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Control of Pre-phonatory Glottal Shape by Intrinsic Laryngeal Muscles

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Objectives: Surgical manipulations to treat glottic insufficiency aim to restore the physiologic pre-phonatory glottal shape. However, the physiologic pre-phonatory glottal shape as a function of interactions between all intrinsic laryngeal muscles (ILMs) has not been described. Vocal fold posture and medial surface shape were investigated across concurrent activation and interactions of thyroarytenoid (TA), cricothyroid (CT), and lateral cricoarytenoid/interarytenoid (LCA/IA) muscles.

Study Design: In vivo canine hemilarynx model.

Methods: The ILMs were stimulated across combinations of four graded levels each from low-to-high activation. A total of 64 distinct medial surface postures (4 TA × 4 CT × 4 LCA/IA levels) were captured using high-speed video. Using a custom 3D interpolation algorithm, the medial surface shape was reconstructed.

Results: Combined activation of ILMs yielded a range of unique pre-phonatory postures. Both LCA/IA and TA activation adducted the vocal fold but with greater contribution from TA. The transition from a convergent to a rectangular glottal shape was primarily mediated by TA muscle activation but LCA/IA and TA together resulted in a smooth rectangular glottis compared to TA alone, which caused rectangular glottis with inferomedial bulging. CT activation resulted in a lengthened but slightly abducted glottis.

Conclusions: TA was primarily responsible for the rectangular shape of the adducted glottis with synergistic contribution from the LCA/IA. CT contributed minimally to vocal fold medial shape but elongated the glottis. These findings further refine laryngeal posture goals in surgical correction of glottic insufficiency.

Key Words: larynx, pre-phonatory posture, intrinsic laryngeal muscles, vocal fold medial surface.

Level of Evidence: NA, Basic science

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INTRODUCTION

The three-dimensional glottal shape and stiffness during phonation are determined by the intrinsic laryngeal muscles (ILMs). Phonatory shape and stiffness are also targeted by many phonosurgical interventions. These include adduction procedures, such as injection laryngoplasty, medialization thyroplasty, and arytenoid adduction,[1] and length-changing procedures such as thyroplasty types 3–4,[2] and glottoplasty.[3] Current interventions use a superior endoscopic view to assess the vocal fold antero-posterior length and the degree of adduction. The third dimension of glottal posture, the medial vocal fold surface shape, is not commonly visualized or considered. Thus, current voice optimization procedures focus on vocal fold length and medialization although omitting medial surface shape, which plays a significant role in voice production and quality.[4,5]

During each glottal vibratory cycle, the inferior surface of the vocal fold medial surface serves as the origin for the mucosal wave/upheaval,[6] which then propagates superiorly as the glottal shape cycles between convergent, rectangular, and divergent shapes.[7] Because the vocal fold medial surface serves as the most critical region for the origin of the mucosal wave,[5] further understanding the vocal fold medial surface shape due to ILM activation is critically important to understanding the mechanics of voice production and optimizing phonosurgical treatment of voice disorders.

The vocal fold pre-phonatory shape, defined as the glottal shape before the onset of sustained oscillation, is determined by the relative activation state of each ILM. ILM effects on vocal fold posture have previously been investigated and basic understanding of their roles have been elucidated.[8–13] The thyroarytenoid (TA) muscle adducts, shortens, and creates mid-membranous bulging of the vocal fold. The lateral cricoarytenoid/interarytenoid (LCA/IA) muscle complex primarily adducts the posterior glottis. The cricothyroid (CT) muscle elongates the vocal fold and opposes the TA muscle. Previously, we demonstrated the interactions of these ILMs during single or pairwise activation, which showed a range of glottal channel shapes dependent on the muscle pairing and level of activation.[8] Although the effects of single and paired

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muscle activation on the medial surface shape have been described, the effects of concurrent activation of all ILMs, as occurs in vivo to set glottal posture,[14,15] have not been investigated. Although computational studies have been used to study control of medial surface shape, they have been limited in the number of concurrent ILM interactions.[16–18] In the current study, we evaluated vocal fold medial surface shape as a function of concurrent activation of all phonatory ILMs to assess glottal posture changes in a more physiologic activation state compared to prior studies. As the LCA adducts the posterior glottis and stabilizes the cricoarytenoid joint to allow CT and TA muscles to interact and control the shape and stiffness of the body and cover layer during phonation,[10] we particularly focus on the interactions between TA and CT as a function of LCA/IA activation level. We hypothesize that the pre-phonatory posture is a well-adducted glottis with a uniform and rectangular medial surface contour.

METHODS

Hemilarynx Model

This study was approved by the Institutional Animal Care and Use Committee. One male mongrel canine was used. Hemilarynx surgery and experimental set-up were detailed previously and relevant sections are briefly described.[19,20] A left hemilaryngectomy was performed and a grid of 33 India ink landmarks was tattooed onto the right vocal fold medial surface (Figure 1B). A transparent right-angle glass prism was positioned with its hypotenuse parallel to glottal midline at a distance so the

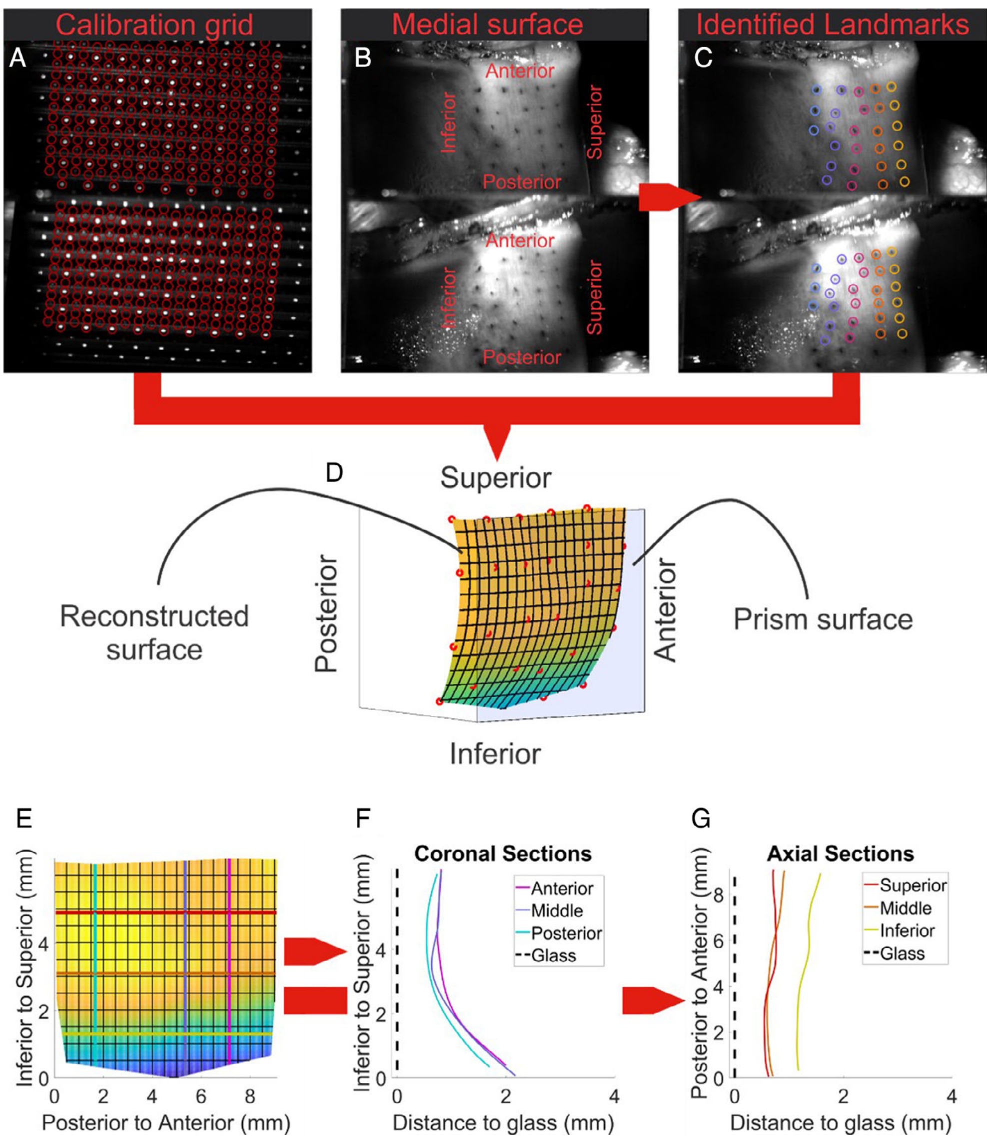

Fig. 1. Experimental setup and generation of reconstructed surface and parameters. (A) Calibration plate. (B) Marked right hemilarynx with prism-generated stereoview. (C) Identified landmarks in custom software of vocal fold. (D) Reconstructed surface with prism. (E) Reconstructed surface with coronal and horizontal section planes marked. (F) Coronal sections generated across anterior, middle, and posterior vocal fold length. (G) Axial sections generated across superior, middle, and inferior vocal fold medial surface. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

vocal fold did not contact the prism during maximal adduction (0.5 mm lateral to glottal midline). This allowed imaging of the medial surface without potential deformation induced by contacting the glass prism. The two prism faces provided two distinct stereo views of the same vocal fold that were used for 3D reconstruction (Figure 1B).

Neuromuscular Conditions Tested

Each ILM was activated via neuromuscular stimulation of respective nerve branches. The recurrent laryngeal nerve (RLN) was identified 5 cm below the larynx and followed distally to the posterior cricoarytenoid (PCA) muscle nerve branch, which was transected to eliminate its abductory effects. The RLN was dissected to the TA branch, which was suture ligated distal to its takeoff and attached to a cuff electrode. A cuff electrode was placed around the RLN trunk for activation of the LCA/IA muscle complex. The superior laryngeal nerve (SLN) was identified, and a cuff electrode was applied to the external (motor) branch for CT muscle activation.

Each neuromuscular stimulation lasted 1500 ms with 1 ms cathodic pulses at 125 Hz for RLN branches and 75 Hz for the SLN, followed by a 3-second rest. ILM activation was performed over four equally graded levels from threshold activation (just a hint of motion) to maximal activation (saturation of vocal fold movement). Vocal fold motion and deformation were captured at 3,000 frames per second using a high-speed digital camera (Phantom v210, Vision Research Inc., Wayne, NJ). The distance from the camera to the prism and larynx was held constant. The frame for analysis of glottal posture was selected at 200 ms after muscle activation to allow adequate time for setting of final pre-phonatory posture based on prior findings of ILM dynamics.[8,21]

3D Reconstruction Method

Figure 1 illustrates the experimental design for surface landmark tracking, 3D reconstruction, and parameter extraction.

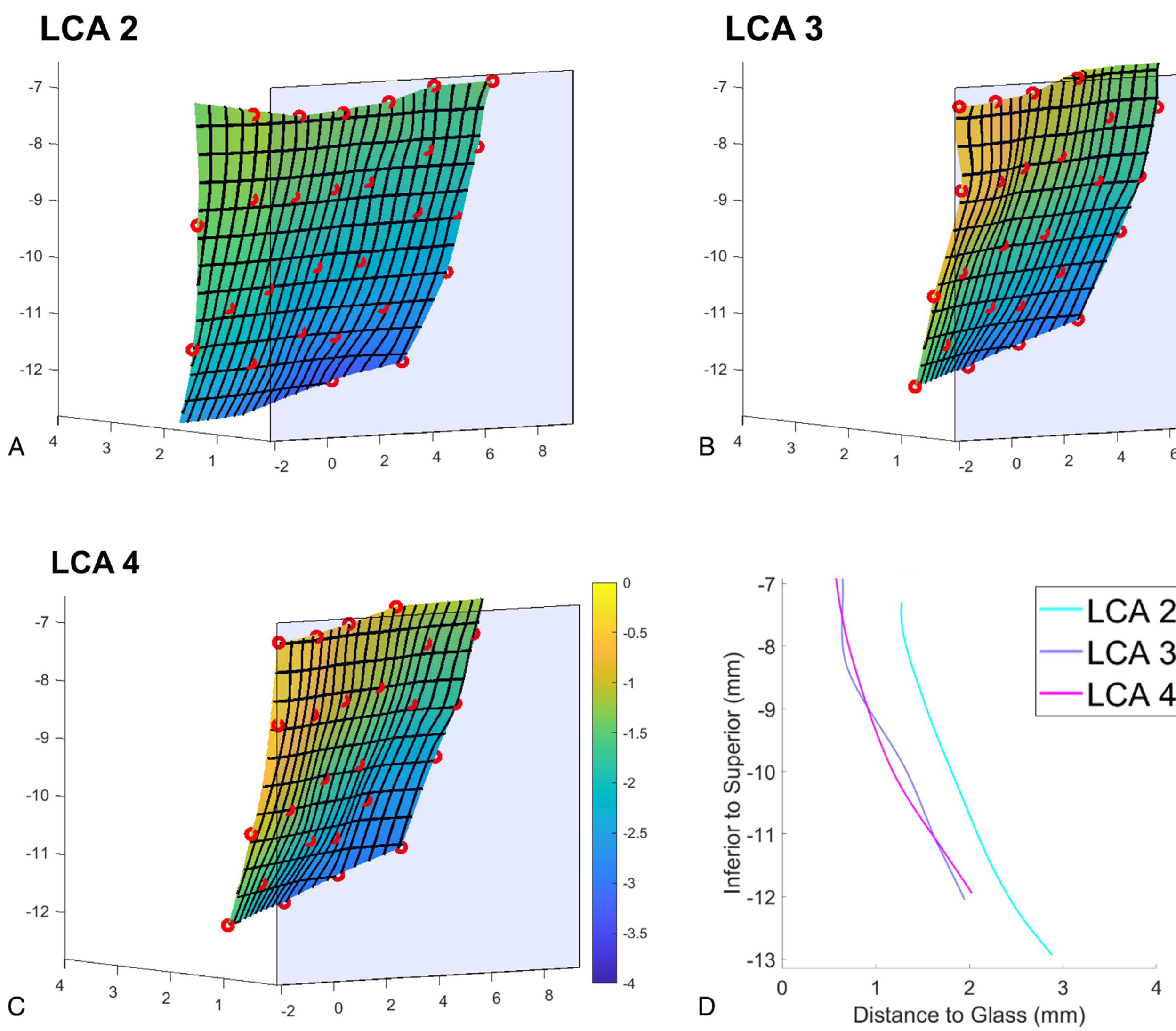

Fig. 2. Effects of increasing LCA/IA activation at low levels of CT and TA (Level 1). (A) LCA 2, (B) LCA 3, (C) LCA 4). (D) Coronal sections at the posterior vocal fold with increasing LCA activation (CT 1, TA 1). LCA activation resulted in adduction of the posterior vocal fold (A–C) without change in the medial surface shape (D). Colormap demonstrates distance from vocal fold to prism. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

The calibration plate was placed directly in contact with the glass prism (Figure 1A). Vocal fold surface was reconstructed using manually tracked landmarks (Figure 1C) using custom software (GLabel, Friedrich Alexander University Erlangen-Nürnberg) by two authors (one for initial marking, one for review) to minimize marking error. Subsequently, the 3D positions of landmarks were reconstructed using a custom algorithm based on the work of Döllinger et al.[22] 3D surface shape between landmarks was then reconstructed using cubic spline interpolation (Figure 1D).[23] Interpolation error was minimized by interpolating from the grid of 33 known points. A potential linearization error may be introduced if the camera was not perpendicular to the prism leading to distortions of the surface in the x- and y-direction. Although angle between the camera and prism was not measured, the camera appeared to be perpendicular to the prism. To assess the accuracy of reconstruction methodology, a comprehensive study reconstructing objects of known dimensions up to 15 mm and at various camera angles from the calibration plate was performed to assess shift and deformation (unpublished data). Based on these estimations, the maximum error of reconstructed points 4 mm from prism surface is expected to be <0.2 mm deviation. This error is low enough to allow for accurate reconstruction of the 3D surface shape.

The medial surface contour was visualized at fixed landmarks at the anterior, mid-membranous, and posterior coronal sections (Figure 1E). Activation levels 1 and 2 elicited similar effects across all ILMs (suggesting inadequate graded stimulation between those levels) and are, therefore, presented interchangeably. Vocal fold length was defined as the distance between the anterior-most and posterior-most landmarks of the vocal fold at the superior edge. Vocal fold adduction was measured at the superior edge of both the mid-membranous and posterior vocal fold. Vocal fold adduction was defined as the distance between the vocal fold and the glass prism. A smaller distance indicated a greater degree of adduction. Axial sections were generated at the superior, middle, and inferior aspects of the vocal fold (Figure 1G). The convergence measure was defined as the distance between the superior and inferior axial sections with a smaller distance between the sections indicating a more rectangular glottal shape and a larger distance indicating a more convergent shape.

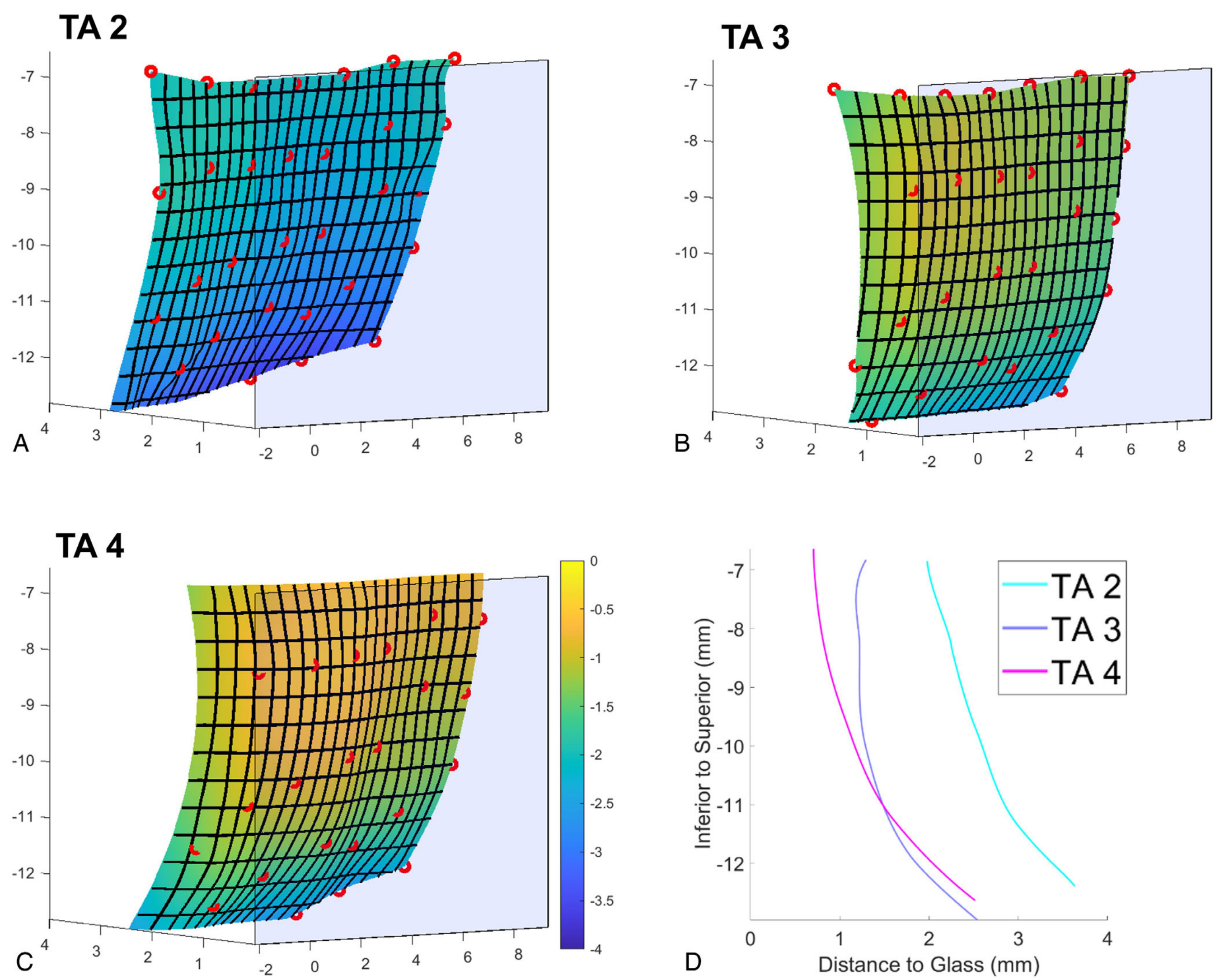

Fig. 3. Effects of increasing TA activation at low levels of CT and LCA (Level 1). (A) TA 2, (B) TA 3, (C) TA 4, (D) Coronal sections at the mid-membranous vocal fold. TA activation resulted in adduction at the mid-membranous vocal fold (A–C) and transition to a rectangular glottal shape (D). [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

Statistical Analysis

Kendall rank correlation coefficients were calculated to explore relations between laryngeal posture measures. To better delineate the roles of the ILMs in determining laryngeal posture, linear regression models were developed for glottal adduction, length, and convergence measures. The false discovery rate (the expected percentage of false positive tests) was controlled at 5% using the Benjamini–Yekutieli procedure to factor in potential unknown dependencies between parameters.[24] All statistical analyses were completed in RStudio (Version 1.3.959).

RESULTS

ILM Effects on Posture

A total of 64 pre-phonatory medial surface shapes were evaluated across combinations of four activation levels for each ILM (4 TA × 4 CT × 4 LCA/IA levels). Successful neuromuscular stimulation was confirmed by reviewing posture changes on high-speed video. At rest, the vocal fold medial surface shape was convergent and consistent with prior studies.[9] TA activation resulted in inferomedial bulging and glottal adduction, LCA/IA activation resulted in adduction of the posterior glottis, and CT activation resulted in vocal fold lengthening.[8,9] These findings confirmed appropriate neuromuscular activation. However, activation levels 1 and 2 (lower activation) resulted in similar postures, suggesting suboptimal graded activation between those two levels. Levels 1 and 2 are presented interchangeably. The interactions of the ILMs on shape were then evaluated.

Generally, the glottal pre-phonatory shape was rectangular or convergent depending on the relative activation levels of ILMs. LCA/IA activation adducted the entire vocal fold without visibly changing the medial surface shape. The posterior vocal fold was adducted more than the mid-membranous vocal fold (Figure 2A–C). In contrast, TA affected both vocal fold adduction and medial surface contour. At low levels of other ILM activation, TA resulted in adduction, mid-membranous inferomedial bulging, and contour shape change from a convergent to a rectangular glottis (Figure 3). Increasing TA at low LCA/IA activation resulted in enhancement of mid-membranous infero-medial bulging (Figure 4A), which was diminished at high levels of LCA/IA as the entire vocal fold became more medialized and the entire vocal fold surface contour became more uniform and rectangular (Figure 4B).

The effects of CT activation on medial surface shape were apparent only in the mid-membranous vocal fold and particularly at concurrent high levels of TA and low levels of LCA/IA activation (Figure 5). At high TA and low LCA/IA, increasing CT activation appeared to slightly abduct the vocal fold superior surface and generated a very slightly divergent contour (Figure 5A). This effect was diminished at higher LCA/IA levels (Figure 5C). When both adductors were concurrently activated at low levels, the medial surface remained convergent throughout and CT activation had no effects. Similarly, when both adductors were concurrently activated at high levels, the medial surface remained rectangular and CT activation had no effects.

TA and CT interactions at higher LCA activation (i.e., fully or nearly fully adducted at the vocal process) are expected to be the physiologic pre-phonatory posture for most voice and speech types. Figure 6 illustrates CT/TA interactions at high LCA activation. Increasing TA activation created a more rectangular glottal shape. Increasing CT activation slightly abducted the glottis with minimal changes in glottal shape.

Interactions between Laryngeal Posture Measures

Exploratory analysis between outcome measures was performed using Kendall correlation. The mid-membranous and posterior glottal gap measures correlated well ($\tau = 0.80$, $p < 0.05$). There was no correlation between vocal fold length and glottal gap measures

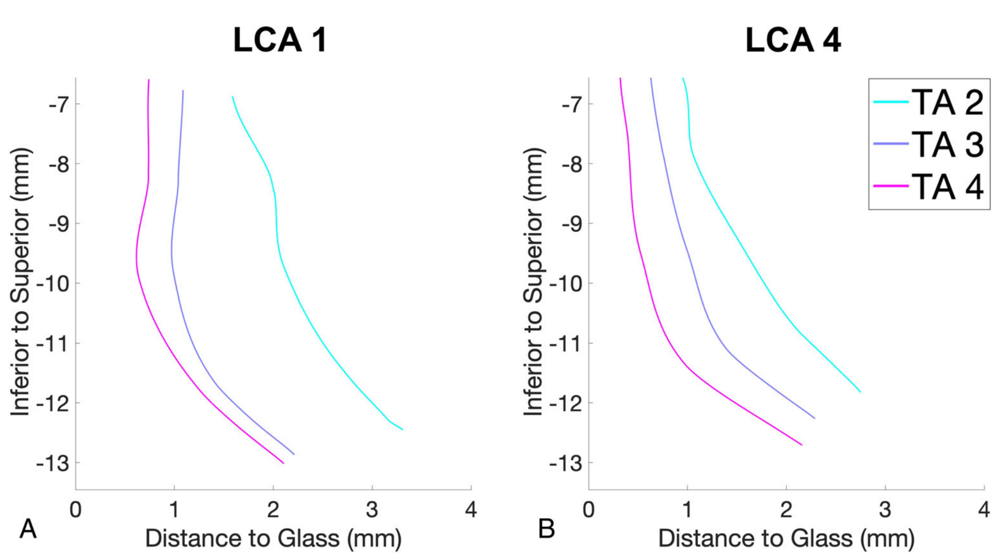

Fig. 4. Effects of increasing TA activation at lower and higher LCA activations. (A) LCA 1, (B) LCA 4. Inferomedial bulging apparent with higher TA activation was diminished with higher LCA activation. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

(τ = 0.24 and 0.11 for mid-membranous and posterior gap, respectively). There was no correlation between vocal fold length and medial surface contour ($\tau = 0.27$).

Predictive Models of Outcome Measures

To better delineate the roles of the ILMs in determining laryngeal posture, linear regression models were

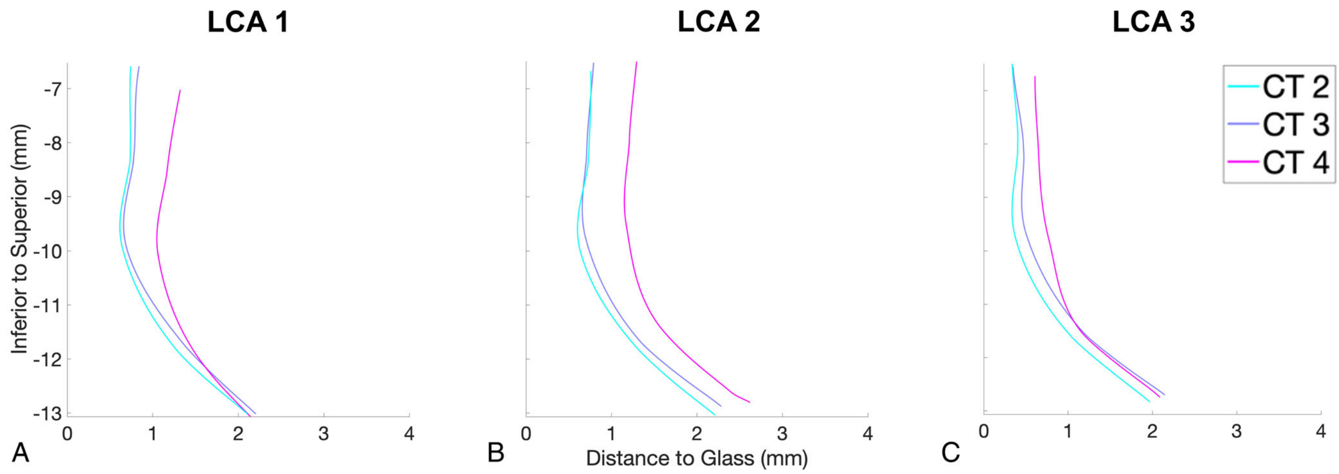

Fig. 5. Effects of increasing CT activation at high TA activation (Level 4) as a function of increasing LCA activation. (A) LCA 1, (B) LCA 2, (C) LCA 3. CT activation at low LCA resulted in abduction of the superior edge of the vocal fold and the formation of a slightly divergent glottis (A, B). This effect was absent at increased LCA activation (C). [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

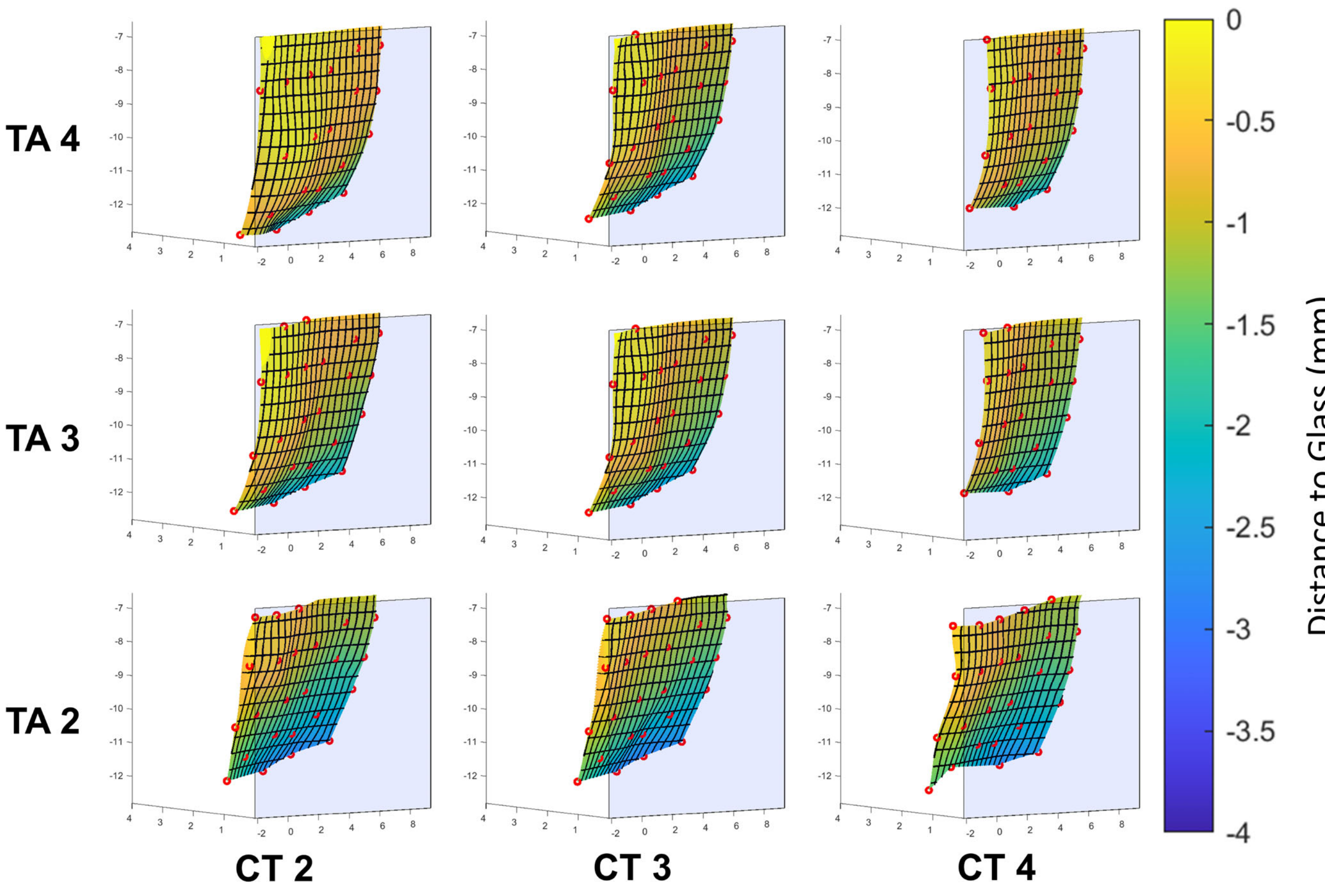

Fig. 6. Interactions between CT and TA at higher LCA activation (LCA 4). Increasing TA activation resulted in mid-membranous bulging, although increasing CT activation showed no effect on medial surface contour. Colormap demonstrates distance from vocal fold to prism. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

TABLE I.
Linear Predictive Model of Laryngeal Posture Measures.

Outcome measure	Muscle CT	LCA/IA	TA
Length (mm)	0.09 (0.009)*	0.05 (0.009)*	−0.11 (0.009)*
Mid-membranous adduction (mm)	−0.07 (0.024)*	0.24 (0.025)*	0.23 (0.024)*
Posterior adduction (mm)	−0.08 (0.027)*	0.40 (0.027)*	0.23 (0.027)*
Convergence (mm)	−0.03 (0.110)*	0.02 (0.110)	−0.09 (0.110)*

Note: Linear predictive model of laryngeal posture measures. Effect estimates (Standard Error) are provided. Asterisk denotes significance $p < 0.05$. Negative effect estimates for adduction measures indicate decreasing adduction (increased glottal gap) with increasing CT activation.

developed for length, convergence, and glottal gap measures (Table I). Vocal fold length showed the greatest increase per activation level of CT followed by LCA. TA showed the greatest effect on vocal fold length with a greater decrease in vocal fold length per unit TA activation than increase in length per unit CT activation. The medial surface contour was determined by TA. The posterior glottal gap was closed most effectively by LCA activation, followed by activation of the TA. In contrast, LCA and TA were equally effective in closing the mid-membranous gap. At both the mid-membranous and posterior glottis, CT activation resulted in slight abduction of the vocal fold.

DISCUSSION

Neuromuscular activation of the ILMs determines the pre-phonatory posture. Previously, laryngeal posture has been described in two dimensions: anteroposterior length and mediolateral excursion. In this work, we highlight the third dimension of laryngeal posture, the vocal fold superoinferior medial surface, which serves as the origin of the mucosal wave.[25,26] Previous studies on pre-phonatory posture were limited to evaluating effects of individual or pairs of ILMs.[8] In this study, we extend those findings by focusing on the interactions between concurrent activation of all the ILMs and reconstructed the resulting vocal medial surface shape. Thus, assessment of a more physiologic posture was possible. Although our findings are consistent with previous investigations on ILM effects on vocal fold medial surface shape[8,9] the current study refined previous results and found that TA activation resulted in the generation of a rectangular glottal configuration with mid-membranous bulging and that the addition of LCA contributed to a more adducted and uniform medial surface contour.

The goal of phonosurgery is to recreate the “plane, position, and contour of the normally adducted vocal fold.”[27] Understanding the three-dimensional vocal fold pre-phonatory posture allows for targeted surgical intervention for glottal incompetence that expands beyond the current approach to the vocal fold as a two-dimensional structure. Current paradigm of medialization techniques, such as injection laryngoplasty and medialization thyroplasty, is defined by the goal of ameliorating the superiorly-visualized glottal gap.[28] More recently, some have proposed a goal of generating a less convergent and more rectangular glottal configuration.[29] However, the underlying physiologic medial surface posture of the vocal fold was not understood.

With limited understanding of the physiologic pre-phonatory posture, phonosurgical procedures may have high rates of poor voice outcomes. Medialization thyroplasty, for example, has a 6% revision rate, and up to 10% of phonosurgeons have reported inadequate improvement in voice quality after medialization thyroplasty or arytenoid adduction.[30,31] Endoscopic evaluation after unsuccessful thyroplasty may show adequate closure at the superior edge of the vocal fold,[28] suggesting that adequate glottal closure at the superior edge is not the sole determining factor for successful voice outcomes. Failure of medialization procedures, then, may also be due to inadequate inferomedial glottal closure or suboptimal medial surface contour. MRI studies following medialization surgery suggest that failure of the medial edge of the implant to conform to the deficit may be a cause of unsuccessful surgery.[32] Medial surface bulging due to TA activation is expected to create a large rectangular medial surface area over which the mucosal wave can propagate.[5] Given the critical role of the medial surface in mucosal wave generation and propagation, phonosurgical techniques that do not address this aspect of voice production may be prone to poor voice outcomes.

This study evaluated the pre-phonatory posture resulting from activation of the phonatory ILMs to better define goals in phonosurgical interventions. In the future work, we aim to incorporate vocal fold vibration and study the resulting acoustics. The animal model used in this study introduces certain limitations. The in vivo canine model is non-human, but the canine larynx reasonably approximates the human larynx in anatomy and physiology and allows systematic activation of the ILMs not possible in humans.[33,34] Slight histological differences, such as the absence of a vocal ligament in the canine, are not expected to affect our laryngeal posture outcomes when the ILMs serve the same functions in the human and canine larynx. The neuromuscular activation conditions we used may not reflect fine patterns of individual muscle control in physiologic phonation but individual stimulation of muscles and their evaluation in combination may represent a reasonable approximation for physiologic phonation. Due to ethical considerations of the use of multiple large animals, the minimum number of animals required to test/retest experimental conditions and confirm data patterns are used. As such, voice research relying on large animals emphasizes data trends and adds to previous findings. Our current findings are experimentally robust, consistent with previous studies, and reflect fine experimental control of laryngeal muscle activation and their effects on pre-phonatory posture in an in vivo larynx. However, although the generalizability of results from a single subject is limited, it should be considered together with findings from previous results with individual and paired activation of ILMs.[8,9] The results of the current study are consistent with prior research but also add new findings to this body of literature.

CONCLUSION

The pre-phonatory glottal posture is set by stimulation of the ILMs when activated in combinations across various levels. We performed systematic activation of the laryngeal muscles and quantitative evaluation of resulting medial surface shape in an in vivo model. The TA is the primary determinant of rectangular medial surface shape. LCA adducts the posterior vocal fold and maintains a convergent glottal shape but is synergistic with TA to maintain a smoother and rectangular medial surface shape. The pre-phonatory postures described in this study may provide insight into phonosurgical management. For example, surgeries that affect the medial surface of the vocal fold, such as injection laryngoplasty and medialization thyroplasty, may benefit from consideration of the pre-phonatory postures that are generated by the ILMs.

FUNDING INFORMATION

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