

EFFECT OF TENSION, STIFFNESS, AND AIRFLOW ON LARYNGEAL RESISTANCE IN THE IN VIVO CANINE MODEL

STEVEN BIELAMOWICZ, MD

LOS ANGELES, CALIFORNIA

JOEL A. SERCARZ, MD

LOS ANGELES, CALIFORNIA

GERALD S. BERKE, MD

LOS ANGELES, CALIFORNIA

DAVID C. GREEN, MD

SEATTLE, WASHINGTON

JODY KREIMAN, PHD

LOS ANGELES, CALIFORNIA

BRUCE R. GERRATT, PHD

LOS ANGELES, CALIFORNIA

This study used an in vivo canine model of phonation to determine the effects of airflow on glottal resistance at low, medium, and high levels of recurrent laryngeal nerve (RLN) and superior laryngeal nerve (SLN) stimulation. Static and dynamic trials of changing airflow were used to study the effects of airflow on glottal resistance during phonation. As reported previously, glottal resistance varies inversely as a function of airflow. Increasing levels of RLN stimulation resulted in a statistically significant increase in glottal resistance for each level of airflow evaluated. Variation in SLN stimulation had no statistically significant effects on the relationship between flow and resistance. At air flow rates greater than 590 milliliters per second (mL/s), glottal resistance approached 0.1 mm Hg per mL/s for all levels of RLN and SLN stimulation tested. These data support the collapsible tube model of phonation.

KEY WORDS — airflow, glottal resistance, glottis, larynx, resistance.

INTRODUCTION

Laryngeal airway resistance during phonation is defined as the ratio of subglottal pressure to transglottal airflow. These measures are obtained by using methods described by Smitheran and Hixon,¹ in which oral airflow and oral pressure measures provide estimates of transglottal airflow and subglottal pressure. Glottal resistance was then calculated from these oral measures. Currently, these aerodynamic measures are often included in a clinical phonatory function evaluation because of their diagnostic implications.² For example, abnormally low measures of glottal resistance may be associated with glottal incompetence, as found in recurrent laryngeal nerve (RLN) paralysis.³ On the other hand, high glottal resistance may be associated with spasmodic dysphonia or a hyperfunctional voice disorder.⁴

Direct measures of subglottal pressure and translaryngeal airflow in excised and in vivo canine models of phonation have also been made. Muta and Fukuda⁵ studied airflow and pressure relations in excised canine larynges. They found that subglottal pressure increased with increasing airflow, with the rate of increase slowing at high flow levels. Thus, laryngeal resistance decreased as airflow increased in their study. The relationship of subglottal pressure and airflow was first evaluated in an in vivo canine

model in 1969 by Koyama et al.⁶ An increase in subglottal pressure with increasing airflow was again found. Koyama et al also studied the effect of superior laryngeal nerve (SLN) stimulation on pressure versus flow relationships, by comparing trials with various levels of SLN stimulation for fixed levels of RLN stimulation. They found an increase in glottal resistance between trials with no SLN stimulation and trials with low SLN stimulation. However, no statistical tests were performed to determine the significance of these results.

To further analyze the effects of airflow on laryngeal resistance, Smith et al⁷ used an in vivo canine model of phonation to evaluate the effect of increasing airflow on subglottal pressure, while keeping laryngeal nerve stimulation constant. They noted that subglottal pressure increased with increasing levels of airflow during trials in which flow increased dynamically. Contrary to previous findings, Smith et al reported a linear decrease in laryngeal airway resistance with increasing airflow. An explanation of the changes in laryngeal resistance with increasing airflow may be found in the work of Sercarz et al,⁸ who used digitized stroboscopic images to measure glottal area per cycle of phonation. Glottal area per cycle of phonation was calculated by integrating the area under the glottal area versus time waveforms created from the measured stroboscopic images.

From the Division of Head and Neck Surgery, University of California—Los Angeles School of Medicine, and the Division of Head and Neck Surgery, West Los Angeles Veterans Administration Medical Center, Los Angeles, California. Supported by the American Laryngological Association Research Award. This study was performed in accordance with the PHS Policy on Humane Care and Use of Laboratory Animals, the NIH *Guide for the Care and Use of Laboratory Animals*, and the Animal Welfare Act (7 U.S.C. et seq.); the animal use protocol was approved by the Institutional Animal Care and Use Committee (IACUC) of the University of California—Los Angeles.

Presented at the meeting of the American Laryngological Association, Los Angeles, California, April 17-18, 1993.

REPRINTS — Gerald S. Berke, MD, Division of Head and Neck Surgery, UCLA Medical Center, Los Angeles, CA 90024.

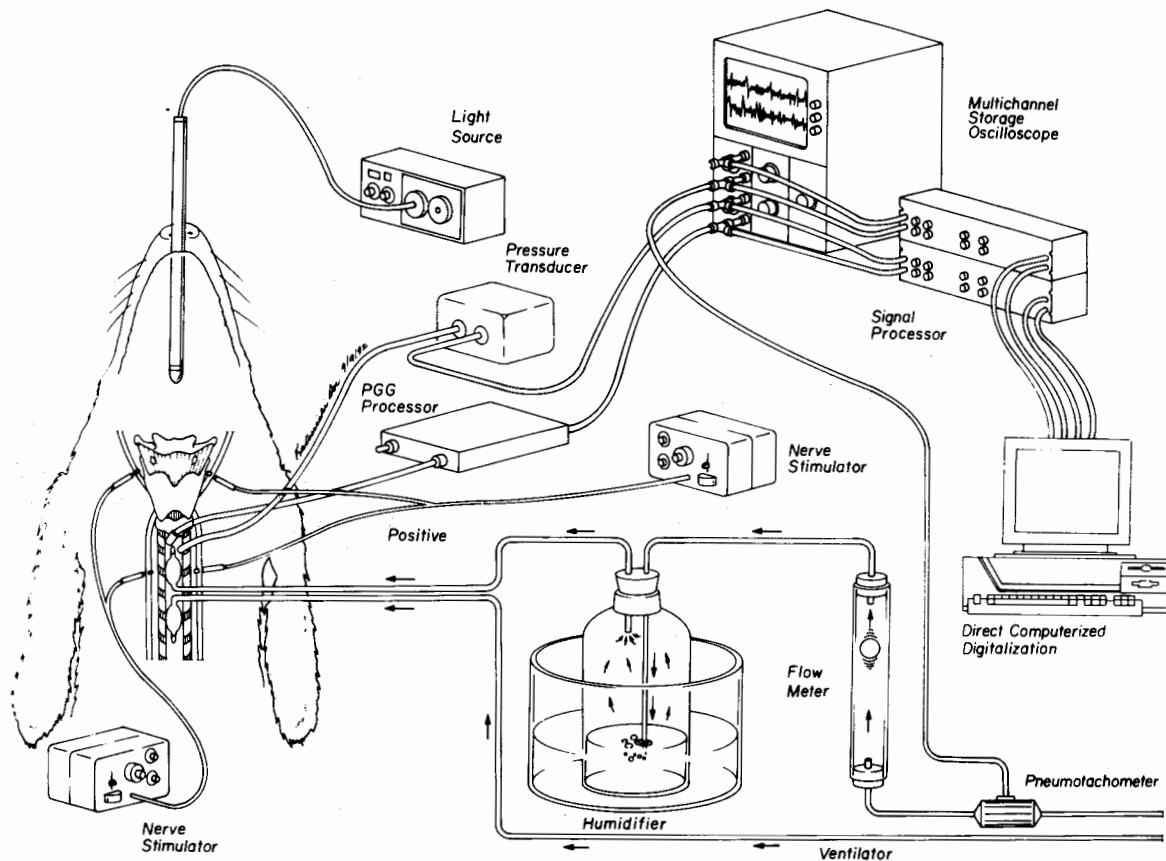


Fig 1. In vivo canine model of phonation.

Multiple trials were made with various levels of airflow. They found that glottal area per cycle increased with increasing levels of airflow, suggesting that the decrease in laryngeal resistance with increasing airflow reported by Smith et al may be due to an increase in glottal area.

Sercarz et al⁸ evaluated the effects of airflow on glottal resistance using the formula for rigid wall resistance:

$$R = k \frac{1}{2} \rho \frac{u}{A^2}$$

in which k = constant, ρ = density, u = airflow, and A = peak glottic area. In that study, they found an increase in glottal resistance with increasing airflow. This is contrary to the concept of flow through a rigid tube, in which increasing flow is always associated with an increase in rigid tube resistance (due to constant area). These findings are explained by the pliable nature of the glottis and the nonlinear relationship of glottal area to resistance.

However, an understanding of laryngeal resistance involves a knowledge of not only the effects of glottal airflow, but also the effects of different levels

of laryngeal nerve activation. No study has evaluated the effects of various levels of both RLN and SLN stimulation on glottal resistance. Previous studies have revealed that an increase in RLN stimulation was associated with a decrease in glottal area per cycle, while an increase in SLN stimulation levels resulted in an increase in glottal area per cycle (Bielamowicz et al, unpublished observations). Also, increasing levels of RLN stimulation resulted in an increased medial adductory compression of the vocal folds.⁹ This suggests that measures of glottal resistance should increase with increasing levels of RLN stimulation and decrease with increasing SLN stimulation. This hypothesis was tested in the present study by measuring changes in subglottal pressure and airflow for various levels of SLN and RLN stimulation.

MATERIALS AND METHODS

Animal Model. The in vivo canine model of phonation is depicted in Fig 1 and has been described in detail in previous reports.¹⁰ Two male mongrel dogs, approximately 25 kg each, were selected for this study. Each animal was premedicated with acepromazine maleate intramuscularly. Intravenous thiopental sodium was administered to a level of corneal anesthe-

TABLE 1. SUBGLOTTAL PRESSURE MEASURED FROM OSCILLOSCOPE AT BEGINNING OF EACH DYNAMIC TRIAL FOR EACH COMBINATION OF RLN AND SLN STIMULATION LEVEL DURING EXPERIMENT 1

SLN Stimulation Level	mA	RLN Level		
		Low	Medium	High
Low	0.5	25	35	55
Medium	0.8	25	35	55
High	1.2	25	35	55

Data are in centimeters of water.
SLN — superior laryngeal nerve, RLN — recurrent laryngeal nerve.

sia. Additional thiopental sodium was used to maintain this level of anesthesia throughout the procedure.

The animal was placed supine on the operating table. A midline incision was made to expose the trachea from the hyoid bone to the sternal notch. A low tracheostomy was performed at the level of the suprasternal notch and cannulated with an endotracheal tube for ventilation. A second tracheostomy was performed superiorly, and a cuffed endotracheal tube was passed in a rostral direction with the tip positioned 10 cm below the vocal folds. The cuff was inflated and air was passed through this rostral endotracheal tube from a laboratory wall outlet. Airflow was humidified and heated by bubbling through 5 cm of heated water so that the temperature of the air was 37°C when measured at the glottal outlet. Upstream subglottal pressure was measured with a catheter-tipped pressure transducer (Millar Instruments, model SPC-330; Houston, Tex). The subglottal pressure transducer was passed rostrally through the superior tracheotomy and placed 2 cm below the glottis.

Airflow was controlled by a valve at the laboratory wall outlet and measured with a U-tube flowmeter (Gilmont Instruments, model F1500; Great Neck, NY) and a pneumotachometer (Fluid Precision Inc, model 183, Billerica, Mass) prior to airflow humidification. Before recording any trials, the pneumotachometer was calibrated with known levels of airflow from the flowmeter. The Gilmont flowmeter allowed macroscopic control of airflow, while the output of the pneumotachometer was digitized and later analyzed by a personal computer.

One-centimeter segments of the RLN and SLN were isolated bilaterally, and Harvard bipolar electrodes (South Natick, Mass) were applied. A constant current nerve stimulator (WR Medical Electronics Co, model S2LH; St Paul, Minn) was used to stimulate the RLN, and a constant voltage source (Grass Instruments, model 54H; Quincy, Mass) was used to stimulate the SLN. These nerves were stimulated at 70 to 80 Hz, with a current of 0.5 to 2.0 mA (RLN) or 0.5 to 1.2 mA (SLN) for 1.5 milliseconds' pulse duration.

A custom photoglottography (PGG) sensor was placed on the trachea, immediately inferior to the larynx. A xenon light source was secured in the oral cavity to provide transglottal light for excitation of the PGG sensor.

Digitization. A 12-bit analog-to-digital converter housed in a personal computer digitized 3 channels: 1) pneumotachometer signal, 2) PGG waveform, and 3) subglottal pressure signal. Signals were low-pass filtered at 3 kHz and digitized at 20 kHz for 2.8 seconds. The subglottal pressure and PGG signals were verified on a Tektronix oscilloscope (model 5116; Beaverton, Ore) before recording.

Research Design. Two experiments examined the effect of airflow on laryngeal resistance by using the ratio of subglottal pressure to airflow as the formula for resistance. Since glottal area measurements were not obtained in the present study, resistance was not calculated from the formula for rigid wall resistance. While some authors have used PGG as a measure of glottal area, we, as well as other authors, have found this method to be unsatisfactory, because light passing through the translucent vocal folds contributes to the PGG waveform.¹¹ This makes accurate location of a baseline difficult. Others have pointed out that the light density distribution within the vocal folds does not remain constant throughout a glottal cycle and may adversely affect the PGG signal.¹²

Because each experiment included a large number of phonatory trials, each experiment was performed on a different dog to prevent fatigue effects. In the first experiment airflow varied dynamically. The second experiment used static flow levels. The static trials were used to evaluate the results obtained in the dynamic trials and to alleviate certain difficulties inherent in the dynamic portion of the experimental design, as discussed below.

EXPERIMENT 1

Method. The profile of laryngeal resistance was examined by independently varying airflow, RLN stimulation, and SLN stimulation while measuring subglottal pressure. Values of laryngeal resistance were calculated by dividing subglottal pressure by airflow. Low, medium, and high RLN and SLN stimulation were provided as described in Table 1. Current was set at 0.5, 0.8, and 1.2 mA for low, medium, and high levels of SLN stimulation. Levels of RLN nerve excitation were controlled to produce periodic vocal fold vibration during each trial. The initial RLN stimulation current was determined by monitoring the subglottal pressure oscilloscope trace. For each level of nerve stimulation, the glottal airflow was set initially at 318 milliliters per second (mL/s).

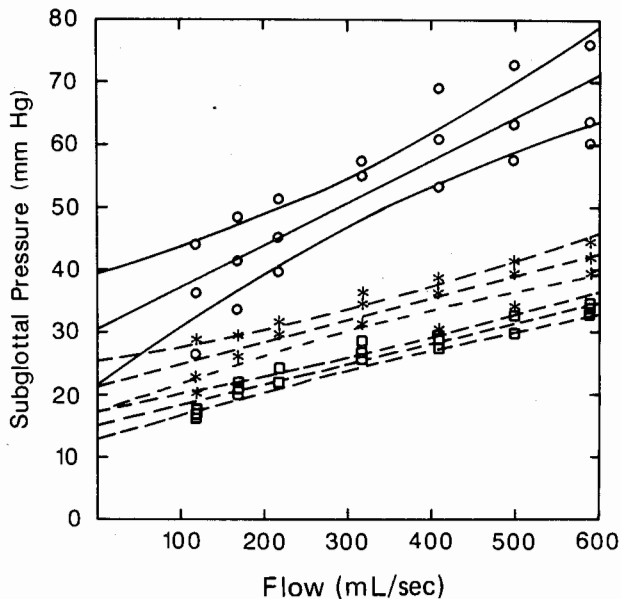


Fig 2. Subglottal pressure versus airflow data for low (squares), medium (asterisks), and high (circles) levels of recurrent laryngeal nerve stimulation. Best fit lines for each level of recurrent laryngeal nerve stimulation are plotted along with curves for 95% confidence intervals.

Current for low levels of RLN stimulation ranged from 0.5 to 0.8 mA as necessary to obtain an initial subglottal pressure of 25 cm H₂O. Medium levels of RLN stimulation varied from 0.8 to 1.5 mA such that the subglottal pressure was 35 cm H₂O at the start of each dynamic trial, while high levels of RLN stimulation produced a subglottal pressure of 55 cm H₂O with 1.5 to 2.0 mA current. After these levels of RLN stimulation were determined for each trial, the RLN level remained constant. The subglottal pressure was allowed to vary freely with changing airflow during each trial. Levels of nerve stimulation were randomized across trials to minimize fatigue effects.

The nerves were stimulated and phonation began. Next, airflow was dynamically increased from 318 mL/s to 590 mL/s by manually increasing flow at the wall outlet over 2.8 seconds, while the stimulation amplitude to the RLN and SLN nerves remained constant. Finally, flow was dynamically decreased from 318 mL/s to 70 mL/s while RLN and SLN stimulation again were held constant. Airflow levels of 70 and 590 mL/s were the end points for the trials because they defined cessation of phonation for this animal. Phonatory trials were obtained for each condition of the experimental design outlined in Table 1. Because of the phonatory fatigue of the dog, multiple trials for all conditions of the experimental design could not be performed.

The subglottal pressure and pneumotachometer signals were analyzed on a personal computer with CSpeech software (Paul Milenkovic, Madison, Wis).

First, the pneumotachometer waveform was used to determine airflow levels of 119, 169, 218, 318, 409, 499, and 590 mL/s by converting voltage output into mL/s via the pneumotachometer calibration curve. Once these flow regions within trials were identified, the subglottal pressure tracing was analyzed for each level of airflow by averaging 5 phonatory cycles around the previously measured region of airflow. Peak subglottal pressures were then plotted against flow for each level of RLN and SLN stimulation. Linear regression curves were then used to fit a line to the data points for each condition, and 95% confidence intervals about each line were calculated to determine the significance of differences between the curves. For each combination of subglottal pressure and airflow, resistance was calculated. Resistance values were then plotted against flow for all combinations of RLN and SLN stimulation.

In addition, the fundamental frequency (F₀) was calculated from the PGG waveform by averaging the period lengths of 10 consecutive cycles of stable phonation in a phonatory trial. Three regions of stable phonation were measured for each trial, and the 30 values were averaged. The F₀ was calculated by obtaining the reciprocal of the average cycle duration.

Results. A 1-way analysis of variance showed no significant effect of SLN stimulation level on subglottal pressure ($F_{2,69} = 0.02$, $p > .05$). Therefore, the data were collapsed across SLN stimulation levels for all subsequent analyses.

Subglottal pressure increased with airflow for the 3 levels of RLN stimulation, as shown in Fig 2. This Figure also includes regression lines with 95% confidence intervals, and reveals a statistically significant increase in the slope of the subglottal pressure versus airflow curves with increasing levels of RLN stimulation. Flow and F₀ were significantly correlated ($r = .65$, $p < .05$). Therefore, multiple regression was used to predict subglottal pressure from flow levels and F₀ values for each level of RLN stimulation. Significant relationships were found for all levels of RLN stimulation (low stimulation: $F_{2,18} = 228.86$, $p < .05$, $R^2 = .96$; medium stimulation: $F_{2,18} = 91.31$, $p < .05$, $R^2 = .91$; high stimulation: $F_{2,18} = 34.25$, $p < .05$, $R^2 = .79$). For low and medium levels of RLN stimulation, flow and F₀ had independent and significant effects on subglottal pressure (Table 2). For the high level of RLN stimulation, effects were apparently not independent, and neither factor had a significant effect on pressure when the other was controlled for, although the overall regression was significant. Thus, both flow and F₀ affect subglottal pressure for low and moderate levels of RLN stimulation. The effects of

TABLE 2. RESULTS OF MULTIPLE REGRESSION ANALYSIS SHOWING EFFECTS OF FLOW AND F0 ON SUBGLOTTAL PRESSURE AT LOW, MEDIUM, AND HIGH RLN STIMULATION

RLN Stimulation	Variable	Co-efficient	Standard Co-efficient	t	p (t)
Low	Constant	9.34	0.00	6.45	<.05
	Flow	0.02	0.49	4.26	<.05
	F0	0.14	0.51	4.49	<.05
Medium	Constant	14.58	0.00	7.98	<.05
	Flow	0.00	0.49	4.23	<.05
	F0	0.14	0.52	4.50	<.05
High	Constant	17.63	0.00	2.39	<.05
	Flow	0.03	0.36	1.26	>.05
	F0	0.19	0.55	1.94	>.05

Both flow and F0 affect subglottal pressure for low and moderate levels of RLN stimulation. Effects of flow and F0 are not separable for high levels of RLN stimulation.

F0 — fundamental frequency, RLN — recurrent laryngeal nerve.

flow and F0 are not separable for high levels of RLN stimulation.

Resistance values decreased as flow increased for each level of RLN stimulation in experiment 1, as shown in Fig 3A. Logarithmic curves provided good

fit (r^2) for each level of RLN stimulation. The RLN stimulation condition had a significant effect on resistance ($F_{2,54} = 17.17, p < .05$). Scheffé comparisons showed that resistance was significantly greater for the high RLN stimulation condition than for the low or medium stimulation conditions. However, the low and medium RLN stimulation conditions did not differ significantly. As suggested by the findings above, the SLN stimulation level had no significant effect on resistance ($F_{2,54} = 0.21, p > .05$), and no RLN level by SLN level interaction was observed ($F_{4,54} = 0.12, p > .05$).

EXPERIMENT 2

Method. Because of concerns over the compressibility of nonideal gases and the compliance of the airflow delivery system during the dynamic trials, the results of experiment 1 were confirmed with static airflow trials. The static trials were also designed to eliminate any possible confounding effects of adjusting the RLN stimulation level at the beginning of the dynamic trials in experiment 1 by the oscilloscope trace of the subglottal pressure. A second dog was used in this experiment, for the in vivo model described in experiment 1.

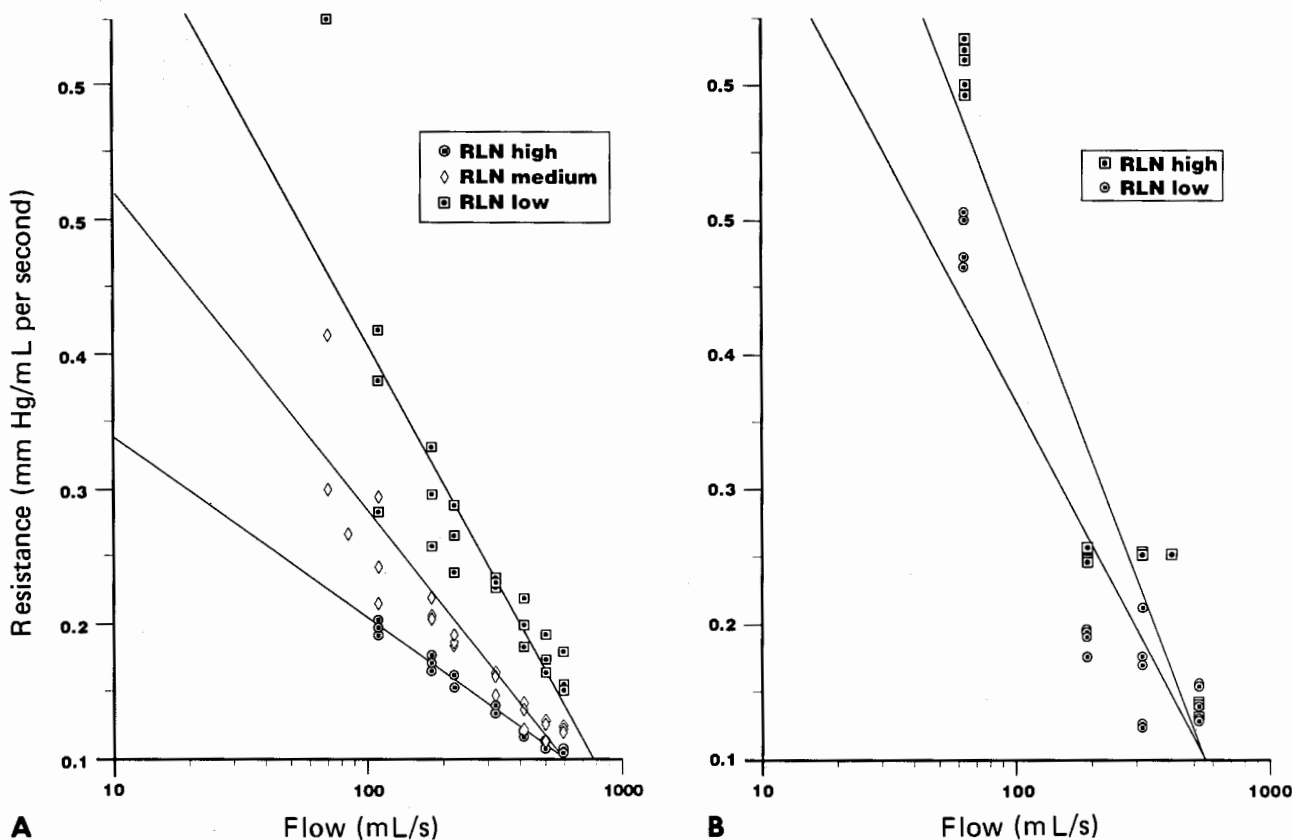


Fig 3. Best fit logarithmic curves for resistance versus airflow. A) For 3 levels of recurrent laryngeal nerve (RLN) stimulation in experiment 1. Correlation coefficients (r^2) for low, medium, and high RLN stimulation are .979, .857, and .786, respectively. B) For 2 levels of RLN stimulation in experiment 2. Correlation coefficients (r^2) for low and high RLN stimulation are .814 and .903, respectively.

A constant level of airflow was confirmed throughout each trial with the Gilmont flowmeter. The RLN and SLN stimulation levels were also held constant within a trial. Current for the low SLN stimulation condition was set at 0.7 mA; current for the high SLN stimulation condition was set at 1.5 mA. The RLN stimulation levels ranged from 0.6 to 0.8 mA for the low stimulation condition, and from 1.3 to 1.5 mA for the high stimulation condition. A narrow range of current was needed to achieve stable phonation in this animal model. The subglottal pressure oscilloscope tracing was not used in this experiment. Flow was set at 63, 183, 318, or 523 mL/s.

Three trials were performed for each experimental condition. However, no trials were performed at a flow level of 318 mL/s when RLN and SLN stimulations were set at high levels because of equipment failure at the end of the experiment. As in experiment 1, the PGG signal was used to determine the F₀ for each trial.

• **Results.** A 2-way analysis of covariance examined the effects of RLN and SLN stimulation on subglottal pressure in the second dog while controlling for the effects of F₀. As above, increasing RLN stimulation produced significant increases in pressure ($F_{1,46} = 4.44, p < .05$). No significant effect of SLN stimulation was observed ($F_{1,46} = 0.58, p > .05$), and no interaction occurred ($F_{1,46} = 0.67, p > .05$). The F₀ did not significantly covary with subglottal pressure in this experiment ($F_{1,46} = 1.32, p > .05$), so F₀ was not included in subsequent analyses.

Linear regression was used to examine the relationship between pressure and flow for each level of RLN stimulation. Subglottal pressure increased significantly with flow for both RLN stimulation conditions (low stimulation: $F_{1,28} = 52.91, p < .05, r^2 = .65$; high stimulation: $F_{1,19} = 24.69, p < .05, r^2 = .57$).

Figure 3B shows that increasing RLN stimulation resulted in an increase in glottal resistance in experiment 2. Logarithmic best fit curves showed a high degree of correlation (r^2) for the resistance versus airflow plots. The RLN stimulation conditions differed significantly ($F_{1,47} = 4.47, p < .05$). The SLN stimulation condition did not significantly affect resistance ($F_{1,47} = 0.32, p > .05$), and no interaction effect was observed ($F_{1,47} = 0.01, p > .05$). These findings confirm the results of experiment 1.

As seen in Fig 3, resistance versus flow curves for the different RLN stimulation conditions converged at approximately 0.1 mm Hg per mL/s as airflow increased. This trend was apparent for both dynamic and static trials. Above this value of glottal resis-

tance, stable phonation ceased regardless of the level of RLN stimulation, SLN stimulation, or airflow.

DISCUSSION

This study examined the effects of airflow, RLN stimulation, and SLN stimulation on laryngeal resistance during phonation by measuring the effect of these variables on subglottal pressure. Dynamic trials provided resistance versus airflow curves of the *in vivo* canine model of phonation for various RLN and SLN stimulation conditions. Static trials confirmed the effects of airflow, RLN stimulation, and SLN stimulation on laryngeal resistance found in the dynamic trials.

As previously reported, subglottal pressure increased linearly with increasing airflow when RLN and SLN stimulation were held constant.⁷ The resistance versus airflow curves (Fig 3) demonstrated a decrease in glottal resistance with increasing airflow in both the dynamic and static trials. These findings are consistent with data from the excised canine laryngeal studies of Muta and Fukuda,⁵ who reported a decrease in laryngeal resistance with increasing levels of airflow. The anatomic changes associated with increasing levels of airflow are best explained by the area study of Sercarz et al.⁸ They discovered that the glottal area per cycle of phonation increased and glottal resistance decreased with increasing levels of airflow. They speculated that this was due to a greater dilation of the subglottal vault prior to the opening of the glottis with increasing levels of airflow. In addition, Sercarz et al.⁸ noted that increasing airflow resulted in an increase in F₀ due to a shorter closed period during phonation. The authors hypothesized that this finding was due to a decrease in the time required to reach the phonatory threshold pressure, ie, the minimum subglottal pressure required to overcome the glottal closing forces and thus produce glottal opening for each cycle of phonation.

Fundamental frequency increased with increasing airflow in both experiments reported here. This relationship between airflow and F₀ is consistent with data reported by Smith et al.⁷ They found that the primary determinant of increasing F₀ with increasing airflow was due to the effect of increasing subglottal pressure with increasing airflow. In 1989, Titze¹³ provided theoretic formulas that predicted the linear effect of increasing subglottal pressure on vibratory frequency of the vocal folds. He stated that the effect of subglottal pressure on F₀ resulted from the vocal folds' having "amplitude-dependent frequency" due to the nonlinearity introduced by the dynamic stiffness of the vocal folds. His experimental data from excised canine larynges strongly support the hypoth-

esis that F0 increases with increasing subglottal pressure.

Previous studies have shown that vocal fold thickness decreases and glottal area per phonatory cycle increases with increasing SLN stimulation¹⁴ (also Bielamowicz et al, unpublished observations). On the basis of these data, we anticipated a decrease in glottal resistance with increasing SLN stimulation. However, variations in SLN stimulation had no statistically significant effects on glottal resistance during phonation in the present study. The limited effects of SLN stimulation on glottal resistance are consistent with the findings of Hirano,¹⁴ who reported that SLN stimulation levels had no significant effects on glottal area. The present study contradicts the *in vivo* canine study of Koyama et al,⁶ who found that increasing levels of SLN stimulation tended to increase glottal resistance. However, their conclusions were not supported by statistical analysis.

The effects of various levels of RLN stimulation on glottal resistance have not been studied previously. Increasing levels of RLN stimulation caused a statistically significant increase in the glottal resistance. A previous study has shown that glottal area per cycle of phonation decreased with increasing levels of RLN stimulation (Bielamowicz et al, unpublished observations). We hypothesize that this narrowing of the glottal opening caused an increase in glottal resistance during phonation.

The present results support the collapsible tube model of phonation described by Berke et al¹⁵ in 1991. While the collapsible tube model of vocal fold vibration has not been universally accepted, we believe it provides a useful framework for understanding our results. This model uses measures of external pressure (eg, laryngeal muscle medial adductory forces), downstream pressure drop (eg, transglottal pressure gradient), and elasticity (eg, vocal fold stiffness) to explain why oscillation occurs at moderate flow rates but not at very low or high rates of flow. Berke et al found that spontaneous oscillations begin in the larynx with increasing airflow after the closing forces of the fixed glottal resistor are overcome. During this range of airflow, the glottis is seen as a partially collapsed tube. As airflow increases, the

glottis dilates and the pressure drop across the glottis (ie, laryngeal resistance) decreases. Given constant muscular contraction and tissue elasticity, vibration ensues. The collapsible tube model of phonation predicts that increases in airflow will result in an increased vibratory frequency and a decreased glottal resistance (eg, pressure drop), as reported here. This suggests that at low flow rates a higher resistance system exists when the glottis is narrowed, while high flow results in a distended glottis and a lower resistance to airflow. The tube also continues to dilate with increased flow, approaching a circular configuration at very high flow rates.

The collapsible tube model of phonation further predicts that increasing stimulation to the RLN (ie, increasing external pressure on the tube) will produce an increase in glottal resistance at low flow rates, but at high flow rates glottal resistance measures would converge toward the same value despite the extent of laryngeal muscular contraction. Consistent with this theory, all resistance versus airflow curves from this study approached 0.1 mm Hg per mL/s at the highest levels of airflow. Levels of airflow greater than 590 mL/s resulted in the cessation of phonation as the glottis obtained a circular configuration. Therefore, a resistance of 0.1 mm Hg per mL/s defines the minimum level of glottal resistance required for stable phonation, regardless of the level of SLN or RLN stimulation.

CONCLUSION

This study evaluated pressure-flow relationships during phonation in the *in vivo* canine model of phonation. We found that an increase in airflow resulted in an increase in subglottal pressure, a decrease in laryngeal resistance, and an increase in F0. Increasing levels of SLN stimulation were not found to have a statistically significant effect on laryngeal resistance. However, increasing RLN stimulation caused a significant increase in the glottal resistance for each level of flow tested. In addition, despite the RLN stimulation used, measures of glottal resistance with increasing airflow converge toward the same value. These findings are consistent with the collapsible tube model of phonation.

ACKNOWLEDGMENTS — The authors thank Dr Ming Ye, Dr Hong-Shik Choi, and Manuel Natividad for their technical assistance.

REFERENCES

1. Smitheran JR, Hixon TJ. A clinical method for estimating laryngeal airway resistance during vowel production. *J Speech Hear Disord* 1981;46:138-46.
2. Netsell R, Lotz W, Shaughnessy AL. Laryngeal aerodynamics associated with selected voice disorders. *Am J Otolaryngol* 1984;5:397-403.
3. Tanaka S, Gould WJ. Vocal efficiency and aerodynamic aspects in voice disorders. *Ann Otol Rhinol Laryngol* 1985;94:29-33.
4. Shipp T, Izdebski K, Schutte HK, Morrissey P. Subglottal air pressure in spastic dysphonia speech. *Folia Phoniatr (Basel)* 1988;40:105-10.
5. Muta H, Fukuda H. Pressure-flow relationships in the experimental phonation of excised canine larynges. In: Fujimura

A, ed. Vocal physiology: voice production, mechanisms and functions. New York, NY: Raven Press, 1988:239-47.

6. Koyama T, Kawasaki M, Ogura J. Mechanics of voice production: regulation of vocal intensity. *Laryngoscope* 1969;79:337-54.
7. Smith ME, Green DC, Berke GS. Pressure-flow relationships during phonation in the canine larynx. *J Voice* 1991;5:10-7.
8. Sercarz JA, Berke GS, Bielamowicz SA, Kreiman J, Ye M, Green DC. Changes in glottal area associated with increasing airflow. *Ann Otol Rhinol Laryngol* (in press).
9. Berke GS, Hanson DG, Gerratt BR, Trapp TK, Macagba C, Natividad M. The effect of airflow and medial adductory compression on vocal efficiency and glottal vibration. *Otolaryngol Head Neck Surg* 1990;102:212-8.
10. Berke GS, Moore DM, Hanson DG, Hantke DR, Gerratt BR, Burstein F. Laryngeal modeling: theoretical, in vitro, in vivo. *Laryngoscope* 1987;97:871-81.
11. Hartmann W, Wullstein H. Untersuchungen über den Bewegungsvorgang an den schwingenden Stimmklappen von Kehlkopfpräparaten mit verbesserter Photozellenmethode. *Arch Ohren Nasen Kehlkopfheilkd* 1938;144:348-60.
12. Coleman RF, Wendahl RW. On the validity of laryngeal photosensor monitoring. *J Acoust Soc Am* 1968;44:1733-5.
13. Titze IR. On the relation between subglottal pressure and fundamental frequency in phonation. *J Acoust Soc Am* 1989;85:901-6.
14. Hirano M. Phonosurgery: basic and clinical investigations. *Otol Fukuoka (Jibi To Rinsho)* 1975;21:239-40.
15. Berke GS, Green DG, Smith ME, Arnstein DP, Honrubia V, Natividad M. Experimental evidence in the in vivo canine for the collapsible tube model of phonation. *J Acoust Soc Am* 1991;89:1358-63.