A virtual trajectory model predicts differences in vocal fold kinematics in individuals with vocal hyperfunction[a)]

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A simple, one degree of freedom virtual trajectory model of vocal fold kinematics was developed to investigate whether kinematic features of vocal fold movement confirm increased muscle stiffness. Model simulations verified that increases in stiffness were associated with changes in kinematic parameters, suggesting that increases in gesture rate would affect kinematic features to a lesser degree in vocal hyperfunction patients given the increased levels of muscle tension they typically employ to phonate. This hypothesis was tested experimentally in individuals with muscle tension dysphonia (MTD; $N=10$) and vocal nodules ($N=10$) relative to controls with healthy normal voice ($N=10$) who were examined with trans-nasal endoscopy during a simple vocal fold abductory-adductory task. Kinematic measures in MTD patients were less affected by increased gesture rate, consistent with the hypothesis that these individuals have elevated typical laryngeal muscle tension. Group comparisons of the difference between medium and fast gesture rates (Mann–Whitney, one-tailed) showed statistically significant differences between the control and MTD individuals on the two kinematic features examined ($p<0.05$). Results in nodules participants were mixed and are discussed independently. The findings support the potential use of vocal fold kinematics as an objective clinical assay of vocal hyperfunction.
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I. INTRODUCTION

A. Vocal hyperfunction

Vocal hyperfunction refers to "conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or 'imbalanced' muscular forces" (Hillman *et al.*, 1989), characterized by excessive laryngeal and paralaryngeal tension (Aronson, 1980; Morrison *et al.*, 1983; Koufman and Blalock, 1991; Dworkin *et al.*, 2000a; Roy, 2008) that commonly accompanies disordered voice production. Estimates of the prevalence of vocal hyperfunction indicate that the condition may account for 10%–40% of cases referred to multidisciplinary voice clinics (Roy, 2003), yet there is no commonly accepted objective measure for its detection and assessment. Vocal hyperfunction could be due to psychological and/or personality factors, misuse of muscles, learned adaptation following a short-term respiratory illness such as a cold, or compensation for underlying vocal disease (Aronson, 1980; Morrison *et al.*, 1983; Morrison *et al.*, 1986; Rammage *et al.*, 1987; Morrison and Rammage, 1993; Hsiung and Hsiao, 2004). However, claims of increased muscle tension are largely based on subjective accounts of the symptom complex. Although Redenbaugh and Reich (1989) showed increased surface electromyographic signals on the anterior neck in individuals with a "hyperfunctional voice disorder" relative to normal controls, no study has shown objective evidence for increased tension of intrinsic laryngeal muscles and structures.

Individuals who display disordered voice production that is associated solely with vocal hyperfunction in the absence of other laryngeal pathology are often diagnosed with muscle tension dysphonia (MTD; Hsiao *et al.*, 2001), according to a common definition of MTD. Benign lesions such as nodules and polyps on the vocal fold surface have been assumed to be related to hyperfunctional behavior for many years (Hillman *et al.*, 1990). For instance, in 1962, Godfrey Arnold wrote that "vocal nodules and polyps represent a local tissue reaction to the mental strain imposed by inappropriate emotional adjustment to the demands made by society" (Arnold, 1962). Vocal fold nodules are defined as benign lesions that occur bilaterally between the anterior and middle third of the vocal fold (Aronson, 1980; Marcotullio *et al.*, 2002). One clinic found that 92% of cases of vocal nodules were coincident with symptoms of vocal hyperfunction (Morrison *et al.*, 1983) indicating a strong relationship between the incidence of vocal hyperfunction and vocal nodules. However, due to the possible effects of compensation once there is organic pathology on the vocal folds (i.e., re-

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TABLE I. Abductory and adductory mean angular velocities.

Experimental							Modeling		
	Dailey *et al.*		CTRL		MTD				
Gesture rate	AB	AD	AB	AD	AB	AD	Stiffness	AB	AD
Medium	396 (237)	318 (113)	366 (109)	390 (83)	312 (70)	361 (84)	Medium	434	357
Fast	420 (173)	464 (185)	398 (83)	481 (99)	390 (90)	420 (102)	High	443	438
% increase	6.1 %	46 %	8.8 %	23 %	25 %	16 %	% increase	2.2 %	23 %

Velocities are presented in °/s. Experimental velocities are means with (standard deviations). CTRL=control participants ($N=10$) from the current work. MTD=MTD participants ($N=10$) from the current work. AB=abduction; AD=adduction.

active hyperfunction), no clear cause-and-effect relationship between vocal hyperfunction and nodules has been established. Therefore, while individuals diagnosed with MTD, by definition, have vocal hyperfunction, this may not be true in all cases of vocal nodules.

An obstacle to efficacious use of voice therapy for the treatment of vocal hyperfunction is the limited ability of the therapist to reliably ascertain if and when there are changes in the degree to which a patient is hyperfunctioning during voice production. Current measures for assessing the success of therapy approaches are patient report, voice-related quality of life assessment, voice quality, aerodynamic changes, and visual examination of the vocal folds using videoendoscopy. However, none of these measures provide unambiguous assessment of the underlying hyperfunctional status of the larynx during phonation, particularly in cases where the interpretation of measures is confounded by the presence of organic pathology such as vocal nodules (e.g., nodules can impact aerodynamic results by interfering with glottal closure). If differences in vocal fold kinematics between vocally normal individuals and individuals with vocal hyperfunction (with or without organic changes) exist, these could be used as an objective clinical assay of vocal hyperfunction to aid in the assessment and treatment of patients with hyperfunctionally related voice disorders. The primary goal of the current study was to explore two kinematic parameters as possible indicators of vocal hyperfunction.

B. Vocal fold kinematics

Using rigid (trans-oral) endoscopy and visual detection of vocal fold landmarks, Cooke *et al.* (1997) examined the linear kinematics of vocal fold adductory gestures during different types of glottal attack ("hard," "normal," and "breathy") in individuals with healthy normal voice. Overall articulator stiffness ("stiffness parameter") was approximated as the ratio of the maximum (linear) adduction velocity to the extent of the motion (Munhall and Ostry, 1983), and was found to be increased for hard voice onset relative to normal and breathy vocal onset. Cooke *et al.* (1997) suggested that this finding offers quantitative support for the idea that hard vocal onset involves greater laryngeal muscle tension than normal vocal onset.

More recently, Dailey and colleagues (2005) monitored the angle of the vocal folds (glottic angle, 2Ψ) as a function of time during a "sniff–/i/" task in women with healthy normal voice using flexible (trans-nasal) videoendoscopy. The sniff–/i/ is a rhythmic repetition of quick intake of air through the nose ("sniff") followed by production of the vowel /i/. They found that the mean angular velocities of the glottic angle during abduction and adduction were higher when participants were asked to perform the repetitions of the sniff–/i/ task more quickly; however, a greater influence of gesture rate was seen on adduction than on abduction. Specifically, an increase in gesture rate caused a 46% average increase in the mean angular velocity during adduction, but only a 6.1% average increase during abduction (see Table I for reproduction of the results of Dailey *et al.*, 2005). The authors speculate that this increased velocity during "quick adduction" is likely the result of the "greater control over recruitment of motor units in adduction." Although there are known differences in muscle fiber type distributions between abductory and adductory intrinsic laryngeal muscles (see Hoh, 2005 for review), it is possible that the effect of rate shown in Dailey *et al.* (2005) can be explained by the mechanical properties of the system.

Increases in speaking rate have been shown to result in kinematic changes of the lip, tongue, and jaw (Adams *et al.*, 1993; Hertrich and Ackermann, 2000). Interestingly, Hertrich and Ackermann (2000) found that the rate of steepness of the ratio of maximum velocity to motion amplitude (a variant of the stiffness parameter) of the jaw increases with increases in speaking rate. Thus, it is possible that increased gesture rate results in a global increase in laryngeal muscle stiffness. If so, a simple model of laryngeal adduction and abduction may capture these changes in velocity based on increases in stiffness. If this simple model results in a glottic angle similar to data gathered by Dailey *et al.* (2005), this would indicate applicability of such an approach to modeling the abduction and adduction of human vocal folds. As the overall stiffness of the muscles modeled is increased (modeling an increase in gesture rate of the task), there may be changes due purely to the mechanics causing the differences in adductory and abductory velocity changes noted by Dailey *et al.* (2005). Further, correspondence of the stiffness parameter measured by Cooke *et al.* (1997) with increases in the stiffnesses of modeled muscles will validate the relationship between muscle stiffness and that kinematic parameter. This is an important objective; although Kelso *et al.* (1985) present a convincing argument for this quantity as a "mass-normalized" or specific stiffness quantity, the parameter does not have dimensions physically appropriate for stiffness, but rather [1/time].

This work employs a simple model of vocal fold abduction and adduction in order to confirm the relationship of intrinsic laryngeal muscle stiffness with kinematic estimators of stiffness. Based on the results of the model, the work experimentally tested the hypothesis that increases in gesture rate of a repetitive abduction-adduction task would affect kinematic estimators of stiffness less in individuals with vocal hyperfunction than in individuals with healthy normal voice. This hypothesis is based on the supposition that individuals with vocal hyperfunction may have increased laryngeal muscle stiffness, which would mitigate stiffness increases caused by increases in gesture rate.

II. MODELING VOCAL FOLD ABDUCTION AND ADDUCTION

A. Model formulation

Although virtual trajectory models (also referred to as equilibrium point models) have been applied to speech systems by many researchers (Perrier *et al.*, 1996; Sanguineti *et al.*, 1998) with some success, previously described biomechanical models of the larynx have largely focused on the dynamics of vocal fold tissues to produce and manipulate the pitch of voicing (Story and Titze, 1995; Jiang *et al.*, 1998; Alipour *et al.*, 2000). Models of abduction and adduction (vocal fold posturing) have been created by several investigators (Farley, 1996; Hunter *et al.*, 2004; Titze and Hunter, 2007). These rigorous models of the larynx explore the role of various muscle activations on the control of abduction and adduction, but their simulations require the estimation of complex muscle activation functions, adding complexity and uncertainty. In the present work, a simple one-joint (unilateral) virtual trajectory model based around the arytenoid joint was designed to examine key aspects of the biomechanics of laryngeal abduction and adduction. A virtual trajectory model was chosen based on the simplicity of the approach: virtual trajectory models assume that higher levels of the central nervous system encode movement by specifying a series of postures (the virtual trajectory), and that the dynamic details of movement are a result of the peripheral neuro-muscular system. Thus, for a simple model of abductory and adductory gesturing, the virtual trajectory is the only input required. The model employed greatly simplifies the physiological system and ignores the translation of the arytenoids as well as their rotation in the sagittal plane, and also assumes symmetry of the control of the bilateral arytenoid joints, formally modeling only one side. Panel A of Fig. 1 shows a schematic of the key anatomy of this transverse section at the level of the vocal folds while panels B and C show the geometric model employed in the abductory and adductory states, respectively.

Model geometry was based on anatomical measurements of women, in order that simulation results could be compared with data from Dailey *et al.* (2005), who examined vocal fold motion in 21 women aged 19–27 years; thus, the modeling results are semi-specific to these smaller larynges. The model is based on a transverse section at the level of the glottis and includes three muscles: the thyroarytenoid (TA), lateral cricoarytenoid (LCA), and posterior cricoarytenoid

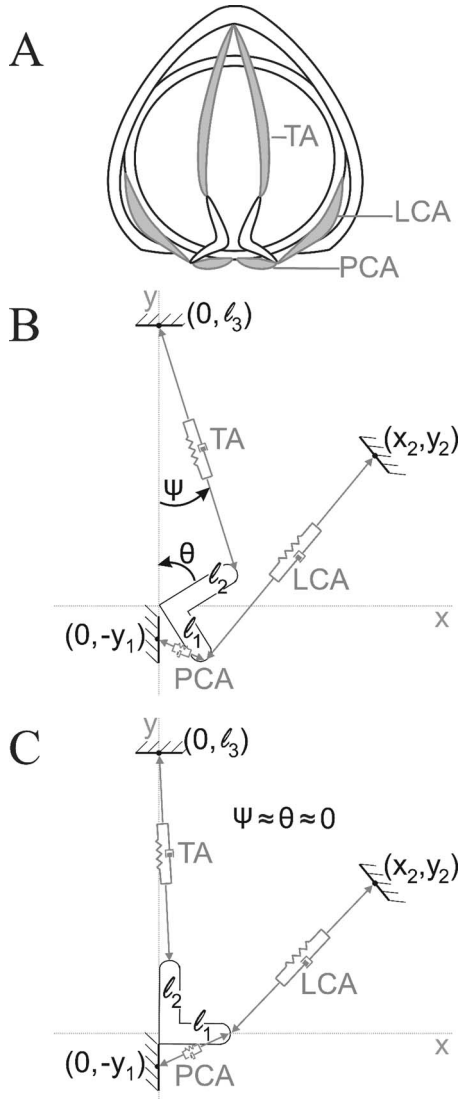

FIG. 1. Panel A—a schematic of a transverse section through the larynx at the level of the vocal folds. Panel B—model of vocal fold abduction. Double-sided arrows indicate muscles modeled as parallel springs and dampers. Panel C—model of vocal fold adduction.

(PCA). The muscles are modeled as simple springs, each represented by a parallel stiffness and damping, with the single input from the neural system defined as the virtual joint angle, θ_v, the angle between the muscular process of the arytenoid and the line perpendicular to the midline of the glottis. Damping values of 10% of the stiffness were used, mimicking the work of Flash (1987) and colleagues, who found that these values often produced results closest to those seen physiologically. The initial values used in the model and the basis for those choices are shown in Table II. These length and distance parameters were taken from 3D attachment points as reported by Kim *et al.* (2004). Assuming that the plane containing the thyroarytenoid is in the 2D plane modeled, these distances are consistent with projections onto the transverse plane. Approximations of spring constants were rough approximations based on animal data, so no effort was made to project stiffness onto the transverse plane. A sensitivity analysis was performed to explore alternative values. Although varying the model values did quan-

TABLE II. Initial values for constant parameters.

Parameter	Physical meaning	Value	Reference
ℓ_1	Length of arytenoid in the x-direction	0.014 m	Measurement "Ab" from Kim *et al.* (2004) (human women only)
ℓ_2	Length of arytenoid in the y-direction	0.0126 m	Measurement "ab" from Kim *et al.* (2004) (human women only)
ℓ_3	Distance from the origin to the vocal fold apex	0.0229 m	Measurement "jF" from Kim *et al.* (2004) (human women only)
M	Total mass of the arytenoid cartilage	0.5 g	Measured by James T. Heaton and James B. Kobler on cadaver larynges
d	Diameter of the "cylinders" of the arytenoid	0.00365 m	Estimated as 2/3 of the length "VW" in Kim *et al.* (2004) (human women only)
y_1	Distance from the origin to the insertion of the PCA	0.00348 m	Measurement "ON" from Kim *et al.* (2004) (human women only)
x_2	Distance from the insertion of the LCA to the y-axis	0.0144 m	Estimated as $\frac{1}{2}$ of the length of "hh" in Kim *et al.* (2004) (human women only)
y_2	Distance from the insertion of the LCA to the x-axis	0.0114 m	Estimated as $\frac{1}{2}$ of the length of "jF" in Kim *et al.* (2004) (human women only)
k_{PCA}	Stiffness of the PCA	119 N/m	Estimated from Young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
k_{LCA}	Stiffness of the LCA	76.5 N/m	Estimated from Young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
k_{TA}	Stiffness of the TA	640 N/m	Estimated from Young's modulus data and average length data from Hunter *et al.* (2004) and Alipour *et al.* (2005)
b_{PCA}	Viscosity of the PCA	11.9 N/m/s	10% of k_{PCA} estimate as in Flash (1987)
b_{LCA}	Viscosity of the LCA	7.65 N/m/s	10% of k_{LCA} estimate as in Flash (1987)
b_{TA}	Viscosity of the TA	64 N/m/s	10% of k_{TA} estimate as in Flash (1987)

titatively alter the modeling results, the overall effects were qualitatively similar. The muscle models used in this work are a simplified version of the Hill model, which is composed of a spring in series with the parallel combination of a spring, damper, and force generator (McMahon, 1984). This highly simplified muscle model was chosen because of its computational tractability and the uncertainty associated with choosing multiple muscle parameters. It should, however, be acknowledged that actual muscle physiology differs significantly from that of a simple spring.

In this formulation, the specific form of the system dynamics is as shown in Eqs. (1) and (2), where the net torque is equal to the product of the moment of inertia, I, and the angular acceleration, as well as to the sum of the torques, τ, imposed by the three muscles, designated with the subscripts *TA*, *LCA*, and *PCA*.

$$\tau_{net} = I\ddot{\theta}, \tag{1}$$

$$I\ddot{\theta} = \tau_{TA}(\theta, \dot{\theta}, \theta_v) + \tau_{LCA}(\theta, \dot{\theta}, \theta_v) + \tau_{PCA}(\theta, \dot{\theta}, \theta_v). \tag{2}$$

The torque imposed by each muscle is a function of its force and moment arm, MA, as is indicated in the example in Eq. (3), both of which are dependent upon the current angle of rotation θ. The force produced by each muscle (musc; e.g., LCA, PCA, and TA) is a function of current muscle length, ℓ, with respect to the virtual length, ℓ_v, a relationship, which is made explicit in Eq. (4).

$$\tau_{musc} = MA_{musc}(\theta)F_{musc}(\theta, \dot{\theta}, \theta_v), \tag{3}$$

$$F_{musc} = k_{musc}(\ell_v(\theta_v) - \ell(\theta)) - b_{musc}\dot{\ell}(\dot{\theta}). \tag{4}$$

The moment of inertia of the arytenoid cartilage was calculated based on its dimensions and mass (see Table II) as 20.8 g mm^2. Its geometry was approximated as three cylinders, meeting at the origin of rotation.

The exact form of the virtual trajectory employed is a key factor in the performance of a virtual trajectory model. The most straightforward approach might be to supply the system with a step function in which the virtual trajectory moves directly from the initial position to the final position ("final position control"). Although this approach may be appropriate for some ballistic movements of the upper limb (e.g., through a reduction in so-called submovements during fast movements), it has been shown to be inadequate for modeling slow and medium speed arm reaching movements (Bizzi *et al.*, 1984). In this model, use of final position control lead to movement simulations that were not physiologic. Thus, the virtual trajectory selected for use was a gradual shift from initial to final position. Specifically, the virtual trajectory was chosen based on an accepted criterion function minimizing the mean-square jerk and optimizing the "smoothness" of the resulting trajectory (Hogan, 1984). The criterion function is minimized as is shown in Eq. (5), where θ is the angle and D is the desired duration of the movement.

$$\min C = \min\left(\int_0^D (\dddot{\theta}_2)^2 dt\right). \tag{5}$$

Dynamic optimization was used to find the minimum mean-square jerk trajectory, which was found to be in the form of a fifth-order polynomial in time. The coefficients for the polynomial were solved for with the following boundary conditions [Eqs. (6)–(11)], where θ_{start} and θ_{finish} are the initial and final values of the desired θ, respectively, and D indicates the duration of the movement.

$$\theta_v(0) = \theta_{start}, \tag{6}$$

$$\theta_v(D) = \theta_{finish}, \tag{7}$$

$$\dot{\theta}_v(0) = 0, \tag{8}$$

$$\dot{\theta}_v(D) = 0, \tag{9}$$

$$\ddot{\theta}_v(0) = 0, \tag{10}$$

$$\ddot{\theta}_v(D) = 0. \tag{11}$$

These boundary conditions lead to an equation for a minimum mean-square jerk virtual trajectory, shown as

$$\theta_v(t) = \theta_{\text{start}} + (\theta_{\text{finish}} - \theta_{\text{start}})\left[10\left(\frac{t}{D}\right)^3 - 15\left(\frac{t}{D}\right)^4 + 6\left(\frac{t}{D}\right)^5\right]. \tag{12}$$

The virtual trajectory used as inputs to the model of laryngeal abduction and adduction were θ_v values from 0° to 35° (abduction) and 35° to 0° (adduction) and D=250 ms. These motions result in changes in the glottic angle ranging from 0° (adduction) to 60° (abduction), and were chosen to correspond with the results reported by Dailey *et al.* (2005).

The initial values of stiffness chosen (see Table II) were unable to recreate movements approximately within the 250 ms as reported in the literature (within 250+10 ms; Dailey *et al.*, 2005). In order to create realistic motions, a scaling factor was applied to all muscle stiffness parameters. The minimum scaling factor necessary to produce movements fast enough was a value of 3, a reasonable increase given the uncertainty in the estimates used, as well as the fact that stiffness-based estimates were made from static measurements. Because muscle stiffness increases with force (Hoffer and Andreassen, 1981), dynamic stiffnesses are likely to be higher than measured static values. For instance, modeling work by Flash and colleagues found physiologically relevant simulation results when values of dynamic stiffness were at least two times higher than the static values (Flash, 1987).

The described virtual trajectory model was used to simulate abduction and adduction at normal stiffness (scaling factor=3) as well as at high stiffness, in which the scaling factor was increased by a factor of 4 (scaling factor=12). The variation in stiffness was used to approximate the changes in muscle stiffness thought to be a result of increasing the gesture rate during the sniff–/i/ task. Thus, normal stiffness was used to correspond to the medium gesture rate and high stiffness to the fast gesture rate. Various increases in muscle stiffness were explored to approximate high stiffness. Small variations about the chosen scaling factor (10–14) did not show significant differences in performance; however, extremely high scaling factors (>15) resulted in minimal increases in the effects on the modeled movement, due to a ceiling effect. Specifically, at these very high stiffnesses, movements did not significantly differ from the given virtual trajectory, and further increases in stiffness caused imperceptible changes.

The resulting abductory and adductory movements created by the model at normal and "high" stiffness were used to calculate several kinematic parameters of interest. The mean angular velocity was calculated as in Dailey *et al.* (2005): the slope between the points of intersection of the movement with the values 20% and 80% of the maximum angle was used as the mean velocity for each gesture. The stiffness parameter was calculated as the ratio between the adductory maximum angular velocity and the maximum glottic angle.

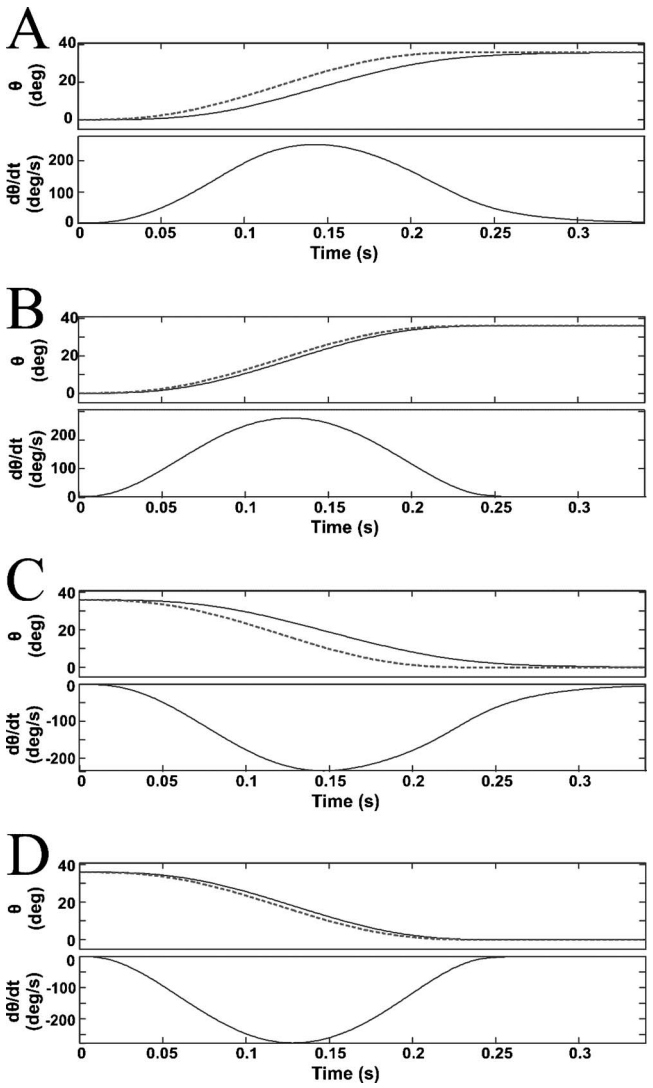

FIG. 2. Simulated θ position and velocity for (a) abduction at low stiffness, (b) abduction at high stiffness, (c) adduction at low stiffness, and (d) adduction at high stiffness. The gray dotted line indicates the virtual trajectory for each movement.

B. Modeling results and predictions

This simple model was able to simulate simple abductory and adductory vocal fold movements consistent with the data of Dailey *et al.* (2005). Figure 2 shows the model output θ, $d\theta/dt$, as well as the virtual trajectory, θ_v during abduction and adduction at high and normal stiffness. For both abduction and adduction, the 20%–80% mean angular velocity was found at both high and normal stiffness (see Table I). These mean values were consistent with those found during "regular" and "fast" gesture rates by Dailey and colleagues (2005); further, the percent increase between medium and high stiffness showed the same trend as that between regular and fast gesture rates seen by Dailey *et al.* (2005).

Although Dailey *et al.* (2005) speculated that the increased velocity during "quick adduction" was likely the result of the "greater control over recruitment of motor units in adduction," the purely mechanical model employed was able to reproduce the qualitative difference between abduction and adduction changes in velocity. However, the model results do not quantitatively agree with the published data (23% versus 46%, found in Table I). This could be due to a number of factors: over-simplification of the mechanical system, poor parameter choices, or true neural recruitment differences as postulated in Dailey *et al.* (2005). It is likely the differences are related to the overly simplified model and uncertainty in model parameters.

At the normal model stiffness values (scaling factor=3), the stiffness parameter was found to be 6.4, increasing to 7.8 at high model stiffness values (scaling factor=12). The increase found between normal and high model stiffness is roughly comparable to the average difference seen between normal and hard onset stiffness values measured using linear velocities previously (Cooke *et al.*, 1997).

These modeling results support the idea that the differential changes in the abductory and adductory velocities are a mechanical consequence of higher stiffness, and that this higher stiffness is consistent with increasing the gesture rate of abduction and adduction in normal speakers (Dailey *et al.*, 2005). Because the underlying characteristic of vocal hyperfunction is thought to be an increase in the tone of laryngeal musculature, these modeling results suggest that increases in gesture rate will affect the speed of adduction and the stiffness parameter far less in patients with vocal hyperfunction since their typical stiffness may already be elevated, a hypothesis that this work subsequently experimentally tested.

III. VOCAL FOLD KINEMATICS IN INDIVIDUALS WITH VOCAL HYPERFUNCTION

A. Experimental methods

1. Participants

Participants were ten adult female volunteers diagnosed with MTD prior to any therapeutic intervention (mean age =23.9 years, SD=5.4 years), ten adult female volunteers diagnosed with bilateral vocal nodules prior to any therapeutic intervention (mean age=25.8 years, SD=9.4 years), and ten females with healthy normal voice (mean age=24.8 years, SD=4.0 years). All participants were female due to the prevalence of vocal nodules in the female population compared to the male population (Morrison *et al.*, 1983; Morrison *et al.*, 1986; Titze, 1989; Pontes *et al.*, 2002). No participants with MTD or nodules had a history of any other speech or language disorder. The individuals with healthy normal voice reported with no complaints related to their voice, and had no abnormal pathology of the larynx as observed during flexible endoscopy with stroboscopy.

2. Procedures

Participants were examined using trans-nasal (flexible) endoscopy in order to visualize the vocal folds. Trans-nasal endoscopy was chosen rather than rigid endoscopy to provide the best view of normal laryngeal function, and to avoid the biomechanical effects of tongue protrusion created by rigid endoscopy. The endoscopy procedure involved the insertion of a flexible endoscope through the nose and past the soft palate to the pharynx above the level of the vocal folds, and in many cases also involved local anesthetization of the nasal passageways on one side with a light topical anesthetic (1% lidocaine) and a decongestant (0.5% neosynephrine). Endoscopic video recordings were made with a fiberscope containing a distal imaging chip (Pentax EPK-1000 or EPM-3500) digitized at 30 frames per second with a frame size of 480×360 pixels. During the exam, the participants produced 3–4 trials of the sniff–/i/ (between 5–6 separate gestures) at both "medium' and "fast" rates. Gesture rate was cued for participants using a metronome with visual (LED) cueing. The medium rate was performed at 72 gestures/minute, and the fast rate at 104 gestures/minute. These speeds were chosen based on the typical rate used in the MGH Voice Clinic currently (medium) and a reasonable (easily reproducible) excursion from this value for the fast rate. All participants were able to produce the gestures at both rates. Production of the sniff–/i/ was modeled by the first author for each participant prior to endoscopy and for some participants during endoscopy as well. Participants were encouraged to practice the sniff–/i/ several times prior to endoscopy.

3. Data analysis and statistics

Video data were analyzed using the custom MATLAB software employed in Dailey *et al.* (2005). The glottic angle was marked on each frame of the exam video recording and an asymmetric sigmoid[1] was fit to each set of angles corresponding to a single abduction or adduction movement, by minimizing the sum of square errors. To avoid introduction of errors due to possible vertical translation of the endoscope during recording, angular rather than linear kinematics were used since they are more robust to changes in vantage point. Marking of glottic angles was performed by two individuals. In order to assess inter- and intra-rater reliability, approximately 5% of samples were independently analyzed by both researchers, yielding inter-rater reliability as measured with Pearson's r of 0.990, and intra-rater reliabilities of 0.995 and 0.986 for each researcher. A sigmoidal fit was used rather than the cubic polynomial fit employed by Dailey *et al.* (2005), because it produced better fits to the kinematic data, which show two asymptotes in time. Figure 3 shows an illustration of a set of adduction angles (2Ψ) with their fit, along with the specific kinematic features extracted for use in parameter calculation. Using the sigmoidal fit, the 20%–80% angular velocity of each abduction and adduction was calculated as well as the stiffness parameter (the ratio of the adductory maximum angular adduction velocity to the extent of the motion). Tests on the null hypothesis that the difference between increases in adduction and abduction velocities and the changes in the stiffness parameter with increased gesture rate would be of the same level in the MTD and control groups were performed with non-parametric Wilcoxon–Mann–Whitney tests using MINITAB® Statistical Software (Minitab Inc., State College, PA).

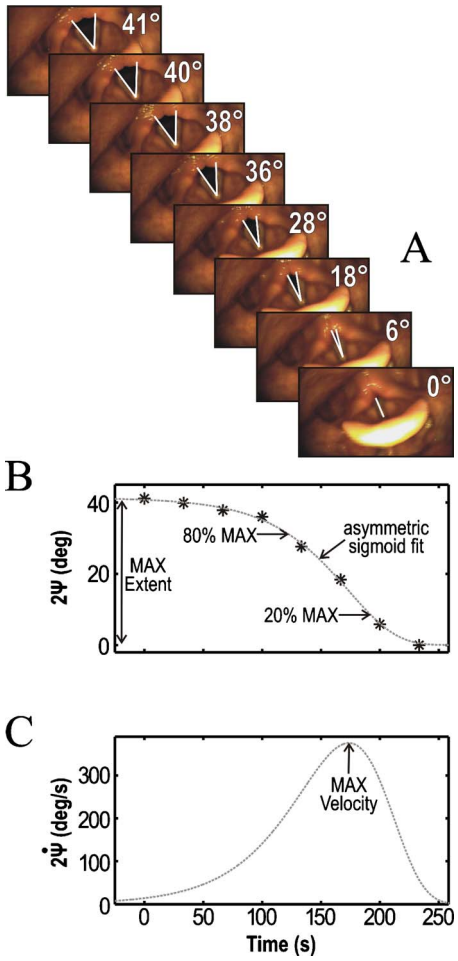

FIG. 3. (Color online) Overview of the experimental methodology. Panel A—an example of the images associated with one adductory movement with marked angles. Panel B—an example of the asymmetric sigmoid fit to the angles (2Ψ) marked in one adductory movement. The 20%, 80%, and maximum angle extents of the sigmoidal fit are marked. Panel C—an example of the angular velocity ($2d\Psi/dt$) of the asymmetric sigmoidal fit shown is in Panel B. The maximum velocity used for calculation of the stiffness parameter is noted.

B. Experimental results

Boxplots of the changes in the stiffness parameter with increased gesture rate (fast versus medium) are shown in panel A of Fig. 4 for individuals with MTD and controls. Also shown are the change values for individuals with vocal nodules. The changes in the stiffness parameter in these individuals presented a marked bimodal distribution. For this reason, these individuals were not analyzed as a single group, but were examined case-by-case. Due to the relatively small sample sizes in the groups and the non-normal distributions, differences between the medians of stiffness parameter changes between the MTD and control groups were examined with non-parametric testing. Median changes in the stiffness parameter in the MTD and control groups were 1.0 and 2.6, respectively. The two groups differed significantly, as evaluated by the Wilcoxon–Mann–Whitney test (one-tailed, $N_1=N_2=10$, $W=131.0$, $p=0.03$).

Differences in the increase in 20%–80% velocity in adductory versus abductory motion between the medium and fast gesture rate were examined similarly. Boxplots of the

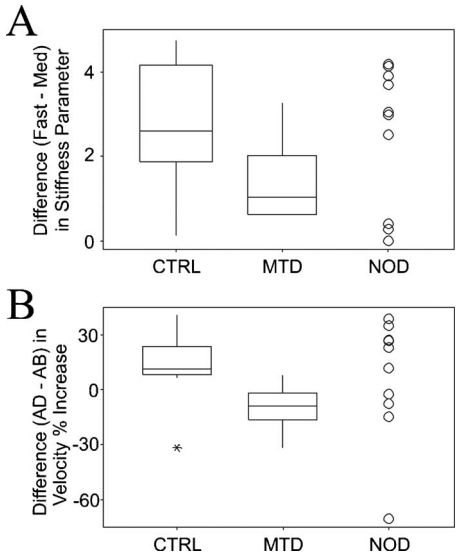

FIG. 4. Panel A—boxplots and an individual value plot of the differences in the stiffness parameter during adduction between the medium and fast gesture rate conditions. CTRL=individuals with healthy normal voice, MTD =individuals with MTD, NOD=individuals with vocal fold nodules. Panel B—boxplots and an individual value plot of the change in the difference in 20%–80% abduction and adduction velocities between medium and fast gesture rate.

differences between the adductory and abductory percent increase with increased gesture rate are shown in panel B of Fig. 4 for the MTD and control groups, with values shown for individuals with vocal nodules. Although less marked than with the stiffness parameter, the differences in velocity increase in individuals with nodules presented with a somewhat bimodal distribution, or at least a significant outlier. For this reason, individuals with vocal nodules were not analyzed as a single group, but were examined on a case-basis. Median differences between the adductory and abductory 20%–80% velocity percent increase with increased gesture rate in the MTD and control groups were −8.9% and 11.3%, respectively. The two groups differed significantly, as evaluated by the Wilcoxon–Mann–Whitney test (one-tailed, $N_1=N_2=10$, $W=144.0$, $p=0.002$). The average values of abductory and adductory 20%–80% velocity are tabulated in Table I.

Figure 5 shows a scatter plot of the differences in adductory and abductory 20%–80% velocity percent increase with faster gesture rate and the changes in the stiffness pa-

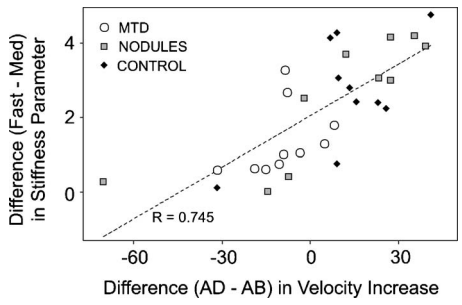

FIG. 5. Differences in the stiffness parameter between the medium and fast gesture rate conditions are plotted with respect to the differences between medium and fast gesture rate 20%–80% abduction and adduction velocities.

rameter with faster gesture rate. The two kinematic measures were found to be statistically significantly correlated, as assessed by Pearson's r ($r=0.745$, $p<0.001$).

IV. DISCUSSION

A. Average abduction and adduction velocities in individuals with healthy normal voice

The mean 20%–80% abduction and adduction velocities and resulting percent increase between the medium and fast conditions showed similar qualitative results to those of Dailey *et al.* (2005); see Table I. The percent increase in the adduction velocity was greater than the percent increase in abduction velocity when gesture rate was increased. However, when compared to the results of Dailey *et al.* (2005), the differences between abduction and adduction were less striking. Some differences in methodology could explain the differences between the current results and those of Dailey *et al.* (2005). The medium and fast rates used in this study (72 rpm and 104 rpm) were precisely controlled using a metronome, whereas the rates used by Dailey *et al.* (2005) were likely more varied. The average rates used in that study were not reported, but may also have been produced at significantly different rates. Another methodological difference that could affect these results is the choice of function used for data fitting. Data fitting is necessary to estimate the kinematic parameters due to the sparse sampling rate of the videoendoscopy, but basing kinematic measures on a function fit to data rather than the raw data is a possible source of estimation error. While Dailey *et al.* (2005) used a cubic polynomial to fit their data, a sigmoidal function was selected for this work. Cubic polynomials have a maximum of two extremas and a single inflection point, which is consistent with vocal fold abduction and adduction; they also have a maximum of three roots, and do not contain horizontal asymptotes, causing curvature that is inconsistent with the raw kinematic data. An asymmetric sigmoidal function was chosen to fit the data due to its characteristic two horizontal asymptotes and single inflection point. This difference in data fitting methodology could have contributed to the quantitative difference between the current work and that of Dailey *et al.* (2005).

B. Experimental confirmation of the modeling hypothesis in individuals with MTD

Compared with control participants, individuals with MTD showed dissimilar changes in both kinematic parameters with increases in gesture rate. These results are consistent with the modeling hypothesis, and suggest that these individuals have elevated laryngeal muscle stiffness, and that increases in gesture rate therefore have smaller effects on the laryngeal muscle stiffness. This work objectively corroborates claims that individuals with MTD have increased muscle tension, albeit indirectly.

The median changes seen experimentally in the stiffness parameter in the MTD and control groups were 1.0 and 2.6, which were roughly similar to the change seen in the stiffness parameter (1.4) between the medium and high stiffness conditions using the model. However, stiffness parameter changes in controls were somewhat larger than those predicted by the model.

Interestingly, the mean velocities in the individuals with MTD during both abduction and adduction were reduced relative to controls (see Table I). This is at apparent odds with the findings of Cooke *et al.* (1997), who found significantly higher maximum linear velocities during hard glottal attack (a behavior consistent with the features of vocal hyperfunction) than during normal glottal attack in individuals with healthy normal voice. However, it is misleading to compare the average velocities reported in the present study with maximum velocities such as those reported by Cooke *et al.* (1997). Further, the task conditions between these two studies were quite different. In Cooke *et al.* (1997), participants were asked to produce the vowel /i/ with various onset characteristics, but with no limitations in timing, whereas the restriction in this study to metronome timing for the production of the /i/ and sniffs created a different behavioral situation.

C. Vocal fold kinematics in individuals with vocal fold nodules

In individuals with vocal fold nodules, the difference between the percent increase in the 20%–80% abduction and adduction velocities, and the change in the stiffness parameter from medium to fast gesture rates showed mixed results. Some individuals presented with kinematic measures consistent with those hypothesized for individuals with vocal hyperfunction, while others displayed values consistent with those hypothesized for controls. Although it is possible that this effect is due to estimation error in the kinematic measures caused by the presence of the lesion, a more likely possibility is that some individuals with vocal fold nodules present with vocal hyperfunction, and that others do not. Although organic developments on the vocal fold surface have been assumed to be related to vocal hyperfunction (Hillman *et al.*, 1990), there is still much unknown about the underlying mechanisms of vocal hyperfunction and its role in developing organic disorders (Hillman *et al.*, 1990). In some individuals, nodule formation may be the result of other factors such as excessive vocal use, laryngopharyngeal reflux, etc., rather than inappropriate muscle tension. Further, once nodules are present it is not possible to determine whether any signs of hyperfunction are related to the etiology of the nodules, or if these are primarily a phonatory reaction (straining) to the presence of the nodules. Progress in this area suffers from two shortcomings. First, there is the lack of a gold standard in vocal hyperfunction assessment with which to compare potential objective measures, such as kinematic parameters. Second, previous investigations have not been able to delineate the actual role of hyperfunction in the etiology of voice disorders like vocal nodules. Future work should collect the kinematic measures used in the present study with multiple available modes of assessment to attempt to ascertain correspondence of kinematic measures with other possible indicators of vocal hyperfunction in the population of vocal fold nodules. In parallel with the identification of better measures, there is also a need for studies

that can correlate levels of vocal hyperfunction across time (longitudinal cross-sectional) with changes in vocal status (deterioration or improvement) to gain better insights into the role of laryngeal stiffness in voice disorders.

D. Use of vocal fold kinematics as an indicator of vocal hyperfunction

Both the difference between the percent increase in the 20%–80% abduction and adduction velocities and the change in the stiffness parameter from medium to fast gesture rates showed statistically significant differences between individuals with MTD and controls, but the velocity measure provided somewhat better discrimination between the two groups. The velocity measure and stiffness parameter changes were statistically significantly correlated, indicating that they may be measuring the same phenomenon. However, the modest Pearson's correlation ($r=0.745$) between the two suggests that there is likely noise in the measurement and estimation process.

Although the individuals with healthy normal voice showed the expected results as a group, some individuals presented with vocal fold kinematics that were inconsistent with the rest of the control group. This is not a completely unexpected result, given that past studies examining some indicators of vocal hyperfunction have indicated that individuals without voice disorder may display symptoms of vocal hyperfunction (Colton and Casper, 1996; Behrman *et al.*, 2003; Stager *et al.*, 2003). These studies have shown that some individuals without an expressed voice disorder showed visible compression during endoscopy (Behrman *et al.*, 2003; Stager *et al.*, 2003), as well as frequent hard glottal attack (Colton and Casper, 1996, p. 80), both of which are thought of as symptoms of vocal hyperfunction. It is possible that some of the individuals examined in this study who reported with healthy normal voice also engaged in vocal hyperfunction, without it leading to a voice disorder. Although this leads to less discrimination between groups and may make these kinematic measures less useful for diagnosis purposes, it does not necessarily rule out their use for follow-up and assessment of individuals with vocal hyperfunction throughout rehabilitation. A further possibility is that these measures could be used preventatively, to identify individuals engaging in vocal hyperfunction so that they may be able to avoid developing vocal pathology.

Another factor to consider with respect to the use of vocal fold kinematic measures in the assessment of vocal hyperfunction is the measurement technique of nasal endoscopy. This technique is usually well-tolerated, but can be a source of stress for many individuals. The stress of undergoing this procedure could temporarily induce vocal hyperfunction in individuals who otherwise have healthy normal voices, making it more difficult to differentiate between typically hyperfunctional individuals and those specifically reacting to the procedure. Alternatively, the stress of endoscopy could differentially elicit hyperfunctional behaviors in predisposed individuals, effectively exacerbating their symptoms. Several studies have suggested a role for hyperreactivity to emotional stressors and anxiety in the etiology of MTD (Goldman *et al.*, 1996; Roy *et al.*, 2000; Demmink-Geertman and Dejonckere, 2002, 2008), especially in women. It is possible that inducing stress in individuals with MTD may intensify symptoms of vocal hyperfunction, without causing similar effects in healthy controls. Well-controlled studies of individuals with MTD and healthy normal voice are needed to validate this hypothesis.

As described in the methods, local anesthetization of the nasal passageways on one side with 1% lidocaine was performed on most participants, and some portion of the anesthetic dripped back into the laryngopharyngeal area. The effect of anesthesia on vocal fold kinematics is unclear. Gould and Okamura (1974) explored the effects of topical anesthetic on aerodynamic measures and selected electromyographic patterns during voice production. They found that none of the measures studied were affected by anesthesia during the production of voice at comfortable pitch and intensity, but that in most combinations of increased or decreased pitch and intensity, the application of anesthesia resulted in increased glottal resistance and subglottal pressure. More recently, Rubin *et al.* (2009) found that application of topical anesthetic does not perceptibly affect vocal fold motion. However, Dworkin *et al.* (2000b) used a 3 cc injection of 4% lidocaine through the cricothyroid membrane to treat three therapy-resistant cases of MTD, finding that the application caused immediate and long-standing resolution of symptoms. Although the dosage used there was quite different than in the present work, it is possible that the use of the lidocaine temporarily reduced symptoms, leading to smaller differences between the MTD and control groups. This possible effect is difficult to study due to the large percentage of individuals unwilling to tolerate nasal endoscopy without use of anesthesia.

E. Summary and future work

A simple, one degree of freedom, virtual trajectory model of vocal fold kinematics was used to confirm that vocal fold movement during a repetitive abduction/adduction gesture suggested excessive muscle tension in individuals with MTD relative to healthy normal controls. The model simulations support the hypothesis that increases in the modeled muscle stiffnesses were correlated with changes in a kinematic parameter known to increase with gesture rate and changes in a kinematic parameter known to increase with hard glottal attack. These modeling results suggested that increases in gesture rate would affect kinematic features to a smaller degree in patients with vocal hyperfunction because of the presence in these individuals of increased stiffness of intrinsic laryngeal muscles and structures during typical phonation. This hypothesis was tested experimentally, finding that kinematic measures in individuals with MTD showed less change with increased gesture rate, consistent with the hypothesis that these individuals have elevated laryngeal muscle tension during phonation. However, kinematic results in individuals with vocal fold nodules were mixed. The findings support the potential use of vocal fold kinematic data as an objective clinical assay of vocal hyperfunction.

Suggested future work includes correlation of vocal fold

kinematic data in individuals with vocal hyperfunction with other modes of vocal hyperfunction assessment, as well as exploring the role of stress inducement in exacerbating the known symptoms of vocal hyperfunction. The simple model utilized was able to simulate simple abductory and adductory vocal fold movements. However, increasing stiffness of the model did not *quantitatively* correspond to the difference in abduction and adduction seen at faster repetition rates. As speculated, this could be due to over-simplification of the mechanical system, poor parameter choices, or true neural recruitment differences as postulated in Dailey *et al.* (2005). A first step to possible improvements to this model would be to reduce the level of simplification imposed on the mechanical system. This could be accomplished by incorporating (1) translation of the arytenoids, (2) rotation of the arytenoids in the sagittal plane, and (3) the effects of the cricothyroid and interarytenoid muscles. Further development of this model may allow for more accurate modeling of vocal fold abduction and adduction.

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[1] $\theta_{\text{fit}}(t)=a/(1+\exp\{(-(t-c\,\log\{2^{1/d}-1\}-b)/c)\})^d$, in which parameters a, b, and c were fit to the data.

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