

A pressure-regulated model of normal and pathologic phonation

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Recent evidence suggests that the lung-thorax system functions as a constant pressure source during phonation. However, previous animal models used a constant flow source. This article describes an in vivo canine model that maintains a constant subglottic pressure during phonation to more closely simulate the pulmonary system. At any given subglottic pressure, increasing levels of recurrent laryngeal nerve stimulation resulted in a significant rise in resistance followed by a plateau. Increasing levels of superior laryngeal nerve stimulation, however, produced no significant change in glottal resistance. Three experimental conditions were studied: normal, unilateral recurrent laryngeal nerve paralysis, and paralysis followed by arytenoid adduction. In normal canines, maximal vocal efficiency values were the highest, indicating the best match between pressure and resistance. The vocal efficiency values were significantly lower in recurrent laryngeal nerve paralysis, indicating pressure-resistance mismatch. Arytenoid adduction increased the maximal vocal efficiency values and decreased the mismatch observed in the paralyzed state. These findings may provide insight into an understanding of normal and pathologic laryngeal behavior. (OTOLARYNGOL HEAD NECK SURG 1994;111:807-15.)

The majority of laryngeal research with animal models has used flow-regulated systems, in which the flow generated by the input source is treated as an independent variable and the resulting subglottic pressure (P_{sub}) is treated as a dependent variable.¹⁻⁴ One possible reason for this approach was that experiments were easier to perform with a constant flow model, in both live animals and excised larynges. Currently, many researchers believe that a constant pressure driving source is more physiologic and a better approximation of actual laryngeal function.

In this study a constant pressure source was studied in an in vivo canine phonation model for the first time.

Thus far, support for the constant pressure model has been largely theoretical. In the two mass laryngeal models proposed by Ishizaka and Flanagan,⁵ a constant pressure in the lungs was used to approximate the P_{sub} . Later, Warren and coworkers^{6,7} stated that pressure regulation was the primary goal of the lung-thorax system so that driving forces above a minimal level were provided to the vocal tract.

Further evidence for pressure regulation has been provided by experiments that use interlabial leak tubes.⁸ Flow through the leak tube increased with the cross-sectional area of the tube, suggesting that pressure was being regulated. This was the first attempt at experimental use of the constant pressure model. However, the authors did not investigate laryngeal function in this pressure-regulated system.

Although evidence points toward a pressure-regulated speech production mechanism, its physiology is poorly understood. To study the relationship in the larynx among aerodynamic and neuromuscular variables with a constant air pressure system, we designed a new in vivo canine phonation model. This design permitted independent control of many variables governing phonation. Simply stated, the constant pressure model is more physiologic because it

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Supported by the Veterans Affairs Merit Review Funds and a National Institute on Deafness and Other Communication Disorders grant (no. R01 DC00855-01).

Presented at the Research Forum at the Annual Meeting of the American Academy of Otolaryngology-Head and Neck Surgery, San Diego, Calif., Sept. 18-21, 1994.

Received for publication March 2, 1994; accepted July 8, 1994.

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23/10/58970

maintains a constant P_{sub} to simulate the function of the pulmonary system during speech.

We investigated the phonatory characteristics under three conditions: normal, unilateral recurrent laryngeal nerve (RLN) paralysis, and paralysis followed by arytenoid adduction. The arytenoid adduction was used to medialize the paralyzed vocal fold. If a constant pressure model is to be of value to laryngologists, it should be able to provide insight into both normal and pathologic laryngeal behavior. Therefore this project was designed to provide a model for examining laryngeal function in unilateral vocal fold paralysis, before and after treatment.

METHODS AND MATERIAL

In Vivo Canine Model

This study was performed in accordance with the Public Health Service Policy on Humane Care and Use of Laboratory Animals, the National Institutes of Health 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 of the University of California, Los Angeles.

Methods are based on those of prior in vivo canine studies.¹⁻³ Six adult male mongrel dogs, each weighing 25 to 30 kg, were used in this study. Each dog was anesthetized with acepromazine maleate intramuscularly. Intravenous sodium pentobarbital was administered to a level of corneal anesthesia throughout the procedure.

The animals were placed supine on an operating table, and a midline incision was made from the hyoid bone to the sternal notch (Fig. 1). A distal tracheotomy was performed at the level of the suprasternal notch, and an endotracheal tube was inserted for ventilation. A proximal tracheotomy was also performed, and a cuffed endotracheal tube was passed in a rostral direction with the tip positioned 10 cm below the glottis. This tube was attached to the pressure-regulating system.

The external branches of the superior laryngeal nerves (SLNs) were isolated at their entrance into the cricothyroid muscle. Both RLNs were isolated 5 cm inferior to the larynx. Harvard bipolar electrodes (South Natick, Mass.) were applied. Electrical isolation between RLNs and SLNs was verified by direct observation of the laryngeal muscles during stimulation of each nerve.

A Grass nerve stimulator (model 54H; Quincy, Mass.) was used to provide variable voltage stim-

ulation to both SLNs. A second stimulator (model 2SLH; WPI Medical Electronics Co., St. Paul, Minn.) was used to provide a constant current stimulus to the RLNs. The frequency of stimulation was 80 Hz, with a pulse duration of 1.5 milliseconds for both stimulators.

The arytenoid adduction was performed as described by Isshiki et al.⁹ The thyroid cartilage on one side was exposed to the posterior margin. A 3-0 nylon suture through the muscular process was passed to the anterior thyroid lamina with a 16-gauge angiocatheter. The tension on the stitch was adjusted to the maximum acoustic intensity.

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. The exit port of the pressure-regulating system was connected to a Gilmont flowmeter (model f1500; Gilmont Instruments, Great Neck, N.Y.). Airflow was humidified and heated by bubbling through 5 cm H₂O so that the temperature of the air was 37°C when measured at the glottal outlet.

Pressure-regulating System

The pressure-regulating system consisted of a cylindrical container measuring 75 cm in height and 42 cm in diameter (Fig. 1). A hollow T-shaped tube with an inner diameter of 1.5 cm was placed within the cylinder and attached to the inside of the cylinder. The tip of the vertical axis of the T was 2.5 cm above the floor of the cylinder. 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. Water pressure was measured with a 100-cm ruler attached to the inner surface of the cylinder. With this configuration, pressure buildup above the adjusted water level would be vented through the submerged tip of the T tube, resulting in a constant P_{sub} . As soon as bubbles appeared because of vented air, the pressure was maintained at a constant level. This constant pressure level is equal to the distance between the bottom of the vertical axis and the water surface in centimeters of H₂O.

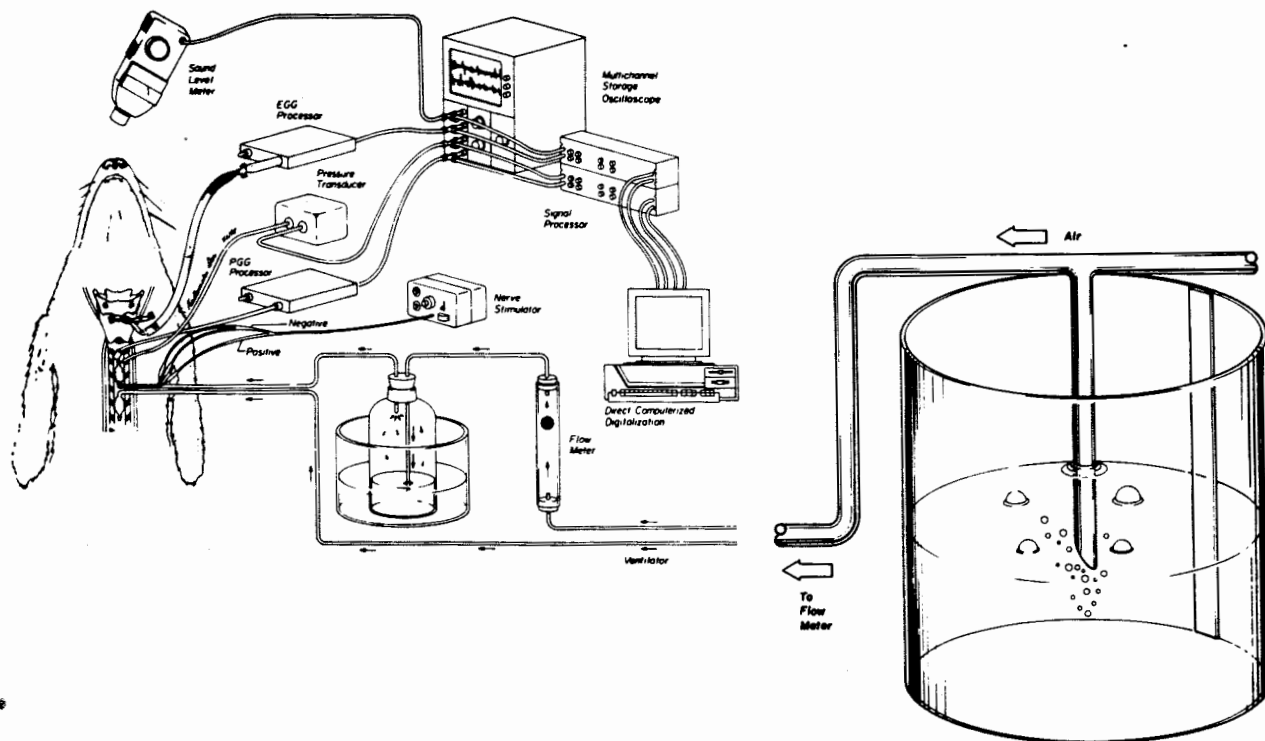


Fig. 1. Schema of experimental setup with constant-pressure driving source. EGG, Electroglottography; PGG, photoglottography.

Experimental Design

In three animals (dogs 1 through 3), target values of P_{sub} ranged from 25 to 70 cm H₂O and varied by canine. Phonation was produced by stimulating the RLN at multiple 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 per subject ranged from 103 to 222. Each trial lasted 5 seconds. RLN stimulation steps and the order of target pressures were randomized.

In the other three animals (dogs 4 through 6), the experiment was done under normal conditions, paralysis, and paralysis followed by arytenoid adduction. Target values of P_{sub} ranged from 45 to 65 cm H₂O in steps of 10 cm H₂O (45, 55, and 65 cm H₂O). Two levels of SLN stimulation were used: low (0.5 mV) and high (0.8 mV). For the normal condition, both RLNs were stimulated. For paralysis, the RLN was stimulated only on one side to simulate acute unilateral vocal fold paralysis. Immediately after this evaluation, arytenoid adduction was performed on the side not stimulated. During phonation after the

arytenoid adduction, only the RLN opposite the side of adduction was stimulated.

Acoustic and Aerodynamic Measures

The P_{sub} and acoustic signals were low-pass filtered at 3 kHz and digitized at 10 samples per second with a 12-bit analog-to-digital converter (Scientific Solutions, Solon, Ohio). The data were analyzed with C-Speech software for IBM (Paul Milenkovic, University of Wisconsin, Madison, Wis.). The vocal efficiency was calculated as described before.

Following Yanagi et al.,¹⁰ vocal efficiency is the ratio of the acoustical power (P_r) to aerodynamic power (P_a): Vocal efficiency = P_r/P_a . P_r is calculated with the following formula:¹¹ $P_r = 2r^2P_e^2/P_0c$. This formula applies to the sound power radiating with no directivity into a hemisphere of area $2r^2$, where r is the distance away from the source. P_0c is the specific acoustic impedance and equals 41.1 dynes \times second/cm³ in air at 20° C. P_e is the root-mean-square sound pressure in dynes per cubic

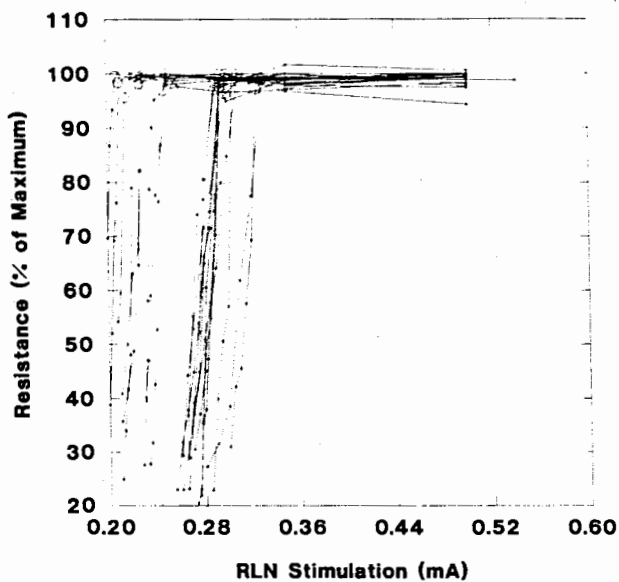


Fig. 2. Resistance (cm H₂O/cm³/sec) vs. level of RLN stimulation in a representative canine.

centimeter at a distance r from the sound source. P_a is the product of flow and the P_{sub} .

RESULTS

Laryngeal Airway Resistance (Dogs 1 Through 3)

Laryngeal airway resistance is calculated as the ratio of P_{sub} to flow. Laryngeal airway resistance increased significantly with increasing RLN stimulation (dog 1: $F [1, 56] = 51.70, p < 0.01, r^2 = 0.48$; dog 2: $F [1, 105] = 46.31, p < 0.01, r^2 = 0.31$; dog 3: $F [1, 82] = 170.78, p < 0.01, r^2 = 0.68$) until a plateau was reached at a point corresponding to the flow minimum (Fig. 2, open circles). Once this peak resistance was reached, phonation ceased, and further increases in RLN stimulation produced no further changes in resistance (dog 1: $F [1, 43] = 2.91, p > 0.01$; dog 2: $F [1, 113] = 3.27, p > 0.01$; dog 3: $F [1, 61] = 1.42, p > 0.01$); (dog 1: $F [1, 7] = 2.67, p > 0.01$; dog 2: $F [1, 19] = 96.91, p < 0.01, r^2 = 0.84$; dog 3: $F [1, 13] = 27.19, p < 0.01, r^2 = 0.68$). Results from dog 1, although not statistically significant, are in the same direction as those for dogs 2 and 3. Lack of statistical significance may reflect the relatively limited number of trials and thus the small number of degrees of freedom for this dog. As subglottic pressure increased, so did the resistance level at which this peak occurred. Similarly, as P_{sub} increased, so did the amount of RLN stimulation needed to achieve peak resistance (dog 1: $F [1, 7] = 50.78, p < 0.01, r^2 = 0.88$; dog 2: $F [1,$

19] = 39.82, $p < 0.01, r^2 = 0.68$; dog 3: $F [1, 13] = 95.12, p < 0.01, r^2 = 0.88$).

Normal, Paralysis, and Postadduction Data (Dogs 4 Through 6)

Vocal efficiency is the ratio of subglottic power to aerodynamic power and is a function of intensity, flow, and P_{sub} . A four-way analysis of variance (ANOVA) examined differences among dogs, P_{sub} s (45, 55, or 65 cm H₂O), SLN stimulation levels, and experimental conditions (normal, paralyzed, and postadduction) in overall levels of vocal efficiency. Significant main effects were found for all independent variables (dog: $F [2, 562] = 110.65, p < 0.01$; SLN stimulation: $F [1, 562] = 81.66, p < 0.01$; subglottic pressure: $F [2, 562] = 100.17, p < 0.01$; experimental condition \times subglottic pressure: $F [4, 562] = 6.19, p < 0.01$). However, these effects are small, and each accounts for less than 1.8% of the variance in the underlying data. Accordingly, interaction terms will not be discussed further here. Note that although each dog was used as its own control in this study, a repeated-measures analysis could not be performed because of differences in both the number of steps and the overall levels of RLN stimulation used for the different conditions.

Figure 3 shows differences in peak vocal efficiency with P_{sub} and experimental condition for a representative set of trials in a canine. A three-way repeated measures ANOVA (dog \times subglottic pressure \times experimental condition, with repeated measures on experimental condition) showed that peak efficiency varied significantly with experimental condition ($F [2, 18] = 359.33, p < 0.01$). Planned comparisons showed that all three experimental conditions differed significantly ($p < 0.01$) from one another, with the highest peak efficiencies in the normal condition, the lowest in the paralyzed condition, and intermediate values for the arytenoid adduction condition. No significant effects of P_{sub} or dog on peak efficiency were observed (subglottic pressure: $F [2, 9] = 5.79, p < 0.01$; dog: $F [2, 9] = 5.35, p < 0.01$), and no significant interactions occurred.

A similar repeated-measures ANOVA examined the effects of dog, subglottic pressure, and experimental condition on the minimum vocal efficiency at which phonation occurred. A significant main effect of experimental condition was again observed ($F [2, 18] = 401.53, p < 0.01$), with all conditions differing significantly from one another (planned comparison, $p < 0.01$). Minimum efficiency was highest for the normal condition, lowest for the paralyzed

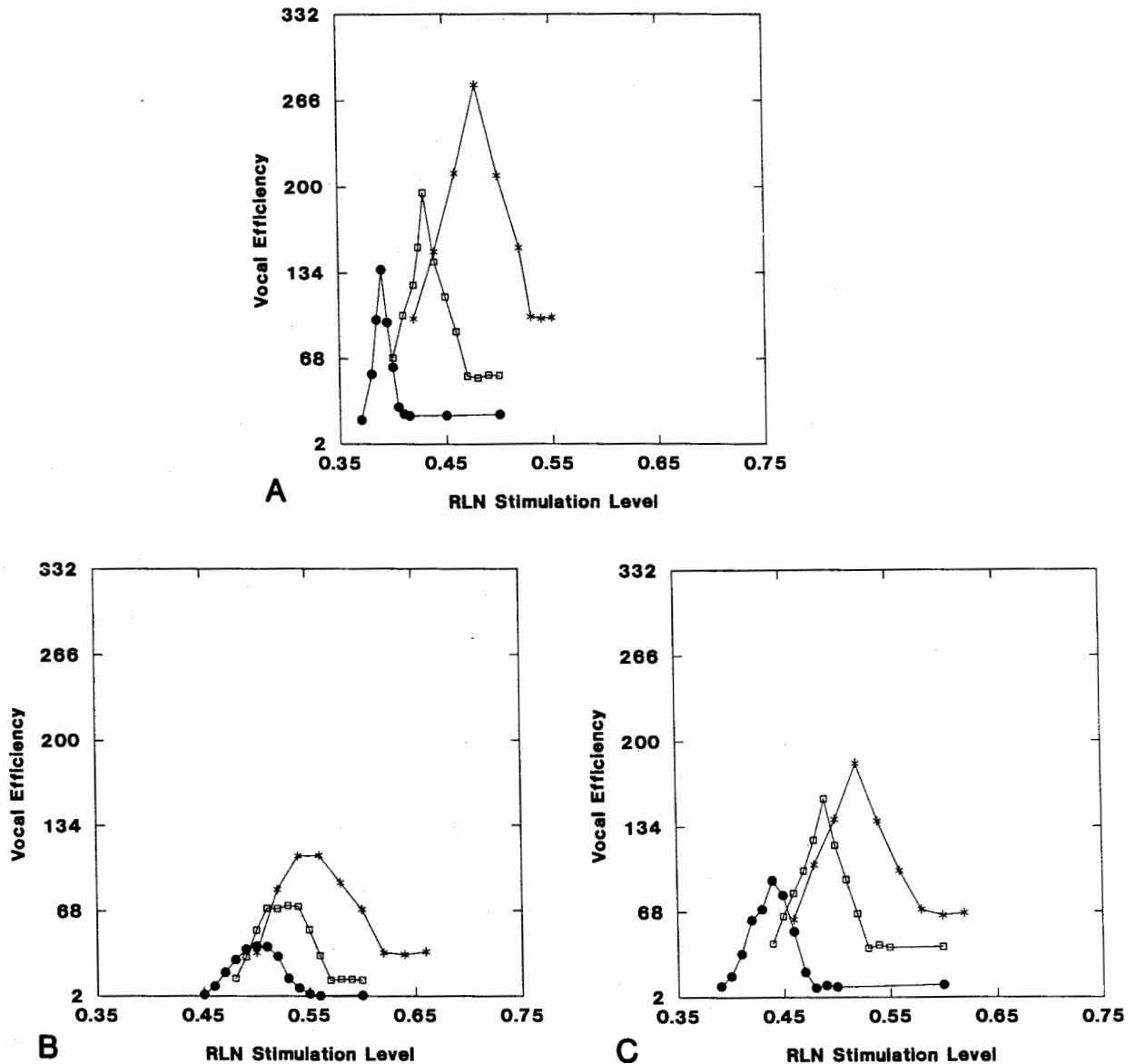


Fig. 3. Vocal efficiency (%) vs. RLN stimulation level (mA) at three levels of P_{sub} in a representative animal. **A**, Normal laryngeal phonation; **B**, unilateral vocal fold paralysis; **C**, paralysis followed by arytenoid adduction. From left to right curve within each panel (**A**, **B**, and **C**), P_{sub} levels are 45, 55, and 65 cm H_2O .

condition, and intermediate for the postadduction condition.

In normal laryngeal phonation, vocal efficiency increased with RLN stimulation until a peak was reached. This peak corresponded with optimal phonation characterized as loud and clear. After the peak, increasing RLN stimulation produced a decrease in vocal efficiency until a plateau was reached (Fig. 3A). In paralysis, the vocal efficiency increased with increasing RLN stimulation level until a plateau was reached (maximal vocal efficiency). This

plateau corresponded with optimal phonation. After the plateau, with increasing RLN stimulation level, there was a decrease in vocal efficiency until a second (lower) plateau was reached (Fig. 3B). After adduction, the overall shape of the vocal efficiency vs. the RLN stimulation curve was similar to the normal curve except that the peak vocal efficiency value was smaller than that of normal laryngeal phonation (Fig. 3C). Figure 4 shows vocal efficiency vs. RLN stimulation level curves in a representative animal under three experimental conditions at the

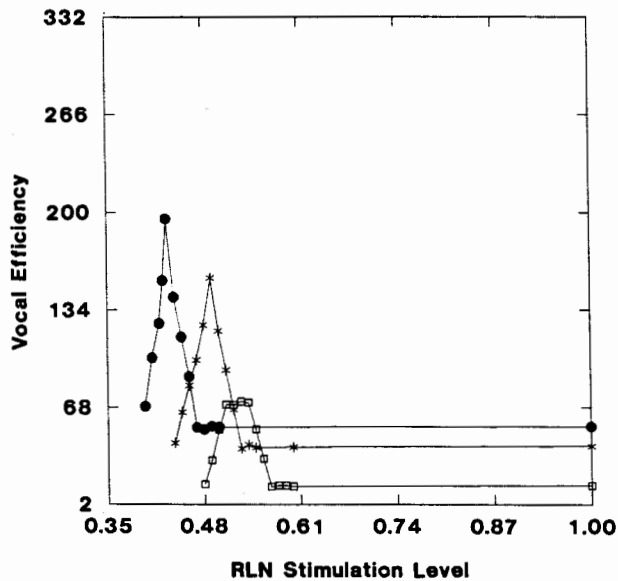


Fig. 4. Vocal efficiency (%) as a function of RLN stimulation level (mA) at 55 cm H₂O P_{sub} under normal (left), paralysis (right), and arytenoid adduction (middle) experimental conditions. Circles are data points for paralysis, squares are data points for postadduction, and asterisks are data points for normal conditions.

P_{sub} level of 55 cm H₂O. This figure demonstrates the differences in shape of vocal efficiency curves under three experimental conditions.

Within each experimental condition, the amount of RLN stimulation necessary to generate peak efficiency varied significantly with P_{sub} (normal: F [1, 16] = 19.35, $p < 0.01$, $r^2 = 0.55$; paralyzed: F [1, 16] = 16.40, $p < 0.01$, $r^2 = 0.51$; postadduction: F [1, 16] = 10.37, $p < 0.01$, $r^2 = 0.39$). Similarly, the RLN stimulation level at which efficiency plateaued varied significantly with P_{sub} (normal: F [1, 16] = 27.38, $p < 0.01$, $r^2 = 0.64$; paralyzed: F [1, 16] = 16.76, $p < 0.01$, $r^2 = 0.51$; postadduction: F [1, 16] = 17.98, $p < 0.01$, $r^2 = 0.53$).

DISCUSSION

We have demonstrated several interesting features of phonation with the constant pressure system. This model treats the lung-thorax subunit as a large reservoir with relatively small outflow resistance. The pressure provided to the subglottis would therefore be determined by the inherent aerodynamic and mechanical properties of the pulmonary source. On the other hand, muscular activity within the larynx could produce a resistance much greater than that of the lung-thorax outflow. Changes in the laryngeal resistance would serve to regulate the flow through the larynx.

It appears that in a normally innervated larynx, muscular contraction can be adjusted to maintain maximal efficiency of phonation. In paralysis, the ability to adjust resistance in the setting of varying P_{sub} is impaired. Therefore, optimal phonation cannot be achieved. After treatment for paralysis (arytenoid adduction in this experiment), there is better control of laryngeal resistance, which improves the response to changing P_{sub}s and results in more efficient phonation.

Situated between the trachea and the pharynx, the larynx acts as a biomechanical valve offering opposition to the flow of air.¹² This vocal tract resistance may be calculated from the ratio of translaryngeal pressure (subglottic minus supraglottic pressure) to the translaryngeal flow. Although supraglottic pressure varies instantaneously with laryngeal vibration, it is approximately equal to atmospheric pressure. The resistance may therefore be calculated by use of the following equation: Resistance = Subglottic pressure/Translaryngeal flow.

The concept of laryngeal resistance is actually more complex than this formula suggests.¹³ The airflow velocity distribution across the larynx and changes in the cross-sectional area of the glottis are the main determinants of the pressure losses within the larynx. The vena contracta or the area of sudden narrowing within the larynx leads to pressure loss. Flow separation and the creation of turbulence result in pressure recovery. However, the formula above is a close approximation of the resistance changes that occur in the vocal tract as a whole.

The formula above was used to calculate the vocal tract resistance in this study. The resistance did not change significantly with increasing levels of SLN stimulation. In constant flow models, Berke et al.¹ found no elevation in P_{sub} with increases in SLN stimulation. They and others also concluded that SLN stimulus level had little effect on vocal tract resistance.^{4,14,15} In these studies, the vocal folds remained closed for a shorter portion of each vibratory cycle because their compliance was reduced by increasing the level of SLN stimulation.¹⁵ The change in the geometry of the glottis after contraction of the cricothyroid muscles could account for this change in compliance.

Increasing levels of RLN stimulation in this study resulted in a rise in resistance followed by a plateau. This is also consistent with findings of previous studies that used a constant flow source.^{2,4} Two factors may account for this plateau. First, the posterior chink in the canine vocal fold may act as a shunt, limiting the degree to which muscular con-

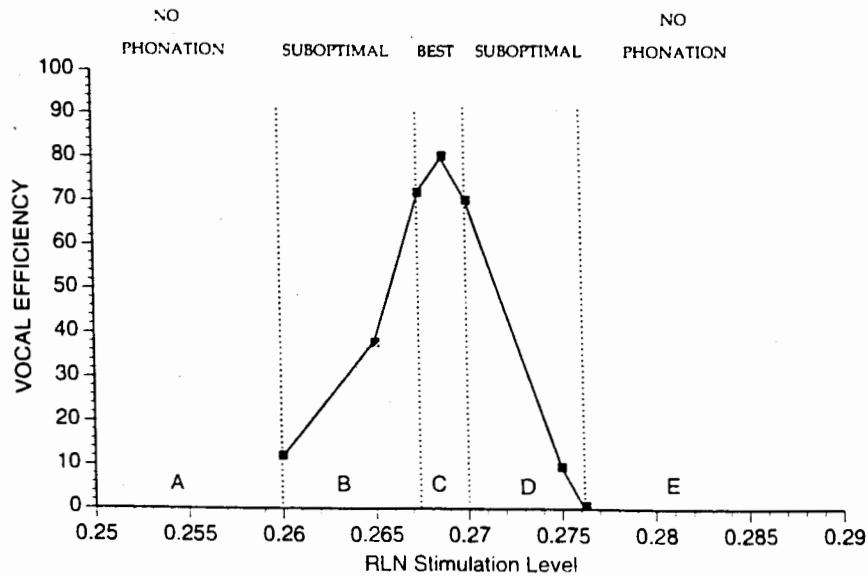


Fig. 5. Five modes of phonation (A through E) in a representative graph of percent vocal efficiency vs. RLN stimulation level in milliamperes.

traction can raise resistance. Further, because of the inherent elastic properties of the vocal folds, there is some degree of air escape at any given P_{sub} . This elastic modulus increases with increasing muscular contraction and then plateaus. Thus the posterior glottal chink and the plateau of the elastic modulus of the folds could account for the plateau in resistance.

Vocal efficiency is an objective measure of the voice, first described by Van den Berg in 1956.¹⁶ Clinically, vocal efficiency has been shown to be useful in voice evaluation and to decrease with some forms of laryngeal disease, such as invasive carcinoma and vocal cord paralysis.^{17,18} This investigation suggested that for each level of SLN stimulation and pressure, increasing levels of RLN stimulation resulted in a normal distribution of the vocal efficiencies (Fig. 5). The value of RLN stimulation at which peak efficiency was achieved also corresponded to the value at which the most subjectively clear phonation was produced. Any deviation from this stimulation level produced a reduction in vocal efficiency and a deterioration in vocal quality.

The pattern of rising and falling vocal efficiency in this experiment appears to describe five "modes" of phonation (Fig. 5, regions A-E). At any level of P_{sub} and SLN stimulation, a minimum amount of RLN activity was required to induce phonation. Below this minimum level, no phonation occurred (Fig. 5, region A). In region A, P_{sub} had not reached a constant level because low levels of vocal tract resistance precluded development of a P_{sub} large enough

to overcome the pressure of the water in the cylinder.

Once P_{sub} reached a constant level, phonation began (region B), although its quality was perceptually weak, hoarse, and breathy. The vocal efficiency was correspondingly low. After this point, any increase in RLN stimulation produced a fall in flow and a concomitant fall in needed aerodynamic power. This fall in aerodynamic power led to an increase in vocal efficiency.

In general, sound intensity remained constant until maximal vocal efficiency was reached (region C) and phonation became maximally clear and strong. As RLN stimulation increased above this level, sound intensity began to fall at a faster rate than the fall in flow (region D). Consequently, the incremental reduction in acoustic power was greater than that of the aerodynamic power, and vocal efficiency began to decline. As RLN stimulation increased still further, intensity continued to fall, whereas flow stabilized at a constant minimum level. Above this level of stimulation, no phonation occurred (region E).

From the previous discussion it follows that a normal larynx is capable of optimizing phonation at any given P_{sub} . With an increase in P_{sub} in this study, there was a shift to a curve with a higher peak vocal efficiency. This was achieved by adjusting the laryngeal resistance and consequently the intensity and the flow. Fant¹⁹ has shown that the primary determinant in increasing acoustic intensity is P_{sub} . To increase intensity in a normal person, pulmonary

driving force or P_{sub} must be increased. However, if this were done without a concomitant rise in laryngeal resistance, flow would increase, and vocal efficiency would fall. To maintain peak vocal efficiency, laryngeal resistance must be altered. Stated differently, there is an optimal range of laryngeal resistance at which vocal efficiency is maximized. Efficient phonation therefore involves a match between P_{sub} and laryngeal resistance.

In unilateral vocal fold paralysis, there was a marked change in the vocal efficiency curve. Compared with the normal condition, the curve was shifted to the right, implying that higher levels of RLN stimulation were required to produce phonation. The vocal efficiency curve rose to a maximum level, reached a plateau, and then fell. These maximum vocal efficiency values in paralysis were much lower than those of normal.

It appears that in unilateral vocal fold paralysis, because of inadequate medial fold compression and an excess airflow leak, patients are unable to adjust the resistance with changes in P_{sub} . In addition, they can only phonate over a very limited range of P_{sub} . Therefore, they cannot switch to a more efficient curve. This results in a pressure-resistance mismatch and production of sound at suboptimal efficiencies.

After arytenoid adduction, the vocal efficiency curve demonstrated a rise followed by a fall, similar to the shape of the vocal efficiency curve under normal conditions. In this study, arytenoid adduction was performed because it produced a reliable change in glottal configuration after RLN paralysis. Compared with the paralysis curve, the arytenoid adduction curve was shifted back to the left, but not as far back to the left as the normal curve. The maximal vocal efficiency value was increased compared with that of paralysis, but it was not as large as that of the normal curve.

Therefore, arytenoid adduction results in improved optimal phonatory efficiency compared with unilateral vocal fold paralysis. This is most likely because of medial positioning of the paralyzed vocal fold, which allows adjustment of laryngeal resistance to achieve a more efficient phonation. Stated differently, in comparison to paralysis, after arytenoid adduction a better match of P_{sub} and laryngeal resistance is attained.

These patterns of vocal efficiency variation may be clinically important because there seems to be a relationship between vocal efficiency and laryngeal aerodynamic and biomechanical elements. In various forms of vocal fold medialization (Teflon injection, type I thyroplasty, arytenoid adduction, and RLN

reinnervation), adjusting the position of the paralyzed hemilarynx results in a more complete medial fold compression and less airflow leak. Hence the larynx regains some of its ability to adjust its resistance with changes in P_{sub} by shifting to more efficient phonatory curves. However, because the paralyzed hemilarynx is fixed at midline as the result of the treatment, the maximal vocal efficiency is not as high as that of the normal larynx. This constant pressure study has produced insight into the biomechanical behavior of normal and pathologic phonation and has increased understanding the effects of surgery to improve voice.

CONCLUSION

This new model of phonation with a constant pressure source enables us to fit the lung-thorax system into the scheme of the laryngeal phonation. It appears that in normal phonation maximal vocal efficiency values are the highest, indicating the best match between pressure and resistance. The vocal efficiency values are decreased significantly after paralysis, indicating pressure-resistance mismatch. Arytenoid adduction increases the maximal vocal efficiency values and decreases the mismatch observed in the paralyzed state. These findings may be used in the future to design phonosurgical procedures to maximize vocal efficiency, minimize pressure-resistance mismatch, and optimize phonation.

REFERENCES

1. Berke GS, Moore DM, Gerratt BR, Hanson G, Natividad M. Effect of superior laryngeal nerve stimulation on phonation in an *in vivo* canine model. *Am J Otolaryngol* 1989;10:181-7.
2. Berke GS, Moore DM, Gerratt BR, Hanson DG, Bell TS, Natividad M. The effect of recurrent laryngeal nerve stimulation in an *in vivo* canine model. *Laryngoscope* 1989;99:977-82.
3. Moore DM, Berke GS. The effect of laryngeal nerve stimulation on phonation: a glottographic study using an *in vivo* canine model. *J Acoust Soc Am* 1988;83:705-15.
4. Bielowicz SA, Berke GS, Kreiman J, Sercarz JA, Green DC, Gerratt BR. Effect of tension, stiffness, and airflow on laryngeal resistance in the *in vivo* canine model. *Ann Otol Rhinol Laryngol* 1993;102:761-8.
5. Ishizaka K, Flanagan JL. Synthesis of voiced sounds from a two mass model of the vocal cords. *Bell Sys Tech J* 1972;51:1233-68.
6. Warren D. Compensatory speech behaviors in individuals with cleft palate: a regulation/control phenomenon? *Cleft Palate J* 1986;23:251-60.
7. Warren D, Dalston R, Morr K, Hairfield M, Smith L. The speech regulating system: temporal and aerodynamic responses to velopharyngeal inadequacy. *J Speech Hear Res* 1989;32:566-76.
8. Putnam A, Shelton R, Kastner C. Intraoral air pressure and

- oral airflow under different bleed and bite block conditions. *J Speech Hear Res* 1986;29:37-49.
9. Isshiki N, Tanabe M, Sawada M. Arytenoid adduction for unilateral vocal cord paralysis. *Arch Otolaryngol* 1978;104:555-8.
 10. Yanagi E, Slavit DH, McCaffrey TV. Study of phonation in the excised canine larynx. *OTOLARYNGOL HEAD NECK SURG* 1991;4:586-95.
 11. Koyama T, Harvey JE, Ogura JH. Mechanics of voice production. III. Efficiency of voice production. *Laryngoscope* 1971;81:210-7.
 12. Smitheran JR, Hixon TJ. A clinical method for estimating laryngeal airway resistance during vowel production. *J Speech Hear Disorders* 1981;46:138-46.
 13. Tully A, Brancatisano A, Loring SH, Engel LA. Relationship between thyroarytenoid activity and laryngeal resistance. *J Appl Physiol* 1990;68:1988-96.
 14. Tully A, Brancatisano A, Loring SH, Engel LA. Influence of posterior cricoarytenoid muscle activity on pressure-flow relationship of the larynx. *J Appl Physiol* 1991;70:2252-8.
 15. Slavit DH, Lipton RJ, McCaffrey TV. Phonatory vocal fold function in the excised canine larynx. *OTOLARYNGOL HEAD NECK SURG* 1990;6:947-56.
 16. Van den Berg JW. Direct and indirect determination of the mean subglottic pressure. *Folia Phoniatr (Basel)* 1956;8:1-24.
 17. Iwata S. Aerodynamic aspects of phonation in normal and pathologic larynges. In: Fujimura O, ed. *Vocal physiology: voice production, mechanisms, and functions*. New York: Raven Press, 1988:423-31.
 18. Tanaka S, Gould WJ. Vocal efficiency and aerodynamic aspects in voice disorders. *Ann Otol Rhinol Laryngol* 1985;94:29-33.
 19. Fant G. I. *Speech production. A. Preliminaries to analysis of the human voice source*. *STL-QPSR* 1982;4:1-27.

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