Hippocrates is recognized as the Father of Medicine, laying the groundwork for medical ethics and practices.

King Ashoka (273–232 BC) established numerous veterinary hospitals, marking the construction of the world's first veterinary hospital, which was based on ancient Vedic texts.

The Atharvaveda contains references to ailments and treatments for animals, highlighting the early understanding of veterinary care.

Shalihotra is noted as the first veterinarian, particularly skilled in horse care, while Palakpaya, an expert on elephants, authored Gaja Ayurveda.

Additionally, Nakul and Sahadev were known as horse experts during the Mahabharata era.

The first formal veterinary school was founded in Lyon, France, in 1762.

In India, the first army veterinary school was established in Poona in 1862, followed by the Bombay Veterinary College in 1886, which became the first veterinary college in the country.

The Tamil Nadu Veterinary and Animal Sciences University (TANUVAS) later emerged as India's first veterinary university, further advancing the field of veterinary education.

- Sporadic – affects 1 or 2 animals in distant places
- Epidemic – large population in large area
- Endemic – disease found In large population in small area for long time
- Pandemic- epidemic in multiple countries
- Diagnosis – identifying the disease and nature of disease.
- Differential diagnosis – rule out other disease with similar symptoms
- Tentative diagnosis- based on clinical examination
- Confirmatory diagnosis- disease diagnosed through clinical, clinic pathological and laboratory examination
- Snap shot diagnosis – diagnosis at glance
- Palpation - handling tissues, organs or parts of animal body
- Percussion - striking or tapping a part of the body.

  -body surface is struck so as to set deep parts in vibration and cause to emit audible sounds.
- Auscultation: It is a method of listening to the functional sounds produced by internal organs
    - direct and indirect

- **Ballottement**: Ballottement is a technique used to detect floating viscera or masses in the abdominal cavity
- Helio (pertaining to Sun) treatment: using sunlight in rickets
- Placebo: for mental satisfaction of animal; no relation with disease
- Empirical treatment: based on experience
- Rational: scientific method of treatment
- Palliative treatment: to extend life in incurable diseases
- Prophylactic treatment: to prevent spread of disease

Shock

- life-threatening condition associated with reduced blood supply to vital organs in the body
- When heart (pump) is affected, it is called pump failure or heart failure and it leads to a condition called cardiogenic shock.
- When the functioning of circuit is affected it is called circuit failure and it leads to a condition called circuit shock

Circuit shock (failure)

- Hypovolemic shock: reduction in the circulating volume of blood as a result of loss of plasma or free water. Eg. Fluid loss and dehydration due to diarrhoea, vomiting, burn etc.
- Haemorrhagic shock: reduction in circulating blood volume due to rapid loss of blood. Eg. Traumatic injury, umbilical haemorrhages in neonates, abomasal ulcer, pulmonary haemorrhage, parvo viral enteritis in dogs
- Maldistributive shock: reduction in circulating blood volume due to increased capillary permeability, pooling of blood in capacitance vessels (such as the veins in the splanchnic circulation), or pooling of plasma in thoracic or abdominal cavities. Eg. Endotoxemia in neonatal septicaemia, salmonellosis, coliform mastitis
- Obstructive shock: Obstructive shock occurs when venous return of blood back to heart is reduced or prevented by a mechanical obstruction. Eg. Pericardial tamponade, pulmonary artery thrombosis etc.

Signs

- Pale grey to white and dry mucous membranes, Low body temperature, Cold skin and extremities, and a prolonged CRT (> 3-4 seconds).
- Heart rate is increased, Pulse is small and weak – ‘thready pulse’, it is difficult to raise veins

- Anuria or oliguria
- Plasma L-lactate: <1.5 mmol/L is normal; >4 mmol/L indicates widespread anaerobic respiration and >10 mmol/L indicates a very poor prognosis.

Hypersenstivity

- HYPERSENSITIVITY It represents an accelerated immune response to an antigen (allergen), which is harmful to body. also known as allergy or atopy.
- When the hypersensitive reaction is sudden and clinically severe it is called anaphylaxis. Severe anaphylaxis may result in anaphylactic shock.
- Epinephrine is the most effective treatment for anaphylaxis and anaphylactic shock. 1:1000 solution @ 0.01-0.02 ml/ kg given IM or SC.
- Type I Hypersensitivity or immediate hypersensitivity: the antigen or the allergen reacts with circulating or cell bound antibody to release biochemical mediators. Rapidly developing immune response to an antigen characterized by humoral antibodies of IgE type.
- Bite of insects, bee etc. Dust, pollens etc.
- Type II Hypersensitivity or Auto immune reaction: In autoimmune reactions, body produces antibodies against proteins present in the body. lysis of cells due to antigen- antibody reaction on the surface of cells in the presence of complement.
- Blood transfusion, Hemolytic anemia, Infections such as Equine infectious anemia, rickettsia, parasites (trypanosomiosis, babesiosis)
- Type III Hypersensitivity (Arthus type reaction/ Arthus phenomenon): Type-III hypersensitivity reaction is characterized by the formation of immune complexes as a result of antigen – antibody reaction and their deposition in body tissues leading to inflammatory reaction.
- Arthus phenomenon is the development of an inflammatory lesion, with erythema, oedema, haemorrhage and necrosis.
- Type IV Hypersensitivity reactions (Cell mediated/ delayed hypersensitivity reactions): DTH reaction is mediated by sensitized T-Iymphocytes and is the manifestation of cell- mediated immune response.Eg. Tuberculin test.

Oedema

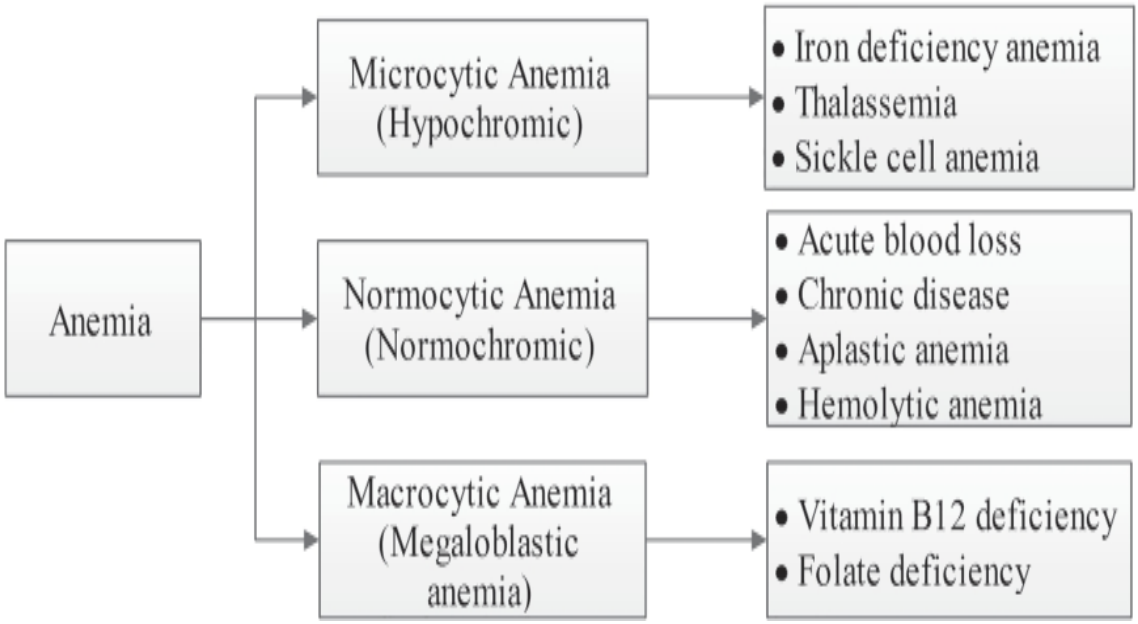
- swelling of soft tissues due to the abnormal expansion of interstitial fluid.
- Anasarca: A generalised and pronounced swelling of the body due to accumulation of fluid in the subcutaneous tissue
- Ascites: accumulation of fluid in peritoneal cavity

Mechanism

- Increased capillary hydrostatic pressure: impaired venous return
- Decreased plasma oncotic pressure: protein deficiency
- Increased capillary permeability/ Sodium retention
- Obstruction of the lymphatic system: farcy, ulcerative lymphangitis

Anemia

- absolute decrease in RBC numbers, hemoglobin concentration or PCV
- regenerative (blood loss, blood parasites, haemolytic anaemias) or non regenerative (chronic kidney diseases)
- Regenerative anaemias: haematopoiesis in the bone marrow is active and it effectively compensate for the decreased red cell mass by increasing RBC production and releasing reticulocytes.
- Nonregenerative anemia: the bone marrow responds inadequately to the increased need for RBCs. Anemia due to decreased erythropoietin or an abnormality in the bone marrow is nonregenerative.
- A reticulocyte count of <60,000 cells per microlitre of blood is considered as non-regenerative.



- Poikilocytosis is a medical term that describes the presence of abnormally shaped red blood cells (RBCs) in the blood
- Anisocytosis is a medical term that means red blood cells (RBCs) are different sizes.

- Macrocytic Normochromic: Due to vit. B12 and folic acid deficiency
- Macrocytic hypochromic- hmges
- Normocytic normochromic: Aplastic anemia due to bone marrow depression, Bracken fern poisoning, prolonged use of sulphonamides and Chloramphenicol, renal failure

Microcytic hypochromic: iron deficiency anemia

- Deficiency of Cu, Vit. C, pyridoxine, riboflavin, nicotinic acid
- Ceruloplasmin: Cu containing enzyme – transport of iron to bone marrow
- Superoxide dismutase: antioxidant, keep RBC intact--- Cu part of enzyme
- Co for maturation of RBCs
- Haematinic mixture: Fe, Cu, Co

Clinical Signs

- Lethargy and exercise intolerance
- Mucous membrane pallor
- Tachycardia
- Low grade haemic murmur
- Prominent femoral pulse
- Tachypnoea
- Episodic collapse (excitement or stress-induced)
- Jaundice (in haemolytic anemia)

Diagnosis

- Decreased Hb and TEC
- PCV - 30 to 37 – mild anemia,
- 20-29- moderate anemia
- < 20 – severe anemia
- < 12 poor prognosis
- **Red cell morphology**
- Reticulocyte count - Indicates degree of regeneration

- **Erythrocytic Indices:** Mean Corpuscular Volume (MCV) & Mean Corpuscular Hemoglobin Concentration (MCHC)

CONGESTIVE HEART FAILURE

- failure of the heart to adequately pump blood
- clinical syndrome initiated by myocardial failure and is characterized by pulmonary or systemic venous congestion or both and low cardiac output.
- Treated by Cardiac glycosides like Digoxin, Digitoxin

Left side failure

- Resting respiratory rate, depth increased
- Cough
- Moist crackles at lung base
- Dull percussion note over ventral lungs o Dyspnea, cynaosis at rest
- Possibly murmur referable to left AV or aortic valves

Right side failure

- Listlessness depression
- Reluctant to walk o Shuffling, staggery under, eventual recumbency
- Anasarca ventrally under the jaw, down the neck and under the abdomen
- Ascites, Hydrothorax
- Possibly palpable enlargement of liver beyond right costal arch
- Urine volume small, concentrated, minor albuminuria
- Jugglar vein distension (other veins also)
- Abnormally high and visible jugular pulse
- Epistaxis in some horses
- Hydropericardium

Tetralogy of Fallot

- Narrowing of the valve between the heart and the lungs, called pulmonary valve stenosis.
- A hole between the bottom heart chambers, called a ventricular septal defect.

- Over riding aorta – aorta on top of both ventricles
- Thickening of the right lower chamber of the heart, called right ventricular hypertrophy.

PNEUMONIA

- inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleuritis.
- increase in the respiratory rate, changes in the depth and character of respirations
- coughing, abnormal breath sounds on auscultation and, in most bacterial pneumonias, evidence of toxemia.

Cattle

- Pneumonic pasteurellosis (shipping fever) - M. haemolytica, P multocida with or without parainfluenza-3 virus
- Enzootic pneumonia of calves - parainfluenza-3, adenovirus-I, -2 and 3. Contagious bovine pleuropneumonia - Mycoplasma mycoides
- Lungworm pneumonia - Dictyocaulus viviparus
- Mycotic pneumonia associated with Mortierella wolfii in adult cattle

Pigs

- Enzootic pneumonia - Mycoplasma sp. with Pasteurella sp.
- secondarily Pneumonic pasteurellosis - P multocida
- Pleuropneumonia - Actinobacillus pleuropneumoniae
- Interstitial pneumonia - septicemic salmonellosis
- Bordetella bronchiseptica, Salmonella choleraesuis

Horses

- aerobic bacteria
  - alpha-hemolytic Streptococcus spp., Pasteurella spp.,
  - Escherichia coli and Enterobacter spp.
  - anaerobic bacteria
    - Bacteroides spp.,
    - Preuotella spp.,

    - Fusobacterium spp.
    - Clostridium
- Newborn foals -Streptococcus spp., E. coli, Actinobacillus equuli and other agents causing septicemia
- In immunodeficient foals, pneumonia associated with adenovirus or Pneumocystis jiroveci
- Older foals - R. equi, equine herpesvirus-1 (the EVR virus), equine influenza virus
- Goats o Pleuropneumonia associated with Mycoplasma strain F 38 or Mycoplasma capri,

Three risk factors interact in the pathogenesis of specific pneumonias:

- Animal
- Environmental & managemental
- Pathogen

Clinical Signs

- Rapid, shallow breathing is the cardinal sign of early pneumonia o
- Dyspnea occurs in the later stages when much of the lung tissue is nonfunctional
- Polypnea may be quite marked with only minor pneumonic lesions; the rapidity of the respiration is an inaccurate guide to the degree of pulmonary involvement
- Coughing is another important sign, the type of cough varying with the nature of the lesion.

Bacterial bronchopneumonia is usually accompanied by a moist and painful cough. In viral interstitial pneumonia the coughing is frequent, dry and hacking, often in paroxysms.

Crackles develop in bronchopneumonia as bronchiolar exudation increases, but in uncomplicated interstitial pneumonia, clear, harsh breath sounds are audible. In viral interstitial pneumonia, wheezes may be audible due to the presence of bronchiolitis.

Diseases affecting Digestive System

- STOMATITIS: Stomatitis is inflammation of the oral mucosa and includes glossitis, palatitis and gingivitis.
- Glossitis: Inflammation of the tongue.
- Palatitis/ lampas: Inflammation of the palate
- Gingivitis: Inflammation of the mucosa of the gums.
- - FMD, BVD, MCF, IBR, Fusobacterium necrophorum – necrotic stomatitis in ruminants

- CD, leptospirosis, hepatitis, Uremia, pyometra – in Dogs

* Pemphigus vulgaris: autoimmune disease of dogs causing chronic bullous eruptions in mouth

PHARYNGITIS

- Inflammation of pharynx
- Paroxysmal coughing
- Cattle- Actinobacillus lignieressi, Fusobacterium necrophorum, IBR
- Horse- Strangles, EHV, parainfluenza virus
- Pigs – Anthrax
- **PHARYNGEAL OBSTRUCTION -** stertorous respiration, enlargement of retropharngeal lymph nodes

PHARYNGEAL PARALYSIS

- inability to swallow

Etiology: Peripheral Nerve injury,

- Guttural pouch infection in horses,
- Trauma,
- Rabies,
- Botulism,
- Central lesion.

Inability swallow and regurgitation

Roaring due to laryngeal paralysis

CUD DROPPING due to partial pharyngeal paralysis.

Aspiration pneumonia

Oesophageal obstruction

- Cattle: choke in cervical region
- Horses: thoracic part of oesophagus
- Barium radiography help in diagnosis

Gastric Dilation/ Volvulus

- stomach dilates with gas and/or fluid (known as “gastric dilatation”) and subsequently rotates around its short axis (known as “volvulus”).
- In most cases the stomach is rotated between 180 to 360 ° in a clockwise direction.
- Deep-chested breeds of dogs due to increased potential for rotational instability of the stomach.
- Abdominal radiography—a right lateral abdominal radiograph is the imaging modality of choice; a “double bubble” or ‘Popeye’s arm' sign like compartmentalized stomach is considered pathognomonic.

Ileus

- Ileus is a state of functional obstruction of the intestines or failure of peristalsis.
- paralytic ileus or adynamic ileus where there is loss of intestinal tone and motility as a result of reflex inhibition.
- Eg: in acute peritonitis, excessive handling of viscera during surgery, and prolonged and severe distension of the intestines as in intestinal obstruction or enteritis, acid base imbalances.
- Impaction of the large intestine in horses and infarction of intestinal wall following an acute mechanical obstruction will also result in ileus.

Jaundice

- yellow staining of tissues caused by excess bilirubin (hyperbilirubinaemia).
- Bilirubin: waste product of hemoglobin metabolism
- Hepatocytes conjugate the insoluble (indirect) bilirubin with various carbohydrates, (glucuronide) making the bilirubin water soluble (direct bilirubin) and excreted into duodenum through common bile duct.
- In the intestine it undergoes bacterial deconjugation to urobilinogen, which is mostly resorbed into the portal system and returned to the liver.
- fraction of urobilinogen is excreted in the urine and the remainder stays in the intestinal tract where it is converted to stercobilin, which imparts normal faecal colour.

Pre-hepatic jaundice (haemolytic jaundice)

- increased production of bilirubin; exceeds the normal concentration that the liver is able to take up and process.
- Intravascular haemolysis eg: babesiosis, chemical toxicity (zinc, paracetamol, onion etc), transfusion reaction etc.
- Extravascular haemolysis. Eg. Immune mediated haemolytic anaemia, oxidative injury etc.

Hepatic jaundice (intra-hepatic cholestasis or toxic jaundice): reduced elimination of bilirubin and other bile constituents due to impaired hepatocellular handling. Cholangiohepatitis, cirrhosis, amyloidosis, infectious hepatitis etc

Post-hepatic or obstructive jaundice: ineffective delivery of bilirubin into the duodenum (extrahepatic cholestasis) or bile leakage into the abdominal cavity (bile peritonitis). Melena or acholic faeces will be observed. Pancreatitis, Duodenitis, cholelithiasis, Lantana poisoning etc.

Van den Bergh reaction

- Direct positive van den Bergh reaction
  - A purple color appears immediately when diazotized sulfanilic acid reacts with conjugated bilirubin.
- Indirect positive van den Bergh reaction
  - A purple color appears within 30 minutes when diazotized sulfanilic acid and methanol react with unconjugated bilirubin.
- Biphasic van den Bergh reaction
  - A purple color appears immediately when the serum contains high concentrations of both unconjugated and conjugated bilirubin. The color intensifies further when alcohol is added.
- Obstructive/ post hepatic jaundice: direct positive
- Prehepatic/ Hemolytic: Indirect
- Hepatocellular: Biphasic positive
- Jaundice develops when bilirubin levels exceed 2.0 mg/dl to 3.0 mg/dl. Normal value in dogs is 0.1- 0.6 mg/dl

PANCREATITIS

- A 'hunched up' posture or praying posture
- Diarrhea and yellow, greasy stool

- Steatorrhea: increased fat excretion in stool
- Serum amylase and lipase activities
- TLI assay—TLI (trypsin like immunoreactivity) is pancreatic specific
- ELISA for trypsinogen activation peptide (TAP)

SIMPLE INDIGESTION

- dietary abnormalities including indigestible roughage, particularly when the protein intake is low, moldy ,overheated, and frosted feeds, and moderate excesses of grain and concentrate intake.
- Prolonged or heavy oral dosing with antimicrobials
- Changes in the pH of its contents markedly affect the motility of the rumen
- Over eating of grains: acidity
- High protein diet/ excessive legume fodders: alkalinity

CLINICAL FINDINGS

- A reduction in appetite is the first clinical finding, followed closely in milking cows by drop in milk production.
- Both occur suddenly; the anorexia may be partial or complete
- Rumination ceases and the ruminal movements are depressed in frequency and amplitude and sometimes are almost absent.
- doughy rumen

Treatment

- Ruminotorics
- Parasymapthominetics like physostigmine, neostigmine
- excessive quantity of grain is the cause of the simple indigestion, the use of alkalinizers, such as magnesium hydroxide is recommended when the rumen contents are excessively acid.
- Acetic acid or vinegar, 5 to 10 L, is used when the rumen contents are alkaline as a result of the ingestion of high-protein concentrates.
- Reconstitution of the flora by the use of rumen fluid transfers from healthy cows is highly effective.

Ruminal Tympany (Bloat)

- Normally, the bulk of this gas is eliminated by eructation or belching

- Bloat is the overdistension of the rumen and reticulum with gases derived from fermentation.

Frothy bloat (primary tympany)

fermentation gases are trapped in a stable, persistent foam which is not readily eructated. This type of bloat occurs most commonly in two settings:

- Animals on pasture, particularly those containing alfalfa or clover (pasture bloat). These legumes are rapidly digested in the rumen, which seems to results in a high concentration of fine particles that trap gas bubbles. Additionally, some of the soluble proteins from such plants may serve as foaming agents.
- Animals feed high levels of grain, especially when it is finely ground (feedlot bloat). Again, rapid digestion and an abundance of small particles appear to trap gas in bubbles.
- Pasture bloat: The vital factor is the frothiness of ruminal contents. The stable dispersion of feed particles are responsible for the frothiness.
- Feedlot bloat: Feedlot bloat is due to feeding finely ground grain. High carbohydrate content increases encapsulated bacteria that produce slimes. The slime entraps the gases of fermentation. Maximum stability of foam occurs at pH of about 6.
- Excessive green or legumes intake →Chloroplast released from the legume leaf forms monomolecular foams that trap gas bubbles → Frothy bloat (Pasture bloat)
- Saponin, pectin, hemicellulose – help in production of foam in rumen
- **Pectin methyl estrase** present in leaf hydrolyse pectin to polygalactouronic acid and acid pectin which possess gel formation property.
- Foam may block cardia and hence eructation

Free gas bloat (secondary tympany)

- occurs when the animal is unable to eructate free gas in the rumen.
- partial obstruction the esophagus (foreign bodies, abscesses, tumors) or interfere with rumenoreticular motility (i.e. reticular adhesions, damage to innervation of the rumen)
- A ruminant cannot eructate when lying on its back, and if a cow falls into a ditch and is unable to right itself, she will bloat rapidly.
- Saliva has **Antifoaming property due to mucin** which prevent froth formation by reducing surface tension. If volume, composition or rate of secretion of saliva is altered → possibility of **gel formation.**
- Low pH due to excessive CHO feeding help mucinolytic bacteria proliferation →bacteria also produce **insoluble slime due to mucopolysaccharide** which increase viscosity →froth formation

Clinical Signs:

- distention of the rumen compresses thoracic and abdominal organs. Blood flow in abdominal organs is compromised, and pressure on the diaphragm interferes with lung function.
- The cause of death is usually hypoxia due to pulmonary failure.
- Anorexia, overdistension of the left flank
- Protrusion of the paralumbar fossa above the ventral column and enlarged abdomen
- Signs of abdominal pain – kicking at belly, looking at flank, rolling on ground, grinding of teeth
- Dyspnea and grunting
- Dyspnea manifested by open mouth breathing, protrusion of the tongue, and extension of the head
- Treatment: antifoaming agents have been used to relieve frothy bloat.
- vegetable oils (corn, peanut) or mineral oil, which are administered in 100-300 ml volumes to cattle.
- A number of effective commercial products are available that include such agents as polaxalene (a surfactant) or alcohol ethoxylate (a detergent).

ACUTE CARBOHYDRATE ENGORGEMENT OF RUMINANTS /RUMEN OVER LOAD/ RUMINAL LACTIC ACIDOSIS

- Sudden ingestion of toxic doses of carbohydrate rich feed such as grain is the most common cause of the disease.
- Less common causes include engorgement with apples, grapes, bread, bakers dough, sugar beet, beverage waste etc.
- Toxic dose of carbohydrate - cattle- 25-62 g / kg body weight of ground cereal grain or corn.

PATHOGENESIS OF ACUTE LACTIC ACIDOSIS

- Changes in rumen microflora
- Ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2-6 hours by a marked change in the microbial population in the rumen.
- increase in the number of *Streptococcus bovis* which utilise the carbohydrate to produce large quantities of lactic acid.
- Continuous production of lactic acid result in the reduction of rumen pH to 5 or less, which results in the destruction of cellulolytic bacteria and protozoa

- The concentration of volatile fatty acids increases initially contributing to the decrease in the ruminal pH. The low pH allows lactobacilli to use the large quantities of carbohydrate in the rumen to produce excessive amount of lactic acid, resulting in ruminal lactic acidosis.
- increase ruminal osmolality, and water is drawn from the systemic circulation, causing haemoconcentration and dehydration.
- Ruminal osmolality increases from the normal of 280mosmol/L to almost 400mosmol/L
- As the rumen pH declines, the amplitude and frequency of rumen contractions are reduced and at about pH of 5, there is ruminal atony.
- The absorbed lactic acid is buffered by plasma bicarbonate buffering system.
- In sever cases of lactic acidosis, the reserves of plasma bicarbonate are reduced, the blood pH declines steadily
- blood pressure declines, causing a decrease in perfusion pressure and oxygen supply to peripheral tissues and resulting in a further increase in lactic acidosis from cellular respiration.
- High concentration of lactic acid in the rumen causes chemical rumenitis, which is the precursor of mycotic rumenitis in those that survive.
- This occurs 4-6 days later. The low pH of rumen favours the growth of Mucor, Rhizopus and Absidia spp. Which invade the ruminal vessels, causing thrombosis and infarction.
- Wide spread necrosis and gangrene may affect the entire ventral half of ruminal walls and lead to the development of acute peritonitis
- toxaemia resulting from the gangrene is usually sufficient to cause death.
- Hepatic abscesses commonly occur as a complication as a result of combination of rumenitis caused by lactic acidosis and allowing Fusobacterium necrophorum and Acranobacter (Corynebacterium) pyogenes to enter directly to ruminal vessels and spread to the liver.
- Laminitis
    - Laminitis occurs in acute, subclinical and chronic forms associated with varying degrees of severity of ruminal acidosis.
    - Vasoactive substances (histamine and endotoxins)are released during the decline of ruminal pH and bacteriolysis and tissue degradation.
    - These substances causes vasoconstriction and dilatation, which injure the microvasculature of corium.

CLINICAL FINDINGS OF LACTIC ACIDOSIS

- Depression, dehydration, inactivity, weakness, abdominal distension, diarrhea and anorexia are typical.
- Body temperature is usually below normal at 36.5°C–38.5°C (98°F–101°F)
- In general, the prognosis is better in those with heart rates below 100/min than those with rates up to 120-140/min.
- although low-pitched tinkling and gurgling sounds associated with the excessive quantity of fluid
- The pH of the rumen fluid is usually below 5.

Treatment

- Prevention of further access to feed and water
- Sodium bicarbonate: Systemic acidosis is treated with IV solutions of 5% sodium bicarbonate @v5L/450kg
- Antihistamines for laminitis, NSAID for shock therapy, thiamin or brewer's yeast to promote lactic acid metabolism,
- parasympathomimetic for stimulation of gut motility.
- Calcium borogluconate for hypocalcemia.
- Rumenotomy
- Rumen lavage

Subacute ruminal acidosis (SARA)

- disorder of ruminal fermentation in dairy cattle caused by the ingestion of large amounts of concentrates and inadequate amounts of fiber administered in order to increase milk production in early lactation
- Etiology- Decreased fibre & increased concentrates during periparturient period
- Clinical findings include laminitis, intermittent diarrhea, suboptimal appetite or cyclic feed intake, a high herd culling rate, loss of body condition in spite of adequate energy intake, liver abscesses
- **Milk-fat depression and suboptimal milk production**
- A decrease in dry matter intake is commonly reported in herds with SARA.

Reticuloruminal milk accumulation ("ruminal drinking")

- milk is ingested straight into the rumen because of a failure of the reticular groove reflex, resulting in ruminal acidosis in calves on a liquid diet.

- The reticular groove is a muscular structure extending from the cardia to the reticulo-omasal orifice.
- Its correct closure is a precondition for the direct passage of ingested milk or milk replacer into the abomasum.
- When the reticular groove partially or completely fails to close, milk spills into the reticulorumen and is fermented to short-chain fatty acids or lactic acid.
- recurrent ruminal tympany, inappetence, unthriftiness, and clay like feces.
- After being placed on milk diet and fed with a bucket.

Ruminal parakeratosis

- disease of cattle and sheep characterized by hardening and enlargement of the papillae of the rumen.
- It is most common in animals fed a high-concentrate ration during the finishing period.
- It also occurs in cattle that are fed rations of heat-treated alfalfa pellets
- calves with prolonged ruminal acidosis due to reticuloruminal milk accumulation ("ruminal drinking").

TRAUMATIC RETICULO PERITONITIS

- Hardware Disease
- caused by the ingestion and migration of a foreign body in the reticulum
- Perforation of the wall of the reticulum allows leakage of ingesta and bacteria, which contaminates the peritoneal cavity
- Initially, peritonitis is generally localized and adhesions takes place
- The object can penetrate the diaphragm and enter the thoracic cavity (causing pleuritis and sometimes pulmonary abscessation) and pericardial sac causing pericarditis

Clinical Findings

- anorexia, fever, tachypnea, tympany and an arched stance with abducted elbows
- Arched back; anxious expression; a reluctance to move; and an uneasy, careful gait
- A grunt produced by applying pressure to the xiphoid or by firmly pinching the withers (Xiphoid or Grunt test)
- Marked jugular pulse and brisket edema
- Death in TRP results from Cardiac Tamponade, severe toxemia and shock

CLINICAL PATHOLOGY

- leukocytosis(neutrophilia with a left shift) – band neutrophils more in number
- cattle also will show hyperfibrinogenemia, with fibrinogen concentrations greater than 1,000 mg/dL
- Hyperproteinemia
- TRP may include hypochloremia, hypokalemia, and metabolic alkalosis

Vagal indigestion- Hoflund syndrome, Chronic indigestion syndrome

- Functional disturbance of the fore stomachs and abomassum.
- Not one specific disease, this is a syndrome which causes rumen distention
- Paple shaped abdomen- apple-shaped on the left and pear-shaped on the right
- Transrectal palpation reveals an L-shaped rumen

TYPES OF VAGAL INDIGESTION

Based on the site of the functional obstruction

- Type I is failure of eructation or free gas bloat,
- Type II is a failure of omasal transport,
- Type III is abomasal impaction,

- Type IV is partial obstruction of the fore stomach.

TYPE I VAGAL INDIGESTION

- FREE GAS BLOAT - Partial esophageal obstruction due to foreign body or extra esophageal obstruction due to lymphosarcoma, thyroid tumors, lung abscess.
- FAILURE OF ERUCTATION - Inflammatory lesion adjacent to vagus nerve (localized peritonitis)

TYPE II VAGAL INDIGESTION

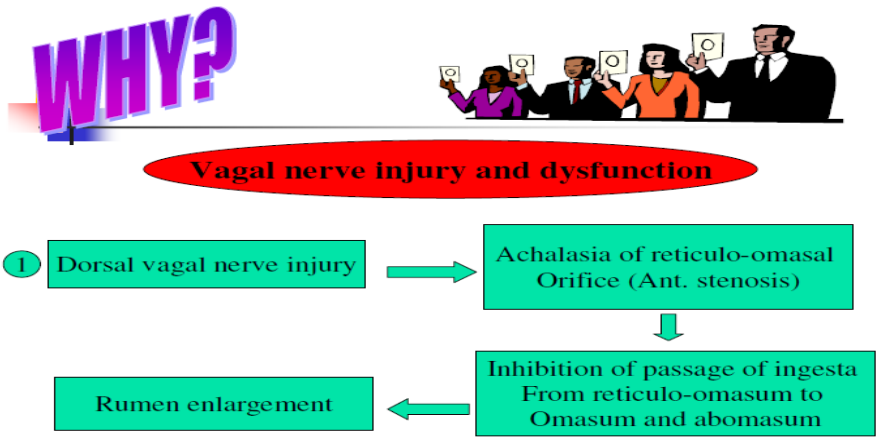
- FAILURE OF OMASAL TRANSPORT due to
- Abscesses
- Lymphosarcoma, Papilloma, Squamous cell carcinoma,
- Liver abscess exerting pressure of vagus.

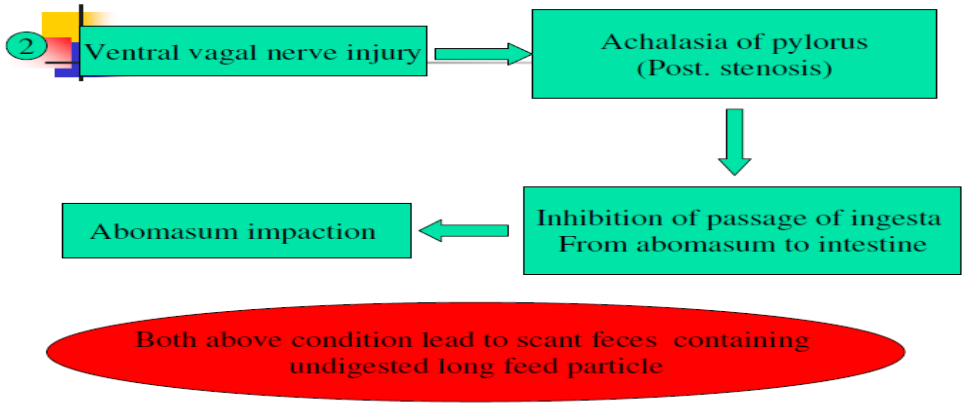
TYPE III VAGAL INDIGESTION

- ABOMASAL IMPACTION
- PYLORIC STENOSIS/OBSTRUCTIONS
- Traumatic reticulo peritonitis, Foreign body, Vagal neuritis

TYPE IV VAGAL INDIGESTION

- Most difficult type- uterus enlarges, abomasum pushed forward which interferes with normal motility





- proximal functional stenosis: passage of ingesta from the ruminoreticulum to the omasum and abomasum is impaired, resulting in rumen overload.
- distal functional stenosis: abomasal emptying is impaired, leading to abomasal overload and reflux of abomasal contents into the omasum and ruminoreticulum.
- Rumen chloride concentration - used to differentiate proximal stenosis (mean, 20 mmol/L) and distal stenosis (mean, 57 mmol/L).
- As a result of abomasal reflux, the abomasal contents get refluxed back into reticulorumen
- Causing dehydration, hypochloremic hypokalemic metabolic alkalosis, azotemia and significant increase in ruminal fluid chloride concentration

ATROPINE TEST

- Inj. Atropine sulphate 40 mg s/c
- A rise in heart rate of more than 20 beats/mt (>16%) in 10 minutes is suggestive of Vagal Indigestion

ABOMASAL DISPLACEMENT

- Abomasum - wandering organ due loose attachment with greater and lesser omentum
- Easily displaced to
  - left (left displacement of abomasum or LDA)
  - displace right (Right displacement of abomasum or RDA)
  - Abomasal volvulus

- RDA and abomasal volvulus cases are also commonly seen in the postparturient period (2-4 weeks after calving)
- association with calving is not as strong as with LDA – can occur at any stage
- LDA is much more common than RDA (30 LDA to 1 RDA);

LDA

- Slab sided abdomen
- Intermittent anorexia, gradual decrease in milk yield, rapid loss of body condition, dull, listless, shifting of weight from one leg to other due to abdominal pain, Scanty faeces, constipated.

RDA: Sunken eyeballs, scant, soft and dark colored feces.

- Abdominal pain with kicking at abdomen, depression of back and crouching are also recorded

1. Dilatation (displacement) phase

2. Volvulus phase (more severe)

Diagnosis

- Ping and Splash sounds
- Ping in LDA : between ribs 9 and 13 in the middle to upper third of the left abdomen
- ping associated with RDA:located in the area between ribs 10 and 13 on the right abdomen
- ping cranial to rib 10 usually indicates the presence of abomasal volvulus
- Liptik test: distinguish between rumen and abomasal ping (pH 2-4 means abomasum and 6-7 means rumen

Treatment

- Treatment with 500 ml of 25% calcium borogluconate
- early treatment with fluids and electroytes intravenously and orally mineral oil(5-10 litres/day orally)
- magnesium hydroxide (500gm per adult cow orally every 2 days) are used.

Surgical /laparoscopic treatment:

A- Blind/ Closed:

1- Rolling

2- Rolling + Toggle pin/Bar suture fixation

B- Opened:

1 *Standing: Left & right flank*

- Flank Abomasopexy (Utrecht technique/ Dirksen technique)
- Flank Omentopexy
- Flank Omento-abomasopexy

2 *Recumbent:* Median Abomasopexy, Paramedian Abomasopexy Paracostal Abomasopexy

FMD (Foot and Mouth Disease)

Genus : *Aphthovirus* Family : *Picornaviridae*

- 7 serological types:
- Type O
  - Type A
  - Type C- not reported since 1995, not present in vaccines now
  - Asia 1
  - South African Territories (SAT) 1
  - (SAT) 2
  - (SAT) 3
  - In India O,A,C, Asia-1
  - O is the most common cause of FMD outbreaks in India

Virus properties

- inactivated outside the pH range 6–9 and by desiccation and at temperatures >56°C.
- 56°C for 30 minutes is sufficient to inactivate most strains of FMD virus
- FMD virus can survive for
  - up to 20 weeks on hay or straw bedding
  - in dry fecal matter for up to 14 days in summer
  - in fecal slurry for up to 6 months in winter

- in urine for 39 days
- in soil for 3 (summer) to 28 (winter) days.

Structure

- structural proteins of the virus (termed VP1, VP2, VP3, and VP4)
- proteins VP1, VP2, and VP3 are exposed on the outside surface of the virus, whereas VP4 is entirely internal

Host range

- virus primarily affects cloven-hoofed animals of the order Artiodactyla. Livestock hosts include cattle, pigs, sheep, and goats.
- Cattle>Pigs > Buffalo>Sheep>Goat> camels
- Wild animals : Deer, elephant, impala, kudu, tapirs, llama, alpaca, antelope, hedgehogs, porcupines, kangaroos, guinea pigs.
- Resistant animals : Horse, donkey, mule, camel
- Pig- Amplifier Host (can expire up to 3,000 times more virus than cattle)
- Sheep& Goat- Maintenance host
- Cattle – indicator host

Transmission

- transmitted via direct contact with infected animals or indirect contact with secretions or excretions (including semen and milk) from infected animals or by mechanical vectors (people, horses, dogs, cats, birds, vehicles) or air movement over land or water.
- inhalation, ingestion, or through skin wounds and mucous membranes. Breeding is a possible route of transmission for the SAT viruses in African buffalo populations.
- respiratory system - major route of infection in ruminant species & pigs, but pigs are much more susceptible to infection by the oral route than are ruminants.
- Virus was reported to travel over water >250 km (~150 miles) from Brittany, France, to the Isle of Wight, UK, in 1981, but it usually travels no more than 10 km (~6 miles) over land.
- most favorable cells for reproduction virus is middle layer of stratum spinosum(Epidermal cells)
- Excretion of the FMD virus can begin up to four days before clinical disease becomes apparent
- Most excretion of the virus ceases four to six days after the appearance of vesicles, when circulating antibodies develop.

- The virus tends to persist in foot lesions for a day or two longer than in mouth lesions, so that foot lesions may be a better source of virus for diagnostic purposes in older cases.
- The FMD virus has been detected in the milk and semen of experimentally infected cattle for 23 and 56 days respectively.

Clinical signs

- The first indication of the disease is a fever (to 42°C), which is accompanied by severe depression, inappetence and a sudden cessation of milk production.
- This is followed within a day or so by the development of vesicles, the predilection sites for which are the tongue, lips, gums, dental pad, nares, interdigital skin of the feet, coronary bands, bulbs of the heels and teats of milking animals.
- Lesions very quickly progress to vesicles initially 1-2 cm in diameter but that rapidly enlarge and often coalesce. They are filled with a clear straw-coloured fluid and their overlying epithelium is blanched. The vesicles rupture within 24 hours to leave raw, painful ulcers surrounded by ragged tags of necrotic epithelium.
- Early signs of FMD in pigs include fever, inappetence and reluctance to move. The most pronounced vesicles are on the feet.
- Cattle: profuse salivation, the saliva hanging in long, ropy strings, a characteristic smacking of the lips and drooling of saliva
- Vesicles may also develop on the teats, which results in severe mastitis.
- Morbidity is high (reaches 100%) and mortality is very low (less than 2%)
- cause of death in young ones – acute myocarditis and Gastroenteritis
- Tigroid heart appearence on necropsy

Approximate age of lesions	**Appearance of lesions**
1 day	Unruptured vesicles containing some fluid, early signs of necrosis in overlying epithelium
1-2 days	Unruptured, fluid-filled vesicles, overlying epithelium necrotic
1-3 days	Vesicles ruptured, erosions present and ragged pieces of epithelium adhering to the margins of the lesions. In the earlier phase, the exposed centre of the lesion is bright red; later the redness begins to change as fibrin deposition occurs

- 4 days-1 week — Erosions with little epithelium attached, margins of lesions becoming "smoother" (no longer ragged) because of early healing with regrowth of epithelium at the edge of the lesion
- 7-10 days — Healing advanced with fibrous tissue formation

Hairy Panters

- -sequelae to FMD in cattle
- -due to endocrine damage, is a chronic syndrome of dyspnea, deleterious effects on testes
- -causing production of poor quality of semen, anaemia, overgrowth of hair and lack of heat tolerance
- The maximum reported carrier periods for different species are three and a half years for cattle; nine months for sheep; four months for goats; and five years or more for African buffaloes.
- virus can be recovered intermittently from such animals by oesophageal-pharyngeal (OP) probang collections

Diagnosis

- Complement fixation test (CFT) - important test to identify the type of virus
- indirect ELISA – DIVA strategy – non structural proteins antibody test
- VNT and ELISA are OIE-prescribed tests for international trade.

FMD Control Program

- (started in 2003-04
- ring vaccination in case of outbreaks
- vaccine strains – O, A, Asia-1
- inactivated tissue culture oil adjuvant vaccine - 4 months onwards
- Estimated national loss due to FMD – Rs.20,000 crore per annum
- overall aim of the National Animal Disease Control Programme for FMD and Brucellosis (NADCP) is to control FMD by 2025 with vaccination and its eventual eradication by 2030

ANTHRAX
(Splenic fever/wool sorter's disease/Carbuncles)

- noncontagious zoonotic disease caused *Bacillus anthracis*.
- gram-positive, aerobic, spore-forming bacillus.

- Characterized by septicaemia, and sudden death with the exudation of dark tarry coloured and un-clotted blood from natural orifices
- The endospores of *B. anthracis* can survive for decades in soil
- Two parts of bacteria – capsule and body; Capsule contains Glutamyl Peptide- contains fibrinolysin property- prevent blood clotting, Anti phagocytic activity, Spreading factor
- **polyD- glutamic capsule** encoded by virulence plasmid **pX02**, and a tripartite toxin comprised of edema (factor) lethal (factor II) and protective antigen (factor III) encoded by plasmid **pX0l**.
- *B anthracis* in Category A (highest priority), and it has been manufactured as a biological warfare agent

Host affected

- Wild and domestic herbivores (eg, cattle, sheep, goats, camels, antelopes).
- People exposed to tissue from infected animals, to contaminated animal products, or directly to *B. anthracis* spores under certain conditions.
- The disease is almost invariably fatal except in Swine
- **Algerian sheep** are said to be resistant.
- Absent in birds

Transmission

- Ingestion,
- Abrasions
- Inhalation(wool sorter's disease)
- biting flies may mechanically transmit *B anthracis* spores from one animal to another
- Contaminated bone meal, meat or hay with contaminated soil

Pathogenesis

1. B. Anthracis endospores (abrasion, inhalation, or ingestion)
2. Phagocytised by macrophages and carried to regional lymph nodes.
3. Within hours, most spores germinate into vegetative form & produce s virulence factors and enter the bloodstream.
4. For virulence : antiphagocytic capsule and 3 toxin components: (Protective antigen, Lethal factor, Edema factor)

5. The three exotoxin components combine to form two binary toxins, oedema toxin and lethal toxin

6. Oedema toxin is a calmodulin-dependent adenylate cyclase that alters water homeostasis causing oedema and impairs neutrophil function, rendering the host further susceptible to infection.

7. Lethal toxin leads to the release of reactive oxygen intermediates as well as the production of pro inflammatory cytokines, tumour necrosis factor and interleukin-1b responsible for rapid circulatory collapse.

8. The release of these toxins results profuse haemorrhage, tissue oedema, and a paucity of acute inflammatory reaction .

The typical incubation period for anthrax is 3–7 days (range, 1−14 days).

- Depends on species affected, dose and the route.
- In cattle and sheep it is septicaemic and rapidly fatal, most animals are found dead without pre-monitary signs.
- Pyrexia with temperatures up to 42°C (108°F), depression, rumination ceases, milk production is materially reduced, congested mucosae and petechiae may be observed ante-mortem.
- Animals which survive for more than one day may abort or display subcutaneous oedema and dysentery.
- In pigs lesions localized to tonsils or cervical lymph nodes and surrounding tissues.
- Rigor mortis is frequently absent or incomplete.
- Dark blood may ooze from the mouth, nostrils, and anus
- An extremely large soft spleen is characteristic of the disease in cattle.

Diagnosis

- History of sudden death and oozing of unclotted tarry blood from natural orifices.
- Demonstration of organism through ear vein/Spleen of carcass.
- McFadyean Reaction: capsule pink bacteria blue
- Serological tests like Ascoli’s test, a precipitation test.
- Agar gel precipitation test.

Control

- Infected animal is isolated and treated separately.
- Strict quarantine measures.

- Proper disposal of carcase (Burning/Burial). In burial, a deep pit is dug and layered with 6-12 inches of limestone/Calcium carbonate to prevent spore formation by releasing Co2 through reaction with putrefied carcass. Then carcase is placed and layered again with 6 inches of lime and pit is closed with soil.
- Vaccination: Alum Precipitated Anthrax spore vaccine (Sterne's spore vaccine)
- For cattle/sheep/Goats-1ml, camel-2ml, Elephant-3ml

Haemrrhagic Septicemia
(Shipping/transport fever, stockyard's disease, Barbone disease, Galaghotu in Hindi.)

- An acute highly fatal disease characterized by acute septicaemia, high fever, swelling at the ventral aspect of throat , neck and brisket causing dyspnoea
- Pasteurella multocida type 1 or B, G –ve, Known as **Bipolar organism**
- B:2 and E:2 in the Carter Heddleston serotyping system, or 6:B and 6:E, respectively, in the alternative Namioka Carter system.
- Buffalo>Cattle>Pig>Horse>Sheep & Goat
- Affect any age and group
- Maximum occurrence- During Monsoon
- Precipitating factors: Stress (Transportation, Heavy worm, Starvation and Viral infections like IBR, Parainflenza-3)

Transmission

  - Ingestion or inhalation
  - The initial site of proliferation –tonsillar region.
  - The source of infective bacteria is thought to be the nasopharynx of bovine carriers.
- Infection occurs by contact with infected oral or nasal secretions from either healthy carrier animals or animals with clinical disease, or by ingestion of contaminated feed or water. Infection begins in the tonsil and adjacent nasopharyngeal tissues.
- Organism in environment → enters terminal bronchioles and alveoli→ changes in lungs in presence of predisposing factors→ destroying the W.B.C.s and macrophages→ releases histamines and PGF2alpha and fibroblastic elements→ leading to septicemia in body and inflammatory changes in lung parenchyma--→ produce pneumonia with the help of secondary invaders (Parainfluenza-II, Bovine herpes virus and other bacteria) → Death due to asphyxia
- High Fever (106-107º F), Profuse salivation, Petechiae om mucousa.

- Edematous swelling (Under throat, neck and brisket region) which are hot and painful
- Increased respiration, Grunting sounds followed by dyspnoea & Death
- In peracute cases of hemorrhagic septicemia that result in death within 8–24 hours, animals often have fever, hypersalivation, nasal discharge, and labored respiration; however, because of the short duration of disease, these clinical signs may easily be overlooked.
- Acute disease can persist for up to 3 days, and less often up to 5 days, and is characterized by fever of 104°–106°F (40°–41.1°C), apathy or restlessness and reluctance to move, hypersalivation, lacrimation, nasal discharge that begins as serous and progresses to mucopurulent. Subcutaneous swelling in the pharyngeal region that extends to the ventral neck and brisket (and sometimes the forelimbs), progressive respiratory distress, cyanosis, terminal recumbency, and sometimes abdominal pain with diarrhea are also seen

Diagnosis

- Clinical findings
- Seasonal occurrence make tentative diagnosis
- Animal inoculation
- Serological (HAT,HIT,CFT etc.)
- Treatment:
    - Various sulfonamides (130-150 mg/Kg, IV for 3-5 days), tetracyclines, penicillin and chloramphenicol are effective if administered early.
    - Anti-inflammatory
    - Antihistamins
    - Supportive therapy

Prevention is by vaccination.

- Three kinds of vaccine are widely used: plain vaccine (HS broth vaccine),
- alum-type precipitated vaccine,
- oil-adjuvant vaccine.
- The most effective bacterin is the oil-adjuvant-one dose provides protection for 9-12 month; administered annually.
- The alum-precipitated-type bacterin is given at 6 months intervals.
- Age of vaccination: Above 5-6 months of age

Pasteurellosis of Sheep and Goats

- ***Pasteurella multocida***
- ***Mannheimia haemolytica* (formerly *Pasteurella haemolytica*)**
- ***Bibersteinia trehalosi***
- *M. haemolytica* is a normal inhabitant of the upper respiratory tract of sheep. most common strain isolated from sheep and goat respiratory pasteurellosis is *M haemolytica* A2
- Pneumonic pasteurellosis - seen in all age groups.
- Septicaemic pasteurellosis - very young lambs
- systemic pasteurellosis in weaned lambs
- Mastitis in ewes

Toxins

- Endotoxin, leukotoxin, and capsular polysaccharide
- leukotoxin - toxic to ruminant leukocytes, resulting in fibrin deposition in lungs and on pleural surfaces
- lipopolysaccharide endotoxin - adverse reactions in the lungs and systemic circulatory failure and shock
- Capsular polysaccharide - prevents the phagocytosis of the bacteria and assists in attachment to the alveolar epithelial surface

Pneumonic pasteurellosis: sudden deaths in lambs from the septicaemic pasteurellosis and progress to pneumonic pasteurellosis in the ewes and also in the lambs as they get old.

Septicaemic pasteurellosis*:* young lambs from 2 days to 2 months of age but most commonly seen at 2-3 weeks. *M. haemolytica* biotype A causes this disease.

Systemic Pasteurellosis*:* *M.haemolytica* biotype B predominantly in weaned lambs from 5-12 months of age

- *Pasteurella multocida* has been reported to be isolated from polyarthritis in young lambs
- *Mannheimia haemolytica* has been reported from cases of mastitis, especially in sheep.

Pasteurellosis in Pigs

- *Pasteurella multocida*

- normal inhabitant of the porcine upper respiratory tract
- produces an exudative bronchopneumonia, sometimes with pericarditis and pleuritis.

Listeriosis

- Synonyms: Circling disease(Sheep), Silage Sickness
- Etiology: *Listeria monocytogenes- intracellular*
- ability to grow at 4°C is an important diagnostic aid (the "cold enrichment" method) for isolation of the organism from brain tissue but not from placental or fetal tissues
- Host affected: Affects domestics animals especially ruminants
- *Encephalitis* is the most readily recognized form of listeriosis in ruminants with unilateral paralysis.
- *Septicemic or visceral listeriosis* is most common in monogastric animals, including pigs, dogs, cats, domestic and wild rabbits, and many other small mammals.

Clinical Symptoms: 3 forms are recognized:

1. Nervous
2. Septicemic
3. Abortive

- Affected animals shows usually, *meningoencephalitis* (common in adult ruminants), *septicaemia* (Common in monogastric and young animals) and *abortions*.
- In cattle and sheep: keratoconjunctivitis and iritis (ocular listeriosis), often unilateral and have been attributed to direct contact with contaminated silage.
- Abortions are recorded in cattle, sheep and goats (can occur at any stage but most commonly 7 month onwards) but rarely in pigs.
- Anton's eye Test- For Demonstrating L. monocytogens

TUBERCULOSIS

Kshaya roga, Pearl's disease, consumption, Scrofula

- infectious disease of all domestic animals and human beings caused by acid fast bacteria *Mycobacterium bovis* / *M. tuberculosis* / *M. avium* and is characterized by emaciation, progressive development of tubercles in any of the organs.
- *Mycobacterium bovis* - Primary host is cattle and secondary host can be any species of domestic animals and human beings.

- *Mycobacterium avium* - Primary host is birds and secondary host can be pigs, cattle, horse, sheep etc.
- *Mycobacterium tuberculosis* - Primary host is human beings and secondary host can be pigs, monkeys, dogs, cattle, etc.
- **Robert Koch** has identified the causative agent of TB and in honour of him every year 24th March will be celebrated as **World tuberculosis** day

Culturing

- Dorsett's egg medium or Petragnani's medium or Lowenstein-Jensen medium.
- **MGIT BACTEC 460 TB** (Mycobacterium Growth Indicator Tube) System is gold standard for TB bacterial drug sensitivity and culture

Transmission

- Infected cattle are the main source of infection for other cattle
- source of infection is exhaled air, sputum, faeces, milk, urine, vaginal and uterine discharges and discharges from the opened peripheral lymph nodes.
- Commonly entry is effected by inhalation or ingestion. It can be transmitted through coitus or A.I.
- drinking of infected milk by young animals is a common method of transmission where the disease is endemic.

Pathogenesis

- **Primary complex** - In the primary complex the portal of entry and the regional lymph nodes are affected and the lesions are seen.
- If the entry is through inhalation, these types of lesions are seen in the lungs.
- If the portal of entry is through ingestion, this type of lesion is seen in GI tract (but not in cattle).
- Calcification of the lesions commences about 2 weeks later

Post primary dissemination

- Organisms from the primary complex can get into the blood directly or through the lymphatics.
- Then through the circulation the organisms go to various tissues and organs. Here commonly miliary tubercles are produced.
- In pigs, localization as non-progressive abscesses in the lymph nodes of the head and neck is the most common finding.

(l) Nodular tubercle which will be greyish white in colour and bigger in size.

(2) Miliary tubercle which is small in size and appear like a millet seed. commonly seen in generalized TB.

- Nodules group together on the pleura or peritoneum and appear like bunch of grapes or a chain of pearls.
- '**Pearl's disease**' is used in TB in cattle. This is the pathognomonic lesion of tuberculosis in cattle.

Clinical findings

- ***Cattle:*** Emaciation**,** Inconsistent appetite and fluctuating temperature. Coughing - slow, suppressed and moist
- Then due to enlargement of lymph node and its pressure on the different organs, the following can be seen. :
- Bronchial lymph node - dyspnoea
- Mediastinal lymph node - persistent and recurrent tympany
- Retropharyngeal lymph node - dysphagia and noisy breathing.
- **Mastitis:** characteristic feature is a marked induration and hypertrophy which usually develops first in the upper part of the udder, particularly in the rear quarters. Enlargedsupramammary lymph nodes
- **Sheep and Goats:** broncho-pneumonia
- **Horses**: involvement of the cervical vertebrae in which a painful osteomyelitis causes stiffness of the neck and inability to eat off the ground.

Necropsy findings

- **Cattle:** Nodular or miliary tubercles in lungs, pleura, liver, peritoneum, kidney, lymph nodes and osteomyelitis of ribs, vertebrae and flat bones of pelvis. Bunch of grapes like lesions on the pleura and peritoneum.
- **Buffaloes:** Similar to cattle but calcification is uncommon.
- **Horses:** The lesions appear like sarcoma and rarely caseation or calcification is seen. Primary complex is in the alimentary canal.
- **Sheep and goat:** Very similar to cattle.
- **Pigs:** Can be localized or generalized. Primary complex is alimentary or respiratory tract.

Diagnosis

- Serological tests: CFT, FAT, Precipitation, HAT, HIT and ELISA have been used for the diagnosis of TB.
- Single intradermal tuberculin test: purified protein derivative (PPD) used
- Stormont test
- Short thermal test
- Anergic animals are those with visible lesions of tuberculosis but which do not react to a cutaneous, delayed hypersensitivity test
- False-negative reactions may be given by:
    - Advanced cases of tuberculosis
    - Early cases until 6 weeks after infection
    - Cows which have calved within the preceding 6 weeks
    - Animals desensitized by tuberculin administration during the preceding 8-60 days
    - Old cattle
    - Low-potency tuberculin or bacterial contamination of the tuberculin
    - Variable dose with multidose syringes.

Treatment & Prevention

- Anti tubercular drugs
    - Isoniazid
    - Streptomycin
    - Rifamycin
    - Paramino salicylic acid
    - Ethambutol
    - Pyrizinamide
- Test and slaughter policy
- Bacille calmette and Guerin (BCG) vaccine - attenuated vaccine

PARATUBERCULOSIS (Johne's Disease)

- infectious disease mainly of cattle and also of sheep and goats, caused by *Mycobacterium avium* subspecies *paratuberculosis* (MAP)
- Characterized by progressive emaciation of all species affected and chronic diarrhoea and thickening and corrugation of the intestine in cattle.
- Incubation period is usually years
- The primary site targeted by Johne's disease is the ileum (M cells of Peyer's patches)

Transmission

- The animals affected are - mainly cattle, and followed by buffaloes, sheep, goat, camel, Llamas, yaks, deers, reindeers and antelopes.
- Ingestion of contaminated feed and water
- Intra-uterine transmission - from dam to the foetus possible
- Nursing from an infected dam (via contaminated teats or direct shedding of the organism into the colostrum/milk) or ingestion of faecally contaminated solid feed and water.
- Usually young ones are highly susceptible and they pick up the infection. But when they become 2 years and above of age, they exhibit the clinical manifestations of the disease.

Pathogenesis

- Organisms enter the mucosa and sub-mucosa of the intestine.
- Attract lot of mononuclear cells & macrophages which fuse to form epithelioid cell
- Results in syncytia formation and stops at symplasma stage.
- no caseation, calcification and encapsulation in cattle.
- But in sheep, caseation is commonly seen and calcification is seen in 25% of the cases.
- Because of the massive infiltration of macrophages in mucosa and submucosa, thickening occurs and mucosa is thrown in the form of folds resulting in corrugations resembling that of brain. This is the pathognomonic lesion.

Clinical findings

- Chronic diarrhoea which is not responding to the commonly used antibiotics is the most important clinical sign.
- The faeces are of the consistency of thick pea soup
- Excessive mucous, shreds of epithelium, blood/blood clots are absent.

- Immediately after parturition diarrhoea will start. Bottle jaw, progressive emaciation, weakness, dehydration and finally prominent rib cage with marked sunken eye balls will be seen and then death.
- In sheep and goat, progressive emaciation and shedding of wool are seen along with Diarrhoea of less severity.

Necropsy findings

- mucosa of the intestine is thickened 3-4 times and there will be corrugations on the mucosa.
- Most commonly these are seen on the ileo-caecal valve and can be seen on ileum, caecum and colon and in advanced stages from duodenum to rectum.

Diagnosis

- Rectal pinch method
- By lesions: Presence of corrugations on the mucosa of the intestines is a pathognomonic lesion of Johne's disease.
- Serological tests: CFT is commonly used and will detect 90% of the cases, FAT, AGPT, ELISA
- Johnin test

Control

- Testing the herd with johnin test and culturing and disposing of the positive cases
- Vaccination:
  - Vallee's vaccine: It is a live non-attenuated vaccine
  - Sigurdsson's vaccine: It is a old vaccine and contains killed organisms
  - Latest vaccine: In 1982, a vaccine containing live attenuated organisms along with oil of paraffin and pumice stone

Brucellosis
(Contagious abortion, Bang disease)

- Brucella caused by Gram-negative coccobacilli, strict intracellular
- It is a zoonotic disease, primarily affecting goats, sheep, cattle, buffaloes, pigs and other animals and transmitted to humans by contact with infected animals or through ingestion of their products.
- Species resistant to brucella- Cats

- Genus Brucella encompasses 9 recognized spp—6 terrestrial sp. & 3 marine spp.
- Terrestrial sp. are B.melitensis, B.abortus, B.suis, B.canis, B. ovis, B.neotamae.

-Marine spp are B.delphini, B. pinnipediae (seals), B. cetaceae (whales & dolphins)

- B.abortus- Cattle, buff
- B.Melitensis- sheep, goat (zoonotically most important)
- B.ovis – sheep (non zoonotic)
- B.suis – Pigs
- B.canis- dogs
- B.neotamae – rodents (non zoonotic)
- -Brucellae are strict aerobes.

-Br. Abortus is capnophilic, many strains requiring 5-10% C02 for growth.

- The media employed currently are Farall's media, serum dextrose agar, serum potato infusion agar, trypticase soy agar, or tryptose agar.
- **Erythritol** has a specially stimulating effect on the growth of Brucella
- NOTE: Erythritol - sugar present in foetal and placental tissue, gravid uterus, seminal vesicle and Testes
- Two important surface antigens are abortus antigen A and melitensis antigen M.
- The proportion of A : M antigen
- Brucella abortus- 20:1
- Brucella melitensis:1:20
- Koster's stain is mostly useful in demonstrating Brucella in smears from the cotyledons in bovine abortion

Transmission

1. Oral entry : Ingestion of contaminated animal products (often raw milk or its derivatives). contact with contaminated fingers.

2. Aerosols: Inhalation of bacteria. Contamination of the conjunctivae.

3. Per cutaneous infection: through intact skin or abrasions or by accidental inoculation.

4. By artificial insemination where in the bull is infected and the organisms are found in the semen.

5. Congenital infection may occur in calves born from infected dams.

Pathogenesis

- Intracellular location & survival of the organism contribute to its virulence & pathogenesis.
- organisms are phagocytosed by PMNs in which some survive and multiply.
- transported to lymphoid tissues and foetal placenta.
- Enter the body and then get into the blood and localizes in the pregnant uterus, udder, testicles and accessory male sex glands, lymph nodes, joint capsules and bursae
- Upon invading the gravid uterus, it spreads to the uterus and causes severe **ulcerative endometritis of intercotyledonary space**.
- Then it invades the allantochorion, foetal fluids and placental cotyledons and results in **destruction of the villi**.
- Because of this abortion occurs in 5^{th} month or more than 5^{th} month of gestation

Clinical symptoms

- Abortion is the most obvious manifestation (abortion storm in last trimester)
- Infection cause stillborn or weak calves, retained placentas reduced milk yield.
- Usually, general health is not compromised in uncomplicated abortions.
- Seminal vesicles, ampullae, testicles, and epididymis may be infected in bulls; therefore, organisms are present in the semen.
- Testicular abscesses may occur in male
- Long standing infections may result in arthritic joints fluid filled knee in some cattle.
- Retention of the placenta and metritis are common sequelae to abortion.
- reproductive failure, such as abortion or birth of unthrifty newborn in the female
- Orchitis and epididymitis with frequent sterility in male
- Persistent infection is characteristic with shedding in mammary and reproductive secretions
- B. abortus in Horse- Fistulous withers/ poll evil
- B. Ovis in sheep - Acute orchitis & epididymitis in Rams
- Necrotic, leathery placenta in brucellosis

Diagnosis

- Culture and isolation: Culture The 'gold standard' diagnostic test continues to be based on isolation and characterization of the organism from the placenta & organs (Stomach, lungs) and lymph nodes of the fetus.
- Rose Bengal Plate test- Slide agglutination---- Screening / Qualitative test
- **Rapid plate agglutination test (RPAT):** screening test used for the diagnosis of Brucellosis in a herd. This test is first conducted by using the serum and if this test is positive, then Standard Tube Agglutination Test (STAT) is performed to confirm Brucellosis previously. Now a days ELISA is being used as a confirmatory test.
- **Standard tube agglutmation test (STAT):** After the RPAT is positive and then this test is used. Then the following criteria are used to differentiate vaccinated animals from infected i.e., non vaccinated animals
- Complement Fixation Test
- Brucellin test: SID
- Strauss reaction: Development of orchitis in G. pig after intraperitoneal injection of infective material.
- **Rivanol and 2-mercaptoethanol test**

  - Here the suspected serum is mixed with either rivanol or 2-mercaptoethanol and kept for some time. Then the STAT is done.

  - Here these agents will destroy the IgM. That means the level of IgG is measured in these tests.

  - This test is used to differentiate the vaccinated from infected animals. If the elevated titre is due to IgM then it is considered as vaccinated animal and if the elevated titre is due to IgG then it is considered as infected animal.

- Milk ring test – herd screening test
    - This is a frequently used serological test for demonstration of antibodies in the milk of an animal.
    - This is a screening test used to detect the presence of Brucella infection in infected cattle.
    - In a positive test, if antibodies are present in the milk, the bacilli are agglutinated and raised with the cream to form a blue ring at the top, leaving the milk unstained.
    - In a negative test, the milk remains uniformly blue without formation of any colored ring.

Vaccination

- Cotton strain S-19 vaccine: calfhood vaccination of 4-8 months Females only of B.abortus Live attenuated vaccine.

* Not injected in males- bacteria will settle in testes and make the animal permanent carrier of brucellosis.

* Rose Bengal-51 (RB-51) vaccine: live B.abortus in 4-12 months of age

*** Killed Brucella Vaccine (Duphavac):** K45/20A strain has been used with adjuvant

* Sheep & Goats: live B.melitensis Rev-1

RINDERPEST
(RP, Mata, cattle plague)

- acute highly contagious disease of ruminants and swines caused by a paramyxo virus and it is characterized by high fever and focal erosive lesions mostly confined to the mucosa of the alimentary tract.
- Most cloven-hooved animals (order Artiodactyla) are susceptible
- **Etiology:**
- Family: Paramyxo viridae
- Genus : Morbilli virus
- Species: RP virus, PPR virus, CD virus, Measles virus
- Many strains of RP virus are available with varying pathogenicity. All the strains are almost antigenically identical. Hence one strain gives protection against the other strain.

Transmission

- Most important mode of transmission is by **close contact between** infected and non infected animals.
- ingestion of contaminated feed and water and by inhalation
- The source of infection are all the secretions and excretions of the affected animal i.e. saliva, nasal discharge, tears, urine and faeces etc.

Pathogenesis

- virus enters the body through naso-pharyngeal mucosa, goes to the tonsils then to regional lymph nodes.
- It replicates and through the lymph enters the blood in mononuclear cells.

- through circulation the virus localizes in the epithelial tissue like the oral mucosa and the gastro-intestinal tract & causes congestion and hemorrhages
- haemorrhages and erythema running transversely across the colonic mucosa and on the rectal folds of mucosa will be clearly visible and gives an appearance of "zebra markings" which is a pathognomonic lesion of R.P.
- pin point greyish-white necrotic patches (1-5 mm) in the oral cavity and abomasum and the intestines. These lesions are seen on the tongue especially on the ventral aspect of the tongue and they appear like as though sprinkling of the bran on the mucosa.
- These will be sloughed off leading to formation of erosion and later on small ulcers which can coalesce together to form large ulcers.

Clinical Signs

- moderate fever like 104°F, depression, anorexia, restlessness, dry muzzle and constipation. Within 1-2 days nasal and lacrimal discharges appear which will be in the beginning serous like and later on becomes mucoid or mucopurulent. There will be excessive thirst, photophobia, leucopenia and rough hair coat. By 2nd or 3rd day oral lesions appear and salivation is seen. The type of salivation is drooling salivation. The temperature goes on increasing and by 3rd or 5th day it can be 106-108 °F.
- diarrhoea starts and as a result of which the temperature drops down and can be normal and at the ending stage it will be subnormal. Later on there will be dysentery. The animals will be extremely dehydrated, weakness, sunken eyeball, marked depression, subnormal temperature, recumbency, coma and death.
- Pregnant animals can abort 3-6 weeks after onset of illness. The course of the disease is 6-12 days.
- **Sheep and Goats:** The clinical signs seen are fever, discharges from the eyes and nostrils, salivation, diarrhoea, dysentery and pneumonic signs. The oral lesions are not commonly seen.
- **Skin form:** is also being recorded wherein the systemic signs are absent and small pustules are seen on the neck, inside the thighs and scrotum.

Necropsy findings:

- **Erosions in mouth and erosions and haemorrhages in the abomasum and intestines.**
- **Zebra marking in rectal mucosal folds.**
- **Crater like lesions in peyer's patches.**

Eradication Program

- NREP: started in May 1992 and provisionally declared RP free in 1998

- OIE recognised India RP free 2004
- The eradication campaign concluded in 2011 with an international declaration of global freedom from rinderpest

PESTE DES PETIS RUMINANTS
(PPR, Goat Plague or Kata)

- acute, highly contagious viral disease of goats and sheep caused by a morbillivirus (Paramyxovirus) and is characterized by fever, anorexia, necrotic stomatitis, diarrhoea, oculo-nasal purulent discharge and respiratory distress.
- PPR is caused by a morbilli virus (belonging to the paramyxoviridae family) and is closely related to rinderpest virus, canine distemper virus and measles virus
- Morbidity rates - 50-90%. The case fatality rates are higher in goats (55-85%) than in sheep (less than 10%).
- Goats are more susceptible to PPR than sheep and kids over 4 months and less than 1 year of age are most susceptible.

Transmission

- Infection is mainly by **inhalation** and also can occur through the conjunctiva and oral mucosa.
- Insects are not regarded as vectors of the virus.
- Large amounts of the virus are present in all body excretions and secretions of the affected animals.

Pathogenesis

- PPR virus penetrates the retropharyngeal mucosa and enters the blood and causes the viraemia.
- localizes in the alimentary, respiratory and lymphoid systems and damages them.
- This results in severe diarrhoea, dehydration and death. In some, diarrhoea is followed by pneumonic signs.

Clinical findings

- **Acute form:** It is mainly seen in goats and is almost similar to RP.
- Initially high fever, dullness, sneezing and serous discharge from the eyes and nostrils are seen.
- After a day or two, there will be discrete necrotic lesions in the mouth which can spread to the entire oral mucosa forming diphtheritic plaques. Then there will be halitosis and anorexia.

- The nasal and ocular discharges become mucopurulent, which will dry up leading to matting of the lids and partial occlusion of the nostrils. Within 3-4 days of fever, there will be diarrhoea which is profuse and the faeces may contain mucous and even blood. Then coughing and dyspnoea will occur which will be worsened by secondary bacterial invaders.
- High morbidity (up to 100%) and up to 90% mortality

Sub acute form: This form is common in sheep and can be seen in goats also. The clinical feature is mild and few animals may die within 2 weeks. However, majority of the animals will recover.

Necropsy findings

- Discrete or extensive areas of erosion, necrosis and ulceration are present in the oral, pharyngeal and upper oesophageal mucosa and can extend upto abomasum and small intestine. Zebra markings are seen in the ileo-caecal region, colon and rectum. The retro-pharyngeal and mesenteric lymph nodes are enlarged and the spleen may be enlarged.
- Purulent or fibrinous broncho-pneumonia and pleuritis due to complications with secondary bacterial invaders can be seen.

Diagnosis:

- The clinical signs and necropsy lesions are suggestive of PPR.
- Isolation and identification of PPR virus confirms the diagnosis. For this purpose ocular, nasal and pharyngeal secretions and faeces of affected animals should be collected and submitted to a laboratory.
- Serological tests like Agar gel precipitation test (AGPT), counter immuno- electrophoresis technique, serum neutralization; complement fixation test (CFT), dot- ELISA and cDNA probes
- **PPRV-Sungri/96 vaccine virus**

CLOSTRIDIAL DISEASES

- Clostridial diseases are caused by bacteria of the genus clostridium (anaerobes)
- Clostridia are widespread in the environment and are normally found in soil and faeces.
- also present in the gastrointestinal tract and as spores in tissues of healthy animals
- 4 types- Histotoxic, hepatotoxic, enterotoxic, neurotoxic

Cl. chauvoei	**Cattle, sheep (pigs)**	**Black quarter (Black leg)**

Cl. septicum	Cattle ,Sheep ,Chicken	Malignant oedema Braxy ,Necrotic dermatitis
Cl. novyi Type A	Sheep Cattle and Sheep	Big head of rams Gas gangrene
Type B	Sheep, Cattle	Black disease (necrotic hepatitis)
Type C	buffalo	Osteomyelitis
C. haemolyticum	Cattle, sheep	Bovine Bacillary haemoglobinuri

Quails Disease/ ulcerative enteritis – C. colinum

Enterotoxigenic clostridium

C. perfringens - Type A	**Humans** **Lambs** **Broiler chickens**	**Gas gangrene,** **Food poisoning, Enterotox mic Jaundice** **(Yellow lambs disease) Nec otic enteritis**
C. perfringens - Type B	Lambs (Under 3 weeks old)	Lamb dysentery
C. perfringens - Type C	Piglets, lambs, calves and foals Broiler chickens Adult sheep and goat	Struck, Haemorrhagic Enter toxae (Clostridial enteritis) Necrotic enteritis
C. perfringens - Type D	Sheep(except neonates)	Pulpy kidney disease
C. perfringens - Type E	Calves and lambs	Enterotoxaemia

Neurotoxic: C. tetani- Tetanus
C. Botulinum- Botulism

BRAXY (Bradsot, Bradapest, Cling)

- acute infectious disease of sheep caused by *Cl. septicum* and is characterized by sudden onset, high fever, inflammation of abomasal wall, abdominal pain, toxaemia and short duration of illness.
- ***Transmission***: *C. septicum* is a **soil-borne** organism and in many areas can be considered as a **normal inhabitant of the ovine intestinal tract**.
- **Pathogenesis:** primary **abomasitis**, associated with the ingestion of frozen grass or other feed, permits invasion by *C. septicum*, resulting in a **fatal toxemia.**

MALIGNANT OEDEMA (Gas gangrene, Big head, Clostridial Myonecrosis)

- **fatal wound infection** of all the species of domestic animals caused by the bacilli of the genus Clostridium, characterized by the inflammation at the site of infection and fever
- portals of entry of the organisms are through the **surgical or accidental wounds**.
- The disease "**swelled head**," a form of malignant edema, occurs in young rams **6 months to 2 years old**
- Potent **necrotoxins** produced in the wounds will cause extensive oedema and necrosis which will end in gangrene
- **foul smelling discharges from the wounds are-noticed**.

ENTEROTOXAEMIA: enterotoxaemia is used because the organisms in the intestine multiplies and produce toxins which will be absorbed into the circulation resulting in toxaemia.

- **caused by *Cl. perfringens***

C. Perfingens type	Toxin release	Disease

A	**alpha (significant toxin)** **Enterotoxin**	**Necrotic enteritis in chickens, Necrotizing enterocolitis in pigs, Canine haemorrhagic gastroenteritis,** gas gangrene **in humans**
B	Alpha, beta (Significant)and Epsilon	***Lamb dysentery* , Haemorrhagic enteritis in less than 3 weeks calves and foals**
C	Alpha, beta (Significant)and Enterotoxin	***Struck in adult sheep*,Necrotic enteritis in chickens, Haemorrhagic enteritis in neonatal piglets**
D	Alpha , Epsilon(Significant)	***Pulpy kidney in sheep***
E	Alpha	***Haemorrhagic enteritis in calves, Enteritis in rabbits***

PULPY KIDNEY DISEASE

- Synonym - "Over eating disease"
- caused by *Cl. perfringens* type D
- diarrhoea, convulsions, paralysis and sudden death.
- Morbidity rate can be upto 10% and the case fatality rate can reach 100%.
- Most common in lambs between 3-10 weeks of age
- Sustained production of **Epsilon toxin,** which exists as a prototoxin and requires activation by **proteolytic enzymes**, leads to toxaemia and the development of clinical signs.
- Enterotoxaemia vaccine (IVRI): it is a formalin killed vaccine. The dose is 2.5 ml, s/c

<u>Lamb dysentery</u>

- ✓ Lamb dysentery, caused by **C. *perfringens type B***

Factors which predispose to the development of enterotoxaemia

A**. Low proteolytic activity in the neonatal intestine:**

- Presence of trypsin inhibitors in colostrum
- Low level of pancreatic secretion

B. **Incomplete establishment of normal intestinal flora in neonates**

C**. Dietary influences in older animals:**

- Abrupt change to a rich diet
- **Grazing** on energy-rich diet

Struck

- ✓ caused by ***C. perfringens type C***
- ✓ ***'struck', an*** acute enterotoxaemia in adult sheep, **manifests** sudden death
- ✓ The ***Beta toxin*** plays the major role in the pathogenesis of the disease.

BACILLARY HAEMOGLOBINURIA (Red water disease)

- highly fatal toxaemia of cattle and sheep
- *Clostridium haemolyticum* and is characterized by high fever, jaundice, haemoglobinuria and necrotic infarcts in the liver.
- Haemolysin toxin which causes lysis of the RBCs leading to haemoglobinuria and jaundice.
- Necrotizing toxin is responsible for the formation of the thrombus in the sub- terminal branch of portal vein leading to large anaemic infarct which is the most characteristic lesion.
- Fever (103-106°F), depression, weakness, anorexia, jaundice and passing of coffee decoction coloured urine
- **Anaemic infarcts in the liver which may be one or more in number with a diameter of 5-20 cms. are very characteristic.**

BOTULISM- food poisoning

- Limber neck, Shaker foal syndrome, Western sickness, Bulbar paralysis, loin disease

-rapidly fatal motor paralytic condition caused by ingestion of *Clostridium botulinum* exotoxin

- Intoxication not infection (Disease which occur by ingestion of pre-formed toxin)

- The toxin does not enter CNS but affect PNS where it block release of acetylcholine and inhibitory neurotransmitters like GABA, glycine etc. causing **flaccid paralysis**.
- animals die of asphyxia
- For the poisoning to occur, the toxin should be present in the food before ingestion, since the organism cannot generate the toxin in the alimentary tract

A, B, C, D, E, types

- A – Limber neck – Poultry
- B – Shaker foal syndrome
- D – Lamsiekte
- C – Forage poisoning in horses
- E – Fish and fish products
- Source of infection in animals is almost always carrion which include domestic animals, wild animals and birds.
- Animals having pica will ingest such materials containing the toxins.

Clinical signs

- Cattle -Pica-Chew bones (due to deficiency of phosphorus) which may contain toxin in the decaying meat
- Cattle and Horse: Forage Poisoning (Ingestion of hay contaminated with dead animals or poultry litter)
- Wound botulism – Horses
- Botulism in birds is exhibited by torticollis – '**Limber neck – ascending flaccid paralysis**
- Death – **Respiratory failure and asphyxia**
- No characteristic lesions
- **In birds disease is known as Limber neck**
- **In Horse also known as Shakers foal syndrome – toxiinfection**
- **occurs in foals usually less than 4 weeks old**

BLACK QUARTER
(Black leg, Emphysematous gangrene, Symptomatic anthrax, **Quarter ill)**

- infectious disease of cattle, buffaloes and sheep caused by ***Clostridium (feseri) chauvoei***, a Grampositive, spore-forming, rod-shaped bacterium.and is characterized by high fever, emphysematous sero-haemorrhagic swelling of the heavy muscles and lameness.
- clostridial myositis of skeletal muscles
- case fatality rate in blackleg approaches 100%.
- spores are highly resistant to the environmental conditions, disinfectants and persist in the soil for many years as it is a **'soil borne' organism.**
- True blackleg is common only in cattle but infection with this organism initiated by trauma occurs occasionally in other animals.
- In cattle the disease usually occurs without a history of trauma but in sheep is almost always a wound infection.
- Cattle and sheep are commonly affected. Cattle in the age group of 6 months to 2 years and rapidly growing on a high plane of nutrition are highly susceptible. Sheep of any age group can be affected.
- BQ commonly occurs every year immediately after the onset of monsoon rains, where there will be sudden change in the weather condition which will act as a stress, on the body
- In pigs, blackleg is not common

Transmission

- Through the alimentary mucosa after ingestion of contaminated feed and water in cattle
- Through the wounds in sheep due to shearing, docking and lambing

Pathogenesis

- organisms go to the intestine and then gets into the blood and localizes in heavy muscles especially of the hind quarters and shoulders
- There organisms lie dormant and only during stress the spores develop into vegetative forms.
- Death within 24 hrs to 60 hrs. Death may be due to toxaemia. From the affected muscles the organisms can get into the blood and can localize in lungs, liver, heart, kidneys and spleen and can produce gas, oedema and necrosis.
- In cattle and sheep atypical outbreaks of sudden death occur in which the lethal lesion is a clostridial cardiac myositis
- RBC lysis
- Iron released & due to necrosis of muscle H_2S is release

- $H_2S + Fe = Fe_2S$ - which is black in color& responsible for black color of affected muscles.
- Release of gas is due to fermentation of sugar in anaerobic medium.

Clinical findings

- Peracute form – sudden death without showing any signs. oozing of blood tinged fluid or blood from the natural orifices which will coagulate immediately.
- Acute form- high fever (106-108°F), depression, anorexia, decreased milk production and dullness. Then there will be acute lameness.
- hot and painful swelling in heavy muscles in the beginning and soon becomes cold and painless. Later on such lesions can be seen in the base of the tongue, heart muscle, diaphragm, muscle, brisket and udder.
- The course of the disease can be 12-36 hours and also can be 3-5 days. During the final stages the animal will be recumbent, and then coma and death occurs.
- In sheep, acute lameness in one or several limbs is noticed. Oedema and crepitation cannot be felt before death. High fever, anorexia and depression are also seen and death occurs very quickly.

Necropsy findings

- Cattle found dead of blackleg are often in a characteristic position; lying on the side with the affected hind limb stuck out stiffly.
- presence of dark, discoloured, swollen tissue with a rancid odor, a metallic sheen on the cut surface and an excess of thin sero-sanguinous fluid containing bubbles of gas.
- In sheep the muscle lesions are more localized and deeper and the subcutaneous edema is not so marked, except around the head. Gas is present in the affected muscles but not in such large amounts as in cattle
- 'False blackleg' may be associated with C. septicum and C. novyi but this disease is more accurately classified as malignant edema.

Diagnosis

- **By history** of occurrence of BQ immediately after the onset of monsoon rains and clinical signs
- **By necropsy findings**
- Isolation of the causative agent
- **Serological tests** like Fluorescent antibody test (FAT) and Enzyme linked immunosorbent assay (ELISA)

Treatment

- penicillin and surgical debridement of the lesion
- Large doses (40 000 IU/kg BW) should be administered, commencing with crystalline penicillin intravenously and followed by longer-acting preparations.
- Blackleg antiserum is unlikely to be of much value in treatment unless very large doses are given.
- Metronidazole which is effective against anaerobes can also be administered along with penicillin @ 5-10 mg/kg body weight, i/v.

Prevention

- annual vaccination of all cattle between 3 and 6 months with two vaccinations given 4 weeks apart followed by an annual booster vaccination is recommended.
- Maternal immunity persists for at least 3 months and will interfere with active immunity in calves vaccinated before this age
- In an outbreak all unaffected cattle should be vaccinated immediately and injected with penicillin at a dose of 10 000 IU/kg BW intramuscularly or a combination of penicillin and benzathine penicillin.
- formalin killed vaccine containing *Cl. chauvoei* and *Cl. septicum*.
- Raksha BQ- Aluminium Hydrogen oxide gel absorbed inactivated vaccine

TETANUS
Locked jaw, Saw Horse disease

- Highly fatal disease of all domestic animals caused by the neurotoxin and characterized by hyperaesthessia, tetany and convulsions

Etiology:

- *Clostridium tetani* , G +ve, anaerobe.
- terminal spore gives a “drum stick like” appearance.
- **gelatin slab culture**, the colonies appear like **'fig tree' growth**
- The organism produces a highly potent toxin i.e. Tetanospasmin (Neurotoxin), Tetanolysin (Hemolysin) and Fibrinolysin

Host Range:

- Susceptibility is Horse> Sheep/Goat > Cattle >Pigs
- Birds are resistant.

Transmission

- The organisms enter the body through the deep punctured wounds
- **Neonatal tetanus** occurs when there is infection in the umbilical cord associated with unsanitary conditions at parturition.
- **idiopathic tetanus** occur occasionally in young cattle without a wound being apparent, usually in association with the grazing of rough, fibrous feed, and it is probable that toxin is produced in wounds in the mouth or gastrointestinal tract or is ingested preformed in the feed.

Pathogenesis

- The organisms go to the deeper part of the wound and multiply when there is lowering of the local tissue oxygen tension. They produce 3 types of toxins:
- **Tetanolysin** is the haemolysin.
- **Fibrinolysin**.
- **Tetanospasmin** gets into the diffuses to the systemic circulation, is bound to motor end plates, and travels up peripheral nerve trunks via retrograde intraaxonal transport to the CNS.
- It may potentiate the normal sensory stimuli leading to the convulsions and tetany. **The death is due to asphyxia which is due to fixation of the muscles of respiration.**

Clinical Findings

- Localized stiffness, often involving the masseter muscles and muscles of the neck, the hind limbs, and the region of the infected wound, is seen first.
- drooling of saliva, Animals stands with tail raised, erected ears, dilated nostrils, and retracted eyelids and feels difficulty in movement.
- Generalized stiffness one day later with tonic spasm and hyperaesthesia.
- Excitation, sudden movement or noise or other stimuli - → increases intensity
- A spasm of head muscles causes difficulty in prehension and mastication of food, hence named lockjaw.
- Prolapse of third eyelid—Early signs in horse.
- Tetany occurs and as it progress, the animal adopt saw horse appearance
- Due to stimuli, there is hyperaesthesia, tetany and convulsions.
- Excessive sweating and marked opisthotonos are developed.

- The hind limbs are stuck out stiffly behind and fore limbs forwards.
- Death occur due to asphyxia.

Diagnosis:

- History and Clinical signs
- Culture of organism by anaerobic method
- Microscopic Examination- Tennis racket or Drum stick appearance of spores

Treatment

- Eliminate the causative bacteria
- Neutralize residual toxin
- Control muscle spasms until the toxin is eliminated or destroyed
- Maintain hydration and nutrition
- Provide supportive treatment

*Theiler's Disease: Serum hepatitis in response to Tetanus toxoid causing death

❖ **To kill the organism:**

✓ Penicillin @44000 IU/Kg BW, IM or, around or in wound if present

❖ **To neutralize toxin:**

✓ ATS @3 lakh IU/Horse, IV, SC or IM at 12 hrs intervals 3 times.

TETANUS TOXOID

- Large Animals: 5ml, I/M
- Small Animals: 3 ml, I/M
- Tetanus antitoxin is often routinely given to mares following foaling and to new born foals

Active immunity:

- Foals --- primary vaccination at 3-4 months
- Pregnant mares- booster injection 4-6 weeks before foaling to provide adequate colostral immunity to the foal.

MASTITIS

- inflammation of the mammary gland caused by varieties of bacteria and fungi and is characterized by physical, chemical and bacteriological changes in the milk and by pathological changes in the milk and glandular tissue

Etiology

1. By contagious pathogens – Staph aureus, Strep agalactiae, Corynebacterium bovis, Mycoplasma bovis

2. By teat opportunistic pathogens- coagulase -ve Staph sp.

3. Environmental – Coliforms (E.coli, Klebsiella), Corynebacterium bovis, Strep uberis & dysgalactiae

- ***Bacteria:*** Innumerable genera and species of bacteria cause mastitis. The **predominant organism is *Staphylococcus aureus*** followed closely by ***Streptococcus agalactiae*.**
- *E. coli, Str. uberis. Str. dysgalactiae. Str. zooepidemicus, Str. faecalis, Str. pyogenes. Campylobacter jejuni, Haemophilus somnus. Str. pneumoniae, Corynebacterium pyogenes. Cor. ulcerans, Klebsiella Spp., Enterobacter aerogenes, Mycobacterium bovis;*
- ***Fungi and Yeast:*** Fungal agents like *Trichophyton Spp., Aspergillus fumigatus, A. nidulus and Pichia Spp.*
- ***Yeasts*** like *Candida* Spp., *Cryptococcus neoformans, Saccharomyces* Spp,
- ***Algae*** like *Prototheca trispora* and *P. zopfii.*
- ***Viruses:*** Viruses causing IBR, BVD, FMD, cow pox, pseudo cowpox, and ulcerative mammilitis

Prevalance increases with age (most at 3rd and 4th lactation)

Economic losses- per year loss of 72.4 million rupees out of which 60% approx. 41.5 million rupees loss due to subclinical mastitis

H.F most susceptible (H.F>Crossbred> Desi/ Indian origin), high yielder (out of Indian origin>> low yielder (desi /indian)

Buffalos less occurrence than cow because of long teat canal

Transmission:

The organism can enter the udder through various routes.

l. By milkers hands

II. Trauma or injury to the teat

III. Intramammary infusions

IV. Haematogenous: The common example is tuberculous mastitis where in the organism from the lesions gets into the blood and then localizes in the udder producing mastitis.

V. **Inhalation:** The organism like Mycoplasma enters the animals through inhalation and gets into respiratory tract.

Pathogenesis:

- The development of mastitis occurs in three stages and they are:
- **Invasion**: It is the stage wherein the organisms pass from the teat orifice into the teat canal.
- **Infection:** In this stage the organisms multiply rapidly and invade the mammary tissue rapidly.
- **Inflammation:** Various types of inflammation like peracute, acute, sub-acute, chronic and chronic granulomatous can occur.
- **Chronic granulomatous mastitis:** Stap. aureus, Mvcobacterium bovis and Myco. tuberculosis. This type of inflammation is called "**Botryomycosis**' wherein it is characterized by the presence of sulphur granules
- **Gangrenous mastitis : Stap. aureus**. Here due to thrombus formation the blood supply to the necrotic part of udder is reduced resulting in putrefaction, the affected part becomes blue or black in colour and sloughing of the part occurs.

Clinical signs

- **Peracute mastitis*:*** Both the systemic signs and local signs are seen in peracute mastitis
- ***Etiology:*** *Stap. aureus, E. coli, Coryne. pyogenes, Klebsiella spp*.,etc.
- ***Systemic signs*:** Sudden in onset, high fever (105-107°F), marked depression, complete anorexia, sudden drop in milk production, atony of the rumen, rapid pulse and respiratory rates, weakness and sunken eyes. As it progresses the animal is unable to stand and will be recumbent
- **Acute mastitis:**
- **Etiology:** The organisms which cause peracute mastitis, can also cause acute mastitis and in addition, the other organisms like Strep. agalactiae. Mycoplasma mycoides, Pseudomonas spp., etc., and fungi can also cause the same.
- Both systemic and local signs are seen.
- **Systemic signs:** Marked systemic signs may or may not be seen. If seen it will be almost similar to that of peracute mastitis.
- **Local signs:** Similar to peracute mastitis. Initially the secretions can be **watery and blood tinged,** later on it may be yellowish and pus like. The supramammaiy lymph nodes are enlarged. Slight to severe udder oedema may occur.

- The peracute and acute mastitis are commonly seen immediately after parturition. In both cases the udder is hard. This is due to massive infiltration of neutrophils, fibrin and fluid. The course of the disease is 3-5 days.
- **Subacute mastitis*:***
- ***Etiology:*** *Staph. aureus, Strep. agalactiae, Strep, dysgalactiae* etc. The systemic signs are not seen and the local signs are less marked.
- ***Subclinical mastitis:***
- ***Etiology:*** Similar to subacute mastitis.
- In this type of mastitis both the **systemic and local signs are absent**. This type of mastitis can be only detected by certain laboratory tests like California mastitis test (CMT), Wisconsin mastitis test, white side test, leucocyte count, Mastrip test, etc.
- **Chronic mastitis and chronic granulomatous mastitis*:***
- ***Etiology:*** *Stap. aureus - Botryomycosis, Mycobacterium bovis, Nocardia asteroides*
- and *Cryptococcus neoformans* and others
- ***Systemic signs* -** Absent.
- ***Local signs*:** Decrease in size of udder and the udder will be hard and upon palpation nodules can be felt. Milk may contain few clots.
- **Streptococcal mastitis by *Strep. agalactiae*:** This organism is an **obligatory parasite**. It can cause acute, subclinical and chronic mastitis in cows, ewes and does. Most important cause of mastitis in these animals.
- **The organisms are located in teat canal and teat cistern.**
- The **CAMP test**, which has served as the universally used means of identifying *S. agalactiae* for many years, has been displaced by a commercial **latex agglutination test**
- **Coliform mastitis**: It is a soil borne infection and it occurs when the hygienic conditions are poor. It is also known as **"Environmental mastitis"** because the bacteria are transmitted from the environment to the cow rather than cow to cow transmission.
- **Mastitis due to *Corynobacterium* (Actinomyces) pyogenes: summer mastitis** in UK because it is commonly recorded during summer. The secretion will be **pus like and foul smelling**.
- **Mastitis due to *Nocardia asteroides*:** The entry of organisms into the udder is through the contaminated intramammary infusions. It causes chronic granulomatous inflammation.
- **Tuberculous mastitis:** *Mycobacterium bovis* - granulomatous inflammation

- **Mastitis due to *Mycoplasma*: *Mycoplasma agalactiae var bovis*** - colostrum like or soft cheese like or yellow coloured strand milk

Diagnosis

- 1.Physical examination- organoleptic taste (color, taste, texture)
- 2.Chemical examination – pH increased in mastitis more than 7.4
- 3.California mastitis test (CMT)
- 4.Somatic cell count (SCC)
- 5.Catalase test
- 6.Hotis test
- 7.NAG-ase test
- 8.Bromothymol blue test (BTB)
- 9.Chloride test

Test for diagnosis OF sub-clinical mastitis

1. CMT

2. SCC

3. NAG-ase(N-acetyl d-Glucoamindase)- enzyme present in somatic cells

4. WISCONSIN MASTITITS TEST

5. White side test: Using NaOH

- Chloride test: Normal content of chloride in. milk is **0.08 – 0.14 gm**% but in mastitis its level is increased.
- **pH of milk:**

  -Normal pH : 6.4 - 6.8

  -Mastitis : 7.4 and above

California Mastitis Test

- No change - Normal
- Traces - No distinct wave, tiny lumps are seen.
- + - Thickening and definite wave. Does not adhere to the bottom of the cup

- ++ - Marked thickening and adhere to the bottom of cup.
- +++ - Too much thickening which will appear like a ball.
- The anionic detergent present in the CMT react with DNA present in the leukocyte which is responsible for thickening of contents.
- Because the anionic detergent, **trietholamine sulfonate**, present in the CMT reagent is not readily available in our country it has been substituted by **sodium lauryl sulfate**

Somatic cell count

- SCC normally less than 2 lac/ml in millk
- -----more than 2 lac/ml -- mastitis
- ----Somatic cells - macrophages(60-80%), Lymphocytes(10-25%), neutrophils <11%

Hotis test:

- **Method:** Take 0.5 ml of 0.5% Bromocresol purple reagent in a test tube
- *For Strep. agalactiae*.

Koestler number (Koestler, 1920) = % Chloride/ % lactose

    - It is usually in normal milk less than 2, in suspected milk from 2 to 3, and in mastitic milk more than 3

- Treatment
    - Antibiotics
    - NSAIDs
    - Antihistaminics
    - Vitamin C
    - Coagulants
    - Topical applications
    - Intramammary infusions

Control of mastitis

- Udder hygiene (0.5-1% Iodine) and proper milking methods (Most commonly full hand milking)
- Dry cow therapy – use of LA intra-mammary infusion after last milking of lactation
- Blanket therapy , selective cow and selective quarter therapy

- Vaccination
- Lysigin –bactrin based vaccine
- J-5, J Vac bactirin against E.Coli
- Endovac Dairy- salmonella toxoid

POSTNATAL DISEASES

- Neonatal disease - morbidity or mortality between birth and 14 days.
- **Early postnatal disease (within 48 hours of birth):**. Most diseases occurring in this period are noninfectious and 'metabolic', e.g. hypoglycemia and hypothermia due to poor mothering, hypothermia due to exposure to cold, low vigor in neonates due to malnutrition.

Delayed postnatal disease (2-7 days of age)

- Desertion by mother, mammary incompetence resulting in starvation and diseases associated with increased susceptibility to infection due to failure of transfer of colostral immunoglobulins (the predisposing causes to these occur in the first 12-24 hours of life) .
- Examples include colibacillosis, joint ill, lamb dysentery, septicemic disease, most of the viral enteric infections in young animals, e.g. rotavirus and coronavirus

Late postnatal disease (1-4 weeks of age)

- There is still some influence of hypo gammaglobulinemia, with late onset enteric diseases and the development and severity of respiratory disease, but other diseases not directly associated with failure of transfer of immunoglobulins such as cryptosporidiosis, white muscle disease and enterotoxemia
- Postnatal disease causing mortality from birth to 2 months of age includes:
    - lack of maturity 36%
    - structural defect 23%
    - birth injury 5%
    - convulsive syndrome 5%
    - alimentary disorder 12%
    - generalized infection 11 %
    - other (miscellaneous) 9%.
- Minimal gestational ages for viability (in days) for each of the species are:
- Calf - 240

- Foal - 300
- Lamb - 138
- Piglet - 108.
- mortality rates in dairy calves that varied from a low of approximatly 2% to a high of 20%
- The best estimate for the average on -farm calf mortality rate is 6%
- Mortality on well managed farm around 5% from birth to 30 days
- Mortality in twin-born calves is approximately three times that of single born calves.
- Highest risk of death in calves: upto 2 weeks due to septicemia and enteric disease
- Respiratory disease common after 2 weeks
- Failure of transfer of colostral immunoglobulins is a major determinant of this mortality
- serum IgG1 concentrations - 500 mg/dL - protection against septicemic disease
- 1000 mg/dL or more - sufficient to reduce the risk of infectious disease in most environments.

Transfer of immunity

- GGT concentrations are high in the colostrum of ruminants (but not horses) and serum GGT activity in calves and lambs that have sucked or been fed colostrum are 60-160 times greater than normal adult serum activity and correlate moderately well with serum IgG
- Serum GGT concentrations less than 50 IU/L indicate failure of transfer of passive immunity.
- In healthy calves a serum total protein of 5.2 g/dL or greater is associated with adequate transfer of passive immunity.

Neonatal Hypothermia

- The critical temperature for neonatal calves is approximately 13°C
- Bos taurus calves are more resistant to cold stress than Bos indicus.

Neonatal infections

- Following invasion via the nasopharynx and the gastrointestinal tract
- the usual pattern of development is a bacteremia followed by septicemia with severe systemic signs
- bacteremia with few or no systemic signs,
- localization in various organs

- If the portal of entry is the navel, local inflammation occurs -'navel ill'

COLIBACILLOSIS
NEONATAL DIARRHOEA COMPLEX

- *Escherichia coli* was considered as the primary pathogen in and the term **Colibacillosis** has been in common use
- *E. coli* has been divided into two groups namely **enteropathogenic** organisms and **septicaemic groups**.
- Enteropathogenic organisms are not invasive but localized in intestine especially in ileum and cause diarrhoea through the production of endotoxins.
  - *Salmonella:* Commonly *S. typhimurium* and *S. dublin* occurs in 2-6 week old calves.
- *Cl. perfringens type B and C*: There are several types like A, B, C and E and type C is most common producing a necrotizing toxin.
- *Reovirus* like agent/Rota virus/Neonatal calf diarrhoea virus
- *Corona virus*
- *BVD Virus*
- Chlamydia psittaci
- *Fungi: Canida, mucor, aspergillus*
- ***Protozoa:***
  - Coccidiosis: Affects young calves of 3 weeks and above of age. It can be a severe problem when overcrowding and poor sanitation and stress occurs. *Eimeria bovis* and *E. zurnii* are most predominant species
  - Cryptosporidia
- The mechanism involved in diarrhoea can be:
  - Defective absorption of fluids
  - Increase in secretions
  - Increase or normal intestinal motility
- Reo virus infection, tall columnar intestinal epithelial cells were lost and replaced by low cuboidal cells.
- Corona virus attacks the columnar epithelial cells of the entire villi resulting in marked villus atrophy.

- *Chlamydia* destroys the epithelial cells of the villi and crypts.

Effect of diarrhea

- dehydration and marked haemo-concentration which results in decreased circulating blood volume.
- metabolic acidosis: loss of HCO_3 ions from the blood into the intestine leading to decrease in blood HCO_3 levels. This will reduce the blood pH
- Hypokalaemia followed by hyperkalemia
    - Hypoglycaemia occurs as a result of suppression of the liver function by endotoxins and also due to malabsorption of lactose present in the milk and anorexia.

Enteric-toxaemic colibacillosis

- Affected new born animals collapse and die in a short time of 2 to 6 hours.
- clinical signs are coma, sub-normal temperature, cold extremities, collapse of superficial veins, slowness and irregularity of the heart and mild convulsive movements. Scouring is absent.

Septicaemic colibacillosis

- Most common during the first few days of life. The illness is acute and the course varying from 24-96 hours.
- Affected animals are depressed, weak and anorectic. There is a marked increase in heart rate and although the temperature is high initially, it will fall rapidly to sub-normal level when diarrhoea and dysentery occurs.
- Post septicaemic localization in the joints may cause arthritis with lameness, pain and swelling in the joints.
- In addition, localization in the meninges can lead to meningitis with clinical signs like recumbency, opisthotonus, colic, paddling convulsions, nystagmus and pus in the anterior chamber of the eye.
- Localization in the lungs can cause Pneumonia but this is less common.

Enteric colibacillosis

- This form can occur during the first three weeks of life but commonly occurs during the first week.
- The faeces are watery or pasty and usually chalky-white to yellow in colour and occasionally are streaked with blood.
- Defaecation is frequent, tail and buttocks are soiled and the faeces have an offensive rancid smell.

- There is usually a systemic reaction with a temperature upto 105°F and an increase in pulse rate.
- The animal ceases to drink, is dull and listless and rapidly becomes dehydrated. Various degrees of dehydration can be noticed.
- There may be abdominal pain on palpation, some time tenesmus is evident and the back may be arched. Without treatment death usually occurs in 3-5 days. All cases of colibacillosis should be carefully examined for evidence of omphalophlebitis.

Treatment

- **To overcome the metabolic acidosis, HCO_3 ions should be supplemented**
- Base deficit = Estimated HCO_3 level in blood - Normal HCO_3 level in blood.
- For the field Veterinarians, the base deficit is approximately: 5% dehydration:10 mEq of HCO_3/litre
- 10% dehydration : 5-20 mEq of HCO_3/litre
- 15% dehydration : 25 mEq of HCO_3/litre
- lactated Ringer's solution can be administered i/v. This solution contains the alkalizing agent sodium lactate which will be metabolized by the liver to yield HCO_3
- **To overcome hypokalaemia, Potassium to be supplemented** @ minimum of 10 mEq of potassium/litre of fluid given.
- Whole blood given at the rate of 10-20 mL/kg body weight, preferably by the intravenous route, will often save a calf that appears to be in shock associated with neonatal diarrhea.

Vaccination

- *E coli* bacterins containing K-99 pili antigen to be administered i/m to pregnant cows twice at 4 and 2 weeks before parturition.
- Combination of parenteral and oral administration of vaccines with K-88 pili antigen to pregnant sows

NAVEL ILL AND JOINT ILL

- Navel ill is a condition where there will be inflammation of umbilical vein and umbilical cord.
- Inflammation of the external aspects of umbilicus, umbilical veins and umbilical arteries is known as **omphalitis, omphalophlebitis** and **omphalo- arteritis** respectively.
- commonly occurs immediately after the birth of young ones of all the species of domestic animals and can be seen upto 3 months of age.
- Omphalitis in 2-5 days old & **omphalophlebitis in 1-2 months old**

- Joint ill is a condition wherein inflammation of one or more joints occurs
- due to improper disinfection of the umbilical cord after birth. It occurs usually as a mixed infection of *E. coli, proteus spp., brucella, campylobacter. leptospira, streptococci, staphylococci, Spherophorous necrophorus* and *Actinomyces (Corynebactgrium) pyogenes.*
- commonly occurs in young calves and can be seen in other species also.
- This condition is usually associated with fever, diarrhoea, depression, anorexia and weakness.
- In these two conditions, the affected area is swollen, painful to touch and filled with pus.
- Incoordination and limping in the beginning with acute lameness and recumbency later on. Ripening of the area which breaks open to discharge the pus will occur and the hairs are matted with the discharge.

Diarrhea

- Bacterial, viral, protozoal, parasitic, chemicals, physical agents, nutritional deficiency or dietary
- Winter dysentery in adult cattle by Corona virus
- Cryptosporidium: 5-35days old calves
- Eimeria: over 3 weeks to 12 months cattle
- Rhodococcus equi: Foals 2-5 months of age
- Osmotic diarrhea: saline purgatives, overfeeding, indigestible feeds and disaccharidase deficiencies, TGE, rotavirus, corona virus
- Exudative diarrhea: Salmonellosis, BVD, inorganic aresenic poisoning
- Secretory diarrhea: enterotoxigenic E. coli
- Abnormal intestinal motility.
- Dehydration, abdominal pain, septicemia and toxemia with fever
- Animal Dehydration Fluid (%) deficit (L)
- 500 kg horse 10 50
- 75 kg foal 10 7.5
- 45 kg calf 10 4.5

Actinobacillosis (wooden tongue)

- -*Actinobacillus ligniersi* – morse code appearence

- normal commensal organism in the oral flora of the cattle
- chronic sappurative granulomatous inflammation of the tongue and phatryngeal lymph node having Sulphur granules
- Hypoglossal nerve affected
- Causes swelling and hardening of tongue so called wooden tongue
- Intravenous sodium iodide (70 mg/kg of a 10% to 20% solution) is given once and then repeated 1–2 times at 7- to 10-day intervals.
- Potassium iodide - 5-10 g/cow, orally, once a day for 6-10 days.

Actinomycosis- lumpy jaw

- Chronic suppurative granulomatous disease characterized rarefying osteomyelitis of mandible and maxilla
- Actinomycosis bovis
- Demonstration of gram-positive rods in yellowish sulphur granules from aspirated purulent material
- Poll evil and Fistulous withers in Horses

Ulcerative lymphangitis

- Cattle and Horses- Ulcerative lymphangitis
- Sheep, Goat- Caseous Lymphadenitis
- Cause - *Corynebacterium pseudotuberculosis*
- Inflammation of skin and lymphatics resulting in nodules and abcess

Epizootic lymphangitis

- Pseudoglanders, Pseudofarcy, Equine Histoplasmosis
- Histoplasma capsulatum
- In horses, mules and donkeys

Campylobacteriosis/ vibriosis

Species	Hosts and diseases
C. foetus subsp. *venerealis*	Endometritis, sterility, abortion in cattle
C. foetus subsp. *foetus*	Abortion in sheep (sometimes in cattle); possible cause of enteritis in sheep, cattle, pigs
C. jejuni (*C. foetus* subsp. *jejuni*)	Abortion in sheep; enteritis in humans, monkeys, cattle, foals, dogs, cats, and fowl
C. coli	Enteritis in humans, monkeys, and other animals. Also fowl

- C. fetus ssp. Veneralis: specific venereal disease in cattle transmitted by coitus or AI
- Infection in cows is characterized mainly by characterized by early fetal death, infertility, and occasional abortions.
- Recovered cows are usually resistant to reinfection
- Vaginal mucous agglutination test – Detection of specific IgA
- Campylobacter foetus subsp. foetus: infects sheep
- not a venereal disease and is characterized by late term abortion
- Campylobacter jejuni & C. coli – Enteric Campylobacteriosis in animals & human
- C. jejuni and C. coli cause enteritis and Gulliean Barre syndrome

CBPP

- Contagious bovine pleuropneumonia / lung Plaque/ Lung Sickness
- Mycoplasma mycoides spp. Mycoides ‘small colony’
- cattle most susceptible
- lack cell wall and hence sensitive to tylosin, kanamycin, tetracyclines and resistant to penicillins.
- Pulmonary sequestration present
- marbling of lungs (inter-lobular septa are prominent)
- Lungers - CBPP recovered animals
- Because of the distension of the inter-lobular septa the pressure on the vessels and lymphatics leads to less supply of blood and results in necrotic changes and results in necrotic anaemic patches.

- Around the patches encapsulation can occur resulting in the formation of a sequestrum, which contains good amount of organisms, which can live upto 3 years.
- High Fever (105- 106°F), anorexia, marked depression, atony of the rumen reduction in milk yield occurs.
- Expiratory grunting, pleuritic friction rub and moist rales can be heard on auscultation of lungs.
- drug of choice is tylosin

CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP)

- - *Mycoplasma capricolum subsp. Capripneumoniae* (Mccp). (mycoplasma F38)
- characterized by fever, cough, dyspnoea, open mouth breathing and salivation in goats
- Morbidity is 100% and mortality can be 60-100%.
- Sequestrate not formed

LEPTOSPIROSIS

- Weill's disease/Infectious Jaundice/Rice field worker's disease/Sugarcane worker's disease
- infectious disease of domestic animals caused by various serovars of Leptospira and is characterized by septicaemia, haemolytic anaemia, haemoglobinuria, jaundice, abortion, mastitis and interstitial nephritis.
- pathogenic *Leptospira* (*L interrogans*) and the saprophytic *Leptospira* (*L biflexa)*
- They are usually visualized using *dark-field microscopy*.
- *Silver impregnation* and immunological staining techniques.
- For culturing of the Leptospirae, Stuart's, and Fletcher's media containing 5-10 % serum is used.

Etiology

- ***Cattle and Horses****: L. pomona, L. icterohaemorrhagiae, L. canicola, L. grippotyphosa, L. hardjo.*
- ***Sheep:*** *L. pomona*
- ***Goats:*** *L. grippotyphosa*
- ***Pigs:*** *L. pomona, L. canicola, L. icterohaemorrhagiae*.
- ***Dogs:*** *L. canicola, L. Icterohaemorrhagia, L. pomona, L. grippotyphosa, L. hardjo etc*..

Host Affected: All domestic animals and human.

Transmission: Pathogenic Leptospira can persist in the renal tubules or in the genital tract of carrier animals

- urine, aborted foetuses, and infected uterine discharges
- Urine is the chief source of contamination

Clinical Signs

- Cattle: Acute, subacute or chronic form can occur.
- Acute form: High fever (103-107°F), anorexia, depression, petechial haemorrhages on the mucosa. Jaundice, haemoglobinuria and haemolytic anaemia, characterized by increase in heart and respiratory rates, pale mucous membranes and also dyspnoea.
- Acute mastitis
- Sub-acute form: Very similar to acute but milder in degree.
- Chronic form: Abortion during the last trimester of gestation.
- Horses: Sub-acute form occurs and the condition called periodic ophthalmia is associated with this disease. Here photophobia, lacrimation, conjunctivitis, keratitis, hypopyon and iridocyclitis occur. Repeated occurrence of this condition results in blindness.
- Dogs: Canine typhus, Stuttgart's disease
- Per-acute, acute and chronic forms have been recorded.
- infectious jaundice, renal failure, abortions, still birth

Treatment

- streptomycin - drug of choice since it is excreted by the kidneys. It is administered @ 10 mg/kg body wt. bid, i/m for 3-5 days.
- Tetracyclines and doxycycline also can be used
- Sodium bicarbonate is administered to make the urine alkaline wherein the haemoglobin is soluble so that it can be eliminated without causing any damage to the renal tubules.

Diagnosis:

- Clinical signs, together with a history suggestive of exposure to contaminated urine, may suggest acute leptospirosis
- Organisms may be detected in fresh urine by dark-field microscopy
- Serological tests:

✓ <u>Microscopic agglutination test (MAT) – most common test – Gold standard test</u>

- ✓ ELISA: Tests for the detection of both IgM and IgG are available and include rapid tests which can be performed 'in the field'.
- ✓ Complement fixation test
- ✓ Fluorescent antibody procedures are often used for the demonstration of leptospires in tissues.

Ephemeral fever/Three days sickness

- Dengue fever of cattle/ Lazyman's disease/ Bovine Epizootic fever
- acute insect borne viral disease of Cattle buffalo
- Rhabdoviridae- Ephemerovirus - Bovine ephemeral fever virus
- Disease of wet season
- Transmission: is by sand fly- ceratopoganidae species.
- Not transmitted by
  - culicoides species
  - contact or fomites

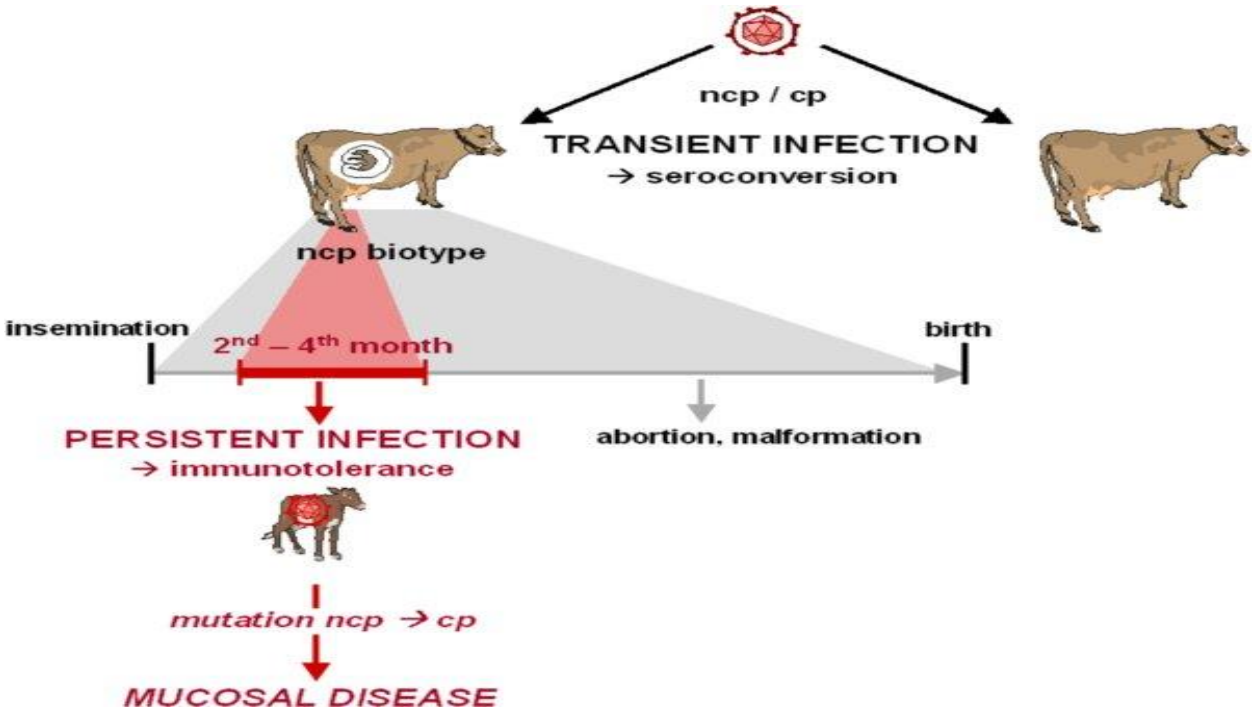
Clinical Signs

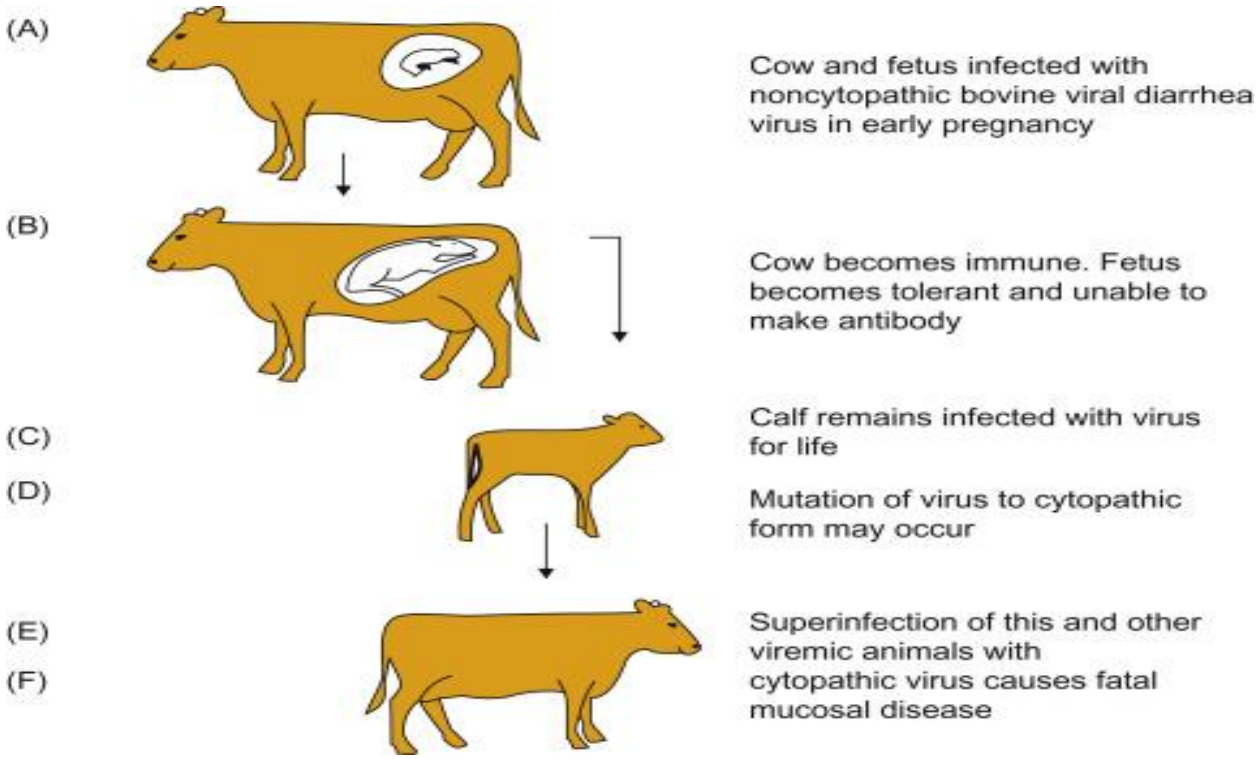
- Morbidity rates may be as high as 80%; overall mortality rate is usually 1%–2%,
- biphasic to polyphasic fever (40°–42°C [104°–107.6°F])
- peripheral lymph nodes are enlarged, anorexia, sharp fall in production of milk
- increase in heart and respiratory rates.
- Muscular shivering, stiffness of the muscles, swelling in the shoulders, neck and back; limping,
- lameness/recumbency can be seen or weakness of the hind limbs.
- Rx: NSAIDs & Supportive care for recumbent cows

BVD/Mucosal Disease

- Caused by Pestivirus of Flaviviridae family
- Clinical disease associated with BVD virus infection is most common in young cattle (6–24 months old).

- Virus types: 1& 2, Each genotype, 1 and 2, are divided into two biotypes- cytopathic (cp) and non-cytopathic (NCP) based on how they replicate in cell culture
- Most (>95%) of the field isolates are NCP
- bovine viral diarrhea is misleading in that the disease does not specifically affect the digestive tract but rather has immune suppression as a hallmark sign.
- Antigenic similarity with Classical swine fever and Ovine Border's disease
- BVD : immunocompetent cattle in post natal life
- **Persistently infected animals which are immune tolerant** – mucosal disease
- mode of transmission – direct, indirect contact & venereal
- Transplacental infections(b/w 80-150 days Teratogenic effects)





Clinical Signs BVD

- wide spectrum of clinical disease varying from **sub-clinical infection to fatal disease**
- Infertility or abortion – foetal abnormalities can occur with infections later in pregnancy resulting in brain abnormalities.
- Calf with cerebellar hypoplasia unable to stand and "stargazing"
- Oral **stomatitis and necrotic erosions in oral cavity**
- Mild to profuse watery Diarrhoea
- High morbidity and low mortality

Mucosal Disease

- diarrhoea, ulceration in mouth and gastro-intestinal tract and lameness
- **Failure to conceive**
- Early embryonic death / abortion / congenital deformity
- Fetal loss
- Low morbidity and high mortality

Malignant Catarrhal Fever

- MALIGNANT HEAD CATARRH / Gangrenous Coryza
- Severe, fatal, lymphoproliferative disease of ruminants caused by herpesviruses. Clinical signs include fever, oral and nasal erosions, enlarged lymph nodes, and centripetal corneal opacity.

1. Alcelaphine herpesvirus-1 (AHV-l)
    - wildebeest-associated MCF virus, transmitted to cattle from blue wildebeest

2. Ovine herpesvirus- 2 (OvHV-2) – major cause of spread
    - sheep-associated MCF virus transmitted to cattle from sheep

- Neither agent transmit from cattle to cattle
- neither of the viruses cause any disease in their principal host, the wildebeest and the sheep

Low morbidity high mortality (almost 100%)

Species Affected: Cattle, bison, giraffe, antelope, deer, nilgai

Transmission

- **AHV-1: In utero, Contact with nasal and ocular secretions, Aerosols during close contact**
- OHV-2: Respiratory (aerosol), Transplacental rare, Contact with nasal secretion

Clinical Signs:

1. Per acute form: sudden death, diarrohea before 12-24 hours of death

2. Head and eye form - Majority of cattle cases ;
    - Early Stages - Reddened eyelids, Bilateral corneal opacity, Crusty muzzle, nares, Nasal discharge, Salivation
    - Later Stages - Erosions on the tongue, buccal mucosa, necrosis, ulcer in oral cavity

- Swelling of Joints, superficial lymph nodes, Incoordination, head pressing, nystagmus, hyperesthesia

3. Intestinal form - Initially like head and eye form, but death occurs from severe diarrhea

4. Mild form- Inoculated animals; recovery expected

Blue tongue

- sore muzzle/ Muzzle disease

- infectious disease mainly of sheep and occasionally of cattle caused by an orbivirus (Reo virus) and is characterized by catarrhal stomatitis, rhinitis and enteritis and lameness due to laminits and coronitis.
- arthropod-borne orbivirus in the family Reoviridae
- insect borne disease (By Genus Cullicoides) ; also venereal transmission
- The sheep ked (Melophagus ovinus) acts as a mechnical vector
- fever (105-106°F), anorexia, increase in heart and respiratory rates
- Severe congestion of the oral mucosa especially of the tongue resulting in cyanosis (blue colour), hence known as 'blue tongue' disease.
- Later on ulcers occur on the lateral borders of the tongue and thus the saliva can be blood mixed.
- Swelling and oedema of the lips, gums, dental pad and tongue can occur
- appearance of dark red purple band on the skin just above the coronet

Pox Diseases

Genera	Members
Orthopox	Cow pox (vaccinia variola), Horse pox, camel and buffalo pox, rabbit pox.
Parapox	Orf (sheep), bovine pustular stomatitis, Pseudocowpox / milker's nodules Ectromelia / mouse pox(An important disease of laboratory and wild mice)
Capri pox	Sheep pox, goat pox, Lumpy skin disease.
Suipox	Swine pox.
Avipox	Fowl pox, pigeon pox, turkey pox and other galliformes
Leporipox	Myxoma virus, rabbit (Shope) fibroma virus.

Cow pox

- typical pox lesions on the teat and udder
- calf– around mouth; bulls– scrotum
    - Transmission: By rodents, cats, milkers hand and milking machine.

Pathogenesis

- virus enters the prickle cell layer of skin and causes inflammation resulting in congestion and this is known as ‘roseola’ stage.
- Epithelial cells undergoes hypertrophy which leads to the formation of papules and this is known as ‘papular’ stage.
- Then the fluid accumulates leading to the formation of vesicles and this is known as ‘vesicular’ stage.
- These vesicles are umbilicated. Then there will be infiltration of neutrophils into the vesicles and pus formation occurs which is known as ‘pustular’ stage.
- This break open and dries up and forms scabs.
- Normally these scabs will detach and fall off which is known as stage of ‘desquamation’.

SHEEP POX AND GOAT POX

- Benign and malignant form occurs in sheep and goats.
- The benign form occurs in the adults where the lesions are seen at the base of the tail. The malignant form is seen in the lambs where the lesions are seen in the oral and nasal cavity.
- **Etiology:** The virus belongs to the genus Capri pox.
- Sheep pox vaccine: live un-attenuated sheep pox virus suspended in 50% glycerol saline.

LSD

- Infected cattle develop **fever, lacrimation, nasal discharge, and hypersalivation, followed by the characteristic eruptions on the skin**
- **nodules are well circumscribed, round, slightly raised, firm, and painful**
- Regional lymph nodes are swollen and edema develops in the udder, brisket, and legs.
- Secondary infection sometimes occurs and causes extensive suppuration and sloughing; as a result, the animal may become extremely emaciated
- Goat pox vaccine : used in cases of LSD

- Goat pox vaccine provides immunity against both SPV & GPV in Sheep
- Sheep Pox vaccine failed to protect goat against GPV
- **LIVE ATTENUATED FREEZE DRIED VACCINE**
- **Pseudocowpox--- Milker's nodules**
- numerous & larger leisons than cow pox; horse shoe shaped ring after scab drops
- contagious ecthyma / contagious pustular dermatitis/ Sore mouth --- Orf virus in sheep
- Guarnieri bodies : intracytoplasmic inclusion bodies In all pox infections

Enzootic bovine leukosis

- bovine leukemia virus (BLV)
- causes lymphosarcomas
- infection by exposure to infected lymphocytes not virus
- transmission – iatrogenic, infected animal, mechanical, transplacental, venereal

Infectious bovine rhinotracheitis

- infectious pustular vulvovaginitis/ infectious pustular balanoposthitis/ red nose/ BHV-1 infection
- Bovine herpes virus-1
- BHV-1.1(respiratory)
- BHV-l.2a and 1.2b(genital) – abortigenic potential
- BHV- l.3 (BHV-5; encephalitic)
- mode of transmission : respiratory & genital
- most common in cattle over 6 months of age
- Latency: in the neurons of trigeminal nerve or sacral ganglia and reactivated in stress
- abortions in 4-8 months of gestation (6.5 months specifically)
- infectious pustular vulvo vaginitis in females
- infectious pustular balanoposthitis in males
- intranuclear inclusion bodies of cowdry type A in vaginal tissues in IPV

Rift valley fever

- - infectious enzootic hepatitis of cattle and sheep caused by Phlebovirus
- -vector borne zoonotic disease
- -High mortality in young animals and abortions in Pregnant animals
- - main transmission route: Aedes mosquito
- - per acute and acute type incidences in epidemic form
- - incubation period : 1-3 days

Swine erysipelas (Diamond skin disease)

- Erysipelothrix rhusiopathiae
- Up to 50% of pigs - colonized with *E rhusiopathiae* - resides in the tonsillar tissue - shed the organism in their feces or oro nasal secretions
- Infection is by ingestion or through skin abrasions.
- sudden and unexpected deaths, febrile episodes, inappetence, painful joints, and classical diamond shaped skin lesions in acute form
- Non suppurative arthritis and vegetative endocarditis in chronic form
- Primarily disease of pigs but can affect other animals also
- Zoonotic in nature

HOG CHOLERA
(Swine plague, Classical swine fever**)**

- infectious disease of pigs caused by a Toga virus and it is characterized by septicemia, hemorrhages through out the body and high morbidity and mortality rate.
- **Etiology:**
- Family: Togaviridae
- Genus: Pesti virus
- It is antigenically related to BVD virus.

Transmission

- All breeds and ages are susceptible and adults are more likely to survive an acute infection

- **Transmission:** By inhalation, ingestion, Insemination.
- In areas free of the disease, introduction is usually by the importation of infected pigs or the feeding of garbage containing uncooked pork scraps.
- Infected pigs shed a large amount of the virus in all normal secretions
- The disease usually occurs in epidemics, often with a morbidity of 100% and a case-fatality rate approaching 100%, when a virulent strain of the virus infects a susceptible population.
- excreted in the urine for some days before clinical illness appears and for 2-3 weeks after clinical recover

Pathogenesis

- virus gets into the tonsils and multiplies.
- Through lymphatics gets into the circulation and replicates leading to septicemia
- The virus is having an affinity for the endothelium of vessels. Here it replicates and causes hydropic degeneration and proliferation of the endothelium.
- This leads to partial/complete occlusion of vessels leading to infarcts.
- It is also having affinity for lymphoid tissue leading to depletion of B- lymphocytes in the lymphoid organs and also leucopenia.
- Neutralizing antibodies occur as early as 9 days after infection in recovering pigs and after 15 days in fatally infected pigs
- **Carrier sow syndrome**: can give birth to normal healthy appearing piglets which are persistently infected and immunotolerant; these pigs along with those with chronic infections are responsible for the perpetuation of the virus in the pig population
- Early immunosuppression is an important feature of the development of CSF59 with the depression of CD1+, CD4 + and CD8+

Clinical Signs

- **Peracute:** Withtout signs many young ones will be found dead.
- **Acute:** High fever (105-107^{0}F), anorexia, depression, disinclined to move upon force and hanging of the tail. Constipation followed by diarrhoea and vomiting can occur. Diffused purplish colouring of the skin on the ventral aspect of abdomen, thorax, inside of the thigh and perenium occurs. Later on necrosis on the edges of ear, tail and lips of vulva is seen. Nervous signs are also seen in the early stages, circling incoordination, muscular tremors and convulsions.

- **Chronic:** Milder form occurs as a result of low virulent strains of virus and many of the clinical signs of acute form are seen. Skin lesions are common. This chronic form appears to be common now a day.
- It can also cause foetal resorptions, mummified fetuses, stillbirths and congenital anomalies like cerebellar hypoplasia.

Necropsy findings

- Petechial haemorrhages on serosa and mucosa and these haemorrahges are clearly noticeable under the capsule of the kidneys, ileo-caecal valve, urinary bladder, larynx and lymph nodes. Infarcts in the mucosa of gall bladder, larynx and lymph nodes. The spleen is enlarged and having marginal infarcts. Infarcts in the mucosa of gall bladder is almost a pathognomonic lesion of hog cholera. The mucosa of colon will be having circular raised button ulcers and now a days it is not common.
- Histopathologically non suppurative encephalitis with perivascular cuffing is a very important lesion and the lesions of the blood vessels are noticed.
- Kidney – found petechiae on the cortex extending deeply into the parenchyma. These gives a characteristic **Turkey egg appearance.**

African swine fever

- ASFV of genus Asfivirus and family Asfarviridae
- only disease caused by DNA virus and transmitted by arthropod
- Tick : Ornithodorus moubata
- very high morbidity and almost100% in infected pigs
- symptoms almost similar to CSF

Pseudorabies

- Aujeszky's disease, Mad itch, Infectious bulbar paralysis
- Caused by Herpes Virus
- primarily affects pigs but may occur in other species of animals
- Rodents and pigs are the primary hosts of the virus.
- Pigs ---reservoir host and source of infection to other species of animals
- Dog & Cat- Dead end host
- Pigs- incoordination in posterior limbs (Dog sitting posture)

- Cattle- severe pruritus
- non suppurative meningoencephalitis

GLANDERS

Synonyms: Farcy/Malleus

- acute to chronic contagious disease of horse, mules and donkeys characterized by nodules or ulcers in the respiratory tract and on the skin.

"Glanders" terms used when the principal lesions are seen in the nostrils, submaxillary glands and lungs & "farcy" when lesions are on the surface of limbs or body.

Etiology:

- *Pseudomonas mallei*
- *Actinomyces mallei*
- *Burkholderia mallei* (Gram –ve bacillus)

Host affected:

- In horses **chronic** form occurs commonly and in mules and donkeys **acute** forms occurs.

Acute (Mules/Donkey)	Chronic (Horses)
Fever	Fever
Cough	Cough
Dyspnea	Malaise (Uneasiness)
Thick nasal discharge	Nasal discharge
Ulcers on nasal mucosal	Ulcers & skin nodules
Involvement of lymphoid & blood system leading to death	Swelling of joints & leg edema
Usually occurs for 1-2 weeks	Weight loss

Diagnosis:

- Signs & symptoms
- Culture & isolation of bacteria
- Serological tests :-
    - ✓ Indirect hemagglutination,

- ✓ Immunofluorescence
- ✓ ELISA – Most reliable in horses

– not used in donkey or mule

- **Mallein test**

Prevention & Control

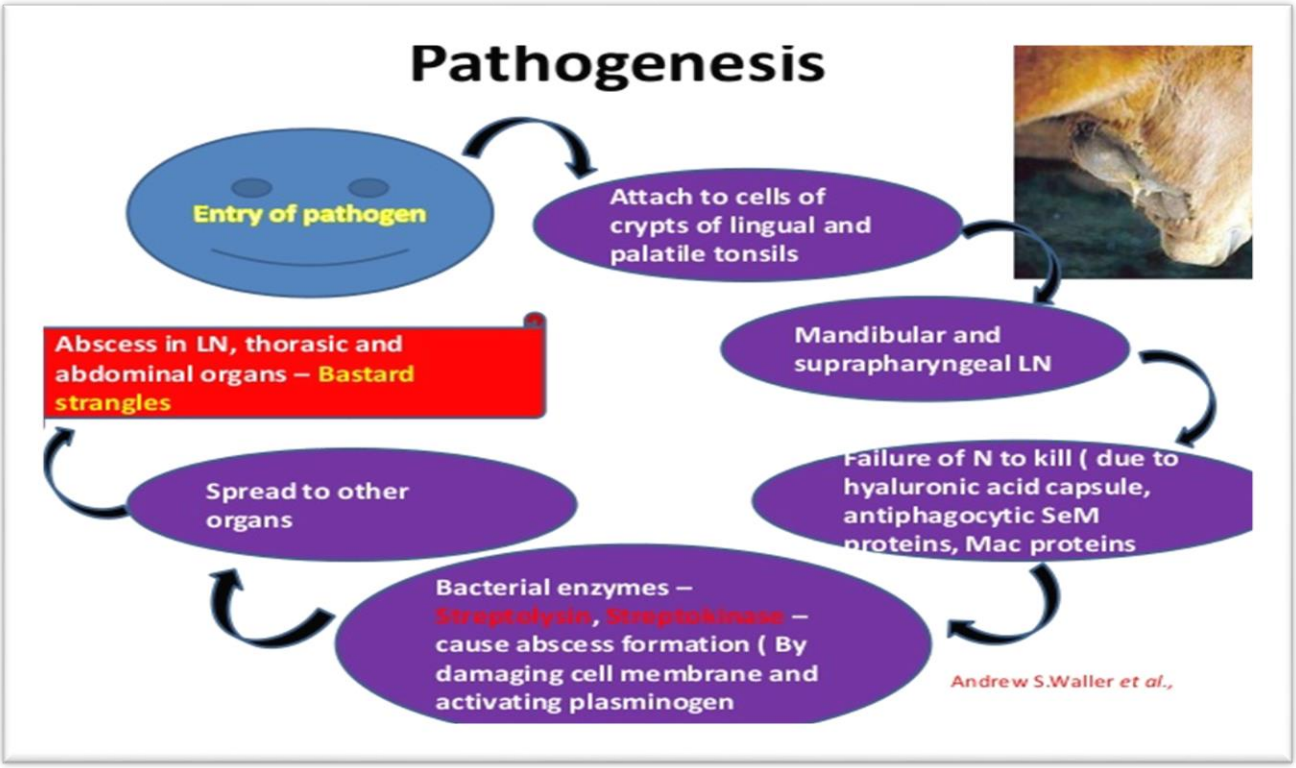
- ❖ No vaccine is available for animal use.
- ❖ Positive animals should be isolated & slaughtered immediately & carcass should be disposed off by incineration or deep burial
- ❖ **Glanders & Farcy Act ,1899.**

Strangles (Synonyms: Equine distemper)

- acute infectious disease of equine, characterized by upper respiratory tract infection and abscess of lymph nodes in throat region.
- *Streptococcus equi*, G+ve
- World wide (Mostly 1-5 years of age)
- As a sequelae of strangles, purpura haemorrhagica (allergic reaction to streptococcal antigen) or empyema of guttural pouches

Transmission

- Direct or indirect contact
- Direct contact (Incubating strangles/just recovered animals/carriers)
- Indirect contact (contaminated stable (buckets, feed, walls, doors) or pasture environment (grass, fences, but almost always the water troughs



- Susceptible horses develop strangles *within 3 to 14 days* of exposure.
- More typically of strangles, horses develop a nasal discharge (initially mucoid, rapidly thickening to purulent), a soft cough, and slight but painful swelling between the mandibles along with swelling of the submandibular lymph node.
- With the progression of the disease, abscesses develop in the submandibular (between the jawbones) and/or retropharyngeal (at the back of the throat) lymph nodes.
- The lymph nodes become hard and very painful, and may obstruct breathing
- The lymph node abscesses will burst in 7 to 14 days, releasing thick pus heavily contaminated with *S. equi*.

1. Bastard strangles (Metastatic strangles):

- ✓ Dissemination of infection to unusual sites other than the lymph nodes draining the throat. E.g abdominal or lung lymph nodes may develop abscesses and rupture

2.Purpura haemorrhagica:

- immune-mediated acute inflammation of peripheral blood vessels that occurs from the formation of immune complexes between the horse's antibodies and bacterial components.

3. Guttural pouch empyaema (filled with pus): Persistent infection in the guttural pouch may lead to inspissation (drying) of pus and, in some cases, the formation of a solid, stone-like, concretion called ***chondroid***.

- Animals that have persistent infection of the guttural pouches become the carriers, the major source of infection to spark outbreaks in susceptible horses
- **Procaine penicillin** used in treatment

Equine infectious anemia

- Swamp Fever, Mountain Fever, Slow Fever, Equine Malarial Fever, Coggins Disease, American Surra
- EIAV – lenti virus
- Ist case in India: 1987 in Karnataka
- Clinical disease occurs in horses and ponies and donkeys may be asymptomatic
- mechanical vectors : Horse flies, stable flies, deer flies
- Normocytic normochromic anaemia

Acute & subacute

- * intermittent fever
- * Jaundice
- * oedema
- * Enlargement of Spleen
- * petechiae under tongue and conjuctiva

Chronic

- relapses during recovery period
- Glomerulonephritis – due to Ag Ab complex
- depigmentation in eye with prominent choroidal vessel

Diagnosis: Coggin's Test - AGID

African horse sickness – equine plague

- non contagious arthropod borne disease of equids
- respiratory and circulatory form

- Orbi virus
- endemic in Africa
- Horses> mule, zebra> donkey
- transmitted by cullicoides

Acute or pulmonary form (DUNKOP)

- severe dyspnoea
- pyrexia
- coughing with abundant frothy discharge from the nostrils
- common in virulent outbreaks and mostly the affected horses die

Sub acute or Cardiac form (DIKKOP)

- remarkable swelling of head, neck and supraorbital fossa associated with cardiac dyspnoea
- no oedema in limbs
- occurs in low immune animals
- low mortality

Very mild form - only rise in temperature to 41°C

Mixed form : Combination of pulmonary form and cardiac form

PM leisons:

- Dunkop: extensive oedema of lungs and thorax may contain several litres of fluid.
- Mottled red appearance of lungs
- Dikkop: oedematous infiltration of subcutaneous tissue

Equine influenza

- orthomyxo virus
- mucopurulent nasal discharge and dry hacking cough
- H7N7 & H3N8

Colic: any abdominal pain and can come from any abdominal organ, not just the gastrointestinal tract. Colic is the number one cause of death in horses, excluding old age

SPASMODIC COLIC

- It is the most common type of colic
- caused by changes to the diet, inadequate deworming, teeth problems, and excessive intake of cold water following exercise
- About 80% of colics are Spasmodic
- In a Spasmodic Colic, a section of the gut goes into a spasm

IMPACTIVE COLIC

- The most common cause of colic is impactions of the large intestine.
- Most impaction colic cases occurs at the pelvic flexure.
- Poor dental care, irregular feeding or where the horse suddenly starts to eat its bedding are important factors

OBSTRUCTIVE COLIC

- This term refers to an uncommon sudden occlusion of the intestinal lumen by organized impaction.
- This may be due to enteroliths, phytoliths, hardened fecal mass, foreign body (hair ball) & clump of parasites.
- It divided into two group

- Vascular/ Functional obstruction

- Mechanical obstruction.

SAND COLIC

- This is most likely to occur in horses that graze sandy or heavily grazed pastures.
- Ingested sand or dirt → accumulates in the pelvic flexure, right dorsal colon and the caecum of the large intestines→ irritates the lining of the bowel → diarrhea.
- The weight and abrasion of the sand/dirt →inflammation of bowel wall →reduced colonic motility→ peritonitis in severe cases.
- Sand→ irritation and impaction of intestines

DIFFERENTIAL DIAGNOSIS OF COLIC IN HORSE

Profiles	Spasmodic colic	Tympanitic colic	Impactive colic	Obstructive colic
Pain	Intermittent	Continuous	Continuous	Continuous
Temperature	Normal	Slight rise	Slight rise	Slight rise
Pulse rate	Increased during attack	Increased	Increased	Increased
Respiration	Increased during attack	Increased; Dyspnoea	Increased; Dyspnoea	Increased; Dyspnoea
Visible mucous	Not much altered	Congested	Congested	Congested
Abdominal distension	Absent	Marked	Present but not marked	Marked
Sweating	Only Patchy	Evident	Evident	Generalized sweating
Faeces	No faeces	No faeces	No passage of faeces	No passage of faeces
Muscular tremor	Absent	May occur	May occur	May occur
Vomition/ regurgitation	Absent	Absent	Frequent	Absent
Intestinal sound	Present	Present	Absent	Usually absent
Rectal examination	Negative	Negative	Usually negative	Positive

SYMPTOMS OF COLIC

- Loss of appetite
- Increased pulse rate
- Excess salivation
- Frequent attempts to urinate or defecate
- Abdominal pain
- Pawing, stretching and rolling
- Flank watching
- Biting the stomach
- Decreased faecal output
- Repeated lying down and rising
- Depression, lip curling & dog sitting posture

CANINE PARVO INFECTION

Etiology:

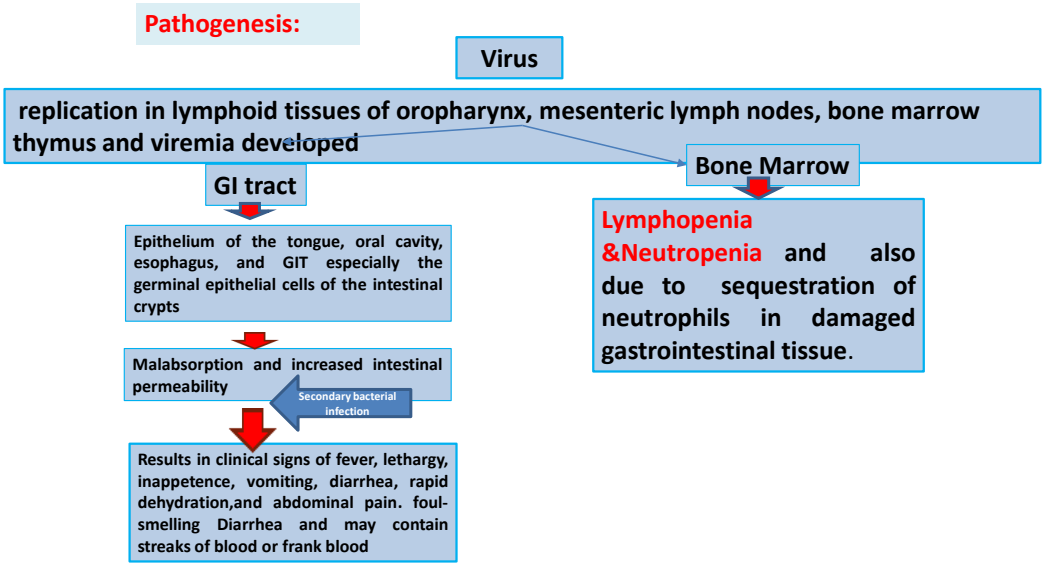
- canine parvovirus-2 (CPV-2),
- antigenically related to Feline Panleukopenia and Mink Enteritis

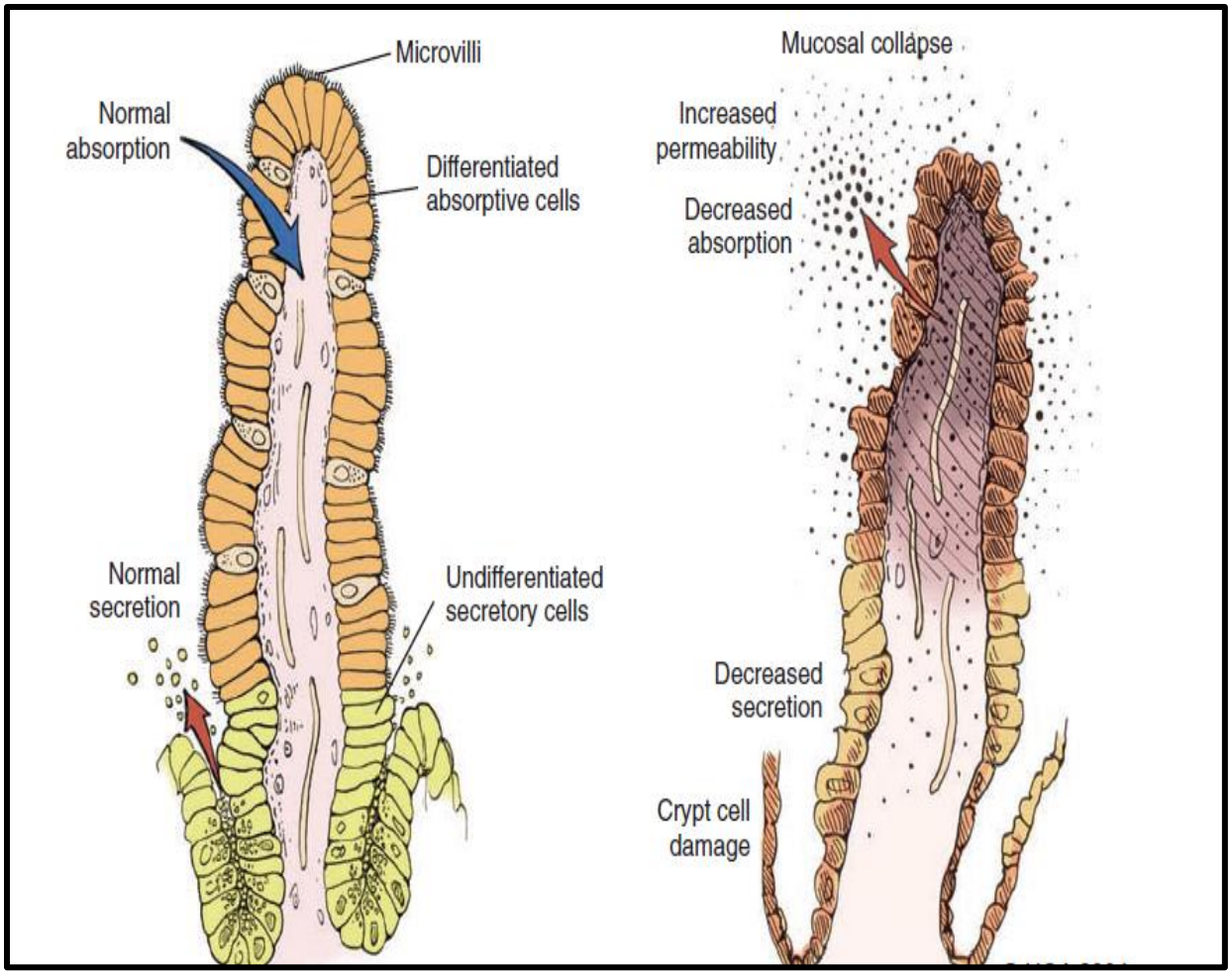
Host affected:

- Dogs, especially less than 12 weeks of age, in unvaccinated or improperly vaccinated adult dogs.
- Rottweiler And Doberman – most severe form observed

Transmission:

- Faecal-oral route and virus that persists on fomites.





CPV infection has been associated with three main tissues

— GI tract, bone marrow, and myocardium — but the skin and nervous tissue can also be affected

- **foul-smelling, bloody diarrhea in a young dog because of putrefaction of proteins**
- **CBC (leukopenia, neutropenia, and lymphopenia)**
- **Increase permeability of villi membrane**

Infected dog sheds virus for 7-14 (10days) in faeces after recovery

CANINE DISTEMPER (Carre's disease, hard pad disease, old dog encephalitis. Saint Vitus dance)

- Canine distemper virus (CDV), belongs to the genus Morbillivirus (family, Paramyxoviridae)
- closely related to human measles virus and rinderpest virus
- Dogs and other Canidae such foxes, and wolves; Procyonidae (raccoons, pandas); Mustelidae (ferrets, mink, skunks, otters). Large wild Felidae can also be affected

- Biphasic fever

CLINICAL SIGNS

Forms of CD virus infection

- Respiratory form
- GI form
- Nervous form
- ocular form
- cutaneous form

Respiratory form

GI form lead to inappetence, vomiting, diarrhea, electrolyte abnormalities, and dehydration

Ocular form : keratitis and corneal ulcers

Nervous form: progressive neuronal necrosis and atrophy which do not resolve, so dogs that recover often have residual neurologic deficits.

3 peculiar signs manifested are as

- ✓ 1.*Old dog encephalitis* - poorly characterized progressive immune-mediated demyelinating leucoencephalomyelitis , which occurs weeks to years after recovery from the acute infection
- ✓ 2.Myoclonus (involuntary twitching of isolated muscle groups) or chorea is common
- ✓ 3.chewing gum seizures: which localized to the head and jaw, with accompanying foamy hypersalivation.
- ✓ Other neurologic signs include, seizures, tremors, opisthotonos,, ataxia, and, less commonly, behavioral abnormalities, compulsive pacing, head tilt and circling
- Ocular form : keratoconjunctivitis sicca (KCS), keratitis, and optic neuritis
- Enamel and dentin hypoplasia in recovered animal.

Cutaneous form: hyperkeratosis in the regions of footpad and nasal epithelium. Puppies with hyperkeratosis feet, also known as hardpad, have thickened, crusty footpads

- The most common secondary infections in distemper are secondary bacterial infections that contribute to bronchopneumonia is Bordetella bronchioseptica.
- The transplacental infections may cause infertility, stillbirth, or abortion and with neurologic signs in puppies that are less than 4 to. 6 weeks of age

INFECTIOUS Canine hepatitis
(ICH / Rubarth's disease)

ETIOLOGY:

- canine adenovirus 1(CAV-1), a DNA Virus
- CAV 2 used in vaccines
- Systemic disease
- Any age but, young dogs, in the first 2 years of life, are more likely to die than older one

HOST RANGE

- Dogs and other canis including foxes, wolves, coyotes, skunks, and bears
- The virus has a predilection for hepatocytes, vascular endothelium, and mesothelium
- In dogs, causing acute hepatitis, respiratory or ocular disease.

TRANSMISSION

- Most often via the oral route by contact with urine from infected dogs.
- Recovered animal shed virus up to 6 months in their urine

CAV-1 not provide cross immunity and CAV-2 wile CAV-2 do… That's why CAV-2 used in vaccines

CLINICAL FINDINGS

PERACUTE FORM:

- Death within few hour after the onset of clinical signs
- High temperature, enlarged tonsils and red colouration of buccal mucosa
- **Saddle shaped curve of fever**

ACUTE FORM:

- Tucked up abdomen with pain on palpation at liver region
- Defective clotting mechanism
- " **Blue Eye disease**", a transit corneal opacity
- ***Intranuclear inclusion bodies***

Kennel cough - Canine Infectious Tracheobronchitis

Etiology: Canine parainfluenza virus (CPIV) and Bordetella bronchiseptica

Other infectious agents : Canine adenoviruses (especially CAV-2), canine herpesvirus, reoviruses (types 1, 2, and 3), and mycoplasmas

- Transmission through fomites (e.g., personnel, cages, food and water bowls)
- Dogs infected with CPIV or CAV-2 shed the virus for only 1 week following recovery
- However, dogs infected with B. bronchioseptica or mycoplasmas can become chronic carriers with persistent shedding

Rabies (hydrophobia)

- Lyssa, Mad dog disease, Jalatanka, Hydrophobia, Rage, Tollwut

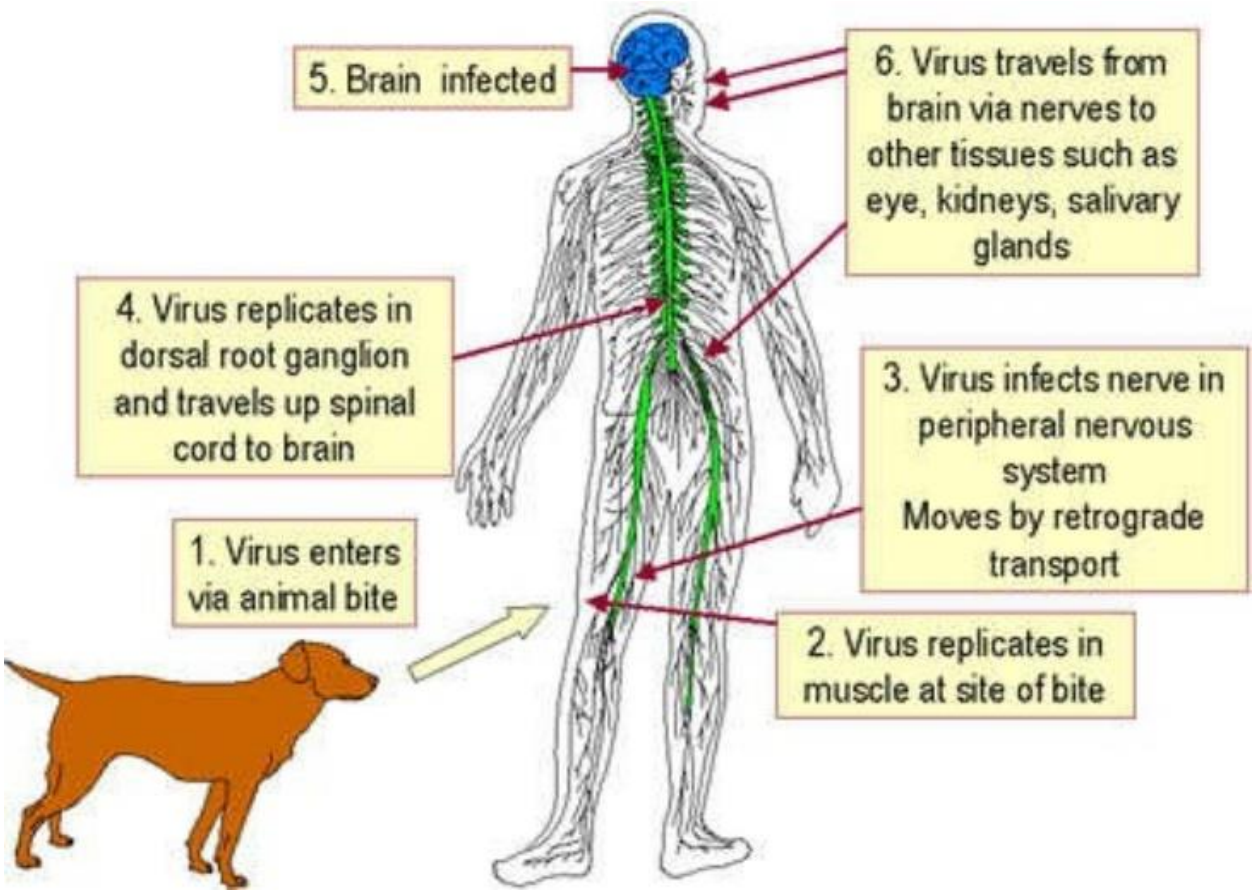
- Non Suppurative viral encephomyelilitis of warm blooded animals - altered behavior, aggressiveness, ascending paralysis and death

- Caused by Lyssavirus type 1 (Rhabdoviruses) – neurotropic virus
- suscepitble to most of the disinfectants like 70% alcohol, carbolic acid and iodine
- Street virus: This is the virus which is existing in nature.
- Fixed virus: This strain is obtained by passaging the street virus in the brain of rabbits, 50 or more times. It is called fixed virus because its characteristics like incubation period, formation of negri bodies etc., are fixed.
- zoonotic disease of warm-blooded animals
- Transmitted by bites or licks of rabid animals
- Characterized by a long and variable incubation period (varies from days to months) a short period of illness due to encephalomyelitis ending in death
- Blood sucking Bats Play important role In Rabies transmission
- Urban cycle and sylvatic cycle – 10^6 infectious units/ml of saliva in wild animals like jackals, fox, wolf

Transmission

- According to WHO, dogs were responsible for 91% of all human rabies cases followed by cats (2%), other domestic animals (3%), bats (2%), foxes(1%) and all other wild animals (less than 1%).
- through the bite of an animal

- can be transmitted to human beings when they handle the oral cavity of the rabid animal with bare hands having abrasions, wounds/cuts



Clinical Signs in Dog

- Furious form- mad dog syndrome causing change in behavior, tendency to run away, biting behavior paralysis of pharyngeal muscles, excessive salivation, coma and death. If not die then passes to paralytic phase
- Dumb or paralytic form-when furious phase is extremely short or absent animal enters in dumb phase
- lazy, sleepiness, paralytic behavior in throat and masseter muscle, lower jaw paralysis and death within 3 day
- Paralysis of the throat and masseter muscle, dropping of lower jaw is common sign in dumb form in dogs

Farm animals

- cattle most commonly affected

- knuckling of hind fetlocks, sagging and swaying of hindquarters, flaccidity of tail to one side, drooling of saliva, pumping of anus, tenesmus followed by recumbency in paralytic form
- decreased sensation over hind quarters – best criteria for detection of rabies
- furious form: alert, hypersensitive, violently attack, bellowing, abdominal pain, diarrohea, paralysis of hind quarters and death

Microscopic lesions

- Main lesions consists of collections of proliferating glial cells known as "Babes nodules"
- Negri bodies in rabid brain- intracytoplasmic basophilic granules
- **Seller's stain method or Mann's method**
- Negri bodies in herbivores – purkinje cell's of cerebellum
- in carnivores – hippocampus

Dignosis:

- ✓ **Corneal imprint method:** From a case suspected for rabies, corneal imprint is taken and then the FAT is performed.
- ✓ **FAT (Fluorescent Antibody Test) – most widely used**
- ✓ gold standard for rabies diagnosis is FAT
- ✓ **Recommended by WHO & OIE**
- ✓ mouse inoculation test: brain preserved in 50% Glycerol saline used
- ✓ **ELISA, RT-PCR, virus neutralization test**

Treatment & Prevention:

- **Prophylactic vaccine**
- **post bite vaccination schedule: 0,3,7,14,28/30,90 days**

Rabies virus with a potency of ≥ 2.5 IU per dose

Salmonellosis

- *Salmonella*-causative agent of salmonellosis (most cases associated with serovars) of *Salmonella enterica*
- The most common type of infection is the carrier state.
- Clinical disease is characterized by two major syndromes:

systemic septicemia (also termed as typhoid) and an enteritis.

- Other less common clinical presentations include abortion, arthritis, respiratory disease, necrosis of extremities, and meningitis
- Young calves, piglets, lambs, and foals may develop both the enteritis and septicemic form.
- **cattle**— *S.* Typhimurium - Commonly affects calves under 6 months of age , Dublin - affects both calves and adults equally, and Newport;
- **sheep and goats**—S. Typhimurium, Dublin, Abortusovis, Anatum, and Montevideo;
- **pigs**—S. Typhimurium and Choleraesuis;
- **horses**— Typhimurium, Anatum, Newport, Enteritidis
- **poultry**— Enteritidis, Typhimurium, gallinarum, and pullorum.
- Salmonella spp. are mainly transmitted by the fecal-oral route.
- Vertical transmission occurs in birds, with contamination of the vitelline membrane, albumen and the yolk of eggs.

a. Enteritis

- -salmonellae invade the intestinal wall and produce enterotoxins that cause nausea, vomiting and diarrhea

b. Enteric fever (Typhoid):

- Two serotypes Typhi and Paratyphi can cause typhoid
- Wandering macrophages that contain salmonellae act as "taxi/cabs" that deliver salmonellae to various reticuloendothelial tissues.
- Pullorum disease or bacillary white diarrhoea (Salmonella Pullorum)
- Fowl typhoid (Salmonella Gallinarum)

Rickettsial Diseases

- Rickettsial diseases (rickettsioses) and related diseases (anaplasmosis, ehrlichiosis, Q fever, scrub typhus) are caused by a group of gram-negative, obligately intracellular coccobacilli. All, except for *Coxiella burnetii*, have an arthropod vector.

Canine Ehrlichiosis

- Tick-born disease of dogs,
- Canine Typhus ; Tracker Dog Disease & Tropical Canine Pancytopaenia (TCP)

- *Ehrlichia canis* - A parasite of *a monocyte in the peripheral blood*
- High fever and Lowered peripheral blood cell counts(pancytopenia)
- Dogs and some species of wild canidae
- German Shepherd-more susceptible

Transmission:

- *Rhipicephalus sanguineus (*brown dog tick) -Major vector
- Others - *Boophilus, Dermacentor, Ixodes, Hyalomma & Argas*

Clinical Signs:

- Acute stage: Fever and epistaxis or other hemorrhages - Lowered peripheral blood cell
- Subclinical phase-No outward signs and Last for the rest of the dog's life-Carrier states
- Chronic phase-The third and most serious stage
- Pancytopenia
- Extensive haemorrhage (bleeding)with epistaxis
- Lameness
- Neurological, ophthalmic disorders, and kidney disease

Necropsy Lesion:

- Emaciated carcass
- Pale mucous membranes
- Oedema of the limbs, ascites and hydro-pericardium
- Hemorrhages in GIT, internal organs, s/c tissues and eye
- Enlarged lymph nodes and spleen

Q Fever – Abattoir disease

- Nature of the disease
- zoonotic disease
- abortions in Ruminants and pets
- biological weapon

- Caused by: Coxilla Burneti
- Susceptible Species: Cattle, sheep, goats, and other domestic mammals,
- Transmission: Inhalation of a spore-like small-cell variant, Contact with the milk, urine, feces, vaginal mucus, or semen of infected animals.

Clinical Signs:

- Late Abortion
- Stillbirth
- Weak calves
- Repeat breeding
- Oxytetracycline is the DOC
- Necropsy Lesion
- Necrotsing placentitis in ruminants
- C. burnetii can localize in mammary glands, supramammary lymph nodes, placenta, and uterus, from which it may be shed in subsequent parturitions and lactations.

Psittacosis/Chlamydiosis

- Psittacosis/Chlamydiosis: disease in psittacine birds, humans and animals
- Ornithosis: Disease affecting birds other than psittacines.
- C. trachomotis – mainly humans and other mammals .
- C. psittaci- humans, mammals and poultry.
- C. pneumoniae- humans.
- C. pecorum- ruminants and other mammals.
- Transmission
    - Directly - Close contact
    - Indirect - Fomites, biting insects, mites and lice
    - Mainly by inhalation - Infected dust
    - No transovarian transmission

Host affected:

Turkeys are highly susceptible

Ducks, pigeons and chickens are rarely affected

Young birds are more susceptible than older birds

Psittacine birds, ducks and turkeys cause severe infection in human being

- Elementary body: A small, infectious form, measures 250- 300 nm diameter which is surrounded by a rigid walled.
- Reticulate body : A larger, non infectious form, measures 400- 600 nm diameter, flexible walled, develops from a small elementary body.
- Chlamydia psittaci: chlamydiosis in birds
- findings like conjunctivitis, fibrino- pericarditis, splenomegaly and anemia
- Cattle, sheep & goat
- – respiratory and reproductive disorder, mastitis, enteritis, conjunctivitis, encephalomyelitis

Parasitic Diseases

Trypanosomiasis

- intercellular in blood and lymph except T. cruzi
- transmitted by blood sucking Parasites except T. equiperdum
- Surra/ Tibarsa / Murine caused by T. evansi
- first reported by Sir Griffith Evans in 1818 in India- horse and camel in Dera Ismail khan
- blood sucking flies – major role in transmission
- intermittent fever, anemia, oedema of dependent parts, nervous signs

I Salivaria (pathogenic)			
1	*T. vivax*	Cattle, Sheep, Goat & Cattle	Glossina spp (Tse-tse flies)
2	*T. congolense* (Nagana)	Cattle, Sheep, Horse & Pig	Glossina spp
3	*T. brucei* (Nagana)	Cattle, Sheep, Goat, Horse, Pig, Dog & cat	Glossina spp, Tabanids
4	*T.evansi*	Equines, Dog, Cattle , Sheep, Goat, Cat, camel, Elephant	Tabanids, Stomoxys & other biting flies
5	*T. equinum* (Mal-de Caderas)	Equines	Tabanids, Stomoxys & other biting flies
6	*T.equiperdum* (Dourine)	Equines	Coitus (rarely tabanids)
7	*T. simiae*	Pigs, cattle, horses, Sheep & Goat	Glossina spp
8	*T. suis*	Pigs, Sheep, Goat & dogs	Glossina spp
9	*T. rhodesiense* (Sleeping sickness)	Man, ruminants (domestic & wild) Zoonotic	Glossina spp
10	*T. gambiense* (Sleeping sickness)	Man, also animals	Glossina spp

1	*T. theileri*	Cattle	Tabanids, Hippobosca maculata
2	*T. melophagium*	Sheep	Melophagus ovinus (sheep ked)
3	*T. theodori*	Goat	Hippoboscid
4	*T. cruzi* (Chaga's disease)	Man, Dog & cat	Reduvlid bugs

Surra

- T. evansi – intermittrnt fever, oedema of dependent parts followed by nervous symptoms

- Vectors: Tabanids, Stomoxys, Haematopota, Hippobosca, Ornithodorus tick.
- in dogs : from ingestion of infected meat
- transmitted due to intermittent feeding habit of flies
- forms: per acute, acute, subacute, chronic, latent
- microcytic hypochromic anemia

Pathogenesis

A. Progressive anaemia may be due to

- Haemolysin released by the parasites which leads to haemolysis of RBCs.
- Increased erythrophagocytosis.

B. Hypogylecmia – due to malfunction of adrenal, pancreas and thyroid glands, the metabolism of carbohydrate hampered.

- Trypanosomes also consume large quantity of blood sugar (glucose)

Clinical Signs:

Cattle

- Per acute - Death within in 2-3 hour; nervous form and death in convulsion
- Acute - staggering gait, encircling movements, nervous excitement
- beating head against wall /manger, apparent blindness, stamping of feet, bellowing, twitching of muscle, shivering of body, coma and death within 6-12 hours
- PM findings: Splenomegaly, hepatomegaly, enlargement of lymph nodes and kidney, petechial haemorrhage at the junction of skin and mucous membrane

EQUINES

- Severe in horses while Donkeys are resistant
- Intermittent fever and progressive weakness and anemia are main symptoms
- Transient local/ urticarial eruptions/ plaques on neck/ flanks; edema of legs and lower parts of the body

Canines- edema of head & thorax, corneal opacity and blindness, oedema of larynx leads to changes of voice similar to those found in rabies.

Diagnosis

- Blood smear examination: Wet, Dry, buffy coat smear – intercellular organisms
- Mercuric chloride test, Thymol turbidity test: for camels
- Stilbamidine test: Bovines
- Formal gel tube test: equines and camels

Treatment

- Quinapyramines (Antrycide prosalt) - Prophylactic - Drug of choice.
  - Quinapyramines sulphate – therapeutic
  - Quinapyramine chloride – prophylactic
- Antrycide prosalt @ 3-5mg/kg as 10% solution by S\C route
  - 4-6 months immunity
- Suramins (Sulphonated naphthalamines) – Curative
- Diamidines: Diminazene aceturate (Berenil)@ 3.5mg/kg – Curative
- Homidium bromide & Isometadium chloride

Dourine

- Equine Syphilis, Breeding paralysis
- Horses are highly susceptible than mules/ donkeys
- Transmitted by coitus
- Dourine Act 1910

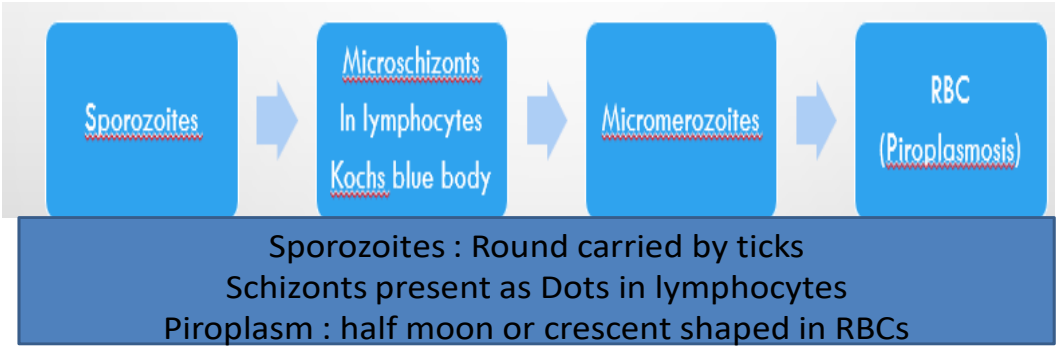
Clinical Signs:

- Phase of edema (4-6 weeks): edema of genitalia, nymphomania, mucoid vaginal or urethral discharge and ulcers in vaginal mucosa; In severe cases, abortion and frequent micturition
- Stallion- Swelling of prepuce and scrotum
- Urticaria phase: Oval plaques under skin on flank called Dollar spots
- Paralytic phase: In coordination, unilateral paralysis of hind limbs, nostrils, ears, lips, recumbency and death(50-70%)

Theileriosis

- Bovine theileriosis – tick borne disease caused by T.annulata and T.hirci
- fever, anemia along with respiratory and digestive disorders
- Bovine Tropical theileriosis/Mediterranean coast fever: caused by Theileria annulata
- East coast fever: Theileria parva
- It is transmitted by Rhipicephalus appendiculatus

T. annulata	**Tropical theileriosis (common in India)**
T. parva	East coast fever in African countries
T. sergenti	Bovine theileriosis in Japan, korea
T. hirci	Virulent in sheep, goat
T. ovis	Avirulent



Transmitting agent Theileria annulata -Hyalomma anatolicum anatolicum (Three host tick) - acts as constant source of infection to susceptible animals

- Exotic and Cross breds – most susceptible

Clinical Signs

- enlargement of prescapular lymph node (in young calves- parotid L.N.)
- Fever, severe anemia, weight loss
- Generalized anemia, icterus and abortion
- Cerebral form in young calves – Nervous signs

Diagnosis:

lymph-node aspirates examination - Koch Blue bodies

Punched necrotic ulcers in abomassum

Treatment: Buparvaquone @2.5mg per kg body weight

- Parvaquone, Halofuginone and Menoctone drugs are effective against clinical infections with *Theileria annulata*

Prevention: Raksha vac –T : vaccine available against theileria

Babesiosis

- Piroplasmosis, Texas Fever, Tick Fever, Red Water Disease
- found inside the RBCs (intraerythrocytic) of the infected hosts.

- fever, anemia, hemoglobinemia, hemoglobinuria and Jaundice
- large piroplasm characteristically pear shaped- lie in pairs forming an angle in RBCs

- The angle between the two parasite in large form is acute and in small form is obtuse

Zoonotic Species :-

- ***Babesia divergens*** **(Small form)**
- ***Babesia bovis*** **(Small form)**
- ***Babesia microti*** **(Small form)**

Animal	Species involved
Cattle, buffalo	Babesia bigemina; Babesia bovis; Babesia divergens; Babesia major.
Horse	Babesia equi; Babesia caballi.
Sheep	Babesia motasi; Babesia ovis.
Pig	Babesia trautmanni.
Dog	Babesia canis; Babesia gibsoni.
Cat	Babesia felis.

Buffalos: B. bigemina and B.bovis

Maltese cross pattern – B.equi

Cattle	**Boophilus annulatus, Rhipicephalus and Hemosyphilus**
Equine	**--do----,Dermacentre**

Dog	**Hemophysalis, Rhipicephalus**
Sheep	**Hemophysalis, Rhipicephalus, Dermacentre**
Pig	**Rhipicephalus, Boophilus**

Symptoms

- High fever
- Urine becomes coffee colour due to haemoglobinurea. Hence the disease called red water disease.
- Anaemia
- Pale mucous membrane (Jaundice)
- Pipe-stem diarrhea
- Hepatomegaly and splenomegaly
- Death in untreated case.

Treatment

- Inverse age relationship
- intravascular hemolysis leading to Hemolytic anemia
- Death due to anemic anoxia
- Treatment – Diaminazine aceturate, Imidocarb

Anaplasmosis/ Gall Sickness

- Anaplasma marginale – cattle in India

- A. centrale – cattle in U.S.
- A. ovis – Sheep & Goats
- disease of adult cattle and clinical anaplasmosis is mostly seen in cattle above 18 months of age.
- major vector: Ixodid ticks & tabanus fly
- Boophilus (A.marginale) and Dermacentre (A.centrale) also transmit the disease

Metabolic Diseases

Milk Fever (Parturient paresis, Hypocalcaemia, Calving paralysis)

- Disease of female
- Occurs in cattle, Buffalo, sheep and goats at or within 72 hrs. of parturition
- caused by hypocalcaemia and characterized by weakness, recumbency, and ultimately shock and death
- "Fever" is a misnomer
- Sometime it is associated with low level of Ca, P & Mg, - "Milk Fever Complex"

Etiology

- onset of lactation results in the **sudden loss of calcium through milk**.
- Serum calcium levels fall from normal of 10-12 mg/dl to 2-7 mg/dl
- sudden increase in the requirement of calcium for the production of colostrum
- impairment of absorption of calcium from the intestine at parturition
- mobilization of calcium from storage in the skeleton may not be rapid enough to maintain normal serum levels which may be due to parathyroid insufficiency.
- Deficiency of Vit D3
- Improper Ca: P
- Excessive Ca feeding during Dry period

PATHOGENESIS

- Clinical signs **appears when Ca falls below 5.5 mg/dl**
- Hypocalcemia causes muscle weakness, hypothermia and depression of consciousness
- Atony of skeletal & smooth muscles and marked reduction in cardiac output

- Reduction in arterial blood pressure
- Reduction in ruminal & abomasal tone & motility
- Clinical signs of early excitement, muscle twitching, tetany of hind limbs, hypersensitivity and convulsions of head & neck.
- Failure of neuromuscular transmission of stimuli in cows with parturient paresis

Clinical findings

Stage I/Stage of excitment:-

- ➢ Temp. Normal
- ➢ Hypersensitiveness
- ➢ Tremor of the muscle of the hind limb
- ➢ Hind limb become rigid & thereby ataxic for which animal is prone to fall

Stage II/ sternal recumbency

- ✓ Sub normal temp.
- ✓ Cold skin & extremities
- ✓ Loss of anal reflex
- ✓ Low venous pressure(Difficult to raise the jugular vein)
- ✓ Suspended defecation & urination

Stage III/ lateral recumbency

- ✓ Markedly sub normal temp.
- ✓ Extremely cold skin & extremities
- ✓ Bloat
- ✓ Difficult/Impossible to raise the jugular vein
- ✓ Suspended defecation & urination

Clinical Pathology

Ca level= 5.5 – 6.5mg/100ml (N.L. = 8.5 – 11.5mg/100ml)

Mg level= 0.8 – 1.2mg/100ml (N.L. = 1.8 – 3.2mg/100ml)

P level= 2.3 – 2.8mg/100ml (N.L. = 3.5 – 6.0mg/100ml)

Diagnosis

- History of parturition
- High milk yield
- Typical clinical signs viz. sternal or lateral recumbency with subnormal body temperature
- Clinical pathology viz. Low serum Ca level
- Clinical response to calcium therapy

Treatment

- Calcium borogluconate (25%) @ 400-800 ml or 1 gm/45 kg body weight slow IV (Av. 450ml bottle contain 8-11g Ca)
- Antihistaminic like Avil 10 ml IM in L.A.
- Ca, Vit.D3 & Vit.B12 inj (Caldee-12 inj.) 10-15 ml IM in

Unusal reaction to Ca therapy can be treated with:-

- Atropine sulphate can be used to overcome cardiac arrhythmia
- Magnesium sulphate 10% solution @100-400ml IV to antagonize cardio-excitatory effects of calcium

Prevention & Control

- Feeding of low Ca diet(8 g/day/450 kg b.wt.) during last 2 week of gestation
- Incomplete milking after calving
- Protect from cold stress & transportation stress
- Use of dietary straw to stimulate rumen function
- Oral Ca supplement, one at calving and a second dose the next day
- A single dose of Vit D3 @ 10 million unit i.e. 1 million units/ 45 kg body weight IM one week before calving has been reported to be effective
- calcium-binding agents such as zeolite, zinc oxide etc

Downer Cow Syndrome

- Complication of periparturient hypocalcaemia clinically characterised by prolonged recumbency even after two successive treatments with calcium

- Etiology: low Ca levels
- Low P, Mg, K, levels
- Following periparturient paresis a cow may develop DCS due to nerve injuries and over stretching of nerves or due to pressure on nerves while in recumbency

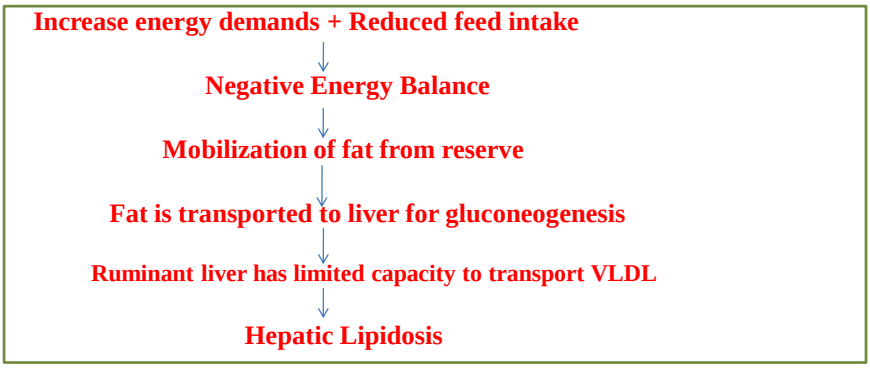
Clinical Signs:

- Creeper/alert downers:
  - Cow is unable to rise and remains in recumbent position.
  - Cow remains bright and alert.
  - Appetite, rumination, defecation and urination are usually normal.
  - Temperature is usually normal but may turn towards sub-normal range in the terminal stage disease.
  - The affected cow usually crawls around utilizing the forelimbs whereas hind limbs remain in flexed position. This type of stance is ascribed as "creeper cow"
- Non alert downers:
  - dull, depressed, lateral recumbency
  - Complete loss of appetite
  - Decubital ulcers
- downer cow which continues to remain down for more than 7 days ends fatally.

Fat Cow Syndrome

- FATTY LIVER IN CATTLE, FAT-MOBILIZATION SYNDROME, HEPATIC LIPIDOSIS
- disease of high producing cows which were Overfed during dry period may develop fatty liver syndrome just before or after calving (Transition period)
- Etiology- Mobilization of excessive body fat to liver during periods of negative energy balance at time of parturition or in early lactation of dairy cows and late pregnancy of beef cows

Pathogenesis



Clinical findings

- Anorexia
- Depression
- Weakness
- Persistent weight loss
- Recombency
- Ketosis (Do not respond to glucose treatment)
- Some animals show nervous signs like star gazing and tremors of head and neck
- Coma and death

Treatment

- Continuous supplementation of glucose along with calcium and magnesium salt to correct negative energy balance
- Long acting protamine zinc insuline @ 200 units/ cow every 12 hrly
- S/c administration of choline chloride (25 g in 250 ml NSS)
- Niacin@ 6-12g/cow/day helps in reducing hepatic lipidosis
- Antioxidants
- Anabolic steroids @300mg/cow

Eclampsia

- Commonly found in bitches with large litters
- Clinical Signs: Panting and restlessness, Tremors, twitching, tetany, muscle spasms, & gait changes (stiffness and ataxia)
- Slow IV administration of 10% calcium gluconate @ 0.5-1.5 mL/kg slow IV

BOVINE KETOSIS (Acetonemia, Ketonemia)

- metabolic disease of lactating dairy cows characterized by weight loss, pica, inappetance, decreased milk production, and neurologic abnormalities that usually occur during the first 6-8 wk of lactation.
- accumulation of abnormal amount of ketone bodies in tissues and tissue fluids which are **acetone, acetoacetic acid and beta hydroxy butyric acid**
- More common in 6 to 10-year-old animals during 3^{rd} to 5^{th} lactation
- More incidences in confined **stabled dairy cows that are improperly fed and conditioned during the dry period and early lactation.**
- More occurrence during first 6 weeks of lactation
- High yielders are more susceptible

Etiology

- **negative energy balance in the 6-8 wk after parturition due to**
- undernutrition
- high milk yield
- hepatic insufficiency
- hormonal deficiency: like insulin, thyroxine, glucocorticoids
- deficiency of certain minerals like cobalt, chromium, phosphorus
- infectious and systemic disease that results in loss of appetite
- excessive feeding of silage which is rich in butyrate

Clinical Signs

Digestive form – more common

- selective appetite: refusal to concentrates

- marked drop in milk production
- firm dry mucus-covered stools
- Lethargy, marked weight loss and emaciation
- acetone odour to the breath, urine, or milk
- decreased ruminal motility
- Nervous form: less common – due to isopropyl Alcohol
- frenzy and aggression
- apparent blindness
- Pica/ depraved appetite
- compulsively lick metal stanchions, mangers, or their own bodies.
- Head or nose pressing may occur along with chewing and bellowing.
- Abnormal walking with staggering, circling, and falling.

Clinical pathology

- Blood glucose levels: < 25 mg/dl (NL 40-60mg/dl)
- Blood ketone levels: 20-100 mg/dl (NL <10)
- Ketone bodies in urine 80-100mg/dl (NL <10)
- Milk ketone levels: 10 to 40 mg/dl (normally absent)
- Increased total lipids, triglycerides, cholesterol
- Urine and milk samples test positive for ketone bodies (Rothera's test)

Diagnosis

- History and clinical findings: recent parturient, high yielder, selective appetite, **sweetish smell in breath** and milk, dropped milk yield
- Clinical pathology
- Response to treatment

Treatment: The treatment should be initiated through:

- **Replacement therapy**
- **Hormonal therapy**

- **Miscellaneous therapy**

Replacement therapy

- Routine treatment is IV administration of 500 mL of 50% glucose and IM administration of the glucocorticoid like Dexamethasone, Isofluperodone
- **Propylene glycol (225 g, b.i.d. for 2 days, followed by 100 g, daily for 2 days) or other glucose precursors are also administered PO in many cases**

Hormonal therapy

- Glucocorticosteroids (Dexamethasone, 40mg
- **Insulin as protamine zinc @200-300 IU/Animal, SC every 24-48 hrs. It is given in conjunction with either glucose or corticosteroids.**
- Anabolic steroids (trenbolone acetate), lactational ketosis and ketosis in late pregnant cows that are overfat, stressed, or have twin fetuses, but banned in food animals.

Miscellaneous treatments

- Vitamin B12 and cobalt
- Cysteamine (a biological precursor of coenzyme A) and also sodium fumarate have been used to treat cases of the disease, but not generally adopted.
- The recommended dose rate of cysteamine is 750 mg IV for three doses at 1-3 day intervals.

Prevention

- Cows should be properly conditioned during late lactation and the dry period.
- They should be fed so that body score at calving will be 3.5 on a 5-point scale.
- The ration should contain adequate amounts of essential vitamins and minerals.
- Avoid sudden changes in feed to avoid indigestion.
- *Propylene glycol* in early lactation - 350 to 1000 mL daily for 10 days after calving
- *Ionophores (Monensin)* decreases in Gram positive bacteria, protozoa, and fungi and increases in Gram-negative bacteria. The net effect of these changes in bacterial flora is increased propionate production and a decrease in acetate and butyrate production.
- Various feed additives such as propylene glycol, sodium propionate, yeasts, niacin, choline, etc. have been recommended.

Pregnancy Toxaemia / ovine ketosis

- Metabolic disorder primarily of disease of sheep due to energy deficit in the diet leading to massive mobilization of fat reserves, resulting in hypoglycaemia and hyperketonaemia

Etiology: similar to ketosis (decline in plane of nutrition during last 2 months of pregnancy)

- Occurrence during last 6 weeks of gestation
- More common in ewes carrying twins or triplets
- Incidences increases with age
- More in intensive farming system
- Mainly due to starvation or malnutrition
- CFR up to 100%

Clinical Signs:

- Separation from group, inappetence, grinding of teeth
- Sweetish smell from breath
- Nervous signs
- Recumbency, coma and death

Clinical pathology: Hypoglycemia, ketonemia, ketonuria, metabolic acidosis, increased BUN and creatinine, increased values of liver profile enzyme

Treatment:

- Replacement therapy: Dextrose 5-2O% i/v 500ml
- Protamine Zinc insulin 20-40 units
- Correction of acidosis: Soda bicarb or RL Removal of foetus

Post Parturient Haemoglobinuria (PPH)

- acute, sporadic life-threatening disease of high yielding buffaloes and cattle characterized by hypophosphatemia, intravascular hemolysis, hemoglobinuria and anemic anoxia.
- Buffaloes are more susceptible than Cattle
- More common during 3rd – 6th lactation & 5-10 years of age.
- Mostly occur between 2-4 weeks after calving

- Cold weather and malnutrition

Etiology: Deficency (def.) of P in diet: - Soil def. in P, Hay, straws & plant rich in oxalate are naturally deficient in P

- Impaired absorption of P: - Excess Ca & Fe in diet, Vit. D def., Improper Ca:P ratio
- Due to ingestion of some plants: Feeding cruciferous plants like cabbage, turnip, kales, rape, rye, alpha alpha
- Forage low in Cu, Se & Mo
- Plant rich in saponin, oxalate

Pathogenesis

- Def. of P results in decrease in RBC glycolysis and ATP synthesis. Subnormal concentration of ATP predisposes RBC to altered function & structure, so results in increased fragility & haemolysis
- Increased fragility of RBC due to loss of integrity of phospholipids membrane of erythrocytes because of P deficiency.

Clinical findings

- The animal voiding "dark red-brown" to almost "black" urine.
- Milk is yellowish or reddish in colour
- Pale m.m., or even icteric m.m. in severe cases
- Tachycardia, increased pulse and respiration rates, dyspnoea.
- Normal/subnormal/slightly elevated rectal temperature.
- The extremities and teats are cold to touch
- The disease slowly recovered, as convalescence is prolonged for up to 3-4 weeks (chronic cases) and pica is often observed during this stage
- Death occur due to anaemic anoxia

Clinical Pathology: Serum level of P 0.5-1.5mg/dl (Normal 4-7mg/dl)

- Haemoglobin drops to 6-8 gm%
- TEC drop to 2-3 millions/cumm of blood (
- PCV drop to 2.5-15 (N= about 35)
- Serum bilirubin and BUN raised.

- Low Cu level of blood
- macrocytic, hypochromic anemia

Treatment

- IV administration of 60 g of sodium acid phosphate in 300 ml of distilled water and a similar dose Sc, followed by further Sc injections at 12-hourly intervals on three occasions and similar daily doses by mouth for another 2 to 3 days.
- Blood transfusion in severe cases.
- Oral dosing with bone meal (120 g twice daily) or dicalcium phosphate or a suitable source of Ca & P daily for 5 days is recommended
- Haematinic inj.
- Toldimfos sodium 10-15 ml I/V or I/M for 4-5 days
- Ascorbic acid(Vit. C) @15-20 mg/kg I/V daily for 3-4 days
- Liver extract with B complex

Lactation Tetany / Grass Staggers/ Hypomagnessium tetany

- Highly fatal metabolic disease of ruminants characterized by hypomagnesemia, tetany and hyperaesthesia
- Etiology:
  - Dietary deficiency of magnesium: grazing on young green grass or cereals, soil deficient in Mg
  - Decreased absorption of Mg: presence of chelating agents, diet rich in Nitrogen and Potassium, Diarrhea
  - Increased loss of Mg: excess loss through milk

More common in ruminants in 4-7 years age group

Both pregnant and lactating females affected

More common in inclement weather condition

More commonly observed in animals feeding on young grass which is poor in Mg and rich in N, K

Pathogenesis:

hypomagnesemia -→ 1. Neurointoxication due to hypomagnesemia 2. Change in Mg: Ca leading to release of acetylcholinesterase enzyme leading to metabolism of Ach and impaired neuronal transmission →tetany and hyperesthesia

Clinical signs:

- Acute form: sudden anorexia, muscle twitching, staggering gait, hyperesthesia, tetany, clonic convulsions, high fever and death within an hour
- Subacute form: onset gradual over 3-4 days
- Anorexia, frequent urination and defecation, muscle tremor, trembling of legs, hyperesthesia, convulsions and death
- Chronic form: dullness, anorexia, emaciation, poor milk yield
- Serum Mg levels: decreased to 0.5 mg/dl (NL 1.7-3%)

Equine Myoglobinuria

- Exertional rhabdomyolysis, Tying-up, "Cording-up" Syndrome of Horses, Paralytic myoglobinuria, Monday Morning Disease
- Severe, often fatal multifactorial disease of horses of sudden occurrence characterized by stiffness of muscles, lameness, hardening of muscles and red colored urine

Etiology:

- Carbohydrate overloading
- Local hypoxia
- Thiamine deficiency
- Vitamin E & Se deficiency
- Hormonal imbalance
- Electrolyte imbalance

– Mostly affect draft horses

– Disease occurs during after rest of at least 2 days on full working ration

PATHOGENESIS: large store of glycogen formed during the period of rest in the muscles metabolized to lactic acid

– Accumulation of lactic acid leads to:

- Degeneration of the muscles and liberation of myoglobin (muscle haemoglobin)
- Swelling of muscle (lactic acid is hydrophilic)

Clinical Signs

- In very mild cases: only poor performance
- Mild cases: stiffness in gait
- Severe cases:
- Profuse sweating, stiffness in gait & reluctance to move
- Horse assumes a dog-sitting position followed by lateral recumbency, laying down & repeated attempts to rise, often with nervous signs
- Rapid respiration, weak pulse & temp. may rise to 40.5 C
- Hard board like muscles particularly of hind legs
- Dark-red brown urine (myoglobinuria)

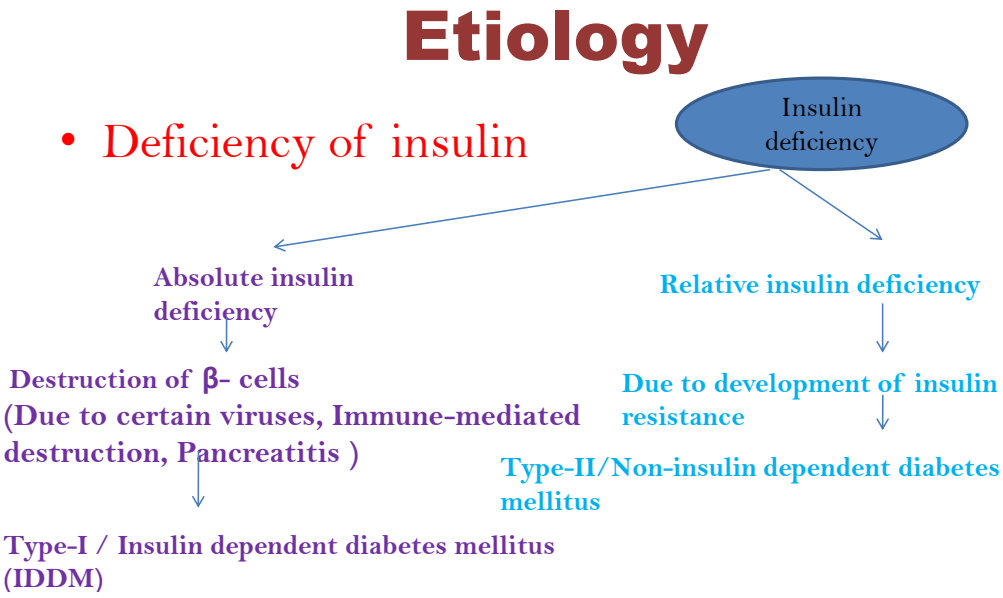
Treatment

- Animal should be kept as quiet as possible, and attempts should be made to keep it standing
- Good nursing care & precautions taken to prevent development of decubital ulcers
- Nervous, restless animals, or those showing evidence of pain, should be given sedatives such as chloral hydrate or tranquilizers
- Give Na bicarbonate IV or orally for alkalization of urine to minimise nephrotoxicity
- I/V injection of large quantities of fluids and electrolytes to maintain high rate of urine flow to avoid renal tubule blockage and subsequent uraemia
- I/M injection Thiamine daily to increase the tolerance of blood to lactic acid by increasing lactic acid metabolism
- NSAIDs such as Flunixin and Phenylbutazone may be used to control the pain
- Antihistaminics, Vit. E & selenium may be useful

Diabetese Mellitus (Hypoinsulinism)

- endocrine disorder, characterized by polyuria, polyphagia, loss of body condition and development of cataract"
- Diabetes is more common in middle-aged dogs of 4-14 years (Highest at 7-9 yrs)

- Females are more affected compared to males
- Poodles, Daschunds, Terrier and Beagle breeds are more susceptible



Clinical Findings

- Polyuria
- Loss of body condition in spite of good appetite
- Weakness
- Lethargy
- Recurrent episodes of skin infection
- Cyctitis
- Bilateral cataract
- Ketotic breath

Avian Diseases

Respiratory Disease Complex

- number of respiratory viruses (Infectious Bronchitis, Avian pneumovirus, Lentogenic Newcastle disease virus, vaccinal and field strains) and bacteria (*Ornithobacterium rhinotracheale*, *E. coli*)

- Predisposing factors: Dust, ammonia and other gases, and other factors associated with poor ventilation
- Signs
  - Snick.
  - Sneezing.
  - Head swelling.
  - Conjunctivitis.
  - Nasal exudate.
  - Rattling noises.
- Post-mortem lesions
  - Severe tracheitis with variable exudate - catarrhal to purulent.
  - Airsacculitis.
  - Pericarditis.
- Treatment: Antimicrobial treatment of specific bacterial infections.
- Prevention: Effective ventilation, sanitation of drinking water, carefully applied appropriate viral vaccines.

RANIKHET DISEASE

- Ranikhet disease, Avian pnuemoencephalitis, Pseudo Fowl Plague, Doyle's Disease, Avian Distemper
- highly contagious and destructive disease of domestic fowls and wild birds caused by a paramyxo virus 1 and characterized by high morbidity and greenish colored diarrhea.
- Chickens are the most susceptible and waterfowl the least susceptible of domestic poultry

✓ On the basis of virulence:

1.LENTOGENIC VIRUS(MILD)

2. MESOGENIC VIRUS(MODERATE)

3.VELOGENIC VIRUS(SEVERE)

1. Doyle's form/ Velogenic viscerotropic form/ Exotic NCD	- **Very acute form** - **Hemorrhagic lesions in digestive tra t**
2. Beach form/ Neurotropic velogenic form	- Neurological & respiratory signs - High mortality
3. Beaudett's form/ mesogenic form	- Acute respiratory
4. Hitcher's form/ lentogenic form	- Mild in apparent
5. Asymptomatic enteric form	

Transmission

- Infected birds shed virus in exhaled air, respiratory discharge, and feces. Virus is shed during the incubation period, during the clinical stage, and for a varying but limited period during convalescence.
- Chickens are readily infected by aerosols and by ingesting contaminated water or food.
- movement of infected birds: poultry; wild, exotic, and companion birds; racing and show birds
- movement of poultry products
- movement of people and contaminated equipment or litter (main mode of virus transmission between poultry flocks and farms)

Clinical signs

- ***In velogenic form :***

- ✓ Depression
- ✓ Closed eyes and facial swelling.
- ✓ Drooping wings and anorexia with greenish/yellowish diarrhea.
- ✓ in neural form--- Torticollis, incoordination or even paralysis of legs and arched back position of the body.

- ***In mesogenic form :***

- ✓ Severe respiratory distress.
- ✓ Marked drop in egg production.
- ✓ Some times soft shelled egg or shell less egg.

PM lesions

- petechial hemorrhages on tips of proventriculus glands
- non suppurative encephalomyelitis
- diptheritic ulcers in small intestine
- Caecal tonsils necrosis

Diagnosis

1) By clinical signs

2) By lesion

3) Isolation of virus- From carcasses- the materials to be collected are lung, spleen, trachea bone marrow. In live bird – the materials to be sent are tracheal or cloacal swabs.

- The suspected material is injected into allantoic cavity of 9-11 day chicken embryos. Every day the eggs are candled Those which dies with in 24 hours are discarded and the other which dies after 24 hours will be taken in to account. Hence the embryos are markedly hemorrhagic and stunted. The allantoic fluid is used for Haemagglutination inhibition test to confirm the virus.

Serological test:

1. Haemagglutination test (HAT)

2. Haemagglutination inhibition test (HAIT) commonly used test for the diagnosis of N.D.

3) FAT 4) CFT 5) AGPT 6) ELISA 7) Phage neutalization test.

Vaccination

- ***Live lentogenic vaccines:***

✓ They have F, Hitchner B1, LaSota and V4 vaccines.

✓ Least harmful vaccines(LaSota and B1 most widely used).

✓ LaSota is not used for first vaccination but often as a booster after one or more B1 strain/ F strain vaccines.

- F1 – intraocular or intra nasal at 4-10 days age (1st week)
- F1booster – 5-6 weeks intraocular or drinking water
- ***Live mesogenic vaccines:***

✓ They are Mukteswar (R2b), Roakin, Komarow, and H (Hartford shire).

✓ They are capable of causing severe disease and must be given after earlier vaccination with least harmful vaccines (Live lentogenic vaccines).

✓ Capable of producing a high secondary immune response.

- 8-9 weeks S\C
- inactivated vaccine – 18-19 weeks

Infectious bronchitis

- Gasping Disease
- Highly infectious and contagious respiratory disease of chicks
- IB virus – coronaviridae family
- Different serotypes

  Respiratory tract - massachusetts and connecticut

  Nephrotoxic - T, Gray and Holte

Clinical Signs - Only the chicken is naturally infected. The respiratory form is more severe in young chicks, whereas the nephritic form is mainly seen in birds under 10 weeks of age.

- The virus first attack tracheal cells, multiply and then reaches different organs like kidneys, oviduct etc.

- Respiratory form: The respiratory syndrome is the most common in birds of all ages. Signs include rales, gasping and sneezing, watery nasal discharge, lachrymation, and facial swelling.
- **In young chicks a yellow cheesy plug at the tracheal bifurcation**

- Reproductive form: due to damage to the functional oviduct. This results in reduced egg production and quality.
    - Eggs: smaller than normal, misshapen, lacking symmetry, or show corrugations in outline. shells may be depigmented, have calcareous deposits or the shell may be absent entirely.
    - Internally, the albumen loses its viscosity (thickness and stickiness) ("watery whites"), and the chalazae are often broken so that the yolk floats free.
- Blind layers: may be partial or complete failure of the oviduct to develop, or at maturity the ova are taken up by the malformed oviduct, and are shed into the body cavity

Nephrotic form: Swollen, pale kidneys, with tubules and ureters distended with urates

Diagnosis

- Dwarfing & curling of chick embryo – infected viral material inoculated in allantoic cavity of 9-12 days embryo
- ELISA, FAT, AGID, PCR

Infectious Laryngotracheitis

- Etiology: Herpes virus
- virus appears to infect naturally only the fowl
- affects only the respiratory tract and conjunctiva.
- Fowls of all ages are susceptible.
- greatest susceptibility occurs in the very young chicks (and broilers), the disease is mostly seen in the field in birds from 3-9 months old.
- Transmission by inhalation, ingestion and fomites
- Virus multiplication is limited to respiratory tissues, with no evidence of viraemia.
- trigeminal ganglion is the main site of virus latency
- Infection may result in peracute, acute, mild, or asymptomatic disease
- Acute form: severe dysponea, coughing and respiratory rales with hemorrhagic tracheitis of adult birds
- Subacute/ mild form: lacrimation, tracheitis, laryngitis, conjunctivitis
- Intranuclear inclusion bodies
- POCK lesions on CAM

Avian Influenza

- Fowl plague (Rinderpest - cattle plague, swine fever - swine plague, PPR as goat plague)
- Orthomyxo virus/ type A influenza virus – Highly pathogenic or low pathogenic
- Contains haemagglutinin (HA) and neuraminidase (NA)
- haemagglutinin is responsible for the attachment 9f the virion to cell surface receptors and is responsible for the haemagglutinating activity of the virus. H(1-16)
- Neuraminidase enzyme activity is responsible for the release of new virus from the cell. N(1-9)
- H5N1 & H5N7 - highly pathogenic strains
- all the outbreaks have been of either H5 or H7 sUbtype.
- Host range: chickens, turkeys, ducks, geese, guinea-fowl, pheasants, and quails, and wild birds
- Ducks acts as reservoir and spread virus to chicken and Turkeys
- Infected birds excrete virus from the respiratory tract, conjunctiva, and faeces
- HPAI: sudden onset of high mortality, which may approach 100% within a few days
- cessation of egg laying, respiratory signs including coughing, sneezing, rales, excessive lacrymation; pronounced depression and decreased activity; decreased feed consumption and emaciation; oedema of head and face; cyanosis of unfeathered skin; nervous disorder and diarrhoea.
- LPAI: drop in egg production or complete cessation, respiratory disease, anorexia, depression, sinusitis
- Diagnosis: agglutination of RBCs, CFT, FAT, RT-PCR, Agar gel precipitation test
- H9N2 – used for vaccination

Duck viral hepatitis (DVH)

- an acute, highly infectious viral disease typically affecting ducklings less than six weeks old.
- duck hepatitis A virus (DHAV)-1,2,3of the genus *Avihepatovirus* in the Picornaviridae family.
- most widespread and virulent is DHAV type 1
- Affected ducklings **become lethargic, lose balance, paddle spasmodically, and die within minutes, typically with opisthotonos**.
- Postmortem lesions:

- main lesions are in the liver, which is enlarged and has a number of petechial and ecchymotic haemorrhages.
- In addition, fatty kidneys described as "duck fatty kidney syndrome"

Egg Drop Syndrome

- EDS - major cause of loss of egg production throughout the world.
- caused by an adenovirus belonging to group Ill.
- EDS 1976 is characterized by otherwise healthy birds producing thin-shelled, or shell-less eggs.
- EDS virus is a naturally occurring adenovirus of ducks and geese which has infected fowl.
- Mainly vertical transmission

Infectious Coryza (Fowl Coryza)

- highly contagious disease of the upper respiratory tract of chickens caused by Haemophilus paragallinarum.
- limited to chickens. Chickens of all ages are susceptible, but older birds react more severely.
- main source of infection are diseased and carrier birds. can be spread by drinking water contaminated by nasal discharge, as well as by airborne means over a short distance.
- sero-mucoid nasal and ocular discharge and facial oedema.
- In severe cases marked conjunctivitis with closed eyes, swollen wattles, and dificult breathing can be seen. Feed and water consumption is decreased. This results in a drop in egg production, or an increase in the rate of culling.

Marek's Disease

- Range paralysis/ skin leucosi/Neural leucosis/ neural lymphomatosis/ Lymphoproliferative disease/ fowl paralysis
- Lymphoproliferative disease of chickens caused by herpes virus characterized by a mononuclear infiltration of the peripheral nervous system
- presence of Lymphomas in various visceral organs, nerves, gonads, skin and muscles
- Gallid Herpes virus – 3 serotypes
- Serotype 1 comprises oncogenic strains of MDV.
- Serotype 2 is a group of naturally apathogenic (non-pathogenic) strains of MDV. Serotype 3 is the apathogenic and antigenically related herpesvirus of turkeys (HTV).

- Serotype 1 strains have been subdivided into three pathotypes: 1. Mildly virulent (mMDV), 2. Virulent (vMDV), and 3. Very virulent (vvMDV).

Transmission

- virus is present in desquamated feather follicle epithelial cells, and in oral, nasal, and tracheal secretions.
- The feather follicle cells are the most important source of infection, and are responsible for the infectivity of dander (minute scales from feathers or skin), poultry house dust, and litter. Infectivity in these materials can last for at least one year at room temperature
- Airborne spread of virus and infection through the respiratory tract is considered to be the most important route.
- The virus is not transmitted through the egg, and thus chicks are hatched free of infection.
- Once contracted, the infection persists throughout the life of the chicken, and infected 'birds continue to contaminate the environment by shedding the virus.

Pathogenesis

1. **Early cytolytic infection (productive restrictive phase)**: causing degeneration & infection in B cells
2. **Latent infection**: At about 6-7 days, the infection switches to latency. infection of T cells responsible for neoplastic transformation. The latent infection is persistent, and can last for the life of the bird.
3. **Second phase of cytolytic infection**: permanent immunosuppression. A secondary cytolytic infection occurs in the Feather Follicle Epithelium two weeks after primary infection and infectious cell-free virus is produced and shed into the environment in feather debris and dander.

4. Proliferative phase: infected T cells escape immune system of host and multiply to form lymphoid neoplasms

- Infection of FFE is unique in that it is the only known site of complete virus replication
- MATSA: marek’s disease associated T-surface antigen - host antigen and is not tumour-specific. It is simply a marker for activated T cells.

Clinical Signs

- Marek's disease affects chickens from about 6 weeks of age. It occurs usually between 1 2 and 24 weeks of age, but older chickens m ay aIso be affected. The incubation period may be as short as 3-4 weeks in some, and several months in others.
- Disease forms: Acute MD → 1.5-2.5 months aged

- Classical MD→ 3-5 months age

Classical Marek's Disease

- The signs depend on the peripheral nerves affected.
- Involvement of brachial and sciatic nerves is common, and leads to progressive spastic paralysis (i.e., paralysis accompanied by muscular rigidity) of the wings and legs.
- Sometimes, when the cervical nerves are involved, there may be torticollis (twisting of the neck); and if the vagus and intercostal nerves are affected, respiratory signs may develop.
- Split leg stance- characteristic- Usually unilateral – one leg stretched forward other extended backward
- Mortality: 10-15%

Acute Marek's Disease

- Mortality in this form is usually much higher than in the classical form. Mortality of 10-30% of the flock is common, and outbreaks involving up to 80% of the flock are recorded.
- Many birds die suddenly without preceding symptoms. others appear depressed before death
- Visceral form : lymphoid tumours
- heavy mortality
- enlargement of one or more visceral organs

Transient Paralysis

- This is an uncommon encephalitic manifestation of Marek's disease in birds between 5 and 1 8 weeks of age. Birds suddenly develop varying degrees of paresis (partial paralysis) or paralysis of the legs, wings and neck. Mortality is low. The disease is characterized by recovery, and signs usually disappear within 24-48 hours.

PM lesions

- In classical Marek's disease, the characteristic lesion is enlargement of one or more peripheral nerves. Affected nerves are up to 2-3 times the normal thickness.
- Acute Marek's disease is characterized by diffuse lymphomatous involvement, and enlargement of the liver, gonads, spleen, kidneys, lungs, proventriculus and heart. Sometimes, lymphomas also arise in the skin in association with feather follicles (known as "skin leukosis")
- In the nervous system, peripheral nerves are affected by proliferative, inflammatory or chronic but minor lesions, termed type A, B, and C
- A lesion- infilteration of lymphoblasts and medium to large lymphocytes

- B lesions – proliferation of schwan cells, interneuritic edema, lymphocytes proliferation
- C lesions : lymphoblasts infilteration
- feather follicle epithelium shows I/N inclusion bodies

Diagnosis

- clinical signs, and gross and microscopic lesions
- fluorescent antibody (FA) tests, immunoperoxidase tests, agar-gel precipitation (AGP) tests, and enzyme-linked immunosorbent assays (ELISA). Polymerase chain reaction (PCR)
- Tumor associated markers
- Pock lesions on CAM: Pock lesions are localized areas of necrosis and inflammation that appear on the chorioallantoic membrane (CAM) of embryonated chicken eggs when infected with MDV.

Vaccination

* HVT vaccine at day 1

* Herpes virus of Turkey – HVT vaccine - Leads to production of MATSA

- plaque-forming units (PFU) of Marek's disease vaccines
- CVI988/Rispens Vaccine: A commonly used vaccine, administered at a dose of 4,000 PFU at hatch to provide protection against Marek's disease virus challenges
- General Standard Dose: The standard dose for Marek's disease vaccines is typically around 1,000 PFU per chicken
- Other Vaccines: Some recombinant vaccines are administered at a target dose of 2,500 PFU per bird

Avian Leuksois

- Lymphoid leucosis/ Big liver disease/ Visceral lymphoid leucosis/ Lymphomatosis/ Avian Sarcoma leucosis
- Etiology: avian leukosis/sarcoma viruses (ALSV) of avian type “C" oncoviruses of the Family Retroviridae
- Six subgroups A, B, C, D, and J, on the basis of differences in their viral envelope glycoproteins; E – non oncogenic
- "onc genes", responsible for oncogenic transformation.

- **tumors of hemopoietic tissues and sarcomas, including lymphoid leukosis, myeloid leukosis, and erythroid leukosis, induced by a related group of avian leukosis/sarcoma viruses. Disease usually occurs in chickens aged 16 weeks or older. (around age of sexual maturity)**

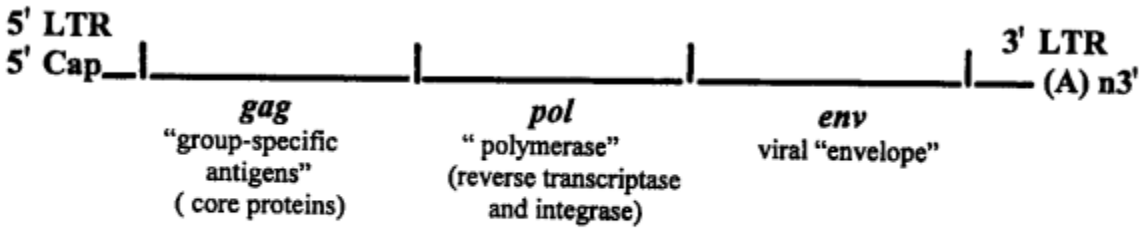


Fig. 3. Diagram showing the gene sequence of the proviral DNA of a standard replicatively competent retrovirus.

Transmission

- Vertically & Horizontally
- Vertical route important as it maintains infection from one generation to another
- Virus produced by albumen secreting glands of oviduct
- Virus-neutralizing antibodies from infected hens are passed through the yolk to chicks, and provide a passive immunity against contact infection which lasts for 3-4 weeks.

Clinical Signs

- Inappetence, weakness, diarrhea, dehydration, and emaciation
- **depressed egg production in the absence of tumor formation is more important economically than are deaths**

Lymphoid leukosis (LL)

- commonest neoplasm caused by the ALSV. It is characterized usually by enlargement of the Liver by infiltrating Lymphoblasts. LL appears between the 14th and 30th week of age. In field outbreaks, cases mostly occur after 14 weeks of age.
- Three lines of evidence indicate that lymphoid leukosis is a malignancy of the bursa-dependent lymphoid system.
- The first is that removal of the bursa prevents LL.
- Necropsies performed on chickens dying with LL have revealed macroscopic tumours of the bursa in almost every case.

- Cells of LL tumours, transplantable tumours, and lymphoid cell lines cultured in vitro all have B cell markers (i.e., they are B-Iymphoytes) and IgM on their surface.
- viral promoter gene a ctivates a host c-myc gene in B cells, and this results in n eoplastic transformation of B cells.
- virus multiplies in Bursa→ cause tumours of B lymphocytes---→ migrates to various organs
- Grossly visible tumours almost always involve liver, spleen, and bursa of Fabricius.

Erythroblastosis (Erythroid Leukosis)

- affecting mainly adult chickens.
- There is always anaemia, which is associated with the presence of a large number of immature red cells in the blood.
- The disease originates in the bone marrow and a leukaemia is present from the outset.
- It is a peculiarity of this disease that the malignant cells remain within the blood vessels throughout the course of the disease.
- This leads to erythrostasis in sinusoids in organs such as the liver, spleen, and bone marrow, giving them a cherry-red colour which characterizes this condition at postmortem. The liver and spleen are moderately enlarged.

Myeloblastosis (Myeloid Leukosis)

- Myeloblastosis involves an extravascular proliferation of cells of the granulocytic series.
- It is a sporadic disease mainly of adult chicks. It may occur as a myeloblastosis, originating in the bone marrow and involving immature cells.
- In diffuse myeloblastosis, the liver and spleen are diffusely and greatly enlarged. The liver usually has a granular appearance.
- Myelocytomatosis, like myeloblastosis, also originates in the bone m arrow, but in this condition the cells affected are more mature.
- have a predilection for the visceral surface of flat bones such as ribs, skull, sternum and pelvis.

Osteopetrosis

- This is a bone disorder affecting mainly the long bones, particularly of the legs and wings. Excessive osteoblast proliferation and bone formation result in gross thickening of the diaphyses of the long bones. Occlusion of the marrow cavity may eventually give rise to anaemia.

Diagnosis

- The presence of infection can be demonstrated easily by detecting neutralizing antibodies in serum.
- The main techniques for ALSV detection are the ELISA test, non-producer (NP) cell activation tests, phenotypic mixing (PM) test,
- **complement fixation avian leukosis virus (COFAL) test**
- resistance-inducing factor (RIF) test

Feature	Marek's disease	Lymphoid leukosis
Susceptible age	6 weeks or older	Not less than 16 weeks
Symptoms	Usually paralysis	Non-specific
Incidence	Usually above 5% in unvaccinated flocks	Rarely above 5 %
Gross lesions		
Neural enlargement	Frequent	Absent
Bursa of Fabricius	Diffuse enlargement or atrophy	Nodular tumours
Tumours in skin, muscle, and proventriculus	May be present	Usually absent
Microscopic lesions		
Neural involvement	Yes	No
Liver tumours	Usually perivascular	Focal or diffuse
Splenic changes	Diffuse	Often focal
Bursa of Fabricius	Interfollicular tumour and/or atrophy of follicles	Intrafollicular tumour
Central nervous system	Yes	No
Lymphoid proliferation in skin and feather follicles	Yes	No

Category of neoplastic lymphoid cell	T cell	B cell

FOWL POX

- Avian pox/ Fowl Diptheria/ Fowl Canker/ Wet pox/ Sore Head
- Caused by Avipox virus –double dtranded DNA virus - largest known virus affecting birds
- single elementary body (Borrel body), i.e., virus particle, separated from the inclusion body (Bollinger body) was capable 0 f causing typical infection
- nodular, proliferative skin lesions on the non-feathered parts of the body (cutaneous form), or fibrino-necrotic and proliferative lesion in the mucous membrane of the upper respiratory tract, mouth, and oesophagus (diphtheritic form).
- Fowl pox virus infects birds of both sexes, and of all ages and breeds.
- first virus to be grown on the chorio-allantoic membrane (CAM) of the embryonated chicken egg. The presence of virus was easily determined by growths, or" pocks" on the membrane.
- The viral particles in these lesions were so large that they could be stained and seen under the light microscope. These particles were called Borrel bodies/granules
- They a re mainly present in a structure in the cytoplasm of the infected cell called a Bollinger body - eosinophilic

Transmission

- Some break in the skin is required for the virus to enter the epithelial cells, replicate, and cause disease. Spread of the virus from one bird to another by direct contact is a major factor in t he spread of disease.
- Cells of the mucosa of the upper respiratory tract and mouth appear to be highly susceptible to the virus.
- biting insects, such as mosquitoes, transmit the disease.

Clinical Signs

- Cutaneous lesions appear on the un feathered skin of the head, neck, legs, and feet.
- Fowl pox can cause a drop in egg production in laying birds.
- The cuatneous lesions can vary in appearance. First, there is a papule, which rapidly progresses through t he vesicle to pustule, and finally to the crust or scab stage.
- After about 2 weeks, the scab drops off, and a healed lesion is left, which may or may not leave a scar.
- mortality is usually low

diphtheritic form ("wet pox") small white nodules are observed in the upper respiratory and digestive tracts. These nodules coalesce (fuse) to form raised yellow plaques on the mucous membranes. Most lesions are found in the mouth, but others are present in the larynx, trachea, and oesophagus

- Mortality as high as 50% has been reported with the diphtheritic form
- Ocular form: cheesy exudates under eyelids causing blindness

PM lesions

- characteristic Lesion of the cutaneous form is a local epithelial hyperplasia involving epidermis and underlying feather follicles, with formation of nodules.
- In the diphtheritic form, slightly elevated, white opaque nodules develop on the mucous membranes.
- yellow, cheesy, necrotic pseudo-diphtheritic or diphtheritic membrane. If t he membranes a re removed, they leave bleeding erosions.
- Characteristic eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies)

Diagnosis

- clinical signs
- Borrel bodies
- Ground-up scabs inoculated onto the chorio-allantoic membrane of 9-12 day-old embryonated eggs produce characteristic pock lesions.
- Inoculation in susceptible chickens
- agar gel precipitation test (AGPT), passive haemagglutination, serum neutralization (SNT), indirect fluorescent antibody, immunoperoxidase, and ELISA. The most sensitive test is the ELISA.

Vaccination

- Where fowlpox is prevalent, chickens and turkeys should be vaccinated with a live-embryo- or cell-culture-propagated virus vaccine.
- The most widely used vaccines are **live, attenuated fowlpox virus** and **pigeonpox virus isolates** of high immunogenicity and low pathogenicity.
- Vaccinated birds should be examined 1 week later for swelling and scab formation (“take”) at the site of vaccination.
- Absence of vaccine take indicates lack of potency of vaccine, passive or acquired immunity, or improper vaccination.

PM lesions

- characteristic Lesion of the cutaneous form is a local epithelial hyperplasia involving epidermis and underlying feather follicles, with formation of nodules.
- In the diphtheritic form, slightly elevated, white opaque nodules develop on the mucous membranes.
- yellow, cheesy, necrotic pseudo-diphtheritic or diphtheritic membrane. If t he membranes a re removed, they leave bleeding erosions.
- Characteristic eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies)

Diagnosis

- clinical signs
- Borrel bodies
- Ground-up scabs inoculated onto the chorio-allantoic membrane of 9-12 day-old embryonated eggs produce characteristic pock lesions.
- Inoculation in susceptible chickens
- agar gel precipitation test (AGPT), passive haemagglutination, serum neutralization (SNT), indirect fluorescent antibody, immunoperoxidase, and ELISA. The most sensitive test is the ELISA.

Vaccination

- Where fowlpox is prevalent, chickens and turkeys should be vaccinated with a live-embryo- or cell-culture-propagated virus vaccine.
- The most widely used vaccines are **live, attenuated fowlpox virus** and **pigeonpox virus isolates** of high immunogenicity and low pathogenicity.
- Vaccinated birds should be examined 1 week later for swelling and scab formation (“take”) at the site of vaccination.
- Absence of vaccine take indicates lack of potency of vaccine, passive or acquired immunity, or improper vaccination.

INFECTIOUS BURSAL DISEASE

- Gumboro disease, Infection bursitis, Infectious Nephritis, Avian Bursitis
- Birna virus : antigenic similarity with Orbi virus and Parvo virus
- host: young chicks 2-7 weeks generally (3-6 weeks specific)

- contagious infection of young chicken targeting Lymphoid tissue
- predilection for bursa leading to B lymphocytes deficiency
- ***Most earliest symptom*** is ***picking of their own vent*** by some birds.
- Vertically not transmitted
- 2 forms immunosuppressive and clinical
- more virulent in layers than broilers
- Spiking death curve: mortality begins on 3rd day post infection, then reaches to peak and recedes in period of 5-7 days

Immunosuppressive form

- Subclinical form accompanied by rapid lymphocytic destruction
- Clinical Form: Pecking of vent
- depression, white diarrhea, soiled vents, anorexia, ruffled feathers, unwillingness to move, trembling, closed eyes, lying down in exhaustion and finally death.

Pathogenesis

- Virus entry → multiplication in bursal follicles, spleen and gut associated lymphoid organs → Nephrosis due to deposition of immune complex in renal glomeruli
- Aplastic anemia, subcutaneous and muscular hemorrhages
- consistent lymphopenia

PM signs

- Ecchymotic haemmorhages in thigh muscles and under skin
- atrophy of bursa
- urate deposits in kidney tubules
- intracytoplasmic inclusion bodies

Vaccination

- *Live mild strain(Lukert type)*
- *Live intermediate(Georgia type)*
- *Inactivated vaccines*: most effective when chickens are primed with live virus vaccines.

Vaccination for commercial layers

- ❖ Lukert strain intraocular and IBD killed @ 0.2 ml s.c at 3 to7 days of age.
- ❖ At 14 to 18 days of age IBD intermediate strain @0.1 ml/ bird in drinking water or intraocular in broilers.
- ❖ These above 2 vaccines are sufficient for broilers.
- ❖ For commercial layers, intermediate vaccine in drinking water is repeated at 28 to 32 days and again at 42 to 46 days of age.

Avian Encephalomyelitis (Epidemic Tremor)

- infectious viral disease of young chickens, turkeys, pheasants, and quail characterized by ataxia and rapid tremors, especially of the head and neck.
- Field strains are mainly enterotropic, whereas egg-adapted strains are mainly neurotropic.
- Horizontal and vertical transmission
- AE usually makes its appearance when chicks are 1-2 week of age.
- dull expression of the eyes followed by a progressive ataxia from incoordination of the muscles, inclination to sit on their hocks.
- Finally, they come to rest, or fall on their sides and Fine tremors of the head and neck

Fowl Cholera

- Avian pasteurellosis/Avian cholera/Avian haemorrhagic septicaemia
- *Pasteurella multocida* subspecies *multocida*
- subspecies *septica* and *gallicida* may be found
- Rats, Cats & Pigs are also a reservoir for *P. multocida*
- *adults birds > 16 weeks age – generally infected*
- capsule is regarded as a major virulence factor of avian P. multocida
- The main site of infection for *P. multocida* is the respiratory tract

Clinical signs

In the acute form:

- Marked depression

- Anorexia
- Mucus discharge from the orifices
- fetid (foul-smelling) Green watery diarrhea
- Blue or purple coloration of skin and swelling of comb and wattles

In chronic infections:

- Arthritis and Otitis
- Abscesses of the head (cranial bones, infraorbital sinuses, subcutaneous tissue, comb and wattles), oviduct
- Torticollis
- Dysponea

Postmortem Findings

- pinpoint hemorrhages on heart, gizzard, proventriculus & intestine
- Enteritis
- Pneumonia
- multiple pinpoint necrotic areas and enlargement in liver

Fowl Typhoid

- Salmonella gallinarum
- infectious enteric disease of adult and layers
- Fowl typhoid is more a disease of adult chickens, with high mortality and morbidity.
- Vertical and lateral transmission occurs
- yellow (sulphur) coloured diarrhoea
- Emaciation, anaemia, pale comb and wattles – in chronic form

PM lesions

- coppery bronze sheen in liver (bronze coloration in liver)
- Fibrin attachment to the surface of heart
- Necrosis of heart- small gray nodular granuloma

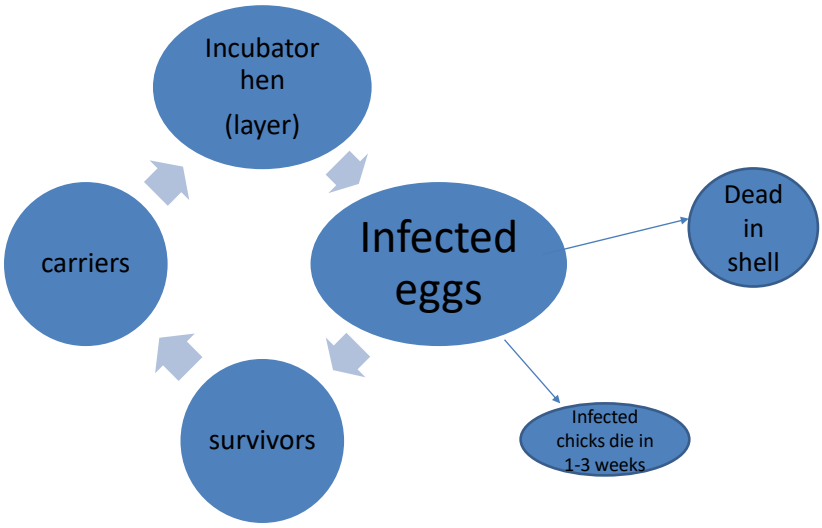
- Vaccine: live attenuated rough Haiftan strain of S.gallinarum

Paratyphoid

- S. heidelberg, S. enteritidis, S. hadar, S. montevideo, S. kentucky,and S. Typhimurium
- Transmission: contaminated feed and various animal and insect vectors.
- PT salmonellae can be spread vertically to the progeny of infected breeder flocks, and horizontally within and between flocks.
- 3 toxins: enterotoxin, cytotoxin, endotoxin associated with lipopolysaccharide
- usually seen in chicks, poults (young turkeys) or ducklings and rarely in birds over 4 weeks of age.
- inflamed, unabsorbed yolk sac, necrotic lesions in the lungs, liver and heart, haemorrhagic enteritis
- characteristic lesion is typhlitis

Pullorum disease

- Bacillary white diarrohea
- Salmonella pullorum
- acute septicemic disease in young chicks and poults
- chronic infection in adult birds
- birds <4 weeks old are most commonly affected



- Acute form:
- huddle near a heat source
- anorectic
- appear weak
- have whitish fecal pasting around the vent (diarrhea)

* Chronic form: synovitis, lameness, inflamed hock joint

Avian Mycoplasmosis

- Chronic Respiratory Disease, CRD, Infectious sinusitis of turkeys
- Mycoplasma gallisepticum (MG) most pathogenic avian mycoplasmosis
- M. synoviae: Chicken and Turkey
- M. melegridis: Tukeys
- M. iowae : chicken and turkey
- Transmission: Horizontal and vertical
- disease of respiratory system characterized by coryza, sneezing, moist rales and open mouth breathing

Post Mortem Lesions

In uncomplicated cases in chickens, the lesions typically include:

1. Mild sinusitis

2. Tracheitis

3. Airsacculitis

If chicken is infected concurrently with E. coli:

1. Thickening and turbidity of the air sacs

2. Exudative accumulations

3. Fibrinopurulent pericarditis

4. Perihepatitis

Infectious Synovitis

- M. synoviae
- exudative tendinitis and Synovitis, subclinical infection of URT
- transmission : horizontal and vertical
- common in chickens 4-6 weeks and Turkey 10-12 weeks
- swelling of hocks and foot pad

Avian spirochetosis

- An acute bacterial infection transmitted by tick
- *Borrelia anserina*
- transmitted by tick *Argas persicus*
- *intestinal spirochetosis and blood spirochetosis*
- highly variable and nonspecific clinical signs
- enlarged and mottled spleen

Avian Chlamydiosis (Psittacosis, Ornithosis, Parrot fever)

- caused by Chlamydia psittaci infectious form (elementary body)
- Among domestic poultry, turkeys are most susceptible and then ducks and pigeons, while chickens are rarely affected.
- Elementary bodies are found in faeces and also in respiratory excretions. Spread occurs mainly through the inhalation of infected, contaminated dust.
- depression with ruffled feathers, anorexia, purulent nasal discharge and conjunctivitis, sometimes tracheitis with rales, and grey-green diarrhoea which may contain blood
- epithelium of the kidney may be packed with large numbers of "LCL bodies". These are minute, spherical basophilic bodies (discovered by Levinthal, Coles, and Lillie)

Avian Coli bacillosis

- E. coli is a normal inhabitant of the digestive tract of mammals and birds, and most strains are non-pathogenic.
- Certain serotypes, however, can cause disease in poultry.
- Colisepticaemia, egg peritonitis, yolk sac infection, and coligranuloma (Hjarre's disease) - colibacillosis"

- Colisepticaemia The disease is usually seen in young chickens. It is the most serious form of colibacillosis.
- Birds between 4 and 12 weeks of age are usually affected,
- first sign is a drop in food consumption. The birds then a ppear I istless, stand dejectedly with ruffled feathers, develop laboured breathing, and a characteristic "snicking" (i.e., making short sharp sound).
- Lesions: airsacculitis, peritonitis, perihepatitis, and pericarditis.
- fibrinous pericarditis, with the pericardial sac thickened, white and adhering to the surface of the heart, is a characteristic finding. The surface of the liver is usually covered by a thin layer of fibrinous material.
- “Egg peritonitis“ covers a number of reproductive disorders of poultry. These include peritonitis, salpingitis (inflammation of oviduct), and impaction of the oviduct.
- Yolk Sac Infection /"mushy chick disease" and "omphalitis", - probably the commonest cause of mortality in chicks during the first week after hatching.
- E. coli can be involved either as the primary and sole causal agent, or as a secondary opportunist.
- Yolk sac infection can be associated with an inflamed navel, or bacteria can multiply in the hatching egg following faecal contamination of the shell.
- The main finding is an inflamed unabsorbed yolk sac, yolk being abnormal in colour and consistency.

Aspergillosis

- "brooder pneumonia" or "mycotic pneumonia“
- saprophytic moulds - Aspergillus fumigatus and A. flavus
- extremely common in nature, occurring on foodstuffs as a white, fluffy, sporulating mould
- Ordinarily they are not pathogens, but rather opportunists.
- primary target of the agent is the respiratory system, other manifestations of the disease also occur in poultry
- Newly hatched chickens, turkeys, and ducks are highly susceptible to infection.
- Stress of cold, high ammonia and dusty environments increase the incidence and severity of infection
- Aspergillus sp. also produce toxins, known as aflatoxins, which cause mycotoxicosis (aflatoxicosis)

- Infection is by inhalation of spores
- Within the first 3-5 days chicks infected in the hatchery show dyspnoea, polypnoea (very rapid breathing), and begin open-mouthed breathing (gasping, 'gaspers') due to airway obstruction.
- birds which survive become lethargic and stunted, develop conjunctival swelling, blindness, and exhibit torticollis and other central nervous system abnormalities
- biphasic mortality pattern
- Lesions: granulomas appear as separate 1-15 mm diameter white plaques or caseous nodules in lungs and air sacs

Thrush (Crop Mycosis, Candidiasis)

- Also known as "moniliasis", "oidiomycosis", "sour crop", and "mycosis of the digestive tract“
- caused by Candida albicans
- C. albicans is ubiquitous in the environment, and is usually present in the upper gastrointestinal tract of normal birds. Candidial overgrowth occurs in prolonged administration of antibiotics.
- Birds with superficial oral, oesophageal or crop infections fail to gain weight.
- Diagnosis: Pseudomembranes and diphtheritic membranes in the crop, oesophagus and mouth (Turkish Towel Appearance)

Favus

- usually caused by Trichophyton megninii (T. gallinae)
- Lesions appear in unfeathered skin (comb, wattle, shank) by superficial invasion of the stratum corneum by hyphae.

Aflatoxicosis

- Aflatoxins are highly toxic and carcinogenic metabolites produced by the fungi Aspergillus flavus and A. parasiticus
- B1, B2, G1, G2 and M1
- Young poultry are more sensitive to aflatoxin than are adults.
- Ducks being 10 times more sensitive than chickens, and turkeys intermediate between the two.
- Pathogenesis: After ingestion, aflatoxin undergoes biotransformation into numerous highly reactive metabolites having a variety of negative effects on metabolism.
- Metabolites bind to DNA and RNA, reduce protein synthesis, and decrease c ell-mediated immunity, and to a lesser extent humoral immunity.

- These metabolic alterations Iead to liver, kidney and spleen enlargement, and bursal, thymic and testicular atrophy.
- yellow discoloration of the liver, with multifocal haemorrhage.
- Anemia
- marked immunosuppression
- aflatoxin blocks ova maturation and reduces feed efficiency and egg production.
- Affected adult hens have decreased egg production, and the hatchability of eggs is reduced

Histomoniasis

- Also known as "blackhead", "infectious enterohepatitis", histoplasmosis is a disease of caeca and liver caused by the protozoan parasite Histomonas meleagridis.
- it is primarily a disease of turkeys. The turkey is considered the most susceptible host
- The existence of H. meleagridis is closely associated with caecal nematode Heterakis gallinarum and several species of earthworms common in the soil of poultry premises.
- Ulceration of the caecal wall may lead to perforation of the organ and cause generalized peritonitis. Liver lesions in turkeys appear as circular depressed areas of necrosis up to 1 cm in diameter, and are surrounded by a raised ring.

Helminthic Diseases of Poultry

Table 53. Some important tapeworms of poultry

Tapeworm	Main final host	Intermediate host
Amoebotaenia cuneata	Chicken	Earthworms
Choanotaenia infundibulum	Chicken	Housefly, beetles
Davainea proglottina	Chicken	Slugs, snails
Hymenolepis cantaniana	Chicken	Beetles
Hymenolepis carioca	Chicken	Stable fly, dung beetles
Raillietina cesticillus	Chicken	Beetles
Raillietina echinobothrida	Chicken	Ants
Raillietina tetragona	Chicken	Ants

Table 54. Some important nematodes of poultry

Nematode	Location	Intermediate host
Ascaridia galli	Small intestine	None
Capillaria anatis	Small intestine, caecum, cloaca	None
C. annulata	Oesophagus, crop	Earthworm
C. bursata	Small intestine	Earthworm
C. caudinflata	Small intestine	Earthworm
C. contorta	Mouth, oesophagus, crop	None or earthworm
C. obsignata	Small intestine, caecum	None
Heterakis gallinarum	Caecum	None
Syngamus trachea	Trachea	None
Trichostrongylus tenuis	Caecum	None

Coccidiosis

- protozoan disease of poultry caused by various species of Eimeria and is characterized by sudden onset, diarrhoea, dysentery, weakness and mild to very heavy mortality rate.
- The bird after exposure to one species will be immune to that particular species, thus indicating that there is no cross protection.
- The birds exposed to Marek's disease virus will be highly susceptible to coccidiosis because such birds will be in the state of immuno-suppression because T-cells are affected.

Species	Site of development	Pathogenicity	Disease type
E. necatrix	**Jejunum, ileum, caeca**	+++++	**Hemorrhagic**
E. tenella	**Caeca**	+++++	**Hemorrhagic**
E. brunetti	**Caeca and rectum**	++++	**Hemorrhagic**

E. maxima	**Jejunum, ileum**	**+++**	**Malabsorptiv**
E. mitis	**Ileum**	**++**	**Malabsorptiv**
E. acervulina	**Duodenum, ileum**	**++**	**Malabsorptiv**
E. praecox	**Duodenum, jejunum**	**+**	**Malabsorptiv**

- Any age group can be affected but commonly occurs in a severe form in young ones. It causes severe economic loss which may be due to:
  - Mortality rate which may vary from 5-20%.
  - Reduction in gain in weight which is commonly seen in sub- clinical form of coccidiosis wherein feed conversion rate is decreased.

Coccidiosis is largely a disease of young birds because immunity is low and it quickly develops after exposure and gives protection against later disease outbreaks. However, birds can be infected at any age if not exposed earlier. Chickens get the infection by eating oocysts once these are sporulated after being shed in the droppings of infected birds.

- **Transmission:** is by the ingestion of feed and water containing the sporulated oocysts.

Pathogenesis

- depends on the following factors :
  - number of sporulated oocysts ingested by the bird.
  - The species of Eimeria involved.
  - Nutritional status of the bird.
  - Management practices.
  - Concurrent diseases.

Clinical findings

- Sudden onset, diarrhoea, dysentery, where in the droppings may have blood clots or fresh blood or sometimes only blood. Weakness, depression, huddling together of the birds and anaemic signs

like pale combs and wattles and conjunctiva, increase in heart and respiratory rates will be seen. Finally it will end in death of the bird.

- In sub-clinical form, the symptoms are not seen and only the loss in gain in weight can be seen.

Caecal coccidiosis – E. tenella

Intestinal coccidiosis – E. necatrix

Rectal coccidiosis - by *E brunette*

E maxima develops in the small intestine

Diagnosis: Diagnosis in majority of the cases is based upon examination of faecal samples for oocyst findings and typical post-mortem lesions.

Treatment	Example	Mechanism of Action
Ionophores	**Lasalocid, Monensin, Narasinm Salinomycin, and Semduramicin**	**Disruption of ion gradient across the parasite cell membrane**
Chemicals	**Quinolone drugs (Decoquinate and nequinatem buquinolate) Pyridones (Meticlorpindol)**	**Inhibition of parasite mitochondrial respiration**
	Sulphonamides	**Inhibition of the folic acid pathway**
	Amprolium Diclazuril,	**Competitive inhibition of thiamine uptake**
	Halofuginone, and Robenidine	**Mode of action unknown**
	Nicarbazin	**Inhibition of the development of the first and second generations of the schizont stage of the parasites**

Veterinary Jurisprudence

COMMON OFFENCES AGAINST ANIMALS AND THE RELATED LAWS

- Government of India legislated an exclusive law to prevent cruelty against animals in the year 1960.
- Prevention of Cruelty to animals act- 1960
- mentions three offences against animals which are mischief, cruelty, and bestiality.

MISCHIEF

- killing, poisoning, and maiming of an animal.
- Poisoning is the most common method of mischievous killing of animals.
- Mischief is punishable under Section 428 and 429 of Indian Penal Code (IPC) Act
- Section 428 of the IPC Act speaks about mischief by killing, poisoning, maiming, or rendering useless any animal or animals of the value of rupees ten or upwards. These offences shall be punishable with imprisonment for a period of up to two years or with a fine or both.
- Section 429 of the IPC Act speaks about mischief by killing, poisoning, maiming, or rendering useless any elephant, camel, horse, mule, buffalo, bull, cow or ox of any value or any other animal of the value of rupees fifty or upwards. These offences shall be punishable with imprisonment for a period of up to five years or with a fine or both.
- Maiming refers to causing injury or wound so that the affected part of the body remains permanently damaged.
- commonly adopted maiming practices are
    - cutting the tendons of legs and neck
    - injuring udder in milch animals
    - breaking of bones
    - injuring with sharp objects etc

CRUELTY

- Cruelty is punishable under Section 11 and Section 12 of the Prevention of Cruelty to Animals (PCA) Act, 1960.
- Section 11 of the PCA Act speaks about kicks, beats, over-drives, over-loads, tortures, or any treatment that cause unnecessary pain or suffering to the animal.

- Activities that do not fall under the category of cruelty to animals are dehorning, castration, euthanasia of stray dogs or other animals which are terminally ill, killing of animals as food for mankind, experimentation on animals for new discovery etc.
- Section 12 of the PCA Act provides for penalty for those who practice phooka or doom dev or injecting substances to improve milk production which is injurious to the health of the animal.
- The penalty includes a fine or imprisonment for up to two years or both.

BESTIALITY/ Zoophilia

- **BESTIALITY :- Means carnal intercourse with man, woman or animal against the order of nature**
- bestiality is punishable under Section 377 of the IPC
- Section 377 of the IPC Act prohibits all unnatural offences. Voluntary carnal intercourse against the order of nature with any man, woman or animal
- punishable with a fine or imprisonment for a term of up to ten years or imprisonment for life.
- The presence of organisms of gonorrhoea (bean-shaped, gram negative diplococci) in the vagina of the animal is a definite sign of bestiality
- **Examination of seminal stains may be carried out by the following method**
- **Physical Examination :-** Average volume of ejaculate, average concentration (million per ml), pH, colour, consistency.
- **Chemical Examination :-** (1) Florence's Test and (2) Barberio's Test
- **Microscopic Examination**
- **Serological Examination**:- (precipitation test)

FRAUDS IN THE SALE OF LIVESTOCK

- Alternation of description
- The description is altered by: -

(i) Castration of an entire

(ii) Clipping the mane and tail of horses

(iii) Docking

(iv) Colouring of white patches by hair dye

(v) Bishoping: This is done to make the horse appear younger - filing down the Galvayne's Groove or changing the shape or length of the teeth, or using silver nitrate to artificially create cups.

* Frauds in the sale of livestock are punishable under Section 420 of IPC Act which deals with “cheating and dishonestly inducing delivery of property”.

FRAUDS IN THE SALE OF LIVESTOCK PRODUCTS

- Reduction of fat, addition of thickening agents, addition of preservatives, urea or coloring material
- Adulteration of milk and milk products is punishable under IPC Section 272. This Section speaks about ‘Adulteration of food or drink intended for sale’.
- Section 273 – sale of such products
- Whoever adulterates any article of food or drink, or involves in any such activities, or sells such articles shall be punished with imprisonment up to 6 months or a fine or both.

LIVESTOCK INSURANCE

- Government of India first launched livestock insurance in the year 1970.
- Premium rates generally vary from 4% to 6% per annum.

Offences

- Cognisable offence: An offence listed in the first Schedule of the CrPC for which the culprit is liable for a death sentence or imprisonment for life or for a term exceeding 2 years is referred to as a cognisable offence. The police can arrest the accused without a warrant in cognisable offences. Eg. Hurting a man or an animal with a weapon.
- Non-cognisable offence: Offences punishable with imprisonment for a term less than 2 years. Police cannot arrest an accused person without a warrant.
- Summons or Subpoena: It is a document compelling a witness to appear before a Court of law on a particular day and time to give evidence.
- Inquest: Inquest is an inquiry into the cause of death. It is conducted in all unnatural deaths. The two types of inquests are the police inquest and the Magistrate’s inquest.

Supreme Court – Highest Judicial tribunal & have power to award any punishment as per law

		including death sentence
2	The High Court	Has powers to award any sentence authorised by law including death sentence
3	Principal Sessions Court/ Additional Sessions Court	Can award any sentence authorised by law including death sentence. Death sentence should be confirmed by the High Court.
4	Assistant Sessions Court	Up to 10 years imprisonment. Fine is unlimited.
5	Chief Judicial Magistrate/ Chief Metropolitan Magistrate	Up to 7-year imprisonment. Fine is unlimited.
6	Judicial First Class Magistrate	Up to 3-year imprisonment. Fine up to Rs.10,000/-
7	Judicial Second Class Magistrate	Up to 1-year imprisonment. Fine up to Rs.5000/-

- Documentary evidence include all documents including electronic reports produced for the inspection of the Court. Documentary evidences (in the context of vetero-legal cases) include veterinary certificates and vetero-legal reports.
- Veterinary certificates include certificate of health or illness, treatment certificate, insurance certificate, death certificate etc. issued by a registered veterinarian.
- Vetero-legal reports include post-mortem reports, wound certificate etc.
- Issuing or signing a false certificate is punishable under Section 197 of IPC Act, 1860.
- It is punishable with a fine and imprisonment up to seven years, and erasure of name from the register of Veterinary Council of India.

ANIMAL WELFARE LAWS, RULES AND REGULATIONS

- The Prevention of Cruelty to Animals Act, 1960 is the most important law related to animal welfare
- The Prevention of Cruelty to Draught and Pack Animals Rules, 1965
- The Performing Animals (Registration) Rules, 2001.
- The Transport of Animals, Rules, 1978.

- The Prevention of Cruelty to animals (Transportation of animals on foot), Rules, 2001.
- The Prevention of Cruelty to Animals (Dog Breeding and Marketing) Rules, 2017
- The Prevention of Cruelty to Animals (Pet Shop) Rules, 2018.

Animal Welfare Board of India

Establishment of Animal Welfare Board of India

- Established in 1962 under Section 4 of the Prevention of Cruelty to Animals Act, 1960 (No. 59 of 1960),
- started under the stewardship of Late Smt. Rukmini Devi Arundale
- Headquarter at Ballabgargh, Faridabad
- implementation of the provisions of the Prevention of Cruelty to Animals Act, 1960.
- Two statutory organizations viz. Animal Welfare Board of India (AWBI) and committee for the Purpose of Supervision and Control of Experiments on Animals (CPCSEA)

Constitution of a committee for control and supervision of experiments on animals (CCSEA)

- Section 15, Chapter IV (Experiments on animals) of the PCA Act, 1960
- take all such measures as may be necessary to ensure that the animals are not subjected to unnecessary pain or suffering, before, during or after the performance of experiments on them.
- The CCSEA will : Register institutions/establishments/breeders carrying out experiments on animals/breeding animals.
- Ensure that the experiments are carried out by qualified individuals and with full responsibility of the person in charge of the institution.
- Monitor and inspect the housing of animals of breeders/establishments and ensure that it is as per specified standards.
- Participate in the meetings of the Institutional Animal Ethics Committees
- principles of 3 R that is to REDUCE, REDEFINE, and REPLACE the use of animals in experiments.

LAWS RELATED TO PREVENTION AND CONTROL OF DISEASES AND OFFENCES AFFECTING PUBLIC HEALTH

- Prevention and Control of Infectious and Contagious Diseases in Animals Act, 2009
- There are seven Chapters in this Act with 45 Sections.

(1) Chapter I: Preliminary

(2) Chapter II: Control of Scheduled diseases

(3) Chapter III: Infected areas

(4) Chapter IV: Infected animals

(5) Chapter V: Enforcement and penalties

(6) Chapter VI: Precautionary measures on causative organism

(7) Chapter VII: Miscellaneous

LAWS RELATED TO POISONS AND ADULTERATION OF DRUGS

- The laws in force related to poisons/ drugs in India are

(i) the Poisons Act, 1919

(ii) The Dangerous Drugs Act, 1930

(iii) the Drugs and Cosmetics Act, 1940.

Of these the Dangerous Drugs Act, 1930 regulates the traffic and misuse of dangerous drugs like opium (Papaver somniferum), Indian hemp (Cannabis sativa), cocoa leaves and their derivatives.

Punishments under the IPC Act, 1860: -

Section 274: Adulteration of drugs Section

275: Sale of adulterated drugs

SOCIETIES FOR PREVENTION OF CRUELTY TO ANIMALS

- The Societies for Prevention of Cruelty to animals (SPCAs) are established as per the PCA (Establishment and Regulation of Societies for Prevention and Cruelty to Animals) Rules, 2001 (under PCA Act, 1960).

Wildlife Institute of India: WII was established in 1982, as an autonomous institute under the Ministry of Environment, Forest, and Climate Change.

LIVESTOCK IMPORTATION ACT, 1898

Glanders and Farcy act, 1899

Dourine act, 1910

Disaster Management Act, 2005.

PostMortem examination of veterolegal cases

- The objective of the postmortem examination of an animal is to ascertain the time since death and the cause of death.
- It includes systemic examination of the dead animal, recording of gross pathological lesions, laboratory examination (histopathology, microbiology, parasitology, and toxicology) of the samples collected and their correlation with the history to make a diagnosis of the disease.

Rules:

- To be done by written order from the police officer or the Executive Magistrate.
- Carefully read the police report first.
- Examination to be done in day light. (Before sunset / after sunrise).
- Examination to be thorough and complete.
- All the details to be noted in the PM reports on the spot of the PM examination.
- The notes and the report to be sent to the Court must tally with each other (Police and owner).
- Should have fair knowledge of the normal and pathological appearance of viscera.
- Time and date of arrival of carcass to be noted.
- No unnecessary delay in conducting PM.
- No unauthorised person should be allowed to be present at the time of PM except investigating police officer

Procedure for PM

- External Examination

  1) Note the general condition of the carcass.

  2) Note the species, age, breed and sex of the animal.

  3) Examine the body surface for injuries, burns, swellings, etc.

  4) Look for the discharge from the natural orifices.

  5) Examine the visible mucous membranes.

  6) Examine the external genitalia.

  7) All the bones should be carefully examined for presence of fracture and joints for dislocation

- Internal Examination:

- Necropsy
- Examination of Organs
- Submission of specimens in case of Poisoning
- Time of Death: Determination of time of death is important in vetero-legal cases. Usually it is available from the owner or the clinician.
- Rigor Mortis: • Rigor mortis is the stiffening of muscles after death.
- The time of onset of rigor mortis and its disappearance give an approximate time of death. Generally, it lasts for 24 hrs.
- However, it is quite variable and is best related to body temperature, metabolic activity at the time of death and atmospheric temperature at the place of death.
- Stiffening of head muscles (jaw, eye lids)- occurs within 2-8 hrs. In summer, it occurs within 30 minutes to 3 hours
- Stiffening of head, neck, and distal fore limb muscles- Occurs within 12 hrs
- Stiffening of large muscles of hind limb and disappearance of rigor in other muscles - Within 20 hrs
- Disappearance of rigor mortis from the whole body - 24-30 hrs
- Carcass bloated with putrid foul smell- More than 30 hrs

PM changes

- Algor Mortis: cooling of the carcass after death.
- Livor mortis: staining of tissues with haemoglobin after death of animals. It gives a pinkish discolouration to the tissues
- Hypostatic congestion: Due to gravitational pull, blood accumulates in the dependent parts of body after death.
- Post-mortem emphysema: It is the post-mortem swelling of the body due to decomposition, by gas producing organisms including saprophytes.
- Post-mortem clot: It is clotting of blood after death mainly due to excessive release of thrombokinase from dying leucocytes and endothelial cells.
- Adipocere: Adipocere is a wax-like organic matrix formed by the anaerobic bacterial decomposition of body fat.

Wounds

- The vetero-legal wounds may be classified as-
    - Bruises (Contusions)
    - Abrasions
    - Incised wounds
    - Lacerated wounds
    - Punctured wounds
    - Gunshot wounds

BRUISES or CONTUSIONS

- Bruises or contusions are injuries which are caused by a blow from a blunt weapon

Age: Age of the Bruise is determined by the colour change on the skin

These colour change start from periphery and ends to the centre

The colour is red and turns blue or bluish black with in 3 days, become greenish on 5^{th} -6^{th} day and yellow from 7-12^{th} day

ABRASIONS

- Abrasions are injuries involving loss of the superficial epithelial layer of the skin Produced by a blow or a fall on a rough surface.
- Abrasions caused during life appear as bleeding surface and soon converted into reddish brown crust or scab
- Abrasions produced after death are dark brown and there is complete absence of bleeding

Incised wound

- It is produced by sharp cutting instruments
- It is mostly intentional

Character: Always broader than the edge of the weapon

It is some what spindle shaped with gapping

Its edges are smooth, even, clean cut, well

defined and usually everted

Bleeding is intensive

Lacerated wound

- These wound do not corresponds in shape or size to the object producing them. Their edges are torn, jagged, irregular and swollen or contused

Punctured wound

- They are popularly called stab and are termed as penetrating wound.
- Character: The wound is wedged shaped
- The depth of a punctured wound is much larger than its length or width
- There may be little external haemorrhage, but profuse internal haemorrhage.

Firearm wounds/Gunshot wounds

- The appearance of these wounds depend upon
    - ✓ The kind of the weapon employed
    - ✓ The nature of projectile
    - ✓ The velocity of the projectile
    - ✓ The range at which the weapon was fired
    - ✓ The part of body struck

Burns and Scalds

Burns

- Injury produced by flame, heat or some heated solid substance like metal or glass
- Lighting
- Electricity
- Xrays
- Corrosive substances

Scalds

- Moist heat injuries
- Injury produced by steaming liquid at or near its boiling point or in its gaseous form

Classification of Burns

- First degree: erythema or simple redness

- Second degree: inflammation and formation of vesicles
- Third degree: destruction of cuticle and skin. Nerve endings exposed resulting in severe pain
- Fourth degree: destruction of whole skin and nerve endings
- Fifth degree: penetration of deep fascia and muscles resulting in great scarring and deformity
- Sixth degree: whole limb, inflammation of adjacent tissues and organs
- Causing of death: Shock, Suffocation or accidental injuries

Lightning: immediate death due to paralysing effect on nervous system or in later stages due to burn and lacerations

Electricity: AC more dangerous than DC due to high voltage

- Cause of death is sudden stoppage of heart or from inhibition of respiratory centre in brain stem

WELFARE OF ANIMALS DURING TRANSPORTATION

- Different classes of animals shall be kept separately during transport.
- Diseased animals, whenever transported for treatment, shall not be mixed with other animals
- Troublesome animals shall be given tranquilizers before loading during transport
- only 6 large animals in a lorry can be transported. Any violation of these laws is termed illegal.
- The permissible loading in a truck is only 4 buffaloes or 40 sheep/goats, and shall not carry more than five cattle without calves or four with calves.
- In the case of trucks whose wheel base is over 142 inches shall not carry more than six cattle without calves or five with calves.
- Animals in tempo are not allowed.
- Transport of Animals, Rules, 1978.
- Transport of Animals (Amendment) Rules, 2009.

TRANSPORT OF CATTLE

- When cattle is to be transported by rail an ordinary goods wagon shall carry not more than ten adult cattle or fifteen calves on broad gauge
- Not more than six adult cattle or ten calves on meter gauge

- Not more than four adult cattle or six calves on narrow gauge.
- While transporting cattle by goods vehicle, only six cattle should be loaded.

TRANSPORT OF EQUINES

- By rail, ordinary goods wagon shall carry not more than eight to ten horses or ten mules or ten donkeys on broad gauge
- Not more than six horses or eight donkeys on meter- gauge.
- If equines are to be transported by good-vehicles each vehicle should not carry more than four to six equines.
- For the transport of equines by sea, horses may normally be accommodated in single stalls and mules in pens, each pen holding four to five mules.

TRANSPORT OF SHEEP AND GOATS

Broad Guage

- If the area of wagon is less than 21.1 Square Meters number of sheep or goat allowed is 70

Meter Guage

- If the area of wagon is 21.11 square Meters and above number of sheep or goat allowed is 100

Narrow Guage

- If the area of Wagon less than 12.5 Square Meters the number of sheep or goat allowed is 50
- If the area of wagon is 12.5 Square Meters and above the number of sheep or goat allowed is 60

Goods vehicle of capacity of 5 or 4.5 tons, which are generally used for transporting animals, shall not carry more than forty sheep or goats.

TRANSPORT OF POULTRY BY RAIL, ROAD AND AIR

Kind of Poultry	**Number in a standard container**
Month old chickens	24
Three-month old chickens	12

Adult stock(excluding geese and turkeys)	12
Geese and turkeys	10 youngs, 2 growings, 1 grown up
Chicks	80
Poult	60

TRANSPORT OF PIGS BY RAIL OR ROAD

- By road, good vehicles shall not carry more than 20 pigs.

Broad Gauge

- If the area of wagon is less than 21.1 Square Meters number of pigs allowed is 35

Meter Gauge

- If the area of wagon is more than 21.1 Square Meters number of pigs allowed is 50

Narrow Gauge

- If the area of wagon is less than 12.5 Square Meter number of pigs allowed is 25
- If the area of wagon is more than 12.5 Square Meter the number of pigs allowed is 30

Overloading of animals treated as animals cruelty under Section 11 of The Prevention Of Cruelty to Animals Act,1960.

What is the maximum load of certain pack animals?

1) Small bullock or buffalo	**100 kilograms**
2) Medium bullock or buffalo	150 kilograms

3) Large bullock or buffalo	175 kilograms
4) Pony	70 kilograms
5) Mule	200 kilograms
6) Donkey	50 kilograms
7) Camel	250 kilograms

General conditions for the use of draught and pack animals

Section 6 of The Prevention of Cruelty to Draught and Pack Animals Rules, 1965, lays down the general conditions for the use of draught and pack animals. No person is allowed to use any animal for drawing any vehicle or carrying any load :

- For more than nine hours in a day in the aggregate.
- For more than five hours continuously without a break or rest for the animal.
- In any area where the temperature exceeds 37 degree C during the period between 12 noon and 3 p.m.