**Vagal Indigestion Syndrome in Ruminants**

**(Chronic indigestion)**

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.