UPDATE ON ESOPHAGEAL DISORDERS IN DOGS – FROM BENCH-TOP TO BEDSIDE

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INTRODUCTION

The normal swallowing reflex is a four-stage process, characterized by the oral preparatory phase, oral phase, pharyngeal phase, and esophageal phase.[1] Disorders affecting the oropharynx and esophagus are relatively common in dogs, and are typically associated with dysphagia. Abnormalities of the pharyngeal phase of swallowing are associated with pharyngeal weakness secondary to neuropathies or myopathies, pharyngeal tumors or foreign bodies, and obstruction of the proximal esophageal sphincter (PES) secondary to hypertrophy of the cricopharyngeus muscle. Synchrony between constriction of the pharyngeal muscles and relaxation of the cricopharyngeus muscle is essential to allow passage of the bolus into the esophagus. The esophageal phase is involuntary and begins with the relaxation of the PES and movement of the bolus into the esophagus.

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.[2,3] 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 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 dyssynchrony) such that PES opening lags behind bolus presentation from pharyngeal contraction.[2,3] 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 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, Cocker spaniel, Springer spaniel, Maltese, and miniature Dachshund (cricopharyngeal dysphagia), English bulldog, French bulldog, Chinese Char Pei, Boston terrier, and Pug (sliding hiatal hernia), Bouvier des Flandres, Golden Retriever, and Cavalier King Charles Spaniel (muscular dystrophy), Boxer and Newfoundland (inflammatory myopathy), and German Shepherd (vascular ring anomaly). In addition, large breed dogs are predisposed to megaesophagus and 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. The gag reflex should be evaluated by placing a finger in the pharynx; however, the presence or absence of a gag reflex does not correlate with the efficacy of the pharyngeal swallow nor adequacy of deglutitive airway protection.[4]

Observation of animal eating and drinking:

The importance of the clinician carefully observing the dysphagic animal while it is eating (kibble and canned food) and drinking in the hospital cannot be overemphasized, and is instrumental in helping to localize the problem to the oral cavity, pharynx, or esophagus. Dogs with an abnormal oral phase of swallowing typically have difficulty with prehension and/or aboral transport of a bolus to the tongue base, and these disorders can often be diagnosed 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. These patients have abnormal transport of bolus from the oropharynx to the hypopharynx or hypopharynx to the proximal esophagus. Cricopharyngeal dysphagia is associated with the abnormal transport of a bolus through the PES, and signs are similar to those seen with pharyngeal disorders.

Cervical and Thoracic Radiographs

The pharynx of healthy 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.

Neuromuscular Minimum Database:

A comprehensive database is warranted in animals with OPD, and should consist of a complete blood count, serum chemistry including creatine kinase (CK) and electrolyte concentrations, urinalysis, evaluation of thyroid function, and the acetylcholine receptor (AChR) antibody titer for acquired myasthenia gravis. A persistently elevated CK could be an indication of an inflammatory myopathy, whereas markedly elevated CK concentrations may suggest a necrotizing or dystrophic myopathy. A normal CK does not rule out a myopathy, particularly when the myopathy is focal (masticatory muscle myositis) or in the chronic stage of disease. Acquired myasthenia gravis is an important neuromuscular cause of OPD, and can be associated with focal signs including pharyngeal, esophageal, and laryngeal weakness without clinically detectable limb muscle weakness. Pharyngeal weakness, as the only clinical sign of myasthenia gravis, was described in 1% of the myasthenic dogs.[5]

Videofluoroscopic Swallow Study:

Contrast videofluoroscopy involves real time image capture of the animal as it is swallowing liquid barium or barium-soaked kibble, and is one of the most important procedures for assessing the functional integrity of the swallow reflex and esophageal motility. 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. 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. One problem with videofluoroscopy is that animal positioning is not standardized in veterinary medicine. Alterations in body position (sternal versus lateral recumbency) do not appear to affect measurements of pharyngeal constriction ratio or the timing of swallowing in healthy dogs, however; cervical esophageal transit is significantly delayed when dogs are imaged in lateral recumbency. Swallow studies performed with the dog in sternal recumbency are significantly more likely to result in generation of a primary peristaltic wave for both liquid and kibble boluses. Thus, it is important to recognize that the retention of liquid or kibble boluses in the cervical esophagus may not be considered abnormal when clinically ill dogs are imaged in lateral recumbency because this may be related to body position. The fluoroscopic swallow study typically involves assessment of 5 swallows each of 5-10mL liquid barium (60% w/v) followed by 5 swallows of kibble soaked in barium.

The timing of the swallow can be easily determined when viewing the swallow study frame by frame in the video, where each frame represents $1/30^{th}$ of a second in the NTSC system, the analog television system used in the US. The frame in which the epiglottis is observed to close over the larynx is considered as the starting point for all time measurements, and frames are counted until the observation of maximal contraction of the pharynx, opening of the PES and closing of the PES. The swallow is considered completed when the epiglottis is observed to re-open, and usually takes 5-6 frames in healthy dogs.[2] More recently, a contrast videofluoroscopy method for quantifying pharyngeal contractility has been described in the dog.[6] 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.[6] 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.

Laryngoscopy/Pharyngoscopy and Esophagoscopy:

Thorough laryngeal examination is important in all animals with OPD to rule out laryngeal paralysis associated with a polyneuropathy. Geriatric, large-breed dogs can experience a progressive generalized neuropathy, with associated pharyngeal weakness, OPD, and esophageal dysmotility.[7] Pharyngoscopy and esophagoscopy provide anatomic information about the structures involved in the oropharynx and esophagus, but both procedures are of limited diagnostic utility for evaluating functional disorders in anesthetized animals. Unsedated transnasal videoendoscopy is an easily accomplished and useful procedure in people that is often performed as an outpatient procedure. The author has assessed the feasibility of fiberoptic endoscopic evaluation of swallowing (FEES) via transnasal intubation in fully awake dogs; however, the procedure is limited to larger dogs that can be readily restrained for the procedure. Although esophagoscopy and survey radiography provide anatomic information about the structures involved with the swallowing reflex and esophagus, neither provides information about esophageal function. This is an important limitation of these diagnostic procedures, particularly in animals that are dysphagic secondary to dynamic disorders such as cricopharyngeal disease or esophageal dysmotility. Esophagoscopy is helpful for diagnosing esophagitis, esophageal strictures (that can be missed on barium swallow studies), and hiatal hernias.

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.[8] 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. HRM has been successfully implemented in awake dogs following intranasal insertion.[9]

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 is 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[TM] 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.[10]

Electrodiagnostic Testing:

Electrodiagnostic evaluation, including electromyography and measurement of motor and sensory nerve conduction velocities, does not provide a specific diagnosis in most cases, but can provide important information as to the severity, distribution, and character of a myopathic or neuropathic disease process, and assist with guiding the optimal anatomic site for biopsy. Electrodiagnostic testing should also include evaluation of the pharyngeal muscles and tongue. The health status of the animal must be taken under consideration as the procedure is performed under general anesthesia.

Muscle and Nerve Biopsies:

Muscle and nerve biopsies are usually integral to reaching a specific diagnosis. This procedure should be performed after the serum AChR antibody titer has been determined to be negative. If the onset of clinical signs is recent and the antibody test negative, retesting in 4-6 weeks is suggested, as a significant number of dogs with early clinical signs can have antibody titers below the detection limits of the test at initial testing, but test positive 4-6 weeks later. Muscle biopsies are usually obtained from a large proximal pelvic limb such as the vastus lateralis or a thoracic limb such as the triceps muscle; however, biopsies of the pharynx and cricopharyngus muscle should also be obtained in dogs with oropharyngeal dysphagia. Muscle biopsies should ideally be performed relatively early in the disease process when warranted before irreversible muscle fibrosis and myofiber loss is extensive.

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 ESOPHAGEAL DISORDERS

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.[11] 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 underscored 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 dsyphagia 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.[12] Cricopharyngeal myotomy involves transecting the cricopharyngeal muscle to the level of the pharyngeal mucosa. Endoscopic CO_2 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.[13] This procedure has been performed in a handful of dogs at UC Davis and is less invasive than the traditional open myectomy procedure; however, the potential for recurrence of dysphagia as a consequence of incomplete myectomy of the cricopharyngeus muscle appears greater compared to the open procedure.

An alternative and less invasive procedure for the management of cricopharyngeal dysphagia involves the injection of botulinum toxin injection into the cricopharyngeus muscle.[14] 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, 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.[14] In people with cricophayryngeal achalasia, physicians frequently balloon dilate the proximal esophageal

sphincter during the botulinum injection procedure. We have successfully managed dogs with severe cricopharyngeal dysphagia with injection of botulinum toxin, although the benefits of the toxin wore off after approximately 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. Dogs diagnosed with cricopharyngeal dyssynchrony are challenging to manage, because there is no direct evidence of an obstructing "bar" (hypertrophied cricopharyngeal muscle) on fluoroscopy or surgery. A comprehensive search for an underlying neuropathy/myopathy should be performed prior to intervention, and if unable to be documented, these dogs should be managed with injection of Botox prior to consideration of cricopharyngeal myectomy.

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.[15] 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.[16] Endoscopic antireflux therapies are currently being utilized in people, although clinical trials in dogs are lacking. Three main methods are currently employed: endoscopic intraluminal valvuloplasty,[17] endoscopic radiofrequency therapy,[18] and endoscopic injection or implantation of foreign material.[19] 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®) 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™ (polymethyl-methylacrylate), Gatekeeper® hydrogel, and Enteryx® (ethylene vinyl alcohol copolymer).[20] 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.

It is clear that there are a plethora of research studies using animal models to evaluate less-invasive approaches to prevent reflux in people. These models provide an ideal template to further our investigations in an effort to provide additional options for the surgical and endoscopic management of congenital or acquired hiatal hernias and reflux disease in the dog.

Megaesophagus

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.

Repeat acetylcholine receptor antibody testing is important in dogs, as many dogs with focal myasthenia gravis have been misdiagnosed with idiopathic megaesophagus on the basis of a single normal acetylcholine receptor antibody test. Repeat testing should be done approximately 6-8 weeks following the first 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. This can often be accomplished with the advent of a Bailey chair or similar device. 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

Esophageal strictures are a relatively common problem in dogs and less commonly cats, and can be caused by benign and malignant causes, although the latter are relatively uncommon in dogs and cats. *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.

Mechanical dilation of the stricture is best accomplished using balloon dilation or bougienage. 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. However, a retrospective case series in 20 dogs and 8 cats with benign esophageal strictures that underwent bougienage treatment suggested that this procedure was safe and effective for most dogs and cats with benign esophageal strictures, with outcomes similar to balloon dilation.[21] The balloons used for balloon dilation 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 (generally ≤ 50% of the original esophageal stricture diameter). The procedure is repeated 3-5 days later in an effort to maximally dilate the stricture.

The administration of triamcinolone into the stricture site using a four-quadrant approach before the balloon dilation procedure has been associated with a reduced rate of restructure formation.[22] 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. Topical mitomycin C has also been shown to be beneficial for preventing restricture formation.[23] 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.

Esophagitis

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. Proton pump inhibitors can initially be administered q 12 hrs for 3-4 days to decrease acid production more rapidly, before continuing with once daily administration. H2-receptor antagonists can also be administered at night to help minimize nocturnal acid breakthrough. Proton pump inhibitors should be given approximately 30 minutes before a meal.

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