SWALLOWING DISORDERS IN DOGS – MORE COMMON THAN YOU THINK!

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INTRODUCTION

Disorders affecting the oropharynx and esophagus are relatively common in dogs, and are typically associated with dysphagia. Despite the frequency of this problem, the diagnosis of disorders affecting the oropharyngeal, esophageal or gastroesophageal phase of swallowing can be extremely challenging, and warrant a thorough physical examination, followed by clinical observations of the animal eating and drinking. The causes of dysphagia may be secondary to either a neurologic or muscular disturbance of the swallowing reflex (functional) or secondary to strictures, traumatic injury, foreign bodies, or neoplastic processes (structural) involving the oropharyngeal region or esophagus. The swallowing reflex involves a coordinated process involving the tongue, hard and soft palate, pharyngeal muscles, esophagus, and gastroesophageal junction. The swallowing reflex is also dependent on normally functioning striated muscle and neuromuscular transmission, the integrity of the trigeminal, facial, glossopharyngeal, vagus, and hypoglossal nerves, their nuclei in the brainstem, and the swallowing center in the reticular formation of the brain.

Dogs with an abnormal oral phase of swallowing typically have difficulty with prehension and/or abnormal transport of a bolus to the tongue base. These disorders can often be diagnosed on physical or neurological examination, or by watching the animal eat. Oropharyngeal dysphagias affecting the pharyngeal phase of swallowing can be more challenging to diagnose, and often present with non-specific signs such as gagging, retching and the necessity for multiple swallowing attempts prior to the successful movement of a bolus into the proximal esophagus.¹ These patients have abnormal transport of bolus from the oropharynx to the hypopharynx or hypopharynx to the esophagus. Cricopharyngeal dysphagia is associated with the abnormal transport of a bolus through the proximal esophageal sphincter (PES). Signs are similar to those seen with pharyngeal disorders. Cricopharyngeal dysphagia may result from incomplete or lack of opening of the PES (true cricopharyngeal achalasia) or from abnormal
timing of PES opening (cricopharyngeal asynchrony) such that PES opening lags behind bolus presentation from pharyngeal contraction.\textsuperscript{1,2} In addition, a subset of dogs have combined disorders affecting both the pharyngeal and cricopharyngeal phases of swallowing, necessitating the implementation of videofluoroscopic swallow studies to accurately diagnose this disorder.

**ASSESSMENT PROCESS**

Assessment of dogs with signs and symptoms of feeding and/or swallowing disorders encompass multiple dimensions that include, but may not be limited to: a) review of the signalment; b) review of drug history and history of recent anesthesia; c) physical examination (prefeeding assessment); d) clinical feeding and swallowing evaluation; and e) instrumental evaluation of swallowing.

**Signalment**

The importance of the animal’s signalment cannot be overemphasized. Puppies can be diagnosed with a variety of congenital functional disorders affecting swallowing, including pharyngeal weakness, cricopharyngeal dysphagia, esophageal dysmotility, and sliding hiatal hernias. Breeds that have a hereditary predisposition or a high incidence of oropharyngeal or esophageal dysphagia include the Golden Retriever (cricopharyngeal dysphagia), Cocker and Springer spaniels (cricopharyngeal dysphagia), English Bulldog (sliding hiatal hernia), Chinese Char Pei (sliding hiatal hernia), Bouvier des Flandres (muscular dystrophy), Boxer (inflammatory myopathy), and German Shepherd (vascular ring anomaly). In addition, large breed dogs are predisposed to masticatory muscle disorders.

**Physical Examination**

Physical examination of the animal must include careful examination of the pharynx using sedation or anesthesia if necessary. The pharynx and neck should be carefully palpated for masses, asymmetry, or pain. The chest should be carefully auscultated for evidence of aspiration pneumonia. Evaluation of cranial nerves should be performed including assessment of tongue and jaw tone, and abduction of the arytenoid cartilages with inspiration. Complete physical and neurological examination may identify clinical signs supportive of a generalized neuromuscular disorder, including muscle atrophy, stiffness, or decreased/absent spinal reflexes. Contrary to
popular belief, the presence or absence of a gag reflex does not correlate with the efficacy of the pharyngeal swallow nor adequacy of deglutitive airway protection.³

Cervical and Thoracic Radiographs

The pharynx of normal animals is evident on radiographs because it is air filled. The size of the air-filled space can be decreased by local inflammation or neoplasia, laryngeal edema, or elongation of the soft palate. Pharyngeal size can also appear increased with dysfunction of the pharynx or upper esophageal sphincter, chronic respiratory (inspiratory) disease, and chronic severe megaesophagus. The normal esophagus is not visible on survey radiographs. An exception occurs following aerophagia due to excitement, nausea, dyspnea, or anesthesia.

Other Diagnostic Modalities

Confirmation of disorders affecting cricopharyngeal function, esophageal motility, and gastroesophageal function require additional diagnostic modalities, including videofluoroscopic assessment of swallowing, esophagoscopy, esophageal pH testing with/without concurrent impedance testing, esophageal manometry, and electromyographic analysis. These modalities are complimentary to one another, and their implementation is based on the results of clinical observation of the animal drinking and eating, and clinical suspicion for disorders affecting the oropharynx or esophageal motility. Although esophagoscopy and survey radiography provide anatomic information about the structures involved with the swallowing reflex, neither provides information about esophageal function. This is an important limitation of these diagnostic techniques, particularly in animals that are dysphagic secondary to dynamic disorders such as cricopharyngeal disease or esophageal dysmotility.

Contrast Videofluoroscopy

Contrast videofluoroscopy involves real time image capture of the dog as it is swallowing liquid barium or barium-soaked kibble. Videofluoroscopy is used to determine the normal sequence of events that make up a swallow and to measure the timing of these events in relation to one another.⁴ Additionally, the movement of certain anatomic structures is measured in relation to a fixed point to further assess function. Swallowing events that occur out of sequence, at inappropriate times or with reduced vigor can cause significant morbidity. For purposes of the
radiographic examination of swallowing, it must be remembered that videofluoroscopy captures only a brief window in time and it does not simulate a real meal. A normal study should not be used to conclusively rule out a diagnosis of hiatal hernia in a dog. More recently, a contrast videofluoroscopy method for quantifying pharyngeal contractility has been described in the dog. The pharyngeal constriction ratio (PCR) is calculated by dividing the pharyngeal area at maximum contraction by the pharyngeal area at rest. As pharyngeal contractility diminishes, the ratio approaches 1.0. This simple procedure provides important information regarding the strength of pharyngeal contraction in dysphagic dogs, and facilitates the improved selection of dogs diagnosed with cricopharyngeal dysphagia for surgical intervention.

**Esophageal Manometry**

Esophageal manometry measures pressure within the esophageal lumen and sphincters, and provides an assessment of the neuromuscular activity that dictates function in health and disease. Manometric techniques have improved in a step-wise fashion from a single pressure channel to the development of high-resolution manometry (HRM) with up to 36 pressure sensors. Advances in computer processing allow pressure data to be presented in real time as a compact, visually intuitive “spatiotemporal plot” of esophageal pressure activity. This spatiotemporal plot provides objective measurements of the forces that drive food and fluid from the pharynx to the stomach. This diagnostic modality is limited to academic institutions conducting clinical research studies on esophageal motility and reflux.

**Esophageal pH/Impedance Testing**

Esophageal pH/impedance testing is a useful diagnostic tool that is used to diagnose acid and non-acid reflux in animals with suspected gastroesophageal reflux (GER), unexplained esophagitis, or hiatal hernias. The technology of esophageal pH testing has advanced tremendously in recent years, and clinicians have several choices when selecting esophageal pH probes. The catheter-free Bravo pH Monitoring System from Medtronic is the first catheter-free system used to measure esophageal pH levels in human patients who are suspected of having GER, and is revolutionizing the way esophageal pH testing is done, because it allows people to maintain their regular diet and activities during pH testing. The Bravo system is an alternative to the traditional pH trans-nasal pH catheter that can cause patient discomfort, and is easily
dislodged by dogs and cats if the animal is not closely monitored. The Bravo System consists of two primary components: a) A small pH capsule the size of a gelcap that is attached to the wall of the esophagus and transmits data to a receiver; and b) a pager-sized receiver worn by the patient or animal that receives pH data from the Bravo capsule. After the test is completed, data from the receiver is downloaded to pH analysis software (Polygram™ Net pH Analysis Software) using infrared technology. The main disadvantage of the Bravo system is that one can only record esophageal pH, and the system does not utilize impedance technology that allows one to measure both acid and non-acid reflux. Esophageal pH testing has been extensively utilized in anesthetized dogs in an effort to identify risk factors for GER, and the effects of body position and prokinetic agents on GER.  

The veterinary profession has made tremendous strides in our ability to evaluate and diagnose disorders of the upper gastrointestinal tract in dogs. The diagnosis of cricopharyngeal and esophageal functional disorders is challenging, but can be facilitated with a comprehensive and structured approach, including observation of the animal eating and drinking, a thorough physical and neurological examination, evaluation of survey radiographs of the chest and neck, and selection of optimal advanced diagnostic modalities based on initial diagnostic findings, clinical suspicion, and availability of these tools.

Treatment of Cricopharyngeal Dysphagia

Definitive treatment for cricopharyngeal dysphagia has been reported with myotomy or myectomy of the cricopharyngeal muscles; however, a recent study documented a high failure rate in 14 dogs with cricopharyngeal dysphagia following cricopharyngeal myotomy and myectomy. Clinically, six dogs showed no improvement after surgery, three of which worsened postoperatively. Eight of the 14 dogs were euthanized due to problems related to cricopharyngeal dysphagia, including persistent dysphagia (8 dogs) and aspiration pneumonia (5 dogs). This study underscores the importance of critically assessing dogs for surgical intervention, and has revised our therapeutic approach to dogs with cricopharyngeal dysphagia. Likely reasons for the high failure rate observed in the dogs of the study following myotomy or myectomy of the cricopharyngeus muscle included incomplete work-up of dogs prior to surgical intervention, resulting in failure to accurately diagnose dogs with focal myasthenia gravis,
laryngeal paralysis, and other underlying polyneuropathies/myopathies. In addition, a few dogs with severe aspiration pneumonia had exacerbation of their pneumonia following the surgical procedure, and these dogs should have been better stabilized prior to surgery. Finally, dogs with concurrent pharyngeal weakness and cricopharyngeal dysphagia likely had exacerbation of their dysphagia following the myotomy or myectomy procedure. For these reasons, all dogs with cricopharyngeal dysphagia are currently worked up comprehensively to assess pharyngeal function via fluoroscopy, rule out concurrent or systemic polyneuropathies/myopathies. Dogs that are diagnosed with underlying neuropathies are managed conservatively with alterations of feeding practice or low-profile gastrostomy devices if the underlying neuropathy or myopathy cannot be specifically managed.

In veterinary medicine, the standard surgical approach for myotomy or myectomy has remained constant over the years. The cricopharyngeal and thyropharyngeal muscles can be approached by either a standard ventral midline approach, with 180° rotation of the larynx on its longitudinal axis or via a lateral approach with 90° rotation of the larynx. Cricopharyngeal myotomy involves transecting the cricopharyngeal muscle to the level of the pharyngeal mucosa. Endoscopic CO₂ laser cricopharyngeal myotomy is being increasingly utilized in people, and this procedure has been shown to be safe and effective, with decreased anesthesia time and morbidity compared with the more traditional transcervical cricopharyngeal myotomy. This procedure has not been performed in the dog to date, although it may well have application in appropriately selected animals.

Dogs with cricopharyngeal achalasia which is less common than cricopharyngeal dysphagia, can be managed surgically with myotomy or myectomy of the cricopharyngeus muscle, or via laser myotomy. An alternative and less invasive procedure involves the injection of botulinum toxin injection into the cricopharyngeus muscle. Botulinum toxin A (BTA) is a neurotoxin synthesized from the bacillus Clostridium botulinum. It acts at the presynaptic cholinergic nerve terminals to block the release of acetylcholine at the myoneuronal junction. In a dose-related manner, it weakens contraction when injected into the target muscle. The toxin has been used successfully in people for the treatment of esophageal achalasia, a condition characterized by hypertonicity of the lower esophageal sphincter. It has also been reported to be
of benefit for the management of people with cricopharyngeal achalasia. For clinical use, botulinum toxin activity is defined in units such that one unit represents the median lethal dose in mice. Because of its short half-life (4 h), the toxin is reconstituted shortly before injection with 0.9% sterile saline to a concentration of 25 units/cc. The average injection dose in people ranges between 25-50 units. In people with cricopharyngeal achalasia, physicians frequently balloon dilate the proximal esophageal sphincter during the botulinum injection procedure. We have successfully managed 4 dogs with severe cricopharyngeal achalasia with injection of botulinum toxin, although the benefits of the toxin wore off after approximately 2-4 months. This is an expected and well-documented phenomenon in people, and can be easily reapplied if warranted. Alternatively, a more permanent myotomy or myectomy of the cricopharyngeus muscle can be performed at this time. The limited duration of botulinum toxin’s effect is a benefit, as animals that respond favorably to the toxin should do well following surgical myotomy or myectomy. In contrast, animals that do poorly following botulinum injection can be supported with an enteral feeding device until the effects of the toxin have worn off. These animals should not undergo surgical correction of their cricopharyngeal disease, as the disorder will be exacerbated. Dogs diagnosed with concurrent cricopharyngeal dysphagia associated with dyssynchrony should not receive botox or surgical intervention. A comprehensive search for an underlying neuropathy/myopathy should be performed, and if unable to be documented, these dogs should be managed with a low-profile gastrostomy device.

**Treatment of Gastroesophageal Reflux Disease**

Gastroesophageal reflux disease (GERD) is a common chronic disorder in the Western world. The basic cause of GERD has been well characterized—the fundamental defect is a loss of integrity of the gastroesophageal barrier. What is less clear is the most appropriate means of addressing this reflux. GERD has a variety of symptoms in people, ranging from typical presentations of heartburn and regurgitation (without esophagitis) to atypical presentations, such as severe erosive esophagitis and its associated complications. Because of its symptomatic diversity, physicians may select from a variety of therapeutic approaches.

In dogs, most cases of GERD occur secondary to hiatal hernia; however, the author has documented cases of severe flaccidity of the lower esophageal sphincter in the absence of a
hiatal hernia. Medical therapy aims at decreasing acidity by suppressing proton secretion and has been well established. Available medications include antacids and alginates, H2-receptor antagonists, motility agents, and proton pump inhibitors (PPIs). All of these medications do little to prevent the reflux of gastric contents, and antireflux surgery is usually warranted. In people, antireflux surgery is commonly performed laparoscopically, and is aimed at reinforcing and repairing the defective barrier through plication of the gastric fundus.\textsuperscript{14} The earliest performed successful procedures were the Nissen and Toupet fundoplications, to which several modifications have since been made. It has been demonstrated in preliminary studies and long-term outcomes of such open surgery and preliminary studies of such laparoscopic surgery that antireflux surgery is an effective approach, with overall outcomes superior to those achieved with medications.\textsuperscript{14}

**Endoscopic Antireflux Therapies**

Endoscopic implantation of bovine dermal collagen and Teflon paste injected into the lower esophageal sphincter of dogs with surgically induced GERD was first reported in the late 1980’s.\textsuperscript{15} In recent years, endoscopic intraluminal antireflux approaches have attracted the attention of physicians, surgeons, and commercial companies, especially after the approval of two endoscopic intraluminal methods by the United States FDA in 2000. The common element is prevention of acid reflux by construction of a functional or controlled barrier in the lower esophageal sphincter zone. Three main methods are currently employed: endoscopic intraluminal valvuloplasty,\textsuperscript{16} endoscopic radiofrequency therapy,\textsuperscript{17} and endoscopic injection or implantation of foreign material.\textsuperscript{18} The endoluminal suturing method is highly demanding technically, and its short-term results are encouraging, although largely dependent on the experience of the endoscopist. Several prospective cohort studies have shown that the radiofrequency procedure (Stretta\textsuperscript{®}) significantly improves GERD symptoms and quality of life while reducing esophageal acid exposure and eliminating the need for antisecretory medications in the majority of patients within 6–12 months. Most recently, some researchers have studied the endoluminal implantation of polymers, such as Plexiglas\textsuperscript{™} (polymethyl-methylacrylate), Gatekeeper\textsuperscript{®} hydrogel, and Enteryx\textsuperscript{®} (ethylene vinyl alcohol copolymer).\textsuperscript{19} The preliminary results of these studies showed that the implantation method was feasible and safe; however, the only multicenter trial related to outcome that has been published has included just 1 year of follow-up.
Megaesophagus and its Management

Idiopathic megaesophagus is the most common type of megaesophagus in the dog and cat. The syndrome may be manifested either in puppies at the time of weaning or in adulthood. The etiology of idiopathic megaesophagus is unknown. The congenital form of the disease may be due to a delay in maturation of the esophageal neuromuscular system; a theory that explains why young dogs may improve with careful feeding management. Idiopathic megaesophagus has been shown to be inherited in the wire-haired fox terrier and the miniature schnauzer. A breed predisposition also exists for the German Shepherd, Great Dane and Irish Setter. The site and pathogenesis of the lesion in idiopathic megaesophagus is unknown. Suggested hypotheses include abnormalities of the afferent limb of the reflex arc (receptors, neurons) or of the swallowing center in the CNS. Idiopathic megaesophagus may also occur rarely in the cat. Secondary megaesophagus may result from a large number of systemic diseases including, myasthenia gravis, SLE, polymyositis, polymyopathies, dermatomyositis, polyneuropathies, dysautonomia, botulism, distemper, neoplasia, brain stem disease, lead and thallium toxicity, Addison's disease, hypothyroidism, pituitary dwarfism, and thymoma. Many obstructive esophageal diseases (neoplasia, granuloma, vascular ring anomaly, stricture, periesophageal masses and foreign bodies) can also lead to megaesophagus if they are of sufficiently chronic duration.

It is important to recognize that some dogs with focal myasthenia gravis manifested with diffuse megaesophagus diagnosed on thoracic radiographs can have a normal acetylcholine receptor antibody test at initial testing, and have an abnormal (positive) acetylcholine receptor antibody test when repeated 2-3 months later. Repeat acetylcholine receptor antibody testing is important in dogs, as many dogs with atypical focal myasthenia gravis have been misdiagnosed with idiopathic megaesophagus on the basis of a single normal acetylcholine receptor antibody test. Additional diagnostic procedures that can be performed based on the animal’s signalment, history, and neurological examination include an EMG, nerve conduction velocities, and muscle biopsies. Videofluoroscopy is essential for the diagnosis of functional esophageal disorders (esophageal dysmotility) not associated with esophageal dilation and has some prognostic value in megaesophagus via assessment of the severity of peristaltic dysfunction. Esophagoscopy is less reliable than radiography and fluoroscopy, although it can
be used to rule out underlying causes of megaesophagus such as esophagitis, neoplasia, and radiolucent foreign bodies.

*Medical management* of generalized megaesophagus involves modification of feeding practices. Treatment of the underlying cause (secondary megaesophagus) is of paramount importance. Dogs with megaesophagus generally tolerate a liquid or semi-liquid gruel better than solid food. Feeding from an elevated position allows gravity to help move the liquid into the stomach. If possible the animal should be held in a vertical position for 5 - 10 minutes after eating. Multiple feedings rather than one large single meal may also help minimize food accumulation in the esophagus. We have successfully placed low-profile-gastrostomy tubes for feeding in many dogs with idiopathic megaesophagus in an effort to minimize aspiration pneumonia. The silicon tubes used are extremely durable and are usually replaced on a yearly basis. The frequency of aspiration pneumonia has been markedly reduced in comparison to oral feeding and this therapeutic modality should be considered when a client is willing to dedicate the time to tube maintenance and feeding. The prognosis for dogs with megaesophagus is very variable depending upon the underlying etiology, the degree of dysfunction and the systemic status of the dog. The long-term *prognosis* is poor in most cases, although some cases can be managed successfully for years. The prognosis is improved if treatment of an underlying disease is possible.

**Esophageal Strictures and Esophagitis**

Esophageal strictures are a relatively common problem in dogs and less commonly cats, and can be caused by benign and malignant causes. Esophageal tumors are relatively rare in dogs and cats, and this discussion will focus on benign esophageal strictures. *The most common cause of esophageal stricture formation is gastroesophageal reflux in association with general anesthesia.* This phenomenon has been reported to occur in up to 65% of cases of esophageal stricture, with a median onset of clinical signs occurring 7.5 days post-anesthesia. The incidence of gastroesophageal reflux (GER) in dogs during anesthesia varies from 16-55%, and occurs secondary to a decrease in lower esophageal sphincter (LES) pressure. General anesthesia is associated with transient lower esophageal sphincter relaxation (TLESR) that is mediated by a vago-vagal neural pathway. In addition, reduction of LES pressure has also been shown to occur
secondary to a variety of anesthetic agents, including atropine, morphine, acepromazine, thiopentol, xylazine, and isoflurane.

The second most common cause of esophageal stricture formation is from esophagitis induced by administration of doxycycline or clindamycin. The proposed mechanism of tablet-induced esophagitis and stricture formation is from tablet retention in the esophagus due to poor esophageal clearance with a “dry” swallow. Other important causes of esophagitis and secondary stricture formation include chronic vomiting of acid contents from the stomach, foreign body ingestion, and swallowing of caustic substances. Esophagitis is associated with a weakening of the LES pressure that can result in further reflux of gastric contents and increased damage to the esophageal mucosa. Damage to the muscularis layer of the esophagus is often associated with fibroblastic proliferation and contraction leading to stricture formation. The clinical signs associated with severe esophagitis and/or esophageal stricture formation include odynophagia (painful swallowing), dysphagia, increased salivation, regurgitation, anorexia, coughing (secondary to aspiration pneumonia), and weight loss. These signs are often insidious at the onset and are often missed by owners, but are progressive as the esophageal lumen gets progressively narrowed.

Diagnostic Approach to Esophageal Strictures

The diagnostic procedures of choice include cervical and thoracic radiographs followed by an esophagram. Administration of liquid barium or barium-soaked kibble followed by thoracic radiographs is often diagnostic for detection of esophageal stricture(s); however, alterations in esophageal motility are best assessed utilizing a dynamic study such as videofluoroscopy. Esophagoscopy is an invaluable diagnostic tool to assess the esophageal mucosa and obtain biopsies, remove esophageal foreign bodies, and confirm the presence of an esophageal stricture that is occasionally missed on esophagram.

Treatment of Benign Esophageal Strictures

Mechanical dilation of the stricture is best accomplished using balloon dilation. The theoretical advantage of balloon dilation is that the forces applied to the stricture are a radial stretch, in contrast to the longitudinal forces applied with the rigid bougienage instrument. The
balloons are available in various diameters (up to 20-mm) and are made of a rigid plastic material that can withstand a relatively high pressure up to 45 psi. The balloons are manufactured to either pass through the biopsy channel of the endoscope, or alongside the endoscope with the use of a guide-wire. If a guide-wire is used, it is passed through the channel of the endoscope (or imaged with fluoroscopy) and advanced beyond the stricture into the stomach or caudal esophagus. The scope is removed as the guide-wire is advanced through the channel, thus leaving the guide-wire near its original position. The balloon catheter is then passed over the guide-wire (with the balloon deflated) until it is positioned within the stricture. The position of the balloon is visualized through the endoscope or via fluoroscopy. An inflating device that has a pressure reading is attached to the balloon and the pressure is slowly increased to the pressure specified by the manufacturer for that particular balloon. The balloon is kept inflated for 1-2 minutes before it is deflated. Sequentially larger balloons are used or increasing pressure is applied to the balloon until the desired amount of mucosal tearing and stricture dilation has been attained. The technique is extremely subjective in veterinary medicine, and we generally balloon dilate the stricture(s) 3-5 times during each dilation procedure.

The author has recently started injecting the steroid, triamcinolone, into the stricture site using a four-quadrant approach before the balloon dilation procedure. We generally inject approximately 2.5 mg triamcinolone into each of the quadrants using a Wang needle (or similar transbronchial needle) that can be threaded down the biopsy channel of the endoscope. The steroid is generally used for the first 2-3 dilation procedures that are performed 3-4 days apart. The rationale for the steroid injection is to prevent or minimize restricture formation that is a relatively common problem in some patients. One randomized trial of human patients with esophageal stricture showed that triamcinolone injection was associated with a longer dilation-free time interval. Only 13% of the patients who received the triamcinolone required repeat dilations compared with the 60% of the placebo-injected patients. Anecdotal reports of Mitomycin’s benefits for preventing restricture formation has also been discussed; however, there is no optimal method to apply the Mitomycin to the stricture site. Clinicians generally apply 5 mg of Mitomycin using a soaked gauze sponge that is placed endoscopically at the stricture site for approximately 5 minutes. The site is then rinsed with 60 mL of water following the removal of the sponge.
Intraluminal stents are being used with increasing frequency in veterinary medicine for patients that have failed balloon dilation or for patients with recurrent stricture formation. Stents are available both covered (polypropylene) and uncovered. The covering helps prevent the ingrowth of tissue within the stent. Available stent materials include Nitinol (nickel plus titanium), Elgiloy (cobalt, nickel, plus chromium), stainless steel, polyester plastic/silicone, or a biodegradable material such as PDS. The selection of a particular stent is based upon the characteristics of the stricture such as its location and length, and the need for removal of the stent. Once the stent is deployed, it must be anchored in place or it will rapidly migrate into the stomach. The stent can be secured in place using a suturing device (GI Stitch, Pare Surgical) that can be used through a double channel endoscope.

Medical management of esophagitis involves a combination of proton pump inhibitors such as omeprazole, sucralfate suspension, and a potent prokinetic agent such as cisapride to minimize further gastric reflux and facilitate gastric emptying. The author discourages the use of parenterally or orally administered steroids to dogs and cats with benign esophageal strictures.

REFERENCES:
15. O’Connor KW. *Gastrointest Endosc* 1988;34:106-112;