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Modeling laryngeal muscle activation noise for low-order physiological based speech synthesis

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Abstract

Physiological-based synthesis using low order lumped-mass models of phonation have been shown to mimic and predict complex physical phenomena observed in normal and pathological speech production, and have received significant attention due to their ability to efficiently perform comprehensive parametric investigations that are cost prohibitive with more advanced computational tools. Even though these numerical models have been shown to be useful research and clinical tools, several physiological aspects of them remain to be explored. One of the key components that has been neglected is the natural fluctuation of the laryngeal muscle activity that affects the configuration of the model parameters. In this study, a physiologically-based laryngeal muscle activation model that accounts for random fluctuations is proposed. The method is expected to improve the ability to model muscle related pathologies, such as muscle tension dysphonia and Parkinson's disease. The mathematical framework and underlying assumptions are described, and the effects of the added random muscle activity is tested in a well-known body-cover model of the vocal folds with acoustic propagation and interaction. Initial simulations illustrate that the random fluctuations in the muscle activity impact the resulting kinematics to varying degrees depending on the laryngeal configuration.

Index Terms: Speech synthesis, vocal folds, voice, muscle activation

1. Introduction

Lumped-element models of voiced speech are capable of investigating a wide range of controlled scenarios at minimal computational cost. These models can mimic and predict complex physical phenomena observed in speech production and have been shown to be useful tools for the investigation, diagnosis, and treatment of voice disorders [1] [2]; furthermore, they have received significant attention due to their ability to efficiently perform comprehensive parametric investigations that are cost prohibitive with more advanced computational tools [3]. Recently, these reduced order models have been recently shown advantageous in that they can yield data that are difficult or impossible to clinically acquire, by means of Bayesian estimation methods [4]. In spite of these notable advancements, several physiological aspects of these reduced order models remain to be incorporated, particularly when describing their connection with the nervous system.

Reduced order vocal fold (VF) models are constituted by a set of coupled components, namely a lumped element description of the VFs, an analytical solution of the glottal flow behavior, and a plane wave representation of the sub and supra glottal acoustic fields. Herein, the VF model is typically configured using physiological rules of muscle activation [5], that allow for a meaningful construction of the VF model parameters. However, the activation rules are based upon several untested assumptions and neglect any neural description of the laryngeal muscle activity. The lack of such neural descriptions results in perfectly constant muscle activations and unnatural sound quality for the synthetic voices.

In this study, a physiologically-based laryngeal muscle activation scheme that accounts for random fluctuations is presented. The proposed method is expected to improve both the physiological relevance of the overall speech production model and the ability to model muscle related pathologies, such as muscle tension dysphonia and Parkinson's disease. At the same time, natural random fluctuations in the model parameters are expected to enhance the resulting sound quality of the voice synthesizer.

2. Methods

2.1. Physiological and morphological aspects of muscular activation

From a neural perspective, the basic unit of a muscle is the *motor unit* (MU), which consists on a *motor neuron* and *muscle fiber* innervated by the axon of the neuron. Large muscles with wide and gross movements have thousands of fibers per motor neuron, while muscles that perform precise contractions have fewer fibers per motor neuron. When a neuronal signal is sent, a MU action potential is generated, which stimulates the fibers to contract synchronously. The electric impulse generated, which corresponds to the sum of all action potentials, is known as motor-unit action potential.

For each spike that stimulates a fiber, a single twitch (or fiber contraction) is produced on them, lasting for a fraction of time. Successive twitches may add up to generate a stronger action [6]. Figure 1 illustrates the temporal summation effect of successive twitches. If spikes are fired at a higher rate, the resulting contraction is larger, thus having a wave summation effect, resulting in temporal variability. This effect is also illustrated in Figure 1, in which neuronal variability can be observed at a single fiber level.

Amplitude and temporal properties of a twitch depend on the type of fiber that comprises a MU. In our scheme, all muscle fibers in a MU are assumed to be of the same type, and although there are many types of fibers, they can be simplified into two principal groups: Type I or *slow*, and Type II or *fast* [7]. Slow fibers are fatigue-resistant, with the smallest force

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or twitch tension and slowest contraction. Fast fibers can also be fatigue-resistant, with large forces and faster contraction, or easily fatigable but having the largest force and fastest contraction. Figure 2 shows typical slow and fast twitches for laryngeal muscles, using an alpha waveform as a template [8].

A muscle is composed of many groups of motor units. Normally, not all MUs in a muscle are identical, having a proportion of slow and fast MUs. Also, MUs are not triggered independently: if the activity in motor neurons increases, then additional motor units are also activated to correspond to the increasing contraction strength. This effect is known as *recruitment* [9] [10].

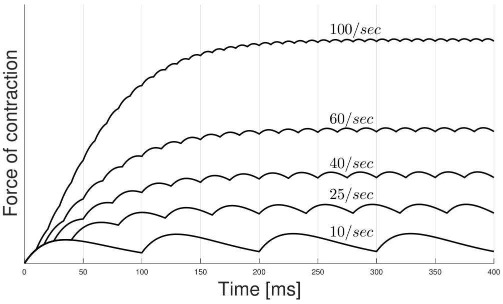


Figure 1: *Force of contraction of a single muscle fiber as a function of time for various motor neuron firing rates. Wave summation effect creates variability allong time.*

2.2. Wave summation model

MUs are recruited into group of motor units (GMUs), where we assume that all GMUs are identical for a given muscle; that is, they have the same proportion of slow and fast MUs. In addition, the *rule of five* is assumed for the recruitment of GMUs, in which each subsequent GMU is recruited if an active GMU increases its firing rate by aproximetly $5[Hz]$ [11]. Considering N Groups of MUs, in which the first one is firing at F[hz], then the set of equations that governs the recruitment of GMUs are :

$$p_m(F,N) = \sum_{j=1}^{N_a}\sum_{k=0}^{\infty} n_s \cdot p_s(t-k/F_j) + \sum_{j=1}^{N_a}\sum_{k=0}^{\infty} n_f \cdot p_f(t-k/F_j) \quad (1)$$

$$p_{s,f}(t) = \frac{t}{\tau_{s,f}} e^{-(t-\tau_{s,f})/\tau_{s,f}} \quad (2)$$

$$N_a = min\,(N, F/5) \quad (3)$$

$$F_j = F - 5(j-1), \qquad j = 1, ..., N_a \quad (4)$$

where p_m is the resulting time series of contraction force for a given muscle m, n_s and n_f are the numbers of slow and fast fibers in a GMU, respectively, p_s and p_f are the slow and fast twitch responses (for the slow and fast fibers), respectively, and τ_s and τ_f are the respective time constants for the twitches. Equation 1 defines p_m as a successive summation of twitches, considering the effect of each GMU. An infinite spike train is considered as an input, represented by the summation in k. Each time a spike arrives, a twitch is triggered. This twitch is mathematically represented by equation 2.

Equation 3 defines N_a as the maximum number of active GMUs, considering a firing rate F, and equation 4 defines the firing rate of recruited GMUs (rule of five). Each GMU (labeled by $j = 1, ..., N_a$) is firing at a different rate F_j, as noted by Mårtensson [12]. A single GMU can only fire at a maximum rate of F_{max}. If $F_j > F_{max}$, then F_j is set at F_{max}. Logically, this is applied after the recruitment step, so there may be some GMUs firing at F_{max} while there are others below this value.

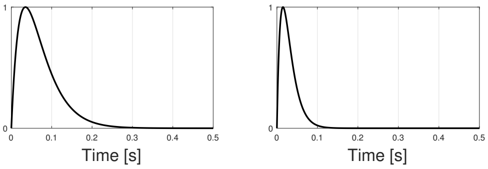


Figure 2: *Fast (right) and slow (left) twitches waveforms. Each time a MU fires a spike, a corresponding twitch is triggered as a response. The sum of successive twitches is the basis of the wave summation model.*

In an effort to capture the inherent variability in neural firing, and thus the contraction response, a degree of randomness is also added to various components. First, a the rule of five, each time a new GMU is recruited, the firing rate for the new GMU is set at a value between 3 to 7 (normal distribution with a mean of 5 and a standard deviation of 2), instead of 5 Hz fixed. Also, it is known that intervals between spikes are not regular. Based upon the results of Moritz *et al* [13] that describes the inter spike interval (ISI) in muscular activation, a normally distributed ISI with a coefficient of variation (CV) of 0.2 was considered.

We now normalize the resulting force of contraction to describe the resulting muscle activation. Thus, the activation a_m for a given muscle m is defined as follows:

$$a_m(F,N) = \frac{\boldsymbol{p_m}(F,N)}{max\,\{p_m(F_{tet},N)\}} \quad (5)$$

in which $\boldsymbol{p_m}$ is the time series for the force of contraction with random components (note the bold notation to differentiate from p_m which has no random components). For the normalization step, the model described in equation 1 is used (without any random components). The muscle activation a_m is normalized, so the muscle is considered fully activated (or *tetanized*) if $a_m = 1$. Conversely, a muscle has no activation related if $a_m = 0$, which has no firing rate associated. However, this situation is never used because a fully relaxed muscle has a residual muscle tension. F_{tet} is the fire rate at which all GMUs are firing at maximum capacity, considering p_m (with no random components). F_{tet} should be higher that F_{max} due to the rule of five.

2.3. Model Parameters

The model proposed can be used to simulate any muscle in which muscle activation is required. In this case, two muscles were simulated: the *thyroarytenoid* (TA) and the *lateral cricoarytenoid* (LCA), due to their importance in voice phonation. Both TA and LCA are considered fast muscles, with 10% of slow fiber and 90% of fast fiber [12]. The parameters related to the model are presented in Table 1

Table 1: *Parameters for CT and LCA*

Muscle	TA	LCA
Type of muscle	Fast	Fast
Number of MU per GMU	350	370
Fibers per MU	10	17
Fibers per GMU	3500	6290
Slow fibers per GMU n_s	350	629
Fast fibers per GMU n_f	3150	5661

For the twitch waveforms, the *alpha* function was used [8], which is described in equation 2. For the time constants, values of $\tau_s = 35[ms]$ and $\tau_f = 15[ms]$ were used for slow and fast twitches respectively [12] (see equation 2). At the same time, N=10 GMUs were considered for every simulation [12].

The simulation time was 5 seconds, discarding the first second, so the transient of the system was not considered in the statistical analysis. It is important to notice that each time a signal was generated, the mean value of a_m was different. To correctly estimate the mean value of a_m, 40 simulations were considered for each frequency of fire.

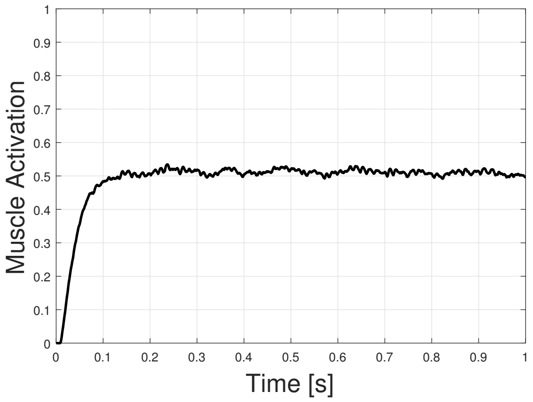


Figure 3: *Example of muscle activation for the TA muscle, firing at* $100[Hz]$. *Activation is around* 0.5. *For this case,* $1[s]$ *is simulated. It can be seen that in this case, it takes around* $0.2[s]$ *to get to stationary state.*

3. Results

The relationship between rate of fire and mean activation is first characterized. Although this was done for the TA muscle, the result is the same for LCA. The mean activation was computed by averaging all mean activations obtained at each fire rate, starting from $10[Hz]$, up to $300[Hz]$. The result is illustrated in Figure 4. The range between $40[Hz]$ and $180[Hz]$ is known as dynamic range. In this range, the model has a linear behavior. Below this range, some GMUs start to become inactive, thus decreasing the effect on mean activation. On the other side, above $180[Hz]$ GMUs start to fire at their maximum capacity, reaching tetanization at around $200[Hz]$. When tetanization is reached, activation saturates just below $a_m = 1$.

The linear relationship provides a mapping between fire rate and mean activation, thus allowing an analysis of the effect of noise in the activation on models that consider this parameter as fixed. For the purpose of this study, the body-cover model (BCM) developed by Titze and Story [14] was considered. This low-dimensional model was chosen due to its simplicity, but also because it enables a natural connection between contraction of the cricothyroid and thyroarytenoid muscles and stiffness parameters. This dependence is ruled by a set of physiologically-based equations described also by Titze and Story [5].

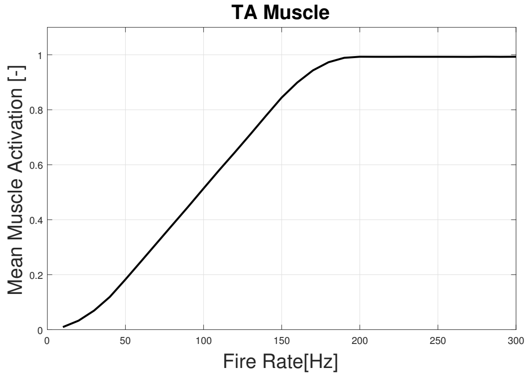


Figure 4: *Mean activation vs firing rate, for TA muscles. In this case,* F_{max} *is set at* $150[Hz]$*, so the tetanization frequency* F_{tet} *is approximately* $200[Hz]$*, due to the rule of five. At this frequency, all 10* $GMUs$ *are firing at* F_{max}. *Between* 40 *and* 180 *[Hz] a linear behaviour can be observed.*

3.1. Effect of Neural Noise on the Body-Cover Model

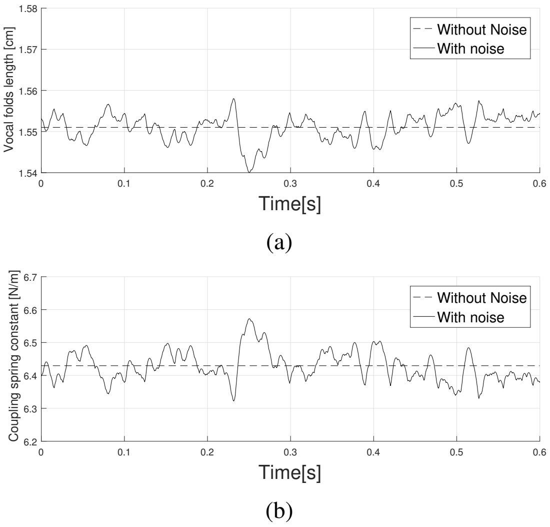
The BCM model is controlled by a set of parameters that depends on the muscular activation of different muscles, like TA and LCA. For the following simulations, fire rates for TA and LCA are set at values in which mean activations are the same as fixed activations in the simulation without noise. This values are $a_{TA} = 0.25$ and LCA at $a_{LCA} = 0.5$. Also, fixed activation for the non-simulated muscle *cricothyroid* is set at $a_{CT} = 0.2$.

Figures 5a and 5b show that with a noisy simulation of TA activation, a variation in time can be incorporated in parameters of the body-cover model. Also, this are just two of the many parameters that are affected by this.

For a given combination of muscle activations, the BCM can enter on a self-oscillating state, or can start a damped oscillation. With this behavior in mind, a comparison between a constant and a noisy activation was made, so the effect of noise can be directly seen on the VF oscillation. Figure 6 illustrates this idea. Muscle activation for LCA is simulated, introducing noise in the model and showing how it reflects on a glottal area graph. As it can be seen, with a combination of activations of $a_{TA} = 0.25$, $a_{LCA} = 0.5$ and $a_{CT} = 0.2$, the model enters in a stable oscillation state. With noise however, this oscillating state becomes very irregular in its amplitude, but keeping its fundamental frequency relatively constant.

The muscle activation plot (MAP) that Titze and Story presented in their study [5] can be used to understand the results presented in Figure 6. Data points show region of self-sustained oscillation with the convergence rule, and for the given combination, oscillation is effectively happening. However, self-sustained oscillation state is limited by a narrow band around 0.5 for lateral cricoarytenoid activity. This implies that if noise is induced in LCA activation, then the BCM could continuously step in and out of the self-sustained oscillation state, which explains the irregular behavior in glottal area waveform.

Figure 7 shows a second comparison, in which noise in TA



(a)

(b)

Figure 5: *Effect of noise in TA muscle activation on vocal fold length (5a) and coupling constant (5b) of the BCM. Dashed line shows the value with no noise on the activation, considering the mean value of the noisy case.*

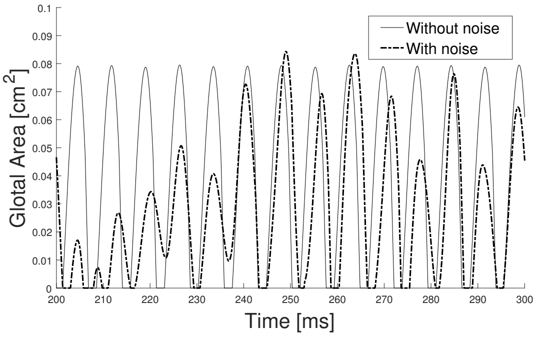


Figure 6: *Comparison between a simulation using the normal and noisy activation. Activations on cricothyroid (CT) and thyroarytenoid (TA) are set at* 0.2 *and* 0.25 *respectively. Fire rate for lateral cricoarytenoid is set at* $97[Hz]$*, which gives a mean activation around* 0.5.

activation is induced. In this case, differences are minimum, showing that in this case the model is almost not affected by noise in this activation. In the two previous cases, fundamental frequency is not affected, althought the waveform is different. This illustrates that the effect of the neural noise is variable depending upon the model configuration and requires further evaluation in a parametric study.

4. Conclusion

The wave summation model can be used to include neural variability in muscular activation over time. Although the proposed neural scheme has been tested only in the Body-Cover Model, it is possible to use it in other vocal fold numerical models, like a bar-plate model [5]. The scheme looks promising in the context of studying the effect of noisy muscle activation in vocal fold numerical models where the behavior of the body cover model can vary significantly in terms of its glottal area.

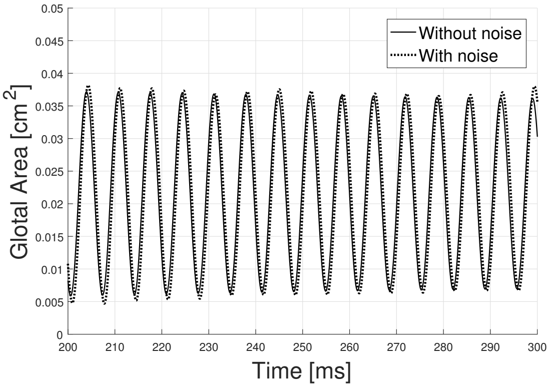


Figure 7: *Comparison between a simulation using the normal and noisy activation. Activation on cricothyroid (CT) and lateral cricoarytenoid (LCA) are set at* 0.2 *and* 0.5*, while TA had a mean value of* 0.25.

The neural noise scheme for muscle activation presented in this study can be use to simulate a different set of muscles simultaneously. In future efforts, *cricothyroid* will be simulated, given its impact on fundamental frequency regulation [15]. A comprehensive sensitivity analysis will be performed to assess the effect of the neural noise in various configurations.

5. Acknowledgements

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6. References

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