Laryngeal paralysis results when the abductor muscles of the larynx are disrupted. The larynx does not open during inspiration because the arytenoid cartilages fail to retract. The disease may be unilateral but more often occurs bilaterally. Laryngeal paralysis is a common, important cause of upper respiratory obstruction in dogs and is increasingly being recognized in cats. Although laryngeal paralysis was once thought to be an isolated clinical entity, recent reports suggest that the condition is only one manifestation of a generalized neuromuscular disorder. This article provides a metaanalytical overview of the current literature on laryngeal paralysis, describes recent advances in diagnostic techniques, and reviews surgical procedures used for correction. A review of laryngeal paralysis in cats is also provided.

EPIDEMIOLOGY

Laryngeal paralysis is most often diagnosed in geriatric large- and giant-breed dogs but can also occur in a number of small breeds. Most studies report that male dogs are more commonly affected than female dogs. Reported canine male:female ratios range from 3.7:1 to 1:1. The mean age range of dogs treated surgically for laryngeal paralysis is 9.5 to 12.2 years of age. Labrador retrievers are most commonly affected with acquired laryngeal paralysis. Other commonly affected large and giant breeds include the St. Bernard, Irish setter, and Afghan hound.

One report described the prevalence of laryngeal paresis and paralysis in a population of dogs undergoing general anesthesia at a university veterinary teaching hospital. The investigators performed laryngoscopy on 250 dogs, assigning each dog a subjective score of 0 (i.e., normal) to 4 (i.e., completely paralyzed). One-quarter of the dogs examined had some degree of laryngeal paresis. Laryngeal scores were significantly and directly related to age, body weight, and body condition score. Labrador retrievers and rottweilers were at least twice as likely to be affected as other breeds. No effort was made to standardize the anesthetic protocol. This could be important because the most commonly used anesthetic drugs depress laryngeal motion.
LARYNGEAL PARALYSIS IN IMMATURE DOGS

Laryngeal paralysis occurs in immature Bouvier des Flandres, Siberian huskies, dalmatians, rottweilers, Leonbergers, and bullterriers. Laryngeal paralysis has also been reported in a young Afghan hound, a cocker spaniel, a dachshund, and a miniature pinscher. Hereditary laryngeal paralysis was first described in the Bouvier des Flandres and is transmitted as an autosomal dominant trait in this breed. This can be seen as a single clinical entity or as part of a polyneuropathy. Microscopic, degenerative lesions of the nucleus ambiguous of the brain stem have been described. However, these changes alone fail to explain the distal distribution of neurogenic atrophy and common peroneal nerve changes in an 8-month-old male Bouvier des Flandres in another report. 

Less is known about hereditary laryngeal paralysis in other canine breeds. Laryngeal paralysis in young Siberian huskies and husky crossbreeds is thought to occur most often as a single clinical entity in dogs with blue eyes and white faces with “freckles.” Preliminary breeding studies have been unable to describe the mode of heritability.

Five rottweilers (three of which were related) with laryngeal paralysis–polyneuropathy complex had an onset of clinical signs at 9 to 13 weeks of age. Nine dogs had polyneuropathy, manifested as weakness, hypotonia, and hyporeflexia, principally distal to the elbow and stifle. Nine dogs had megaesophagus. The mean observation period between onset of clinical signs and euthanasia or death was 3.7 months. No sex predilection was identified.

Fourteen dalmatians with laryngeal paralysis–polyneuropathy complex had an onset of clinical signs at 2 to 12 months of age. Nine dogs had polyneuropathy, manifested as weakness, hypotonia, and hyporeflexia, principally distal to the elbow and stifle. Nine dogs had megaesophagus. The mean observation period between onset of clinical signs and euthanasia or death was 3.7 months. No sex predilection was identified.

Laryngeal paralysis was seen in Leonbergers 1 year of age and older. Clinical signs included exercise intolerance, weakness, gait abnormalities, change in phonation, and dyspnea. Affected dogs had distal limb muscle atrophy, decreased spinal and cranial nerve reflexes, and decreased to absent movement of the laryngeal and pharyngeal muscles. Electromyogram studies suggested denervation of distal muscles. Nerve conduction velocities were decreased. Peripheral nerve biopsies showed a loss of axons and a shift toward smaller diameter myelinated fibers. Laryngeal paralysis appears to follow an X-linked pattern of inheritance in Leonbergers.

Figure 1. Graph depicting the most common clinical signs of laryngeal paralysis in dogs. These figures represent data from several retrospective studies.

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Percentage of cases</th>
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<tr>
<td>Stridor</td>
<td>90%</td>
</tr>
<tr>
<td>Exercise intolerance</td>
<td>80%</td>
</tr>
<tr>
<td>Respiratory distress</td>
<td>70%</td>
</tr>
<tr>
<td>Change in phonation</td>
<td>30%</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>20%</td>
</tr>
<tr>
<td>Cough or gag</td>
<td>10%</td>
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<tr>
<td>Fever</td>
<td>5%</td>
</tr>
<tr>
<td>Collapse</td>
<td>0%</td>
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</table>

CLINICAL SIGNS

Clinical signs of laryngeal paralysis in dogs include stridor, exercise intolerance, respiratory distress, change in phonation, cyanosis, cough or gag, fever, and collapsed (Figure 1). Hyperthermia and heatstroke may be observed, resulting from inability to adequately ventilate through panting. Clinical signs occur inconsistently until laryngeal paresis develops into paralysis. Some animals also show other signs related to neuromuscular dysfunction, such as limb weakness or dysphagia.

CAUSE AND PATHOPHYSIOLOGY

The cause of laryngeal paralysis can be genetic, as mentioned previously, or acquired. The cause of the acquired form is most commonly described as idiopathic. Other causes of laryngeal paralysis include neoplasia, trauma, infection, or a surgical complication in the cervical or thoracic region. Myasthenia gravis has been implicated as a cause of laryngeal paralysis. The mechanism of idiopathic laryngeal paralysis in dogs is described as a progressive, noninflammatory, degenerative disease of the recurrent laryngeal nerves. Histopathologic characteristics of the recurrent laryngeal nerves include loss of axons, beading of myelin, and perineural fibrosis. Neurogenic atrophy of the cricoarytenoideus dorsalis muscle has been noted.
Bilateral laryngeal paralysis occurs in 81% to 100% of dogs with laryngeal paralysis presenting for surgery.\textsuperscript{7,12} Dogs may have symmetric or asymmetric laryngeal paralysis.\textsuperscript{10} Everted laryngeal saccules, elongated soft palate, laryngeal edema, and moderate to severe laryngeal collapse may play a role in upper airway obstruction secondary to laryngeal paralysis.\textsuperscript{6} Patients with laryngeal paralysis commonly have diffusely inflamed laryngeal mucosa. The reason for this is unknown.

**LARYNGEAL PARALYSIS AS ONE MANIFESTATION OF POLYNEUROPATHY**

Changes in distal tibial and common peroneal nerve biopsy samples in young and old dogs with laryngeal paralysis and polynuropathy have been described.\textsuperscript{3} The predominant changes in teased nerve fiber studies were fiber degeneration or demyelination and remyelination. Electrophysiologic changes indicative of a dying back neuropathy (i.e., axonal degeneration specifically targeting the distal part of long and large diameter nerve fibers) were noted in all dogs. The study authors suggest that laryngeal paralysis is only one clinical sign of an underlying, more generalized polynuropathy with variable clinical expression of neurologic signs.

In one report,\textsuperscript{12} 56% of dogs treated surgically for laryngeal paralysis had posterior weakness before or after surgery. Instances of confirmed neurologic disease range from 2% to 22% of dogs treated surgically for laryngeal paralysis.\textsuperscript{5,10,11} Many reports\textsuperscript{5-7,9,11,12} of surgical treatment of laryngeal paralysis did not include a complete neurologic examination as part of the minimum database.

**CONCURRENT DISEASE**

The relationship between hypothyroidism and laryngeal paralysis is unclear; however, many dogs with laryngeal paralysis are concurrently hypothyroid.\textsuperscript{3,11,26} Hypothyroidism could represent a causative or predisposing factor or could merely be coincidental.\textsuperscript{5-7,9,11,12} Resolution of laryngeal paralysis in supplemented hypothyroid dogs has been poorly described.\textsuperscript{27} In contrast, hypothyroid polyneuropathy (with no concurrent laryngeal paralysis) usually responds to thyroid supplementation. A possible explanation for this disparity could be that laryngeal paralysis is usually diagnosed as an end-stage disease after irreversible atrophy of the cricoarytenoideus dorsalis muscle has already occurred.\textsuperscript{1} Dogs receiving adequate thyroid supplementation have reportedly developed laryngeal paralysis.\textsuperscript{5}

Dogs with laryngeal paralysis are reportedly 21 times more likely to have megaesophagus compared with control groups.\textsuperscript{24} Laryngeal paralysis was diagnosed in 11.8% of dogs with acquired megaesophagus. Concurrent megaesophagus is a negative prognostic indicator.

It has been speculated that laryngeal paralysis associated dysphagia or megaesophagus predisposes patients to aspiration pneumonia.\textsuperscript{28} Reported instances of pneumonia at the time of presurgical evaluation ranged from 7% to 10%.\textsuperscript{5,10} Nearly one-quarter of dogs treated surgically for laryngeal paralysis developed aspiration pneumonia at some point.\textsuperscript{5}

**DIAGNOSIS**

Clinical suspicion is an important tool in diagnosing laryngeal paralysis.\textsuperscript{1} Clinical suspicion was reportedly 91.6% sensitive and 98.5% specific for severe laryngeal paralysis in 250 dogs anesthetized at a veterinary teaching hospital. This comparison used laryngoscopic observation as the definitive diagnostic procedure.

The accepted standard of diagnosis is direct visualization of the arytenoid cartilages by laryngoscopy with the patient under light anesthesia\textsuperscript{6,7,9,11,12} (Figure 2). The presence of abnormal laryngeal function in clinical cases has been based on the subjective opinion of the surgeon. There is a reported 95% agreement between two observers in assigning 17 dogs a laryngeal paralysis score.\textsuperscript{1} A potential problem with diagnosing laryngeal paralysis is that anesthetic agents normally depress laryngeal movement. In normal dogs, anesthetic depths necessary to alleviate jaw tone to safely and easily visualize the larynx may prevent laryngeal motion.\textsuperscript{13,29} The paralysis may be bilateral (Figure 3) or unilateral (Figure 4).

A comparison of various anesthetic protocols for laryngeal function in normal dogs has been reported.\textsuperscript{13}
Two anesthetic protocols, intravenous thiopental alone and intramuscular acepromazine with intramuscular butorphanol plus isoflurane by mask, had the least effect on laryngeal motion. Intravenous propofol, intravenous ketamine plus intravenous diazepam, intramuscular acepromazine plus intravenous thiopental, or intramuscular acepromazine plus intravenous propofol caused more depression of laryngeal motion. This study suggests that use of the latter anesthetic protocols could cause misdiagnosis of laryngeal paralysis. Another report suggests that intravenous thiopental and intravenous propofol are superior to intravenous ketamine plus intravenous diazepam because they more effectively alleviate jaw tone.

Another study evaluated use of doxapram HCl (1.1 mg/kg IV) as a respiratory stimulant in dogs anesthetized with acepromazine, butorphanol, and isoflurane. Depth of respiration increased in normal dogs, but arytenoid motion did not change in response to doxapram HCl injection. Dogs affected by laryngeal paralysis developed paradoxical arytenoid motion and a decrease in glottal area. The authors concluded that doxapram HCl administration may be useful in differentiating between normal and affected dogs. Affected dogs may experience extreme glottic constriction and require intubation during examination.

Transnasal laryngoscopy in dogs has been recently described. This technique is an adaptation of the well-accepted means of diagnosing laryngeal paralysis in horses. The authors were able to successfully assess normal and abnormal laryngeal function in dogs sedated with an opioid and acepromazine. This technique eliminated the need for general anesthesia to diagnose laryngeal paralysis. A 2.5-mm flexible endoscope was passed through the nasal passages of dogs treated with intranasal lidocaine. The technique successfully differentiated between three normal dogs and four dogs affected with laryngeal paralysis. All of the dogs resisted lidocaine application and initial placement of the endoscope. If no laryngeal motion was observed, the authors mechanically stimulated the laryngeal mucosa to differentiate normal from abnormal dogs. Normal dogs began moving the arytenoids, whereas affected dogs did not. Limitations of the technique include the need for a small, flexible endoscope and a large patient.

Use of ultrasonography has been described for diagnosing laryngeal paralysis in dogs. With direct transoral laryngoscopy used as a definitive diagnostic procedure, motion of the cuneiform processes of the arytenoid cartilages was correctly observed in 10 of 10 normal dogs and 29 of 30 dogs with unilateral or bilat-
eral laryngeal paralysis. The one instance of disagree-
ment was when ultrasonography suggested unilateral
involvement but laryngoscopy showed bilateral involve-
ment. Advantages of this technique are that it is rapid
and noninvasive and does not require sedation or anes-
thesia. Disadvantages are that it requires technical
expertise and expensive equipment. Difficulties are
encountered with large, obese dogs and very calm dogs
with shallow breathing.

Computer programs using the normalized glottal gap
area have been used to quantitatively measure the glottal
opening and thereby laryngeal function.\(^{13,33}\) This has
enabled investigators to compare anesthetic agents used
for diagnosis and the efficacy of surgical techniques in
relieving upper airway obstruction.

Historically, tidal breathing flow–volume loops
(TBFVL) and arterial blood gas analysis have been used
to characterize the type and severity of upper airway
obstruction and response to surgical correction.\(^{34,35}\)
TBFVL has not been used clinically in any of the major
surgical technique reviews.\(^{7-12}\) The logistical challenges of
TBFVL likely preclude its clinical use because it requires
a voluntary maximal tidal volume, which is difficult to
achieve in animals. Blood gas analysis is probably not
routinely conducted because it was shown that blood gas
abnormalities are directly related to severity of clinical
signs and are not specific for a particular disease.\(^{35}\)

Electromyography of the cricoarytenoideus dorsalis
muscle has been successfully used to confirm laryngeal
paralysis.\(^5\) However, this technology requires specialized
equipment and a highly trained electrophysiologist.

**EVALUATING FOR CONCURRENT
DISEASE**

The possibility of concurrent disease makes additional
diagnostic testing necessary in patients suspected of
having laryngeal paralysis. Thoracic radiographs should
be taken to rule out aspiration pneumonia, intrathoracic
mass, and megaesophagus.\(^{5,9,10,24}\) Cervical radiographs
have been recommended to rule out neoplasia.\(^{11}\) If dys-
phagia or regurgitation is reported, a barium esoph-
ogram should be considered. Conversely, dogs diagnosed
with megaesophagus and/or aspiration pneumonia
should be screened closely for laryngeal paralysis.\(^{24}\)
Serum acetylcholine receptor antibody tests could be
considered to rule out myasthenia gravis, especially in
dogs with megaesophagus.\(^{24}\) Thyroid function tests
should be conducted to rule out hypothyroidism.\(^{27}\)

**Although laryngeal paralysis most often
affects geriatric large-breed dogs, it can affect
young dogs of a number of breeds.**

A complete neurologic examination should be per-
fomed on all dogs and cats with laryngeal paralysis.
Patients with neurologic abnormalities in addition to
laryngeal paralysis should be considered as candidates
for electrophysiology and muscle and nerve biopsies.
Alternatively, animals presenting with polyneuropathy
should be carefully evaluated for laryngeal paralysis.\(^1\)

**MEDICAL TREATMENT**

Medical treatment of laryngeal paralysis involves
symptomatic treatment of hypoxia, hyperthermia,
excitement, and obesity. Oxygen therapy, cooling, exer-
cise restriction, stress avoidance, and caloric restriction
are advised. Identification and treatment of concurrent disease may be beneficial. Some benefit may be gained by thyroid supplementation in hypothyroid dogs. Corticosteroids have been advocated to decrease laryngeal inflammation. There is no proven efficacious drug therapy for similar polyneuropathies in humans.

**SURGICAL TREATMENT**

A variety of surgical procedures are used to treat laryngeal paralysis. Some are aimed at enlarging the laryngeal opening by removing one or both of the vocal folds (i.e., ventriculocordectomy) and arytenoid cartilages (i.e., partial laryngectomy). Some procedures seek to lateralize one or both arytenoid cartilages (tieback), whereas others seek to widen the larynx by widening the thyroid cartilage (i.e., laryngofissure). A description and brief review of each of these techniques follow.

**Ventriculocordectomy**

Ventriculocordectomy has been described for treating laryngeal paralysis. The dog is anesthetized with an injectable agent and positioned in sternal recumbency with the head elevated. Alternately, a tracheostomy may be performed and the patient maintained under inhalant anesthesia. The mouth is opened with the aid of a dental speculum and the tongue retracted cranially. An assistant elevates the soft palate and depresses the epiglottis with the blade of a laryngoscope, permitting visualization of the larynx. Long forceps and scissors or biopsy forceps are used to reach inside the laryngeal opening and remove the vocal folds from the larynx (Figure 5). To prevent laryngeal “webbing,” the most ventral one-quarter of the vocal fold should not be excised. Simple removal of the vocal folds in some patients provides enough of an airway that the animal does not need additional surgery. This is especially true for inactive animals. The complication of “webbing” may be minimized by using a ventral laryngotomy approach to the vocal folds. The folds are removed under direct visualization and the mucosa reapposed over the excised edges using a 4-0 monofilament absorbable suture.

**Partial Arytenoidectomy**

Partial arytenoidectomy is often performed in combination with unilateral vocal cord excision as described above. One or both of the vocal folds are removed. Biopsy forceps are then used to remove the medial portions of the corniculate processes of the arytenoid cartilages (Figure 6). Surgeons should attempt to remove 2 to 3 mm of the medial border of this car-
It is best to remove only cartilage on one side of the larynx to prevent “webbing” at the dorsal laryngeal opening. It may be necessary to use a rongeur to remove portions of the cartilage. A sufficient amount of laryngeal tissue is removed from one side to provide an adequate laryngeal opening (Figure 7). Hemorrhage is controlled by intermittently packing the larynx with gauze or saline or epinephrine sponges.

Following the partial laryngeal excision, the larynx is packed with gauze for approximately 10 minutes to provide hemostasis. After hemorrhage has been controlled, the upper trachea is aspirated and lavaged, if necessary, to remove residual blood clots. The animal is maintained under anesthesia for another 10 minutes to ensure that additional hemorrhage does not occur. If a tracheostomy is used for these procedures, the tube is left in place for 24 to 48 hours to provide airway management until laryngeal swelling has subsided.

**Laryngeal Tieback**

The objective of laryngeal tieback is to enlarge the laryngeal opening by surgically retracting the arytenoid cartilage on one side of the larynx. This is accomplished by dissecting the arytenoid cartilage from its attachments and retracting it caudolaterally to the dorsocaudal aspect of the cricoid cartilage. The procedure may be done on the left or right side. The animal is anesthetized, and the surgical site over the lateral aspect of the larynx is prepared for aseptic surgery. The neck is extended over a small sandbag to elevate the larynx for increased surgical exposure. A skin incision is made from the level of the ramus of the mandible, ventral to the jugular vein, to a level just caudal to the bifurcation of the jugular vein. The subcutaneous musculature and connective tissues are separated and the jugular vein and its bifurcation retracted dorsally by Gelpi retractors. The thyropharyngeus muscle is incised at its attachment to the rim of the thyroid cartilage. The muscular process of the arytenoid cartilage can usually be palpated as a small protrusion on the lateral surface of the larynx. The cricoarytenoid muscle that

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**Figure 7.** Laryngoscopic view of a cat with bilateral laryngeal paralysis before and after partial arytenoidectomy.
attaches at this point is usually atrophied, making the process particularly prominent.

Dissection begins under the muscular process with small blunt scissors, mosquito forceps, or a periosteal elevator, and the arytenoid cartilage is disarticulated from the cricoid cartilage immediately beneath the muscular process. When dissecting and placing sutures through the muscular process, surgeons should be extremely careful not to break the process, which is fairly easy to do in an elderly animal. This articulation is identified by the presence of articular cartilage. Once totally separated from the cricoid cartilage, the muscular process of the arytenoid cartilage is freely movable. To gain total mobility, it may be necessary to sever the sesamoidian band that connects the left and right arytenoid cartilages across the dorsal aspect of the larynx. This small band of tissue is approximately 1 mm in diameter and, when excised, allows the arytenoid to become totally mobilized.

Two sutures of 0 (for large dogs) or 3-0 (for small dogs and cats) monofilament nonabsorbable suture with a sturdy half-circle taper point needle are placed through the muscular process of the arytenoid and through the dorsocaudal extremity of the wing of the thyroid cartilage\(^8\) (Figure 8). When these sutures are tied tightly, the arytenoid is pulled laterally, opening the larynx (Figure 9).

An alternative and more physiologic suturing technique involves placing the tieback suture from the muscular process of the arytenoid to the dorsocaudal border of the cricoid cartilage.\(^{13}\) Sutures from the arytenoid to the cricoid in this procedure approximate the same
function and location as the cricoarytenoideus dorsalis muscle. This suture is more demanding to place because exposure of the dorsocaudal cricoid can be difficult. In placing these sutures, surgeons must be very careful to retract the esophagus dorsally to avoid injury when operating on the left side. Preferably, two simple sutures should be placed from the muscular process of the arytenoid to the caudal rim of the cricoid immediately lateral to the dorsal midline (Figure 10). For right-handed surgeons, the suture is passed from the cricoid cartilage cranial to the muscular process. For left-handed surgeons, the suture is passed from the muscular process caudally to the cricoid cartilage. After these sutures have been placed and one has been tied, the anesthetist should examine the larynx with a laryngoscope to ensure that the tieback procedure has resulted in proper lateralization of the arytenoid cartilage (Figure 11). It is very easy to overabduct the arytenoid cartilage with this procedure, resulting in dysfunction of the larynx after surgery due to inability of the epiglottis to totally close the abducted larynx. It is necessary to move the muscular process only a few millimeters caudally (Figure 11). The second suture is tied after the first has been confirmed to be properly placed. Absorbable sutures are used to reapproximate the severed thyropharyngeus muscle and the subcutaneous tissue. The skin is closed with monofilament nonabsorbable sutures.

**Figure 10.** Drawing illustrating a cricoarytenoid tieback with placement of two sutures from the muscular process of the arytenoid cartilage to the dorsocaudal aspect of the cricoid cartilage. (Haines DK © 2005 The University of Tennessee College of Veterinary Medicine)

**Figure 11.** Laryngoscopic view of a patient with bilateral laryngeal paralysis before and after cricoarytenoid lateralization. Note that the arytenoid is more lateralized than with the thyroarytenoid tieback in Figure 9.

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**Modified Castellated Laryngofissure**

A seldom-used technique is the modified castellated laryngofissure. The patient is anesthetized and placed in dorsal recumbency, with the neck arched ventrally, and the skin over the larynx is prepared for surgery.\(^{39,40}\)
A skin incision is made over the larynx to the fourth tracheal ring. The sternohyoides muscle is divided to expose the thyroid and cricoid cartilages. A tracheotomy tube is inserted into a vertical incision between the second and third tracheal rings. A “step” incision is made in the thyroid cartilage, and the larynx is opened (Figure 12). The vocal folds are removed under direct visualization. The arytenoids are bilaterally lateralized by monofilament mattress sutures placed through the thyroid cartilage and the arytenoid cartilage dorsal to the vocal process, with the knot outside the laryngeal lumen. Two or three 3-0 monofilament nonabsorbable sutures are preplaced between the step and the cranial segment of the opposite cartilage incision. The castellated cartilage incision is closed by aligning the step against the cranial segment of the opposite cartilage incision, thereby spreading the larynx by the height of the step. The step is fixed in position by tightening the preplaced sutures. Sutures from the thyroid around the basihyoid bone help secure the closure. Loose tissue and corner edges of cartilage are trimmed away to prevent them from entering the lumen of the larynx. The sternohyoideus and sternothyroideus muscles are tightly approximated to close the laryngeal defect. The subcutaneous tissue and skin are closed routinely. The tracheostomy tube is removed in 3 to 4 days.

Other Surgical Options
Neuromuscular pedicle grafting has been investigated in dogs. It is not commonly performed because it does not provide immediate relief of upper airway obstruction. Neuromuscular pedicle grafts require 36 to 44 weeks to return laryngeal movement to normal. Permanent tracheostomy has been recommended as a final alternative in treating laryngeal paralysis. Permanent tracheostomy can be problematic in dogs that like to swim.

COMPARISON OF DIFFERENT SURGICAL TECHNIQUES
A 2001 report provides the most comprehensive comparison of different surgical techniques. The findings show that both unilateral arytenoid lateralization and partial laryngectomy offer superior clinical outcome over bilateral arytenoid lateralization. Complication rates between unilateral arytenoid lateralization (30%) and partial laryngectomy (40%) were not significantly different. However, dogs treated with partial laryngectomy were significantly more likely to die of complications than were dogs treated with unilateral arytenoid lateralization. These complications included aspiration pneumonia, respiratory distress, failure of surgical repair, and death. The complication rate may be higher than in some other studies because of a longer duration of follow-up. There was not a significant difference in implant...
failure between dogs treated with thyroarytenoid lateralization and those treated with cricoarytenoid lateralization. Postoperative death rates were highest in dogs treated with bilateral arytenoid lateralization (67%) compared with unilateral arytenoid lateralization (14%) and partial laryngectomy (30%). Factors predisposing patients to death or complications were age, temporary tracheostomy placement, concurrent respiratory tract abnormalities, concurrent esophageal disease, postoperative megaesophagus, concurrent neoplastic disease, and concurrent neurologic disease. A common complication of the tieback procedure is persistent postoperative cough, especially after eating or drinking. This is because the arytenoid cannot adduct and the epiglottis does not completely close with swallowing.

The most commonly recommended treatment of laryngeal paralysis in dogs is unilateral arytenoid lateralization. This procedure appears to offer good resolution of clinical signs with fewer complications than partial laryngectomy. Unilateral arytenoid lateralization is technically easier and quicker than castellated laryngofissure.

One report compared the two methods of unilateral arytenoid lateralization (i.e., thyroarytenoid versus cricoarytenoid lateralization). It was shown that cricoarytenoid lateralization increased the size of the glottic opening compared with thyroarytenoid lateralization. Cricoarytenoid lateralization did not, however, result in improved clinical outcome but did require significantly less operative time (i.e., 25 minutes compared with 43 minutes).

**LARYNGEAL PARALYSIS IN CATS**

Laryngeal paralysis is becoming more commonly recognized in cats. No sex or breed predilection has been identified. The median age of cats diagnosed with laryngeal paralysis is 11 years (range: 4 months to 17 years of age).

Half of the affected cats in one report were domestic shorthair. Other breeds represented included domestic longhair, Siamese, Abyssinian, and Balinese. The same study reported that clinical signs began at younger than 3 years of age in 31% of affected cats. Another study reported that clinical signs began in two of four cats younger than 1 year of age.

Clinical signs of laryngeal paralysis in cats include tachypnea or dyspnea, stridor, exercise intolerance, change in phonation, dysphagia, weight loss, cough, anorexia, lethargy, cyanosis, and fever. As in dogs, laryngeal paralysis in cats may occur in conjunction with polyneuropathy. In one report, two of four affected cats had generalized neuromuscular disease.

Little is known about the pathophysiology of laryngeal paralysis in cats. Based on the age of cats treated surgically for laryngeal paralysis, apparently, there are congenital and acquired causes. How often cats are affected with idiopathic laryngeal paralysis is unknown. In one report, 75% of affected cats had bilateral laryngeal paralysis. Other causes of laryngeal paralysis include neoplasia, trauma, or a surgical complication in the cervical or thoracic region.

**CONCLUSION**

Laryngeal paralysis is a common, important cause of upper respiratory obstruction in dogs and is increasingly being recognized in cats. Recent advances in diagnostic techniques should help veterinarians correctly identify...
the problem. Thorough evaluation for concurrent disease is crucial in providing excellent patient care. Several surgical alternatives seem to be acceptable, if not ideal, in treating the problem. Arytenoid tieback surgery appears to give the best overall results.

REFERENCES


### ARTICLE #4 CE TEST

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1. Which has reportedly caused laryngeal paralysis in dogs?
   a. idiopathic nerve degeneration
   b. postsurgical complication
   c. neoplasia
   d. all of the above

2. Laryngeal paralysis occurs most often in dogs.
   a. old, large-breed
   b. old, small-breed
   c. young, large-breed
   d. young, small-breed

3. Laryngeal paralysis in the Bouvier des Flandres is inherited as an ____________ trait.
   a. X-linked
   b. autosomal dominant
   c. autosomal recessive
   d. none of the above

4. A possible association exists between laryngeal paralysis and
   a. hypoadrenocorticism
   b. hyperadrenocorticism
   c. hypothyroidism
   d. insulinoma

5. Which anesthetic(s) is expected to have the least impact on laryngeal function?
   a. thiopental
   b. propofol
   c. diazepam plus ketamine
   d. All of the above have a similar effect on laryngeal function.

6. Administering doxapram to a dog with laryngeal paralysis would not be expected to cause ____________ during laryngeal examination.
   a. increased respiratory effort
   b. increased arytenoid abduction
   c. paradoxical arytenoid motion
   d. glottic constriction

7. Which statement regarding laryngeal paralysis is true?
   a. It is advisable to have an endotracheal tube ready when performing transnasal laryngoscopy because dogs may experience severe glottic constriction and require intubation during examination.
   b. Ultrasonography of the larynx correlates poorly with laryngeal function.
   c. Inappropriate choice of anesthetic protocols could lead to incorrect diagnosis of laryngeal paralysis.
   d. Hypothyroidism has been shown to cause laryngeal paralysis.

8. A common complication of partial arytenoidecrctomy is
   a. surgical repair failure
   b. uncontrollable hemorrhage
   c. epiglottic paralysis
   d. laryngeal “webbing”

9. Sutures can be passed between the ____________ cartilages to perform arytenoid lateralization.
   a. arytenoid and thyroid
   b. thyroid and cricoid
   c. arytenoid and cricoid
   d. a and c

10. The ____________ cartilage is cut in a “stepwise” fashion during castellated laryngofissure.
    a. cricoid
    b. thyroid
    c. arytenoid
    d. epiglottis