Characteristics of an In Vivo Canine Model of Phonation With a Constant Air Pressure Source

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Many previous studies of laryngeal biomechanics using in vivo models have employed a constant air flow source. Several authors have recently suggested that the lung-thorax system functions as a constant pressure source during phonation. This study describes an in vivo canine system designed to maintain a constant peak subglottic pressure (P_{sub}) using a pressure-controlling mechanism.

Increasing levels of recurrent laryngeal nerve (RLN) stimulation resulted in a significant rise in resistance followed by a plateau. For a given P_{sub}, flow decreased significantly and precipitously with increasing stimulation and then quickly plateaued. Vocal intensity increased with increasing RLN stimulation until a peak was reached. After this peak, intensity dropped until a plateau was reached, corresponding to the flow minimum. At a given P_{sub}, increasing levels of RLN stimulation resulted in a normal distribution of vocal efficiencies.

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INTRODUCTION

Recent debate has centered on the question of whether the lung-thorax system acts as a constant air pressure source or a constant air flow source in providing the vocal tract with the aerodynamic energy needed for speech production. The majority of biomechanical studies examining pressure-flow characteristics of the larynx have been designed with the flow generated by the lung-thorax system as an independent variable and the subglottic pressure (P_{sub}) as a dependent variable, implying that the system is flow regulated.[1–4]

The concept of pressure regulation was supported by the interlabial tube leak experiments of Putnam et al.[5] These investigators discovered that as the cross-sectional area of the leak tube increased, flow through the tube also increased. They concluded that pressure was controlled, while flow was allowed to vary. The concept of active pressure regulation has been more recently challenged by the theory that the lung-thorax unit provides a constant air pressure without the need for any active feedback mechanism.[6] In this view, muscular activity and the elastic recoil of the lung-thorax unit combine to maintain pressure levels passively.

Some authors have assumed a constant pressure source in their theoretic formulations of sound production. In Ishizaka and Flanagan's network model for the synthesis of voiced sounds,[7] a constant excess pressure in the lungs was employed as an approximation of P_{sub}. The notion of pressure maintenance was addressed in 1986 by Warren.[8] He claimed that the primary goal of the lung-thorax system was the regulation of air pressure in order to generate a driving force above a minimum level. In another study, Warren et al.[9] assumed that pressures were maintained by reflexive changes in the resistance of the vocal tract or by alterations in the level of muscular contraction. This implied the existence of a feedback mechanism through which the pressure or the acoustic quality of the voice was monitored and regulated. To block the auditory feedback loop, auditory masking studies of articulatory quality were performed.[10] These studies demonstrated that peak intraoral pressures remained above a certain level regardless of the auditory masking, suggesting that constant pressures are maintained without the need for an auditory perceptual feedback regulation mechanism.

The present investigation was undertaken to examine the characteristics of a constant pressure source of phonation using an in vivo canine model. The

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unique feature of this model is that it permits independent regulation of airflow, P_{sub}, and nerve stimulation level. The effects of variations in recurrent laryngeal nerve (RLN) and superior laryngeal nerve (SLN) stimulation on airflow, resistance of laryngeal airway, and vocal efficiency at various levels of P_{sub} are examined.

MATERIALS AND METHODS

In Vivo Canine Model

Three adult male mongrel canines, each weighing 25 to 30 kg, were used in this study. Each dog was screened to assess its suitability for the experiment, assuring bilateral symmetrical vocal fold motion and normal gross laryngeal anatomy.

The methods follow those used in previous in vivo canine studies.[1] Each dog was anesthetized with acepromazine maleate administered intramuscularly. Intravenous pentobarbital sodium (Nembutal) was administered to a level of corneal anesthesia. Additional pentobarbital was used to maintain this level of anesthesia throughout the procedure.

In each procedure, the animal was placed supine on an operating table, and an incision was made in the midline from the sternal notch to the hyoid bone. The strap muscles were then retracted laterally to expose the larynx and the trachea. At the level of the suprasternal notch, a distal tracheotomy was performed, and an endotracheal tube was inserted for ventilation. An additional proximal tracheotomy was performed, and a cuffed endotracheal tube was passed in a rostral direction with the tip positioned 10 cm below the glottis. The cuff of the rostrally directed tube was inflated to just seal the trachea. The tube was then attached to the pressure-regulating system.

The recurrent laryngeal nerves were isolated in the tracheoesophageal groove approximately 5 cm from the inferior border of the thyroid cartilage. The external branches of the superior laryngeal nerves were isolated at their entrance into the cricothyroid muscle. Bipolar electrodes (Harvard laboratories, South Natick, Mass.) were applied to both RLNs and SLNs. Electrical isolation between the RLNs and SLNs was verified by direct observation. Maximum stimulation of the recurrent laryngeal nerves to the point at which the strap muscles were also noted to contract did not produce contraction of the cricothyroid muscle. In addition, no lengthening or thinning of the vocal folds occurred during maximum RLN stimulation. Isolated maximum SLN stimulation to the point at which the strap muscles were noted to contract did not demonstrate tensing or bulging of the vocalis muscle on direct laryngoscopic observation.

A nerve stimulator (Model 54H; Grass Instruments, Quincy, Mass.) was used to provide variable voltage stimulation to both superior laryngeal nerves. A second stimulator (Model 2SLH; WPI Medical Electronics Co., St. Paul, Minn.) was used to provide a constant current stimulus to the recurrent laryngeal nerves. For both the Grass and WPI units, the frequency of stimulation was 80 Hz and the pulse duration was 1.5 msec.

Electroglottography (EGG) electrodes (Synchrovoice, Harrison, N.J.) were sutured in direct contact with either side of the thyroid cartilage, and the reference electrode was sutured to the skin. A 1.0-cm button was placed through the epiglottis to improve visualization of the vocal folds. A sound intensity meter (Quest, Culver City, Calif.) was suspended 30 cm away from the glottis outside of the air flow stream. This was used to measure the acoustic intensity of the phonation.

A catheter-tipped pressure transducer (Model SPC 330; Millar Instruments, Houston, Tex.) was inserted through the upper tracheotomy to rest 2 cm below the glottis. The transducer was calibrated against a manometer from 0 to 120 mm Hg. The laboratory flow outlet was attached to the pressure-regulating system described below. A Gilmont flowmeter (Model f1500; Gilmont Instruments, Great Neck, N.Y.) was connected to the exit port of the flow-regulating system. Air flow was humidified and maintained at a temperature of 37°C by bubbling through 5 cm of water (H_2O).

Pressure-Regulating System

The pressure-regulating system used in this experiment is described in a previous work from this laboratory.[11] Briefly, it consisted of a cylindrical container measuring 75 cm in height and 42 cm in diameter. A hollow T-shaped tube was placed within this container. One end of the horizontal section of the T was connected to the laboratory-wall flow outlet; the other end was connected to the flowmeter. The cylinder was filled with water to submerge the vertical tip of the T tube. A ruler was attached to the inner surface of the cylinder to measure water pressure. Thus, the pressure buildup above the adjusted water level would be vented through the submerged tip of the T tube, resulting in a constant pressure.

Experimental Design

Target values of P_{sub} ranged from 25 to 70 cm H_2O in steps of 5 to 10 cm H_2O and varied by dog. Phonation was produced by stimulating the RLN at varying levels. The minimum level of RLN stimulation needed to produce phonation was determined at three levels of SLN stimulation: low (0.5 mV), medium (0.6 mV), and high (0.8 mV). The number of trials ranged from 99 to 116. Each trial lasted 5 seconds. The RLN stimulation steps and the order of target pressures were randomized.

Acoustic and Aerodynamic Measures

The EGG, P_{sub}, and acoustic signals were low-pass filtered at 3 kHz and digitized at 10 kHz using a 12-bit analog-to-digital converter (Scientific Solutions, Inc., Solon, Ohio). The signals were monitored on an oscilloscope (Tektronix 5116, Beaverton, Ore.) during data collection, and they were recorded on a personal computer. The data were then analyzed using C-Speech software for IBM.

Using the method of Yanagi et al.,[12] vocal efficiency was calculated according to the following formula: $V_{eff}=P_r/P_a$, where V_{eff} is vocal efficiency, P_r is the acoustical power, and P_a is the aerodynamic power.

P_a is a function of P_{sub} in centimeters of H_2O and airflow rate (Q) in cubic centimeters per second:

$$P_a=P_{sub}\times Q\times 980.6\times 10^{-7}\ (\text{watts})$$

Acoustical power radiating from a source is defined as $P_r=I\times 4\pi R^2$ (watts), where I is the sound intensity in watts

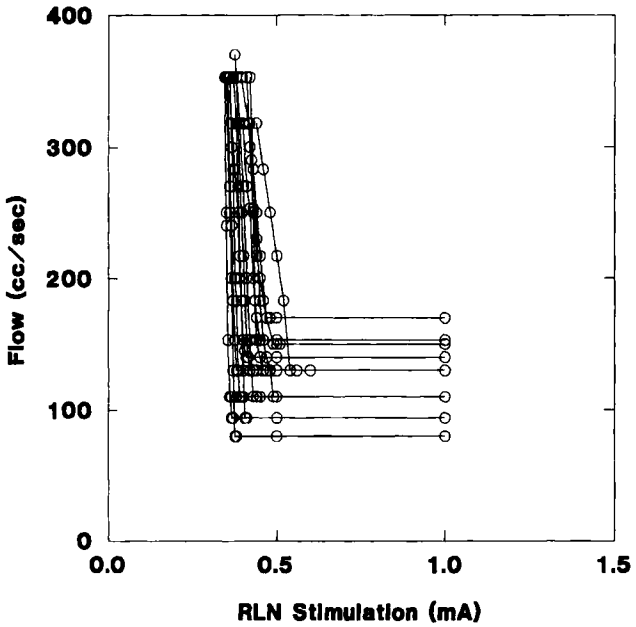

Fig. 1. Air flow versus the level of recurrent laryngeal nerve (RLN) stimulation at a constant subglottic pressure (P_{sub}) and superior laryngeal nerve (SLN) stimulation in a representative animal (P_{sub}=45 cm H_2O; SLN stimulation=low).

per square centimeter and R is the distance in centimeters between the sound meter and the vocal folds.

RESULTS

A two-way multivariate analysis of variance examined differences among dogs and SLN stimulation levels for dependent measures of air flow and vocal intensity. Changes in SLN stimulation had significant effects on intensity (F[2,463]=12.31, P<.01), but this effect accounted for very little variance in the underlying data (r^2=.045). Flow did not vary significantly with changes in SLN stimulation (F[2,463]=0.159, P>.01). Dogs differed significantly on both variables (intensity: F[2,463]=28.69, P>.01; flow: F[2,463]=15.19, P<.01), but no significant interactions between dogs and SLN stimulation levels were observed (intensity: F[4,463]=0.48, P>0.01; flow: F[4,463]=2.39, P>0.01).

Because SLN stimulation had no significant effect on flow, SLN levels were combined. The relationship between air flow and RLN stimulation is shown in Figure 1 for a representative animal. Each line in this figure represents a single trial. For a given level of P_{sub}, flow decreased significantly and precipitously with increasing stimulation and quickly plateaued. Table I shows significant tests for changes in air flow with RLN stimulation in three dogs at various levels of P_{sub}. Changes in flow with RLN stimulation are statistically significant (P<.01, adjusted for multiple comparisons).

Phonation occurred at a wide range of flow and stimulation levels (Table II). However, the range of RLN stimulation levels that produced phonation at any given level of P_{sub} was extremely narrow (0.008 to 0.10 mA). For dog 1, the average level of RLN stimulation was 0.065 mA (range: 0.05 to 0.08 mA; SD=0.011 mA); for dog 2, the mean RLN stimulation level was 0.025 mA (range: 0.008 to 0.05 mA, SD=0.011 mA); and for dog 3, the mean stimulation level was 0.047 mA (range: 0.015 to 0.10 mA, SD=0.023 mA). Linear regression showed that as P_{sub} increased, so did the RLN stimulation levels that produced phonation: for dog 1, F(1,7)=33.89, P<.01, r^2=.83; for dog 2, F(1,19)= 65.71, P<.01, r^2=.78; and for dog 3, F(1,13)=66.06, P<.01, r^2=.84.

For all three animals, both the amount of RLN stimulation needed to initiate phonation and the amount of RLN stimulation present when phonation ceased increased significantly with increasing P_{sub}. Onset of phonation in dog 1 was F(1,7)=8.61, P<.01, r^2=.55; in dog 2, F(1,19)=13.68, P<.01, r^2=.42; and in dog 3, F(1,13)=118.45, P<.01, r^2=.90. Cessation of phonation in dog 1 was F(1,7)=50.78, P<.01, r^2=.88; in dog 2, F(1,19)=43.48, P<.01, r^2=.70; and in dog 3, F(1,13)= 97.93, P<.01, r^2=.88.

Recall that laryngeal resistance is calculated as the ratio of P_{sub} to flow. Laryngeal airway resistance increased significantly with increasing RLN stimulation: for dog 1, F(1,56)=51.70, P<.01, r^2=0.48; for dog 2, F(1,105)=46.31, P<.01, r^2=.31; and for dog 3, F(1,82)= 170.78, P<.01, r^2=.68. The increase continued until a plateau was reached at a point corresponding to the flow minimum (shown as open circles in Figure 2, corresponding to data from a representative animal).

TABLE I.
Significant Tests for Changes in Air Flow With RLN Stimulation.

Dog	Target P_{sub}	F	df	P	r^2
1	50	42.86	1,19	<.01	.69
	60	21.63	1,19	<.01	.53
	70	13.33	1,14	<.01	.49
2	25	15.69	1,12	<.01	.57
	35	14.19	1,12	<.01	.54
	45	23.87	1,13	<.01	.65
	55	42.23	1,15	<.01	.74
	65	64.88	1,15	<.01	.81
3	25	18.48	1,10	<.01	.65
	35	21.35	1,15	<.01	.59
	45	63.93	1,17	<.01	.79
	55	60.70	1,19	<.01	.76
	65	12.40	1,14	<.01	.47

RLN = recurrent laryngeal nerve; P_{sub} = subglottic pressure; F = flow; df = degrees of freedom.

TABLE II.
Ranges of Flow and RLN Stimulation That Produced Phonation for Each Dog.

Dog	Flow (cm^3/sec)	RLN Stimulation (mA)
1	94 to 370	0.29 to 0.43
2	65 to 360	0.195 to 0.325
3	80 to 370	0.345 to 0.540

RLN = recurrent laryngeal nerve.

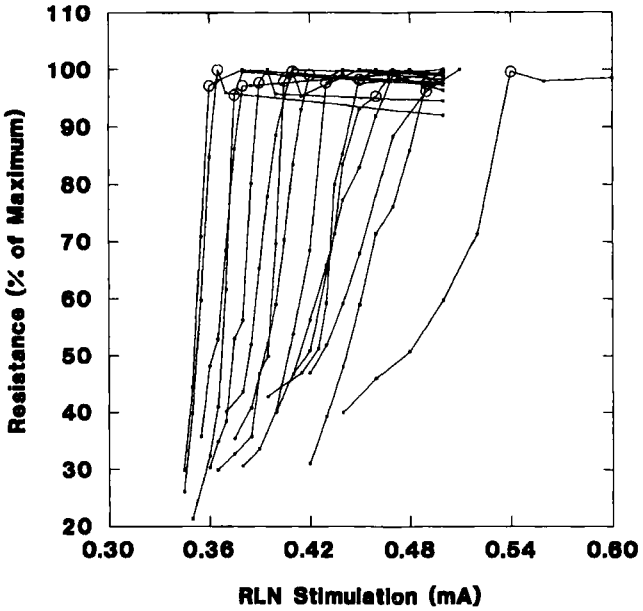

Fig 2. Percentage of maximum resistance versus the level of RLN stimulation (mA) at a constant subglottic pressure and SLN stimulation in a representative canine (P_{sub}=45 cm H_2O; SLN stimulation=low).

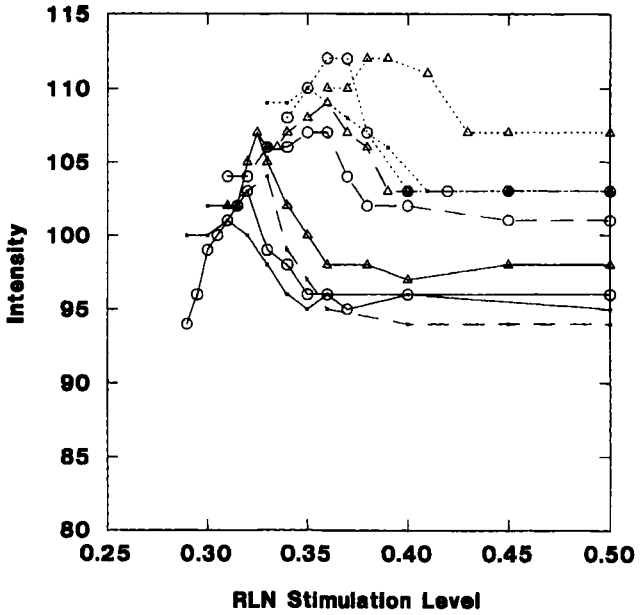

Fig. 3. Vocal intensity versus RLN stimulation level (mA).

Once the peak resistance was reached, phonation ceased and further increases in RLN stimulation produced no further changes in resistance: for dog 1, F(1,43)=2.91, $P>.01$; for dog 2, F(1,113)=3.27, $P>.01$; and for dog 3, F(1,61)=1.42, $P>.01$. As P_{sub} increased, so did the resistance level at which this peak occurred: for dog 1, F(1,7)=2.67, $P>.01$; for dog 2, F(1,19)=96.91, $P<.01$, $r^2=.84$; and for dog 3, F(1,13)=27.19, $P<.01$, $r^2=0.68$. Similarly, as P_{sub} increased, so did the amount of RLN stimulation needed to achieve peak resistance: for dog 1, F(1,7)=50.78, $P<.01$, $r^2=0.88$; for dog 2, F(1,19)=39.82, $P<.01$, $r^2=0.68$; and for dog 3,

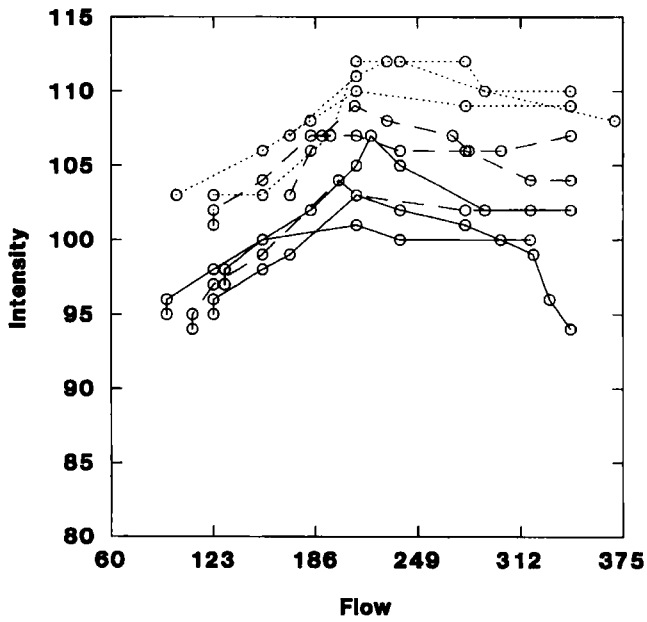

Fig 4. Vocal intensity versus air flow (cm^3/second).

F(1,13)=95.12, $P<.01$, $r^2=.88$.

Variations in vocal intensity with RLN stimulation are shown in Figure 3 for various levels of P_{sub}. For all levels of P_{sub}, intensity increased with increasing RLN stimulation until a peak was reached. As with laryngeal resistance, the location of this peak varied with P_{sub}; that is, more RLN stimulation was required to attain peak vocal intensity as P_{sub} increased: for dog 1, F(1,7)=12.37, $P<.01$, $r^2=0.64$; for dog 2, F(1,19)=20.15, $P<.01$, $r^2=.52$; and for dog 3, F(1,13)=117.18, $P<.01$, $r^2=.90$. After that peak, intensity dropped until a plateau was reached at a point corresponding to the flow minimum.

The particular intensity level at which the plateau occurred varied significantly with SLN stimulation level: F(2,240)=8.57, $P<.01$. Scheffé post-hoc comparisons indicated that intensity plateaued at higher levels for the high SLN stimulation condition than for the low or medium stimulation conditions, which did not differ, $P<.01$. Finally, the relationship between intensity and flow is shown for selected levels of P_{sub} in Figure 4. For all flow levels, intensity first increased and then decreased slightly with increasing flow.

Recall that vocal efficiency is the ratio of subglottic power to aerodynamic power and is a function of intensity, flow, and P_{sub}. Accordingly, changes in vocal efficiency with RLN stimulation (Fig. 5) reflect aspects of intensity, flow, and P_{sub}. As with intensity, vocal efficiency increased with RLN stimulation until a peak was reached. After the peak, increasing RLN stimulation produced a decrease in vocal efficiency until a plateau was reached. For each dog, the amount of RLN stimulation needed to produce maximum vocal efficiency increased with P_{sub}: for dog 1, F(1,7)=14.08,

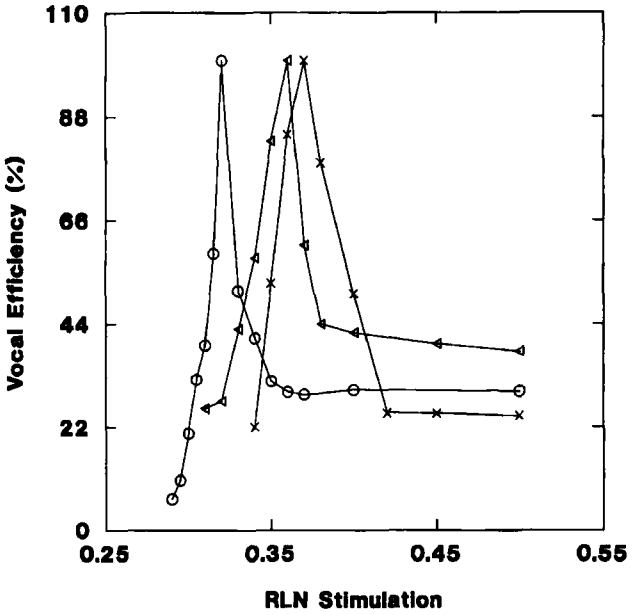

Fig. 5. Percentage of vocal efficiency versus RLN stimulation level (mA) at three levels of P_{sub} and a constant level of SLN stimulation.

$P<.01$, $r^2=.67$; for dog 2, $F(1,19)=12.97$, $P<.01$, $r^2=0.41$; and for dog 3, $F(1,13)=95.57$, $P<.01$, $r^2=.88$. The level of vocal efficiency at which the plateau occurred also increased significantly with P_{sub}: for dog 1, $F(1,7)=16.47$, $P<.01$, $r^2=.70$; for dog 2, $F(1,19)=7.95$, $P<.01$, $r^2=.30$; and for dog 3, $F(1,13)=29.65$, $P<.05$, $r^2=.70$.

DISCUSSION

To understand the concept of a constant air pressure source, one needs to envision the vocal tract as composed of at least two subunits: the larynx subunit and the lung-thorax subunit. The lung-thorax is similar to a large reservoir with negligible outflow resistance. As such, the aeromechanical properties of the lung-thorax subunit would be the main determinants of pressure provided to the subglottis. Coupled in series with the lung-thorax is the larynx subunit. This subunit is capable of producing much larger resistances than the lung-thorax subunit. Consequently, flow through the larynx would be regulated by changes in laryngeal resistance. However, laryngeal resistance has minimal effects on the subglottic pressure.

Laryngeal resistance is determined by the interplay of several elements.[13–15] Changes in the cross-sectional area of the larynx and the airflow velocity distributions across the larynx are the main factors responsible for pressure loss within the larynx. The vena contracta or the area of sudden narrowing within the larynx leads to pressure loss, while flow separation and the creation of turbulence result in pressure elevation.

It is possible to approximate the overall resistance of the vocal tract using the following formula: Resistance=Subglottic Pressure/Transglottic Flow. Thus, vocal tract resistance is calculated from the ratio of translaryngeal pressure (i.e., the subglottic pressure minus the supraglottic pressure) to the translaryngeal flow. Since supraglottic pressure is approximately equal to atmospheric pressure, it may be disregarded.

Some controversy exists regarding the effects of increasing levels of SLN stimulation on vocal tract resistance. It is known that vocal fold compliance is reduced as the level of SLN stimulation is increased. The change in the geometry of the glottis secondary to contraction of the cricothyroid muscle could account for this change in compliance. Photoglottographic techniques have revealed that with increasing SLN stimulation, the vocal folds remain approximated for a shorter portion of each vibratory cycle.[1] However, the present study did not demonstrate any significant change in vocal tract resistance with increasing levels of SLN stimulation. Similar conclusions have been described in other studies.[16,17]

Vocal tract resistance rose significantly and reached a plateau with increasing levels of RLN stimulation, resulting in a sigmoidal resistance curve. Two factors may account for the plateau in this curve. Since the vocal tract is attached to the surrounding structures, progressive narrowing will lead to an increasing elastic impedance. Thus, increasing muscular contractions will raise the resistance by a smaller increment as the elastic impedance rises. Moreover, it is known that there is a posterior chink in the canine vocal fold, which would, in effect, act as a shunt, limiting the degree to which muscular contractions can raise the resistance.

This investigation suggests that for each level of SLN stimulation and P_{sub}, there is a small range of values of RLN stimulus level that produces the highest vocal efficiency. Any deviation from this optimal value leads to a reduction of vocal efficiency. It is noteworthy that the optimal value of RLN stimulation at which the highest vocal efficiency was achieved appeared to coincide with the most subjectively clear phonation. Furthermore, at any level of P_{sub} and SLN stimulation, a minimal amount of RLN activity was required to induce phonation. Below this minimum level of RLN, no phonation occurred. It should be kept in mind that at this minimum level of RLN stimulation, P_{sub} had not yet reached a constant level. This was because the vocal tract had not developed enough resistance to generate a P_{sub} capable of overcoming the pressure of water in the cylinder.

Once P_{sub} reached a constant level, any increase in RLN stimulus level led to a fall in flow and a concomitant fall in aerodynamic power. Since less flow was needed to generate the same sound intensity, a fall in aerodynamic power led to an increase in vocal efficiency. Sound intensity remained constant until maximum vocal efficiency was reached. As RLN stimulation level was increased above this optimal level,

TABLE III.
Range of RLN Stimulation Needed for Phonation at Various Subglottic Pressures.

Subglottic Pressure ($cm H_2O$)	SLN Stimulation (mA)		
	Low	Medium	High
20	0.0200	0.0125	0.0100
30	0.0300	0.0300	0.0250
45	0.0325	0.0310	0.0305
50	0.0375	0.0350	0.0325
60	0.0500	0.0450	0.0450

RLN = recurrent laryngeal nerve; SLN = superior laryngeal nerve.

TABLE IV.
Minimum Level of RLN Stimulation Necessary for Phonation at Various Subglottic Pressures.

Subglottic Pressure ($cm H_2O$)	SLN Stimulation (mA)		
	Low	Medium	High
20	0.195	0.195	0.205
30	0.210	0.205	0.205
45	0.225	0.230	0.240
50	0.245	0.245	0.255
60	0.250	0.250	0.255

RLN = recurrent laryngeal nerve; SLN = superior laryngeal nerve.

sound intensity began to fall at a faster rate than flow. Consequently, there was a greater incremental reduction in acoustic power than aerodynamic power. Thus, vocal efficiencies began to decline. As the maximum RLN stimulation level was reached, intensity continued to fall, while as mentioned above, flow stabilized at a constant minimum level. Above this maximum level of stimulation, phonation did not occur.

From the previous discussion, one may conclude that for any constant level of P_{sub}, there is an optimal range of resistances at which vocal efficiencies are maximized. Thus, efficient phonation involves a pressure-resistance match. Any pressure-resistance mismatch will lead to the production of sound at suboptimal efficiencies characterized by weak, hoarse, and breathy phonation. Some clinical examples of this mismatch are spasmodic dysphonia and unilateral vocal fold paralysis. In both cases, excessive effort is required by the patient to approach the normal range of efficiency.

The assumption that the lung-thorax unit provides constant pressures for phonation necessitates the concept of threshold pressure. This can be defined as the minimum pressure below which no phonation occurs.[1] In the present study, as P_{sub} declined, so did the range of RLN stimulation capable of producing phonation (Table III). Consequently, in a constant pressure system, the threshold pressure is one at which the range of RLN stimulation necessary for phonation becomes zero. Our data obtained by lowering the target P_{sub} until this range became zero are consistent with a previous report which indicated that for the canine model of phonation, the threshold pressure is about 20 $cm H_2O$.[1]

An interesting observation is that the minimum RLN stimulation level required for phonation increased with increasing subglottic pressure (Table IV). Conversely, less resistance was required for phonation at lower P_{sub}. Conceptually, as P_{sub} is raised, more energy is available to blow apart the vocal folds. The folds are drawn together by the muscular, elastic, and Bernoulli restoring forces. Vocal fold vibration results from an alternating balance between the pressure on the folds and opposing restoring forces.[18] As less force is available for vocal fold separation, less force will be necessary to produce phonation.

It is not clear how RLN stimulation translates into actual muscular contraction. It is conceivable that a very narrow range of RLN stimulation may result in a great variation in muscular activity. This conversion factor may be determined by obtaining quantitative electromyographic data from the intrinsic laryngeal muscles. In addition, stroboscopic analyses may be useful in determining laryngeal configuration at various parts of the glottic cycle.

It is also important to note that the human laryngeal function is more complex than what is explained by this model. When stimulating the RLN, all the fibers are stimulated. However, there are selective activities that occur in normal phonation with variable function of each of the muscles innervated by the RLN. Therefore, pressure may not necessarily be constant with more variables at the glottis. This important possible limitation of the constant pressure model of phonation should be taken into consideration when extrapolating its findings.

CONCLUSION

The results of the present investigation demonstrate some characteristics of an in vivo canine model of phonation with a constant pressure source. The data indicate that relationships previously described using constant flow models may also be derived from a constant pressure system. Some phonatory phenomena not previously seen in constant flow models are also described. As discussed above, the concepts of pressure-resistance mismatch and threshold pressure have important clinical implications in the study of laryngeal dysfunctions.

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