

Determination of Vocal Fold Mucosal Wave Velocity in an In Vivo Canine Model

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The vocal fold traveling wave is essential to normal voice production. The present investigation examined whether the velocity of the traveling wave (TWV) could be consistently determined in an in vivo canine model and if traveling wave velocity is affected by changes in the amplitude of recurrent laryngeal nerve stimulation (RLNS) and superior laryngeal nerve stimulation (SLNS). The results showed that traveling wave velocity increased with an increase in recurrent laryngeal nerve stimulation at low constant superior laryngeal nerve stimulation, but was poorly correlated with increases in recurrent laryngeal nerve stimulation at a high level of superior laryngeal nerve stimulation. Furthermore, traveling wave velocity was significantly correlated with superior laryngeal nerve stimulation at constant recurrent laryngeal nerve stimulation. This study demonstrated the feasibility of objectively measuring traveling wave velocity in vivo.

INTRODUCTION

A rational approach toward the development of improved techniques for the detection, diagnosis, and treatment of vocal pathologies depends on an improved understanding of voice mechanisms. To achieve these goals, the properties of phonatory performance need to be understood. Although many objective measures of phonation are routinely performed, some fundamental properties of laryngeal behavior are not well understood.

One important property of the vocal folds that has not been measured is the velocity of the vibrating glottic mucosa, defined as the distance between two points on the traveling wave, divided by the time it takes for the wave to travel between these two points on the vocal fold. Although the "traveling wave" velocity (TWV) of the glottic mucosa may be a fundamental

parameter of phonation, little is known about the factors that control it during normal and pathological states.

The traveling wave moves from the inferior to the superior portion of the vocal folds, and then moves laterally onto the surface of the vocal folds. The wave resembles a wave breaking on a shoreline. Through its motion, this wave has a primary influence on the rise and fall in the amount of air flowing through the glottis during voicing. These glottal air pulses form the vocal source, which influences the final acoustic signal emitted, which gives rise to the perception of voice. Consequently, the characteristics of the mucosal wave should have a profound effect on the sound produced by the larynx.

Historically, investigators have emphasized the importance of glottal mucosa in phonation.¹ In experiments with excised larynges in 1886, Lermoyez found that, in falsetto register, vibration was restricted to the mucosa and excluded the deeper layers of the lamina propria.² Saito, *et al.*³ radiographically tracked the movements of lead particles inserted at various points in the vocal fold. He observed that larger fold trajectories were found for particles in the mucosa relative to smaller trajectories in the underlying muscle. Fukuda, *et al.*⁴ showed that, as fundamental frequency increased, the size of the trajectory grew smaller and was limited to a smaller portion of the vocal fold.

A number of anatomical factors are pertinent to the formation and characteristics of the traveling wave. Hirano^{5,6} proposed the body-cover theory of vocal cord vibration. This theory suggests that the layers of the vocal fold divide into two groups which have different rheological properties, and thus are expected to act differently during vocal fold vibration. The cover is composed of squamous epithelium and the superficial and intermediate layers of loose connective tissue (or lamina propria). The cover is very pliable and has no intrinsic contractile properties; it can propagate a surface wave that facilitates energy transfer from the glottal airstream to the vocal fold tissue.⁷⁻⁹ The cover's tension is controlled by vocal fold length, primarily via contraction of the cricothyroid muscle.

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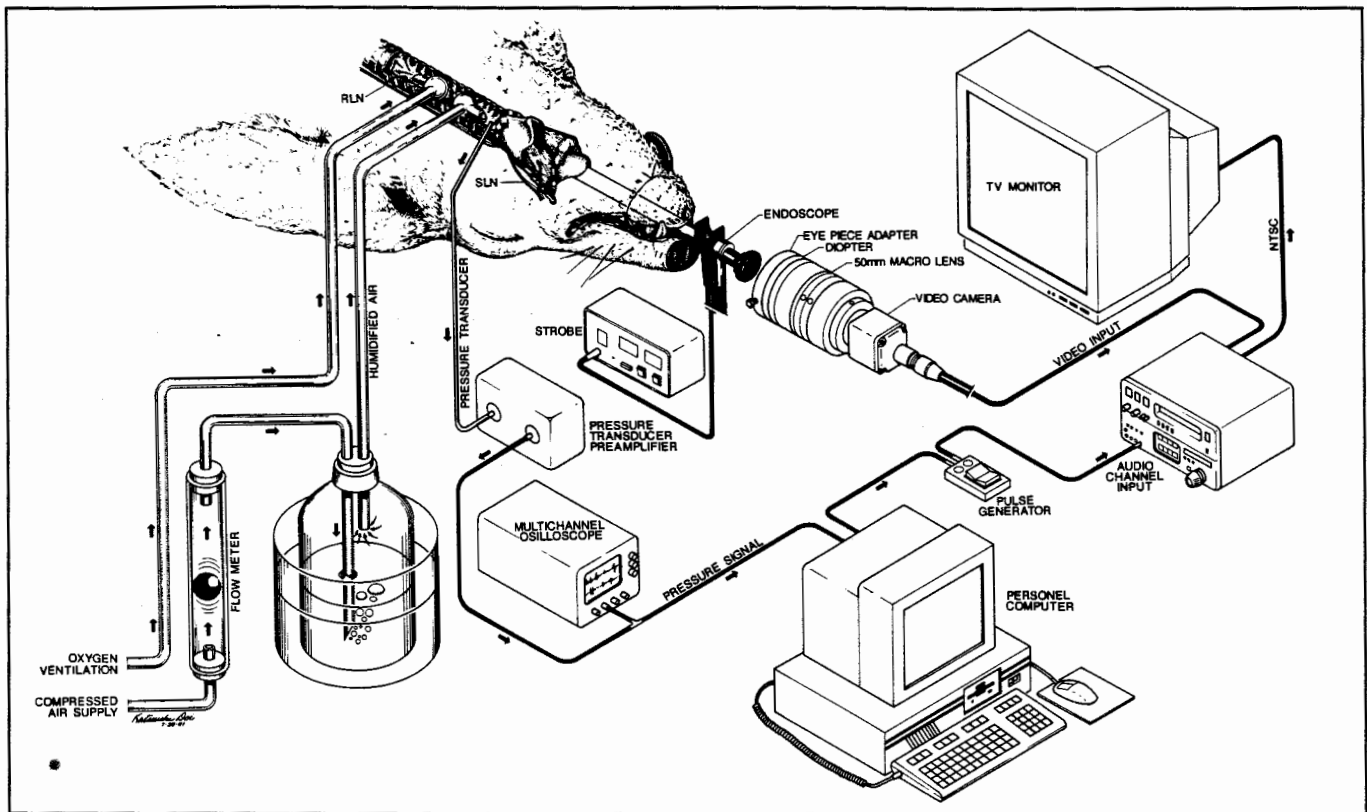


Fig. 1. Schematic representation of experimental setup for determination of traveling wave velocity (TWV).

The inner layer, or body, is composed of the deep layer of the lamina propria and thyroarytenoid muscle. It is stiffer than the cover because of the muscular layer, and has active contractile properties that primarily determine its tension. The muscular portion has been described as affecting the vocal fold configuration on which the mucosal cover vibrates.^{1,10} The passive stretch of the cover and the active contraction of the body thus are two contributors to overall vocal-fold stiffness, and play important roles in determining the fundamental frequency.

Baer¹¹ was the first to quantify the wave nature previously observed by Timcke, *et al.*^{12,13} Each point along the mucosal edge of the vocal fold moved in a quasi-elliptical trajectory in the coronal plane. This pattern of movement implied a vertical as well as horizontal motion to the mucosal wave. Baer estimated that the wave traveled at approximately 1 m per second, but its velocity varied at different locations on the vocal folds.¹¹

The study of traveling-wave velocity in pathological states of the larynx holds promise for surgical treatment of voice disorders. For example, Trapp, *et al.*¹⁴ studied states of simulated laryngeal paralysis in a canine model. Teflon® (polytef) injection of the paralyzed vocal fold to the point where the mechanical compliance or stiffness between the two vocal folds became roughly equivalent was associated with return of the traveling wave in the affected fold, and a

subsequent increase in the amplitude of the upper harmonics in the source spectrum. This study indicated that stiffness asymmetries between the vocal folds and abnormal mucosal-wave motion produced abnormal vocal fold vibration and dysphonia. These findings suggest that the presence of symmetric vocal fold stiffness implies symmetric vocal-fold traveling-wave motion. Thus, if TWV could be accurately determined intraoperatively, the targeting of normal values could optimize the results of phonosurgery.

The purpose of this study was twofold: 1. To determine whether traveling-wave velocity could be reliably measured in an *in vivo* canine model. 2. To test the hypothesis that altering vocal fold stiffness by varying the amount of recurrent laryngeal nerve (RLN) and superior laryngeal nerve (SLN) stimulation would alter traveling wave velocity.

METHODS

The experimental preparation was similar to that previously reported in studies using the *in vivo* canine model.¹⁵ Figure 1 diagrams the experimental setup. Four healthy adult male mongrel dogs (25 kg) were studied. The first three dogs were used to practice the tattooing technique, study the specifics of dot placement, and analyze video images in order to reliably determine TWV. All four dogs had similar results, but only the data from the fourth dog were analyzed.

The animals were premedicated with acepromazine

maleate intramuscularly. Intravenous pentobarbital was then administered to a level of corneal sensation loss; additional amounts were used to maintain this level of anesthesia throughout the experiment. The animals were placed in a supine position and direct laryngoscopy was performed to confirm normal laryngeal anatomy. A 7-mm orotracheal tube was inserted and the animal was given assisted ventilation with 95% oxygen.

A midline neck incision was made to expose the trachea and larynx. The strap and sternocleidomastoid muscles were retracted laterally. A low tracheotomy was performed at the level of the suprasternal notch, through which an endotracheal tube was passed to allow ventilator-assisted respiration. 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 resting 10 cm below the glottis. The cuff of this tube was inflated to just seal the trachea. Room air from a compressed air tank was bubbled through 5 cm of water at 37°C for warming and humidification, and passed through this rostral tube. Flow was controlled with a needle valve (Whitey, Highland Heights, Ohio) and measured with a flowmeter (Gilmont #1500, Great Neck, NY). 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.

To determine traveling wave velocity, the vocal folds were tattooed by placing Higgins waterproof black India ink in the submucosal plane. A custom-made stainless-steel ink-dot applicator with two sharp prongs 1.5 mm apart was used. This applicator was gently advanced deep to the glottic mucosa, forming two distinct indelible dots. One pair of dots was placed at the medial edge of each vocal fold, halfway between the anterior commissure and the vocal process of the arytenoid corresponding to the upper and lower lips of the fold. Figure 2 demonstrates the stroboscopic images and corresponding schematic diagram of the vocal folds to illustrate the position of the upper and lower dots. The vertical distance between these two dots, 1.5 mm, was used in the TWV calculation. Each dot was 0.5 mm in diameter.

A 1-cm segment of the external branch of each superior laryngeal nerve (SLN) was isolated and Harvard subminiature electrodes (South Natick, Mass.) were applied to each nerve. The recurrent laryngeal nerve (RLN) was isolated bilaterally 5 cm inferior to the larynx and Harvard electrodes were applied. A Grass model 54H stimulator (Quincy, Mass.) provided voltage stimulation to both SLNs. A constant current stimulator (WR Medical Electronics RLN Stimulator, Model S2LH, St. Paul, Minn.) was used to provide varying amounts of current to the RLNs. The frequency of stimulation was 80 Hz with a pulse duration of 1.5 msec for both nerve stimulators. Voltage varied from 0 to 2 V for the SLN stimulator, and currents ranged from 0.12 to 0.33 mA for the RLN stimulator. Electrical isolation between the RLN and SLN was verified by direct observation. Maximal stimulation of the RLNs to the point at which the strap muscles contracted (approximately 9 V) was not observed to produce contraction of the cricothyroid muscle. In addition, no lengthening or thinning of the vocal folds occurred during maximal RLN stimulation. Isolated maximal stimulation of the SLNs to the point of strap-muscle contraction did not demonstrate tensing or bulging of the vocalis muscle on direct laryngoscopic exam. No arytenoid adduction nor phonation could be elicited by maximal SLN stimulation.

Flow, Pressure, and Stroboscopic Measurements

The rate of air flow was 330 mL per second. Air flow was controlled with a Gilmont flow meter (Model 1500, Great Neck, NY) and passed through the air-warming chamber prior to entry into the larynx.

A Millar catheter-tipped pressure transducer (SPC 330-3F, Houston, Tex.) was inserted through the upper tracheotomy to rest 2 cm below the glottis. The transducer was calibrated at the temperature of the animal's trachea by submerging it in a water bath at 37°C to a depth just covering the sensor (0.5 cm) and then calibrating it against a Mercury manometer from 0 to 100 mm Hg. The fundamental frequency of each trial was calculated by measuring the vocal period from the subglottic pressure curve. The recorded subglottic pressure waveforms were low-pass filtered at 3000 Hz and digitized at a rate of 20 kHz.

Videostroboscopy was performed using a Storz laryngostroboscope (Model 8000, Culver City, Calif.) connected with a fiberoptic cable to a 0° Storz telescope for observation of vocal fold vibratory excursion and traveling wave speed. Images were recorded using a CCD camera (Toshiba IK C30A, Buffalo Grove, Ill.) and 3/4-inch videotape recorder (SONY VO-9850, Park Ridge, NJ). Recorded video images were viewed on a SONY video monitor (PVM 1341) and analyzed frame by frame.

Experimental Design

Each animal's laryngeal nerves were simultaneously stimulated at various intensities to produce phonation at a constant air-flow rate of 330 mL per second. Each animal experiment was divided into three parts.

First, the effect of varying RLN stimulation on TWV at a constant low SLN stimulation level was investigated. While the SLN was stimulated bilaterally at 1.0 V, the animals were phonated at five different RLN intensities corresponding to five different target subglottic pressures. Two trials with a RLN stimulation at 0.15 mA, corresponding to a subglottic pressure of approximately 50 mm Hg, were performed. Next, trials at a lower RLN stimulus of 0.12 mA, corresponding to subglottic pressures averaging 40 mm Hg, were performed. The remaining trials used three higher RLN stimuli (0.17, 0.20, and 0.24 mA), corresponding to target subglottic pressures of 60, 70, and 80 mm Hg, respectively. At least two trials were performed at each level of RLN and SLN.

Second, the effect of varying SLN stimulation on TWV at constant RLN-stimulation levels was examined. While the RLN was stimulated bilaterally at 0.17 mA, the animals were phonated at four different levels of SLN stimulation. Trials were recorded at SLN-stimulation intensities of 0.5 V, 1.0 V, 2.0 V, and without SLN stimulation.

Lastly, the effect of varying RLN-stimulation intensities on TWV was examined for a constant high SLN stimulation level. The animals were phonated at an SLN-stimulation intensity of 1.7 V and at RLN-stimulation intensities of 0.16, 0.21, 0.25, 0.30, and 0.33 mA.

Wave Velocity Calculation

The traveling wave velocity (TWV) was defined as the distance between the dots placed at the upper and lower lips of the vocal fold's medial edge, divided by the time it takes

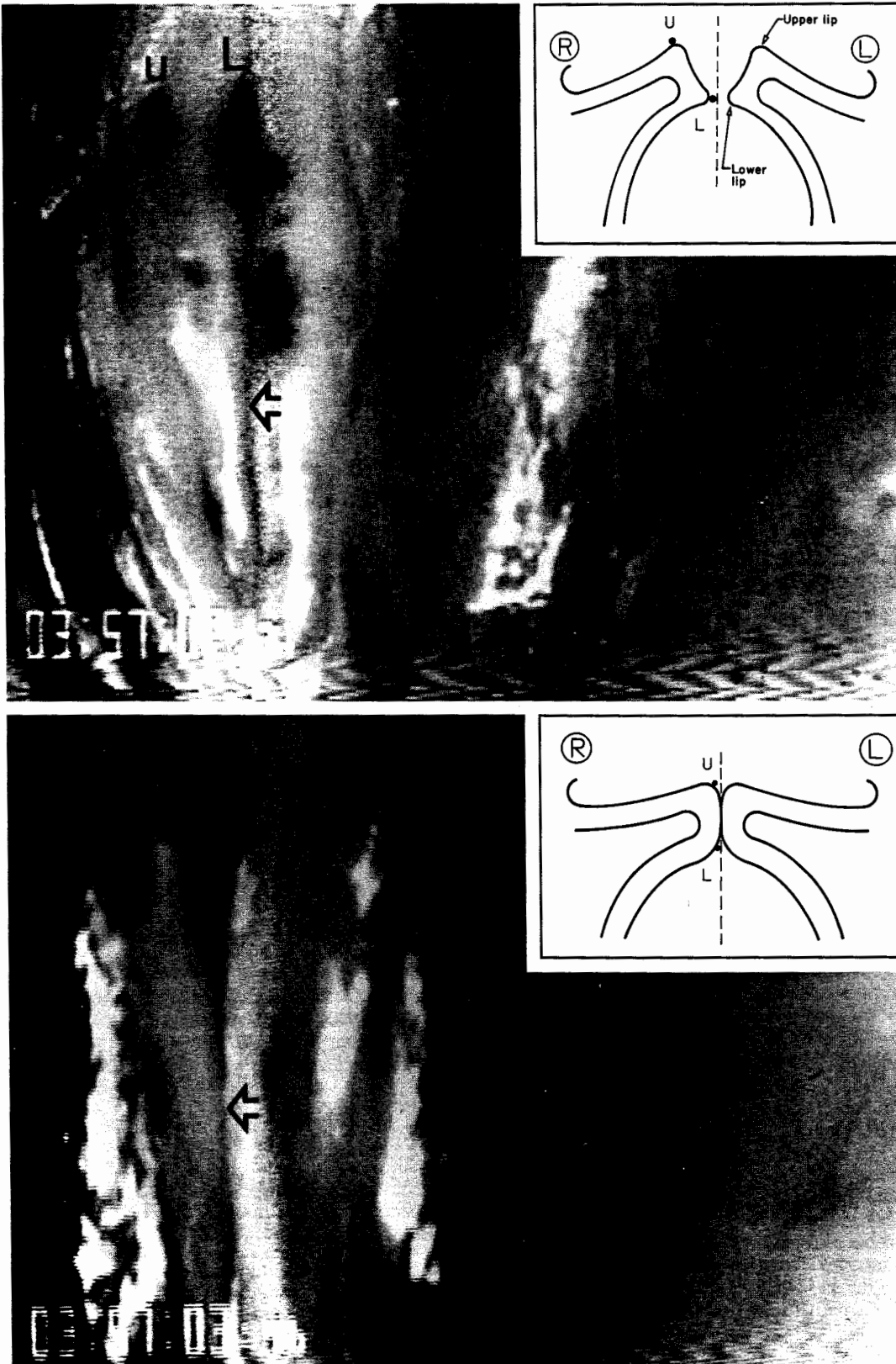


Fig. 2. Stroboscopic images and corresponding schematic diagrams of the canine larynx at two different times during the vocal period. The arrow denotes the midline. U represents the dot placed on the upper lip of the right vocal fold and L represents the dot placed on the lower lip. **Top.** The medial edge of the traveling wave is positioned at the lower dot. **Bottom.** Later in the vocal period, the medial edge of the vocal fold is positioned at the upper dot. The number of videostroboscopic images between these two events during the vocal period is counted, and the distance between the upper and lower dot is measured in order to calculate the TWV.

the wave to travel from the lower to the upper dot. Time was considered as that fraction of the vocal period (inverse of frequency) that the mucosal wave traveled from the lower to upper dot. This was calculated by knowing three values:

1. the number of video fields occurring during the time the mucosal wave traveled from the lower to upper dot (VF_{Betw}), 2. the number of fields in a vocal period (VF_{Per}), and 3. the fundamental frequency (F_0). This yields the following

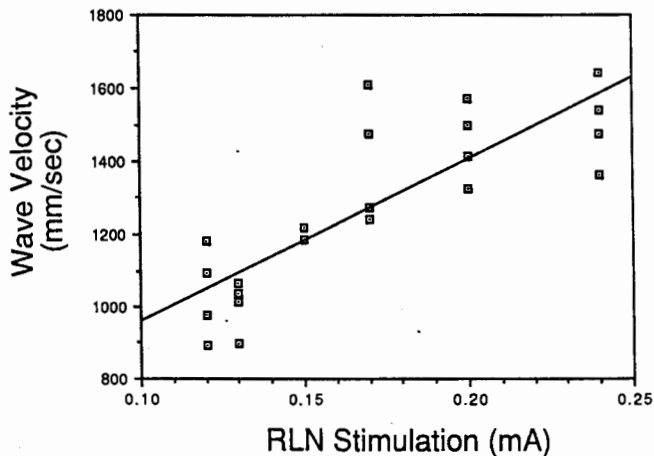


Fig. 3. Scatter diagram of relationship between traveling wave velocity (TWV) and recurrent laryngeal nerve stimulus (RLNS) at low constant superior laryngeal nerve stimulus (SLNS) with the linear regression line.

equation:

$$\text{Traveling Wave Velocity} = \frac{(1.5 \text{ mm})(F_0)(VF_{Per})}{(VF_{Betw})}$$

* Measurements to calculate TWV were performed only when periodic phonation was observed. The number of video fields that elapsed during the time that the mucosal wave traveled from the lower to upper dot was counted. For each trial, this counting procedure was performed by three experimenters independently. The number of video fields in each vocal period was automatically held constant at 120 per vocal cycle by the stroboscope (there are two video fields per frame). Lastly, the distance between the upper and lower dots on each cord was confirmed using a microcaliper and an operating microscope after the larynx was excised.

Data Analysis

Separate calculations were made for each right and left vocal fold for all the trials at each level of RLN and SLN stimulation in the one animal. Trials with aperiodic vibration or poor video images were excluded. The TWV of the left and right vocal folds for each individual trial were also compared to assess consistency of our methods.

RESULTS

Figure 3 demonstrates the relationship of the TWV measured from both vocal folds with increasing RLN stimulation at a constant low SLN stimulation (1 V). A linear regression line provided the best fit to the present data set, although the nature of the "true function" relating these qualities is difficult to determine from our small data set. TWV ranged from 897 mm per second at 0.13 mA to 1640 mm per second at 0.24 mA. The Pearson's correlation of $r = .83$ ($P < .05$) shows a moderately high association between RLN stimulation and TWV at this level of SLN stimulation ($P < .05$). In contrast, the correlation of $r = -.41$ ($P < .05$) demonstrates a low to moderate, inverse relationship between these two variables at high SLN stimulation (1.7 V) (Fig. 4). TWV ranged from 864 at

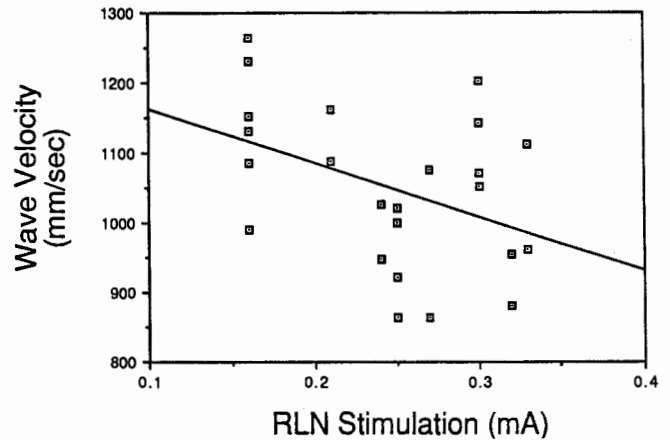


Fig. 4. Scatter diagram of relationship between TWV and RLNS at high constant SLNS with the linear regression line.

0.27 mA to 1232 mm per second at 0.16 mA in this condition.

At a constant, moderate level of RLN stimulation (0.17 mA), changes in SLN stimulation correlated with TWV at $r = .70$ ($P < .05$). Figure 5 demonstrates this relationship. TWV ranged from 990 mm per second without SLN stimulation to 1609 mm per second at 1 V of stimulation.

Figure 6 shows the relationship between subglottic pressure and TWV for all stimulation levels of RLN and SLN ($r = .79$, $P < .05$). TWV ranged from 860 mm per second at 40 mm Hg to 1640 mm/s at 82 mm Hg. Additionally, TWV significantly correlated with F_0 ($r = .81$).

To look at the reliability of our method for measuring TWV, the TWV values for the right and left vocal folds for all trials were compared. The mean absolute difference between the two folds was 9.1% (102 mm per second) of the mean TWV (1168 mm per second), and ranged from 2.2% (22 mm per second) to 21.8% (211 mm per second), with a standard deviation of 5.5%. Thus, the values for the two vocal folds were relatively close.

DISCUSSION

The present investigation was designed to study whether the velocity of the vocal fold traveling wave could be measured in an in vivo canine model, and to test the hypothesis that TWV increases with increasing muscular contraction of the vocal fold under conditions of constant air flow.

The results showed that for constant low SLN contraction, changes in RLN contraction correlated moderately highly with increased TWV. Thus, the presumed increase in stiffness of the body of the vocal fold was associated with increasing TWV. Similarly, the presumed increase in stiffness of both the body and cover of the vocal fold that occurred as SLN stimulation increased during constant RLN stimula-

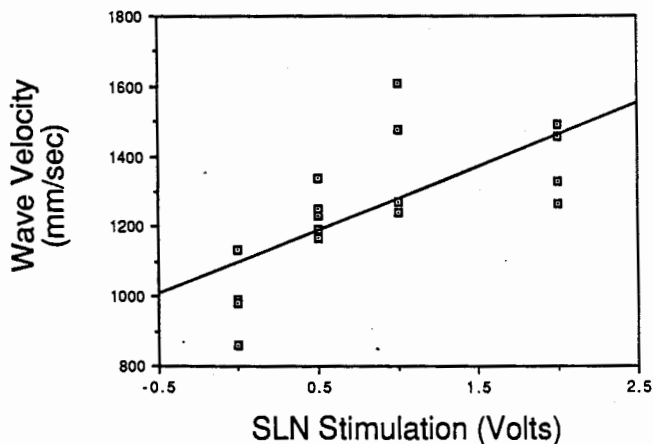


Fig. 5. Scatter diagram of relationship between TWV and SLNS at constant RLNS with the linear regression line.

tion was associated with increasing TWV. However, the low correlation between changes in RLN stimulation and TWV at a constant high level of SLN stimulation was an anomalous finding. As frequency increased, the TWV increased over a range of 900 to 1600 mm per second.

- Stimulation of the RLN activates the thyroarytenoid, lateral cricoarytenoid, interarytenoid, and posterior cricoarytenoid muscles. Contraction of the thyroarytenoid has been noted to cause a medial bulging at the midportion of the vocal cord¹⁶ and a more rounded, thickened cord.^{6,17} Physiologically, thyroarytenoid contraction should increase the stiffness of the body of the vocal cord, leading to an increase in fundamental frequency. Furthermore, TWV should increase with increasing RLN stimulation because velocity of an object in a viscoelastic medium is proportional to the square root of the stiffness. Thyroarytenoid contraction also leads to a slackening of the mucosal cover of the vocal fold. This slackening was visibly apparent at low constant SLN stimulation.

In contrast, at very high levels of SLN stimulation, the mucosal cover of the vocal fold appeared maximally stretched and, presumably, was unable to slacken during vocalis contraction. In this condition, a low, inverse correlation between the TWV and increasing RLN stimulation was noted. Previous research has suggested that increased thyroarytenoid muscle activity may either increase or decrease fundamental frequency depending on cricothyroid muscle activity.¹⁸ Titze, *et al.* have shown that at low cricothyroid activity, the thyroarytenoid is better able to increase the net tension of the vibrating portion of the vocal fold, because the tension of the cover is rather low.¹⁹ At high cricothyroid activity, on the other hand, the cover is maximally stretched and it becomes difficult for the body to bring about a substantial increase in the net tension of the vibrating system.

Another possible explanation for the lack of correlation between traveling-wave velocity and RLN

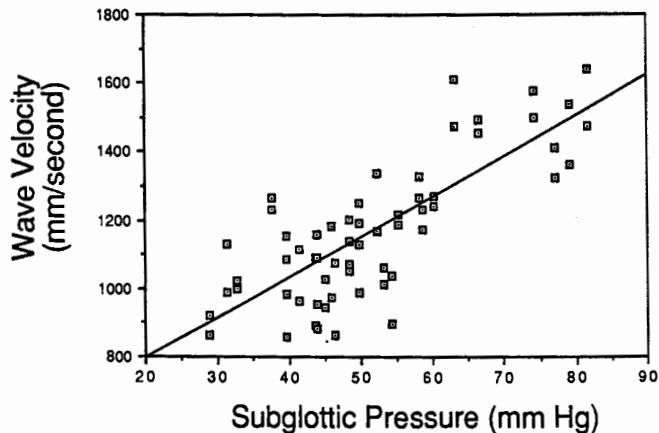


Fig. 6. Scatter diagram of relationship between TWV and subglottic pressure with the linear regression line.

stimulation at high SLN stimulus is the morphological differences between the canine and human larynx. The canine has a thicker superficial layer of the lamina propria and virtually lacks a vocal ligament. The cover seems to be more loosely connected to the body in the dog than in the human. This potential difference in the distribution of cover and body tissue in the vibrating portion of the vocal fold may be important.¹⁹ In the human, when the thinner cover is slack and the amplitude of vibration is sufficiently large to include a portion of the muscle in vibration, increasing thyroarytenoid activity will raise pitch. This is because the increase in muscle tension outweighs the decrease in tension in the cover that may result from a small decrease in vocal fold length. This effect increases with increased vibrational amplitude or loudness because more of the muscle is involved in vibration. However, at high SLN stimulation the thick canine lamina propria may cause the relative amplitude of vibration to be very small, such that none of the muscle is vibrating. In this condition, changes in thyroarytenoid muscle activity would not affect the overall stiffness of the vibrating part of the vocal fold. Thus, the relative thickness of the vocal fold cover in the canine may also help explain our results.

The results demonstrated that TWV is correlated with fundamental frequency, subglottic pressure, and presumably, vocal fold stiffness. It should be emphasized that correlation does not imply causality. For example, it is quite possible that the same factors that produce an elevated subglottic pressure, *i.e.*, vocal fold stiffness, also increase TWV and that an elevated subglottic pressure, in itself, would have no direct influence on TWV. Given the design of this study, it is difficult to isolate the direct effect of one variable upon another, such as subglottic pressure on TWV.

The determination of TWV in the *in vivo* canine model required tattooing of the vocal fold. This process created easily visible, distinct, and indelible marks on the vocal fold. The exact placement of the

dots on the vocal fold was crucial. For example, the level at which the dots were placed on each vocal fold had to be the same, since TWV could be different at various locations on the vocal fold.¹¹ It also became apparent that the dots had to be made close together. This was because, at high SLN stimulation, the mucosal cover was maximally stretched and less of the vertical dimension of the vocal fold was involved in the mucosal-wave motion. Furthermore, the ink must be placed in the submucosal plane. Any mark placed topically on the vocal fold mucosa washed off or became distorted during phonation. Although this tattooing method is invasive, no vocal fold bleeding occurred. More importantly, the vocal fold motion appeared unchanged after the marks were created. This method for determining TWV proved effective in the canine model, but is too traumatic for human application.

As suggested by Baer, TWV is not constant. Variation in this velocity may be one of the determining factors in control of fundamental frequency (F_0). At first glance it may appear that F_0 should be directly related to TWV. However, some factors that affect F_0 have no influence on TWV. For example, F_0 is also dependent on the rate in which the airstream fills the subglottic vault.²⁰ This filling occurs during the time in the glottal cycle that the vocal folds are closed and at rest prior to any mucosal movement. In contrast,

TWV is measured only during the open interval of the glottal cycle when mucosal wave movement is present.

Future studies should explore TWV asymmetries. TWV asymmetries due to unilateral changes in mass or stiffness may be a unique characteristic of dysphonia. Loss of normal wave morphology in paralysis, scarring, and neoplasia have been documented. If wave velocities between normal and abnormal vocal folds could be compared over time, this information could be used to more objectively guide both the medical and surgical treatment of these lesions.

CONCLUSION

The results of this study indicate that traveling wave velocity can be determined consistently in an in vivo canine model. It suggests that TWV increases with an increase in RLN stimulation at low constant SLN stimulation, but is poorly correlated at a high level of SLN stimulation. In addition, the results showed TWV increased with an increase in SLN stimulation. This study may improve our understanding of TWV as an objective measure of laryngeal function during normal and pathological voicing. Future studies should work toward documenting how TWV differs for a variety of vocal fold lesions, including paresis and paralysis. In addition, reliable, practical, and noninvasive methods for determining TWV in humans need investigation.

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