Pathology of Alimentary system

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- The digestive system is one of the largest organ systems in the body; it includes the alimentary tract, salivary glands, tonsils, liver, pancreas, and peritoneum.
- This system is involved, either directly or indirectly, in most of the diseases that affect animals.
- In fact, any well transcribed clinical record will include a large component of information relative to its status; i.e., feed consumption, water intake, appearance of the oral mucous membranes, bowel movements, consistency of the feces, even Body temperature etc..

- Alimentary disorders are very frequent in animals.
- Normal gross appearance: Both the mucosal & viscera (serosal/ abdominal cavity) surfaces are Smooth and shiny.
- Diagnostic procedures include: Clinical exam (endoscopy, U/S, laparoscopy), Biopsy, Fecal exam, Necropsy & histopathology and Other lab tests.
- Signs of GIT Disease: Dysphagia (difficulty of swallowing), Vomiting, Diarrhea, Weight loss, Abdominal pain, Melena (the passage of dark tarry stools), and Suboptimal performance

Objective

• At the end of this session students will able to determine pathological conditions affecting the mouth, pharynx, salivary glands, oesophagus and crop.

Pathology of Oral Cavity and Associated Structures

1. Inflammation of The Oral Cavity

- Stomatitis refers to diffuse inflammation of the oral mucosa and includes
 - Glossitis: inflammation of the lingual mucosa,
 - Lampas: inflammation of the hard palate,
 - Gingivitis: inflammation of the gums
 - Chilitis: inflammation of the lips.
 - Pharyngitis: inflammation of the pharynx, and
 - Tonsillitis: Inflammation of the tonsils.

- Stomatitis may be **caused** by physical agents (foreign bodies, maloccluded teeth, hot water, etc.), Chemical Agents (acid, alkali, etc.), Bacterial Agents (Fusobacterium necrophorum, etc.), Viral Agents (vesicular exanthema, bovine virus diarrhea, malignant catarrhal fever, etc.), and Fungi (usually Monilia spp.).
- Clinically, characterized by partial or complete loss of appetite, chewing movements and smacking of the lips, profuse salivation and slow and painful mastication.

- Morphological changes of stomataitis are Macules (redness), Papules, Vesicles (blisters), Erosions, Ulcers, Necrosis with pseudomembranes, Granulomas.
 - ❖ Macule: Circumscribed lesion, 1 cm or smaller in diameter, characterized by flatness and distinguished by coloration (white, black, red, etc.)
 - ❖ Vesicle: Fluid-filled raised lesion 1 cm or less across (Bulla is greater than 1 cm. Blister is the common term for either)

Types of Stomatitis

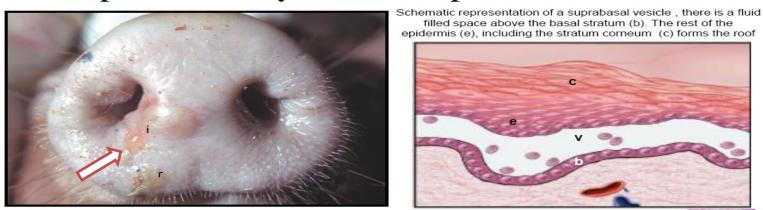
A. Vesicular stomatitides

- Seen in Foot and mouth disease, infectious vesicular exanthema and infectious vesicular stomatitis.
- Vesicles, blebs or blisters containing clear fluid are formed on the mucosa.
- Rupture of the blisters results in the formation of erosions, which subsequently heal.
- Catarrhal and vesicular stomatitis may develop into ulcerative variety.

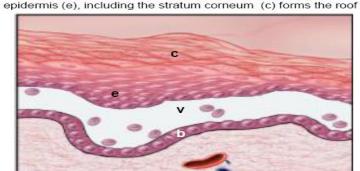
Pathogenesis of vesicular stomatitides

• Epithelial damage (viral) → intracellular edema (ballooning degeneration) \rightarrow vesicles \rightarrow bullae \rightarrow erosions \rightarrow ulcers \rightarrow cellular infiltration

• Lesions present in any stratified epithelium



Intact (i) & ruptured (r) vesicles, pig, Vesicular exanthema.

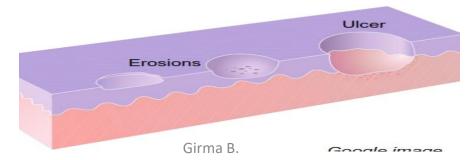


filled space above the basal stratum (b). The rest of the



B. Erosive & Ulcerative Stomatitides

- Erosion: Discontinuity of a body surface due to partial loss of surface epithelium
- Ulcer: Full-thickness epithelial loss revealing the underlying submucosa
- **Pathogenesis:** Epithelial necrosis & inflammation without vesiculation
 - Lesions in stratified squamous epithelium of mouth (may extend to esophagus and forestomach)



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Erosive & Ulcerative Stomatitides

Sharp, discrete erosions & ulcers, BVD

Some specific diseases/conditions:

- BVD/MD
- MCF
- Rinderpest
- Peste des petits ruminants
- Bluetongue
- Herpesvirus in cats, horses, primates
- Feline calicivirus
- Uremia



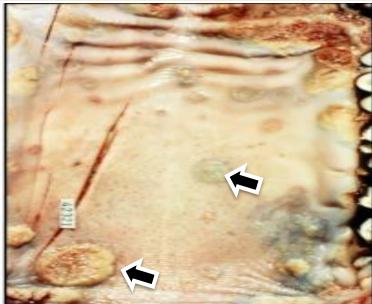


C. Papular Stomatitides

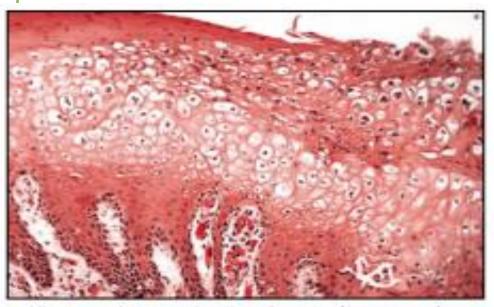
- Caused by parapoxviruses (Contagious ecthyma & Bovine papularstomatitis)
- Seen Mainly in young animals
- Papule: Elevated dome-shaped or flat-topped lesion 1
 cm or less across
- Pathogenesis: Epithelial degeneration, hyperplasia and inflammation → papule formation → ulceration & slow healing

Contagious ecthyma (scabby mouth

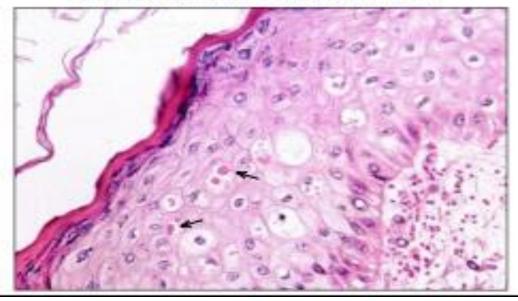




Coin-shaped papules & ulcers in papular 4/22/2020 stomatitis, cow



Ballooning degeneration & Intracytoplasmaic Inclusion bodies (arrows) in papular stomatitis, cow



Girma B.

D. Necrotizing (deep) Stomatitides

- Often secondary to erosions/ulcers and trauma
- Opportunistic bacteria penetrate mucosa and invade deeper tissues
- Ulcers/erosions are often covered by yellow-grey pseudomembranes
- Results in chronic inflammation (abscess, granuloma or cellulitis)

Eg. Calves (Calf diphteria) & pigs (Fusobacterium necrophorum)

2. Oral cavity Neoplasm

- A wide variety of neoplasms may arise from tissues of the mouth and related structures.
 - In dogs, viral oral papilloma occur most frequently.
 They tend to disappear spontaneously in 1-3 months if not removed.
 - Squamous cell carcinomas frequently occur in cats, dogs, cattle, and horses.
 - Fibrosarcomas are the most common sarcoma of the oral cavity. Osteogenic sarcomas may arise in the alveolar processes of the mandible and maxilla.

3. Developmental Abnormalities

a) Palatoschisis (cleft palate): direct communication between nasal and oral cavity.

Causes:

- Genetic (Charolais, along with arthrogryposis)
- Toxic plants in cattle, sheep & pigs
- Steroid administration (primates)
- > Results in starvation or aspiration pneumonia & death

Palatoschisis (cleft palate), a direct communication between the oral and nasal cavities, bovine





Palatoschisis (cleft palate), pig



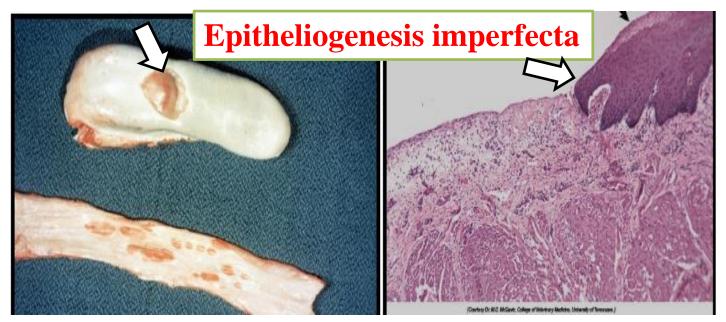
Arthrogryposis, piglet

b) Cheiloschisis (cleft lip or hare lip)

- Failure of fusion of the upper lip along the midline or philtrum
- May be uni-or bilateral, superficial or extend into the nostril
- Usually of no clinical significance

c) Epitheliogenesis imperfecta

- Incomplete development of the stratified squamous epithelium of skin, adnexa, and/or oral mucosa.
- May lead to bacterial infection or dehydration





Dental Caries:

- Caries refer destructive decalcification of the dental enamel which is followed by destruction of underlying tissues.
- Caries usually develop when the normal alkaline condition of the mouth changes to acid.
- After the **pH change**, anaerobic streptococci which are found in the mouth of all animals, attack **sugars** and **CHO**, resulting in lactic acid production.
- The lactic acid accumulates around teeth resulting in decalcification.

• A developing carie usually presents the appearance of a "black cavity" in the tooth. Eventually, the entire tooth turns black or a dark color.





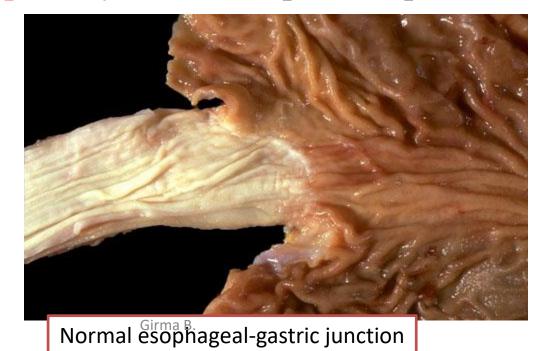
Pathology of Salivary Glands

- **Ptyalism (sialosis):** refers to excessive secretion of saliva resulting in an abnormal accumulation in the mouth.
 - it is associated with stomatitis, encephalitis, heavy metal poisoning.
- Ranula: refers to cystic dilatation and an accumulation of saliva in the salivary duct of the sublingual region.
- Mucocele: refers to an accumulation of salivary secretion adjacent to salivary ducts.
- Sialolithus: refers to calculi or concretions that form in salivary glands.
- Sialoadenitis: refers to inflammation of the salivary glands.

Pathology of Esophagus

Clinical consequences of diseases of esophagus:

- Manifested by regurgitation, dysphagia, multiple swallowing attempts & excessive salivation
- May lead to respiratory disease (aspiration pneumonia)

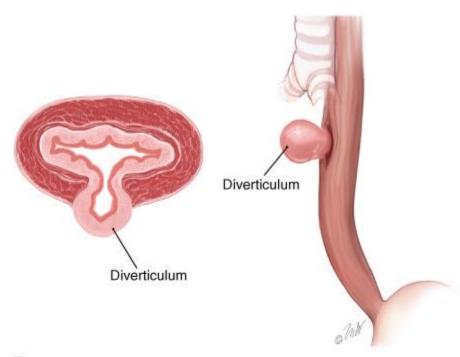


1. Inflammation of The Esophagus:

- Inflammation of the esophagus is referred to as esophagitis.
- Caused by Trauma (foreign bodies, etc.), ingestion of caustic chemicals (lye, etc.) and infectious diseases (papular stomatitis, malignant catarrhal fever, etc).

2. Dilation of the Esophagus:

- Esophageal dilatation (Esophagectasia) refers to an increased caliber of the lumen.
- The condition may be either localized, generalized, congenital or acquired.
- Remember, dilatations should be differentiated from diverticula of the esophagus.
 - Dilatations involve the entire circumference of the affected portion; whereas,
 - Diverticula involve only a portion of the circumference.



В

Source: Sugarbaker DJ, Bueno R, Krasna MJ, Mentzer SJ, Zellos L: *Adult Chest Surgery:* http://www.accesssurgery.com

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- Acquired dilatations may be seen in any animal species.
 - The dilated segments are located proximal to the point of obstruction (stenosis, foreign bodies, etc.).
- Megaesophagus refers to a diffuse (non-localized) dilatation of the esophagus.
 - This condition frequently occurs in puppies.

1. Megaesophagus

- Dilation due to insufficient/ uncoordinated peristalsis
- Often no microscopic lesions
- Congenital form
 - Vascular ring
 - Idiopathic





Congenital
megaesophagus, dogs.
The dilated portion is cranial
to the heart and is due to
persistent right 4th aortic
arch.

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3. Esophageal Rupture and Chock:

• Esophageal rupture may be a consequence of impaction, foreign bodies, external penetrating wounds, neoplasms or improper instrumentation.

 Ruptures at any location within the esophagus usually prove fatal because material contaminated with bacteria can gain entry into the fascial planes of the neck resulting in phlegmon or gangrene. 2 & 3. Esophageal rupture and obstruction (choke)

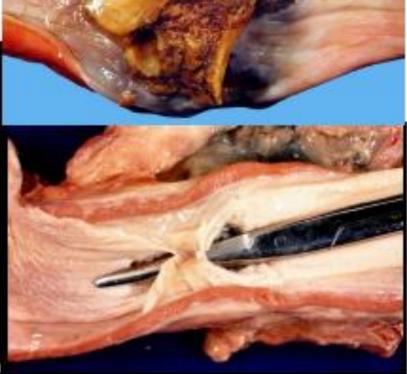
Obstruction sites:

- Larynx
- Thoracic inlet
- Base of the heart
- Diaphragmatic hiatus

Sequelae:

- Cellulitis
- Perforation
- Stenosis*
- Pleuritis/ pyothorax
- Pneumonia
- Bloat
- * Narrowing of the esophageal lumen

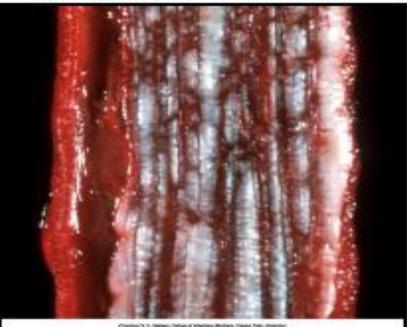




Examples of foreign bodies causing choke, ulceration (left), and perforation right top), dogs. If the animal survives, healing is by fibrosis, which may lead to stenosis, horse (bottom right)

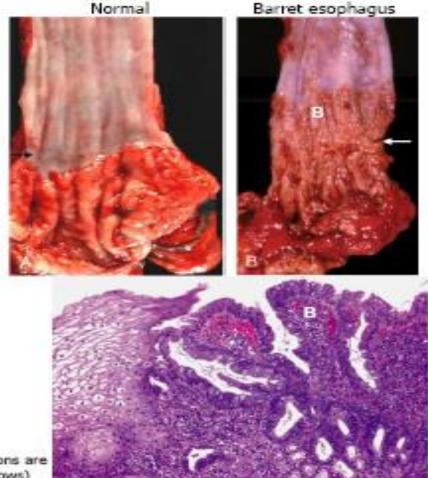
4. Reflux esophagitis

 Chemical damage of the squamous epithelium due to repeated gastric acid reflux.



Acid reflux esophagitis, horse. The dark red streaks are areas of epithelial loss secondary to gastric acid reflux. The white streaks are areas of unaffected mucosal epithelium.

Barret esophagus (B= intestinal metaplasia human), right. Lesions are most severe on the esophageal mucosa adjacent to the cardia (arrows) It is a complication of long-standing gastroesophageal reflux in humans



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Barrett Esophagus:

Replacement of squamous epithelium by columnar epithelium with goblet cells

Complication of long-standing reflux esophagitis

5. Parasitic lesions

- Sarcocystosis (Sarcocystis sp)
- Spirocerca lupi
- Gongylonema spp
- Gasterophilus & Hypoderma spp – Fly larvae



Sarcocystosis, esophagus, duck. Note the pale elongated nodules within the esophageal muscle.



Charrier Co. (6.) Michael Charge of Hospitals Robotics Charriery of Sections

Gongylonemiasis, esophagus, cow. Note The serpentine nematodes underneath the mucosa

6. Neoplasms of The Esophagus:

- Neoplasms of the esophagus are uncommon.
- However, neoplasms of epithelial and connective tissue origins have been reported.
 - Sarcoma as associated with S. lupi
 - Papilloma (commen in esophagus)
 - Papilloma virus
 - Squamous cell carcinoma
 - Pteridium aquilinum(Bracken fern)
 - Lymphoma

Pathology of the Crop of Birds

1. Inflammation of crop

- Inflammation of crop is called **ingluvitis**.
- a. Acute catarrhal ingluvitis
- Caused by Trauma by foreign bodies, Chemical agents (phosphorus, fertilizers), Toxins from decomposed food, Infectious diseases.
 - Parasites Acuaria sp; Capillaria sp
- Grossly the Lesions include cogestion, edema and tympanites.
- b. Diphtheritic ingluvitis: This is found in fowl pox.

2. Obstruction of Crop In Birds

Aetiology

- Ingestion of large quantities of dry grain which swell in the crop and form a hard mass.
- Atony or paralysis of wall leading to stasis of food.
- Foreign bodies like wire etc.

Grossly

 The stagnated food gets decomposed, gas accumulates and inflammation sets in.

Sequelae

- Rupture due to distention by food and gas or due to penetration by the foreign body.
- Death because of
 - Heart failure due to pressure on heart.
 - Asphyxia due to compression of trachea.
 - Intoxication due to absorption of toxins from decomposed foods.
 - Starvation, since, food does not enter the proventriculus.

Pathology of the Fore stomachs of Ruminants

- The forestomachs of ruminants are closely associated anatomically and functionally and disease of one usually affects the others.
- The two major functions of the forestomach are:
 - Bacterial digestion and fermentation and
 - Physical maceration by contraction of the stomach walls.
- Diseases of forestomach: Most are related to management practices

1. Simple Indigestion:

- Simple indigestion is characterized by anorexia, lack of ruminal movement and constipation.
- It develops subsequent to atony of the forestomachs.
- It is common in dairy cattle and stall-fed beef cattle because of the variability in quality and the large amounts of food consumed.
- Atony of the forestomachs is commonly related to abnormalities in the dietary intake, such as indigestible roughage, mold and overheated or frosted feeds.
- Simple indigestion is not usually a fatal disease.

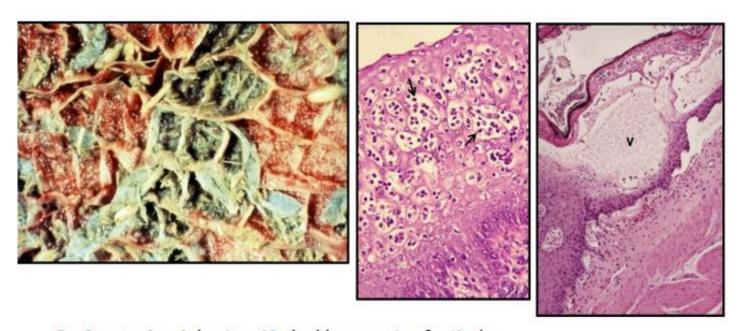
2. Carbohydrate Engorgement:

- Crbohydrate engorgement in ruminants develops following the ingestion of large amounts of highly fermentable carbohydrate rich feed.
- Clinically, the disease is characterized by severe toxemia, ruminal stasis, dehydration, weakness, recumbency and a high mortality rate.
- Acute carbohydrate engorgement is also referred to as grain overload, rumen overload and acidosis.

Pathogenesis of grain overload

- Sudden change to high CHO diet → overgrowth of gram + bacteria → ↑ lactic & dissociated fatty acids → pH < 5 & ruminal atony:
 - Acidic pH → eliminates normal gram-negative bacteria
 - Acidic pH → damages mucosa → microvesicles
 - Organic acids → osmotic effect → fluids into rumen
- Sudden death from dehydration, acidosis & endotoxemia
- Survivors often have grave sequelae

At necropsy: the rumen is filled with foul-smelling, fermenting feed and acute passive congestion of the ruminal wall is prominent. A few petechial and ecchymotic hemorrhages may be found over the heart and serosal surfaces.
 Lesions of grain overload



Grain overload, bovine: Marked hyperemia of reticular mucosa (top left); moderate cellular infiltration (arrows) and vesiculation (v) of the mucosa (histo).

Sequelae of grain overload

• Bacterial rumenitis

- Arcanobacterium pyogenes
- Fusobacterium necrophorum
- Healed ulcers ("stellate scars")
- Liver abscesses
 - Often subclinical
 - May rupture into vena cava
 - fatal septic embolism
- Mycotic rumenitis
- Well demarcated, circular hemorrhagic infarcts
 - Can become systemic
 - placentitis & abortion

3. Ruminal tympany (Bloat)

- Over-distension of the rumen and reticulum with gases of fermentation.
- These gases may be present in the form of a persistent foam mixed with the rumen ingesta (frothy bloat) or in the form of free gas separated from the ingesta (free gas bloat).

3.1. Primary Ruminal Tympany (Frothy Bloat):

- It is dietary in origin and occurs in cattle on legume pasture and in feedlot cattle which are fed diets containing high levels of grain.
- Frothy bloat is due to the production of a stable foam which traps the normal gases of fermentation in the rumen.
- The small gas bubbles which have mixed with the rumen contents do not coalesce, **eructation cannot occur**, and pressure within the rumen increases.
- Leguminous bloat is due to the foaming qualities of the soluble leaf proteins in legumes and other forages frequently associated with bloat.

- Evidence suggests that primary ruminal tympany or frothy bloat develops when:
 - The ingesta contains foaming substances (especially soluble leaf proteins, feeding finely ground grain and large amounts of grain),
 - The pH of the rumen is suitable for the growth of encapsulated bacteria which produce extracellular polysaccharides (slime), and
 - Insufficient saliva to exert a buffering effect on the pH of rumen may promote the tendency for foam to develop.
 - salivation is insufficient either because of the failure of the diet to stimulate it or because of the individual salivary paucity of the animal.

Pathogenesis of primary bloat

- Ruminal fermentation & acid production → low pH (5-6)
- Legumes & CHO's → ↓salivary secretion → ↑viscosity
 of rumen contents → foam → physical blockage of the
 cardia

- Accumulation of gases → ↑ intra-abdominal pressure
- → diaphragmatic & abdominal vein compression → respiratory & circulatory failure → death

Pathogenesis of primary bloat

Pasture bloat

- Legumes -> release of chloroplast particles (containing soluble proteins) -> rumen microbes colonize particles & degrade proteins (insoluble proteins) -> gas bubbles get trapped among the particles & do not coalesce -> stable foam
- Organic acids (from legumes) + salivary bicarbonates → CO2

Feedlot bloat

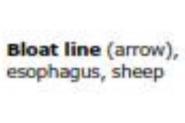
- CHO's (fine particles) & microbes which produce slime → it entraps gasses → stable foam
- CHO's → growth of encapsulated bacteria (↑ polysaccharides)
- → ↑viscosity of rumen contents → stable foam

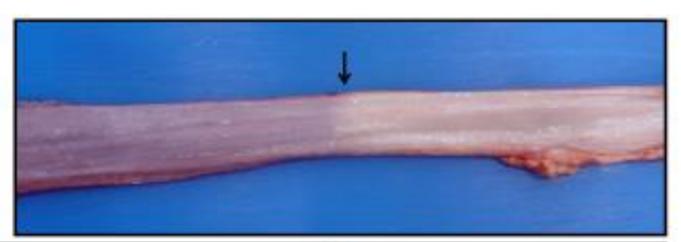
2. Secondary Ruminal Tympany (Free Gas Bloat):

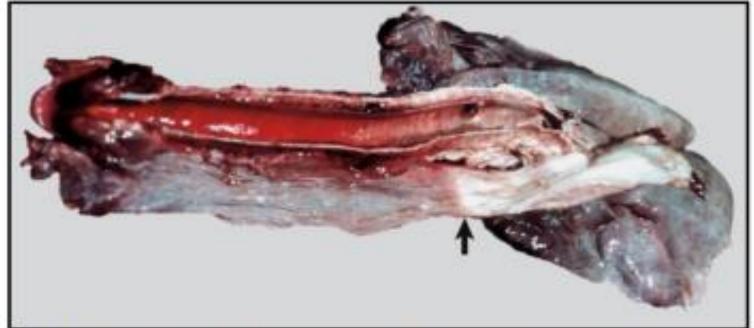
- Usually due to some physical obstruction which prevents the gases of normal fermentation from escaping via the esophagus (interference with eructation).
- Physical obstruction to eructation occurs in esophageal obstruction caused by foreign bodies, stenosis, pressure from enlargements outside the esophagus, etc.
- The free gas is usually present on top of the solid and fluid ruminal contents. Treatment in these cases consists of passage of a stomach tube or trocarization which results in the expulsion of large quantities of gas.

Signs & lesions in bloat

- Lesions of primary tympany are difficult to detect if there is time interval between death & postmortem (foams can collapse)
 - Abdominal distension
 - Animal found dead & rolled on back
 - Large volume of frothy rumen content
- Marked congestion of head, neck & hind limbs
- "Bloat line" is a sharp demarcation between pale bloodless caudal and congested cranial mucosa of the esophagus. This is diagnostic for bloat.







Bloat line, esophagus and trachea at the thoracic inlet, cow. There is a sharp demarcation between the caudal (white) and the cranial (congested) mucosa of the esophagus (arrow).

4. Foreign Bodies In the Rumen:

• It has been estimated that approximately **50%** of adult cattle have foreign bodies in the rumen and reticulum.

2. Foreign bodies

- 2.1 Trichobezoar (hair balls)
- 2.2 Phytobezoars (plant balls)
- 2.3 Lead substances → poisoning

2.4 Sharp metals → hardware disease (traumatic reticuloperitonitis or reticulopericarditis)



Trichobezoar, from stomach and duodenum, human.







• Impaction of The Rumen:

- Is a rumen which is firm and almost completely filled with very dry ingesta.
 - Atony is present and there is usually no fermentation or digestion occurring.

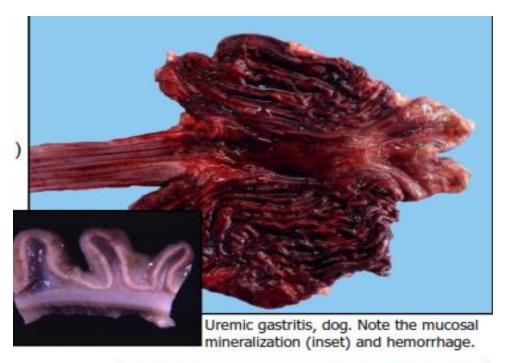
Postmortem Maceration of Ruminal Epithelium:

- Within a few hours after death, large patches of ruminal epithelium detach when the ingesta is disturbed.
- This postmortem change is referred to as maceration.

Pathology of the True Stomach

1. Inflammation of the Stomach:

- Inflammation of the monogastric stomach is referred to as Gastritis, whereas inflammation of the abomasum is commonly referred to as Abomasitis.
- **Grossly**: the mucosa is reddened and swollen and there is increased secretion of mucus.
- Microscopically: there is desquamation of the epithelium together with leukocytic infiltration.





Hypertrophy of the gastric mucosa (Hypertrophic gastropathy), dog. Note the prominent folds and redundant "cerebrifrom" mucosa.





hemorrhagic abomasitis

Noah's arkive

2. Acute Gastric dilation and volvulus (GDV)

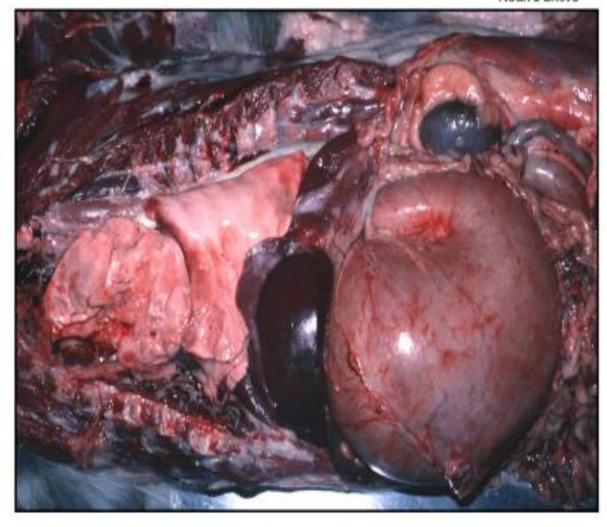
- Dilatation of the stomach can be caused by Failure of eructation and pyloric outflow by foreign bodies, gross overeating and drinking excessive amounts of water, etc.
- Mostly in large dog breeds, rarely horses and pigs.
- Subsequent to acute dilatation, rupture of the stomach may occur in the equine, but this is not common in other species.

Pathogenesis

- Follows large meal (dry or highly fermentable)
- Failure of normal eructation & pyloric outflow
- → Excess gas production → gastric dilation → functional obstruction of cardia & pylorus → torsion (rotation on its mesenteric axis)→compression of lung & posterior vena cava →circulatory collapse (shock) → death from respiratory & circulatory failure

Gross changes:

- Severe abdominal distension
- Clock-wise rotation of stomach
- Hemorrhagic infarction
- Rupture of stomach
- V-shaped bending of enlarged spleen
- Congestion of intestines



Gastric dilation and volvulus, dog. The stomach is filled with fluid and gas and the serosa is congested. The spleen is engorged, and displaced to the right and cranially.



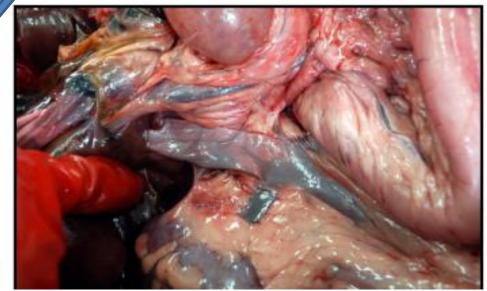


GDV, V-shaped bending of enlarged spleen

GDV, Stomach rotates on its mesenteric axis



GDV, compressed caudal vena cava (decreased venous return to the heart)



3. Displacement of The Abomasum

a) LDA mostly in dairy cows

- Common GIT disorder requiring surgery
- Caused by Feeding high grain diet (volatile fatty acid decrease motility)
- Occurs most commonly in large high producing adult dairy cows during late pregnancy or immediately after parturition.
- Abomasal atony is considered to be the primary dysfunction in this condition.
- Pathogenesis: The gravid uterus displace the rumen & abomasum cranially & to the left → rupture of attachment of greater omentum → LDA

b) RDA mostly in calves

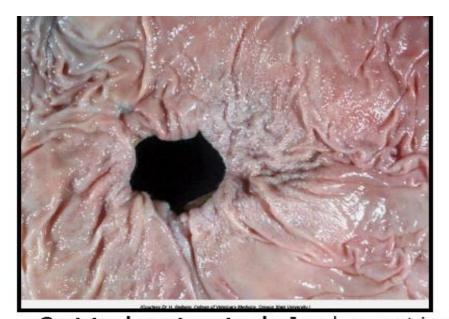
- Over distended abomasum → displaced
 dorsally (and to the right), → rotated on its
 mesenteric axis (torsion) →RDA.
- LDA & RDA results haemodynamic disturbance mainly congestion of the displaced abomasum and serosal congestion
 - if prolonged → Ischemic necrosis

4. Ulceration of the Stomach:

- Gastric ulcerations occur in all animal species, but they are most frequent in farm animals (cattle, swine and horses)
- less important than in humans
 - where they are usually traumatic or associated with
 erosive or ulcerative diseases (bovine virus diarrhea, etc.)
- Main signs: Hematemesis, Melena, Anemia, Abdominal pain.

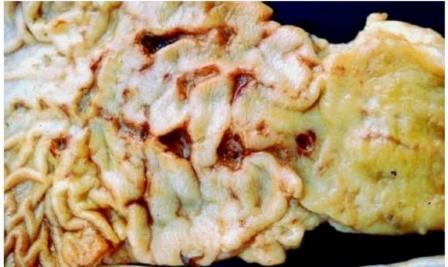
- Ulcers → central area of necrosis surrounded by active hyperemia & inflammation (red periphery).
- Epithelial necrosis → erosion → ulceration →
 bleeding → perforation → peritonitis

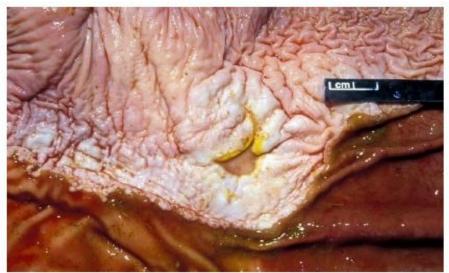






Gastric ulcers in animals. In calves, gastric ulcers may perforate (top left) and cause fatal peritonitis. In pigs, they occur as a oval or rectangular crater (top right), often as incidental finding, but could be fatal. In dogs, they may result from mast cell tumors (bottom left). In horses, lesions are often subclinical and affect the squamous mucosa (bottom right).





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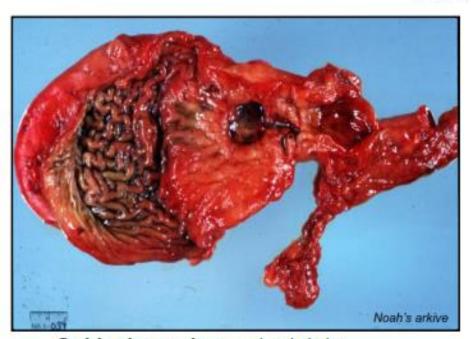
5. Neoplasms of The Stomach:

- Primary neoplasia is relatively uncommon in domestic animals.
- However, neoplasms with multicentric origin;
 e.g., lymphosarcoma, Squamous cell carcinoma (horses), Adenocarcinoma (dogs)
 have been noted to occur with more frequency.

Gastric leiomyoma, dog



Pathologic Basis of Veterinary Disease, 4thed., Mosby-Elsevier



Squamous cell carcinoma, stomach, equine

Gastric adenocarcinoma, ulcerated, dog

6. Parasitic diseases of stomach

1. Parasitic diseases

Ruminants

- Haemonchosis
- Ostertagiosis
- Trichostrongylosis

Equine

- Gastric bots
- Draschia megastoma
- Trichostrongylosis

Swine

Hyostrongylosis

Pathology of Small and Large Intestines

Structure of intestine

- Long coiled tube, large surface area
- Folded mucosa
- Villi (7-14 fold increase)
- Microvilli (15-40 fold increase)

Function of intestine

- Digestion, absorption, excretion
- Fermentation vat (cecum)
- Good defense mechanisms

Diseases of Small and Large Intestines

- Congenital anomalies
- Obstruction and functional disorders
- Inflammation
- Specific enteric diseases (mostly infectious)
- Neoplastic diseases

1. Congenital anomalies of intestine

Segmental defects

- Stenosis (partial occlusion)
- Atresia (complete occlusion)
 - Atresia ani, atresia coli, atresia ilei

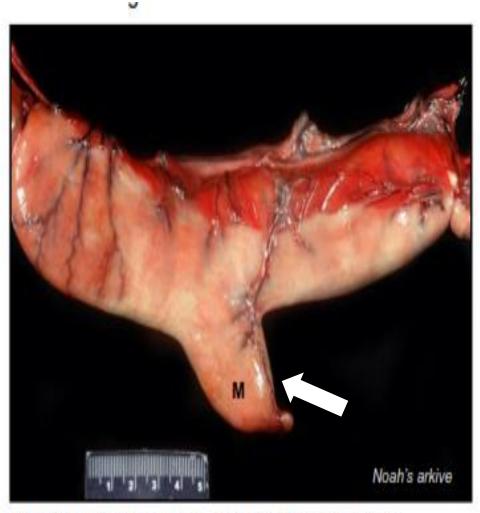
• Persistent Meckel's diverticulum (Out pouching of a hollow)

Derived from omphalomesenteric duct (stalk of the yolk sac)

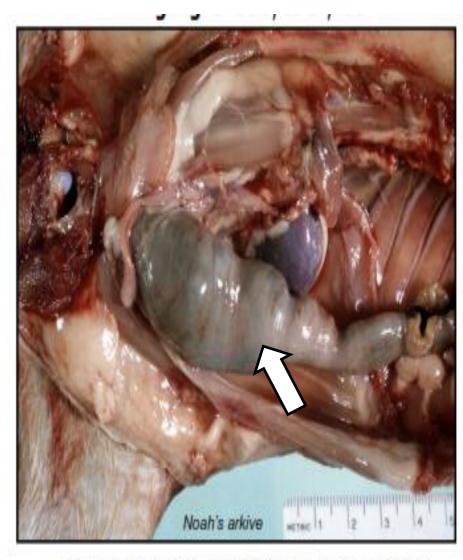
Megacolon

- Reduction/abscence of ganglion cells of myenteric plexus (aganglionosis)
 - 2ary to atresia ani
 - Damage to the colonic innervation

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Meckel diverticulum. The blind pouch (M) is located on the antimesenteric side of the small bowel.



Megacolon, cat. Fecal-filled, enlarged colon

2. Obstruction and functional disorders

- Can be caused by Obturation (intraluminal),
 Compression (external), Stenosis (strictures or narrowing), Intestinal displacement, Functional (absence of peristalsis).
- Consequences of obstruction is Death from
 - Toxemia (bacterial overgrowth),
 - Shock (dehydration, etc),
 - Starvation

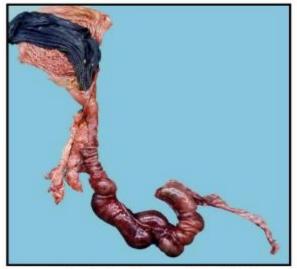
Gross lesions

- Distended abdomen
- Dilated bowel proximal to obstruction
- Collapsed and empty distal part
- Congested/infarcted area of obstruction



Foreign body (sock), accordion-folded intestine, dog

Perforation



Foreign body (sock), accordion-folded intestine, dog



Congenital stenosis, lamb

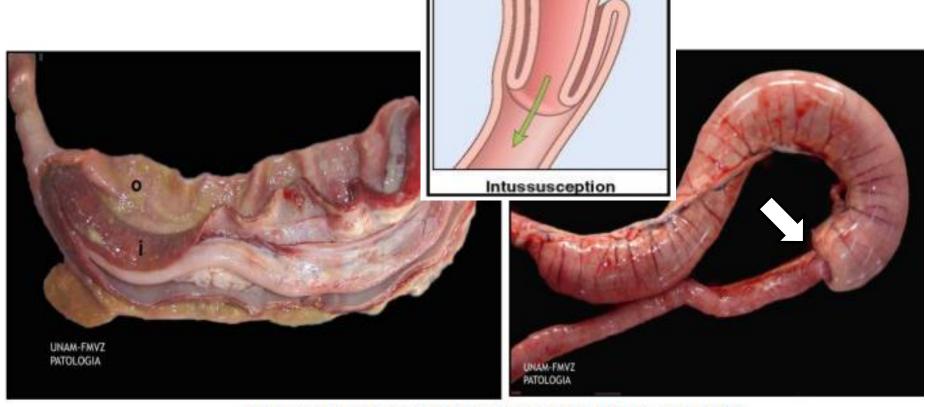


Enterolith, horse

3. Intussusception

- is Telescoping (entrapped or enveloping) segment
 - this means a portion of intestine enters in caudal segment due to violent peristaltic movement.
- Caused by Irritability/hypermotility
- It results obstruction, passive congestion and oedema.
- Pathogenesis:
 - Vascular strangulation → congestion/edema → ischemia → infarction → gangrene (necrosis or rotting)

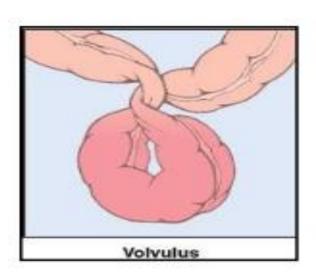
• Sharp line of demarcation between viable colon and nonviable colon caused by obstruction of venous blood flow frequently seen.

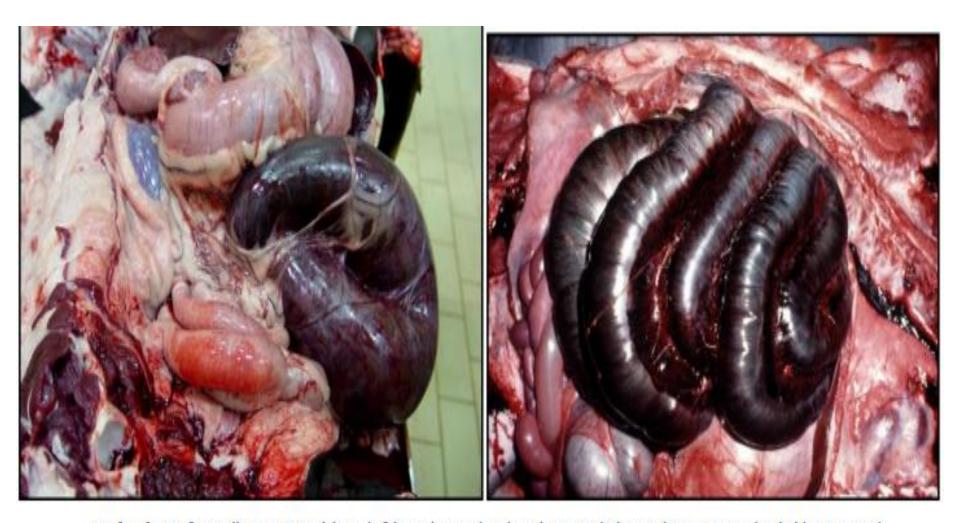


Intussusception, dogs. Intussusceptum (i), intussuscipiens (o)

4. Volvulus

- The loop of intestine passes through a tear in mesentery
- It causes obstruction at both ends of loop.
- Common Left colon in horses

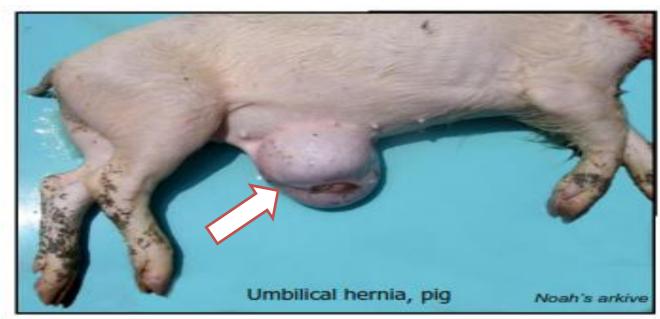


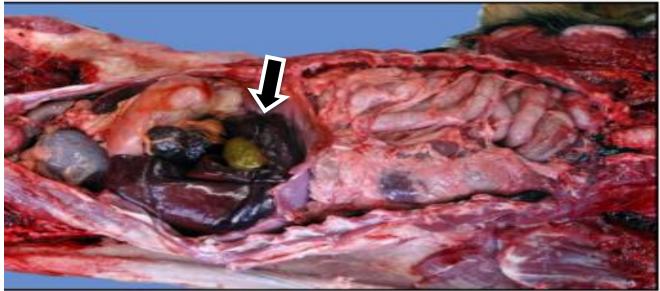


Volvulus of small intestine (dog, left) and spiral colon (pig, right) resulting in marked dilation and venous infarction.

5. Herniation

- is the protrusion of the abdominal viscera through a natural or artificial opening.
 - Displacement through a foramen
- It could be Internal (Foramen of Winsloe, Omental or mesenteric tears, Renosplenic ligament) and External (Diaphragmatic, Umbilical, Scrotal or femoral, ventral)
 - External hernia consists of a hernial sac, hernial ring and The hernial contents.
- which causes passive congestion, Strangulation (interference with blood flow), edema, obstruction in intestines and Perforation.

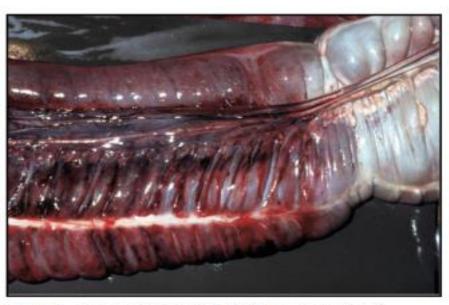




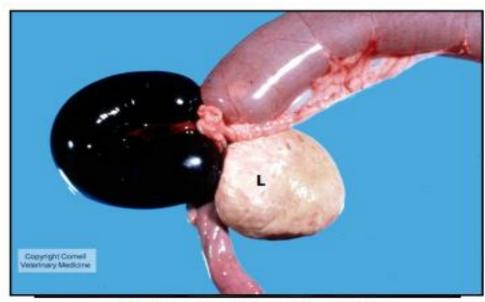
Herniation through the diaphragm, dog

6. Torsion

- Torsion is twisting of intestine upon itself or axis causing obstruction.
- common in Cecum
- Results → Obstruction and infarction → strangulation



Torsion, large colon, horse. Note the sharp line of demarcation between viable colon (to the right) and nonviable colon (to the left) caused by obstruction of venous blood flow.



Pedunculated lipoma, horse (L).

The tumor wrapped around the mesentery and strangled the bowel.

7. Inflammation of intestines

• It leads to

- Diarrhea

- An increase in feces mass, frequency, and/or fluidity
- Consequences of diarrhea are Dehydration, Acidosis,
 Hypoproteinemia & ascites, Electrolyte imbalance →
 Death

Dysentery

- Painful, bloody diarrhea
- Causes of enteritis are viruses, bacterias, Parasites, Dietary factors, Ingested intoxicants, Allergies and Idiopathic

• Types of enteritis: gross lesions

- Catarrhal (dark bloody)
- Hemorrhagic (bloody)
- Fibrinous/fibrinonecrotic
- Ulcerative (sore of moucous membrane)
- Proliferative/hyperplastic
- Granulomatous (mass of granular tissue)



Necrohemorrhagic enteritis, small intestine, horse. Necrosis and sloughing of the mucosa caused by the ingestion of cantharidin, a toxin in ingested blister beetles (*Epicauta* spp).



Microscopic features in enteritis

Infiltration of lamina propria

- Crypt necrosis & inflammation (cryptitis, crypt

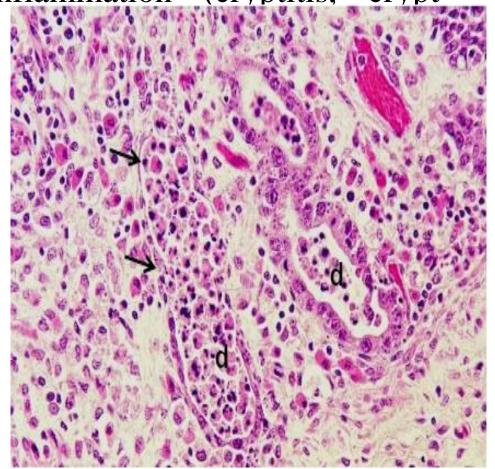
abscess)

Crypt dilation

Crypt hyperplasia

Necrosis of enterocytes

- Villous atrophy



8. Concretions/ Enteroliths

- Calculi found in the intestine are called enteroliths.
- Sometimes they are found in the large intestines of horses.
- When animals are fed on wheat or bran which are rich in magnesium phosphate, intestinal calculi can occur.
- Normally magnesium phosphate is dissolved by the gastric juice and then absorbed in the intestines.
- If an animal suffering from chronic catarrhal gastritis in which gastric juice is not secreted, much of the magnesium phosphate reaches the intestines in an undissolved state.

- This combines with ammonia that is formed from the decomposition of protein (which is also abundant in wheat and bran) to form triple phosphate.
- This triple phosphate crystalises around foreign bodies like a grain of sand, a piece of metal or undigested plant fibre.
- Enteroliths do not form in the small intestines because the movement of the food is too rapid there to allow the deposition of salts and formation of calculi and Bacterial decomposition of proteins to form ammonia does not take place there.

• Grossly: Enteroliths may sometimes attain a large size, some may weigh as much as 20 lbs and are usually round and smooth.



Enterolith, horse

Adhesion

- Intra-abdominal adhesions generally affect the small intestine and usually cause obstruction of the intestinal lumen, although they may cause strangulating obstruction.
- These adhesions develop in response to peritoneal injury and, most often, are the result of previous small-intestinal surgery, chronic small-intestinal distention, peritonitis, or larval parasite migration.
- The tissue response to ischemia, traumatic tissue handling, foreign material, hemorrhage, or dehydration results in the formation of fibrinous (and subsequently fibrous) adhesions.

2. Peritonitis

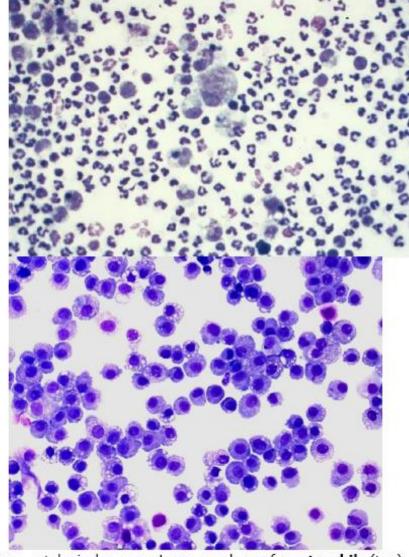
- Very common in large animals
- Usually bacterial, less often viral, parasitic
 & chemical (e.g. bile, pancreatic enzymes)
- Bacteremia/septicemia in young animals
 - Omphalitis or mucous membranes → infected monocytes with limited bactericidal power → serosal, synovial & meningeal surfaces

Classified as:

- · Primary or secondary
- Acute or chronic
- · Localized or diffuse
- · Septic or nonseptic
- · Type of predominant exudate

Sequellae:

Fibrous adhesions → intestinal obstruction



Microscopic images from cytological smears. Large numbers of **neutrophils** (top), **macrophages** and reactive **mesothelial cells** (the latter two look very similar, bottom) are usually present in pleural or peritoneal fluids associated with inflammatory processes.

