

FUNCTION OF THE INTERARYTENOID MUSCLE IN A CANINE LARYNGEAL MODEL

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The interarytenoid (IA) muscle has rarely been studied in the living larynx. In this work, the role of the IA muscle in phonation was studied in three dogs by means of an *in vivo* phonation model. The isolated action of the IA muscle was studied by sectioning and stimulating its nerve branch. As IA activity increased, subglottic pressure increased significantly until a plateau was reached. In the absence of superior laryngeal nerve stimulation, the fundamental frequency rose with increasing IA activity. In the presence of superior laryngeal nerve stimulation, however, no significant change in fundamental frequency was observed with increasing IA activity. Measurement of adductory force demonstrated that the IA muscle adducts primarily the posterior vocal fold. In this canine model, phonation was not possible without IA stimulation, owing to a large posterior glottic chink.

KEY WORDS — interarytenoid muscle, phonation, recurrent laryngeal nerve, superior laryngeal nerve, videolaryngoscopy, vocal efficiency.

INTRODUCTION

Speech is initiated in the larynx by an alteration of the airstream that accompanies contraction of the intrinsic laryngeal muscles. Electromyography (EMG) of human laryngeal muscles confirms that there is an increase in the activity of the interarytenoid (IA), cricothyroid (CT), thyroarytenoid (TA), and lateral cricoarytenoid (LCA) muscles before and during phonation.¹⁻³ This study will focus on the activity of the IA muscle during phonation in the canine model.

It has been demonstrated that the IA holds the arytenoid cartilages in close contact, closing the posterior portion of the glottis during phonation in both humans² and dogs.⁴ Complete adduction of the vocal folds cannot be achieved solely by IA contraction, however, because the synergistic activation of several muscles is required. Studies of intrinsic muscle function have demonstrated that the LCA muscle is an adductor of the vocal folds, while the CT muscle is a vocal fold tensor. The TA muscle can produce both effects simultaneously.⁴ The precise role of the IA muscle in human phonation has not been determined.

Anatomically, the IA muscle consists of a mass of transverse fibers attaching to the medial surface of the muscular process of the arytenoid. The effect of the IA muscle depends on the configuration of the

glottis. In previous studies performed on intact human larynges, Hirano et al^{2,5} showed that IA activity was greatest for a hypertense style and smallest for a hypotense style of phonation. The effect of the IA muscle on vocal fold adduction was similar to that of the TA muscle, but with less consistency and strength because of its inability to completely approximate the folds acting by itself.⁵ The TA muscle adducts the membranous portion of the vocal folds tightly, while the IA muscle adducts the vocal process of the arytenoid.

The intrinsic muscles also have predictable effects on quality of phonation. According to Hirano's body-cover theory,⁶ the fundamental frequency (F₀) of phonation is determined primarily by the tension on the cover of the membranous vocal folds. The cover includes the mucosa and the superficial lamina propria of the fold.^{6,7} A central aim of the present investigation was to examine the correlation of the F₀ with IA activity. Berke et al⁸ concluded that recurrent laryngeal nerve (RLN) stimulation causes an increase in F₀, but did not separate the effect of the IA. Shipp and McGlone⁹ measured subglottic pressure (P_{sub}), airflow, and EMG of human intrinsic laryngeal muscles in an attempt to correlate muscular activity with F₀. They found that F₀ increased with increasing CT and TA activity. However, no correlation existed between the F₀ and IA or PCA activity.

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Several researchers have concluded that increases in F0 in the chest register are accompanied by increases in the activity of most of the intrinsic laryngeal muscles, primarily CT and TA.^{5,10-12} By varying the F0 and measuring the EMG of the various human laryngeal muscles, Gay et al¹¹ found that IA activity increased with increasing F0. However, there was considerable variability in this relationship between subjects. Hirose¹³ found correlations between F0 and CT, LCA, and TA EMG activities. However, no relationship between IA activity and F0 was identified.

This study used an *in vivo* canine laryngeal model to determine the relationship between IA muscle stimulation and acoustic and aerodynamic measures of phonation. Terminal IA branches were identified and stimulated to obtain isolated muscle activity.

MATERIALS AND METHODS

In Vivo Canine Model. An *in vivo* canine laryngeal model was used, as previously described by this laboratory.^{14,15} Three adult mongrel dogs were premedicated intramuscularly, each with a 3-mL injection of acepromazine maleate. Intravenous pentobarbital sodium (Nembutal) was administered up to the point of corneal anesthesia. The animal was placed supine on the operating table and intubated. An incision was made from the sternal notch to the hyoid bone. The larynx and trachea were exposed by retracting the strap and the sternocleidomastoid muscles. An endotracheal tube was passed through after a distal tracheotomy was performed at the level of the suprasternal notch. This allowed ventilator-assisted respiration. A proximal tracheotomy was also performed through which a cuffed tube was passed with its tip resting 10 cm below the glottis. The cuff was inflated to just seal the trachea. Humidified warm air was passed through the cephalad tube. Room air was warmed by bubbling through 5 cm of water at 37°C. The rate of airflow was controlled with a Gilmont flowmeter (model 1500, Great Neck, NY). The epiglottis was suspended for better visualization of the larynx with a button, 1 cm in diameter.

The RLN was isolated at the tracheoesophageal groove and a stimulating electrode was applied to it. The IA branch was identified as the first terminal branch of the anterior division and confirmed by electrical stimulation; it was cut distally from the branching point, leaving the other terminal branches unharmed. The first branch of the RLN in the dog is the nerve of Galen. Following the RLN distally, one encounters the PCA branch. The third branch from the RLN supplies the IA muscle.¹⁶

Stimulation of the nerve branches was provided by

custom-designed rubber electrodes (monopolar, flexible, conductive neoprene with silicone, coated by insulative silicone KE45W). One electrode was applied to each trunk of the RLN, approximately 5 cm proximal to the takeoff of the posterior division (hereafter referred to as the trunk). The second electrode was applied to the sectioned terminal IA branch. Two more electrodes were applied to the external branch of the superior laryngeal nerve (SLN) bilaterally. The SLN electrodes received electrical input from a Grass model 54H stimulator (Grass Instruments, Quincy, Mass). The electrical stimulation of the trunk was provided by a constant current stimulator (WR Medical Electronics RLN Stimulator, model S2LH, St Paul, Minn). The frequency of nerve stimulation was 80 Hz with a pulse duration of 1.5 milliseconds. The intensities used for stimulation ranged from 0.1 to 2 mA.

Glottography, Pressure, and Intensity Measurements. Photoglottography (PGG) was performed with a photosensor (Centronics OSD 50-2, Mountainside, NJ) placed on the trachea 3 cm below the larynx. Supraglottic illumination was achieved with a halogen flashlight. Electroglottography (EGG) was done with EGG electrodes (Synchrovoice, Briarcliff Manor, NY) placed on both sides of the thyroid cartilage.

The Psub was measured with a Millar catheter-tipped pressure transducer (model SPC 330, Houston, Tex). Calibration of the Millar against a manometer from 0 to 120 mmHg was accomplished just before insertion. The Millar was passed through the upper tracheotomy and placed 2 cm below the glottis. A linear sound level meter (Quest Electronics model 208L, Oconomowoc, Wis) was placed 30 cm from the larynx to measure intensity. The PGG, EGG, Psub, and acoustic signals were low-pass filtered at 3 kHz, digitized at 20 kHz, and recorded on a personal computer. The data were analyzed by means of C-Speech software.

Videostroboscopy-Videolaryngoscopy. Videostroboscopy-videolaryngoscopy was performed during phonation to obtain recorded visual images of the vocal folds and processes. A 0° Karl Storz telescope was connected to the Storz laryngostroboscope (model 8000) via fluid-filled cables. The image from the telescope was recorded by a Storz CCD (charge-coupled device) video camera (model 9000) and a Sony U-matic videocassette recorder (VO-5800).

Measurement of Force. A tensionometer developed previously in this laboratory was used for force measurement.¹⁷ This instrument was designed to measure the adductory force of the vocal folds produced by stimulation of various nerve branches. The footplate of the tensionometer was placed in contact

with the vocal fold of the anesthetized dog. The footplate attaches to a deflection bar that measures the force. The device uses the Shimpo Digital Force Gauge (Shimpo force gauge DF-0.5R, Shimpo American Corp) that combines a light beam detection design with a microcomputer for measurement of push-pull forces. The gauge can measure from all angles and has a 0.1-g resolution. The force gauge was placed on a lightweight platform, allowing frictionless movement during the measurement process. Coarse, intermediate, and micrometer adjustments facilitated the device's maneuverability and precise movement. As little as 0.1 mm of movement of the footplate can be achieved with the micrometer adjustment. The force gauge is attached to the deflection bar through a mechanical linkage that allows an exact transition of force between the footplate and the gauge with minimum loss. The tensionometer only measures the horizontal (transverse) component of the force that is exerted on the footplate by the vocal fold. The area of the footplate is 0.04 cm². The force per unit area is calculated by dividing the force (measured by the tensionometer) by the area of the footplate (0.04 cm²).

Vocal Efficiency and Open Quotient. The sound intensity meter was suspended 30 cm away from the glottis outside of the airflow stream to measure the acoustic quality of phonation. Vocal efficiency (VE) was calculated as the ratio of the acoustic power of the voice to the subglottic power. The total sound power was calculated with the formula¹⁸

$$\text{Total sound power} = 2r^2P_e^2/P_{0c}$$

This formula applies to a sound radiating with no directivity into a hemisphere of area $2r^2$, where r is the distance from the source. The P_{0c} is the specific acoustic impedance and equals 41.1 dynes \times s/cm³ in air at 20°C. The P_e is the root mean square sound pressure in dynes per square centimeter at a distance r from the sound source. The subglottic power is the product of the flow rate and the P_{sub} .

The open quotient (OQ) is the ratio of the duration of the glottal open phase to the total period of the glottal cycle. It can be calculated from the PGG and EGG waveforms, as described in previous studies.^{7,8}

Experimental Design of Dynamic Study. During the experiment the constant airflow was 388 mL/s. The trunk was stimulated with 1.5 to 2.0 V to initiate phonation. Stimulation of the IA branch was varied gradually from 0 to 1.5 V over 1.5 seconds in three trials. The EGG, PGG, and acoustic signals were digitized. Three more trials were performed by the same procedure in the presence of SLN stimulation of 3 V. Data were evaluated at 100-millisecond inter-

vals. The F_0 , P_{sub} , and OQ were measured across 15 consecutive cycles for each interval.

Experimental Design of Static Study. The airflow was held constant at 388 mL/s and the RLN stimulation was adjusted as described above. Three levels of stimulation to the IA branch of the RLN were used: low, medium, and high. These levels were determined subjectively by the quality of phonation: low stimulus levels presented phonation threshold, high stimulus levels produced maximum-intensity phonation, and medium levels were approximately halfway between the previous two levels.

Three trials were recorded for each level of IA branch stimulation. Each trial lasted 1.5 seconds. In order to reduce fatigue of the IA muscle, the stimulations were separated by 3 to 5 minutes. The P_{sub} , F_0 , VE, and OQ were measured from 10 consecutive cycles selected at random from a stable section of phonation.

RESULTS

Videostroboscopy-Videolaryngoscopy. The laryngeal configurations produced by stimulation of the bilateral IA branches of the RLN are depicted in Fig 1. Figure 1A shows the larynx in the resting state. Figure 1B depicts the larynx during bilateral IA branch stimulation. The IA activation results in adduction of the vocal processes of both arytenoids, with a midfold glottal gap. With stimulation of the bilateral RLN trunks with the IA nerve sectioned, medial bulging of the bilateral membranous vocal folds occurs (Fig 1C). However, the vocal processes remain separated and a large posterior glottic gap is present. Combined stimulation of the trunk and IA branch results in full adduction of the folds (Fig 1D). No phonation occurred without IA branch stimulation because of the large posterior laryngeal gap. Moreover, bilateral IA branch stimulation alone did not result in phonation, since a longitudinal vocal fold gap was present. Simultaneous stimulation of both RLN trunks and IA branches resulted in normal phonation, including symmetric mucosal traveling waves.

Dynamic Study. Figure 2 shows plots of the P_{sub} versus time for each dog studied without and with SLN stimulation, respectively. The IA branch stimulation is increased from 0 to 1.5 V within 1.5 seconds depicted in each graph. As these Figures show, P_{sub} increased with IA branch stimulation until a plateau was reached (at IA stimulation of 1.0 V when SLN stimulation was absent, and at 0.8 V when SLN stimulation was present). Prior to this plateau, the SLN stimulation condition had no significant effect on

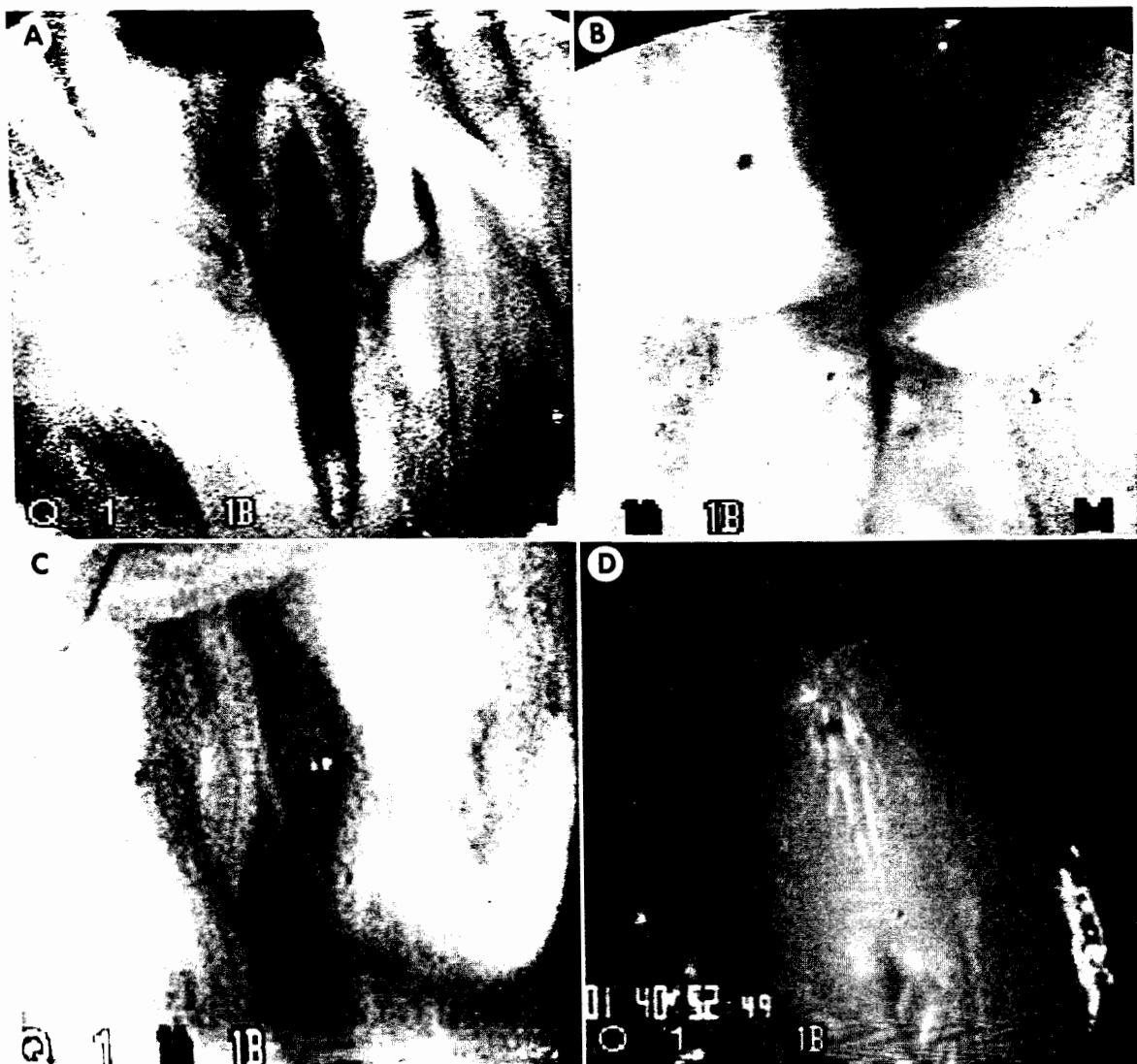


Fig 1. Canine laryngeal configuration in various states. A) Resting state. B) Stimulation of bilateral interarytenoid (IA) muscle nerve branches. C) Stimulation of bilateral recurrent laryngeal nerve trunks. D) Combined stimulation of bilateral IA muscle branches and bilateral recurrent laryngeal nerve trunks.

P_{sub} ($F(1,88) = 1.24, p > .01$). Further, no significant differences among dogs were observed ($F(2,88) = 2.86, p > .01$) and no dog-by-SLN stimulation condition interaction occurred ($F(2,88) = 1.69, p > .01$). For the combined data, prior to the plateau, the increase in P_{sub} with IA stimulation was significant (dog 1: $r^2 = .91, p < .01$; dog 2: $r^2 = .89, p < .01$; dog 3: $r^2 = .97, p < .01$). As IA stimulation increased beyond the plateau level, significant differences between the SLN stimulation conditions emerged ($F(1,80) = 7,775.69, p < .01$), as did significant differences among dogs ($F(2,80) = 2408.21, p < .01$), and there was a significant interaction term ($F(2,80) = 1,271.88, p < .01$).

The F_0 -versus-time graphs are plotted in Fig 3 under similar conditions of increasing IA branch stimulation level (0 to 1.5 V) for three dogs studied. The SLN stimulation had the expected significant

effect on F_0 ($F(1,174) = 440.40, p < .01$). Further, the effect of IA stimulation depended on SLN stimulation. When SLN stimulation was absent (Fig 3A), F_0 increased significantly with increasing IA stimulation (dog 1: $r^2 = .92, p < .01$; dog 2: $r^2 = .99, p < .01$; dog 3: $r^2 = .99, p < .01$) until a plateau was reached, as above. When SLN stimulation was present (Fig 3B), IA stimulation had no significant effect on F_0 (dog 1: $F(1,28) = 0.10, p > .01$; dog 2: $F(1,28) = 1.21, p > .01$; dog 3: $F(1,28) = 0.44, p > .01$).

Static Study. Figure 4 shows the changes in P_{sub} , F_0 , and VE at three different levels of IA stimulation (low, medium, and high) without and with SLN stimulation, respectively, in a representative animal. In the absence of SLN stimulation, the P_{sub} , F_0 , and VE increased with increasing IA stimulation level. When SLN stimulation was present, the P_{sub} and VE rose with increasing IA stimulus level. The F_0 , however,

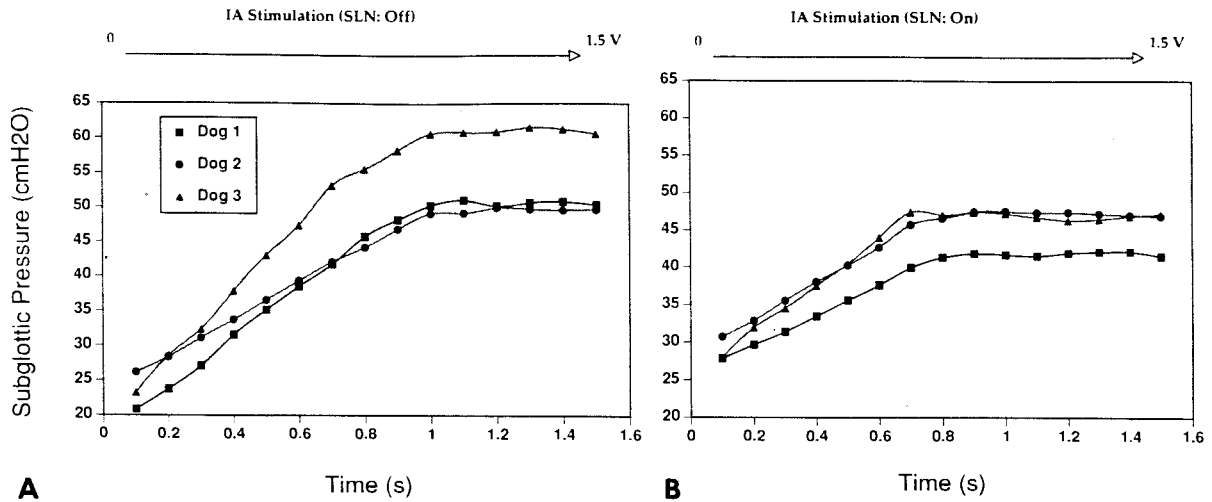


Fig 2. Plots of subglottic pressure versus time for all three dogs with increasing IA muscle nerve branch stimulation. A) Without superior laryngeal nerve (SLN) stimulation. B) With SLN stimulation.

remained unchanged. Although SLN stimulation had the expected significant effect on OQ ($F(1,174) = 3,258.59, p < .01$), no further variation with IA stimulation was observed ($F(1,178) = 0.02, p > .01, r^2 = .00$). Correlation analyses revealed significant ($p < .01$) associations between VE and both F_0 ($r^2 = .65$) and P_{sub} ($r^2 = .42$). For this reason, F_0 and P_{sub} were dropped from the analyses, and a three-way ANOVA was used to evaluate the effects of SLN stimulation level, IA stimulation level, and subject on VE. Parallel analyses using P_{sub} and F_0 as the dependent measures replicated the findings reported here for VE, and will not be described further. Results showed significant main effects of SLN stimulation level ($F(1,36) = 282.22, p < .01$) and IA stimulation level ($F(1,36) = 109.56, p < .01$), but no significant differences among subjects in VE and no significant interaction effects.

Adductory Force Measurements. Figure 5 com-

pares the adductory force–area ratio generated in the middle of the vocal fold with that of the posterior vocal fold in one dog. At all stimulation levels, the force-area ratio is larger posteriorly than at the midfold level. The posterior to middle force-area ratio varies from 1.47 to 5.21 with a mean of 3.15 and an SD of 1.03. Figure 6 compares the midfold to posterior fold force-area ratio generated in three dogs at a high IA branch stimulation level. Similar graphs were obtained for low and medium levels of stimulation.

A four-way ANOVA examined differences among dogs, recording position on the vocal fold (midfold versus posterior fold), side (left versus right), and stimulation levels in their effect on force. All main effects were significant ($p < .01$; dog: $F(2,108) = 8,598.56$; fold position: $F(1,108) = 22,171.78$; side: $F(1,108) = 2,427.73$; stimulation level: $F(2,108) = 5,409.01$). Scheffé post hoc comparisons indicated that each stimulation level differed significantly from

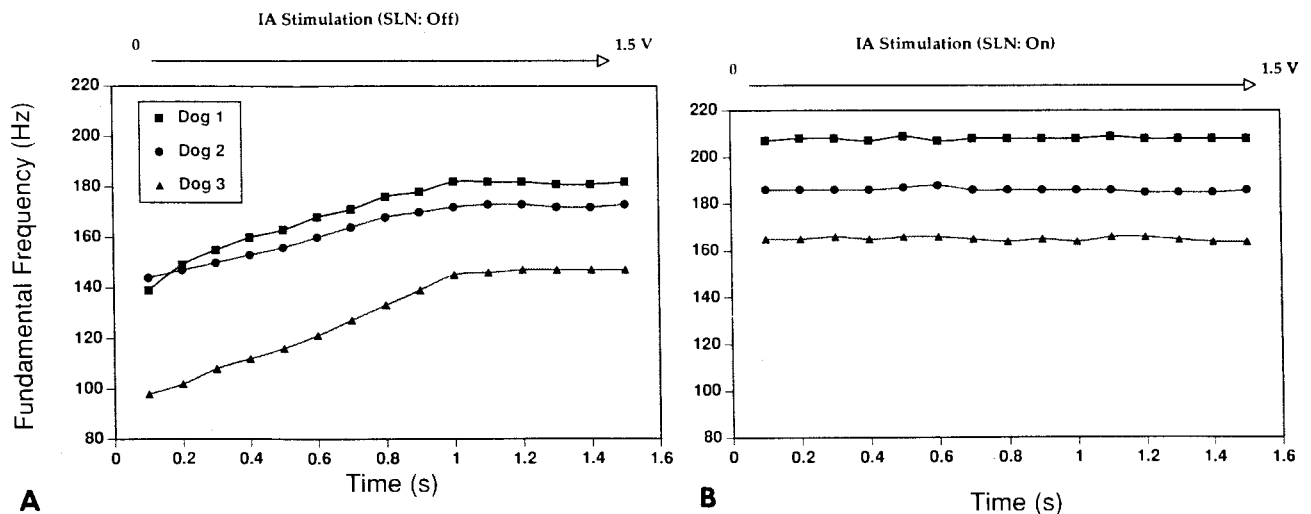


Fig 3. Plots of fundamental frequency versus time with increasing IA muscle nerve branch stimulation in three dogs. A) Without SLN stimulation. B) With SLN stimulation.

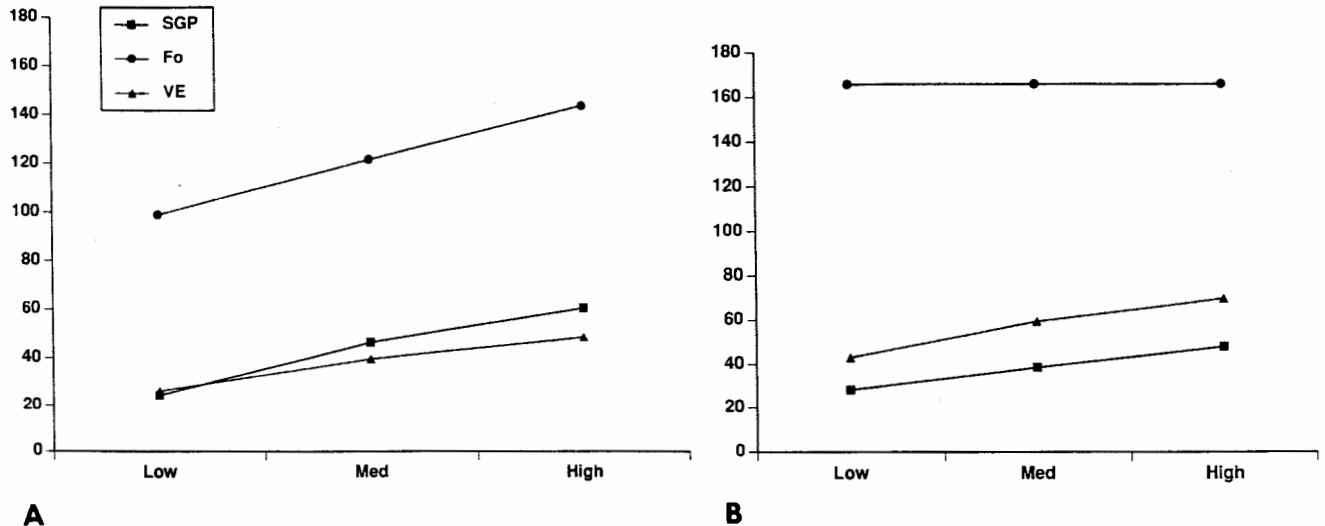


Fig 4. Graphs of subglottic pressure (SGP; in centimeters of water), fundamental frequency (F₀; in hertz), and vocal efficiency (VE; in percent) at three levels (low, medium, high) of IA stimulation in representative animal. A) Without SLN stimulation. B) With SLN stimulation. All vocal efficiency values are times 10⁻⁵.

the others in the adductory force–area ratio generated.

All interaction terms in the ANOVA model proved statistically significant. However, examination of the relevant means indicated that these effects reflected differences in effect magnitude across conditions, rather than any reversal in the data (eg, the difference in the forces recorded at the two positions was greater for dog 3 than for dogs 1 and 2). Further, the largest effect accounted for only 5.6% of the variance in data. For these reasons, interactions will not be discussed further.

DISCUSSION

The aim of this study was to determine the function of the IA muscle in the living larynx. The data presented here suggest that the IA muscle is necessary for vocal fold closure and for the rise in subglottic pressure that occurs in each glottal cycle. These findings are consistent with Van den Berg's¹⁹ myo-

elastic-aerodynamic theory, which states that a sufficient increase in P_{sub} is essential for normal voice production. In order to initiate phonation, P_{sub} must overpower vocal fold resistance just enough to open the folds. As the vocal folds approximate in response to Bernoulli forces and the inherent elasticity of the folds, P_{sub} rises until there is adequate force to reopen them. In the absence of IA stimulation, the dog was unable to phonate because of a large posterior glottic chink that resulted in a low P_{sub}. Optimal phonation required some IA stimulation to provide posterior closure. Videostroboscopy was not possible without IA activation because of the glottal gap and lack of vocal fold entrainment. With simultaneous stimulation of the RLN trunk and IA branch, normal videostroboscopy was obtained.

Previous studies have related muscle activation to acoustic measures of voice production. Changes in the length, mass, and stiffness of the membranous portion of the vocal folds caused by intrinsic muscle

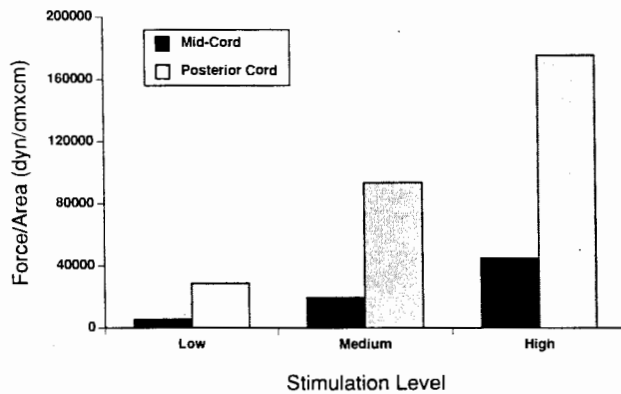


Fig 5. Comparison of adductory force per unit area measured at midfold with that at posterior fold at low, medium, and high IA stimulation levels in one dog.

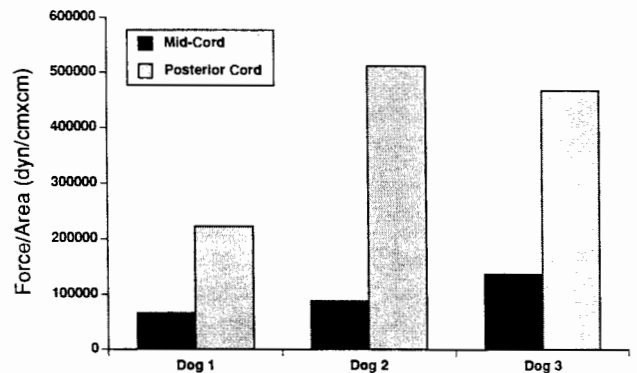


Fig 6. Comparison of adductory force per unit area at midfold and posterior fold at high IA nerve stimulation level in three dogs.

contraction cause predictable changes in the F_0 .³ A low F_0 is associated with a low pitch, a low level of intrinsic muscle stiffness, and a larger area of approximation of the vocal folds; the inverse is true for a high F_0 . The CT and TA are the muscles with the greatest impact on vocal fold tension and hence F_0 .^{11,15}

This experiment assessed the relationship between IA contraction and F_0 by providing constant RLN trunk stimulation with progressive increases in IA stimulation. The result depended on the status of SLN activation. It was found that increasing IA stimulation, in the absence of SLN activation, caused pronounced increases in F_0 . However, in the presence of SLN stimulation, changes in IA stimulation did not affect F_0 significantly. In the presence of SLN stimulation, the effect of the CT apparently overpowers that of the IA and no additional change in F_0 with IA activation occurs.

Vocal efficiency has proven to be useful in assessing glottal insufficiency, and therefore provides another objective measure of glottal closure. The VE is defined as the ratio of the acoustic power to the subglottic power and is directly proportional to intensity. Inadequate closure of the vocal folds can lead to decreases in VE.²⁰ Increases in IA stimulation increase VE regardless of SLN stimulation levels, apparently owing to improvement of posterior glottis closure, resulting in a greater P_{sub} .

Hirose¹³ electrically stimulated the IA muscle in a canine model and found that it adducts the membranous vocal fold to some extent. This suggests that the IA muscle has the ability to change the length and stiffness of the membranous vocal folds when it adducts the cartilaginous (posterior third) folds. In addition to closing the posterior glottis, the IA muscle may have a role in producing the sensitive adjustments in vocal fold stiffness that occur during phonatory tasks. In contrast with the other intrinsic laryngeal muscles, with the possible exception of the LCA, when the IA muscle contracts, it not only adducts the cartilaginous folds, but also moves the attached membranous portion.²¹

Anatomically, the IA muscle is bilaterally innervated by the RLN and consists of the oblique IA and the transverse IA muscles.^{20,22} The horizontal mus-

cular fibers of the transverse IA muscle lie deep to the oblique IA and attach to the length of the dorsolateral ridge and dorsomedial surface of both arytenoid cartilages. The muscular fibers of the oblique IA muscle are in the shape of an X, and attach from the medial half of the muscular process of one arytenoid cartilage to the apex of the contralateral arytenoid cartilage. The IA muscle spans the cricoarytenoid joint. Rotation around the vertical axis and sliding along the long axis of the cricoid facet are allowed. Movements about this joint are limited owing to the nature of the joint: a tight saddle joint capsule and supporting ligaments.³ Judging from their origin and insertion, the oblique IA muscle is most likely primarily responsible for moving the *apex* of the arytenoid cartilages medially, while the transverse IA muscle pulls the *base* of the arytenoid cartilages medially to produce adduction.^{10,11}

To further determine the site of action of the IA muscle, the adductory properties of the muscle were assessed at the membranous midfold level and at the vocal process. A recently developed tensionometer device was used to measure the horizontal force vector of the IA muscle. The IA muscle caused primarily adduction of the vocal process, with a smaller effect on the membranous fold. As IA activity increased, the force of adduction at both levels increased. This result was expected from the posterior location of the IA fibers.

The finding that the dog would not phonate without IA stimulation in our model may not apply to human phonation. The canine larynx is relatively diamond-shaped compared to the more triangular human larynx. In the absence of IA stimulation, a larger posterior glottic gap is present in dogs than in humans. Human research may provide insight into the issue of the need for IA contraction in phonatory tasks.

In summary, the IA muscle has a significant effect on closure of the glottis in the canine model, producing higher subglottic pressure and allowing phonation to occur. The role of the IA muscle was verified with electrical stimulation. Force measurements suggest that the action of the IA muscle is experienced primarily at the posterior vocal fold. Human experiments are required to further illuminate the role of the IA muscle in physiologic phonation.

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