Physiologic Motion After Vocal Cord Reinnervation: A Preliminary Study

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This study attempted to reestablish physiologic vocal cord motion, rather than synkinesis, to a reinnervated vocal cord. One mongrel dog underwent a division and reanastomosis of the anterior branch of the right recurrent laryngeal nerve and simultaneous separation and reimplantation of a posterior division nerve-muscle pedicle into the posterior cricoarytenoid muscle. After 21 weeks, spontaneous physiologic vocal cord movement and electromyographic (EMG) activity were recorded during respiratory obstruction and laryngeal mechanical stimulation. Acoustic measures and histologic data are also presented from the reinnervated and normal vocalis muscle and from the recurrent laryngeal nerve. This study demonstrated that physiologic vocal cord motion can be achieved after laryngeal reinnervation using this technique.

INTRODUCTION

One major hindrance to the return of physiologic laryngeal function after reinnervation of a paralyzed vocal cord is synkinesis.[1] Laryngeal synkinesis results, after recurrent laryngeal nerve (RLN) injury, from the misdirected regeneration of axons to the intrinsic laryngeal muscles. This misdirected axonal regrowth results in the simultaneous contraction of all the reinnervated muscles during nerve stimulation. In the case of the vocal cord, simultaneous contraction of these muscles usually results in vocal cord adduction from the greater bulk of the adducting muscles.[2]

Despite the lack of physiologic motion seen in reinnervated vocal cords, ansa cervicalis nerve transfer has been used as a treatment for unilateral vocal cord paralysis.[3] In humans, the reinnervated cord does not exhibit physiologic motion but maintains its bulk, intrinsic stiffness, and tone. This may result in a superior voice when compared to other treatments such as Teflon® injection.

There are other situations in which physiologic vocal cord motion may be more critical such as in bilateral RLN paralysis or with laryngeal transplantation.[4] Bilateral recurrent vocal cord paralysis usually causes respiratory obstruction due to the loss of respiratory abduction. Several attempts at restoring physiologic vocal cord motion after paralysis have been made using the nerve-muscle pedicle or nerve transfer techniques.

Several investigators have achieved physiologic abduction using a variety of methods. Murakami and Kirchner[5] obtained physiologic abduction after RLN paralysis by sectioning the adductor branch after reinnervation by the RLN. Fex[6] also obtained physiologic abduction in 8 of 23 cats studied by anastomosing the phrenic nerve to the abductor branch of the RLN. Rice and Burstein[7] anastomosed the sternothyroid branch of the ansa cervicalis to the abductor branch of the RLN in 5 dogs after cutting the RLN lower in the neck. In the 4 surviving dogs, the reinnervated cord abducted 50% to 70% of the normal side during inspiration. This degree of abduction was abolished when the ansa was cut. Baldissera, *et al.*[8] studied bilateral vocal-cord paralysis in the cat by anastomosing a superior root of the right phrenic to the right RLN stump. This was followed by section of the adductor branch on the right to achieve abduction on inspiration in the right reinnervated cord. The cut adductor branch on the right was then anastomosed to the left abductor branch to achieve abduction on the left. They obtained bilateral vocal cord abduction on inspiration in 5 of 6 cats treated. However, no physiologic adduction was observed using this method.

Efforts to obtain physiologic abduction and adduction have also been made. Iwamura[9] unilaterally anastomosed a split vagus nerve to the adductor branch and the phrenic to the abductor branch of the RLN in 6 dogs. He obtained normal electromyographic and physiologic motion from the reinnervated side in 4 of the 6 animals and slight paradoxical vocal-cord adduction during inspiration in the remaining 2 animals. This paradoxical cord adduction was thought to be due to synkinetic growth of the vagus nerve resulting in the activation of the thyroarytenoid from fibers that used to innervate the posterior cricoarytenoid.

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Crumley[10] attempted to restore physiologic vocal-cord motion by anastomosing the phrenic to the abductor branch of the RLN and either the RLN, superior laryngeal nerve (SLN), or ansa cervicalis to the adductor branch. From this study, he concluded that the phrenic and ansa cervicalis were the best nerves for transfer. Marie, *et al.*[11] anastomosed the sternothyroid branch of the ansa cervicalis to the adductor branch of the RLN and the phrenic nerve to the abductor branch in 7 dogs. Functional reinnervation was achieved in only 1 dog, but the phonatory quality of the cord adduction was not studied. In a second dog, adduction of the cord was noted during obstructive dyspnea but not phonation.

The nerve-muscle pedicle technique, in addition to nerve transfer, has also been used to reinnervate the paralyzed vocal cord. Before the work of Meikle, *et al.*,[12] questions existed whether reinnervation actually occurred after nerve-muscle pedicle implantation. This study confirmed the presence of true muscle reinnervation using EMG activity, evoked EMG, direct nerve stimulation with muscle contraction, and horseradish peroxidase uptake. Tucker[13] reported appropriate abduction of the reinnervated cord in approximately 50% of patients with a bilateral vocal cord paralysis after omohyoid nerve-muscle pedicle implantation into the posterior cricoarytenoid muscle (PCA). In an attempt to improve on this percentage, Broniatowski, *et al.*[14] used an implanted laryngeal pacemaker in six dogs to directly stimulate an ansa cervicalis nerve-muscle pedicle which had been implanted into the PCA. All animals demonstrated marked abduction with inspiration. Maniglia, *et al.*[15] used a cricothyroid nerve-muscle pedicle implant into the PCA in dogs and found that this technique resulted in a greater degree of vocal-cord abduction when compared to direct nerve anastomosis. None of the dogs obtained physiologic cord adduction in that study.

A prior study in this lab of ansa cervicalis nerve transfer to the RLN, involving 13 dogs, demonstrated excellent reinnervation but a lack of physiologic cord motion due to synkinesis.[16] The goal of this initial study, using one dog, was to test the feasibility of reestablishing physiologic adduction and abduction after complete unilateral vocal paralysis by reanastomosis of the anterior branch to the anterior branch of the RLN and reimplantation of a PCA nerve-muscle pedicle into the PCA muscle. This was attempted with the expectation that, if laryngeal synkinesis could be avoided using this technique, a major obstacle to future laryngeal transplantation would be removed.

MATERIALS AND METHOD

Experimental Design

One mongrel dog (25 kg) underwent a complete division and immediate reanastomosis of the anterior branch of the right RLN and simultaneous separation and reimplantation of a posterior division nerve-muscle pedicle into the PCA muscle under general anesthesia. After 21 weeks, spontaneous vocal cord movement and EMG activity were recorded during respiratory obstruction and laryngeal mechanical stimulation. Measurements of vocal efficiency and acoustic analysis (jitter, shimmer, and signal-to-noise ratio) were made on the reinnervated animal with and without electrically stimulating the reinnervated RLN. Evoked electromyography was then performed to measure the nerve conduction velocity and the response amplitude. In addition, measurements of vocal efficiency and acoustic analysis (jitter, shimmer, and signal-to-noise ratio) were made on a group of eight control dogs during "normal" electrically stimulated phonation and a simulated RLN paralysis. Tissue samples were then taken of both vocalis muscles and recurrent laryngeal nerves in the reinnervated dog.

Surgical Reinnervation Technique

The animal to be reinnervated was premedicated with acepromazine and then underwent general anesthesia with endotracheal intubation. The ventral neck was shaved and prepared with Betadine.® The animal was placed supine on an operating room table, and a midline neck incision was made from the hyoid bone to 4 cm below the cricoid. The dissection was carried down to the larynx and trachea, retracting the strap muscles. Both recurrent and superior laryngeal nerves were identified. The attachments of the right thyrohyoid and the pharyngeal constrictor to the thyroid cartilage were separated, and the course of the right RLN was followed into the larynx. The anterior and posterior divisions of the RLN were identified and confirmed using a nerve stimualtor. The anterior division was cut sharply with a No. 15 blade and immediately reanastomosed with four 10-0 nylon sutures using standard microsurgical technique.

Due to the extremely small size of the posterior division of the RLN, a nerve-muscle pedicle was removed from the PCA and then reimplanted into a slit in the muscle using 10-0 nylon sutures. Prior to reanastomosing the anterior division and reimplanting the posterior division nerve-muscle pedicle, all attachments of the right RLN to the intrinsic laryngeal muscles were freed, and there was no evidence of intrinsic laryngeal muscle movement to right RLN electrical stimulation. The wound was closed in three layers with 3-0 Vicryl. At 21 weeks postoperatively, the laryngeal phonatory characteristics were evaluated using the in vivo canine model.[17]

In Vivo Canine Model

Each dog (reinnervated and controls) was premedicated with acepromazine 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 (Fig. 1). Both recurrent and 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 endo-

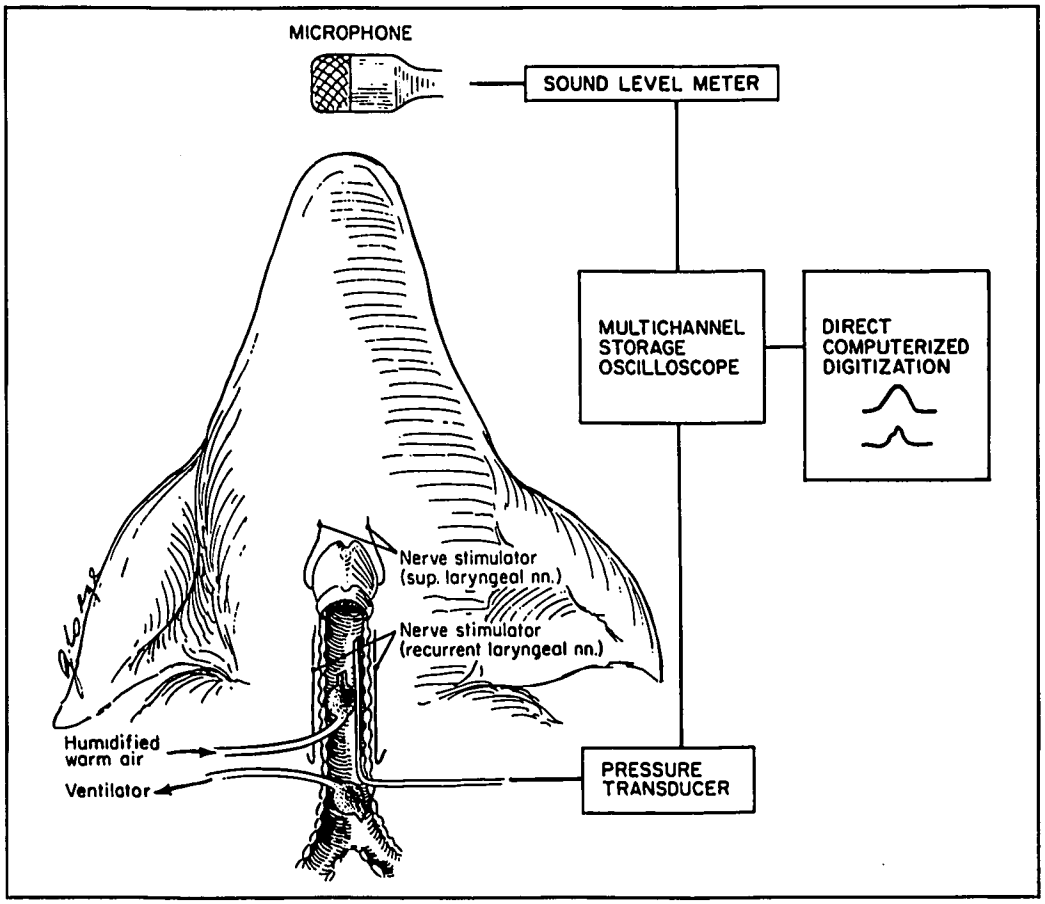

Fig. 1. Schematic representation of experimental setup for the in vivo canine model of phonation.

tracheal 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 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 each superior (external branch) and recurrent laryngeal nerve was isolated, and Harvard miniature electrodes were applied around each nerve. The electrodes were then insulated from surrounding tissue. Two constant-current nerve stimulators (WR Medical Electronics Co., St. Paul, Minn.: Model S2LH) were used to stimulate the recurrent and superior laryngeal nerves independently. These nerves were stimulated at a 70- to 80-Hz stimulus frequency with a 0.5- to 2.0-mA intensity for a 1.5-msec duration. Phonation was produced with an airflow of 318 to 523 cc/s applied through the larynx by the rostral endotracheal tube.

Acoustic Measures

Acoustic measures were obtained in the eight control animals during stimulation of all four laryngeal nerves to simulate normal phonation and with no stimulation of one RLN to simulate an RLN paralysis. Acoustic measures were made in the reinnervated animal both with and without stimulation of RLN on the right during stimulation of the other three laryngeal nerves. The sound-level measurements were made with a 1-inch Quest condenser microphone placed 30 cm from, and level with, the glottic outlet. The microphone was directed 90 degrees from the direction of the sound source. The sound-level measurements were made in decibels with a Quest sound-level meter 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 using a Millar Mikro-Tip catheter pressure transducer (Model No. 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. Due to the variation in subglottic pressure during phonation, the peak pressures attained during the glottic cycle were used. These peaks were identified using a commercially available software package for the PC system ("C-Speech," Paul Milenkovic, University of Wisconsin, Madison). 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 using the method of Koyama, *et al.*,[18] where total sound power = $2\,r^2P_e{}^2/P_oc$. This formula applies for a sound power radiating with no known directivity into a hemisphere of area ($2\,r^2$), a distance "r" away from the source. The product P_o (the density of the

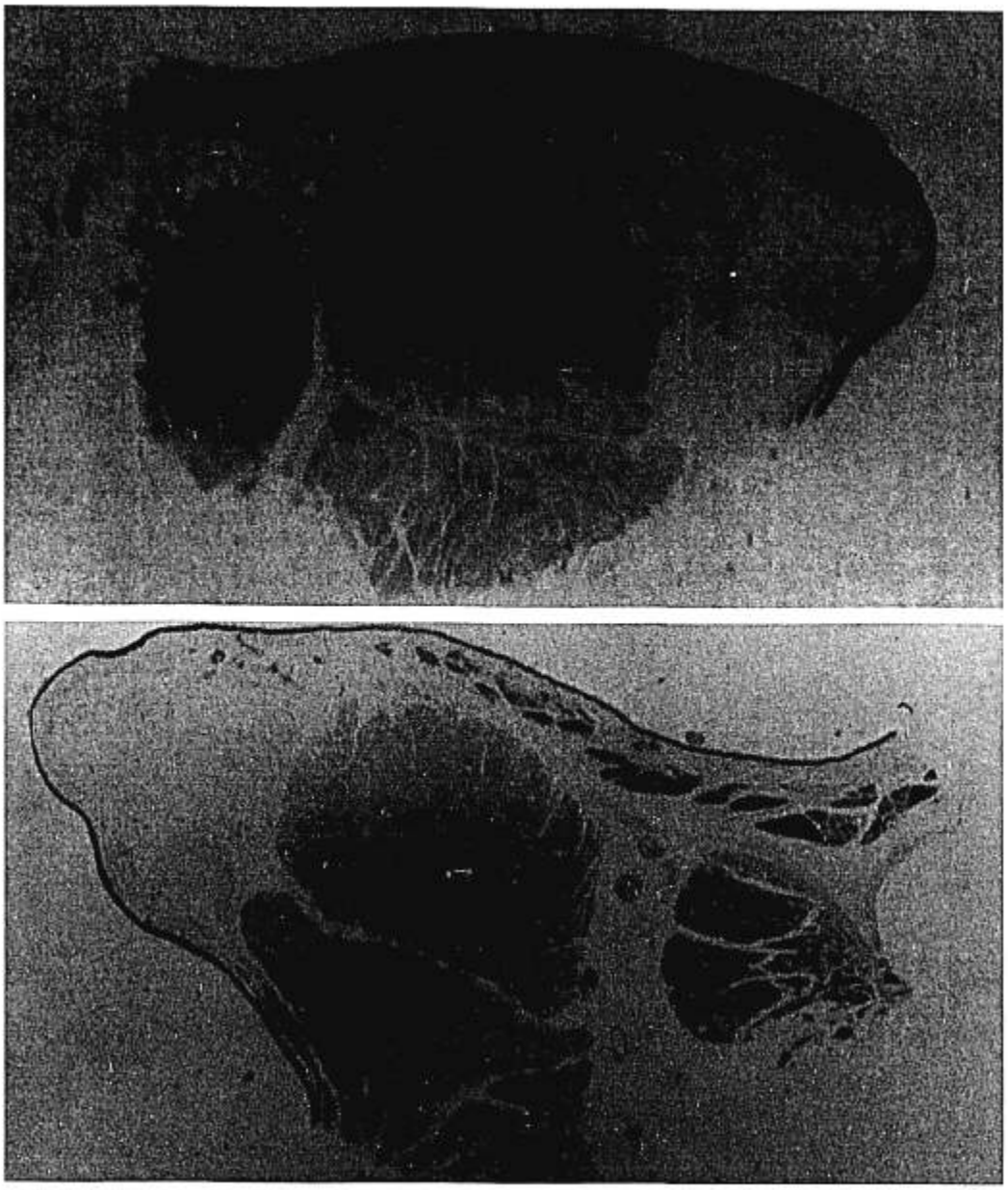

Fig. 2. **Top.** Cross section of normal cord. **Bottom.** Cross section of reinnervated cord (H&E, original magnification × 25).

medium) and c (the velocity of propagation) is the specific acoustic impedance of the medium which is 41.1 dyne s/cm^3 in air at 20°C. The term P_e is the root:mean-square (rms) sound pressure in dyne/cm^2 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 using a commercial software program, "C-Speech." Jitter, shimmer, and signal-to-noise ratio were calculated for each trial. The background noise in the laryngeal lab was 35 dB lower than the experimental values using the C-scale. To normalize for varying fundamental frequency, jitter was calculated as a fraction of the period of the fundamental frequency.

Videostroboscopy and Videolaryngoscopy

Videolaryngoscopy was performed during single-nerve stimulation of the RLN on the normal side or reinnervated side. The current stimulus was increased, starting from zero, to observe the effect of stimulation from each nerve separately. The image was detected by a Jed-Med CCD (charge-coupled device) video camera (model 70-5110) and a Sony U-matic videocassette recorder (VO-5850). Videolaryngoscopy was also performed under a lighter plane of anesthesia to detect the degree of spontaneous motion of the cords during laryngeal stimulation with a cotton swab and also to detect obstructive respiratory dyspnea.

Videostroboscopy was performed both with and without stimulation of the RLN on the right in the reinnervated animal, and with and without stimulation of one RLN in the normal control animals during stimulation of the other three laryngeal nerves. For stroboscopic imaging of the larynx, a Bruel & Kjaer laryngostrobe unit (model 4914) was used. The stroboscope was connected to a Storz 0-degree telescope via a fluid-filled light cable. The video images

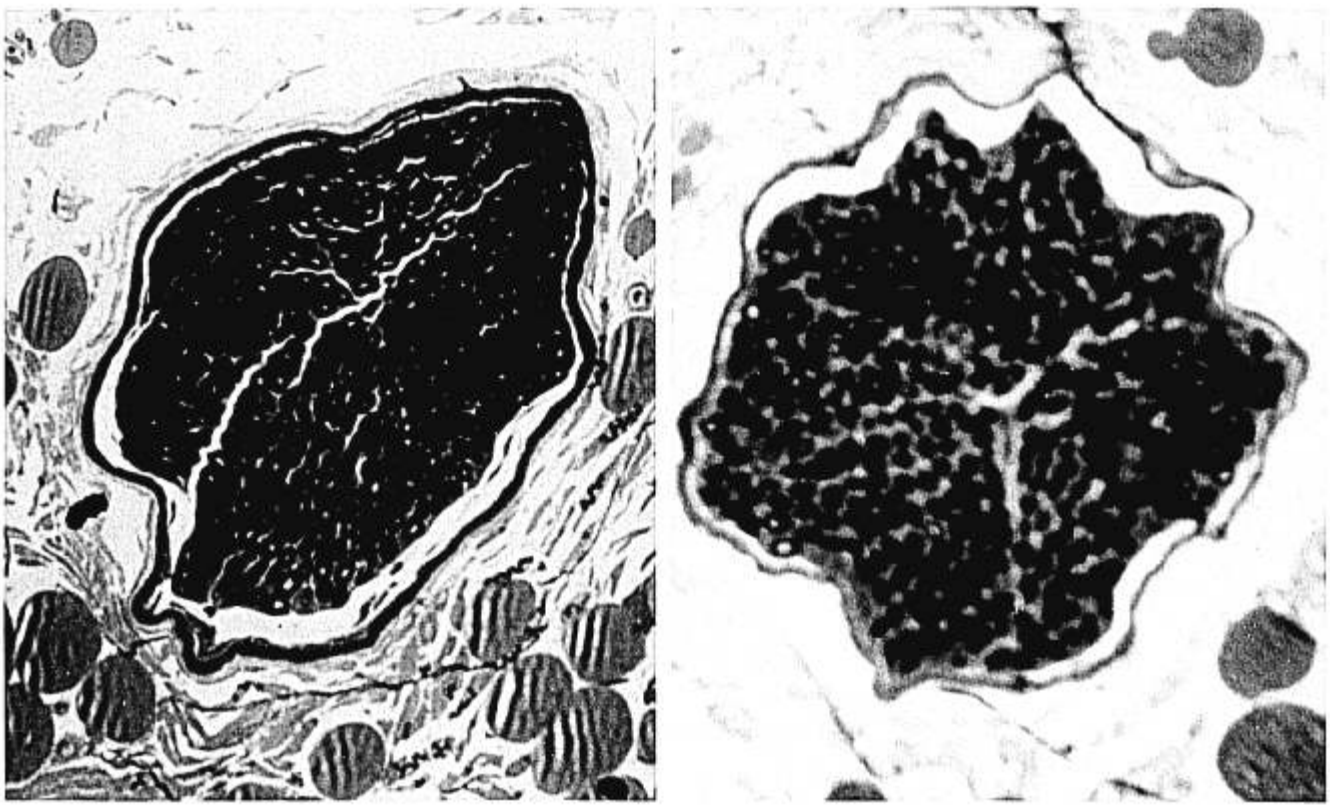

Fig. 3. **Left.** Cross section of recurrent laryngeal nerve (RLN). **Right.** Cross section of reinnervated RLN (osmium tetroxide and toluidine, original magnification × 25).

were analyzed frame by frame using the videorecording unit.

Evoked Electromyography

Conventional spontaneous EMG recordings were initially made during light anesthesia in order to detect fibrillation potentials in the right thyroarytenoid and PCA muscle. The response was detected with a bipolar needle placed transorally into the thyroarytenoid or PCA muscle. EMG recordings were then made in the PCA and thyroarytenoid muscle on the right and left during respiratory dyspnea and laryngeal mechanical stimulation.

Evoked electromyography was then performed in the reinnervated animal by stimulating each RLN individually with a single 0.5-msec pulse of variable voltage through a Harvard miniature electrode. The signal was digitized at 20 kHz and stored in a personal computer. The latency of the response was recorded in milliseconds at two points along the distal RLN and on either side. From these data, a conduction velocity for each nerve between the stimulated points was calculated. In addition, the amplitude of the evoked EMG was recorded in millivolts as a measure of the amount of depolarizing muscle.

Histologic Analysis

The larynx of the reinnervated animal was removed, divided into right and left halves, and placed in 10% formalin. A 3-mm thick cross section of each vocalis muscle was taken midway between the anterior commissure and the tip of the vocalis process for hematoxylin and eosin staining.

A 1-cm segment of the RLN was removed at a site 1 cm proximal to the cricothyroid joint bilaterally and fixed in 4% paraformaldehyde and 0.4% glutaraldehyde. The nerves were then stained with osmium, embedded in Epon/Araldite, and thin sections (0.5 μm) were cut transversely.

RESULTS

Nerve and Vocal Cord Histology

As shown in Figure 2, there was little sign of atrophy in the reinnervated thyroarytenoid muscle when compared to the normal side. Examination of the RLN on both sides revealed axons present in the reinnervated side, as seen in Figure 3.

Videolaryngoscopy of Spontaneous Movement and Single Nerve Stimulation

Under light anesthesia, with no external stimuli, there was no spontaneous movement of either the normal or reinnervated cord. Mechanical stimulation of the supraglottis with a cotton swab did not induce vocal cord adduction or spontaneous movement from either vocal cord. This may have been due to the level of anesthesia. When fully awake, the reinnervated animal had a very strong bark with no evidence of breathiness. Obstructive respiratory dyspnea resulted in abduction of both vocal cords. The degree of abduction was approximately 50% stronger on the normal side than on the reinnervated side. When fully

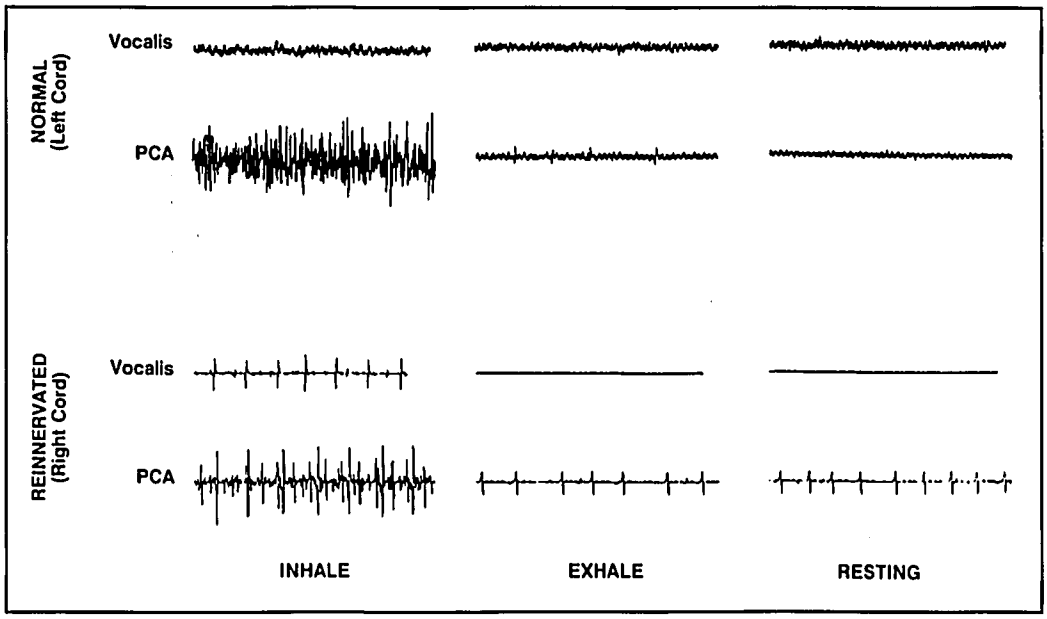

Fig. 4. Spontaneous electromyographic (EMG) activity from the posterior cricoarytenoid (PCA) and thyroarytenoid during the respiratory cycle.

awake, the reinnervated animal had no evidence of respiratory obstruction.

Electrical stimulation of the normal left RLN caused adduction of the left vocal cord with thyroarytenoid contraction at a threshold of 0.25 mA. Electrical stimulation of the right reinnervated RLN also caused adduction of the right vocal cord with thyroarytenoid contraction at a threshold of 0.05 mA.

Videostroboscopy

The results of videostroboscopy are summarized in Table I. Videostroboscopy of normal phonation in the control animals revealed similar glottal dynamics to phonation in the reinnervated animal during stimulation of all four laryngeal nerves, including the reinnervated RLN. There was complete glottic closure, two mass (upper and lower margin motion) motion of the mucosa on both cords, and complete glottic symmetry. Videostroboscopy, without stimulation of the right reinnervated RLN in the reinnervated animal, resulted in a picture similar to a simulated unilateral vocal cord paralysis. There was a mild posterior glottic-chink incompetence and one mass motion of the paralyzed cord. Without stimulation of the right RLN, the reinnervated cord stroboscopically appeared to track greater lateral excursions on opening than the normal cord, which remained primarily in the midline.

TABLE I.
Summary of Stroboscopic Analysis.

Animal Group	Glottic Closure	Mucosal Motion	Cord Motion
Control group (N = 8)			
Normal	Complete	Two mass bilaterally	Symmetric
Paralysis	Incomplete; posterior glottic leak	One mass; paralyzed cord	Asymmetric
Reinnervated animal (N = 1)			
Right RLN stimulated	Complete	Two mass; bilaterally	Symmetric
Right RLN not stimulated	Incomplete; posterior glottic leak	One mass; paralyzed cord	Asymmetric

Spontaneous and Evoked Electromyography

There were no spontaneous fibrillation potentials in either thyroarytenoid or PCA muscle. Mechanical stimulation of the supraglottis with a cotton swab did not induce any thyroarytenoid-muscle EMG activity from either vocal cord. As seen in Figure 4, obstructive respiratory dyspnea resulted in EMG evidence of PCA activation just prior to and during inspiration and cessation during expiration in both vocal cords. There was no thyroarytenoid EMG activity in either vocal cord throughout the respiratory cycle.

TABLE II.
Vocal Efficiency.

Dog	Normal	Paralysis
Control 1	3.0	0.098
Control 2	14	1.5
Control 3	2.8	0.055
Control 4	23	0.065
Control 5	11	0.2
Control 6	43	1.4
Control 7	70	0.065
Control 8	49	1.3
Mean	27	0.58
Reinnervated Animal 1	Right RLN stimulated (34.4)	Right RLN not stimulated (0.643)

All values are times 10^{-4}.

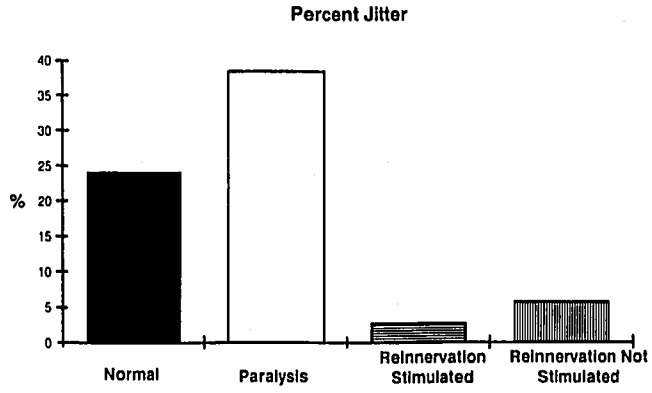

Fig. 5. Mean jitter for normal phonation and unilateral RLN paralysis in the control group (N = 8) and with and without stimulation of the right reinnervated RLN in the experimental animal (N = 1).

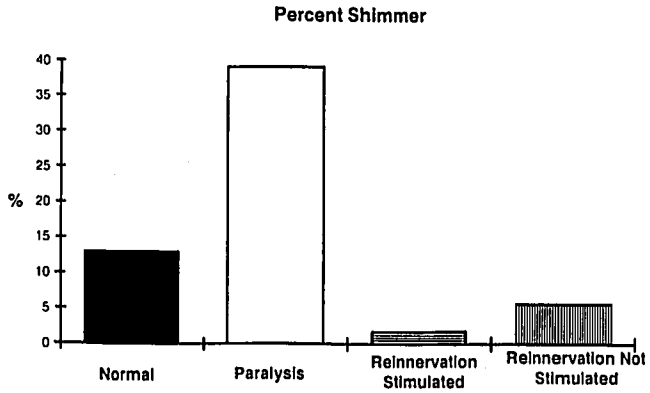

Fig. 6. Mean shimmer for normal phonation and unilateral RLN paralysis in the control group (N = 8) and with and without stimulation of the right reinnervated RLN in the experimental animal (N = 1).

The amplitude of the thyroarytenoid muscle response to evoked EMG was 10.0 mV on the reinnervated right side and 5.0 mV on the normal side. The amplitude of the PCA muscle response to evoked EMG was 2.8 mV on the reinnervated right side and 13.0 mV on the normal side. The latency of the thyroarytenoid-evoked EMG to stimuli at two separate stimulation points on the nerve was measured and from these data, a conduction velocity was calculated for the normal and reinnervated side. The conduction was 31 m/s for the normal side, and 44 m/s for the reinnervated side. The surface temperature of the nerve tissue was 31°C.

Vocal Efficiency

Table II shows the vocal efficiency of all eight control animals during normal phonation and simulated RLN paralysis. For the normal canine larynx, the vocal efficiency varied from 3×10^{-4} to 1×10^{-3} with a mean of 2.7×10^{-3}. For recurrent laryngeal paralysis, the vocal efficiency decreased in every case by at least a factor of 10. The values for paralysis varied from 5.5×10^{-6} to 1.3×10^{-4} with a mean of 5.8×10^{-5}. The vocal efficiency for normal phonation was significantly lower than that for unilateral RLN paralysis ($P<.05$).

Table II also shows the vocal efficiency for the reinnervated dog. With stimulation of the right reinnervated RLN during phonation, the vocal efficiency was 3.4×10^{-3}. Without stimulation of the reinnervated RLN, the vocal efficiency was 6.4×10^{-5}.

Acoustic Analysis

Figures 5, 6, and 7 demonstrate the mean jitter, shimmer, and signal-to-noise ratio, respectively, for normal phonation, unilateral RLN paralysis (control animals N = 8), and right reinnervated RLN with and without stimulation (reinnervated animal N = 1). The mean jitter for normal phonation was 24.1%; for paralysis, 38%; for reinnervation with stimulation, 2.61%; and without stimulation, 5.57%.

The mean shimmer was 13.0% for normal phonation, 39.1% for paralysis, 1.78% with reinnervated RLN stimulation, and 5.68% without stimulation.

The mean signal-to-noise ratio was 10.9 dB for normal phonation, 4.9 dB for paralysis, 28.05 dB with reinnervated RLN stimulation, and 15.15 dB without stimulation.

DISCUSSION

The isolated presence of vocal cord mobility on laryngoscopy or spontaneous EMG activity may not be sufficient evidence of reinnervation due to the action of the other intrinsic and extrinsic laryngeal muscles and the ability of neighboring nerves to reinnervate adjacent paralyzed muscles.[19] This study demonstrated right vocal cord reinnervation using the combination of (evoked and spontaneous EMG) observed motion from direct nerve stimulation, histologic evidence of preserved vocalis muscle bulk, and axonal regrowth in the RLN.

The amount of reinnervation that has occurred in a muscle can be estimated by the amplitude of depolarization on the evoked EMG. For the thyroarytenoid muscle, this study obtained the values of 10.0 mV on the reinnervated right side and 5.0 mV on the normal side. These values are in agreement with the studies of Steiss and Marshall[20] who obtained values of 1.2 to 26.0 mV in the normal canine. The higher value in the reinnervated thyroarytenoid muscle may have been due to synkinesis within this muscle. Intramuscular synkinesis may tend to simultaneously depolarize a greater muscle bulk than the normal side, which would depolarize the same muscle bulk but over a longer time frame. Although the absolute bulk of depolarized muscle, as calculated by the integral under the depolarization curve, may be the same on both the normal and reinnervated sides, the peak-to-peak amplitude for the reinnervated cord may be higher. The evoked EMG-response amplitude in the PCA muscle was 2.8 mV on the reinnervated right side and 13.0 mV on the normal side. This reduced bulk of PCA-

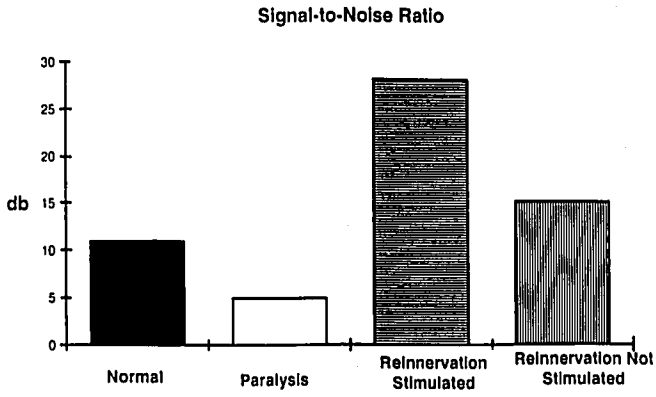

Fig. 7. Mean signal-to-noise ratio for normal phonation, unilateral RLN paralysis in the control group (N = 8), and with and without stimulation of the right reinnervated RLN in the experimental animal (N = 1).

muscle depolarization on the reinnervated side may account for the smaller degree of cord abduction on this side seen during respiratory dyspnea.

Successful laryngeal transplantation will require the solution of several problems. The foremost is prevention of laryngeal rejection and cancer recurrence by the host in the face of multiple contaminated surgical wounds.[21] Clinical experience with immunocompromised heart and liver transplantation patients at the UCLA Center for Health Sciences and elsewhere, has shown that contaminated pharyngeal wounds will heal adequately.[21] The second is reliable revascularization to the larynx via modern microsurgical techniques. The third, and perhaps most challenging, is the return of physiologic motion to the vocal cords.

One major obstacle to physiologic cord motion after reinnervation is synkinesis. Any nerve that is anastomosed to the distal RLN stump sends axons to all muscle groups previously innervated by the RLN. Stimulation of the anastomosed nerve after transfer to the RLN stump generally results in vocal cord adduction due to the 4:1 ratio of adductor to abductor muscles. One method of avoiding synkinesis is by selectively anastomosing the adductor and abductor branches of the RLN to the donor nerves. Several investigators have used this method with a variety of donor nerves in an attempt to reestablish physiologic motion. They have achieved only sporadic success.

This study has demonstrated the feasibility of obtaining physiologic motion of the paralyzed vocal cord by direct reanastomosis of the anterior-to-anterior branch of the RLN and reimplantation of a posterior branch of the PCA nerve-muscle pedicle. Respiratory dyspnea caused selective activation of the PCA on inspiration in the reinnervated cord by EMG without activation of the thyroarytenoid muscle. Although isolated spontaneous thyroarytenoid-muscle activation (without PCA stimulation) could not be demonstrated by mechanical laryngeal stimulation under light anesthesia, the reinnervated animal did exhibit a strong nonbreathy bark while awake. In addition, electrical stimulation of the reinnervated RLN did cause vocal cord adduction and thyroarytenoid muscle contraction with a vocal efficiency and acoustics similar to normal phonation. Using this method of reinnervation, it may be possible to achieve physiologic vocal cord motion after laryngeal transplantation. Not only was there adequate spontaneous abduction for an inspiratory airway, there was selective thyroarytenoid reinnervation for an adequate voice and glottic closure. The technique of intralaryngeal dissection of the RLN with reanastomosis of the anterior branch and nerve-muscle pedicle implantation of the PCA may avoid vocal cord synkinesis and solve one of the major problems of functional laryngeal transplantion.

CONCLUSION

Vocal cord synkinesis was avoided and physiologic motion was restored after reinnervation using a technique of intralaryngeal dissection of the RLN with reanastomosis of the anterior branch and nerve-muscle pedicle implantation of the PCA. The reinnervated vocal cord displayed isolated inspiratory PCA activation by EMG, with enough spontaneous abduction for an inspiratory airway. This technique also resulted in selective thyroarytenoid reinnervation by the RLN. The vocal efficiency and acoustics of the reinnervated animal during electrical stimulation of the reinnervated RLN were similar to normal electrically stimulated phonation.

BIBLIOGRAPHY

1. Crumley, R.L.: Laryngeal Synkinesis: Its Significance to the Laryngologist. *Ann Otol Rhinol Laryngol,* 97:87–91, 1988.
2. Gordon, J.H. and McCabe, B.F.: The Effect of Accurate Neurorrhaphy on Reinnervation and Return of Laryngeal Function. LARYNGOSCOPE, 78:236–250, 1968.
3. Crumley, R.L., Izdebski, K. and McMicken, B.: Nerve Transfer Versus Teflon® Injection for Vocal Cord Paralysis: A Comparison. LARYNGOSCOPE, 98:1200–1204, 1988.
4. Tucker, M.T.: Laryngeal Transplantation: Current Status. LARYNGOSCOPE, 84:787–796, 1974.
5. Murakami, Y. and Kirchner, J.A.: Vocal Cord Abduction by Regenerated Recurrent Laryngeal Nerve. *Arch Otolaryngol,* 94:64–68, 1971.
6. Fex, S.: Functioning Remobilization of Vocal Cords in Cats With Permanent Recurrent Laryngeal Nerve Paralysis. *Acta Otolaryngol (Stockh),* 69:294–301, 1970.
7. Rice, D. and Burstein, F.: Restoration of Physiologic Vocal Fold Abduction With the Ansa Cervicalis Nerve. *Arch Otolaryngol,* 109:480–481, 1983.
8. Baldissera, F., Cantarella, G., Marini, G., *et al.*: Recovery of Inspiratory Abduction of the Paralyzed Vocal Cords After Bilateral Reinnervation of the Cricoarytenoid Muscles by One Single Branch of the Phrenic Nerve. LARYNGOSCOPE, 99:1286–1292, 1989.
9. Iwamura, S.: Functioning Remobilization of the Paralyzed Vocal Cord in Dogs. *Arch Otolaryngol,* 100:122–129, 1973.
10. Crumley, R.: Selective Reinnervation of Vocal Cord Adductors in Unilateral Vocal Cord Paralysis. *Ann Otol Rhinol Laryngol,* 93:351–356, 1984.
11. Marie, J.P., Dehesdin, D., Ducastelle, T., *et al.*: Selective Reinnervation of the Abductor and Adductor Muscles of the Canine Larynx After Recurrent Laryngeal Nerve Paralysis.

Ann Otol Rhinol Laryngol, 98:530–536, 1989.
12. Meikle, D., Trachy, R. and Cummings, C.: Reinnervation of Skeletal Muscle: Comparison of Nerve Implantation With Neuromuscular Pedicle Transfer in an Animal Model. *Ann Otol Rhinol Laryngol,* 96:152–157, 1987.
13. Tucker, H.M.: Human Laryngeal Reinnervation: Long-Term Experience With the Nerve-Muscle Pedicle Technique. LARYNGOSCOPE, 88:598–604, 1978.
14. Broniatowski, M., Kaneko, S., Jacobs, G., *et al.*: Laryngeal Pacemaker. II. Electronic Pacing of Reinnervated Posterior Cricoarytenoid Muscles in the Canine. LARYNGOSCOPE, 95:1194–1198, 1985.
15. Maniglia, A., Dodds, B., Sorensen, K., *et al.*: Newer Technique of Laryngeal Reinnervation: Superior Laryngeal (Motor Branch) as a Driver of the Posterior Cricoarytenoid Muscle. *Ann Otol Rhinol Laryngol,* 98:907–909, 1989.
16. Green, D.C., Berke, G.S. and Graves, M.C.: A Functional Evaluation of Ansa Cervicalis Nerve Transfer for Unilateral Vocal Cord Paralysis: Future Directions for Laryngeal Reinnervation. *Otolaryngol Head Neck Surg,* 104:453–466, 1991.
17. Berke, G., Moore, D., Gerratt, B., *et al.*: The Effect of Recurrent Laryngeal Nerve Stimulation on Phonation in an In Vivo Canine Model. LARYNGOSCOPE, 99:977–982, 1989.
18. Koyama, T., Harvey, J.E. and Ogura, J.H.: Mechanics of Voice Production. III. Efficiency of Voice Production. LARYNGOSCOPE, 81:210–217, 1971.
19. van Harreveld, A.: Re-innervation of Denervated Muscle Fibers by Adjacent Functioning Motor Units. *Am J Physiol,* 144:477–493, 1945.
20. Steiss, J.E. and Marshall, A.E.: Electromyographic Evaluation of Conduction Time and Velocity of the Recurrent Laryngeal Nerve of Clinically Normal Dogs. *Am J Vet Res,* 49:1533–1536, 1988.
21. Bradford, C.R., Hoffman, H.T., Wolf, G.T., *et al.*: Squamous Carcinoma of the Head and Neck in Organ Transplantation Recipients: Possible Role of Oncogenic Viruses. LARYNGOSCOPE, 100:190–194, 1990.

Vestibular Rehabilitation Course Set in Ann Arbor

The University of Michigan Medical School is sponsoring a course, "Vestibular Rehabilitation," May 14–15, at the Towsley Center in Ann Arbor, Mich.

The seminar is intended to provide an important management tool for care of balance disorder patients by offering a broad introduction to the principles and practice of vestibular rehabilitation. Individual workshops for each member of the multidisciplinary teams of otologists, physical therapists and audiologists or vestibular testing specialists will be geared to specific needs.

The course is under the direction of Neil T. Shepard, PhD and Steven A. Telian, MD.

For more information, contact Angela Stewart, Towsley Center for Continuing Medical Education, Department of Post Graduate Medicine, University of Michigan Medical School, P. O. Box 1157, Ann Arbor, MI 48106-9869.

Research Study Club to Sponsor Conference

The Research Study Club of Los Angeles is sponsoring the 61st Annual Midwinter Otolaryngology Clinical Conference February 7–9. The conference will be held at the Moseley-Salvatori Conference Center, 637 So. Lucas, Los Angeles.

Thomas Calcaterra, MD, will be the program chairman. Guest faculty include Eugene B. Kern, MD; Wayne Larrabee Jr, MD; Harold Pillsbury III, MD; and James Y. Suen, MD.

For more information, contact Ms. Louise Ball, Executive Secretary, P. O. Box 1216, Murrieta, CA 92564; or phone (714) 677-4482.