DISEASES OF THE FORESTOMACH

Normal Structure and Function of Relevance to Disease

- the **reticulum** is the most cranial compartment of the forestomach of ruminants
- it acts mechanically to reduce ingesta to fine particles
- heavy ingesta (e.g. grain, rocks, nails) tends to fall into this compartment
- lighter materials (e.g. grass, hay) tend to enter the rumen

- the **rumen** is the large fermentation vat with an anaerobic environment and constant temperature and pH in which micro-organisms break down ingested cellulose and other substrates mechanically and chemically into short chain volatile fatty acids (VFA) that can be directly absorbed across the mucosa into blood
- VFA provide more than 70% of the dietary energy supply to ruminants
- virtually all of the acetic, proprionic and butyric acids formed in the rumen are absorbed across the ruminal mucosa (butyric acid is first metabolised by themucosal epithelium to the ketone body, β-hydroxybutyrate)
- continuous removal of VFA prevents acidification of the ruminal pH

- ruminants produce a massive volume of saliva (estimated at 100-150 L of saliva per day in adult cows)
- the saliva provides fluid for fermentation in the rumen and is an important source of bicarbonate to buffer the acids produced locally in the rumen

- **rumination** (regurgitation, re-mastication and re-swallowing of ingesta) increases the surface area of the ingested substrates to promote subsequent fermentation by the ruminal bacteria

- **eructation** (or belching) is necessary to release carbon dioxide and methane gases produced by bacterial fermentation of ingesta in the rumen

- co-ordinated movements of the rumen and reticulum are necessary for normal mixing and fermentation of ingesta, regurgitation and eructation
- the strength and rate of contractions is controlled by the brainstem via branches of the vagus nerve
- stretch receptors and chemoreceptors in the rumen modulate contractions via vagal nerve axons that relay signals to the brainstem
- **abnormal acidity of ruminal contents** +/- **ruminal distension → decreased ruminal motility**

- the **oesophageal groove** in suckling ruminants permits ingested milk to bypass the rumen and reticulum and pass directly into the omasum and thence abomasum, where it can be curdled by rennin and thence digested enzymatically
- the groove is formed by muscular folds of the reticulum
- closure of the groove is controlled by neural stimulation from suckling and by milk proteins so that milk, colostrum and milk replacer (from a nipple, bottle or open bucket) bypass the rumen and reticulum whilst water can enter these chambers

- the function of the **omasum** is poorly understood
- it may function in absorption of residual VFA and bicarbonate

- fluids tend to pass rapidly through the omasal canal but particulate material tends to be retained between the laminae until periodic contractions force it into the abomasum

- the most significant disorders of the forestomach of ruminants involve **abnormalities of chamber motility** and/or **imbalance of the microbial flora**
- potential sequelae include **bloat**, **ruminal acidosis**, and **secondary bacterial** or **fungal infection**

EFFECTS OF DIET ON RUMINAL PAPILLAE

- ruminal papillae are rudimentary in newborn and suckling ruminants (which are functionally monogastric)
- the timing and extent of subsequent development of the papillae is influenced by the diet
- exposure to roughage and **especially concentrates** promotes development of the papillae
- papillary growth is stimulated by proprionate and butyrate produced by fermentation, possibly mediated by insulin-like growth factor-1

- a high plane of nutrition (with production of abundant VFA in the rumen) → large papillae to promote absorption of VFA
- a poor plane of nutrition → small, blunt papillae

- papillae may vary in shape from long and flat to conical, spade-shaped or hair-like

- **adequate fibre** (approximately 15% of the diet) → long, slender papillae

- **high concentrate rations with inadequate roughage** → stumpy, club-shaped papillae that tend to form clumps, nodules and rosettes, especially in the ventral sac of the rumen
  - mucosa undergoes hyperplasia, hyperkeratosis and often hyperpigmentation
  - clumping is encouraged by hyperplasia of secondary papillae
  - plant fibres (e.g. rachilla of barley) and animal hairs may adhere to the mucosa and contribute to the matted appearance, and may also penetrate the mucosa to cause inflammation (e.g. “barley beef rumenitis” in feedlot cattle)

- **vitamin A deficiency** in calves can also cause hyperkeratosis of the forestomach mucosa

POST MORTEM EXAMINATION OF THE RUMEN

- assessment of the forestomach compartments (especially the rumen) at necropsy may provide important clues to the metabolic state of a ruminant at death and the cause of death

- the volume, odour, consistency and pH of the ruminal contents, the height of the ruminal papillae, and the integrity of the mucosa should all be assessed at necropsy

- depending on the diet, the **pH of ruminal fluid** in health is **5.5-7.5**

- overly dry ruminal contents = **dehydration**
- excessively watery ruminal contents = **vagal indigestion or grain overload**

- voluminous, frothy ruminal contents = **primary bloat**
- acidic pH (< 5), fermented odour +/- grains = **grain overload (lactic acidosis)**
- alkaline pH and ammoniacal odour = **urea toxicity**

- odour of cooked turnips or a pungent insecticidal smell – may indicate **organophosphate poisoning**
- rancid odour and putrefied milk = **failure of the oesophageal groove reflex**

- lead flakes, motor oil or paint flakes – suggest **lead poisoning**
- aromatic odour (akin to wood polish) and pine needles – ***Taxus* spp. (yew) poisoning** (contain toxic alkaloids)
- characteristic foliage in other plant poisonings – e.g. ***Prunus* spp. (plums, cherries, peaches, apricots, almonds)** (contain hydrogen cyanide)

- once the luminal content has been assessed, the forestomach compartments should be hosed out gently with water to remove the content and allow close inspection of the mucosa for any evidence of hyperaemia, erosion, ulceration or scarring

- the ruminal mucosa usually sloughs within a few hours of death so that the mucosa can be peeled away from the lamina propria in sheets
- **adherence of the mucosa post mortem is abnormal** and may reflect **rumenitis** or **fibrosis**

BLOAT

- **bloat = dilation of the rumen, ruminal tympany, hoven**

- can be **primary** (typically acute) or **secondary** (typically chronic)
- up to 50% mortality rate

- ruminal fermentation generates an enormous volume of gas in health (30-50 L/hour in adult cows, 5 L/hour in adult sheep or goats)
- **eructation** or belching of carbon dioxide and methane takes place with almost every secondary ruminal contraction
- anything that interferes with eructation in a ruminant can cause life-threatening bloat

Primary Bloat (Frothy Bloat)

- in health, only a small volume of foam is produced in the rumen and it is unstable
- primary bloat is an acute condition of the rumen in which stable foam forms and prevents eructation of gas

- most often associated with consumption of **succulent legumes** (e.g. alfalfa and clover) (**pasture bloat**) → bloat within 1-3 days

- succulents stimulate less saliva production → increased viscosity of ruminal fluid → promotion of foam formation
- soluble proteins released from legume chloroplasts are degraded by ruminal flora → rise to the

surface of the ruminal fluid and become denatured and insoluble → stabilisation of the foam
- legume acids also drop the ruminal pH to 5.4-6 (optimal for formation of stable foam)

- some cattle may be genetically predisposed to develop primary bloat because they produce less saliva

- primary bloat may also develop in feedlot cattle on **high concentrate and low roughage** diets (**feedlot bloat**)
- onset of feedlot bloat is usually more gradual than that of pasture bloat
- high concentrate/low roughage diets → decreased saliva production and a gradual change in the ruminal flora → increased bacterial production of polysaccharides → increased viscosity of the fluid → stable foam formation

- irrespective of the underlying cause, excess ruminal foam prevents formation of a free gas cap and clearance of the cardia
- failure of eructation → distension of the rumen → massive increase in intra-abdominal pressure → pressure on the diaphragm and lungs → dyspnoea → hypoxaemia
- high intra-abdominal pressure +/- direct compression of the intra-abdominal caudal vena cava by the rumen also causes impaired venous return to the heart → circulatory failure with congestion of the distal hindlimbs

Post Mortem Findings

- often sudden death, with animals found lying on their backs with a sawhorse posture, and with the abdomen markedly distended by gas
- dark, poorly clotted blood may ooze from orifices due to hypoxaemia
- congestion, oedema and haemorrhage of the subcutis, muscles and lymph nodes of the head and neck +/- mucosa/submucosa of the proximal trachea, paranasal sinuses and nasal cavity
- pale muscles of proximal hindlimbs with congestion distally
- +/- **bloat line** in the **oesophagus** at the **level of the thoracic inlet** – sharp or gradual demarcation between blanched distal oesophagus and proximal congested oesophagus
- bulging diaphragm
- lungs pale and compressed into cranial thorax
- pallor of compressed abdominal organs, especially the liver
- rumen distended by bulky foamy contents (foam may disappear if necropsy examination is more than 10-12 hours after death)

Secondary Bloat

- usually a **chronic** condition (+/- episodes of acute exacerbation) referable to **a physical or functional obstruction preventing eructation of gas from the rumen**

- e.g. physical obstruction of the oesophagus by a tumour, foreign body or stricture
- e.g. fibrous adhesions, tumours, abscesses or peritonitis interfering with contraction of the rumen and reticulum
- e.g. functional obstruction of the oesophagus due to vagal nerve injury
- e.g. functional obstruction of the oesophagus due to organophosphate poisoning

- post mortem findings are as per primary bloat **but without stable foam formation** (and with the possible presence of an identifiable physical obstruction)

- **other causes of secondary bloat** include:

- **excess indigestible roughage** → recurrent episodes of bloat
  - if digestible nutrients (starch, sugars) in the diet are inadequate, excess roughage cannot be digested and accumulates in the rumen and reticulum → distension → inhibition of contractions → failure of eructation

- **failure of the oesophageal groove reflex in bucket-fed calves** ("ruminal drinkers") → milk or milk replacer flows into the rumen → putrefaction → mild rumenitis and hyperkeratosis
  - clinical signs include inappetence, recurrent bloat, abdominal pain and passage of clay-like faeces
  - may see a similar problem in calves fed by stomach tube

FOREIGN BODIES

- foreign bodies may lodge in the rumen or reticulum
- most common in **cattle** due to their non-selective eating habits
- rare in sheep and goats

- **trichobezoars** = spherical masses largely composed of **hair** or **wool**
  - more common in younger animals
  - may be associated with inadequate dietary fibre, boredom, pica or skin disease (e.g. lice)
  - may become impregnated with mineral
  - usually incidental

- **phytobezoars** = spherical masses largely composed of **plant fibres**
  - may be due to rough feed or oral or dental disease that impairs mastication
  - may become impregnated with mineral
  - usually incidental

- bezoars are only of importance if they are regurgitated to obstruct the oesophagus or if they obstruct the reticulo-omasal orifice, the abomasal pylorus or the intestines

- **foreign bodies containing lead** – can cause lead poisoning when dissolved and absorbed

- **wood shavings or straw** – e.g. calves with inadequate roughage
  - may cause diffuse mixed bacterial cellulitis of the forestomach +/- abomasum

- **sharp objects** – can result in traumatic reticuloperitonitis (TRP) (see below)

TRAUMATIC RETICULOPERITONITIS

= hardware disease, traumatic reticulopericarditis

- sharp foreign bodies (especially wires or nails over 4 cm in length) may perforate the reticulum during contractions
- perforation is usually in a cranioventral direction
- bacteria such as *Trueperella (Arcanobacterium) pyogenes* and *Fusobacterium necrophorum* extend along the perforation track from the reticulum

- prevalence of this condition has been reduced through prophylactic use of rumen magnets and also replacement of baling wire with twine

- **incomplete perforation** is often inconsequential but may → focal suppurative or granulomatous inflammation in the wall of the reticulum with minor overlying peritonitis

- **penetration through the wall of the reticulum** → acute local peritonitis → chronic local fibrous adhesions
  - if the foreign body is short or bent, it may protrude no further and may even be withdrawn back into the reticulum

- **penetration of diaphragm** → pleuritis +/- pneumonia

- **penetration of pericardial sac** → florid suppurative or fibrinosuppurative pericarditis → eventual right-sided congestive heart failure

- **other possible sequelae** include:

  - penetration of the myocardium or a large regional artery → fatal haemorrhage
  - penetration of the liver or spleen → abscessation
  - right lateral deviation with penetration of the abomasum
  - ventral penetration → subcutaneous abscess near xiphoid
  - penetration of the side of the reticulum → chronic suppurative inflammation in grooves between the reticulum, omasum and abomasum → **vagus indigestion**

VAGUS INDIGESTION

- **vagus indigestion = a clinical syndrome resulting from a physical or functional outflow obstruction of the forestomach or abomasum → distension of the rumen and reticulum**

- characterised by persistent ruminal atony or irregular motility, gradual bilateral abdominal distension, inappetence and decreased milk production

- **causes** include:

- **traumatic reticuloperitonitis**

- **physical obstruction of outflow from the rumen/reticulum, omasum or abomasum** - e.g. foreign bodies, neoplasms, abscesses adjacent to the reticulo-omasal orifice

- **damage to the vagus nerve** anywhere along its length (neck, pharynx, intra-thoracic, intra-abdominal) – especially entrapment in fibrous adhesions of TRP or due to trauma following right displacement and volvulus of the abomasum

- **degeneration of muscle in the wall of the oesophageal groove** → flaccidity

- **pregnancy** – possibly due to displacement of the abomasum by the uterus

- many cases are **idiopathic** (i.e. of unknown cause)

Post Mortem Findings

- rumen distended with watery fluid lacking odour; unmacerated food particles float on the surface of the fluid
- omasum can be very large and impacted with dry feed
- abomasum may be distended and impacted with dry feed

RUMENITIS

- **rumenitis = inflammation of the rumen**

Lactic Acidosis

- **lactic acidosis = ruminal acidosis, grain overload, rumen overload, carbohydrate engorgement**

- **the most common cause of rumenitis**

- occurs in dairy and feedlot beef cattle, sheep and goats
- due to dietary overload with rapidly fermentable carbohydrate (e.g. grain, bread, root crops) or a sudden increase in the amount of carbohydrate in the diet
- the amount of carbohydrate consumed is less important than the rapidity of the dietary change

- may trigger a **severe acute metabolic disease → high morbidity and mortality**
- survivors have **chemical rumenitis** and are **predisposed to development of secondary infections with *Fusobacterium necrophorum* and/or fungi**

- fermentation of the carbohydrate → increased VFA production → decreased ruminal pH
- at a pH of ≤ 5.0, ruminal Gram-negative bacteria (which predominate in health) and protozoa die
- streptococci (especially *Streptococcus bovis*) begin to proliferate and produce lactic acid → further decrease in the pH
- at a pH of 4.5-5.0, streptococci then decline and lactobacilli proliferate

- low pH → ruminal atony and reduced secretion of saliva (→ decreased buffering of ruminal acid

by salivary bicarbonate)
- lactic acid acts osmotically → fluid moves from circulation into the ruminal lumen → ruminal distension and then diarrhoea → profound dehydration and hypovolaemic shock → death

Post Mortem Findings

- **diagnosis can be difficult in the acute phase**
- evidence of dehydration – e.g. sunken eyes, tacky subcutis, haemoconcentration
- ruminal contents may be voluminous and porridge-like with a distinctive fermented odour
- grain may be obvious in the rumen but can be scanty or overlooked if finely ground
- in more advanced cases, rumen contents may appear normal but intestinal contents are very watery
- acute chemical rumenitis may not be obvious grossly
- detachment of the ruminal mucosa may reveal patchy hyperaemia of the lamina propria

- **a rumen pH < 5.0 is diagnostic** but the pH may rise in later stages of the disease as putrefaction and release of toxic amines develop
- no protozoa are identifiable in wet mount smears of ruminal fluid (but protozoa will die off anyway as the post mortem interval increases)

- **histopathology of the rumen is diagnostic**
- ruminal papillae are swollen with severe hydropic degeneration of the mucosal epithelial cells +/- coagulative necrosis
- mild to severe infiltration of the mucosa and lamina propria by neutrophils
- +/- formation of mucosal vesicles, erosions and/or ulcers

- if the animals survive, the ulcers heal by granulation tissue → permanent white scars lacking papillae

Secondary Bacterial Rumenitis (Necrobacillary Rumenitis)

- ruminants that survive a mild episode of lactic acidosis may develop secondary bacterial infection of the damaged ruminal mucosa
- usually involves *Fusobacterium necrophorum*, a member of the normal ruminal anaerobic flora → usually multiple, superficial, 2-15 cm diameter foci of coagulative necrosis with a hyperaemic margin, especially in papillated areas of the ventral sac and occasionally the pillars
- if the animal survives, the necrotic lesions slough → ulcers → healing by granulation tissue → white stellate scars lacking papillae

- embolism of infection via portal vein branches to the liver → multiple foci of coagulative necrosis that ultimately liquefy to form conventional hepatic abscesses (= **hepatic necrobacillosis**)

Secondary Fungal Rumenitis

- mycotic infection of the rumen is **most often a complication of lactic acidosis**
- usually involves zygomycete fungi (e.g. *Absidia*, *Mucor*, *Rhizopus* spp.)
- much more severe than necrobacillary rumenitis and often fatal

- fungal hyphae invade blood vessels of the ruminal wall → **vasculitis, thrombosis and infarction**

- lesions are typically circular, sharply demarcated and fibrinohaemorrhagic
- the necrotic ruminal mucosa is usually difficult to detach
- **always suspect fungal infection if fibrinohaemorrhagic lesions extend from the ruminal mucosa to the serosal surface**

- in fatal cases, most of the ventral sac of the rumen and parts of the reticulum and omasum are involved
- hyphae may also embolise via portal vein branches to the liver → hepatic infarcts

- fungal infection of the forestomach compartments may also develop in ruminants secondary to mucosal ulceration caused by:

  - **broad spectrum antibiotic administration** with disruption of normal flora - especially in calves
  - **septicaemia**
  - **reflux of abomasal contents**
  - **primary viral infection** – especially bovine viral diarrhoea virus (BVDV) infection

Viral Rumenitis

- the following viral infections may cause lesions in the mucosa of the forestomach compartments:

  - bovine herpesvirus-1
  - bovine papular stomatitis
  - contagious ecthyma (scabby mouth, orf)
  - BVDV/mucosal disease
  - malignant catarrhal fever
  - bluetongue
  - rinderpest
  - foot and mouth disease
  - bovine adenovirus

- the virus-induced mucosal erosions, ulcers, vesicles, papules or pustules may become secondarily infected by *F. necrophorum* or fungi

Toxic Rumenitis

- e.g. accidental consumption of **excess urea** as a non-protein nitrogen supplement or in fertiliser → production of ammonia in the rumen → caustic injury to the mucosa

PARASITES

- ***Gongylonema* spp.** – spirurid nematodes found in the oesophageal and ruminal mucosa +/- lamina propria → red blood-filled serpentine tracks → heal by scar tissue

- ***Paramphistomum* and related spp.** – conical flukes
  - found in the rumen and reticulum of cattle, sheep and wild ruminants
  - red, plump, pear-shaped parasites that reside between the ruminal papillae
  - usually non-pathogenic but large numbers may cause atrophy and hyperkeratosis of the papillae

NEOPLASIA

- most neoplasms of the ruminant forestomach are of no clinical significance
- large lesions in strategic locations may cause **secondary bloat** or **vagus indigestion**

- **papilloma** - a benign neoplasm of squamous epithelial cells
  - rare in sheep
  - common in cattle in some areas due to infection with bovine papillomavirus type 4

- **fibropapilloma** - a benign neoplasm of squamous epithelial cells and lamina proprial fibrocytes/fibroblasts
  - due to infection with bovine papillomavirus 2
  - resembles a papilloma but has proliferating fibroblasts in the lamina propria beneath the thickened epithelium

- **squamous cell carcinoma** – a malignant neoplasm of squamous epithelial cells
  - rare in sheep and most cattle
  - can be common in cattle in some locations (e.g. Scotland, northern England, Kenya) due to exposure to bovine papillomavirus 4 and carcinogens (e.g. ptaquiloside) in bracken fern *(Pteridium aquilinum)* or other native plants

- **lymphoma** - a malignant neoplasm of lymphocytes
  - rare in cattle, and usually as an extension from lymphoma involving the abomasum

- **fibroma** - a rare benign neoplasm of fibrocytes/fibroblasts producing collagen