IMPACTION OF THE RUMEN AND RETICULUM

- Introduction
- ▫ Here the rumen stops functioning, the musculature does not contract and so the food ingested stagnates.
- Occurrence
- ▫ This is a common condition in cattle.
- Predisposing causes
- ▫ Lack of exercise and debility predispose the animal to atony of rumen.

- Aetiology
- ▫ Overfeeding with large amounts of highly fermentable carbohydrate feeds. ▫ Tight packing of the rumen leaves no room for bacterial growth and normal ruminal fermentation and digestion. This leads to weak contractions of the ruminal and reticular walls and so the food does not get propelled. ▫ Lack of water ▫ Defective mastication and salivation due to defects in teeth or lesions of the tongue. ▫ Penetration of the wall of the rumen or reticulum by sharp objects like wire, nail etc., ▫ Paresis of rumen which may occur due to injury to vagus by pressure from abscesses, tumors, tubercular nodules, swollen lymph glands and ruminal displacements.

- Pathogenesis
- ▫ The pathogenesis of atony and impaction of the rumen after ingestion of large quantities of carbohydrate rich feeds is as follows o The carbohydrates are fermented by gram positive organisms, notably Streptococcus bovis, with the formation of lactic acid, resulting in to lowering of pH of the ruminal contents to as low as 4 to 4.5 from a normal 5.5 to 7.5. o Due to the production of lactic acid the osmotic pressure of ruminal contents increases and so fluid is drawn into the rumen from the blood leading to hemoconcentration, anuria, dehydration and circulatory collapse. o As the pH of the ruminal constituents falls, the motility of the rumen decreases and there may even be complete stasis. o In such an atonic rumen, in which the normal microflora are lost, Fusiformis necrophorus and fungi of the family Mucoraceae (those belonging to the genera Mucor, Rhizopus and Absidia) invade the ruminal wall producing ruminitis and ulcers. o At the lowered pH, normal microfloras of the rumen are destroyed, the lactobacilli and streptococci thrive and the salivary secretion ceases so that buffering action of the saliva is absent. o Absorption of the lactate causes acidosis.

- Clinical signs
- ▫ The stagnated food becomes putrified with the liberation of the foul smelling gases. ▫ In some animals, diarrhoea may be present if the putrid ingesta finds its way into the intestines causing enteritis. ▫ Anorexia develops and regurgitation stops.

- Gross pathology
- ▫ At necropsy, the rumen will be found to contain hard, caked, undigested food with evil smelling odor. ▫ In animals that die of acute atony, the contents of the rumen and reticulum are thin, porridge-like and bulky. ▫ The cornified epithelium is soft and peels off easily, exposing hemorrhagic areas underneath. ▫ The blood is dark and thick. ▫ Lungs show bleeding into the alveoli and bronchi.
- ▫ Heart musculature is flabby.
- Histopathology
- ▫ In animals that survive for three days and more, demyelination of the nervous system may occur.
- Sequelae
- ▫ In mild cases, if the primary cause is removed, normal state may be regained. ▫ In severe cases, toxemia will cause death. ▫ Enteritis, peritonitis and ketosis

GASTRITIS

- Definition
- ▫ Inflammation of the stomach is called gastritis.

- TYPES OF GASTRITIS
- ▫ It may be primary or may be secondary to some other infections, as canine distemper, viral diarrhoea, swine erysipelas. ▫ Gastritis may be acute or chronic.
- Acute gastritis
- ▫ Acute gastritis may be catarrhal, fibrinous, suppurative, haemorrhagic or necrotic, depending upon the cause and their severity.
- ▫ By far the most common is the catarrhal and to a lesser extent, the hemorrhagic.
- Pathogenesis
- ▫ In gastritis, food does not get digested ▫ Motility of the gastric wall is retarded ▫ Irritation may produce pain and vomiting.

- Catarrhal gastritis
- ▫ Gross pathology o The gastric mucosa is covered with mucus. o The mucosa in some places may show ulceration. o The mucosa is thick and red. ▫ Histopathology o The mucosa shows catarrhal exudation, hyperemia and leuococytic infiltration. o Some of the gastric glands may be damaged and lost.
- Acute hemorrhagic gastritis
- ▫ Occurrence o This is a common condition. ▫ Aetiology o caustic chemical poisoning o uremia o acute infectious diseases like pasteurellosis, braxy, leptospirosis (in dogs). ▫ Gross pathology o Due to haemorrhage, the mucosa is bright red in color and the gastric contents are blood stained. o Digested blood (acid haematin) imparts a brownish coloration to the contents.

- Parasitic Gastritis
- ▫ Occurrence o This is very common in animals. ▫ Aetiology o Cattle and sheep: Hemonchus contortus, Ostertagia ostertagi, Trichostrongylus axei o Horses: Habronema larvae, Trichostrongylus axei and Gastrophilus equi larvae. o Pig: Hyostrongylus rubidus, Physocephalus sexalatus. Simondsia paradoxa, Ascarops strongylina . o Cats: Gnathostoma spinigerum ▫ Pathogenesis o The strongyles are blood suckers and they produce minute injuries on the mucosa.
- o The larvae may burrow into the mucosa for completion of their life cycle and thereby cause damage to the epithelium and glands. o Heavy infestation besides causing anemia will produce catarrhal gastritis. ▫ Gross pathology o Gastrophilus sp. in the stomach may produce ulcers o Habronema larvae live in granulomatous nodules which may be infected by secondary bacteria and form abscesses.

- Chronic gastritis
- ▫ Aetiology o Usually the same causes as for the acute but operating for a longer time. o Sometimes it may be secondary to chronic gastric dilatation and cirrhosis. ▫ Pathogenesis o Ischaemia in gastric dilatation and passive hyperemia and failure of detoxication in cirhosis decrease the local resistance thereby facilitating infection. ▫ Gross pathology o The mucous membrane is thickened and covered with tenacious, viscid glassy mucus. o This condition is usually of a hypertrophic type with thickening of the gastric wall. ▫ Histopathology o There is exfoliation of the epithelium o Hyperplasia of gastric glands and muscle fibres along with cellular infiltration and hyperplasia of basal lymphocytic nodules. o The mucosa may be thrown into polypoid folds (polypoid gastritis). o The interstitial connective tissue hyperplasia exaggerates the mucosal foldings. o Occlusion of glands results into development of retention cysts.

- TORSION
- ▫ Torsion is a twisting of intestines on its axis.
- VOLVULUS
- ▫ Volvulus is a twisting of the bowel on itself as occurs when it passes through a tear in the mesentery.
- INTUSSUSCEPTION
-
- ▫ Intussusception is telescoping of a portion of intestine into another, usually the anterior into the posterior.
-

HERNIA

- Definition
- ▫ Hernia of the abdominal organs is the protrusion of the abdominal viscera through a natural or artificial opening.

ENTERITIS

- ▫ Enteritis is inflammation of the whole of the intestinal tract. But usually it is applied to the inflammation of the small intestines. ▫ The inflammation of the colon is called colitis , that of cecum typhlitis, that of rectum proctitis and of cloca cloacitis or vent gleet.
- Introduction
- ▫ Enteritis is of immense economic importance.
- Occurrence
- ▫ Enteritis is very common in domesticated animals and fowls. ▫ Since enteritis occurs along with gastritis (the same irritants causing gastritis passing on to intestines produces enteritis also) gastro-enteritis is a frequent condition met with.
- Etiology
- ▫ Causes are many and varied and they include chemicals, bacteria, viruses, protozoa, rickettsia, helminths, fungi, , disturbed metabolic processes as in ruminants, venous congestion as in portal hypertension and congestive cardiac failure, toxins of Clostridia, coliforms and spoiled or mouldy feeds and avitaminosis.

- Gross pathology
- ▫ In enteritis, the whole length of the bowel may not be affected, inflammation localizing only at one part or the other
- TYPES OF ENTERITIS
- Based on the nature of the exudate and the changes produced in the intestinal tract, enteritis is classified as follows.
- ▫ Catarrhal Enteritis ▫ Hemorrhagic Enteritis ▫ Fibrinous Enteritis ▫ Suppurative Enteritis ▫ Necrotic Enteritis

CIRRHOSIS

- ▫ Cirrhosis of the liver is chronic hepatitis characterized by degeneration and hyperplasia of hepatic cells and fibrosis. ▫ The stimulus for the fibroblastic proliferation is some irritant, chronic and severe enough to produce degeneration and necrosis of the parenchymatous cells. ▫ The irritant may reach the liver through (a) The portal vein (b) hepatic artery and (c) Bile ducts.
- The classification of cirrhosis
- ▫ Portal or nodular cirrhosis ▫ Multinodular or Atrophic or Gindrinker's or Laennec's cirrhosis ▫ Biliary cirrhosis (Monolobular or hypertrophic cirrhosis)