An In Vivo Canine Model for Testing Treatment Effects in Laryngeal Hyperadduction Disorders

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Adductor spastic dysphonia is a voice disorder characterized by a strained, squeezed, effortful voice produced by true and false cord hyperadduction. An in vivo canine model has been developed to simulate hyperadduction of the true cords. Using this model, the thyroarytenoid muscle was found to have a greater effect on intraglottic and subglottic pressure than cricothyroid muscle contraction. The intraglottic and subglottic pressure was reduced after simulated recurrent laryngeal paralysis. This model can be used in future studies to compare laryngeal treatment modalities for disorders that have a component of vocal cord hyperadduction, such as spastic dysphonia.

INTRODUCTION

Spastic dysphonia is a form of nervous hoarseness first described by Traube in 1871.[1] The diagnosis can be difficult and involves a subjective evaluation of the patient's speech and phonation.[2] Laryngoscopy, acoustic analysis, glottography, and electromyography have not been found to be uniquely diagnostic. Aronson has divided this disorder into two types: adductor and abductor. The adductor type is characterized by a strained, squeezed, effortful voice produced by true and false cord hyperadduction. The abductor type is characterized by an abrupt widening of the glottis, which produces aphonia or a breathy dysphonia.[3]

The exact cause of spastic dysphonia is unknown. In the past, the disorder was thought to reflect psychiatric problems. There was often little improvement with counseling or neuropharmacologic medication. Despite the name, spastic dysphonia is not thought to be a spastic disease. Blitzer, *et al.* found no irregularity of firing, dyssynchrony, or slow firing of motor potential units by electromyography indicative of a spasticity. They found, through clinical and electromyographic (EMG) evaluation, that many patients with "spastic dysphonia" actually have a dystonia. Dystonia is a neurologic disorder of motor control processing characterized by abnormal, often action-induced, involuntary movements or uncontrolled spasms. The etiology is usually idiopathic. The dystonia might be restricted to the larynx or present in other areas of the body as well, such as blepharospasm.[4]

Cohen, *et al.* studied the blink reflexes of 12 patients with spastic dysphonia and no symptomatic eye involvement.[5] These patients were found to have increased excitability of blink reflexes compared to healthy controls, which suggested that a focal laryngeal dystonia might subclinically involve other anatomical structures.

Blitzer, *et al.* found the mean age of onset for spastic dysphonia was 34.6 years old with a female to male ratio of 1.4:1.[6] A family history of dystonia was present in 23% of the patients with primary laryngeal dystonia. The majority of the patients had adductor spastic dysphonia, but three rarer forms of laryngeal dystonia were described. The first of these was the abductor form of laryngeal dystonia, in which the patient had spasmodic contraction of the posterior cricoarytenoid during phonation that resulted in a breathy voice. The second rare form was compensatory abductor spastic dysphonia, in which patients with adductor dysphonia did not contract their vocal cords to avoid spasm during phonation. Finally, there was compensatory adductor spastic dysphonia, which was found in patients with abductor dysphonia who tried to prevent breathiness.

Ludlow and Connor, using speech tasks and laryngeal motor exercises, found evidence that only the muscles involved in vocal cord adduction were affected in patients with spastic dysphonia. They also found that spastic dysphonia patients phonated with a higher intensity, which they attributed to the patients using a higher subglottic pressure to overcome the excessive adductory force.

Ludlow, *et al.* studied intrinsic laryngeal muscle

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activation using electromyography and found abnormally high resting levels in both the thyroarytenoid and cricothyroid muscles. During phonation there were bursts of thyroarytenoid and cricothyroid muscle activity with an imbalance between these muscles that favored the thyroarytenoid, and resulted in excessive adduction and shortening of the vocal folds.[7]

There are a variety of treatments for adductor spastic dysphonia which include speech therapy, psychotherapy, biofeedback, systemic medicine, nerve section, botulinum toxin injection and thyroplasty.

Dedo was the first to describe recurrent laryngeal nerve section as a treatment for spastic dysphonia.[8] The aim was to achieve a voice that was slightly breathy but easier to produce. After a 3-year follow-up, Izdebski, *et al.* reported a 10% to 15% recurrence rate using patient self-evaluations.[9] Patients who experienced a recurrence still thought they had a better voice than before the nerve section. The most common complaint from the patients was an inability to speak loudly when necessary.

Aronson and DeSanto reported an initial success rate of 97% at 6 months for recurrent laryngeal nerve section.[10] This success rate fell to 36% at 3 years' follow-up. The failures showed gradual hyperadduction of the intact cord against the paralyzed cord, which was thought to be due to a worsening of the patient's underlying neurologic condition.

Biller, *et al.* (1979) reported crushing the recurrent laryngeal nerve as a treatment for spastic dysphonia.[11] Although all patients experienced initial improvement, only 13% were improved at 3 years' follow-up.[12]

Friedman, *et al.* (1987) reported a clinical and canine study using direct unilateral electrical stimulation of the recurrent laryngeal nerve to improve spastic dysphonia.[13] All five patients in this study experienced improvement during the period of electrical stimulation. The stimulation was thought to inhibit excessive impulses to the recurrent laryngeal nerve from the brain or rectify an irregular pattern. The stimulation did not cause any gross change in cord position or cardiac arrhythmias. Implantation of electrodes around the recurrent laryngeal nerve in the canine for 6 months caused only perineural and epineural fibrosis, with no evidence of damage to the nerve itself.

Blitzer, *et al.* reported the first series of patients with focal laryngeal dystonia to be treated with local cord injections of botulinum toxin (BOTOX).[14] BOTOX acts presynaptically at nerve terminals to prevent calcium-dependent release of acetylcholine. There were five patients in this series and, within 2 to 3 days, all experienced benefit that lasted 3 to 6 months. Although the authors initially injected only one cord, bilateral injections are now performed. There were no systemic side effects from the injections, but the authors stated that the long-term systemic or local laryngeal effects were unknown.

Ludlow, *et al.* studied patients with adductor spastic dysphonia who failed unilateral recurrent laryngeal nerve section, using electromyography. They found that some of the failures were due, at least in part, to reinnervation of the thyroarytenoid by the previously sectioned recurrent laryngeal nerve, where compensation by the nonoperated side might have also played a role in others.[15] These patients were treated successfully with BOTOX injections. Some patients experienced a reduction in the electromyographic activity of both cords despite a unilateral injection into the previously operated cord. This finding was also demonstrated in patients who have not had previous surgery,[16] which suggested that one aspect of spastic dysphonia might be hypersensitivity to afferent muscle action feedback.

To date there have been no animal models to aid in evaluating different treatment modalities or to gain a better understanding of the mechanism of hyperadduction in disorders such as spastic dysphonia. This model was developed to investigate the role of hyperadduction of the arytenoids, as well as thyroarytenoid and cricothyroid muscle contraction on intraglottic and subglottic pressure. The model was then used to compare the variety of laryngeal treatments used to relieve the hyperadductive component of adductor spastic dysphonia. Intraglottic pressure was used as a measure of the hyperadductory force of the cords.

MATERIALS AND METHODS

In Vivo Canine Model

Figure 1 schematically depicts the in vivo canine model for spastic dysphonia. 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 and a pneumotachygraph with a differential pressure transducer (Fluid Precision Inc., Billerica, Mass., Model #183) to record input airflow. The air flow was humidified and heated by bubbling it 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 button was used to suspend the epiglottis from a fixed point to provide direct visualization of the larynx through the oral cavity.

A 1-cm segment of recurrent and superior laryngeal nerves was isolated and Harvard miniature electrodes were applied around

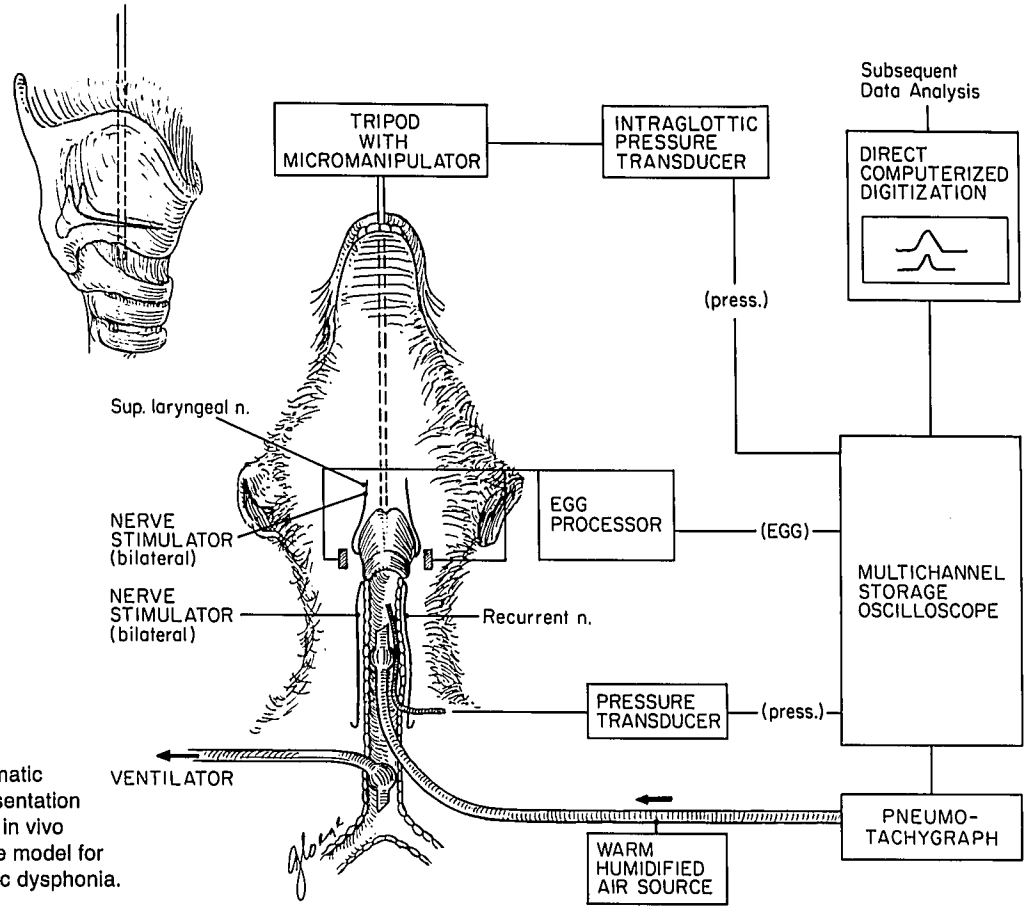

Fig. 1. Schematic representation of the in vivo canine model for spastic dysphonia.

each nerve. The electrodes were then insulated from surrounding tissue. A constant current nerve stimulator (WR Medical Electronics Co. St. Paul, Minn., Model S2LH) was used to stimulate the recurrent laryngeal nerve (RLN) and a constant voltage source (Grass Model 54H, WPI 301-T) was used to stimulate the superior laryngeal nerves (SLN). These nerves were stimulated at a 70- to 80-Hz stimulus frequency, with 0.5- to 1.5-mA (RLN) or 0- to 1.4-V (SLN) intensity for a 1.5-msec duration. There was no observed contraction of the cricothyroid muscle during maximal stimulation of the RLN at 2.0 mA. Similarly, there was no bulging or contraction of the thyroarytenoid muscle, or movement of the arytenoid during maximal stimulation of the SLN at 3.0 V. Phonation was produced with an airflow of 50 to 450 cc/sec applied through the larynx by the rostral endotracheal tube.

Subglottic pressure was measured using Millar Mikro-Tip catheter pressure transducers (Model No. SPC-330, size 3F). The subglottic pressure transducer was passed rostrally through the superior tracheotomy, and was placed 5 cm below the glottis.

The intraglottic pressure was also measured using a Millar Mikro-Tip catheter pressure transducer as described by Sin, *et al.*[17] The intraglottic pressure transducer was placed between the true cords midway between the anterior commissure and the vocal process of the arytenoids. The membrane of the catheter was placed flat onto the surface of the cord, midway between the upper and lower margins of the cord. The pressure transducers were calibrated before each experiment against a mercury manometer.

Electroglottographic (EGG) signals were obtained with a laryngograph (Synchrovoice, Harrison, NJ) with the two recording electrodes sutured into place on the right and left thyroid ala, just above the cricothyroid muscles. The reference electrode was secured to an adjacent strap muscle.

The EGG, glottic air flow, intraglottic and subglottic pressure signals were low-pass filtered at 2 kHz, digitized at 5 kHz, and stored on the hard disk of a personal computer. Due to the cyclic variation in subglottic pressure during phonation, the peak pressures attained during the glottic cycle were used. Measurements of the waveforms were made using a commercially available software package for the PC system ("C-Speech," Paul Milenkovic, University of Wisconsin, Madison).

Surgical Technique

In order to examine the separate effects of thyroarytenoid and cricothyroid contraction on intraglottic pressure, it was necessary to independently maintain forceful adduction of the arytenoids. This was achieved by performing a bilateral arytenoid adduction as described by Isshiki, *et al.*[18] 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 using a Keith needle. The anterior tension on the stitch was adjusted to 200 g of force.

Experimental Design

After performing a bilateral arytenoid adduction, the static intraglottic pressure was measured with laryngeal nerve stimulation for 2 seconds with no air flow through the larynx. This was done as a relative measure of the adductive pressure between the cords. The air-flow rate was then gradually increased from 0 to 450 cc/sec over the next 8 seconds. As the subglottic pressure increased, the

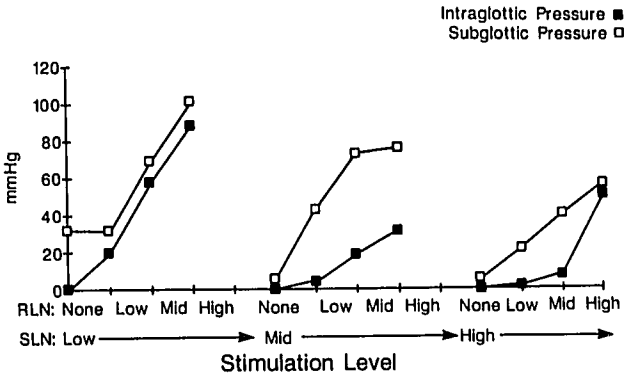

Fig. 2. Intraglottic and subglottic pressures as a function of RLN and SLN stimulation.

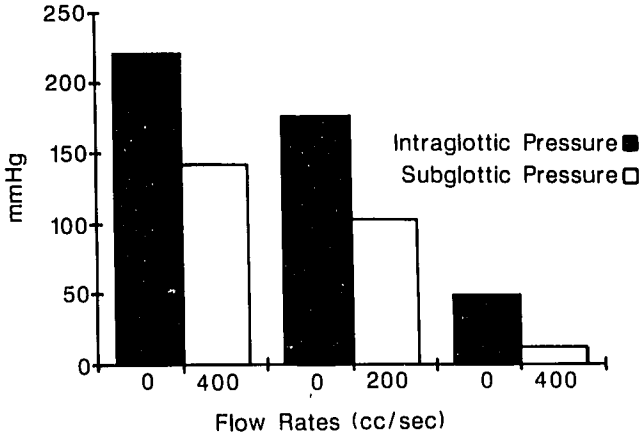

Fig. 3. Intraglottic and subglottic pressures at glottic flow rates of 200 and 400 cc/s.

onset of phonation was detected by the oscillation of the EGG signal. The peak subglottic pressure at the onset of phonation was recorded. The intraglottic and subglottic pressures were recorded for bilateral high (1.4 mAmp), middle (0.9 mAmp), low (0.8 mAmp), and no recurrent laryngeal nerve stimulation, in combination with high (1.4 V), mid (1.0 V), and no superior laryngeal nerve stimulation. The intraglottic pressure was then measured before and after a simulated recurrent laryngeal paralysis, at high recurrent laryngeal nerve stimulation and low, middle, and high superior laryngeal nerve stimulations. Subglottic pressure during phonation was also measured during hyperadduction, before and after simulated recurrent laryngeal nerve paralysis.

RESULTS

Mechanism of Hyperadduction

Figure 2 graphically depicts the role of the vocalis and cricothyroid muscle contraction on intraglottic pressure in the presence of arytenoid hyperadduction. In the presence of a constant level of SLN stimulation and no recurrent laryngeal nerve (RLN) stimulation, arytenoid hyperadduction alone caused no increase in intraglottic pressure. Increasing levels of RLN stimulation, with constant SLN stimulation, caused an increase in the intraglottic pressure due to thyroarytenoid muscle contraction, in the presence of arytenoid hyperadduction. The intraglottic pressures ranged from a low 0 mm Hg with no RLN stimulation, to a high of 88 mm Hg during high levels of stimulation.

In the presence of hyperadduction of the arytenoids and constant RLN stimulation, increasing levels of SLN stimulation with cricothyroid contraction caused little change in the intraglottic pressure. This indicated that one of the necessary factors in raising intraglottic pressure was vocalis muscle contraction rather than cricothyroid contraction in the presence of arytenoid hyperadduction.

Control of Subglottic Pressure

Figure 2 graphically depicts the role of the vocalis and cricothyroid in controlling subglottic pressure in the presence of hyperadducted arytenoids. In the presence of a constant level of SLN stimulation and no RLN stimulation, arytenoid hyperadduction alone caused little or no increase in subglottic pressure. During a constant level of SLN stimulation, increasing levels of RLN stimulation caused an increase in the subglottic pressure. For hyperadduction of the arytenoids alone, without RLN stimulation, increasing levels of SLN stimulation did not increase subglottic pressure. In the presence of constant RLN stimulation, increasing levels of SLN stimulation caused little change in the subglottic pressure. Subglottic pressure recordings indicated a low of 4 mm Hg and a high of 101 mm Hg during high levels of RLN stimulation.

Although the subglottic pressures were higher than the intraglottic pressures, for each of the corresponding levels of nerve stimulation, the two pressures did seem to parallel each other. This correlation implied that, in the presence of hyperadducted arytenoids, vocalis contraction controls intraglottic pressure, which then determined (along with other factors) the subglottic pressure.

It is important to note that, in the presence of hyperadducted arytenoids, the minimum subglottic pressure at which phonation occurred was determined by the degree of vocalis contraction. Also, there was no correlation between subglottic pressure and the glottic air-flow rate at the onset of phonation.

The Effect of Flow Rate on Subglottic Pressure

To investigate the role of glottic air-flow rate on the minimum subglottic pressure in the presence of hyperadducted arytenoids, the subglottic pressure was measured for two similar intraglottic pressures at a flow rate of 200 and 400 cc/sec. Figure 3 shows that, despite a doubling of the flow rate, the subglottic pressure was determined to a greater degree by the intraglottic pressure. This small nonproportional change in subglottic pressure with flow rate was in agreement with the findings of Smith and Berke.[19]

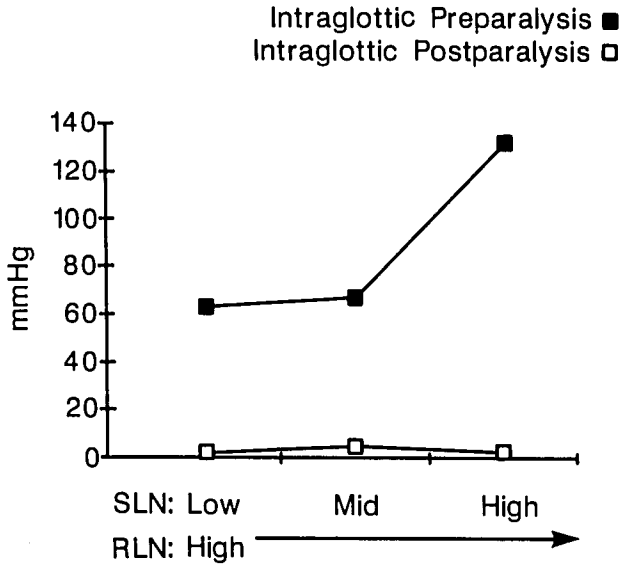

Fig. 4. Intraglottic pressures during high RLN stimulation and different levels of SLN stimulation, before and after unilateral recurrent laryngeal nerve stimulation.

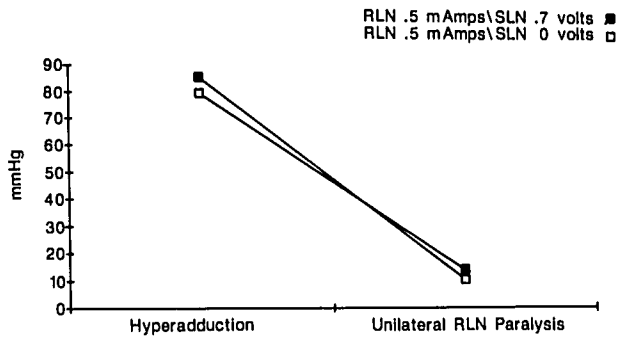

Fig. 5. Subglottic pressure during hyperadduction (high vocalis stimulation) before and after recurrent laryngeal nerve stimulation at two different cricothyroid stimulation levels: high and low.

Effect of Recurrent Laryngeal Paralysis on Intraglottic Pressure

Figure 4 graphically depicts the intraglottic pressure before and after a simulated unilateral RLN paralysis in the presence of hyperadduction of the arytenoids. The comparisons were made during these relatively high levels of RLN stimulation (high intraglottic pressure), and three different levels of SLN stimulation. For low, mid, and high levels of RLN stimulation, the intraglottic pressure fell from 63, 68, and 133 mm Hg preparalysis to 2.5, 5, and 3.5 mm Hg after unilateral RLN paralysis, respectively. This decrease indicated that the paralytic state lowered intraglottic pressure and therefore reduced hyperadduction.

It was interesting to note that, despite the preparalysis differences in intraglottic pressure, all three of the postparalysis intraglottic pressures were within 2.5 mm Hg. This implies that intraglottic pressure (and therefore subglottic pressure) was determined by the thyroarytenoid muscle with the least adductive force.

Effect of Recurrent Laryngeal Nerve Paralysis on Subglottic Pressure

Figure 5 graphically depicts subglottic pressure before and after a simulated unilateral RLN paralysis, in the presence of hyperadduction of the arytenoids. The comparisons were made during high levels of RLN stimulation (high intraglottic pressure), and two different levels of SLN stimulation. The subglottic pressure before unilateral RLN paralysis was very similar for the high and low levels, 85 and 79 mm Hg respectively, of SLN stimulation. The recurrent paralysis produced a decrease in subglottic pressure to 10 mm Hg and 14 mm Hg, respectively. In a fashion similar to the intraglottic pressure, paralysis caused a decrease in subglottic pressure to similar levels, which implied that subglottic pressure was also determined by the thyroarytenoid muscle with the least adductive force.

DISCUSSION

Although controversy remains as to the exact etiology and classification of adductor spastic dysphonia, one common mechanism involved in the pathophysiology of this disorder was hyperadduction of the true vocal cords.[20] From EMG studies, results of recurrent nerve section, and results of botulinum toxin injection into the vocalis, it might be surmised that stimulation of the thyroarytenoid muscle contributed to hyperadduction of the vocal cord, and resulted in high intraglottic pressures. Our results were in agreement with this conclusion. The role of the cricothyroid was less important in determining intraglottic pressure.

In this study, the relationship between the subglottic and intraglottic pressure during phonation indicated that, in the presence of hyperadducted arytenoids, the intraglottic pressure might play an important role in determining the subglottic pressure at a given flow rate. This study also suggested that thyroarytenoid contraction might be involved in controlling subglottic pressure by controlling intraglottic pressure. In comparison, cricothyroid contraction did not have a significant effect on subglottic pressure.

In normal phonation, in the absence of pulmonary disease, expiratory lung pressure has been found to be similar to subglottic pressure.[21] Sawashima and Kiyoshi[22] found that, at phonatory airflow rates of 100 cc/sec, the difference between expiratory lung pressure and subglottic pressure for normal subjects was 5 mm H_2O at most. The differences were less than 10 mm H_2O (= 1 cm H_2O or 0.74 mm Hg) at 200 cc/s and were greater than 10 mm H_2O at 400 cc/s.[22] During chest wall and diaphragmatic contraction, expiratory lung pressure and subglottic pressure rose to some

minimum value for phonation. This study indicated that this minimum subglottic pressure was determined primarily by the intraglottic pressure. In spastic dysphonia, there were bursts of thyroarytenoid and cricothyroid muscle activation resulting in periods of hyperadduction and shortening of the vocal folds. During these periods of hyperadduction, the intraglottic pressure might become so elevated that the expiratory lung pressure was unable to raise the subglottic pressure to a level sufficient to initiate phonation. This may explain why some patients with spastic dysphonia are able to sing or laugh in a normal way, but unable to speak normally at conversational levels.[23] The subglottic pressure for human phonation in normal conversation was maintained between 3 and 10 cm H_2O, but for singing it reached a peak of 50 to 70 cm H_2O at loudest intensities.[24] When the patient sang, the higher expiratory lung pressure might have overcome the periods of hyperadduction.

There are several limitations of the in vivo canine model of hyperadduction as a model for spastic dysphonia. Spastic dysphonia was thought to be a central nervous system (CNS) disorder resulting in episodes of vocal cord hyperadduction. Hyperadduction was simulated, in this canine model, by high levels of peripheral RLN electrical stimulation. The central nervous system component of this disorder was not a part of this model and, thus, we could not test for treatment effects directed at the CNS, such as certain muscle relaxants or psychotherapy. In addition, no attempt was made to duplicate the dynamic pattern of laryngeal sensory or motor nerve stimulation that might be experienced in spastic dysphonia. There could possibly be more moment-to-moment variation in laryngeal muscle contraction that was not contained in our model. Only one component of spastic dysphonia, vocal cord hyperadduction, was modeled and only the effects of treatments directed at the larynx, such as RLN section, could be tested.

Simulated hyperadduction from high levels of RLN stimulation resulted in high intraglottic and subglottic pressures in this canine model. After unilateral recurrent laryngeal nerve paralysis, intraglottic and subglottic pressures fell. Despite the large differences in the intraglottic pressures for different levels of RLN stimulation preparalysis, all intraglottic pressures decreased to similar values after unilateral RLN paralysis. This effect of unilateral RLN paralysis, in the presence of hyperadducted arytenoids, on intraglottic pressure indicated that the intraglottic pressure followed the thyroarytenoid muscle with the least adductive force despite the persistent difference in RLN stimulation to the opposite thyroarytenoid. This finding was consistent with the work of Shin, *et al.*, who found that unilateral RLN section significantly decreased peak glottic closing pressure during swallowing, but bilateral RLN paralysis did not significantly differ from the unilateral paralysis.[17]

A similar pattern was seen for subglottic pressure. This finding was expected from the earlier data which showed that subglottic pressure was largely determined by intraglottic pressure.

Our measurements were performed after bilateral arytenoid adduction to simulate hyperadduction of the arytenoids. This allowed us to isolate the role of the vocalis in adduction from the other muscles innervated by the recurrent laryngeal nerve. The individual role of the posterior cricoarytenoid muscle was probably small because it was mainly active during abduction.[25] The separate roles of the lateral cricoarytenoid and interarytenoid muscles were unknown but, as adductors of the cord, they should make up some component of the hyperadduction. It was clear from this study that hyperadduction of the arytenoids alone, in the absence of RLN, was not enough to raise intraglottic or subglottic pressure, even with increasing levels of SLN stimulation. The inferior pharyngeal constrictors might also play a role. Shin, *et al.* found that an inferior constrictor myotomy, after bilateral RLN section, significantly decreased peak glottic closing pressure compared to bilateral RLN paralysis alone.[17]

It was clear that paralysis of one thyroarytenoid muscle was sufficient to lower subglottic pressure; however, this resulted in asymmetric tension of the cords and a poor acoustic result. One theoretical advantage that treatment with botulinum toxin had over unilateral recurrent laryngeal nerve section was that treatment could be bilateral, which preserved the symmetric tension of the vocalis. BOTOX injection could also preserve the normal movement of the arytenoids. The disadvantage of BOTOX was that the injections had to be repeated and the long-term effects were unknown. It would have been optimal to produce bilateral permanent complete or partial vocalis paralysis, without paralyzing the other intrinsic laryngeal muscles. This would have improved hyperadduction, preserved symmetry of cord tension, and yet preserved true cord abduction and adduction. The in vivo canine model might help provide the answers to these questions.

CONCLUSIONS

The level of RLN stimulation and thyroarytenoid muscle contraction were the primary contributors to intraglottic pressure and hyperadduction. In the case of unilateral recurrent laryngeal nerve paralysis, the intraglottic pressure fell to the level of the thyroarytenoid muscle with the least adductive force. The level of SLN stimulation and cricothyroid contraction also played little role in determining intraglottic pressure. Subglottic pressure seemed to parallel intraglottic pressure, indicating that intraglottic pressure played a large role in determining subglottic pressure.

An in vivo canine model was useful in simulating the effects of laryngeal hyperadduction in spastic

dysphonia. This model was also useful in investigating and optimizing the results of laryngeal treatment modalities to relieve hyperadduction in disorders such as spastic dysphonia.

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