Enhancement of physiological and mechanical modelling of the skeletal muscle controlled by Functional Electrical Stimulation

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Abstract

This work establishes a mathematical muscle model which describes the complex physiological system of the skeletal muscle based on the macroscopic Hill and microscopic Huxley concepts. A new study published by Sorine opened the possibility of controlling the heart muscle model by using a chemical control input to stimulate the contractile element of the model. Starting with this concept, we have proposed a new physiological and mechanical skeletal muscle model where we take into account the muscular masses and the viscous frictions caused by the blood and the muscle tendon connections. This model is presented under differential equations where the input is the electrical signal provided from the stimulator and the outputs are the muscle force and stiffness.

1 Introduction

Skeletal muscle modelling as the actuator propellant of the animal motion presents a special challenge to the bio-mathematicians not only because the behaviour of this structure is highly complex and nonlinear, but also because any successful mathematical model of skeletal muscle must be able to predict a wide variety of experimentally established phenomena.

The main issues concern the muscle modelling and its applications such as controlling the human movement of a paralysed limb using Functional Electrical Stimulation (FES). Starting with the last version of the muscle modelling [5][7], we have proposed a new physiological skeletal muscle model by adding the muscular mass and its effect. This model leads us to define the signal input and to characterize the parameters of the muscle model presented by differential equations form where the outputs are the muscle force and stiffness. The input model represents the actual electrical signal as provided by the stimulator "PROSTIM" or the SUAW implant [6] offering the possibility of tuning three independent parameters: amplitude, pulse width and frequency [6][7].

Our muscle model is composed of two parts: activation model and mechanical model, shown on figure 1 [7].

The first one depends on the parameters of the stimulation intensity, pulse width and frequency, but also on phenomenon such as fatigue and calcium dynamics. The second part deals with the mechanical behaviour and is based on the work of Hill [1], Huxley [2], Zahalak [3] and Sorine [4].

1.1 Mechanical muscle model

Our model represents the original form of skeletal muscle. We have a contractile element $E_c$ controlled by two variables ($\alpha, u$) in series on each side with two elastic elements $E_{s1}$ and $E_{s2}$ that represent the muscle tendon, figure 2.

Let us suppose that all the elements of the models are without masses, then, we introduce the muscle mass between the elastic and contractile element on both sides where the two masses are equal ($m_1=m_2$). The muscular masses move in a viscous medium then, we introduce viscous friction forces in parallel with the elastic elements. A parallel elastic element $E_p$ is often added to account for the resistance of passive muscle to stretch [5].
1.2 Activation model

The activation model is composed of two models, fibre recruitment model and chemical control generator. The recruitment depends on the amplitude current and the pulse width of the stimulus signal. The larger the intensity or pulse width is, the higher the number of recruited motor units is: less excitable and deeper fibres are then excited. The chemical control input $u$ results from the electrical activity in muscle fibre including the calcium dynamics and time delay of the action potential propagation.

2 Methods

2.1 Mathematical equations of the mechanical muscle models

From [7] and the dynamic relation between the springs we can write the following differential equations:

$$
\begin{align*}
    \ddot{\epsilon}_r &= (s_a a \sigma - s_a \sigma) u + (b k_s - s, a a \sigma) \dot{\epsilon}_r \\
    \dot{\epsilon}_r &= a \dot{\epsilon}_r - p r \dot{\epsilon}_r - q \epsilon_r \\
    \dot{\epsilon}_r &= a \epsilon_r - a_r \epsilon_r - a_r \epsilon_r
\end{align*}
$$

The parameters of the model are given by:

$$
\begin{align*}
    a &= \frac{L_0}{L_{c0}} ; \quad b = \frac{L_0}{A_0} ; \quad r = \frac{A_0}{m L_{c0}} ; \quad \dot{r} = \frac{A_0}{m L_{c0}} \\
    a &= \frac{L_0}{L_{c0}} ; \quad b = \frac{L_0}{A_0} ; \quad p = \frac{\lambda_1}{m_1} ; \quad q_i = \frac{k_i}{m_2} \\
    a &= \frac{L_0}{L_{c0}} ; \quad b = \frac{L_0}{A_0} ; \quad p = \frac{\lambda_2}{m_2} ; \quad q_i = \frac{k_i}{m_2} \\

S_1, S_2 and S_r represent the sign of the chemical control and the velocity of the contractile element. The signs introduce a non linear complexity in the mathematical equations of the muscle model.

$A_0$ represents the value of the cross sectional area. $m_1$ and $m_2$ represent the muscle masses. $\lambda_1$ and $\lambda_2$ represent the parameters of the viscous friction of each side. $L_{c1}, L_{c2}, L_c$ and $L$ are the lengths of elastic, contractile and parallel elements [7]. The relative deformation of elastic, contractile and parallel elements are given by:

$$
\begin{align*}
    \epsilon_i &= \frac{L_0 - L_{c0}}{L_{c0}} , \quad \epsilon_1 = \frac{L_{c1} - L_{c0}}{L_{c0}} , \quad \epsilon_2 = \frac{L_{c2} - L_{c0}}{L_{c0}} , \quad \epsilon = \frac{L - L_0}{L_0}
\end{align*}
$$

2.2 Fibre recruitment model

The aim of the fibre recruitment model is to model the number of fibre recruited in each impulse. This model depends on the amplitude current and the pulse width of the stimulus. We used a non linear function (2) "sigmoid function" to represent this effect see figure 3.

$$
(2) \quad r(d) = A_0 e^{-k_s (d - d_0)} + e^{-k_r (d - d_0)} + C_r
$$

Where $d$ represents the product of the amplitude current I by the pulse width $PW$. $A_0, C_0, k_0, d_0$ four parameters determines the characteristic of the curve.

2.3 Chemical control generator

The muscle contraction results directly from the high intracellular calcium concentration in muscle cell. In [4], Bestel introduces a model of the heart with chemical control input connected to the calcium dynamics. Based on this hypothesis, we propose a model that connects the fibre electrical activity started by the signal stimulation and the chemical control input $u$ of the mechanical muscle contraction at the microscopic scale (sliding Actin - Myosin filament). This model depends on different components, the generator of $u$ resulting from the calcium dynamics and the calcium concentration $[Ca^{2+}]$ resulting from the action potential on the fibre. Figure 4 shows the link between the different components [4, 7].

Figure 3: Fibre recruitment model for artificial stimulated muscle

Figure 4: The link between stimulation signal, calcium concentration $[Ca^{2+}]$ and the chemical control $u$. 
3 Simulation and results
The proposed muscle model is evaluated by analysing and comparing simulation results with data from literature in first step and from our own measurements in the next step. We will focus on the behaviour of the muscle model in isometric mode when the constraint developed by the muscle involves no displacement.

We have implemented this model in MATLAB/SIMULINK. The parameters used in simulation are issued from the literature presented in table 1. The model describes sufficiently the influence of the recruitment model characterized by the two parameters: amplitude and pulse width of the signal stimulation, figure 5. The model also allows accurate prediction of the force-frequency characteristic during stimulation at different frequency levels, figure 6.

![Figure 5: Simulated forces response of the contractile element at fixed frequency with amplitude I and pulse width PW.](image)

![Figure 6: Simulated force response at fixed pulse trains with various frequency. (a) Force response on the boundary of muscle, (b) Force response of the contractile element (between masses).](image)

4 Discussion and Conclusions
The objective of this on-going study is to develop an experimentally estimated muscle model of the human bodies, which can be used during the restoration of movement of paralysed limbs through FES. The first set of simulations agrees with the data found in the literature. The new approach provides a macroscopic model based on the microscopic physiological phenomenon and thus can be linked to physiological measurements. The future work will concern the identification protocols based on an experimental measurement carried out on an animal isolated muscle. The next is to perform the identification on paraplegic patients using a measuring chair, developed in our lab, that can run in different modes: isometric, isotonic, isokinetic contractions of the studied muscle.

Based on this concept of modelling, we proposed several families of model adapted to applications - isolated muscle, isometric contraction, muscle contraction in a biomechanical model (hip, knee, ankle...) – optimising the compromise between complexity and accuracy. Besides, the structure of our model allows including new boxes such as muscle fatigue and detailed Calcium dynamics.

In a short term, this model will be used to perform simulations, to synthesize stimulation pattern for open loop movement generation under FES, and in a long term to develop control strategies to achieve balanced standing for instance.

References