General Medicine

Medicine is the science of treating diseases - the healing art. Veterinary medicine is that branch of medical science which deals with the diagnosis, treatment, prevention and general study of the diseases of animals, especially the domesticated ones. In order to practice veterinary medicine, one must be armed with thorough knowledge of anatomy, physiology, parasitology, pathology, microbiology, pharmacology and animal ethology to distinguish diseased from healthy ones.

How is the profession of animal worker different from that of a medical profession?
1. Animal patients cannot express its subjective feelings.
2. A fair percentage of animal owners/attendants give wrong statement about the animal’s illness, making the task more troublesome.
3. Animal patients do not cooperate while examination.
4. Various species of animals have different structures and functions of body systems making the process of diagnosis more complicated.
5. A wide variation in normal physiology in different species of animals with distinct behaviour of them makes the task of diagnosis more cumbersome.

Branches of veterinary medicine
Veterinary medicine may be divided into two branches:
1. **Clinical veterinary medicine** (bed-side medicine, internal medicine, curative medicine): that branch of veterinary medicine which covers the art of making a correct diagnosis and extends the remedial and curative measures against diseases of animals.
2. **Preventive veterinary medicine**: that branch which ensures measures to maintain the health when the disease is imminent. It deals with all measures to control and prevent animal diseases.
1. General Clinical Conditions

General clinical conditions are those conditions which are not confined to any particular diseases or organs/system. Their manifestation could cut across all the organs/systems.

1. Toxaemia

Toxaemia is a clinical systemic state caused by wide spread activation of host defense mechanism to the presence of toxins in the blood, usually with tissue or organ damage, fever, and severe intestinal upset. Toxaemia may occur as a result of either antigens or due to metabolic toxins and may be classified as follows.

![Toxin Diagram]

### Etiology

1. **Antigenic toxin** - produced by bacteria and to a lesser extent by helminthes. Both act as antigens and stimulate the development of antibodies. Antigenic toxins are divided into two-
   a. Exotoxins- exotoxins are protein substance produced by bacteria which diffuse into the surrounding medium e.g., those produced by *Clostridium spp.*
   i. Ingested preformed as in **botulism**
   ii. Produced in large quantities by heavy growth in the intestines as **enterotoxaemia** *(enterotoxins)*. Enterotoxin exerts their effect principally on the mucosa of the intestine causing disturbances of fluid and electrolyte balance. E.g., enterotoxin released by *E.coli*, which causes a hyper secretory diarrhea in the neonatal farm animals
   iii. From growth in the tissue as in **black leg**

b. Endotoxins- the endotoxins are lipopolysaccharides found in the outer wall of the bacteria. Endotoxins are released in to the immediate surroundings when the bacteria undergo rapid proliferation or most commonly when the bacterial cell wall breaks and liberates them. They can get access to the blood when there is a severe localized infection, such as coliform mastitis in dairy cattle, or disseminated infection, such as coliform septicaemia in new born calves.
2. **Metabolic toxins**
These may accumulate as a result of incomplete elimination of toxic materials normally produced by body metabolism, or by abnormal metabolism. E.g., Uraemia (ammonia in blood).

**Clinical findings**

*Acute enterotoxaemia in calves*
- Depression, anorexia, and muscular weakness
- Calves may not suckle voluntarily
- Scant feces are common. But diarrhea may also occur
- Tachycardia
- Weak and rapid pulse
- Fever initially but later temperature drops to normal or subnormal
- Terminal muscular weakness to the point of collapse and death occurs.

*Chronic enterotoxaemia*
- Lethargy
- Separation from the group
- Inappetence
- Failure to grow or produce
- Emaciation

*Severe endotoxaemia*
- Depression
- Hyperthermia followed by hypothermia
- Tachycardia followed by decreased cardiac output
- Decreased systemic blood pressure
- Diarrhea
- Congested mucosa with an increased capillary refill time
- Cool skin and extremities
- Muscular weakness leading to recumbency

**Clinical pathology (laboratory examination)**

*Hematology* - Leukocytosis and neutrophilia in mild endotoxaemia
- Leucopenia, neutropenia and lymphopenia in severe toxemaia

*Serum biochemistry* - Low blood glucose level
- High blood non protein nitrogen
- Anaemia and
- Albuminuria

**Treatment principles**
1. Removal of foci of infection
2. Fluid and electrolyte therapy
3. Antimicrobials
4. NSAIDs

Endotoxaemia requires a quick and comprehensive treatment plan as follows;
1. Fluid and electrolyte- a balanced electrolyte solution by IV route over several hours
2. Broad spectrum antimicrobial such as oxytetracycline and chloramphenicol
3. Antiserum in case of *E.coli* endotoxaemia in horse and cattle
4. Glucocorticoids, NSAIDs and anti-prostaglandins

A beneficial response is noted by the following:
- Correction of the peripheral vasoconstriction
- Restoration of an acceptable pulse quality
- Return of urine output
- Return of central venous pressure and arterial blood pressure
- Restoration of cardiac output.

2. Fever
Denotes the elevation of body temperature regardless of environmental temperature and is mainly a general reaction of the body to the action of harmful agents or pyrogens.

**Cause**
Microbes, foreign bodies, chemicals and immune reactions

**Pathogenesis**

![Diagram](https://via.placeholder.com/150)

**Clinical findings**
- Dullness, lowering of head, drooping of ear and disinclination to move.
- Inappetance, increased thirst, constipation
- Erection of hair, dry muzzle, congested mucous membrane
- Coloured urine, shivering, rapid respiration

**Management of fever**
- Antipyretics
- Antibiotics
3. Anaemia

Anaemia refers decrease in the quantity of haemoglobin or the number of erythrocytes or both per unit volume of blood. It is clinically manifested pale mucous membrane, tachycardia and loss of muscular strength and vigour. Anemia can be classified into three types based on the aetiology:

- **Nutritional**
  - Haemolytic
  - Intracellular
  - Babesiosis
  - Haemoglobinuria

- **Depressed Erythropoiesis**
  - Extracellular
  - Injury
  - Parasitism

- **Internal**
  - Hypoplastic
  - Co, Cu, Fe, Vit B12, Folic acid
  - Drugs
  - Radiations

- **External**
  - Haemorrhagic
  - Injury
  - Autoimmune diseases

- **Parasitic**
  - Injury

**Figure 1.2.** Classification of anaemia based on the causative agents.

**Pathogenesis**

**Figure 1.3.** Pathogenesis of anaemia.

**Clinical signs**

- Paleness of mucous membrane
- Muscular weakness and lethargy.
- Inappetance
- Tachycardia
- Laboured breathing in later stages
- Shock.

**Diagnosis**
From clinical signs and Laboratory investigation
Haemogram
- Lower RBC count
- Lower Hb count.

**Treatment**
- Correction of primary cause.
- Haematinics:
  - Inj. Imferon (Fe) - on alternate days or twice weekly
  - Cofecu Tab. - 1 tab daily x 15 days
  - Haematinic mixture @ 1 dose x 10-15 days

  One dose haematinic mixture
  - Ferric sulphate - 5 gm
  - Copper sulphate - 0.5 gm
  - Cobalt sulphate - 0.1 gm

- Inj.Vitamin B complex with liver extract.
- Whole blood transfusion or plasma extenders like Haemacel (gelatin, Na, K and Cl) @ 10-15 ml/kg BW in severe cases

**Blood transfusion**
Collect about 500ml of whole blood from the healthy donor animal of the same species in a sterile bottle with an anticoagulant (10ml per 100 ml of blood). Blood from animals could be refrigerated for about a week. Before blood transfusion, blood has to be warmed to the normal body temperature of the species. Transfusion can also be done soon after collection. Transfusion is done through i/v route at the rate of 100 drops per minute. The quantity to be given is about 10-15 ml/kg body weight. Matching blood group is not required in animals for the first transfusion. Anaphylactic shock can occur in the second or third transfusion. To prevent anaphylactic reactions, cross matching of blood for compatibility should be carried out.

Two samples of about 10-15 ml of whole blood is collected; one with an anticoagulant and the other for serum. The whole blood with anticoagulant is centrifuged to separate the blood cells from plasma. The cells are then washed in saline solution. Two to three drops of the donor cells suspension is added to the serum of the donor itself (control 1) in a test tube, and to the serum of the recipient in another test tube. This is then allowed to stand for an hour. Similarly cell suspension of the recipient is added to its own serum (Control 2) and the serum of the donor. Any significant haemolysis detected after 1 hour indicates incompatibility of the blood of the donor and the recipient and therefore in such case, blood should not be selected for transfusion.
4. Oedema

Oedema means excessive accumulation of fluid in the tissue space involving a disturbance in the mechanism of fluid exchange in tissues. Depending on the cause, non-inflammatory oedema can be:

<table>
<thead>
<tr>
<th>Cardiac oedema</th>
<th>Hepatic oedema</th>
<th>Pulmonary oedema</th>
<th>Renal oedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac weakness</td>
<td>Liver damage</td>
<td>Pulmonary infection</td>
<td>Acute glomerulo-nephritis</td>
</tr>
<tr>
<td>Venous congestion</td>
<td>Obstruction in portal circulation</td>
<td>Pulmonary circulation overloading</td>
<td>(−) plasma (+) sodium ions</td>
</tr>
<tr>
<td>Increased vascular permeability</td>
<td>Fluid in peritoneal cavity</td>
<td>Increased capillary permeability</td>
<td>Water reabsorption</td>
</tr>
<tr>
<td>Fluid in tissue spaces (oedema)</td>
<td>Oedema</td>
<td>Oedema</td>
<td>Oedema</td>
</tr>
</tbody>
</table>

**Figure 1.4.** The process of oedema (pathogenesis) based on the organ affected.

Other causes of oedema are

**Nutritional Oedema**

Cause: Blood sucking parasites/ liver fluke/ protein wasting enteritis/

- Protein deficient diet
  - Hypoproteinaemia
    - Decreased plasma protein
      - Decreased plasma osmotic pressure
        - Ascites

**Mammary Oedema**

Cause: Late pregnancy/ early parturition in first calvers

- Increased blood supply to mammary gland
  - Increased vascular permeability
    - Oedema of mammary gland and ventral abdomen
Allergic Oedema
Cause: Contact with Allergen (Pollens, insects etc)

- Increased release of histamine like substances
- Increased blood supply/ capillary permeability
  - Urticaria/ wheels

Mechanical /Obstructive oedema
Cause: Obstruction of blood/ lymph vessels (in Thrombophlebitis, Lymphangitis, tumour, parasites)

- Venous congestion
  - Oedema

Vitamin A deficient Oedema
Vit A deficiency may cause Anasarca (generalised Oedema)

General treatment
- Correction of the primary cause
- Diuretics - Frusemid inj. @ 0.4-4.44 mg/kg BW BID
- Protein rich diet
- Salt free diet
- Rest
- Aminoacids
- Corticosteroids

Cardiac oedema
- Digitalis

Hepatic oedema
- Deworming
- Antibiotics
- Liver extracts

Nutritional oedema
- Plasma extenders- albumin, dextran\(^1\), haemoglobin, hydroxyl-ethyl starch.
- Vitamins and minerals

\(^1\) A branched polysaccharide produced by the action of bacteria on sucrose. Use: blood plasma substitute, food additive.
Mammary oedema
• Glycerine + Magnesium sulphate astringent

Allergic Oedema
• Antihistamines
• Corticosteroids.

5. Dehydration
It denotes the loss of water and dissolved salts from the body.

Normally water intake + metabolic water = water loss through urine, sweat, faeces and respiration. But sometime there is either failure of water intake or excessive loss of water resulting into dehydration.

Failure of water intake due to
• Deprivation of water.
• Decrease thirst
• Inability to drink due to painful lesion in the mouth (Dysphagia- difficulty in swallowing).

Excessive loss of water from the body due to
• Acute gastro-enteritis (Diarrhoea)
• Polyuria due to nephritis.
• Copious sweating
• Skin injury such as burns
• Acute acidosis
• Ascites
• Purgative, diuretic and corticosteroid therapy

Pathogenesis and clinical manifestations
• Breakdown of carbohydrate, fat and protein to produce metabolic water under anaerobic conditions may lead to acidaemia.
• Urine concentration may lead to oliguria and anuria resulting in uraemia.
• Transient fever may occur due to insufficient water to dissipate heat.

Clinical signs
• Sunken eyeball
• Dry mucous membrane
• Loss of skin elasticity
• Lethargy
• Increased capillary refill time
• Twitching of muscles
• Respiratory and circulatory failure
• Shock and animal may die if there is more than 40 % loss of water.
Diagnosis
- From clinical signs (especially skin tenting)
- Laboratory examination involves looking at the Blood picture. Increase in Packed Cell Volume (PVC) indicates dehydration.
- Assessment of degree of dehydration by skin-tent test

Table 1.1. The level of clinical symptoms exhibited and fluid supplementation required for different degrees of dehydration.

<table>
<thead>
<tr>
<th>Degree of dehydration (%)</th>
<th>Sunken eyes</th>
<th>Retention of skin fold (sec)</th>
<th>Haematocrit value (%)</th>
<th>Fluid required (ml/kg b.wt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-8 (Mild)</td>
<td>Not sunken</td>
<td>absent</td>
<td>40-45</td>
<td>15-25</td>
</tr>
<tr>
<td>6-8 (Moderate)</td>
<td>Barely visible</td>
<td>2-4</td>
<td>50</td>
<td>30-50</td>
</tr>
<tr>
<td>8-10 (severe)</td>
<td>Pronounced</td>
<td>6-10</td>
<td>55</td>
<td>50-80</td>
</tr>
<tr>
<td>&gt; 10 (Shock)</td>
<td>More pronounced</td>
<td>20-45</td>
<td>60</td>
<td>80-100</td>
</tr>
</tbody>
</table>

Treatment
- Rehydration Therapy:
  - Inj. Dextrose, Dextrose Normal saline (DNS) - 10-15 ml/kg b.w - slow i/v.
  - Oral administration of fluid.
- Inj. Corticosteroids- i/v or i/m.
- Treatment of primary cause.
Drip should be kept at 30-60 drops per minute.

Table 1.2. Different types of fluid and electrolyte supplements used in dehydration.

<table>
<thead>
<tr>
<th>Type</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal saline (0.9g of NaCl in 100 ml distilled water)</td>
<td>Correct salt and water loss</td>
</tr>
<tr>
<td>Glucose saline (DNS)</td>
<td>Correct salt and water loss</td>
</tr>
<tr>
<td>0.9g NaCl in 100 ml Distilled water containing 5g glucose</td>
<td>Best fluid to initiate fluid therapy.</td>
</tr>
<tr>
<td>Glucose solutions (5%, 10%, 25%)</td>
<td>Correct water deficit, Maintain nutrition (10-25 % DS), Treat hypoglycaemia</td>
</tr>
<tr>
<td>Ringers lactate solution (NaCl- 6.9 g, KCl -0.3 g, CaCl₂ 3.0 g, Sod. lactate-3.0 g, water-1000 ml)</td>
<td>Correct deficiency of sodium and potassium chloride</td>
</tr>
</tbody>
</table>

Table 1.3. Fluid of choice for different clinical conditions in animals.

<table>
<thead>
<tr>
<th>Disturbances</th>
<th>Fluid of choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dehydration (isotonic)</td>
<td>0.9 % saline</td>
</tr>
<tr>
<td>Acidosis</td>
<td>1.3 % Na₂CO₃</td>
</tr>
<tr>
<td>Acidosis with Na⁺ and K⁺ deficit</td>
<td>Ringer’s lactate along with 0.1 % KCl and 0.5 % sod.bicarbonate</td>
</tr>
</tbody>
</table>
Replacement therapy: replacement by saline on a volume per volume basis. Losses from gastric drainage are replaced by isotonic solution containing excess chloride ion and potassium ion.

Maintenance therapy: maintenance therapy is done to compensate ongoing losses.
2. Clinical Disorders of the Digestive System

1. Inappetence and anorexia

![Appetite Diagram]

**Figure 2.1.** Classification of change in appetite in animals.

Inappetance means partial loss of appetite or reduced feed intake while anorexia is the complete loss of appetite.

**Causes**
- Primary
  - G.I disorders (Indigestion)
  - Affection of oral cavity.
- Secondary
  - Febrile conditions
  - Vitamin deficiencies- Vitamin B₂ (thiamine), Vitamin B₁₂
- Apparent
  - Unpalatable feeds.

**Correction**
- Removal of primary cause.
- Vit B complex with liver extract
- Stomachiac/rumentorics.

2. Hyperorexia

It means excessive appetite for solid food

**Causes**
- Heavy endoparasitism (bulimia)
- Pituitary disturbances.

**Correction**
- Treatment of primary cause.

3. Depraved appetite/Pica (Allotriophagia)

Perverted appetite or the tendency of animals to eat abnormal substances.

**Types of Allotriophagia**
- Coprophagia- eating of faeces
Osteophagia- eating of bones
Geophagia- eating of soil
Infantophagia- eating of its own infants
Pilophagia- licking or eating of hair
Iron Mongering- Licking of metallic substances
Licking disease- eating of papers, cloth, wood, leather, etc

Causes
- Vit B deficiency
- Mineral deficiency (Phosphorus, iron, cobalt, etc.)
- Insufficient feed
- Chronic endoparasitism
- Chronic gastritis
- Gingivitis accompanied with teething
- Neurological disorders (Rabies)
- Boredom (Vices in Horse)

Correction
- Removal of primary cause.
- Inj Vit B complex.
- Inj. Tonophosphane
- Vitamin- mineral mixtures - 30 gm daily x 15 days.
- Cofecu tab - 1 tab x 15 days
- Provision of roughage in non-ruminants
- Periodic deworming.

4. Stomatitis
Stomatitis is the inflammation of the mucous membrane of the oral cavity. It often occurs simultaneously with glossitis, gingivitis and palatitis (Inflammation of tongue, gum and palate respectively).

Aetiology

Physical causes:
- Traumatic injuries to oral mucosa due to rough fodder, spines etc.
- Laceration with sharp objects like nails, bone, etc.
- Malpositioning of teeth
- Faulty drenching technique.
- Too hot or to cold food

Chemicals:
- Irritant chemicals and drugs such as acid and alkali, turpentine oil, etc.
Infective causes:
- Virus - Rinderpest, FMD, Mucosal disease complex, Bovine Malignant catarrah, vesicular exanthema, pox, Infectious rhino-tracheitis, canine distemper, etc.
- Bacteria - Streptococci, Staphylococci, Spherothorus necrophorus, Actinobacillus, etc.
- Fungus - Monilla spp, Candida spp, Aspergillus spp.
- Parasites - Plant lies, mites, leeches
- Nutritional - Vitamin B deficiency (riboflavin)
- Allergy - Ingestion of stinging plants and insects.

Clinical sign
- Anorexia due to pain
- Profuse salivation, foaming at the mouth with stingy saliva hanging down from the mouth. Saliva may contain pus or epithelium.
- Smacking of the lips.
- Oral mucosa become red and swollen
- Vesicles (Blisters) on the oral mucosa which might later rupture leaving raw ulcers.
- Halitosis (Foul smelling of mouth)
- Enlargement of local lymph nodes - sub maxillary and pharyngeal lymph nodes.
- Toxaemic reaction in systemic diseases.

Diagnosis
- From clinical signs
- Examination of swab or scrapping to determine the causative agent
- Vesicular fluid should be collected in 50% glycerin buffer for confirmatory tests.

Treatment
- Foreign bodies if any may be removed
- Collutory (Mouth wash)
  - Potassium permanganate (1: 10000) or 0.1% soln.
  - Copper sulphate - 2% soln.
  - Alum - 2% soln.
  - 1-2% Boroglycerine
- Antibiotics - in systemic diseases and to prevent secondary infection.
- Antihistamines
- Inj. Vitamin B complex
- Supportive therapy
  - Inj. Vitamin A and C
  - Fluid therapy.
- Give soft food and grass
5. **Pharyngitis**
Inflammation of the pharyngeal mucosa

**Aetiology**

**Physical**
- Injury from rough hard food and foreign bodies
- Irritation by smoke, gases, etc.
- Too hot or too cold food
- Faulty drenching

**Chemical**
- Ingestion of irritant chemicals and drugs.

**Infectious**
In cattle - IBR (Infectious bovine rhinotracheitis), *Pasturella, Mycobacterium tuberculosis, Spherophorus necrophorus, Actinobacillus corynebacterium*, etc.
In Horse- *Streptococcus equi*, parainfluenza virus, gastrophilus larvae, etc
In pig - *Streptococci, staphylococci, bacillus anthracis*, etc
In Dog- *Bordetella broncoseptica, Streptococci, Staphylococci*, Canine distemper, Canine hepatitis, etc.

**Clinical signs**
- Animal dull and depressed
- Inappetance or anorexia
- Extension of head and neck, protrusion of tongue and open mouth breathing
- Painful cough especially during feeding and drinking
- regurgitation of food and water through the nostril
- Throwing of chewed grass in case of cattle and horse
- High body temperature
- Purulent nasal discharge
- Pawing at the mouth with forelegs in dogs
- Salivation

**Diagnosis**
- From clinical signs.

**Treatment**
- Antibiotics
- Local application of Mendel’s soln.
  - Iodine  600 mg
  - Pot. Iodide 1 gm
  - Peppermint oil 250 gm
  - Glycerin 30 ml
- Sodium iodide - 30 gm in 300 ml of distilled water- i/v
- Inj. Antihistaminics
- Tr. Benzoin inhalation
- Expectorant
- Vitamin A
- Fluid therapy
- Give soft palatable food
- Keep the animal in a well ventilated and clean place.
- Drenching of liquids should be avoided.

6. **Choke (Esophageal obstruction)**
Sudden closure /occlusion of otherwise normal esophagus by foreign body or food mass. It is characterized by hyper salivation, deglutition inability and regurgitation. Depending upon the degree of occlusion of oesophagus, choke can be either complete or partial.

**Etiology**

**Internal occlusion**
- Due to clumsy greedy feeding habit- mango seed, potato, cabbage, turnip, etc may stuck up in the oesophagus while swallowing.
- Foreign bodies- stone, piece of metal, wood, glasses, etc
- Sharp bone, solid food, fish hook, rubber ball, needle, toys, stick, etc

**Mural obstruction** (**space occupying lesions on the esophageal wall**)
Granulomatous mass, papilloma, neoplasms, *Spirocerca lupi, etc.*

**Extra mural obstruction**
- Tuberculosis/abscess of mediastinal lymphnodes
- Neoplasms of cardiac end of stomach
- Oesophageal diverticulum
- Displacement of the heart and lung base.

**Pathogenesis**
In acute choke with complete closure, there is spasm of esophageal musculature and forceful movement of the rumen/stomach to expel the lodged object. Excessive salivation due to parasympathetic stimulation. Slimy saliva softens and lubricate food mass/ foreign object and thereby help to dissolve or dislodge the obstructed mass.

There are three sites of obstruction

a. Immediately behind the pharynx (oropharynx)

b. Cervical part of the esophagus, and

c. Thoracic part of the esophagus
Clinical findings

Cattle and buffalo
- Salivation, restlessness, refusal of food, protrusion of tongue
- Grunting
- Stretching of head and neck from side to side
- Palpation of lump in the cervical esophagus
- Fatal tympany
- Respiratory distress

Sheep and goat
- Restlessness, retching, hyper salivation
- Tympany
- Dyspnoea

Horse
- Anxious look and restlessness,
- Refusal to eat, excessive salivation, regurgitation, cough,
- Pawing on the ground,
- Dyspnoea

Pig
Salivation, restlessness, vomiting, dysphagia and dehydration

Dog and cat
- Distress, dysphagia,
- Salivation, vomiting,
- Foul breath, mouth held high,
- Pawing in the mouth,
- Feeling of mass on cervical part by digital pressure and
- Dehydration

Diagnosis
1. History of ingestion of foreign body
2. Clinical signs
3. Passage of stomach tube
4. Palpation of cervical esophagus for foreign bodies
5. Use of mouth gag to examine pharyngeal obstruction
6. Use of stomach tube to judge the nature and location of obstruction
7. Radiography of full length of esophagus
8. Contrast radiography to locate the obstruction
9. Surgical exploration for prompt diagnosis and repair
Differential diagnosis
- Oesophagitis
- Esophageal dilatation
- Esophageal stenosis
- Oesophagesal diverticulum
- Gastro esophageal intussusceptions
- Esophageal tumor

Line of treatment
- Removal of foreign body using long handled forceps in small animals
- Sedation of animal in acute cases with much discomfort to the animal.
- Atropine sulphate to relax esophageal spasms in large animals.
- Trocarization in large animals to relief from bloat.
- Digital manipulation using a mouth gag for solid obstruction in pharyngeal region.
- Surgical interference

7. Gastritis (Abomasitis)
The inflammation of the mucous membrane of stomach is referred to as gastritis. It is observed in all animals but more frequent in canine and feline animals.

Etiology

Physical cause
- Overfeeding and inferior quality food.
- Ingestion of foreign bodies
- Too cold/ hot food and water
- Abnormal food- moldy food, fermenting green foods
- Poisonous plants
- Food allergy

Chemical causes
- Irritant chemicals- arsenic, lead copper, phenol, etc
- Fungal toxin
- Herbicide, rodenticide, chemical fertilizers
- Strong acid/ alkali
- Drugs- aspirin, phenylbutazone, salicylate, ethylene glycol

Infective causes
- Viruses: malignant catarrh virus, T.G.E., Canine distemper
- Bacteria: *E.coli, salmonella, Clostridium perfringens*, etc
- Parasites: *Trichostrongylus, Haemonchus, Ostertagia*, etc
- Neurogenic causes: stress and strain, fatigue, change of environment
Clinical findings

Carnivorous animals:
- Severe discomfort, abdominal pain, nausea and vomiting. Vomitus may contain blood.
  Undigested decomposed feed in vomitus.
- Offensive smell from mouth
- Intense thirst
- Scant/ less feces
- Muscle weaknesses and flaccidity.
- Tendency to lie down on stomach

Herbivorous animals
- Dull and depression
- Depraved appetite
- Foul smelling feces
- Pain in abdomen. Colic in horses
- Retching
- Offensive breath
- Constipation or diarrhoea

Diagnosis
1. History
2. Vomition, pain on abdominal palpation
3. Radiography and contrast radiography to study changes of gastric mucosa
4. Endoscopy for direct visualization of gastric mucosa
5. Gastric analysis e.g.: pH determination using pH paper

Line of treatment
1. Food be withheld for 12-24 hours
2. Correction of primary condition
3. Antacids 3-4 times a day 10-15 minutes before meal
   - Aluminium hydroxide
   - Magnesium hydroxide
   - Magnesium carbonate
4. Preparations containing chlorpromazine, triflupromazine or ranitidine (histamine blockers) should be given.
5. Gut acting antibiotics- neomycin and streptomycin or sulphanamide to check bacterial fermentation and gas production.
6. Supplementary therapy: Vitamin B- complex to improve GI tone and appetite.

Drugs like salicylates, phenylbutazone, indomethacin, steroid, etc are contraindicated in gastritis.
8. Gastric Ulcer
It is the interruption in the continuity of mucosa/submucosa with the tendency to extend. Gastric ulcers are characterized by abdominal pain, inappetence, constipation or diarrhea along with hemorrhage (melena).

Etiology
1. Physical causes: Foreign bodies, sharp metal and glasses, coarse food, hair balls, etc.
2. Chemical causes: irritant chemicals, corrosive substances, and drugs.
3. Infective causes: Viral, bacterial, fungal, helminthes.
4. Miscellaneous causes: Neoplasms, cirrhosis, uremia, stress in calving, vagal indigestion, abomasal displacement, etc

Clinical signs/findings
- Variable appetite
- Vomiting. Vomitus may contain blood
- Abdominal pain
- Rapid breathing
- Loss of body weight
- Anemia
- Blood in the feces or tarry colored feces
- Peritonitis and death

Clinical pathology
- Examination of vomitus
- Examination of feces
- Blood examination for microcytic anaemia

Diagnosis
- History of food and foreign body ingestion
- Clinical findings
- Radiography/contrast radiography to detect crater
- Fluoroscopy to evaluate gastric motility
- Gastroscopy to identify ulcer
- Cytology through biopsy

Line of treatment
1. Medical treatment as for gastritis treatment
2. Histamine receptor antagonists as Cimetidine and ranitidine
3. Anticholinergic drugs to reduce gastric motility
4. Iron and vit-B complex parenterally
5. Haemostatics to reduce hemorrhage
6. Surgical intervention may be required if medical treatment fails.
9. **Gastro-enteritis**

It is defined as inflammation of the mucous membrane of the stomach and the intestines.

**Aetiology**

**Physical:**
- Overfeeding - excess milk feeding in calves.
- Ingestion of foreign bodies.
- Ingestion of spoiled food like mouldy and decomposed food.
- Defective mastication (chewing).
- Food allergy in dogs and cats.
- Poisonous plants.

**Chemical:**
- Irritant chemicals like acid and alkali.
- Fungal toxins.
- Herbicides, rodenticides and chemical fertilizers.
- Drugs like Aspirin, Phenylbutazone, paracetamol, etc.

**Infective:**

- **Virus** - Rinderpest, Mucosal disease complex, Bovine Malignant Catarrah, FMD Viral rhinopneumonitis, Transmissible Gastroenteritis (TGE) in pigs, Swine fever, canine distemper, infectious canine hepatitis, etc.
- **Bacteria** - *E. coli*, *Salmonella*, *Leptospira*, *Clostridium*, *spherophorus necrophorus*, *Erysipelas*, *mycobacterium paratuberculosis*, *Pasturella*, etc.
- **Fungus** - *Aspergillus spp*, *Monila spp*.
- **Protozoa** - *Eimeria spp.* (coccidiosis).
- **Parasites** - *Trichostrongylus*, *Hemonchus contortus*, *Trichuris*, *Ascaris*, *strongylus*, *Paramphistomon*, *Bunostonum*, *Fasiola spp*, *Monieza* in calves, etc.
- **Deficiency** - Copper deficiency.
- **Neurogenic** - Stress and fatigue due to overworking and adverse environmental changes.
Pathogenisis

Clinical signs
- Diarrhoea. Faeces may be foul smelling and may be green, tarry or black in colour. Sometimes blood may be present (Coccidiosis).
- Partial or complete loss of appetite
- Abdominal pain
- Borborygmus
- Tenesmus
- Dehydration
- Muscular weakness
- Temperature is usually normal but may be raised in systemic diseases.

Gastritis in dogs
- Retching and vomiting
• Increased thirst
• cloudy urine
• Dirty coating of tongue and halitosis

**Diagnosis**

• From clinical signs.
• Examination of feacal sample or faecal culture to establish the causal agent.
• Rule out systemic diseases.

**Table 2.1. Common conditions that must be differentiated for gastroenteritis.**

<table>
<thead>
<tr>
<th>Systemic diseases</th>
<th>Differential symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>FMD, RP, MDC</td>
<td>High body temperature.</td>
</tr>
<tr>
<td></td>
<td>Vesicular lesion in the oral cavity</td>
</tr>
<tr>
<td>Johne’s disease</td>
<td>Bubbles in the faeces, chronic diarrhoea and emaciation (Hidebound condition)</td>
</tr>
<tr>
<td>Poisoning</td>
<td>Body temperature may be subnormal, there may be haemorrhage, in-coordination and convulsion, acute respiratory distress</td>
</tr>
<tr>
<td>Helminthiasis</td>
<td>Gradual loss of body condition, anaemia, potbellied condition and submandibular oedema.</td>
</tr>
<tr>
<td>Facioliasis</td>
<td>Clay or black colored faeces</td>
</tr>
</tbody>
</table>

**Treatment**

• Replacement therapy to replenish lost fluid.
  o Inj. DNS- 10-15 ml/kg b.wt by slow i/v drip.
• Astringent mixtures (substances which helps to control diarrhoea).
  o Neblion powder- Large Animals- 30 gm bid orally
    ▪ Small animals- 110-15 gm
  o Pesulin bolus- Large animals - 2-4 boli bid.
  o Kaolin (natural aluminium silicate)
    ▪ Horse/cattle- 50-250 g
    ▪ Calf and foal- 16-50 g
    ▪ Dog- upto 8 g
  o Bismuth salts (horse and cattle-15-30 g
    ▪ Calf and foal- 2-4 g
    ▪ Dog- 0.3- 2 g
    ▪ Cat- 0.1-0.3 g
• Antimicrobials
  o Sulpha drugs- 2-4 boli daily x 3 days orally
  o Tetracycline tab- 2-4 tab bid x 3 days orally
  o Tetracycline water soluble powder orally
• Inj. Antihistaminics
• Anthelmintics
• Supportive therapy:
In gastritis in dogs
1. Withhold food and water for 24 hours
2. Antacid to neutralize the acidity
   o Tab. Digene - 1 tab tid
   o Triple carb- tid orally
     ▪ Magnesium carbonate- 2gm
     ▪ Bismuth carbonate- 2 gm
     ▪ Sod. bicarbonate- 2 gm
3. Inj. Sedative- trifluropromazine or chlorpromazine
4. Antimicrobials
5. Deworming in case of parasitic infestations
6. Vitamin B complexFluid therapy

10. Enteritis (Enteropathy; Malabsorption; diarrhea)
A chronic or acute inflammation of the entire intestinal mucous membrane characterized by frequent passing of loose feces, flatulence, abdominal distension and borborygmus.

Etiology
Physical agents: over feeding-simple indigestion, coarse food, mouldy food, decompose food
Poisonous plants as Pteridium aquilinum
Chemical agents: corrosive chemicals, arsenic, copper, molybdenum, micro toxin, carbon tetrachloride, cathartics
Infectious agents
  • Bacterial agents: E.coli, Salmonella, etc
  • Viral agents: Rinderpest, BVD, Parvovirus, Rotavirus, Coronavirus, etc.
  • Fungal agents: Aspergillus, Candida, etc.
  • Protozoal agents: Eimeria, Giardia,, Cryptosporidia, etc
  • Helminthes: Paramphistomes, Ascaris, etc
Deficiencies- Copper deficiency, Disaccharidase deficiency in young calf
Secondary causes: peritonitis, renal insufficiency, etc
Diarrhea may be categorized into four types:
1. Osmotic diarrhea: due to poorly absorbed solutes or due to ions like Mg++, sulphates.
2. Secretory diarrhea: Secretory function exceeds the absorptive capacity of small bowel and colon. This type occurs in TGE, Rotavirus, Corona virus infections.
3. Exudative diarrhea: inflammatory reactions lead to ulcerative changes of the small or large intestine. There is accumulation of mucus, blood, proteins, cellular debris in the lumen.
4. Motility dependent diarrhea: absorption of fluid and electrolytes may be dampened due to increased motility of the gut.
Clinical finding
- Rise of temperature in acute case.
- Often goes low in long standing case.
- Diarrhea, mal-absorption and dehydration are the main clinical observations.
- Partial or complete loss of appetite
- Fluid or semi-solid feces containing partially digested foods, mucus, fibrin and blood.
- Feces may have foul or fishy odour.
- Abdominal pain
- Accumulation of gas
- Gurgling sound or fluid rushing sound- borborygmus
- Excessive straining
- Pin-bones smeared with sticky feces.
- Chronic diarrhea may produce anemia in animals.

Laboratory tests
- Helminthoscopy/ helmintho-ovoscopy/ helmintho-larvoscopy
- pH of feces- usually acidic in case of malabsorption
- culture of feces to isolate pathogenic microbes
- D-xylose test for malabsorption
- Lactose intolerance test
- Chemical detection of fecal fat and trypsin level, etc

Diagnosis
- Attempt to differentiate between large bowel and small bowel diarrhea
- Differentiate maldigestion and malabsorption
- Infective diarrhea- high rise of temperature with systemic affects accompanied by toxemia and septicaemia
- Hemogram to delineate the diarrhea-
  o Viral diarrhea- leucopenia followed by leukocytosis
  o Bacterial diarrhea- leukocytosis
  o Dietary diarrhea- animal bright but may pass voluminous feces
- Poisoning- subnormal temperature with dehydration ad haemorrhage.
  o Haemorrhagic enteritis is a feature in Arsenic poisoning
- Helminthiasis (endoparasitism) - slow course, appetite maintained but there is gradual loss of body weight, anemia and protruded abdomen and intermandibular swelling
- Para tuberculosis- bubble in the feces
- Viral disease as RP, FMD, mucosal disease, etc mouth lesion in addition to diarrheal problem.

Line of treatment
Treatment aims at
- correction of primary cause
- correction of dehydration, acidosis and electrolyte balance,
- regulation of diet and
Symptomatic treatment.

Supportive therapy:
- Correction of fluid and electrolyte imbalance
- Drugs reducing motility e.g. Codiene sulphate, loperamide (also antisecretory), etc.
- Intestinal adsorbents like Bismuth, pectin, kaolin and activated charcoal.
- Anti-secretory agents like aspirin, bismuth, chlorpromazine, atropine, etc.

Specific therapy:
- Antimicrobial therapy
- Anthelminthic drugs

Control of diarrhea in new born animals
- Removal of newly born animals from contaminated environment to prevent from further exposure
- Removal of infectious agent from the environment with appropriate sanitary and hygienic measures
- Elevation of non-specific resistance by increasing vitality with proper nutrition
- Elevation of specific resistance by feeding of colostrums within one hour at 6% body weight.

11. Constipation
Difficult or infrequent passage of hard feces

Etiology
1. Lack of exercise
2. Dry hard food especially bones
3. Impaction of colon due to foreign body, bones, hairs, etc.
4. Lack of fibrous food in the diet
5. Unaccustomed environment
6. Lack of bile salts
7. Disease of anal glands and peri-anal region-Abscess, fistula, stricture, etc
8. General muscular weakness
9. Dehydration
10. Lack of calcium and glucose
11. Ascites
12. Astringents and diuretic drugs

Clinical findings
- Diminished frequency of defecation
- Feces dry and hard
- Tenesmus
- Feces with mucus and blood
- Dull and depression
- Gradual diminished food intake with complete anorexia later on.
- Gaseous distension of intestine
Diagnosis

- Retention of feces
- Unfruitful and ineffective attempt to defecate
- Radiography and barium enema to detect stones
- Proctoscopy to detect mega colon

Treatment

1. Correction of the predisposing factors
2. Mineral oil preparations e.g., Paraffin orally or as enema
3. Fibrous diet, bran in the food
4. Calcium therapy orally or parenterally increases rumen motility.
5. Neuromuscular purgatives as neostigmine or carbachol
6. Other laxatives/ cathartic/ purgatives:
   - Biscodyl (Dulcolax®) @ 10 mg (total dose) in dogs and cats
   - Docusates (Laxicon®) @ 5-15 g (total dose) in cattle and horse
     - @ 15-20 mg (total dose) in dogs and cats.
**Rumen dysfunctions**

Rumen function is dependent on the rumen environment which is made optimal by
- A normal rumen pH (5.8 to 7.2)
- An optimal number and balance of micro flora consisting of bacteria, fungi and protozoa.
- A normal rumen contraction (2-3 per minute)

Disturbances of any of these factors will result into ruminal dysfunction. Rumen dysfunction can be:
- Simple indigestion with impaction
- Acid indigestion
- Alkaline indigestion
- Tympany

1. **Simple indigestion**

It can be defined as a simple digestive disturbance which is clinically manifested by inappetance/anorexia, atony of rumen, infrequent defecation or constipation.

**Etiology**

*Dietary factors:*
- Intake of indigestible course roughage, placenta, plastic, leather, etc.
- Ingestion of spoiled and moldy feed.
- Ingestion of large quantities of concentrates.
- Sudden change in feed, for example, sudden change to green pasture.

*Animal factors:*
- Old age
- Faulty mastication
- Depraved appetite
- Greedy feeding
- disturbance of vagus nerve function

*Drugs:*

Prolonged use of oral antibiotics may disturb the microbial population resulting into change in pH and indigestion.

**Clinical sign**

- Animal is dull and depressed
- Inappetance followed by anorexia
- Suspended rumination
- Atony of rumen ( absence of rumen contraction )
- Rumen feels doughy on palpation through the paralumbar fossa
- Occasional grinding of teeth
- Drop in milk yield
- Defecation less frequent which may lead to constipation.
- Faeces may be coated with mucus
- Indigestion with toxaemia may show milk fever like symptoms.
• Temperature, pulse and respiration remain normal.

**Diagnosis**

• From clinical signs
• Rumen pH
• Rumen function tests.

The condition could be confused with some other diseases.

**Table 2.2. Conditions that show similar signs like simple indigestion.**

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Differential symptoms</th>
</tr>
</thead>
</table>
| Acetonaemia (Ketosis) | • Commonly occurs in animals 2-3 weeks after parturition  
| | • Rapid emaciation  
| | • Hypoglycaemia  
| | • Ketone bodies in urine and milk |
| Traumatic reticuloperitonitis | • High body temperature  
| | • Abdominal pain  
| | • Arch back |
| Left abomasal displacement | • History of recent parturition  
| | • bulging and abomasal sound on the left flank |

**Treatment**

• Fluid therapy
• Rumenotorics (Drugs which enhances the rumen environment)
  o Himalayan Batisa-30gm daily orally.
• Correction of rumen pH.
  o In acidic - Antacid - Sod. Bicarbonate  
  o In Alkaline - Vinegar - 5-10 % soln. 200-500ml orally  
• Inj. Vitamin B complex with liver extract.
• Rumenal cud or fluid transplantation from healthy animals.
• Withhold feed for 24 hours and massage the rumen (upwards)
• Surgical intervention - Rumenotomy as a last resort if the indigestion is persistent due foreign bodies in the rumen.

2. **Acid Indigestion (Acidosis, grain engorgement)**

**Aetiology**

• Excessive ingestion of carbohydrate-rich feed which are highly fermentable. E.g. cereals, sugarcane, potato, apples.
• Hotel and ceremonial leftovers.
• Feeding excessive powdered grain
• Decomposed silage
• Enthusiastic feeding after calving.
Clinical symptoms
- Animal becomes anorexic, dull and depressed.
- Distended rumen (but not so much as in tympany)
- Rumen doughy to palpation
- Rumen palpation may reveal fluid waves and sound
- Expiratory grunt
- Tachycardia (increased heart rate)
- Scant pasty faeces.
- Normal temperature.
- Animal may be recumbent.

Table 2.4. The rumen pH and pulse rate in mild, moderate and severe acid indigestion.

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rumen pH</td>
<td>5.5-6.5</td>
<td>4.5 - 5.5</td>
<td>&lt; 4.0</td>
</tr>
<tr>
<td>Pulse/min</td>
<td>60-80</td>
<td>100</td>
<td>120</td>
</tr>
</tbody>
</table>

Pathogenesis

Diagnosis
- History of feeding large amounts of cereal grains.
- Clinical symptoms
Treatment

- Neutralization of acidity.
  - Use of systemic alkalinizer - sodium bicarbonate - 5% solution- 500-1000 ml half by i/v and the other half by oral route. Magnesium carbonate can also be used.
- Use of antibiotics- either orally or by intra-ruminally. Antibiotics are diluted with distilled water to make up volume. - streptopenicillin or oxytetracycline (short acting).
- Inj. Antihistaminics- i/m
- Inj. Vitamin B complex with liver extract
- Rumentorics- H. Batisa
- Last resort: Rumenotomy and evacuation of rumen content with replacement by cud from healthy animals can be performed as the last resort.

3. Tympany (Bloat)

It is a clinical condition where the rumen and the reticulum are over distended with the gases of fermentation either due to excessive fermentation or due to impairment of eructation mechanism in ruminants.

![Diagram showing Primary and Secondary Bloat]

**Primary bloat:** is of dietary origin. Free gas bloat is due to excessive production of gas over and above the rate of eructation and is due to excessive fermentation of easily fermentable feedstuff while frothy bloat is due to trapping of gases in the solid and liquid phase of the rumen content. This is mainly due to the increase in surface tension by saponin, pectins, etc contained in certain feed stuff like legumes.

**Secondary bloat:** collection of gases in the rumen due to failure of eructation as in oesophageal obstruction.

**Aetiology**

- Excessive ingestion of legumes like clover, alfalfa, etc especially when they are wet and the rumen is empty in the morning.
- Ingestion of plants at pre-bloom stage.
- Grazing in lush green pasture
- Excessive intake of highly soluble and fermentable carbohydrate containing feeds like cereals, potato, sugar, and molasses.
- Lack of fibrous feed in the ration.

**Clinical signs**

- Anorexia
- Extension of the head and neck
• Clamping of mouth and grinding of teeth
• Distension of the left flank
• Percussion of the left paralumber fossa reveal drum like sound.
• Dyspnoea characterized by mouth breathing
• Salivation and abduction of elbow
• Tachycardia
• Hypermotility of rumen in early stages but later rumen becomes atonic.

Figure 2.3. Severity of bloat (A=mild, B=moderate, and C=severe)

**Diagnosis**

- From clinical signs.
- Frothy and free gas bloat can be distinguished by trocarisation of the rumen with a large bore needle. Frothy bloat is characterized by small amount of bubbles mixed with rumen fluid. In free gas bloat a large amount of free gases are expelled through the canula or bore needle.

**Table 2.** Differential diagnosis for bloat.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Differential symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oesophageal obstruction</td>
<td>Drooling of saliva and retching</td>
</tr>
<tr>
<td>Tetanus</td>
<td>Rigidity of muscles, protrusion of third eyelid and hypersensitivity</td>
</tr>
<tr>
<td>Black Quarter</td>
<td>High temperature, lameness and localised swelling of muscles particularly on the thigh which crepitates on pressure</td>
</tr>
</tbody>
</table>

**Treatment**

**First aid**

- Withdraw all food and water.
• Keep the animal on a slope with the head at the higher level to relieve pressure on the diaphragm.
• Wooden stick can be tied in the mouth to stimulate chewing and salivation
• Massaging the rumen upwards.
• Make the animal walk.

Emergency treatment
• Trocarisation with Trocar and Canula to reduce pressure.
• In most cases, pricking the rumen with a large bore needle is adequate to relieve the gases.
• Passing of stomach tube to clear obstruction and expelling of gases.

Medicinal treatment
• Carminatives (substances which helps to expel out the gases)
  o Timpol - 30 gm orally
  o Turpentine oil- 30 ml
  o Linseed oil- 300-500 ml
  o Mineral oil /liquid paraffin- 300 to 500 ml
  o Water- 500 ml
• Antizymotics (drugs which inhibit fermentation and therefore prevent formation of gases)
  o Formaline- 30 ml
  o Water- 300 ml
• Antibiotics to control the rumen microflora
  o Tetracycline powder orally
  o Oxytetracycline/ Streptopenicillin - 5 ml diluted in 20-30 ml distilled water- intra-ruminally.
• Antihistamines
• Calcium borogluconate - slow i/v to stimulate rumen contraction
• Rumenotorics - H. Batisa to improve rumen environment.
• Inj. Vit B with liver extract.

In recurrent or repeated tympany:
• Check rumen pH and correct it
• Transplantation of fresh ruminal fluid from healthy animals
As a last resort perform exploratory rumenotomy.

4. Abomasal Displacement
Abomasum may be displaced to the left or to the right. Abomasum is displaced from its normal site on the left side of the abdominal floor between rumen and left abdominal floor. It is a disease of high yielding dairy cows.

Etiology
• Anatomical predisposition as it has no rigid attachment
• Greater curvature of the organ
• Pregnancy
• Atony of the abomasums due to poor exercise
Heavy feeding with high fat and protein diet
Post parturient diseases as hypocalcaemia, ketosis, mastitis, metritis, etc.

Clinical findings
- Left-sided displacement of abomasum
- Inappetance or complete anorexia
- Marked drop in milk yield
- Scanty pasty feces
- Presence of ketone bodies in urine
- Distended abdomen with bulging of lower left paralumber fossa
- Tympany- resonant sound on percussion.
- Animal becomes thin with bulged abdomen
- Ruminal atony
- “Tinkling”, “splashing” or “ping” sound is heard on auscultation on lower left paralumber fossa.

Diagnosis
Clinical examination
- Auscultation with palpation/percussion: auscultation of lower left para lumbros fossa with ballottement will reveal splashing sound.
- Rectal examination: distended abdomen may be felt.
Laboratory examination
- Ketonuria is a constant finding
- Abomasal fluid paracentesis through 10th 11th intercostals space at the middle third of the wall will reveal fluid with pH of 2 and no protozoa.

Treatment
- Rolling of the animal on its back and massaging form left to right
- Surgical correction
- Relief gas by trocarization
- Symptomatic treatment with dextrose, glucocorticosteroids and antibiotics.

5. Traumatic Reticuloperitonitis (Hardware Disease)
Traumatic reticuloperitonitis, or TRP, is a disease in adult cattle caused by the ingestion and migration of a foreign body in the reticulum. Cattle are more likely to ingest foreign bodies than small ruminants since they do not use their lips for prehension and are more likely to eat a chopped feed.

Etiology
- Accidental ingestion of foreign body- metallic objects such as a piece of wire, nails while feeding
- Iron mongering
- Lack of oral discrimination in animal
A large number of adult dairy cattle have metallic foreign bodies in their reticulum without signs of clinical disease. It is likely that a predisposing factor in otherwise normal cows, such as tenesmus or a gravid uterus, causes migration of the foreign body into the reticular wall.

**Pathogenesis**
Foreign body after ingestion is entrapped in reticulum due to honey-comb appearance of reticulum. When it passes through the thoracic cavity, it may pierce lungs and heart. It may also pierce the liver and spleen in abdominal cavity and result in peritonitis.

**Clinical signs**
The classic signs associated with TRP are consistent with an acute, localized peritonitis and include:
- Sudden anorexia
- Ruminal stasis (constipation)
- High fever
- Tachypnea,
- Animal is disinclined to move and
- Arched stance with abducted elbows (indicating cranial abdominal pain).

If the foreign body has penetrated the diaphragm and pericardium, affected cattle also can have muffled heart sounds, jugular pulses, and brisket edema secondary congestive heart failure caused by pericarditis.

**Diagnosis**
- Clinical signs
- Laboratory tests
  a) *Complete blood count* – increased total count with neutrophilia
  b) *Serum biochemical profile* – hyperproteinemia with a hyperglobulinemia.
  c) *Abdominocentesis* – Normal peritoneal fluid of an adult cow is straw-colored, clear, and odorless. Turbid samples or samples containing gross pus or fibrin are indicative of peritonitis, at least locally.
- Other diagnostic methods- contrast radiography of the reticulum

**Complications of TRP**
Reticular abscesses are a common complication of TRP. Also, if the foreign body migrates through the diaphragm and into the pericardium, it can cause septic pericarditis and subsequent congestive heart failure. Less common complications include reticular fistulation, vagal indigestion, and diaphragmatic hernia.¹

**Treatment**

**Conservative treatment**
- Conservative treatment consists of instillation of a magnet to recover or immobilize the metal foreign body if the foreign body is composed of magnetic metal.
- Front feet elevation
- Reduced roughage feeding
• Administration of antibiotics
  Affected cattle should also receive 3-7 days of systemic antibiotic therapy (penicillin, ceftiofur, ampicillin, or tetracycline), stall rest and other supportive therapy as indicated.

Surgical treatment
An exploratory laparotomy/ruenotomy is indicated for removal of the foreign body if a magnet is already in place or conservative therapy is not successful.
3. Disorders of the Respiratory system

Respiratory disorders are more common in young animals and are mostly associated with the presence or exposure to some predisposing factors.

Cardinal signs of respiratory involvement

- Nasal discharge- either clear or purulent
- Cough
- Alteration in rate and character of respiration
- Abnormal lung sound on Auscultation
- High body temperature depending on cause and extent of affection.

Predisposing Factors

- Exposure to adverse climate
- Dusty surrounding
- Overcrowding in ill-ventilated places
- Malnutrition
- Stress
- Aspiration of food

Exciting factors are irritation and inflammation due to:

- Virus: Equine parainfluenza virus in horse, mucosal disease complex, rinderpest, viral pneumonia, etc in cattle, swine influenza and swine fever in swine and canine distemper, kennel cough in canine.
- Bacteria: Streptococci, Staphylococci, Pasteurella spp, Mycoplasma, Pseudomonas, Corynebacterium, etc.
- Fungus: Aspergillus, Rhinosporidium, etc.
- Parasites: Schistosomaisis, *Linguata serrata*, *Oestrus ovis*, Dictyocaulus, Strongyle, Ascaris, etc.
- Allergy: Dust, fumes and pollens.
- Physical: Aspiration of fluid and drugs due to faulty drenching.

1. Upper Respiratory tract Infection (URTI):

The disorders of the nasal cavity (Rhinitis), Larynx (laryngitis), Trachea (tracheitis) and Bronchi (Bronchitis) can be grouped under URTI.

Aetiology

Predisposing causes

1. Exposure of animal to cold and humid weather
2. Keeping animal in ill-ventilated shed
3. Inhalation of dust and smoke
4. Inhalation of chemicals vapour like ammonia, chlorine, sulphur
5. Sudden change of environment temperature from too hot to too cold or vice versa
6. Stress and strain
Exciting causes

Viruses: canine distemper virus, mucosal complex disease and rinderpest in cattle, infectious bovine rhinotracheitis virus, blue tongue virus, atrophic rhinitis, Aujesky’s disease virus in pigs,
Bacteria: Streptococcus, Staphylococcus, Strangles and glanders in horses, Spherophorus necrophorus in pigs, haemorrhagic septicaemia in cattle.
Fungus: Aspergillus fumigatus, Rhinosporidius, Mucor and Rhizopus.
Parasites: Schistosomas nasalis, Oestrus ovis in sheep, Syngamus, etc.
Allergic origin: Dusts, fumes, smoke, pollen grains.

Clinical signs

- Nasal discharge and sneezing
- Cough which may aggravate during feeding.
- Rise in body temperature
- Palpation of these regions may elicit cough
- Lungs clear on Auscultation.

Diagnosis

Examination of nasal swab and nasal scrapings

Treatment

1. Correction of the aetiological factor
2. Flushing nasal exudates with normal saline
3. Antibiotics- procaine penicillin, Streptopenicillin, Sulphamethazine, sulphadimidine may be used.
4. Antiseptic inhalant to remove nasal exudates- oil turpentine, Tr. Benzoin, Tr. Camphor.
5. Chlorbutenol 1% solution (nasal decongestants) in liquid paraffin as nasal drops.
6. Thiabendazol@ 10 mg/kg BW suspension with 10-20 ml water twice daily using nasal tube. Oral thiabendazole @20 mg/kg BW daily for 6 weeks to combat fungal diseases.

2. Pneumonia (Pneumonitis)

It is the inflammation of the lung parenchyma. It is usually accompanied by the inflammation of the bronchioles (bronchopneumonia) or pleura (Pleuropneumonia). The disease is more common in young animals.

Clinical symptoms

- Anorexia and dullness
- Cough
- Nasal discharge - clear or muco-purulent.
- Rapid shallow respiration in early stages and dyspnoea later on.
- High body temperature
- Reluctance to move and tendency to lie down
- Abduction of elbow
- Extension of head and neck with frothy mouth.
- Auscultation reveals moist rales and dry rales in chronic cases.
Diagnosis
- Cultural examination of nasal discharge and swab
- Blood examination for differential count

Sometimes it is possible to tentatively point to a particular group of causal agents as in Table 3.1.

**Table 3.1. Causal agent for pneumonia based on different symptoms exhibited by animal.**

<table>
<thead>
<tr>
<th>Causal agent</th>
<th>differential signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteria</td>
<td>moist painful and paroxysmal cough&lt;br&gt;Neutrophilia</td>
</tr>
<tr>
<td>Viral</td>
<td>Dry unproductive and hacking cough&lt;br&gt;Neutropenia</td>
</tr>
<tr>
<td>Parasitic</td>
<td>oedema of the throat, eyelid and head&lt;br&gt;Eosinophilia</td>
</tr>
<tr>
<td>Mycotic</td>
<td>short moist cough&lt;br&gt;Nasal discharge may contain greenish yellow mass</td>
</tr>
<tr>
<td>Aspiration</td>
<td>Nasal discharge may contain aspirated materials&lt;br&gt;History of drenching with a few days</td>
</tr>
</tbody>
</table>

Treatment
- Antiseptic/inhalation expectorants
  - Eucalyptus oil or Tr. Benzoin - 30 ml in a bucket of hot streaming water.
- Antimicrobials for 5-7 days
  - Sulpha drugs orally or parenterally<br>Antibiotics- Oxytetracycline, Gentamicin - i/m or in severe cases i/v
- Inj. antihistamines
- Inj. corticosteroids
- Inj. analgesics and antipyretics – Analgin, paracetamol, phenylbutazone
- Cough electuary: bid x 5-7 days
  - Ammonium carb - 4 gm
  - Pot. Iodide - 5 gm
  - Pulv Camphor - 2 gm
  - Pulv. Glycerrihzes - 30 gm
  - Treacle - Q.S
  - Caflon powder - 30 gm bid x 5 days
- Supportive therapy
  - Inj Vit A, B and C
  - Dextrose Normal saline if the animal is not eating.

3. Pulmonary Emphysema

It is the distension of the lung cause by over distension of the alveoli. The alveolar wall may rupture with or without escape of air into the interstitial spaces. It is mostly chronic in nature.

Aetiology
- Dust and dusty feed
- Prolonged exhaustive work with poor nutrition
• Chronic bronchitis and Bronchial stenosis
• Parasites - *Dictyocaulus viviparus* (lung worm)
• Allergy - Ingestion of Plants like *Eupatorium adenophorum*

**Clinical signs**
- Occasional cough
- Laboured breathing with open mouth
- Double expiratory movement and formation of heave lines on the costochondral junction of the thoracic rib.
- Auscultation reveal crepitant rales
- Poor exercise tolerance
- Loss of body condition and anaemia
- Death.

**Treatment**
- Animal to be kept in clean well ventilated place
- Inj. Antibiotics - 7 days
- Inj. Antihistaminic - 5 days
- Inj. Vitamin B complex with Liver extract
- Anthelmintics
- Cough mixtures
4. Disorders of Urinary system

Cardinal signs of urinary system disorders
- Abnormal constituents of urine. These are usually protein (albumin), blood, haemoglobin, cells, tissues casts, pus, glucose, ketone bodies, etc
- Abnormal volume of urine
- Pain and dysuria
- Uraemia

1. Nephritis
It refers to inflammation of kidney(s). Nephritis is of three types.

![Nephritis Diagram]

**Figure 4.1.** Different types of nephritis based the tissue affected.

**Aetiology**
- virus - Infectious canine hepatitis
- Bacteria- *E.coli, Streptococci, Corynbacterium renale, Leptospira*
- Parasites - *Dirofilaria immitus, Dictyophyme renale*
- Toxins
- Autoimmune diseases

**Clinical signs**
- The animal becomes dull and depressed
- Slight rise in body temperature
- Polydypsia (Increased thirst)
- Wasting of muscles and loss of body condition
- Rough and staring body coat
- Vomiting.
- Frequent micturition (urination) with pain
- Polyuria followed by anuria
- Cloudy urine
- Urine may contain blood, haemoglobin, cells and tissue cast, pus and albumin. (Blood in the whole of the urine)
- Puffy eyelids in glomerular nephritis
- Stiff gait, arching of back and pain in the lumbar region on palpation.
Uraemia.

**Diagnosis**
- From clinical signs:
- Urine analysis

**Treatment**
- Inj. Antibiotics - for 7-10 days (Gentamicin, neomycin and cephalosporidines are contraindicated)
- Inj. Corticosteriods.
- Inj. Frusemide (Lasix)
- Inj. Vit B complex
- Inj. Dextrose normal saline - slow i/v
- Protein supplement - Aminovit
- Low sodium diet
- Give plenty of water

2. **Cystitis**
Cystitis is the inflammation of the urinary bladder. It is most common in females due to the short urethra. Interference with the act of micturition and retention of urine may enhance bacterial multiplication. Invasion of bladder will lead to shedding of epithelium, erosion and haemorrhage

**Aetiology**
Infection could be either ascending (from urethra) or descending (from kidney)
Bacteria - *Corynbacterium renale, E. coli, Pseudomonas, Streptococci, Staphylococci, etc*

**Clinical finding**
- Animal becomes dull and depressed with inappetance.
- frequent attempt to urinate with small quantity of urine
- Pain during urination (dysuria). Animal remain in urinating posture for sometime even after voiding the urine
- Male dogs adopt squatting posture during urination as in females.
- Abdominal pain
- Urine may be cloudy containing blood (haematuria) and cells. (blood may be found in the last part of the urine)
- Rise in temperature.
- Body coat appears rough and starring.

**Diagnosis**
- From clinical signs.
- Urine analysis.

**Treatment**
- Inj. Antibiotics: for 7-10 days
Urine pH should be considered while using antibiotics here as some antibiotics can act only in acidic pH while some in alkaline pH. Normally urine of herbivorous animals are alkaline while that of carnivorous is acidic.

**Table 4.1. Drugs of choice for different pH of urine.**

<table>
<thead>
<tr>
<th>Acidic pH</th>
<th>Alkaline pH</th>
<th>Both pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxyteracycline</td>
<td>Gentamicin</td>
<td>Sulpha drugs</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Streptopenicilin</td>
<td>Ampicillin</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td>Norfloxacin</td>
<td>Nalidixic acid</td>
</tr>
</tbody>
</table>

- Urinary antiseptics- Hexamine. Urinary acidifier such as sodium acid phosphate has to be given about 20-30 minutes prior to hexamine as it is effective only in acidic pH.
  
  Hexamine- 5 gm
  Sodium acid phosphate- 20 gm to be given orally for 10-15 days

- Urinary acidifiers- acid urine has bacteriostatic effect. - Sodium acid phosphate.

- Inj. Corticosteriod.

- Inj antihistaminics

- Inj. Dextrose-i/v

- In haematuria-
  - Inj. Vit C or vit K.
  - Inj Iron
  - Inj Vit B complex

- Give plenty of drinking water.

**3. Acute renal failure**

A clinical syndrome associated with a rapid decline in the renal function that occurs over a period of hours to days. It is characterized by complications resulting from kidney’s inability to regulate fluid, electrolytes, acid-base balance and metabolic waste products.

**Etiology**

1. Nephrosis- due to toxic causes or ischemic causes
2. Nephritis is usually due to infectious causes.

**Toxic causes (nephrotoxicosis)**

Kidney is highly vulnerable to toxic effects because of high cardiac output (20%). Most nephrotoxins cause necrosis of tubules by way of direct tubular injury, cellular interference, respiration, vasoconstriction of kidney and decreased perfusion.

1. Nephrotoxic drugs
   a. Aminoglycosides- streptomycin, gentamycin, neomycin, kanamycin, amikacin.
   b. Amphotericin B
   c. Tetracycline
   d. NSAIDs.
II. Endogenous pigments- Hemoglobin and myoglobins are nephrotoxic as they cause tubular obstruction and vascular injury

III. Miscellaneous conditions
   a. Ethylene glycol toxicosis
   b. Administration of methylene blue
   c. Hypercalcaemia

Ischemic causes
Prerenal ischaemia
Renal ischaemia due to hypovolemia, decreased cardiac output, renal vasoconstriction, renal vascular thrombosis or systemic vascular dilatation.

Nephritis (infectious causes)
Leptospirosis, ehrlichiosis, bacterial endocarditis.

Clinical signs
- Clinical signs are non-specific.
- Lethargy
- Inappetence
- Vomition
- Diarrhea
- Dehydration
- Oral ulcers (rarely)
- Polyuria in early period and oliguria during later parts.

Diagnosis
1. history- infectious disease, toxins, drugs, prolonged dehydration, etc
2. physical examination- fever in infectious conditions, icterus, ecchymoses, etc
3. Laboratory tests- A correct diagnosis requires one or more of the following laboratory evaluations to be carried out.
   - Serum biochemistry for BUN, Creatinine, and acid-base balance
   - Urine analysis
   - Radiography
   - Ultrasonography
   - Determine urine volume.
4. Serology
5. Renal biopsy, and
6. Determine life threatening complications if any (dehydration, hyperkalaemia and severe metabolic acidosis).

Management
1. Correct fluid, electrolyte and acid-base disorders.
   a. 0.9% saline is the fluid of choice.
   b. Bolus of dextrose (25% or 50%) to correct hyperkalaemia @0.1- 0.5 g/kg BW.
c. Sodium bicarbonate to correct metabolic acidosis @ 1-5 mEq/kg BW diluted to 1.3%.

2. Initiation of diuresis in oliguria
   a. Furesemide @2-3 mg/kg BW bid after administration of fluid
   b. Mannitol 25% @0.25-0.50 g/kg BW IV

3. Other symptomatic treatment as and when required
   a. Antibiotics
   b. Antiemetics
   c. Repeat fluid if required
   d. Administer blood or control bleeding

4. Chronic renal failure
   It is defined as slowly progressive and irreversible impairment of renal functions. Thos slow process allows time for a number of adaptive mechanisms. Some of these adaptive mechanisms are initially beneficial and maintain homeostasis but with time they are non-adaptive and contribute to the progression of the renal disease.

   **Etiology:**
   1. Tubulo-interstitial disorders- chronic interstitial nephritis, amyloidosis, neoplasia, hydronephrosis.
   2. Glomerular disorders- amyloidosis, parasites, glomerulosclerosis, hemolytic anaemia syndrome, idiopathic diseases
   3. Vascular disorders- infarction, emboli, systemic- or glomerular hypertension, hypotension (reduces GFR)

   **Clinical signs**
   Clinical signs are varied and affects all organs
   1. GIT- Uremia causes anorexia, weight loss, nausea, vomition, diarrhea, uremic stomatitis, urineferous breath, etc.
      Polyuria also would cause polydipsia.
   2. Arterial hypertension due to Na+ retention
   3. Neuromuscular consequences- dullness, drowsiness, lathergy, seizure, gait imbalance, coma.
   4. Eye- injected and congested scleral and conjunctival blood vessels.
   5. Anaemia

   **Diagnosis**
   - Clinical signs
   - Laboratory tests-
     ➢ Blood parameters- anaemia, acidosis, azotemia, hyperphosphatemia, hypokaelemia.
     ➢ Fixed creatinine value to a higher level.

   **Management**
   1. Reducing protein intake to reduce ammoniacal compounds.
   2. B-complex vitamins
   3. Antemetics
4. Correction of hypocalcemia
5. Correction of hypokalaemia
6. Correction of metabolic acidosis.

5. **Obstructive Urolithiasis (urinary calculi or stone)**

It means obstruction of urinary tract by formation of urinary calculi or stones. Common sites for urinary calculi are the urinary bladder, kidney, urethra at the ischial arch and the sigmoid flexure.

**Aetiology**
- Feeding high concentrates
- Pasture containing high oxalate and silica
- Hypervitaminosis D (excess of vitamin D)
- Avitaminosis A (Lack of vitamin A)
- High estrogen intake
- Less water intake

**Pathogenesis**

Injury, high oestrogen intake, Avitaminosis A, infections

- Defoliation of epithelial cells
- Nidus formation
- Calculi

Precipitation of solutes like Ca, Mg, NH₄⁺, CO₃⁻, Phosphates, Oxalates

- Change in urine pH
- Enhance solute precipitation
- Urine concentration
- Inadequate water intake

**Figure 4.2.** Pathogenesis of urinary calculi due to different causes.

Calculi are mostly:
- In dogs: Calcium phosphate, calcium oxalate
- In cattle and horse: Calcium/ magnesium/ ammonium phosphate
- In pig: Calcium/ magnesium carbonate, magnesium phosphate
Clinical findings
- Anorexia and lethargy
- Small amount of urine frequently in partial obstruction
- Anuria in complete obstruction. Animal make repeated attempts to urinate but in vain.
- Abdominal pain
- Per rectal examination may reveal distended urinary bladder or flattened bladder if it has ruptured
- Rupture of urinary bladder lead to accumulation of urine in abdominal cavity resulting in distension of ventral abdomen.
- Accumulated urine leads to severe cellulitis, uraemia
- Coma and death

Diagnosis
- From clinical signs
- Abdomencentesis (Pricking of ventral abdomen) may reveal urine

Treatment
- Location of the site of obstruction can be done by passing a urinary catheter.
- Tab cystone 1 tab t.i.d. x 15-20 days
- Inj. Antibiotics
- Surgical intervention- urethrotomy and removal of the calculi.

6. Haematuria
It means presence of whole blood in the urine which imparts a dark brown coloration to the urine. In most cases, haematuria could be confused with haemoglobinuria. These two conditions can be differentiated by either centrifugation or sedimentation of the urine sample.
In Haematuria, the whole blood settles at the bottom while the supernatant fluid becomes normal in color while in haemoglobinuria, there is no sediment and the color of the urine remains unchanged.
7. Urinary incontinence

The condition of constant dribbling of urine due to less voluntary control of micturation is called micturition.

Aetiology

Neurogenic causes
Paralysis of bladder due to damage of nerve supply to bladder. This may result due to trauma, fracture or luxation of vertebrae.

Non-neurogenic causes
This is due to abnormal disposition of urinary organs e.g., ectopic ureter, abnormal urethral sphincter, etc.

Endocrine causes
Estrogen responsive incontinence and testosterone responsive incontinence in castrated dogs

Obstructive causes
This develops due to calculi in the urethra, stricture of urethra, neoplasms of urethra.

Line of treatment
1. Attempt should be made to control the primary cause
2. Measures to prevent infection
3. Empty distended bladder
4. Neurovitamins to induce contraction power of urinary bladder