

The digestive system is anatomically and functionally divided into segments: The oral cavity, oesophagus, stomach, small and large intestines and rectum. It consists of (a) alimentary canal and (b) accessory organs.

(a) Alimentary Canal

- This includes:
- (i) Buccal/oral cavity
  - (ii) Oesophagus
  - (iii) Crop (in birds)
  - (iv) Fore stomach as seen in ruminants
  - (v) Stomach/Abomasum
  - (vi) Intestines – small and large
  - (vii) Anus.

(b) Accessory Organs

- These are:
- (i) Liver
  - (ii) Pancreas
  - (iii) Salivary gland
  - (iv) Bile organ
  - (v) Tonsils
  - (vi) Anal sacs

The alimentary system is directly or indirectly involved in most diseases that affect animals, and it is accessible in large measure to routine clinical examination.

In this course, emphasis will be placed on the pathology and pathogenesis of diseases and conditions that are prevalent in Africa. In veterinary practice, gastro-intestinal tract problems are among the most commonly encountered conditions. Many of the diseases are life threatening and others cause great economic losses to the producers. Unfortunately, some of these diseases, especially intestinal conditions of cats and dogs are not fully understood and treatment is often empirical.

**I. The basic reactions of the system to injury include:**

- (a) Inflammation
- (b) Cellular degeneration and necrosis resulting in vesicles, erosion, ulcers, villous atrophy e.t.c.
- (c) Proliferation of cells as a reparative process but may lead to neoplasia.
- (d) Altered rate of secretion, absorption and/or motility.

**II. Factors predisposing the system to disease include:**

- (a) Direct exposure to the environment
- (b) Management (related to feeding and confinement).
- (c) Entry of pathogenic organisms and toxins through feed and water.
- (d) Relatively loose suspension of its segments in the abdominal cavity
- (e) Large absorptive surface make it possible for rapid absorption of poison and toxin.

**III. Protective Mechanism include:**

- (a) Endogenous secretion and fluid in-take
- (b) GIT motility and rhythmic contractions of wall and movement of villi
- (c) Rapid epithelial turnover (e.g. 2-8 days for enterocytes covering villi)
- (d) Local immune response (Payer's patches and tonsils)
- (e) Eructation, Belching and Vomiting.

**IV. Signs of Alimentary disease specific and non-specific. These include:**

- Diarrhea
- Vomiting
- Anaemia
- Bottle jaw in sheep
- Hypersalivation
- Painful mastication
- Ascitis/Pot Belly
- Erosions/ulcers in the mouth.

## **PATHOLOGY OF BUCCAL CAVITY AND MUCOSA**

### **DEVELOPMENTAL ABNORMALITIES**

A variety of development abnormalities occur in oral cavity. Some are incompatible with life unless surgically corrected. Some have been showed to have hereditary component while most are idiopathic. Developmental abnormalities. Occur sporadically in all species and may be due to genetic defect or toxicity.

- A. Primary Cleft Palate (cheiloschisis) is sometimes referred to as harelip since it is a normal feature of the rabbit. It is a failure of fusion of upper lip along the midline or philtrum. It arises from incomplete fusion of the frontonasal process with maxillary processes.

- B. Secondary Cleft Palate (palatoschisis/cleft palate): Arises from inadequate growth of either of the two palatine shelves from the maxillary processes. May occur with arthrogyrosis in charolais cattle and appears to be hereditary. Intoxication with *Veratrum californicum* in sheep and *Crotalaria* in pigs may also lead to cleft palate. It can also be caused by steroid administration during pregnancy in primates including human beings.

Significance: Depending on the size of the defect which can involve only the soft palate or both (soft and hard) palates. The most important sequela are starvation due to an inability to create a negative pressure in the mouth and hence failure to suckle and aspiration pneumonia. Since no effective separation is present between the oral and nasal cavities the lesion results in direct communication between oral and nasal cavities, leading to aspiration pneumonia and death.

- C. Growth abnormalities of the jaws: Manifested as brachygnathia superior or inferior, prognathism and agnathia. May cause serious dental occlusion abnormalities.
- D. Epitheliogenesis imperfecta: Incomplete development of cutaneous epithelium including the tongue. Area appears ulcerated. Tooth development can be affected.

## **INFLAMMATORY CONDITIONS OF THE MOUTH**

- (a) Terms used include:

Stomatitis – oral mucosa in general

Cheilitis – lips

Glossitis – tongue

Gingivitis – gums or gingival

Pharyngitis – pharynx

Tonsillitis – tonsils

Sialoadenitis – salivary glands

Angina – soft palate

- (b) Causes include:

Infectious agents locally or following septicemia

Trauma

Chemical; injury

Auto-immune disease

Nutritional factors

Idiopathic

- (c) Importance is that stomatitis can be indicator of certain major systemic diseases such as viral stomatitis and uraemic stomatitis. Affected animals will show anorexia, painful mastication, hypersalivation or foul mouth odour. This leads to loss of condition and unthriftiness.
- (d) Morphologic manifestation are a combination of inflammatory and degenerative changes resulting in vesicles, erosions, ulcers, papules or pseudomembranes (diphtheritic) if superficial or granuloma if deep.

## **STOMATITIDES**

### Types of Stomatitides

1. Catarrhal
2. Vesicular
3. Erosive & Ulcerative
4. Proliferative/Papular
5. Deep (Granulomatous)
6. Eosinophilic stomatitides
7. Lymphoplasmacytic stomatitides.

### **1. CATARRHAL STOMATITIDES**

This is non-specific stomatitis, seen more often in man than animals.

### **2. VESICULAR STOMATITIDES**

These are of special interest in veterinary medicine because they are characteristic of some viral epizootic diseases that have major economic impact on our food industry. These diseases include foot & mouth disease (FMD), vesicular stomatitis, swine vesicular disease and vesicular exanthema. All are highly contagious and have short incubation periods. In dogs & cats, vesicular stomatitis is more likely part of some auto-immune disease of the skin and muco-cutaneous junctions e.g. pemphigus vulgaris and bullous pemphigoid. Also in cats with calicivirus infection.

#### Grossly

Small ulcers and fluid filled vesicles of the lips, buccal mucosa surface and margin of the tongue.

#### Pathogenesis

Viral replication in epithelium (Stratum spinosum) → cell lysis → Intracellular oedema and ballooning degeneration → vesicles → bullae → erosion → ulcers and cellular infiltration. Lesions may also be present in the nasal mucosa, oesophagus, rumen, interdental spaces, teats and vulva. Hydropic degeneration of the cells of the stratum spinosum is characteristic of vesicular stomatitis.

### **3. EROSIIVE AND ULCERATIVE STOMATITIDES**

### Pathogenesis:

The agents cause epithelial necrosis and inflammation without vesiculation. Erosions and ulcers occur in stratified squamous epithelium of mouth, oesophagus and forestomachs and in some cases extend to the intestines. Some specific diseases include:

- (i) Bovine viral Diarrhoea (Mucosal Disease)
- (ii) Malignant catarrhal fever
- (iii) Rinderpest (cattle) peste des Petits Ruminants (PPR) in goats & sheep.
- (iv) Blue tongue by an orbivirus sheep
- (v) Feline rhinotracheitis
- (vi) Chronic uraemia
- (vii) Herpesvirus infection in Primates
- (viii) Eosinophilic granuloma or Rodent ulcer in cats
- (ix) Oral Eosinophilic granuloma (Siberian husky dogs).
- (x) Canine distemper in the dog
- (xi) Noma: an acute gangrenous stomatitis that has been recognised in human beings, Rhesus monkeys & dogs.

#### **4. PROLIFERATIVE/ PAPULAR STOMATITIDES**

Pathogenesis: Agents cause epithelial degeneration and hyperplasia as well as cellular infiltration, resulting in a firm elevation of affected area (papule). Healing process is slow. Seen mainly in young ruminants. Examples include:

#### **PATHOLOGY OF RUMINANT FORESTOMACHS**

Most diseases of the organ are related to management/husbandry practices.

##### **1. RUMINAL TYMPANY (BLOAT)**

- Over-distension of rumen and reticulum with gases of fermentation and may be primary or secondary.

##### **(a) Primary bloat:**

Occurs in cattle feeding on legumes or concentrates which promote formation of stable foam in rumen. The foam is mixed with ruminal content and does not escape during eructation and is not easily relieved by intubation or cannulation. Succulent legumes e.g. alfalfa, clovers are most dangerous because of their content of their leaves i.e. cytoplasmic proteins which are soluble proteins. Susceptibility to bloat varies with individual cattle and may be inherited.

Pathogenesis: Fermentation of soluble proteins and production of non-volatile acids lead to lowering of rumen pH (5-6) and formation of stable foam. This is further enhanced by diminished salivation increased production of bacterial polysaccharides, depressed eructation and individual susceptibility. Rapid accumulation of gases (methane, CO<sub>2</sub>) leads to increased intra-ruminal and intra-abdominal pressure. Death occurs from compression of the diaphragm and large abdominal veins leading to respiratory and circulatory failure.

Gross: Animal found dead and rolled on its back due to marked abdominal distension. Rumen content is frothy. Also marked congestion of head and neck anterior to thoracic inlet and well demarcated as "bloat line" in oesophagus. Distinguish from PM bloat.

(b) Secondary or Obstructive bloat:

Results from physical obstruction or stenosis of oesophagus leading to defect in eructation. The condition is common during dry season when animal feed is scarce.

## **2. FOREIGN BODIES**

Trichobezoar – hair

Phytobezoar – plant material

Lead substances – intoxication

Sharp metals, nails, wire – traumatic reticuloperitonitis

Sharp objects in reticulum often penetrate the wall and may move through diaphragm and pericardium to the heart, causing inflammation all the way.

Also called hardware disease.

## **3. INFLAMMATION (RUMENTITIS, RETICULITIS & OMASITIS)**

It is often an extension of stomatitis and oesophagitis as seen in some viral infections but the most important and specific form of inflammation is that due to grain overload (synonyms ruminal lactic acidosis, engorgement toxæmia rumen overload). It is seen mainly in intensive beef and dairy production and it occurs when the animals have sudden access to a higher level of carbohydrates. Primary bloat may co-exist.

Pathogenesis: Sudden change to a much higher highly fermentable, CHO rich carbohydrate diet promotes growth of gram cellulolytic negative/positive bacteria (streptococci and lactobacilli) leading to the production of excess lactic and volatile fatty acids and a ruminal pH less than 4.5. The acids damage the rumen mucosa and attract large quantity of fluid from the circulation, leading to dehydration, acidosis, rumen atony and toxæmia. Death may occur in 24 hours or disease may become subclinical and yet have significant sequelae.

Gross: Nonspecific, but rumen pH below 5 is helpful + absent at grain. There is Hyperaemia of mucosa . Intestinal content is increased, watery and acidic.

Histo: Vesiculation Hydropic degeneration and coagulative necrosis followed by: neutrophilic infiltration of the epithelium of ruminal papillae.

Sequelae:

(i) Necrobacillary rumenitis

Etiology: *Fusobacterium necrophorum*

Produces ulcers and stellate scar tissue in forestomachs.

Bacteraemia leads to liver abscesses, vena caval syndrome e.t.c.

(ii) Mycotic rumenitis

Etiology: *Rhizopus, Mucor, Absidia*

A more severe complication and could also be secondary to any other damage to the forestomachs and to prolonged antibiotic treatment especially in calves.

Gross: Well-demarcated, often circular areas of haemorrhagic infarction covered with a fibrino-haemorrhagic exudates. Lesion may be present in the liver.

Histo: Acute inflammation with haemorrhage, oedema, coagulative necrosis and presence of numerous non-septate branching hyphae, vasculitis and thrombosis.

(iii) Polioencephalomalacia

Proliferation of thiaminase producing bacteria in the acidic rumen has been linked to some cases of polioencephalomalacia.

(iv) Laminitis

(v) Renal cortical necrosis

## DISEASES OF STOMACH AND ABOMASUM

### 1. PHYSICAL INFLUENCES

(a) Acute Gastric Dilatation and Volvulus

Seen especially in large breeds of dogs following a heavy meal, especially dry dog food. Starts as dilatation and torsion and progresses to volvulus (rotation on mesenteric axis). Occurs also in horses and pigs.

Pathogenesis: Uncertain but probably includes swallowing air or ingestion of large quantity of readily digestible and fermentable food → gas production by bacteria including *Clostridium perfringens* → functional obstruction of cardia and pylorus → further gastric distension → compression of lungs and occlusion of posterior vena cava → hypovolemic shock.

Gross: severe abdominal distension due to accumulation of fluid with gas in stomach, varying degrees of clockwise rotation of stomach (180 – 360°), V-shaped bending of enlarged spleen, and congestion of intestines. Stomach may show haemorrhagic infarction and rupture if there is volvulus.

(b) Displaced Abomasum/Abomasal Displacement

In dairy cows, displacement is usually to the left but would also be to the right. In calves, it is mostly to the right. Most cases occur between 2 weeks prepartum and 2 weeks postpartum in older, high producing dairy cows. Often not fatal except when complicated by volvulus in right displacement and in preruminant calves.

Pathogenesis: Uncertain but related to parturient stress → abomasal atony and increased gas production → displacement to left or right. →

(c) Chronic Gastric Dilatation

Secondary to other diseases, e.g. acute gastric dilatation in dogs, “wind-sucking” in horses, poor nutrition in ruminants and chronic diarrhea in calves.

(d) Abomasal Dilation and Emptying defect

A syndrome that occurs in sheep, especially Suffolks. Characterized by chronic inappetence and weight loss, and at necropsy they have a markedly distended abomasum containing digested ingesta resembling rumen contents. The cause is unknown.

(e) Omasal Impaction and Rupture

## 2. GASTRIC ULCERS

Seen commonly in cattle, pigs, dogs and cats but not as much as in humans where the world drug market for peptic ulcers was estimated to be \$8 billion in 1992 (*Science*, Vol. 267: p. 173, 1995). Involves necrosis and loss of surface epithelium and exposure of underlying tissues to gastric juice (HCl & pepsin) and pathogens. Erosion of blood vessels leads to internal hemorrhage, anaemia and melena. Perforation of the wall leads to peritonitis. Although most cases in humans are now believed to be caused by *Helicobacter pylori*, the causes in animals are still poorly understood. In generally,



they involve an imbalance between the aggressive (acid & pepsin) and protective (mucus & bicarbonate) factors that maintain the integrity of the mucosa. The causes include the following:

- (i) Hypersecretion of acid by parietal cells as seen in mastocytoma (via XS histamine) and in gastrin secreting pancreatic islet cell tumor (Zollinger-Ellison syndrome) in dogs. Histamine binds to H-2 receptors on parietal cells and induce increased HCl secretion as well as hypermotility.
- (ii) Anti-inflammatory drugs (such as Aspirin) by interfering with prostaglandin synthesis (a gastroprotective agent) and by direct necrosis of epithelial cells.
- (iii) Stress and XS glucocorticoids which may interfere with cytoprotective factors in mucosa.
- (iv) Secondary to dietary changes, foreign bodies, infectious agents (e.g. *Helicobacter pylori*?) and uraemia. In pigs, severe septicaemia is often accompanied by acute gastric infarction and ulceration but this is in the glandular portion.

Gross: depends on the species

Cattle: Multiple punched out areas in the ventral-most part of pylorus from a few mm to 15 cm in size. May be nonclinical or cause severe disease including perforation.

Pig: Affects squamous portion (pars oesophagea) and appears as round, oval or stellate crater, up to 10 cm in diameter. Ulcer may be covered by a fibrinonecrotic membrane or blood clot overlying a thick bed of granulation tissue. Large amount of blood may be present in stomach and intestine. First changes are parakeratosis and hyperkeratosis before ulceration. Glandular stomach may be affected as well.

Horses: Foals mainly and similar to pigs.

Dog: Body of stomach but more in duodenum.

### 3. GASTRITIS

In dogs, acute and chronic gastritis may occur but are not common and except in cases of uraemia, the cause is often undetermined. In chronic cases, the mucosa may become atrophic or hypertrophic and *Helicobacter pylori* or H fells is being associated with some of these. In ruminants, acute gastritis occurs in the form of braxy caused by Clostridium septicum, especially in sheep. Mycotic gastritis is usually secondary to insults caused by other agents such as viruses, steroids e.t.c.

### 4. NEOPLASIA

Gastric tumors are generally uncommon and may arise from the mucosa, connective tissue or smooth muscle. These include:

- (a) Lymphosarcoma in cattle, pigs and dogs as part of systemic disease.
- (b) Squamous cell carcinoma in horses.
- (c) Adenocarcinoma (most common gastric tumor in dogs). May appear as thick, ulcerated, plaque on antrum or body. Provokes severe scirrhous reaction and frequently metastasizes to regional lymph nodes, liver and lung. Benign papillary adenomas are rare.

## **DISEASES OF SMALL AND LARGE INTESTINES**

### **1. CONGENITAL ANOMALIES**

- (a) Segmental defects occur in form of aplasia, stenosis or atresia in piglets and calves and atresia ilei in calves are the most common.
- (b) Persistent Meckel's diverticulum extends from anti-mesenteric side of intestine to umbilicus. Derived from omphalomesenteric duct.

### **2. OBSTRUCTIONS AND FUNCTIONAL DISORDERS**

Obstruction may be due to stenosis, obturation, compression or may be functional as in grass sickness in horses (equine dysautonomia). Foreign bodies, enteroliths, and bezoars may caused obturation whereas tumors, abscess, e.t.c. cause compression. Enteroliths (intestinal calculi) form by the concentric deposition of salts around a central nidus. The consequences of the presence of foreign bodies vary and they may lead to death from toxemia or from shock due to dehydration, hypokalemia and acid-base imbalance. Death may also be due to starvation if the obstruction is in distal part. At necropsy, animal is dehydrated and abdomen is distended due to dilation of bowel proximal to obstruction. Distal portion is collapsed and empty. Specific conditions include:

- (a) Adynamic (Paralytic) ileus: Disorder of stomach and intestines characterized by absence of normal tone and peristalsis due to sympathetic inhibition. Seen following extensive manipulation of intestines during surgery or peritonitis. Intestines are distended with gas and fluid and wall is flaccid.
- (b) Intussusception: Telescoping or invagination of one segment of the intestine (intussusceptum) into another (intussusciens). Seen in all species, cause is often undetermined but could be associated with endoparasitism, malnutrition, enteritis and diarrhoea. Affected area is swollen, dark-red or black and heavy because of the mass within. Venous stasis leads to ischemic necrosis and gangrene of affected segments. Following surgical correction, reperfusion injury may occur.
- (c) Volvulus & Torsion: Twisting of intestine upon its mesenteric axis (volvulus) or along its long axis (torsion) leads to luminal obstruction and ischemic injury. Seen mostly in

horses and swine. Rotation may be 180° – 720°. Torsion of caecum is seen in horses, cattle and swine.

(d) Herniation: Displacement of intestine through normal or pathological foramina in peritoneal cavity (internal) or outside the abdominal cavity but within a hernial sac formed by parietal peritoneum (external).

- Internal hernias are less common and may occur at the epiploic foramen of Winslow, omental or mesenteric tears, pelvic area following castration.
- External hernias may be ventral, umbilical, scrotal femoral, perineal or diaphragmatic may or may not lead to clinical disease.
- Sequelae include incarceration, infarction, paralytic ileus and perforation.

### III. INFLAMMATION OF INTESTINES

- Enteritis, typhlitis, colitis, proctitis depending on portion affected and generally result in diarrhoea or dysentery.
- Diarrhoea: Increased volume and fluidity of faeces while dysentery is bloody diarrhea or enteritis accompanied by abdominal pain. Diarrhoea leads to dehydration, acidosis, hypoproteinemia, electrolyte imbalance and death especially in young animals. Neonatal diarrhea is of great economic importance in food producing animals and irrespective of the cause, fluid therapy is essential.

Pathophysiologic mechanisms of diarrhoea include:

- (a) Mucosal hypersecretion e.g. enterotoxigenic colibacillosis and human cholera.
- (b) Malabsorption and maldigestion e.g. in villous atrophy, infiltration of lamina propria or defective pancreatic secretions.
- (c) Increased mucosal permeability e.g. portal hypertension, lymphatic obstruction or epithelial necrosis.
- (d) Osmotic diarrhea secondary to accumulation of non-absorbable solutes.
- (e) Motility disorder (intestinal hypermotility).

In general, decreased absorption of fluid, electrolytes or nutrients in either small or large intestine or increased secretion of fluid and electrolytes in the small intestine will result in diarrhoea.

Gross Morphologic types of Enteritis

- (a) Catarrhal: Mildest form and not diagnostic

- (b) Haemorrhagic: Severe form seen in arsenic poisoning, clostridial infections, Campylobacteriosis, coccidiosis and anthrax.
- (c) Fibrinous/fibrinonecrotic: Also severe form. Seen in *Fusobacterium necrophorum* infections, salmonellosis and campylobacteriosis. Fibrin forms a pseudomembrane or diphtheritic membrane which may detach as casts.
- (d) Ulcerative e.g. hog cholera, BVD, mycotic
- (e) Proliferative or hyperplastic e.g. campylobacter-like (Ileal symbiont intracellularis bacteria, ISI) infection.
- (f) Granulomatous e.g. Johne's disease

Causes of enteritis are many and some of the more important infectious causes are discussed later in the course. Dietary factors such as milk replacers in farm animals, deficiency of vitamins, and allergies may predispose to enteritis.

## **ACCESSORY GLANDS**

### **PATHOLOGY OF SALIVARY GLANDS & LIVER**

1. Salivary Mucoceles: Thin-walled, fluid filled pseudocysts resulting from trauma and accumulation of saliva within the mucosa and submucosa.
2. Ranula: Mucocele of sublingual salivary gland.
3. Sialolithiasis: Calcium concretions in ducts of salivary gland.
4. Sialoadentitis: Usually as part of stomatitis but there are a few viruses that specifically affect the glands e.g. coronavirus causing sialodacryoadentitis in rats and rabbits virus in domestic animals.
5. Tumors: Seen from time to time.

In a recent histological evaluation of 245 salivary gland submissions from dogs and cats, 30% were malignant neoplasms, 26% sialoadentitis, 9% sialocele, 8% infarction, 16% normal and 11% miscellaneous conditions (Spangler & Culbertson, JAVMA, 198: 465-469, 1991).

## **B. LIVER PATHOLOGY**

Liver is the largest gland of the digestive system and it is made up of lobules which is the functional unit of the organ. Functions of the liver include: Metabolism, detoxication, Bile secretion, erythropoiesis (in young animals) phagocytosis (via the kuffer cells), blood clotting etc.

### Reaction of Livers to Injury

- Hypertrophy
- Degeneration (fatty degeneration)
- Necrosis

- Hyperplasia – as seen in Vit E and/or selenium deficiency; Aflatoxicosis.
- Fibrosis – proliferation of fibrous connective of the liver lobule without damaging the hepatocytes.
- Cirrhosis – this is the combination of fibrotic tissue proliferation, hepatocyte damage and hepatic regeneration.

### CONGENITAL ABNORMALITIES

- (a) Abnormal lobulation: pigs have 4 lobes while dog have 5. Any deviation for this is abnormal.
- (b) Accessory Liver
- (c) Congenital cysts.

### B. FUNCTIONAL DISORDERS

- 1) Hepatic displacement – This is sequel to diaphragmatic hernia
- 2) Hepatic Rupture: rupture is due to trauma, fatty degeneration and hepatomegaly. Sequelae of liver rupture include: internal haemorrhage, defective blood clotting and adhesion of liver to other visceral organs.
- 3) Liver Torsion: This condition is common in pigs whose liver is big, large and distended. There is twisting of the liver against the mesentery or intestine.
- 4) Liver Atrophy: This is reduction in the liver size. It may be due to malnutrition, salinity and chronic disease e.g. Tuberculosis.
- 5) Hepatomegaly: It is the enlargement of the liver. Causes include: severe congestion and oedema, engorgement with bile, acute diffuse hepatitis, nodular hyperplasia due to aflatoxicosis and vit. E and/or selenium deficiency, fatty liver, amyloidosis and neoplasm. Sequelum depends on the aetiology.

### C. PIGMENTATION OF THE LIVER

It may be any of these forms

- (a) Congenital melanosis: Here the liver is darker than normal due to deposition of melanin.
- (b) Haemosiderosis: The liver is golden yellow or golden brown in colour. It is due to haemosiderin deposits.
- (c) Icterus: The liver is yellowish as found in Acute Leptospirosis in dogs and acute Haemolytic icterus in cats due to Haemobartonella felis
- (d) Lipochrome Pigment: The liver is also yellow due to presence of a wear & tear pigment

## D. LIVER PHOTSENSITIZATION

This condition occurs when certain photodynamic substances present in the ingested feed accumulate in the liver due to failure of metabolism. Consequence is that these substances attract ultraviolet ray from sunlight and become photosensitized and cause cancer, ulcer et.c e.g. facial eczema in sheep.

## E. INFLAMMATION

Inflammation of the liver tissue is called hepatitis. In this condition, there is pronounced cloudy swelling and fatty infiltration of the hepatocytes. The term is often used to describe all stages of the process from necrosis to healing by fibrosis.

Classification

1. Infectious hepatitis and
2. Non-infectious-hepatitis, also known as toxic hepatitis.

There are 2 type of toxic hepatitis

- (a) Acute toxic hepatitis
- (b) Chronic toxic hepatitis

(I) Infectious hepatitis

Here infectious agents are involved and these include: Viruses e.g. infectious canine hepatitis virus. Rifty valley fever virus, wesselbron disease and Herpes virus infection.

Bacteria – Mycobacteria spp., Treponema spp (human) Toxoplama spp, Histoplasma (turkey) Necrobasillus spp Leptospira spp, Staphylococcus spp e.t.c.

Parasite – Hydatid cyst, cysticercus cyst, Echinococcus spp Fasiola spp and Emerica spp (Rabbit) .

Nematodes include: Ascarid Suum and Strphanurus dentatus

Sequelae – Fibrosis, Cirrhosis, hepatic abscess, pyemia, toxaemia and hepatic rupture.

## E. CIRCULATORY DISTURBANCES IN LIVER

It may be in these forms.

- (I) Congestion
  - (i) acute – right sided heart failure
  - (ii) Chronic – congestive heart failure
- (i) Acute congestion is usually due to right sided heart failure

Characteristic

- There is blood stagnation in hepatic veins and central veins
- Fatty degeneration in the peripheral tissue of the liver. Here the liver will be creamy in colour.

- Nut meg liver – i.e. the liver has mottled or mosaic or variegated appearance.
- Expanded and widened central vein.

(ii) Characteristic of chronic congestion

- Stagnation of RBC causing ischaemia
- Distension of central vein due to more blood coming in than can be evacuated.
- Diapedesis (extravasation Rbc)
- Chronic congestion of the liver is usually due to congestive heart failure.

(2) Haemorrhage

Causes include:

- Detective blood clotting
- Chemical poison e.g. warfarin poisoning
- Infections agents – viruses, bacteria e.t.c.
- Genetic abnormality – Haemophilia.
- Grossly the liver will be enlarged.

(3) Telangiectasis

This is sinusoidal dilatation due to engorgement of blood or bile in areas where hepatocytes are lost.

Grossly such liver enlarge liver has focal blue-black areas of discolouration.

Aetiology is not clear but Fusiformis necrophorus and nutritional disorder have been incriminated.

Sequelum – the function of the liver is not affected but such liver is usually condemned during meat inspection.

(4) Embolism – causes include, parasite, fat, cysts.

Sequelae: Liver rupture and suppurative inflammation.

(5) Infarction: Occurs infrequently because of the dual blood supply to the liver – portal vein and hepatic artery. Causes include: liver torsion especially if individual lobes are affected which can cause vascular occlusion.

(6) Gross: The area is sharply delineated and appear dark red.