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VIDEOSTROBOSCOPY OF HUMAN VOCAL FOLD PARALYSIS

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Previous stroboscopic studies of human vocal cord paralysis have been infrequent and have lacked documentation of the site of lesion. In order to study human laryngeal paralysis, the recurrent and superior laryngeal nerves were infiltrated unilaterally with lidocaine hydrochloride in three human volunteers. Vagal paralysis was simulated by combined (superior and recurrent) infiltration in one volunteer. Additionally, 20 patients with untreated laryngeal paralysis were studied from the voice laboratory at UCLA. In addition to videostroboscopic analysis, photoglottography and electroglottography were performed and synchronized with the stroboscopic images. The most significant finding in stroboscopy of the paralyzed larynx was the asymmetry of traveling wave motion. The traveling wave on the normal vocal fold had a faster wave velocity that created a phase difference in the vibration of the two folds. The wave also traversed a greater distance along the vocal fold mucosa on the normal side. No patient or volunteer with untreated laryngeal paralysis had a symmetric traveling wave, either in superior or recurrent laryngeal nerve paralysis. Synchronization with glottography indicated that the differentiated electroglottographic waveform provides useful information about the timing of glottic opening and closure in states of asymmetric laryngeal vibration. Implications for future studies and for the diagnosis of laryngeal paralysis are discussed.

KEY WORDS — laryngeal paralysis, larynx, mucosal wave, stroboscopy, voice disorders.

INTRODUCTION

Stroboscopy has provided valuable insights into laryngeal vibration and mucosal motion. However, few studies have systematically assessed the findings of stroboscopic analysis in laryngeal paralysis. Stroboscopy has a long history of clinical use in laryngology. Oertel is credited with the first clinical examination of the larynx with a stroboscope in 1878.[1] His primitive system employed a rotating perforated disc and laryngeal mirror to intermittently illuminate the glottis. Modern stroboscopes, while expensive, are considerably less cumbersome and have gained widespread use, particularly in Japan and Europe.[2]

Much of the behavior of the vibrating vocal folds is not visible through indirect laryngoscopy. Because an image on the human retina persists for approximately 0.2 seconds, the vibrating vocal folds appear as a blur along their medial edge. Stroboscopy creates the illusion of slow motion by generating light flashes at a rate slightly out of synchrony (approximately 2 Hz) with the fundamental frequency of phonation. This transforms the duration of a typical laryngeal cycle from 5 milliseconds to somewhere between 0.25 and 1 second. The "cycle" therefore represents a montage of many laryngeal cycles, rather than documentation of a single cycle as in high-speed photography.

Stroboscopic analysis was instrumental in the development of the cover-body theory of Hirano and Kakita,[3] which proposes that the stiff underlying body formed primarily by the vocalis muscle is responsible for the transverse movements of the vocal folds, while the looser mucosal cover vibrates primarily in the vertical dimension and forms a traveling mucosal wave.[3] As confirmed through supraglottic and subglottic videostroboscopy, the vocal folds vibrate as an upper and a lower margin during phonation. The lower margins separate first, forming a subglottic vault filled with a small volume of air, which is released as a puff into the vocal tract. The lower margins then return to the midline, and a gradual closing of the upper margin follows. In unilateral paralysis, there is a loss of stiffness of the underlying body, reducing the distinction between the upper and lower margins and creating a diminution or loss of the traveling wave.[4]

In addition to its research value, stroboscopy has been used for decades in clinical laryngology. Von Leden[5] emphasized the usefulness of stroboscopy in differentiating functional from anatomic laryngeal lesions, and for the early detection of invasive cancers of the vocal folds. However, stroboscopy is limited when aperiodicity, severe hoarseness, or breathiness impairs its ability to synchronize the flashes accurately.[2]

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Previous research supported the widespread view that laryngeal paralysis is accompanied by a loss of the mucosal traveling wave on the paralyzed fold.[2] Schoenharl[6] studied laryngeal paralysis with stroboscopy and found that in 55 of 62 cases the traveling wave was absent. The presence of the mucosal wave in patients with a paralyzed fold was interpreted as a sign of some degree of reinnervation, suggesting an improved prognosis for eventual complete recovery.[2,7] Fex[7] published a study in 1970 on the stroboscopy of laryngeal paralysis. He agreed with previous authors that in acute recurrent laryngeal nerve (RLN) paralysis the glottic wave is absent. The mucosal traveling wave was found to be an accurate reflection of thyroarytenoid muscle tonus, and its return indicated recovery of RLN function even when vocal fold abduction could not be detected.[7] The possibility that the return of the traveling wave was promoted by vocal cord fibrosis was not discussed.

Isshiki et al[8] published a detailed account of the effects of asymmetric vocal fold extension, using excised larynges and a computer model to assess the effect of unilateral changes of cricothyroid (CT) contraction and subsequent vocal fold stiffness. According to Isshiki et al, the vibratory findings produced by asymmetric laryngeal tension depend on the degree of glottic closure. They found that in simulated superior laryngeal nerve (SLN) paralysis the true vocal folds vibrated at the same frequency but out of phase, with the more tense fold (active CT) preceding the less tense one. This finding was later confirmed by Isshiki's group in a study performed on live dogs.[9]

Another study of canine laryngeal paralysis was performed by Moore et al[4] employing videostroboscopy. The authors found that canine RLN paralysis produces a difference in the timing of the onset of vocal fold lateral displacement and loss of the normal two-mass laryngeal vibration. The mucosal wave was markedly diminished but not lost in RLN paralysis.[4]

The morphological findings in laryngeal paralysis, including the Wagner-Grossman theory of vocal fold position in RLN and vagal paralysis, can be appreciated through indirect laryngoscopy alone and will not be the focus of this research.

The purpose of this study is to update earlier analyses of asymmetric laryngeal vibration by using videostroboscopy. First, it will describe the stroboscopic appearance of the larynx in paralysis. Previous reports of vibrational findings in laryngeal paralysis have been limited by the difficulty in defining the actual type of paralysis. Temporary induced paralysis was used in this study and provides a known site of lesion and the ability to compare laryngeal vibration before and after paralysis. Second, videostroboscopic images were synchronized with glottographic waveforms to more accurately assess the timing of glottic opening and closure on the glottographic waveforms. Third, an analysis of a clinical series of patients with various types of vocal fold paralysis was performed to verify that the findings noted in induced paralyses are similar to those in a typical laryngology practice. Finally, asymmetric vibration of the laryngeal mucosa provides an opportunity to utilize recently developed techniques for the objective analysis of videostroboscopic images for the degree of vocal fold symmetry.

MATERIALS AND METHODS

Method of Paralysis Induction. Three adult male volunteers with a mean age of 36 years were studied in this experiment. The volunteers appeared normal on laryngeal examination and had no history of laryngeal disorders. Vocal fold paralysis was induced by infiltrating 2% lidocaine hydrochloride with a 25-gauge needle into the expected location of the nerve. The left RLN was infiltrated in the tracheoesophageal groove approximately 2 cm inferior to the cricoid cartilage. The left SLN was infiltrated as described by Abelson and Tucker.[10] Ten cubic centimeters were necessary, with injection both at the posterior edge of the thyroid ala and inferiorly near its passage posterior to the thyrohyoid muscle. To create a combined ("vagal") paralysis, an SLN injection was performed, verified by stroboscopy, and immediately followed by an RLN injection. Subject 1 had SLN, RLN, and combined paralysis induced with lidocaine. Subject 2 had the RLN only injected, and subject 3 had the SLN only injected.

Videostroboscopic Analysis. The method of videostroboscopic analysis was modified from previous reports by Bless et al[1] and Kitzing.[2] Emphasis was placed in the analysis on the characteristics of the traveling mucosal wave. The onset, extent, and velocity of the traveling wave were analyzed and estimated by advancing the videotape frame by frame. Phase differences were noted in cases of asymmetric vibration. Other characteristics included the degree of glottic closure, the lateral excursion of the fold during vibration (termed amplitude by Bless et al[1]), and the regularity of glottic cycles, reflecting the presence or absence of severe frequency perturbation.

Stroboscopy. The experimental equipment required for these studies is depicted in Fig 1. Stroboscopy was performed with a Bruel & Kjaer 4914A (Orange, Calif) stroboscope. A microphone (Sennheiser MD

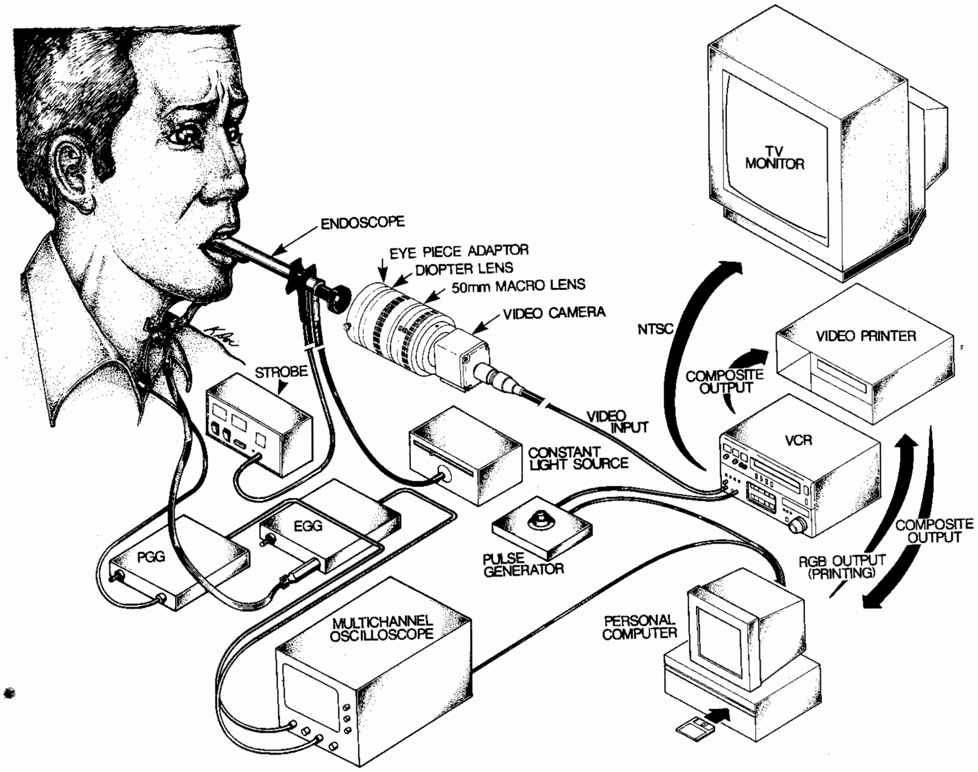

Fig 1. Experimental setup for recording of videostroboscopic images and concurrent glottographic waveforms. Computer includes Frame Grabber hardware, which allows digitization of videostroboscopic images, and Image Pro software for analysis of images.

4026) transduced the speech signal to allow synchronization with the video images. A Toshiba Charge Coupled Device (CCD) color video camera (model 1K-C30A, Buffalo Grove, Ill) imaged the glottis. The images were recorded onto a $^{3}/4$-in professional videocassette recorder (Sony VO-9850, Teaneck, NJ) equipped with a time coder. Still images from the videotape were made with a color video printer (Sony Mavigraph, Teaneck, NJ).

Glottography. Videostroboscopy and glottography were performed simultaneously during phonation of the vowel /i/ produced at a constant pitch and loudness. Transduction of photoglottography (PGG) waveforms required constant illumination of the larynx with a miniature light source (Karl Storz 481-C, Culver City, Calif) in addition to the stroboscopic light source. A 90° telescope (Wolfe, Rosemont, Ill) with two light inputs was used to visualize the glottis. Light transmission for PGG waveforms was transduced with a photosensor (Centronics OSD 50-2, Mountainside, NJ) held in position on the skin overlying the CT membrane. Electroglottography (EGG) recording electrodes were strapped into position onto the subject's neck, overlying the thyroid lamina at the level of the true vocal folds. A Synchrovoice electroglottograph (Harrison, NJ) recorded the EGG waveforms.

Synchronization. Videostroboscopic images were correlated with glottographic signals to provide preliminary information about the timing of events in laryngeal paralysis. Further details about the synchronization method are being published in a separate report from this laboratory.[11] Briefly, a 5-millisecond square wave pulse was digitized and simultaneously recorded on the audio channel by the videotape recorder. By correlating the 5-millisecond square wave pulse with the vertical synchronization trace of the video signal, the position on the glottographic waveform could be precisely correlated with individual video images. A Hitachi oscilloscope (model V-1050 F, Torrance, Calif) was used to extract the vertical trace of the video signal for recording.

Electroglottography, photoglottography, synchronizing pulse and vertical trace of the video signal were digitized via a Labmaster 12-bit analog-to-digital board housed in an IBM compatible computer. The EGG and PGG signals were verified on a Tektronix 5116 (Beaverton, Ore) storage oscilloscope prior to recording. The waveforms were analyzed by using a commercially available software package for the PC system (C-Speech, Paul Milenkovic, University of Wisconsin, Madison, Wis).

Videostroboscopic Image Evaluation. Images were analyzed by using an image processing software package previously described[12] (Image Pro II, Media Cybernetics, Silver Spring, Md). The hardware necessary for image analysis included a Frame Grabber to digitize and analyze video images (Data Transla-

TABLE 1. SUMMARY OF STROBOSCOPIC EVALUATION OF LIDOCAINE-INDUCED LARYNGEAL PARALYSIS

Subject	*Site*	*Regularity**	*Closure*	*Symmetry of Vibration and Characteristics of Mucosal Wave*
1	SLN	Regular	Complete	Moderate asymmetry of TW; greater velocity and excursion of TW on normal side, particularly at low frequency of vibration; decreased tension of TVF on injected side; occasional shifting of glottis from side to side during vibration
1	SLN + RLN	Variable aperiodicity	Small posterior glottic chink	TVFs vibrated at different level during portions of cycle (Fig 3C); profound asymmetry of TW (Fig 3); normal TVF crossed midline during vibration; TW velocity and excursion greater on normal side; loss of TVF tension; TVF higher on normal side
1	RLN	Regular	Variable	Marked diminution of TW excursion and velocity on side of injection; greater TVF excursion on normal side (see symmetry ratio, Fig 4A)
1	RLN paresis	Regular	Complete	Asymmetry of vibration with greater excursion and velocity of TW on normal side; normal TVF adduction and abduction
2	RLN	Variable aperiodicity	Large chink	Marked asymmetry of TW (Fig 2B); greater velocity of TW on normal side; vibration out of phase; normal TVF precedes paralyzed TVF
3	SLN	Regular	Complete	Diminished mucosal wave on side of paralysis; flaccidity of TVF on injected side

SLN — superior laryngeal nerve, RLN — recurrent laryngeal nerve, TW — traveling wave, TVF — true vocal fold.

*Regularity indicates ability of subject to maintain constant frequency and therefore facilitate stroboscopy. High jitter (frequency perturbation) results in irregular vibration and difficulty in analyzing glottic wave.

tion, DT-2853 60SQ, Marlboro, Mass). The desired portion of the video image can be outlined by use of a pointing device, and then the area of the trace or the length of a line is calculated by and expressed in pixel units by the software.

Images were digitized from the most closed and most open portions of the vocal cycle, and the width of the vocal folds was measured during these intervals. A measure was then computed of the symmetry ratio, which expresses the excursion of one vocal fold in proportion to that of the other fold. Symmetric vibratory movement is marked by an equal excursion of both vocal folds from the midline during phonation. When the lateral motion of one fold is markedly reduced, as in unilateral RLN paralysis, the symmetry ratio approaches zero. Further details regarding the objective analysis of videostroboscopic images have been recently published by Sercarz et al.[12]

Chronic Paralysis Study. We reviewed the videotapes and clinical files of all patients with a diagnosis of vocal fold paralysis evaluated in the UCLA Voice Laboratory during the years 1988 to 1991. Patients were excluded if the paralyses had been previously treated or if inadequate videotape or clinical information was available. All patients underwent stroboscopy and glottographic (PGG and EGG) recordings. In each case, medical records were analyzed to determine the probable site of the paralysis. Three cases of vagal paralysis had documented sites of lesion. Two patients had laryngeal paralysis following penetrating trauma to the skull base and subsequent high vagal paralysis. The third had undergone a tumor with sacrifice of cranial nerve X at the skull base.

Two clinical cases were diagnosed as SLN paralysis. One patient developed a dysphonia following thyroid surgery marked by a reduced ability to modulate pitch and vocal fatigue. Later exploration revealed a lack of function of the SLN on direct nerve stimulation. Another patient suffered neck trauma and developed a dysphonia marked by abnormal laryngeal vibration and normal vocal fold abduction and adduction. In the latter patient, there was a rotation of the posterior commissure in the direction of the paralysis side.

Fifteen additional patients received diagnoses of unilateral RLN paralysis based on history and indirect laryngeal examination.

RESULTS

Induced Paralysis. The three subjects underwent stroboscopy prior to induction of paralysis. In each case, vibration was normal with equivalent velocity and excursion of the traveling wave and symmetric tension of the vocal folds bilaterally. Closure was complete in each case.

In each case of SLN paralysis, the volunteer reported anesthesia of the larynx and an inability to sing at a high pitch in addition to the findings described.

Table 1 summarizes the videostroboscopic findings in induced vocal fold paralysis. Following RLN injection, the vocal process on the paralyzed side remained in the paramedian position. There was no rotation of the posterior glottis that was appreciable in any of the induced paralysis states.

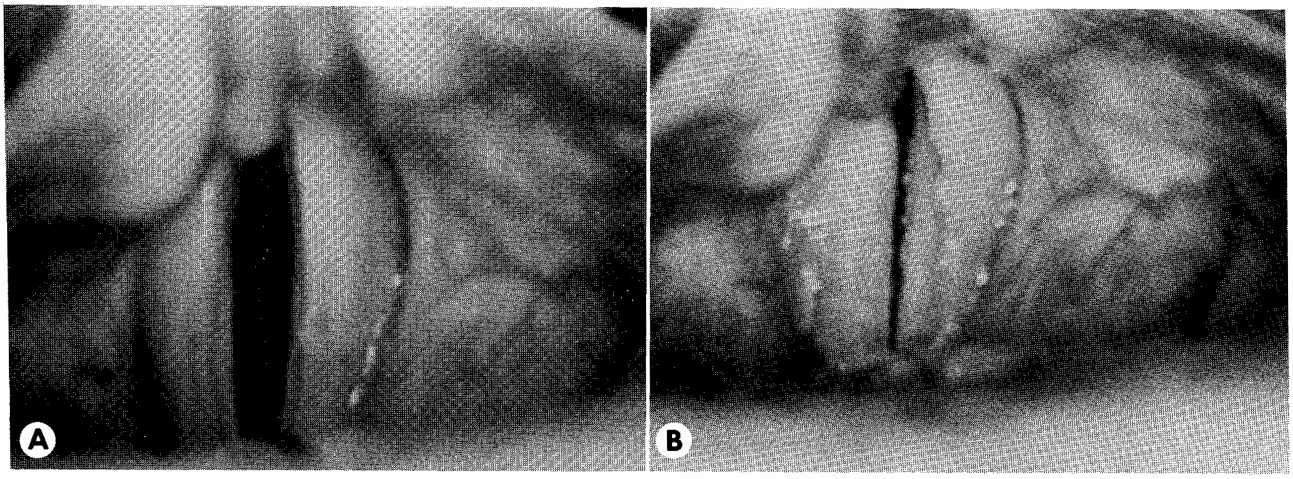

Fig 2. (Subject 2) A) Normal phonation, showing symmetric traveling mucosal wave. B) Following left recurrent laryngeal nerve injection. Right mucosal wave has completed its vibration. Wave of lesser excursion is now traveling along left (paralyzed) vocal fold.

The most consistent finding in each case of induced paralysis was a diminution of traveling wave velocity and less lateral excursion on the side of the injection, whether SLN, RLN, or combined. Although the traveling wave was attenuated in each case on the side of the paralysis, the wave was observed during each trial. For subject 1, who underwent all three types of paralysis, the traveling wave asymmetry was greatest in the vagal paralysis, followed by RLN and SLN in that order.

Figure 2A is normal phonation from subject 2 with a symmetric traveling wave. Figure 2B, following left RLN injection, demonstrates asymmetric vibra-

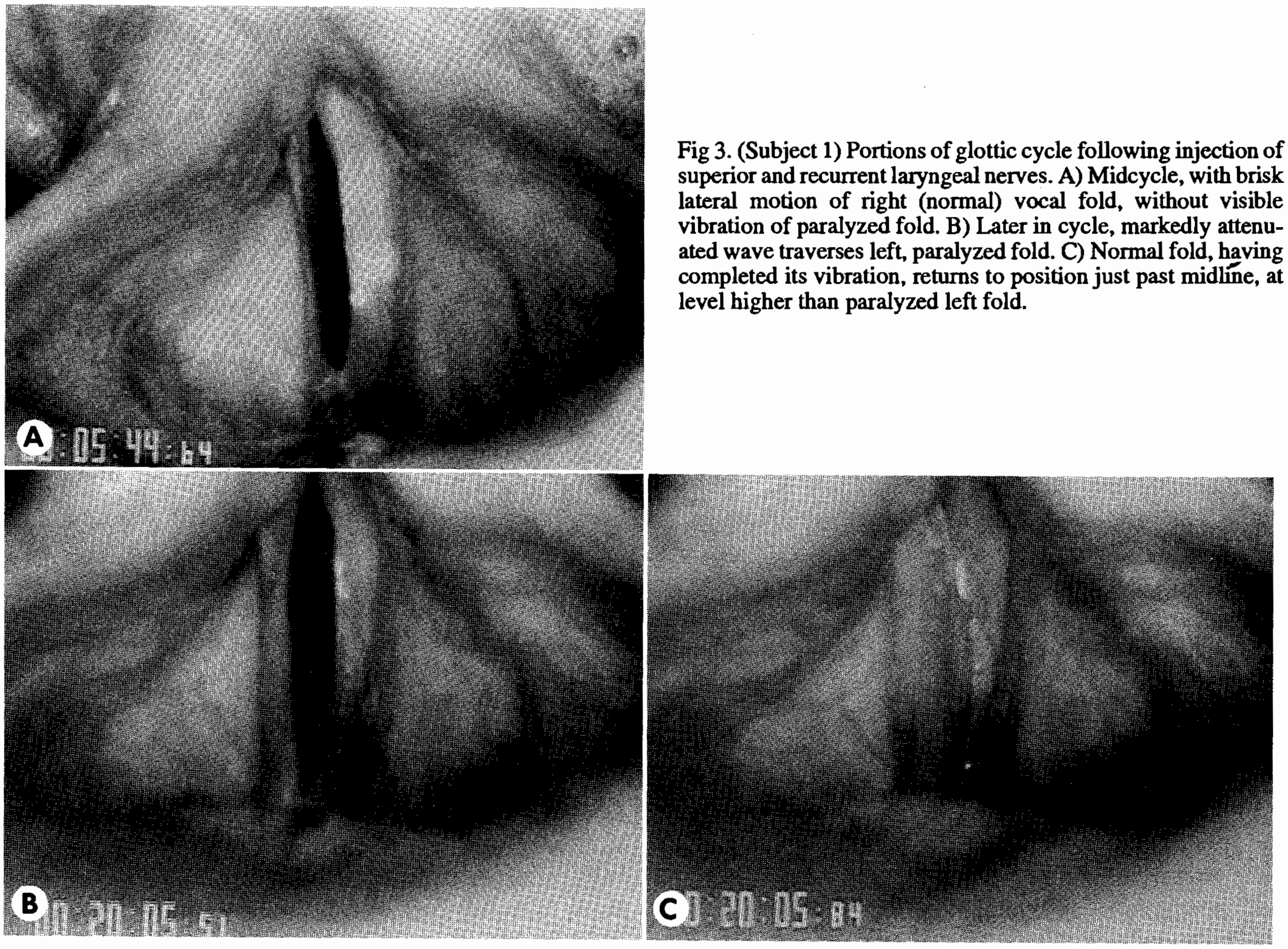

Fig 3. (Subject 1) Portions of glottic cycle following injection of superior and recurrent laryngeal nerves. A) Midcycle, with brisk lateral motion of right (normal) vocal fold, without visible vibration of paralyzed fold. B) Later in cycle, markedly attenuated wave traverses left, paralyzed fold. C) Normal fold, having completed its vibration, returns to position just past midline, at level higher than paralyzed left fold.

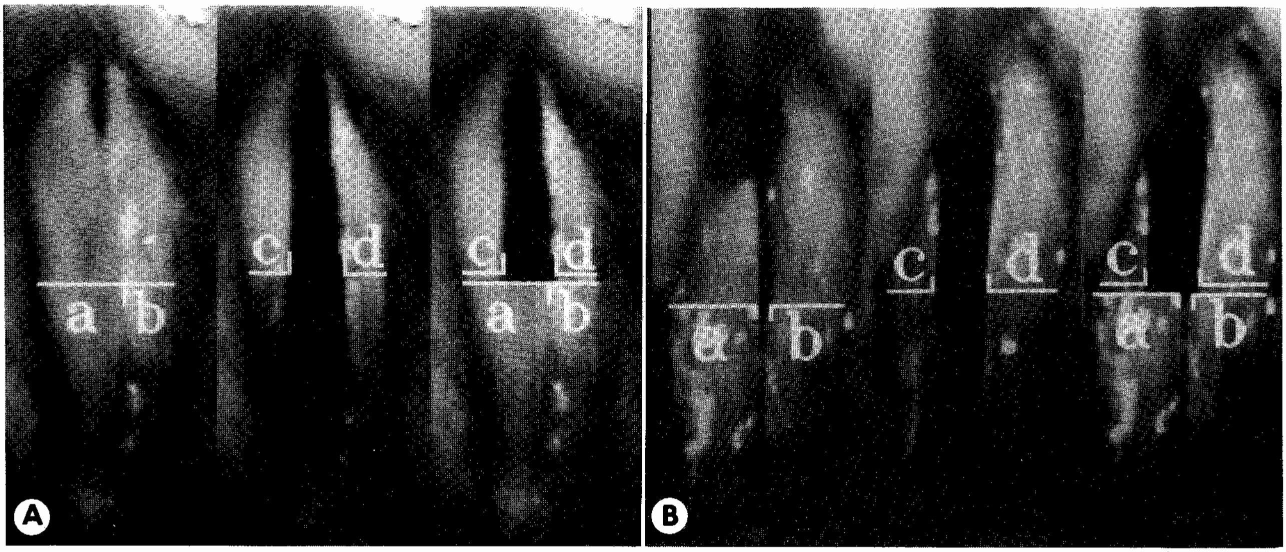

Fig 4. Symmetry ratio for two volunteers with induced recurrent laryngeal nerve paralysis (see text). A) Subject 1. B) Subject 2.

tion. The normal (right) vocal fold has completed its vibration, and the paralyzed (left) fold is shown with an attenuated traveling wave. Figure 3 is a series of still video frames from subject 1 with a left combined RLN and SLN paralysis. Figure 3A is early in the glottic cycle, with a vigorous excursion of the uninjected (right) vocal fold. Later in the glottic cycle, a minimal mucosal wave is seen on the left vocal fold (Fig 3B). Finally, the right vocal fold, positioned at a higher level than the paralyzed left vocal fold, crosses the midline, and the glottis closes (Fig 3C).

Figure 4 depicts measurement of the symmetry ratio for subjects 1 and 2 following RLN injection. The lengths a and b (most closed portion of the glottal cycle) and c and d (most open) represent the width of the right and left vocal folds. The calculated symmetry ratio for subject 1 was 0.139 (Fig 4A) and for subject 2 was 0.151 (Fig 4B). In a perfectly symmetric case, the ratio is 1.0, and in a case with no lateral vocal fold movement in one of the folds the ratio is 0.

Figure 5 demonstrates the results of synchronizing the glottographic signal to the videostroboscopic image from subject 1 following RLN injection. Figure 5A is a stroboscopic image taken immediately following opening of the glottis. Figure 5B is the synchronized glottography. There is an upward deflection in the differentiated EGG (dEGG) waveform that correlates well with the onset of vocal fold opening. Figure 5C,D is synchronized from a point at midcycle. Figure 5E,F is from an image immediately preceding the point of glottic closure. The downward deflection in the dEGG waveform closely correlated with the instant of laryngeal closure. Similar findings are documented in Fig 6, which shows the results of synchronizing strobe images and glottography in subject 1 with SLN paralysis at the moment of opening (Fig 6A,B) and closure (Fig 6C,D). Again, the timing of opening and closure of the vocal folds is predicted by the dEGG waveform.

Clinical Cases. The results of a series of 20 consecutive patients analyzed at the UCLA Voice Laboratory will now be described. Of the 15 RLN patients, 3 could not be included because aperiodic vibration or a large glottic gap prevented analysis of the mucosal traveling wave. The results of the 12 remaining patients with unilateral RLN paralysis and adequate stroboscopy are summarized in Table 2. Ten of 12 patients had a mucosal wave present on the paralysis side. The wave asymmetry was similar to that observed in the induced paralysis: the normal wave had a greater velocity and traveled farther along the vocal fold mucosa. There was a phase shift, with the normal side vibrating sooner than the side of the paralysis. The asymmetry was marked in the majority of patients with unilateral RLN paralysis.

The findings in the two patients with SLN paralysis parallel those in the group with induced paralysis. There was normal abduction and adduction of the vocal folds in both cases. Glottic closure and regularity (lack of noticeable frequency perturbation) were normal. Analysis of the mucosal wave revealed mild to moderate asymmetry of the traveling wave. On the normal (nonparalysis) side, the wave appeared earlier, had a greater velocity, and traveled farther along the surface of the vocal cord mucosa.

The mucosal wave findings in vagal paralysis are similar to those in RLN paralysis. Only one of the three patients had the frequently cited finding of rotation of the posterior glottis in the direction of the

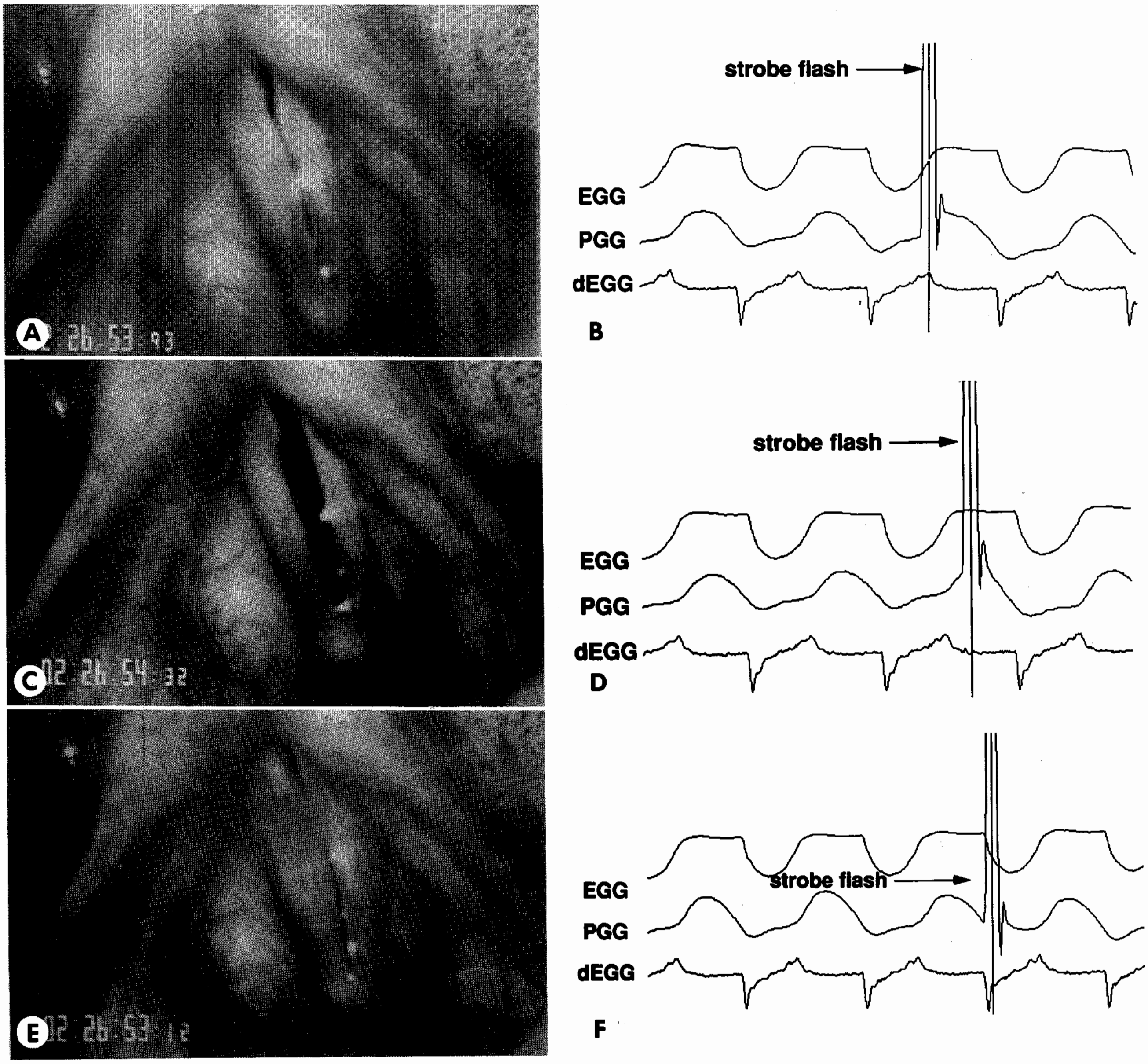

Fig 5. (Subject 1) Correlation of glottographic information with stroboscopic images in recurrent laryngeal nerve paralysis. EGG — electroglottography, PGG — photoglottography, dEGG — differentiated EGG. A) Immediately following vocal fold opening. B) Corresponding glottographic waveforms, with strobe flash occurring just after positive deflection in dEGG waveform. C) Later in cycle, at mid-opening. D) Glottographic waveforms, with strobe occurring near peak of PGG waveform. E) Image of glottis at point of closure. F) Corresponding waveforms; nadir of dEGG waveform corresponds to strobe flash.

paralysis. In one of the patients with vagal paralysis, the glottic gap was wide throughout the cycle and there was very irregular vibration of the mucosal wave bilaterally. The other two patients had marked asymmetry of the traveling wave, vibration out of phase, and differences in the velocity and excursion of the traveling wave similar to those found with induced vagal paralysis in this study.

DISCUSSION

There are several advantages of studying induced vocal fold paralysis stroboscopically. Temporary chemical paralysis allows comparison with normal stroboscopy in the same individual. It also provides an opportunity to study recovering RLN weakness and observe the gradual recovery of symmetry, as in the study of vocal fold paresis in subject 1. Before discussing the stroboscopic findings in particular paralytic states, we will review the overall relationship between tension of the vocal fold and vibratory characteristics.

Unlike previous studies[2,6,7,13] of stroboscopy in vocal cord paralysis, the data in the present report indicate that the mucosal wave is always *affected* but

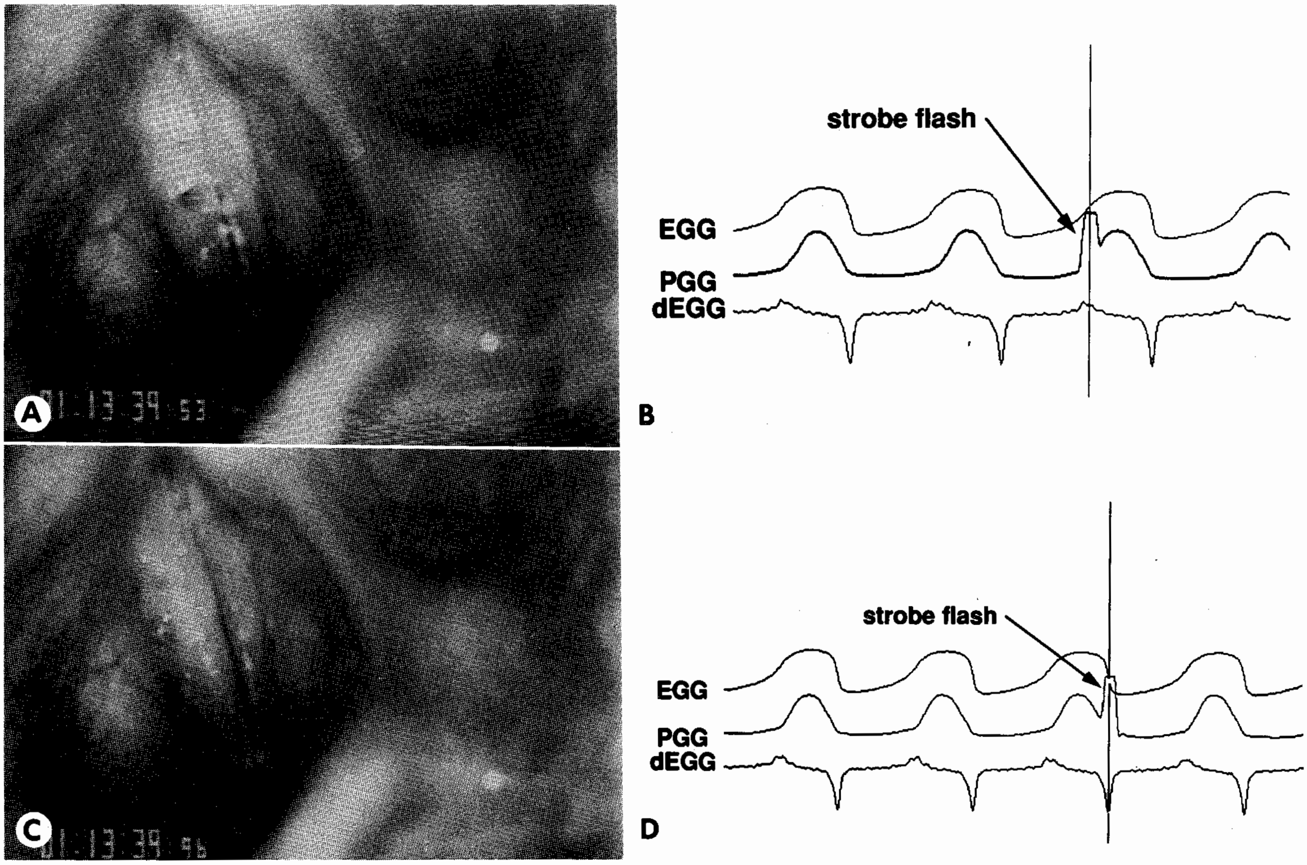

Fig 6. (Subject 1) Correlation of glottographic information with stroboscopic images in superior laryngeal nerve paralysis. Abbreviations as in Fig 5. A) Immediately following vocal fold opening. B) Corresponding glottographic waveforms, with strobe flash occurring just after positive deflection in dEGG waveform. C) At point of closure of glottis. D) Corresponding glottographic waveforms, with strobe again occurring at nadir of dEGG waveform.

not invariably *absent* in RLN and vagal paralysis. All patients with laryngeal paralysis of any type, experimental or clinical, had an unambiguous asymmetry of laryngeal vibration demonstrated stroboscopically. Furthermore, the data presented indicate that the asymmetry follows a pattern: the normal vocal fold traveling wave has a greater velocity than that of the paralyzed fold, is observed earlier in the glottic cycle, and traverses farther over the surface of the vocal fold mucosa. The symmetry ratio data were presented to document the greater excursion of the vocal fold margin on the nonparalyzed side during the most open portion of the cycle in unilateral RLN paralysis.

The findings described here are similar to those reported by Tanabe et al[9] in a report on asymmetric glottic vibration studied in canine larynges. It is not surprising that there was a visible difference in the velocity of the traveling waves on two vocal folds. Basic research on the propagation of waves in elastic media have indicated that there is a direct relationship between the stiffness of the material or reactance to deformation and the velocity of a harmonic traveling wave.[14] The lack of thyroarytenoid stiffness and/or CT contraction is probably the central cause of the vibratory differences in laryngeal paralysis. In a morphologic study of the vocal fold as a vibrator, Hirano[15] stated that the CT and thyroarytenoid muscles had the greatest effect on the stiffness relationship between the body and the cover of the vocal fold; this finding may explain why either muscle may elicit similar changes in the traveling wave velocity and excursion.

For the two patients with absent glottic waves, each had poor glottic closure that reduced the degree of vocal fold contact. This tended to decrease the ability of stroboscopy to detect the very subtle mucosal wave that generally occurs on the paralyzed vocal fold.

Because of its sensitivity in demonstrating slight differences in vocal fold vibration, stroboscopy is particularly useful in the study of laryngeal paralysis. In subtle cases, such as RLN paresis or isolated SLN paralysis, an abnormality of traveling wave motion may be the easiest finding to elicit. The results of this study suggest that traveling wave asymmetry may be one of the most salient observations to make in suspected paralysis, because traveling wave asymmetry

TABLE 2. RESULTS OF PATIENTS WITH RECURRENT LARYNGEAL NERVE PARALYSIS

	No. of Patients
Glottic closure	
Complete	2
Mild to moderate incomplete closure	5
Severely incomplete closure	5
Glottic wave: extent of wave excursion along vocal fold mucosa	
Symmetric	0
Mild to moderate asymmetry	2
Marked asymmetry	8
Absent on paralysis side	2
Glottic wave: estimated speed of glottic wave	
Symmetric	0
Greater velocity (earlier wave) on normal side	10
Greater velocity on paralyzed side	0
Absent wave on paralyzed side	2
Vocal fold lateral displacement during vibration*	
Equivalent	0
Mild to moderate asymmetry	4
Marked asymmetry	8

Of 15 patients with clinical diagnosis of recurrent laryngeal nerve paralysis, 12 had adequate regularity of vibration for suitable stroboscopy. Their results are presented here.

*Lateral motion during vibration was invariably greater on side opposite paralysis (normal side). See Fig 4, showing symmetry ratio in induced paralysis.

is a consistent finding in untreated laryngeal paralysis. In subject 1, traveling wave asymmetry was also identified in RLN paresis.

Despite this consistency, there is a significant variability of findings among different patients and even within the same individual phonating at two different fundamental frequencies. For example, in RLN paralysis, CT muscle contraction at high pitch adds vocal fold stiffness and tends to reduce the degree of asymmetry. One patient in the study developed RLN paralysis following thyroid surgery 20 years before the examination; the paralysis is still largely uncompensated. He apparently compensates with CT tension, producing speech at a high pitch level (paralytic falsetto) that provides glottic closure and only moderately asymmetric traveling wave motion.

Both RLN paralysis and combined SLN and RLN paralysis produced the above changes in the traveling wave. Although the combined paralysis was characterized by a greater degree of asymmetry and a more flaccid or "wavy" paralyzed cord, it is doubtful from our data whether stroboscopy alone can be of significant assistance in differentiating vagal from RLN paralysis.

Isolated SLN paralysis is a rarely noted clinical entity that probably often goes undetected.[10,16,17] The SLN provides sensation to the supraglottic larynx through its internal branch and motor fibers to the CT muscle via its external branch. The motor branch is in close proximity to the superior thyroid vessels and is therefore vulnerable to injury during thyroid surgery. According to Ward et al[17] and other authors,[10] paralysis of the CT muscle causes 1) lack of longitudinal tension of the true vocal folds, 2) a tilt of the larynx because of the downward motion of the contralateral intact CT muscle, and 3) a rotation of the posterior glottis toward the side of the paralysis because of the unopposed CT muscle's pulling the anterior thyroid cartilage toward the intact side.

Unfortunately for the clinician, as stated by Dedo in his study on experimental and clinical vocal fold paralysis, the expected findings in SLN paralysis "...are not consistent or obvious enough to be useful in clinically diagnosing superior laryngeal nerve paralysis."[16(p1503)] Neither of the two volunteers undergoing SLN injection for this study had an unequivocal rotation of the posterior glottis toward the paralyzed side, as described in the literature. The asymmetry of the traveling wave, however, was easy to observe stroboscopically.

Although the SLN patients demonstrated that their folds were vibrating at the same frequency, there was a difference in velocity and a phase lag, with the nonparalyzed fold completing the vibratory cycle before the paralyzed fold. This finding has been previously reported in both live dogs and excised larynges.[8,9] Similar asymmetry occurred, although to a greater degree, in the present study in RLN paralysis.

It is possible that the expected rotation of the posterior glottis was too subtle to be appreciated in these patients. In a canine study, the glottic rotation following unilateral CT paralysis was only 3.5° to 17°.[9] At the lower end of this range, the rotation probably is imperceptible.

Following SLN injection, the asymmetric vibration and morphology, supraglottic anesthesia, and inability to sing at a high pitch produced a constellation of findings consistent only with SLN paralysis, in the opinion of the authors. It is difficult to prove complete SLN paralysis, however, even if a loss of CT activity is documented on electromyography. It is possible, but unlikely, that there is another explanation for the stroboscopic findings.

Clinical correlation is required in the diagnosis of an isolated SLN paralysis with stroboscopy. Other possible causes of asymmetric laryngeal vibration should be considered, including scarring or a submucosal mass, which could rarely produce a similar stroboscopic picture.[2,18] In the setting of suspected SLN injury — for example, a postthyroidectomy patient with a loss of the ability to sing or modulate pitch in continuous speech — an asymmetric mucosal traveling wave or asymmetric vocal fold excursion suggests SLN paralysis, even if other characteristics such as posterior glottic rotation or tension asymmetry are not observed on indirect laryngoscopy alone. In such a situation, laryngeal electromyography provides a sensitive method to verify the presence of a paralysis.[19]

A method of synchronizing glottographic waveforms with videostroboscopic images has recently been developed in our laboratory.[11] Figures 5 and 6 indicate that in studies of patients with asymmetric laryngeal vibration, the first derivative of the EGG waveform provides information about the timing of laryngeal opening and closure, provided that adequate vocal fold contact occurs during phonation. The upward deflection in the EGG first-derivative waveform is a good indicator of the moment of opening, whether in normal patients or in those with asymmetric vibration. The nadir of the dEGG waveform has previously been shown to closely correspond to the moment of laryngeal closure.[20,21] The largest positive peak in the dEGG waveform has been shown to correlate with opening.[20] Our data from synchronizing the videostroboscopic images with glottographic waveforms (Figs 5 and 6) indicate that a similar relationship exists in both RLN and SLN paralyses. Childers et al[20] noted that there was a significant variation in dEGG waveforms dependent on the experimental conditions, such as frequency, which somewhat limits the accuracy of estimating glottic events based on dEGG.

Computerized digital analysis of stroboscopic images holds the promise for quantifying traveling wave abnormalities associated with laryngeal disorders. Figure 4 demonstrates the symmetry ratio applied to laryngeal paralysis. The ratios can provide an objective measure, allowing interpatient and intrapatient comparisons of asymmetry.

Although this study did not report data describing the results of reinnervation or treatment on videostroboscopic findings in RLN paralysis, stroboscopy can document a return of normal symmetry of laryngeal vibration, particularly when the thyroarytenoid muscle is reinnervated. Crumley[22] recently updated his experience with ansa hypoglossi nerve transfer for the treatment of unilateral vocal fold paralysis. He used stroboscopic analysis to document reinnervation, observing return to symmetric vibration in four of five patients assessed following reinnervation.[22]

In conclusion, stroboscopy is a useful tool in the evaluation of patients with suspected laryngeal paralysis. Paralysis of the SLN, RLN, or both results in asymmetric laryngeal vibration that is easily identified even by inexperienced observers. The mucosal wave has a greater velocity and travels further along the mucosa on the normal fold. The probable cause of these vibratory findings is the reduced stiffness in the paralyzed cord, which reduces the velocity and extent of the traveling mucosal wave. Stroboscopy can identify abnormal vibration in patients with otherwise normal findings on indirect examination. Our early experience suggests that stroboscopy cannot reliably distinguish RLN paralysis from vagal paralysis. Studies are being planned in the canine model to better quantify the traveling wave findings in laryngeal paralysis.

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