

3. Gastro-Intestinal Tract, updated 02-12-2018 - pages 27 to 31

The Equine GI Tract and Physiology

- Lips
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- Teeth
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- Esophagus
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- Stomach
 - Simple stomach divided into two regions
 - Cardiac region is lined with a non-glandular, squamous epithelium, similar to the mucosa of the mouth, and esophagus.
 - This is the area where most gastric ulcers occur.
 - This is the location where *Gasterophilus intestinalis* and *G. nasalis* fly larvae emerge and live in the fall and winter, and release to pupate in the spring. These parasites cause gastritis, mucosal damage and perforating ulcers.
 - Glandular fundus, the lower half of the equine stomach is the fundus which contains the secretory gastric mucosa. Gastric ulcers can also occur here
 - Pyloric region
- Small Intestines
- Ileocecal junction

- Cecum
- Cecocolic junction

- Colon
 - Ventral Colon is divided into: several sections
 - Analogous to the Ascending Colon)
 - The Origin of the large colon, starting at the cecocolic junction; high in the right posterior quadrant of the abdomen
 - The right ventral colon descends to the lowest and most anterior location of the abdominal cavity, adjacent to the sternum
 - Divided into three sections
 - Right ventral colon, descends from a location high in the Right
 - Sternal flexure – the location when interference of sand impaction
 - Left ventral colon

 - Pelvic flexure
 - Separates the Ventral Colon from the Dorsal Colon
 - Easily palpated via the rectum
 - The most common location for fibrous impaction in the

 - Dorsal Colon
 - Analogous to the Transverse Colon
 - Divided into three sections
 - Left dorsal colon
 - Diaphragmatic flexure
 - Right dorsal colon

 - Small Colon (also called the Descending Colon)
- Rectum

Ruminant Stomach, GI Tract and Physiology

Four divisions of the ruminant stomach

- Reticulum (1st compartment)

Functions in concert with the rumen in fermentative digestion of fibrous feed components. Sometimes referred to as the "first stomach". The reticulum, along with the rumen are the "proventricular" or cardiac region of the ruminant stomach. The reticulum, rumen, and the omasum are lined with a squamous epithelium, similar to the esophagus and cardiac region of other mammalian species. The main function of the reticulum, similar to the rumen; is bacterial fermentation of roughages feeds.

The reticulum lies directly below the esophageal entry point, and heavy items usually end up in the reticulum,

- Boluses, magnets, telemetry devices, and metallic hardware are usually found in the reticulum.
- Wire, nails and other sharp metallic items are the cause of "hardware disease"

Common Condition

- Hardware Disease - A wire or other sharp metallic items swallowed mistakenly by a cow will usually end up in the in the reticulum were it is able to abrade and irritate the reticular mucosa leading to a traumatic reticulitis.

- Wire may perforate the reticulum during a peristaltic wave and cause peritonitis
- A wire may continue to migrate through the diaphragm; leading to pleuritis and perforation of the pericardial sac causing purulent pericarditis. Pericarditis can then lead to cardiac tamponade and congestive heart failure.

- Clinical Signs and diagnostic tests:

A moderate fever if peritonitis is present, 103.5 to 105 degrees F. The animal may be non-febrile before reticulum perforation occurs. The animal may also be non-febrile late in the disease process

Loss of appetite, weight loss, ruminal ileus upon auscultation, and a gaunt appearance to the abdominal area.

Abdominal pain due to peritonitis causes a reluctance to walk, a reluctance to lay down or rise, and the animal will assume an arched back posture. The animal's posture will not return to a flat backed or lordotic posture when the skin of the back is pinched. The animal will have a positive "Grunt Test" when sudden upward pressure is applied to the sternum.

Peripheral edema, and other signs of congestive heart failure will appear if pericarditis is present

Blood work reveals leukocytosis & elevated fibrinogen if peritonitis is present.

Radiographs to identify metallic objects in the reticulum & pericardial areas

- Treatments:

If a cattleman or veterinarian thinks; maybe it is remotely possible. Na! Well Maybe! The animal could have hardware disease?? : GIVE THE ANIMAL A MAGNET!

If the animal is febrile use antibiotics to treat for peritonitis

Surgery can be used. A Rumenotomy may be performed to remove hardware from the reticulum. Pericardial Marsupialisation can be performed on animals with pericarditis.

- Rumen (2nd compartment)

The largest part of the ruminant stomach

Is actually a large fermentation vat

- Cows do not masticate the feed that they eat very well. The feed is masticated only well enough to form a feed bolus that is swallowed.
- The un-masticated, partially saliva saturated feed is less dense than fully masticated feed that is fully saturated with saliva. The feed bolus that is swallowed will float on the surface of the rumen fluid layer.
- After a ruminant finishes eating, the feed on the surface of the rumen ingesta is regurgitated and re-masticated. This is called "chewing the cud".
- When the cud is swallowed it is saturated with saliva and due to its density it will fall into the lower layers of the reticulum and rumen contents.
- One very important aspect of chewing the cud is the large amount of saliva that is added to the feed. This saliva acts as a buffer to lowering of the pH caused by volatile fatty acid products of fermentation.

Common conditions

- Bloat - Due to the bacterial fermentation that occurs in the ventral sac of the rumen volatile fatty acids and gases are produced by the microbes. The gas accumulates in the dorsal sac of the rumen. On a regular basis the ruminant animal will eructate a portion of the gas that accumulates there.
 - There are three basic types of bloat
 - Esophageal obstruction, Often a feed item such as a turnip or a sugar beet top that gets stuck at any location such as the larynx, the esophagus at the base of the heart or the cardiac orifice of the rumen. Treatment usually involves oral exam and removal of observable obstructions. If the obstructing object was swallowed it can be pushed with a stomach tube into the rumen
 - Frothy Bloat, This form of bloat occurs when an animal eats fresh young legumes that are highly fermentable, low in fiber, high in soluble carbohydrates, and high in protein (18 to 24% protein). The high protein and rapid fermentation cause a rapid production of stabilized gas bubbles, rising from the fermenting fluid layer that form a stable froth in the dorsal sac of the rumen. Cattle are unable to eructate the gas trapped in froth. Regurgitation may sometimes reveal what looks like a large volume of frothy saliva running from the mouth. When passing a stomach tube no gas is released from the rumen, only froth is seen in the tube. Treatment involves administering a defrothing agent that will lower the surface tension of the gas bubbles. FDA approved pharmaceuticals for this purpose are Poloxalene and Simethacone. Mineral oil can be used if it is all that is available.
 - Vagal indigestion and chronic (repeated) bloating occurs occasionally in weanling calves (and older animals less frequently). Vagal indigestion is a condition that occurs secondarily to pleuritis. Pleuritis itself is a secondary condition to bronchopneumonia in cattle. The vagus nerves run down each side of the mediastinum, immediately underneath the visceral pleura. If infection extends into the nerve itself it will affect conduction in both sensory and motor neurons. This may result in decreased peristalsis at any level of the GI tract and it may reduce sensory perception of fullness. Lack of sensory perception causes animals to overeat, covering the esophageal orifice, preventing regurgitation. Lack of motor function may interfere with ruminal - omasal transit and gastric emptying. The pathogenesis is complex and can vary from animal to animal. Treatment simply involves passing a stomach tube and if the animal turns into a chronic bloater, it should be isolated and limit fed an adequate diet.
- Ruminant Acidosis
 - Causes: Overfeeding soluble carbohydrates (grains, potatoes or other high starch crop residues) with inadequate quantity of forage. Feeding the grain

component of a diet separate from and prior to feeding the forage component of the ration. This allows grains to sink in the fluid layer of the rumen to the floor of the reticulum and rumen. This creates local areas in the reticulum and ventral sac of the rumen with extremely low pH under 5.0. A pH <5.5 is considered to be a low grade ruminal acidosis. A pH <5 is severe acidosis even if only locally in the ventral rumen.

- Prevention: decreased carbohydrates in the ration. Replace carbohydrate with fat in high energy requirement situations. Always feed the hay component of the ration prior to feeding grains. This causes an increase in rumen buffering due to salivation stimulated by consumption of forage. Also forage consumption creates a buoyant forage mat for the grain to remain suspended in. Do not feed a component ration. It is always better if it is possible. To feed Total Mixed Ration (TMR) instead. A TMR prevents local areas of low pH.
- Pathogenesis: When the pH is too low in the rumen it will cause a shift in the rumen microbes population to more acidophilic species that may not be as beneficial to the animal. Also during the increase in acid in the gut bacteria will die or sporulate as the bacteria cannot live in their own waste products. When bacteria die they release endotoxins and exotoxins that can cause subclinical disease and poor performance. Frank disease such as "Enterotoxemia" caused by sporulation and lysis of *Clostridium perfringens* organisms is also a possibility.
- Treatment: Prevention is the best treatment, but buffers in the feed such as sodium bicarbonate can be used. Acute acidosis is usually treated with magnesium oxide. In a severe acute case of overeating grain (i.e. She broke into the grain storage room!) a rumenotomy can be performed to manually remove the grain. Intravenous sodium bicarbonate may be needed in patients such as this.

- Omasum (3rd compartment)

Round basketball sized organ

Mucosal folds form interdigitating leaves

- Mucosal leaves fill entire lumen
- Very large mucosal surface area for absorption

Main functions:

- Dehydration of rumen contents
- Absorption of volatile fatty acids (end products of fermentation)

Common condition

- Impaction
 - Undigestible materials such as baler twine can easily cause impaction and obstruction of the omasum
 - Torsion

- Abomasum (4th compartment)

"The true stomach" actually the fundus region of the ruminant stomach

Acid and enzymatic digestion of ingesta

Anatomy is similar to the simple stomach of other species

In addition to digesting consumed feed:

- Bacteria and protozoa from rumen fermentation are digested, greatly increasing the protein and nutrient quality of the ingesta
- Ruminal microorganisms can convert ammonia, urea and other nitrogenous compounds into protein

Common condition: Displaced Abomasum (DA) there are two different types with many variations regarding etiology and pathogenesis.

Displaced abomasum is not a primary condition. It

- Left displaced abomasum (LDA) is a simple displacement without volvulus or torsion.

- Right displaced abomasum (RDA) is an actual 180 degree counterclockwise (viewed from behind) abomasal torsion. It has been reported that the torsion can occur proximal to the omasum abomasum paired unit, or commonly the torsion occurs at the omasum - abomasum junction. It results in stasis of the abomasal contents an accumulation of chloride in the abomasal lumen.
- The etiology of DA, regardless of the side of the displacement, is that the displacement is secondary to any condition that causes the animal to be "off feed", (anorexic), have GI ileus, reduced ingesta volume and relative increased gas volume in the rumen. This allows ruminal gas to pass into the abomasum causing buoyancy of both the rumen and abomasum. The decreased rumen size, buoyancy of the stomach compartments, and a tendency for sick cows to lay fully recumbent; allows movement of the abomasum away from the ventral, right, paramedian, normal position in the abdomen.
- The main predisposing factors for a DA include: hypocalcemia, mastitis, metritis, ketosis & fatty liver syndrome, ruminal acidosis, laminitis and other lameness. These conditions have their highest incidence in the immediate post-partum period, therefore the highest incidence of DA is also post-partum.
- Initial treatment is directed at the initial predisposing factors. Simultaneous or subsequent abomasal replacement surgery with an abomasopexy, a pyloropexy or an omentopexy must be performed to secure the abomasum in its proper position. An RDA is an emergency surgery that must be done on a Stat. basis due to the hypochloremia and metabolic alkalosis that occurs in conjunction with the abomasal torsion. An LDA surgery should be scheduled within one day or two. Timely surgery will prevent adhesion formation which may interfere with successful LDA surgery if it is delayed for too long.

The Ruminant Hind Gut (Intestines)

- Small Intestines
- Cecum
- Large (or Spiral) Colon
 - Centripetal Spiral Colon
 - Centrifugal Spiral Colon
- Descending Colon
- Rectum

Equine Digestive Disturbances

Primary categories

- Gastritis
 - Parasitic gastritis
 - Bot fly larva
 - Stomach worms
 - Both parasites perforate and cause lesions to gastric mucosa
 - Gastric ulcers
 - Primarily a disease of young horses
 - Decreased appetite and poor performance are main symptoms
 - Colic and other signs are rarely seen
 - Up to 50% of foals may be afflicted
 - Gastric perforation and hemorrhage are occasional life-threatening sequela
 - Helicobacter infection has not been proven but may be a factor

Proximal Enteritis

- Extension of a small intestinal disease discusses below
- Enteritis (small intestinal)
 - Small intestinal enteritis frequently causes diarrhea in neonatal foals.
 - Adults will seldom display diarrhea due to the fluid absorbing capacity of the large colon.
 - Adults are more likely to show colic like signs as main sign of small intestinal enteritis.