DISEASES OF RESPIRATORY SYSTEM

General Medicine

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Prepared by Prof. S . Haque, Ph.D

Dept of Veterinary Clinical Medicine

College of Veterinary Medicine and Animal Sciences(UOG)

DISEASES OF RESPIRATORY SYSTEM

- The **respiratory system** (or **ventilatory system**) is a biological system consisting of specific organs and structures used for the process of respiration .
- Respiratory system is involved in intake and exchange of oxygen and carbon dioxide between an living animal and the environment.
- Inspiration and expiration collectively known as breathing or ventilation.
- The exchange of gasses occurs in the alveoli air sacs in the lungs.
- Mostly in animals respiratory movements are **thoracoa - bdominal.**
- In horses through nostrils as they are nasal breather.

DISEASES OF RESPIRATORY SYSTEM

- Respiratory system is divided into two parts:

  1. Upper Respratory tract --

     It consists of the nose, nasal sinuses, throat and trachea or windpipe .

  2. lower respiratory tract –

     It consists of the 'small airways' (bronchi and bronchioles) and the alveoli (the small air sacs deep in the lung tissue where oxygen exchange occurs).

DISEASES OF RESPIRATORY SYSTEM

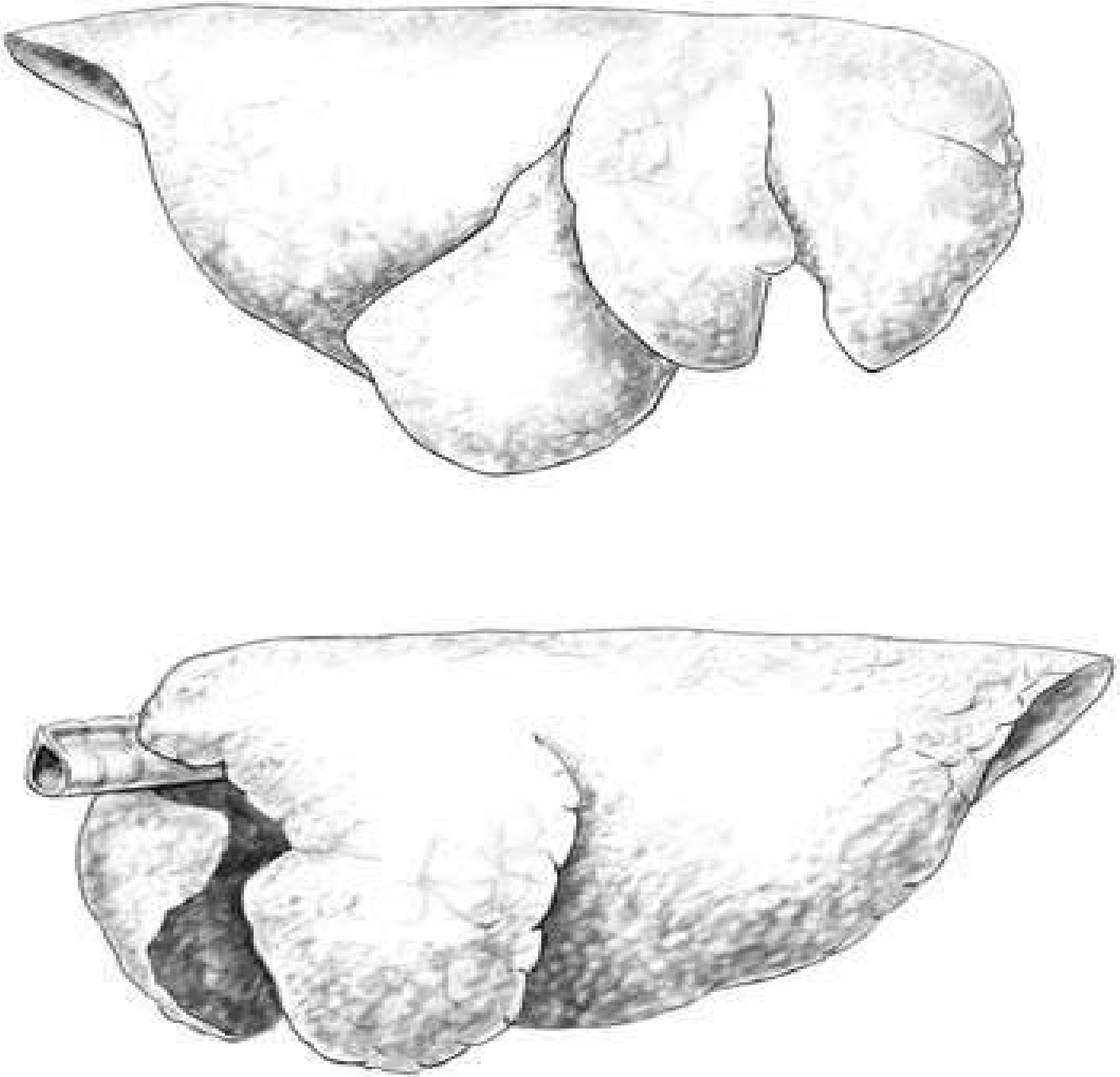
- The lower airway is composed of the trachea and the right and left lungs.

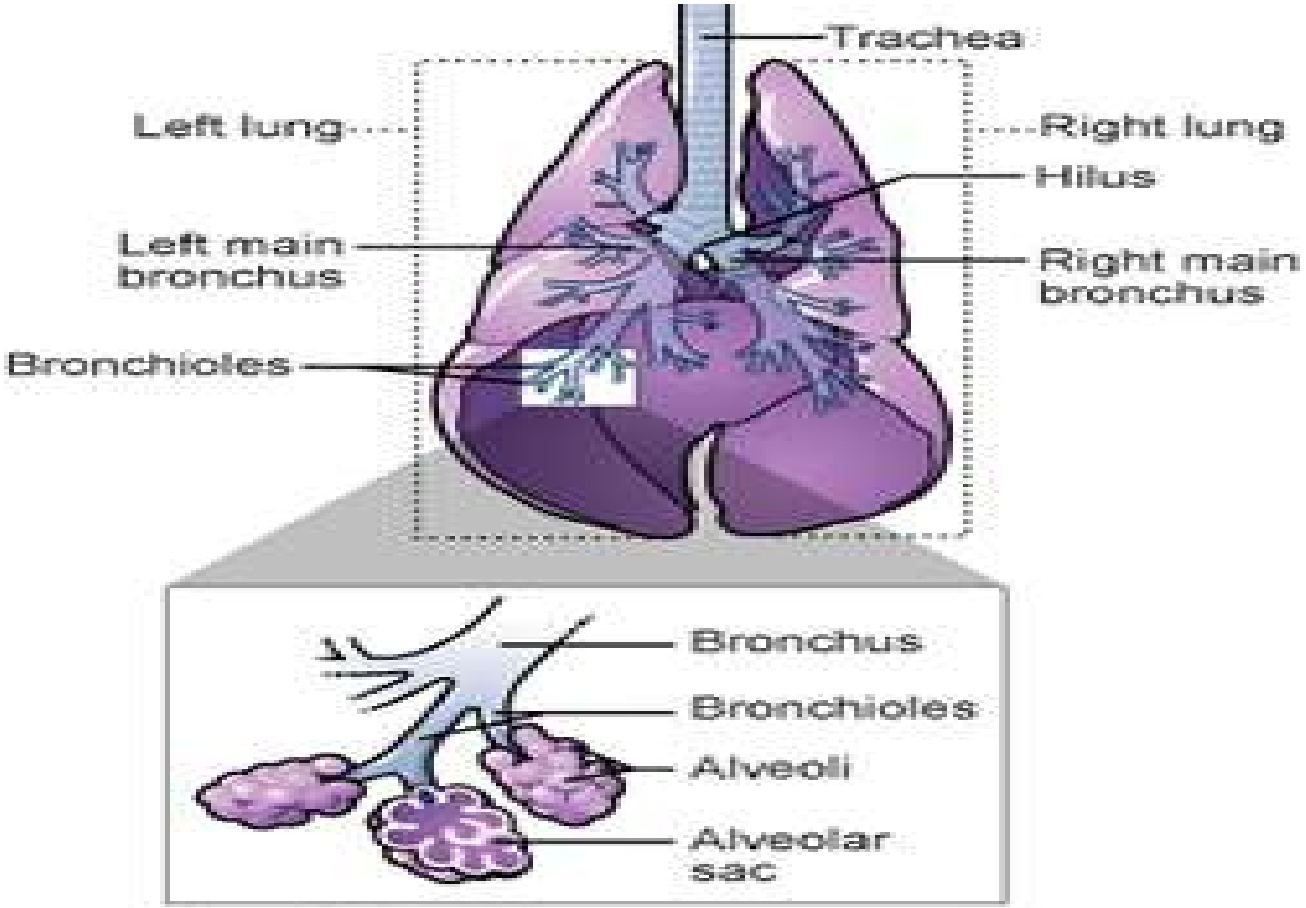
The *left lung is composed of three lobes:*

- *The apical lobe*(cranial) .
- The cardiac lobe (middle).
- The diaphragmatic lobe (caudal).

The *right lung is composed* of four lobes:

- The apical lobe .
- The intermediate lobe .
- The cardiac (middle) lobe.
- The diaphragmatic lobe.





PNEUMONIA

Definition:

1. Pneumonia is inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleuritis.

2. Pneumonia is inflammation of the lungs.

Inflammation is the body's response to damage.

When lung cells are damaged, due to any cause, lungs become inflamed, and the animal has pneumonia.

- Lungs is like sponges that inflate and deflate as an animal breathes.
- Like sponges, lungs are composed of tiny, thin-walled sacs (alveoli).
- The sacs (alveoli)contain air that is replaced with every breath.
- The walls of the sacs contain small blood vessels.

PNEUMONIA

- The extremely thin walls of the air sacs allow oxygen in the air to move into the blood, and carbon dioxide (CO_2) in the blood to move into the air sacs.
- The CO_2 is expelled when the animal exhales.
- This process is called gas exchange.
- Anything that interferes with this process may cause poor performance, disease or death.

ETIOLOGY

- Viruses, bacteria, or a combination of both.
- Fungus.
- Metazoan parasites
- Physical and chemical agents.
- Bronchogenic (inhalation)
- Hematogenous(Sepitcemia)
- Verminous(parasites)

Cattle:

- Pneumonic pasteurellosis (shipping fever) - M. haemolytica,P. multocid ,Histophilus somnus, Actinomyces pyogenes and Streptococcus spp.
- Mycoplasma mycoides.
- Mycotic pneumonia due to Mortierella wolfii in adult cattle.
- Lungworm pneumonia(Dictyocaulus viviparous)

ETIOLOGY

Horses:

- Glanders and epizootic lymphangitis (Histomonas farcinicus) lead to pneumonia.
- Bordetella bronchiseptica in adult horses.
- Equine herpesvirus infection, equine influenza.
- Strangles is a diseases of the upper respiratory tract but invasion of lower respiratory tract also.
- Rigrous exercise in winter, damage air ways.

ETIOLOGY

Sheep and goats:

- Pneumonic pasteurellosis (Mannheimia spp.) as acute primary pneumonia.
- Streptococcus zooepidemicus,Salmonella abortus-ovis and Corynebacterium pseudo tuberculosis .
- Mycoplasma strain F 38 or Mycoplasma capri, cause a devastating pneumonia in goats.
- Chronic interstitial pneumonia is a common sequel.
- M. mycoides var. mycoides is most common.
- Pigs: Mycoplasma sp.,Pasteurela multocida and septicemic salmonellosis cause pneumonia

EPIDEMIOLOGY

- The susceptibility of the animal (No registance).
- Three risk factors are involve in pathogenesis:
    1. Animal
    2. Environmental and management
    3. Pathogen.

Some of common risk factors include:

- The weaning of beef calves in cold climates
- The long transportation of beef cattle.
- Poor ventilation, poor hygeine and Auction mart.

PATHOGENESIS

Pulmonary defense mechanisms

- Major airways and the lung parenchyma prevent the entry or neutralize or remove injurious agents (bacteria).
- Many infections of the respiratory tract originate from aerosolized particles.
- These respiratory infections deposited in the respiratory tract.

PATHOGENESIS

- Biochemical, physiological and immunological defense mechanisms protects the lungs.

Major defense mechanisms of the respi tract:

- Aerodynamidc filtration by the nasal cavities
- Sneezing
- Local nasal antibody
- The laryngeal reflex
- The cough reflex
- Mucociliary transport mechanisms
- Alveolar macrophages
- Systemic and local antibody systems

PATHOGENESIS

- In bacterial pneumonias,toxins is produced.
- Inflammatory exudate in the bronchi produce adventitious lung sounds such as crackles.
- Restricted gaseous exchange occurs due to reduction of alveolar spaces and obstruction of air passages.
- Cyanosis is most likely to develop at this stage due to poor oxygenation.

PATHOGENESIS

- Viral infection reduces the defence mechanism and lead to bronchitis and pneumonia.
- Allergens also cause irritation and inflamation and exudation leading to pneumonia.
- Endotoxins(Gm-ve) leads to endotoxemia and damage the blood vasculature in the lungs.

PATHOGENESIS

- Bacteria produces cytotoxin (leukotoxin) that kills various leukocytes(Manheimia disease).
- It reduces the primary pulmonary defense.
- Damage to micro vasculature in the lung.
- This lead to effusion of fluid, inflamation and pneumonia.
- Endothelial damage by endotoxin.
- Diffuse infection like E.coli mastitis.

PATHOGENESIS

- Endotoxin activates macrophages and neutrophils, provoking the release of a multitude of inflammatory mediators.
- Interleukin -1, interleukin- 6 and platelet activating factor.
- This lead to endothelial damage, leaky vessels.
- Release of arachidonic acid by Interleukins .
- This is metabolized by the cyclooxygenase pathway to yield prostaglandins.

PATHOGENESIS

- The destruction of leukocytes and respiratory epithelial cells and the release of DNA increases the viscosity of respiratory secretion
- Yellow or green respiratory secretions are due to the enzyme myeloperoxidase.
- This enzyme is released from leukocytes in the static secretion.

Interstitial pneumonia(Shipping fever)

- **Interstitium** is a collection of support tissues that incluces the alveolar epithelium, pulmonary capillary endothelium and basement membrane.
- Interstitial pneumonia(IP) occurs when fluid and white blood cells accumulate in interstitium.
- The air sac walls become thicker than normal.
- This increases the distance between the air and the blood, thus interfering with gas exchange.

Interstitial pneumonia(IP)

- Actually, in the early phases of severe IP, fluid and white blood cells ‘leak’ out of the walls and accumulate in the air sacs.

Causes of IP in Cattle:

- Viruses --bovine respiratory syncytial virus(BRSV).
- Feed Toxins and some toxic plants
- Fog Fever: Sudden exposure to lush pasture grasses (high in L-tryptophan).
- lungworms
- Allergens and hay mould.

Clinical Findings

- Lowest milk yield
- Dull nad poorer condition
- Usually have a dry staring coat
- Variable rectal temperature up to 39.5°C.
- Cough frequently with occasional nasal discharge.
- The respiratory rate is increased with an abdominal respiration.

Clinical Findings

- Some cattle stand with an arched back with the neck extended and the head held lowered suggestive of thoracic pain.
- *Affected animals cough frequently and have a purulent nasal discharge.*
- Rapid, shallow breathing
- Dyspnea , Polypnea.
- Bacterial bronchopneumonia is usually accompanied by a moist and painful cough.

Pneumonia(Showing Open Mouth breathing)

CALF PNEUMONIA

Etiology: Multifactorial involving infectious agents, the environment and the immune status of the calves.

- *Mannheimia haemolytica*
- *Haemophilus somnus*
- Infectious Bovine Rhinotracheitis(IBR)
- Bovine Respiratory Syncytial Virus (RSV)
- Parainfluenza III Virus (PI3).

Fog fever

- Fog fever is an uncommon condition causing severe respiratory distress in adult cattle one to two weeks after moving on to a lush silage/hay aftermath in June/September.

Cause:

- Ingestion of large amounts of the amino acid L-tryptophan along with lush green grasses.
- Its conversion in the rumen to 3-methyl indole and indole acetic acid cause Fog fever.

Fog fever

Clinical presentation of fog fever:

- Usually not more than 2 to 5 per cent of cattle at risk are severely affected with sudden onset of severe respiratory distress.
- The animal stands with its neck extended, head lowered, and moves very reluctantly.
- The nostrils are flared and the animal breathes through its mouth.
- There is an expiratory grunt and frothy saliva around the protruding tongue.

Fog fever

- The rectal temperature is normal. Coughing is not a frequent feature of fog fever.
- The mortality rate in severely affected cases is around 95% and often precipitated by movement or handling.
- May be confused with Lungworm,nitrate poisoning and grass staggers

Inhalation pneumonia (Aspiration pneumonia or Foreign-body pneumonia *in cattle*)

- Inhalation pneumonia most commonly results from inhalation of rumen contents following hypocalcaemia especially if the cow had become recumbent laterally.
- Faulty administration of drenches.
- Sudden death may follow drenching but this is unusual.
- Recovery from anesthesia.

Aspiration Pneumonia(Foreign-body pneumonia)

- Aspiration pneumonia is a pulmonary infection characterized by inflammation and necrosis caused by inhalation of foreign material.
- Aspiration pneumonia can occur in any animal, particularly following drenching or inadvertant placement of a tube into the trachea in large animals

Aspiration Pneumonia(Foreign-body pneumonia)

- Aspiration pneumonia is a pulmonary infection characterized by inflammation and necrosis caused by inhalation of foreign material.
- The severity of the inflammatory response depends on the material aspirated, the type of bacteria aspirated, and the distribution of aspirated material in the lungs.

Aspiration Pneumonia(Foreign-body pneumonia)

Clinical presentation(If survive)

- Typically, the cow has a painful expression and stands with a arched back.
- Stance with the neck extended and the head held lowered and walks slowly.
- The animal does not eat.
- The rectal temperature is elevated within a range 39.5 to 40.0°C. after 24 hours.

Aspiration Pneumonia

- There is a bilateral mucoid/purulent nasal discharge and the animal coughs frequently.
- The respiratory rate is elevated with an obvious abdominal component.
- The cow has halitosis (Bad breath)
- The milk yield is greatly reduced
- No time for treatment.
- If survive antibiotics , corticosteroids and NSAIDS.

Severe necrotic pneumonia and pleurisy (right lung) following inhalation of rumen content

Pleural abscesses

Clinical presentation:

- There may be no history of respiratory disease.
- Affected cattle typically present with a history of poor growth, weight loss, and/or poor milk yield over several weeks.
- Affected cattle often stand with a roached back.
- Stand with the neck extended and the head held lowered with a painful expression.

Pleural abscesses

- The respiratory rate is elevated with obvious abdominal respiration as the abscess often occupies a large proportion of the chest.
- A large pleural abscess may contain up to 50 litres of pus.
- The cow's appetite is reduced.
- The rectal temperature is only marginally elevated (39.0 to 39.5°C). There are no ocular or nasal discharges.

Necropsy of the bull confirmed a massive pleural abscess containing over 50 litres of pus.

Symptoms of Horse Pneumonia

- Pneumonia is very rare in horses, but if it does occur, it can be fatal.
- Foals, injured horses and old horses are mostly at risk of getting pneumonia.
- Any kind of upper respiratory infection can lead to pneumonia in horses.
- The horse may display signs of discomfort and fatigue.

Symptoms of Horse Pneumonia

- Loss in appetite and this is normally accompanied by fever.
- Lot of nasal discharge and cough.
- Reluctant to exercise and many refuse to move as they are very weak.
- Breathing is slow and labored.
- Weight loss in horses with pneumonia.
- Pneumonia is the result of a serious bronchitis

Differential Diagnosis

1. Bronchitis
2. Pleurisy
3. Pleural effusion
4. Lung abscess
5. Lung worms
6. Hypomagnesemic tetany
7. Nitrate poisning
8. Black disease

CLINICAL PATHOLOGY

- The laboratory examination of the exudates and secretions of the respiratory tract.
- Nasal swabs, tracheobronchial aspirates and broncho alveolar lavage samples can be examined for isolation of viruses, bacteria and fungi,
- Cytological examination and antimicrobial sensitivity test

CLINICAL PATHOLOGY

- Thoracocentesis can be used to obtain pleural fluid for analysis, cytological and bacreriological test .
- Hematological examination can indicate if the infection is bacterial or viral in nature.
- Serological test when viral interstitial pneumonia is suspected.
- When lungwonn pneumonia is suspected, fecal samples can be examined for detection of the larvae.

NECROPSY FINDINGS

- Gross lesions are usually observed in the anterior and dependent parts of the lobes.
- Even in fatal cases where much of the lung is destroyed, the dorsal parts of the lung lobes may be unaffected.
- Bronchopneumonia is characterized by presence of serofibrinous or purulent exudate in the bronchioles.
- There is lobular congestion or hepatization.

NECROPSY FINDINGS

- In interstitial pneumonia the bronchioles are clean and the affected lung is sunken, dark red in color and has a granular appearance.
- There is often an apparent firm thickening of the interlobular septae.
- In chronic bronchopneumonia of cattle there is consolidation,fibrosis and fibrinous pleuritis.
- Bronchi filled with exudate, bronchiectasis and pulmonary abscessation.

DIAGNOSIS

- Diagnosis is made on the basis of clinical findings for each type of pneumonia.
- Diagnosis can be made on the basis of laboratory findings.
- Diagnosis can be made on the basis of xray examination of chest area.
- Diagnosis on the basis of Isolation of bacteria.
- Diagnosis on the basis of Serological test for viruses.

TREATMENT

Antimicrobial therapy: The common bacterial pneumonias of all species will recover quickly (24-72 h) if treated with an adequate dose of the drug of choice early.

- Azithromycin, erythromycin oral for calves only @10 mg/kg bwt.
- **Tulathromycin @ 1ml/kg I/M(**Each mL of DRAXXIN 25 contains 25 mg of tulathromycin)

TREATMENT

- Fluoroquinolone(danofloxacin, enrofloxacin)
- **Danofloxacin** 18% Injectable Solution.
- The beta-lactam antimicrobials(penicillin, ceftiofur , Cefotaxim and Ceftriaxone).
- Aerosolization and inhalation of antimicrobials has the theoretic advantage
- Nonsteroidal anti-inflammatory drugs(NSAID).
- Corticosteroids

TREATMENT

- Theophylline has been evaluated as a bronchodilator to relieve respiratory distress in cattle with pneumonia.
- When it was given orally at a dose of 28 mg/kg BW daily for 3 days.
- Affected animals should be housed in warm, well-ventilated shed.
- Oxygen therapy in serious pneumonic cases.

TREATMENT

- If there is respiratory distress, dehydration, anorexia.
- Hospitalization for oxygen therapy and/or intravenous fluids and medications may be necessary.
- 95-105 mmHg is normal oxygen concentration in blood.
- Less than 75 mmHg need oxygen therapy.
- Hypertoni saline solutin should be given.

PLEURISY

Prepared by Prof. S . Haque, Ph.D

Dept of Veterinary Clinical Medicine

College of Veterinary Medicine and Animal Sciences(UOG)

PLEURISY

Definition:

1. Peurisy (Pleuritis) refers to inflammation of the parietal and visceral pleura.
2. Pleurisy is the term used to designate inflammation of the pleura or membrane lining the chest and covering the lungs.
3. Pleurisy results from an inflammation of pleural sac.

The pleura is a two layered sac that holds the lungs and separates them from the chest wall, diaphragm, and heart.

PLEURISY

- The pleura that lines the inside of the chest is called the parietal pleura.
- The pleura that covers the lungs is called the visceral pleura.
- The pleura is separated by a thin layer of fluid.
- Through this fluid lungs expand and contract easily during breathing (Lubricant).
- Pleurisy can cause pain with breathing.
- May be large amount of fluid to in pleural sac.
- Pleurisy often complicates with pneumonia.

ETIOLOGY

- Pneumonia can progress to pleuritis, and pleuritis can cause consolidation and infection of the lungs.
- Primary pleuritis: It is due to perforation of the pleural space and subsequent infection(TRP).
- Secondary pleuritis: It is develop due to infectious lung diseases.
- One layer is continuous with the outer wall of each lung, and the other layer lines the muscular chest wall and diaphragm.

ETIOLOGY

Cattle:

- Secondary to Mannheimia haemolytica pneumonia in cattle, especially feedlot cattle
- Tuberculosis (Bovine TB).
- Contagious bovine pleuropneumonia(CBP)
- Histophilus somnus(respiratory disease in bison, domestic sheep, and cattle).
- Sporadic bovine encephalomyelitis(CNS disease).

ETIOLOGY

Horses: Sudden chills, as exposure to cold draughts and cutting winds when the animal is overheated.

- It may also be due to direct injury from external injury to the walls of the chest.
- Thoracic hemangiosarcoma is recorded as a cause of chylothorax in the horse
- Influenza in horses.

ETIOLOGY (Sheep and goats)

- Pleuropneumonia associated with Mycoplasma spp.
- Mycoplasma mycoides subsp.
- Mycoides and Haemophilus spp.
- Streptococcus dysgalactiae in ewes.

Pigs

- Glasser's disease(caused by Haemophilus parasuis and cause Infectious polyarthritis).
- Pleuropneumonia associated with Actinobacillus (Haemophilus)
- Pleuropneumoniae and Haemophilus influenzae suis.

PATHOGENESIS

- Due to Inflamation there is production of serofibrinous inflammatory exudate, which collects in the pleural cavities.
- This causes collapse of the ventral parts of the lungs.
- Thus reduces vital capacity of the lungs and interfere with gaseous exchange.
- If the accumulation of fluid is too much there may be pressure on the atrium(Rt.atria)and a diminished return of blood to the heart.

PATHOGENESIS

- Fluid is resorbed in animals lungs that survive of the acute disease.
- Then adhesions of both pleura develop, restricting the movement of the lungs and chest wall.
- In all bacterial pleuritis, toxemia is common and usually severe.
- Toxemia develop when amounts of pus accumulate in the pleural sac.
- Continous friction increases the permiability of pleura and fluid leaked out in the pleural sac.

CLINICAL FINDINGS

- The clinical findings of pleuritis vary from mild to severe.
- It depend on the species and the nature and severity of the inflammation.

  In peracute to acute stages of pleuro-pneumonia there are:
- Fever, toxemia, tachycardia aand anorexia
- Depression, nasal discharge and coughing.
- Exercise intolerance, breathing distress and flared nostrils.

CLINICAL FINDINGS

- The nasal discharge depends on the presence or absence of pneumonia.
- It may be absent or copious and its nature may vary from muco hemorrhagic to mucopurulent.
- The odor of the breath may be putrid, which is usually associated with an anaerobic lesion.

CLINICAL FINDINGS

Pleural pain (pleurodynia) is common and manifested as:

- Pawing, stiff forelimb gait, abducted elbows and reluctance to move or lie down.
- In the early stages of pleuritis, breathing is rapid ,shallow and markedly abdominal.
- Movement of the thoracic wall is restricted.
- The inspiration is short and jerky, the expiration is longer.

CLINICAL FINDINGS

- Temperature on first days as high as106° F
- On 2nd or 3rd day falls to 103° or 104°.
- Pressure between the ribs gives pain and usually causes the animal to flinch and grunt.
- The pain is caused by the friction of the dry, inflamed pleural surfaces of the lung and chest on each other.
- At this stage auscultation detects a dry friction murmur.
- It resemble somewhat the sound made by rubbing two pieces of sole leather together.

CLINICAL FINDINGS

- The muzzle is hot and dry, the mouth slimy, and the secretions scanty.
- After 3 to 4 days the severity of the symptoms lessened.
- Pain decreases, the stiffness disappears, and the patient eats a little.
- The pulse softens, but remains quicker than normal.

CLINICAL FINDINGS

- Now, day by day the patient loses strength.
- The friction sound disappears as the exudation moistens the pleural surfaces.
- Percussion now shows a horizontal line of dullness, which day by day rises higher in the chest area.
- Pleural friction rubs are audible only during the initial stages of the disease

CLINICAL FINDINGS

- Pleural friction rubs are not audible when fluid accumulates in the pleural space.
- Subcutaneous edema of the ventral body wall extending from the pectorals to the prepubic area is common in horses.
- Ventral edema is less noticeable in other species.
- This may be due to blockage of lymphatics particularly of sternal lymph nodes.

CLINICAL FINDINGS

- In cattle, an inflammatory pleural effusion is often limited to one side of lung
- Pleural sacs(Rt and Left) do not communicate each other.
- Bilateral pleural effusion may indicate a bilateral pulmonary disease process.
- Right- sided congestive heart failure or hypoproteinemia may be the reason for bilateral pulmonary effusion.

CLINICAL FINDINGS

- The respiration grows more frequent and labored.
- The countenance(appearance) is anxious and eyes sink some what in their sockets
- Haggard (exhausted).
- In untreated and unfavorable cases death occurs during the second or third week.
- Either due to asphyxia or heart failure.

DIAGNOSIS

Medical imaging:

- Radiographic examination may reveal the presence of a fluid line and fluid displacement of the mediastinum and heart to the unaffected side and collapse of the lung.
- In cattle, pleural effusion cannot be located precisely by radiography because only laterolateral radiographs of the thorax can be taken.

DIAGNOSIS

- Ultrasonography is superior for the visualization of small volumes of pleural fluid that cannot be detected by auscultation and acoustic(waves in liquids)percussion of the thorax.
- Pleuroscopy using a rigid or flexible fiberoptic endoscope allows direct inspection of the pleural cavity.

CLINICAL PATHOLOGY
Thoracocentesis (pleurocentesis)

- Thoracocentesis to obtain a sample of the fluid for laboratory examination is necessary for a definitive diagnosis.
- The fluid is examined for its odor, color and viscosity, protein concentration and presence of blood or tumor cells, and is cultured for bacteria and antibiotic sensitivity test.
- It is important to determine whether the fluid is an exudate or a transudate.

CLINICAL PATHOLOGY

- Pleural fluid from horses affected with anaerobic bacterial pleuropneumonia may be foul-smelling.
- Pleural fluid reveals an increase in leukocytes count up to 40 000-100000 /mic.L and protein concentrations of up to 50 g/L (N=5.0 g/dL) .
- The fluid should be cultured for both aerobic and anaerobic bacteria and Mycoplasma spp.

CLINICAL PATHOLOGY

Hematology:

- In peracute bacterial pleuropneumonia in horses and cattle,leukopenia and neutropenia.
- In acute pleuritis with severe toxemia, hemo concentration, neutropenia with a left shift.
- In subacute and chronic stages normal to high leukocyte counts are often present.

NECROPSY FINDINGS

- In early acute pleurisy there is marked edema, thickening and hyperemia of the pleura.
- Engorgement of small vessels and the presence of shreds of fibrin.
- These can most readily be seen between the lobes of the lung.
- In the exudative stage the pleural cavity contains an excessive quantity of turbid fluid containing flakes and clots of fibrin.

Fibrinous pleuritis in a horse

NECROPSY FINDINGS

- The pleura is thickened and the central parts of the lung are collapsed and dark red in color.
- Pneumonia is present and may be pericarditis.
- In the later healing stages, adhesions connect the parietal and visceral pleurae.
- Type I fibrinous adhesions is associated with pneumonia
- Type II fibrinous proliferative adhesions are idiopathic(of unknown cause).

TREATMENT

- The principles of treatment of pleuritis are pain control, elimination of infection and prevention of complications.
- The primary aim of treatment is to control the infection in the pleural cavities using the systemic administration of antimicrobials.
- This should be selected on the basis of culture and sensitivity of pathogens from the pleural fluid.

TREATMENT

- Before the antimicrobial sensitivity results are available it is recommended that broad spectrum antimicrobials be used.
- Long term therapy daily for several weeks may be necessary.
- Drainage and lavage of pleural cavity.
- Drainage of pleural fluid removes exudate from the pleural cavity and allows the lungs to re-expand.

TREATMENT

Criteria for drainage include:

- An initial poor response to treatment.
- Strepto penicillin can be given.
- Large quantities of fluid causing respiratory distress.
- Bacteria in cells of the pleural fluid.
- Clinical experience suggests that drainage improves the out come .
- Thoracotomy can be done.

PNEUMOTHORAX

Definition:

- Pneumothorax is defined as air in the pleural cavity via lung, mediastinal or thoracic wall disease or injury.
- Pneumothorax refers to the presence of air (gas) in the pleural cavity.
- Entry of air into the pleural cavity in sufficient quantity causes collapse of the lung.
- There is impaired respiratory gas exchange with consequent respiratory distress.

Etiology

- Spontaneous cases occur without any identifiable inciting event.
- Open pneumothorax in which gas enters the pleural space by ruptured or lacerated lung.
- Through an open wound in the chest wall.
- Closed pneumothorax refers to gas accumulation in the pleural space in the absence of an open chest wound.

Etiology

- Tension pneumothorax: It occurs when a wound acts as a one-way valve.
- Air entering the pleural space during inspiration but being prevented during expiration by a valve-like action of the wound margins.
- Trauma pneumothorax: when a wound penetrates the thoracic wall, including the parietal pleura.

Pathogenesis

There are three mechanisms Pathogenesis :

1) An imbalance between protease secreted by neutrophils and macrophages.

- Antiprotease activity results in destruction of alveolar walls and interstitial matrix.

2) Inappropriate maintenance of lung structure and repair follows injury.

Pathogenesis

3) The condition develops secondary to obstruction of airways due to chronic bronchitis/ bronchiolitis or congenital airway.

- This creates a "check valve" lesion.
- The air is able to enter alveoli on inspiration or through collateral ventilation but is unable to leave freely and causes air trapping in lung.

Pathogenesis

- Entry of air into the pleural cavity results in collapse of the lung.
- This can be partial or complete.
- Collapse of the lung results in alveolar hypoventilation, hypoxemia, hypercapnia, cyanosis, dyspnea, anxiety.
- Hyper resonance on percussion of the affected thorax.

Pathogenesis

- The degree of lung collapse varies with the amount of air that enters the cavity:
- Small amounts are absorbed very quickly .
- Large amounts may cause fatal anoxia.
- Recurrent airway obstruction, or “heaves” in horses is due to chronic bronchitis and bronchiolitis.
- Causes alveolar hyperinflation by air trapping.
- It is partially reversible with bronchodilators.

Clinical findings

- Acute inspiratory dyspnea, which may terminate fatally within a few minutes if the pneumothorax is bilateral.
- If unilateral, the rib cage on the affected side collapses and shows decreased movement.
- There is a compensatory increase in movement and bulging of the chest wall on the unaffected side.

Clinical findings

- On auscultation of the thorax, the breath sounds are markedly decreased in intensity or absent.
- The mediastinum (central compartment of the thoracic cavit) may bulge toward the unaffected side.
- May cause moderate displacement of the heart.
- The heart sounds on the affected side have a metallic note and the apex beat may be absent.

Clinical findings

- On percussion of the thorax on the affected side, a hyperresonance is detectable over the dorsal aspects of the thorax.
- Affected animals are anxious, tachypneic and in variable degrees of respiratory distress.

Clinical findings

- Tachypnea.
- Dyspnea.
- Changes in breathing pattern - shallow and rapid, becoming irregular or deep and slow.
- Evidence of other problems:
  - Coughing, nasal discharge and other signs of severe lower respiratory tract disease, eg tracheal foreign body , pleuropneumonia .

Clinical findings

- Depends on the amount of air and whether uni- or bi-lateral:
  - Small amounts on one side - no clinical signs.
  - Larger amounts, especially if bilateral → respiratory distress
- Cyanosis if arterial oxygenation impaired.
- Sudden change in breathing pattern and oxygenation levels subsequent to procedure such as thoracocentesis or positive pressure ventilation.

Differential diagnosis

- Hemothorax.
- Fractured ribs.
- Pneumonia [Lung: pneumonia - neonatal bacterial]
- Pleuropneumonia .
- Ruptured diaphragm .
- Acute obstructive pulmonary disease [Lung: recurrent airway obstruction (RAO] .

Diagnosis

- Definitive diagnosis is based on demonstration of pneumothorax by radiographic or ultrasonographic examination.
- Clinical findings
- Imagings

Treatment

- Emphysema is an irreversible lung lesion.
- Emergency decompression of pleural cavity.
- Inserting a needle into the pleural cavity.
- Connecting it to a tubing and submerged into a flask of saline or water.
- This creates a water seal drainage.
- Thoracostomy tubes attached to Heimlich thoracic drainage valves are effective in preventing aspiration of air.

EMPYSEMA

Definition:

- Emphysema is a long-term, progressive disease of the lungs that primarily causes shortness of breath due to over-inflation of the alveoli(air sac).
- Emphysema means over distension of alveoli without any change in the pulmonary tissues.
- There is reduction in the air space & loosing the elasticity of the alveoli.

EMPYSEMA

- Emphysema is a lung condition in which tiny air sacs in the lungs - alveoli - fill up with air.
- As the air continues to build up in these sacs, they expand, and may break or become damaged and form scar tissue.
- The patient becomes progressively short of breath.

EMPYSEMA

Classification:

- Acute alveolar emphysema:- mostly in cattle & is temporary distension of the alveoli.
- Interstitial emphysema:- in which rupture of one or more alveoli & the air escape to the interlobular & intralobular spaces mostly in cattle & is fatal.
- Chronic alveolar emphysema or Heaves Disease in horses.
- In Heaves Disease over distension of the alveoli till the complete loss of elasticity.

Etiology

A-Acute alveolar emphysema:

- Persisting cough.
- Obstruction of the alveoli with food particles.
- Foreign bodies.
- Larvae of ascaris
- Dictycoulus viviparous
- Exudates in case of bronchitis.
- Traumatic reticuloperitonitis
- Pulmonary abscess.

Etiology

B-Interstitial emphysema:

- Heavy infestation with lungworm.
- Damage of the lung tissue.
- In case of parturition
- Over distention & rupture of the alveoli.
- In Horses it may be due to heavy work & exercise.
- Dusty foods causes continuous coughing.

Pathogenesis

- Over stretching of the supporting & elastic tissue of the pulmonary parenchyma leads to excessive dilatation of the alveoli.
- Collapse of one part of lungs(Alveoli)cause the alveoli of other parts to be distended to compensate.
- Incomplete evacuation during expiration.
- The retention of CO2 stimulate the depth of respiratory development of anoxia .
- Reduced metabolism of all body tissues

Clinical findings

1-Acute alveolar emphysema

- Dyspnea is more severe.
- On percussion , Hyperresonant sound in the anterior & posterior parts of the lung.
- On ausculatation , Rales associated with bronchitis.
-

Clinical findings

Interstitial emphysema:

- Dyspnea.
- Subcutaneous emphysema over the trunk, shoulder & inlet of thorax and may spread all over the lung (in cattle)
- In other species,perforation of pleura.
- On percussion , Hyperresonant sound.
- On auscultation , Crepitant rales.

Clinical findings

Chronic alveolar emphysema:

- Signs appear gradually only during exercise.
- Prolonged expired phase in each respiratory cycle.
- Increased depth of respiration
- On percussion hyperresonsnt sound
- On auscultation crepitant rales.
- Expansion of the lung over the heart.
- Absence of heart sound.
- Extension of the lung till 14,th rib(11^{th} rib)

Clinical pathology &Necropsy findings

- Retention of CO2 increases the alkali reserve.
- Lungs are distended & became pale in colour.
- In interstitial emphysema,the interalveolar septae are distended with air
- There may be congestive heart failure.
- Histopathologically bronchitis is present in most cases.

Diagnosis

Exercise test in horses :-

- Count the respiration per minute before exercise.
- Allow it to exercise.
- Count the respiration every 5 minutes.
- Then put the animal in the stable for the rest.
- Again count the respiratory movements every 5 minutes.
- The normal respiratory rate is 10-14 /minute.
- The rate after exercise is 50-60 movements/minute.
- This rate will come back to normal after 15-18 minutes.
- Rate in diseased horses after exercise is 80-120/ minute. This rate not return back before 30 minutes.

Treatment

- Beladona 25-40 gm. daily for adult horses suffering from chronic alveolar emphysema.
- Oxygen therapy may be recommended.
- Atropine to relieve dyspnea specially if odema.
- Atropin@0.04 mg/kg . (15 mg/mL)–I/m or I/v
- Antihistaminics therapy because histamine release induce bronchoconstriction.

DISEASES OF THE NEW BORN

- In terms of survival, the first few weeks are the most crucial of a young calf's life, as their bodies are not yet developed to the point where they can effectively fight off illnesses. With so many potential health hazards around, it is important that as a livestock farmer you are at least aware of these 5 common calf diseases, and know what steps to take in order to prevent them and, if needs be, treat them.

Scour

- Possibly the most prevalent cause of calf death in the first month of life, symptoms of scour include:
- Severe diarrhoea
- Panting as though physically exhausted
- Sunken eyes
- A reluctance to feed either from a bottle or direct from their mother
- Lethargy

Dehydration

- Dehydration resulting from diarrhoea is often the direct cause of death from Scour.
- Scour can be brought on by bacteria (e.coli or salmonella usually) or a virus, and it is not always easy to determine which. Because of this it is difficult to tell what kind of medication, anti-bacterial or anti-viral, is best to administer, which is why taking preventative measures is essential.
- There are a number of effective routes you can take in order prevent calf scours, including pre-natal vaccinations, thoughtful colostrum management, and generally ensuring the good hygiene of living and sleeping areas.
- If one of your calves has scour, the best thing you can do is quarantine it immediately and take steps toward replacing the electrolytes, glucose, minerals and vitamins lost through diarrhoea.

Salmonellosis

- As well as causing Scour, Salmonella bacteria can also cause Salmonellosis; the most common symptoms of which are:
- Dysentery
- Pneumonia
- Arthritis
- Jaundice
- Septicaemia
- All the symptoms of Scour

Pneumonia

- The single most common and deadly killer of calves, Pneumonia is the primary cause of a number of viral infections, and can be brought about as a result of bacterial infections (such as Salmonellosis). Pneumonia is an opportunistic calf disease that will strike when their immune system is already weak or compromised, and the symptoms of Pneumonia include:
- Panting
- Coughing
- Wheezing
- Loss of appetite
- Mucosal discharge
- Lethargy & depression
- Body temperature over 39.6°C

DISEASES OF URINARY SYSTEM

Prepared by Prof. S . Haque, Ph.D

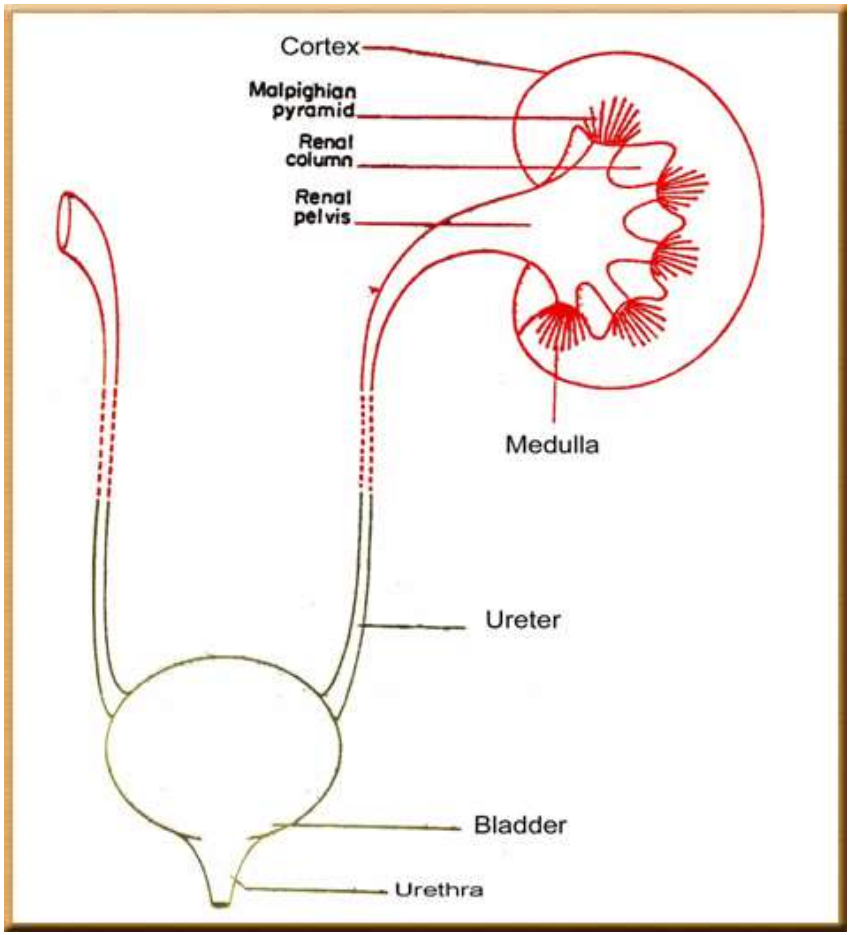
Dept of Veterinary Clinical Medicine

College of Veterinary Medicine and Animal Sciences(UOG)

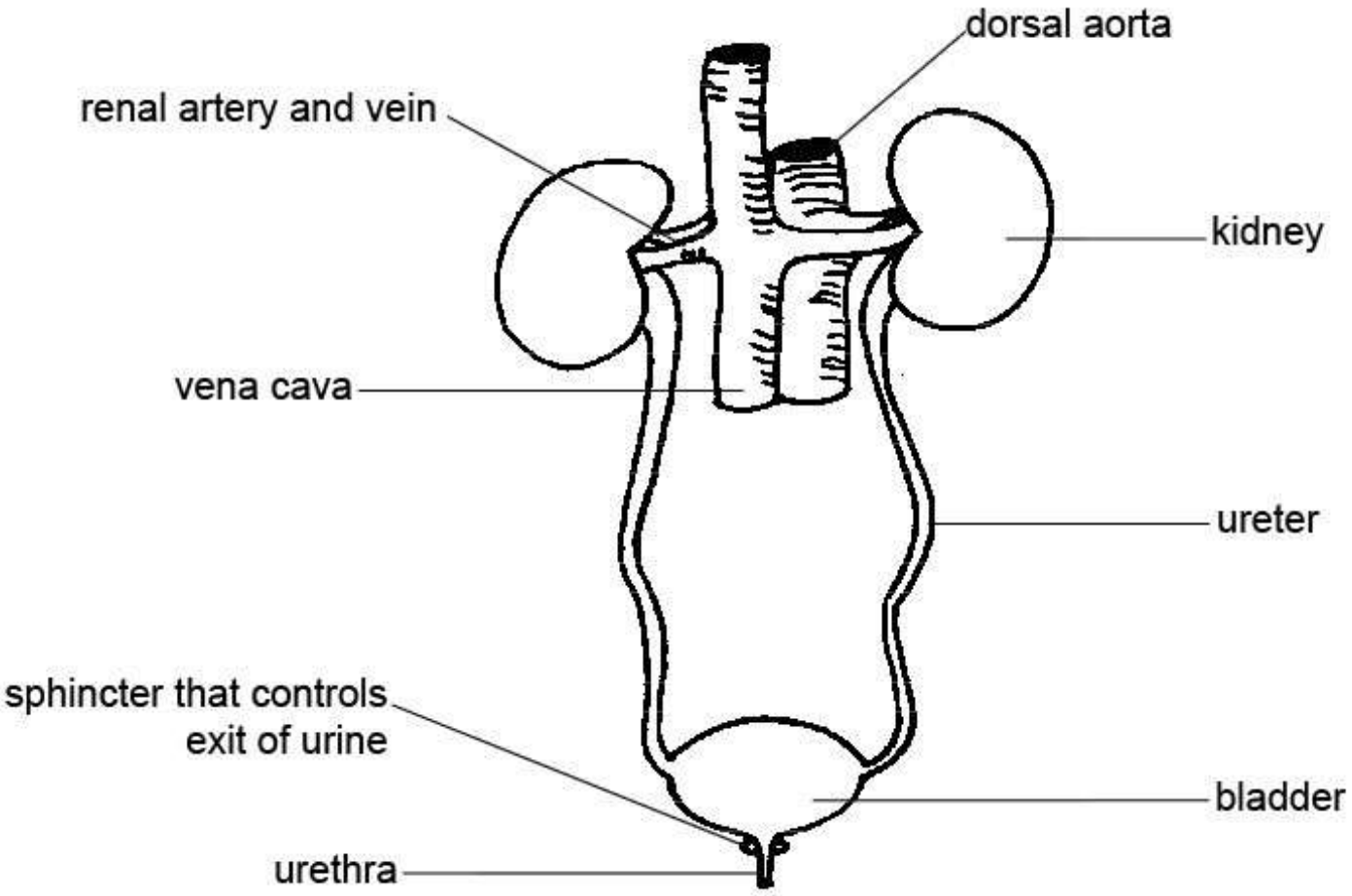
DISEASES OF URINARY SYSTEM

The parts of the urinary system are:

- A pair of kidneys
- A pair of ureters
- Bladder
- Urethra.



DISEASES OF URINARY SYSTEM



DISEASES OF URINARY SYSTEM

- The incidence of urinary system disease in cattle is quite low but more in dog and cat.
- Basically there are two main functions of the kidney:

1. To excrete the waste products of metabolism (including urea or uric acid, creatinine, ammonia).
2. To regulate desirable constituents like water glucose and amino acids.

DISEASES OF URINARY SYSTEM

- Reabsorption of water is by anti diuretic hormone.
- *it is* released by t he posterior pituitary gland.
- Kidney secretes two important endocrine substances:
- Erythropoietin – Help in RBCs synthesis
- Renin which regulates aldosterone secretion by the adrenal cortex.
- Renin convert angiotensinogen to angiotensin -I.
- ACH convrt angiotensin -I to angiotensin-II.
- Angiotensin-II is vasoconstrictor.

DISEASES OF URINARY SYSTEM

- Disease of the kidneys, the ureters, bladder and urethra interferes with glomerular filtration.
- It disturbs the excretion of metabolic waste products, and upsets protein, solute and water homeostasis.
- When loss of function is only partial it is called as renal insufficiency.
- When complete loss of function, called renal failure(ARF and CRF).
- Renal ischaemia may cause Renal Insufficiency

DISEASES OF URINARY SYSTEM

The functions of the urinary system are:

- To regulate the chemical composition and volume of the body fluids – *osmoregulation*
- To remove nitrogenous waste products and excess water from the body – *excretion*
- To act as an endocrine gland by the secretion of the hormone *erythropoietin.*

Maintain ph of the blood as it secrete phosphate

RENAL ISCHEMIA

- Definition: Necrosis or imfarction of kidney tissues due to low blood supply to kidney.
- Reduced blood flow through the kidneys usually results from general circulatory failure.
- Characterised by transitory oliguria , anuria and uremia if the Circulatory failure is not corrected.

ETIOLOGY

- Any condition which predisposes the animal to marked hypotension and release of endogenous pressor agents like chatechlamine(Vasoconstrictor).

Ischemia may be classified into :

- Acute renal ischemia
- Chronic renal ischemia

ETIOLOGY

Acute renal ischemia:

- General circulatory failure .
- Shock.
- Dehydration.
- Acute hemorrhagic anemia.
- Acute heart failure.
- Diarrhea.
- Vomition .

Chronic renal ischemia: Due to Congestive heart failure

PATHOGENESIS

- Acute ischemia of the kidneys occurs when compensatory vasoconstriction affects the renal blood vessels in response to a sudden reduction in cardiac output.
- As blood pressure falls, glomerular filtration decreases.
- Metabolites that are normally, excreted accumulate in the blood stream.

PATHOGENESIS

- The concentration of urea in the blood increases, giving rise to the name pre renal uremia.
- As glomerular filtration falls, tubular resorption increases, causing reduced urine flow.
- Up to a certain stage, the degenerative changes are reversible by restoration of renal blood flow.
- But if ischemia is severe enough and of sufficient duration, the renal damage is irreversible.

PATHOGENESIS

- Acute circulatory failure cause more degenerative lesions to kidney than chronic congestive heart failure.
- The parenchymatous lesions vary from tubular necrosis to diffuse cortical necrosis in which both tubules and glomeruli are affected.
- The nephrosis due to hemoglobinuria appears to be caused by the vasoconstriction of renal vessels
- It is less due to direct toxic effect of hemoglobin on renal tubules.

PATHOGENESIS

- Uremia in acute hemolytic anemia and in acute muscular dystrophy with myoglobinuria may be exacerbated by plugging of the tubules with casts of coagulated protein.

CLINICAL FINDINGS

- Renal ischemia does not appear as a distinct disease.
- Its signs are masked by the clinical signs of the primary disease.
- Oliguria and azotemia will go unnoticed in most cases if the circulatory defect is corrected in the early stages.
- Renal insufficiency may cause a poor response to treatment with the infusion of other fluids.
- Mostly in hemorrhagic or hemolytic anemia, in shock or dehydration.

CLINICAL FINDINGS

- The general clinical picture is one of acute renal failure.

  There is uremia which reflect as :

- Recurrent vomition
- Lethargy
- Complete loss of appetite.
- Oliguria or anuria.
- Hypothermia, shock and death.

CLINICAL PATHOLOGY

- Urinalysis: On urinalysis, proteinuria is an early indication of damage to the renal parenchyma
- Serum biochemistry: serum urea nitrogen and creatinine concentrations are mostly raised.
- Large volumes of urine of low specific gravity after a period of oliguria.
- It is usually a good indication of a return of normal glomerular and tubular function.

NECROPSY FINDINGS

- Lesions of renal ischemia are present primarily in the cortex, which is pale and swollen.
- There may be a distinct line of necrosis visible at the corticomedullary junction.
- Histologically there is necrosis of tubular epithelium and, in severe cases, of the glomeruli.
- In hemoglobinuria and myoglobinuria hyaline casts are present in the tubules.

Dignosis

- On the basis of clinical findings and lab findings.
- Serum creatinine raised: Normal -0.9 to 1.2mg%
- BUN—20 40 mg%
- Specific gravity of urine—it is increased.
- Normal sp gravity of urine - cattle and horses – 1.015 to1.050
- Leukocytosis— normal leukocytes value is 8000-11000 cubic milli liter.
- Anemia – Low Hb – normal value is 10 to 14gm%

TREATMENT

- Treatment must be directed at correcting fluid, electrolyte and acid-base disturbance as soon as possible.
- If renal damage has occurred, supportive treatment is given like dextrose 5% @ 20ml/kg
- Ringers lactate is given to maintain blood ph.
- Use of urine acidifiers such as ammonium chloride at a dose rate of 50-100 mg /kg twice a day orally.
- Antibotics can be given if infection is there.

CYSTITIS

PREPARED BY PROF. S . HAQUE

DEPT. VETERINARY CLINICAL MEDICINE

CYSTITIS

Definition: By definition cystitis means inflammation of the urinary bladder.

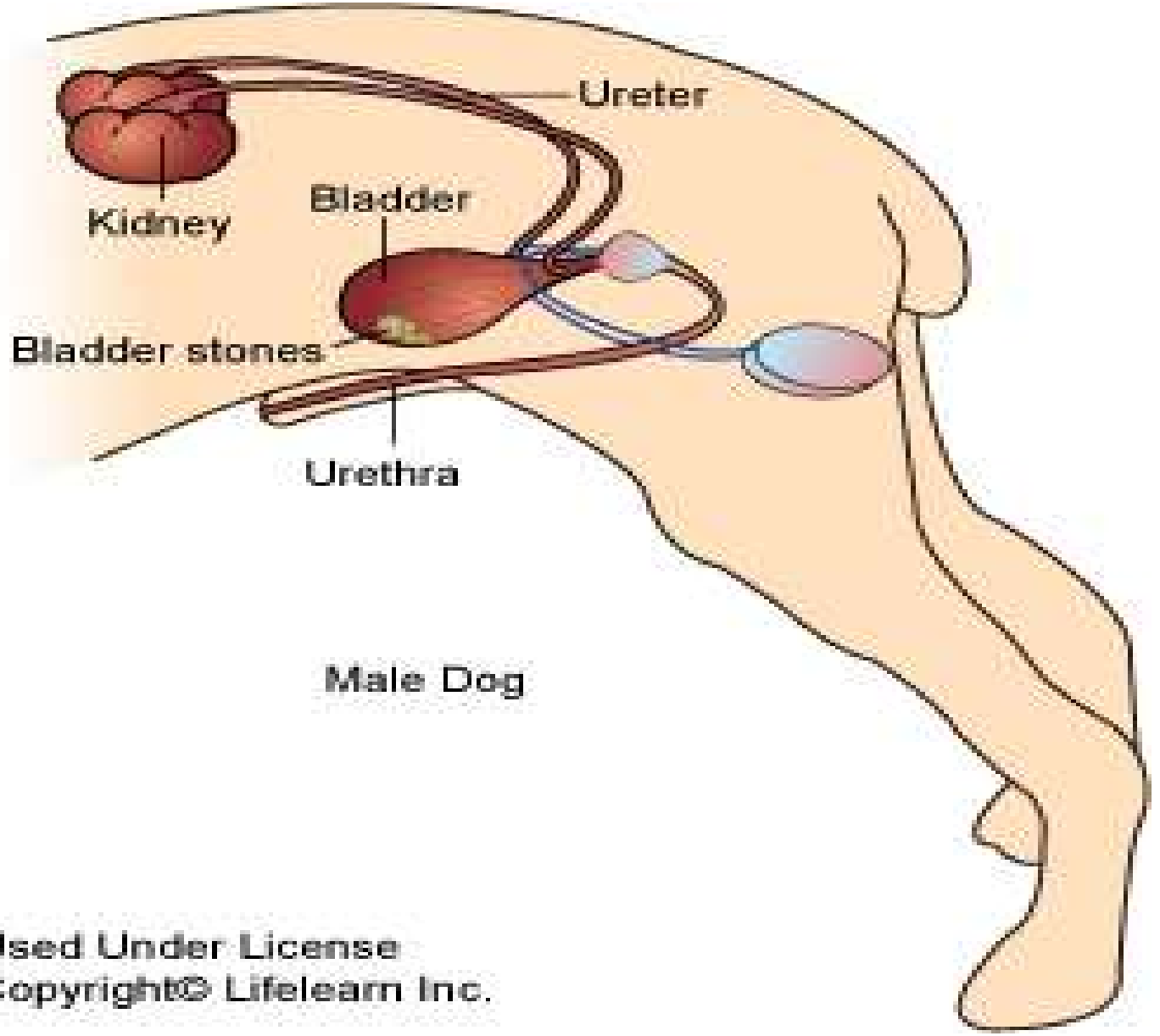
- A number of diseases and conditions can cause the inflammation of urinary bladder that is called cystitis.
- The bladder wall can become irritated by crystals, bacteria, and chemical changes of the urine.

Etiology

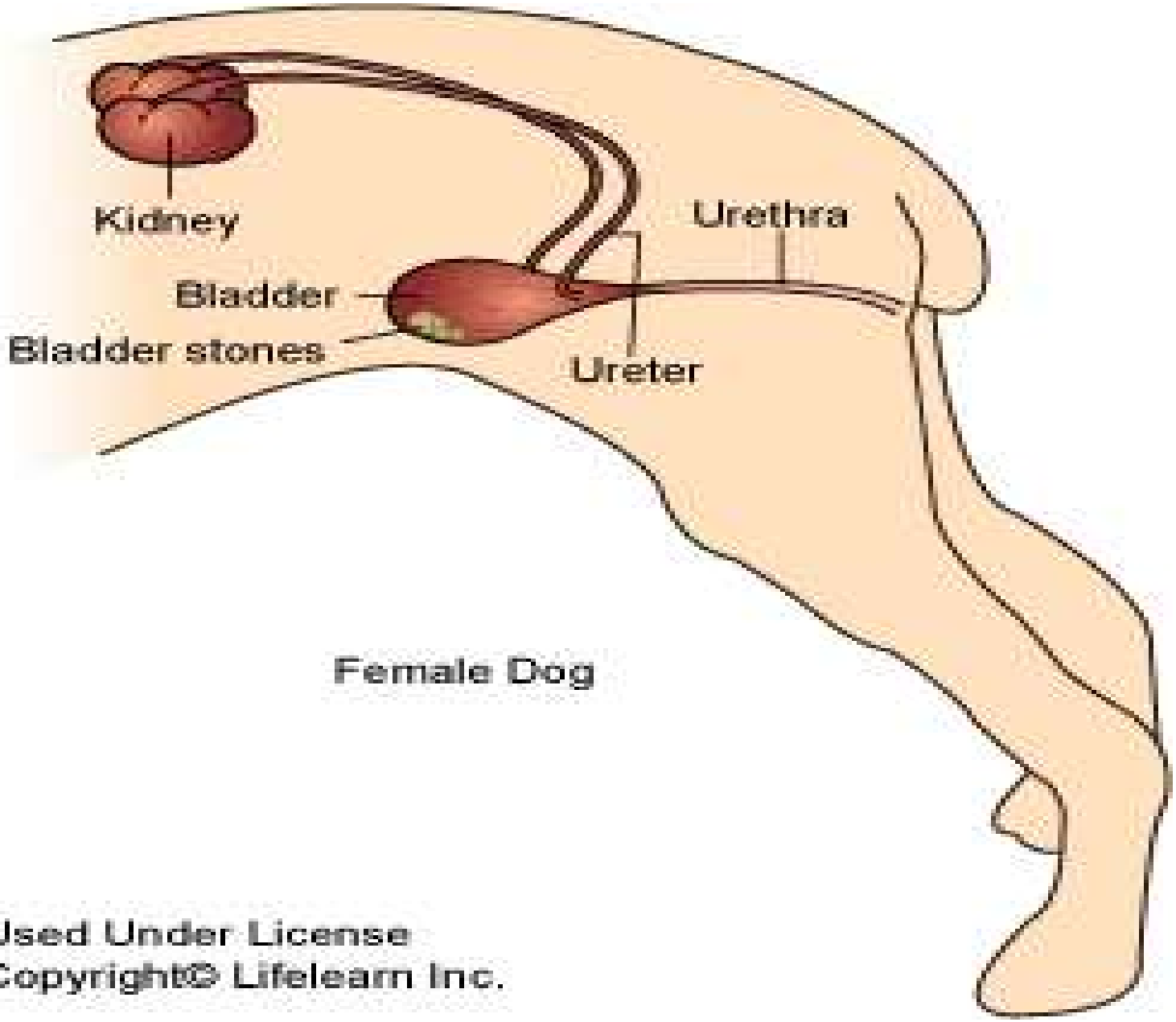
- The most common cause of cystitis in dogs is an infection caused by bacteria.

Other common causes include:

- Bladder stones
- Tumors or polyps in the bladder.
- Abnormal anatomy (especially in female dogs).
- Some dogs may have interstitial or "sterile cystitis“.
- It is a condition that causes inflammation and associated clinical signs without any infection.



Male Dog



Female Dog

Etiology

- Unlike dogs cystitis in cats is frequently caused by stress rather than by an infection.
- Feline *interstitial* cystitis, sometimes called feline *idiopathic* cystitis or FIC.
- It is an inflammation of the bladder that causes symptoms of lower urinary tract disease.
- Feline interstitial cystitis can occur in both female and male cats.
- It is a chronic disease that can be difficult to treat and frustrating for cats and cat owners alike.

Clinical findings

- The most common clinical sign is *hematuria* or blood in the urine.
- Cystitis often causes discomfort and pain.
- Dogs with cystitis will spend several minutes squatting and producing only a small amount of urine.
- Dog urinate more frequently than normal.
- Bacterial infections usually cause hematuria and *dysuria* (straining to urinate.)

Clinical findings

- Bladder stones are often very rough and cause irritation as they rub against the bladder wall.
- Tumors or polyps are usually not irritating to the bladder, but they can cause bleeding and straining to urinate.
- A diverticulum is a small pouch in the wall of the bladder that usually causes hematuria and dysuria secondary to the chronic bacterial infection
- Bacteria often reside deep in the diverticulum and are extremely difficult to cure without surgical removal of the pouch.

Clinical findings

Symptoms associated with interstitial cystitis include:

- Frequent attempts to urinate
- Straining to urinate
- Urinating in inappropriate places in the house
- Crying out during attempts to urinate
- Blood-tinged urine

Diagnosis

- A history of hematuria, dysuria, and pollakiuria is strong evidence of some form of cystitis.
- Abnormally frequent urination- pollakiuria
- **Bladder palpation** is the first "test" for bladder stones, since stones may be large enough to be felt .
- Urinary stones can occur as a result of cystitis.
- The bacteria form a nidus (a central point) around which the stone develops.

- A **urine culture and sensitivity** determines if bacteria are present and what antibiotics are likely to be effective for killing bacteria.
- **Bladder radiographs** (x-rays) are taken to evaluate the bladder for common types of bladder stones.
- Mineral composition of some stones requires special radiographs, using contrast materials.
- Plain radiographs rarely show bladder tumors, polyps, or diverticula.
- A plain radiograph can be made without sedation or anesthesia in most dogs.
- **Complete blood count (CBC).**

Treatment

- Treatment is based on the cause.
- Bacterial infections are generally treated with antibiotics.
- Some bladder stones can be dissolved with special diets while others require surgical removal.
- Benign bladder polyps can usually be surgically removed, but malignant bladder tumors are difficult to treat successfully.
- A bladder diverticulum should be removed surgically.
- Pain relief or anti-inflammatory medications to relieve discomfort and improve urine outflow.

Treatment

- Increased water consumption, either by drinking more water or eating a moist diet.
- This will dilute the urine and help flush out bacteria, inflammatory debris, and dissolved crystals.
- NSAID should not be given in cats as it causes renal failure.
- Propper antibiotics shoul be given to cats.
- Chronic forms of cystitis may require the *use of long-term antibiotics to* be given at bedtime.

UROLITHIASIS

Prepared by Prof. S. Haque, Ph.D

Dept. of Veterinary Clinical Medicine

UROLITHIASIS

Definition:

- Urolithiasis can be defined as the formation of sediment any where within the urinary tract.
- An urolith may be defined as the aggregation of crystalline and matrix materials'
- Urolith may be in one or more locations within the urinary tract.
- Term urolith is derived from the Greek word:
  ouron = urine
  lithos = stone.

EPIDEMIOLOGY

- Species affected: Cattle, calves, horses, sheep and goats.
- Mostly castrated males suffer more.
- Females have short urethra of wide diameter and suffer less.
- Females usually pass the calculi.
- Obstructive urolithiasis is the most common urinary tract disease in breeding rams and goats.
- Urolithiasis occurs sporadically in horses.
- The prevalence in horses is as low as 0.04-0.5%

ETIOLOGY

- Urinary calculi, or uroliths, form when inorganic and organic urinary solutes are precipitated out of solution.
- The precipitates occur as crystals or as amorphous(non-crystalline solid) deposits.
- Calculi form over a long period by a gradual accumulation of precipitate around a nidus.
- An organic matrix(cells like osteoblast) is an integral part of most types of calculus.

ETIOLOGY

There are three main groups of factors that contribute to urolithiasis:

1. Those that favours development of a nidus around which precipitation and concretion can occur.
2. Those that facilitate precipitation of solutes on to the nidus.
3. Those that favor concretion by cementing precipitated salts to the developing calculus.

ETIOLOGY

Nidus formation:

- A nidus favors the deposition of crystals around itself.
- A nidus may be a group of desquamated epithelial cells or necrotic tissue.
- It may be formed as a result of occasional infection in the urinary tract.

ETIOLOGY

1. Vitamin A deficiency- it causes desquamation of epithelial cells around which crystals deposit.

- The pH of urine affects the solubility of some solutes.
- Mixed phosphate and carbonate calculi being more readily formed in an alkaline than in acid urine(medium).
- Urine of cattle, horses, sheep and goats are alkaline . Normal ph in these sps is 8 to 8.4

ETIOLOGY

- Ammonium chloride or phosphoric acid added to the rations of steers increases the acidity of the urine and less calculi.
- Low intake of water and concentration of urine.
- The mucoprotein content of urine of steers and lambs is increased by heavy concentrate and low roughage rations.
- Mucoprotein act as a cementing agent.

ETIOLOGY

- Stasis of urine favors precipitation of solutes.
- It provide cellular material for a nidus.
- Ingestion of plants with a high oxalic acid.
- Diets high in magnesium increases incidence of obstructive urolithiasis.
- Pasture containing plants with high levels of silica,oxalates and estrogen cause uroliths in animals of all ages and sexes

PATHOGENESIS

- Calculi may be present in kidneys, ureters, bladder, and urethra.
- In a few animals pyelonephritis, cystitis, and urethral obstruction may occur.
- Obstruction of one ureter may cause unilateral hydronephrosis, in compensation to the contralateral kidney.

PATHOGENESIS

- Simple urolithiasis has relatively little importance.
- obstructive urolithiasis is a fatal disease unless the obstruction is relieved.
- Rupture of the urethra or bladder occurs within 2-3 days if the obstruction is not relieved and the animal dies of uremia or secondary bacterial infection.

PATHOGENESIS

- Rupture of the bladder is more likely to occur with a spherical, smooth calculus that causes complete obstruction of the urethra.
- Rupture of the urethra is more common with irregularly shaped stones that cause partial obstruction and pressure necrosis of the urethral wall.

PATHOGENESIS

Occurrence:

- Urethral obstruction may occur at any site but is most common at the sigmoid flexure in wethers or rams.
- It occur at all sites where the urethra narrows.
- Urolithiasis is mpre common in males.
- Less common in femals as it pass out.
- Urolithias due to siliceous calculi is most common in beef feeder cattle.

Factors favoring concretion and cementing

- Mucoprotein, especially mucopolysaccharide fraction may act as commenting agent.
- High concentration diet with low roughage.
- High phosphorus contents in the diet.
- High concentration diet rapid gain in body weight.
- Rapid turnover of supporting tissues.
- High level of mucoprotein in the urine.

Chemical composition of calculi

- It varies and depends mainly on the dietary intake of individual elements.
- Calcium, ammonium, and magnesium carbonate are the common calculi in cattle and sheep.
- Calcium carbonate and phosphate are more common in the Horse.
- Silicon calculi are common in grazing cattle and sheep.
- Calculi may be found as one stone, or more.
- It may be large and smooth or small and rough.

CLINICAL FINDINGS

Obstruction of the urethra by a calculus:

- It is common in steers and wethers.
- Syndrome of abdominal pain with kicking at belly and swishing of the tail.
- Repeated twitching of the penis.
- Shaking the prepuce .
- Strenuous efforts to urinate
- Straining, grunting and grating of the teeth.
- Passage of only a few drops of blood stained urine.

CLINICAL FINDINGS

- A heavy precipitate of crystals is often visible on the preputial hairs or inside
- Some animals with urethral obstruction will have a dry prepuce because of the absence of urination.
- This sign is not specific for urolithiasis.
- In urolithiasis may be dribbling of urine.
- Dribbing of Blood-Stained urine if complete anuria is not present.

CLINICAL FINDINGS

Cattle with incomplete obstruction:

- They are - 'dribblers‘.
- Pass small amounts of bloodstained urine frequently.
- Occasionally a small stream of urine will be voided followed by a complete blockage.
- This confuses the diagnosis.
- Here the calculus is triangular in shape and allows small amounts of urine to move around the obstruction at irregular intervals.
- However, these are rare cases.

CLINICAL FINDINGS

In rams and bucks:

- Obstructive urolithiasis.
- sudden depression
- Inappetence
- Stamping the feet
- Tail swishing
- Kicking at the abdomen
- Anuria
- Passage of only a few drops of urine .

Rupture of the bladder(Cystorrhexis)

- If the obstruction is not relieved, urethral rupture or bladder rupture usually occurs within 48 hours.
- With urethral rupture, the urine leaks into the connective tissue of the ventral abdominal wall and prepuce.
- Bladder rupture also cause uroabdomen.
- This causes an obvious fluid swelling, which may spread as far as in the thorax.

Rupture of the bladder(Cystorrhexis)

- This results in a severe cellulitis and toxemia.
- The skin over the swollen area may slough, permitting drainage.
- A fluid wave is detectable on tactile percussion.
- Abdomen soon becomes distended.
- The animal may continue in this state for as long as 2-3 days before death occurs.

CLINICAL PATHOLOGY

- The urine usually contains erythrocytes and epithelial cells and highernumber of crystals.
- Bacteria may also be present if secondary invasion of the traumatic cystitis and pyelonephritis has occurred.
- Serum urea nitrogen and creatinine will be increased.

CLINICAL PATHOLOGY

- Rupture of the bladder will result in uroabdomen.
- Urine has a markedly low sodium and chloride concentration.
- Urine is of high osmolality in comparision to plasma.
- Intra and extra cellular fluid will move to abdomen leading to more enlargement.
- Abdominocentesis is necessary to detect uroperitoneum after rupture of the bladder.
- X ray and Ultrasonography to detect calculi.

NECROPSY FINDINGS

- Calculi may be found in the renal pelvis in those dying of renal calculi diseases.
- Pyelonephritis.
- Unilateral ureteral obstruction is usually accompanied by dilatation of the ureter and hydronephrosis.
- The urethra or urethral process may be obstructed by one or more stones.
- When rupture of the urethra has occurred the urethra is eroded at the site of obstruction with extensive cellulitis.

Differential Diagnosis

- Pylonephritis – urine culture for bacteria.
- Cystitis --frequent, painful urination and dysuria Pollakiuria : Painful dribbling of urine.

Diagnosis:

- Clinical findings
- Xray
- Ultrasonography
- Blood and urine examination
- Abdominocentesis
- Tactile percusdion of abdomen for uroabdomen.

Diagnosis

- Inspection of the ventral abdomen for edema.
- Inspection and palpation of the preputial orifice for crystals.
- Palpation of the penis in the area of the sigmoid flexure.
- Inspection and palpation of the urethral process of the exteriorized penis.

TREATMENT

- Recent studies suggest that administration of specific solutions into the bladder can rapidly dissolve most uroliths.
- Instillation of 30-200 mL of an acetic acid solution in the bladder of calves or cattle.
- Smooth muscle relaxants such as phenothiazin derivatives (Aminopromazine@ 0.7 mg/kg of BW)
- This relax the urethral muscle and permit passage of the obstructing calculus.

TREATMENT

- Normograde hydro pulsion is only occasionally successful.
- This technique involves catheterization of the urethral orifice with suitably sized urinary catheter.
- Intermittent injection of 0.9% NaCl into the urethra in an attempt to flush out the calculi.
- Surgical treatment includes perineal urethrostomy to relieve bladder pressur and for the removal of calculi.

NEPHRITIS

NEPHRITIS

Definition: Nephritis is the Inflamation of kidney

Types of Nephritis:

- Pyelonephritis
- Glomerulonephritis
- Interstitial Nephritis
- Nephrosis

Definition: Pyelonephritis is the bacterial inflammation of renal parenchyma and renal pelvis.

- Pyelonephritis develops most commonly from ascending infection through the lower urinary tract.
- It is characterized clinically by pyuria, dysuria or stranguria(Difficult dribbling), suppurative nephritis, cystitis and ureteritis.

Etiology

- In most cases it results following bacterial infection of the lower urinary tract.
- Spread from embolic nephritis of hematological origin (descending infection) such as septicemia of cattle caused by *Pseudomonas aeruginosa*.
- E.coli
- Specific pyelonephrit is caused by *Corynebacterium renale* in cattle
- *Eubacterium suis* in pigs is also recorded.

Etiology

- *Dioctophyma renale* is found in dogs, minks, cats, and other fish-eating mammals.
- The adult worms live in the renal pelvis, causing a suppurative pyelitis (inflammation of the pelvis).

Transmission

- Transmission is by direct contact (such as tail switching).
- Service by infected or contaminated bulls
- Poor urinary catheterization techniques.
- Poor management
- Urine deposit in ditches in cow house

Relationship of the ureter to the pelvis (or calyx)

- The dog and cat have a single calyx and renal papilla (uni pyramidal)
- The cow has multiple calyxes and papilla (multi pyramidal) with an external lobulated kidney.
- The pig kidney has multiple papillae and calyxes (multi pyramidal) without external lobulation.

Pathogenesis

- Tubulointerstitial nephritis is mostly hematogenous.
- Pyelonephritis usually results from an ascending UTI.
- Urinary obstruction (urolithiasis, prostatic hyperplasia) will lead to urinary stasis.
- This predisposes the animal to bacterial cystitis.
- Once an infection is established in the bladder, it may ascend up the ureters via vesico ureteral reflux.
- Once the renal pelvis is infected, bacteria gain access to the parenchyma via the collecting ducts.
- This is pyelonephritis

Pathogenesis

- In severe cases of reflux, bacteria present in urine, flows up into the tubules via the collecting ducts.
- This carry bacteria as far as the urinary space of glomeruli.
- This is intra renal reflux.

Pathogenesis

Development of pyelonephritis is depending up on:

- Common presence of infection in U.Tract.
- Stagnation of urine.
- Reflux of urine from the bladder.

Urine stasis can occur as a result of:

- Blocking of the ureters by inflammatory swelling or debris.
- Pressure from the uterus in pregnant females.
- Obstructive urolithiasis.

Pathogenesis

- Infection ascends the ureters and invades renal pelvis
- Extends to involve the medulla and cortex.
- Signs of toxemia and fever, usually accompany extensive bilateral infection.
- Uremia may develop if the lesions are sufficiently extensive.
- Pyelonephritis is always accompanied by hematuria. Due to:
- Inflammatory lesions of ureters and bladder.

Clinical findings

- First sign passage of blood stained urine in some cases.
- In other cases, the first sign may be an attack of colic.
- Swishing of the tail, kicking at the abdomen and treading(Specific way) of the feet.
- Straining to urinate.
- Such attacks are caused by obstruction of the ureter or renal calyx by pus or tissue debris.
- Pyelonephritis (along with toxic nephrosis) is one of the most common clinically recognized renal disorders of cattle.

Clinical findings

- The onset is gradual with fluctuating temperature (39.5º C).
- Capricious appetite, loss of condition and fall in milk yield over a period of weeks.
- The most obvious sign is the presence of pus, blood, mucous and tissue debris in the urine.
- Particularly at last portion of voided urine.
- Urination is frequent, painful and may occur in a dribble rather than a stream.

Clinical findings

- Rectal examination may be negative in the early stages.
- In later, thickening and contraction of bladder wall and enlargement of one or both ureters.
- kidney may show enlargement, absence of lobulation and pain on palpation.
- In many cases (chronic) there are no distinct clinical signs.
- Weight loss and in such cases urinalysis is important for diagnosis.

Clinical pathology

- Urine analysis for blood and protein.
- Urine pH is more than 8.5 (Alkaline).
- Urine specific gravity is 1.008-1.212.
- Microscopic examin - pyuria, debris, blood cells.
- Bacteriological culture- may be bacteria
- Proteinuria
- Seum creatinine--1.5 mg / dL and above
- Elevations of BUN -100 mg / dL.
- This carry a grave prognosis.

PM Findings

- The renal calyxes contain a purulent exudate and the lobules have a hyperemic or necrotic medulla.
- White streaks extend upward from the medulla into the cortex.
- This is an example of pyelonephritis.

Treatment

- General principles for treatment of U.T. infections.
- Acidification of urine by feeding monobasic sodium phosphate (100 gm daily for several days).
- Procain penicillin remains the antibiotic of choice in this cases. 15.000 IU / Kg B.Wt i/M for at least 5 weeks.
- Unilateral Nephroctomy may be carried out in valuable animals.
- Improving appetite, milk yield and clearing of urine expect good prognosis.

GLOMERULAR NEPHRITIS

- Glomerulonephritis is also known as glomerular nephritis.
- It is a term used for several kidney diseases (usually affecting both kidneys).
- It is characterised by inflammation either of the glomeruli or of the small blood vessels in the kidneys.

GLOMERULAR NEPHRITIS

- As it is not strictly a single disease, its presentation depends on the specific disease entity: it may be present in:
- hematuria
- proteinuria
- nephrotic syndrome
- acute kidney injury (ARF)
- chronic kidney disease (CRF)

Etiology

- Primary causes are intrinsic (Inherent an ddeep rooted) to the kidney.
- Secondary causes are associated with certain infections:
- Bacterial
- Viral or parasitic pathogens
- Drugs
- Systemic disorders

Pathogenesis

- A glomerulus, a functional unit that represents the first step in the filtration of blood and generation of urine.
- Glomerulonephritis refers to an inflammation of the glomerulus, which is the unit involved in filtration in the kidney.
- This inflammation typically results in one or both of the nephrotic or nephritic syndromes.

Pathogenesis

- Podocytes, cells which line the glomerulus, are negatively charged and have small gaps, preventing the filtration of large molecules.
- When damaged by inflammation, this can result in an increased permeability to proteins
- It allow only 4 nanogram particle to pass
- Inflammatory damage to cells lining the glomerulus destruct the epithelial barrier.
- This eading to blood being found in the urine.

Pathogenesis

- At the same time, reactive changes may result in a decreased kidney blood flow.
- This result in a decrease in the production of urine.
- The renin-angiotensin system(RAAS) is subsequently activated due to the decrease in perfusion of juxtaglomerular apparatu.
- Renin is secrete which convert angiotensinogen to angiotensin –I
- ACE convert angiotensin –I to angiotensin –II
- Angiotensin –II is a vasoconstrictor.

Pathogenesis

- Inflammation that affects the cells surrounding the glomerulus, podocytes, increases the permeability to proteins, resulting in proteinuria.
- With decreased proteins in the blood, there is a decrease in the oncotic pressure of the blood.
- This results in edema of the body.
- More recent studies have shown that extensive sodium retention in the distal nephron (collecting duct) is the predominant cause of water retention and edema in the nephrotic syndrome.

Pathogenesis

- Glomerulonephritis is an ucommon clinical renal disease in sheep and occasionally as a clinical entity in cattle.
- cattle are usually not examined until the disease process is advanced.
- **The three types are seen :**
- Spontaneous proliferative glomerulonephritis.
- Glomeoulonephritis of pregnancy toxemia in sheep.
- Mesangiocapillary glomerulonephritis.

Clinical and lab. Findings

- Weight loss and generalized edema.
- Marked oliguria or even anuria in some cases.
- Proteinuria, hypoalbuninemia and anemia.
- Serum Cr. And BUN is elevated in advanced cases.

Treatment

- Is usually unrewarding.
- Plasma transfusion.
- Anabolic steroids.
- Antibiotics can be given

Interstitial Nephritis

- **Interstitial nephritis** (or tubulo-**interstitial nephritis**) is a form of **nephritis** affecting the **interstitium** of the kidneys surrounding the tubules.
- **Interstitial** nephritis is a **kidney** condition characterized by swelling in between the **kidney** tubules.
- This disease can be either acute(occurs suddenly) or Chronic .
- It is ongoing and eventually ends in kidney failure.

Interstitial Nephritis

- Interstitium: It is the support tissue of **renal** parenchyma.
- In histology, it corresponds to the space between glomeruli, tubules, vessels and nerves.
- Medullary interstitium is the tissue surrounding the loop of Henle in the **renal** medulla.
- It functions in **renal** water reabsorption by building up a high hyper tonicity, which draws water out of the thin descending limb of the loop of Henle and the collecting duct system.

Interstitial Nephritis

- The **renal interstitium** is defined as the intertubular, extraglomerular, extravascular space of the **kidney**.
- It is bounded on all sides by tubular and vascular basement membranes.
- It is filled with cells, extracellular matrix, and **interstitial** fluid.

Interstitial Nephritis

- It is a common disease of the dog, it may be acute ,diffuse or chronic focal but always non-suppurative.
- Acute type is usually caused by leptospirosis in dogs.
- Chronic poisoning with caustic-treated roughages in cows leads to chronic form of this disease.

Interstitial Nephritis

- **Clinically is characterized by:**
- Polyuria with low specific gravity urine.
- Vomiting , polydepsia and depression.
- Arched back in severe cases and temperature may be elevated.

Treatment:

- Restoration of water and electrolyte balance is important.
- Parentral administration of antibiotics.

Nephrosis

- Nephrosis includes degenerative and inflammatory lesions of the renal tubules .
- Uremia may develop acutely or as terminal stages after a chronic illness.
- It is manifested by polyuria, dehydration and loss of weight.

Etiology

- Most cases of nephrosis caused by toxins.

Toxins:

- Mercuric compounds, selenium and organic copper compounds.
- Oxalates in plants and fungi.
- Thiabendazale anthelmintics.
- Over dose w sulphonamides.
- **Haemodynamic factors:**
- Dehydration leading to concentration of toxins in the tubules.
- Severe renal ischemia.
- Heamoglobinurea causing hemoglobinuric nephrosis.

Pathogenesis

- In acute nephrosis there is obstruction to glomerular filterate flow through the tubules.
- There is obstructive oliguria and uremia.
- In chronic cases there may be impairment of tubular reabsorption of solutes and fluids.

Clinical signs

- In acute stage there is oliguria, protinuria and other symptoms of uremia as:
- Anorexia, hypothermia, depression, weak pulse.
- In cow there is continuous mild hypocalcemia.
- Polyuria in chronic cases.

Diagnosis and Treatment

Diagnosis:

- Clinical signs.
- Clinical pathology.
- Necropsy findings.

Treatment:

- Correct the primary cause.
- Water and electrolytes therapy.
- Antibacterial agents.

DISEASES OF NERVOUS SYSTEM

Prepared by Prof. S. Haque

Dept. of veterinary clinical medicine

DISEASES OF NERVOUS SYSTEM

A. **Central nervous system consist of :**

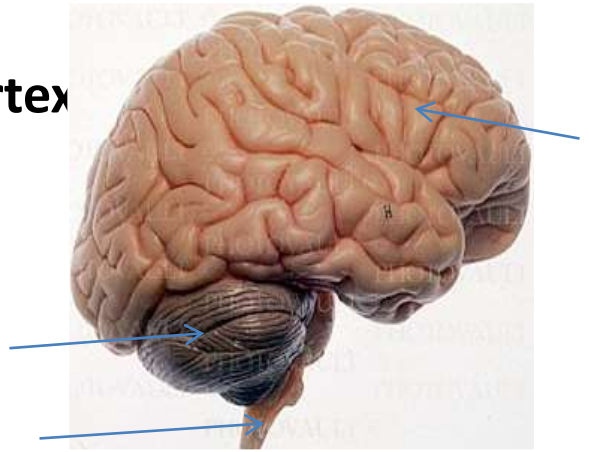
1 . Brain

2 . Spinal cord

Cerebral Cortex

cerebellum

Brain stem



B . Peripheral nervous system comprises of :

a . Efferent (Motors)

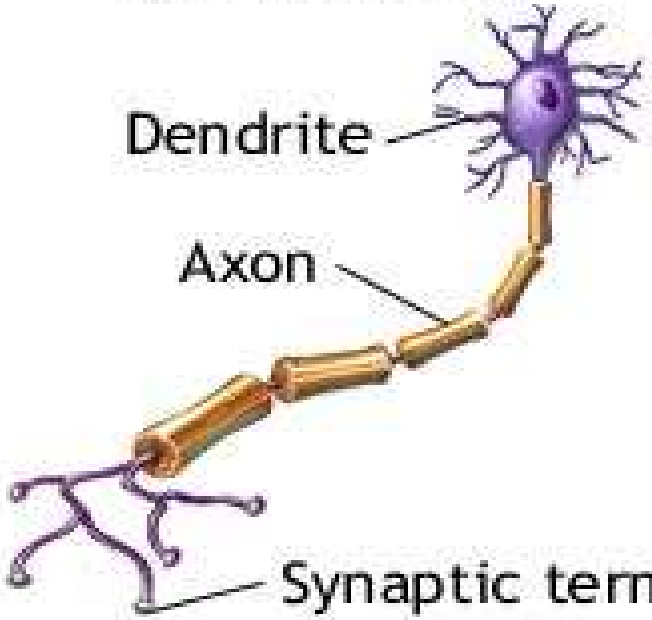
b. Afferent (sensory)

CEREBRAL HEMISPHERES

- It is made up of cerebral cortex and basal ganglia .
- Two Hemispheres are connected with Corpus Callosum.
- It is collectively called as cerebrum and is associated with consciousness .
- It constitute 90% of the brain structure .
- Cortex contain nearly 75% of neuron cell body.

THE NEURONS

Younger brain



Neurons in younger brain

ENCEPHALITIS

- Encephalitis is inflammation of brain.
- Inflammatory disease of the central nervous system (brain and spinal cord) is one of the most common causes of neurological disease in veterinary medicine.
- Encephalitis is more common in dogs and cats

ENCEPHALITIS

- The clinical diagnosis is named after the area of the nervous system that is most affected. For example,
- **“Encephalitis” refers to inflammation of the brain.**

  Encephalo = brain and

  itis = inflammation) .
- Encephalitis” is used as a general term for inflammatory diseases of the nervous system.

ENCEPHALITIS

- **Myelitis**”-- refers to inflammation of the spinal cord.
- If the meninges are primarily affected, the disease is called “**meningitis”**.

Encephalitis + Meningitis=**Meningoencephalitis**

- If the meninges, brain and spinal cord are all involved then the disease is called **“Meningoencephalomyelitis”.**

Classification

1. Infectious encephalitis
2. Idiopathic encephalitis

Infectious encephalitis is caused by :

i. Bacteria.
ii. Viruses.
iii. Parasites.
iv. Fungi.
v. Tick transmitted (rickettsial) diseases.

Classification

- **Idiopathic encephalitis**: In medical term it means encephalitis of unknown cause.
- It is diagnosed if an infectious cause cannot be found.
- **Idiopathic encephalitis** is the most common cause of inflammatory brain disease.

ETIOLOGY

IN all species of animals:

- **Sarcocystosis** – A protozoal disease of cattle , horses , sheep cats and dogs with a two host life cycle causing nervous disorders.

Viral infections :

i. Rabies

ii. Pseudorabies

iii. Japanese B encephalitis .

ETIOLOGY

Cattle : Baacterial infection

i. **Listeriosis.**

ii. **Haemophilus somnus** – a causative organism localises in several tissue and organ including nervous tissue causing menengoencephalitis, synovitis and pleuritis.

iii. **Heartwater** – It is a rickettsial disease caused by cowdria ruminantium in cattle sheep and goats characterised by fever , nervous sign , edema of body cavities and diarroea.

ETIOLOGY

Cattle (viral infection):

i. Bovine malignant catarrh .

ii. Infectious bovine rhinotracheitis virus.

Sheep (viral infection):

i. Louping ill –It si also called as ovine encephalomyelitis and is caused by virus.

ii. Scrapie – It is virus like disease of adult sheep causing nervous disorder including encephalitis.

iii. Visna –It is menengioencephalitis of sheep.

Horses : Viral infection

i. Infectious equine encephalomyelitis.

ii. Borna disease : It is a viral disease of horses transmitted by ticks (Hayaloma analoticum) and is characterised by fever ,halomyelitis, somnolence and paralysis.

iii. **West Nile virus (WNV)**: causes equine encephalomyelitis.

iv. **Eastern Equine Encephalitis(EEE) virus:**

This is commonly called as sleeping sickness which cause encephalitis in equine.

Dogs and cats :

- **Virus** – Canine distemper virus, feline infectious peritonitis virus and rabies virus

- Parasites – *Toxoplasma gondii and Neospora caninum*

- **Fungi** – *Cryptococcus neoformans and Coccidioidomyces immitis* (“Valley Fever”)- cause fever and lungs congestion.

- Tick transmitted disease –***Ehrlichia canis.***

  Lyme disease –(Borreliosis- skin rashes)

Etiology

Parasitic causes:

- Migration of parasitic larvae:
- **Hypoderma bovis and**
- **Oestrus ovis**

occasionally migrate to brain and spinal cord .

Pathogenesis

- Entrance of the virus to the nervous tissue occur in several ways.
- Normally blood-brain barrier is the filtering agent and also protect the brain by not allowing the bacteria or viruses to enter.
- But when there is damage to endothelium, infection readilly occurs .

eg. **Tick born fever (EEE) and louping ill .**

Pathogenesis

- Entry of the agent to the brain may also occur by the progression of the virus along with nerve trunk:

  eg. Rabies and seudorabies.

- Entry via the olfactry nerve is also possible.
- The clinical sign of encephalitis appear due to stimulatory, inflamatory and lethel effect on the nerve cells (neurons).
- In case of listeriosis lesion is in pons-medulla.

Clinical symptoms

1. There may be initial exitment or mania.
2. Mental depression including head pressing.
3. If irritation persist it include the symptoms of:
    i. **Colonic convulsion**
    ii. **Nystagmus**
    iii. **Champing of the jaw**
    iv. **Exessive frothy salivation**
    v. **Muscles tremor specially of face and limb.**

Clinical symptoms

4. Loss of nervous function which include :
   - **i.** Paresis
   - ii. Knuckling of lower limb joints .
   - iii. Spastiticity of limbs.
   - iv. Ataxia.
   - v. There may be complete paralysis.
   - vi. Deviation of head
   - vii. Walking in circle.

Clinical symptoms

- Clinical signs of encephalitis usually reflect the area of the brain that is affected.
- Seizures, blindness, behavior changes, mental depression and circling are seen with forebrain (cerebral) diseases.

- Imbalance or incoordination, head tilt, tremors or facial paralysis are associated with brainstem disease.

Clinical symptoms

- If the neurologic abnormalities are focal, it may be brain tumor.
- Most animals with encephalitis will develop neurologic abnormalities over a short period of time (days to weeks).

Clinical Pathology

- Clinical pathology may be of the considerable assistance in the diagnosis of encephalitis.
- Test and techniques are different as per the specific diseases.

CBC especially DLC will be helpful for diagnosis.

- The brain and spinal cord are surrounded by CSF and it reflects what is going on inside CNS.
- A significant increase in white blood cells and proteins in CSF usually indicates encephalitis.

Diagnosis

- Diagnosis of encephalitis cannot depend entirely on recognition of the tipical syndrome.
- Because simillar symptoms may be exibitted by many other brain diseases.
- Magnetic Resonance Imaging (MRI) helps to differentiate between encephalitis and other causes of neurologic disease.
- Certain brain tumors can cause inflammation in spinal fluid and thereby “mimic” encephalitis.

Treatment

- Once an animal has been diagnosed with encephalitis, antibiotics are usually started to treat for the common infectious diseases. Like:
- Cefotaxim@10mg/kg body wt. parentrally.
- If the animal has seizures, anticonvulsants like Phenobarbital @ 2mg/ kg body wt orally.
- Low doses of steroids is given in inflammation in the CSF or in severe clinical signs.
- Dexamethasone@0.5mg/kg BW= 2weeks in tapering doses.

Treatment

- The treatment for idiopathic encephalitis usually involves suppression of the immune system.
- This is in clear opposition to the treatment for infectious encephalitis.
- It is important that infectious diseases be ruled out before starting immuno suppression.
- High dose therapy with the steroid (I/M)only like Prednisone @ 2mg/kg body wt. is the most common treatment for idiopathic encephalitis

MANINGITIS

Prof. S. Haque

Dept. of veterinary clinical medicine

ETIOLOGY

Cattle:

- Viral diseases - Bovine malignant catarrh, sporadic bovine encephalomyelitis.
- Bacterial diseases - listeriosis, H. somni, chronic lesions elsewhere in the body.

Horses:

- Strangles, Pasteurella haemolytica (also donkeys and mules), Streptococcus suis, Streptococcus equi.

ETIOLOGY

Sheep:

- Melioidosis, Streptococcus aureus (tick pyemia) in newborn lambs .
- Pasteurella multocida in lambs
- Mannhemia (Pasteurella) haemolytica in lambs.

Pigs:

- Glasser's disease(Haemophilus parasuis) erysipelas, salmonellosis; S. suis type- 2 in weaned and feeder pigs.

ETIOLOGY

- Streptococcal and coliform septicemias are probably the commonest causes of meningitis in neonatal farm animals.
- Hematogenous infection occurs from other sites also.

Calf –

- E. coli. The disease occurs most commonly in calves under several days of age and can occur in less than 24 hours after birth.

ETIOLOGY

- *E. coli* is the main bacterial pathogen associated with septicemia in calves, *Salmonella, Campylobacter, Klebsiella* and different *Staphylococcus* species have also been isolated from the blood of septic calves.

PATHOGENESIS

- The integrity of blood brain barrier(BBB) is due to the presence of tight junctions between the cerebral endothelial cells forming the BBB and between the blood and choroid plexus epithelial cells (blood- CSF barrier)
- The presence of endothelial cell tight junctions and basement membrane also restricts entry of certain therapeutic agents into the CSF and CNS.

PATHOGENESIS

- During CNS inflammation both microglial cells and brain endothelial cells cause histocompatability complex (MHC) class II and thus activate T lymphocytes.
- These activated T cells, microglial and endothelial cells release cytokines which increase permeability of the BBB .
- Cytokines increase permeability of the BBB.
- Now the bacteria can enter.

PATHOGENESIS

- Exactly how bacteria get into the meninges is poorly understood.
- Sustained and high grade bacteremia in the highly perfused dural venous system and choroid plexuses, adherence of fimbriae from some strains of E. coli.
- Phagocytosis of pathogens by circulating monocytes and endocytosis(protein transport) through microvascular endothelial cells.

PATHOGENESIS

- Inflammation of the meninges causes local swelling and interference with blood supply to the brain and spinal cord
- As a rule penetration of the inflammation along blood vessels and into nervous tissue is of minor importance and causes only superficial encephalitis.

PATHOGENESIS

- Pyogenic bacteria often cause a fatal choroiditis(inflammation of the retina and choroid) with exudation into CSF.
- The signs produced by meningitis are thus a combination of those resulting from irritation of both central and peripheral nervous systems.

PATHOGENESIS

- Spinal meningitis: Muscular spasm with rigidity of the limbs and neck, arching of the back and hyperesthesia with pain on light touching of skin.
- Cerebral meninges: Irritation signs, including muscle tremor and convulsions are common manifestations.
- Since meningitis is usually bacterial in origin, fever and toxemia can be expected if the lesion is sufficiently extensive.

PATHOGENESIS

- Defects of drainage of CSF occur in both acute and chronic inflammation of the meninges.
- Produce signs of increased intracranial pressure(vomiting without nausea and altered level of consciousness,.
- The signs are general although the accumulation of fluid may be localized to particular sites such as the lateral ventricles.

CLINICAL FINDING

- Calves may have an extended head and neck and attempts to flex and thrashing of the limbs.
- Calves with meningitis almost always have abnormal mentation.
- As the disease progresses, the animal becomes comatose and non responsive and may develop seizures.

CLINICAL FINDING

- Blindness is common in cerebral meningitis but not a constant clinical finding.
- In young animals, ophthalmitis.
- The pupillary light reflex is usually much slower than normal.
- Examination of the fundus of the eyes may reveal evidence of:
- Optic disk edema
- Congestion of retinal vessels and exudation.

CLINICAL PATHOLOGY

Cerebrospinal fluid:

- CSF collected from the lumbosacral space or cisterna magna in meningitis contains:
- Elevated protein concentrations, has a high cell count and usually contains bacteria.

Hematology:

- The hemogram usually reveals a marked leukocytosis, reflecting the severity of the systemic illness secondary to septicemia.

NECROPSY FINDINGS

- The CSF is often turbid and may contain fibrin.
- A local superficial encephalitis is commonly present.
- Hyperemia, the presence of hemorrhages, and thickening.
- opacity of the meninges, especially over the base of the brain

DIFFERENTIAL DIAGNOSIS

- It is often difficult to differentiate meningitis from encephalitis and acute cerebral edema.
- Examination of the CSF is the only means of confirming the diagnosis before death.
- Analysis of CSF is very useful in the differential diagnosis of diseases of the nervous system of ruminants.

TREATMENT

- The infection is usually bacterial, and parenteral treatment with antimicrobial.
- Presumably, the blood-brain and blood-CSF barriers are not intact in meningitis:
- Penicillincillin With streptomycin
- Ampicillin
- Cefotaxim
- ceftriaxone

Control and Prevention

- Calf should receive 3-4 litres of colostrum in the first six hours after birth.
- Followed by similar volume in the next 12- 18 h.
- One of the main portals of entry of bacteria into the calf's bloodstream after birth is via the navel.
- Concentrated iodine solution --applied
- Chlorhexidine-based dip or OTC spray twice.
- Once immediately after birth and also 12 h later.

ENCEPHALOMALACIA

Prof. S. Haque

Dept. of veterinary clinical medicine

ENCEPHALOMALACIA

Def: By definition encephalomalacia means softening of the brain tissue.

- The degenerative diseases of brain are grouped together under the name of encephalomalacia.

It is of two type :

- LEUKOENCEPHALOMALACIA --- SOFTENING OF WHITE MATTER.
- POLIOENCEPHALOMALACIA --- SOFTENING OF GREY MATTER

ETIOLOGY

Ruminants: In ruminants it is caused by diferrent etiological factors which are as follow:

- Plants poisons e.g. , Astragalus sps. , Oxytropis sps., Vicia sps. and Kochia scoparia.
- Residual lesion after intoxication with Clostridium perfringes type D in sheep.
- Thiamin deficiency in cattle and sheep.
- Mannosidosis (**Mannosidosis** is a lethal disorder linked to genes and nervous disorder also.
- Swayback and enzootic ataxia due to nutritional deficiency of copper in lambs.
- Due to prolonged parturition in calves.
- Acute lead poisoning
- Sodium toxicosis
- Water deprivation
- Sulpher intoxication

PATHOGENESIS

- Pathogenesis of LEUKOENCEPHALOMALACIA is unknown but it is assumed that it might be due to endothelial injury .
- POLIOENCEPHALOMALACIA appears to be a consequence of acute edematous swelling of the brain and cortical ischaemia.
- In Swayback disease there is defective demyelination.
- In newborn calves and foals there is cavitation in brain with loss pf neurons and axons .
- Whether the lesion is in the grey matter or in the white matter the syndrome is loss of function.
- Irritation signs is more when grey matter is damaged.

PATHOGENESIS

- Ruminants depend on dietary thiamine.
- In adult ruminants, thiamine is produced by rumen microbes.
- Thiamine inadequacy can be caused by decreased production by rumen microbes
- Factors that interfere with the action of thiamine, eg, plant thiaminases.
- Thiaminases can be produced by gut bacteria or ingested as preformed plant products.
- They can either destroy thiamine or form antimetabolites that interfere with thiamine function.
- Sulpher poduce Hydrogen sulfide (H_2S) gas, which has the odor of rotten eggs and destroy R/microflora.

CLINICAL FINDINGS

- Paralysis is accompanied by dullness, somnolence , blindness, ataxia , head pressing , circling with terminal coma.
- Irritation signs including muscle tremor , opisthotonus , nystagmus and convulsion.
- Blindness followed by recumbency, tonic-clonic seizures, and coma.

CLINICAL FINDINGS

- The head is held in an elevated position.
- There is cortical blindness with absent menace response but normal bilateral pupillary light reflex.
- Dorsomedial strabismus may develop.
- Animals may show ataxia and sometimes a hypermetric gait.

CLINCAL PATHOLOGY

- There is no specific clinicopathological test .
- Test for specific diseases are required .

DIAGNOSIS

- The onset is quite sudden and there is depression of consciousness and loss of motor function.
- It resemble clinically most of the diseases of brain increasing intracranial pressure
- It is non progressive and the affected animal may survive for longer time in comparision to other brain diseases.

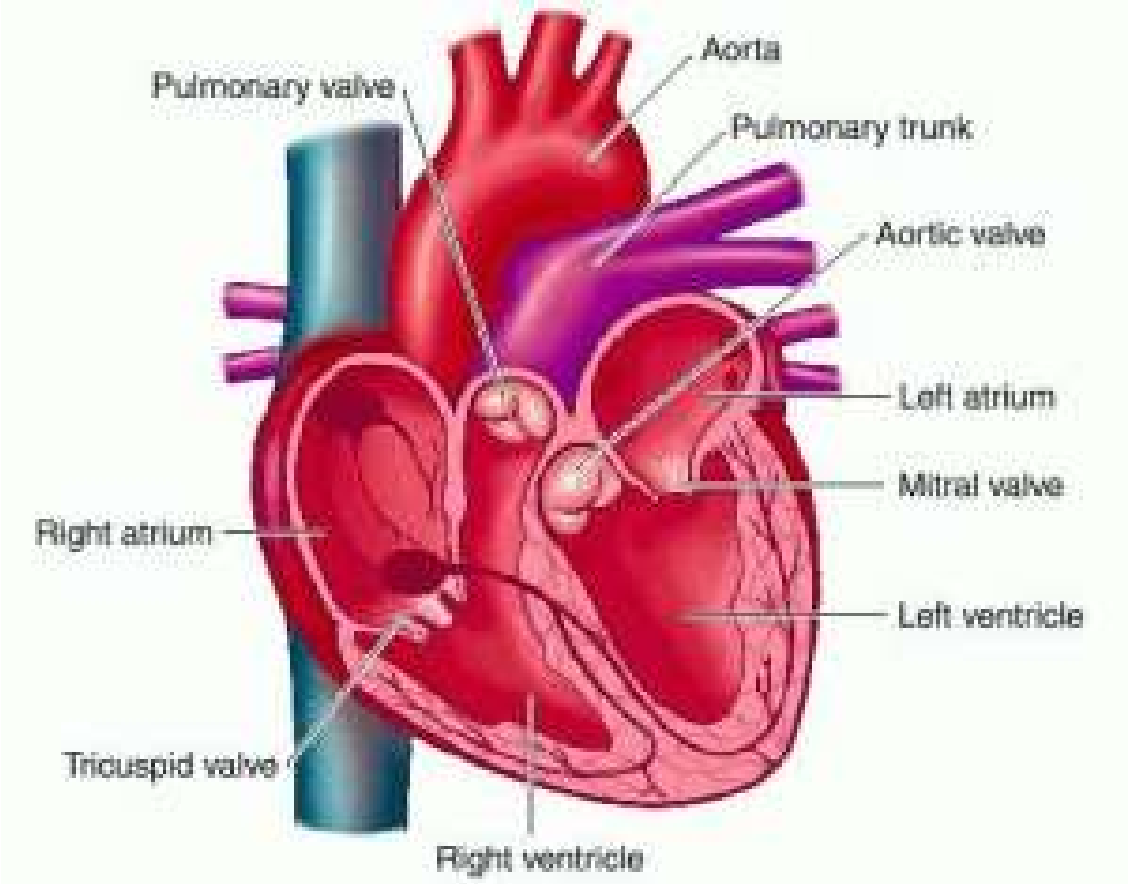
Differential Diagnoses

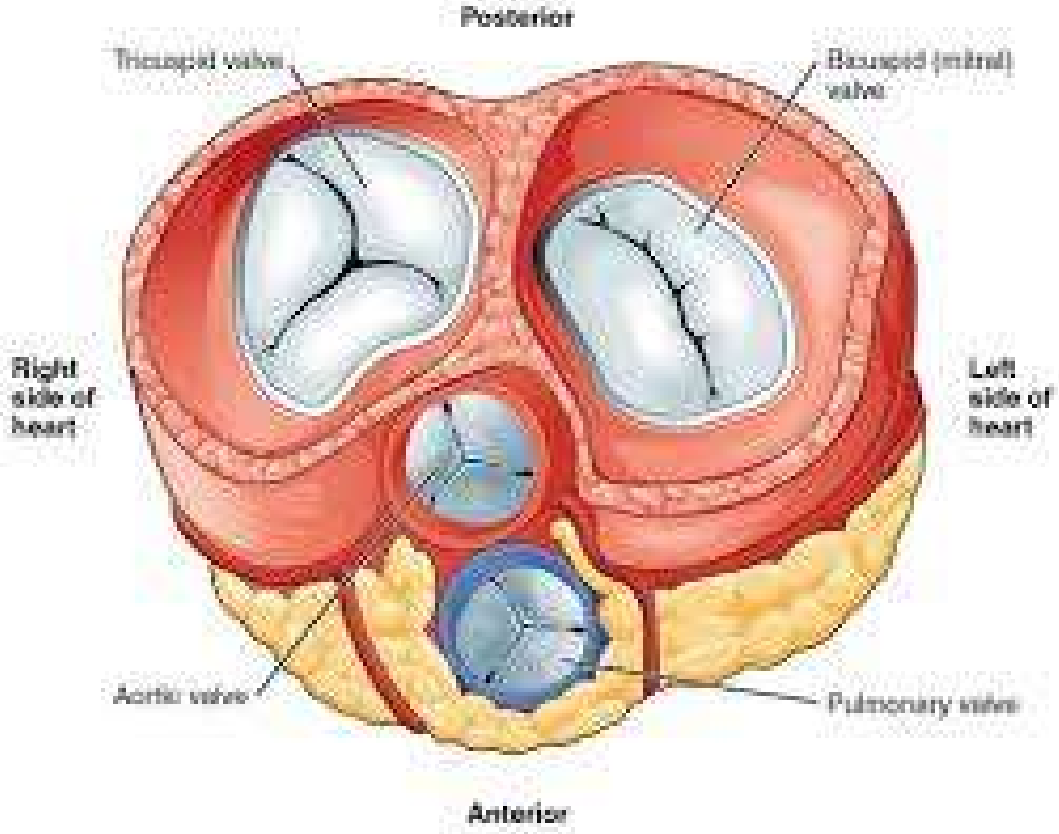
- For cattle include acute lead poisoning, water deprivation/sodium toxicosis
- *Histophilus* meningoencephalitis
- Rabies with nervous involvement
- vitamin A deficiency.
- Differential diagnoses for sheep include pregnancy toxemia, type D clostridial enterotoxemia and listeriosis.

TREATMENT

Specific diseases can be treated with specific drugs .

- Encephalomalacia is irreversible .
- Thiamin administration @ of 10 mg/kg tid for cattle or small ruminants for 3 days –I/M or I/V.
- Reduction of cerebral edema can be treated with dexamethasone at a dosage of 1–2 mg/kg, IM or SC.
- Anticonvulsants -Phenobarbital @ 2mg/ kg b wt orally.
- Fluid therapy
- Dietary supplementation of thiamine at 3–10 mg/kg feed has been recommended for prevention.


Aorta
Pulmonary valve
Pulmonary trunk
Aortic valve
Left atrium
Mitral valve
Right atrium
Left ventricle
Tricuspid valve
Right ventricle


Posterior
Tricuspid valve
Bicuspid (mitral) valve
Right side of heart
Left side of heart
Aortic valve
Pulmonary valve
Anterior

History and Physical Examination

1. The **history** and **physical examination** remain the **most crucial steps** in establishing the correct diagnosis.
2. Findings from a careful history and physical examination **give an idea** about the **presence of heart disease**.
3. Results of the cardiovascular physical examination will usually **allow the clinician** to make a **tentative diagnosis**.

Common presenting complaints for cardiac disease

- Coughing
- Dyspnea (difficulty in respiration)
- Syncope (fainting/unconsciousness)
- Ascites (fluid accumulation in abdomen)
- Cyanosis (bluish color mucous membrane)
- Hemoptysis (blood in cough)
- Weakness and exercise intolerance (tiredness due to walking/running)
- Weight loss

Terminology

- **Preload**: The volume of blood within ventricle **just before it contracts** is termed the preload.
- **Afterload:** the **resistance** to ejection of blood "perceived" by the **left ventricle** is termed the afterload (Peak tension in the wall of ventricle).
- **Systolic Function**: an adequate amount of blood must be pumped out of the heart with every beat to perfuse the peripheral tissues and meet the metabolic needs of the body.
- **Diastolic Function:** Normal diastolic function is the ability of the heart to fill sufficiently at regular filling pressures.
- **Systolic dysfunction:** is characterised by **impaired pumping ability** and reduced ejection fraction (amount of blood ejected out by heart, N=50-65%).

Heart failure

- Heart failure is a clinical syndrome that occurs secondary to severe, overwhelming cardiac disease.
- It occurs because the heart is no longer able to maintain normal cardiac output.
- Initial changes in **heart chamber volume** or **wall thickness** are best understood in relation to **preload** and **afterload**.

- **Clinical Signs based on location of cardiac chamber failure:**
- **Left Heart Failure**: Left atrial pressure increases as left heart diseases becomes severe eg, in MVD, regurgitant blood flow creates a **back pressure on pulmonary veins**, and **pulmonary capillaries** leading to **leakage (transudation) of fluids** out of the capillaries into the lung tissue causing **pulmonary edema**. This pulmonary edema causes **tachypnea**, **dyspnea** and **coughing**.

- **Right Heart Failure**: The **right atrium** receives venous return via the **cranial and caudal vena cavae.** Right atrial pressure increases in right heart disease.
- **Clinical manifestations** of right heart failure include **jugular venous distension**, **hepatomegaly, ascites**, and peripheral edema.
- Management

1. Diuretics: **Furosemide** is the most widely used

- Dogs @ 1–2 mg/kg, bid
- Cats @ 0.5–2 mg/kg every 12–24 hr
- Cattle @ 2.2-4.4 mg/kg IV bid; Horses @ 1-2 mg/kg IM-IV bid

2. Positive Inotropes: **Pimobendan** (increases heart contraction) @ **0.2–0.3 mg/kg, PO, bid**, in dogs.

3. **Enalapril :**

- Dogs @ **0.5 mg/kg, PO, bid.**
- Cats @ **0.5 mg/kg/day.**

Nutritional Considerations:

1. Salt (Sodium) restriction
2. Supplementation with **omega-3 fatty acids**
3. **Taurine** supplementation especially in cats
4. **L-Carnitine**: in **Boxer dogs** plays an important role in fatty acid metabolism and energy production.
5. **Coenzyme Q10:** possesses general **antioxidant properties.**

Pericarditis: Inflammations of **pericardium** characterized by **chest pain**, **abduction of elbow**, **difficult respiration**, **muffled heart sound**, **edema of brisket region** & finally death due to congestive heart failure & toxemia.

- **Etiology**: primary due to the bacterial infection & secondary due to the other diseases.

1. Route of infection- It may be hematogenous as occurs in septicaemia and other specific diseases like H.S, B.Q, Strangle or pneumonia.
2. It may be also occur due to the inflammatory process of neighboring tissue like myocardium, pleura and mediastinum lymph node as seen in diseases like T.B & CBPP.
3. It may be traumatic in origin like traumatic pericarditis.

Pericarditis is also found as apart of following diseases in different animals:

- **Cattle**: - Pasterullorisis ,CBPP, T.B , Mycoplasma , Pseudomonas , Haemophilus inf. Coliform infection of new born.
- **Horses**: - Complication of strangle, influenza, T.B.
- **Sheep** :- H.S, Mycoplasma infection & Salmonella infection .
- **Swine** :- Erysipelas, Hog cholera, H. S., Haemophilus & streptococcal infection.
- **Dogs**:- Secondary complication of C.D, Parvoviral infection , Leptospira infection

Pathogenesis

- Hyperemia in the pericardium leads to the deposition of fibrous exudate between pericardium & epicardium which produces **frictional rubbing sound** when pericardium & epicardium rub together during cardiac movement. It may also cause accumulation of fluid within the pericardium causing muffling heart sound leading to cardiac tamponade.
- Severe toxaemia may be present in cases of suppurative pericarditis.

CLINICAL FINDING

- Pain over the pericardium
- Restricted movement, abduction of elbow,
- Arching of back
- Shallow abdominal respiration
- Muffled heart sound
- Edema of brisket region
- Increase in pulse rate , well marked jugular pulsation &
- Finally the animal die of congestive heart failure & toxemia .

DIAGNOSIS

- Based on clinical sign & history.
- Auscultation of heart: during early stage **friction rub sound** of heart followed by **muffling** of heart sound.
- On palpation & percussion of cardiac area, animal exhibits a sign of **pain** .
- **Bamboo Test**: Monitor the heart rate of animal. Apply a bamboo to raise it by single thrust. If HR is increased that indicates some foreign body in cardiac area and there will be a grunt sound produced by the animal which is auscultated over the trachea simultaneously.
-

TREATMENT

- ✓ Antimicrobial therapy i.e. Broad spectrum antibiotic
- Oxytetracycline: 5-10 mg/kg
- Streptopenicillin: 5-10mg/kg
- ✓ If infection is brought under control, drainage of pericardial sac also be done to provide temporary relief to the animal.

Traumatic Pericarditis (TP)

- Perforation of **pericardial sac by sharp foreign body** originating from reticulum causes pericarditis which is characterized by toxemia, tachycardia, fever, engorgement of jugular vein, anasarca, hydrothorax & abnormal heart sound.
- **Etiology**: As TRP (traumatic reticulo peritonitis), most cases occur in the advanced pregnancy or after parturition. About 8% of TRP cases turn to be traumatic pericarditis.

Pathogenesis

- **TP** occurs in the cattle due to the ingestion of sharp foreign body particularly metallic in nature. Sharp penetrating metallic **foreign body gets settled** initially in the **ventral sac** of the rumen. Due to rumino-reticular **motility**, it may **penetrate the rumen and reticulum anteriorly** followed by penetration of **diaphragm** which may later advance to **penetrate the pericardium** resulting into pericarditis **known** as **traumatic pericarditis** (**TP**). Since foreign body may also carry infection and can lead to toxemia or their may be accumulation of fluid within pericardial sac leading to heart failure and finally death.

Clinical signs

- Serve depression due to toxemia
- **Complete anorexia** , rapid weight loss
- **Abduction of elbow**
- Prominent **jugular pulsation (pulse occurs >1/3rd length of Jugular Vein)**
- **Brisket edema**, extruding up to the ventral abdominal wall
- Increased respiration rate & respiration is more abdominal.
- On auscultation, heart sound may be normal (initially) or muffled (later, due to fluid accumulation).

Diagnosis

Clinical examination: clinical signs are very much evident

- Withers test and bamboo test to detect pain in the anterior region.

Laboratory examination: CBC-Increased **TLC (initially few days only)** with severe Neutrophillia shift to left.

X-ray examination which reveal the presence of some foreign in the cardiac area.

TREATMENT

- Not satisfactory
- Some cases may respond heavy doses of broad spectrum antibiotic or sulphonamide for long time.
- Drainage of pericardial sac gives temporary relief.
- In some cases pericardiotomy can be done with variable success.

Prevention: feeding of magnet to localize in the reticulum.

ANAEMIA

- Anemia may be defined as the decrease in quantity of hemoglobin and number of erythrocytes or both, per unit volume of blood, clinically characterized by pale mucous membrane, tachycardia, and muscle weakness.

Classification of anemia

1. On the basis of Etiology
2. On the basis of Morphological features

On basis of etiology, anemia can be of three types:

1. Hemorrhagic anemia.
2. Haemolytic anemia.
3. Dyshaemopoietic anemia.

- **Acute hemorrhagic anemia**: Occurs due to severe trauma, physical injury, accident, epistaxis (nasal bleeding), hemoptysis (cough with blood), surgical bleeding, excessive bleeding during parturition or poisoning like sweet clover poisoning.
- **Chronic hemorrhagic anemia**:

a) Due to **heavy ectoparasitic infestation** because they suck blood e.g ticks, fleas or lice etc.

b) Endoparasitic infestation or due to **blood sucking parasites** e.g Hemonchus, Bunostomum, Ancylostomiasis in dog.

c) Malabsorption and **hypoproteinemia** causing parasites e.g Fascioliasis, Amphistomiasis, Trichostrongylosis and ascariasis.

d) Coccidiosis in young animals causes chronic haemorrhages in small intestine.

e) Enzootic Bovine Haematuria occurs due to ingestion of **bracken fern**, which causes tumor formation in bladder, and passing of blood in urine.

2. Hemolytic anemia: It occurs due to destruction of RBC's in various diseases, may be due to blood parasites, bacterial, viral, chemical agents, poisonous plants and metals.

1. Bacterial disease:

- *Leptospirosis* in dogs & cattle
- *Clostridium hemolyticum* infection

2. Viral disease:

Equine infectious anaemia.

3. Protozoan disease:

Babesiosis in all species (**COFEE coloured urine in cattle**)

Anaplosmosis & Thelariosis in ruminants.

4. Post parturient hemoglobinuria (PPH) or nutritional hemoglobinuria because of deficiency of phosphorous.

5. Cu poisoning in sheep

6. Phenothiazine therapy in horses

7. Ingestion of excessive cold water intake in calves.

- **3. Dyshemopoietic anemia:** It occurs due to depression of bone marrow & decreased erythropiosis either due to nutritional or aplastic causes.
- **(A) Nutritional causes**: Most common cause of anemia in animal is nutritional anemia e.g. Fe, Cu & Co deficiency or Vitamins deficiency:
- **Iron deficiency -** with exception of anaemia in piglets, iron deficiency anemia rarely occurs. However, may occur in animals grazing on pastures deficient in iron. Piglet anaemia occurs because sows milk is poor in Fe content and Fe absorption from piglet intestine is very less & requirement of iron in young growing piglet is more.
- **Vitamins deficiency**
- a) **Pyridoxine** (B6) - mainly occurs in calves. It helps in utilization of iron in the synthesis of haemoglobin.
- b) **Nicotinic acid -** Restriction of niacin may decrease folic acid synthesis especially in dogs.
- c) **Riboflavin deficiency-** B12 is needed in metabolism of erythrocytes. It rarely occurs in animals

- **(B) Aplastic anemia :** Indicates decreased erythropoiesis due to depression of bone marrow and conditions are :
- 1. **Nephritis**: Uremia produced in nephritis has got depressing effect on bone marrow(↓ **Erythropoietin**). Similarly in advance cases of chronic interstitial nephritis.
- 2. **Neoplastic disease**: Bone marrow is replaced by neoplasm or may be aplasia of bone marrow
- 3. Rapid **exposure** to **X-Rays** or **radioactive** substances depresses the bone marrow.
- 4. **Drugs toxicity**: Continuous use of sulphonamides, Chloramphenicol depresses the bone marrow.

On basis of morphology, anemia can be of following types:

1. **Macrocytic Normochromic Anaemia:**

This types of anemia denotes the presence of **immature erythrocytes** in the blood. It is due to def. of **Vitamin B12 and Folic acid** deficiency as these are necessary for synthesis of RNA & DNA. It can be due to **cobalt** deficiency in **ruminants.**

2. Macrocytic Hypochromic anemia: Occurs in **regenerative phase** after hemorrhages. It may be due to various reasons like

a) Hemorrhages after injury or coagulation defects

b) Massive destruction of RBC's due to protozoan infection or drug toxicity

3. Normocytic normochromic anemia: Also called as **aplastic anemia** or **hypoplastic anemia**. Occurs in acute or sub acute systemic disease due to suppression of bone marrow activity.

4. Microcytic hypochromic anemia: It is the **iron deficiency** anemia

1. Iron deficiency may be due to
   a) Lack of dietary iron (**Fe**)
   b) Chronic blood loss
2. Defective utilization of iron stores as occurs in deficiency of Cu (as Cu acts as catalysts in utilization of iron in Hb formation), pyridoxine and riboflavin.

PATHOGENESIS

- Because there is decreased concentration of haemoglobin, **oxygen carrying capacity of blood is decreased** so the tissue suffers from hypoxia/anoxia. Skeleton and cardiac tissue suffer maximum because there demand for oxygen is more than other tissues.
  - In **acute hemorrhagic anaemia** there is loss of circulatory blood volume and plasma. Fluid loss is quickly repaired by equilibrium from tissue fluid and evacuation of blood stored in **spleen** and **liver**. Plasma proteins are also restored by synthesis in liver. However, erythropoisis require long time to remove the anemia.
  - **Hemolytic anemia** generally leads to haemoglobinuria and may cause nephrosis and depression of renal function and may lead to development of uremia.

- **CLINICAL SIGNS**: **Paleness** of visible mucus membrane, **Tachycardia** and increased intensity of heart sounds, Blood is **thin** and **watery**, **laboured** breathing due to hypoxia/anoxia.
- Additional sign accompanying anemia may be **Jaundice**, **hemoglobinuria** and **hematuria**.
- Clinical signs do not appear **until level falls to 50% of normal** and about 25% level is fatal.
- **Jugular pulsation** is evident in sever case of anemia.

- **Diagnosis:** based on history and clinical signs

TREATMENT

1) Treatment of primary cause is essential

2) Blood transfusion is indicated in severe cases, e.g. severe hemolytic anemia.

- Amount of blood collected 10-15 ml/Kg body weight I/V in emergency.

3) Injectable hematinics and Oral Haamatinic can be given ($FeSo_4$: $CuSo_4$: $CoSo_{4)}$

4) Preparation of Co and Cu

Arterial Thrombosis

- A thrombus is a **localized** or **generalized** intravascular or intracardiac blood clot (Thrombosis is the method of formation of a thrombus).

✓ Thrombi may occur in **any vasculature** in the body and form as the result of **trauma** or **pathological processes** affecting the blood vessel endothelium.

✓ Some diseases such as infective endocarditis and heart worm increase the risk of thrombi formation.

✓ It may occur due to **endothelial injury**, altered blood flow e.g. abnormal stasis or **hypercoagulability**.

✓ Thrombosis is often associated with other disease processes such as disseminated intravascular coagulation **(DIC).**

✓ Spontaneous venous thrombosis is rare, but can be seen in cattle with traumatic reticulo-peritonitis in the caudal vena cava.

Types

- **Arterial thrombosis**
  - ✓Arterial thrombosis is common in man, but it is uncommon in domestic animals.
  - ✓Verminous arteritis may occur with or without aneurysm and is seen in horses as a result of Strongylus vulgaris infestation (root of cranial mesenteric artery, renal artery and aorta as a consequence of larval migration in the vessel walls)
- **Cardiac thrombosis**
  - ✓ Cardiac thrombosis is usually valvular, but can occasionally be mural.
  - ✓ In farm animals, and rarely in the horse, infective/inflammatory thrombosis occurs subsequent to endocarditis. In dogs and horses, cardiac thrombosis is generally of degenerative/non-infectious cause; endocarditis may occur, though uncommonly.

- **Venous thrombosis**
  - ✓ Venous thrombosis is a fairly common type of thrombus in the veterinary species because veins are relatively thin-walled and are therefore more susceptible to distortion, inflammatory damage and iatrogenic venepuncture damage.
  - ✓ Also, veins have relatively slower blood flow rates allowing cell aggregates to persist more readily.
  - ✓ Most venous thrombosis in domestic animals results from extension of inflammatory reactions, erosion/disruption caused by malignant tumours, pressure from adjacent space-occupying masses or venepuncture damage.
- **Capillary thrombosis**
  - ✓ Capillary thromboses are microthrombi that are only appreciable histologically.
- **Signalment**: Can occur in any specie of animal, but is most commonly described in **dogs** and **cats.**

- **Clinical Signs**
  - ✓ Signs depend on the area affected and the size of the vessel blocked.
  - ✓ There will always be poor perfusion below the affected area, or **malfunction** and **necrosis** of the affected organs.
  - ✓ The area affected will often appear **pale**, **cold**, have **poor perfusion** and be **painful** to touch.
- **Diagnosis**

✓ History and clinical signs can be indicative of the condition.

✓ Ultrasonography may show blood stasis and may also demonstrate the presence of a thrombus.

✓ Angiography may show lack of opacity in the affected region.

✓ On microscopic examination, the thrombus appears as a layered mass which is attached to the vessel wall. The composition consists of red blood cells, neutrophils and platelets bound together by fibrin.

Treatment

- ✓ The mainstay of treatment is to diagnose and treat the underlying problem.
- ✓ It is important to give **pain relief** and **IV Fluids** as palliative care.
- ✓ The use of an **anticoagulant** such as **heparin** for short-term treatment or **aspirin** for long-term treatment should be considered.

Edema

Edema results from **four causes**:

1. Increased **hydrostatic pressure** in capillaries and veins due to chronic (congestive) heart failure or obstruction to venous return e.g heart failure, udder edema, local edema by compressive lesions etc.

2. Decreased **plasma oncotic pressure e.g** chronic blood loss by endoparasites- strongylus, hemonchus; Johne's disease and amyloidosis etc.

3. Increased **capillary permeability** in endotoxemia, part of the allergic response, vasculitis and damage to the vascular endothelium e.g endotoxemia

4. **Obstruction** to **lymphatic flow e.g** lymphangitis, tumor etc.

PATHOGENESIS

- Edema is the excessive accumulation of fluid in the interstitial space of tissue caused by a **disturbance in the mechanism of fluid interchange** between capillaries, the interstitial space and the lymphatic vessels.
- It can also occur where there is increased **vascular permeability** due to vascular damage.
- These processes can lead to pleural and pericardial effusion, pulmonary edema

TREATMENT (Aimed at correcting the cause)

- Hypoalbuminemia may require the administration of **plasma or plasma substitutes**.
- Parasitic gastroenteritis requires administration of the appropriate **anthelmintic**, obstructive edema requires removal of the physical cause, and increased permeability edema require resolution of the cause of endothelial damage. **Diuretics** may relieve the effects of pressure temporarily.

Shock

- **Shock** is defined as **inadequate cellular energy production** and most commonly occurs secondary to **poor tissue perfusion** from low or unevenly distributed blood flow. This leads to a critical decrease in oxygen delivery (DO2) compared to oxygen consumption (VO2) in the tissues.

- Shock may be due to a reduction in venous return (circuit failure) secondary to hypovolemia, hemorrhage, maldistribution of blood or obstruction to venous return.

Classification of Shock

- A common classification includes **hypovolemic**, **distributive**, and **cardiogenic causes**. For all forms of shock **except** cardiogenic shock, the main **therapy involves** rapid administration of **large volumes of isotonic crystalloid fluids**.

Etiology: The circulatory system consists of a pump (the heart) and a circuit (the vasculature). Circulatory shock can result from abnormal functioning of the pump or circuit, or both.

- **End points** of resuscitation such as **normalization of heart rate** and **blood pressure**, improved pulse quality, and **resolution of lactic acidosis** are necessary to tailor therapy to the individual patient. Oxygen therapy and avoidance of stress are key components to the treatment of cardiogenic shock.

Functional Classifications and Examples of Shock
Hypovolemic: A decrease in circulating blood volume
Hemorrhage
Severe dehydration
Trauma
Cardiogenic: A decrease in forward flow from the heart
Congestive heart failure
Cardiac arrhythmia (abnormal heart beat-in Rhythm)
Cardiac tamponade
Drug overdose (anesthetics, b-blockers, calcium channel blockers, etc.)
Distributive: A loss of systemic vascular resistance
Sepsis (mediators of inflammation cause hypotension & decreased myocardial contractility)
Obstruction (heartworm disease, saddle thrombosis)
Anaphylaxis

Clinical findings:

- Depression and weakness
- Subnormal temperature
- Elevated heart rate with weak thread pulse
- Cold skin and extremities
- Prolonged capillary refill time (hypodynamic stage)
- Progressive development without aggressive fluid therapy and collapse and death from irreversible shock.

- **Hypovolemic shock** occurs when there is a reduction in circulating blood volume due to plasma or free water loss.
- **Hemorrhagic shock** occurs when there is a reduction in circulating blood volume due to rapid blood loss.
- **Maldistributive shock** occurs when there is a 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 is a large third space such as the thoracic or abdominal cavities
- Regardless of the cause for circuit failure and inadequate venous return, **tissue hypoperfusion** results, leading to **impaired oxygen uptake** and **anaerobic metabolism**, **lactate acidemia**, and **acidosis.**

Management

- ✓ Identification and removal of the cause
- ✓ For Hypovolemic and Maldistributive shock: The rapid administration of intravenous fluids (e.g Isotonic crystalloid solutions, Hypertonic saline solution and colloids) is the single most important therapy in animals with hypovolemia or mal distributive shock. The goal is to increase venous return and thereby restore circulatory function and tissue perfusion.
- ✓ For hemorrhagic shock: replenish the blood volume, Drugs to assist coagulation and arrest hemorrhage (Hemostatic Drugs) are used (**Aminocaproic acid--**10 g in 1 L of saline for an adult horse IV) has been recommended.
- Ancillary treatment: Corticosteroids use is controversial, COX inhibitors (Flunexin) in sepsis, antibiotics use in maldistributive shock should be immediately used.

Respiratory system

General Terminology

- **Eupnoea**: normal breathing is referred as eupnoea.
- **Polypnoea**: increase in respiration rate is referred as polypnoea.
- **Hyperpnoea**: increase in respiration rate with increase in depth is referred as hyperpnoea.
- **Oligopnoea**: decrease in respiratory frequency is referred as oligopnoea.
- **Dyspnoea**: difficulty in respiration or painful respiration is referred as dyspnoea.
- **Pleural Rub**: it denotes frictional or rubbing sound produced due to the rough layers of pleura.
- **Moist Rales**: characterised by interrupted bubbling sound produced during respiration. Sound is produced due to the presence of fluid secretion within alveoli or bronchi or both.

- **Crepitant Rales**: sound is produced by separation of sticky walls (due to exudate) of alveoli and bronchioles.
- **Ozena**: an atrophic rhinitis marked by a **thick mucopurulent** discharge, **mucosal crusting**, and a **strong odor** or a disease characterized by intranasal crusting, atrophy, and fetid odor.
- **Pneumothorax**: is the abnormal accumulation of air in the pleural space. It develops when air leaks into the space between lung and chest wall.
- **Hydrothorax**: fluid accumulation in the pleural cavity is referred as hydrothorax.
- **Snoring** - when there is pharyngeal obstruction, as in tuberculous adenitis of the pharyngeal lymph nodes.
- **Roaring** - in paralysis of the vocal cords
- **Sneezing** - due to nasal irritation

PULMONARY EMPHYSEMA

- ✓ Pulmonary emphysema is **distension of the lung** caused by **overdistension of alveoli** with **rupture of alveolar walls** with or without escape of air into the interstitial spaces.
- ✓ Pulmonary emphysema is **always secondary to some primary lesion** which effectively **traps an excessive amount of air** in the alveoli.

ETIOLOGY

- Pulmonary emphysema is an important lesion only in cattle, although occasional cases occur in pigs. The **bovine lung is highly susceptible** to the development of emphysema from many different causes, including those of non- respiratory in origin.

Cattle

- ✓ Acute interstitial pneumonia
- ✓ Parasitic pneumonia with pulmonary edema in acute anaphylaxis
- ✓ Perforation of the lung by foreign body as in traumatic reticuloperitonitis

Horses

- ✓ Bronchiolitis due to viral infection of the respiratory tract in young horses.

All species

- ✓ Secondary to bronchopneumonia Poisoning by oleander, Bryophyllum pinnatum and moldy sweet potatoes.
- ✓ Acute chemical injury - as in inhalation of welding fumes
- ✓ Chlorine gas poisoning

PATHOGENESIS

- Emphysema occurs because of destruction of the connective tissues of the lung, including the supporting and elastic tissue of the pulmonary parenchyma.
- In **interstitial emphysema** there is the additional factor of **distension of the connective tissue with air and compression collapse of the alveoli**.
- The development of interstitial emphysema **depends largely upon the amount of interstitial tissue** that is present and is most common in **cattle and pigs.**
- The **pathophysiological consequences of emphysema** depend upon the **inefficiency of evacuation** of pulmonary air-space and **failure of normal gaseous exchange** in the lungs.

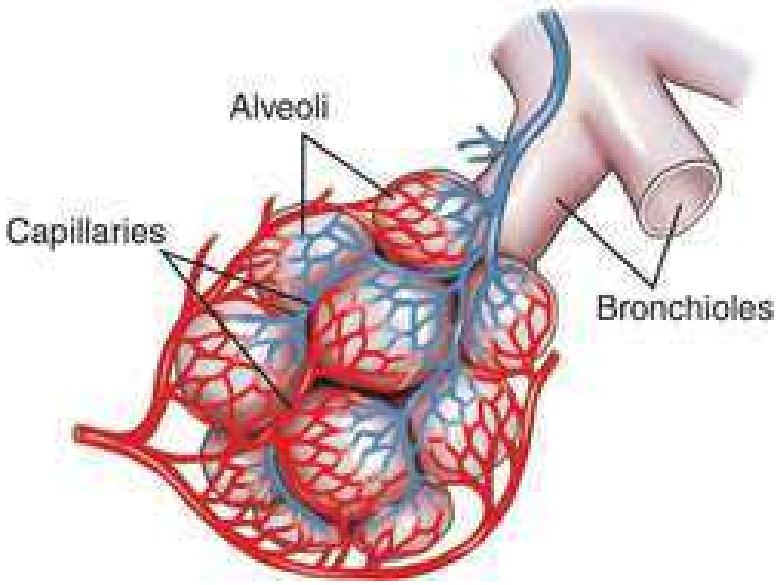
- **Retention of carbon dioxide** stimulates an increase in the depth of respiration. **Anoxia** develops and metabolism of all body tissues is reduced. The characteristic effect of emphysema is to produce an **increase in expiratory effort** necessitated by the failure of normal elastic recoil.
- Interference with the pulmonary circulation results from collapse of much of the alveolar wall area and a consequent diminution of the capillary bed.
- Characteristically, diffuse pulmonary emphysema causes severe expiratory dyspnea with a grunt on expiration and loud crackling lung sounds on auscultation over the emphysematous lungs. In severe cases in cattle, open-mouth breathing is common.
- In case of **cattle and swine** as there is more interstitial tissue, **interstitial emphysema** is common.
- In case of **dog and horses** more of alveolar tissue is there, so **alveolar emphysema** is common.

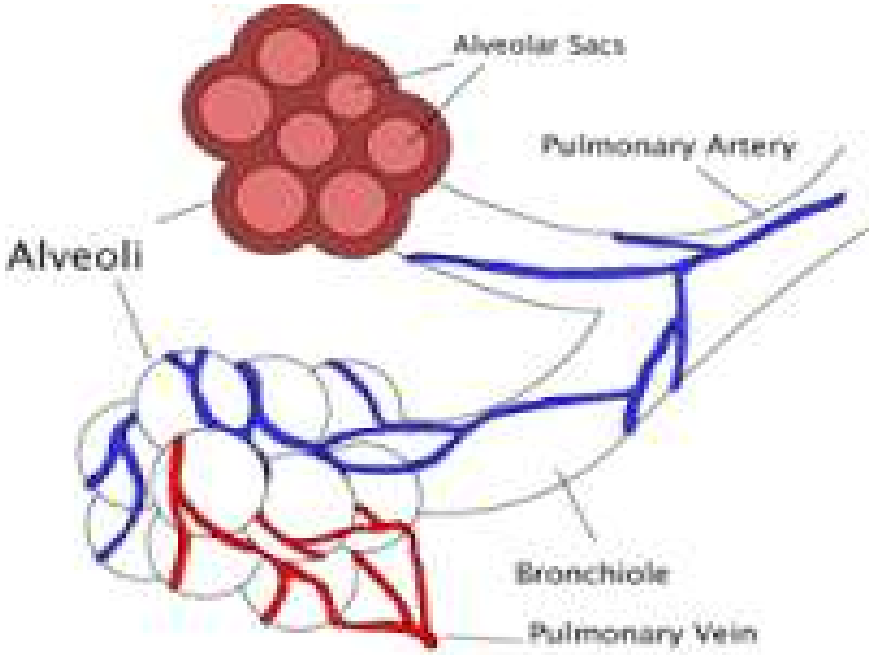
TREATMENT

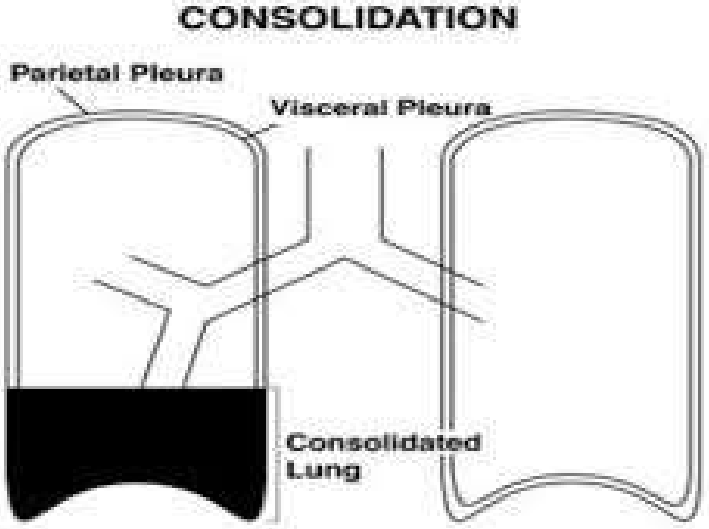
- The treatment of pulmonary emphysema will depend on
- Species affected
- Cause of the emphysema and
- Stage of the disease.
- There is **no known specific treatment** for the pulmonary emphysema associated with acute interstitial pneumonia in cattle.
- The emphysema secondary to the infectious pneumonias will usually resolve spontaneously if the primary lesion of the lung is treated effectively.
- Antihistamines, atropine and corticosteroids have been used for the treatment of pulmonary emphysema secondary to interstitial pneumonia in cattle.

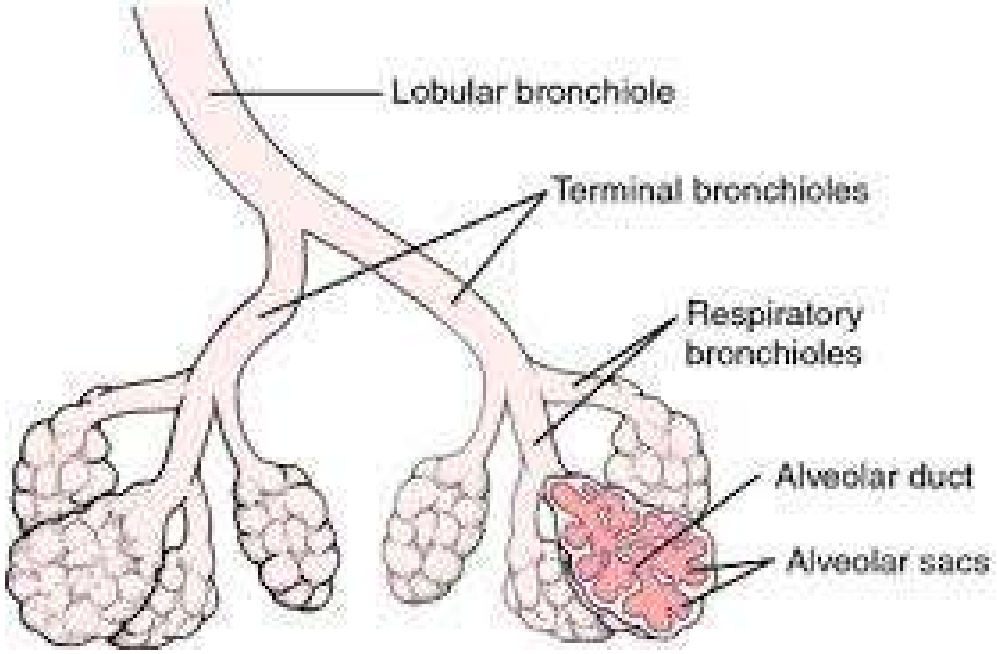
PNEUMONIA

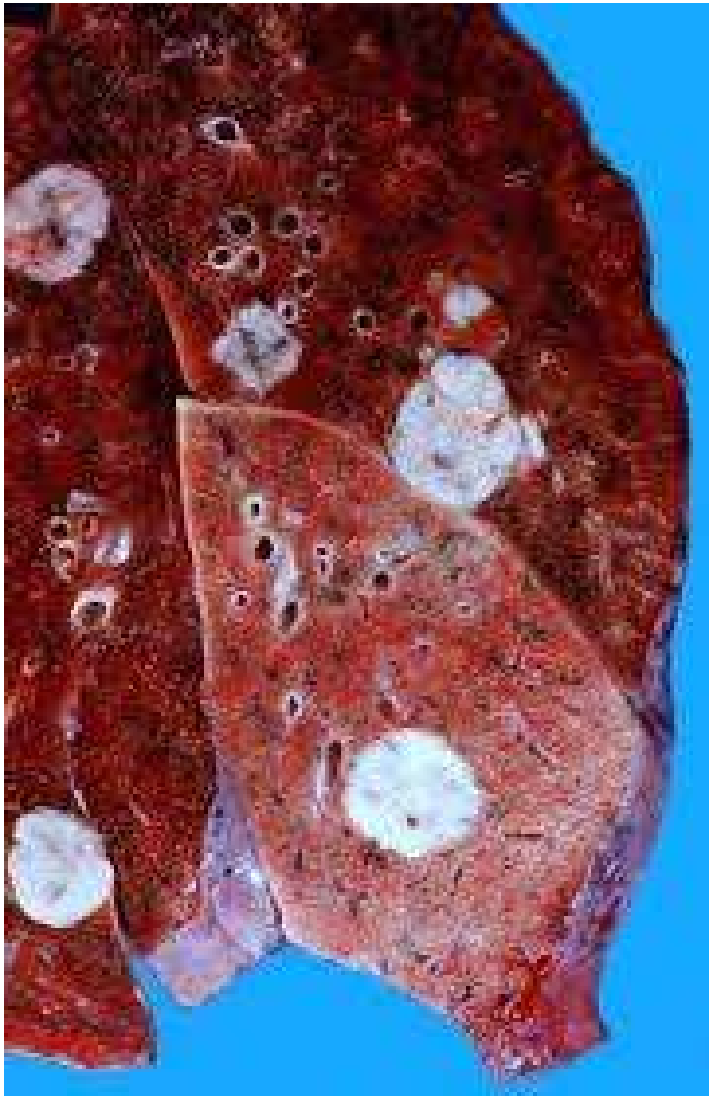
- Inflammation of pulmonary **parenchyma** accompanied by inflammation of **bronchioles** and **pleura** clinically manifested by increase in respiratory rate, nasal discharge, coughing and abnormal lung sounds on auscultation. In most of bacterial pneumonia, there is toxemia also.
- **ETIOLOGY**:- Caused by **bacteria**, **virus** or combination of **both**, **fungus** and **parasite**.
- In most of **pneumonia in farm animals are bronchogenic in origin** but some originates by haematogenous route.
- 1) **Bacterial Pneumonia**:-
- Specific diseases like CBPP and CCPP (***M. mycoides*** var ***Capri***). Pneumonic pasturellosis caused by ***P. haemolitica*** and **P. multocida** in cattle, sheep and swine
- Bacterial calf pneumonia caused by ***Klebshiella***.
- Pneumonia is part of diseaes like T.B (***M. tuberculi***), necrobacillosis (***Fusiformis neoforus***) in cattle , salmonellosis in pigs , strangles (***S. equi***) and glanders (***M. mallei***) in equines.


Alveoli
Capillaries
Bronchioles


Alveolar Sacs
Pulmonary Artery
Alveoli
Bronchiole
Pulmonary Vein


CONSOLIDATION
Parietal Pleura
Visceral Pleura
Consolidated Lung


Lobular bronchiole
Terminal bronchioles
Respiratory bronchioles
Alveolar duct
Alveolar sacs



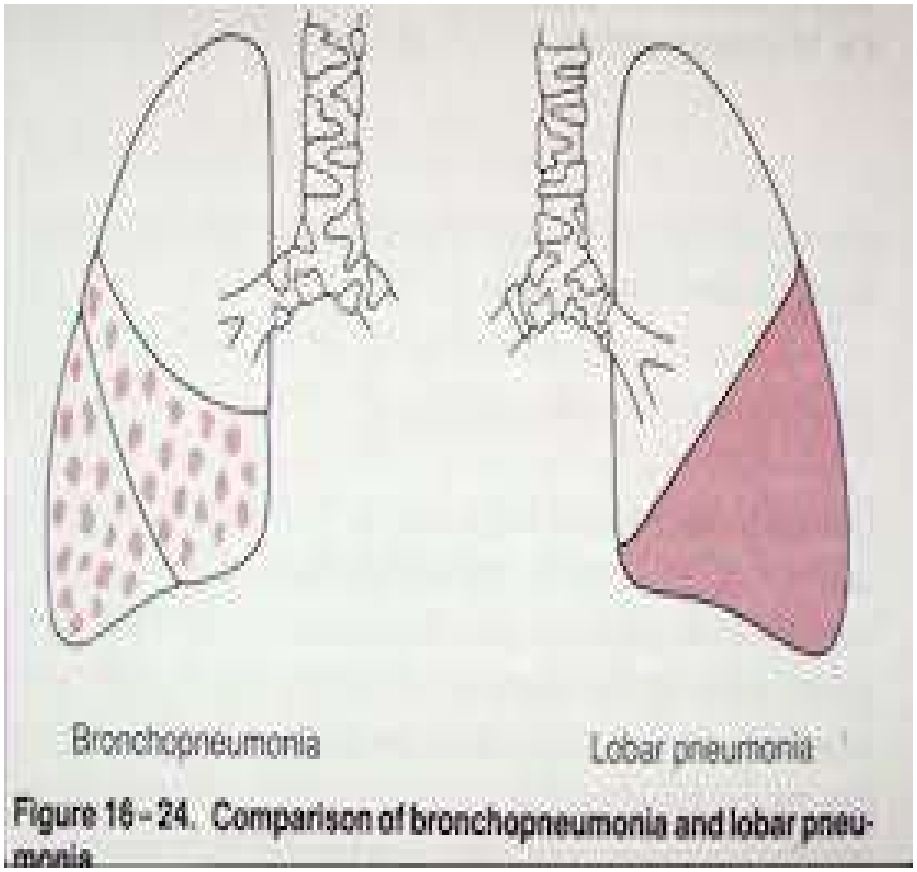


Figure 16 - 24. Comparison of bronchopneumonia and lobar pneumonia

Mucous Filled-Lung Consolidation

- 2) **Viral diseases**: -
- Viral pneumonia of calf due to **Para influenza 3**, **adeno virus**, **reovirus**, bovine herpes virus.
- Viral pneumonia of sheep
- Viral pneumonia of Swine
- Equine Viral Rhino pneumonitis
- Equine viral Arteritis
- 3) **Fungal diseases**: -
- Aspergellosis (***A. flavus*** and ***A. fumigatus***) in cattle and swines.
- Histoplasmosis by ***H.capsulatum*** of dogs.
- 4) **Parasitic**: -
- Verminous pneumonia of cattle caused by ***Dictycaulus viviparous***
- Verminous pneumonia in sheep by ***D.filaria***
- Verminous pneumonia in horse by ***D.arnfieldi***

PATHOGENESIS

Bacterial Pneumonia

- Organism gain **entry** by **inhalation** and or by **haematogenous route**: -
- 1) **Inhalation**: - first there is Bronchitis and broncheolitis from which infection spread either by peripheral extension or by coughing and lymphatic ducts. **Depending upon virulence and kind** of organism, lesions can be **acute fibrinous** as in case of CCPP ,CBPP and H.S. It can be **necrotic** in necrobacillosis or granulomatous as in T.B.
- 2) **Haematogenous route**:- Organisms are present in blood causing septicemia. These organism lodges in the pulmonary capillaries and form microabcess.

Viral Pneumonia:-

- It is mostly by **inhalation** and infection reaches to bronchioles and alveoli causing **mild bronchiolitis** and there is enlargement and proliferation of **alveolar epithelial cells** leading to **alveolar edema** but absence of acute inflammation.

Parasitic pneumonia:-

- After ingestion of parasitic infected larvae with food and water they enter lymphatic system from intestine, then to **mesenteric lymph node to pulmonary capillaries** to alveoli and finally to bronchi, here they **cause inflammation mechanically** and partially by **metabolic products**. Mature parasite migrate to bronchioles and set up inflammation there and **epithelium becomes thickened and hyperplastic**.
- Regardless of way in which lesions develop there is **interference** with gaseous exchange which leads to **anoxia** and **hypercapnoea** resulting in **dyspnoea** and **polypnoea**.
- In **bacterial pneumonia** there is **added effect of toxin** production by the organism and necrosed tissue.
- There is **accumulation of inflammatory exudates** in bronchi manifested by abnormal lung sounds. In **viral pneumonia** there is negligible bronchitis and **direct consolidation** of alveoli and so **no abnormal lung sound**.

CLINICAL SIGNS: -

- 1) Respiratory movements: in early stages, respiration is **rapid and shallow** and in later stages, there is both inspiratory and expiratory polypnoea &dyspnea.
- 2) **Cough** in **early** pneumonia is **dry painful** and **unproductive (no mucous production)** and less exudates is there and in later stages moist less painful and **productive (increased mucous production).**
- 3) **Nasal discharge** is **serous** in early in early stages and become mucoid to mucopurulent as diseases progresses.
- 4) Odor of breath is sometimes foul.
- Other signs include rise in body temp., anorexia, dullness, toxemias, increased pulse, gradual loss of body condition and reluctance to move.
- 7) In consolidation of lung, **bronchial tones are only sounds of affected lungs (less sound produced)** but moist rales can be heard on periphery of affected area.

CLINICAL PATHOLOGY: -

- **Nasal swab**, **Blood examination** (Leucocytosis) and **X-ray** examination is helpful only when the lungs are consolidated and show Granulomatous lesions.

DIAGNOSIS: history, physical examination, X-ray

TREATMENT: -

- 1) Isolation of animal, keeping the animal in chill free condition and well ventilated area.
- 2) Use of **broad spectrum antibiotics** in proper doses for 5-7 days.
- 3) Anti-inflammatory drugs
- 4) Suitable expectorants should be given to expel out exudates.
- 5) Medicated inhalation by use of TT oil, camphor, eucalyptus oil or Tenture bezoin Co.

Pneumothorax refers to the presence of air (or other gas) in the pleural cavity. **Entry of air** into the pleural cavity in sufficient quantity **causes collapse of the lung** and **impaired gas exchange** with consequent respiratory distress.

ETIOLOGY

- Pneumothorax is defined as either **spontaneous**, **traumatic**, **open**, **closed**, or **tension**. Spontaneous cases occur without any identifiable inciting event.
- **Open pneumothorax** describes the situation in which gas enters the pleural space other than from a ruptured or lacerated lung, such as through an open wound in the chest wall (**no lung injury**).
- **Closed pneumothorax** refers to gas accumulation in the pleural space in the absence of an open chest wound.
- **Tension pneumothorax** occurs when a wound acts as a one-way valve, with air entering the pleural space during inspiration but being prevented from exiting during expiration by a valve-like action of the wound margins.

- **Rupture of the lung** is a common cause of pneumothorax and can be either **secondary to thoracic trauma**, for example a **penetrating wound** that injures the lung, or lung disease. Most cases of pneumothorax in **cattle** are associated with pulmonary disease, notably **bronchopneumonia** and **interstitial pneumonia**. **Pleuropneumonia** is the most common cause of pneumothorax in **horses.**

Pathogenesis

- Entry of air into the pleural cavity results in **collapse** of the lung. There can be **partial** or **complete** collapse of the lung. Collapse of the lung results in **alveolar hypoventilation**, **hypoxemia**, **hypercapnia, cyanosis**, **dyspnea**, anxiety, and **hyperresonance on percussion** of the affected thorax. Tension pneumothorax can also lead to a direct decrease in venous return to the heart by compression and collapse of the vena cava.

CLINICAL FINDINGS

- There is an acute onset of **inspiratory dyspnea**, which may terminate fatally within a few minutes if the pneumothorax is bilateral and severe. If the collapse occurs in only one pleural sac, the **rib cage on the affected side collapses** and shows **decreased movement.**
- . On **auscultation of the thorax**, the breath sounds are markedly decreased in intensity and **commonly absent**.
- On percussion of the thorax on the affected side, a **hyperresonance** is detectable over the dorsal aspects of the thorax.

Diagnosis: by X-ray

TREATMENT

- The treatment depends on the cause of the pneumothorax and the severity of the respiratory distress and hypoxemia. Emergency decompression (**thoracocentesis**)of the pleural cavity is done by using a needle into the pleural cavity, connected to a tubing followed by aspiration of accumulated air.

PLEURITIS (PLEURISY)

- Pleuritis refers to **inflammation of the parietal and visceral pleura**. Inflammation of the pleura almost always **results** in **accumulation of fluid** in the pleural space. Pleuritis is characterized by varying degrees of toxemia, painful shallow breathing, pleural friction sounds.

ETIOLOGY

- Pleuritis is almost always associated with **diseases of the lungs**. **Pneumonia can progress to pleuritis**, and pleuritis can cause consolidation and infection of the lungs. **Primary pleuritis** is usually **due to perforation** of the pleural space and subsequent infection.
- Secondary pleuritis develops subsequent to the following conditions:
- **Pigs**: Glasser's disease , Pleuropneumonia

- **Cattle**: Secondary to pneumonia in cattle, Tuberculosis, CBPP & TRP.
- **Sheep and goats**: Pleuropneumonia associated with Mycoplasma spp., Streptococcus dysgalactiae in ewes.
- **Equines**: Strangles in horses and Secondary infection of equine influenza.

PATHOGENESIS

❖ Contact and movement between the parietal and visceral pleura causes pain due to stimulation of pain end organs in the pleura.

❖ Respiratory movements are restricted and the respiration is rapid and shallow.

- There is production of serofibrinous inflammatory exudate

CLINICAL FINDINGS

- **Pleural pain (pleurodynia)** is common and manifested as pawing, stiff forelimb gait, abducted elbows and reluctance to move or lie down.

- **Pleuritic friction sounds:** These may be audible over the thoracic wall.
- **Subcutaneous edema** of the ventral body wall extending from the pectorals to the prepubic area is common in **horses** with severe pleuritis

- **Pleural effusion**: In **cattle**, an inflammatory pleural effusion is often limited to one side because the pleural sacs do not communicate. **Bilateral pleural effusion** may indicate either a **bilateral pulmonary disease**

TREATMENT

- **Antimicrobial therapy**: to control the infection in the pleural cavities
- **Drainage and lavage of pleural cavity**: Clinical experience suggests that drainage improves the outcome.
- **Use of dexamethasone** at **0.1 mg/kg BW** to reduce the degree of pleural effusion. In acute cases of pleurisy in the horse analgesics such as **phenylbutazone** are valuable to relieve pain and anxiety, allowing the horse to eat and drink more normally.
- **Fibrinolytic therapy**: Fibrinolytic agents such as **streptokinase** have been used in human medicine to promote the thinning of pleural fluid

Urinary System

Terminology

- **Azotemia**: means increase in creatinine and blood urea nitrogen (BUN) leading to renal failure.
- **Proteinuria**: is the abnormal presence of albumin in the urine (usually >30 gm).
- **Oliguria**: means decrease in the urine production and is usually seen in acute kidney failure (in addition to other diseases).
- **Polyuria**: is the increase in urine production and is usually seen in chronic kidney failure (in addition to other diseases).

Renal Ischemia

- Renal ischemia results from **decreased renal perfusion** with subsequent **reduced glomerular filtration** in dehydrated patients. Renal ischemia will lead to reduced ATP production. Decreased intracellular ATP leads to **several metabolic and structural changes** within renal tubular cells, leading to **ingress of intracellular calcium** result in movement of water into the cell and **cell swelling**, which may contribute to **tubular obstruction**. Tight junction integrity is lost and causes cell desquamation. Damage to the kidney during ischemia will cause leaking of blood substances which are normally retained back during filtration such as RBC, WBC, Albumin, Glucose etc.
- Decreased renal perfusion may be **Prerenal** (dehydration, congestive heart failure, and clot in the renal artery) and **Renal** (infections, drug toxicity, metal toxicity etc).

- In Prerenal cases, animal is **able to concentrate urine** whereas in renal causes, kidney is not able to concentrate urine as normally so leads to renal failure (decreased urine production, and azotemia i.e increased Creatinine and blood urea nitrogen-BUN).

- Cattle with **severe dehydration** resulting from gastrointestinal obstruction or diarrhea frequently develop **renal infarcts**. Renal infarcts result in some RBCs, WBCs, and protein in the urine. Finding these abnormal constituents in urine from dehydrated patients should arouse suspicion of renal failure and alert the clinician to the need for rehydration and avoidance of nephrotoxic drugs.

Treatment: should be **directed toward the primary disease** and the patient **rehydrated** with IV fluids to **improve renal perfusion**, urine production, and to correct existing Prerenal azotemia.

Nephritis

- **Nephritis**: is the inflammation of the kidney characterised by azotemia and oliguria.
- **Glomerulonephritis**: is the inflammation of the glomeruli. Glomerular diseases affect primarily the glomeruli. The inflammation of the glomeruli may be followed by structural damage of other parts of the kidney. However, destruction of the glomerulus renders the remainder of the nephron non-functional and progressive destruction of glomeruli can lead to decreased glomerular filtration rate, azotemia and renal failure.
- Glomerulonephritis, a rare clinical condition in cattle, causes progressive renal failure and **severe proteinuria, hypoalbuminemia**, **weight loss**, and **ventral edema**.

Etiology

Cattle

- Glomerulonephritis is thought to be an auto-immune disease and develops either as a result of **antigen-antibody complexes** deposited in the glomeruli leading to damage.
- **Damage to the glomeruli interferes with normal filtration** such that protein loss from the kidney occurs and renal failure follows.

Dogs and Cats

- In dogs and cats, most GN is **idiopathic**, and the cause is never determined. Important causes in dogs and cats include:

✓ Feline Leukemia Virus and Feline Infectious Peritonitis in cats.

✓ **Pyometra** and heartworm disease in dogs.

✓ Chronic bacterial, parasitic, and neoplastic disease

✓ Autoimmune diseases.

Clinical Signs:

- **Cattle:** Weight loss, decreased appetite and production, poor hair coat, and ventral edema are typical signs in cattle affected with glomerulonephritis. Some patients have diarrhea.
- **Dogs and cats**: The most common signs observed are anorexia, weight loss, lethargy, polyuria, polydipsia, oral ulcers, Melena (black color feces) and vomiting.

Diagnosis

- Because the clinical signs are similar for glomerulonephritis and amyloidosis, **renal biopsy** is essential to confirm the diagnosis.
- Rectal palpation of an enlarged left kidney may be the only specific physical abnormality detected. Proteinuria will be detected by urinalysis.

Pathology: Azotemia (increase in BUN and Creatinine) and proteinuria.

Treatment

- Early or acute cases may be treated by supportive care for renal failure and specific therapy.
- For Glomerulonephritis: immunosuppressive drugs are given (e.g Corticosteroids, azathioprine, cyclophosphamide, and cyclosporine).

Interstitial Nephritis

- IN occurs rarely as a clinical disease in farm animals but is commonly observed in dogs. ***Leptospira canicola*** causes interstitial nephritis in dogs, cats, and cattle. The common clinical signs include moderate fever, dullness, depression, anorexia, and melena. There are pinpoint hemorrhages on the kidney surface, white foci or streaks on cortico-medullary area.

Treatment

- Early or acute cases may be treated by supportive care for renal failure and specific therapy (Renal Safe antibiotics).

Cystitis and Pyelonephritis

- Bovine cystitis is an **inflammation of the urinary bladder** of cattle that may ascend the ureters to cause infection of the kidneys (pyelonephritis). A similar condition is seen in sheep. The condition is **sporadic** and worldwide in distribution. Cystitis and pyelonephritis are most **often seen after parturition** (in one study, the average days to onset after parturition was 83), with **multiparous cows being at highest risk**. Cystitis and pyelonephritis are **rare in male cattle**.
- Among pets **cystitis** is more common in **bitches** than in the male dogs.

Etiology and Pathogenesis:

- Presently **E coli and Trueperella** (formerly Arcanobacterium or Corynebacterium) pyogenes are now the bacteria **most frequently** isolated from cows with pyelonephritis.
- **Pyelonephritis** develops from an **ascending infection** from the bladder.
- The organisms attack the mucosal lining of the bladder and ureters usually after some traumatic insult such as parturition.
- The stresses of parturition, peak lactation, and a high-protein diet (Increases pH-i.e. Alkaline; good medium for bacteria to grow) are all contributing factors

Clinical Signs of Cystitis

- First sign: passage of blood-stained urine
- Clinically characterised by **frequent painful urination** with the **passage of blood and inflammatory cells.**

Clinical signs of Pyelonephritis

- The infection proceeds **backwards (ascending)** from urinary bladder to the ureters, causing inflammation and then to the **kidneys** leading to the involvement of the kidney, the animal exhibits discomfort manifested by **frequent attempts of urination**, colic with restlessness, tail switching, polyuria, hematuria, or pyuria.
- In chronic cases, animal may show **polyuria, polydipsia, and stranguria.** As the disease progresses, the **bladder becomes thickened and inflamed**. The **ureters become thickened** and dilated with a purulent exudate. The involved **kidneys develop multiple small abscesses** on the surface that may extend into the cortex and medulla.

Diagnosis:

- Diagnosis is based on **clinical signs--hematuria**; a history of recent parturition; palpation of the left kidney for enlargement, loss of lobulation, and pain; **ultrasonographic** inspection of the kidneys, ureters, and bladder; **microscopic examination** of the urine for WBCs and bacteria; **urine culture** to identify the organism.

Treatment:

- Early diagnosis and prompt, sustained treatment are needed for a successful recovery.

- The treatment of choice for pyelonephritis due to Corynebacterium spp is **penicillin** (22,000 IU/kg, IM, bid) or **trimethoprim-sulfadoxine** (16 mg combined/kg, IM, bid) for ≥3 wk.
- ***E coli*** infections require a broad-spectrum antimicrobial. **Ceftiofur** (1.1–2.2 mg/kg/day, IM or SC) or **gentamicin** (2.2 mg/kg, IM, bid) for ≥3 wk have been used successfully in some cases.

- **Urolithiasis**: The process of formation of uroliths or stony concretions in any part of the urinary tract is known as urolithiasis.
- **Nephroliths**: presence of uroliths in the kidneys is referred as nephroliths.
- **Cystolithiasis**: presence of uroliths in the urinary bladder is referred as cystolithiasis.

Urolithiasis in Ruminants

- Uroliths in cattle, sheep, and goats are common, although, uroliths can be found anywhere within the urinary tract.
- Obstruction caused by urethroliths causes **urine retention** and leads to **bladder distention**, and **abdominal pain**.
- Untreated cases of urolithiasis may lead to **urethral / bladder rupture**, and **death** from **uremia** or **septicemia**.

- Urolithiasis is usually seen in **winter** in **steers** and **wethers** on full feed, or during **severe weather** conditions with **limited water intake**, especially when the water has a **high mineral** content.
- Uroliths occur in either sex, but obstructive urolithiasis occurs primarily in **males** (anatomical difference) and is found more common in **male goats**.

- **Etiology and Pathogenesis**

- In ruminants it is primarily a **nutritional disease**.
- Calves, lambs, and kids **castrated** at an early age and fed **high-grain diets** with roughly a **1:1 calcium:phosphorus ratio** or a **diet high in magnesium**.
- Diets high in calcium may result in calcium carbonate uroliths.

- The mineral **composition of water**, along with **dietary mineral imbalances**, contributes more to initiating **urolith formation** than **lack of water**.

- **The distal aspect of the sigmoid flexure of cattle** and the sigmoid flexure and **urethral process** of sheep and goats are **the most common sites** for uroliths to lodge.
- **Castration of young males** also **predisposes to urolith-induced urethral obstruction** by **removing hormonal influences** necessary for mature development of the **penis and urethra**.

Clinical Findings

- Clinical signs may be associated with partial or complete urethral occlusion.
- **Stranguria** (Pain during urination)
- **Partial obstruction**—cause to dribble blood-tinged urine
- Urine may dry on the preputial hairs and leave detectable mineral deposits.

- **Complete urethral obstruction**: signs are tenesmus, tail twitching, weight shifting, and other signs consistent with colic
- **Sequelae** of complete urethral obstruction: urethral perforation, hydronephrosis, or urinary bladder rupture
- **Diagnosis**:
- Diagnosis based on the history, clinical signs, and physical examination is usually straightforward.
- Rectal palpation may reveal an **enlarged, distended bladder**, or the bladder may be nonpalpable, consistent with bladder rupture.
- In small ruminants, the distended bladder can be **felt by abdominal palpation** and visualized on **ultrasound examination**.
- Calcium carbonate and calcium oxalate calculi can be **seen on radiographs** of the urethra in small ruminants; **struvite calculi are not seen on radiographs**.

Treatment (Obstructive Urolithiasis)

- Treatment involves **establishing a patent urethra** and correcting **fluid and electrolyte imbalances**.
- In many instances, **surgical management** of the obstruction is all that is necessary. Uroliths trapped within the urethral process of sheep and goats may be removed by **gentle manipulation** or by **amputation of the urethral process**.
- Severely uremic and depressed animals require rehydration and correction of acid-base and electrolyte abnormalities, especially hyperkalemia or hyperammonemia.
- Treatment with IV normal saline is indicated. Animals with **early clinical signs** of obstructive urethral disease may benefit from conservative therapy using **antispasmodics** and **tranquilizers**.

Control

- Cystotomy followed by dietary management is believed to be a more effective long term solution to urolithiasis.
- **Struvite calculi prevention**: are to **increase urinary chloride excretion**, **decrease urine pH**, and **provide a calcium:phosphorus ratio of 2:1** in the complete ration.
- **Intensive concentrate feeding**, such as in many finishing programs, **frequently leads to urolith formation and urethral obstruction**. Adjunct measures to minimize the formation of urethral calculi include **adding sodium chloride up to 4% of the total ration**.
- **Ammonium chloride can be used as a urinary acidifying agent (7–10 g/head/day** for a 30-kg lamb or kid; **50–80 g/head/day for a 240-kg steer**).
- **Lower calcium diets** should be used **if** calcium carbonate or **calcium oxalate is a concern**.

Musculoskeletal System

Myopathy

- It denotes the **non-inflammatory degeneration** of skeletal muscles

Etiology

- **Nutrition**: vitamin-E and selenium deficiency are the chief factors for nutritional myopathy in farm animals.
- **Effect of Exercise**: post-exertion or exercise may cause muscular degeneration. This condition in horse is known as “tying up”.
- **Inherited or congenital**: some animals may get the problem at birth as an inherited defect.
- **Chemical agents**: some poisonous plants containing mycotoxins producing muscular degeneration.
- **Drugs**: prolonged use of drugs like corticosteroid has been suggested as a factor.

- **Local ischemia**: recumbency for more than a day in one position may produce compression of blood vessels. This will produce ischemia of muscles leading to degeneration and necrosis.
- **Nerve damage**: nerve damage may lead to muscular atrophy of muscles innervated by the nerve.
- **Mitochondrial myopathy**: damage of mitochondria may be observed in muscular diseases. Mitochondrial abnormalities will interfere in energy production.
- **Endocrinal myopathy**: Cushing's disease in dog caused by excess production of glucocorticoids is responsible for muscle abnormalities.

Pathogenesis

- In myopathies due to **vitamin-E and or selenium deficiency** in herbivores there is **degeneration of muscle fibres**. It may cause swelling and hyaline degeneration of muscle fibres. Potassium content of muscles may fall and sodium content rises in some form of myopathies. During **prolonged exercise** there is anoxic condition. Anoxic condition leads to the formation of **sarcolactic acid** from muscle glycogen. Sarcolactic acid causes myofibrillar degeneration and coagulation of muscle protein. Due to intense reaction necrosis of muscles may result. As a result of necrosis **myoglobin is leaked out** and excretes through urine causing myoglobinuria (red colour urine). Excess of myoglobin may cause myoglobinuric nephropathy. There is excessive **release of muscle enzymes** due to the muscle damage such as **creatine phosphokinase** (**CPK**). In neurogenic myopathy there is paralysis of muscles (flaccid type).

Clinical findings

- Weakness of the muscles.
- Inability to rise and walk.
- Abnormalities in gait
- Animal becomes paretic or recumbent.
- Heart rate is increased
- Muscles are swollen, hard and painful.
- Urine may become red or coffee coloured
- Edema of head and neck
- Stiffness of joints and lameness
- Animal may be unable to rise (downer cow syndrome)
- There may be atrophy of muscles
- Sudden death may occur due to heart failure

Clinical Pathology

- Elevation of CPK enzyme (normal level=10=30 IU).

Diagnosis

- Clinical signs and clinical pathology

Line of treatment

- Vitamin-E and selenium supplementation are to be made
- In acidosis condition sodium bicarbonate is to be given
- There should be provision for good bedding

Myositis

- Inflammation of muscle fibres is known as myositis.

Etiology

- Traumatic injury of the muscle or tearing of muscle fibres.
- Diseases which produces myositis includes Black quarter, Blue tongue, FMD, Sarcosporidiosis, Trichinosis, and Cysticercosis.
- Exercise induced myositis in horses.
- Eosinophilic myositis in dogs and cats.

Clinical findings

- Severe pain in muscles exhibited as lameness.
- Elevation of body temperature
- Muscles are swollen, firm and painful
- Stiffness in gait due to muscular rigidity
- Crepitation's in muscles in black quarter
- Signs of toxemia may develop

Diagnoses: based on clinical signs and muscle biopsy

Treatment

- Specific therapy to specific etiological agent

Arthritis

- Inflammation of the **joint** is known as **arthritis**. It accompanies inflammation of the synovial membrane. Inflammation of the **synovial membrane** is known as **synovitis**. **Osteomyelitis** is inflammation of bone marrow, cortex, and possibly periosteum.
- Arthritis is seen in farm as well as small animals (dogs). Sometimes all the joints are affected simultaneously known as **polyarthritis**. Osteoarthritis denotes inflammatory as well as degenerative changes of the bone.

Etiology

- **Bacterial infections**: *E. coli*, *streptococcus spp.*, *Corynebacterium equi*, *Salmonella spp.*, *Erysipelothrix insidiosa*, *Haemophilus spp*.
- **Mycoplasmal infection**: *Mycoplasma mycoids*, *Mycoplasma agalactiae*.
- **Chlamydial infection**: chlamydia spp. (polyarthritis)
- **Viral infections**: Bovine Viral Diarrhoea

Pathogenesis

- Arthritis may occur as a result of trauma or infection. Infection is mostly by hematogenous route. Localisation of infection in joints occurs following bacteremia. The synovial membrane shows inflammatory changes characterised by edema and deposition of fibrin. The joint capsule becomes painful and is swollen with fluid. Depending upon the nature or inflammation, arthritis may be of different types viz. acute serous arthritis, acute serofibrinous arthritis, chronic osteoarthritis, purulent arthritis, exudative arthritis etc.

Clinical Findings

- Pain in the affected joints
- Deformity of the affected joints
- Joints are thick, swollen and hot
- Crepitation sound on movement of the joint

- Pain during movement
- Lameness
- Animal may become recumbent
- In chronic cases, Ankylosis set in and working capability of the animal is permanently lost.
- **Clinical pathology:** Analysis of synovial fluid and in vitro antibiotic sensitivity are to be done to arrive at a diagnosis
- **Diagnosis**: based on clinico pathological examination. Radiography is useful for diagnosis.

Treatment

- Antibacterial therapy for a period of 4-6 weeks in case of infectious arthritis
- Analgesics and anti-inflammatory drugs to reduce pain and inflammation.
- Surgical intervention to remove purulent material and fibrin from joints to minimize cartilage destruction.

Nervous System

Encephalitis

- Encephalitis refers to the **inflammation of the brain**.
- It may be defined as inflammation of the encephalon (brain) characterised by initial hyperexcitability followed by paralysis and unconsciousness. It may be a primary disease or a secondary lesion or consequence to disease elsewhere in the system.

Etiology

- **Viral agents**: virus invasion causes death of the neurons e.g Rabies, Pseudo rabies, Japanese encephalitis.
- **Cattle**: Sporadic bovine encephalomyelitis, Bovine malignant catarrh, rabies etc.
- **Horse**: Infectious encephalomyelitis, Borna disease.
- **Pig**: Viral encephalitis, Hog cholera, African swine fever.
- **Sheep and Goat**: Scrapie, Louping ill, Visna.
- **Dog**: canine distemper, Rabies.

- **Bacterial agents**: Listerial infection, Necrobacillus infection, Enterotoxemia, Salmonella infection etc
- **Parasitic infections**: Migrating Larvae, Multiceps multiceps, Toxoplasmosis, Nervous Coccidiosis
- **Toxic agents**: Lead poisoning, Arsenic Poisoning, Salt poisoning etc.
- **Fungal agents**: Cryptococcosis

Pathogenesis: infectious agents cause **irritation** and **degenerative** changes in the brain tissue. There will be formation of **multiple necrotic foci** or **micro-abscesses**. Lesions depend upon the site and nature of causative agents. Bacteria and viruses affect primarily **vasculature** e.g Bovine malignant catarrh and Sporadic bovine encephalomyelitis.

- **Viruses** may enter nervous tissue by **progressive peripheral trunk** e.g Rabies, Pseudo-rabies and Listeria monocytogenes. Entry of infectious agents can also occur via **olfactory nerve**.

- There may be acute edematous swelling. This may obstruct blood flow and interfere with cerebral function. There is gradual increase in intracranial pressure.

Clinical findings

- High rise of temperature
- Mania, aggressiveness(rabies), depression
- Circling movement (Listeria infection), head pressing (cerebral edema)
- Clonic convulsion (canine distemper), muscular tremor and pawing on the ground
- Frothy salivation (epilepsy) from commissure of mouth
- Champing of jaws (canine distemper) and hyperaesthesia
- Depression of consciousness
- Spastic type of paralysis
- Ataxia or incoordination of gait
- Nystagmus of eye ball
- Unilateral facial paralysis

Pathology

- Examination of cerebro spinal fluid (CSF) for cellular, bio-chemical and microbiological analysis.

Diagnosis

- **Acute cerebral edema**-history of salt poisoning
- **Poisoning**-salivation, history of poisoning, no fever, blindness, acute onset
- **Encephalomalacia**-history of grain engorgement, thiamine deficiency, respond to vitamin B_1 therapy
- **Meningitis**-temperature reaction, hyperaesthesia, rigidity of muscles

Treatment

- Use of sedative and tranquilizer
- High dose of broad spectrum antibiotics-Cefotaxime, Chloramphenicol (cause bone marrow suppression).
- Use of corticosteroid to reduce inflammation.
- Specific antidote against poisoning
- Use of mannitol for cerebral edema @ 0.5-1.0 gram/kg body weight in dogs

Encephalomalacia

- Encephalomalacia refers to **softening (degeneration)** while leukoencephalomalacia and polio-encephalomalacia refer to softening of **white matter** and **grey matter** respectively. Encephalomalacia includes the degenerative diseases of the brain. The changes are comprised of demyelination, necrosis and edema.

Etiology

- It may occur due to metabolic disorders
- **Hepatic encephalopathy**: in hepatoencephalopathy, **urea cycle is deranged**, hence, ammonia is not converted to urea due to damaged liver leading to ammonia accumulation in the blood. Ammonia intoxication leads to damage of CNS.
- **Copper deficiency**: it causes tissue oxidation by cytochrome-oxidase. Anoxic condition due to copper deficiency causes demyelination. Along with iron, copper prevents anemia. Deficiency of copper leads to anemic anoxia. Anemic anoxia causes demyelination.

- **Thiamine deficiency**: causes include **bracken fern (**contains Thiaminase enzyme**) poisoning**, ingestion of **mouldy corn** and **excess grain** (reduces pH, growth of thiaminase producing bacteria). cerebral changes are due to lack of diphosphothiamine essential in pyruvic acid metabolism of the nervous system. Thiamine depletion leads to disturbed function.
- **Infection with clostridium perfringens type D**: (pulpy kidney disease)
- **Ingestion of toxic chemicals**: lead, mercury, arsenic, salt poisoning.
- **Deficiency of vitamin E**: it produces leukoencephalomalacia in pig.

Pathogenesis

- High level of thiaminase causes destruction, inactivation, impairment of absorption and phosphorylation of thiamine. Two factors are responsible for activation of thiaminase. They are thiamine antimetabolic and phenothiazine tranquilizers. These tranquilizers cause activation of thiaminase and destruction of thiamine.
- Due to destruction of thiamine, pentose phosphate pathway is inhibited leading to production of lactate, pyruvate and alpha-ketoglutamic acid. Lactate cause degenerative changes in the brain. Sometimes cortical ischemia may lead to focal cortical necrosis.

Clinical findings

- **Acute form**: muscle tremors (more on head), frothy salivation and champing of jaws, head pressing & blindness, tono-clonic convulsions, opisthotonos condition, nystagmus of the eyeball and finally death in young age group.
- **Sub-acute form**: anorexia, depression, circling movements, incoordination of gait, and head pressing.

Pathology

- There will be increased level of blood lactate and pyruvate. Decreased level of thiamine and increased thiaminase level.

Line of treatment

- No specific therapy
- Vitamin B1 (thiamine) @ 5-10 mg/kg b.wt. and repeated after 4-6 hr interval.
- Mannitol to reduce intracranial pressure
- Ruminal contents may be replaced with fresh ruminal fluid

Meningitis

- Meningitis is an inflammation of pia and arachnoid membranes. The inflammation of dura is known as pachymeningitis. As a general term, the inflammatory condition of meninges is known as meningitis. It is manifested by fever, hyperaesthesia and rigidity of muscles.

Etiology

- **Cattle**
  - **Viral meningitis**: sporadic bovine encephalomyelitis, BMC, IBR.
  - **Bacterial meningitis**: streptococcal infection, staphylococcal infection, coliform orgaisms, Listeria infection, etc.
- **Horse**
  - Strangle

- **Pig**
  - Glassers disease, Salmonellosis, otitis etc.
- **Goat**
  - Pasteurellosis
- **Dog**
  - Rabies, CD, otitis, etc.

Pathogenesis

- Inflammatory response of meninges causes **edematous swelling** of the membranes. This **interferes** with **blood supply** of brain. There is **increased intracranial pressure** due to deranged drainage of CSF.

Clinical findings

- Acute onset with chill and rise of temperature upto 104-106 ^{0}F
- Excitation and restlessness due to motor irritation.
- Tonic spasm of the muscles of the head and neck.
- Head and neck becomes rigid.
- Circling
- Incoordination
- Cerebral meningitis is characterised by hyperaesthesisa of skin, blindness, trismus, opisthotonos and rigidity of muscles of neck and back.
- Vomiting is common in early stages in pigs.
- Young animals suffer from opthalmitis
- Respiration: cheyne-stokes or biot's respiration is often observed.

Pathology

- CSF examination, culture for organisms, high protein (20-270 mg/dl) and high cell count. Peripheral blood will reveal neutrophilic leucocytosis.

Diagnosis:

- History and clinical signs
- Neutophillic leucocytosis in peripheral blood examination.

Treatment: similar to encephalitis.