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VOCAL FOLD MEDIALIZATION BY SURGICAL AUGMENTATION VERSUS ARYTENOID ADDUCTION IN THE IN VIVO CANINE MODEL

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There are a variety of methods for treating unilateral vocal cord paralysis, but to date there have been few studies that compare these phonosurgical techniques by using objective measures of voice improvement. Vocal efficiency is an objective voice measure that is defined as the ratio of the acoustic power produced by the larynx to the subglottic air power. Vocal efficiency has been found to decrease with glottic disorders such as vocal cord paralysis and carcinoma. This study compared the effects of vocal fold medialization by surgical augmentation to those of arytenoid adduction on the vocal efficiency, videostroboscopy, and acoustics (jitter, shimmer, and signal-to-noise ratio) of a simulated unilateral vocal cord paralysis in an in vivo canine model. Arytenoid adduction was superior to surgical augmentation in vocal efficiency, traveling wave motion, and acoustics.

KEY WORDS — flaccid laryngeal paralysis, laryngoplasty, phonosurgery, recurrent laryngeal nerve, stroboscopy, vocal efficiency.

INTRODUCTION

There are a variety of methods for treating unilateral vocal cord paralysis. These include Teflon injection,[1] thyroplasty,[2] arytenoid adduction,[3] and nerve[4] and nerve-muscle pedicle transfer.[5] Most of these methods have been reported to improve the voice. However, recently, a theoretical paper comparing effects of the above treatment modalities on laryngeal vibration found significant differences among them.[6] Unfortunately, to date there have been few studies that compare these phonosurgical techniques by using objective measures of voice improvement. Most authors simply describe the voice as improved over the paralyzed state.

Vocal efficiency is an objective measure of the voice that was first studied by van den Berg[7] in 1956. He defined the efficiency of the voice as the ratio of the acoustic power of the voice to the subglottic power. The subglottic air power can be estimated as the product of the mean glottic airflow rate and the mean subglottic pressure. The physiologic control of vocal efficiency has been studied by several investigators. Koyama et al,[8] using an in vivo canine model, found higher levels of vocal efficiency when cricothyroid contraction was added to recurrent laryngeal nerve stimulation. Tanaka and Tanabe,[9] also using an in vivo canine model, found that increased contraction of the thyroarytenoid or cricothyroid muscle increased glottic resistance (the ratio of glottic airflow to subglottic pressure), while vocal efficiency remained constant. They further observed that contraction of the lateral cricoarytenoid muscle increased both glottic resistance and vocal efficiency. They postulated that thyroarytenoid muscle contraction plays a greater role in intensity control during normal phonation than lateral cricoarytenoid contraction by changing cord stiffness and shape, while lateral cricoarytenoid contraction plays a greater role in pathologic cases with incomplete glottic closure by enhancing cordal adduction. Clinically, vocal efficiency has been shown to decrease with some forms of laryngeal disease, such as invasive carcinoma and vocal cord paralysis.[10,11]

Although vocal efficiency is a useful objective measure of voice, it does have its shortcomings. This measure may not correspond with vocal quality. The voice may be quite harsh with a normal vocal efficiency. Also, vocal efficiency does not indicate the degree of control the patient has over the glottis. Typically, vocal efficiency increases with intensity, and an early vocal abuser may have a higher vocal efficiency despite actively abusing his or her voice.[9]

A number of acoustic measures of voice quality have been used clinically. These include jitter, shimmer, and signal-to-noise ratio. Jitter is defined as the fluctuation in the time interval between successive peaks of the fundamental frequency. Shimmer is the cycle-to-cycle variation in the amplitudes of the peaks. Signal-to-noise ratio is the ratio of the sound energy in the acoustic signal to the noise in the voice signal.[12,13] Lieberman[14] was the first to report an increased jitter in pathologic phonation. Lieberman[14] and Koike et al[15] found, using high-speed cinematography, that pitch perturbations reflected variations in the glottic area and periodicity. Koike et al[15] and Zyski et al[16] found increased jitter

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and shimmer in patients with laryngeal tumors and unilateral vocal cord paralysis. Efforts have been made to use these measures as screening devices for laryngeal disease,[17] but few studies have used these measures to document the results of laryngeal surgery.[18]

An examination of current literature demonstrates that the optimal procedure for voice improvement in a patient with unilateral vocal cord paralysis is in a state of flux.[19] Teflon injection has been very successful in improving the voice and reducing aspiration since its introduction in 1962 by Arnold.[1] Rubin[20] studied the histology of Teflon-injected vocal cords and showed, using high-speed cinematography, improved vibration of the paralyzed cord, which was placed in a more medial position after injection. Von Leden et al[21] found voice improvement, postinjection, using both acoustic analysis and laryngeal function studies. Rontal et al[22] also demonstrated improvement in voice spectrograms postinjection. However, a number of concerns regarding Teflon injection are emerging.

The degree of improvement with Teflon injection is sensitive to the amount and position of injection.[23] Trapp et al[24] found, in the dog, that overinjection or underinjection of Teflon into the paralyzed cord would result in a lack of two-mass (upper and lower margin) vibration. Crumley et al[4] found, in humans, that Teflon-injected vocal cords lacked a mucosal wave. They postulated that the early voice fatigue often seen after Teflon injection may be due to sound's being generated by only the noninjected cord. Failure to achieve an improved voice has been reported to occur in about 10% of cases.[25] Acoustic studies by Trapp et al[24] using the in vivo canine model have shown that phonation after Teflon injection requires a high flow rate and has a high jitter. Cormier et al[26] measured forced inspiratory and expiratory airflow before and after injections and found a transient subclinical inspiratory airway obstruction at 24 hours after injection in all seven of the patients studied. This airway obstruction was thought to be due to postinjection inflammation of the paralyzed cord, which resolved in approximately 10 days. Rubin[20] demonstrated this inflammatory reaction histologically and warned that the voice may be worse up to 5 days after injection until the reaction subsides. He also showed that injection close to the free margin of the cord could increase its stiffness and interfere with its vibratory characteristics. Other complications of this procedure include granuloma formation, migration of Teflon, imprecise placement of Teflon, and overinjection with possible airway obstruction. The long-term effects of Teflon injection on the cord and the voice have not been reported. As an alternative to Teflon injection, a number of surgeons have begun to treat paralysis by laryngeal framework surgery.

Surgery on the laryngeal framework as a treatment for unilateral vocal cord paralysis began with Payer[27] and was later modified by Meurman[28] and others. The first systematic study and classification of these procedures was by Isshiki et al,[2] who coined the term thyroplasty. There are four types of thyroplastic operations. Type I provides lateral compression to the paralyzed cord, narrowing the glottic chink. Type II creates lateral expansion of the glottis. Type III shortens and relaxes the cord bilaterally. Type IV lengthens and stretches the cords. Isshiki et al[29] studied the results of these four types of thyroplasties in treating unilateral vocal cord paralysis in the canine larynx. In their study, the degree of voice improvement was evaluated subjectively as "improved" or "rough," and the mechanical effect on the larynx was studied only with laryngoscopy. They recommended using a type I thyroplasty for a unilateral recurrent laryngeal nerve paralysis and types I and IV together for a unilateral superior and recurrent laryngeal nerve paralysis. Clinically, there are four types of manual compression tests that can be performed on the larynx to help decide if thyroplasty would be helpful. These are the lateral compression test, the dorsal compression test, the cricothyroid approximation test, and combinations of the above.[30] Improvement in the voice during the lateral compression test helps indicate the degree of improvement after type I thyroplasty.

Koufman[31] reported a series of 11 patients who underwent a modification of the type I thyroplasty (medialization laryngoplasty) for treatment of unilateral vocal cord paralysis (both recurrent and vagal). On a five-point scale, improvement was seen in 10 of 11 patients. Escajadillo[32] reported a modification of the type I thyroplasty performed in dogs and humans. This modification resulted in a "near normal" or "normal" voice in 4 of 5 patients. Koufman found several advantages of medialization laryngoplasty over Teflon injection. The medialization is theoretically easily reversible, whereas Teflon injection is more difficult to reverse. The patient's discomfort is less with this procedure under local anesthesia than with a direct laryngoscopy under topical and local anesthesia. The surgeon can add to and subtract from the degree of medialization to fine-tune the voice, whereas the Teflon injection can only add to the medialization.

After their initial experience with the type I thyroplasty, Isshiki et al[3] noticed that unilateral laryngeal paralysis patients with a large posterior glottic chink or a difference in the level of the cords were not achieving optimal results. If the posterior glottic chink was large, it continued to leak air after type I thyroplasty. In five patients (two of whom had failed a type I thyroplasty), Isshiki performed an arytenoid adduction in which a suture was placed around the muscular process of the arytenoid and through the anterior thyroid cartilage, and tied it with

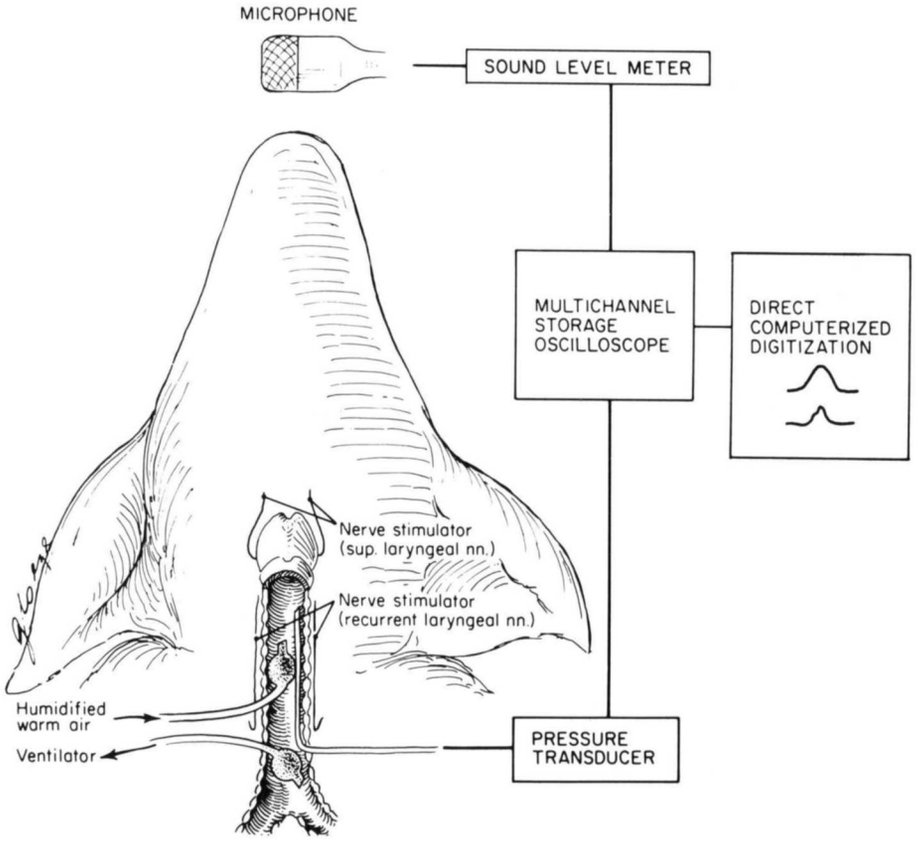

Fig 1. Schematic representation of experimental setup for in vivo canine model of phonation.

enough tension to adduct the arytenoid to the midline. This was performed as an open neck dissection under local anesthesia. Postoperatively, all five patients had improved spectrograms.

The first reported arytenoid procedure for vocal cord adduction was by Morrison,[33] who used an open procedure to release the posterior cricoarytenoid muscle from the muscular process, and sutured the arytenoid in the midline after removing a piece of cricoid to adjust the level of the cord. Montgomery[34] used a laryngofissure to gain access to the glottis and then pinned the arytenoid to the cricoid in the midline position.

This study used an in vivo canine model to compare vocal fold medialization by surgical augmentation to arytenoid adduction in improving the vocal efficiency, videostroboscopy, and acoustic properties (jitter, shimmer, and signal-to-noise ratio) of a unilateral vocal cord paralysis.

MATERIALS AND METHOD

In Vivo Canine Model. Mongrel dogs (25 kg) were premedicated with Innovar intramuscularly. Intravenous Pentothal was administered to a level of corneal anesthesia and additional Pentothal was used to maintain this level of anesthesia throughout the experiment.

The animal was placed supine on the operating table, and a midline incision was made to expose the trachea from the hyoid to the sternal notch. Both recurrent laryngeal nerves were identified and preserved. Both superior laryngeal nerves were identified along their course to the cricothyroid muscles. A low tracheotomy was performed at the level of the suprasternal notch, through which an endotracheal tube was passed to allow ventilator-assisted respirations. A second tracheotomy was performed in a more superior location, through which a cuffed endotracheal tube was passed in a rostral direction and positioned with the tip 10 cm below the vocal folds. The cuff was inflated to just seal the trachea. Humidified heated air was passed through this rostral endotracheal tube from a compressed air tank. Flow was controlled with a valve and measured with a Gilmont flowmeter. The air was humidified and heated by being bubbled through 5 cm of heated water so that the temperature of the air was 37°C when measured at the glottic outlet. A 1-cm four-holed plastic shirt was used to suspend the epiglottis from a fixed point to provide direct visualization of the larynx through the oral cavity (Fig 1).

One-centimeter segments of recurrent and superior laryngeal nerves were isolated, and Harvard miniature electrodes were applied around each nerve. The electrodes were then insulated from surrounding tissue. Two constant-current nerve stimulators (model S2LH, WR Medical Electronics, St Paul, Minn) were used to stimulate the recurrent and superior laryngeal nerves independently. These nerves were stimulated at 70- to 80-Hz stimulus fre-

quency with 0.5- to 2.0-mA intensity for 1.5 milliseconds. Phonation was produced with an airflow of 318 to 523 mL/s applied through the larynx by the rostral endotracheal tube.

Surgical Technique. Arytenoid adduction was performed as described by Isshiki et al.[3] The thyroid cartilage was exposed down to the posterior margin. The constrictor muscles were elevated and sectioned off. Dissection proceeded on the inner surface of the thyroid cartilage. The mucosa of the pyriform recess was elevated to identify the muscular process of the arytenoid. One 4-0 braided nylon suture was placed around the muscular process of the arytenoid and then out through the thyroid ala by using a Keith needle. The tension on the stitch was adjusted with a clamp to allow maximum acoustic intensity for a given airflow rate and subglottic pressure.

The surgical augmentation used for vocal cord medialization followed the method of Hiroto.[35] This method was chosen because of the more caudal position of the vocal fold with respect to the thyroid cartilage in the dog as compared to the human. The external perichondrium of the medial lower margin of the thyroid cartilage was incised at the junction between the cricothyroid membrane and the thyroid cartilage, on the side of the paralyzed vocal fold. A tunnel was created, by using a Freer dissector, through the inner perichondrium. A rectangular piece of polystyrene plastic (Styrofoam) tapered at the anterior and posterior edge, 2 to 4 mm in thickness, was then placed in this tunnel to medialize the paralyzed cord. This piece of Styrofoam was reshaped as necessary to achieve optimal volume for a given airflow rate and subglottic pressure.

Acoustic Measures. Acoustic measures were made with a 1-in Bruel & Kjaer condenser microphone placed 30 cm from and level with the glottic outlet. The microphone was directed 90° from the direction of the sound source. The sound level measurements were made in decibels with a Bruel & Kjaer sound level meter type 2209 on the C-scale. The acoustic signal was also digitized, after C-scale filtering, at 20 kHz and stored on the hard disk of a personal computer.

Subglottic pressure was measured with a Millar Mikro-Tip catheter pressure transducer (model SPC-330, size 3F) passed rostrally through the superior tracheotomy. It was placed 5 cm below the glottis. This signal was low-pass-filtered at 3 kHz, digitized at 20 kHz, and stored in a personal computer. Owing to the variation in subglottic pressure during phonation, the peak pressures attained during the glottic cycle were used. These peaks were identified by using a commercially available software package for the personal computer system (C-Speech, Paul Milenkovic, University of Wisconsin, Madison, Wis). The pressure transducer was calibrated before each experiment against a mercury manometer.

Vocal efficiency was calculated as the ratio of the acoustic power of the voice to the subglottic power. The total acoustic power was calculated by the method of Koyama et al,[8] in which total sound power = $2r^2Pe^2/Poc$. This formula applies for a sound power radiating with no known directivity into a hemisphere of area $2r^2$, a distance "r" away from the source. The product of Po (the density of the medium) and c (the velocity of propagation) is the specific acoustic impedance of the medium, which is 41.1 dynes x s/cm^3 in air at 20°C. The term Pe is the root mean square sound pressure in dynes per cubic centimeter at the distance "r" from the sound source. The subglottic power was calculated as the product of the flow rate times the peak subglottic pressure.

Acoustic analysis of the digitized acoustic signal was performed with a commercial software program (C-Speech). Jitter, shimmer, and signal-to-noise ratio were calculated for each trial.[10] The background noise in the laryngeal laboratory was 35 dB lower than the experimental values, in using the C-scale. To normalize for varying fundamental frequency, jitter was calculated as a fraction of the period of the fundamental frequency.

Videostroboscopy. For stroboscopic imaging of the larynx, a Storz laryngostrobe unit (model 8000) was used. The stroboscope was connected to a Storz 0° telescope via a fluid-filled light cable. The image was detected by a Jedmed CCD (charge-coupled device) video camera (model 70-5110) and a Sony U-matic videocassette recorder (VO-5850). The video images were analyzed frame by frame with the videorecording unit.

Experimental Design. Eight adult mongrel dogs were used for this study. Measurements for vocal efficiency and acoustic analysis (jitter, shimmer, and signal-to-noise ratio) were obtained on a minimum of two to four trials, followed by videostroboscopy, for each experimental state: normal (simultaneous bilateral recurrent laryngeal nerve and superior laryngeal nerve stimulation), unilateral recurrent laryngeal paralysis, paralysis plus arytenoid adduction, paralysis plus surgical augmentation, and paralysis plus adduction and augmentation together. A mean value for vocal efficiency, jitter, shimmer, and signal-to-noise ratio was then computed from the trials for each experimental state. The order of the surgical manipulations was changed for each dog to reduce any ordering effects. Stimulation of the recurrent and superior laryngeal nerves in the normal state was optimized to create the greatest intensity, because vocal efficiency has been found to increase with intensity.[10]

Statistical Analysis. Each experimental state (normal, unilateral recurrent laryngeal nerve paralysis,

TABLE 1. VOCAL EFFICIENCY

Dog	*Normal*	*Paralysis*	*Arytenoid Adduction*	*Augmentation*	*Adduction and Augmentation*
1	3.0	0.098	2.4	0.69	4.6
2	14	1.5	18	1.3	8.9
3	2.8	0.055	2.5	0.095	2.4
4	23	0.065	9.8	0.065	11
5	11	0.2	11	0.68	45
6	43	1.4	2.1	3.9	3.3
7	70	0.065	14	0.35	4.9
8	49	1.3	57	1	63
Mean	27	0.58	15	1	18

All values are times 10^{-4}.

paralysis plus arytenoid adduction, paralysis plus augmentation, and paralysis plus adduction and augmentation together) was compared for statistical significance by using a one-way analysis of variance for repeated measures for the dependent variables vocal efficiency, jitter, shimmer, and signal-to-noise ratio.

RESULTS

Vocal Efficiency. Table 1 lists for each dog the vocal efficiency for each experimental state. For the normal canine larynx the vocal efficiency varied from 3 x 10^{-4} to 1 x 10^{-3} with a mean of 2.7 x 10^{-3}. For recurrent laryngeal nerve paralysis the vocal efficiency decreased in every case by at least a factor of 10. The values for paralysis varied from 5.5 x 10^{-6} to 1.3 x 10^{-4} with a mean of 5.8 x 10^{-5}. Even the least efficient normal larynx was more efficient than the most efficient larynx with recurrent laryngeal nerve paralysis.

The efficiency of the paralyzed larynx after arytenoid adduction varied from 2.1 x 10^{-4} to 1.8 x 10^{-3} with a mean of 1.5 x 10^{-3}. In every case, the arytenoid adduction improved the vocal efficiency of the paralyzed larynx. The degree of improvement varied by a factor of 1.5 to 150.

The efficiency of the paralyzed larynx after surgical augmentation varied from 6.53 x 10^{-6} to 3.9 x 10^{-4} with a mean of 1.0 x 10^{-4}. In all cases but two, there was an improvement in the efficiency over the paralyzed state. The degree of improvement varied by a factor of 0.3 to 23.

When both arytenoid adduction and surgical augmentation were used together on the paralyzed larynx, the vocal efficiency varied from 2.4 x 10^{-4} to 4.5 x 10^{-3}, with a mean of 1.8 x 10^{-3}. The degree of improvement over the paralyzed larynx varied by a factor of 2.4 to 222. In every case, arytenoid adduction and augmentation together improved the efficiency of the paralyzed larynx.

The vocal efficiency scores for arytenoid adduction and arytenoid adduction and augmentation to-

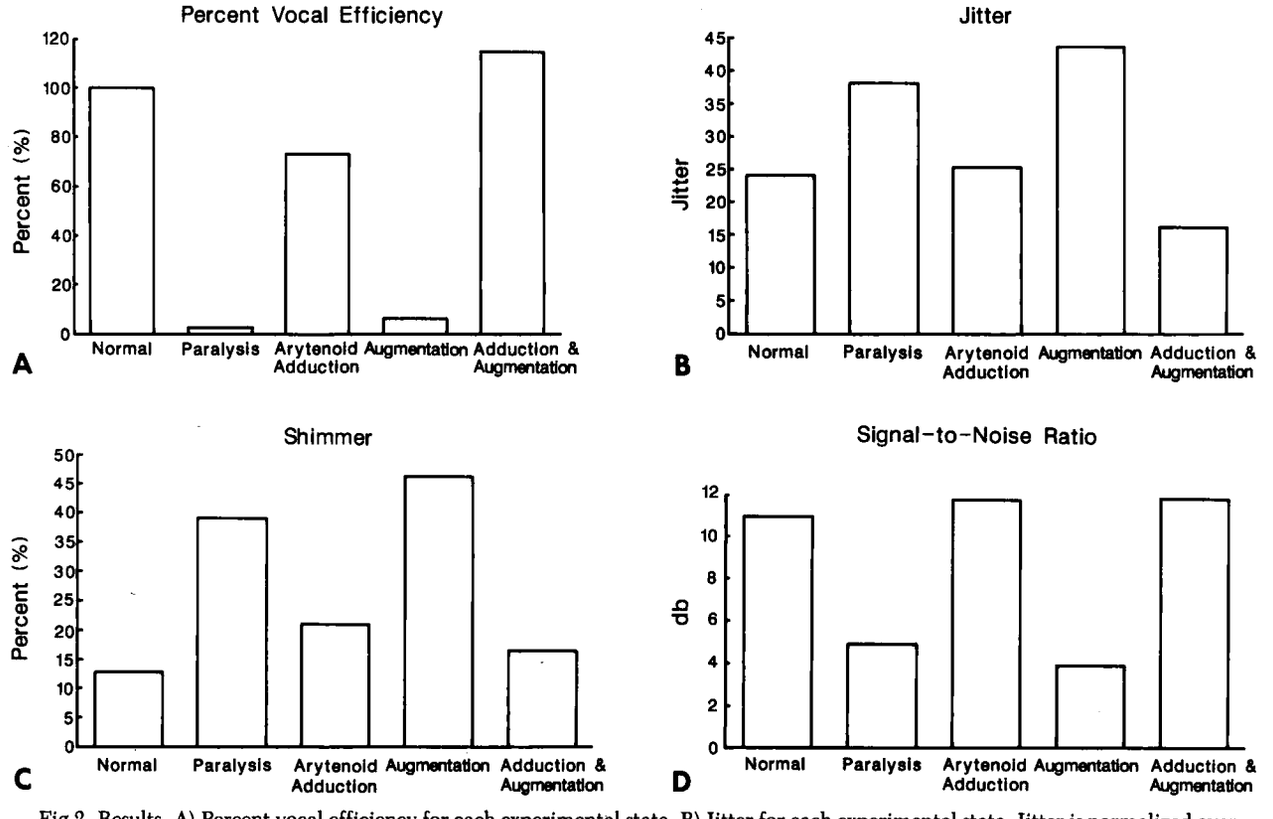

Fig 2. Results. A) Percent vocal efficiency for each experimental state. B) Jitter for each experimental state. Jitter is normalized over period (jitter ms/period ms) and expressed as times 10^{-1}. C) Shimmer for each experimental state. D) Signal-to-noise ratio for each experimental state, expressed as dB = 20log10(signal intensity/background intensity).

TABLE 2. SUMMARY OF STROBOSCOPIC ANALYSIS

Laryngeal State	*Glottic Closure*	*Mucosal Motion*	*Cord Motion*
Normal	Complete	Two-mass bilaterally	Sym
Paralysis	Incomplete; PGL	One-mass on paralyzed cord	Asym
Arytenoid adduction	Complete	Two-mass bilaterally	Asym
Augmentation	Incomplete; PGL	One-mass on paralyzed cord	Sym
Adduction and augmentation	Complete	Two-mass bilaterally; Asym movement of upper margin	Sym

PGL — posterior glottic leak, Sym — symmetric, Asym — asymmetric.

gether were significantly different from those for recurrent laryngeal nerve paralysis. Augmentation was not found to be significantly different from the paralysis state. Arytenoid adduction was not found to be significantly different from adduction and augmentation together ($F_{4,28} = 4.51$; $p < .01$).

In order to aid in comparisons between dogs, the efficiency of the normal larynx for each dog was expressed as 100% efficient, and the efficiency of the other states of the larynx was expressed as a percentage of normal. These results are shown in Fig 2A. The mean percent efficiency, across all dogs, was 3.0% for unilateral recurrent laryngeal nerve paralysis, 73% for arytenoid adduction, 7% for augmentation, and 115% for arytenoid adduction and augmentation together.

Acoustic Analysis. Figure 2B-D illustrates the jitter, shimmer, and signal-to-noise ratio, respectively, for each experimental state. The mean jitter values for unilateral recurrent laryngeal nerve paralysis and surgical augmentation were similar at 38 and 43, respectively. Jitter values for the normal larynges, arytenoid adduction, and arytenoid adduction and augmentation together were significantly lower ($F_{4,28} = 2.63$; $p = .05$) at 24, 25, and 17, respectively.

The mean shimmer values for unilateral recurrent laryngeal nerve paralysis and surgical augmentation were also similar at 39 and 47, respectively. Shimmer values for the normal larynges, arytenoid adduction, and arytenoid adduction and augmentation together were also significantly lower ($F_{4,28} = 4.17$, $p < .01$) at 13, 21, and 16, respectively.

The signal-to-noise ratios for unilateral recurrent laryngeal nerve paralysis and augmentation were similar at 5 and 4, respectively. Signal-to-noise values for the normal group, arytenoid adduction, and arytenoid adduction and augmentation together were significantly higher ($F_{4,28} = 5.88$; $p < .001$) at 11, 12, and 12, respectively.

In general, a unilateral recurrent laryngeal nerve paralysis resulted in a weaker acoustic signal (signal-to-noise ratio) with an increase in frequency and amplitude perturbation (jitter and shimmer). Arytenoid adduction or arytenoid adduction and augmentation together corrected these measures to a greater degree than surgical augmentation alone.

Stroboscopic Analysis. The stroboscopic data are summarized in Table 2. Phonation in the normal larynx revealed complete glottic closure and two-mass (upper and lower margin) motion of the mucosa on both cords, with complete symmetry. Unilateral recurrent laryngeal nerve paralysis revealed a mild posterior glottic chink incompetence and one-mass motion of the paralyzed cord. In addition, the paralyzed cord made greater lateral excursions than the normal cord, which remained more in the midline.

Surgical augmentation resulted in continued glottic incompetence at the posterior chink. The mucosa of the paralyzed cord had one-mass motion, while the normal cord had two-mass motion. The excursions of the two cords from midline were symmetric (as opposed to the paralyzed state).

Arytenoid adduction resulted in complete glottic closure and two-mass motion of the vocal fold mucosa bilaterally. The paralyzed cord demonstrated a greater lateral excursion from the midline, similar to that in the paralyzed state.

Arytenoid adduction and augmentation together resulted in complete glottic closure, with two-mass motion in the vocal fold mucosa bilaterally, but the upper margin of the paralyzed cord had a wider excursion of oscillation, crossing the midline after contact with the opposite cord. The motion of the bulk of the cords was symmetric, however.

DISCUSSION

In addition to the sound produced by the larynx, vocal efficiency is affected by the vocal tract and mouth radiation. In this study these last two factors were held constant so that glottic efficiency was measured. Koyama et al[8] found in dogs the normal range for vocal efficiency to be 0.06×10^{-4} to 2.0×10^{-3}, which agrees with our values of 3×10^{-4} to 1×10^{-3} with a mean of 2.7×10^{-3}. This value for canine vocal efficiency also approximates the range for humans of 0.45×10^{-5} to 45×10^{-5} found by van den Berg,[7] 0.3×10^{-4} to 14.0×10^{-4} by Isshiki,[36] and 0.11×10^{-4} to 6.8×10^{-4} by Iwata.[10]

In this study we examined vocal fold medialization by surgical augmentation and arytenoid adduction in treating unilateral recurrent laryngeal nerve paralysis in the dog. Vocal efficiency improved from approximately 3% of normal in the paralyzed state to only 7% after augmentation. The jitter, shimmer, and signal-to-noise ratio of surgical augmentation were also comparable to those of the paralyzed state.

Examination of the videostroboscopic images

demonstrated that some of the failures of augmentation to improve vocal efficiency were from a leak of air in the region of a posterior glottic chink. This leak of air was a source of lost subglottic air power that was not converted to acoustic power. Titze,[37] using the excised canine larynx, found vocal efficiency to decrease with increasing glottal width. From both canine and human anatomic studies it has been shown that a type I thyroplasty cannot medialize the vocal process of the arytenoid, owing to the overlap of the cricoid cartilage under the posterior thyroid ala. Any attempt at medializing the vocal process using the thyroid lamina would just push against the posterior cricoid.[38]

The noise of turbulent flow from this posterior glottic leak may have contributed to the low signal-to-noise ratio of the surgical augmentation. Isshiki et al[39] found, using a life-size silicone model of the larynx, that turbulent noise was produced at the glottic chink when the speed of airflow through the chink exceeded a certain value (the critical Reynolds number). The intensity of the noise in Isshiki's model was proportional to the area of the glottic chink. The turbulent noise produced was distributed over a wide frequency range as in white noise.

There are some limitations in using the acute canine model of laryngeal paralysis. The posterior glottic chink between the vocal processes is longer in the dog than in the human. This difference may limit the performance of a surgical augmentation procedure in the dog. The problem of a leak in the posterior glottic chink may not be as significant a factor in the human. Using an acute model of laryngeal paralysis does not allow for atrophy of the paralyzed cord or compensation by the normal cord with time. These are interesting problems that deserve further investigation.

In this study, arytenoid adduction resulted in an average vocal efficiency that was 73% of normal (compared with 3% in the paralyzed state and 7% after surgical augmentation). The jitter and shimmer after arytenoid adduction were similar to those of the normal larynx and lower than those for the paralyzed state or after surgical augmentation. The signal-to-noise ratio for arytenoid adduction was also similar to normal and higher than for the paralyzed state or surgical augmentation. The values for arytenoid adduction and augmentation together seemed to follow those of arytenoid adduction alone.

It is difficult to speculate if the advantage of the arytenoid adduction was due to the dog's large posterior glottic chink. Although this study indicates that arytenoid adduction may be superior, in terms of vocal efficiency and acoustic analysis, to augmentation, careful preoperative and postoperative evaluation in humans is necessary.

The stroboscopic vibratory patterns of the vocal cords continued to demonstrate abnormalities after all treatment modalities. The larynges with arytenoid adduction, despite having two-mass motion of both mucosal folds, continued to have wide lateral excursions of the paralyzed cord. The augmentation failed to yield two-mass motion of the paralyzed cord, in addition to leaving a posterior chink leak. Even using arytenoid adduction and augmentation together resulted in an upper margin of the paralyzed cord that vibrated in wide excursions, crossing the midline after meeting the opposite cord. The reason these procedures failed to result in normal vocal cord vibration may be the lack of intrinsic vocalis muscle tone and stiffness provided by innervation. This intrinsic stiffness may be preserved by using reinnervation procedures.

Crumley et al[4] have recommended nerve transfer from the ansa hypoglossi to the recurrent laryngeal nerve as a treatment for unilateral vocal cord paralysis. This procedure was performed on five patients and the voice was thought to be superior to that produced by Teflon injection, owing to the restoration of normal stiffness, mass, and symmetry of the cord. Results on spectral analysis were improved postoperatively,[40] and stroboscopic examination revealed synchronous mucosal waves. This technique required an open procedure but did not expose or manipulate the larynx. Tucker and Rusnov[5] have also described a technique for laryngeal reinnervation using an ansa hypoglossi neuromuscular pedicle passed through a window in the thyroid cartilage.

How the reinnervation procedures would compare to surgical augmentation or arytenoid adduction in terms of vocal efficiency or acoustic analysis is presently under investigation.

Vocal fold medialization by surgical augmentation or arytenoid adduction, in this study, improved the vocal efficiency of the paralyzed larynx, but to different degrees. Under conventional reporting standards both results would be described as "good" or "improved." It was only through more objective measures, such as vocal efficiency and acoustic analysis, that any quantitative difference could be found. In this canine model, the vocal efficiency and acoustic properties of arytenoid adduction were superior to those of augmentation. As the number of treatments for unilateral vocal cord paralysis increases, it becomes important to document the results of each treatment by using objective measures. Practical approaches for achieving this goal have been reported, and it is our hope that the use of these methods will continue.[41-46]

CONCLUSIONS

Vocal fold medialization by surgical augmentation was compared to arytenoid adduction as a treatment for unilateral vocal cord paralysis. In this canine model, the vocal efficiency and acoustic properties of arytenoid adduction were superior to those of surgical augmentation.

REFERENCES

1. Arnold GE. Vocal rehabilitation of paralytic dysphonia. VIII. Phoniatric methods of vocal compensation. Arch Otolaryngol 1962;76:76-83.

2. Isshiki N, Okamura H, Ishikawa T. Thyroplasty type I (lateral compression) for dysphonia due to vocal cord paralysis or atrophy. Acta Otolaryngol (Stockh) 1975;80:465-73.

3. Isshiki N, Tanabe M, Sawada M. Arytenoid adduction for unilateral vocal cord paralysis. Arch Otolaryngol 1978;104:555-8.

4. Crumley RL, Izdebski K, McMicken B. Nerve transfer versus Teflon® injection for vocal cord paralysis: a comparison. Laryngoscope 1988;98:1200-4.

5. Tucker HM, Rusnov M. Laryngeal reinnervation for unilateral vocal cord paralysis: long-term results. Ann Otol Rhinol Laryngol 1981;90:457-9.

6. Smith ME, Berke GS. The effects of phonosurgery on laryngeal vibration: Part 1. Theoretic considerations. Otolaryngol Head Neck Surg 1990;103:380-90.

7. Van den Berg JW. Direct and indirect determination of the mean subglottic pressure. Folia Phoniatr (Basel) 1956;8:1-24.

8. Koyama T, Harvey JE, Ogura JH. Mechanics of voice production. III. Efficiency of voice production. Laryngoscope 1972; 82:210-7.

9. Tanaka S, Tanabe M. Glottal adjustment for regulating vocal intensity. An experimental study. Acta Otolaryngol (Stockh) 1986;102:315-24.

10. Iwata S. Aerodynamic aspects for phonation in normal and pathologic larynges. In: Fujimura O, ed. Vocal physiology: voice production, mechanisms and functions. New York, NY: Raven Press, 1988:423-31.

11. Tanaka S, Gould WJ. Vocal efficiency and aerodynamic aspects in voice disorders. Ann Otol Rhinol Laryngol 1985;94: 29-33.

12. Ludlow C, Coulter D, Gentges F. The differential sensitivity of frequency perturbation to laryngeal neoplasms and neuropathologies. In: Bless DM, Abbs JH, eds. Vocal fold physiology: contemporary research and clinical issues. San Diego, Calif: College-Hill Press, 1983:381-92.

13. Milenkovic P. Least mean squares measures of voice perturbation. J Speech Hear Res 1987;30:529-38.

14. Lieberman P. Some acoustic measurements of the fundamental periodicity of normal and pathologic larynges. J Acoust Soc Am 1963;35:344-53.

15. Koike Y, Takahashi H, Calcaterra TC. Acoustic measures for detecting laryngeal pathology. Acta Otolaryngol (Stockh) 1977;84:105-17.

16. Zyski B, Bull G, McDonald W, Johns M. Perturbation analysis of normal and pathologic larynges. Folia Phoniatr (Basel) 1984;36:190-8.

17. Iwata S. Periodicities of pitch perturbations in normal and pathologic larynges. Laryngoscope 1972;82:87-96.

18. Fukuzawa T, Blaugrund S, El-Assuooty A, Gould W. Acoustic analysis of hoarse voice: a preliminary report. J Voice 1988;2:127-31.

19. Von Leden H, Abitbol A, Bouchayer M, Hirano M, Tucker H. Phonosurgery. J Voice 1989;3:175-82.

20. Rubin HJ. Histologic and high-speed photographic observations on the intracordal injection of synthetics. Trans Am Acad Ophthalmol Otolaryngol 1966;70:909-21.

21. Von Leden H, Yanagihara N, Werner-Kukuk E. Teflon in unilateral vocal cord paralysis. Preoperative and postoperative function studies. Arch Otolaryngol 1967;85:666-74.

22. Rontal E, Rontal M, Rolnick MI. The use of spectrograms in the evaluation of vocal cord injection. Laryngoscope 1975;85: 47-56.

23. Dedo H. Avoidance and treatment of complications of Teflon injection of the vocal cord. J Voice 1988;2:90-2.

24. Trapp TK, Berke GS, Bell TS, Hanson DG, Ward PH. Effect of vocal fold augmentation on laryngeal vibration in simulated recurrent laryngeal nerve paralysis: a study of Teflon and Phonogel. Ann Otol Rhinol Laryngol 1989;98:220-7.

25. Lewy RB. Teflon injection of the vocal cord: complications, errors, and precautions. Ann Otol Rhinol Laryngol 1983; 92:473-4.

26. Cormier Y, Kashima H, Summer W. Subclinical reduction in airflows after Teflon injection of vocal cord. Laryngoscope 1980;90:1027-31.

27. Payer. Plastik am Schildknorpel zur Behebung der Folgan einseitiger Stimmbandlahmung. Dtsch Med Wochenschr 1915; 43:1265-70.

28. Meurman Y. Operative mediofixation of the vocal cord in complete unilateral paralysis. Arch Otolaryngol 1952;55:544-53.

29. Isshiki N, Morita H, Okamura H, Hiramoto M. Thyroplasty as a new phonosurgical technique. Acta Otolaryngol (Stockh) 1974;78:451-7.

30. Isshiki N. Recent advances in phonosurgery. Folia Phoniatr (Basel) 1980;32:119-54.

31. Koufman JA. Laryngoplasty for vocal cord medialization: an alternative to Teflon®. Laryngoscope 1986;96:726-31.

32. Escajadillo JR. Technique for external repositioning of the paralyzed vocal cord with Silastic implant. Ann Otol Rhinol Laryngol 1988;97:234-8.

33. Morrison LF. The "reverse King operation." A surgical procedure for restoration of phonation in cases of aphonia due to unilateral vocal cord paralysis. Ann Otol Rhinol Laryngol 1948; 57:945-56.

34. Montgomery WW. Cricoarytenoid arthrodesis. Ann Otol Rhinol Laryngol 1966;75:380-91.

35. Hiroto I. Surgical voice improvement for unilateral recurrent laryngeal nerve paralysis. Otol Fukuoka (Jibi To Rinsho) 1976;22:473-6.

36. Isshiki N. Regulatory mechanism of voice intensity variation. J Speech Hear Res 1964;7:17-29.

37. Titze IR. Regulation of vocal power and efficiency by subglottal pressure and glottal width. In: Fujimura O, ed. Vocal physiology: voice production, mechanisms and functions. New York, NY: Raven Press, 1988:227-37.

38. Isshiki N. Phonosurgery: external laryngeal surgery not requiring entry into the vocal tract. In: Arnold GE, Winckel BD, eds. Surgical care of voice disorders. Vienna, Austria: Springer-Verlag, 1984:59-84. (Disorders of human communication; vol 8.)

39. Isshiki N, Kitajima K, Kojima H, Harita Y. Turbulent noise in dysphonia. Folia Phoniatr (Basel) 1978;30:214-24.

40. Crumley RL, Izdebski K. Voice quality following laryngeal reinnervation by ansa hypoglossi transfer. Laryngoscope 1986;96:611-6.

41. Berke GS, Hanson DH, Trapp TK, Moore DM, Gerratt BR, Natividad M. Office-based system for voice analysis. Arch Otolaryngol 1989;115:74-7.

42. Sataloff RT, Spiegel JR, Carroll LM, Darby KS, Rulnick RK. Objective measures of voice function. Ear Nose Throat J 1987;66:307-12.

43. Bastian RW. Factors leading to successful evaluation and management of patients with voice disorders. Ear Nose Throat J 1988;67:411-20.

44. Bastian RW, Levine LA. Visual methods for the office diagnosis of voice disorders. Ear Nose Throat J 1988;67:363-79.

45. Thumfart WF. Electrodiagnosis of laryngeal nerve disorders. Ear Nose Throat J 1988;67:380-93.

46. D'Antonio L, Netsell R, Lotz W. Clinical aerodynamics for the evaluation and management of voice disorders. Ear Nose Throat J 1988;67:394-9.