VETERINARY SYSTEMIC PATHOLOGY

DIGESTIVE SYSTEM

MOUTH AND PHARYNX

CLEFT LIP (Chelioschisis) AND CLEFT PALATE (Palatoschisis)

- Clefts of the mouth are midline fusion defects which occur during embryonic development.
- In cleft palate, the ingesta is likely to enter the respiratory passages and cause aspiration pneumonia and death.

CARIES

- Definition
  - Caries means decay of teeth in which the enamel is decalcified followed by softening and discoloration.
- Occurrence
  - Caries is rare in domestic animals.
  - This occurs occasionally in pet dogs with imbalanced and inadequate diets.
- Etiology
  - Disturbances in calcium and phosphorus metabolism as well as dietary deficiencies of these minerals.
  - It is frequently seen in fluorine poisoning.
- Pathogenesis
  - The organic acids especially lactic acid, that are formed due to the action of bacteria on carbohydrates dissolve the salts of the enamel.
  - Then, the same acids corrode into the less strong dentine, which contains in its structure 30% of protein.
  - The damage to the dentine is deeper and most widespread.
  - The opposed surfaces of adjacent teeth may be more frequently affected.
- Clinical signs
  - The affected teeth which are shaky and painful interfere with mastication.
- Gross pathology
  - The affected teeth usually have one or more depressed areas, which are brown or black in colour.
  - In the early incipient stages, “Enamel Flecks” which are yellow stained spots on the enamel can be seen.

STOMATITIS

Definition

- This is diffuse inflammation of the mucous membrane of the mouth.
- But when confined to particular parts of the mouth, it is known as:
  - Gingivitis for inflammation of the gums.
  - Glossitis for inflammation of the tongue.
  - Lampas for inflammation of the palate.
  - Cheilitis for inflammation of the lips.
  - Pharyngitis for inflammation of the pharynx and
  - Tonsillitis for inflammation of the tonsils.

Occurrence: Stomatitis is common in animals.

Aetiology

- It may be a primary affection or may occur as secondary to other diseases viz. gastritis or infectious diseases.
- The causes are
  - Physical

        - **Trauma** by awns, thorns, burrs, wood pieces, glass pieces, sharp bits, irregular sharp teeth, sharp edged feeding utensils.
        - **Thermal injuries**: Hot drenches and eating frozen foods.
    - Chemical : caustic alkalies, corrosive acids, fertilizers.
    - Deficiency of vitamins
        - Hypovitaminosis A especially in fowl
        - Niacin deficiency : Black tongue in dogs
    - Microorganisms
        - Bacteria: *Actinomyces bovis; Actinobacillus lignieresi; Fusobacterium necrophorum; Pseudomonas aeruginosa; Corynebacterium pyogenes; Streptococci* and*Staphylococci.*
        - Fungi: *Candida albicans* and *Oidium pullorum* in poultry.
        - Viruses: Foot and Mouth disease; Rinderpest: Virus diarrhoea -mucosal disease; Infectious canine hepatitis; contagious ecthyma; vesicular exanthema; fowl pox and blue tongue.

Gross pathology

- The lesion starts as catarrhal inflammation of the mouth and pharynx with reddening and swelling of the mucosa which is covered by small, whitish raised spots (Aphthous stomatitis).
- These spots may latter develop into small crusts or into ulcers.

Histopathology

- Microscopically, in case of ulcer of tongue, the surface epithelium of the tongue is lost and the underlying area is infiltrated with leucocytes.

TYPES OF STOMATITIS

Vesicular Stomatitis

- Vesicles, blebs or blisters containing clear fluid are formed on the mucosa.
- Seen in Foot and mouth disease, infectious vesicular exanthema and infectious vesicular Stomatitis.
- Rupture of the blisters results in the formation of erosions, which subsequently heal.
- Catarrhal and vesicular Stomatitis may develop into ulcerative variety.

Fibrinous and necrotic Stomatitis: This is seen in infection by *Fusobacterium nerophorum*.

Diphtheritic Stomatitis

- Fowl pox produces diphtheritic stomatitis and pharyngitis in which a grayish membrane is found.

Gangrenous Stomatitis: Very severe irritants may cause gangrenous Stomatitis.

Thrush

- Thrush is found in birds.
- Here grey or yellowish thick tenacious material gets attached to the mucous membranes.

Sequelae

- Starvation as prehension and mastication are prevented
- Spread of infection to other parts- oesophagus, lungs, stomach etc.

TUMORS OF MOUTH AND PHARYNX

- Tumors of the mouth and pharynx are common.
- Most common neoplasms in the dog and calves are the infectious papillomas, occurring as clusters on the lips and gums. These are caused by papilloma virus.
- Epulis is a fibroblastic tumour consisting of dense fibrous tissue with varying amounts of epithelium and a few giant cells. They usually occur in the gums
- Carcinoma, sarcoma, fibroma and melanoma are other tumors occasionally seen.

SALIVARY GLAND

FEATURES OF SALIVARY GLAND

- Pathological processes are rare in the salivary glands of animals because
  - The glands are in fairly well protected situations.
  - The salivary secretions have some anti-bacterial properties.
  - There is good flushing by the secretions

PATHOLOGICAL CONDITIONS IN SALIVARY GLAND

Foreign bodies

- Foreign bodies are occasionally found in the ducts especially of the parotid and sub maxillary glands.
- These are usually awns, wood pieces and kernels of grain causing inflammation.
- Sometimes, these may produce obstruction and consequent dilatation of the ducts.

Dilatation

- Dilatation of the salivary ducts may occur when the flow of saliva is obstructed by foreign bodies, inflammatory exudates etc.

Ranula

- When the dilatation of the salivary duct and gland occurs as cyst on the floor of the mouth it is called a ranula.
- This is smooth, rounded cyst containing a clear fluid.
- This can be easily ruptured.

Sialoliths

- Sialoliths are salivary calculi
- These are common in horses.
- These are formed by the precipitation of minerals around nuclei of foreign matter in the ducts.
- Salivary calculi are usually single and sometimes may be very large preventing the flow of saliva.
- These calculi produce stasis, distension of ducts and finally atrophy of the gland.

INFLAMMATION

Occurrence: Sialadentitis is very rare in animals.

Aetiology

- It may be due to traumatic injury or due to infection by bacteria.
- This may be associated with strangles in horses, mastitis in cattle and distemper in dogs.

Gross pathology

- The glands are swollen and red.
- Abscesses may be found in glands.
- Sometimes, cystic dilatations may occur.

Sequelae

- Stasis of saliva in the ducts facilitates infection.
- Inflammation of the salivary glands may cause atrophy of the gland.

NEOPLASM

- Neoplasms of salivary glands are not common in animals.

OESOPHAGUS

CHOKE

Definition : Choke is obstruction of the esophagus.

Occurrence: It occurs in horses and cattle, but more common in the former.

Aetiology

- Old age

- In cattle, large objects of food- beet root, carrot, apples, potatoes, fetal membranes, sticks and wire. In dogs, large bones.
- Impacted masses of feed due to improper chewing, bad teeth and rapid gulping of dry feed.
- Lesions of esophagus – stenosis or diverticulum cause repeated choking.
- Enlarged lymph nodes- mediastinal and cervical.
- Enlarged thyroids.
- Neoplasms of adjacent tissue especially thymus - thymoma in new-born animals.

Gross pathology

- In the horses choke occurs in the thoracic area while in cattle and dogs the pharynx is obstructed.
- Choke may be complete or incomplete.
- *Complete choke*
    - In complete choke, feed will be returned and water will flow through the nostrils when animal is watered.
    - Aspiration of the feed will cause secondary foreign-body pneumonia.
    - In cattle, complete obstruction will cause dangerous tympany.
    - Because of pressure, ischemia and resultant necrosis and gangrene may develop.
    - Infection may spread to the surrounding tissues- cellulitis or to the lungs- gangrenous pneumonia.
    - Resultant sapremia or toxaemia is the cause of death in fatal case.
- *Partial choke*
    - Partial obstruction will give rise to dilatation of esophagus above the obstruction - the esophageal diverticulum .

Sequelae

- Death due to gangrenous pneumonia, bloat, cellulitis or asphyxiation.
- Esophageal diverticulum
- Rupture of esophagus

DILATATION (ECTASIA)

Types: The dilatation of oesophagus may be fusiform or cylindrical.

Occurrence: Fusiform dilatation of the oesophagus is more common.

Aetiology

- Accumulation of food proximal to a stenosed area.
- In ruminants accumulation of food during regurgitation on the distal side of stenosed area.
- Trauma from horns etc., rupturing the muscular coat.
- Relaxation of the esophageal muscles consequent to the nervous lesions.

Sequelae

- The food gets accumulated in the dilated area of esophagus leading to pressure and rupture.
- The food may become decomposed and produce softening of the epithelium, inflammation, ulceration, gangrene and death.
- In ruminants, bloat occurs.

OESOPHAGITIS

Definition: This is inflammation of the oesophageal mucosa.

Occurrence: It is rare in animals because of the thick and resistant condition of the mucosa.

Aetiology

- Trauma by probing, stomach tube or foreign bodies.
- Chemicals – corrosives.

- In the fowl, thallium sulphate poisoning. Avitaminosis A in Fowls.
- Viral enteritis and mucosal disease in cattle.
- Parasites – bot fly larvae in horses and hypoderma larvae in cattle.
- Persistent vomiting in dogs and pigs.

Gross pathology

- The mucosa is red and swollen.
- In the catarrhal variety, the exudate is mucous.
- The ulcerative variety is met with in conditions caused by trauma (Stomach tube) and in viral enteritis and mucosal disease in cattle.

Sequelae

- Usually recovery occurs.
- Stenosis may result in severe cases.
- When pyogenic bacteria enter the place of obstruction (in choke) which had become necrotic due to pressure, suppurative oesophagitis occurs.

NEOPLASM

- Neoplasms of the oesophagus are not common.
- The connective tissue tumours of the dog have been reported. In the thoracic portion of the oesophagus, fibrosarcomas and osteogenic sarcomas are found in connection with*Spirocerca lupi* infection.
- The osteogenic sarcoma is evidently a metaplastic manifestation of the fibrosarcoma.
- Metastases of these tumors are sometimes found in the lungs and other tissues.
- Carcinoma in cat and horse and papilloma in cattle are other tumours met with.

CROP

OBSTRUCTION

Aetiology

- Ingestion of large quantities of dry grain which swell in the crop and form a hard mass.
- Atony or paralysis of wall leading to stasis of food.
- Foreign bodies like wire etc.

Gross pathology: stagnated food gets decomposed, gas accumulates and inflammation sets in.

Sequelae

- Rupture due to distention by food and gas or due to penetration by the foreign body.
- Death because of
    - Heart failure due to pressure on heart.
    - Asphyxia due to compression of trachea.
    - Intoxication due to absorption of toxins from decomposed foods.
    - Starvation, since, food does not enter the proventriculus.

INGLUVITIS

Definition: Inflammation of crop is called ingluvitis.

Acute catarrhal ingluvitis

- *Aetiology*
    - Trauma by foreign bodies.
    - Chemical agents : phosphorus, fertilizers
    - Toxins from decomposed food.
    - Infectious diseases.
    - Parasites – *Acuaria sp ; Capillaria sp*
- *Gross pathology*
    - Lesions include cogestion, edema and tympanites.

Diphtheritic ingluvitis: This is found in fowl pox.

TYMPANITES OR BLOAT

Physiological considerations

- Normally animals get rid of gases produced in the rumen by eructation.
- Saliva which has important antifoaming properties plays a significant part in the prevention of bloat. Mucin in saliva prevents formation of froth.

Aetiology

- Bloat (or accumulation of gas) can occur
    - When the gas is produced at too rapid a rate than can be eructated.
    - When the eructation mechanism is faulty.

Types

- Based on course, bloat may be acute or chronic.
- Based on nature of gas, bloat may be dry or frothy.

Acute bloat

- This may be due to complete choke in esophagus.
- It may also be due to sudden changes of feed.
- Excessive feeding on legumes that are wet with dew or rain.

Chronic bloat

- The chronic variety occurs whenever there is any hindrance to eructation in the esophagus either within or without pressure by tumors, foreign bodies, enlarged lymph nodes, abscesses, constrictions or diverticula.
- It may occur in lesions of the rumen causing decreased contractions of the ruminal wall as in atony, serosal adhesions, paresis, diffuse lymphomatosis.

Dry bloat

- The dry bloat is less harmful, since in this condition, the gases can be more easily got rid of by eructation.

Frothy bloat

- In the frothy bloat, the gas is trapped as small bubbles in the fluid forming a foamy mass which is not easily eructated.

Aetiology

- The following are supposed to produce frothy bloat
    - Saponin found in plants is a good saponifying agent.
    - Water-soluble proteins of legumes are capable of forming froth. This is probably of greater importance, since, bloat is observed even in animals fed with low- saponin plants.
    - Factors which increase the viscosity and lower the surface tension of ruminal contents produce froth.
    - Normally, in rumen due to bacterial activity, fatty acids are produced which increase the surface tension. If the production of these fatty acids is decreased, the surface tension will be lowered favouring froth production. This is the theory behind the use of vegetable oils in the treatment of bloat.

Pathogenesis

- Some legumes contain HCN, which is toxic, causing paralysis of the ruminal or reticular musculature and so inhibits eructation.
- Some legumes contain phosphatase which with arsenates accelerates fermentation producing a large quantity of CO_2.
- H_2S, CO_2 and CO produced in large quantities causes paralysis of ruminal muscles.
- If fed excessively on green plants only, which do not contain sufficient stiff fibres, the mucosa of the rumen is not adequately scratched to elicit the reflex contraction of the musculature.

- Mechanical stimulation of cardia, especially by roughages, increases the rate of secretion of saliva. But with ingestion of young succulent legumes, too little saliva may be secreted and so foaming is not counteracted and bloat results.
- Ruminal mucinolytic bacteria may destroy salivary mucin thereby producing frothy bloat. Polysaccharides produced by capsulated ruminal bacteria may be another etiological factor in bloat.
- Interference with the nerve pathways that are responsible for the eructation reflex may also lead to tympany. The receptors for this reflex are in the reticulum and the afferent and efficient nerve fibres are in the vagus nerve. Any lesions in this nerve may, therefore, lead to bloat.
- Distended rumen compresses other abdominal organs and causes passive congestion since the pressure on thin-walled veins impedes circulation.
- Along with this, there is forward thrust on the diaphragm, pressing on the lungs, which become smaller and sometimes atelectatic.
- The result of this is hypoxia and ultimate asphyxia and death.

Gross pathology

- In animal that dies of bloat, besides congestion of the abdominal viscera, one may notice hemorrhages on the pleura, pericardium, tracheal mucosa and on bronchial lymph nodes as well as in the lymph nodes of head and neck.
- Blood is tarry, as in Anthrax.
- Liver is pale.
- The rumen or diaphragm may be ruptured sometimes.
- Within few hours of death, the ruminal epithelium peels off.

Sequelae: If quickly relieved, acute bloat can be cured. If not, death may supervene due to asphyxia.

Prevention: Hay or straw must be fed liberally when animals are fed with succulent legumes.

IMPACTION OF THE RUMEN AND RETICULUM

Introduction

- Here the rumen stops functioning, the musculature does not contract and so the food ingested stagnates.

Occurrence: This is a common condition in cattle.

Predisposing causes: Lack of exercise and debility predispose the animal to atony of rumen.

Aetiology

- Overfeeding with large amounts of highly fermentable carbohydrate feeds.
- Tight packing of the rumen leaves no room for bacterial growth and normal ruminal fermentation and digestion. This leads to weak contractions of the ruminal and reticular walls and so the food does not get propelled.
- Lack of water
- Defective mastication and salivation due to defects in teeth or lesions of the tongue.
- Penetration of the wall of the rumen or reticulum by sharp objects like wire, nail etc.,
- Paresis of rumen which may occur due to injury to vagus by pressure from abscesses, tumors, tubercular nodules, swollen lymph glands and ruminal displacements.

Pathogenesis

- The pathogenesis of atony and impaction of the rumen after ingestion of large quantities of carbohydrate rich feeds is as follows
    - The carbohydrates are fermented by gram positive organisms, notably *Streptococcus bovis*, with the formation of lactic acid, resulting in to **lowering of pH of the ruminal contents** to as low as 4 to 4.5 from a normal 5.5 to 7.5.

- Due to the production of lactic acid the **osmotic pressure of ruminal contents increases** and so fluid is drawn into the rumen from the blood leading to hemoconcentration, anuria, dehydration and circulatory collapse.
- As the pH of the ruminal constituents falls, the **motility of the rumen decreases** and there may even be complete **stasis**.
- In such an atonic rumen, in which the normal microflora are lost, *Fusiformis necrophorus* and fungi of the family *Mucoraceae* (those belonging to the genera *Mucor*,*Rhizopus* and Absidia) invade the ruminal wall producing ruminitis and ulcers.
- At the lowered pH, normal microfloras of the rumen are destroyed, the lactobacilli and streptococci thrive and the salivary secretion ceases so that **buffering action of the saliva is absent**.
- Absorption of the lactate causes **acidosis**.

Clinical signs

- The stagnated food becomes putrified with the liberation of the foul smelling gases.
- In some animals, diarrhoea may be present if the putrid ingesta finds its way into the intestines causing enteritis.
- Anorexia develops and regurgitation stops.

Gross pathology

- At necropsy, the rumen will be found to contain hard, caked, undigested food with evil smelling odor.
- In animals that die of acute atony, the contents of the rumen and reticulum are thin, porridge-like and bulky.
- The cornified epithelium is soft and peels off easily, exposing hemorrhagic areas underneath.
- The blood is dark and thick.
- Lungs show bleeding into the alveoli and bronchi.
- Heart musculature is flabby.

Histopathology

- In animals that survive for three days and more, demyelination of the nervous system may occur.

Sequelae

- In mild cases, if the primary cause is removed, normal state may be regained.
- In severe cases, toxemia will cause death.
- Enteritis, peritonitis and ketosis.

TRAUMATIC RETICULITIS

Occurrence: This is a very common condition in older cattle.

Pathogenesis

- These animals ingest and swallow, along with their feed, a wide variety of sharp objects like needles, nails, pieces of fencing wire and screws.
- Sometimes contraction of the rumen and reticulum during pregnancy cause the sharp object to pierce the wall of the reticulum.
- Usually, it pierces the antero-ventral wall, which is near the diaphragm.
- The passage through it is usually slow and so the track formed by the moving nail is thickened by a dense fibrous wall.
- Piercing the diaphragm, the foreign body may enter the pericardium and even the heart, producing inflammation enroute.
- Traumatic pericarditis with serofibrinous or purulent exudates may occur.
- Sometimes, it may take a downward slope and pierce the chest wall near the xyphoid cartilage forming an abscess there.

- At the point where the object pierces the reticulum, a localized peritonitis develops causing adhesions between the reticulum and the diaphragm at this place.
- The sharp object may, sometimes, penetrate the lungs or the liver or the spleen causing abscesses in these organs.
- Penetration of vagus nerve may result into vagal indigestion.

Gross pathology

- The thick-walled blackened track followed by the foreign body and adhesions of the reticulum to the diaphragm may be clearly seen.
- Along the track, fistulae connecting one abscess to the other may be found.
- Fibrinous pericarditis with hypertrophied myocardium may be seen if foreign body has entered the pericardium.

Sequelae

- If the sharp object is not contaminated, only mechanical injury to the affected parts is seen.
- Vagus indigestion, when the ventral branch of the vagus is affected by the inflammatory and scar tissue formed by the penetrating foreign body.
- Diaphragmatic hernia may occur due to weakening of the diaphragm by lesions produced by the foreign body .
- Abscesses of lung, liver and spleen
- Fatal because if the sharp object penetrates any larger artery.

ULCERS OF FORE STOMACH

Occurrence: Ulcers of the fore stomachs may be seen in cattle occasionally.

Aetiology

- Usually due to *Fusobacterium necrophorum* infection.
- These ulcers may be found in animals that are started on heavy grain feed and also in calves kept on milk.
- Chemical ulceration due to corrosives may occur, though rare.
- Ulcers are seen in bovine viral diarrhoea (BVD).

Sequelae: Hepatic abscesses are considered to be complications of ruminal ulcers.

PARASITES IN THE RUMEN

- Rumen of adult ruminants may contain conical flukes which may be free in the contents or attached to the rumen mucosa.

POSTMORTEM CHANGES: Postmortem changes in the rumen include bloating with gas, sloughing of the mucosa and blackish discolouration.

STOMACH

MALPOSITIONS

Diaphragmatic hernia of stomach

- It may be met with in dogs and cats due to automobile accidents.
- The diaphragm is ruptured and the stomach enters the thoracic cavity.

Abomasal displacement

- Introduction
  - Abomasum may be displaced either to the left or to the right.
  - The left-sided displacement is more common in which it lies between the rumen and left abdominal wall.
- Occurrence
  - This condition is met with more frequently after parturition.
- Predisposing causes
  - Atony of the abomasum due to feeding large quantities of concentrates.

    - Post- parturient diseases like milk fever, mastitis, metritis and ketosis may also cause atony of the abomasum.
- Aetiology
    - Acomplication of surgical treatment for chronic indigestion.
    - Violent activity like jumping in estrus may be a cause in non-parturient cases.
    - A hereditary predisposition may exist.
- Pathogenesis
    - During pregnancy, the rumen may be lifted by the expanding gravid uterus and the abomasum may slip under the rumen.
    - After parturition, when the uterus recedes, the rumen drops to its normal position displacing the abomasum.
    - The greater curvature of the body of the abomasum which is more mobile slips under the ventral ruminal sac.
    - Atony of the abomasum due to feeding of concentrates after parturition prevents its correction.
    - In cases treated surgically for chronic indigestion, the incisions made are the weak spots where the abomasum may slip through.
- Clinical signs
    - Vague and are like those of chronic indigestion
    - Due to pressure, normal function of the abomasums is interfered with. Because of the abnormal position the function of the esophageal groove may be affected.
    - Anorexia alternating with voracious appetite
    - Ruminal tympany
    - Abdominal pain
    - Dullness
    - Listlessness
    - Rapid loss of weight
    - Tucked-up-appearance.
    - The dung is scanty but soft (not caked as in chronic acetonemia).
    - Frequent abnormal tinkling abomasal sounds may be heard at the level of paralumbar fossa.
- Clinical pathology
    - Mild ketonuria is present.

Diagnosis : Laparotomy may be needed for diagnosis.

Differential diagnosis

- Displacement of abomasums must be differentiated from
    - Chronic acetonemia
    - Traumatic reticulitis
    - Vagal indigestion
    - Diaphragmatic hernia
    - Pyelonephritis
    - Lymphomatosis.

Torsion of the stomach

- Occurrence : Torsion of the stomach may sometimes be seen in old dogs.
- Aetiology
    - This is due to sudden movements (jumping, rolling etc) especially when the stomach is full.
- Pathogenesis
    - With the esophagus as the pivot, the heavy stomach rotates clockwise.

    - As the blood vessels are compressed, there may be congestion and hemorrhage.
    - The twist closes both the openings of stomach and so gastric tympany develops with resultant dyspnoea.
    - In some cases the stomach may rupture.
- Gross pathology
    - There is twisting of the stomach around the esophagus.
    - The contents of the stomach are blood stained.

Rupture of stomach

- Occurrence : This is common in horses
- Aetiology
    - Usually due to tympanites and dilatation.
    - Trauma
    - Violent gastric contractions

Acute dilatation of the stomach

Aetiology

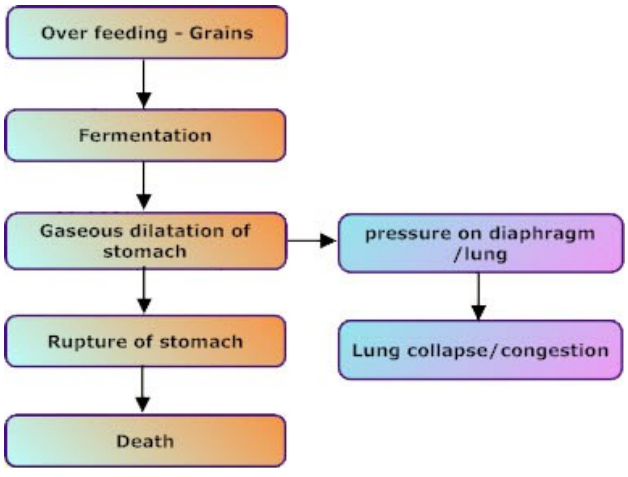
- It is due to excessive amounts of food or gas accumulating in the stomach.
    - Overeating; especially with grain in horse.
    - Excessive fermentation when easily fermentable foods are eaten.

Accessory causes

- Incomplete mastication due to poor teeth
- Hard work immediately after feeding
- Diseases of the stomach wall
- Atony of the gastric musculature
- Reflex closure of the pylorus
- Obstruction of the pylorus (by rags in calves)
- Compression of the pylorus by lipoma in horses and lymphocytoma in cattle.

Pathogenesis

- The accumulated feed and gas in the stomach cause such a stretching of the gastric wall that contractions are interfered with.
- The stomach which is dilated presses upon other organs
- Pressure on diaphragm and lung results into dyspnoea and congestion.
- Sometimes the diaphragm may rupture.
- Obstruction of the pylorus due to a tumor or cicatricial constriction and wind sucking may cause recurrent dilatation with resultant hypertrophy of the muscular wall of the stomach.



Clinical signs

- Stretching of stomach wall causes severe pain.

- Dilatation of the stomach causes vomition.

Gross pathology
- The stomach is dilated 4 to 5 times its normal size
- Lung is congested

Sequelae
- Fatal dehydration and alkalosis.
- Rupture of stomach.

GASTRITIS

Definition : Inflammation of the stomach is called gastritis.

Occurrence : Gastritis is a fairly common condition in animals.

Predisposing causes
- Close confinement and unsanitary conditions where bacteria thrive contaminating feeds and feeding utensils

Aetiology
- Causes may be the same for both acute and chronic gastritis but of different severity and acting for different lengths of time.

Physical
- Faulty dentition prevents mastication
- Foreign bodies may traumatize the gastric mucosa.
- Feeding very coarse material (eating bedding in horses and dogs)
- Feeding with frozen foods
- Overfeeding causing dilatation of stomach

Feeding
- Spoiled, mouldy and fermented hay and silage or feeding easily fermentable foods.
- When heavily fatigued animals are fed, the feed is not easily digested, stagnates, ferments and so produces irritation.
- Too sudden changes of feed
- Toxic plants

Chemicals
- Uremia; (Excretion gastritis is caused by the excretion of the toxic substances through gastric glands)
- Caustic and corrosive chemicals like mercury, lead, copper, arsenic and phosphorus

Stress
- In stress, adrenaline is produced in large quantities which is responsible for increased gastric secretion and gastritis.
- This is seen in nervous dogs and in calves separated from their mothers.

Bacterial
- In calves - enterotoxemia and colibacillosis; in pigs - erysipelas, vibrionic dysentery, salmonellosis and colibacillosis.

Viruses
- Pig- hog cholera; transmissible gastro-enteritis in baby pigs
- Cattle - rinderpest, mucosal disease.
- In chicken - Ranikhet disease causes haemorrhagic proventriculitis.

Fungi : Mucormycosis, moniliasis and aspergillosis cause gastritis in many animals.

Parasites
- Stomach worms - *Trichostrongylus sp., Hemonchus sp., Ostertagia sp.*, larval paramphistomes in ruminants.
- Larvae of *Habronema sp.* and *Gastrophilus equi* in horses.
- In pigs *Hyostrongylus rubidus*, *Ascarops strongylina* and *Physocephalus sexalatus.*

TYPES OF GASTRITIS

- It may be primary or may be secondary to some other infections, as canine distemper, viral diarrhoea, swine erysipelas.
- Gastritis may be acute or chronic.

Acute gastritis

- Acute gastritis may be catarrhal, fibrinous, suppurative, haemorrhagic or necrotic, depending upon the cause and their severity.
- By far the most common is the catarrhal and to a lesser extent, the hemorrhagic.

Pathogenesis

- In gastritis, food does not get digested
- Motility of the gastric wall is retarded
- Irritation may produce pain and vomiting.

Catarrhal gastritis

- *Gross pathology*
  - The gastric mucosa is covered with mucus.
  - The mucosa in some places may show ulceration.
  - The mucosa is thick and red.
- *Histopathology*
  - The mucosa shows catarrhal exudation, hyperemia and leuococytic infiltration.
  - Some of the gastric glands may be damaged and lost.

Acute hemorrhagic gastritis

- *Occurrence* : This is a common condition.
- *Aetiology*
  - caustic chemical poisoning
  - uremia
  - acute infectious diseases like pasteurellosis, braxy, leptospirosis (in dogs).
- *Gross pathology*
  - Due to haemorrhage, the mucosa is bright red in color and the gastric contents are blood stained.
  - Digested blood (acid haematin) imparts a brownish coloration to the contents.

Parasitic Gastritis

- *Occurrence* : This is very common in animals.
- *Aetiology*
  - *Cattle and sheep*: *Hemonchus contortus, Ostertagia ostertagi, Trichostrongylus axei*
  - *Horses: Habronema* larvae, *Trichostrongylus axei* and *Gastrophilus equi* larvae.
  - *Pig: Hyostrongylus rubidus, Physocephalus sexalatus. Simondsia paradoxa, Ascarops strongylina* .
  - *Cats: Gnathostoma spinigerum*
- *Pathogenesis*
  - The strongyles are blood suckers and they produce minute injuries on the mucosa.
  - The larvae may burrow into the mucosa for completion of their life cycle and thereby cause damage to the epithelium and glands.
  - Heavy infestation besides causing anemia will produce catarrhal gastritis.
- *Gross pathology*
  - *Gastrophilus sp.* in the stomach may produce ulcers
  - *Habronema* larvae live in granulomatous nodules which may be infected by secondary bacteria and form abscesses.

Chronic gastritis

- *Aetiology*
  - Usually the same causes as for the acute but operating for a longer time.
  - Sometimes it may be secondary to chronic gastric dilatation and cirrhosis.
- *Pathogenesis*
  - Ischaemia in gastric dilatation and passive hyperemia and failure of detoxication in cirhosis decrease the local resistance thereby facilitating infection.
- *Gross pathology*
  - The mucous membrane is thickened and covered with tenacious, viscid glassy mucus.
  - This condition is usually of a hypertrophic type with thickening of the gastric wall.
- *Histopathology*
  - There is exfoliation of the epithelium
  - Hyperplasia of gastric glands and muscle fibres along with cellular infiltration and hyperplasia of basal lymphocytic nodules.
  - The mucosa may be thrown into polypoid folds (*polypoid gastritis)*.
  - The interstitial connective tissue hyperplasia exaggerates the mucosal foldings.
  - Occlusion of glands results into development of retention cysts.

GASTRIC ULCERS

Introduction

- Usually gastric ulcers run an acute course, heal promptly and seldom become chronic as in man.

Occurrence

- Gastric ulcers are common among animals.
- Small superficial defects are known as *erosions* which are common among ruminants.
- Calves are more often affected.

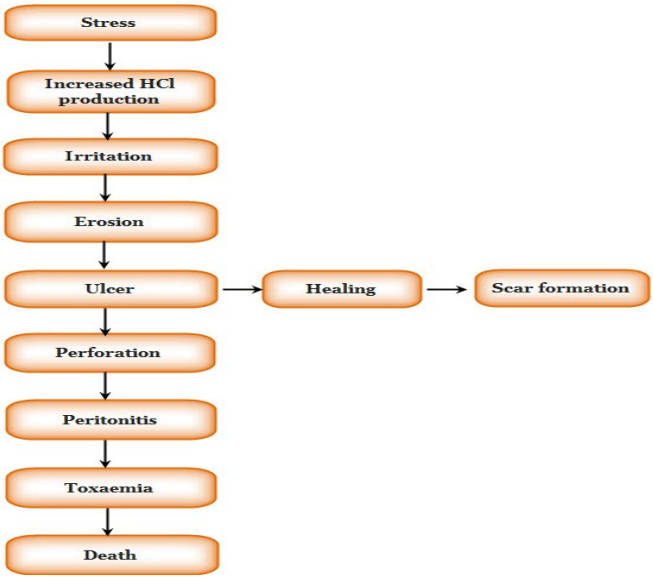
Aetiology

- *Trauma*
  - This is the commonest cause of abomasal ulcers in calves.
  - In early weaning of calves (before fourth week of age), when they are put on roughages before the full functional development of forestomachs and start of rumination, the coarse plants irritate the tender gastric mucosa producing focal gastritis and ulcer.
- *Obstruction of pylorus in cattle*
  - In calves - by foreign bodies or coarse feed.
  - In adult cattle - traumatic reticulitis and abomasal displacement.
- *Chemicals*
  - May act either directly on the mucosa or indirectly through their action on the nerves.
  - Grazing on pastures heavily fertilized with nitrogen.
- *Nutrition*: Nutritional hepatic dystrophy (due to deficiency of vitamin E) in pigs.
- *Infections*
  - Erosions are common in the abomasum of cattle in rinderpest, mucosal disease, bovine malignant catarrh and pox.
- *Fungi*: *Mucor sp* and *Monilia sp* cause gastric ulcers in pigs.
- *Circulatory disturbance*

- In intense passive hyperemia with focal hemorrhages lead to epithelial nercrosis.
- Such vascular stasis and hemorrhages are found in - foot and mouth disease, canine distemper, rinderpest, rabies, purpura hemorrhagica and uremia.

- *Nervous effects*
  - The sympathetic and the vagus nerves control the secretion and blood circulation of abomasum.
  - Any factors that disturb these nerves may produce stress, hemorrhages and increased secretion by gastric glands.
  - Stress in pregnancy and abomasal displacement may cause such disturbances and so abomasal ulcers are common in post-parturient dairy cows.
  - Vasoconstriction produced by adrenaline during stress is another factor.
  - Vagus indigestion causes atony of abomasum and ulcers.
- *Parasites*: In horses, larvae of *Gastrophilus* species and *Habronema megastoma*.
- *Neoplasms of the stomach* : Lymphocytoma of stomach wall is accompanied by ulceration.

Pathogenesis



Gross pathology

- The mucosal erosions (in cattle) are of the size of a millet.
- Slowly, by the action of the gastric juice the erosions may enlarge and become deeper to form ulcers.
- The ulcers are usually demarcated, having raised borders with a punched-out appearance. The base of the ulcers may be the submucosa or muscular coat or in some cases even the serosa. When the serous coat is the base, perforations are likely to occur.

Histopathology

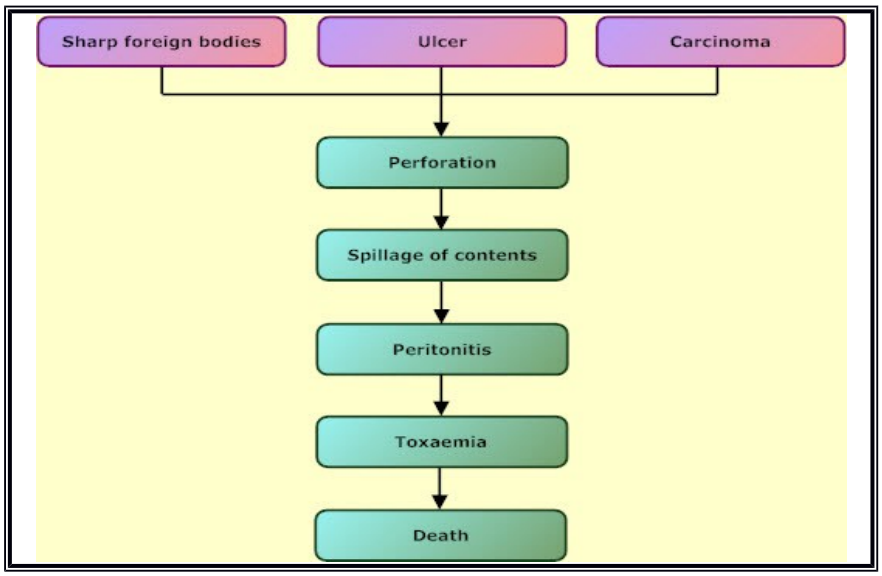
- The erosion is covered by an exudate consisting of mucus, fibrin and inflammatory cells.
- The epithelium is exfoliated.
- The sub-mucosa may be infiltrated by large number of leucocytes.

Sequelae / Fate of the ulcers

- Ulcers develop very rapidly. The abomasal ulcers develop in three to four days.
- Most of the gastric ulcers heal by the formation of granulation tissue. Destroyed glands do not regenerate. Healed gastric ulcers have a star-like appearance.

- Sometimes blood vessels may be eroded and fatal hemorrhages may result. But, this is not so common in animals as in man.
- Gastric ulcers of animals rarely perforate. Perforation, when it occurs, may happen in six to seven days.
- Hemorrhages and perforation resulting into fatal peritonitis are the occasional complications.
- Lactation stress may be one of the causes of perforation of abomasal ulcers in cattle.
- Progressive anaemia and emaciation may result in some cows.

Perforation of stomach wall



Neoplasms of the stomach wall

- Most common is lymphosarcoma of the abomasum in the bovines.
- Other tumors seen are leiomyoma and adenocarcinoma.

INTESTINE

CONGENITAL ANAMOLIES

Atresia of the intestine: It may be found as a hereditary defect among cattle.
Atresia of rectum: In pigs and foals, atresia of rectum is an inherited lethal characteristic.
Imperforate anus: In cattle, imperforate anus at birth is a semi-lethal character.
Persistent omphalomesenteric duct (Meckel's diverticulum)

- In pigs and horses, it is probably a hereditary defect.
- It may become obstructed or inflammed or even ruptured resulting in peritonitis.

Stenosis of the intestines sometimes occurs congenitally.

PM CHANGES

- Postmortem changes in intestine include ballooning of intestine, thickening of intestinal mucus, blackening of intestinal mucosa (pseudomelanosis coli) and sloughing of intestinal mucosa.

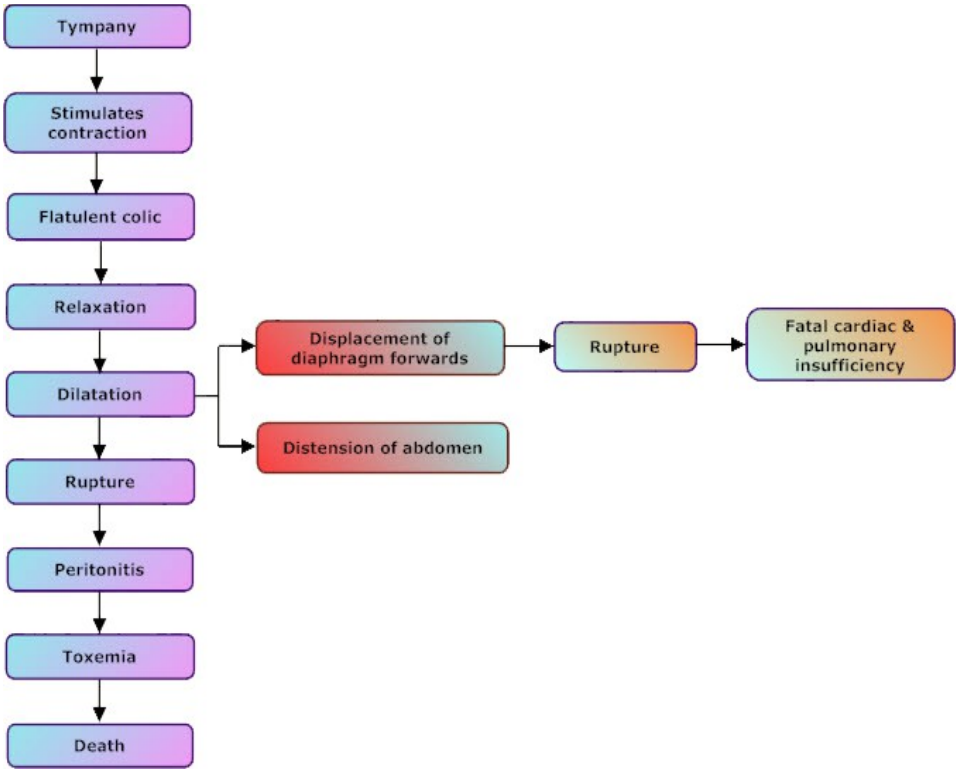
INTESTINAL TYMPANY

Definition: Distension of intestine with gas
Occurrence: Very frequent in horses, rabbits and dogs.
Etiology

- *Acute*
  - As for gastric tympany
  - Intestinal obstruction
  - Atresia of intestine

- *Chronic*
    - Accumulation of contents in certain parts of intestine
    - Dry, coarse insufficiently masticated feed
    - Decreased secretion
    - Intestinal adhesion / stenosis
    - Regional ileus
    - Enlarged prostate gland
    - Anal adenitis

Pathogenesis



CIRCULATORY DISTURBANCES

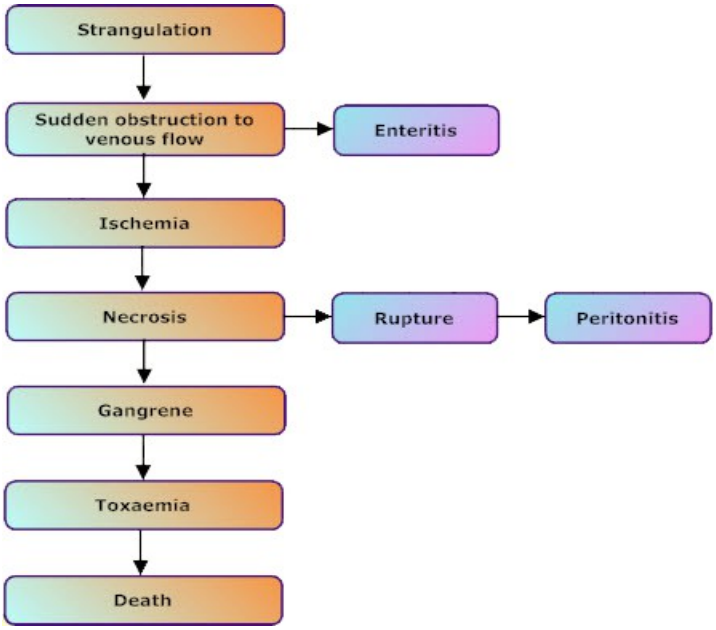
- Acute passive hyperemia
- Chronic passive congestion
- Hemorrhages
- Infarction
- Thrombosis
- Embolism

ACUTE PASSIVE HYPEREMIA

Occurrence

- Occurs in conditions where there is sudden obstruction to the venous out flow as in prolapse, torsion, hernia or intussusception.

Pathogenesis



Gross pathology

- There may be effusion into the peritoneal cavity.
- The involved part is dark-red and swollen due to haemorrhage and oedema.
- The intestinal wall is edematous with hemorrhages.

CHRONIC PASSIVE CONGESTION

Occurrence

- It is common in liver diseases or obstruction of the portal vein or may be part of the general venous congestion (with lesions in the heart and/or lungs).

Gross pathology

- The intestinal veins are dilated and stand out prominently.
- The intestinal wall is thickened and edematous
- Ascites is found

Sequelae

- The intestines become predisposed to enteritis.
- In long standing cases, there is fibrous thickening of the wall and atrophy of the glands.

HEMORRHAGES

Occurrence: Haemorrhages are common on the mucosa and serous surface of the intestines.

Etiology

- Specific infections in which this is a lesion.
- Enterotoxaemia (in sheep)
- Blood sucking parasites
- Hemorrhagic diseases

INFARCTION

- If circulation is obstructed either by embolus or by extramural pressure, infarction may result.
- Saprophytes normally resident in the bowel invade the infarcted part and produce gangrene

THROMBOSIS

Occurrence

- Thrombosis of the intestinal vessels is common in the horses and is mostly due to larvae of *Strongylus vulgaris*.

Pathogenesis

- The infective larvae burrow into the mucosa and travel along the intestinal arteries, crawling against blood flow along the intima and reach the anterior mesenteric artery where they settle.
- At that place the endothelium is damaged and a thrombus is formed.
- This thrombus may become organized and canalized.
- Complete organization of the thrombus is prevented by the penetration of larvae which produce irritation.
- The damage caused by the penetration of larvae as well as their continued presence weakens the wall of the artery leading to aneurysm. Replacement of the elastic tissue by fibrous tissue with lack of resilience of the arterial wall also contributes to aneurysm.
- The wall may sometimes rupture.
- Calcification of the fibrosed wall may sometimes further weaken the wall.
- Of various branches of the anterior mesenteric artery, it is in the right branch that thrombus more often occurs. This branch supplies the ventral colon and so thrombosis of the vessel produces ischemia of these parts.
- Thrombosis more often occurs in the right branch of anterior mesenteric artery, hence, ischaemia is more frequent in ventral colon.

Sequelae

- Ischemia of the parts results into atony of the intestinal musculature leading to decreased peristalsis, stagnation and impaction by the ingesta.
- Gas may be produced if fodder is succulent.

EMBOLI

- Thrombotic emboli may occlude some branches of the artery resulting in infarction, gangrene, shock and death.

MECHANICAL OBSTRUCTION

Aetiology

Congenital: Atresia and imperforation of intestine

Acquired

- *Stenosis*
  - Due to pathological lesions: hematoma, neoplasms, abscesses, chronic inflammatory scars, displacements like torsion, volvulus, intussuception and hernia.
- *Impaction*
  - Foreign bodies like bone, stones, cartilage, rubber ball, rags and golf balls
  - Hair balls in cats
  - Impacted undigested coarse food, especially in the horse
  - Dogs- coproliths
  - Impacted meconium in new born animals.
  - Parasites- masses of round worms in pigs and fowls and tape worms in sheep
  - Enteroliths
  - Neoplasms- lipoma in horses.

Accessory causes

- Sudden changes in feeds
- Faulty dentition

Pathogenesis

- Obstruction causes weak peristaltic movements of the bowel above the points of obstruction, resulting in dehydration of the contents at that place.
- Spasms with violent contractions of the gut above the place of obstruction causes intense pain (colic) and sometimes rupture may occur.

Clinical signs: Vomiting is usually associated with intestinal obstruction in dogs and cats.

Gross pathology

- The place of obstruction is usually distended.
- The contents are hard, which pressing on the mucosa may cause necrosis and erosion.
- Ultimately stenosis may develop at this part.

Sequelae: Rupture and peritonitis leading to death may result if the obstruction is not relieved.

TORSION

Definition: Torsion is a twisting of intestines on its axis.

Occurrence

- It is frequent in horses.
- This may also be seen in other animals

Etiology

- Torsion occurs more often in the small intestines which have a long mesenteric attachment.
- In the horse, the right colon is fixed by ligaments and so torsion occurs in the left and transverse colon.
- In the cattle torsion of cecum is more common.

Pathogenesis

- The changes that occur in torsion are acute passive congestion leading to edema, hemorrhage, gangrene, peritonitis and death.

VOLVULUS

Definition

- Volvulus is a twisting of the bowel on itself as occurs when it passes through a tear in the mesentery.

Occurrence

- This condition is more frequently seen in horses .
- May also be met with in other animals.

Etiology

- Violent movements as in rolling and struggling.
- Violent peristaltic movements.
- *Foreign bodies:* sand or enteroliths by their weight make the part heavy and aid in its winding around other parts.
- *Gas:* Accumulation of gas makes the part bulge and twist round other viscera.

Gross pathology

- The affected portion is swollen and darkened in color.
- The wall is very easily torn.
- Peritonitis may be evident in some cases.

INTUSSUSCEPTION

Definition

- Intussusception is telescoping of a portion of intestine into another, usually the anterior into the posterior.

Occurrence : It occurs mostly in the jejunum and cecum in dogs and cattle.

Pathogenesis

- Along with the portion of intestines, its mesentery is also dragged along and so there is compression of the thin-walled veins resulting in acute local passive hyperemia.

Gross pathology: The affected part is dark-red or bluish and swollen.

Sequelae

- Usually gangrene and peritonitis supervene terminating in death.
- In some stray cases, the invaginated portion may be sloughed off healing occurring by granulation tissue. Epithelium covers the scar. But at the site of scar circular stenosis may form.

INCARCERATION

Definition

- Incarceration of the intestine is trapping of the intestine internally from pressure on its external surface.

Aetiology

- Incarceration may occur due to
    - The loop of intestine may pass through the epiploic foramen of Winslow
    - Occasionally a persistent urachus may cause incarceration
    - When the bowel passes through a fissure of the mesentery, congenital or acquired, incarceration may supervene.
    - Adhesion of the intestine to other abdominal organs.
    - An adhesion to the uterus.

Pathogenesis

- The changes in this condition are due to stagnation of the intestinal contents followed by venous stasis.

Sequelae

- Incarcerated portion of intestine undergoes infarction, gangrene and leads to peritonitis, toxaemia and death.
- Sometimes rupture, shock and death may occur.

PROLAPSE OF RECTUM

Definition : This is protrusion of the rectum through the anus.

Etiology

- The causes are straining, irritation, abdominal pressure, diarrhoea, increased peristalsis and constipation.

Gross pathology

- The changes are due to venous compression and acute local passive hyperaemia.
- The rectum is hyperaemic and will be found hanging through the anus.
- It may be edematous and soon becomes gangrenous.

Sequelae

- If not attended to early, the prolapsed rectum will be pecked by birds or injured by other animals.
- Due to swelling, fecal matter cannot be voided.
- Antemortem prolapse can be distinguished from the post-mortem prolapse by the absence of congestion in the latter.

HERNIA

Definition

- Hernia of the abdominal organs is the protrusion of the abdominal viscera through a natural or artificial opening.

Occurrence : Hernia of intestines is commonly seen in domestic animals, especially the pig and horse.

Etiology

- The intestines may pass through a natural opening, the internal inguinal opening which is patent in the males.
- The umbilicus, if not healed is another site of hernia.
- Trauma when the abdominal muscles may rupture or even the diaphragm may tear resulting in the intestines passing through the opening.
- Violent straining during parturition or defecation

Types

- Depending upon the location, hernia may be *external* or *internal* (diaphragmatic, pelvic).
- If the hernial contents can be returned in to the abdominal cavity, it is called *reducible hernia* .
- If the hernial contents cannot be so returned it is called *irreducible hernia* .
- The causes of the latter are
    - i. Adhesions between the visceral mass and the hernial sac.(The adhesions arise due to inflammation of the peritoneum)
    - ii. Accumulation of ingesta in the loop of intestines making it too bulky to be reducible and
    - iii. Venous stasis, oedema and incarceration, whereby the volume is so increased and the bowel cannot be reduced.
- If the hernia does not have a parietal peritoneal covering of the viscus, it is called a *false hernia*. In such cases, opening of the skin will reveal the bowel. The condition is called *eventration.*

External hernia

- External hernia consists of
    - A hernial sac formed by the parietal peritoneum and the covering skin
    - A hernial ring which is the opening in the abdominal wall and
    - The hernial contents.

Types of External Hernia

Ventral hernia

- The ventral hernia occurs when the abdominal muscles are ruptured.
- This is common in horses (spontaneous in pregnant mares) and occasionally in cattle.
- Trauma - Horn injuries, kicks, automobile accidents, laparotomy and castration scars.
- In pregnant ewes, this may be due to muscular degeneration of nutritional origin.

Umbilical hernia

- *The umbilical when the bowel passes through a congenital or acquired defect of umbilicus*
- It is seen in foals, calves and pups.

Inguinal hernia

- In inguinal hernia, the bowel passes through the internal inguinal ring.
- This is not so common in animals as in man, because of horizontal position: seen in colts and pigs.

Scrotal hernia

- Here the intestines slide into the tunica vaginalis along the inguinal canal in contact with the spermatic cord.
- The testes may undergo thermal atrophy as it is in contact with the intestines.

Femoral hernia

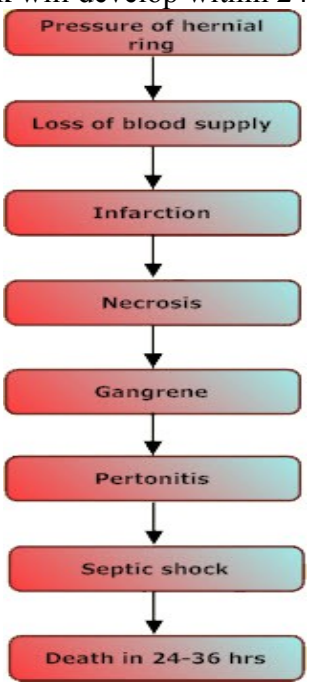
- Femoral hernia may develop when the omentum and intestines pass through the femoral triangle along the femoral artery.
- Here the bowel is found on the inner surface of the thigh.

Perineal hernia

- The perineal hernia may occur in old dogs.
- It may be due to violent straining as in case of enlarged prostate.

Strangulated hernia

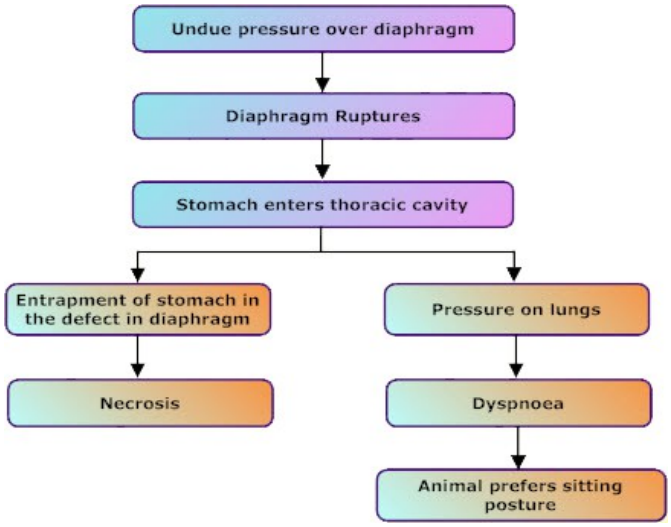
- This is one in which the blood supply is cut off by the pressure of the hernial ring through which the intestines pass.
- If not relieved in time, this condition is fatal since infarction, gangrene, peritonitis and shock will develop within 24 to 36 hours.



Internal hernia

Diaphragmatic hernia

- *Aetiology*
  - Physical trauma from external side
  - Increased intra-abdominal pressure
- *Pathogenesis*



ENTERITIS

Definition

- Enteritis is inflammation of the whole of the intestinal tract. But usually it is applied to the inflammation of the small intestines.
- The inflammation of the colon is called *colitis* , that of cecum *typhlitis, that* of rectum *proctitis* and of cloca *cloacitis* or *vent gleet*.

Introduction : Enteritis is of immense economic importance.

Occurrence

- Enteritis is very common in domesticated animals and fowls.
- Since enteritis occurs along with gastritis (the same irritants causing gastritis passing on to intestines produces enteritis also) gastro-enteritis is a frequent condition met with.

Etiology

- Causes are many and varied and they include chemicals, bacteria, viruses, protozoa, rickettsia, helminths, fungi, , disturbed metabolic processes as in ruminants, venous congestion as in portal hypertension and congestive cardiac failure, toxins of Clostridia, coliforms and spoiled or mouldy feeds and avitaminosis.

Gross pathology

- In enteritis, the whole length of the bowel may not be affected, inflammation localizing only at one part or the other.

TYPES OF ENTERITIS

Based on the nature of the exudate and the changes produced in the intestinal tract, enteritis is classified as follows.

- Catarrhal Enteritis
- Hemorrhagic Enteritis
- Fibrinous Enteritis
- Suppurative Enteritis
- Necrotic Enteritis

CATARRHAL ENTERITIS

Acute Catarrhal Enteritis

- This is the mildest of inflammation of the intestinal tract, occurring in a diffuse manner throughout the bowel.

Predisposing causes

- In calves and lambs avitaminosis A is a predisposing factor while in young pigs deficiency of animal proteins and trace elements predispose them to infections.
- In such a state, the organisms are able to gain a foothold and thrive causing the disease.

Aetiology

- Causes include mild irritants like foreign bodies, sand, coarse feeds, chemicals, bites of parasites (hook worms), *Vibrio coli* (causing winter diarrhoea in cattle) and drugs.
- Acute catarrhal enteritis may be noticed in
    - Enteritis in sucklings – scours in calves, lambs, foals and piglets caused by *E.coli, Pasteurella, Salmonella, Proteus, Vibrio* and *Streptococci*.
    - Enterotoxemia in sheep.
    - Viral Diarrhoea -Mucosal Disease in cattle.
    - Virus gastroenteritis in pigs.
    - Salmon poisoning in dogs.
    - Salmonellosis, infectious cloacitis, pullet disease and ornithosis in fowls.
    - Oral antibiotic therapy may cause enteritis in two ways
        - These may themselves be irritants or

        - They may so alter the intestinal flora that there is over growth of other bacteria (*Staphyloccocci, Proteus sp; Pseudomanas sp*) and fungi (*Candida albicans*) which are normally kept under restraint and so enteritis results.
    - Immature amphistomes

Gross pathology

- One should be able to distinguish this condition from the normal hyperemia that occurs during active digestion.
- The intestinal contents are watery
- The mucosa is reddish in color and slightly thickened, covered with a mucinous exudate.
- The Peyer's patches are prominent being hyperplastic, outlined by a zone of hyperemia.

Histopathology

- The intestinal contents consist of mucus, fibrin and exfoliated epithelial cells.
- Goblet cells are numerous and produce large amounts of mucin.
- The tips of villi may be hyperaemic and edematous.
- There is infiltration with leucocytes in the lamina propria and to a little extent in the submucosa.

Sequelae

- When cause is removed inflammation may subside and the bowel returns to normal.
- But if irritant persists, the condition may develop into the chronic state.

Chronic Catarrhal Enteritis

Aetiology

- It may develop from the acute condition or more usually it may arise gradually as in Johne's disease, intestinal helminthiasis, chronic venous congestion (due to congestive cardiac failure) and cirrhosis of liver.

Gross pathology

- The wall of the intestines is greatly thickened.
- The mucosa is smooth (covered by thick mucus)
- The corrugations are sometimes present at right angles to the length of the intestines (*Chronic polypoid enteritis*)

Histopathology

- The mucosa is thickened
- The mucosa is covered with thick mucus.
- The characteristic appearance is the presence of numerous macrophages, plasma cells, lymphocytes and connective tissue cells in the lamina propria and even in the sub-mucosa.
- The intestinal glands are atrophied due to the pressure of the infiltrating cells.
- Sometimes retention cysts due to closure of the mouths of glands are found (chronic cystic enteritis).

HEMORRHAGIC ENTERITIS

This is a more severe form of enteritis, characterized by the presence of erythrocytes in the exudate.

Aetiology

- This is mostly seen in septicemic bacterial and viral diseases e.g Anthrax and Rinderpest.
- Poisoning by arsenic and croton oil
- Colibacillosis
- Enterotoxemia

- Coccidiosis
- Continous feeding of dogs with horse meat causes an anaphylactic condition manifested by hemorrhagic enteritis.
- Vitamin B deficiency in dogs and pigs
- This condition may also be found in ancylostomiasis and uraemia of dogs.

Gross pathology

- Always patchy in distribution
- The intestinal contents are blood stained.
- Blood found in the anterior portion of the intestines is digested and so is brown in color while in the posterior portion it is bright red.

Histopathology

- Red blood cells may be found in the exudate of the mucosa.
- The villi may show necrotic changes.
- The intestinal wall shows haemorrhages.
- Thrombosis of some enteric vessels is evident.

Sequelae

- If treated in time, prognosis may be favourable.
- Being very severe, death usually occurs.

FIBRINOUS ENTERITIS

Introduction: This is of the diphtheritic type of enteritis.

Occurrence: It occurs in cattle, pigs and cats, and rarely in horse and fowl.

Etiology

- *Chemicals:* Salts of mercury and arsenic.
- *Bacteria:* Campylobacteriosis, *Salmonella cholerae suis* and *Escherichia coli*
- *Parasites: Echinostomum*, a fluke in turkeys.

Gross pathology

- The characteristic finding is the presence of strands of fibrin on the mucosa of intestine.
- The wall of the intestine is edematous.
- In more severe conditions, a thick, grayish or whitish-gray membrane may be covering the intestinal mucosa.
- The inflammation may extend into the submucosa and petechial hemorrhages may be seen.
- The mesenteric lymph nodes are swollen, haemorrhagic and juicy.

Histopathology

- The exudate consists of strands of fibrin containing in its meshes varying number of neutrophils and exfoliated epithelial cells.
- Mucous membrane shows edema, hyperemia and infiltration by neutrophils.
- Coagulative necrosis of the epithelium occurs in some places, which along with the exudate forms a membrane which is adherent to the intestine.

Sequelae

- Being a very severe condition, death is a common sequel.
- In those that are able to withstand, recovery with complete healing occurs.

SUPPURATIVE ENTERITIS

Occurrence : This is not a common condition in animals.

Aetiology

- It may result due to infection by pyogenic organisms (*Streptococci, Salmonella* and *Shigella*) infecting the wounds caused by helminths.

Gross pathology : Macroscopically, the exudate contains pus.

Histopathology: The exudate besides mucus contains exfoliated cells, neutrophils and bacteria.

NECROTIC ENTERITIS

Here necrosis of the intestinal epithelium and underlying tissues occurs.

Etiology

- Severe irritants: Chemical- croton oil, mustard gas, wood preservatives; insecticides.
- Bacteria- *Fusiformis necrophorum and Salmonella sp.*
- Viral diseases - Rinderpest, Viral Diarrhoea- Mucosal Disease, Hog cholera
- Protozoal diseases - Coccidiosis and histomoniasis.
- Vitamin deficiency – Niacin deficiency in swine.

Gross pathology

- Patchy necrotic areas are seen
- Fibrin may be found on the necrotic mucosa.
- When the necrotic material is removed, a red, raw, bleeding surface is seen.
- The necrosis of the mucosa extends into the sub-mucosa also.
- The mesenteric lymphatic nodes are swollen and juicy.
- In hog cholera, the characteristic lesion is the "button ulcer", which is a spherical ulcer in the mucosa of the colon. This is circumscribed with sharp edges.

Histopathology

- Necrosis of enterocytes is seen.
- The mucosa shows hyperemia and cellular infiltration.
- The ulcer reveals a demarcated zone of necrosis in the mucosa and sub-mucosa.
- The button ulcers are tiny areas of infarction that arise by the occlusion of small arteries by the swollen and proliferated endothelium.

Sequelae

- The condition is mostly fatal.
- If the condition is one of niacin deficiency, restitution of the deficiency may cure the condition.

CONCRETIONS

Enteroliths

Definition: Calculi found in the intestine are called enteroliths.

Occurrence: Sometimes they are found in the large intestines of horses.

Physiological considerations

- Normally magnesium phosphate is dissolved by the gastric juice and then absorbed in the intestines.

Predisposing causes

- Disturbance in the colloid protection of dissolved salts
- Change in bacterial flora with altered fermentation conditions
- Sluggish intestinal movements that occur in the dilatation of the bowel
- Relaxation of intestinal muscle met with feeding of bran.

Aetiology

- When animals are fed on wheat or bran which are rich in magnesium phosphate, intestinal calculi can occur.

Genesis

- They are formed of triple phosphates which are deposited concentrically, layer after layer, over a nucleus/nidus of sand or a metal piece or an undigested vegetable fibre.
- When excessive amounts of magnesium phosphate are fed to an animal, and that too to one suffering from chronic catarrhal gastritis in which gastric juice is not secreted, much of the magnesium phosphate reaches the intestines in an undissolved state.

- This combines with ammonia that is formed from the decomposition of protein (which is also abundant in wheat and bran) to form triple phosphate.
- This triple phosphate crystalises around foreign bodies like a grain of sand, a piece of metal or undigested plant fibre.

Enteroliths do not form in the small intestines because

- The movement of the food is too rapid there to allow the deposition of salts and formation of calculi and
- Bacterial decomposition of proteins to form ammonia does not take place there.

Gross pathology

- Enteroliths may sometimes attain a large size, some may weigh as much as 20 lbs and are usually round and smooth.

PHYTOBEZOARS

Definition: These are food balls (phyto= plant, bezoar=concretion) made of plant fibres.
Occurrence: They may also be found in the crops of birds and stomach of ruminants.
Genesis

- These arise from plant fibres and awns which are impregnated with triple phosphate and rolled into balls.

Gross pathology : These have a velvety surface, light in weight and are brown in color.

TRICHOBEZOARS

Synonym: Piliconcretions; Hair balls
Definition: Tricho= hair; bezoars= concretions
Occurrence: Hair balls are found mostly in the rumen of calves.
Etiology

- Animals having itching skin conditions (animals infected with mange or lice) may lick each other when the loose hair may be swallowed.
- Similarly, calves kept together in enclosures, suck and lick each other's ears, tails etc., and swallow hairs.

Genesis

- The hair is rolled into balls during ruminal contractions.
- Mucus of rumen may form a smooth coat over such balls.

Sequelae of concretions

- More often these concretions are found only at postmortem examination.
- The enteroliths, phytobezoars and trichobezoars are usually of no consequence unless they obstruct the passage, when, sometimes, even rupture may occur.
- Cattle may regurgitate a food ball into the esophagus which may be choked.

COLI GRANULOMA IN FOWLS

Synonym: Hjarre's Disease
Definition: A granulomatous condition of the fowl intestine and liver, caused by a mucoid strain of E.coli
Distribution : It has been reported in adult fowls in many parts of the world including India.
Gross pathology

- *Intestine*
  - A large number of grayish- white nodules varying in size from a millet seed to a hazel nut are found projecting from the serosal surface of the intestines.
  - These are distributed diffusely from the duodenum to the ceca.
  - In some places, the whole circumference of the bowel may be involved.

Histopathology

- The lesion is a granuloma involving all the structures of the intestinal wall with exfoliation of the mucosa and fibrous thickening of the serosa.

- A typical nodule consists of the following structures
    - a central structure less mass which is calcified in some places;
    - peripheral to this is a zone of caseo-necrotic material with cells in varying stages of degeneration and necrosis;
    - peripheral to this is a zone of granulation tissue, with epithelioid cells and a few giant cells.
- *E.Coli* can be demonstrated in and isolated from the lesions.
- Similar lesions are found in the liver.
- Differences between the lesions of coli granuloma and tuberculosis
    - The lesions of coli-granuloma unlike those of tuberculosis do not form conglomerates.
    - Unlike tuberculosis, lesions of coli granuloma are not found in spleen and bone.
    - In tuberculous lesions, the acid-fast organisms can invariably be found while in coli-granulomatous lesions only *E.coli* is seen.

ANAL GLANDS

- Anal glands of dogs may sometimes be inflammed and may become purulent if infected by pyogenic organisms.
- There may also be swelling of the glands due to retention of the secretion.
- These swellings may cause constipation.

NEOPLASMS

- Lymphocytoma is the most common neoplasm met with in animals. Masses of the neoplastic cells are found in the wall of the bowel.
- Adenoma of the anal glands has been met with in dogs.
- Other tumors met with are: papilloma, adenocarcinoma, lipoma, leiomyoma and sarcoma.

LIVER

Physiological considerations

- At any point of time, 25% of the blood in the body flows through the liver.

Functions of liver

- *Secretion of bile*
    - Bile contains pigments and bile salts. Bile pigments are not useful to the body. On the other hand, retention of these (hyperbilirubinemia) is toxic to the body.
    - Bile salts play an important role in digestion, especially of the fats
    - Bile contains mucin and related substances which helps in emulsification of fats in the bowel.
    - Bile acts as bacteriostatic in intestine.
- *Protein metabolism*
    - Uric acid is converted into a harmless allantoin in animals.
    - Highly toxic ammonium salts are detoxified by converting them into urea.
    - The non-nitrogenous residues obtained after deamination of amino acids, are converted into glucose and ketones which are used by the body.
    - From amino acids
        - Plasma proteins (albumin, globulin, fibrinogen, prothrombin) and tissue proteins are formed and
        - Protein reserves are stored in the liver.
- *Carbohydrate metabolism*
    - Glycogen is synthesized and stored in the liver.

    - Excessive carbohydrates ingested are converted into lipids and stored in the fat depots.
    - At times of need, gluconeogenesis from proteins and fats occurs in the liver.
- *Fat metabolism*
    - Fats that are characteristics of animals are also synthesized from fatty acids and glycerol by liver.
    - With the assistance of choline, liver is able to transform the depot fats into tissue fats (phospholipids) so that the tissues can utilize them.
- *Erythropoiesis*
    - In the bird, liver is the site for erythropoiesis.
    - In other animals, during fetal life, erythropoiesis occurs in the liver. In these animals in severe anemia, erythropoiesis takes place in the liver even in the adult as part of extramedullary hemopoiesis.
- *Iron metabolism*
    - The reticulo-endothelial cells of the liver are capable of destroying the red blood cells and the minerals released (Fe, Cu and Co) are stored in the liver for use again by the body.
- Detoxication
    - Some toxic substances, especially putrefactive products from the alimentary tract are detoxified by the liver.
    - Bacterial toxins and hormones produced in excess of requirements are inactivated.
    - Many drugs used therapeutically are also made harmless by the liver e.g. Morphine, barbiturates, phenol and camphor.
- *Vitamin metabolism and storage*
    - Failure of bile excretion due to hepatic damage interferes with the absorption of fat-soluble vitamins.
    - Vitamin A is stored in the liver
    - Vitamin K is utilized for the formation of prothrombin
    - Some members of the vitamin B group, especially thiamine, riboflavin and niacin are partly metabolized in the liver where they may also be stored.

Liver Function Tests

- The liver has a great reserve power and it has enormous ability to recover from injury. So, the tests are not adequate clinically to evaluate the correct state of the health of liver and hence it is not wise to put too much reliance on these tests.
- Since the functions of the liver are carried out by the activity of enzymes, inadequacy or absence of the particular enzyme may affect one function and so a decrease of one function does not mean that other functions are affected.

Postmortem Changes

- Postmortem decomposition of liver occurs rapidly since gas-forming organisms (*Cl. welchii*) invade from the intestines which are close by. Liver, particularly rich in nutrients, is a good medium for the growth of these bacteria. Gas bubbles form in the blood vessels. The parenchyma and the blood vessels adjacent to the bowel are stained by hemoglobin bluish-black. The presence of gas gives the liver a foamy appearance – "**Foamy-liver**".
- **Imbibition of bile** occurs in the liver tissue surrounding the gall bladder.

Effects of liver failure

- With severe hepatic diseases, a great many vital processes will be affected. More important pathological conditions met with are:
    - Jaundice due to retention of bile pigments.

- Bleeding be due to
  - Lack of absorption of Vitamin K
  - Failure of prothrombin formation
  - Lack of formation of fibrinogen
- Hypoglycemia due to impairment of glucose metabolism making the animal weak and irritable.
- Hypoproteinemia due to failure to synthesize plasma proteins results into generalized edema and emaciation.
- Anemia due to iron and protein deficiencies: liver stores iron and so in liver diseases iron stores are depleted. In liver diseases protein synthesis also does not occur.
- Toxemia due to failure of detoxication of proteins and intestinal toxins.
- Renal failure – Hepatorenal syndrome: In severe hepatic injury, the toxins that are not detoxified are excreted through kidneys resulting into toxic nephrosis. Renal dysfunction leads to uraemia .
- Pyrexia: The thermoregulatory center is affected by the circulating toxins since they are not detoxified.

DEGENERATIONS

Cloudy swelling

- *Occurrence* : Cloudy swelling is common in the liver.
- *Aetiology*
  - Hypoxia
  - Poisons
    - Chemicals- salts of heavy metals – arsenic and lead.
    - Plant toxins – glucosides, saponin.
    - Drugs – carbon tetrachloride which was used as an anthelmintic
  - Bacterial toxins seen in all infectious diseases.
  - Viruses.
- *Gross pathology*
  - The organ has a dull, parboiled appearance.
  - The liver is enlarged
  - The capsule is tense.
  - Borders are rounded.
  - Consistency is softer.
  - On section, it bulges at the cutting surface.
  - Lobular markings are indistinct.
- *Histopathology*
  - Hepatocytes are swollen and have a pale granular cytoplasm due to swelling of mitochondria. The nuclei may be indistinct.
- *Sequelae*
  - Recovery of the cells occurs if the cause is removed.
  - If the cause persists, the condition may progress to fatty degeneration or necrosis.

Hydropic degeneration

- Etiology
  - Toxins and septicaemic infections
  - Ether,chloroform and carbon tetra choloride poisoning
  - Euthanasia
- Gross pathology : Gross lesions are more or less similar to that of cloudy swelling

- Histopathology
    - The cytoplasm of hepatocyte may contain one, two or more vacuoles in the cytoplasm.
- *Sequelae*
    - Recovery of the cells occurs if the cause is removed.
    - If the cause persists, the condition may progress to necrosis.

Fatty change

- *Occurrence*: Fatty change in the liver is common in animals.
- *Aetiology*
    - Hypoxia/anoxia: Chronic venous congestion.
    - Nutritional deficiency: Inadequate choline and apoproteins
    - Metabolic diseases– Diabetes mellitus in dogs and cats, acetonemia/ketosis in cattle, pregnancy toxaemia in ewes, and deficiency of thyroxine and anterior pituitary hormones.
    - Bacterial toxins
    - Poisons: Inorganic – phosphorus, arsenic, antimony; Organic - chloroform, carbon tetrachloride, tannic acid, tetrachlorethylene, alkaloids of phytotoxins, aflatoxin and senecios.
- *Pathogenesis*
    - The liver is too sick to metabolize the dietary as well as the fat brought to it from the depots. Defect may be anywhere in the process from metabolism of fatty acids to the formation and release of lipoproteins.

Gross pathology

- The liver is enlarged with round margins, has a smooth surface and is pale or yellowish.
- Consistency is friable.
- On section it bulges on the cut surface.
- Fat droplets are seen on the blade.
- Very fatty livers as in pregnancy toxemia of sheep, float in water.

Histopathology

- The hepatic parenchymal cells contain fat droplets, either as a single large globule or as multiple small globules.
- If single and large the nucleus may be pushed to a side.
- The sinusoids are compressed and so appear ischaemic.
- Usually the distribution of the lesions in the liver may be diffuse or zonal.
- In chronic venous congestion, the lesion is in the centrilobular hepatocytes.
- In poisoning, when poison is brought through the portal vein, the fatty changes are found at the periphery of the lobule.
- Rupture of affected hepatocytes may give rise to fatty cysts.

Sequelae

- If the cause is removed early, the condition can be completely corrected.
- In continued presence of the pathogen, cirrhosis will eventually result.

HEPATITIS

- Hepatitis is an alterative inflammation of liver in which the various degenerative processes like cloudy swelling, fatty change and necrosis are caused by irritants which also produce inflammation. Besides, in liver, these degenerative changes are accompanied by lymphocytic or leucocytic infiltrations typical of an inflammatory reaction.

- Hepatitis is classified as alternative inflammation because the inflammatory process is caused by the same etiological agents that also produce degeneration and so alteration in the parenchymatous cells is produced.
- Hepatitis may be either infectious, non-infectious or toxic. This may again be acute or chronic. The chronic variety is usually called **Cirrhosis**.

Infectious hepatitis: Infections in which the liver is only or primarily affected:

- Infectious canine hepatitis (Rubarth's disease)
- Wesselsbron disease of sheep, a viral disease found in South Africa.
- Viral hepatitis of ducks.
- Viral hepatitis of poultry.

Infections in which liver is also affected along with other organs (Secondary)

- Suppurative conditions
- Salmonellosis
- E. coli infection in poultry
- Coligranuloma of poultry
- Pasteurellosis
- Brucellosis
- Glanders
- Actinomycosis
- Botryomycosis
- Necrobacillosis
- Tuberculosis
- Rift valley fever
- Toxoplasmosis
- Histoplasmosis
- Coccidioidomycosis
- Leptospirosis
- Blackhead in turkeys (Histomoniasis)

Routes of infection

- Infection to the liver may be conveyed through several routes. The following are the more important.
    - **Portal vein** : Ingested organisms enter the portal vein and so are conveyed to the liver.
    - **Hepatic artery:** Organisms when present in the blood as emboli or in a bacteremic state reach the liver.
    - **Umbilical vein of the new born animals:** When the umbilical vein is contaminated, organisms grow well in the partially coagulated blood which acts as a good medium and reach the liver. *Fusobacterium necrophorum* and pyogenic bacteria are the commonest organisms involved in producing hepatic necrosis and abscesses respectively.
    - **Bile ducts:** Infection may ascend from the duodenum. Obstruction of bile ducts causing biliary stasis may facilitate such infections.
    - **By direct extension** from neighbouring organs as in traumatic reticulitis.

ACUTE TOXIC HEPATITIS

- This is characterized by necrosis which is usually preceded by degenerative changes like cloudy swelling and fatty changes.
- Hepatic necrosis is classified as per anatomical distribution into
    - Focal necrosis
    - Centrilobular necrosis
    - Midzonal necrosis
    - Periportal necrosis
    - Diffuse necrosis
    - Paracentral necrosis

FOCAL NECROSIS

- In this variety, numerous small necrotic areas are seen scattered in the liver and may be found in any part of the lobule.

Aetiology

- Obstruction of biliary passages

- Bacterial – in bacteremic or septicemic affections – Pasteurellosis, Johne's disease, Salmonellosis, Tularemia, Listeriosis in new-born.
- Viral – as in equine viral rhinopneumonitis in the foetus.
- Due to parasitic migration.

Sequelae

- Focal necrosis of the liver is not of significant consequence since the function of the liver is not much affected.
- Healed lesions show some scarring but this also disappears after sometime.

CENTRILOBULAR NECROSIS

Definition : In this condition, the cells nearest to the central vein are affected.

Aetiology

- *Anoxia*
  - Low atmospheric pressure
  - Acute hemorrhagic anemia
  - Shock – due to reduced blood pressure, reduced oxygen tension and reduced volume flow.
  - Congestive cardiac failure
- *Toxins* : Blood borne especially carbon tetrachloride.

Gross pathology

- The liver is enlarged and paler than normal. In severe cases the organ may be dark red due to venous stasis.
- The lobular markings are exaggerated. This is due to the difference in color at the center and periphery. When congestion of the central part is present, the periphery is paler due to degenerative changes in the cells.
- On the other hand, if necrosis of the cells in the center occurs, then the center will be pale while the periphery is darker.

Histopathology

- The cells round about the central veins have disappeared, blood taking up their places.
- In area nearer to the periphery of lobule, the cells may show fatty changes or cloudy swelling.
- Infiltration of the periportal connective tissue by lymphocytes is seen after some days.

Sequelae

- Single affection may heal by regeneration.
- Repeated attacks will result into fibrosis and atrophy.
- Pseudolobulation with proliferated bile ducts and resulting nodulation is the ultimate result found in frank post-necrotic cirrhosis (Hobnail liver).

MID-ZONAL NECROSIS

- This lesion is found in mid-way between the central vein and the periphery of hepatic lobe.
- This condition is not seen in animals.
- Mid-zonal necrosis is seen in yellow fever of man.

PERIPORTAL NECROSIS

- In this condition the cells adjoining the portal tract become necrotic.

Aetiology

- Here the toxins should have been conveyed by the portal vein.
- This is more commonly seen in phosphorus poisoning.

Histopathology: Inflammation of the portal triads results in cirrhosis similar to portal cirrhosis.

MASSIVE NECROSIS / ACUTE YELLOW ATROPHY

- In this condition, there is necrosis of considerable number of the cells in a lobule. This may be a severe manifestation of various types of necrosis described above.

Etiology

- Dietetic: Deficiency of sulphur-containg amino acids, Tocopherols and Selenium.
- Poisons: Carbon tetrachloride, chloroform, phosphorus.

Gross pathology: The liver is yellow and smaller in size.

Histopathology: There is fatty change and necrosis of hepatocytes with loss of parenchyma.

Sequelae

- Since whole parenchyma of the lobule is dead no regeneration occurs.
- The reticulum and fibrous frame work collapse and there is post-necrotic scarring. Death will occur ultimately.

PARACENTRAL NECROSIS

- This is a peculiar type of wedge-shaped necrosis occurring only on one side of the central vein extending up to the periphery.
- This type is encountered in Rift-valley fever and in uremic conditions.

SAW DUST LIVER

- The foci of necrosis may be few or many, and appear to the naked eye as though saw dust is sprinkled on the liver.

Aetiology: In well-fed young cattle, at postmortem, focal necrosis of the liver is commonly seen.

Pathogenesis

- Here the irritant is borne by the portal veins from the gut and so the lesions are found nearer the portal areas.

Clinical signs : The animals do not manifest any symptoms while alive.

Histopathology

- These spots are evidently scars resulting from inflammatory reaction.
- The lesion consists of hepatic cells which have undergone coagulative necrosis. There is also infiltration by lymphocytes and neutrophils.

TRAUMATIC INJURY

- Traumatic injury to liver may occur due to automobile accidents, crushing by dam in case of young ones etc.
- At the site of rupture of liver blood clots and fibrin may be present.

CIRRHOSIS

- Cirrhosis of the liver is chronic hepatitis characterized by degeneration and hyperplasia of hepatic cells and fibrosis.
- The stimulus for the fibroblastic proliferation is some irritant, chronic and severe enough to produce degeneration and necrosis of the parenchymatous cells.
- The irritant may reach the liver through (a) The portal vein (b) hepatic artery and (c) Bile ducts.

The classification of cirrhosis

- Portal or nodular cirrhosis
- Multinodular or Atrophic or Gindrinker's or Laennec's cirrhosis
- Biliary cirrhosis (Monolobular or hypertrophic cirrhosis)
- Other forms of cirrhosis

PORTAL OR NODULAR CIRRHOSIS

Aetiology

- Frequently one may not be able to ascertain the cause.
- The irritant is mild and acting for a long time.
- Usually, the causes are the same as described for acute focal toxic hepatitis.

- Toxic plants: *Crotalaria saggitalis* in horses; plants of *Senecio* family in horses, cattle and sheep; *Atalaya intermedia* in horses; *Amsinckia intermedia* (tar weed) in horses, swine and cattle; plants containing high selenium content-in horses. (Wheat loco weed).
- Chemicals: Pitch in tar paper, repeated exposure to chloroform, carbon tetrachloride and phosphorous.
- Long continued intestinal toxaemia

Pathogenesis

- When the irritant is conveyed via the portal veins, changes are first at the periphery of the lobules –area next to the portal tract. The following changes take place:
    - Stimulation of the interlobular connective tissue to proliferate
    - Depending upon the severity of the irritant degeneration or necrosis of the hepatic tissue.
    - New irregular blood vessels are formed which anastomose with the network of the portal vein and hepatic artery resulting into arterio-venous shunts. Thus ischaemia of some parts of the liver occurs leading to further hepatic necrosis.
    - Infiltration of lymphocytes and macrophages occur into the islands of Glisson.
    - Hyperplasia of the surviving hepatocytes replaces the destroyed cells.
    - The newly formed after maturation contracts, interfering with blood circulation.
    - Due to resultant ischaemia, hyperplasia does not progress further.
    - As the fibrous tissue grows into the lobule, the hepatic cells become atrophied.
    - The fibrosis constricts the central vein impeding the out flow of blood, thereby rendering the irritant to stay longer in the liver.
    - Growth of the fibrous tissue into the lobule divides the parenchyma into small islands of hepatic cells-pseudolobulation.
    - In the new fibrous tissue, especially in the portal areas, new bile ducts are formed. These are not functional, lacking an outlet and so stasis of bile occurs.
    - If irritant enters the liver through the hepatic artery , changes of damage are first noticed in the tissues of portal canal and inter-lobular connective tissue. The features here are:
        - lymphocytic infiltration
        - proliferation of the connective tissue slowly encroaches into the lobule which produces changes as described above.

Gross pathology

- In the early stages the liver may be large. But as the condition progresses, due to atrophy of the parenchyma, the liver is reduced in size.
- Due to biliary stasis, the color of the organ is tawny or yellowish-gray or green and it is to this color that the name “Cirrhosis” was first applied.
- The liver surface is uneven and nodular (Hobnail liver).
- The liver is hard and firm.
- On section, the liver cuts with difficulty giving a peculiar grating sound due to the dense fibrous tissue formed.

Histopathology

- The architecture of the liver is lost.
- The characteristic picture is the increase in fibrous tissue within and around the lobules.
- In the portal area small new bile ducts and inflammatory cells (lymphocytes and macrophages) are present.
- Deposition of the bile pigment is present

- Central veins in some lobules are either absent or are placed eccentrically (Indication of pseudolobulation).
- The parenchymatous cells show various stages of degeneration - cloudy swelling, fatty change and even necrosis.
- Hyperplasia that is present gives nodularity to the organ. These regenerating young cells are plump, robust and stain more intensely.

Sequelae

- Once the fibrous tissue is stimulated to proliferate, this proliferating fibrous tissue itself becomes an irritant.
- So, even if the original irritant is removed or destroyed, cirrhosis progresses with more and more fibrous tissue formation until the condition terminates fatally.

MULTINODULAR OR ATROPHIC OR GINDRINKER'S OR LAENNEC'S CIRRHOSIS

Occurrence

- This is portal cirrhosis of man.
- In the dog, a similar condition is met with, though not due to similar etiology but to toxins absorbed from the intestines.

Aetiology

- Deficiency of Vitamin B complex and lipotropic factors, especially in drunkards produces this condition.

Pathogenesis

- Lack of Vitamin B complex and lipotropic factors will result into a highly fatty liver and subsequently a severe fibrovascular proliferation.

Gross pathology : The gross changes are more or less similar to portal cirrhosis.

Histopathology

- The hepatocytes show severe fatty changes.
- The bulging cells, pressing on the sinusoids produce ischemia resulting in necrosis of the parenchyma.
- New capillaries form and invade the lobule and connect the central vein with the portal vessels.
- There is proliferation of the fibrous tissue which is infiltrated by chronic inflammatory cells.
- The fibrous tissue divides the parenchyma into smaller lobules.
- Some surviving cells proliferate and form nodules (Hobnail).
- Contracting fibrous tissue makes the liver smaller and hence “Atrophic cirrhosis” results.

BILIARY CIRRHOSIS (MONOLOBULAR OR HYPERTROPHIC CIRRHOSIS)

- Biliary cirrhosis in animals is rare because cholangitis and cholangiostasis do not occur in them.
- Liver flukes that inhabit the bile ducts do not cause extensive cirrhosis but only a local fibrosis.

Aetiology

- In man this type of cirrhosis occurs consequent on obstruction and infection of the biliary tract.
- The causes are
    - Cholangitis-the inflammatory exudate clogs the bile ducts
    - Stone in the common bile duct.
    - Obstruction of biliary passages by flukes (*Chlonorchis sinesis*) and ascarids.
    - Stricture of the bile duct.
    - Extramural pressure on the bile ducts from tumour of pancreas

Gross pathology: The liver is enlarged, greenish and the surface is either smooth or finely granular.

Histopathology

- Here, the connective tissue encircles individual lobules (hence monolobular).
- The bile ducts may be dilated and tortuous.
- There is great infiltration of the connective tissue with chronic inflammatory cells.
- Newly formed non-functional bile ducts are also found.
- Hepatic cells reveal degenerative changes.

SEQUELAE / EFFECTS OF CIRRHOSIS

Sequelae / Effects of Cirrhosis

- Due to disturbance in portal circulation
    - Ascites due to
        - Increased hydrostatic pressure in portal veins due to compression of veins and distortion of the portal and hepatic veins as well as sinusoids. The effect is more in portal cirrhosis.
        - Decreased colloid osmotic pressure due to decreased production of plasma proteins, particularly albumin.
        - Hormones are not inactivated by a damaged liver. Hence mineralocorticoids and the anti-diuretic hormone leads to retention of sodium chloride and generalised oedema.
    - Varicosity and rupture of esophageal veins may lead to hematemesis.
    - Splenomegaly.
    - Chronic gastroenteritis as a result of chronic venous congestion of abdominal viscera.
    - Caput medusae in man – This is dilatation of the cutaneous veins around the navel.
- Loss of Inactivation of hormones and detoxification
    - Estrogens normally are inactivated in the liver in the male. In hepatic cirrhosis this does not occur and so gynecomastia and testicular atrophy occur.
    - Toxins – exogenous or endogenous are normally detoxified by the liver. If this is not done, the toxins affect the brain, producing degenerative changes resulting in “walking disease” in horses.
- Jaundice – due to pressure on the bile canaliculi by the compressed cord cells (by fibrous tissue). So there is obstructive jaundice resulting in digestive disturbances.
- Bleeding due to deficiency in production of prothrombin.
- Anemia – since iron and erythrocyte maturation factor cannot be stored.
- Vitamin A deficiency since Vitamin A cannot be stored in the liver.

ABSCESS

- Infection by pyogenic bacteria cause abscesses in liver.
- The bacteria enter the liver by way of portal veins and hepatic arteries mostly.
- Infection may also occur from the umbilical vein in the young animal.
- In the adult and older cattle, infection may occur from traumatic reticulitis.
- In countries where cattle are fattened for slaughter, abcesses are frequently encountered in the liver.
- The cause is *Fusobacterium necrophorum,* which gains entry through the portal vein. In these animals, highly concentrate diet produces ruminal disturbances, resulting into ulcers. From infected ulcers, *Fusobacterium necrophorum* reaches the liver, where it produces first coagulative necrosis and subsequently abscess on liquefaction.

NEOPLASMS

- Tumours of the liver may be primary (arising from the liver parenchyma and bile ducts) or secondary metastases from elsewhere.

Primary neoplasms

- The most common primary tumours are hepatomas, hepatocellular carcinomas and cholangiocellular carcinoma.
- In dogs, hemangiomas are common.
- Primary fibroma may also be found in the liver.
- In chicken, lymphoid leucosis and Marek's disease primarily affect liver.

Secondary

- Metastases of any malignant tumor may be found in the liver.
- Metastases of lymphocytoma and pancreatic carcinoma are mostly seen
- In the cow, metastases of uterine carcinoma are common.
- Mammary gland carcinoma in the dog metastasizes in the liver.

CHOLANGITIS

Inflammation of bile duct is called cholangitis.

Aetiology

- Mostly liver fluke infection
- Hepatic coccidiosis by *Eimeria stiedae* in rabbits

Pathogenesis

- Cholangitis is caused by the irritation of the spines on the cuticle of the parasites as well as the toxins liberated by the liver flukes.

Gross pathology

- The lumen of the bile duct is dilated and its wall is thickened due to fibrous tissue proliferation around it.
- These ducts stand out as thick cords.
- In some cases, calcification makes them hard (Clay pipe appearance)

Histopathology

- The bile duct mucosa is hyperplastic with papillary projections.
- The bile duct wall is infiltrated by macrophages and lymphocytes.
- The lumen contains mucus.
- The fibrous tissue proliferates around the walls of the bile ducts and may extend to a short distance into the parenchyma of the liver.

Sequelae: Occlusion of the bile ducts may give rise to obstructive jaundice.

CHOLECYSTITIS

- Inflammation of gall bladder is called cholecystitis.
- This is rare in animals.
- It is caused when stasis of bile occurs by
    - the presence of foreign bodies
    - parasites
    - concretions
    - Extramural pressure on bile duct
- E.Coli and *Salmonella* are frequently associated with cholecystitis.
- Infection is usually ascending from the duodenum.
- The retained bile itself acts as an irritant.
- Usually the catarrhal variety is noticed with congested mucosa and increased secretion of mucus by the glands.

CHOLELITHIASIS

Occurrence

- Gall stones or choleliths are not as common in animals as in man.
- These are found mostly in cattle

Etiology: Almost always gall stones occur as a result of cholescystitis.
Pathogenesis

- The dead cells, bacteria or mucus may form the nuclei for development of cholelithiasis.
- Sand particles and food materials that may reach the gall bladder through the bile duct from the duodenum during violent peristalsis may also form nuclei of the choleliths.
- Cholesterol is normally held in solution by loose combination with bile salts. This combination may be easily broken up.
- In cholecystitis, the bile salts are rapidly absorbed leaving the cholesterol which is precipitated.
- Gall stones are composed of cholesterol, bilirubin, bile salts, calcium and organic matrix.

Gross pathology

- The gall stones may be found in the gall bladder or bile ducts but unlike in man, bile ducts are more often affected because of frequency of parasitic involvement.
- They may arise in the bile passages of the liver also.
- There may be numerous small stones or a few large ones in the gall bladder.
- The larger ones may be faceted due to rubbing against one another.
- These may be dark brown or yellowish-green in color.
- They are light and friable.

Sequelae

- Most of the gall stones are "silent" and cause no symptoms, but some may cause colic, nausea and dyspepsia.
- If the bile passages are obstructed, obstructive jaundice may occur.
- If the obstruction of the bile duct is complete, rupture of gall bladder may occur sometimes.

PANCREAS

EXOCRINE DISORDERS

- In animals, diseases of the pancreas are uncommon.

Acute pancreatic necrosis

- This condition may sometimes be met with in dogs, cat, swine and horses.
- Ruminants are usually not affected.

Aetiology

- Occlusion of the pancreatic duct (by parasites) or injuries or circulatory disturbances or regurgitation of bile or bacterial infection (via blood or ascending infectionfrom the intestines) may be the causes.

Pathogenesis

- The essential lesion is necrosis of pancreas by its own enzymes especially the proteolytic enzymes.
- The enzymes escaping out of the pancreatic tissue digest the surrounding peripancreatic fat (fat necrosis) first and the pancreatic parenchyma subsequently.
- Entering lymph channels, the lipase may produce fat-necrosis in distant organs, even as far away as anterior mediastinal region.

Gross pathology

- In fatal cases, there is a small quantity of fluid in the abdominal cavity.
- Haemorrhages may be present in the omentum.
- In the mesentery and around the pancreas whitish areas or nodules of fat necrosis with an inflammatory zone surrounding them are found.
- The pancreas is swollen and soft, yellowish or slightly hemorrhagic.

- The lesions may be widespread or localized. If limited to a little area, encapsulation may occur. On section, yellowish-grey, soft (pus like) areas of necrosis may be visible.

Histopathology

- Pancreas may show the following changes
  - Peripancreatic fat will reveal necrosis, granulomatous reaction and calcification.
  - Necrosis of the parenchymatous cells
  - Edema, haemorrhage and infiltration by a few leucocytes
  - Thrombosis of vessels.

Sequelae

- Death in acute cases after manifesting severe abdominal pain and cardiovascular collapse in shock.
- Chronic fibrosing pancreatitis may result if the episodes are repeated. In the some dogs, the organ is enlarged due to great increase in the scar tissue.
- Steatorrhoea occurs due to loss of the pancreatic juice. In this condition the feces is greasy and foul smelling.
- Diabetes mellitus with glycosuria may be seen in dogs.

DIABETES MELLITUS

Definition

- Diabetes mellitus is essentially the derangement of the carbohydrate metabolism due to insulin deficiency and characterized by hyperglycemia and glycosuria.

Physiological considerations

- Carbohydrates are absorbed as glucose, which is converted into glycogen and stored in the liver.
- When needed by the tissues, muscles in particular, glycogen is converted into glucose – 6 – phosphate and then oxidised releasing energy.
- These processes are regulated and controlled by various hormones. The most important of these is insulin.

INSULIN

- This is a hormone produced by the Beta cells of the islets of Langerhans.
- Insulin has the following functions
  - helping in the storage of glycogen in the liver, facilitating the entry of hexoses across the cell membrane into the cell (muscles especially)
  - stimulation of hexokinase for formation of hexose – 6 – phosphate and inhibition of activity of hepatic glucose – 6 – phosphatase and thus preventing over production of glucose.

Insulin deficiency

- Insulin deficiency may arise when
  - Insulin may not be adequately synthesized due to necrosis of pancreas.
  - Insulin may not be liberated into the circulation though synthesized by the Beta cells.
  - Diminished production of insulin due to "work-exhaustion". This occurs when insulin-antagonists act for a long time like
    - **Insulinase** , a proteolytic enzyme which destroys insulin.
    - **Glucagon** and **epinephrine** are anti-insulin by virtue of their capacity to stimulate hepatic phosphorylase and produce glycogenolysis and hyperglycemia.
    - **Growth hormone** (S.T.H) antagonizes
      - the effect of insulin on hexokinase;

        - the ability of insulin to transport glucose across the cell memebrane.
        - by stimulating insulinase and
        - by probably stimulating the release of glucagons.
    - **Thyroxine** increases the metabolic rate and gluconeogenesis.
    - **Adrenal cortical hormones** antagonize by gluconeogenesis and supporting the action of growth hormones.
- The above antagonists first stimulate the islets of the pancreas, which become hyperplastic, releasing excess of insulin to arrest the hyperglycemia produced by them. In time, the cells become exhausted and atrophied.

Pathogenesis

- Diabetes mellitus may be present from birth as an inherent defective carbohydrate metabolism and this will be manifested later as diabetes due to various causes, viz, stress due to pregnancy, ACTH therapy, Cushing's syndrome, overeating, streptococcal infections and acromegaly. This error is due to an inherited recessive Mendelian factor.
- Insulin in diabetes may be in the body as inactive complex.
- There may be autoimmunity so that patient's antibodies against insulin may inactivate the insulin in the body.
- When this is above the renal threshold (in dog normal is 160 to 180mg per 100 ml) renal tubules are unable to completely reabsorb the glucose of the glomerular filtrate and so**glycosuria** results.
- Now because of glucose in the urine, its osmotic pressure rises and this prevent the reabsorption of the water by the tubules and so **polyuria** also results giving rise to increased thrist, polydypsia and dehydration.
- Glycogen stores of the liver are depleted due to glycolysis.
- So sufficient amounts of pyruvic acid and oxaloacetic acids are not formed to combine with active acetate formed from the fats. So this active acetate accumulates, condenses and forms ketone bodies, which in excess produce **ketonaemia** and **ketonuria**. Being acidic, the ketone bodies neutralize the alkali reserve resulting into acidosis which terminates in air hunger and coma.
- Since tissues are unable to utilize glucose (except nerve cells and red blood cells which do not require insulin for glucose utilization), catabolism of proteins and fats take place as source of energy.
- Since fat of fat depots has to move into liver for phosphorylation (without which it cannot be utilized in the tissues) fatty change of liver occurs.
- Ketone bodies are formed in excess due to catabolism of excess of fats and so ketonaemia occurs. (Normally small quantities of ketone bodies are produced but these are metabolized in the body).
- Ketonaemia gives rise to ketonuria. The breath and urine have the characteristic sweet odor.
- These ketoacids interact with sodium and potassium salts and so these bases are lost in the urine and acidosis develops.
- Acidosis, dehydration and ketonemia give rise to coma.
- Protein is catabolised to amino acids from which glucose (gluconeogenesis) and fatty acids are formed.
- Excess of amino acids are deaminised in the liver and so there is elevation of blood and urine non-protein nitrogen.
- With the depletion of carbohydrates, fats and proteins, body weight decreases in spite of consuming considerable quantities of food.

Animals affected

- Diabetes mellitus may be found in dogs and cats.
- In dogs it is a disease of older animals, especially in females, due to chronic pancreatitis. For some unknown reason, such dogs develop cataract in the eye.
- In lambs, diabetes mellitus is seen in those that are overfed on carbohydrates. Glycosuria is met with in enterotoxaemia in sheep.

Clinical pathology : Lipemia is evident with the serum appearing white.

Gross pathology

- The animal is emaciated and dehydrated.
- The liver is highly fatty.
- The pancreas may either be normal or show pancreatitis and necrosis with fibrosis.

Histopathology

- *Pancreas*
  - Beta cells: Vacuolation, necrosis and hyalinization of Beta cells have been noticed.
  - The epithelium of ducts show vacuolation and is due to glycogen infiltration.
- *Kidney*
  - Glycogen infiltration of the epithelial cells of the Henle's loops and the distal convoluted tubules is noticed.
- *Liver* : The liver cells are loaded with fat.
- *Other organs* : The retinal and vascular lesions of man are not met with in animals.

PATHOLOGICAL CONDITIONS

ASITES OR HYDROPERITONEUM

Definition : This is edema of the peritoneum

Occurrence : This is common in dogs and cats but may also be encountered in sheep and cattle.

Aetiology

- Hypoproteinemia – gastrointestinal trichostrongylosis and Johne's disease in which there is protein loss.
- Cachectic diseases: anaemia and starvation.
- General chronic venous congestion – cardiac or pulmonary lesions.
- Portal obstruction due to hepatic lesions – cirrhosis, hydatidiasis, fascioliasis, neoplasia (secondary) and pressure upon the vein by neoplasms, abscesses and enlarged lymph nodes.
- Urinary obstruction in male cattle and sheep with or without rupture of bladder.
- Increased capillary permeability due to histamine release in shock or due to toxins as in edema disease of pigs
- Lymphatic obstruction in peritoneal cavity.
- Carcinomas – primary (malignant mesotheliomas) or secondary (extensive implantation carcinomas).

Gross lesion : The peritoneal cavity contains excess of clear colourless or serous fluid

HAEMORRHAGES

Occurrence : Hemorrhages in the peritoneum are common in all animals

Etiology

- It may be due to trauma of abdominal organs or sweet clover disease.
- Small focal hemorrhages are common in acute toxemias (enterotoxemia) and infectious diseases – anthrax, hemorrhagic septicemia and infectious canine hepatitis. These hemorrhages are found on the serosa of the diaphragm, stomach and intestines.

- Hemorrhages in the peritoneum are also caused during the course of certain parasitic diseases such as distomiasis, *Strongylus edentatus* infection etc.

PERITONITIS

Occurrence: Peritonitis is a very common condition in most of the domestic animals

Types: It may be localized or generalized.

Etiology

- Bacteria: *E.coli, Streptococci, Staphylococci, Corynebacteria, Clostridia, Pasteurella* group and Anthrax in pigs.
- Viruses of bovine encephalitis
- Helminths
- Chemicals – introduced for medication
- Endogenous – Bile and pancreatic juice.
- Visceral gout of birds
- Glasser's disease of swine

Routes of entry

- Externally through surgical wounds or from trauma.
- By rupture of an abdominal organ.
- Extension through the walls of stomach, intestine or uterus when their mucosa is inflammed.
- Through osteum abdominale of an infected oviduct.
- From an infected umbilicus.
- Direct extension from an infected kidney.
- By blood stream as in bovine viral encephalitis.
- By way of lymphatics from scrotal infection and infection of abdominal wall.

Pathogenesis

- The irritant first produces a serous inflammation which later becomes fibrinous or fibrinopurulent.
- The fibrin is helpful in localizing the inflammation by forming adhesions. Being a very large absorptive surface of the body, toxins are speedily absorbed from the peritoneum damaging other parenchymatous organs.
- In Glasser's disease of swine, diffuse serofibrinous peritonitis is seen.
- Tuberculosis of the peritoneum is very frequent in cattle, less frequent in dogs and rarely met with in other animals.
- In visceral gout of birds, *uric acid peritonitis* occurs and is characterized by the deposition of urates on the serous membrane which consequently shows inflammatory changes.
- One of the protective mechanisms of nature is the mobilisation and movement of the omentum which covers and sticks to the area of inflammation thereby restricting its spread. But this has its own drawbacks, since adhesions may form between it and the inflammed parts.
- Fibrin that forms, if not removed within 6 to 10 days is organized, thereby inhibiting the movements of the intestines and impeds the digestive process.

NEOPLASMS

- The primary tumors, mesotheliomas (malignant) arise from the serosa and are common in the young and newborn animals.
- The secondary tumors are metastases from the liver or uterus.
- Transcoelomic implantation of ovarian tumour found in women is not common in animals.

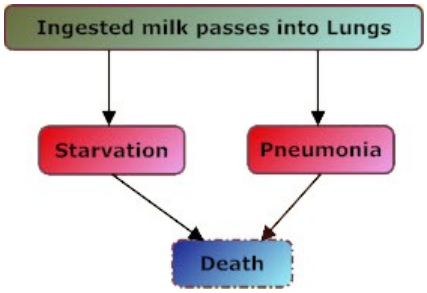
RESPIRATORY SYSTEM

ANOMALIES

CLEFT PALATE

- Abnormal connection between the nasal cavity and the mouth. It is otherwise called palatoschisis.
- Fairly common defect seen in newborn animals.
- Affected animals do not survive long.

Sequelae



NASAL CAVITY

NASAL CONGESTION

Causes

- Physical -compression as in case of overlay in piglets
- Environmental
  - Exposure to cold air (To warm the breathed air blood vessels in the nasal passage dilate).
- Pathological
  - Inflammation (Rhinitis).

EPISTAXIS

Synonym : Nosebleed
Occurrence : Infrequent in animals
Aetiology

- Physical : Trauma- Violent exercise induces pulmonary haemorrhage in horses
- Bacterial : Anthrax
- Parasites : *Eimeria canis* in dogs; *Oestrous ovis* in sheep

Clinical signs :Bleeding may be unilateral or bilateral

SINUSITIS

Aetiology : Bacteria - *Haemophilus paragallinarum* in chicken.
Route of entry : Through the wound produced by dehorning.
Gross pathology

- The head will be swollen
- The eyelids may be closed
- The subcutis contains edematous fluid
- The sinus contains mucus or cheesy material

Sequelae : Spread to eustachian tubes in human being interfere with hearing.

RHINITIS

Definition : Inflammation of the nasal mucosa
Classification

- Primary or Secondary
  - Primay rhinitis - occurs independently
  - Secondary rhinitis - partial manifestation of some of the acute and chronic infectious diseases.
- Based on the nature of the exudates : Serous , Catarrhal , Purulent , Fibrinous.
- Based on the age of lesion: Acute, Subacute, Chronic.
- Based on severity of insult: Mild, Moderate, Severe.

- Based on aetiological agent : Bacterial, Viral, Mycotic, Toxic.

Predisposing Factors

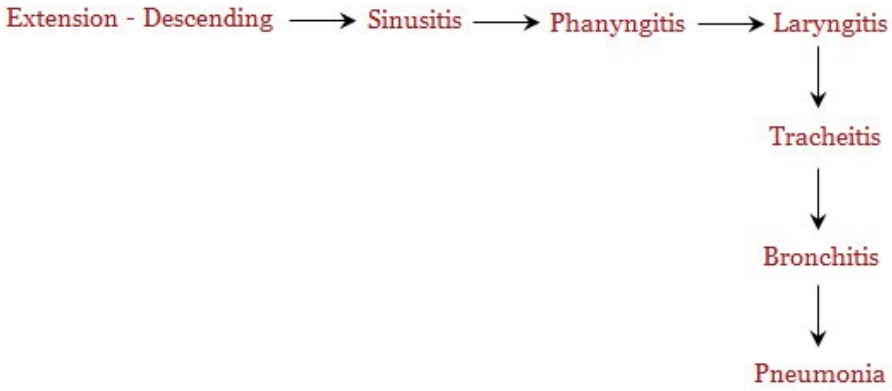
- Environmental changes
- Stress
- Immunosuppression
- Prolonged antibacterial therapy

Etiology

- *Physical*
  - Irritants – Dust, pollen
  - Foreign bodies – rice chaff, insects, flies
  - Injuries
- *Chemical* -Irritating gases, Formalin vapour, Ammonia gas arising from deep litter in poultry
- *Biological*
  - Bacteria
    - *Alcaligens, Bordetella bronchiseptica* – Dogs
    - *Actinobacillus lignieresi, Actinomyces bovis, Mycobacterium tuberculosis* – Cattle
    - *Fusobacterium necrophorum* - calf
    - *Pasteurella multocida* – Pasteurellosis in cattle and swine
    - *Pseudomonas mallei* - glanders; Streptococcus equi – strangles in horse
    - *Haemophilus paragallinarum*(Infectious coryza), *Mycoplasma galliseptium* – chicken
    - *Pseudomonas aeruginosa, Staphylococci*
  - Virus
    - Human - Common cold virus
    - Cattle -Infectious bovine rhinotracheitis (IRBT), bovine malignant catarrhal fever and rinderpest
    - Dog- Canine distemper
    - Cats -Feline viral pneumonitis and feline viral rhinotracheitis
    - Horse -Equine rhinopneumonitis and equine influenza
    - Pigs - Swine influenza and swine inclusion body rhinitis
    - Poultry -Infectious laryngotracheitis and fowl pox
  - Fungus
    - Aspergillus fumigatus
    - Cryptococcus neoformans in cats
    - Rhinosporidium seeberi in cattle
  - Parasite
    - Sheep – Oestrus ovis larvae
    - Dog – Linguatula serrata
    - Cattle – Schistosoma spindale

Route of infection: Inspired air

Sequelae: Rhinitis might extend to cause bronchitis and pneumonia



Classification of rhinitis: Rhinitis is classified as acute and chronic rhinitis.

ACUTE RHINITIS

Definition : Acute inflammation of the nasal mucosa

Causes

- Physical
  - Irritants – Dust, pollen
  - Foreign bodies – chaff, insects, flies
- Chemical irritants
- Infectious agents

Gross lesions

- Mucous membrane
  - Colour: Congested
  - Thickness: Swollen
  - Moisture: Dry /moist / exudate
  - Exudate: Serous / mucous / mucopurulent / suppurative (Purulent) / fibrinous

Histopathology

- Lumen: contains inflammatory exudate with inflammatory cells
- Mucosa:
  - Epithelium shows hydropic degeneration
  - Lamina propria: Hyperaemic, infiltration with inflammatory cells.

CHRONIC GRANULAR RHINITIS

Species affected : Jersey breed of cattle.

Clinical signs

- Nasal discharge - Mucous or mucopurulent
- Nasal pruritus.

Gross pathology : Tiny nodules – cobblestone appearance.

Histopathology

- Mucosa: Many closely packed tiny polyps covered by acanthotic squamous epithelium projecting into the lumen.
- Lamina propria: contains oedema, engorged capillaries, fibroblasts, eosinophils, mast cells and plasma cells.
- Ducts of mucosal glands: Metaplastic changes pseudostratified columnar epithelium.

BOVINE NASAL GRANULOMA

Definition : Granulomatous inflammation of the nasal mucosa.

Aetiology

- Bacterial - Actinobacillosis.
- Parasite - Blood fluke – Adult parasite of *Schistosoma nasale* causes snoring disease in cattle.

Signs

- Interference with normal respiration (snoring sound is heard).
- Profuse nasal discharge – sometimes mixed with blood.
- Gross changes: Lumen is occluded by granulomatous growth.
- Nasal mucosa contains minute ulcers.

Histopathology

- Feature resembles that of pseudotubercle characterised by: central schistosome egg surrounded by eosinophilic rays of dead cells (actinobody).
- Around the actinobody cellular infiltration consisting of lymphocytes, neutrophils, eosinophils and a few giant cells are seen.

Diagnosis

- Nasal discharge – characteristic boomerang shaped ova with spine at one end is chaacteristic of *Schistosoma nasale*.

BULL NOSE

Synonym: Rhinohyperplasia.

Definition

- Slowly developing condition of nasal mucosa with enlargement of nose seen usually in young pigs.

Aetiology

- *Infectious*
    - Bacteria -
        - *Fusobacterium necrophorum*
        - Staphylococci, Streptococci
        - *Pseudomonas aeruginosa*
        - *Corynebacterium pyogenes*

Clinical signs: Dyspnoea

Gross changes

- Nose: swollen and enlarged due to edema
    - Bones of nasal cavity, nasal mucosa and Skin adjacent to the nares show areas of suppuration or caseous necrosis
    - Maxillary and nasal bones – distorted
    - Sinus: occluded with exudates
    - Lymphadenitis

Histopathology

- Edema seen in early lesion.
- Fibrosis seen in chronic inflammation.

NASAL POLYPS

Polyps are non neoplastic masses that resemble tumours.

Species affected: Occurs following chronic rhinitis mostly in horses, also recorded in cats and sheep

Site: Nasal passage or nasopharynx.

Origin: Fibrous tissue of submucosa protrude into the lumen of the nose.

Signs: Respiratory distress.

Gross pathology

- Seen as a growth or mass.
    - Shape: Ovoid.
    - Surface: Smooth and glistening.
    - Consistency: Soft and slimy.
    - Covered by: Nasal mucosa.

Histopathology

- Nasal polyp has a core of well vascularised stromal tissue along with infiltration of lymphocytes, macrophages and eosinophils.

PORCINE ATROPHIC RHINITIS

Definition: Catarrhal inflammation of nasal turbinates which later undergoes atrophic changes

Aetiology

- Combines infection of *Bordetella bronchiseptica* and toxigenic strain of *Pasteurella multocida*

Signs

- Slight catarrh (mucopurulent exudate) or have intermittent epistaxis
- Sneezing
- Dyspnoea progresses slowly over months.
- Anorexia leads to poor growth rate, lameness and fractures.
- Inanition
- Death

Clinical pathology: Paratharmone level is increased.

Gross pathology

- Nasal mucosa is swollen.
- Mucosa of turbinate bones: Small depressed, congested foci lead to erosion and covered by tenacious mucopurulent exudate.
- Turbinate bone resorption leads to hard fibrous tissue replacement which later becomes soft. Then disappearance of nasal septum occurs in some months and only a blood clot remains at the end.
- Excessive bone resorption occurs around the skull sutures.

Histopathology

- Mucosa is infiltrated with lymphocytes, macrophages and a few neutrophils.
- Bone: Rarefaction of bony tissue occurs in 2 -4 weeks postinfection.
- Periostesal region: fibroplasia.
- Nasal mucosa: squamous metaplasia
- Parathyroid shows hyperplastic changes.

Sequelae : Recovery is rare.

TUMORS

Benign	Malignant
Osteoma	Osteosarcoma
Chondroma	Chondrosarcoma
Myxoma	Myxosarcoma
Fibroma	-
Angioma	-
-	Adenocarcinoma
-	Squamous cell carcinoma

Adenocarcinoma

- Arises from mucosal lining of ethmoid bone and olfactory mucosa

- Endemic in certain regions of the world
- Reported in cattle, sheep, goat, pigs and horses
- Causes severe necrosis and haemorrhages
- It can metastasize rapidly.

LARYNX

ROARING

Definition: Roaring is impaired sound due to laryngeal hemiplegia in horses.

Etiology

- Injury and degeneration of the left recurrent laryngeal nerve (primary axonal disease) or secondary nerve damage by repeated trauma by pulsation in the aorta, lead poisoning, pressure on the nerve by aneurysms, enlarged lymph nodes, abscesses, tumours, oesophageal diverticula and other traumatic conditions.

Pathogenesis

- Hyaline degeneration and fibrosis of the left doesal and lateral cricoarytenoideus muscle atrophy and paralysis leads to incomplete dilatation of larynx.
- The arytenoids cartilage cannot open and so will stand in the way of air passing freely into the wind pipe. This condition is accentuated when the animal is exercised. A noise is heard by brushing of air with the arytenoid cartilage and the noise is called roaring.

Gross pathology: Affected muscle is pale and atrophic.

Histopathology

- Left recurrent laryngeal nerve – Demyelination and wallerian degeneration
- Atophic changes of muscle fibres (neurogenic atrophy)

LARYNGITIS

- Laryngitis is the inflammation of the laryngeal mucosa.

Aetiology

- Extension of infection from nasal cavity and pharynx in infectious diseases - Canine distemper in dog; Strangles and glanders in horses; Infectious laryngotracheitis in poultry.
- Irritant vapours of chemicals.
- Mechanical injury by kicks, bites, grass awns; Injuries while passing probes or stomach tube.
- Excessive barking.
- Specific diseases – calf diphtheria, tuberculosis, glanders and actinomycosis.

Gross pathology

- Mucosa of larynx is swollen. The mucosa is haemorrhagic and dry at first, later becomes coated with mucus or mucopurulent discharge.

Histopathology

- Mucosa is covered with exudate - mucus, blood or necrotic material
- Lamina propria is infiltrated with leucocytes

CROUPOUS OR MEMBRANOUS LARYNGITIS

Causes: Infectious laryngotracheitis in fowls.

Gross pathology: The laryngeal mucosa is covered with grayish fibrinous deposits.

Histopathology

- A membrane consisting of fibrin and leucocytes is formed over the necrotic laryngeal mucosa.

Sequelae: The affected fowls may die of asphyxia.

TRACHEA

TRACHEITIS

Definition : Tracheitis means inflammation of the trachea.

Causes

- Bacteria: *Escherichia coli and* Mycoplasma gallisepticum
- Virus: Ranikhet disease virus, Infectious bronchitis virus and infectious laryngotracheitis virus
- Parasite: *Syngamus trachea*.

Gross pathology

- Tracheal mucosa is congested
- Tracheal lumen contains mucus or blood tinged mucus containing red worms.

Histopathology

- The mucosal epithelium is denuded and the lamina propria is infiltrated with leucocytes.

Sequelae: In heavy worm infections, asphyxia leads to death.

ACUTE TRACHEOBRONCHITIS

Occurrence: Usually encountered along with upper respiratory disease and pneumonia.

Route of infection

- Nasal
- Aspiration into trachea
- Extension from pharynx and sinuses

Causes

- Inhalation of irritants like industrial fumes, smokes, feed particles and dust.
- Faulty drenching of medicaments.
- Bacterial – Pasteurellosis.
- Viral – Infectious bovine rhinotracheitis, Ranikhet disease and infectious bronchitis of fowls.
- Parasites - Lung worms.

Gross pathology

- The mucosa is covered by exudates which may be catarrhal, fibrinous or purulent.
- The mucosa is thick and reddish.
- In gangrenous bronchitis, there is extensive necrosis of the mucosa which becomes sloughed.

Histopathology

- Lumen contains mucus, leucocytes, dead epithelial cells, lung worms and their ova.
- Mucosal epithelium shows necrotic changes.
- Lamina propria - Congested and infiltrated with inflammatory cells of which neutrophils predominate.

Sequelae

- Recovery may occur in mild cases.
- Bronchiectasis.
- Chronic bronchitis, peribronchitis and bronchopneumonia.
- Abscess may develop if infected.

BRONCHI

BRONCHOSTENOSIS

Definition: Narrowing of the bronchial lumen due to obstruction or peripheral pressure.

Aetiology

- Aspiration of foreign bodies.

- Accumulation of exudates and infiltration in to the wall causing reduction of diameter of the bronchus.
- Parasites within the lumen.
- Pressure from outside the bronchial wall by abscesses, enlarged lymph nodes, tumours and exudates of pleural cavities.
- Spasms of the muscles of the bronchi as in allergy.

Sequelae

- A partial closure of the bronchi or bronchioles results in ballooning of the alveoli as air that enters during inspiration is not expelled. Repeated inspiration will therefore lead to ballooning of the alveoli.
- Complete obstruction of the bronchus results in collapse of the lung.

BRONCHIECTASIS

Definition: Bronchiectasis means dilatation of the bronchus.

Aetiology and pathogenesis

- Usually due to chronic inflammation of the bronchus. Due to destruction of the elastic tissue of the bronchial wall, contractile power of the bronchus is lost and so the bronchus dilates. At the place of dilatation, exudates accumulate thereby further dilating the bronchus.
- In chronic pneumonia, the bronchial wall is weak. The fibrous tissue on contraction pulls the bronchial wall which gets dilated.
- In bronchostenosis, air accumulates during inspiration below the level of obstruction and causes dilatation of the bronchus. When the bronchi are completely closed resulting in atelectasis, the negative pressure in the pleural cavity pulls the bronchial wall and cause dilatation.

Clinical signs: Persistent cough, Debilitation.

Gross pathology

- Cylindrical form of bronchiectasis is more common in cattle and cause uniform dilatation of the bronchus.
- Saccular form is less common in which there is an out pouching of the bronchial wall due to focal necrosis as occurs in lung worm infections in cattle and sheep.

Histopathology

- Destruction and disappearance of the elastic tissue, musculature and even the cartilage of bronchus
- Mucosa: disappearance of lining epithelium
- Wall: lamina propria infiltrated with mononuclear cells
- Lung: collapsed and carnified; pleural adhesions may develop

Sequelae: Prognosis: The course is chronic and unfavorable.

Complications

- Development of abscesses with metastasis
- Bronchiolitis with emphysema
- Bronchopneumonia
- Secondary amyloidosis

BRONCHITIS

Definition: Bronchitis means inflammation of the bronchial mucosa.

Causes: Viral – canine tracheobronchitis and infectious bronchitis in fowls.

Gross Pathology

- Trachea is congested and contains mucus exudate
- Bronchial lumen is obstructed by yellow plugs of caseous material.

Histopathology

- Bronchial mucosa is thickened due to edema and cellular infiltration.

- Bronchial lumen may contain neurophil or heterophil rich exudate

CHRONIC BRONCHITIS

Aetiology

- Mild, continuous irritant like smoke and dust.
- Extension of chronic infection of upper respiratory tract as in chronic sinusitis.
- Most common cause in animals is lung worm infection, abscess, tuberculosis.
- Pathological – bronchiectasis.
- Chronic venous congestion – as in heart diseases.

Gross pathology

- Size: May be dilated.
- Lumen: Exudate – Mucoid or mucopurulent mixed with worms.
- Colour: Pale (sometimes congested).
- Surface: Velvety.
- Mucosa: Thickness: Thick.

Histopathology

- Lumen – Contains worms and eggs in case of parasitic bronchitis.
- Mucosa - glands may be atrophic; ciliated epithelium replaced by cuboidal epithelium.
- Lamina propria: Infliltration by lymphoid cells.
- Peribronchial glands: hyperplastic (resemble goblet cells).
- Walls: Lymphoid follicles may be formed; Fibrosis may cause polypoid projections into the lumen (Brochiolitis obliterans).

Sequelae

- Bronchi – Bronchiectasis.
- Lungs – Bronchopneumonia; atelectasis; emphysema leads to greater strain on the heart and chronic venous congestion occurs ultimately.

LUNGS

ATELECTASIS

Definition: Failure of alveoli to open and contain air or absence of terminal dilatation.

Types

- Congenital.
- Acquired.

Congenital atelectasis

- *Definition*: In congenital atelectasis, animal is born dead and has not breathed.
- *Causes*
  - Obstruction of the bronchi by – mucus; inhaled liquor amnii.
  - Damage to the respiratory center that may occur in injury to the brain.
  - Muscular weakness.
- *Gross pathology*
  - Lungs: colour - reddish blue (due to dilatation of alveolar capillaries).
  - Consistency - firm.
  - Density: Sink in water (lack of air).
- *Histopathology*
  - Lung: Alveoli – collapsed or may contain fluid; alveolar epithelium – cuboidal.

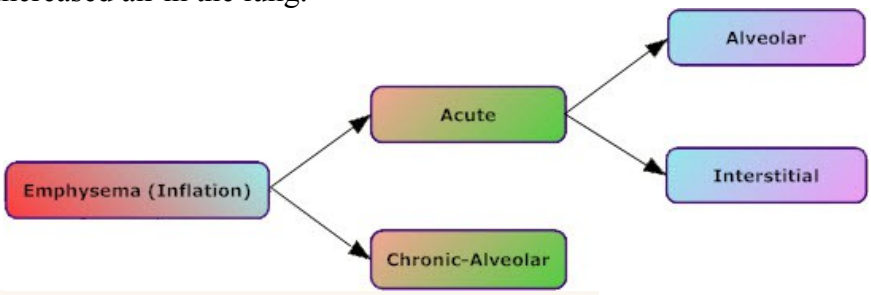
Acquired Atelectasis

- *Causes*
  - Obstruction of the bronchial lumen
    - Lumen: Mucus, pus, foreign bodies or masses of parasites.

        - Outside: Enlarged lymph nodes, cyst, abscess, tumour or aneurysm.
        - Result: Air in the alveoli is resorbed.
    - Compression
        - Extrapulmonary: abdominal distension due to tympany of rumen or ascites, hydrothorax, hydro pericardium or pneumothorax.
        - Crushing injury: Young piglets may be crushed by the dam.
- *Gross pathology*
    - Lungs - There is never a total collapse but only focal collapse.
        - Colour: Dark or reddish blue in colour.
        - Surface: Affected part depressed from the level of surrounding healthy lung.
        - Density: Sink in water.
        - Consistency: Leathery.
    - Pleura
        - Thick
        - Surface: wrinkled.
- *Histopathology*
    - Alveoli appear as small or elongated clefts.
    - Alveoli are devoid of air.
    - Epithelium: Desquamated in later stage.
    - Capillaries: Dilated and engorged with blood.
    - Interlobular septal tissue – proliferate, later (fibrosis).

EMPHYSEMA

Definition : Increased air in the lung.



ACUTE PULMONARY ALVEOLAR EMPHYSEMA

Causes

- Physical: Over exertion during struggling leads to over ventilation.
- Nutritional: Feeding on lush green pastures.
- Pathological
    - Over exertion during coughing (Bronchitis/ Bronchopneumonia).
    - Compensatory at areas adjacent to pneumonia.
    - Loss of elasticity of wall of alveoli.
- Allergy: Feeding mouldy forage or mouldy sweet potatoes.
- Poisons: Parathion poisoning.

Course: Temporary.

Gross pathology

- Certain areas of lung are unduly distended with air.
- Pale or white areas project above surrounding tissue.
- Cut surface is dry.

Histopathology

- *Alveoli*
    - Size: greatly distended (Giant alveoli).

- Wall: Sometimes ruptured combined to form bullae.

ACUTE INTERSTITIAL EMPHYSEMA

Definition

- In this condition air collects in the interlobular septa beneath the pleura and wherever there is interstitial tissue in the lungs.

Occurrence: Often accompanies the acute alveolar emphysema.

Species affected: More often seen in cattle and sheep.

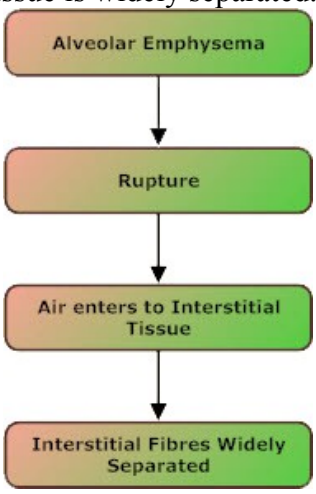
Causes

- Violent respiratory effort (struggling) during death from loss of blood.
- Perforation of lung by mechanical means- foreign body through rumen and reticulum.
- Excessive bellowing in estrum or when separated from calf.
- Forced breathing as in old hunting dogs.
- Pulmonary strongylosis.
- Pathological: pneumonia.

Gross pathology

- Air may escape through thoracic inlet into the subcutis of neck.
- In Severe case,
  - Air collects beneath the pleura and other interstitial tissue of lung.
  - Air accumulate along the spine – from pole to base of tail.
- Interlobular septa are thickened and seen as criss cross straight lines usually at wider intervals.
- Interlobular septum: shiny, well outlined and filled with large and small air bubbles.

Histopathology: Lung interstitial tissue is widely separated.



CHRONIC ALVEOLAR EMPHYSEMA

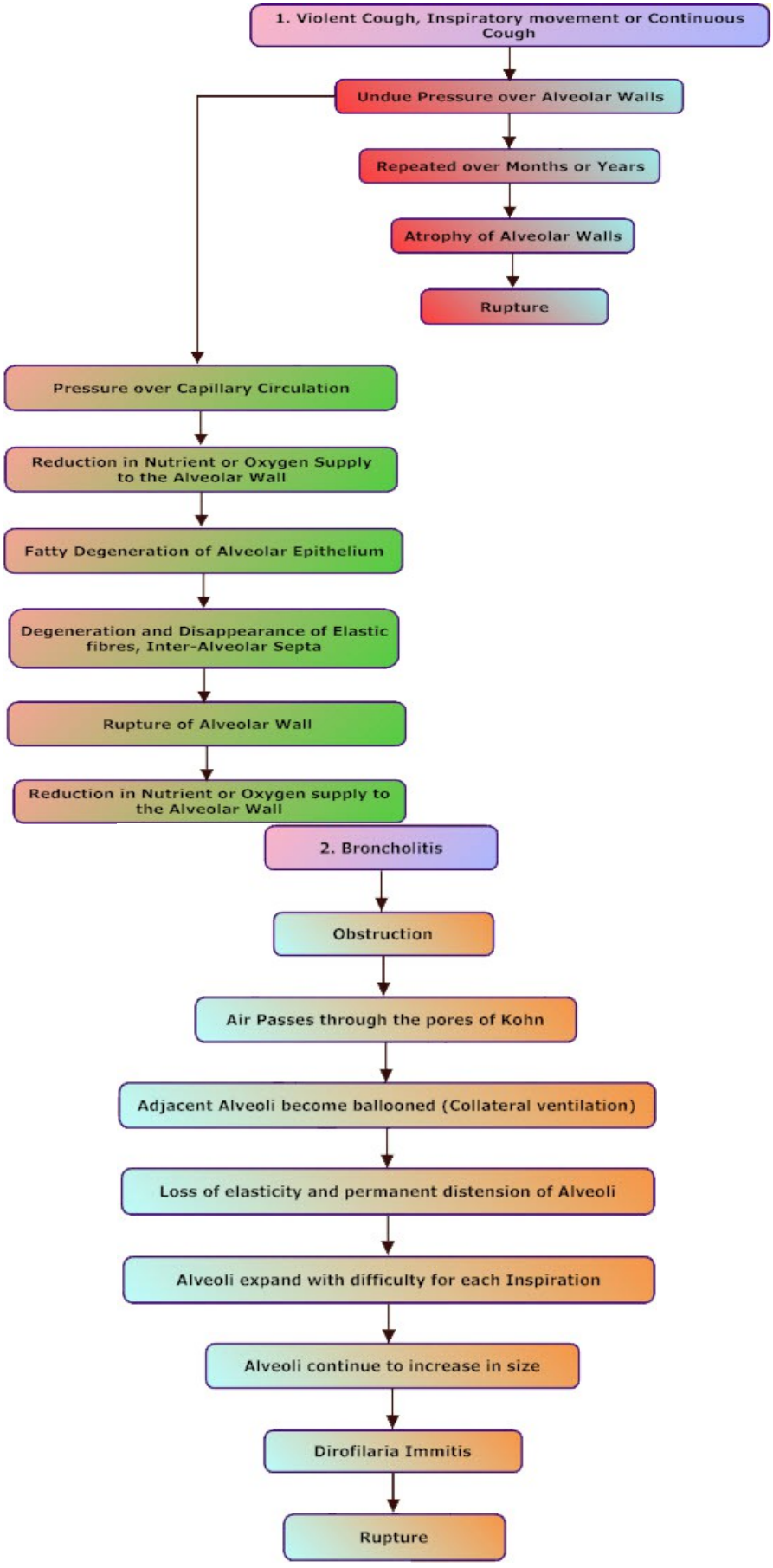
Synonym : Broken wind/heaves.

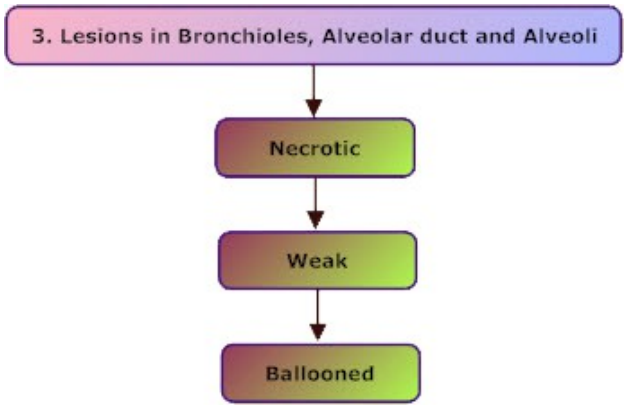
Species affected: More often seen in horses.

Causes

- Heavy meal in working horses causes distension of abdominal organs which prevents expansion of diaphragm. Such animals make violent respiratory effort.
- Physical: Dust / Mould causing coughing.
- Pathological: obstruction of bronchi as in bronchiolitis.
- Allergy: Dust, pollen grains and fungi.

Pathogenesis


1. Violent Cough, Inspiratory movement or Continuous Cough
Undue Pressure over Alveolar Walls
Repeated over Months or Years
Atrophy of Alveolar Walls
Rupture
Pressure over Capillary Circulation
Reduction in Nutrient or Oxygen Supply to the Alveolar Wall
Fatty Degeneration of Alveolar Epithelium
Degeneration and Disappearance of Elastic fibres, Inter-Alveolar Septa
Rupture of Alveolar Wall
Reduction in Nutrient or Oxygen supply to the Alveolar Wall
2. Broncholitis
Obstruction
Air Passes through the pores of Kohn
Adjacent Alveoli become ballooned (Collateral ventilation)
Loss of elasticity and permanent distension of Alveoli
Alveoli expand with difficulty for each Inspiration
Alveoli continue to increase in size
Dirofilaria Immitis
Rupture



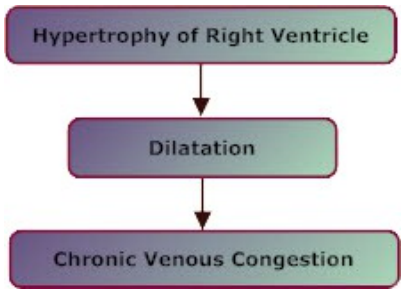
Gross pathology

- Size: Voluminous.
- Colour: Pale due to decreased blood circulation.
- Consistency: Pits on pressure easily.
- Surface: Indentation of ribs seen clearly.

Histopathology

- Alveoli: Size - Over distended and some rupture with confluence of neighbouring alveoli produces giant alveoli.
- Outline - Rounded.
- Walls size - Atrophied.

Sequelae



ANTHRACOSIS

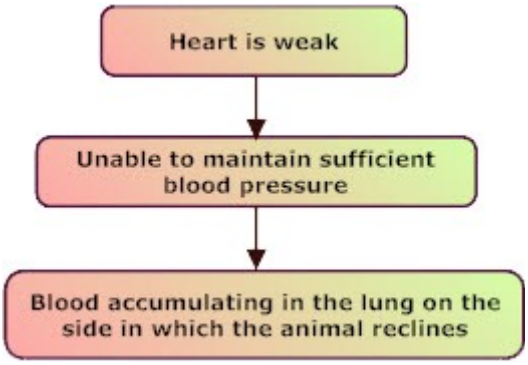
- Inhalation of carbon particles in animals living in highly polluted areas lead to the deposition of carbon particles in the lung. Such particles are found in the macrophages in the alveoli as well as around the bronchioles of the lung .

VASCULAR DISTURBANCES OF THE LUNG

HYPOSTATIC CONGESTION

Etiology : Animals in moribund or recumbent conditions.

Pathogenesis



PASSIVE CONGESTION

Occurrence: Occurs generally in older animals.

Etiology

- Cardiac lesion - Chronic pericarditis, myocardial weakness or lesions in the mitral valve.
- Extra-thoracic lesion – Bloat.
- General vascular dilatation – shock.

Gross pathology of lungs

- Colour: Dark red.
- Size: Larger.
- Consistency: Firm.
- Cut section: Blood oozes out.

Histopathology

- Capillaries in the alveolar wall are distended with RBCs
- Alveolar lumen: Contains RBCs / macrophages that have engulfed RBCs (heart failure cells).

Sequelae

- Edema of lung – Brown induration.
- Dyspnoea.
- Bronchopneumonia.
- Generalized hypoxia – fatty changes in liver and kidneys.

Active hyperemia: Commonly seen in pneumonia and acute general infectious diseases.

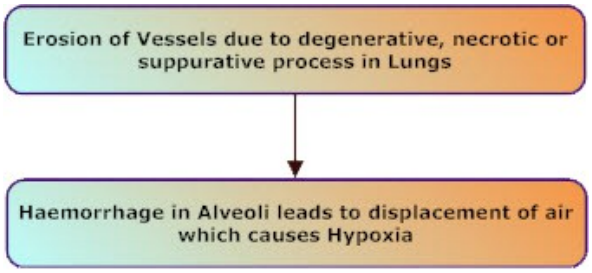
Pulmonary Haemorrhage

Aetiology

- Injury from a foreign body: Gunshot wounds, sharp objects piercing the thoracic cavity.
- Fracture of ribs : External injury, automobile accidents.
- Extreme over exertion as in young horses : Exercise induced pulmonary haemorrhage.
- Pathological
    - Erosion of blood vessels and rupture into a bronchus.
    - Growing abscess in the lung of cattle.
    - Extreme cardiac action as in death due to asphyxia.
    - Defect in coagulating ability – Hemophilia, purpura hemorrhagia or bracken fern poisoning.
- Biological
        - Bacteria disease – anthrax, pasteurellosis or leptospirosis.
        - Viral disease: Hog cholera, african swine fever.
        - Toxins: Uremia (injury to capillary endothelium occurs).

Signs: Haemoptysis (Blood in sputum due to pulmonary haemorrhage).

Pathogenesis



Gross pathology

- Hemorrhages may be in the form of petechiae, ecchymoses or hematocyst.

- Appearance: Mosaic pattern of red discolouration.
- Costo-caudal portion is haemorrhagic in horses.
- Trachea and Bronchi: contain foamy blood.

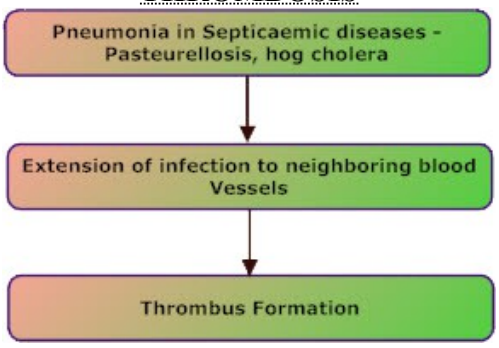
Histopathology

- RBCs in bronchi, bronchioles and alveoli.
- Abundant macrophages laden with hemosiderin (Siderophages)
- Interstitial tissue: Mild fibrosis.

Differential diagnosis

- Aspirated blood after the carotid artery and trachea have been cut at the time of of slaughter (Trachea and bronchial lumen will contain free/loosely attached blood clots.
- Epistaxsis.

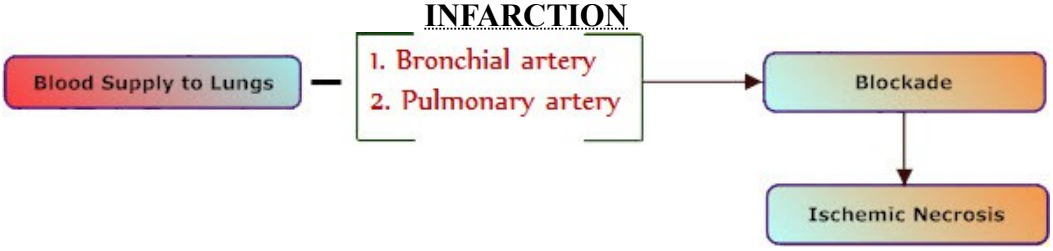
THROMBOSIS



EMBOLISM

- *Aetiology*
  - Emboli may emnate from
    - Thrombosis: Heart worm infection in dog, mesenteric veins in horse by *Strongylus vulgaris* and uterine vessels in metritis in cattle.
  - Pelvic veins – Mastitis in cattle.
  - Posterior venacava - extension of infection from hepatic abscess in cattle.
  - Ascarid larva.
  - Tumour cells – metastasis in malignant tumours.
  - Fat – due to fractures of large bones in man.
- *Sequelae*
  - Septic emboli cause pneumonic foci, lung abscess.
  - Thrombosis.
  - Parasitic cysts.

INFARCTION

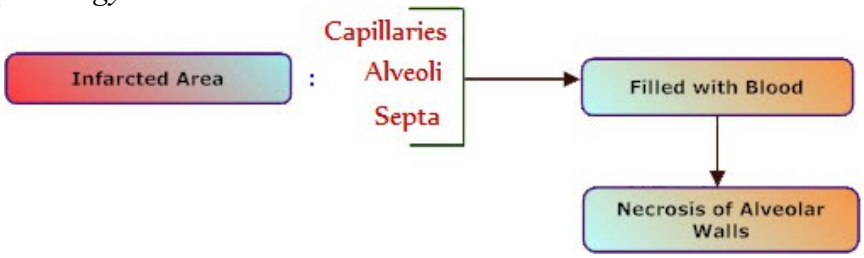


Gross pathology

- *Lung infarct*
  - Shape: Cone shaped
  - Apex: Thrombus or embolus
  - Base: At pleural surface
  - Colour: Dark red
  - Appearance: Solid looking

- Consistency: Firm
- Cut surface: Bulge initially and later stages depressed from surrounding surface

- *Histopathology*



OEDEMA

Characterised by excess fluid accumulation in the lung

Occurrence

- Common condition. , Usually proceeds to pneumonia.

Aetiology

- Nutritional - Hypoproteinemia.
- Inhalation of smoke.
- Chemicals: Inhalation of ammonia, chlorine, nitric oxide and phosgene.
- Bacterial toxins either exo or endotoxins; produced locally or elsewhere.
- Allergy
  - Vaccination with strain 19 against brucellosis.
  - Helminthic infection.
- Pathological
  - Acute anemia (Trypanosomiasis).
  - Passive congestion of lungs as in lesions of myocardium.
  - Lesions of mitral valve.
  - Mulberry heat disease of swine.
  - Shock.
- Therapeutic - Increasing venous pressure in pulmonary circulation due to over administration of intravenous fluids.
- Poisons - Alpha naphthyl thiourea (ANTU) – (increase capillary permeability).

Gross pathology

- Trachea or bronchi – contain froth due to churning action of tracheal air on exposure to protein containing fluid).
- Lung
  - Size: Enlarged.
  - Consistency: Firm.
  - Cut surface: Oedematous fluid drips – Clear, yellow frothy fluid.
  - Interstitial tissue – Oedematous.
  - Alveolar septa stand out prominently.

Histopathology

- Bronchi and alveolar lumen contain a pink stained homogeneous material proportional to the amount of protein present.

Sequelae

- The edematous fluid is a good medium for the growth of micro- organisms and hence pneumonia is frequent following pulmonary oedema.

INFLAMATION OF LUNGS

Pneumonia: Inflammation of lungs with copious exudates filling the alveoli

Introduction: Pneumonia is common in domestic animals especially sheep except in cats.

Routes of infection

- Through respiratory passage (bronchogenous) – Bacteria: *Staphylococcus, Streptococcus, E. coli, Corynebacterium pyogenes*; Fungus: *Aspergillus fumigatus*; Virus- Ranikhet disease virus in fowls, influenza virus in pigs.
- Blood (Hematogenous) – Bacteria – *Pasteurella, Salmonella*; parasitic larvae *(Strongylus vulgaris)*
- Through exterior:
    - Penetrating wounds – Contaminated bullets and knives
    - Through abdominal cavity: Foreign bodies penetrating rumen -----> Diaphragm ----> lung

Predisposing causes

- Recumbency for a considerable time -----> pulmonary edema
- Chronic under nutrition
- Malnutrition
- Severe hunger
- Fatigue
- Parasitism
- Long travel by train or ship
- Exposure to cold air
- Cardiac weakness -----> pulmonary edema
- Dipping in winter month
- Pathological: extension of infection from rhinitis/laryngitis

Priming

- by *Haemophilus suis,* Porcine pneumonia virus cause pneumonia
- *Pasteurella multocida* paves the way for pleuropneumonia like organisms and parainfluenza-3 infection
- Natural inhabitant fauna of upper respiratory tract flares up during stress/ viral infection
- Immune depression by Canine distemper leads to *Bordetella bronchiseptica* infection in dogs

Aetiology

- Physical – irritants
    - Solid – Dust, pollen or foreign bodies
    - Gas - smoke, hot and cold air
- Chemicals - Anaesthetics, war gases and medicinal agents
- Parasites
    - *Dictyocaulus viviparous* - cattle
    - Dictyocaulus filaria - Sheep and goat
    - *Protostrongylus rufescens*
    - *Ascaris lumbricoides var suum* – Pigs
    - *Metastrongylus apri*
- Bacteria
    - **Cattle:** *Pasteurella multocida, Pasteurella hemolytica, Staphylococcus spp, Streptococcus spp, Mycoplasma bovirhinis, Mycobacterium tuberculosis, Mycobacterium bovis, Mycobacterium avium, Acinomyces pyogenes and Actinobacillus lignieresi.*
    - **Sheep:** *Pasteurella multocida, Escherichia coli, Pasteurella hemolytica, Staphylococcus spp, Streptococcus spp, Mycobacterium ovis, Acinomyces pyogenes, Actinomyces ovis and Actinomyces pseudotuberculosis.*
    - **Horse:** *Actinomyces equi* and *Pseudomonas mallei*

- **Pig:** *Bordetella brochiseptica, Streptococcus spp, Hemophilus suis, Mycoplasma hyopneumonia and Acinomyces pyogenes.*
- **Dog:** *Bordetella brochiseptica, Escherichia coli, Staphylococcus spp, Streptococcus spp* and *Klebsiella pneumoniae.*
- **Cat:** *Escherichia coli* and *Pseudomonas aeruginosa.*
- **Rabbit:** *Pasteurella multocida, Actinomyces pseudotuberculosis and Pseudomonas aeruginosa.*
- **Rat:** *Mycoplasma pulmonis and Diplococcus pneumonia*
- **Poultry:** *Pasteurella multocida, Escherichia coli, Hemophilus paragallinarum, Mycoplasma galliseptitum and Mycobacterium avium.*

Virus

- Dogs: Canine distemper, canine herpesvirus, canine adenovirus1,2, parainfluenza, SV5, canine reovirus –1
- Cattle: Infectious bovine rhinotracheitis, bovine respiratory syncytial virus
- Sheep: Sheep pox
- Cats: Feline herpesvirus
- Horses, mule and donkeys: Equine influenza-A, equine herpesvirus 1-4,
- Poultry: Ranikhet disease

Chlamydia

- *Chlamydophila psittaci* – Parrots and other birds, man
- *Chlamydophila spp*. – Cattle, cats

Fungus

- *Aspergillus fumigatus*
- *Cocidiodes immitis*
- *Cryptococcus neoformans*
- *Blastomyces dermatidis*
- *Histoplasma capsulatum*
- *Nocardia spp*
- *Geotrichum candidum*

DEVELOPMENT OF PNEUMONIA (STAGES OF PNEUMONIA)

Stage of congestion

- Early stage in the developing of pneumonia
- *Course*
  - Depending on the irritant this may develop within a few minutes (chemical) to a few hours (infectious agents).
- *Gross pathology*
  - Colour: Active hyperemia/ congested
  - On section: Blood tinged fluid escapes (edema and congestion)
  - Size: Swollen
  - Density: Affected part float in water
- *Histopathology*
  - Alveolar – capillaries: Prominent and congested
  - Lumen: may contain fluid mixed with erythrocytes.

Stage of red hepatisation

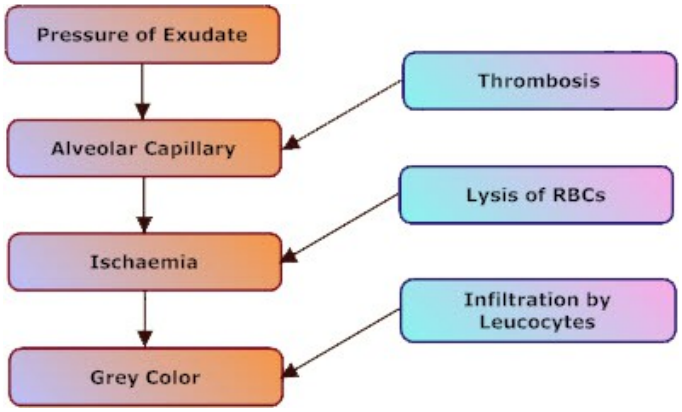
- *Course* : Develops in two days
- *Gross pathology*
  - Appearance: Affected portion of lung is quite conspicuous being readily discernible from the healthy area and resemble liver, hence called hepatisation.

- Margin: Distinct line of demarcation is found between the affected and normal part.
- Surface: pleura is thickened and reddish.
- Lung colour: Red
- Consistency: Consolidated.
- Density: Sink in water.
- Lumen size dilated.
- Pleural fluid: Quantity is increased.

- *Histopathology*
  - Alveolar lumen – exudate – fibrinous; contain RBCs, neutrophils and desquamated epithelial cells.
  - Septum – Widely separated.
  - Lymphatics - Dilated.
  - Peribronchial or perivascular lymphatics: Obstructed by fibrinous plugs.

Stage of grey hepatisation

- *Pathogenesis*



- *Gross pathology*
  - Appearance -Consolidated
  - Colour - Affected are greyish coloured
  - Density - Sinks in water
- *Histopathology*
  - Alveolar capillary: Congested or haemorrhagic.
  - Lumen: Reduction in erythrocytes or almost complete disappearance compared red hepatisation and fibrin strands pass form one alveolus to another through the pores of Kon.
  - Later the fibrin is liquefied and the nuclei of polymorphs become blurred and less distinct.

Stage of resolution

- *Pathogenesis* : Alveolar exudate is removed by liquefaction through lymphatic and veins.
- *Histopathology*
  - Alveolus: Lumen exudate – disappears and looks granular - Polymorphs – absent or a few degenerated.
  - Infiltration of macrophages occur.
- *Sequelae*
  - Lung returns to normal state of functional activity.
  - Death is due to hypoxia / cardiac failure.

CLASSIFICATION OF PNEUMONIA

Pneumonia is classfied as follows based on the extension of lesion and aetiology

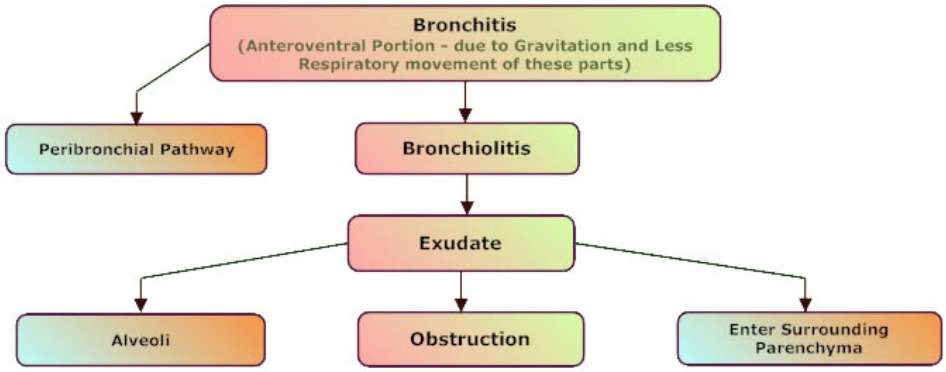
- Lobar pneumonia
- Bronchopneumonia
- Interstitial pneumonia
- Aspiration pneumonia
- Metastatic suppurative pneumonia
- Verminous pneumonia
- Mycotic pneumonia
- Pulmonary adenomatosis
- Maedi

- **LOBAR PNEUMONIA**

- Synonym : Croupous pneumonia
- Cause
- *Bacteria*
    - *Diplococcus pneumonia* – Man
    - *Pasteurella multocida* - Cattle sheep swine
    - Contagious pleuropneumonia - cattle and goat
- Pathogenesis
- Upon inhalation of bacteria bronchiolitis occurs.
- Bronchiolitis leads to obstruction of bronchiole and hence the flooding exudates infect the surrounding parenchyma.
- Through the terminal bronchiole alveolus may be affected.
- Through the pores of Kohn neighbouring alveoli are affected.
- Through the peribronchial pathway neighbouring alveoli are also affected.
- Spread of infection is rapid via blood and lymph which leads to thrombosis of pulmonary vessels
- Gross pathology
- Man: Whole lobe is affected
- Animals: Apical, cardiac, intermediate and anteroventral portion of diaphragmatic lobe is affected
    - Due to difference in age of lesions in different areas in the same animal, there are different colored areas
    - Lung appears marbled. Interlobular septa prominent and widened
- Histopathology
- Alveolus: Exudate – Fibrinous (croupous) - less in animals
- Inter lobular septa: Distended with plasma and fibrin

- **BRONCHO PNEUMONIA**

- Synonym : Lobular pneumonia or catarrhal pneumonia
- Occurrence : Bronchopneumonia is the commonest type of pneumonia found in animals
- Introduction
- Most varities of pneumonia in animal may start as lobular pneumonia but end up as a lobar variety
- All gradations may be met within the same animal
- Aetiology
- *Bacteria*
    - *Rhodococcus equi* – foal (may also be a complication of strangles)
    - *Salmonella cholera suis* – haematogenous route
- *Virus*
    - Parainfluenza –3 virus, adenovirus, reovirus, respiratory syncytial virus in calves and sheep
- *Bacteria and virus*
    - A combination of *Hemophilus suis* + swine influenza or *Pasteurella* + *Hemophilus suis* + hog cholera may cause pneumonia in pigs
-
-
- Pathogenesis



- Gross pathology
- Bronchi – contain exudate which may be haemorrhagic
- Pneumonic lesion: Distribution - patchy (Anterovental portion of lung)
- Part of lobe or lobule or several lobules may be involved
- Colour: Red
- Consistency: Firm
- Density: Sink in water
- Lymphatic: Prominent
- Lymph nodes: Size - Swollen
    - Colour – Haemorrhagic
- Histopathology
- Bronchial lumen contain catarrhal exudate or leucocytic exudate
- Alveolus: leucocytic infiltration
- Adjacent portion lesion: Compensatory emphysema
- Pleura over affected area: Exudate – fibrinous
- Sequelae
- If extensive area of lung is involved, hypoxia occurs and it leads to anoxia which causes cadiac failure / toxaemia and finally death.
- If pneumonic area is invaded by saprophytes, putrefactive changes lead to gangrene
- If the bacteria in the pneumonic area enter the blood, septicaemia occurs. Spread of infection to different parts of the body occurs. Usually meningitis and arthritis occurs.
- If pyogenic bacteria is the cause of pneumonia (*Corynebacterium equi* in foals and *Bordetella bronchiseptica* in dogs), suppuration of the affected area leads to abscess
- Complete resolution of bronchial exudates leads to atelectasis of the affected pulmonary tissue
- If the exudate in the alveoli undergoes incomplete resolution, fibrosis of lung and pleura (Organisation) occurs. The affected area looks like flesh **(Carnification)**
- **INTERSTITIAL PNEUMONIA**
- Definition
- In interstitial pneumonia, the inflammatory changes takesplace primarily in the alveolar wall and alveolar interstitium.
- Aetiology
- Respiratory viral diseases - e.g. Swine influenza, Feline pneumonitis
- Secondary as in septicaemic diseases – Erysipelas, Leptospirosis, endocarditis of right side, salmonellosis
- Psittacosis group of organisms
- Larval migration of ascarids and other intestinal parasites
-

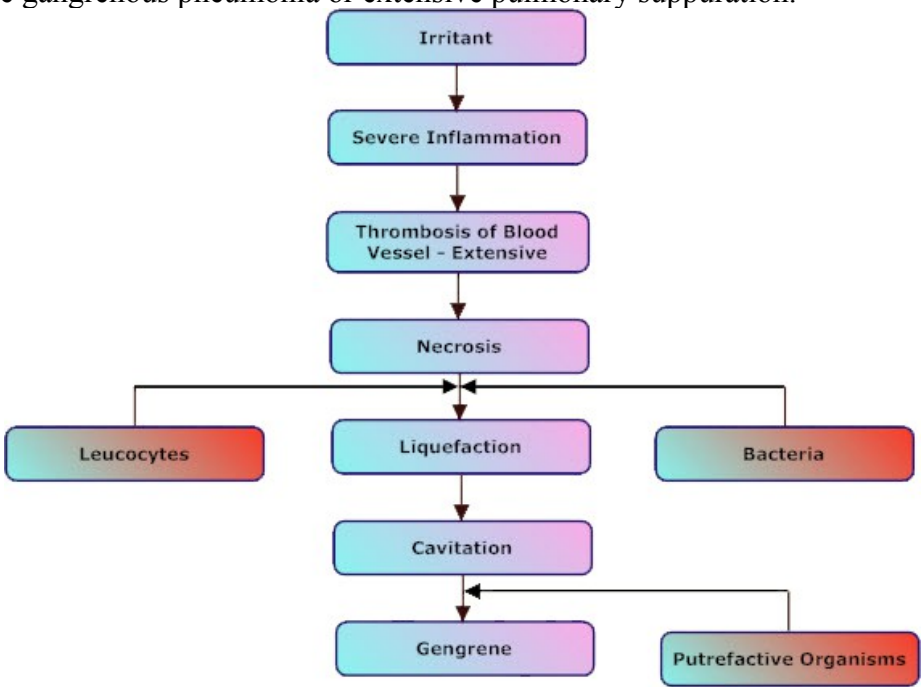
- Pathogenesis
- The condition may be acute or sub-acute in intensity
- Bacteria and parasitic larvae injure the capillary endothelium directly and make them more permeable. Hence fibrinous exudates occurs in the alveolar septa
- Viruses affect the alveolar epithelium and which proliferates and make them thick. Hence, the alveolar capillaries are devoid of oxygen supply. This hypoxia injures the capillary endothelium and makes them more permeable to produce edema in the alveolar walls
- Gross pathology
- Lungs
    - do not collapse when the thorax is opened
    - Colour may vary from red to pale gray or mottled
    - Pleural surface may show costal impressions
    - Cut surface has visible meaty appearance
    - Lack of visible exudation unless complicated by secondary bacterial infection
    - Consistency of lung is elastic or rubbery
    - Interlobular septa are widened
- Histopathology
- Alveolar walls and interlobular septa are thickened by
- exudate of either serous or fibrinous variety
- infiltration of interstitium by neutrophils, lymphocytes and macrophages
- proliferating fibroblasts
- proliferation of the alveolar epithelial cells
- formation of the hyaline membrane over the alveoli and alveolar ducts
- Alveolar epithelium
    - becomes cuboidal and prominent (called cells of tripier; Fetalisation or epitheliolisation)
    - become rounded and desquamated, forming macropages with phagocytic properties. In some cases giant cells also form by the fusion of macrophages or division of nuclei without the division of cytoplasm. Predominance of these cells gives rise to giant cell pneumonia
    - covered by hyaline membrane
    - perivascular areas are infiltrated with lymphocytes and macrophages
- Sequelae
- Complete resolution may occur
- If complete resolution does not occur, healing occurs by fibrosis. The organized tissue is like flesh - carnification
- Interstitial pneumonia is the seat of secondary bacterial infection and may lead to bronchopneumonia or bronchointerstitial pneumonia.

- **ASPIRATION PNEUMONIA**

- Synonym
- Foreign body pneumonia, dipping pneumonia, drenching pneumonia, mechanical pneumonia, inhalation pneumonia, necrotic pneumonia and gangrenous pneumonia
- Introduction: Aspiration pneumonia is a common and serious disease of farm animals
- Predisposing factors
- Drenching of animals when the tongue is drawn out or when the head is held high or when the animal is coughing or bellowing.
- Vagal paralysis
- Acute pharyngitis

- Tumours of pharyngeal region
- Oropharyngeal diverticula
- Cleft palate
- Encephalitis
- Etiology
- Physiological: Aspiration of milk or gruel in pail fed calves.
- Feeding on fluid feeds in inadequate troughing, inhalation occurring in the struggle for food.
- Animals fed with dusty feeds inhale many dust particles and bacteria could be readily isolated form the lung. This rarely results in the development of pneumonia as much of the dust filtered out in the bronchial tree and does not reach the alveoli.
- Regurgitation in weak animals
- Vomiting may be followed by aspiration
- Most cases occur after careless drenching or passage of a stomach tube during the treatment for other illness
- Animals suffering form paralysis or obstruction of the larynx, pharynx or esophagus may aspirate food or water when attempting to swallow.
- Foreign bodies, food material or medicines enter the respiratory tract as a result of some disturbance of deglutition as in paralysis of the pharynx (parturient paresis of cattle) or when administered by the use of force.
- Aspiration of septic material - Rupture of pharyngeal abscess during palpation of the pharynx or passage of a nasal tube may cause sudden aspiration of infective material
- Penetration of sharp foreign bodies through rumen and reticulum.
- Inhalation of irritant gases or smoke is an infrequent cause. Inhalation of vapour will cause atelectasis, pulmonary oedema and congestion.
- Hematogenous – from gangrenous lesion elsewhere in the body eg. Gangrenous metritis or mastitis
- Direct infection by *Fusobacterium necrophorum*
- Dipping pneumonia
- It is common 3-7 days after dipping in case of weak sheeps. Sheep may inhale the dipping fluid resulting in purulent pneumonia.
- After dipping in arsenic tanks contaminated with *Actinobacillus lingniresi*, inhalation pneumonia results.
- Dipping of weak sheep by keeping their heads under dipping fluid for too long also result in inhalation of fluid
- Drenching pneumonia
- Wrong administration of any drench allowing it to go down the wind pipe e.g. carbon tetrachloride
- Pathogenesis
- An uncommon but important effect of aspiration with lodgment of food at the glottis leads to asphyxia or sudden death due to vagal inhibition and cessation of respiration and circulation.
- If large quantities of fluid are aspirated after faulty passage of a stomach tube into the trachea, death may be almost instantaneous. However with aspiration of smaller quantities, the outcome may depend on the composition of the aspirated material.
- Absorption from the lungs is very rapid and soluble substances such as chloral hydrate and magnesium sulfate exert their systemic pharmacological effects very rapidly.

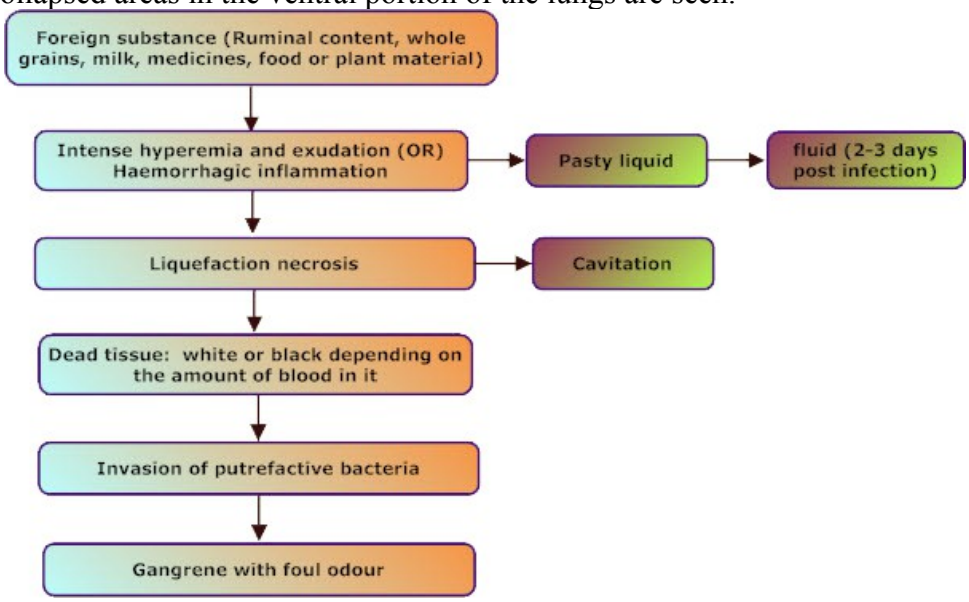
- With insoluble substances and vomitus the more common occurrence is the development of pneumonia with profound toxaemia which is usually fatal in 48-72 hrs.
- The severity of aspiration pneumonia depends largely upon the bacteria which are introduced, although in animals the infection is usually mixed causing in many cases an acute gangrenous pneumonia or extensive pulmonary suppuration.



- Signs
- Pulse – accelerated
- Polypnoea - Labored breathing
- Purulent nasal discharge which in some cases may be reddish brown or green
- Cough
- Occasionally evidence of the aspirated material can be seen in the nasal discharge or in expectorated material e.g. Oil droplets
- Rales
- Long breath sounds on ventral half of lung with moist crackles
- On auscultation, in early condition, fluid sounds may be heard over one or both sides of the chest followed by wheezing sounds and localized pleuritic friction rubs
- Putrid or sweetish odour on breath is characteristic of gangrene, the intensity increase as the disease progresses.
- Acute case
- Sudden onset of marked dyspnoea
- Anxiety
- Distress
- Death may occur within a few minutes.
- Animals that survive the acute stage of aspiration pneumonia shows emaciation, chronic cough and dull sounds at auscultation and percussion.
- Gross pathology
- If the sheep dies rapidly, it may have frothy mucus in the airway
- Tracheo-bronchial mucosa are hyperemic
- Lungs
    - The pneumonia is usually in the anteroventral parts of the lungs and may be unilateral or bilateral with foul smelling exudates

- The Pneumonic areas tend to be cone shaped with the base towards the pleura
- The affected parts are greenish or black in colour and consolidated.
- Occasionally with suppuration and necrosis, the focus becomes soft or liquefied and has reddish brown appearance with foul odour.
- Cavitation may occur in some cases.
- A zone of congestion demarkates the affected and unaffected area.
- Fibrinous pleuritis with pleural exudation may also be noticed.

- Chronic cases
- Serosanguineous fluid in the pleural or pericardial cavities and necrotic, haemorrhagic and collapsed areas in the ventral portion of the lungs are seen.



- Histopathology : Lesion is similar to that of bronchopneumonia
- Diagnosis
- History: Drenching within the previous 1-3 days
- Pneumonia of anteroventral lobes following recent drenching
- Differential diagnosis
- Tubercular pneumonia
- Chronic contagious bovine pleuropneumonia
- Pleurisy and pericarditis following traumatic reticulitis
-
- Sequelae
- The presence of saprophytic and putrefactive organism in the aspirated material leads to gangrene and fatal consequence
- Prognosis: poor
- Mortality: High
- **METASTATIC SUPPURATIVE PNEUMONIA**

- Synonym : Embolic pneumonia
- Route of infection
- Haematogenous - Embolic deposition of pyogenic organism from lesions elsewhere in the body *viz* – suppurative metritis or mastitis, navel ill and valvular endocarditis.
- Gross pathology
- Multifocal suppurtive foci evenly scattered through all the lobes of both lungs- predominently the diaphragmatic lobes

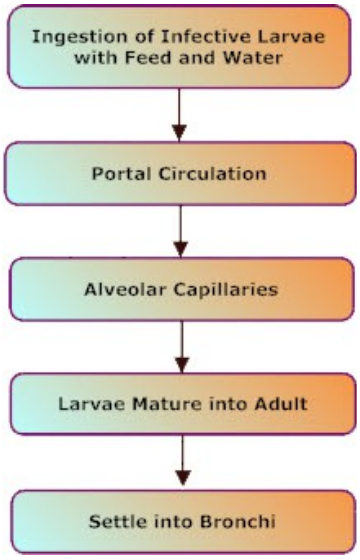
- Greatest numbers of foci are concentrated near the pleural surface (this area contains the largest proportion of small arteries and arterioles in which an embolus may be lodged).
- Emboli may induce a granuloma or an abscess. The abscess wall thickness depends on duration. It may later becomes organized and produce a scar.
- Histopathology : Areas of suppuration are usually concentrated near the blood vessels

- **VERMINOUS PNEUMONIA**

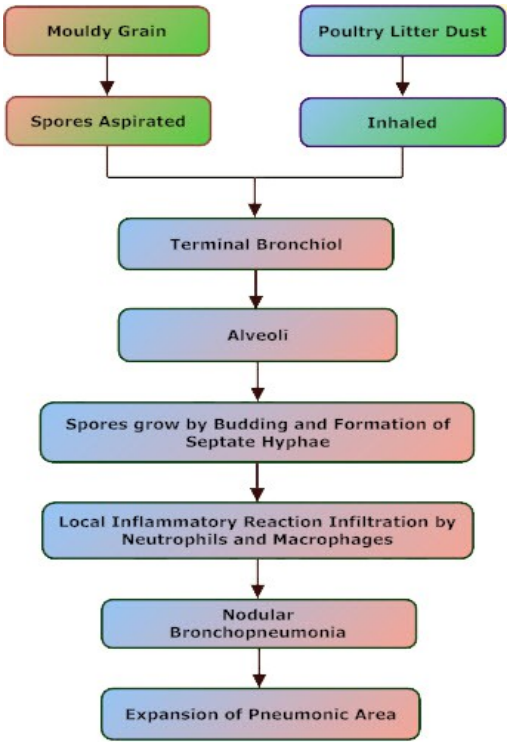
- Etiology

Habitat	Parasite	Host
	Dictyocaulus viviparous	Cattle
	Dictyocaulus filaria and *Protostrongylus rufescens*	Sheep
Bronchi	*Dictyocaulus arnifieldi*	Horse
	Metastrongylus apri	Swine
	Aleurostrongylus abstrasus	Cat
Bronchi and cyst in lungs	*Paragonimus westermanii* (Fluke)	Cattle, sheep, dog, man
Pulmonary arteries	*Angiostrongylus vasorum*	Dog
Alveoli and blood vessels	*Mullerius minutissimus (capillaris)*	Sheep, goat
Lung	*Ascaris* larvae	Swine
Lung(aberrant location)	Liver fluke	Ruminants

- Life cycle



- Histopathology
- Lesions resemble bronchopneumonia
- Bronchial lumen may be occluded with worm/embryonated eggs, mucin, desquamated epithelial cells and inflammatory cells like neutrophils, eosinophils, lymphocytes and macrophages.
- Obstruction of bronchial lumen may lead to bronchial ectasia, atelectasis and emphysema
- Bronchial epithelium – Thick and hyperplastic
- Alveolar walls are thickened and show evidence of necrotic foci surrounded by neutrophils, eosinophils, macrophages and a few giant cells
- In heavy infections, haemorrhages along with ova may be noticed in the bronchial lumen
- *Mullerius capillaris*
- Grossly, lungs contain multifocal, wedge shaped nodules mostly in the diaphragmatic lobe.
- Microscopically, granuloma develops around worms which die and lead to calcification.
- *Paragonimus westermanii*
- Usually found in pairs in inflammatory cysts in lungs
- Cysts have communication with the bronchi for the passage of eggs
- Cysts without such outlets become organized.
- Ascaris larvae
- Diffuse interstitial pneumonia with haemorrhagic foci, atelectasis, interlobular oedema and emphysema are seen
- Exudate is rich in eosinophils.
- Liver flukes
- Produce nodules with fibrous capsule
- Parasite dies as it cannot thrive in a strange locality
- Sequelae : Secondary bacterial infection
- **MYCOTIC PNEUMONIA**
- Etiology
- Oppurtunistic fungi – *Aspergillus fumigatus* is the most common cause
- Systemic (deep) mycosis: *Blastomyces dermatitides, Coccidiodes immitis, Cryptococcus neoformans* and *Histoplasma capsulatum* may cause pneumonia
- Pneumonia caused by *Aspergillus fumigatus*
- Distribution: Ubiquitous
- Animals affected: Frequently affects birds (Broooder pneumonia). Also affects immune suppressed or animals undergoing prolonged antibiotic therapy.
- Pathogenesis of brooder pneumonia
- Age of affected birds: Brooders
- Route of infection - Respiratory tract



- Gross pathology
- Larynx, trachea and bronchi contain grayish white or greenish thick cheesy material
- Lungs may show nodular or miliary type lesions
- Air sacs – Thickened and may show growth of fungus as a saucer shaped lesion
- Diagnosis
- Microscopic examination of crushed nodule reveals dichotomously branching septate hyphae.
- Histopathology
- Granulomatous inflammation characterized by central caseous area with septate hyphae surrounded by infiltrating cells that include macrophages, epithelioid cells, foreign body giant cells.
- Capsule is formed by fibrous tissue
- In nonprogressive lesion, hyphae are very short
- Lesion resembles that caused by actinomyces – the asteroid body

- **PULMONARY ADENOMATOSIS**

- Definition : Alveoli have adenomatous or glandular appearance
- Occurrence: Sporadic or as outbreak
- Species affected
- Sheep (India) cattle, pigs, horse and man
- In sheep, it is called Jaagsiekte which means driving sickness
- Etiology
- Food allergy in cattle
- Chemicals - Nitrogen peroxide from corn silos in man causes silo filler's disease
- Bacteria –Mycoplasma; Pleuropneumonia like organisms in pigs and horses
- Virus in sheep
- Mouldy foods in cattle
- Pathogenesis
- Incubation period - 7 months
- Death occurs 2-3 months after the on set of signs

- when driven coughing in produced
- Gross pathology
- *Lung*
    - Appearance – bulky and nodular; Discrete small nodules coalesce to form bigger nodules
    - Weight - heavy
    - Surface - imprints of ribs
    - Unaffected lung – emphysematous
    - Bronchial lymph nodes - metastasis seen
- Histopathology
- *Alveoli*
    - Lumen – seldom contain exudates
    - Epithelium: desquamated or epithelium undergoes hyperplasia and hypertrophy (lined by cuboidal or columnar epithelium) to produce papillary projections
- *Bronchi*
    - Epithelium shows papillary projection into the lumen
    - Lumen – contain desquamated epithelium and alveolar septal cells
- *Stroma*
    - Infiltrated with lymphocytes and plasma cells
- Sequelae
- Thickening of alveolar epithelium prevents normal exchange of gases and hence hypoxia and dyspnoea occurs
- Decrease in pulmonary capillary bed causes dilatation of right side of heart
- Fibrosis of lung
- Cattle: Course is more acute so fibrosis is not usually observed
- **MAEDI**
- Meaning : Dyspnoea in Icelandic Languge
- Occurrence : Iceland, Holland and North America. Maedi like disease like disease is reported in India
- Species Affected: Sheep
- Age Susceptibility: Older (over 2 years)
- Etiology: Virus
- Pathogenesis
- Incubation period: Long – 3 years.
- Contagious disease
- Occurs as out break
- Signs: Dyspnoea, inanition and progressive debility for 3 –6 months
- Gross pathology
- Lungs
    - Lesion distribution: Uniform
    - Collapsibility: Do not collapse when thorax is opened
    - Colour: Greenish blue
    - Cut surface: Dry
    - Size: Large
    - Weight: Heavier
- Lymph node: Size: Swollen
- Histopathology

- Lesion resembles chronic interstitial pneumonia
- Alveoli:
  - Lumen: Contains desquamated epithelial cells
  - Walls: Thickened.
- Infiltrated with lymphocytes, macrophages and short reticular cells
- Macrophages cytoplasm contain 1-3μ diameter bluish grey inclusion bodies (Stainable by Giemsa stain)
- Bronchi:
  - Epithelium: hypertrophied and hyperplastic
  - Walls – smooth muscles increased (false appearance of fibrosis)
- Peribronchial and perivascular area markedly infiltrated with lymphocytes
- Lymph nodes are hyperplastic
- Sequelae: No healing occurs

Disease	Jaagsiekte	Maedi
Course	Shorter	Longer
Distribution of Lesion	Focal	Diffuse
Alveoli	Adenomatous	Not Adenomatus
Inclusions	Absent	Present
Involvment of Lymph nodes	Not affected	Lymph adenitis present

- Tumours of Lung
- Primary tumours o Lung are rare.
- The common tumour is lymphocytoma
-
- **PLEURA**
- Vascular disturbance
- Congestion
- Edema (hydrothorax)
- Abnormal contents
- Air in pleural cavity: Pneumothorax
- Pus in pleural cavity: Empyema
- Inflammation: Pleuritis
- Pleural congestion
- *Cause*
  - Passive congestion
  - Acute poisoning
- Abnormal contents
- Hydrothorax
- *Definition*

- Excessive fluid accumulation in thoracic cavity

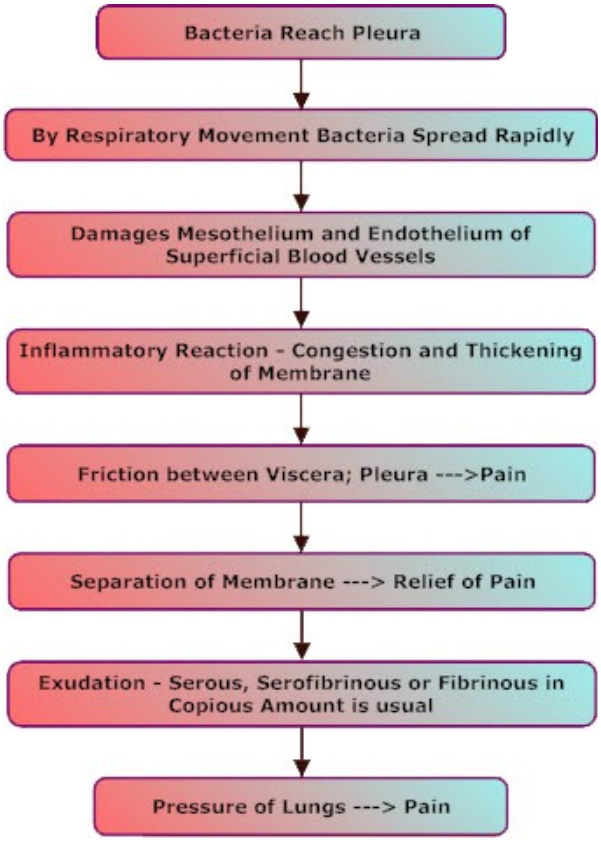
- *Cause*
  - Passive congestion
    - Acute poisoning
    - General dropsy conditions
    - Chronic cardiac diseases
    - Interference with lymphatic circulation – tumours and lymphadenitis
- *Gross pathology*: Pleural cavity contains serous fluid
- Pneumothorax
- *Definition* : Presence of air or gas in the pleural cavity
- *Etiology*
  - Internal - Rupture of bullae in lung
  - External - Chest wall – by piercing sharp objects
  - Physical injury to the chest wall
  - Bacterial – gas forming organisms introduced through a septic wound.
  - Pathological – Rupture of lung tissue as in tuberculosis, infarct, lung abscess and emphysema
- *Gross pathology*
  - Pleural cavity: Contain gas or pus mixed with gas
  - Lungs: Collapsed
- *Sequelae*
  - Pneumothorax
  - Pressure on lungs - atelectasis
  - If air has entered through a small wound and pathogenic bacterial are absent the wound usually heals up and the imprisoned air is rapidly absorbed.
- Haemothorax
- *Definition* : Accumulation of blood in pleural cavity
- *Etiology*
  - Physical – trauma – fracture of ribs
  - Pathological – rupture of aneurysm or neoplasm

- **PLEURITIS**

- Synonym: Pleurisy
- Definition: Inflammation of pleura
- Occurrence: Common is animals
- Predisposing causes
- Fatigue
- Over exertion
- Sudden changes of temperature
- Prolonged transport
- Etiology :Mostly secondary to pneumonia

S	Route of Infection	Examples
1	By direct extension frm underlying lungs, mediastinal glands and oesophagus	Abscess / Gangrene of lungs and pnemonia; Sharp bones, pins may penetrate and convey bacteria
2	Introduction through thoracic wall	Trauma by knives, bullets and fracture of ribs
3	Introduction form rumen	Via reticulum and diaphragm as in traumatic reticuloperitonitis
4	Blood Stream	Septicaemic Diseases
5	Lymphatic Infection	Bacterial : Tuberculosis, swine erysipelas, contagious bovine pleuropneumonia, haemorrhagic septicaemia, black quarter, Staphylococcus, Streptococcus (Strangles) HS Viral: Equine Influenza

- Pathogenesis



- Gross pathology

- Pleural exudate: Serous, serofibrinous or purulent (Empyema)
- Amount: copious – usual
- Sequelae
- Pleural exudate if serous absorbed
- Serofibrinous or fibrinous exudates is organized and leads to adhesions
- Entry of saprophytes along with foreign bodies causes gangrenous pleurisy
- Acute pleurisy
- *Etiology*
    - Pathological – Extension from lungs directly to pleura or through lymphatics
    - Bacteria: Pyogenic organisms (suppurative or purulent pleuritis- EMPYEMA)
- *Gross pathology*
    - Usually unilateral (Bilateral in horse owing to the communication between pleural cavities)
    - Pleura
        - Colour - Hyperaemic / haemorrhagic
        - Appearance- Dull
        - Moisture - Dry
        - Contents – Exudate is serous, serofibrinous or fibrinous; the exudates may be scanty or dry if fibrinous; several pints in dogs; several gallons is horse.
- *Sequelae*
    - Serous pleurisy – Resolution
    - Serofibrinous pleurisy – Resolution if fibrin is in small quantity
    - Exudates - Organized - granulation tissue - adhesion between parietal and visceral pleura
    - Emphyema - Absorption of fluid - Exudate inspissated - Calcification sometimes occur
    - Fluid accumulation -Extensive pulmonary collapse - Death in horse
- Chronic pleurisy
- *Etiology*
    - Pathological: Acute pleurisy leads to chronic pleurisy
    - Bacterial: Tuberculosis, actinobacillosis, contagious bovine pleuropneumonia and contagious caprine pleuropneumonia.
- *Gross pathology*
    - Pleura – adhesion may be local or diffuse
- *Histopathology*
    - Pleura – Thickness- thick due to fibrous tissue proliferation
    - In case of contagious bovine and caprine pleuropneumonia, the interlobular septa are thickened and the alveoli are infiltrated with mononuclear cells and giant cells.

- **TUMOURS OF PLEURA**

- Occurrence: Rare
- Primary: Mesothelioma - malignant tumours may occur
- Secondary - Metastasis of melanoma may happen

- **AIR SACCULITIS**

- Definition: Inflammation of the air sacs is called air sacculitis
- Species affected: Avian – Commercial chicken
- Etiology
- Mycoplasma gallisepticum
- Escherichia coli

- Gross pathology
- Air sacs
    - Pin point pale foci seen in early change
    - Cloudy –Mild inflammation
    - Thick – Moderate
    - Thick with whitish cheesy exudate – severe
    - Thick with yellowish cheesy material indicate complicated infection
- Histopathology
- The air sacs are thickened with congestion, edema, fibrinous exudates and infiltrated with lymphocytes, macrophages and giant cells.

- **NEOPLASMS**

- Common tumours of the lung include adenocarcinoma and lymphosarcoma in animals and Marek's disease in chicken.

- **CARDIOVASCULAR SYSTEM**

- **HEART**
- The heart is located in the thoracic cavity between the lungs attached to mediastinum
- Function: The heart acts as the pumping station and pumps blood to different parts of the body.
- Compensation
- Compensation is the ability of the heart to adapt to varying physiological needs and pathological abnormalities.
- e.g: whenever there is resistance to blood flow as in case of pulmonary fibrosis or chronic nephritis, the heart has to contract forcibly to force the blood through the blood vessels.
- Hence the heart muscles have to do more function and become hypertrophied. The hypertrophy of cardiac musculature is a compensatory mechanism.
- Decompensation
- When the heart is unable to cope up with the demands and becomes fatigued and fails, the state of the heart is termed decompensation.
- The heart is unable to compensate for the ever-increasing workload. The decompensation is a gradual process.
- The decompensated heart has dilated ventricles.
- Causes of heart failure
    - Increased resistance to outflow: it occurs in case of hypertension, narrowing of valvular orifice, thrombosis and arteriosclerosis.
    - Alteration in the venous return: when the venous return is high or low continuously, the heart may fail at the end.

- Impaired cardiac contraction: this occurs when the myocardial contractility is lost as in the case of myocardial necrosis due to coronary vascular insufficiency

- **ANOMALIES**

- **AURICULAR DEFECT**

- Patent foramen ovale
- Patent foramen ovale has been met with in calves.
- In this condition the foramen ovale which communicates the right auricle with the left during foetal life becomes persistent even after birth.
- Hence, the blood flows from right auricle to left auricle i.e the unoxygenated blood is pumped through left ventricle through out the body.
- This leads to blue babies in human beings. Paradoxical embolism and hypertrophy of the right ventricle are the sequelae of this condition.

- **VENTRICULAR DE FECTS**

- Interventricular septal foramina
- Interventricular septal foramina below 5 mm are of no consequence. But when it exceeds 5 mm functional changes are observed in the ventricles.
- As the blood flows from left ventricle to right ventricle during systole of left ventricle, greater flow of blood in to the right ventricle occurs and so the right ventricle has to contract more forcibly and so hypertrophy of the right ventricle occurs.
- Endocardial fibroelastosis
- This condition is reported in dogs, cattle and cats.
- There is thickening of elastic fibres in the subendocardial connective tissue. Death occurs soon after birth of young ones.
-

- **DEFECTS IN BLOOD VESSELS**

- Patent ductus arteriosus
- The shunt which connects the pulmonary artery with the aorta should obliterate within a few weeks after birth.
- Sometimes the shunt may be patent and hence blood might enter from aorta in to the pulmonary artery causing increased pressure in the pulmonary artery.
- This leads to hypertrophy of right ventricle.
- The unoxygenated blood from pulmonary artery might also enter aorta causing mixing of oxygenated blood and hence the affected animals may be cyanotic.
- Persistence of the right aortic arch
- This is seen mostly in dogs and rare in bovines.
- The ductus arteriosus of the right aortic arch encircles and constricts the oesophagus. Hence, the proximal portion of oesophagus dilates.
- The affected pups vomit immediately after taking food.
- Coarctation of the aorta
- This is narrowing of the lumen of the aorta.
- It may occur close to the heart or between the origin of the common brachiocephalic artery and ductus arteriosus.
- Because of the narrowing of the aorta there is resistance to the flow of blood. This leads to hypertrophy of the left ventricle.
- Transposition of the aorta

- In this condition, the aorta arises from right ventricle or from both the ventricle. This is incompatible with life.
- Subaortic stenosis
- This is seen in case of Alsatians and boxer breeds of dogs. In this condition, a ring of fibrous tissue occurs just below the cusps of aortic valves.
- This causes stenosis resulting in hypertrophy of the left ventricle and finally its dilatation.
- Pulmonic stenosis
- In this condition the pulmonary valves fuse together resulting in stenosis.
- This results in hypertrophy of the right ventricle.
- Congenital aneurysm of the aorta and pulmonary artery
- Focal dilatation of the aorta or pulmonary artery causes pressure atrophy of the neighbouring structures.
- Fatal haemorrhage might also result from thinning and rupture of the aneurysms.
- **MULTIPLE DEFECTS IN THE HEART**
- Tetralogy of Fallot
- This refers to four defects in the heart.
- They are
    - Interventricular septal defect
    - Dextraposed aorta
    - Stenosis of pulmonary valves and
    - Hypertrophy of right ventricle.
- The affected animals are stunted and their mucous membranes are cyanotic. In human beings, this condition also gives rise to blue babies.
- Other defects of the heart
- *Acardia*
    - Acardia is a condition in which there is complete absence of heart. This condition is incompatible with life.
- *Diplocardia* : Diplocardia is a condition in which two hearts are present.
- *Ectopia cardis*
    - In ectopia cardis, the heart is found outside the thorax usually in the neck region or abdominal cavity.
    - This condition has been reported in cattle. This is not always fatal.
- **PERICARDIUM**
- Haemorrhages on pericardium
- Haemorrhhage may be in the form of petechiae, ecchymoses or extravasation.
- Causes of petechiae
- Shock
- Toxaemia caused by toxins of bacteria and viruses
- Hypoxaemia
- Purpura haemorrhagica of horses
- Causes of ecchymoses and extravasations: Sweet clover poisoning
- Abnormal contents of pericardium : Pericardial sac may contain excess serous fluid, blood, pus or gas.
- Hydropericardium
- *Definition* : Hydropericardium means excess of serous fluid in pericardial sac.
- *Causes*
    - Cachexic diseases

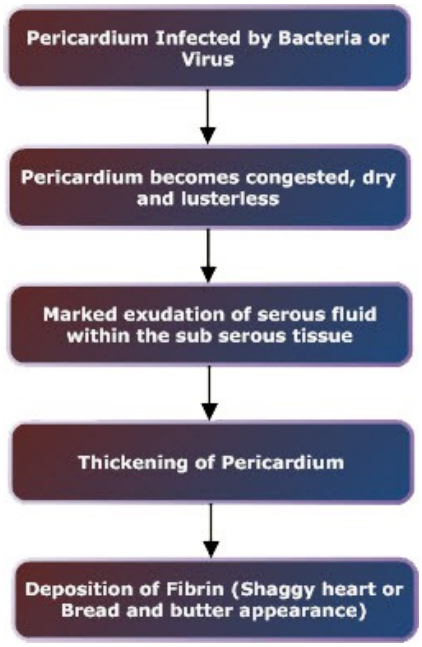
- Congestive heart failure- leads to increased pressure in coronary veins and capillaries
- Liver insufficiency leads to stasis of portal circulation, hypoproteinaemia
- Renal disease
- Chronic stomach worm infection leading to hypoproteinaemia
- Anaemia especially haemolytic
- Damage to capillary endothelium as occurs in
  - Many infections due to toxins
  - Anoxic conditions
- Tumors in pericardium or myocardium
- Salt toxicity
- Hydropericardium syndrome in chicken

- *Gross appearance*
  - The fluid is straw-coloured and clear. In infections, the fluid may contain floccules.
  - This is due to damage of the capillary endothelium and much protein flows out into the exudates.
  - If the fluid in the pericardial sac persists for a long time, it may become turbid and organized giving a shaggy (bun-butter) appearance to the pericardium and epicardium.
- Hemopericardium
- *Definition* : Hemopericardium means accumulation of blood in the pericardial sac.
- *Causes*
  - Trauma to heart
  - Rupture of heart, aorta or coronary artery
- *Gross appearance*
  - If the blood clot completely encloses the heart, the condition is known as cardiac tamponade.
- Pyopericardium
- *Definition:* Pyopericardium means pus in the pericardial sac
-
- *Causes*
  - Rupture of myocardial abscess , Purulent pericarditis
  - Tuberculosis
- *Gross appearance* : Pericardial fluid is turbid
- Pneumopericardium
- *Definition* : Pneumopericardium means accumulation of gas in the pericardial sac
- *Causes*
  - Production of gas by gas producing organism which may enter the pericardium with a penetrating body.
  - Gas may escapes into the pericardium in traumatic reticulitis
  - Gas may enter into the pericardium often a lesion involving pericardium and lungs breaks down
  - Gas may enter from outside in compound fracture of ribs.

- **PERICARDITIS**

- Definition : Inflammation of pericardium is called pericarditis.
- Incidence : Pericarditis is a common condition in animals but rare in dogs.
- Causes
- *Chicken* : Escherichia coli infection and salmonellosis.
- *Dogs*

    - Leptospirosis and as a complication of distemper produced by secondary bacterial invaders.
- *Cattle* : Pasteurellosis, coliform infection of new born and tuberculosis
- *Sheep* : Pasteurellosis and Salmonellosis
- *Swine*
    - Pasteurellosis, Streptococcal infections, Salmonellosis, Glassers disease caused by *Hemophilus suis*, Swine erysipelas, pneumonia caused by Mycoplasma spp. and hog cholera
- *Horses* : Streptococcal infections
- Routes of infection
- Haematogenous: Occurs in septicaemic and other specific diseases
- Lymphogenous: From the inflammatory processes of neighbouring tissues – myocardium, pleura, bronchial or mediastinal lymph nodes caused by foreign bodies entering through reticulum.
- Trauma as in traumatic pericarditis and bullet wounds
- **TYPES OF PERICARDITIS**
- Based on the lesion or exudates pericarditis is classified as
    - Fibrinous pericarditis
    - Suppurative pericarditis
    - Uric acid pericarditis
- Fibrinous pericarditis
- *Pathogenesis*



- *Gross appearance*
    - Initially the pericardium is congested, dry and lusterless.
    - Later becomes thickened with fibrin.
    - Pericardial sac may contain increased serous fluid or have fibrinous exudate.
    - The deposition of fibrin on the pericardium and into the sac gives the appearance of bread and butter - Such a heart is also called shaggy heart.
- *Microscopic features*
    - Desquamation or proliferation of mesothelial cells

    - Subserosa is infiltrated with neutrophils, lymphocytes, plasma cell and macrophages
- *Sequelae*
    - If the cause is removed, complete resolution occurs
    - In serve infections, adhesions develop with organization of fibrinous exudate and the heart finally fails
    - Occassionally caseation or calcification of the exudates may occur.
- Suppurative pericarditis
- *Definition* : Inflammation of pericardium with accumulation of pus.
- *Causes*
    - Salmonellosis in poultry
    - Traumatic reticulopericarditis in cattle
    - Secondary to suppurative pleuritis and bronchopneumonia
- *Clinical pathology*
    - Blood shows severe leucocytosis with shift to left in traumatic reticulopericarditis in cattle
    - Pericardial fluid contains more number of leucocytes
- *Gross appearance*
    - Pericardial sac is filled with pus
    - Pericardium is thickened with fibrin on both the surfaces
- *Microscopic appearance*
    - Pericardium is thickened with fibrin and leucocytic infiltration especially of neutrophils.
- *Sequelae*
    - Resolution is impossible
    - Toxins from exudate causes toxaemia and death
    - Organization of exudate leads to adhesions between pericardium and epicardium of heart. The condition is called cor rugosum or shaggy heart.
    - When the exudates and adhesions compress the heart (constrictive pericarditis), initially hypertrophy of myocardium occurs. But later diastolic filing and systole is hampered. The exudates also compress the great veins near the heart and hence blood flow into the heart is obstructed. This leads to chronic venous congestion and the heart fails finally. The associated lesions are nutmeg liver, cardiac cirrhosis, splenomegaly, ascites, hydrothorax and deposits of fibrin on the liver and spleen(sugar icing)
- Uric acid pericarditis
    - In fowls suffering from visceral gout, the urate irritates pericardium and cause pericarditis.
- *Gross appearance*
    - The chalky white fine granules deposited on the pericardium gives the appearance of fine frost.
- *Microscopic appearance*
    - Pericardium contains needle shaped urate crystals surrounded by granulation tissue.
- **EPICARDIUM**
- Serous Atrophy of Epicardial Fat
- *Definition*
    - Serous atrophy means the fat present in the grooves of heart becomes transformed into a gelatinous mass cause cachectic disease.
    -

- *Gross appearance*
    - The fat is lost and in its place edematous fluid accumulates
    - The interstitial tissue of heart is also edematous
    - White foci of necrosis may be seen in the lesion.
- Epicardial Haemorrhage
- *Etiology*
    - Bacteria – *Pasteurella*
    - Virus – Ranikhet disease
    - Pathological - Toxaemia, septicaemia and asphyxia
-
- **PATHOLOGY OF MYOCARDIUM**
- Pathology of myocardium includes
    - Hypertrophy
    - Dilatation
    - Cardiac failure
    - Degeneration
    - Necrosis
    - Circulatory disturbances
    - Inflammation
    - Neoplasms
    - **HYPERTROPHY**
    - Definition
- Hypertrophy of heart muscle means increase in size of the individual myocardial fibres.
    - Occurrence
- Left side of heart is more often affected
- Ventricles suffer more frequently
    - Physiological hypertrophy
- The ventricles become thicker due to greater strain on heart as in race horses and grey hounds
    - Pathological hypertrophy -Etiology
- *Intracardial hindrances*
    - Intracardial hindrance may be due to stenosis or insufficiency of heart valves
    - Stenosis or insufficiency of aortic valves leads to hypertrophy of left ventricle.
    - Stenosis or insufficiency of mitral valve leads to hypertrophy of left auricle.
    - Both the lesions lead to brown induration of lungs which ultimately causes hypertrophy of right ventricle
    - Stenosis or insufficiency of valves of pulmonary artery leads to hypertrophy of right ventricle.
- *Extracardial hindrances* - Etiology of right ventricular hypertrophy
    - Pulmonary emphysema
    - Chronic interstitial pneumonia
    - Brown induration of lungs
    - Pneumoconiosis
    - Types of hypertrophy
- Mere hypertrophy is called simple hypertrophy
- If the lumen of the chamber becomes narrowed, it is called concentric hypertrophy
- If the hypertrophy is accompanied by dilatation, it is called eccentric hypertrophy
    - Gross pathology
- Heart is enlarged and walls become thicker
- Hypertrophy of right side increases the width of the base of heart
- Hypertrophy of left side increases the length of the heart
- Bilateral hypertrophy causes the heart to be rounder
- Usually hypertrophy and dilatation of heart occur together

- Sequelae
- The thickened heart muscle fibers need more nutrition, which may not be adequately supplied by coronary vessels.
- Because of inadequate blood supply waste products cannot be eliminated. Both the factors lead to degeneration of myocardial fibers.
- Continuous work load further stresses the heart and makes it weak. Ultimately atrophy of heart muscle takes place.
- Atrophied heart muscle undergoes decompensation and so dilates which ends in heart failure.

- **DILATATION**
  - Definition
- Due to deficient emptying during systole one or more chambers of heart may undergo enlargement
  - Introduction : Very great dilatation of heart of man is called cor bovinum
  - Occurrence : Right ventricle is more commonly affected
  - Etiology
- Sudden acute dilatation occurs in severe acute intoxicating conditions and infections causing myocardial degeneration and myocarditis
- Chronic dilatation usually occurs along with hypertrophy in which it is a terminal lesion
- Dilatation of heart that is a sequel to stasis in pulmonary circulation is called cor pulmonale .
  - Gross pathology
- The heart is rounder and globular
- The walls are thinner
- Papillary muscles are attenuated
  - Sequelae : Dilatation of heart leads to congestive heart failure

- **CARDIAC FAILURE**
  - Definition
- It is a syndrome of failing circulation in various organs due to decompensation and loss of contractility.
  - Introduction
- The failure of one side of heart leads to failure of the other side also, hence cardiac failure is usually bilateral.
- To arrive at a diagnosis, it is essential to examine other organs too along with heart.
  - Left-sided heart failure
- *Etiology*
  - Aortic valvular disease
  - Mitral valvular disease
  - Nephritis in dogs
  - Hypertension
  - Congenital heart disease
  - Myocardial degeneration
  - Myocarditis
  - Adhesive pericarditis
- *Pathogenesis*
  - Left sided heart failure leads to diminished blood supply to various organs
  - Anoxia of brain causes increased irritability, restlessness and in far advanced cases stupor and coma

- Renal anoxia causes impaired renal function. Salt and water are retained which causes increased blood volume and edema in dependant parts of the body
- Diminished excretion of nitrogenous substances leads to prerenal uremia
- Pulmonary congestion occurs due to venous stasis
- Pulmonary congestion leads to pulmonary edema. Edema fluid irritates the respiratory mucosa and causes cough
- Impaired exchange of gases
- Hypoxia leads to stimulation of carotid sinus and respiratory centre which causes reflex dyspnoea

- *Clinical signs*
  - Clinical signs are due to the affections of lungs, kidneys and brain
  - Restlessness
  - Dyspnoea
  - Cough
  - Edema in dependant parts of the body
  - coma
- *Lesions*
  - Stasis in the pulmonary circulation is the reason for the pathology seen in left sided heart failure
- *Lungs*
  - Due to damming back of blood in the lungs pulmonary congestion occurs
  - Because of alveolar congestion edema fluid accumulates in alveoli
  - Sometimes small capillaries rupture leading to small haemorrhages in alveoli (Heart failure cells can be seen)
  -
  - Right-sided heart failure
  - Usually right sided heart failure occurs along with left sided heart failure
  - Rarely right sided heart failure occurs in a pure form
- *Etiology*
  - Causes as those for left sided heart failure
- *Causes for the pure form of right sided heart failure*
  - Increased resistance to flow of blood in the lungs as in emphysema and chronic interstitial pneumonia
  - Hydropericardium – As blood entering the heart is blocked
  - Constrictive pericarditis - As blood entering the heart is blocked
  - Endocarditis of tricuspid valves producing incompetence and stenosis
  - Myocardial degeneration and infarction
  - Myocarditis – As the right ventricle is much thinner than the left, it fails more rapidly when myocardium is damaged
- *Pathogenesis*
  - Pulmonary congestion that occurs in left sided heart failure ultimately affects the right ventricle and auricle
  - There is damming back of blood in the systemic and portal venous circulation with consequent decreased flow of blood into the left auricle from the lungs
  - Venous stagnation produces interstitial edema
  - Anoxia occurs later which affects visceral organs
  - Decreased renal blood flow causes reduced glomerular filtration rate and hence reduced sodium filtration. Also salt is reabsorbed and the retention of

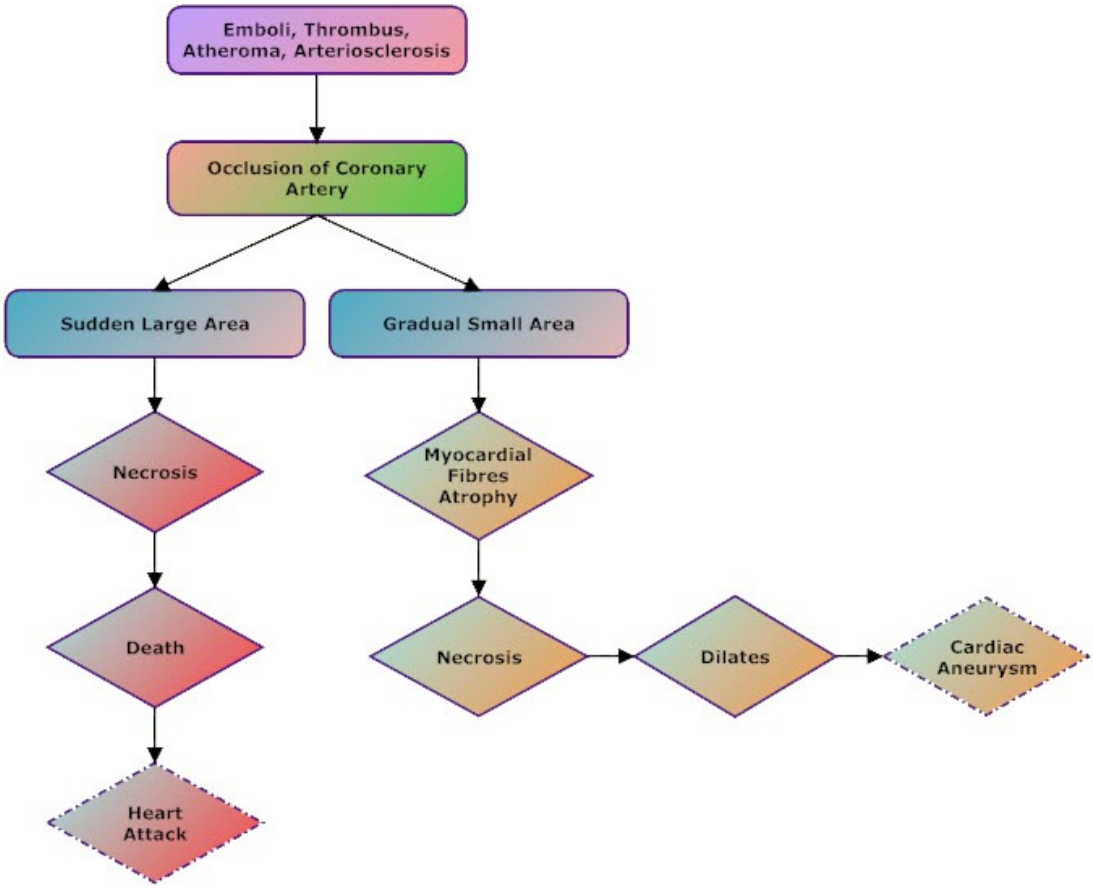
salt also pulls more water from the tubules which ultimately increases the blood volume. So edema occurs.

- *Clinical signs*
  - Cyanosis
  - In the dogs ascites is manifested, in cats pleurisy and in the horse and ox edema is seen subcutaneously in the dependent parts
  - Icterus
  - Diarrhoea
  - Epistaxis in horses
- *Lesions*
  - Stasis in the systemic circulation is the reason for the pathology seen in right sided heart failure
  - Liver
- *Gross pathology* : Enlarged and congested
- *Histopathology*
  - Haemorrhages around the central vein
  - Atrophy and necrosis of hepatic cords around the central vein
  - Later in chronic stage fibrosis around the central vein occurs (Cardiac cirrhosis)
  - Kidneys
- *Gross pathology* : Congested
  - Spleen
- *Gross pathology* : Enlarged and congested
- *Histopathology*
  - Haemorrhages may be present liberating haemosiderin followed by fibrosis. Such areas may be calcified
  - Metaplasia of reticulum to fibrous tissue causes hardening of the organ
  - Stomach and intestine
- *Gross pathology* : Congested

DEGENERATIONS

- Myocardial degenerations includes cloudy swelling, fatty degeneration , fatty infiltration and hyaline degeneration.
  - Cloudy swelling
- Etiology : Toxaemia, septicaemia, febrile conditions and autolysis
- Gross pathology
  - Heart is slightly enlarged, pale, friable and the myocardium has a “cooked up” appearance
- Histopathology
  - Myocardial fibres: Slightly swollen
  - Cytoplasm: Granular
  - Cross striations: Indistinct
  - Fatty degeneration
- Etiology
  - More severe irritant
  - Prolonged infections
  - Nutritional deficiency – Vitamin E, iron (Piglet anaemia)
  - Toxaemia
  - Poisons – As, P, Chloroform
  - Pyometra
  - Purpura haemorrhagica in horses

- Gross pathology
    - Heart
        - Size: Enlarged
        - Colour: yellowish – stripping of the endocardial surface especially on the papillary muscles (Tigering / Thrush – breast heart)
- Histopathology : Myocardial fibres contain fat droplets; the nuclei are degenerated.
    - Fatty infiltration
- Definition : The fat of coronary adipose tissue infiltrated between the myocardial fibres
- Etiology : Obesity
- Histopathology : Crowding of myocardial fibres
- Sequelae : Heart failure
    - Hyaline degeneration
- Etiology
    - Vitamin E deficiency (White muscle disease in claves; stiff lamb disease in lambs)
    - Copper deficiency in cattle (Falling disease)
    - Poisoning – gossypol, mercury
    - Myocarditis
- Histopathology : Cardiac muscle fibres are homogeneous and glassy
- Sequelae
    - Hyaline degeneration is a prenecrotic lesion usually terminating in necrosis and calcification
    - **NECROSIS**
- Myocardial necrosis is usually focal
    - Etiology
- Deficiency of vitamin – E in claves and lambs; thiamine in pigs
- Virus – FMD in calves; equine viral arteritis and swine fever
    - Gross pathology : Heart contains scattered grayish spots.
    - Histopathology : Coagulative necrosis of myocardial fibres
    - Calcification
- Myocardial calcification is a dystrophic calcification occurring in necrotic myocardial fibres
- Etiology
    - Deficiency of vitamin – E in claves and lambs
    - Hyaline degeneration is followed by calcification
    - Excessive vitamin-D and calcium therapy in puppies
- Histopathology
    - Myocardium or Purkinje fibres - calcified
    - **CIRCULATORY DISTURBANCES**
- Haemorrhages
- Infarction
    - Haemorrhages
- *Etiology* : Toxaemia, septicaemia and death due to asphyxia
- *Gross pathology*
    - Epicardium shows petechiae
    - Subendocardium shows ecchymoses
    - Myocardial infarction
- *Occurrence* :Uncommon



- **INFLAMMATOIN**

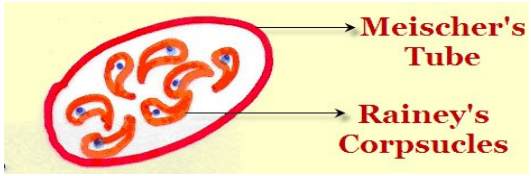
- Inflammation of myocardium includes myocarditis, parasitic myocarditis and specific inflammation.
  -
  - Myocarditis
- Primary - rare
- Secondary - Seen in many systemic diseases
  - Non-suppurative myocarditis
- *Route of infection* : Haematogenous
- *Etiology*
  - Bacteria
    - *Pasteurella*(cattle)*, Leptospira, Spherophorus*
    - As metastatic foci– *Streptococcus, Escherichia coli*
  - Viral
    - Foot and mouth disease (FMD) in calves, infectious canine hepatitis, equine infectious anaemia
  - Pathological
    - Generalised septicaemia, toxaemia, bacteremia
    - Extension of infection from valvulitis and pericarditis
- *Gross pathology* : Heart may show focal pale, grayish or yellowish areas
- *Histopathology*
  - Acute case
    - Degeneration of myocardial fibres in FMD / coagulative necrosis in spherophorus infection

        - Infiltration with lymphocytes, macrophages, plasma cells and eosinophils
    - Chronic cases : Fibrosis is seen
- *Sequelae* : In case of FMD – young calves may die.
    - Acute suppurative myocarditis
- *Spread of infection*
    - Through coronary artery
    - Extension of infection in traumatic reticulopericarditis
    - Direct extension of infection from purulent pericarditis, endocarditis, pleura, lungs and bronchial lymph nodes
- *Etiology* : Pyaemia seen in joint ill, mastitis or metritis
- *Gross pathology*
    - Myocardium may contain abscess with hyperaemic borders. Some abscesses have capsule. In some cases scars may be noticed.
- *Histopathology*
    - Myocardium may contain suppurative foci with numerous neutrophils
    - Fibrosis and calcification may also be seen

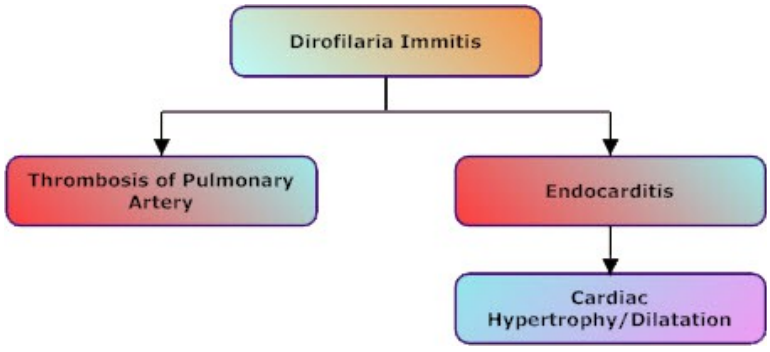
PARASITES OF MYOCARDIUM

- Sarcocystis tenella

- Found universally in the myocardium of cattle, sheep and pigs.
- The muscle fibres contain the Miescher's tubes in which are found the spores called Rainey's corpuscles.



- Hydatid cysts of Echinococcus granulosus of dogs contain scolices and clear fluid.
- *Bladder worms like Cysticercus bovis, C. cellulosae, C. ovis may be found in the myocardium*
- *Toxoplasma gondii*
    - May be found as pseudocysts in the myocardium.
    - If the cysts rupture focal myocarditis occurs. The lesion consists of necrosed centre surrounded by neutrophils, lymphocytes and macrophages. Sometime necrosed centre may be calcified.
    - Heart worm



SPECIFIC DISEASES

- Tubercular myocarditis may occur from haemotogenous infection
- Actinomycosis may also affect myocardium.

NEOPLASMS

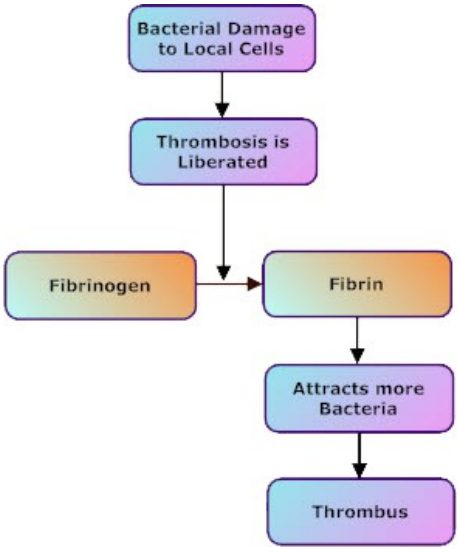
- Primary tumours of the heart are uncommon; Marek's disease may cause lesion in the heart of chicken
- Secondary tumours are common e.g. Lymphosarcoma in cattle
    -
    - **PATHOLOGY OF ENDOCARDIUM**
        - **Endocarditis**
    - Definition : Endocarditis means inflammation of endocardium of heart
    - Occurrence: Common in animals – Swine are more frequently affected
    - Types
- Inflammation of valves is common and is called valvular endocarditis
- Inflammation of septal endocardium is called mural endocarditis
    - Sites of occurrence

○ S	○ Frequents ite
○ H	○ Aortic valve
○ C	○ RightA-V valve

○ D	○ Left A-V valve

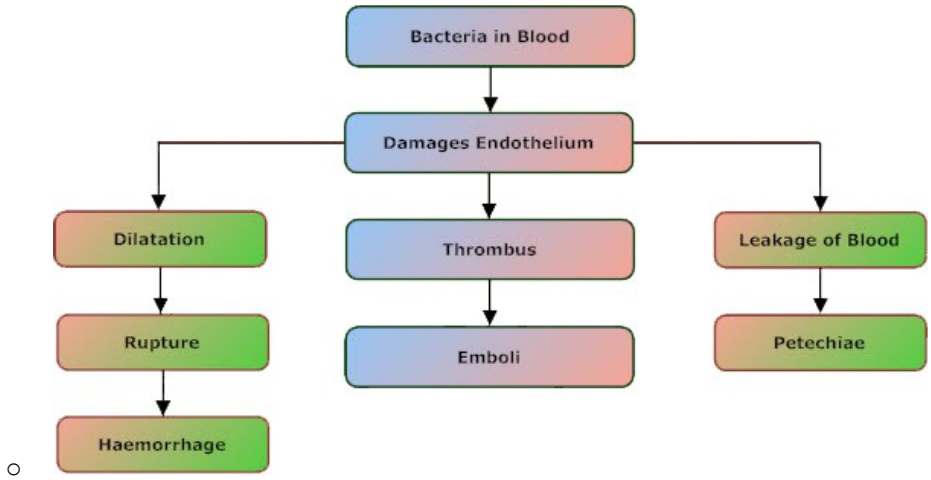
- Sources of infection

- Septicaemic infections
- Oral infection
- Umbilical infection
- Suppurative metritis and mastitis
- Traumatic reticulopericarditis (TRP) in cattle
- Liver abscess, peritoneal abscess
- Streptococci of intestinal origin (white scours in calves) or oral origin (in dogs)
  - Etiology
- Bacteria
  - *Erysipelothrix rhusiopathiae* in swine
  - *Streptococcus equi* (Strangles in horses), *Shigella equirulis*
  - Streptococci in cattle, swine and dogs
  - Leptospirosis in dogs
  - *Corynebacterium pyogenes* in cattle
- Parasites ;Migrating larvae of Strongylus in horses
- Pathological : Chronic septicaemic diseases
  - Pathogenesis
- The valves are more often affected as they are exposed to the circulating bacteria and the force of blood.
- The auricular side of valves is damaged usually. In the valves it is the edges of valves that are frequently affected.



- The enlarged valves injure the adjacent valves during its movement and the thrombi may break off and form emboli.
    - Gross pathology
- The thrombus is cauliflower like and friable and is called as vegetation . Endocarditis in which these vegetations are present is called vegetative endocarditis .
- In swine excessive vegetations are common
- In cattle affected with black quarter, roughening of left auricular endocardium occurs
- In dogs with leptospirosis and uraemia, ulcerative endocarditis is common. Greenish ulcers are present in the left auricle and ventricle, pulmonary artery and aorta.
    - Histopathology
- The lesion consists of a thrombus with central bacterial clumps
- Leucocytes are present in the intima
- From the basal area of the valve fibrous tissue invades the thrombus. But as long as the organisms are alive, complete organization and healing does not take place
- In cattle *C. pyogenes* causes excessive fibrosis
    - Sequelae
- Vegetations in the valves cause valvular insufficiency or stenosis. When tricuspid valve is affected hypertrophy and dilatation of the right auricle and general venous congestion. When mitral valve is affected, hypertrophy and dilatation of the left auricle, pulmonary congestion, edema and pneumonia takes place. When pulmonary valve is involved, dilatation of the right ventricle, general venous congestion and hypertrophy of the right ventricle occurs. If aortic semilunar valves are affected, dilatation of the left ventricle, hypertrophy of the left ventricle and later general venous congestion will occur. Finally congestive heart failure will occur.
- From the thrombus (vegetation), pieces of thrombus may be detached to form the emboli. If the emboli are contaminated with bacteria (arising from left side of heart) abscesses will be formed in the kidneys (acute embolic nephritis), spleen and liver. Emboli arising from the right side, pulmonary abscesses are formed. Emboli of coronary vessels cause myocardial abscesses. If the emboli are bland and sterile, infarcts are formed in the kidneys and spleen from emboli of left side heart origin. Emboli arising from right side cause thrombosis in lungs. Myocardial infarction occur if emboli enter coronary circulation
- From the vegetations, infection extend to the chordae tendinae, which become weaker , degenerated and then may rupture
- Extension of inflammation from the valves to the walls causes mural endocarditis
    - **ENDOCARDIOSIS**
    - Synonym : Chronic valvular disease
    - Occurrence : Common is 4 – 5 years old dogs
    - Site of occurrence : Mitral valve is commonly involved
    - Gross pathology
- Valve – Thick, shrunken, nodular, distorted
- Chordae tendinae - Thick or ruptured
- Lungs – Acute pulmonary edema
    - Histopathology
- Valves are thickened due to fibroelastic tissue with abundant mucous ground substance
    - Sequelae : Congestive heart failure
    - **ARTERITIS**
    - Definition

- Inflammation of the wall of artery is called arteritis. Inflammation of tunica intima is called endarteritis
    - Occurrence : Uncommon
    - Types : Based on the course, arteritis is classified as acute and chronic arteritis
    - Acute arteritis
- *Route of infection*
    - Perivascular extension of infection is common as in pneumonia, mastitis and metritis
    - Vasavasorum
    - Haematogenous from lumen- As in case of umbilical infection, the pulmonary vessels are the favoured places where emboli lodge
- *Etiology*
    - Physical trauma
    - Nutritional – vitamin E and C deficiency (microangiopathy in pigs)
    - Bacteria –Pyogenic
    - Virus – Equine viral arteritis, equine herpes virus, infectious canine hepatitis and feline infectious peritonitis
    - Parasite
    - Fungi
    - Toxins – Uremia
- *Histopathology*
    - Intima and media – Infiltrated with lymphocytes
    - Connective tissue cells – Swollen
    -
    -
- *Sequelae*
    - Inflammation of the intima results in the formation of a thrombus at the site called thromboendarteritis .

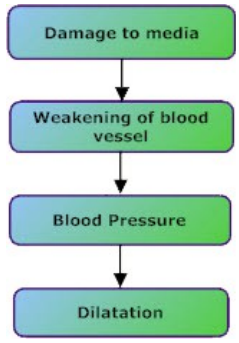


    - Chronic arteritis
- *Etiology* : Larvae of *Strongylus vulgaris* in the anterior mesenteric artery of horse
- *Gross pathology*
    - Artery: Dilated / aneurysm forms which may rupture with fatal haemorrhage
    - Wall: Loss its resiliency
    - Intima: Rough, has thrombus which contain larvae - Shows series of fine tracts
- *Histopathology*
    - Artery wall - Loss of Elastic tissue -Fibrosis

- Media- Degeneration of muscles
- Intima – Has thrombus which contains parasite and fibrin deposits.

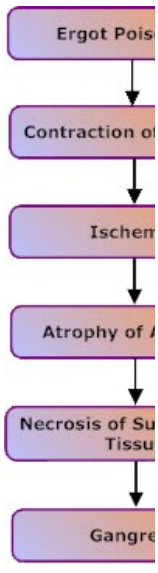
ANEURYSM

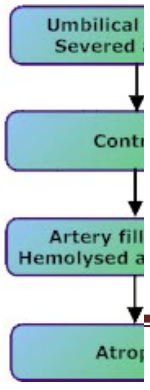
- Definition: Localized circumscribed dilatation of an artery, vein or cardiac chamber.
- Pathogenesis



- Etiology

- Physical – trauma
- Infected emboli or tuberculous lesion may cause destruction of media and weakening of alveoli
- Parasite – *Strongylus vulgaris, Spirocerca lupi*
- Toxins- Uremia
- Disease of artery wall – atheroma
- Congenital weakness of vessel wall
  - Gross pathology
- Intima
  - Intact
  - Depressed
  - Rough
  - Covered with thrombus
  - Histopathology
- Media / intima
  - Wholly or partially ruptured
  - Fibrosed
  - **TYPES OF ANEURYSM**
    - **True aneurysm**: The sac is formed by the wall of the artery
    - **False aneurysm**
- Here the wall of the artery is ruptured and hence the sac is formed by the surrounding connective tissue.
  - **Dissecting aneurysm**
- This is not a true aneurysm. Here no dilatation of the wall occurs.
- In aorta the wall is dissected and so blood circulates within the media of the wall itself.
- The outer wall may rupture leading to fatal haemorrhage.
- This is usually due to degenerative lesion in the media.
  - **Saccular aneurysm**: Here the dilatation is characterized by formation of pouch on one side of wall
  - **Fusiform aneurysm**

- Here a long segment of the vessel is uniformly dilated around the whole circumference.
- This is usually seen in the aorta and its branches.
    - **Miliary (Berry) aneurysm**
- This aneurysm occurs in the minute arteries as in the brain. This is congenital in origin.
    - **Arteriovenous aneurysm (Aneurysmal varix)**
- It is an abnormal acquired communication between an artery and a vein due to simultaneous injury to both.
- There is pulsation in veins since blood passes directly into it.
    - **Cirsoid aneurysm**
- It is a mass of dilated pulsating and intercommunicating arteries and veins.
- It is usually subcutaneous in location. Most are congenital and a few may be due to trauma.
    - **Cardiac aneurysm:** This is focal dilatation of cardiac chamber.
    - **Bacterial aneurysm**
- This is due to bacterial infection which weakens the wall.
- Aneurysm develops at those sites.
- This is usually associated with vegetative endocarditis.
    - **Parasitic aneurysm**
- This is due to parasites which weaken the wall.
- e.g. *Stongylus vulgaris* in the anterior mesenteric artery in horses.
- *Sequelae*
    - Thrombus formation or emboli formation which may occlude the vessel e.g. *Stongylus vulgaris* in the anterior mesenteric artery in horses lead to colic.
    - Atrophy of vessel wall - fibrosis - calcification
    - Compensatory hypertrophy of tunica adventitia
    - Rupture of vessel wall - haemorrhage
    - Pressure atrophy of surrounding tissue
    - Extension of inflammation e.g. In *Stongylus vulgaris* infection of the anterior mesenteric artery in horses, spread of inflammation to adjacent autonomic ganglia lead to colic.
    - Growth Disturbances
- *Atrophy*
    - **Ergot poisoning**
    - 

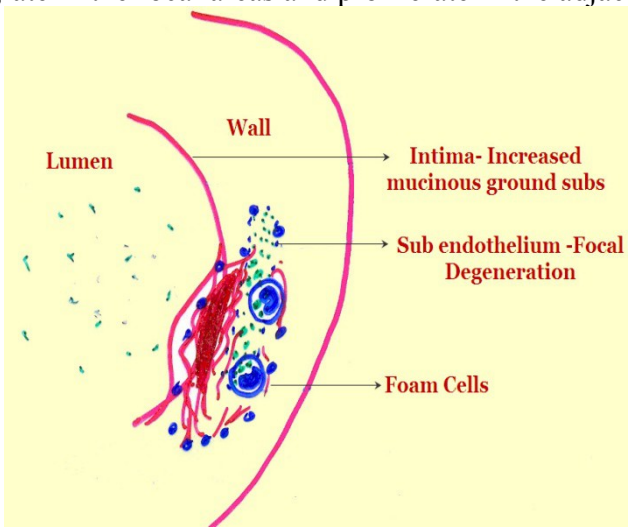
    - **Umbilical arteries severed at birth**
    - 

    -
    -
- *Hypertrophy*
    - Compensatory hypertrophy of the wall of the artery is common
    - Usually the elastic lamina and muscles are involved to withstand the increased blood pressure
- *Occurrence*
    - Dog: Chronic interstitial nephritis in renal artery
    - Cat: *Toxocara cati* infection in pulmonary artery
    -
    -

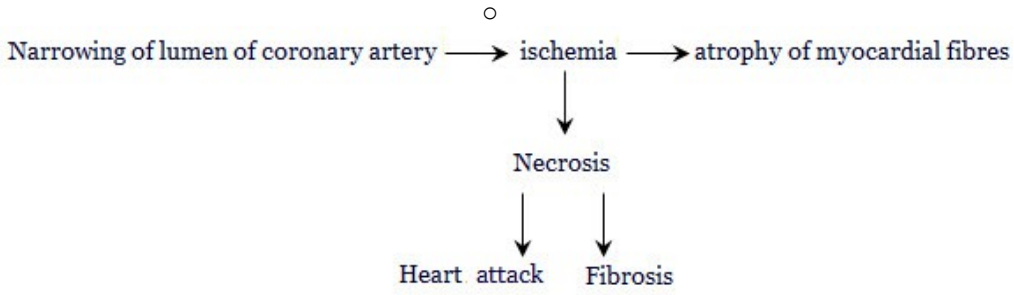
- **CIRCULATORY DISTURBANCES**
  - **THROMBOSIS**

- *Strongylus vulgaris* produces thrombosis of anterior mesenteric artery in horses
  - Thrombangitis obliterans
- This is an acute inflammatory reaction involving all layers of the wall of artery resulting in thrombosis.
- There is proliferation of endothelial cells and fibroblasts.
- Organization of thrombus and subsequent canalization may occur. e.g. Tuberculosis
  - **ATHEROSCLEROSIS**
  - Definition
- Athere means a soft, mushy, gruel like substance.
- In this condition such a substance is deposited in the intima of larger elastic arteries.
  - Occurrence : Man and pigs
  - Site of occurrence
- Commonly affected – Aorta and its primary branches
- Occasionally affected -Cerebral and coronary vessels
- Pigs – Abdominal aorta
  - Etiology
- Contributing factors in human beings
  - Heredity
  - Lack of physical activity
  - Stress
  - hypercholesterolaemia
  - Diabetes mellitus
  - Hypothyroidism
  - Obesity
  - Hypertension
  - Senility
  - Intramural haemorrhages
  - Smoking
  - Toxins
  - Pathogenesis
- The lesion commences as a focal degenerative change in the subendothelial tissue in man and fowls. Whereas in pigs the lesion commences in the middle and outer layers and extend to the intima
- The mucinous ground substance of intima is increased
- In the subendothelial area fine fat droplets appear. These fatty deposits consist of cholesterol, neutral fats, fatty acids and cholesterol esters
- Macrophages are thought to transport fat from the blood into this area. Macrophages are loaded with fat (foam cells) and start accumulating in the subendothelial layer. Macrophages later die and liberate the fats in to the surrounding area. The cholesterol crystals stimulate the proliferation of the connective tissue around these foci, especially towards the luminal side. This newly formed tissue becomes hyalinised. This is the atheromatous plaque with central debris consisting of granular, lipid rich material and acicular crystals of cholesterol.
- This lesion is well supplied with capillaries
- Haemorrhages and haemosiderin granules may also be seen
- A few lymphocytes may be seen around the lesion
- With further deposition of lipids, fibrous tissue increases in quantity
- The atheroma enlarges in size and reaches the endothelial layer and may be pushed into the lumen

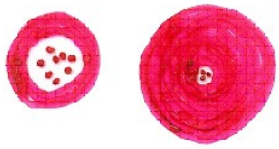
- Degenerative changes are noticed in the media also. The edema first separates the muscle fibres and elastic fibres. In these areas collagen is formed and scarring occurs
- Elastic fibres disintergrate in the focal areas and proliferate in the adjacent areas



  - Gross pathology
- The affected vessel is enlarged, less pliable and the wall is thickened
- The intima of aorta may reveal round or oval fatty areas (0.1 to 2.0 c.m diameter)
  - Histopathology - Aorta
- The microscopic lesion is as described in the pathogenesis
- The endothelium shows hydropic degeneration and appears thickened
- Foam cells appear in the area (Subendothelial or middle and outer layers)
- Fibroblastic proliferation occurs around the leison
- Muscle fibres show hydropic degeneration
  - Sequelae
- Upon healing- dense inflammatory scar containing cholesterol cleft occurs. It may be calcified and islands of heterotopic bone may be formed in it.
- The luminal side may be ulcerated - thromobus - emboli
- Weakening of wall - aneurysm



- Extension - Endocardium
  - **PARASITIC AORTITIS**
  - Onchocerciasis
- Etiology : *Onchocerca armillata*
- Species affected : Cattle, buffalo and, goats
- Site of occurrence : Aortic arch
- Gross pathology : Intima – corrugated with irregular tracts
- Histopathology: Focal granuloma – atheromatous plaque with calcification
  - Spirocercosis

- Etiology : *Spirocerca lupi*
- Species affected : Dogs
- Pathogenesis : During the migratory life cycle of the parasite the larvae at one stage pass through the aorta
- Gross pathology : Aorta – has atheromatous ulcers
    - **ARTERIOSCLEROSIS**
    - Definition
- Thickening of wall with narrowing of lumen of a small artery and arteriole is called arteriosclerosis
    - Species affected : It is not common in animals
    - Site of occurrence : Kidney, spleen, pancreas, adrenal and small intestines
    - Etiology
- Johne's disease
- Toxins
- Hypertension
- Hypothyroidism in old dogs
    - Gross pathology
- *Artery*
    - Thick, hard
    - On section, stand prominently
    - Lumen – narrow
    - Loss of elasticity
    - 
    - Histopathology
- Wall of artery – Thick with homogenous pink collagenous fibrosis
- Intima to media - Hyaline degeneration, fibrosis and calcification
    - Types
- Hyaline – occurs due to slowly developing hypertension
- Hyperplastic – occurs due to sudden elevation of blood pressure
- Endothelial cells, fibroblasts and muscle cells – Proliferate in a concentric manner and has onion skin appearance
    - Sequelae
- Hypertrophy and dilatation of left ventricle
- Focal myocardial fibrosis
    - **MONCKEBERG'S MEDIAL SCLEROSIS**
    - Occurrence : Cattle and old dogs
    - Etiology
- Stress
- Hypervitaminosis – D
- Chronic interstitial nephritis
    - Pathogenesis
- Endothelium – Intact
- Muscular tissue- Degeneration Fatty / hyaline → Necrosis → Calcification (Heterotopic ossification)
    - **MANCHESTER WASTING DISEASE**

OF CATTLE

- Calcific arteiosclerosis

- *Etiology*
  - Excessive Vitamin D
  - Toxins causing hypercalcaemia
- *Signs*
  - Progressive wasting
  - Arched back
- *Gross pathology*
  - Major arteries – thick, hard and brittle
  - Intima – plaque
- *Histopathology*
  - Medial layer of muscle fibres in arteries show degenerative changes and calcium deposition
  - Intimal connective tissue show moderate proliferation

EQUINE VIRAL ARTERITIS

- Etiology : Toga virus
- Signs

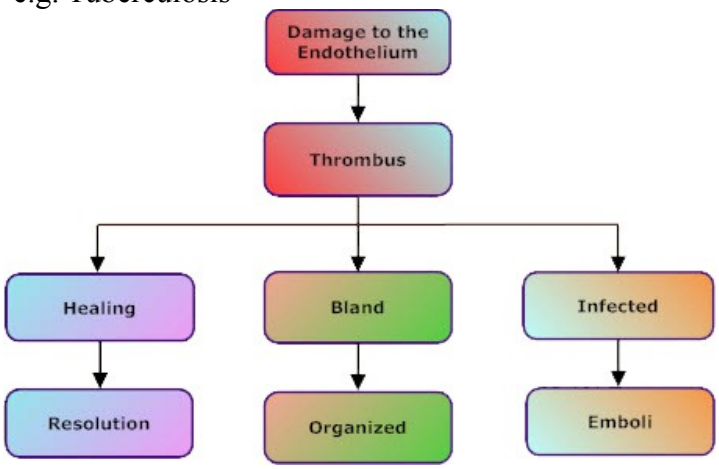
- Fever
- Conjunctivitis
- Palbebral edema
- Abortion
  - Gross pathology
- Serous membrane shows petechiae
- Serous cavities contain more fluid
  - Histopathology
- Small arteries – media – muscle fibres- necrosed
- Adventia shows edema and lymphocytic infiltration

POLYARTERITIS NODOSA (PERIARTERITIS NODOSA)

- It is a collagen disease affecting all layers of the wall of artery
  - Site of occurrence
- Small and medium sized arteries in musculature, myocardium, subepicardial fat and mammary gland
  - Etiology
- Bacteria – Streptococci
- Virus – Bovine malignant catarrhal fever
- Bovine viral diarrhoea
- Equine infectious anaemia
- Allergy to drugs - Sulphonamides
- Sarcosporidia
  - Histopathology
- Adventitia shows edema, necrosis and infiltration with neutrophils and eosinophils. After the acute phase the exudates is organized and infiltration with macrophages and lymphocytes occur
- Media shows fibrinoid necrosis
- Intima contains thrombus which may occlude the lumen. The wall may be weak and produce aneurysm which gives nodular appearance

- **VEINS**
- Phlebitis
- Phlebitis means inflammation of veins

- *Acute phlebitis*
  - It is caused by intravenous infusion of irritants, traumatic reticuloperitonitis and through extension from wounds
- *Chronic phlebitis* : e.g. Tuberculosis



- Varicose veins

- *Definition* : Varicose veins are dilated and tortuous veins.
- *Occurrence*
  - Not common in animals – Veins of scrotal plexus in horses and supramammary veins in cows
  - Common in human beings
- *Etiology*
  - Trauma, high blood pressure, hindrance to flow as in mitral stenosis, pulmonary emphysema and cirrhosis of liver, ageing and standing for long time in human beings
- *Histopathology - Veins*
  - Muscular hypertrophy is followed by atrophy
  - Elastic tissue is replaced by fibrous tissue and the affected areas may dilate.
  - Thombosis may occur at those locations
  - Telangiectasis
- Here sinusoids of liver are widely dilated locally.
- It is seen in bovine liver due to *Spherphorus* infection.
- In human beings it is seen in subcutis.

HAEMOPOIETIC SYSTEM

- *TERMINOLOGIES*
- Erythrocyte
- Normal erythrocyte

- Normocytic, normochromic erythrocyte is reddish coloured with central depression (biconcave disc) and palor not more than 1/3 of the surface area.
- Avian erythrocytes are nucleated
- This is not due to increased hemoglobin content but due to increased thickness of erythrocyte.
  - Hypochromasia

- This is decreased staining intensity of erythrocyte which may be either due to decreased thickness of erythrocyte or decreased hemoglobin content.
- This is seen in iron and copper deficiency.
    - Eccentrocyte
- Here the erythrocyte has haemoglobin on one side.
- This is seen in hemolytic anaemia.
    - Annulocyte (Pessary cell)
- Here the erythrocytes have a narrow rim of hemoglobin surrounding a large central pale area
    - Target cell
- Here the erythrocyte has central pigmented material surrounded by a clear unpigmented area and the outside border has haemoglobin pigmentation (resembles a bull's eye).
- These are seen in chronic disease, liver diseases and lead poisoning.
    - Pappenheimer bodies (Siderotic granules)
- These are purplish coccoid granules seen at the periphery of erythrocytes in anemias due to impaired heme synthesis (sideroachrestic anemias).
- The granules contain iron in ferritin.
    - Polychromasia (Polychromatophilia)
- This denotes staining of erythrocytes with many colours i.e. red, blue and intermediate colours.
- This may be an immature cell or it may be seen in blood loss.
    - Cabot rings
- Cabot rings are bluish thread like rings seen in the erythrocytes.
- These are seen in hemolytic and toxic anemias and lead poisoning.
    - Heinz bodies
- These are refractile inclusions found in the erythrocytes.
- These are associated with denatured protein.
- These are seen hemolytic anaemia and in horses undergoing phenothiazine therapy.
- They are not visible when the blood smear is fixed in methanol.
    - Howell – Jolly bodies
- These are single or double spherical bluish bodies situated eccentrically usually.These are remnants of nuclear material.
- These are normally seen in young pigs and dogs. Horse blood also contains these bodies in one per cent of erythrocytes.
- In bovine these should be differentiated from *Anaplasma marginale.* The *Anaplasma marginale* is uniform in size while Howell – Jolly bodies vary in size.
- These are seen in hemolytic anaemia, splenectomy and lead poisoning.
    - Basophilia
- Here the erythrocytes take a pale bluish or bluish stain instead of red stain. This is because of retention of ribonucleic acid which takes up bluish stain. This indicates incomplete maturation of erythrocytes, lack or deficiency of hemoglobin. This is seen in anemia.
    - Basophilic stippling (Punctate basophilia)
- Here the erythrocyte has blue staining granules scattered through out.
- These are remnants of RNA. This condition is seen in anaplasmosis in bovines, haemonchosis in sheep and in lead poisoning.
    - Spherocyte

- These are dome shaped, thick erythrocytes.
- These are seen in immune mediated hemolysis and blood loss.
- These are not seen in animals.
    - Leptocyte
- Leptocytes are thin erythrocytes with larger surface without increase in volume.
- These are seen in chronic diseases and liver diseases.
    - Megaloblast
- This is an immature erythrocyte comparable to prorubricyte.
- This is seen in Vitamin B_{12} or folic acid deficiency.
    - Macrocyte
- The erythrocyte has larger diameter than the normal erythrocyte.
- Hence the mean corpuscular volume is higher.
- This is an immature cell and is seen in blood loss.
    - Microcyte
- Here the erythrocyte has smaller diameter than the normal.
- This is seen in iron deficiency.
    - Anisocytosis
- Anisocytosis means variation in the size of erythrocytes.
- In cattle blood slight anisocytosis is common.
    - Poikilocyte : Here the erythrocytes have varied shapes.
    - Crenation
- Here the erythrocytes have abnormal notching.
- This is seen in splenic disease and haemangiosarcoma.
    - Schizocyte
- This is a small irregular erythrocyte fragment that may have two or three pointed extremities.
- This is seen in intravascular coagulation and vasculitis.
    - Dacrocyte
- This is a teardrop shaped erythrocyte.
- This is seen in myelofibrosis
    - Keratocyte
- Here the erythrocyte has half-moon shape with spicules.
- This is seen in intravascular coagulation
    - Meniscocyte (Drepanocyte)
- These are crescent shaped erythrocyte.
- This is seen in sickle cell anemia.
    - Ovulocyte
- These are elliptical erythrocytes.
- These may be seen in advanced anemia with poikilocytosis.
    - Reticulocyte: Reticulocyte is a precursor of erythrocyte and cannot divide but can undergo maturation.
    - Normoblast : Normoblast will have the remnant of nuclear material
    - **POLYCYTHEMIA**
    - Definition
- Polycythemia means increase in the circulating erythrocyte.
- Blood picture: Normocytic, normochromic
    - Types

- *Relative polycythemia*
    - This is seen when there is reduction in the circulating blood volume.
    - Hence there is increased concentration of normal number (hemoconcentration).
    - This is seen in excessive fluids loss as in dehydration due to continued vomiting, diarrhea, sweating, shock and collapse.
- *Absolute polycythemia*
    - Here the blood volume is normal but the erythrocyte number is more.
    - This condition may be primary, secondary or due to pathological causes.
    - Primary – Polycythemia vera – a tumor of bone marrow may cause polycythemia.
    - Secondary – Physiological: Seen permanently in high altitude
        - Termporary: Seen in splenic contraction.
        - e.g Sporting dogs, racing horses
        - Neonates have more number of erythrocytes
    - Pathological – Prolonged anoxia e.g. Cardiac and pulmonary disease, Tetralogy of Fallot

OLIGOCYTHEMIA

- Definition
- This is decrease in the quantity of erythrocytes in the peripheral blood.
- RBCs (absolute)
    - Types
- *Relative oligocythemia*
    - Here is increase in the blood volume (hemodilution) with normal number of erythrocyte.
- *Absolute oligocythemia* : This is otherwise called as anemia.

ANAEMIA

- Definition : Anaemia means decrease in the quantity or quality of erythrocytes below normal.
- Clinical signs : Visible mucous membrane will be pale
- Types
- Production is low but destruction is normal: Dyshaemopoietic anaemia
- Production is normal but destruction is high: This is seen in haemolytic and haemorrhagic anemia.

DYSHAEMOPOIETIC ANEMIA

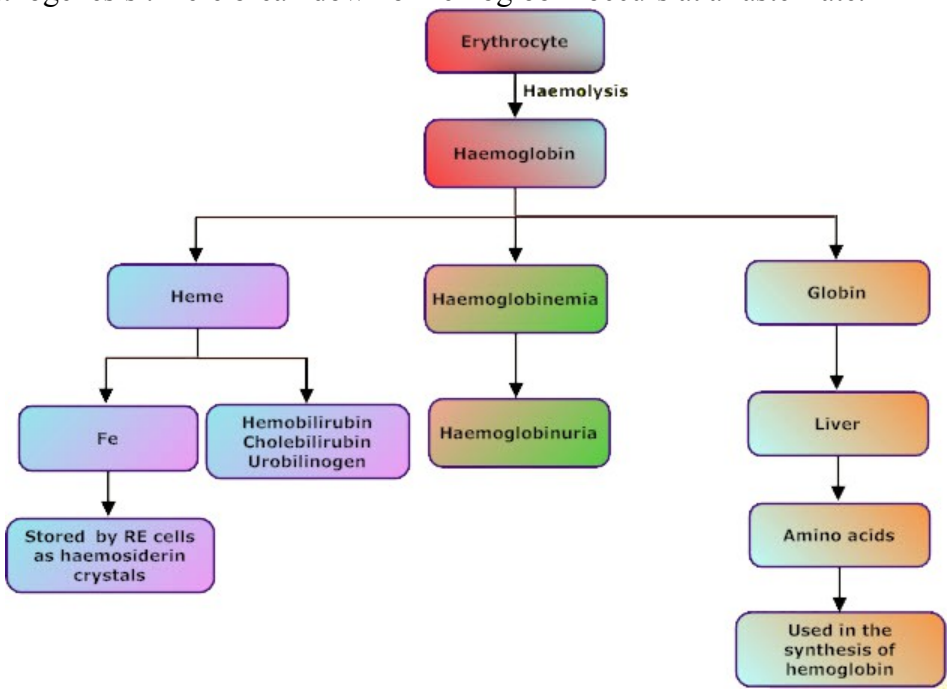
- Definition
- In dyshemopoietic anemia, there is defect in the formation of haemoglobin.
- The defect may be either in the formation of protein or hemoglobin.
    - Decreased heme synthesis
- *Porphyrinopathy*
    - Porphyrins are needed for the synthesis of heme.
    - If certain enzymes are lacking, then heme is not synthesized .
    - So excessive amounts of porphyrins are found in the body (Porphyria)and in urine (Porpyrinuria).
    - Congenital porphyriesa occurs in bovines and pigs.
    - Porphyrins are photosensitive. The following changes are seen
        - Skin undergoes photosensitization (Photosensitive dermatitis)
        - Teeth looks pink (Pink tooth)
        - In bone, ostoheamochromatosis occurs

        - In kidneys, porphyrins are deposited in the tubular epithelium and interstitium. Urine also contains porphyrins (porphyrinuria). The urine turns red on exposure to light.
    - Decreased protein formation
    - Blood picture is macrocytic and normochromic or hypochromic
    - Bone marrow shows numerous megaloblasts and giant metamyelocytes
- *Dietetic deficiency of extrinsic factor*
    - Cobalt is needed for the synthesis of Vitamin B_{12} by the microbes in the rumen. Vitamin B_{12} and folic acid deficiency causes arrest of maturation of prorubricyte and metamyelocytes. As Vitamin B_{12} is needed for the synthesis of DNA and RNA, its deficiency leads to delayed maturation of nucleus whereas hemoglobin synthesis continues. When hemoglobin synthesis reaches a certain concentration in the erythrocytes, the nucleus leaves them and so macrocytes result giving rise to macrocytic anemia.
    - Dietetic deficiency of folic acid: As folic acid is essential for the maturation of erythrocytes, in its absence maturation is slowed and so macrocytic anemia occurs.
- *Deficiency of the intrinsic factor*
    - Gastric mucosa secretes an enzyme which helps in the absorption of Vitamin $B_{12.}$
    - In its absence (gastritis), Vitamin B_{12} may not be absorbed and so results in macrocytic anemia.
- Failure to store the erythrocyte maturation factor
    - The erythrocyte maturation factor i.e. Vitamin B_{12} is normally stored in the liver.
    - In diseases of liver, this vitamin is not stored hence there is deficiency of Vitamin B_{12} which in turn causes macrocytic anemia.
- *Failure to use the erythrocyte maturation factor*
    - Failure of mobilization of erythrocyte maturation factor i.e. Vitamin B_{12} or its utilization leads to macrocytic anemia.
    - This is called achrestic anemia (Achrestic means failure to utilize).
- *Hypopituitarism*
    - Anterior pituitary seems to exert a potent influence in erythropoiesis directly or through the thyroid which influences metabolism of carbohydrates, releasing energy.
    - Decreased hemoglobin formation
    - Blood picture is initilally normocytic and hypochromic, but later becomes microcytic hypochromic.
- *Iron deficiency*
    - Iron deficiency may occur in the following conditions
        - Deficient intake: Milk of sows is deficient in iron. If piglets are not allowed for access to iron in soil, they suffer from iron deficiency.
        - Defective absorption: Excessive phosphorus and phytic acid forms insoluble complexes with iron which are excreted through the feces.
        - Increased requirement: Young growing animals and pregnant animals require more iron
- *Copper deficiency*
    - Copper acts as a catalyst in the utilization of iron in hemoglobin synthesis, hence its deficiency leads to iron deficiency.
    - Some cells undergo additional mitosis and so microcytic erythrocytes occur.

- *Dietetic deficiency of ascorbic acid*
    - Vitamin C is dietary reducing agent and so facilitates the reduction of Fe+++ to Fe++ which is easily absorbed.
    - It is also needed for the synthesis of folic acid and its further conversion to more active folinic acid.
- *Dietetic deficiency of pyridoxine*
    - Pyridoxine is required for the utilization of iron in hemoglobin synthesis.
    - So its deficiency leads to iron deficiency.
- *Dietetic deficiency of nicotinic acid*
    - Nicotinic acid interferes with respiration of immature red cells.
- *Dietetic deficiency of riboflavin*
    - Riboflavin is needed for the metabolism and arrangement of amino acids of hemoglobin.
- *Deficiency of thyroxine*
    - Thyroxine along with vitamin C is required for the conversion of folic acid to folinic acid.
    - In myxedema, the secretion of intrinsic factor is depressed as well as absorption of Vitamin B_{12}.
    - So a normocytic or macrocytic anemia may be encountered.
    - Toxic inhibition
    - Here the bone marrow is normal and active but is unable to utilize haematinics.
    - The blood picture is microcytic normochromic and no regenerative forms are seen.
- *Etiology*
    - Physical - Irradiation can damage the hemopoietic system.
    - Chemical - Nitrogen mustard, hair dyes
    - Metals - Arsenic, bismuth and gold
    - Bacteria - In chronic infections like tuberculosis and brucellosis normocytic normochromic anemia is seen. In these conditions there is hypoferremia. In these chronic diseases there is greater utilization of iron by the tissue hence the iron is sided tracked instead of to the bone marrow, so hemoglobin is not formed and anemia results.
    - Parasite - Esophagastomiasis causes decreased absorption from the intestine
    - Pathological - Chronic interstitial nephritis leads to decreased erythropoietin production
    - Poisons - Insecticides
    - Drugs - Sulpha, streptomycin and chloromycetins
    - Aplastic anaemia
    - Here the bone marrow is aplastic and inactive.
    - The blood picture is normocytic normochromic.
    - No regenerative forms are seen.
- *Etiology -Types*
    - Primary : Idiopathic and is rare
    - Secondary
        - Exhaustion caused by chronic haemorrhage due to gastric and intestinal ulcer, blood sucking worms, neoplasm and deficiency of vitamin K, vitamin C and prothrombin.
        - Toxic agents which cause toxic inhibition also cause aplastic anemia.

        - Metabolic: A type anemia occurs in piglets born of sows which suffered from protein malnutrition during pregnancy.
    - Myelophthisic anaemia
    - Myelophthesic anemia means anaemia resulting from replacement of bone marrow by other tissues.
    -
    -
- *Blood picture*
    - Erythroblasts and immature granulocytes are seen. Hence, it is also called leuco-erythroblastic anaemia .
- *Etiology*
    - Myelophthesic anemia may occur in the following conditions
        - Replacement of myeloid tissue by connective tissue which occurs in osteodystrophy
        - Primary tumour like Hodgkin's disease
        - Metastatic tumour - lymphatic leukemia in dog and cat
- *Sequelae of dyshemopoietic anemia*
    - After restitution of hematinics, reticulocyte number increase (Reticulocyte shower).
    - This is called as regenerative anemia .
    - **HEMOLYTIC ANAEMIA**
    - Definition : In hemolytic anemia, there is intravascular destruction of RBCs.
    - Blood picture
- Blood picture is macrocytic normochromic which becomes microcytic and hypochromic as iron stored is used up.
- Many regenerative forms and spherocytes are also seen.
    - Etiology : Hemolytic anemia may occur in the following conditions
    - Poisoning - Copper poisoning
- As copper is poorly excreted if excessive quantities are ingested, the excess copper is stored in the liver and poisoning may result.
- Excessive dose of copper given for deworming
- Ingestion of large quantities of salt lick containing copper sulphate
- Eating forage contaminated with copper from mines or having too much concentration of copper from soil
- Too much of supplemental mineral mixture containing copper sulphate
- In stress conditions more amount of copper is released and may cause hemolysis.
- Lead poisoning
- Poisons – Onion poisoning (n- propyl disulphide); Castor seeds (ricin) toxicity
- Naphthalein balls accidentally ingested by pets
- Drugs – Phenothiazine poisoning- In horses with usual dosage, phenothiazine may be toxic. Cattle may also be susceptible.
- Snake venom which contains lecthinase acts on lecithin and converts it into lyolecithin which is highly hemolytic.
    - Hypersensitivity
- Hypersensitivity to certain drugs like sulphanilamide, quinine, paraaminosalicylic acid and some antipyretic drugs may cause hemolytic anemia.
    - Nutritional
- Hypophosphatemia in cows leads to postparturient haemoglobinuria and postparturient hemoglobinemia.

- Cold water – Ingestion of excessive quantities of cold water in calves leads to intravenous hemolysis and haemoglobinuria
    - Infections
- Bacteria – *Leptospira, Staphylococci, Streptococci* and *Clostridium*
- Virus – Equine infectious anemia and feline infectious anemia
- Protozoa – *Anaplasma, Babesia*, *Ehrlichia canis,* eperythrozoonosis and haemobartonellosis
    - Pathological : Hypersplenism in dogs leads to severe anaemia of macrocytic or normocytic type.
    - Abnormal auto-antibodies to
- malignancy – Lymphatic neoplasms, ovarian tumours and gastrointestinal carcinomas.
- Collagen disease – Disseminated lupus erythematosis
- Idiopathic cause
    - Abnormal iso-antibodies
- Haemolysins in plasma may be produced by incompatible blood transfusion, blood products and in icterus neonatorum.
- In icterus neonatorum, the blood group antigen of the foetus pass into the dam's blood and so isoantibodies are produced.
    - Eg . Icterus neonatorum
    - Incompatible blood transfusion
    - Blood products
    - Pathogenesis : Here break down of hemoglobin occurs at a faster rate.



    - Clinical signs
- Anemia
- Icterus
- Hemoglobinuria
- Dyspnoea
- In postparturient hemogobinuria, signs of phosphorus deficiency are seen viz. pica shifting lameness, decreased productivity and lordosis
- In cold hemoglobinuria in calves, there is cardiac insufficiency.
    - Clinical pathology : Erythrocytes show increased hypotonic fragility

- Gross pathology
- Carcass smells of onion in case of onion toxicity
- Mucosa – Icteric
- Spleen – swollen and darker
- Liver – yellowish and friable; shrunken in later stages.
- Kidneys – Dark brown
- Ur . Bladder – distended with blood coloured urine
- Bone marrow is active
  - In postparturient hemoglobinuria
- Liver – pale and slightly enlarged
- Kidney is black coloured due to deposition of hemoglobin
- Body cavities – contain excessive fluid
- Lung shows edema
  - Hypersplenism in dogs
- Spleen is enlarged
- In cold hemoglobinuria in calves, there is pulmonary edema.
  - Histopathology - In postparturient hemoglobinuria
- Liver – Centrilobular necrosis
- Central vein – thrombus formed by ghost corpuscles
  - Sequelae
- In mild cases, recovery may occur.
- Cold hemoglobinuria in calves – spontaneous recovery occurs
- Hemoglobinuria
- Jaundice
- Increased storage of iron in the form of hemosiderin crystals
- Spontaneous agglutination of erythrocytes
- Death
  - **HAEMORRHAGIC ANEMIA**

- In haemorrhagic anemia, there is extravascular destruction of RBCs and blood loss is greater than production.
- Anaemia depends on
  - Amount of blood cost
  - Rate at which blood is lost
  - Diet controlling the balance between blood loss and production
    - If there is balance between blood loss and production, the blood picture is normocytic- normochromic anemia with many regenerative forms.
    - If the balance of blood loss and production is maintained with difficulty, then the bone marrow works faster and macrocytes appears.
    - Later if there is depletion of iron stores, macrocytic-hypochromic anaemia results with numerous regenerative forms.
    - Ultimately if the bone marrow is exhausted aplastic anemia with normocytic - normochromic anemic picture is observed but with out any regenerative forms.
  - Types
- Acute haemorrhagic anaemia
- Chronic haemorrhagic anaemia
- Purpura and haemorrhagic disease
  - Acute haemorrhagic anaemia
- Acute haemorrhagic anaemia may be due to

- Injury
- Stomach ulcers in pigs; Abomasal ulcers in cows
- Ancylostomiasis and haemonchosis
- Coccidiosis in poultry
- Bovine enzootic hematuria
- Poisoning by warfarin, sweet clover and bracken fern:

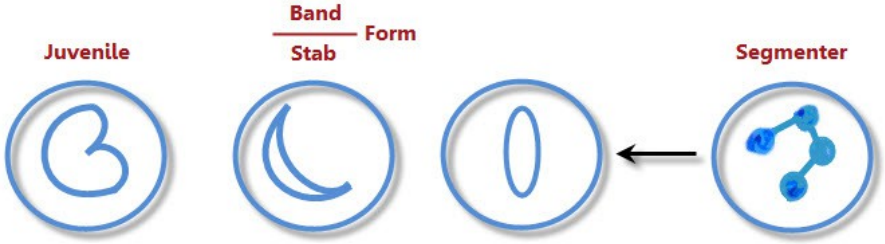
- In bracken fern poisoning, there is acute thrombocytopenia which is the direct cause of hemorrhage.
- Bracken fern is also a cumulative poison and cause granulocytopenia which leads to bacteremia and bacterial embolism
  - Gross pathology
- Gastrointestinal tract shows submucosal haemorrhages which may lead to ulcers
- Myocardium, liver and kidney show haemorrhages.
- In cumulative poisoning, there are haemorrhages and infarcts in the heart and kidneys.
  - Chronic haemorrhagic anaemia
- Chronic haemorrhagic anemia may be due to
  - Ectoparasite - ticks, lice, fleas
  - Protozoa - coccidiosis in dogs
  - Nematode - *Haemonchus, Fasciola, Bunostomum* in cattle and sheep; Strongyles in horses; *Ancylstomum* in dogs.
  - Haemorrhagic disease - Chronic bovine hematuria
  - Gastrointestinal ulcers and vascular tumours
  - Purpura and haemorrhagic diseases
- Purpura is accumulation of blood under the skin due to spontaneous rupture of the capillaries.
- Haemorrhages result even due to mild damage.
- It is a syndrome and not a disease. The causes are
  - Vascular disorders
  - Impaired clotting mechanism
  - Other coagulation defects
  - Unknown causes
  - **VASCULAR DISORDERS**
  - Purpuric infection (Symptomatic purpura)
- This is seen in various diseases characterized by petechiae viz. anthrax, haemorrhagic septicaemia.
- Here the cause is injury to the endothelium of blood vessels by bacterial toxins.
- In viremic diseases like infectious canine hepatitis and hog cholera, the viruses directly damage the endothelium by their multiplication in the endothelial cells.
  - Allergic purpura or purpura haemorrhagica
- This is a symptom of post-infectious toxaemia as in strangles, fistulous withers, poll evil and empyema of guttural pouches.
- Here the defect is due to development of allergy resulting in increased capillary permeability.
- *Gross pathology*
  - Mucous membranes shows haemorrhages
  - Subcutis, peritoneal cavity and muscles show edema
  - Congenital purpura
- Congenital purpura may develop in the foetus.
- Here iso-agglutinins formed against the platelets of the mother pass via placenta and produce thrombocytopenia in the foetus.

- Senile purpura
- This is seen in old men.
- The skin is very much atrophied.
- There is no subcutaneous fat.
- Vessels of skin are easily injured and haemorrhage occurs.
  - Vitamin C deficiency
- In vitamin C deficiency of human beings, there is increased capillary permeability and fragility since cement substance of the capillary wall is not synthesized
  - **IMPAIRED CLOTTING MECHANISM**
  - Impaired clotting might be due to thrombocytopenia.
  - It may occur as
- Primary thrombocytopenia or secondary thrombocytopenia.
  - Primary thrombocytopenia
- It might be due to auto-antibodies formed against platelets.
- Bracken fern poisoning
  - Secondary thrombocytopenia
- Secondary thrombocytopenia may occur in the following conditions
- Damage to the bone marrow
  - By chemicals like nitrogen mustard
  - Individual sensitivity to therapeutic doses of sulphanilamide, quinine, gold salts, oxytetracycline, streptomycin, sodium salicylate, DDT etc.
  - Animal toxins – snake venom and extensive burns
  - By physical agents: ionizing radiation and heat stroke
  - Septicaemic infections
- Myelophthesic replacement as in leukemias
- Hypersplenism causing destruction of thrombocytes
- Aplastic anemia: Here because of complete atrophy of bone marrow, there is no production of platelets.
- Bracken fern poisoning
  - **OTHER COAGULATION DEFECTS**
  - Hemophilia
- Hemophilia is an inherited defect caused by sex linked recessive gene.
- Here the blood does not clot after an injury.
  - Prothrombin deficiency : Prothrombin deficiency may occur due to impaired formation
  - Liver diseases
- In liver diseases, clotting factors like fibrinogen, prothrombin, factors V, VII and IX are not synthesized.
- Deficiency of bile that may occur in hepatic diseases also cause deficiency of vitamin K.
  - Vitamin K deficiency
- In fowls and pigs which are given antimicrobial therapy may have less microbes in the intestine and the fowl and pig may suffer from deficiency of vitamin K due to their poor synthesis by microbes.
- In obstructive jaundice, bile secretion into the intestine is less and so vitamin K can not be absorbed. In turn in vitamin K deficiency may lead to poor synthesis of prothrombin and factor VII.
- In diseases of small intestine as in sprue and colitis, absorption of vitamin K is interfered.
  - Poisoning by dicoumarin, warfarin and sweet clover

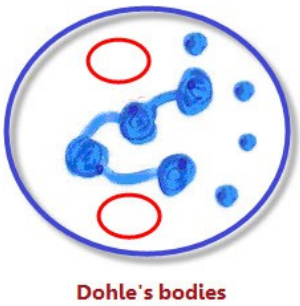
- Sweet clover contains coumarin which is converted into dicoumarol. This is a powerful anticoagulant. It antagonizes the activity of vitamin K and so depresses the formation of prothrombin and factors VII, IX and X.
- Gross pathology
- In cattle and sheep there are extensive haemorrhages under the subcutis, on the serous membranes and in the viscera. Anemia also occurs.
- **Warfarin** which is used as a rodenticide may be accidentally ingested by pets. It is chemically similar to dicoumarol and causes extensive haemorrhages.
    - Excessive production of circulating anticoagulants
- **Heparin** is a powerful anticoagulant and is produced in excessive quantities during anaphylactic shock and so lead to continuous bleeding.
- **Snake venoms** contain anticoagulants and so bites by such snakes lead to fatal bleeding.
    - **UNKNOWN FACTORS**
- Mouldy com poisoning in cattle and swine leads toacute haemorrhages in various parts of the body
- Epistaxis in horses: It is due to a non sex linked recessive character in horses. The affected horses have very thin nasal blood vessels which may rupture due to strenuous exercise.
    - **SECONDARY EFFECTS OF ANEMIA**
- Secondary effects of anemia are due to anoxia and may cause
    - Dyspnoea
    - Tachycardia
    - Rapid fatigue
    - Hyperplasia of hemopoietic tissue: Regenerative forms occur in the blood. Extramedullary hemopoiesis may occur in the liver and spleen
    - Fatty degeneration of parenchymatous organs
    - Edema because of increased capillary permeability
    - Petechiae
    - Compensatory hypertrophy of heart in the early stages. If decompensation sets in then hydropericardium and ascites results.
    - Signs
- Pale skin and mucosa
- Glossitis
- Anorexia
- Flatulence
- Vomiting
- Constipation
- Diarrhoea
- Fever
- Splenomegaly
- Albuminuria
- **PATHOLOGY OF LEUCOCYTES**
- Leukopenia: Leukopenia means decrease in the number of circulating leukocytes.
- Leucocytosis
- Increase in number of leukocytes is called leukocytosis. It is a temporary phase and is useful to the animal. It is a reversible condition.
- Leukemia :Leukemia is cancer of leucopoietic tissue and is progressive, irreversible and fatal.

• ↓ **Leucopenia**	• **Leucocytosis**	• ↑ **Leukemia**
• Temporary	• Cancer, Progressive	•
• Useful	• Fatal	•

- Neutrophilia
- Neutrophilia means increase in the number of neutrophils in the peripheral blood.
- Heterophil is the counterpart of mammalian neutrophil
- *Shift to left*
    - In shift to left, the peripheral blood contains more number of immature neutrophils.
    - This is noticed when bone marrow is stimulated.
    - Schilling has propsed a hemogram to estimate the shift to left.
    - This hemogram has four stages
        - Myelocytes: It is an immature form of neutrophil
        - Juveniles: It is also an immature form of neutrophil in which the nucleus is indented
        - Band form or stab: It is also an immature form of neutrophil in which the nucleus is curved or bent
        - Segmenter: It is the mature form of neutrophil



- *Regenerative reaction*
    - Regenerative reaction is characterized by a little more number of immature forms of blood cells due to increased activity of the bone marrow.
- *Degenerative reaction*
    - Here the immature forms are far more in number than the mature forms.
    - In this condition, there is a depression of maturation of leukocytes in the bone marrow.
    - It is due to a very severe infection.
    - Here the prognosis is unfavourable.
    - In severe infections, neutrophils contain large toxic granules called “Dohle’s bodies”. They are aggregates of rough endoplasmic reticulum.
    - Some neutrophils contain vacuoles which are due to leakage of hydrolytic enzymes released from the ruptured lysosomes under the influence of bacterial toxins.
- *Toxic neutrophils*



- *Basket cells*
    - Ruptured immature neutrophils are called as basket cells.
- *Etiology*
    - Neutrophilia may be seen in the following conditions

        - Acute bacterial infections
        - Acute haemorrhage and hemolysis
        - After surgical operations
        - Malignancy
        - Metabolic conditions like uremia, burns
- Eosinophilia: Eosinophilia means increase in the number of eosinophils in the peripheral blood.
- *Etiology*
    - Eosinophilia is seen in the following conditions
        - Allergic conditions
        - Skin affections – Eczema, Scabies
        - Parasitic infections
        - Following administration of certain poisons and drugs – arsenic, copper, sulpha drugs, chlorpromazine and digitalis
        - Chronic eosinophilic myositis of dogs
        - Following splenectomy
        - Diseases affecting the haemopoietic system -Chronic myelocytic leukemia and Hodgkin's disease
- Lymphocytosis: Absolute increase is rare where as relative increase is common.
- It may be seen in the following conditions.
    - Certain viral infections like influenza
    - Chronic bacterial infections like brucellosis and tuberculosis
    - Convalescence
    - Lymphatic leukemia
    - Following vaccination
- *Smudge cells*
    - Lymphocytes that are damaged during the preparation of smear are called smudge cells.
- Monocytosis : Monocytosis means increase in the number of monocytes in the peripheral blood.
- It is seen in the following conditions
    - Protozoal disease – Trypanosomiasis
    - Ehrlichiosis in dogs
    - Chronic bacterial disease – Tuberculosis and brucellosis
    - Convalescence following acute diseases
    - Monocytic leukemia
- Agranulocytosis
- Agranulocytosis means complete disappearance of granulocyte series of leukocytes from the peripheral blood.
- In addition there is also leucopenia.
- *Etiology*
    - It may occur in case of suppression of leucopoiesis and in inhibition of maturation of the granulocyte series in the bone marrow.
        - Toxic chemicals and drugs acting on the bone marrow like benzol, barbiturates and arsenical preparations
        - x- ray irradiation
        - Bacterial toxins of *Staphylococcus aureus, Streptococcus hemolyticus* and *Streptococcus viridans*
        - Feline pan leucopenia causes total destruction of myelopoiesis
    -

- *Histopathology*
    - Bone marrow initially shows hyperplasia of the bone marrow and later myeloid hypoplasia in chronic cases.

- **LEUCOPENIA**

- Definition: Leucopenia means reduction in the number of leucocytes in the peripheral blood.
- Etiology
- Leucopenia is observed in the following conditions
    - Diminished production of leucocytes
    - Increased destruction of leucocytes
    - Altered distribution of leucocytes
- Diminished production
- Physical agents – Ionising radiation, X- rays
- Chemical agents – Benzol, nitrogen mustard
- Certain bacterial infections – Brucellosis and tuberculosis
- Viral diseases – Rinderpest, Canine distemper, Infectious canine hepatitis and mucosal disease
- Fungal diseases – Histoplasmosis
- Rickettsial diseases – Tick borne fever
- Cachectic states and starvation
- Metabolic disturbances – Hypothyroidism and hypopituitarism
- Hemopoietic disorders – Myelophthesic and aplastic anemia
- Bracken fern poisoning
- Unknown mechanism – Cirrhosis of liver
- Increased destruction
- Physical agents – Large doses of ionizing radiation
- Loss of leucocytes in pus and inflammatory exudates
- Bacterial toxins – *Clostridium welchii* and *Pasteurella*
- Protozoal infections - *Theileria parva*
- Destruction by leucocytic antibodies which occur when amidopyrene is administered
- Hypersplenism as in tuberculsis and Hodgkin's disease
- Altered distribution
- In anaphylactic shock the leucocytes are trapped in the sinuses of the liver, spleen and lungs
- In stress, liberated cortisone causes eosinophilia and lymphopenia

- **LEUKEMIA**

- Introduction
- Leukemia is aprimary neoplastic disease of the bone marrow and other reticuloendothelial tissues.
- Definition : Leukopenia means decrease in the number of circulating leukocytes.
- Occurrence
- Leukemia is not so common in animals as in man.
- Leukemia affecting lymphopoietic tissue is more common in animals
- Lymphatic leukemia is common in bovines and dogs.
- Types
- When the neoplastic cells flood the blood, then it is called leukemic leukemia.
- When the blood picture is relatively normal then the condition is called aleukemic leukemia

- Neoplasm of the granulopoietic tissue is called granulocytic leukemia. It is further classified based on the cell type involved as neutrophilic, eosinophilic and basophilic types.
- Neoplasm of the lymphocytic tissue is called lymphatic or lymphocytic leukemia.
- Neoplasm involving the monocytes is called monocytic leukemia
- If the lymph nodes are not affected but the liver and spleen are enlarged and the smear shows numerous myeloblasts then the condition is termed as myeloid leukemia
- If the bone marrow is normal but the lymph nodes show neoplasms then the condition is known as lymphatic leukemia
- Neoplasms involving the reticulum cells is called reticulum cell sarcoma and giant follicular lymphoma
- Lymphocytic leukemia
- In animals the condition is also called lymphocytoma, malignant lymphoma, lymphomatosis or lymphosarcomas.
- Involvement of all the lymph nodes indicates a multicentric origin.
- Metatasis are found in almost all the organs where the normal structure is replaced by neoplastic ells.
- Clinical signs
- Laboured breathing
- Anemia
- Gradual loss of condition
- Digestive signs if the liver and abomasum are involved
- Paraplegia if brain and spinal cord are affected
- Clinical pathology
- In the circulating blood immature forms of leucocytes or blast cells are frequently found (It is impossible to say to which cell type the precursor blast cells belong)
- In dogs with lymphocytic leukemia, leukemic stage is absent hence blood picture shows only moderate neutrophilia
- Gross pathology -Lymphatic leukemia
- Superficial lymph nodes are enlarged
- Mucous membranes are pale
- Splenomegaly and hepatomegaly are often seen in dogs
- Tumour masses are found in many lymph nodes and internal organs
- Sequelae : Leukemia is always fatal
-

-

- **LYMPHOID SYSTEM**

- **PATHOLOGY OF LYMPH NODES**

- **LYMPHANGITIS**

- Definition: Inflammation of lymph vessel is called lymphangitis
- Types
- Simple Lymphangitis
- Purulent Lymphangitis
- Simple lymphangitis
- *Etiology*
    - Extension of inflammation
    - *Histoplasma farciminosum* infection which occurs as epizootic
    - Seen in lungs in case of pneumonia as in swine fever, tuberculosis and contagious bovine pleuropneumonia in cattle and bronchopneumonia in dogs and horses.

- Johne's disease
- Actionomycosis
- *Corynebacterium ovis* which causes ulcerative lymphangitis

- *Signs*
  - The affected vessels are painful to touch
  - Edema in the nearby area
  - The nearest lymph gland that drains the area is enlarged and painful
- *Pathogenesis* : The occluded vessels prevents drainage of lymph and hence edema occurs
- *Gross pathology*
  - The inflammed lymphatics appear as thickened and reddish streaks
  - In subcutis, they appear as thick cords
- *Histopathology* : The vessels are dilated and the wall is infiltrated with leucocytes.
- Purulent lymphangitis
- It is associated with suppurative condition of the tissue drained.
- The affected vessels contain thrombus and the wall is intensely infiltrated with leucocytes

- **TUMORS OF VASCULAR SYSTEM**

- The common tumours of vascular system include haemangioma and lymphangioma.

- **LYMPH NODES**

- Anatomical Features of Lymph Nodes
- Lymph nodes have cortex and medulla
- Ruminants have hemal lymph nodes
- Chicken do not have lymph nodes
- Physiological considerations : Lymph nodes act as a filter for foreign particles.

- **DISTURBANCES OF GROWTH**

- Hypoplasia
- *Etiology*
  - Hypoplasia of lymph nodes is seen in
    - Infection
    - Toxic agents
    - Hormonal mechanisms
    - Stress
- *Histopathology* : In alarm reaction of stress, diffuse dissolution of lymphocytes is seen
- Atrophy
- *Etiology*
  - Atrophy of lymph nodes is seen in
    - Starvation
    - Senility
    - Chronic wasting disease
    - Viral infections
    - Ionizing radiation
    - Stress

        - Excessive doses of – adrenal cortical hormones and sex hormones
- *Clinical pathology* : In atrophy of lymph nodes lymphopenia is seen
    - Hyperplasia
- *Etiology*
    - Hyperplasia of lymph nodes is seen in
        - Reaction to subcute to chronic type of irritants
        - Chronic enteritis, chronic pneumonia and canine distemper as a generalized or local phenomenon.
- *Gross pathology*
    - The affected lymph nodes are whitish-gray, enlarged and firm
    - The follicles are prominent
- *Histopathology*
    - There is great enlargement of the germinal centres with zone mature lymphocytes surrounding them.

DEGENERATIVE CHANGES

    - Amyloid degeneration
- In general Amyloidosis, amyloid may be found in the lymph nodes
- Deposition of Amyloid starts in the germinal centres and spreads outwards

NECROSIS

- Necrosis of the whole or part of a lymph node may occur
    - Etiology
- When infectious agents grow locally as in anthrax and erysipelas, necrosis of the lymph nodes draining the affected area occurs.
    - Gross pathology
- Necrotic area is dry and circumscribed.
- In some infectious gas bubbles may be seen

CIRCULATORY DISTURBANCES

    - Hemorrhages
- *Etiology*
    - Hemorrhages are seen in the lymph nodes in the following conditions
        - Local trauma
        - Passive venous congestion
        - Severe infectious diseases
        - Haemorrhagic diathesis
- *Gross pathology*
    - Lymph nodes show petechiae, focal or diffuse reddened areas (haemorrhages)
    - Hemal lymph nodes should not be confused with hemorrhagic lesions.
- *Histopathology*: Erythrocytes may replace the normal lymphocytes

GAS ACCUMULATION

    - Emphysema
- *Occurrence*
    - Mesenteric lymph nodes show emphysema in association with intestinal emphysema in pigs.
    - Bronchial nodes show emphysema in pulmonary interstitial emphysema in cattle.
- *Gross pathology*
    - The affected lymph nodes are enlarged, soft and puffy.
    - The cut surface looks like a sponge.
- *Histopathology*

- Lymph node sinuses contain vesicles.
- The sinus endothelial cells become macrophages and even giant cells.
- These cells occurring as clusters cause pressure atrophy of the lymphoid tissue.

PIGNMENTATIONS

- **Exogenous Pigmentation**

- Exogenous pigmentation of lymph nodes is common in pulmonary and mesenteric lymph nodes.
- These pigments are not of clinical significance.
  - Anthracosis
- Coal dust is common in the bronchial and mediastinal lymph nodes of dogs which live in industrial or highly polluted areas and in pit ponies.
- The coal particles are found in the macrophages of the medullary cords
  - Ingested pigments
- In ruminants - Mesenteric and other nodes develop a grey exogenous pigmentation of the medulla due to some pigments in the ingested feed.
- Tattoo pigments – Regional lymph nodes contain the tattooed pigments
  - **Endogenous Pigmentation**
- Endogenous pigmentation includes hemosiderin, bile pigments and melanin.
  - Hemosiderin
- Hemosiderin is the most common endogenous pigment.
- Occurrence
  - Hemodsiderin pigment is seen in the lymph nodes draining the areas where hemorrhage has occurred.
- Gross pathology : The affected lymph nodes are brownish in colour.
- Histopathology
  - The reticular and sinusoidal macrophages contain the brownish amorphous crystals of hemosiderin.
  - Bile pigments : Bile pigments may be found in the hepatic lymph nodes.
  - Melanin pigments : Melanin pigments may seen in superficial lymph nodes of old grey horse.

LYMPHADENITIS

- Definition: Lymphadenitis means inflammation of the lymph nodes.
- Etiology: Lymph node is usually affected by any irritant that may be present in area it drains.
- Types
- Acute serous lymphadenitis
- Haemorrhagic lymphadenitis
- Suppurative lymphadenitis
- Chronic lymphadenitis
- Specific lymphadenitis
- Nonspecific lymphadenitis
  - Acute serous lymphadenitis
- *Etiology*
  - Acute serous lymph adenitis is seen in lymph nodes draining acutely infected or inflammed areas

    - In some septicaemic diseases, the lymph nodes through out the body are affected
    - e.g. anthrax, haemorrhagic septicaemia, swine erysipelas and hog cholera
    - Mesenteric nodes are affected by the absorption of irritants from the gastrointestinal tract.
- *Gross pathology* : The affected lymph node is enlarged, moist and reddish.
- *Histopathology*
    - The affected lymph node shows
        - Hyperemia
        - Edema
        - Proliferation of lymphatic parenchyma and reticuloendothelial tissue
        - Infiltration of mononuclear cells and a few neutrophils in lymph sinuses
    -
    -
    - Haemorrhagic lymphadenitis
- *Etiology*
    - Haemorrhagic lymphadenitis occurs when there is stronger irritant than the serous variety
    - e.g. Anthrax, Theileriasis
- *Gross pathology* : The cut section of the gland reveals bloody fluid
- *Histopathology* : Micoscopically the lymph node sinuses contain large number of erythrocytes
    - Suppurative lymphadenitis
- *Etiology*
    - Suppurative lymphadenitis is caused by pyogenic organisms.
    - e.g. *Streptococus equi* in horses (Strangles), *Corynebacterium ovis* in sheep (Caseous lymphadenitis)
- *Gross pathology* : The affected lymph node shows abscesses
- *Histopathology*
    - The lymph node in the affected area contain liquefied necrotic material along with infiltration of leucocytes especially neutrophils.
    - Several small purulent foci may be present which may coalesce to form large abscesses.
    - Chronic lymphadenitis
- *Etiology* : Chronic lymph adenitis is seen in Johne's disease
- *Gross pathology*: The affected lymph nodes (mesenteric lymph nodes) are large, hard,dry.
- *Histopathology*
    - The affected lymph node shows hyperplasia of reticulum cells
    - Numerous endothelial cells are swollen, rounded, cast off and fallen into the lymph sinuses that are much distended (Sinus catarrh)
    - Macrophages become predominant cells
    - Fibrosis is also evident
    - Reactive hyperplasia of lymph node is also present
    - Specific lymphadenitis
- Lymphadenitis is characteristically seen in the following diseases

- In tuberculosis, Johne's disease, glanders and actinobacillosis granulomatous lymphadenitis with caseation and calcification is present.
- Strangles in horses,
- Caseous lymphadenitis in sheep
- Bovine lymphangitis and lymphadenitis caused by *Pasterurella pseudotuberculosis rodentium*
- Brucellosis in guinea pigs
- Epizootic lymphangitis
- Tularemia in rodents
- Nonspecific lymphadenitis

- *Etiology*
  - Irritant chemicals
  - Toxins form trauma, burns and bacteria
  - **NEOPLASMS**
  - Primary tumours
- Primary benign tumours of lymph nodes are not common
- Primary malignant neoplasm i.e. Lymphosarcoma is common
  -
  - Secondary tumours
- Common tumours involving lymph nodes are carcinoma and malignant melanoma
- Occasionally sarcoma is seen
  -
  - **PATHOLOGY OF SPLEEN**
  - ANATOMICAL FEATURES OF SPLEEN

- Location: Left side of abdominal cavity
- Position: Visceral surface is related to rumen and reticulum
- Shape: Round in fowl; Oblong in cattle, Socks shaped in dog; Sythe shaped in horse Oyster shell shaped in sheep and goat; Spear shaped in cat.
- Size (Weight): Cattle - 900 gms; Horse - 1000 gms; Dog - 50 gms
- Colour: Bright red (Dog), reddish brawn (fowl) bluish red (cattle and horse).
- Surface: Capsule – contractile and muscular
- Parietal – convex
- Visceral – Concave
- Consistency: Soft
- Ends: Dorsal; Ventral end is wider in dogs. Ends are called as base and apex in horse
- Border: Anterior border is concave and posterior border is convex in horse
- Attachments: Spleen is attached to stomach by gastrosplenic omentum and to diaphragm by suspensory ligament.
- Blood supply: Spleen gets the blood supply through the splenic artery and venous flow by splenic vein.
- Nerve supply: Spleen has splenic plexus for its nervous connection
  - FUNCTIONS OF SPLEEN

- Storage organ for blood as in horse, dog and cat
- Graveyard of erythrocytes – Conversion of haemoglobin to bilirubin and storage of iron
- Lymphoid organ

- Produces extramedullary haemopoiesis in severe anemia
- Produce antibodies by plasma cells
- Helps in phagocytosis of worn out blood cells and foreign particles
    - **GROWTH DISTURBANCES**
        - Accessory spleen
- Accessory spleens are acquired during life.
- They are produced through the traumatic rupture of spleen.
- The implanted pieces may be noticed in the gastrosplenic omentum.
    - Labulated spleen
- Lobulated spleen is a primitive stage in the development of spleen which is arrested from its development through the foetal growth.
    - Doughnut spleen
- Doughnut spleen is a circular spleen with a hole in the middle.
- Noticed rarely in horse
    - Aplasia & Hypoplasia of spleen : Sometimes noticed
    - Atrophy of spleen
- Atrophy of lymphoid tissue may take place.
- Chronic wasting disease
- Viral infections
- Ionizing radiation
- Chronic stasis of blood leads to induration (fibrosis) and finally atrophy of spleen. The spleen is much reduced in size. Here the capsule appears shriveled.
- The parenchyma is scanty and Malpighian corpuscle are not visible. This is seen in swine, dogs and cats.
- Such newly formed hyperplastic nodules do not contain the central arteriole.
    - Hyperplasia of spleen
- Focal hyperplasia of spleen is commonly observed in old dogs.
- Grey, round, soft projecting nodules (Nodular hyperplasia) are seen on the surface of spleen.
    - **DEGENERATIVE CHANGES**
        - Hyaline degeneration : Hyaline degeneration is seen in the wall of the arterioles
        - Amyloid infiltration
- Occurrence : Amyloid infiltration occurs as part of the generalized Amyloidosis
    - **Types**
    - Focal amyloidosis
- Occurrence : Focal amyloidosis is more common in occurrence
- Portion of spleen affected
    - In focal amyloidosis, the central arteries of the Malpighian corpuscles are affected.
- Gross pathology
    - The affected arteries of spleen are prominent, pale, and translucent and stand out against a red back ground, like boiled sago hence it is called “sago spleen”.
    - Diffuse amyloidosis
- Portion of spleen affected
    - In diffuse Amyloidosis, the arterioles and fibres of the reticuloendothelial system are affected.
- Gross pathology
    - The spleen is enlarged with rounded edges and the cut surface is smooth and translucent.

- As the spleen looks like bacon it is called " bacon spleen "
- **PIGMENTATIONS**
- Hemosiderosis

- Normally hemosiderin is stored in the spleen in the reticular macrophages.
- Increased amounts of hemosiderin pigments may be seen in hemolytic anaemias.
- Haemosiderin is stained bluish with Perl's stain

- **RUPTURE OF SPLEEN**
- Occurrence : Rupture of spleen is common in dogs
- Etiology

- Rupture of spleen may be seen in
  - Automobile accidents
  - Sharp blows in abdomen
  - Splenomegaly due to congestion Amyloidosis, hyperplasia or tumours
  - Gross pathology : Spleen may be divided in to two pieces
  - Sequelae
- Hemorrhages into the peritoneum may lead to fatal results
- Healing leads to formation of scars

- **CIRCULATORY DISTURBANCES**
- Congestion: Due to wide variation in size, congestion of the spleen is difficult to interpret.
- Acute congestion

- Acute congestion is common in acute infectious diseases and in acute bacterial intoxication as in enterotoxaemia
  -
  - Acute passive congestion
- *Etiology*
  - Acute passive congestion of the spleen is seen in
    - General venous congestion
    - Euthanasia by barbiturates
    - Paralysis of splanchnic nerve
- *Gross pathology*
  - Spleen is very much enlarged and soft
  - On section the cut surface bulges and dark blood oozes
  - Chronic passive hyperaemia
- *Occurrence* : Not common among animals
- *Etiology*
  - Chronic passive hyperemia may be seen in
    - Chronic general venous congestion
    - Cirrhosis of liver leading to congestion of portal vein
    - Partial or complete of venous return as in
      - Thrombosis or pressure by cysts, tumours and abscesses on veins draining the spleen
      - Torsion of stomach and spleen in dogs
      - Torsion of splenic ligament in the pigs
- *Gross pathology* : The spleen is moderately enlarged and firm
- *Histopathology*
  - There is gradual replacement of pulp cells and follicles by fibrous tissue.
  - Trabeculae also have more fibrous tissue.
  - Haemosiderin accumulates in the phagocytes.

- Thrombosis of splenic veins
- *Occurrence*
  - Thrombosis of splenic veins is rare.
  - Occasionally it is seen in
  - Traumatic reticulitis and portal thrombosis in cattle
  - Extension from parasitic abscess in horses
  - Embolism of splenic artery
- Valvular vegetations may lead to embolism of splenic artery and its braches with subsequent infarction.
  - Infarction
- *Occurrence* : Infarction of the spleen is common
- *Etiology*: If the artery is occluded by an embolus, the whole organ may undergo infarction.
- *Pathogenesis*
  - Proliferation of endothelial cells in hog cholera leads to occlusion of follicular braches of the spleen with resultant haemorrhagic infarcts.
- *Gross pathology*
  - The infarct is red in the beginning but later the base turns pale with the diffusion of haemoglobin
- *Histopathology*
  - At the infarcted area the lymphoid cells are either necrotic or replaced by erythrocytes.
  - Haemorrhages
- In dogs, due to automobile accidents, if rupture of spleen does not occur than the blood may collect under the capsule giving rise to subcapsular haematomas.
  - **HYPERSPLENISM**
  - Definition : Hypersplenism is a condition in which there is excessive activity of phagocytes.
  - Occurrence : This condition may or may not be coexisting with splenomegaly
  - Etiology
- Depression of bone marrow function or inhibition of the maturation of cells of bone marrow by spleen through some hormonal influence
- Antibody formation against blood cells leading to destruction of RBCs, WBCs and thrombocytes
- Hypersequestration: In this condition there is increased blood stasis leading to increased fragility of erythrocytes. Such red cells are easily phagocytosed.
  -
  - Sequelae
- In this condition there is excessive
  - hemolysis resulting in hemolytic jaundice
  - Leucocytolysis resulting in leucopenia
  - Thrombocytolysis resulting in thrombocytopenia and so purpura
  - **SPLENOMEGALY**
  - Definition : Splenomegaly means enlargement of the spleen
  - Etiology
- Chemicals – Barbiturates, chloroform
- Bacteria – Anthrax, salmonellosis, erysipelas (in swine) and tuberculosis and spirochaetosis
- Virus – Equine infectious anaemia

- Protozoal diseases – Anaplasmosis, babesiosis, theileriasis, eperythrozoonosis
- Ehrlichiosis in dogs
- Fungus – Histoplasma
- Pathological – Torsion of spleen, acute congestion in bacteremic and toxaemic condition, congestive heart failure, cirrhosis and extramedullary hemopoiesis occurring due to destruction of bone marrow
- Tumours- metastatic melanoma in horses, haemangiomas, haemagiosarcomas, lymphomatosis and myelogenous leukemia in dogs and lymphoid leucosis in fowls
    - Pathogenesis
- In cirrhosis, toxins from the intestine are not detoxified and hence enter the blood circulation and thence to spleen where the spleen is stimulated to produce antibodies.
    - Histopathology
- There is
    - marked hyperplasia of the reticuloendothelial system as well as white pulp
    - diffuse infiltration of neutrophils
    - stasis of blood and resultant fibrosis (in congestive heart failure)
    - Myeloid metaplasia – seen in extramedullary haemopoiesis
    - Stimulation of plasma cell production for antibody formation
    - **SPLENITIS**
    - Definition: Splenitis means inflammation of the spleen.
    - Types: Splenitis may be acute or chronic.
    - Acute splenitis
- *Etiology*
    - Acute splenitis is seen in
    - Acute generalized infectious diseases as in
        - Salmonellosis and swine erysipelas
        - Infectious anaemia of horses
        - Anaplasmosis and eperythrozoonosis
- *Pathogenesis* : In acute splenitis, the infectious organisms grow in the spleen
- *Gross pathology*
    - The spleen is enlarged, dark and soft.
    - Splenic pulp is fluid in consistency
    - Abscesses may be seen in infection by pyogenic organisms
- *Histopathology*
    - Necrosis of the pulp
    - Sinusoids are congested and contain neutrophilic infiltration
    - In the red pulp there is proliferation of reticular cells and macrophages
    - There is certain amount of proliferation of lymphocytes as well as plasma cells
    - Chronic splenitis
- *Etiology*
    - Chronic splenitis is seen in chronic diseases like tuberculosis, Pseudotuberculosis, glanders, actinomycosis and histoplasmosis
- *Gross pathology*: The spleen is enlarged, firm and tough.
- *Histopathology* : Abscesses or chronic granulomatous foci may be seen
    - **TUMORS**
- Primary neoplasms of the spleen are rare. Lymphomas, haemangiomas, lymphosarcomas fibrosarcomas may be encountered.
- Secondary metastasis are not common since “Spleen is a poor soil for the growth of tumours”

- **HARDERIAN GLAND**
- **Introduction**: Harderian gland in chicken is a lymphoid organ present on the medial side of eye balls.
- **Pathology** : It is affected in infectious bursal disease leading to plasma cell depletion and atrophy.
- **BURSA OF FABRICIOUS**
- **Pathology of bursa of Fabricius**

- Bursa of Fabricius is primarily affected in infectious bursa disease in chicken.
- The bursa is enlarged, edematous, haemorrhagic or may contain cheesy material.
- Microscopically, the bursa may show lymphoid depletion, edema, haemorrhage, heterophilic infiltration,necrosis, cystic changes, glandular transformation, atrophy and indentation of surface epithelium.

- **URINARY SYSTEM**
- **NORMAL STRUCTURE OF KIDNEYS**
- Colour: Reddish brown (normal)/ Yellow in cats
- Normal location: Sublumbar
- Normal shape: Bean shaped
- Normal surface: smooth unlobulated / monolobular – canine
- Histological consideration: No tissue is found between the tubules
- **ANOMALIES OF KIDNEY**
- Ectopic kidney

- Location : Inguinal region, Pelvic cavity
- Occurrence : Pigs and dogs
- Sequelae : Ureteral obstruction leads to secondary hydronephrosis
    - Fused kidneys: Fused kidneys look like horse shoe
    - Persistent Lobuation of kidneys
- Normal in foetal life
- Occurrence : Dogs, sheep and swine
    - Renal Agenesis : Renal agenesis means absence of one or both the kidneys
    - Renal Aplasia
- It is usually found at necropsy
- It is seen in beagle and Doberman breeds of dogs

- If unilateral, the condition is compatible with life
- If bilateral it is usually incompatible with life.
    - Renal Hypoplasia
- Definition
    - Renal hypoplasia means that the affected kidney is smaller and the unaffected kidney shows compensatory hypertrophy.
    - Criteria for considering the kidneys are hypoplastic include absence of acquired disease, 50% reduction in size, 1/3 rd reduction in mass and reduced number of glomeruli (5-12 glomeruli / LPF normal: 30-35)
- Occurrence : Cattle, pigs and foals
- Sequelae : If unilateral, the other kidney undergoes compensatory hypertrophy
    - Duplication of kidneys
- Three kidneys may be seen
- Occurrence - Pigs
    - Renal Cysts
- Common congenital defect seen in calf, pigs, lambs, cat, dog and foal.
- Etiology
    - Tubular obstruction
    - Weak tubular basement membrane
- Pathogenesis
    - Cysts in kidneys arise due to lack of continuity between the nephron and collecting ducts and so urine formed in the nephron is not evacuated but collects to form the cyst.
- Gross pathology
    - The kidneys may contain one or more cysts. Kidneys with many cysts are known as congenital polycystic kidney.
    - Ureteral Aplasia
- Occurrence
    - Rare
        - Ureter empties into urethra / vagina, neck of bladder, prostate or vas deference.
    - Persistent (Patent) Urachus
- Occurrence: Foals
- Signs: Dribbling of urine
- Sequelae: Cystitis
    - Bladder Diverticulum : Cystitis and cystic calculi
    - **FACTORS AFFFECTING RENAL FUNCTIONS**
    - Extrarenal Factors Interfering Kidney Function
- Hemoconcentration
- Low blood pressure
- Obstruction to flow of urine
    - Intrarenal Factors Interfering Kidney Function
- Injury to glomeruli
- Injury to tubules
- Alteration in blood supply
    - **ERRORS IN RENAL FUNCTIONS**
    - Proteinuria
- *Definition*
    - Proteinuria means presence of protein (mainly albumin) in urine. The resultant hypoproteinemia causes renal edema.

- *Etiology*
    - Increased permeability of glomerular capillaries
    - Amyloidosis
    - Tubular injury -Nephrosis
    - Renal infarction
    - Nephritis - Glomerulonephritis
    - Congestive heart failure
    - Glycosuria
- *Definition* : Glycosuria means presence of glucose in urine.
- *Etiology*
    - Diabetes mellitus
    - Enterotoxaemia
    - Intravenous injection of large quantities of dextrose
    - Injection of ACTH
    - Rabies
    - Ketonuria
- *Definition* : Ketonuria means presence of ketone bodies in urine.
- *Etiology*
    - Diabetes mellitus
    - Acetonemia / Ketosis of cattle
    - Pregnancy toxemia in ewes
    - Starvation
    - Anuria
- *Definition* : Anuria means absence of urine formation
- *Etiology*
    - Glomerulonephritis
    - Tubular degeneration
    - Extensive destruction of tubular epithelium
    - Urinary obstruction
    - Extreme dehydration
    - Decreased blood pressure
- *Pathogenesis*
    - In glomerulonephritis, there is swelling of capillary endothelium and infiltration of inflammatory cells. These changes cause compression of capillaries of glomeruli and so blood flow through them is blocked. So urine is not filtered.
    - In tubular degeneration, fatty changes and cloudy swelling of tubular epithelial cells cause increase in pressure within the kidneys which compresses the blood vessels.
    - In urinary obstruction, the stagnated urine causes back pressure to the kidneys which in turn opposes the filtration pressure and thus the formation of urine.
    - In Extensive destruction of tubular epithelium, the filtered urine passes through the tubules in to the lymphatics and veins.
    - Oliguria
- *Definition* : Oliguria means reduced excretion of urine
- *Etiology*
    - Glomerulonephritis
    - Tubular degeneration
    - Urinary obstruction

    - Extreme dehydration
    - Decreased blood pressure
- *Pathogenesis* : As in anuria but of lesser magnitude
    - Polyuria
- *Definition* : Polyuria means excessive passage of urine
- *Etiology*
    - Diabetes mellitus
    - Diabetes insipidus
    - Tubular degeneration
    - Chronic interstitial nephritis
    - Hypercalcemia
    - Hypomagnesemia
    - Pyuria
- *Definition* : Pyuria means presence of pus in urine
- *Etiology* : Suppurative inflammation of kidneys or urinary passage
    - Hematuria
- *Definition* : Hematuria means presence of blood in urine
- *Etiology*
    - Trauma / Calculi
    - Chemicals – Phenol, turpentine
    - Septicemia – Haemorrhagic septicemia, anthrax
    - Parasites – *Dioctophyma renale*
    - Inflammatory reaction - acute nephritis, pyelonephritis, cystitis and urethritis
    - Renal infarction
    - Chronic bovine hematuria
    - Neoplasm – carcinoma of bladder or kidneys
- *Signs* : Urine is coloured red
- *Pathogenesis*
    - Hematuria is due to haemorrhage from any part of urinary system – from glomeruli to urethra
    - Haemoglobinuria
- *Definition* : Haemoglobinuria means presence of haemoglobin in urine
- *Etiology*
    - Haemoglobinemia is seen in babesiosis, leptospirosis and infection by Clostridium and Streptococci
    - Chemicals – Chronic copper poisoning and potassium chlorate poisoning
    - Post parturient haemoglobinuria in cattle
- *Signs* : Urine is brown or coffee coloured
- *Pathogenesis*
    - Here the red blood cells are lysed and so haemoglobin diffuses out and enters the glomerular filter.
    - The red blood cells can not be sedimented upon standing of urine.

CIRCULATORY DISTURBANCES

    - Hyperemia
- *Etiology*
    - Active hyperemia is seen in
    - Acute – Nephritis
        - Septicemia
        - Bacterial intoxication

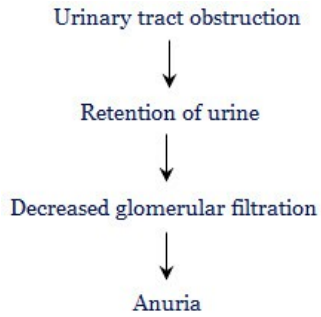
- *Gross pathology*
    - The kidneys may be slightly enlarged
    - Cut surface – Ooze blood
    - Congestion
- *Etiology*
    - Hypostatic
    - Generalized passive congestion
    - Medullary congestion – Prominent
    - Edema
- *Occurrence*
    - As the capsule is inelastic and the parenchyma is firm, edema of kidney is not common.
- *Etiology* : Inflammatory edema may be seen in acute interstitial nephritis
    - Haemorrhage
- *Etiology*
    - Vasculitis
    - Vascular necrosis
    - Direct trauma to kidney
    - Petechiae
    - Cortical surface
- *Etiology*
    - Seen in
        - Swine fever
        - African swine fever
        - Swine erysipelas
        - Streptococcosis
        - Salmonellosis
    - Ecchymoses
- *Etiology* - Herpes virus infection in neonatal puppies
    -
    -
    - Extravasations
- *Etiology*
    - Direct trauma
    - Bleeding disorders – Hemophilia, disseminated intravascular coagulation
    - Infarct
- *Etiology*
    - Cows
        - Thrombosis of the uterine veins after parturition
        - *Corynebacterium pyogenes* and Streptococcal infection
    - Pig
        - Erysipelas infection
- *Occurrence*
    - Very common in cattle especially cows
    - Anaemic (Pale) type is common
- *Gross pathology*
    - The infarcts are wedge shaped with the base towards the cortex and the apex towards the medulla or pelvis.
- *Sequelae*

- If the condition is not septic, the infarct will be healed by scar tissue formation and the surface will be pitted.

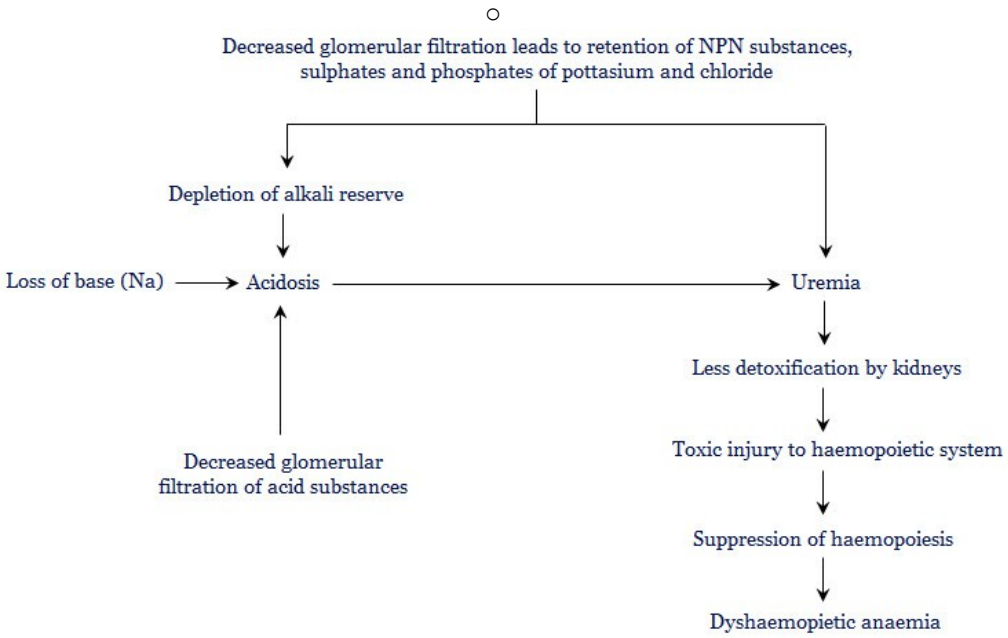
UREMIA

- Definition

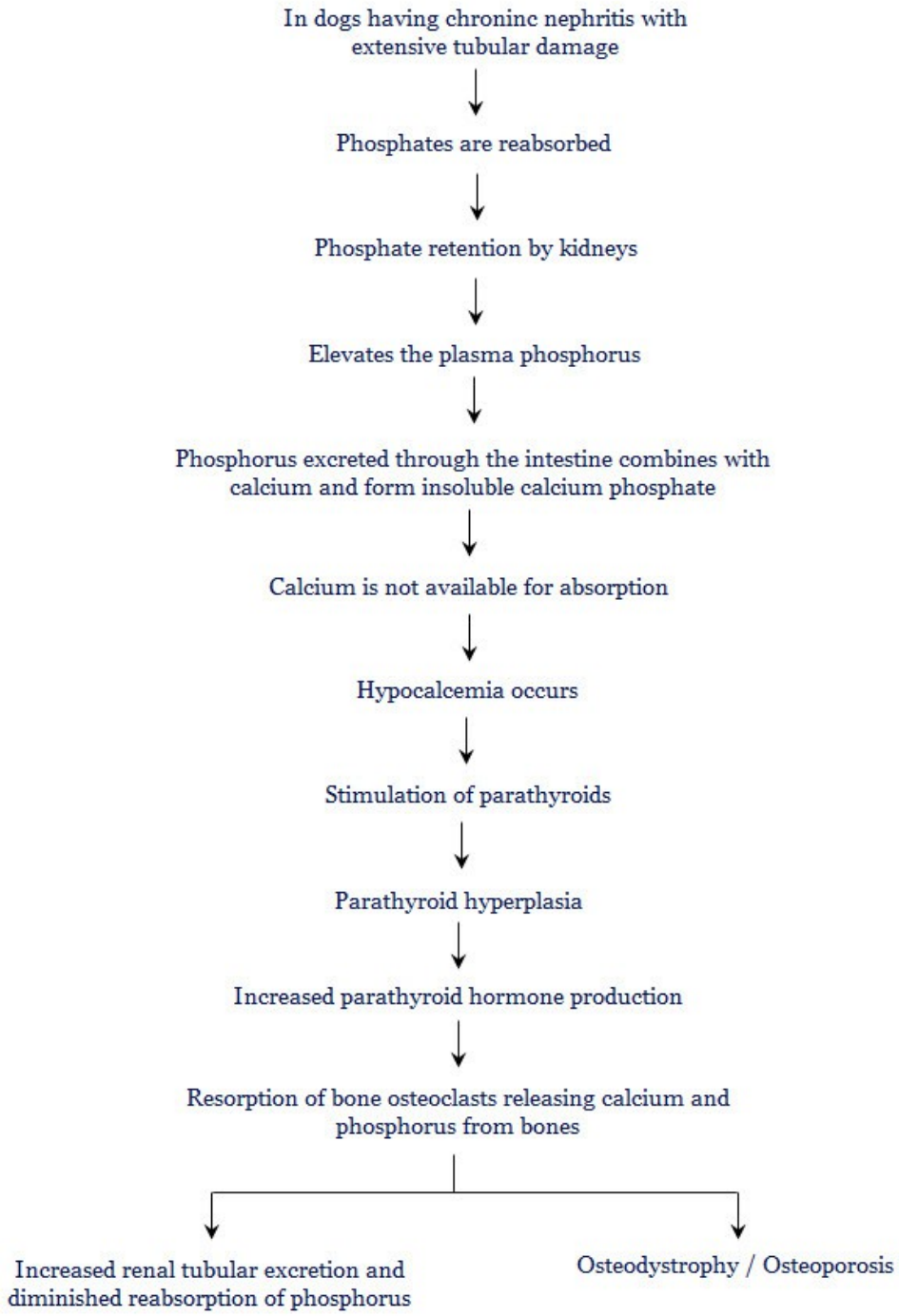
- Uremia is a toxaemic syndrome resulting from renal insufficiency
- This is due to toxic action by-retained non-protein nitrogenous substances (NPN) Substances in blood (Azotemia) and partly to acidosis. The NPN substances include urea, creatine, uric acid and ammonia.
    - Occurrence
- Common – Nearly 5 % of all dogs examined at autopsy have some kind of uremia
- This is more common in males than in females in all species of animals
    - Etiology
- *Pre-Renal*
    - Here waste products of protein catabolism are retained in the blood which may occur in the following conditions.
    - Low Blood Pressure: Decreased glomerular filtration may occur in trauma, shock, intestinal haemorrhage and severe dehydration.
    - Increased protein catabolism – High protein intake and followed by rapid breakdown of proteins, gangrene, diabetes mellitus, fever, large infarcts
    - In conditions like vomition, diarrhea, excessive sweating and intestinal obstruction, there is salt deficiency, dehydration and electrolyte imbalance
- *Renal*
    - Decreased glomerular filtration occurs in glomerulonephritis and extensive amyloidosis
    - Decreased tubular resorption occurs in toxic tubular nephrosis and chronic interstitial nephritis
    - Decreased tubular secretion - Hyperkalemia → Acidosis
    - Toxic tubular necrosis
    - Decreased detoxifying mechanism
    -
-
    -
- *Post-Renal*
    - The urinary tract may be obstructed by calculi, post inflammatory strictures, carcinoma of bladder, prostatic enlargements and expanding pressure by tumours over the urinary passage.



    - Pathogenesis



- Dyshaemopoietic anaemia may also be caused by decreased intake of iron and vitamin B -12 due to anorexia and vomiting.
- Decreased tubular resorption causes loss of water and electrolytes
- Damaged kidney → Increased renin production → Release of hypertensin → Acts on plasma hypertensinogen → Increased blood pressure → Left side cardiac hypertrophy
- This is only an attempt by nature but in chronic renal disease the damage is irreparable with more and more phosphorus retention and consequent hyperparathyroidism and bony lesions.
- As the bone calcium is removed the bone becomes soft. But nature tries to strength the bone by producing the cheaper fibrous tissue which becomes hyperplastic. This is more evident in the bones formed by intramembranous ossification especially those of the head and jaw
  ○



- Signs

- Polyuria leading to polydipsia
- Anuria
- Icterus
  - Clinical pathology
- Blood urea level is elevated which is a good index of the toxaemia that develops in uremic conditions
- Elevation of other NPN substances – uric acid, creatine, ammonia
- Elevation of amino acids in blood
- Elevation of sulphates and phosphates of potassium and chlorides

- Decreased calcium level
    - Gross pathology
- Mouth – Ulcers
- Stomach – Ulcers
- Haemorrhagic gastroenteritis (Excretion gastritis)
- Serosa contains deposits of calcium urates and urea
- Anaemia / Icterus
- Parathyroids – Enlarged
- Skeleton especially of head and jaw–The bones are every soft, pliable and can be bent (Hence the bones called rubber nose, rubber jaw)
- Pericardial fluid – Increased and contains fibrin
- Enlarged left ventricle
- Lungs – Edematous
    - Histopathology
- Left auricle – Endocardium – Necrotic
- Aorta / pulmonary artery – Intima up to initial few centimeters undergoes necrosis → Necrotic area becomes fibrosed → Calcification
- Left ventricle – myocardial fibres → Hypertrophic
- Arteriole & capillaries shows medial hypertrophy
- Liver – Degenerative changes
- Neuronal injury by toxic materials in blood
- Bone marrow – suppression of hemopoiesis
- Bones - Resorption of bone by osteoclasts
- Parathyroids - Hyperplastic
- Metastatic calcification
    - Gastric mucosa
    - arynx
    - Trachea
    - Lungs
    - Visceral pleura
- Kidney: Nephritis
- The deposits of calcium urates and urea on serous membranes and joints cause inflammation and pain.

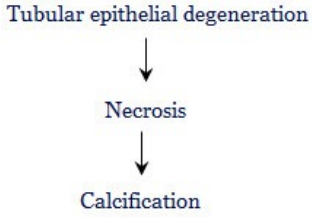
    - **PM CHANGES**
- Postmortem changes in the kidneys include greyish or blackish discolouration, distortion and softening to pulpiness.

    - **NEPHROSIS**
    - Definition: Nephrosis includes cloudy swelling, fatty degeneration and even necrosis of tubules.
    - Site of occurrence
- Proximal convoluted tubule, Henle's loop and distal convoluted tubule are affected in descending order of occurrence
    - Toxic nephrosis
- *Etiology*
    - Toxic nephrosis may be due to inorganic, organic and endogeneous substances. Here the toxins are conveyed to the kidneys by way of blood.
- *Inorganic origin*
    - Salts of mercury (Mercuric perchloride - fungicide) acts as cumulative toxicity. This is common in cattle, horses and swine. Mortality is high

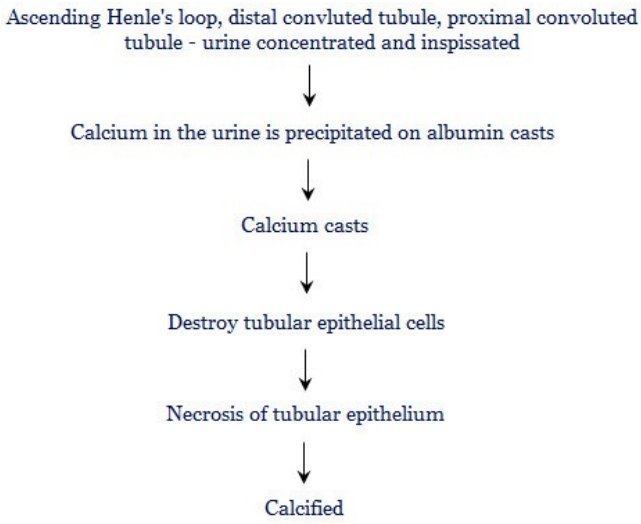
    - Potassium dichromate, copper sulphate, bismuth bisulphate, cadmium, arsenic and phosphorus
- *Gross pathology*
    - Kidney: Size: Enlarged
    - Colour: pale
    - Cut surface: Bulges
- *Histopathology*
    - Kidney – Tubule
    - Proximal tubular epithelium desquamated or show coagulative necrosis
    - Calcium is deposited in the luminal debris, necrotic epithelium and basement membrane
    - Lumen contains granular or hyaline casts
    - Moderate dilatation of the lumen of the tubules
    - Intertubular tissue contains edema fluid and lymphocytic infiltration
    - If death does not occur within a week, regeneration of the tubular epithelium occurs. The new cells are flat and dark staining. By the third week regeneration is complete
    - Organic origin
- It may be due to carbon tetrachloride, chlorinated hydrocarbons, oxalates, sulphonamides, turpentine, phenol, ethylene glycol and toxic plants.
    - Oxalate nephrosis
- *Source of poisoning*
    - Ethylene glycol (Used as antifreeze – ingested by dogs and cats)
    - Oxalate rich plants ingested by sheep
- *Signs* : Anuria develops due to obstruction of the tubule by crystals
- *Gross pathology* : Kidneys are enlarged and grayish brown in colour
- *Histopathology*
    - Kidney – Tubule
    - Proximal convoluted tubules contain doubly refractive, rectangular crystals of calcium oxalate
    - Epithelium reveals vacuolar degeneration
    - May show focal necrosis
    - Sulphonamide nephrosis
    - It is caused by continuous sulphonamide therapy in calves but with inadequate sodium bicarbonate and water.
- *Signs* : Anuria
- *Histopathology*
    - Tubules contain crystals
    - Epithelium of proximal convoluted tubule and Bowman's capsule shows hydropic change
    - Distal convoluted tubule shows swelling and proliferation of the epithelium
    - Endogenous origin
    - Seen in obstructive jaundice (Hepatorenal syndrome), ketosis, septicemia and toxemia
- *Gross pathology* : Kidneys – swollen pale / brown
- *Histopathology*
    - Kidney – Tubular epithelium
    - Shows cloudy swelling and early necrosis
    - Deposition of blood pigments in renal tubular epithelium (Noticed in jaundice)

- Anoxic Nephrosis

- *Definition*
  - When adequate supply of oxygen is lacking degeneration of the tubules may occur. This is called anoxic nephrosis.
- *Etiology*
  - Urine retention leads to increased intrarenal pressure
  - Shock – severe burns
  - Intestinal obstruction
- *Gross pathology* : Kidneys: Pale and soft
  - Lower Nephron Nephrosis (Crush syndrome, hemoglobinuric nephrosis)
- *Etiology*
  - Excessive hemolysis or haemoglobinemia - protozoa, poisons, incompatible blood transfusion
  - Severe burns
  - Equine azoturia
  - Crushing injuries- automobile accident
- *Signs*
  - Urine is acidic
  - Anuria
- *Gross pathology*
  - Kidneys – Swollen and pale or haemoglobin stained Medulla has reddish streaks
- *Histopathology*
  - Kidney
    - Asending Henle's loop and distal convoluted tubule - Tubular epithelial cells contain fine particles of haemoglobin
    - Tubular lumen – haemoglobin is precipitated or deposited as casts
  - **NEPHROCALCINOSIS**
- Here calcium is deposited as calcium phosphates or carbonates
  - Gross pathology
- Kidney shows white streaks or spots
- Three types of disturbances of calcium metabolism are met with.
  - Dystrophic calcification
  - Calcium casts
  - Deposition of calcium in interstitial tissue
  - Dystrophic calcification
- It is a type of dystrophic calcification (Primary epithelial) seen in subacute mercury poisoning
  - Pathogenesis



  - Calcium casts



- Interstitial tissue shows infiltration of histiocytes and fibrosis
- Deposition of calcium in interstitial tissue

- *Occurrence*
  - Occurs in interstitial nephritis as seen in leptospirosis of dogs aged 6 months to 4 years
- *Histopathology*
  - Here first calcium is deposited in the basement membrane of the tubules and later extends to the interstitial tissue
  - The capillary vessels may also reveal calcium masses in their walls.

NEPHRITIS

- Objective: To know about the various types of inflammations and neoplasm affecting kidneys.
- Definition : Inflammation of kidney is called nephritis
- Classification of nephritis

- Nephritis is classified as follows
  - Suppurative Nephritis
    - Pyaemic Nephritis
    - Pyelonephritis
  - Non-Suppurative Nephritis
    - Interstitial Nephritis
    - Tubular Nephritis
    - Glomerulonephritis
  - Specific Nephritis
    - Tuberculous Nephritis

SUPPURATIVE NEPHRITIS

- Suppurative nephritis is classified as follows
  - Pyaemic nephritis
  - Pyelonephritis
  - Pyaemic nephritis
- *Synonym*
  - Embolic nephritis
- *Etiology*

- Haematogenous origin– *E. coli, Staphylococci, Streptococci, Corynebacterium* and *Shigella*
- As metastatic foci – From infection of umbilical vein, mammary gland, uterus, pericardium, cardiac vegetations and lung. In the foal infection may be acquired *in utero*

- *Pathogenesis*
  - The bacteria in clumps or emboli from cardiac vegetations may get lodged in glomeruli or intertubular capillaries where abscesses are formed.
- *Gross pathology*
  - Kidney
    - Cortex may contain numerous tiny round abscesses
    - In medulla, the abscesses are elongated
    - All the abscesses are of the same size, being of same age
- *Histopathology*
  - Bacterial emboli may be seen in small blood vessels (glomeruli or intertubular capillaries)of kidney
  - Around blood vessels leucocytic infiltration may be seen
- *Sequelae* : Pyaemic nephritis might lead to death
  - Pyelonephritis
- *Definition*
  - Inflammation of pelvis and the adjacent parenchyma of kidney is called pyelonephritis.
- *Occurrence* : Met within cows but may also be found in sheep and swine.
- *Predisposing factor* : Stasis of urine
- *Etiology*
  - Bacteria like *Corynebacterium renale, Corynebacterium pyogenes*, coliforms, *Streptococci, Salmonella, Pseudomonas* and *Proteus. C. renale* has selective affinity for the pyramids of the pelvis in cows. The condition occurs more frequently in the post-parturient period because at that time infection of the uterus and vagina may occur and spread very easily through the short and broad urethra
  - Anomalies of the urinary system – *Pervious urachus* in calves and kinking of ureters in pigs
- *Pathogenesis*
  - By ascending infection from stasis of urine as it occurs in pregnant animals where gravid uterus presses upon the bladder and ureters, urethritis, cystitis, obstruction by calculi, prostatic enlargement, uterine prolapse, tumour, abscess and fibrosis in the urinary passage.
  - By Haematogenous route the infection may also occur
  - At first the infection occurs at the pelvis than it spreads to the adjacent renal parenchyma by way of large uriniferous tubules
- *Signs* : Haematuria / pyuria
- *Gross pathology*
  - Urinary bladder – Enlarged
  - Ureters – Distended; Wall is thickened and their mucosa is roughened
  - Pelvis is widely dilated with pus and contains triple phosphates
  - Calyces are widely dilated and filled with purulent material containing calcium particles. The walls are red and ulcerated
  - Papillae are either absent or dirty grey in appearance with erosions and a zone of hyperemia around

- Kidney- Beneath the capsule on the cortex is many tiny abscesses. In the medulla gray streaks may be found in the early stages
- Due to increasing accumulation of purulent material, back pressure on the kidney parenchyma leaves only a thin rim or cortex in some cases

- *Histopathology*
  - Pelvis - The epithelial lining of the pelvis may be necrosed and there may be leucocytic infiltration underneath and renal pyramid is infiltrated with neutrophils and a few lymphocytes. They may be found as a streak among the tubules in the medullary region.
  - Glomerular loops and Bowman's capsule are filled with leucocytes and bacteria
  - Tubules may contain cell casts and bacteria
  - The walls of collecting tubules and interstitial tissue may be necrosed which may be demarcated from the healthy area by a zone of leucocytes and hyperemia
- *Sequelae* : Pyelonephritis might lead to death

NON-SUPPURATIVE NEPHRITIS

- Non-suppurative nephritis is classified as follows
  - Interstitial nephritis
  - Tubular nephritis
  - Glomerulonephritis
  - **Interstitial nephritis**
- *Occurrence*
  - Most common in older male dogs
  - Sometime s observed in horses, swine, sheep and cattle
  - This is usually found at autopsy in normal looking animals
- *Etiology*
  - Haematogenous origin- Occurs in enteritis, bronchopneumonia, chronic peritonitis, salmonellosis, leptospirosis, streptococcal and staphylococcal infections
  - Ascending infections due to retention of urine – Tubular damage, metritis, pyometra, prostatitis and cystitis
  - In swine it is due to erysipelas, corynebacteria, E.coli and non-hemolytic streptoccoi, staphylococci and leptospira
- *Types*
  - Interstitial nephritis may be focal or diffuse
  - Focal interstitial nephritis
- *Occurrence*
  - Focal interstitial nephritis is usually noticed during necropsy in calves which died of some other cause
- *Etiology*
  - Incomplete resolution of diffuse interstitial nephritis
  - *E.coli* infection in calves
  - Pullorum disease In fowls
  - *Erysipelothrix rhusiopathiae* in pigs
- *Signs* : Affected animals may not show any signs
- *Gross pathology*

    - Kidneys will have white spotted appearance – small pin point sized grey-white circumscribed areas that are found scattered on the cortex under the capsule.
    - Such spots can also be noticed deep in the cortex as well as in the outer medulla on sectioning the kidney.
    - Some foci may bulge on the cortex which may be clearly seen through the capsule.
- *Histopathology*
    - Interstitial tissue contains edematous fluid and lymphocytic and plasma cell infiltration
    - Proliferation of fibrous tissue in the interstitial tissue causes tubular degeneration, atrophy and later complete disappearance of tubules. Some of the tubules are compressed with resulting dilatation of the proximal portion with cystic fluid
    - Tubular lumen may contain albuminous or granular casts
- *Sequelae* : Complete resolution with obliteration of the lesion may occur
    - Diffuse interstitial nephritis : Diffuse interstitial nephritis may be acute or chronic type
    - Acute diffuse interstitial nephritis
- *Signs* : Oliguria or polyuria
- *Clinical pathology*
    - Urine may show marked albuminuria and casts
    - Blood may show elevated BUN and creatine
- *Gross pathology*
    - Kidney is normal size or enlarged
    - Capsule strips off easily
    - Cortex is mottled (gray and red)
- *Histopathology*
    - Kidney
        - In cortex and outer medulla - The grossly appeared gray areas are diffusely infiltrated with lymphocytes, plasma cells and neutrophils and exudates.
        - Glomeruli are found to be normal
        - The tubular epithelium especially of the proximal convoluted tubule shows degenerative changes which may be so severe as to cause death by uremia
- *Sequelae*
    - Acute diffuse interstitial nephritis may pass off with resolution and healing
    - It may progress to chronic diffuse interstitial nephritis especially with repeated attacks
    - Chronic diffuse interstitial nephritis
- *Pathogenesis*
    - The damaged tubules may not be able to concentrate the glomerular filtrate
    - Connective tissue proliferation replaces the renal parenchyma
- *Signs* : Oliguria or polyuria
- *Clinical pathology*
    - Urine specific gravity – Low
    - High phosphorus level in blood
- *Gross pathology*
    - Kidney is pale and shrunken

- When stripping the capsule, the cortex is torn.
- Cortical surface is coarsely granular or uneven in appearance (Hence the name small granular contracted kidney)
- Cortex is very narrow
- Cut surface is sometimes cystic in appearance. The retention cysts vary in number and size. If many cysts are present, it may resemble a polycystic kidney

- *Histopathology*
  - Here the inflammatory process begins in the interstitial tissue and involvement of glomeruli is secondary (Glomeruli either not involved or only a few are affected)
  - Fibroblastic proliferation replaces normal renal parenchyma and the glomeruli appear as a hyaline nodule. Bowman's capsule may be surrounded by fibrous tissue. Calcium may be deposited in the glomeruli.
  - The tubules may undergo atrophy and may disappear later. Proximal convoluted tubular epithelium undergoes degeneration. Some of the tubules may become cystic proximal to the part constricted by fibrous tissue and contain granular and hyaline casts. The remaining tubules in the cortex may appear adenomatous with tall columnar epithelium due to compensatory work
  - As the degeneration of the tubules take place, leakage of urine in to the interstitial tissue causes vascular changes and fibroplasias which may become self perpetuating
  - The walls of blood vessels in the glomeruli are thicker and the lumen is narrow.
- *Sequelae*
  - In chronic diffuse interstitial nephritis, retention of phosphorus causes parathyroid hyperplasia. This excess activity of parathyroid causes increased demineralization from bones and causes metastatic calcification in the left atrial endocardium, pulmonary artery, laryngeal mucosa and pleura
  - Chronic diffuse interstitial nephritis leads to failure of renal function, uremia and finally death.
  - **Tubular nephritis**
- Tubular nephritis is dealt under nephrosis
  - **Glomerulonephritis**
  - Definition: It is the inflammation of the kidneys involving primarily the glomeruli.
  - Occurrence
- The condition is not common in animals as in man
- It is sometimes noticed in dogs, cats, swine, horses and mink
  - Etiology
- Horses used for antisera production suffer from this disorder.
- It is seen as a sequel to bacterial and viral diseases elsewhere in the body- Focal golmerulonephritis is seen in acute septicaemic infections such as acute swine erysipelas and infections by *Coliforms, Leptospira, Streptococci, Staphylococci, Pasteurella* and *Salmonella*.
- It is sometimes seen as enzootic among minks vaccinated against distemper
  - Pathogenesis
- Glomerulonephritis is due to antigen-antibody reaction to a foreign protein. The antigen –antibody complex gets deposited and damages the glomerular capillaries.
- The two mechanisms by which antigen-antibody reaction is brought about is

    - Antibodies are produced by the animal against the glomerular basement membrane. The antibody, complement and fibrin are deposited beneath the endothelium of glomerular capillaries.
    - Antibodies produced elsewhere in the body (which is not of renal origin) gets deposited beneath the epithelium of glomerular capillaries on the basement membrane.
- Here the antigen –antibody complexes affect the endothelium of glomerular capillaries, release histamine and increase their permeability.
- The primary changes are in the glomeruli.
- The tubular and interstitial lesions are secondary to the glomerular affection since their blood supply depends on the efferent arteriole emanating from the glomerulus.
    - Classification of glomerulonephritis
- Based on the course glomerulonephritis is classified as follows
    - Acute glomerulonephritis
    - Subacute glomerulonephritis
    - Chronic glomerulonephritis

1. Acute glomerulonephritis
    - Pathogenesis

- Here proliferation of the endothelial and epithelial cells takes place and blocks the lumen of glomerular capillaries and hence glomerular ischemia develops.
- There is increased permeability of the basement membrane.
- Inflammatory cells infiltrate the glomerular tuft which further aggravates the condition.
- Hence efferent arterioles become ischemic and so nutrient supply to the tubules is decreased.
- Later degenerative changes are produced in the tubules.
    - Signs : Oliguria
    - Clinical pathology
- Urine – Concentrated urine –Specific gravity is high and contain albumin, RBCs, leucocytes, renal cells and casts
- Blood – Increased BUN
    - Gross changes
- Kidneys
    - Enlarged and pale
    - Capsule easily peels and is tense
    - On the cortical surface red dots indicating congested glomeruli are seen. Sometimes small haemorrhages are also noticed
    - On section, kidney slightly bulges on the edges.
    - Histopathology
- Kidney
- Glomerulus
    - Glomerular capillaries hyperaemic initially and followed by proliferation of the endothelial and epithelial cells.
    - Fibrin thrombi may form in the glomerular capillaries and cause hemorrhages.
    - Increased cellularity of glomeruli by inflammatory cells
    - Collagen like material may be deposited between the capillaries
    - The subepithelial basement membrane becomes thickened
    - The capsular space is completely occupied by the swollen tuft, leukocytes, precipitated protein and a few erythrocytes.

- Tubules
    - Tubular lumen contains casts of protein, leucocytes and erythrocytes.
    - Tubular epithelium may contain hyaline droplets
    -
    - Electron microscopy
- Basement membrane is swollen.
- There is deposition of electron dense material between the endothelium and basement membrane as well as between epithelium and basement membrane.
    - Sequelae
- In mild cases the condition may resolve by healing
- If not, it may progress to subacute and chronic glomerulonephritis

2. Sub acute glomerulonephritis
    - Gross changes

- Kidney is enlarged and pale (Large white kidney)
- Capsule is tense and peels off easily
- Cortical surface is smooth
- Cortex is wider than normal, yellowish (so there is distinct colour contrast between it and the medulla) and may reveal few haemorrhages.
- As the cortex is yellow, there is distinct color contrast between it and the medulla
    - Histopathology
- *Glomerulus*
    - The proliferation of the endothelial and epithelial cells of the glomerular tuft is more pronounced
    - The epithelial cells of the parietal layer of Bowman's capsule proliferate and form a crescent shaped tissue of several layers thick – Epithelial crescents
    - Adhesions develop between the epithelial crescents and the glomerular tuft due to the deposition of collagen like material. Hence the tufts are obstructed and subsequently destroyed.
- *Tubule*
    - The tubular lumen contains casts of protein, necrotic epithelial cells and leucocytes
    - The tubular epithelium undergoes fatty changes which progress to hyaline droplet degeneration and necrosis.
- *Interstitial tissue*
    - Edematous with infiltrated inflammatory cells together with some amount of collagenous tissue.
    - 3. Chronic glomerulonephritis
- The subacute glomerulonephritis may imperceptibly merge with the chronic phase.
- Only a small portion of acute glomerulonephritis culminate into chronic form.
    - Signs : Polyuria and late urine decreases in volume
    - Clinical pathology
- Urine – has decreased specific gravity, contains albumin, RBCs, leucocytes and casts
- Blood –Progressive anaemia, elevated BUN values
    - Gross changes
- Kidneys are shrunken and contracted with a finely granular surface
- The capsule is adherent and when peeled some amount of cortex is removed
- On section, cortex is narrower and small retention cyst are seen due to obstruction of the tubules
- The cortical marking are obscured
    - Histopathology

- *Glomerulus*
    - Most of the glomeruli are affected and are fibrosed. Some show hyaline changes, while a few are atrophied and may disappear altogether.
    - Adhesions may develop between the tufts and capsular epithelium
- *Interstitial tissue*
    - Infiltrated with lymphocytes and more marked fibrosis is evident
    - In the scarred tissue, many tubules are disappeared
    - Arteries are thickened and narrowed due to proliferation of media and intima
    - Some of the tubules still connected to functional glomeruli are dilated.
    - Specific nephritis: Tuberculous nephritis may be seen in generalized tuberculosis as in bovines

NEOPLASMS

- Primary neoplasms of the kidney are not common
- Secondary tumours may be found more often than the pimary tumours
- Common secondary neoplasm of kidneys is lymphosarcomas

PATHOLOGY OF URINARY BLADDER

- The common pathological conditions of urinary bladder include the following
    - Anomalies
    - Rupture
    - Prolapse
    - Hernia
    - Cystitis
    - Bovine enzootic hematuria

ANOMALIES

- Persistent Urachus
- *Occurrence*
    - This is sometime seen in foals. Urachus is a tube that connects the bladder to the umbilicus in the foetus.
    - Just before birth, this is severed from the umbilical cord and becomes obliterated.
    - But in some instances, it is still patent after birth and said tot be “pervious”.
- *Sequelae* : Through the pervious urachus infection of the bladder may occur
    - Diverticula of the Bladder: Diverticula and division of the bladder into several small cavities is rare

RUPTURE

- Rupture of urinary bladder may occur in
    - Trauma by automobile accident, gun shot wounds or faulty catheterization
    - Obstruction of urethra by calculi, enlarged prostate, neoplasm or inflammatory debris

PROLAPSE

- When cows, sows, mares which have short broad urethra strain during parturition, prolapse of bladder occurs through the urethra. If not corrected, necrosis and gangrene may supervene.

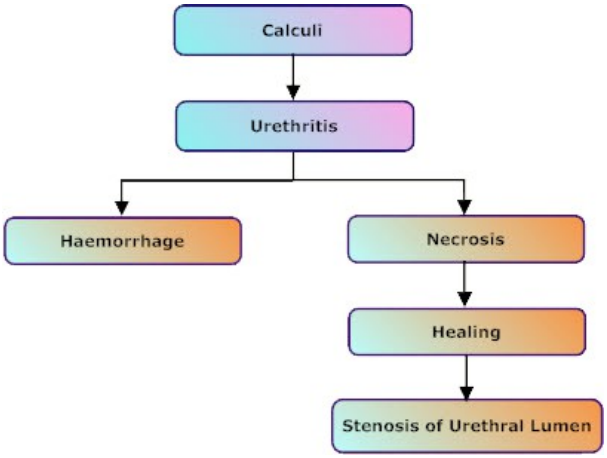
PERINEAL HERNIA

- Occurrence : Male dogs
- Etiology
- When dogs having prostate enlargement strain during urination perineal herniation of the bladder may occur.

- **CYSTITIS**
  - Occurrence: Cystitis is common in females
  - Predisposing Causes
- Paresis of bladder
- Retention of urine
  - Routes of infection
- Ascending – suppurative endometritis and vaginitis
- Descending – suppurative nephritis and pyelonephritis
- Pervious urachus in foals and calves
- Catheterization
- Expansion from nearby organs
  - Etiology
- Trauma
- Bacteria – *E.coli, Corynebacterium renale, Staphylococci, Streptococci* and *Proteus vulgaris*
  - Gross pathology
- Acute Catarrhal form
  - Urine – cloudy
  - Mucosa –Thick, edematous and congested
- Chronic catarrhal form
  - Here the mucosa is very much thickened
- Follicular form
  - In the dogs follicular form is frequently seen. Here the mucosa is studded with small grey nodules
- Chronic polypoid cystitis
  - This is seen in cattle
  - Here the mucosa is thrown into folds
- Haemorrhagic form
  - Urine – reddish
  - Mucosa – haemorrhagic
- Purulent form
  - Urine – opaque
- Fibrinous form
  - Urine contains flakes of fibrin
- Histopathology
  - Urinary bladder
    - Epithelium is degenerated and desquamated
    - Mucosa and submucosa may be infiltrated with leucocytes and may form dense sheaths around the blood vessels
    - In the purulent variety, the infiltration of leucocytes is heavy and may extend into the muscular coat. Congestion of the blood vessels is seen. In sever conditions ulceration of the mucosa may occur.
    - In the chronic catarrhal cystitis, the mucosa is very much thickened due to fibrosis. There is frequently desquamation of surface epithelium. The muscle layer may undergo hypertrophy and may be infiltrated with leucocytes.
    - In Chronic polypoid cystitis, the mucosa is thick, folded with infiltration of leucocytes and proliferation of connective tissue.
  - **BOVINE ENZOOTIC HEMATURIA**
  - Synonym : Chronic bovine hematuria

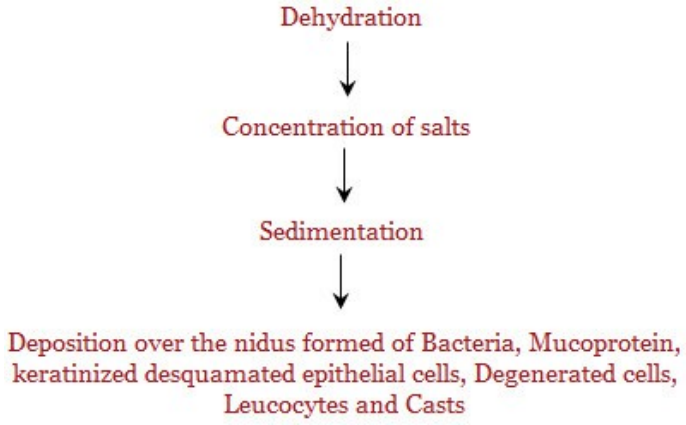
- Occurrence : Cattle – Hilly areas
-
- Etiology

- Bracken fern toxicity
- Babesiosis
- *Aspergillus kamala*
- High oxalates in feed
- Low calcium, phosphorus and manganese in diet
- Viral etiology
  - Pathogenesis : The course of the disease is chronic (a few months to years)
  - Signs
- Initially blood is present in the last few drops of urine
- Later bloody urine may be passed
  - Gross pathology
- Urinary bladder
  - Mucosa initially shows petechiae or ecchymoses
  - Mucosa is thick and reddish and may show cauliflower like tumours
- Kidney : Haemorrhagic and may show cystic dilatation in the later due to obstruction
  - Histopathology
- Urinary bladder
  - Mucosa – Initially haemorrhages may be seen. Epithelium may be hyperplastic. Metaplasia to squamous or columnar epithelium may occur frequently.
  - Capillaries – Proliferate and growths similar to haemangiomas may occur
- Kidneys may show glomerulonephritis, tubular degeneration and interstitial nephritis.
  - **NEPOPLASMS**
  - Occurrence
- Occasional
- Primary tumours include papilloma, leiomyoma, transitional cell carcinoma and squamous cell carcinoma
- The most common secondary tumour is the lymphosarcoma
  - **PATHOLOGY OF URETHRA**
  - Histological consideration : Urethra: Lining mucosa is tough to withstand infection
  - Introduction
- Affections of the urethra are rare. The most common affection is the obstruction of the lumen.
  - Obstruction



- **UROLITHIASIS**
  - Definition: Presence of calculi in urinary system
  - Terminology
- Stone in kidney is called nephrolith which may be microconcretions
  - Pelvis – pyelolith
  - Ureter - ureterolith
  - Bladder- cystolith
  - Urethra – urethrolith
  - Occurrence
- Common in the ox
- In horses, calcium carbonate is common
- In ruminants, triple phosphates are common
- In pigs, triple phosphates are common
- In dogs (Dalmatian breed) and cats oxalates and urates are common
- In cats, cystine stone is common.
- In chicken, urate deposition is common
  - Site of occurrence
- In ox, sigmoid curve is the common site. In dogs, *os penis* is the frequent site of lodgement of calculi.
  - Types
- Carbonates of – Ca, Mg, NH_4
- Phosphates of Ca, Mg, NH_4 - white, smooth, chalky and may easily break
- Silicates
- Oxalates (Mulberry calculus) – Very hard, rough, spiny and laminated. They may be solitary and large in the bladder.
- Cystine calculi are small, irregular, friable and yellow. They become greenish on exposure to air.
- Xanthine stones are brownish red, laminated and easily broken
- Urates are small, hard and brown in colour. They show concentric rings on section.
- In alkaline urine, carbonates and phosphates of calcium, magnesium and ammonium (Seen in Staphylocccal infection) stones are common.
- In acidic urine, oxalates, urates, xanthine, cystine (Seen in *E.coli* infections) stones are common.
  - Etiology
- **Nuritional deficiency:** In vitamin – A deficiency, the transitional epithelium of the urinary tract undergoes metaplasia to keratinized stratified squamous epithelium. The keratinized epithelial cells get exfoliated and may form the nidus.

- Deficiency of green fodder leads to excretion of mucoprotein in urine which acts as nidus for the formation of calculi.
- **Bacterial infection:** Here the bacteria and exudates form the nidus. Bacterial infection also alters the reaction of the medium which favours the deposition of the salts.
- **Concentration of salts** occurring in
    - Dehydration, excessive sweating
    - Poor quality of water - Hard water or high mineral content in drinking water favours high concentration of minerals in excreted urine.
    - Hypervitaminosis – D
    - Ingestion of plants with high oxalates
    - In born error in metabolism of salts
- **Sulfonamide medication** : If sulfa drugs are used with out sufficient sodium bicarbonate and water, acetyl salts are formed and precipitated as calculi.
- **Hormones:** If diethyl stilbestrol pellets are used for fattening lambs, urinary epithelium is transformed into keratinized squamous epithelium. The desquamating cells form a nidus for the calculi.
- Hypercalcemia occurring in tumours of parathyroid and in decalcification of bones in prolonged confinement is associated with urolithiasis
    -
    - Formation of calculi



    - Gross pathology
- Size of the stones depends on its location.
- Renal calculi may be microscopic in size in the tubules.
- Pelvic calculi may reach upto 8 c.m. in size.
- Cystic calculi may vary in size from a grain of sand to a tennis ball size.
- Cystolith
    - May be the largest stones
    - Calculi may be small and numerous and have a smooth surface
    - If the calculi are large, because of friction during contractions of the bladder facets may develop on the calculi.
    - Histopathology
- In chicken, urate deposition is seen in the tubules. The urates are highly irritating. Hence, the urates are surrounded by lymphocytes, macrophages and giant cells. The whole area is surrounded by fibrous tissue.
    - Sequelae
- Irritation by calculi leads to inflammation of the affected area
- Partial obstruction of ureter by calculi leads to hydronephrosis

- Complete obstruction of ureter by calculi leads to atrophy of the kidney
- Urethral obstruction by calculi leads to retention of urine which causes dilatation of bladder, uremia and catarrhal or fibrinous cystitis. The dilated bladder may rupture.

URINARY CASTS

- Definition

- Urinary casts are albuminous exudates into tubules from blood vessels. Their presence in the urine is called cylindruria .
  - Clinical significance : Casts in urine indicate some type of nephritis
  - Types
- Casts are classified according to their microscopic appearance as below
  - Epithelial casts : Contain epithelial cells of tubules. These are seen in acute tubular nephritis. They may be yellowish due to imbibition of haemoglobin.
  - Blood casts : Seen in renal haemorrhage, acute nephritis and in acute congestion of kidneys.
  - Fibrin casts are yellowish and seen in haemorrhages.
  - Hyaline casts: Hyaline cats are pale, colorless, homogenous and cylindrical. They have straight parallel sides and rounded ends. These are seen in congestion or nephritis.
  - Granular casts : Granular casts may be coarsely granular or finely granular. They may be curved or straight with rounded or broken ends. These consists of albumin, fat, epithelial cells, WBCs and RBCs.
  - Waxy casts : Waxy casts are more opaque than hyaline casts. They are seen in chronic nephritis and amyloid degeneration.
  - Pseudocasts – conglomeration of various substances on mucous threads. They have no connection with the renal disease. They may be of following types.
    - Fatty casts contain numerous fat globules. They are seen in degenerated epithelial cells.
    - Pus casts contain pus cells. They are seen renal suppuration.
    - Bacterial casts are seen in bacterial multiplication of stagnant urine.
    - Cylindroids : Consists of mucus and fat globules. They are striated and are not true casts.
  - Clinical Pathology : Casts in urine give positive reaction to protein

HYDRONEPHROSIS

- Definition

- Hydronephrosis means accumulation of urine in the renal pelvis with its dilatation due to obstruction to free flow of urine.
  - Etiology : Hydronephrosis may be congenital or acquired
  - Acquired hydronephrosis
- Partial obstruction of bladder due to luminal obstruction by calculi, external compression of renal pelvis, ureter, bladder or urethra due to an expanding tumour, scar tissue development in the ureter, compression of the ureters by surrounding inflammatory tissue, neoplasms, gravid uterus, ovarian or uterine tumours, haemorrhagic cystitis, displacement of the bladder in perineal hernia and enlargement of the prostate in the dogs.
  - Pathogenesis
- For hydronephrosis to develop, the obstruction must be partial leading to gradual stasis. Whereas complete obstruction causes atrophy of the corresponding kidney
- Obstruction of urinary passage from urethra to renal pelvis produces stasis of urine which with its back pressure causes atrophy of the renal parenchyma.

- Gross pathology

- Hydronephrosis may be unilateral or bilateral
- Extreme degree is observed only in the unilateral condition with partial obstruction proximal to the bladder. Here the whole kidney looks like a bag with paper thin capsule
- In less severe cases, the tubules are atrophied and some of which may be widely dilated. The cortex is thinner and grayish in colour.

- Sequelae

- In the accumulated fluid bacterial infect ion may occur. If the infection is by suppurative organisms pyonephrosis and pyelonephritis may occur
- Bilateral hydronephrosis leads to uremia which causes death
- If unilateral, hypertrophy of other kidney occurs

- **REPRODUCTIVE SYSTEM**
- **ANOMALIES**
- **FREEMARTIN**
- Definition

- The bovine freemartin is a genetic female born cotwin with a normal male with which it has exchanged whole blood.

- Pathogenesis

- The structural modifications of female genitalia are under the influence of androgenic hormones produced by the male foetus.

- Gross pathology

- The gonads are undifferentiated.
- The mullerian duct system is not differentiated fully.
- Ovaries are small.
- There are usually portions of tubular system.
- The uterus is small and incomplete.
- The cervix is usually absent.
- The vagina is fairly developed
- The clitoris is quite prominent.
- The vulva has frequently long tufts of hair.
- One feature of the reproductive tract, which is very useful in distinguishing this condition from severe cases of aplasia of Mullerian ducts is the presence of seminal vesicles.
- Epididymis may be present or absent.

- Histopathology

- The histologic appearance of the gonad is one of a quite undifferentiated structure. There are small tubular structures resembling primitive seminiferous tubules with lining cells similar to the sertoli cells. There are interstitial cells, which in the new born freemartins resemble fibroblasts.
- In the freemartin which is allowed to live the age of one or more years, the interstitial cells develop and resemble luteal cells in the ovary or Leydig cells of the testis.

- In the older animals, these develop into multiple large masses of orange or tan colored masses which resemble both interstitial cell tumor or corpora lutea on gross examination.
- Most freemartins do not develop ovarian follicles.
- The seminal vesicles are usually small and have abundant fibrous stroma. The epithelium resembles that of seminal vesicles of a castrated bull.
- Endometrial glands are present and produce fluid resulting in cystic distension of vestigial remnants.

ARRESTS IN THE MULLERIAN DUCT DEVELOPMENT

- These defects are of significance only in cattle and swine.
  - White heifer's disease
- This condition is seen more commonly in short horn cows due to arrest in the Mullerian duct system and consists of a number of abnormalities.
- Depending on the intensity of arrest in the development it may be classified into three groups.
  - Group A
- The animals in this group have the following characteristics
  - Hymenal constriction
  - Absence of anterior vagina, cervix, uterine body
  - Occasional submucous vaginal channels.
  - Cystic dilatation of uterine horns
  - Presence of well marked Wolffian bodies.

  - Group B
- The animals in this group have the following characteristics
  - Uterus unicornis. The abnormal horn is being present as a flat muscular band.
  - Hymenal constriction may or may not be present.
  - Group C
- The animals in this group have the following characteristics
  - Hymenal constriction
  - The rest of genitalia is comparatively developed.
  - If constriction is complete, gross utero-vaginal distension results.

INTER SEXES

  - Definition
- The intersex is an individual with congenital abnormality, where the diagnosis of the sex is confused.
  - Occurrence
- Intersexes are common in pigs but not to the same degree as in goats. In bovines, intersex seems to be a rare condition.
  - Types
- Intersexes may be of two types
  - True hermaphrodites in which gonads of both sexes are present
  - Pseudo-hermaphrodites have gonads of only one sex but has reproductive organs with some characteristics of the opposite sex. Such animals are classified as male and female pseudohermaphrodites depending on the gonads present.
  - Pseudo-hermaphroditism
- It is very common in goats and studied in detail from genetic point of view.

- The incidence of hermaphroditism in Sannen breed of goats is high and mostly present as male pseudo-hermaphroditism.
- It is caused by a recessive gene.

OTHER DEFECTS

- The more common failures of fusion occur in or adjacent to the cervix.
- Complete failure of fusion results in double vagina and double cervix.
- The anterior vagina may be partitioned by a dorsal septum in conjunction with a double cervix. A dorso-ventral band may be present across the external os (double external os). The cervix and vagina being properly fused.
- The failure of fusion may involve only a part of the cervix, chiefly the caudal part, so that there is one uterine body and bifurcated cervical canal with duplication of external os.
- If the cervix and uterine body are completely divided then the condition is known as uterus didelphys

PATHOLOGY OF OVARY

PHYSIOLOGICAL CONSIDERATIONS

- The ovary is under the control of anterior pituitary through two hormones, Follicle Stimulating hormone (FSH, and the Luteinising hormone (L H). Hence any pituitary endocrine disturbance affecting the gonadotropin levels affect the ovaries also.
- Placenta also elaborates certain hormones which have considerable effect on the ovarian function.
- Ovary produces two hormones, estrogen and progesterone, which have very important functions on the development of tubular female genital organs and control of estrus cycles.

DEVELOPMENTAL ANOMALIES

- Supernumerary and accessory ovaries are very rarely seen in cow.

Supernumerary ovary

- Supernumerary ovary is an extra gonad which is entirely separate from the normally placed gonad and appears to arise from a separate analogue.

Accessory ovary

- Accessory ovary is situated near the normally placed gonad and may be connected to it giving the impression that it developed from a normal ovary.

Anomalies in the position of the ovaries

- Anomalies in the position of the ovaries occur and vary according to the length of the broad ligament.
- It is not unusual to find cows with a short broad ligament on one side resulting in the ovary being located closer to the body wall than normal.
- In cows with uterus unicornis the gonad of the affected side is generally located near the body of the uterus. On the other hand in bitches with uterus unicornis, the ovary may be located some distance from the uterus.
- In such cases, the ovary may be overlooked during routine ovariohysterectomy.

DISTURBANCE OF GROWTH

Aplasia Or Absence Of Ovaries

- Aplasia or absence of ovaries is occasionally seen, especially in swine and sheep.

Hypoplasia of the ovary

- *Occurrence*
  - Both ovaries may be affected, or sometimes only a single ovary or a part of the ovary may be affected.
- *Etiology*

    - In the Swedish highland breed of cattle, hypoplasia is determined to have been caused by a single recessive autosomal gene.
- *Pathogenesis*
    - Hypoplasia of the ovary occurs due to the failure of migration of primordial germ cells from the yolk sac to the developing gonad during embryonic stage.
    - Thus the ovary becomes devoid of germinal epithelium, which is the precursor for the follicular system.
- *Gross pathology*
    - The ovaries are small and rudimentary in the form of a thin band with wrinkled rough and irregular surface similar to that of new born calves.
- *Histopathology*
    - Tunica albuginea is thick and covered by low cuboidal epithelium.
    - Follicles are completely absent.
    - In ovarian hypoplasia, the ovary consists of predominantly medullary tissue.
    - The stroma is dense and made up of thick fibrous tissue with several anovular cords of Type I and Type II, blood vessels and rete tubules.
    - Polyoogonia
- Polyoogonia is a condition in which each follicle, which normally contains only one ovum may contain several ova without disturbing the function of genital organs.

    - **HEMORRHAGE**

- Hemorrhage in the ovary occurs in the following conditions
    - During ovulation
    - While enucleating corpus luteum manually in the cow
    - While expressing ovarian cysts in the treatment of sterilitv and
    - In hens affected with avian leucosis complex, pullorum disease and heat stroke.

    - **CONGESTION**

- Congestion of ovary may be seen in heat storke and systemic infections.

    - **PERIOOPHORITIS**

    - Definition: Inflammation of the serosal surface of the ovary is called perioophoritis
    - Occurrence
- It is more common than oophoritis.
- It is usually chronic in nature.
    - Etiology
- Perioophoritis in bovines is seen in tuberculosis or setariasis
- In porcine, it occurs in brucellosis
    - Gross pathology
- It is usually a localized serositis which is seen as red fibrin or serosal tags
- In severe cases, the ovarian surface is shaggy and often encapsulated with adhesions of bursa.
- Serosal granulomas may occur in bovine peritoneal tuberculosis or porcine brucellosis or in setariasis in bovines. These appear as reddish nodules or tags. These infective granulomas remain strictly localized to the surface of the ovary and do not penetrate its substance.
    - Histopathology
- Affected area of the ovary contains numerous leucocytes attached to the surface of the ovary.

- The tunica albuginea is thickened by fibrous tissue with occasional lymphocytic and plasma cell infiltration.
- In tuberculosis, ovaries may be covered by tuberculous granulomatous tissue with Langhans' type of giant cells.
- In Setariasis, granulomatous foci with sections of larvae along with neutrophils and surrounded by mononuclears, epithelioid cells and fibrous capsule may be seen.
    - Sequelae
- Fine bursal adhesions may result but are too delicate to interfere with ovulation.
- Severe adhesions may interfere with the ovulation and entry of ovum into the fallopian tube.

    - **OOPHORITIS**
    - Definition : Oophoritis means inflammation of the ovary
    - Occurrence : It is rare in animals.
    - Pathogenesis : It is usually the result of an ascending infection from the oviduct or uterus.
    - Gross pathology
- Peritonitis may involve the ovaries when adhesions may occur. This is especially true in the case of bovine tuberculosis of the peritoneum and brucellosis of swine, when infective granulomas may be found on the surface of the ovaries.
- Sometimes abscesses may be found in or about the capsule.

    - **CYSTIC OVARIES**
    - Etiology ; Endocrine disturbance is the main cause
    - Occurrence
- Cysts of the ovary are more frequent in the cows, sows and mares.
- Cysts are found in high milk yielding cows more frequently
    - Gross pathology: The ovaries contain one or more cysts of varying size.
    - Types : Ovarian cysts may be either follicular or luteal type.
    - Follicular cysts
- *Definition*
    - In this condition, the graffian follicle does not rupture as it should normally and so liquor folliculi accumulates and so the cysts enlarge up to as much as 11 cm in diameter.
- *Occurrence*
    - The incidence of cystic ovary is high in high lactating animals immediately after parturition.
- *Etiology*
    - Defective feeding and faulty animal husbandry practices may be some of the contributory factors.
    - There may be an involvement of hereditary predisposition.
    - The cause of the cystic ovaries is considered to be failure of release of LH or failure of release of LH in insufficient quantities to cause ovulation.
    - It may also be due to imbalance of FSH and LH.
- *Clinical signs*
    - Clinically, nymphomania (persistent sexual desire) is observed in cows and bitches.
- *Gross pathology*
    - The following extra-ovarian lesions are observed
        - Relaxation of the sacro-sciatic ligament.
        - Vagina and vulva are edematous.

        - Cervix – enlarged with patent os
        - Uterus enlarged and edematous
        - Endometrium (cystic endometrial hyperplasia, "swiss-chesse" type): The hyperplastic glands secrete excess of mucin, producing retention cysts.
        - Enlargement of the thyroid gland
        - In the dog, mammary tumors and uterine fibroids occurs
- *Histopathology*
    - Ovary
        - The granulosa cells which are normally of several layers degenerate as atretic bodies.
        - In most of the follicular cysts a single layer of granulose cells is left appearing as a string of pearls lining the antrum. In some, even the single layer of granulose cells disappears leaving the membrana propria to line the antrum.
        - The cumulus oophorus and the ovum degenerate.
    - Pituitary gland
        - Changes have been noticed in the basophil cells of Hypophysis which are believed to elaborate the gonadotrophic hormones. The cells assume a bigger and bizarre shape, with large nuclei and large nucleoli. The basophilic granules are at first large and numerous. Later, the cytoplasm becomes clear, homogeneous and later even becoming acidophilic.
        - Side by side with these changes, the acidophils become hypertrophied and densely laden with granules
    - Adrenal gland
        - Increase in the width of the zona fasciculata occurs.
- *Sequelae*
    - The glandular cells of theca interna continue to secrete adding to the estrogenic pool.
    - Pituitary gland produces more growth hormone and prolactin.
    - Enlargement of the thyroid gland causes hyperthyroidism.
    - **Lutein cysts**
- Normally, after ovulation, corpus luteum forms from the proliferation and luteinization of the cells of theca interna and the follicular epithelial cells.
- A small central cavity is present.
- But in lutein cysts, there is abnormal accumulation of fluid in this cavity.
    - Occurrence : These are more common in cows and sows than in other animals.
    - Etiology
- The cause is probably non-release of adequate quantities of luteinising hormone by the anterior pituitary.
    - Gross pathology
- The ovary is large, round or oval and soft.
- The corpus luteum is not visible on the surface as in the case of normal one.
- The cyst has a narrow internal lining of yellowish-brown luteal tissue and contents are opalescent, light yellow and gelatinous.
    - Histopathology
- *Microscopically*
    - The cyst wall comprises of three layers surrounding the central cavity containing homogeneous contents.

- The inner layer consists of a thin band of loose connective tissue separating the adjacent luteal tissue from cystic contents.
- The middle layer has varying thickness of lutein tissue.
- The outer layer consists of concentrically arranged dense bands of connective tissue merging with ovarian stroma.
- Sequelae

- Increased production of progesterone by these cysts renders the uterus susceptible to infection. Hence pyometra is the usual sequel.

- **INRA-FOLLICULAR LUTEINISATION**
  - Definition

- Formation of luteal tissue within the follicles even before rupture is called as intrafollicular luteinisation.
  - Occurrence : This condition is noticed only on histopathology.
  - Etiology : It may be due to hormonal disturbance between FSH and LH
  - Gross pathology
- Usually the ovaries are of medium size.
- The follicle is located in the center of ovarian stroma.
  - Histopathology
- Tunica theca is thick and lined by several layers of granulosa cells.
- In the center there are irregularly arranged lutein cells with large vacuolated cytoplasm and large nucleus having scanty chromatin.

- **EMBEDDED CORPUS LUTEUM**
  - Definition

- Small encapsulated yellowish-brown lutein tissue is located in the middle of cortical stroma and associated with endometritis.
  -
  -
  - Histopathology
- The embedded corpus luteum has lutein cells of normal appearance but the fibroblastic proliferation forming irregular masses is conspicuous.
  - Sequelae : The embedded CL inhibits the oestrum resulting in anoestrous condition.

- **SMALL AND SCLEROSED OVARIES**
  - Occurrence: This is the commonest abnormality noticed in ovaries of buffaloes.
  - Etiology
- An imbalance of gonadotrophic hormones
- Subactivity or inactivity could be due to hypofunction of thyroid as evidenced by low blood levels of thyrotropic hormone in buffaloes with small ovaries.
- Nutritional error might have a significant role.
  - Gross pathology
- The ovaries are small with smooth surface.
- Neither follicle nor corpus luteum is apparent on the surface.
- The cut surface reveals dense stroma with no developing follicles.
  - Histopathology
- *Microscopically*
  - The surface epithelium is usually absent.
  - Tunica albuginea is thickened with dense fibrous tissue.
  - The cortex is reduced in thickness.

- Developing follicles are completely absent.
- A few atretic follicles may be seen.
- The stroma is dense with thick fibrous strands running in different directions.
- Aggregates of thick walled, closely packed capillaries are seen in the stroma.

EPOOPHORON

- Epoophoron consists of intercommunicating, short, closely packed, acinar structures in the loose connective tissue of mesovarian attachment at either pole of ovary.
- The lumen is narrow and lined by cuboidal to columnar epithelium with large lightly stained nucleus.
  - Etiology : These have the origin from anterior mesonephric tubules.

RETE OVARII

- It consists of tubular net work of anastomosing canals, separated by thick bands of connective tissue at the hilus.
- These tubules are lined by low cuboidal to columnar epithelium with round or elongated lightly stained nucleus.
- At times the epithelium may be hyperplastic and assume adenomatous appearance.
  - Occurrence : Rete ovarii is seen prominently in the old ovaries.
  - Etiology :The origin is from mesonephros.

ANOVULAR CORDS

- Definition
- Anovular cords originate either from groups or nests of epithelial cells which never had oocytes from normal follicles in the early stages of development replacing the follicles.
  - Occurrence: These are seen in the ovaries of bovines as scattered or in groups in the ovarian stroma.
  - Types : Three types of anovular cords are recognized.
- *Type I anovular cords*
  - These are elliptical and surrounded by a thin layer of PAS positive membrane. The cords are filled with 3 to 4 rows of irregularly arranged epithelial cells with no ovum.
  - The nucleus of the cells adjacent to basement membrane is oval with diffuse chromatin.
  - The cytoplasm is stained light and contains a net work of very thing eosinophilic fibrils.
- *Type II anovular cords*
  - These are elliptical or round and slightly larger than type I cords.
  - The cells are arranged in one or two layers of cells.
  - The lumen contains a moderate amount of PAS positive material.
- *Type III anovular cords*
  - These are larger than type II, the diameter reaching 200 microns.
  - There are two layers of epithelial cells with PAS positive amorphous substance in concentric layers in the lumen.
  - The connective tissue around the anovular follicles is arranged in several circular layers and in these epithelioid and eosinophilic cells is often found.
  - Sequelae
- Presence of anovular cords is directly proportional to the severity of hypoplasia of ovaries.
- In very severe cases Type II and Type III forms are seen.

- In less severe cases Type II and Type I cords and in early cases of hypoplasia Type I cords are predominantly seen.

FOLLICULOIDS

- These are seen in ovaries on microscopic examination and are present in one or both the ovaries of aged bovines.
  -
  - Etiology
- The common association of anovular cords with folliculoids and their close morphological resemblance suggest that the anovular cords might be precursors of the folliculoids.
- Probably under constant stimulation of gonadotrophin, particularly in aged animals, the anovulatory follicles proliferate to form folliculoids.
  - Types
- Two types of folliculoids are recognized.
  - Trabecular type
  - Colloid type
  - Trabecular Type
- These have distinct connective tissue PAS positive capsule with the invaginations of septa into the lumen dividing it into smaller cavities.
- The septa are lined on either side by single or double layers of granulosa-like cells. These cells are elongated having large vesicular nucleus and scanty cytoplasm.
- Several rossette like structures consisting of eosinophilic irregular bodies surrounded by radially arranged single or double layers of cells are present in the cavity. These structures have a resemblance to Call-Exner bodies, characteristic of Granulosa-cell tumor but the origin and morphogenesis of rossettes is different.
  - Colloid type
- A few irregular shaped PAS positive colloid bodies are characteristically seen in the lumina of solitary folliculoids.
- The cellular elements are few.
- Two types of colloid bodies are seen. One type is large, irregular in size and shape with a laminated appearance. The other type is small and spherical with homogenous structure. Both the types are surrounded by a single layer of granulosa like cells.

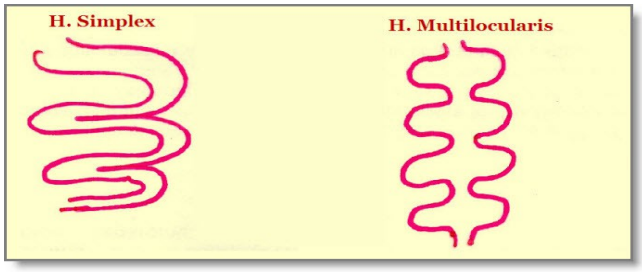
PAROVARIAN CYSTS

- These occur frequently in most species of domestic animals in the vicinity of ovary in mesosalpinx and vary in size from a few mm to 1 cm or more in a diameter.
- The cysts are lined by a single layer of cuboidal epithelium.
- The wall of the cyst contains smooth muscle.
  - Etiology : Parovarian cysts arise from the remnants of either Mullerian or Wolffian ducts.

TUMORS

- These are comparatively rare in animals.
  - Primary tumors
- Of the primary tumors, cystadenoma and cystadenocarcinoma are the most frequent and met with in the bitch and hen.
- These may be unilateral or bilateral and may be unilocular or multilocular.
- The cavities contain clear fluid and lined by either cuboidal or low columnar cells. Sometimes cilia may be noticed in these cells.
- More frequently, there may be papillary projections from the lining, filling the cavity. Usually, such papilliferous tumors are malignant.
- Peritoneal implantation of the ovarian carcinoma may be observed.

- Metastasis is by way of lymphatics.
- Tumors consisting entirely of the epithelial cells mentioned above are known as solid carcinomata and are seen in fowls and rarely in bitches. Transcoelomic spread occurs in this tumor.
- Other tumors, called endocrine tumors of the ovary that may be seen rarely are: granulosa-cell tumor, theca-cell tumor, arrhenoblastoma and dysgerminoma. Of these, the granulosa-cell tumor is more often encountered in the cow and bitch.
    - Secondary tumors
- The following secondary tumors may be found – lymphosarcomas, mammary tumor of the bitch and intestinal carcinoma of the cow.
- Teratomas and dermoid cysts may be found in the ovaries of animals and birds.
    - ***AFFECTIONS OF BURSA***
    - Bursitis
- Bursitis is generally due to the extension of inflammation from the peritoneum or from the infundibular end of the oviduct.
- The ascending infection from the oviduct is common in cases of retained placenta and septic metritis.
- Perimetritis may also contribute to bursitis.
- Excessive pressure during enucleation of corpus luteum may cause inflammation of ovarian bursa.
    - Adhesions
- Adhesions occur as a consequence of bursal inflammation or due to hemorrhage caused by ovulation or enucleation of corpus luteum.
- The adhesions may be in-between the membranes.
- The bursal adhesions cause infertility by interference with the transport of gametes.
    - **PATHOLOGY OF FALLOPIAN TUBES**
    - Occurrence : The diseases of the fallopian tubes (oviducts) are not common in animals.
    - Sequelae
- Affections of the oviduct are of importance since the ovum is transported to the uterus via these tubes and any disease of the tubes, therefore, will interfere with pregnancy and reproduction.
    - Malformations
- Absence of fallopian tubes or segmental aplasia may be met with.
- Accessory tubes, reduplication of the tubes are other malformations seen.
- These conditions are more important in the cow and sow.
    - **HYDROSALPHINX**
    - Definition: Hydrosalpinx denotes a cystic dilatation of a part of the oviduct, containing clear fluid.
    - Occurrence
- Hydrosalpinx is more important in the cow and sow.
- It may affect one or both the tubes.
    - Etiology
- This condition arises due to some obstruction in the oviduct and is usually a result of salpingitis in which, occlusion of the lumen may arise.
    - Types
- Based on gross changes and histopathology two types are recognized
    - Hydrosalpinx simplex and
    - Hydrosalpinx follicularis or multilocularis.



- Hydrosalpinx simplex

- *Gross pathology*
  - In the simple form, the fallopian tube is considerably distended, elongated and tortuous forming several coils in the mesosalpinx.
  - The distension is more often located in the ampulla.
  - The wall is thin, translucent and distended with varying amounts of clear fluid.
- *Histopathology*
  - Microscopically,
    - The mucosal folds in the simple form are considerably atrophied and lined by low cuboidal to columnar epithelium devoid of cilia.
    - The lamina propria and muscular coat are thin.
  - Hydrosalpinx follicularis or multilocularis
- *Gross pathology*
  - In the follicular form, unlike the simple form, the tube is distended with a little fluid but is hard, tortuous and irregularly beaded.
  - On cross section the lumen presents a multilocular appearance.
- *Histopathology*
  - In the follicular form, the fibrous septa are usually thin, but in some places, thickening is marked.
  - The trabeculae are lined by low cuboidal or flat epithelium.
  - Infiltration of lymphocytes, a few plasma cells and eosinophils are seen in the lamina propria.

SALPINGITIS

- Definition: Salpingitis means inflammation of the oviduct.
- Introduction

- This is the most common disease of the oviduct and is usually not diagnosed while the animal is alive.
- This is of great economic importance since salpingitis is one of the causes of sterility.
  - Etiology
- The organisms that are incriminated in causing salpingitis are
  - *Streptococcus viridans*
  - *Staphylococcus aureus*
  - *Mycobacterium tuberculosis* and
  - *Brucella suis.*
  - Epizootiology
- The organisms may enter the oviduct
  - By way of the blood stream – in generalised infection as in tuberculosis.
  - Through the *osteum abdominale* – spread of peritonitis (descending)
  - Through the *osteum uterinum* – extension of endometritis (ascending)
- Sometimes irritants may be introduced by uterine insufflations or surgical operations.
  - Gross pathology
- Macroscopically,

- There may not be visible changes in the tubes except for slight enlargement and congestion of the mucosa.
- In the milder forms, there may not be any exudate in the lumen.
- In more severe cases, catarrhal or fibrinous exudate may be present consisting of dead cells and debris.
- Histopathology

- Miscroscopically,
  - There may be mononuclear infiltration besides congestion of the mucosa, desquamation of epithelium and proliferation of stromal elements.
  - Plasma cells are particularly abundant.
  - These microscopical changes may be evident even in normal looking (grossly) tubes.
  - Sequelae
- The mucosa of the fallopian tubes does not possess much of regenerative capacity and so when once the epithelium is lost it is not restituted.
- Sterility occurs in salpingitis for the following reasons:
  - The exudate or proliferating cells may occlude the lumen of the tubes.
  - The inflammatory exudate is toxic to the spermatozoa, causing their death.
  - The ciliated epithelium and contractile muscle necessary for transport of ovum is destroyed, preventing the movement of the ova to the uterus.
  - Fibrosis in chronic salpingitis may cause occlusive stenosis.
  - **IN BIRDS**
  - Occurrence : In birds, salpingitis is a common affection.
  - Etiology
- *Primary*
  - Bacteria: *Salmonella pullorum* (decending infection from ovary) and *E. coli.*
  - Parasites: *Prosthogonimus macrorchis.*
- *Secondary*
  - Pullorum disease, vitamin A deficiency.
  - Gross pathology
- Macroscopically,
  - The body cavity contains masses of yolk material and fibrin strewn about, which may cause loops of intestines to become adherent.
  - The oviduct is swollen and distended.
  - The mucosa of the oviduct is red and edematous.
  - The cloaca and parts round about are soiled.
  - Histopathology : catarrhal, fibrinous, hemorrhagic or purulent salpingitis may be encountered.
  - Sequelae : Ascending infection may cause oophoritis or peritonitis.
  - **PYOSALPINX**
  - Definition : Pyosalpinx means pus in the salpinx
  - Etiology: It occurs in suppurative salpingitis, which is usually a sequel to suppurative metritis.
  - Pathogenesis
- Pus accumulates in some segments of the tube due to occlusion of the lumen in certain places by inspissated exudate or inflammatory thickening or by chronic granulation tissue.
  - Histopathology

- The wall of the oviduct is infiltrated by neutrophils, lymphocytes and plasma cells which are also found in the exudate that collects in the lumen.
- Metaplasia of the epithelium to squamous variety is common.
    - Sequelae: Pyosalpinx invariably ends in sterility.
    - **TUBERCULOSIS**
    - Occurrence: Tuberculosis of fallopian tubes is common in cows.
    - Types: Two varieties are seen.
- *Caseous tuberculous salpingitis*
    - Here adhesions may be present between the ovary and the tube and between the individual coils of the tube.
    - The tube is very much thickened and swollen.
    - The lumen contains the disintegrating tissue masses.
    - The mucosa is much thickened and caseated.
- *Nodular tuberculous salpingitis*
    - In this type, military tubercles are found in the mucosa as a result of generalized tuberculosis.
    - Neoplasms
- The most common neoplasm in the oviduct of fowls is the leiomyoma.
- It is also frequently met with in the right mesosalpinx.
    -
    - **PATHOLOGY OF UTERUS**
    - **MALFORMATIONS**
- The following malformations may sometimes be seen:
- Aplasia, hypoplasia, duplication of cornu and longitudinal division of the uterus by a septum may be encountered.
    - Etiology: the failure of development of Mullerian duct system.
    - Gross pathology
- The conditions are bilateral in the case of infantile genitalia and freemartins and unilateral in the case of uterinum unicornis.
- Sometimes cystic dilatations are noticed in the undeveloped segments.
    - **RUPTURE**
    - Etiology
- Rupture of the uterus may occur during parturition due to violent contractions or due to obstetrical manipulation during dystocia.
- Rupture may occur in prolonged dystocia and torsion due to weakening of the wall.
- Another rare cause may be over distension of the uterus with introduced fluids.
    - Sequelae
- Rupture may involve only the mucosa in which case healing will occur.
- If the whole wall of the uterus is involved, death may supervene, due to hemorrhage (or) inflammation of the uterus spreading to the peritoneum (or) entry of the placenta into the abdominal cavity.
    - **MALPOSITIONS**
    - Torsion of the uterus
    - Torsion of the uterus is most common in the cow, especially during the terminal stages of pregnancy.
- Introduction: The condition assumes importance only if the twist is 180° or more.
- Gross pathology
    - The veins of the broad ligament and ovarian ligaments are compressed while acute hyperemia occurs in the arteries.

- Sequelae
    - As the cervix is tightly closed in the twist, parturition cannot take place unless the disorder is corrected.
    - Minor twists are self corrected.
    - The uterus is liable to rupture easily in this condition as the walls become weakened and friable.
    - If the condition is not corrected the dam will die of gangrene, sepsis and peritonitis.
    - Hernia
- Etiology
    - Displacement of the uterus, especially in advanced pregnancy, through a ruptured diaphragm into thoracic cavity may occur in dogs and cats (possessing sufficiently long broad ligaments) as a result of automobile accidents.
    - Displacement of the uterus in abdominal and ventral hernias is also met with. In the bitch perineal hernia is also seen.
    - Herniation into inguinal and femoral canals may occur, when it is called a metrocele if a peritoneal lining of the sac is present.
    - Prolapse of the uterus
- Occurrence
    - Prolapse of the uterus through the vulva is most common in the cows, but may also be seen in other animals.
- Etiology
    - It may be due to strong uterine contractions for expelling the fetus, the placenta or the exudate.
- Predisposing causes
    - Forced traction during dystocia, post-parturient hypocalcemia and retained placenta are the predisposing causes.
- Gross lesions
    - The prolapsed uterus may be enlarged, edematous, congested and haemorrhagic
- Sequelae
    - The sequelae are similar to those found in intussusceptions viz. acute congestion, hemorrhage, necrosis, infection, gangrene and death.
    - Sows and poultry may injure everted uterus.

CIRCULATORY DISTURBANCES

- Hyperemia and edema of the endometrium
- Physiological hyperemia and edema of the endometrium are found during estrus.
- Acute hyperemia is present in metritis.
- Chronic general venous congestion is found with cardiac and pulmonary lesions hindering normal blood flow through these organs.
    - Hemorrhage
- *Physiological*
    - Haemorrhage occurs during estrus, analogous to the menstrual discharge.
    - This is more often found in heifers and bitches.
    - The source of the blood is capillaries of the endometrium.
    - Ecchymoses on the serosa and musculature are normal in heifers during estrus.
- *Pathological*

- Hemorrhage can occur during parturition and dystocia. One of the common causes is manual intervention in dystocia when rupture of the uterus with hemorrhage may take place.
- Hemorrhage from the arteries of the broad ligament, especially in the sow, is a surgical hazard during cesarean operation, if too much traction is applied on the uterus.
- Torsion of the uterus as well as prolapse may be responsible for hemorrhages.
- Lastly, massive hemorrhages may be met with in cattle in sweet clover poisoning.
- In dogs metrorrhagia may be the result of hormonal disturbances. There may be severe lowering of FSH.
- Thrombosis of the uterine vein

- Thrombosis of the uterine vein may be observed in septic metritis, torsion or prolapse.
- The affected veins are dilated and tortuous and do not collapse at death.

- **GROWTH DISTURBANCES**

- Atrophy of uterus

- *Etiology*
  - Atrophy of the uterus may occur in the following conditions
    - Senility
    - Oophorectomy after a normal full growth of uterus is attained.
    - Hypopituitarism due to
      - wasting disease
      - primary lesion of the pituitary.
  -
  - Hypoplasia of uterus
- *Etiology*
  - Hypoplasia may be observed in
    - Nutritional deficiency
    - oophorectomised young animals
- *Gross pathology*: The uteri of affected animals may not attain full size.
  - Hyperplasia of uterus
- *Synonym*: It is also known as cystic hyperplasia of the endometrium.
- *Occurrence*: Hyperplasia of the endometrium is observed in all species of animals but is more often met with in the dog.
- *Etiology*
  - Hyperplasia of endometrium occurs due to increased estrogen or progesterone secretion.
  - This condition is also noticed in animals having
    - granulosa-cell tumor
    - papillary cystadenoma
    - persistent corpora lutea
    - ovarian follicular cysts.
  - Feeding on legume pastures: Lucerne and clover contain substances having estrogen activity cause hyperplasia of the endometrium in the ewe and cow.
- *Gross pathology*
  - Macroscopically
    - Endometrium of both the horns contains cysts of different sizes of microscopic to as big as 5 mm in diameter.
    - The cysts may completely fill the lumen giving it a “swiss-cheese” appearance. They contain clear fluid.

        - The lumen of the horns may contain mucus which may flow out of the vulva.
        - In those animals in which infection has taken place, there will be pus in cystic glands as well as in the lumen and may flow out of the vulva. The uterine horns show alternating constrictions and dilatations which appearance mimics pregnant uterus.
- *Histopathology*
    - Microscopically,
        - The endometrium may show thickening due to polypoid proliferation.
        - Hyperplasia may involve the surface epithelium or glandular apithelium.
        - No inflammatory changes may be present in the uncomplicated cases but only cyst formation of the glands which are increased in number and irregularly distributed unlike the normal orderly arrangement may be seen.
        - The cysts contain a single layer of epithelium enclosing clear watery fluid.
        - In some animals, there may be plasma cell infiltration of the lamina propria.
        - In those animals in which infection has taken place, there will be neutrophilic infiltration of the lamina propria.
- *Clinical features*
    - Abnormal uterine bleeding.
    - Disturbances in estrus cycle
        - irregularity
        - longer or short duration
        - diminished or enhanced characteristics of the different phases.
    -
    - Sterility
    - Abortion
    - Prolonged lactation
    - Development of secondary infection, characterized by high leucocytic count with a shift to the left.

INFLAMMATION

- If the inflammation is restricted to the endometrium of the uterus alone then the condition is known as endometritis.
- On the other hand, if the whole thickness of the wall is involved metritis is the term used. Metritis is inflammation of the uterus and is found in all animals.
- Inflammation of the serosa is known as perimetritis .

ENDOMETRITIS

- Etiology: Endometritis may be caused by
- Infection
- *Trichomonas fetus, Vibrio fetus, Brucella* , weaker strains of pyogenic cocci and coliforms.
- A severe metritis might have subsided and leave a low grade inflammation of the endometrium.

    - Irritants: Introduction of too hot fluids or too irritating chemicals into the uterus may injure the delicate mucosa.
    - Routes of infection
- Ascending infection from the vagina. This is the most important route. Infection may occur during coitus or during artificial insemination or during manual handling of the uterus for therapeutic purposes.
- Descending infection from the abdominal cavity through the fallopian tubes.
- Through lymphatics from the peritoneum.
- By way of blood: this is important in tuberculosis.
    - Gross pathology
- Macroscopically,
    - No gross lesions are evident.
    - An increased secretion of tenacious mucus may be visible.
    - The mucosa may be swollen, red or haemorrhagic and rough instead of having a smooth surface and covered with fragments of necrotic material.
    - Histopathology
- Microscopically,
    - In the mild catarrhal variety there may be slight but diffuse infiltration by lymphocytes, plasma cells and macrophages.
    - The blood vessels are engorged.
    - In acute suppurative endometritis, the mucosa is infiltrated with neutrophils and the surface epithelium shows degenerative and necrotic changes.
    - In chronic endometritis, the endometrial mucosa is fibrosed, infiltrated with mononuclear cells and the glands have become shrunken.
    - In cystic endometritis, the endometrium has many cysts and more fibrous tissue.
    - More severe forms of inflammation invariably involve all the layers of the wall and must be considered as metritis.
    - Clinical considerations and sequelae
- Though mild in appearance, endometritis must be attended to promptly or else conception may not occur.
- The inflammatory exudate being toxic is lethal to the ovum whether fertilized or not.
- The condition may progress to the chronic stage when permanent sterility may supervene.
    -

M

- Depending upon the virulence and nature of the organisms, metritis may be acute catarrhal, acute suppurative and chronic suppurative.
    - Predisposing causes
- Infection is facilitated by the following which may cause an initial injury:
    - Parturition, dystocia, mechanical injuries by the obstetrical instruments, projecting fetal bones after embryotomy, excessively warm irrigating fluids, chemical antiseptics and disinfectants introduced into the uterus.
    - The parturient uterus, with its lochia is a good medium for the growth of bacteria.
    - Retained placenta and albuminous exudate are also ideal for the propagation of micro organisms and so infection is common in those animals in which

lochia is plentiful and involution is delayed. The latter condition may result due to weakened and injured uterine musculature in prolonged dystocia.
    - Nutritional deficiencies and hormonal disturbances are also considered as predisposing factors by preparing the soil for infection.
    - Etiology
- Metritis is caused by bacteria that invade the injured uterus viz. *Corynebacterium pyogenes, Streptococci, Staphylococci, E. coli, Spherophorus, Clostridium sp.*
- Mycobacterium tuberculosis and other chronic granulomatous infections may also invade the uterus.
    - **ACUTE CATARRHAL METRITIS**
- This is acute endometritis and is difficult to be differentiated from the uterus during estrous cycle.
    - **CHRONIC NON-PURULENT ENDOMETRITIS**
    - Gross pathology
- The uterus becomes thickened. This thickening is not uniform because of the fixed position of the outlet ducts of the gland and so assumes a polypoid appearance. This is known as chronic polypoid endometritis.
    - Histopathology
- Because of the proliferation and the subsequent contraction of the superficial connective tissue, the mouth of the glands may be closed rendering them cystic. This condition is known as chronic cystic endometritis.
    - Sequelae
- If the causes of the endometritis are removed, the leucocytes are replaced by fibrous tissue which on contraction produces atrophy of the glands and the mucosa and so chronic atrophic endometritis results.
- In cows the mucosa in chronic endometritis may undergo degeneration and subsequently be calcified and the condition then is known as endometritis calcificans.
    - **ACUTE SUPPURATIVE METRITIS**
- This condition usually arises from infection by pyogenic organisms and is a frequent complication of dystocia, retained placenta or abortion.
    - Gross pathology
- The mucosa of the uterus is covered by a purulent, often reddish exudate. This may frequently contain shreds of disintegrated fetal membranes.
- It is very much reddened, thickened and rough.
- The uterine wall is thickened and friable.
    - Histopathology
    - There is infiltration of the endometrium by large number of neutrophils. After several days, macrophages, lymphocytes and plasma cells infiltrate the endometrial stroma.
    - Infection extending into the uterine veins results in thrombosis of those vessels.
        - The muscle fibres atrophy and disappear or show Zenker's degeneration.
    - Subserosa is edematous and infiltrated with leucocytes.
    - Serous coat also shows inflammatory changes.
    -

-
- **CHRONIC SUPPURATIVE METRITIS PYOMETRA**
- Introduction

- Pyometra literally means pus in the uterus. But usually this term is applied to chronic suppurative metritis.
  - Occurrence
- This condition is seen in dogs, cats, cows and swine.
- In cattle, pyometra is encountered as a result of retention of placenta. Since it is a very good medium for bacteria to thrive, the placenta putrifies.
  - Predisposing causes: Incomplete involution in cows.
  - Etiology
- In dogs, increased progesterone activity is the prime cause. The bacteria causing pyometra in bitches are *Corynebacterium pyogenes, E coli, Proteus sp*. and Staphylococci.
- In the cow, *Trichomonas fetus* infection is a common cause.
  - Pathogenesis
- Persistent corpus luteum releases large amount of progesterone in bitches which causes "Pseudopregnancy" where in hyperplastic endometrium occurs. This hyperplastic endometrium is infected to produce pyometra.
  - Gross pathology
- *Cow*
  - Thin cream-like pus may be discharged through the vulva, soiling the tail and the perineum.
  - When the animal lies down, due to pressure on the abdomen, pus which may stagnate due to gravity during a standing position may flow out.
  - The uterus is dilated and involution may not be complete.
- *Bitch*
  - The abdomen is enlarged as in a full pregnancy.
  - The exudate is always retained in the uterus as the cervix is completely closed.
  - The horns are dilated and thin-walled and contain chocolate-colored fluid.
  - The serosal surface may show congested vessels and evidence of inflammation.
  - The mucosa may be thickened irregularly and in some places it may be ulcerated and hemorrhagic and covered with necrotic shreds of membrane appearing as though bran is sprinkled. Retention cysts may sometimes be seen.
  - Histopathology
- *Cow*
  - The appearances are similar to those of endometritis viz. congestion of the blood vessels and infiltration by inflammatory cells especially neutrophils, lymphocytes and plasma cells.
  - These cells accumulate under the epithelium, leading to its purulent softening and separation of the necrotic area of tissue and these appear as bran-like material grossly.
- *Bitch*
  - The condition is more acute and so greater infiltration of neutrophils and lymphocytes occurs.
  - There is hyperplasia of the endometrium, producing pseudostratification or papillary proliferation.

- Sometime squamous metaplasia may be observed.
-
-
- Extragenital lesions

- These are found in the dog and cat and may be due to the 'toxic' effect on other organs as well as to periodical bacteremia that may occur.
- The lesions are
  - Intense leucocytosis: The total white cell counts vary from 30,000 to 160000 per c.m.m. An extreme shift to the left and toxic granulation of neutrophils are found.
  - Anemia due to depression of the bone marrow.
  - Extramedullary leucopoiesis, especially in liver, spleen, kidneys, adrenals, lungs and lymph nodes.
  - Congestion and degenerative changes in the liver.
  - Lesions of the kidney – glomerulonephritis, tubular degeneration, hemorrhages in the medulla, infarcts, pyelonephritis.
  - Adrenals – necrosis of the cortex and hemorrhages in the medulla.
  - "Sinus Catarrh" of the lymph nodes.
  - **SCLEROTIC METRITIS**
  - Sclerotic metritis
- *Definition*
  - Sclerotic metritis is characterized by complete destruction of endometrium as a result of severe chronic endometritis.
  - A thick dense connective tissue layer replaces the endometrium.
- *Pathogenesis*
  - The focus of infection in the connective tissue layers is responsible for purulent exudate in the uterine cavity.
- *Clinical signs*
  - Cow is usually anestrus
  - On rectal examination, the cervix thickened, the uterus appears hard and firm and the corpus luteum is found deeply embedded.
- *Sequelae*
  - The uterine caruncles and endometrium are destroyed resulting in permanent sterility.
  - **PERIMETRITIS AND PARAMETRITIS**
  - Definition
- Perimetritis and Parametritis are characterized by varying amounts of adhesions between uterus and broad ligaments, with other pelvic and abdominal organs.
  - Etiology
- The adhesions may occur due to
  - severe metritis
  - douching with strong irritant solutions
  - perforation of rectum with leakage of its contents
  - torsion of uterus
  - vaginal and cervical lacerations during difficult birth
  - excessive bleeding following enucleation of corpus luteum or vigorous massage of infected uterus.

- The condition may also be due to peritonitis or tuberculosis of the genital organs.
    - Gross pathology
- In infection of the uterus by the bacillus of Black Quarter produces gas gangrene and abortion.
- The muscles and peritoneal coat are involved with inflammatory changes and gas formation. This condition is known as Physometra .
    - Sequelae
- Necrobacillary metritis that may occur in cows due to puerperal infection by *Spherophorus necrophorus* is always fatal.
    - **TUBERCULOSIS**
    - Routes of infection
- Infection may be descending from tubercular peritonitis or it may be hematogenous as occurs in generalized tuberculosis.
    - Types
- Two types of tuberculosis of uterus are seen.
    - *Disseminated military tuberculosis* in which the tubercles are found uniformly in the mucosa.
    - *Diffuse caseating endometritis* in which the body or the cornua are diffusely thickened. The lumen contains large quantity of serous or purulent exudate containing large caseating masses. The mucosa is very much thickened and caseated.
    - **ACTINOMYCOSIS**
    - Occurrence: Incidence of actinomycosis is not common.
    - Gross pathology: Large swellings with extensive pelvic adhesions are produced.
    - Sequelae: Prognosis is generally poor
    - **BRUCELLOSIS OF UTERUS OF PIG**
    - Etiology: Brucellosis of the uterus in pigs caused by *Brucella suis*.
    - Gross pathology
- Some aged pigs reveal pea sized military yellowish-white nodules in the uterine mucosa
- These nodules may be single or may occur in groups.
- The mucosa which may be raised may sometimes be ulcerated. In the centre, the nodules contain a little pus.
- Fetal membrane may show petechiae and whitish flaky material
    - Histopathology: Histologically the nodules are granulomas.
    - Diagnosis: Animals having these lesions give positive test for brucellosis.
    - **MUCOMETRA / HYDROMETRA**
    - Introduction
- The two conditions are considered together as the difference is probably only in physical properties and depends on the degree of hydration of mucin, which in turn may be related to the relative activity of estrogenic hormone.
    - Gross pathology
- The accumulation of thin or viscid fluid in the uterus is concurrent with the development of endometrial hyperplasia or is proximal to an obstruction of the lumen of the uterus, cervix or vagina.
- In the first instance the amount of the fluid may be several litres and the greater the volume of the fluid the less viscous it is. Small amounts of mucin give the mucosal surface a gummy stickiness.

- In the second instance, that of obstruction to the lumen, the volume of fluid depends on the site of obstruction. The fluid is slightly cloudy and watery.
- In cows with cystic ovaries, the large volume of fluid is usually associated with functional cysts of the follicles.
    - Sequelae
- An abnormally long and tortuous cervix may result in a form of mucometra caused by the retention of uterine secretion.
- Animals with mucometra are sterile.
- If affected uterus becomes infected, an intractable pyometra results.
    - Histopathology
- Microscopically,
    - The endometrium is thin and lined by a single layer of cuboidal to low columnar cells.
    - The uterine glands are reduced in number.
    - The endometrial stroma is edematous.
    - **UTERINE ABSCESS**
    - Etiology
- Uterine abscesses are usually man made lesions produced either by an insemination pipette or some instrument used for uterine medication.
    - Gross pathology
- These are commonly present on the dorsal wall of the uterus at the anterior extremity of the body and are of variable sizes containing thick yellow pus.
    - **ENDOMETERIOSIS**
    - Definition
- Endometriosis means presence of endometrial glands and stroma in places other than endometrium.
    - Occurrence: Endometriosis is occasionally noticed in bitches and bovines.
    - Types
- If these are seen between the muscle bundles of myometrium, the condition is called endometriosis interna or adenomyosis.
- If these are seen in places other than uterus such as mesosalpinx, ovary, cervix or intestinal serosa, it is called endometriosis externa.
    - Histopathology
- The endometrial glands show changes in response to ovarian activity and often there are hemorrhages.
    - **MUMMIFICATION OF FOETUS**
    - Occurrence
- Mummification of a dead fetus is seen occasionally in any, but usually in multiparous species and most commonly in the cow.
- In the mare it is typically one of twin fetuses which is mummified.
    - Types
- In bovines, haemic mummification is seen
- In mare and sows, it is of paperaceous type.
    - Pathogenesis
- A prerequisite for mummification is the absence of infection unlike in maceration.
- The fluids are reabsorbed and the membranes become closely applied to the desiccated fetus.
- The time required for complete mummification depends on the size of the fetus but probably requires as long as 6 to 8 months.
    - Gross pathology

- The whole fetus becomes brown or black and rather leathery, moist on the surface with sticky mucus without odour.
    - Sequelae
- In uniparous animals, the mummified fetus is usually retained indefinitely or if aborted may only be delivered into vagina.
- In the case of multiparous animals, it may be delivered along with viable fetuses.
- Animals which had and recovered from mummified fetus usually breed normally on subsequent occasion.

    - **MACERATION OF FOETUS**
        - Definition
- If the foetus *in utero* is infected it may be softened by the toxic products liberated by the infecting organisms, then it is called as maceration of foetus.
    - Pathogenesis : Maceration of foetus depends on the presence of infection in the uterus.
    - Sequelae
- If the early embryo succumbs to uterine or embryonic infection maceration is usually followed by resorption from the uterus or expulsion along with a small amount of purulent discharge.
- If the fetus is about three months, complete foetal maceration does not occur and bones resist maceration. These may be discharged or be retained in the pus of pyometra indefinitely, often near the cervix.
    - Histopathology
- Advanced uterine lesions accompany the macerated foetus.
- The uterine wall is thickened and the reaction within it varies from the acute exudative inflammation of pyometra to more or less complete sclerosis and replacement by granulation tissue in long standing cases.

    - **NEOPLASMS**
- In the domestic animals neoplasms are not common.
- The most common uterine tumor is the lymphosarcoma, a local manifestation of a generalized condition. In the uterus the neoplastic cells may aggregate as nodules or may diffusely infiltrate the organ.
- Adenocarcinomata of the uterus are encountered with metastases in the lungs and liver.
- In the bitches, uterine fibroids may be seen.
- In chicken, Marek's disease nodules may be seen in the oviduct
    -
    - **PATHOLOGY OF CERVIX**

        - **MALFORMATIONS**
- Malformations occur more frequently in the cervix than in other parts of the reproductive tract.
    - Duplication of cervix
- Varying degrees of persistence of the median wall of the Mullerian ducts which are destined to develop into cervix result in the formation of a complete or partial duplication of cervix.
- Incomplete double cervix occurs much more frequently than a complete duplication and usually involves the portion of the cervix adjacent to vagina.
- *Sequelae*
    - In the case of both incomplete and complete double cervix, if the insemination is done pregnancy may occur but it may result in dystocia.
    - Absence of external os

- Absence of external os may be commonly encountered.
- In this case, the expulsion of uterine secretions cannot occur resulting in accumulation of fluids in uterine horns leading to hydrometra.
    - Double external os
- *Definition*
    - This is the presence of a dorso-ventral band adjacent to external cervical os giving an impression as though two cervical openings are there.
- *Etiology*
    - This condition is inherited and conditioned by a single recessive gene with low penetrance.
- *Sequelae*
    - It may not interfere with conception or pregnancy but may cause dystocia occasionally.
    - The fetal membranes may be caught on this dorso-ventral band.
    - Hypoplasia of cervix
- *Definition*
    - The cervix may be very small and there may be deficiency in number of cervical rings.
- *Sequelae*
    - Hypoplastic cervix is usually defective in protecting the uterus against bacterial invasion from vagina.
    - Tortuosity of cervical canal: Here the cervix may be kinked or tortuous.
- *Sequelae*
    - In the S-shaped kink of cervix, insemination pipette cannot be inserted into cervix.
    - Extreme degrees of tortuosity of the cervical canal may cause of infertility in heifers.
    - Cervical dilatation and diverticula
- *Definition*
    - Dilatation and diverticula usually occur in heifers at the level of third and fourth cervical rings.
- *Sequelae*
    - The cervical canal is usually very small anterior to the defect so it is difficult to insert insemination pipette.
    - With age tenacious mucus tends to accumulate in the area of the defects.
    - Prolapse of cervical rings
- *Definition*: Here the first and sometimes the second cervical rings protrude into the vagina.
- *Occurrence*: It is a condition which usually develops with age
- *Etiology*
    - Repeated parturition.
    - Lacerations and hemorrhages which occur during parturition
- *Sequelae*
    - It results in the formation of excess fibrous stromal tissue, enlargement of cervical rings, vascular defects and occasionally squamous metaplasia of the affected rings.
    - **CERVICITIS**
    - Definition: It is the inflammation of the cervix
    - Etiology

- The causes include
    - Mechanical injuries
        - during parturition
        - copulation and
        - phooka – criminal stimulation of the vagina or os for higher milk yield.
    - Diseases of uterus and vagina: Cervicitis, always occurs whenever metritis or vaginitis is present, since cervix is located between these two.
    - The cervicitis normally follows abnormal parturition such as abortion, premature birth, dystocia, retained placenta, post partum metritis, pneumovagina and vaginitis.
    - The organisms responsible for cervicitis are the same as those of metritis.
    - Cysts in Cervix
- Retention cysts of the cervix are seen in cows.
- These are usually small. Bigger ones may partially occlude the cervical canal.
    - Neoplasms of Cervix: Squamous cell carcinoma may be encountered in cervix.
    -
    - **PATHOLOGY OF VAGINA**
        - **DEVELOPMENTAL ABNORMALITIES**
    - Double vagina: It is due to persistent median septum along the vaginal passage.
    - Median vertical bands
- Median vertical bands connecting the floor with the roof of vagina at the hymenal border is more common.
    - Heterotopic vulval opening
- Heterotopic vulval opening may be located in the inguinal region behind the udder instead of below the anus in the perineum.
        - **CYSTS IN VAGINA**
    - Definition
- Dilatation of the Gartner's canals (which are remnants of Wolffian ducts) produce cysts in vagina.
    - Etiology
- Cysts may be noticed in the cow poisoned with highly chlorinated naphthalenes and in those having ovarian follicular cysts.
    - Gross pathology
- Multiple cysts are located on the floor of the vagina as parallel rows.
- These contain a thin clear fluid and are lined by simple cylindrical epithelium.
        - **RUPTURE**
    - Etiology: Rupture of vagina may occur during parturition or during coitus (especially in sows).
    - Sequelae: Infection of the rupture may lead to abscess, phlegmon, gangrene and peritonitis.
        - **VAGINITIS AND VULVITIS**
    - Etiology
- Causes include physical trauma, chemical, nutritional deficiencies, bacterial and viral agents that are the same as for metritis and abortion.
    - Gross pathology: Appearances are similar to those of inflammations of other mucous membranes.
    - Granula Vaginitis

- *Synonym*: It is otherwise called nodular venereal disease.
- *Etiology*
    - The causative agent is not clearly established, many organisms have been listed: hemophilus, pleomorphic rods and viruses.
- *Occurrence*: The incidence of the disease is highest in naturally served herds.
- *Gross pathology*
    - The lesions are most commonly seen in the region of the clitoris, below the lips of the vulva. Sometimes lesions extend up to the dorsal commissure.
    - Raised orange-red areas about 3 mm in diameter are noticed in the posterior part of the vagina up to the urinary meatus.
    - The raised areas or granules are isolated and in severe cases may coalesce.
- *Histopathology*: The granules are lymphoid follicles or lymphoid accumulations.
- *Sequelae*
    - The epithelium over the granules is easily injured and bleeding may occur.
    - This disease is described as one of the causes of infertility.

VESICULAR VENERAL DISEASE

- Synonym

- Vesicular vaginitis
- Coital exanthema
    - Etiology
- The cause is supposed to be a virus, which is considered to be the same causing infectious bovine rhinotracheitis and infectious keratoconjunctivitis in cattle, an epitheliotropic virus.
    - Transmission: This is a highly contagious disease, frequently transmitted by coitus.
    - Epizootiology
- Incubation period is 1 to 3 days.
- The course of the disease is about 10 days.
    - Clinical signs
- Early fever may be seen during viremic phase.
- Within 24 hours of infection, there is mucopurulent discharge from the vagina.
    - Clinical pathology: Leucopenia may be seen during viremic phase.
    - Gross pathology
- The vaginovulval mucosa is covered with thick mucus.
- Starting with hyperemia, hemorrhages may be observed later in the submucosal lymphoid follicles.
- Pustules may be found in the vagina and vulva over the lymphoid follicles.
    - Histopathology
- The epithelial cells undergo hydropic degeneration
- Acidophilic intranuclear inclusion bodies are found in the epithelial cells, since the virus is epitheliotropic.
- Neutrophilic infiltration is present near these lesions.
- In the lamina propria, infiltration by lymphocytes and plasma cells may be found together with edema and hyperemia.
    - Sequelae
- Recovery is the rule with transient immunity. Resolution occurs in about 8 days.
- Rupture of these lesions results in ulceration.
- Extension of infection to cervix and uterus results in cervicitis and metritis.

SPECIFIC BOVINE VENEREAL EPIDIDYMITIS AND VAGINITIS

- Introduction: It is chronic viral disease of cattle, transmitted by coitus and is found in Africa.
- Clinical signs

- The disease is characterized by mucopurulent vaginal discharges in females.
- In the bulls the disease causes swelling of epididymis.
  - Sequelae: Permanent adhesions of fallopian tubes may occur

- **PNEUMOVAGINA**

  - Occurrence: It is common in mares
  - Etiology

- It is due to the deformities or injuries to the vulva and its suspensary apparatus.
- It is also due to the vice of crib biting.
  - Pathogenesis

- In crib biting, the mare makes an inspiratory effort holding something hard in its mouth.
- Because of this effort, and because of the already existing negative pressure of the uterus, and due to the inability of the vulva to keep back the external air from entering the vagina, air enters the vagina and causes ballooning of the vaginal wall.
- Along with air, urine and dung also gain entrance into the vaginal cavity.
  - Sequelae

- The contamination of vagina leads to vaginitis, cervicitis and endometritis.
- *Klebsiella* can generally be isolated from the exudates of the vagina.

- **TUMORS**

- Fibromas may be found, which may be soft or hard, pedunculated or sessile.
- Leiomyomas are also seen. Some have fair amount of collagen fibres when they are known as fibromyomas. These are comparatively harder than the pure leiomyomas.
- Transmissible venereal tumor of the bitch is frequently seen in the vagina.

- **ABORTION**

  - Definition: Expulsion of a dead fetus prior to the normal full gestation period is called abortion.
  -
  - Pathogenesis

- Abortion is mostly due to infection of the fetus, placenta or the uterus since these conditions cause death of the fetus.
- A dead fetus is a foreign body and so is expelled from the uterus.
- The following are the causes
  - Abortion
  - Vibriosis
  - Trichomoniasis
  - Listeriosis
  - Epizootic bovine abortion
  - Leptospirosis
  - Abortion in mares by *Salmonella abortus equi*
  - Equine viral abortion
  - Equine rhinopneumonitis virus (Equine influenza)
  - Equine viral arteritis
  - Abortion caused by the virus of Infectious Bovine Rhinotracheitis
  - Mycotic abortion in cattle and sheep

- **BRUCELLOSIS**

  - Etiology

- *Brucella abortus, B. melitensis* and *B. suis* affect the cow, sheep goat and pig.
- *Brucella ovis* affects only the sheep.
    - Bovine brucellosis: The typical abortion occurs at about the 7th month of gestation in the cow.
- *Routes of infection*
    - Alimentary canal – ingestion of feed or water contaminated by fetal membranes, fetus or uterine discharge.
    - vagina – by coitus or artificial insemination
    - conjunctiva
    - Skin
    - Contamination of healthy udder from an infected one during milking.
- *Pathogenesis*
    - The organism produces abortion in the following manner:
        - The organism has special affinity for the pregnant endothelium.
        - First placentitis is produced by the invading organism.
        - Sero-purulent exudate accumulates between the endometrium and chorion.
        - Edema and infiltration of the chorion by macrophage, lymphocytes and plasma cells occurs.
        - Necrosis and hyalinization of chorio-allantois.
        - Thus the membranes become separated from the uterine endometrium.
        - Fetus dies.
        - Severance of blood supply to fetus.
        - Dead fetus is a foreign body and so expelled (abortion). A few weeks after abortion or parturition, the organism can no longer be detected in the uterus.
    - Milder cases: a live fetus may be born, which is usually weak and may succumb soon after.
    - Chronic cases: In the chronic cases, there is fibrotic adhesion of the placenta to the endometrium resulting in retention of placenta. In these cases, calves may be born alive.
- *Gross pathology*
    - In the aborted fetus may be found
        - croupous or catarrhal pneumonia
        - edema of the pericardium, umbilical cord and skin
        - serosanguineous exudate in the serous cavities
        - suppurative or hemorrhagic gastro-enteritis.
- *Histopathology*: Hyperplasia of lymph nodes and spleen are prominent lesions.
- Diagnosis
    - Brucellosis is usually diagnosed with Rose bengal plate agglutination test using the coloured antigen
    - Cutural examination
    - Brucellosis of swine
    - Coital infection is more frequent. Abortion occurs between the 2nd and 3rd months of pregnancy.
- *Gross pathology*
    - Serosanguineous exudate in the serous cavities and subcutis
    - Fetal membrane show yellowish grey mucopurulent exudate
    - Brucellosis in dogs
- *Etiology*: *Brucella canis* causes abortion in dogs.

- *Transmission*
    - This is a highly infectious disease
    - Transmission may be by contact, through infective discharges or by venereal transfer.
- *Clinical signs*: In bitches, abortion occurs between the 7th and 9th weeks of gestation.
- *Histopathology*: In male dogs, epididymitis and testicular atrophy may occur.
- *Sequelae*: In male dogs, complete sterility may result.
    - Ovine Brucellosis
- *Etiology*: Ovine brucellosis is caused by *Brucella ovis*
- *Routes of infection*: Infection is probably by ingestion.
- *Pathogenesis*
    - In ewes abortion occurs due to placentitis.
    - In rams after an initial bacteremia and mild systemic reaction, the organisms localize in the epididymis and cause sterility.
- *Clinical signs*: In ewes abortion may occur in late pregnancy or still births may also result.
- *Clinical pathology*: Semen is of poor quality and contains leucocytes and brucellae.
- *Gross pathology*
    - Ram
        - There may be acute inflammation of the scrotum with edema.
        - The condition may become chronic with enlargement of epididymis, thickening of the scrotum and atrophy of testes.
    - Ewe
        - There is purulent exudate on the placenta and edema of allantois.
        - There may be elevated, firm, yellowish-white plaques in the inter-cotyledonary areas
        - The cotyledons are enlarged and edematous.
    - Brucella melitensis abortion in goats
- *Routes of infection*: Infection is by ingestion
- *Clinical signs*: Abortion may occur, but sometimes live kids may be born.
- *Pathogenesis*
    - In the goats an acute systemic reaction develops and later localization of the organisms in the placenta causes placentitis and thus abortion results.
- *Sequelae*
    - After abortion the uterine infection persists for over 5 months and the mammary glands remain infected for many years.
    - In some cases, spontaneous recovery may occur.
    - Viable kids are infected and infection persists in a latent form and at maturity clinical symptoms are manifested.
    - **VIBRIOSIS**
    - Occurrence: Vibrio abortion occurs in cows and sheep
    - Routes of infection
- Infection is by ingestion in sheep
- In cows, through coitus or artificial insemination infection occurs.
    - Pathogenesis
- The pathogenesis of abortion is similar to that of brucellosis, the initial lesion being a placentitis followed by exudation, necrosis, vasculitis, separation of the placenta from the endometrium, death of the fetus and abortion.
    - Clinical signs
- In the cow abortion occurs between the 5th and 7th months of pregnancy

- In the sheep, abortion occurs at 2nd month of pregnancy.
    - Histopathology
- Vibrio causes acute catarrhal endometritis, cervicitis and vaginitis.
- *Fetus*
    - In the fetus, edema of the subcutis, serofibrinous pleurisy, peritonitis and pericarditis may be found.
    - Fatty degeneration of the liver and kidney
    - Hemorrhages in the renal cortex.
    - Sequelae: Infection causes repeat breeding.
    - **TRICHOMONIASIS**
    - Etiology: It is caused by *Trichomonas fetus*.
    - Transmission: It is transmitted to the cow through coitus
    - Epizootiology
- The bull harbours the flagellate in the mucous membrane of the penis, terminal portion of urethra and prepuce.
    -
    -
    - Pathogenesis
- In the cow, within three days after infection, vulvitis and vaginitis develop, from where infection spreads producing cervicitis, endometritis and placentitis.
- There is copious grayish-white thin exudate and abortion will occur within about 16 weeks of pregnancy.
- Sometimes the dead fetus may be macerated in the exudate. Or the fluid may be absorbed (if not infected by other bacteria) and the fetus may be mummified.
- In chronic infection with fibrosis, placenta may be retained.
    - Sequelae
- If pyogenic organisms invade the uterus (*Corynebacterium, Staphylococci* and *Streptococci*), pyometra will result.
- Infection causes repeat breeding.
    - **LISTERIOSIS**
    - Etiology: Listeriosis is caused by *Listeria monocytogenes.*
    - Occurrence: Listeriosis causes abortion in cattle and sheep.
    - Pathogenesis
- Listeriosis primarily affects the brain.
- It may sometimes infect the pregnant uterus and the organisms become septicemic in the fetus and cause its death.
- Abortions usually occur during the last trimester of pregnancy.
    - Gross pathology
- *Fetus*
    - The fetus shows
        - hemorrhage in the kidneys
        - anasarca
        - areas of necrosis and granulomas in the liver, spleen, lungs and kidneys
        - catarrhal gastro-enteritis
        - cardiac vegetations and
        - hemopericardium.
    - **EPIZOOTIC BOVINE ABORTION**
    - Etiology

- A virus of the family psittacosis lymphogranuloma group is found to cause abortion in cows and ewes (in sheep it is called enzootic abortion).
    - Epizootiology: An arthropod vector is important in transmission.
    - Pathogenesis
- The virus causes death of the fetus.
- It injures the vascular endothelium of the fetus.
    - Clinical signs
- In an out break 75% of affected animals may abort.
- Abortion occurs during the last trimester.
    - Gross pathology
- *Fetus*
    - Lesions in the fetus are
        - Skin at the groin shows erythematous patches.
        - Petechiae found on the skin.
        - Subcutaneous edema
        - Hemorrhages on the conjunctiva, on the mucosa of ventral surface of the tongue and on the tracheal mucosa.
        - Body cavities are filled with serosanguineous fluid.
        - Petechiae found on the internal organs.
        - The pathognomonic lesions are found in the liver which is enlarged, friable, pale red to reddish-orange in color and has a coarsely granular surface (due to chronic venous congestion).
    - Histopathology
- *Fetus*
    - The characteristic lesion found in all the organs is a focal inflammation consisting of neutrophils, lymphocytes and macro-phages.
    - The liver may show chronic venous congestion (dilatation of the central vein and sinusoids and consequent pressure on the hepatic cells causing necrosis) or granulomatous lesions in the hepatic capsule, in the portal triads or in the adventitia of the central veins.
    - Meningitis and focal encephalitis
    - Infiltration of the adventitia of meningeal and parenchyma cells of the brain by pleomorphic mononuclear cells, arranged concentrically is a characteristic appearance.
    - Mild degenerative changes in the kidney, pancreas and lung may be noticed.
    - There is hyperplasia of the reticulo-endothelial tissue of the spleen, thymus and lymph nodes and so these organs are enlarged.
    - The granulomas wherever they are seen (liver, kidney, spleen, lymph node) consist of central necrotic areas surrounded by neutrophils, epithelioid cells and lymphocytes, surrounded by fibrous tissue.
    - Sequelae: Animals rarely abort more than once probably due to development of immunity.

LEPTOSPIROSIS N CATTLE

- Etiology: In cattle various strains of leptospira produce abortion
- Pathogenesis: Abortion is due to fetal death.
- Clinical signs: Leptospira causes abortion after 6^{th} month of pregnancy.
- Gross pathology

- *Fetal membranes*
    - The placenta is avascular with collapsed blood vessels.

- Cotyledons are atonic, yellow-brown in color and leathery.

- *Fetus*
  - In the fetus, edema of the subcutis, peritoneum, umbilical cord and pericardium are seen.
  - Histopathology
- Focal interstitial nephritis with round-cell infiltration, glomerulo-nephritis, and infiltration of eosinophils into the cortex.
- Round-cell infiltration into the periportal tissue of the liver.

  - **ABORTION IN MARES BY SALMONELLA ABORTUS EQUI**
  - Clinical signs: Abortions occur late in pregnancy.
  - Gross pathology
- The organisms produce a purulent hemorrhagic placentitis.
- Allanto-chorion is edematous and exhibits necrosed areas with a wall of hemorrhagic reaction separating it from the surrounding tissue.
  - Sequelae: Infection is followed by development of immunity.

  - **EQUINE VIRAL ABORTION**
  - Etiology
- Equine viral abortion is caused by
  - (1) Equine rhinopneumonitis virus
  - (2) Equine viral arteritis
  - Equine rhinopneumonitis virus (Equine influenza)
- *Clinical signs*: Abortion occurs in the 9^{th} or 10^{th} month.
- *Gross pathology*
  - The fetus shows edema of the subcutis
  - Jaundice
  - The lungs are heavy and voluminous (edema of the lungs).
  - Liver shows focal necrotic areas. Visible under the capsule are grayish-white foci. Such necrotic areas are seen in the spleen and lung also.
  - Petechiae are found throughout the body.
- *Histopathology*: Bronchial and alveolar epithelial cells show acidophilic intranuclear inclusions
  - Equine viral arteritis
- *Pathogenesis*: The virus of equine arteritis brings about the death of the fetus.
- *Gross pathology*: The fetus shows hemorrhages in the splenic capsule and respiratory mucosa.

  - **ABORTION CAUSED BY THE VIRUS OF INFECTIOUS BOVINE RHINOTRACHEITIS**
  - Etiology
- Abortion in cows may be caused by the IBR virus when the animal suffers from respiratory affection caused by this virus.
- Vaccination of cows with the IBR virus vaccine also brings on abortion.
  - Clinical signs
- Abortion occurs during the last trimester of pregnancy.
- No symptoms are noticed prior to abortion.
- There may be history of vaccination by IBR vaccine or of a respiratory affection. Fetus is usually decomposed when aborted since abortion occurs only 24 to 36 hours after its death.
  - Gross pathology
- Serous cavities contain serosanguineous fluid.

- There is edema of lungs and placenta.
- Fetus shows petechiae on the heart.
    - Histopathology
- There is focal necrotizing hepaititis and placentitis.
- Renal cortex shows hemorrhagic necrosis.
    - Diagnosis
- By clinical signs
- Affected animals are serologically positive
- Virus isolation from cotyledons.

MYCOTIC ABORTION IN CATTLE AND SHEEP

- Etiology
- Abortion in cattle may occur due to infection by fungi of the following species. *Aspergillus, Absidia, Mucor* and *Rhizopus*.
    - Pathogenesis
- The infection is a secondary one, the primary lesions being in the lungs, abomasum (ulcers) and the intestines.
- Infection is through the blood stream.
- Separation of the placenta from the cotyledons causes death of the fetus.
    - Clinical signs
- Abortion in affected cows occurs during the later half of the gestation period between 6th and 8th months.
- Placenta is retained.
    - Gross pathology
- Infection occurs first in the placentomes which show necrotic plaques and the fungus can be demonstrated in these locations.
- In the cow, the chorion-allantois is thick and leathery.
- The fetus may show circumscribed grayish plaques on the skin resembling ring worm lesions.
    - Histopathology
- The typical lesion consists of focal collection of inflammatory cells with macrophages predominating.
- Extensive necrosis of the placentomes
- In the uterine wall, the intercaruncular areas show red patches covered in places by a thin yellowish-grey pseudomembrane. Thrombosis and perivascular necrosis occur in these places and hyphae are found both in the tissues and over the mucous surface. Some degeneration of circular muscle is noticed and small arteries are hyalinised throughout uterine wall. Hyperemia and hemorrhages are common in the affected area.

ABORTION BY OTHER CAUSES

- Abortion may occur in infections by various organisms which first produce metritis followed by placentitis and abortion or birth of weak fetus.
- The following are noteworthy
    - *Cattle*
        - *Salmonella sp*
        - *Corynebacterium pyogenes*
        - *Streptococci*
        - *Stapylococci*
        - *M. tuberculosis*
        - *Actinobacillus*
        - *Pasteurella*

- *Mares*
  - *Streptococcus zooepidemicus*
  - *Klebsiella genitalium*
  - *Shigella equirulis*
  - *E. coli*
- *Ewes*
  - Virus of ovine abortion
  - *Salmonella abortus ovis*

- Poisoning by ergot
  - Ergot being an ecbolic produces violent contractions of the uterine muscle resulting in abortion.
- Neutralisation of the effect of progesterone by estrogens
  - Progesterone maintains pregnancy while estrogen terminates it by inducing uterine contraction.
- Poisons
  - Chlorinated naphthalenes (which are anti-vitamin A and so may produce metaplasia of the uterine epithelium, infection and separation of the placenta)
  - Purgatives
  - Nitrates through ingestion of plants containing large quantities of this chemical.
  -
- Faulty nutrition of the mother
  - Deficiencies of minerals and vitamins may lead to abortion.
- Vaccination of mother during pregnancy against bacterial and viral diseases.
- Severe and acute septicemic diseases of the mother
  - Abortion may frequently occur in leptospirosis, dourine, viral diarrhoea, hog cholera, erysipelas and infectious rhino-tracheitis.
- Hereditary predisposition.
- Torsion of the umbilical cord (rare).
- Traumatic injury to the placenta (very rare).
  - **PATHOLOGY OF MAMMARY GLAND**
    - **MASTITIS (MAMMITIS)**
  - Definition: Mastitis or inflammation of the udder
  - Introduction

- All domestic animals suffer from this condition, but it is in the cow that mastitis is of importance because of the economical loss the owner may suffer.
  - Etiology
- Mastitis may be caused by trauma of various kinds.
- The most common causes are the infectious agents.
  - Bovine Mastitis
- The bacteria that have been found to cause mastitis, are *Streptococcus agalactiae: Streptococcus dysagalactiae; Staphylococcus aureus* and *albus; Corynebacterium pyogenes; E. coli; Pseudomonos aeruginosa, Pasteurella multocida; Brucella abortus: Mycobacterium tuberculosis; Actinomyces bovis; Actinobacillus lignieresi; Nocardia; Mycoplasma*and *Cryptococcus neoformans*.
- *Staphylococcus aureus* has been found to be the major cause of mastitis in cows.
- In India, *Staphylococcus aureus* and *pyogenes* have been isolated from large number of mastitis cases. In some herds, on the other hand, gram negative organisms (*E. coli* and*Aerobactor aerogenes*) have been isolated.
  - **STREPTOCOCCUS MASTITIS**

- When once udder becomes infected with Streptococcus agalactiae, it never becomes free of this organism.
- Though some kind of equilibrium develops between the udder and the organism, at times acute exacerbations may occur when the organisms multiply and increase in great numbers.
- In a herd all cows are not equally affected.
    - Routes of infection
- The route of infection appears to be through the teat canal.
- Wounds that occur in cow pox or those caused by suckling calves facilitate infection.
    - Source of infection
- Contaminated cups of milking machines, milkers hands and farm utensils are other sources of infection.
    - Resistance to infection
- The teat canal is lined by the same type of epithelium that covers the teat, but this epithelium secretes a type of smegma (rich in fatty acids) and this inhibits the streptococci.
    - Pathogenesis
- Milk being a good medium for the growth of bacteria infection is much more serious in a lactating udder than in a dry one.
- The acute systemic symptoms are due to the action of the bacterial toxins that diffuse into the general circulation.
- The development of mastitis can be described under three phases
    - The invasion phase in which the bacteria are able to enter the teat orifice and be present in the teat canal and cistern.
    - The infection phase in which the organisms are able to overcome the resistance and multiply
    - The inflammatory phase in which the organisms invade the udder.
    - Gross pathology
- One or more quarters may be affected.
- The gland is swollen and slightly hard.
- The secretion may be serous or may contain floccules and sometimes it may be purulent also.
- On section of gland, the silky pink color of normal udder is lost but is red or white. Lobulation is distinct.
- When fibrosis has set in it can easily be seen surrounding the lobules and the ducts.
    - Histopathology
- Streptococci are numerous in the ducts
- The epithelium of the acini becomes vacuolated and desquamated.
- When the streptococci invade the epithelium of the ducts inflammation results and due to the rapid development of the granulation tissue beneath the epithelium it is thrown into folds of polypoid thickening.
- The organisms may penetrate the interstitial tissue and cause edema and infiltration by neutrophils which destroy some of the organisms.
- The lymphatics in the stroma become widely dilated due to infiltration by leucocytes that migrate from the regional lymph node.
- The exudation process gives rise to pathological fibrosis and involution of the acini. Subsequently macrophages and fibroblasts increase in number while neutrophils decrease. There may be stagnation of secretion in the smaller ducts and at this stage the udder may be firm and indurated due to the inflamed interaveolar tissue and retained secretion.

- When acute stage passes off and the damage caused is slight regeneration of the acini may occur. But if there is large scale destruction, regeneration is not possible and so the acini collapse and are replaced by granulation tissue. Interstitial spaces are infiltrated by lymphocytes. Such a gland is reduced in size and becomes hardened in consistency – "the shrunken quarter".
    - **TYPES OF MASTITIS**
    - Mastitis caused by *Streptococcus dysagalactiae*
- Mastitis caused by *Streptococcus dysagalactiae* is more severe than that caused by *Streptococcus agalactiae* and more destructive leaving non-functional udder.
    - Mastitis caused by *Streptococcus uberis*: *Streptococcus uberis* produces a mild and chronic mastitis.
    - Mastitis caused by *Staphylococcus aureus*
- *Staphylococcus aureus* usually affects younger animals; especially after, parturition.
- Infection is supposed to be contagious and through the teat canal.
- In acute cases mortality may be high due to toxemia
- There may be a peracute and fulminating type or more commonly a chronic type.
- *Gross pathology*
    - Pitting edema is seen in the flank, inguinal region and the ventral aspect of the abdomen anterior to the udder.
    - The udder is hard, swollen and very painful.
    - Secretion of milk is very little and that too blood stained.
    - Uninfected quarters are also swollen because of the action of tkyy2qhe toxin (alpha) that has diffused into them.
    - Gangrene may supervene when infection spreads to the blood vessels causing thrombosis. The udder then becomes cold and greenish or blue.
    - Gas may be present in the affected gangrenous area producing crackling sound on pressure. In such cases death may supervene or if the animal survives, the udder is totally lost.
- *Histopathology*
    - Differing from the streptococcal variety, in this type organisms persist in the interstitial tissue producing the granulomatous lesions
    - In less severe cases necrotic foci are found surrounding which is zone of leucocytes and this in turn is enveloped by fibrous tissue. This granulomatous lesion is known as botriomycosis , in the centre of which can be seen gram positive cocci. The udder tissue contains numerous such granulomata. Fibrosis ultimately occurs resulting in the shrunken quarter.
    - Mastitis caused by *Staphylococcus pyogenes*
- Staphylococcus pyogenes produces a very acute type of mastitis accompanied by severe systemic disturbances and fever.
- Gangrene may supervene.
- Animals die in a few days.
    - Mastitis caused by *Corynebacterium pyogenes*
- *Corynebacterium pyogenes* is the cause of the so called " Summer Mastitis " affecting both immature and lactating glands.
- The organism being pyogenic, large amount of pus is produced, resulting in abscesses.
- There may be fistula discharging the pus to the exterior as well as large scale necrosis and sloughing. The latter are due to thrombosis.

- Fibrosis with loss of function results in those animals that survive.
    - Mastitis caused by Coliform organisms
- Coliform organisms, *E coli* and *Aerobactor aerogenes*, produce, sometimes, an acute inflammation of the udder.
- Infection is supposed to be by blood stream though galactogenic infection cannot be ruled out.
- *Clinical signs*
    - The affected quarter is hot, painful and edematous.
    - Clotted milk, sometimes blood tinged may be seen.
    - Infection may subside, with involution of the quarter or it may become chronic with acute exacerbations developing later.
    - Sometimes severe general toxic symptoms may be noticed with death following due to the potent toxins.
    - **Gangrenous Mastits**
- In severe cases of mastitis caused by virulent strains of organisms thrombosis of the mammary vessels occurs resulting in infarction and gangrene. *Staphylococcus aureus* and *E coli*with *Clostridium welchii* produce this condition. Usually all the four quarters may be affected.
- The udder becomes cold, and bluish within 3 to 4 days after infection.
- In many cases death may supervene.
    -
    -
    -
    - **Cryptococcal mastitis**
    - Etiology
- Cryptococcal mastitis is a surgical hazard that may be encountered in repeated intra-mammary infusions.
- *Cryptococcus neoformans* produces an acute inflammatory reaction.
    - Clinical signs
- One or more quarters may be affected.
- The gland becomes hard.
- The milk turns to a watery, flaky secretion.
    - Gross pathology
- The gland is fleshy and interlobular septa are distended with edema.
- There is large-scale destruction of the glandular tissue and the alveolar and ductal epithelium is liquefied to form a viscid, mucoid material.
- Organisms may be found in the supra-mammary lymph gland.
- Metastases, in some cases, may be found in the lungs.
    - Histopathology
- In sections, the double refractile fungus can be seen in large numbers, some of which are found engulfed by the histiocytes.
- In some isolated chronic cases granulomatous nodules with interlobular and intralobular fibrosis occurs together with infiltration by histiocytes and lymphocytes.
    - **Brucella Mastitis**
- In infection by Brucella, udder is the reservoir of the organism, which is excreted through the milk.
- Regional lymph nodes may or may not be swollen.
- The udder may not show any changes or only scattered lesions may be observed which are not very characteristic.

- Histologically the lesion is a granuloma which is intralobular. It consists of lymphocytes, plasma cells, histiocytes and a few Langhans' type giant cells.
- In some cases, instead of the granuloma an interstitial mastitis is seen with infiltration of lymphocytes, histiocytes, plasma cells and fibrous tissue.
    - **Mycoplasmal Mastitis**
- This is caused by several strains of *Mycoplasma sp*.
- Cows of all ages are affected.
    - Source of infection
- Faulty milking machines
- Unsterilized teat syringes
- Contamination of teats during milking
- Inhalation
    - Clinical signs
- All four quarters may be involved
- Sudden drop in milk yield.
- Milk becomes abnormal grossly.
- There is cessation of lactation and the animal will not be useful again for dairy purposes.
    - Gross pathology
- The condition is a purulent mastitis
- Milk is thick and cheesy and may be tinged with blood. There may also be clots or granular material in some specimens.
- The udder is swollen and later it gets atrophied.
- The organism may also invade the blood and then affect the joints and other tissues causing systemic symptoms. There is arthritis with swelling of the joints and lameness.
    - **OTHER ORGANISMS WHICH CAUSE MASTITIS**
- The following organisms also may cause mastitis
    - *Pseudomonas aerugionosa*
    - *Pasteurella multocida*
    - *Nocordia asteroids*
    - *Candida sp*
    - *Mycobacterium tuberculosis.*
    - **MASTITIS IN EWES**
    - Etiology
- The organisms responsible for mastitis in ewes are *Staphylococcus aureus, Pasteurella hemolytica, Corynebacterium pyogenes, Streptococci* and *Coliforms.*
- *Staphylococcus aureus* produces a more acute disease than in the cow.
- Mastitis caused by other organisms is less severe than that produced by *Staphylococcus aureus*.
    - **MASTITIS CAUSED BY STAPHYLOCOCCUS AUREUS**
    - Routes of infection
- Infection is usually ascending through the teat canal.
- Injuries made by suckling lambs provide a route of entry for the organisms.
    - Clinical signs
- Morbidity is 25%

- Mortality is greater than in the cows, being 25 to 50%.
- There are severe systemic disturbances with intense edema of the udder, which may be extended up to the belly
- Gangrene may supervene, when the udder assumes blue color and so the condition is called **"blue bag".**
    - Histopathology
- There is serous or serofibrinous or purulent exudate in the acini, interlobular septa and in the interacinar septa.
- Alveolar exudate contains desquamated epithelial cells and leucocytes.
- Large scale necrosis may occur with abscess formation. Abscesses may rupture on the skin.
- Ultimately, the gland becomes fibrosed.
    - Sequelae: If the gland becomes fibrosed, it may become functionless
    - **CONTAGIOUS AGALACTIA OF SHEEP AND GOAT**
    - Introduction: This is a disease primarily of goats but slightly infective to nearby sheep also.
    - Animals affected: Both adult goats and kids are susceptible.
    - Etiology: The causal agent is *Mycoplasma agalactiae*.
    - Epizootiology
- The infective agent is eliminated in the secretions and discharges.
- Organisms are voided in the milk.
    - Routes of infection
- Infection is probably by ingestion, though it may also occur by way of teat canal and the conjunctiva.
    - Pathogenesis
- Though the udder is mostly affected, the disease may run a septicaemic course in which mortality is heavy (10 to 33%).
- Later, infection localizes in the eyes, joints and the udder.
    - Clinical signs
- Pregnant animals may abort.
- If live kids are born, they may be found infected.
- Lameness, if joints are affected
    - Histopathology
- *Mammary gland*
    - The inflammation commences in the interstitial issue, with fibrosis. Later acini may be involved and they may be atrophied as the fibrous tissue increases and encroaches on the acini.
    - Ultimately the udder is completely fibrosed and so last.
- *Eye*
    - In about half the number of cases mucopurulent conjunctivitis and keratitis complicated by ulceration are noticed.
- *Joints*
    - Mostly the carpal and tarsal joints are affected showing arthritis and periarthritis.
    - The peri-articular tissues appear swollen due to inflammatory edema.
    - **MASTITIS IN PIGS**
    - Staphylococcal mastitis
- *Staphylococcus aureus* causes sporadic cases of mastitis.

- The condition is chronic with the formation of large fibrous nodules in the gland. The nodules may open out through sinuses and then pus containing small granules may be discharged.
- The udder is lost ultimately.
    - Mastitis caused by Coliform bacteria
- *Introduction*
    - This is usually found in sows heavily fed with concentrates and in those which farrow in unhealthy pens.
    - Heavy losses are encountered.
- *Clinical signs*
    - There may be severe systemic reaction.
    - The sow does not get up and does not allow piglets to suckle. So the piglets die of starvation. If they suckle, they suffer from enteritis.
- *Gross pathology*
    - The udder is swollen, hard, discolored and painful.
    - There may be concomitant metritis.
- *Sequelae*
    - The udder is lost
    - If the pig survives, it will not be useful for breeding.
    - Mastitis caused by *C. pyogenes*
- This is very frequent in pigs.
- The infection may be primary or it may be secondary from metastatic involvement from a focus somewhere else.
- In the udder there are abscesses with central collection of greenish pus.
- Fistulae may be present opening out on the skin from the abscesses.
- Affected animals are no more useful for breeding.

    - **NEOPLASMS**

- Tumors of the mammary gland are common only in the canine species in which the mixed tumor (fibrochondro-adenocarcinoma etc.) is frequently seen.

    -
    - **OVIDUCT OF THE BIRDS**
    - Abnormal eggs
    - The following types of abnormal eggs may be encounted.

- *Double yolked eggs*: In this condition two yolks enter the oviduct simultaneously and so are enveloped in a single layer of albumen, membrane and shells.
- *Ovum in ova*: In this condition there is fully formed smaller egg within a large one. A fully formed egg, due to reverse peristalsis reaches anterior parts and comes in contact with another ovum and gets attached to it. Then this combined mass while moving downwards is enclosed with albumin and shell.
- *Yolk-less eggs*: In this variety, the fully formed egg does not contain yolk. The explanation offered is the yolk that is released does not enter the oviduct due to closure. But at the same time stimulates the oviduct to produce various layers of albumin, membrane and shell or a nidus of exudate may be coated by albumin etc. to form a yolk-less egg.
- *Soft shelled eggs*: This variety is also known as leathery eggs. In this a shell is lacking. The causes are various and include deficiency of calcium, vitamin D or there may be disturbances of secretion of calcium by the shell gland. This disturbance may be due to:
    - Poisons – chemicals such as Zinc sulphide used as sprays on tree.
    - Infective agents as in infectious bronchitis or

    - Too rapid peristalsis at that part.
- *Layered eggs*: In this the various layers are duplicated. This is due to forward and backward movement of the egg mass so that various substances are deposited again and again.
- *Egg concretion*: When there is some inflammatory process of the oviduct the egg may be retained and so more and more layers of albumin, membrane and calcium are deposited. There may be deposition of fibrin also and the whole mass may be dehydrated and calcified to form a huge mass of concretion.
- *Foreign bodies with eggs*: Eggs may contain various foreign bodies. These may be
    - Feed, feathers, droppings, small pieces of wood etc that have reached through the cloaca, upper parts of the oviduct by antiperistalsis and get incorporated into an egg.
    - Parasites – *Prosthogonimus macrorchis* which is found in the Bursa of Fabricius normally may go up the oviduct and be incorporated in an egg. So also *Ascaridia galli* may go up the oviduct from rectum and be incorporated in an egg.
    - Blood clots may be found when hemorrhage occurs due to rupture of vitalline membrane.
    - Abnormal location of eggs
- Sometime eggs in various stages of development may be found in the abdominal cavity.
- They may be there because
    - Yolk does not get sucked by the infundibulum and so is found in the abdominal cavity.
    - By antiperistalsis, partially formed egg may be thrown out through the infundibulum into abdominal cavity.
    - The oviduct may rupture so that the egg is found in the peritoneal cavity.
- *Sequelae*
    - The eggs and their contents acting as foreign bodies produce peritonitis and death.
    - Sometimes, if secondary infection occurs the organisms multiply quickly since the egg contents are good medium, death results.
    - Egg bound
- *Definition*
    - Egg bound in the fowl is a condition in which the egg is lodged tightly in the oviduct or cloaca and is not laid.
- *Etiology*
    - This condition may be due to
        - Narrowing of the oviduct as a result of inflammation, thereby making it difficult for the egg to be laid.
        - Far too large an egg like a double yolked egg.
        - Paralysis of the muscles of the oviduct. In this condition the egg is not propelled further towards the cloaca.
- *Sequelae*
    - This gives rise to local irritation, inflammation, peritonitis and death if not relieved in time.

CRYPTORCHIDISM

    - Definition: Retention of testes in the abdominal cavity is called cryptorchidism.
    - Occurrence: Cryptorchidism is more often seen in horses and swine.

- Etiology: In horses, it is hereditary and caused by a sex- linked dominant factor.
- Pathogenesis

- As the abdominal cavity temperature is higher than the scrotal temperature, the testes do not develop normally.
- Hence, spermatogenesis is arrested.
  - Clinical signs
- Cryptorchidism may be unilateral or bilateral.
- The horses with this condition sometimes have increased sexual urge and becomevicious and difficult to control.
  - Gross pathology: Testes – small and softer
  - Histopathology
- The Leydig cells may be normal or hyperplastic.
- The seminiferous tubules lack spermatogenic activity
- The interstitial space has more fibrous tissue
  - Sequelae
- If the cryptorchidism is bilateral, the affected animal will be sterile.
- Dogs: Sometimes tumours like seminoma and sertoli cell tumour develop.

HYPOPLASIA OF TESTES

- Occurrence

- Hypoplasia of testes is seen in all animals.
- But it is of greater economic importance in bulls.
  - Etiology
- In Swedish Highland cattle, it is hereditary.
- Hormonal disturbances
- Vitamin deficiencies
- Some poisons
  - Gross pathology : Hypoplastic testes is small and harder
  - Histopathology
- Severe condition
  - The seminiferous tubules are narrowed and have only one layer of cells which do not show mitotic activity (cessation of spermatogenesis)
  - The basement membrane is thick and hyalinized
  - Peritubular connective tissue is increased (Hence the testes is harder)
  - Leydig cells may be increased in number
- Mild condition
  - Varying degrees of spermatogenesis is found and some show giant Spermatids.

CONGESION IN TESTES

- Congestion of testis may be seen in heat storke or systemic infections

HYDROCELE

- Definition: Hydrocele is a condition in which clear serous fluid accumulates in the tunica vaginalis.
- Etiology

- This condition may be seen along with generalized edema or ascites
- Trauma (Here the fluid may be turbid or blood stained)

HAEMATOCELE

- Definition: Hematocele means presence of blood in the tunica vaginalis
- Etiology

- Trauma

- Haemoperitoneum
- Leptospirosis
- Infectious canine hepatitis
    - Gross pathology
- Haemolysed blood is seen in the tunica vaginalis in leptospirosis and infectious canine hepatitis.

TESTICULAR DEGENERATION

- Definition: Degeneration of the seminiferous epithelium
- Introduction: This is the commonest type of bull infertility encountered
- Occurrence: Testicular degeneration may be unilateral or bilateral
- Predisposing factors
- Failure of thermoregulation due to excess scrotal fat, short cremaster muscle, scrotal / inguinal hernia, periorchitis, edema of scrotum and dermatitis.
    - Etiology
- Physical – Excessive heat, freezing temperature, trauma,haematoma and laceration of scrotum and radiation
- Localised / systemic infection causing fever, toxaemia, orchitis, inflammatory changes of tunica vaginalis, scrotum and epididymitis.
- Vascular lesion caused by torsion of testes, spermatic cord compression, testicular biopsy, inflammation of testicular artery (seen in horses caused by migrating strongyl larvae)
- Artery – thrombus, hyalinization in arteries lead to testicular degeneration
- Obstructive lesion in the head of epididymis interferes with the flow of spermatozoa and testicular tubular secretions. The back pressure cause degeneration of germinal layers of the seminiferous tubules.
- Nutritional deficiency of vitamin - A, phosphorus, protein and energy
- Toxic substances like arsenic, metals, cadmium chloride and naphthalene
- Hormonal imbalance of FSH and LH and improper administration of hormones
- Auto immunization with auto / isologus spermatozoal materials
    - Pathogenesis
- Avitaminosis causes testicular degeneration through inhibition of release of gonodotrophins.
- Cadmium chloride causes lesions in the vascular endothelium resulting in thrombosis
- Radiation affects highly sensitive spermatogonia-B and late spermatogonia –A.
- The testicular degeneration caused by highly cholorinated naphthalene is generally reversible.
    - Signs - Clinical Pathology
- *Semen*
    - Densty : Poor
    - Count : Low
    - Motility : Less
- Abnormal sperms increase to 30 -50 %. Head abnormalities like detached heads, variation in size and shape of heads are common.
- Presence of proximal protoplasmic droplets, looped middle piece and tail, tight coils of middle piece and tail are other abnormalities encountered.
    - Gross Pathology
- Tunica albugenia – Thick, wrinkled
- *Testes*
    - Size may be normal or smaller
    - Soft and flabby

    - Do not bulge on cut surface
- *Chronic cases*
    - Testes - size is smaller; Consistency: Firm
    - In necrotic area calcium deposits appear as yellowish white flakes.
    - Histopathology
- Histological changes vary with severity and stage of degeneration
- The degenerative changes may not involve the tubules uniformly
- The entire length of some tubules may be affected, while in others only partial
- Tunica albuginea is condensed, thick and wrinkled.
- *Seminiferous tubules*
    - Tubules collapsed, degenerated and degenerated tubules may contain calcium deposits.
    - In acute degeneration deposition of calcium is observed in the connective tissue.
    - Germinal epithelium is denuded leaving the basement membrane exposed
    - In early stages of degeneration, failure of maturation of spermatozoa and degeneration of spermatids occurs. Later spermatids undergo necrosis. Some of these give rise to multinucleate phagocytic giant cells
- Spermatogonial cells – Cytoplasm is vacuolated; Nucleus is pyknotic
- Lastly even the very resistant sertoli cells may be denuded. The tubules then collapse and are replaced by connective tissue
- In the lumen of stenotic tubules, large polyhedral and binucleate cells with granular eosinophilic cytoplasm are observed. These may contain a golden yellow **wear and tear pigment** in the cytoplasm. These cells are thought to arise from the altered spermatogonia with an unusual capacity to survival. In addition, stagnation of spermatozoa and tubular secretions, fragmentation of basement membrane occurs
- Contact of degenerated sperms with the connective tissue causes granulomatous reaction
- *Chronic case*
    - Sometime tubules are replaced by dense hyaline connective tissue
    - Basement membrane – Thick and hyalinized
    - Interstitial tissue is increased

ORCHITIS

    - Definition: Inflammation of testis is called orchitis
    - Introduction
- Since the testes is located compactly in the tunica albuginea, a tough fibroelastic membrane, swelling of the testes to any appreciable degree does not occur.
    - Occurrence: Orchitis occurs more frequently in sheep, cattle and swine.
    - Etiology
- Trauma – This is more common in rams as they have pendulous testes.
- Bacteria – The infection may extend from the lower part i.e. epididymis or may be haematogenous.
- The most common bacteria causing orchitis are
    - *Brucella abortus* in bulls
    - *Brucella suis* in boars
    - *Corynebacterium pyogenes, Corynebacterium ovis* and *Pasteurella pseudotuberculosis rodentium* in rams
    - *Salmonella abortus equi* in donkeys and stallions
    - *Pseudomonas mallei* in stallions

    - *Mycobacterium tuberculosis*
    - Clinical signs
- *Brucella abortus* in bulls
    - It is an acute condition
    - The scrotum is swollen, hot and painful
- Chronic cases: Testes is shrunken and hard
    - Gross pathology
- Inflammatory exudate may accumulate in the tunica vaginalis and scrotum which is responsible for the swelling of the scrotum. The exudate is fibrino purulent and sometimes haemorrhagic.
- Adhesion of the parietal and visceral layers of tunica vaginalis may occur.
- Due to pressure by the exudate and the action of the pathogen, necrosis of the testes occurs with suppuration and abscess formation
- Abscesses may open out on the scrotum
    - Histopathology
- There is microcyst formation
- Seminiferous epithelium show degeneration and desquamation
- Interstitial tissue is infiltrated with lymphocytes, macrophages and plasma cells
- In suppurative orchitis, there will be a suppurative area filled with neutrophilic exudate.
- The bacteria can be seen in large numbers in the epithelial cells and necrotic areas
- *Chronic cases*
    - Tubules and intertubular connective tissue contain military granulomas
    - Tubular epithelium shows degenerative changes and atrophy
    - Interstitial tissue has more amount of fibrous tissue
    - Sequelae: In many cases an accompanying epididymitis is present
    - Orchitis in boars
- Testes has multiple abscesses consisting of a central necrotic and caseated material surrounded by epithelioid cells, Langhan's type giant cells, plasma cells, lymphocytes and a connective tissue capsule.
    - *Salmonella abortus equi* in donkeys and stallions
- It causes acute suppurative orchitis
- The affected animal has febrile reaction
- The seminiferous tubules rupture and the sperms spill into the interstitial space and so a foreign body reaction is set up
    - *Pasteurella pseudotuberculosis rodentium* in rams
- It is characterized by suppurative orchitis
- Here the pathogen is transmitted from the rodents by tick bites
    - Tuberculous orchitis
- The lesion is usually a secondary Haematogenous infection from a primary focus somewhere else
- Testes contain typical miliary calcified nodules
- Microscopically the nodules has typical histology of tubercle nodule or it may be in the form of diffuse caseating lesion radiating from the rete testes
- Very often epididymis is also affected
    - Strau's test
- The test is used for the diagnosis of following diseases
- *Malleomyces mallei* (Glanders)
- Malleomyces pseudomallei (Melioidosis)
- *Cryptococcus neoformans* (Epizootic lymphangitis)

- *Coynebacterium ovis* (Ulcerative lymphangitis and ovine lymphangitis)
- *Brucella abortus*
- *Pseudomonas aeruginosa*
- It is an experimental infection of male guinea pigs with the above organisms.
- It is characterized by suppurative orchitis and periorchitis in 2-3 days following an intraperitoneal injection of a small amount of the culture of the organism.
    - **EPIDIDYMITIS**
    - Definition: Inflammation of the spermatic cord is called epididymitis.
    -
    - Etiology
- It is usually seen along with orchitis – orchitis may extend into epididymitis
- Bacteria – *Brucella ovis* in rams
    - Gross pathology
- In the chronic stage, the epididymis enlarged 3- 4 times and becomes hard
- Occlusion of the lumen by the debris and exudate results in spermatocyst formation. If the cyst ruptures into tunica vaginalis, a dense adhesion takes place involving the visceral and parietal layers
- No lesion may be seen in testes
    - Histopathology
- Usually the tail of epididymis is affected
- Granuloma is formed
- The organism incites an inflammatory reaction at the site of its localization with edema, infiltration with lymphocytes and macrophages. Later neutrophils arrive at the site.
- The epithelium shows papillary hyperplasia and later hydropic degeneration
- The interstitial tissue shows increased fibrous tissue
- If there is rupture of spermatocyst, the tunica vaginalis has a foreign body reaction
    - Sequelae
- Due to stasis of sperms, secondary degenerative changes with calcification may occur in the seminiferous tubules.
    - **PATHOLOGY OF SPERMATIC CHORDS**
    - Funiculitis
- *Definition*: Inflammation of the spermatic cord is called funiculitis
- *Occurrence*: Common in pigs due to contamination of the castrated wound
- *Etiology*
    - Physical injury caused during castration
    - Contamination of the castrated wound with bacteria - *Staphylococcus aureus*
- *Types*
    - Acute and necrotizing in pigs
    - Chronic (Scirrhous cord) in horses and cattle
- *Histopathology*
    - There is excessive formation of granulation tissue at the site of castration wound.
    - Abscesses with thick walls may be present in this tissue (Botriomycosis). Centre has granules (colonies of bacteria) surrounded by zone of club and inflammatory cells (Leucocytic infiltration).
    - In the horse, testes and spermatic cord has verminous granulomas caused by larvae of *Strongylus spp* may be found occasionally.
    - **PATHOLOGY OF SEMINAL VESICLES**

    - Growth Disturbances
- Segmental aplasia of ampulla and seminal vesicles
    - This usually occurs in association with segmental defects of the epididymis.
- Duplication of seminal vesicles: It may occur unilaterally or bilaterally
    - Seminal Vesiculitis
- *Definition*: Inflammation of the seminal vesicle is called seminal vesiculitis
- *Introduction*
    - If it occurs in bulls used for artificial insemination, it is of serious concern as the pathogenic organisms can be transmitted to a wide population.
- *Occurrence*: Rare.
- *Etiology*
    - Bacteria – *Brucella abortus, Corynebacterium pyogenes* and *Mycoplasma*
- *Gross pathology*
    - Seminal vesicle
        - In most of the acute stages it is enlarged and tender on palpation. There is a tendency for loss of lobulation.
        - If it is not enlarged, massage the vesicles and stripping of the ampullae will force the inflammatory cells into the urethra and the exudate will drib from the penis.
        - Semen collected following this procedure has marked increase in leucocytes and there may be clumping of the exudate and the sample will have the appearance of the curdled milk.
- *Histopathology*
    - Acute stage: Alveoli and interstitial tissue are infiltrated with neutrophils
    - Chronic stage
        - Interstitial tissue is infiltrated with lymphocytes, plasma cells and macrophages.
    - **PATHOLOGY OF PROSTATE**
    - **Hyperplasia / Hypertrophy of prostate**
    - Animals affected: Dogs
    - Occurrence: Dogs above five years old and house-bred are commonly affected
    - Etiology
- Urinary retention in household dogs
- Increased testosterone level in dogs
    - Clinical signs
- Due to pressure on the rectum by the enlarged prostate constipation may be produced.
- Difficulty in micturition is attributed not to the pressure on or narrowing of urethral lumen, but to paresis of the bladder resulting from pressure of the enlarged gland on the parasympathetic nerves.
    - Gross pathology
- Prostate
    - Enlarged and sometimes the bilobed appearance of the gland is lost
    - The surface may be smooth, nodular or cystic
    - Histopathology
- The microscopic picture resembles that of hyperplastic adenoma
- Acinar cells are increased both in number and size.
- The epithelium is tall and is frequently thrown into folds as papillary projections into the lumen.

- Some acini may be cystic with increased amount of secretion which presses upon the epithelium, flattening it.
- The interlobular connective tissue may be increased.
- There is always infiltration of lymphocytes and plasma cells in the interstitial tissue.
- Bladder may show compensatory muscular hypertrophy.
    -
    - **PATHOLOGY OF PENIS AND PREPUCE**
    - Definition
- Inflammation of glans penis is called **balanitis.**
- Inflammation of the prepuce is called posthitis. Usually both occur together as balanoposthitis.
    - Occurrence: In the dog, it is common condition.
    - Etiology
- In the dog, the cause may be trauma or bacteria.
- In other animals, balanoposthitis is associated with various organisms including *Pseudomonas aeruginosa; Corynebacterium pygenes* and *C.renale.*
- This condition is also met with in bulls that cross cow suffering from "Infectious pustular vulvovaginits":
    -
    - Gross pathology
- There is catarrhal exudate
- Mucosal lymph follicles may be enlarged.
- In the bulls (known as infectious pustular balanoposthitis) pustules form on the preputial lining and glans penis, giving them a granular appearance. Infection does not extend into urethra. Edema of penis and prepuce may cause paraphimosis.
    - Histopathology
- There is catarrhal exudate with infiltration of leucocytes into the degenerated epithelium.
    - Phimosis
- Definition
    - It is a condition in which the penis cannot be extended from the prepuce, due to inflammatory swelling.
    - **PARAPHIMOSIS**
- Definition
    - It is a condition in which extended penis due to inflammatory enlargement, cannot withdrawn into the prepuce.
    - **NEOPLASMS**
- In the bull, transmissible fibro papilloma is encountered. These are multiple and cauliflower-like.
- In the horse squamous cell carcinoma may be wet with
- In the dog transmissible venereal tumor is common.
    -
    -
    - **MUSCULOSKELETAL SYSTEM**
    - **SKELETAL SYSTEM**
    - **NORMAL BONE FORMATION**
    - Bone cells
- Bone has three types of cells. They are as follows

    - Osteocytes are the ordinary bone cells that are found in the lacunae. These are old cells that cannot divide.
    - Osteoblasts are bone producing mesodermal cells and line the deep layer of periosteum, the endosteum and the Haversian canals. These cells like fibroblasts have great power of proliferation and produce alkaline phosphatase. Osteoblasts secrete precursors of collagen and mucopolysaccharides. The latter act as the cement substance and in this is embedded collagen. These form the matrix of the bone called osteoid.
    - Osteoclasts are the phagocytes of bone and are multinucleated. Foreign body giant cells can be formed from them. These are under the control of parathyroid and under its influence remove bone.
    - Normal calcification
- The cartilage cells nearest the diaphysis become degenerated and calcified.
- Then the capillaries nearby invade and corrode the cartilage, on the remnant trabeculae of which the osteoblasts arrive and form the organic matrix, the osteoid, over which calcium salts are deposited.
    - Various factors which govern bone formation
- *Proteins:* Sufficient amount of protein must be fed for the formation of the ground substance- osteiod.
- *Minerals:* sufficient amount of calcium and phosphorus must be supplied in the food in correct proportion and the intestinal tract must be` healthy and of correct pH for their absorption. Normally, the Ca : P ratio in food should be 2:1.
- *Vitamins:* Vitamins A, D and C control bone formation.
    - Vitamin A deficiency produces inanition and growth rate is retarded. It is concerned with the metabolism of endothelial cells and so is required for the proliferation of endothelial cells of the capillaries, for their transformation into osteoblasts and for the erosion and removal of the calcified cartilage.
    - Vitamin D controls absorption and utilization of calcium and phosphorus.
    - Vitamin C controls the formation of osteoblasts and so controls deposition of osteoid.
- *Alkaline phosphatase:* The alkaline phosphatase found in bone is formed by the osteoblasts. This enzyme splits the organic phosphate compounds liberating excess of phosphate which upsets the local calcium phosphate balance leading to the precipitation of calcium salts. It is in this manner that mineralization of the ostoeid (the organic matrix of the bone) takes place.
- *Endocrines*
    - Parathyroid controls calcium and phosphorus metabolism.
        - It increases the phosphate diuresis and
        - It produces hypercacemia through its action on the osteolcasts which with-draw calcium form the bone.
    - Anterior Pituitary
        - The growth hormone influences the growth of connective tissue especially bones. Gigantism occurs when there is increased secretion of growth hormone and the bony growth is enormous.
    - Thyroid
        - Thyroxin controls the metabolism of carbohydrates and fats and so energy production is under its control. Indirectly therefore, bone formation is influenced by the thyroid as energy production is controlled by it. In hypothyroidism, there is retardation of

endochondral bone formation and osteoporosis which occurs due to negative metabolism balance.

- Gonads and Adrenal cortex
  - Bone, growing and mature is affected by estrogens and androgens. These hormones accelerate the epiphyseal closure and maturation of the bone. In deficiency of these hormones, there is disproportionate elongation of immature lone bones.

ANOMALIES AND ABNORMALITIES OF BONE

- The following terms indicate certain anomalies and abnormalities in the skeletal system.

Amelia	Absence of limbs. The scapula and pelvic girdle may be intact or rudimentary.
Abrachia	Absence of anterior limbs.
Apodia	Absence of posterior limbs.
Micromelia	All parts of limbs are present but are of smaller size.
Perodactyly	Absence of all toes in a limb.
Brachydactylism	Abnormal shortening of toes.
Polydactylism	Presence of more number of digits; seen in horse and pig.
Syndact	Fusion of toes seen in cattle and pigs.

ylism	
o Prognathism	o Having a long jaw; pig-mouth condition in horse
o Brachygnathism	o Having a short jaw; parror-mouth in horse.
o Kyphosis	o Abnormal dorsal curvature with prominence of spine (hump back) is called kyphosis. It is rare in animals.
o Lordosis	o It is the curvature of the spine with a ventral convexity due to heavy loads or heavy abdominal organs; terminal parts of the thoracic spine and the lumbar spine are involved. The spinous processes rub against each other and so periostic osteophytes develop.
o Scoliosis	o Abnormal lateral curvature of the spinal column is called scoliosis. It may be congenital and sometimes inherited. It may be due to disease of bones like achondroplasia, osteodystrophy.
o Torticollis (Wry neck)	o This is twisting of the neck with an unnatural position of the head.
o Osteodystrophy	o It denotes disturbance in the growth of bone. Osteodystrophies may be acquired or congenital. It may be due to • Lack of minerals and vitamins (Rickets and osteomalacia etc.) • Excessive hormones- gigantism acromegaly, osteoporosis. • Unknown causes.

o

o **RICKETS**

o Definition

- This is a condition seen is young growing animals in which there is a failure of adequate calcification of bones.
- Similar condition in adult mature animals in which growth of bones has stopped is known as osteomalacia – literally meaning softening of bones.
    - Etiology
- Rickets is a deficiency disease caused by deficiency of calcium phosphorus or Vitamin D. The deficiency of these may arise in several ways.
    - Deficiency of calcium
- *Deficiency of calcium in the diet*: Inadequate calcium in the diet may not occur in animals.
- *Increased requirements in growing animals*: if adequate quantities are not allowed in the ration deficiency may arise in growing animals in which the needs for calcium and phosphorus are great.
- *Failure of absorption of calcium*: Calcium is mostly absorbed as CaH PO4 and for this the medium must be acidic. If the intestinal contents are excessively alkaline, calcium cannot be easily absorbed.
- *Improper balance of calcium and phosphorus*: Excess of phosphorus in the ration (feeding too much of bran etc), may combine with calcium and form a relatively insoluble $Ca_3(PO_4)$ which is excreted in faces.
- *Formation of insoluble complexes*: Oxalates and phytates present in some green leaves and grains respectively may form insoluble compounds in monogastric animals and are lost in the feces.Excess of oxalic acid in leaves and excess of lactic, tartaric, and malic acids in silage bind calcium in large quantities. If feed is too coarse, then greater amounts of hippuric acid are formed from cellulose.If food is too rich in protein, acid breakdown products of proteins bind calcium. When sulphur is fed to chicks as a coccidiostat, it combines with calcium to form insoluble compounds which are lost in feces.
- *Poor utilization of calcium* may occur leading to calcium deficiency. Too rich or poor fat reduce the utilization of calcium. Reduced body movement may also reduce the utilization of calcium.
- *Steatorrhoea*: In dogs fatty acids from fats that are not assimilated may combine with calcium, forming calcium soaps which are lost in the feces. This is common in man in Coeliac disease. Since Vitamin D absorption is conditioned by absorption of fat, in steatorrhoea, Vitamin D also is not absorbed and this still further affects the absorption of calcium since vitamin D is not only necessary for calcium absorption but it also increases calcium absorption by the intestinal mucosa.
- *Renal disease*: In nephritis, phosphorus is not excreted as it should be and so accumulates in the blood and body. The excess phosphate ions are excreted through the intestinal tract, where they combine with calcium to form insoluble compound which is lost in the feces.
    - Deficiency of phosphorus
- *Inadequate amounts of phosphorus in diet*: In certain parts of the world soil is deficient in phosphorus and so animals maintained solely on the plants from such soils develop phosphorus deficiency which is clinically manifested as osteophagia (pica).
- *Increased requirements*: In growing animals, if adequate quantity of phosphorus is not allowed in the ration deficiency will result.
- *Formation of insoluble complexes*: Excess of Calcium, iron and aluminum form insoluble phosphorus compounds and so phosphorus deficiency results.

- *Change of reaction of intestinal contents*: An acid medium is required for the absorption of CaH PO_4. But if the reaction changes, absorption cannot occur.
- *Steatorrhoea*: Vitamin D is necessary for the absorption of phosphorus also. In steatorrhoea vitamin D is not absorbed and so is not available to the body and so phosphorus is also not absorbed.
    - Deficiency of vitamin D
- Insufficiency of vitamins D may occur in young animals due to the following causes
    - *Deficiency of sunlight***:** Since vitamin D can be formed in the skin by the action of ultraviolet rays on ergosterol, deficiency of sunlight may cause deficiency of vitamin D. Smoke and smog in industrial places filter the ultraviolet rays and so rickets may supervene.
    - *Diseases of liver***:** If sufficient bile is not secreted, absorption of vitamin D may be interfered with.
    - *Steatorrhoea***:** Vitamin D is not absorbed in the absence of fat.
    - Pathogenesis
- The essential defects in rickets are
    - The cartilage cells are resistant to degeneration and are not calcified.
    - The blood vessels fail to invade and corrode the cartilage.
    - On the persistent and growing cartilage, there is overgrowth of the osteoid.
    - The osteoid is not calcified.
    - In the osteochondral zone, fibrous tissue proliferates.
    - Clinical signs
- Stunted growth - shortening of bones results in rachitic dwarfism.
- Kyphosis and scoliosis
- “pot-bellied” appearance of the abdomen
- On pressure and due to weight the bones bend - bowing of the limbs (“bow legs”)
- Bending of knee and fetlocks
- Enlargement of ends of bones and joints
- Over-extension of pasterns with overgrowth of hooves
- Cranium is more dome shaped and the fontanels are wide
- Jaws cannot be closed
- Teeth may be poorly formed and irregular
- “Rickety-rosary” (enlarged chondrocostal joints appearing as a string of beads)
- Deformity of the pelvic bones (which later may cause material dystocia)
- Crooked sternum in birds.
    - Clinical pathology
- There may be decrease in serum calcium and phosphorus and increase in alkaline phosphatase.
    -
    - Gross pathology
- The epiphysis cartilages are abnormal wider and soft and so can easily be cut. The osteochondral zone is softer than normal.
    - Histopathology
- The epithyseal cartilage cells persist and grow and so the zone of cartilage is wider and longer. Therefore chondrocostal and osteochondral junction are widened and enlarged.
- In the osteochondral junction irregular tongues of surving and resistant cartilage cells appear to be arranged in a disorderly and crooked manner.
- Osteoid which is pink staining is abundant while the blue staining bony trabeculae are few and widely separated.

- Overgrowth of fibrous tissue occurs at the osteochondral zone and in the marrow. There is therefore reduction of myeloid cells.
    - Diagnosis
- X ray shows enlargement of epiphyseal plate, enlargement of epiphyseal line and bending bones.
    - Sequelae: Restitution of the deficiencies corrects the disorder. But deformities persist e.g. "bowlegs"

OSTEOMALACIA

- Synonym: This condition is otherwise known as adult rickets.
- Occurrence

- It occurs in animals in which endochondral ossification has ceased.
- Osteomalacia may be seen in pregnancy when maternal calcium is drained to the fetus and in high yielding cows in which large amounts of calcium are excreted through the milk.
    - Etiology: The causes for osteomalacia are similar to those of rickets.
    - Pathogenesis
- Because of either excessive demands of the body for minerals or deficient in the dietary intake of the minerals to meet the demands of the mineral requirement by the body bone resorption occurs.
    - Clinical signs
- Kyphosis and lordosis are frequently seen together with narrowed pelvis.
- Because of lack of mineralization the skeleton becomes soft and fragile and so fractures and deformities occur.
- The articular heads of some of the bones may sometimes separate.
    - Histopathology
- Presence of excess of osteoid
- Failure of calcification of matrix (atrophy of the bone substance) and.
- The medullary cavity is enlarged.
- Compact bone becomes spongy in extreme cases and ultimately a membranous sac covered over by the periosteum containing traces of bone is left.
- Active resorption of bone by osteoclasts
- Reduction in size and number of the trabeculae of spongiosa

OSTEODYSTROPHIA FIBROSA

- Synonym
- In the horses, osteodystrophia fibrosa is known as "Bran disease" or Big head" or "Miller's disease"
    - Occurrence
- Frequently occurs in animals.
- It is common among horses maintained by millers.
    - Etiology
- Feeding of animals with bran alone.
- If the calcium phosphorus ratio in feed is reversed or becomes 1:3 or wider, osteodystrophia fibrosa results.
    - Pathogenesis
- Since bran is a cheap by-product in the milling of wheat, horses of the millers are maintained exclusively on bran which has high phosphorus content.
- This phosphorus combines with calcium of the food and forms insoluble Ca_3PO_4 in the intestine and is excreted in the feces.
- Therefore sufficient amount of calcium and phosphorus are not available to the body and so hypocalcemia results.

- This in turn causes osteomalacia.
- Hypocalcemia stimulates the parathyroid which becomes hypertrophic producing excess of parathormone.
- This hormone acting on the bone (through the osteoclasts) decalcifies it producing osteomalacia.
- The bones become soft as calcium is withdrawn from them.
- So as to strengthen the bones, fibrous tissue proliferation occurs.
- Since soft bones bend and twist, irritation is produced and this causes inflammation to occur which ultimately is responsible for fibrosis.
- This fibrosis is most apparent under the periosteum hence the bone appears larger than normal.
- The bone marrow may also be replaced by the fibrous tissue.
    - Clinical signs
- Initially
    - abnormal gait - stiffness
    - shifting lameness
- These symptoms are later followed by
    - Anorexia
    - Anemia (the anemia is myelophthisic in origin – due to swelling of the jaws)
    - Cachexia.
    - Dyspnoea due to narrowing of the nasal passages
    - Difficulty in mastication
    - Loosening and loss of teeth
    - Fractures (common in occurrence)
    - Gross pathology
- All bones are not equally affected. The bones that are most active are affected. This affection is first noticed in the facial bones.
- The facial bones of the horse appear swollen and hence the name “Big Head”.
    - Histopathology
- Microscopically,
    - large masses of fibrous tissue are seen in which are found remnants of bony trabeculae.
    - sometimes the fibrous tissue may be of such proportion as to resemble a fibroma.

OSTEITS FIBROSA CYSTICA

    - Synonym: Von Recklinghausen’s disease
    - Definition
- This condition is a form of osteodystrophia fibrosa characterized by decalcification of bone, substitution by fibrous tissue and formation of cysts.
    - Etiology
- This condition is caused by hyperparathyroidism.
- Hyperparathyroidism may be**:**
    - Primary: As in a tumor of the Parathyroids.
    - Secondary
        - Dietary calcium insufficiency
        - Dietary phosphorus insufficiency
        - Chronic renal disease.
    - Pathogenesis
- The parathyroids are sensitive to blood calcium level.

- Any decrease in blood calcium stimulates the parathryoids and hyperplasia occurs with increased production of parathyroid hormone.
- Normally glomeruli filter phosphates some of which are reabsorbed by the tubules. But in renal disease the phosphate excretion is much reduced and so the phosphate level of the blood raises – hyperphosphatemia. To compensate for the rise and to keep the Ca: P ratio constant, calcium is withdrawn from the bones.
- Besides the retained phosphate is excreted through the bowel where it combines with calcium and forms an insoluble compound and so is lost from the body resulting in hyocalcemia. Which stimulates parathyroid liberating excess of hormone and this withdraws calcium from the bone through the mediation of osteoclasts to maintain normal blood calcium level.
- The function of parathyroids is therefore homeostasis to maintain the optimum Ca: P level in the blood.
- The activity of parathyroids is increased two folds to facilitate excretion of phosphorus in the urine.
- When calcium is removed from the bones, they become soft and weak and so to strengthen them, there is fibrous tissue proliferation. This change though found in all the bones, it is more prominent in the bones of the head.
    - Gross pathology
- In the dog, the lower jaw becomes so soft that it is as pliable as rubber. " Rubber –jaw syndrome ".
- Since the newly formed connective tissue is poorly supplied with blood. Degeneration, softening and cyst formation occur – hence the name osteitis fibrosa cystica .
    - Histopathology
    - Osteoid and fibrous tissues are more.
    - Attempts to form a new bone in some places is evident by the presence of ostoblastic activity.
    - In other places, osteoclasts are seen nibbling away spicules of bone.
    - Cysts of varying sizes and hemorrhages are seen.

OSTEOPOROSIS

- Definition
- In this disorder, there is reduction in the bony matrix. But what is present is fully mineralized (whereas in rickets and osteomalacia tissue matrix is formed but inadequately mineralized).
    - Etiology
- Senility: Due to decreased osteoblastic activity or decreased sex hormones.
- Local pressure on bones may cause atrophy: for example tumors, *Coenurus cerebralis*, hydatid cysts and pulsating aterial aneurysms in contact with vertebra.
- Lack of protein as in loss of protein (renal disorder), lack of production as in liver disease or defective absorption due to intestinal disorders. Protein is essential for the formation of osteoid, without which bone cannot be formed.
- Deficiency of vitamin A leads to decreased production of osteoblasts
- Deficiency of Vitamin C: Osteoblasts and osteoid are not formed.
- Disuse: for proper healthy condition of the bone to be maintained exercise is necessary. If a part is immobilized for a long time, bone of that part becomes thinner and porous due to increased activity of the osteoclasts and inactivity of osteoblast (due to lack of normal stimulus of stresses and strains).
- Loss of nerve supply to the part results in paralysis and so the part cannot be moved and osteoporosis will result.

- Deficiency of trace elements : - copper deficiency in dogs; manganese deficiency in pigs and zinc deficiency on fowls.
- Hyperthyroidism: Osteoclastic activity is probably increased.
- Hyperparathyroidism: Increased resorption of bone occurs.
- Cushing's syndrome: Excess of glucocorticoids probably suppresses the osteoblastic activity. Bodies of vertebrae are severally affected.
- Lack of either androgens or estrogens: In human pathology, osteoporosis is frequently observed after menopause. The sex hormones appear to have some, influence over the osteoblastic activity.
- Poisons – For example lead poisoning in sheep and goats causes osteoporosis.
    - Pathogenesis: In this condition, destructive processes exceed the productive in remodeling of bone.
    - Clinical pathology: The blood levels of calcium and phosphorus are normal.
    -
    - Gross pathology
- The bones appear lighter and thinner – atrophied.
- The cortex is thinner but the marrow cavity is wider.
- The bones become porous, brittle and so are prone to fractures.
    - Histopathology
- The bony trabecula is thinner with decreased number of osteoblasts.
- Osteoclastic activity denotes destruction of bone.
    - Diagnosis: Osteoporosis can be diagnosed by lzuka's test.
    - **PULMONARY OSTEOARTHROPATHY**
    - Synonym: This condition is known as Marie's disease in humans
    - Occurrence: This is found in all animals but with greater incidence in dogs.
    - Etiology
- This disease is noticed in the following conditions:
    - Chronic disease of heart and lungs – congenital heart disease, bronchiogenic carcinoma bronchiectasis, emphysema, chronic tuberculosis and neoplastic condition of the lungs
    - When there is interference in the vascular supply to the extremities.
    - Passive congestion of the affected parts.
    - *Spirocerca lupi* infection.
    - *Dirofilaria* infection
- It is surmised that anoxia, probably with some obscure toxins is the causative factor.
- The skeletal changes are the result of reflex vasomotor disturbances in limbs secondary to circulatory disturbances in the lungs.
    -
    -
    - Gross pathology
- There is formation of new bone mostly under the periosteum of bones of limbs, which is therefore pushed out (Perisoteal hyperostosis).
- As the osteophytic (Osteophyte means a boney excrescence) formation is not even, the bony surface is rough.
- The articular surfaces are free.
- Joints may be swollen due to periarticular proliferation.
- In the lung, foci of new bone formation are seen.
    - **OSTEITIS AND OSTEOMYELITIS**
- Inflammation of the bone is called osteitis
- Inflammation of periosteum is periostitis.

- Inflammation of bone marrow is known as osteomyelitis.
- Inflammation of vertebrae is spondylitis .
- Osteitis and osteomyelitis may be acute or chronic.
    - Acute purulent osteomyelitis
- *Occurrence*: This condition is not so frequent in animals as in man.
- *Etiology*
    - Acute purulent osteomyelitis is always caused by bacteria which gain entry into the bone in the following ways.
        - Direct
            - Through compound fractures
            - Gunshot and other wounds
        - By lymph vessels in draining neighboring purulent areas such as:
            - Purulent arthritis
            - Purulent periostitis
            - Gathered –nail wound
            - Suppurative otitis media
        - By blood stream: From a suppurative lesion elsewhere and in pyemia.
    - The organisms that cause it are pyogenic bacteria, *Spherophorus necrophorus, Erysipelothrix rhusiopathiae, Salmonella* and *Cryptococcus neoformans*.
- *Acute periostitis*
    - Acute periostitis may be non-suppurative and is usually caused by trauma (concussion).
    - It is seen in horses as " sore shins " (due to working on hard roads).
- *Gross pathology*
    - The periosteum shows hyperemia with purulent exudates accumulating between the cortex and periosteum.
    - The exudates may separate the periosteum from the bone and necrosis of the cortex results.
    - Periosteum may be ruptured, liberating the pus into the nearby tissue.
    - Since periosteum is in continuity with the endosteum and medulla, pus may pass on to these structures.
    - Then, necrosis of the bone occurs due to separation of both periosteum, and endosteum on which the nutrition of the bone depends.
    - Suppurative osteomyelitis
- In suppurative osteomyelitis pus is found in the medullary cavity and it may burst through the cortex. But more often, such drainage is difficult and the condition progresses to chronic stage.
- In the young growing animals abscesses are found at the chondrocostal joints and in the epiphyseal plates.
- The necrosed bone is separated from the healthy bone by the action of osteoclasts and a sequestrum is formed. Osteoblasts nearby are active and produce new bone which forms a case as it were around the sequestrum and this is known as involucrum. Pus is discharged to the outside from the sequestrum through small openings in the involucrum called cloacae .
- *Sequelae*
    - Resolution and healing with timely treatment.
    - Pathological fracture due to extensive destruction of the bone.
    - Suppurative arthritis may occur due to extension of infection to the neighbouring joint; metastatic abscesses.

- If suppurative osteomyelitis is extensive and present for a long time amyloid degeneration may occur.
- Chronic osteomyelitis
- Death due to pyemia and septicemia.
- Chronic osteomyelits

- *Etiology*
  - It may be a sequel to osteomyelitis.
  - Repeated injury or concussion especially in horses
  - Bacteria of low virulence – *Actinomyces, Brucella, Mycobacterium tuberculosis, Salmonellosis*
  - Fungi – Coccidioidomyocosis.
- *Gross pathology*
  - In the case of chronic trauma and concussion, exostosis will occur.
  - Exostosis is the formation of granulation tissue of the bone, just as fibrosis occurs in chronic inflammation of soft tissues
  - So in the bone, chronic inflammation results in the formation of new bone. This is essentially a result of chronic ossifying periostitis.
    - In the horse special names are given to exostoses occurring in certain locations.
    - Ring bone: This is the exostosis found on the 1st or 2nd phalanx. This is a painful condition causing lameness.
    - Splint: This is exostosis at the end of maetacarpal or metatarsal bones. It is not usually painful and so no lameness seen
    - Spavin: This is exostosis on the medial portion of the distal tarsal bones. This causes lameness as the bony growth pinches the cunean tendon, which passes over it.
- *Histopathology*
  - The exostoses or osteophytes have the structure of a compact bone but do not have haversian system.
  - The lesions produced by bacteria in chronic osteomyelitis are granulomas.
  - In chronic osteomyelitis, centre of pus are surrounded by granulation tissue and inflammatory cells, consisting mostly of mononuclears and a few giant cells.
  - Due to activity of the osteogenic layer of the periosteum new bone is formed and so the shaft is thickened and marrow narrowed- osteosclerosis .
  - In actinomycosis and tuberculosis there is rarefaction of bone – rarefying osteitis. In tuberculosis, there is extensive destruction of bone with the formation of caseous material but new bone is not formed.
  -
  -
  - **FRACTURE**
  - **Definition** : A fracture is a break in the continuity of a bone and is usually due to trauma.
  - **Varieties of the fractures**

Simple fracture	Fracture of bone without an opening over the overlying skin.

○ Compound fracture	○ Fracture with an opening on overlying skin.
○ Greenstick fracture	○ Here one side of the bone is broken while the other is intact as occurs when a green stick is bent.
○ Impacted fracture	○ When one fragment of a fractured bone is firmly driven into the other.
○ Transverse fracture	○ Fracture at right angles to the axis of the bone
○ Oblique fracture	○ Break extends in an oblique direction.
○ Linear fracture	○ Here bone is split lengthwise.
○ Multiple fractures	○ Here are two or more lines of fracture of the same bone but not communicating with each other.
○ Comminuted fracture	○ The bone is splintered into many pieces.
○ Pathological fracture	○ The fracture is not due to trauma only but due to some bone disease existing. e.g. Osteosarcoma.
○ Arti	○ When joint surface of a bone is involved.

cular fracture	
○ Depressed fracture	○ In the skull where the involved bone is depressed below the surface.

- Sequelae

- *Healing of fracture*
  - Along with fracture of bone, there is hemorrhage as the blood vessels nearby are torn and ruptured. The capillaries of the haversian canals also contribute to the hemorrhage.
  - Because of ischemia (due to cessation of local circulation) bone cells die and these incite and inflammatory reaction.
  - The accumulated blood clots
  - In twenty four hours, this clot is invaded by fibroblasts and capillaries from the periosteum and is organized. This fibrovascular tissue is strong enough to keep the two broken ends together and is known as a soft tissue callus . (Callus, Latin for a hard substance).
  - Osteoblasts derived mostly from the deeper layer of the periosteum invade the blood clot along with the capillaries and within 4 or 5 days trabeculae are formed around central spaces which become Haversian canals. This is the osteoid laid down by the osteoblasts.
  - This osteoid is well formed by the end of second week.
  - Osteoblasts are also formed by metaplasia of the fibrous tissue.
  - Later calcium salts are deposited on the osteoid to form bone.
  - The newly formed bony tissue unites the two ends of the fracture bone and is known as provisional callus.
  - The callus formed by the periosteum and located sub-periosteally is called external callus , that present in the medullary region is called internal callus and that between the ends of the shaft the intermediate callus or in-line callus .
  - The callus formed is larger than the outline of the bone and so bulges on the periosteal side.
  - In the beginning there is no orderly arrangement of the trabeculae and haversian systems.
  - Later the provisional callus is removed by osteoclasts and remodeled by osteoblasts into regular bone. This is called definitive callus .
  - It may take several months for this definitive or hard callus to form.
  - Finally during the remodeling processes, excess of the callus is removed.
  - If the gap between the two ends of a broken bone is too wide, the fibroblasts of the provisional callus may become cartilage cells by metaplasia and this is later converted into bone – endochondral ossification.
  - Factors that interfere with healing
- Non-alignment of the two ends of the bone: Due to this deformity, excessive callus formation and displacement of bone may occur.

- Inadequate immobilization: A false joint or pseudoarthrosis may occur if the fractured ends are not firmly immobilized. The provisional callus is not sufficiently mineralized and so permits bending at the fracture area.
- Fragments of necrotic bone: This is more common in comminuted fractures where the necrotic bone acts as a foreign body producing inflammation and prevents healing.
- Presence of foreign bodies hinders normal and rapid healing. These may be bullets, muscle, fat or clothing.
- Infection: This is common in compound fracture, leading to necrosis and osteomyelitis, which retard the process of healing.
- Senility: In older animals healing is slow due to decreased vascularity and retarded metabolic processes.
- Deficiency of calcium, phosphorus, vitamin D and proteins: These may occur in dietary deficiency, starvation, metabolic or infectious diseases e.g. renal diseases, malabsorption diseases due to gastro-intestinal pathology, parathyroid disorders and excessive loss of protein as in albuminuria or heavy stomach worm infections.
- Pathological: Presence of osteodystrophy or neoplasms prevents healing of fractures.
    - **TUMORS**
- Primary tumours of bone include fibroma, myxoma, lipoma, chondroma, osteoma, chondrosarcoma, fibrosarcoma, osteogenic sarcoma, and giant cell tumor.
- Secondary tumours of bone are metastatic carcinoma and sarcoma from other parts of the body.
    - **PATHOLOGY OF JOINTS**
        - **ARTHRITIS**
    - Pathology of Joints
- Normally, the young cartilage is whiter and translucent. As it ages it becomes opaque, yellowish and less and less elastic.
    - Definition: Inflammation of the joint is called arthritis.
    - Terminology
- Inflammation of hip joint is called Coxitis
- Inflammation of stifle joint is called gonitis .
    - Types
- Based on course: Arthritis may be acute or chronic.
- Based on etiology: Bacterial
    -
    -
    -
    - Gross lesions
- In case of chicken, affected with staphylococcal infection, calcium defeciency and reoviral infections, the articular cartilage of femur may be eroded.
    - Acute arthritis
- *Etiology*
    - Contusion or strain in which there is stretching of the joint capsule.
    - Bacteria: Routes of infection may be (a) via blood stream (b) by extension from neighbouring tissue and (c) by puncture wounds.
- *Gross pathology*
    - Trauma usually produces a serous type of inflammation in which there is increased production of synovia distending the joint capsule.
    - The condition is mild showing a slight hyperemia of the articular cartilage and the synovial membrane.

- **Bacterial Arthritis**

- Bacterial arthritis is classified as
  - Non-suppurative and
  - Suppurative
  - Non-suppurative arthritis
  - In non-suppurative arthritis, there is acute serous or serofibrinous exudate.
- *Etiology* : The causes are
    - *Erysipelothrix rhusiopathiae* (serous polyarthritis) in sheep and pig
    - *Hemophilus influenza suis* in pig.
- *Gross pathology*
  - The exudate contains yellowish flakes, which are often compressed into flat structures which float in the joint fluid.
  - The Synovial membrane is thickened and studded with hemorrhages.
- *Histopathology*
  - Synovial membrane: hyperemia and neutrophilic infiltration are common.
  - Articular cartilage may be eroded.
  - Suppurative arthritis
- *Etiology*
  - This condition is usually associated with Navel ill.
  - The bacteria localize in the joints because of the rich blood supply there and also probably to the weak defenses in that region.
- *Routes of infection*
  - Infection may be
    - Primary through penetrating wounds of joints.
    - Secondarily by extension of suppurative process from neighbouring lesions or metastatic lesions in pyemia.
  - The following organisms are incriminated

Animal affected	Causative bacterium	Nature of lesions
Calf	E. coli Corynebacteriumpyogenes	Purulent arthritis with destruction of joint. Organisms may be found in pure culture or mixed with other organisms Septicemia and acute arthritis Synovia is cloudy

		○ Many organisms are present in the joint.
○ Colt	○ Shigella equirulis ○ Streptococci	○ Swollen joints ○ Purulent exudate
○ Sheep	○ Staphylococci ○ Brucella abortus	○ Purulent exudates with joint destruction
○ Swine	○ Streptococci ○ Brucella abortus	○ Purulent exudate

- *Gross pathology*
  - The affected joint is swollen.
  - Sometimes, the pus in the joint may be discharged though a break in the skin resulting in an open joint .
  - Synovial fluid is purulent. White yellow green pus may be present in the joint depending on infective organism. In mycoplasma infection, pus is thin and colourless.
  - Particles of disintegrated bone are found in the pus like grins of sand.
  - The articular cartilage may be inflammed and eroded.
  - There may be inflammation of the periarticular tissue.
- *Histopathology*: there is infiltration by neutrophils.
- *Sequelae*
  - The condition has unfavorable prognosis in young animals. Due to pain, they will not be able to move about.
  - In suppurative arthritis the articular cartilage is destroyed and infection may spread to the underlying bone leading to suppurative osteomyelitis, necrosis and caries of bone.
  - If the condition becomes chronic, there is excessive fibrosis and ankylosis of the joint.
  - **Chronic Arthritis**
  - Etiology
- Chronic arthritis may be a sequel to acute arthritis.
- Primary
  - Chronic trauma to a joint
  - Bacterial: Tuberculosis in ox and pig; Fowl Cholera in fowls.
- In chronic stages of fowl cholera, *Pasteurella avium* may get localized in the joints and tendon sheaths.
  - Gross pathology
- There is an accumulation of a cloudy or cheesy material giving the joint a swollen appearance.

- Chronic serous arthritis

- Due to destruction of the articular cartilage, there may be fibrous adhesion between the articular surfaces.
- Subsequently the two bones may fuse together producing ankylosis of the joint.
  - Tuberculous arthritis
- Tuberculous arthritis is characterized by the granulomatous inflammation.
- It is manifested in 3 forms.
  - *Miliary form*
    - Here military nodules are found in the synovial membrane.
    - The neighbouring tubercles may coalesce and project into the joint cavity as 'pearls'.
    - This form is seen in pigs.
  - *Infiltrating tuberculosis* (Chronic organ tuberculosis)
    - This is seen in cattle characterized by diffuse tuberculous granulation tissue containing epithelioid cells and giant cells.
  - *Caseating tuberculosis*
    - It is characterized by synovitis with caseation but without specific granulation tissue.
  - Myocaplasmal arthritis in swine
- *Etiology*: Mycoplasma granularum is a common cause of arthritis in 100 to 200 pound swine.
- *Predisposing causes*
  - Stress
  - Heavy muscling
  - Genetical background
- *Pathogenesis*
  - The disease is usually an acute one, with sudden onset of lameness.
  - The course runs for 3 to 10 days.
  - Subsequently flare up cause longstanding chronic arthritis.
- *Gross pathology*
  - In the acute form, there is increased serosanguineous synovial fluid in the femuro-tibial, coxo-femoral, cubital or scapulohumoral joints.
  - The synovial membranes are swollen, hyperemic and discolored but the joint capsule and articular surfaces appear normal.
- *Histopathology*
  - Microscopically, in the synovial membrane, hyperplasia of synovial lining cells, villous hypertrophy and extensive mononuclear infiltration are noticed.

DEGENERATIVE ARTHROPATHY

  - Synonym: Osteoarthritis-deformans
  - Definition
- In arthropathy, no inflammation occurs initially, but is an ageing process.
- This condition should be distinguished from the conditions of joint resulting from arthritis.
  - Etiology
- The following causes are incriminated
  - Probably it is an ageing process.
  - Faulty circulation.
  - Obesity
  - Absorption of products of faulty digestion.

- Repeated trauma as in concussion sustained by working horses on hard road; sprains.
- Pathogenesis

- The cartilage being avascular depends on the synovial fluid for its nutrition. So, many changes that may occur in the synovial fluid as a result of ageing process may contribute to the degeneration of the cartilage.
- To start with, cartilage cells undergo hydropic degeneration and fatty changes.
- The fibrils of the cartilage become visible.
- Subsequently fissures form on the cartilage followed by fibrosis of the ground substance.
- The cartilage soon becomes separated and eroded, exposing the bone underheath.
- The older cartilage has lost is power of regeneration and growth and so repair does not occur.
- When ulceration of the cartilage occurs, the bone is exposed and subjected to stress and becomes sclerosed and hard (eburnation).
- Granulation tissue grows from the exposed bones of the two articular surfaces and thus fills the articular cavity. This tissue subsequently becomes ossified resulting in ankylosis. At the margin or edges of the joint are formed periostitic exostoses.
- The Synovial membrane becomes fibrous and thickened.
- The villi become thickened, fibrous and long and contain fatty tissue.
- Occasionally near the area of degeneration, some cartilage cells proliferate and form into small nodules which may be calcified following degeneration. These nodules may become detached into the articular cavity.

- **RING BONE**
  - Definition

- This is a condition of degenerative arthropathy affecting the inter-phalangeal articulation of horses, resulting in ankylosis and lameness.

  - Pathogenesis

- In this condition, the articular cartilages may be destroyed resulting in ankylosis. This is due to union of the articular ends of the bones by granulation tissue which becomes ossified. So, ankylosing arthrosis results-articular ring bone.
- More often there is chronic inflammation of the periosteum and the ligamental apparatus due to repeated concussion and this result in periarticular ring bone, in which the exostoses may fuse bridging the joint and fixing it.

- **SPAVIN**
  - Definition

- This is arthropathy of the tarsal joint affecting its dorsal and medial parts. Anklyosis may result.

  - Pathogenesis

- The condition first starts with degeneration of the cartilages of the second and third tarsal bones. Subsequently other tarsal and metatarsal bones may be involved.
- The normal white or bluish cartilage undergoes degeneration, becoming opaque and fibrous. It breaks down and ulcerates.
- Granulation tissue from the exposed bone grows and fuses with that growing from the opposite end. When this becomes ossified, the joint becomes ankylosed. No pertarticular changes may be noticed. So such a condition is known as occult spavin.
- In some cases, the synovial membrane may become thickened due to irritation.
- The fibrous layer of the articular capsule proliferates and then becomes ossified, resulting in large exostoses, which can be easily seen on the internal and medial aspects of the hock joint.

- These exostoses pinch the cunean tendon and so pain may be caused, resulting in lameness.

ANKLYOSING SPONCYLOSIS

- Definition: In this condition, the small vertebral articulations become ankylosed.
- Pathogenesis

- In the dog it is due to the protrusion of the nucleus pulposus irritates the periosteum and the ventral spinal ligament resulting in exostoses which may subsequently fuse and join the vertebrae.
- In bulls also this condition is met with in those that are used for stud. Due to frequent trauma attendant on their work, there is constant irritation. The lumbosacral region is more often affected causing paralysis or ataxia.

PROTRUSION OF INTER - VERTEBRAL DISCS

- Animals affected: This condition is met with in man and dog
- Anatomical features

- Normally, the Intervertebral disc consists of a central nucleus pulposus which is semisolid mucoid connective tissue. This is enclosed in a thick fibrous covering, the annulus fibrosus.
  - Pathogenesis
- Due to violent trauma and degenerative changes in senility, there may be a rupture in the annulus, from which the nucleus pulposus escapes and becomes displaced.
- The susceptibility of the disc to degeneration is inherited.
- Usually two forms of displacement occur:
  - dorso-lateral prolapse of the nucleus pulposus into the spinal canal.
  - ventral prolapse beneath the spinal ligaments. In this variety due to formation of osteophytes ankylosing spondylosis results.
- In the chondrodystrophic breeds (Dachshunds, Pekingese, French bull dogs) at a very early age, the nucleus pulposus become cartilaginous, which later becomes degenerated and calified.
- So the nucleus puposus, which is normally a gel and so able to withstand shocks and transmits pressures uniformly to the annulus fibrosus, becomes transformed into a cheesy mass which crumbles easily.
- This material transmits pressure to localized portions of the annulus, which also undergoes degeneration. Its lamellae become hyalinised and later split.
- The protrusion of the disc may occur at any level, but occurs more frequently in the lumbar region or in the posterior thoracic region.
- The displayed nucleus puposus presses upon the spinal cord producing nervous lesions.
- Pressure on the spinal cord may produce hemorrhage and necrosis in the involved area.
- Wallerian degeneration of the nerves may be noticed in the spinal nerves arising from the affected region as well as demyelination of nerve tracts.
- In other breeds, the above changes occur in mid or later life.
  - Clinical signs
- Violent reaction to stimuli-spastic type; Pain with exaggerated reflex movements which may be intermittent or occur over long or short periods.
- Rapid progressive complete or partial paralysis of the posterior region may be noticed.

- Early death due to respiratory failure.

BURSITIS

- Definition: Inflammation of the bursa over the joint is bursitis.
- Occurrence

- This is of frequent occurrence in animals.
- Examples:
- Hygroma of the carpal joint in cows
- Capped elbow or hock joints of horses.
  - Etiology
- Over-use
- Trauma, especially if repeated.
- Infection (Brucella infection in cows produces hygroma and in horses " fistulous withers" and " poll evil ")
  -
  - Gross pathology
- The inflammation may be serous, serofibrinous or purulent.
- Trauma produces serous type - one example is the serous bursitis of hock joint in the horses. This is called Bog spavin .
- Here the joint is filled with serous fluid.

POLE EVIL

- It is the inflammation of the bursa between ligamentum nuchae and atlas and axis.

FISTULOUS WITHERS

- Definition: It is the affection of bursa between the ligamentum nuchae and the thoracic spines.
- Etiology

- Causes may be traumatic, parasitic (*Onchocerca cervicalis*) or *Brucella abortus* and *Actinomyces bovis*.
  - Pathogenesis
- The suppurative and granulomatous reaction is attributed to the two organisms and the infection occurs hematogenously.
  - Gross pathology
- The inflammation is a suppurative granulomatous one in which fistulae open on the surface of the skin.

NAVICULAR DISEASE

- Definition: This is bursitis and arthritis involving the distal sesamoid or navicular bone in the horse.
- Clinical signs: Usually the fore limb is affected.
- Pathogenesis

- First there is serous inflammation of the lining membrane of the podotrochlear bursa, with hyperemia.
- This is followed by erosion and ulceration of the articular cartilage, over which the flexor tendon passes.
- Due to the changes in the cartilage, the tendrils of the tendon become frayed and ultimately rupture of the tendon may occur.
- Later the bone is inflammed, becomes rarefied and may fracture.

INFECTIOUS SYNOVITIS OF CHICKEN

- Introduction: This is a chronic disease of chicks.
- Etiology: *Mycoplasma syoviae* and *Mycoplasma gallisepticum*.
- Pathogenesis

- Infection occurs in chicks, 12-14 weeks of age, by ingestion and the incubation period is 24 to 80 days.
    - Clinical signs
- Morbidity and mortality are low.
- Emaciation
- Retarded growth
- Pale comb
- Distended hock joints
- Swollen foot pads
- Lameness.
    - Gross pathology
- In the early stages, creamy exudate is found in the synovial membrane of the joints, especially those of the hock and foot.
- This material becomes caseous as the disease progresses.
- The surface of the affected joints becomes yellow or orange.
- In the early septicemic stage, the spleen, liver and the kidneys may be swollen.
    - Histopathology
- The following changes are noticed microscopically
    - The characteristic lesion is a purulent synovitis of the leg joints.
    - In the liver and spleen there is proliferation of the reticular cells of the reticulo endothelial system.
    - Bile duct proliferation
    - Fibrinous pericarditis
    - Sometimes a focal infiltration by mononuclears necrosis of the myocardium
    - Thymus and bursa of Fabricius may be atrophied due to degeneration of lymphoid tissue.
    - Brain may show gliosis and degeneration of Purkinje cells.
    - Sequelae: With timely treatment using antibiotics the disease can be cured.
    -
    - **MUSCULAR SYSTEM**
        - **DISEASES OF SKELETAL MUSCLES**
    - **Atrophy of skeletal muscle**
    - Etiology
- *Senility*
    - In old age there is gradual atrophy of all muscles.
    - In dogs, milch cows, horses and ewes which are allowed to grow old, atrophy may be observed.
    - It is likely that there may be under nutrition or the animal is not able to metabolise available nutriments in old age.
    - The muscle cells are not able to assimilate the nutrients and so catabolism exceeds anabolism.
- *Disuse*
    - This is seen in fractures of bones when the parts are immobilized for long periods and the muscles therefore are not utilised.
    - Disuse of the limb may occur due to pain as in rupture of a tendon, acute arthritis, ankylosis and disease of the bones and muscles.
- *Starvation*
    - Here sufficient food is not available to make up for the catabolism that takes place.

- *Atrophy of wasting diseases, cachexia and malnutrition*
    - In chronic wasting diseases like tuberculosis and Johne's disease; in debilitating conditions like neoplasia; in cachexia and in malnutrition, the food digested is either not effectively metabolized or is not used by the body and so atrophy results.
- *Pressure*
    - Continous pressure on the muscle, producing ischemia locally and interfering with movement will cause atrophy.
    - The cause of pressure may be tumors, abscesses, cysts, ill fitting collars and saddles as well as infiltration of lymphoid cells in neoplasm of these cells.
- *Denervation*
    - When a nerve is injured or severed, the muscles supplied by it become paralysed and atrophied
    - Examples
        - Atrophy of laryngeal muscles when the recurrent laryngeal nerve is injured (Roaring)
        - Atrophy of supraspinatus muscles when the supracapular nerve is injured.
        - Atrophy of muscles in lesions of the central nervous systems - poliomyelitis, protrusion of intervertebral disc, tumors etc.
    - Gross pathology
- The muscle which is normally pink, loses this color and turns pale, grey or brown.
- It is firmer due to replacement by fibrous tissue.
- Due to uneven atrophy of different muscles, disfigurement may occur.
- Skeleton becomes prominent.
    - Histopathology
- The size of the muscle fibres is reduced.
- Sarcoplasm may become so reduced and in some places may even disappear, that the sarcolemmal nuclei become prominent.
- There may be deposition of "wear and tear" pigments at the poles of nuclei giving the muscle a brown color-brown atrophy
- The cell nuclei may proliferate and fill the empty sheath.
- The etiological factors for atrophy also cause degenerative and necrotic changes like cloudy swelling, fatty degeneration and coagulative necrosis.
- In later stages there may be infiltration of fat in some areas (atrophia lipomatosa) and fibrosis in others.
    - **LOSS OF MYOGLOBIN**
- Skeletal muscles may lack myoglobin in some areas as in case of Heat stroke in chicken. COOKED UP APPEARANCE.
    - **MYOSITIS**
    - Definition: Inflammation of the muscle is called myositis.
    - Types: This may be acute or chronic.
    - Etiology
- The routes of infection and causes are
    - Trauma
    - By direct extension from lesions of neighbouring arthritis, osteitis or periostitis
    - In pyemia, hematogenously
    - By parasitic infection.

- **ACUTE MYOSITIS**
  - Acute non-suppurative myositis

- *Etiology*: Best examples of acute non suppurative myositis is black quarter is cattle and sheep.
- *Gross pathology*
  - In this condition, the organisms, *Clostridium chauveoi* causes inflammation and necrosis of the muscles with production of gas.
  - Local hemorrhage is present and the area is black due to formation of black iron suphide.
  - Regional lymph nodes are acutely congested
  - Serous cavities contain blood stained serous fluid.
- *Histopathology*
  - There is necrosis of the muscle, infiltration by neutrophils and clumps of the anaerobe.
  - The muscle fibres are torn by the gas bubbles.
  - Parenchymatous organs show fatty changes.
  -
  - Suppurative myositis
- *Etiology*
  - Infections may occur in lacerating and penetrating wounds or by extension from adjoining areas.
  - Haematogenous infection may occur from other foci as in strangles and Glanders.
- *Gross pathology*
  - The usual changes of suppurative inflammation are found, viz, abscess or phlegmon.
- *Histopathology*
  - Microscopical appearances are typical of any other suppurative inflammation with a great outpouring of neutrophils.
  - The muscle fibres undergo liquefaction following coagulative necrosis.
- *Sequelae*
  - As there is loss of muscle tissue, healing is by means of fibrous tissue proliferation and scar formation.
  - If severe, septicemia may result.
  - **CHRONIC MYOSITIS**
    - Etiology
- Actinomycosis
- Actinobacillosis
  - Gross pathology : The muscles of the tongue, cheek and throat are affected.
  - Histopathology
- The lesion consists of chronic suppurative myositis in which the “Sulphur granules” are noticed in a mass of inflammatory granulation tissue.
- There is infiltration by large number of lymphocytes, neutrophils and plasma cells.

- Muscle fibres are destroyed.
    - **PARASITIC MYOSITIS**
    - The following parasites are found to infect the muscles of animals.
- Toxoplasma
- Trichinella spiralis
    - This is found in man, pig and other animals.
    - The larvae are encysted in many muscles, especially those of diaphragm, intercostals muscles and tongue.
    - The cysts are parallel to the muscle fibres which undergo granular degeneration of the sarcoplasm.
    - Intense infiltration by eosinophils, plasma cells, histiocytes and lymphocytes occurs
    - Sarcolemmal nuclei proliferate
    - The encysted larvae may be alive for as long as 20 years.
- Sarosporidiosis
    - The sarcosporidia are present in the skeletal and cardiac muscle of all species of animals.
    - No specific disease has been attributed to these parasities.
    - Though light infections cause no perceptible symptoms, heavy infections may be responsible for lameness, weakness, paralysis, emaciation and sometimes even death.
    - Parasitised muscle fibres are destroyed by the parasite and the adjacent cells undergo pressure atrophy.
    -
- Cysticercus (Measles)
    - Cysticercus cellulosae
        - The bladder worm of *Taenia solium,* a tapeworm of man infects the muscles of pig.
        - The muscles of the shoulder, neck, diaphragm, tongue, intercostals, abdominal and cardiac muscles are affected.
        - Heavy infection may result in fatal anemia and cachexia.
    - Cysticercus bovis
        - It is the intermediate stage of tapeworm *Taenia saginata* of man, found in the muscles of cat.
        - All muscles may be affected but those of tongue, mastication and heart are more often infected.
    - Cysticercus ovis is the intermediate stage of dog tape worm *Taenia ovis* and is found in the muscles of sheep.
    - **DISEASES OF UNKNOWN ORIGIN**
    - **Eosinophilic myositis**
    - Etiology
- Cause: Unknown.
- Some kind of allergy
- Vitamin E deficiency.
- *Cattle and Sheep*
    - Very rarely, at slaughter, yellowish green areas may be noticed in the lingual, oesophageal, cardiac and diaphragmatic muscles of cattle and sheep.

- The green color fades on exposure to light
- Microscopically, large numbers of eosinophils, histiocytes, plasma cells and lymphocytes are found between the muscle fibres and in tissue spaces.
- Though extensive degeneration of the muscles may not be noticed, in some places necrosis and invasion of muscle fibres by the eosinophils are observed.
- In more chronic cases fibrosis is evident.
- There is some suspicion that the condition may be a manifestation of allergy and sarcosporidia are suspected as the sensitizing factor.

- *Dogs*
  - German shepherds and Alsatians are more often affected.
  - Clinically, the condition involves the masseter, temporal and pterygoid muscles chiefly.
  - Other muscles may be affected.
  - The muscles of mastication are enlarged bilaterally so that opening of the mouth is painful and mastication is interfered with.
  - The eyes bulge out, resulting in keratitis and corneal ulceration, since eyelids cannot close completely.
  - The local tonsillar and mandibular lymph nodes are also swollen.
  - Blood picture reveals high eosinophilic count up to as much as 90%.
  - Temporary remissions may occur but are followed by repeated attacks and the animal finally dies.
  - As the animals cannot eat, they die finally of inanition.
  - Gross pathology
- The affected muscles are swollen, hard to the touch, show grey and red streaks and white and yellow spots.
- Hemorrhage is present.
- The regional lymph nodes are congested and swollen.
  - Histopathology
- There is heavy infiltration of eosinophils, lymphocytes, plasma cells and macrophages into the muscle, producing atrophy, and hyaline necrosis, vacuolar degeneration and lysis of muscle fibres.
- Hemorrhage is common.
- The necrotic muscle is removed by macrophages and fibrosis follows.
- In the liver periportal lymphocytic infiltration is found.

WHITE MUSCLE DISEASE

- Synonym: Stiff-lamb disease
- Definition: This is coagulative necrosis of the muscles due to various causes.
- Animals affected: occurs in calves and lambs and can be produced in rabbits and guinea-pigs.
- Etiology

- *Vitamin E deficiency:* Vitamin E is an antioxidant and in its absence, oxidation in the muscles is increased to 400 times the normal and so degeneration and necrosis occur
- Vitamin E deficiency may occur in the following manner.
  - Dietetic deficiency.
  - Feeding too much of cod liver oil. The unsaturated fatty acids in the cod liver oil antagonize Vitamin E.
- *Selenium deficiency:* Selenium is required in minute quantities. In its absence muscle necrosis occurs.
- Selenium deficiency can occur in the following manner.

    - Deficiency in the soil; animals that are grazed on fodder grown on soils deficient in selenium suffer from the disease.
    - Excess of sulphur used in fertilizers inhibits the uptake of selenium by plants.
- *Vitamin B deficiency:* It is found that Thiamine deficiency produces cardiac necrosis. Deficiency can occur in animals when the ruminal flora is not active to synthesize the vitamin as occurs in cobalt deficiency.
- *Abnormal ruminal fermentation:* Some toxic products produced in the rumen cause muscle necrosis.
- *Deficiency of choline* produces muscle necrosis in rabbits (experimentally)
- *Vitamin A deficiency:* Vitamin A deficiency produces this disease in swine.
- *Multiple deficiencies:* In starvation as occurs during drought and malnutrition, multiple deficiencies of vitamins and minerals (phosphorous) may occur and muscle necrosis may be encountered .Similar lesions are seen in hypothyroidism.
    - Types
    - The clinical picture is classified into three main types.
- *The stiff type*
    - The head is carried low and has a dropping posture.
    - Animal experience difficulty in rising and walking. While walking, the gait is stiff.
    - The weight-bearing and active muscles, for example, muscles of the croup and quarters, diaphragm, heart and intercostals are mostly affected.
    - In lambs, this is the form encountered and the animals are always recumbent and do not like to move. On forcible movement, they have stiff gait and wobble. (Stiff lamb disease).
- *The respiratory type*
    - Here the muscles of respiration (diaphragm and intercostal muscles) are affected and the animal may show symptoms of respiratory distress.
    -
    -
- *The cardiac type*
    - In this type, animals show considerable weakness, inability to stand, rapid pulse and low blood pressure.
    - Since the heart is affected and weakened, exertion brings on respiratory distress and even death in animals with cardiac involvement alone
    - Sudden death occurs without any other symptoms.
    - Clinical pathology
- Clinically the serum glutamic oxaloacetic transaminase (SGOT) level will be more than 300 units while the normal is less than 100 units.
    - Gross pathology
- Those muscles which are continuously active, viz. diaphragm and intercostal muscles show the changes.
- The whole muscle bundle may not be affected but only a part of it will show the change.
- The muscles are bilaterally affected and are pale like fish flesh.
- The paleness is due to loss of myoglobin which is excreted in the urine. The change in color is also due to changes in optical characteristics of the muscle protein when it becomes coagulated.
- The muscles become hard and wooden
- Pneumonia, edema, hydrothorax, C.V.C of liver and hydropericardium will be seen when heart is involved.

- Heart shows yellowish or grey streaks or patches and the left ventricle is more often affected.
    - Histopathology
- The muscles are swollen with loss of striation and with widespread hyaline degeneration. This progresses to coagulative necrosis.
- Fibres are fragmented and may completely disappear.
- Marked sarcolemmal prolifearation is present.
- Some fibres may be calcified
- Infiltration by macrophages and lymphocytes is seen.
- Similar lesions may be found in the heart.
- In some places healing by fibrous tissue is evident.
- One noteworthy feature is that the nerves and C.N.S are normal without showing lesions.

AZOTURIA

- Synonym: Equine myoglobinuria; Monday-morning sickness; Paralytica hemogloburia
- Definition: Azoturia literally means "nitrogen in the urine."
- Clinical signs

- Azoturia is found to occur suddenly in horses going to work after complete rest for a few days but maintained on full work-rations.
- The animals suddenly stop; sweat, shiver and show great suffering from pain in the lumbar regions
- The affected muscles are those of gluteal, lumbar and femoral regions and are swollen and board-like.
- Soon the animal passes coffee colored dark-brown or black urine since it contains large quantities of myoglobin.
- Animals lie down and soon die.
- Those that survive are weak and it takes a long time for them to recuperate and for the atrophied muscles to regain their normal state.
    -
    - Pathogenesis
- In normal muscle contraction, muscle glycogen is converted into pyruvic acid
- Due to inadequate oxygen 1/5 of this is oxidised to CO_2 and H_2O to liberate energy. The rest is converted into lactic acid which is converted into glycogen in the liver and used again
- When the animal is at rest but well fed, the muscles are well stored with glycogen.
- When the animal is put to work suddenly much of this glycogen is converted to lactic acid in the muscles and large amounts of this stimulate extreme contraction of the muscles, which become hard (board-like)
- In the contracted state of muscles, blood circulation is poor and so oxygen supply is reduced.
- Under this hypoxic condition, lactic acid is formed (from pyruvic acid) which still further contracts the muscles and so greater curtailment of blood flow occurs leading to still greater reduction of oxygen supply.
- Thus a vicious circle is established. The net result being that the muscles do not get sufficient amount of oxygen and nutrition, and so necrosis results.
- Necrosed muscle liberates myoglobin which is excreted in the urine.
- Large masses of myoglobin in the urine appear to produce renal blockade, renal ischemia and lower nephron nephrosis wherein the epithelium of the distal convoluted

tubules as well as that of Henle's loops are degenerated some of which become necrosed and desquamated.

- This condition causes degenerative changes in the tubules and so anuria and fatal uremia results
- Death is due to renal insufficiency leading to uremia.
    - Clinical pathology: Urine shows granular reddish casts and few hyaline casts
    - Gross pathology: affected muscles are swollen, pale and have increased amount of interstitial fluid.
    - Histopathology
- The changes in the muscle are those of Zenker's degeneration in which the muscle becomes a homogenous hyaline mass without striation.
- The fibres may be fragmented.
- There may be disappearance of all the constituents of the muscle fibres excepting the sarcolemmma and fibrous stroma. This is the cause of atrophy noticed in surviving animals.
- In animals that survive regeneration may occur but it is a very slow process.
- In the kidney lesions are found mostly in the tubules. The epithelium of the proximal tubules may be degenerated and all stages from cloudy swelling to necrosis are encountered. Desquamation of epithelium occurs. Similar changes may be noticed in the epithelium of Henle's loops and distal convoluted tubules. The lumens of the tubules may contain besides the desquamated cells masses of myoglobin. These form granular pigmented casts. A few hyaline casts may also be found.
    - **TUMORS OF SKELETAL MUSCLE**
    - Primary
- Rhabdomyoma, rhabdomyosarcoma, lipoma liposarcoma, fibroma, fibrosarcoma and myxoma.
    - Secondary
- Metastases of carcinoma and sarcoma are not frequent since muscle does not afford a suitable 'bed' or soil for them to grow.
- The following may be found occasionally – Lymphosarcoma, adenocarcinoma melanoma and angios
    - **SKIN AND ITS APPENDAGES**
    - **ABNORMALITIES OF HAIR**
    - Hypotrichosis: Presence of less hair is called hypotrichosis
    - Apennosis: Congenital lack of feathers (local or general) in fowls is called apennosis
    - Defluxion/Effluvium : Shedding of hair is called defluxion/effluvium
    - Alopecia
- Loss or lack of hair, wool, or feather is called alopecia
- *Etiology*
    - Ageing: Falling of old hairs and further growth does not occur
    - Nutritional: Deficiency of Vitamin- A, biotin, zinc and fatty acid deficiency
    - Pathological: Dermatitis, eczema and mange
    - Dry Lusterless Hair
- *Etiology*
    - Nutritional: Fatty acid deficiency
    - Minerals-Deficiency of Zn
    - Greying of hairs : Nutritional deficiency of copper
    - Rough hair : Roughness of hair may be seen in fatty acid deficiency

- Breakage of hairs / fibres

- Here the hair shaft is weak and easily breaks
- *Etiology*
  - Chemicals Poisoning: Chronic poisoning with thallium
  - Nutritional: Feeding excessive palm oil, soy oil, whale oil as milk replacers
  - **ALTERATION IN SKIN SURFACE**
  - Seborrhea
- *Definition* : Increased secretion of sebum leading to greasy skin and hair is called seborrhea
- *Occurrence*
  - Horses -Greasy heel
  - Newly calved dairy cows
- *Predisposing factors*
  - Standing in unhygienic barns in horses
  - Standing in mud floor in cows
- *Etiology*
  - Nutritional: Vitamin - A deficiency and fatty acid deficiency
  - Pathological - dermatitis
- *Pathogenesis*: Seborrhea is due to increased secretion of sebum
- *Clinical signs*
  - Horses: Back of pastern – Appears greasy excoriation and soreness is seen. The affected part is sensitive to touch resulting in lameness
  - Newly calved dairy cows: The lesions are commonly found in the groin between the udder and the thighs or in the udder itself between the two halves. There is voluminous sebum secretion and the skin may be necrosed. Bad smell exudes. The affected skin may be shed.
  - Icthyosis: Scaly skin is called icthyosis (The skin is scaly normally in fishes and reptiles)
  - **DRIED MATERIALS ON THE SKIN**
  - Scales
- Bran like grayish thin flakes on the skin is called scales
- *Etiology*
  - Scales may be seen in
    - Dandruff
    - Chronic dermatitis
    - Fine scaling occurs in fatty acid deficiency
- *Histopathology* : The scales consist of imperfectly keratinized superficial layers of the epidermis.
  - Dandruff / Pityriasis
- Dandruff is the presence of excessive scales on the skin
- *Predisposing causes*: Presence of dirt on the skin
- *Etiology*
  - Nutritional: Vitamin A deficiency and fatty acid deficiency
  - Disturbance of skin metabolism
  - Parasites: Lousiness
  - Poisoning: Chronic iodine poisoning
  - Chronic dermatitis
- *Histopathology*:, they are imperfectly keratinized superficial layers of epidermis.

- Crust (Coagulated exudate): Crust is a material formed by drying of exudates or secretions on the surface of skin
- Comedo: Comedo is a plug of keratin and dried sebum in a hair follicle

DISORDERS OF KERATINIZATION

- Acantholysis: Separation of keratinocytes is called acantholysis
- Hyperkeratosis: Excessive formation of keratin over the skin surface is called heperkeratosis
- Parakeratosis

- *Definition*: Accelerated imperfect keratinization is called parakeratosis
- *Gross pathology* : Grossly, there is production of scales.
- *Histopathology* : stratum granulosum is reduced in size. Nuclei of horn cells are retained.
  - Dyskeratosis
- *Definition*
  - In dyskeratosis, cells of Malphigian layer undergo abnormal, premature imperfect keratinization. It suggests developing malignancy.
- *Histopathology*
  - Microscopically, there is large number of mitotic figures with hyper chromatism and loss of polarity.

DEFECTS OR DEFECIENCIES IN THE SKIN

- **Erosion / Excoriation**
- Definition: Partial or complete loss of superficial epithelium
- Etiology

- Erosion may be caused by
  - Injury by sharp objects
  - Continuous discharges from the nearby skin lesion
  - **Ulcer**: Break in the continuity of epidermis exposing the underlying dermis. It is deeper than erosion.
  - **Fissure / Rhagade**
  - Definition : Deep linear defect in the epidermis often extending into the dermis
  - Occurrence : Occur in the dry crusty skin in which elasticity is lost
  - Etiology
- Cracking of soles occurs in pigs due to biotin deficiency
- Cracking of thickened skin occurs in vitamin –C deficiency in calves

SEPARATION OF LAYERS OF SKIN

- **Epidermolysis** : Separation of epidermis and dermis
- **Thickened skin** : Thickened skin may be caused by fatty acid deficiency
- **Hyperkeratosis** : Abnormal thickening of stratum corneum is called hyperkeratosis
- Etiology

- Hyperkeratosis occurs in hypothyroidism due to diseases of pituitary or atrophy of thyroid gland
- Diseases of other organs: Cirrhosis in dogs and cattle

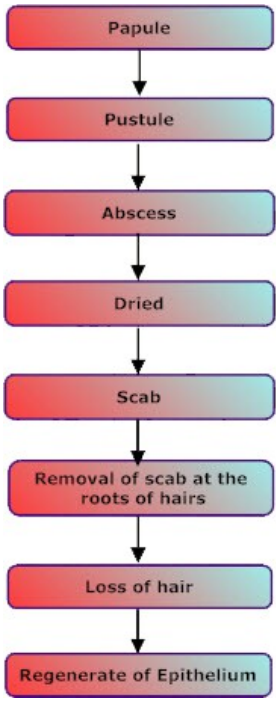
Acanthosis

- Thickening of epidermis due to hyperplasia of cells of stratum spinosum is called acanthosis.
  - Acanth-horny
  - sis -formation/process

- It may or may not be associated with hyperkeratosis or parakeratosis.
    - **Pseudoepitheliomatous hyperplasia**
    - Definition
- Severe acanthosis with deep downward growth of rete pegs (resembling carcinoma) is called pseudoepitheliomatous hyperplasia.
    - Etiology
- Pseudoepitheliomatous hyperplasia may be seen in
    - Margins of burns
    - Indolent ulcers
    - Chronic focal inflammations
    - **Lichenification**
    - Definition: Thickening of skin in irregular areas is called lichenification
    - Etiology: Chronic dermatitis
    - **Pachyderma**
    - Synonym : Elephantiasis
    - Definition
- Thickening of skin involving all the layers is called pachyderma (Elephantiasis). Here individual cells are normal. Here connective tissue may be hyperplastic.
    - Etiology
- Bacteria: Actinomycotic pachyderma in swine-Here the ears are very much enlarged and hard
- Nonspecific dermatitis: Seen in hind limbs of horses and scrotum of old dogs.
    - **SOLID ELEVATION OF SKIN**
    - Pimple or Papule
    - It is a small, circumscribed, cone shaped elevation of skin.
- *Histopathology* : The deep corium is infiltrated with leucocytes.
    - Proud flesh
- If irritant, movement or trauma prevents healing of wounds then excessive granulation tissue is produced and is known as proud flesh.
    - Keloid
    - Keloid is a connective tissue proliferation below the scar. It may recur after excision. It is not a true tumour.
- *Occurrence*
    - Horses
    - In Negroes, it is due to genetic or familiar predisposition
    - Calcinosis Circumscripta
- *Definition* : Raised, elevated or bulging mass of 1-10 cm diameter under the skin
- *Occurrence* : Common in dogs
    -
- *Gross pathology*
    - On section, white granular masses are present. This chalky paste like material may be enucleated.
- *Histopathology*
    - The mass contains calcium and may be separated by thin connective tissue septa. It is surrounded by granulation tissue consisting of lymphocytes, macrophages, plasma cells and giant cells.
        - **FLUID ACCUMULATION IN THE SKIN**
    - Hydropic or Balooning degeneration

- *Definition*: Intracellular edema is called hydropic or ballooning degeneration. Here cells are swollen and become isolated from one another. The prickles disappear (acantholysis).
- *Etiology*: Hydropic degeneration is an early stage in vesicle formation. e.g. viral diseases
    - Spongiosis
- *Definition*: Intercellular edema of the epidermis is called spongiosis
- Etiology : It is seen in inflammation.
    - Wheal / Urtica
- *Definition:* It is small sharply circumscribed flat round or irregular shaped elevation of skin.
- *Etiology* : Due to edema seen in u rticaria or allergic conditions
    - Urticaria : Urticaria is an allergic swelling of skin
- *Etiology*
    - Primary causes include
        - Nutritional: Unusual food
        - Parasite: Larvae of warble fly
        - Contact with caterpillar hairs (nettles)
        - Poisoning: Insect bites
        - Medicines like penicillin
    - Secondary causes include
        - Horses: Bacteria: Strangles
        - Swine: Bacteria: Erysipelas
        - Fowls: Bacteria: Chronic fowl cholera (Wattle disease)
- Gross pathology: Skin contains wheals
- Histopathology
    - First vascular dilatation leads to erythema and later followed by exudation in the skin
    - Plaque: Several urtica coalesce to form large plaques
    - Blister / Vesicle
- *Definition*
    - It is a cavity (diameter <5mm) in the epidermis or beneath the epidermis containing serum, plasma or blood. It is raised above the surface of skin. It is covered by a thin rim of epithelium. It is a form of severe spongiosis.
- *Sequelae*: Many vesicles may coalesce to form a bulla.
    - Bulla / Bleb
- Bulla is a space containing fluid situated intraepidermally or subepidermally. It is a large vesicle (size >5mm).
    - Pustule: Pustule is a small circumscribed cavity in the epidermis containing pus.
    - **CHANGES IN COLOUR OF SKIN**
    - Black Mole: Black mole is a hyperpigmented spot in the skin
    - Hyperpigmentation
    - In hypothyroidism due to diseases of pituitary or atrophy of thyroid gland, diffuse or local hyperpigmentation occurs.
- *Etiology* : Nutritional deficiency of zinc
    - Leucoderma
- *Etiology*
    - Scars after healing of wounds
    - Congenital defect in certain breeds of dogs

- *Sequelae*: If exposed to sunlight, the affected area is inflamed.
    - Erythema
- *Definition* : Erythema is a reddish spot in the skin
- *Etiology*
    - Nutritional deficiency of zinc
        - Erythema is due to focal congestion of capillaries. It is the first stage of inflammation of skin. In some cases, the inflammatory process does not proceed beyond the stage of congestion and it is these cases that may be termed erythema secondary to some specific fevers
        - e.g. Swine fever and swine erysipelas infection
    - Makula
- Makula is a discoloured spot of skin due to focal hyperaemia or haemorrhage. It is not elevated above the surface of skin.
    - Contusion (Bruise)
- Contusion or bruise is an injury caused by blunt force resulting in breach of the subcutaneous tissue, disruption of blood vessels and infiltration into surrounding tissue of blood. There is no loss of continuity of skin.
    - Albinism
- *Definition*: Albinism is complete absence of melanin pigment in the skin (white skin)
- *Occurrence* : Species affected: Man, horses, Dogs (Bull dogs and Collies), cats, mice, rats and rabbits.
- *Etiology*: Absence of tyrosinase (since the epidermal cells are DOPA negative)
        - **DISCOLOURED AND ELEVATED SPOTS IN THE SKIN**
    - Eruption ; Eruption is a small rapidly developing elevated red spot.
    - Boil or Furuncle
- *Definition* : It is a small suppurative inflammation (Abscess) of skin involving a hair follicle or sebaceous gland.
- *Contributing causes*
    - Sweating
    - Contamination by filth
    - Decreased vitality of skin
- *Etiology*
    - Bacterial
        - *Staphylococcus aureus*
        - *Corynebacterium pyogenes*
    - Viral cause: Complication of canine distemper
    - Fungi: Ringworm
    - Parasite: Mange
- *Gross pathology*: Local lymph nodes may be enlarged



- *Histopathology*: Hair follicle contains inflammatory exudate consisting of leucocytes.
  - Carbuncle: A cluster of boils situated close to each other. They open onto the skin through several pores.
  - Acanthosis Nigrican: Acanthosis nigricans is a raised rough black patch of skin.
- *Occurrence* : This condition is mostly seen in dogs.
- *Etiology*
  - Hormonal: In dogs it is due to hypothyroidism
  - Pathological
    - Adenocarcinoma of liver
    - Sertoli cell tumour leads to increased estrogen production
    - Hypoplasia of pituitary
- *Gross pathology*
  - Bilateral skin lesion (ventral side of body, axilla, inner side of thighs, inguinal and circumanal region) is characteristic.
  - Small poorly circumscribed patches of skin will be thick, folded with alopecia and increased pigmentation (Black coloured)
- Histopathology
  - Dermal papillae –Elongated
  - Blood vessels – Congested
  - Epidermis – Prickle cell layer – Increased in thickness with prominent rete pegs
  - Hyperkeratosis
  - Pigment: Present in cells of all layers
  - Glands and hair follicles – Atrophied
  - **OTHER LESIONS OF THE SKIN**
  - Folliculitis: Inflammation hair follicle is called folliculitis
  - Acne: Inflammation of sebaceous gland is called acne
  - Miliaria: Blockage of sweat duct is called miliaria
  - Scar / Cicatrix
- Scar is a healed skin defect having neither hair follicle nor sweat gland.

- Affected area is puckered or wrinkled due to high amount of mature fibroblasts.
- Initially white but later becomes pigmented.
- *Histopathology*
    - Scar is covered by surface epithelium.
    - It consists of mature fibroblasts containing more collagen fibres which cause shrinkage.
    - The fibroblast nuclei are less prominent.
    - They require fewer nutrients hence avascular and white in colour.
    - It contains neither hair follicle nor sweat glands.
    - Dryness of skin: Occurs in vitamin –E deficiency in cats
    - Seborrhoea
- *Definition* : Seborrhoea means increased secretion of sebum
- *Occurrence*
    - Cattle standing in mud
    - Horses standing in unhygienic barns
- *Etiology* : Dermatitis - Seborrhoea in dairy cows
- *Gross pathology*
    - Seborrhea of dairy cows usually occurs in newly calved cow. The groin between udder and thigh or between two halves of udder emits bad smell and the skin may shed.
    - In greasy heal of horses, excoriation on the back of postern leads to soreness and appears greasy. On touch it is highly sensitive. It leads to lameness.

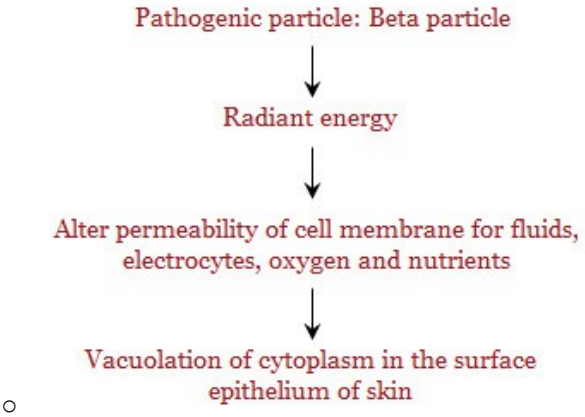
ANOMALIES

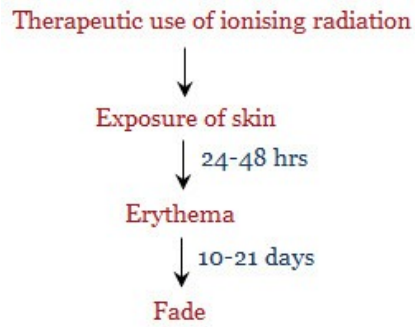
- Epitheliogenesis Imperfecta
- It indicates a condition wherein the skin fails to develop around nose, ear and feet.
- *Occurrence* : Species: Calves, piglets, pups, lambs, kids and foals
- *Etiology* : Genetic defect: Autosomal recessive character
- *Sequelae* : The skin may be infected leading to septicemia quickly.
    - Hypotrichosis Congenita
- *Synonym* : Congenital alopecia
- *Types:* Hypotrichosis congenita may be partial or complete.
- *Occurrence* : Species: Calves, foals, dogs (Dachshunds)
- *Etiology* : Hereditary - Mendelian recessive trait in calves
- *Sequelae* : Affected animals are easily susceptible to cold and sun burns.
    - Alopecia
- *Definition*
    - Loss or lack of hair, wool, or feather is called alopecia. Further growth of hair does not occur at the affected area.
    - If the baldness is confined to more or less circumscribed patches it is known as alopecia areata
- *Etiology*
    - Alopecia is due to various causes which include
        - Physiological: Temporary loss of hair in lactating mares and bitches. In aged animals old hairs fall out and further growth does not occur.
        - Nutritional: Feeding of excessive palm oil, soy oil, whale oil as milk replacers causes alopecia (The fibres also break easily). Deficiency of Vitamin like biotin (in pigs), vitamin-C (in calves; also produces cracking of thickened skin) and vitamin- E in Cats (Loss of hair on the head and extremities; also produces dryness of skin) produces alopecia. Deficiency of minerals like iodine in newborn piglets has alopecia due

to mothers fed on iodine deficient feed during gestation period. Deficiency of Vitamin- A, biotin, zinc and fatty acid may also cause alopecia.
        - Plant poisoning due to ingestion of *Tamarindus indica*, Senecio and *Chrysocoma tenuifolia* in sheep and goats.
        - Chemicals Poisoning : Chronic poisoning with thallium - Here fibres break and shaft is weakened. Chronic poisoning with arsenic or selenium also leads to alopecia.
        - Pathological: Dermatitis and eczema
        - Inflammatory skin disease – In ectoparasitic (mange) or mycotic infection due to itching irritation occurs. The animal rubs against wall or hard objects which lead to denudation of hairs.
        - Injury to peripheral nerve causes alopecia in the affected area
        - Diseases of other organs: In cirrhosis of dogs and cattle (hyperkeratosis is also found)and chronic interstitial nephritis in dogs, partial alopecia occurs
        - Atrophy of testicles in adult dogs
        - Disturbance of estrous cycle in bitches
        - Following severe febrile disease – In sheep affected with blue tongue partial or complete loss of wool occurs.
- *Endocrine imbalance*
    - In male dogs having sertoli cell tumours, hyperestrogenism leads to symmetrical alopecia.
    - In Cryptorchidism (unilateral or bilateral), due to deficiency of testosterone bilateral alopecia occurs.
    - In bitches having hypoestrogenism, alopecia occurs at posterior part of abdomen, inside the thighs and under the tail.
    - In hypothyroidism due to diseases of pituitary or atrophy of thyroid gland, bilateral alopecia occurs. In addition hyperpigmentation and hyperkeratotsis are noticed.
- *Psychic disturbance*: Psychological upset in human beings leads to alopecia areata
- *Hereditary*
    - It is a congenital defect in calves due to single autosomal recessive character. Symmetrical alopecia occurs during 6^{th} week to 6 month of post natal life. It commences on head, back, hind quarters and extends to other parts (completely bald).
    - Congenital Icthyosis
- *Definition* : Skin of affected animals look like scales of fish
- *Etiology* : Hereditary - Simple recessive lethal gene in new born calves
- *Gross pathology*
    - Skin
        - Devoid of hair
        - Hard, horny plates with fissures separating them is present
- *Sequelae* : Affected animals usually die in 1-2 days
- *Histopathology*: Acanthosis, Hyperkeratosis.
    - **PHYSICAL INJURIES OF SKIN**
    - **COLD INJURY / FROST BITE**
    - Physiological considerations
- Well acclimatized long haired animals can tolerate temperatures of -50°C for indefinite periods.

-
    - Occurrence
- Cold injury occurs most commonly on the tips of the ears and tail of cats, the scrotum of male dogs and bulls and the tips of the ears, tail and teats in cattle.
    - Gross pathology
- Gross lesions include
    - Alopecia
    - Scaling
    - Dry gangrene
    - Sloughing of affected skin
    - Histopathology
- Histopathological alterations in the affected skin includes
    - Necrotizing vasculitis
    - Hyperemia
    - Haemorrhage
    - Neutrophilic infiltration
    - Thrombosis of small vessels
    - **S**
    - Anatomical features of skin
- The skin is the largest organ in the body
- Colour is black in buffaloes
- Thickness varies in different animals - Goat, sheep, dog, horse, cattle, buffaloes, hippos, pigs and elephant.
- In Non-hairy regions, the epidermis is thick e.g. Sole of foot, palm of hand
    - Physiology of skin
- Organ of heat regulation
- Provides a covering to the body
- Gives colour to the body surface
- Organ of excretion
- Organ of sensation for air, particulate matter, liquids, heat, touch and pressure
- Protects the body from physical, chemical and microbial invasion
- Can be invaded by various agents like bacteria, fungus and parasites
- Needed nutrients are
    - Protein
    - Essential fatty acid
        - Linoleic and Linolenic acids
    - Vitamins
        - Water soluble – Biotin
        - Fat soluble- A and E
    - Minerals-Copper and Zinc
    - **THERMAL INJURY**
    - Etiology
- Thermal injuries in domestic animals can be caused by hot liquids, steam, fires, and friction from rope “scalds”, electrical burns from chewing electrical wires and lighting strikes.
    - Pathogenesis
- The lowest temperature at which skin can burn is 44°C.

- Dry heat causes desiccation and carbonization of skin
- Moist heat causes “boiling” or coagulation of skin
- Longer exposure to lower temperature is more damaging than short exposure to higher temperatures
    - Gross pathology
- Animals struck by lightning may show a jagged line of singed hair running down the shoulder or flank.
    - Histopathology
- Burns are classified into four degrees according to depth of injury
    - First degree burns involve only the epidermis
    - In second degree burns, the epidermis and part of the dermis are damaged
    - In third degree burns, the destructive effect of the burns extends full thickness through the epidermis and dermis causing coagulation necrosis of connective tissues, blood vessels and adnexa.
    - Fourth degree burns are similar to third degree burns but also extends to the subcutaneous fascia.
    - **DIRECT EFFECT OF SOLAR RADIATION**
    - Solar dermatitis / Sun burn
- Sun burn occurs most frequently in cats, dogs, pigs, cows and goats.
- The lesions in cats typically affect the tips of the ears, nose, eyelids and lips of white, blue –eyed animals.
- The initial lesion is erythema followed by alopecia, scaling and crusting.
    - Photosensitization dermatitis
- Photosensitization dermatitis occurs in animals when photodynamic or fluorescent pigments are deposited in sunlight exposed skin.
- Phylloerythrin, a degradation product of chlorphill and haematoporphyrin may accumulate in the skin.
- The initial reaction is eythema followed by edema which is more prominent in sheep than in cattle.
- Histologic lesions include coagulative necrosis of the epidermis and dermis.
    - **DERMATITIS CAUSED BY IONIZING RADIATION**
    - Pathogenesis: Skin sensitivity: Radio responsive (comparatively radio-resistant)





- Histopathology

- Blood vessels:
  - Vascular endothelial cells- Swollen
  - Intima- Thrombosis occurs which obstructs the vessels
  - Wall of the blood vessels: Fibrous tissue becomes hyalinised
  - Severe exposure of skin to ionizing radiation leads to the following changes in the skin
- Dermatitis
- Edema
- Parakeratosis
- Necrosis of epidermis
- Damage to hair follicle - Loss of hair (Epilation) which may not grow again
- Destruction of sweat and sebaceous glands
- Ulcerated area may be infected secondarily by saprophytes leading to gangrene
- *Dermis*
  - Hyperaemia
  - Edema
  - Fibrous tissue: Hyalinisation
  - Infiltration by lymphocytes and neutrophils occur
  - Sequelae
- If dermis is not affected complete healing occurs.
- If there is widespread necrosis with involvement of the dermis, scar tissue may be formed. But this scar is very weak. Epidermis is thin and inadequately keratinized. Carcinomas are supposed to arise from such scars of old X-ray burns.

DERMATITIS

- Definition

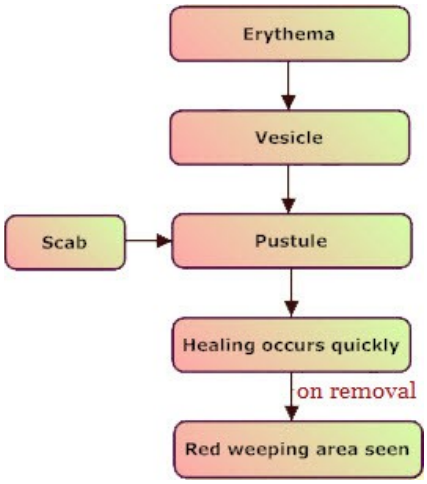
- Inflammation of the skin is called dermatitis
- If the skin is moist, then it is called moist eczema (Acute condition)
- If the skin is dry, then it is called dry eczema (Chronic condition)
  - Dermatosis : Non inflammatory lesion of skin is called dermatosis.
  - Predisposing factors
- Continuous sweating
- Hereditary
  - Etiology
- *Physical*
  - Continuous pressure leads to decubital ulcers on the skin
  - Trauma- abrasions, scratches etc
  - Excessive exposure to heat or cold

    - Excessive sweating causes maceration of the stratum corneum. The macerated area may be easily invaded by bacteria
    - Beta radiation
    - Photosensitization
    - Sun burn in unpigmented animals
- *Nutritional*
    - Fatty acid deficiency causes acute moist dermatitis.
    - Deficiency of Vitamin like biotin (in pigs)
- *Chemicals irritants*: Acids, bases and Chronic poisoning with thallium
    - Allergic
- Allergen
    - Endogenous
    - Exogenous
    - Endogenous
- Nutritional - overeating
- Stasis of food - Constipation
- Undigested protein
- Digested parasites
- Exogenous
- Fungal
- Parasitic - Lice infestation in chicken
- Chemicals e.g. Medications on the skin
    - Types of dermatitis
- Based on nature of exudates
    - Serous dermatitis
    - Papular dermatitis
    - Suppurative dermatitis
    - Necrotic dermatitis
    - Parasitic dermatitis
    - Gross pathology
- *Moist eczema*
    - Initially, vesicles and bullae are formed which may be infected to produce pustule. The vesicles may rupture to produce a watery discharge or weeping on the surface. Later pustule may dry forming scabs.
- *Dry, chronic eczema*
    - Scratching and rubbing of skin leads to thickening of skin (lichenification), scales, scabs/crusts and fissures.
    - Histopathology
- *Moist eczema* : Spongiosis, edema and cellular infiltration in the dermis may be seen.
- *Dry, chronic eczema*
    - Hyperkeratosis
    - Acanthosis with prolonged rete pegs
    - Dermis shows fibrosis and infiltration with lymphocytes
    - **SEROUS DERMATITIS**
    - Definition : Serous dermatitis is a mildest type of dermatitis and the exudate is serous in nature.
    - Etiology

- Physical: Trauma – Mild friction e.g. abrasions, scratches and ill fitting saddle and harness
- Thermal
    - Exposure to heat and cold
    - Sun burn especially in unpigmented animals
    - Mild Chemicals
    - Gross pathology
- Skin: Colour- Red
- Surface – Raised
- Sweat and sebum are secreted in large quantity (Seborrhoea)
    - Histopathology
- Spongiosis
- Infiltration by a few leucocytes
- Blood vessels show hyperemia
    -
    -
    - Sequelae
- Upon removal of the cause serous dermatitis resolve quickly and no permanent damage occurs
- If the cause is more intense or persists, a cute dermatitis develops
    - **ACUTE VESICULAR DERMATITIS**
    - Definition: It is an acute type of dermatitis characterized by vesicles.
    - Etiology
- Physical
    - Thermal – Hot and cold application
    - Photosensitization , More intense sun burn
    - Pressure – Decubital ulcers
- Chemicals : Stronger chemicals -Acids, Bases and Thallium poisoning
- Bacteria: Anthrax, tuberculosis and swine erysipelas
- Viral: Pox diseases, contagious ecthyma, FMD, rinderpest and mucosal disease
- Fungus : Ringworm, *Candida albicans* (moniliasis) and epizootic lymphangitis
- Parasitic : Nematodes, *Stephanofilaria* and ectoparasites
- Protozoa: Leishmaniasis
    - Gross pathology
- Skin
    - Color – Red (Erythema)
    - On palpation heat can be felt in the affected area
    - Swelling of the affected skin due to edema leads to formation of vesicles or blebs which contains clear fluid. If the roof of the vesicle dies the vesicle may rupture revealing a red base
    - Histopathology
- Epidermis shows spongiosis
- Prickle cells undergo hydropic degeneration which may rupture to form vesicle and bullae
- The roof of vesicles is formed by stratum corneum.
- If the vesicle is infiltrated with leucocytes, it develops into a pustule. The pustule on its rupture leaves discontinuity in the skin and the coagulated exudates forms crusts

on the skin. The undersurface of crusty area is infiltrated with neutrophils. Upon healing the surface epithelium regenerates.

- Dermis shows edema and is infiltrated with lymphocytes and macrophages
- Stratum germinativum: Blood vessels show hyperaemia.
    - **PUSTULAR OR SUPPURATIVE DERMATITIS / IMPETIGO**
    - Definition : Impetigo is a form of pustular dermatitis
    - Predisposing factors
- Dirty litter
- Humid places
- Skin that is continuously drenched by discharge from wounds or natural opening becomes soft and is easily infected.
- Milking infected udder may lead to spread of infection to other animals
    - Etiology
- Physical: Injury by thorns in sheep. If the injured area is infected impetigo develops.
- Bacteria
    - *Staphylococcus aureus* in cows causing mastitis
    - *Streptococcus* - rarely
- Viral
    - Dogs- Canine distemper
    - Sheep and goats – Contagious pustular dermatitis
- Route of infection : Bite wounds in pigs by dogs and other pigs
    - Gross pathology
- Pustule may be seen
    - Pigs: Location - face, snout, ears and over the eyes
    - Dogs affected with canine distemper pustules are seen under the belly
    - Cattle: Bacteria – Staphylococcal mastitis. In the udder, the base of teat is the common location of lesion.



    - Histopathology
- Pustule contains serofibrinous exudate with neutrophils
- Dermal vessels are congested.
    - Sequelae : If the dermis is diffusely inflammed, then it is called phlegmon.
    - **NON-NEOPLASTIC CYSTS**
    - Epidermoid Cyst
    - Species affected: Dogs
- *Pathogenesis*

    - If the hair follicular duct is occluded, then the desquamated epithelium and keratin are trapped inside and develop into a cyst.
    - The development of cyst is slow.
- *Gross pathology*
    - Location: If in the dermis, it is fixed. If in the subcutis, it is movable.
    - Shape: Round or oval
    - Size: Few millimeters to few centimeters
    - Number of cysts: Solitary, clusters or generalized
    - Wall: Thin
    - Margin: Well demarcated
    - Contents: Color is gray or brown; May contain hair/wool
    - Consistency: Semisolid or dry
- *Histopathology*
    - At the centre of cyst, keratin is deposited concentrically.
    - The cyst wall has a collagenous capsule, surrounded by squamous epithelium and is devoid of adnexae.
- *Sequelae*
    - Usually benign and rarely become ulcerated if infected or malignant
    - If the contents escape into tissue foreign body reaction sets in.
    - Dermoid Cyst
- It is a round or oval soft mobile cyst at the junction of dermis and subcutis.
- Its wall contains skin appendages.
- It communicates to outside though tiny pores.
- It is a benign lesion. It may contain desquamated cells, hairs, keratinized greasy substance, calcium and cholesterol.
    - Sebaceous Cyst
- It is a dilatation of duct or sebaceous gland leading to cyst formation.
- It may contain greasy sebum and cholesterol.
    - Sudoriferous Cyst
- It is a cyst formed by the occlusion of sweat gland.
- It contains watery fluid.
- The capsule is thin and lined by single layer of columnar or cuboidal epithelium.
    - **TUMORS OF SKIN**
    - Epidermal origin
- The common tumours arising from epidermal origin include papilloma, squamous cell carcinoma, basal cell carcinoma and melanoma (Benign and malignant)
    - Dermal origin
- Fibroma, fibrosarcoma, keloid of horses, equine sarcoid, venereal tumour of dogs, mastocytoma and myelocytoma
    - Adnexal tumours
- Adenoma and adenocarcinoma of sweat glands, sebaceous glands and perianal glands; hair matrix tumour and basal cell carcinoma.
    - Tumours arsing from subcutaneous tissue
- Lipoma, lymphangioma, hemangioma and hemangioendothelioma
    -
    - **PATHOLOGY OF APPENDAGES OF SKIN**
        - **PATHOLOGY OF SUBCUTIS**
    - Emphysema
- Etiology: Air entering through cutaneous wound

- Pathogenesis: Movements of skin exert a sucking or pumping action for the air to enter the subcutis
    - Cellulitis: Cellulitis means inflammation of subcutis. Subcutis may have congestion, haemorrhage, edema or purulent exudate.

PATHOLOGY OF HOOF

    - Laminitis
- *Synonym* : Founder or fever in feet
- *Definition*: Laminitis is inflammation of sensitive laminae of hoof
- *Occurrence*
    - Horses and cattle may be affected
    - It may be seen in fore feet, all four feet or occasionally in hind feet
    - Can occur in a single foot – usually as a complication of a severe lameness or orthopedic disease in the contralateral limb.
- *Etiology*
    - Laminitis may be a local manifestation of a more generalized metabolic disturbance
    - Horses
        - Diarrhoea occurring due to excessive ingestion of CHO and use of irritant purgatives like aloes
        - Drinking very cold water after the animal is over heated
        - Long drives / severe exercise on hand roads leads to concussion
        - Standing for long time
        - Fat ponies which are kept at pasture without much exercise develops chronic laminitis
    - Cattle
        - Feed allergy caused by Cottonseed cake, mouldy hay and barley (Allergic laminitis)
        - Retained placenta, post parturient metritis, mucosal disease and endotoxaemia.
    - Cattle and Sheep : Overfeeding with concentrate
- *Pathogenesis in horses*

Course may be acute, subacute or chronic

↓

Toxins (Indol, phenol) formed from tyrosine and tryptophan binds to sulphate ions of blood

↓

The balance between sulphur of blood and sulphur in chondroitin sulphate of hoof is altered

↓

Collagen losses it's strength and elasticity

↓

Keratogenic lamellae losses it's supporting tissue

↓

Degeneration and destruction of keratogenic lamellae

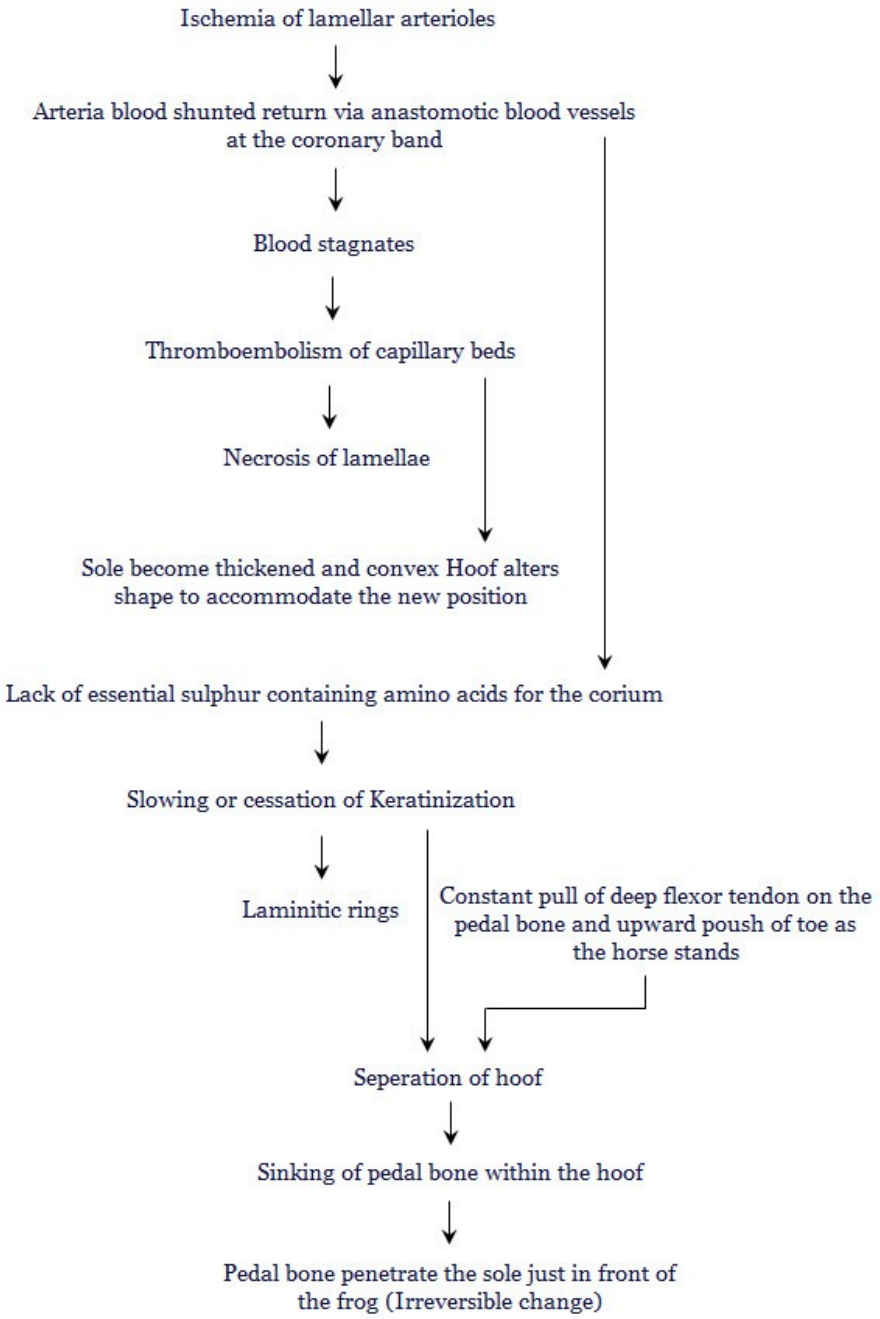
↓

Do not produce sufficient keratohyaline. Loss of eleidin granules and onychogenic fibrils

↓

Hoof becomes uneven

-
-



- *Clinical signs*
  - Horses
    - Onset: Sudden
    - High temperature
    - Rapid pulse
    - Accelerated respiration
    - Distress
    - Does not like to bear weight on affected limb
  - Cattle and sheep
    - Depressed
    - Anorectic
    - Stands reluctantly
    - Hoof (Touch) palpation – Heat is apparent near the coronary band

        - Sole of feet has increased sensitivity
        - Shifting lameness
        - Reluctance to walk
        - Each foot once lifted is replaced as quickly as possible
- *Gross pathology*
    - Hoof
        - Acute congestion with venous stasis
        - Pressure of increased blood and edema on sensitive tissue which presses on the tough hoof causes extreme pain.
        - Haemorrhage occur into the sensitive laminae
        - Suppuration occurs in severe conditions
        - As the keratogenic lamellae are affected, the formation of hoof is uneven
    - In cattle and sheep
        - At the coronary band, blood stained exudate seep
    - Hoof: Bands of irregular horn growth
        - Laminitic rings (close at the toe and diverging at the heel) can be seen
        - Narrow and elongated
            - At heel-vertical
            - At toe – horizontal.
            - Sole – Thick
            - Outline – convex / Flat
    - Radiography : Pedal bone in cattle shows osteoporosis
- *Sequelae*
    - If laminitis subsides in 3-4 days, no permanent damage occurs.
    - If persists, permanent damage to hoof occurs. In a few days, separation of sensitive laminae from inner laminae occurs. Due to weight of the body, *os pedis* takes a more perpendicular position and may pierce the sole. The weight is borne by heels and so the toes grow exceedingly long with convex lower surface. Concentric rings on hoof parallel to coronet may occur.
    - If the divergence is more than11° in cattle and sheep, the prognosis is guarded to unfavorable.
    - Secondary infection
    - Laminitis in Goats
- *Etiology*
    - Sudden access to concentrates
    - High grain and low roughage diet
    - High protein diets
    - Complication of acute infections like metritis, mastitis and pneumonia especially after kidding
- *Signs*
    - Touching the coronet elicits a severe pain reaction
- *Gross pathology*
    - Only fore feet are affected in less severe cases

PATHOLOGY OF NAIL

    - Nail Abscess
- Synonym: Septic laminitis, lamellar suppuration and toe abscess
- Definition: Acute bacterial infection of the laminar matrix of the hoof usually restricted to toe and abaxial wall.
- Occurrence

- Sporadic
- Fore feet are affected commonly

- Etiology
  - Bacteria - *Spherophorus necrophorus, Corynebacterium pyogenes* and
  - Streptococci
- Route of infection
  - Through – fissures between wall and sole; vertical and horizontal fractures of horn impacted with mud & feces
- Signs
  - Severe lameness
  - Affected digit is hot and tender
  - At coronet sinus may be seen

PATHOLOGY OF HORN

- Horn fracture

- Etiology: Fracture of horn occurs due to traumatic injury like fighting between animals.
- Types : Horn fracture may be complete or incomplete
- Sequelae
  - If properly immobilized, incomplete fracture of distal portion may heal
  - Fracture of horn causes haemorrhage into the frontal sinus and bleeding from the nostrils. Accumulation of blood in the frontal sinus might lead to purulent sinusitis and empyema of the sinus.
  - Horn avulsion
- Synonym: Evulsion of horn
- Definition : Separation of horny covering of the horn core is called avulsion of horn.
- Etiology : Horn avulsion occurs when there is injury to the base of horn as well as in horn cancer.

THE EYE AND EAR

PATHOLOGY OF EYE

THE EYE

- Anatomical features

- The eye ball is located in the bony cavity of the skull knows as the orbit. It is protected by eyelids, which have skin on the outside and stratified squamous epithelium (the conjunctiva) lining the surface that comes into contact with the eyeball.
- The borders of the eyelids have eyelashes containing hair. Just behind the eyelashes are a row of tiny sebaceous glands, the meibomian glands, the secretion of which serves to lubricate the eyelashes, preventing their adhesion.
- The lens is a peculiar structure composed entirely of epithelium. It has neither stroma nor vascular tissue. In front it is bathed by the aqueous humor and is nourished by it.
- Actually the anterior surface of the lens forms the posterior boundary of the anterior chamber. Its anterior surface is in contact partly with the iris. Its posterior surface fits into the depression of the vitrous- the hyaloid fossa.
  - Physiological considerations
- Usually, the conjunctival mucosa is free of bacteria either due to the flushing action of the tears or to the bacteriostatic property of the lysozyme.

CONGENITAL ANOMALIES

Anophth	This is complete absence of one or both eyes. This

almia congenitus	condition was reportedin foals, pups, calves and piglets. Histological examination may reveal remnants of some ocular structures.
o Microphthalmia	o Microphthalmia is a condition in which one or both eyes are small. The condition was reported in swine and dogs
o Cyclops	o Cyclops is a condition in which there is only one eye due to fusion of the orbits and is seen in monsters.
o Ankyloblepharon	o Ankloblepharon is a condition in which both the eyelids are fused together
o Starbismus	o Starbismus is squint of human beings. In animals this condition is bilateral with the two eye globules tuning inwards. Squint is seen in Siamese cats in which it is congenital. This has also been reported in calves of beef breed and in collie dogs.
o Entropion	o Entropion is turning in of the eyelids and is hereditary congenital condition seen in sheep, dogs and foals.
o Ectropion	o Ectropion is turning out of eyelids. Usually the lower eyelid is affected. This is also a hereditary condition.
o Coloboma	o Coloboma is a congenital anomaly due to failure of the closure of embryonic ocular cleft. Here the eyelids, ciliary body, lens or iris may be affected showing fissures or gaps in their continuity. In the eyelids a small wedge shaped portion may be missing or even larger area may be lacking. Similarly small areas of iris and lens may be absent.
o Dermoids of cornea	o These tumours occur as congenital condition probably due to the sublethal factor. The cornea of one or both eyes is partly covered by skin.Because of constant friction and irritation by hair conjunctivitis,pannus formation, keratitis or ulceration may result.
o Congenital anterior synechia	o Congenital anterior synechia is a condition in which where is adhesion between iris and the posterior surface of the cornea.
o Congenital opacity of cornea	o Congenital opacity of cornea may be met with, sometimes, as a result of anterior synechia. In certain breeds of cattle hereditary opacity of cornea has been reported.
o Microphakia	o Here the lens is small and is spherical. It may be opaque or transparent.

o Luxation of the lens	o Displacement of the lens or luxation of the lens may be met within some animals. The dislocated lens is opaque. In certain breeds of dogs, it is a developmental defect.
o Cataract	o Cataract is a condition in which the lens becomes opaque. In some animals it is a congenital condition.
o Congenital aplasia of retina and hypoplasia of the optic nerve	o Congenital aplasia of retina and hypoplasia of the optic nerve may be met with in calves and they are born blind.

o
o
o

o **PATHOLOGY OF EYELIDS**

o Trichiasis

- Trichiasis is the turning in of the eyelashes and therefore cornea is irritated by the hair.
- Keratitis and attendant lesions may be encountered.

o Blepharitis

- *Definition* : Blephartis is inflammation of eyelids.
- *Etiology*
  - This may be a part of generlised dermatitis. But it is usually a complication of a) distemper in dogs and cats b) trauma, c) conjunctivitis or d) inflammation of the lachrymal glands.
- *Sequelae*
  - Complications of blepharitis are i) Ankyloblepharon, when adhesion between eyelids takes place, ii) symblepharon, where there is union between the conjunctiva lining the eyelids and that covering the eyeballs.

o Hordeolum or stye

- Hordeolum or stye is the inflammation or even abscess formation of the follicles of an eyelid and is very painful condition.

o Chalazion

- Chalazion is the abscess formation of the meibomian glands. Sometimes a cyst may form in these glands.

o Edema of the eyelids

- This usually results due to i) trauma, ii) infection, iii) conjunctivitis, iv) allergy and v) fracture of the orbital ring.
- The following conditions are accompanied by edema of eyelids.
  - Horse: Influenza, pink eye, purpura and allergic condition.
  - Cattle: Malignant catarrhal fever, distomiasis, stomach worm affection, traumatic pericarditis and penetration of a foreign body into eye like husks etc.
  - Pig: Gut edema, chronic hog cholera.
  - Dog and cat: Distemper, allergy, stings by nettles and ants.

o Neoplasm

- Papilloma, basal-cell carcinoma and angioma may be found. Adenoma of the Meibomian glands may also be met with.
- Squamous cell carcinoma which may arise from the conjunctiva or the skin of eyelid or from the membrane nictitants is very common among cattle.

PATHOLOGY OF ORBIT

- Exophthalmos : Exophthalmos means protrusion of the eyeball.
- Enophthalmos: Enophthalmos means sinking of the eyeball into the orbit.
- Orbital cellulitis

- *Definition*: Inflammation of the orbit is called orbital cellulitis.
- *Occurrence*: Rare
- *Etiology*
  - Trauma by foreign bodies
  - Extension from periodontitis of posterior molars in dogs.
  - Orbital contusion complicated by fracture of the ring of the orbit.
  - Extension of opthalmitis
  - Nutritional deficiency in cats
  - Non-inflammatory edema with sight exophthalmos occurs in edema disease and mulberry heart disease of swine, congestive edema of head, purpura hemorrhagica and urticaria.
  - This condition is usually suppurative in nature.
  - Xerophthalmia: Xerophthalmia means dryness of the eye ball. It is due to vitamin A deficiency.

PATHOLOGY OF LACHRYMAL GLANDS

- Dacryoadenitis

- Definition: Dacryoadenitis is the inflammation of the lachrymal glands.
- Occurrence: It is rarely met with as a complication of conjunctivitis or trauma.
- Gross pathology
  - There is diffuse congestion and enlargement of the gland, causing protrusion of the embrane nictitans.
  - The ducts are dilated with inflammatory exudates
  - An abscess may form rupturing on the upper eyelid.
  - Occlusion of lachrymal canal
- Etiology
  - Occlusion of lachrymal canal may be congenital or acquired. In dogs of certain breeds (Sealyheams and Poodles) there may be congenital absence of the puncta.
  - The canal may be occluded in the following conditions. A)entropion, b) inflammatory swellings found in conjunctivis and rhinitis, c) atrophic rhinitis of swine, d) miller's disease in horses and e) neoplasms of the nasal passage.
  - Neoplasm of the lachrymal gland: Adenoma of the lachrymal gland is very rare.

PATHOLOGY OF CONJUNCTIVA

- Conjunctivitis

- *Definition*: Inflammation of the conjunctiva is called conjunctivitis.
- *Occurrence*: Conjunctivitis is commonly met with in animals.
- *Etiology*
  - The causes of conjunctivitis are
    - Bacteria: Some bacteria can penetrate the intact conjunctival mucosa- Brucella, Listeria and Pasteurella tularensis. Secondary infection

by *Staphylococcus aureus, Pseudomonas aeruginosa* and *E. coli* may occur following trauma or debiliting viral diseases.
        - Chemicals: Disinfecting fluids, lime in white washing material, irritant gases such as formalin vapour, sulphur, smoke, acids, alkalies, sheep dips, parasiticides, skin dressing, iodism.
        - Foreign bodies: Awns, oak husks, mud, dust and sand
        - Parasites: *Thelazia lachrymalis* in horse, *T. rhodesii* in the ox, *T. callipeda* in dog and *T. leesi* in camel. Allergy to pollen, horse serum etc.
- *Clinical signs*
    - There is congestion of the conjunctiva and increased production of tears which flow over the face as the lachrymal canal may be closed due to the swelling of the membrane .
    - The tears are clear at first but soon become turbid and thick due to the presence of leucocytes and mucoid material. It may also contain flecks.
    - The eyelids may be glued by the sticky material.
- *Gross pathology*
    - Infections by pyrogenic organisms as occurring in distemper of dogs and periodic opthalmia of horses produce purulent conjunctivitis.
    - Croupous or diphtheritic conjunctivitis is mostly encountered in fowls. In cattle infection by *S. necrophorous* causes croupous conjunctivitis. In this condition there is gray or chocolate colored membrane covering the eyeball.
- *Sequelae*
    - Infection may spread to the cornea and keratitis may result.
    - In the purulent and croupous varieties, keratitis and ulceration of the cornea are very commonly seen.

PATHOLOGY OF CORNEA

- Pannus

- Pannus is a condition in which vascular granulation tissue is found between the corneal epithelium and the Bowman's membrane.
- Calcification of the granulation tissue may sometimes occur.

- Keratitis

- *Definition*: Inflammation of the cornea is called keratitis.
- *Etiology*
    - The causes of keratitis are the same as detailed for conjunctivitis.
        - a) Bacteria: Brucella, Listeria and Pasteurella tularensis.
        - Secondary infection by *Staphylococcus aureus, Pseudomonas aeruginosa* and *E. coli* may occur following trauma or debiliting viral diseases.
        - b) Chemicals: Disinfecting fluids, lime in white washing material, irritant gases such as formalin vapour, sulphur, smoke, acids, alkalies, sheep dips, parasiticides, skin dressing, iodism.
        - c) Foreign bodies: Awns, oak husks, mud, dust and sand
        - d) Parasites: *Thelazia lachrymalis* in horse, *T. rhodesii* in the ox, *T. callipeda* in dog and *T. leesi* in camel.
        - e) Allergy to pollen, horse serum etc.
- *Clinical signs*
    - Symptoms include
        - Photophobia and blepharospasm (in which eyelids are tightly closed).
        - Vascularization of cornea

        - Corneal opacity
        - Corneal ulceration
- *Histopathology*
    - Cornea
    - Congestion and edema
    - Infiltration by leucocytes.
    - Ulceration
    - Corneal ulceration
- *Etiology*
    - Corneal ulceration occurs during acute or chronic conjunctivitis.
        - It may also occur as a result of suppurative conjunctivitis or due to trauma (thorns, nails, barbed wire, cat scratches, horn gores).
        - Nutritional imbalance due to deficient proteins and vitamins
        - Impaired nerve supply
        - Virulent organisms can cause ulcers due to the activity of their toxins.
- *Sequelae*
    - Healing of a corneal ulcer is slow and is similar to healing of an open wound. The scar tissue contracts and a tiny scar, which is opaque, is left, which never completely disappears. Depending on its density, the corneal scar is known as nebula, macula or leucoma.
    - If the Descemet's membrane is also perforated, aqueous humor is lost.
    - There may be prolapse of the iris through the rupture (Staphyloma), followed by dislocation of the lens.
    - Secondary infection of an ulcerated cornea can infect the whole globe (panophthalmia) and the eye will be completely lost.
    - Infectious keratoconjunctivitis in cattle (Pink eye)
- *Epizootiology*
    - This condition may occur as an epizootic in many parts of the world
    - It is common in summer and autumn
    - Probably flies transmit the disease from animal to animal.
- *Etiology*
    - The causative organism is *Moraxella bovis* which is gram negative and is found in the tears. An endotoxin that causes necrosis of the skin is produced by this organism.
    - A mild conjunctivitis is also caused by the virus of infectious bovine rhinotracheitis.
- *Clinical signs*
    - Symptoms include
        - Conjunctivitis
        - Copious lachrymation
        - Photophobia
        - Blepharospasm
        - A slight elevation of temperature accompanied by anorexia
        - Within one or two days following the onset of the above symptoms, corneal opacity develops in the centre followed by ulceration in two more days. Ulceration in the young expands and vascularization may occur.
        - The cornea may become completely opaque.
        - There may be purulent discharge from the eyes.

    - A mild conjunctivitis is also caused by the virus of infectious bovine rhinotracheitis and is characterized by congestion of the conjunctiva with increased lachrymation which is serious.
    - Cornea may become slightly cloudy.
- *Sequelae*
    - As the condition subsides, opacity decreases and completely recovery occurs in three to five weeks.
    - Recovery is followed by immunity which lasts for a year. Immunity is local and may be due to persistence of the organism in the conjunctivitis sac.
    - Infectious keratoconjunctivits in sheep – Contagious ophthalmia
- *Synonym*: Pink eye
- *Occurrence*
    - Occurs as outbreaks in various parts of the world in summer.
    - Among goats, the condition is mild.
- *Etiology*: Infectious keratoconjunctivits in sheep is caused by *Rickettsia conjunctiva*
- *Clinical signs*
    - Symptoms include
        - Conjunctivitis
        - Keratitis
        - Increased lachrymation
        - Blepharospasm
        - Opacity of the cornea with vascularization
        - The discharge which is watery at first becomes purulent subsequently.
    - Though the condition is mild and not fatal, animals become temporarily blind and become weak and undernourished as they cannot graze properly and adequately.
- *Sequelae*
    - Recovery starts within three to four days and by the tenth day is complete.
    - In some animals, certain amount of opacity of the cornea remains.
    - Recovery followed by partial immunity and the recovered animals are carriers for a year.

PATHOLOGY OF LENS

- Because of the peculiar structure of the lens, the changes that could occur in this structure are of limited range. These are
    - changes in its position and
    - degenerative or metabolic changes in which the transparency of the structure is altered.
    - Luxation of the lens
- The lens is anchored by the suspensory ligaments to the ciliary body.
- If these ligaments are ruptured, the lens may be displaced into the anterior chamber or into the hyaloid fossa or into the vitreous.
- *Etiology*
    - Luxation of the lens may occur in the following conditions
        - The condition may be a congenital anomaly.
        - Trauma is the most important cause in the acquired variety and found mostly in dogs, especially in sealyheam and terrier breeds. Excessive barking may traumatize the ligaments.
        - Since this condition occurs in older dogs (after the age 3 years) degenerative changes (causes unknown) in the suspensory ligaments may be a cause.

        - A predisposing genetical factor may play a part.
        - Glaucoma which may be the result of cyclitis, may cause secondary luxation.
- *Sequelae*
    - If it is displaced into the anterior chamber, opacity of the cornea occurs due to the pressure of the lens on the endothelium. In such a situation, the nutrition of the lens is altered and it becomes opaque.
    - Glaucoma may develop due to hindrance in the filtration of the aqueous humor (consequent on the abnormal position)
    - Inflammatory changes may be produced by the unnatural position of the lens and so adhesion between the cornea, lens and iris may be brought about.
    - When the capsule of the lens is ruptured, the lens may be liquefied.
    - Cataract
- *Definition*: Opacity of the lens is known as cataract.
- *Occurrence*
    - Cataract is common in dogs and rare in other animals. Among dogs, there is hereditary and breed susceptibility.
- *Etiology*
    - Cataract may be due to the following causes
    - Congenital
        - Failure of the hyaloid artery (which is present in the embryo entering at the optic papilla and extending to the posterior surface of the lens and nourishing the vitreous humor) to regress and disappear completely, leaving remnants of its wall or its small branches.
        - Impairment of translucence of the lens due to abnormal arrangements of the lens fibres or there may be fluid or droplets of fluid between the nucleus and cortex of the lens.
        - A hereditary predisposition may precipitate the occurrence of cataract in later life.
        - Deficiency of vitamin E: Cataracts are found in chicks born of fowls fed vitamin E deficient diets.
    - Acquired
        - Degeneration of the lens due to
            - Trauma
            - Luxation
            - Degenerative ocular disease as in retinopathies and retinal detachment in dogs, as found in Pekingese.
            - Senility- common in old stallions
            - Diabetes mellitus: This is seen occasionally in dogs. Probably increased sugar content alters the osmosis in the aqueous humor.
            - Impaired nutrition, as occurs in opthalmitis and affections of the uveal tract
            - Nutritional disease – deficiency of vitamin D; deficiency of vitamin C in the lens; deficiency of cystein
            - Toxins
                - toxins circulationg in diseases like influenza and periodic ophthalmia in horses and distemper in dogs may cause degeneration of the lens,

        - toxins of uremia as occurs in chronic interstitial nephritis.
    - Poisons: Ergot in cattle and pigs
    - Absorbed radiation
- *Pathogenesis*
    - The lenticular tissue is capable of the following changes:
        - proliferation of the capsular epithelium
        - necrosis of the fibre of the lens and
        - increased sclerosis of the fibres forming the nucleus.
- *Types*
    - Cataract may be partial or complete depending on its situation.
    - It may be congenital or acquired, the former being the most common.
    - Depending on the nature of the lesion, cataract is classified as follows:
        - Subcapsular cataract
        - Cortical cataract
        - Lamellar cataract
        - Nuclear cataract
    - Subcapsular cataract
- This is seen in horses, dogs and birds.
- In this condition there is abnormal proliferation of the lens epithelium. Usually the proliferation occurs at the front surface of the lens, when it is known as anterior polar cataract.
- Sometimes the proliferation of the cells may extend beyond to the posterior surface due to the degeneration of the lens. It is known as posterior polar cataract.
- The cells, because of proliferation become disorganized and form thicker layers producing opacity. This condition may be a result of posterior synechia or due to repeated attacks of periodic opthalmia in horses and is usually associated with cortical cataract.
    - Cortical cataract
- This is the most common form and involves the lens fibres, either at the front (anterior cortical cataract) or at the back (posterior cataract).
- Usually this type follows a corneal ulcer.
- Due to accumulation of interstitial fluid consequent on the altered metabolism of the epithelial cells, the fibres become disintegrated and disorganized.
- The cataract is stellate spreading from the centre to the periphery.
- It is a progressive condition.
    - Lamellar cataract
- This occurs in young animals.
- It may be congenital or acquired and results due to some injury during development and is seen in puppies following an attack of distemper or in those that have suffered from rickets (due to vitamin D deficiency).
- It is non-progressive and is located between the nucleus and the cortex.
    - Nuclear cataract
- Nuclear cataracts are probably the results of senile changes in which the fibres at the centre become denser thereby making the nucleus dull or hazy.
- Sequelae
    - Blindness or impairment of vision develops.
        - **PATHOLOGY OF UVIAL TRACT**
    - Anterior synechia

- Anterior synechia is the condition in which there is adhesion of the iris to the posterior surface of the cornea.
- This results due to iritis.
- Anterior synechia causes glaucoma.
    - Posterior synechia
- Posterior synechia is the adhesion of the posterior surface of the iris to the anterior surface of the lens capsule.
- This condition results due to iritis.
- In this condition pupil cannot dilate.
    - Iridocyclitis
- This is the inflammation of iris and ciliary body and is also known as anterior uveitis.
- This condition in horses is known as periodic opthalmia.
    - Periodic opthalmia or equine recurrent iridocyclitis or moon blindness
- This is a disease of equidae, in which one or both eyes may be affected.
- The disease may abate only to reappear (in the same or the other eye) in a more severe manner causing greater damage to the eye, which finally becomes blind on repeated attacks, hence it is called periodic ophthalmia.
- *Etiology*
    - The exact cause is obscure. The following causes are suggested.
    - Deficiency of riboflavin
    - Infection by *Leptospira*
    - Hypersensitivity to *Leptospira*
    - Virus transmitted by flies.
- *Clinical signs*
    - The disease starts with photophobia, blepharospasm, lachrymation and tightly contracted pupil, which does not dilate even in darkness.
    - The conjunctiva and sclera are congested.
    - The cornea may show vascularization.
    - The iris is dull and yellowish but not the normal brown.
    - The pupil becomes clouded due to the presence of the exudates and leucocytes in the anterior chamber.
    - Particulate matter settles at the lower border of the iris and posterior surface of the cornea forming the keratic precipitates.
    - Within a week or ten days, the disease subsides so the photophobia vanishes and the animal is able to see. Though the disease appears to subside, the eye is never completely normal since some amount of anterior synechia and posterior synechia are present.
    - Repeated attacks cause posterior synechia, subcapsular cataract (due to the alteration in the nutrition of the lens consequent on inflammatory changes of uveal tract), and gradual absorption of the lens after the capsule is ruptured, liquefaction of the vitreous humor, shrinkage and atrophy of the eye.
    - The choroids and retina separate due to accumulation of the exudates.
    - The eye is finally lost.
    -
    - **PATHOLOGY OF IRIS**
    - Mydriasis
- Dilatation of the pupil is known as mydriasis.
- This can be brought about by various drugs like atropine, hyocyamine and stramonium, cocaine, adrenaline and amphetamine.

- Mydriasis may be seen in hypertension, injury to the third cranial nerve, strychnine poisoning and in the later stages of chloroform anesthesia.
    - Myasis
- Constriction of the pupil is known as myasis.
- This can be brought about by pilocarpine, physocarpine and ergotamine.
- Myasis may also be met with in keratisis, ulceration of the cornea, inflammatory conditions of the uveal tract and meningitis.
    - **PATHOLOGY OF RETINA**
    - Progressive retinal atrophy in dogs
- Occurrence : This condition is found in dogs (Irish setters).
- Etiology: It is due to a recessive inherited factor.
- Clinical signs
    - It is manifested in the early stages as night blindness, finally resulting in total blindness.
    - It may take some years even for complete blindness to develop.
- Histopathology
    - The degeneration first starts in rods. Gradually the outer and inner nuclear layers become degenerated and atrophied.
    - The retina in both the eyes becomes atrophied.
    - Detachment of retina
- Retina lies just in contact with the inner surface of the choroids but not attached to it.
- So the retina can be lifted by accumulating edematous fluid or exudates or blood.
- This occurs in inflammatory conditions of the choroids or even in retina.
- Inflammatory exudate pours between the choroids and retina thus separating them.
- Etiology
    - In injuries to the eye, especially thorn pricks in cattle, there may be hemorrhage resulting in the separation of the two structures.
    - In ophthalmitis, organizing exudates may cause traction on the retina which may be detached from its normal position.
    - Liquefaction or loss of vitreous humor may be another cause for detachment of the retina.
    - Retina may be pulled away by the traction of shrinking fibrous band of the vitreous.
- Histopathology
    - When the retina is detached, the rods and cones first become atrophied and disappear, followed by the nuclear layer and the optic fibre layer.
    - Gliosis may also occur.
- Sequelae: Finally cataract, glaucoma and shrinkage of the eye result.
    - **PATHOLOGY OF OPTIC NERVE**
    - Atrophy
- Etiology
    - Congenital
    - Acquired
        - Retinitis, papilledema, retinal degeneration, glaucoma; choroidoretinitis, trauma on occiput, hemorrhages; poisons; morphine, areca nut; deficiency of vitamin A.
    -
- Sequelae

- The optic papillae become thinned with disappearance of the interstitial capillaries.
- Retinal degeneration follows atrophy of the optic nerve fibre.
- Total blindness results ultimately.
- **GLAUCOMA**
- Definition

- Glaucoma is a condition in which there is increased intraocular pressure leading to secondary changes in the eyeball.
  - Etiology
- Increased intraocular pressure may result from a) too excessive a secretion of the aqueous humor or b) hindrance in its drainage.
- It is obstruction to drainage is the most common cause of glaucoma among animals.
  - Primary glaucoma
- If the causes that give rise to obstruction of the flow, leading to glaucoma cannot be determined with certainty, the condition is known as primary glaucoma.
- Probably congenital glaucoma is of this category.
  - Secondary glaucoma
- If the causes for such obstruction can be determined, the condition is known as secondary glaucoma .
- The pathological lesions causing obstruction may be due to
  - Occlusion of the pupil, which may occur as a result of posterior synechia. In this condition due to organization of the inflammatory products, the iris becomes adherent to the lens and so fluid accumulates in the posterior chamber, iris is pressed forward and filtration triangle and the spaces of Fontana are blocked by this protruding iris.
  - Occlusion of the filtration angle by the products of inflammation, which may be acute or chronic. Majority of secondary glaucoma occur this way. So obstruction may occur in iridocyclitis in which anterior synechia occurs; inflammation following trauma of the eyeball - luxation of the lens, intraocular hemorrhages, detachment of the retina and intraocular neoplasms.
  - Clinical signs
- Glaucoma may be unilateral or bilateral.
- The globe is enlarged
- Exophthalmos may be noticed (buphthalmos)
- Cornea may be edematous and opaque.
- Corneal vascularisation and pannus may result due to chronic corneal edema.
  - Histopathology
- The cornea is flattened
- There is opacity of the cornea, the lens and vitreous humor.
- The iris is displaced anteriorly.
- Anterior synechia may occur.
- Degeneration of the lens may occur.
- Blood vessels become sclerosed.

- Atrophy of the choroids to a thin membrane may occur.
- Due to pressure a depression is excavated in the optic disc, which assumes the shape of the cup (cupping of disc).
- The nerve fibres become atrophied. Due to atrophy of the nerve fibres, retina becomes degenerated and atrophied.
- Ganglion cell layer disappears and blindness results.
    - **AURICULAR DEFECT**
    - Definition: Inflammation of the eye ball is called ophthalmitis.

	Disease	**Animal**	**Lesion**
	Mucosal disease	Cattle	Conjunctivitis
	Infectious bovine rhinotracheitis	Cattle	Purulent conjunctivitis
	Infectious canine hepatitis	Dog	Corneal edema
	Rabies	Dog	Negri bodies in ganglion cells
	Canine distemper	Dog	Intranuclear and intracytoplasmic inclusions seen in the ganglion cell layer. Retinitis and optic

			neuritis results in blindness.
○	○ Swine fever	○ Pigs	○ Retinitis and uveitis
○	○ Marek's disease	○ Chicken	○ Iridocyclitis
○	○ Ranikhet disease	○ Chicken	○ Conjunctivitis
○	○ Morexella bovis	○ Cattle	○ Infectious keratoconjunctivitis
○	○ Tuberculosis	○ Cattle, pigs and cats	○ Nodular uveitis
○	○ Coliform infection	○ Calves	○ Purulent ophthalmitis
○	○ *Mycoplasma agalactia*	○ Sheep and goat	○ Corneal opacity
○	○ Rickettsi	○ Sh	○ Infectious

	a ovis	ee p	keratoconjunctivitis
○	○ *Aspergillus fumigatus*	○ Chicken	○ Panophthalmitis
○	○ Trypanosomiasis	○ Horse	○ Diffuse interstitial keratitis
○	○ Dirofilariasis	○ Dog	○ Keratitis
○	○ Thelaziasis	○ Various animals like cattle	○ Mild conjunctivitis

- **NEOPLASMS OF THE EYE**
- Primary neoplasms

- Squamous cell carcinoma, especially in the bovines, is the most common neoplasm.
- Adenomas and adenocarcinomas of the lachrymal gland and Harderian glands may be met with.
- Adenoma and Adenocarcinoma of the ciliary epithelium and iris may occur.
  - Secondary matastases
- Secondary matastases of carcinoma, sarcomas, melanoma, lymphosarcoma, meningioma and the venereal tumor may be met with.

- **POSTMORTEM CHANGES**

- Postmortem changes in the eye includes dryness of the eyeball, pupillary dilatation and detachment of the retina.

- ***DISEASES OF THE YEAR***

- **External ear**

- The external ear consists of the concha, the external auditory meatus and the ceruminous glands.
  - Otitis externa
- *Definition*: Inflammation of the external ear is called otitis externa.
- *Etiology*
  - Foreign bodies like awns may lodge in the ears of dogs and cause irritation and inflammation. The foreign body may sometimes rupture the tympanum.

- Ectoparasites
  - *Psoroptes communis* – causes profuse exudation in to the meatus, which thus contain tenacious brown material. This is seen in sheep more often.
  - *Otodectes cynotis* causes otitis in dogs and cats. Due to irritation the dogs may shake their heads often and this leads, in the long-ear breeds (Dachshand) to hematomas. Secondary infection by bacteria may produce profuse exudates and tympanum may be ruptured.
  - *Otobius megnini* or the spinose ear tick causes otitis in cattle. Though only lymph is sucked by the larvae and the nymphs, secondary bacterial infection of the wounds results in otitis.
  - Fungi that produce dermatomycosis may also cause otitis.
  - *Stephanofilaria zaheeri* causes dermatitis of the ears in buffaloes and may cause otitis.
- Specific disease
  - In swine, *Actinomyces bovis* causes a typical actinomycotic granulomatous condition of the ears, which become thick and indurated. The characteristic symptoms are the presence of thick pus in the external auditory meatus (Otorrhoea) and the thickening of the lining of the meatus.
  - Shaking of the head is an important symptom of the presence of pus in the meatus. Stagnant pus may lead to rupture of the tympanum with subsequent occurrence of otitis media and even otitis interna.

- *Histopathology*
  - The skin becomes much thickened and the sebaceous glands contain eosinophilic material.
  - This is a chronic condition in which there is hyperplasia of the epidermis, hyperkeratosis of the hair follicles and the infiltration of the inflammatory cells.
- *Neoplasms*
  - Neoplasms of the ear are rare
  - Adenoma of the ceruminous glands may occur. Sarcoid in the equines and chondroma and chondrosarcoma may rarely be met with.
  - **Middle ear**
- Middle ear consists of the tympanic cavity, the ossicles and the eustachian tubes. In horse, guttural pouches are diverticula of the eustachian tubes.
- The epithelium lining the tympanic cavity is continuous with the nasal mucosa through the eustachian tubes and so infection from the nose and pharynx can extend into the middle ear.
  - Otitis media
- *Definition*: Inflammation of the middle ear is called otitis media.
  -
- *Etiology*
  - Infection can occur through the external auditory meatus through the eustachian tubes. Normally, there is no communication between the external and middle chambers as, the tympanum seals the passage. But in condition in which there is profuse exudate in the external auditory meatus, ear drum can be ruptured by pressure and infection of the middle ear occurs.
  - Infection can occur via the eustachian tubes from the nasal passages and the pharynx. The organisms found in such cases are *Corynebacterium pyogenes* in

calves, swine and sheep; *Pseudomonas aeruginosa* and Streptococci in swine; *Pasteurella* in cats and staphylococci and *Mycobacterium tuberculosis* in different animals.

- *Sequelae*
  - The inflammatory exudate that accumulates in otitis media, unless drained becomes inspissated and organized, especially around the ossicles, immobilizing them and so deafness may ensure.
  - Other sequelae of otitis media are extension of infection into the inner ear (otitis interna), deafness, paralysis of the 7th cranial nerve, meningitis and encephalitis due to extension of infection into the cranial cavity through the 8 th cranial nerve with resultant death.
  -
  -
  -
  - **NERVOUS SYSTEM**
  - **FUNCTIONAL DISTURBANCES**
  - Loss of consciousness: It results from the effect of various toxic agents upon the brain.
  - Coma
- Definition: Complete loss of consciousness is called coma.
- Etiology: Nervous depression results from pressure upon the brain tumor, or even collection of fluid within the ventricles.
- Clinical signs
  - In this condition, the animal lies outstretched and motionless, its reflexes are gone, the pupils are dilated, respiration is slow and irregular, heart beat weak, and skin cool.
- Sequelae: It usually ends fatally.
  - Nervous depression
- Nervous depression results from pressure upon the brain as in the case of haemorrhage within the cranial cavity, brain tumour or collection of fluid within the ventricles.
- **Etiology** :Equine encephalomyelitis
- **Clinical signs**: There is loss of feeling, sleepiness, and muscular in-coordination.
- Nervous excitement
  - Nervous excitement results from congestion and inflammation of the brain and its coverings.
- Etiology: Rabies
- Clinical signs: There is delirium, mania and convulsions.
  - **NEUROMUSCULAR DISTURBANCES**
- Disturbances in nervous functions also affect muscles in two ways.
- In the first case there is increased muscular activity (spasm) and in the other loss of muscular contractility (paralysis and paresis).
  - Spasms
- In muscle spasms there are sudden, violent involuntary contractions.
- They may be continuous (tonic spasms) or intermittent (clonic spasms).
  -
  -
  - Tremor
- When the spasms are mild and are confined to groups of muscles, they are called tremors .

- Etiology
    - Epidemic tremor (avian encephalomyelitis) of chicks
    - Convulsions
- If the muscle spasms are widespread and involve the whole body, including the limbs, they are called convulsions.
- Etiology
    - They are often seen in puppies infested with ascarids.
    - Epilepsy: When tonic and clonic spasms alternate, and are accompanied by loss of consciousness, they are termed epilepsy.
    - Paralysis: There is complete immobility of a muscle
    - Paresis : Paresis means incomplete loss of motion
- The underlying cause is the defective innervation of the muscle. The defect may lie in the motor centres or in the conduction paths. It prevents the flow of motor impulses and immobility result.
    - Hemiplegia: Hemiplegia is the paralysis arising in the brain cortex and in the peripheral nerves and is unilateral.
    - Paraplegia : A bilateral paralysis of the posterior parts of the body and hind limbs resulting from injury to the spinal cord is called paraplegia.
        - **REACTONS OF NERVOUS TISSUE TO INJURY**
    - Paths of infection
- Infection may reach the CNS by way of the blood stream or the lymph stream or it may pass along the axis cylinders of motor or sensory nerves.
    - Neurones
- Degenerate neurons are normally found in healthy brain but more so in the young animals.
- Since autolysis occurs very early after death in the nervous system it is essential to know how to distinguish between autolytic and degenerative changes.
- The following are the changes noticed postmortem
    - Imbibition of large amounts of fluid giving a spongy, wet appearance to the tissue
    - Neurons and glia shrink, leaving a clear space between the cell and the surrounding parenchyma
    - shrinkage and condensation of the nucleus (Postmortem pyknosis)
    - Fragmentation, fading and disappearance of the nucleus and cytoplasm and Nissl granules
    - Axis cylinders, which are normally unstained, take stain diffusely.
    - Etiology
- The neurons being highly specialized are easily susceptible to injury by hypoxia or toxic materials.
- The following are some of the causes that bring about the degenerative changes in the neurons
    - Inoroganic salts : lead, arsenic.
    - Organic : Anesthetic agents.
    - Metabolic: Toxic products of uremia.
    - Infectious agents : neurotropic viruses.
    - Nutritional deficiency: Deficiency of B1, Copper, Cobal
    - Vascular: Ischemia causing anoxia
    - Effects of vaccination: Allergic encephalitis occurs after vaccination with vaccines containing brain tissue.

- Toxic agents
  - In liver disease, toxic agents, exogenous or endogenous are not detoxified and so these pass on to the brain and produce degenerative changes.
  - Mercury, *lathyrus* and chlorinated hydrocarbons poisoning.
- Lesions

- When a neuron is destroyed, it cannot be replaced.
- When the nervous system is sick and fails to perform its functions, the structural changes may be of three types.
  - Macroscopic alterations.
  - Microscopic alternations, or
  - The changes may be of a biochemical nature, and nothing may be visible even microscopically.
  - The following reactive changes are noticed in the neurons
- Shrinkage characterized by cells becoming very irregular, nucleus pyknotic, clumping and condensation of Nissl substance and tortuousness or sclerosis of the processes. This is seen in senility and chronic infections.
- Swelling of the nerve cells: Here the cytoplasm stains very faintly and only the cell outline may be discerned with fragmentation of the processes. This is a reversible condition and occurs in severe intoxications and systemic infections.
- Vacuolation of the nerve cells may be seen in toxic conditions and in viral encephalomyelitis.
- Chromatolysis: in this condition the Nissl substance becomes fine and dispersed and later may disappear. The nucleus may be eccentric. This change is seen in injury to the axon. It is suggested that the Nissl substance being a ribonucleoprotein is actively involved in the synthesis of axoplasm and so chromatolysis may denote an exhaustion phase. When axon regenerates and is repaired, Nissl substance is restored. In viral infections and other severe intoxications chromatolysis occurs when the Nissl substance disappears. The cytoplasm also shows degenerative changes: swelling and rounded contours. Unlike in the axonal trauma, in viral infections and injury to central axons chromatolysis is irreversible since the neurons are destroyed.
- Neuronophagia: when the nerve cell dies, microglia and oligodendroglia invade the cell and remove it by phagocytosis.
- Satellitosis: Normally every neurone has one or two oligodendroglia near them. They are called satellite cells in this location. Whenever a neurone is damaged, oligodendroglia and microglia crowd around such cells, without actually invading them, and this phenomenon is known as “satellitosis”. Both satellitosis and neuronophagia are indications of necrosis of a neurone.
- There is destruction of myelinated tracts in the white matter in allergic encephalitis.
- In mercury poisoning, neuronal degeneration and necrosis of the brain and demyelination of the nerve tracts extending to the spinal cord are noticed.
- In *lathyrus* poisoning there is degeneration of neurons in the spinal cord accompanied by gliosis and ultimate atrophy of the spinal cord.
- In poisoning by chlorinated hydrocarbons, there is Nissl degeneration and necrosis of neurons, especially of the ganglia and brain stem.

  - **REACTION OF GLIAL CELL TO INURY**

  - Astrocytes

- Astrocytes are star shaped, are the supporting cells found throughout the central nervous system.

- These cells react to injury and proliferate and this process is known as " gliosis ", which may be uniformly diffuse or may be focal.
- When nervous tissue is destroyed, repair does not take place by fibrosis. The fibrous tissue from the adventitia of the blood vessels may repair sometimes. But, if a cavity arises due to softening and absorption of the brain, it is not filled in but a cyst is formed with a thin fibrous capsule around which the astrocytes proliferate.
- Inclusion bodies may be present in the astrocytes also (gemistocytes in Canine Distemper).
    - Oligodendroglia
- Oligodendroglias are the cells containing dark round nuclei and are found mostly in the white matter, in long rows between the fibres.
- They may also be found in small numbers as satellite cells around the nerves and blood vessels.
- In places where medullated nerve fibres are destroyed oligodendroglia are found to disappear.
- The cells invade the dead neurons and engulf them (neuronophagia).
    - Microglia
- Microglial cells are the phagocytes of the central nervous system and are found in both white and gray matter.
- With ordinary stains, only their oval dark nuclei are noticed. Their branching cytoplasmic processes require special stains to be seen.
- Microglia is amoeboid and phagocytic and become hypertrophied and proliferates. Hypertrophied cells that engulf the dead tissue are rounded and the cytoplasm is foamy containing lipids and are called " Gitter cells " (from Gitter –Zellen, German), "Compound granular corpusles" or "fat granule cells".
- Often these gitter cells migrate to the perivascular spaces (space of Vichow-Robin).
    - The meninges
- The coverings of the central nervous system consist of the dura and pia-arachnoid.
- The dura in the cranium is attached to the cranial periosteum, in the spinal column it is separated widely from the vertebral periosteum.
- Pia closely follows the brain and is separated from the arachnoid by the subarachnoid space. The space contains the cerebrospinal fluid and has the spongy network of the arachnoid trabeculae.
- The meninges are composed of cells that are mesodermal in origin. Their reaction to injury is by inflammation and fibrosis.
    - Blood vessels
- The blood vessels of the brain have some peculiarities. These are
    - The arterioles and venules are very thin walled, devoid of elastic and muscular tissue.
    - Veins do not have valves
    - The blood vessels acquire a menigothelial sheath as they pass through the subarachnoid space and a second outer sheath derived from the pia.. So a perivascular is formed between these sheaths- the space of Virchow-Robin, which is continuous with the perinerual and interstitial space of the C.N.S.
- It is in this perivascular space that the cells accumulate and give rise to " perivascular cuffing ". Depending on the nature of the pathogen, the cells vary. In bacterial infections of the brain, inflammatory exudate collects in the space of Virchow-Robin.
- The adventitial wall is the source of macrophages and the fibrous tissue elements that compose the capsule in abscesses that arise in some bacterial infections.
    - Blood- brain barrier

- Histologically, the blood brain barrier is composed of the vascular endothelium of blood vessels, the basement membrane, and the perivascular glial membrane which is the close application of the cytoplasmic foot processes of innumerable astrocytes to the endothelium of the capillaries.
- The blood- brain barrier is essentially a defence mechanism against noxious agents.
- The brain effectively prevents a wide variety of toxic substances of large molecules from reaching the brain.
- It successfully prevents the entry of most bacteria and viruses from the circulating blood into the brain.
- It is only when certain toxic substances, including bacteria and viruses, break down the barrier that they are able to enter into the brain tissue and set up the infection, or induce other pathological changes.
    - Cerebrospinal fluid
- Cerebrospinal fluid is found in the ventricles, spinal cord and the subarachnoid plexus in the lateral ventricles.
- In infections, C.S.F may show considerable changes. It may become cloudy or even bloody and the globulin content may be very high.
    - **CONGENITAL ANOMALIES**
- Anencephaly is the absence of most of the brain, and is seen in most animal species
- Acrania is the complete failure of cranial development
- Amyelia is the absence of spinal cord.
- Cranioschisis in congenital fissure of the cranium
- Encephalocele is the protrusion of meninges, alone or with part of the brain through a defect in the cranium.
- Microcephaly is the presence of an abnormally small brain.
- Meningocele is hernia of the meninges, which protrude through an opening of the skull or spinal column.
- Rachicele is hernia of the spinal cord.
- Spina bifida is a congenital defect in walls of spinal canal caused by lack of union between the laminae of the vertebrae. Through this defect the spinal cord may herniate. The condition has been reported in cattle, dogs and sheep. Sometimes there is no protrusion and swelling to indicate the defect. This means the condition is hidden and is then called spina bifida occult .
    - **DISTURBANCES OF CIRCULATION**
    - **HYPEREMIA**
- Hyperemia may be acute or chronic.
    - Acute general active hyperemia
- Acute general active hyperemia is present, when bacterial or viral diseases affect the entire central nervous system as in rabies, viral equine encephalomyelitis and hog cholera.
    - Acute focal active hyperemia: Acute focal active hyperemia is seen in near the infarcts, abscesses and tumours.

- Chronic general passive hyperemia

- This occurs when there is a passive hyperemia due to lesions in the heart or lung or an obstruction to the flow of blood from the brain such as thrombosis of both jugular veins.
- Histologically, there is increased number of glial cells throughout the brain and spinal cord, which indicates chronicity of the condition.
  - Chronic focal passive hyperemia
- This occurs when a tumour or abscess presses upon a vein, or a thrombus forms within a vein causing a reduction in the flow of blood from a local area of the brain.
  - **A**

- Anemia may be general or local.
  - General anemia
- This occurs as when anemia involves the entire individual.
- This may be seen in parasitic anemia as in gasterointestinal parasitism, excessive hemorrhage and in anemias associated with deficiency of iron, copper and the vitamin B complex.
- The brain and spinal cord are whiter than normal, and the blood vessels contain decreased amount of blood and are therefore less prominent.
- Histologically, areas of liquefactive necrosis as well as gliosis and neuronal degeneration may be present. These result from oxygen deficiency.
  - Local anemia or ischemia
- This occurs from a deficiency of arterial blood in a local area of the brain or spinal cord.
- This may be caused by thrombosis and embolism.
- Thrombosis and embolism of cerebral arteries are rare in animals and may occur in the brain and spinal cord.
  - Emboli may be
    - detached vegetations from the cardiac valves or may arise from lesions of the lungs, left atrium or coronary artery,
    - clumps of bacteria
    - tumor cells
    - parasites (larvae of ascaris, onchospheres of tapeworms, young trichinella etc).
    - agglutinated erythrocytes.
  - Thrombus can arise from lesions of cerebral vessels (atheroma) or can occur in diseases that damage the vascular endothelium: - trauma causing fractures of the skull, invasion of the vessel wall by neoplastic cells, abscesses and hog cholera.
  - If the collateral blood supply is inadequate, infarction results. The infarcted area is pale or red depending upon the blood supply. The infarcted area is liquefied to form a cyst.
  - **HEMORRHAGE**

- Petechiae
- Petechiae are common in acute septicemic diseases (Anthrax, Hemorrhagic septicemia, Hog cholera, Leptospirosis) or in infections by pyogenic organisms.
- These also occur after thrombosis or in degeneration of the vessel walls or in general hemorrhagic diseases as in bracken fern poisoning.
- Hyperemia of the brain and meninges together with petechial hemorrhage and edema are found in the following conditions
    - Electrocution,
    - Lightning stroke and
    - Sunstroke
    - Rupture of an artery
- Rupture of an artery will give rise to large areas of hemorrhage with clots causing apoplexy.
- Rupture may occur in injuries – automobile accidents, gun-shot wounds, diseases of wall of blood vessels (atheroma) with hypertension as in arteriosclerosis, chronic-nephritis, bursting of an aneurysm as in parasitic aneurysm in horses.
    - Clinical signs
- The first symptom in cerebral hemorrhage is shock, later passing on to coma and terminating in death.
- Animals that survive the first shock suffer from some degree of paralysis due to pressure on and damage to neurons.
    - Gross pathology
- Hemorrhages may be found subdurally or epidurally.
- They may also occur in the substance of the brain.
- When hemorrhage is present in the ventricles, the cerebrospinal fluid may be blood tinged.
- The blood clot in the brain first contracts separating the serum which is absorbed. The clot that remains is liquefied and a cyst is formed with a clear fluid – the " apoplectic cyst ".
    - Histopathology: The capsule of the apoplectic cyst is formed by the neuroglia.
    - **EDEMA OF BRAIN**
- Edema of brain may be focal caused by local lesions such as
    - Trauma accompanied by hemorrhage and laceration
    - Cerebral and meningeal hemorrhages
    - Meningitis
    - Focal necrosis
    - Neoplasms
    - Generalised edema of brain
- Generalised edema of brain may be found in
    - Causes that give rise to general edema of the body
    - Enterotoxemia caused by *Clostridium welchii*
    - Viral encephalitis
    - Salt poisoning in pigs
    - Poisoning by Lead, organic mercury compounds and ANTU
    - Sunstroke
    - Shock
    - Diffuse meningitis
    - Gross pathology
- Macroscopically

    - The brain appears more moist and heavy.
    - The gyri are widened while the sulci are narrowed.
    - Swollen gyri that press against the skull appear flattened.
    - On section, the gray matter appears wider while the internal white matter is softer. The ventricles appear narrowed.
    - Histopathology
- Microscopically
    - The white and gray matter appears to have a loose texture and the interfibrillar space is widened.
    - Neurones and glia appear swollen.
    - Edematous fluid accumulating around the space of Virchow-Robin (perivascular space) widens these areas.

HYDROCEPHALUS

- Definition
- Hydrocephalus is a condition in which there is abnormal accumulation of cerebrospinal fluid in and around the brain.
    - Etiology
- In animals this is a congenital condition due to some error in development, obstructing the pathways of fluid passage.
- Vitamin A deficiency during intrauterine life may cause internal hydrocephalus in calves and pigs.
    -
    - Types
- If the accumulation is in the ventricles, the condition is called internal hydrocephalus.
- But if fluid accumulation occurs in the sub-dural space or pia-arachnoid, it is called external hydrocephalus.
    - Internal hydrocephalus
- It can arise whenever there is obstruction to the free passage of the cerebrospinal fluid. The obstruction can occur at the formation of Monro, the aqueduct of Sylvius or the formina of Luschka. Cysts (hydatids and coenurids), tumors or inflammatory exudates are the usual causes for the blockage.
- Congenital narrowing of the lumina may also be a contributory cause.
- *Gross pathology*
    - Due to pressure of the accumulating fluid the ventricles dilate and the adjoining nerve tissue atrophies.
- *Sequelae*
    - If hydrocephalus develops before the cranial sutures fuse, the cranial bone may grow to a large size.
    - The cranium is greatly enlarged causing foetal dystocia.
    - External hydrocephalus
- Usually, the external hydrocephalus is “acquired”. It results due to either too much fluid formed and not rapidly drained by the arachnoid villi or to hindrance to the drainage of normally produced fluid as occurring in congenitally constricted tentorial aperture.
- It is usually the result of rupture of the thin dorsal wall of the third ventricle which allows the fluid to escape into the subarachnoid space between the cerebral hemispheres and the cerebellum. Hence, the external hydrocephalus is called the “ communicating hydrocephalus ” which is less common than the internal variety.
- *Gross pathology*
    - The accumulated fluid exerts pressure on the surface of the brain.

- So there is a general atrophy of the brain and widening of the sulci between the convolutions.

- *Sequelae*
  - The result of hydrocephalus is pressure atrophy of the surrounding nervous tissue causing depression, in-coordination, ataxia and death.

- **CONGESTION OF BRAIN**

- Congestion of brain may be seen in heat stroke, poisoning (nervous) and systemic infections.

- **DISTURBANCES OF GROWTH**
  - Aplasia: Aplasia of portions of the brain and spinal cord are observed in young animals.
  - Hypoplasia

- This is relatively more common than aplasia.
- *Etiology*
  - Hypoplasia is seen in congenital posterior paralysis in calves and swine, spastic paresis in cattle.
  - Cerebellar hypoplasia

- This anomaly is seen in calves and cats mostly but may also be found in other animals. In the Jersey calves and cats, this defect is inherited.
- Cerebellar hypoplasia was encountered in calves born of cows which were affected with virus diarrhea-Mucosal Disease while pregnant. Similarly modified hog cholera virus causes, cerebellar hypoplasia in the fetal pig.
- It is also eported in dogs, lambs and goats.
- *Gross pathology*: At necropsy, cerebellum may be found to be rudimentary or even absent.
- *Histopathology*
  - Microscopically, molecular and granular layers are reduced in size. There is a relative reduction in Purkinje cells.

- *Sequelae*
  - Animals may usually die shortly after birth.
  - Animals that survive for a short while, show locomotor disturbance and in-coordination.
  - Hypertrophy

- This may result from increase in size of the glial cells, microglia showing the greatest degree of hypertrophy.
- The neuron does not increase in size.
  - Hyperplasia

- This results from an increase in the number of glial cells. Glia, especially the microglia, increases in number in case of hypoxia.
- Hyperplasia of the neurons does not occur.
  - Metaplasia

- This does not occur in the nervous tissue proper.
- It may occur in the connective tissue of the meninges and blood vessels, in which case cartilage and bone may be found.
  - Atrophy

- Atrophy of the cerebrum may occur in hydrocephalus.
- Pressure atrophy may also occur in the vicinity of tumors, abscesses, haematocysts and depression fractures of the skull

- **DISTURBANCES IN CELL METABOLISM**
  - Cloudy swelling

- Etiology
    - The neurons and the glia undergo cloudy swelling as a result of hypoxia produced by toxic substances or infectious agents.
- Histopathology
    - The cells become larger, cellular outline more round and cellular structures indistinct.
    - Fatty degeneration: This appears as fat droplets in the cytoplasm of the neurons.
    - Hydropic degeneration
- It is a continuation of cloudy swelling. In this droplets of edematous fluid are observed in the cytoplasm of the neurons and glia.
    - Amyloid infiltration: This is uncommon in the central nervous system of most domestic animals.
    - Glycogen infiltration: This does not occur in the central nervous system.
    - **Pigmentation**
- In cattle and sheep, melanin is most frequently encountered in the pia mater of the anterior one-fourth of the brain.
- Focal areas of melanin may be found in other portions of the meninges and even within the brain and spinal cord.
    - **Calcification**
- This is most commonly found in meninges than in the brain and spinal cord proper.
- It occurs in the presence of dead tissue and faulty circulation, examples being abscesses, infarcts, parasitic lesions, sites of old hemorrhage and in necrotic neurons.
    -
    - **TRAUMATIC INJURY TO THE NERVOUS SYSTEM**
- The brain being soft, is susceptible to shock that emanates from impact, especially from fast moving objects.
- A sudden blow on the cranium may result in fracture of the cranial bones which may not be depressed.
- Fracture of the skull causes considerable damage to the meninges and brain. Hemorrhage may occur and nerve fibres disrupted.
- Hemorrhage aggravates the condition by pressure of the accumulated blood on the brain tissue.
- On the other hand a blow on the vertebral column results in fracture or dislocation.
    - Concussion
- Concussion occurs when the skull receives a sharp blunt blow suddenly, not accompanied by fracture.
- Gross pathology
    - Lesion consists of small hemorrhage in the brain and under the skin at the site of injury.
- Sequelae
    - There is loss of consciousness.
    - The condition is not fatal. Recovery is the rule.
    - Laceration
- In this condition there is discontinuity of the tissue and usually occurs in automobile accidents.
- Blunt objects may cause laceration and a contrecoup laceration occurs on the brain on the side opposite to that on which the injury is struck. This is due to striking of the brain on the skull on the opposite side, since normally the brain is smaller than the cranium and is slightly movable. In such places hemorrhages are common.

- Penetrating wounds, usually caused by gun-shot wounds, are followed by severe hemorrhage. Fractures are also common in such injuries. Penetrating wounds are usually followed by secondary infections and are fatal.

NECROSIS

- Coagulative necrosis

- This involves the neurons and the glia.
- *Etiology*
  - The causes are severe injury to the cells brought about by hypoxia, chemical poisons, bacterial toxins and viruses.
- *Gross pathology*: No changes are seen macroscopically.
- *Histopathology*
  - Microscopically, the cells are swollen and become more globular in shape.
  - The cytoplasm stains more intensely with eosin.
  - The Nissl substances may eventually disappear (chromatolysis or tigrolysis).
  - The nucleus shows pyknosis, karyorrhexis, or karyolysis.
  - Microglia accumulates around the necrotic neurons, the process being known as satellitosis. When the microglia phagocytose the necrotic neuron, the process is called neuronophagia.
  - Liquefactive necrosis
- Necrosis of the brain is almost always liquefactive in nature.
- *Etiology*
  - Infarction is one of the common causes of liquefactive necrosis.
  - It may also occur when the central nervous system is invaded by pyogenic bacteria.
  - Encephalomalacia is commonly seen in the following conditions
  - Defeciency of Vitamin E in young chickens (crazy chick disease); moludy corn poisoning in horses (cornstalk disease); Acute pancreatitis in all animals; Antenatal copper deficiency in lambs (sway back); cobalt deficiency (Enzootic marasmus); Enterotoxemia in lambs; Mulberry heart disease in swine; Vitamin B deficiency (Chastek paralysis) in fur- bearing animals and in calves and sheep, when it is called cerebro-cortical necrosis ; Blue tongue in sheep; Rift valley disease of Kenya; Distemper of dogs, toxoplasmosis and lead poisoning; Infarction due to an embolus consisting of tumor cells or parasites or a piece of a thrombus or due to thrombosis of an artery; poisoning by mercuric salts.
- *Pathogenesis*
  - This is the most common type of necrosis encountered in the brain and spinal cord compared to coagulative and caseous type because the nervous tissue contains little coagulable albuminous material but is rich in lipoids.
  - When necrosis occurs in the nervous system, the autolytic enzymes released from lysosomes of the dead cells cause disintegration of myelin into a liquid mass that consists mainly of lipoid.
  - The lysosomal enzymes released from neutrophils induce liquefaction of myelin, neuroglia and other structures, and is known as encephalomalacia . Softening of gray matter is known as poliomalacia and that of white matter leucomalacia .
- *Histopathology*
  - The lesions seen are thickening of blood vessels, endothelial hyperplasia and liquefaction of brain substance. Thrombosis and hemorrhage may be found some cases. Around the area, there may be proliferation of capillaries and the

formation of a capsule by the cells of meninges. Astroglia proliferate and surround the area of encapsulation. The involved tissue undergoes liquefaction and a serous fluid is present.
    - Caseous necrosis
- *Etiology*
    - The cause of this type of necrosis is infection of the brain by *Mycobacterium tuberculosis*.
- *Gross pathology*
    - The necrotic area is seen as a dry, crumbly, yellowish-white mass.
    - It may contain areas of calcification.
- *Histopathology*
    - Microscopically, all cellular or architectural structures are lost.
    - The necrosed area is surrounded by a zone of inflammation.
    - Necrosis of nerve fibres of the peripheral nerves, the tracts and central nervous system is first indicated by fatty degeneration of the myelin sheaths of the nerve fibre affected. This change occurring in the brain and spinal cord is called demyelination. Ultimately the axon may disappear.
- *Sequelae*
    - Demyelination alone can render the nerve fibre non-functional.
    - If demyelination alone has taken place, regeneration is possible with restoration of function.

    -
    -
    -
    - **GANGRENE**
- This could occur if the brain is invaded by saprophytic microorganisms, as in the case of traumatic injuries of the skull or as septic emboli from areas of gangrene in the lungs.

    - **INFLAMMATION**
    - Terminology of inflammation affecting the nervous system
- Encephalitis – is inflammation of the brain.
- Myelitis – is inflammation of the spinal cord.
- Encephalomyelitis – is inflammation of the brain and spinal cord.
- Meningitis – is inflammation of the meninges.
- Pachymeningitis - is inflammation of the dura matter.
- Leptomeningitis – is inflammation of the pia matter.
- Meningoencephalomyelitis – is inflammation of the meninges, brain and the spinal cord.
- Poliomyelitis – is inflammation of gray matter in the spinal cord.
    - The same general laws of inflammation apply to the brain and spinal cord as elsewhere.
    - Since there are no mucous membranes, catarrhal inflammation does not occur.
    - Serous inflammation also probably does not occur: If it does it resembles edema.
    - Hemorrhagic exudates are rarely met with and fibrinous inflammation is limited practically to the meninges.
    - Purulent, lymphocytic and proliferative inflammations are the types which are regularly encountered in the central nervous system.
    - Types of inflammation

- *Fibrinous encephalitis, myelitis and meningitis*
    - These are seen in cattle and sheep during *Pasteurella* infection of the central nervous system.
    - They are characterized by cardinal signs of inflammation and increased fibrin content in the sub-arachnoid and Virchow-Robin space.
- *Suppurative (purulent) encephalitis, myelitis and meningitis*
    - These are observed in all species of animals.
- *Routes of infection*
    - By direct extension from suppurative conditions of the middle ear, nasal passage, cribriform plate or from meninges.
    - Through blood stream (in septicemic disease) and lymphatic vessels accompanying nerves (Listeriosis).
    - Infection of penetrating wounds of the skull.
    - Suppurative myelitis will result due to infection of the wound made while docking the tail.
- *Etiology*
    - The pyogenic organisms responsible are Staphylococci, Streptococci, Corynebacterium, Pasteurella, Listeria and pleuroneumonia like organisms.
- *Gross pathology*
    - There are the usual cardinal signs of inflammation.
    - The principal constituent of the exudates is pus.
    - The inflammation may be focal or diffuse.
- *Histopathology*
    - The lesions may be microscopic and consists of focal collection of neutrophils and lymphocytes.
    - The abscesses do not have well developed capsules as mesodermal cells that form it are few. Astroglia proliferate and form a poorly defined capsule around the cerebral abscess.
- *Sequelae*
    - The accumulation of pus causes pressure and destruction of the local tissue.
    - If an important area is involved, severe effects follow.

    - **LISTERIOSIS (LISTERELLOSIS)**

- This is the most frequent cause of a purulent reaction in the brain of farm animals.
- Infection by *Listeria monocytogenes* produces suppurative meningoencephalomyelitis in cattle, sheep and goats.
- The disease is characterized by the presence of multiple microabscesses which contain the organisms.

    - **LYMPHOCYTIC MENINGENCEPHALOMYELITIS**

- This is the most important form of inflammation of central nervous system in animals.
    - Etiology
- This type of inflammation is caused mainly by viruses. The viruses may be
    - Neutrotropic: The virus affects almost only the nervous system. Examples of these are the Rabies in dogs and Borna disease in the horses.
    - Organotropic: The viruses that affect other tissues may also infect the nervous tissue by chance. Examples: Canine distemper; Hog cholera; Epidemic tremor of fowls; Malignant catarrhal fever and Rinderpest.
- Others that may produce encephalitis are the casual agents of psittacosis and ornithosis, PPLO, rickettsia and trypanosomes.
- Allergic: Post-vaccinal encephalitis with vaccines containing nerve tissue.

- Routes of entry
- Blood stream – Hog cholera
- Nerves – Rabies
- Neurolymphogenous – infection ascending along the lymph pathways of cerebrospinal nerves.
    - Pathogenesis: The virus entering a neurone kills it.
    - Gross pathology
    - No gross lesions of significance are noticed.
    - Hyperemia and edema of the pia-arachnoid may be seen.
    - Occasionally localised areas of softening may be found.
    - Petechiae also may be found
    - Histopathology : the following are the characteristic features noticed:
    - In this the lymphocyte is the principal constituent of the exudates.
    - Congestion and hemorrhages
    - Cuffing of blood vessels: i.e. accumulation of lymphocytes in the space of Virchow-Robin. The cells being trapped in the Virchow-Robin spaces as they leave the vessels is called perivascular lymphocytic infiltration or perivascular cuffing . Later plasma cells and macrophages may also be found in these places. This lesion is found in the white and grey matter.
    - Edema
    - Gliosis: diffuse proliferation of the astrocytes throughout the brain giving the tissue a dense and cellular appearance.
    - Satellitosis – appearance of scavenger cells or " Hortega cells " of " Gitter cells " around the necrotic area. These are the microglia and oligodendroglia which remove the dead neurons and debris by neuronophagia . The gitter cells contain lipid and are the only cells seen in the area where neurons were situated previously. Sometimes in more chronic cases, the microglia increase in number, their nuclei become elongated and their cytoplasm contains iron deposits and such cells are known as " Rod cells "
    - Neuronophagia.
    - Demyelination of the nerve fibres may be present.
    - Rarely proliferation of blood capillaries may be seen
    - Inclusion bodies may be found in the neurons or astroglia in a number of diseases; rabies, canine distemper, infectious canine hepatitis, Borna disease
        - **SPECIFIC INFLAMMATIONS OF BRAIN AND SPINAL CORD**
    - Rabies
- It is an acute viral disease of domestic animals characterized by a very severe lymphocytic inflammation of nervous system.
- There is diffuse and severe meningoencephalomyelitis.
- A characteristic feature of the disease is the presence of intracytoplasmic inclusion bodies in the cells of the hippocampus and cerebellum.
    - Pseudorabies
- It an infection viral disease of cattle, pigs, dogs and cats.
- In pigs, there is a diffuse lymphocytic meningocephalomyelitis which does not seem to occur in cattle.
    - Hog cholera (swine fever) encephalitis
- In hog cholera 80 to 90 per cent of the animals suffer from an acute diffuse lymphocytic meningocephalomyelitis.
    - Canine distemper encephalitis

- The virus of canine distemper also produces a typical diffuse lymphocytic meningoencephalomyelitis.
- However, not all the affected dogs develop lesions in the central nervous system.
    - Infectious viral equine encephalomyelitis
- This is an acute viral disease of horses and mules and is also characterized by a typical diffuse lymphocytic meningoencephalomyelitis.
- It terminates fatally in about 50 percent of the cases.
    - Borna disease
- This is an acute diffuse viral meningoencephalomyelitis of the horses that occur in Europe, especially in Germany.
- It has not been reported in India.
    - Louping ill
- This is an acute diffuse viral lymphocytic meningoencephalimyelitis of sheep in Scotland, England and Ireland.
    - Avian viral meningoencephalomyelitis or Epidemic tremors
- It is an infectious viral disease of chicks.
- Microscopically, the disease is characterized by a diffuse lymphocytic inflammation of the entire central nervous system.
    - Ranikhet disease (Pneumoencephalitis of poultry)
- It is a viral disease of chickens which is also characterized microscopically by diffuse meningoencephalomyelitis.
    - Tuberculosis meningoencephalomyelitis
- The lesion in the central nervous system consists of a central area of caseous necrosis which may be partially calcified.
- This indicates that generalized tuberculosis is present.
    - Enterotoxaemia: In enterotoxaemia of sheep, there is perivascular edema and haemorrhage.
    -
    - **PARASITIC ENCEPHALOMYELITIS**
    - **MYIASIS**
- *Hypoderma bovis* larvae may be found in the fat of the vertebral canal in cattle. At times they may invade the spinal cord or the brain.
- The larvae of *Oestrus ovis* have also been reported to invade the brain.
    - **CESTODIASIS**
- Tapeworm cysts are found in the central nervous system of the domestic animals.
    - Etiology: Cysts of *Multiceps multiceps*, *Taenia pisiformis* and *Tanenia echinococcus*.
    - Pathogenesis
- The ova of these tapeworms are ingested by the animal. In the intestine, the hexacant embryo comes out, pierces the intestine, and is carried by blood stream to various places in the body.
- Some of the larvae, particularly those of *Multiceps multiceps*, reach the brain where they encyst.
    - Gross pathology
- The path of migration of the larvae in the brain is macroscopically visible as red streaks due to the presence of hemorrhage.
    - **GID OR STURDY**
- The larval stage of *Multiceps multiceps*, a dog tapeworm is known as coenurus cerebralis .

- It causes, a rather uncommon disease of the central nervous system of sheep, known as “gid” or “sturdy”.
    - Clinical signs
- The symptoms depend on whether the bladder worms are located in the brain or spinal cord.
- Cysts usually affect the lumbar portion of the spinal cord, which results in in-coordination and paralysis of the posterior extremities.
- In acute diffuse lymphocytic meningoencephalomyelitis, the animal dies.
    - Gross pathology
- In both brain and spinal cord, they form cysts reaching 50 mm in diameter or more. Each cyst is filled with clear fluid and contains even up to 500 scolices.
- As the cyst enlarges there is pressure atrophy of the surrounding nervous tissue. So the convolutions may be flattened and cortex becomes thinned. Even the cranial bones may be subjected to pressure atrophy and some may be punctured even.
    - Histopathology
- In severe infection, an acute diffuse lymphocytic meningoencephalomyelitis is produced.
- The chronic irritation induces a chronic lymphocytic meningitis, encephalitis, or myelitis, depending on the location of the parasite.
    - Sequelae: Death of the larvae will result in calcification of the cysts.
    - **NEMATODIASIS**
- Various nematode larvae are found in the central nervous system. Strongyle larvae are found in the horses. The larvae of ascarids, strongyloides, hookworms and microfilaria of*Dirofilaria immitis* may be found in the capillaries or in the nervous tissue of the brain, spinal cord or meninges. Their lesions are characterized by a chronic lymphocytic inflammation.
    - **Cerebrospinal nematodiasis (Neurofilariasis; Kumri)**
    - Occurrence: The disease occurs in India, Srilanka, Burma, Korea and Japan.
    - Etiology: This is found in sheep, goats and horses and is caused by *Setaria digitata.*
    -
    - Synonym
- In horses it is known as Kumri (Hindustani for “weakness of the loin”) in our country, whereas in sheep and goats it is known as “ lumbar paralysis ”.
    - Pathogenesis
- *Setaria digitata* is a natural parasitic of cattle.
- But when the microfilaria finds entry into heterologous hosts, like sheep and goat, they wander away to the central nervous system.
    - Clinical signs
- Symptoms are basically neuroparalytic and include motor weakness, in-coordination and loss of balance.
- Severe cases exhibit paralysis of one or all limbs. The hind limbs being most frequently and severely affected.
    - Gross pathology
- Macroscopically, lesions are found in the brain and spinal cord and the severity depends on the number of the parasites present.
- Narrow tortuous tracks of hemorrhage and softening may be found denoting the path taken by the parasite.
    - Microscopically,

- The lesions consist of a central space surrounded by a degenerated and necrotic tissue.
- The necrosis is liquefactive in type.
- Hemorrhages may be present in this area.
- Due to damage by the larvae the axis cylinders in the affected area are swollen and degenerated and appear enlarged and fragmented.
- The myelin sheath becomes swollen and distorted, accompanied by glial proliferation.
- Lymphocytes, eosinophils and microglia infiltrate around the area.
- Perivascular cuffing of nearby vessels is observed.
- The larvae may not be visible in these lesions as they might wander off.
- Careful microscopic examination of the tissue and cerebrospinal fluid is necessary to see the larvae.
- Sequelae: The causes may terminate fatally, or recovery may follow.
- **WOBBLES**
- Occurrence

- Cerebrospinal nematodiasis is known as "wobbles" among young horses and mules.
- Animals of one to two years of age are affected.
  - Other etiological factors
- Trauma of the spinal cord at the cervical region is suspected to be the cause some authors.
- Weakness inherited genetically is also suggested.
  - Clinical signs
- Suffering animals move with difficulty and on motion sway (or wobble) from side to side. They may fall frequently and show difficulty in rising.
  - Gross pathology
- Lesions are found only in the cervical portion of the cord where tracts of hemorrhagic necrosis and bilateral symmetrical areas of malacia are noticed.
- In some cases, through parasites may be found in the central nervous system neither symptoms nor lesions are observed.
  -
  -
  - Histopathology
- Microscopically, liquefactive necrosis, haemorrhage, degeneration and demyelination of the peripheral nerve tracts, perivascular cuffing, satellitosis, neuronophagia and gliosis are observed.
  - **TOXOPLASMOSIS**
- *Toxoplasma gondi* , a protozoan parasite is found in the central nervous system of domesticated animals.
- Lesions consist of a central area of coagulative necrosis, surrounded by microglia and neutrophils. There is also lymphocytic meningitis, lymphocytic perivascular cuffing and gliosis.
  - **ALLERGIC ENCEPHALITIS (POSTVACCINAL ENCEPHALITIS)**
- This sometimes occurs in dogs following rabies vaccination.
- It occurs 2 to 3 weeks after vaccination.
- There is motor paralysis of one or more limbs, which may later involve most of the body.
- It is characterized by a lymphocytic meningoencephalomyelitis.
- Death is the usual outcome.

- **CONGENITAL MYOCLONIA OF PIGS (TREMBLES)**
- Definition: It is characterized by clonic convulsive movements due to hyperirritability of muscles.
- Occurrence: New born pigs are affected by this condition.
- Clinical signs

- Clonic convulsions
- After a few weeks of affection, the symptoms may disappear and the pigs may then thrive.
- In severe cases they die in inanition as the piglets are unable to suckle properly.
  - Etiology
- The following causes are suspected: heredity factors, hypothyroidism, virus infection, nutritional deficiencies of the sow during pregnancy.
  - Gross pathology
- No visible changes may be noticed.
- Edema, thickening and hemorrhage in the cerebellum may be noticed.
- Congestion and hemorrhage may be observed in the brain, lymph glands, liver, kidney, lungs, spleen, thymus and ocular muscles.
  - Microscopically,
- There may be delay in the myelin sheath formation in the spinal cord.
- Ganglion cells of corpus striatum may reveal changes of shape and vacuole formation.
- Vasculitis affecting small arteries (sometimes obliterating them) is seen in various organs.

- **EPILEPSY**
- Definition

- Epilepsy is a sudden brief (petit mal) or prolonged (grand mal), loss of consciousness usually preceded by convulsions.
  - Types
- *Symptomatic epilepsy*
  - It may occur in animals due to organic brain lesions such as neoplasms or inflammation or trauma; disturbances in brain metabolism due to visceral pathology or metabolic diseases or poisons; cerebrospinal nematodiasis; verminous infestations or profound toxemias.
  -
- *True or Idiopathic epilepsy*
  - It is an inherited condition in Brown Swiss cattle and Cocker spaniels.
  - The inheritance is through a recessive factor.
  - Between attacks the animals are preferably well and the condition persists for life.
  - Clinical signs
- A true grand mal epileptiform seizure is manifested by an early period of alertness, followed by a state of tetany, which gives way after a few seconds to a clonic convulsion with padding, opisthotonus, champing of jaws and salivation. The clonic convulsions are followed by a period of relaxation. This may spread from the initial area to the rest of body, which is referred to as Jacksonian epilepsy.
- The animal is unconscious throughout the seizure.
- Evacuation of the bladder or bowel or both is common during the seizure.
- The animals may quickly regain its normal state after the seizure or act dazed or uncoordinated for a few minutes.
- The temperature may be elevated or normal.
- The pulse is frequent and respiration rate is increased.

- The blood, cerebrospinal fluid and urine are normal.
- The attacks are always recurrent and the animals are normal in the intervening periods.

SWAYBACK OR ENZOOTIC ATAXIA

- Occurrence: The disease is seen in new born lambs in certain parts of the world.
- Etiology

- Swayback is attributed to a deficiency of copper.
- The ewes which are maintained on a copper deficient diet or grazed on lands with molybdenum-rich grasses may manifest anemia and produce "steely" wool. Lambs of such ewes show demyelination and suffer from "swayback".
  - Clinical signs
- The symptoms noticed are severe ataxia, locomotor disturbance, paralysis and inability to walk.
- Affected animals may be blind and so are unable to move.
- Death may also be due to broncho-pneumonia.
  - Macroscopically,
- Lesions are not prominent in mild cases. But in severe cases, cavities containing gelatinous material may be found in the white matter due to liquefactive necrosis with secondary internal hydrocephalus.
- The lesions are bilaterally symmetrical.
- Flattening of cranial bones occurs due to cystic degeneration and increased intracranial pressure.
  - Microscopically,
- Diffuse symmetrical destruction of the white matter in the cerebrum is noticed, which is liquefactive necrosis.
- There is destruction of descending myelinated tracts.
- Gitter cells are numerous in the area.
- There is reduction in the cytochrome oxidase activity of neurons.

MENINGITIS

- Definition: Inflammation of the meninges is called meningitis.
- Pachymeningitis

- *Definition*: Inflammation of the dura mater is called pachymeningitis.
  -
- *Etiology*
  - The inflammation of the dura mater is usually secondary to infection of the middle ear or adjacent bone.
  - It may be suppurative or non-suppurative.
  - In the suppurative variety which is more common, local abscesses may be found on the dura and the peridural spaces.
  - Subsequently chronic fibrosis may develop when the dura is thickened with local adhesions.
  - Infection may spread to the arachnoid causing leptomenigitis.
  - Leptomenigitis
- *Definition*
  - Leptomenigitis is the inflammation of the pia-arachnoid.
  - When associated with inflammation of the brain, which is usually the case, the condition is known as meningoencephalitis.
- *Etiology*
  - Leptomeningitis may be suppurative or non-suppurative.

- The causes are
  - Mechanical injuries – fractures
  - Extension from adjacent tissues – as in viral encephalitis (swine fever, rabies etc)
  - Bacterial infection from neighboring areas- middle ear, nasal cavity and sinuses (usually streptococci and staphylococci: other bacteria that may cause leptomeningtitis are: - *Listeria, Corynebacterium pyrogenes, Pseudomonas*, coliforms, *Pasteurella*; chronic meningitis is produced by *Toxoplasma, Mycobacterium tuberculosis* and *Cryptococcus*. In swine *Leptospira pomona* causes non-suppurative meningitis).
  - Hematogenous infection in septicemic conditions – navel ill; enzootic pneumonia, Colibacillosis, purulent pneumonia, metastasis from infections such as mastitis, metritis or peritonitis
  - Parasitic invasion as in *Multiceps* infection in sheep.
  - Hemorrhagic meningitis is seen in acute lead and copper poisonings.
- *Pathogenesis*
  - Due to the movement of the C.S.F, the inflammation is usually diffuse.
  - The causative organisms grow on the surface of the pia-arachnoid and in its spaces.
  - Injury to the blood vessels is responsible for the inflammatory exudates.
- *Gross pathology*
  - Hyperemia is severe in meningitis.
  - In suppurative meningitis, the exudate which is yellow or greenish accumulates in the pia-arachnoid space.
  - Pus may also be found in the spinal fluid. When it accumulates in the lateral ventricles, the convolutions tend to be flattened.
  - Due to gravity the inflammatory fluid collects at the base of the brain.
- *Histopathology*
  - Suppurative inflammation is characterized by the infiltration of neutrophils while mononuclears (lymphocytes and macrophages) predominate in the non-suppurative variety.
  -
  -
- *Diagnosis*
  - Examination of the spinal fluid collected from a lumbar puncture gives, valuable information as to the nature of infection.
  - **PATHOLOGY OF SPINAL CORD**
  - **Myelitis**
  - Definition
- Myelitis is inflammation of the spinal cord.
- Usually myelitis is found along with encephalitis when the condition is known as encephalomyelitis.
  - Types
- Myelitis may be suppurative or non-suppurative and the lesions are comparable to those of the brain.
  - Etiology
- Sometimes non-suppurative myelitis may occur without any attributable cause. In such cases trauma (automobile accidents) are believed to be the cause.
- Fractures of spinal column and protrusion of inter-vertebral disc may be other causes.

    - Gross pathology
- Macroscopically, there may be congestion of the pia, petechiae on and inside the spinal cord and in advanced cases, softening of the nervous tissue.
    - Microscopically,
- Congestion, infiltration by inflammatory cells and degenerative changes of the nerve cells are seen.
- The nerve cells are swollen.
- Nissl substance disappears and the nucleus assumes an eccentric position.
- Degenerative changes of the nerve fibres are also observed.
    - **PATHOLOGY OF THE PERIPHERAL NERVES**
    - Degeneration
- *Types*
    - When a nerve cell undergoes degeneration due to the action of an irritant, the degenerative process also affects the nerve fibre of that cell. This is known as descending degeneration .
    - Degeneration can also begin in the nerve fibre and progress towards the nerve cell (ascending degeneration).
- *Histopathology*
    - Microscopically, both axis cylinder and myelin sheath are simultaneously involved (total degeneration).
    - Loss of the myelin substance is called demyelination.
    - When a nerve fibre (axon gets severed from cell body, the distal part of the nerve fibre undergoes characteristic degenerative changes known as Wallerian degeneration . The axis cylinder disintegrates and disappears; the myelin sheath (medullary sheath) also degenerates and is transformed into a chain of lipoid droplets which can be stained black by marchi's method. The cells of the sheath of Schwann proliferate and get converted into phagocytes which remove the remnants of axis cylinder and the lipoid droplet. Similar changes occur in the proximal part up to the first node of Ranvier.
    - Regeneration
- Nerve fibres in the central nervous system cannot regenerate as it is lacking a sheath of Schwann, but the peripheral nerves regenerate fairly rapidly.
- In a degenerated nerve fibre there are also attempts at repair.
- Repair in a nerve fibre is a prolonged process and it requires 10 to 12 months for complete healing.
- Schwann cells play a leading role in the healing of nerves.
- If the sheath of Schwann (neurilemma) is intact, the Schwann cells proliferate and arrange themselves in both proximal and distal ends in the form of a tube. Along this tube new axis cylinders grow and unite the two severed ends. They fall to heal the gap if it is more than one inch. In such cases, the gap is filled in by granulation tissue which originates from the three connective tissue coverings of the nerve and its bundles. The Schwann cells proliferate at both ends.
- In case of amputation, the axon fibrils coil up and form a nodule called amputation neuroma which is covered by fibrous tissue.
- When a peripheral nerve is cut degenerative changes occur in the neurons. These changes are called Nissl's Degeneration in which the cells become enlarged and the nucleus is pushed to a side (eccentric). Chromatolysis of Nissl's substance occurs after breaking up.
- When regeneration of the nerve fibre starts the neuron tends to return to normal. Nissl granules reappear, nucleus takes up a central position and the cell becomes smaller.

- Neuritis

- *Definition*: Neuritis is inflammation of the peripheral nerve.
- *Etiology*
  - Trauma
  - Viruses – Marek's disease, Ranikhet disease.
  - Toxins – bacterial mostly. Neuritis occurs in infectious diseases as in strangles, protozoal – dourine, viral - rabies and distemper.
  - Chemical poisons – lead, mercury, arsenic, alcohol
  - Plant poisons – *Lathyrus sativus*.
  - Nutritional deficiency – deficiency of the members of vitamin B group.
  - Allergic factors.
- *Gross pathology*
  - More often no naked eye changes are noticed.
  - The nerve may be swollen, reddened, soft or flabby.
- *Histopathology*
  - Microscopically, inflammation of the nerves is usually accompanied by degenerative changes.
  - Degenerative changes even leading to Wallerian degeneration are found.
  - Edema and infiltration by inflammatory cells of interstitial connective tissue can be seen.
  - The exudates may be serous (serous neuritis) or purulent (purulent neuritis). The latter variety may destroy the nerve completely.
  - Marek's disease
- It is a lymphoproliferative disease of the domestic fowl which has an unusual predilection for peripheral nerves.
- It occurs in classical and acute forms.
- The classified form is characterized by peripheral nerve enlargement, and paretic and paralytic symptoms.
- The nerves that are commonly affected are the branchial and sciatic plexuses, celiac plexus, abdominal vagus and intercostals nerves.
- Microscopically, the nerves are infiltrated with lymphoid cells, which include primitive and activated reticular cells, lymphoblasts and small medium and large lymphocytes.

  - **NEOPLASMS AFFECTING THE NERVOUS SYSTEM**
  - Primary tumors

- Primary tumors of the brain and spinal cord are rare. However, tumors of the central nervous system are most common in dogs and least common in the pig and sheep.
- Primary tumors include those of the neuroglia (gliomas, astrocytoma, oligodendroglioma), nerve cell and fibres (neuromas), ganglion cells (ganglioneuroma), ependymal cells (ependymomas) and of meninges (meningiomas).
- The brain tumors are of limited malignancy, metastases not occurring elsewhere. Pressure on the brain by the developing neoplasms produces various symptoms depending upon the part of the brain involved and the functional disturbances in turn are dependent on the neurons of the part which are responsible for them. Death is the invarible outcome.
  - Secondary tumors
- Central nervous system is also prone to secondary tumors, which are metastatic, their primary sites being the lung or some other organ.

  - **POSTMORTEM CHANGES IN THE BRAIN**

- Post mortem changes in the brain include hypostatic congestion and softening of the brain.
    -
    -
    - **ENDOCRINE GLANDS**
    - **PITUITARY GLAND**
    - Anatomical features: The gland is divided into adenohypophysis and neurohypophysis.
    - Physiological functions
- This gland is called as master gland in the body and controls all other glands in the body by its hormones.
    - **PATHOLOGY OF PITUITARY GLAND**
    - Aplasia
- Aplasia of the hypophysis has been observed in Holstein-Friesian cattle, and is controlled by an autosomal recessive gene.
- Histologically there is evidence of failure or differentiation of acidophils.
    - Atrophy
- Atrophy may be caused by the pressure of cysts or Tumors.
- Atrophy may be present to a great extent without signs of hypophyseal deficiency.
- Basophils are more sensitive to pressure than acidophils.
    - Cysts
- Occurrence: Cysts are relatively common in dogs (short muzzled dogs and cross breeds).
- Etiology
    - They may be developmental cysts (Rathke's cleft cyst; Craniopharyngeal cyst: Evagination cyst) or acquired cyst (common in cattle and horses due to nutritional deficiency of Vitamin A or due to senility).
- Gross pathology
    - Craniopharyngeal end of Rathke's pouch may persist resulting in the formation of pharyngeal hypophysis, a cystic non-glandular structure.
    - Cysts may be single or multiple and may be unilocular or multilocular.
    - These are seen commonly in the periphery of pars distalis and pars tuberalis.
- Histopathology
    - Cysts are lined by cuboidal, columnar, ciliated columnar or squamous epithelium.
- Sequelae
    - Though cysts may be encountered only, occasionally, they cause functional disturbances.
        - **PITUITARY LESIONS IN DISORDERS OF OTHER ORGAN**
    - Hypothyroidism
- Adenohypophysis is enlarged and the large Beta basophils are hypertrophied. The granules of these cells become finer and disappear eventually.
- In severe cases, these Beta cells become vacuolated giving a spongy appearance to the medulla, where these cells are found in large numbers.
    - Gonadal deficiency
- Gonodotropin producing basophils become degranulated and are transformed into hypertrophic aminophils, a change characterized by enlarged nucleus, thinness and irregularity of nuclear membrane.
    - Stress

- In chronically stressed animals, basophils of both types store granules. This accounts for gonadal inactivity in such animals.
- The acidophils are hyperactive and may be completely degranulated cells or a few granulated cells persist adjacent to sinusoids.

HYPERPITUITARISM

- This condition is manifested by overgrowth and proliferation of bones.
- In man this is called " Gigantism " in young growing individuals in whom the ossification of bone has not yet stopped and " acromegaly " in adults in whom no more growth occurs.

Gigantism

- Gigantism is due to increased secretion of somatotropin in the young. The individual grows very tall and the skin and subcutaneous tissues show fibrous hyperplasia. Since STH is diabetogenic, glycosuria is a symptom.
- As this condition occurs in adenoma of the acidophils and since the neoplastic growth produces pressure atrophy on the basophils impotence in the male and amenorrhoea in the female are observed.
- If the patient lives beyond the age of epiphyseal fusion acromegaly may result.

Acromegaly

- Acromegaly (akros=extremity; megale=enlarged) is the condition that is observed in the adult. Since no growth in the bone is possible, the bone becomes thicker and broader.
- The hands and the feet are abnormally large and the fingers are crooked and knotty. The facial bones become long and thick, especially the jaw, resulting in prognathism.
- Viscera are enlarged (splanchnomegaly or macrosplanchnia) and fibrous hyperplasia of skin and subcutaneous tissue is common. Nose, lips and ears become large.
- Kyphosis is also seen in some. Impotence in the male and amenorrhoea in the female are the other symptoms.
- Diabetes mellitus occurs due to diabetogenic action of the hormone. Eye lesions may be noticed due to pressure on the optic chiasma by the tumor.

Cushing's syndrome

- Basophil adenoma may produce this disorder.
- Conversely, hyper function of the adrenal cortex may cause changes in the pituitary.

HYPOPITUITARISM

- Since the pituitary is enclosed on three sides by hard bone, even slight enlargement of some part will cause atrophy on others and so corresponding decrease in the activity of the cells results.

Etiology

- Causes of pituitary hypofunction
- Pressure by: a) Tumors b) Cysts
- Inflammation and sclerosis
- Infarction and necrosis
- Hydrocephalus – bulging of the floor of the ventricles
- Abnormal development
- Tuberculosis

Pituitary dwarfism or infantilism

- Hypofunction in the young children causes in pituitary dwarfism or infantilism . This condition is not seen in animals.

Symmond's disease (Pituitary cachexia) or Sheehan's Syndrome

- This condition was described in dogs.
- *Etiology*

    - This is found only in females, due to postpartum necrosis of the pituitary consequent on thrombosis following hemorrhage.
    - Hence severe hypopituitarism develops.
- *Clinical signs*
    - The characteristic clinical signs are severe cachexia, loss of sexual function, weakness, low metabolic rate, loss of hair and pigmentation, mental apathy, drowsiness, microsplanchnia, extreme dehydration and emaciation.
- *Lesions*
    - Since tropic hormones are not secreted, there is atrophy and fibrosis of the thyroid, adrenal glands, ovaries and parathyroids together with the symptoms and lesions consequent on the deficiency of the hormones secreted by these glands and structures.
    - Frohlich's Syndrome- Dystrophia adiposogenitalis
- This develops probably due to the pressure by a tumor or hydrocephalus and is mostly found in ladies.
- In man, it is feminizing with the characteristic distribution of fat as in a female.
- *Clinical signs*
    - The characteristic features are
        - Obesity: There is disproportionate and excessive accumulation of fat on the abdomen, buttocks and thighs, while other parts are thinner.
        - Genital hypoplasia and decreased sexual function
        - Idiocy or mental retardation
        - Thin skin and hair
        - Reduced sweat secretion
    - Diabetes insipidus
- *Etiology*
    - Normally under the influence of the antidiuretic hormone of the neurohypophysis, 80% of the water in the glomerular filtrate is reabsorbed by the epithelium of the Henle's loops and distal convoluted tubules. But if the secretion of the ADH is interfered with due to failure of the hypothalamic-hypophyseal system, reabsorption of water from the glomerular filtrate does not occur and so large quantities of urine with low specific gravity are passed and this condition is known as diabetes insipidus.
    - Lesions of the pars nervosa or any causes that injure the hypothalamus will produce diabetes insipidus. The secretion of ADH by the pars nervosa is under the control of stimuli from the hypothalamic nuclei.
    - The causes that may produce this condition are
        - Trauma- surgical or fractures
        - Pituitary tumor or metastases from bronchogenic carcinoma ort mammary carcinoma
        - Meningitis- pressing on the stalk
        - Encephalitis.

    - **TUMORS**
- Chromophobe adenoma, acidophilc adenoma and basophilic adenoma of pituitary were rarely reported.
- Carcinomata of the above three types of cells may be noticed. But these are only locally invasive.
    - **THYROID GLAND**
    - Anatomical features
- Thyroid gland is located in the cervical region.

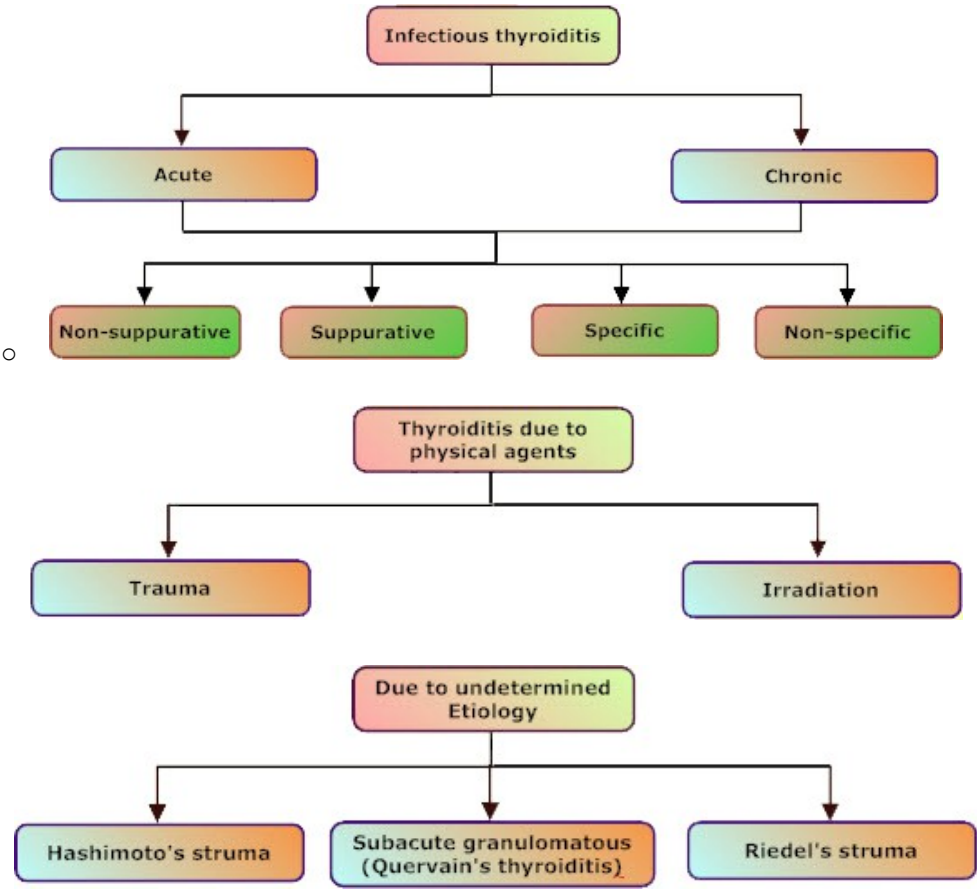
- It consists of two lobes connected by a narrow isthmus.
    - Physiological functions
- Thyroid gland secretes a hormone called thyroxine.
- Thyroxine production is under the influence of thyrotropic hormone from anterior pituitary
- Thyrotropic hormone releasing factor from hypothalamus controls the production of the thyrotropic hormone.
- Thyroxine maintains a high rate of metabolism in the animal
- Thyroxine is required for estrus and libido.
- If the thyroxine level is decreased, the animal is dull and lethargic.
- If the thyroxine level is increased, the animal becomes nervous, irritable, hyperactive and jumpy.
- Parafollicular or "C" cells of the thyroid produce thyrocalcitonin which enhances the excretion of sodium, phosphates and calcium.
    - **PATHOLOGY OF THYROID GLAND**
    - Cysts in thyroid gland
- Cysts in the thyroid gland may be ultimobrachial cysts, thyroglossal duct cysts or parenchymal cysts.
    - Atrophy of thyroid gland
    - Atrophy of thyroid gland may occur secondary to lesions in the pituitary gland
- *Histopathology*
        - Fatty metaplasia of interstitial tissue takes place.
        - Follicles become smaller and are filled with basophilic colloid and corpora amylaceae
        - Connective tissue stroma is prominent.
    - Hypothyrodism
- *Definition*: Deficiency of thyroxine is called hypothyroidism
- *Etiology*
    - Atrophy and fibrosis of thyroid gland
    - Thyroiditis
    - Hypopituitarism
    - Thyroidectomy
- *Effects*
    - The condition found in young growing children is called cretinism .
    - Hypothyroidism in adults is called myxedema .
    - Hypothyroidism in dogs leads to obesity, alopecia, thick skin and lethargy.
    - Calves usually are either born dead or die within a day or two. The calves have goitre which causes fetal cystocia.
    - Hypothyroidism in cattle causes sluggishness, agalactia, silent heat, retained placenta, still births and a tendency to purulent endometritis. This occurs in areas where there is incidence of endemic goitre.
    - Due to deficiency of thyroxine, the basal metabolic rate is abnormally low and so all the vital processes are slowed down.
        - **GOITRE**
    - Definition: Goitre is non-inflammatory and non-neoplastic enlargement of the thyroid gland
    - Goitrogens
- The substances that are responsible for the production of goitre are called goitrogens or goitrogenic substances.

- Examples: Cabbage, soyabean, sulphonamides, phenothiazine, thiocyanates and thiourea.
    - Pathogenesis
- A low level of thyroxine is a stimulus for the production of the thyrotropic hormone which causes hypertrophy and hyperplasia of the follicular epithelium.
- In general, there is increase in BMR.
    - Types of goitre
- Parenchymatous goitre
- Colloid goitre
- Nodular or adenomatous goitre
- Exophthalmic goitre
    - Parenchymatous goitre
- Synonym: Hyperplastic goitre or goitre of cretinism
- Etiology
    - Iodine deficiency in animals due to deficiency of iodine in soil and water in the locality
    - Congenital
- Gross pathology: The thyroid gland is enlarged, meaty and firm.
- Histopathology
    - The follicular epithelium is hypertrophic. The cells become tall and plump.
    - Later, there is hyperplasia of these cells with papillary projections into the lumen.
    - Colloid goitre
- Synonym: Simple goiter
- Occurrence: Simple goitre is the most frequent form of goitre in animals
- Etiology
    - Low levels of iodine in soil and water.
    - Ingestion of goitrogenic substances.
    - Excessive demands as in adolescence or pregnancy
    - Diseased conditions interfering with assimilation of iodine as in gastroenteritis
- Clinical signs
    - Difficulty in swallowing and dyspnoea due to the pressure of the enlarged gland is noticed.
- Gross pathology: Thyroid gland is enlarged. The cut section is translucent.
- Histopathology
        - The acini are widely dilated and contain watery, faintly staining colloid.
        - Sometimes neighboring acini may coalesce to form cysts.
        - Leakage of the colloid into the interstitial space may occur due to rupture of cysts.
        - Colloid is deficient in iodine and thyroxine.
        - The epithelium is flattened.
        - Since this condition is supposed to be an involutionary phase of hyperplastic goitre, here and there some acini may show papillary projections of the epithelium.
    - Nodular or adenomatous goiter
- Occurrence: Nodular goiter is seen in old and senile horses, dogs and cattle.
- Predisposing causes: Sexual excitement and pregnancy
- Etiology

    - This may be an outcome of alternating hyperplasia, hypertrophy and involution affecting gland.
    - Hereditary predisposition (endogenous factor)
    - Exogenous factor like deficiency of iodine
    - Fluorine containing compounds have goitrogenic effects.
- Gross pathology
    - Nodules of varying sizes, microscopic to several centimeters in diameter are found
    - On section, the nodules are translucent and may contain cysts or vesicles filled with gelatinous colloid.
- Histopathology
    - Microscopically, the picture is variable. All gradations may be found in different nodules from the picture of a colloid goitre to that of hyperplastic goitre. But in a nodule, the picture is constant.
    - In some nodules dilated acini filled with colloid and having flattened epithelium may be present. In other places, acini containing papillary projections of the epithelium obliterating the lumen may be found.
    - Retrogressive changes, leading to necrosis with subsequent softening and liquefaction are responsible for cyst formation (pseudocyst).
    - Cystic goiter may also result due to formation of the cysts by the confluence of smaller colloid filled follicles. These are lined by flattened epithelium.
    - The connective tissue which is increased undergoes hyalinization.
    - Calcification (calcareous goitre) and metaplasia into bone (osseous goitre) of the connective tissue may also be encountered.
    - Hemorrhage into follicles due to erosion of vessels causes hemorrhagic goitre . The coagulation of the fibrin produces rubber colloid.
- Sequelae
    - The nodules are inert and functionless. But in others, thyroid hormone may be actively secreted when toxic adenoma is applied to the condition. This condition is met with in horses and dogs with signs of hyperthyroidism.
    - Exophthalmic goitre
- Synonym: Grave's disease, Basedow's disease or primary thyrotoxicosis.
- Occurrence: This disorder of the thyroid gland is found in man and is probably not seen in animals.

THYROIDITIS

- Definition: Thyroiditis means inflammation of the thyroid.
- Occurrence: Thyroiditis is less common in animals.
- Etiology
- In generalized infections, thyroid may also get involved.
- Subcutaneous suppurative inflammatory lesions may spread to the thyroid.
- Classification
- Thyroiditis may be classified as
    - Infectious thyroiditis,
    - Thyroiditis due to physical agents,
    - Due to undetermied Etiology



- Hashimoto's struma (Struma lymphomatosa)

- *Occurrence*: It is relatively common in dogs
- *Predisposing causes*: There is a genetic predisposition. Incidence is high in Beagle breed of dogs.
- *Etiology*
  - The cause is an autoimmune reaction.
  - Thyroglobulin leaks out into the blood due to defect in the basement membrane and stimulates antibody production.
  - These antibodies are being formed by plasma cells and lymphocytes which infiltrate the gland.
  - Three specific antigen-antibody systems have been described. 1. Microsomal 2. Thyroglobulin and 3. An altered colloid.
- *Lesions*
  - Gross lesions are not characteristic.
  - Histologically, there rare multiple isolated lymphoid nodules with well formed germinal centres replacing the parenchymal tissue.
  - The interstitial tissue and the parenchyma show dense streaks or collections of lymphoid cells, plasma cells and large mononuclears.
  - Larger or oval oxyphilic cells are occasionally seen in the follicles (Askanazy cells).
  - Subacute granulomatous (Quervain's) Thyroiditis
- The disease follows an acute respiratory infection caused by a virus.
- It is associated with epithelial necrosis, disappearance of epithelial cells and infiltration by histiocytes, mononuclears, fibroblasts and multinucleated giant cells.

- Riedel's struma

- Here the gland is very firm, hard and is adherent to the surrounding tissue.
- Histologically, there is severe fibrosis and diffuse, moderate infiltration with lymphocytes and mononuclear cells.
- The involvement is unilateral compared to the Hashimoto's struma. There is no giant cell reaction.

- **NEOPLASM**

- Primary tumors

- Adenoma is reported in the horse.
- Adenocarcinoma and adenoma in the old dogs are more common.
- These tumors are found in areas of endemic goitre.

- Secondary tumors of the thyroid

- Metastases from mammary tumors and lymphosarcomas are common in the thyroid.

- **PATHOLOGY OF THE PARATHYROID GLANDS**
- Physiological functions

- Parathyroid gland secretes a hormone called parathormone which maintains calcium-phosphorus balance in the body by regulating the osteoclastic activity in the bone.

- **Hyperparathyroidism**
- It may be primary or secondary.

- *Primary parathyroidism*
  - It is due to an adenoma, in which increased parathormone is secreted.
  - Changes consist of osteopathy, nephrocalcinosis and urolithiasis.
  - In nephrocalcinosis, there is diffuse calcium deposition in the renal tubular epithelium.
- *Secondary hyperparathyroidism*
  - Etiology
    - It is found in
    - Inadequate intake of calcium in diet; or imbalance of calcium: phosphorus ratio.
    - Hypocalcemia due to vitamin D deficiency
    - Steatorrhoea
    - Chronic renal failure – Phosphorus excretion in interfered with.
    - Pregnancy and lactation – calcium is side-tracked to the fetus and milk respectively.
  - Pathogenesis
    - In these conditions there is a lowering of blood calcium or rise in the level of blood inorganic phosphorus leading to hyperplasia of the parathyroids.
  - Sequelae
    - Osteritis fibrosa cyctica and Rubber Jaw Syndrome are the sequelae in these conditions.
  - **THE ADRENAL GLANDS**
- The adrenocorticotropic hormone (ACTH) controls the form and function of the cortex. Decrease or increase of ACTH is followed by atrophy (regressive transformation) or hypertrophy (progressive transformation) of the adrenal respectively.
- When ACTH is lacking, atrophy of all zones occurs. Differentiation of the cells is lost. The cytoplasm and nuclei become smaller and the storage capacity of the lipid is lost. The capsule becomes thickened and the fibrous tissue of the gland is increased.

- Continuous ACTH administration causes hypertrophy and hyperplasia of the cortex, which, therefore, becomes broader. The cytoplasm and the nuclei of the cells become increased in size and the storage capacity of the cells for lipid is increased.
    - **ADRENAL CORTEX**
- The adrenal cortex has three zones viz. *zona glomerulosa, zona fasciculata* and *zona reticulata.*
- The three zones of the cortex produce three different physiologically active hormones viz. glucocorticoides, mineralocorticoides and sex hormones.
    - Glucocorticoids
- Glucocorticoides are secreted by the Zona fasciculata.
- The most important glucocorticoids are the hydrocortisone or cortisol (compound F) and cortisone (compound E).
    - Physiological functions
- Shifting of fat stores: Circulating fatty acids are more and these are used for energy. Some are converted into glycogen of the liver, sparing glucose.
- Anti-inflammatory function: Glucocorticoids suppress connective tissue response to injury, activity of the fibroblasts, vacularisation and granulation tissue formation and intercellular ground substance formation.
- Decreases the secretion of pepsin and HCl.
- Decreases the hyaluronidase activity.
    - Adreno-cortisol insufficiency
- *Dogs*
    - Primary adreno-cortisol insufficiency has been met with in dogs and they are manifested as anorexia, diarrhoea, asthenia, polydipsia, azotemia, hyponatremia, cardiovascular effects, eosinophilia, dehydration, anuria, hair loss, emesis and hyperkalemia.
- *Shipping fever*
    - A condition of hypoadrenalism can arise due to stress of transport, cold, hunger, fatigue, lack of water and food, fright and infectious agents. This stimulates the production of ACTH, which in turn stimulates the cortex which finally becomes exhausted.
    - Infectious organisms may then invade and cause shipping pneumonia and fever.
    - Mineralocorticoids
- Mineralocorticoides are secreted by the cells of *zona glomerulosa*. These hormones control the electrolyte and water balance. The chief hormone is called aldosterone.
- In deficiency of aldosterone, sodium, chloride, carbonate and water are lost-extracellular fluid is decreased, thereby minimizing the cardiac output and lowering the blood pressure. This leads to failure of circulation, culminating in shock, coma and death.
    - Sex hormone
- Sex hormones of adrenal cortex are produced by the zona reticularis.
- The hormones are mostly androgens (masculanising) and a few only estrogens (feminizing)
    - **ADRENAL MEDULLA**
- The hormones of the medulla are adrenaline and noradrenaline and are produced in the ratio of 4:1.
    - Reactions of adrenal glands to stress
- Adrenal cortex reacts to stress by depletion of lipids.
- Three major patterns occur based on variation of lipid content.

- Focal lipid depletion with alteration in cell type
- Focal depletion of lipid with alteration in cell type and degenerative changes and
- Lipid reversion.

- In the first type cells are hypertrophic and compact.
- In the second, degenerative changes are characterized by cytoplasmic degeneration. Solid cords of cells in zona fasciculata break up to form pseudotubules and tubules with lumina formation. The lumen of the tubules contains detached cells, pale staining fluid or erythrocytes. Occasionally infarcts affecting mid zona-fasciculata may be seen.
- In the third, lipid reversion occurs in local depletion i.e. lipid is absent in outer fasciculate, scanty in reticularis but abundant in the remaining fasciculate. This picture is characteristic of conditions in which the adrenal recovers from stress.

- **HYPOCORTICALISM**
  - Congenital hypoplasia

- Congenital hypoplasia of the adrenal gland has been reported in dogs and a hereditary predisposition has been indicated.
- Adrenal hypoplasia associated with maldevelopment of hypophysis is relatively frequent in calves.
  - Hypofunction of adrenal cortex
- Acute hypofunction caused by extirpation is rare
- Chronic hypofunction (Addison's disease): The manifestation of Addison's disease may occur only if there is bilateral destruction of the glands. This condition is exceedingly rare in animals. It is observed in tuberculosis and hypopituitarism. In this condition, there is general weakness, anemia, low blood pressure and brown pigmentation of skin.
  -
  - Adrenal Hyperplasia
- Cortical hyperplasia is relatively common, particularly in dogs.
- Hyperplasia may be of zona glomerulosa, zona fasciculate or zona reticularis. It may be focal or diffuse and may or may not be associated with clinical manifestation.
- Progressive and regressive transformations have been described in stress reaction.
- The progressive transformation is characterized by cellular hypertrophy, increased formation of new cells and storage of fat, while the regressive transformation is characterized by depletion of fat and hypofunction of cortex.
- In severe degree of progressive transformation, there may be formation of accessory cortical nodules. There is proliferation of reserve cells in the sudanophobic zone and transformation of these cells into zona fasciculata type.
- In the hyperplastic zone acini and secretory activity may be seen and this is a morphological expression of severe hyperactivity, wherein the gland takes resource to exocrine type of secretion.
- Accessory cortical nodule formation is characterized by formation of nodules on the capsule of the adrenal. Histogenesis of the cells which form the nodule is from the subcapsular blastema.
- The reserve cells in the subcapsular blastema proliferate and invade the capsule which gets split up and later the proliferating cells get encapsulated by fibrous tissue.
- The small size of the lesion, absence of encapsulation and acini formation are all features of cortical hyperplasia while adenoma is characterized by large sized nodules, encapsulation, acini formation and the type cells are columnar, lipid-depleted cells.
- Regressive transformation is seen in acute diseased conditions.

- Hyperfunction of adrenal cortex – Cushing's syndrome

- In hyperfunction of the adrenal cortex, there is excess of circulating hydrocortisone. This may be due to basophile adenoma of the anterior pituitary.
- Zona fasiculata is increased and the adrenals are yellow in colour.
- Hyalinisation of the basophile cells of the anterior pituitary together with disappearance of basophile granules is a frequent finding in Cushing's syndrome.
  - Adrenogenital syndrome (Adrenal virilism)
- A syndrome in which "little girls become little boys" and " little boys –little men".
- In this condition there is an excess of androgens – masculanising hormones.
- In female fetus, if the excess of hormone occurs during the first few weeks of intrauterine life, pseudo-hermaphroditism results. If it occurs later in females or in boys, precocious puberty may result.
  - **NEOPLASMS**
- Adenoma of adrenal gland may be seen in dogs and horses. Pheochromocytoma or medullary chromaffinoma is also reported in dogs, cattle, horses and sheep.
  - **PINEAL GLAND**
  - Introduction
- Pineal gland is a tiny gland located above the posterior extremity of the third ventricle of brain.
- It secrets a hormone called melatonin .
- Melatonin antagonizes the action of the melanocyte stimulating hormone of the posterior pituitary.
- It inhibits thyroid hormone secretion rate and secretion of the adrenal steroids.
  - Pineal dysfunction
- Pineal hyperfunction is associated with delayed puberty.
- Pineal hypofunction results in precocious puberty.
  - Neoplasms of pineal gland: Tumors reported in pineal gland include pinealoma, glioma and teratoma.
  - **THYMUS**
  - Introduction
- Thymus is an unpaired organ situated in the thoracic cavity in the anterior mediastinum in the livestock.
- It is an important site of lymphopoiesis in the embryo and new-borns.
- Clones of thymic cells are believed to migrate to the other lymphoid tissues like Peyer's patches, spleen and lymph nodes, colonise and enable them to give an antibody response to specific antigen stimulation in later life.
- In toxaemic cases, the thymus shows haemorrhages as in chicken and young animals.
  - Status thymolymphaticus
- It is associated with sudden death in dog, cats and cattle without obvious cause.
- In such animals, the thymus is enlarged due to hyperplasia. Other lymphoid organs are also enlarged.
  - Myasthenia gravis
- It is an autoimmune mechanism characterized by great muscular weakness.
- The thymus is hyperplastic with increased number of germinal centres. In the blood, antinuclear antibodies can be detected.
  - Neoplasms of thymus: Neoplasms of the thymus include thymoma and lymphosarcoma.
  -
  -
  -

-
-
-
-