A viscoelastic laryngeal muscle model with active components

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Accurate definitions of both passive and active tissue characteristics are important to laryngeal muscle modeling. This report tested the efficacy of a muscle model which added active stress components to an accurate definition of passive properties. Using the previously developed three-network Ogden model to simulate passive stress, a Hill-based contractile element stress equation was utilized for active stress calculations. Model input parameters were selected based on literature data for the canine cricothyroid muscle, and simulations were performed in order to compare the model behavior to published results for the same muscle. The model results showed good agreement with muscle behavior, including appropriate tetanus response and contraction time for isometric conditions, as well as accurate stress predictions in response to dynamic strain with activation. © 2014 Acoustical Society of America. [<http://dx.doi.org/10.1121/1.4866173>]

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I. INTRODUCTION

Muscle mechanics include both passive and active properties. The passive mechanical force in muscle tissue is produced by the resistance of elastin and collagen fibers in the soft tissue as the muscle is stretched. It is dependent solely on muscle strain (stretch) and increases nonlinearly with increasing strain. Passive tissue also exhibits viscoelastic effects such as rate-dependence, hysteresis, stress relaxation, and creep. Active muscle force, on the other hand, is produced by engagement of the sarcomeres in response to neural stimulation of the muscle. The magnitude of the active force depends on the current length of the muscle, the contraction velocity, and the activation level or amount of neural stimulation provided to the muscle. As with all biological systems, both active and passive muscle properties vary for each individual muscle and specimen.

Both active and passive tissue properties are important to laryngeal function and voice production. Vocal fold vibratory motion and fundamental frequency are governed largely by passive stiffness of the vocal fold lamina propria (Titze, 1988; Zhang *et al.*, 2006) which commonly experiences strains of more than 30%, as well as cyclic strain modulations (such as in quick speech inflection or vocal embellishments like vibrato) where the viscoelastic effect may play a role. Activation of the thyroarytenoid and cricothyroid (CT) muscles regulates vocal fold length and shape, factors which in turn affect phonation pitch and threshold pressure (Titze *et al.*, 1988). Other vocal fold posturing movements of adduction and abduction are vital to healthy respiration and

contribute to ease of phonation and voice quality (Murry *et al.*, 1998). These motions involve participation from all five (paired) intrinsic laryngeal muscles (Titze and Hunter, 2007), which undergo varying levels of stimulation, large strains, and low-frequency (<10 Hz), non-periodic, cyclic behavior where time- and rate-dependent effects have an influence.

Since passive elastic and viscoelastic properties as well as active tissue characterizations influence vocal fold dynamics, it is crucial to provide accurate material definitions in dynamic models of the vocal folds. Much effort has been placed into the development of accurate passive tissue models for the vocal folds. Polynomial and exponential equations have been employed to capture the nonlinear stress-strain properties of vocal fold tissue (Alipour-Haghighi and Titze, 1991; Min *et al.*, 1995). Other viscoelastic models based on the standard linear solid model have been developed to describe the passive behavior of the vocal fold lamina propria (Chan and Titze, 2000; Chan and Siegmund, 2004). Zhang *et al.* (2006) created a two-network passive model, one branch describing the nonlinear elastic equilibrium stress based on a hyperelastic Ogden model, and the second branch accounting for short-term viscoelastic effects of rate-dependence and hysteresis. Building upon this model, a third network was added to model long-term viscoelasticity, namely stress relaxation and creep (Zhang *et al.*, 2009). This three-network model was able to simulate reasonably well all of the passive properties of vocal fold tissue (nonlinear elasticity and both short- and long-term viscoelasticity).

Laryngeal muscles, with active properties as well as passive, have also been extensively modeled. Most of the models in the literature (e.g., Butenweg and Kob, 2004; Farley, 1996; Titze and Story, 2002; Gommel *et al.*, 2007; Yin and Zhang, 2013) have all followed a similar approach. They can be described schematically like a basic Hill muscle

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model, which consists of a contractile element and a nonlinear spring element in parallel. The active muscle stress, generated by the contractile element, is based on non-dimensional factors that represent the dependency of the stress on muscle length, contraction velocity, and activation level. The passive stress, produced by the spring element as the muscle is stretched, is calculated with exponential equations that capture the nonlinear passive stress-strain response of the muscle. One recent study used a Mooney-Rivlin model instead (Yin and Zhang, 2013). In most of the cases, active and passive forces are calculated separately. After they are calculated, they are summed to yield the total stress in the system (stresses add because the elements are in parallel). The exception to this general Hill-based approach is the modified Kelvin model used by Hunter *et al.* (2004) and Hunter and Titze (2007), where active and passive stresses relate to each other through coupled differential equations.

Many of these models have shown reasonable results as far as active muscle behavior. An area lacking in the muscle models, however, has been the implementation of accurate passive properties, particularly a complete definition of the viscoelastic effects. As mentioned above, the majority of these models have used exponential relationships for passive properties, which match the nonlinear elastic stress-strain response of the muscle during extension but do not capture the hysteretic response during cyclic loading, rate-dependence, or time-dependent tissue properties. Few studies have used more complex models that include viscoelasticity (Hunter *et al.*, 2004; Hunter and Titze, 2007), with these constitutive models being limited in their ability to simulate all the viscoelastic effects. No active model has employed the three-network Ogden model, which has demonstrated the ability to model the appropriate elastic and viscous properties of laryngeal tissue, to simulate active and passive stresses of laryngeal muscle.

Therefore, the purpose of this study was to test the efficacy of a muscle model which combines active properties with a complete passive model. Using the previously developed three-network Ogden model to model passive stress, a Hill-based contractile element stress equation was utilized for active stress calculations. Methods for development of the model are described, and results showing the model

response in comparison to published data for laryngeal muscles are presented and discussed. The scope of the present study was not to model any particular voice phenomenon at this time, but rather to fuse active properties with the accurate three-network Ogden passive model and determine whether the combination could reasonably simulate general laryngeal muscle mechanics. Once accomplished, the model, with its additional capability, could then be used to study more complex voice questions. With this in mind, potential future applications of the model were also discussed.

II. METHODS

First described is the formulation of the muscle model, including active and passive properties. Next, the parameters of the presented model are discussed. Finally, the simulations of the model are examined.

A. Muscle model formulation

Figure 1 shows a schematic of the mathematical muscle model, which is derived from a basic Hill muscle model and consists of active (top) and passive (bottom) branches. The active branch consists of a single contractile element which generates stress due to muscle activation. The passive branch is typically displayed as a spring element and uses an exponential relationship to calculate passive stress based on muscle strain (stretch). However, here it has been replaced by a schematic representing an Ogden material-based constitutive model for estimating passive properties (described briefly below). The total stress generated in the muscle is the sum of the active stress (σ_{CE}) and passive stress (σ_{PE}):

$$\sigma = \sigma_{CE} + \sigma_{PE}. \quad (1)$$

Mechanically, the stresses add because the elements are in parallel. In muscle modeling, this is also a generally accepted approach when the model is not mechanically based. Many studies have utilized this technique when defining active and passive properties with separate material definitions (e.g., Hedenstierna *et al.*, 2008). The present model follows this precedent in combining a Hill-based contractile

FIG. 1. Schematic of muscle model. Active branch (top) consists of Hill type contractile element. Passive branch (bottom) consists of three-network Ogden model. Total stress is summation of active and passive stresses.

element for the active component with a complex Ogden-based material model for the passive part.

1. Passive properties

The passive model was developed previously and was not altered as a part of this study but is presented here briefly for completeness. Additional details can be found elsewhere (Zhang *et al.*, 2009; Chan *et al.*, 2009). It is the same three-network constitutive model presented by Zhang *et al.* (2009), shown to sufficiently model the complex mechanical behavior of vocal fold passive tissues. The model consists of a time-independent equilibrium network based on the hyperelastic Ogden material model to give nonlinear elastic properties (Network A) and two viscoplastic networks to approximate time-dependent viscoelastic tissue effects. Network B captures the hysteretic response in cyclic loading, while Network C represents effects of stress relaxation and creep. The nominal stress in Network A is calculated as

$$\sigma_A = \frac{2\mu_A}{\alpha} (\lambda_A^{\alpha-1} - \lambda_A^{-(\alpha/2)-1}). \quad (2)$$

Here μ_A is the initial shear modulus and α is a dimensionless constant which describes the degree of nonlinearity of the elastic response. The elastic stretch λ_A is equal to the applied stretch λ_u ($\lambda_A = \lambda_u$), where stretch is related to engineering strain ($\lambda = 1 + \epsilon$). The same also applies for the stretch rate in network A ($\dot{\lambda}_A = \dot{\lambda}_u$).

In Networks B and C the elastic stretch rate can be expressed as follows:

$$\begin{aligned} \dot{\lambda}_i^e &= \dot{\lambda}_u \frac{\lambda_i^e}{\lambda_u} - \frac{2}{3} \lambda_i^e \cdot Z \cdot \text{sgn}(\lambda_i^e - 1) \\ &\times \left\{ \sqrt{\frac{1}{3} \left[\left(\frac{\lambda_u}{\lambda_i^e} \right)^2 + \frac{2\lambda_i^e}{\lambda_u} \right]} - 1 + \delta \right\}^{c_i} \\ &\times \left| \frac{2\mu_i}{\alpha} \left[(\lambda_i^e)^\alpha - (\lambda_i^e)^{-(\alpha/2)-1} \right] \right|^{m_i} \quad (i = B, C). \end{aligned} \quad (3)$$

It is dependent on the applied stretch λ_u and stretch rate $\dot{\lambda}_u$. It is also dependent on the stress exponent m_i , which characterizes the dependence of the inelastic deformation on the stress level in the network; the stretch exponent c_i ($-1 < c_i < 0$), which characterizes the dependence of the rate of inelastic deformation on the current magnitude of inelastic deformation; and the viscosity scaling constant Z_i , which defines the absolute magnitude of the inelastic deformation. In addition, the initial shear modulus for each respective network (μ_i) and α (same as for

Network A) are required. The value δ ($\delta < 0.001$) is a small positive number added to avoid singularities in the inelastic stretch rate when the inelastic stretch is close to unity.

Equation (3) is solved numerically to find the elastic stretches for Networks B and C. The total passive stress is then obtained by summing the stresses in all three networks [Eq. (4)].

$$\begin{aligned} \sigma_{PE} &= \sigma_A + \sigma_B + \sigma_C \\ &= \frac{2\mu_A}{\alpha} \left[\lambda_u^{\alpha-1} - \lambda_u^{-(\alpha/2)-1} \right] \\ &\quad + \frac{2\mu_B}{\alpha} \left[(\lambda_B^e)^{\alpha-1} - (\lambda_B^e)^{-(\alpha/2)-1} \right] \\ &\quad + \frac{2\mu_C}{\alpha} \left[(\lambda_C^e)^{\alpha-1} - (\lambda_C^e)^{-(\alpha/2)-1} \right]. \end{aligned} \quad (4)$$

The Ogden model input parameter values used for this study, found by optimization to simulate properties of the vocal fold cover (Jampala *et al.*, 2012), are listed in Table I.

2. Active properties

As stated above, the active branch consists solely of a Hill contractile element. In finite element implementations of the Hill model, the stress due to an active muscle force can be described by the following equation:

$$\sigma_{CE} = a(t) \cdot \sigma_{\max} \cdot \sigma_{TL}^* \cdot \sigma_{TV}^*. \quad (5)$$

The contractile element stress magnitude is based on $\sigma_{\max}$, the maximum active muscle stress under isometric conditions and full nerve stimulation, is determined experimentally. This stress is scaled by an activation level, $a(t)$, which represents the amount of stimulation provided to the muscles by the nerves and ranges from 0 (no activation) to 1 (full or maximum activation). Further scaling is provided by the terms σ_{TL}^* and σ_{TV}^* , which are the normalized stress as a function of muscle stretch and normalized stretch rate, respectively.

Normalized stress functions are defined by mathematical expressions and matched to experimental data. Normalized stress as a function of stretch represents the dependency of force generated by the muscle on muscle length. A typical curve for the force-length relationship (e.g., Bahler *et al.*, 1967; Winters and Stark, 1985) is shown in Fig. 2 (top). An optimum length exists where isometric muscle stress is maximal and decreases as the muscle becomes shorter or longer. Here normalized stress is calculated as a function of stretch. Further, σ_{TL}^* is defined by a Gaussian-type equation after Winters and Stark (1985),

TABLE I. Three-network Ogden material model parameters.

Network A		Network B				Network C			
α	μ_A	Z_B	c_B	m_B	μ_B	Z_C	c_C	m_C	μ_C
17.6746	4.743	0.022	-0.80902	1.2527	78.3221	0.005	-0.005	1.1	0.74

FIG. 2. Typical curves for normalized force-length (top) and force-velocity (bottom) relationships of muscle.

$$\sigma_{TL}^* = \exp \left[- \left(\frac{\lambda - \lambda_{opt}}{s_f} \right)^2 \right] + m\lambda, \quad (6)$$

where stress is normalized by σ_{max} , stretch λ is the current muscle length L normalized by the initial length L_0 ($\lambda = L/L_0$), and λ_{opt} is the optimal stretch (at which peak stress occurs). In addition, the shape of the Gaussian curve is governed by the shape factor, s_f . The parameter m is a slope parameter, which is sometimes needed to capture asymmetry in the curve.

Normalized stress as a function of normalized stretch rate accounts for the dependency of muscle force on shortening/lengthening velocity. Figure 2 (bottom) gives a typical F - V curve (Bahler *et al.*, 1967; Winters and Stark, 1985). Force decreases hyperbolically as shortening velocity increases, and increases hyperbolically but not to the same extent as the muscle lengthening velocity increases. When velocity is zero, the force equals the isometric force as a function of length. In the present model, a normalized stress is used in place of force (yielding a value between 0 and 1), and a normalized strain rate is used instead of velocity, ranging between -1 and 1 . The hyperbolic equations used to

estimate the curves for muscle contraction and lengthening, respectively, are as follows:

$$\sigma_{TV}^* = \begin{cases} 1 - \left(\frac{1 + s_p}{-s_p + \dot{\epsilon}^*} \right) \dot{\epsilon}^* & \text{if } \dot{\epsilon}^* \leq 0 \\ 1 + \left(\frac{0.4(1 + 0.5s_p)}{0.5s_p + \dot{\epsilon}^*} \right) \dot{\epsilon}^* & \text{if } \dot{\epsilon}^* > 0. \end{cases} \quad (7)$$

Here stress is normalized by σ_{max} and the muscle strain rate $\dot{\epsilon}$ is normalized by the maximum muscle strain rate $\dot{\epsilon}_{max}$ ($\dot{\epsilon}^* = \dot{\epsilon}/\dot{\epsilon}_{max}$), which is again determined by experiment. The parameter s_p controls the shape of the curve, which varies from muscle to muscle.

3. Active stress response

Physiologically, when a muscle is activated a series of nerve impulses are sent to the muscle at a given frequency. With a high enough frequency, the nerve signal is more or less continuous, resulting in constant muscle activation, termed tetanus. For a muscle to go from no activation to a given level of activation, the response is not immediate. There is a delay in the muscle response and then the force gradually builds in the muscle over a tetanic contraction time before evening out at its full amplitude (e.g., Sherwood, 2007).

Consideration was made for the active stress to simulate a realistic response to active stimulation. Activation level, $a(t)$, was put into the contractile element stress calculation as a step input (for an immediate change in activation level and, consequently, contractile element stress). In order to simulate the appropriate gradual response, two coupled differential equations representing two first-order systems, each with a different time constant, were solved with the contractile element stress as the input [Eq. (8)].

$$\begin{aligned} t_1 \dot{\sigma}_1 + \sigma_1 &= \sigma_{CE}, \\ t_2 \dot{\sigma}_2 + \sigma_2 &= \sigma_1. \end{aligned} \quad (8)$$

The output stresses, σ_1 and σ_2 , are an intermediate first-order response and final second-order response, respectively, to the contractile stress input, and t_1 and t_2 are the time constants governing the response for each system. These equations were adapted from the system equations used by Titze (2006) to describe the first- and second-order stress response of muscle as predicted by the modified Kelvin model.

B. Model parameter selection

The goal in selecting input parameters was to closely match published data for laryngeal muscles. Experimental data for active properties of laryngeal muscles are limited, with the majority of the data that exists having been taken from canine CA muscle. Since this is the only muscle for which all of the necessary parameters could be derived, the parameters chosen for this study were taken from published properties of canine CA muscle.

The inputs to the model include σ_{max} ; σ_{TL}^* , governed by λ_{opt} , s_f , and m ; σ_{TV}^* , defined by s_p with velocity normalized

by $\dot{\epsilon}_{\max}$; and the $a(t)$ response, using t_1 and t_2 . The studies from which these inputs were derived and the parameter values chosen are described below.

1. Maximum active stress: $\sigma_{\max}$

Alipour-Haghighi *et al.* (1991) showed a force-elongation curve with the peak stress at approximately 134 kPa (120 gf). On the other hand, Titze (2006) gave a maximum active stress value of 87 kPa for canine CA muscle. Alipour and Titze (1999) showed data for several samples of canine CA muscle with a range of roughly 80 to 110 kPa for $\sigma_{\max}$.

For the present model, two maximum stress values are used. A value of 134 kPa, from Alipour-Haghighi *et al.* (1991) is used in our general active model behavior and sinusoidal strain simulations (described later), while 155 kPa is chosen for the remaining simulations to obtain an optimal match to the literature data. While this second value is slightly above those reported in the literature, it is reasonable to assume that it is not unrealistic considering the amount of variation present in soft tissues.

2. Maximum strain rate: $\dot{\epsilon}_{\max}$

A maximum strain rate of 2.2 l_0/s was reported by Titze (2006) for the muscle. Alipour and Titze (1999) showed a range from 1.2 to 3.5 l_0/s , with curve fits yielding roughly 2.5 l_0/s for the maximum strain rate. We chose $\dot{\epsilon}_{\max}$ to be 2.25 l_0/s to be a representative value.

3. Normalized stress: σ_{TL}^*

A force-elongation curve (Fig. 3) for canine CT muscle was given by Alipour-Haghighi *et al.* (1991). From this curve, we estimated the optimum stretch to be approximately 1.3, based on a 17 to 20 mm initial muscle length with 5.5 mm stretch. Titze (2006) found the value to be 1.2, and data from Alipour and Titze (1999) suggested a range from 1.1 to 1.4. A shape factor, s_f , of 0.3 matched the curve from Alipour-Haghighi *et al.*, while a value around 0.5 was needed to approximate the data in Alipour and Titze. We

FIG. 3. Normalized stress as a function of stretch for the present model and for published data of canine CT muscle from Alipour-Haghighi *et al.* (1991).

used the same shape factor as Alipour-Haghighi *et al.* of 0.3 and chose λ_{opt} to be 1.2 for all of our simulations except the general active model behavior and sinusoidal strain simulations (described later), in which we used a value of 1.5 to better match our results to the published simulation of sinusoidal strain. Similar to the maximum stress parameter, while this optimum stretch was just out of the published range, we could assume that it was realistic given the amount of variation in soft tissue properties.

4. Normalized stress rate: σ_{TV}^*

Alipour and Titze (1999) approximates the force-velocity relationship with a hyperbolic function and found curve fits for the pars recta (PR) and pars oblique (PO) portions of the CT. Figure 4 shows these curves normalized along with the curve used for our model. Using our definition for force versus velocity, a shape factor of 0.2 gives the best combination of overall fit to Alipour and Titze's data and simulation results in comparison to published data.

5. Activation level: $a(t)$

A similar set of coupled first-order systems to ours was used by Titze (2006) for the active branch of the Kelvin model. The time constants for these systems were $t_i = 0.02 + 0.1 \epsilon$ s and $t_s = 0.08$ s, corresponding to an internal contractile element activation time and series spring element time response, respectively. Using $t_1 = 0.02$ and $t_2 = 0.08$ in our model brought about a tetanic contraction time of 77 ms to 50% of the maximum (steady-state) stress and 207 ms to 90% maximum stress. These contraction times were chosen because they are similar to those reported previously: 84 ms—50%, 194 ms—90% CT-PR, 109 ms—50%, 253 ms—90% CT-PO (Alipour and Titze, 1999); and <100 ms—50%, >200 ms—90% (Alipour-Haghighi *et al.*, 1991).

C. Simulations

The mathematical model was coded in MATLAB, from which simulations of muscle activity were performed to

FIG. 4. Normalized stress as a function of normalized muscle strain rate for the present model and for canine CT muscle from Alipour and Titze (1999).

observe model behavior and compare the results to published data of laryngeal muscle behavior. For each simulation, the MATLAB script required the input of a time step and total simulation time, a prescribed strain at each time step, and the desired activation level at each time step. A time step size of 0.001 s was used for all simulations, while the other inputs varied based on the particular simulation to be run.

Four types of simulations were carried out, each briefly described below. The first was done to observe general model behavior, and the remaining ones replicated experiments from the literature on actual laryngeal muscle for comparison purposes.

1. General active model behavior simulation

To observe general behavior of the active model, a simulation was performed in which the model was subjected to various step and gradual changes in activation level over a period of 3.0 s, with a constant zero strain (i.e., no change in length). An isometric simulation was chosen in order to exclude passive stresses and because the active model response could more easily be interpreted without dynamic changes.

2. Tetanic response simulation

Two simulations of the tetanic response of the model were run, similar to tests by Alipour-Haghighi *et al.* (1991) using canine CA muscle. In one, full activation was applied from the beginning of the simulation and held throughout the simulation time of 1.5 s. In the other, full activation was applied at 0.06 s and turned off at 2.0 s to simulate both tetanus and the muscle deactivation response. Additionally, the deactivation response from this second simulation (i.e., only data from 2.0 s on) was normalized and compared to another experiment by Alipour-Haghighi *et al.* (1991), which considered only muscle deactivation from a full activation state. For these simulations, a strain value of 0.135 was applied at the start and held constant throughout the simulation. This simulated a slight pre-strain that was applied in the literature simulations to set the muscle to an *in situ* length. The pre-strain created a starting force value of approximately 4 gf in the model, compared to the 1 to 5 gf reported by Alipour-Haghighi *et al.* These simulation responses were compared to the tests from Alipour-Haghighi *et al.* (1991).

3. Twitch response simulation

A test of the model twitch response was performed next. Similar to an experimental twitch test done by Perlman and Alipour-Haghighi (1988) on laryngeal muscle, the muscle model was activated fully for 2 ms and then deactivated. The response was compared to data from Perlman and Alipour (1988), and successive twitch simulations were performed, gradually increasing the activation duration to obtain a better match to the published data. The other simulation that will be discussed below had an activation duration of 20 ms. As with the tetanus simulations, a pre-strain of 0.135 was applied and held throughout the twitch simulations. This gave a starting force value of about 4 gf, which matched the literature twitch data.

4. Sinusoidal strain simulation

The muscle model was subjected to a sinusoidal stretch and release, ranging from 0% to 30% strain at a frequency of 1 Hz, the same as that performed by Alipour-Haghighi *et al.* (1991) with canine CT muscle. Full activation was applied after 1.75 s so that the first part of the simulation showed only passive stresses with the latter portion displaying the combined active and passive stresses. Muscle and model responses were compared. In contrast to the other simulations performed for this study, this simulation demonstrated the dynamic response of both the active and passive components of the model (Figs 3 and 4).

III. RESULTS

The following describes the results of the simulations performed with the present model in MATLAB per Sec. IIC.

A. General active model behavior

In order to observe general behavior of the active model, the model was subjected to various levels and types of change in activation level over time without changing length (i.e., isometric contraction). Figure 5 shows the activation input and the resulting model active stress. In addition to the final active stress, the intermediate active stress was included, which occurred after the contractile element stress was solved in the first of the two first-order differential equations for active response.

Overall, the model behaved as expected for a linear second-order system. As the activation level was initiated and changed, the model exhibited a transient response before reaching the steady state value prescribed by the activation level. The transient output response consisted of a time delay and smooth, gradual changes in stress in comparison to the step changes and sharp transitions of the input. The intermediate stress showed a first-order response based on the time constant for the first differential equation (system). After passing through the second system, the active stress curve followed a second-order transient response resembling that seen in muscle. As anticipated by subjecting the stress to another first-order system, the time delay was increased and transitions were further smoothed from the intermediate stress. Also noteworthy is the fact that these effects were more pronounced when the stress was passed through the second system than when passed through the first, due to the larger time constant for the second system. Deactivation of the muscle model (decrease in activation, as opposed to an increase) yielded an identical response, only inverted. When activation level was held constant long enough for the transient response to die out, the stress transitioned to a steady value that continued until a change in activation level occurred.

Another important observation was that, while the second order system affected active stress during the transient response, the steady-state stress was equal to the input contractile element stress. To illustrate, the simulation was performed with 0% strain, which gives $s \sigma_{TL}^* = 0.14$. Because no change in length occurred, σ_{TV}^* equaled 1. Multiplying

FIG. 5. Model active stress response to varied activation level input, showing activation and intermediate and final active stresses.

these by the maximum active stress allowed an active stress of 18.8 kPa with full activation. The active stress reached its steady state at this value with full activation, and only 25% of that was achieved with 25% activation. This effect was due to a coefficient of 1 for the stress (not the derivative of stress) in the linear differential equations. If a non-unity or time-dependent coefficient were used, the steady-state stress and/or scaling in the output would differ from the input making the output difficult to predict and potentially unrealistic.

B. Tetanic response

The tetanic response of the model was compared to data for canine CT muscle presented by [Alipour-Haghighi et al. \(1991\)](#). Both contraction (response to activation) and relaxation (response to deactivation) were considered.

Tetanic contraction responses of the model and CT muscle are shown in Figs. 6 and 7. Figure 6 gives normalized stress over time with activation occurring immediately at the start of the simulation, which is then held throughout. Model stress was normalized by the steady-state stress in order to compare with the already normalized muscle data. Muscle curves are shown for both the pars recta (CT-PR) and pars oblique (CT-PO) portions of CT muscle. Overall, the model response was a good fit to the literature data. The contraction

time to the maximum stress matched well and the curve, as it ramped up, was reasonably close. There was some deviation as activation initiated, particularly the model appeared to have a faster time response than the muscle, although this deviation was minimal and was not much greater than the difference between the curves for the PR and PO.

Figure 7 shows force over time, comparing the model response to a different sample of CT muscle. Here activation occurred at 0.06 s and was turned off at 2.0 s. Contraction responses of the model and literature data were close again, with a seemingly identical match at the initiation of the contraction and some deviation as the force neared its peak. The curvatures of the responses differ, mainly due to a longer than typical contraction time for the experimental muscle data, which reached 90% of the maximum force around 300 ms instead of the 200 to 250 ms reported as typical in literature and was also simulated in the current model. The maximum force was not reached until close to 1000 ms, approximately twice that of the model contraction time. While the peak forces also differ slightly, the difference is only about 2%, with values of 135 and 138 gf for the model response and experimental muscle data, respectively.

FIG. 6. Normalized tetanic stress response of model compared to canine CT muscle data from [Alipour-Haghighi et al. \(1991\)](#).

FIG. 7. Tetanic force response, including contraction and relaxation, of model compared to canine CT muscle data from [Alipour-Haghighi et al. \(1991\)](#).

Relaxation responses for separate samples of CT muscle are shown in Figs. 7 and 8, plotted with the response of the model to deactivation after 2.0 s in Fig. 7 and at the beginning of the simulation in Fig. 8. Figure 8 gives curves for the CT-PR and CT-PO portions. Both plots show differences between the model and measured CT muscle responses. While the model response was similar in shape to the muscle data, it exhibited a sharper decrease after deactivation and the response is slower. The model had a half-relaxation time of 77 ms, whereas the CT muscle relaxation times were 60, 57, and 67 ms for Fig. 7 and the PR and PO curves in Fig. 8, respectively. Because of the mismatch in curvature, the difference continued to increase below half relaxation. Time to 90% relaxation for the model was 207 ms, which was on average 85 ms slower than that for the CT muscle samples.

Of further importance is the observation of steady-state stress over time. Figures 6 and 7 show the stress over time after maximum stress was achieved and activation was held constant. When the model transient response died out, the stress remained at a constant maximum stress until some change in activation level occurred. On the other hand, the experimental muscle stress gradually decreased over time after maximum stress was reached. This stress relaxation is typical of biological tissue. The stress typically drops exponentially and will reach an equilibrium stress eventually. The data sets from Fig. 6 showed differences of 4% and 1% between model and experimental muscle stress for the PR and PO, respectively, after the first 1.0 s of tetanus (about 0.5 s at steady state). For such a short period, this difference may not be significant. However, over time the difference can become much more pronounced, and the eventual difference between the peak model stress and the CT muscle equilibrium stress will likely be substantial.

C. Twitch response

The twitch response of the muscle model was compared to a typical twitch response for laryngeal muscle from Perlman and Alipour-Haghighi (1988) (Fig. 9). Perlman and Alipour-Haghighi provided 85 V stimulation to the muscle for

FIG. 8. Normalized force resulting from tetanic relaxation simulation for present model compared to canine CT muscle portions reported by Alipour-Haghighi *et al.* (1991).

FIG. 9. Muscle twitch force for two simulations with the present model compared to typical twitch curve for laryngeal muscle from Perlman and Alipour (1988).

2 ms. For the present study the model was first stimulated with full activation for the same time of 2 ms. For the most part, the observed model response did not compare well with the experimental muscle data. The peak force produced by the model (about 5.2 gf) was only 22% of the approximately 24 gf peak of the CT muscle. The half-relaxation time (from peak force) of the model was 77 ms (same as with tetanic relaxation because of the dependence on system time constant), which was considerably greater than the 25 ms for the CT muscle. However, twitch contraction times were close with 37 and 34 ms for the model and CT muscle, respectively.

As discussed previously, additional twitch simulations were performed with the model, varying the stimulation duration in order to get a better match of peak force. It was found that when the model was stimulated for 20 ms the twitch curve gave a close match for peak force with the muscle. This curve generally had a similar shape to the experimental muscle twitch response. Because the activation duration was increased, the response had a longer contraction time (47 ms), and half-relaxation time was still 77 ms.

Also of interest was a comparison of the intermediate and final active stresses, shown in Fig. 10 along with

FIG. 10. Model active force response with twitch stimulation, showing activation and intermediate and final active stresses.

FIG. 11. Model force compared to CT muscle force presented by [Alipour-Haghighi et al. \(1991\)](#), in response to sinusoidal strain with activation after 1.75 s.

activation input. The simulated twitch was created by activating the model fully for 20 ms at the start of the simulation. The intermediate stress had a first-order ramp response until activation turned off, then a sharp change from increasing to decreasing stress followed by a first-order relaxation response. The final active stress was significantly different than the intermediate stress. The second-order response created a differently shaped curve and a continuous transition around the peak. As expected, the contraction and relaxation times were increased from the intermediate stress. The peak now occurred at the intersection with the intermediate stress curve. This slower response time caused the amplitude of the active stress to be much smaller than the peak intermediate stress because it did not increase much by the time the activation was turned off and relaxation began.

D. Sinusoidal strain

Depicted in Fig. 11 is the model stress resulting from a sinusoidal strain input with activation, compared to the same simulation performed on CT muscle by [Alipour-Haghighi et al. \(1991\)](#). The muscle was fully activated at 1.75 s, thus resulting in only passive stress for the first half of the simulation and combined passive and active for the latter half. Overall, the curves were remarkably similar. Shapes and amplitudes of each oscillation were generally the same. Peak stresses matched, both for passive only (first peak) and passive and active combined (third peak).

The present model response differed somewhat from the experimental muscle response, the main dissimilarity being in peak stress. While the model predicted the first passive peak stress well, the peak stress during the second oscillation had decayed more in the model than in the CT muscle. The third peak stresses agreed well again, but the fourth peak stress was greater for the model than the CT muscle. Other small differences between the data sets existed, particularly as stress increased. The model had a faster response when only passive stress was present and a slightly delayed response with activation. The greatest discrepancy was seen on the upslope of the third peak as activation started. The fourth peak might have shown similar deviation if the peak stresses were equal. Passive stress downslopes also showed slight disagreement between the model and CT muscle curves.

IV. DISCUSSION AND CONCLUSION

The purpose of this study is to examine a new muscle model which added active properties to an accurate passive material model. The results overall suggest that the combination of the three-network Ogden model and a Hill-based active model estimates well the passive and active stresses of muscle during both isometric and dynamic conditions. In comparison to isometric tetanic contraction of CT muscle, the model stress response shows appropriate contraction times and matching curvature. Tetanic relaxation and twitch responses are also similar to those exhibited by real muscle. Furthermore, the model provides an excellent approximation of the muscle force (stress) behavior resulting from dynamic stretch and release with activation, as compared to published data for canine CT muscle.

Aside from mimicking important behavior of canine CT muscle, there are a few reasons that this model is favorable for computational simulations of laryngeal muscle. First, because it is a one-dimensional model (which is the predominant behavior of laryngeal muscles): (1) It has a simple, discrete method of calculation; (2) it is not computationally expensive; and (3) it can be solved for individual time steps. Eventually, complex two- or three-dimensional finite element models of the vocal folds or larynx could use components of the current muscle model. Second, because the steady state output stress equals the input contractile element stress, the model is predictable and convenient. No extra consideration is necessary to determine the output stress magnitude after passing through the two systems. This input–output relationship may not be the same physiologically (i.e., a given percentage of stimulation may not produce the same percentage of maximum stress), but such a relationship is desirable for modeling purposes. Finally, while the passive and active stresses are coupled and certainly affect each other to an extent in actual muscles, it is desirable that they are separate in modeled muscles. Since the three-network Ogden model is an accurate representation of passive mechanical properties of laryngeal tissue on its own, it was of importance for this study to include an active component without affecting the accuracy of the passive model. Also beneficial is the ability to separately consider active and passive contributions. The model as it stands is able to estimate separately and combined the appropriate behavior for laryngeal muscle.

This new muscle model represents an improvement over previous models. Most importantly, the new model can model passive stress, particularly viscoelasticity. Viscoelasticity in the Kelvin model is accounted for with a single time constant to describe stress relaxation, which is admittedly inaccurate (Titze, 2006) since it is known that soft tissue relaxation is described with an infinite number of time constants (Fung, 1972; Alipour and Titze, 1985). On the other hand, the three-network Ogden model better matches the viscoelastic effects because, in its present form, it includes two time-dependent viscoplastic networks. Additional networks can also be added to further increase its ability to match viscoelastic effects of biological tissue. These additional capabilities allow the new muscle model to be useful for simulating voice phenomena in which tissue viscoelastic effects may play a significant role, not possible with previous models.

While not as crucial, other additional modeling capabilities in the current model are still noteworthy. For example, one obvious benefit to the current model over previous laryngeal models is its ability to change the shape factor in the force-velocity equation. Also of note is the slight difference in how the system responds between the two models. This new model applies the second-order response to the active stress alone, while the most previous laryngeal models like the modified Kelvin (Hunter *et al.*, 2004) model, the active stress is passed first through a single first-order system and then through a second first-order system after the addition of passive stress. Although the modified Kelvin model has the benefit of coupled active and passive responses, the new model can accurately describe the active and passive stresses separate from one another. While these differences exist, they likely do not significantly influence certain behavior of either model or its ability to generally describe many characteristics of laryngeal muscle. In fact, the responses of both models in mimicking the sinusoidal stretch and release experiment are similar (see Titze, 2006, for Kelvin model response).

As with any estimation of biological materials and processes, the present model has limitations and potential for improvement in future studies. One such limitation of the present model was in predicting stress relaxations for the simulations considered here. In the tetanic responses, the experimental CT muscle stress showed relaxation of the peak stress/force over time while the model apparently exhibited none. In fact, there was a small amount of relaxation in the passive stress produced by the pre-strain, but because the passive stress was small and the effect was long-term, the amount of decay during the simulation was negligible compared to the peak stress/force. The discrepancy between peak stress for the model and CT muscle responses in the sinusoidal strain simulation was also due to differing levels of stress relaxation. Namely, the second peak was under-predicted by the model because relaxation of the passive stress alone was faster than for this particular CT muscle. Later, at the fourth peak, the model stress was over-predicted because of a slower relaxation rate with activation than was present in the CT muscle. In the future, the prediction of stress relaxation can be improved in two ways. First,

the Ogden model parameters can be recalibrated to match the stress relaxation behavior of any muscle passive behavior, as well as contribute to appropriate active stress decay. Second, while in some cases it may be desirable to use the model to predict an ideal situation where active stress is able to be held constant, a more realistic response can be obtained by incorporating an active stress decay into the model.

Another area that did not show agreement with experimental muscle data was the model twitch response. It required a substantially longer duration of stimulation to produce similar peak force as the sample of laryngeal muscle it was compared to, and the relaxation was much slower than averages reported for CT muscle. The exact reasons for this under-prediction of peak stress are uncertain. It is known that the peak stress in the twitch can be improved by changing the system coefficients. However, this would consequently alter contraction/relaxation times to undesirable values. It is possible that other parameters used in the model did not adequately reflect the properties of the specific laryngeal muscle sample used for the published simulation, as details about the type of muscle and its properties were not reported by Perlman and Alipour-Haghighi (1988). If so, appropriately adjusting parameters will improve the response. It is also possible that twitch must be described in a different manner than the present approach. In any case, since muscle twitch is typically not of concern when simulating voice phenomena in laryngeal models, this limitation of the model does not lessen its usefulness for laryngeal modeling.

Tetanic relaxation was an area of weakness for the present model in that predicted relaxation times were significantly longer than those measured in real muscle. This also contributed to the poor match with the twitch response. Altering the system equations to include non-unity or time-dependent coefficients can make it so that the relaxation time can be different than the contraction time as would be necessary for both values to be correct. Unfortunately, as mentioned earlier, this would change the input-output relationship such that the input stress would not equal the output steady state stress. Thus, it was deemed more desirable for this model to have some error in the relaxation time than to have such a complicated response. Other approaches for the active stress response may be incorporated into this model in the future to allow for accuracy of both contraction and relaxation times, as well as an improved twitch response.

Some additional steps to be taken in developing the model include further verification by comparison to experimental data and a more in-depth characterization of the model behavior through performing a sensitivity analysis. Further verification would likely only slightly modify the model parameters but may be important when modeling different muscles which all work in concert, such as the various intrinsic muscles of the larynx while moving the vocal folds. Sensitivity analysis is important to show the effects of varying input parameters and would provide insights into optimizing the response to minimize discrepancies with experimental data, as well as show the range of capabilities of the model. As was touched upon in Sec. I, the goal of this paper was to determine the efficacy of adding active

properties to the passive Ogden model and seeing how well the combination is able to replicate muscle behavior in experimental simulations. This paper has shown that their combination produces a suitable response for modeling laryngeal muscle.

Biomechanical models of the larynx are improving, becoming more complex and realistic representations of voice and speech. With these improvements, the potential effects of clinical and surgical intervention can eventually be simulated. However, for such simulations to be useful in real world contexts, they must include, for example, such laryngeal fatiguing behaviors as a school teacher speaking for an extended period of time or a singer performing a high note for 10 s with vibrato. These simulations would require both active muscle modeling as well as passive muscle modeling (including addition of stress relaxation over time). Previously, no reported laryngeal muscle model could adequately capture the case of a long sustained sung high note while including the stress relaxation that the singer would have to be constantly compensating for with active muscle contraction. The muscle model presented here is a significant step forward in allowing such simulations to be possible.

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