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The role of thyroarytenoid muscles in regulating glottal closure in an in vivo canine larynx model

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This study investigated the effectiveness of individual laryngeal muscles in regulating the mean glottal flow and glottal closure pattern during phonation in an in vivo canine larynx model. Phonation experiments were performed with parametric stimulation of the thyroarytenoid (TA), lateral cricoarytenoid (LCA), interarytenoid (IA), and the cricothyroid (CT) muscles. For each stimulation level, the subglottal pressure was gradually increased to produce phonation. The subglottal pressure, the volume flow, and the outside acoustic pressure were measured together with high-speed recording of vocal fold vibration from a superior view. The results show that the TA muscle played a dominant role in regulating both the membranous glottal width and the glottal closure pattern during phonation, indicating an important role of the TA muscle in regulating voice quality. The TA muscle activation was also the most effective in regulating the mean glottal flow, and thus an important laryngeal adjustment in airflow conservation, particularly at high subglottal pressures or loud voice production, although increasing TA activation decreased the vocal intensity. This study also presented a complete set of data on muscular control of the glottal width and voice production, which can be used in validation of computational models of vocal fold posturing and voice production.

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1. INTRODUCTION

Recent numerical and experimental studies have shown that the medial surface shape, particularly the vertical thickness of the vocal folds plays an important role in voce production and control. Increasing vertical thickness has been shown to be an effective laryngeal mechanism in maintaining the adductory position of the vocal folds against the subglottal pressure and conserving airflow expenditure during phonation (Zhang, 2016; Desjardins et al., 2021), particularly at high subglottal pressure or loud voice production. More importantly, changes in the vertical thickness have an important effect on the closed quotient (the fraction of one oscillation cycle that the glottis remained closed) of vocal fold vibration (van den Berg, 1968; Sundberg and Hogset, 2001; Zhang, 2016; Zhang & Chhetri, 2019; Wu and Zhang, 2019), and thus are essential to producing voice qualities ranging from breathy, modal, to pressed.

Considering that the thyroarytenoid (TA) muscle is the primary muscle in regulating the vertical thickness of the vocal folds (Hirano, 1988; Zhang, 2016b), these studies suggest that the TA muscle plays an important role in maintaining sufficient vocal fold adduction, conserving airflow expenditure, and regulating the glottal closure pattern and voice quality. While there have been many studies on laryngeal muscular control of phonation (e.g., Hirano et al., 1969, 1970; Tanaka and Tanabe, 1986; Kempster et al., 1988; Titze et al., 1989; Mendelsohn et al., 2015), they often focused on the effect on fundamental frequency and vocal intensity. Few studies attempted to isolate and investigate the role of individual laryngeal muscles in the control of the glottal airflow and the glottal closure pattern during phonation, largely due to difficulties in accessing the larynx in human subjects or reproducing the effect of TA muscle stimulation in excised larynges. One exception was the in vivo canine experiment by Choi et al. (1993), which showed a dominant effect of the TA muscle on the open quotient of vocal fold vibration. However, the experiment was performed only for a few selected laryngeal stimulation conditions, and the interaction between different laryngeal muscles was not fully investigated.

The goal of this study was to investigate the effectiveness of individual intrinsic laryngeal muscles, particularly the TA, in regulating the glottal flow and glottal closure patterns in an in vivo canine larynx model. Unlike excised larynx or human subject experiments, the in vivo canine larynx model with graded nerve stimulation allows systematically stimulating individual laryngeal muscles, particularly the TA muscle, and observing its effect on voice production. Our previous studies using this model (Chhetri et al., 2012, 2014) focused on phonation characteristics at onset, and the glottal closure pattern was not investigated. Specifically, in this study, we parametrically stimulated the TA muscle, the cricothyroid (CT) muscle, the lateral cricoarytenoid (LCA) muscle, and the interarytenoid (IA) muscle, and quantified the effect on the glottal airflow and glottal closure pattern, as measured by the closed quotient.

A second goal of this study was to provide a complete data set of laryngeal muscular control of vocal fold geometry and voice production, for the development and validation of computational models of phonation. In particular, although continuum models of laryngeal muscle activation have been developed (Hunter et al., 2004; Yin and Zhang, 2014, 2016; Geng et al., 2020), validation of these laryngeal muscle models is often difficult due to the lack of experimental data for comparison. On the other hand, due to the high nonlinearity of the vocal fold tissue properties and large deformation involved in vocal fold posturing, such continuum models are computationally expensive. A more quantitative description of laryngeal muscular control of vocal fold posturing, together with mechanical testing data of the vocal folds (Alipour-Haghighi and Titze, 1991; Kelleher et al., 2013; Zhang et al., 2017) would lead to the development of empirical models of vocal fold posturing (e.g., Titze and Story, 2002) that more accurately relate laryngeal muscle activation levels to vocal fold properties of importance to voice production such as vocal fold length, glottal width, and the anisotropic stiffness conditions.

2. METHODS

The in vivo canine larynx model study protocol was approved by UCLA Institutional Animal Research Committee. The larynx was exteriorized in the neck via suprahyoid pharyngotomy and supraglottic laryngectomy to improve access to the larynx for high-speed video recording. The recurrent laryngeal nerves (RLNs) were identified and nerve branches to Galen's anastomosis and posterior cricoarytenoid (PCA) muscles were divided and followed distally until the TA and LCA/IA branches were identified. The TA branches were tied off with silk sutures and tripolar cuff electrodes (Ardiem Medical, IN, PA) were placed distally to activate the TA muscles. The LCA/IA muscles were stimulated through electrodes placed on the main RLN nerves about 5 cm from the larynx after the TA branches were tied off. The CT muscles were stimulated by electrodes placed

on the external branches of the superior laryngeal nerves (SLNs). The internal sensory branches of the SLN were cut bilaterally. For each muscle, the nerve stimulation range from threshold to maximal activation was first determined. The stimulation range was then divided into 8 equal-spaced levels (0-7) for the LCA/IA and CT muscles, and 5 levels (0-4) for the TA muscles. A total of 320 stimulation conditions were investigated, each with a stimulation period of 1,500 milliseconds with 100 microsecond-long rectangular unipolar cathodic pulses at pulse repetition rates of 100 Hz. Each stimulation condition was followed by a 3.5 second pause to allow muscle recovery.

For each stimulation condition, the volume flow Q was increased linearly from 300 ml/s to 1400 ml/s within the 1.5 seconds. The outside acoustic sound was recorded using a probe microphone (B&K model 4128) at a sampling rate of 50 kHz, from which the fundamental frequency (F0) and output sound pressure level (SPL) were extracted. The mean subglottal pressure was measured using a pressure transducer (MKS Baratron 220D, MKS Instruments) mounted flush with the inner wall of the subglottal tracheal tube, from which the glottal resistance (GR, the ratio between the mean subglottal pressure and mean flow rate) was calculated. From the high-speed recordings of vocal fold vibration, the glottal area function was extracted using a region-growing algorithm, similar to Lohscheller et al. (2008). The glottal area function was used to calculate the closed quotient (CQ) or the fraction of one oscillation cycle that the membranous glottis remains closed. In addition, feature points were created on the vocal folds by ink during experimental preparation (Fig. 1a), which were used to track vocal fold motion and estimate the glottal gap width along the medial-lateral direction and vocal fold elongation in the anterior-posterior direction, as illustrated in Fig. 1b.

Two larynges were investigated in this study. Despite of some differences, the observations in the two larynges were qualitatively similar. For clarity, only results from one of the larynxes are presented in this study.

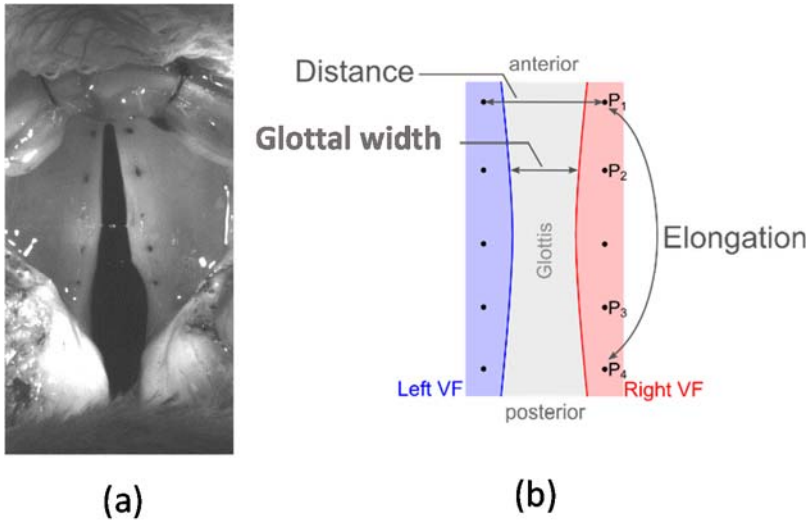

Figure 1. (a) Superior view of the vocal folds with feature points that were used to calculate vocal fold elongation and glottal gap width at different anterior-posterior locations.

3. RESULTS

A. MUSCULAR CONTROL OF GLOTTAL WIDTH AND VOCAL FOLD ELONGATION

Fig. 2 shows the glottal width under different conditions of laryngeal muscle activation at four anterior-posterior locations, with each location corresponding to an anterior-posterior location marked by a straight line connecting a pair of feature points on the left and right vocal folds (Fig. 1b). The glottal width at the location corresponding to the middle pair of suture points was not calculated because the suture points were smaller in size and thus difficult to track. Fig. 2 shows that for this particular larynx, the glottal width was primarily controlled by the TA muscle, which had a dominant effect on both the anterior and posterior glottal width. For low levels of TA activation, the LCA/IA muscle activation also significantly reduced the glottal width,

particularly at the posterior membranous glottis. The effect of CT muscle activation on the glottal width was generally small, with increasing CT activation increasing the posterior glottal width but decreasing the anterior glottal width.

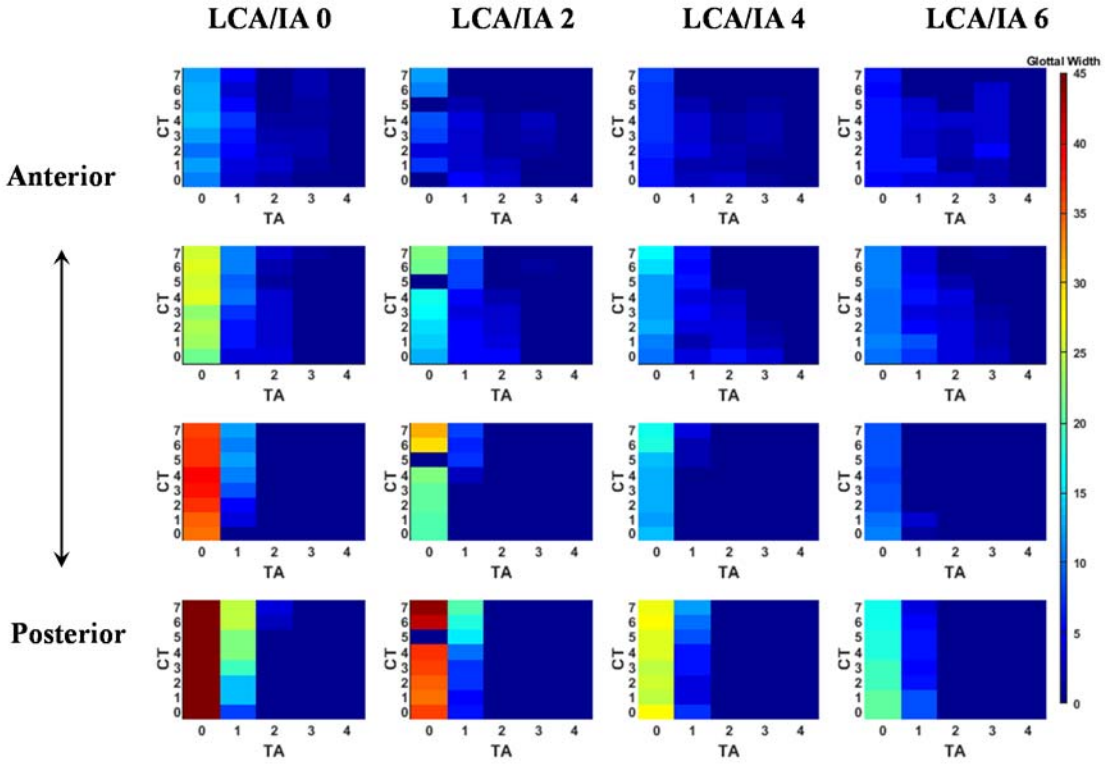

Figure 2. Prephonatory glottal widths in pixels at four anterior-posterior locations at different stimulation conditions of the TA, CT, and LCA/IA muscles.

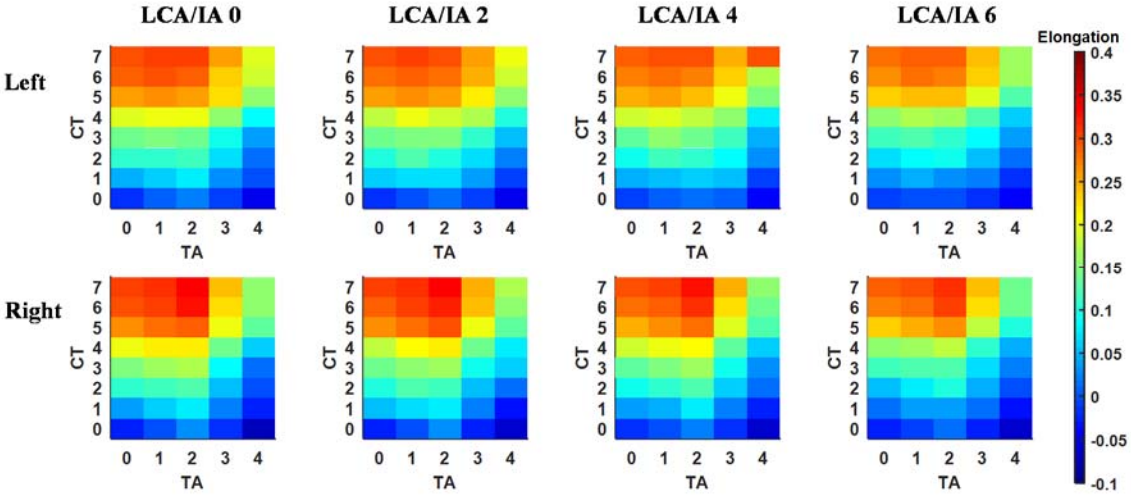

Figure 3. Elongation of the left (top) and right (bottom) vocal folds at different stimulation conditions of the TA, CT, and LCA/IA muscles.

Fig. 3 shows vocal fold elongation (changes in vocal fold length along the anterior-posterior direction) under different muscle stimulation conditions. Vocal fold elongation was estimated by changes in the distance between the most-anterior feature points and the most-posterior feature points. This change in distance was further normalized by the original distance at condition of no muscle activation. Vocal fold elongation varied between about -0.1 (shortened by 10%) and 0.4 (elongation by 40%). Elongation was primarily controlled by the CT muscle, and to a lesser degree, the TA muscle, with elongation increasing with increasing CT activation and decreasing TA activation. Note that noticeable vocal fold shortening occurred only at high levels of TA activation. Taken together with figure 2, this showed that TA activation adducted the vocal folds at low activation levels and shortened the vocal folds at high activation levels. Activation of the LCA/IA muscles slightly decreased vocal fold elongation, but this effect was almost negligible.

B. MUSCULAR CONTROL OF THE MEAN GLOTTAL FLOW, GLOTTAL RESISTANCE, AND GLOTTAL CLOSURE

Figure 4 shows the mean glottal flow at different conditions of laryngeal muscle activation and subglottal pressures. Note that the regions in the figure without data indicate conditions for which either no vibration was observed or the desired subglottal pressure was not reached in the flow range between 300ml/s and 1400 ml/s. As expected, the mean glottal flow increased with increasing subglottal pressure. This effect of increasing subglottal pressure on the glottal flow can be effectively counterbalanced by activation of the TA muscle and the LCA/IA muscles. In comparison, the effect of CT muscle activation on the mean glottal flow was smaller.

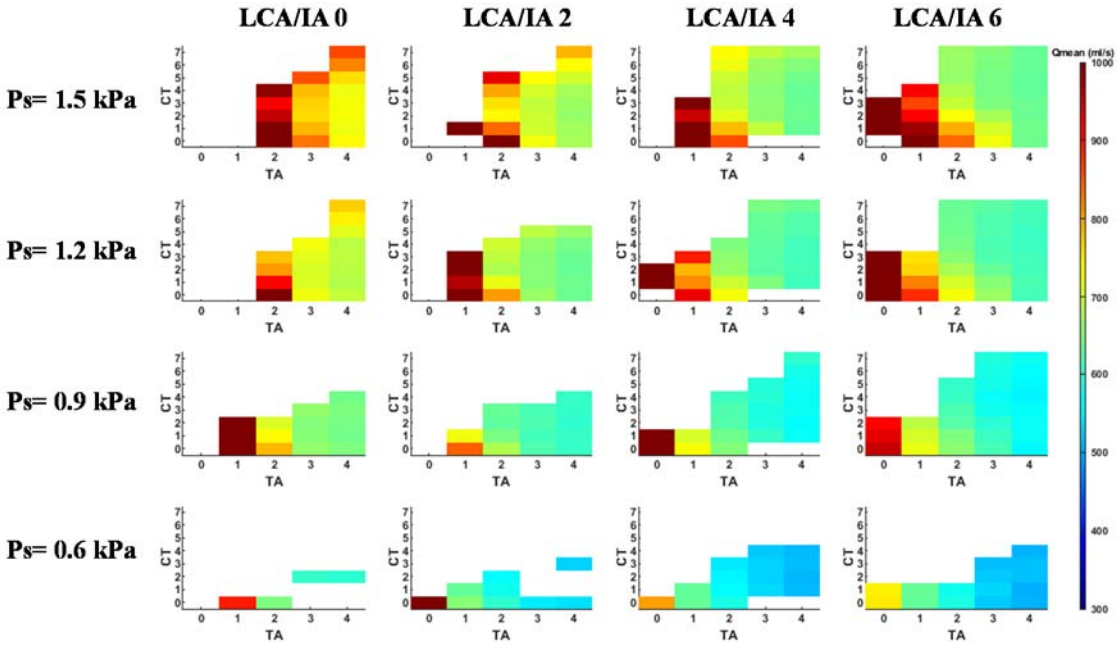

Figure 4. The mean glottal flow rate at different stimulation conditions of the TA, CT, and LCA/IA muscles and subglottal pressures.

The relative effectiveness of different laryngeal muscles in regulating the glottal flow is more clearly illustrated in the top panel of figure 5, which shows the mean glottal flow as a function of the subglottal pressure when each intrinsic laryngeal muscle was individually stimulated. For a given subglottal pressure, TA activation clearly had the largest impact on the mean glottal flow, whereas CT activation had the least and almost negligible effect. The dominant effect of the TA muscle activation on the mean glottal flow was further confirmed in a multiple linear regression shown in Table 1.

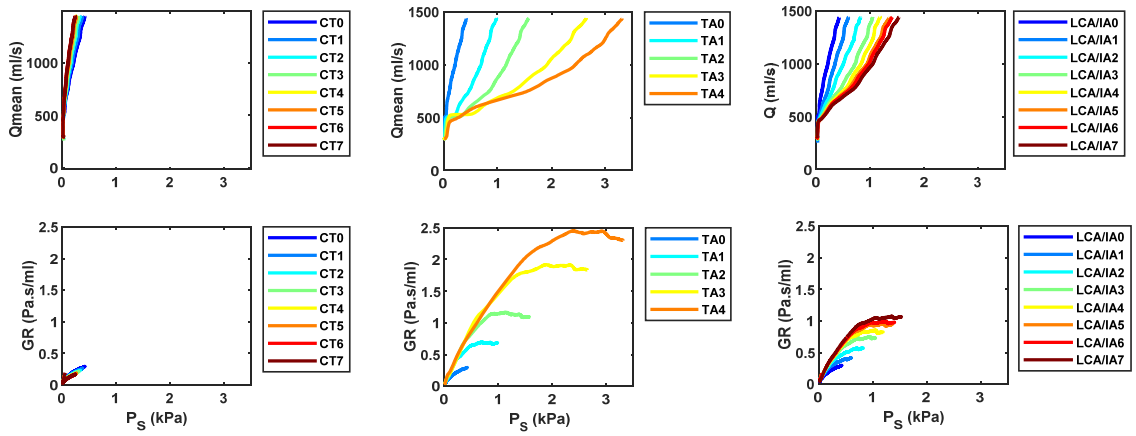

Figure 5. Mean glottal flow (top) and glottal resistance (bottom) as a function of the subglottal pressure at selected conditions of the TA, CT, and LCA/IA muscle activation.

Table 1. Standardized coefficients and R2 values of multiple linear regressions between the independent variables (subglottal pressure Ps and activation of the TA, CA, LCA/IA muscles) and four output measures of voice production (mean glottal flow Qmean, glottal resistance GR, closed quotient CQ, SPL, and F0). All parameters were statistically significant with p<0.005 unless denoted by *.

Parameter	Qmean	GR	CQ	SPL	F0
Ps	0.554	0.707	-0.126	0.769	0.028*
TA	-0.699	0.469	0.617	-0.257	-0.132
CT	-0.042	0.037	0.257	-0.187	0.737
LCA/IA	-0.310	0.242	-0.107	0.325	0.070
R^2	0.692	0.849	0.499	0.713	0.546

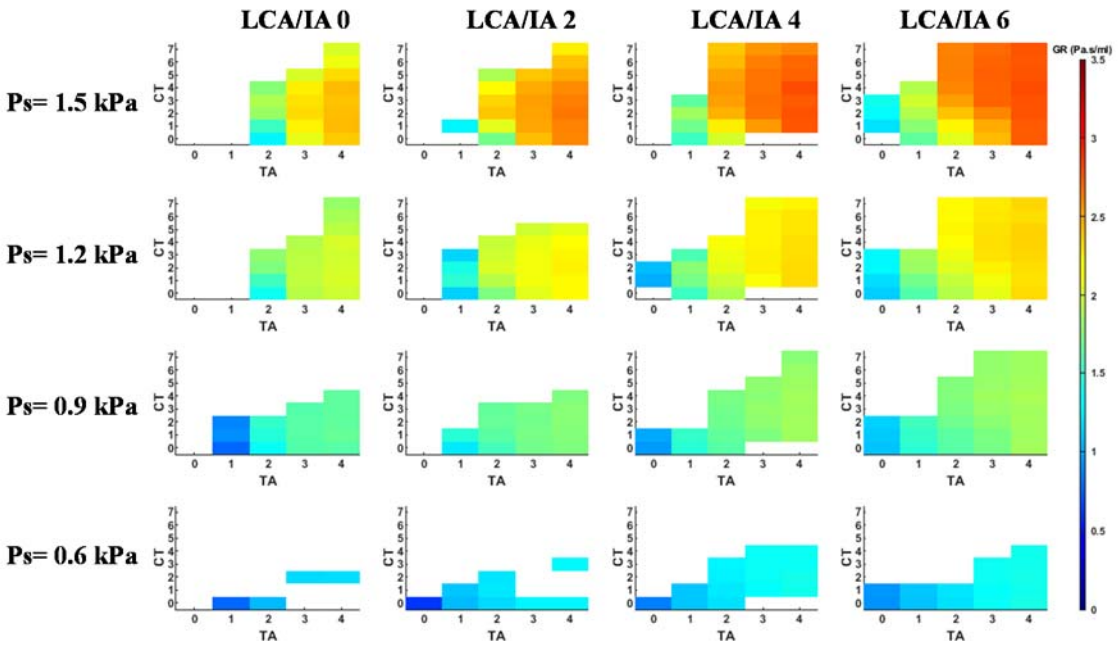

Figure 6. The glottal resistance (GR) at different stimulation conditions of the TA, CT, and LCA/IA muscles and subglottal pressures.

The glottal resistance followed a similar pattern as the mean glottal flow, as shown in figure 6. The subglottal pressure had the largest effect, followed by the TA and LCA/IA muscles. The CT muscles had the smallest effect. It is worth noting that the glottal resistance increased significantly with increasing subglottal pressure, even without any simultaneous laryngeal adjustments. Thus, an increase in the glottal resistance does not always indicate increased laryngeal adduction.

Figure 7 shows the closed quotient of vocal fold vibration at a function of the subglottal pressure and laryngeal muscle activation. One consistent observation across different conditions was that the closed quotient increased with increasing TA activation, which was also confirmed in the multiple linear regression in Table 1. Increasing CT activation also appeared to increase the closed quotient, especially at low levels of CT activation. However, the effect was much weaker than that of the TA muscles.

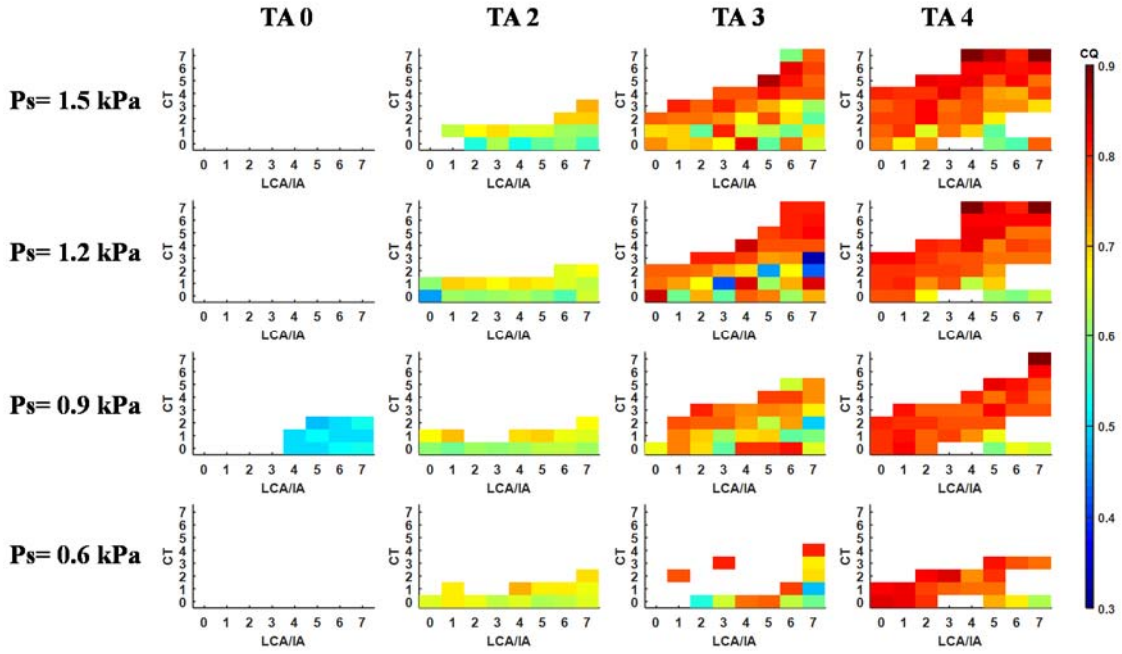

Figure 7. The closed quotient of vocal fold vibration at different stimulation conditions of the TA, CT, and LCA/IA muscles and subglottal pressures.

C. MUSCULAR CONTROL OF VOCAL INTENSITY AND FUNDAMENTAL FREQUENCY

Figure 8 shows the vocal intensity under different muscle activation and subglottal pressure conditions. Consistent with previous studies, vocal intensity was primarily controlled by the subglottal pressure. In comparison, the effect of muscle activation was smaller. In general, the vocal intensity increased slightly with increasing the LCA/IA activation. In contrast, the vocal intensity generally decreased with increasing activation of the TA or CT muscles, particularly for low levels of LCA/IA activation.

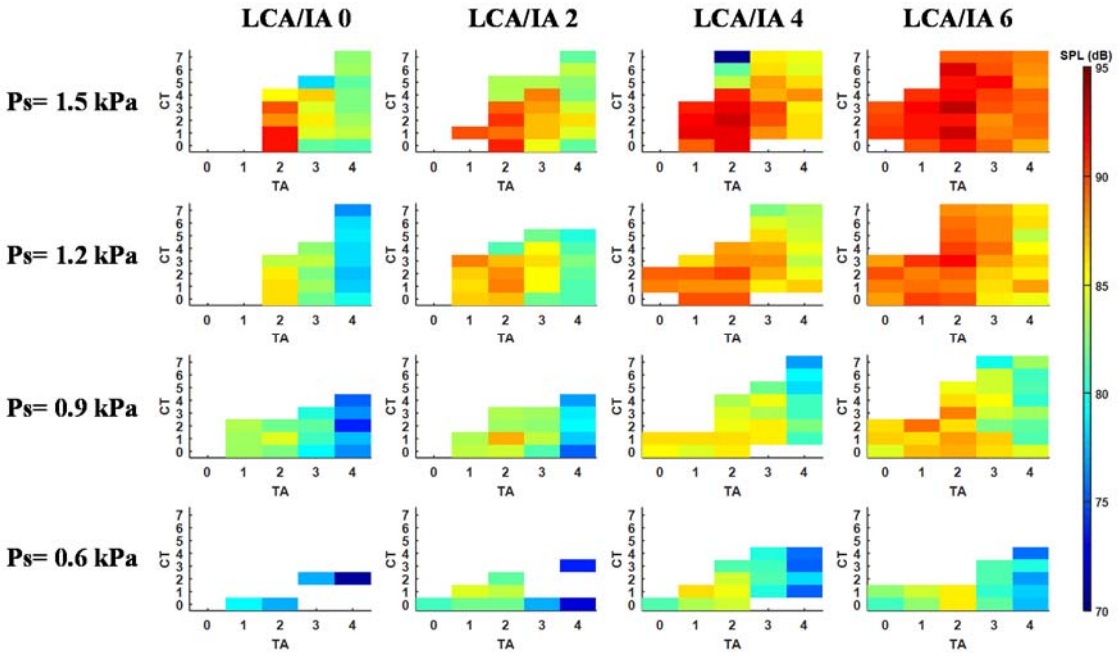

Figure 8. SPL at different stimulation conditions of the TA, CT, and LCA/IA muscles and subglottal pressures.

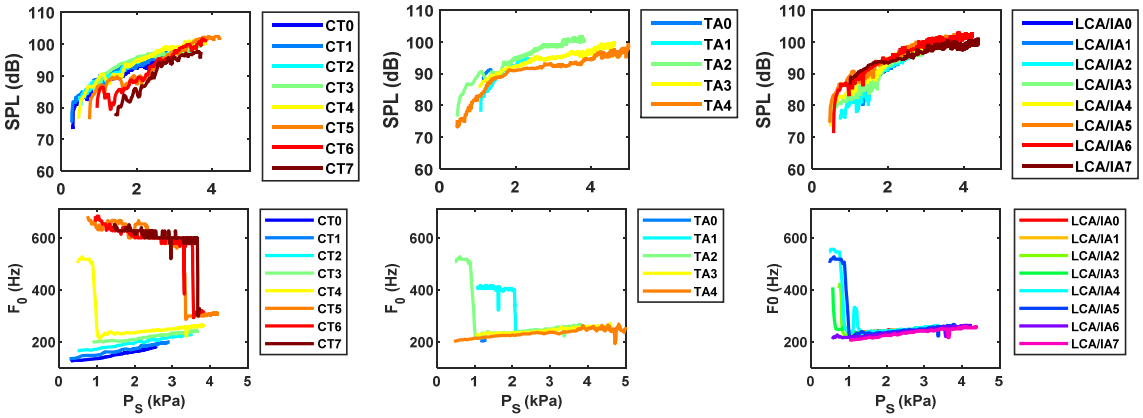

Figure 9. SPL (top) and F0 (bottom) as a function of the subglottal pressure at selected conditions of the TA, CT, and LCA/IA muscle activation.

There was noticeable interaction effect between the subglottal pressure and laryngeal muscle activation, which is more clearly illustrated in the top row of figure 9. Figure 9 shows that the effect of laryngeal muscle activation on the SPL was generally much larger at lower subglottal pressures, particularly around phonation onset, and the effect decreased with increasing subglottal pressure. For the LCA/IA muscles, the effect of its activation was reversed at high subglottal pressures, with the SPL decreasing with increasing LCA/IA activation levels. Figure 9 also shows that the maximum SPL often occurred at intermediate levels of laryngeal muscle activation. In figure 9, maximum SPL occurred at level 3 out of the 7 levels for the CT muscle, level 2 out of 4 levels for the TA muscle, and level 5 out of 7 levels for LCA/IA muscles.

Figure 10 shows the F0 at different muscle activation and subglottal pressure conditions. In general, the F0 increased with increasing CT muscle activation. The F0 also decreased with increasing TA muscle activation, particularly at high levels of TA activation. The effect of the subglottal pressure was generally small and inconsistent (figure 9). For example, the F0 can either increase or decrease with increasing subglottal pressure, depending on the specific activation levels of the CT or TA muscles (figure 9). Figures 9 and 10 also show large F0 jumps triggered by changes in either laryngeal muscle activation or the subglottal pressure, which had been reported before in Choi et al. (1993) and Chhetri et al. (2014).

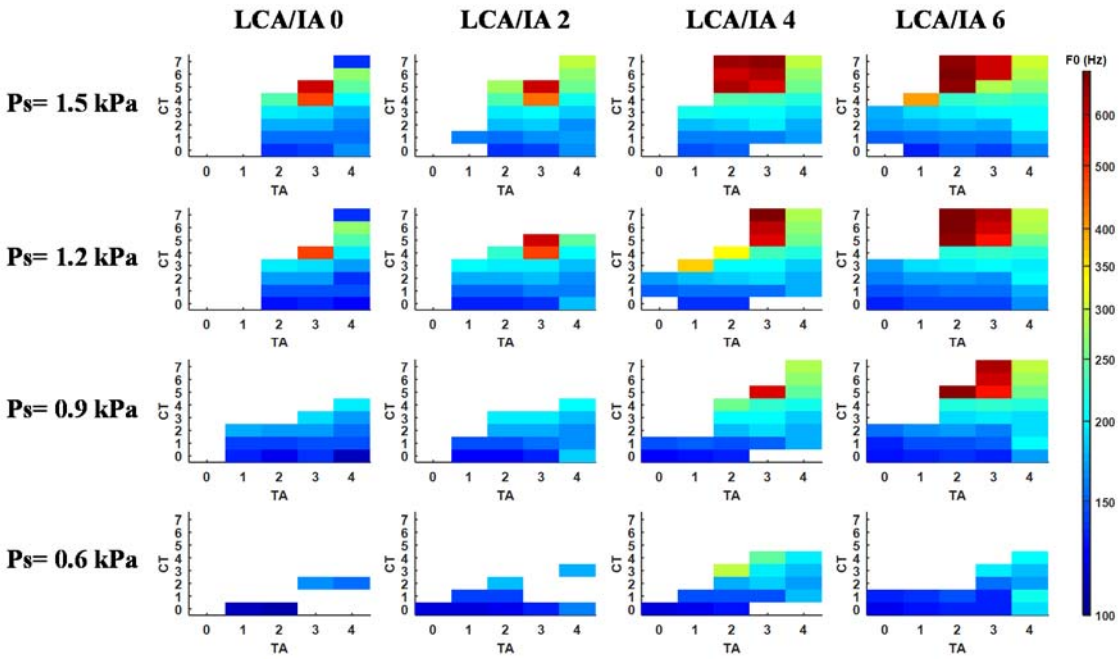

Figure 10. F0 of vocal fold vibration at different stimulation conditions of the TA, CT, and LCA/IA muscles and subglottal pressures.

4. DISCUSSION AND CONCLUSIONS

Our results showed that the TA muscle played a dominant role in regulating both the membranous glottal width and the glottal closure pattern during phonation. While arytenoid adduction is generally considered an important parameter in controlling the glottal closure pattern, our results showed that the closed quotient was primarily determined by the activity of the TA muscle. This indicates a dominant role of the TA muscles in regulating voice quality, particularly transitions between a modal voice and a pressed voice. This dominant role of the TA muscle in controlling the closed quotient was likely due to two reasons. First, similar to observations in previous studies (Choi et al., 1993; Chhetri et al., 2012), our results showed that the LCA/IA muscles alone were unable to completely close the membranous glottis, and a complete closure of the membranous glottis required the activation of the TA muscle. Second, TA muscle activation is known to increase the vertical thickness of the vocal folds, which has been shown to be the primary regulator of the closed quotient of vocal fold vibration (van den Berg, 1968; Sundberg and Hogset, 2001; Zhang, 2016).

Our results also showed that while increasing LCA/IA activation increased the output SPL, increasing activation in either the TA or CT muscle decreased the output SPL. The opposite effect of the TA and LCA/IA muscles on the SPL, despite that both reduced the glottal width, was likely due to changes in the medial surface shape caused by TA muscle activation, which increases the vertical vocal fold thickness and tends to decrease the SPL.

Our results also showed an important role of the TA muscle in regulating the mean glottal airflow, with the mean glottal flow decreasing with increasing TA activation. This flow-reducing effect of the TA muscle was particularly dominant at high subglottal pressures. The mean glottal flow can also be effectively reduced by increasing activation of the LCA/IA muscles, but the effect was much reduced at high subglottal pressures. In comparison, the effect of the CT muscle activation on the mean glottal flow was much smaller and became noticeable only at high subglottal pressures.

Previous studies have also shown that the mean glottal flow during phonation remains relatively constant with increasing vocal fold intensity (Isshiki, 1964; Hirano, 1981; Holmberg et al., 1988). In some conditions, the mean glottal flow even decreased slightly with increasing intensity. As an increase in vocal intensity is often accompanied by an increase in the subglottal pressure, laryngeal adjustments are therefore required to counterbalance the glottis-opening effect of the subglottal pressure (Isshiki, 1969; Holmberg et al., 1988; Zhang,

2011, 2015). Our results suggest that, in order to maintain the mean glottal flow, such adjustments likely include increased activation of the LCA/IA at low and moderate intensities, and increased TA activation at high vocal intensity. An increased activation of the LCA/IA is preferred because it reduces airflow but also increases output SPL. An increase in either the TA or CT muscle activation would generally decrease the output SPL, and thus neither would be a useful adjustment when attempting to increase vocal intensity. However, there are situations in which the need for airflow conservation becomes important, for example when speaking loudly, at a very low lung volume, or for a long breath group duration (Desjardins et al., 2021). In these situations, increasing TA muscle activation can significantly reduce the mean glottal flow rate, particularly at high subglottal pressures when the LCA/IA activation becomes less effective in reducing the mean glottal flow. While an increased vocal fold tension through CT activation is often considered to accompany an increase in vocal intensity, increasing CT activation decreases the output SPL and its effectiveness in conserving airflow is limited. Thus, speakers are less likely to increase CT activation when attempting to increase vocal intensity within the same register.

A second goal of this study was to provide experimental data against which computational models can be validated, either qualitatively or quantitatively. Many of the observations in this study are consistent with predictions from our computational models. For example, our previous studies (Zhang, 2015, 2021) showed that the SPL is primarily determined by the subglottal pressure, but also decreases with increasing vertical thickness or increasing vocal fold stiffness, and first increases then decreases with decreasing glottal width, which is consistent with the observation in this study. The dominant role of the TA muscles in regulating the closed quotient was consistent with the observation in Zhang (2016). The relative effectiveness of the TA, CT, and LCA/IA muscles in regulating the glottal flow observed in this study was consistent with the relative effectiveness of vertical thickness, stiffness, and initial glottal width reported in Zhang (2015) and Desjardins et al. (2021). The more dominant role of the TA muscle compared with the LCA/IA muscle in controlling the membranous glottal width was also observed in the numerical posturing model of Yin and Zhang (2014, 2016). These qualitative agreements support the validity of these models in predicting the general trends in voice production and control.

While results from the second larynx in this study were generally similar with the results from the first larynx, notable differences were observation. For example, the TA muscle was more effective in regulating vocal fold elongation in the second larynx compared to the first larynx, especially at low activation levels, indicating variability in the relative strength between the CT and TA muscles across larynges. Variability in muscular control of vocal fold posturing between larynges will be reported in future studies when more data have been collected.

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