

FUNCTION OF THE THYROARYTENOID MUSCLE IN A CANINE LARYNGEAL MODEL

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Fundamental frequency is controlled by contraction of the thyroarytenoid (TA) and cricothyroid (CT) muscles. While activity of the CT muscle is known to tense and thin the vocal folds, little is known about the effect of the TA muscle on vocal fold vibration. An *in vivo* canine laryngeal model was used to examine the role of the TA muscle in controlling phonation. Isolated TA muscle activation was obtained by stimulating sectioned terminal TA branches through small thyroid cartilage windows. Subglottic pressure measures, electroglottographic and photoglottographic signals, and acoustic signals were obtained in 5 mongrel dogs during dynamic and static variations in TA muscle activity. Results indicated that TA muscle activation is a major determinant in sudden shifts from high-frequency to modal phonation. Subglottic pressure increased and open quotient decreased gradually with increasing TA activation.

KEY WORDS — fundamental frequency, *in vivo* canine laryngeal model, phonation, recurrent laryngeal nerve, thyroarytenoid muscle.

INTRODUCTION

Vocal control during speech or singing is achieved by coordination of the respiratory, laryngeal, and articulatory muscles. Among laryngeal muscles, the cricothyroid (CT) and thyroarytenoid (TA) have been considered tensors of the vocal folds. Arnold¹ claimed that the TA and the CT muscles are in an agonist-antagonist relationship, one being an internal tensor and the other an external tensor of the folds.

The CT muscle is stimulated by an external branch of the superior laryngeal nerve (SLN), and CT muscle activation lengthens and thins the vocal folds. Many studies have clarified the function of the CT muscle in phonation, using electromyography (EMG),^{2,4} morphological techniques,⁵ and *in vivo* canine experiments.⁶ According to the body-cover theory,^{7,8} tension in the cover (the epithelium and the superficial and intermediate layers of the lamina propria) is the primary determinant of fundamental frequency (F₀). Contraction of the CT muscle increases the length of the vocal folds, thus increasing the stiffness of the cover to produce an increase in F₀.

The role of the TA muscle in F₀ regulation is less clear. Hirano et al³ showed that EMG activity in the TA increased with increasing F₀. However, EMG levels dropped when the vocal register shifted to falsetto. Faaborg-Andersen² also found increases in

EMG activity with increased F₀, but across register boundaries EMG activity increased less than for a similar pitch range without a register shift. In contrast, Gay et al⁴ reported no drop in EMG activity as F₀ increased through 90% of a subject's pitch range. Finally, Larson and Kempster,⁹ Kempster et al,¹⁰ and Titze et al¹¹ found both increases and decreases in F₀ after direct electrical TA muscle stimulation during phonation. Apparently, F₀ correlates positively with TA muscle activity at lower F₀s and lower vocal intensities, but at higher F₀s and lower intensities, an increase in TA activity tends to lower the F₀.

The present study used an *in vivo* canine laryngeal model^{12,13} to examine the effect of TA muscle stimulation on F₀, subglottic pressure, vocal intensity, and open quotient (OQ) during phonation. Isolated TA muscle activation was obtained by stimulating sectioned terminal TA branches of both recurrent laryngeal nerves (RLNs) through small thyroid cartilage windows. This technique enabled us to manipulate levels of TA and SLN stimulation independently, and thus to separate their effects on phonation.

METHODS

In Vivo Preparation. Five mongrel dogs were premedicated with an intramuscular injection of 3 mL acepromazine maleate, followed by intravenous pentobarbital sodium (Nembutal) titrated to loss of

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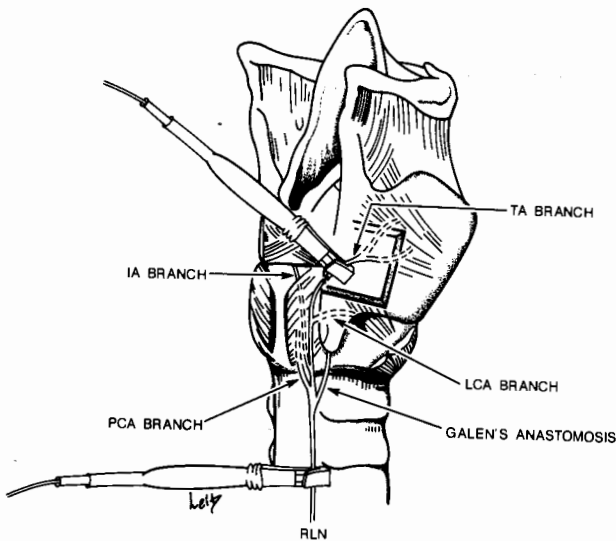


Fig 1. In vivo canine model. Cartilage windows were made at lateral margin of thyroid cartilage to expose thyroarytenoid (TA) branch of recurrent laryngeal nerve (RLN). This branch was cut distal to lateral cricoarytenoid (LCA) branch, and electrode was applied. IA — interarytenoid, PCA — posterior cricoarytenoid.

corneal reflex. Each animal was placed supine on an operating table and direct laryngoscopy was performed to confirm normal laryngeal anatomy. A 7-mm oral endotracheal tube was inserted and connected to a respirator. The animal was shaved, prepared, and draped, and a vertical midline incision was made. The strap muscles and sternocleidomastoid muscles were retracted laterally to expose the larynx and trachea.

The external branch of the SLN was isolated at its entrance into the CT muscle. The RLN was isolated at the tracheoesophageal groove, and its identity was confirmed with electrical stimulation. The inferior constrictor muscle was cut at the lateral margin of the thyroid cartilage. A cartilage window was made with heavy scissors at the lateral-inferior border of the thyroid cartilage. By means of meticulous dissection, the terminal branches of the RLN were located without injuring the intrinsic laryngeal muscles. After stimulation confirmed the lateral cricoarytenoid (LCA) and TA branches, the TA branch was cut 2 mm distal to the LCA branching.

Specially designed rubber electrodes (monopolar, flexible, conductive neoprene with silicone, coated by insulative silicone KE45W) were applied bilaterally as shown in Fig 1. The first was applied around the sectioned terminal TA branch. The second was applied to the main trunk of the RLN to stimulate the other adductor branches (eg, LCA and interarytenoid branches). A third electrode was applied to the external branch of the SLN.

Electrodes attached to each SLN were connected to a Grass model 54H stimulator (Grass Instruments, Quincy, Mass). Electrodes attached to the sectioned TA branches and to the RLN trunks were connected to separate channels of a second nerve stimulator (custom-made, 2-channel, constant-voltage DC stimulator). A ground electrode was inserted into the subcutaneous tissue of the neck flap. Nerves were stimulated with 80-Hz pulses with a 1.5-millisecond duration. Intensity varied from 0 to 3 V, as described below.

A low tracheotomy allowed replacement of the endotracheal tube connected to the respirator, and the oral intubating tube was removed. The epiglottis was suspended with a small button and a 2-0 silk suture for better visualization of the larynx. An additional higher 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 H₂O at 37°C for warming and humidification and passed through the cephalad tracheotomy tube.

Glottography, Pressure, and Intensity Measurements. Electroglossography (EGG) electrodes (Synchrovoice, Briarcliff Manor, NY) were placed in direct contact on either side of the thyroid cartilage. The reference electrode was sutured to the inside of the skin flap.

A photosensor (Centronics OSD 50-2, Mountain-side, NJ) was placed on the animal's trachea, approximately 3 cm below the larynx. A halogen flashlight provided supraglottic illumination for photoglottography (PGG).

A catheter-tipped pressure transducer (Millar Instruments, model SPC 330, Houston, Tex) was inserted through the upper tracheotomy and rested 2 cm below the glottis. The transducer was calibrated against a manometer from 0 to 100 mm Hg just before insertion. Intensity was measured with a linear-scale sound level meter (Quest Electronics, model 208L, Oconomowoc, Wis) positioned 30 cm from the larynx.

The PGG, EGG, subglottic pressure, and acoustic signals were low-pass-filtered at 3 kHz and digitized at 20 kHz with a 12-bit analog-to-digital converter installed in a personal computer. The signals were verified on an oscilloscope (Tektronix 5116, Beaverton, Ore) prior to recording. A multipurpose computer program (CSpeech 3.1) was used to analyze the subglottic pressure, glottography (EGG and PGG), and acoustic signals (Fig 2).

Videostroboscopy. Stroboscopic images of vocal fold movement during phonation were recorded with

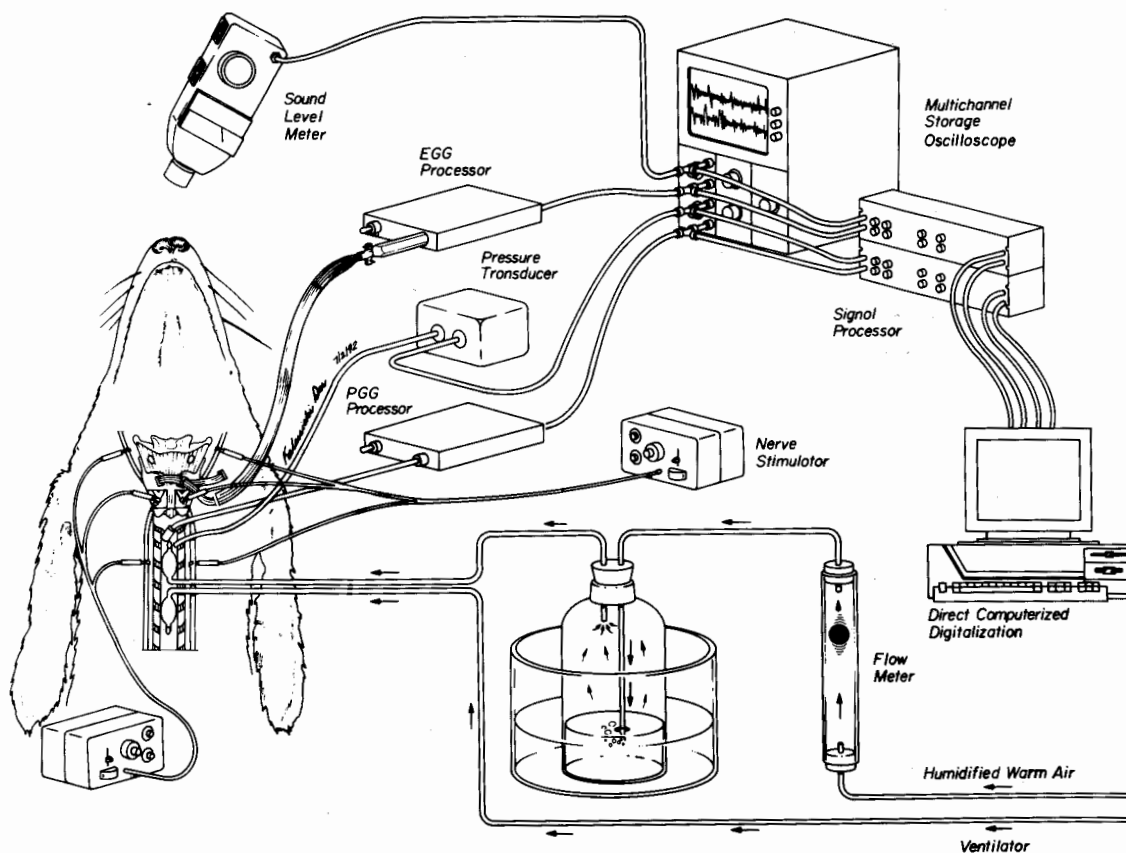


Fig 2. Schematic drawing of experimental model. EGG — electroglottographic, PGG — photoglottographic.

a Storz laryngostroboscope (model 8000). A Storz 0° telescope was connected to the stroboscope via fluid-filled cables. The image from the telescope was recorded with a Storz CCD (charge-coupled device) video camera (model 9000) and a Sony U-Matic videocassette recorder (VO-5800).

Experimental Design of Dynamic Study. Airflow remained constant at 388 mL/s during the experiment. Phonation was induced by stimulating the trunk of the RLN. The level of stimulation was set between 1.5 and 2.0 V as necessary to initiate phonation. Stimulation to the TA branch was then varied gradually from 0 to 3 V over a 3-second trial, during which EGG, PGG, and acoustic signals were digitized. Finally, a second trial was performed by the same procedure in the presence of SLN stimulation at a constant intensity of 3 V.

Data were evaluated at 300-millisecond intervals. Values of F₀, subglottic pressure, and OQ were averaged across 10 consecutive cycles for each interval.

Experimental Design of Static Study. Airflow was held constant at 388 mL/s, and RLN stimulation was adjusted as described above. Three levels of stimulation to the TA branch of the RLN were used: zero (0 V),

low (1.5 V), and high (3 V). Three levels of SLN stimulation were also used (0 V, 1.5 V, and 3 V).

Each trial lasted 0.5 second, and 2 or 3 trials were obtained for each level of TA branch stimulation. Trials were separated by at least 3 to 5 minutes to reduce fatigue effects. Subglottic pressure, F₀, and OQ were measured from 10 consecutive cycles selected at random from a stable section of phonation.

RESULTS

Videostroboscopic Findings. Figure 3 shows the laryngeal configurations produced by the different stimulation conditions. The resting state is shown in Fig 3A. Stimulation of the sectioned TA branch (Fig 3B) produced unique medial bulging of the membranous vocal fold, as reported by Hirano.⁷ Some vocal fold adduction was also noted. However, the posterior commissure and the vocal processes were separated widely. Conversely, with stimulation of the RLN trunk (which contains all the adductor fibers except the TA branch), the vocal processes and the posterior commissure were adducted completely, but a chink remained in the middle of the membranous vocal fold (Fig 3C). Combined stimulation of the TA branch and the trunk of the RLN produced total adduction of the vocal fold (Fig 3D).

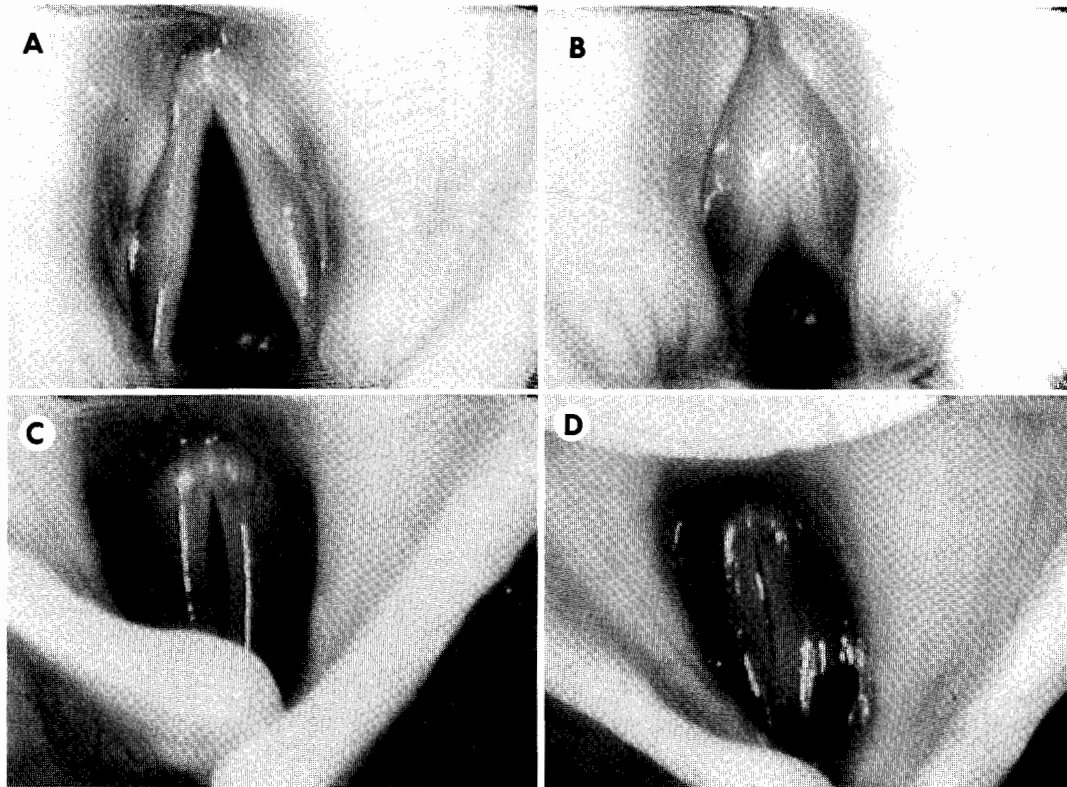


Fig 3. Photographs taken from videotape. A) Resting state. B) Stimulation of TA branch. Medial bulging of membranous vocal fold with some adduction was noted. C) Stimulation of RLN trunk without TA branch. Chink is seen in midmembranous vocal fold. D) Stimulation of RLN together with TA branch. Vocal fold is completely closed.

Dynamic Study. A 2-way (dog by SLN stimulation condition) multivariate analysis of variance (MANOVA) for dependent variables F₀, pressure, and OQ showed significant differences among dogs in levels of F₀ ($F_{4,80} = 6.55$, $p < .05$) and OQ ($F_{4,80} = 8.81$, $p < .05$). However, no significant interactions occurred between dog and SLN level for any dependent measure (F₀: $F_{4,80} = 1.60$, $p > .05$; pressure: $F_{4,80} = 1.45$, $p > .05$; OQ: $F_{4,80} = 1.00$, $p > .05$). Because differences among dogs did not interact with SLN condition, all 5 dogs were combined in the analyses described below.

Some of the dependent measures were moderately but significantly correlated (pressure and OQ: $r = -.69$, $p < .05$; F₀ and OQ: $r = .49$, $p < .05$). Accordingly, multiple regression was used to examine the relationship between TA stimulation and these measures. Separate analyses were undertaken for the SLN-on and SLN-off conditions. When SLN stimulation was absent, changes in TA stimulation produced significant changes in pressure and in OQ ($F_{3,41} = 23.51$, $p < .05$, $r^2 = .63$; pressure: $t = 3.65$, $p < .05$; OQ: $t = -2.04$, $p < .05$; Fig 4A,B). The F₀ did not vary significantly with TA stimulation ($t = -0.79$, $p > .05$). In the presence of SLN stimulation, only pressure varied significantly with TA stimula-

tion ($F_{3,41} = 53.49$, $p < .05$, $r^2 = .80$; pressure: $t = 6.93$, $p < .05$; Fig 4C). No significant changes in OQ or in F₀ were observed (OQ: $t = -0.80$, $p > .05$; F₀: $t = -1.11$, $p > .05$).

Although no overall significant effect of TA stimulation on F₀ was observed, Fig 5A suggests that when SLN stimulation was present all 5 animals experienced sudden shifts in phonatory frequency as TA stimulation increased. The F₀ did change significantly at this point for all dogs ($F_{1,35} = 485.47$, $p < .05$). Further, for every dog subglottic pressure began to rise at the point at which the frequency shifts occurred. However, simple regression confirmed that TA stimulation had no other significant effect on F₀, either before ($F_{1,16} = 3.26$, $p > .05$) or after ($F_{1,30} = 0.70$, $p > .05$) the frequency shift. A frequency shift occurred for only 1 dog when SLN stimulation was absent (Fig 5B).

Static Study. Means for the dependent variables for each level of TA and SLN stimulation are given in Table 1. A 2-way (TA stimulation level by SLN stimulation level) MANOVA showed significant effects of TA stimulation on all the dependent measures (F₀, pressure, intensity, and OQ). The effect of SLN stimulation level was significant only for intensity

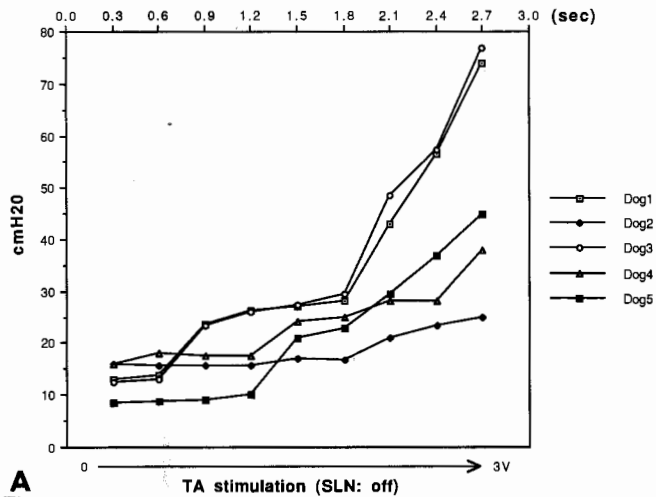
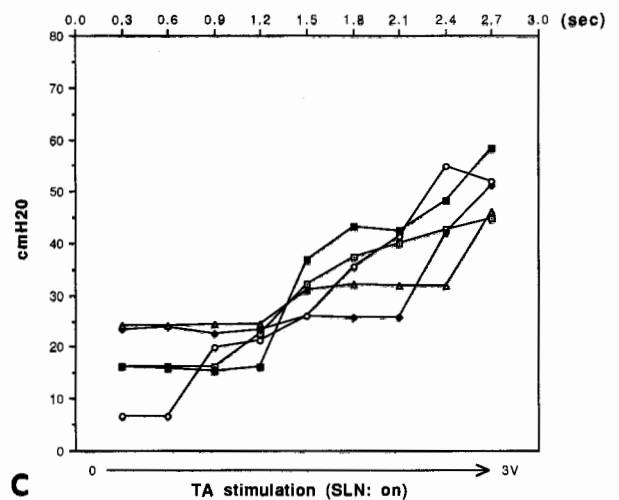
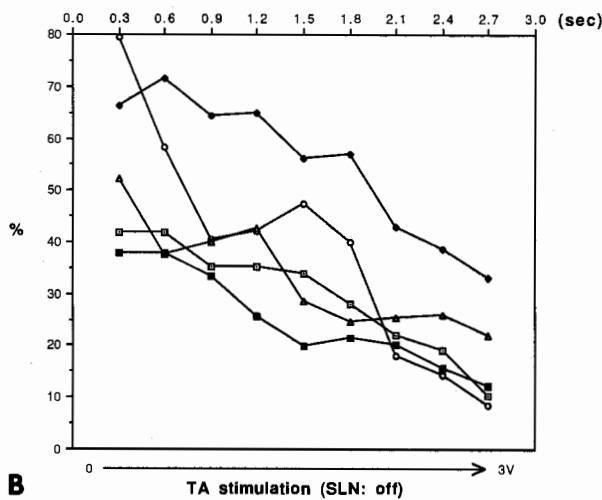


Fig 4. Effects of TA stimulation on subglottic pressure and open quotient. Measurements were made every 0.3 second while intensity of TA muscle stimulation was varied dynamically from 0 to 3 V in 3 seconds. A) Subglottic pressure; superior laryngeal nerve (SLN) stimulation absent. B) Open quotient; SLN stimulation absent. C) Subglottic pressure; SLN stimulation present.



(Table 2). Because the dependent variables were significantly correlated, step-down analysis was used to determine which effects of TA stimulation represented independent effects of stimulation, and which

were merely artifacts of these correlations.

In the step-down analysis, the MANOVA described above was repeated with pressure treated as a

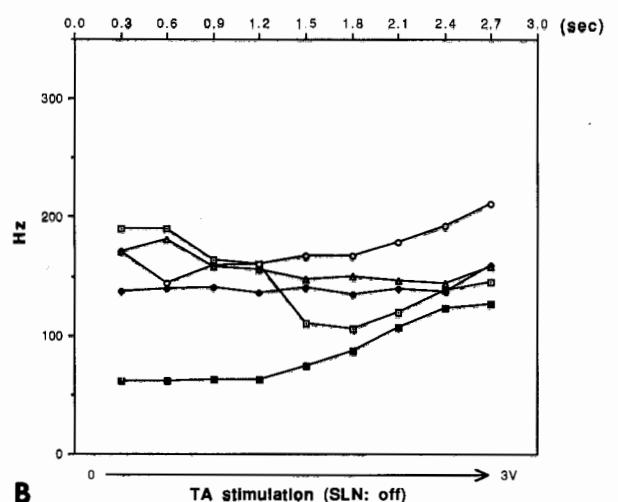
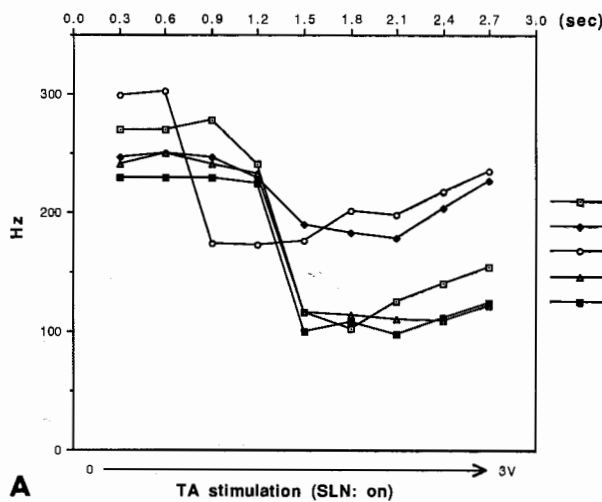


Fig 5. Effect of TA stimulation on fundamental frequency, which was measured every 0.3 second while intensity of TA muscle stimulation was varied dynamically from 0 to 3 V in 3 seconds. A) SLN stimulation present. B) SLN stimulation absent.

TABLE 1. MEANS AND STANDARD DEVIATIONS FOR DEPENDENT MEASURES UNDER VARIOUS LEVELS OF TA AND SLN STIMULATION (STATIC TRIALS)

Independent Variables		Dependent Variables			
TA Stimulation	SLN Stimulation	F0	Subglottic Pressure	Open Quotient	Intensity
Absent	Absent	155.48 ± 42.21	19.94 ± 8.03	60.92 ± 15.63	79.90 ± 5.19
Absent	Low	166.00 ± 16.35	20.82 ± 8.74	47.02 ± 4.99	90.00 ± 10.15
Absent	High	208.26 ± 24.90	19.66 ± 6.65	52.85 ± 10.99	90.40 ± 6.88
Low	Absent	132.52 ± 36.32	38.04 ± 8.21	33.26 ± 4.36	89.20 ± 3.22
Low	Low	120.49 ± 32.31	27.02 ± 3.19	32.56 ± 12.23	87.00 ± 2.00
Low	High	161.18 ± 41.40	34.40 ± 11.14	35.78 ± 6.16	95.50 ± 3.37
High	Absent	204.84 ± 61.11	57.96 ± 20.42	27.38 ± 7.38	96.20 ± 3.19
High	Low	191.76 ± 74.34	54.74 ± 23.47	24.70 ± 10.48	96.80 ± 2.10
High	High	186.00 ± 50.48	49.46 ± 9.52	25.06 ± 3.67	97.80 ± 0.86

TA — thyroarytenoid, SLN — superior laryngeal nerve, F0 — fundamental frequency.

covariate and F0, intensity, and OQ as dependent variables. The effects of TA stimulation on F0 and OQ were still significant after controlling for the correlation between these variables and pressure (F0: $F_{2,80} = 50.98$, $p < .05$; OQ: $F_{2,80} = 55.84$, $p < .05$), but no significant effect on intensity was observed ($F_{2,80} = 2.30$, $p > .05$). Next, F0 was added as a covariate, and the MANOVA was again repeated with dependent variables intensity and OQ. The effect of TA stimulation on OQ remained significant after controlling for correlations with F0 and pressure ($F_{2,79} = 17.77$, $p < .05$).

As in the dynamic trials, sudden shifts in F0 occurred as TA stimulation increased (Fig 6). (Recall that there was no overall effect of SLN stimulation on F0.) Post hoc Scheffé comparisons showed that the F0s obtained in the low-TA stimulation condition differed significantly from those in the TA stimulation-absent and high-TA stimulation conditions ($p < .01$), but that the TA stimulation-absent and high-TA stimulation conditions did not differ significantly from one another ($p > .01$).

DISCUSSION

To summarize our findings, TA stimulation had a significant effect on pressure in dynamic trials, re-

TABLE 2. SIGNIFICANCE TESTS FOR STATIC TRIALS

Independent Variables	Dependent Variables	df	F	p
Thyroarytenoid stimulation level	Fundamental frequency	2, 81	11.99	<.05
	Pressure	2, 81	54.69	<.05
	Intensity	2, 81	32.60	<.05
	Open quotient	2, 81	71.66	<.05
Superior laryngeal nerve stimulation level	Fundamental frequency	2, 81	2.72	>.05
	Pressure	2, 81	1.15	>.05
	Intensity	2, 81	11.64	<.05
	Open quotient	2, 81	2.90	>.05

gardless of SLN stimulation level. The effect on OQ was also significant, but only in the absence of SLN stimulation. Across SLN stimulation levels in static trials, TA stimulation had significant and independent effects on pressure, F0, and OQ. The effect on intensity was apparently an artifact of the intercorrelation among dependent measures. Across TA stimulation levels, SLN stimulation had a significant effect only on intensity. Sudden shifts in F0 occurred as TA stimulation increased in both the static and dynamic trials.

Laryngeal EMG studies of human^{3,4} and canine¹²⁻¹⁴ phonation have helped establish which muscles control F0, intensity, and frequency (register) shifts during vocalization. However, such studies are not ideal for clarifying the functions of individual muscles or for distinguishing the effects of laryngeal muscles from the contributions of the respiratory system. Despite differences in structure between human and canine larynges,⁵ in vivo canine studies are well suited for examining these questions,¹⁵ because the basic gross function of the major laryngeal muscles is quantitatively the same for the 2 species.¹⁶

Most authors distinguish 3 vocal registers: falsetto or light, modal or heavy, and vocal fry.¹⁷ Falsetto is traditionally characterized by the absence of complete glottal closure, high F0, and low intensity. Modal register is accompanied by complete glottal closure for each vibratory cycle. Vocal fry is characterized by an extremely long closed phase relative to the length of a vibratory cycle. In this study, when phonation was evoked by stimulating the RLN trunk without the TA branch, a weak, falsettolike voice was produced. Regardless of the level of SLN stimulation, the subglottic pressure was lower (below 25 cm H₂O), and OQ was higher than when TA stimulation was present. We could easily hear the sudden drop in F0 and an associated change in voice quality.

When SLN stimulation was present, the drop in F0

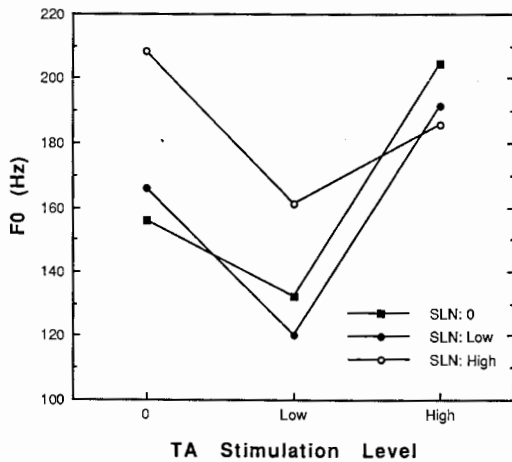


Fig 6. Effect of TA stimulation on F0 in static trials, with and without SLN stimulation.

was more pronounced. Human EMG data^{3,7} suggest that register is regulated primarily by the vocalis muscle, although register, pitch, and intensity are not independent parameters in normal human phonation. Register shifts from falsetto to modal have been associated with increasing vocalis muscle activity. Our data support the suggestion that the TA muscle is responsible for sudden downward frequency shifts.

In the static study, there was a definite decrease in F0 from the TA stimulation-absent condition (high pitch) to the low-TA stimulation condition (modal; Fig 6). Similar results were produced in the dynamic study when SLN stimulation was also present (Fig 5A). In contrast, as TA stimulation increased further during modal phonation, F0 increased. These opposing effects of TA activity on F0 control have been reported by several authors.^{9-11,18} An explanation may be found in the body-cover hypothesis.^{5,8} Hirano⁵ suggested that the TA muscle should be able to stiffen the body of the vocal fold while slackening the cover. Titze et al^{11,18} refined this argument, suggesting that nonuniform stiffening of adjacent vocal fold tissue creates an uncertainty about the effective stiffness of the vibrating tissue. Nonuniform stiffening presumably occurs most markedly when the vocal fold is allowed to shorten. If the vibrating cross-sectional area is primarily nonmuscular tissue, then the effective stiffness should be lowered by TA contraction. On the other hand, if the vibrating cross-sectional area is primarily muscular tissue, then the effective stiffness could be raised. The F0 would be expected to change approximately as the square root of the effective stiffness (or equivalently, the effective longitudinal tension).

According to this logic, the dual effects of TA stimulation on F0 control in our data can be explained. Figure 7 shows a representative dynamic

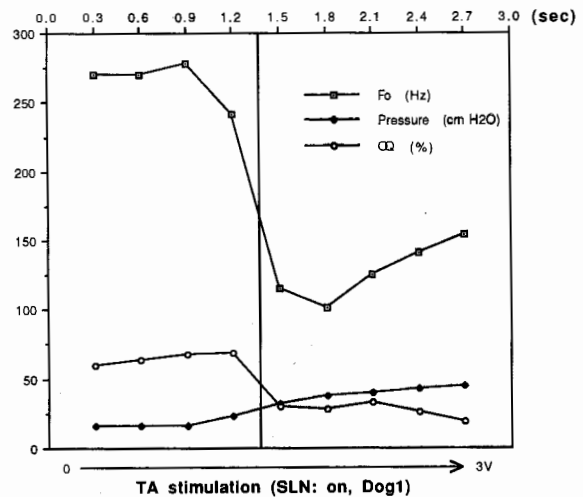


Fig 7. Representative dynamic trial from individual dog. SLN stimulation is present. Fundamental frequency (F0), subglottic pressure, and open quotient (OQ) were measured every 0.3 second while intensity of TA stimulation varied dynamically from 0 to 3 V in 3 seconds. In this plot, y-axis represents units for each variable. Time is on x-axis. Vertical line—sudden shift from high frequency to modal phonation.

trial for an individual dog with SLN stimulation present. The starting point (0.9 second) and the ending point (1.8 seconds) of the F0 decrement correlate exactly with the rapid increment of subglottic pressure (0.9 to 1.8 seconds). After 1.8 seconds, subglottic pressure has built up to a certain extent and F0 begins to increase with increasing subglottic pressure. After this point (1.8 seconds), the increase in tension in the muscle outweighs any decrease in tension in the cover that may result from a small decrease in vocal fold length.

Loudness can be controlled through respiratory and laryngeal mechanisms. In this study, the effect of respiratory control can be ignored, because flow was set constantly at 388 mL/s. Isshiki^{19,20} postulated that laryngeal control of intensity is dominant for very low pitches and that expiratory muscle control is dominant for extremely high pitches. In modal register, the muscle that exhibits the greatest variation in activity with changes in intensity is the TA; LCA and IA activity increase with vocal intensity, but less consistently than TA activity.¹⁷ Titze suggested that subglottic pressure is the primary variable for control of vocal intensity,²¹ and elevation of subglottic pressure also affects F0.²² In this study, TA activation definitely increased subglottic pressure (Fig 4A,C and Table 1). However, intensity was not simply correlated with subglottic pressure.

As TA stimulation increased, the OQ decreased gradually (Fig 4B and Table 1). The drop in OQ with TA activation has been reported previously in hu-

mans.²³ At the point of sudden pitch shift from high to modal, however, rapid changes in OQ were noted (Fig 7). Both the drop in F0 and the sudden drop in OQ (change of the duty cycle) were correlated with the change in vocal quality observed at this point.

Some researchers claim that the TA consists of 2 muscles: the vocalis, which is the most medial part lying adjacent to the vocal cords, and the TA proper, which lies lateral to the vocalis and comprises the main bulk of the muscle.^{9,24,25} They suggest that the TA is a complex muscle, and different subcomponents may have different functions. Because the TA muscle is one of the most important muscles for the laryngeal control of phonation and singing, development of a more elaborate in vivo model (possibly incorporating a constant level of subglottic pressure to drive phonation) to further clarify the delicate functions of the TA

muscle will be helpful.

CONCLUSION

A modified in vivo canine model was used to clarify the function of the TA muscle in phonation. Results indicated that TA muscle activation is a major determinant in sudden pitch shifts from high frequency to modal phonation. The F0 increased with increasing TA activation in modal register. The F0 decreased with TA activation when the evoked voice was high in F0. This effect was more pronounced in the presence of simultaneous SLN stimulation. Subglottic pressure increased gradually and OQ decreased gradually with TA activation. However, the change in OQ was more abrupt at the point at which sudden shifts in F0 occurred. This phenomenon seems to be related to changes in voice quality during sudden frequency shifts.

REFERENCES

1. Arnold GE. Physiology and pathology of the cricothyroid muscle. *Laryngoscope* 1961;71:687-753.
- * 2. Faaborg-Andersen K. Electromyography of laryngeal muscles in humans: techniques and results. *Folia Phoniatr (Basel)* 1965;3:1-71.
3. Hirano M, Vennard W, Ohala J. Regulation of register, pitch and intensity of voice. *Folia Phoniatr (Basel)* 1970;22:1-20.
4. Gay T, Hirose H, Strome M, Sawashima M. Electromyography of the intrinsic laryngeal muscles during phonation. *Ann Otol Rhinol Laryngol* 1972;81:401-9.
5. Hirano M. Morphological structure of the vocal cord as a vibrator and its variations. *Folia Phoniatr (Basel)* 1976;26:89-94.
6. Moore MD, 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.
7. Hirano M. Phonosurgery. Basic and clinical investigations. *Otol Fukuoka (Jibi To Rinsho)* 1975;21:239-440.
8. Fujimura O. Body-cover theory of the vocal fold and its phonetic implications. In: Steven KN, Hirano M, eds. *Vocal fold physiology*. Tokyo, Japan: University of Tokyo Press, 1981:271-90.
9. Larson CR, Kempster GB. Voice fundamental frequency changes following discharge of laryngeal motor units. In: Titze IR, Scherer RC, eds. *Vocal fold physiology: biomechanics, acoustics, and phonatory control*. Denver, Colo: The Denver Center for the Performing Arts, 1983:91-104.
10. Kempster GB, Larson CR, Kistler MK. Effects of electrical stimulation of cricoarytenoid and thyroid muscles on voice fundamental frequency. *J Voice* 1988;2:221-9.
11. Titze IR, Luschei ES, Hirano M. Role of the thyroarytenoid muscle in regulation of fundamental frequency. *J Voice* 1989;3:213-24.
12. Berke GS, Moore DM, Hansen DG, Hantke DR, Gerratt BR, Burstein F. Laryngeal modeling: theoretical, in vitro, in vivo. *Laryngoscope* 1987;97:871-81.
13. Green DC, Berke GS, Ward PH. Vocal fold medialization by surgical augmentation versus arytenoid adduction in the in vivo canine model. *Ann Otol Rhinol Laryngol* 1991;100:280-7.
14. Rubin HJ. Experimental studies on vocal pitch and intensity in phonation. *Laryngoscope* 1963;73:973-1015.
15. Choi HS, Berke GS, Ye M, Natividad M. Function of the posterior cricoarytenoid muscle in phonation: in vivo laryngeal model [Abstract]. *Otolaryngol Head Neck Surg* 1992;107:240.
16. Takase S. Studies on the intrinsic laryngeal muscles of mammals. *Comparative anatomy and physiology*. *Otol Fukuoka (Jibi To Rinsho)* 1964;10(suppl 1):118-58.
17. Hirano M. The laryngeal muscles in singing. In: Hirano M, Kirchner JA, Bless DM, eds. *Neurology: recent advances*. Boston, Mass: College-Hill, 1987:209-30.
18. Titze IR, Jiang J, Drucker DG. Preliminaries to the body-cover theory of pitch control. *J Voice* 1987;1:314-9.
19. Isshiki N. Regulatory mechanism of voice intensity variation. *J Speech Hear Res* 1964;7:17-29.
20. Isshiki N. Vocal intensity and air flow rate. *Folia Phoniatr (Basel)* 1965;17:92-104.
21. Titze I. Regulation of vocal power and efficiency by subglottal pressure and glottal width. In: Fujimura O, ed. *Vocal fold physiology*. New York, NY: Raven Press, 1988:227-38.
22. Titze I. On the relation between subglottal pressure and fundamental frequency in phonation. *J Acoustic Soc Am* 1989;85:901-6.
23. Kempster G, Preston J, Mack R, Larson C. A preliminary investigation relating laryngeal muscle activity to changes in EGG wave forms. In: Titze IR, Scherer RC, eds. *Vocal fold physiology: biomechanics, acoustics, and phonatory control*. Denver, Colo: The Denver Center for the Performing Arts, 1983:339-48.
24. Kahane JC. Anatomy and physiology of the organs of the peripheral speech mechanism. In: Lass NJ, McReynolds LV, Northern JL, Yoder DE, eds. *Speech, language, and hearing 1: Normal processes*. Philadelphia, Pa: WB Saunders, 1982:109-55.
25. Wu B-L, Biller HF, Sanders I. Histochemical evidence for separate thyroarytenoid and vocalis muscle within the canine larynx [Abstract]. *Otolaryngol Head Neck Surg* 1990;103:191.