Getting Harder to Breathe: Upper Airway Disease in Dogs

Drs. Michael King & Mike Higgins

Brachycephalic Syndrome

Any of the brachycephalic breeds can suffer from this combination of conditions, though most often seen in bulldogs and pugs.

Though brachycephalic syndrome is caused by an overall narrowing of the upper airways, there are three main primary components:

- Stenotic nares
- Elongated Soft palate
- Hypoplastic trachea

Secondary obstruction can then occur to the airway with everted laryngeal saccules, and in more severe cases laryngeal collapse. These form due to chronic negative pressure within the larynx, due to the airway resistance rostrally. For the same reasons these patients are seen to have a higher incidence of hiatal hernia and gastroesophageal reflux (some reports suggest up to 98% of brachycephalics affected).

Presentation

Predominant presenting signs consist of sterterous breathing, and potentially periods of acute respiratory distress. Though this is most often seen from a very early age, it can seemingly worsen suddenly later in life if secondary obstruction occurs. This is why even in relatively mildly affected patients, treatment is recommended to reduce the risk of later issues.

Diagnosis

While stenotic nares can be diagnosed on routine physical examination, the soft palate, and larynx can only be properly assessed under an anesthetized oral examination. As recovering these patients form anesthesia presents some risk, this assessment is often performed immediately prior to surgical intervention.

Radiographs of the chest can help identify hypoplastic trachea (which unfortunately cannot be treated surgically), as the average tracheal diameter in brachycephalic breeds (as a proportion of thoracic inlet size) is only around 12%, compared with 20% in other breeds (<16% is considered hypoplastic). It also can highlight other concurrent lower airway pathology such as

aspiration pneumonia, or non-cardiogenic pulmonary edema, which is commonly seen in such cases.

Radiographs of the cervical region are also indicated in patients where there is suspicion of additional airway obstruction, such as possible neoplasia, or other space- occupying lesions.

Treatment

The goal of surgery is to decrease upper airway resistance, permitting greater airflow, and also decreasing the degree of negative pressure occurring within the rima glottis during inspiration. As airflow is proportional to the radius of the airway to the 4th power, even a relatively minor increase in airway size can dramatically improve breathing.

The nares are formed by several nasal cartilages, with the shape of the dorsolateral cartilage causing the major part of the visible narrowing in brachycephalics. The most common method of treatment is a vertical wedge resection. By removing a lateral wedge of cartilage and mucosal tissue on each of the alar wings, the nostrils are widened significantly. Alternatively an alapexy can be employed to abduct the alar tissues, though is more technically demanding. An older method (the “Trader” technique) achieves increased nostril diameter by excising a portion of the dorsolateral cartilage, and allowing it to heal by second intention. It has been recommended by some authors for use in patients with very small noses.

Unfortunately the narrowing continues to some degree throughout the nasal passages, and frequently includes abnormalities within the conchi. Despite this, correcting the most rostral obstruction alleviates a lot of resistance.

The soft palate is resected back to the level of the tonsillar crypts laterally, and the tip of the epiglottis centrally. It is important to measure this correctly, as insufficient excision results in persistent airway obstruction, but excessive resection can allow nasopharyngeal reflux.

Excision can be done with sharp excision using metzenbaum scissors and apposing the mucosa, with laser, or electrocoagulation devices for hemostasis. There is no difference in outcome between the techniques, and is more down to the experience of the surgeon than the equipment used. Intra- or post-operative dexamethasone (0.5-2mg/Kg IV) can be given to decrease swelling, and help with recovery from anesthesia.

Everted laryngeal saccules are seen in anyway from 40-60% of patients with brachycephalic syndrome. The most effective treatment is to decrease the upper airway obstruction that has caused their formation, with some authors suggesting that with resection of an elongated palate, and correction of stenotic nares, no removal of saccules is necessary. Most surgeons though advocate removal concurrently, which is via simple excision using metzenbaum scissors or a scalpel. Some mild hemorrhage is seen, but usually resolves quickly. Gentle suction of the trachea is sometimes indicated before recovery from anesthesia.

Post-operative Care.
Extubation should be delayed as much as possible to protect the airway during recovery. Supplemental flow-by oxygen is recommended during recovery, and for the first few hours after. An oral gag can also help maintain patency of the oropharynx while the patient recovers control of its airways.

Occasionally if there is significant swelling (particularly if present preoperatively, in patients where they have presented in respiratory distress) a temporary tracheostomy can be needed to help in recovery.

Overall the prognosis for patients with brachycephalic syndrome treated surgically is good with around 90% of cases reporting significant improvement in signs, though sterterous breathing usually continues to some degree.

Patients exhibiting stage II or III laryngeal collapse (medial collapse of the arytenoid cartilages) have a much poorer prognosis, as once this has begun, the changes within the cartilage are irreversible. While an improvement has sometimes been achieved with a laryngeal tieback procedure, often a permanent tracheostomy is required in severe cases.

Laryngeal Paralysis

The larynx consists of a series of cartilages, supported by the hyoid apparatus, that in the healthy dog act to maximize airflow, and protect the airway from food particles. Rostrally the epiglottis, and the paired arytenoids function as gatekeepers to the trachea, with the thyroid and cricoid cartilages providing both structure, and a base for several of the laryngeal muscles to originate from.

Failure of innervation of some of the intrinsic laryngeal muscles is what results in laryngeal paralysis. Most importantly the cricoarytenoid dorsalis muscle, which inserts on the muscular processes of the arytenoids, and acts to abduct the arytenoids during inspiration.

The most common presentation for laryngeal paralysis is older Labradors and Golden Retrievers, with a history of increased respiratory stridor, a change in bark, and potentially episodes of acute respiratory distress. As these signs become exacerbated in warmer weather, laryngeal paralysis often becomes more evident in spring and early summer.

Neurologic Aspects of Laryngeal Paralysis:
The underlying pathogenesis of laryngeal paralysis is thought to be a motor neuropathy occurring in two major forms.

The first form is a hereditary disease that has been best described for the Bouvier des Flandres breed. It is an autosomal-dominant inherited disease most often seen in dogs less than 12 months of age. Other breeds proposed to be affected by this disorder include the Siberian Husky, Bull Terrier, Rottweiler and white-coated German Shepherd dogs. Age of onset is typically between 4 and 6 months of age, and is characterized by neuronal degeneration in the nucleus ambiguus of the brainstem.

Acquired (or idiopathic) laryngeal paralysis is the second and most common form encountered. There is evidence that older dogs presenting with laryngeal paralysis may have underlying generalized neuromuscular disease (NMD). Clinical signs relating to NMD include exercise intolerance, muscle atrophy and loss of segmental spinal reflexes.

Both forms of this neuropathy are diagnosed by historical and clinical features and by ruling-out other causes of laryngeal paralysis (e.g., neuromuscular junction disorders, hypothyroidism). Aside from laryngoscopy, electrodiagnostic evaluation may be performed. Abnormal EMG activity in the cricoarytenoideus dorsalis muscle and neurogenic atrophy observed in biopsy samples of this muscle support the diagnosis. If neurologic signs are present, a complete neurologic exam and electrodiagnostic evaluation should be performed.

Clinical signs tend to progress in both forms of this disease. Absent patellar reflexes have been associated with a worse prognosis in affected dogs due to their association with generalized NMD. Aside from preventing stressful situations and restricting exercise, there is no effective medical treatment to impede the disease progression. However, most patients will do well with corrective surgery. Pet owners should be alerted to monitor for progression of neurologic signs.

Diagnosis

Though a suspicion of laryngeal paralysis can readily be made based on signalment and clinical signs, definitive diagnosis requires an oral exam to observe laryngeal function. As any anesthetic agent can suppress laryngeal function, and assessment needs to take that into consideration. Thiopental is the most commonly described drug with the least observable effect on larynx function, though not always as readily available as other agents. Propofol is therefore often the most appropriate anesthetic used.

Under a light plane of anesthesia, abduction of the arytenoids is assessed in combination with inspiration. This can be deceiving, as in cases with laryngeal paralysis the arytenoids move passively with airflow, though the arytenoids abduct on expiration. The deeper the respiratory effort the easier it is to accurately assess laryngeal function, and administration of Doxapram (1mg/Kg IV) can help increase breathing rate and depth.

Thoracic radiographs are important due to the high risk of aspiration pneumonia in patients with laryngeal paralysis. Esophageal dysfunction and megaesophagus is also common, and thought to be associated with the neuropathy underlying these cases. The presence of megaesophagus can be a poor prognostic indicator, as abnormal esophageal function increases the risk of aspiration dramatically.

Treatment

The goal of therapy is to rapidly improve the clinical signs associated with laryngeal paralysis and increase airflow. The most commonly employed technique is a unilateral cricoarytenoid lateralization, as it has the most consistent outcome. This involves a lateral approach to the larynx, usually on the left. By transecting the thyropharyngeus muscle, and reflecting the thyroid cartilage ventrally, the muscular process of the left arytenoid is identified and exposed. A non absorbable suture is then passed from around the cricoid cartilage and through the muscular process, to simulate the position of the contracted cricoarytenoid dorsalis muscle, as it abducts the arytenoid into a permanently open position.

A variety of techniques for partial laryngectomy have been described, though have poorer long-term outcomes due to recurrence of clinical signs, and increased risk of aspiration pneumonia. Castellated laryngofissure aims to widen the airway by increasing the width of the larynx itself, through a stepped incision ventrally in the cartilage. It is uncommonly performed due to inconsistent outcomes and technical difficulty.

In patients where a cricoarytenoid lateralization is not possible, or if there has been recurrence of signs following treatment, a permanent tracheostomy can be created as a salvage procedure. As this carries its own long-term management issues, it is not recommended as a primary treatment in standard laryngeal paralysis cases.

Prognosis

Overall the outcome following unilateral cricoarytenoid lateralization is very good, with improved exercise tolerance, decreased respiratory noise, and less incidence of respiratory distress. The risk of aspiration pneumonia remains present though, and is seen in between 10 and 30% of dogs at some point following treatment.

References:

1. Bahr, K. L., Howe, L., Jessen, C., & Goodrich, Z. (2014). Outcome of 45 Dogs With Laryngeal Paralysis Treated by Unilateral Arytenoid Lateralization or Bilateral Ventriculocordectomy. Journal of the American Animal Hospital Association, 50(4), 264–272.
2. Nelissen, P., & White, R. A. S. (2012). Arytenoid Lateralization for Management of Combined Laryngeal Paralysis and Laryngeal Collapse in Small Dogs. Veterinary Surgery 41(2), 261-265
3. Stanley, B. J., Hauptman, J. G., Fritz, M. C., Rosenstein, D. S., & Kinns, J. (2010). Esophageal dysfunction in dogs with idiopathic laryngeal paralysis: a controlled cohort study. Veterinary Surgery, 39(2), 139–149.
4. Zikes, C., & McCarthy, T. (2012). Bilateral Ventriculocordectomy via Ventral Laryngotomy for Idiopathic Laryngeal Paralysis in 88 Dogs. Journal of the American Animal Hospital Association, 48(4), 234–244.
5. McKeirnan, K. L., Gross, M. E., Rochat, M., & Payton, M. (2013). Comparison of Propofol and Propofol/Ketamine Anesthesia for Evaluation of Laryngeal Function in Healthy Dogs. Journal of the American Animal Hospital Association, 50(1), 19–26.
6. Nelissen, P., Corletto, F., Aprea, F., & White, R. A. S. (2012). Effect of Three Anesthetic Induction Protocols on Laryngeal Motion during Laryngoscopy in Normal Cats. Veterinary Surgery, 41(7), 876–883.
7. MacPhail, C (2014). Laryngeal Disease in Dogs and Cats. Vet Clin North Am Small Anim Pract, 44(1), 19–31.
8. Thieman KM1, Krahwinkel DJ, Sims MH, Shelton GD. (2010). Histopathological confirmation of polyneuropathy in 11 dogs with laryngeal paralysis. Journal of the American Animal Hospital Association, 46(3), 161-7.
9. Shelton GD. (2010) Acquired laryngeal paralysis in dogs: evidence accumulating for a generalized neuromuscular disease. Veterinary Surgery, 39(2), 137-8.
10. Lodato, D. L., & Hedlund, C. S. (2012b). Brachycephalic Airway Syndrome: Pathophysiology and Diagnosis. Compendium on Continuing Education for the Practicing Veterinarian, 34(7), 1–5.
11. Lodato, D. L., & Hedlund, C. S. (2012a). Brachycephalic Airway Syndrome: Management. Compendium on Continuing Education for the Practicing Veterinarian, 34(8), 1–7.
12. Schuenemann, R., & Oechtering, G. U. (2014b). Inside the Brachycephalic Nose: Intranasal Mucosal Contact Points. Journal of the American Animal Hospital Association, 50(3), 149–158.

13. Schuenemann, R., & Oechtering, G. (2014a). Inside the Brachycephalic Nose: Conchal Regrowth and Mucosal Contact Points After Laser-Assisted Turbinectomy. Journal of the American Animal Hospital Association, 50(4), 237–246.
14. White, R. N. (2011). Surgical management of laryngeal collapse associated with brachycephalic airway obstruction syndrome in dogs. Journal of Small Animal Practice, 53(1), 44–50.