EVALUATING THE LAR-PAR PATIENT – MORE TO SWALLOW THAN REALIZED?
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Key Points
- The condition known as “idiopathic” laryngeal paralysis is a common condition in older dogs. Surgical outcomes are generally excellent for alleviation of clinical signs.
- In the majority of cases, the condition represents an early onset of more generalized, progressive neurodegeneration, and renaming to Geriatric Onset Laryngeal Paralysis Polyneuropathy (GOLPP) has been suggested.
- Over two-thirds of affected dogs have esophageal dysfunction at time of presentation for their laryngeal paralysis, based on esophagram and throat-clearing, although less than one third clinically regurgitate at this time.
- About one third of affected dogs have signs of neurological weakness at time of presentation for their laryngeal paralysis. This percentage and severity of signs increase with time.
- It is critical that we fully evaluate these patients. In doing so, not only will we be able to completely characterize the disease, but we will be able to give owners a more accurate prognosis.
- It is also critical that we provide long-term post-operative management to affected dogs to optimize their quality of life.

When acting effectively, the five laryngeal cartilages act to protect the airway from aspiration, modulate airflow, facilitate expectoration and coughing, increase intra-abdominal pressure, and allow vocalization. The rima glottidis airflow is controlled by active abduction or adduction of the paired arytenoids dorsally and tensing of the vocal cords ventrally. The only true abductor muscle is the \textit{m. cricoarytenoideus dorsalis}. All intrinsic laryngeal muscles (except the \textit{m. cricothyroideus}) are innervated via the recurrent laryngeal nerves, which arise from the vagus in the thorax, and course cranially alongside the trachea to the larynx. Laryngeal innervation shows remarkable variation within and between species; and frequent erroneous extrapolations between species have occurred. The pararecurrent laryngeal nerve arises with the recurrent laryngeal nerve, and innervates the cranial thoracic and cervical esophagus.

A late-onset, acquired laryngeal paralysis has been well documented in the literature for almost 40 years, presenting as a common condition in older dogs, particularly the Labrador retriever, but also other purebreds (such as German Shepherds, Golden Retrievers, Australian Shepherds, Borzois, Greyhounds, Newfoundlands, Brittany Spaniels) and mixed breeds. Because a specific cause was not identified, the term “idiopathic laryngeal paralysis” became the universal descriptor. A changed bark is noted about half the time, and two-thirds of dogs have gagging, throat-clearing, ‘choking’ or coughing, usually associated with eating and drinking. The condition is insidious in onset, characterized by signs of upper respiratory obstruction (stridor, dyspnea, exercise intolerance), with exacerbation often leading to severe compromise and collapse. Many dogs will either present as emergencies to the veterinarian, or become emergent upon routine appointment. The acutely distressed patient requires immediate therapy to alleviate their dyspnea and hypoxia, including oxygenation, fluids, sedation, fan cooling.
Surgical intervention is common, usually in the form of a unilateral crico- or thyroarytenoid laryngoplasty, immediately providing effective alleviation of signs of upper respiratory obstruction. The most clinically significant complication is aspiration pneumonia which appears to occur in around 10-24% of cases. Aspiration pneumonia can occur as early as the night of surgery, but can also develop months or even years later. Its appearance has been attributed to the surgical procedure increasing susceptibility to laryngotracheal aspiration, which is a reasonable explanation. Although several different techniques and modifications have been proposed to reduce this complication, no significant reduction in the incidence of aspiration pneumonia has been reported. Luckily, most cases respond well to management of pneumonia, especially when owners are educated to watch for the earliest signs.

A 2-year, prospective study compared esophageal function (via standardized esophagram) in dogs with “idiopathic laryngeal paralysis” with age- and breed-matched controls. The severity of esophageal dysfunction was then compared to see if could be related to the development of aspiration pneumonia during a one year follow-up. Clinical neurologic status was also assessed at every recheck over the study period. A total of 66 dogs were enrolled – 32 affected dogs, 34 controls. After unilateral cricoarytenoid laryngoplasty, affected dogs were re-examined, including thoracic radiography, at 1, 3, 6, and 12 months. Neurologic examinations repeated at 3, 6, and 12 months. The most significant findings of the study were:

1. Seventy percent of the affected dogs had esophageal dysfunction (compared to controls), most notable in the liquid phase.
2. Dysfunction was more pronounced in the cranial esophagus (corresponding to the pararecurrent laryngeal innervation).
3. The 18% of affected dogs that experienced aspiration pneumonia in the study period had significantly worse esophageal dysfunction than those dogs that did not develop aspiration pneumonia.
4. One third of affected dogs had generalized neurologic signs on enrollment, and all dogs had signs of polyneuropathy at study end (12 months).

Investigators concluded that the disorder we have been calling “idiopathic laryngeal paralysis” for many years, is actually a chronic, progressive, polyneuropathy with early manifestations of laryngeal and esophageal dysfunction. These findings have also now been found by others. A more accurate term for the disease may be “geriatric onset laryngeal paralysis polyneuropathy”, or GOLPP.

Recognition of the generalized nature of this condition has probably been hampered by the fact that the laryngeal surgery is usually performed in a referral setting, and dogs return to their regular veterinarian for extended follow up; and that the neurodegeneration is slow and insidious in nature. In such cases, subsequent neurologic deterioration and swallowing issues may not have been linked to the laryngeal dysfunction. Additionally, affected dogs are often in marked respiratory distress and veterinary attention is focused on the upper airway. Without careful and rigorous neurologic assessment, early neurologic dysfunction may be misinterpreted as weakness from hypoxia or orthopedic conditions (which are also common in these dogs).

It is critical that we fully evaluate these patients. In doing so, not only will we be able to completely characterize the disease, but we will be able to give owners a more accurate prognosis. Recommended diagnostics and evaluation protocols include:
Standardized history: A questionnaire is designed so that all relevant questions will be asked in a standardized manner and responses scored. A similar post-op questionnaire can be used for follow up examinations.

Neurologic examination; Orthopedic examination: It is clear that neurologic issues need to be discerned from orthopedic issues, and a careful and complete neurologic examination should always be performed.

Pre-operative standing esophagram: Ideally, evaluation of esophageal function in all phases of swallowing (oropharyngeal, esophageal, gastroesophageal) should be performed in a standing position. An esophageal stanchion can be built, so that dogs can eat naturally. Esophageal transit times can be recorded, and esophageal function and gastro-esophageal reflux scored. Hiatal herniation should also be noted. If a dog has very poor esophageal function, findings can be discussed with the owners in depth, and in consideration of the degree of the dog’s respiratory compromise.

Laryngoscopy: Laryngeal function should be noted on induction using a standard induction protocol, including 1 mg/kg doxapram IV to enhance respiratory excursions.

Muscle and nerve biopsies: At time of laryngeal surgery, biopsies can be taken of the m. cricoarytenoideus dorsalis, and the resulting histopathological examination will provide the gold standard for diagnosis. Biopsies of the m. tibialis cranialis and peroneal nerve will also give the veterinarian and owner a point-in-time status of the dog. Current investigations suggest that these biopsies may be of significant prognostic value.

Anesthesia protocol: In all cases, even if esophageal function is normal on esophagram, the esophagus should be suctioned immediately following induction, intraoperatively and immediately post-operatively, before recovery. Additionally, all dogs are placed on a metoclopramide CRI before surgery (1-2 mg/kg/day) and continued into the next day. Hydromorphone is avoided as a premedicating agent.

Electrodiagnostics: If available, electromyography and motor NCS can be performed, per standardized protocol. Typical time for electrodiagnostics is around 30-45 minutes. Current studies are evaluating changes seen in affected dogs and will eventually be able to report on the value of this test.

It is also critical that we provide long-term management and guidance to the owners of affected dogs following surgery. Owners are dedicated to these pets and are emotionally (and financially!) invested in optimizing their quality of life.

Management of esophageal dysfunction: Based on preliminary results from a positional esophagram study just completed, the recommend feeding angle is a 30 degree incline plane with head up, and maintaining the dog in sitting position for 10 minutes post prandially. Metoclopramide administered per os TID can help most dogs with moderate esophageal dysfunction, and cisapride can be added in dogs with severe dysfunction. There are not reliable results for the effects of these drugs, but most owners feel that metoclopramide is helpful when given before feeding and before bedtime.

Physical therapy: Water treadmill physiotherapy, balancing and coordination exercises are highly recommended for all dogs long-term. The aim is to maintain muscle mass and minimize
proprioceptive deficits in the face of insidious neurodegeneration. Home exercise is also encouraged, with daily long, slow walks.

*Owner education:* The development of owner handouts to inform owners of GOLPP dogs on this disease and its progression is recommended. Owners need to be educated to identify early signs of aspiration pneumonia (inappetance, lethargy, fever), as early treatment is most successful. These dogs are longtime companions and almost always regarded as much-loved members of their human family. As the condition progresses relentlessly over months to several years, euthanasia is typically requested by owners when their pet becomes non-ambulatory, or experiences repeated episodes of aspiration pneumonia from regurgitation, gagging, and/or dysphagia. Occasionally dogs will go into a cart for several months.

*Follow up:* It is vital for us to follow affected dogs out for the remainder of their life – currently recommended every 3 months until their demise. It is already clear that most dogs will progress at a fairly steady rate, with euthanasia requested within 2-3 years. However, some dogs will progress at remarkably rapid rate. There is also a small group of dogs in which we have noted a surprisingly slow rate of neurologic deterioration. We encourage owners to participate in our post mortem donor scheme so that we can analyze brain and spinal cord tissues.

In addition to studying the natural history of this disease, pedigrees and DNA can be collected from affected dogs. We welcome the participation in many parts of our investigations by ACVS board-certified surgeons and their teams.

*References*