

Laryngeal Paralysis in Dogs

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Abstract: Laryngeal paralysis is a common cause of upper airway obstruction in large-breed dogs. Although congenital forms have been reported, the disease is usually an acquired condition in older dogs. Clinical signs include voice change, inspiratory stridor, and dyspnea. Laryngeal paralysis is diagnosed by observing the absence of arytenoid abduction during laryngeal examination under a light plane of anesthesia. The most common method of surgical treatment is unilateral arytenoid lateralization. Most dogs experience significant improvement in respiration following surgery; however, they have an increased risk of aspiration pneumonia for the remainder of their lives.

Laryngeal paralysis is a well-recognized disease of large-breed dogs that results in upper airway obstruction and dyspnea. The condition results from dysfunction of the caudal laryngeal nerves, which are the terminations of the recurrent laryngeal nerves. The caudal laryngeal nerves provide innervation to all the muscles of the larynx except the cricothyroideus muscle. Dysfunction of these nerves results in the loss of arytenoid abduction by the cricoarytenoid dorsalis muscle and the inability to actively constrict the glottis or relax the vocal folds¹ (FIGURES 1 AND 2).

Etiology

Laryngeal paralysis can be congenital or acquired. A hereditary form has been described in Bouvier des Flandres, dalmatians, rottweilers, and Siberian huskies and is usually reported in dogs younger than 1 year.²⁻⁵ Acquired laryngeal paralysis may result from trauma or iatrogenic injury to the recurrent laryngeal nerve (e.g., during thyroidectomy) or compression of the recurrent laryngeal nerve by a cranial mediastinal or cervical mass.⁶ More commonly, however, laryngeal paralysis is classified as idiopathic in older dogs. Although the underlying etiology is unknown, idiopathic laryngeal paralysis is most likely part of a generalized peripheral neuropathy.⁷ In one recent study, muscle and peripheral nerve biopsy samples obtained from 11 dogs with acquired

laryngeal paralysis displayed neurogenic atrophy of the cranial tibial muscle and axonal degeneration of the peroneal nerve in all cases, regardless of whether the dogs had signs of peripheral neuropathy.⁸ Within 2 years after diagnosis of laryngeal paralysis, clinical signs of generalized lower motor neuron disease were

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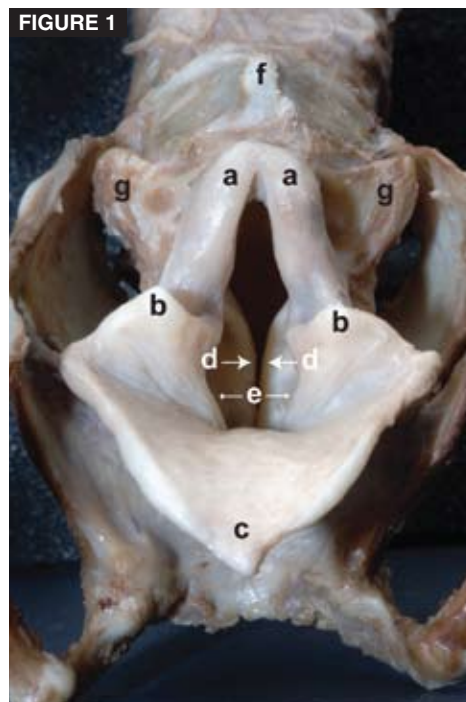


FIGURE 1
Cranial view of a dissected canine larynx. (a) Corniculate process of arytenoid cartilage, (b) cuneiform process of arytenoid cartilage, (c) epiglottis, (d) vocal fold, (e) laryngeal ventricles, (f) cricoid cartilage, (g) muscular process of arytenoid cartilage.

present in all dogs in the study.⁸ Although laryngeal paralysis has been reported in dogs with hypothyroidism, the association between the two conditions is unknown.^{9,10} Myasthenia gravis has also been suggested as a cause of laryngeal paralysis in dogs.¹¹

Signalment and Clinical Signs

Laryngeal paralysis is most commonly reported in older, large-breed dogs, especially Labrador retrievers.^{9,12-14} The average age at the time of presentation is approximately 10 years.^{9,12,14} Males are affected more frequently than females.¹²⁻¹⁴ Clinical signs progress as laryngeal dysfunction becomes more severe. Early in the disease process, owners may notice a voice change, inspiratory stridor, and exercise intolerance. Owners may initially believe that the dog's reluctance to move is simply a sign of aging. Dysphagia can also occur, possibly in association with peripheral neuropathy.^{9,14} Owners may also report vomiting; however, they may actually be seeing regurgitation from concurrent esophageal disease or gagging and retching from a soft palate that has elongated as a result of inspiratory dyspnea. Once the laryngeal muscles are paralyzed bilaterally, dogs may develop severe dyspnea, cyanosis, and syncope. Exercise, obesity, excitement, and increased ambient temperature can exacerbate clinical signs, leading to an emergency presentation.⁹ Affected dogs may develop pneumonia or pulmonary edema, which can contribute to respiratory distress. Inability to constrict the glottis properly during swallowing, regurgitation, or vomiting increases the risk of aspiration. Pulmonary edema can develop in cases of upper airway obstruction as a result of changes in intrathoracic pressure and hypoxia, which cause increased permeability of alveolar capillary membranes.^{15,16}

Diagnosis

If an affected dog is stable, it should undergo a thorough physical examination. The thorax should be auscultated for evidence of pneumonia or pulmonary edema, such as harsh crackles, wheezes, or rales, and for cardiac murmurs or arrhythmias. Arterial pulses should be palpated for rate, rhythm, symmetry, and strength to assess for cardiovascular abnormalities that

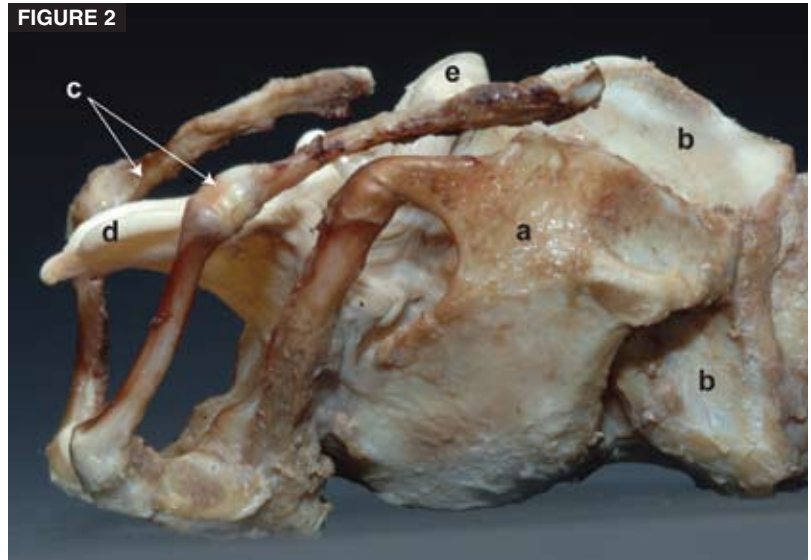


FIGURE 2
Lateral view of a dissected canine larynx. (a) Thyroid cartilage, (b) cricoid cartilage, (c) hyoid apparatus, (d) epiglottis, (e) corniculate process of arytenoid cartilage.

could contribute to exercise intolerance. A complete neurologic examination should be performed to evaluate for signs of polyneuropathy, such as decreased postural reactions, deficits in spinal reflexes, and cranial nerve abnormalities.⁷

A rectal temperature should be obtained, and all dogs should be evaluated for systemic signs of heatstroke, such as petechial hemorrhages associated with disseminated intravascular coagulation, excessive panting, collapse, hyperemic mucous membranes, and abnormalities in mentation, regardless of body temperature at time of presentation.^{17,18} The primary means of heat loss in dogs is evaporation while panting. Dogs affected by acute signs of laryngeal paralysis are more susceptible to hyperthermia due to a lack of heat dissipation through an obstructed respiratory tract. Heatstroke from sustained hyperthermia can progress to multiorgan failure and death.^{17,18} If the body temperature is $\geq 106^{\circ}\text{F}$ (41°C) or systemic signs of heatstroke are evident, additional diagnostics (e.g., coagulation panels, immediate evaluation of glucose and electrolytes) and supportive treatment should be instituted.

Complete blood count and serum biochemistry profile results are typically normal unless concurrent diseases are present. In dogs with

QuickNotes

Acquired laryngeal paralysis may be associated with a generalized peripheral neuropathy.

peripheral weakness, exercise intolerance, megaesophagus, or other signs of generalized polyneuropathy, free thyroxine and endogenous thyroid-stimulating hormone concentrations are measured to rule out hypothyroidism, and acetylcholine receptor antibody titers are measured to rule out myasthenia gravis.^{7,19} The association of laryngeal paralysis with hypothyroidism or myasthenia gravis is unclear, however, as medical treatment for either of these conditions is unlikely to restore laryngeal nerve function.

Thoracic radiography is important for ruling out other causes of dyspnea and exercise intolerance and for determining whether concurrent conditions are present in dogs with laryngeal paralysis. The lung fields should be assessed for evidence of aspiration pneumonia and noncardiogenic pulmonary edema, which can occur with upper airway obstruction. Dogs with laryngeal paralysis from polyneuropathy or neuromuscular junction disease such as myasthenia gravis may develop megaesophagus, which significantly increases the likelihood of aspiration pneumonia^{11,12} (FIGURE 3). A contrast esophagram with videofluoroscopy may be required to make a definitive diagnosis of decreased esophageal motility.²⁰ The risk

of aspiration largely outweighs the diagnostic benefits of contrast esophagography; therefore, this procedure is not performed routinely in dogs with laryngeal paralysis.

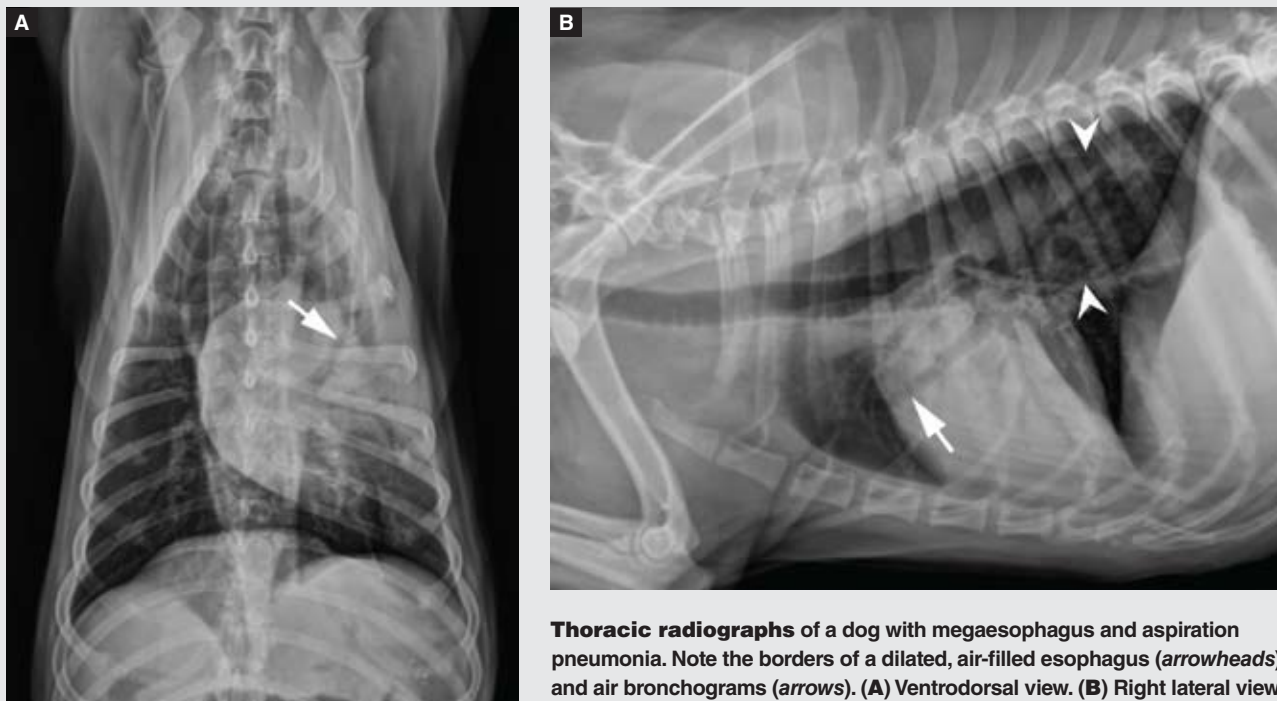
Laryngeal paralysis is most commonly diagnosed with transoral laryngoscopy under a light plane of anesthesia. Excessive administration of any anesthetic can inhibit laryngeal motion; however, some drugs may reduce arytenoid abduction under a light plane of anesthesia. In a comparison of seven different anesthetic protocols,²¹ acepromazine plus thiopental, acepromazine plus propofol, and ketamine plus diazepam resulted in no laryngeal motion in 67%, 50%, and 50% of normal dogs, respectively. Thiopental and propofol as single agents inhibit laryngeal motion less than these drug combinations.^{21,22} However, compared with propofol, thiopental as a single agent results in significantly more arytenoid motion during inspiration and is therefore preferred for evaluation of laryngeal function.^{21,22} Often, dogs receive acepromazine when they present with anxiety and respiratory distress. In the comparison study, laryngeal function was evident in all normal dogs that received acepromazine and butorphanol sedation and were

QuickNotes

Every dog suspected of having laryngeal paralysis should undergo thoracic radiography.

FIGURE 3

Thoracic Radiographs.



Thoracic radiographs of a dog with megaesophagus and aspiration pneumonia. Note the borders of a dilated, air-filled esophagus (arrowheads) and air bronchograms (arrows). (A) Ventrodorsal view. (B) Right lateral view.

BOX 1

Anesthetic Regimens for Diagnosing Laryngeal Paralysis in Dogs²¹

Preoxygenate for 3 to 5 minutes before induction.

- ▶ Thiopental (12–16 mg/kg IV to effect)
- ▶ Propofol (4.5–7 mg/kg IV slowly to effect) and doxapram (1 mg/kg IV)
- ▶ Acepromazine (0.2 mg/kg IM) and butorphanol (0.4 mg/kg IM) 20 minutes before mask induction with isoflurane

QuickNotes

In dogs with laryngeal paralysis, paradoxical movement can be mistaken for active arytenoid abduction during laryngeal examination.

examined under a light plane of anesthesia induced by mask inhalation of isoflurane.²¹ In animals in which laryngeal function has been depressed by sedatives and opioids, doxapram (1 mg/kg) can be administered intravenously to stimulate respiration²³ (BOX 1).

Although a portable laryngoscope can be used to visualize the rima glottidis, retraction of the tongue and pressure on the epiglottis with the laryngoscope blade may affect laryngeal function. Therefore, many clinicians prefer to use a transoral video endoscope. Laryngeal paralysis has also been diagnosed with transnasal laryngoscopy and laryngeal ultrasound.^{24,25}

If possible, blood oxygen saturation should be monitored with a pulse oximeter during laryngoscopy to ensure that the hemoglobin saturation remains $\geq 95\%$.²⁶ Flow-by oxygen can be administered by attaching flexible tubing from an oxygen source to the blade of the laryngoscope or to the insufflation port of the video endoscope to reduce the risk of hypoxia. During laryngeal examination, laryngeal motion should be correlated with the phase of respiration. It is helpful to have an assistant call out when each inspiration and expiration occurs. In normal dogs, the rima glottidis remains open at rest, closes slightly during expiration, and opens widely during inspiration. Inability of the arytenoid cartilages to abduct during inspiration is diagnostic for laryngeal paralysis. In questionable cases, doxapram is administered intravenously.²³ Respiration is usually stimulated within 8 seconds after administration.

Unless the examiner is aware of the phase of respiration, it is easy to mistake paradoxical movement of the larynx for active abduction. Lack of arytenoid cartilage abduction during inspiration narrows the rima glottidis, increasing resistance to airflow. Rapid, forceful inspiration creates negative pressure within the larynx, which pulls the flaccid arytenoid cartilages medially, worsening the obstruction.²⁷ The cartilages are forcefully separated by airflow as the animal exhales. Therefore, dogs with laryngeal paralysis and paradoxical motion have inward movement of the arytenoid cartilages on inspiration and outward, passive movement of the cartilages during expiration. Intubation may be required in some patients with severe paradoxical motion and resultant hypoxia.²³

Medical Management

Dogs that present with acute cyanosis or in collapse require emergency treatment. Supplemental oxygen should be provided to help alleviate hypoxia. An intravenous catheter should be placed for administration of fluid and medications. Severely dyspneic or anxious dogs may require sedation with acepromazine (0.005 to 0.02 mg/kg IV) and butorphanol (0.2 to 0.4 mg/kg IV) or other sedatives. If laryngeal edema is suspected, an antiinflammatory dose of a glucocorticoid such as dexamethasone (0.1 to 0.5 mg/kg) or prednisolone sodium succinate (0.5 to 1 mg/kg) can be administered intravenously. Dogs that are significantly hyperthermic ($\geq 106^\circ\text{F}$ [41°C]) are treated with sedatives, IV fluids, cool water baths, and fans. The rectal temperature should be monitored continuously until it has stabilized within a normal range and external cooling has been discontinued. Dogs that are cyanotic, severely dyspneic, or hypoxic ($\text{SpO}_2 < 95\%$) despite supplemental oxygen therapy may require intubation and light anesthesia until laryngeal swelling resolves. If an intubation period of several hours or longer is expected, a tracheostomy tube should be placed to avoid exacerbation of laryngeal swelling from the endotracheal tube and prolonged periods of anesthesia.²⁸ It is possible for severe cases to progress to respiratory muscle fatigue, which may require mechanical ventilation.²⁹ There is no reliable bedside measurement for detection of respiratory muscle fatigue; the diagnosis is based on changes in breathing patterns, such as inward movement of the abdomen during inspi-

VIDEO



To see videos of normal and paralyzed laryngeal abduction, please visit CompendiumVet.com.

ration, uncoordinated alterations between ribcage and abdominal movements, and increased PaCO₂ on blood gas analysis.²⁹

Dogs that have mild clinical signs or are asymptomatic at rest may be managed conservatively by reducing stress, excitement, and exposure to high ambient temperatures and with weight loss as needed. Owners should be informed that laryngeal paralysis is usually progressive and that many dogs require surgery as clinical signs become more severe or quality of life is affected.

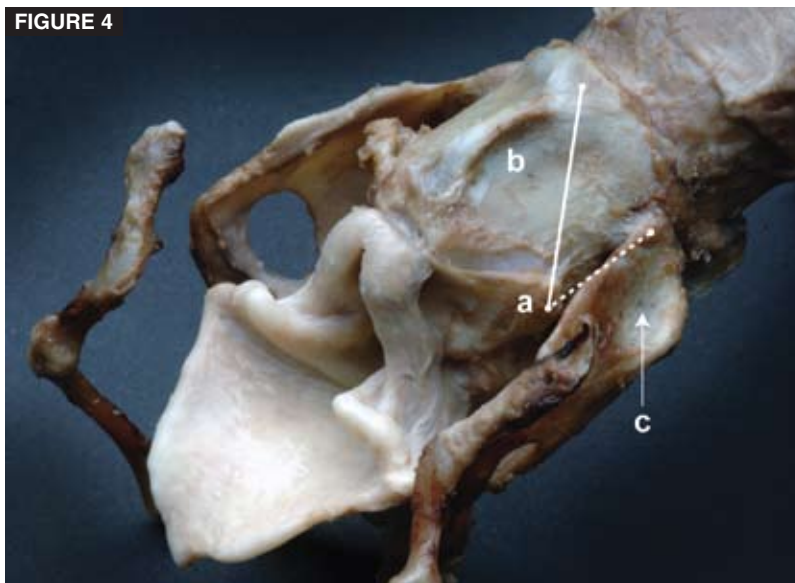
Surgical Treatment

The goal of surgery is to enlarge the size of the rima glottidis to decrease resistance to airflow during inspiration. Surgical techniques include unilateral arytenoid lateralization (UAL), partial arytenoidectomy, vocal fold resection, castelated laryngofissure, and muscle–nerve pedicle transposition.^{30–32} Some dogs may require concurrent soft palate resection because prolonged negative airway pressure can increase soft palate length and thickness. Castelated laryngofissure is rarely performed, and muscle–nerve pedicle transposition has not been evaluated in dogs with spontaneous laryngeal paralysis; therefore, these procedures are not described in this article.

In animals undergoing vocal fold resection for laryngeal paralysis, the vocal fold and process are removed unilaterally or bilaterally. The procedure is often performed transorally with scissors. If bilateral vocal cordectomy is performed, the ventral 1 to 2 mm of the vocal fold should be left in place to reduce the risk of scar formation and subsequent glottal stenosis. Partial arytenoidectomy involves unilateral resection of the corniculate process of the arytenoid cartilage. This procedure can also be performed through a transoral approach with cup biopsy forceps and may be combined with a vocal fold resection. In one study,¹² complications were reported in 40% of dogs undergoing unilateral laryngectomy (arytenoidectomy, vocal cordectomy, or a combination of both) for treatment of laryngeal paralysis, and 30% of the dogs died from respiratory-related causes.

UAL is the most commonly performed procedure for laryngeal paralysis.^{12,14} With this technique, a suture is placed between the arytenoid and cricoid or thyroid cartilages to prevent inward motion of the arytenoid car-

FIGURE 4



Dorsolateral view of a dissected canine larynx. (a) Muscular process of arytenoid cartilage, (b) cricoid cartilage, (c) thyroid cartilage, (solid line) suture placement for cricoarytenoid lateralization, (broken line) suture placement for thyroarytenoid lateralization.

tilage during inspiration³³ (FIGURES 4 AND 5). Active abduction of the arytenoid with the suture is not required to reduce laryngeal airway resistance.^{34,35} If the soft palate is elongated, it is resected before recovery from anesthesia. Bilateral arytenoid lateralization increases the risk of postoperative complications and respiratory-related death and is not recommended.¹¹

Complications are reported in 10% to 28% of dogs that undergo UAL (BOX 2) and include aspiration pneumonia (8% to 33%), coughing and gagging (16%), suture failure or return of clinical signs (4% to 8%), gastric dilatation–volvulus (4%), respiratory distress (2% to 4%), and sudden death (3%).^{12,14,36} Aspiration pneumonia may occur shortly after surgery or at

BOX 2

Complications of Unilateral Arytenoid Lateralization

- ▶ Aspiration pneumonia
- ▶ Coughing/gagging
- ▶ Surgical repair failure
- ▶ Respiratory distress
- ▶ Gastric dilatation–volvulus
- ▶ Seroma formation
- ▶ Sudden death

QuickNotes

Administration of doxapram during laryngeal examination facilitates differentiation of laryngeal paralysis from drug-induced laryngeal dysfunction.

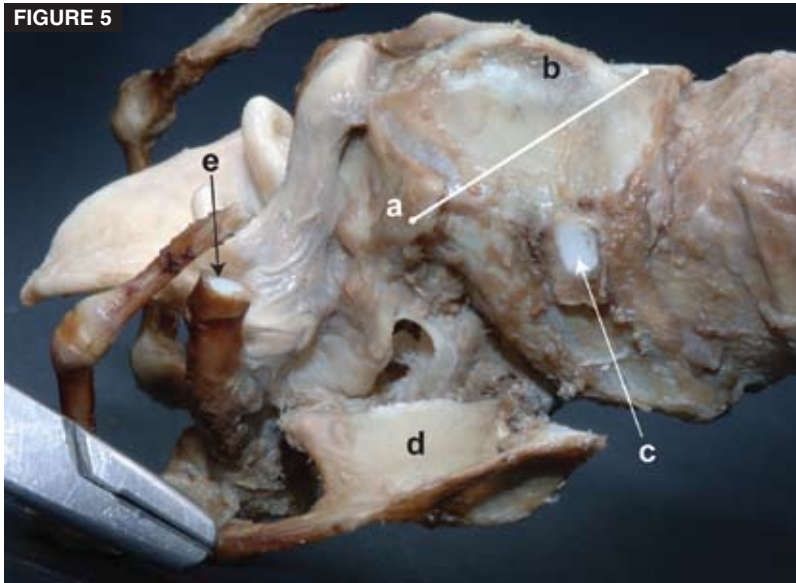


FIGURE 5
Lateral view of a dissected canine larynx. (a) Muscular process of arytenoid cartilage, (b) cricoid cartilage, (c) cricothyroid articulation, (d) thyroid cartilage retracted laterally, (e) articulation of thyroid cartilage and thyrohyoid bone, (solid line) suture placement for cricoarytenoid lateralization.

any time for the remainder of the dog's life. The use of metoclopramide reduces the risk of perioperative aspiration pneumonia.³⁶

Median survival times after UAL range from 1 to 5 years, with approximately 14% of dogs dying from diseases related to the respiratory tract.^{12,14} Factors associated with a higher rate of complications or death include increasing age, placement of a temporary tracheostomy tube, and presence of concurrent respiratory tract abnormalities, postoperative megaesoph-

agus, and neurologic disease.¹² In one study, five of six dogs that developed megaesophagus in conjunction with aspiration pneumonia died.¹² Because polyneuropathy is suspected as an underlying etiology for laryngeal paralysis, affected dogs should be monitored frequently for evidence of neuromuscular weakness and esophageal dysfunction. The association between temporary tracheostomy tube placement and increased postoperative complications should be interpreted with caution because dogs that require tracheostomy tubes are likely to be in critical condition. Clinicians should not hesitate to place a tracheostomy tube in animals with severe inspiratory dyspnea.

Despite complications, approximately 90% of dogs have a reduction in respiratory signs and improved exercise tolerance after UAL. Most owners report an improvement in quality of life and are satisfied with their decision to go to surgery.^{12,14}

Conclusion

Laryngeal paralysis is a common cause of upper airway obstruction in older, large-breed dogs and is likely associated with a generalized polyneuropathy in most animals. Surgical therapy is frequently indicated, and UAL is currently the recommended treatment. Respiratory signs significantly improve in most patients after surgery; however, postoperative complication rates can be high, and patients have a lifelong risk of developing respiratory tract disease. **C**

QuickNotes

Aspiration pneumonia is the most common complication after surgery for laryngeal paralysis.

References

1. Evans HE, Kitchell RL. Cranial nerves and cutaneous innervation of the head. In: Evans HE, ed. *Miller's Anatomy of the Dog*. Philadelphia: WB Saunders; 1993:953-987.
2. Venker-van Haagen AJ, Bouw J, Hartman W. Hereditary transmission of laryngeal paralysis in Bouviers. *JAAHA* 1981;17:75-76.
3. Braund KG, Shores A, Cochrane S, et al. Laryngeal paralysis-polyneuropathy complex in young dalmatians. *Am J Vet Res* 1994; 55:534-542.
4. Mahony OM, Knowles KE, Braund KG, et al. Laryngeal paralysis-polyneuropathy complex in young rottweilers. *J Vet Intern Med* 1998;12:330-337.
5. Polizopoulou ZS, Koutinas AF, Papadopoulos GC, et al. Juvenile laryngeal paralysis in three Siberian husky x Alaskan malamute puppies. *Vet Rec* 2003;153:624-627.
6. Klein MK, Powers BE, Withrow SJ, et al. Treatment of thyroid carcinoma in dogs by surgical resection alone: 20 cases (1981-1989). *JAVMA* 1995;206:1007-1009.
7. Jeffery ND, Talbot CE, Smith PM, et al. Acquired idiopathic laryngeal paralysis as a prominent feature of generalised neuromuscular disease in 39 dogs. *Vet Rec* 2006;158:17.
8. Thieman KM, Krahwinkel DJ, Shelton D, et al. Laryngeal paralysis: part of a generalized polyneuropathy syndrome in older dogs. *Vet Surg* 2007;36:E26.
9. Burbidge HM. A review of laryngeal paralysis in dogs. *Br Vet J* 1995;151:71-82.
10. Jaggy A, Oliver JE, Ferguson DC, et al. Neurological manifestations of hypothyroidism: a retrospective study of 29 dogs. *J Vet Intern Med* 1994;8:328-336.
11. Dewey CW, Bailey CS, Shelton GD, et al. Clinical forms of acquired myasthenia gravis in dogs: 25 cases (1988-1995). *J Vet Intern Med* 1997;11:50-57.
12. MacPhail CM, Monnet E. Outcome of and postoperative complications in dogs undergoing surgical treatment of laryngeal paralysis: 140 cases (1985-1998). *JAVMA* 2001;218:1949-1956.
13. Snelling SR, Edwards GA. A retrospective study of unilateral arytenoid lateralisation in the treatment of laryngeal paralysis in 100 dogs (1992-2000). *Aust Vet J* 2003;81:464-468.
14. Hammel SP, Hottinger HA, Novo RE. Postoperative results of unilateral arytenoid lateralization for treatment of idiopathic laryngeal paralysis in dogs: 39 cases (1996-2002). *JAVMA* 2006;228:1215-1220.
15. Algren JT, Price RD, Buchino JJ, et al. Pulmonary edema associated with upper airway obstruction in dogs. *Pediatr Emerg Care* 1993;9:332-337.
16. John PJ, Mahashur AA. Pulmonary oedema associated with airway obstruction. *Can J Anaesth* 1991;38:139-140.
17. Bruchim Y, Klement E, Saragusty J, et al. Heat stroke in dogs: a retrospective study of 54 cases (1999-2004) and analysis of risk factors for death. *J Vet Intern Med* 2006;20:38-46.
18. Flournoy WS, Macintire DK, Wohl JS. Heatstroke in dogs: clini-

cal signs, treatment, prognosis, and prevention. *Compend Contin Educ Pract Vet* 2003;25:422-431.

19. Shelton GD. Myasthenia gravis and disorders of neuromuscular transmission. *Vet Clin North Am Small Anim Pract* 2002;32:189-206, vii.
20. Washabau RJ, Hall JA. Diagnosis and management of gastrointestinal motility disorders in dogs and cats. *Compend Contin Educ Pract Vet* 1997;19:721-737.
21. Jackson AM, Tobias K, Long C, et al. Effects of various anesthetic agents on laryngeal motion during laryngoscopy in normal dogs. *Vet Surg* 2004;33:102-106.
22. Gross ME, Dodam JR, Pope ER, et al. A comparison of thiopental, propofol, and diazepam-ketamine anesthesia for evaluation of laryngeal function in dogs premedicated with butorphanol-glycopyrrolate. *JAAHA* 2002;38:503-506.
23. Tobias KM, Jackson AM, Harvey RC. Effects of doxapram HCl on laryngeal function of normal dogs and dogs with naturally occurring laryngeal paralysis. *Vet Anaesth Analg* 2004;31:258-263.
24. Radlinsky MG, Mason DE, Hodgson D. Transnasal laryngoscopy for the diagnosis of laryngeal paralysis in dogs. *JAAHA* 2004;40:211-215.
25. Rudorf H, Barr FJ, Lane JG. The role of ultrasound in the assessment of laryngeal paralysis in the dog. *Vet Radiol Ultrasound* 2001;42:338-343.
26. Proulx J. Respiratory monitoring: arterial blood gas analysis, pulse oximetry, and end-tidal carbon dioxide analysis. *Clin Tech Small Anim Pract* 1999;14:227-230.
27. Smith MM. Diagnosing laryngeal paralysis. *JAAHA* 2000;36:383-384.

28. Bishop MJ, Hibbard AJ, Fink BR, et al. Laryngeal injury in a dog model of prolonged endotracheal intubation. *Anesthesiology* 1985;62:770-773.
29. Barton L. Respiratory muscle fatigue. *Vet Clin North Am Small Anim Pract* 2002;32:1059-1071, vi.
30. Greenfield CL, Walshaw R, Kumar K, et al. Neuromuscular pedicle graft for restoration of arytenoid abductor function in dogs with experimentally induced laryngeal hemiplegia. *Am J Vet Res* 1988;49:1360-1366.
31. Toth A, Szucs A, Harasztosi C, et al. Intrinsic laryngeal muscle reinnervation with nerve-muscle pedicle. *Otolaryngol Head Neck Surg* 2005;132:701-706.
32. Fulton IC, Stick JA, Derksen FJ. Laryngeal reinnervation in the horse. *Vet Clin North Am Equine Pract* 2003;19:189-208, viii.
33. Mathews KG, Roe S, Stebbins M, et al. Biomechanical evaluation of suture pullout from canine arytenoid cartilages: effects of hole diameter, suture configuration, suture size, and distraction rate. *Vet Surg* 2004;33:191-199.
34. Bureau S, Monnet E. Effects of suture tension and surgical approach during unilateral arytenoid lateralization on the rima glottidis in the canine larynx. *Vet Surg* 2002;31:589-595.
35. Greenberg MJ, Bureau S, Monnet E. Effects of suture tension during unilateral cricoarytenoid lateralization on canine laryngeal resistance in vitro. *Vet Surg* 2007;36:526-532.
36. Greenberg MJ, Reems MR, Monnet E. Use of perioperative metoclopramide in dogs undergoing surgical treatment of laryngeal paralysis: 43 cases (1999-2006). *Vet Surg* 2007;36:E11.

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1. The most common cause of acquired laryngeal paralysis is

- a. hypothyroidism.
- b. myasthenia gravis.
- c. trauma.
- d. idiopathic.

2. The muscle responsible for abduction of the arytenoid cartilages during inspiration is the _____ muscle.

- a. cricoarytenoideus dorsalis
- b. cricoarytenoideus lateralis
- c. thyropharyngeus
- d. arytenoideus transversus

3. Laryngeal paralysis has been identified as a congenital condition in

- a. Labrador retrievers.
- b. Great Danes.
- c. Afghan hounds.
- d. Bouvier des Flandres.

4. Which is an early sign of laryngeal paralysis?

- a. syncope
- b. cardiac murmur
- c. voice change
- d. cyanosis

5. Which anesthetic protocol decreases laryngeal function in at least 50% of normal dogs?

- a. acepromazine/thiopental
- b. acepromazine/propofol
- c. ketamine/diazepam
- d. all of the above

6. Regarding partial laryngectomy, which statement is true?

- a. In dogs undergoing bilateral vocal cordectomy, the entire vocal fold should be removed.
- b. Partial arytenoidectomy is performed by removing the corniculate process of the arytenoid cartilage.
- c. Complications are reported in 10% of dogs undergoing unilateral partial laryngectomy for laryngeal paralysis.
- d. Approximately 5% of dogs undergoing unilateral partial laryngectomy die from respiratory-related diseases.

7. The most common complication after unilateral arytenoid lateralization is

- a. respiratory distress.
- b. aspiration pneumonia.
- c. seroma formation.
- d. suture failure.

8. Which factor is associated with a higher rate of complications or death after UAL in dogs with laryngeal paralysis?

- a. young age
- b. obesity
- c. the need to place a temporary tracheostomy tube
- d. perioperative metoclopramide

9. Which statement is true?

- a. Shortening an elongated soft palate increases the risk of postoperative aspiration after arytenoid lateralization.
- b. During UAL, the arytenoid cartilage should be maximally abducted with sutures to enlarge the glottic opening.
- c. Bilateral arytenoid lateralization increases the risk of postoperative complications and respiratory-related death.
- d. The risk of aspiration pneumonia significantly decreases 1 year after UAL.

10. Approximately _____ of dogs experience improvement in upper airway resistance and exercise tolerance following arytenoid lateralization.

- a. 30%
- b. 50%
- c. 75%
- d. 90%