The effect of air flow and medial adductory compression on vocal efficiency and glottal vibration

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This study used an in vivo canine model to investigate the effects of varying vocal fold resistance by electrically stimulating the recurrent laryngeal nerve while monitoring medial adductory compression of the vocal folds, glottal airflow, and vocal intensity. The effects of increasing airflow on glottal vibration were also examined stroboscopically and by measurement of open quotient. The results indicated that increasing intensity by medial adductory compression was more efficient than by increasing airflow. Increasing airflow produced a significantly greater open quotient and vocal fold vibratory excursion. (OTOLARYNGOL HEAD NECK SURG 1990;102:212.)

The study of the factors affecting vocal intensity is complicated by difficulty in measuring all the variables of interest. Most often, investigators have measured subglottal pressure (Psub), glottal airflow (U), and vocal intensity (I). Vocal fold resistance has not usually been measured directly, but rather has been calculated from these other measures. This study, using an in vivo canine model, investigated the effects of varying vocal fold resistance by electrical stimulation of the recurrent laryngeal nerve (RLNS) while monitoring medial adductory compression (MAC) of the vocal folds, glottal airflow, and vocal intensity.

BACKGROUND

Van den Berg[1] was one of the first investigators to emphasize the importance of subglottic power in vocal intensity, which he estimated as the product of mean subglottic pressure and mean flow rate. Rubin et al.[2] used a tracheal puncture technique to study the effect of Psub and U on intensity. They concluded that subglottic pressure had a much greater effect on intensity production than airflow, which had little or no effect.

Isshiki[3] reported that resistance or subglottic pressure had the greatest effect on intensity at low fundamental frequencies (F_0s), whereas at high frequencies airflow had the greater effect. Koyama et al.,[4] using an in vivo canine model, concluded that airflow was more important than RLNS in controlling the intensity of voice production. They assumed that RLNS was directly proportional to laryngeal stiffness. Other authors have not concurred with their conclusions.[2,3,5]

Timcke et al.[6] reviewed the relationship of open quotient (OQ) to intensity of voice production. Using high-speed cinematography, they found that as open quotient (i.e., the proportion of time the glottis was open within each glottal cycle) decreased, vocal intensity usually increased.

The above studies and others[7,8] appear to have assumed that resistance of the vocal folds is proportional to vocal fold stiffness, implying a linear relationship of resistance to airflow. One aim of this study is to reexamine the validity of this assumption.

Previous in vivo animal studies have demonstrated that, under conditions of steady subglottic flow, increase in electrical stimulation to the recurrent laryngeal nerves was associated with induced phonation of a higher pitch and greater loudness.[5,9] This study examined effects of variation in airflow and electrical stimulation of the laryngeal nerves on intensity and open quotient.

METHODS

In vivo preparation. Mongrel dogs were anesthetized with an intramuscular injection of 2 ml ketamine, followed by intravenous pentobarbital titrated to loss

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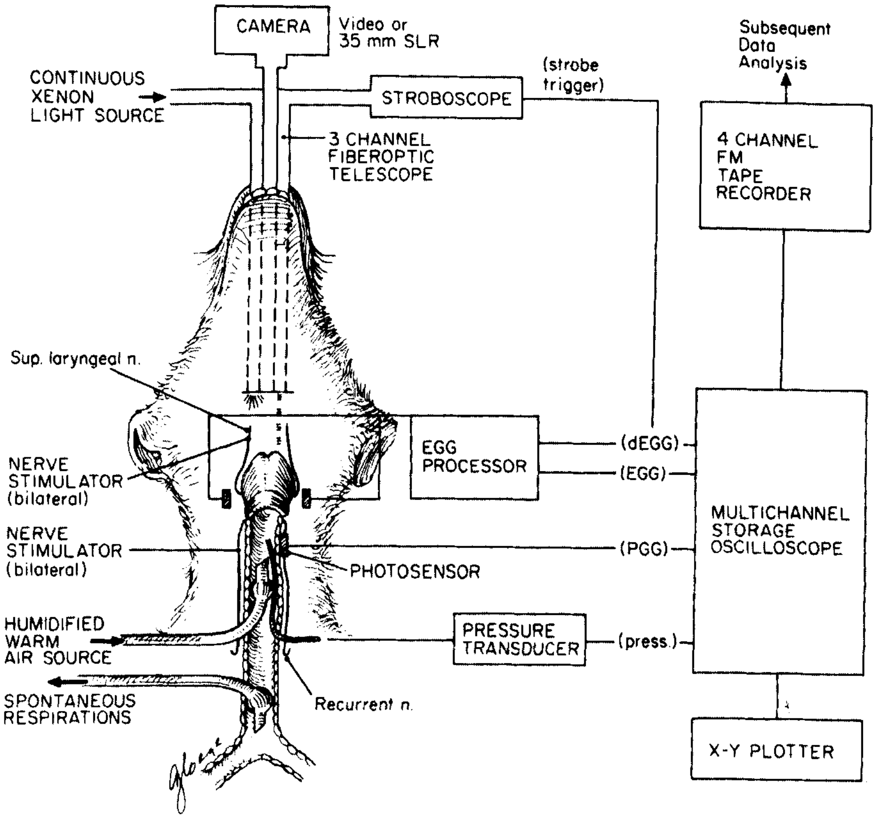

Fig. 1. An in vivo canine preparation allowed simultaneous monitoring of video stroboscopy, photoglottography, electroglottography, and subglottic pressure and airflow.

of the corneal reflex. The animals were placed in supine position on an operating table (Fig. 1) and direct laryngoscopy was performed to confirm normal laryngeal anatomy. A 7-mm oral endotracheal tube was inserted, through which the animal breathed spontaneously. Through a vertical midline incision, the strap muscles and sternocleidomastoid muscles were retracted laterally to expose the larynx and trachea. The external branches of the superior laryngeal nerves were isolated at their entrance into the cricothyroid muscle. Harvard bipolar electrodes were applied to the nerves. The recurrent laryngeal nerves were isolated 5 cm inferior to the larynx, and bipolar electrodes were applied. A suture through the thyrohyoid membrane was used to suspend the epiglottis anteriorly to improve visualization of the vocal folds. A distal tracheotomy was made for placement of an endotracheal tube to permit the animal to breathe spontaneously. An additional proximal tracheotomy was performed through which a cuffed tracheotomy tube was placed, with its tip resting 10 cm below the glottis. The cuff of the superiorly directed tube was inflated to just seal the trachea. Room air was bubbled through 5 cm H_2O at 37° C for warming and humidification and passed through the cephalad tracheotomy tube. The temperature in the animal's trachea was measured at 15-minute intervals to assure a constant air temperature of 37° C.

A Grass model 54H stimulator (Grass Instruments, Quincy, Mass.) provided variable voltage stimulation to both recurrent laryngeal nerves. A second (WPI 301-T) stimulator was used to provide a low level of constant current stimulus for the superior laryngeal nerves. Voltages ranged from 0.5 to 0.9 volts for the Grass stimulator. Currents ranged from 0.1 to 0.15 mA for the WPI stimulator. Frequency of stimulus was 80 Hz, with a pulse duration of 1.5 msec for both the Grass and WPI units.

Medial adductory compression. Medial adductory compression (MAC) of the vocal folds was measured by a device consisting of a small soft polyethylene (1 × 3 × 3 mm) H_2O-filled sac connected to a 2-mm diameter catheter. The device was calibrated against a mercury manometer with a pediatric blood pressure transducer for pressure levels from 10 to 90 mm Hg.

The device was held between the arytenoids and vocal processes, with the catheter arising from the sub-

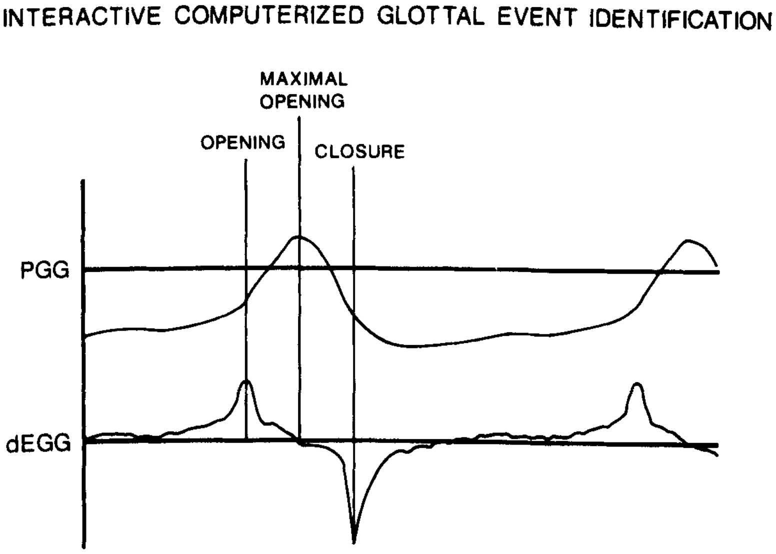

Fig. 2. Open quotient was calculated from simultaneously obtained photoglottography and the first derivative of electroglottography. The points of opening and closing were marked at respective peaks in increasing and decreasing velocity of the EGG impedance signal. Peak glottic area was estimated from the peak voltage of the photosensor signal. Open quotient was then calculated as the percentage of the glottal cycle during which the vocal folds were open.

glottic tracheostomy. A measurement was taken before and after each trial to ascertain a relative gauge of MAC.

Photoelectric, intensity, and pressure measurements. A photosensor (Centronics OSD 50-2, Mountainside, N.J.) was placed on the animal's trachea approximately 3 cm below the larynx. A halogen flashlight provided supraglottic illumination for photoglottography (PGG). A microphone (Sennheiser, Old Lyme, Conn.) was placed 15 cm from the vocal folds and connected to a Storz model 8000 laryngostroboscope (Storz, Culver City, Calif.) for frequency analysis of the induced phonation. The stroboscope source was connected with a fiberoptic cable to a 0-degree Storz telescope for observation of vocal fold vibratory excursion.

Electroglottography (EGG) electrodes (Synchrovoice, Briarcliff Manor, N.Y.) were placed in direct contact on either side of the thyroid cartilage while the reference electrode was sutured to the skin.

A catheter-tipped pressure transducer (Millar model #SPC 330, Houston, Texas), inserted through the upper tracheotomy, rested 2 cm below the glottis. The transducer was calibrated at the temperature of the animal's trachea by submerging the transducer in a water bath at 37° C to a depth just covering the sensor (0.5 cm) and then calibrating it against a manometer from 0 to 120 cm H_2O pressure.

Intensity was measured with a linear scale sound level meter (Quest Electronics Model #208L, Oconomowoc, Wis.) positioned 1 m from the anterior canine. Rotation of the sound level meter at constant radius, 1 m from the animal's mouth, showed less than a 2-dB fluctuation in intensity, indicating isotropic sound radiation.

PGG, EGG, Psub, and MAC signals were digitized at 20 kHz using a 12-bit A/D board and a 16-bit personal computer microprocessor. The signals were monitored on oscilloscopes (Tektronix 5116, Beaverton, Ore.) and (Hitachi V1050-F, Carson, Calif.). Files were stored on disk, and a 0.5-second sample of stable phonation was used for analysis. A multipurpose computer software program was used to choose points of opening and closing by using the differentiated EGG.[10] Peak opening was chosen using the peak of the PGG (Fig. 2). Twenty-five consecutive cycles were used to calculate a mean open quotient for each trial.

Experimental design. The first part of this study compared the effects of airflow vs. laryngeal nerve stimulation on intensity. Four related experiments were performed on five animals.

1. Subglottal air flow was provided at a constant 318 cc/second. Superior laryngeal nerve stimulation was set to sustain half maximal contraction of the cricothyroid muscle at 0.1 mA. Voltage was increased to the recurrent laryngeal nerve until phonation ensued and then voltage was increased in steps to result in change in F_0 of 20 Hz per increment. Intensity was measured at each F_0 increment (Fig. 3).

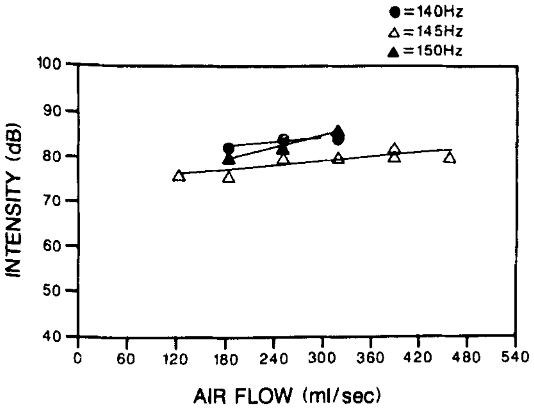

Fig. 3. Intensity measured in dB SPL is plotted against rate of subglottal airflow as fundamental frequency was varied by change in voltage of stimulation of the recurrent laryngeal nerves ($n = 2$).

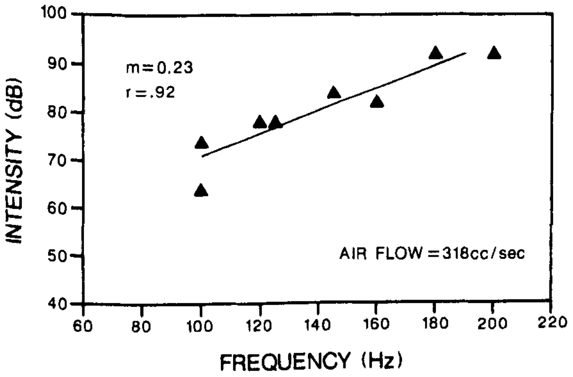

Fig. 4. Intensity in dB SPL is plotted against F_0 at a constant airflow of 318 cc/second; m = slope, r = Pearson's correlation, $n = 1$.

2. Stimulus voltage to the recurrent laryngeal nerves was held constant at 0.5 volts, and stimulation to the superior laryngeal nerves was held at a constant current of 0.1 mA. Subglottal airflow was increased by increments of 60 cc/second from 120 to 480 cc/second, and the F_0 and intensity of the induced phonation were measured (Fig. 4).
3. Stimulus voltage to the recurrent laryngeal nerves was varied in 0.1-volt increments, and MAC and intensity of phonation were measured. This experiment was performed at three different rates of subglottal flow (150, 300, and 450 cc/second). It was observed that equal increments of increasing voltage did not correspond to proportional increases in MAC. This was the result of impedance fluctuation from electrode-nerve connection or nerve-muscular fatigue. For this experiment,

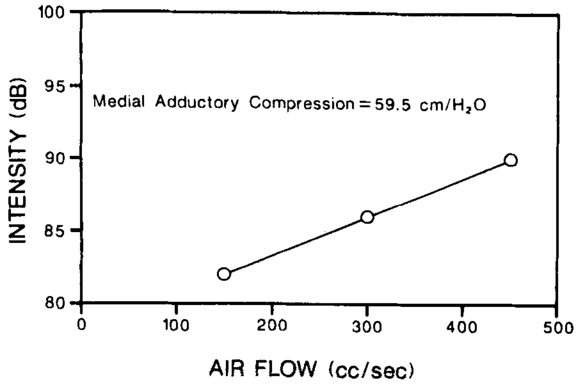

Fig. 5. At a constant medial adductory pressure, intensity in dB SPL is plotted against three levels of subglottal air flow ($n = 1$).

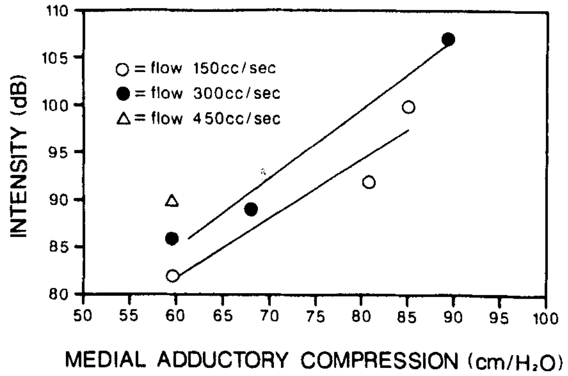

Fig. 6. For three different flow rates, intensity in dB SPL is plotted against mean adduction pressure measured between the vocal folds ($n = 1$).

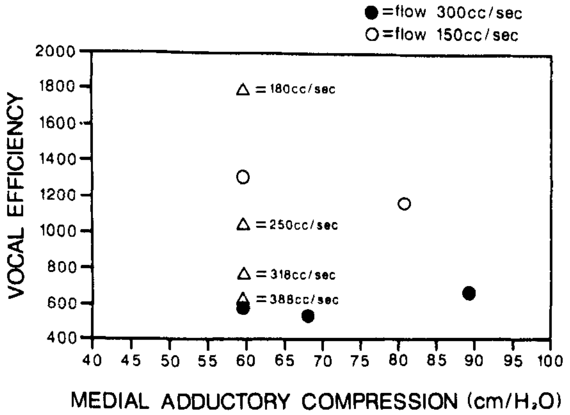

Fig. 7. The *open triangles* demonstrate vocal efficiency for four rates of subglottic flow (180, 250, 318, and 388) at a 59.5 cm H_2O constant medial adductory pressure. The *open circles* and *closed circles* represent vocal efficiency, with variation in medial adductory pressure at subglottic flow rates of 150 and 300 cc/second, respectively ($n = 2$).

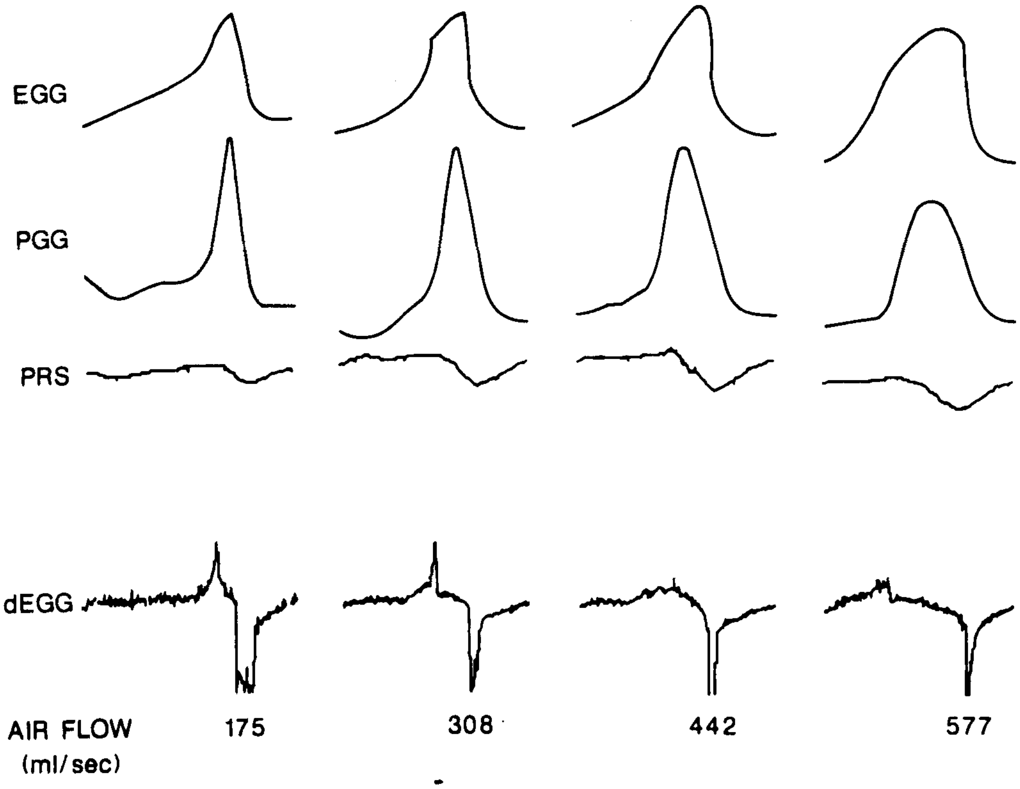

Fig. 8. Representative samples of signals, representing impedance (EGG, with increasing impedance up), light transillumination (PGG, increasing light up), subglottic pressure (PRS), and the first derivative of the EGG signal are demonstrated for four subglottal flow rates. The duration of the open period increased with rate of flow.

phonation at 450 cc/second was elicited solely at an MAC of 59.5 cm H_2O (Figs. 5, 6, and 7).

4. MAC was maintained constant at 59.5 cm H_2O by stimulation of the recurrent laryngeal nerves at 0.7 volts. Subglottal airflow was varied (180, 250, 318, and 450 cc/second) while intensity was measured (Fig. 7).

Vocal efficiency, the ratio of the radiated intensity of production to the subglottic power, was calculated from experiments 3 and 4 using the following equation:

$$(I \times R^2 \times 4\,pi)/(U \times Psub)$$

in which I = intensity (dB), R = radius (cm), U = mean flow (cc/sec), and Psub = mean subglottic pressure (cm H_2O).

The second part of this study examined the effect of airflow on open quotient. MAC was held constant at 59.5 cm H_2O. This was done at a constant superior laryngeal nerve stimulation of 0.1 mA and recurrent laryngeal nerve stimulation of 0.4 to 0.8 volts. Airflow was varied in four levels (175, 308, 442, and 577 cc/second). Seven animals were studied. Not all preparations produced phonation at 175 cc/second, so analysis was limited to the three flow rates of 308, 442, and 577 cc/second.

RESULTS

Fig. 3 shows the experimental effect of varying airflow at a constant level of RLNS. There was little change in I or F_0 for increasing levels of flow. In contrast, Fig. 4 demonstrates that at a constant level of U (318 cc/second), increasing F_0 by greater RLNS produced approximately a 25-dB increase in I. A similar comparison is seen in Figs. 5 and 6. Fig. 5 demonstrates the effect of increasing flow while monitoring constant medial adductory compression at 59.5 cm H_2O. As U was increased from 150 to 450 cc/second, I increased by only 8 dB. Fig. 6 shows the effect of increasing MAC at three different rates of air flow (150, 300, and 450 cc/second). MAC showed a profound influence on the intensity of production during constant flow.

Fig. 7 shows that at a constant level of MAC (59.5 cm H_2O), increasing levels of U (180, 250, 318, and 388 cc/second) were associated with a decrease in the efficiency of vocal production *(triangles)*. When flow was held constant at 150 cc/second *(open circles)* or 300 cc/second *(closed circles)*, increasing MAC had relatively less effect on vocal efficiency. These data indicated that producing intensity by increasing medial adductory compression was more efficient than by increasing airflow.

Fig. 8 shows signals from simultaneously obtained EGG, PGG, subglottic pressure, and differentiated EGG in a typical experiment for four levels of airflow at constant MAC. It was observed that as airflow increased, there was a longer glottal open period. Subglottic pressure changed relatively little as flow was increased.

Fig. 9 shows mean data for seven experiments. Open quotient significantly increased with greater airflow (ANOVA for repeated measures within subjects, $p < 0.001$; F[1,12] = 30.84). Post hoc Neuman-Keuls testing indicated that all levels were significantly different. Stroboscopic examination of the glottis at constant MAC demonstrated an increase in the lateral excursion of the vocal folds with increasing flow.

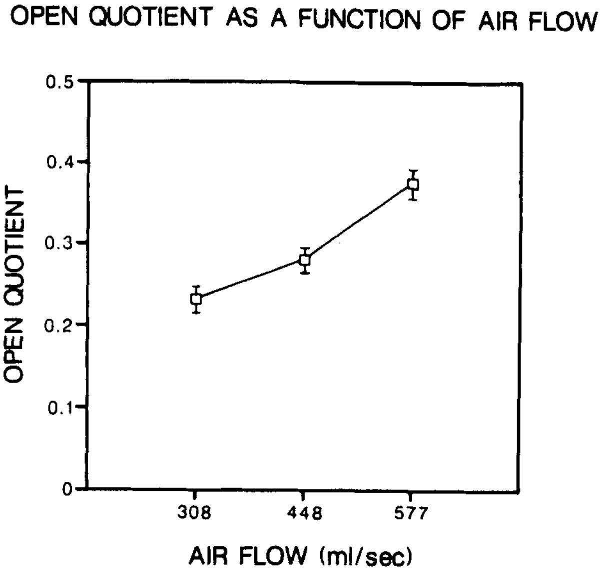

Fig. 9. The *open* and *line bars* represent mean data and standard deviation, respectively, for variation in open quotient for three magnitudes of subglottal airflow for seven subjects.

DISCUSSION

Little if any increase in subglottic pressure was observed when flow was increased during constant MAC. In contrast, greater RLNS significantly increases subglottic pressure.[11] Open quotient increased with greater airflow in this study, whereas OQ decreased with greater RLNS.[11]

Results of this study indicate that the phonating larynx may be analogous to a small balloon filling with air each cycle until it bursts, leading to resealing and refilling again. Once the stiffness of the walls of the balloon are overcome and it begins to fill, the pressure within the balloon stays constant and is independent of the rate of airflow used to inflate it. Furthering this analogy, it appears that greater airflow (U) can inflate the balloon to a larger circumference (OQ) without changing the intraballoon pressure (Psub).

Fant[12] has pointed out that the primary factor involved in determining voice intensity is the lung or subglottic pressure. This occurs because of the increase in the mean particle velocity produced by the transference of potential energy from the elevated subglottic pressure to the kinetic energy of the molecules in the supraglottic airflow jet. Two additional covarying factors include increase in the velocity of vocal fold closing caused by increased medial adductory compression and an increase in the fundamental frequency or pulse repetition. These intensity-related factors are a function of laryngeal nerve stimulation, but not of airflow.

The living innervated animal model of phonation used for this study appears to be closer to the physiology of normal human phonation than would be expected of excised larynges or other models with fixed mechanical properties of the aperture walls. During phonation induced in these experiments, the resistance of the vocal folds varied, even though the measured medial adduction compression was kept constant. The pressure measured as MAC probably resulted from a number of forces, including adduction of the vocal processes, intrinsic muscular contraction of the thyroarytenoid, and linear tensile stretch of the vocal fold tissues. It seems, however, that MAC represents a reasonable indirect measure of muscular effort that results in resistance to flow of air through the glottis.[13] As flow increased at an otherwise steady state of medial adductory compression, subglottic pressure was observed to remain constant. This necessarily implies a decrease in the dynamic resistance of the vocal fold walls in response to greater airflow. Stroboscopy of the vocal fold movements indicated that the relative stability of subglottic pressure as subglottic flow increased was associated with greater lateral movement of the vocal folds in each cycle and a greater cross-sectional area for air escape in each cycle (resulting in greater measured open quotient). Thus, the resistance of the vocal folds is not linear across various levels of airflow, and the assumption of glottal resistance as the quotient of Psub and U may consequently be incorrect. It appears that the mechanical properties of the folds changed in response to the effect of greater flow, without comparable change in the muscular effort indicated by medial adductory compression. The concept of a flow-controlled nonlinear resistance has been proposed for a number of fluid mechanical systems, such as collapsible tubes.[14] It has been suggested that vocal fold oscillation may demonstrate a specific subset of collapsible tube behavior.[15] Data from these experiments would appear to support that theory. Further exploration of the nature of flow-controlled resistance in the larynx should be encouraged.

CONCLUSIONS

1. During constant medial adductory compression of the vocal folds, increasing airflow from 100 to 500 cc/second increased intensity by only 5 to 10 dB.
2. During constant airflow, increasing medial adductory compression from 60 to 90 cm H_2O increased intensity by 30 dB.
3. Vocal efficiency markedly decreased for increasing airflow and remained essentially unchanged for increasing levels of medial adductory compression.
4. Increasing airflow during constant medial adductory compression produced a significant increase in the open quotient and a larger vocal fold vibratory excursion.
5. The dynamic resistance of the larynx is controlled by the flow during phonation and is not constant, even for constant medial adductory compression.

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