Chapter four

1. Non infectious disease

1.1. Disturbance of the Digestive Tract

1.1.1. Abnormalities of prehension, mastication and swallowing

a. **Prehension** is the act of taking feed and water into the mouth; it varies in different species of animals. Tongue, lip and teeth are the main organs of prehension. The abnormal act is mostly due to painful conditions of the organs of prehension, e.g. stomatitis, gingivitis, broken teeth, glossitis, paralysis of lips, malposition of jaws, etc.
b. **Mastication** is the function of crushing ability of teeth, which needs the unique mobility of tongue and massive action of the masseter muscles of the cheeks. It is impaired due to painful conditions of the various organs of the oral cavity, e.g. stomatitis, gingivitis, glossitis, or the presence of foreign body. Dropping of food from the mouth clinically manifests due to occlusion or painful reaction in the oral cavity and this condition is called "**cud dropping**".
c. **Swallowing (deglutition)** is the process of transferring materials from the mouth to the stomach through the passage of oropharynx and esophagus. The structures involved in swallowing are floor of the mouth, tongue, hard and soft palate, esophagus, and laryngeal muscles. Causes of inability to swallow includes foreign body, tumor or inflammatory swelling in pharynx or esophagus, painful condition of pharynx or esophagus, esophageal dilatation due to paralysis, esophageal diverticulum, or esophageal spasm at site of mucosal erosion.
d. **Polyphagia** is excessive or increased feed intake. This can be seen in case of starvation, chronic pancreatitis and metabolic disease like diabetes mellitus (chronic hyperglycemia).
e. **Anophagia or Aphagia** is decreased feed intake. It may be due to painful conditions of the mouth and pharynx or to any bacterial or viral infection producing toxemia or septicemia. Vitamin deficiency like thiamin and cyanocobalamin may also produce inappetance.
f. **Pica or Allotriophagia** refers to ingestion of materials other than the normal feed. It is often due to deficiency in the diet such as lack of salt or inadequate trace elements such as phosphorus or copper.
g. **Starvation and Thirst**

Starvation is complete deprivation of feed as in drought, flood, bushfire or human intervention. Starvation may lead to hypoglycemia; acidosis, & ketosis.

Clinical manifestations include: hunger, weight loss, dramatic fall in milk yield, faecal output decline, weakness, recumbency, staggery gait.

Treatment -provide small amounts of highly digestible carbohydrate and proteins orally; avoid lipids, which may exacerbate ketosis.

Thirst: - the subjective sensation of the need to drink fluid which is manifested in farm animals by polydipsia, or allotriophagia in the form of drinking abnormal fluids, especially urine.
Etiology: • in dehydrated animals due to diarrhoea, vomiting, sweating,
• Polyuria, excessive salt intake in the feed, water deprivation or diabetes insipidus.

Clinical findings: partial water deprivation: restlessness, wondering around water supply, aggressiveness, sunken eyes, hollow abdomen, skin tenting; tremor, excitement, frothing at mouth, stiff gait, terminally very excited, urine specific gravity increased.
Treatment: replacement therapy with fluids and electrolyte, slowly in small amounts to avoid water intoxication.

4.1.2. Syndromes associated to digestive tract disturbances and their treatment

1. Diarrhea and Constipation

Diarrhea

Definition: frequent defecation of fluid or semi fluid faces

Etiology: Enteritis; Malabsorption, e.g. due to villous atrophy; Functional diarrhea as in excitement

Clinical findings: increased frequency of defecation; increased faecal water content and volume; thinner consistency of faces; decreased alimentary tract transit time; dehydration.

Diagnosis: History of feed ; change in frequency, color, consistency & elements of faces

Treatment: -fluid therapy & dietary adjustment and removal of the cause of the GIT diseases

Constipation: the decreased frequency of defecation accompanied by dried faces.

Etiology:• Severe debility as in old age; Deficiency of fibres /bulk in the dietary; Chronic dehydration; Partial obstruction of large intestine; Painful conditions of the anus; Paralytic ileus &Terminal stages of pregnancy in cows.

Diagnosis: - retention of faces, unfruitful or ineffective attempt to defecate

Treatment: - correction of predisposing factors, mineral oil preparation, e.g. liquid paraffin by mouth or as enema, fibrous diets, calcium, vit A, &vit B complex therapy

2. Vomiting and Tenesmus

***Vomiting*:** the act of forceful emptying of stomach contents by vigorous contractions of diaphragm and abdominal muscles. It is the most protective disturbance of digestive tract. It is frequently observed in dog and cat, sometimes in pig, seldom in cattle, horse, sheep or goat.

Etiology: Gastric irritation and ulceration; Gastric dilatation, emotional conditions

- Foreign body in oesophagus or pharynx
- Most toxins and some drugs

Vomition *may be central or peripheral*

Central: - occurs when there is stimulation of the vomiting centre in the medulla. It is induced by drugs and toxaemic conditions, e.g. arsenic poisoning, poisonous plants, acute chemical poisoning (phosphorous) or fear and tension

Peripheral- irritation of mucosa of the alimentary tract, e.g. gastritis, gastric dilatation, foreign body in the pharynx, oesophagus, overloading of the stomach,

Clinical findings: restlessness extends head and neck, contraction of abdominal wall, retching, protrudes tongue and large volume of digesta pours from mouth, stomach rupture (a terminal event in horse).

Treatment: elimination of the primary cause of vomition, fluid therapy and antiemetic drugs like chlorpromazine

***Tenesmus*:** it is a condition in which defecation is accompanied by signs of pain like groaning, grunting, with repeated tensing of the abdominal musculature.

Etiology: problem in pelvic cavity (inflammation of lower bowel or lower reproductive tract, e.g. coccidiosis, vaginitis, retained placenta, dystocia spinal cord lesions like abscess, or due to rabies), constipation, or old age.

Clinical findings: frequent straining at defecation

Treatment: the primary cause should be treated.

- Supportive treatment with fluid and electrolyte
- Epidural anaesthesia-to avoid straining

3. Bloat (ruminal tympany)

Definition: it is over distension of the rumen and reticulum with the gases of fermentation, either in the form of persistent foam mixed with the rumen contents or in the form of free gas separated from the ingesta. Thus, there are two classes of bloat**:** ***Frothy or primary bloat*** is dietary in origin and occurs in cattle on legume pasture and in feedlot cattle on highly fermentable grain diets; ***Free gas or secondary bloat or chronic bloat*** is usually due to failure of eructation of free gas because of a physical interference with eructation.

Etiology:

A. Frothy bloat: there are two major factors:

1. Dietary factor-- it is the major cause of frothy bloat. It has a primary and secondary factor

Primary factors include grazing on very green succulent lush pastures or immature (pre-blooming) rapidly growing legumes, excessive intake of cereal crops, peas, beans, cabbage, potato, etc. this is known as pasture bloat.

Secondary factor seen in feedlot, due to feeding finely ground grains that produces enormous quantity of gas under the influence of microbial fermentation. This bloat is known as feedlot bloat. The froth causes physical obstruction to the cardiac and impedes eructation.

2. Animal factors—some are susceptible while others are not. This is due to certain inherited characters. There is also age factor (young animal is more susceptible).

B. Free gas bloat: physical or functional defects in the eructation of the normally produced gas. This is caused by the mechanical obstruction of the esophagus (choke), stenosis of esophagus, interference with esophageal groove function, vagus damage (vagus indigestion), reticulitis, gastritis, tumor mass, etc.

Clinical findings: sudden onset of abdomen distension; Inappetence to anorexia, marked dyspnoea with mouth breathing (extension of the head and neck, protrusion of tongue, abducted elbow),

- Respiratory rate increased, tachycardia, depression, and salivation
- Death usually commence 3-4 hours later.

Note: The cause of death in case of bloat is dyspnoea due to the increased intraruminal pressure by the distended rumen on diaphragm to such an extent that it depresses the cardiovascular and respiratory system. The outcome of this condition is hypoxia and ultimately asphyxia and death.
Diagnosis: Based on the clinical signs **;** Percussion revealing tympanic sound on the left Para lumbar fossa **;** History of access to lush pasture or finely ground grain**;** Season of the year.

Treatment:

- Keep the animal where the anterior portion is elevated over posterior part to reduce pressure on diaphragm
- Trocarization (puncture of the rumen at Para lumbar fossa with trocar and canula) or introduction of stomach tube in case of free gas bloat
- Use of antifoaming agents like mineral or vegetable oil or liquid paraffin (dose500mg-1liter) in case of frothy bloat
- Correction of the predisposing factors.

4. Simple indigestion

Definition: it is a minor disturbance in ruminant GIT function related to a change in the quality and quantity of the diet. It is a milder form of CHO engorgement.

Etiology: or predisposing factors include

- Sudden change of feed

• Poor feed quality, e.g. mould, spoiled and indigestible roughage
• Prolonged oral antibiotic therapy
• Animal fatigue or stress e.g. shipping

Clinical findings: reduced appetite, depression, and absence of rumination, firm and doughy rumen without abnormal distension, normal physical clinical parameters.

Treatment: • Correction of the suspected predisposing factors;

• Correction of the rumen environment by cud transfer from a healthy or slaughtered animal;
• Administration of rumen stimulants like neostigmine or physiostigmine;
• Use of Vitamin B-complex and mineral oils.

5. Rumen over load /CHO engorgement / lactic acidosis

Definition: it is a condition in which large amounts of highly fermentable carbohydrate rich feeds causes an acute disease due to the excessive production of lactic acid in the rumen. It is characterize by ruminal stasis, severe toxaemia, diarrhoea, weakness and recumbency, and dehydration

Etiology: sudden ingestion of toxic doses of carbohydrate rich feeds especially grain. A sudden ingestion of toxic doses of carbohydrate rich feeds results in production of lactic acid that affect the microbial population of the rumen (marked increase in the number of gram positive bacteria) that further increases production of large quantity of lactic acid. This lactic acid will be absorbed in to the blood streams and causes rumen stasis, chemical rumenitis, diarrhea, dehydration, and muscular weakness.

Clinical findings: ruminal distension, abdominal pain (belly kicking) and ruminal stasis, subnormal body temperature, shallow and rapid respiration, diarrhoea, the faeces contain undigested fragments of the feed, dehydration, dullness and anorexia, etc.

Diagnosis: • history of sudden access to toxic doses of grain; Characteristic clinical signs especially anorexia, rumen stasis and depression, rumen fluid PH, complete absence of ruminal protozoa.

Treatment: - correction of ruminal and systemic acidosis by using alkalinizing agents through stomach tube and /or IV by sodium bicarbonates.

- Balanced electrolyte solutions.
- Cud transfer (10-20lit) from healthy animals;
- Restore ruminal motility by using rumen stimulants.
- Prevent animals from further access to high quality feeds, e.g. grain.

6. Colic

Definition: Colic is a complex symptom exhibited by animals when there is painful condition of any abdominal organs. It is common in equine specie, however, it may occur in other animals as well.

Etiology: depending on its cause, colic is classified into the following classes even though they are not mutually exclusive.

a. Spasmodic (spastic) colic: It is characterized by a periodic spastic contraction of the intestinal muscles or visceral pain. It is the most common type in equine species. This may occur due to irritation of GI mucosa by unsuitable foodstuff, excitement, or drinking of cold water following work.

b. Tympanic (flatulent) colic: it results from excessive gas accumulation in the intestinal tract where over distension of viscera stimulates pain and pressure receptors causing mild to severe colic.

c. Impactive colic/ obstructive colic: It may result due to obstruction or impaction of GIT with impacted food mass, stone, sand, foreign body. It can be seen in stomach, cecum, colon, etc.

Causes: physical agents like:

1. Feed related: -coarse roughage may predispose the horse to improper digestion of feedstuffs with a resultant impaction.
2. Water related-insufficient amounts of water create dry ingesta prone to impaction;
3. Poor teeth- similar to poorly digestible feeds, improper mastication causes some impactions.
4. Parasitic agents: migrating larval forms of S.vulgaris interfere with circulation and innervations of various parts of the large intestine, which affects gut motility leads to impaction.

d. Extra-luminal colic: due to mechanical distortion or obstruction of intestine with a consequence of interference with blood supply, e.g. volvulus, strangulation, intussusceptions, etc.

Clinical findings: the cardinal sign of colic is due to abdominal pain manifested by pawing, rolling, sweating, kicking at the abdomen, getting up and down frequently, flank-watching, hyper motility of intestine, increased respiratory and heart rate, absence of faeces.

Diagnosis: clinical findings and history of the animal related to management of feed.

Treatment: treatment should aim to relief pain targeting to the predisposing factors.

- Use of analgesics like flunixinmeglumine, xylazine, phenaylbutazole
- Use of laxatives in case of impactive colic for softening the mass
- Surgical exploration or laparotomy in case of extra-luminal colic
- Regular deworming of horses and improvement of husbandry.

7. Oesophageal obstruction (Choke)

Definition: it is an obstruction or occlusion of esophageal lumen or tube.

Etiology: Inruminants by solid objects, such as apples, pears, potatoes, etc. In horses most frequently due to greedily eating behaviours, by dry grains or a bolus of hay, less often by corn, potatoes, and occasionally on medical boluses.

•Tumors, swollen lymph nodes, abscesses of the surrounding tissue etc.

Clinical findings: The site of obstruction in large animals most frequently occurs in cervical and less often in the thoracic region.

- Sudden stoppage of eating & forceful attempts to swallow;
- Profuse salivation and food and saliva are regurgitated through nostrils; coughing and continuous chewing movements; in cattle bloat and salivation are typical signs.

Diagnosis: from history and prominent signs.

- Cervical oesophageal choke may be located by external palpation or passage of a stomach tube.
- Diagnosis of thoracic obstruction may be confirmed by the careful passage of a stomach tube.

Note: in any animal, signs of difficulty of swallowing and salivation should be considered as a case of rabies

Treatment: a) Conservative treatment:

- Sedate animals to control pain that is caused by obstruction and allow muscles relaxation
- Passage of stomach tube to the obstructed site with repeated pushing with great care and softens with water to loosen the obstructing material and drop down to stomach
- Relief the bloat by trocarization.

b. Radical treatment (esophagotomy): surgical removal of the obstructing materials if conservative measures failed.

8. Intestinal obstruction

Definition: it is the occlusion of the intestinal lumen either from the external pressure or from other physical occlusions or intestinal displacement. It is characterized by abdominal pain,

absence of defecation or often the passage of blood and mucus tinged faces. It occurs in all large animals but is most common in horses.

Etiology: physical obstructions due to intestinal accidents like:

Intussusception is a form of obstruction in which a part of intestine turns in (invaginate) on itself..

Volvulus is a form of obstruction produced by the twisting of a loop of intestine round its mesenteric axis.

Torsion is obstruction caused by the bowel twisting on its own long axis.

Strangulation is an obstruction caused by volvulus or intussusception in which blood supply to a loop of intestine trapped in a hernia form; its development causes tissue necrosis and consequently toxemia and death

Incarceration is a condition in which some part of intestine passes through a torn peritoneum and lie under the skin just like hernia.

Clinical findings: Signs associated with abdominal pain**;** Increased respiratory and pulse rate**;** Dehydration, gastric dilatation**;** Absence of defecation.

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

Treatment: some impaction may be treated with mineral oil. However, most true obstructions usually require surgery (laparotomy), use of analgesics.

9. Rectal prolapse

Definition: is the protrusion of one or more layers of the rectum through the anus due to persistent tenesmus and associated with intestinal or urogenital diseases. Prolapse may be classified as either incomplete (where only rectal mucosa is everted) or complete (where all rectal layers are protruded).

Etiology: severe enteritis, parasites, lacerations, neoplasia of the rectum or distal colon, urolithiasis, retained foetal membrane, dystocia, etc.

Clinical findings: an elongated, cylindrical mass protruding through the anal orifice

Diagnosis is by typical clinical signs.

Treatment: identify and eliminate the cause of the rectal prolapse

replacement of viable prolapsed tissue to its proper anatomic location or amputation of the segment if necrotized & caudal epidural anaesthesia to reduce straining , facilitate repositioning of the prolapse and to permit surgical manipulation.

1.2.Metabolic disease

Amongst domestic farm animals' metabolic diseases are very importance in dairy cows and pregnant ewes. In dairy cows, the incidence of metabolic diseases is highest in the period commencing at calving and extending until the peak of lactation is reached, and their susceptibility appears to be related to extremely high turnover of fluids, salts and soluble organic materials during the early part of lactation. The term "production disease" encompasses those diseases previously known as "metabolic diseases".

1.2.1. Parturient paresis/milk fever/hypocalcaemia

Definition: it is a metabolic disease occurring most commonly about the time of parturition in adult females and is characterized by hypocalcaemia, general muscular weakness, circulatory collapse and depression of consciousness.

Etiology: sudden loss of calcium through milk at or near the time of parturition. Serum phosphorus is decreased but Serum magnesium is increased. Hypocalcaemia causes general muscle weakness, circulatory collapse, depression and unconsciousness.

Clinical findings: there are three different stages of milk fever:

Stage1 is a brief excitement and tetany with hypersensitiveness and muscle tremor of the head, flank and limbs. Animal is able to stand but restlessnes, shuffling feet, bellowing, and open-mouth breathing with tongue extension.

Stage2 is sternal recumbency.

In this case the patient is unable to stand, depression, anorexia, subnormal temperature, loss of defecation and urination, GIT atony (i.e. mild bloat, constipation), decreased heart sound, retained foetal membrane, lies on her brisket with the head characteristically turned into the flank over one shoulder or rested on the ground &eye blinking.

Stage3, lateral recumbency.

Lose consciousness progressively to the point of coma, unresponsive to stimuli, severe bloat because of lateral recumbency and GIT atony, heart sound becomes inaudible and pulse may be undetectable.

Diagnosis: • clinical signs and history of animals (calving, lambing)

- Laboratory tests: total serum Ca, P and Mg determination (hypophosphatemia, hypermagnesemia)

• Favorable response to therapy of IV injections of calcium solutions.

Treatment: IV administration of calcium borogluconate slowly, within 30 minutes of treatment animal will stand and relief from bloat.

Prevention: -feeding diets low in Ca and normal to high phosphorus during late pregnancy

- Administration of Vit D_3 and Ca in the form of Ca gel at calving.

Ketosis in cattle/Acetonemia of cattle

Definition: it is a disease of lactating cows characterized Clinically by loss of body weight, reduced milk yield, nervous signs (e.g. walking in circles, straddling or crossing of the legs, head-pushing, apparent blindness, aimless movements and wandering, vigorous licking of the skin and inanimate objects, depraved appetite and chewing movements with salivation), Laboratoricaly by ketonemia, ketonuria, hypoglycaemia, & ketone bodies in milk.

Etiology: ketosis is basically the result of a negative energy balance early in lactation. High milk production causes energy (glucose) drain and the need for energy exceeds the capacity. A defecit in digestion or metabolism may result in inadequate amounts of glucose (hypoglycemia) available at the cellular level. In an effort to correct this condition, body fat and limited protein stores are mobilized for gluconeogenesis in the liver with a parallel rise in ketone body formation. Theseketone bodies have a toxic effect to the CNS if the level rises beyond the physiological limit. It occurs most commonly during the first month of lactation, less commonly in the second month, and only occasionally in late pregnancy. The highest frequency is between 20-30 days after calving. It can occur in cows of any age, rarely at first lactation, reach its peak at fourth.

The risk factors include low energy intake, high protein intake, excessively fat cows, inter current diseases e.g. milk fever, foot rot.etc…

Clinical findings:

Wasting syndrome: - gradual decrease in milk yield and appetite, weight loss, depression, disinclination to move, ketone odor on breath or milk, ruminal movements decreased in amplitude & number.

Nervous syndrome:- circling, leg-crossing posture, head-pressing, blindness, aimless wandering, vigorous licking of self/fixed objects, depraved appetite, chewing movements with excessive salivation, incoordination, bellowing etc.

Diagnosis: based on clinical findings and history of lactation.

Treatment: IV injection of 500ml 50% glucose solution result in rapid improvement, however, relapse is common; IM administration of glucocorticoids; supportive treatment by oral administration of propylene glycol for 2 days or other glucose precursors to overcome relapse conditions must be included.

Hypomagnesaemia in cow / Lactating Tetany /Grass Tetany /Grass Staggers/ Wheat pasture poisoning

Definition: It is a metabolic disease of cattle, horse, buffalo, sheep and goat characterized by hypomagnesaemia, and usually hypocalcaemia, and clinically by hyper aesthesia, in coordination, tonic-clonic muscular spasms, convulsions, and death due to respiratory failure.

Etiology: a deficiency of magnesium in blood stream is the principal cause of hypomagnesictetany.

Factors affecting magnesium homeostasis:

a. Tetanogenic pasture. Young, green grass has a lower content of available Mg than mature grass, heavy applications of potassium-rich and nitrogen-rich fertilizers reduce the availability of soil Mg.

a. Ammonia formation. Excessive production of ammonia in the rumen from protein rich diet prevents, by a process of chelating the absorption of magnesium.
b. Starvation: during bad weather, transport, and movement to unaccustomed pasture may bring about such a period of partial starvation.
c. Lactation. Considerable quantity of magnesium is excreted through milk during lactation resulting to hypomagnesaemia if not replenished through diet.
d. Scour. Scour causes decreased absorption of magnesium.

Clinical findings:

Acute: • Suddenly ceases to graze ; Unusual alertness and appear uncomfortable

- Staggering gait and fall down with obvious tetany of the limbs that is rapidly followed by clonic convulsions lasting for about a minute
- Opisthotonus, nystagmus, Champing of the jaws , Frothing at the mouth
- Temperature rises to 40-40.5^{o}C, high pulse and respiratory rates

Sub acute: -The onset is more gradual over a period of 3-4 days

- Slight inappetence; Wildness of the facial expression ; Exaggerated limb movements
- Spasmodic urination and frequent defecation are characteristic
- The appetite and milk yield are diminished and ruminal movements decrease
- Muscle tremor and mild tetany of the hind legs and tail with trembling
- Straddling gait may be accompanied by retraction of the head

Chronic tetany: -May not show clinical signs although serum Mg level is low. May show vague syndrome like dullness, unthrift ness, and indifferent appetite. Chronic tetany may turn to sub acute form.

Diagnosis: in coordination, hyperesthesia and tetany are the major clinical abnormalities especially if they occur in ruminants exposed to grazing green cereal crops or lush grass-dominant pasture. Lactating animals are likely to be affected first.

Treatment: combined calcium-magnesium preparation (e.g. 500ml of a solution containing 25% Caborogluconate and 5% Mg hypophosphite for cattle, 50ml for sheep IV followed by a subcutaneous injection of a concentrated solution of a magnesium salt. If signs of respiratory distress or increase in heart rate are noticed the injection should be stopped immediately and, if necessary, a calcium solution injected.

Control: • Feeding of magnesium supplements, 120g magnesium oxide per day.

- Spraying with a 2% solution of magnesium sulfate at fortnightly intervals or by application of very finely ground magnesium oxide to the pasture (30kg/ha) before grazing commences.

- Feeding on hay and unimproved pasture.

Ovine ketosis /Pregnancy toxemia of ewes/ Twin lamb disease/

Definition: it is a highly metabolic disease of sheep and goat that occurs in late pregnancy and characterized by hypoglycemia, ketonaemiaand low liver glycogen content.

Etiology: Negative energy balance and multiple fetuses predispose the disease. A deficiency of blood glucose level is the main cause. This deficiency may be related to low intake of grains or for carrying twin or triplets

Clinical findings:

- Anorexia is the first signs. This is followed by depression of consciousness,
- Twitching of muscles, rapid respiration and ataxia,
- Blind (no corneal reflex) and stand with their head pressed against some object, Walk in circle,

• Spasms of the head and neck muscles, spasticity of the limb muscles, Recumbency, paralysis and death.

Diagnosis: based on the history of pregnancy and inadequate amount of grain feeding. When female goat or sheep is sick in advanced pregnancy, suspect for this disease. • By the typical clinical signs.

Treatment: once the advanced signs have developed, no treatment is effective.

- Cesarean section or induced abortion early in the course of the disease usually leads to recovery.
- Gluco-corticoids are probably only effective through increasing blood sugar.
- Provide palatable feed and water and protection from extreme weather

- Twice daily force-feeding with finely ground-dried grass given by stomach tube may be good practice with especially valuable animals

Prophylaxis-obesity should be avoided in early pregnancy and adequate good feed supplied during the last 6 weeks of pregnancy.

- Minimum changes of feeds should be made during advanced pregnancy,
- Avoid over stress, arrange adequate exercise in pregnancy period, and rule out parasitic burden.

1.3. Mineral and Vitamin Deficiencies/ imbalance

Deficiency of Minerals

At least 15 mineral elements are nutritionally essential for animals. The macrominerals are calcium, phosphorus, potassium, chlorine, magnesium, and sulfur. The trace elements, or microminerals, are copper, selenium, zinc, cobalt, iron, iodine, manganese and molybdenum. The trace elements are involved as component parts of many tissues and one or more enzyme activities and their deficiency leads to a wide variety of pathological consequences and metabolic defects.

Copper Deficiency

Copper deficiency occurs primarily in young ruminants in a range of clinical manifestations including unthriftiness, diarrhea, lameness, demyelination of the central nervous system in neonates, anemia in the later stages of deficiency and falling disease.

Etiology:

1. Primary copper deficiency is due to inadequate intake of copper in the diet when forage is grown on deficient soils or on soils in which copper in unavailable.

2. Secondary (conditioned) copper deficiency is due to impairment in the utilization of the copper by tissues even though blood copper levels are high in response to the physiological needs of the tissues for the element. In this case the dietary intake is sufficient. It is conditioned by the presence of other materials in the diet e.g. molybdenum, sulfate, zinc, iron, and lead, which impede the utilization of copper by body tissue. Molybdenum and sulfate alone or in combination can affect copper metabolism through the formation of insoluble complex of copper-thiomolybdates in the rumen that are not absorbed from the intestine. This effect also operates in the fetus and interferes with copper storage in the fetal liver.

Clinical findings:

- Anemia →. Copper is essential for the synthesis of hemoglobin along with iron.
- Diarrhea (scouring) → high molybdenum content in the diet is usually responsible for this disorder. Diarrhea is usually seen in cattle. This diarrhea is persistent in nature with the passage

of watery, yellow green to black feces with inoffensive odor. The feces are released without effort, often without lifting the tail. It is defined as "peat scours" and "teart".

- Bone deformities → copper deficiency reduces the strength and stamina of bones due to loss of collagen. Bones become porous (osteoporosis) and there is tendency of spontaneous fracture.
- Nervous disorder → copper deficiency produces demyelination of central nervous system. Usually seen in lamb and kids. There is necrosis and neuronal degeneration of spinal cord and brain stem. Such animal shows nervous manifestations known as "neonatal ataxia", "enzootic ataxia" and "sway back". Paralysis is the ultimate outcome of this problem.
- Pigmentary disorder → copper deficiency causes depigmentation of hairs and wools known as "achromotrichia". Copper interferes with the formation of melanin from tyrosine as such black hairs turn grey or brown (bleached). Wools lose crimp, become straight, rough and staring. There are abnormalities in the growth of hairs and wools. Hair color around the eyes is strikingly altered and this is ascribed as "spectacle disease".
- Myocardial degeneration → copper deficiency produces fibrosis characterized by sudden death. This condition is described as "falling disease". Acute heart failure is the cause of death.
- Infertility → copper deficiency has been considered as the cause of delayed estrus in cattle. Copper deficient animals may yield dead fetus and abortion.

Diagnosis: based on history of diet, clinical findings and estimation of copper levels on the soil, diet, blood and liver. Low level of copper reduces hemoglobin level and RBC number.

Treatment: supplementation of copper sulphate through oral route is recommended in clinical cases. Dose: 4g of copper sulfate for calves from 2-6 months of age and 8-10gfor mature cattle given weekly for 3-5 weeks. The requirement of copper is about 5 ppm of the dry diet. 0.5% copper can be added in mineral mixture. Supplementary selenium may enhance copper absorption.

Cobalt Deficiency

Cobalt plays a vital role in the rumen of ruminants for the synthesis of vitamin B_{12} (Cyanocobalamin). Vitamin B_{12} plays a crucial role in the production of red cells. Cattle and sheep are similarly affected and the signs are identical in both species. Cattle are slightly less susceptible than sheep; lambs and calves are more seriously affected than adults. Goat is less vulnerable than sheep.

Etiology: the disease is caused by a deficiency of cobalt in the diet and characterized by anorexia and wasting.

Clinical findings: No specific signs are characteristic of cobalt deficiency. When sheep, cattle or goat are confined to cobalt-deficient diets, there is a gradual decrease in appetite, failure of growth or weight loss, rapid muscular wasting (marasmus), pica is likely to occur especially in

cattle, severe anemia (pallor of mucus membrane), lactation and wool production are severely retarded. In late stage there are infertility, diarrhea and lacrimation.

Diagnosis: - is based on treatment response to oral dosing with cobalt &parentral administration of vitamin B_{12} and determination of vitamin B_{12} content of the feces. On oral dosing with cobalt the signs disappear and vitamin B_{12} in the feces return to normal.
Treatment: - Oral dosing with cobalt or IM injection of vit. B_{12} or oral dosing with large amount of vitamin B_{12}.
Prevention: - the recommended safe level of cobalt in the diet for sheep and cattle is 0.11mg cobalt/ kg dry matter diet, or supplementation of the diet with cobalt is necessary.

Iodine Deficiency

Goiter (thyroid hyperplasia) is the cardinal sign of iodine deficiency. The major clinical manifestation is neonatal mortality, with alopecia and visible and palpable enlargement of the thyroid gland.

Etiology: primarily due to deficient iodine intake or secondarily conditioned by a high calcium intake.

Iodine deficiency reduces the ability of thyroid gland to produce thyroxin hormone. It stimulates the secretion of TSH by pituitary gland. It causes hyperplasia of the thyroid gland and goiter.

Clinical findings: thyroid hyperplasia, loss of libido in the bull, failure to express estrus in cow, hair or wool loss (alopecia), high incidence of abortion, stillbirth and weak newborn animals.

Diagnosis: easily diagnosed if goiter is present but the occurrence of stillbirths without goiter may be confusing. Estimation of iodine levels in the blood and milk is needed.

Treatment: potassium iodine 200mg/kg dry feed or provision of iodine salt preparations. Recommended intake of iodine for lactating and pregnant cows is 0.8-1mg/kg dry feeds; for dry cow are 0.1-0.3mg/kg dry matters. Treatment must be undertaken with care, as over dosage will cause toxicity.

Iron Deficiency

Functionally iron is necessary for hemoglobin formation. A deficiency of iron in the diet causes anemia and failure to thrive. It is the most common problem of piglets kept under artificial conditions.

Etiology:

•**Primary:** is most likely to occur in newborn animals whose sole source of iron is the milk of the dam, milk being a poor source of iron. Deposits of iron in the liver of the newborn are

insufficient to maintain normal hemopoiesis for more than 2-3 weeks, and are particularly low in piglets.Continued blood loss by hemorrhage in any animal may bring about a sub clinical anemia and an associated iron deficiency.

•**Secondary**: due to chronic bleeding, high infestation of cattle with sucking lice, high burden of blood-sucking strongylid parasites in horses.

Clinical findings: the highest incidence occurs at 3 weeks of age although the disease can occur in pigs up to 10 weeks of age. Significantly lower growth rate of anemic pigs than normal pigs. Obviously reduced food intake.

• Diarrhea is very common, but the feces are usually normal in color. Severe dyspnea, lethargy, and marked increase of the apex beat with exercise. The mucosa and skin are pale; Edema of the head and forequarters.

• Death usually occurs suddenly.

Diagnosis: is based onclinical findings, age (neonates are highly affected), laboratory test to determine Hb level and treatment response (IM injection of iron-dextran preparations or other iron preparations).

Treatment: the treatment is usually parenteral and consists of organic iron preparations such as iron-dextran, iron-sorbitol-citric acid complex, iron sacccharate or gluconate. The dose rate is 0.5-1g elemental iron in one injection once each week. Vitamin B_{12} is often used in the same injection at a dose rate of 5000 mg per week in a single dose.

Calcium, Phosphorus and Vit D Deficiency

A dietary deficiency or disturbance in the metabolism of calcium, phosphorus or vitamin D, including imbalance of the calcium: phosphorus ratio, is the principal cause of the osteodystrophies. The interrelation of these various factors is often very difficult to define.

Calcium Deficiency (hypocalcicosis)

Calcium deficiency may be primary or secondary, but in both cases the end result is an osteodystrophy, the specific disease depending largely on the species and age of the animals affected.

Etiology: Primary calcium deficiency is due to an absolute deficiency in the diet. This occurs rarely.

Secondary calcium deficiency is due to marginal calcium intake aggravated by high phosphorus in the diet. Such a diet depresses intestinal absorption and retention of calcium in the body, and the absorption of calcium from bones is increased. This can also occurs accompanied by a vitamin D deficiency in animals confined indoors.

Clinical findings: the Clinical signs, apart from the specific syndromes, are less marked in adults than they are in young animals.

Non-specific syndrome includes: - decreased rate or cessation of growth

- Dental mal-development which is characterized by deformity of the gums, poor development of the incisors, failure of permanent teeth to erupt for periods of up to 27 months and abnormal wear of the permanent teeth due to defective development of dentine and enamel, occurring principally in sheep.

- Soft bones, bendable ribs, easly fracture.
- Reduced fertility, difficult parturition.
- Tetany in pigs and young cattle.

Specific syndrome includes rickets, osteomalacia, osteodystrophiafibrosa, bovine degenerative arthropathy, and ovine hypocalcemic paresis.

Diagnosis: clinical signs, and diagnostic response to diet supplementation with calcium.

Treatment: -calcium borogluconate solution injection (as in milk fever) for tetany

- Supplement diet with calcium (ground limestone, bone meal)
- Reduce excessive phosphorus intake
- Aim at calcium: phosphorus ratio of 2:1 (optimum) up to 1:1; if urolithiasis is a problem ratio should be 2.5:1.

Phosphorus Deficiency (hypophosphatosis)

Phosphorus deficiency is usually primary and is characterized by pica, poor growth, infertility and, in the later stages, osteodystrophy.

Etiology: Phosphorus deficiency is usually primary under field conditions but may be exacerbated by a deficiency of vitamin D and possibly by an excess of calcium. Unlike calcium, a dietary deficiency of phosphorus is widespread under natural conditions.

Clinical findings: primary phosphorus deficiency is common only in cattle.

Young animals grow slowly and develop rickets. In adults there is an initial sub clinical stage followed by osteomalacia. Retarded growth, low milk yield and reduced fertility are the earliest signs of phosphorus deficiency. Osteophagia is common and may be accompanied by a high incidence of botulism. Cows in late pregnancy often become recumbent and although they continue to eat are unable to rise.

Diagnosis: based on clinical findings

Treatment & control: urgent treatment is 30g sodium dihydrogen phosphate in 300ml water IV for cattle. Daily supplementation for cattle of 15g phosphorus minimal, 40-50g optimal, as bone meal, rock phosphate, soft (colloidal clay) phosphate in form of free-access lick, mineral mix fed in concentrate or hay, pasture, in drinking water.

Vitamin D Deficiency (hypovitaminosis D)

Vitamin D deficiency is usually caused by insufficient solar irradiation of animals or their feed and is manifested by poor appetite and growth, and in advance cases by osteodystrophy.

Etiology: a lack of ultraviolet solar irradiation of the skin, coupled with a deficiency of calcium and phosphorous. Fish liver oil is the richest source of vitamin D.

Clinical findings: reduced productivity, poor weight gains, inappetence, and infertility. In late stages there is rickets in young and osteomalacia in adults.

Diagnosis: based on history of feeding vitamin D deficient diets and less exposure to sunlight, clinical signs, response to vitamin D therapy.

Treatment and control: Arrange exposure to solar irradiation; Include sun-dried hay in diet

Deficiencies of Vitamins

Vitamin A Deficiency (hypovitaminosis A)

Vitamin A is essential for the regeneration of the visual purple necessary for dim-light vision, for normal bone growth and for maintenance of normal epithelial tissues. Deprivation of the vitamin produces effects largely attributable to disturbance of these functions.

Etiology: Vitamin A deficiency occurs either as a primary disease, due to inadequate intake of this vitamin or its carotenoid precursors in the diet, or as a secondary disease in which the dietary supply of the vitamin or its precursor is adequate, but their digestion, absorption or metabolism is interfered with to produce a deficiency at tissue level. Major sources of this vitamin are preformed vitamin A from animal sources (fish oil, egg yolk, liver) and provitamin carotene from plants. Dark green leafy vegetables are the good sources of carotenes.

Clinical findings:

- Poor appetite and emaciation in growing animals
- Impairment of vision (*night blindness*)
- Skin thickening and keratinization (xerodermia)
- Conjunctivalxerosis: dryness, loss of transparency, thickening, wrinkling
- Corneal xerosis and ulceration,
- Xeropthalmia (dry eye)
- Susceptible to infection, so this vitamin is considered as anti-infection vitamin.
- In the urinary system, there is degeneration and cornification of epithelium. These favor the formation of renal stones. Low vitamin A and high calcium diet accentuates the formation of calcium phosphate calculi in the bladder and kidney.

Diagnosis: based on history of diet, clinical accounts, estimation of vitamin A level and respond to vitamin A supplementation.

Treatment: High dose of vitamin parenterally, even ten times of it may be warranted in some vulnerable sick animals. Doses: Calf 1,000,000 to 1,500,000iu, cow 250,000 to 2,500,000iu, lamb 125,000 to 250,000iu, horse 200,000 to 300,000iu.

Vitamin K Deficiency

This vitamin is adequately available in the plants and synthesized by herbivorous animals in the gut. Therefore, deficiency of it is rarely seen in animals.

Etiology: - prolonged use of oral antibiotics can retard the synthesis of this vitamin due to reduction or destruction of beneficial bacteria. High dosing with vitamin A can also interfere with bacterial synthesis of this vitamin.

This vitamin helps the formation of prothrombin and takes part in the normal coagulation process. Thus deficiency of vitamin K causes prothrombinemia and hemorrhage. Bile salt helps its absorption.

This vitamin should be used when there is bleeding, e.g. bleeding from nostril or other orifices, hepatitis and hemorrhagic gastroenteritis, bleeding due to warfarin and salicylate poisoning.

Treatment: in horse and cattle 80-250mg or 20mg/kg Bwt IM at 12-hour interval till bleeding ceases.

Vitamin B Complex Deficiency

Vitamin B complex deficiency seldom occurs in ruminants as they can synthesize adequate amount of these vitamins in the rumen through the bacteria. But, prolonged use of antibiotics and sulphonamides through oral route cause reduction of bacterial population and thus impede the synthesis of this vitamin. High carbohydrate diet may also reduce the bacterial population and synthesis of this vitamin.

Ascorbic acid (Vitamin C)

It regulates oxidation-reduction potential. It acts as co-enzyme in the oxidation of tyrosine and phenylalanine. It helps wound healing. It maintains capillary integrity.

Deficiency signs: scurvy in human beings. Infertility, slow healing and susceptibility to infection in animals.

Selenium And/ Or Vitamin E Deficiency

Selenium is an essential nutrient for animals. It is as a component of the enzyme glutathione peroxidase, which protects cellular membranes and lipid containing organelles from peroxidative damage acting in conjunction with vitamin E to maintain integrity of these membranes.

Etiology: selenium deficiency occurs in animals fed plant material grown on soils poor in the element. Vitamin E deficiency may be caused by a primary deficiency in the feed, or by the presence in the diet of polyunsaturated fatty acids that destroy the vitamin.

Clinical findings: The clinical effects are manifested principally in skeletal muscles but occur also in most other organs.

A. Acute enzootic muscular dystrophy

Sudden death, without prior illness, often after exercise, excitement, sudden onset of dullness, dyspnea, frothy, sometimes blood-stained nasal discharge in some.

• Many cases laterally recumbent

•Heart rate 150-200/minut, often grossly irregular

• In spite of treatment, death may occur after 6-12-hour course.

B. Sub acute enzootic muscular dystrophy (white muscle or stiff lamb disease)

• Sternal recumbency; Patient anxious to stand but most unable to do so
• Standing patients have rigidity, tremor, stumbling gait, easy falling
• Weakness and stiffness followed by paralysis
• Large, upper limb muscle masses symmetrically swollen, firm
• Myopathy as muscular degeneration or "white muscle disease"
• Retention of placenta in cattle
• Dyspnea, transient fever in some
• Heart rate moderately elevated

Diagnosis: history of diets, clinical signs, lesions in muscles and estimation of vitamin E. Respond to vitamin E therapy may confirm diagnosis.

Treatment: administration of vitamin E and selenium-containing medicine.

4.4. Diseases Caused By Chemical Agents and Poisonous Plants

A. Lead poisoning (plumbism)

Lead is one of the commonest causes of poisoning in dogs and farm animals, particularly cattle, sheep and horses especially young cattle.

Etiology: - accidental ingestion of sources of lead compounds or ingestion of feed, usually forage, containing lead from pollution of the environment, e.g. licking lead-bearing paint and metallic lead, lubrication grease, car battery, grazing beside rubbish dumps, discarded paint cans Small proportion is absorbed usually in the form of lead acetate or carbonate and deposited in the liver and kidneys in acute poisoning and in the bones in chronic poisoning, a small amount also in the brain.

Clinical findings: -

Acute form: - is common in young animals.

• Staggery gait, muscle tremor, salivation, eye rolling, bellowing, blindness, hyperesthesia (oversensitive), convulsion, head pressing and death.

Sub acute form: - common in adult**.**

- Extreme depression, immobility, complete anorexia, blindness, staggery gait, tremor, hyperesthesia, salivation, teeth grinding, kicking at belly, complete ruminalatony, initial constipation followed by fetid, black diarrhea, recumbency, quiet death after 3-4-day course.

Treatment: If there is extensive tissue damage, particularly of the nervous system, treatment may not be successful.

The following treatments are used singly or in combination:

• Immediate relief of acute nervous signs in calves by IV pentobarbital sodium
• Emptying of rumen and reticulum completely, supplemented by wash-out
• Calcium versenate 150mg/kgBW
• IV or Sc (110mg/kgWB) Calcium disodium edetate (CaEDTA)
• Parenteral thiamin hydrochloride (2mg/kgWB) reduces deposition of lead in tissues.

B. Common salt poisoning (sodium chloride poisoning)

Definition: the ingestion of excessive quantities of sodium chloride causes inflammation of the alimentary tract with the production of gastroenteritis and diarrhea. It is called "water deprivation sodium ion toxicosis" because it can be resulted when excessive quantities of salt are ingested and intake of potable water is limited.

Lactating females are most susceptible.

Etiology: •is directly related to water consumption.

- Thirsty cattle get access to saline bore water
- Animals on low salt diet allowed access to ad lib salt
- Animals on normal salt intake (2% of ration) temporarily deprived of water
- Prepared feeds to housed cattle contain excess salt

Clinical findings: • In cattle and sheep signs of acute poisoning include: -

Salivation, increased thirst, vomiting (regurgitation), abdominal pain & diarrhea, opisthotonus, nystagmus, tremor, blindness, paresis, fetlock knuckling, recumbency, convulsions, death after course of 24 hours.

Treatment: • the toxic feed or water must be removed immediately

In advanced cases animals may be unable to drink and water may have to be administered by stomach tube

Symptomatic treatment includes alimentary tract sedatives when gastroenteritis is present and the provision of isotonic fluids when dehydration has occurred. When there is evidence of cerebral edema it may be necessary to administer a sedative and cerebral decompression may be attempted by use of diuretics or hypertonic solutions injected parenterally.

C. Nitrite poisoning: Nitrate is a source of nitrite; nitrite poisoning causes a syndrome of respiratory distress because of the formation of methemoglobin (nitrite ion in contact with RBC reacts with hemoglobin to form a stable methemoglobin), which results in anemic anoxia.

Etiology:the nitrate may be reduced to nitrite in the plant before ingestion often hay in the stack (if hot and wet), overheated in the sun or damp for some time before feeding, by bacterial action or heating. The formation of methemoglobin results in anemic anoxia.

Clinical findings: dyspnea, tremor, stumbling gait, and mucosal cyanosis, fast, weak pulse.

Treatment: • methylene blue IV 1-2mg/kg as 1% solution

• Feeding chlortetracycline or sodium tungstate reduces nitrate to nitrite conversion.

D. Bracken fern poisoning

In ruminants the effect is depression of bone marrow activity with pancytopenia expressed primarily as ecchymotic hemorrhages and often followed by bacterial invasion of tissues. There is also a relation between bracken and the disease enzootic hematuria. Carcinomas of the intestine and urinary bladder in sheep and cattle have also been attributed to the ingestion of bracken for long periods. It is a retinal degenerant that causes bright blindness.

Clinical findings:

a. Pancytopenic disease of cattle: there is an access to bracken for from 2 to 8 weeks. Initially there is loss of condition and dryness and slackness of the skin. Clinical signs occur suddenly and include high fever (40.5-43c), dysentery or melena, bleeding from the nose, eyes and vagina, and drooling of saliva, nasolabial ulcers, petechiae in mucosae and skin, death after 1-3 days in most.
b. Enzootic hematuria: high incidence of bladder carcinomas, intermittent high-volume hematuria, hemorrhagicanemia, discrete, firm lesions palpable rectally in bladder wall, entire wall thickened in advanced casts, cystitis a common complication.
c. Bright blindness of sheep: in sheep on bracken for years, blind but bright, alert, pupils dilate, poor light, menace reflexes, retinal degeneration, leukopenia.
d. Alimentary tract neoplasia: carcinomas in sheep on long-term grazing bracken pasture, lesions in small intestine, liver, mandible, pharynx, esophagus of cattle.

Diagnosis: based on pasture, feeding, season, clinical findings, especially high fever, sudden onset in acute form, large hemorrhage.

Treatment: thiamine 50-100mg once a day for 6 days, batyl alcohol (to stimulate bone marrow function) plus antibiotics (to control secondary complication), vitamin B complex IM as a supportive treatment to stimulate appetite.

E. Fluorine poisoning

Fluorosis is a chronic disease caused by the continued ingestion of small but toxic amount of fluorine in the diet (drinking water). It is characterized by mottling and excessive wear of developing teeth and osteoporosis. Acute fluorine poisoning usually occurs as a result of the inhalation of fluorine-containing gases or accidental administration of large amount of fluoride and is manifested by gastroenteritis. Irrespective of treatment used, no improvement in dental or osseous lesions can be anticipated but amelioration of the other clinical signs may occur.

4.5. Allergy and anaphylaxis

Trauma/wound, stress

4.3.1 Diseases Due To Physical Agents:

A. Heat (sun) stroke (Hyperthermia or heat stress): It is a disturbance of the heat regulating mechanism of the body due to high environmental temperature, high humidity and inadequate ventilation.

Etiology: • prolonged exposure to high environmental temperature

- Predisposing factors: physical effort (muscle exertion), obesity and stagnation of the air.
- Animals like dog, suffer from hyperthermia due to their inability to sweat. In addition sheep with fleece and large dense cattle are especially prone to heat stroke.

Clinical findings: elevation of temperature

- Increased heart rate and respiratory rate
- Sweating and restlessness
- Weakness, depression, panting,, frothy discharge from mouth and nostrils, tachycardia, protrusion of tongue, congestion of mucus membrane of conjunctiva, cardiac arrhythmia, muscle tremor, coma and death.
- In pregnant animals hyperthermia may lead to abortion

Treatment: •cold applications like immersion in water or spraying of water and rectal enema.

- Provision of adequate drinking water, shelter and ventilation.
- IV administration of isotonic saline solutions

B. Cold (over-cooling, hypothermia)

It is a disturbance in thermo regulation accompanied by drop in body temperature below the normal level.

Etiology: •increase in heat loss due to exposure to cold stress; wet skin exposed to high air current

• Decrease in heat production or combination of both factors.

-Newborn animals born in cold weather and their mother is incapable to lick it dry.

- Emaciated and malnourished animals are more susceptible to over-cooling**.**

Treatment: •Adequate covering with sack or blanket

- Dextrose 5-10% solution IV in severe cases
- Corticosteroid preparation to prevent shock.

C. Burn

Definition: the destruction of epithelium or deeper tissues by direct heat, radiant heat, flames, electricity or corrosive chemicals. Destruction of skin causes fluid loss, entry of bacteria, dehydration, toxemia, and shock.

Clinical findings: Reddening of the skin, formation of vesicles and blister, diffuse edema of the skin and subcutaneous tissue, charring and devitalization of tissue, sloughing of the skin, constant exudation or effusion of serum, considerable loss of protein and fluid, reluctant to move, shock, and implications including infection

Treatment: •if burns are severe and involve more than 50% of the body, euthanasia should be considered.

- In less severe and small area burns, initially the application of cold packs may reduce pain and edema, clipping of the hair over and around the burn, remove the sloughing tissues and debris and clean with iodine or saline solutions, local application of antibacterial creams e.g. silver sulfadizine.

D. Trauma

It is a condition in which part of the body is damaged by wound or injury. Depending on its nature trauma can be classified in to: -

1. Blunt trauma: - is commonly associated with thoracic and abdominal bleeding, organ rapture, fractures and neurological injuries.
2. Penetrating trauma: - is localized to the path of penetrating object, e.g. animal bite, gunshot etc.

Etiology: sharp objects, car accident, gunshot, animal bite, etc.

Diagnosis and treatment: -The traumatized animal should be approached as if multiple injuries are present. The neck and spine should be immobilized until a thorough examination for spinal fracture, thoracic auscultation and abdominal palpation is done.

- Active hemorrhage should be stopped with pressure.
- Bandages must support fractures of the extremities.
- Keep careful monitoring in the 12-24 hours after the trauma occurred, which would allow early detection of potentially life threatening complications.

Assignment and Home study #2, from Chapter 2, 3 and 4 of the hand out and the given reference (20-Point)

Q1. Farm animal Diseases can be categorized as Vector-borne diseases, Soil-borne diseases & Contact diseases.

- Clearly describe each category with at least three examples?

Q2. What is the difference between infectious and non-infectious disease? Give at least 3 examples for each?

Q3. Briefly describe and define the concept Etiology, Epidemiology, Pathogenesis, clinical sign and Diagnoses of an infectious disease?

Q4. Farm animal diseases can be Bacterial, Viral, Parasitic, fungal, metabolic diseases and etc.

- **Give at most 5 examples for each category?**

Q5. Write a short note (Definition, Etiology, Epidemiology, Diagnoses, prevention and control) of the following disease?

1. Shipping fever

2. Mastitis

3. Fascioliasis

4. Sheep pox

5. Fowl pox

6. Rabies

7. Taeniasis

8. Infectious Bursal disease