A Neuromuscular Elbow Model for Analysis of Force and Movement Variability in Slow Movements

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Abstract— In this paper, we present a neuromuscular elbow model with both motor unit pool recruitment and Hill-based contraction dynamics. The model builds upon various models reported in the literature and provides a way to quantify force and movement variability in both isometric and non-isometric contractions. The model's accuracy in estimating muscle force variability at low force levels (at less than 20% maximum voluntary contraction) is evaluated in isometric contraction case and compared with experimental results from the literature. This comparison suggests that the model is accurate in estimating force variability within the low force range and can be used to explore effects of muscle force variability in increased kinematic variability during slow movements.

Index Terms— Movement variability, muscle force variability, movement intermittency, muscle models, motor-unit pool models.

I. Introduction

Although speed-accuracy trade-offs and planning and execution of rapid goal-directed movements have garnered significant research interest, far fewer studies have reported results on the lower end of the movement speed spectrum. Not only do very interesting observations exist that are unique to slow movements, but an explanation of these observations is highly relevant to motor function recovery and motor skill learning, where movements are typically slow at the initiation of therapy or learning, and movement speed increases through practice, exercise or therapy. Specifically, in their study on movement intermittency in slow movements, Doeringer and Hogan [1] showed that voluntary movements become considerably intermittent (or non-smooth) with decreasing movement speed.

Hamilton et al. [2] demonstrated that larger muscles produce less variable forces, due to increased total number of motor units. Hamilton et al. also stated that it is likely that a similar relationship is in effect for the number of active motor units during a contraction, within the same muscle, and that this mechanism is responsible for the increased coefficient of variation[1] (CV) for low force levels, a well-documented observation in several studies in the literature [2]–[4]. This observation provided motivation to explore whether the increase in muscle force generation variability for small forces due to low number of active motor units can explain the increased variability in slow movements.

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[1]Coefficient of variation corresponds to standard deviation divided by the mean.

In this paper, we present a neuromuscular elbow model to be used in evaluating force and movement variability in slow movements. Specifically, this work is part of a modeling-based approach to answer the question "can increased muscle force variability in low force levels explain increased variability or intermittency of slow movements?" In this study, we report results for only isometric conditions due to space constraints, although the overall model is structured to be able to handle non-isometric conditions as well. The paper is structured as follows: Section II presents the model in detail, Section III presents the force variability results of the model and discusses the implications of these results for future work. Section IV concludes the paper.

II. Neuromuscular Elbow Model

There are mainly two types of muscle models in the literature. First is motor unit (MU) pool-based models commonly used to gain insight into isometric force variability. Second is Hill-based muscle contraction models that are commonly used to study numerous types of biomechanical movements and their control.

Fuglevand et al. [5] proposed a MU pool-based model that included both surface electromyogram (sEMG) and force predictions under isometric conditions, in comparison with experimental recordings. This model has become widely accepted and has been adopted by many studies, and later was extended to study effects of synchronization in MU pools [6].

Hill-based models originated from Hill's seminal work on energetics of muscle contraction [7]. Zajac's review of muscle and tendon models [8] is the most comprehensive reference on Hill-based muscle models. These are lumped-parameter models that approximate the force-length and force-velocity properties (or equivalently the dynamic behavior) of the musculotendon complex at a fidelity sufficient to study biomechanics of multi-muscle or multi-joint movements.

Selen et al. [9] proposed a combination of these two types of models in their work where they studied whether co-activation of muscles can be used as a strategy to decrease variability. He showed that Hill-based models alone with Gaussian stimulation noise cannot account for the force variability observed experimentally in the literature [4], [10]. A model combining Hill-based muscle contraction with MU pool recruitment and rate coding dynamics, however, was found to be in agreement with experimental results and used to explain that co-activation indeed can be a valid strategy to reduce movement variability.
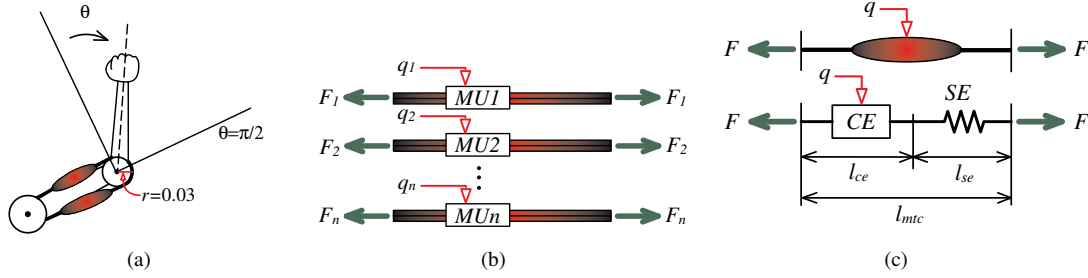
Fig. 1. (a) An equivalent agonist-antagonist pair of uniarticular muscles drive the elbow joint in my model. (b) Each muscle consists of $n = 60$ motor units. q denotes the active state for each motor unit. (c) Although Hill-based contraction models are commonly used in explaining lumped-parameter behavior of whole muscles, in our model, each motor unit has a contraction behavior defined by an active contractile element (CE, muscle tissue) and a series elastic element (SE) with nonlinear stiffness (tendon). See text for explanation of all variables. Also see the functional block diagrams corresponding to each level of the model in Fig. 2

Selen et al.'s model [9] provided an attractive starting point for our model since it combined widely accepted MU pool models of force variability with Hill-based contraction dynamics and musculoskeletal dynamics to study kinematic variability. Our model most closely resembles that of Selen et al. [9], and the contraction model is described in more detail in the studies by Van Soest and Bobbert [11] and by Ridderikhoff et al. [12]. The motor unit pool model is similar to that of Selen et al. [9], however, we used parameter values from Fuglevand et al. [5] for a portion of the parameters, due to better agreement with experimental data for the biceps muscle. A list of parameters with their units and detailed explanations are not provided here due to space constraints and the the reader is suggested to refer to the mentioned references for this information.

1) Activation and Contraction Model: Similar to Selen et al. [9], we used an agonist-antagonist pair of muscles to drive the elbow. Our activation and contraction model most closely follows the model by Ridderikhoff et al. [12]. Schematic representations of the model are illustrated in Fig. 1 and functional block diagrams of three levels of the model are given in Fig. 2. The activation-contraction model block diagram is given in Fig. 2(a). The first order activation dynamics of each motor unit is defined by

$$\dot{\gamma} = \frac{c \times nFR - \gamma}{\tau_\gamma}, \quad \tau_\gamma = \begin{cases} \tau_{act} = 89\text{msec}, & c \times nFR \geq \gamma \\ \tau_{rel} = 178\text{msec}, & c \times nFR < \gamma \end{cases} \quad (1)$$

where γ is the intramuscular concentration of Ca^{+2}, $c = 0.1373 \times 10^{-3}$ is a gain coefficient, nFR is normalized firing rate of motoneurons and τ_γ is the time constant, defined differently based on activation and relaxation cases [12]. Active state q is dependent on the Ca^{+2} concentration and the length of the contractile element l_{ce} through the nonlinear relationship

$$q = \frac{q_0 + (\rho\gamma)^3}{1 + (\rho\gamma)^3}, \quad \rho = Gl_{ce}\frac{\lambda - 1}{\lambda l_{ce,opt} - l_{ce}} \quad (2)$$

where $q_0 = 0.005$, $G = 52700$, and $\lambda = 2.9$.

The force exerted by each motor unit is calculated by the equation

$$F_{se} = \begin{cases} k_{se}l_{se}^2, & l_{se} \geq 0 \\ 0, & l_{se} < 0 \end{cases} \quad (3)$$

where $l_{se} = l_{mtc} - l_{ce} - l_{se,slack}$ is the tendon elongation length, $l_{se,slack} = 0.170$ m is tendon slack length, $l_{mtc} = 0.312$ m is musculotendon complex length (it is given by $l_{mtc} = 0.312 \pm r\theta$ for non-isometric simulations) and k_{se} is the stiffness of the tendon. k_{se} values for each motor unit are determined at a tendon elongation of $0.0333 \times l_{se,slack}$ [8], [13] and depend on the maximum force F_{max} each motor unit can generate. F_{max} varies exponentially among motor units and is described in detail later. Note that $F_{ce} = F_{se}$ due to the serial configuration.

The force-length relationship is given by

$$F_{isom} = max\left(1 - \frac{1}{w^2}\left(\frac{l_{ce}}{l_{ce,opt}} - 1\right)^2, 10^{-5}\right) \quad (4)$$

where F_{isom} is the normalized maximum isometric force at muscle length l_{ce} and $w = 0.56$ is parameter for the width of the relationship. $l_{ce,opt} = 0.136$ m is the optimum fiber length.

Concentric (shortening) contractions ($F_{ce} \leq qF_{max}F_{isom}$) are governed by the equation

$$\dot{l}_{ce} = -v_{scale}l_{ce,opt}\left(\frac{F_{isom} + v_{shape}}{\frac{F_{ce}}{qF_{max}} + v_{shape}} - 1\right) \quad (5)$$

where

$$v_{scale} = min(1, 3.333q)B_{rel}$$
$$v_{shape} = \begin{cases} A_{rel}F_{isom}, & l_{ce} \geq lce,opt \\ A_{rel}, & l_{ce} < lce,opt \end{cases}$$
$$A_{rel} = 0.41, \quad B_{rel} = 5.2.$$

Eccentric (lengthening) contractions ($F_{ce} > qF_{max}F_{isom}$) are governed by the equation

$$\dot{l}_{ce} = \frac{p_1}{\frac{F_{ce}}{qF_{max}} + p_2} + p_3 \quad (6)$$

Fig. 2. Block diagrams of the neuromuscular elbow model. See text for explanations of the blocks and parameter values. (a) Block diagram of the activation and contraction model. (b) Block diagram of the motor unit pool model. Expanded version of the contraction block (highlighted in yellow) is given in (a). (c) Block diagram of the elbow neuromusculo-skeletal system with an antagonist pair of muscles. Detailed version of the Muscle 1 block (highlighted in yellow) is given in (b).

where

$$p_1 = -\frac{v_{scale}}{\sigma} \frac{((1 - F_{ecc})F_{isom})^2}{F_{isom} + v_{shape}}$$

$$p_2 = -F_{ecc}F_{isom}$$

$$p_3 = -\frac{p_1}{F_{isom} + p_2}$$

In these equations, $F_{ecc} = 1.5$ represents the maximum eccentric force as a fraction of F_{isom} and $\sigma = 2$ is the ratio of eccentric and concentric contraction curves at zero velocity. The activation and contraction model constitutes a sub-component of the motor unit pool model (see Fig. 2(b), highlighted yellow block), which is described next. We implemented a linear eccentric contraction condition to avoid numerical problems in very low active states, similar to the one defined by Ridderikhoff et al. [12], which is not reported here, and the reader is referred to [12] for details.

2) Motor Unit Pool Model: A schematic representation of the motor unit pool model is illustrated in Fig. 1(b). Fig. 2(b) provides a block diagram of the model. The formulations below follow closely that of Selen et al. [9], however some parameters are replaced with values in [5].

A MU is recruited when excitatory input (E) exceeds the recruitment threshold (RTE). RTEs of motor units vary exponentially based on the equation

$$RTE(mu) = \exp(mu(\ln RR)/n) \quad (7)$$

where mu represents the index of the motor neuron, $RR = 75$ is the recruitment range and $n = 60$ is the total number of MUs. The firing rate of a recruited neuron is given by the equation

$$FR(mu) = g(E - RTE(mu)) + mfr, \quad E \geq RTE(mu) \quad (8)$$

where the gain $g = 1.36$ and minimum firing rate $mfr = 8$ pps. FR saturates at 41.6 pps for all motoneurons. The interspike interval ($ISI(mu)$) is calculated from the inverse of $FR(mu)$. A Gaussian noise with CV of 0.2 is added to the $ISI(mu)$, and this constitutes the main source of variability in the model. After the noise is added, a normalized firing rate ($nFR(mu)$) is calculated by inverting $ISI(mu)$ with noise, and dividing by maximum FR. To avoid saturation of motor unit activation and hence force variability due to saturation in q dynamics [9], $nFR(mu)$ is halved. $nFR(mu)$ provides both the normalized stimulation input to the muscle activation-contraction model, and the duration of this input, which corresponds to the $ISI(mu)$. The maximum force value for each motor unit was calculated from the equation

$$F_{max}(mu) = h\exp(mu(\ln RF)/n) \quad (9)$$

where $RF = 108.1$ denotes the range of forces, giving a 1:100 ratio for the ratio of maximum forces of the first and the last MUs. h is a constant used to obtain a total maximum force of 2000 N for the whole muscle. The force outputs of all MUs are summed to obtain the total muscle force following the independent tendon model used by Selen et al. [9]. A time step of 1 msec and Euler's integration method are used for the model.

III. RESULTS AND DISCUSSION

To evaluate the accuracy of the model in replicating experimental force variability under isometric conditions, plots of SD of force and CV of force vs mean force level are generated and presented in Fig. 3. In these plots, each data point is based on 20 simulations of the isometric model for 3 sec, and only data within the last 2 sec (after force stabilization) is used. Both mean and SD (across 20
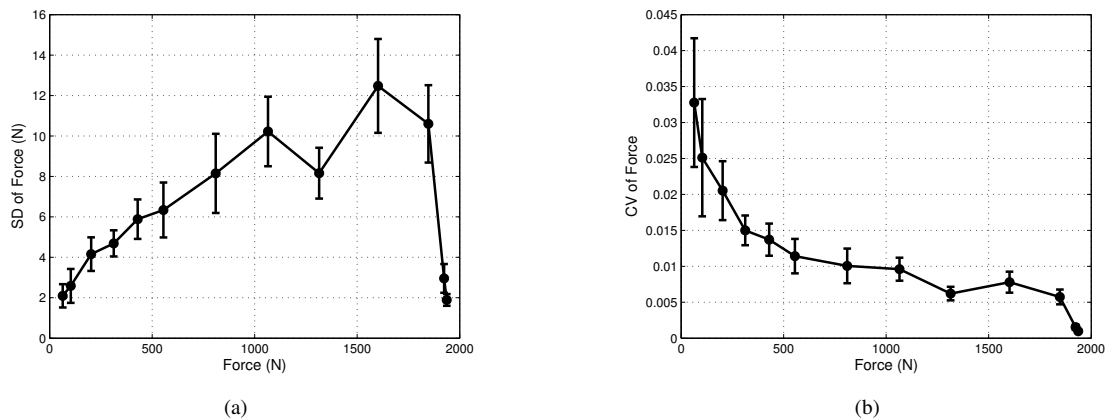
(a)

(b)

Fig. 3. Standard deviation (a) and coefficient of variation (b) of force, estimated from the isometric muscle model. Markers represent mean values, while error bars represent standard deviation, both across twenty simulations. A comparison with experimental results by Taylor et al. [4] shows that the model captures an accurate representation of experimental results.

simulations) of SD and CV of force are illustrated in these plots. When compared with results by Taylor et al. [4] (see Fig. 4 in this reference), it can be observed the model captures an accurate representation of experimental results. The trends in the SD of force closely resemble those from experiments. The CV of force values from the model match the experimental values reasonably well in the first quarter of mean force levels. This low range of forces are the most relevant range due to our focus on slow movements. The model achieved a reasonable match with experimental observations within the low force range and future work will use the model in predicting kinematic (speed) variability during slow movements. This way the model described in detail here will be used in future work to seek an answer to the question whether increased variability or intermittency of slow movements can be explained by increased muscle force variability in low force levels.

IV. Conclusion

We presented a neuromuscular model in detail for the elbow, with the ultimate goal of using the model to explore the effect of muscle force generation variability on kinematic variability, especially during slow movements. The model includes motor unit pool dynamics with Hill-based activation and contraction dynamics for each motor unit. This structure makes it possible to use the model for estimating both force and kinematic variability in isometric and non-isometric conditions. Results in this paper are limited to the isometric case and are found to be in agreement with experimental force variability characteristics reported in the literature.

V. Acknowledgements

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