Laryngeal paralysis is defined as failure of abduction of one or both arytenoid cartilages during inspiration. Congenital and acquired forms of laryngeal paralysis have been reported, with the acquired form being more common and predominantly diagnosed in older large-breed dogs. Causes of the acquired form of laryngeal paralysis include trauma, infection, neoplasia, iatrogenic, and idiopathic polyneuropathy. Idiopathic polyneuropathy is the most commonly reported cause.

Surgical treatment of laryngeal paralysis is required to treat clinical signs, improve exercise tolerance, and improve quality of life. The goal of surgical treatment is to decrease LAR without increasing the risk of aspiration pneumonia. Unilateral arytenoid lateralization is the most commonly performed surgical treatment for laryngeal paralysis. Bilateral ventriculocordectomy via a transoral or ventral laryngotomy approach has been used to treat dogs with laryngeal paralysis.

Aspiration pneumonia is a complication of laryngeal paralysis that can develop before or after surgery. Potential mechanisms for the development of aspiration pneumonia are laryngeal and esophageal dysfunction as well as gastroesophageal reflux. Furthermore, an increased diameter of the rima glottidis after unilateral lateralization may prevent the epiglottis from closing the rima glottidis during swallowing, which increases the risk of developing aspiration pneumonia.

One of the potential advantages of ventriculocordectomy is that it does not affect the diameter of the rima glottides dorsally. Therefore, it should minimize the risk of aspiration pneumonia by reducing LAR. In one study, resistance of the larynx (ie, upper airways) was not affected after ventriculocordectomy in dogs with experimentally induced chronic laryngeal paralysis. However, the protective effect of the epiglottis against aspiration pneumonia was not evaluated in that study because the investigators could not differentiate when the epiglottis was in an open or closed position.

The purpose of the study reported here was to evaluate the effect of bilateral ventriculocordectomy via ventral laryngotomy on laryngeal airway resistance in canine cadaver larynges.

**OBJECTIVE**
To evaluate the effect of bilateral ventriculocordectomy via ventral laryngotomy on laryngeal airway resistance (LAR) in canine cadaver larynges.

**SAMPLE**
6 clinically normal canine cadaver larynges.

**PROCEDURES**
LAR was determined for each specimen before (baseline) and after bilateral ventriculocordectomy with the epiglottis open and closed. After ventral laryngotomy was performed, the vocal cords were sharply excised, and the incised mucosal edges were apposed with 4-0 glycomer 631 suture in a simple continuous pattern. The thyroid cartilage was apposed with 3-0 polypropylene suture in a simple continuous pattern.

**RESULTS**
With the epiglottis closed, baseline median LAR was 27.6 cm H₂O/L/s (range, 21.2 to 30.6 cm H₂O/L/s), which did not differ significantly from the median LAR after bilateral ventriculocordectomy (24.7 cm H₂O/L/s [range, 20.6 to 27.7 cm H₂O/L/s]). With the epiglottis open, baseline median LAR was 7.3 cm H₂O/L/s (range, 5.4 to 7.8 cm H₂O/L/s), which did not differ significantly from the median LAR after bilateral ventriculocordectomy (7.2 cm H₂O/L/s [range, 6.6 to 7.6 cm H₂O/L/s]).

**CONCLUSIONS AND CLINICAL RELEVANCE**
Bilateral ventriculocordectomy did not affect LAR with an open epiglottis in canine cadaver larynges. Therefore, it may not be an effective treatment for laryngeal paralysis. It also did not affect LAR with a closed epiglottis, which may indicate protection against aspiration pneumonia.
via ventral laryngotomy in larynges of canine cadavers on LAR with the epiglottis open and closed. We hypothesized that bilateral ventriculocordectomy via ventral laryngotomy would not result in a significant change of LAR in the larynx of a canine cadaver with the epiglottis open or closed.

Materials and Methods

Sample
The larynx was harvested from each of 6 cadavers of purpose-bred dogs immediately after the dogs had been euthanized; dogs were euthanized for reasons unrelated to the present study. All larynges were visually inspected to determine that they were anatomically normal. None of the dogs had clinical signs of upper airway obstruction at the time of euthanasia. All extrinsic soft tissues were removed from each larynx: only the laryngeal cartilages, intrinsic laryngeal musculature, and first 4 tracheal rings were obtained. Specimens were wrapped in sponges moistened with saline (0.9% NaCl) solution and stored at 2°C until testing, which was performed approximately 48 hours after specimens were harvested.

Experimental procedures
The LAR was calculated for each specimen before (baseline) and after bilateral ventriculocordectomy via ventral midline laryngotomy with the epiglottis open and closed. The order in which the 6 specimens were tested was determined by means of a random-number generator. Larynges were periodically moistened with saline solution throughout testing.

Ventriculocordectomy
A stay suture of 3-0 polypropylene suture was placed through the tip of the epiglottis. The ends of the stay suture were passed down the trachea and used to close the epiglottis.

Bilateral ventriculocordectomy was performed via ventral laryngotomy as described elsewhere. First, the right vocal fold and vocal muscle were identified, sharply dissected with curved Metzenbaum scissors, and completely excised. The mucosa was sutured over the ventriculocordectomy site with 4-0 glycomer 631 suture in a ventrodorsal-dorsoventral direction with a simple continuous suture pattern. The mucosa of the laryngeal saccule was used to achieve a tension-free closure (Figure 1). The same procedure then was performed on the left side. The thyroid cartilage was closed with 3-0 polypropylene suture in a simple continuous pattern, which provided alignment and apposition of the thyroid cartilages without overlap.

LAR measurement
The LAR was determined in accordance with a previously described technique. Each larynx was mounted in a chamber, and the pressure in the chamber was measured by use of a water manometer, extension tubing, and 3-way stopcock, which was the same system that had been used in another study. The extension tubing was attached to the chamber at a point 2 cm from the air inflow stream. The water manometer was calibrated to zero at the level of the center of the laryngeal lumen for each specimen before each test. Outflow pressure was atmospheric pressure. Airlow was controlled with a precalibrated flowmeter.

For each test, a larynx was secured into the testing chamber, and the epiglottis was open or closed. The manometer was filled with saline solution, and the 3-way stopcock was turned to prevent additional access to the testing chamber. The flowmeter was set to the desired airflow. The 3-way stopcock was then opened to provide access to the testing chamber, and the manometer was allowed to equilibrate.
to the pressure within the chamber. Pressure of the testing chamber was recorded when the manometer pressure reading remained at a constant value for a period of 10 seconds. Each test was repeated 3 times to ensure repeatability. The median of the 3 values was then recorded.

The LAR was calculated by use of the following equation: \( \text{LAR} = \frac{\Delta P}{AF} \), where \( \Delta P \) is the pressure gradient, and \( AF \) is the airflow. The \( \Delta P \) was the difference between the pressure on one side of the larynx (the pressure measured in the testing chamber) and the pressure on the other side of the larynx (atmospheric pressure). Because the transducer was calibrated to zero at the level of the larynx, the pressure recorded during the experiment was \( \Delta P \).

The LAR at baseline was first measured for each larynx with the epiglottis open and again with the epiglottis closed. Each larynx was secured into the testing chamber with the epiglottis open. Airflow was set at 40 L/min, and the pressure in the chamber was measured. The epiglottis was closed (by pulling the epiglottic stay suture); airflow was reduced to 10 L/min, and the pressure in the chamber was again measured.

Bilateral ventriculocordectomy was then performed. Each larynx was again mounted in the chamber, and the LAR was measured with the epiglottis open and closed.

**Statistical analysis**

Data were reported as median and range. The effect of bilateral ventriculocordectomy on LAR was analyzed by use of a Wilcoxon signed rank test with the epiglottis open and closed. Values of \( P < 0.05 \) were considered significant.

**Results**

With the epiglottis open, median LAR at baseline was 7.3 cm H\(_2\)O/L/s (range, 5.4 to 7.8 cm H\(_2\)O/L/s), which did not differ significantly (\( P = 0.90 \)) from the median LAR after ventriculocordectomy (7.2 cm H\(_2\)O/L/s [range, 6.6 to 7.6 cm H\(_2\)O/L/s]; Figure 2). With the epiglottis closed, the median LAR at baseline was 27.6 cm H\(_2\)O/L/s (range, 21.2 to 30.6 cm H\(_2\)O/L/s), which did not differ significantly (\( P = 0.13 \)) from the median LAR after ventriculocordectomy (24.7 cm H\(_2\)O/L/s [range, 20.6 to 27.7 cm H\(_2\)O/L/s]).

**Discussion**

In the present study conducted with anatomically normal larynges of canine cadavers, LAR with the epiglottis open or closed was not affected by bilateral ventriculocordectomy. Previous studies\(^{14-16,20,21}\) of laryngeal paralysis have involved the use of larynges from canine cadavers. Several procedures have been found to significantly decrease LAR in canine cadavers, validating the use of this method.\(^{14-16,20,21}\)

In the present study, baseline LAR measurements obtained before bilateral ventriculocordectomy were similar to those reported in other studies.\(^{14-16,21}\) The LAR in isolated cadaveric specimens in the present study is lower than the value reported for dogs with induced laryngeal paralysis\(^{19}\); however, authors of that study\(^{19}\) did not completely isolate the larynx. Those authors reported resistance for the upper airways (including the nares, nasal cavity, and oral pharynx), instead of the LAR that was reported in the present study.

Cadaveric specimens do not have tone in the cricoarytenoideus dorsalis muscle, thus causing passive adduction of both arytenoid cartilages, which is similar to the situation for dogs with bilateral laryngeal paralysis. Airflows in the present study (10 to 40 L/min) were similar to those in spontaneously breathing dogs.\(^{19,22,23}\) Assuming that this passive adduction is similar to that in a dog with laryngeal paralysis in vivo, a similar LAR should be created at airflows similar to those generated by a live animal.

Ventriculocordectomy has been performed via a transoral approach or ventral laryngotomy, primarily to devocalize dogs.\(^{17,24}\) The transoral approach has been associated with severe scar tissue formation that compromises the diameter of the larynx.\(^{17}\) Ventral laryngotomy with suturing of the mucosa over the ven-
Ventriculocordectomy to prevent cicatrix formation has been recommended.\textsuperscript{23} Ventriculocordectomy with suturing of the mucosa has been used with or without partial arytenoidecitiay or arytenoid lateralization to treat dogs with laryngeal paralysis.\textsuperscript{3,12,17,18,24–27} For the present study, we used the same ventral laryngotomy approach with mucosal suturing as has been reported for other studies\textsuperscript{9,18} related to laryngeal paralysis.

In the study reported here, ventriculocordectomy did not affect LAR with the epiglottis open. Similar results were reported for a study\textsuperscript{19} that involved the use of awake dogs with induced laryngeal paralysis. In that study,\textsuperscript{19} upper airway resistance was above reference limits and did not decrease after ventriculocordectomy; upper airway resistance did not return to within reference limits after surgery and was high from 3 to 42 days after surgery. There were several possible reasons that ventriculocordectomy failed to reduce LAR in the present study. First, the suture lines were bulging into the lumen of the larynx after ventriculocordectomy, which could have interfered with airflow and increased LAR. This bulging could worsen during the postoperative period because of postoperative edema and scar tissue formation.\textsuperscript{9,19,24} Second, it was also possible that the irregular surface of the sutured mucosa created enough turbulence in the larynx that it increased the LAR. Finally, it is possible that anatomically normal vocal cords like those used in the present study do not contribute substantially to LAR.

Investigators of a study\textsuperscript{19} that involved dogs with experimentally induced laryngeal paralysis found that upper airway resistance was increased after administration of respiratory stimulants. However, the effect of the respiratory stimulant on upper airway resistance was blunted after ventriculocordectomy, likely because the flaccid vocal cords were no longer interfering with the flow of air. Therefore, those distended vocal cords may have contributed to upper airway obstruction during laryngeal paralysis, which would explain the reason that 63% to 90% of owners have reported excellent satisfaction for palliation of clinical signs in their dogs after ventriculocordectomy for laryngeal paralysis.\textsuperscript{12,17,18}

A lack of reduction of airway resistance after ventriculocordectomy is compatible with the clinical observation of respiratory distress, coughing, and noisy breathing in the short term.\textsuperscript{9,18} Noisy breathing and coughing after surgery were reported for 29% of dogs after ventral laryngotomy and ventriculocordectomy.\textsuperscript{18} In another study,\textsuperscript{9} investigators also performed bilateral arytenoid lateralization in addition to ventriculocordectomy performed with a ventral laryngotomy. In that study,\textsuperscript{9} all dogs were discharged from the hospital within 24 hours after surgery, but only 55% of owners were satisfied with the outcome in the short term. Stridor and labored breathing were among the most common problems reported by the owners.\textsuperscript{9}

In 3 studies,\textsuperscript{12,17,18} the majority (63% to 90%) of owners reported excellent satisfaction for palliation of clinical signs after ventriculocordectomy alone as a treatment for laryngeal paralysis. As indicated by results of a survey conducted for 1 of those studies,\textsuperscript{18} owner satisfaction was based only on resolution of clinical signs and not on improvement in the level of exercise. Investigators of another study\textsuperscript{25} reported that dogs with induced laryngeal paralysis were exercise intolerant and unable to trot for 30 m at 42 days after ventriculocordectomy. Therefore, ventriculocordectomy might not be sufficient to palliate clinical signs during exercise because it may not reduce laryngeal or upper airway resistance.

In the present study, ventriculocordectomy did not affect the LAR with the epiglottis closed because the rima glottidis was not affected by the procedure. Therefore, the epiglottis can still protect the airway, which is in contrast to unilateral lateralization if excessive abduction is created.\textsuperscript{14–16} Aspiration pneumonia has been detected in 7.9% of dogs with laryngeal paralysis prior to surgical treatment.\textsuperscript{2} In a study\textsuperscript{18} of 88 dogs with laryngeal paralysis treated with ventriculocordectomy, only 3 (7.1%) dogs had aspiration pneumonia, which was significantly less than the percentage of dogs with aspiration pneumonia after treatment of laryngeal paralysis with unilateral lateralization (23.6% to 31.8%).\textsuperscript{2,15}

The study reported here had several limitations, including the small sample size and use of anatomically normal cadaveric larynges. Acute postoperative swelling may be encountered with bilateral ventriculocordectomy, but this was not evaluated in this cadaveric study. We could not evaluate the effect of scar tissue formation, which has been reported in patients after ventriculocordectomy. Ventricleulocordectomy did not change LAR in the anatomically normal canine cadaver larynx (open or closed glottis), which indicated that the LAR may not change in the immediate postoperative period; therefore, an anatomically normal larynx may not be the best structure to test, but we believe it provided good baseline information. However, cadaveric larynges have been used previously to evaluate the reduction of LAR for various techniques.\textsuperscript{15,16,20} It would be interesting to repeat the study with larynges of dogs with laryngeal paralysis to evaluate the effect on LAR. In dogs with laryngeal paralysis, the vocal cords usually are extremely flaccid and distended and vibrate in the ventral part of the rima glottides, which creates the stridorous sounds heard in dogs with laryngeal paralysis. Because of changes (eg, edematous vocal cords) in larynges of dogs with laryngeal paralysis, it would be beneficial to perform future studies to evaluate LAR by use of larynges of dogs with laryngeal paralysis. More in vitro and in vivo studies are needed to determine the factors that account for the positive results seen in clinical studies.

For the present study, we concluded that bilateral ventriculocordectomy did not affect LAR with the epiglottis open or closed in isolated anatomically normal larynges from canine cadavers. On the basis of the results of this cadaveric study, the authors
would not recommend ventriculocordectomy as the preferred technique for treating laryngeal paralysis. Further studies are needed to determine the ideal surgical treatment for laryngeal paralysis that would improve airflow while maintaining functional airway protection.

Footnotes
a. Surgipro, Medtronic Inc, Minneapolis, Minn.

b. Biosyn, Medtronic Inc, Minneapolis, Minn.

References