UREMIA

- Dogs and cats
- Chronic kidney failure
- Ulcerative stomatitis with foul odor
- Buccal mucosa and especially tongue dark cyanotic
- Ammonia formation and has a corrosive effect on the mucous membrane.
- Elevated urea levels in the blood and saliva create a tendency to bacterial infection, and urease-producing bacteria normally found in the oral microflora generate ammonia from the urea in saliva.

Uremic Ulcers, Hard Palate, Dog.
Ulcers present on the transverse palatine ridges and periodontal gingiva are secondary to vascular damage associated with increased concentrations of plasma blood urea nitrogen and creatinine from kidney failure. Affected animals often have an ammoniacal or uremic odor to the breath.

Brown DL, Van Wettere AJ, Cullen JM (2017) Alimentary system in: Pathologic Basis of Veterinary Disease, 6th edition, Zachary JF, ed., elsevier, St. louis, Missouri, pp. 345.

DEEP STOMATITIS

Lesions of the oral mucosa may permit **the entry of pyogenic bacteria**, often **normal oral flora**, into the **connective tissues** of the submucosa and **muscle**.

Purulent inflammation or **cellulitis** may develop in the lips, tongue, cheek, soft palate, and pharynx.

Abscesses may form and may **fistulate** through the mucosa or skin.

Usually caused by ***Fusobacterium necrophorum*** and other **anaerobes.**

DEEP STOMATITIS

- ✓**Oral Necrobacillosis**
- ✓Noma
- ✓**Actinobacillosis**
- ✓Oral dermatophilosis of cats

Oral Necrobacillosis

- ***Fusobacterium necrophorum***
- **Necrotizing lesions in the upper and lower alimentary tract, and liver.**
- Secondary invader following previous mucosal damage.
- Once established in a suitable focus, *F. necrophorum* proliferates, causing extensive coagulative necrosis.

- The best-known form of necrobacillary stomatitis is **calf diphtheria.**
- **Fatal** in **young** animals, in which **extension** often occurs to other organs.
- In **adults**, oral necrobacillosis tends to remain **localized** to the **oral cavity**, where it may complicate vesicular and ulcerative stomatitides.

Actinobacillosis

- It is a chronic suppurative inflammatory disease of ruminants,
- characterized by inflammation of soft tissue of the head especially tongue (localized firm swelling of dorsum), less commonly pharyngeal lymph nodes, facial skin, nares and esophageal groove.
- ***Actinobacillus lignieresii*** is part of the normal oral flora, and in cattle is associated with deep stomatitis.
- Typically a ***disease of soft tissue***, spreading as a ***lymphangitis*** and usually involving the regional lymph nodes.

Actinobacillosis

- The chronically occurring picture is clinically defined **as "wood tongue".**
- **Predisposing factor : Oral mucosa injuries** by fibrous feed materials or by foreign bodies and during oral manipulation by hand of owner or veterinarian.
- The lesion is a ***pyogranuloma,*** appearing grossly as a nodular, firm, pale, fibrous mass a few millimeters to 1 cm in diameter, containing in the center minute yellow **"sulfur"** granules-like.

Actinobacillosis

- Microscopically; **Finger like clubs or projections** abutting the bacterial colonies are surrounded by cellular granulation tissue consisting of epithelioid cells, a few giant cells and leucocytes.
- Fibroblasts form masses of fibrous tissue beyond these infiltrating inflammatory cells.
- The granulomatous growth of the tissue marked by inflammation and abscessation in this infection contains foci of pus having clubs radiating from the centres of the masses called **rosettes.**

Parasitic Diseases of The Oral Cavity

- ***Sarcosporidiosis and cysticercosis:*** striated muscles of the tongue
- ***Gongylonema spp.:*** Mucosal lining of the tongue
- ***Trichinella spiralis*** **:** Muscles of the tongue and mastication
- ***Gasterophilus spp.*** (horse) and ***Oestrus ovis*** (sheep) : Pharyngeal mucosa (focal ulceration and mild inflammation)
- ***Gasterophilus nasalis:*** Migrate from the lips and invade the gums around and between the teeth and behind alveolar process to cause small suppurating pockets.

Neoplastic and Like Lesions of The Oral Cavity

- Oral papillomatosis
- Squamous cell carcinomas
- Melanomas
- Fibrosarcomas
- Mast cell tumors
- Granular cell tumors
- Neuroendocrine carcinomas
- Plasmacytomas
- Vascular tumors
- Miscellaneous tumors

Diseases of Teeth and Dental Tissues

Developmental Anomalies of Teeth

- Anodontia (absence of teeth)
- Pseudoanodontia (Failure of the teeth to erupt from the gums)
- Oligodontia (fewer teeth than normal)
- Pseudo-oligodontia and Pseudopoliodontia result from failed eruption.
- Polyodontia (excessive teeth)
- Heterotopic polyodontia (an extra tooth, or teeth, outside the dental arcades)
- Odontogenic cysts (epithelium-lined cysts derived from epithelium associated with tooth development)
- Dentigerous cysts (cysts that contain part or all of a tooth.)

Degenerative Conditions of Teeth and Dental Tissue

- **Pigmentation of the teeth** (**chronic fluorosis**, pulpal hemorrhages or inflammation, putrid pulpitisler, icterus, congenital erythropoietic porphyria, tetracycline, impregnation of mineral salts with chlorophyll and porphyrin pigments from herbage, poisoning of lead)
- **Odontodystrophies** (Fluorine poisoning, Vitamin A-calcium-phosphorus deficiency)

Dental attrition (oligodontia, diastasis dentinum), wave mouth-step mouth, shear mouth

- It occurs due to chewing movements.
- Irregularities in dental attriations are among the most common dental disorders in herbivores.

- *Subnormal resistance* to wear on the part of the **molar teeth** is common, and *results in wave mouth* **(weave mouth)** or **step mouth**, in which *successive teeth in an arcade wear at different rates.*

- Due to insufficient lateral movements of the jaws, slopes are formed on the chewing surfaces of the **molar teeth.**
- *The cheek-facing parts of the* ***maxillary teeth*** *and the tongue-facing parts of the* ***mandibular teeth*** become **sharper.**
- The teeth wear progressively sharper, and can result in the teeth *passing each other like shear blades*; hence the term **shear mouth**.

ODONTODYSTROPHIES

- *Odontodystrophies are diseases of teeth caused by **nutritional, metabolic, and toxic insults.***
- The most prominent effects of odontodystrophies appear **in enamel** (irreparable).
- Harmful effects are seen **on ameloblasts** that are forming and mineralizing enamel.
- Enamel defects vary in severity from isolated opaque spots or pits on the surface to deep and irregular horizontal indentations.
- It is seen bilaterally and symmetrically in **incisor and canine teeth.**

Several nutritional and toxic conditions produce odontodystrophy.

Fluorine poisoning

- calcification of the enamel **x**, →leading to hypomineralization and hypoplasia.
- chalk-like areas
- loses its shiny and translucent appearance and turns into dull, white areas
- Dental attrition occurs due to softening of enamel and possibly dentin. Bone and kidney lesions are also formed in chronic fluoride poisoning

In **vitamin A deficiency,** ameloblasts do not differentiate normally, and their organizing ability is disturbed.

Calcium deficiency retards eruption and causes enamel hypoplasia and mild dentin hypoplasia

Phosphorus deficiency, combined with vitamin D deficiency, depresses dentin formation slightly

Infectious and Inflammatory Diseases of Teeth and Periodontium

- Supragingival plaque
- Subgingival plaque
- Dental calculus (tartar)
- Materia alba
- Dental caries
- Pulpitis
- Periodontal disease and gingivitis

  Subgingival plaque-gingivitis-gingival recession-loss of alveolar bone-chronic periodontitis-exfoliation of teeth

Neoplastic and Like Lesions of The Teeth

- EPULIDES
  - Gingival vascular hamartoma
  - Pyogenic granuloma
  - Giant cell epulis
  - Fibrous epulis
  - Fibromatous and ossifying epulis
  - Acanthomatous epulis
- SQUAMOUS CELL CARCINOMA

ODONTOGENIC TUMORS

- Ameloblastoma (Adamantinoma, enameloblastoma)
- Calcifying epithelial odontogenic tumor
- Ameloblastic fibroma (Fibroameloblastoma)
- Ameloblastic fibro-odontoma
- Complex odontomas
- Compound odontomas
- Odontoameloblastoma
- Odontogenic myxoma
- Cementoma

TONSILLITIS

TYPE	DISEASE
❑CATARRHAL+PURULENT	Gourme Distemper Hepatitis contagiosa canis Canine parvovirus infection
❑ CATARRHAL+NECROTIC	Rinderpest / BVD-MD
❑ NECROTIC	Necrobacillosis / Aujeszky
❑ DIPHTHEROID	Swine plaque Panleukopenia
❑ DIPHTHEROID+NECROTIC+HEMORRHAGIC	Anthrax (Swine) Pasteurellosis

Salivary Glands

- **Ptyalism** (increased secretion of saliva)
- **Aptyalism** (reduced or ceased secretion)
- **Salivary calculi** (sialoliths)
- **Dilations of the duct** (ranula)
- **Salivary mucocele** or **sialocele*** *is an accumulation of salivary secretions in single or multiloculated cavities, not lined by secretory epithelium, in the soft tissues of the mouth or neck.*
- **Sialoadenitis** (Rabies, Coryza gangrenosa bovum, Gourme, Distemper, vitamin A deficiency)

ESOPHAGUS

Anomalies of esophagus

- ✓Congenital duplication
- ✓Segmental aplasia
- ✓Esophageal atresia
- ✓Esophagorespiratory fistulae
- ✓Congenital esophageal diverticula
- ✓Epithelial inclusion cysts
- ✓Presence of papillae
- ✓Gastric heterotopia

- **Aplasia** is **a birth defect where an organ or tissue is wholly or largely absent**. It is caused by a defect in a developmental process.
- **Atresia** is a condition in which an orifice or passage in the body is (usually abnormally) closed or absent.

ESOPHAGITIS

- **Reflux esophagitis** occurs because of **a loss of functional integrity of the lower esophageal sphincter**.
- It can be due to **airway occlusion** and increased intra-abdominal pressure, the pharmacologic effects of preanesthetic agents, or abnormality of the hiatus.
- Esophagitis due to the action of **gastric acid, pepsin, and probably regurgitated bile salts and pancreatic enzymes, on the esophageal mucosa.**
- **Erosive and ulcerative esophagitis**
  - **Mucosal disease**
  - **Rinderpest**
  - **Malignant catarrhal fever**

Focal necrosis (**Bovine papular stomatitis, IBR, Peste des petits ruminants, bovine herpesvirus infection, calicivirus in cats**)

ESOPHAGEAL OBSTRUCTION, STENOSIS AND DILATIONS

Stenosis:
the abnormal narrowing of a passage in the body.

Obstruction:
the action of obstructing or the state of being obstructed.

Dilation:
is the action or condition of becoming or being made wider, larger, or more open.

Esophageal Obstruction and Stenosis

Esophageal obstruction can have **intrinsic or extrinsic causes**, and the lesions are generally obvious.

Intrinsic causes

- *Choke,* or esophageal impaction, is the most common example

(when large or inadequately chewed and lubricated foods, masses of grain or fibrous ingesta, or medically administered boluses lodge in the lumen)

Complications of obstruction include:

- *pressure necrosis and ulceration of the mucosa*
- perforation or
- esophageal diverticula or fistulae.
- **Fibrosis and scarring** of large ulcers may result in **narrowing lumen and stenosis.**
- Sharp objects, such as bones, are most likely to cause ***perforation.***

Esophageal Obstruction and Stenosis

Extrinsic causes

- ***Vascular ring anomalies:***

The most common causes of external compression and constriction of the esophagus are **vascular ring anomalies** seen in dogs.

- a persistent right aortic arch is present. However, other vascular anomalies such as an aberrant subclavian artery or a double aortic arch may be the cause of the esophageal constriction.
- ***Persistence of the right fourth aortic arch*** is the most common of these anomalies, and occurs when the right aortic arch develops instead of the normal left aortic arch.

Breeds commonly affected include the Great Danes, German Shepherds and Irish setters.

Regardless of the type of vascular anomaly, **the constriction prevents solid foods from passing to the stomach** and prevents the puppy from thriving well.

ESOPHAGEAL DIVERTICULA

- Irregular outpouchings or herniations of the esophageal mucosa through a defect in the esophageal tunica muscularis.

✓**Pulsion diverticulum**

✓**Traction diverticulum**

✓**Diverticula** is a blind tube leading from a cavity or passage.

ESOPHAGEAL DIVERTICULA

Pulsion diverticula

Increased intraluminal pressure associated with

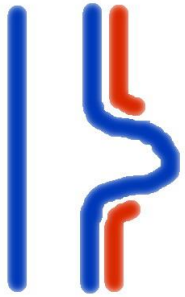
- **foreign bodies,**
- **Obstruction or stenosis**

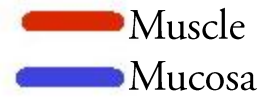
✓the mucosa is forced out through the distended or ruptured muscularis.

✓large spherical structures with a narrow neck

✓common in the horse and dog.

✓have a layer of epithelium lining the inner aspect of a wall of fibrous connective tissue,





Traction diverticulum

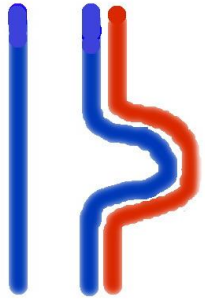
is the result of

- Contraction of a paraesophageal fibrous adhesion, following perforation and inflammation,

✓drawing with it a pouch of esophageal mucosa

✓small and inconsequential.

- Traction:have a wall comprised of all layers of the esophagus.



➢ **Megaesophagus** (Esophageal ectasia)

Dilation of the esophageal lumen, and is the result of atony and flaccidity of the esophageal muscle.

- ✓ **Congenital idiopathic megaesophagus (CIM)** (Great Danes, German Shepherds, Irish Setters)
- ✓ **Secondary megaesophagus**
  - ✓ Myastenia gravis
  - ✓ Administration of cholinesterase inhibitors
  - ✓ Hypoadrenocorticism
  - ✓ Giant cell axonopathy in dogs
  - ✓ Immun mediated polymyositis
  - ✓ Polyradiculoneuritis
  - ✓ Distemper
  - ✓ Systemic lupus erythematosus
  - ✓ Lead poisoning
  - ✓ Chagas disease

Ingesta accumulates in the esophageal lumen

↓

putrefaction and esophagitis

motor dysfunction

results in failure of peristaltic pushing of the food through the lower esophageal sphincter into the stomach.

Parasitic Diseases of The Esophagus

- ✓Sarcosporidiasis
- ✓*Hypoderma lineatum*
- ✓*Spirocerca lupi*
- ✓*Gasterophilus* spp.
- ✓*Gongylonema*

Neoplasms of Esophagus

- Papillomas
- Squamous cell carcinomas

FORESTOMACHS

Examination of ruminal contents in necropsy

- **Urea toxicity** ..... Ammoniacal odor, alkaline pH
- **Organophosphates toxicity** ... Pungent insecticidal smell
- **Plant toxicity** ..... *Taxus* spp. aromatic odor- cedar oil
- **Lead poisoning** ..... Motor oil, paint flakes or metallic lead
- **Primary tympany** ..... Foaming of the rumen contents

Postmortem Changes

- Rumenomalasia
- Postmortal tympany
- Postmortal rupture

HYPERPLASTIC CHANGES IN THE RUMINAL MUCOSA

Ruminal papillae

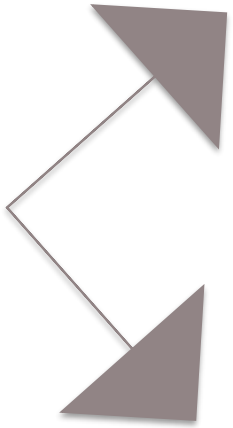
- Newborn calves — **Rudimentary, flat**
- Coarse roughage (apprx. 15%) — **Thin, long, white to grey**
- High-concentrate rations
- Barley rations
- Deficiency in Vitamin A
- Chloronaphthalene poisoning

- **Dark, clumped and Club-shaped**
- **Atrium ruminis and ventral caudal sac**
- **Microscopically Acanthosis, hyper- and parakeratosis and hyperpigmentation, secondary papillae hyperplastic, fibrosis.**

Similar changes like high-concentrate rations, matted apperance
Leukocytic reaction, microabscesses

Dilation of The Rumen

Excessive collection of gas in the rumen and reticulum is called TYMPANI.



Acute or primary tympany (characterized by foaming of the rumen contents, which prevents gas from being eructated).

Chronic or secondary tympany(the gas is free but retained because of some physical or functional defect of eructation).

PRIMARY TYMPANY (FROTHY BLOAT)

- There is apparently a delicate balance between **profoaming and antifoaming factors in the rumen.**
- The formation of foam is dependent on ***soluble proteins***
- **Soluble proteins**, released from chloroplasts, are degraded by the rumen microflora, and they rise to the surface where they are **denatured**, become **insoluble**, and **stabilize the foam.**
- The optimal pH (isoelectric point) for foam production by soluble proteins ranges from **5.4 to 6.0.**
- ***Pectins*** are considered to increase viscosity of ruminal fluid and may act as foam-stabilizing agents.
- **Plant lipids may act as antifoaming agents** by **competing for metal ions with the soluble proteins,** thus inhibiting the **denaturation of these proteins and resulting in decreased foam production.**

PRIMARY TYMPANY (FROTHY BLOAT)

- High levels soluble proteins → Rumen microflora → Denatured, insoluble, Stabilize the foam

PH 5.4-6.0

- Pectins → Increase viscosity of ruminal fluid → Stabilize the foam

- Plant lipids (antifoaming agents) → Competing for metal ions with The soluble proteins → Resulting in Decreased Foam production

PRIMARY TYMPANY (FROTHY BLOAT)

- Saliva has properties that **may promote or prevent foaming** in the rumen. When secretion
- of saliva decreases, the viscosity of ruminal contents increases, which makes foaming.
- Succulent and high-concentrate feeds **reduce salivary secretion**, thus increasing viscosity of rumen contents.
- The composition of saliva also affects foam production in several ways. **Combination of salivary bicarbonate with organic acids** results in the production of large amounts of *carbon dioxide,* increasing bubble formation.
- **Rations high in concentrate and low in roughage not only reduce saliva secretion, but also change the ruminal microflora.**
  - They promote the growth of large numbers of encapsulated bacteria, which increase the concentration of polysaccharides, and these, in turn, increase the viscosity, promoting foam.
  - These bacteria are also often mucinolytic and may destroy salivary mucins.

PRIMARY TYMPANY (FROTHY BLOAT)

The amount and composition of saliva

When secretion of saliva decreases

Succulent, high-concentrate feeds → The viscosity of ruminal contents increases

Combination of salivary Bicarbonate with organic acids → Production of large amounts of carbon dioxide

Salivary mucoproteins → Increase viscosity

Mucins in saliva (Parotid, submaxillary) → Reduce viscosity

Buffering action of saliva → Raise pH of the ruminal contents and soluble proteins produce stable foam

Rations high in concentrate -low in roghage → Reduce saliva secretion, change ruminal microflora, large numbers of encapsulated bacteria

Increase the concentration of polysaccharides

Increase the viscosity of ruminal contents

Destroy salivary mucins

The Cause of Death

- Increased intra-abdominal pressure on the diaphragm inhibits respiration and adversely affects cardiac function.
- The caudal vena cava is compressed, decreasing venous return to the heart.

NECROPSY FINDINGS IN PRIMARY TYMPANY

- Excessive dilations in forestomachs
- Blood exudes from the orifices
- Subcutaneous hemorrhages-cranial extremities
- Edema, congestion, hemorrhage of the cervical muscle and of the lymph nodes of the head and neck
- The tracheal mucosa is hemorrhagic
- Blood clots (bronchi, paranasal and frontal sinuses)
- The lungs compressed into the cranial thorax
- Pressure ischemia of the abdominal viscera (especially liver)
- Lymph nodes and the muscles of the hind legs are pale
- Subcutaneous edema-vulva, perineum
- The ruminal contents are bulky and foamy(10-12 h)

NECROPSY FINDINGS IN PRIMARY TYMPANY

- **Bloat line** in the esophageal mucosa

(congestion-petechial/ ecchymotic hemorrhages– pale mucosa)

- Postmortal inguinal hernia and diaphragmatic rupture

SECONDARY TYMPANY (FREE GAS OR SECONDARY BLOAT)

It is usually the result of a physical or functional defect in eructation of gas produced by normal rumen fermentation.
The more common physical problems include internal or external obstructions of the esophagus or esophageal groove by tumor, foreign body, or esophageal stenosis of any cause.

- **Physical problems**

✓Internal or external obstructions of the esophagus

✓Esophageal stenosis

✓Reticular adhesions

✓Abscesses

✓Peritonitis

✓Tumor masses that interfere with contractions of the forestomach

SECONDARY TYMPANY

- **Functional causes**

✓Organophosphate intoxication and vagal damage due to adhesions, lymphosarcomatous infiltrates

- **Other causes**

✓Bucket-fed calves ingest large amounts of milk-*ruminal drinkers*

✓Animals fed rations that have too much undigestible roughage

FOREIGN BODIES IN THE FORESTOMACHS

- Cattle are notoriously **lacking in alimentary finesse**, a deficiency that allows an amazing variety of foreign bodies (**metallic, wood or plastic foreign bodies**) to be deposited in the forestomachs (**specially rumen and reticulum**).
- Bezoars-spherical masses

  * **Trichobezoars = hair or wool**

  * **Phytobezoars = plant fibers**

Not important unless regurgitated to lodge in the esophagus or passed on to obstruct the reticulo-omasal orifice, the pylorus, or the intestine, which is very infrequent.

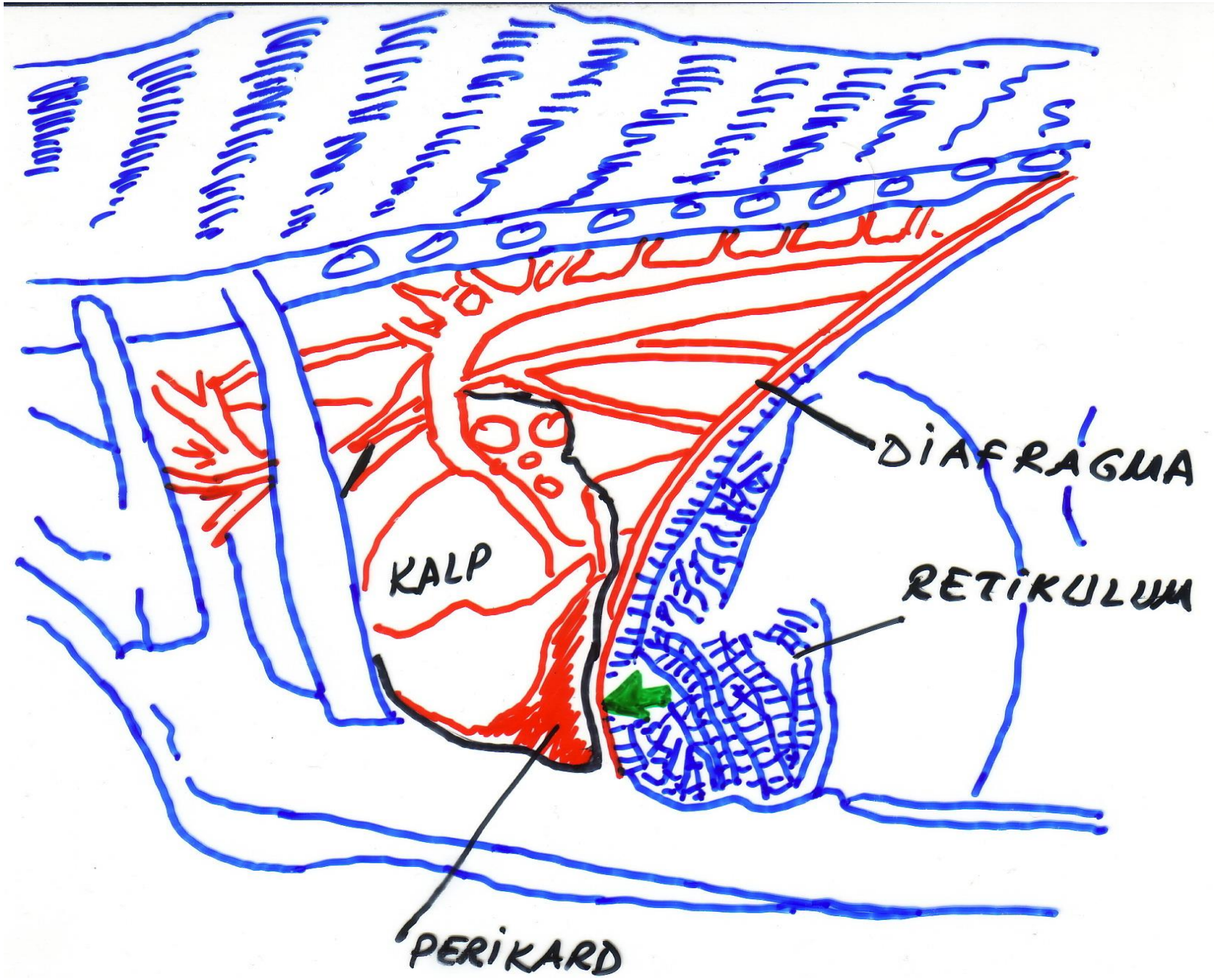
Otherwise, these bezoars are an **incidental finding**

- The sequel to penetration **by sharp objects** in adult cattle is **traumatic reticuloperitonitis**.

TRAUMATIC RETICULOPERITONITIS

Perforation of the forestomachs by foreign bodies is caused by a long, thin, and sharp foreign body, usually a wire or nail, penetrating the reticular wall.

The prophylactic use of magnets has become common in many herds, and this probably contributes to the marked decrease in fatal cases of traumatic reticuloperitonitis observed over the past few years.



The perforation is usually in the cranioventral direction and is followed by *acute local peritonitis.*

If the foreign body is short or bent, with the next reticular movement, only *chronic local peritonitis* with adhesions develops.

The foreign body may advance to perforate the diaphragm and pericardium, resulting in *traumatic pericarditis.*

Traumatic reticuloperitonitis

- **Incomplete perforation**
- **Penetration**
  - **ANTEROVENTRAL** → **Traumatic reticuloperitonitis**
  - **VENTRAL abscess** → **Subperitoneal and subcutaneous**
  - **ARTERIES** → **Rupture and hemorrhage**
  - **THORACIC CAVITY** → **Pneumonia, pleuritis**
  - **RIGHT LATERAL** → **Lesion at wall of the abomasum**
  - **LIVER AND SPLEEN** → **Metastatic abscesses**
  - **RETICULUM, OMASUM, ABOMASUM** → **Suppurative inflammation, vagus indigestion**

VAGUS INDIGESTION

- **CLINICALLY**
  - Persistent ruminal atony or irregular motility
  - Bilateral abdominal distension
  - Inappetence
  - Decrease milk production

CAUSES / I

- Migration of the foreign body is penetration of the side of the reticulum, leading to suppurative inflammation in the grooves between the reticulum, omasum and abomasum
- *Looseness of Sulcus oesophagicus*
- Degeneration of muscle and intramuscular nerve plexus
- Lesions of **the vagus nerves**

CAUSES / II

- Vagal lesions-pharyngeal and cervical areas, or intrathoracic or abdominal
- Adhesions following reticular perforation
- Trauma following abomasal volvulus
- Peritonitis
- Abscessation

TYPE II VAGUS INDIGESTION

(FAILURE OF OMASAL TRANSPORT)

- **Impairment of movement** of ingesta from the reticulorumen to the omasum associated with abscesses adjacent to the reticulo-omasal orifice.

- **AT NECROPSY**

- The abomasum may be distended and impacted with dry ingesta, because of functional pyloric stenosis or abomasal stasis.
- The omasum can be very large and impacted with dehydrated ingesta.
- The rumen is distended with enough fluid-no ruminal fermentation
- Morphologic lesions of the vagus nerves
- Adhesions or neoplasms involving the forestomachs and abomasum.

RUMENITIS

- Inflammatory lesions in the forestomachs occur in a number of **viral, parasitic, mycotic, toxic, and nutritional diseases** of the alimentary tract in ruminants.

- *Primary bacterial or mycotic inflammatory lesions* of the rumen are uncommon, they can occur subsequent to **chemical rumenitis, primary viral rumenitis, sepsis, or intensive antibiotic treatment.**

VIRAL RUMENITIS

- In neonatal calves IBR (Necrosis of ruminal mucosa)
- Bovine papular stomatitis
- Contagious ecthyma
- CGB (Focal or diffuse rumenitis)
- Adenoviral infection (Multifocal fibrinohemorrhagic rumenitis)
- BVD-MD (Ruminal erosions and ulcers)
- Rinderpest (Ruminal erosions and ulcers)
- Foot and mouth disease (Ruminal erosions and ulcers)
- Bluetongue (Extensive hemorrhage and ulceration)
- Sheeppox (Nodules)

NUTRITIONAL DISEASES

- Mild inflammation of the forestomachs occurs in some young calves fed milk from a pail, when, because of laxity of the reticular groove reflexi the milk spills into the rumen and reticulum in large quantity.
- Putrefaction in these compartments leads to mild rumenitis, edema and mild neutrophil infiltration of the mucosa
- **Ammonia toxicity (Accidental consumption of excessive quantities of urea)**
- **Ingestion of toxic levels of sulfur**
- **Plant toxicoses**
- **Feeding rations deficient in fiber**

CHEMICAL RUMENITIS

Develops after overeating on rapidly fermentable carbohydrate, usually grain.

RUMINAL ACIDOSIS

Ruminal acidosis usually follows the ingestion of excess carbohydrate in the form of grain, or other fermentable feedstuffs occasionally used, such as root crops, bread, waste baked goods, brewers' waste, and apples.

- Shortly after the ingestion of a toxic amount of carbohydrate, ruminal pH begins to fall.
- As ruminal pH drops, ruminal atony develops, mainly as the result of an **increase in the concentration of the nondissociated volatile fatty acids**, *lactic, propionic, and butyric*.
- They act on receptors that mediate inhibition of reticuloruminal motility. SO**, stasis** is seen.
- There is also cessation of salivary secretion, **so that the buffering effect of saliva is absent.**
- The increase in ruminal organic acids, mainly **lactate**, causes an increase **in ruminal osmotic pressure.**
- This results in movement of fluid from the blood into the rumen,
- Plasma volume is reduced; hemoconcentration, anuria, and circulatory collapse follow.

- The low ruminal pH is lethal to much of the normal flora and fauna. The protozoa appear to be particularly sensitive but many types of bacteria are also lost.
- **Fusobacterium necrophorum** is a normal inhabitant of the anaerobic ruminal environment. This bacterium is commonly responsible for complications of ruminal acidosis, producing
- characteristic lesions in the forestomachs and in the liver. Characterized necrotic epithelium and liver.
- Because of the acidosis mycotiv rumenitis can be occured by mucor, Rhizopus and absidia.
- **Mycotic rumenitis** is much more severe and extensive than necrobacillary rumenitis, and is often fatal. *The basis for the lesion is submucosal venular thrombosis caused by fungal invasion,* causing venous infarction of the tissue field involved.

CLINICALLY

- Intensive toxemia
- Dehydration
- Ruminal stasis
- Weakness
- High mortality

AT NECROPSY

- The eyes are sunken, the blood may be thick and **dark due** to **dehydration and hypoxia**
- **General venous congestion**
- **Porridge-like rumen content** which has a distinct fermentative odor
- Absence of protozoa
- **Blue coloration** in the ventral sac of the rumen, reticulum and in the omasum.

MICROSCOPIC EXAMINATION

- Ruminal papillae appear enlarged
- Cytoplasmic vacuolation of the epithelial cells
- Neutrophilic reaction is evident in the mucosa and submucosa
- Focal areas of erosion and ulceration

NECROBACILLARY RUMENITIS

MACROSCOPIC -MICROSCOPIC EXAMINATION

- Forestomachs and hepatic lesions- Coagulative necrosis

"**rumenitis-liver abscess complex**"

- The papillated areas of the ventral sac and occasionally the pillars-multiple irregular patches 2-15 cm across:

The papillae are swollen, dark slightly mushy and are matted together by fibrinocellular inflammatory exudate

- Affected papillae are necrotic, ulceration

(If the animal recovers; a stellate scar often remains)

MYCOTIC RUMENITIS

- Should be suspected when inflammation in the wall of the forestomachs extends to the serosa and is *hemorrhagic* and *angiocentric*.

- The basis for the lesion is submucosal venular thrombosis caused by fungal invasion, causing venous infarction of the tissue field involved.

MYCOTIC RUMENITIS

At NECROPSY

- The inflammation extends to the peritoneum, causing **hemorrhagic and fibrinous peritonitis** that mats the *omentum to the rumen*
- In fatal cases, most of the ventral sac and parts of the reticulum and/or omasum are involved
- The lesions are very striking and the walls have been massively infarcted

MICROSCOPIC EXAMINATION

- **Necrotic** epithelium
- **Necrotic** vasculitis and submucosal venular thrombosis
- Granulomatous inflammation in chronic cases
- The rumenitis is characterized by hemorrhagic necrosis of all structures in the walls; by copious fibrinous exudate; and by rather scant leukocytic reaction
- Fungal hyphae are in the necrotic tissues and the lumina of the thrombosed blood vessels
- Metastases sometimes occur in the liver

NEOPLASIA OF THE FORESTOMACHS

- **Papillomas**
- **Fibropapillomas**
- **Squamous cell carcinomas**
- **Lymphoma**

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