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Function of the Interarytenoid(IA) Muscle in Phonation: In Vivo Laryngeal Model

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Thyroarytenoid(TA), lateral cricoarytenoid(LCA), and IA muscles are referred to as the adductors of the vocal fold. The TA is known to shorten the vocal folds and to adduct the membranous vocal fold, and the LCA adducts the inter-vocal process region and IA adducts the posterior commissure. Even though IA has an important role for the positioning of the vocal folds during respiration and phonation together with the action of the posterior cricoarytenoid muscle, little is known about the effect of IA on voice parameters during phonation. An in vivo canine model was used in five mongrel dogs to examine the role of the IA muscle in controlling phonation. In two out of five dogs, sound could not be elicited without stimulating the IA branches of the recurrent laryngeal nerves. When the IA was dynamically and statically stimulated , subglottic pressure, vocal intensity and fundamental frequency were increased. However, open quotient was not changed markedly. These results suggest that the IA affects the voice parameters mainly by controlling subglottic pressure during phonation.

Key Words: Inter-arytenoid muscle, In vivo laryngeal model

A variety of voiced sounds are produced by the coordinated activity of the larynx and the supra-laryngeal articulators, which use respiratory air flow as the source energy. As a phonatory control mechanism, the larynx enables us to turn the direct flow of air from the lungs into an alternating on-off flow, which from an acoustic point of view is far more efficient. According to the myoelastic-aerodynamic theory of phonation, presented by Van den Berg (1958), a prerequisite for the initiation of the vibration cycle is the adduction of the vocal folds within 3 mm of each other by the action of the adductor muscles especially the lateral cricoarytenoid (LCA) and the interarytenoid (IA) muscles.

Thyroarytenoid (TA), LCA, and IA muscles are referred to as the adductors of the vocal fold. The TA muscle shortens the vocal folds and adducts the membranous vocal fold, in addition increases glottal resistance. An increase in glottal resistance is one of the most important factors contributing to an increase in vocal intensity. The TA muscle has dual effect on the control of fundamental frequency. In addition, it plays important roles in regulating the vocal register and also affects the open quotient (Choi *et al.* 1993; Hirano, 1987; Titze *et al.* 1989).

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The LCA adducts the inter-vocal process region and IA adducts the posterior commissure. According to results from an human electromyographic (EMG) examination, LCA is found to participate in the control of the vocal register, the fundamental frequency and the vocal intensity. It is not a leading factor, but an important supporting factor (Hirano, 1987).

While contracting, the IA muscle approximates the posterior ends of the arytenoid cartilages, thereby playing an important role in both the phonatory and the sphincteric mechanisms of the larynx (Ballenger, 1985; Williams, 1989). Together with the action of the posterior cricoarytenoid muscle, the IA plays an important role for positioning the vocal folds during respiration and phonation. However, little is known about the effect of the IA on voice parameters during phonation. Hirano (1987) investigated functions of the IA in his limited EMG studies and reported that the IA does not affect the mechanical characteristics of the vibrator as much as the previous two muscles do. Its main role is to close the glottis. It seems, however, that the IA supports other muscles in controlling the vocal register, the fundamental frequency, and the vocal intensity.

Recently, the in vivo canine laryngeal model has been used to investigate the role of the individual intrinsic laryngeal muscles (Choi *et al.* 1993; Choi *et al.* 1993). Compared to the EMG study, the in vivo canine model study has an advantage in that the isolated particular intrinsic muscle can be stimulated under constant air flow, while the remaining intrinsic muscles remain stimulated at a constant level.

In this study, the similar setup of in vivo canine laryngeal model was utilized to clarify the function of the IA muscle in phonation.

METHODS

In Vivo Preparation

Five mongrel dogs were premedicated with an intramuscular injection of 3 ml acepromazine maleate, followed by an intravenous pentobarbital sodium (Nembutal) titrated to remove any corneal reflex. Each animal was placed supine on an operating table, whereupon direct laryngoscopy was performed to confirm a normal laryngeal anatomy. A 7 mm. oral endotracheal tube was inserted and connected to a respirator. The animal was shaved, prepared, and draped. A vertical midline incision was then made. The strap muscles and sternocleidomastoid muscles were laterally retracted to expose the larynx and trachea.

The external branch of the superior laryngeal nerve (SLN) was isolated at its entrance into the cricothyroid (CT) muscle. The recurrent laryngeal nerve (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. The terminal branches of the RLN were then located without injuring any of 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 branch. Branches innervating the posterior cricoarytenoid (PCA) muscle and Galen's anastomosis were transected. (Fig. 1a)

Specially designed rubber electrodes (monopolar, flexible, conductive neoprene with silicone, coated with insulative silicone KE45W) were applied bilaterally. 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: the LCA branch and the IA branch. 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). Separate electrodes were attached to the sectioned TA branches and to the RLN trunks, and those 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. The nerves were then

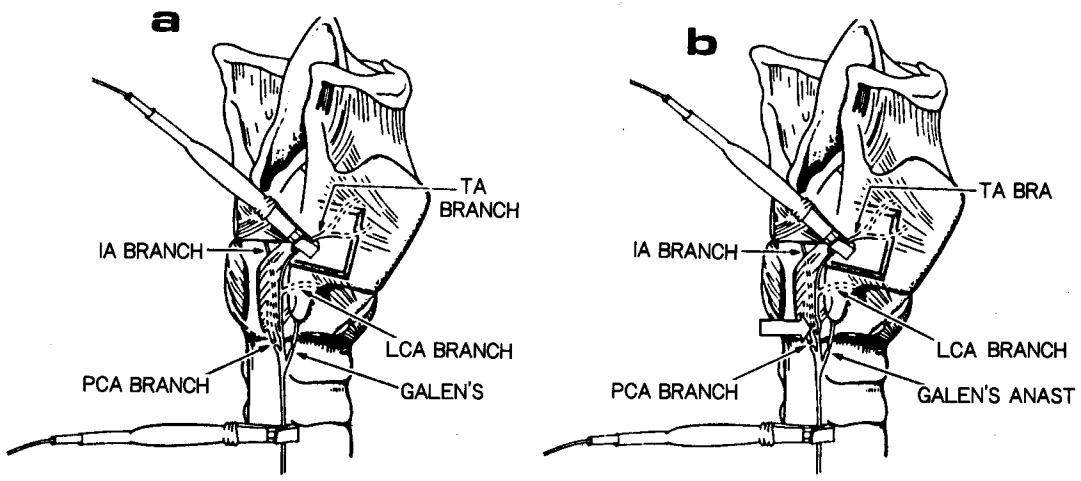

Fig. 1. *In vivo canine model. Cartilage windows were made at lateral margin of thyroid cartilage.*
a) LCA+IA setup; TA, PCA branches and Galen's anastomosis of the RLN were cut. Electrodes were applied to the TA branch and trunk of RLN.
b) LCA (cut IA) setup; IA branch (large open arrow) was cut additionally. Electrodes were applied at the points.
LCA: lateral cricoarytenoid, IA: interarytenoid, TA: thyroarytenoid, PCA: posterior cricoarytenoid, RLN: recurrent laryngeal nerve

stimulated with 1.5-millisecond pulses of 80 Hz. Intensity varied from 0 to 3 V, as described below.

A low tracheotomy allowed the 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 to better visualize the larynx. An additional higher tracheotomy was also performed through which a cuffed tracheotomy tube was placed with its tip resting 10 cm below the glottis. The cuff of the higher directed tube was inflated to just seal the trachea. Room air which passed through the cephalad tracheotomy was bubbled through 5 cm of H_2O at 37℃ for warmth and humidification.

Glottography, Pressure, and Intensity Measurements: Electroglottography (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, Mountainside, NJ) was placed on the animal's trachea, approximately 3 cm below the larynx. A halogen flashlight provided supraglottic illumination for the 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 its insertion. The 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 to 20 kHz by 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 monitor subglottic pressure, glottography (EGG and PGG), and acoustic signals (Fig. 2).

Videostroboscopy: Stroboscopic images of

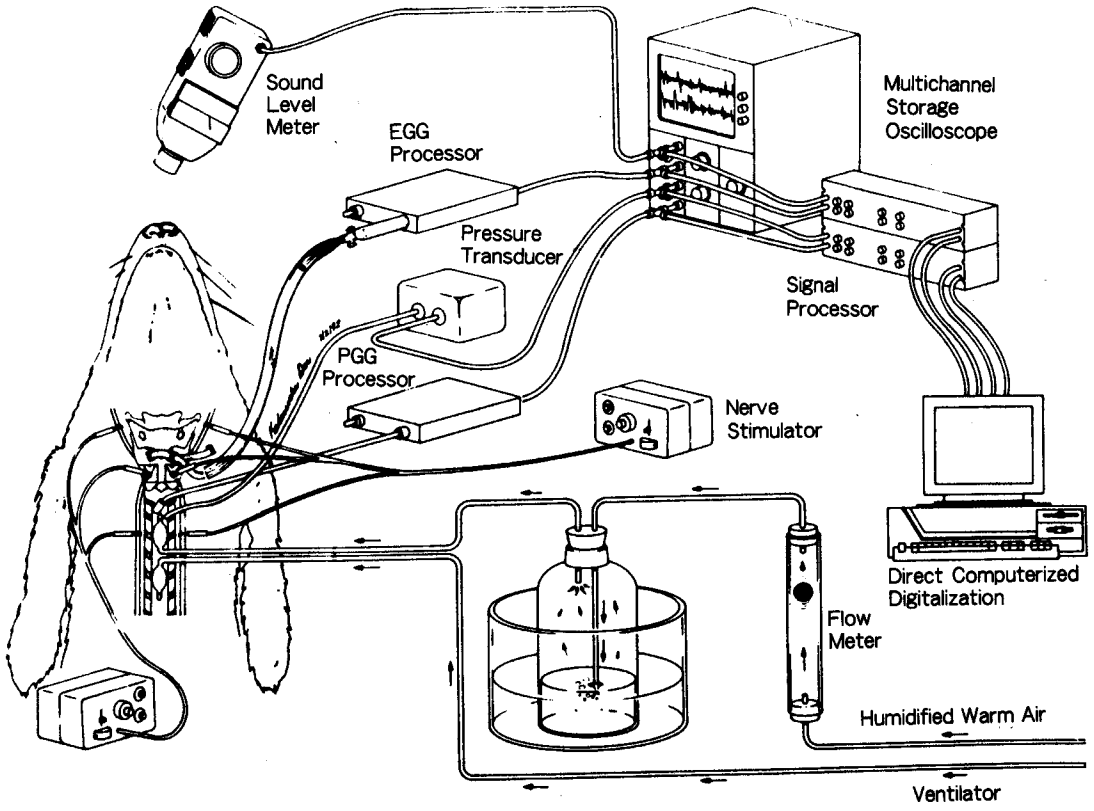

Fig. 2. *Schematic drawing of experimental model. EGG-electroglottographic, PGG-photoglottographic*

the vocal fold movement during phonation were recorded by a Storz laryngostroboscope (model 8000). A Storz 0° telescope was connected to the stroboscope via fluid-filled cables. Then 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 (V0-5800).

Experimental Design of Subtraction Methods: Comparison of LCA+IA Stimulation and only LCA (cut IA) Stimulation

Neither the LCA nor the IA branch was too small and too short to be stimulated separately by the applied electrode respectively. As a result, we had adopted subtraction methods. Under constant air flow and constant electrical stimulation to the TA and CT muscles, the effect of the LCA + IA (the trunk of the RLN only has LCA and IA branches in this setup) stimulation was measured at first (Fig. 1a) and then the effect of only LCA stimulation was measured after transecting the IA branches bilaterally(Fig. 1b).

Dynamic Study: Airflow remained constant at 388 mL/s during the experiment. Stimulation of the SLN was set to 0.5 V and the level of TA stimulation was set between 0.5 V and 1 V as needed. Stimulation to the trunk of the RLN (LCA+IA, LCA: cut IA) varied gradually from 0 to 3 V over a 3-second trial, during when subglottic pressure, EGG, PGG, and acoustic signals were digitized. Data was evaluated at 300-millisecond intervals. The averages of the values for Fo, subglottic pressure, and OQ accross 10 consec-

utive cycles for each interval were recorded.

Static Study: Likewise, airflow was held constant at 388 mL/s, and stimulation to the SLN and TA was adjusted as described above. Two levels of stimulation applied to the trunk of the RLN-LCA+IA and to only the LCA (cut IA)-were used: low (0.7 V), and high (1.5 V). Each trial lasted 0.5 seconds, and the results of 2 trials were obtained for each level of RLN stimulation. Each trial was separated by at least 3 to 5 minutes to reduce fatigue effects. Subglottic pressure, Fo, and OQ were measured from 10 consecutive cycles, randomly selected from a stable section of phonation.

RESULTS

Videolaryngoscopic examination

Three levels of the vocal fold adduction were identified. The TA muscle adducts the anterior membranous portion of the vocal folds (Fig. 3b, 4b), while the LCA muscle adducts the midportion of the glottis, mainly the inter-vocal process area (Fig. 4c). Comparing Fig. 3d to Fig. 4d, the IA muscle is responsible for adduction the posterior commissure.

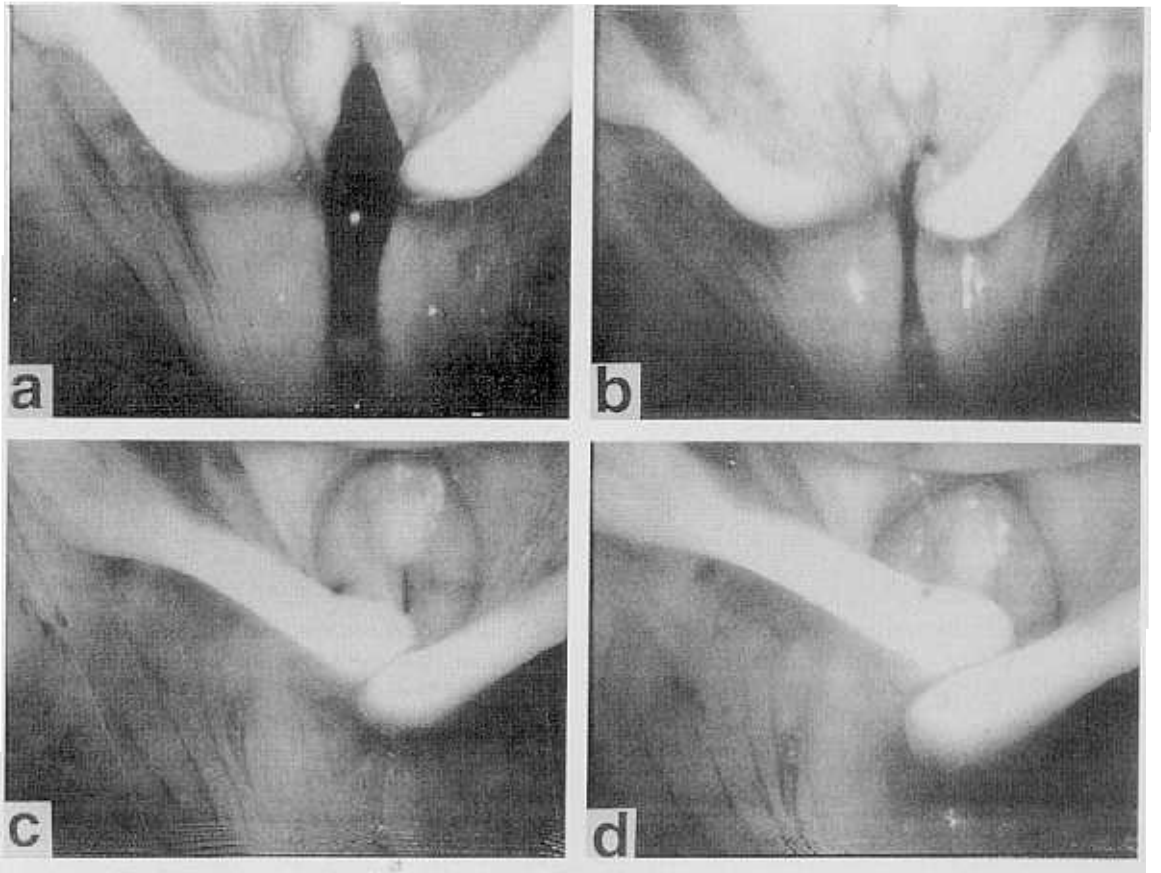

Fig. 3. Photographs taken from video tapes: LCA+IA setup
a) resting, b) TA stimulation, c) LCA+IA stimulation, d) LCA+IA+TA stimulation
LCA: lateral cricoarytenoid, IA: interarytenoid, TA: thyroarytenoid

Dynamic study

Figures 5 and 6 reveal how subglottic pressure is effected by stimulation of both LCA+IA, and LCA (cut IA) in each of the 5 dogs. High LCA+IA stimulation tends to be associated with high subglottic pressure and vice versa (Fig. 5). This association is measured by the correlation test, and the correlation coefficient (r) was 0.93. Analysis of the variance approach to simple linear regression demonstrated that subglottic pressure had increased with LCA+IA stimulation level ($t=6.71$, $p<0.01$). High LCA (cut IA) stimulation also tends to increase the subglottic pressure (correlation coefficient $r=0.90$, $t=5.57$, $p<0.01$), but the increment of subglottic pressure was much smaller than that of the LCA+IA stimulation (Fig. 6). Analysis of the variance for the multiple regression of subglottic pressure on LCA+IA stimulation and LCA (cut IA) stimulation revealed that cutting the IA produced significant changes in subglottic pressure ($r^2=0.88$, $0<0.002$). These results appeared to be the consequence of the air leakage through the big gap in the posterior commissure (Fig. 4c, d) during phonation.

Static study

After cutting the IA in dog 3 and in dog 5, a voice could not be induced even with maxi-

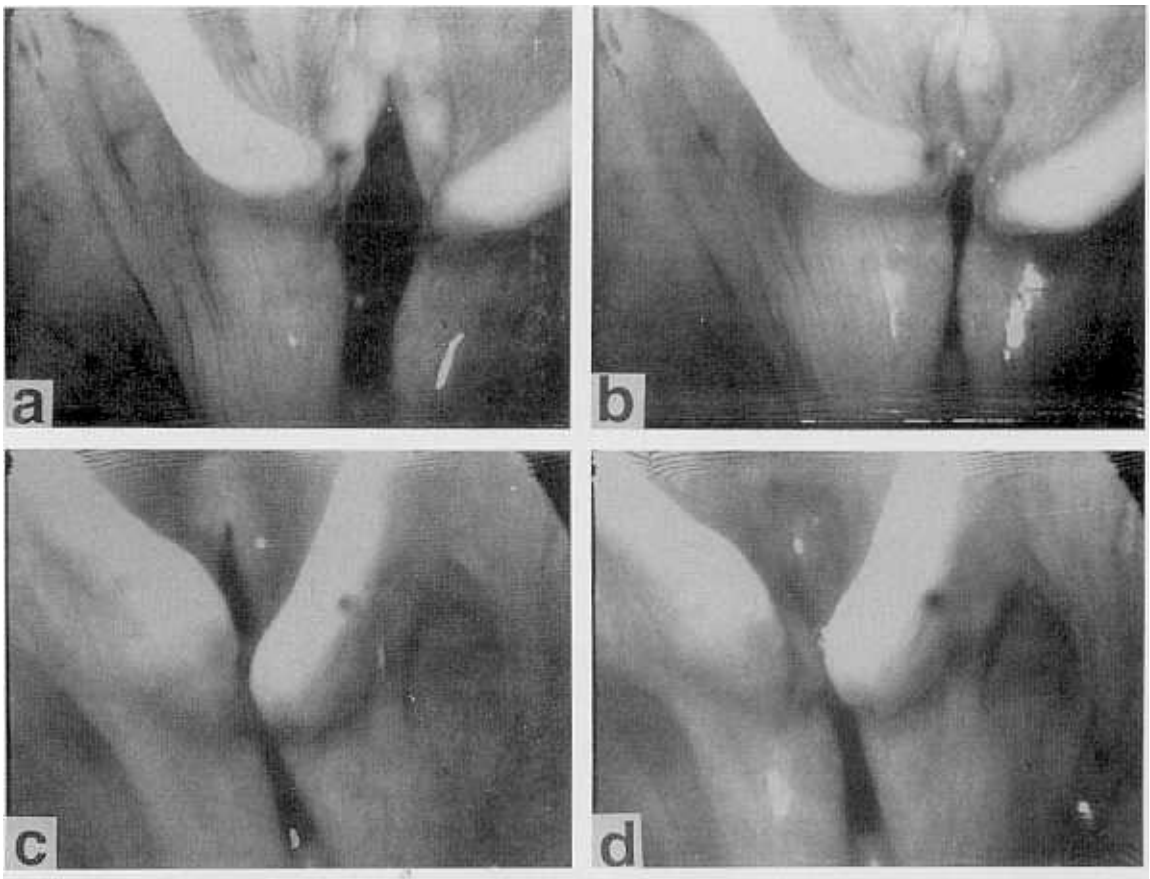

Fig. 4. Photographs taken from video tapes: LCA(cut IA) setup.
a) resting, b) TA stimulation, c) LCA(cut IA) stimulation, d) LCA (cut IA)+TA stimulation, LCA: lateral cricoarytenoid IA: interarytenoid, TA: thyroarytenoid