Subacute Ruminal Acidosis

(Chronic ruminal acidosis, Subclinical ruminal acidosis)

By Ingrid Lorenz , DMV, DMVH, DECBHM, School of Agriculture, Food Science and Veterinary Medicine, University College Dublin

Ruminant animals are adapted to digest and metabolize predominantly forage diets; however, growth rates and milk production are increased substantially when ruminants consume high-grain diets. One consequence of feeding excessive amounts of rapidly fermentable carbohydrates in conjunction with inadequate fiber to ruminants is subacute ruminal acidosis, which is characterized by periods of low ruminal pH that resolve without treatment and is rarely diagnosed. Dairy cows, feedlot cattle, and feedlot sheep are at risk of developing this condition.

Etiology and Pathophysiology:

Ruminal pH fluctuates considerably during a 24–hr period (typically between 0.5–1 pH units) and is determined by the dynamic balance between the intake of fermentable carbohydrates, buffering capacity of the rumen, and rate of acid absorption from the rumen. In general, subacute ruminal acidosis is caused by ingestion of diets high in rapidly fermentable carbohydrates and/or deficient in physically active fiber. Subacute ruminal acidosis is most commonly defined as repeatedly occurring prolonged periods of depression of the ruminal pH to values between 5.6 and 5.2. The low ruminal pH is caused by excessive accumulation of volatile fatty acids (VFAs) without persistent lactic acid accumulation and is restored to normal by the animal's own physiologic responses.

The ability of the rumen to rapidly absorb organic acids contributes greatly to the stability of ruminal pH. It is rarely difficult for peripheral tissues to utilize VFAs already absorbed from the rumen; however, absorption of these VFAs from the rumen can be an important bottleneck.

Ruminal VFAs are absorbed passively across the rumen wall. This passive absorption is enhanced by finger-like papillae, which project away from the rumen wall and provide massive surface area

for absorption. Ruminal papillae increase in length when cattle are fed higher-grain diets; this presumably increases ruminal surface area and absorptive capacity, which protects the animal from acid accumulation in the rumen. Dairy cows are especially at risk in the transition period, because the ruminal mucosa needs several weeks to adjust to high-grain diets, and in peak lactation, when high levels of easily fermentable carbohydrates are fed to avoid excessive negative energy balance.

One mechanism by which affected animals resolve ruminal acidosis and return ruminal pH to normal is by selecting long forage particles, either by choosing to preferentially consume long dry hay or by sorting a mixed ration in favor of longer forage particles. Another mechanism is by reducing overall feed intake. Depressed dry-matter intake becomes especially evident if ruminal pH falls below ~5.5. Intake depression may be mediated by pH receptors and/or osmolality receptors in the rumen. Inflammation of the ruminal epithelium (rumenitis) could cause pain and also contribute to intake depression during subacute ruminal acidosis.

Absorption of VFA inherently increases as ruminal pH drops. These acids are absorbed only in the protonated state. Because they have a pKa of ~4.8, the proportion of these acids that is protonated increases dramatically as ruminal pH decreases below 5.5. Lactate levels in the ruminal fluid of cattle with subacute ruminal acidosis, if measured, are usually not increased; however, the pathogenesis of excessive lactate production in the rumen is well described. Ruminal carbohydrate fermentation shifts to lactate production at lower ruminal pH (mostly due to Streptococcus bovis proliferating and shifting to lactate instead of VFA production); this can offset gains from VFA absorption. Ruminal lactate production is undesirable, because lactate has a much lower pKa than VFAs (3.9 vs. 4.8). For example, lactate is 5.2 times less protonated than VFAs at pH 5. As a result, lactate stays in the rumen longer and contributes to the downward spiral in ruminal pH.

Additional adaptive responses are invoked if lactate production begins. Lactate-utilizing bacteria, such as Megasphaera elsdenii and Selenomonas ruminantium, begin to proliferate. These

beneficial bacteria convert lactate to other VFAs, which are then easily protonated and absorbed. However, the turnover time of lactate utilizers is much slower than that of lactate synthesizers. Thus, this mechanism may not be invoked quickly enough to fully stabilize ruminal pH. Periods of very high ruminal pH, as during feed deprivation, may inhibit populations of lactate utilizers (which are sensitive to higher ruminal pH) and leave them more susceptible to severe ruminal acidosis.

Besides disrupting microbial balance, feed deprivation causes cattle to overeat when feed is reintroduced. This creates a double effect in lowering ruminal pH. Cycles of feed deprivation followed by overconsumption greatly increase the risk of subacute ruminal acidosis.

Low ruminal pH during subacute ruminal acidosis also reduces the number of species of bacteria in the rumen, although the metabolic activity of the bacteria that remain is very high. Protozoal populations are particularly limited at lower ruminal pH; the absence of ciliated protozoa in ruminal fluid is often observed during bouts of subacute ruminal acidosis. When fewer species of bacteria and protozoa are present, the ruminal microflora are less stable and less able to maintain normal ruminal pH during periods of sudden dietary change. Thus, periods of subacute ruminal acidosis leave animals more susceptible to future episodes of ruminal acidosis.

The pathophysiologic consequences of ruminal acidosis have mainly been described in feedlot cattle and in cattle surviving acute ruminal acidosis. Low ruminal pH may lead to rumenitis, erosion, and ulceration of the ruminal epithelium. Once the ruminal epithelium is inflamed, bacteria may colonize the papillae and leak into the portal circulation. These bacteria may cause liver abscesses, which may eventually lead to peritonitis around the site of the abscess.

Caudal vena cava syndrome is caused by the release of septic emboli from liver abscesses; this septic material then travels via the caudal vena cava to the lungs. These bacteria proliferate in lung tissue and may ultimately invade pulmonary vessels, causing them to rupture. This is

observed clinically as hemoptysis and even peracute deaths due to massive pulmonary hemorrhage.

Subacute ruminal acidosis has traditionally been associated with claw horn lesions, assumed to be caused by subacute laminitis. However, this pathophysiologic mechanism has not been experimentally characterized or reproduced. In recent years, alternative explanations for the development of claw horn lesions have been suggested.

Clinical Findings:

The main clinical signs attributed to subacute ruminal acidosis are reduced or cyclic feed intake, decreased milk production, reduced fat, poor body condition score despite adequate feed intake, and unexplained diarrhea. High rates of culling or unexplained deaths may be noted in the herd. Sporadic cases of caudal vena cava syndrome may also be seen. The clinical signs are delayed and insidious. Actual episodes of low ruminal pH are not identified; in fact, by the time an animal is observed to be off-feed, its ruminal pH has probably been restored to normal. Diarrhea may follow periods of low ruminal pH; however, this finding is inconsistent and may be related to other dietary factors as well.

Diagnosis:

Subacute ruminal acidosis is diagnosed on a group rather than individual basis. Measurement of pH in the ruminal fluid of a representative portion of apparently healthy animals in a group has been used to help make the diagnosis of subacute ruminal acidosis in dairy herds. Animal selection should be from highest-risk groups: cows between ~15–30 days in milk in component-fed herds and cows between ~50–150 days in milk in herds fed total mixed rations. Ruminal fluid is collected by rumenocentesis, and its pH is determined on a pH meter. Twelve or more animals are typically sampled at ~2–4 hr after a grain feeding (in component-fed herds) or 6–10 hr after the first daily feeding of a total mixed ration. If >25% of the animals tested have a ruminal pH <5.5, then the group is considered to be at high risk of subacute ruminal acidosis. This type of

diagnostic tool should be used in conjunction with other factors such as ration evaluation, evaluation of management practices, and identification of health problems on a herd basis.

Milk fat depression is a poor and insensitive indicator of subacute ruminal acidosis in dairy herds.

Treatment and Prevention:

Because subacute ruminal acidosis is not detected at the time of depressed ruminal pH, there is no specific treatment for it. Secondary conditions may be treated as needed.

The key to prevention of subacute ruminal acidosis is allowing for ruminal adaption to high-grain diets, as well as limiting intake of readily fermentable carbohydrates. This requires both good diet formulation (proper balance of fiber and nonfiber carbohydrates) and excellent feed bunk management. Animals consuming well-formulated diets remain at high risk of this condition if they tend to eat large meals because of excessive competition for bunk space or after periods of feed deprivation.

Field recommendations to feed component-fed concentrates to dairy cattle during the first 3 wk of lactation are usually excessive. Feeding excessive quantities of concentrate and insufficient forage results in a fiber-deficient ration likely to cause subacute ruminal acidosis. The same situation may be seen during the last few days before parturition if the ration is fed in separate components; as dry-matter intake drops before calving, dry cows preferentially consume concentrates instead of forage and develop acidosis.

Subacute ruminal acidosis may also be caused by errors in delivery of the rations or by formulation of rations that contain excessive amounts of rapidly fermentable carbohydrates or a deficiency of fiber. Recommendations for the fiber content of dairy rations are available in the National Research Council report, Nutrient Requirements of Dairy Cattle (see Nutritional Requirements of Beef Cattle). Dry-matter content errors in total mixed rations are commonly related to a lack of adjustment for changes in moisture content of forages.

Including long-fiber particles in the diet reduces the risk of subacute ruminal acidosis by encouraging saliva production during chewing and by increasing rumination after feeding. The provision of adequate long-fiber particles reduces the risk of ruminal acidosis but cannot eliminate it. If a total mixed ration is fed, it is important that the long-fiber particles not be easily sorted away from the rest of the diet; this could delay their consumption until later in the day or cause them to be refused completely. Sorting can be prevented by providing long-fiber particles less than ~5 cm in length, by having adequate (~50%–55%) moisture in the mixed ration, and by including ingredients such as liquid molasses that help ration ingredients stick together.

Ruminant diets should also be formulated to provide adequate buffering. This can be accomplished by feedstuff selection and/or by addition of dietary buffers such as sodium bicarbonate or potassium carbonate. The dietary cation-anion difference (DCAD) is used to quantify the buffering capacity of a diet; diets for animals at high risk of ruminal acidosis should be formulated to provide a DCAD of >250 mEq/kg of diet dry matter, using the formula (Na + K) – (Cl + S) to calculate DCAD.

Supplementing the diet with direct-fed microbials that enhance lactate utilization in the rumen may reduce the risk of subacute ruminal acidosis. Yeasts, propionobacteria, lactobacilli, and enterococci have been used for this purpose. Ionophore (eg, monensin sodium) supplementation may also reduce the risk by selectively inhibiting ruminal lactate producers and by reducing meal size.

Bloat in Ruminants

(Ruminal tympany)

By Peter D. Constable , BVSc (Hons), MS, PhD, DACVIM, College of Veterinary Medicine, University of Illinois at Urbana-Champaign

Bloat is an overdistention of the rumenoreticulum with the gases of fermentation, either in the form of a persistent foam mixed with the ruminal contents, called primary or frothy bloat, or in

the form of free gas separated from the ingesta, called secondary or free-gas bloat. It is predominantly a disorder of cattle but may also be seen in sheep. The susceptibility of individual cattle to bloat varies and is genetically determined.

Death rates as high as 20% are recorded in cattle grazing bloat-prone pasture, and in pastoral areas, the annual mortality rate from bloat in dairy cows may approach 1%. There is also economic loss from depressed milk production in nonfatal cases and from suboptimal use of bloat-prone pastures. Bloat can be a significant cause of mortality in feedlot cattle.

Etiology and Pathogenesis:

In primary ruminal tympany, or frothy bloat, the cause is entrapment of the normal gases of fermentation in a stable foam. Coalescence of the small gas bubbles is inhibited, and intraruminal pressure increases because eructation cannot occur. Several factors, both animal and plant, influence the formation of a stable foam. Soluble leaf proteins, saponins, and hemicelluloses are believed to be the primary foaming agents and to form a monomolecular layer around gas rumen bubbles that has its greatest stability at about pH 6. Salivary mucin is antifoaming, but saliva production is reduced with succulent forages. Bloat-producing pastures are more rapidly digested and may release a greater amount of small chloroplast particles that trap gas bubbles and prevent their coalescence. The immediate effect of feeding is probably to supply nutrients for a burst of microbial fermentation. However, the major factor that determines whether bloat will occur is the nature of the ruminal contents. Protein content and rates of digestion and ruminal passage reflect the forage's potential for causing bloat. Over a 24-hr period, the bloat-causing forage and unknown animal factors combine to maintain an increased concentration of small feed particles and enhance the susceptibility to bloat.

Bloat is most common in animals grazing legume or legume-dominant pastures, particularly alfalfa, ladino, and red and white clovers, but also is seen with grazing of young green cereal crops, rape, kale, turnips, and legume vegetable crops. Legume forages such as alfalfa and clover have a higher percentage of protein and are digested more quickly. Other legumes, such as

sainfoin, crown vetch, milk vetch, fenugreek, and birdsfoot trefoil, are high in protein but do not cause bloat, probably because they contain condensed tannins, which precipitate protein and are digested more slowly than alfalfa or clover. Leguminous bloat is most common when cattle are placed on lush pastures, particularly those dominated by rapidly growing leguminous plants in the vegetative and early bud stages, but can also be seen when high-quality hay is fed.

Frothy bloat also is seen in feedlot cattle, and less commonly in dairy cattle, on high-grain diets. The cause of the foam in feedlot bloat is uncertain but is thought to be either the production of insoluble slime by certain species of rumen bacteria in cattle fed high-carbohydrate diets or the entrapment of the gases of fermentation by the fine particle size of ground feed. Fine particulate matter, such as in finely ground grain, can markedly affect foam stability, as can a low roughage intake. Feedlot bloat is most common in cattle that have been on a grain diet for 1–2 mo. This timing may be due to the increase in the level of grain feeding or to the time it takes for the slime-producing rumen bacteria to proliferate to large enough numbers.

In secondary ruminal tympany, or free-gas bloat, physical obstruction of eructation is caused by esophageal obstruction due to a foreign body (eg, potatoes, apples, turnips, kiwifruit), stenosis, or pressure from enlargement outside the esophagus (as from lymphadenopathy or sporadic juvenile thymic lymphoma). Interference with esophageal groove function in vagal indigestion and diaphragmatic hernia may cause chronic ruminal tympany. This also occurs in tetanus. Tumors and other lesions, such as those caused by infection with Actinomyces bovis, of the esophageal groove or the reticular wall are less common causes of obstructive bloat. There also may be interference with the nerve pathways involved in the eructation reflex. Lesions of the wall of the reticulum (which contains tension receptors and receptors that discriminate between gas, foam, and liquid) may interrupt the normal reflex essential for escape of gas from the rumen.

Ruminal tympany also can be secondary to the acute onset of ruminal atony that occurs in anaphylaxis and in grain overload; this causes a decrease in rumen pH and possibly an esophagitis and rumenitis that can interfere with eructation. Ruminal tympany also develops with

hypocalcemia. Chronic ruminal tympany is relatively frequent in calves up to 6 mo old without apparent cause; this form usually resolves spontaneously.

Unusual postures, particularly lateral recumbency, are commonly associated with secondary tympany. Ruminants may die of bloat if they become accidentally cast in dorsal recumbency or other restrictive positions in handling facilities, crowded transportation vehicles, or irrigation ditches.

Clinical Findings:

Bloat is a common cause of sudden death. Cattle not observed closely, such as pastured and feedlot cattle and dry dairy cattle, usually are found dead. In lactating dairy cattle, which are observed regularly, bloat commonly begins within 1 hr after being turned onto a bloat-producing pasture. Bloat may develop on the first day after being placed on the pasture but more commonly develops on the second or third day.

In primary pasture bloat, the rumen becomes obviously distended suddenly, and the left flank may be so distended that the contour of the paralumbar fossa protrudes above the vertebral column; the entire abdomen is enlarged. As the bloat progresses, the skin over the left flank becomes progressively more taut and, in severe cases, cannot be “tented.” Dyspnea and grunting are marked and are accompanied by mouth breathing, protrusion of the tongue, extension of the head, and frequent urination. Rumen motility does not decrease until bloat is severe. If the tympany continues to worsen, the animal will collapse and die. Death may occur within 1 hr after grazing began but is more common ~3–4 hr after onset of clinical signs. In a group of affected cattle, there are usually several with clinical bloat and some with mild to moderate abdominal distention.

In secondary bloat, the excess gas is usually free on top of the solid and fluid ruminal contents, although frothy bloat may be seen in vagal indigestion when there is increased ruminal activity. Secondary bloat is seen sporadically. There is tympanic resonance over the dorsal abdomen left

of the midline. Free gas produces a higher pitched ping on percussion than frothy bloat. The distention of the rumen can be detected on rectal examination. In free-gas bloat, the passage of a stomach tube or trocarization releases large quantities of gas and alleviates distention.

Lesions:

Necropsy findings are characteristic. Congestion and hemorrhage of the lymph nodes of the head and neck, epicardium, and upper respiratory tract are marked. The lungs are compressed, and intrabronchial hemorrhage may be present. The cervical esophagus is congested and hemorrhagic, but the thoracic portion of the esophagus is pale and blanched—the demarcation known as the “bloat line” of the esophagus. The rumen is distended, but the contents usually are much less frothy than before death. The liver is pale because of expulsion of blood from the organ.

Diagnosis:
Usually, the clinical diagnosis of frothy bloat is obvious. The causes of secondary bloat must be ascertained by clinical examination to determine the cause of the failure of eructation.

Treatment:
In life-threatening cases, an emergency rumenotomy may be necessary; it is accompanied by an explosive release of ruminal contents and, thus, marked relief for the cow. Recovery is usually uneventful, with only occasional minor complications.

A trocar and cannula may be used for emergency relief, although the standard-sized instrument is not large enough to allow the viscous, stable foam in peracute cases to escape quickly enough. A larger bore instrument (2.5 cm in diameter) is necessary, but an incision through the skin must be made before it can be inserted through the muscle layers and into the rumen. If the cannula fails to reduce the bloat and the animal’s life is threatened, an emergency rumenotomy should be performed. If the cannula provides some relief, an antifoaming agent can be administered

through the cannula, which can remain in place until the animal has returned to normal, usually within several hours.

Cannulation of rumen, cow

Cannulation of rumen, cow. Illustration by Dr. Gheorghe Constantinescu.

Cannulation of rumen, cow

When the animal's life is not immediately threatened, passing a stomach tube of the largest bore possible is recommended. A few attempts should be made to clear the tube by blowing and moving it back and forth in an attempt to find large pockets of rumen gas that can be released. In frothy bloat, it may be impossible to reduce the pressure with the tube, and an antifoaming agent should be administered while the tube is in place. If the bloat is not relieved quickly by the antifoaming agent, the animal must be observed carefully for the next hour to determine whether the treatment has been successful or whether an alternative therapy is necessary.

A variety of antifoaming agents are effective, including vegetable oils (eg, peanut, corn, soybean) and mineral oils (paraffins), at doses of 250–500 mL. Dioctyl sodium sulfosuccinate, a surfactant, is commonly incorporated into one of the above oils and sold as a proprietary antibloat remedy, which is effective if administered early. Poloxalene (25–50 g, PO) is effective in treating legume bloat but not feedlot bloat. Placement of a rumen fistula provides short-term relief for cases of free-gas bloat associated with external obstruction of the esophagus.

Control and Prevention:

Prevention of pasture bloat can be difficult. Management practices used to reduce the risk of bloat include feeding hay, particularly orchard grass, before turning cattle on pasture, maintaining grass dominance in the sward, or using strip grazing to restrict intake, with movement of animals to a new strip in the afternoon, not the early morning. Hay must constitute at least one-third of the diet to effectively reduce risk of bloat. Feeding hay or strip grazing may be reliable when the pasture is only moderately dangerous, but these methods are less reliable

when the pasture is in the pre-bloom stage and the bloat potential is high. Mature pastures are less likely to cause bloat than immature or rapidly growing pastures.

The only satisfactory method available to prevent pasture bloating is continual administration of an antifoaming agent during the risk period. This is widely practiced in grassland countries such as Australia and New Zealand. The most reliable method is drenching twice daily (eg, at milking times) with an antifoaming agent. Spraying the agent onto the pasture is equally effective, provided the animals have access only to treated pasture. This method is ideal for strip grazing but not when grazing is uncontrolled. The antifoaming agent can be added to the feed or water or incorporated into feed blocks, but success with this method depends on adequate individual intake. The agent can be “painted” on the flanks of the animals, from which it is licked during the day, but animals that do not lick will be unprotected.

Available antifoaming agents include oils and fats and synthetic nonionic surfactants. Oils and fats are given at 60–120 mL/head/day; doses up to 240 mL are indicated during dangerous periods. Poloxalene, a synthetic polymer, is a highly effective nonionic surfactant that can be given at 10–20 g/head/day and up to 40 g/head/day in high-risk situations. It is safe and economical to use and is administered daily through the susceptible period by adding to water, feed grain mixtures, or molasses. Pluronic agents facilitate the solubilization of water-insoluble factors that contribute to formation of a stable foam. A pluronic detergent (Alfasure®) and a water-soluble mixture of alcohol ethoxylate and pluronic detergents (Blocare 4511) also are effective but are not approved by the FDA. Ionophores effectively prevent bloat, and a sustained-release capsule administered into the rumen and releasing 300 mg of monensin daily for a 100-day period protects against pasture bloat and improves milk production on bloat-prone pastures.

The ultimate aim in control is development of a pasture that permits high production, while keeping incidence of bloat low. The use of pastures of clover and grasses in equal amounts comes closest to achieving this goal. Bloat potential varies between cultivars of alfalfa, and low-risk LIRD (low initial rate of digestion) cultivars are available commercially. The addition of legumes with

high condensed tannins to the pasture seeding mix (10% sainfoin) can reduce the risk of bloat where there is strip grazing, as can the feeding of sainfoin pellets.

To prevent feedlot bloat, rations should contain ≥10–15% cut or chopped roughage mixed into the complete feed. Preferably, the roughage should be a cereal, grain straw, grass hay, or equivalent. Grains should be rolled or cracked, not finely ground. Pelleted rations made from finely ground grain should be avoided. The addition of tallow (3%–5% of the total ration) may be successful occasionally, but it was not effective in controlled trials. The nonionic surfactants, such as poloxalene, have been ineffective in preventing feedlot bloat, but the ionophore lasalocid is effective in control.

Traumatic Reticuloperitonitis
(Hardware disease, Traumatic gastritis)

By

Peter D. Constable
, BVSc (Hons), MS, PhD, DACVIM, College of Veterinary Medicine, University of Illinois at Urbana-Champaign

Traumatic reticuloperitonitis develops as a consequence of perforation of the reticulum. It is important as a differential diagnosis of other diseases marked by stasis of the GI tract, because it causes similar signs. Traumatic reticuloperitonitis is most common in mature dairy cattle, occasionally seen in beef cattle, and rarely reported in other ruminants.

Cattle commonly ingest foreign objects, because they do not discriminate against metal materials in feed and do not completely masticate feed before swallowing. The disease is common when green chop, silage, and hay are made from fields that contain old rusting fences or baling wire,

or when pastures are on areas or sites where buildings have recently been constructed, burned, or torn down. The grain ration may also be a source because of accidental addition of metal.

Etiology:

Swallowed metallic objects, such as nails or pieces of wire, fall directly into the reticulum or pass into the rumen and are subsequently carried over the ruminoreticular fold into the cranioventral part of the reticulum by ruminal contractions. The reticulo-omasal orifice is elevated above the floor, which tends to retain heavy objects in the reticulum, and the honeycomb-like reticular mucosa traps sharp objects. Contractions of the reticulum promote penetration of the wall by the foreign object. Compression of the ruminoreticulum by the uterus in late pregnancy and straining during parturition increase the likelihood of an initial penetration of the reticulum and may also disrupt adhesions caused by an earlier penetration.

Perforation of the wall of the reticulum allows leakage of ingesta and bacteria, which contaminates the peritoneal cavity. The resulting peritonitis is generally localized and frequently results in adhesions. Less commonly, a more severe diffuse peritonitis develops. The object can penetrate the diaphragm and enter the thoracic cavity (causing pleuritis and sometimes pulmonary abscessation) and the pericardial sac (causing pericarditis, sometimes followed by myocarditis). Occasionally, the liver or spleen may be pierced and become infected, resulting in abscessation, or septicemia can develop.

Relationship between the reticulum, diaphragm, and heart and pericardium in large ruminants
The relationship between the reticulum, diaphragm, and heart and pericardium in large ruminants. Illustration by Dr. Gheorghe Constantinescu.

Relationship between the reticulum, diaphragm, and heart and pericardium in large ruminants
Clinical Findings:

The initial penetration of the reticulum is characterized by the sudden onset of ruminoreticular atony and a sharp fall in milk production. Fecal output is decreased. The rectal temperature is often mildly increased. The heart rate is normal or slightly increased, and respiration is usually shallow and rapid. Initially, the cow exhibits an arched back; an anxious expression; a reluctance to move; and an uneasy, careful gait. Forced sudden movements as well as defecating, urinating, lying down, getting up, and stepping over barriers may be accompanied by groaning. A grunt may be elicited by applying pressure to the xiphoid or by firmly pinching the withers, which causes extension of the thorax and lower abdomen. The grunt can be detected by placing a stethoscope over the trachea and applying pressure or pinching the withers at the end of an inspiration. Tremor of the triceps and abduction of the elbow may be seen.

In chronic cases, feed intake and fecal output are reduced, and milk production remains low. Signs of cranial abdominal pain become less apparent, and the rectal temperature usually returns to normal as the acute inflammation subsides and peritoneal contamination is walled off. Some cattle develop vagal indigestion syndrome (see Vagal Indigestion Syndrome in Ruminants) because of the adhesions that form after foreign body perforation, particularly those on the ventromedial reticulum.

Cows with pleuritis or pericarditis due to foreign body perforation usually are depressed, tachycardic (>90 bpm), and pyrexic (104°F [40°C]). Pleuritis is manifest by fast, shallow respiration; muffled lung sounds; and possibly pleuritic friction rubs. Thoracentesis may yield several liters of septic fluid. Traumatic pericarditis is most commonly characterized by muffled heart sounds; however, early in the disease process pericardial friction rubs or gas and fluid splashing sounds (washing machine murmur) can be heard on auscultation. Jugular vein distention and congestive heart failure with marked submandibular and brisket edema is a frequent sequela of traumatic reticulopericarditis. Prognosis is grave with these complications. Penetration through the pericardium into the myocardium usually results in extensive hemorrhage into the pericardial sac or ventricular arrhythmias and sudden death.

Diagnosis:

This can be based on history (when available) and clinical findings if the cow is examined when signs initially appear. Without an accurate history and when the condition has been present for several days or longer, diagnosis is more difficult. Other causes of peritonitis, particularly perforated abomasal ulcers, can be difficult to distinguish from traumatic reticuloperitonitis. Differential diagnoses should include conditions that can produce variable or nonspecific GI signs, eg, indigestion, lymphosarcoma, or intestinal obstruction. Abomasal displacement or volvulus should be excluded by simultaneous auscultation and percussion. Pleuritis or pericarditis of nontraumatic origin produces signs similar to those associated with foreign body perforation.

Although not always necessary, laboratory tests may be helpful. In many cases, there is a neutrophilia with a left shift. Plasma fibrinogen concentrations may be increased, and serum haptoglobin, amyloid A, and total plasma protein concentrations may be markedly increased. Severely affected cattle may have coagulation abnormalities, as evidenced by prolonged prothrombin time, thrombin time, and activated partial thromboplastin time. The acid-base status and serum electrolyte levels are typically normal, because abomasal and small-intestinal absorption can remain normal. However, marked hypokalemic, hypochloremic metabolic alkalosis can be seen, presumably because adynamic ileus from peritonitis can affect abomasal and GI motility and resorption of abomasal secretions. The metabolic alkalosis can be created or exacerbated by treatment with alkalinizing agents such as magnesium hydroxide used as a laxative. Peritoneal fluid analysis can help determine whether peritonitis is present, particularly the concentration of d-dimer and the neutrophil percentage in the peritoneal fluid. However, the peritonitis frequently becomes walled off, and in these cases peritoneal fluid may be within the reference range unless obtained from within the lesion. The presence of a magnet in the reticulum can be determined by movement of a magnetic compass in the region of the cranioventral abdomen; the presence of a magnet in the reticulum makes traumatic reticuloperitonitis very unlikely unless the penetrating object is not magnetic.

Ultrasonography of the ventral abdomen using a 3-MHz transducer is the most accurate way to diagnose localized peritonitis near the reticulum and characterize the reticular contraction frequency. It rarely identifies the presence of a penetrating object. Ultrasonography of the heart and thorax is very useful in the diagnosis of pleuritis and pericarditis as a sequelae of traumatic reticuloperitonitis and has replaced radiography in the diagnosis of reticuloperitonitis.

Lateral radiographs of the cranioventral abdomen can detect metallic material in the reticulum but should be taken only after oral administration of a magnet. To determine whether the reticulum is currently perforated, the foreign body must be visible beyond the border of the reticulum, unattached to the magnet in the reticulum, or positioned off the floor of the reticulum. A depression in the cranioventral aspect of the reticulum or identification of an abscess (by gas accumulation outside a viscus), soft-tissue masses, or a fluid line in the cranial abdomen are also reliable radiographic findings of penetration. Portable radiographic units cannot penetrate the reticular area of standing adult cattle, and the cow may need to be transported to where there is equipment with sufficient power. The cow should not be placed in dorsal recumbency to obtain radiographs, because such manipulation places stress on adhesions and may lead to a localized peritonitis becoming a diffuse peritonitis due to gravitational spread of infection.

Electronic metal detectors can identify metal in the reticulum but do not distinguish between perforating and nonperforating foreign bodies.

Treatment:

Treatment of the typical case seen early in its course may be surgical or medical. Either approach improves the chances of recovery from ~60% in untreated cases to 80%–90%. Surgery involves rumenotomy with manual removal of the object(s) from the reticulum; if an abscess is adhered to the reticulum, it should be aspirated (to confirm it is an abscess) and then drained into the reticulum. Antimicrobials should be administered perioperatively. Medical treatment involves administration of antimicrobials to control the peritonitis and a magnet to prevent recurrence.

Because of the mixed bacterial flora in the lesion, a broad-spectrum antimicrobial agent such as oxytetracycline (16 mg/kg/day, IV) should be used. Penicillin (22,000 IU/kg, IM, once to twice daily) is widely used and effective in many cases despite its limited spectrum. Affected cows should be confined for 1–2 wk; placing them on an inclined plane (elevated in front) is believed by some to limit further penetration of the foreign object, but supporting studies are lacking. Supportive therapy, such as oral or occasionally IV fluids and SC calcium borogluconate or calcium gluconate, should be administered as needed. Rumen inoculation (4–8 L of ruminal fluid from a healthy donor) is beneficial in some cases with prolonged ruminal stasis and loss of normal flora.

More advanced cases, those with obvious secondary complications, or those that do not respond to initial medical or surgical therapy should be evaluated from an economic perspective; if the cow is of limited value, slaughter should be considered if the carcass is likely to pass inspection.

Prevention:

Preventive measures include avoiding the use of baling wire, passing feed over magnets to remove metallic objects, keeping cattle away from sites of new construction, and completely removing old buildings and fences. Additionally, bar magnets may be administered PO, preferably after fasting for 18–24 hr. Usually, the magnet remains in the reticulum and holds any ferromagnetic objects on its surface. There is good evidence that giving magnets to all herd replacement heifers and bulls at ~1 yr of age minimizes the incidence of traumatic reticuloperitonitis.

Vagal Indigestion Syndrome in Ruminants

(Chronic indigestion)

By

Peter D. Constable

, BVSc (Hons), MS, PhD, DACVIM, College of Veterinary Medicine, University of Illinois at Urbana-Champaign

Vagal indigestion syndrome is characterized by the gradual development of abdominal distention secondary to rumenoreticular distention. The distention was originally thought to be the result of lesions affecting the ventral vagus nerve. Vagal indigestion syndrome is seen most commonly in cattle but has been reported in sheep.

Etiology and Pathogenesis:

Diseases that result in injury, inflammation, or pressure on the vagus nerve can result in clinical signs of vagal indigestion syndrome. However, vagal nerve damage is not present in most cases of vagus indigestion, and the most common cause is traumatic reticuloperitonitis (see Traumatic Reticuloperitonitis). Conditions resulting in mechanical obstruction of the cardia or reticulo-omasal orifice (eg, papillomas or ingested placenta) can also result in vagal indigestion if ruminoreticular distention is present and the condition is subacute to chronic.

Historically, there were four types of vagal indigestion described based on the purported site of the functional obstruction. Type I was failure of eructation or free-gas bloat, type II was a failure of omasal transport, type III was secondary abomasal impaction, and type IV was indigestion of late gestation. Types I and IV are rare, and this categorization system has minimal clinical relevance.

Type I vagal indigestion, or failure of eructation, results in free-gas bloat and has been attributed to inflammatory lesions in the vicinity of the vagus nerve, such as localized peritonitis, adhesions (usually after an episode of traumatic reticuloperitonitis), or chronic pneumonia with anterior mediastinitis. Other potential causes for type I vagal indigestion include pharyngeal trauma, which affects a more proximal part of the vagus nerve, and esophageal compression by abscesses or neoplasia, such as lymphosarcoma. Vagal indigestion can develop in cattle after abomasal volvulus without abomasal impaction. These cases would presumably fall into the category of type I vagal indigestion with damage to the vagal nerve near the reticulum and omasum.

Type II vagal indigestion, more correctly termed failure of omasal transport, develops as a result of any condition that prevents ingesta from passing through the omasal canal into the abomasum. Adhesions and abscesses (reticular or single liver abscesses) are the most common cause of failure of omasal transport and are usually located on the right or medial wall of the reticulum near the route of the vagus nerve. Reticular abscesses and adhesions are almost invariably the result of traumatic reticuloperitonitis. Mechanical obstruction of the omasal canal by ingested material (eg, plastic bags, rope, placenta) or masses (eg, lymphosarcoma, squamous cell carcinoma, granulomas, or papillomas) can also cause chronic ruminoreticular distention due to failure of omasal transport.

Type III vagal indigestion is a secondary abomasal impaction. Primary abomasal impaction develops due to feeding of dry, course roughage, such as straw, in a chopped or ground form with restricted access to water and usually during extremely cold temperatures (see Dietary Abomasal Impaction). Secondary abomasal impaction is seen most commonly after an episode of traumatic reticuloperitonitis or occasionally as a sequela of abomasal volvulus. Mechanical fixation of the reticulum to the ventral abdominal floor in cows with reticuloperitonitis interferes with the normal sieving action of the reticulum, with passage of large fiber particles (>2 mm long) into the abomasum. The abomasum has difficulty in emptying the larger particles of food because of the increased viscosity, and they accumulate in the abomasum, resulting in abomasal impaction.

Type IV vagal indigestion, or partial forestomach obstruction, is poorly defined. It typically develops in cattle during gestation and is more appropriately termed indigestion of late gestation. The condition is thought to be related to the enlarging uterus shifting the abomasum to a more cranial position, which inhibits normal abomasal emptying.

Clinical Findings:

The clinical signs vary to some extent with the location of the obstruction. In all cases, there is a gradual development (over days to weeks) of abdominal distention secondary to ruminoreticular distention. Distention of the dorsal and ventral sacs of the rumen results in an “L-shaped” rumen on rectal examination. Left dorsal and left and right ventral distention of the abdomen causes a “papple” (pear plus apple) shape as viewed from behind.

Cattle with vagal indigestion syndrome have a diminished appetite, which typically improves temporarily if distention is relieved. Milk production gradually decreases, fecal output is reduced, and the rumen develops a “splashy” fluid consistency. The feces are characteristically very scant and sticky and may contain longer than normal particles. The strength of rumen contractions is decreased; however, rumen motility is often increased (3–4 contractions/min). It is commonly possible to see movements of the left abdominal wall that mirror the movements of the hyperactive rumen. However, rumen contraction sounds are not audible because the contents have become frothy due to the prolonged contractions and failure of the rumen to empty.

Temperature and respiratory rate are usually normal; however, these can be increased depending on the cause. Bradycardia is present in 25%–40% of cases and is due to decreased feed intake rather than a direct stimulation of the vagus nerve. Tachycardia develops as the disease progresses and cattle become dehydrated. Over time, the animal develops a rough hair coat, loses condition, and becomes weak (in some cases to the point of recumbency), with marked clinical signs of dehydration.

On rectal palpation, the rumen is distended with gas or froth that occupies the entire left abdomen, pushing the left kidney to the right of the midline. The ventral sac of the rumen is enlarged and palpable to the right of the midline (the characteristic “L-shaped” rumen). It is important to recognize that diagnosis of vagal indigestion syndrome requires the presence of a markedly increased ruminoreticular volume. Palpation of the lower half of the right side of the abdomen below the costochondral junction may detect an impacted abomasum that feels doughy. Hematologic findings vary. The PCV can be increased because of dehydration or

decreased because of bone marrow depression (anemia of chronic disease). The WBC may be normal, increased, or decreased. If an inflammatory condition such as peritonitis is present, the neutrophil to lymphocyte ratio is typically reversed, and a neutrophilia may be present. Lymphocytosis can be seen with vagal indigestion due to lymphosarcoma. Leukopenia may be present with diffuse peritonitis. Increased serum globulin and total protein can be seen with abscesses.

Metabolic status is normal, or metabolic alkalosis may be present. The serum chloride concentration varies with the site of the obstruction. It is usually normal if the lesion is proximal to the abomasum. A low serum chloride concentration is consistent with reflux of chloride from the abomasum into the rumen (internal vomiting) and obstruction at the level of the abomasum (type III). Metabolic alkalosis is typically present if serum chloride is decreased. Rumen chloride concentration is increased in type III vagal indigestion and provides a useful method to differentiate type II from type III vagal indigestion. The serum potassium concentration is usually low due to decreased potassium intake in the feed. Serum calcium concentration is often moderately decreased because of ongoing milk production, but it is rarely low enough to cause recumbency. Serum urea and creatinine concentrations increase with dehydration due to prerenal azotemia.

Diagnosis:

Diagnosis is based on the presence of subacute to chronic ruminoreticular and abdominal distention. Because vagal indigestion is by definition a subacute to chronic disease, this diagnosis should not be made in cattle that have not been sick for at least several days, which excludes acute rumen tympany and acute frothy bloat. Other causes of abdominal distention, such as ascites and uterine enlargement, are included in the differential diagnosis and can almost invariably be excluded by rectal palpation because of the absence of ruminoreticular distention. Occasional cases of longstanding obstruction of the cecum or small intestine can cause severe ruminoreticular and abdominal distention; however, palpable cecal or small-intestinal distention

is also palpable rectally. In addition, the rumen is distended but not L-shaped, and a characteristic ping is present in the case of cecocolic volvulus.

Diagnosing the specific cause of vagal indigestion is more difficult but is important because of differences in treatment and prognosis. Physical examination, rectal examination, CBC, blood acid-base determination, and serum biochemical values are often useful. Peritoneal fluid analysis can support the diagnosis of peritonitis if total protein or nucleated cells are increased. Lateral radiographs of the reticulum should be taken to identify an opaque linear foreign body (eg, wire) or reticular abscess. Ultrasonography of the cranioventral abdomen can indicate the presence of focal peritonitis and the reticular contraction rate. Definitive diagnosis often requires exploratory surgery (left paralumbar fossa laparotomy and rumenotomy).

Treatment and Prognosis:

If the value of the animal justifies treatment, surgery is almost always needed to identify and potentially correct the underlying cause. Medical management alone is usually ineffective. A left paralumbar fossa laparotomy and rumenotomy provides the opportunity for definitive treatment in some cases. Emptying the rumen at the time of surgery may help restore normal rumen motility. Stimulation of low-threshold tension receptors in the reticulum occurs under normal circumstances and causes reflex reticuloruminal contractions. However, severe distention causes stimulation of high-threshold receptors that have the opposite effect and inhibit contractions.

Supportive or symptomatic therapy should be provided in all cases, which typically involves correcting dehydration as well as calcium and electrolyte deficits, commonly with oral fluids and electrolytes. Severely dehydrated animals and those with longstanding disease require IV fluids. Fresh water and normal feed should be available. Transfaunation at surgery or via oroesophageal intubation may help reestablish normal rumen flora in cattle with chronic anorexia. Antimicrobials (procaine penicillin or oxytetracycline) should be given if the underlying cause is infectious or if a rumen fistula is created.

Treatment of type I vagal indigestion (failure of eructation) also typically involves creating a rumen fistula to allow free gas to escape. If surgery is not economically feasible and the underlying cause of vagal indigestion has been identified and treated, a rumen trocar can be placed temporarily. Such trocars are commercially available and must be secure and self-retaining to prevent potentially fatal leakage of rumen contents into the peritoneal cavity. The trocar should not be removed for at least 2 wk to allow firm adhesions to form between the rumen and body wall.

The prognosis for animals with type I vagal indigestion is usually favorable. After creation of a rumen fistula, the signs of vagal indigestion resolve in nearly all cases. However, animals with chronic respiratory disease or pharyngeal trauma may not recover from the underlying condition. Leakage of ingesta from fistulas can cause off-flavored milk. Peritonitis can develop from leakage around the fistula or after rumenotomy; however, this should not happen with good surgical technique.

Type II vagal indigestion (failure of omasal transport) rarely responds to supportive or symptomatic therapy without surgical intervention. Left paralumbar fossa laparotomy and rumenotomy can be used to identify adhesions in the vicinity of the reticulum, reticular or hepatic abscesses, or obstruction of the omasal canal. Removal of foreign bodies, wires, and some masses at surgery and lancing of perireticular abscesses into the reticulum affords a fair to good prognosis. A diagnosis of lymphosarcoma at surgery warrants a grave prognosis. Reticular abscesses identified at surgery should be cautiously drained into the reticulum, and antibiotics given for 10–14 days. Reportedly, 83% of cattle with reticular abscesses respond favorably to treatment. Identification of adhesions in the vicinity of the reticulum warrants a fair to good prognosis with surgery, antibiotic therapy, and appropriate supportive treatment. Hepatic abscesses must be drained by a second surgery. Large-bore cannulas placed through the body wall, through the adhesions, and into the abscess will drain the purulent material. However, recurrence is more of a problem with hepatic abscesses than with reticular abscesses.

Animals with type III vagal indigestion (secondary abomasal impaction) diagnosed without surgery usually do not receive further treatment because of the poor prognosis, particularly if there is a history of traumatic reticuloperitonitis or abomasal volvulus. If the diagnosis is made at surgery or if the abomasal impaction is thought to be dietary, dioctyl sodium sulfosuccinate can be infused directly into the abomasum via the reticulo-omasal orifice after emptying the rumen. A nasogastric tube can be passed into the abomasum via the reticulo-omasal orifice at surgery and left in place for continued treatment (3–4 L of mineral oil daily for 3–5 days). If possible, impacted material should be removed manually through the reticulo-omasal orifice. Other lesions, such as abscesses in the medial wall of the reticulum, should be identified and drained. Abomasotomy and removal of abomasal contents, using a right paracostal approach with the cow in left lateral recumbency, can be performed as a last resort. However, recurrence of the impaction is common. Pyloric obstruction in cattle is rare and is most often due to a foreign body obstructing the lumen. Pyloromyotomy is almost never effective in resolving abomasal impactions.

Type III vagal indigestion has a poor prognosis regardless of the cause or the treatment. However, cattle with mild to moderate primary abomasal impactions will respond to therapy, although severely affected animals will not (see Dietary Abomasal Impaction). Cattle with secondary impactions due to traumatic reticuloperitonitis or as a sequela of abomasal volvulus seldom recover. Animals with foreign bodies (eg, trichobezoars) obstructing the pylorus have a good prognosis if the obstruction is removed.

Therapeutic induction of parturition has been recommended for treatment of cattle with type IV vagal indigestion (indigestion of late gestation), and some cows have improved with this treatment; however, because type IV vagal indigestion is a poorly defined condition, the prognosis is always guarded. A more specific prognosis is based on response to therapy and identification of a specific lesion at exploratory celiotomy and rumenotomy.

Prevention:

The most common cause of vagal indigestion syndrome is traumatic reticuloperitonitis, which causes adhesions and abscesses that interfere with both reticular motility and the appropriate stratification of feed particles for passage through the abomasum. Therefore, prevention of traumatic reticuloperitonitis is important. Good management practices may prevent some cases of vagal indigestion associated with chronic pneumonia. Early diagnosis of abomasal volvulus, with same-day surgical correction, may prevent some cases.