Hand book
of
Veterinary Internal Medicine

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Second Edition

SUMMERY OF IMPORTANT INTERNAL MEDICINE DISEASES FOR FIELD VETERINARIANS

PREFACE

This book is intended to be used as quich reference for those involved in farm animal care.

We spend along time in choosing, collecting and arranging the material found in this book according to the best, recent, international texts and references.

This book is arranged in four parts

1. Field Cases of Internal Medicine Diseases.
2. Key to Diffrential Diagnosis.
3. Clinical and Laboratory Diagnosis.
4. Therapeutic Index.

This book is supported with many colored clinical illustrated photos distributed in 18 colored plates.

We hope this book will fill a gap in the veterinary field in Egypt and the Arabian contrier.

Hamed Attia and Hatem Selim

Content

Part I

Part II

1. Key of diffrential diagnosis

Part III

Clinical examination

Stomatitis

1. Definition and Causes

It is inflammation of the oral mucosa includes glossitis and gingivitis. It may be due to trauma, foreign body injury, sharp teeth, and irritant substances. Avitaminosis, Bacterial, Viral, or Mycotic infections. Secondary Stomatitis such as Foot and mouth disease, Actinomycosis Rinder pest & Malignant catarrhal fever.

2. Clinical Findings:

Partial or complete anorexia. Profuse salivation, slow and painful mastication. Changes in the mucous membrane of the mouth (Catarrhal, Errosive, Follicular, Vesicular, Pustular, Puppular, Pseudo-aphthus, Ulcerative, Diphtheretic, Gangrenous, Phlegmonous, Mycotic Stomatitis or Allergic Stomatitis). Fetid odour is present in breath. Fever may be present if stomatitis is secondary to systemic diseases

3. Treatment

R/ Gentian violets 2%.
Wash the mouth cavity with mild antiseptics 3 times daily.

R/ Tincture iodine 0.5-1% (in case of ulcerative stomatitis)
Touch the ulcer 3 times daily.

R/ Tincture iodine 3.5% in 10% glycerin Paint the mouth cavity after (in case of diphtheretic stomatitis)

R/ Atropine sulphate 1% 3-5 cc / cattle & horse S/C or I/ M or I/V. (to decrease salivary secretions)

R/ Pentomycine (pencillin & streptomycin) 1 ml/25 kg BW I/M once daily / 3-5 days.

R/ Dextrose 25% 1 - 2 liter, I/V (as supportive treatment)

4. Important Notes

1. You may use syringe without needle for washing the mouth.
2. Easily digested food as barseem, bran mash or rice and soup.
3. Isolation of the diseased animals in case of infectious disease.
4. Mild antiseptics can be used as 1 % boric acid, 5% alum water, 2% copper sulfate, spoonful of vinegar / liter water and/or 2% potassium permanganate.

Parotitis

1. Definition and Causes

It is inflammation of salivary glands (parotid, sub maxillary and/or sublingual). It is caused by mechanical injuries as trauma from outside or entrance of foreign body or specific infectious disease as Tuberclosis and Actinobacillosis in cattle and strangles in horse.

2. Clinical Findings:

Enlargement of the gland (may be seen and palpated in upper & lower pharyngeal region). Difficult in prehension, mastication and swallowing. Abscess may develop in the gland and evacuate in the mouth cavity. Profuse salivation. The inflammation may extend to the larynx causing edema. Salivary cyst "permanently enlarged" due to the entrance of small food particles in the excretory duct.

In chronic form, painless solid swelling of the gland is found.

3. Treatment

R/ Apply hot fomentation.

R/ Iodine or camphor ointments 10%.
Apply two times daily as resorbant ointments

R/ Atropine sulphate 1% 3-5 cc / cattle and horse S/C or I/ M or I/V. (to decrease salivary seretions).

R/ Dexatrin (Oxytetracyclene, dexamethazone & tripellinamine) 1 ml / 25 Kg Bwt. I/M / 3-5 days.

4. Important Notes

1. **Preparation of iodine ointment:** 20 g potassium iodide dissolve in small quantity of alchol then add 10 g iodine crystal, mix well until dissolve all iodine particles, then mix with 100 g Vaseline.

2. Abscess or calculi in the duct can be treated surgically.

3. Chronic cases, local application of tincture iodine or to inject the infected gland with lugol iodine 5 cc/ every week.

4. Potassium iodide may be used 8 g / orally for few days.

Pharyngitis

1. Definition and Causes

It is inflammation of pharyngeal mucosa. It is caused by mechanical (sharp object, hard food or foreign body), thermal (hot food), chemical (corrosive, acid and/or alkaline drugs) or infectious agents (oral necrobacillosis, strangles, anthrax & parasites).

2. Clinical Findings

Painful swallowing, the animal refuse to eat and drink. Regurgitation of fluid and food through the nostril in severe cases. Drooling of saliva. Opening the mouth is painful, head is usually extended. Normal compression of the throat from outside reveal hot painful swelling and causes coughing. Mucopurulent nasal discharge may be present. If local swelling is severe, there may be obstruction to respiration. The retropharyngeal and parotid lymph nodes are commonly enlarged.

3. Treatment

R/ Streptopenicid (pencillin & streptomycin) the large animals 2 vials I/M every 12 hours./ 3-5 days.

R/ Expectyl 30 ml orally in cattle and horse / 12 hours / 3-5 days, as expectorants syrups (human preparation).

R/ Bisilvon 1 ampoule / 70 kg Bwt; I/M, as mucolytic drugs.

4. Important Notes

1. The pharynx is consider as a food and air passage, pharyngitis may be see as symptoms of either disturbances in respiration or intake of food.
2. Remove the primary cause
3. Easily digested food as berseem, bran mash or rice and soup
4. Sometimes parental feeding is necessary
5. Medicated steam inhalation in horse (pail contain boiling water sprinkled with tibn 2 gallons containing an ounce of compher or turpentine).
6. Other cough suppressant such as Codilar and/or Codiphan.
7. Other expectorant drugs such as Bronchistal and/or Isilin.

Chock

1. Definition and Causes

This condition means sudden closure of the normal esophagus. It may be acute or chronic. It is caused by swallowing of bones or lodgment of large pieces of ligaments in dogs. Feeding on dry materials such as bran causes column occluding the esophageal canal in horse. Feeding on large pieces of roots, cobs of maize, upper part of can sugar, root of turnips, potatoes and stump and root of cabbage may causes obstruction of the esophagus in cattle. Esophagitis, Tuberculosis and/or neoplasm in mediastinum lymph node causes stenosis or complete obstruction of the esophagus from outside.

2. Clinical Findings

Continued efforts to swallow and to eructate. The animal moves its head from side to side with restlessness. Open its mouth, protrudes its tongue, profuse salivation and cough spasmodically. Refuse food and drink, if the animal tries to eat it will result in immediate regurgitation. Tympany in cases of complete obstruction in ruminants.

Incomplete obstruction in dogs, causes mild symptoms, complete obstruction resulted in profuse salivation and dribbling of frothy mucous or blood.

3. Treatment

R/ Comblene 0.5-1 cc/ 100 kg Bwt. I/M. or As sedative and minor tranquilizer.

R/ Atropine sulphate 1% 3-5 cc / cattle & horse S/C or I/ M or I/V, to decrease salivary secretion.

R/ Novalgin 1ml / 8 kg Bwt. I/ M or I/V, as analgesic drugs.

4. Important Notes

1. Foreign bodies in the anterior part of the esophagus removed by the hand
2. Foreign bodies in cervical portion of the esophagus, strong pressure by thumb from outside to push the foreign body towards the pharynx.
3. Foreign bodies in thoracic portion of the esophagus, removed by using of stomach tube to push it toward the stomach.
4. In cases of dry column of bran in esophagus of horse, introduce the stomach tube through the nostrils till it reachs the column then pump water to penetrate the bran then lower its head and neck downwards. You may repeat this process several times.
5. Sometimes esophagotomy is required
6. Minor tranquilizer such as Neurazin 1 ampoule /70 kg Bwt; I/M

Vomiting

1. Definition and Causes

It is forcible expulsion of the stomach contents through the nose or the mouth. It is caused by irritation of the stomach mucosa or vomiting center in the medulla oblongata, diseases of brain and drugs causing central vomiting action (apomorphine). Plant poisoning or other poisoning or autointoxication. Gastritis or overeating, obstruction of the pylorus (Gastrophilus larvae) and small intestine. Involvement of organs such as the kidneys, liver and pancreas.

2. Clinical Findings

The animals put the posterior legs under the body, strechs head and neck and expel large quantities of stomach content. A yellow or green liquid usually indicates the presence of bile from the proximal duodenum. While foamy or frothy material is usually associated with excessive mucous during gastritis.

3. Treatment

R/ Sodium bicarbonate 5-10 g / orally / 12 hours / dog, as antacid.

R/ Atropine sulphate 1% 3-5 cc / cattle & horse S/C, I/ M or I/V, antispasmodic drug

R/ Primpran, 1 ampoule / 70 kg Bwt; as antiemetic drugs

4. Important Notes

1.Egg albumin, oils, sugar, honey, treacl and/or starch, as demulcents.
2. Other antacid drugs as Mucogel, Epicogel susp and/or Alkasilon.
3.True emesis is not possible in the horses and ruminant, but sometimes occurs in all these species particularly in young ruminants in adults the animal seldom lives long after this event.
4. In horse vomiting occurs via the nose.
5. Other spasmolytic drugs from human preparation. as Buscopan, Novatropine, Spasmocin, Spasmopyralgin-M or Atropine 0.1%. (1 ampoule / 70 kg Bwt. I/M).

Gastritis

1. Definition and Causes

It is inflammation of the stomach. It is commonly associated with enteritis. It is caused by overfeeding, bad teeth and foreign bodies and also feeding on frozen food, damaged food or coarse fibrous foods as straw bedding. It is also caused by poisons such as caustic and irritant materials, excessive production of lactic acid in the rumen. Bacterial infections e.g. necrobacillus, leptospira in dog etc. Viral infections as rinder pest, equine influenza, hog cholera, infecious canine hepatitis. Fungus agents can produce diffuse or ulcerative gastritis in newborn animals. Parasitic infestation such as nematodes e.g. trichostrongylus, ostertagia spp, hemonchus, paramphystomes, habronema and ascaris migration.

2. Clinical Findings

A). Acute Gastritis:

Repeated vomiting with forceful movements, the vomitus contains much mucous, blood or foreign material. Abdominal pain. Diarrhea may develop. Excessive vomiting lead to dehydration, alkalosis, tetany and rapid breathing. Fever in severe cases.

B). Chronic Gastritis:

Decrease appetite. Vomiting occurs not frequently, but usually after feeding, the vomitus contains much viscid mucus. The animal is emaciated due to lack of food intake and incomplete indigestion. Anemia in bovine in cases suffered from bloodsucker stomach worm, sometimes bottle jaw will develop.

3. Treatment

R/ Bismuth subnitrate for large animals 20 - 40 g / dog / orally. or white egg, as coating drugs.

R/ Sodium bicarbonate 5-10 g / dog orally as antiacid.

R/ Saline or Ringer lactate solution 0.5 - 1 liter / I/V as fluid therapy.

R/ Atropine sulphate 0. 1% 1 ampoule/70 kg Bwt. I/ M or I/V, as antispasmodic drug.

R/ Cortigen B $_6$ 1 ampoule / 20 kg Bwt; as antiemetic drugs

R/ Amoxicillin 15 % 1 ml / 100 Kg Bwt, I/V or I/M / 3-5 days.

4. Important Notes

1. Gastric lavage and enema to remove irritant chemical or poisoning.
2. To alleviate the gastric inflammation, withhold food and water for a period of at least 12-24 hours and replaced by parentral administration then soft palatable, highly nutritious food is necessary e.g. bran mashes to cattle and horses. Chicken with rice and soup to dogs.
3. In cases of hematomesis (Bloody vomiting), inject vitamin k & calcium preparation, sometimes blood transfusion is necessary.

Simple Indigestion

1. Definition and Causes

It is a disorder and inactivity in the rumen and reticulum due to the presence of undigested food in the rumen, which undergoes fermentation. It is caused by the atony of the fore-stomach; dietary abnormalities such as indigestible roughage, low protein intake; mouldy food; moderate concentrate intake and insufficient drinking water. Secondary indigestion occurs in cases of toxemia and septicemia.

2. Clinical Findings

The common symptoms are a sudden reduction in appetite; dullness; sharp decrease in milk yield; decrease in rumen contraction (sometimes rumenstasis); constipation (firm feces). Diarrhea may be present if the cause is damaged food.

3. Treatment

R/ Magnesium sulfate 300-400g/ cow orally as a purgatives.

R/ Supermach 2 sachet / cow orally, daily for 2 days, as a stomachic. (increase the number and activity of microflora and microfauna).

R/ Dry yeast about 50 g dissolved in a sufficient quantity of warm water and given orally.

4. Important Notes

1. Allow massage of the rumen from the left flank.
2. Rectal enema, back racking and exercise are necessary.
3. It is contra-indicated to give carbachol or magnesium sulfate in pregnancy, severe constipation and acute impaction.
4. It is better to transfer 1-2 liters of rumen juices from healthy animal.
6. Other stomachics such as laxavit, bykodigest, vapcodiges, muvdigest, rumstomaton or tonovit can be used.

Acute Impaction

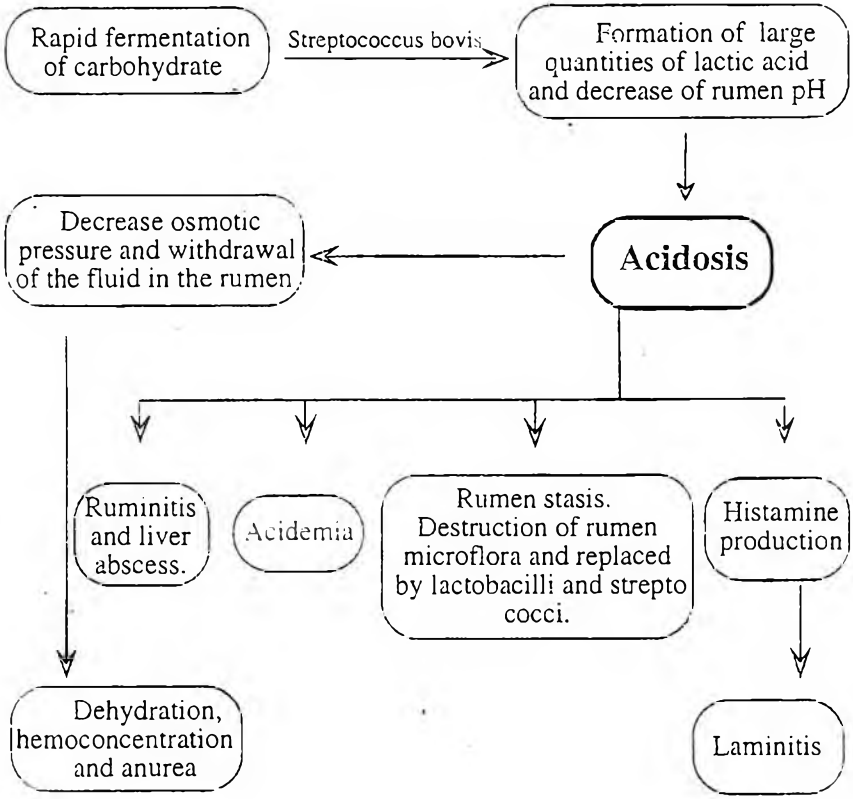
1. Definition and Causes

Ingestion of large amount of highly fermentable carbohydrate foods causes an acute illness due to excess production of lactic acid in the rumen. A crushed grain seems to cause more problems than whole grain.

2. Clinical Findings

The common symptoms are depression, anorexia, grinding of teeth, abdominal pain, kicking at the belly, rumenstasis and constipation, dry muzzle and nose, sunken eyes (as a result of dehydration). Other symptoms are increased respiration (40-60/M) and pulse (120/M). Temperature is usually below normal. The animal suffers from staggering in gait, laminitis, recumbancy, decrease response to stimuli and death may occur in 1-3 days.

3. Pathogenesis



Acute Impaction

Line of Treatment

1. Evacuation of stomach content.
2. Antacid.
3. Fluid replacement.
4. Oral antibiotic.
5. Antihistaminic.
6. Stomachic.

3. Treatment

R/ Liquid Paraffin 1 liter /100 kg Bwt. / Cattle / orally.
R/ Sodium Bicarbonate 1g / kg Bwt, orally in Cattle.
R/ Sodium Bicarbonate 2 - 3%, 0.5 - 1 liters, I/V very slowly, In cases of acidemia.
R/ Lactate Ringer 3 - 4 liters I/V.or S/C, in Cattle.
or Saline 0.9% or Dextrose 5%.
R/ Teramycin powder 5 - 8 g, orally in Cattle
or Penicillin 5 - 7 million IU
R/ Anti-Stamin 1 cc / 10 kg BW I/M, I/V & S/C in Cattle, as antihistaminic drug .
R/ Supermach 2 sachet / Cow orally daily in the second and third days, as a stomachic increase the number and activity of microflora and microfauna).

4. Important Notes

1. Other antacid such as magnesium salt (carbonate, oxide or trisilicate) or calcium carbonat can be used.
2. Other stomachics (e.g. Superflora, Bykodigest or Muvdigest) .
3. Apply rectal enema by warm water and soup or liquid paraffin; back racking and ruminal massage.
4. The animal must exercise 2-3 times daily. The amount of water must be decreased.
5. In severe impaction, surgery is recommended.
6. Prognosis is bad in cases of subnormal temperature with recumbancy.
7. Rumen transplantation is recommended after correction of the pH.
8. In cases of liver absces, systemic antibiotics (e.g. Uvomycin 1 ml / 10 kg) can be used.

9. **Rumen alkalosis:** It is an acute indigestion resulting from feeding on a large amount of urea, nitrogenous substances or line seed cake. The symptoms are the same as those of acidosis; in addition to tremors, muscular weakness and nervous signs. The treatment is almost the same, except replacing the sedatives and the antacids with antalkaline drugs such as vinegar 1 - 2 liters mixed with cold water / orally.

Traumatic reticulitis

1. Definition and causes

It is a disease of cattle resulting from perforation of the wall of reticulum by sharp penetrating objects (wire or nails ..).

2. Clinical Findings

The common symptoms are complete anorexia; a sudden fall in milk yield; rumenstasis; recurrent tympany; an increase of pulse, respiration and temperature; subacute abdominal pain and arching of the back. Pain can be detected by vigorous palpation of the abdominal wall just behind the xiphoid cartilage.

3. Diagnosis

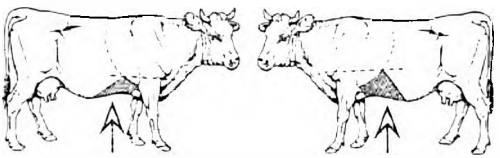
1. Clinical signs
2. Pain tests
   * Walking on downhill.
   * Turning in a narrow circle.
   * Pinching of the wither.
   * Side stick method.
   * Strong percussion on the xiphoid region.
3. Min detector to detect any foreign body of magnetic nature
   * It is not useful if the foreign body is copper or non-magnetic.
   * It gives positive results to non-penetrating magnetic objects.
4. Laboratory examination
   * Total Leucocytic counts rises up to 8000 - 12000 / cumm
   * Neutrophilia.

4. Treatment

Rumenotomy is recommended to remove any foreign body and decomposed food material

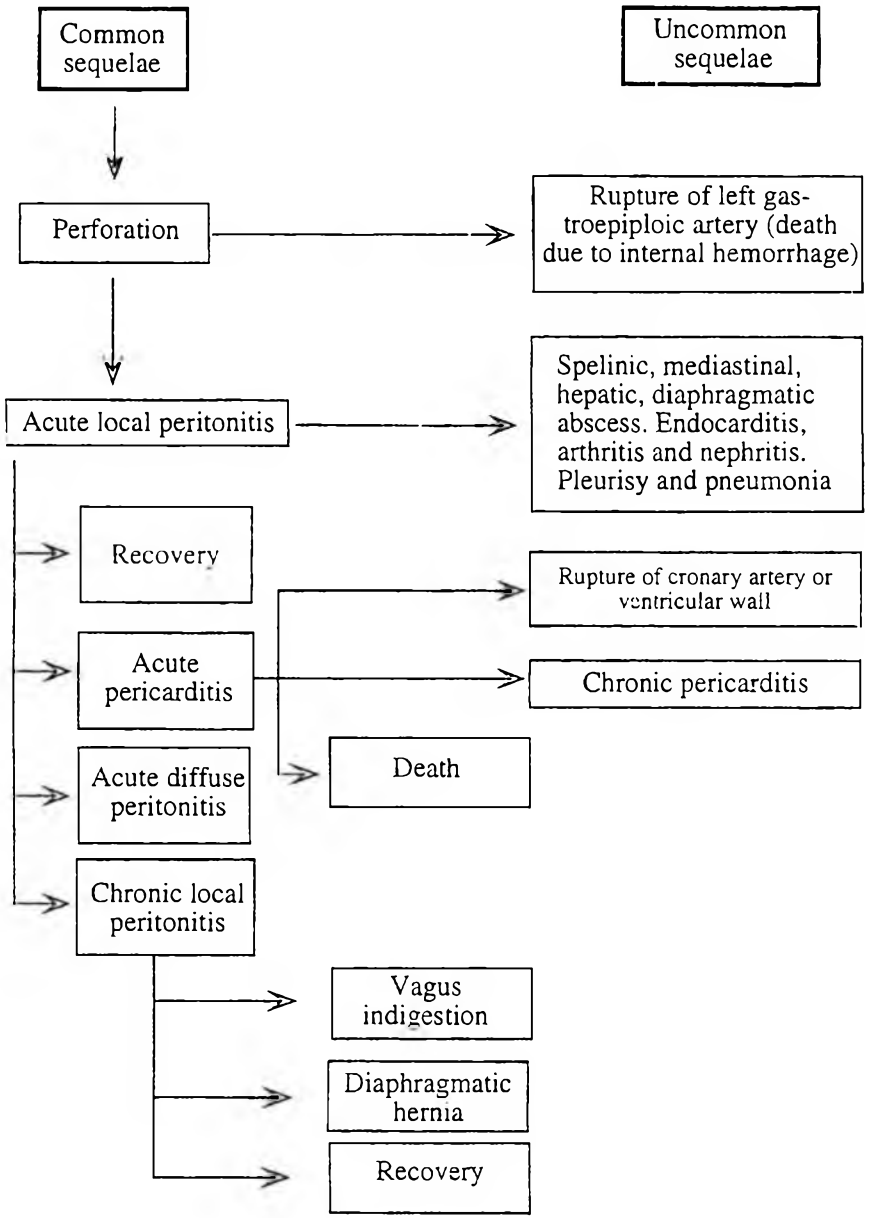
5. Important Notes

1. The prehension of food in cattle by tongue predisposes it to ingest foreign body.
2. Pain tests are positive when accompanied by a grunt of pain.
3. The oral administration of a magnet to immobilize the foreign body inside the rumen is recommended.
4. The administration of antibiotic drugs is necessary to control the infection.
5. It is necessary to elevate the forefeet of the animal by standing on a sloop to avoid the occurrence of traumatic pericarditis.



Site of reticulum (shaded) between shoulder joint, umbilicus and caudal edge of the lung.

Sequelae of traumatic reticulitis



Tympany

1. Definition and Causes

It is an over distention of the rumen and reticulum with gases of fermentation either separated from ingesta (simple tympany) or mixed with ingesta (frothy tympany). It is caused by grazing on young rapidly growing legumes and young grass cereal crops (cabbages, barseem, beans...); the sudden change from dry to green ration; feeding on mouldy fermented food or the ingestion of large amount of milk in calf. It may be secondary to impaction or stenosis of the esophagus. Sometimes, recurrent tympany occurs due to traumatic reticulitis, tumors inside or outside the esophagus, the enlargement of mediastinal lymph nodes in cases of tuberculosis. Persistent ruminal tympany occurs in cases of diaphragmatic hernia and vagal indigestion.

2. Clinical Findings

The common symptoms are distention of the left side of the abdomen, discomfort, kicking at the belly, dyspnea, protrusion of the tongue, extension of the head, increased pulse and respiratory rates, decrease in the ruminal movement and milk production. In the severe cases, cyanosis of mucous membrane, bulging of the eyes and death may occur due to respiratory failure.

3. Treatment

a) Emergency treatment

Reduce the intra-ruminal pressure as soon as possible by passing a stomach tube or trocarization; back racking; massage on the tongue and rectal enema. In severe cases, rumenotomy is required.

b) Medical treatment

R/ Liquid Paraffin 0.5 - 1 liter , as a defoming agent.

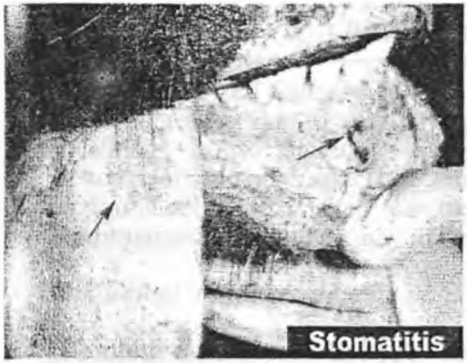
R/ Muv-antibloat one bottle for Cattle orally or intra-ruminal. Dimethicone or Bloatzal, orally as antifrothy preparation.

R/ Supermach 2 sachet / Cow orally daily in the second and third days, as a stomachic to increase the number and activity of microflora and microfauna).

4. Important Notes

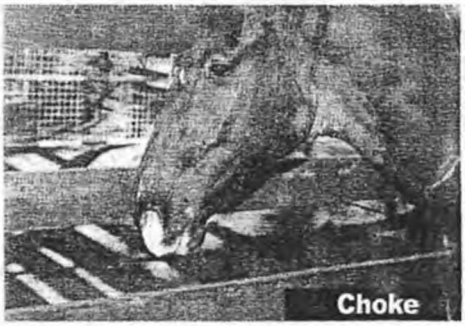
1. Administration of vegetable oil 60 cc orally or mixed with water as a prophylactic.
2. Other oils such as line seed oil, corn oil, mineral or vegetable oils 1 liter / 100 kg Bwt can be used orally.
3. Leave trocar and canula in the rumen for a period ranging between 12-24 hours to get rid of the gases (no value in frothy tympany).
4. Gradual change of ration from dry to green is necessary.
5. Other antibloat drugs as bloatzal, trimethcone, sicadine can be used.

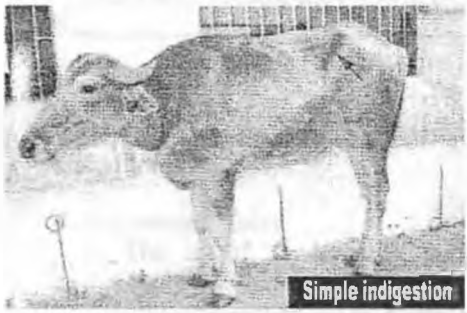
Plate 1 Diseases of digestive system


Stomatitis


Pharyngitis

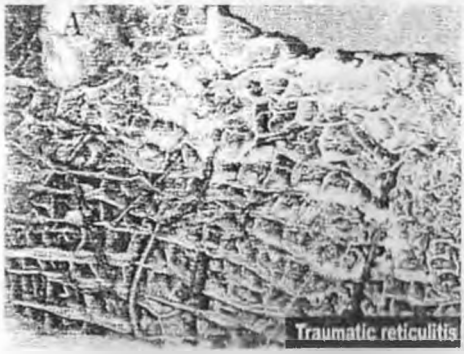

Parotitis


Choke


Simple indigestion


Tympany


Traumatic reticulitis


Traumatic reticulitis

Vagal Indigestion

1. Definition and Causes

In digestion due to varying degree of paralysis of the forestomach resulting from injured vagus nerve. Caused by traumatic reticuloperitonitis (affect ventral branch of vagus nerve), actino-bacillosis, parasites (sarcospiridia and cysticercous taenicollis) and enlarged lymph node may injury to the nerve.

2. Clinical Findings

Ruminal distension with hyper-motility

Moderate to severe ruminal tympany, emaciation, abdominal distesion and rumen moving vigorously and continuosly but sounds reduced in volume.

Ruminal distension with hypo-motility

This type occur commonly in late pregnancy and after calving. The cow is clinically normal in all, except: anorexia, passes only small amounts of soft pasty faeces, distended abdomin, no response to treatment with purgatives or parasympathetic stimulants, atony of the rumen, mild bloat, rectal palpation reveals distension rumen and abomasum blocking of the pelvic inlel. Loss of weight rapidly, weakness, recumbancy and death.

3. Treatment

Animals suspected to be suffering from such affection must be slaughtered.

4. *Important Notes*

1. The major abnormality appears to be in the development of achalasia (dysfunction) of the reticulo-omasal and pyloric sphincters, resulting of accumulation of food material in the rumen.

2. **Diaphragmatic Hernia:** This means protrusion of a part from the rumen and reticulum through a rupture in the diaphragmatic musculature. Caused by weakened diaphragm by lesions of traumatic reticulo-peritonitis or congenital defect. The same syndrome as vagus indigestion accompanied with hypermotility. Irregular appetite, loss of condition, moderate rumen tympany, grinding of teeth, small amounts and pasty faeces and the animal may vomit. Bradycardia and systolic murmur. Diagnosis of the problem mainly by rumenotomy. Animals suspected to be suffering from such affection must be slaughtered.

Abomasal Displacement

1. Definition and Causes

It is a common disease of mature cows in which the abomasum displaced from its normal position in the abdominal floor either to the right (between the liver and right abdominal wall) or to the left (between the rumen and left abdominal wall) or into an anterior position (between the reticulum and diaphragm). The predisposing factors are feeding on grain in late pregnancy, vigorous movement during transportation and during parturition.

2. Clinical Findings

Sudden anorexia, decrease in milk production and loss of body weight. Severe abdominal pain, rumenstasis and tympany. Small volume of feces and pasty in consistency. Auscultation of an area below a line from the center of the left flank to behind the left elbow reveals the presence of splashing or tinkling sound (more fluid in nature than the rumen) every 15 minutes. An obvious bulge caused by distended abomasum may develop in the anterior part of flank region. The swelling is tympanitic and gives a resonant sound on percussion.

3. Diagnosis

The disease must be suspected in every case of ketosis where there is no response to treatment (Keton smell in the mouth and breath). Rectal palpation reveals the distended abomasum to the left of the rumen. In anterior displacement abomasum sounds can be heard just above the heart area on both sides of the chest. Exploratory labaratomy is necessary in many cases to confirm a diagnosis of displacement.

4. Treatment

Surgical interference is the best method of the treatment.

5. Important Notes

1. Displacement to the right has no relationship to pregnancy or parturition.

2. The disease is not fatal but affected animals become useless for milk production.

3. Rolling of the animal may correct the displacement.

Abomasal ulcers of cattle

1. Definition and Causes

It occurs in mature cattle and calves and may cause acute Abomasal hemorrhage, indigestion & melena. It is caused by Abomasal hairballs, displacement, impaction, torsion & lyphosarcoma. Coccidiosis, internal parasites, over fertilized plant, excess roughage in young calves, and vagus indigestion.

2. Pathogenesis

Non perforating ulcers ⟶ abomasum thickening and chronic gastritis.
Ulcers causing severe blood loss ⟶ penetration of wall of abomasum vessels ⟶ hemorrhage and anemia. Perforating ulcers ⟶ leakage of abomasum content ⟶ local or diffuse peritonitis.

3. Clinical Findings

Abdominal pain, sudden onset of anorexia, decrease in milk prduction and tachycardia (90 - 100). Melena (the feces are scanty, black, and tarry), anemia, in severe hemorrhage death may occur & in less severe cases may recovere through 4 - 6 days.

4. Treatment

R/ Sodium Bicarbonate 1g / kg Bwt. orally in cattle.
The dose may repeat 4 times a day. It may be injected directly into the abomasum.
R/ Calcium carbonate 80 - 120 g / orally / cattle.
R/ Iron Dextran 4 ml / 10 kg I/M , as hematinics.
R/ Cobalt and B vitamins, as tonic.
R/ Oxycomplex (Oxytetracyclene) 3 cc/ 100Kg Bwt, I/M / 3-5 days.

5. Important Notes

1. Blood transfusion (1 liter /50 kg Bwt.), it is indicated in weakness, takycardia, dyspnea and low hematocite (12%).
2. Surgical interference with limited success in cattle and better in calves.
3. Other antacid such as magnesium salt (carbonate, trisilicate or oxide), calcium carbonate or almonium hydroxide (gell or phosphate).
4. Other astringent and protectants such as Bismuth subnitrate or carbonate, magnesium trisilicate or starch.

Enteritis

1. Definition and Causes

Inflammation of the intestinal mucosa characterized by increase motility of the gut, decrease absorption and increase secretion. It is caused by bacterial enteritis (Colibacillosis, Salmonellosis and Enterotoxaemia due to Clostridium Perfiring). Viral enteritis (Rinder pest, Mucosal disease, Rota and Crona virus). Chemical agents as poisoning by Arsenic, Phosphorus, Cupper, Mercury, Sodium Chlorid, lead and nitrates. Nutritional deficiency (nicotinic acid and other B vitamins). Parasitic enteritis (Paramphistomum, Trichostrongylus spp; Ostertagia spp; Cooperia spp; Nematodirus; Ascaris, Coccidiosis and Tape worm infestation).

2. Clinical Findings

A. **Acute Enteritis:** Abdominal pain, straining and colic may develop. Feces are soft and fluidly with unpleasant odor and may contain blood or shreds of mucous. Auscultation reveals sounds of increased motility. Pale mucous membrane in parasitic infestation while congested in infectious diseases and icteric in hepatic dysfunction. Increase in body temperature in case of infectious diseases. Dehydration and vomiting may develop.

B. **Chronic Enteritis:** Pain is seldom, odor is not changed and much mucous and emaciation are present.

3. Treatment

R/ Tannic acid 5-10 g / cattle / orally, as astringent.
R/ Calcium carbonate or starch 80-120 g orally / cattle.
R/ Diaclean 1 sachet / 50 kg, as antimicrobial drugs.
R/ Atropine sulphate 1% 3-5 cc / cattle & horse S/C or I/ M or I/V.
R/ Saline and dextrose 5%, 1-2 liters, as fluid therapy and electrolytes.
R/ Super-Lyte 1 sachet / oraly / dissolve in 2 litre of water.
R/ Finadyne Cattle & horse 1 / 45 kg Bwt. I/M & I/V.

4. Important Notes

1. In cases that are suffered from toxicity:
   a. Washing the stomach with mineral oil, saline and purgative using stomach tube to help evacuation and removal of toxic material. Washing of the stomach is contra-indicated in case of poisoning with corrosive.
   b. General antidote (Atropine Sulphate) or specific antidote.
   c. Oral administration of egg albumin to protect the mucosa.
   d. I/V injection of calcium and cardiac tonic as adcopherin I/M.
2. Anthelmintic for parasitic enteritis.
3. Other antimicrobial such as New Diaclean 1/2 sachet for calf & 2 sachet for adult or Trimetasol 1 cc/ 32 kg Bwt.

Diarrhea

1. Definition and Causes

It is a disorder in the intestine characterized by frequent evacuation of the bowel, feces are watery and it may be tinged with blood. It is causes by dietetic errors such as ingestion of mouldy, fermented, spoiled food. Chemical irritants such as arsenical preparations or mercury. Poisonous plant and/or sudden change of the diet. Secondary to bacterial, viral or parasitic. Also copper and cobalt deficiency.

2. Clinical Findings

Frequent evacuation of watery feces and may be stained with blood. Straining, colicy pain and expulsion of gases. Dehydration (sunken eye, rough coat and non elastic skin). Rise of body temperature in cases of bacterial or viral diseases.

3. Treatment

R/ Sulphaguanidine 20 g / 100 kg Bwt orally as antiseptic drugs.

R/ Calcium carbonate or starch 80-120 g orally / cattle.

R/ Tannic acid or catchue 5-10 g / cattle orally as astringent.

R/ Saline 1-2 liter I/V according to the degree of dehydration.

4. Important Notes

1. Patent preparations of antibiotic and antiseptic such as Biodiristin, New Diaclean (1/2 sachet for calf & 2 sachet for adult) or Trime (1cc/ 32 kg Bwt.). Kapect or Diastop or Lomotil one bottle / head.
2. You may prepar a mixture from chloramphenichol, sulfaguanidine, neomycin, tannic acid and starch.
3. Adminstration of Tyvert 1 ml/ 5 kg Bwt. per os. In cases of nematodiasis.
4. Adminstration of Mansonil or Yomesan.1 tablet / 20 kg Bwt. In cases of paramphistomiasis.
5. Adminstration of Dovenix 1cc / 25 kg, Bwt. S/C, In cases of fascioliasis.
6. Drug specific in equine as equivalan (oral past in graduated syring), Pancure, Banminth, piperazin citrate (200-300 mg/kg, Bwt specific to ascaris in all animals).
7. Drug acting orally in dogs and cats as Antiver, Fluvermal or Vansil 1 table spoonful 2 times daily.
8. In cases of presence of toxins, it is preferable to give laxative or mild purgative as Paraffin oil 1 liter / large animal and 100-200 cc / small animal.
9. Adminstration of Sulphadimidine and amprolium in cases of coccidiosis.

Dietetic scours

1. Definition and Causes

Passage of soft, fluid feces in young calves associated with rapid loss of weight with normal appetite. It is caused by dietary abnormalities such as drinking too rapidly, feeding of excessive quantities of milk at too long intervals and temperature below body heat. Feeding of milk high in fat or sudden changes from whole milk to milk substitutes.

2. Pathogenesis

Failure of esophageal reflex in pail fed calves, the milk deposited in the rumen where it undergoes putrefaction. Poor clotting of milk resulted from milk with a very low level of casein or calcium or with high level of sodium or pH. Poorly clotting of milk passes in to the intestine where protein putrefaction causes scour

3. Treatment

R/ Tannic acid or catchue, as astringent and coating.

R/ Trimetasol (Sulpha & trimethoprim) 1 cc/ 32 kg Bwt. orally.

R/ Vit-Lyte (oral electrolyte)
Reverse case may require I/V injection of saline & ringer Lactate.

R/ Limewater (1 part to 2 parts of milk) helps digestion.

4. Important Notes

1. Milk feeding should be stopped, then oral electrolyte solution for 24 hours, Milk is then gradually re-introduced.

2. Calves should be fed at least three times a day on a low fat contents milk.

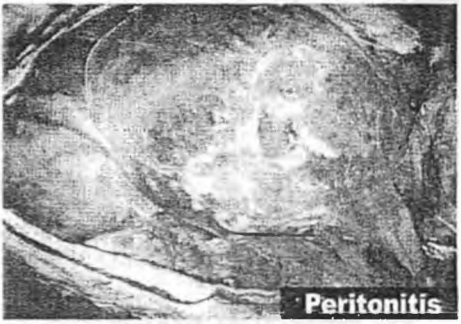
3. Foals should be muzzled and allowed only limited access to the mare.

4. Piperazine Citrate 50% 4 g / 10 kg Bwt. orally for treatment of ascaridia in calves and foals


Melena


Abomasal ulcer


Left abomasal displacement


Peritonitis


Calf scour


Diarrhea


Naso-gastric tube


Fascioliasis


Diarrhea


Ascites

Constipation

1. Definition and Causes

It is difficult evacuation of the feces because the feces are retained for a long time in the intestine. It is caused by atony of the intestine, irregularity in feeding, small amount of water given to the animal, constant feeding on starchy food, sudden change in diet, lack of exercise, feeding on bones. Diseases of the liver. It may occur in cases of impaction, tympany, prostatitis and fever.

2. Clinical Findings

Loss of appetite. The animal is dull and depressed and there is abdominal pain. The faces are hard in consistency and may be stained with blood. Defecation is accompanied by straining.

3. Treatment

R/ Magnesium Sulfate, 60-100 gm/ cattle / orally, as mild purgative.

R/ Lin seed oil 1/2 liter for large animals, as Laxative, purgative and lubricant.

4. Important Notes

1. Other Laxative, purgative and lubrican such as Laxofin, Laxolac, Abilaxine or Laxomag

2. Give easily digested food, regulate the feeding time, apply sufficient quantity of water to the animal and reduce the amount of carbohydrate to the animals.

3. Enema with soft soap and warm water, also it is better to add some oily material. The amount of fluid required for such enema is about 10 liters for a large animal and from 1/2 - 1 liter for small animal. The enema must be repeated at intervals to stimulate and regulate the peristaltic movement of the intestine.

4. In severe cases paraffin oil is recommended but magnesium Sulfate is contra - indicated in pregnant animals and severe constipation.

Spasmodic colic

1. Definition and Causes

It is severe attacks of abdominal pain caused by functional disturbance of the intestine. It is caused by drinking cold water when hot weather and more sweating after work. The body exposure to cold or wetness.

2. Clinical Findings

It characterized by intermittent fits of colic, rapid course (short duration) and favorable termination. Occur suddenly, each fits lasts from 5-15 minutes (short Attacks) and during these attacks the animal kicks, lies down on the ground and rolls with violence but often rise again after rolling. These efforts resulted in slight increase in body temperature, accelerated respiration, rapid pulse and the animal sweat in patches. Hypermotility (increases in the peristaltic sound). Defecation takes place at short intervals and the feces may be semi-solid or fluid in character. The mucous membrane may be congested.

Diffrential diagnosis: Diseases causes chest pain, pain on urination and abdominal pain in horse see key of diffrential diagnosis.

3. Treatment

R/ Novalgin 20-25 ml, I/V & I/M, as sedative.

R/ Atropine sulphate 1% 3-5 cc / horse S/C or I/ M or I/V, as spasmolytics drugs.

4. Important Notes

1. It is pereferable to give analgesic and antispasmodic I/V in saline.
2. Rectal enema using warm water and soft soap.
3. Warm compresses applied to the abdomen, act as counter irritant.
4. Side effect of Atropine Sulfate: dryness of the mouth and skin, decrease heart rate followed by increasing with tachycardia, decreasing the motility of intestine, so that, contra-indicated in flatulent and obstructive colic.
5. Other spasmolytic drugs such as Boscopan, Glucolinamine, Spasmopyralgin -M, Atropine 0.1% (1 amp/70 kg, Bwt. I/M).

Flatulent colic

1. Definition and Causes

This form of colic is due to the excessive distension of the bowel with gases particularly the caecum and colon. It is caused by feeding on large quantities of succulent food such as barseem. Ingestion of spoiled or mouldy food or grains which has tendency to swell. Sudden change in the ration. Atony of the bowel. Obstruction of the bowel by sands.

2. Clinical Findings

Sudden attacks of abdominal pain which is continuous. Affected horse may roll and bow violently and lies down very carefully. This effort resulted in quick pulse and accelerated respiration. Abdominal distension occurs due to accumulation of gases in the intestine, which can be observed in the flanks region (especially in the right flank). Percussion gives tympanic sound (drum like sound). Decrease of peristaltic movement. The mucous membrane is congested.

3. Treatment

R/ Analagin 20-25 cc I/V, as sedative.

R/ Liquid paraffin 2-4 liters / orally / horse every 12 hours.

R/ A mixture of Ammonium carbonate 50 grams & charchol 50 g (Neocarbotrina tablet) as carminatives drugs.

R/ Ringer Lactate 2 - 4 liters I/V.

4. Important Notes

1. Apply massage externally in flank regions and internally through the rectum using the hand to stimulate peristalsis.

2. In severe cases, you must get rid of this intestinal tympany from the caecum by trocarisation through the right and left flanks. However peritonitis may occur due to infection.

3. Rectal enema using warm water and soft soap.

4. You may use carminatives drugs of human preparation such as Disflatyl (tablets), Flatidyl (tablets), Maxiflat (tablets), or Biskaol (powder).

5. Oral fluid to soften intestinal masses, doses are empirical.

Intestinal impaction

1. Definition and Causes

Colic due to impaction of the intestine. It is caused by over feeding on coarse food rich in cellulose and bran. Ingesting food, which contain large amounts of mud or sand. Defective teeth and/or obstruction of the intestine by large foreign bodies or parasite.

2. Clinical Findings

Acute colic: Restlessness and beats the ground with the forelimbs, the animal lies on the ground and rolls, quick pulse & continuous pain during attack is present. During urination the animal throw the hind legs more backward and outward and urine comes out at intervals.

Subacute colic which occurs slowly, started with dullness and abdominal discomfort, the animals looks at the flank and kicking its belly. Anorexia. There is constipation and the feces are passed in small amounts and hard in consistency and covered with thick, sticky mucous. Intestinal sound are absent or much decreased in intensity. Moderate decrease in pulse. Rectal palpation revealed that balloon shape impacted colon.

Differential diagnosis between impaction in the small intestine and colon by rectal palpation.

3. Treatment

R/ Novlagin 20-25 cc I/V
R/ Liquid paraffin 2-4 liters / orally / horse every 12 hours.
R/ Ringer Lactate 2 - 4 liters I/V.
R/ Finadyne 1 ml / 45 kg Bwt, I/V & I/M,as anti-inflammatory drugs
R/ Supermach 1-2 sachet / horse orally, as digestant and apetizer.

4. Important Notes

1. Contra-indicated to give Atropine Sulfate and/or Magnesium Sulfate in obstructive colic.
2. In cases of sandy colic you must notice the presence of sand in feces.
3. In cases of obstruction due to parasites, fecal examination reveals the presence of eggs of parasites, anthelmintic drugs is recommended.
4. The symptoms varies according to the location of the impaction, when the duodenum is affected, the symptoms occurs after feeding with few hours, when the ileum is affected symptoms appear after several hours
5. Rectal enema using warm water and soft soap.
6. Reduce the amount of carbohydrate given to the animal.
7. Surgery may be necessary if the condition persists and repeated
8. Oral sedative as: chloral hydrate 30, oil of turpentine 30, spritus ether nitrosi 30, compher 15, Tr. nix vomica 10 then lin seed oil ad 1 litre. give at once by the stomach tube for a horse.
9. In cases of overfeeding of carbohydrate you must give orally and I/V antiacid.

Obstructive colic

A. Intestinal torsion (Volvulus)

Obstruction due to the rotation of sigment of the intestine around its mesenteric axis. It is either partial or complete. It is caused by severe attack of colic which may leads to the torsion due to rolling, jumping or sudden fall of the animal. Injections of large dose of carbacoal which leads to the sudden increase in the peristaltic movements. Heavy infestation with parasite (Ascaris) cause irregularity in peristaltic movement of the intestine resulted in torsion.

B. Intestinal strangulation

It is the occlusion of the intestinal lumen by pressure from outside. It occurs when a lope of the intestine passes through a natural or artificial opening in the peritoneum and held there as in case of inguinal hernia in stallion. Also in case of pedunculated tumor which cause strangulation.

C. Invagination (Intussusception)

Acute intestinal obstruction caused by telescoping of a section of the bowel into a portion immediately behind it, especially in ileo-caecal junction. The affected part form a sausage shaped, painful swelling composed of three segments. It caused by violent intestinal peristaltic movement or presence of tumors in the lumen of the bowel.

Diagnosis

Signs of colic with absence of defecation, peristaltic movement is very weak or absent rectal palpation revealed absence of feces and the intestine distended with gases.

3.Treatment:

* Surgically remov s the obstruction.
* Try to give large doses of liquid paraffin and rectal enema.
* Sedative must be given when pain is severe.

4. Important Notes

1. Complete torsion is unfavorable, the animal die within 12- 24 hours.
2. In partial twist the course and prognosis depends upon the severity.
3. Obstruction in the small intestine causes a more acute and severe syndrome than those in the large intestine.
4. Obstructions of the small intestine or colon in horses usually kill within 24 hr. While similar obstructions in cattle are not usually fatal in less than a week.
5. Hydration of the fluid in the lumen of the intestine causes abdominal pain and dehydration.
6. *Embolic colic (Special type of Colic):*
   It is disorder in the intestine due to the presence of larvae of strongylus vulgaris in the anterior mesenteric artery of the horse, causing aneurysms, emboli and thrombi of the mesenteric artery and its branches. Characterized by intermittent attacks of colic occurs suddenly during work. The symptoms as in spasmodic colic, beside that the feces are bloody stained, fecal examination is required to detect the egg of parasite. No curative treatment in such cases.

Jaundice

1. Definition

Jaundice is the most important clinical sign associated with liver diseases, in which bile pigments accumulates in blood (bilirubinaemia) and then partly excreted by the kidney (bilirubinuria) and partly deposited in the tissue such as mucous membrane (conjunctiva, nasal & oral MM.) and unpigmented portion of the skin. The sweet, milk and exudates also contain bile.

2. Causes

a) **Pre-hepatic** (hemolytic)

Bacterial toxins e.g. bacillary hemoglobinuria and leptospirosis

Invasion of erythrocytes by protozoa or viruses e.g. babesiosis, anaplasma and infectious equine anemia. Inorganic or organic poisons e.g. chronic copper poisoning, hypophosphataemia, overeating of onion. arsenic, phosphorous or lead poisoning.

Immunological reactions e.g. allergic reaction (hemoglobinuria).

b) **Hepatic** (toxic, infective and obstructive) causes of diffuse hepatitis.

c) **Post-hepatic** (obstructive).

Extra-hepatic biliary obstruction by calculi or compression by tumor masses. The common causes are obstruction by nematodes and Inflammation of the bile ducts by extension from enteritis or by Infestation with trematodes.

3. Clinical Findings

Jaundice usually began with symptoms with indigestion, latter on the mucous membrane and unpigmented portion of the skin becomes yellow in color, this change in color is best seen in conjunctiva sclera. The color ranges from lemon yellow to orange yellow or greenish yellow. The urine is also stained with bile pigment, the sweet, milk and exudates also contain bile. There is constipation, feces have a fetid odor and pale in color.The animal is dull and depressed. In dogs and cats, acute jaundice produces convulsion and repeated vomiting.

4. Treatment (Treat the primary cause in addition to the following):

R/ Glucose 25% & 40%, I/V injection.

R/ Cal De Mag, I/V injection of calcium.

R/ Varolex B_{12} 1 vial / cattle, I/M / daily /3 - 5 days.

R/ Multivitamin cattle .20 - 30 cc & Sheep and goat 5 - 10 cc I/M

R/ Supermach 2 sachet /cow orally, daily for 2 days, as a stomachic.

5. Important Notes

1.The diet should be high in carbohydrate and calcium and low in protein and fat as much as protein may leads to ammonia intoxication.

2.You may use hepatic preparations such as Sorbit , Sorbitol, Sorbosan, Hepaton, Rowachol, Legalon, Zymagallin or Dioron.

Hepatitis

1. Definition and Causes

Diffuse degenerative and inflammatory diseases, which affect the liver. The clinical signs of hepatic dysfunction appear only when three-quarters of the liver parenchyma are inactive. Causes by toxins such as Inorganic poisons (phosphorous, arsenic, hexachlorothane and gossypol), Bacterial (Salmonella and leptospira), Parasitic hepatitis, (liver fluke infestation and migration of larvae of ascaris) and congestive heart failure.

2. Clinical Findings

Anorexia accompanied by constipation punctuated by attacks of diarrhea. The feces are light in color than normal. Vomiting in some animals. Nervous signs and dummy syndrom. Pain on palpation the abdomin and liver. Jaundice and edema. Photosensitizations in animal fed green fodder and exposed to sunlight. And ascites. Endocrine abnormalities

Nutritional and metabolic abnormalities

3. Diagnosis

1. Clinical sign
2. Biochemical tests estimation of serum total, direct and indirect bilirobin. In addition to AST, ALT, ALP, LDH, SD, cholesterol, uric acid, albumin, globulin and total protein.
3. Biopsy of the liver.
4. Sonography on the liver

Differential Diagnosis: Encephalopathy and Acidosis.

4. Treatment Treat the primary cause in addition to the following:

R/ Spectrama Vet 1 cc / 40 kg, Bwt, S/C or I/M / daily/3 - 5 days.

R/ Glucose 25% & 40%, I/V injection.

R/ Cal De Mag, I/V injection of calcium.

R/ Varolex B_{12} 1 vial / cattle I/M / daily / 3 - 5 days.

R/ Multivitamin cattle 20 - 30 cc & Sheep and goat 5 - 10 cc I/M

R/ Brewer yeast or egg yolk, as digestive aids.

5. Important Notes

1. The diet should be high in carbohydrate and calcium and low in protein and fat as much as protein may leads to ammonia intoxication
2. You may use hepatic preparations such as Sorbit, Sorbitol, Sorbosan, Hepaton, Rowachol, Legalon, Zymagallin or Dioron
3. Injection of Rolenol in case of fascioliasis, 0.5 ml / 10 Kg Bwt, S/C.

Peritonitis

1. Definition and Causes

It is inflammation of the peritoneal sac, which is accompanied by abdominal pain, which usually varies in degree according to extent of the affection. It is caused by traumatic reticulo-peritonitis in cattle. Rupture of the stomach or intestine when acute dilatation or obstruction occurs. Rupture of the vagina or uterus. Secondary due to pleuritis, tuberculosis, actinobacillosis and migration of parasitic larvae to the peritoneal cavity. Septic surgical operation or during intraperitoneal injection or trocarization in case of tympany

2. Clinical Findings

A. Acute diffuses peritonitis: There is severe abdominal pain, which is manifested by tenderness and rigidity of abdominal wall during palpation, the animal shows pain. Lack desire to move, persistent standing & if the animal lies with great care and grunting. Arched back. Grunting commonly occurs at each step and when the animal defecates or urinates. Moderate increase in pulse, respiration (costal type respiration) and temperature. Congested mucous membrane. Finally the animal is recumbent and unable to rise, subnormal temperature, very weak pulse and heart rate 100-110 / minute.

B. Acute local peritonitis: Similar to those of acute diffuse peritonitis but signs are less severe. Pain is localized in small area, temperature and pulse are not evident.

C. Chronic peritonitis: It is chronic syndrome of indigestion and toxemia.

3. Diagnosis

1. Peritonitis should always be suspected in the presence of acute abdominal pain, paralytic ilius or absence of intestinal sounds and vomiting.
2. **Differential Diagnosis:** Pleuritis, Enteritis, Acute pancreatitis (Dogs), Acute nephritis, Intestinal obstruction & Cholecystitis.

4. Treatment

R/ Glucose 25% & 40%, I/V injection.

R/ Cal De Mag, I/V injection of calcium.

R/ Muv-Ampiclox,, 5-10 /100 kg Bwt / 3-5 days/ I/M

R/ Novalgen 30 cc / cattle. Given I/V to relief pain.

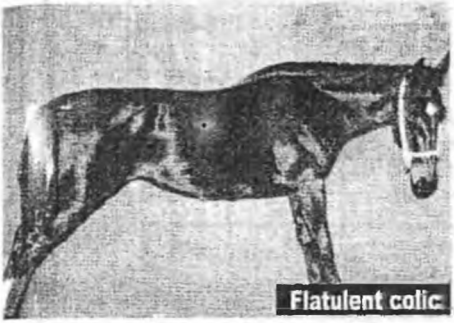
5. Important Notes

1. Do not give food to the animal in the first 2 days
2. Horses and Dogs are usually taking the acute diffuse type of peritonitis, while cattle usually take the chronic type.
3. Peracute cases usually die within 24-48 hours.

Plate 3 Diseases of digestive system


Abdominal pain


Ileo-cecal intussusception


Flatulent colic


Spasmodic colic


Jaundice


Jaundice


Rumen protozoa


Ruminal impaction by a plastic bag in goat

Epistaxis

1. Definition and Causes

It is bleeding from the nostril or from sinuses. It is caused by traumatic injury, foreign bodies, neoplasm, over exhaustion (Race Horse) and bad use of stomach tube. It may be secondary to parasitic diseases (oestrus ovis in sheep and gastrophilus nasals in equine) and/or infectious diseases as anthrax, glanders and hemorrhagic septicemia.

2. Clinical Findings

There is bleeding from the nostrils (unilateral or bilateral). The blood is bright red in color and may be scanty or profuse. It sometimes mixed with mucous. Anemia and loss of condition. Death occurs in untreated cases.

3. Diagnosis

Try to locate the place of injury by use of endoscope

Bright red bleeding ⟶ nasal origin.
Bright red and frothy bleeding ⟶ lung origin.
Brownish, acidic and may mixed with ingesta ⟶ stomach origin.

3. Treatment

R/ Alum 2%, irrigate the affected nostril 3 times daily.

R/ Adrenaline 2%. or tannic acid, plug the affected nostril by a piece of gauze soaked in the above solution.

R/ Cal De Mag 100 - 200 cc I/V - I/M.

R/ Amri - K ampoule 3 ampouls / horse I/M.
or VITAK 30 gm orally daily / one week.

R/ Ringer lactate solution 1-2 liters I/V, as supportive treatment.

4. Important Notes

1. Complete rest and cold application on forehead

2. Tracheotomy can be performed and plug the two nostril with gauze soaked in astringent solution in bilateral bleeding.

3. Do not give Adrenaline injection because it raises blood pressure.

4. Bleeding due to parasite you must give anthelmintic drugs.

5. Other vitamin K ampoules such as Phytomenadion and Konakion.

Rhinitis

1. Definition and Causes

It is inflammation of the mucous membrane of the nose and usually involving the upper respiratory tract. It is caused by inhalation irritant vapor such as ammonia or chloride. The presence of some foreign bodies in the nose as grains or dust. It may be secondary due to microorganism as staphylococcus, streptococcus, and diphtheroids or parasitic as estrous ovis in sheep. Also it may be associated with some specific diseases as strangles glanders and equine influenza in horses.

2. Clinical Findings

Catarrhal rhinitis: Redness and swollen of the mucous membrane of the nostril. Bilateral nasal discharge (watery, mucoid, mucopurulent or purulent). Snoring sound when discharge blocks the nostril.

Cropous rhinitis: Characterized by the presence of greyish patches or yellow fibrous membrane → shed off → bleeding surface → heal → forming trace of scar.

The nasal discharge may contain shreds of mucous membrane and also the submaxillary lymph glands are swollen.

3.Treatment

R/ Alum 1%, tannic acid 0.5, boric acid 2% or potassium permanganate 0.3%. Irrigation of the nasal cavities 2-3 times daily.

R/ Saline solution or Sodium Bicarbonate 1%.
irrigation of the nose with above solution to hasten the shedding of pseudomembrane.

R/ Borgal 24% (Sulphadoxin & Trimethoprim). 3 ml / 50 kg Bwt. I/V & I/M, a second dose after 48 hours may be needed.

4. Important Notes

1.Thick tenacious must be removed gently.

2. Crusts can be removed with warm water and simple ointment.

3. Put the animal in well-ventilated place, complete rest and easily digested food.

4. Medicated steam inhalation in horse. The pail contains boiling water sprinkled with tibn (2 gallons containing an ounce of compher or turpentine is added).

Laryngitis, Tracheitis and Bronchitis

1. Definition and Causes

It is inflammation of larynx, trachea and bronchi. It is caused by sudden exposure to cold, inhalation of irritant gases or extension of infection from other parts of respiratory tract. It may be infectious disease such as IBR in cattle, equine viral influenza and strangles in horse.

2. Clinical Findings

Acute form: Nasal discharge (mucoid or mucopurulant). Dry painful coughing then moist later. Increase in pulse, respiration and temperature. Dyspnea accompanied by loud stridor and harsh breath sound. Congested mucous membrane. Auscultation the chest area hears dry rales in case of thick exudate, moist rales in case of watery exudate and criptant rales in case of severe swelling of mucous membrane.

Chronic form: The same as acute form but the course of the disease takes longer times. Severe cough but not painful, normal temperature and by auscultation we notice only dry rales.

3. Treatment

R/ Streptopenicid (pencillin & streptomycine), 2 vial I/M / 12 hrs.
or GENTA 50 (Gentamycin sulfate), 8 ml / 100 kg Bwt. I/M & I/V.

R/ Expectyl or Bronchistal 30 ml in cattle orally 3-5 day, as expectorant

R/ Bisilvon 1 amp./ 70 kg BW, I/M, as mucolytic drugs.

R/ Buta-fenil 5-10 cc / 450 Kg BW I/M, as anti-inflammatory drug.

R/ Saline, dextrose 5% or ringer lactate As supportive treatment.

R/ Adcoferine 5 cc I/M daily or Lanoxin ampoule, as heart tonic.

R/ Cevarol 1 ampoule/ 70 kg Bwt. I/M daily, as vitamin C.
or VITAC 30gm orally daily / one week.

4. Important Notes

1. Dry rales occurs when air is being forced through a bronchial tube which is partially constricted, either by dry tenacious thick exudate or severe swelling of the mucous membrane.
2. Moist rales occurs when bronchi contain light, thin watery mucous (pus - blood - liquid - exudate) moving from place to another.
3. Cripitant rales occur when the opposing walls of bronchial mucosa become adherent to one another and have to be separated by the stream of incoming air.
4. Tussivan or Codaphen in case of dry cough.

Pneumonia

1. Definition and causes

It is inflammation of lung tissue and bronchioles. It is caused by:

In cattle: Pasteurella multocida, salmonella and TB.
IBR, bovine respiratory syncytial virus, parainfluenza 3.
Dictyocaulus viviparous (verminus pneumonia).

In Horse: Streptococci (stran gles), corynbacterium and E coli.
Adenovirus, equine herbs virus 1 (EVR).
Dictyocaulus arnfeldi, para-ascaris equorum.

In sheep Pasteurella multocida, corynbacterium pseudo-tuberculosis.
Ovine respiratory syncytial vVirus, parainfluenza 3.
Dictyocaulus filaria

2. Clinical Findings

Off food, dullness, decrease in milk production and rumenstasis. Painful cough, congested mucous membrane and nasal discharge. Increase in pulse rate but weak heart beat, labored respiration and abducted elbow. Continous or recurrent fever. In verminus pneumonia, protrusion of the tongue, expectoration of masses of mucous sometimes mixed with worm.

Stages of pneumonia	Auscultation	Percussion
Congestion	Exaggerated vesicular sound	incomplete dull sound
Red hepatization	Absence of sound (consolidation) only heart and bronchial sound	complete dull sound
Gray hepatization	Exaggerated vesicular sound	incomplete dull sound
Resolution	Vesicular sound	resonant sound

Pneumonia

Line of treatment:

1- Antibiotic	2- Expectorant.
3- Mucolytic .	4- Anti-inflammatory.
5- Heart tonic.	6- Vitamine C.

3. Treatment

R/ Cidotryl vial 10% 1ml/40 kg. (S/C or I/M). For 3-5 days.
or Borgal 24% 3 ml / 50kg. (I/V or I/M). For 3-5 days.

R/ Expectyl 30 ml in cattle orally 3-5 day, as expectorant

R/ Bisilvon 1 amp./ 70 kg BW, I/M, as mucolytic drugs.

R/ Buta-fenil 5-10 cc / 450 Kg BW I/M, as anti-inflammatory drug.

R/ Saline, dextrose 5% or ringer lactate As supportive treatment.

R/ Adcoferine 5 cc I/M daily or Lanoxin ampoule, as heart tonic.

R/ Cevarol 1 ampoule/ 70 kg Bwt. I/M daily, as vitamin C.
or VITAC 30gm orally daily / one week.

4. Important Notes

1. In lobular pneumonia: It affects a group of lobules. The disease is slow in appearance, recurrent attack of fever 3 - 4 days. Percussion and auscultation on the chest hearing different stages of pneumonia in different area. Hyperresonant sound around the affected area.
2. Vaccination for pneumonia as Cattle Master four for Infectious Bovine Rhino Trachietis (IBR), Bovine Respiratory Syncytial Virus, Parainfluenza 3 and Mucosal disease. Vaccination to the dam in the 7 month of pregnancy and 2 weeks before parturition 5 cc S/C
3. Tyvert 1 ml/ 5 kg Bwt. per os. In cases of verminus pneumonia.

Drenching Pneumonia

1. Definition and Causes

It is a common serious disease in farm animal occur when foreign materials take their way into the lungs. It is caused by administration of liquid medication (Mineral Oil, Magnesium Sulfate...), during passage of the stomach tube, vomiting, and/or rupture of pharyngeal abscess during palpation of the pharynx.

2. Pathogenesis

Large amount of fluid is aspired ⟹ death may occur quickly.
Small amount ⟹ depends on the composition of aspired fluid
Soluble fluids as Magnesium Sulfate and Chloral Hydrate ⟹ absorbed rapidly. Insoluble oil, pus and vomits ⟹ fatal 48 - 72 hrs.

3. Clinical Findings

Cough, moist rales, consolidation of the lung and putrid odour in the breath especially in gangrenous pneumonia.

4. Diagnosis

Case history, clinical signs, moist rales and fetid breath.

5. Treatment

R/ Advocin (danofloxacin), 1 ml / 50 kg Bwt, 3 - 5 days, I/M or S/C.

R/ Predef 2 X, 10 cc / I/M / 2 days, as anti-inflammatory drug.

R/ Saline, dextrose 5% or ringer lactate, as supportive treatment.

R/ Adcoferine 5 cc I/M daily, as heart tonic.

R/ Cevarol 1 ampoule/ 70 kg Bwt. I/M daily, as vitamin C.

R/ Lasix 3 ampoule / cattle I/M, as diuretics.

6. Important Notes

1. In case of recumbancy the diseased animals should be changed regularly at least once every hour.
2. Severe cases not treated

3. Pulmonary absces:

Abscess are caused by infected emboli in other organs (metritis, mastitis and endocarditis. Also mycosis and aspirating pneumonia lead to pulmonary abscess. Symptoms as in pneumonia, inaddition purulent nasal discharge and fetid breath.Treatment by using overdose of antibiotic.

Pleurisy

1. Definition and Causes

It is an acute inflammation of the pleura. Caused by an extension of infection from respiratory tract, traumatic perforation of thoracic wall or sequel of traumatic reticuloperitonitis. Infectious pleurisy as in Contagious Bovine Pleura- Pneumonia, Infectious Equine Pneumonia and Strangles.

2. Clinical Findings

Increase of temperature and pulse rate and painful cough. Palpation and percussion on chest area reveals pain. Accelerated respiration and wholly abdominal. Inspiratory dyspnea, abducted elbow to relief pressures from lung and pleura. There is a loss of appetite, dullness and depression.

Stages of Pleurisy	**Auscultation**	**Percussion**
a. Dry stage	Friction sound	Resonant sound
b. Exudative stage (above the line)	Vesicular soun	Resonant sound
(under the line)	No sound	Dull sound
c. Adhesive stage	No sound	

3. Treatment

R/ Cidotryl Vial 10% (Enrofloxacin), 1 ml / 40 kg BW, 3 - 5 days, I/M or S/C

R/ Tussivan, Codilar or Codaphen 30 ml / cattle / orally 3-5 days as cough suppressants drugs.

R/ Predef 2 X 10 cc / I/M / 2 days, as anti-inflammatory drug.

R/ Saline, dextrose 5% or ringer lactate As supportive treatment.

R/ Adcoferine 5 cc I/M daily or Lanoxin ampoule, as heart tonic.

R/ Cevarol 1 ampoule/ 70 kg Bwt. I/M daily, as vitamin C.

4. Important Notes

1. Frictional sound means adhesion between parietal and visceral layers of pleura.
2. In Exudative stage, the exudate goes downwards by gravity to the floor of the chest cavity (pleural sacs) will give rise to line of demarcation (Pleuritic line) which is horizontal. The Pleuritic line will be changed according to the position of the animal.

Chronic Alveolar Emphysema

1. Definition and Causes

It is a permanent dilatation of the alveoli without any changes in the lung tissue. It is involve one lobe or both lobes. It is caused by chronic bronchitis, traumatic perforation of the lung, pulmonary abscess or allergic.

2. Clinical Findings

Prolonged cough, which is weak and low (usually at morning). Difficulty in breathing (expiratory dyspnea). Double expiratory movement (the first is normal but the second is wholly abdominal). Percussion on chest area gives hyper-resonant sound. The abdomen is barrel shape and decrease in the area of the lung. Heaves line is developed as a groove in the flank along the line of the coastal arch.

3. Treatment

R/ Aminophilline 3 - 5 amp. I /M - I/V, as bronchdialator drugs.

R/ Finadyne 1cc/45kg (I/M or I/V) as anti-inflammatory

R/ Expectyl, Bronchistal, Tussilar Co or Isilin, 30 ml / cattle / orally 3-5 days, as cough suppressants drugs.

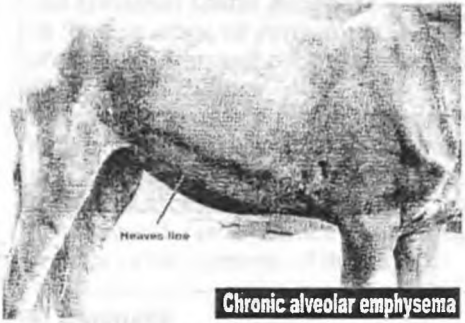
4. Important Notes

1. No direct treatment but to stop the progress of the disease by symptomatic relief
2. Supplying the animal with non nourished food contain dust for a long time predisposing for the disease.
3. Allergic emphysema due to the sensitivity of some horses to mouldy (Aspergillus fumigatous) and dusty food.
4. Full recovery can not be expected.
5. Good nourished food free from dust is necessary for animal.
6. Oxygen therapy for life threat phases in valuable equines.

Plate 4 Diseases of respiratory system

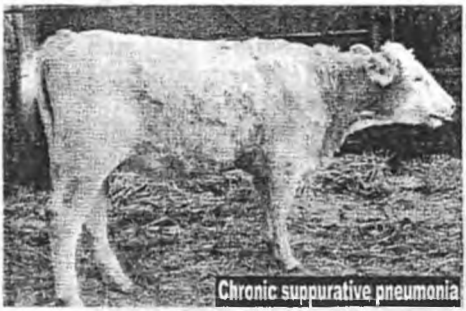

Unilateral epistaxis


Bilateral nasal discharge

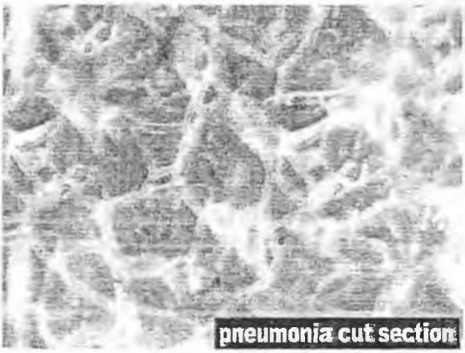


Chronic alveolar emphysema


Guttural pouch tympany


Chronic suppurative pneumonia


Atypical interstitial pneumonia


pneumonia cut section


Verminous bronchitis

Traumatic pericarditis

1. Definition and Causes

It is perforation of the pericardial sac by an infected foreign body migrating from the reticulum causing pericarditis. The predisposing factors are, late stage of pregnancy, parturition, severe tympany, prehension of food with tongue.

2. Pathogenesis

In the early stage of pericarditis ⟶ hyperemia and deposition of fibrous exudate ⟶ frictional sound is heard.
In the middle stage of pericarditis ⟶ increase inflammatory fluid and frictional sound disappear ⟶ replaced by muffling sound.
In the late stage of pericarditis ⟶ accumulated fluid prevents complete filling and congested heart failure ⟶ toxemia.

3. Clinical Findings

Sharp drop in milk production, reduced appetite, abduction of the elbows and arching of the back. Jugular pulsation, engorgement of the veins in the late stage due to congestive heart failure. Edema in the brisket region. Auscultation of the heart reveals tachycardia, muffling of the heart sound (as it comes from a distant place) and absence of lung sounds in the ventrum of the thorax.

4. Diagnosis

1. History of the case & clinical signs.
2. Pain tests & mine detector.
3. Auscultation of the heart:

First stage (dry stage):

Frictional sound is heard due to friction between parietal and visceral layer of pericardium.

Second stage (exudative stage):

Dribbling sound is heard when small amount of exudate is formed. Splashing sound (Tinkling sound) when inflammation go on and exudate increase and sometimes mixed with gases.

Third stage (Muffling stage):

Muffling sound, the exudate usually rich with fibrin and pus due to septic infection) and the heart sound is low as it comes from distant place.

5. Treatment

Surgical treatment is not practical and it is better to slaughter the animal.

6. Important Notes

For short time survival to calving:

1. Repeat pericardial drainage by means of pericardiocentesis
2. Medical treatment:

Antibiotic, Cardiac tonics, Laxative and diuretics

Congestive heart failure

1. Definition and Causes

The heart is unable to maintain circulatory equilibrium, result in congestion of venous circulation. It is caused by endocarditid (valvular stenosis or insufficiency), myocardial diseases (myocarditis or myocardial degeneration), pericarditis (traumatic and non-traumatic) and pulmonary or systemic hypertension.

2. Pathogenesis

Increase load of ejection of blood from the heart ⟶ increase heart rate, dilatation and hypertrophy.

Right side heart failure: Venous congestion lead to:

Liver congestion ⟶ portal congestion ⟶ digestive trouble (diarrhea)

Kidney congestion ⟶ tubular damage ⟶ oliguria and proteinuria.

Left side heart failure: Lead to pulmonary congestion, anoxia and edema

3. Clinical Findings

Increase heart and respiratory rates. Dyspnea, cyanosis and abnormal respiratory sound. Edema (anasarca, ascitis, hydrothorax and hydropericardium). Increase weight due to edema. Diarrhea and oliguria. Enlargement of the liver and engorgement of the vein.

4. Treatment

The animals suffering from congestive heart failure due to traumatic pericarditis must be slaughtered

5. Important Notes

1. Treat the primary cause.
2. Diuretics as Edemx 1 ampoule /70 kg BW.
3. Heart tonics as Adcoferene (5-10 cc I/M), or Pregazole 10 cc/I/M

Acute Heart Failure

1. Definition and Causes

It is inability of the heart to maintain the proper blood supply to satisfy the metabolic requirement of the body all the times. It is caused by rapid intravenous injection (such as calcium), pericarditis (traumatic and non-traumatic), excessive tachycardia or bradycardia, occlusion of coronary vessels and also during anathesia.

2. Pathogenesis

When excessive tachycardia the diastolic period is so short that filling of the ventricles is impossible and cardiac output is reduced. Tissue anoxia especially in the brain and the clinical signs are nervous in type. Pale mucous membrane due to reduction in arterial blood flow. In less acute cases respiratory distress because of pulmonary edema.

3. Clinical Findings

Dyspnea, staggering and falling, pale mucous membrane, convulsion and death. No treatment in such cases.

4. Treatment

R/ Adrenaline 1 ampoule/ 70 kg Bwt. I/V & I/M, as vasoconstrictor drugs in vasogenic failure only.

R/ Saline solution 1 - 2 liter I/V, in cases of dehydration.

R/ Blood transfusion in cases of hemorrhage & plasma in cases of shock

5. Important Notes

Do not give any cardiac stimulant and avoid vasoconstrictor drugs in hematogenic failure, dehydration, hemorrhage & shock.

Peripheral circulatory failure

1. Definition and Causes

It is reduction of cardiac output due to failure of venous return to the heart. It is caused by collection of blood in dilated splachenic vessels (vasogenic failure), this occurs due to liberation of histamine during surgery or exhaustion of adrenal cortex (milk fever). Also occur due to hemorrhage and/or in dehydration (hematogenic failure).

2. Clinical findings

Muscular weakness, subnormal temperature, increases heart rate, anorexia, convulsion and death.

3. Pathogenesis

Compensatory mechanism results in vasoconstriction and evacuation of blood stored in the spleen. Cardiac output fails and anoxia of tissue began, leads to severe damage of CNS and renal parenchyma.

4. Treatment

R/ Adrenaline 1 ampoule/ 70 kg Bwt. I/V & I/M, as vasoconstrictor drugs in vasogenic failure only.

R/ Saline solution 1 - 2 liter I/V, in cases of dehydration.

R/ Blood transfusion in cases of hemorrhage & plasma in cases of shock

5. Important Notes

Do not give any cardiac stimulant and avoid vasoconstrictor drugs in hematogenic failure, dehydration, hemorrhage & shock.

Anemia

1. Definition and Causes

Deficiency of erythrocytes count and/or hemoglobin concentration in the blood. It is caused by hemorrhage (internal or external), heavy parasitic infestation (hock worms or coccidiosis), hemolytic anemia (babesia, bacillary hemoglobinuria, leptospirosis, hypophosphatemia, water intoxication, poisonous plants). It may be due to reduction in the erythrocytes count and/or hemoglobin concentration as in nutritional deficiency, and reduction in the hemopoetic activity as in chemical poisonous, x rays, biological toxin and/or tumor of bone

2. Clinical Findings

Pale mucous membrane, muscular weakness, depression, and inability to work, sweating and coldness of extremities. Respiratory distress due to increase in depth in respiration without much increases in rate. In severe hemolytic anemia muscular tremor, labored breathing, subnormal temperature and death as result from anoxia.

3. Treatment: Treat the primary causes

R/ Blood transfusion (1 liters /100Kg B.W.) I/V.

R/ Varolex B_{12} 1 vial / cattle, I/M / daily /3 - 5 days.

R/ Brewer yeast or egg yolk. As digestive aids.

R/ Multivitamin cattle 20 - 30 cc, sheep & goat 5 - 10 cc I/M.

R/ Arsinal 15 cc I/M daily / 3 days.

6. Important Notes

1. Whole blood or plasma should be transfused between the similar breeds. Give at first small amounts (50 - 100 ml S/C) 30 minutes before the transfusion to detect the anaphylactic reaction.
   The rest amount (3 - 6 liter or 1 liter / 100 kg Bwt) should be injected I/V within 72 hours. You may draw blood directly from the donar and inject into the recipient or anticoagulant is added 0.25 g of Sodium Citrate / 100 ml of blood and also it better to add antibiotic, it may be stored in the refrigerator for 3 - 4 days. Filtration by sterile gauze is necessary before use.
2. Splenomegally and jaundice in hemolytic anemia are due to blood parasites.
3. Hemoglobinuria occurs in rapid hemolysis and 40-50% of RBC is destroyed.

Edema

1. Definition and Causes

Excessive accumulation of fluid transudate in the tissue spaces and body cavities caused by increase hydrostatic pressure (congestive heart failure) or decrease osmotic pressure (liver cirrhosis, renal disease or heavy parasitic infestation), also obstruction of lymphatic vessels, allergic condition or infectious diseases such as black leg and malignant edema.

2. Pathogenesis

Increase in hydrostatic pressure or decrease in osmotic pressure lead to return of the fluid to capillaries and accumulation in the serous cavities, results in edema.

3. Clinical Findings

Edematous swelling are soft, painless, and pit under pressure. Distension of the abdomen in ascitis (fluid thrill on tactile palpation). Embarrassment of respiration, collapse of ventral parts of the lungs, muffled heart and respiratory sound, moist rales in pulmonary edema

3. Treatment: Correct of the primary cause of the disease.

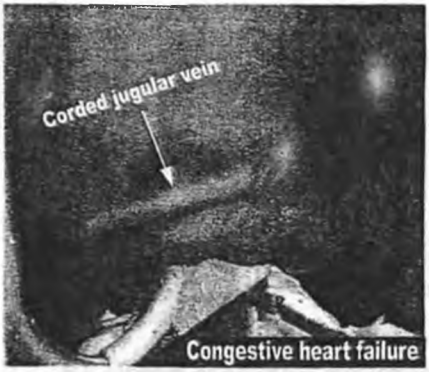
R/ Lasix 1 ampoule / 70 kg Bwt or Super-Retic orally 20 g daily 5 days or Nephton 20 g daily / 5 days as diuretics.

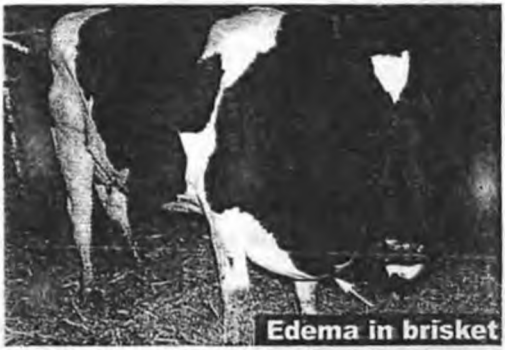
R/ Iodine ointment 10%. 2 times daily in local edema.

4. Important Notes

In hydropericardium, drainage from pericardial sac, aspiration of fluid must be carried slowly to avoid acute dilatation of splanchinic vessels and peripheral circulatory failure (it well temporary relief because the fluid is rapidly reaccumulate).

Plate 5 Diseases of cardiovascular system





Pyelonephritis

1. Definition and Causes

It is highly fatal chronic purulent infection of the pelvic portion of the kidney, ureter and bladder developed by ascending infection from urinary tract. It is caused by infection with corynbacteria renal.

2. Clinical Findings

A). **Postparturient type:** Loss of conditions, emaciation, fetid discharges from genital tract, pus and blood in the urine and vagina.

B). **Ureter colic type:** Restlessness, kicking of the abdomen, arched back, passage of blood clots and casts through the Ureter frequent hemorrhage cause paleness of mucous membrane.

C). **Cystitis type:** Frequent urination, straining and passage of small amount of blood urine.

Diagnosis

Rectal palpation revealed enlarged kidney (Left kidney can palpate), loss of lobulation and painful on palpation. Presence of blood (RBC), pus and casts in the urine.

3. Treatment

R/ Pentomycin 1 ml / 25 kg BW I/M 5-7 days

R/ Coliurinal eff. 10 g / 200 ml water as urinary antiseptic.

R/ Sodium acid phosphate 125 g orally/daily, to change urine pH.

R/ Super-Retic 20 gm orally daily / 5 days / cattle or Nephton or Potassium Citrate orally or Lasix ampoles I/M as diuretics.

R/ Novacid or Novalgen 25 cc I/V, as sedatives.

Nephritis

1- Defination and causes:

It is inflammation of the kidneys. Caused by bacterial infection, mineral irritant or poisoning, excessive skin damage, trauma of the kidney, cold, severe gastroenteritis and/or constipation.

2. Clinical findings

Oliguria and increase of albumin content in the urine. The animal stands with arched back and stiffness gait, tenderness and pain on manipulation of the kidney, fever in case of infectious agent, colic in equine and vomiting in dogs. When both kidneys are affected uremia and coma may found. In late stage edema appears in the lower part of the chest, abdomen and legs.

3- Diagnosis:

Case history, clinical signs, albuminuria and with the presence of epithelial tissue in the urine.

3. Treatment

R/ Procaine penicillin as 5-7 million IU every 12 hours I/M /5-10 days cattle & mare.

R/ Coliurinal eff. 10 g / 200 ml water as urinary antiseptic.

R/ Sodium acid phosphate 125 g orally/daily, to change urine pH.

R/ Lasix 3 ampoules / cattle I/M

R/ Super-Retic 20 gm orally daily / 5 days / cattle or Nephton or Potassium Citrate orally as diuretics.

R/ Novacid or Novalgen 25 cc I/V or I/M as a sedative.

Cystitis

1. Definition and Causes

It is inflammation of the bladder, characterized by frequent and painful urination. It is caused by bacterial infection, injuries to the urethra during coitus or calculus, faulty cathetetization and also after calving. It may be secondary to vaginitis or endometritis.

2. Clinical Findings

Frequent and painful urination and passage of small amount of urine. Arched pack and fits of colic in acute cases. Retention of urine may occur if the urethra becomes blocked with pus or blood. Painful palpation of the bladder.

3. Treatment

R/ Procaine penicillin, cattle 5-7 million IU every 12 hours I/M /5-10 days
R/ Uricol eff. 10 g / 200 ml water as urinary antiseptic.
R/ Sodium acid phosphate 125-g orally / daily.
R/ Lasix 3 amp. /cattle I/M or Potassium Citrate orally, as diuretics.
R/ Novacid or Novalgen 25 cc I/V, as sedatives.

4. Important Notes

1. Hematuria: It means the presence of blood constituents in the urine. It is caused by trauma of the kidney, septicemia accompanied by vascular damage, anthrax (pre-renal). Acute glomerulonephritis, tubular degeneration by bacterial toxins and sulfanilamide intoxication (renal). Cystitis, urolithiasis, rough manipulation of the catheter, tumor of renal tract and also hemorrhage of genital tract (post-renal).

2. Clinical findings: Blood clots in the urine in severe cases. In common cases the color varies from deep red to brown coloration. In less severe cases only cloudiness settle to form red deposits. Blood originating from the kidney is mixed with urine and present in equal concentration in all samples, while blood originating from urethra appears in the beginning of urination. Blood originating from urinary bladder appears in the end of urination.

3. Diagnosis

1. Centrifugation to the urine sample or let the test tube for a while their will be a blood sediment.
2. Microscopically RBC will be seen on the slide and urine strips test.

4. Diffrential diagnosis from hemoglobinuria

Presence of hemoglobin in the urine due to rapid destruction of large number of RBC. It is caused by babesiosis, bacillary hemoglobiuria, water intoxication, leptospirosis, chronic copper poisoning and hypophosphatemia. Centrifugation to the urine sample their will be no blood sediment.

Urolithiasis

1. Definition and Causes

It is obstruction of the urethra in castrated male ruminants characterized clinically by complete retention of the urine, unsuccessful effort to urinate, distension of the bladder and sequels of urethral perforation may occur and also may rupture of the bladder.

2. Pathogenesis

Unilateral obstruction of ureter ⟶ hydronephrosis
Bilateral obstruction of ureter ⟶ anuria and uremia
Obstruction in the urethra (sigmoid flexure) ⟶ rupture of urethra & urinary bladder

3. Clinical Findings

The animal is restlessness with frequent attempts to urinate and only drops of urine pass. Palpation of urethra may reveal obstruction (pulsation). Hematuria, anorexia, vomiting (dog & cat). Rupture of urethra may lead to swelling the sheath of prepuce.). Rupture of bladder lead to peritonitis, urine smell and exploratory puncture of the ventral abdominal wall reveals the presence of urine. The presence of calculus in the urinary bladder leads to cystitis. Symptoms of uremia develops and animal dies from coma.

3.Treatment

R/ Neurazine 3 ampoule in cattle &1 ampoule in calf I/M,as tranqulizer
R/ Voltarin 1 ampoule / 70 kg Bwt / cattle & horse, I/ M as smooth muscle relaxant.
R/ Novalgen 25 cc I/V, as sedative
R/ Procaine penicillin as 5-7 million IU every 12 hours I/M /5-10 days
R/ Urolithin eff. 10 g / 200 ml water as urinary antiseptic.
R/ Sodium acid phosphate 125-g orally/daily.

4. Important Notes

1. Stages of calculus formation

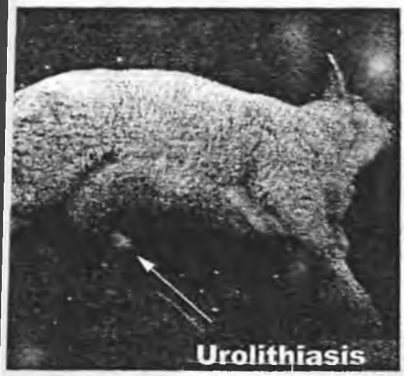
*Nidus formation: Desquamated epithelial or necrotic tissues, leukocytes, albumin or organic elements. Caused by local infection of urinary tract and/or vitamin A deficiency.

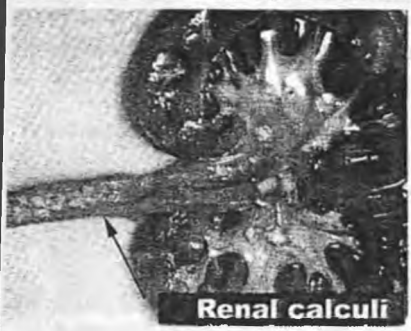
*Precipitation of salts (urine is highly saturated with solutes). Increase of colloidal state of the urine due to excessive intake of mineral salts, excessive oxalate, concentrated urine in dehydration and change in pH of urine.

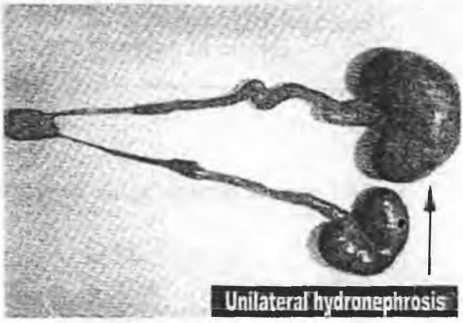
*Concretion: It is the cementing of precipitate salts to form the calculus as mucoprotein, which increased by feeding on heavy concentrate with low roughage content.

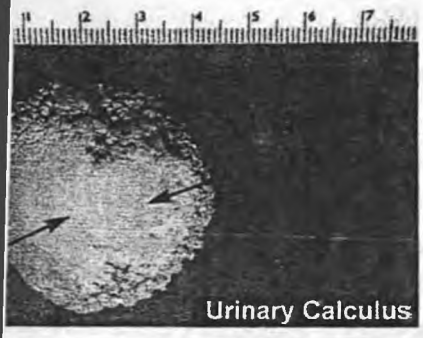
2. Surgical interference in rams by cutting the urethral process.

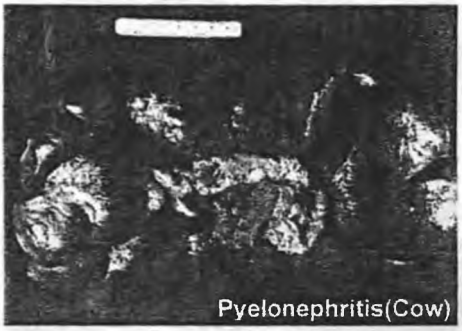
Plate 6(a) Diseases of urinary system

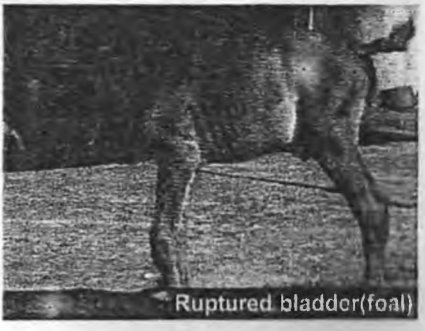












Encephalitis

1. Definition and Causes

It is inflammation of the brain. It is caused by viral infection (rabies, malignant head catarrhal fever, equine infecious encephalomylitis, scrapie and louping ill in sheep), bacterial (listeriosis, salmonellosis and swine erysipelas), parasitic, mycotic, physical (sun stroke) and chemical agents (irritant gases).

2. Clinical Findings

Rise of temperature, anorexia, depression and increase heart rate. Excessive response to normal stimuli with excitment and mania. Clonic convulsions, accampanied by nystagmus, muscle tremor of the face and limbs and frothy salivation. Incoordination and walking in circles.

3. Treatment

R/ Streptopenicid (pencillin & streptomycin) large animal 2 vials I/M every 12 hours./ 3-5 days.
or

R/ Borgal 24% (Sulphadoxin & Trimethoprim). 3 ml / 50 kg a second dose after 48 hrs. may be needed, I/V & I/M.

R/ Novacid 25 cc I/V, as antipyretic drugs in case of fever.

R/ Predef 2 X 10 cc / I/M / 2 days, as anti-inflammatory drug.

R/ Dextrose 25%, as supportive treatment.

R/ Neurazine 3 ampoules in cattle & 1 ampoule in calf I/M.
Used during the excitment stage only.

4. Important Notes

1.Other disease causing nervous manifestation such as hypomagnesemia , hypoglycemia, milk fever and vitamin A & copper deficiency. Urea poisoning, spinal abscess, meningitis, sinusitis & trauma in C.N.S. Tetanus, enzootic ataxia, IBR, coenurosis and babesiosis.

Meningitis

1. Definition and Causes

It is inflammation of the meninges. It is caused by viral infection (malignant head catarrhal fever and bovine encephalomylitis), bacterial (listeriosis, salmonellosis and tuberculosis). Extesion of inflammation as in case of encephalitis.

2. Clinical Findings

Rise of temperature, rigidty of the neck, excitment and mania followed by convulsions and death.

3. Treatment

R/ Streptopenicid (pencillin & streptomycin) large animal 2 vials I/M every 12 hours./ 3-5 days

R/ Novacid 25 cc I/V, as antipyretic drugs in case of fever.

R/ Predef 2 X 10 cc / I/M / 2 days, as anti-inflammatory drug.

R/ Dextrose 25%, as supportive treatment.

R/ Neurazine 3 ampoules in cattle & 1 ampoule in calf I/M.
Used during the excitment stage only.

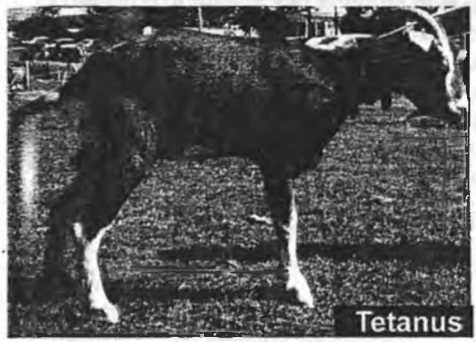
4. Important Notes

1. **Militis:** It is inflammation of spinal cord and usually associated with viral encephalitis and Rabies. It is uaually end by paralysis.
2. **Hydrocephalus:** It is accumulation of CSF in the cerebral ventricles. It may be congenital due to embryonic defect or acquired due to obstruction of drainage by inflammation, tumor and Avitaminosis A.
3. **Cerebral Apoplexy (Brain hemorrhage):** It means rupture of blood vessle of the brain. Caused by increase of blood pressure or traumatic injuries of the skull. Characterized by nervous shock, unconciousness, convulsion, coma and death. The formed hematoma will compress on part of the brain causing loss of functions controlled by centers located in this part lead to hemiplegia (paralysis of one side of the body), paraplegia (paralysis of posterior part and hind legs of the body) or monoplegia (paralysis of one limb or one muscle).
4. **Chorea:** It is nervous disease characterized by involuntry movments of individual muscle or group of muscle without loss of sensation. It is usually a sequel to certain diseases such canine distemper, encephalitis, meningitis and brain tumers. The muscles of the neck, eye lids and lips are usually affected.
5. **Trauma of the spinal cord:** It caused by dislocation, frcuture and/or concusion of the vertebrae. Migration of parasitic larvae as hypoderma bovis, toxacara canis and cerebrospinal nematodiasis. Characterized by flacid paralysis and fall in blood pressure. Recovery may occur 1-3 weeks if nervous tissue not destroyed.

Plate b(b) Diseases of nervous


Facial Paralysis

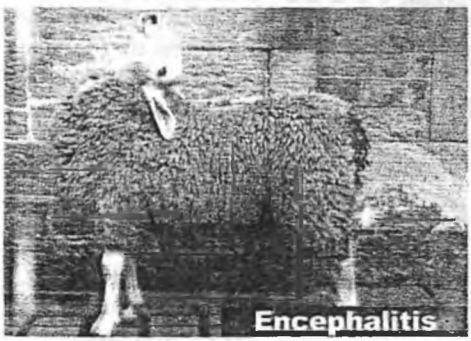

Rabies


Tetanus


Otitis media


Meningitis


Meningitis


Encephalitis


Encephalitis

Milk Fever
Parturient Paresis

1. Definition and Causes

It is a febrile disease occurs most commonly at/or after parturition (12-72 hours). It occurs in high producing cows 5-10 years. It is caused by Hypocalcemia.

2. Clinical Findings

A). Excitement stage: Restlessness, hypersensitivity, tremor and tetany. Protrusion of tongue and grinding of teeth. Shaking of head with stiff gait.

B). Sternal Recumbancy: Depression, drowsy, sternal recumbancy and unable to rise. The head turned in to the flank. Hypothermia with cool skin. Decreased hearts sounds and increase heart rate (180/minute). Dry muzzle and eye, dilated pupil, ruminal stasis, secondary bloat and constipation. No anal reflex (anal relaxation).

C). **Lateral recumbancy:** Cows always comatose, lateral recumbancy and unable to set up. Hypothermia and increase heart rate up to 120/minutes. The animal dies after 12-24 hours.

Complication: Hypomagnesemia, dystokia, and uterine prolapse

Clinical pathology:

Serum calcium is below 5 mg/dl, may be 2 mg/dl (Normal level 10 mg/dl). In organic phosphorus is decreased to 1.5-3 mg/dl

Increase magnesium levels to 5 mg/dl.

3. Treatment

R/ Calcium borogluconate 25% (large cow 800 - 1000 cc & small cow 350 - 500 cc) half dose I/V and half dose S/C. daily till recovery.
or Ca. D. Mg or CAL-BOR-MAG or Calcium gluconate.

R/ Predef 2x or dexamethazone, 10 cc I/M, as corticosteroid drugs.

R/ VITA-JECT, AD_3E 5-10 cc I/M, as a single dose of vitamins.

R/ Adcoferine 5-10 cc I/M, as heart tonic.

R/ Glucose 25% 2-3 L I/V daily, as a supportive treatment.

4. Important Notes

1. You must give the animal easily digested food and clean water supply.
2. Clean bedding and rotation of the animal to avoid ulceration and hypostatic congestion.
3. Avoid sudden or complete emptying of the udder.
4. Under dosing of calcium therapy is common error.
5. Administration of vitamin D and feeding ration containing calcium in late pregnancy as prophylactic therapy. Calcium injection after calving or 2 days before and after parturition as prophylactic dose.
6. Subnormal temperature and lateral recumbancy are cosidered as a bad prognosis.

Hypoglycemia

1. Definition and Causes

It is an impairment of metabolism of carbohydrates and volatile fatty acids leading to intoxication from ketoses in the blood. It is caused by hypoglycemia occurring in the first month of lactation in cattle and late pregnancy in ewes.

2. Clinical Findings

A). Wasting form: Decrease in appetite, milk production and body weight. Depression and disinclination to move and eat. Decrease ruminal movement, but normal pulse, respiration and temperature. Ketoses smell on the breath and milk. Feces are firm and dry. Woody cow due to wasting and loss of skin elasticity.

B). Nervous form: The animal walks in circles and crossing the legs. Apparently blindness. Vigorous licking of the skin. Depraved appetite. Hyperthesia with moderate tremor and tetany. Recurrent attack of nervous signs may occur 8 - 12 hours.

Clinical pathology: Hypoglycemia as the serum glucose levels 20 - 40 mg% (Normal range 50 - 70 mg%). Increase of ketones of blood (10 - 100 mg%), urine (80 - 1300 mg%) and milk up to 40-mg %.

3. Treatment

R/ Glucose 25% 1-2 liter I/V twice daily for 3 - 5 days.

R/ Treacle and glycerol 0.5 - 1 liter orally.

R/ Predef 2X 10 cc I/M daily for 2 days (increase blood glucose).

R/ Ca. D. Mg 500 ml I/V.

R/ Cobalt and B_{12} to (help in the proper metabolism of propionic acid).

4. Important Notes

1. Avoid sudden changes of diet from good quality roughage to high protein diet (more ketoenic).
2. Starvation m ay result in gluconeogensis that results in increased level of ketones.

Ovine Ketosis
Pregnant Toxemia

1. Definition and Causes

It is an impairment of metabolism of carbohydrates and volatile fatty acids leading to intoxication from ketoses in the blood. It is caused by hypoglycemia occurring in the first month of lactation in cattle and late pregnancy in ewes.

2. Clinical Findings

The syndrome is similar to the nervous form in cows, in addition to ataxia and locomotor dysfunction, twitching of the muscles around the eyes and the ears, dysphasia and dyspnea. In the terminal stage, the animal appears to be blind, not responding to stimuli, recumbant, comatose, cyanosis and death usually occurs 3-7 days after the first clinical signs are observed. The mortality rate in pregnant toxemia is about 90%.

3. Treatment

In mild cases

R/ Glucose 25% 200-300 ml I/V twice daily for 3 - 5 days.

R/ Treacle and glycerol 100-200 ml orally.

R/ Predef 2X 2cc I/M daily for 2 days (increase blood glucose).

R/ Ca. D. Mg 100 ml I/V.

R/ Cobalt and B_{12} to (help in the proper metabolism of propionic acid).

In severe cases

Induce abortion or cesarian section is necessary.

4. Important Notes

1. Avoid sudden changes of diet from good quality roughage to high protein diet (more ketogenic).
2. Starvation may result in gluconeogensis that results in increased level of ketones.

Hypophosphatemia
Postparturant hemoglobinuria

1. Definition and Causes

It is a metabolic disease of high producing dairy cows, usually occurring 2-6 weeks after parturition. The diseases usually occur in buffaloes at the 5th -7th months of pregnancy. It is caused by low phosphorus level in the blood resulting from low phosphorus intake either by ration or grazing on pasture for long period 3-4 months as barseem.

2. Clinical Findings

Anorexia, pica and decrease of milk yield. Hemoglobinuria, anemia, general weakness & pale mucous membrane associated with normal body temperature, finally jaundice and dehydration may occur. In the terminal stage, gangrene and/or sloughing of the digit and tips. Ketosis and locomotion disturbances may develop. Sometimes death occur due to anemic anoxia

Clinical Pathology

Decrease of serum inorganic phosphorus from 4-5 mg.% to 2-3 mg.% and 0.4 mg.% in severe cases.
Decrease of serum glucose level.
Decrease of RBC, Hb and PCV.

Differential diagnosis

Babesiosis (fever, hemoglobinburia, tick on the animal, blood film), bacillary hemoglobinburia, water intoxication, leptospirosis and urinary tract affections (hematuria).

3. Treatment

R/ Sodium Acid Phosphate or Sodium Dibasic Phosphate 20% (60 g dissolved in 300 ml DW to be given by I/V route) followed by S/c dose after 12 hours intervals for 3-5 days. Also oral administration of 80 gm Sodium Acid Phosphate or 120 gm bone meal in the ration daily till complete recovery.
R/ Catozal or Tonophosphane 50 cc I/M or I/V daily 3 - 5 days
R/ Super-Phos (vitamin A & D, Iron and phosphorous) 100 g orally daily/ week.
R/ Predef 2X 10-cc I/M daily/3 days (gluconeogenesis).
R/ Arsinal 15 cc I/M daily / 3 days.
R/ Glucose 25% for treatment of ketosis. As supportive treatment.
R/ Blood transfusion 3 - 4 liter I/V, see method incase of anemia

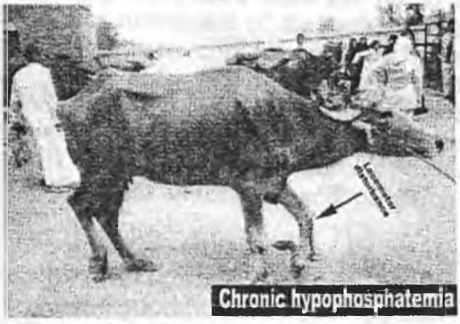
4. Important Notes

1. The diseases mainly occur in winter as the animals feeding mainly on barseem (Nov.- May.). Addition of bran and bone meal to the ration are necessary during barseem season.
2. When the cows down before calving it is better to give phosphorus therapy as prophylactic dose.

Plate 7 Metabolic diseases


Hypocalcemia (lateral recumbancy)


Hypocalcemia (sternal recumbancy)


Chronic hypophosphatemia


Hypophosphatemia


Ketosis (wasting form)


Ketosis (nervous form)


Pregnancy toxemia


Pregnancy toxemia

Hypomagnesemia tetany

1. Definition and causes

It is a highly fatal disease of lactating cow and small ruminant, after parturition by two months. It is caused by hypomagnesemia. It occurs due to feeding grass pasture low in magnesium content, grasses with high potassium content (cereal crops), pasture top dressing with nitrogen partial starvation and recurrent diarrhea.

2. Clinical Findings

Acute stage: Sudden onset of anxiety, muscle tremor, ear twitch, hyperthesia, staggering in gait and easy falling. Tetanic-clonic convulsions with opisthotonus. Jaw champing, frothy salivation and bellowing. Protruded third eye-lid to cover most of the eyeball, and continuos movement of the eyeball. The eye-lid may be retracted. Quite period between convulsions. Hypothermia, increases respiratory and heart rate. Response to treatment with magnesium solution I/V very good (untreated cases die after 30-60 minutes).

Subacute stage: Loss of appetite, suppressed rumination and low milk yield. Staggering in gait, muscular tremor. Mild tetant of hind limb and tail. Frequent defecation and urination. Spontaneous recovery in few days.

Chronic stage: Gradual loss of condition, some sudden deaths, dullness, depressed milk yield. The cow finally may pass into convulsions and dies unless prompt treatment is applied.

Differential diagnoses: nervous form of ketosis, rabies, acute lead poisoning, vitamin A deficiency and tetanus.

3. Treatment

R/ Neurazine 3 ampoules in cattle & 1 ampoule in calf I/M.
To handle the animal quietly before treatment.

R/ Magnesium Sulfate (33 g in 500 ml DW), filter and sterilize before use (slowly I/V please follow heart and pulse rate). Followed by S/C injection of 200 cc Magnesium Sulfate (25-50%). In calf 100 cc Magnesium Sulfate 10% S/C.

R/ Ca. D. Mg or CAL-BOR-MAG. 500 ml I/V.

R/ Magnesium (Oxide, Sulfate or Phosphate) 60 g orally / daily.

4. Important Notes

1. **Calf tetany:** Stiffness gait due to stiffness joint. Hyperthesia and frightened easily. Stretching of the head either upward or sideways. Tetanic spasms of jaw muscle with rapidly open and close the mouth with salivation. Retracted eyelids show great of the white. Calf tetany observed in calves from 3 months age and upward, that receiving exclusively milk diet.
3. Avoiding use of potash fertilizer (interfere with Mg. absorption & also hyperkalemia increase the urinary excretion of Mg.).
4. Lactating cows need 20 g. of Mg. daily to absorb only 4 g. The winter pastures (clover) fulfill this amount, while grasses gives only 10 g.

Azoturia

1. Definition and Causes

It is acute disease of equine occur during exercise after a period of inactivity and feeding on full ration.

2. Clinical Findings

Acute form: Signs develops 15 - 60 minute after the beginning the exercise. Profuse sweating, stiffness of gait and disinclination to move. The signs may disappear if the horse is given complete rest. Dog sitting position followed by lateral recumbancy. Sever pain accompanied by restlessness. Gluteal and quadriceps muscles are hard, edematous, and painful. Deep red brown (coffee coloured urine). Retention of urine and constipation may present.

Subacute form: Signs are mild and myoglobinurea are absent. Lameness and limitation of movement of hind limb. If exercise is stopped lameness may recover 2 - 4 days.

3. Treatment

R/ Sodium Bicarbonate 150 - 300 g orally for acidosis.

R/ Sodium Bicarbonate (2%) 1 - 2 liter for treatment of acedemia.

R/ Finadyne 1 ml / 45 kg Bwt. I/M as anti-inflammatory.

R/ Insulin 100 - 200 units S/C daily 3 - 4 days.

R/ Viteselen (vitamin E & selenium) 5 ml - I/M.

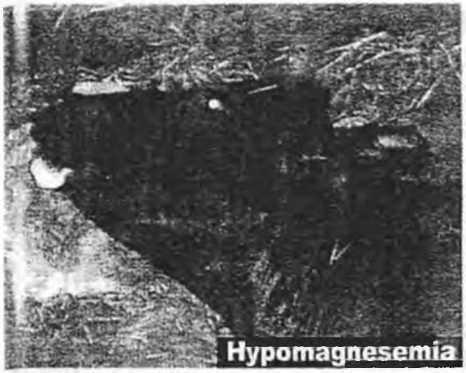
R/ Saline & ringer Lactate 2-4 liters I/V.

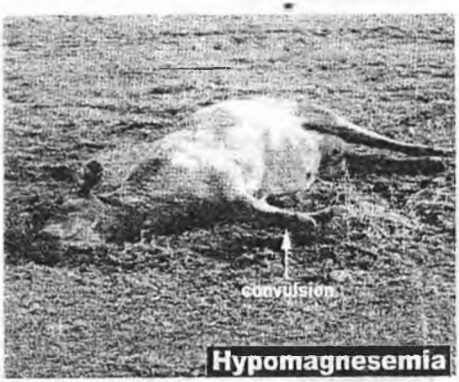
R/ Paraffin oil 2-3 liter orally as a purgative drugs.

4. Important Notes

1. Give the animal easily digested food as green fodder and hay and avoids food rich in nitrogen.
2. Apply catheter to avoid urine retention.
3. Hot fomentation to relief discomfort.
4. Application of heavy bedding, turn the animal every 4 hours to avoid hypostatic congestion.
5. Recumbancy is usually abad prognosis.
6. Reduce the grain ration to half when the horse is at rest.

Plate 8 Metabolic diseases and downer cow syndrome


Hypomagnesemia



Hypomagnesemia


Calf tetany

Calf tetany


Downer cow

Downer cow

Azoturia

Azoturia

Rickets

1. Definition and Causes

It is a disease of young growing animals characterized by defective calcification of growing bone. It is caused by calcium, phosphorus and/or vitamin D deficiency.

2. Clinical Findings

Stiffness in gait, enlargement of the limb joints specially in the forelegs and costochondral junction (button like projection). The long bone shows abnormal curvature, lameness and tendency to lie down. Arching of the back. Irregular and delayed rupture of the teeth. Dyspnea and chronic rumen tympany in severe cases. Finally, hypersensitivity, tetany, recumbancy and death.

3 . Clinical Pathology

Decrease in serum calcium level 4 - 5 mg% (normal 10 mg%).
Decrease in serum phosphorus level 1 - 2 mg% (normal 6 mg%).

4. Treatment (less sever cases)

R/ Calcium gluconate 100 - 200 cc I/V or I/M/ calf.
50 - 100 cc I/V or I/M / lamb.

R/ Super-Phos (vitamin A & D, Iron and phosphorous) 25 g / daily / calf & 10 g / daily / lamb.

R/ Tonophosphan, Calves 5 -10 ml, lambs 1-2 cc, I/M & I/V.
or Catosal. Calves 5 -10 ml, lambs 1-2 cc, I/M & I/V.

R/ VITA-JECT, 3 - 5 cc in lambs & 7 - 10 cc in calves, I/M.

R/ Super-Vitamix 25 g / daily / calf & 10 g / daily / lamb.

6. Important Notes

1. In severe deformity treatment is of no value.
2. Allow exposure of the animal to sunlight and avoid dampness.
3. Bone meal and bran daily in the ration.
4. Daily requirement of Calcium (g) Phosphorus (g) Vitamin D (U/kg)

Cattle	40	20	10
Horse	14	13	10
Sheep	5	3	10

Osteomalacia

1. Definition and Causes
It is a disease of mature animal affecting bone in which endochondrial ossification has been completed. It is caused by calcium, phosphorus and/or vitamin D deficiency. Lactation and pregnancy are predisposing causes for this disease.

2. Pathogenesis
Increase resorption of bone mineral to supply the needs of pregnancy and lactation ⟶ osteoporosis, weakness and deformity of bone.

3. Clinical Findings
In the early stages: Lower productivity, fertility and loss of condition. (the main cause phosphorus deficiency).

In the late stage: Painful condition of the bone and joints, stiffness in gait, lameness (shifting from leg to leg). Crackling sound while walking and arched back. Deformity of pelvis and dystokia may occur. Permanent recumbence and death from starvation.

4. Treatment

R/ Calcium gluconate 0.5 - 1 liter I/V / Cattle.

R/ Super-Phos (vitamin A & D, Iron and phosphorous) 50 g orally daily/week.

R/ Tonophosphan, 25 - 30 cc I/M & I/V / Cattle.
or Catosal. 25 - 30 cc I/M & I/V / Cattle.

R/ Vitamin AD3E Cattle 10 cc I/M

R/ Super-Vitamix 50 g / daily / cattle.

5. Important Notes

1. Bone meal and bran daily in the ration

2. In severe deformity treatment is of no value.

3. Allow exposure of the animal to sunlight and avoid dampness.

Copper deficiency

1. Definition and Causes

Hypocupremia occur in cattle and newborn animals. The primary causes are inadequate intake of copper in the diet. Secondary causes such as an increase in molybdenum and zinc in the diet. Also parasitic infestation (bunostomum) decrease copper absorption.

2. Pathogenesis

Copper play an important role in tissue oxidation —copper def.→ inadequate keratinization of the skin, wool and hair.

Copper is necessary for the reutilization of iron —copper def.→ anemia → myocardial degeneration → anemic hypoxia

Copper help in the formation of myelin sheath —copper def.→ demyelination in lamb.

Increase molybdenum in the diet → reduce copper storage and utilization.

3. Clinical Findings

*General symptoms: Unthriftness, loss of milk production and anemia. Rough coat and increase tendency to bone to fracture. Poor growth in calf, stiffness and enlargement in joint.

*Falling disease syndrome in cattle: The animal through up their head, bellow and fall with attempt to rise and end with death.

*Beat scour syndrome of cattle and sheep: Persistent diarrhea with the passage of watery yellow green to black feces with an offensive odour.

*Steely wool syndrome in sheep: Fine wool becomes limp, glossy and losses its crimp developing a straight steely appearance. Anemia and scouring.

*Enzootic ataxia (Swayback) in lambs and goat kids: Incoordination of the hind limb, accelerated heart and respiratory rates, excessive flexion of joints and knuckling over the fetlock. Falling and paresis which start at the hind limb and the animal die from starvation.

4. Treatment

R/ Copper Sulphate

Cattle 8 - 10 g / orally / weekly for 3 - 5 weeks.
Calves 4 g / orally / weekly for 3 - 5 weeks.
Lamb 2 g / orally / weekly for 3 - 5 weeks.

R/ Vitamin AD3E cattle 10 cc I/M

R/ Super-Vitamix 50 g / daily / cattle.

5. Important Notes

Minimum dietary requirement of Copper 10 mg / kg for cattle and 5 mg / kg for sheep

Zinc deficiency

1. Definition and Causes

It is a chronic non-inflammatory disease affecting the epidermis of the skin. It is caused by deficiency of zinc in the diet or by deficiency of unsaturated fatty acid. Secondary deficiency due to excess of calcium or copper in the diet.

2. Pathogenesis

Zinc deficiency ⟶ decrease feed intake ⟶ depression in growth rate. Failure of keratinization ⟶ parakeratosis.

Retard testicular development ⟶ complete cessation of spermatogenesis.

3. Clinical Findings

Clinical signs developed within two weeks after deficiency. Parakeratosis and alopecia in muzzle, vulva, anus, tail, head, ears, back of hind legs, flank and neck. Stiff gait, swelling of the hocks and knees and wrinkling of the skin of the legs and scrotum. Stunted growth and decrease in the weight of the newborn animals. Wool eating and infertility in sheep.

4. Treatment

R/ Zinc Sulfate 2 - 5 g / Cattle, and 40 mg / orally daily.

or

R/ Zinc Sulfate or Carbonate 200 mg / kg daily in the ration for 3 - 5 weeks.

R/ Super-Vitamix 50 g / daily / cattle.

5. Important Notes

1. For prophylaxis oral administration of zinc sulphate in the dose of:

Cattle 25 mg orally	Calves 25 mg orally
Sheep 5 mg orally	Lambs 2.5 mg orally

2. Restriction of calcium in the diet.

Iodine deficiency

1. Definition and Causes
The cardinal signs of iodine deficiency is goiter. It is caused by the deficiency of iodine intake or high intake of calcium in the diet.

2. Pathogenesis
Iodine deficiency ⟶ decrease thyroid production of thyroxin and stimulation of secretion of thyrotropine hormone by the pituitary gland ⟶ hyperplasia and enlargement of thyroid gland.

3. Clinical Findings
Loss of condition, decrease milk production, failure of estrous in cow, weak off spring and partial or complete alopecia. Enlargement of thyroid gland.

4. Diagnosis
Clinical signs.
Clinical pathology plasma protein bound protein below 8 ug / 100 ml blood is considered deficiency (normal 10 - 14 ug / 100 ml blood).

5. Treatment

R/ Potassium Iodide 0.8 - 1 mg / kg dry matter / feed, daily intake in lactating & pregnant Cattle.

6. Important Notes

1. For prophylaxis individual dosing of pregnant ewe on two occasions during the fourth and the fifth months of pregnancy with 280 mg of Potassium Iodide or 390 mg Potassium Iodine is effective to prevent goiter in lambs.

2. Restriction of Calcium in the diet.

3. Weekly painting inside thigh with Tincture Iodine 4 ml in Cattle and 2 ml in sheep.

4. Over dosing of Iodine may lead to toxicity.

Cobalt deficiency

1. Definition and Causes

Cobalt is an essential dietary element for cattle and sheep because it is necessary for the synthesis of vitamin B_{12} by the bacterial flora in the rumen. Cobalt deficiency in the diet causes the disease.

2. Pathogenesis

Cobalt play an important role in the formation of thiamin (B_1), nicotinic acid and cynocobalamin (B_{12}). Decrease in cobalt ⟶ loss of appetite ⟶ death due to starvation.

3. Clinical Findings

No specific signs are characteristic for cobalt deficiency. Gradual decrease in appetite, pale mucous membrane, loss of body weights emaciation, weakness and pica. Retardation of growth, lactation and wool production. Infertility, diarrhea and lacrimation.

4. Diagnosis

The response of animal to dietary supplementation with cobalt is generally accepted as diagnostic test.

5. Treatment

R/ Cobalt Sulfate 1 g / Cattle and 0.5 g / Sheep and Calf orally.

R/ Varolex B_{12} with , liver extract
Cattle 1 vial, Sheep 1/2 vial I/M.

R/ Catozal or Tonophosphane
50 cc I/M or I/V daily 3 - 5 days as a general tonic.

Vitamin A deficiency

1. Definition and Causes

Deficiency of vitamin A is caused by an insufficient supply of the vitamin in the ration or defective absorption from the alimentary canal. Secondary causes such as chronic disease of the liver or intestine, continued injection of mineral oil, high environmental temperature and high nitrate content in the feed which reduce the conversion of carotene to vitamin A.

2. Pathogenesis

*Night vision: Vitamin A is essential for photochemical bases of light adaptation. Low vitamin A in the blood will result to night blindness.

*Bone growth: Vitamin A is necessary to maintain the normal position and active of osteoblast and osteoclast. Vitamin A deficiency lead to narrowing of foramina so that arteries, veins and nerves may become partially included. Stenosis for optic foramina will lead to total blindness.

*Epithelial tissue: Vitamin A deficiency lead to atrophy of all epithelial cells which has secretory function (salivary and urogenital) dystokia, infertility and enteritis.

*Embryonic development: Vitamin A is essential for organ formation so that its deficiency lead to congenital defect and congenital hydrocephalus.

3. Clinical Findings

Night blindness (inability to see in dim light), xerophthalmia (thinking and clouding of the cornea in dogs and calves and thin serous mucoid discharge in other species). Heavy deposits of bran like scale on the skin rough coat dry with excessive keratinization. Emaciation, disturbances in reproductive efficiency in both male and female. Nervous symptoms (paralysis of skeletal muscle, ecephalopathy and blindness). Edema, enteritis and otitis media are common signs of vitamin A deficiency.

4. Treatment

R/ Vitamin AD_3E, Cattle 10 cc & Sheep & Goat 5 cc I/M.

R/ Code liver oil 15-60 cc / Horse, Cattle 4-15 cc / Sheep and 1-8 cc / Dog and Cat.

5. Important Notes

1. Other AD_3E, vitamins can be used such as VITA-JECT, calves 7-10 ml & I/M Lamb 3-5 ml, S/C & I/M or Multivitamin cattle & 20 - 30 cc & Sheep & goat 5 - 10 cc I/M
2. Daily requirement 30 IU/kg Bwt. of vitamin A or 75 IU carotene/kg Bwt. (increase to 50% in pregnant animals and rapidly growing animals). Injection intraruminal give good results.
3. Green fodder and colostrum are rich in vitamin A.

Vitamin E deficiency

1. Definition and Causes

Deficiency of vitamin E occurs when the animals are fed on poor hay or straw, oxidation during rancidification of the oils causes the destruction of vitamin E. The presence of myopathic agents in the oil may also cause deficiency. Unsaturated fatty acid in fish and vegetable oils appear to be important myopathic agent in many outbreaks of enzootic muscular dystrophy.

2. Pathogenesis

Degeneration of skeletal muscle & diaphragm ⟶ dyspnea.
Degeneration of heart muscle ⟶ congestive heart failure.
Acute degeneration ⟶ liberation of myoglobin in the blood ⟶ myoglobinuria.
Selenium has an important role in transportation and retention of vitamin E

3. Clinical Findings

*Subacute form: Stiffness, weakness and trembling of the limbs. Inability to stand. Rotatory movement of hocks of calves. Muscles are hard, rubbery, often swollen and atrophied. Dyspnea and inability to move for eating and death occurs from starvation.

*Acute form: Sudden onset of dullness, respiratory distress, frothy blood stained nasal discharge, increase heart rate and irregular heart beat. Death occurs 6 - 12 hours.

4. Treatment

R/ Viteselen, Calves, Sheep & Goat 1-5 ml, I/M, as a source of vitamin E and selenium

R/ Vitamin AD_3E, Cattle 10 cc & Sheep & Goat 5 cc I/M.

5. Important Notes

1. Non inflammatory bilateral hyaline degeneration of skeletal muscle and / or myocardium in post mortal lesion.
2. Prophylaxis administration 25-mg sodium selenite and 250 mg alpha Tocopherol Acetate I/M injection to the pregnant cows at 6 Th. month of pregnancy.
3. Giving new born calves 2 mg of Sodium Selenite and 100 mg Alpha Tocopherol Acetate.
4. Wheat Germ Oil 10 cc / Calves and 2 cc / Lamb, orally.

Vitamin B deficiency

1. Aueurine - Thiamin (Vitamin B_1)

1. Sources

It is available in most plants, yeast and wheat germs. Ruminal bacteria can synthesize it. Milk, meat, egg and fish are the main sources for carnivores.

2. Clinical Findings

Muscular incoordination especially in the hind legs. Opisthotonus, (paralysis with head turned over the back) convulsion. Vomiting and diarrhea.

2. Riboflavin (Vitamin B_2

1. Sources

It has a direct effect on the metabolism of carbohydrate, amino acids and aldehyde. Deficiency is confined to a simple stomach animals and pre-ruminating ruminants.

2. Clinical Findings

Decrease in growth rate, anemia, eye discharge and infertility.

3. Pyridoxine hydrochloride (Vitamin B_6)

1. Sources

It is found in yeast, kidney, milk, molasses, cereal and wheat by product.

2. Clinical Findings

Dermatitis (hyperkeratosis of the skin of the nose, paws and ears. Muscular weakness, nervous manifestation. In dogs microcytic and hypochromic anemia are seen.

4. Nicotinic Acid (Nicotinamid - Niacin)

1. Sources

It is found in the food of animal and plant origins. It is necessary for the synthesis of coenzyme II and I.

2. Clinical Findings: Black tongue in dogs.

5. Cyanocobalamine (Vitamin B_{12})

1. Definition and Causes

Vitamin B12 deficiency occurs in case of cobalt deficiency (microbial synthesis of the vitamin occurs in the rumen of cattle and in the intestine of the horse in the presence of adequate cobalt).

2. Clinical Findings

Anorexia, cessation of the growth, loss of the condition and muscular weakness.

General treatment

R/ Tri B, 1 ampoule / 70 kg Bwt. I/M, Trivarol or Trivacid

Vitamin k deficiency

1. Definition and Causes

Vitamin k is essential in the formation of prothrombin by the liver (prothrombin is essential in the clotting of the blood). Vitamin k deficiency is rare in the domestic animals because of the high content in most plants and the synthesis of the vitamin by microbial activity in the alimentary tract.

Absorption of vitamin k from the intestine is depend on the presence of bile and fat in the intestine. Storage is mainly in the liver and excretion is via urinary tract.

2. Treatment

R/ Amri-K ampoule 1 / 70 kg Bwt, I/M injection.
or VITAK 20 g daily / week

3. Important Notes

Therapeutic uses of vitamin K in epistaxis, coccidiosis, abomasal ulcers, sweat clover poisoning, hepatitis and gastro-enteritis.

Vitamin C deficiency

1. Definition and Causes

Ascorbic acid (vitamin C) acts as co-enzymes in certain oxidative process (tyrosine and finyl alanine). It is necessary for normal folic acid function and normal healing. It plays a role in treatment of infertility and it is important in detoxification of toxins and chemicals (Arsenic, Sulphonilamine, and Salysilates).

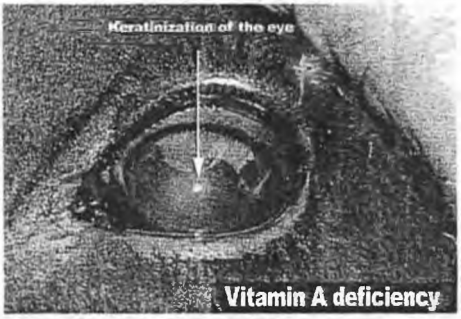
2. Treatment

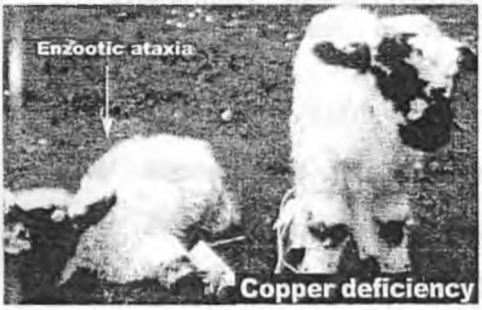
R/ Cevarol ampoule 1 / 70 kg BW I/V or I/M injection.
or VITAC 20 g daily / week

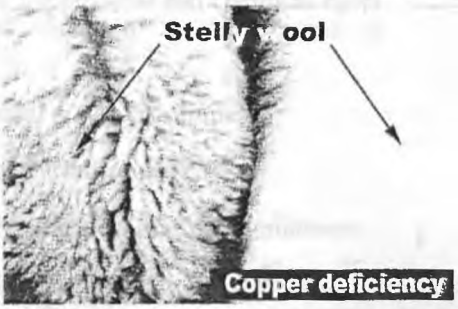
3. Important Notes

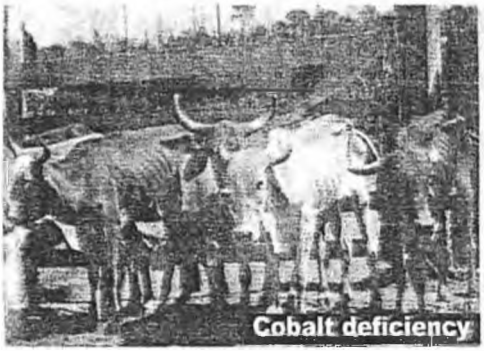
Therapeutic uses of vitamin C in respiratory affection, viral affection, toxicity, wound healing, some cases of infertility in cattle, indigestion and diarrhea in horses.

Plate 9 Deficiency diseases









Dermatitis

1. Definition and Causes

Inflammation of the dermis and epidermis. It is caused by bacteria (actinomycotic dermatitis), viral (pox), fungal (sporotrichosis of horse), physical agent (sunburn, excessive heat or excessive cold and trauma), chemical (irritant chemical), allergic and nutritional deficiency (vitamin B).

2. Pathogenesis

Inflammation of the deepest layer of the skin involving the blood vessels and lymphatic which lead to increase the thickness of the skin. Increase temperature of the inflammed parts. Pain or itching and erythema in the unpigmented area of the skin

3. Clinical Findings

The affected area shows erythema, vesicular lesion and edema of the skin. The next stage may be the healing stage (scab formation) or necrosis and gangrene of the affected area. Systemic reaction when the affected area are extensive. Shock and toxemia may be present.

4. Treatment

Treat the primary cause and remove off the physical and chemical agent from the environment. In case of infection sensitivity test is recommended.

R/ Garamycin or Teramycin as a local antibiotic ointment.

R/ Betamethzone as a local corticosteroidc ointment.

R/ Canastin, Dermatin or Teniacure as a local antifungal ointment.

R/ Zinc Oxide 10% as a local emollient ointment.

R/ Salicylic Acid 3% , as a local keratolytic ointment

R/ Avil 1 ampoule/70 kg Bwt, as antihistaminic drugs

R/ GENTA 50 (Gentamycin sulfate), 8 ml / 100 kg Bwt. I/M & I/V.

4. Important Notes

1. Antibiotic, anti-inflamatory and antifungal ointment as Kenacomb.

2. Dusting powder as mixture of 2g Zinc Oxide, 5g Tannic Acid and 20 g starch.

Eczyma

1. Definition and Causes
It is moist catarrhal inflammation of the skin. It is caused by either exogenous allergens (external parasites, some soup & some antiseptic washes) or endogenous allergens (ingested protein, autointoxication due to overfeeding or constipation and/or internal parasites) and / or vitamin A deficiency.

2.Pathogenesis
Erythema ⟶ intercellular edema ⟶ small vesicle ⟶
Rupture of the vesicle and scab formation.

3. Clinical Findings
Patches of erythema, followed by appearance of small vesicles, which rupture and cause weeping of the surface. Scab formation follows. Lesions may isolate or diffuse over large areas. Itching and irritation. Chronic eczema may follow an acute attack. Alopecia due to scratching and rupping of the skin.

4. Treatment: Treat the primary cause.

R/ Zinc Oxide 10% as a local emollient ointment.

R/ Salicylic Acid 3% , as a local keratolytic ointment

R/ Calcium borogluconate 25%, Horse and Cow 100 - 200 cc, I/V.

R/ Predef 2x 10 cc I/M- 2 days. or Finadyne 1 ml / 45 kg Bwt. I

R/ Lin seed oil 1/2 liter for large animal in case of constipation.

4. Important Notes

1. Antibiotic, anti-inflamatory and antifungal ointment as Kenacomb.
2. Dusting powder as mixture
   2g Zinc Oxide, 5g Tannic Acid and 20 g starch.
3. Enema with soft soap and worm water

Urticharia

1. Definition and Causes

It is a type of hypersensitivity (Nettle Rash) due to antigen antibody reaction results in release of histamine. It is caused by infection such as strangles & dourine in horse, distemper in dog. External toxicity such as mechanical irritant as bits of insects, chemical and medication as carbolic acid and turpentine oil. Internal toxicity administration of some hormones, antibiotics, foreign protein, serum and various bacterial product as mallen and tuberculin. Internal parasites and ingestion of mouldy food may result in Urticharia.

2. Clinical Findings

Clinical signs develop rapidly (within few minutes) and may proceed by general disturbances as loss of appetite, depression and fever.
Cutanious lesions are firm, flat-topped or convex wheels of various sizes. Erected hair & swelling of the affected parts. Lesions may be present in the mucous membrane of the mouth, nose, conjunctiva, rectum and vagina. Sometimes papules and vesicles develop in the surface.
Urticharia due to infection is usually associated with fever, edema of the extremities and head.

3. Treatment

Treat the primary cause.

R/ Finadyne 1 ml / 45 kg Bwt. I/V for 3-5 days.
as antiinflammatory and antihistaminic.

R/ Calcium borogluconate 25%, Horse and Cow 100 - 200 cc, I/V.

R/ Cevarol and Tri B, 1 ampoule / 70 kg Bwt.

4. Important Notes

Allergic dermatitis:

It is various generalized or localized cutaneous due to hormonal imbalances, hepatic dysfunction, inadequate nutrition, seasonal and climatic factors. Hereditary is another factor in the development of allergy. It is characterized by swelling and redness of superficial layer of the skin and pruritis. Treatment as uricharia.

Photosynsetization

1. Definition and Causes

It is irritation of the skin of some animals due to exposure to direct sun light. It is caused by feeding of some photodynamic plants (toxic or green plants) containing agents which when ingested deposited in the skin and activated by sunlight.

2. Pathogenesis

The lesion mainly occurs in the unpigmented area of the skin especially in the dorsal parts of the body. Most photosensitizing substances including phylloerythrin (the normal breakdown product of the chlorophyll in the alimentary tract) is excreted in the bile. In hepatic or biliary insufficiency, excretion of these substances is retarded and photosensitization occurs.The penetration of light rays to sensitized tissues causes the liberation of histamine, local cell death and tissue edema. Nervous signs may occur.

3. Clinical Findings

Skin lesion shows characteristic distribution and usually around the eyelids, ears, muscles, face, laterals aspect of the testis, vulva and perineum.

The first sign is erythema followed by edema. Irritation is intense and the animal rubs the affected part. Dyspnea due nasal obstruction. Dysphasia due to swelling of the lips. Rise in the temperature. Nervous manifestation, posterior paralysis and blindness.

4. Treatment

Immediately removal from sun light and prevention of ingestion of further toxic plants

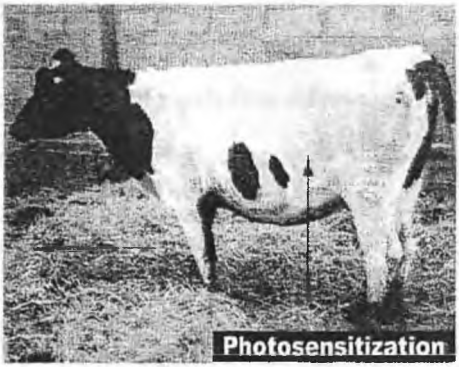
R/ Garamycin or Teramycin as a local antibiotic ointment.
R/ Betamethzone as a local corticosteroidc ointment.
R/ Canastin, Dermatin or Teniacure as a local antifungal ointment.
R/ Zinc Oxide 10% as a local emollient ointment.
R/ Salicylic Acid 3% , as a local keratolytic ointment
R/ Avil 1 ampoule/70 kg Bwt, as antihistaminic drugs
R/ GENTA 50 (Gentamycin sulfate), 8 ml / 100 kg Bwt. I/M & I/V.
R/ Lin seed oil 1/2 liter for large animal.

4. Important Notes

1. Antibiotic, anti-inflamatory and antifungal ointment as Kenacomb.
2. Dusting powder as mixture of 2g Zinc Oxide, 5g Tannic Acid and 20 g starch.
3. Enema with soft soap and worm water

Plate 10 Skin diseases


Alopecia


Photosensitization


Alopecia


Urticaria


Eczema


Facial eczema


Ulcerative dermatitis

Alopecia

1. Definition and causes

It is a loss of hair or wool coat. It is a manifestation of much skin disease (dermatitis, eczema, scabs, and mange).

*Congenital alopecia: It may be localized or generalized, temporary or permanent. Hereditary factors plays an important part in etiology.

*Acquired alopecia: it is usually associated with severe systemic disorders as chronic wasting gastro-enteritis or verminous bronchitis. May occur with certain infectious diseases (strangles, distemper, influenza, pathologic disorder of the genital organ result in certain endocrine imbalances especially in dogs)

2. Pathogenesis and Clinical Findings

The first lesions are edema of the prickle cell layer, dilatation of the intracellular lymphatic and leucocytic infiltration. Imperfect keratinization follows. The lesion is usually confined to the flexure aspect of the joint. Thickening of the skin (gray coloration) scales, cracks and fissure and removal of the scale leaves arrow red surface.

3. Treatment

Washing the lesion with soapy water followed by the application of an astringent preparation

R/ Salicylic acid 3%. As keratolytic ointment.

R/ Multivitamin
cattle & 20 - 30 cc & Sheep & goat 5 - 10 cc I/M.

R/ Supermach
1 sachet /cow orally, daily as a source of vitamin and trace element..

R/ Mineral mixture 50 g / daily / cattle.

4. Important Notes

1. Metabolic disorder, endocrine disturbances, vitamin or dietary deficiencies should be corrected.

2. In gonadal disorders castration or the administration of gonadal hormones is effective.

Hyperthermia

1. Definition and Causes

It is an elevation of body temperature due to excess heat production or absoption of heat from high envirnment temperature and/or deficient heat loss.

2. Pathogenesis

Heat stroke will cause vasodilatation of the cranial vessle, results in drop in blood pressure. Increases in heart and respiratory rates. The temperature is elevated. The urine secretion is reduced. Depression of nervous system activity and depression of respiratory center, usually causes death by respiratory failure. Circulatory failure also occurs due to myocardial weakness.

3. Clinical Findings

Rise in the body temperature over 39 °C. The stops work and refuse to continue. Staggering gait and the animal falls to the ground unconcious. Mucous membranes are congested, irregular and slow pulse. Abortion may occur if the period of hyperthermia is prolonged and high incidence of embryonic mortality. Convulsion are evident and the animal dies in a state of coma within 2 hours.

4. Treatment

Cold application: Including immersion, spraying, rectal enemas or cold packs. Put the animal in well ventillated place together with adequet drinking water.

R/ Saline, dextrose 5% or ringer lactate 1-2 liters I/V.

R/ Novalgen or Analgen or Novacid 25 cc I/V. As antipyretic drugs.
or Acetyle Salysilic acid as Asprine or Aspegic ampoules.

5. Important Notes

1. Antipyretic drugs is of no value when the temperature is over 41 °C in cattle, sheep and horse and over 40 °C in camel. Firstly try to reduce the temperature by using of cold application until reach 40 °C then you can useantipyretic drugs.
2. In cases of fever due to bacterial, viral and/or blood parasites, broad spectrum antibiotic, anti-inflammatory and anti-blood parasites drugs are recommended.
3. **Hypothermia:** Means decrease in the body temperature. It is caused by decrease of muscle tone as in hypocalcemia and acute ruminal impaction and during anathesia and sedation, associated with profuse diarrhea, shock, hemorrhage, anemia and before death. Dealing with such cases by worming the animal and injection of calcium preparation and glucose 25% I/V.

Dehydration

1. Definition and Causes

It is loss of body fluid. It is caused by failure of water intake or excessive loss of fluid due to diarrhea, vomiting, polyurea, skin wounds or by copious sweating. Severe dehydration also occurs in acute impaction, acute intestinal obstruction, abomasal dilatation and torsion and diffuse peritonitis.

2. Clinical Findings

Dryness of muzzle, oral cavity, cornea and skin.Eyeball is sunken and received in to sockets. Skin be ome wrinkled and loss its elasticity. Emaciation, weakness and severe loss of body weight. Anorexia, severe thirst, decreases in gastrointestinal motility, indigestion and constipation. Bradycardia and cardiac arrhythmia. Oliguria or anuria. Coldness of extremities, subnormal temperature, recumbency, coma, circulatory failure and death.

4. Treatment

R/ Electrolytes or Super-Lyte or Vit-Lyte or Rehydran. 100 g/calves or lambs, dissolve in 2 liers of water or milk. As Oral electrolye

R/ Saline, dextrose 5% or ringer lactate or blood transfusion 1-2 liters I/V or I/P.

R/ Sodium Bicarbonate 1.3% for mild acidosis. 1-2 liters slowly I/V or 3 - 5% 5 ml / kg Bwt / for severe acidemia.

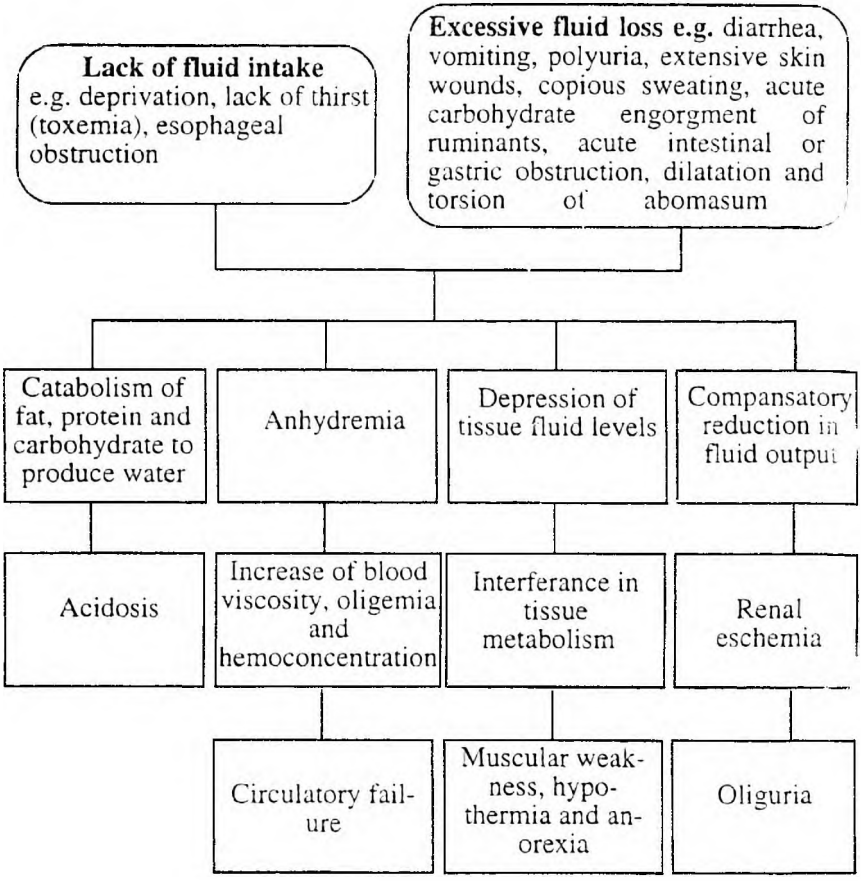
5. Important Notes

1. Sodium, chloride and bicarbonate are found extracellular, while potassium is found intracellular. Loss of fluid starts in the intravascular space then interstitial and intracellular fluid.

2. Hypertonic saline solution such as Sodium Chloride 7.5% (5 ml / kg Bwt) injection is continued by Ringer Solution 0.9 Sodium Chloride facilitate intracellular rehydration. It is used in serious cases as in hemorrhage or circulatory shock.

3. Isotonic Solution as Saline Solution (0.9 % Sodium Chloride), Ringer Solution (Na cl, k cl & ca cl) & Ringer Lactate Solution.

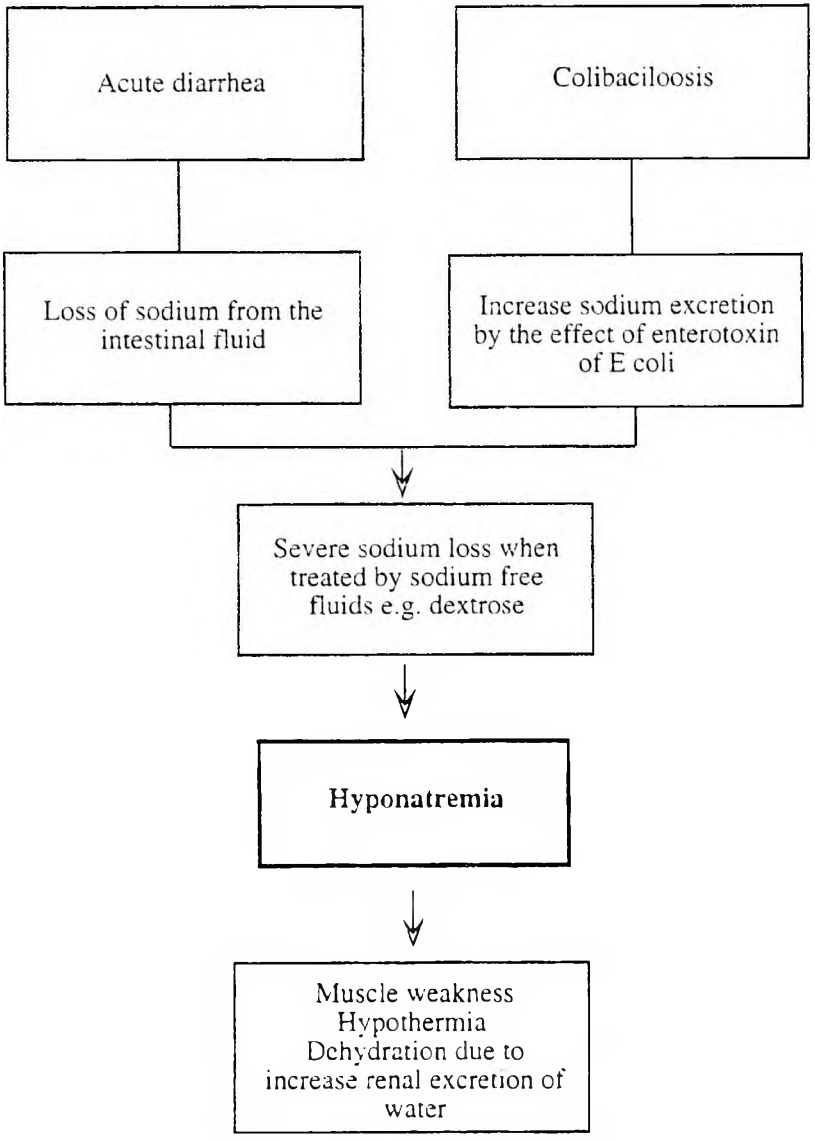
Etiology and pathogenesis of dehydration



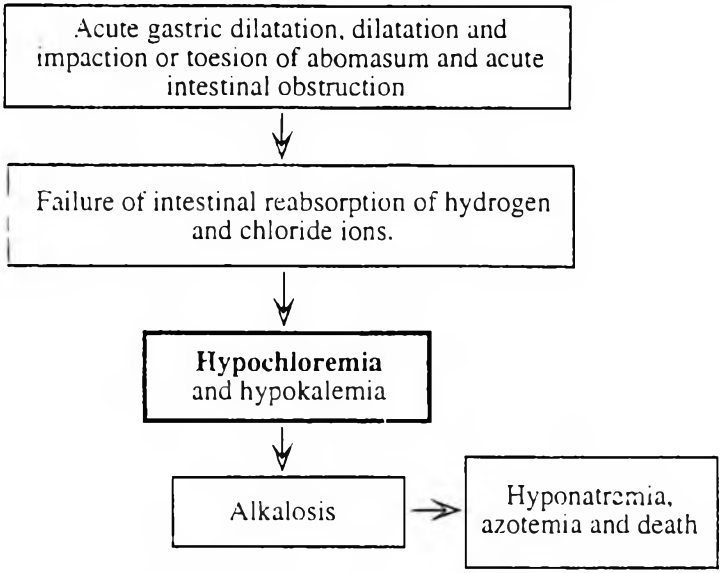
Degree of severity of dehydration and treatment

Body weight loss %	Sunken eyes shrunken face	Skin fold test persists for sec	PCV %	Fluid required ml/kg Bwt.
4-6	+	-	45	20-25
6-8	++	2-4	50	30-50
8-10	+++	6-12	55	50-80
10-12	++++	20-45	60	80-120

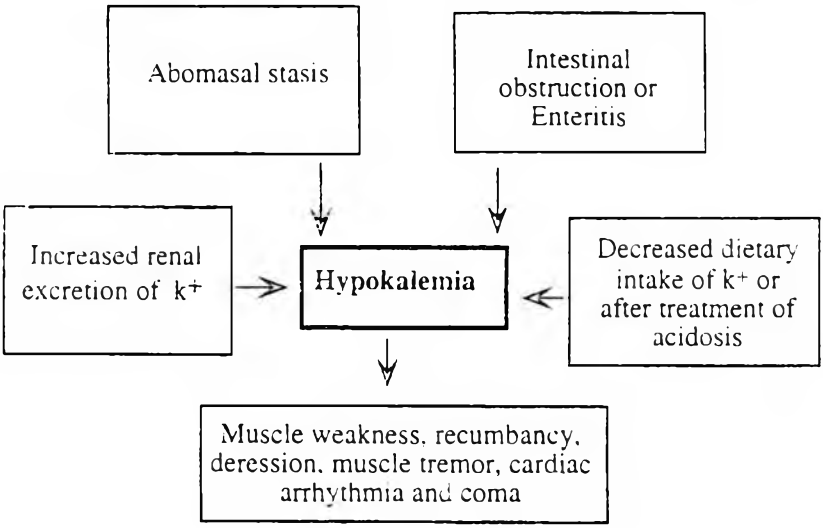
Etiology and pathogenesis of hyponatremia



Etiology and pathogenesis of hypochloremia



Etiology and pathogenesis of hypokalemia



Key to differential diagnosis of diseases of farm animals

1- Diseases causing sudden death
In cases of Hypocalcemia, Hypomagnesemia, Acute Pneumonia, Peracute Mastitis, Poisonous, Heavy Worm Infestation, Enterotoxemia, Salmonellosis, Tetanus, Snake Bite, Calculi, Acute Liver Fluke, Lamb Dysentery, Sun Stroke, Anthrax, Malignant Edema, Itussuception.

2- Diseases causing sudden death in young animal
In cases of Enzootic ataxia, Colibacillosis, Vitamin A Deficiency, Brucellosis, Toxopalsmosis, Arthritis, Enterotoxemia, Feeding troubles, Navel Ill, Pneumonia, Coccidiosis, Jaundice, Cobalt & Copper deficiency, Necrobacillousis, Injury at Birth & Genetic factors.

3- Diseases causing lameness, Stagger and / or Paralysis
In cases of Enzootic ataxia, arthritis, foot rot, foot abscess, Injuries, Fracture, wound, laminitis, improper shoeing, sprain, sole abscess.
Acute Mastitis, Black Leg, Listeriosis, Foot and Mouth Diseases Post-vaccine Lameness, Tetanus & Blue Tongue.
White Muscle Disease, Rickets, Calcium Deficiency, Grass Tetany, C.N.S. Diseases, Vitamin A Deficiency, Copper Deficiency, Pregnant Toxemia & Plant poisoning.

4- Diseases causing convulsion
In cases of Hypomagnesemia, Hypoglycemia, Milk Fever and Vitamin A & Copper Deficiency. Poisoning, Urea Poisoning, Spinal Abscess, Meningitis, Sinusitis & Trauma in C.N.S. Colibacillosis, Tetanus, Enzootic Ataxia, IBR, IPV, MHCF, Coenurosis, Listeriosis Babesiosis.

5- Diseases causing wasting (emaciation)
In cases of abomasal impaction or displacement, copper, selenium, and/or phosphorus deficiency, malnutrition, foreign body, tumers, tick and/or lice infestation and chronic diseases such as Tuberculosis and Johnes diseases.

6 - Diseases causing fever
In cases of pneumonia, pleurisy, enteritis, pyelonephritis, acute mastitis, metritisand septic infection. Bacterial, viral diseases and blood parasites diseases.

7- Disease problems arising from intensive managment of cattle
Rumen acidosis, laminitis, urolithiasis, tympany, liver abscess, Avitaminosis A, Avitaminosis E, hypomagnesemia, hypocalcemia and ketosis.

8- Diseases causing polyphagia (increase in food intake)
In cases of starvation, internal parasites, functional diarrhea, chronic gastritis, diabetes mellitus, hyperthyrodism and abnormalities of digestion particulary pancreatic deficiency.

9- Diseases causing anophagia (poor appetite or decrease food intake)
In cases of stomatitis, pharygitis and hyperthermia. Thiamine, cobalt and zinc deficiency. Heavy infestation with trichostrongyloid helminth. Some sheep which have been at pasture become completly anophagic if housed.

10- Diseases causing pica (ingestion of material other than food)
In cases of salt, cobalt and/or phosphorus deficiency. Chromic abdominal pain due to peritonitis or gastritis. Rabies and nervous form of ketosis.

11- Diseases causing weight loss or failure to gain weight
Malnutrition due to trace element deficiency, falty absorption and digestion, excessive loss of protein and carbohydrate, congestive heart failure, chronic diseases (Trypanosomiasis, Enzootic pneumonia, chronic peritonitis and parasitic infestation).

12- Diseases causing scouring
In cases of GIT Nematodes & Coccidiosis, Liver Abscess and Cancer. Rota and Corona virus. Colibacillosis, Salmonellosis, Enterotoxemia & Lamb Dysentery. Feeding Troubles (milk replacer or concentrate), Poisoning, Mineral Deficiency and Imbalances & Vitamin A Deficiency.

13- Diseases causing vomiting
In cases of diseases of brain and drugs causing central vomiting action (apomorphine), plant poisoning or other poisoning or autointoxication. Gastritis or over eating in dogs. Obstruction of the pylorus (Gastrophilus larvae) or small intestine. Involvement of organs such as the kidneys, liver, uterus and pancreas.

14- Diseases causing diarrhea
In cases of GIT Nematodes, Fascioliasis, Paramphistomiasis & Coccidiosis,
Salmonellosis, johne's disease, BVD, MHCF, Enterotoxemia & rinder pest. Enteritis, indigestion (spoiled feed, overfeeding, or sudden change), abomasum displacement or torsion, peritonitis, copper deficiency, heart failure, uremia, renal failure, overdose of rompone, laxative, parasympathomimitics, toxic plant, toxicity by (arsenic, sulfur, salt, zinc, copper, levamisol).Traumatic reticulitis, Vegal indigestion, Liver Abscess, vitamin A deficiency, selenium deficiency, zinc deficiency & water intoxication.

15- Diseases causing abdominal pain in horse
In cases of intestinal tympany, intestinal obstruction, intestinal muscle spasm (cramps), intestinal impaction, colitis, colonic displacement, colonic volvolus, ileal impaction, intestinal foreign body (sand), renal colic, parasympathomimitic drugs, peritonitis, small intestinal strangulation obstruction, uterine torsion, ascarid impaction, gastric dilatation, hernias (diaphragmatic or umbilical), intussusception, plant poisoning urolithiasis & tetanus.

16- Diseases causing abdominal pain in ruminant
In cases of rumen impaction, ruminitis, liver abscess, tympany, traumatic reticulo-peritonitis, vagus indigestion, Abomasal ulcer, Abomasal displacement, Abomasal impaction.Urolithiasis, cystitis, pyelonephritis & uterine torsion.

17- Diseases causing jaundice
In cases of copper poisoning, Photosensitization, Hepatitis, Plant poisoning, Toxemic Jaundice, Phosphorus Poisoning, Leptospirosis, Nitrite poisoning, Jaundice in Newborn Lambs and Salmonella aborts infection.

18- Diseases causing pain on urination
In cases of urolithiasis, urethritis, cystitis, rupture bladder, bladder calculus, vaginitis, prolapsed prepuce, perpetual injuries or infection & Pyelonephritis in cattle.

19- Diseases causing albuminuria
In cases of cystitis, glomerulonephritis, renal infarction, and inflammation of genital organs and poisoning (lead, arsenic, and mercury...).

20- Diseases causing red or brown urine
Hematuria: In cases of trauma of the kidney, Anthrax, acute glomerulonephritis, cystitis, urolithiasis, rough manipulation of the catheter, tumor of renal tract and also hemorrhage of genital tract
Diseases causing **hemoglobinuria:** In cases of Babesiosis, Bacillary Hburia, Water intoxication, Leptospirosis, chronic copper poisoning and hypophosphatemia.
Myoglobinuria: In case of Azouturia

21- Diseases causing muffled heart sound
In cases of traumatic pericarditis, chronic heart failure, emphysema, pneumothorax, abscess, obesity, large or thick chest wall & tumor in the chest.
Jugular venous distension or pulsation. Pericarditis, right heart failure, chronic heart failure, tricuspid insufficiency, jugular venous phlebitis or thrombosis white muscle disease, Brisket disease and overhydration.

22- Diseases causing cough
In cases of pharyngitis, Laryngitis, Bronchitis, Emphysema & Pleurisy, Pneumonia (Bacteria, Virus, Parasitic, Drenching Traumatic, Abscess). Choke & Vitamin A Deficiency. IBR, IPV, MHCF & MD.

23- Diseases causing nasal discharge:
In cases of laryngitis, lung worms, nasal Bot, pneumonia, drenching pneumonia, snuffles, Pasteurellosis, dusty yard & Blue tongue.

24- Diseases causing eye discharge:
In cases of foreign bodies, IBR, BMCF, Pink Eye, rinder pest & thieleria.
Photosensitization, hyperkeratosis & vitamin A deficiency.

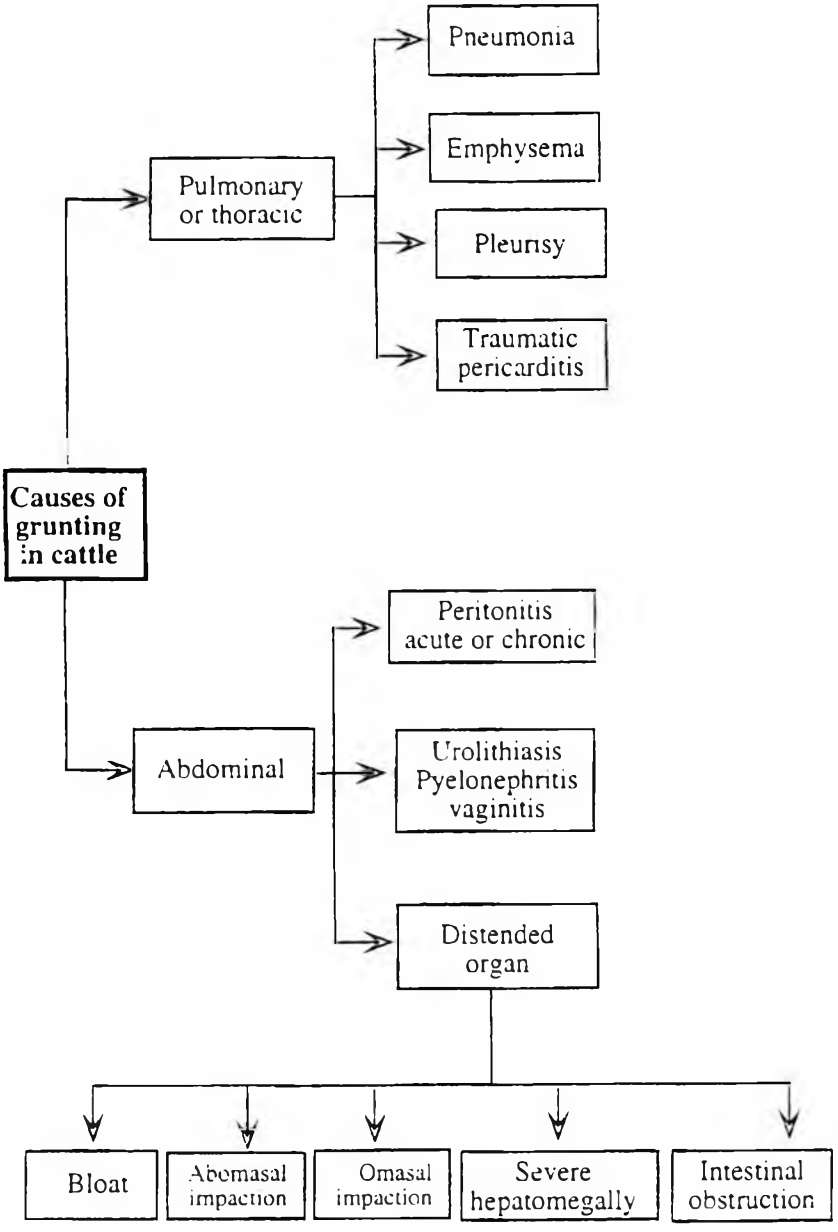
25- Diseases causing chest pain in ruminant
In cases of pneumonia, pleura-pneumonia, pleuritis, traumatic reticulo-peritonitis, traumatic pericarditis, thrombosis of caudal vena Cava, acute bovine emphysema, choke, fractured ribs, osteomylitis & mediastinal abscess or tumor.

26- Diseases causing chest pain in horse
In cases of pneumonia, pleura-pneumonia, pleuritis, choke, fractured ribs, osteomylitis, mediastinal abscess or tumor & white muscle disease.

27- Diseases causing skin lesion
In cases of dermatitis, photosensitization, eczema, drug reaction, allergy, mange, ring worm, lice and Tick infestation, hyperkeratosis, articaria, local irritation, and papilloma in cattle.

28- Diseases causing disease problems arising from intensive managment of cattle
In cases of rumen acidosis, laminitis, urolithiasis, tympany, liver abscess, Avitaminosis A, Avitaminosis E, hypomagnesemia, hypocalcemia and ketosis.

29- Diseases causing downer cow syndrome
In cases of hypocalcemia, traumatic injuries of medial thigh muscle, traumatic injuries to the nerves of the limbs, calving paralysis (obturator or sciatic paralysis), pelvic fracture, malnutrition, coxofemoral luxation and lymphosarcoma


Causes of grunting in cattle
Pulmonary or thoracic
Pneumonia
Emphysema
Pleurisy
Traumatic pericarditis
Abdominal
Peritonitis acute or chronic
Urolithiasis Pyelonephritis vaginitis
Distended organ
Bloat
Abomasal impaction
Omasal impaction
Severe hepatomegally
Intestinal obstruction

Clinical Examination and diagnosis

1. History

In animal disease investigation, the history taking has a very significant role because animal cannot speak. So it depends on the skill of a veterinarian, how he takes out information of illness of animal from his owners.

A) Animal data

Veterinarian should include the owner's name and address alongwith species, breed, six, age, name and number of animals.

B) Previous illness

You should record the previous disease of animal, previous treatment, last pregnancy, sudden death, previous vaccination, sudden change of diet.....

C) Present disease

How long the animal has been ill, first sign of the disease, symptoms of the disease, number of affected animal, appetite, type and quantity of food, rumination, defecation, amount of drinking water, urination, amount of milk, posture, locomotion and cough, dyspnea.

D) Observations of the veterinarian

The veterinarian should not wholly depend upon the owner 's complian. He must take his own observation (clinical signs, lesion, and diagnosis).

2. Normal respiration, pulse and temperature

Animal species	Respiration	Pulse	Temperature
Camel	5-12 / minute	30-50 / minute	36 - 38 C
Horse	10-14 / minute	28-40 / minute	37 - 38 C
cattle	10-30 / minute	55-80 / minute	38 - 39 C
sheep and goat	20-30 / minute	70-90 / minute	39 - 40 C
Dog and cat	15-30 / minute	70-90 / minute	38 - 39 C

Site of taking pulse

A) Camel ——→ posterior tibial artery
B) Cattle ——→ ventral coccygeal artery
C) Horse ——→ external maxillary artery
C) Sheep & goat ——→ femoral artery
C) Dog & cat ——→ femoral artery

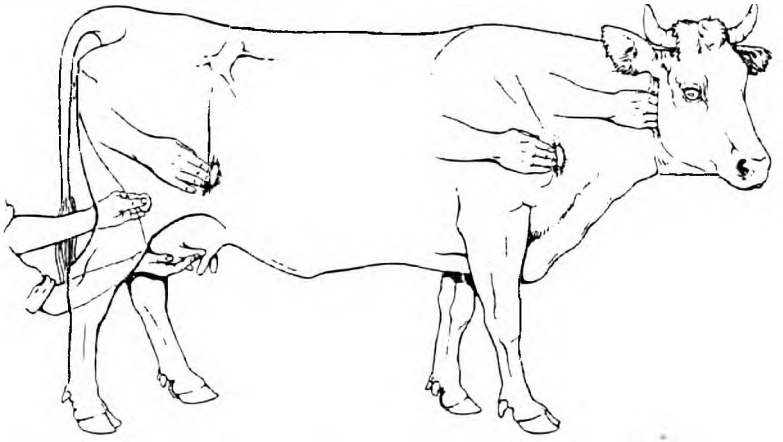
4. Examination of mucous membrane

The mucous membrane examined are conjunctival , nasal, oral & vaginal. The normal color of mucous membrane is rosy red in equines and pale rosy red in ruminants.

Abnormal color of mucous membrane are:

A) Pale m. m. in cases of blood loss, iron deficiency, some parasitic diseases (hemolytic) & wasting diseases.
B) Congested m. m. in cases colic, fever & respiratory diseases.
C) Icteric m. m. in cases of liver diseases, blood parasites & infectious anemia.
D) Cyanosed m. m. in cases of defective oxygenation of the blood & respiratory trouble.

4. Examination of lymph node



Animal species	Name of LN	Site of LN	Enlarged LN
Cattle & sheep	Prescabular	Above shoulder point	TB, Theileriosis, Leukemia, and local affection.
	Prefemoral	Above stifle joint	as above
	Submaxillary	Intermaxillry space	actinomycosis
	suprammary	At the posterior base of udder	mastitis
Horse	Submaxillary	Intermaxillry space	Strangles, glanders and epizootic lymphangitis

5. Examination of the skin

Examination includes condition, surface, elastisity and horn structures. The normal coat is smooth and shiny.

Abnormality in skin coat:

A) Skin lustreless, dry and rough ⟶ Nutritional deficiency.
B) Greasy hair ⟶ Seborrhiec eczema
C) Erection of hair ⟶ Urticaria
D) Loss of hair ⟶ Eczema, dermatitis, mange, ring worm, iodine def. & hyperkeratosis.
E) Alopecia ⟶ Copper def., hypothyrodism selenium & mercury poison.

6. Auscultation of the heart

In all animals the heart lies in lower two thirds of the thoracic, just above the elbow joint (left side).

The heart sounds are classified into two groups:

A) The first sound (systolic sound) is dull, loud and prolonged, arises from cntraction of ventricle, closure of atrio-ventricular valve & and tension of cordae tendinae resemble lubb.

B) Second sound (diastolic sound) is short and sharper. It is due to closure of semilunar valves resemble Dup.

Abnormal heart sound

They may originate in the cavities in the heart or from pericardium.

A) Murmur:

It may systolic or diastolic due to improper closure of atrio-ventricular valve & aortic valve respectivelly.

B) Pericardial sound

It occurs in traumatic pericarditis and classified into 3 stages:

First stage (dry stage):

Frictional sound is heard due to friction between parietal and visceral layer of pericardium.

Second stage (exudative stage):

Dribbling sound is heard when small amount of exudate is formed.

Splashing sound (Tinkling sound) when inflammation go on and exudate increase and sometimes mixed with gases.

Third stage (Muffling stage):

Muffling sound, the exudate usually rich with fibrin and pus due to septic infection and the heart sound is low as it comes from distant place.

7. Auscaltation of the lung.

Most of thoracic cavity area is occupied by the lungs. The area of auscaltation and percussion of the lung is triangular area formed by the points, (a) posterior angle of the scabula, (b) olecranon process of the ulna & (c) second last intercostal space. At a poit on horizontal line from scabula to the external angle of the ilium.

The normal sound by auscaltation: Vesicular sound in lung (resemble V) & bronchial sound (resemble Ch) at larynes and trachea.

Abnormal respiratory sounds:

A. Rales

Dry rales: occur when air is being forced through a bronchial tube which is partially constricted, either by dry tenacious thick exudate or severe swelling of the mucous membrane.

Moist rales: occur when bronchi contain light, thin watery mucous (pus - blood - liquid - exudate) moving from place to place.

Cripitant rales: occur when the opposing walls of bronchial mucosa become adherent to one another and have to be separated by the stream of incoming air.

B. Emphysematous sounds:

Emphysematous sound are harsh and crackling, heared during inspiration. It occurs in pulmonary emphysema & edema.

F) Frictional sounds: Are heared in dry stage of pleuritis

8. Examination of the abdomin.

The abdominal cavity is occupied by the rumen, intestine & associated organs. The abdominal cavity is separated from chest cavity by the diaphragm.

Rumen

Location: The rumen can be examined in the left side (left flank region).

Auscaltation : Normal ruminal movement 2 - 5 / 2 minutes. Increase in cases of vagal indigestion & gastric stenosis. Decrease in ruminal movement and/or stasis in cases of indigestion, severe tympany, rumen acidosis and traumatic reticulo-peritonitis.

Reticulum

Location: The reticulum is located on the left side at the ventral end of 6th or 7th rib separated from the heart by the curve of the diaphragm.

Auscaltation: Reticular movement are heared as a rumbling gurgle. The reticulum normally contract every 40-60 seconds into phases interupted by a period of a pause.

Abomasum

Location: In the abdominal floor on the right side behind the xiphoid cartilage.

Auscaltation: Neither percussion nor auscaltation can be done in the investigation of the abomasum. It is only valuable and diagnostic in the displacement to the left side. Splashing or tinkling sound (more fluid in nature than the rumen) every 15 minutes..

Cecum

Location: The cecum is cone shape can be examined in the right side. Its round base in the right flank & its apex above xiphoid cartilage.

Auscaltation: Normal intestinal movement is peristaltic sound. Increase of intestinal movement in spasmodic colic. Decrease or absence of intestinal movement in flatulent colic and intestinal impaction.

Liver

Location: The liver is situated in concavity of diaphragm. It is on the right side of midian plane & contact with right portion of diaphragm and some of its portion is incontact with last 2-3 ribs. It can be examined by palpation and percussion through the costal arch.

Owner's name: ..... **Date:** .....
Owner 's adress: .....
Owner 's Tel No: .....

Animal species: Age: Sex:

Case History:

Previous illness	Appetite:
Previous treatment	Type of food:
Last pregnancy	Quantity of food:
History of sudden death	Rumination:
Previous vaccination	Defecation:
Sudden change of diet	Drinking water:
Source of disease	Urination:
How long the animal has been ill	Amount of milk:
Number of affected animals	Posture:
First sign of the disease	Locomotion:
Symptoms of the disease	Cough:

Pulse: **Respiration:** **Temperature:**

Mucous membrane Colour Swelling Exudate	**Jugular vein & Eye Capillaries** Abnormal pulsation	**Lymph node** Size consistancy Movability Tenderness	**Skin** Ext. parasites Lesion Dehydration
Heart Rate Rythm Ab. sound	**Lung** Rate Rythm Ab. sound	**Liver** Palpation Percussion Pain	**Rumen** **Caecum** Rate Amplute

Laboratories Examination

Blood Exam	Fecal & Urine Exam	Skin scrab Exam	Serum Exam
Film RBC WBC DLC			

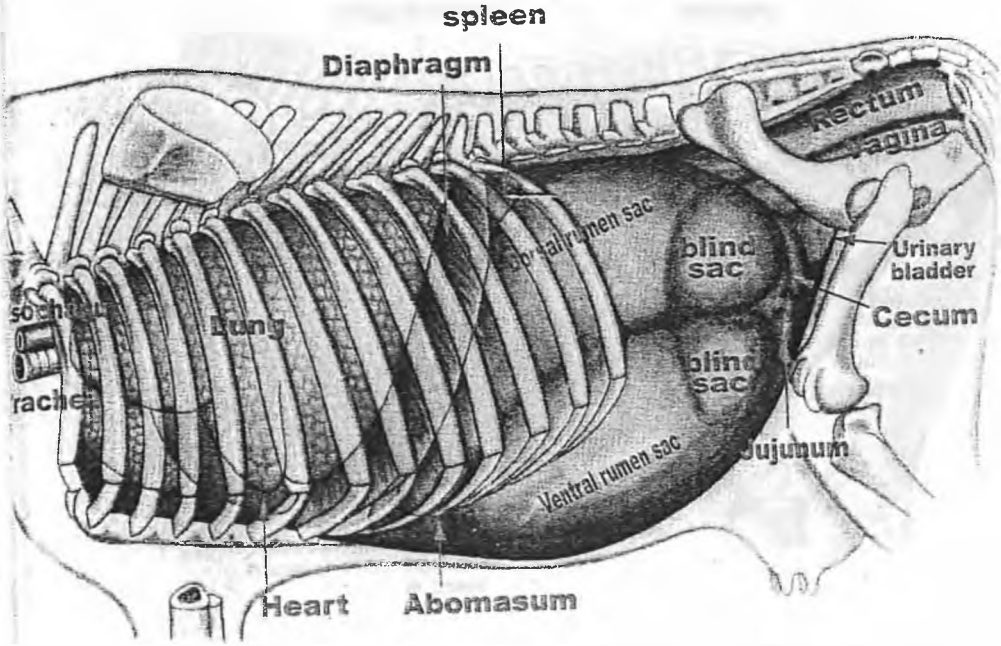
Diagnosis

Treatment

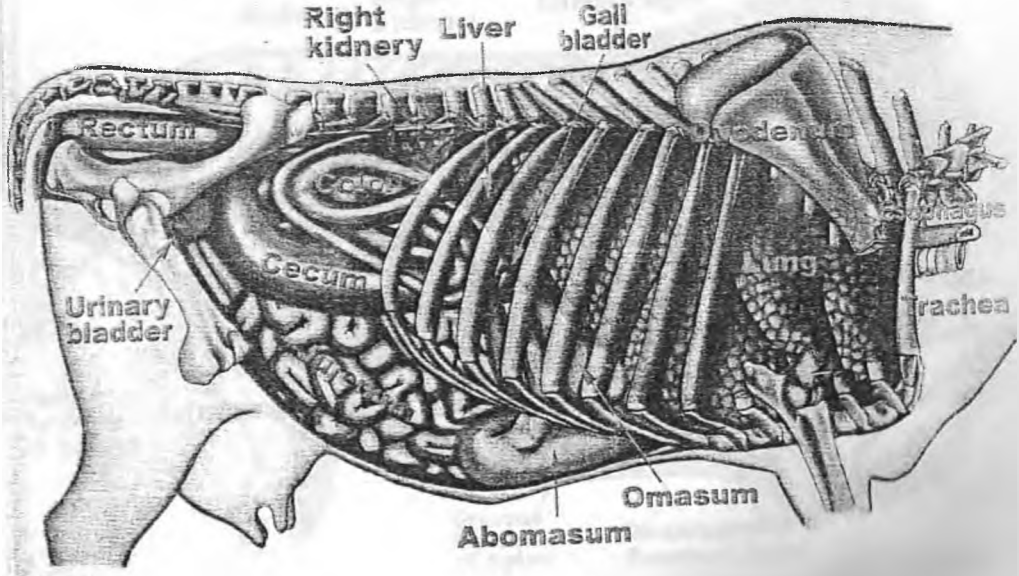
R/

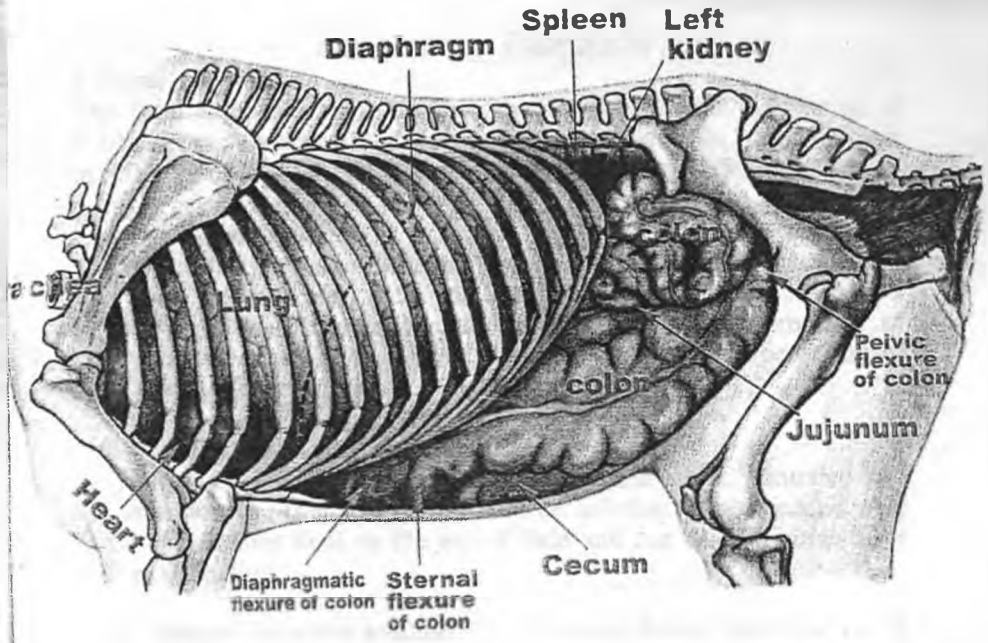
R/

Plate 11 Topographic anatomy of cattle (left side)

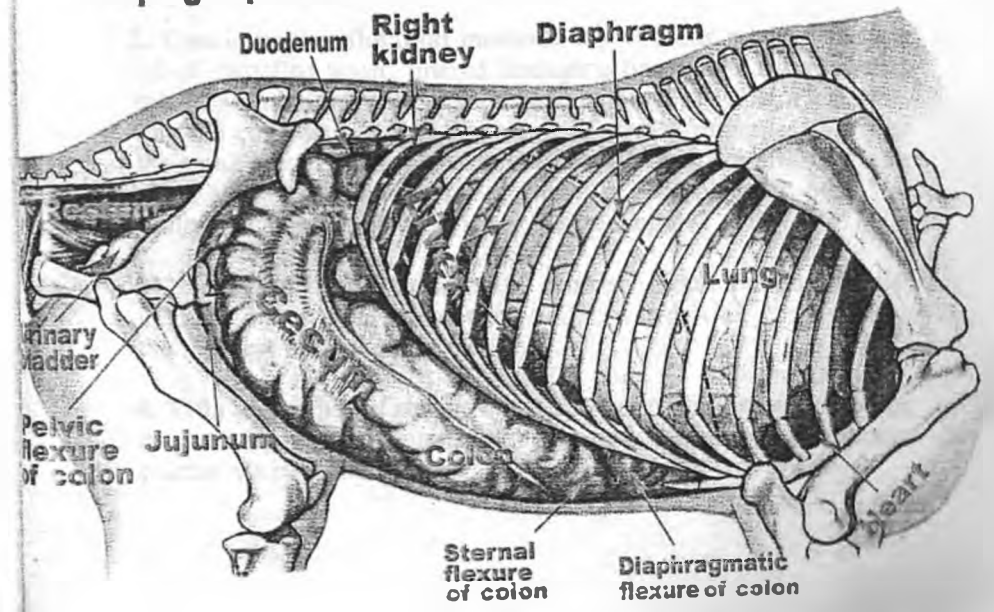


Topographic anatomy of cattle (right side)





Topographic anatomy of horse (right side)



Laboratory Diagnosis

1. Fecal examination

The fecal sample is collected directly from the animal. Collection of 5-10 g feces in a clear dry glass container. In delay exam, store the feces in refrigerator at 4 ^{o}C. The feces can be examined by different methods:

A) Direct method:

A clean dry glass slide is used. Place a drop of distilled water in the middle of the slide, add small amount of feces, mix and place a cover slipe.Examine it under microscope for the presence of parasitic ova. If no parasitic ova is detected it should be examined by qualitative method.

B) Qualitative concentration method:

Feces is mixed with either of the saturated suger, saturated salt solution or 41% magnesium sulfate solution. The parasitic ova, being lighter float on the top of fluid and can be concentrated for examination.

1. Simple flotation method: 1 g of feces mixed with few ml of disitalled water, filtered through a fine sieve. The filtrate is mixed with 4-5 ml of saturated salt solution. It should be then placed in a tube or cylender and filled up to the top with solution, cover the tube with glass slide and left it 30-60 minutes at room temperature. Remove the cover slide and examine under the microscope.

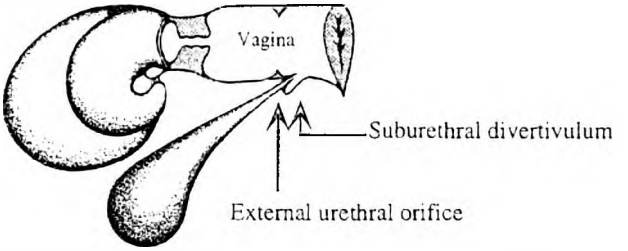
2. Concentration flotation method: 1 g of feces mixed with few ml of disitalled water, filtered through a fine sieve. The filtrate is mixed with saturated suger solution in a ration of 1:3 in a test tube, mix the contents and centrifuge at 1500 rpm / 5 minutes. Transfer the small amount of superficial contents of tube on a clean and dry glass slide and examine for the presence of parasitic ova. The sediment can be examined for eggs of trematodes.

3. Baermen's technique in cattle &horse: Small amount of feces in guaze inside funnle filled with w orm water. After 2 hrs. examin the first few drop to detect the larva

4. Vida technique in sheep: Pellet of feces mix with worm water in petredish for 10 minute then crushed the pellets by forceps examin after 10 minute.

2. Urine examination

Urine samples can be collected in cows and sheep either by stimulation of the urethra through valva or by catheterization. In delay exam, store the urine in refrigerator at 4 °C.



1. Chemical examination:

A. Reaction (pH): The reaction of urine is determined by using pH strips or pH meter. Normal urine is alkaline in cattle and horse (7.4-8.4) and acidic (6-7) in dogs and cats. Acidic urine is abnormally observed in cases of starvation, fever, treatment with sodium acid phosphate, while the alkaline urine is abnormally observed in cases of cystitis, urine retension and treatment by carbonate, acetate and nitrate of sodium or potassium.

B. Glucose: Normally there is no any glucose content in the urine. Glucosuria occurs due to hyperglycemia and in diabetes mellitus, acute or chronic pancreatitis, hyperadrenaline and certain drugs (pencillin, tetracyclene and chloramphenicol). Glucosuria can be detected in the urine by using of Benedict ,s test or urine strips.

C. Protein: The main protein in the urine is albumin which comes under certain disease conditions such as cystitis, glomerulonephritis, renal infarction, inflammation of genital organs and poisoning (lead, arsenic and mercury). It can be detected by using of sulfosalcylic acid test or urine strips test.

D. Ketones bodies: Ketones bodies are acetone, acetoacetic acid and beta hydroxybutyric acid which are formed as a result of breakdown of fatty acids. It abnormally occurs in acetonemia, pregnancy toxemia, fatty degeneration of the liver and abomasal displacement. It can be detected by using of Rother ,s test or urine strips test.

E. Bilirubin: It may be:

1) **Pre-hepatic** (hemolytic):It occurs in case of bacillary hemoglobinuria and leptospirosis, babesiosis, anaplasma, infectious equine anemia, chronic copper poisoning, hypophosphataemia and/or heavy metal poisoning.
2) **Hepatic** It may be toxic, infective or obstructive
3) **Post-hepatic**: Obstruction by calculi or compression by tumor.

Plate 13 Fecal examination of cattle and sheep

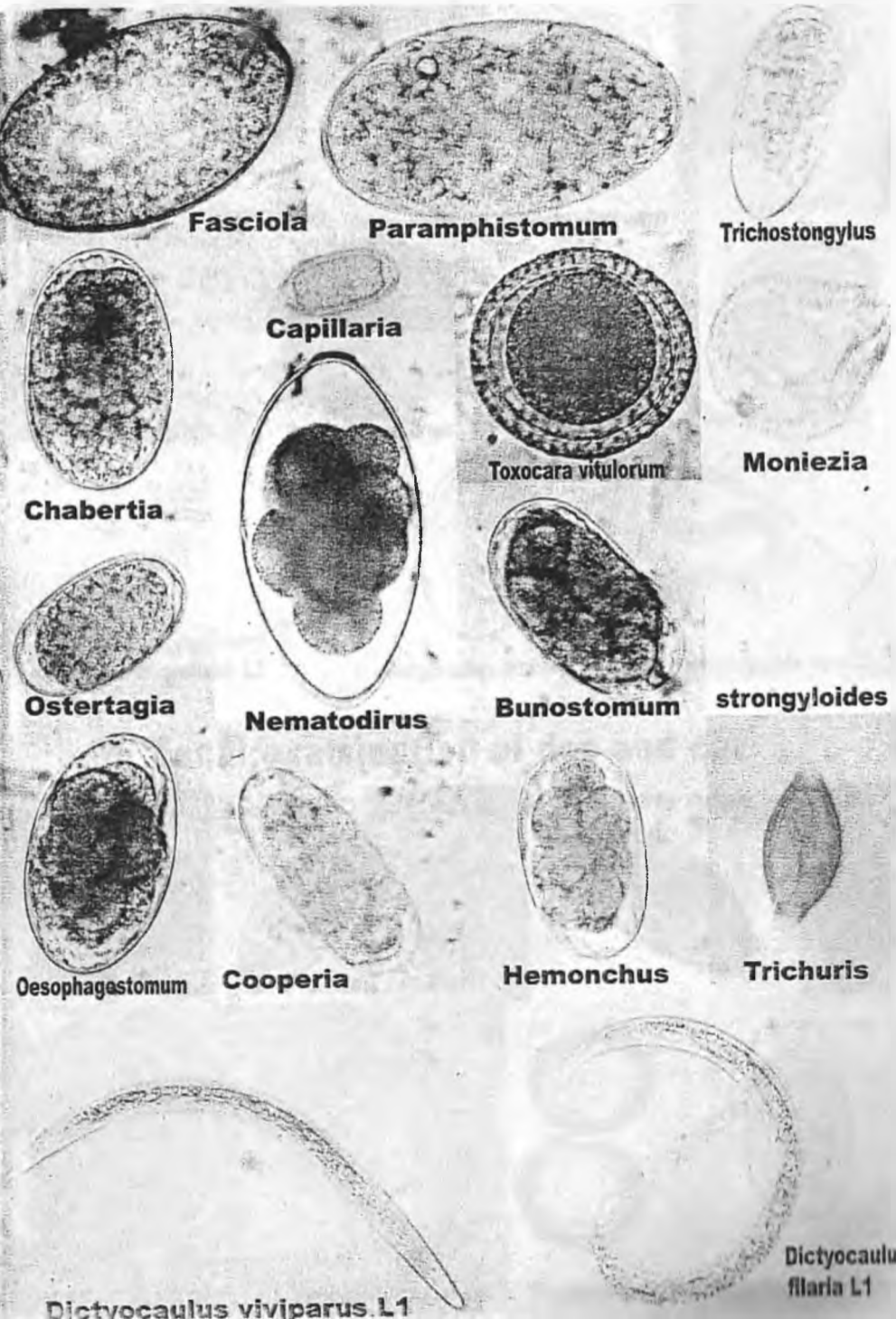
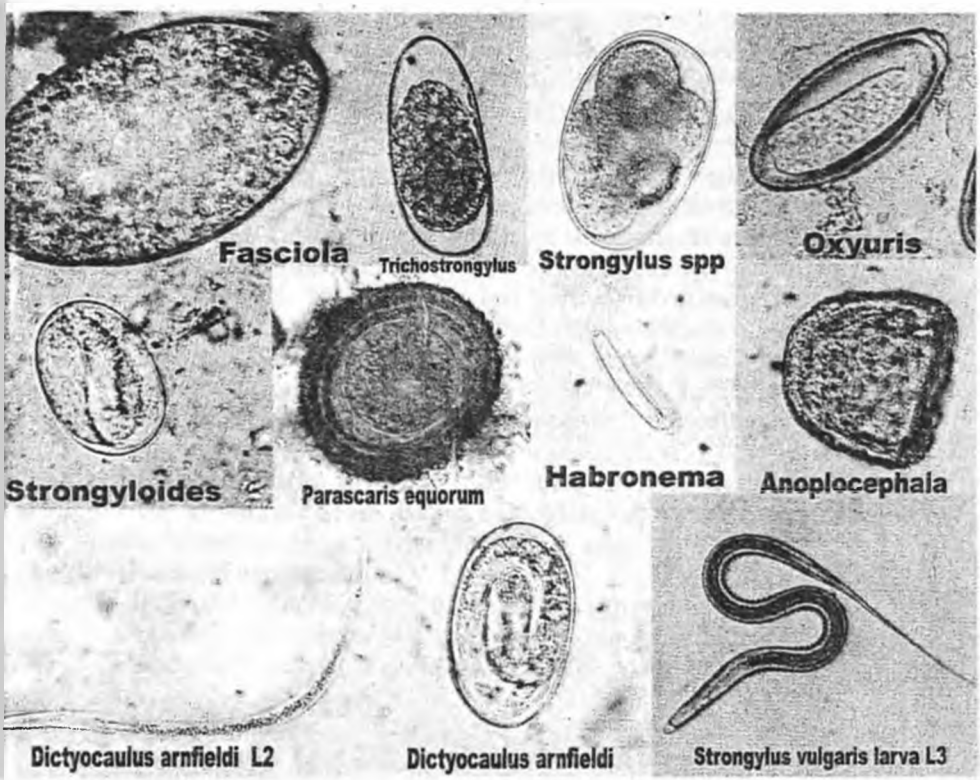
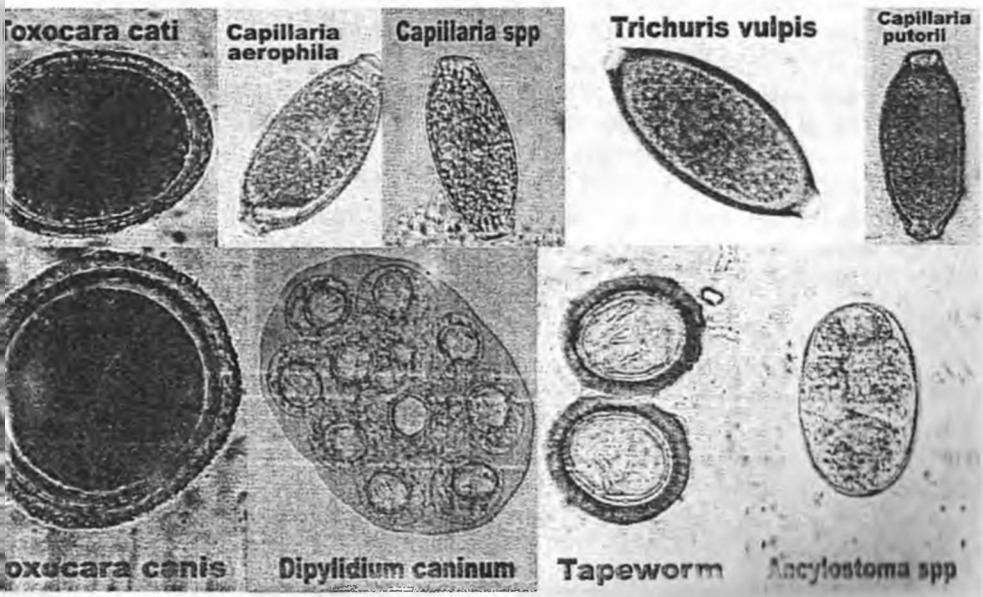


Plate 14 Fecal examination of horse



Fecal examination of dog and cat



F. Blood, hemoglobinuria & myoglobinuria:

1) **Hematuria:** The color is red and clowdy.It is caused by trauma of the kidney, anthrax (pre-renal), acute glomerulonephritis, tubular degeneration(by bacterial toxins and sulfanilamide intoxication (renal) ; cystitis, urolithiasis, rough manipulation of the catheter, tumor of renal tract and also hemorrhage of genital tract (post-renal). If the blood comes during onset of urination, the source of hemorrhage is coming from urethra. If the whole urine is mixed with blood the possible source may be kidneys but if only last portion of urine is red and containing blood , it will be come from the bladder.
2) **Hemoglobinuria:** The color is brown to red, caused by water intoxication, babesiosis, bacillary hemoglonburin, leptospirosis, chronic copper poisoning and hypophosphatemia.
3) **Myoglobinuria:** Brown to black color of urine, occurs in case of Azouturia disease. Blood, hemoglobin & myoglobin can be detected by using of Benzidine test or urine strips test.

2. Microscopical examination

Take 5-10 ml urine in a centrifuge tube and Centrifuge it at 1000 rpm/10 minutes. Discard the supernatant and place a drop of sediment on dry glass slide. Cover it with a cover slip and examine under microscope.

I. Organized sediment:

A. Epithelial cells: They increases in cystitis, and other inflammatory condition of urinary tract.

B. Leucocytes (pus cells) : The number of leucocytes increases in nephritis, pyelonephritis, urothritis and cystitis. Presece of 10 leucocytes per high power field of 15 ml urine sediment are considered as an infammatory condition. The leucocytes are larger than erythrocytes and have granular appearance.

C. Erythrocytes: It is spherical in shape, like a faint colorless ring (Shadow cells). Presece of large number of erythrocytes is an indication of hemorrhage from urogenital system.

D. Casts: Presence of casts indicate mild form of renal irritation (hayline casts), nephritis and degeneration of kidney (epithilial casts), hemorrhage in the renal tubules (red cell cast) and suppurative infection as in pyelonephritis and kidney abscess (leucocytic cast).

E. Parasites: Capillaria plica (bladder worm of dog and cat). dioctophyma renale (kidney worm of dog). Other worms/or ova may be present in the urine sediment as a fecal contamination.

I. Un-organized sediment:

A. **Crystals:** Crystals occur as a result of acute liver disease, carbon tetrachloride poisoning and phosphorus poisoning. In alkaline urine, the crystal may be triple and amorphous phosphates or calcium carbonate and amonium urates. In acidic urine the crystals present may be amorphous urate, uric acid or calcium oxalate.

3. Cultural examination

Urine is collected in a sterile container, inculated on culture media directly or after centrifugation. Antibiotic sensetivity tests can be done.

3. Skin scraping examination

The scrapings must be collected deeply from the most affected part of the skin. the affected part should be moistened with mineral oil.

A. Direct method: The skin scrapings are placed on clean and dry glass slide with one drop 10 sodium hydroxide and cover it with cover slipe. Examine under low power of microscope.

B. Sedimentation methods: The skin scrabings are kept in 10% potassium or sodium hydroxide, 2-4 hours then transfer to centrifuge tube and certrifugated at 3000 rpm/10 minutes. The supernatant is discarded and one drop of the sediment examine under microscope.

C. Examination of skin scrapings for fungi:

Examination by Wood 's lamp: The Wood 's lamp has UV light, which is directed on the skin or scrabings collected in petridishes. If the fungus microsporum is present, it gives yellow green fluorescence, while no fluorescence in negative infection.

Microscopic examination: Collection of skin scrapings should be from the center as well as from the periphery of the lesion. Swab the lesion with 95% alcohol to remove any saprophytic organism. The skin scrapings are collected in sterilized petridishes containing 10% sodium hydroxide or potassium hydroxide. Put one drop on clean slide and cover it with cover slip and apply vaseline around the rim of cover slip. The slide is warmed gently for few second. Then examine for the presence of chains of hyphae and spores.

4. Examination of milk

Collection of the samples: The udder of the animal should be cleaned with water and antiseptic solution like potasium permanganate (1:1000). The hands of examiner should be cleaned with soap and antiseptics. Disinfect the teats with alcohol 70%, collect the milk sample from each teat in separate tube (5-10 ml) and discar the first 3-4 streams of milk. Tube should be stoppered and transported to the laboratory in ice for examination.

I. Physical examination of milk:

A. Color: Noramlly the color of milk is white but in acute mastitis it may become redish (presence of blood). Yellowish coloration occur during colostral period, feeding of carrots and tetracyclene therapy.

B. Reaction: The pH of normal milk is 6.4 to 6.6 but in mastitis it becomes alkaline up to 7.4 due to presence of sodium bicarbonate in the milk. The reaction can be determined by using pH strips or pH meter.

C. Odor: Noramlly the odor of milk is pleasant but in mastitis due to Actinomyces pyogenes it becomes obnoxious. In ketosis the odor of milk becomes sweet and fruity.

C. Cosistancy: The colostrum is viscus. In acute and subacute mastitis the milk contains fine and large flakes. The watery consistancy occurs due to poor feeding and chronic mastitis.

II. Chemical examination of milk:

A. White slide test: 4-5 drops of milk are placed on a clean dry slide. Add a drop of 4% sodium hydroxide and mix with glass rod. In mastitis it becomes thickened and flakes appear.

B. California mastitis test: This test is based on increased the number of leucocytes and increased alkalinity in mastitic milk. Take 0.5 ml milk from each quarter in plastic peddle cups and add equal quantity of California reagent, mix well by circular movement of peddle on a horizontal plane.

1. Liquid milk with no streaks or precipitation: negative for mastitis
2. Streaky milk: the weak positive
3. Slimy: ++.
4. Gelatinous: +++.

C. Leucocytic count: Mark the area on central portion of slide (1 squar cm). Put 0.1 ml, spread the milk sample by bacteriological loop in this area. Dry the smear and dissolve the fat by rinscing it in xylene for 2-5 minute. Fix the smear with alcohol 4-5 minutes and stain with methylene blue for 1 minute. Remove the excess stain by immersing the slide in alcohol. Count the leucocyte under oil immersion lense. The cell of 10 field are counted and averaged and multiplied by 500.000 to get the total number of leucocytes in the milk.

III. Bacteriological examination of milk:

A. Direct microscopical examination.

B. Cultural examination: The milk is collected in sterilized vials and stores in refrigerator and send to the laboratory, for isolation of the organism and antibiotic sensetivity test.

5. Rumen juice examination

Examination of rumen juice (RJ) gives rapid diagnostic test for monitoring the function of the rumen as well as the nutritional health of the animals. The rumen juice is collected from animals by using stomach tube, that was introduced through the mouth, then moved to and fro to obtained a representative sample from different areas of the rumen. The pH of the rumen juice, ammonia concentration, and volatile fatty acids must be measured as soon as possible.

Counting of rumen ciliate protozoa:

The rumen contents were fixed and stained with 4 times volume of methyl-green formaline saline (MFS) solution (100 ml formaldehyde 35%, 900 ml DW, methyl green 0.60 g and sodium chloride 0.80 g), then stocked in dark place until examination. After gentle mixing of fixed rumen juice sample, one drop was poured on hemocytometer slide, covered with a cover slip and examined under a light microscope.

The number of rumen protozoa per 1 ml was calculated as follow:

Calculation: Number of protozoa/ 1 ml RJ = $n \times 5 \times 10^4$

Identification of rumen ciliate protozoa:

Differential counts were also made using the same slide. Identification of genera and species of the ciliate must be recorded.

The genera of rumen ciliates were detected in Egyptian ruminant:

Buetschlia, Dasytricha, Isotricha, Oligotricha, Charonina, Entodinium, Diplodinium, Eodinium, Eudiplodinium, Epidinium, Metadinium, Polyplastron, Elytroplastron, Ostracodinium, Ophryoscolex and Caloscolex.

Notes

The dilution rate (1 ml rumen juice & 4 ml MFS) = 5.

Count the number of protozoa in one large corner square of WBC=n. The depth of hemocytometer is 0.1 so that you must multiply by 10. The number of protozoa/ µl RJ = n X dilution X depth.

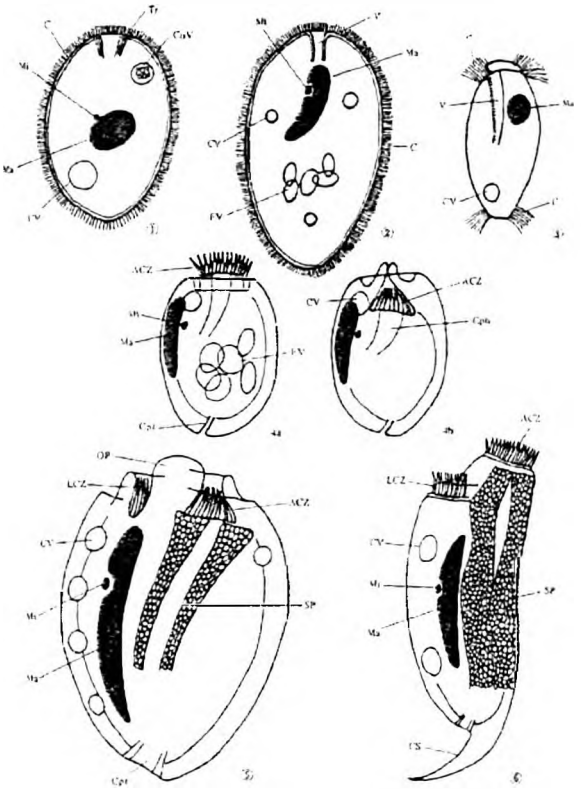
The number of protozoa/ 1 ml RJ = n X dilution X depth X 10^3

Identification of rumen ciliate protoxoa:

Differential counts were also made using the same slide. Identificatin of genera, species and forma of the ciliate must be recorded.

Distribution and composition of ciliate species in the rumen are affected by many factors, such as host species, keeping area of the host and feeding condition of host.

Diagram
Key for the identification of rumen ciliates



General morphology of rumen ciliate protozoa

1 Buetschliidae: Body is ovoid, uniform somatic ciliature, there is peculiar CoV
2 Isotricha spp: Body is ellipsoidal uniform somatic ciliature, no concretion vacuole
3. Charonina ventriculi: Ciliary zones are present at the anterior and posterior ends & distinct vestibulum
4. Entodium: Ciliary tufts only in the adorsal area & small in size
5. Diplodinium: Ciliary tufts in the adorsal area and antero-left side& skeletal plate is preset
6. Epidinium: Ciliary tufts in the adorsal area and antero-left side & skeletal plate is preset

ACZ: adorsal ciliary zone; C: cilia; CoV: concretion vacuole; Cph: cytoproct; CS: caudal spine; CV contractile vacuole; FV: food vacuole; LCZ: left ciliary zone; Ma: macronucleus; Mi: micronucleus; OP: operculum; SP: skeletal plate; Tr: trichite; V: vestibulum.

Key for the identification of rumen ciliates

For the identification of rumen ciliate, the following character should be noted

1. Shape: Spherical, ovoid, ellipsoidal, elongate or asymetrical.
2. Location of ciliary zone: Entire body surface, anterior and posterior body surface or anterior body surface only.
3. Number of ciliary zones: One or two.
4. Concretion vacuole: Present or absent.
5. Operculum: Present or absent.
6. Skeletal plate: Present or absent.
7. Number of skeletal plates: One, two, three, four or five.
8. Shape of skeletal plate: Broad or selender.
9. Number of contractile vacuole: One, two, three, four, five or more.
10. Shape of macronucleus: Spherical, ellipsoidal, rod or more complicated.
11. Location of micronucleus: Anterior, middle or posterior.
12. Number of caudal spine: Zero, one, two, three, four, five or more.
13. Size: Diplodiniinae and Ophryoscolecinae are bigger in size than Entodiniinae.
14. The micronucleus is located very close to the macronucleus, so that it is difficult to find it in a specimen fixed with MFS solution.
15. The caudal spines of large ciliate can note be adjusted to bring them all in to focus.
16. The skeletal plate are strongly stained with iodine, so mix a drop of diluted tincture iodine with a drop of 10% formaline on a glass slide to observe the skeletal plates.
17. In general, in specimens collected from hosts just after feeding, it is difficult to observe the organelles.

Diagram of key for the identification of rumen ciliates

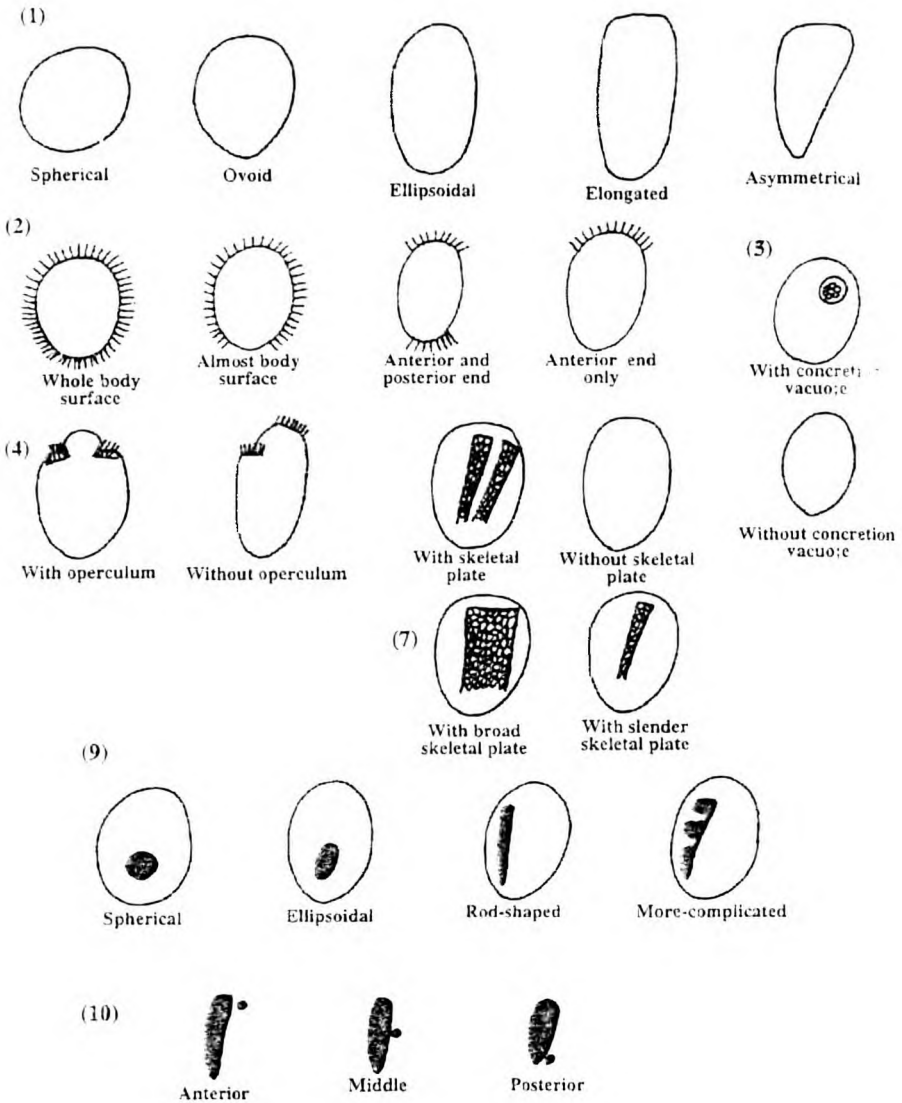
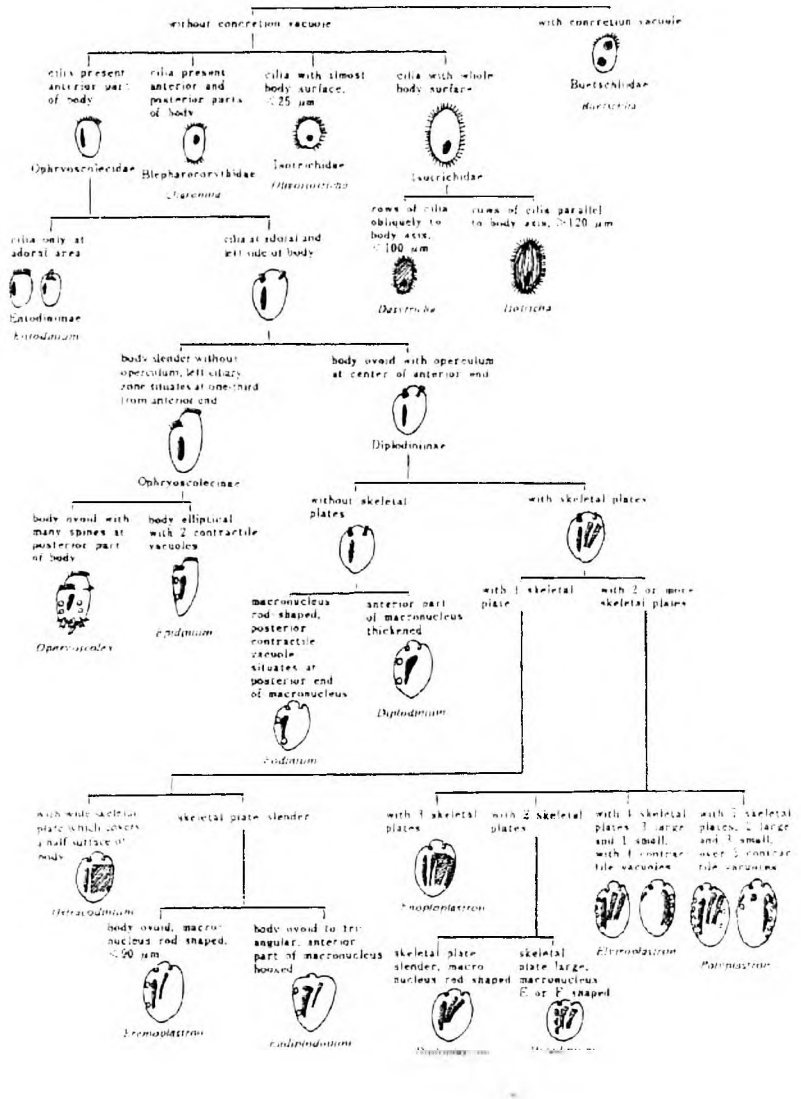


Diagram of key for the identification of rumen ciliates



Genus

species

forma

Entodinium

1. Entodinium ovinum

(The body is oval shape, the macronucleus is rod- shaped)

2. Entodinium parvum

(The body is symmetrical and elongated, the anterior end is flattened)

3. Entodinium simplex

(The body is ovoid, the posterior end is round, the macronucleus is rod- shaped)

4. Entodinium nanellum

(The body is relatively elongate, the anterior end is flattened)

5. Entodinium bimastus

(The body is ovoid, the posterior part is tappers, the macronucleus is rod- shaped)

6. Entodinium exigum

(Body is round, anterior end is flattened, the macronucleus is short and thick)

Entodinium longinucleatum (EL)

7. EL spinonucleatum

(Macronucleus is rod shape and its length is half of the body, three caudal spins, one is short and located on the right side and two are long and located on the left side.

8. EL acutonucleatum

(Similar to the entodinium longinucleatum with three caudal spinse, one is located on the right side and two are on the left side)

9. EL longinucleatum

(The body is ovoid, the macrocucleus extend from the anterior to the posterior part of the body, no caudal spine)

10. Entodinium minimum

(The body is asymmetrical, posterior part of the body is slender)

11. Entodinium dubardi

(Similar to simplex)

13. Parentodinium africanum

Entodinium caudatum (EC)

12. EC dubardi

14. EC caudatum

(One long right caudal spine and two short left spines, contractile vacule is located in the anterior part of the body, the macronucleus is rod- shaped, its anterior is thick and its posterior is thin)

18. EC lobosopinosum

(Similar to **ECC**, one long and one short spines were divided in the same manner)

15. Entodinium dilobum

(There are two broad and short spines at the right and left posterior end of the body)

Genus

species

forma

16. Entodinium rhomboideum

(The body is rhomboid, the macronucleus is rod - shaped)

17. Entodinium furca

19. Entodinium birostratum

20. Entodinium rostratum

(The body is a symmetrical, right surface is convex and left side is concave, there is left spine in the posterior end)

21. Entodinium bovis

(The body is round and the anterior end bccomes small)

41. Entodinium ovumrajae

Oligoisotricha

22. Oligoisoticha bubali

(The body is ovoid and small, the posterior end is slighly concave and the macronucleus is eleptical)

Charonina

23.Charonina ventriculi

(The body is relatively elongate, The vestibulum is clear and long, cilia cover only the anterior and posterior end of the body)

Eodinium (Eo)

24. Eo posterovesiculatum

(The body is ellipsoidal and small, the left side of the anterior end is depressed for the contractile vacuol)

Eudiplodinium

Eudiplodinium maggi

(The body is ovoid to triangular and very big in size, the macronucleus resembles 7 shape)

Eudiplodinium bubalus

(The body is ellipsoidal, posterior end has a small caudal lobe on the right side, two contractil vacuoles are present)

26. Eudiplodinium bovis

(Similar to preceding species, but slightly larger in size)

34. Eudiplodinium dilobum

(Similar to Eu bovis, but there are two process at right posterior end of the body)