

A musculotendon model including muscle fatigue

Jong - kwang Lim and Moon - hyon Nam

Agency for Defense Development
P.O.BOX 35-1, Yousoung, Daejeon, 305-600, Republic of Korea
Tel: +82-42-821-3181, Fax: +82-42-821-2221-137342, Email: ljk0305@chollian.net
and
Department of Electrical Engineering
Kon-Kuk University
93-1, Mojin-Dong, Kwangjin-Gu, Seoul, 143-701, Republic of Korea
Tel: +82-2-450-3482, Fax: +82-2-454-0428, Email: monroe@kkucc.konkuk.ac.kr

Abstract

A musculotendon model is investigated to show muscle fatigue under the repeated functional electrical stimulation (FES). The normalized Hill-type model can predict the decline in muscle force. It consists of nonlinear activation and contraction dynamics including physiological concepts of muscle fatigue. A muscle fatigue as a function of the intracellular acidification, pH_i is inserted into contraction dynamics to estimate the force decline. The computer simulation shows that muscle force declines in stimulation time and the change in the estimate of the optimal fiber length has an effect only on muscle time constant not on the steady-state tetanic force.

1. Introduction

Muscle fatigue defined by the decline in muscle force must be minimized to maintain mobility of paraplegics as long as possible. A musculotendon model with muscle fatigue is investigated to predict muscle force in time and its adoption under the change in the estimate of the optimal fiber length. By using this model we hope to find the optimal stimulation patterns in the future.

2. Musculotendon Model Under FES

A normalized musculotendon model including muscle fatigue is shown in Figure 1. When applying FES to limbs the stimulated muscles across a joint are contracted. The muscle force generated by muscle contraction moves limbs to flex or extend. Then the

muscle length is changed in accordance with limbs movement. The changes in muscle length and force are normalized by the optimal fibre length, l_{m0} , and the maximum isometric force, F_0 , respectively.

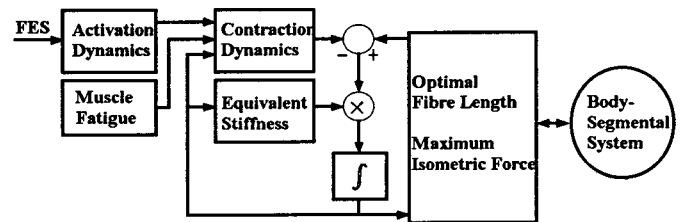


Figure 1. A normalized musculotendon model including muscle fatigue as a function of pH_i .

Activation Dynamics

The activation is a series of processes of the depolarization of muscle membrane by FES input $u(t)$, Ca^{2+} diffusions from T-tubules to sarcoplasmic reticulum (SR), the coupling Ca^{2+} with troponins ($Ca^{2+}Tn$) and Ca^{2+} uptake.^[1] By stimulating the contact parts between nerve terminals and fibre end plates we assume there are no any failures of neuromuscular transmissions, membrane depolarizations, and Ca^{2+} diffusions.

By the application of $u(t)$ the transients^[1] of Ca^{2+} concentrations of in SR and $Ca^{2+}Tn$, $m(t)$ and $n(t)$ are described by

$$\frac{dm(t)}{dt} = \frac{1}{C_m} (k_f m_0 u(t) - k_b m(t)) \quad (1)$$

$$\frac{dn(t)}{dt} = \frac{k_n}{C_n} (F(m(t)) - G(n(t))).$$

Where $F(m(t))$ and $G(n(t))$, the rates of coupling and decoupling between Ca^{2+} and troponin are given by

$$F(m(t)) = \frac{m^2(t)}{m^2(t) + M} \quad (2)$$

$$G(n(t)) = n(t)(g_1 + g_2 n(t) + g_3 n^2(t) + g_4 n^3(t)).$$

Assuming that Ca^{2+} Tn activate contractile elements without any loss muscle activation in fresh state can be expressed as

$$a(t) = n(t), \quad 0 \leq a(t) \leq 1. \quad (3)$$

Contraction Dynamics

Contraction dynamics describing mechanical activity of muscle can be expressed using the modified force - shortening velocity relationship^[2] to satisfy the relation,

$\tilde{F}(l_m) = (F(l_m)/F_0) = 1$. By normalizing^[3] for the optimal fibre length ($l_{m0} = l_m / \tilde{l}_m$), the maximum shortening velocity ($v_{max} = v_t(t) / \tilde{v}_t(t)$), and the maximum isometric force $F_0 = F(t) / \tilde{F}(t)$ it is calculated by

$$\tilde{v}_m(t) = \begin{cases} \frac{a_f(t) - \tilde{F}(t)}{a_f(t) + a_1 \tilde{F}(t)}, & 1 \geq \frac{\tilde{F}(t)}{a_f(t)} \\ \frac{a_f(t) - \tilde{F}(t)}{a_2(a_f(t) - a_3 \tilde{F}(t))}, & 1 < \frac{\tilde{F}(t)}{a_f(t)} \leq \tilde{F}_{max} \end{cases} \quad (4)$$

where $\tilde{v}_m(t)$ is the change of the normalized muscle length due to limb movement. The muscle fatigue is included as a term $a_f(t)$ ^[4]. The equation (5) expresses the lengthening velocity^[5] by external force where \tilde{F}_{max} is the maximum force that muscle can exert, $F_{max}/F_0 = 1.8$ ^[3].

Finally muscle force is estimated as

$$\frac{d\tilde{F}(t)}{dt} = \tilde{k}_{eq} [\tilde{v}_{mt}(t) - \tilde{v}_m(t)] \quad (6)$$

where \tilde{k}_{eq} is the equivalent stiffness^[2] and is calculated as a function of muscle force with the equation (7).

$$\frac{1}{\tilde{k}_{eq}} = \frac{1}{\tilde{k}_s} + \frac{1}{\tilde{k}_t}$$

$$\tilde{k}_s = \frac{\sigma_s + \tilde{F}(t)}{\epsilon_s}$$

$$\tilde{k}_t = \begin{cases} \frac{(\sigma_{t1}/\sigma_{t0}) + \tilde{F}(t)}{\epsilon_{t1} \tilde{l}_t}, & \epsilon_t \leq \epsilon_{tt} \\ \frac{E_t}{\sigma_{t0} \tilde{l}_t}, & \epsilon_t > \epsilon_{tt} \end{cases} \quad (7)$$

$$\epsilon_t = (l_t/l_{t0}) - 1 = \epsilon_{t1} \ln[1 + (\sigma_{t0}/\sigma_{t1}) \tilde{F}(t)]$$

\tilde{k}_s is the stiffness of the series element of muscle. \tilde{k}_t is the tendon stiffness. It is exponentially increasing when

tendon strain ϵ_t is less than ϵ_{tt} and is proportionally increasing otherwise^[3]. l_{ts} is the slack length of tendon.

Muscle Fatigue

Muscle fatigue due to the repeated stimulations has the linear relation of the decline of pH_i ^[6]. For the fresh and fatigued muscle pH_i is 7.03 and 6.51, respectively.^[7] The fitted muscle fatigue profile^[4] can be obtained by

$$pH(t) = p_1 \tanh[p_2(t - p_3)] + p_4 \quad (8)$$

$$\tilde{F}(pH(t)) = \frac{p_5 + p_6 pH(t)}{p_5 + p_6 pH(0)} \quad (9)$$

Let $a_f(t)$ in contraction dynamics set as

$$a_f(t) = a(t) \tilde{F}(pH(t)). \quad (10)$$

3. Computer Simulation

Computer simulation shows the capability of model to predict the decline in muscle force. With knee joint fixed at the specific position we consider the situation that FES is applying to rectus femoris of quadriceps muscle through surface electrode.

Method

By changing the frequency of stimulation pulse train $u(t)$ to 1Hz(twitch), 5Hz, and 20Hz(tetanic contractions) $\tilde{F}(t)$ are calculated. The length of muscle in isometric condition is constant and therefore its derivative \tilde{v}_m becomes to zero. Let the changes in the estimates of the optimal fibre length are the $\pm 32\%$ of maximum ($l_{m0} \pm 1 \sigma$). There are no the corresponding change in the maximum isometric force shown as equations (4) to (7). Table 1 shows the constants used in simulation.

Table 1. Constants in simulation(not shown their units)

activation dynamics	$C_m = C_n = 1, m_0 = 1000$ $k_f = 10, k_b = 90, k_n = 52, M = 500$ $g_1 = 0.081, g_2 = 1.936,$ $g_3 = -7.638, g_4 = 7.406$
contraction dynamics	$a_1 = 4, a_2 = 14.479, a_3 = 0.524,$ $\sigma_{t0} = 32 \times 10^6, \sigma_{t1} = 11.4 \times 10^6,$ $E_t = 1.2 \times 10^6$ $\epsilon_{tt} = 0.02, \epsilon_{t1} = 0.023$
muscle fatigue	$p_1 = -0.260, p_2 = 0.065,$ $p_3 = 58.321, p_4 = 6.768,$ $p_5 = -66.692, p_6 = 10.948$
muscle characteristics	$l_{m0} = 0.082, l_{ts} = 0.411$

Results

During muscle fatigue the time course of pH_i is typically shown as Figure 2.

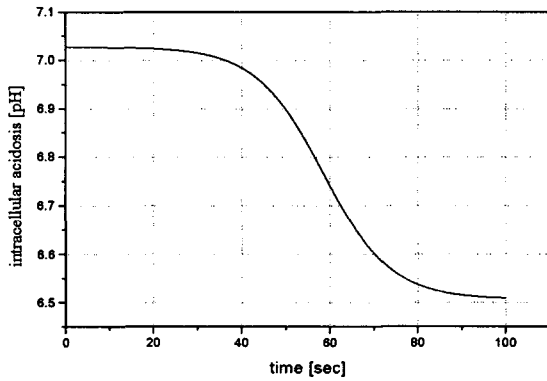


Figure 2. The intracellular acidosis is decreased during muscle fatigue.

The twitch (1Hz) and tetanic responses (5 and 20Hz) are shown in Figure 3. The twitch and tetani decline rapidly after 50[sec] and approach 35% and 27% of the maximum force in fresh after 90[sec].

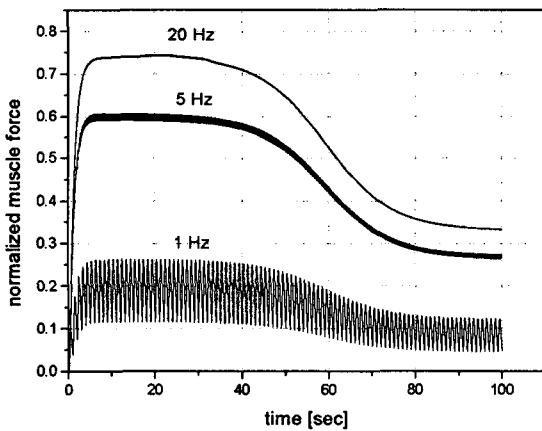
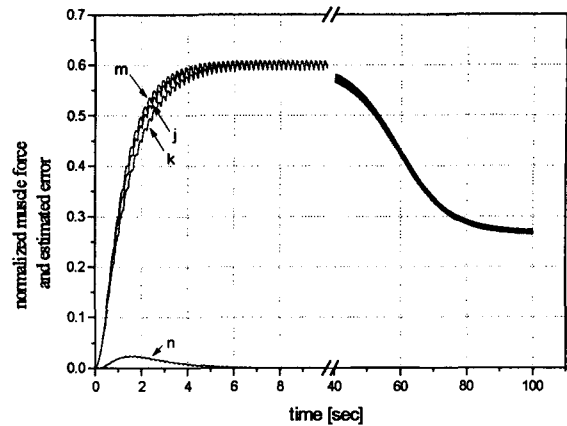
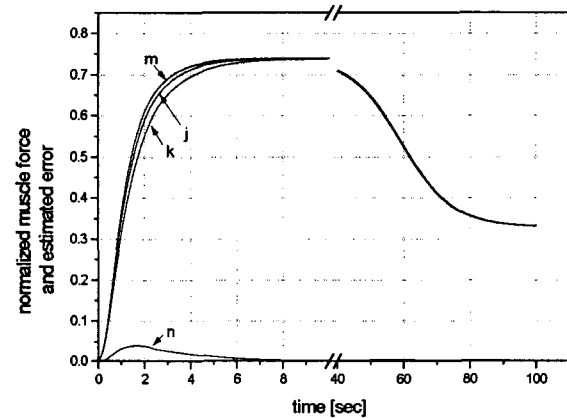


Figure 3. The twitch and tetanic force are declined during muscle fatigue.

Figure 4 shows that the effects of the change in the estimated optimal fibre length on twitch and tetani under the repeated 5Hz and 20Hz stimulation. We can see that the more change in the positive direction, the more rise muscle force. The changes of the estimated optimal fibre length affect the equivalent stiffness to change rising time of muscle force but its tetanic amplitude. The same result is presented by Zajac(1989)^[3].



(a) 5Hz stimulation



(b) 20Hz stimulation

Figure 4. The change of optimal fibre length under 20 Hz stimulation affects only the rising phase of force, not steady tetanic force. (without (j), negative 32% (k) or positive 32% (m) changes of l_{m0} and average absolute estimated error, $n=(j-k)+(j-m)/2$.)

4. Discussion

Muscle fatigue term in contraction dynamics represents the physiological fact that cycling rate of cross bridges^[8] decline in accordance with force during muscle fatigue. However it is lack of expressing fatigue dynamics in time. Since the cycling rate of cross bridges is equal to the coupling and decoupling rates of Ca^{2+} with troponins under the lossless contractions, the decline in force can be described by the modified activation dynamics. By replacing muscle fatigue term to the changes of the cycling rate of cross bridges or the rate of Ca^{2+} uptake muscle fatigue dynamics will be studied.

5. Conclusions

A musculotendon model including muscle fatigue can predict the force decline during muscle fatigue. Also its steady force level is not affected by the change of the estimated optimal fibre length.

6. References

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