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ORIGINAL PAPER

Modeling the influence of the extrinsic musculature on phonation

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Abstract
Neck muscles play important roles in various physiological tasks, including swallowing, head stabilization, and phonation. The mechanisms by which neck muscles influence phonation are not well understood, with conflicting reports on the change in fundamental frequency for ostensibly the same neck muscle activation scenarios. In this work, we introduce a reduced-order muscle-controlled vocal fold model, comprising both intrinsic muscle control and extrinsic muscle effects. The model predicts that when the neck muscles pull the thyroid cartilage in the superior–anterior direction (with a sufficiently large anterior component), inferior direction, or inferior–anterior direction, tension in the vocal folds increases, leading to fundamental frequency rise during sustained phonation. On the other hand, pulling in the superior direction, superior–posterior direction, or inferior–posterior direction (with a sufficiently large posterior component) tends to decrease vocal fold tension and phonation fundamental frequency. Varying the pulling force location alters the posture and phonation biomechanics, depending on the force direction. These findings suggest potential roles of particular neck muscles in modulating phonation fundamental frequency, with implications for vocal hyperfunction.

Keywords Extrinsic laryngeal muscles · Muscle tension dysphonia · Sustained phonation · Fundamental frequency

1 Introduction

In the context of phonation, there are two critical components underlying the mechanics of vocal fold (VF) vibrations, namely prephonatory posture (i.e., neutral glottal configuration), and mechanical (e.g., stress level) and geometrical (e.g., length, thickness, and width) properties of VFs, which are determined primarily via the controlled activation of the intrinsic laryngeal muscles. Several clinical and numerical studies have been conducted to uncover the function of the intrinsic laryngeal musculature during phonation (Titze and Story 2002; Chhetri et al. 2012; Movahhedi et al. 2021; Titze et al. 1989). Recent studies have further attempted to elucidate how activation of intrinsic laryngeal muscles varies between healthy speakers and those with vocal pathologies in order to gain insight into the underlying mechanisms of voice disorders (Serry et al. 2021; Alzamendi et al. 2022).

Extrinsic laryngeal muscles are also involved in the biomechanics of phonation. Kirzinger and Jürgens (1994) recorded electromyographic (EMG) activity of some extrinsic and intrinsic laryngeal muscles in squirrel monkeys during the production of calls of different voice characteristics, observing noticeable activation levels of various extrinsic laryngeal muscles that depended on the nature of the calls. Moisik and Gick (2017) developed a three-dimensional numerical posturing model of the larynx, which incorporates different intrinsic and extrinsic laryngeal muscles, with the aim of investigating laryngeal maneuvers that are likely to be associated with different glottal articulatory states. Their findings suggested that certain extrinsic muscles may play a role in some articulatory states (e.g., glottal configurations of aryepiglotto-epiglottal stops and fricatives). They did not, however, consider how these laryngeal maneuvers influence phonation.

The impact of extrinsic muscles on phonation remains poorly understood, with conflicting findings reported in the literature on the influence of specific muscles. For example,

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Atkinson (1978) reported negative correlations between the activation of the sternohyoid (SH) and sternothyroid (ST) muscles (see Fig. 3) and phonation fundamental frequency, f_o, based on a statistical analysis of voice measures and EMG activity of laryngeal muscles. In contrast, Hong et al. (1997) studied an *in vivo* canine laryngeal model and found that contraction of the SH and ST muscles corresponds to a rise in subglottal pressure, shortened cricothyroid (CT) distance, lengthened VFs, and increased f_o and vocal intensity. Moreover, they found that activation of the thyrohyoid (TH) muscle corresponds to lower subglottal pressure, wider CT distance, shortened VFs, and lower f_o and vocal intensity. Roubeau et al. (1997) reported, based on recorded EMG measurements in two singers during glissandos, that the relation between the activation of ST, SH, and TH and fundamental frequency is context-dependent. They noted that at low and high f_o values, the TH, ST, and SH muscles are active; however, the activity of these muscles becomes less prominent at moderate f_o values. For a comprehensive review of the conflicting findings in the literature regarding the roles of extrinsic muscles, see Vilkman et al. (1996).

There are several insightful investigations in the literature that shed light into the biomechanics of extrinsic laryngeal muscle activation during phonation. Honda et al. (1999) conducted magnetic resonance imaging on three male subjects and recorded the positions of the articulators and the larynx during vowel production with different f_o values. They noted that in the high f_o range, the hyoid bone, which is attached to several extrinsic laryngeal muscles, moves horizontally while the larynx height remains relatively constant. In contrast, in the low f_o range, the entire larynx moves vertically and the cricoid cartilage rotates along the cervical lordosis (the spine curvature in the neck region). In addition, they found that as f_o decreases, the hyoid bone tends to move posteriorly and then inferiorly. They noticed that the rotation of the cricoid cartilage, in the direction corresponding to increasing the CT distance, is significant in the low f_o range. Finally, they suggested two mechanisms underlying the impact of extrinsic muscles on phonation in the high and low f_o ranges: In the high range, the extrinsic muscles attached to the superior margin of the hyoid bone pull it anteriorly, leading to pulling the thyroid cartilage forward, causing increased VF tension. On the other hand, in the low f_o range, the extrinsic muscles attached to the inferior margin of the hyoid bone may be contributing to lowering the larynx, leading to increased CT distance and lowered VF tension.

In a similar study, Hong et al. (2015) conducted videofluoroscopic examinations of the movements of the hyoid bone, thyroid cartilage, and cricoid cartilage in addition to the CT distance during fundamental frequency elevation. They reported that vertical movements of the hyoid bone and the cricoid and thyroid cartilages positively correlate with fundamental frequency, whereas CT distance negatively correlates with it. Moreover, they found that the upward movement of the larynx has a more dominant effect on fundamental frequency elevation than the CT distance. They noticed that the extrinsic muscles contribute to pulling the hyoid bone and larynx upward, and that the larynx and trachea tilt forward when the fundamental frequency is higher. Moreover, the authors noticed that the hyoid bone and the thyroid cartilage move more antero-vertically than the cricoid cartilage during fundamental frequency elevation; they thus speculated that this motion may result in increased length and tension level of the VFs and, subsequently, increased f_o values.

Muscle tension dysphonia (MTD) (Morrison and Rammage 1993), or non-phonotraumatic hyperfunction (Hillman et al. 2020), is a class of voice disorders that is characterized by excessive and/or unbalanced activation of intrinsic and extrinsic laryngeal muscles (Roy 2008; Hocevar-Boltezar et al. 1998), with no observable organic changes in the vocal organs. This excessive and/or unbalanced activation is often accompanied by various symptoms, including supraglottal compression (Morrison and Rammage 1993), incomplete glottal closure (Hillman et al. 2020; Morrison and Rammage 1993), and abnormal fundamental frequency (Van Stan et al. 2021; Nguyen et al. 2009; Altman et al. 2005). Due to the relevance of neck muscles in MTD, some studies attempted to investigate the involvement of extrinsic laryngeal muscles in MTD patients. Angsuwarangsee and Morrison (2002) assessed the tension levels of extrinsic laryngeal musculature in patients with MTD using a palpation technique and tension grading system. The authors reported a strong relationship between the TH muscle tension and MTD, especially in subjects who suffer from gastroesophageal reflux. They suggested that this correlation is due to the TH muscle tensing to induce a supraglottal compression to block the stomach acid from reaching the oral cavity. Lowell et al. (2012) used a radiographic technique to measure the hyoid bone position, laryngeal position, and the hyolaryngeal space during phonation in patients with MTD in comparison with control participants. They reported that normalized vertical hyoid and laryngeal positions during phonation are significantly higher for patients with MTD than for control participants. O'Keeffe et al. (2023) utilized perilaryngeal-cranial surface EMG measurements, collected from healthy subjects and vocal hyperfunctional patients, to construct functional muscle networks (O'Keeffe et al. 2022) for each subject/patient and computed different network parameters under different vocal conditions. They reported notable differences in these parameters between healthy speakers and speakers with vocal hyperfunction, where these differences are condition-specific, making functional muscle networks a promising assessment tool. Despite the aforementioned efforts, the underlying mechanisms of MTD, and their relation with

extrinsic laryngeal muscles, are not well understood due to the wide spectrum of symptoms and the long time span over which MTD takes place. Therefore, an extensive investigation of the role of extrinsic muscles can help in elucidating some of the underlying mechanisms of MTD.

Motivated by the need for a refined understanding of the role of extrinsic laryngeal muscles in normal and pathological speech, the goal of this work is to investigate how the pulling action of neck muscles exerted on the larynx affects the mechanics of phonation. Numerical modeling is selected as the modality of investigation in order to isolate and control the pulling action of neck muscles and study its effect on the mechanics of phonation, which is difficult, if not impossible, to attain in clinical settings. Numerical phonation simulations have been shown to be effective in elucidating some of the underlying mechanisms of normal and pathological speech (Alzamendi et al. 2022; Zañartu et al. 2014; Dejonckere and Kob 2009).

Organization of the remainder of this paper is as follows: A review of the anatomy and biomechanics of the larynx and intrinsic and extrinsic laryngeal muscles is introduced in Sect. 2; model development is discussed in Sect. 3; a simplified theoretical analysis of the adapted model is introduced in Sect. 4; numerical simulation results are presented in Sect. 5; the discussion of the results and their implications are presented in Sect. 6; and the salient conclusions are presented in Sect. 7.

2 Anatomy and biomechanics of the larynx and surrounding muscles

2.1 Laryngeal cartilages and intrinsic muscles

The larynx consists of several cartilages (see Figs. 1 and 2): the cricoid cartilage, which has a ring-like shape, that sits on top of the trachea and forms the base of the larynx; the thyroid cartilage, which forms an anterior cover of the larynx, is located anterior to and slightly above the cricoid cartilage, and is attached to it at the posterior end at the cricothyroid joint; and two arytenoid cartilages resting posteriorly on the cricoid cartilage. The VFs are two paired membrane-like structures that are attached anteriorly to a common point on the thyroid cartilage and posteriorly at the vocal processes in the corresponding arytenoid cartilages.

The posture of the laryngeal cartilages, and subsequently the glottal geometry and VF tension levels, is primarily controlled by the intrinsic laryngeal muscles (see Fig. 2). The intrinsic muscles consist of the cricothyroid (CT), thyroarytenoid (TA), lateral cricoarytenoid (LCA), posterior cricoarytenoid (PCA), and interarytenoid (IA) muscles. The CT muscle is responsible for stretching the VFs, the TA, LCA, and IA muscles are responsible for adducting the VFs, with the TA muscle also shortening the VFs, and the PCA muscle is responsible for abducting the VFs (see, e.g., Alzamendi et al. (2022)).

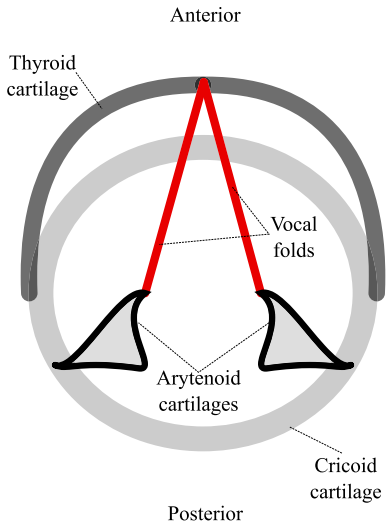

Fig. 1 (Color online) Superior schematic view of the thyroid, cricoid, and arytenoid cartilages, and the vocal folds

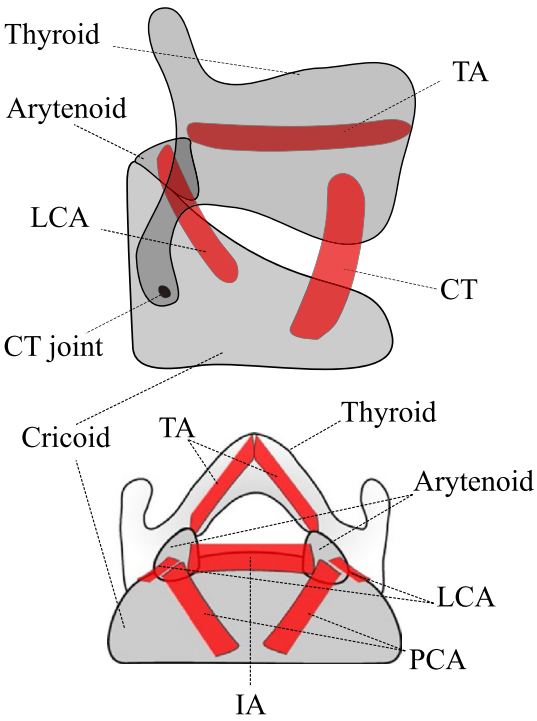

Fig. 2 (Color online) Side (top) and posterior (bottom) schematic views of the larynx with intrinsic laryngeal muscles

Intrinsic laryngeal muscles have been investigated extensively in many clinical and numerical studies in order to

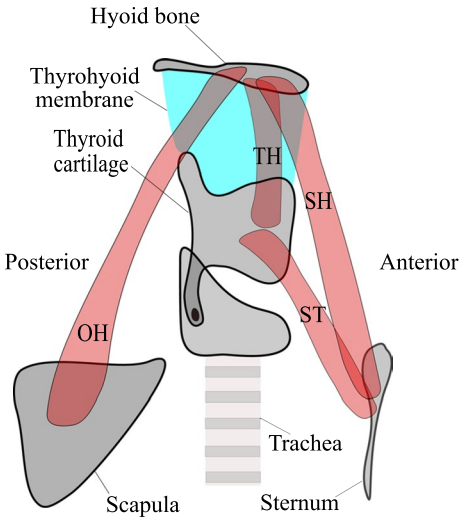

Fig. 3 (Color online) Lateral schematic view of the infrahyoid muscles: thyrohoid (TH), sternohyoid (SH), sternothyroid (ST), and omohyoid (OH) muscles: partially inspired by Fig. 5 in Hong et al. (1997)

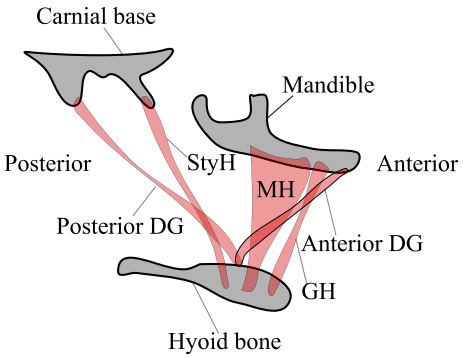

Fig. 4 (Color online) Lateral schematic view of the suprahyoid muscles: digastric (DG), geniohyoid (GH), mylohyoid (MH), and stylohyoid (StyH) muscles: inspired by Fig. 2 in Pearson et al. (2011)

elucidate their roles in phonation. In general, it has been found that the CT muscle is the primary muscle modulating phonation fundamental frequency, where increasing its activation leads to increasing fundamental frequency (Löfqvist et al. 1989; Chhetri et al. 2014). On the other hand, the role of the TA muscle in modulating fundamental frequency is more complex as it can either increase or decrease the fundamental frequency, with controversy in the literature regarding its exact role (see the discussion in Movahhedi et al. (2021)). Furthermore, it has been found that activation of the LCA and IA muscles is positively correlated with fundamental frequency (Choi et al. 1995), whereas PCA activation exhibits a negative correlation with fundamental frequency (Choi et al. 1993).

2.2 Extrinsic muscles

The extrinsic muscles, which surround the larynx, are involved in modulating the glottal posture, in addition to their roles in other physiological functions, such as head–neck movement (Mortensen et al. 2018) and swallowing (Hashimoto et al. 2020). Extrinsic muscles can be categorized into two groups, depending on their location relative to the hyoid bone (see Auvenshine and Pettit (2018) and Figs. 3 and 4), a horseshoe-shaped bone that is located superior to the larynx.

Infrahyoid muscles (see Gervasio et al. (2010)) refer to the group of neck muscles that are positioned below (inferior to) the hyoid bone (see Fig. 3). These muscles include the sternohyoid (SH), which connects the hyoid bone to the sternum, the sternothyroid (ST), which connects the thyroid cartilage to the sternum, the thyrohyoid (TH), which connects the thyroid cartilage to the hyoid bone, and the omohyoid (OH), which connects the scapula to the hyoid bone (Sukekawa and Itoh 2006). The thyrohyoid membrane, a set of connective tissues, also connects the hyoid bone to the thyroid cartilage.

Suprahyoid muscles refer to the group of muscles that are located above (superior to) the hyoid bone. This group includes the anterior and posterior digastric (DG) muscles, the geniohyoid (GH), the mylohyoid (MH), and the stylohyoid (StyH) muscles (see Fig. 4). The GH and StyH muscles originate from the mandible and cranial base, respectively, and are inserted into the hyoid bone. The MH consists of anterior and posterior portions, which connect the mandible to the hyoid bone. The anterior and posterior branches of the DG muscle arise from the mandible and cranial base, respectively, and share a common tendon with an insertion point in the hyoid bone (Pearson et al. 2011). Pearson et al. (2011) investigated the influence of suprahyoid muscles on the movement of the hyoid bone in the inferior–superior and anterior–posterior directions using a human cadaver model. They concluded that the MH, DG, StyH, and GH muscles have the likelihood to move the hyoid bone in the superior direction, with the MH muscle having the highest potential. Moreover, the MH, DG, and GH muscles are likely to move the hyoid bone in the anterior direction, with the GH muscle having the highest potential, whereas the StyH muscle has the tendency to move the hyoid bone in the posterior direction.

3 Modeling

In an effort to capture the net effect of the extrinsic muscles on VF vibration, we propose the following modeling framework: we adapt the phonation model of Alzamendi et al. (2022), which integrates the two-dimensional (2D) posturing model introduced by Titze and Hunter (2007) and the triangular body-cover model (TBCM) developed by Galindo et al. (2017). As explained in Alzamendi et al. (2022), this model includes independent activation of all five intrinsic muscles. We utilize the acoustic, aerodynamic, and TBCM modules of Alzamendi et al. (2022) and introduce modifications to the posturing module to include the thyroid cartilage pulling effect due to extrinsic muscles. The model assumes the configuration of the VFs and the position of the arytenoid cartilages to be symmetric about the mid-coronal plane, and that the activation levels of the intrinsic laryngeal muscles are identical on the right and left sides. Therefore, only one VF, in addition to associated intrinsic and extrinsic muscles, is considered in our analysis.

According to the posturing model of Titze and Hunter (2007), the VF strain, ϵ, is linearly decomposed into three components: strain due to VF adduction, ϵ_a; strain due to the rotation of the CT joint, ϵ_r; and strain due to the translation of the CT joint, ϵ_t. That is,

$$\epsilon = \epsilon_a + \epsilon_t + \epsilon_r. \tag{1}$$

The adductory strain ϵ_a is obtained from the translation and rotation of the arytenoid cartilages. We refer the readers to Titze and Alipour (2006) and Titze and Hunter (2007) for detailed discussion and governing equations for ϵ_a.

In this work, we incorporate the net pulling effects from the neck muscles on the thyroid cartilage by modifying the rotational and translational stains, ϵ_r and ϵ_t. In our derivation, and similar to Titze and Hunter (2007); Titze and Alipour (2006), the rotational and translational strains are assumed to be decoupled; that is, the CT joint undergoes only rotation (translation) when estimating rotational (translational) strain. In the subsequent discussion, we derive static equations for the rotational and translational strains, as we are concerned with sustained phonation scenarios, where the mechanical and geometrical properties of the VFs in such scenarios are assumed constant over a sufficiently long time window. We note that the static equations can be easily extended to model dynamic changes in strain by adopting the derivations in Titze and Alipour (2006, Chapter 3).

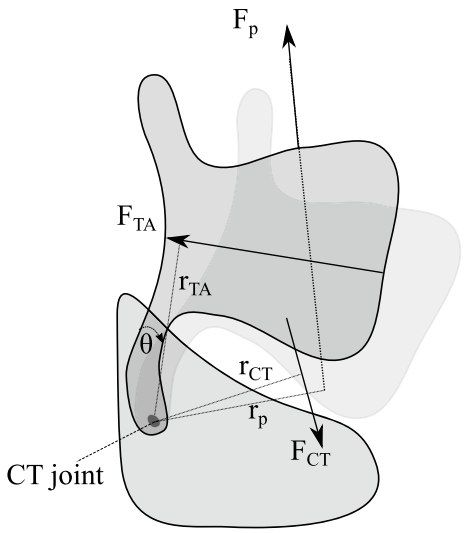

Fig. 5 Free body diagram of the rotation of the CT joint

3.1 Rotational strain

Derivation of the rotational strain follows Titze et al. (1988) and Titze and Alipour (2006). The strain ϵ_r is related to VF elongation due to CT joint rotation, ΔL_r, through the relation

$$\epsilon_r = \Delta L_r / L_0, \tag{2}$$

where L_0 denotes the resting VF length. CT joint rotation is modulated by the forces from the CT muscle, the TA muscle, and the vocal ligament, in addition to the net larynx pulling force from the extrinsic muscles (see Fig. 5), and is given by the static moment equation

$$k_r \theta = r_{\mathrm{CT}} F_{\mathrm{CT}} - r_{\mathrm{p}} F_{\mathrm{p}} - r_{\mathrm{TA}} F_{\mathrm{TA}}, \tag{3}$$

where k_r is the rotational stiffness of the CT joint, θ is the rotational displacement of the CT joint, and F_{CT} and F_{TA} are the forces from the CT muscle and the TA muscle and vocal ligament, respectively. The variable F_{p} represents the net force on the thyroid cartilage due to the extrinsic muscles. The terms r_{CT}, r_{TA}, and r_{p} denote the moment arms corresponding to the respective forces. Elongation due to rotation, ΔL_r, can then be obtained by using a small angle approximation as

$$\Delta L_r \approx r_{\mathrm{TA}} \theta. \tag{4}$$

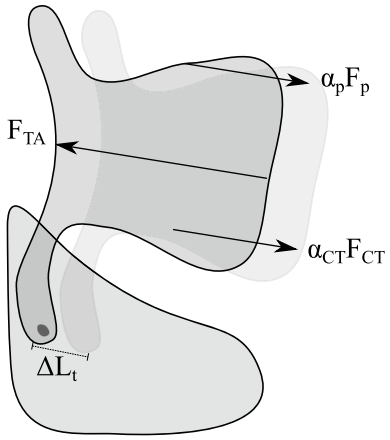

Fig. 6 Free body diagram of the translation of the CT joint

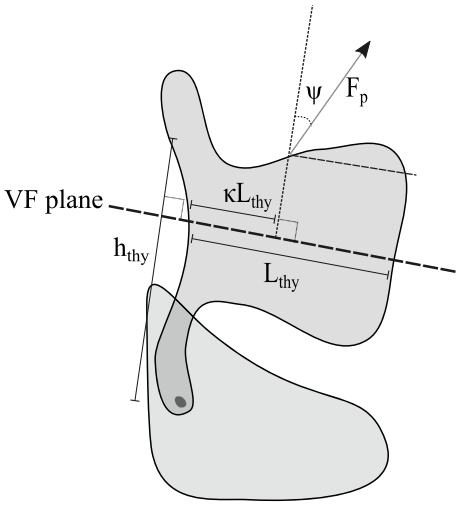

Fig. 7 Schematic plot of the net pulling force F_p, and its direction and location

3.2 Translational strain

Similar to the rotational strain derivation, strain due to translation of the CT joint, ϵ_t, is estimated by

$$\epsilon_t = \Delta L_t / L_0, \tag{5}$$

where ΔL_t is the elongation of the VF due to translation of the CT joint parallel to the VF plane[1], which is given by the static equation

$$k_t \Delta L_t = \alpha_{CT} F_{CT} - F_{TA} + \alpha_p F_p, \tag{6}$$

where k_t denotes the translational stiffness of the CT joint, and α_{CT} and α_p denote the direction cosines of the CT muscle and the resultant force due to extrinsic muscles, respectively (see Fig. 6). As in Alzamendi et al. (2022), the forces F_{TA} and F_{CT} are related to VF strain; we refer interested readers to Alzamendi et al. (2022); Titze and Alipour (2006) for detailed discussions.

3.3 Net extrinsic muscle force: magnitude, direction, and application location

In general, the force F_p and its direction cosine α_p and moment arm r_p are dependent on extrinsic muscle activation and the displacement of the thyroid cartilage with respect to the surrounding laryngeal structure. For simplicity, we set F_p, its direction ψ, defined with respect to a line perpendicular to the plane of the VFs (see Fig. 7), and its location of application κL_{thy} from the thyroid posterior margin, as prescribed parameters and investigate their effects on the mechanics of phonation. Herein, L_{thy} is the length of the lamina of the thyroid cartilage along the plane of the VFs and κ denotes the normalized location of the pulling force from the posterior margin. We assume that the direction ψ, once specified, remains unchanged during the rotation of the CT joint. Moreover, we assume that F_p is applied at the superior end of the thyroid lamina. The distance from the CT joint (which is assumed to be located approximately at the tip of the inferior cornu) to the superior end of the thyroid cartilage lamina in the direction perpendicular to the VF plane is denoted by h_{thy}. Using trigonometry, the direction cosine and moment arm of F_p are given by

$$\alpha_p = \sin(\psi), \tag{7}$$

and

$$r_p = \kappa L_{thy} \cos(\psi) - h_{thy} \sin(\psi), \tag{8}$$

respectively. Note that the direction cosine α_p is a function of the angle ψ only, whereas the moment arm r_p is a function of ψ, κ, and the lengths L_{thy} and h_{thy}.

To the best of our knowledge, there exist no direct measurements of L_{thy} and h_{thy} in the literature as dimensions of the thyroid cartilage are typically collected after removing the VFs and surrounding tissues and cartilages; however, available laryngeal measurements can be used to obtain rough estimates of these lengths. Based on measurements of male cadaver thyroid cartilages from a European population (Kovač et al. 2010), L_{thy} can be estimated as the projection of the superior width of the thyroid cartilage (denoted BE in Kovač et al. (2010)) on the medial plane, and h_{thy} can be estimated as the posterior height of the thyroid cartilage

[1] We define the VF plane to be the plane parallel to the superior surfaces of the VFs.

(denoted AD in Kovač et al. (2010)) minus the length of the superior horn (denoted AB in Kovač et al. (2010)). This yields values of $L_{\text{thy}} \approx 32\,\text{mm}$ and $h_{\text{thy}} \approx 27\,\text{mm}$ for male specimens; we adopt these values in this study. For female specimens, the values of L_{thy} and h_{thy}, based on the measurements in Kovač et al. (2010), are estimated to be approximately 22 mm for both lengths.

It has been found that the typical length of the TH muscle in a male cadaver subject is about 34 mm (Van Ee 2000), and that, in swallowing maneuvers, the hyoid bone moves in the anterior–posterior direction about 1 cm (Hashimoto et al. 2020). Considering these measurements and assuming that at rest the hyoid bone is almost perpendicular to the plane of VFs, we estimate the angle ψ to be of the order of 0.1 rad in scenarios wherein the supra- and infrahyoid muscles are jointly activated. In order to extensively investigate the influence of the thyroid cartilage pulling in the superior direction on phonation mechanics, we consider ψ values in the larger range of $[-1, 1]$ rad. On the other hand, to qualitatively assess the effect of pulling the thyroid cartilage in the inferior direction exerted by some neck muscles (e.g., ST), we additionally consider the ψ-range $[\pi - 1, \pi + 1]$ rad.

The cross-sectional area of the TH muscle in a male cadaver specimen is approximately 0.37 cm^2 (Van Ee 2000). By considering the average sarcomere length of dissected neck muscles of 2.6 μm and the method proposed in Van Ee (2000, Equations 7.2 and 7.3), the isometric maximum force in the TH muscle is estimated to be of the order of 10 N. This is of the same order of magnitude of estimates of isometric maximum forces for other neck muscles used in musculoskeletal modeling (Mortensen et al. 2018, Table 2). As we expect that neck muscles will not be activated to their maximum capacity in phonation, we consider the range [0, 1] N for the force magnitude F_{p} in this study.

As the moment arm r_{p} depends on the location of the pulling force and this location may change depending on the activation levels of the various extrinsic muscles, we consider $\kappa \in [0, 1]$. That is, the location of the resultant pulling force from the extrinsic muscles may be anywhere along the length of the thyroid cartilage.

4 Net extrinsic muscle effect: analytical insights

To gain insight into the influence of thyroid cartilage pulling on VF strain, we define

$$\begin{aligned}\tilde{\epsilon}_{\text{p}} &= \left(-\frac{r_{\text{TA}}}{k_r L_0} r_{\text{p}} + \frac{1}{k_t L_0}\alpha_{\text{p}}\right) F_{\text{p}} \\ &= \frac{1}{L_0}\left(\left[\frac{r_{\text{TA}} h_{\text{thy}}}{k_r} + \frac{1}{k_t}\right]\sin(\psi) - \frac{r_{\text{TA}}\kappa L_{\text{thy}}}{k_r}\cos(\psi)\right) F_{\text{p}}\end{aligned} \tag{9}$$

to be the strain accumulated in the VFs due to F_{p}; (in other words, $\tilde{\epsilon}_{\text{p}}$ is the contribution of F_{p} to ϵ_r plus the contribution of F_{p} to ϵ_t). Assuming that the pulling effects of the extrinsic laryngeal musculature on VF strain do not alter the applied forces from the intrinsic muscles, and that the rotational and translational stiffnesses, k_r and k_t, are displacement-independent, $\tilde{\epsilon}_{\text{p}}$ positively correlates with VF strain, ϵ, when activation of intrinsic laryngeal muscles is fixed[2]. In this section, we consider thyroid cartilage pulling in all directions (i.e., $\psi \in [-\pi/2, 3\pi/2]$ rad) while adopting the force magnitude range [0, 1] N and κ in the range [0, 1].

First, it can be deduced from Eq. (9) that, when $\cos(\psi) \geq 0$ ($\psi \in [-\pi/2, \pi/2]$ rad, i.e., upward pulling), there is a negative correlation between $\tilde{\epsilon}_{\text{p}}$ and κ. This correlation becomes positive for $\psi \in [\pi/2, 3\pi/2]$ rad (downward pulling).

Next, let us consider the range of ordered pair values $(\psi, F_{\text{p}}) \in [-\pi/2, \pi/2]\,\text{rad} \times [0, 1]\,\text{N}$, which corresponds to thyroid cartilage pulling in the superior direction. To investigate the sensitivity of $\tilde{\epsilon}$ to F_{p}, we compute the partial derivative of $\tilde{\epsilon}_{\text{p}}$ with respect to F_{p}, $\partial_{F_{\text{p}}}\tilde{\epsilon}_{\text{p}}$, which yields

$$\partial_{F_{\text{p}}}\tilde{\epsilon}_{\text{p}} = \frac{1}{L_0}\left(\left[\frac{r_{\text{TA}} h_{\text{thy}}}{k_r} + \frac{1}{k_t}\right]\sin(\psi) - \frac{r_{\text{TA}}\kappa L_{\text{thy}}}{k_r}\cos(\psi)\right). \tag{10}$$

We see that the effect of the force magnitude F_{p} is direction-dependent, that is, it has a functional dependence on ψ. We can deduce from Eq. (10) that for $\pi/2 \geq \psi \geq \psi_c$, where

$$\psi_c := \tan^{-1}\left(\frac{k_t r_{\text{TA}}\kappa L_{\text{thy}}}{k_t r_{\text{TA}} h_{\text{thy}} + k_r}\right), \tag{11}$$

changes in $\tilde{\epsilon}_{\text{p}}$ are positively correlated with the force magnitude F_{p} ($\partial_{F_{\text{p}}}\tilde{\epsilon}_{\text{p}}$ is nonnegative). On the other hand, if $-\pi/2 \leq \psi \leq \psi_c$, the correlation becomes negative ($\partial_{F_{\text{p}}}\tilde{\epsilon}_{\text{p}} \leq 0$). Note that the angle ψ_c is positively correlated with κ, implying that this critical angle increases as the pulling force location moves anteriorly.

Using clinical measurements of canine and human male cadaver models, see Table 1, and setting $\kappa = 0.5$, we estimate that

$$\frac{k_t r_{\text{TA}}\kappa L_{\text{thy}}}{k_t r_{\text{TA}} h_{\text{thy}} + k_r} \approx 0.51, \tag{12}$$

which, according to Eq. (11), yields

$$\psi_c \approx 0.47\,\text{rad}. \tag{13}$$

[2] These simplifying assumptions are not adopted in the full numerical phonation model used in the simulations presented in Sect. 5.

Table 1 Some laryngeal parameter values (rounded), based on measurements from canine and male cadaver specimens (Titze and Alipour 2006; Kovač et al. 2010)

Parameter	Value
r_{TA}	1.6 cm (Titze and Alipour 2006)
h_{thy}	27 mm (based on measurements from Kovač et al. (2010))
L_{thy}	32 mm (based on measurements from Kovač et al. (2010))
L_0	1.6 cm (Titze and Alipour 2006)
k_t	500 N/m (Titze and Alipour 2006) (zeroth order)
k_r	0.05 N.m/rad (Titze and Alipour 2006) (zeroth order)

Both the translational and rotational stiffnesses are typically given as (polynomial) functions of the CT joint translation and rotation (Titze and Alipour 2006, Equations 3.48 and 3.53). The reported stiffness values are the zeroth order terms of such polynomial functions

Considering now the sensitivity of $\tilde{\epsilon}_{\mathrm{p}}$ to ψ, the gradient $\partial_\psi \tilde{\epsilon}_{\mathrm{p}}$ is given by

$$\partial_\psi \tilde{\epsilon}_{\mathrm{p}} = \frac{1}{L_0}\left(\left[\frac{r_{\mathrm{TA}} h_{\mathrm{thy}}}{k_r} + \frac{1}{k_t}\right]\cos(\psi) + \frac{r_{\mathrm{TA}}\kappa L_{\mathrm{thy}}}{k_r}\sin(\psi)\right)F_{\mathrm{p}}. \tag{14}$$

Eq. (14) implies that for $\pi/2 \geq \psi \geq \bar{\psi}_c$, where

$$\bar{\psi}_c := -\tan^{-1}\left(\frac{k_t r_{\mathrm{TA}} h_{\mathrm{thy}} + k_r}{k_t r_{\mathrm{TA}}\kappa L_{\mathrm{thy}}}\right) = -\frac{\pi}{2} + \psi_c, \tag{15}$$

the strain $\tilde{\epsilon}_{\mathrm{p}}$ increases with ψ for fixed F_{p}; on the other hand, the strain decreases with ψ, for fixed F_p, when $-\pi/2 \leq \psi \leq \bar{\psi}_c$. Eq. (15) indicates an increase in $\bar{\psi}_c$ when κ increases and that the difference between ψ_c and $\bar{\psi}_c$ is always $\pi/2$. The estimate in Eq. (12), where κ is set to 0.5, implies that

$$\bar{\psi}_c \approx -1.1 \text{ rad}. \tag{16}$$

This indicates that for the range $\psi \in [-1, 1]$ rad, $\tilde{\epsilon}_{\mathrm{p}}$ is always positively correlated with ψ, when $\kappa = 0.5$.

Due to the structure of Eq. (9), the above findings can be easily extended to the range $(\psi, F_{\mathrm{p}}) \in [\pi/2, 3\pi/2]\,\mathrm{rad} \times [0, 1]\mathrm{N}$, which corresponds to thyroid cartilage pulling with an inferior component. For inferior pulling, $\tilde{\epsilon}_{\mathrm{p}}$ is positively correlated with F_{p} in the ψ-range $[\pi/2, \psi_c + \pi]$ rad, whereas the correlation is negative for $\psi \in [\psi_c + \pi, 3\pi/2]$ rad. The strain $\tilde{\epsilon}_{\mathrm{p}}$ is positively correlated with ψ in the range $[\pi/2, \pi + \bar{\psi}_c]$ rad and negatively correlated in the range $[\pi + \bar{\psi}_c, 3\pi/2]$ rad.

The effects of F_{p} and ψ on $\tilde{\epsilon}_{\mathrm{p}}$ in the superior and inferior pulling ranges are depicted in Fig. 8. This figure and the associated analysis show clearly that in the vicinity of the critical angles, ψ_c, $\psi_c + \pi$, $\bar{\psi}_c$, and $\bar{\psi}_c + \pi$, the influence of net neck muscle pulling can result in either an increase or decrease in VF strain. Moreover, the influence of the parameter κ on the critical angles ψ_c and $\bar{\psi}_c$ is depicted in Fig. 9, which illustrates the positive correlation between κ and the aforementioned angles. In Sect. 5, we extensively investigate through numerical simulations the influence of

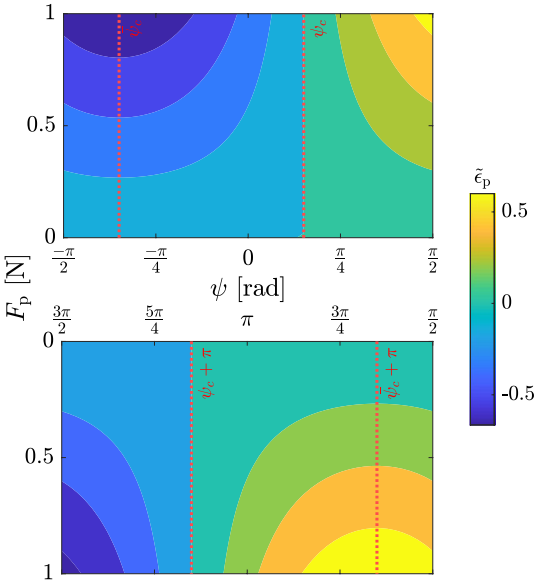

Fig. 8 (Color online) Contour plot of the strain $\tilde{\epsilon}_{\mathrm{p}}$ as a function of F_{p} and ψ from Eq. (9): parameter values of Eq. (9) are depicted in Table 1 and $\kappa = 0.5$. Scenarios for upward (superior) and downward (inferior) pulling forces are shown in the top and the bottom panels, respectively

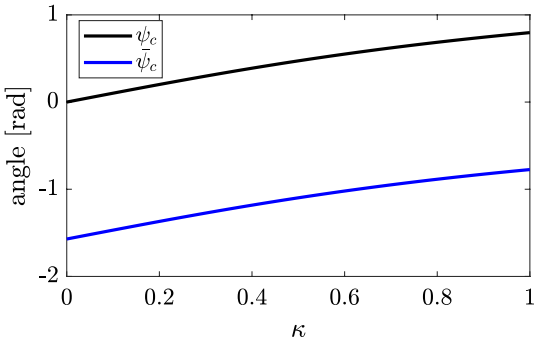

Fig. 9 (Color online) Plots of the critical angles ψ_c and $\bar{\psi}_c$ as functions of κ from Eqs. (11) and (15): adopted parameter values are listed in Table 1

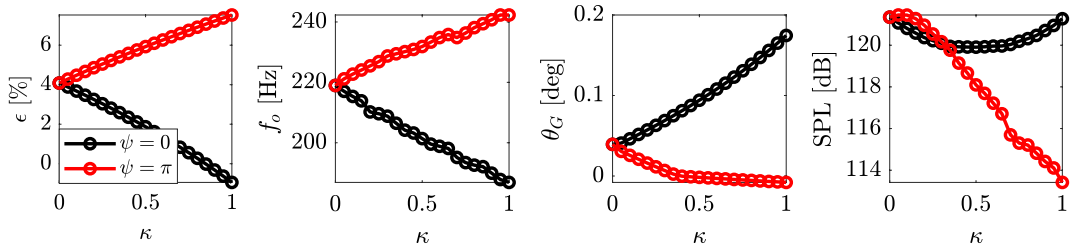

Fig. 10 (Color online) Vocal fold strain ϵ (first column), sustained phonation fundamental frequency f_o (second column), glottal angle θ_G (third column), and sound pressure level SPL (fourth column) as functions of κ, where $a_{\mathrm{CT}} = a_{\mathrm{TA}} = 0.4$ and $F_{\mathrm{p}} = 1$ N for ψ: $\psi = 0$ rad (black) and $\psi = \pi$ rad (red)

the force F_{p}, its direction ψ, and its location parameterized by κ, on the VF posture and on different voice measures.

5 Numerical simulations

In this section, we conduct numerical simulations using the modified TBCM (see Sect. 3), where the values of F_{p}, κ, and ψ are set in the ranges [0, 1] N, [0, 1], and $[-1, 1] \cup [\pi - 1, \pi + 1]$ rad, respectively. The focus herein is to investigate the influence of the pulling force parameters on the biomechanics of posturing and phonation.

The remaining parameters of the TBCM model are set as follows: we consider intrinsic muscle activation values in the ranges $a_{\mathrm{LCA}} = 0.5$, $a_{\mathrm{IA}} = 0.5$, $a_{\mathrm{PCA}} = 0$, $a_{\mathrm{CT}} \in [0.2, 0.6]$, and $a_{\mathrm{TA}} \in [0.2, 0.6]$, where a_{LCA}, a_{IA}, a_{PCA}, a_{CT}, and a_{TA} denote the non-dimensional normalized muscle activations for the LCA, IA, PCA, CT, and TA muscles, respectively. The above ranges correspond to phonation scenarios with low/normal activation levels of the CT and TA muscles, and fully (or near fully) adducted VFs (modal phonation). Moreover, we set the subglottal pressure $P_s = 800$ Pa, which is within the typical range during speech (Zhang 2016; Espinoza et al. 2017). The passive model parameters correspond to a male subject, and acoustics are modeled using wave reflection analog (Kelly and Lochbaum 1962; Story 2005), where subglottal and supraglottal acoustic tracts are coupled with the TBCM. Similar to Galindo et al. (2014); Zañartu et al. (2014), the subglottal tract area function is adapted from respiratory system measurements of human cadavers (Weibel et al. 1963), covering only the trachea and bronchi. Moreover, a supraglottal area function corresponding to a male tract configured to produce the American English open back unrounded vowel /*a*/ is adopted (Story 2008).

Each phonation simulation is run for 200 ms, with a sampling frequency of 44100 Hz. The first 100 ms of each simulation is discarded to remove transient effects. From the sustained oscillations portions, we compute the fundamental frequency, f_o, and the sound pressure level, SPL. Additionally, from the posturing computations, we estimate the VF strain, ϵ, and the glottal angle, θ_G, which is defined as the total included angle between the two VFs at the anterior attachment point.

Figure 10 depicts plots of vocal fold strain ϵ, sustained phonation fundamental frequency f_o, glottal angle θ_G, and sound pressure level SPL, as functions of the parameter κ, which specifies the location of the pulling force. The figure shows that, for the case of upward pulling ($\psi = 0$), increasing κ, which corresponds to moving the pulling force location anteriorly along the thyroid cartilage length, leads to decreasing VF tension and fundamental frequency, in agreement with the analysis in Sect. 4, and slight variations in the glottal angle and SPL. Moreover, the figure indicates that, for the case of downward pulling ($\psi = \pi$ rad), increasing κ leads to a general increase in VF tension and fundamental frequency, also in alignment with the analysis in Sect. 4, and insignificant variation in the glottal angle. However, for the case of downward pulling, a notable decrease in SPL is observed. We speculate that this decrease may be due to the fact that increased VF tension leads to VF oscillations of small amplitude, which may induce low-amplitude acoustic pressure.

Figure 11 displays contour plots of the VF strain, glottal angle, phonation fundamental frequency, and SPL as functions of F_{p} and ψ for various intrinsic muscle activation values and $\kappa = 0.5$. The first column shows that for all simulations when the angle ψ is in the approximate range [-1,0.5] rad the strain is negatively correlated with the force magnitude F_{p}, with the correlation becoming positive for the approximate range [0.5, 1] rad. This agrees reasonably with predictions from the theoretical analysis in Sect. 4. For the inferior thyroid cartilage pulling scenarios ($\psi \in [\pi - 1, \pi + 1]$ rad), Fig. 11 shows that the VF strain increases with F_{p} in the approximate range $[\pi - 1, \pi + 0.4]$ rad. When the inferior pulling force has a sufficiently large posterior component (ψ is in the approximate range $[\pi + 0.4, \pi + 1]$ rad), VF strain decreases as F_{p} increases. In addition, the first column of Fig. 11 shows that when ψ is in the range $[-1, 1]$ rad, VF strain is positively correlated with the angle ψ (for fixed F_{p}), which agrees with our theoretical

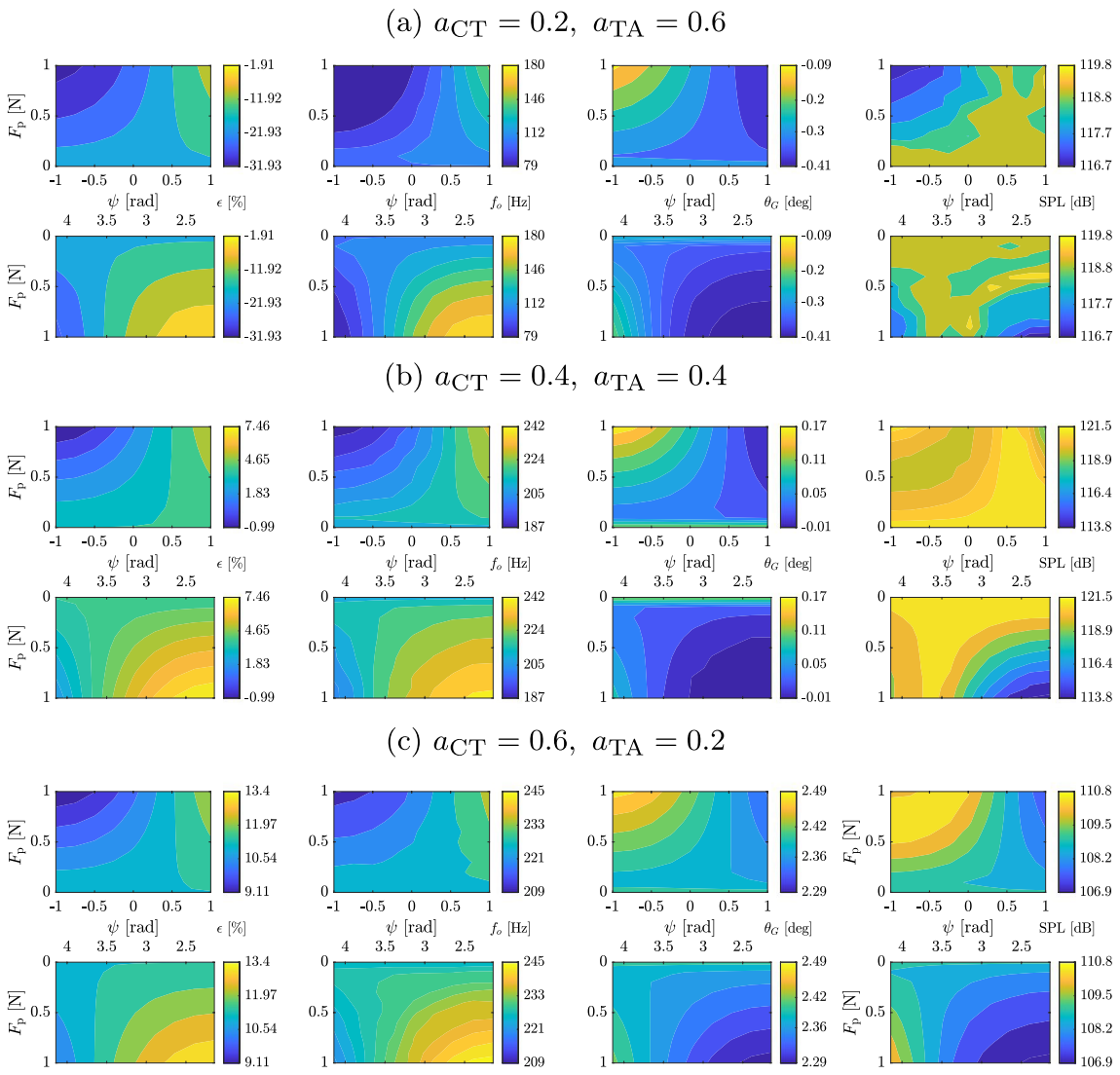

Fig. 11 (Color online) Contour plots of vocal fold strain ϵ (first column), sustained phonation fundamental frequency f_o (second column), glottal angle θ_G (third column), and sound pressure level SPL (fourth column), as functions of the force F_{p} and the angle ψ for different combinations of CT and TA activation levels with $\kappa = 0.5$: (a) $a_{\mathrm{CT}} = 0.2$, $a_{\mathrm{TA}} = 0.6$, (b) $a_{\mathrm{CT}} = 0.4$, $a_{\mathrm{TA}} = 0.4$, and (c) $a_{\mathrm{CT}} = 0.6$, $a_{\mathrm{TA}} = 0.2$

predictions in Sect. 4. For the case of inferior pulling, ψ negatively correlates with the VF strain. These results indicate that pulling the thyroid cartilage superiorly, or superiorly and posteriorly, leads to VF shortening. When superior pulling incorporates an anterior component with a sufficiently large tilting angle, the VFs are elongated. In addition, the results indicate that inferior pulling of the thyroid cartilage typically lengthens the VFs, except in situations when the pulling has a sufficiently large posterior component, which causes the VFs to shorten and the strain to decrease.

Unsurprisingly, the second column of Fig. 11 shows that fundamental frequency exhibits similar trends to VF strain, as fundamental frequency typically correlates with VF elongation (see, e.g., Zhang et al. (2006)). This implies that thyroid cartilage pulling in the superior or the superior–posterior directions causes a drop in fundamental frequency, which agrees with the analysis in Hong et al. (1997). Thyroid cartilage pulling in the superior–anterior (with sufficiently large anterior component), inferior, or inferior–anterior directions causes the fundamental frequency to rise, which agrees qualitatively with the findings in Hong et al. (2015); Hong et al. (1997). The third column of Fig. 11 shows the influence of the pulling force F_{p} and its direction ψ on glottal angle. It can be seen that for the approximate ranges for ψ, $[-1, 0]$ rad and $[\pi + 0.5, \pi + 1]$ rad, increasing the force F_{p} leads to increasing the glottal angle; that is, the net extrinsic muscle action acts to abduct the VFs. In contrast, for the approximate ranges of ψ, $[0.5, 1]$ rad and $[\pi - 1, \pi]$ rad,

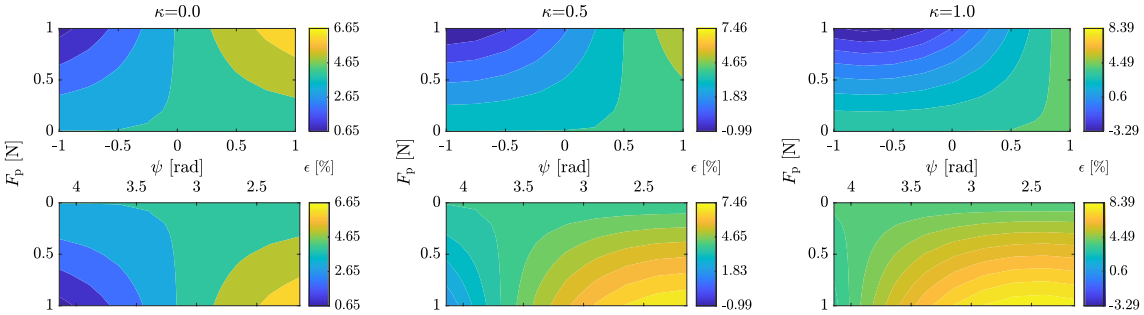

Fig. 12 (Color online) Contour plots of vocal fold strain ϵ as a function of the force F_p and the angle ψ for $a_{CT} = 0.4$, $a_{TA} = 0.4$ and different values of κ: $\kappa = 0$ (first column), $\kappa = 0.5$ (second column), and $\kappa = 1$ (third column)

the glottal angle decreases with the force F_p, indicating an adducting action. We note, however, that for the considered ranges of F_p and ψ, changes in glottal angle are quite modest. The fourth column of Fig. 11, which presents SPL, exhibits complex trends. However, in most of the considered muscle activation scenarios, the changes in SPL are within 4 dB.[3] Care should be taken when investigating the independence/dependence between intrinsic and extrinsic muscles. Despite the qualitative similarities of contour plots across different intrinsic muscle activation combinations in Fig. 11, the significance of extrinsic musculature effects on different laryngeal and phonation variables depends upon the activation levels of the intrinsic muscles.

Figure 12 displays contour plots of VF strain as a function of F_p and ψ for various values of κ. These contour plots are similar in trends to those presented in Fig. 11, where the aforementioned variable changes behavior in the vicinity of critical angles (recall the definitions of ψ_c and $\tilde{\psi}_c$ in Sect. 4). Such similarities have also been observed for the variables f_o, θ_G, and SPL (not shown). The figure illustrates how the critical angles corresponding to ψ_c and $\bar{\psi}_c$ increase as κ increases, which is in agreement with the theoretical analysis in Sect. 4.

6 Discussion

The results presented in Sect. 5 highlight how the magnitude, direction, and location of the net force on the thyroid cartilage due to the extrinsic laryngeal musculature influence glottal configuration and phonation characteristics, including fundamental frequency and SPL. Our analysis suggests that the suprahyoid muscles, which tend to pull the hyoid bone anteriorly (and thus pull the thyroid cartilage in the same direction), have the potential of increasing phonation fundamental frequency. Our analysis, in coordination with Fig. 4, indicates that the GH muscle, which tends to move the hyoid bone anteriorly and superiorly (Pearson et al. 2011), may act to raise fundamental frequency during phonation, agreeing with the analysis of Honda et al. (1999). Moreover, our analysis suggests that suprahyoid muscles that exert superior or superior–posterior pulling on the thyroid cartilage (e.g., StyH (Pearson et al. 2011)) may lower fundamental frequency, which agrees with the observations of Honda et al. (1999) regarding the posterior motion of the hyoid bone and lowering f_o. Furthermore, our analysis indicates that neck muscles responsible for pulling the thyroid cartilage inferiorly (e.g., ST) may induce higher VF tension and consequently higher f_o, which agrees with the analysis of Hong et al. (1997). Finally, and most importantly, our analysis indicates that the effect of co-activated extrinsic laryngeal muscles depends upon the magnitude, direction, and location of the net force and subsequent net VF elongation, which all vary with different muscle co-activations, in alignment with the analysis of Vilkman et al. (1996). This may explain, in part, inconsistencies appearing in the literature regarding the roles of extrinsic muscles in phonation. Given the experimental difficulties in assessing levels of muscle activity of human subjects, and in light of our findings on the dependence of VF strain on net force direction and application location, we postulate that inconclusive and contradictory findings from empirical studies may be associated with uncontrolled activation of additional neck muscles beyond those being studied in a given experimental campaign (see also the discussion in Vilkman et al. (1996)). That is, in light of existing literature, our work suggests that to have a robust analysis and refined insight into the influence of neck muscles, the net effect of laryngeal muscles needs to be considered by investigating the larynx and hyoid bone movements, as in Hong et al. (2015), and measuring the EMG activity of all extrinsic laryngeal muscles.

With regard to non-phonotraumatic hyperfunctional phonation, findings in the literature indicate excessive activation of neck muscles is common in patients with MTD (Roy 2008).

[3] The case of inferior thyroid cartilage pulling with $a_{CT} = a_{TA} = 0.4$ exhibits changes in SPL within 8 dB.

These patients also typically suffer from abnormal phonation fundamental frequency, where some studies indicate that abnormality in f_o leans toward lower fundamental frequency (Altman et al. 2005), whereas recent studies indicate that MTD patients, especially female patients, exhibit higher f_o in comparison with control subjects (Van Stan et al. 2021). In addition, clinical data reporting higher hyoid bone position in MTD subjects (Lowell et al. 2012) imply relatively higher activation of suprahyoid muscles. These observations, in addition to the results obtained herein, suggest that for individuals with MTD with abnormally low f_o, the net pulling effect exerted on the thyroid cartilage by the suprahyoid muscles could potentially be in the superior or superior–posterior directions. This indicates that suprahyoid muscles contributing to the pulling of the hyoid cartilage in these directions (e.g., StyH (Pearson et al. 2011)) may exhibit higher activity in such patients. Besides the suprahyoid muscles, other extrinsic muscles that contribute to superior pulling of the thyroid cartilage, such as TH, may exhibit higher activity in MTD subjects, contributing to the clinically observed lower f_o. This partially agrees with the assessment of Angsuwarangsee and Morrison (2002), who reported higher activation of the TH muscle in some MTD patients. In the case of MTD patients who exhibit abnormally high f_o (Van Stan et al. 2021), we hypothesize, based on the analysis in the current study, that suprahyoid muscles that are associated with superior–anterior pulling of the hyoid bone (e.g., the GH muscle (Pearson et al. 2011)) may exhibit high activity in such patients. Finally, in the case of MTD subjects who suffer from tense neck muscles without abnormality in fundamental frequency, we speculate that an array of neck muscles are co-activated, such that their opposing effects neutralize each other (the net force is near zero), resulting in no significant changes in VF elongation.

There are a number of limitations in the present analysis that warrant consideration. The posturing model in this work considers only the net pulling effect of extrinsic muscles without incorporating the mechanics of individual muscles and connective tissue that will influence hyoid bone movement. Furthermore, our model parameters are based on crude estimates of the pulling force direction and magnitude due to the paucity of relevant clinical measurements. Moreover, our modeling framework carries over the limitations of the phonation model of Alzamendi et al. (2022), wherein for example, the direction cosines associated with intrinsic laryngeal muscles are assumed to be displacement-independent. Despite these limitations, the model is capable of capturing qualitatively some effects of extrinsic laryngeal muscles during phonation, in alignment with clinical observation. Still, the conjectures from the analyses above necessitate extensive clinical investigation into the involvement of neck muscles during phonation in vocally healthy subjects and patients with MTD.

7 Conclusion

In this work, we adapted a previously introduced muscle-controlled phonation model to incorporate the effects of extrinsic muscles on the mechanical and geometrical properties of the VFs, and subsequently applied the proposed model to elucidate the effects of larynx pulling on the mechanics of phonation. Theoretical analysis and simulations using the adapted model indicate that if the thyroid cartilage is pulled in the superior, superior–posterior, or inferior–posterior directions, VF tension tends to reduce and, subsequently, phonation fundamental frequency decreases. On the contrary, superior pulling with a sufficiently large anterior component, inferior pulling, and inferior–anterior pulling tend to raise the VF tension leading to a fundamental frequency rise. The analysis also indicated a dependence of the VF tension level and fundamental frequency on the location of the net force application, where in the case of superior pulling, moving the force location anteriorly leads to a drop in tension and fundamental frequency, whereas a rise was found when pulling in the inferior direction. A comparison of the numerical results in this paper with reported clinical findings suggested potential involvements of particular neck muscles during phonation and the potential relevance of some neck muscles in patients with MTD.

In future work, we aim to investigate the influence of individual neck muscles by adopting a more refined posturing model that incorporates the hyoid bone and supra- and infrahyoid muscles, with the specified origin and insertion points, where models used in investigations of neck stabilization, swallowing, and phonatory postures (e.g., Hashimoto et al. 2020; Mortensen et al. 2018; Moisik and Gick 2017) may be adapted in the refined analysis. In addition, we aim to adapt our modeling framework to investigate the influence of tracheal pulling on the mechanics of phonation, which was highlighted, e.g., in Vilkman et al. (1996).

Author Contributions M.A.S. and S.D.P. conceived of the study. M.A.S. and G.A. developed the model. M.A.S. generated preliminary results and figures. All the authors analyzed and interpreted the results. M.A.S. wrote the first draft of the article. All the authors reviewed and approved the manuscript.

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Availability of data and materials Not applicable.

Declarations

Ethical Approval Not applicable.

Conflict of interest M.Z has a financial interest in Lanek SPA, a company focused on developing and commercializing biomedical devices and technologies. His interests were reviewed and are managed by the Universidad Técnica Federico Santa María in accordance with its conflict-of-interest policies.

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