Vet Med: Applied GI Physiology- Supplemental Notes

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ERIN MALONE DVM PHD

Contents

This book is designed to accompany the lectures and discussions on GI Physiology at the UMN CVM. Suggestions always welcome. Please send ideas, concerns, edits to Dr. Malone using this link

You can also use the hypothes.is app to annotate (improve, suggest, ask questions etc). You can access this book via this link

GI glossary – for questions or additions

GI questions– for your questions or answers

Finding additional resources

For starter explanations, try these:

- Wikipedia– useful for physiology, unknown terms and other more general knowledge questions
- Vetfolio – has been free for students; great summaries of topics and conditions
- Merck Veterinary Manual online- good for pathogenesis and presentation; not as good for therapies
- Vet Clinics of North America Equine, Food Animal, and Exotics– see the search function at the top left. Does require a subscription. Available through many University libraries. Peer-reviewed summaries of topics.
- VIN – abstracts are posted in related feeds. Free for students and contains a variety of other resources (3D anatomy, drug calculators, financial aid info). The help from the boards is variable; sometimes excellent, sometimes dangerous.
- Specialty sites with info for animal owners (many are free for students): ACVS.org, ACVIM.org, AAEP.org, AASRP.org, AABP.org, AASV.org, sites with .gov or .edu
- Plumb's Veterinary Drug Handbook and app (lots of versions out there in different formats and prices)

- SCOVE Edu+ – online source of online resources

For more detailed explanations or comparison of therapies, diagnostic tools etc:

- Google scholar– look for the ones with a pdf. You can sort by year, type of article etc using the left hand bar
- Veterinary medical library Pub Med and other databases; video how to for UMN system; or go visit the librarian in person
- UMN library search– For articles and books. Great for specific articles you are trying to access or specific topics, particularly if not likely to be in pubmed.
- Ivis – international veterinary information service – is a great place to find specific meeting proceedings
- Extreme googling
- Surgery textbooks- great for how to perform surgery and the perioperative care required
- Revisit sites above

Unless it is fiction or for fun, don't read every word.

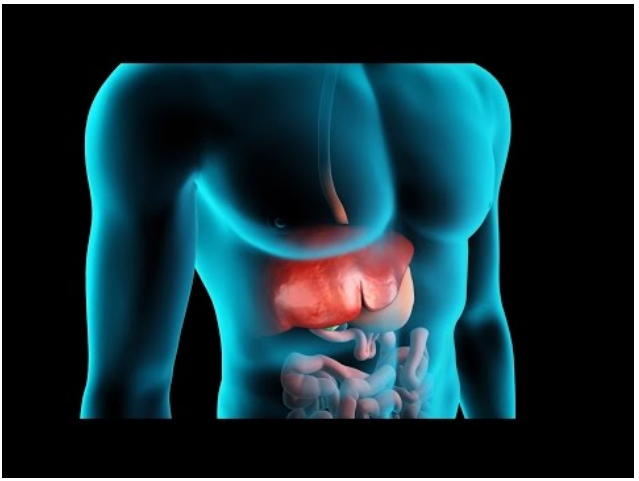
- Textbooks – read the chapter summary and/or skim the headings; then read the applicable sections.
- Journal articles – start with the abstract and/or introduction, follow with the last paragraph, finish with any middle bits that are useful
- Online materials – start with Ctrl-F if you have a specific question; otherwise go with the section headings and introductory sections as above.

PART I

LIVER

Liver function, liver dysfunction and testing for liver abnormalities

1. Liver function



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The liver receives nutrient rich blood directly from the GI tract through the portal vein. The blood is modified by the liver before it moves onto other organs.

The liver is responsible for over 500 functions. The most crucial liver functions include:

Metabolism management

- we use nutrients from the gut and from liver stores to

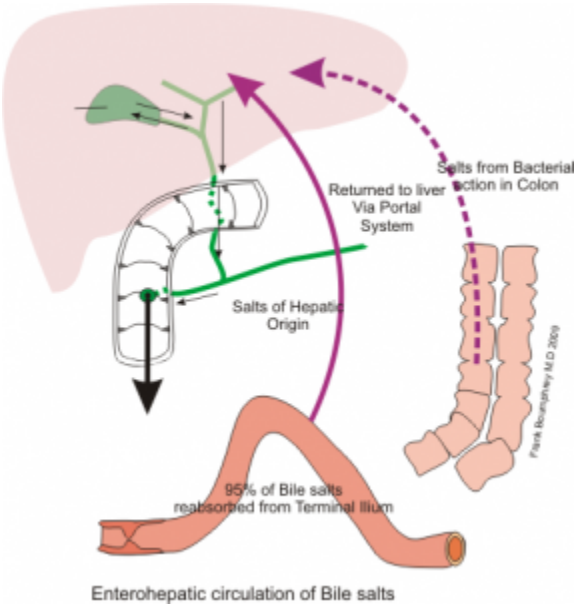
balance energy, build proteins, etc.
- all nutrient rich blood from the gut goes to the liver via the portal vein
- the liver either stores or releases compounds based on current metabolism needs
    - catabolism – break down nutrients for storage
    - anabolism – build up nutrients from stored components

Energy and nutrient storage

- the liver stores fats and carbohydrates in the form of triglycerides, lipoproteins and glycogen
- the liver stores B12 (cobalamin) and C vitamins, the fat soluble vitamins ADE and K, iron and copper

Bile production and recirculation

- bile is necessary for fat digestion/absorption
    - bile is important for emulsification of fats (turning large droplets into smaller droplets)
    - bile is important for absorption of fat-soluble vitamins (A,D, E and K)
- the **enterohepatic recirculation system** returns most of the bile to the liver for re-use



- *https://upload.wikimedia.org/wikipedia/commons/f/f4/Bile_recycling.png*

Protein formation

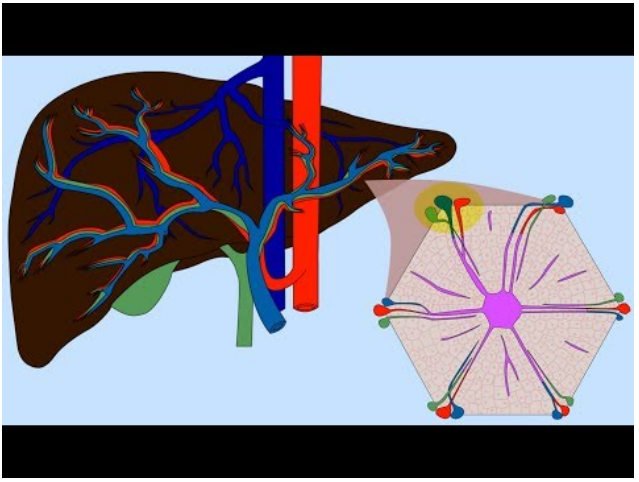
- the liver creates proteins required for coagulation, binding of other proteins, transport of iron and other compounds, immune function, and vascular pressure (oncotic pressure)

Detoxification

- the liver denatures toxins, potential toxins, ammonia (urea cycle) and old red blood cells

Drug metabolism

- the liver may activate or inactivate drugs, depending upon the drug, drug combination and specific enzymes
- cytochrome p450 enzymes are essential



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Resources

What is bile – video

Liver function Khan academy – includes P450 enzymes, bioavailability and more

Secretion of bile and the role of bile acids in digestion -short, sweet and useful

Hepatic lobule – review of the microscopic anatomy – portal triad; Khan Academy

The liver- functions – Neural academy video

Just for fun

I am a gall bladder

2. Liver dysfunction

With liver dysfunction, problems can arise with energy levels and energy reserves, vitamin and mineral deficiencies, protein levels, coagulation, toxins, and drug effects.

The liver does have a very large functional reserve. It can still function normally with only 25% of "good" liver remaining. The liver can actually regrow itself (this works well for liver transplants).

Energy levels

Since the liver controls our metabolism, with liver dysfunction we can run out of energy. Clinical signs include weakness, fatigue and seizures.

Nutrient storage

Liver disease can lead to issues with nutrient storage. Symptoms may develop due to low levels of copper, iron and vitamins. Scurvy can occur with liver disease.

Bile Production and Loss

With liver disease, the feces may be pale (not bile colored), fatty and smelly due to limited fat digestion. Weight loss usually accompanies liver disease due to lack of fat absorption.

Enterohepatic recirculation may be limited with bile salts collecting elsewhere in the body and/or not being available for reuse.

Protein Production

Less liver function means fewer proteins produced. This can impact oncotic pressure, coagulation, and other functions where proteins are required.

Edema occurs due to the lowered oncotic pressure. Hemorrhaging and bruising are evident with limited coagulation proteins.

Detoxification

Many agents are not being detoxified. The urea cycle and ammonia metabolism are disrupted. These compounds and others (amino

acids) can act as neurotransmitters, leading to neurological signs related to liver dysfunction.

Drug metabolism

Products aren't excreted normally, leading to build up in organs and the blood. Drugs may be less active or have more side effects.

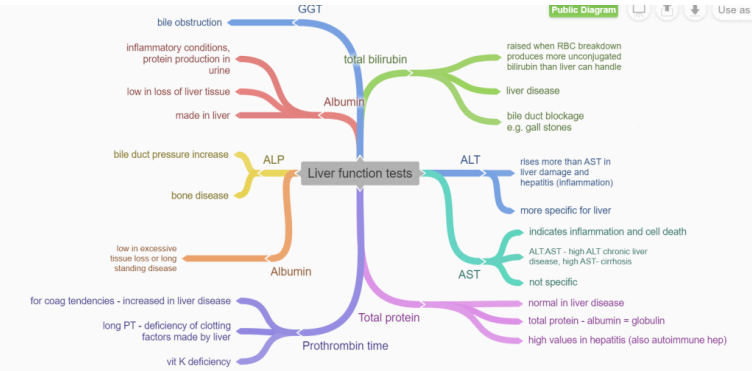
Examples

Function	Dysfunction
Store energy & maintain blood glucose	Weak, fatigued (no energy), seizures
Store vitamins B12 & C (& ADEK)	Skin, bone, urinary, gut, lungs, teeth, & gum disorders, anemia
Produce bile	Steatorrhea, Vitamin ADEK deficiencies
Produce clotting proteins	Bleeding disorders
Produce albumin	Edema, ascites (excessive fluid) due to low protein
Produce immune factors	Frequent infections
Filter and detoxify blood	Neurologic signs
Remove old rbcs	Icterus
Remove ammonia	Neurologic signs
Metabolize drugs	Ineffective drugs, toxic effects

3. Liver function testing

With liver dysfunction, problems can arise with coagulation, protein levels, energy levels, and toxins.

Liver damage can be assessed through analysis of liver enzymes and products as well as by liver function "tests". We look for loss of cellular contents, changes in liver function and signs of liver dysfunction.



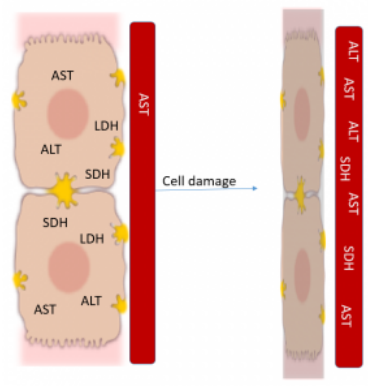
https://coggle.it/diagram/W-sS5oFGxH-0R2dM/t/liver-function-tests

SERUM MARKERS

Leakage enzymes = hepatocellular damage

AST* and ALT will be elevated with hepatocyte damage, while ALP

and GGT are more typically elevated with bile stasis or bile duct pressure.



AST, ALT, LDH, and SDH (transaminases) exist within the hepatocytes and are released into the blood stream when the hepatocytes are damaged.

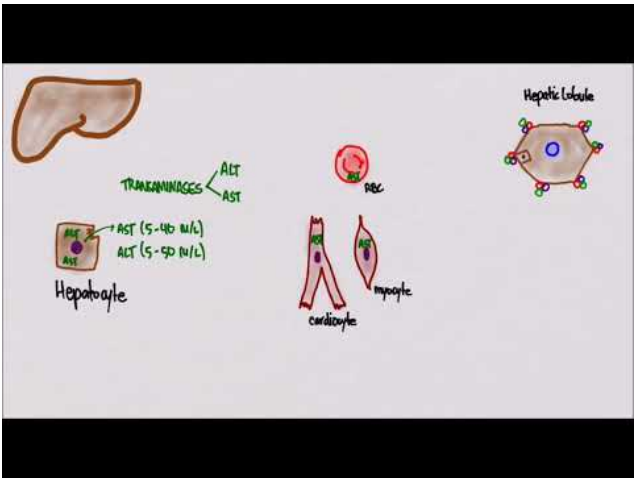
In dogs and cats, ALT is the most useful as it is much more liver specific. However, it can be released from muscle cells too so doesn't necessarily indicate liver damage.

Horses and ruminants have low levels of ALT in hepatocytes so it isn't very useful. SDH is much more useful in those species.

AST is also released from muscle cells in all species but be used to identify hepatocyte damage if muscle enzymes are low.

GDH (GLDH) is used in other countries but is not routinely available in the US.

All leakage enzymes values may also be **normal** in end stage (cirrhotic or scarred) liver disease as the cells have already released theses factors.



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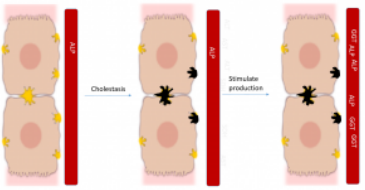
From this video

Induced enzymes = biliary stasis



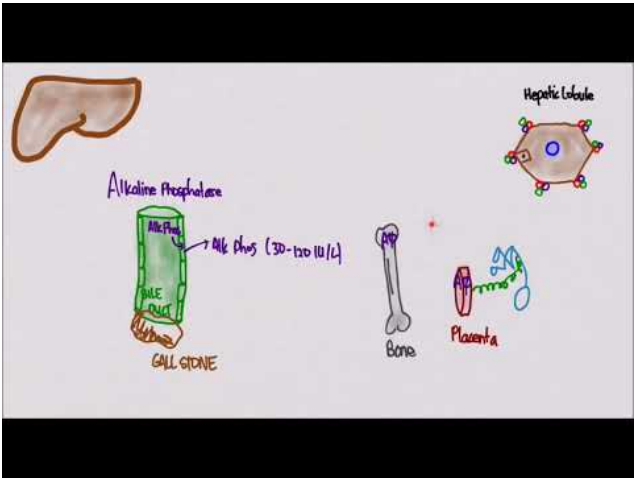
ALP and GGT are produced with biliary stasis. Liver levels are normally low when everything is working well. ALP is also produced in bone, kidney and placenta and is further stimulated by corticosteroids. ALP levels are normally elevated in young growing animals.

GGT is considered to be an induced enzyme but can be released

with acute hepatocellular damage. Many other cell types also produce GGT, especially the pancreas and the kidney. However, the kidney and pancreas elevations do not usually result in increased serum levels of GGT.

GGT is considered more specific but less sensitive than ALP for liver damage in dogs. This means you can have liver damage and normal GGT levels, but if you see GGT levels, you can be pretty sure you have liver damage. In cats, GGT is more sensitive but less specific. GGT will go up with mild liver damage but other things can cause elevations. Generally we perform both tests in small animal species.

In large animal species, just GGT is used in most cases. Keep in mind that high serum GGT in the colostrum can lead to related increases in neonates.



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LIVER FUNCTION TESTS

More specific liver function evaluation includes measuring prothrombin (clotting function), albumin, and bile acid levels.

Serum bile acids

Bile containing bile salts is released into the duodenum in response to elevated cholecystokinin (CCK) and secretin levels. Secretin is released when the pH in the duodenum is lowered (more gastric contents). Secretin stimulates biliary cells to produce bicarbonate and water, expanding the volume of bile and increasing biliary flow. CCK is released when fat-containing chyme is present in the duodenum and it stimulates gall bladder contraction and biliary flow.

The released bile aids in fat emulsification and absorption. Some of the bile salts are reabsorbed passively in the proximal intestine but most are actively transported back into the blood in the ileum. 90-95% of the salts are returned to the liver. In the liver, the bile salts are extracted and reused. This process repeats 10-12x per day, creating the **enterohepatic cycle.** Small amounts of bile salts escape absorption and pass into the colon where they are broken down by bacteria. These products create the brown stool color and yellow urine color.

Increased serum bile acids occur with

1. abnormalities in the portal circulation – congenital or acquired vascular shunts move blood away from the liver, preventing the first pass metabolism and impeding removal of bile acids from the circulation. Bile acids stay in the systemic circulation
2. decreased functional hepatic mass – diffuse liver disease results in impaired uptake of bile salts from the portal vein

3. decreased bile acid secretion in bile – any cause of cholestasis can lead to back pressure and move bile acids back into the circulation

Bile acids are very sensitive indicators of cholestasis and liver damage. Any jaundiced patient will have elevated serum bile acids but they can be useful in determining the cause of the jaundice and help with identifying diffuse liver damage. Generally bile acids are measured both before and after eating in dogs and cats; a single sample is adequate in herbivores.

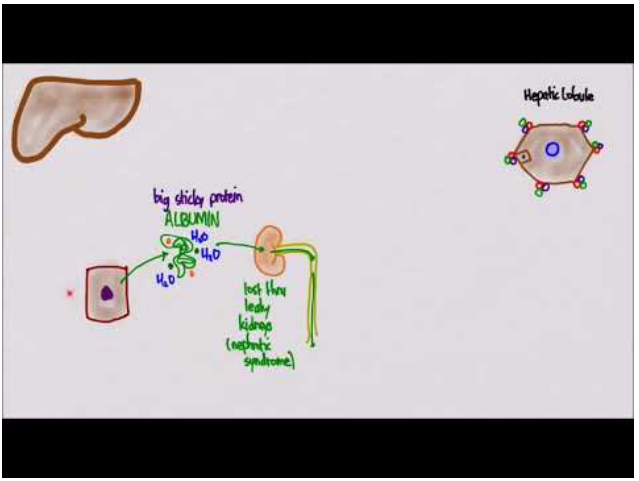
Increases in fecal bile acids are indicative of malabsorption, which can cause diarrhea. However, our ability to accurately assay fecal bile acids is currently questionable.

Plasma ammonium levels

Ammonia is produced by intestinal microbes, reabsorbed into the blood and removed by the liver. The liver uses the ammonia for urea and protein synthesis. Ammonium levels are elevated with changes in hepatic blood flow and decreased functional hepatic mass. Levels can be elevated by strenuous exercise, urea toxicity and other conditions but not with cholestasis. This means ammonia levels provides a nice comparison to bile acid levels. Ammonia measurement is not a very sensitive test but is useful to measure in cases of suspected **hepatic encephalopathy.**

Other liver products

Albumin is produced by the liver. Hypoalbuminemia (low albumin levels) is quite common in dogs with loss of 60-80% of liver function. It is not as commonly seen in horses.



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Globulins are also produced by the liver but may be elevated in liver disease. Elevations in liver disease are likely due to less processing of foreign compounds by the hepatic Kupffer cells, resulting in an increased immune response.

Glucose levels may be either decreased, increased or normal, depending on other mechanisms involved.

Urea is produced in the liver and may be decreased with liver disease; however, urea can be decreased for many reasons unrelated to liver disease.

Cholesterol is produced in the liver. Levels may be low, normal or elevated and depend in part on if cholestasis is a component of the liver dysfunction.

Clotting factors are produced in the liver. Liver dysfunction can lead to coagulopathies and prolonged bleeding times due to low levels of clotting factors.

CLINICAL SIGNS

Loss of liver function can lead to clinical signs, including

- pruritis due to accumulation of bile salts in the skin
- pale smelly fatty feces due to loss of fat absorption
- petechiation, brusing and/or bleeding due to limited production of coagulation proteins and/or storage of vitamin K
- hepatic encephalopathy (neurological signs) due to accumulation of toxic products and limited urea cycle activity
- seizures due to changes in glucose regulation
    - see youtube video
- poor growth due to limited nutrient storage
- icterus or jaundice due to bilirubinemia
- edema or ascites due to changes in colloid oncotic pressure

Key Takeaways

Test	Indicates
AST/ALT/SDH elevated	hepatocellular damage
ALP/GGT elevated	biliary stasis or damage
Serum bile acids elevated	liver damage
Albumin low	liver failure
Prothrombin (bleeding) times prolonged	liver failure
Ammonia elevated	liver failure

Pattern recognition is important

Hepatocellular damage (liver parenchymal damage)			Cholestasis (gall bladder/bile duct damage)		
ALT/ AST/ SDH	GGT/ALP	serum bile acids	ALT/AST/SDH	GGT/ALP	serum bile acids
increased	normal	high	normal	increased	high

End stage liver disease may be accompanied by normal AST/ALT as all cells have released their contents. Diagnosis may be via low albumin, high ammonia, prolonged bleeding times, and high serum bile acids.

*Abbreviations
ALT = alanine aminotransferase
ALP = alkaline phosphatase
AST = aspartate aminotransferase
GDH = glutamate dehydrogenase
GGT = gamma glutamyltransferase
SDH = sorbital dehydrogenase

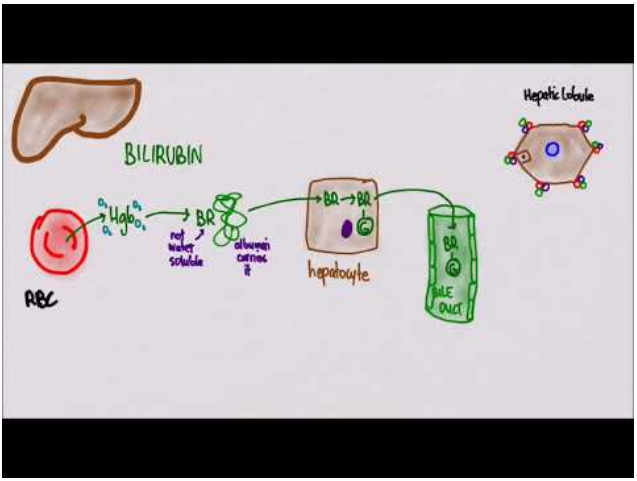
Resources

Veterinary Hematology and Clinical Chemistry – very useful ebook
Eclinpath – liver– webpage, explains multiple tests
Liver function tests– video
Important liver values – video

4. Bilirubin

Bilirubinemia means high levels of bilirubin in the blood and this gives the appearance of jaundice. **Jaundice** appears when bilirubin levels are elevated. Jaundice is most readily observed in the sclera and mucous membranes.

Bilirubin is produced with red blood cell breakdown. Normally, bilirubin is bound to albumin and carried to the liver where it is conjugated and excreted in the bile.



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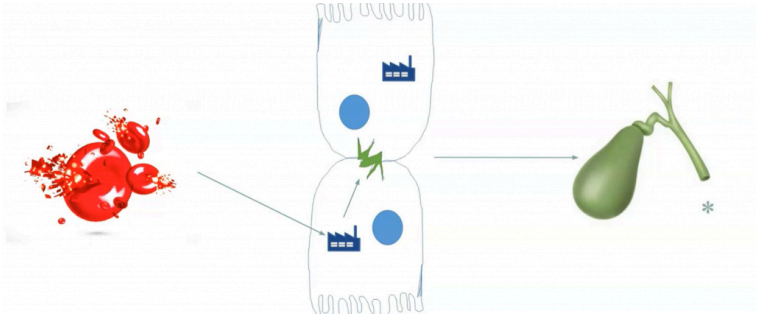
From this video

Bilirubin metabolism requires both functional hepatocytes and functional biliary system.



Unconjugated bilirubin is conjugated in the machinery of the hepatocyte, sent into the bile and then dumped into the lumen of the small intestine

Bilirubin levels can be elevated if

- too much unconjugated bilirubin hits the hepatocytes
- the liver is damaged and leaks bilirubin back into the blood because it can't metabolize it well
- biliary obstruction creates back flow into the circulation

Bilirubinemia can be sorted into three forms related to those three causes:

1. Pre-hepatic bilirubinemia
    - This is generally due to a relative excess of bilirubin delivered to the liver.
    - Hemolysis is a common cause.
    - In this version, more (unconjugated) bilirubin reaches the liver than can be conjugated.
    - This leads to excess unconjugated bilirubin in the blood
    - The liver is fine, the biliary system is fine; the system is overloaded
2. Hepatic bilirubinemia

- This can be due to congenital or acquired hepatic issues.
- The hepatocytes cannot manage to transform the unconjugated to conjugated bilirubin in sufficient amounts.
- Most of these disorders will result in high levels of unconjugated bilirubin but may also leak conjugated bilirubin due to related liver damage

3. Post-hepatic bilirubinemia

    - Bilirubin is conjugated by normal liver machinery; however, it can't be excreted because of an obstruction in the biliary system
    - Back pressure in the biliary system can lead to leakage of conjugated bilirubin
    - Cholestasis from flukes, stones, tumors can cause this
    - Surgery is potentially indicated for post-hepatic bilirubinemia.

Horses are different

Horses get jaundiced just from being off feed! This form is related to the diet components but is NOT associated with bilirubinemia. Horses do get elevated bilirubin when off feed and without liver damage; this is unconjugated bilirubin. The theory is they don't have enough glucose to do the conjugation. Conjugated bilirubin should be normal unless there are other issues.

Resources

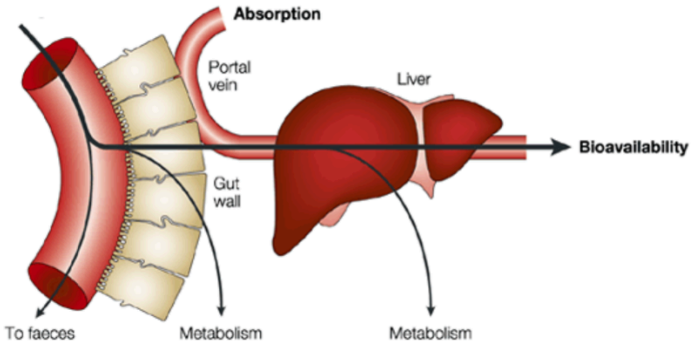
Veterinary Hematology and Clinical Chemistry – very useful ebook

Jaundice – osmosis video
Jaundice explained – 5 min video
Biliary excretion of waste products– CSU vivo site

5. Drugs and the liver

The liver metabolizes the majority of drugs administered orally and most that need to be metabolized to be excreted. This may involve activation or inactivation or drugs and making drugs water soluble so that they can be excreted.

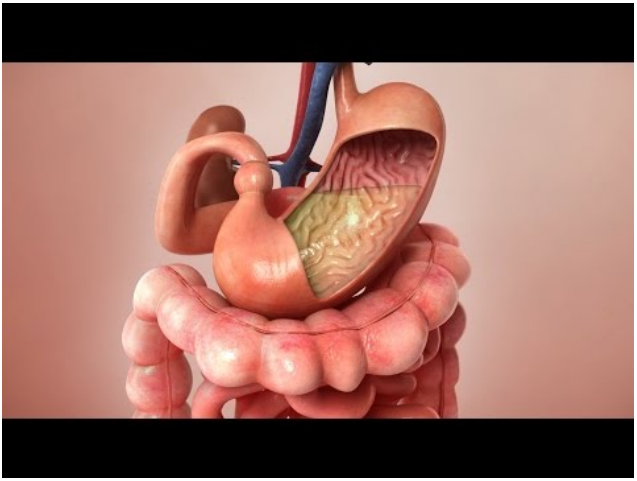
First pass effect- The large reduction in concentration of an orally absorbed drug due to passage of the drug through the liver. Some drug will be lost during absorption from the intestine and more from metabolism in the liver. Remember, the liver sees things as toxins and tries to detoxify them. Drugs are included in that list of things that the liver tries to remove.



https://canna-pet.com/first-pass-effect/

Bioavailability– the fraction of the drug that escapes the liver and reaches the systemic circulation due to the first pass effect. If a drug is given intravenously, it is considered to have 100% bioavailability since it is all in the blood stream. The same drug given orally will have less bioavailability since some is never absorbed

(stays in the gut and ends up in the feces) or is modified by the liver before it hits the vascular system.



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Cytochromes P450 (CYPs) are hemeproteins used extensively by the liver to metabolize drugs. The types and amounts of CYPs vary by species, sex and genetic makeup. Changes in CYPs result in different levels of drug effectiveness and toxicity. Differences in cyp450 is responsible for the unique responses of horses and cats to certain drugs.

Drugs can interact with CYPs in 3 different ways.

- Drugs may just be metabolized by the CYPs. Generally this makes drugs less available to the animal as it leads to faster excretion.
- Drugs may inhibit CYP activity. Erythromycin and omeprazole

inhibit CYP activity. This prevents the CYPs from metabolizing other drugs being administered to the animal, resulting in abnormally high levels of the unmetabolized drugs.
- Drugs may induce CYP activity. Phenytoin, phenobarbital, griseofulvin and rifampin are CYP inducers. When they are present, the CYPs metabolize other drugs at a much higher rate, resulting in low working levels of those drugs.

Obviously it is crucial to know the full list of drugs being administered in order to check for interactions. Consult with a pharmacist if unclear!

Liver disease and drugs

Drug administration needs to be carefully considered and monitored in animals with hepatic damage. Drugs can be more active or less active in an animal with liver disease.

When the liver metabolizes drugs, it can activate or inactivate drugs. With liver disease, drug may not be activated or deactivated as much as usual. Albumin binds active drug and keeps it from working. More active drug may be present in liver failure due to low albumin levels. The liver also conjugates drugs to excrete them. If it can't conjugate properly, the drug builds up in the body.

Most antibiotics are metabolized by the liver. In humans with liver disease, care must be taken with sulfas and with clavulanic acid. Penicillins and cephalosporins are considered safe; regular doses of fluroquinolones and tetracyclines are also low risk.

Drugs can also damage or add to hepatic damage in three different ways :

- immune response to the drug / idiosyncratic reactions
- toxic metabolites that cause more damage
- interactions with cytokines activated by liver damage

Drugs that can cause more damage include clavamox, contraceptives and ibuprofen.

Resources

The liver -function – good focus on drugs and toxins

Cytochrome P450 – 1 page monograph

What are cytochrome P450 enzymes? – includes info on grapefruit juice

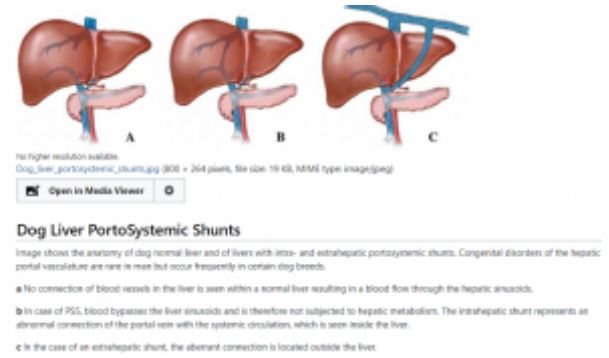
Inducers and inhibitors – video designed for med students

6. Liver shunts

Pathophysiology

Blood is supposed to move (1) from the intestines to (2) the liver to (3) the rest of the organs and then (4) to the heart to be oxygenated. In a normal animal, the liver filters out the nutrients and toxins prior to the blood going to the other organs.

In some animals, blood bypasses the liver through "shunts". These shunts can be outside the liver (extrahepatic) or inside the liver (intrahepatic).



https://embryology.med.unsw.edu.au/embryology/index.php?title=File:Dog_liver_portosystemic_shunts.jpg

Congenital shunts more commonly affect small breeds (Miniature Schnauzers, Yorkshire terriers, Maltese, Pugs and Cairn terriers).

Animals with congenital liver shunts are not able to use the nutrients from the gut and are often the runts, are small in stature, and/or don't grow well. Dogs may be otherwise healthy or have vomiting or polyuria/polydipsia. Hepatic encephalopathy can develop from high ammonia levels (no urea cycle) as can ammonium

urate stones in the urinary tract. If anesthetized, affected animals may be slow to recover from the drugs. Cats more typically show drooling and seizures. Cats often have copper colored eyes.

Diagnosis

Liver enzymes are often normal. Animals may be anemic and hypoglycemic. Serum bile acids are the most useful for testing liver function in these animals and are almost always elevated.

Therapy

Surgery is an option for many of these dogs, with the shunt clamped closed slowly over time so new blood supply develops.

Medical treatment includes fluids and gut protectants to prevent ulcers. Reduction of blood ammonia concentrations can be achieved through warm water enemas, dietary protein reduction, lactulose and antibiotic therapy.

> **Lactulose** is a nonabsorbable disaccharide that is digested by the GI microflora to produce short chain fatty acids. As a result, the colonic pH decreases and NH4+ concentrations increase, which is less absorbable than ammonia. Antibiotic treatment lowers the intestinal bacterial load and may decrease ammonia production. Severe dietary protein restriction can be bad as the liver is already not producing enough

albumin. Some animals may need antiseizure meds.

Resources

Portosystemic shunts in dogs, VCA
Portosystemic shunts, K Tobias
Portosystemic shunts, ACVS
Liver shunts, UTenn

7. Hepatic lipidosis

Pathophysiology

Cats , camelids, and cows tend to get fatty liver when they are off feed or in a negative energy balance. Miniature horses can do this too. Pregnancy toxemia in sheep is related. **Obese animals** are particularly at risk.

The disorder is very common in cats and is responsible for almost half of the cases of feline liver disease. This is the biggest reason why cats should not be allowed to become overweight. A sick cat tends not to eat and this leads to fat mobilization and hepatic lipidosis.

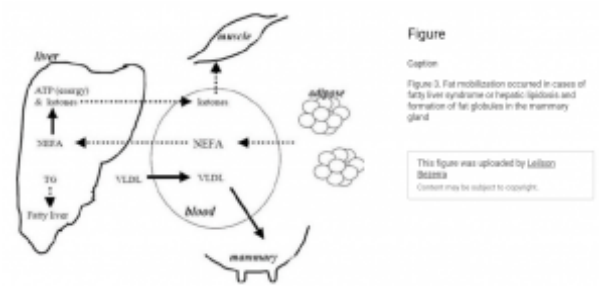
In cattle, this generally occurs in early lactation as the cows cannot eat enough to meet their metabolic demand and try to mobilize fat stores. In animals affected with hepatic lipidosis, metabolic derangements and liver dysfunction occur due to increased mobilization of fatty acids.

- Hyperlipemia occurs when triglycerides are not removed from the blood as fast as they are produced.
- Lipidosis occurs when triglyceride synthesis exceeds formation and release triglycerides into circulation

Normal physiology

With lipolysis, serum free fatty acid (FFA) concentration increases. These FFAs are absorbed by liver cells and metabolized. Some go through carnitine dependent oxidation and produce energy or ketone bodies. In this pathway, triglycerides are produced and stored in hepatocellular vacuoles. They get secreted into the

circulation after incorporation into very low density lipoproteins (VLDLs).



https://www.researchgate.net/publication/272774956_Major_Metabolic_Diseases_Affecting_Cows_in_Transition_Period/figures?lo=1

Lipidosis

With lipidosis, several things could alter the pathway

1. These animals may have insufficient protein to form VLDLs which are important for removal of excessive triglycerides
2. Relative amounts of carnitine may be too low to transport fatty acids thru the liver
3. Insulin resistance. Normally fat breakdown occurs in response to insulin. Poor insulin function may lead to continued lipolysis in the face of energy restriction.

Current thinking is protein deficiency combined with insulin issues.

Diagnosis

Animals become anorexic, lose weight, lose muscle mass, and become icteric. Cattle drop in milk production. The liver enlarges as it fills with fat.

Serum liver enzymes increase due to hepatocellular liver damage. Cholestasis occurs as the bile ducts get filled with lipids and collapse.

Liver ultrasound and/or biopsy is diagnostic in cattle.

Therapy

Treatment in cattle includes providing iv glucose, insulin, vitamins and potentially steroids. Rumen transfaunation may also be useful.

General resources

Hepatic lipidosis in cattle, Purdue, 2003
Clin path evaluation of hepatic lipidosis in cattle, JVIM, 2008
Prediction and diagnosis of fatty liver in dairy cattle, SMJGH, 2017

Resources – Hyperlipemia in horses

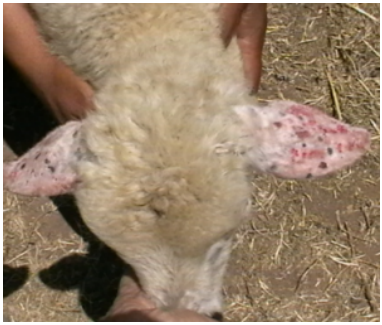
Hyperlipemia in donkeys, The donkey sanctuary
Equine hyperlipidemias, VCNA 2011
Hyperlipemia mini horses and donkeys, JVIM 1994
Severe hypertriglyceridemia in clinically ill horses, EVJ 2003

8. Photosensitization

Primary Photosensitization

Author : Marion Karhatsu

Primary photosensitization occurs when a photodynamic agent is ingested, injected or absorbed through the skin. In animals, this is often related to the ingestion of plants that contain photosensitizing agents. The skin becomes abnormally reactive to ultraviolet light exposure (especially skin not protected by hair, fur or pigment) through the production of unstable, high-energy molecules created by the interaction of light photons with the agent. The resultant cell damage can cause edema, skin ulcerations and necrosis. Primary photosensitization is basically a toxin-induced, severe sunburn.



Clinical signs include severe irritation, restlessness, rubbing, as well as signs of sunburn: redness and swelling of affected areas followed by sloughing skin. Secondary skin infections are possible. Symptoms often first appear on the tips of the ears and are mostly localized to white or lightly pigmented areas. Treatment consist primarily of supportive care and avoiding sun exposure. Corticosteroids may be needed to address strong inflammatory responses. Lesions may resolve within 48 hours, but can take up to 6 months, depending on severity.

Plants that contain primary photosensitizing agents include *Hypericum perforatum* a.k.a. St. John's wort (agent: hypericin) and *Fagopyrum esculentum* a.k.a. buckwheat (agent: fagopyrin). Plants of livestock and poultry veterinary importance are those belonging in the Umbelliferae and Rutaceae families. Examples are *Ammi majus* (bishop's weed) and *Cymopterus watsonii* (spring parsley) that produce photosensitization in cattle and sheep, respectively (agents: furocoumarins/psoralens). Ingestion of *A majus* (snapdragons) and *A visnaga* (toothpick plant) seeds have produced severe photosensitization in poultry.

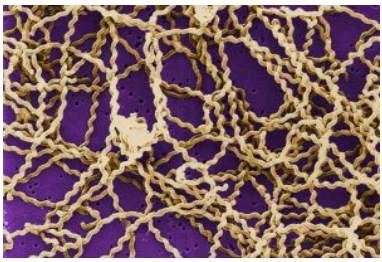
The condition is best prevented by good pasture and feed management to avoid the consumption of photosensitizing agents.

9. Leptospirosis

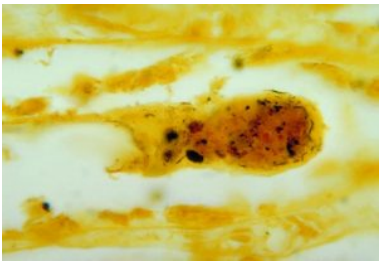
Leptospirosis

Author: Bradley Fountain

Leptospira interrogans, the most common and persistent pathogen species responsible for the disease leptospirosis, is a Gram negative spirochete endemic to most of the world, especially in areas with standing water or seasonal floods. The species contains over 200 known pathogenic serovars and is very zoonotic, transmittable between different animal species and to humans with no significant barriers. The bacteria are shed through the urine. Animals are infected by drinking contaminated water and humans through mucosa or broken skin exposed to the same. The bacteria then travel through the blood and multiply in a variety of organs, causing tissue damage through a mechanism likely related to the organism's lipopolysaccharide coating. The immune system is largely capable of clearing the bacteria from the body except in the kidney tubules, where the bacteria persists and continues to multiply, being shed once more in urine.



Leptospira scannning micrograph



Leptospirosis in the kidney

As a disease, leptospirosis is only partially understood. In humans, the bacteremia caused by initial infection brings on flu-like symptoms. This is followed by a brief period of pseudo-latency when the bacteria are sequestered in organs and begin multiplying, after which signs of disease re-emerge due to related tissue damage. In domestic animals the bacteremic stage can be difficult to detect, as symptoms can be as general as mild malaise or lethargy and moderate fever. If a veterinarian is consulted at this stage, a thorough history and the availability of diagnostics will help determine the outcome. The history can help determine the risk an animal is at for leptospirosis, such as contact with wild animals or potentially contaminated water sources. Routine bloodwork will not pick up a *Leptospira* infection, but can point towards a general bacterial infection that may be treated with appropriate antibiotics. The two of these together may lead a veterinarian to run a serology for leptospirosis, and treat accordingly. Once the disease has progressed to tissue damage the danger becomes more acute, not just for the animal but for the humans working or living with them, and medical intervention not just to treat the animal but establish quarantine protocols is imminently necessary.

While much of the pathology in the disease is concentrated on its occupation of the kidneys, the most common clinical presentation of advanced disease is due to liver damage. The pathogen migrates through the blood, which means large amounts of it can pass into and through the liver, causing wide spread damage. This pathology is evident in many of its common names, such as “rat-catcher’s yellows” and “black jaundice.” In ancient China it was the likely

cause of "rice field jaundice." The jaundice or icterus is caused by hepatocellular damage. This leads to the inability of the liver to process hemoglobin, causing a build-up of unconjugated bilirubin in the blood. However, the true danger is in compromise of the other functions of the liver, including toxin metabolism.

Currently there is no human vaccine for *Leptospira* recognized by the CDC, although there are other countries using them. Vaccines for dogs are in common use in the US, providing immunity for up to four known common serovars. Besides these measures, the best means of prevention is avoiding wild animal urine-through direct contact or through contaminated water, and controlling rodent populations which are considered lifetime reservoirs. As climate change affects areas of flooding and severe weather, further efforts will need to be made to control this already widespread disease.

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Leptospirosis; prevention; information; Adventure Racing – available at https://www.cdc.gov/leptospirosis/index.html Accessed Apr 5, 2019

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Namroodi S, Shirazi AS, Khaleghi SR et al. Frequency of exposure of endangered Caspian seals to Canine distemper virus, Leptospira interrogans, and Toxoplasma gondii. *PLoS One*. 2018;13:e0196070

Wang Z, Jin L, Węgrzyn A. Leptospirosis vaccines. *Microbial Cell Factories*, 2007;6:39

Xu Y, Ye Q. Human leptospirosis vaccines in China. *Human Vaccines & Immunotherapeutics*, 2018;14:984-993

10. Resources for Liver disease

Advanced Physiology

Veterinary hematology and clinical chemistry, Thrall – free ebook through UMN library; very useful for diagnostics

Bilirubin, eclinpath- veterinary

Hepatic safety of antibiotics used in primary care, 2011, human and TB oriented but nice summary

Just for fun

Liver disease song, human version

Approach to the jaundiced cat, 2015

The yellow cat, Todays veterinary practice

Causes of equine hepatic disease, DVM 360, 2009

Equine liver in health and disease, The Horse, 2016

Genetic disorders in sheep, 1990 VCNA – includes congenital bilirubin issues

Grapefruit and medications– more cytochrome P450

Rifampin and contraceptives – this one makes sense now

Porcine circovirus infections-Vet Path, 2000

Cholecystitis in people – Osmosis

Copper toxicity – sheep get copper toxicity; no idea of related pathophysiology

11. Liver exercises

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Challenge Exercises

General

- Would a compound like amino-pro really work?
- What diet would you recommend if your favorite species had liver failure?
- How does ammonia cause hepatic encephalopathy?

- What happens with high levels of serum bile acids in the body?
- What proteins are involved in coagulation?
- How do we use enzyme half lives to determine when liver disease happened? (ALT, AST, ALP)
- Explain common signs associated with liver disease
- How does liver disease lead to photosensitization? What is photosensitization?
- What is the role of cytochrome P450?
- Find and interpret abnormal lab results related to liver function
- Why can't we drink grapefruit juice while on antibiotics?
- How do antibiotics interfere with the Pill?
- How do you evaluate the liver ultrasonographically?
- Create a mnemonic for common causes of liver disease in your favorite species

Dogs

- What are congenital disorders of the liver (besides shunts)?
- How does copper affect liver function?
- Explain 3 toxin (or infection or drug or other) causes of liver disease in dogs
- How do SAM-E and milk thistle help with liver disease?
- What are the most effective tests for liver disease in dogs?
- How would you treat xylitol toxicity?

Cats

- Explain 3 toxin (or infection or drug or other) causes of liver disease in cats
- What are the most effective tests for liver disease in cats?
- Explain tylenol toxicity in cats
- What breeds are prone to liver disease?

Horses

- Explain 3 toxin (or infection or drug or other) causes of liver disease in horses
- What are the most effective tests for liver disease in horses?
- How do choleliths affect horses if they don't have gall bladders? What does CCK do?
- How does tetanus antitoxin cause liver disease?
- Toxic plants in North America include cocklebur, Lantana and Helenium species. What do these do to the liver?
- Iron injections can kill foals. How and why?

Ruminants

- Explain 3 toxin (or infection or drug or other) causes of liver disease in ruminants
- What are the most effective tests for liver disease in ruminants?
- How do we treat liver disease in ruminants?
- How does moldy hay affect ruminants?
- Farms tend to have more chemicals around. Which ones are hepatotoxic?
- Cattle tend to be liver abscesses. What are the risk factors?

- What is bacillary hemoglobinuria?
- What happens with copper toxicity in sheep?

Swine

- What is postweaning multisystemic wasting syndrome? What might labwork look like?
- What are liver milk spots?
- Farms tend to have more chemicals around. Which ones are hepatotoxic?

Pocket pets

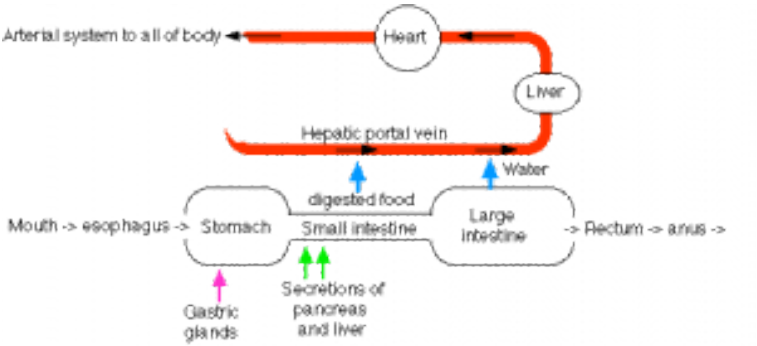
- Which pocket pets get fatty liver?
- What causes liver abscesses in chinchillas? How are they treated?
- What is Tyzzer's disease in gerbils and hamsters? How is it treated?
- What is amyloidosis in hamsters?
- What kind of liver disease do sugar gliders get? How is it diagnosed?

PART II

DIGESTION AND THE PANCREAS

Accessory organs

12. Energy and growth



A surgeon's view of the gastrointestinal tract

Requirements for growth

Many things have to happen for the nutrients we eat to actually turn into energy and components that we can use for growth.

We have to eat the right things and in the right proportions

- Veterinary diets are formulated to solve this problem. The name brand foods (with a nutritionist involved) include essential vitamins and minerals as well as the right balance of proteins, carbohydrates and fats. If pets or livestock eat diets developed by nutritionists, they are usually eating the right things in the right proportions. When we change that diet, we can have issues.
- Diet fads can impact animals. Raw diets are not healthier than dog chow. Cat are obligate meat eaters; dogs are not. Dogs do

not need to eat "low carb". Cats cannot survive as vegetarians.

We have to eat enough food to meet metabolic demands

- We need to be ingesting enough calories to meet the body's demand or we have a "negative" energy balance. Higher energy needs are required for growth, lactation, extra work, illness, neoplasia and the last trimester of pregnancy.
- Nutrients may be lost through various disorders. In particular proteins can be lost through the skin (burn wounds), the GI tract, and the kidneys. If proteins are being lost, more are required in the diet.

The things we eat have to be turned into digestible particles- chewing and swallowing

- Chewing helps break food material up so it can be broken down further more effectively. Older animals may not be able to chew as well. Certain dietary components are also harder to chew or to break down (straw, older hay, corn)

The material has to move through the GI tract

- Most digestion typically occurs in the small intestine. If food isn't eaten, is regurgitate or vomited or get stuck, it may not reach the areas required for digestion.

Digestive enzymes must break down food into its basic components for absorption

- We don't have transport mechanisms for larger proteins, fats or sugars. We can absorb disaccharides, monosaccharides,

monoglycerides, fatty acids, amino acids and some peptides. We use salivary (in some species) and pancreatic enzymes to break larger food molecules down.
- Activation is required for many enzymes to prevent self-digestion. Bile is required for fat digestion. Multistep processes mean more things can go wrong.
- Segmentation (mixing) activity also helps mix the enzymes with the food. If the intestine isn't contracting properly, digestion as well as absorption can be impaired.
- Different species have different enzymes and hence different dietary requirements.

Nutrients must be absorbed

- Absorption of nutrients mostly occurs in the villi (folds of tissue that increase surface area)
- The villi are designed so that the digestion products can easily reach the vessels and lymphatics by passive movement
- If the villi are too thick, damaged or otherwise dysfunctional, absorption may not occur even with proper digestion.
- When motility is sped up, absorption may not have time to occur.

Nutrients must be produced

- Gut microflora play in role in digestion too, particularly in ruminants. In ruminants, diets are designed to primarily feed the microflora. The microflora feed the cow.
- If the microflora are killed off or are abnormal, digestion of hays and grasses will be impaired. In addition, microflora produce certain nutrients that are not otherwise available in diets.

Nutrients must be distributed

- Most nutrients pass out of the GI tract and go into the portal vein system and to the liver before being disseminated through the body. The liver filters out toxins and may further metabolize dietary components. If the portal system or liver function is abnormal, the nutrients may not make it to the rest of the body. Fats must also be disseminated by the lacteals.

Causes of weight loss- Malone lecture

Challenge Questions

Pick one to think through or research:

- When do we(or other animals) gain weight vs lose weight?
- What are the current recommendations for endurance diets (dogs, horses, humans)?
- What vitamins and minerals are required(any species)? What happens with deficiencies?

13. Normal Digestion

Sites of Digestion

There are 5 main “sites” of digestion.

Oral cavity

Chewing and the production of saliva both help with digestion. Chewing breaks down food into smaller components and increases the surface area for digestive enzymes. Saliva in some species contains salivary amylase which start the process of **carbohydrate** digestion. Dogs, cats, sheep and goats do not have salivary amylase.

People (and many omnivores) have salivary amylase. Chewing starches for a longer period of time makes the food taste sweeter.

Stomach

Protein digestion starts in the stomach. Acid and pepsin break down proteins into polypeptides. Pepsinogen is produced by chief cells and is converted to pepsin by HCl (released from parietal cells). Once activated, pepsin can activate itself. Some animals produce gastric lipase, permitting some fat digestion in the stomach. Salivary amylase is inactivated by the stomach acidity.

Liver/gall bladder

Bile is required to digest **fats**. Bile salts are released from the liver and help break up fat globules, allowing them to be broken down into smaller components by pancreatic lipase. After monoglycerides and fatty acids enter the cell, they are recombined into triglycerides. The triglycerides are coated in proteins and transported into the lacteals as chylomicrons.

Image result for bile and fat digestion

Not all species have gall bladders but they all have biliary systems.

Small intestine and pancreas

The small intestine works similarly across species despite dietary differences. The main function of the small intestine is to move nutrients from the diet into the bloodstream. In a herbivore, this includes the non-cellulose parts of the diet (fats, proteins, carbohydrates from grains or oils, not hay).

Once foodstuffs enter the SI, they are mixed with enzymes and buffers from profuse pancreatic secretions and from the SI brush border. The enzymes (amylase, trypsin, chymotrypsin, lipase, carboxypeptidases, endopeptidases) start breaking down the food products into glucose, amino acids, free fatty acids and monoglycerides. Pancreatic enzymes are released in an inactive form (zymogen) and must be activated. Along with peptidases that can digest proteins directly, intestinal cells produce enterokinase which converts the pancreatic zymogen trypsinogen to trypsin. Trypsin then converts other zymogens to active forms. The process is enhanced by calcium levels in the duodenum. These enzymes work on **carbohydrates, fats and proteins**.

Motility and digestion

The intestinal wall contracts rhythmically to mix the ingesta with the enzymes (segmentation).

The simpler components are absorbed through capillaries and lymphatics in the intestinal wall where they can be transported to other organs for use or storage. At the same time, the ingesta is moved from the duodenum, through the jejunum and into the ileum. Gradually more and more of the ingesta is exposed to the gut lining and can be absorbed if it hits the right receptors. Most of the simple sugars and proteins are absorbed in the jejunum. If fats are in low concentration, they can also be absorbed in the jejunum. Excess fats are absorbed in the ileum. The ingesta will often be held and mixed in the ileum until the fat content is minimal (see "ileal brake").

During the process, any structural carbohydrates (hays, grasses, seeds) in the diet are not broken down but are being hydrated by active fluid secretion. Eventually the remaining components are moved into the cecum where microbial digestion will start.

The water exchange that occurs in the SI equals about 1.5x the total extracellular fluid volume of the animal over a 24 hour period. The SI Is the main site of water secretion and water absorption in all species. Colonic water absorption occurs (especially horse colons) but colonic water absorption is still are secondary to the SI.

Digestion by dietary component

Carbohydrates

Carbohydrates are primarily digested by pancreatic amylase in the SI.

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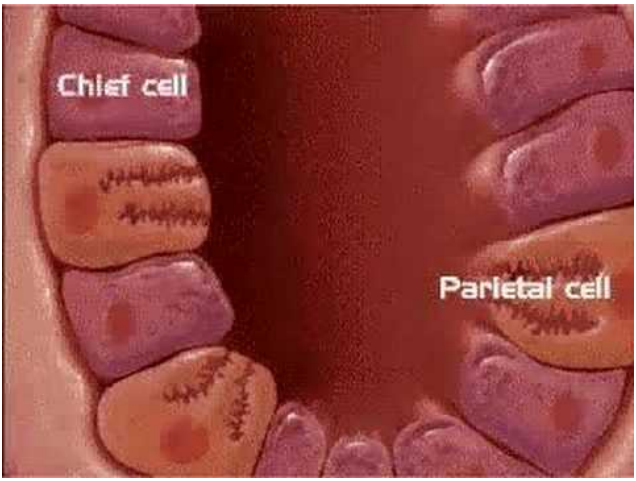
Proteins

Proteins are digested in the stomach (pepsin) and SI (trypsin and chymotrypsin).



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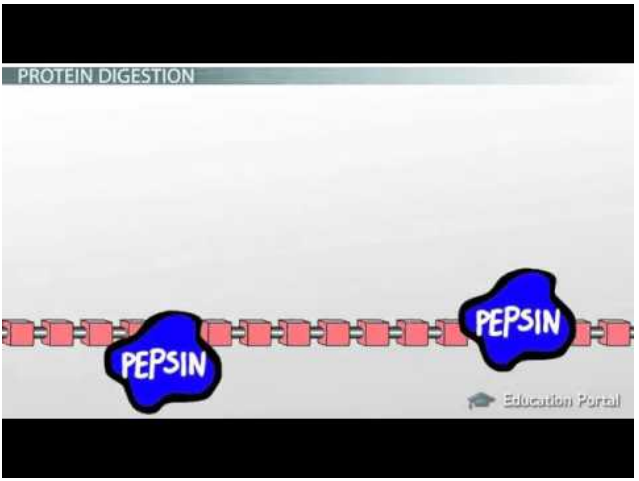

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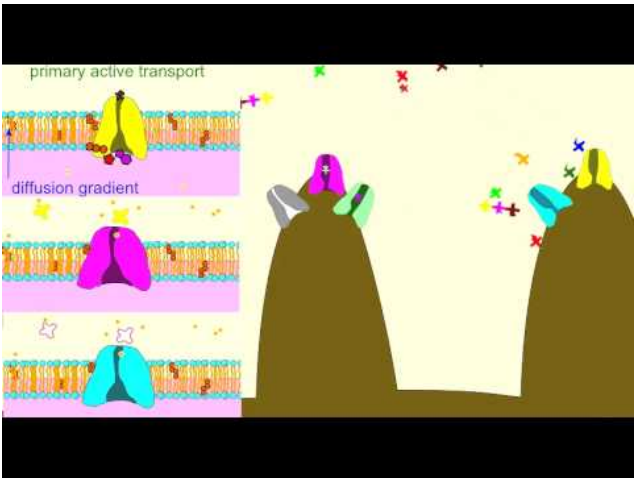

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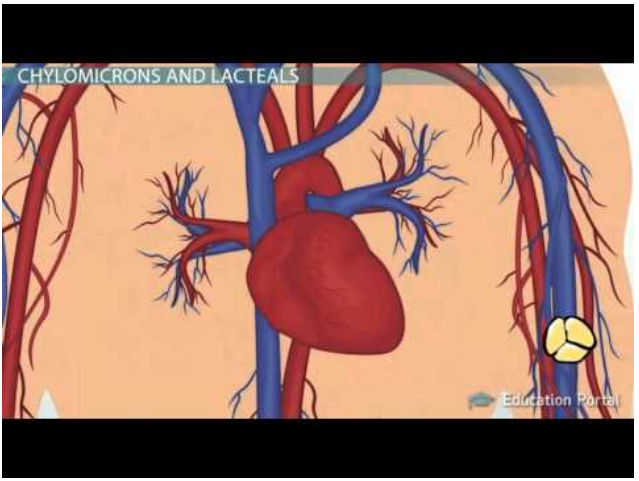
Lipids

Lipids must be emulsified by bile salts before they can be broken down in the SI by pancreatic lipase. The breakdown triglycerides are coated with proteins and absorbed into the lymphatic system.



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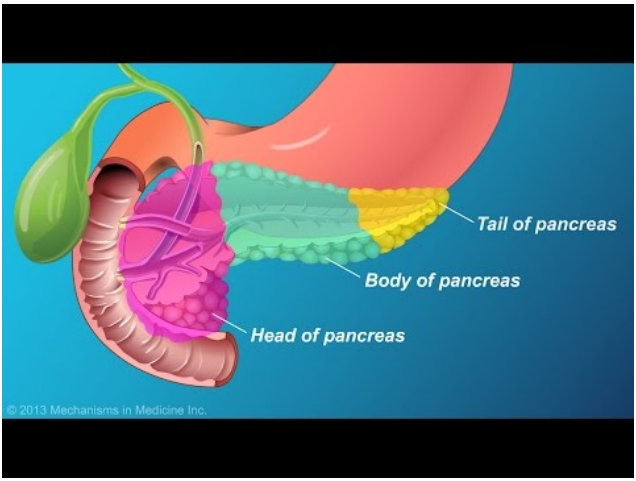

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Challenge questions

- If pandas are really carnivores, how do they eat bamboo?
- What makes feces brown? What would feces look like if the animal had no pancreatic enzymes?
- Why do some animals eat their own feces? Explain

copraphagy and cecotrophy.

Resources- Digestion

GI anatomy and physiology – Osmosis.org; covers it all including motility

Digestion overview – 3D horse digestion guide; equine but nicely done for all species

Comparison pigs, horses and cows – basic but cool shows how GI tracts compare

Pancreas function – Education Portal; great lecture review

Gastric secretions – will help connect to microanatomy

Dietary protein – drawittoknowit- great way to learn the material

What is bile – another good review of fat digestion

Digestive hormones – more if you are interested

Resources – Absorption

Absorption of nutrients – Khan Academy

Nutrient absorption and utliization by ruminants, CSU- I love their approach to physiology

Absorption of lipids, CSU

Control of the GIT-digestion and motility put together- Khan academy – really nice to come back to

Villi structure – really important for diffusion

Just for fun

Unsaturated vs saturated vs trans fats – Malone's favorite; now I get it

The digestive system – species comparison, fun to watch

Grain overload, UIll

Short bowel syndrome, 2006 Compendium

Aspirin absorption – relaxing, explains drug -protein binding, bioavailability and drug metabolism

The naked mole rat – The brainscoop; unique GI tract too!

Why pandas are bad at being pandas – they can't digest bamboo well at all

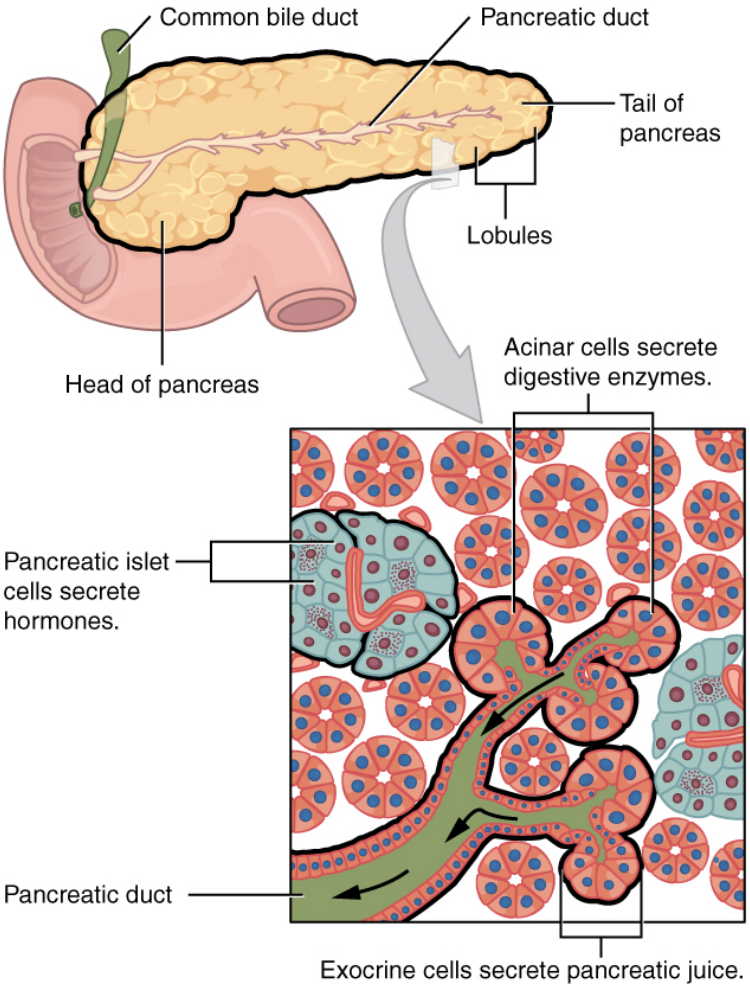
Why did pandas go vegetarian– this girl is just impressive

Protein digestion – fun version

Why you can't eat grass- video

Why is poop brown – video

14. Pancreatic function



https://commons.wikimedia.org/wiki/File:2424_Exocrine_and_Endocrine_Pancreas.jpg

General exocrine function

The pancreas has four main exocrine functions:

1. Enzymatic digestion of food

The pancreas stores the enzymes in zymogen (inactive form). Zymogens are stored in zymogen granules with other enzyme inhibitors.

When the zymogens reach the duodenum, trypsinogen is activated by the endopeptidases. Trypsin then further activates all enzyme types (amylase, trypsin, chymotrypsin, carboxypeptidase and lipase). The process is enhanced by higher levels of calcium in the intestines.

2. Production of antibacterial proteins

Bacteria are kept in control by proteins released from the pancreas. The bacteria are also impacted by gastric pH, other bacteria and peristalsis.

3. Neutralization of gastric acid

The pancreas releases sodium bicarbonate which helps neutralize the gastric acid that reaches the duodenum.

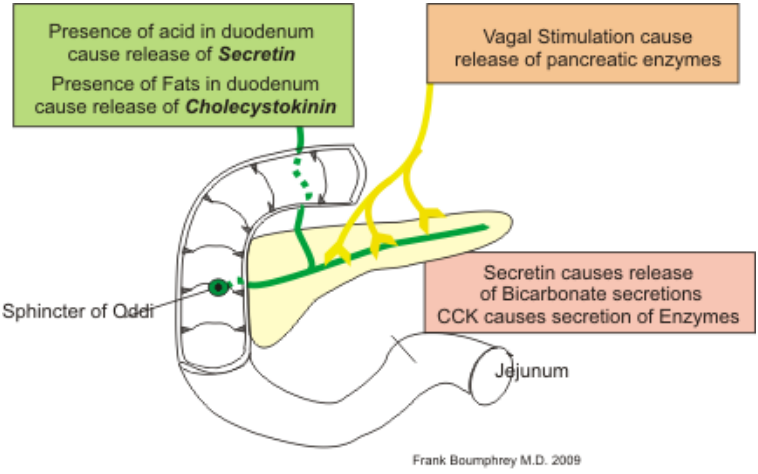
4. Production of intrinsic factor

Intrinsic factor is needed for absorption of cobalamin. In most of our species, it is primarily produced by the pancreas.

Cobalamin (vitamin B12) has a very complex absorption process. It must be first bound to an R protein. This is degraded by the pancreatic enzymes. Now it is bound by intrinsic factor which carry it to the ileum. Receptors in the ileum bind the intrinsic factor and enable absorption.

Cobalamin is required for proper red blood cell formation, brain function and DNA synthesis (basically all growth).

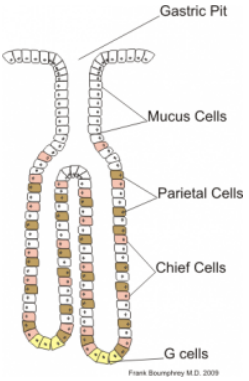
Control mechanisms



Control of Pancreatic Secretions

https://upload.wikimedia.org/wikipedia/commons/6/6e/Pancreas_secretion.png

Gastrin



https://commons.wikimedia.org/wiki/File:Gastric_gland.png

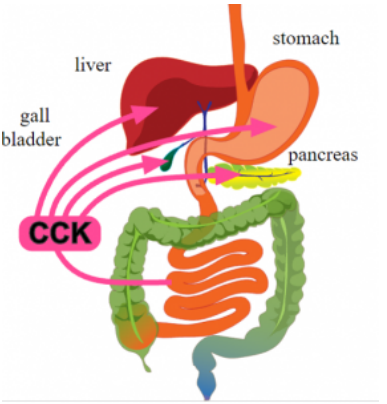
Gastrin starts the process of protein digestion by stimulating the release of HCl from parietal cells. Gastrin is produced by G cells in the gastric pits. It is released when there are higher levels of calcium and/or peptides in the stomach.

Cholecystokinin

Cholecystokinin (CCK) is produced by the duodenal enteroendocrine cells and stimulates the release of bile from the gall bladder and pancreatic enzymes from the pancreas. It is released due to the presence of partially digested fats and proteins in the duodenum.

Secretin

Secretin is produced in the duodenal S cells and stimulates the release of sodium bicarbonate from the pancreas. It also inhibits gastrin release from the stomach. It is released when the duodenal pH is low (acidic).



https://upload.wikimedia.org/wikipedia/commons/8/83/Effects_of_CCK_on_the_gastrointestinal_tract.svg

Resources

Pancreas function, enzymes and role in digestion – video
The pancreas– CSU vivo site

Just for fun

I am your pancreas

15. Pancreatitis

Preventing pancreatitis

The pancreas has high self destructive capacity. It contains enzymes that destroy protein and lipids. It is comprised of cells that have walls of protein and lipids.

Zymogens are activated by:

- Calcium
    - helps trypsin activation
- Proteases
    - released from lysosomal granules
- Trypsin
    - activates other enzymes

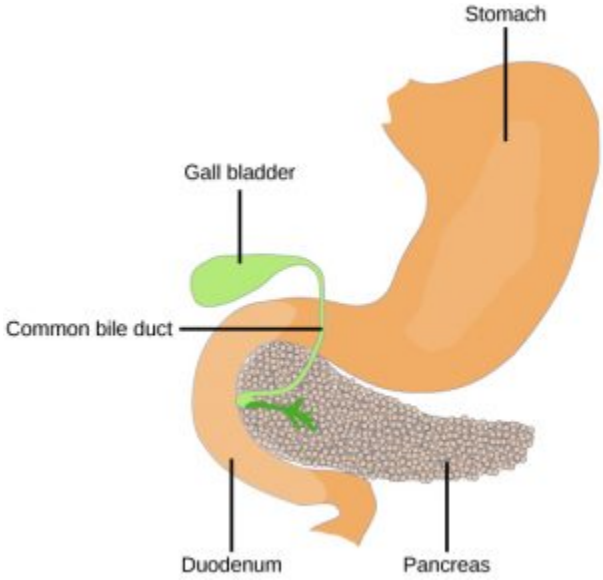
To prevent self digestion, several safety mechanisms are built in:

- Low calcium levels stabilize trypsinogen
    - keep it from changing into trypsin
- Trypsinogen inhibitors are also present in the pancreatic cell
    - SPINK – serine protease inhibitor
- CFTR secretes sodium bicarbonate and flushes the duct system
    - Move activated products into the duodenum
- Sphincter muscle at end of duct next to duodenum

Failure of the safety mechanisms leads to inflammation of the pancreas = pancreatitis

Potential activators

- Idiopathic
  - we don't know
- Ingestion of fatty foods
  - this probably leads to excessive release of CCK
  - more CCK means more trypsin; both can flow up the pancreatic duct
- Endocrine disorders
  - diabetes and pancreatitis are closely linked
  - which comes first?
- Inflammation
  - inflammation means more cells with lysosomal granules
  - inflammatory cells can flow up and down the ducts, activating things
  - this is worse in cats due to the combined bile duct and pancreatic duct



https://upload.wikimedia.org/wikipedia/commons/6/68/Figure_37_05_05.jpg

- pancreatic duct blockage
  - parasites, inflammatory cells, tumors can block the duct
  - this prevents the natural sodium bicarbonate flush mechanisms
  - activated trypsin stays in the pancreas vs being flushed out
- medications
  - several drugs can activate trypsin
  - particularly a concern in animals being treated with cancer chemotherapy
- ischemia- reperfusion injury
  - ischemia – loss of blood flow; reperfusion – return of oxygen
  - I-R leads to free radical formation

    - free radicals can activate lysosomal enzymes
    - this likely occurs with gastric dilatation volvulus in dogs
- blunt force trauma
    - trauma can cause release of lysosomal enzymes
    - hit by car
    - surgery – it is easy to damage the pancreas
- genetic disorders
    - CFTR gene
    - SPINK gene
    - we know some breeds (terriers, Siamese) are more prone to pancreatitis
    - research area
- hypercalcemia
    - high serum levels of calcium will lead to higher levels of calcium in the pancreas
    - hypercalcemia is seen in older cats with hyperthyroidism and in animals with paraneoplastic syndromes

Pathophysiology

When enzymes are activated in the pancreas, they do start digesting the pancreas itself and the intrinsic vessels. The melting pancreas can cause severe peritonitis and/or become infected. Enzymes eventually leak into the bloodstream and get disseminated to the body. While the gut is designed to "control" the enzyme action, this isn't true in the kidney and other areas of the body. Digestion in other areas leads to inflammation and hemorrhage.

Abscesses, cysts and shock develop. Organ failure can follow (particularly renal failure). Many animals go into disseminated intravascular coagulation (DIC = death is coming). As multiple clots form throughout the body, all the clotting factors are used up and the animals start spontaneously bleeding.

Acute pancreatitis

Clinical signs in dogs

- unnoticed
- vomiting
- diarrhea
- abdominal pain
- shock
- hemorrhage/ ecchymoses, petechiation
- death

Vomit and die syndrome – due to vagal nerve stimulation by the pancreatic inflammation, vomiting can lead to cardiac asystole (heart stops).

Many causes of vomiting and diarrhea are not directly related to the GI tract. Pancreatitis is one of the important ones to consider as it can be life threatening and often needs aggressive treatment. Dogs typically exhibit abdominal pain, vomiting, dehydration and death.

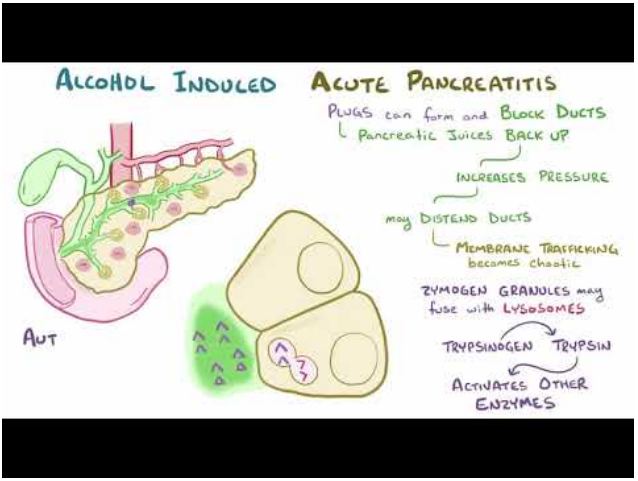
Clinical signs in cats

- unnoticed
- lethargy
- dehydration
- polyuria/polydipsia (drink more, pee more)
- poor appetite
- vomiting
- weight loss

Cats tend to get triaditis – inflammation of the duodenum, biliary system and pancreas. Cats show very vague signs and don't usually die. Cats show mostly lethargy and anorexia, followed by dehydration and jaundice. Vomiting and diarrhea are rare in cats. Many will develop chronic pancreatitis.

Treatment

Both dogs and cats are treated for pain, gastric ulcers, infections, and for dietary deficiencies (vitamin B12 is often required). While going NPO (nothing per os) rests the pancreas, it can have severe consequences of its own, particularly in cats. Dogs are fed a bland diet and cats fed a bland, high calorie diet. As cats are often anorexic, a feeding tube is often required. Fluids and colloids are given as needed.



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Chronic pancreatitis

Chronic pancreatitis is probably an under-diagnosed condition. Animals may be subclinical (not showing signs) but still develop fibrosis (scarring) and atrophy of the pancreas. This means the pancreas cannot return to normal status. However, the pancreas can function normally with only 10% normal tissue.

Affected animals will show similar signs to acute pancreatitis but are not usually at risk of dying unless they get a form of acute pancreatitis on top of their chronic pancreatitis. Abdominal pain can be helpful in differentiating vomiting or diarrhea related to pancreatitis from other causes.

Animals with chronic pancreatitis should be managed to minimize risk factors for pancreatitis. Maintaining a healthy weight is important. Most dogs need to be put on a low fat diet, eating mostly lean proteins and carbohydrates. Cats should stay on food, even if it means using a feeding tube.

Chronic pancreatitis can lead to diabetes mellitus and exocrine pancreatic insufficiency.

Resources

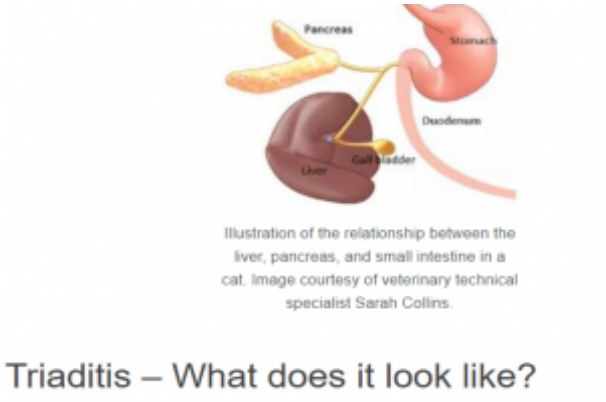
Pancreatitis in dogs – client focused

SPINK gene and pancreatitis – research article
Feline triaditis – vet article

16. Feline triaditis -Inflammatory bowel disease, pancreatitis and cholangitis

Pathophysiology

Anatomy : The **common bile + pancreatic duct** makes cats more likely to share inflammation between the biliary system, the pancreas and the duodenum. This means problems in one tends to lead to problems in the others. Cats with acute necrotizing pancreatitis often have associated suppurative cholangitis and inflammatory bowel disease (triaditis).



https://criticalcaredvm.com/triaditis-in-cats/

Obstruction of the common bile duct allows bile to enter the pancreas and change the duct permeability and pressures. This can lead to pancreatic necrosis.

The association with inflammatory bowel disease may be related to:

- IBD being common in cats
- Vomiting with IBD may raise intraduodenal pressure and push duodenal contents up into the duct
- Cats have a wide variety of intestinal flora that can proliferate and infect things

Diagnosis

The individual diseases can be diagnosed separately, but triaditis should be considered when any combination is seen together:

- Vomiting
    - Vomiting is a sign noted in both intestinal disease and hepatic disease. Remember, vomiting is not a hallmark sign of feline pancreatitis.
- Icterus
    - Icterus can be from hepatic disease. Or it can be from cholangitis secondary to pancreatitis. Bacterial infection in the gut may cause a mild jaundice in the cat.
- Lab values elevated
    - ↑ WBC, ↑ ALT , ↑ ALP, ↑ glucose, ↑ cholesterol, ↑ bilirubin

Therapy

Cholangiohepatitis is divided into two categories: acute and chronic.

Acute or purulent cholangiohepatitis with bacterial infections

Treat with

- long term antibiotics
- maybe steroids
- ursodeoxycholic acid
- SAM-E

SAMe is involved in many liver functions including minimizing oxidative stress and as a methylating agent. Low levels may increase liver damage. SAMe seems to be innocuous (it won't hurt anything to give it)

Chronic cholangiohepatitis

Treat with

- long term corticosteroid and antibacterial therapy
- ursodeoxycholic acid is also helpful.

Ursodeoxycholic acid is a normal component of bile (and black bear bile contains lots) that has 3 main functions

- protection of cholangiocytes against cytotoxicity of hydrophobic bile acids
- stimulation of hepatobiliary secretion
- protection of hepatocytes against bile acid–induced apoptosis

Feline pancreatitis

Feline pancreatitis is generally chronic. Treatment includes fluids, anti-emetics, and steroids.

Acute pancreatitis treatment includes fluids, plasma and anti-emetics. Calcium, antihistamines, and low dose dopamine may be considered.

Cats should be fed. Antibiotics may be given to keep bacteria from colonizing the necrotic tissue.

Hence, common triaditis treatments include:

- Antibiotics (if a bacterial infection is detected)
- Anti-nausea meds
- Appetite stimulants
- Gastrointestinal protectants
- Painkillers
- Steroids (to reduce IBD impact)

Resources

Feline Triaditis: Inflammatory Diseases of the Liver, Pancreas and Small Intestine,World Vet Proceedings, 2011

How I treat feline triaditis, NAVC, 2014

Understanding triaditis in cats, ASPCA

17. Exocrine pancreatic insufficiency

The pancreas can totally fade out in some species. This can be due to a genetic disorder leading to acinar atropy, immune causes or due to chronic pancreatitis. The result is called exocrine pancreatic insufficiency (EPI) and primarily affects dogs.

German shepherds, rough coated collies and beagles are prone to acinar atrophy. EPI develops at about 18 mo of age.

Chronic pancreatitis most affects King Charles spaniels and cats.

As a reminder, the pancreas serves 4 main functions:

1. Enzymatic digestion of food
2. Production of antibacterial proteins
3. Neutralization of gastric acid
4. Production of intrinsic factor

The pancreas has impressive reserve capacity but when **90%** is destroyed, these functions are all lost. This is not reversible.

1. Enzymatic digestion of food

- without these enzymes, food cannot be digested and absorbed. Animals quickly lose weight but maintain a strong appetite. They may even eat inappropriate objects (pica). The unabsorbed food leads to osmotic diarrhea. Fatty stool is seen due to limited digestion. Hair coats are often dull due to lack of absorption of vitamins.

2. Production of antibacterial proteins

- without this secretion, animals can develop bacterial overgrowth or dysbiosis. This can also add to the maldigestion and flatulence issues.

3. Neutralization of gastric acid

- without bicarbonate the acid from the stomach can irritate the duodenum and change the bacterial flora

4. Production of intrinsic factor

- without intrinsic factor, cobalamin is released and cannot be absorbed. Cobalamin is required for DNA synthesis and is crucial for red blood cell production (erythropoiesis). Pernicious or macroocytic anemia is the formation of excessively large red blood cells in the face of cobalamin deficiency

Clinical signs

- weight loss (cachexia)
- polyphagia (eating lots), coprophagia and pica (eating weird stuff)
- small bowel diarrhea with pale greasy feces
- flatulence
- anemia
- lethargy
- diabetes mellitus

Diagnosis

Affected animals are most readily diagnosed by measuring TLI – trypsin-like immunoreactivity and/or response to enzyme treatment.

TLI is preferred as the baseline levels of trypsin are moderate. It is possible to see a decline. Baseline levels of lipase are low so it can be difficult to determine if the pancreas is functional but not currently releasing lipase or dysfunctional and not able to release

lipase. Amylase is just not a good enzyme to measure due to other complexity.

Treatment

1. Provide enzymes for digestion
    - the powder form is considered most effective
    - avoid enteric coated versions because they require bicarbonate to be dissolved.
    - it may take awhile to find the right level for the patient
2. Provide acid control
    - minimize duodenitis and digestion of the enzymes by lowering acid levels
3. Provide B vitamins and fat soluble vitamins
    - folate, cobalamin, A, D, E, and K (and most times they don't need folate since the bacteria produce it)
4. Feed an easily digestible diet
    - moderate fat, low fiber
5. Antibiotics
    - some animals may need control of flora (short term treatment)

Prognosis

Prognosis is good but treatment is lifelong

Key Takeaways

Exocrine pancreatic insufficiency means the pancreas can no longer produce the enzymes and products it usually produces for digestion and gut health.

PANCREATIC FUNCTION	DYSFUNCTION	TREATMENT
Enzyme production	Limited digestion-> flatulence, weight loss, ravenous, poor hair coat, greasy stool	Enzyme supplementation Vitamin A,D, E and K supplementation Highly digestible diet
Antibacterial proteins	Dysbiosis (bacterial overgrowth)	Antibiotics
BIcarbonate secretion	Duodenitis (ulcers)	Antiulcer agents
Intrinsic factor secretion	Cobalamin deficiency, macrocytic anemia	Vitamin B12 supplementation

Resources

Pancreatic acinar atrophy in German Shepherds– review article
Chronic pancreatitis in dogs and cats -review article
Exocrine pancreatic insufficiency – client focused article

18. Diagnosing pancreatic disease

The three enzymes we have historically measured are amylase, lipase and trypsin. Amylase isn't helpful due to poor sensitivity and sensitivity.

The key point is differentiating pancreatitis (excessive function) from exocrine pancreatic insufficiency (insufficient function).

Pancreatic lipase levels are helpful for diagnosing pancreatitis. We expect to see elevated levels when the pancreas is overactive. Normal levels are pretty low so can't use it for diagnosing pancreatic insufficiency.

Trypsin-like levels are already relatively high in normal animals and it can be difficult to determine if an animal has pancreatitis due to challenges with renal clearance etc. However, trypsin-like immunoreactivity can be very useful to detect exocrine pancreatic insufficiency!

Enzyme	Normal	Pancreatitis (high function)	Exocrine pancreatic insufficiency
Pancreatic Lipase	low	**elevated**	low
Trypsin (TLIR)	moderate	moderate or high	**low**

We like measuring pancreatic lipase to diagnose pancreatitis.

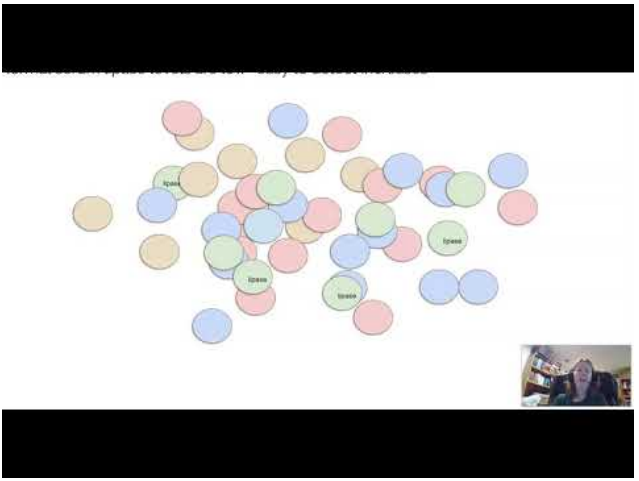
We like measuring trypsin-like immunoreactivity to diagnose pancreatic insufficiency

We can also look at indirect markers of pancreatic function in EPI.

Pancreatic function is required to absorb cobalamin. Low cobalamin levels would be expected with EPI. Folate is produced by bacteria. If we don't have antibacterial protein, the bacteria will expand in numbers and produce more folate.

With EPI, we expect low cobalamin and high folate levels.

Enzyme	Normal	Pancreatitis (high function)	Exocrine pancreatic insufficiency
Pancreatic Lipase	low	**elevated**	low
Trypsin (TLIR)	moderate	moderate or high	**low**
Folate	moderate		**high**
Cobalamin	moderate		**low**



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Resources

Diagnosis of pancreatitis in dogs and cats – review article
Chronic pancreatitis in dogs and cats – article
TLI, folate and B12 – lab test explanation
Trypsin like immunoreactivity – lab test explanation

19. Resources for Pancreatitis and Exocrine pancreatic insufficiency

Useful

Pancreatic function– good video overview

Pancreatitis in dogs, VIN, reviewed 2018

Pancreatitis in dogs and cats, DVM 360, 2011- discusses changes over the past few years and challenges with cats

Exocrine pancreas – Khan academy – basic but good review of earlier material too

Control of the GIT-digestion and motility put together- Khan academy – really nice to come back to

Exocrine pancreatic insufficiency – quick explanation of human version

Advanced Physio

Pancreas, clinical anatomy and physiology– good discussion of CCK and secretin (6 minute mark)

Pancreas, liver and duodenum working together – little longer but well done

Chronic (and acute) pancreatitis -Osmosis

Just for fun

Causes of vomiting – secondary GI – Washington State

Diagnosis of pancreatitis in dogs and cats – JSAP, 2015; latest and greatest

A Multi-Institutional Study Evaluating the Diagnostic Utility of the Spec cPL™ and SNAP® cPL™ in Clinical Acute Pancreatitis in 84 Dogs, JVIM 2012

Fecal markers of inflammation, protein loss, and microbial changes in dogs with the acute hemorrhagic diarrhea syndrome (AHDS), JVECC 2017

Resources for pancreatitis– DVM 360 related articles

Pancreatic insufficiency in people – can you add the physio? How does it compare to animals?

20. Pancreatitis exercises

Challenge Exercises

Dogs and cats

- What hints would lead you to primary vs secondary GI causes of vomiting?
- Investigate the theories related to different causes of pancreatitis
- Use the damnit scheme to create a list of potential causes of pancreatitis
- Why is vitamin B12 so important?
- Why is feeding cats so important?
- How does ultrasound help with pancreatitis diagnosis? radiographs? Can we use CT?
- Is pancreatitis likely to recur?
- What foods are highest risk?
- How is chronic pancreatitis linked to diabetes?
- Why is chronic pancreatitis so hard to diagnose?
-

Do other animals get pancreatitis? What are the causes and signs?

21. Study Questions

Liver

1. What is the first pass effect and why do we care?
2. Explain bioavailability and enterohepatic recirculation.
3. Why is it difficult to extrapolate drug metabolism information across species?
4. How would low albumin levels affect drug activity?
5. What is cytochrome P450 and how is it regulated by drugs?
6. What stimulates gall bladder contraction?
7. Explain the tests used to identify liver damage and assess liver function.
8. How much liver needs to be destroyed before we see changes in liver tests?
9. Why are albumin and clotting factors useful for assessing liver function?
10. What causes a jaundiced appearance?
11. What are causes of elevated bilirubin levels? What types are there?
12. Match the likely elevated liver damage and liver function tests with unconjugated or conjugated bilirubin levels.

Pancreas

1. What enzymes are produced by the pancreas normally?
2. What is autodigestion?
3. What causes autodigestion/ pancreatitis?
4. What are clinical signs associated with acute pancreatitis?
5. How do we diagnose pancreatitis?
6. What clinical signs might you see if the exocrine pancreas is destroyed?
7. What would the poo look like with exocrine pancreatic insufficiency (EPI)?

8. How do we diagnose EPI?
9. What nutrient deficiencies are associated with EPI?
10. How is it treated?
11. How is pernicious anemia associated with exocrine pancreatic insufficiency? What other diseases is it associated with?

Liver shunts, hepatic lipidosis and feline triaditis

1. Explain liver shunts. What liver tests would be abnormal?
2. What is medical treatment of liver shunts designed to accomplish? Surgery?
3. Explain hepatic lipidosis
4. What are the risk factors for hepatic lipidosis? How can it be prevented?
5. What liver tests would be abnormal?
6. What are the goals of treatment?
7. Explain feline triaditis.
8. What is the anatomy that predisposes to triaditis? How does it create issues?
9. What liver tests would be abnormal?
10. What are the goals of treatment?

Practice Quiz

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22. Practice

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PART III
FUNCTION AND MOTILITY

A look at the normal physiology

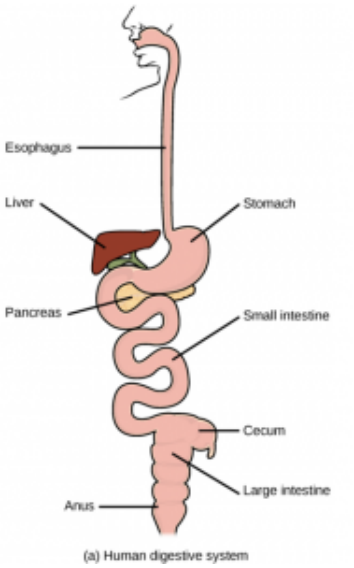
23. Digestion Overview

Monogastrics (carnivores, omnivores)

Anatomy

As described in the chapter on Normal Digestion, in a simple stomached (monogastric) animal, food follows a fairly straightforward and relatively short path

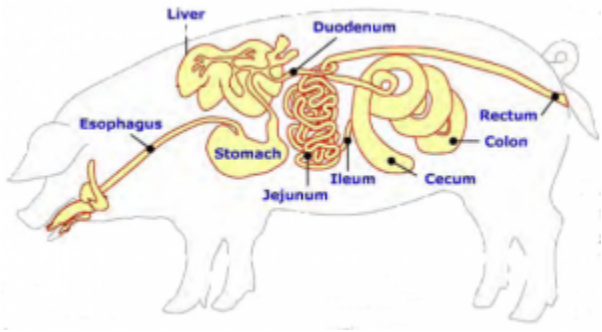
- oral cavity – food is chewed up to increase surface area and may/may not be mixed with salivary enzymes
- esophagus – food moves quickly down the esophagus and through the distal esophageal sphincter
- stomach – holds and grinds food, mixing it with hydrochloric acid and pepsin
- SI – the major area of digestion and absorption as food is mixed with bile and pancreatic enzymes and broken down into component parts
- LI- more water resorption and home of some bacteria that further aid in



Simple stomached animals have GI tracts that closely resemble human ones
https://upload.wikimedia.org/wikipedia/commons/7/7a/Figure_34_01_05ab.jpg

production of essential vitamins
- rectum – holding area until the animal has time to defecate

Pigs do have a spiral colon that is an expanded large intestine (LI) to enable some cellulose digestion.

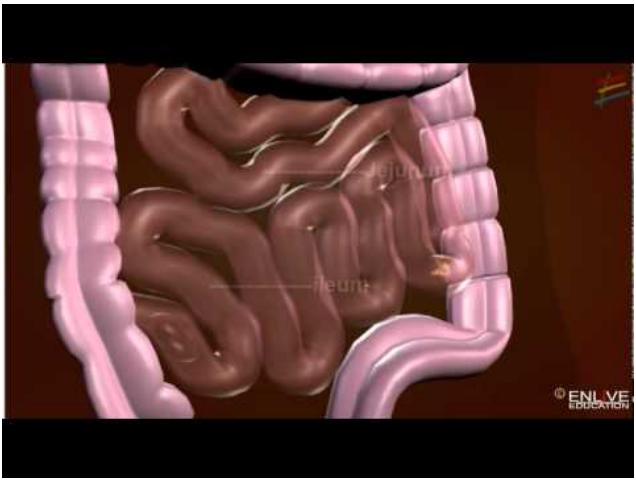


https://extension.oregonstate.edu/sites/default/files/styles/full/public/images/2018-08/pig-fig1.png?itok=E0G84HM7

Digestion overview

In general, carbohydrates (nonstructural) get digested into glucose in the small intestine (SI), proteins into amino acids in the stomach and SI, and fats into lipids in the SI.

The pancreas is essential for digestion and gut health (enzymes, antibacterial protein, intrinsic factor, sodium bicarbonate) as is the liver. Remember the liver gets all of the blood from the GI and further processes the nutrients before sending on a balanced "ration" to the rest of the body.



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Impacts of the kidney and brain

Other organs are also involved less directly in metabolism:

Kidneys can mimic GI disorders. They regulate fluids, electrolytes and **proteins**. With kidney damage, animals can develop proteinuria. This can look like poor digestion or poor absorption of proteins.

The brain can impact GI function in both good and bad ways. The brain responds to the sites and sounds of food, starting and enhancing the process of **digestion**. The brain can moderate **motility** and does this as part of the flight/fight response (aka rest and digest) as well as in periods of stress. The **vomiting** center is in the brain and is involved in all types of nausea and vomiting

including vomiting due to motion sickness, drug responses and intestinal foreign bodies.

Hence, availability and usage of nutrients can be impacted by the GI tract, the liver, the pancreas, the kidney and the brain.

> Note: horses do not vomit (very strong distal esophageal sphincter) and ruminants rarely vomit. For animals with forestomachs, vomiting from the stomach pushes fluid into the forestomach versus out the mouth.

Herbivores

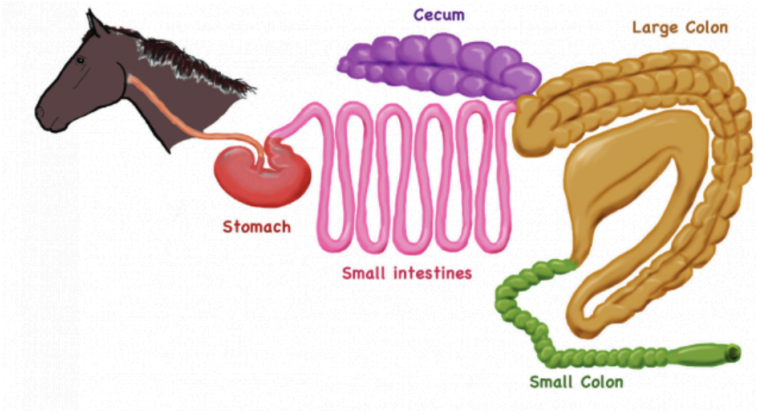
Major variations of the GI tract occur in animals that eat grasses and hays. Very few herbivores have exactly the same set of modifications but all are similar in that they have a fermentation area where bacteria do the work of structural carbohydrate digestion.

Microbes running around video

Horses

Horses are often fed corn, oats, and/or barley as well as sugar beet pulp, fat supplements and proteins. These are basically the same nonstructural carbohydrates, fats and proteins that we feed to monogastrics. Horses also eat grasses and hays. The grasses and hays are known as structural carbohydrates. Cellulose covers the useful carbohydrate in the structural carbohydrates. Animals don't have cellulase so really can't digest cellulose. Bacteria are needed to perform this function; they do have the enzyme cellulase.

The greatly enlarged ascending colon and cecum are fermentation chambers for the digestion of these structural carbohydrates.

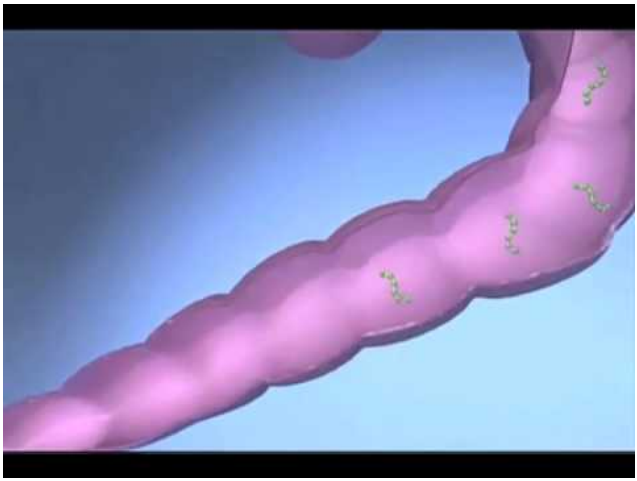


https://nwdistrict.ifas.ufl.edu/phag/2018/09/21/understanding-a-horses-digestive-system/

Horses are grazing animals. They have a relatively small stomach for their size. They eat small amounts almost constantly rather than a large meal once a day or every few days.

Grasses are not easy to digest. Hence the modification of the colon and cecum. Besides expanding the length of the colon, motility is also adjusted to allow backwards and forwards ingesta flow, extending the time the grasses spend in the colon.

Horses do not have a gall bladder. They have a biliary system but, instead of collecting in a gall bladder, bile is just constantly emptying in to the SI.



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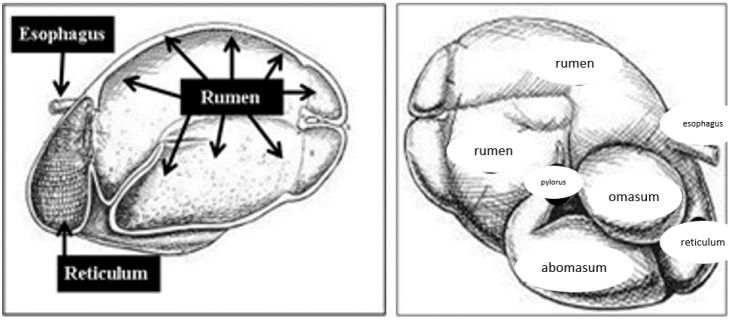
Cattle

Cattle (and sheep and goats) digest grasses in the forestomach or rumen. The forestomach is positioned between the esophagus and the main stomach (abomasum). The forestomach has 3 parts:

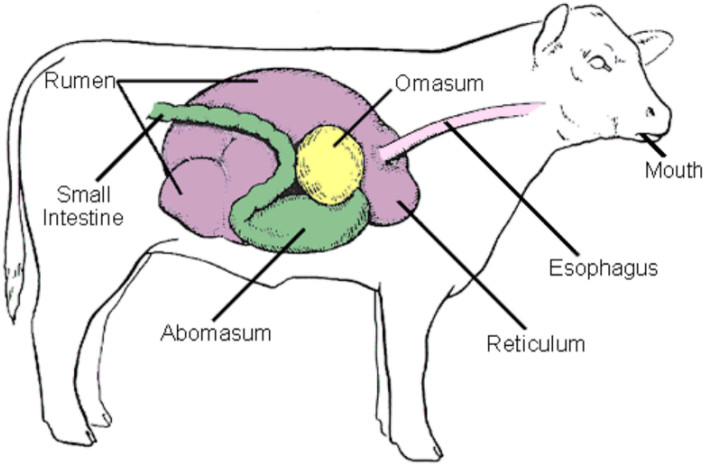
- rumen – large fermentation vat; the inner surface is covered by papilla
- reticulum – sorts particles into those that need more digestion or are ready to be moved down the GIT; honeycomb inner surface
- omasum- in charge of fluid removal; full of "leaves" that enhance water and nutrient absorption



Left side view Right side view

https://www.fda.gov/animal-veterinary/animal-health-literacy/how-cows-eat-grass

The abomasum is a true stomach with gastric acid producing structures, etc.

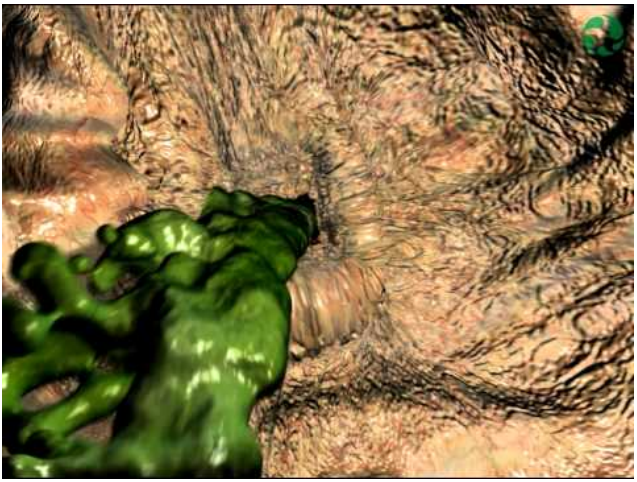


https://www.toppr.com/ask/content/concept/digestion-in-ruminants-200952/

Ruminants can regurgitate (eructate) and rechew the grasses. This

is called chewing their cud. This process allows further digestion of those tough grasses and hays.

Cows do have a spiral colon, similar to that of pigs. They also have an elongated cecum, similar to that of horses. These structures will also help digest cellulose products.



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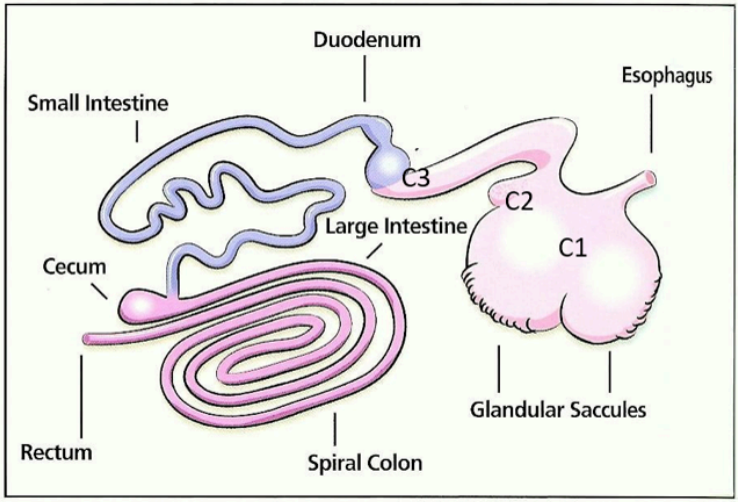
Camelids

Camelids are pretty similar to cattle in that they have a forestomach and chew their cud. They have 3 versus 4 stomach chambers.

- C1 – like a rumen

- C2- like a reticulum
- C3- true stomach; also does water absorption somewhat like an omasum

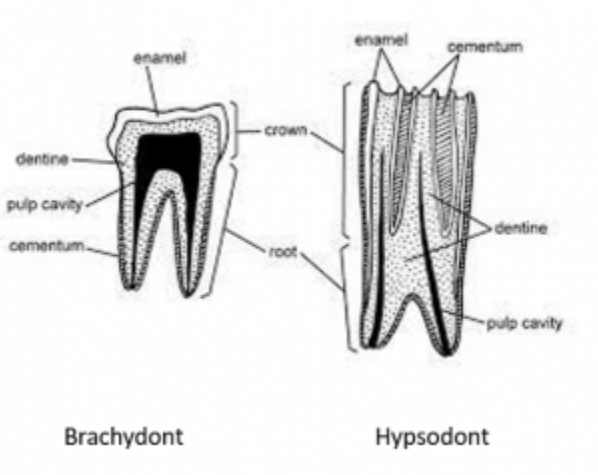
Camelids also have a very tightly coiled spiral colon.



https://www.applemountainalpacas.com/mentoring/alpaca-digestive-system/

Teeth

Teeth match the diet.



https://www.life.illinois.edu/ib/462/Lab%203%20-%20Teeth.pdf

Brachydont species

- dogs and cats
- teeth designed for ripping and tearing
- short roots

Hypsodont species

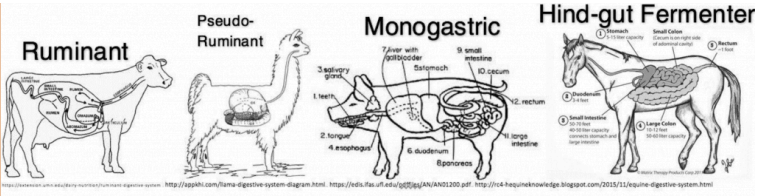
- horses, ruminants
- teeth designed for grinding
- long roots because actually grind teeth down
- cattle and camelids have an upper dental pad rather than upper incisors

Key Takeaways

Key components

- Protein digestion starts in the stomach and continues in the SI while carbohydrates and fats are mostly digested in the SI with pancreatic enzymes
    - Proteins, non-structural carbohydrates and fats are digested in herbivores the same way they are in carnivores and omnivores. Dead bacteria are a protein source in animals with forestomachs.
- The liver is essential for digestion of fats and balances nutrients for the rest of the body; pancreatic enzymes are required for digestion
- Ruminants and camelids regurgitate to further chew food. Herbivores need microbial digestion and more time to further digest structural carbohydrates (fiber, hay)
- Herbivore microbes live in the fermentation section of the GI tract – this can be the forestomach such as cattle and camelids, or the hindgut such as in horses and rabbits.
- Bacteria produce volatile fatty acids (energy source) and B vitamins for the host.
- Dysfunction of the liver, kidney, pancreas, and brain often produce changes in nutrient usage. Microbial health is also necessary for good digestion.

Species comparison



https://www.agupdate.com/tristateneighbor/news/livestock/vet-report-managing-ionophore-toxicity-in-horses/article_cb0d129a-d6cd-11e8-87db-03d11b038b94.html

	Dog	Horse	Cow	Alpaca
Oral cavity	Brachydont Tearing	Hypsodont Grinding	Hypsodont Upper dental pad	Hypsodont Upper dental pad
Esophagus			Regurgitation	Regurgitation
Forestomach			Rumen, reticulum, omasum	C1, C2
Stomach	Simple	Simple	Abomasum	C3
Small intestine				
Cecum	Small	Large	Large	Small
Ascending colon	Small	Large and folded	Spiral	Spiral
Descending colon			Spiral	Spiral
Rectum				

Video Resources

How your digestive system works – confused from the beginning? start here to just get a nice overview

A cow's digestive system – good review and intro to physical exams

Ruminant structure and function – nice overview

More ruminant videos

- Digestion in ruminants
- How does a rumen work

Equine GI tract– review of anatomy

Rabbit coprophagy – excellent video

Digestive system part I crash course – the path of nachos from start to finish; longer but worth watching a few times; it is jam packed with relevant info

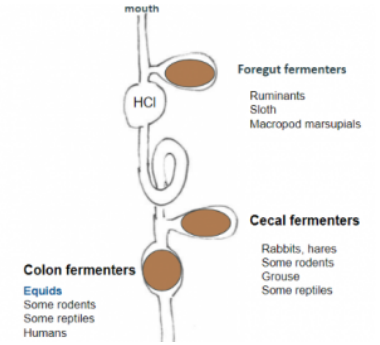
Comparative anatomy slide show

Digestive system overview and comparison – CSU web pages

Malone weight loss lecture

24. Fermentation

Fermentation chambers can occur in several different locations in mammalian gastrointestinal tracts.



Each of us has a unique microbiome that is essential for our good health. The microbes digest any leftover starches and proteins, including those in sloughed intestinal cells. In all species, a healthy microbiome also produces enough water soluble B vitamins and vitamin K to keep us healthy.

Microbes generally have a preferred milieu. If that environment is changed, different microbes can take over and change the level of energy and gas production. For instance, if any of us eat too much, the extra food will reach the microbes. This may also lead to changes in pH and type of microorganisms. At the very least, it will lead to increased intestinal gas.

However, none of us have microbes when we are born. We acquire microbes through a variety of processes. Foal heat diarrhea is believed to be related to foal copraphagy (eating feces); the copraphagy is useful to digest those microbes but leads to the mild diarrhea until the GI tract can adjust.

As a juvenile switches from milk to other diets, the microbial environment becomes more important. Often there is a slight mismatch between the microbiome maturity and the diet. In calves, this mismatch can lead to free gas bloat. This type of "immaturity" bloat can be a recurrent issue, necessitating the placement of a relief valve in the rumen to let the gas off. Ideally, calves with recurrent free gas bloat get a "rumenostomy" (surgical opening into

the rumen) that allows gas to escape when needed, at least until the rumen flora mature.

Foregut fermenters

Ruminants and other foregut fermenters (i.e. camelids) use microbes to produce cellulase. Cellulase breaks down grasses into compounds that microbes can digest. The microbes transform food into essential B vitamins and one of three volatile fatty acids (VFAs)- acetic acid, proprionic acid acid and/or butyric acid.

Ratios of VFAs vary by diet and microbe population. The VFAs are absorbed through the rumen wall and transported to the liver and other parts of the body. The liver can convert propionic acid into glucose; the other acids are used by various other cells of the body (especially by the udder). VFAs provide 60-80% of the cow's energy, meaning a healthy microbial flora is essential.

Microbes also digest protein and are a source of (microbial) protein. Once the microbes are killed in the abomasum, the protein is digested and absorbed by the ruminant.

Hindgut fermenters- cecal and colonic

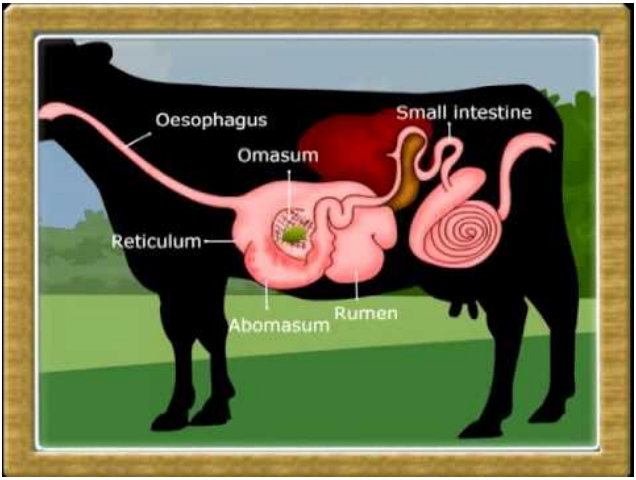
The equine large colon has been highly modified to allow cellulose digestion. It is very elongated to allow maximal exposure of the cellulose products to the microbes. Activity is largely mixing with propulsion slowed to keep the ingesta in the colon for long periods of time (transit time is 30-50h). This maximizes the nutrition that the horse can obtain from the hay diet. The microbes use the hay (and any leftover carbohydrates) for energy and produce volatile fatty acids that are absorbed by the colon wall and used by the horse. These fatty acids supply the major portion of the horse's daily energy requirements and is why they do well on a strictly hay diet. The microbes also produce some essential B vitamins. Unlike in foregut fermenters, the hindgut fermenters can't use the microbial protein unless they eat feces. Horses do not routinely eat their own feces, so their protein needs are met by the food they ingest.

The colon also buffers ingesta coming from cecum and absorbs fluid from the ingesta. A pony resorbs approximately 30 liters of fluid/day from the colon.

As with the small intestine, the colon (with its microbes) is designed to handle frequent eating and continuous flow of ingesta. If horses are fed only twice daily, they will have much more extensive fluid shifts into the ingesta and out of the colon, particularly if they are fed highly fermentable carbohydrates. Increasing carbohydrates entering the cecum and colon also means happy microbes and more gas production.

Rabbits are a cecal fermenter. The digestion process requires a repeat exposure to the feed for full digestion. Similar to birds with retroperistalsis activity, once through the gut is not enough.

Rabbits eat the “night feces” (caprophagy) which are rich in dietary essentials produced by the microorganisms and partially digested cellulose. This permits those nutrients to be removed by the rabbits GI tract.



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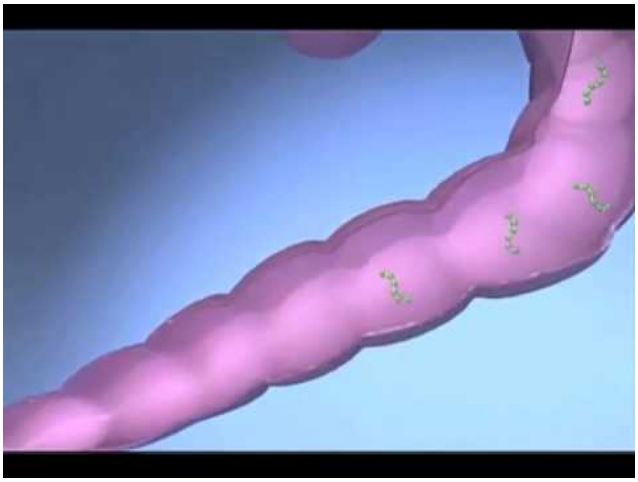

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Resources

Life sciences lecture – UIll
- Rumen physiology and rumination – CSU
- Rumen development in the dairy calf – Dairexnet
- Ruminant structure and function – nice overview
- Rabbit coprophagy explanation

More depth

Feeding the calf – Calf care canada

Calf nutrition and management – free coursera lecture

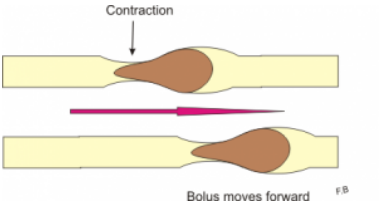
Rumen development and weaning in dairy calves– good images

Rabbit cecal function – not sure about the secretion part but interesting article

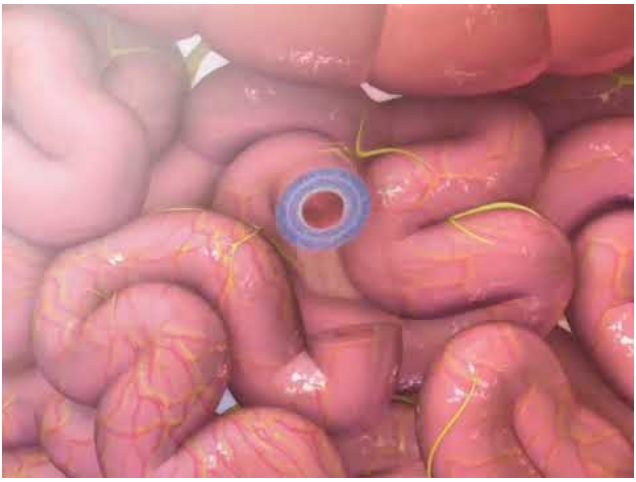
25. Normal GI Activity

Activity types

Peristalsis



Peristalsis is the aborad (away from the mouth) propulsion of ingesta through the parts of the GI tract. To move ingesta, generally the portion of intestine just orad (oral cavity side) to the food squeezes shut through combined circular muscle contraction and longitudinal muscle relaxation. For the bolus to move, the intestine just aborad to the food has to open up (circular muscle relaxation and longitudinal muscle contraction). Due to the connections between gut neurons, the process continues smoothly in most instances.



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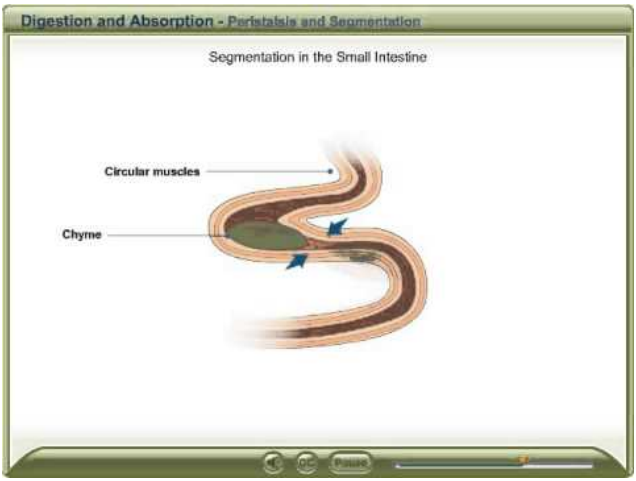
Peristalsis occurs in the esophagus, SI and LI. The stomach contracts and empties but doesn't really have this particular type of peristaltic activity.

Peristalsis leads to intestinal motility.

Segmentation

Segmentation and mixing are also required to ensure the ingesta is broken down into small enough particles and gets exposed to the mucosa surface for absorption. In this activity pattern, the area with the ingesta alternately contracts and relaxes.



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Segmentation occurs in the SI and LI. It should not occur in the esophagus. Mixing does occur in the stomach but we don't call it segmentation.

Segmentation is intestinal activity but not motility.

Mass movement

Mass movements are large waves of motility.

There are species differences in the type of motility patterns. Dogs eat relatively large meals at set times of the day while horses graze continuously. There are major waves of peristalsis that "clean house" in the dog. These mass movements move everything out of the small intestine. In the horse, there are different electrical

and contractile patterns that result in continual activity with fewer periods of mass movement.

Retrograde peristalsis

This is backwards motility

Retrograde peristalsis occurs in the large intestine, particularly in herbivores, to allow full digestion of cellulose. After a time, mass movements shift the colonic contents aborad toward the rectum. Eructation/regurgitation is considered normal in ruminants (cattle, sheep, goats, deer) and camelids. Sometimes retrograde peristalsis occurs in the esophagus and SI but is considered abnormal in most species (aka regurgitation and vomiting).

Normal patterns by site

	Esophagus	Stomach	SI	LI
Dog	Peristalsis	Segmentation "Peristalsis"- gastric emptying	Segmentation Peristalsis	Segmentation Peristalsis Mass movements
Horse	Peristalsis	Segmentation "Peristalsis"- gastric emptying	Segmentation Peristalsis	Segmentation Peristalsis Retrograde peristalsis
Cow	Peristalsis Retrograde peristalsis	Segmentation "Peristalsis"- gastric emptying	Segmentation Peristalsis	Segmentation Peristalsis Retrograde peristalsis
Camelid	Peristalsis Retrograde peristalsis	Segmentation "Peristalsis"- gastric emptying	Segmentation Peristalsis	Segmentation Peristalsis Retrograde peristalsis

Retrograde peristalsis in the stomach/SI of monogastrics is observed as vomiting.

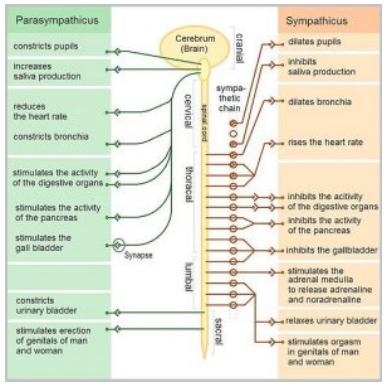
Retrograde peristalsis in the esophagus of monogastrics is observed as regurgitation.

Activity control

The GI tract is under complex control. Typically intestinal activity is stimulated by the sight, smell and ingestion of food. The main activity patterns come from the enteric nervous system and its multitude of neurotransmitters. The main types are adrenergic (norepinephrine), cholinergic (acetylcholine) and non-adrenergic/non-cholinergic ones (NANC or everything else).

Autonomic nervous system (ANS)

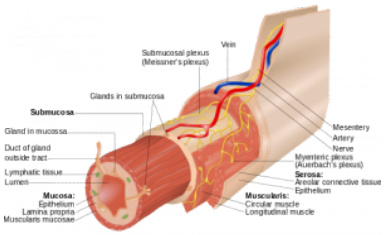
The gut is affected by the parasympathetic nervous system, particularly through the vagus nerve. Stimulation by the parasympathetic system tends to encourage **"rest and digest"** – stimulating gut activity through more acetylcholine release. Blood is directed toward digestive processes.



The sympathetic nervous system also plays a role, tending to inhibit GI activity and blood flow for the **"flight or fight"** response. Blood is shunted away from the guts to more vital organs and leg muscles. A similar shut down occurs with pain, even if flight isn't necessary.

This is a balance – there is always some acetylcholine release and some norepinephrine response. The effect depends on which is in greater amounts. It is possible to block either response using drugs, as well.

Enteric Nervous System (ENS)



Enteric nervous system & intestinal anatomy

The intestine is naturally active, stimulated locally through the **enteric nervous system**. The enteric nervous system is primarily located in the myenteric plexus with extensions to the Meissner's plexus. When this activity becomes coordinated, it can move or mix ingesta.

This nervous system uses acetylcholine (cholinergic), norepinephrine (adrenergic), and non-adrenergic/non-cholinergic neurotransmitters. The ENS can function in the absence of the autonomic nervous system.

Part of the GIT do "talk" to each other to coordinate activity.

For example, the stomach holds ingesta until the food particles are both big and the duodenum is empty. The ileum holds ingesta until lipid levels are low (see ileal brake below). The colon holds ingesta until water and fiber levels are lowered. The rectum holds the feces until the animal is able to stop and defecate.

Enteroendocrine effects

Circulating hormones also affect the gut- there are receptors for

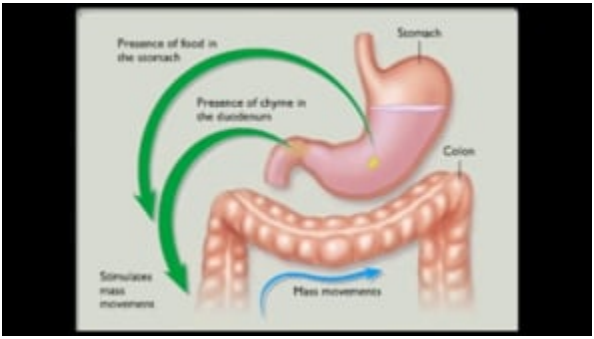
most hormones in the GI tract, including adrenal and thyroid hormones, estrogen and oxytocin. The brain-gut connection is very strong as most all of us have experienced in stressful situations. With dysfunction, the gut can also affect the brain. When the liver isn't functioning well, the amino acids that pass through the liver can become neurotransmitters with direct neurological effects.

Reflexes

The enteric nervous system is connected throughout its length, providing feedback to other sections depending upon fill. Four of the most crucial communication reflexes are:

Gastrocolic reflex

Stomach distension leads to colonic propulsion and defecation. The goal is empty out the old stuff so there is room for the incoming.



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You can view it online here: https://open.lib.umn.edu/vetphysioapplied/?p=5

Intestino-intestinal reflex

Distension in one area of the intestines slows motility of segments proximal. The goal is to avoid adding more ingesta to the already distended section.

Ileal brake (animation)

Higher than usual nutrient levels (especially **fats**) entering the ileum slow motility in the proximal small intestine. The goal is to allow more time for digestion.

Defecation reflex

The defecation reflex is stimulated by movement of food from the colon and distension of the rectum. The internal anal sphincter relaxes and external sphincter contracts. This is a combination of parasympathetic and enteric nervous system effects.

Key Takeaways

- The primary intestinal activity types are peristalsis and segmentation. Mass movements also occur in the colon and retroperistalsis in the colon and esophagus of ruminants and camelids. The normal types of activity vary by section of the GIT.
- Higher sympathetic tone decreases gut activity while higher parasympathetic tone increases it.
- The enteric nervous system is able to control gut activity independently of the ANS/brain; it helps coordinate activity along the GIT
- Ingesta movement is slowed at the pylorus, the ileocecal valve, the colon and the rectum. Further movement depends on the local environment but can be altered by more distant forces.

Resources

The enteric nervous system – video; more than you need to know but good explanation of basics too

Enteric nervous system -CSU blog

Gastric motility – CSU blog

Malone's thesis – good for insomnia

For fun

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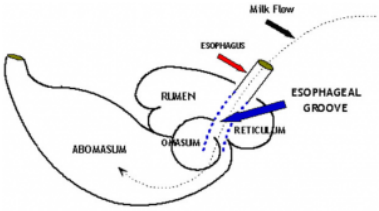
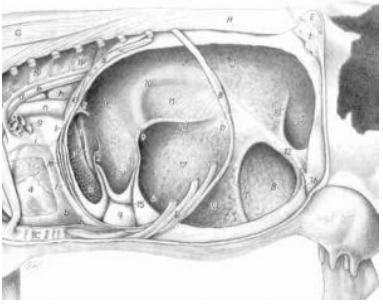
26. Forestomach activity and function

The rumen is nonfunctional at birth and has to develop over time. In the neonate, milk bypasses the rumen by going through the esophageal groove.



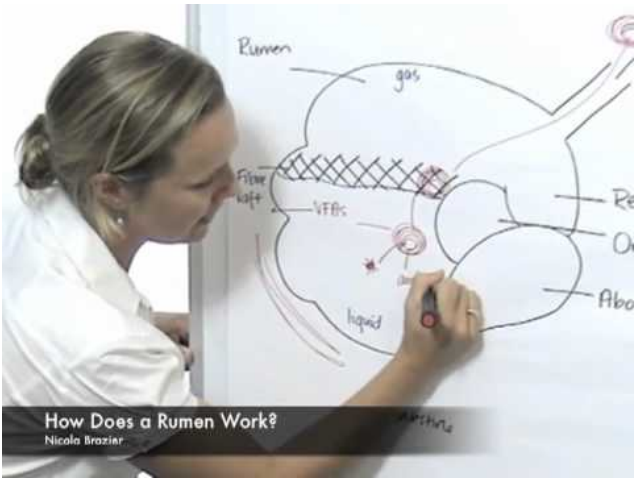
This is a functional change in the esophagus created by high levels of vasopressin. When the calf suckles, the suckling action and milk proteins stimulates formation of the groove, shifting milk past the rumen and reticulum. This avoids any accidental fermentation of milk. If a calf drinks from a bucket or if the groove doesn't form, occasionally some milk will end up in the rumen and cause bloating. These calves are known as "rumen drinkers".

Over time, the rumen acquires microbes (from the diet and the environment), begins to expand and acquires the papilla. The papilla development occurs in direct relation to the intake of grains rather than hays. Calves eating too much milk or poor quality grain do not develop as healthy a rumen as those on good quality and adequate levels of grain.



The rumen is also called a fermentation vat. Once the calf is weaned and the rumen has begun developing, food enters via the esophagus and directed into the rumen. The well functioning rumen has distinct layers. Yesterday's hay and

grains are already somewhat liquefied and sink to the bottom. Gases float to the top and today's hay floats in the middle. These layers can be distinguished on firm palpation through the body wall. Today's hay will be regurgitated and chewed more before it starts to sink. Once in the fluid layer, the older feeds become microbial food and will be digested by the bugs. Larger particles will be recirculated in the rumen until further digested. When the particles are small enough, they will move through the reticulum and into the reticulo-omasal orifice. The omasal leaves will absorb as much water as possible, sending the thickened chyme into the abomasum.



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A similar process occurs in small ruminants and in camelids. Camelids have 3 chambers, named C1, C2 and C3. C3 is the true stomach while C1 acts like the rumen and C2 like the reticulum.

Rumen rolls or rumen contractions occur every 1-3 minutes in

a healthy animal. These can be felt through palpation of the paralumbar fossa and heard with a stethoscope. The brainstem and vagus nerve control rumen activity. It can be altered by changes in content (eating river rocks is bad for rumen activity), pain, pH changes and vagal nerve dysfunction. Hormones also appear to play a role, as seen with pregnancy indigestion.

The rumen should contain large and small motile protozoa as well as a multitude of mostly gram positive bacteria. pH levels are very important to keep the appropriate flora balance and for normal rumen activity. Rumen pH needs to stay between pH of 6 and 7 for best function. Rumen pH <5.5 causes significant disruption on rumen flora. Extra chewing (enough fiber) helps by producing buffering saliva. Dietary rumen buffers can help as can certain additives.

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Resources

Development of the rumen – how feedstuffs affect rumen papilla development

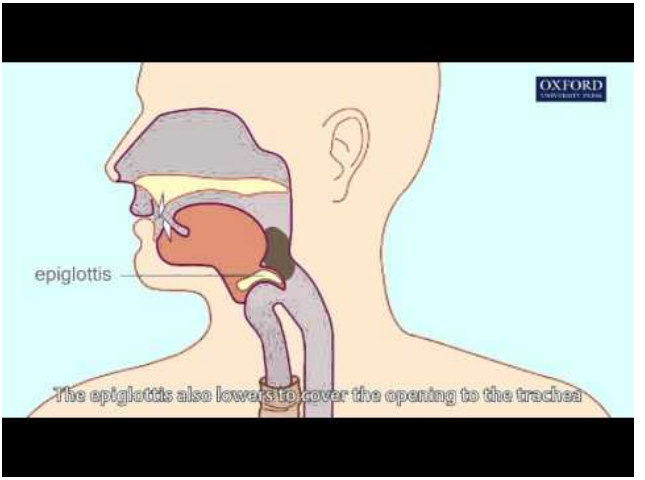
Rumen microbial fermentation – nice overview; you can skip the rainforest bit

How the rumen works ebook – well done and more than you need right now

Rumen physiology and rumination – CSU- always has good stuff

27. Oral cavity and esophageal function

Chewing and swallowing



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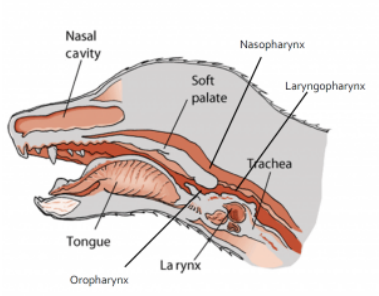
Normal food movement into the esophagus involves **prehension**, **mastication** and **swallowing**.



Prehension

Different species prehend (grab) their food in different ways, with some using lips more than tongue and vice versa. Usually prehension abnormalities are easy to identify as animals visibly struggle to pick up or grab food. Oral exams and cranial nerve evaluations are crucial to localize lesions.

Chewing

Chewing motions vary by species; learning normals is useful. Asymmetrical chewing or dropping of unchewed food particles (quidding) are commonly seen when chewing is painful.

Chewing requires a functional cranial nerve V (trigeminal), normal muscle function and healthy teeth. Issues can be caused by problems with neuromuscular function, dental issues, pain on chewing or foodstuffs that are tough to chew.

Swallowing

Swallowing requires central stimulation and involves opening of the upper esophageal sphincter, movement of the soft palate upwards to close off the nostrils and retroflexion of the epiglottis to cover the airway. Swallowing is controlled by the brainstem and happens reflexively – no effort required in most situations. Cranial nerves IX,

X and XII are all involved. Problems with any of those steps can lead to dysphagia and dropped food.

Esophageal transit

Esophageal motility is controlled by the autonomic nervous system and is involuntary. Central stimulation opens the upper esophageal sphincter and starts a peristaltic wave that generally carries the bolus to the stomach, coordinating the progression of the bolus with opening of the lower esophageal sphincter. If the bolus doesn't get all the way down, local reflexes create a secondary peristaltic wave. Most disorders are related to poor muscle tone or neurogenic disorders of the swallowing component or esophagus.

Key Takeaways

After teeth grind up the food and the tongue shapes it into a bolus, the brainstem kicks in to swallow it. The upper esophageal sphincter opens while the palate protects the nasal passageway and the epiglottis protects the trachea. The bolus moves down the esophagus in a quick peristaltic burst. The lower esophageal sphincter opens automatically to let the bolus move into the stomach.

Resources

Anatomy of a swallow-humans

Normal cat swallow

A most multitalented nerve- cranial nerve X – a good nerve to understand well

Is your pet vomiting or regurgitating? – blog post

Megaesophagus in dogs – proceedings

Esophagus endoscopy in the dog– video

Just for fun

I am your esophagus

Sword swallowing – think about what has to change for this to happen

Giraffe prehension and tongues

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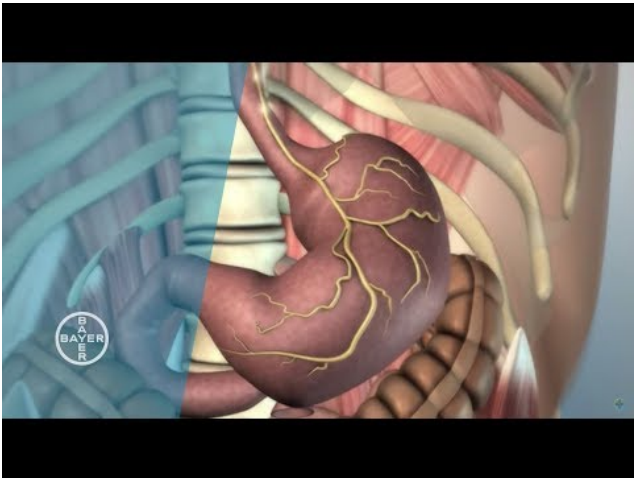
28. Gastric activity and function



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The stomach is designed to hold food, destroy microbes and start the process of digestion.

Food storage

As food enters the stomach, the stomach relaxes in a process called **receptive relaxation** or **accommodation**. The process is mediated

by the vagal nerve and allows for the storage of more food. Predators tend to have larger stomachs with more relaxation capability as they may not eat frequently. Grazers such as horses have smaller stomachs relative to body size.

Acid production

Stimulated parietal cells produce hydrochloric acid (HCl) via a proton pump, creating a very acidic environment. This helps to kill any microbes that entered on or with the food.

The acid proton pump is stimulated by a combination of acetylcholine, histamine and gastrin. Vagal stimulation and release of acetylcholine occurs with swallowing and gastric distension. G cells produce gastrin in response to stretching of the stomach wall, protein in the chyme, and higher gastric pH. Enterochromaffin cells produce histamine in response to gastrin and pituitary peptides.

Activation of the pump is summative – the activation is strongest when all three compounds (acetylcholine, histamine and gastrin) are binding compared to when only one or two compounds are binding the pump.

The proton pump is inhibited by prostaglandins, antihistamines, and somatostatin.

The HCl also activates pepsinogen produced by the chief cells. Pepsinogen is turned into pepsin and can begin protein digestion.

The stomach also contains mucin cells which produce mucous to protect the stomach wall from the acid production.

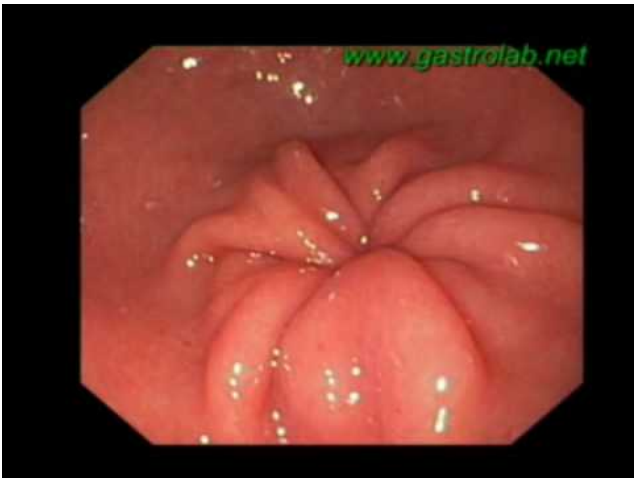
Pepsin production

Once the environment is acidic, pepsinogen is converted to pepsin. Pepsin starts breaking down proteins into peptides. Once proteins

are small enough, the stomach will allow them to move into the duodenum for further digestion.

Stomach motility and emptying

The stomach does contract to grind and mix the food.



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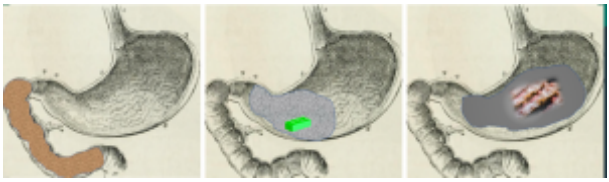
As food is broken into smaller particles, the pylorus opens to allow the "chyme" to move into the duodenum.

The stomach empties faster with liquid contents (especially isotonic liquids) and with large meals (gastric distension).

Mostly things delay stomach emptying. The stomach empties only when the duodenal environment suggests that the prior meal

has moved down the GI tract. The presence of acid, protein byproducts and fats in the duodenum mean the last "meal" is still in the duodenum and there is no room for more. This information is sent back to the pylorus, preventing emptying of more food.



Stomach emptying is delayed when the duodenum is full, the duodenal content has a low pH, high osmolarity, fat and protein byproducts; or when particle size in the stomach is > 1mm; or when the stomach contains high fat foods

When the pH is elevated (from the pancreatic secretions) and minimal food byproducts are present in the duodenum *and* if the stomach contents are small enough, the pylorus will push more gastric content into the duodenum and the cycle starts again.

Cholecystokinin and secretin delay gastric emptying as their release from the pancreas and SI are due to the presence of feed stuffs (CCK released in the presence of lipids and tryptophan) and excessive stomach acid (secretin).

Other factors that can delay stomach emptying include pregnancy, stress, pain and drugs.

Key Takeaways

The gastric fundus relaxes when a meal is anticipated. This allows it to fill without increasing tone. The stomach empties in response to changes in gastric and duodenal contents. Large meals and fluids stimulate emptying. Everything else delays gastric emptying.

Resources

The parietal cell acid secretion – Colostate vivo
How HCl is formed and secreted – animation
Gastric acid physiology – watch 2:30-6:30 in particular; rest is good too

Just for fun

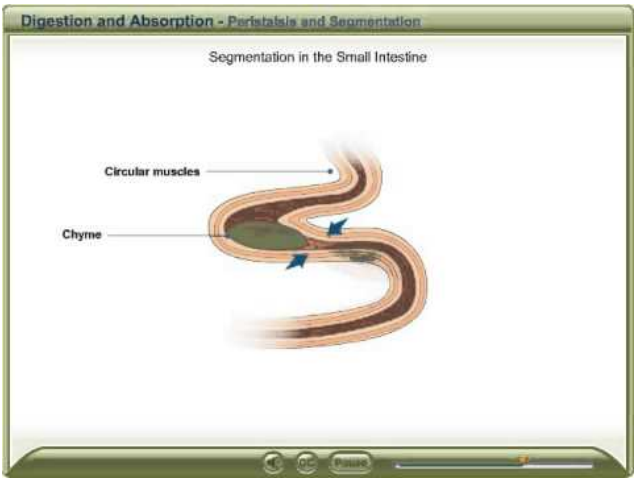
Upper GI movies – stomach peristalsis
I am your stomach

29. SI activity and function

SI Activity

The activity of the SI is highly controlled by the enteric nervous system. The interneurons in the myenteric nervous plexus carefully coordinate a timed ans sequential relaxation and contraction that pushes the bolus of food distally. Food should only move in one direction – retrograde flow is not normal. Mixing in place does occur.

Patterns of small intestinal motility are regular – moving from periods of no spiking activity of the neurons (no contraction), through intermittent spiking/ contraction and through to regular spiking and contraction. The regular spiking phase helps to clear the contents by a more forceful peristaltic wave.



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SI function

The small intestine does most of the work. It is responsible for the majority of fluid absorption as well as for nutrient digestion and absorption. The bile and pancreatic ducts empty into the duodenum, providing the needed factors for fat, carbohydrate and protein digestion. More protein and disaccharide digestion occurs by enzymes released from the enterocytes. The intestinal wall also maintains a firm border to keep out the bacteria and larger components found in the ingesta. Maintaining this tight wall also permits active transport mechanisms to exist by allowing electrolytes to stay in unequal concentrations (not go down the concentration gradient).

The duodenum also helps regulate gastric, liver and pancreatic functions. .

Secretin is released from duodenal S cells when acid levels are high in the duodenum. Secretin stimulates bicarbonate production and release from the pancreas. Secretin also decreases gastrin production, slows gastric emptying and inhibits bile release.

CCK (cholecystokinin) is released from enteroendocrine cells when protein and fat byproducts are identified in the duodenum. CCK stimulates bile release, pancreatic enzyme release and more bicarbonate secretion.

Hormone	Primary Action	Stimulus	Source
Gastrin	Releases HCl and stimulates gastric mucosal growth	Calcium and peptides in the stomach	G cells in gastric pits
Cholecystokinin ("to move the gall bladder"	Stimulates release of pancreatic enzymes and bile	Partially digested fats and proteins in duodenum	Duodenal enteroendocrine cells
Secretin	Releases bicarbonate rich fluid from pancreas. Inhibits gastrin production	Acidification of the duodenum	Duodenal S cells

Resources

Vivo CSU site– more enzymes if you like this stuff (or want to review these)

Just for fun

Intestines peristalting (not a real word) video
I am your small intestine

30. Fluid secretions and water absorption

Water absorption

Why do wombats have square poo?

Animal poo/manure/feces/stools vary drastically. Animal stool varies by species and individual. In general, species that live in arid environments with minimal access to fresh water tend to have drier stool. Wombats do have squarish shaped poo (story here). In general variations occur between species due to differences in GI tract differences:

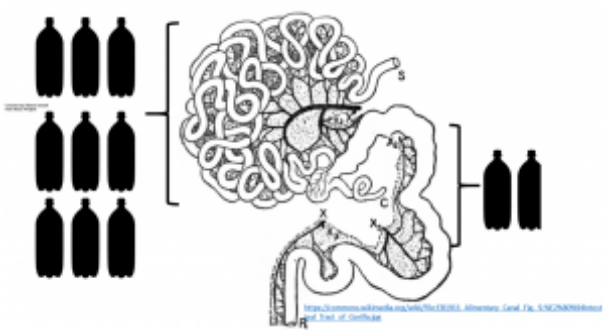
- length
- fore/hindgut fermentation
- activity differences
- microbial populations
- absorptive capability

Variations between individuals occur due to differences in:

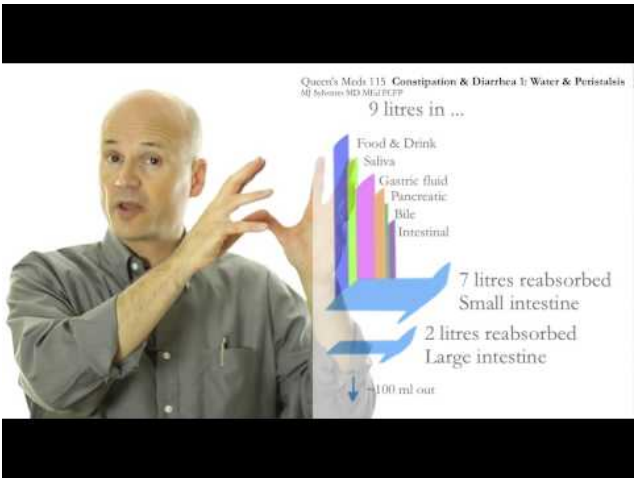
- Diet – affects transit time and secretions
- Stress levels
- Hydration
- Drugs or other disorders

Different parts of the gut have different functions, including different amounts of water absorption. All species primarily absorb

water from the small intestine. Hindgut fermenters (horses) can also absorb quite a bit from the LI. If the total body water content becomes too low, more water is absorbed from the colon, making the stool even drier. Stool that is retained in the colon will also become dry. Astute animal owners can detect changes in their pet through changes in the stool size and consistency.



In humans (and likely gorillas), the small intestine absorbs about 9 liters of fluid daily while the large intestine absorbs just under 2 liters.



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Osmosis is the main force behind water movement. By using active transport pumps, the levels of sodium and glucose are higher in the blood than in the gut lumen. This creates a concentration gradient that permits osmosis (movement of water).

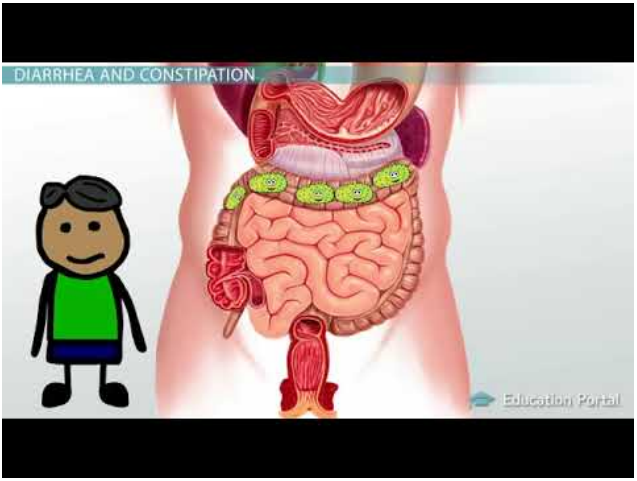
31. LI activity and function

LI Activity and Function

The large intestine is responsible for water removal and microbial nutrient production.

Activity includes segmentation, some reverse peristalsis and mass movements – coordinated long contractions that move the ingeta toward the anus.

The large colon is often modified across species. Carnivores have a relatively short colon.



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LI Function

The main roles of the large intestine are to remove water, move ingesta out of the GI tract and maintain a healthy microbiome. The microbiome helps with immune function, keeps out other bad bacteria and produce essential B vitamins, volatile fatty acids and nitrogen (through self-sacrifice).

The microbes do ferment feedstuffs in all of us – not just ruminants. Particularly when we eat nonsoluble fiber and difficult to digest foods.

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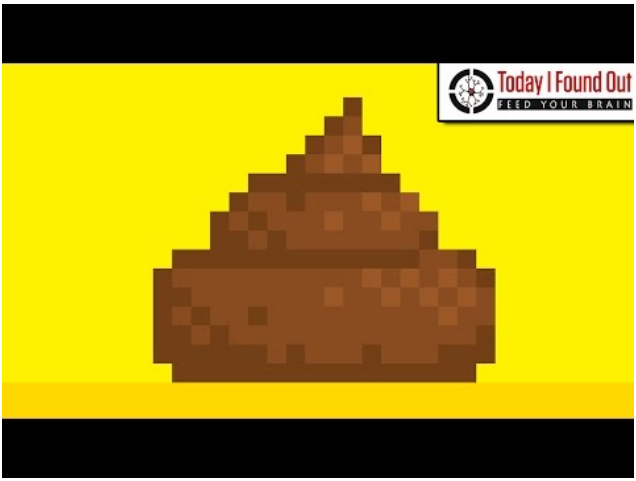

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Just for fun

I am called your large intestine

32. General motility diagnostics

Diagnosis of motility disorders

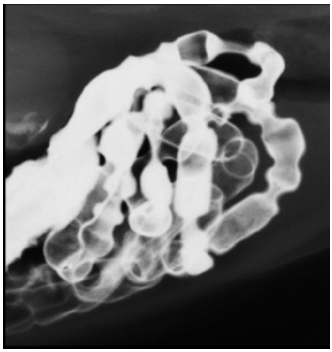
Transit times vary by species and by area of the gut

INTESTINE LENGTHS AND AVERAGE DIGESTA TRANSIT TIMES				
	Dog	Cat	Man	Horse
Small intestine (m)	3.9	1.7	7.0	20
Large intestine (m)	0.6	0.4	1.8	7
Total length (m)	4.5	2.1	8.8	27
Body length (m)	0.75	0.5	1.75	3
Total length: body length ratio	4–5	3	5	9
Mean retention time (hr)	22.6±2.2	13	45.6±11.1	37.9±5.3

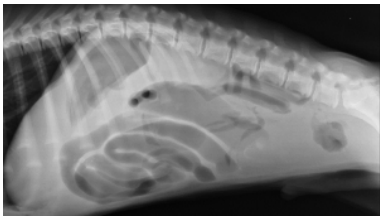
Reproduced from Maskell IE, Johnson JV. Digestion and Absorption. In: The Waltham Book of Companion Animal Nutrition. Editor: Burger IH.Pergamon Press, Oxford, UK. 1993, pages 25-44

In veterinary medicine, we commonly listen for gut sounds. However, this is a very nonspecific and insensitive test. Contrast radiographs, contrast fluoroscopy and ultrasound are the most commonly used tools for more accurate diagnoses.

Contrast radiographs provide a snapshot at one point in that can at least suggest activity or show distension.



normal contrast radiograph



excessive SI distension

Ultrasound can show activity; however, activity doesn't always mean motility (uncoordinated activity can lead to poor motility)

Intestinal ultrasound – types of activity

Intestinal ultrasound equine

Colon scan canine (scan starts at 1:20)

Emptying studies are more accurate than ultrasound but are more involved.

Stomach activity (human) – barium study

Jejunal fluoroscopy

Sheep colon fluoroscopy

What is a fluoroscopy (human)

Indigestible markers can be used to evaluate total transit time. In horses, mineral oil is used as a tool, with passage expected in 12-24 hours. For more detailed evaluation, plastic beads or radioactive markers are used.

Abnormal motility disorders

Lack of motility or activity in ultrasound and contrast studies is generally abnormal.

Reverse flow of ingesta in the esophagus, stomach and SI is abnormal in non-ruminants.

Changes in stool (softer, dryer, different shape or color) are major clues to changes in the GI tract.

33. Additional resources

Overview

Gastrointestinal physiology – great overview for those that want more in depth material-slideshow

Gastrointestinal physiology -another great review in text form

The enteric nervous system – more succinct version of the enteric nervous system

Control of the GIT – Khan academy

Digestion and absorption

Veterinary hematology and clinical chemistry, Thrall – free ebook through UMN library; very useful for diagnostics

Digestive regulation part I – Gastrin – regulation, neurotransmitters etc

Chemical digestion and absorption – brief, useful overview.

Protein digestion – includes biochemistry

Carbohydrates and sugars – more biochemistry and good explanations of different types of sugars

Fats – ditto for fats

Absorption – more by lumenlearning

Absorption of nutrients, Khan Academy

Zymogen activation, Khan Academy

The horses digestive system and how it can fail – if you are really into horses, feed company sponsored

Gastric control

Three phases of gastric secretion

34. Practice

Exercises

Predict clinical signs and related pathology (fluid and electrolyte loss, damage to other structures) that will happen if :

1. the cricopharyngeal sphincter doesn't open?
2. the epiglottis doesn't close?
3. the upper esophageal sphincter doesn't open?
4. the lower esophageal sphincter doesn't open? stays open?
5. the gastroduodenal sphincter doesn't open? stays open?

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Challenge exercises – research and explain a few of these to a friend or relation to boost your understanding and retention

General

- What sort of meals make you feel full?
- What are the current recommendations for endurance runner diets?

- Who tends to have lactose intolerance, what are the signs and treatments?
- Where do vitamins get absorbed?
- How would dysfunction in each of these areas affect weight gain/nutrition?
    - duodenum
    - jejunum
    - ileum
    - lack of absorption of carbohydrates
    - lack of absorption of protein
    - lack of absorption of fats
- What vitamins and minerals are required for your species of choice? how does this compare across species?
- How do Zn and Cu help the digestive health of young animals?
- If the secretory line in the villi is disturbed (no stem cells), what happens re: other cell types and function?

Exotics

- Explain copraphagy – what is it and why is it beneficial?
- Explain cecotrophy
- How do other fermenters compare to cattle or horses? Eg sloths, marsupials, reptiles, lagomorphs, etc

Ruminants

- How do you diagnose and treat rumen acidosis?
- When and why do cows get ketotic? How is it

treated?

- Explain pregnancy toxemia in sheep
- Why do we perform rumen transfaunation in cattle? Would it work in other species?
- How does a transition ration work in cattle (from pregnancy to lactation)?
- Explain "if we can feed the bugs, we can feed the cow"
- Why is feeding a total mixed ration (TMR) considered better than component feeding in dairy cattle?
- Explain subacute rumen acidosis and weight loss

Horses

- What are the side effects of grain overload in horses?
- If a colon resection is performed, how does the horse manage hay?
- Horses don't have a gall bladder. How is bile managed?

Small animal

- If too much small intestine is removed, dogs and cats can suffer from short bowel syndrome. What are the therapies needed?
- What happens if cats are fed a vegetarian diet?
- What happens if we feed dogs raw meat?
- Can a dog or cat eat a paleo diet?
- Can cats get ketosis? How do they use ketone bodies?

Swine

- What does it mean if pigs don't have paneth cells?
- How does Lawsonia affect the viili?

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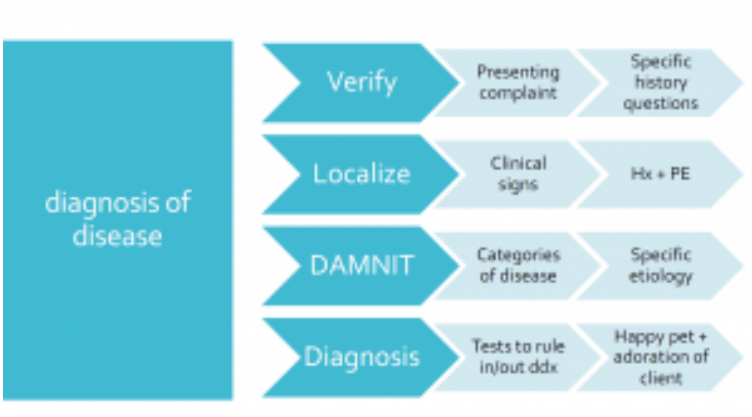
PART IV

ORAL CAVITY AND ESOPHAGUS

Problems with chewing, swallowing and esophageal transit lead to food coming back out the oral cavity and obviously impact nutritional absorption. Differentiating between the three is important to develop accurate differential lists, select diagnostic tests and to choose therapies.

From SAM II Dr. Granick:



Clinical signs associated with Disease by Location

	Oral Cavity	Esophagus	Stomach
Dysphagia	+	+/-	–
Regurgitation	–	+	–
Vomiting	–	–	+
Salivation	+	+	+
Gagging	+	–	–
Drinking	Abnormal	Normal	Normal
Bolus Formation	–	Normal	Normal
Dropping Food	+	–	–
Food ejection	Immediate	Variable	Delayed
Character	Undigested	Undigested	Digested
Odynophagia (painful swallowing)	Occasional	Frequent, but variable	Absent
Swallowing	Multiple attempts	Single/Multiple	Single
Other signs	Discharge	+/- Cough/Dyspnea	Retch

35. Dysphagia

Dysphagia is difficulty chewing and/or swallowing.

Pathophysiology

General types of dysphagia are classified as oral, pharyngeal or cricopharyngeal. They can also be grouped as 1) pain, 2) neuromuscular, 3) obstructive and 4) mechanical (similar to pain).

Patients with dysphagia will present for

- Gagging
- Difficulty drinking
- Unable to make a food bolus
- Ineffective swallowing
- Drooling
- Dropping food
- Nasal discharge or cough

Normal chewing requires cranial nerve V, non-painful dentition, chewable feedstuffs and normal musculature. Dental disorders are a common cause of dysphagia. Abnormal tongue function, painful or nonfunctional muscles and neuropathies can also affect prehension and chewing.

Swallowing requires central stimulation and involves opening of the upper esophageal sphincter, movement of the soft palate upwards to close off the nostrils and retroflexion of the epiglottis to cover the airway. Cranial nerves IX, X and XII are all involved. Problems with any of those steps can lead to dysphagia and dropped food.

Diagnostics

Consider rabies and wear protective gear.

Watch the animal eat or drink.

Usually prehension abnormalities are easy to identify as the food or water never makes it into the mouth. Asymmetrical chewing or dropping of unchewed food particles (quidding) are commonly seen with painful chewing. Basically what goes in is what falls out.

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Oral exams and cranial nerve evaluations

These are crucial to localize lesions. Check for tongue and tooth abnormalities as well as foreign bodies or masses. Full evaluation of cranial nerves is important.

Advanced diagnostics

If there are no visible oral lesions or obvious tooth decay, consider dental radiographs and videoendoscopy. Biopsy any masses observed. Referral may be needed for testing and/or therapy.

Diagnostics include barium swallows, cranial nerve exams and endoscopy.

Therapy

Dysphagia is generally not an emergency unless patients are dehydrated or in respiratory distress.

Surgical repair is indicated for forms of trauma (jaw fracture, tooth fracture). Extracting painful teeth can rapidly resolve issues. Surgical myotomy is indicated for cricopharyngeal achalasia (failure of the upper esophageal sphincter to relax) and arterial occlusion for equine guttural pouch mycoses (fungal infection that affects the nerves involved in swallowing).

Ruminants may have significant metabolic acidosis and dehydration from salivary loss. Both small and large patients may need re-hydration and slurry or nasoesophageal feeding, as well.

Aspiration pneumonia is common in dysphagia cases as the food goes down the trachea instead of the esophagus; antibiotic therapy is required.

Many types of dysphagia can only be treated supportively; for many, there is no available treatment and supportive care is often lifelong.

Key Takeaways

- Dysphagia is difficulty chewing; regurgitation is retroperistaltic esophageal activity.
- Dysphagia can occur due to nerve/muscle issues,

pain, dental issues or foreign bodies/masses. Atypical regurgitation can occur due to nerve/muscle issues, peristaltic disorders, or foreign bodies/masses.

- Diagnostic tests include feed trials, oral exams, radiographs, endoscopy, and/or fluoroscopy

Just for fun

Polymyositis dysphagia case- be sure to look up the terms and conditions described

Achalasia dogs – do more review on this one too!

Barium swallow in a person– mostly normal

Barium swallow – esophageal pathologies– description of a variety of images (human)

Swallowing disorders and achalasia – description of the pathophysiology (human)

Megaesophagus in dogs – proceedings

36. Regurgitation

Regurgitation is the release of food back out of the mouth when the food has not reached the stomach (undigested). It is usually related to esophageal dysfunction or obstruction.

Pathophysiology

Esophageal disorders lead to regurgitation or retrograde flow of the bolus back up and out the mouth. Most disorders are related to poor muscle tone or neurogenic disorders affecting the esophagus.

Physical obstructions can occur with food, treats and toys or with esophageal strictures. Strictures are relatively common after esophageal trauma (eg after choke in horses). Occasionally congenital anomalies (eg persistent right aortic arch) entrap the esophagus, creating a physical obstruction.

Idiopathic (unknown cause) megaesophagus is the most common cause of regurgitation in dogs 7-15 years of age. Congenital megaesophagus and myasthenia gravis can cause similar signs as can several other endocrine disorders.

Diagnostics

Watching the animal is important to differentiate between regurgitation and vomiting. Depending upon the history, further testing may be important to differentiate physical obstruction from functional obstruction.

Regurgitation	Vomiting
Passive	Active-heaving
Minimal warning	Owners hear retching
Owners don't hear it	Prodromal phase – nausea (lip licking, salivation)
Food undigested	Digested food
Tubular in shape	Bile staining possible

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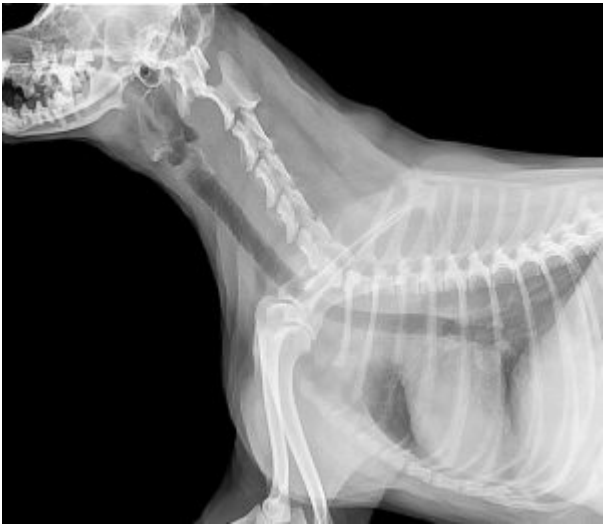
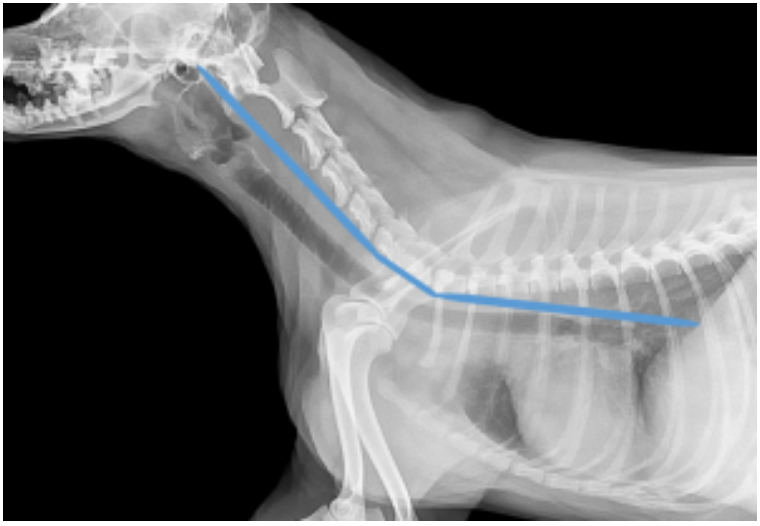
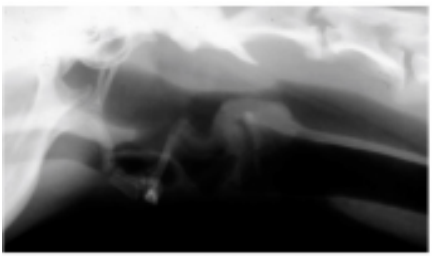
Food, treats and toys causing physical obstruction can be visualized with radiographs (SA) and endoscopy (LA).

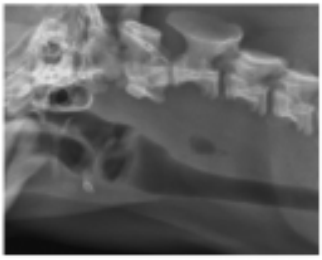
The esophagus is not generally radiographically visible in normal animals.



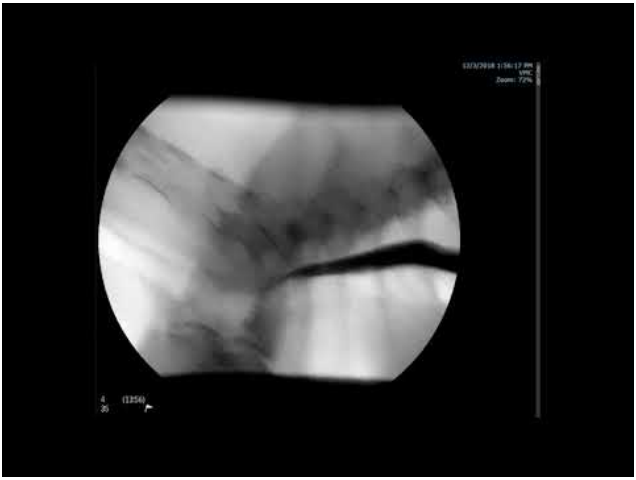


Animals with esophageal dysfunction may have swallowed air present in the esophagus.





Contrast studies using regular films or fluoroscopy can help identify megaesphagus and motility disorders. Food is mixed with barium to be able to follow the food during imaging.



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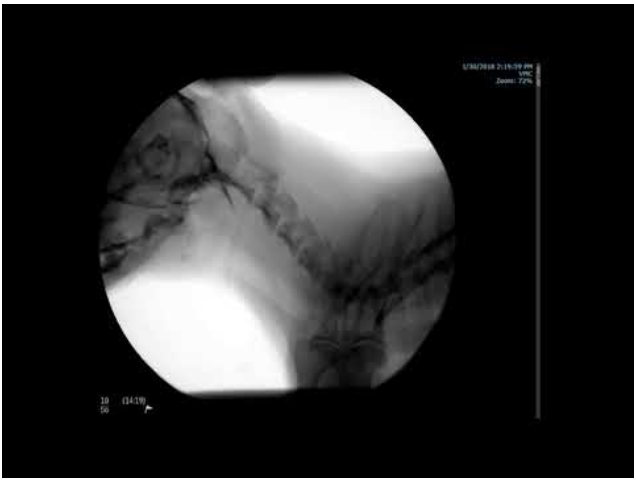

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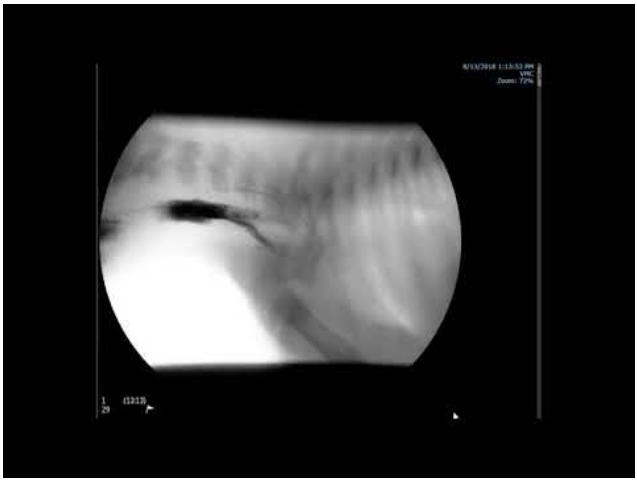

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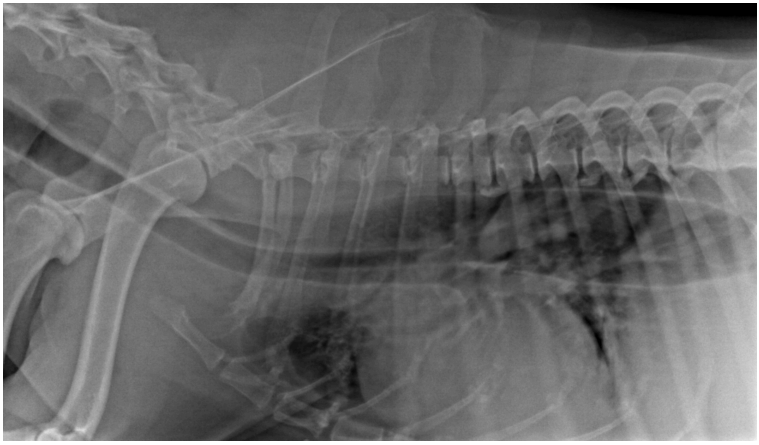
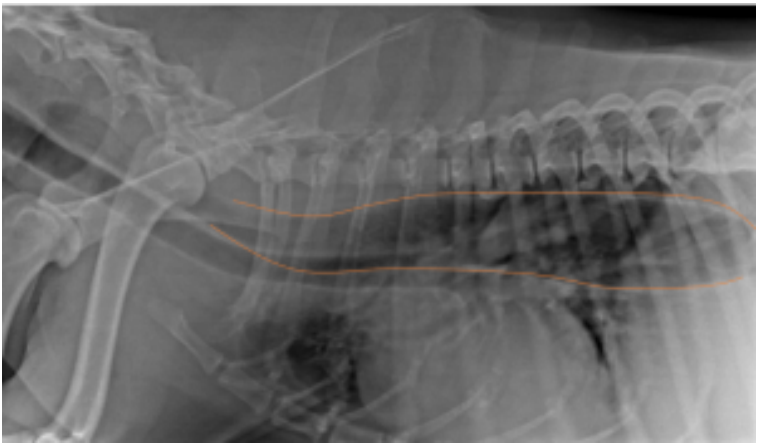
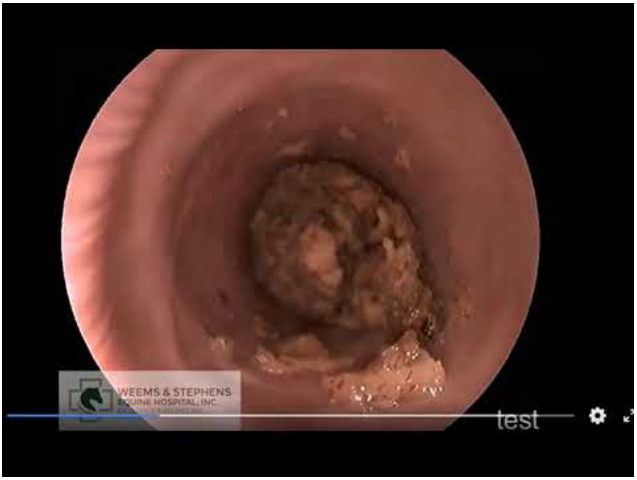
Megaesophagus films





Endoscopy is very useful in horses since they can be scoped while standing and awake.



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Dogs with regurgitation without a diagnosis should be tested for myasthenia gravis (test for anti-acetylcholine receptor antibodies), for thymoma via thoracic radiographs, Addison's disease via ACTH stimulation test, hypothyroidism via a thyroid panel (T_4, FT_4, TSH) and for lead toxicity.

Therapy

Many esophageal obstructions can be removed endoscopically. Myasthenia gravis is treated with anticholinesterase inhibitors. Immune or inflammatory conditions can be treated with systemic or local steroids. Vitamin E and thyroid supplements are given for for related deficiencies.

Most other disorders, including megaesophagus, are treated symptomatically.

Key Takeaways

	Dysphagia	Regurgitation
Eating trial	Watch to see if can chew and swallow	Watch to see if stuff comes up and if abdominal effort
Oral exam	Check for dental issues, oral sores and oral foreign bodies	
Radiographs (plain)	Check for dental issues, foreign bodies and masses	Check for foreign bodies and masses
Radiographs (contrast)	Check for air in the esophagus	Check for normal bolus movement
Endoscopy	Check teeth and pharynx	Check for foreign bodies, masses and activity
Fluoroscopy	Evaluate swallowing	Check for normal bolus movement

Just for fun

How does my stomach work – good explanation of esophageal reflux disease and hiatal anatomy

Vomiting cat – Shrek

How bird vomit helps us understand history – really bird regurgitation

37. Vomiting and Reflux

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Pathophysiology

Vomiting is a common non-specific condition that affects small animals, pot bellied pigs and some exotics. Vomiting is not routinely seen in horses, camelids or ruminants.

With vomiting, the stomach, abdominal muscles, and diaphragm contract. The lower esophageal sphincter opens. The palate and epiglottis move up to protect the airway. (see link)

Vomiting comes with a very broad list of differential diagnoses,

including both GI disorders (obstruction, ulcers, ileus; primary GI) and secondary disorders such as toxin/infection exposure, abdominal pain, endocrine or intracranial processes (secondary GI). They all work through the vomiting center in the brain.

Primary GI causes

Primary GI refers to cases in which the GI tract itself is involved.

Primary GI disorders that cause vomiting can usually be classified as infiltrative, obstructive, infectious, dysmotility and toxins. Examples include canine gastric dilatation volvulus (GDV), foreign body, gastric ulcers, intussusception, parvoviral enteritis, canine distemper, leptospirosis, internal parasites (stomach worms, roundworms), and hemorrhagic gastroenteritis.

Primary GI is more likely if the animal is not overly sick, has something abnormal on abdominal palpation (foreign body, intussusception) or has significant diarrhea. Generally a CBC, profile and UA are most helpful to rule out a secondary GI disorder.

Some of these animals will need surgery!

Secondary GI causes

Secondary GI refers to causes of GI type clinical signs that are related to organ dysfunction that isn't the GI tract.

Secondary GI disorders that cause vomiting include renal disease, hepatic disease, pancreatic or endocrine disease, adrenal disease (Addisons), peritonitis, hyperthyroidism, or CNS disease (cortical disease, vestibular disease). Besides organ failure, disorders include pyometra, diabetic ketoacidosis, motion sickness, ear infections, and septicemia.

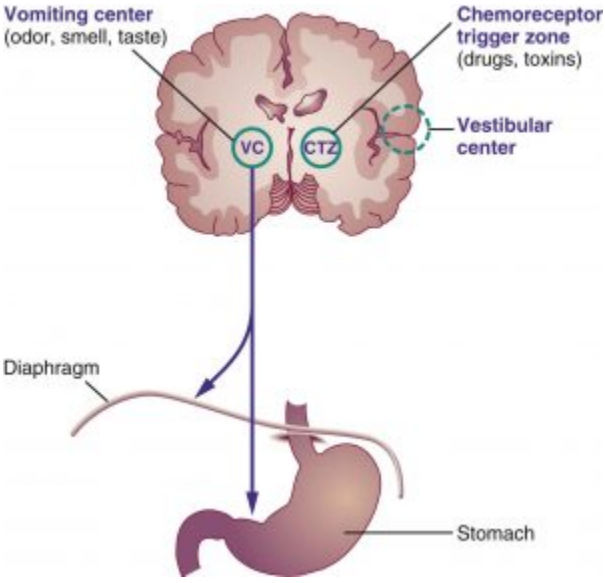
With secondary GI issues, animals are often sick and generally are normal on abdominal palpation.

Generally, surgery and anesthesia is contraindicated!

Differentiating primary and secondary GI issues

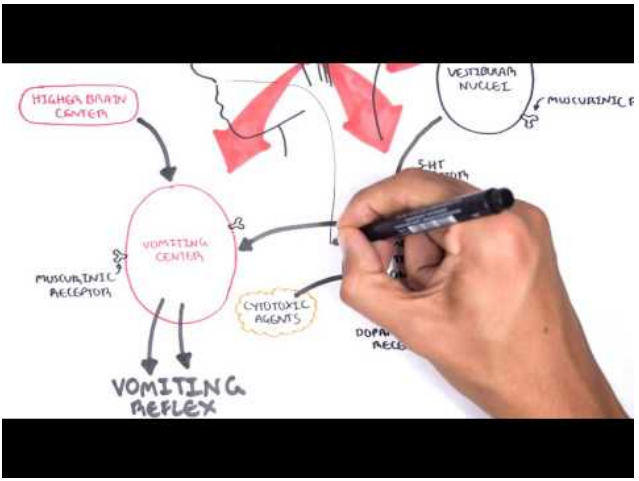
	Primary GI	**Secondary GI**
History	Dietary indiscretion, dietary changes, missing object	Car ride, not spayed, weight changes, head trauma
Physical examination	Pain or masses on abdominal palpation. Otherwise healthy	Sick, fever, thyroid enlargement
Radiographs (plain or contrast)	Check for foreign bodies or masses	Check for enlarged uterus or liver
Fecal/UA	Check for parasites	Check for kidney issues
Blood work	Dehydration	Check for systemic disease, hyperthyroidism

Vomiting reflex



https://basicmedicalkey.com/gastrointestinal-tract-disorders/

All vomiting stimulants act through the vomiting center in the brain.



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The 5 main neurotransmitters involved in the vomiting pathway:

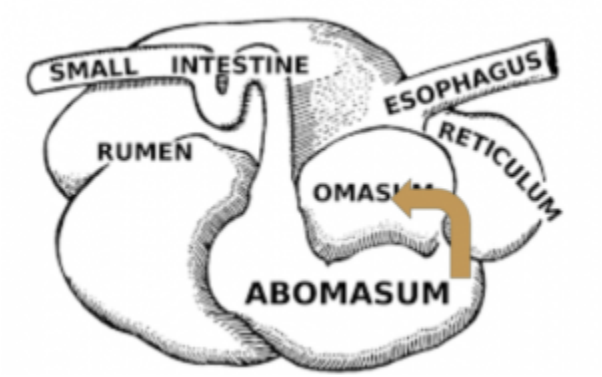
- muscarinic (vagus nerve)
- dopamine
- histamine
- serotonin
- neurokinin

Maropitant (Cerenia^{R}) is a highly effective drug in small animals, blocking Substance P (NK-1) receptors in multiple sites. Ondansetron is usually the second choice.

Reflux

Horses can't vomit but still have the same fluid backup. Due to the strong lower esophageal sphincter, the stomach will often rupture before the animal can actually vomit. When a horse is colicky, it can be life saving to pass a stomach tube and relieve the fluid. Instead of vomiting, we say horses are **refluxing**.

Cattle and camelids don't usually vomit. In these animals the fluid that would be vomited moves into the omasum, reticulum and/or rumen (cattle) and C2 and C1 (camelids).



Electrolyte and acid base changes

The stomach contents are acidic. If the vomitus/reflux is due to an upper GI problem, the animal loses hydrogen and chloride. Ruminants with abomasal outflow obstruction still lose the HCl but it ends up in the rumen. All animals with an upper GI obstruction tend to hypochloremic. Cattle usually have hypochloremic metabolic alkalosis. Dehydration makes animals acidotic so small animals may not show the alkalosis but may actually be acidotic.

Differentiating dysphagia, regurgitation and vomiting

	Difficulty chewing or swallowing?	Retching noises?	Abdominal effort?
Dysphagia	yes	no	no
Regurgitation	no	no	no
Vomiting	no	yes	yes

History and other clinical signs can be useful.

Diagnostics for vomiting

Diagnostics are often NOT performed for acute cases of vomiting. Supportive care is provided and diagnostics only performed if the animal doesn't respond. One common "test" is response to maropitant (antinausea drug). Maropitant usually controls vomiting due to brain or non-GI lesions. If the animal continues vomiting, this is considered an indication of a surgical lesion.

Key factors:

	Primary GI	Secondary GI
History		Head trauma
Physical examination	Abnormal abdominal palpation. Otherwise healthy	Sick. Fever.
Lab work	Dehydration	Abnormalities
Response to maropitant	Not if surgical	Yes

Diagnostics for severe or chronic vomiting might include

SA Tier 1

- Fecal evaluation –internal parasites
- CBC or PCV/TPP- hydration status, signs of infection or inflammation
- UA-urine specific gravity, UTI, proteinuria, glucosuria
- Chemistry – organ function (kidney, liver, pancreas), electrolyte levels
- Baseline cortisol – Addisons in dogs
- Blood gas/istat- electrolyte, acid base status
- T4-hyperthyroidism- cats > 7yo

- Abdominal radiographs-foreign body, gas distension in stomach/SI/LI, feed/fecal obstruction, lymphadenopathy, intussusception
- Thoracic radiographs- pneumonia
- Parvo SNAP test- kittens and puppies with diarrhea and vomiting

SA Tier 2

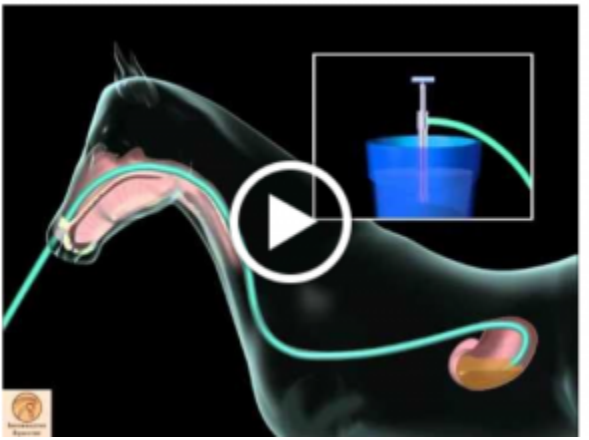
- ACTH stim-adrenal insufficiency
- Abdominal ultrasound/AFAST-free fluid, intestinal activity, abnormal densities, intestinal wall thickness
- Contrast upper GI study
- Bile acids, cPLI/fPLI

SA Tier 3

- CT -cranial, thoracic or abdominal disease/masses
- Endoscopy- esophageal/gastric ulceration/obstruction
- Surgery– abdominal masses, obstruction

LA diagnostics

- Check for reflux by passing a tube into the stomach



- Check rumen pH to determine if it is lower than usual due to the addition of acidic stomach contents

Therapy

Drugs are available to block each part of the vomiting pathway in small animals. In veterinary medicine, the first line agent is typically maropitant.

Maropitant blocks the NK-1 receptors, keeping substance P from binding and starting the vomiting process.

Odansetron blocks 5-HT3 serotonergic receptors and is used if maropitant isn't working.

Metoclopramide is used, particularly if there is a motility disorder, as it stimulates gastric emptying. Metoclopramide blocks dopaminergic function in part.

Antihistamines can also be useful but have sedative effects as well.

In most clinical situations vomiting is acute, self-limited, lasting

for several hours to days. Acute vomiting is often treated supportively. Chronic vomiting represents symptoms present for greater than 7-10 days or unresponsive to initial therapy; such patients need a more involved diagnostic evaluation.

In addition to identifying the underlying cause of the vomiting, complications of vomiting such as dehydration, acid-base and electrolyte abnormalities, gastroesophageal-reflux disease (GERD), weight loss and aspiration pneumonia should be identified early in the patient assessment.

GERD occurs due to damage of the esophagus by refluxed (vomited) gastric juices. Aspiration pneumonia due to inhalation of vomitus. As stomach and biliary fluids are lost, animals may develop related electrolyte and acid-base changes. Saliva contains large amounts of bicarbonate. If the fluid lost is primarily saliva, the animal will become acidotic.

Fix electrolyte and acid-base abnormalities.

Supportive care for vomiting may include

- No food for 6-12 hours, then a light diet
- Fluids
- Antiemetics (maropitant)
- Prokinetics (metoclopramide)
- Antacid treatment
    - H2 blockers (famotidine, ranitidine)
    - Proton pump inhibitors (omeprazole)
    - Coating agents (sucralfate)
- Antimicrobials
- Psyllium

- Specific antitoxins
- Primary disease related therapy (eg insulin)

Pharmacological stimulation of vomiting

Apomorphine is used to stimulate vomiting when an animal has ingested something it shouldn't. Apomorphine works by stimulaing dopamine receptors. Dilute hydrogren peroxide can also be used in certain species; however it is basically being used for its toxic effects, ideally at a low dose.

Vomiting should not be induced if it is more than 1-2 hours after eating (the substance has left the stomach) or if coming back up would be just as bad (alkali or corrosive substances, etc).

Key Takeaways

- With vomiting, the stomach, abdominal muscles and diaphragm contract. The lower esophageal sphincter relaxes. Abdominal muscle contraction is a key factor that differentiates vomiting from regurgitation and dysphagia.
- All vomiting is initiated by the vomiting center. The major neurotransmitters are 5HT3, NK-1 and dopamine. The vagal nerve plays a role in sending

signals to the vomiting center.

- Vomiting can occur due to primary GI issues (problems with the GI tract) or secondary GI issues (problems with other organs).

Resources

Vomiting in pets – WSU CVM

Digestive system part 2 – an additional look at digestion or skip to 8:50 to see the vomiting part

Physiology of vomiting – video – more details but good

Dog vomiting causes and treatment – Purina blog

Colic in horses when is surgery necessary – The Horse article

Just for fun

Vomiting vs regurgitation with Dr. Foley – animated description

Hypertrophic pyloric stenosis – occasionally seen in small animals; we do get similar conditions due to tumors and adhesions ; good anatomy/physio review

Vomiting in pigs– the pig site typically has good info!

Apomorphine administration -> vomiting; useful for toxins that have recently been ingested

Inducing vomiting, VSPN

Pharmacology – Antiemetics– more drugs than vets typically use but good info

How does my stomach work – good explanation of esophageal reflux disease and hiatal anatomy

Physiology of vomiting (dogs) – fancier than it needs to be

Nausea and vomiting (people); shorter than some others and easy to understand

38. Practice

Board prep questions below

Challenge Questions

General

- Explain mechanisms and testing involved in dysphagia associated with
    - Prehension
    - Mastication
    - Swallowing
    - Cranial nerve dysfunction
    - Aspiration pneumonia
- Explain to a client how to differentiate dsyphagia from regurgitation and vomiting
- Explain mechanisms of action for
    - NSAIDs
    - Corticosteroids
- why do animals drool with rabies? red clover? motion sickness?
    - what does saliva help with anyway?

- Use the DAMNIT-V scheme to work through differentials for dysphagia in your favorite species
- How can you use dysphagia to help localize neurological lesions?
- Develop a decision tree for dysphagia or for regurgitation, showing diagnostic, treatment or prognosis options for each category:
    - infection, inflammation, trauma neoplasia, neuromuscular disorders.
- define prehension, mastication , deglutition, odynophagia
- Use a flowchart to identify site of dysfunction in eating
- identify signs related to dysphagia
- match cranial nerves to dysphagia types

Small animal

- Explain mechanisms and testing involved in dysphagia associated with
    - Cricopharyngeal achalasia
    - Myasthenia gravis
    - Tick paralysis
    - Rabies
    - Muscular dystrophy
    - Addison's disease
- Explain mechanisms of action for
    - Edrophonium
    - Pyridostigmine

- Why do we give atropine to small animal patients in anesthesia but not to large animal patients?
- How do we manage megaesophagus patients, particularly for eating?

Equine

- Explain mechanisms and testing involved in dysphagia associated with
    - Guttural pouch infections
    - Temporohyoid osteopathy
    - Tetanus
    - Botulism
    - Strangles
    - Ulcerative stomatitis
    - White snakeroot
    - Moldy corn
    - Overgrown teeth
- What causes choke in horses and how is it treated?

Ruminants

- Explain mechanisms and testing involved in dysphagia associated with
    - White muscle disease
    - Bovine viral diarrhea
    - Bovine leukosis virus infection
    - Malignant catarrhal fever
    - Scrapie
    - Ulcerative stomatitis
    - Locoweed
    - Woody tongue (*Actinobacillus*)

- why do cows salivate so much? what happens if they have choke?

Avian

- what are in raptor pellets?
- What is involved with parent birds feeding baby birds?

What electrolyte disorders could develop with chronic vomiting or refluxing?

When do you use apomorphine to stimulate vomiting in dogs?

How are cats different?

Explain the medical management of these causes of vomiting:

- Motion sickness
- Chemotherapeutic drugs
- Xylitol toxicity

General

Use the DAMN-IT scheme to work through 6 differentials for vomiting in your favorite species

Explain how you can use vomiting to localize neurological lesions

Explain how these drugs help vomiting and what types they help

- maropitant
- metoclopramide
- ondansetron

- prochlorperazine

Small animal

How or why do these cause vomiting?

- motion sickness
- inner ear infections
- gastric ulcers
- pancreatitis
- chemotherapeutic drugs
- renal disease
- toxicity
- infection
- pyometra

Explain the medical management of

- motion sickness
- chemotherapeutic induced nausea
- xylitol toxicity

We often feed a light diet to vomiting dogs and cats. What does that look like?

What can you use /not use to stimulate vomiting in cats?

How does activated charcoal help?

Top 10 animals being sick, part 3– can you tell the difference between the regurgitating and vomiting ones?

More : Top 10 animals being sick, part 1; top 10 animals being sick, part 2

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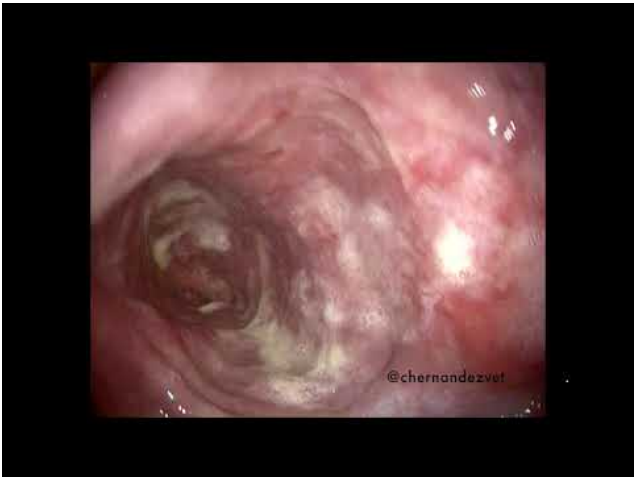
Cases

Can you identify the abnormalities?



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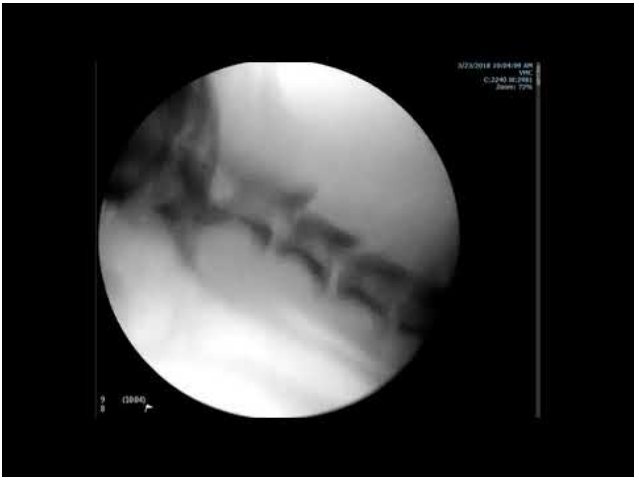

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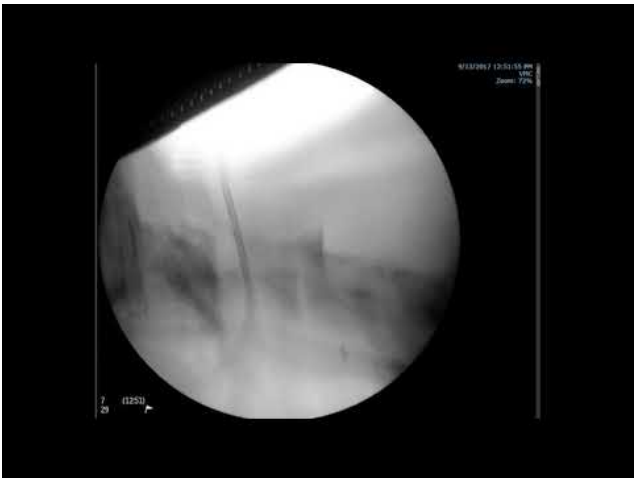

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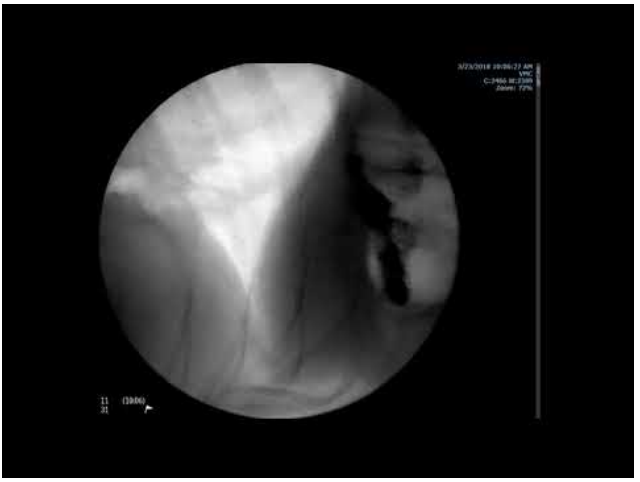

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PART V

GASTRIC AND SMALL INTESTINAL DISORDERS

Upper GI disorders – stomach and small intestine

39. Gastric ulcers

Gastric ulcers

Gastric ulcers are also called peptic (pepsin) ulcers.

Prostaglandins are important for mucus production in the stomach. The mucus protects gastric cells from gastric juices. Non-steroidal (and steroidal) agents prevent prostaglandin production and subsequently prevent mucus production. The cells exposed to the gastric juices can be damaged => gastric ulceration. Neoplasia, chronic inflammation and chronic hyperacidity (gastrinoma, mastocytosis, head trauma) can also cause ulcers. Both young and old animals are at greater risk.

The esophagus and nonglandular portion of the stomach (camelids, horses) can also be damaged by stomach acids with vomiting and perhaps with certain forms of exercise (equine).

In non-ruminants, ulcers can be visualized endoscopically. Ulcers may be accompanied by anemia and hypoproteinemia as well as anorexia, weight loss, and abdominal pain. Fecal occult blood tests can be useful in herbivores. Deep ulcers can perforate, leading to peritonitis. The gastric lining can repair itself, so not all damage leads to ulcers or problems.

Ulcers are treated by:

(1) stopping NSAID use

(2) protecting the ulcers with sucralfate – this drug creates a sticky gel when exposed to acidic environments

(3) blocking HCl production

(a) antihistamines – block the histamine receptor that signals HCl production. Ex – famotidine

(b) proton pump inhibitors – directly blocks the HCl pump. Ex- omeprazole

(4) misoprostol is a synthetic prostaglandin used to prevent or repair damage

In a recent study, misoprostol was more effective than omeprazole in treating equine glandular ulcers. These seem to be different then the more common squamous ulcers that do respond well to omeprazole. As the glandular stomach should have less acid exposure, other mechanisms may be more in play than damage due to acid. Glandular ulcers may be increasing in prevalence.

Drug treatment is harder in ruminants as many drugs do not make it through the rumen or are too expensive for cattle.

Resources

Peptic ulcers– good review of anatomy and physiology as well as ulcers in people

How nsaids create ulcers – simple but useful depiction

Ulcers pathophysiology and treatment – set to classical music

Horse stomach acid – nice short overview (different gastric structure than dogs)

Just for fun

40. Gastric dysfunction

Gastric dysfunction

The stomach can have issues. Animals can develop gastric impactions, emptying disorders, volvuluses and gastric foreign bodies (toys, bones, bezoars). The most common lesions are gastritis and gastric ulcers.

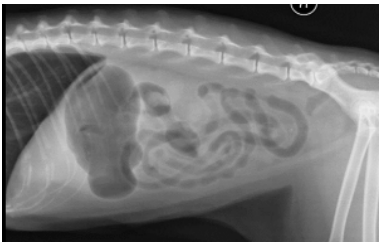
Dogs, cattle and small ruminants are fairly indiscriminate eaters and commonly have foreign bodies in the stomach (forestomach in ruminants). Generally these require surgery. Ruminants can eat sharp objects that puncture the reticulum, creating local or diffuse peritonitis. The heart sits on just the other side of the diaphragm, making these animals also prone to pericarditis and pleuritis.

Gastric impactions in horses can be related to poor motility (Friesians are prone) and to dehydration. Horses that eat fresh cut grass can end up with gastric impactions/ gas due to fermentation. Horses don't usually eat things they shouldn't but occasionally develop gastric foreign bodies. Persimmon seeds can be an issue. Occasionally stomach tubes break and parts end up in the stomach. This is most common in cold climates.

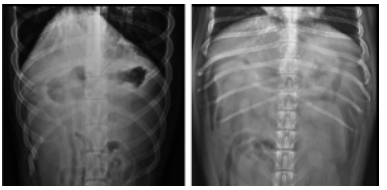
Diagnostics

Gastric function can be evaluated by contrast radiographs in smaller animals. Endoscopy can be helpful in larger animals. Ultrasound may show gastric distension or gastric emptiness but is not often helpful in identifying motility patterns. Breath hydrogen levels and acetaminophen absorption are used in research studies.

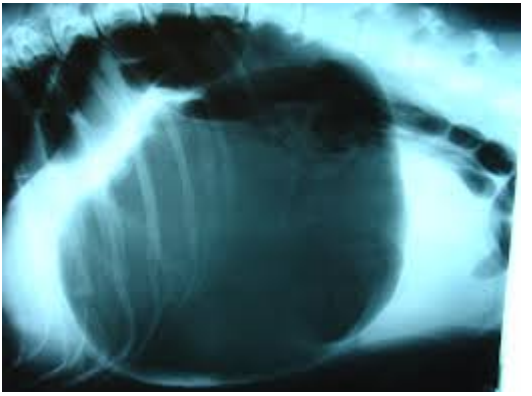
The stomach can be relatively distended in normal animals but should change over time.



normal stomach distension



the stomach should not be static in size on repeat radiographs



This is abnormal gastric distension

Gastric dilatation in a dog

41. Small intestinal obstruction

Pathophysiology

The small intestine may be physically or functionally obstructed.

Functional obstruction is due to lack of motility but there is not physical obstruction. Many animals experience postoperative ileus (especially horses and humans). During POI, inflammation disrupts normal nerve function. Animals can also get ileus from enteritis (inflammation of the intestines). Hypocalcemia is another common cause of functional obstruction – no smooth muscle action means no peristalsis.

Physical obstruction can occur due to intraluminal or extraluminal obstruction. Intraluminal obstructions can be due to food material, tumors, enteroliths, foreign bodies and the like. Extraluminal obstructions can be due to gut twists and displacements, tumors, adhesions, hernias and changes in other peritoneal structures that pinch the gut closed.

All animals can develop intussusceptions – one part of the bowel telescopes (folds into) another part. This is particularly common in younger animals with diarrhea and in older animals with tumors. Brown Swiss cattle of all ages are predisposed (no idea why).

Intestinal torsions are also common – the gut twists on itself. These are most common in younger animals.

Dog and cats are prone to foreign body obstructions. Cats like to eat string and thread. These linear foreign bodies can then "plicate" or "fold up" the SI as they get stuck in the SI.

In cattle, a relatively more recent form of physical obstruction is blood clot disease – hemorrhagic gastroenteritis. We believe this is a clostridial or fungal problem causing bleeding into the gut lumen. It almost always causes death.

An obstruction in the small intestine stimulates local contractions to push the obstruction distally. Secretions are increased to "liquefy" the contents. Scant soft feces may be seen in partial obstructions as fluid but not ingesta can pass.

With complete obstructions, the secretions eventually back up into the stomach and are vomited in small animal patients. In horses, the stomach may rupture unless a nasogastric tube is placed to remove the fluid.

In ruminants, the secretions back up into the abomasum. Abomasal fluid eventually refluxes back into the rumen. A low pH in the rumen will also shut down motility of the forestomachs.

Diagnostics

The most common finding is vomiting or refluxing. Animals are often sick – dehydrated with electrolyte and acid base abnormalities. Abdominal pain is related to intestinal distension or traction on the mesentery. Functional obstruction is typically a systemic problem and affected animals may show other systemic signs such as fevers and depression. In smaller animals, abdominal palpation may be useful but animals may be too painful to allow good palpation. Radiographs are useful in smaller animals, particularly contrast radiographs. Ultrasound can occasionally identify lesions such as intussusceptions.

Vomiting cats and dogs that do not respond to maropitant are likely to have a physical obstruction.

Animals with SI obstructions are not usually bloated.

Therapies

Treatment of functional obstruction includes supportive care while mother nature works, iv lidocaine or other motility agents, and

surgery to decompress the intestines. Moving the fluid out of the SI is often enough to let them restart normal activity. Most physical obstructions require surgical removal.

42. Enteritis / SI diarrhea

Pathophysiology

Diarrhea may be due to one or more of three main forces

Secretory diarrheas

Example – cholera, giardia and ETC (enterotoxigenic E coli) In these cases, the crypt cells are stimulated to release chloride ions. This release then leads to water flow into the gut lumen due to the change in concentration gradient.

Malabsorptive diarrheas

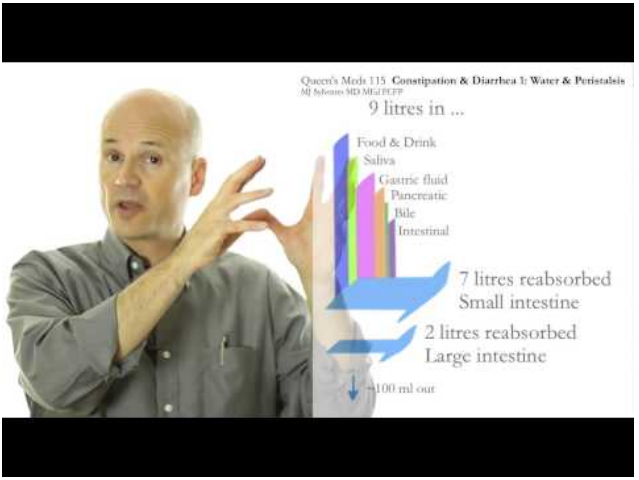
Example – rota/coronavirus diarrhea. In these cases, the enterocytes are destroyed or damaged. Water cannot be resorbed properly. This is often associated with increased osmotic fores as well. This means that food components are not being absorbed and stay in the lumen. This creates a concentration gradient that pulls water from the blood into the gut lumen, creating diarrhea. Many of these organisms also impact the nervous system, causing hyperactive peristalsis. This can also limit digestion and absorption through **decreased transit time.**

Many of these disorders lead to disruption of the tight junctions and allow lots of things to cross the gut barrier – bacteria, large proteins, electrolytes. This can disrupt the active transport systems, cause weight loss and lead to immune system compromise and response.

Osmotic diarrhea

Example – lactose intolerance. When food stuffs cannot be digested, they stay in the lumen, again creating a concentration gradient. Many of these come with bloating as the food stuff may actually be digestible by the colonic bacteria.

Increased flow is due to osmotic forces whereby solutes in the lumen lead to higher osmotic forces moving fluid in the same direction. It can also be due to leakage of tight junctions and damaged cells such as occurs with inflammation and various intestinal toxins. Bleeding is likely related to loss of barrier function and/or cell damage.



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Causes include

- infectious agents (viral, bacterial, fungal, protozoal, parasitic)
- dietary (coprophagy, grain overload, lactose, sand)
- antibiotic associated (changes in normal gut flora)
- changes in blood or lymphatic flow (hypertension, lymphatic obstruction)
- infiltrative disease (immune cells, tumor cells)
- intestinal accidents (ischemia,intussusceptions, short bowel syndrome)
- endocrine disorders (hyper/hypothyroidism, Addisons disease)
- toxins and drugs (NSAIDs)
- pancreatitis, malabsorption
- dysmotility -stress, nervous system dysfunction

Diagnostics

While the signs of diarrhea are pretty easy to recognize, it can sometimes be useful to differentiate small intestinal from large intestinal diarrhea. Small intestinal diarrhea often causes fever, dehydration and eventually weight loss.

Often we want to identify the causative agent in order to identify those that are infectious : parvovirus, Salmonella, African swine fever, giardia etc.

It can be helpful to differentiate SI diarrhea from LI diarrhea. Mixed cases are common but often one type predominates.

Therapy

Therapy is often supportive. Antiparasite drugs can be useful for parasite induced diarrhea. Many cases of bacterial diarrhea are

actually not responsive to antibiotics. Fluid therapy is key. Electrolyte replacement is often required.

Peristalsis may be slowed to permit more water absorption (eg using loperamide) if the cause is dietary – or basically not infectious. We avoid slowing motility with infectious agents as we want to get rid of the infectious agent.

Isolate any animals with potentially infectious conditions!

Treatment is typically supportive.

- Treat dehydration – oral rehydration mixes can help force fluid back out of the lumen; sicker animals may need intravenous fluids
    - We use salt and glucose to work with the transport mechanisms to encourage movement of water from the bowel back into the bloodstream. These work when the enterocytes are still function (eg with secretory diarrheas). The most common use is in calves with E coli diarrhea.
- Treat electrolyte and acid-base distrubances – most animals become acidotic and need an oral alkalinizing agent (acetate, citrate, bicarbonate) or fluid adjustments
- Restore oncotic pressure – give plasma etc if needed
- Clean and protect the perineum (diaper cream)
- Deworm any likely suspects
- Treat endotoxemia with NSAIDs, polymyxin

B, etc
- Treat pain
- Administer gut protectants (pepto bismol, kaopectate, biosponge)
- Potentially give antibiotics
- Potentially give motility modifiers to allow more absorption; contraindicated if infective agents!
    - Loperamide (opioid type) and buscopan (anticholinergic agent) have been used to slow motility and enhance water absorption. With diarrhea related to organisms, out is generally better than in and we avoid these agents.
- Bland diet (I/D; no grain; etc)
- Provide oncotic pressure
- Ice feet to prevent laminitis in horses

Resources

Integrated function – the intestinal phase – includes crypt cell function and general overview

Secretion in the small intestine- another briefer look at secretory action

Diarrhea – all species overview, WSU

Diarrhea in beef and dairy calves – nice high level overview

Foal diarrhea – another high level overview

Just for fun

Approach to diagnosis and therapy of acute diarrhea in dogs-spans physio to disorders, TVP

Swine diseases – diarrhea is important! By Dr. Zhitnitskiy (UMN)

Chronic diarrhea in dogs, JVIM 2017

Feline diarrhea, Cornell

Puppy diarrhea – AKC overview

Challenge quiz – UGA

43. GI colors as diagnostic tools

Vomitus and poo colors, gastric secretions

The green of herbivores is directly related to their diet. Bile and fat digestion create the normal brown color of dog poo. If the vomitus includes the yellow-green color of bile, it has come from the small intestine. If poo is green or brown, it generally contains bile. Yellow or orange stool may indicate excess bilirubin secretion, limited bile secretion, and/or incomplete fat digestion due to limited bile salts, pancreatic dysfunction or rapid transit times. Pancreatic dysfunction will also lead to greasy poo. Gray greasy poo means no bile has been secreted into the lumen to digest fats (so greasy) or add color (so gray). Mucus-covered poo suggests a prolonged transit time.

While peptobismol and charcoal can cause black stool, red, brown or black vomitus or poo is generally due to blood. Gastrointestinal bleeding is a chief complaint that may represent broad etiologies and affects all mammalian species. For practical purposes lower GI bleeding is defined as bleeding that occurs in the lower GI tract and is not digested. It presents as hematochezia (bright or dark red blood). Infectious disease agents are most common causes of hematochezia. Upper GI bleeding is defined as bleeding that occurs in the stomach or small intestine, leading to digested blood in the vomit (coffee grounds) or feces (melena, black tarry poo). The most common causes of upper GI bleeding is gastric or abomasal ulceration. Occasionally upper GI bleeding is not digested, leading to fresh blood in the vomitus (hematemesis). This is usually from the oral cavity, esophagus or stomach (but without digestion).

Resources

What vomit colors mean – short, sweet, and similar across species!

12 things your stool says about your health – pretty easy to follow and useful for colors

Gastric cells and secretions – shorter but scientific version

Just for fun

What the color of your poop means – more rambling, less content but still good

Blood in dog stool – what to look for – scroll down for differentials

44. Additional resources

Stomach physiology

What the stomach cells do – gastrin, parietal, chief cells, g cells, oh my

Physio of the stomach and gastric juices -advanced info about digestion

Alpha -1 proteinase inhibitor– test explanation

Gastric ulcers

Gastric acid physiology– more anatomy, physio with neurotransmitters, hormones and feedback loops

Aspirin and prostaglandins – more about how the drugs work

Abomasal and third compartment ulcers in ruminants and camelids, VCNA 2018

Abomasal ulcers in cattle– DVM 360, 2008

Abomasal ulcers in calves – Dairyherd, 2018

Gastrointestinal protectants in dogs and cats – ACVIM white paper (consensus statement), JVIM, 2018

Consensus statement on Equine gastric ulcer syndrome – European discussion, JVIM, 2015

Equine gastric ulcer syndrome – AAEP

New thoughts on gastric ulcers in horses – KER, 2016

Sucralfate actions- more than I ever knew

Drugs for peptic ulcers– slideshow about humans; includes drug side effects and drugs we don't currently use

Helicobacter and SA gastric ulcers, JVIM, 2000

45. Practice

Note : The zuku questions are designed for year 4 students. You will need to look stuff up for most of those; don't panic please.

Gastric ulcer trivia quiz

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Challenge Exercises – Diarrhea

General

- Review drugs used to treat diarrhea – how do they work?
- Use the DAMNIT-V scheme to classify causes of diarrhea for your favorite species
- What tests are available for particular diarrhea causing agents affecting your favorite species ?
- Do probiotics work in your favorite species? If so, which ones?
- Create a decision tree to determine cause of diarrhea

- Contrast Campylobacter, Salmonella, Clostridium, E coli and fungal diarrheas
- What dietary deficiencies could be associated with chronic diarrhea?

Dogs

- How is albumin and cobalamin used to give prognoses?
- Is vomiting more likely associated with SI or LI diarrhea? Why?
- What constitutes a bland diet?
- Explain the immunology behind inflammatory bowel disease.
- How can exercise help IBD?

Cats

- What constitutes a bland diet?
- How does hyperthyroidism cause diarrhea?

Horses

- What are the risk factors for Salmonellosis in hospitalized horses?
- How does Biosponge work?
- Horses can develop high levels of blood ammonia with diarrhea. How does this happen?
- Coronavirus issues are expanding in the equine world. What does infection with coronavirus look like?

Cattle

- We worry about hospitalized cattle shedding

diarrhea causing bugs. Which do we worry about?

- Explain BVD-mucosal disease
- Outline calf hood diarrhea diseases by age group
- How do you know which oral rehydration formula to choose? What are alkalinizing agents?
- Which will take longer to recover from? Rotavirus or coronavirus? Why?

Swine

- Outline causes of swine diarrhea by age group
- Which diarrheal diseases also have high death rates?

Exotics

- Guinea pigs are prone to enteritis if fed the wrong diet. What is the correct diet and what mechanism is related to the inappropriate diet?

46. Practice

Try out your knowledge! To find more resources to help you with these, try the first tab of this section. Do NOT try to answer them all but pick 1-3 to reinforce the lessons and help you consolidate material in your brain.

Challenge questions- research and explain a few of these to a friend or relation to boost your understanding and retention

All species

- Would or could opioid induced constipation occur in animals?
- How might these drugs help with motility dysfunction?
    - metoclopramide
    - cisapride
    - erythromycin
    - bethanechol
    - tegaserod
    - lidocaine
- How does motility differ across 3 different species?
- Why is stool water content different across species?
- What would be the GI effects related to botulism?
- What would be the GI effects from repeated use of a_2 adrenergic agonists?

- Create your own question related to motility and intestinal activity
- Find a veterinary article about motility disorders or diagnostics and explain to a friend

Small animal

- Why can dogs eat rotten meat and not get sick?
- Explain to a (nonveterinary) relative how edrophonium helps diagnose dogs with myasthenia gravis.
    - Case study : myasthenia gravis in a dog
- Why is poop generally brown and what do other colors mean?
- Explain the findings in cats with dysautonomia
- Why does eating + exercise lead to gastric dilatation volvulus in some dogs?
- What does a GDV look like on radiographs?
- What gastric dysfunction is seen after GDV?
- How does hyperthyroidism affect the stomach in cats?
- Why is diarrhea a risk factor for intusussceptions? Tumors?
- What causes megaesophagus? what animals are prone to it?
- What causes megaocolon? what animals are prone to it?
- What would cause pyloric outflow obstruction and how would it be diagnosed?
- What is a “gravel” sign on radiographs?
- You remove the colon. What will the poo look like?

- Explain gastroesophageal reflux and duodenogastric reflux

Exotics

- What GI track changes would be needed to be a beaver?
    - Beaver dissection
- What happens in the GI track with hibernation?
- What is GI stasis in rabbits and how is it managed?
- What regulates changes in GI motility in cold-blooded animals ?
- What adaptations are present in camels to allow them to live in arid environments?

Equine

- Why would horses being treated for eye disease (eg corneal ulcers) get cecal impactions?
- Horses being treated for orthopedic injuries are more likely to get pelvic flexure impactions. Why might that be?
- What is equine grass sickness and how do affected horses present?
- Why do horses get large colon displacements?
- What is postoperative ileus and what are the main risk factors?
- What causes gastric impactions and how are they treated?
- Why would we give oxytocin to a choked horse? buscopan?
- You remove the colon. What will the poo look like?

Ruminants

- After abomasal volvulus, cows can get abomasal atony. What factors might be involved?
- Why is diarrhea a risk factor for intusussceptions? Tumors?
- Why do cattle get displaced abomasums?
- What is abomasal emptying defect in sheep?
- Why is stool water content different between deer and cattle?
- What would cause pyloric outflow obstruction and how would it be diagnosed?
- How would milk fever (hypocalcemia) affect the GI tract?
- How does normal reticuloruminal activity move ingesta?
- General:

    Explain the actions of drugs used for gastric ulcers. Are there complications associated with their use?

    - peptobismol
    - sucralfate
    - famotidine, ranitidine, cimetidine
    - omeprazole, pantoprazole
    - misoprostol

    What are other side effects of NSAIDs?

    Create a diagnostic decision tree based on colors of vomitus or poo for your favorite species

    Ruminants

Besides NSAIDs, what are other causes of abomasal ulcers in cattle?

How are bleeding ulcers treated in cattle? perforating ulcers?

What clinical signs are associated?

Instead of the vomitus, we can check rumen fluid in cattle. How is rumen fluid analyzed?

Horses

What are risk factors for EGUS in horses?

Explain the difference between glandular and non-glandular ulcers

What are common preventatives for gastric ulcers in horses?

What clinical signs are associated?

Why do we not see steatorrhea in horses?

Small animal

Besides NSAIDs, what are other causes of ulcers in dogs? cats?

What is the role of Helicobacter in small animal gastric ulcers?

Explain the relation of gastrinomas and mast cell tumors with gastric ulcers.

How do bile salts help to digest fats?

What are the clinical signs associated with steatorrhea?

Camelids

What are signs of C3 ulcers in camelids?

How are ulcers treated? Do oral medications work?

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Play along with the Brainscoop podcast – what's the function?

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Practice scat matching for your wildlife hikes! Purely for fun. Not on a test.

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PART VI

FERMENTATION AND LOWER GI DISORDERS

Bloat, flatulence, distension = Large intestine or lower GI

Obstruction of a fermentation site will lead to bloat

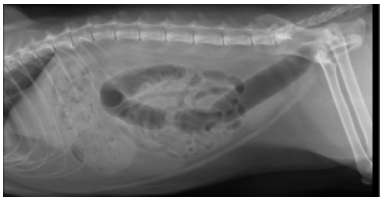
47. Bloat and flatulence

Gas in the GI tract

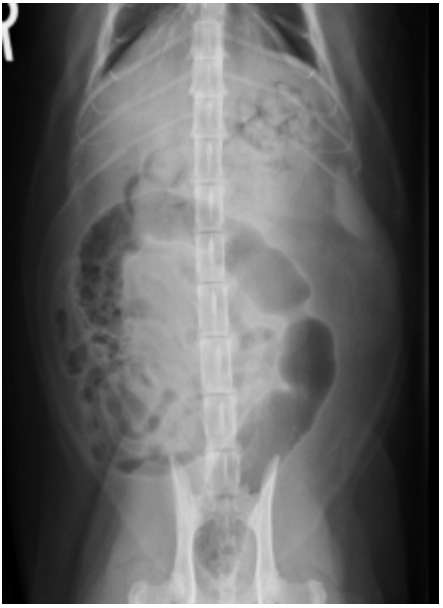
Gas in the Gi tract may be due to swallowed air but is mostly due to bacterial production of gas as they metabolize stuff. Gas is normally released as burps and farts. Bloat is generally due to gaseous distension.

Simple stomached animals (dogs, cats)

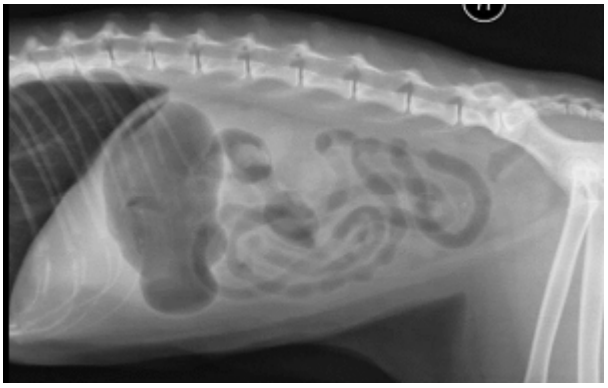
Dogs and cats do have microflora. The amount of microflora is highest in the distal parts of the GI tract (colon). Some gas is usually evident in the colon. Radiographically we identify colonic gas by the "?" appearance and the path to the anus.



LI gas since can trace to the rectum



Some gas can be seen in the stomach, as well. It is abnormal to NOT see some gas in the stomach:



normal stomach distension

Excessive gas in the stomach of a dog often indicates gastric dilation or volvulus, a life threatening condition. in these animals, the

normal gas is trapped, creating severe pain and cardiovascular compromise. These animals need emergency care.

Gastric dilatation and volvulus in dogs, abomasal displacements/torsions in cattle, and large colon displacements/torsions in horses all bear some similarities in terms of

- who gets them – bigger or deep chested animals
- clinical signs – distension, "pings", respiratory and cardiovascular impairment
- pathophysiology – blockage of gas relief
- treatment options – decompression and tacking procedures (pexies)

Fermenters

Fermenters tend to have more bloat problems since they have more microbial activity. Foregut fermenters need to eructate or burp to remove the gas. Hindgut fermenters do more natural farting. Anything that increases the gas production or prevents it expulsion can be very dangerous to the animal involved.

Increased gas production happens with happy microbes : change in diet, change in motility, change in types of microbes

prevention of gas release occurs with physical or functional

obstructions : blockage of the GI tract (esophagus for foregut fermenters, colon for hindgut fermenters), nerve or muscle dysfunction.

Examples of microbial dysfunction

RUMEN BLOAT

Ruminants can develop two types of bloat. *Frothy* and *free gas* bloat.

Frothy bloat develops when animals eat too much clover or alfalfa. The result of digestion is a stable froth that can't be eructated. Treatment involves giving an agent that can break down the froth. This can be problematic if the whole herd if affected. Prevention is better.

Free gas bloat develops when

1. Microbes are happy – change in diet, change in motility, change in types of microbes
2. Muscle dysfunction – hypocalcemia or sedation leads to changes in eructation or motility
3. Nerve damage – changes in vagal function from pneumonia or traumatic reticuloperitonitis lead to loss of eructation or motility
4. Obstruction – esophageal obstruction prevents eructation, a plastic bag blocks omasal outflow

Free gas bloat is treated with passage of a stomach tube, trocharization and/or rumenostomy.

As a juvenile switches from milk to other diets, the microbial environment becomes more important. Often there is a slight mismatch between the microbiome maturity and the diet. In calves, this can lead to free gas bloat. This can be a recurrent issue,

necessitating the placement of a "relief valve", ideally a "rumenostomy" allows gas to escape when needed, at least until the rumen flora mature.

If unrelieved, bloat can compress the venous return to the heart and prevent lung expansion. Both are bad.

FLATULENCE

Flatulence is normal and generally a good thing. Increased flatulence may be associated with changes in diet or exercise. If truly bothersome, pets can be put on charcoal, yucca or zinc acetate.

Prebiotics and probiotics for pets are becoming very popular but healthy animals probably don't benefit much. Prebiotics are feed compounds that are generally believed to be good sources of food for the bacteria. Probiotics are replacement bacteria.

Supplements may be good when an animal has been off feed or on antibiotics.

48. Additional resources

Useful

Why we fart – beer analogies, Khan academy

Bloat in dogs – video for clients

Diagnostics on a dog with bloat– real case

Microbes in the ruminant GI tract, UMN extension- nice overview of roles

Advanced Physio

Lactose intolerance, video- same as before; gut flora ~3:30

Physio of rumen acidosis – nice review of digestion and intro to feeding dairy cattle for milk production

Thiamine deficiency – cool cross species review

Just for fun

Malone's ebook chapter of abomasal displacement – comes with videos and more on the surgeries

Skunk dissection, The brain scoop on scent glands – apparently anal glands with directional nipples!

Why do we fart more on planes? DNews video just for fun party conversation

Ruminal tympany– covers both types of bloat and their pathophysiology

How to properly drench a cow– to avoid drenching the lungs

Rumen function – fistulated cow and microbes

Rumen fluid assessment – very thorough explanation

Harnessing methane from cows for energy

Polioencephalomalacia in goats, MSU extension

B vitamins and horse nutrition, Kentucky Equine Research

Displaced abomasum, UMO extension

DA surgery in cows, Manitoba – start page 19

Colic and colon torsion in the mare, Rossdales equine surgery

Colic and what it means for your horse, The Horse

49. Practice

Challenge Exercises

General

- How is Parkinson's related to your gut bacteria?
- Do prebiotics and/or probiotics work? Are some better than others? What is the difference?
- What is a fecal transplant? Do we do those in vet med?

Dogs

- Compare canine gastric bloat and torsion to abomasal displacements and volvulus- clinical signs, pathology, treatment, risk factors
- Some of the more common flatulence remedies include chicory, Fortiflora and emollients. How do these work?

Horses

- Why do people feed horses B vitamins even if they have healthy gut biomes?
- Compare large colon volvulus with abomasal displacements -clinical signs, pathology, treatment, risk factors

Cattle

- Compare canine gastric bloat and torsion to abomasal displacements and volvulus- clinical signs, pathology, treatment, risk factors
- Why are cattle the most susceptible to nitrate containing plants?

50. LI Obstruction

Etiology

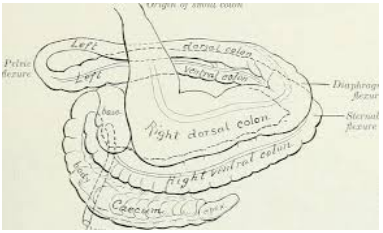
Similar to the small intestine, the large intestine can be functionally or physically obstructed. When the large intestine is obstructed, gas and feed materal are backed up. Gas buildup would indicate a complete obstruction (physical or functional). Obstruction leads to decreased poo output, abdominal distension and discomfort. Mild dehydration can occur along with mild pain signs.

Functional obstruction is due to dysfunction of nerves and/or muscles. Nerves may be local (enteric nervous system) or the autonomic nervous system. Functional obstruction is "ileus" and can be feces, gas or feces + gas buildup. Causes include increased sympathetic tone, decreased motility (eg bed rest, drugs, neuronal changes), and surgery/anesthesia. Horses get "gas colic" with tremendous buildup of gas in the large intestine but without any physical obstruction. The gas distension sets up a pain response which increases sympathetic tone and makes things worse. Cats get megacolon – severe impactions that just sit there.

Physical obstruction can be luminal or extraluminal. Luminal obstructions are usually feed- dried out poo- but can be enteroliths, foreign objects, hair balls, neoplasia etc. Extraluminal obstruction can occur due to twists or displacements (kinks), tumors, adhesions, etc.

In horses, feed impactions are very common. These usually occur at the pelvic flexure – part of the large colon that makes a hairpin turn and narrows. Feed impactions occur due to poor mastication, poorly digestible feedstuffs, decreased water intake, decreased exercise, changes in routine and recent illness (among lots of other things). If the total body water content becomes too low (dehydration), more water is absorbed from the colon, making the

stool even drier. Stool that is retained in the colon will also become dry. Astute animal owners can detect changes in their pet through changes in the stool size and consistency.



The horse large colon is expanded for cellulose digestion and then folded back up to fit inside the peritoneal cavityHorses can also develop enteroliths – stones that form around a particle or foreign object. These are more common on the west coast (something in the water supply) but are not uncommon in Arabs in Minnesota. Displacements are another common cause of obstructions – the bowel moves around and kinks off. Again, this is a common problem in horses since their guts are full of gas producing bacteria and are not tacked down.

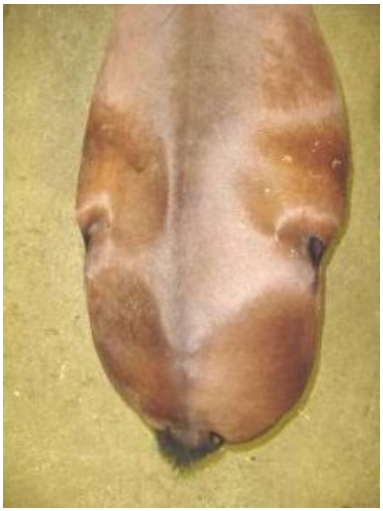
In dogs and cats, foreign bodies are very common. These usually get stuck more proximally (stomach and SI) but can work their way down if smaller. Small animals don't get as gassy unless suffering chronic complete obstructions. Generally in small animals, constipation is due to physical obstruction (pelvic fracture, foreign body, etc), functional obstruction (dysmotility due to chronic obstruction, nerve degeneration, metabolic diseases etc), or neoplasia. Constipation is relatively rare in dogs. Cats can develop idiopathic megacolon. Chronic constipation may result in dilation and dysfunction.

Cattle tend to develop functional obstructions after calving due to low calcium levels (all of the calcium is going into milk production). This leads to ileus and often cecal dilation.

Obstruction in the hindgut leads to impacted dry feces and to gas buildup proximal to the obstruction. Animals may become so gassy that they appear distended. When the obstruction is passed, the manure may be dry and mucous covered due to its longer time in the colon.

Diagnostics

Diagnostics depend a bit on the size of the animal. In all cases, abdominal contour can be useful as can the level of pain (generally mild) and dehydration (mild). Unless an intestinal twist is causing the obstruction, these animals are not usually as sick as those with upper GI obstructions. In fermenters such as cattle and horses, functional or mechanical obstruction distally results in bloat



bloated horse (paralumbar fossa are distended)

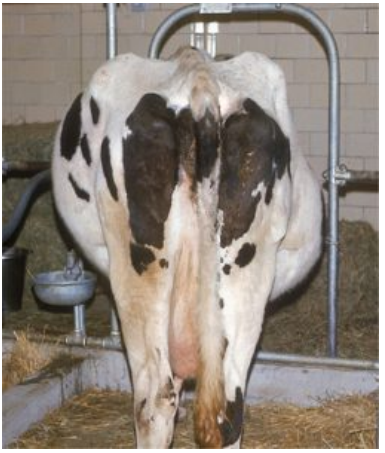
mucus covered manure

Small animals – abdominal distension, abdominal palpation and radiographs are most commonly used. Poo may be small and dry. Limited vomiting. Radiographic changes are often colonic distension with feed material. The colon can be identified by its extension into the pelvis. Diagnostic evaluation may include digital rectal examinations, other imaging, and laboratory workup to identify inflammatory, metabolic or endocrine disorders.

Large animals – abdominal distension, changes on rectal palpation. Poo may be small and dry. Limited reflux. Response to therapy is also useful. We know most cases of equine colic are gas (functional obstruction) and pelvic flexure impactions (feed impactions) so

often treat for those and only explore additional diagnostics if the animal does not respond.

Most animals with complete obstruction develop bilateral abdominal distension. Due to the large rumen, cattle can have unilateral distension and we use the location to help identify what is likely distended.



Papple shaped cow – distended on upper left and lower right

	Upper GI	Lower GI
Reflux/ vomiting	often	not common
Abdominal contour	normal	bloated
Feces	scant	dry, small
Dehydration	moderate to severe dehydration	normal to slight dehydration
Electrolyte issues	low chloride, acid-base issues	normal

Therapy

Animals that are not defecating frequently enough may be treated conservatively at home, medically in the clinic or with surgery. Being able to localize the lesion to the small or large intestine will enhance decision making, as animals with small intestinal obstruction will tend to be sicker and less likely to respond to at home treatment. This is related to the large volume of secretions and electrolyte movement in that section.

For mild impactions, overhydration via enteral fluids (through the GI tract, not intravenously), exercise, analgesics and laxatives are often enough to get things moving.

Buscopan is used in horses for gas colic. This drug actually slows motility (anticholinergic) but that can help with gut spasms. Removing the gut spasm decreases pain and decreases the flight response, leading to an eventual improvement in gut activity. Repeated doses would be bad though – leading to primarily decreased gut function.

Gastric distension can be used to stimulate the gastrocolic reflex and empty the colon.

Laxatives are frequently given to cats and horses to help dry feed material and hairballs slide through. Various types of laxatives can be used. Insoluble fiber is useful in all species to help peristalsis and maintain good fecal water levels.

Lubricants	Softeners	Bulk formers	Stimulants
Mineral oil	Epsom salt	Wheat bran	Bisacodyl
		Metamucil	DSS

51. Colitis / LI diarrhea

Etiology

Many cases of diarrhea are due to infectious agents – bacteria, viruses and parasites. These alter the water secretion and/or water absorption capabilities. In the SI we have three types : secretory, malabsorptive and osmotic. As the LI doesn't have secretory or absorptive capabilities, primary colitis cases are less common and are generally due to osmotic forces. Most times we have both – enterocolitis – due to infectious agents that don't stay in one area of the gut.

Osmotic diarrhea

Example – lactose intolerance. When food stuffs cannot be digested, they stay in the lumen, again creating a concentration gradient. Many of these come with bloating as the food stuff may actually be digestible by the colonic bacteria.

Diagnosis

Small intestinal diarrhea and large intestinal diarrhea do look differently primarily due to the differences in physiology and digestion.

Small intestinal diarrhea	Large intestinal diarrhea
Large volume	Small volume
Normal to slightly increased frequency	Increased frequency
Weight loss	Mucus
Melena	Hematochezia
Flatulence	Tenesmus
Steattorhea	Urgency

Due to the extensive colon and opportunity for water absorption, we often don't even see diarrhea with SI enteritis in horses – all the fluid is resorbed. Most diarrhea in horses will be colitis or enterocolitis.

Therapy

Therapy is generally supportive – fluids and good nursing care.

Therapy plans

- Fluids for dehydration
- Correct electrolyte and acid- base abnormalities
- Bland diet (I/D; no grain; etc)
- Provide oncotic pressure
- Deworm most everyone
- Antibiotics for some cases
- NSAIDs or other pain relief
- Bind endotoxin
- Protect gut
- Ice feet to prevent laminitis in horses

Supplemental resources

Lactose intolerance, humans but generally applies

An overview of calf diarrhea – J Vet Sci, 2014 – much more detailed

Acute colitis in adult horses – Vet Quart 1997, detailed pathophys

Diarrhea pathophysiology in peds – similar categories, slightly different approach

52. Additional Resources

Useful

Bowel obstruction, causes and pathophysiology– really nice overview; yes human but it all is relevant

Equine obstructions– straight forward overview of obstructions and torsions (and cool animations)

Treating mild equine colic -short and sweet, horse owner perspective but also includes neuroanatomy

The equine digestive system- a food factory – an early look at digestion with a motility lens

Digestive track comparison – comparative function and adaptations in one page

Advanced

Comparison mammalian GI tracts– slide show, includes exotics

Constipation and diarrhea – water movement and peristalsis – deeper look at osmosis and water regulation

Small bowel obstruction – in people and includes complications

Normal radiographic anatomy of the canine abdomen

Just for fun

Small bowel ischemia and infarcts – for those of you that like equine colic topics

Irritable bowel syndrome – probably in our vet patients too

Hirschprungs disease – compare to lethal white foals and congenital aganglionosis in SA patients

Ultrasound of a horse with right dorsal displacement– mostly still shots but some live footage too

Coastal hay colic in horses – if you are going to work in the SE USA

Colic in broodmares – more cool animations

Constipation and constipation management – human diets

53. Practice

Exercises

General

- Explain how common laxatives work
    - Mineral oil
    - Dioctyl sodium sulfosuccinate (DSS)
    - Magnesium sulfate (epsom salts)
    - Wheat bran
    - Psyllium (metamucil)
    - Bisacodyl
    - Petrolatum
    - Ranitidine
    - Lactulose
    - Bisacodyl
- Why do all species get constipated with too little exercise?
- How do opioids cause constipation?
- What electrolytes are important for normal smooth muscle (intestinal) activity?
- Create a mnemonic to remember basic causes of impaction/constipation in your favorite species
- Practice using the DAMN-IT scheme to review potential causes of impactions/constipation
- What are 8 F words that would be differentials for a

distended abdomen?

Avian

- How does egg binding compare to mammalian impactions? How is it treated?
- How do crop impactions compare?

Ruminants

- What is abomasal bloat? how does it present and what causes it?
- What are the four types of vagal indigestion in cattle?
- What are potential causes of bovine bloat contours : apple, papple – pear on left, papple – pear on right, and pear?
- What are risk factors for left abomasal displacements? right? are these partial or complete obstructions?
- Why would rapid changes in diet cause DAs?
- How does abomasal volvulus compare to abomasal displacement? duodenal obstruction?
- Why do some young cattle develop recurrent bloat?
- What is frothy bloat and why does it develop? how is that different from free gas bloat?

Equine

- What are common causes of impactions in horses?
- What are common causes of gas colics in horses?
- How are impactions treated in the field?
- How does a dorsal displacement compare to a colon torsion in terms of clinical signs?

- How does an intestinal herniation compare to a strangulating lipoma?
- Proximal enteritis is a form of functional obstruction. How does it compare to a physical obstruction?
- Oral fluids work better than IV fluids for impactions. Explain the path of fluid via both routes.
- What causes gastric impactions in horses? Gastric rupture?
- Miniature horses are prone to small colon impactions? why might that be so?
- What are enteroliths and why do they develop?

Small animal

- What are the risk factors and signs associated with feline megacolon?
- Explain the terms constipation, obstipation and megacolon
- What treatments are used for feline megacolon?
- How common is constipation in dogs? why are dogs so different from other species?
- What causes GDV in dogs?

Camelids

- What are phytobezoars and what signs are associated?

Exotics

- What is rabbit bloat? risk factors? treatment?

Note : The zuku and vetprep questions are designed for year 4

students. You will need to look stuff up for most of those; don't panic please.

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PART VII

DIGESTION AND ABSORPTION ISSUES

Weight loss and vitamin deficiencies

54. Weight loss

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Weight loss occurs for five main reasons:

1. Insufficient calories
    - Lack of intake
    - Excessive utilization
2. Loss of protein or nutrients

- Skin
- Kidneys
- GI Tract

3. Maldigestion
4. Malabsorption
5. Liver disease

Weight loss is often associated with hypoalbuminemia and potentially hypoproteinemia.

Insufficient calories

Insufficient calories can be due to insufficient intake (agroceriosis) or less intake than calorie usage. Increased calories are required with exercise, pregnancy, lactation, hyperthyroidism, parasitism, neoplasia and chronic illnesses.

Protein loss

Loss of proteins can occur through the skin (typically with burn injuries), the kidneys (proteinuria), and the gut. Gut loss maybe associated with ulcers and erosions, inflammatory bowel disease, tumors, crypt cell proliferation, increased lymphatic pressure, and tight junction disruption.

Maldigestion

Maldigestion is associated with liver or pancreatic issues in monogastrics. If the liver doesn't produce or recirculate adequate bile, fat digestion is impaired. If the pancreas doesn't produce functional enzymes, digestion of most dietary substances is impaired. In herbivores, if the microbes are not functional, cellulose digestion is impaired.

Malabsorption

Malabsorption is related to abnormalities in the intestinal wall. This may be due to diffusion or transport issues. the villi must be normal for good absorption. Malabsorption will occur if villi are lost, shortened or thickened. if diffusion gradients are not maintained, absorption will also be impaired. Glucose and protein digestion require active transport and short diffusion distances. Fat absorption is primarily diffusion.

Evaluation

Nutrient analysis can help identify if caloric intake is adequate. If the diet appears sufficient, laboratory testing should include a complete blood count, serum profile, urinalysis and total throxine (T4, cats). Additional testing may include fecal examinations, thoracic or abdominal radiographs, rectal biopsies, and abdominal ultrasounds.

Malabsorption (lack of protein absorption) can be difficult to distinguish from protein loss, particularly protein-losing enteropathies. Often both loss and malabsorption coexist. Low

protein can also develop with renal disease, burn wounds, and liver disease.

Alpha-1 proteinase inhibitor to rule-out protein loss

Protein loss can be identified in dogs and cats by measuring alpha-1-proteinase inhibitor. This protein leaks into the intestinal lumen with severe disease but isn't digested so can be measured in the feces.

Malabsorption tests

Intestinal malabsorption can be measured through 1) D-xylose absorption tests (proximal small intestine) or 2) Vitamin B12 (cobalamin) levels (distal small intestine). Low folate levels may also indicate proximal small intestinal disorders, particularly in cats. In large animals, ultrasound is often combined with duodenal or rectal biopsies and/or abdominocentesis. Additional tests tend to be specific for differentials, such as fecal parasite checks, Lawsonia titers, etc.

D-xylose absorption

D-xylose is a plant sugar that isn't normally found in the bloodstream and is readily absorbed. D-xylose is administered orally and then blood levels measured. If blood levels do not rise sufficiently, malabsorption is diagnosed. If weight loss is present and D-xylose levels are normal, the problem is more likely to be

maldigestion (e.g. pancreatic dysfunction). A flattened peak suggests malabsorption while a delayed peak correlates with delayed gastric emptying.

Cobalamin levels

Cobalamin is an essential co-factor in many mammalian enzyme systems. It comes exclusively from bacteria and is important in amino acid metabolism and DNA synthesis. Absorption is highly complex.

Dietary cobalamin is tightly bound to animal-derived dietary proteins. These proteins are partially digested in the stomach and the cobalamin released. It immediately binds to haptocorrin (aka R-protein). In the small intestine, the haptocorrin is digested and the cobalamin freed again. This time it immediately binds intrinsic factor. Intrinsic factor/cobalamin complexes are absorbed in the ileum. As it is absorbed into the epithelial, the intrinsic factor is removed and cobalamin released into the circulation. In the circulation, cobalamin binds transcobalamin II and is stored in the liver and kidney. (This is different than in people where intrinsic factor is released in the stomach).

Cobalamin deficiency in dogs and cats is generally associated with severe intestinal disease and with exocrine pancreatic insufficiency. Almost all cats and most dogs pancreatic insufficiency are cobalamin deficient, likely related to lack of intrinsic factor. Small intestinal disease must be chronic and severe to see cobalamin deficiencies as body stores are large. Hereditary cobalamin deficiency can also occur in some dog breeds.

Maldigestion tests

Maldigestion can be assessed through 1) trypsin-like immunoreactivity, 2) folate levels and 3) cobalamin levels. Additionally, enzyme levels can be measured for gastrin and pancreatic lipase.

Trypsin-like immunoreactivity

TLI is a more accurate measure of pancreatic function than lipase or amylase levels. Species-specific TLI is considered the optimum test for exocrine pancreatic insufficiency (EPI).

Folate levels

Folate can only be absorbed in the proximal small intestine. Many bacteria produce folate but generally only in the large intestine which has no receptors for folate absorption. In cases of pancreatic insufficiency, bacteria can overgrow in the small intestine due to lack of pancreatic enzymes. This can lead to high levels of folate in dogs with EPI. Cats tend to have low folate levels with EPI as they also get intestinal damage that impacts the folate receptors.

Cobalamin levels

Cobalamin insufficiency is more likely to be associated with exocrine pancreatic insufficiency than with malabsorption as described above. However, if TLI is normal, cobalamin and folate abnormalities are more likely to be associated with intestinal

malabsorption. (When you have bacterial overgrowth, they bind the cobalamin receptor and prevent cobalamin uptake into the cell so this also lowers levels.)

Key Takeaways

Nutritional balance	Weight loss
Lack of input	Agroceriosis, lack of essential vitamins or minerals
Excessive output	Lactation, neoplasia, surgery
Loss	Parasites, pregnancy, gastric ulcers, inflammation
Lack of digestion	Indigestible foods, lack of enzymes, lack of healthy microbes
Lack of absorption	Abnormal villi, abnormal motility

Resources

Treatment of obesity in dogs and cats, Today's Veterinary Practice
Hyperthyroidism in cats, Washington State University

55. Weight loss resources

Useful

Pick one:

- Weight loss in dogs, VCA hospitals
- Weight loss in cats, PetMD
- Weight loss in dairy cattle, Dairy Australia
- Weight loss in horses, The Horse
- Weight loss in alpacas, Dr. Videla, UTenn
- Weight loss in sheep and goats, VCNA 1983
- Weight loss in swine, National Hog Farmer

Diagnostic approach to protein losing enteropathies, Ch 73 Equine Medicine & Surgery , 7th edition

Testing for weight loss in dogs, VCA hospitals

Cobalamin, DVM 360

Treatment of obesity in dogs and cats– Todays Veterinary Practice

Advanced

D-xylose testing, 1988 Gastroenterology

Care of dogs with protein-losing enteropathies, DVM 360

Just for fun

Canine lymphangiectasia, Indiana Animal Disease Diagnostic Laboratory

Johnes disease, APHIS

Coccidiosis in new world camelids, DVM 360

Hyperthyroidism in cats, WSU

Thyroid testing, Idexx

Lactose intolerance – human but applies to animals too

Celiac disease – talking about gluten

56. Weight loss exercises

Challenge exercises

General

- How do veterinary therapies for intestinal diseases compare to humans?
- What are the signs of cobalamin deficiency?
- How are serum MMA tests used to evaluate cobalamin deficiencies?
- How and when do we supplement cobalamin?
- Why is calcium low in animals with protein losing enteropathy?

Small animal

- How do diets impact cobalamin levels? folate?
- Explain lymphangiectasia induced weight loss in dogs
- Explain hyperthyroidism induced weight loss in cats
- Explain lymphoma induced weight loss
- How does histoplasmosis lead to weight loss?
- What are the therapies for protein losing enteropathies in small animals?

Ruminants

- How does Johne's disease induce weight loss?

- How do we evaluate microbial function? (sedimentation rate, methylene blue reduction)
- What causes pregnancy toxemia and how is it managed?

Camelids

- How does coccidiosis induce weight loss?

Equine

- What are common causes of weight loss in horses?
- How might abdominal fluid help with diagnosis of weight loss causes?
- How effective are rectal biopsies at diagnosing weight loss causes?
- What is killing Sable Island feral horses?
- How is maldigestion evaluated in horses? foals?

Exotics

- What are common causes of weight loss and how are they diagnosed?

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57. Vitamin deficiencies

THIAMINE DEFICIENCY

Almost all animals are likely to have problems if they have thiamine deficiency. Generally our microbes produce enough; however, in goats, they only produce just enough. So if something goes wrong (too much grain, wrong plants, not enough food), goats are prone to developing thiamine deficiency and polioencephalomalacia. Goat "polio" is a neurological disorder and animals show "star gazing", blindness and gait changes. If caught early enough, goat polio is highly treatable and animals can make a full recovery. Many goats are given supplemental thiamine as a precaution anytime they are sick or anorexic.

58. Practice

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Exercises

Horses

- What can cause thiamine deficiency in horses?

Small ruminants

- Under what conditions are small ruminants most at risk of thiamine deficiency?

Cats

- What does thiamine deficiency look like in cats? What are risk factors for it?

This is where you can add appendices or other back matter.