Using a new muscle activation model to improve the stability of force estimation with Hill-type models over different activation levels

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Abstract. Hill-type models are attractive in biomechanics simulation due to their low computational cost and commonly measured parameters. They mainly consist of two parts, the activation dynamics, and the contraction dynamics. Since Hill-type models are phenomenological models, it is meant to capture the process's main characteristics using a descriptive system that approximates the behaviour of the real physical one. This paper developed a novel activation dynamics formulation to estimate the force of the triceps surae muscles at two different contraction levels without retuning the model parameters. The new formulation simulates the cell calcium dynamics as well as the recently discovered role of store-operated calcium entry (SOCE) channels. The proposed model reflects the main characteristics required from the activation model, which are the electro-mechanical delay, the process nonlinearity, and the ability to couple the activation process with some parameters from the subsequent force-generating process. The parameters of the model are task-independent to closely approximate the muscle function and to have the ability to simulate different movements adequately. The model shows a more stable performance along with different contraction levels in comparison with two other activation models.

1. Introduction

Biological systems are complex systems, they are consisting of different interrelated elements. Most of these relations are nonlinear and, as a result, properties do not obviously arise from the properties of individual elements. This complexity imposes many challenges in modelling such systems [1]. One of the most important challenges is determining the model structure. A too simple model can lead to misrepresenting some important physiological properties and consequently inaccurate system behaviour. On the other hand, a too complex model can result in missing the big picture and getting lost in detail such as parameter identification. Another problem that arises when dealing with such systems is that our models should evolve continuously to answer the newly raised questions and keep up with our increasing understanding of the system. This means that modelling of the biological systems is a side adventure of experimental work [2].

When it comes to muscle modelling, there are three main categories each of them represents a different modelling direction. The three categories are the simple second-order models, the Huxley-based distributed-parameter models, and the Hill-based lumped-parameter models [2]. The first approach deals with the system as a "black box". These models try to map the input and the output without representing the system elements. Therefore, this type has severe limitations when it
comes to model generality. Since the parameters' value of these models changes as a function of the task or even in different ranges of the same task. Also, these models cannot be used to reveal interrelation between the system elements.

The second category is the Huxley-based models; also known as the physiological or microscopic models. These models try to identify and then model the actual contractile mechanism. These models are so complex and even modified to involve more rate functions and activation-deactivation states. This led to two main challenges. First, the high computational cost even when simulating rather simple movements. Second, the usage of many parameters that are hard to be identified.

The third category is Hill-based models. These models are based on the structural model of A.V. Hill (1938). They are phenomenological models. Therefore, the system is simplified and described using representative elements that approximate, under some assumptions, the behaviour of the distributed system. They are lumped parameter models since they overcome the complexity of the spatial distribution and simplify the system equations from partial differential equations like in Huxley-type models to ordinary differential equations. Both Hill and Huxley models back experimental studies. But when it comes to synthesizing complex motion, Hill-type models of muscle are often used. Due to its low computational cost even for a multiple-muscle model, and few easily defined parameters [2].

Hill-based models consist mainly of two parts, the activation dynamics, and the contraction dynamics. While the activation dynamics formulation simulates the process of force initiation or the muscle fibres' response to the nerve stimulation, the contraction dynamics formulation simulates the force developing within the muscle fibres [3].

The common configuration of the contraction dynamics part is a contractile element (CE) representing active muscle fibre and describes the force-length, force-activity, and the force-velocity (hyperbolic relation mostly known as the Hill's relation) characteristics phenomenologically. The CE comes in series with a viscoelastic element (SEE) which represents the tendon and other material like aponeuroses. While the passive tissues acting in parallel to the active muscle fibre are represented by the parallel elastic element (PEE).

On the other hand, the main characteristics the activation dynamics part should reflect are the electromechanical delay, the process nonlinearity, and the ability to couple the activation process with some parameters from the subsequent force-generating process. But, in contrast to the contraction dynamics, the activation dynamics formulations used in Hill-type models are "Black Box" formulations where only the input/output behaviour is considered without representing the system element and its dynamics. This led to the usual issues regarding this kind of model.

According to a survey conducted by [4], there are two categories of muscle activation models based on the features they describe. The first category aims to reflect the linearity or nonlinearity of the system. This property has been detected during the study of isometric electromyographic signal (EMG)-force relationships for different muscles and many studies have been conducted to identify its causes. The second category aims to capture the electromechanical delay (EMD) of the process. EMD is defined as the time lag between the onset of electrical activation of the muscle and the onset of force production. These models consider the EMD phenomena at the muscle activation as well as the deactivation process, making them two-parameter models. However, to count for the system's nonlinearity and the EMD, one should use two models, one from each category.

2. The Mathematical Model

In this manuscript, we used a new activation dynamics formulation modified by the authors to estimate the force of the triceps surae muscles at two different contraction levels without retuning the model parameters. The model is used along with the Hill-type contraction model. This aims to improve the accuracy and stability of force estimation for human movements by adding some important physiological insights into Hill-type models.

2.1. Activation dynamics part

2.1.1. Classical Ca-model. The original model [5] is two coupled ordinary differential equations that describe the calcium kinetics between its sarcoplasmic reticulum (SR) stores, and muscle protein
filaments. The equations are formulated based on the principle of mass action and uses four parameters \( k_1 - k_4 \) to represent the rates at which the \( Ca^{2+} \) ions bound/unbound to the sarcoplasmic reticulum or protein filament.

\[
\frac{dCa}{dt} = (k_4 Ca_f - k_3 Ca)(1 - Ca_f) + \left\{ \begin{array}{ll} k_1 (C - Ca - Ca_f) & \text{stimulus on} \\ k_2 Ca (C - S - Ca - Ca_f) & \text{stimulus off} \end{array} \right.
\] (1)

\[
\frac{dCa_f}{dt} = -(k_4 Ca_f - k_3 Ca)(1 - Ca_f)
\] (2)

Where \( Ca \) and \( Ca_f \) are the non-dimensional concentration of \( Ca^{2+} \) when it is free and when it binds to filaments, respectively. The constants \( C \) and \( S \) denote the non-dimensional total concentrations of \( Ca^{2+} \) and sarcoplasmic-reticular binding sites, respectively.

Yet this model faces two major problems. First, EMG signals which are the driving force for calcium dynamics are not included. The calcium dynamics in this model relies on the activation period without taking into consideration the magnitude of the signal. The model, therefore, is not EMG-Driven. Secondly, the model assumes a total separation between the calcium release from SR and absorption rate constants. This means that the ions/SR interaction process is a one-direction process (release or reuptake) and cannot happen in both directions simultaneously. This contradicts with recent biological observations [6,7].

2.1.2. Modified Ca-model. A modified model developed by the authors overcomes these issues.

\[
\frac{dCa}{dt} = (k_4 Ca_f - k_3 Ca)(1 - Ca_f) + \varepsilon [k_1 (C - Ca - Ca_f) - k_2 Ca(C - S - Ca - Ca_f)]
\] (3)

\[
\frac{dCa_f}{dt} = -(k_4 Ca_f - k_3 Ca)(1 - Ca_f)
\] (4)

where \( \varepsilon \) is the rate of change of the excitation signals i.e., EMG signals.

The model introduced two major changes to the classical one. First, the model presented the EMG signals as a driving force for the calcium dynamics from/into the SR. This happened by integrating the parameter \( \varepsilon \) into the system, which represents the rate of change of the EMG signals. Electromyographic signals (EMG) measure muscle response or electrical activity in response to a nerve’s stimulation of the muscle. This response or electrical activity leads to the release of stored calcium from the sarcoplasmic reticulum (SR) and consequently triggers muscle contraction. Since \( k_1, k_2 \) are the rate constants of calcium release and reuptake we chose the rate of the EMG signal \( \varepsilon \) to represents the muscle electrical activity. The second, it dropped the pair-wise relation between the rate constant of calcium release and reuptake \( (k_1, k_2) \), this allows for simulating the interactions between the calcium channels. This means that the model allows for concurrently bi-direction relation between the \( Ca^{2+} \) ions and its sarcoplasmic reticulum stores.

Moreover, the model indicates an interaction between the rate of calcium reuptake and the EMG signals, a relationship that has recently been proven and has contributed to a reshaping of our view of the role of EMG signals [6].

2.2. Contraction dynamics part

The modified activation dynamic model works with any Hill-type contraction model. in this paper we will use it along with the contraction model that is widely used in several published articles. it is an updated version of Zajac’s model [8], with parallel elastic and damping components, which are primarily intended to improve numerical stability [9]. See Figure (1).
Figure 1. Hill-type contraction model. PEE is the parallel elastic element, PDE is the parallel damping element, and CE is the contractile element. $L_{MT}$ is the musculotendon length, $L^M$ is the muscle length, $L^T$ is the tendon length, and $\alpha$ is the pennation.

3. Case study: Triceps Surae Muscles Force Estimation

The model is used to estimate the plantarflexion isometric torque with the ankle at 90° degrees, knee extended. EMG data has been collected for each muscle of the triceps surae group i.e., gastrocnemius medialis (GM), gastrocnemius lateralis (GL), and soleus (SOL). Synchronously, the ankle joint torque is recorded from a dynamometer. A maximum voluntary contraction (MVC) is first performed for the normalization, then data has been collected at submaximal levels i.e., 20% and 60% MVC. All the data are from [9]. EMG signals are then filtered, full-wave rectified, enveloped, and then normalized as [10]. These normalized surface EMG data are then numerically differentiated with respect to time using the Matlab® program. The rates of data are used as input to the muscle activation model.

For each muscle, a system of ordinary differential equations represents the activation dynamics, the contraction dynamics, and an auxiliary equation for the muscle velocity is used. This system consists of the proposed activation dynamics equations 3, 4 combined with

$$\frac{dF_T}{dt} = K^T (v^{MT} - v^M \cos \alpha)$$

$$\frac{dl^M}{dt} = v^M$$

where $F_T$ is the tendon force, $K^T$ is the tendon stiffness, $v^{MT}$ is the musculotendon velocity, $v^M$ is the muscle contraction velocity, $\alpha$ is the pennation angle and $L^M$ is the muscle length.

The system’s three-state variables are Caf, $F^T$ and $L^M$, while its control input is $\epsilon$. For isometric contraction, musculotendon length $L^{MT}$ is constant, so musculotendon velocity $v^{MT}$ is set to zero. The Musculoskeletal parameters are from [9]. An open-source MATLAB® graphical user interface (GUI) under public license has been used to numerically integrate the equations [9]. Each muscle force is then multiplied by its moment arm to give its contribution to the joint torque. The total estimated torque is then compared to the measured one to calculate the RMS error.

The joint torque is also estimated using two other activation models. The first formulation (Linear Model) is designed to detect the electromechanical delay within the process using two parameters [4].

$$\frac{du}{dt} = (e - u) \ast (t_1 \ast e + t_2)$$

Where $(u)$ is the neural activation, $(e)$ the excitation signals i.e., processed EMG signals, and $t_1$ and $t_2$ are constants depend on the activation and deactivation time.
The second model (A-Model) is a modification to the latter. It is an algebraic piecewise function which has been added to capture the nonlinearity in the system [11].

\[
a(t) = \begin{cases} 
\gamma \ln(\beta u(t) + 1) & 0 \leq u(t) \leq u_0 \\
mu u(t) + c & u_0 \leq u(t) \leq 1
\end{cases}
\]

(8)

Where \(u\) is the neural activation from eq. (7), \(a\) is the muscle activation, and \(u_0, \mu, c, \beta\) and \(\gamma\) are constants and their values can be determined from the single parameter.

4. Results and Discussion

Outcomes in Table 1 show the Normalized Root Mean Square Error (%RMS) between the measured and estimated torque by each model at both 20% and 60% MVC. The results prove that the proposed Ca-Model and A-Model improve the error at 20% MVC by 34.7% and 34.6%, respectively, compared with the linear model (the linear model considered as the reference). Hence, the result of the new method is in better agreement with the measured torque at 20% MVC than the results of the A-model. Remarkably, the new method provides a considerably better fit with the measured results at 60% MVC torque than both the linear model and A-model. Precisely, the proposed model shows 50.8%, while the A-Model shows only 21.8%, an increase in accuracy compared with the linear model.

Table 1. %RMS errors between the measured and estimated results in 20% and 60% of MVC.

|       | Linear Model | Ca-Model | A-Model |
|-------|--------------|----------|---------|
| 20% MVC | 7.48         | 4.88     | 4.89    |
| 60% MVC | 19.88        | 9.77     | 15.55   |

5. Conclusions

A new muscle activation model has been proposed in this paper. The model has simulated the cell calcium dynamics and has presented the role of store-operated calcium entry (SOCE) channels. Using this model, the activation dynamics of the triceps surae muscles has been successfully simulated. The numerical simulations show that the model has improved the accuracy of force estimation for Hill-type models over different activation levels without the need to readjust the parameters. Hence, a more accurate representation of muscle properties interactions has been recognized. Finally, this work proves that more physiological insights are key for improving Hill-type models.

6. References

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