Laryngeal Paralysis
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Laryngeal paralysis is the lack of abduction of the arytenoid cartilages during inspiration. During expiration, the arytenoid cartilages are passive and therefore expiration is not affected by laryngeal paralysis. The larynx functions to regulate airflow, produce voice and protect the airway during swallowing.

There are two forms of laryngeal paralysis: acquired and congenital. Acquired laryngeal paralysis is most common in Labs, Goldens, Saint Bernards, and Irish Setters. The average age at diagnosis is 9 years, and males are twice as likely to be affected as females. Acquired laryngeal paralysis has also been diagnosed in cats. Any damage to the recurrent laryngeal nerve (which innervates the cricoarytenoideus dorsalis muscle that provides motor to the arytenoid cartilage) can cause laryngeal paralysis but most commonly the etiology is idiopathic.

Congenital laryngeal paralysis is fortunately uncommon and diagnosed in the Dalmatian and Husky (most commonly) in the US. These dogs will present with clinical signs before the age of one. An autosomal dominate mode of transmission has been found in at least one breed with congenital laryngeal paralysis and many affected dogs will have other central neurologic abnormalities at presentation.

Laryngeal paralysis can be difficult to diagnose because signs can be subtle and often are not present in the exam room or are not part of a routine history. Early clinical signs of laryngeal paralysis include: change of voice (hoarse bark), coughing or gagging (especially after eating and drinking), and decreased endurance. These clinical signs are sometimes attributed to normal aging change by many owners. As the disease progresses, often over months to years, you will begin to hear laryngeal stridor. At first this only occurs when the dog is exercising or is very excited (increased respiratory rate induces the stridor). However, the stridorous breathing becomes worse as laryngeal edema and inflammation occur secondary to the turbulent airflow in the larynx due to laryngeal paralysis. Episodes of severe inspiratory dyspnea, cyanosis or syncope can occur in severely affected dogs and these dogs present more commonly in the late spring and early summer as the outdoor temperatures rise. Many dogs will actually present with concurrent signs of heat stroke.

A very important discovery was made in the last few years that dogs affected with acquired, idiopathic laryngeal paralysis are actually suffering from a generalized progressive idiopathic polyneuropathy. A study by Stanley et al. in 2010 looked at dogs diagnosed with laryngeal paralysis and only 25% of dogs had neurologic signs at the time of diagnosis and enrollment in the study. At 6 months and 1 year after diagnosis, 58% and 100% of the dogs, had neurologic signs in the hind limbs (ataxia, decreased proprioception, weakness, and muscle atrophy). One theory is that the recurrent laryngeal and the peroneal nerves are two of the longest nerves in the body and therefore most commonly affected. Although dogs do not progress to hind limb paralysis, this finding is still very important to discuss with owners when their dogs are diagnosed with laryngeal paralysis. There is currently no treatment for the progressive polyneuropathy.

Dogs that present with respiratory distress due to upper airway obstruction should be placed in a cool environment with supplemental oxygen. Dogs that present with acute
cyanosis or collapse due to upper airway obstruction should be intubated or an emergency tracheostomy should be performed. Sedation and steroids are often indicated to help decreased inflammation and acute swelling of the mucosa of the arytenoid cartilages. Fluid administration should be judicious because dogs with severe upper airway obstruction can develop pulmonary edema.

These emergent situations are always very stressful for the dog, the owners and the veterinary staff attending to the patient. If laryngeal paralysis is suspected and has been discussed with the family during a routine exam or consult for respiratory stridor/exercise intolerance, the owners may be more likely to use caution when exercising these dogs and be more aware to present them earlier in the event of respiratory distress. Additionally, a cyanotic dog in distress that presents with a history of collapse while playing ball. Therefore, it is important to take ample time to educate owners about the risk of respiratory distress, collapse or even death for dogs with laryngeal paralysis. Stressing the importance of notifying the ER staff right away that the dog has been diagnosed with suspected laryngeal paralysis. This information can save precious time and save a life.

A definitive diagnosis of laryngeal paralysis is made by direct visualization of the arytenoid cartilages under a light plane of anesthesia (oral laryngoscopy). Ultrasound by a trained radiologist can also be used to diagnose laryngeal paralysis in the awake patient. In a normal dog the arytenoids should abduct with each inspiration. If the plane of anesthesia is too deep the arytenoids will not move at all and can lead to false positive diagnosis. Additionally, paradoxical movement of the arytenoids can occur leading to a false negative diagnosis. Paradoxical movement of the arytenoids occurs due to the change in velocity of the air as it moves through the larynx, and the arytenoids will actually adduct during inspiration and abduct during expiration. Therefore, it is important to know when the dog is inspiring and expiring during your sedated laryngeal exam.

Once the diagnosis of bilateral laryngeal paralysis is made and the discussion of a progressive polyneuropathy, risks and complications of surgery, and discussion of finances has taken place with the owner, surgical treatment can be performed to reposition the arytenoid cartilage in order to decrease airway resistance. Surgery is performed with the goal of decreasing the incidence of respiratory compromise. Decreased respiratory stridor (at rest and with exercise) is often also attained, but owners should be advised that there will still be increased respiratory noise after surgery since surgery is only performed on one side.

There are several surgical techniques described for laryngeal paralysis but unilateral arytenoid lateralization is currently considered the gold standard technique because of its very consistent outcome. Dogs treated with bilateral arytenoid lateralization had a 55 times greater risk of complications and 47 times greater chance of dying than dogs treated with unilateral lateralization (MacPhail 2001). Unilateral lateralization is sufficient to reduce clinical signs of laryngeal paralysis without increasing risk to the patient. Therefore, patients that are only unilaterally affected should not undergo surgery.

Long-term outcome of dogs surgically treated for bilateral laryngeal paralysis is good. After unilateral arytenoid lateralization the risk of aspiration pneumonia is our greatest concern. The risk of aspiration pneumonia is reportedly about 10-21% and 70% of dogs were still alive 5 years after surgery in one study (MacPhail 2001). Poor prognostic indicators of long-term survival are presence of aspiration pneumonia before surgery, development of megaesophagus after surgery, and the need for a temporary tracheostomy (MacPhail 2001).
Dogs treated for laryngeal paralysis are at lifelong risk for aspiration pneumonia, and progressive generalized polyneuropathy with possible esophageal dysfunction.

