



Common Nutrition-Related Gastrointestinal Diseases of Ruminants

by Joanne McCrea

Ruminants have a complex gastrointestinal system that turns fibrous materials into valuable energy and protein sources such as milk and meat. Maintaining a healthy digestive system and preventing nutrition-related disorders enhances an animal's overall health and performance. This publication highlights some common gastrointestinal conditions that may be related to diet. Understanding the gastrointestinal anatomy and physiology of ruminants may be helpful. Refer to the "Gastrointestinal Anatomy and Physiology of Ruminants" publication as a resource. **Note that this article doesn't cover all gastrointestinal issues and is not a substitute for veterinary care.**

ESOPHAGEAL OBSTRUCTION (CHOKER)

Ruminants can experience a blockage in their esophagus — the tube that connects the mouth to the stomach. This can occur if there are external masses or swelling that compress the esophagus, or if foreign bodies become lodged inside. Poorly chopped beet roots, for example, can block the esophagus.

Normally, gas produced during digestion moves into a part of the reticulum called the cardia, which is the area where the esophagus enters the reticulo-rumen. The gas is released through the esophagus in a process called eructation or belching. If this gas can't escape through the esophagus, it will be trapped within the reticulo-rumen and cause a condition known as rumen bloat. Bloat can become so severe that the animal's enlarged abdomen compresses the diaphragm, lungs and vasculature (blood vessels) to the point that the animal cannot breathe.

Signs that a ruminant is suffering from an esophageal obstruction include food or saliva

coming from their mouth or nostrils, muscle spasms or swelling along the neck, coughing and abdominal distention (an enlarged belly). Managing these cases depends on the cause of the obstruction, the animal's health and the available resources. Sometimes, using an orogastric tube (hose-like tubing put into the mouth and esophagus) to break up the blockage will not be effective, and surgery may be necessary.

TRAUMATIC RETICULOPERICARDITIS OR RETICULOPERITONITIS (HARDWARE DISEASE)

Ruminants can accidentally ingest metal objects, such as nails or pieces of wire, which can settle in the reticulum. If these sharp objects puncture the reticulum wall, they can create a pathway for bacteria from the gastrointestinal tract to nearby areas, such as the pericardium (the lining around the heart) or the peritoneal cavity (the abdominal cavity), causing infection and inflammation known as reticulopericarditis and reticuloperitonitis, respectively.

Animals may show signs of infection, such as fever, decreased appetite, weight loss and reduced milk or meat production. Signs of pain in the chest or abdomen include a lowered head, wide-set elbows or hesitancy to move around. Due to changes within the reticulo-rumen, they might also produce less manure and have a distended abdomen. Additional signs that may be seen with reticulopericarditis include swelling around the jugular vein, jaw and brisket.

HYPOMAGNESEMIA (GRASS TETANY)

Hypomagnesemia, also known as grass tetany, is a condition that occurs when animals have low levels

of magnesium in their blood. There are various causes, but it is most commonly seen when lactating animals graze on new, lush green pastures. Fast-growing, young grasses often have low magnesium and high potassium.

Magnesium is an essential mineral for normal nerve signals and muscle function. Animals with hypomagnesemia may show signs such as increased muscle stiffness, nervousness, sensitivity to sounds, excessive excitement, aggressive behavior, muscle twitching and recumbency (lying down and being unable to rise) with a stiff, rigid posture. This condition can worsen rapidly and may become life-threatening. Early treatment with magnesium-calcium solutions, given through an intravenous (IV) infusion, along with additional magnesium supplements, can help. To prevent this issue, provide the animal with an adequate amount of magnesium in its diet.

Pasture-related factors that may increase the risk for grazing animals to develop hypomagnesemia:

- Young grass under 6 inches tall
- Grass growing in soils high in nitrogen, potash (a potassium-rich salt) or calcium, as these nutrients can interfere with an animal's ability to absorb magnesium. Fertilizers will increase the nitrogen and potash content of the soil, while lime treatment will increase the calcium content of the soil.
- Grass that has been grown with soil or water that has high nitrate or sulfate levels
- Grass mixes with certain cereal grains that contain compounds that may be involved in forming tricarballic acid, which can bind magnesium and reduce its absorption.

Certain animal characteristics can also raise the risk of hypomagnesemia:

- Lactating animals need more magnesium than other animals.
- Older animals are not able to access their mineral stores of magnesium as well as younger animals.

- Excess calcium in the diet can decrease magnesium absorption.
- Excess potassium in the diet can decrease magnesium absorption in the rumen.
- Excess sodium in the diet can lead to increased magnesium loss through urine.

BLOAT (RUMINAL TYMPANY)

Bloat happens when the rumen becomes overly full of gas. There are two main types of bloat: free gas bloat, where a large pocket of gas forms, and frothy bloat, where gas is trapped in a thick foam and can't be released by belching.

Bloat can happen if there is abnormal rumen motility or a blockage in the esophagus. To understand this issue, it's important to know how the rumen typically functions. Rumen motility includes primary contractions that mix the rumen contents, and secondary contractions that help the animal release gas through belching. During these secondary contractions, the cardia (the area where the esophagus enters the reticulo-rumen) is cleared of food to allow gas to move from the rumen to the esophagus and be released. Rumen motility is controlled by the vagus nerve, which carries signals between the brain, heart and digestive system. However, there are other sensory nerve cells that will affect rumen motility. For example, if nerves in the rumen wall detect distention (overfilling and stretching) or a decrease in the rumen pH from more acid production in the rumen, the contractions will decrease or stop.

Free gas bloat

Free gas bloat happens when gas builds up due to a blockage in the esophagus or if rumen contractions fail to move gas near the opening of the esophagus and into the esophagus. Rumen contractions can be inhibited by injury of the vagus nerve, increased rumen distention (overfilling and stretching) or increased acid within the rumen. If food cannot exit the rumen due to a blockage anywhere between the rumen and the small intestine, the rumen may become distended. Two places where blockages occur are the omasal orifice (the passageway between the reticulo-rumen and omasum) and the

pylorus (the passageway between the abomasum and the small intestine). To treat free gas bloat, it's necessary to identify the underlying cause.

There are three ways to alleviate free gas bloat. The first, and least invasive, method is to insert a flexible plastic tube called an orogastric tube down the esophagus to help release gas from the stomach. This may not work if there's a blockage in the esophagus. Another option, called rumen trocarization, involves inserting a special needle (a trocar) into the rumen to allow the gas to escape. Lastly, surgery may be necessary to open the rumen and remove the contents. This surgery is called a rumenotomy.

Frothy bloat

Frothy bloat occurs when gas bubbles become trapped within froth or foamy rumen contents. Normally, the gas is in a large pocket and can be eructated, but when it is trapped in this foam, it causes rapid rumen overfilling and distention. Frothy bloat can happen when animals eat a diet high in protein or when rumen microbes produce a mucoprotein slime that traps gas. Legumes such as alfalfa can have a high protein content and may cause frothy bloat when fed. The acidic rumen environment seen in animals that are being fed high-grain diets may cause more mucoprotein slime production and frothy bloat. Frothy bloat can quickly become life-threatening.

Treating frothy bloat depends on the animal's condition. If the animal is stable, a surfactant can be given through an oral gastric tube. A surfactant is a substance that works by reducing the surface tension of the foam, breaking it down and allowing the gas to be released. However, if the animal is in critical condition and struggling to breathe, surgery may be necessary to remove the rumen's contents immediately. Unlike free gas bloat, frothy bloat cannot be treated by rumen trocarization.

RUMEN IMPACTION (HAY BELLY)

Rumen impaction, often called hay belly, occurs when animals eat poor-quality forage, like overmature hay or straw. This can limit the nutrients available for rumen microbes, slowing their activity. When the rumen microbes are not active, fibrous food isn't broken down, production of

volatile fatty acids slows, and the energy the volatile fatty acids provide isn't available to the animal. This will also slow the rumen's mixing contractions, and the fibrous food will accumulate and sink to the bottom of the reticulo-rumen. The food material will not be digested and will build up in the rumen, causing rumen distention. The entire left and lower right sides of the animal's belly may be notably enlarged.

Signs of rumen impaction include decreased appetite, less manure output and dry feces with large pieces of undigested plant material. Due to malnourishment, animals may also suffer from weight loss, a poor coat condition and decreased milk production. In some cases, a blockage in the omasum or the presence of a foreign material that was eaten, such as a plastic bag, can look similar to this condition.

While some cases of rumen impaction might be treatable without surgery, others may require a veterinarian to remove the impacted material through a surgical procedure called a rumenotomy.

RUMEN ACIDOSIS

- pH = 7.0 is neutral
- pH < 7.0 is acidic
- pH > 7.0 is alkaline or basic
- Normal range for rumen pH is 5.5-7.0
- Normal range of cattle saliva pH is 8.1-8.9

Rumen acidosis is a condition that occurs when the pH of the rumen contents drops below 5.5. This disease is complicated and can lead to several other disease conditions that are briefly described below.

Feeds high in starch and sugar (grains) are a valuable nutrient source and are used to meet the energy demands for late pregnancy and milk production. Grains can also support rapid growth and desired meat marbling in beef cattle.

When ruminants eat grains, the starches and sugars are quickly fermented by the rumen microbes and broken into compounds called volatile fatty acids, a process that makes the rumen more acidic. The rumen acidity is normally buffered

(offset) by the saliva. Saliva contains bicarbonate, a basic compound. When fibrous feed is ingested, it is regurgitated, rechewed and reswallowed. During this process of chewing cud, more salivary bicarbonate is added. Given this, fibrous forages are important for managing the rumen pH. High-grain diets may not always contain enough fibrous forage.

Normal rumen pH typically ranges from 5.5 to 7.0. Different types of microbes in the rumen have varying preferences and tolerances for acidity. For example, microbes that are better at digesting cellulose prefer a pH level between 6.2 and 7.0. Ruminants that eat mainly forage (like hay and grass) usually have a rumen pH between 6.0 and 7.0. Those on a grain-heavy diet typically have a rumen pH between 5.5 and 6.0. After being fed grains, the rumen pH temporarily drops a little bit, but should return to the normal range in a few hours. If the pH drops below 5.5 and stays there for a longer period of time, the rumen microbes will be affected. The altered pH in the rumen can cause some microbes to die and others to thrive. Rumen acidosis occurs when the rumen pH stays at less than 5.5.

The consequences of rumen acidosis can be complex and can lead to several health problems, listed here. The descriptions of these diseases are limited to the context of rumen acidosis.

- **Bloat or ruminal tympany:** A condition where gas is trapped in the rumen, causing rumen distention. Some rumen microbes produce harmful byproducts, including mucoproteins, a type of slime that can lead to frothy bloat. Microbes that produce these mucoproteins favor overly acidic environments in the rumen.
- **Endotoxemia (endotoxins in the bloodstream):** This happens when endotoxins — toxic substances released by dying bacteria — enter the bloodstream, triggering an inflammatory reaction that can be life-threatening. Endotoxic shock may occur and can be fatal.
- **Septicemia (sepsis):** A bacterial infection of the blood. Septicemia triggers an inflammatory response, which can lead to septic shock. Septic shock can be fatal.

- **Metabolic acidosis:** An increased amount of acid in the blood caused by a lack of oxygen in the body tissues. Without oxygen, cells will use anaerobic metabolism, which generates lactic acid.
- **Rumen parakeratosis:** A condition where the papillae, finger-like projections in the rumen lining that are important for absorbing nutrients, thicken and harden in response to injury. The abnormal papillae will not absorb nutrients as well as normal papillae.
- **Liver abscesses (hepatic abscesses):** These abscesses occur when excessive bacteria in the bloodstream overwhelm the liver's ability to filter and remove bacteria.
- **Vena caval thromboembolic disease:** A condition caused when a bacteria-infected blood clot, called a septic thrombus, forms in the vena cava, one of the body's largest veins, which transports blood from the lower body back to the heart. Pieces of these infected clots may break off, spread infection or interfere with blood flow elsewhere. Pneumonia, bleeding from the nose (epistaxis), and respiratory distress can occur when septic thrombi are in the lungs.
- **Laminitis:** Inflammation of the lamina (blood vessels and connective tissue between the hoof wall and foot). This is a complex disease with various causes, including endotoxemia or septicemia.
- **Polioencephalomalacia:** This neurological condition results from a disrupted energy supply to the brain. Some microbes produce harmful byproducts, such as hydrogen sulfide gases and thiaminases (an enzyme that breaks down thiamine or vitamin B1). Rumen acidosis can increase the production of these byproducts. Both too much hydrogen sulfide gas and a deficiency in thiamine can cause polioencephalomalacia. Learn more in the publication "Polioencephalomalacia in Ruminants."

General disease process of rumen acidosis

When ruminants eat grains, a large amount of volatile fatty acids is produced, lowering the rumen pH. A proper balance of fiber in their diet is crucial because it stimulates cud chewing, which adds saliva to the rumen, helping neutralize the acidity produced by volatile fatty acids. Grain-based diets may not always contain enough large fiber particles (roughage or neutral detergent fiber) to stimulate cud chewing.

Certain bacteria thrive in acidic rumen environments, such as *Streptococcus bovis* and multiple species of *Lactobacillus*. These bacteria can produce large amounts of lactate. Other microbes may use lactate; however, when more lactate is produced than used, it pulls water from the bloodstream into the rumen. This can lead to dehydration and diarrhea.

Abnormal blood flow, either from dehydration, septicemia or endotoxemia, lowers the amount of oxygen delivered to the body's tissues. Without enough oxygen, tissues will use anaerobic metabolism — a process of generating energy without oxygen. A byproduct of anaerobic metabolism is lactic acid. Excess lactic acid in the bloodstream changes the pH in the blood and causes a condition called metabolic acidosis.

The acidic environment can also damage the lining of the rumen and the papillae, potentially causing permanent scarring called rumen parakeratosis. In severe cases, the rumen lining may slough off (shed). The rumen lining functions as a protective barrier between the rumen contents and the blood of the animal. This damage can lead to fungal infections (rumen mycosis) and create openings for harmful bacteria and toxins to enter the bloodstream. When bacteria and toxins enter the animal's bloodstream, they are filtered and detoxified by the liver. Large amounts of bacteria or toxins can overwhelm the liver's ability to detoxify the blood. This can lead to liver abscesses, septic thrombi (infected blood clots) or a systemic infection of the blood called septicemia.

Vena caval thromboembolic disease occurs when septic thrombi (infected blood clots) form in the large blood vessel between the liver and the heart,

which is called the vena cava. These thrombi (clots) can spread through the body and into the lungs. Septicemia, pneumonia and hemorrhage in the lungs can be seen with this disease.

When the rumen pH is lowered, microbes that require a more basic environment may die off. Some microbes, upon death, release toxins, called endotoxins, into the rumen. These endotoxins and live bacteria can move through the injured rumen lining and enter the animal's circulatory system, causing endotoxemia (endotoxins in the blood) or septicemia (bacteria in the blood). Endotoxemia and septicemia cause inflammatory reactions and may lead to fatal toxic shock or septic shock.

Laminitis, inflammation of the sensitive laminae of the foot, can develop in some cases of rumen acidosis. The sensitive laminae are the feather-like layers of tissue that connect the hoof to the bone beneath the hoof. When the sensitive laminae swell, they are compressed between the bone and the hard hoof wall. This is a painful condition.

If the rumen environment shifts to favor a group of microbes that produce excessive hydrogen sulfide or thiaminase (an enzyme that degrades thiamine), the animal may develop polioencephalomalacia. Polioencephalomalacia is a condition characterized by brain cell inflammation and cell death and manifests as ataxia (staggering gait), abnormal postures, recumbency and death.

Bloat, or ruminal tympany, can occur as a secondary problem to rumen acidosis. Both a lower level of rumen pH and excessive fluid in the rumen will lower rumen motility. An acidic environment may alter the rumen microbes and lead to frothy bloat.

The symptoms of rumen acidosis can vary greatly depending on how severe the situation is, how long it lasts and the presence of secondary problems such as metabolic acidosis, septicemia, bloat and endotoxemia.

Strategies for managing rumen acidosis depend on the animal's condition. General strategies include correcting the pH in the rumen, correcting any fluid and electrolyte imbalances, and using antibiotics if an infection is present.

Some cases may require a rumenotomy to relieve bloat or remove large volumes of grain. Introducing healthy rumen microbes from another animal, a process called transfaunation, may also help restore normal microbial fermentation. In severe cases, salvage slaughter or euthanasia may be recommended.

Rumen acidosis can be classified as either acute rumen acidosis or subacute rumen acidosis.

Acute rumen acidosis occurs when the rumen pH drops and stays below 5.0 and the animal develops signs of metabolic acidosis. Metabolic acidosis is caused by a combination of dehydration, changes in how the body uses oxygen (associated with endotoxemia or septicemia) and too much lactate being absorbed into the gastrointestinal system. These animals may also develop bloat, diarrhea, septicemia and endotoxemia. Over time, they are at risk for rumen mycosis, rumen parakeratosis, liver abscesses, vena caval thromboembolism and laminitis.

As the condition progresses, animals may show a range of symptoms. Initially, the animal may increase how much water they are drinking, followed by decreased food and water intake, bloat, a water-filled rumen (hydrorumen), diarrhea, dehydration, recumbency, stupor and death.

Treating acute rumen acidosis can include giving fluids or sodium bicarbonate through an intravenous line, giving the animal antimicrobials, anti-inflammatories, or magnesium hydroxide, which can help normalize the rumen pH, performing a rumenotomy to remove undigested food, or reintroducing healthy microbes into the rumen to restore normal function (transfaunation). Salvage harvest may be recommended in some cases.

Subacute rumen acidosis (SARA) is seen in animals that develop a rumen pH between 5.5 and 5.0. While animals with SARA won't show signs of metabolic acidosis, they are still at risk for developing other secondary problems like bloat, septicemia and endotoxemia. SARA is most often seen in feedlot cattle that are fed a high-grain diet and in pre- and postpartum animals that have recently had their rations changed. Animals that develop SARA may

have decreased production rates because nutrient absorption from the rumen is decreased by rumen parakeratosis.

The signs of SARA can be subtle and might include decreased appetite, rumen motility, growth rates and milk production. More obvious signs may be related to secondary problems such as laminitis, liver abscesses, vena caval thrombosis and polioencephalomalacia.

Managing SARA often involves changing the animal's diet or adding bicarbonate to balance the acidity in the rumen. Other treatments may include anti-inflammatories, antimicrobials and transfaunation.

Recommendations for preventing rumen acidosis

- Gradually change the animal's diet over a three-week time frame to allow the rumen microbiome to slowly adapt to new feeds.
- Include enough fiber with adequate particle size in their diet to support regurgitation, saliva production (which adds bicarbonate to the rumen), and normal rumen motility, as this helps with digestion and normal rumen function.
- Avoid errors in feeding by establishing feeding routines and good communication about the animals' health status.
- Ensure that animals and feed storage areas are secure to prevent accidental consumption of food.
- Establish consistent feeding routines that prevent excessive volumes of grain from being given at any one time.
- Consider adding bicarbonate to their feed to help if their diet might lead to acidosis.
- If animals are at risk for polioencephalomalacia, consider supplementing their ration with thiamine until a long-term solution is found.
- If animals are at risk for frothy bloat, consider adding surfactants to their grain or providing lick blocks that contain a surfactant available

until a long-term solution is found. Mineral oil and poloxalene are two surfactants used in grazing cattle at risk for legume-induced frothy bloat.

RUMEN ALKALOSIS

Rumen alkalosis is when the rumen pH rises above 7.0. This can happen if the bicarbonate from saliva is not balanced by volatile fatty acids production during rumen fermentation. Poorly fermentable feeds or microbial inactivity may cause limited production of volatile fatty acids and the rumen can become alkalotic. Anorexic animals may also develop rumen alkalosis. Symptoms of mild alkalosis include decreased rumen motility.

Overfeeding nonprotein nitrogen sources or accidental exposure to fertilizers containing ammonia can also cause rumen alkalosis and possibly ammonia toxicity. In these cases, the rumen pH level can rise to 7.5-8.5. Signs of ammonia toxicity include bloat, diarrhea, decreased rumen motility, incoordination, weakness, muscle tremors and hyperexcitability, a condition in which nerve cells are excessively responsive to stimuli. These cases can progress rapidly and be fatal.

To prevent alkalosis, ensure that the feed contains enough digestible carbohydrates to maintain volatile fatty acid production, properly use nonprotein nitrogen ingredients and avoid incidental ingestion of ammonia-containing substances, such as fertilizer.

ABOMASUM DISPLACEMENT AND ABOMASAL VOLVULUS

Abomasum displacement is a condition where the abomasum moves from its normal position. Abomasal volvulus occurs when the abomasum moves into an abnormal position and twists around itself, causing constriction of the blood supply and outflow of food material. The causes of these diseases are not completely understood. Animals that have hyperketonemia or hypocalcemia are more at risk for developing abomasal displacement.

Hyperketonemia is characterized by abnormally high levels of ketone bodies in the bloodstream. Ketone bodies are compounds produced by the

liver when fatty acids are used to provide energy. When glucose isn't readily available, the animal will use more fats for energy and produce more ketone bodies. Hyperketonemia is often seen when pre- and postpartum animals use more energy than they consume and rapidly use their stored fat. Animals may show clinical signs of ketosis (clinical ketosis) or have elevated levels of ketones without clinical signs (subclinical ketosis).

Hypocalcemia is characterized by low levels of calcium in the blood. This disease can occur in postpartum animals that have a high calcium requirement due to lactation.

Both hyperketonemia and hypocalcemia can decrease abomasal motility — contractions that mix food with digestive enzymes and move it along the digestive tract. The abomasum can then fill with gas produced by microbial fermentation, making it more prone to displacement.

The abomasum typically lies on the right side of the animal's abdomen. When it crosses under the rumen and floats upward between the left side of the body and the rumen, it's called a left displaced abomasum (LDA). This is the most common form seen in postpartum cattle.

When the abomasum floats upward along the right side of the abdomen, it's called a right displaced abomasum (RDA). RDAs are less common but can lead to a more serious condition called abomasal volvulus, where the abomasum twists around itself, potentially blocking the outflow of the abomasum and the blood vessels to the abomasum. Abomasal displacement and volvulus are managed with surgery and by treating any underlying health disorders. An abomasal volvulus is an emergency.

Signs of RDA and LDA include decreased appetite, less milk production and less manure output. Signs of abomasal volvulus may look similar to an RDA, with the animal's condition worsening rapidly, leading to severe dehydration, increased heart rate and no rumen motility.

Although not all cases of abomasal disease are associated with hyperketonemia or hypocalcemia, monitoring animals around calving time and

providing a diet that has adequate energy and calcium can help reduce the risk of these conditions.

NUTRITIONAL CAUSES OF RECURRENT OR CHRONIC DIARRHEA

Diarrhea can be a sign of many digestive diseases, including many infectious and parasitic diseases. While infectious diseases aren't covered in this publication, it is worth noting that nutritional deficiencies can weaken an animal's immune system, making it more vulnerable to infections.

Vitamin A deficiency can result in diarrhea. Since young animals have not had time to develop liver and fat reserves of vitamin A, they may be more susceptible and show signs earlier than

adults. Besides diarrhea, other signs of vitamin A deficiency may include night blindness, unhealthy skin and coat, poor reproductive performance, musculoskeletal abnormalities and a weakened immune system. An animal's ability to use vitamin A can be affected by factors such as the amount of nitrate, phosphorus or vitamin E in the animal's diet.

Mineral imbalances that can cause diarrhea include

- Cobalt deficiency
- Copper deficiency
- Iron toxicosis (poisoning)
- Selenium deficiency or toxicosis

Joanne McCrea, Assistant Professor of Large Animal Medicine

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