Canine Megaesophagus

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Symptoms of esophageal disease occur when esophageal motility is disturbed or when the movement of ingesta is obstructed. Swallowing becomes difficult, even painful, and food does not go down. Regurgitation, dysphagia, and ptyalism are common signs, and aspiration pneumonia is a frequent complication. Typically liquid and solid foods pass poorly with motility disturbances of the esophagus, whereas liquids often pass easily with obstruction of the esophagus. A thorough physical examination and often an extensive diagnostic workup are necessary for determining the cause of esophageal disease and designing a treatment plan. Web Box 47-1 lists the primary causes of esophageal dysfunction in the dog.

Functional Anatomy

The esophagus is not simply a tube through which food passes. It is an organ with complex innervation and patterns of motility designed to transport fluid and food efficiently from the pharynx to the stomach. The esophagus begins at the pharyngoesophageal junction, commonly referred to as the upper esophageal sphincter (UES), which prevents reflux and aspiration of ingesta from the esophagus. The body of the canine esophagus consists of two layers of skeletal muscle that propel ingesta to the stomach. The gastroesophageal junction, referred to as the lower esophageal sphincter (LES), is the distal limit of the esophagus and prevents reflux of gastric content into the esophagus.

Disorders of the Esophagus

Motility Disorders
- Megaesophagus
- Congenital
- Acquired
  - Primary (idiopathic)
  - Secondary (see Web Box 47-2)
- Dysautonomia
- Hiatal hernia?

Inflammatory Disease
- Esophagitis
- Gastroesophageal reflux
- Hiatal hernia

Obstructive Lesions
- Foreign body
- Stricture
- Vascular ring anomaly
- Neoplasia

Miscellaneous
- Diverticula
- Bronchoesophageal fistula

?, Not well documented.

Diseases Associated with and Causes of Megaesophagus in the Dog

Central Nervous System
- Distemper
- Cervical vertebral instability with leukomalacia
- Brainstem lesions
- Neoplasia
- Trauma

Peripheral Neuropathies
- Polyneuritis
- Polyradiculoneuritis
- Ganglioradiculitis
- Dysautonomia
- Giant cell axonal neuropathy
- Spinal muscular atrophy
- Toxicity
  - Lead
  - Thallium
  - Acrylamide
- Bilateral vagal damage

Neuromuscular Junction
- Myasthenia gravis
- Botulism

- Tetanus
- Anticholinesterase toxicity

Esophageal Musculature
- Esophagitis
- Systemic lupus erythematosus
- Glycogen storage disease
- Polymyositis
- Dermatomyositis
- Cachexia
- Trypanosomiasis
- Hypoadrenocorticism
- Hypothyroidism?

Miscellaneous
- Pyloric stenosis
- Gastric dilation volvulus
- Pituitary dwarfism
- Thymoma
- Mediastinitis

?, Not well documented.

The UES separates the pharynx from the cervical portion of the esophagus and is formed by the cricopharyngeus and thyropharyngeus muscles dorsolaterally and the cricoid cartilage ventrally. These striated muscles are innervated by the glossopharyngeal, pharyngeal, and recurrent laryngeal branches of the vagus nerve that originate in the brainstem nucleus ambiguus. The muscles of the sphincter remain contracted at all times, except during a swallow, when they relax momentarily to allow passage of a bolus. The muscles contract promptly to maintain closure of the sphincter and protect against esophagopharyngeal reflux and aspiration.

In contrast to the feline esophagus that is composed of smooth muscle in the distal one third, the canine esophageal body is composed entirely of two oblique layers of skeletal muscle and is innervated by the somatic branches of the vagus nerve. The LES is a physiologic sphincter rather than a true anatomic sphincter because it does not consist of a distinct muscle mass. It consists of an outer layer of longitudinal striated muscle and an inner layer of circular smooth muscle that merge with the smooth muscle of the stomach. The LES remains closed except to allow passage of a bolus. Competence of the LES is maintained by the gastric rugal folds, the muscular sling of the right crus of the diaphragm, the oblique angle of the gastroesophageal junction, and gastric compression on the esophagus. This sphincter separates the esophagus from the cardia of the stomach and allows ingesta to pass into the stomach while preventing reflux of stomach content into the esophagus. Cholinergic, nonadrenergic noncholinergic, and myogenic mechanisms play a role in

maintaining gastroesophageal sphincter tone. Increases in tone, which protect the esophagus from gastroesophageal reflux, are mediated by moderately increased intragastric pressure; acid in the cardia; high-protein diet; and a number of hormones such as gastrin, histamine, and acetylcholine (ACh). Conversely, marked increases in intragastric pressure decrease sphincter tone to facilitate eructation and prevent gastric rupture.

Esophageal contraction occurs in response to swallowing (primary peristalsis) and esophageal distention (secondary peristalsis). The oropharyngeal phase of swallowing and the movement of food through the UES initiate primary peristalsis. Afferent vagal receptors in the pharynx and proximal esophagus are stimulated by the presence of food; solids are more effective than liquid in stimulating a swallowing reflex. The origin of the vagus nerve, the nucleus ambiguus for striated muscle, initiates an efferent response via the somatic nerve fibers of the vagus. This neuronal pathway ends at the myoneural junction with a coordinated contraction of the UES and propagation of a peristaltic wave aborally along the body of the esophagus, through the LES, and into the stomach. Remaining intraluminal ingesta within the esophagus stimulate esophageal afferent receptors to initiate a secondary peristaltic wave to clear the lumen. Any disease or lesion affecting any part of this neuromuscular pathway can alter normal esophageal motility and cause megaesophagus (Web Box 47-2).

Megaesophagus

Megaesophagus is a condition characterized by decreased or absent esophageal motility that usually results in diffuse dilation of the esophagus. Megaesophagus occurs as a congenital disorder that becomes clinically apparent at or shortly after weaning, or it can occur as an acquired disorder in a previously normal adult. Acquired megaesophagus can be secondary to a variety of diseases that cause neuromuscular dysfunction, or it can occur as a primary disorder for which the cause is unknown (idiopathic megaesophagus).

Congenital Megaesophagus

Congenital megaesophagus occurs in purebred and mixed-breed dogs. It is known to be inherited in the wirehaired fox terrier as an autosomal-recessive trait and in the miniature schnauzer as either an autosomal-dominant or autosomal-recessive trait with partial penetrance. Congenital megaesophagus occurs with increased prevalence in Great Danes, German shepherds, Labrador retrievers, Newfoundlands, Chinese shar-peis, and Irish setters. Although not proved, the predilection for megaesophagus in these breeds, in addition to reports of entire litters of German shepherds, Great Danes, Newfoundlands, and shar-peis being affected, suggests a hereditary basis for megaesophagus. For this reason owners are advised not to use affected dogs or those closely related to affected dogs for breeding. Clinical signs usually occur by 3 months of age; however, dogs with mild symptoms may not be presented until 1 year of age.

Although the pathogenesis of congenital megaesophagus is unclear, esophageal function studies of affected dogs indicate defects in the vagal afferent innervation of the esophagus. Other studies have confirmed that vagal efferent innervation in affected dogs is normal but that esophageal motor function is decreased, possibly secondary to abnormal biomechanical properties of the esophageal muscle.

Acquired Megaesophagus

Acquired megaesophagus can occur in any breed; however, the breeds at a significantly increased risk for developing the disease include some of the same breeds discussed previously (i.e., German shepherds, golden retrievers, Irish setters, and Great Danes). Secondary megaesophagus can be caused by any disorder that inhibits esophageal peristalsis either by disrupting esophageal neural pathways or by causing esophageal muscular dysfunction. Numerous central and peripheral neuropathies, diseases of the neuromuscular junction, and myopathies have been reported to cause megaesophagus (see Web Box 47-2). Most of these diseases are uncommon, and an exhaustive search to rule out all is unrealistic. However, several diseases routinely should be considered.

Myasthenia gravis (MG) is the most common cause of acquired megaesophagus in the dog. It occurs rarely as a congenital disease and more frequently as an acquired disease; both can cause megaesophagus. Acquired MG is an autoimmune disorder that interferes with normal neuromuscular transmission. Production of autoantibodies against nicotinic ACh receptors decreases the number of receptors available for normal neuromuscular transmission, resulting in skeletal muscle weakness. Two forms of acquired MG, generalized and focal, have been identified. Generalized MG causes exercise-related generalized muscle weakness that worsens after exercise and improves with rest. Most dogs with generalized MG also have megaesophagus. Focal MG causes weakness that affects predominantly esophageal, pharyngeal, or facial muscles. Affected dogs usually have symptoms of megaesophagus.

Diagnosis of MG is made by measuring increased antibody titers to ACh receptors, but serum ACh receptor antibody concentrations tend to be lower in focal MG than in the generalized form. ACh receptor antibodies are negative in up to 15% of generalized MG and up to 50% of focal MG in humans (Dewey, 1997). Seronegative myasthenics exist in the veterinary population as well. Approximately 2% of dogs with generalized myasthenia gravis are seronegative. The percentage of dogs with seronegative focal myasthenia gravis has not been determined (Shelton, 2002). Immunocytochemical staining, which localizes the immune complexes at the neuromuscular junction after incubation of patient serum with normal canine muscle, is a second diagnostic method that is also relatively inexpensive and easy to perform. This test is not specific for antibodies against the ACh receptors; thus a positive result is not definitive. However, it is a useful screening test.

Many dogs diagnosed as having "idiopathic" megaesophagus are likely to have focal MG. In a study by Shelton and associates (1990), serum samples from 152 dogs with idiopathic megaesophagus were tested for ACh receptor antibodies. Results confirmed that 40 of 152 (26%) had antibody titers diagnostic for MG. Another 17 cases (11%) that did not have positive titers had positive immunocytochemical staining of immune complexes. Of those affected, 48% had clinical improvement or remission of clinical signs with treatment.

Occasionally megaesophagus is observed in dogs with primary, secondary, or atypical hypoadrenocorticism. Impaired muscle carbohydrate metabolism and depletion of muscle glycogen stores resulting from glucocorticoid deficiency and decreased catecholamine activity have been suggested as possible causes. Megaesophagus has been reported to resolve with prednisone treatment in dogs with glucocorticoid-deficient hypoadrenocorticism (Bartges and Nielson, 1992).

Hypothyroidism historically has been cited as a possible cause of megaesophagus. However, a definitive association between hypothyroidism and megaesophagus has not been proved. In a case-controlled study by Gaynor, Shofer, and Washabau (1997) of 136 dogs with acquired megaesophagus, 272 control dogs from the general hospital population, and 151 control dogs that underwent thyroid-stimulating hormone response tests, no association between megaesophagus and hypothyroidism was found. In one retrospective study of 29 hypothyroid dogs, four had megaesophagus; one dog showed clinical improvement in esophageal symptoms when treated with thyroid supplement. Radiographic evidence of a dilated

esophagus persisted in all four dogs (Jaggy et al, 1994). There is an association between MG and hypothyroidism, most likely caused by a common immune-mediated disorder. Therefore thyroid function still should be evaluated in dogs with megaesophagus until MG has been ruled out definitively.

Dysautonomia, an idiopathic condition that results in clinical signs attributable to failure of the sympathetic and parasympathetic nervous systems, is becoming a more common cause of megaesophagus. Dysautonomia typically affects young dogs from rural environments with the freedom to roam. Clinical signs are consistent with autonomic dysfunction and include vomiting, regurgitation, weight loss, dysuria, decreased anal tone, mydriasis with absent pupillary light reflexes, decreased tear production, and dry mucous membranes. More than 60% of patients with dysautonomia have radiographic evidence of megaesophagus; 71% of those have concurrent lung disease (Detweiler et al, 2001). Radiographic evidence of megaesophagus with dysautonomia is indistinguishable from that seen with other causes of megaesophagus; however, gastric motility should be normal with causes other than dysautonomia.

Another condition that should prompt evaluation for megaesophagus is laryngeal paralysis. Many dogs with laryngeal paralysis also have concurrent esophageal dysfunction. Laryngeal paralysis may be a risk factor for acquired megaesophagus because both diseases have a common pathogenesis involving the vagus nerve. The vagus nerve may be the only affected nerve, or it may be affected as part of a diffuse polyneuropathy. Similarly, dogs with histories of chronic or recurrent gastric dilation with or without volvulus should be evaluated for megaesophagus. In these cases LES obstruction or esophagitis secondary to vomiting is the proposed mechanism for acquired megaesophagus.

Idiopathic Megaesophagus

Idiopathic megaesophagus is a severe and often fatal disease characterized by a large, dilated esophagus with no apparent motility. It occurs spontaneously, usually in large-breed adult dogs between 5 and 12 years of age. There appears to be no sex or breed predisposition. Unfortunately the majority of adult dogs with megaesophagus are diagnosed as having idiopathic megaesophagus. The etiopathogenesis of this disorder is unknown; however, recent studies have clarified that the abnormality appears to be neurogenic rather than myogenic. Manometric studies have shown that the function of the UES and LES is normal in response to a swallow, indicating that the efferent innervation is intact. However, when the esophagus is distended with an intraluminal balloon to initiate secondary peristalsis, compliance of the esophagus is increased compared with normal dogs, the UES and LES fail to relax as in normal dogs, and inhibition of diaphragmatic electromyographic activity does not occur as in normal dogs. These observations indicate a defect of either afferent sensory innervation of the esophagus or esophageal muscle function (Holland, Stachell, and Farrow, 1996; Tan and Diamant, 1987).

Clinical Signs

Regurgitation is the most common clinical sign observed with megaesophagus. Most owners fail to recognize the difference between regurgitation and vomiting and report vomiting as the primary complaint. The clinician must differentiate between these clinical signs to ensure proper localization and diagnosis of the problem. Regurgitation is characterized as a passive evacuation of fluid, mucus, and undigested food from the esophagus. No consistent temporal relationship occurs between eating and regurgitation caused by esophageal disease. Other signs observed with megaesophagus include ptyalism, halitosis, and vomiting. Cough, nasal discharge, and dyspnea caused by aspiration pneumonia are frequent presenting complaints, especially in young or debilitated dogs. Some dogs appear normal on physical examination, whereas others are underweight to cachectic from poor nutritional intake, depending on the duration and severity of disease. Puppies with congenital megaesophagus are usually smaller than their littermates. Swelling of the ventral neck near the thoracic inlet from esophageal distention with ingesta is present occasionally. Other clinical signs may reflect diseases causing secondary megaesophagus. Careful examination should be performed for neuromuscular disease such as muscle weakness, pain, or neurologic deficits.

Diagnosis

Diagnosis of megaesophagus is based on radiographic identification of a dilated or hypomotile esophagus. Survey thoracic radiographs confirm the presence of generalized megaesophagus in most cases and usually reveal an esophagus dilated with air, fluid, or ingesta. In equivocal cases with mild or segmental dilation or if hypomotility without dilation is suspected, a contrast esophagogram is indicated. Barium liquid and barium meal contrast studies must be performed to detect subtle hypomotility and to rule out a stricture, foreign body, or other obstructive lesion. Although the availability of fluoroscopy in practice is limited, esophageal motility can be assessed well with static contrast radiographs during and immediately after a swallow of contrast material. Esophageal retention of any contrast material in a dog that is symptomatic for esophageal disease is abnormal. Radiographic signs of aspiration pneumonia, even in the absence of clinical or radiographic signs of esophageal disease, should alert the clinician to the potential for esophageal disease and the need for a contrast esophagogram. Dogs that have significant retention of contrast material in the esophagus are at risk for severe aspiration. They should be held in a vertical position for 5 to 10 minutes after the procedure and closely observed for at least an additional 30 minutes.

Esophageal motility is evaluated best using fluoroscopic, manometric, or scintigraphic procedures, which usually are limited to referral practices and teaching hospitals. Fluoroscopy provides visualization of swallow dynamics and helps to identify anatomic abnormalities of the esophagus. Manometry and scintigraphy provide quantitative measures of esophageal motility. Manometry

is most useful to evaluate subtle motility abnormalities not evident on fluoroscopy. Manometry uses a catheter passed into the esophageal lumen for dynamic measurement of esophageal pressures, transit rate, and lower esophageal pressures during a swallow. Scintigraphy is a newer quantitative technique used to measure the transit time of a radiolabeled food bolus as it moves through the esophagus. Scintigraphy has the advantage of evaluating the response of the esophagus to a normal bolus in an awake patient without the influence of foreign material such as barium or an esophageal catheter.

Once the presence of megaesophagus has been confirmed, the clinician must determine whether the disorder is primary (idiopathic) or secondary. Generally most dogs with idiopathic megaesophagus have a large, dilated, aperistaltic esophagus. Contrast material is slow to move into the stomach and may not do so for hours. Dogs with secondary megaesophagus often are not affected as severely and have less dilation and some motility. A recent study to evaluate whether dogs with megaesophagus secondary to myasthenia gravis had less esophageal dilatation radiographically than dogs with other causes of megaesophagus documented a small but significantly increased esophageal diameter in nonmyasthenic dogs. However, because of a large overlap in values, the finding offers limited clinical utility (Wray and Sparkes, 2006).

Formulating a logical and economic diagnostic plan can be challenging. The initial diagnostic plan should be broad and include a complete blood count; a serum chemistry profile that includes a creatine kinase determination, electrolyte determination, and urinalysis; and a fecal examination. Results of these tests help determine which additional diagnostic tests should be considered. A complete blood count may provide a clue to the presence of hypoadrenocorticism, immune-mediated disease, lead toxicosis, or pneumonia. Serum chemistry profiles are useful to detect hypoadrenocorticism or myositis. Proteinuria is supportive of the diagnosis of systemic lupus erythematosus, and a fecal examination may identify *Spirocerca lupi* in dogs from endemic areas. Patients with congenital or idiopathic megaesophagus generally have few if any laboratory abnormalities.

If results of the initial diagnostic tests are inconclusive, an ACh receptor antibody test should be performed to rule out focal or generalized MG. The immunoprecipitation radioimmunoassay is a specific and sensitive test. When the index of suspicion for MG is high, but a diagnosis is not confirmed with initial ACh receptor antibody serology, repeat serology 1 to several months later may seroconvert in a small percentage of cases. Also a motor point muscle biopsy may be submitted to the aforementioned laboratory for immunocytochemical staining. This test is less specific for MG but is highly suggestive for diagnosing seronegative myasthenia gravis. Although hypoadrenocorticism infrequently is associated with megaesophagus, this disease is a potentially treatable cause of megaesophagus. Dogs with unexplained acquired megaesophagus should have adrenal function (adrenocorticotropic hormone stimulation test) evaluated. Furthermore, because of an indirect association to megaesophagus through association with other polyneuropathies such as MG, hypothyroidism should be ruled out with thyroid hormone evaluation. If gastric hypomotility also is suspected on radiographs, several simple pharmacologic tests can aid in an antemortem diagnosis of dysautonomia. Affected dogs should develop miosis in response to ocular installation of dilute (0.1%) pilocarpine solution, have no change in heart rate after administration of atropine, and have no flare response to intradermally administered histamine (Berghaus et al, 2001; Harkin, Andrews, and Nietfeld, 2002).

Esophagoscopy usually is not helpful in determining the cause of megaesophagus. It is indicated if an obstructive lesion or foreign body is suspected but not confirmed by radiographs and to confirm the presence of esophagitis.

Other tests should be considered for individual cases if specific clinical signs or results of preliminary laboratory tests or both indicate the presence of a toxic, neurologic, or muscular disease. Lead, thallium, and anticholinesterase toxicities can be diagnosed by history, clinical signs, and toxicologic assay. Serum creatine kinase determinations, electromyography, and muscle biopsy are used to confirm the presence of myopathy or myositis. Systemic lupus erythematosus is diagnosed by the presence of systemic signs and positive antinuclear antibody or lupus erythematosus tests or both. Laryngeal paralysis can be diagnosed with direct laryngeal examination. Symptoms of central nervous system disease can be evaluated with distemper titers, cerebrospinal fluid analysis, computed tomographic brain scans, or a combination of these methods. Such diseases are infrequent causes of megaesophagus.

Treatment

The goals in the management of megaesophagus are to identify and treat the primary cause, decrease the frequency of regurgitation, prevent overdistention of the esophagus, provide adequate nutrition, and treat complications such as aspiration pneumonia and esophagitis. Dogs with secondary megaesophagus that can be treated specifically for underlying disease may show improvement of esophageal motility with time; however, responses are variable. Treatment of dogs in which an underlying cause cannot be found is entirely symptomatic.

Cases of focal and generalized MG are treated with long-acting anticholinesterase drugs. Either pyridostigmine bromide, 1 to 3 mg/kg orally every 8 to 12 hours, or neostigmine, 0.04 mg/kg intramuscularly every 6 hours (if oral medication is not tolerated), is effective. If pyridostigmine bromide is administered in the syrup form, it should be diluted at a 50:50 ratio in water to avoid gastric irritation (Shelton, 2002). Improvement of clinical signs accompanied by a decrease in ACh receptor antibody concentration indicates a positive response to treatment. Antibody concentrations should be checked every 4 to 6 weeks to determine the course of the disease and therapy adjusted because spontaneous remissions do occur in a large percentage of dogs. Treatment should be continued until serum antibody titers are within the normal range. If clinical remission does occur, esophageal dilation may resolve completely, and medication can be discontinued; however, relapses do occur. The time course

until remission can vary from 1 month to longer than 1 year. Some dogs with focal MG progress to generalized MG, usually within several weeks of the initial onset of clinical signs. Myasthenia is an immune-mediated disease; thus in most cases immunosuppression with corticosteroid therapy or a steroid-sparing agent such as azathioprine may be warranted. A retrospective study by Bartges, Hansen, and Hardy (1997) evaluated 30 dogs with acquired MG and megaesophagus and discovered that glucocorticoid therapy alone resulted in clinical remission in 67% of cases. Multiple case reports of myasthenics experiencing clinical remission and a decrease in ACh receptor antibody titer after therapy with azathioprine are also available (Dewey et al, 1999). Other immune-directed therapy includes plasmapheresis, the filtration of plasma to rid the body of circulating antibodies. Shelton and associates (1990) evaluated 53 dogs with MG and saw spontaneous clinical and immunologic remission in 89% of dogs treated with anticholinesterase therapy alone. Therefore the use of immunosuppressive therapy is somewhat controversial, and the decision must be made on a case-by-case basis. Aspiration pneumonia should be ruled out or treated before the use of immunosuppressants, and the patient monitored closely for signs of developing infection.

Megaesophagus associated with hypoadrenocorticism resolves with corticosteroid and mineralocorticoid replacement. Immune-mediated polymyositis and polyneuritis and systemic lupus erythematosus may respond to immune suppression. Toxic causes of megaesophagus are treated by removal of the offending agent or the use of specific antidotes or both. Treatment of dysautonomia is limited to symptomatic therapy, including cholinergic drugs to relieve some of the signs of parasympathetic dysfunction, such as bethanechol to improve bladder function and pilocarpine to relieve photophobia. Artificial tears and humidifying the air can help eliminate the dryness associated with eyes and oral and nasal mucous membranes. Symptomatic management of the associated megaesophagus is described in the following paragraphs. Despite supportive care, the prognosis for dysautonomia is poor.

The management of idiopathic megaesophagus, as well as most cases of megaesophagus resulting from neurologic disease, is entirely symptomatic and centers on special feeding techniques. A diet should be formulated using a high-calorie food to provide adequate nutritional intake. Meals should be fed in small portions several times daily and the dog should be in an upright position when eating. This can be accomplished by placing the food on an elevated feeding platform. The owners should be encouraged to construct a "Bailey chair"–like feeding platform. An online support group (www.geocities.com/bailey_chair/) gives instructions on building a "Bailey chair" for upright feedings. The patient should remain in an upright position after feeding for at least 15 to 20 minutes to allow for the effect of gravity moving the food into the stomach. Upright feeding provides surprisingly effective symptomatic control of regurgitation in many dogs.

Because dogs with megaesophagus vary in their ability to swallow foods of various consistencies, the type of diet fed should be tailored for each patient. Liquefied foods tend to flow more easily with gravity than do solids, but liquids stimulate little peristaltic activity and may increase the risk of aspiration upon swallowing. Solids stimulate more peristalsis and perhaps pose less risk of aspiration. Barium contrast radiography using liquid, canned, and dry food may help determine the best consistency of food for a particular patient. Ultimately food trials are the best way of determining the consistency of food to be fed. In the authors' experience, feeding small meatballs made of canned food provides the best symptomatic control.

Some dogs cannot tolerate oral feeding, especially if the esophagus is extremely distended, secondary esophagitis is present, or the patient is debilitated severely. Providing nutritional intake in these instances requires gastrostomy tubes for long-term feeding. Surgical, endoscopic, or nonendoscopic techniques for gastrostomy tube placement can be used. The authors have managed successfully the nutritional needs of large dogs with idiopathic megaesophagus for longer than 1 year using gastrostomy tube feeding. Esophagostomy tubes can exacerbate regurgitation and should not be used.

Aspiration pneumonia should be treated with broad-spectrum antibiotic therapy while culture and sensitivity results of a transtracheal wash are pending. Many dogs with megaesophagus appear to acquire esophagitis, which can worsen the clinical signs. Systemic antacid treatment with drugs such as ranitidine or omeprazole and protectant drugs such as sucralfate help control symptoms in some patients. Many types of drugs have been used unsuccessfully in an attempt to improve motility and esophageal emptying in dogs with megaesophagus. Anticholinergic drugs and calcium channel blocking drugs such as nifedipine have been used to decrease LES pressure. Little if any response has been observed. Anecdotal observations indicate that some dogs with megaesophagus improve clinically when treated with prokinetic drugs such as metoclopramide or cisapride. These observations have been questioned because metoclopramide and cisapride increase motility by binding 5-HT$_4$ (serotonin) receptors on enteric cholinergic neurons, resulting in depolarization and contraction of gastrointestinal smooth muscle; canine esophagus consists of striated muscle. In normal dogs cisapride actually has been shown to decrease or slow the rate of transit of a food bolus through the esophagus (Mears et al, 1996). Therefore cisapride and other prokinetic drugs cannot be recommended to improve esophageal motility in dogs with megaesophagus.

If reflux esophagitis is suspected as a cause of esophageal hypomotility or a complication of megaesophagus, a trial with prokinetic drugs should be considered. These drugs do increase LES pressure, potentially decreasing episodes of reflux and subsequent esophagitis. However, this decision must be made carefully because increasing LES pressure actually can diminish esophageal clearance and perpetuate clinical signs in dogs with megaesophagus.

Bethanechol is a drug that has shown promise in stimulating esophageal propagating contractions in some dogs affected with megaesophagus by directly binding to and stimulating cholinergic (muscarinic) receptors (Diamant and Szezepanski, 1974). This study was per-

formed before the availability of testing for MG; thus it is impossible to know whether the documented improved motility is the result of treating megaesophagus secondary to undiagnosed MG or an indication that cholinomimetics can improve some cases of acquired idiopathic megaesophagus. There is still a population of undiagnosed myasthenics composed of both dogs that have not yet seroconverted and those that are seronegative. Therefore dogs with acquired idiopathic megaesophagus should be treated as myasthenics with acetylcholinesterase inhibitors and immunosuppression as described previously.

Surgical treatment of congenital and idiopathic megaesophagus has not proved beneficial. Myotomy of the LES and techniques to plicate or resect the redundant esophagus actually may worsen the clinical signs. If radiographic evaluation of a patient shows failure of the LES to open and manometric studies confirm elevated sphincter pressures that did not relax in response to a swallow, a modified Heller's esophageal myotomy may be indicated.

Prognosis

The prognosis for megaesophagus is variable and difficult to predict. Successful outcomes depend on early diagnosis and aggressive dietary management. Even with diligent care, owners should be warned that aspiration pneumonia is a frequent and often fatal complication.

Congenital megaesophagus has at best a guarded prognosis for the animal to become a healthy and functional pet. Reported recovery rates vary from 20% to 46%. Some dogs improve with maturity, especially if the condition is recognized early and dietary management is begun before severe and irreversible dilation occurs. For example, most miniature schnauzers acquire improved or normal esophageal function by 6 to 12 months of age (Cox et al, 1980). There is one report of hypertrophic osteoarthropathy (HO), congenital megaesophagus, and no other pulmonary or extrathoracic disease in a German shepherd (Watrous and Blumenfeld, 2002). This dog was euthanized at 6 years of age because of cachexia and HO, but in a single report of a human with achalasia and HO the bone changes regressed after successful surgical treatment of the achalasia. One proposed mechanism is a neurogenic basis, in which chronic vagal dysfunction initiated a reflex arc leading to HO.

Adult-onset idiopathic megaesophagus has a guarded prognosis and is compounded by the presence of aspiration pneumonia. However, some affected dogs respond to symptomatic dietary management. Unfortunately many die of aspiration pneumonia or are euthanized because of persistent regurgitation and debilitation within 5 months of the time of diagnosis. Spontaneous recovery rarely occurs. Several online chat rooms on canine megaesophagus are often helpful for clients with pets with this disease.

The prognosis for dogs with secondary megaesophagus is good if the underlying disease can be treated successfully. This is especially true for MG, in which clinical recovery can be expected to occur in at least 50% of the cases. Megaesophagus appears to respond well to corticosteroid therapy in dogs with hypoadrenocorticism. Megaesophagus secondary to dysautonomia carries a grave prognosis. Dogs with megaesophagus from polyradiculoneuritis, polymyositis, systemic lupus erythematosus, and botulism can recover esophageal function after successful treatment of the primary disease.

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