A systemic response is usually associated with severe mastitis associated with E. coli, Klebsiclla spp. or A. pyogenes and occasionally with Streptococcus spp. Or Staphylococcus spp.
* Clinical mastitis episodes are also categorized according to their **severity** and **duration**. Severity is characterized as:
* Severity and duration is characterized as:
  + **Peracute**: severe inflammation, with swelling, heat and pain of the quarter, with a marked systemic reaction, which may be fatal. (as in E. coli and Klebsiella.)
  + **Acute**: severe inflammation without a marked systemic reaction. (as in E. coli and Klebsiella.)
  + **Subacute**: mild inflammation with persistent abnormality of the milk. (as in S. aureus, S.dysagalactiae)
  + **Chronic;** persistent abnormality of the milk and udder.( S. agalactiae and M. bovis)
* **Diagnosis**
* Detection of clinical mastitis
  + The initial diagnosis of clinical mastitis is made during the routine ***physical examination***
  + ***Laboratory culturing*** of milk samples for bacteria and Mycoplasma spp.
* Detection of subclinical mastitis
  + culturing large numbers of milk samples
  + Screening tests; milk from quarters or cows with a positive screening test are then submitted to ***bacteriological culture***.
  + Subclinical mastitis can only be detected by laboratory examination
* Indirect tests include SCCs using automated electronic counters, the ***California Mastitis Test***, increases in ***electrical conductivity of milk***, and increases in the activity of cell associated enzymes (such as ***NAGase***) in milk.
* Hematology and serum biochemistry
  + In severe clinical mastitis there may be marked changes in the leukocyte count, packed cell volume and serum creatinine and urea nitrogen concentration because of the effects of severe infection and toxemia
* Physical examination
  + **Signs of inflammation**
  + **Empty udder**
  + **Differences in firmness**
  + **Unbalanced quarters**
* Culture analysis
  + The most reliable and accurate method
  + **Treatment**
* The treatment of the different causes of clinical and subclinical mastitis may require specific protocols
* IMM Therapy
  + Injection of antibiotics into udder
* Systemic Therapy
  + Antibiotics IV or IM
* Supportive Therapy
  + Remove toxins – Frequent milkout
  + Treat dehydration, swelling and pain
* Dry Cow Therapy
  + Larger dose, longer acting product
* IMM Therapy
  + Use an approved product
  + Use proper technique
  + Have culture reports and sensitivities
  + Don’t give up on a certain antibiotic, often response is seen with longer course of therapy
  + Have a standard protocol
* **Prevention**
* Milk cow with clean, dry teats and teat ends.
  + **Impact:** Milk quality, environmental mastitis
* Prevent transfer of pathogens from cow to cow during milking.
  + **Impact:** Contagious mastitis, milk quality
* Prevent injury to the teats during milking.
  + **Impact**: Mastitis, milk out
  + Provide an environment that allows the cows to remain clean between milking.
  + **Impact:** Environmental mastitis, milk quality, cow comfort
  + Early detection of new infections (clinical and subclinical).
  + **Impact**: Response to treatment, chronic infections, culling
* Proper use of medications.
  + **Impact**: Success of treatment, cost control, residues in milk and meat
* Assume all purchased replacements are infected.
  + **Impact:** Control introduction of new pathogens
* Provide routine milker training
  + **Impact:** All areas of mastitis prevention and control, milk quality
  + **6. PASTEURELLOSIS**
* Septicemic pasteurellosis of cattle (hemorrhagic septicemia or barbone), commonly associated with infection by *P. multocida,* is characterized by a peracute septicemia and a high mortality rate.
* Pneumonic pasteurellosis of cattle, commonly associated with infection by *Mannheimia (formerly Pasteurella) haemolytica* and *P. multocida*

**Shipping Fever**

* Pneumonic pasteurellosis, is known as shipping fever, is an entity within the bovine respiratory disease complex, characterized clinically by acute bronchopneumonia with toxemia.
* *Mannheimia haemolytica* is the most common cause of the pneumonia. and *P.multocida* is isolated occasionally.

**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.
* Environmental and management risk Factors
  + The mixing of cattle from different sources is an important risk factor.
  + Transportation
  + Confinement of cattle
  + Ineffective housing and ventilation
  + Confinement in humid and poorly ventilated barns, exposure to inclement weather, fatigue and deprivation from feed and water are commonly followed by outbreaks of the disease
* Pathogen risk factors
  + The frequency of isolation of *Pasteurella* spp. from the nasal passages of normal healthy unstressed calves is low but increases as the animals exposed to stress.

**Method of transmission**

* + Transmission of pasteurellas probably occurs by the inhalation of infected drop lets coughed up or exhaled by infected animals, which may be clinical cases or recovered carriers in which the infection persists in the upper respiratory tract.
* *M. haemolytica* and *P multocida* are highly susceptible to environmental influences and it is unlikely that mediate contagion is an important factor in the spread of the disease.
* When conditions are optimal, particularly when cattle are
  + closely confined in inadequately ventilated barns, or
  + when overcrowded in trains and
  + held for long periods in holding pens in feedlots,

the disease may spread very quickly and affect a high proportion of the herd within 48 hours

* **Pathogenesis**
* Which are part of the normal flora of the upper respiratory tract, colonize first the upper respiratory tract then the lower respiratory tract.
* Under normal conditions, alveolar macrophages will effectively clear pasteurellas from the alveoli by phagocytic mechanisms.
* When the large numbers of organisms enter and colonize the lung they interact with alveolar macrophages.
* Endotoxin interacts with numerous cell types and humoral mediator systems, resulting in widespread injury to the lung.
* Death is due to hypoxemia and toxemia.
* Complications include pulmonary abscessation, chronic pleuritis with or without pleural effusion, pericarditis and, rarely, congestive heart failure
* **Clinical findings**
* Animals found died without any previous signs may be the first sign of an out break.
* When viewed from a distance, affected cattle are depressed and their respirations are rapid and shallow.
* There may be a weak protective cough, which becomes more pronounced and frequent if they are urged to walk.
* A mucopurulent nasal discharge, a crusty nose, and an ocular discharge are common.
* Auscultation of the thorax of some of these subclinical cases will reveal rapid shallow respirations and an increase in the loudness of the breath sounds.
* The typical case of pneumonic pasteurellosis reveals a fever of 40-41 oc,
* The course of the disease is only 2-4 days.

**Diagnosis**

* Clinical finding and history of the animal.
* Bacterial culture
  + Nasal swabs taken from clinical cases before treatment often yield a pure culture of pasteurellas
* Hematology
  + Plasma fibrinogen concentrations are elevated, paralleling the increase in body temperature, and are a more reliable indication of the presence of the lesion than clinical assessment.

**Treatment**

* The recommendations for the treatment of bovine pneumonic pasteurellosis are based on clinical experience and the results of clinical field trials.
* About 85-90% of affected cattle recover within 24 hours if treated with antimicrobials such as oxytetracycline, tilmicosin, the sulfonamides, and penicillin.
* **7. Foot and mouth disease (FMD)**
* Synonym: Aphthous fever
* Foot-and-mouth disease is a highly contagious, viral disease of domestic cloven-hoofed and many wild animals characterized by erosions in the mucosa of the mouth and hooves.
* The disease is rarely fatal in adult animals but there is often high mortality in young animals
* **Occurrence:** The disease is endemic in South America, central Europe and central African countries notably in Ethiopia and Tanzania

**Hosts**

* + The disease affects cattle and swine as well as sheep, goats, and other cloven-hoofed ruminants.
  + All species of deer and antelope as well as elephant, and giraffe are susceptible to FMD

**Etiology**

* + The organism which causes FMD is an aphthovirus of the family Picornaviridae.
  + There are seven strains (A, O, C, SAT1, SAT2, SAT3, Asia1)
* **Transmission**
  + FMD is found in all excretions and secretions from an infected animal.
  + The virus may be present in milk and semen for up to 4 days before the animal shows clinical signs of disease.
  + Animals that have recovered from infection may serve as carriers of the virus.
  + Infected animals notably breathe out a large amount of aerosolized virus, which can infect other animals via the respiratory or oral routes
* The signiﬁcance of FMD is related to the ease of virus spread through any or all of the following:
  + new animals carrying the virus (saliva, milk, semen, etc.) may introduce the disease to a herd;
  + contaminated pens, buildings or vehicles used to house and move susceptible animals;
  + contaminated materials such as hay, feed, water, milk or biologics;
  + people wearing contaminated clothes or footwear, or using contaminated equipment;
  + meat or animal products, raw or improperly cooked food infected with the virus and fed to susceptible animals, and;
  + aerosol spread of virus from an infected property via air currents.
  + Aerosol transmission possible up to 250 km depending on strain and environmental conditions

**Transmission**

* Aerosols/Airborne, Inhaled
* Direct or indirect contact-droplets,
* Ingestion
* Artificial Insemination
* Vectors (vehicles, equipment, or humans)
* Carrier state

**Clinical signs**

* + The severity of clinical signs will depend on the strain of virus, the age and species of animal.
  + The signs can range from a mild infection to severe.
  + Clinical signs are more severe in cattle and intensively reared pigs than in sheep and goats.
  + Excess salivation, drooling, nasal discharge
  + Lethargy, loss of body condition
  + The typical clinical sign is the occurrence of blisters (or vesicles) on the nose, tongue, lips, oral cavity, between the toes, above the hooves, teats and pressure points on the skin.
* Ruptured blisters can result in extreme lameness and reluctance to move or eat.
* Secondary bacterial infection of open blisters can also occur.
* Other symptoms often seen are fever, depression, hyper salivation, loss of appetite and weight, drop in milk production.
* Blisters usually heal within 7 days or longer, however the impact of the disease on growth or milk production rates may persist after recovery.
* Oral lesions (vesicles)
  + Tongue, dental pad, gums, soft palate, nostrils, muzzle
  + Progress to erosions
  + Excess salivation, drooling, nasal discharge
* Hoof lesions
  + Interdigital space
  + Coronary band
  + Lameness
  + Reluctant to move
  + sloughing of hooves
  + Teat lesions
  + Decreased milk production
* Abortion
* Death in young animals

**Diagnosis**

* Clinical sign
* Serum neutralization test
* Complement fixation test
* Enzyme-linked immunosorbent assay
* PCR
* Virus isolation

**Treatment**

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

**Prevention and control measures**

* control over access to livestock by people and equipment;
  + Control the introduction of new animals to existing stock;
  + Maintain sanitation of livestock pens, buildings, vehicles and equipment ;
  + monitor and report illness;
  + Appropriate disposal of manure and dead carcasses.
  + In endemic countries or zones, culling may be complemented by vaccination for susceptible livestock.
  + Vaccines used must protect against the particular virus strain prevalent in the area.
* **Parasitic Diseases** 
  + Definitions, importance
  + Internal parasites (Nematodes, Trematodes and Cestodes)
  + External parasites (ticks, lices, fleas. Mangemites and flies)
  + Other important parasites (Trypanosomosis and Coccidiosis)
* **Parasitic Diseases**

Parasitic diseases are caused by organisms that live within an animal (endoparasites) or on its surface (ectoparasites).

* *Endoparasites* include worms and flukes;
* *Ectoparasites* include fleas,mites and ticks.
* Disease is seldom caused by one or a few parasitic organisms, but as a rule depends on mass infestations. There are exceptions to this, however, as a single Ascaris may obstruct the bile-duct with fatal results.
* Parasites, with few exceptions, do not spend all their lives in the animal body, but always need to spend a certain proportion of their life-cycle outside the host. They may cause damage to the host in the following ways:

(1) By abstraction of nourishment properly belonging to the host, e.g. many of the intestinal worms;

(2) By mechanical obstruction of passages or compression of organs, e.g. gapes (in chickens) and hydatid;

(3) By feeding on the tissues of the host, e.g. blood-sucking worms or flies;

(4) By production of toxins with varying effects;

(5) By actual traumatic damage, e.g. by piercing and destroying skin (ticks, mites, flies, etc.), by depositing eggs in the tissues (lung-worms), by migrations of larvae (Ascaris and Trichinella), by clinging to surfaces by means of sharp hooks (tapeworms), and in many other ways;

(6) By facilitating the entrance of bacteria, e.g. stomach worms in pigs allow the entrance of Fusiformis necrophorus (the necrosis bacillus);

(7) By transmitting diseases for which they act as intermediate hosts, e.g. ticks and babesiosis;

(8) By causing inflammatory or neoplastic reactions in the invaded tissues, e.g. pneumonia, gastritis, and fluke adenomata in the liver.

* These are only some of the more obvious methods of injuring the host. Apart from the loss due to actual deaths, the depreciation in value of hides, meat, milk, and work is enormous, and, although less spectacular than a bacterial epizootic, the loss is more constant, and in the aggregate is probably even greater than the loss due to bacterial diseases.

**Parasitism**

* Parasitism is the association of 2 organisms, 1 of which (the parasite) benefits by nourishing itself at the expense of the other (the host) but without normally destroying it.
* The following types of parasitic relations are recognized:

**1** (a) ectoparasites, which live on the host; and

* (b) endoparasites, which live within the body of the host;

**2** (a) accidental parasites, which are normally free- living animals but may live for a certain period in a host;

* (b) facultative parasites, which are able to exist free or as parasites, e.g. blowfly larvae; and
* (c) obligatory parasites, which are completely adapted to a parasitic type of life and must live in or on a host, e.g. most parasitic worms;

**3** (a) temporary or transitory parasites, which pass a definite phase or phases in their life-history as parasites and during which time the parasitism is obligatory and continuous, e.g. botflies, ticks;

* (b) permanent parasites, which always live for the greater part of their life as parasites, e.g. lice, tapeworms, coccidia, etc.; and
* (c) periodic, occasional, or intermittent parasites, which only visit the host for short periods to obtain food, e.g. blood-sucking flies, fleas;

4 (a) erratic parasites, which occur in an organ that is not their normal habitat, e.g. Fasciola hepatica in the lungs;

* (b) incidental parasites, which, exceptionally, occur in an animal that is not their normal host; they are incidental only in this first host, e.g. Dipylidium caninum is incidental in man; and
* (c) specific parasites, which occur in a particular species of host or group of hosts, e.g. *D. caninum* is specific for dogs and cats.

**UNIT.4**

**Basic principles of disease prevention, control and eradication of disease**

* Livestock disease management is made up of two key components:

- Prevention (biosecurity) measures in susceptible herds

- Control measures taken once infection occurs.

**Prevention Measures**

* Preventing diseases entering and spreading in livestock populations is the most efficient and cost-effective way of managing disease.
* While many approaches to management are disease specific, improved regulation of movements of livestock can provide broader protection.
* A standard disease prevention programme that can apply in all contexts does not exist.
* But there are some basic principles that should always be observed.

The following practices aid in disease prevention:

* Elaboration of an animal health programme
* Select a well-known, reliable source from which to purchase animals, one that can supply healthy stock, inherently vigorous and developed for a specific purpose.
* New animals should be monitored for disease before being introduced into the main flock.
* Good hygiene including clean water and feed supplies
* Protect the health of your animals and workers
* Reduce the risk of disease exposure to any members of the public who visit your farm
* this is particularly important if you hold open days or farm visits
* Precise vaccination schedule for each herd or flock.
* Observe animals frequently for signs of disease, and if a disease problem develops, obtain an early, reliable diagnosis and apply the best treatment, control, and eradication measures for that specific disease
* Dispose of all dead animals by burning, deep burying, or disposal pit.
* Maintain good records relative to flock or herd health. These should include vaccination history, disease prob­lems and medication.
* **Control and eradication of disease**

**Control of disease**

* 'Control' is the reduction of the morbidity and mortality from disease, and is a general term embracing all measures intended to interfere with the unrestrained occurrence of disease, whatever its cause.
* It is an ongoing process.
* Control can be achieved by treating diseased animals, which therefore reduces disease prevalence, and by preventing disease, which therefore reduces both incidence and prevalence
* **Eradication of disease**
  + Firstly, eradication mean the extinction of an infectious agent ; eradication has not been completed if a single infectious agent survives anywhere in nature.
  + Secondly, eradication has been defined as the reduction of infectious disease prevalence in a specified area to a level at which transmission does not occur
  + Thirdly, eradication has been defined as the reduction of infectious disease prevalence to a level at which the disease ceases to be a major health problem, although some transmission may still take place
  + Fourthly, and most commonly in veterinary medicine, eradication refers to the regional extinction of an infectious agent.
  + Strategies of control and eradication

1. **Quarantine** 
   * Quarantine is the isolation of animals that are either infected or suspected of being so, or of non-infected animals that are at risk.
   * Quarantine is an old method of disease control that is still very valuable.
   * It is used to isolate animals when they are imported from countries where exotic diseases are endemic.
   * It is also used to isolate animals suspected of being infected, until infection is either confirmed or discounted by clinical examination or laboratory testing
   * Similarly, when an infectious agent is not spreading within a herd or flock, quarantine may be adopted as part of a control campaign.

**2. Slaughter**

* + The productivity of animals usually is decreased when they are chronically diseased.
  + If a disease is infectious, affected animals can be a source of infection to others.
  + In such circumstances it may be economically and technically expedient to slaughter the affected animals.
  + Eradication of specific diseases from herds often involves a 'test-and-removal' strategy, in which all animals are tested, and only those testing positive are removed and slaughtered.

**3. Vaccination**

* Vaccines can confer immunity not only to many bacteria and viruses, but also to some helminths.

**4. Restriction of movement of hosts**

* + The movement of animals is often restricted during epidemics and eradication campaigns to reduce the risk of disease transmission
  + Restriction of international animal movements is important in ensuring that countries remain free from exotic diseases

**5. Mixed, alternate and sequential grazing**

* + The level of infection with some nematodes can be reduced by mixed, alternate and sequential grazing
  + The mixed grazing of susceptible animals with stock that are genetically or immunologically resistant to helminths reduces pasture contamination to an acceptable level. Thus, adult cattle (immune) can be grazed with calves (susceptible). Similarly, cattle can be grazed with sheep (susceptible).
  + The alternate grazing of a pasture with different species of livestock again reduces pasture contamination

**6. Control of biological vectors**

* + Infectious diseases transmitted by biological vectors can be controlled by removing the vectors. Insect vectors can be killed with insecticides.
  + The habitat of the vectors can be destroyed

**7. Control of mechanical vectors**

* + Living organisms that mechanically transmit infectious agents can be controlled by destruction and disinfection.
  + Biting fleas that transmit bacteria, for example, can be destroyed by insecticides.
  + People can also act as mechanical vectors; thus the veterinarian must impose a strict procedure for personal disinfection

**8. Therapeutic and prophylactic chemotherapy**

* + Antibiotics, anthelmintics, other drugs and hyperimmune serum are used (therapeutically) to treat diseases, and are administered (prophylactically) at times of high risk to prevent disease and thus to increase productivity.
  + Sometimes, chemotherapy can be used to eradicate disease

**THE END**