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“Laryngeal Paralysis”

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Laryngeal Paralysis

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Laryngeal paralysis is a disease of the larynx resulting from partial or complete failure of the arytenoid cartilages and vocal folds to abduct during inspiration, and causes mild to severe dyspnoea (Griffin & Krahwinkel, 2005; Griffiths, Sullivan, & Reid, 2001; Hedlund, 2007; Schofield, Norris, & Sadanaga, 2007; R. A. S. White, 1989).

Anatomy
The larynx is comprised of five main cartilages. The epiglottis forms the cranial extent of the larynx, paired arytenoid cartilages form the craniodorsal border, the thyroid cartilage forms both the ventral and lateral boundaries, and the cricoid cartilage forms the dorsal portion of the larynx (Palmisano, 2003). The larynx functions to control airway diameter during respiration; prevent inhalation of food or debris to the lower respiratory tract and enable vocalisation (Hedlund, 2003a). Laryngeal movement is coordinated by intrinsic and extrinsic musculature innervated by branches of the vagus nerve (Hedlund, 2003a).

Function
Airflow and airway resistance are regulated by the cranial opening of the larynx – the rima glottidis or glottic cleft. This narrow kite-shaped opening lies between the vocal folds and vocal processes of the arytenoid cartilages; more commonly known as the glottis (Blood & Studdert, 1999; Dyce et al., 1996; Evans & deLahunta, 1996. Ventrally the rima glottidis is bordered by the vocal folds and epiglottis; laterally by the cuneiform processes of the arytenoid cartilages and aryepiglottic folds, and dorsally by the corniculate processes of the arytenoid cartilages (Kirby, 2004). The size and shape of this aperture is regulated by paired intrinsic musculature. The only intrinsic abductor of the larynx – the cricoarytenoideus dorsalis muscle arises from
the dorsolateral surface of the cricoid cartilage and inserts on the muscular process on the lateral surface of the arytenoid cartilage.

**Innervation**

Innervated by the caudal recurrent laryngeal nerve, contraction of the cricoarytenoideus dorsalis muscle widens the glottis by abducting the arytenoids caudolaterally around the cricoarytenoid articulation (Evans & deLahunta, 1996; Palmisano, 2003).

The caudal recurrent laryngeal nerve is a branch of the recurrent laryngeal nerve, providing motor innervation to all intrinsic laryngeal muscles except the cricothyroideus muscle which is innervated by the cranial laryngeal nerve (Palmisano, 2003).

Adductors of the larynx are comprised of the cricothyroideus muscle which depresses the thyroid and so lengthens the vocal folds; the cricoarytenoideus lateralis and thyroarytenoideus muscles which allow closure of the rima glottidis, and the arytenoideus transversus muscle which serves to close the intra-cartilaginous parts of the rima glottidis (Palmisano, 2003).

**The disease – laryngeal paralysis**

Typically the disease is seen as an acquired form in older large breed dogs including Labrador Retrievers, Afghan Hounds, Saint Bernards and Irish Setters (Griffin & Krahwinkel, 2005; Griffiths et al., 2001; Peterson, Rosin, & Bjorling, 1991; R. A. S. White, 1989). In breeds such as the Bouvier des Flandres, Siberian Husky, Bull Terrier, Dalmatian, Leonberger, Rottweiler and the white-coated German Shepherd, laryngeal paralysis may be congenital (Braund et al., 1994; Braund et al., 1989; Griffin & Krahwinkel, 2005; Griffiths et al., 2001; Hedlund, 2007; MacPhail & Monnet, 2001; Monnet, 2003; Peterson et al., 1991; Venker-van Haagen, 1992).

Potential causes of acquired laryngeal paralysis include neoplasia, trauma, infection, iatrogenic or accidental trauma, and generalised polyneuropathy or polymyopathy. Commonly however, the cause of the acquired form remains undetermined and is described as idiopathic (Griffin & Krahwinkel, 2005; Griffiths et al., 2001; MacPhail & Monnet, 2001).
Pathogenically it is described as a progressive, noninflammatory, degenerative disease of the recurrent laryngeal nerves, consequentially resulting in neurogenic atrophy of the cricoarytenoideus dorsalis muscle, leading to an inability of the arytenoid cartilages and vocal cords to abduct during inspiration (Griffin & Krahwinkel, 2005; Griffiths et al., 2001). Narrowing of the rima glottidis results in clinical signs representative of upper airway obstruction and include inspiratory stridor, exercise intolerance, dyspnoea, change of bark and coughing (Griffin & Krahwinkel, 2005; Griffiths et al., 2001; Peterson et al., 1991).

Acquired laryngeal paralysis is frequently idiopathic and has been described as a progressive, non-inflammatory, degenerative disease of the recurrent laryngeal nerves with resulting neurogenic atrophy of the intrinsic laryngeal musculature (Griffin & Krahwinkel, 2005). Unilateral or bilateral laryngeal paralysis is determined by symmetric or asymmetric paralysis of the arytenoid cartilages further described as partial or complete paralysis (C. Broome et al., 2000; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005).

Laryngeal paralysis can be a manifestation of any generalised neuropathy, myopathy or vagal neuropathy. Myasthenia gravis involving intrinsic laryngeal and pharyngeal musculature has been implicated as a cause of laryngeal paralysis (Gaynor et al., 1997; Griffin & Krahwinkel, 2005; Venker-van Haagen, 1992). Gaynor et al. (1997) reported dogs with acquired laryngeal paralysis as having an increased risk of developing acquired megaoesophagus and such dogs are considered a poor candidate for laryngeal surgery. Although hypothyroidism has been reported concurrently in a number of dogs with laryngeal paralysis, a causal relationship remains to be established.

Clinical signs are generally slow in onset and progressive, reflective of gradual laryngeal obstruction (H.M. Burbidge, 1995; H.M Burbidge et al., 1993; Cathy L. Greenfield, 1987). They include inspiratory stridor; exercise intolerance; change in phonation; gagging, retching and coughing associated with swallowing food and water (Bjorling, 1995; Braund et al.,
1989; H.M. Burbidge, 1995; H.M Burbidge et al., 1993; Griffin & Krahwinkel, 2005). However, some cases may present in acute respiratory distress, cyanosis or collapse after exercise, excitement or hyperthermia (H.M. Burbidge, 1995; Griffin & Krahwinkel, 2005). It has been reported that unilateral paralysis is often asymptomatic, whereby pet dogs appear to adapt to their progressing laryngeal dysfunction by reducing their activity accordingly; hence those presenting with clinically significant respiratory distress are likely to have bilateral disease (H.M. Burbidge, 1995; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; Hedlund, 2007; Palmisano, 2003). By contrast, working and racing dogs can present with unilateral paralysis responsible for clinical signs interfering with their function (H.M. Burbidge, 1995; Cathy L. Greenfield, 1987).

In addition to respiratory signs, some dogs exhibit pelvic limb weakness and bilateral tibial muscle denervation (H. Burbidge et al., 1991a; H.M. Burbidge, 1995; Gaber et al., 1985). Gaber et al. 1985 describe a distal polynuropathy where cases presented with varying degrees of neuropathy in all limbs, exhibiting signs of proprioceptive deficits, paresis and hyporeflexia. It has been proposed that laryngeal paralysis may be a clinical manifestation of a more generalised polyneuropathy, suggesting two forms may exist: an early form seen in young dogs with congenital or hereditary disease; and a delayed-onset form seen in older dogs with idiopathic laryngeal paralysis (Braund et al., 1989; H. Burbidge et al., 1991a; H.M. Burbidge, 1995; Gaber et al., 1985; Griffin & Krahwinkel, 2005; Holt & Harvey, 1994; Jaggy & Oliver, 1994; Jaggy et al., 1994). Complete neurological examination has not always been documented in the literature, however, it has been reported that 2% to 22% of cases presented for surgery had confirmed neurologic disease and the occurrence of posterior weakness post-laryngeal surgery has been recorded (Gaber et al., 1985; Griffin & Krahwinkel, 2005; LaHue, 1989; MacPhail & Monnet, 2001).

Clinical suspicion of laryngeal paralysis can be obtained from the history and presenting clinical signs of a patient. The accepted diagnostic procedure is by visualisation of arytenoid cartilage movement during direct
laryngoscopy with the patient under light general anaesthesia (Bjorling, 1995; H. Burbidge et al., 1991a; H.M. Burbidge, 1995; H.M Burbidge et al., 1993; Gaber et al., 1985; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; C.E. Harvey & O'Brien, 1982; Hedlund, 2003b, 2007; Holt & Harvey, 1994; Kirby, 2004; LaHue, 1989; Lane, 1989; MacPhail, 2004; Palmisano, 2003; Peterson et al., 1991; R. A. S. White, 1989; Wykes, 1983). Achieving the appropriate level of anaesthetic depth is essential to alleviate jaw tone and expose the larynx yet maintain the patient light enough to preserve laryngeal motion. (Bjorling, 1995; H. Burbidge et al., 1991a; H.M. Burbidge, 1995; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; Gross, Dodam, Pope, & Jones, 2002; Hedlund, 2003b, 2007; Jackson, Tobias, Long, Bartges, & Harvey, 2004; Kirby, 2004; Lane, 1989; MacPhail, 2004; Palmisano, 2003; Peterson et al., 1991; Wykes, 1983). The larynx should be examined through several respiratory cycles correlating the stage of respiration to arytenoid and vocal fold movement. In a normally functioning larynx the corniculate processes of the arytenoid cartilages and the vocal folds abduct symmetrically during inspiration and passively adduct during expiration. In laryngeal paralysis the affected arytenoid cartilages and vocal folds show little or no abduction and remain in a paramedian position during inspiration; during expiration the cartilages may appear to flutter in the flow of exhaled air (H. Burbidge et al., 1991a; H.M. Burbidge, 1995; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; Peterson et al., 1991; Wykes, 1983). Asymmetrical abduction of these structures is suggestive of laryngeal hemiplegia (Wykes, 1983). Paradoxical movements can occur and although they may reflect a normally functioning larynx, on careful inspection it is apparent that maximal abduction is not occurring during inspiration (Hedlund, 2007). The vocal cords are drawn toward midline due to negative pressure created during inhalation and then appear to abduct either when they passively return to their original position at the end of inspiration or from being forced outward during exhalation (H.M. Burbidge, 1995; Gross et al., 2002).
Various anaesthetic protocols have been described in the literature, most recently Gross et al. (2002) and Jackson et al. (2004). Gross et al. (2002) compared intravenous thiopental, intravenous propofol and a combination of intravenous ketamine and diazepam for assessing laryngeal function. It was concluded that thiopental and propofol were superior to the ketamine-diazepam combination by effectively alleviating jaw tone allowing better exposure of the larynx (Griffin & Krahwinkel, 2005; Gross et al., 2002). Similarly, Jackson et al. (2004) conclude that intravenous thiopental alone and intramuscular acepromazine with intramuscular butorphanol plus isoflurane by mask had the least effect on laryngeal motion (Griffin & Krahwinkel, 2005). Other anaesthetic protocols examined in this study included, intravenous propofol, intravenous ketamine plus intravenous diazepam, intramuscular acepromazine plus intravenous thiopental or intramuscular acepromazine plus intravenous propofol; all of which were reported to depress laryngeal motion and could lead to misdiagnosis of laryngeal paralysis (Griffin & Krahwinkel, 2005; Jackson et al., 2004).

The respiratory stimulant doxapram hydrochloride has been administered to lightly anaesthetised patients to help differentiate normal dogs from those with laryngeal paralysis. In patients with laryngeal paralysis doxapram administration resulted in paradoxical arytenoid motion in response to respiration and a decrease in glottal area (Griffin & Krahwinkel, 2005; MacPhail, 2004; Tobias et al., 2004). Affected patients may require endotracheal intubation during examination, with extreme glottic constriction resulting from vigorous respiration (Griffin & Krahwinkel, 2005; Tobias et al., 2004).

**Evaluation for concurrent disease**

Patients suspected of having laryngeal paralysis also require evaluation for concurrent disease. Diagnostic work-up should include a complete blood count, serum chemistry analysis and urinalysis. Thoracic radiographs to identify existing lower respiratory disease, including aspiration pneumonia, cardiogenic or noncardiogenic pulmonary oedema, mediastinal or thoracic inlet masses, megaoesophagus and pulmonary metastases. Cervical
radiographs are recommended to screen for cervical masses, tracheal collapse or deviation, intraluminal laryngeal or tracheal masses. Esophagography should be considered if megaoesophagus is suspected. Serum acetylcholine receptor antibody tests may be performed on those candidates with clinical suspicion of myasthenia gravis and thyroid function tests in those patients considered to be hypothyroid. A complete neurologic examination should be performed on all patients with laryngeal paralysis. Those with neurologic abnormalities may be considered candidates for electrophysiology, muscle and nerve biopsies (H.M. Burbidge, 1995; Griffin & Krahwinkel, 2005; Kirby, 2004; Peterson et al., 1991).

Treatment
Treatment options for laryngeal paralysis usually involve symptomatic medical therapy to stabilise the patient, with surgery the preferred definitive therapy (H. Burbidge et al., 1991a; H.M. Burbidge, 1995; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; Peterson et al., 1991). Medical management depends on the severity of the patient at presentation. Severe respiratory distress requiring emergency treatment involves supplemental oxygen, cooling, sedation, a non-stressful environment and some authors advocate corticosteroids (Hedlund, 2007). Emergency general anaesthesia and endotracheal intubation alone or with surgery or temporary tracheostomy are sometimes required. Some patients may have postobstructional pulmonary oedema for which furesomide may be helpful or aspiration pneumonia requiring antibiotic therapy (Hedlund, 2007). Milder presenting cases may require exercise restriction, stress avoidance and caloric restriction (Bjorling, 1995; H.M. Burbidge, 1995; Cathy L. Greenfield, 1987; Griffin & Krahwinkel, 2005; Kirby, 2004; Peterson et al., 1991).

The aim of laryngeal surgery is to relieve airway obstruction by enlarging the rima glottidis and prevent subsequent dynamic airway collapse (H.M. Burbidge, 1995; Griffin & Krahwinkel, 2005). A number of surgical techniques have been developed for the treatment of laryngeal paralysis in dogs.
Unilateral arytenoid laryngoplasty is widely accepted as the standard surgical procedure for laryngeal repair (Bjorling, 1995). It has been shown to provide similar clinical improvement to bilateral repair, but with a lower incidence of aspiration pneumonia due to a narrower resultant lumen (Bjorling, 1995; Love, Waterman, & Lane, 1987; MacPhail & Monnet, 2001). In unilateral arytenoid laryngoplasty, the size and shape of the rima glottic area have been influenced by suture placement and suture tension. Greater enlargements in glottic area have been achieved in unilateral cartilage laryngoplasty with suture placement from arytenoid-to-cricoid cartilage (cricoarytenoid laryngoplasty) or a combination of arytenoid-to-thyroid and arytenoid-to-cricoid cartilages (cricothyroarytenoid laryngoplasty) (Bureau & Monnet, 2002; Demetriou & Kirby, 2003; Griffiths et al., 2001; Lussier, Flanders, & Erb, 1996). These have been described as more physiologic suturing techniques replicating the same function and location as the cricoarytenoideus dorsalis muscle (Bureau & Monnet, 2002; Demetriou & Kirby, 2003; Griffin & Krahwinkel, 2005; Lussier et al., 1996; Payne, Martin, & Rigg, 1990).

Complications and Prognosis

Complication rates of 10 to 28% are reported for unilateral arytenoid cartilage lateralisation with a mortality rate of 13.8% (White 1989, Demetriou & Kirby, 2003; Griffin & Krahwinkel, 2005; MacPhail & Monnet, 2001).

Greenfield (1987) quotes that unilateral arytenoid cartilage lateralisation with a thyroartenoid suture gave good results with 89% of patients being functional companion animals after surgery. However 11% required bilateral surgery for resolution of clinical signs. Similarly, Snelling and Edwards (2003) report an 87.7% clinical improvement in dogs treated with unilateral cricoarytenoid cartilage laryngoplasty in the six months following surgery.

Aspiration pneumonia has been reported as a serious consequence of laryngeal surgery. Failure to protect the airway appropriately due to excessive tie-back may lead to aspiration of food, water or saliva. In addition, the cranial laryngeal nerve supplies sensory innervation to the larynx, thereby, desensitisation of the laryngeal mucosa may contribute to
the potential for aspiration pneumonia (H.M Burbidge et al., 1993; Demetriou & Kirby, 2003). Progressive neuromuscular disease secondary to polyneuropathy may also be a major contributing factor in the development of aspiration pneumonia secondary to oesophageal dysfunction (Ross et al., 1991; Smith et al., 1986). MacPhail and Monnet (2001) reported an incidence of aspiration pneumonia of 33/140 cases (23.6%) which underwent laryngeal surgical repair over a 13 year period. Nineteen of these were in the first two weeks following surgery and nine cases were recognised more than one year following surgery. Dysphagia, poor laryngeal function and a permanently opened glottis are all factors which will predispose a patient to the risk of aspiration pneumonia throughout the rest of their life (Ross et al., 1991; Trout et al., 1994). MacPhail and Monnet (2001) found that dogs with neurologic disease were 3.28 times more likely to die of complications related to laryngeal paralysis such as aspiration pneumonia or respiratory distress. Recurrence of clinical signs because of prosthesis failure in dogs receiving UAL surgery has been reported as 5% (5/109 dogs) (MacPhail & Monnet, 2001)
References:


