Simulated effects of cricothyroid and thyroarytenoid muscle activation on adult-male vocal fold vibration

Soren Y. Lowell, and Brad H. Story

Citation: The Journal of the Acoustical Society of America **120**, 386 (2006); doi: 10.1121/1.2204442
View online: https://doi.org/10.1121/1.2204442
View Table of Contents: https://asa.scitation.org/toc/jas/120/1
Published by the Acoustical Society of America

Simulated effects of cricothyroid and thyroarytenoid muscle activation on adult-male vocal fold vibration

Soren Y. Lowell[a] and Brad H. Story
Department of Speech, Language, and Hearing Sciences, University of Arizona, Tucson, Arizona, 85721-210071

(Received 18 January 2005; revised 3 April 2006; accepted 20 April 2006)

Adjustments to cricothyroid and thyroarytenoid muscle activation are critical to the control of fundamental frequency and aerodynamic aspects of vocal fold vibration in humans. The aerodynamic and physical effects of these muscles are not well understood and are difficult to study *in vivo*. Knowledge of the contributions of these two muscles is essential to understanding both normal and disordered voice physiology. In this study, a three-mass model for voice simulation in adult males was used to produce systematic changes to cricothyroid and thyroarytenoid muscle activation levels. Predicted effects on fundamental frequency, aerodynamic quantities, and physical quantities of vocal fold vibration were assessed. Certain combinations of these muscle activations resulted in aerodynamic and physical characteristics of vibration that might increase the mechanical stress placed on the vocal fold tissue.
[DOI: 10.1121/1.2204442]

PACS number(s): 43.70.Bk, 43.70.Aj, 43.70.Gr [AL] Pages: 386–397

I. INTRODUCTION

Intelligible and efficient production of speech and song relies, in part, on the ability to precisely control the fundamental frequency (F_0) of the voice. Average F_0 and changes in F_0 during conversational speech signal critical linguistic information such as questions versus statements (Thorsen, 1980) and syllable and word emphasis, as well as personal information such as personality traits of the speaker (Brown *et al.*, 1974), sex (Bachorowski and Owren, 1999; Gelfer and Schofield, 2000), and age (Jacques and Rastatter, 1990). The primary control of F_0 is thought to be based on the neuromuscular activation of two sets of intrinsic laryngeal muscles: the cricothyroid (CT) and the thyroarytenoid (TA). According to the cover-body theory of vocal fold vibration (Hirano, 1974), physical quantities of the vocal folds such as length, mass, and stiffness are adjusted by the complex activations of these two muscles. Adjustment of these muscles can therefore have a substantial effect on the aerodynamics and mechanics of vocal fold vibration. The relationships between several intrinsic laryngeal muscles and acoustic characteristics of voice (Gay *et al.*, 1972; Kempster *et al.*, 1988; Tanaka and Tanabe, 1986) as well as vocal fold movement (Nasri *et al.*, 1994; Poletto *et al.*, 2004) have been studied in humans and in canine models. Less is known about the effects of intrinsic laryngeal muscle activation on aerodynamic and mechanical properties of vocal fold vibration. This knowledge is critical for understanding normal vocal fold physiology and pathophysiology in voice disorders. Vocal fold vibration during speech production often involves co-contraction of the CT and TA muscles (Poletto *et al.*, 2004; Shipp and McGlone, 1971; Titze *et al.*, 1989). Thus, determining the aerodynamic and mechanical effects of a range of co-activation combinations for CT and TA is important to study and has not been addressed in research to date. This study investigated the simulated effects of increased cricothyroid and thyroarytenoid muscle activation on acoustic, aerodynamic, and mechanical characteristics of adult-male vocal fold vibration.

Through their ability to lengthen or shorten the vocal folds, the CT and TA muscles provide the greatest mechanical advantage in altering vocal fold stiffness and thereby achieving changes in F_0. Whereas the apparent importance of other intrinsic laryngeal muscles such as the lateral cricoarytenoid (LCA) and posterior cricoarytenoid (PCA) in F_0 control has varied between studies (Atkinson, 1978; Gay *et al.*, 1972), the CT and TA muscles consistently emerge as the primary contributors to F_0 change (Gay *et al.*, 1972; Hirano *et al.*, 1970; Shipp and McGlone, 1971). Gay *et al.* (1972) used intramuscular electromyography (EMG) to measure laryngeal muscle activity relative to F_0 change and found that increases in F_0 were correlated with predominant, progressive increases in CT and TA activity. Other investigators systematically varied CT and TA activity to observe the resulting effect on F_0. Van den Berg and Tan (1959) conducted studies on excised human larynges, in which they artificially induced tension to mimic the action of the CT, TA, and LCA, and found that F_0 increased as a result of these manipulations. Direct electrical stimulation of the CT and TA muscles has been shown to increase F_0 (Kempster *et al.*, 1988), thus demonstrating the causal contribution of CT and TA activity to control of F_0.

Simultaneous contraction of the CT and TA muscles during human phonation offers a great deal of flexibility in producing changes in fundamental frequency. Male subjects show both intra- and intersubject variability in the levels of CT and TA activation used to produce a given F_0 (Titze *et al.*, 1989). Differing levels of TA and CT activation will

[a]Current affiliation: The National Institutes of Health, National Institute of Neurological Disorders and Stroke, Laryngeal and Speech Section, Bethesda, MD 20892.

influence aerodynamic and physical characteristics of the vocal folds during vibration. Based on Hirano's (1974) cover-body theory, Titze *et al.* (1989) proposed that TA activation can increase the stiffness of the vocal fold body while simultaneously decreasing the stiffness of the cover. Depending on the degree to which the body participates in vocal fold vibration, TA activation could raise or lower F_0. Increased F_0 would be expected when the body participates significantly in vibration, such as in the low-frequency range or for loud phonation. At the mid-frequency range, TA activation could result in either increased or decreased F_0. At high fundamental frequencies such as in falsetto, and during soft phonation when the cover contributes more to vibration than the body, TA activation would be expected to lower F_0 because it creates a slack cover (Titze, 2000). Changes to the stiffness of the body and cover not only affect F_0, but can also affect dimensions such as amplitude of vibration, degree of excursion, and vertical phase difference between the body and cover during vocal fold vibration, as well as aerodynamic characteristics.

Support for the complex effects of TA activation on F_0 is based on investigations of muscle activation levels of the CT and TA during F_0 change. Hirano and colleagues have demonstrated frequency-dependent, differential activation levels of the CT and TA with F_0 changes (Hirano, 1988; Hirano *et al.*, 1969, 1970). Titze *et al.* (1989) used electromyography (EMG) to study CT and TA muscle activations associated with volitional F_0 change. Results showed that speakers generally raised F_0 by increasing both CT and TA activation levels. However, for a given speaker, several combinations of CT and TA activation could yield the same F_0. In the second portion of the study, Titze *et al.* (1989) used the electrodes for direct stimulation of the TA muscle. Electrical stimulation in one subject resulted in consistent increases in F_0 for low to medium F_0 ranges. In the medium F_0 range, TA stimulation resulted in both increased and decreased F_0. Decreasing F_0 changes were produced with TA stimulation in the falsetto range and for soft phonation. EMG stimulation in the other three subjects, however, yielded less clear results that may have been affected by electrode placement difficulty or measurement error (Titze *et al.*, 1989). Overall, these findings highlight the complexity of the TA muscular role in F_0 control and support the theoretical notion that TA activation can result in both increased and decreased F_0.

Beyond the control of F_0, less is known about the mechanical and aerodynamic consequences of intrinsic laryngeal muscle activation. Physical movement of the vocal folds has been correlated to the activity of several intrinsic laryngeal muscles. Poletto *et al.* (2004) found posterior cricoarytenoid EMG activity to correlate consistently with vocal fold opening, whereas CT and TA activity were correlated with both opening and closing of the vocal folds during speech production. An *in vivo* canine model (Nasri *et al.*, 1994) supported the adductory roles of the TA, lateral cricoarytenoid (LCA), and interarytenoid (IA) muscles.

Few studies have quantified aerodynamic changes associated with intrinsic laryngeal muscle activation changes. Investigators who have included estimations or measures of tracheal pressure or airflow and intrinsic laryngeal muscle activity (Atkinson, 1978; Baer, 1979; Baker *et al.*, 2001; Shipp and McGlone, 1971) have generally focused on the relationship between these aerodynamic variables and the acoustic variables, versus laryngeal muscle activation. In the few studies that have correlated intrinsic laryngeal muscle activity to aerodynamic parameters, conflicting results regarding the correlation of TA activity and laryngeal resistance have been reported (Finnegan *et al.*, 2000; Tanaka and Tanabe, 1986). CT and TA activity were positively correlated to subglottal pressure (Baer, 1979; Shipp and McGlone, 1971), and CT activity was not significantly correlated to airflow (Faaborg-Andersen *et al.*, 1967). In all of these studies, subjects were asked to produce speech utterances at specific frequencies or intensities, while EMG activity and aerodynamic parameters were measured. However, data are lacking on the aerodynamic and mechanical effects that result from changes to intrinsic laryngeal muscle activation levels.

Measuring the isolated effects of intrinsic laryngeal muscle activation is difficult *in vivo*. Although speakers can reliably produce speech at a specified target frequency or intensity, especially if given a means of external monitoring and correction, they typically cannot volitionally control the level of TA or CT activation as targets. Changes to multiple extralaryngeal variables often occur simultaneously with intricate, synergistic activation of intrinsic laryngeal muscles in the control of F_0. These co-occurring changes complicate the interpretation of individual, intrinsic laryngeal muscle effects. One way in which the isolated effects of these muscle activations have been studied is by electrically stimulating the muscle and then observing the response to the stimulation. As summarized above, electrical stimulation of the TA muscle and resulting effects on F_0 were assessed by Titze *et al.* (1989) in four males. Stimulation either increased or decreased F_0, depending on the F_0 at which the subject was vocalizing. Kempster *et al.* (1988) found that electrical stimulation of the TA and CT increased F_0 in the four human subjects that they studied. Intrinsic laryngeal muscle activation was simulated in anesthetized canines using mechanical retraction of cartilages and by electrically stimulating the thyroarytenoid muscle (Tanaka and Tanabe, 1986). The effects of intrinsic laryngeal muscles on subglottal pressure, flow, and voice intensity were then observed. TA muscle contraction resulted in increased subglottal pressure and decreased airflow, with no substantial change to voice intensity. Thus, preliminary work in humans and canines has suggested some causal effects of intrinsic laryngeal muscle activation on acoustic and aerodynamic parameters. However, investigation in humans is hampered by the difficult and invasive techniques of intramuscular stimulation, and the need to minimize the number of stimulations given to any one subject.

The effects of intrinsic laryngeal muscle activity on F_0 can also be studied with computational models that are based on approximations of the physical properties of the vocal folds (Alipour-Haghighi and Titze, 1983, 1991; Ishizaka and Flanagan, 1972; Story and Titze, 1995). Such models cannot necessarily explain how any particular speaker controls their voice, but rather allow for predictive simulations of possible

voice productions. Titze *et al.* (1989) and Titze (1991) developed a model of male F_0 control based on a physiologically motivated and empirically determined relation between vocal fold strain (i.e., length change) and normalized activation levels of the CT and TA muscles. This relation resulted from experiments with excised larynges, *in vivo* animal preparations, and EMG recordings of human speakers, and was subsequently used in the model to specify the passive stress developed in the various tissue layers within the vocal folds. These stresses, along with the active stress contributed by the TA activity, were then combined to predict the vibrational frequency of the vocal folds. This particular model did not, however, include an actual simulation of the self-sustained oscillation of the vocal folds.

More recently, Titze and Story (2002) have incorporated many aspects of the strain-based F_0 control model into a system in which the activations of the CT and TA muscles, specified as input parameters, are transformed into the mechanical parameters (i.e., stiffness, mass, damping) of a low-dimensional self-oscillating vocal fold model. This provides a means by which the vibration of the vocal folds and resulting output quantities such as pressure and airflow can be simulated relative to intrinsic laryngeal muscle activation. Simulations showed that a continuum of muscle activation levels for TA and CT could theoretically produce a constant F_0, with isofrequency contour lines generated to depict these muscle activation combinations (Story and Titze, 1995; Titze, 2000; Titze *et al.*, 1989; Titze and Story, 2002). These simulations suggested that, at certain ranges of CT and TA activation levels, a male speaker would have various options for increasing or decreasing F_0. Thus, computational modeling provides a means to explore the predicted, isolated contributions of intrinsic laryngeal muscles to vocal biomechanics. Follow-up studies in humans are then needed to validate the simulated effects of these muscle activations in humans.

Determining how simulated changes to CT and TA activation may alter parameters such as vocal fold configuration, airflow, and F_0 during phonation is a critical step toward understanding normal and disordered voice physiology. Changes in certain aerodynamic characteristics have been associated with voice disorders. Maximum flow declination rate (MFDR) is indicative of the velocity of vocal fold closure, and an increase in MFDR may result in a greater degree of vocal fold collision forces (Hillman *et al.*, 1989). Sound pressure level (SPL), tracheal pressure, laryngeal resistance, and MFDR are positively correlated (Holmberg *et al.*, 1988, 1989, 1994). Increased MFDR values have been reported in subjects with vocal nodules and vocal polyps (Hillman *et al.*, 1989, 1990)—vocal pathology that is thought to be related to vocal hyperfunction and increased vocal fold impact stress (Boone and McFarlane, 2000; Case, 2002; Gray and Titze, 1988). Higher levels of impact stress occur at the midmembranous portion of the vocal folds during vocal fold vibration, which corresponds with the location at which vocal nodules often develop in humans (Jiang and Titze, 1994).

In addition to collision forces and impact stress, shearing forces are considered potentially harmful to vocal fold tissue. Shearing forces occur during vibration of the vocal folds, and prolonged or excessive phonation may result in excessive shearing that can cause damage to the vocal fold tissue (Courey *et al.*, 1996; Gray and Titze, 1988). Changes in muscle activation levels of the CT and TA could differentially affect mass and tension of the vocal fold layers, and could therefore result in changes to tissue displacement that might be associated with increased or decreased shearing forces.

In this study, the effects of controlled change of CT and TA muscle activity were investigated with the low-dimensional vocal fold model reported by Titze and Story (2002) and Story and Titze (1995) and applicable to the adult-male voice. The first purpose of this study was to determine the effects of CT and TA muscle activation on F_0 when the level of each muscle was independently manipulated. The second purpose was to determine whether increased CT or TA activation produced aerodynamic and mechanical changes to vocal fold vibration that might be harmful to vocal fold tissue. The aerodynamic quantities of maximum intraglottal pressure, maximum glottal flow, and MFDR, and the physical quantities of amplitude ratio (lower to upper cover mass) as well as vertical phase difference, were chosen due to their influence on mechanical stress in phonation and for their associated potential for increasing the risk of vocal fold damage in humans.

II. METHOD

Vocal fold vibration was simulated with a model designed to approximate the body-cover structure of the vocal folds, where upper and lower masses represent the cover, and a third, laterally positioned mass represents the body (Story and Titze, 1995). A schematic diagram of the model is shown in Fig. 1(a); the stiffness and damping elements have been combined to simplify the picture. The upper and lower masses are coupled to each other and to the body with spring and damping elements. The springs account for shearing forces and stiffness of the tissue, whereas the damping elements account for the energy losses that occur in the system. The two-mass representation of the cover allows the vertical phase difference of the mucosal wave to be represented. In addition, the separation of cover and body tissue in the model allows for individual specification of the mechanical properties of each tissue layer. For all of the simulations in this study, bilateral symmetry was assumed such that identical vibrations occur within the right and left vocal folds.

The vocal fold model was coupled to the pressures in the trachea and the vocal tract [see Fig. 1(b)] according to the aerodynamic and acoustic considerations specified by Titze (2002), thus allowing for self-sustained oscillation. Acoustic wave propagation in both the trachea and vocal tract was simulated in time-synchrony with the vocal fold model. This was carried out with a wave-reflection approach (digital waveguide) (e.g., Liljencrants, 1985) that included energy losses due to yielding walls, viscosity, and radiation at the lips (Story, 1995). The shape of the trachea and the vocal tract shown in Fig. 1(b) were maintained for all simulation cases in this study. The cross-sectional area of the epilaryngeal portion was set to be 0.5 cm^2, whereas the uniform tube representation of the pharynx and oral cavity was set at

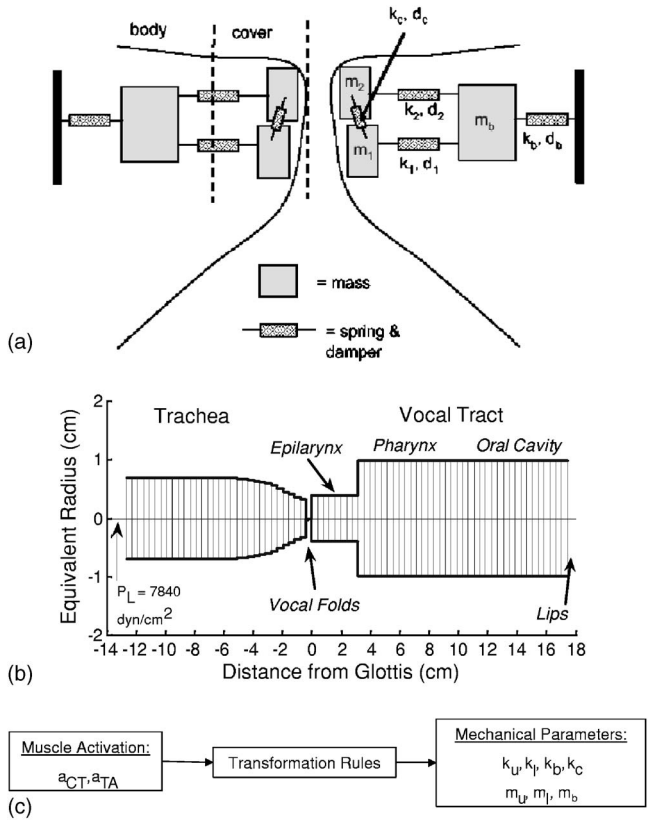

FIG. 1. Schematic representation of the simulation model: (a) three-mass model of the vocal folds, (b) tubular configuration of the trachea and vocal tract, and (c) transformation of CT and TA muscle activation levels into the mechanical parameters of the three-mass model.

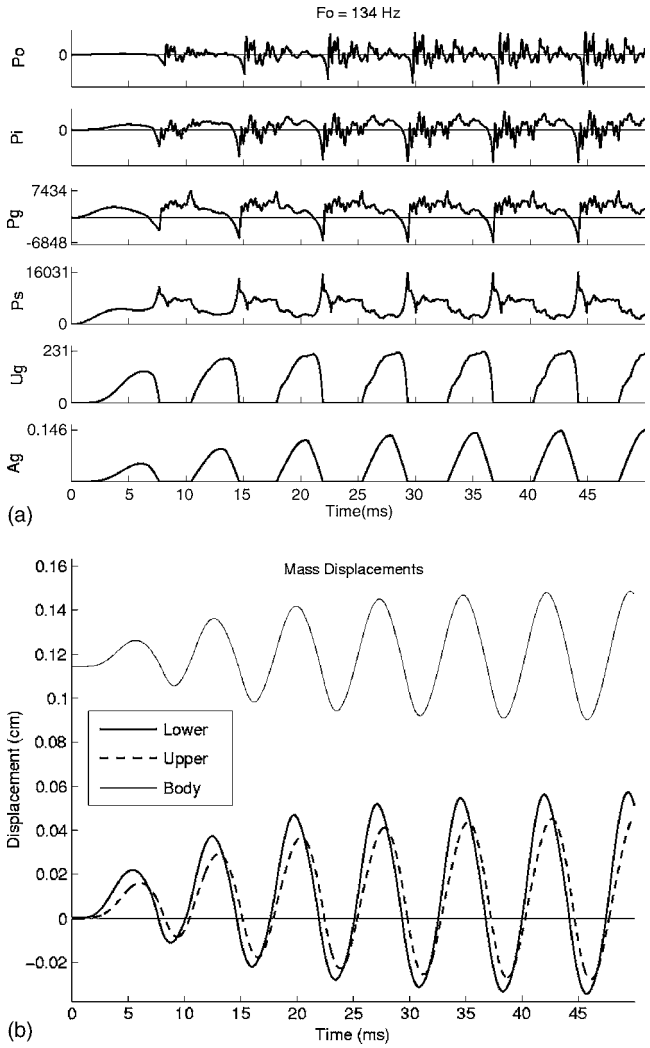

FIG. 2. Waveforms for the condition of a_{CT}=0.24, a_{TA}=0.24, depicting time-varying output pressure (Po), input pressure (Pi), intraglottal pressure (Pg), subglottal pressure (Ps), maximum glottal flow (Ug), and glottal area (Ag) as a function of time (a), and depicting changes in displacement of the lower and upper cover masses (bottom traces) and body mass (upper trace) as a function of time (b).

3 cm^2. A standard input pressure (lung pressure) of 7840 dyn/cm^2 was used for all simulations. A very slight prephonatory adduction angle was used (0.0001 cm). Lateral cricoarytenoid activation was set at a normalized level of 0.5 for all simulations.

The muscle activation levels of the CT and TA were specified in a normalized range from 0.0 to 1.0 and were intended to represent minimum to supramaximal activity within each muscle. As shown schematically in Fig. 1(c), these muscle activation levels were transformed, according to the "rules" developed by Titze and Story (2002), into the mechanical parameters of mass and stiffness. The rest dimensions of each of the vocal fold layers (length, thickness, depth) were also set to be the same as specified by Titze and Story (2002). Based on a comparison of EMG recordings and measurements of voice fundamental frequency reported by Titze *et al.* (1989) to the range of fundamental frequencies produced by the simulation model in Titze and Story (2002), it can be concluded that the normalized muscle activities produce physiologically realistic results.

The output waveforms produced by this model consisted of output pressure (radiated pressure at the lips), input pressure, glottal pressure, subglottal pressure, glottal flow, glottal area, and displacements of the upper and lower masses (Story and Titze, 1995; Titze, 2000; Titze *et al.*, 1989; Titze and Story, 2002). Shown in Figs. 2 and 3 are two different cases of CT muscle activation (a_{CT}) and TA muscle activation (a_{TA}) levels (a_{CT}=0.24, a_{TA}=0.24 for Fig. 2, and a_{CT}=0.24, a_{TA}=0.74 for Fig. 3). Note that the displacements of the upper and lower cover masses are strongly affected by levels of TA muscle activation and result in different waveform shapes for glottal flow (Ug) and glottal area (Ag).

To observe the output quantities of the model over a large range of muscle activation levels, simulations were generated for 2500 settings of a_{CT} and a_{TA}; the ranges of both a_{CT} and a_{TA} were divided into 50 evenly spaced increments from 0 to 1.0. For each simulation, the F_0 was determined with a zero-crossing detector and interpolation applied to the resulting glottal area signal. Additionally, vertical phase difference between the upper and lower masses, amplitude ratio of lower to upper mass displacement, maximum glottal flow, and maximum flow declination rate were computed for each simulation.

A contour plot showing lines of constant F_0 was produced with the "contour" function in MATLAB 7 (Mathworks, 2004), based on the collection of fundamental frequencies from each of the 2500 simulations. The resulting

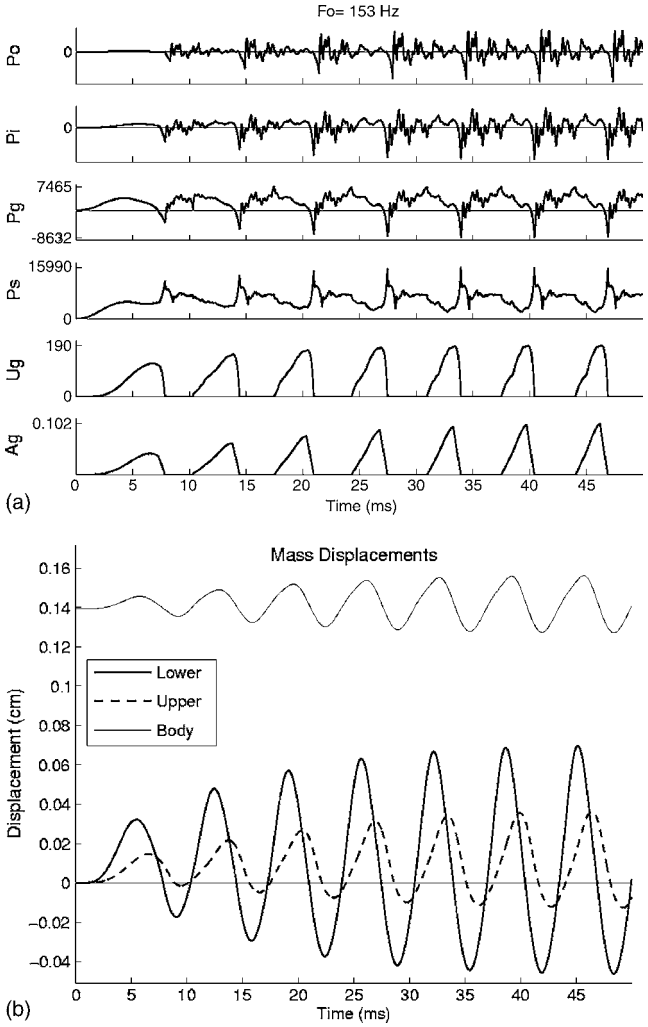

FIG. 3. Waveforms for the condition of a_{CT}=0.24, a_{TA}=0.74, depicting time-varying output pressure (Po), input pressure (Pi), intraglottal pressure (Pg), subglottal pressure (Ps), maximum glottal flow (Ug), and glottal area (Ag) as a function of time (a), and depicting changes in displacement of the lower and upper cover masses (bottom traces) and body mass (upper trace) as a function of time (b).

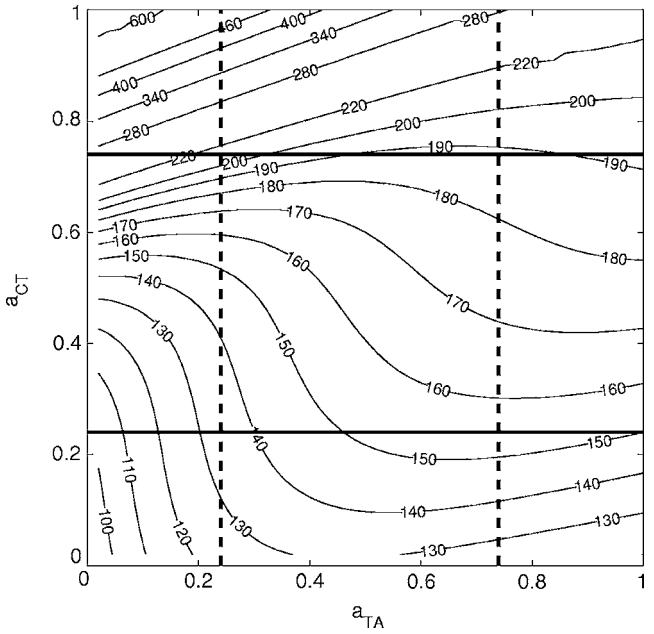

FIG. 4. Muscle activation plot (MAP) depicting isofrequency contour lines for normalized minimum to maximum thyroarytenoid (TA) activation levels and cricothyroid (CT) activation levels.

contour plot is shown in Fig. 4 and indicates how F_0 either changes or remains constant as the CT and TA activation levels change. Hence, it is referred to as a muscle activation plot (MAP).

The lower left portion of Fig. 4 represents CT and TA activation levels that are both low, and fundamental frequencies that are typical of conversational speech (Titze *et al.*, 1989). The upper right portion of Fig. 4 represents high levels of CT and TA activation. A speaker would presumably use muscle activation levels in this region for production of high fundamental frequencies. High CT activation levels and low TA activation levels are represented in the upper left portion, which encompasses the highest F_0 in the MAP. Four lines were drawn on the MAP to represent constant CT activation levels with progressively increasing TA activation (solid lines), and constant TA activation levels with progressively increasing CT activation (dashed lines). These lines were selected to represent low and high levels of constant CT activation (0.24 and 0.74, respectively) with TA activation varying from 0.0 to 1.0, and low and high levels of constant TA activation (0.24 and 0.74, respectively) with CT activation varying from 0.0 to 1.0. Thus, these four lines represent midpoints of the lower and upper portions of the MAP (constant CT) and midpoints of the right and left portions of the MAP (constant TA). Table I shows the values of the mechanical parameters of the three-mass model at the intersection points of the four lines indicated in Fig. 4. These values result from the rule-based transformation implemented by Titze and Story (2002).

By systematically increasing TA or CT activation levels from low to high along the selected lines, the isolated effects of these simulated muscle activations could be assessed. F_0, aerodynamic and physical quantities were assessed for each data line that represented a range from low to high TA activation levels (with constant CT) or low to high CT activation levels (with constant TA). From the output quantities described by the waveforms shown previously in Figs. 2 and 3 (or derivations from those quantities), changes to the following acoustic, aerodynamic, and physical quantities were assessed in response to systematic manipulation of TA and CT

TABLE I. Mechanical parameter values of the three-mass model at four settings of TA and CT activation levels. These result from the rule-based transformation implemented by Titze and Story (2002).

	$[a_{TA}, a_{CT}]$			
Mechanical parameters	[0.24, 0.24]	[0.24, 0.74]	[0.74, 0.24]	[0.74, 0.74]
Lower cover mass: m_1	0.0619	0.0612	0.0867	0.0864
Upper cover mass: m_2	0.0879	0.0869	0.0628	0.0626
Body mass: M	0.0978	0.0968	0.1973	0.1967
Lower cover stiffness: k_1	102 940	487 470	78 484	393 010
Upper cover stiffness: k_2	146 100	691 900	56 833	284 590
Body stiffness: K	188 210	430 190	529 310	481 360
Cover coupling stiffness: k_c	6739	11 857	5383	10 275

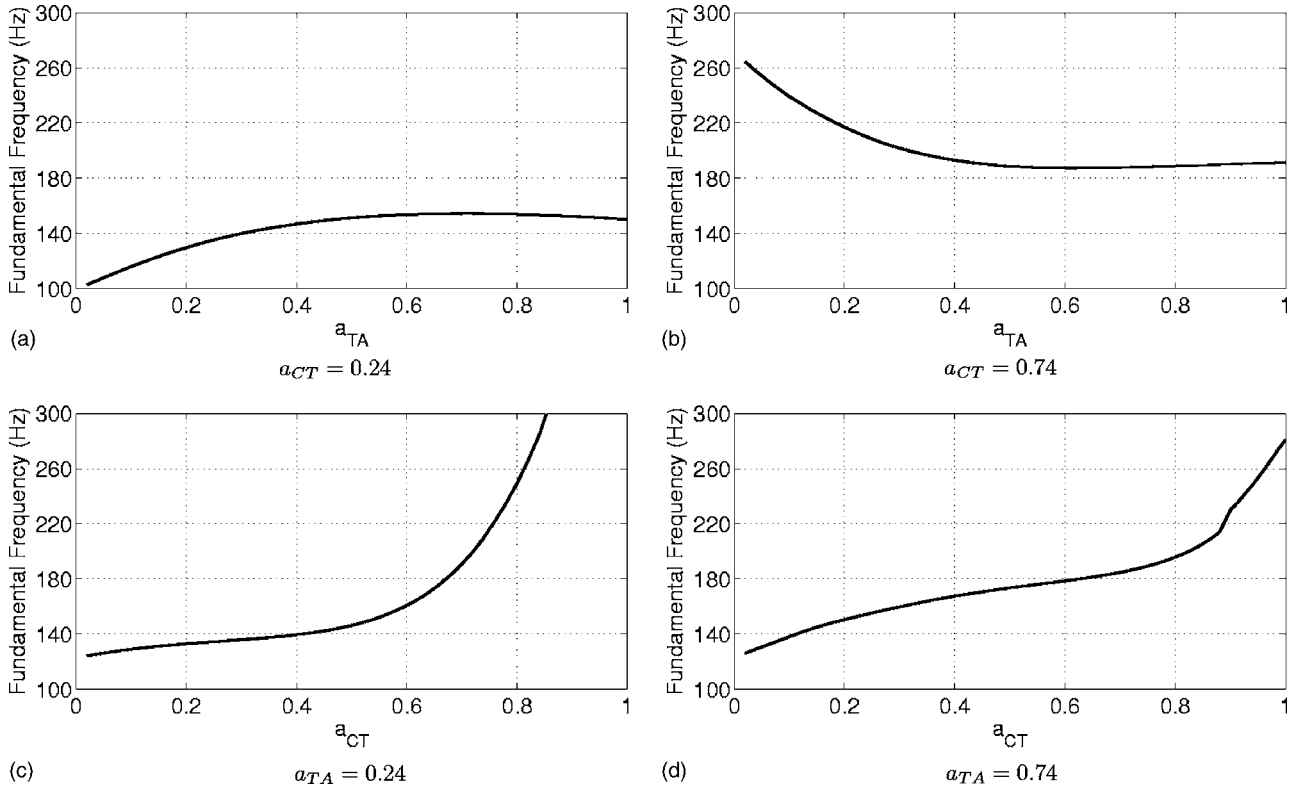

FIG. 5. Changes to fundamental frequency (F_0) as a function of increasing TA activation level when CT was constant at 0.24 (a) and 0.74 (b), and as a function of increasing CT activation when TA was constant at 0.24 (c) and 0.74 (d).

activation levels: fundamental frequency, maximum glottal flow, maximum flow declination rate (MFDR), vertical phase difference (number of degrees within a vibratory cycle that the lower mass leads the upper mass), and amplitude excursion ratios of the lower to upper cover masses. These quantities were chosen due to their potential impact on mechanical stress during vocal fold vibration. MFDR was measured as the most negative portion of the flow derivative within a glottal cycle. Maximum intraglottal pressure was analyzed but was found to be nearly constant at approximately 7300 dyn/cm^2 for all muscular settings, hence it was not included in the Secs. III and IV.

III. PREDICTIONS

To simplify presentation of the simulations, four different muscle activation cases were summarized for each output quantity: TA activation starting at a low normalized level (0.0) and progressively increasing to a high level (1.0) while (1) CT activation was held at a constant, low level of 0.24 (case 1) and (2) a constant, high level of 0.74 (case 2), and CT activation starting at a low normalized level (0.0) and progressively increasing to a high level (1.0) while (3) TA activation was held at a constant, low level of 0.24 (case 3) and (4) a constant, high level of 0.74 (case 4). All plots depict the change that occurred for each quantity as muscle activation was systematically increased.

A. Fundamental frequency

When CT activation level was held at 0.24, F_0 increased with increasing TA activation to an approximate TA activation level of 0.68, as shown in Fig. 5(a). For TA activation levels higher than 0.68, changes to F_0 plateaued. As can be seen by comparing the lower solid line in Fig. 4 and the fundamental frequencies in Fig. 5(a) for this same set of muscle activation combinations, F_0 started low at approximately the 100-Hz isofrequency contour and increased to cross the 150-Hz isofrequency contour as TA activation was maximally increased. At a constant CT activation level of 0.74 (case 2), F_0 decreased from 265 Hz to about 190 Hz as TA activation was increased to about 0.50, and then remained nearly constant, as shown in Fig. 5(b).

When TA activation was held at 0.24 (case 3), F_0 increased from 125 to 580 Hz as CT activation level increased, as shown in Fig. 5(c). The most substantial increase, however, occurred when CT activation exceeded the level of 0.60. To keep all plots in Fig. 5 on the same scale, the increase in F_0 is only shown up to 300 Hz. This increase in F_0 can also be observed in Fig. 4 along the vertical dashed line denoting TA activation level of 0.24. Here the F_0 levels begin below the 130-Hz isofrequency contour, and extend to the 460-Hz isofrequency contour (see Fig. 4). For a constant TA level of 0.74 (case 4), F_0 showed a small, gradual increase as CT activation was increased throughout the range of 0 to 1.0, as shown in Fig. 5(d).

B. Maximum glottal flow (Max Ug)

Max Ug decreased with increased TA activation and CT activation constant at 0.24 (case 1), as demonstrated in Fig. 6(a). As previously shown in Figs. 2(a) and 3(a), changes to the shape of the flow waveform were substantial when TA

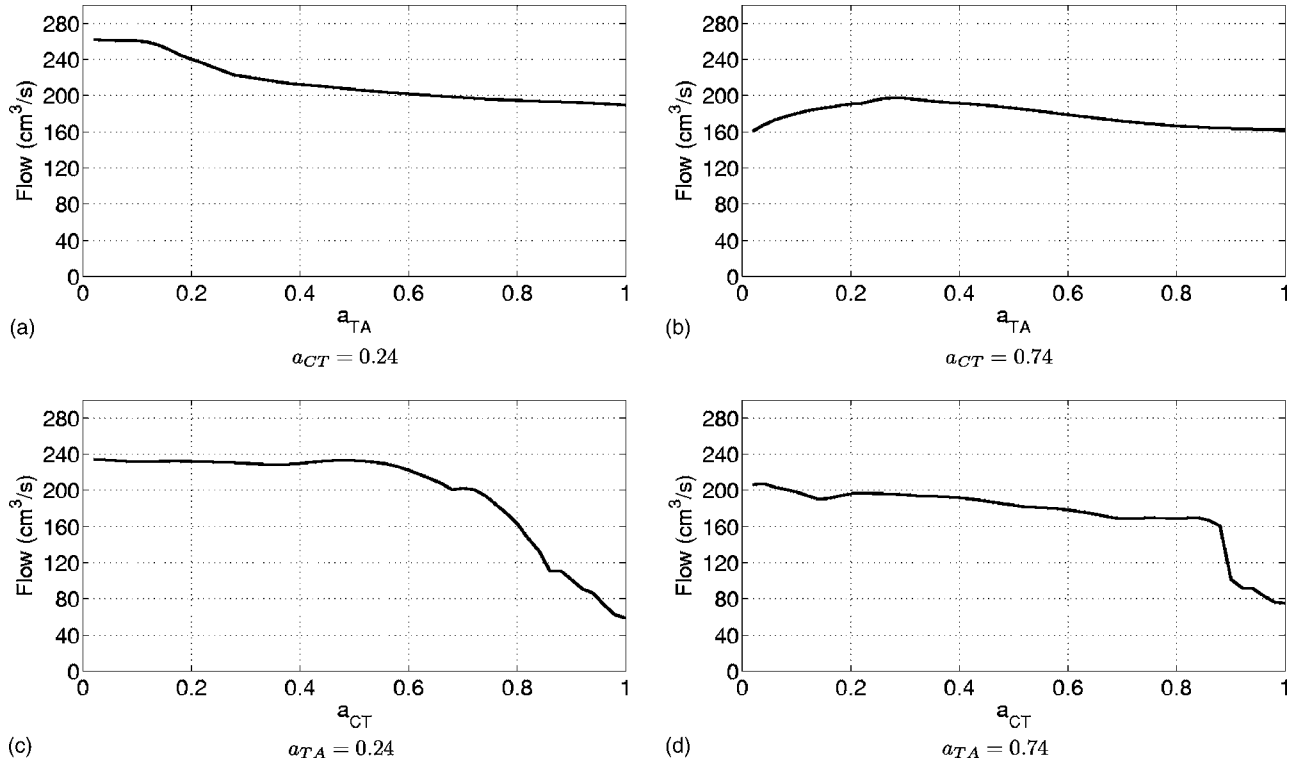

FIG. 6. Changes to maximum glottal flow as a function of increasing TA activation level when CT was constant at 0.24 (a) and 0.74 (b), and as a function of increasing CT activation when TA was constant at 0.24 (c) and 0.74 (d).

was increased and CT was held at a low level. At low TA activation levels, a rounded, more gradual flow waveform was produced that was slightly skewed to the right relative to the glottal area waveform. At high TA levels, the flow waveform had a sharp upper cutoff, with a triangular shape that followed the changes to the glottal area waveform. Changes in Max Ug as a function of increasing TA activation with CT activation constant at 0.74 (case 2) were variable and dependent on TA activation level, as shown in Fig. 6(b). Max Ug generally increased between the TA activation levels of 0.0 to approximately 0.28, and then generally decreased for TA values greater than 0.28.

When TA activation was held at 0.24 (case 3), Max Ug decreased as CT activation was increased beyond 0.60, as demonstrated in Fig. 6(c). Changes in Max Ug as CT activation was increased and TA activation was held at 0.74 (case 4) were small but generally in a decreasing direction, as shown in Fig. 6(d).

C. Maximum flow declination rate (MFDR)

MFDR changes varied when CT activation was held at a constant, low level and TA activation was increased (case 1), as shown in Fig. 7(a). MFDR showed an initial decrease as TA activation was increased from 0 to approximately 0.24. Beyond those low TA values, MFDR showed a gradual, steady increase as TA activation was increased. As demonstrated in Fig. 7(b), MFDR showed a gradual, small decrease as TA activation increased and CT activation was held at a constant, high level (case 2). This decrease leveled off at TA values of approximately 0.70 to 0.80.

As shown in Fig. 7(c), when CT activation was increased and TA activation was held at a constant, low value (case 3), changes to MFDR varied by the level of CT activation. Little change in MFDR was evidenced as CT activation was increased to approximately 0.60. As CT values were increased from 0.60 to 1.0, MFDR showed a fluctuating but substantial decrease. MFDR decreased substantially (from approximately 1 400 000 to 350 000 cm^3/s^2) as CT activation was increased and TA activation was held constant at 0.74 [case 4, Fig. 7(d)].

D. Amplitude ratio (of lower to upper mass)

The amplitude ratios of lower to upper cover masses gradually increased as TA activation increased when CT was held at 0.24 (case 1), as demonstrated in Fig. 8(a). The displacement waveforms shown in Figs. 2(b) and 3(b) exemplify the contrast in amplitude ratios between low and high TA activation conditions. Amplitude ratios minimally changed as TA activation increased and CT activation stayed constant at 0.74 (case 2), as shown in Fig. 8(b).

Amplitude ratios of the lower to upper cover masses showed minimal change as CT activation increased and TA was held at 0.24 [case 3, Fig. 8(c)]. Amplitude ratios decreased as CT activation increased beyond 0.3 and TA was held at 0.74 (case 4), as shown in Fig. 8(d).

E. Vertical phase difference (of the upper and lower cover masses)

With CT activation constant at 0.24 (case 1), vertical phase difference increased substantially (from approximately

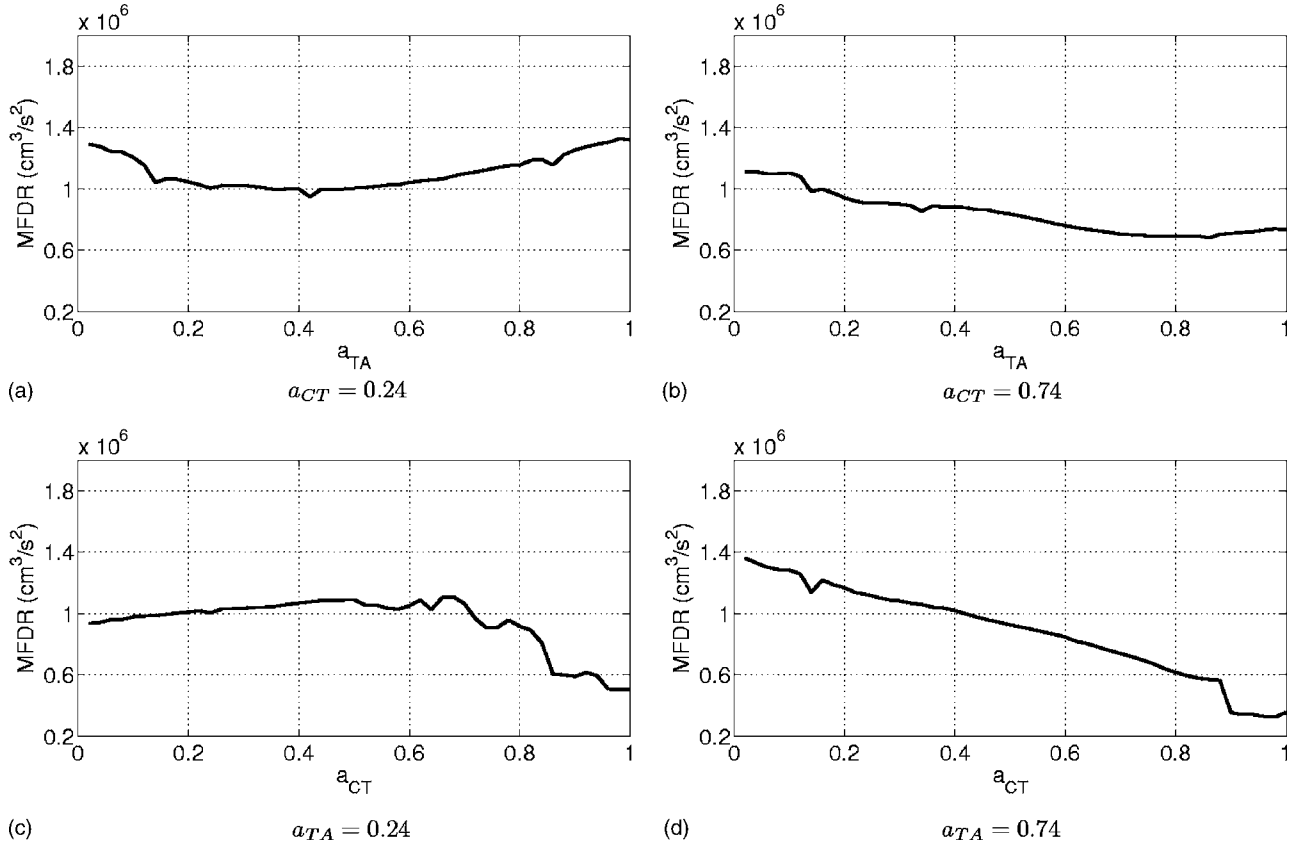

FIG. 7. Changes to maximum flow declination rate as a function of increasing TA activation level when CT was constant at 0.24 (a) and 0.74 (b), and as a function of increasing CT activation when TA was constant at 0.24 (c) and 0.74 (d).

5 to 81 deg) as TA activation increased [Fig. 9(a)]. Figure 9(b) shows that when CT activation was held at 0.74 (case 2), little change in vertical phase difference was demonstrated as TA activation was increased.

At a constant TA activation level of 0.24 (case 3), vertical phase difference decreased (from approximately 58 deg to approximately 17 deg) as CT activation increased to approximately 0.58 [Fig. 9(c)]. Between CT levels of 0.58 and 1.0, vertical phase difference leveled off and then increased slightly. When TA activation was held at 0.74 (case 4), vertical phase difference decreased greatly (approximately 84 deg to as low as 6 deg) as CT activation was increased [Fig. 9(d)].

IV. DISCUSSION

The purpose of this study was to investigate the predicted effects of independent manipulations to CT and TA muscle activation levels on F_0 and vocal fold vibration characteristics of adult males. These intrinsic laryngeal muscles are vital to F_0 control. Understanding the effects of these muscles on aerodynamic and physical quantities of vocal fold vibration, without the complications of other intrinsic and extrinsic laryngeal factors, can provide important insights regarding normal and disordered voice physiology. However, systematic increase of one muscle only, while controlling activation of the antagonist muscle, is difficult for a real speaker to achieve. Use of the three-mass model for vocal fold vibration provided a tool for predicting the independent, simulated effects of manipulating the CT and TA muscles during vocal fold vibration. This study is the first to provide predicted, causal effects of CT and TA activation on vocal fold aerodynamics and biomechanics.

A. Effect on fundamental frequency

Simulated effects of CT activation on F_0 highlighted the contrasting degree to which CT affects F_0, dependent on the level of TA activation present. Whereas increased CT activation consistently resulted in increased F_0, the greatest degree of F_0 change occurred with low TA activation levels when CT activation levels exceeded 60% of the maximum. When TA activation was high, changes in F_0 that resulted from increased CT were much smaller. This can be explained through the cover-body theory of vibration by the notion that at high TA levels, the vocal fold body is already quite stiff. Increases in CT activation would therefore be less effective in increasing overall vocal fold tension, and the increase in F_0 would be less pronounced. The simulated effects of CT activation in this study support the findings from electromyography studies showing the CT muscle to be a primary controller of F_0 (Atkinson, 1978; Faaborg-Andersen *et al.*, 1967).

The simulated effects of TA activation support the notion that TA activation can either raise or lower F_0, as predicted by Hirano (1974) and Titze *et al.* (1989). Electromyography recordings from a small number of subjects (Titze *et al.*, 1989) provided preliminary evidence supporting the bio-

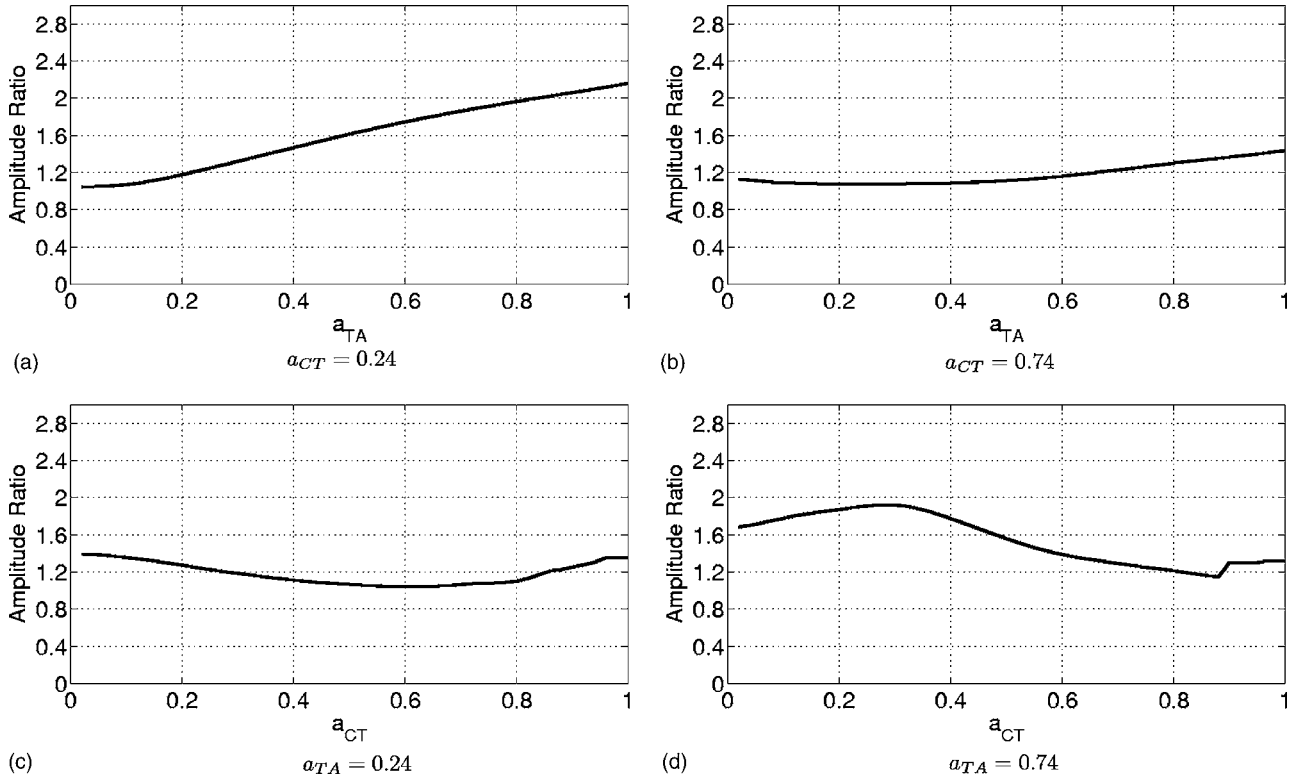

FIG. 8. Changes to amplitude ratio as a function of increasing TA activation level when CT was constant at 0.24 (a) and 0.74 (b), and as a function of increasing CT activation when TA was constant at 0.24 (c) and 0.74 (d).

mechanical theory outlined by Titze *et al.* (1989). However, the findings from this study expanded on that data by running simulations for thousands of muscle activation combinations and determining the precise conditions in which TA activation was predicted to raise or lower F_0, dependent on concurrent CT activation. With low CT activation levels, TA activation increased F_0 up to approximately 60% of its maximum activation, without further changes to F_0 above that activation level. In contrast, when CT activation was high, increasing TA activation resulted in *decreased* F_0 until about mid TA activation levels, after which no further F_0 change was realized. At low CT and TA activation levels, increased TA activation would increase the tension in the relatively slack vocal folds, resulting in an increased F_0 for the fundamental frequencies of approximately 100 to 150 Hz in this adult-male model. In contrast, with high CT activation and low TA activation levels (yielding high fundamental frequencies), the vocal folds would be stiff and tense, and any increased TA activation would shorten the vocal folds and produce sufficient reduction in tension to result in a lowering of F_0.

B. Effect on aerodynamic and physical quantities

Modification of CT and TA muscle activation levels had several pronounced effects on aerodynamic and physical quantities of vocal fold vibration. These quantities influence vocal fold impact stress and shearing stress, parameters that contribute to overall mechanical stress during vocal fold vibration (Titze, 1994). Glottal area and glottal airflow generally decreased as both TA and CT muscle activation levels were increased. Decreased airflow during vibration may be optimal for a speaker who is trying to conserve airflow and driving pressure, and may allow the speaker to limit their frequency of respiratory replenishment or depth of inspiration. However, if this airflow conservation occurs as a result of increased intrinsic laryngeal muscle activation (TA and/or CT), the cost to the speaker relative to muscle expenditure may outweigh the airflow conservation benefits.

Maximum flow declination rate (MFDR) has been used as an indicator of velocity of vocal fold closure (Hillman *et al.*, 1989), and increased MFDR may be associated with increased vocal fold collision forces. MFDR was generally at its highest when the simulated difference in levels of CT and TA activation was greatest. Specifically, when muscle combinations of high TA activation were coupled with low CT activation, MFDR was high. As CT and TA activation levels approached each other, MFDR generally decreased. Therefore, when high TA muscle activation is used with low CT muscle activation, velocity of vocal fold closure may be increased and may result in increased collision forces or impact stress of the vocal folds. When simulated activation for both the CT and TA were high, MFDR values were quite low. Interestingly, speakers have been observed to use approximately equal increases in both CT and TA activation when increasing F_0 (Titze *et al.*, 1989), effectively utilizing a diagonal from the lower left and upper right corners in the muscle activation plot (Fig. 4). This muscle use strategy would apparently result in lower MFDR values. It should be noted that, at times, an increase in MFDR is desirable, such as when increased voice intensity is needed. Two trained male singers showed different muscle use strategies for increasing F_0, primarily relying on increased CT activation

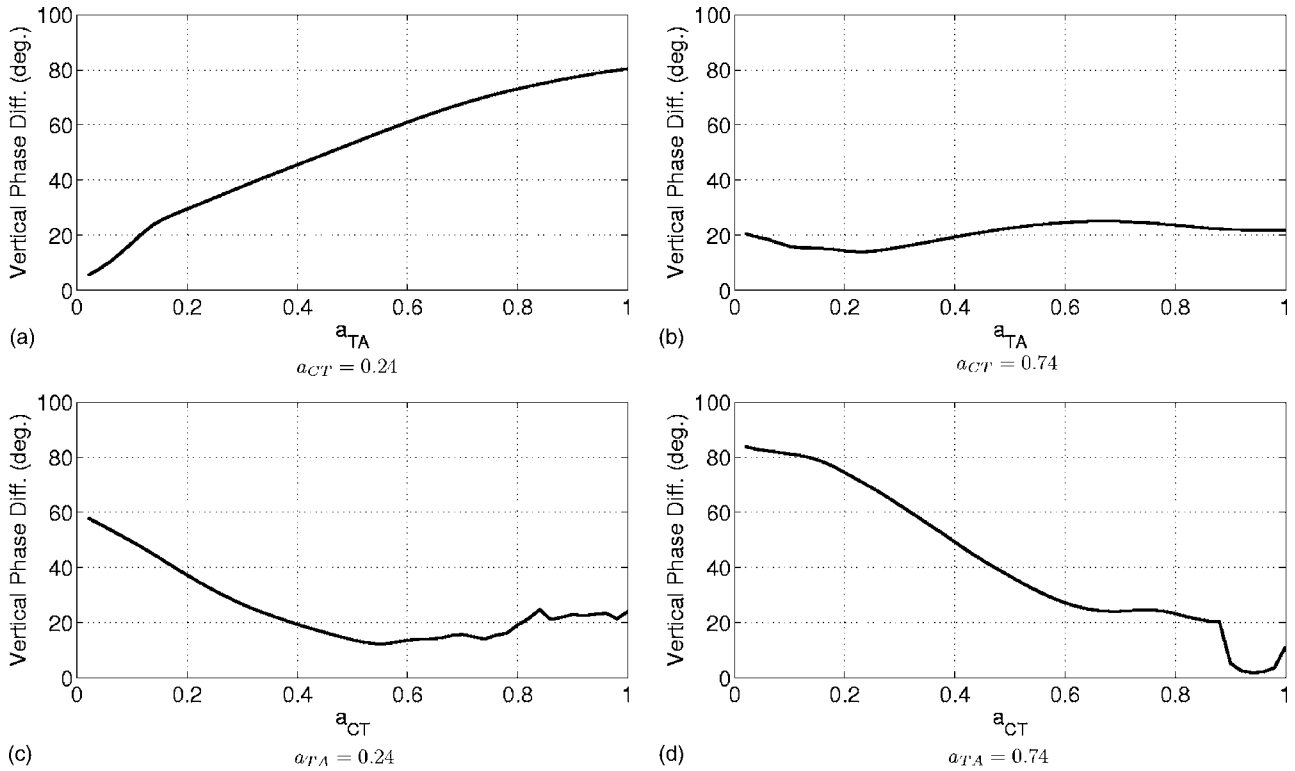

FIG. 9. Changes to vertical phase difference as a function of increasing TA activation level when CT was constant at 0.24 (a) and 0.74 (b), and as a function of increasing CT activation when TA was constant at 0.24 (c) and 0.74 (d).

while maintaining lower TA activation (Titze *et al.*, 1989). This strategy might result in increased MFDR, which would be important for a singer who needs to achieve both increased F_0 and increased intensity.

The amplitude ratio and vertical phase difference of the lower to upper cover masses are important to the self-oscillating nature of vocal fold vibration (Titze, 1988), but excessive vertical phase differences can also contribute to shearing forces (Titze, 1994). No previous studies have documented the effects of varying levels of intrinsic laryngeal muscle activation on these quantities. In this simulation study, substantial changes in the amplitude ratio occurred when TA activation was increased and CT muscle activation was low, creating a large CT to TA activation differential. In this case, the amplitude ratio more than doubled. Thus, at a low F_0 range, when the vocal folds would be relatively slack, increased TA activation resulted in increased excursion of the lower cover mass and decreased excursion of the upper cover mass. These changes were accompanied by a large increase in vertical phase difference between the upper and lower cover masses, due to the upper mass not paralleling the increased excursion of the lower mass, with resulting differences in phase as TA muscle activation was increased. These increases in amplitude ratio and vertical phase difference resulted in substantial changes to the glottal area and glottal flow waveforms. When TA activation was high and CT activation was low, the glottal area waveform became very sharp, with an abrupt cutoff point between the opening and closing phases of vibration.

Abrupt changes to vocal fold dynamics can result in increased mechanical stress during vibration. An increase in vertical phase difference of the upper and lower cover masses during vibration will result in an increase in shearing forces on the vocal folds; the greater phase asymmetry of the upper and lower masses would mean increased shearing of the cover tissue that connects these masses. If shearing forces are harmful to vocal fold tissue, increased levels of TA activation may place an individual at risk for damage or change to the vocal fold tissue due to an increase in shearing forces that occurs as TA activation is increased. The simulated effects of muscle activation support Hillman *et al.* (1989), who suggested that increased levels of muscle activation associated with adducted hyperfunction might result in increased vocal fold stiffness, high velocity of tissue movement, and an increase in vocal fold collision forces, all contributing to an increased risk of vocal fold pathology. These authors theorized that the vocal fold dynamics associated with increased muscle activation would also result in increased amplitude of vocal fold excursion, in turn contributing to increased collision forces. The simulations from the present study indicated that with increased TA activation and relatively low CT activation, the largest increase in amplitude excursion will occur in the lower cover mass, with a greater amplitude differential at these muscle activation levels between the lower and upper cover mass movement. Therefore, in addition to the increased impact forces, increased shearing forces may compound the mechanical stress that is placed on the vocal folds.

There are few studies documenting intrinsic laryngeal muscle activation levels in people with voice disorders. Most of the available literature has been conducted with individuals presenting with spasmodic dysphonia, a voice disorder of

neurologic origin. Several investigators have not found differences in intrinsic laryngeal muscle activation between control subjects and those with spasmodic dysphonia (Van Pelt *et al.*, 1994; Watson *et al.*, 1991). Interestingly, the studies that have found group differences have consistently reported increased activity of the thyroarytenoid muscle in people with spasmodic dysphonia as compared to control subjects (Cyrus *et al.*, 2001; Nash and Ludlow, 1996; Schaefer *et al.*, 1992). Speech samples that elicited these group differences varied from phonation breaks only (Nash and Ludlow, 1996) to speech with and without breaks (Cyrus *et al.*, 2001) to repeated consonant-vowel-consonant tasks (Schaefer *et al.*, 1992). One hypothesis based on the predictions obtained from this study is that increased TA activation may alter displacement characteristics of the vocal fold cover and body, contributing to decreased phonatory stability during vibration. In voice disorders that occur in the absence of neurologic or structural laryngeal pathology (often referred to as functional voice disorders), increased intrinsic and extrinsic laryngeal muscle activation is frequently assumed but has generally not been objectively documented (Eustace *et al.*, 1996; Morrison and Rammage, 1993; Morrison *et al.*, 1983; Stemple *et al.*, 1995). The simulations produced in this study indicate that certain combinations of intrinsic laryngeal muscle activation may result in aerodynamic and physical characteristics of vocal fold vibration that could place an individual at risk for voice problems. However, modeling studies must be followed by *in vivo* speech recordings in people with and without voice disorders to validate these predictions.

There are several limitations to the present study, and critical future directions for research in this area. To simplify the interpretation of results, only a realistic epilaryngeal and tracheal configuration was included, with the remaining vocal tract modeled as an open tube. To more realistically depict the acoustic, aerodynamic, and physical changes associated with variation of muscle activation and epilaryngeal area, modeling of particular vocal tract configurations representing vowels such as /a/ or /i/ would be useful. Furthermore, this study controlled the configuration of the trachea and held input pressure (lung pressure) at a constant value to isolate the effects of CT and TA muscle activation. Manipulation of parameters such as driving pressure would be expected to influence F_0 (Baer, 1979; Hixon *et al.*, 1971). An important step in future studies would be manipulating such parameters in conjunction with CT and TA muscle activations and determining the predicted outcomes. Likewise, the output parameters assessed in this study were limited so that the length and interpretability of the predictions would not be too unwieldy. Due to the finding of increased skewing of the glottal pulse under simulated conditions of high TA activation and low CT activation in this study, future studies might include the speed quotient as a measure of the symmetry of the open phase (Baken and Orlikoff, 2000).

A major limitation to all vocal fold modeling studies is that the effects that are obtained by experimentally manipulating various parameters may not be evidenced in the human with those same manipulations. In this study, it was possible to manipulate intrinsic laryngeal muscles independent of other factors. In humans, these muscle changes would occur with a probable concomitant increase in other laryngeal and pharyngeal muscles, and the effects of those muscle changes would not be reflected in the present modeling study. Thus, modeling studies can provide predictions regarding the effects of controlled manipulation of variables, but must be followed by *in vivo* studies to validate these predictions. In attempting to draw inferences regarding changes to aerodynamic and physical quantities and their implications for risk of vocal fold damage, it is also important to note that the three-mass model of vibration cannot depict tissue damage. Thus, the actual risk for tissue damage associated with these changes to vocal fold dynamics is unknown. Finally, the current model has been developed on male speakers and may have limited applicability to female voice production. Future modifications to this computational model are therefore needed to adequately represent female voice biomechanics, as many voice disorders occur more frequently in women.

C. Conclusions

The number of variables that affect F_0 in speech and the interdependence of these variables make the study of F_0 control difficult *in vivo*. The three-mass model of adult-male vocal fold vibration allows for the isolated simulation of several variables that are critical to F_0 control, such as CT and TA muscle activation. By manipulating activation of one intrinsic laryngeal muscle while holding other variables constant, the simulated effects of that muscle on F_0, as well as on aerodynamic and physical characteristics of vibration, can be studied. The aerodynamic and physical quantities analyzed in this study were chosen due to their contribution to vocal fold dynamics and their influence on various forms of mechanical stress during vibration. F_0 was greatly affected by the simulated manipulation of CT and TA muscle activation, as were the aerodynamic quantities of glottal airflow and MFDR. Physical quantities of amplitude ratio and vertical phase difference were also affected by simulated muscle activation. A simulated increase in TA activation with relatively low CT activation substantially increased both the amplitude ratio and vertical phase difference. These aerodynamic and physical changes would be expected to increase both vocal fold collision forces and shearing forces, which may increase the potential for vocal fold tissue damage.

Alipour-Haghighi, F., and Titze, I. R. (**1983**). "Simulation of particle trajectories of vocal fold tissue during phonation," *in Vocal Fold Physiology: Biomechanics, Acoustics, and Phonatory Control*, edited by I. R. Titze and R. C. Scherer (Denver Center for the Performing Arts, Denver, CO), pp. 183–190.

Alipour-Haghighi, F., and Titze, I. R. (**1991**). "Elastic models of vocal fold tissues," J. Acoust. Soc. Am. **90**, 1326–1331.

Atkinson, J. E. (**1978**). "Correlation analysis of the physiological factors controlling fundamental voice frequency," J. Acoust. Soc. Am. **63**, 211–222.

Bachorowski, J.-A., and Owren, M. J. (**1999**). "Acoustic correlates of talker sex and individual talker identity are present in a short vowel segment produced in running speech," J. Acoust. Soc. Am. **106**, 1054–1063.

Baer, T. (**1979**). "Reflex activation of laryngeal muscles by sudden induced subglottal pressure changes," J. Acoust. Soc. Am. **65**, 1271–1275.

Baken, R. J., and Orlikoff, R. F. (**2000**). *Clinical Measurement of Speech and Voice, 2nd ed.* (Singular, Thomson Learning, San Diego, CA).

Baker, K. K., Ramig, L. A., Sapir, S., Luschei, E. S., and Smith, M. E.

(**2001**). "Control of vocal loudness in young and old adults," J. Speech Lang. Hear. Res. **44**, 297–305.
Boone, D. R., and McFarlane, S. C. (**2000**). *The Voice and Voice Therapy, 6th ed.* (Allyn and Bacon, Needham Heights, MA).
Brown, B. L., Strong, W. J., and Rencher, A. C. (**1974**). "Fifty-four voices from two: The effects of simultaneous manipulations of rate, mean fundamental frequency, and variance of fundamental frequency on ratings of personality from speech," J. Acoust. Soc. Am. **55**, 313–318.
Case, J. L. (**2002**). *Clinical Management of Voice Disorders, 4th ed.* (Pro-ed, Austin, TX).
Courey, M. S., Shohet, J. A., Scott, M. A., and Ossoff, R. H. (**1996**). "Immunohistochemical characterization of benign laryngeal lesions," Ann. Otol. Rhinol. Laryngol. **105**, 525–531.
Cyrus, C. B., Bielamowicz, S., Evans, F. J., and Ludlow, C. L. (**2001**). "Adductor muscle activity abnormalities in abductor spasmodic dysphonia," Otolaryngol.-Head Neck Surg. **124**(1), 23–30.
Eustace, C. S., Stemple, J. C., and Lee, L. (**1996**). "Objective measures of voice production in patients complaining of laryngeal fatigue," J. Voice **10**(2), 146–154.
Faaborg-Andersen, K., Yanagihara, N., and von Leden, H. (**1967**). "Vocal pitch and intensity regulation: A comparative study of electrical activity in the cricothyroid muscle and the airflow rate," Arch. Otolaryngol. **85**, 122–128.
Finnegan, E. M., Luschei, E. S., and Hoffman, H. T. (**2000**). "Modulations in respiratory and laryngeal activity associated with changes in vocal intensity," J. Speech Lang. Hear. Res. **43**, 934–950.
Gay, T., Hirose, H., Strome, M., and Sawashima, M. (**1972**). "Electromyography of the intrinsic laryngeal muscles during phonation," Ann. Otolaryngol. **81**, 401–409.
Gelfer, M. P., and Schofield, K. J. (**2000**). "Comparison of acoustic and perceptual measures of voice in male-to-female transsexuals perceived as female versus those perceived as male," J. Voice **14**, 22–33.
Gray, S. D., and Titze, I. R. (**1988**). "Histologic investigation of hyperphonated canine vocal cords," Ann. Otol. Rhinol. Laryngol. **97**, 381–388.
Hillman, R. E., Holmberg, E. B., Perkell, J. S., Walsh, M., and Vaughan, C. (**1989**). "Objective assessment of vocal hyperfunction: An experimental framework and initial results," J. Speech Hear. Res. **32**, 373–392.
Hillman, R. E., Holmberg, E. B., Perkell, J. S., Walsh, M., and Vaughan, C. (**1990**). "Phonatory function associated with hyperfunctionally related vocal fold lesions," J. Voice **4**(1), 52–63.
Hirano, M. (**1974**). "Morphological structure of the vocal cord and its variations," Folia Phoniatr. **26**, 89–94.
Hirano, M. (**1988**). "Behavior of laryngeal muscles of the late William Vennard," J. Voice **2**(4), 291–300.
Hirano, M., Ohala, J., and Vennard, W. (**1969**). "The function of laryngeal muscles in regulating fundamental frequency and intensity of phonation," J. Speech Hear. Res. **12**, 616–628.
Hirano, M., Vennard, W., and Ohala, J. (**1970**). "Regulation of register, pitch and intensity of voice: An electromyographic investigation of intrinsic laryngeal muscles," Folia Phoniatr. **22**, 1–20.
Hixon, T. J., Klatt, D., and Mead, J. (**1971**). "Influence of forced transglottal pressure changes on vocal fundamental frequency," J. Acoust. Soc. Am. **49**, 105(A).
Holmberg, E. B., Hillman, R. E., and Perkell, J. S. (**1988**). "Glottal airflow and transglottal air pressure measurements for male and female speakers in soft, normal, and loud voice," J. Acoust. Soc. Am. **84**, 511–529.
Holmberg, E. B., Hillman, R. E., and Perkell, J. S. (**1989**). "Glottal airflow and transglottal air pressure measurements for male and female speakers at low, normal, and high pitch," J. Voice **3**(4), 294–305.
Holmberg, E. B., Hillman, R. E., Perkell, J. S., and Gress, C. (**1994**). "Relationships between intra-speaker variation in aerodynamic measures of voice production and variation in SPL across repeated recordings," J. Speech Hear. Res. **37**, 484–495.
Ishizaka, K., and Flanagan, J. L. (**1972**). "Synthesis of voiced sounds from a two-mass model of the vocal cords," Bell Syst. Tech. J. **51**, 1233–1268.
Jacques, R. D., and Rastatter, M. P. (**1990**). "Recognition of speaker age from selected acoustic features as perceived by young and older listeners," Folia Phoniatr. **42**(3), 118–124.
Jiang, J., and Titze, I. R. (**1994**). "Measurement of vocal fold intraglottal pressure and impact stress," J. Voice **8**(2), 132–144.
Kempster, G. B., Larson, C. R., and Kistler, M. K. (**1988**). "Effects of electrical stimulation of cricothyroid and thyroarytenoid muscles on voice fundamental frequency," J. Voice **2**(3), 221–229.
Liljencrants, J. (**1985**). "Speech synthesis with a reflection-type analog," DS dissertation, Dept. of Speech Commun. and Music Acoust., Royal Inst. of Tech., Stockholm, Sweden.
Mathworks (**2004**). MATLAB 7 Version 4; (The MathWorks, Inc., Natick, MA).
Morrison, M. D., and Rammage, L. A. (**1993**). "Muscle misuse voice disorders: Description and classification," Acta Oto-Laryngol. **113**, 428–434.
Morrison, M. D., Rammage, L. A., Belisle, G. M., Pullan, C. B., and Nichol, H. (**1983**). "Muscular tension dysphonia," J. Otolaryngol. **12**(5), 302–306.
Nash, E. A., and Ludlow, C. L. (**1996**). "Laryngeal muscle activity during speech breaks in adductor spasmodic dysphonia," Laryngoscope **106**(4), 484–489.
Nasri, S., Sercarz, J. A., Azizzadeh, B., Kreiman, J., and Berke, G. S. (**1994**). "Measurement of adductory force of individual laryngeal muscles in an in vivo canine model," Laryngoscope **104**(10), 1213–1218.
Poletto, C. L., Verdun, L. P., Strominger, R., and Ludlow, C. L. (**2004**). "Correspondence between laryngeal vocal fold movement and muscle activity during speech and nonspeech gestures," J. Appl. Physiol. **97**, 858–866.
Schaefer, S. D., Roark, R. M., Watson, B. C., Kondraske, G. V., Freeman, F. J., Butsch, R. W., *et al.* (**1992**). "Multichannel electromyographic observations in spasmodic dysphonia patients and normal control subjects," Ann. Otol. Rhinol. Laryngol. **101**, 67–75.
Shipp, T., and McGlone, R. E. (**1971**). "Laryngeal dynamics associated with voice frequency change," J. Speech Hear. Res. **14**, 761–768.
Stemple, J. C., Stanley, J., and Lee, L. (**1995**). "Objective measures of voice production in normal subjects following prolonged voice use," J. Voice **9**(2), 127–133.
Story, B. H. (**1995**). "Physiologically-based speech simulation using an enhanced wave-reflection model of the vocal tract," Ph.D. dissertation, University of Iowa.
Story, B. H., and Titze, I. R. (**1995**). "Voice simulation with a body cover model of the vocal folds," J. Acoust. Soc. Am. **97**, 1249–1260.
Tanaka, S., and Tanabe, M. (**1986**). "Glottal adjustment for regulating vocal intensity," Acta Oto-Laryngol. **102**, 315–324.
Thorsen, N. G. (**1980**). "A study of the perception of sentence intonation-evidence from Danish," J. Acoust. Soc. Am. **67**, 1014–1030.
Titze, I. R. (**1988**). "The physics of small amplitude oscillation of the vocal folds," J. Acoust. Soc. Am. **83**, 1536–1551.
Titze, I. R. (**1991**). "Mechanisms underlying the control of fundamental frequency," *in Vocal Fold Physiology: Acoustic, Perceptual, and Physiological Aspects of Voice Mechanisms*, edited by J. Gauffin and B. Hamarberg (Singular, San Diego, CA), pp. 129–138.
Titze, I. R. (**1994**). "Mechanical stress in phonation," J. Voice **8**(2), 99–105.
Titze, I. R. (**2000**). *Principles of Voice Production* (National Center for Voice and Speech, Iowa City).
Titze, I. R. (**2002**). "Regulating glottal airflow in phonation: Application of the maximum power transfer theorem to a low dimensional phonation model," J. Acoust. Soc. Am. **111**, 367–376.
Titze, I. R., and Story, B. H. (**2002**). "Rules for controlling low-dimensional vocal fold models with muscle activation," J. Acoust. Soc. Am. **112**, 1064–1076.
Titze, I. R., Luschei, E. S., and Hirano, M. (**1989**). "Role of the thyroarytenoid muscle in regulation of fundamental frequency," J. Voice **3**(3), 213–224.
Van den Berg, J., and Tan, T. S. (**1959**). "Results of experiments with human larynxes," Pract. Otorhinolaryngol. (Basel) **21**, 425–450.
Van Pelt, F., Ludlow, C. L., and Smith, P. J. (**1994**). "Comparison of muscle activation patterns in adductor and abductor spasmodic dysphonia," Ann. Otol. Rhinol. Laryngol. **103**, 192–200.
Watson, B. C., Schaefer, S. D., Freeman, F. J., Dembowski, J., Kondraske, G. V., and Roark, R. M. (**1991**). "Laryngeal electromyographic activity in adductor and abductor spasmodic dysphonia," J. Speech Hear. Res. **34**, 473–482.