IS IT VOMITING OR REGURGITATION?  
GASTROENTEROLOGY

David C. Twedt, DVM, DACVIM

There are many gastrointestinal disorders that either frequently go unrecognized or are often misdiagnosed. Appropriate recognition and subsequent therapy may result in resolution of these disorders. The purpose of this lecture series is to provide important practice tips in the diagnosis of various upper gastrointestinal disorders associated with regurgitation and/or vomiting. In some situations, it can be difficult to differentiate between regurgitation and vomiting. If the cause of vomiting cannot otherwise be determined, one should investigate the esophagus as the possible source of the problem.

Regurgitation
Regurgitation is associated with the passive expulsion of esophageal contents and is not associated with retching. The majority of esophageal disorders are associated with motility; however, we are diagnosing more and more inflammatory lesions.

Esophageal Burn
Gastroesophageal reflux disease (GERD) is the result of esophageal mucosal having abnormal contact with refluxed gastric or duodenal fluid and/or ingesta, resulting in esophageal inflammation. The incidence of GERD in veterinary patients is unknown, but it is thought to occur much more frequently than is clinically recognized. The diagnosis of GERD is difficult and often requires endoscopy to demonstrate esophageal ulceration.

Factors responsible for damage to the esophagus include the character of the refluxed material, the competency of the esophageal clearing mechanism, the volume and frequency of the reflux, the time the refluxed material remains in the esophagus, and the integrity of the esophageal mucosal barrier. Common etiologies associated with reflux esophagitis in small animals include general anesthesia, hiatal hernia disorders, and persistent vomiting. Disorders of gastric motility or increased abdominal pressure may be also associated with GERD. Gastroesophageal reflux and hiatal hernias may occur from upper airway obstructions due to increased negative intrathoracic pressure. It appears to be quite common in brachycephalic breeds, presumably secondary to their respiratory disease, and signs often resolve following surgical correction of their upper airway disease.

Signs of GERD include salivation, licking of the lips, anorexia, and regurgitation or vomiting. The owners often report halitosis, salivation, and/or anorexia. Survey radiographs and contrast studies are often inconclusive; however, when reflux is suspected, applying pressure over the stomach filled with contrast may induce reflux that can be demonstrated on radiographs or with fluoroscopy. Endoscopy showing distal mucosal ulceration and a large open lower esophageal sphincter (LES) is consistent with GERD.

Rational therapy for GERD involves first treating the inciting etiology. Specific therapy is to increase LES pressure and protect the mucosa from damage. Therapy should begin with diet, by feeding small, frequent meals that are low in fat to maximize LES pressure and minimize gastric volume. Topical liquid sucralfate binds to ulcers, protecting the mucosa and promoting esophageal healing. Reducing gastric acid production is important. Because the esophagus’s lack of tolerance to acid, I suggest using the proton pump blocker omeprazole (Prilosec, 0.7–1 mg/kg q 12–24 hrs). Gastric prokinetic agents, such as cisapride (Prepulsid 0.1 mg/kg b.i.d. to t.i.d.) or erythromycin (0.1–0.5 mg/kg b.i.d. to t.i.d.), can be used to increase the LES pressure and gastric motility. Cisapride is obtained from compounding pharmacies and is my preference. Recent information suggests that metoclopramide (Reglan, 0.2–0.4 mg/kg t.i.d. to q.i.d.) has poor prokinetic properties.

When the Esophagus Doesn’t Work
“Megaesophagus” is a descriptive term referring to esophageal dilation secondary to an aperistaltic esophagus. In most cases the prognosis for megaesophagus is poor. There are a number of etiologies, however, to consider as a cause of megaesophagus in the dog. Congenital megaesophagus occurs in young dogs inherited or secondary to developmental abnormalities in esophageal innervations. It is inherited in the wirehaired terrier and schnauzer and there is a high incidence in Irish setters, German shepherds, golden retrievers, shar peis, and Great Danes. The prognosis is conflicting; rarely, some may have spontaneous improvement, but this is generally the exception. If there is no evidence of improvement by 3 or 4 months it is unlikely they will improve. Adult-onset idiopathic megaesophagus occurs spontaneously in dogs from 7 to 15 years of age with no specific sex or breed predisposition; though it is more frequent in larger breed dogs. The etiology is unknown, and the treatment is only symptomatic. I recommend upright feeding, which can be accomplished using a Bailey chair (websites provide specific
information). Dietary consistency is variable, and different diets should be tried to see what is best tolerated. Severe debilitation requires gastrostomy feeding tubes. Prognosis is variable, and poor with aspiration pneumonia. In 49 cases, 73% were dead or euthanized within several months of the diagnosis. A very small population of dogs seems to tolerate megaesophagus with minimal complications.

Secondary megaesophagus results from a number of etiologies that directly affect neuromuscular function, including myasthenia gravis (MG), adrenocortical insufficiency, systemic lupus erythematosus (SLE), polymyositis, dysautonomia, and immune-mediated polyneuritis.

**Focal myasthenia gravis (MG)** is associated with generalized weakness of only the esophagus. Both young and old dogs are reported to have focal MG, and the German shepherd and golden retriever seem to be the most frequently affected. The clinical presentation and initial diagnostic findings are similar to that of primary idiopathic megaesophagus. The diagnosis of MG is confirmed by identifying positive Acetylcholine (ACh) receptor antibodies. Approximately half of the dogs with focal myasthenia improve or eventually have remission of clinical signs. Anticholinesterase therapy with pyridostigmine bromide (Mestinon 0.5–1.0 mg/kg t.i.d. to b.i.d.) and/or immunosuppression is suggested. Refer to specific topics on therapy.

**Hypoadrenocorticism** has been reported as a cause of reversible megaesophagus in the dog. Animals may present as typical Addison’s or may be atypical only with megaesophagus. Measurement of cortisol levels pre- and post-ACTH stimulation confirms the diagnosis. We frequently do a baseline cortisol and if it is elevated (> 2 µg/dl) this rules out the disease. Adequate glucocorticoid and/or mineralcorticoid (if typical Addison’s) replacement results in rapid resolution of the megaesophagus, usually within a week of therapy.

**Polymyositis** is possible but difficult to diagnose. Systemic involvement of skeletal muscles and/or elevated creatine kinase (CK) concentrations supports a diagnosis. If all is ruled out I will sometimes try a course of steroids if aspiration is not evident and other diagnostics are negative. I have been pleasantly surprised having some dogs improve.

**Dysautonomia** is a disorder identified in the dog as the result of degenerative changes involving neurons of the autonomic nervous system. The signs of disease are associated with dysfunction of the autonomic nervous system. In addition to megaesophagus and regurgitation, other autonomic disorders include dilated pupils, dry eyes, nictitans protrusion, dilated anal sphincter, distended urinary bladder, fecal and urinary incontinence, and delayed gastric emptying. Dogs with this disorder are usually young, large-breed dogs from a ranching or farm environment. The prognosis for most cases is very guarded.

**A Hard Pill to Swallow**
Esophageal strictures result following deep submucosal ulceration with subsequent fibrosis. In a review of 23 cases, anesthesia-related gastric reflux occurred in 65%; 9% of the cases were associated with foreign bodies; and the remainder were due to other causes, such as pill-associated problems, trauma, or esophageal tube placement. The association of anesthesia and gastroesophageal reflux occurs in 10% to 15% of dogs having anesthesia. If stricture formation occurs, it is approximately 1 to 2 weeks following the anesthesia episode. Animals regurgitate solid food but are able to hold down liquids, and regurgitation is usually immediately following eating. We reported a number of cats developing esophageal strictures secondary to doxycycline tablets occurring in the cervical esophagus. Doxycycline and NSAIDs are the most common drugs to result in stricture formation in humans. Recent studies in our laboratory showed that pills given to cats on a dry swallow are delayed in passage through the esophagus, but if the pill is given with 3–6 ml of water, it will pass into the stomach.

The treatment of esophageal strictures involves either feeding the patient a liquid diet or administering therapy using balloon dilation. Balloons of increasing size are placed in the stricture to mechanically dilate the lumen of the esophagus. Therapy is then directed at management of reflux esophagitis (see below) and steroids to lessen re-stricture formation (triamcinolone injection in the stricture citrix). In a review of 23 cases, 84% were considered to have a good outcome following a mean of three separate balloon dilations performed at weekly intervals.

**Esophageal Foreign Body**
The most common esophageal foreign bodies are bones, but stones, chews, wood, and toys are also common. Terrier dogs are overrepresented with bone foreign bodies, and most of these obstruct at the level of the distal esophagus. The next most common type obstructs at the base of the heart, and the least common at the thoracic inlet. Signs of
acute regurgitation should make an esophageal foreign body high on the differential list. Following diagnosis and routine and/or contrast radiographs, prompt removal is required. The longer the foreign body remains in the esophagus, the greater the mucosal damage, with secondary complications such as strictures or perforation. Attempts should first be directed at conservative removal by either passing a gastric tube to dislodge the foreign body, Foley-catheter-assisted removal, or esophagoscopy. We will use either a rigid or fiberoptic endoscope to remove most foreign bodies. The disadvantage of flexible GI endoscopic removal is the small size of most foreign-body-grasping instruments. Removal of large foreign bodies, such as bones, often requires heavier, rigid, pronged grasping forceps. These can be adjacent to the flexible endoscope or through a rigid, hollow esophageal scope. The advantage of using a rigid, hollow endoscope is that it will both mechanically dilate the esophagus and allow one to pass large, grasping forceps through the center of the endoscope tube to retrieve the foreign body. The foreign body, once grasped, can be pulled into the scope for easy removal. One can also make one’s own esophageal scope by purchasing plastic polyvinylchloride (PVC) pipe of various sizes and lengths. A strong light is then used to illuminate the esophagus through the tube. Pronged grasping forceps can also be purchased from most hardware or auto stores. These are used for picking up small objects in difficult to reach areas and will work for grabbing bones and other foreign bodies. When large distal esophageal bones can’t be retrieved orally, one should attempt to push them into the stomach, where they will eventually be digested.

Single barbed fishhooks attached to a line are easily removed by simply stringing the line through a rigid esophageal scope. The scope is then advanced through the line to the hook. Next the hook is pushed out of the esophageal wall with the end of the scope, and the line is tugged gently to pull the hook into the scope. Then everything is removed—“hook, line and sinker.”

**Vomiting**

When dealing with the vomiting patient, there are four key parts of the overall history: (1) determining whether the patient is actually vomiting; (2) obtaining a detailed vomiting history (including the duration, frequency, character, and association with eating or drinking); (3) obtaining a drug and diet history; and (4) identifying any other clinical signs associated with the vomiting. Vomiting of an undigested or a partially digested meal, especially when the vomiting occurs more than eight hours following eating (a point at which the stomach should usually be empty), suggests a possible gastric outflow obstruction or a condition causing gastric hypomotility. The presence of blood in the vomit, either as fresh "bright red" blood or digested blood that has a "coffee grounds" appearance, indicates gastrointestinal erosion or ulceration.

**Components of Vomiting**

Although vomiting is a complex and poorly understood event, it can be best described simply as a reflex act that is initiated by stimulation of the conceptualized “vomiting or emetic center” located in the medulla oblongata of the brain. The vomiting center can be activated through a humoral pathway, via blood-borne substances, or through various neural pathways. Vomiting is sometimes classified as either centrally mediated (from central nervous system [CNS] mediation) or peripherally mediated (from distant structures in the abdominal cavity). Understanding this concept helps the clinician formulate a differential list of causes.

Neural stimulation of the vomiting center can arise through vagal afferent, sympathetic, vestibular, or cerebrocortical pathways. Activation of peripheral receptors found throughout the body can stimulate these neural pathways. Disease or irritation of the gastrointestinal tract, other abdominal organs, or peritoneum can directly stimulate vomiting through vagal afferent pathways. The vomiting center can also be stimulated indirectly via humoral or blood-borne factors that activate the chemoreceptor trigger zone (CRTZ) located in the area postrema. In this area, the blood-brain barrier is limited, which allows the CRTZ to be exposed to chemical stimuli found in the circulation. Blood-borne substances that can stimulate the CRTZ include certain drugs, uremic toxins, electrolytes, osmolar and acid-base disorders, and a number of metabolic derangements. Drugs such as apomorphine, or cardiac glycosides and bacterial toxins, are examples. Evidence shows that vestibular stimulation in the dog passes through the CRTZ before activating the vomiting center. Motion sickness, inflammation of the labyrinth, or lesions in the cerebellum can result in vomiting via this vestibular pathway.

**Vomiting Workup**

In patients with chronic vomiting, laboratory diagnostics are indicated and should include a minimum database (complete blood cell count, biochemical profile, and urinalysis), fecal examination, and abdominal radiographs and/or abdominal ultrasound. If no obvious diagnosis is made, then an immediate in-depth diagnostic evaluation is required in cases of significant vomiting. Animals with mild signs and minimal debilitation should first undergo
dietary food trials and empirical treatment for gastrointestinal parasites. Adverse reactions to food result from either food allergies or food intolerances. Food trials include hypoallergenic diets (i.e., a novel antigen diet, gastrointestinal diet, a different premium diet, or a hydrolyzed diet) for a minimum of a two-week trial. If the patient is diet-responsive, then the diagnosis is made. There is no one ideal diet to use, and novel protein diets, hydrolyzed diets, or prescription diets are suggested as trials.

Parasites should also be considered and can be a cause of vomiting, including Giardia, ascarids, and hook and whipworms. They are usually diagnosed by using proper fecal examination techniques. Physaloptera spp. infection (the gastric worm) in dogs is uncommon but may be underestimated due to the difficulty of diagnosis. Prevalence rates range from 1% to 25% of stray dogs in the Midwest. The worm burden need not be large to cause clinical signs; in fact, it is not unusual to find only one or two worms causing chronic intermittent vomiting. The adults produce few eggs and do not float well using routine fecal flotation techniques. Diagnosis is most frequently made during endoscopy by viewing the parasite in the stomach or proximal duodenum. The author usually prescribes febendazole at 50 mg/kg daily for 3 to 5 days to eliminate gastrointestinal parasites from the differential list. An in-depth GI evaluation should be considered for the vomiting animal with significant or severe gastric or GI disease or in the patient that has failed to respond to adequate dietary or anthelmintic therapy. Ollulanus tricuspis, a gastric parasite, may be more common than previously thought and is a cause of chronic vomiting and unthriftiness in cats. The vomiting can include mucus, bile, or food, and multiple cats in a household may be affected. The parasite has a direct life cycle with oral-oral transmission; consequently, eggs are not passed into the feces or observed in the fecal examination. The diagnosis of Ollulanus tricuspis is made by examining the vomitus, using the microscope on low power to look for nematode parasites that are approximately 0.75 mm in length. Gastric fluid samples can be obtained by either endoscopy or by inducing vomiting to collect gastric fluid by administering the emetic xylazine (0.44 mg/kg IM). The parasite appears to be eliminated with pyrantel pamoate or febendazole (50 mg/kg/day for 3 to 5 days). Ollulanus tricuspis is reported to be associated with variable chronic inflammatory mucosal infiltrates. When the cause of vomiting is unknown, and since gastric parasites are a possible etiology, anthelmintic trial therapy should be considered. I usually prescribe febendazole (50 mg/kg daily for days) as my broad-spectrum anthelmintic therapy in both dogs and cats. We routinely use the equine liquid formulation.

Helicobacter. Helicobacter has been implicated as a cause of chronic gastritis in humans and more recently in cats and dogs. It is a spiral-shaped, gram-negative bacteria that appears to be resistant to the effects of low gastric pH by virtue of its ability to produce urease, which splits the urea to make a microenvironment with ammonia. Helicobacter spp. was first identified in humans (Helicobacter pylori) and subsequently identified in dogs and cats (Helicobacter felis, Helicobacter heilmannii, and others). Helicobacter is thought to be a normal inhabitant initially, becoming clinically evident and causing a lymphocytic gastritis only after years of exposure, altered immune status, and/or other factors.

Dogs and cats presenting for Helicobacter are usually older (> 6 years) and have chronic vomiting as part of their history. They tend to vomit gastric fluid and occasionally food. Weight loss, unthriftiness, and rarely diarrhea have been noted. I have only seen one cat having hematemesis and a gastric ulcer with Helicobacter. Failure to respond to symptomatic therapy would warrant a Helicobacter workup. The clinical diagnosis is based on identification of the organism on gastric biopsy using a silver or modified Giesma stain. A provisional diagnosis involves either gastric mucosal brush cytology showing many small spiral organisms in the mucus, or the presence of the organism in a gastric biopsy (done by incubating a small biopsy sample in a urea broth containing a pH indicator that demonstrates bacterial urease production by means of a color change). Several commercial test kits are available to detect urease-producing bacteria in biopsy samples, but routine microbiological urea culture tubes work well and are quite inexpensive.

Specific antibiotic therapy appears to eradicate the organism and resolve clinical signs in affected patients. Generally, a combination of antibiotics is required to eliminate the organism from the lumen, gastric mucus, and mucosal cells. Triple therapy using amoxicillin metronidazole and omeprazole is the norm. Recent studies suggest that acid blocking may not be required, and preliminary studies suggest the use of amoxicillin and clarithromycin (7.5 mg/kg b.i.d.) for two weeks to be effective as well. The amoxicillin-clarithromycin combination is the treatment I now use in dogs and cats.

Bilious Vomiting Syndrome. Disorders in motility can on occasion be responsible for GI signs including vomiting. Motility disorders are diagnosed by exclusion of inflammatory and obstructive disease, and specialized contrast studies may help support the diagnosis. One condition thought to be associated with abnormal motility is the clinical
entity referred to as bilious vomiting syndrome, or reflux gastritis. The reflux of duodenal fluid (bile) into the gastric lumen can be responsible for gastric mucosal irritation and erosions. Reflux may in part be associated with abnormal gastric motility and the inability of the empty stomach to maintain intragastric pressure that is greater than duodenal pressure. This idiopathic syndrome is commonly observed in dogs and rarely in cats. Most animals with this syndrome are middle-aged or older, but there does not appear to be a breed, age, or sex predisposition. Vomiting usually occurs late at night or early in the morning, suggesting that fasting or inactivity may modify normal motility patterns and result in duodenal reflux. Chronic intermittent vomiting of bile without food (having an empty stomach) is typical. Between episodes, the animal appears to be normal in all other respects, and the physical examination is unremarkable. Other conditions causing gastritis or duodenitis are also responsible for altered motility and may cause bile reflux. One should always investigate for Giardia or inflammatory bowel disease as possible etiologies to this syndrome. Animals with the bilious vomiting syndrome generally respond to symptomatic therapy. Feeding the animal a late evening meal often resolves clinical signs. It is thought that food possibly acts as a buffer to the refluxed bile or may enhance gastric motility. When diet fails, medical treatment should be considered. Choices include agents for gastric mucosal protection against the refluxed bile or the use of gastric prokinetic agents to improve motility. Often a single evening dose of a medication is all that is required to prevent clinical signs. Drugs that have been used include H2 blockers, sucralfate, antacids, or prokinetic agents, including metoclopramide (0.2–0.4 mg/kg) or cisapride (0.1 mg/kg). Recent evidence suggests that metoclopramide has poor prokinetic effects in the dog. A novel therapy involves erythromycin to improve gastric emptying. Erythromycin given at very low doses induces a motilin-like effect, stimulating migrating motor complexes in the stomach. In dogs a dose of 0.5–1 mg/kg improves gastric emptying of food. Ranitidine also has been shown to have some GI prokinetic effects but there is limited experience using ranitidine for GI motility.

Gastric Ulceration. Animals with gastric ulceration generally vomit blood, either fresh bright red blood or, more frequently, blood that has remained in the stomach and has undergone acid digestion and appears to have a “coffee ground” consistency. Some gastric ulcers may be silent and without obvious clinical signs. With chronic blood loss, iron loss becomes evident, resulting in a microcytic hypochromic anemia. With the presence of blood in the GI tract, the BUN is often increased, having a discordant normal creatinine. In cases of suspected GI ulceration, a fecal occult blood should be performed. Studies suggest that as little as 5 to 10 mls of GI bleeding will be detected on fecal occult exams. When you perform an occult exam, it is important that the patient is not eating a raw meat–based diet, as it will give false-positive results. Radiology is not very reliable for identifying ulcers. Diagnosis of a gastric ulcer is best made using endoscopy.

Factors causing gastric ulceration include nonsteroidal anti-inflammatory drugs (NSAID), severe illness, trauma, surgery, shock, neurological disease, renal disease, liver failure, mastocytomas, neoplasia, and hypoadrenocorticism (Addison’s disease).

The typical patient with hypoadrenocorticism presents with evidence of both mineralcorticoid and glucocorticoid deficiency. Classical signs are an acute adrenal crisis, with typical biochemical abnormalities of hyperkalemia and hyponatremia. Dogs respond to shock fluid and glucocorticoid and mineralcorticoid therapy. However, many Addisonian dogs are presented only because of gastrointestinal signs and lack the typical electrolyte changes that occur from mineralcorticoid deficiency. This atypical Addison’s disease is most common in young females. It appears that larger breeds (> 20 kg) are overrepresented. The history is generally described as a waxing and waning course with inappetence, weakness, or vomiting. Megaesophagus from esophageal hypomotility may occur with regurgitation. Others may present with vomiting blood or melena from gastrointestinal ulceration. The mechanism of ulceration is unknown; however, endogenous glucocorticoids are important in gastric mucosal integrity, and the lack of them may play a role in the ulceration.

Clues to the diagnosis may be found in the CBC with the lack of a stress leukogram (i.e., lymphocytosis and eosinophilia) and a nonregenerative anemia. Hypcholesterolemia, hypoalbuminemia, and hypoglycemia are common. Atypical dogs have normal serum electrolyte concentrations. The diagnosis of glucocorticoid-deficient hypoadrenocorticism is made using an ACTH stimulation test measuring serum cortisol concentrations at zero and 1 hours following injection. In the affected patient, both low pre-ACTH cortisol concentrations and post-ACTH cortisol concentrations (2 µg/dl or less) confirms the diagnosis. A recent report looking at only a single cortisol level suggests that if the value is greater than 2.0 µg/dl, it is highly unlikely to be hypoadrenocorticism. If values are less than 2.0 µg/dl, an ACTH stimulation should be performed.
The therapy for atypical Addison’s disease is glucocorticoid replacement. Some atypical Addison’s dogs may require mineralcorticoid replacement in the future. Prednisone is administered at the physiologic dose of 0.2 mg/kg/day. It is recommended to periodically evaluate the electrolytes to ensure that mineralcorticoid replacement is not required. The gastric ulceration is treated using H₂-receptor antagonists cimetidine (Tagamet, 5–10 mg/kg [IV or oral] q.i.d.), ranitidine (Zantac, 2 mg/kg t.i.d. [IV or oral]), famotidine (Pepcid, 0.5 mg/kg b.i.d. to q 24 hrs), and nizatidine (Axid, 5 mg/kg s.i.d.), or a proton pump inhibitor omeprazole (Prolosec, 20 mg time release capsule). Sucralfate (Carafate), using a dose of 0.5 to 1 gram 3 to 4 times a day, is also suggested. There is some concern that sucralfate will retard absorption of some drugs. It may decrease absorption of H₂-receptor antagonists by 20% to 30%. For this reason I generally give sucralfate 30 to 60 minutes after giving an H₂-receptor antagonists.

The Stomach Can’t Empty. Abnormal gastric retention is associated with a number of conditions, including antral pyloric mucosal hyperplasia. This is a condition characterized by mucosal hyperplasia that is large enough to block the pyloric lumen in the stomach, causing obstruction of gastric outflow. It is most often observed in the small middle-aged or older dog. Many are described as being nervous or high strung in nature. The signs are that of chronic vomiting of undigested food or gastric secretions. Vomiting may occur minutes to hours following eating. The etiology of this condition is unknown. Such things as chronic stress, nervous innervation, or chronic inflammation may play a role. Gastrin and histamine are two factors that are trophic to the gastric mucosa and may possibly be a factor.

The diagnosis is made by identifying gastric retention of barium due to mucosal folds in the antral region of the stomach. There should normally not be rugal folds in the antrum. Ultrasound may support antral thickening. My preferred means of evaluating the stomach radiographically is by performing a double contrast gastrogram. The technique involves administration of barium sulfate suspension (1–2 ml/kg) via stomach tube then rolling the patient to coat the mucosa. The stomach tube is replaced and the gastric lumen is distended with air. Generally cats require sedation using ketamine (1 mg/kg IV), and in dogs that require sedation, acepromazine should be used, as it causes limited alteration in gastric motility. This technique gives good mucosal delineation such as antral polyps and identifies intraluminal foreign bodies or lesions. If no lesion is identified, additional barium can be given to perform a standard upper gastrointestinal study.

Endoscopy confirms the finding of mucosal hypertrophy. The therapy involves surgery, for which there is a reported 80% success rate. Pyloroplasty, Y-U pyloroplasty, and Bilroth I are surgical considerations for this condition.

References and Suggested Reading


Wilson DV, Evans AT, Miller R. Effects of preanesthetic administration of morphine on gastroesophageal reflux
