STOMACH AND ABOMASUM

• Postmortem changes

- * Rigor mortis
- * Hypostasis
- * Sulfmethemoglobin
- * Bile agent
- * Gastromalasia

Foreign bodies

- *Foreign bodies in distemper and rabies dogs
- Trichobezoars long haired cats
- Phytobezoars and trichophytobezoars calves reared on diets low in roughage



PYLORIC STENOSIS



- It is relatively common in dogs, and rare in cats and horses.
- Recurrent vomiting and poor growth in recently weaned animals suggest the clinical diagnosis of a congenital lesion.



CONGENITAL PYLORIC STENOSIS

In some dogs there may be hypertrophy of pyloric smooth muscle, which appears grossly thickened.

• Tonic stenosis of the pyloric sphincter may occur in dogs

✓perhaps because of alterations of the myenteric plexus or

✓Gastrin excess



ACQUIRED PYLORIC STENOSIS

Functional:

- Abomasal hypomotility
- <u>Physical</u> causes of acquired pyloric stenosis or obstruction include:
- Ulceration and stricture of the pyloric canal in any species
- Complication of polyps and tumors
- Chronic hypertrophic pyloric gastropathy in dogs_is the term coined for a syndrome of pyloric obstruction in dogs, associated with mucosal hypertrophy, hypertrophy of circular smooth muscle, or a combination of the two.





GASTRIC DILATION AND DISPLACEMENT

GASTRIC DILATATION in the **HORSE**

<u>Primary</u> gastric dilation in horses happens due to

- Consumption of excess fermentable carbohydrate
- Lush pasture or
- Excessive intake of water.

Secondary (more frequent) Gastric dilation in the horse is often

- Obstruction of stomach, small bowel, or of colic with ileus
- Grass sickness
- Ingestion of *Datura sp.* Seeds, which contain a parasympatholytic alkaloid, can also cause ileus, leading to gastric dilation.



GASTRIC DILATATION IN THE DOG

- Commonly in dogs
- Gastric dilation and volvulus are usually problems associated with overeating, and probably aerophagia,
- Especially in the *deep-chested breeds*, such as Great Danes, St. Bernards, Irish Setters.
- The gas that contributes to the development of dilation is probably the result of aerophagia,
 and possibly the evolution of carbon dioxide by physiologic mechanisms.
- Inability to relieve the accumulation of food, fluid, and gas in the stomach causes the
 organ to dilate and change its intra-abdominal position, so that its long axis rotates from a
 itransverse left-right orientation to one paralleling that of the abdomen.



Appearance of Volvulus

It rotates on the **mesenteric axis clockwise** resulting in a gastric volvulus with an **obstructed esophagus** that prevents eructation and thus further contributes to gastric dilation

The spleen, attached to the stomach by the gastrosplenic ligament, rotates with the stomach and is thus folded back upon itself and located in the right cranial abdomen against the diaphragm.

The splenic vein is compressed, resulting in a **congested spleen**, because the arterial blood supply remains patent longer than venous drainage.



Appearance of Volvulus

• **Venous infarction** of the gastric mucosa

ensues

- The gastric wall are edematous and dark red
 to black
- Bloody content in the lumen of the stomach
- Necrosis of ischemic mucosa occurs, and the stomach may rupture.
- Hemoperitoneum



RESULTS OF VOLVULUS

✓ Obstruction of veins by volvulus and pressure exerted by the distended stomach result in decreased return via the portal vein and posterior vena cava, causing reduced cardiac output and circulatory shock.

✓ Increased intra-abdominal pressure affects on the diaphragm and compromises respiration.

✓ **Death** is inevitable in dogs not treated early.



ABOMASAL DISPLACEMENT

Causes:

- * High-producing, intensivly managed, dairy cattle
- * Abomasal atony
- * Increased gas production

(Influx of high concentrations of volatile fatty acids from the rumen and hypocalcemia may play a part in instigating hypomotility while evolution of gas in the abomasum is directly related to the amount of concentrate in the ration.)

* Postpartum



GASTRIC IMPACTION

<u>Horse</u>

Fibrous roughage and persimmons

İnadequate water intake

Poor mastication

Clinically

✓Anorexia

✓ Mild colic

✓ Loss of body condition

<u>Cattle</u>

Primary:

Restricted water intake and coarse High roughage feed Enceinte

Secondary:

- ✓ Pyloric stenosis (physical or functional)
- ✓ Vagus indigestion
- ✓ Adhesions of the abomasum and omasum
- ✓ Systemic diseases that causes abomasal stasis



RESULTS OF GASTRIC IMPACTION

- Peritonitis
- The laceration is near the omasal-abomasal orifice
- Compression necrosis
- Omasal dilation and ruminal distension



CIRCULATORY DISTURBANCES

Active hyperemia

- * Physiologic
- * Chemical substance
- * Acute gastritis

Passive hyperemia

*Portal hypertension

*Cirrhosis

*Shock





Congestion

Infectious diseases

Stress

Venous infarction

Gastric venous infarction is related to endothelial damage and thrombosis in venules, usually associated with

- Endotoxemia
- Bacterial or toxic damage
- Salmonellosis
- *♦ E. coli* septicemia

Gastric venous infarction is a common lesion in swine and seen in swine

- Coliform gastroenteritis
- >Erysipelas
- Dysentery
- Glasser's disease
- ≻Hog cholera





Edema of the gastric mucosa occurs with

- Hypoproteinemia
- Portal hypertension
- Cattle poisoned by arsenic
- Gut edema of swine





☑Chemical gastritis (abomasitis)

☑Uremic gastritis

☑Braxy (bradsot)

✓Chronic gastritis

⊡Helicobacter gastritis

☑Chronic atrophic gastritis

- * Chronic diffuse gastritis
- * Chronic antritis



Chronic hypertrophic gastritis

Chronic hypertrophic pyloric gastropathy

✓Hypertrophic antritis

✓Eosinophilic gastroenteritis

Mycotic gastritis

Dogs

✓Mycotic abomasitis

☑Abomasitis associated with viral infection



CHEMICAL GASTRITIS

- Chemical gastritis or abomasitis, reflected in
 - diffuse gastric congestion,
 - hemorrhage,
 - necrosis,
 - ulceration

It may be induced by chemicals such as arsenic, thallium, formalin, bronopol, steroidal and nonsteroidal antiinflammatory drugs (NSAIDs), phosphatic fertilizers, and by the toxic principle in bitterweed





- An <u>acute abomasitis of</u>
 - sheep and, less commonly, calves,
- caused by infection with Clostridium septicum.
- It occurs in cold areas and in winter.
- <u>The entry of the agent</u> into the mucosa may be due to the **ingestion of frozen** grass and feed.
- Cold weather is usually associated with the disease,
- Production of exotoxin by *C. septicum* causes the signs and death quickly.



BRADSOT

- At necropsy, there may be **Blood-tinged abdominal fluid**
- The serosa of the Abomasum may be congested or fibrincovered.
- Mucosal lesions may be diffuse or demarcated foci of variable size and shape
- Abomasal folds may be thickened, reddened, and occasionally hemorrhagic or necrotic.
- Most notable is the presence of extensive gelatinous edema and emphysema in the submucosa.
- Gram-positive bacilli are usually evident as individuals or colonies in affected tissue.



UREMIC GASTRITIS

- It is caused by uremia. Uremia occurs due to the renal failure.
- Uremia means high level of urea in the blood. It make vasculitis. Vasculitis leads to both gastritis and stomatitis.
- By the time urea converts to <u>amonia by the bacterial flora of the mounth</u>. Also it makes stomatitis.
- Urea 📩 amonnia
- At the same time, Metabolism of calcium disturbance leads mineralizaiton in gastric.
- Gastrin increases in blood because of kidney failure.



Also, gastrin hormon increase and then acidity increase and make gastritis.



UREMIC GASTRITIS

- Result of chronic renal disease
- Congestion and edema of the gastric mucosa caused by injury to capillaries within the lamina propria associated with elevated concentrations of nitrogen-derived metabolic waste products in the systemic circulation from kidney failure. (Fig. A)
- Mineralization of the glands, vessels, and lamina propria of the gastric mucosa(grossly visible as fine white stippling and lines in the mucosa (Fig. B).



• Ulcer formation.





The pathogenesis of peptic ulcer

(imbalance between the necrotizing effects of gastric acid and pepsin)

HYPERSECRETION OF ACID



Gastrinomas (Zollinger-Ellison Syndrome)

MastocytomaMastocytosis

Increased histamine levels



ULCERATION DUE TO COMPROMISE OF MUCOSAL PROTECTIVE MECHANISMS

- Nonsteroidal antiinflammatory agents (Aspirin, phenylbutazone, indomethacin)
 - Direct toxic effect
 - >Suppression of prostaglandin synthesis
- Helicobacter pylori (in humans)
- Reflux of duodenal contents (containing bile salts)
- Glucocorticoids
- Stress
 - >Reduced mucosal perfusion
 - ≻Ischemia



THE CAUSES OF ULCERS



✓Mastocytoma

✓Mastocytosis

✓Zollinger-Ellison Syndrome

Administration of glucocorticoids and non-steroidal antiinflammatory drugs in high doses

✓Trauma

✓Surgery

✓Damage to the spinal cord



CATTLE

⊠In veal calves, dairy cattle, feeding cattle

- Stressful circumstances
 - Weaned and veal calves
 - × Postparturient cows
- 🗵 Abomasal displacement
- 🗵 Mastitis
- Transportation
- High concentrate rations
- **Eymphosarcoma**
- **Arsenic poisoning**
- 🗷 Rinderpest, Malignant catarrhal fever
- 🗵 Mucosal disease, Theileriosis





Different diseases

Intestinal diseases

Colic

Surgery

Stressful circumstances

Candida spp.

Clostridium botulinum type B

Clostridium perfringens



PARASITIC DISEASES OF STOMACH AND ABOMASUM Horse





DOG-CAT

- Gnasthostoma spinigerum (NODULE)
- Physaloptera preputialis
- P. rara
- P. canis
- Cyathospirura
- Cylicospirura felineus (NODULE)
- Ollulanus tricuspis
- Capillaria putorii
- Cryptosporidium



CATTLE-SHEEP-GOAT

- Ostertagia ostertagi- cattle
- O. circumcinctata
- O. trifurcata
- *O. lyrata* cattle
- O. leptospicularis cattle-sheep-goat
- Haemonchus contortus sheep-goat
- *H. placei* ---cattle
- Marshallagia marshalli
- Mecistocirrus digitatus
- Camelostrongylus mentulatus
- Trichostrongylus axei (Nodule)
- Cryptosporidium

OSTERTAGIOSIS (NODULE)

HAEMONCHOSIS



GASTRIC NEOPLASIA

• <u>Dog</u>

* Adenocarcinoma

Cat

* Adenocarcinoma

* Lymphoma

• <u>Horse</u>

* Squamous cell carcinoma

Cattle

* Lymphoma







CONGENITAL ANOMALIES of the INTESTINE I

Segmental Anomalies

• **Stenosis** (incomplete occlusion or narrowing of the lumen)

- Atresia (complete occlusion of the lumen)
 - ✓Atresia coli
 - ✓Atresia ilei
 - ✓Atresia jejuni
 - ✓Atresia ani
 - Atresia ani and recti

Atresia coli and Atresia ani are the most common anomaly

Atreasia coli is seen particularly in the spiral colon of Holstein calves, and in the large and small colon of Foals While Atresia ani is most often encountered in <u>calves and pigs</u>, in which it is considered to be hereditary. **CONGENITAL ANOMALIES of the INTESTINE II**

Short colon (cats and dogs)

Hypoplasia of the small intestine (foals)

Congenital colonic agangliosis (foals)

Persistent Meckel's diverticulum (swine and horses)

Intestinal diverticula



PERSISTENT MECKEL'S DIVERTICULUM

- Occurs anomaly mostly along the antimesenteric border of the lower small bowel.
- Mainly in swine and horses.
- Derived from the omphalomesenteric (vitelline) duct, which is the stalk of the yolk sac.
- The vitelline membrane can also persist forming a fibrous ligament or *mesodiverticular band* between the distal small jejunum and the diverticulum or umbilicus.

In summary, the duct fails to regress and involute, which remains as a remnant of variable length and location



MISCELLANEOUS CONDITIONS OF THE INTESTINAL TRACT

- Intestinal Lipofuscinosis
- Muscular hypertrophy of the ileum (swine and horses)
- Diverticulosis of the small intestine
- Intestinal emphysema in pigs
- Rectal prolapse (swine, sheep and cattle)



INTESTINAL OBSTRUCTION

 Intestinal obstruction may be the sequel to a physical blockage of the lumen resulting from stenosis (narrowing, stricture) caused by an <u>intrinsic lesion</u> *involving the intestinal wall*, obturation (occlusion) by an intraluminal mass, or <u>extrinsic compression</u>.

FUNCTIONAL OBSTRUCTION





Segmental congenital anomalies of the intestine (stenosis and atresia)

- Acquired stenosis (intramural abscesses, primary neoplasms and scarring following ulceration)
- Foreign bodies
- Enteroliths*, phytobezoars*, trichobezoars*
- Parasites
- Impaction of the colon, by feces in dogs and cats
- Impaction of the ileum , by feces in horses
- Impaction of the cecum or colon in horses

Gravel



Enteroliths (mineral concretions) were common <u>in the colon of horses</u>.

Mineral salts are deposited in concentric lamellae around a central *nidus*—a foreign body such as a nail, wire, stone, or particle of feed Phytobezoars or fiber balls consist largely of plant fibers intermixed with phosphate salts, may be found <u>especially</u> <u>in the colon of horses</u>

Hairballs (trichobezoars) sometimes occur in dogs, cats, and ruminants; in ruminants they occur mostly in the forestomachs.



- TUMORS involve the intestine can make obstruction from outside the lumen, for example a tumor of pancreas.
- ADHESIONS are also common reason that make obstruction. In these situation, obstruction develops gradually as fibrous tissue contracts and adheres the bowel to itself or other abdominal structures.
- ABDOMINAL FAT NECROSIS
- PEDICLES OF SOME TUMORS
- INCARCERATION IN HERNIAS



<u>3. FUNCTIONAL OBSTRUCTION</u>

Failure of the intestinal circular smooth muscle to contract blocks the peristaltic wave, causing **functional obstruction**, a clinical syndrome of <u>pseudoobstruction</u> in which there is no physical occlusion of the lumen of the impacted intestine.

- Paralytic ileus frequently follows abdominal surgery, especially when the intestines are handled roughly or traumatized. It also is associated <u>with peritoneal irritation of any cause</u>, <u>especially peritonitis</u>. Intestinal paralysis resulting from complete obstruction of the intestines is called **ileus**.
- Pseudo-obstruction (neuromuscular dysfunction) a clinical syndrome described mostly in dogs, in which there is no physical occlusion of the lumen of an impacted intestine, may result from segmental or diffuse neuromuscular dysfunction in the gut.
- Ganglioneuritis or neuronal hypocellularity
- Megacolon in Clydesdale foals-hypoganglionosis of the myenteric plexus
- Grass sickness in horses
- Feline dysautonomia or *Key-Gaskell* syndrome
- Intrinsic disease of intestinal smooth muscle (syndrome of intestinal sclerosis)



CLINICAL SYMPTOMS AND CIRCUMSTANCES LEADING TO DEATH IN INTESTINAL OBSTRUCTION

Acute shock

Electrolyte imbalance

- Endotoxemia
- Dehydration

- Gastric and intestinal rupture
- Disseminated
- Paralytic ileus

- Tympany
- Ischemia
- Autointoxication



DISPLACEMENTS OF THE INTESTINES

<u>1. EVENTRATION</u>

Congenital

Displacement of a portion of the gut, usually the small intestine, outside the abdominal cavity.

Schistosomus reflexus Patent umbilicus Congenital diaphragmatic hernia

Acquired (trauma)



2. CECAL AND COLONIC DILATION, TYMPANY AND TORSION

>In ruminants, cecal dilation and torsion

- ✓Occurs in animals fed High-concentrate rations
- \checkmark Has been associated Late gestation and ileus from other causes



An increase in the concentration of dissociated volatile fatty acids, especially butyric acid, causes atony of the cecum, and dilation follows.

Motility is reduced.

If physical obtruction and ischemia are seen at the time in intestines then called strangulation obstruction



In horses, cecal and colonic tympany

✓ Readily fermentable carbohydrate

<u>CLINICALLY</u>

- Severe abdominal distension
- Compression of intra-abdominal organs
- Reduced cardiac return due to postcaval compression
- Reduced respiratory capacity due to compression of the diaphragm
- Severe pain
- Hypovolemia, acidosis, large bowel ruptures
- Laminitis (in recovered horses)



<u>3. DISPLACEMENTS OF THE EQUINE COLON</u>

Right dorsal displacement of the colon

Left dorsal displacement of the colon

Colonic torsion and volvulus

