Disorders of Rumen Distension and Dysmotility

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KEYWORDS
- Vagal indigestion
- Rumen motility
- Abdominal distension

KEY POINTS
- Rumen distension and hypomotility are common clinical signs that are found together.
- The location of abdominal distension and consistency of rumen contents provide key information for determining the cause of abdominal distension.
- Serum chloride and bicarbonate concentrations and rumen chloride concentration allow for differentiation of type 2 and type 3 vagal indigestion.
- Rumenotomy or right flank exploratory surgery can be both diagnostic and therapeutic.

INTRODUCTION
Rumen distension and dysmotility are not uncommon presentations in both cattle and small ruminants. These clinical signs often are linked, as dysmotility can lead to rumen distension and distension can lead to dysmotility. Identifying the underlying cause of the distension and dysmotility and determining if it is truly of gastrointestinal origin is critical to appropriate treatment. Generally, a thorough physical examination combined with some routine diagnostics can accurately identify the reason for rumen dysfunction, and guide appropriate treatment and prognosis.

NORMAL RUMEN CONTOUR AND MOTILITY
Examination of rumen shape, fill, and motility should be a part of the physical examination on all ruminants. Assessment of abdominal shape and rumen fill provides crucial information on feed intake and potential causes of distension. Decreased rumen motility can be a sensitive indicator of disease, although not specific, as many inflammatory processes and increased sympathetic tone will decrease normal rumen motility.1

Abdominal and Rumen Contour
Assessment of abdominal shape is preferably done early in a physical examination while observing a cow from a distance. While standing directly behind the cow,
determine if the cow’s abdomen appears gaunt, normal, or distended. Abdominal shape is not entirely dictated by rumen shape, but rumen size is the most common reason for abnormal distension. Abnormalities identified at this time can be useful in guiding a more thorough examination of the forestomach during the remainder of the physical examination. Nonetheless, practitioners must remember that other conditions, including intestinal distension, peritoneal effusion, pathologic accumulation of uterine fluid, and rupture of the prepubic tendon can affect abdominal shape and must be considered.

In a normal cow or small ruminant, the abdomen should be slightly wider than the stifles bilaterally. Typically, it will be somewhat symmetric, although slight differences from right to left are not uncommon. The most prominent distension on the left in a normal cow is typically around the level of the stifle in the mid abdomen due to fiber accumulation in the rumen. On the right, the normal shape is a slight enlargement below the stifle due to the small intestine.

The rumen should be palpated in the left paralumbar fossa and rectally. The normal rumen stratification can be identified on physical examination. There should be a gas cap in the caudodorsal rumen, a fiber mat throughout most of the rumen, and fluid ventrally. The gas cap, found dorsally, is softer and will immediately return to its previous shape when compressed. The doughy fiber mat is the most easily distinguished layer on palpation, as one can press into the rumen wall and leave an indentation when it is palpated rectally. On palpation through the flank, the fiber mat simply feels firm. The fluid layer is found in the ventral left flank. This area is softer than the fiber mat, but ballottement of this area is difficult due to the weight of the rumen contents.

**Normal Rumen Motility**

Rumen motility should similarly be evaluated as a part of the physical examination of all ruminants. Simultaneous auscultation and palpation in the left paralumbar fossa will allow the examiner to assess the frequency and strength of rumen contractions while also hearing any abnormal sounds associated with the contraction. The normal rate is 1 to 3 contractions per 2 minutes. Each contraction should be strong enough to lift the examiner’s hand on the paralumbar fossa. The sound should grow louder and then softer as the fiber mat turns inside the rumen and brushes along the rumen wall. There should not be any splashes or bubbling sounds associated with the contraction. This assessment of rumen motility measures the contraction rate of the dorsal rumen sac, and does not differentiate primary versus secondary contraction, as the dorsal sac will contract with both patterns. In most cases, simply determining the overall rumen contraction rate is adequate.

Primary contractions are mixing contractions in which the fiber mat is turned in the rumen to ensure that feed material is mixed with the microbial flora contained in the rumen fluid. These contractions occur approximately 1 to 2 times per 2 minutes. Primary contractions are initiated at the reticulum with a biphasic contraction of the reticulum. These contractions can be ausculted at the seventh to eighth intercostal space, just caudal to the elbow on the left side or visualized by ultrasound caudal to the xiphoid and left of midline. The first reticular contraction is smaller, whereas the second contraction completely collapses the reticular lumen. From there, the contraction moves caudally and dorsally as the dorsal sac contracts. This is followed by contraction of the ventral sac and finally by contraction of the cranial sac to complete the cycle. This pattern effectively mixes the fiber suspended in the mat with the liquid in the ventral aspect of the rumen, allowing the bacteria to attach to the undigested fiber. This furthers digestion and increases fermentation. Primary contractions also cause fluid outflow through the omasal canal as the reticular contractions create...
negative pressure in the canal causing fluid and fine feed material to be aspirated through the omasal orifice.\(^5\)

Primary rumen contractions are controlled centrally at the dorsal vagal nucleus in the brainstem. Afferent fibers travel via the vagus nerve to the forestomach and allow for coordinated, regular primary contractions. Without this control, contractions become uncoordinated and insufficient to provide adequate mixing or allow for the removal of fluid and feed material from the rumen.\(^1\) Normally the rumen has 1 to 2 primary contractions per 2 minutes.\(^6\) Moderate rumen distension, as would be found after a recent meal, increases primary contraction rate due to stimulation of stretch receptors in the rumen wall, whereas severe pathologic distension will stop rumen contractions.\(^7\) Abomasal distension will also reduce primary contractions, presumably to decrease the flow of ingesta through the omasal canal into the already overfull abomasum. Stimulation of buccal receptors in the mouth during feeding will also increase contraction rate, while chemoreceptors in the rumen epithelium monitor pH, and will stop rumen contractions if the pH drops below 5.0. Additional inhibitors of primary contractions are systemic disease and increased sympathetic tone (eg, pain, fear).\(^1\)

Secondary contractions are defined as those that cause eructation, although this distinction is less clear in reality, as ruminants can eructate with primary contractions and do not always eructate with each secondary contraction. Further, secondary contractions have been defined differently by various researchers. Some view them as a standalone motility pattern,\(^8\) whereas others have described them as an additional contraction cycle superimposed on a primary, mixing contraction.\(^4\) Again, a strict definition here is likely inaccurate, as it appears that these patterns are not nearly as fixed as one would like, as different methods of recording contractions and species differences (sheep vs cattle) further muddy the characterization of contraction patterns.\(^9\) Nonetheless, secondary contractions typically involve a contraction of the cranial pillar that holds ingesta back in the caudal sac, while a wave of contraction moves cranially across the dorsal sac, which pushes the gas cap forward.\(^10\) This clears the cardia of fluid, which allows it to open, leading to eructation.

Secondary contractions typically occur following every other primary contraction, leading to 1 secondary contraction every 2 minutes.\(^6\) Assessment of secondary contractions can be done by simultaneously listening to the reticulum and feeling rumen contractions in the left flank. As mentioned previously, primary contractions are felt immediately after a reticular contraction. Secondary contractions will be felt without an associated reticular contraction. The primary driver of secondary contractions is the rate of gas production and subsequent distension of the dorsal sac of the rumen.\(^10\) Inhibition of secondary contractions is due to excessive distension and sympathetic tone. Eructation can be prevented even if the motility pattern is normal if the cardia is not able to be cleared of fluid.\(^11\) This can occur if the animal is laterally recumbent or there is froth in the rumen.\(^12\) Damage to the epithelium near the cardia from rumenitis can inhibit the ability of the cardia to sense the presence of gas, and subsequently prevent it from opening.

**PHYSICAL EXAMINATION**

*Abnormal Rumen Contour and Motility*

**Abnormal abdominal and rumen shape**

Finding that the cow’s abdomen is narrower than her stifles suggests prolonged anorexia, as completely emptying the rumen can take several days. Although specific in identifying a significant and prolonged decrease in feed intake, a gaunt abdomen provides little guidance as to the underlying problem.\(^2\)
If a cow is found to have a distended abdomen, first characterize the location of the distension, the organ leading to abdominal distension, and determine if the distension is due to the accumulation of gas, fluid, or feed material. The distension is most commonly found in the mid abdomen and dorsally on the left, ventrally on the right, dorsally on the left and ventrally on the right, or ventrally bilaterally. Other locations (ie, just ventrally on the left or just dorsally on the right) are less common due to the abdominal anatomy of ruminants.3

Once the distension is localized, ballottement of the abdomen and rectal palpation can be used to determine the organ or organs leading the change in abdominal shape and whether the abnormal distension is due to gas, fluid, or feed material. Based on the location and type of distension, the veterinarian can then develop a relatively short differential diagnosis list (Table 1).

Unilateral distension on the left side is almost always due to enlargement of the rumen. Palpation of the rumen at paralumbar fossa and rectally will allow practitioners to determine the reason for the distension. Excessive gas will accumulate dorsally, and will feel like a large balloon (Fig. 1). This is consistent with a type 1 vagal indigestion (failure of eructation). Excessive fluid distension of the left side is consistent with a rumen acidosis and the subsequent fluid shifts that occur due to osmosis. Early type 2 vagal indigestion (failure of rumen outflow) cases may have fluid distension only on the left, but most commonly they are distended bilaterally. An enlarged, doughy rumen is consistent with a feed impaction due to poor-quality feedstuffs or inactivity of the rumen microorganisms. Additional information on these disorders is provided later in this article and elsewhere in this issue.

Distension ventrally on the right side is most commonly either fluid or feed. If the distension is due to fluid, the most likely reasons are type 3 (failure of abomasal outflow) or 4 (failure of pyloric outflow) vagal indigestion or small intestinal distension. With type 3 or 4 vagal indigestion, the abomasum initially becomes distended and then, ultimately, the rumen becomes distended as well. Therefore, most of the animals present with bilateral distension. Cattle with small intestinal distension, on the other hand, may not have rumen distension, as they often present with signs of abdominal pain due to the stretch of the intestinal wall earlier in the disease process. Feed distension on the lower right side is consistent with an abomasal impaction.

Bilateral distension most commonly occurs due to fluid accumulation in the rumen or rumen and abomasum (Fig. 2). As fluid is trapped in the rumen, it initially distends on the left in the midflank. Over time, the ventral sac of the rumen expands greatly toward the right such that there is now distension of both sides. If there is a type 3 vagal indigestion, distension of the abomasum will contribute to the ventral, right-sided distension, and eventually fluid will back up into the rumen and cause the left-sided

<table>
<thead>
<tr>
<th>Type of Distension</th>
<th>Dorsal Left</th>
<th>Ventral Right</th>
<th>Dorsal Left and Ventral Right</th>
<th>Ventral Bilaterally</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gas distension</td>
<td>Type 1 vagal indigestion</td>
<td>Uncommon</td>
<td>Uncommon</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Fluid distension</td>
<td>Rumen acidosis</td>
<td>Type 3 or 4 vagal indigestion, small intestinal distension</td>
<td>Type 2 or 3 vagal indigestion</td>
<td>Peritoneal effusion, hydrops conditions</td>
</tr>
<tr>
<td>Feed material</td>
<td>Rumen impaction</td>
<td>Abomasal impaction</td>
<td>Uncommon</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>
Fig. 1. Dorsal distention of the left flank of a cow consistent with a type 1 vagal indigestion.

Fig. 2. Asymmetric, bilateral distension of a steer (A) and goat (B) consistent with type 2 or 3 vagal indigestion.
distension. In either case, the distension on the left is more diffuse and located in the middle to dorsal region of the flank (“apple” shaped), whereas the distension on the right is in the ventral flank (“pear” shaped). This combination leads to the description of these cows as “papple” shaped due to their asymmetric bilateral distension.

Bilateral ventral distension is generally due to fluid accumulation in the abdomen or uterus, and therefore, rarely gastrointestinal in origin (Fig. 3). Differentials for these animals include pathologic accumulations of fluid in the uterus due to placental or fetal abnormalities, peritoneal effusion, or uroabdomen. Appropriate history, rectal palpation, and abdominoceutesis can be used to differentiate these, but this is beyond the scope of this article.

Abnormalities of rumen motility
Hypermotility of the rumen is a relatively uncommon finding, although in actuality it likely occurs quite frequently. In cases of early rumen distension, hypermotility may be noted as the moderate stretch receptors in the rumen wall are stimulated. The rumen continually senses this distension as a recent meal, and increases the rate of primary contractions in response to this distension. Therefore, in most cases of pathologic rumen distension, there is an early phase associated with rumen hypermotility.13 Due to the early nature of the disease course and mild distension, it is unusual for an owner to present an animal for examination at this stage, and the hypermotility is missed. As the distension increases, the severe stretch then stops rumen contractions, and it is at this stage at which animals are typically examined.

Hypomotility is a much more common finding in clinically ill ruminants. As mentioned previously, systemic inflammation or increased sympathetic tone from a variety of causes will decrease rumen motility. Hence, most cases of rumen hypomotility are from causes outside the rumen. A thorough physical examination is necessary to rule out other causes of decreased rumen contractions. Hypomotility due to rumen diseases are most commonly associated with rumen distension or rumen acidosis. When the rumen is severely distended, rumen contraction rate will slow down and ultimately stop. Some disorders, traumatic reticuloperitonitis for example, may first disrupt normal motility, leading to rumen distension, which then further slows the contraction rate. Other disorders, such a physical obstruction of the omasal canal, lead to a primary rumen distension, and the distension ultimately slows and stops rumen contractions. This distinction is important prognostically, as cases with primary

Fig. 3. Bilateral ventral distension of the abdomen of a cow with hydrops.
motility disorders are less likely to return to productivity after relieving the distension and underlying problem, whereas those with hypomotility due to distension are more likely to return to normal function after relieving the distension.

**Disorders associated with rumen distension and dysmotility**

Ruminants with both rumen distension and dysmotility typically are diagnosed with vagal indigestion, although rumen acidosis and rumen impactions also should be considered depending on the animal’s abdominal shape, rumen fill, and dietary history. In spite of the name, clinical cases of vagal indigestion have been repeatedly shown to not involve the vagus nerve in most cases. Further, Hoflund’s original description\(^\text{14}\) of the disease based on experimental transection of the vagus nerve does little to guide diagnostic and therapeutic decisions. The classification scheme of 4 types of vagal indigestion by Ferrante and Whitlock\(^\text{15}\) provide a more clinically useful approach to understanding these diseases and will be used here. No matter the underlying cause, the disease typically progresses from mild rumen distension leading to hypermotility, then progressive distension causes rumen hypomotility. At this point, the animal usually presents with severe rumen distension, decreased rumen contraction rate, and anorexia.

Type 1 vagal indigestion is associated with a failure of eructation. These animals present with gas distension of the dorsal left flank, and rumen hypomotility. This can occur due to a failure of secondary contractions, an inability to clear the cardia of fluid, failure of the cardia to open, or esophageal obstruction. A loss of secondary contractions appears to be relatively rare, although this may play a role in the bloat that can be seen in some calves with chronic respiratory disease. It is hypothesized that the vagus nerve can become inflamed in the thorax secondary to the respiratory disease. Bloat that is seen within laterally recumbent ruminants is due to fluid flooding the cardia, in spite of normal rumen motility. Similarly, the froth that can be created from consumption of legumes is sensed as fluid at the cardia, and prevents eructation. Damage to the rumen epithelium in the area of the cardia from rumenitis can damage the receptors responsible for sensing the presence of gas at the cardia, allowing it to open for eructation. Obstruction of the esophagus can occur from an intraluminal obstruction (swallowing an apple), an extraluminal mass (tracheobronchial lymphadenopathy in cases of respiratory disease), or a mass at the cardia (papilloma). Note that in all of these cases, the distension arises from a failure to eructate, not from an increased rate of gas production. Even with significant gas production from fermentation, the normal ruminant can increase eructation adequately to eliminate the gas.

Animals with type 2 vagal indigestion present with bilateral distension of the abdomen due to fluid accumulation in the rumen. The abdomen is distended at the midflank and dorsally on the left and ventrally on the right. On rectal examination, the classic finding of an “L”-shaped rumen is felt due to the significant expansion of the ventral sac toward the right flank. The fluid accumulation arises from a failure of rumen outflow with continued food and water intake and saliva production. The obstruction of the omasal orifice can be either functional or mechanical. Functional failures are most commonly due to traumatic reticuloperitonitis leading to inflammation and adhesions around the reticulum. Without normal reticular contractions, primary contractions are disrupted, and fluid is not aspirated into the omasal canal.\(^\text{16}\) Other causes of peritonitis in the cranial abdomen including liver abscesses may present similarly.\(^\text{17}\) Mechanical obstructions can occur secondary to consumption of a foreign body, including rope, hay netting, or placenta.\(^\text{18}\) Masses including fibropapillomas and other neoplasias can also obstruct outflow.\(^\text{19}\) In these cases, primary
contractions are not disrupted initially, and they serve to maintain the foreign body lodged in the omasal orifice. Once the rumen becomes overly distended, then the rumen contractions stop.

Type 3 vagal indigestion presents similarly to type 2 in that the animal has the classic “papple” shape and fluid distension of the rumen. The difference is that the distension is due to a failure of abomasal motility and outflow. Reflux of abomasal fluid leads to the rumen distension, and the abomasum and rumen both contribute to the abdominal distension that is seen externally. The combination of abomasal and rumen distension leads to rumen hypomotility. Like type 2 vagal indigestion, type 3 also can be due to a functional or mechanical failure of abomasal motility. Functional causes include abomasal lymphosarcoma, traumatic reticuloperitonitis, and abomasal damage after an abomasal volvulus. Roughly 15% of cattle with an abomasal volvulus will go on to develop abomasal motility disorders. This appears to be due to ischemic damage to the abomasal wall, peritonitis, and/or damage to the vagus nerve. Mechanical obstructions here are less common, although lymphosarcoma and feed or sand impactions can also physically disrupt pyloric outflow. Iatrogenic causes should be considered, including inappropriately placed pyloropexy or incorrect placement of a toggle suture.

Type 4 vagal indigestion is a less well defined syndrome of partial pyloric obstruction or generalized ileus. These animals have less abdominal distension compared with those with type 2 or 3 vagal indigestion. A common reason for this presentation is late-term pregnancy, as the fetus may physically impede pyloric outflow or proximal intestinal motility. Other causes are related to severe systemic disease, including hypocalcemia, peritonitis, septicemia, and enteritis leading to reduced intestinal motility.

Rumen acidosis is more thoroughly discussed in Nathan F. Meyer and Tony C. Bryant’s article, “Diagnosis and Management of Rumen Acidosis and Bloat in Feedlots,” in this issue, but is worth mentioning here as another cause of rumen distension and hypomotility. Due to the rapid production of volatile fatty acids from grain fermentation that exceeds the absorptive capacity of the rumen, water is pulled by osmosis into the rumen. This accumulation of fluid in the rumen causes a left-sided abdominal distension that may initially appear similar to a type 2 or 3 vagal indigestion. The abnormally low pH of the rumen fluid stops rumen contractions as the rumen attempts to slow fermentation. These animals with rumen acidosis will typically be more depressed and dehydrated than those with vagal indigestion, and examination of the rumen pH allows for easy differentiation of these diseases.

Animals with a rumen impaction will present with a firm, left-sided abdominal distension due to feed accumulation in the rumen. Rumen contraction rate will be variable depending on the degree of distension, and could range from increased to absent. The underlying pathogenesis of this disease could be either a lack of appropriate rumen microbial populations or feeding a low-quality, largely indigestible forage. The former can be seen in young animals who begin consuming large amounts of forage before developing a functional rumen or in an adult animal who has lost the normal rumen bacterial population after acidosis, anorexia, or antimicrobial administration. When fed indigestible forage, the rumen bacteria cannot adequately break down the plant material or the fermentation is excessively slow. This leads to an accumulation of fiber within the rumen as the animal continues to consume a large volume of feed material, yet cannot meet its nutritional needs. Hence, in chronic cases, animals will present with severe rumen distension but extremely poor body condition. The severe weight loss may be overlooked by owners due to the animal’s large abdomen.
Diagnostic Approach to Animals with Rumen Distension and Dysmotility

History and physical examination
When examining an animal with rumen distension and dysmotility, a complete physical examination will generally provide practitioners with a reasonably short list of differentials that can be further assessed with minimal diagnostic testing. Before examining the animal, it is useful to gather an appropriate nutritional and housing history. How much grain is fed? What is the quality of forage that is provided? Any exposure to legumes? Recent construction or building of fences? Evidence of trash or other potential foreign bodies in the pasture or animal’s enclosure? Has the animal had a recent abomasal volvulus, pyloropexy, or toggle procedure? Then the animal is observed before restraint to properly assess abdominal contour, as described previously.

Rumen contraction rate and strength should be assessed by auscultation of the left paralumbar fossa. Most of these animals will have few or no rumen contractions. If the animal does have some contractions, simultaneous auscultation of the reticulum with palpation of the rumen will determine if the contractions are primary or secondary contractions. During the examination, particular attention should be paid to those potential diseases that can lead to vagal indigestion. A withers pinch should be performed. A lack of response could be due to any cause of cranial abdominal pain, although traumatic reticuloperitonitis is the classic disease associated with this finding. Other considerations include a ruptured liver abscess or a perforating abomasal ulcer. Practitioners may get some indication of the underlying problem if the cow responds more severely to sternal pressure on the right or left, as traumatic reticuloperitonitis will typically cause more pain on the left, whereas other causes are more likely located on the right. On auscultation of the thorax, is there evidence of respiratory disease or muffling of the heart associated with traumatic reticulopericarditis? Is there any lymphadenopathy that might be suggestive of lymphosarcoma? On rectal examination, the rumen size and texture is assessed to determine if there is fluid distension of the ventral sac. Also, the pregnancy status of the animal is determined, internal lymph nodes are palpated, and the viscera are palpated for evidence of peritonitis and adhesions.

Ancillary diagnostic testing
Rumen fluid analysis
After completing the physical examination, passing a stomach tube is valuable diagnostically and therapeutically. In many cases of type 1 vagal indigestion, gas will be released when the tube is passed. With type 2 or 3 vagal indigestion, fluid may spontaneously reflux from the tube. If not, fluid should be siphoned off the rumen to reduce the distension and provide a sample for diagnostic evaluation. On collection of the fluid, the pH should be evaluated to rule out rumen acidosis. In cases of vagal indigestion, the pH will be normal (5.5–7.0) or slightly alkaline due to anorexia. The reflux of abomasal fluid with type 3 vagal indigestion is not sufficient to reduce rumen pH out of the normal range. When collecting rumen fluid orally, it is critical to collect several hundred milliliters of fluid to minimize the impact of saliva contamination on the pH. Excessive saliva contamination in a small volume sample will artificially elevate the pH due to the buffering capacity of ruminant saliva. A drop of the fluid should be placed on a microscope slide and evaluated at low magnification to assess protozoal activity. There should be numerous protozoa of varying sizes rapidly moving across the field. This can be used as a proxy measure of general microbial activity, as the protozoa appear to be more susceptible to changes in the rumen environment. In particular, the larger Holotrich protozoa appear to be especially sensitive to changes in the rumen environment. Acidosis or prolonged anorexia in vagal indigestion are the most common causes of decreased
protozoal numbers. This assessment needs to be done relatively rapidly, as these pro-
toza can be quite susceptible to changes in temperature and exposure to oxygen. Bacterial populations can be further investigated by Gram staining a sample of fluid, and measuring the methylene blue reduction time.

A sample of rumen fluid also should be strained for measurement of chloride con-
tent. In normal rumen fluid, the chloride content should be less than 30 mEq/L. Abomasal outflow obstructions (type 3 vagal indigestion) cause an increase in rumen chloride as the chloride secreted into the abomasum refluxes back into the rumen. It remains sequestered there due to the rumen epithelium’s relatively poor ability to absorb electrolytes. This finding is quite useful in differentiating type 2 and type 3 vagal indigestion, as they often present similarly. It has been demonstrated that acetate in the rumen fluid can falsely elevate chloride measurement when assessed using routine potentiometric blood chemistry analysis. This interference is of less concern in ani-
mals with anorexia, as the acetate levels will be lower. Further, a chloride level less than 30 mEq/L can be reliably interpreted as normal, whereas an elevated rumen chlor-
ide concentration could be due to abomasal reflux or increased acetate levels. There-
fore, it is critical to interpret rumen chloride concentrations in concert with blood chemistry analysis (Table 2).

**Blood Chemistry Analysis**

Assessment of serum chloride and bicarbonate can be useful in distinguishing be-
tween type 2 and 3 vagal indigestion for similar reasons as rumen chloride. Reflux
of the chloride and subsequent sequestration in the rumen leads to a severe hypo-
chloremia as the chloride is normally reabsorbed in the duodenum. Similarly, the
hydrogen ions secreted into the abomasum to acidify the contents are associated
with bicarbonate moving into the bloodstream. Normally, the bicarbonate from the
bloodstream is then taken by the duodenum to neutralize the abomasal pH when
ingesta enters the proximal small intestine. When this flow is disrupted, a severe meta-
bolic alkalosis occurs as the bicarbonate remains in the circulation. Hence, animals
with a type 3 vagal indigestion will have a severe hypochloremic metabolic alkalosis.
Those with other rumen motility disorders may have similar electrolyte and acid-base
derangements, but not to the same degree. The hypochloremic, metabolic alkalosis in
these cases is associated with reduced abomasal motility due to anorexia and sys-
temic disease. Other findings on the blood chemistry analysis also can be instructive,
as an increase in globulins would suggest a chronic inflammatory process, such as
traumatic reticuloperitonitis.

**Ultrasound of the Reticulum**

To definitively identify reticular contractions, it is helpful to use ultrasound to visualize
the reticulum as auscultation can be difficult, and does not let one evaluate the
strength of the reticular contraction. The reticulum can be identified to the left of

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Rumen fluid analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>Yellow-green to olive green depending on diet</td>
</tr>
<tr>
<td>pH</td>
<td>5.5–7</td>
</tr>
<tr>
<td>Protozoal activity</td>
<td>Abundant protozoa of different sizes</td>
</tr>
<tr>
<td>Methylen blue reduction</td>
<td>&lt;5 min</td>
</tr>
<tr>
<td>Chloride</td>
<td>&lt;30 mEq/L</td>
</tr>
</tbody>
</table>

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midline, just caudal to the xiphoid. It will appear as a U-shaped structure, and only the wall can be seen due to the gas mixed into the ingesta. The cranial sac of the rumen will appear just caudal to the reticulum. The reticulum will have a biphasic contraction in which the first contraction is smaller, and the second completely collapses the reticular lumen as it moves dorsally. Identification of normal reticular contractions in cases of rumen distension suggests that the problem is less likely a functional motility disorder of the forestomach. A lack of reticular contractions, on the other hand, may suggest either a primary motility disorder or hypomotility due to rumen distension. Interestingly, many animals with rumen hypomotility will have reticular hypermotility, and this was particularly pronounced in cases of type 2 vagal indigestion. Further, imaging of this area can identify abscesses, adhesions, or fluid accumulation associated with traumatic reticuloperitonitis.

**Rumenotomy/Abdominal Exploratory**

Abdominal surgery may ultimately be necessary to accurately diagnose the underlying disease in animals with rumen distension and dysmotility. This has the advantage of being both diagnostic and therapeutic. Before surgery though, one must determine if the animal most likely has a type 1 or 2 vagal indigestion versus a type 3 or 4. This distinction is important, as surgical diagnosis and correction of type 1 and 2 vagal indigestion is best accomplished through a left flank celiotomy and rumenotomy, whereas type 3 and 4 problems are best addressed from a right flank celiotomy and exploratory (Table 3).

**TREATMENT OPTIONS**

Treatment of the most these disorders associated with rumen distension and dysmotility are more thoroughly addressed in Robert J. Callan and Tanya J. Applegate’s article, “Temporary Rumenostomy for the Treatment of Forestomach Diseases and Enteral Nutrition,” Nathan F. Meyer and Tony C. Bryant’s “Diagnosis and Management of Rumen Acidosis and Bloat in Feedlots” and Matt D. Miesner and Emily J. Reppert’s “Diagnosis and Treatment of Hardware Disease,” in this issue, and will commonly require a rumenotomy or abdominal exploratory as discussed previously. A few principles of therapy applicable to any of the previously discussed disorders are discussed as follows.

<table>
<thead>
<tr>
<th>Type of Vagal Indigestion</th>
<th>Location of Abdominal Distension</th>
<th>Rumen Contents</th>
<th>Rumen Chloride</th>
<th>Serum Chloride</th>
<th>Serum Bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1: Failure of eructation</td>
<td>Dorsal left</td>
<td>Gas</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
<td>Normal to mildly increased</td>
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<tr>
<td>Type 2: Failure of rumen outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
<td>Normal to mildly increased</td>
</tr>
<tr>
<td>Type 3: Failure of abomasal outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Increased</td>
<td>Moderate to severely decreased</td>
<td>Moderate to severely increased</td>
</tr>
<tr>
<td>Type 4: Partial failure of pyloric outflow/proximal intestinal obstruction</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal to increased</td>
<td>Mild to moderately decreased</td>
<td>Mild to moderately increased</td>
</tr>
</tbody>
</table>
**Emergency Treatment**

Emergency treatment of severe rumen distension may be necessary even before complete evaluation. As the rumen becomes distended, the animal’s ability to breathe is reduced as the rumen impedes normal movement of the diaphragm. Passage of a large-diameter orogastric tube should always be one’s initial consideration, as this will allow passage of accumulated gas or fluid without the risk of peritonitis associated with rumen trocarization. A surfactant, such as poloxalene, can be administered at this time if there is any suspicion of a frothy bloat. Trocarization of the rumen can be performed in cases of extreme respiratory distress or if passage of an orogastric tube is not possible. A self-retaining, screw-in trocar is best, but no matter what type is used, owners should be made aware of the significant risk of peritonitis.

**Transfaunation**

For any of these diseases, there is likely to be an associated disruption of the rumen microbial populations. This disruption could be due to pH changes following rumen acidosis or due to prolonged anorexia in cases of vagal indigestion. Correction of the underlying cause of the motility disorder is key, but transfaunation with normal rumen fluid can speed the animal’s return to normal productivity by replenishing the microbial populations. Drenching an adult bovine via an ororuminal tube (or adding directly to the rumen during a rumenotomy) with 10 to 16 L fresh rumen fluid appears to be clinically effective. Similarly, transfaunation of 1 to 4 L fresh rumen fluid in sheep and goats can reestablish normal microbial populations.28

**SUMMARY**

Rumen distension and dysmotility (most commonly hypomotility) are often found together in clinical cases. A thorough physical examination to determine the location of the rumen distension, assess the rumen contents, and careful auscultation of rumen contraction patterns will commonly provide the examiner with a relatively short differential diagnosis list. From here, rumen fluid analysis, ultrasound of the reticulum, and blood chemistry analysis can further guide surgical planning. Based on these findings, the practitioner can then make an informed decision concerning the surgical approach: left flank rumenotomy for rumen acidosis, type 1, or type 2 vagal indigestion or a right flank exploratory for type 3 or 4 vagal indigestion.

**REFERENCES**

Disorders of Rumen Distension and Dysmotility


