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13. Further Observations on the Relationship of EMG and Muscle Force*

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Human skeletal muscle may be regarded as an electro-mechanical transducer. Its physiological input is a neural signal originating at the alpha motoneurons in the spinal cord and its output is force and muscle contraction, these both being dependent on the external load.

In this paper some experimental data taken during voluntary efforts around the ankle joint and by direct electrical stimulation of the nerve are described, Some of these experiments are simulated by an analog model, the input of which is recorded physiological soleus muscle EMG. The output is simulated foot torque. Limitations of a linear model and effect of some nonlinearities are discussed.

INTRODUCTION

Modeling of biological systems requires us to face three most challenging problems. First, we sometimes do not know and often cannot measure the inputs to the real system we are modeling. Second, it is usually extremely difficult, if not impossible, to measure all state variables, and third, such systems almost inevitably contain significant nonlinearities which cannot be avoided by common analytical devices of piecewise linearization or small signal analysis. In the following paper we wish to discuss some models of muscle contraction in normal humans, and show how we have attempted to resolve or avoid these difficulties.

Human skeletal muscle may be regarded as an electro-mechanical transducer, converting the electrical signals from the alpha motor-neurons into a mechanical force at its origin and insertion. For all practical purposes we cannot measure this neural input. Associated with muscle activation is the electrical activity of the contracting muscle itself, which we may record as the electro-

myogram (EMG). There is a one-to-one relationship between an action potential in a single motor nerve and the EMG of its muscle fibers. Using surface electrodes on the belly of the muscle, however, we get only a single scalar measure from the many motor units which fire in varying spacial and temporal patterns.

Nevertheless, there is some theoretical justification for expecting the average rectified EMG (refs. 1 and 2) to be proportional to the level of innervation and we have used this signal (ref. 3) as our model's input.

The only other variables we can measure are muscle force and length. For simplicity we have fixed muscle length and measured only the isometric force (actually torque) generated by the gastrocnemius-soleus muscles (GSM) at the ankle joint. This avoids those nonlinearities in both the EMG measurement and in the mechanical parameters which are associated with changing muscle length.

One of the intrinsic state variables of the muscle which we cannot measure is termed the "active state" (ref. 4). This rather loosely used term describes two conceptually distinguishable changes, one the generation of an active force of contraction and the other an increase in the passive

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mechanical impedance of the muscle. Measurements of both these phenomena have been made in animals (ref. *5*) but only the impedance changes have been measured in man (refs. **6** and **7**).

LINEAR MODEL

At the Sixth Annual NASA-University conference we presented a linear model for the EMG-force relationship ((ref. 8), see also (ref. **9**)). The topology of this model is shown in figure **1**. For the linear model, B, K_1, and K_2 are constant, passive elements and activation of the muscle implies generation of an active force F_m.

The model transfer function is given by

$$\frac{F}{\text{EMG}} = K \cdot G_1(S) \cdot G_2(S),$$

where

$$G_i(S) = \frac{1}{T_i S + 1}, \; i = 1, \, 2.$$

Separation of the transfer function like this allows us to tentatively designate $G_1(S)$ as the active state component while $G_2(S)$ may be considered the linearized mechanical component of the muscle model shown in figure **1**. The input is recorded physiological EMG data. For our simulation the model parameters were chosen as $T_1 = 0.105$ sec, $T_2 = 0.105$ sec, and $K = 0.25$ to *0.35*. Figure 2 shows muscle and model response to a rapid contraction. Figure *3* shows the impulse response of this linear model. The "impulse response" of the muscle was obtained by delivering a single maximal shock to its motor nerve.
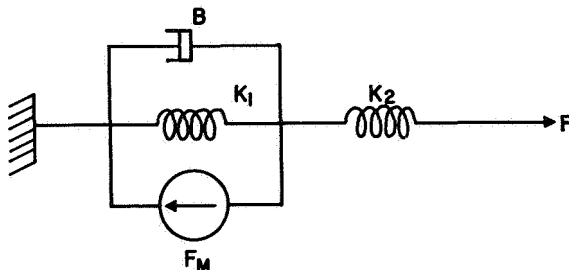

FIGURE 1.—A mechanical model of skeletal muscle. The series and parallel elastic elements (K_2 and K_1) and the viscosity B are defined only by the mechanical behavior of the muscle and should not imply a mechanism. The force generator F_m represents the contractile "active state" of the muscle.
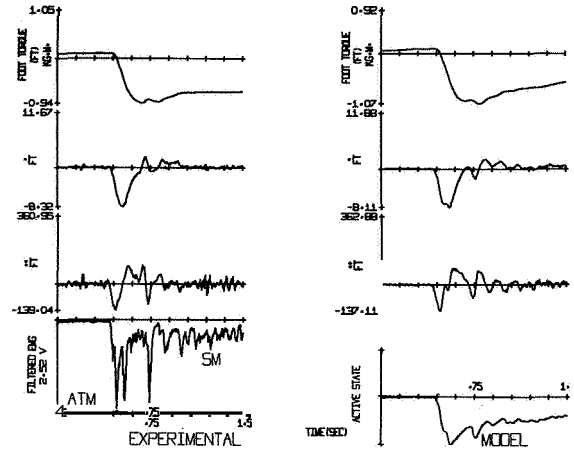

FIGURE 2.—Left: subject's step torque response and EMG. Right: model response to subject's EMG. Instead of replotting EMG on the right, the output of the active state stage G_1 is the fourth curve plotted.
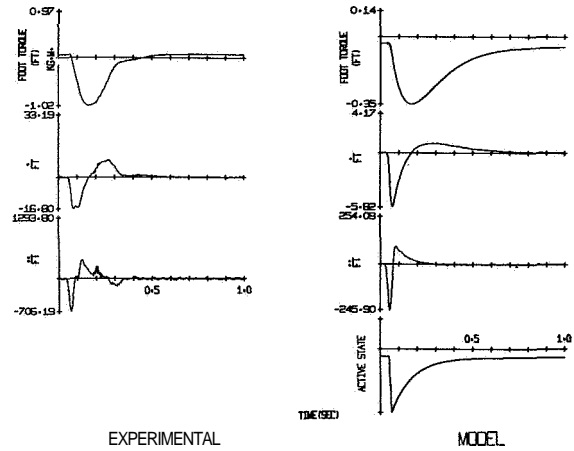

FIGURE 3.—Left: foot twitch in response to a single supramaximal shock to the tibial nerve. Right: model impulse response and the output of the active state stage G_1.

Figure 2 shows that the model is reasonably successful in mimicking rapid muscle contraction. The output of the first stage of the model is plotted as the active state in the lower part of the figure. However, it was shown in an earlier paper (ref. 8) that these time constants are too long to reproduce a sudden relaxation. Reducing one time constant ($T_2 = 0.01$) improves the rate of fall of the force (see fig. 8 of ref. 8). It is clear that a constant coefficient linear model is not adequate to reproduce both contraction and relaxation.

The impulse response in figure **3** shows three interesting deficiencies. The negative peak in the experimental acceleration curve at **300** msec is not due to intrinsic muscle activity but is produced by the stretch reflex loop causing reactivation of the muscle as it relaxes and returns to its resting length.

The second error is the apparent saturation of the velocity curve and the third is the double hump of the acceleration curve between **100** to **250** msec. The active state variable does behave much like measurements in animal preparations indicate it should. It rises abruptly and falls slowly but does not remain on a plateau at its peak value.

NONLINEAR MODEL

The nonlinear model incorporates two limiter type nonlinearities in order to improve the impulse response of the model and its performance with soleus EMG as the input signal. A block diagram of the analog model is shown in figure **4.** The first limiter N_1 is situated after the first stage to limit amplitude of the active state thus producing a plateau at its peak value. The second limiter N_2 is situated in the second stage to limit the maximum rate of change of foot torque. Figure **5** shows the effect of the active state limiter and figure **6** shows the effect of the velocity limiter.

DISCUSSION

The nonlinearities used in this model only show that the simple first order dynamics of the two stages are easily improved upon. However, these nonlinearities (which were used because they were easy to implement) are not suitable because we are still dealing with linear phenomena. What is actually needed are higher order linear stages
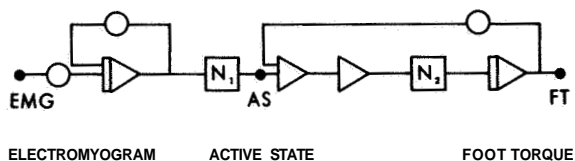
FIGURE 4.—Analog diagram of the model showing the position of the nonlinear elements.
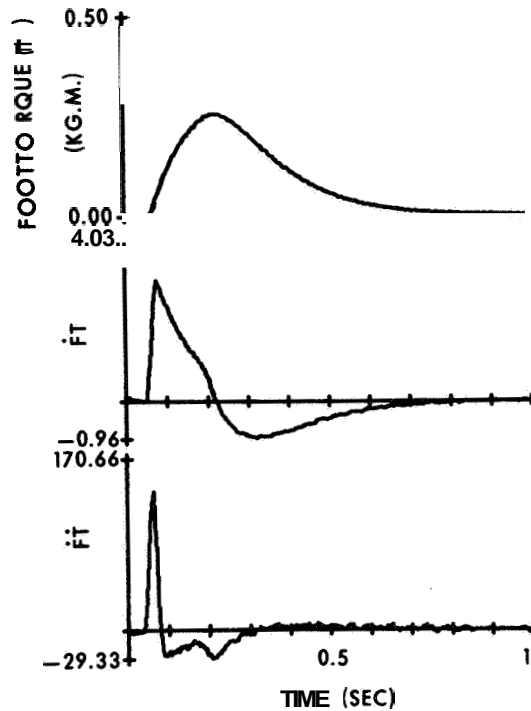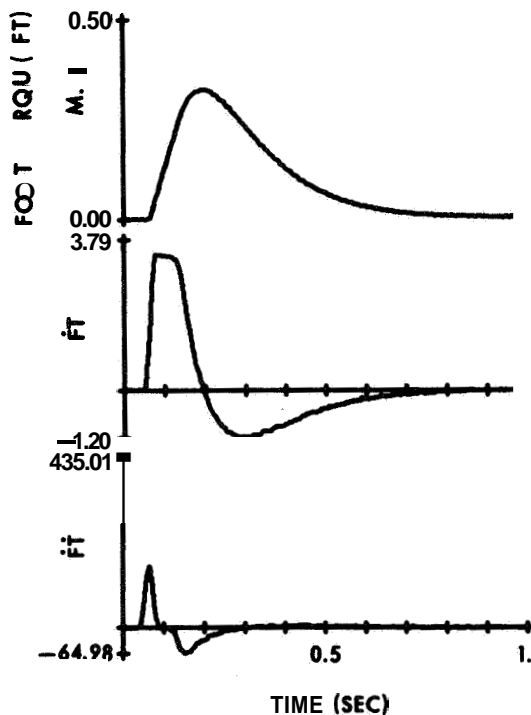
FIGURE 5.—Effect of the active state limiter.

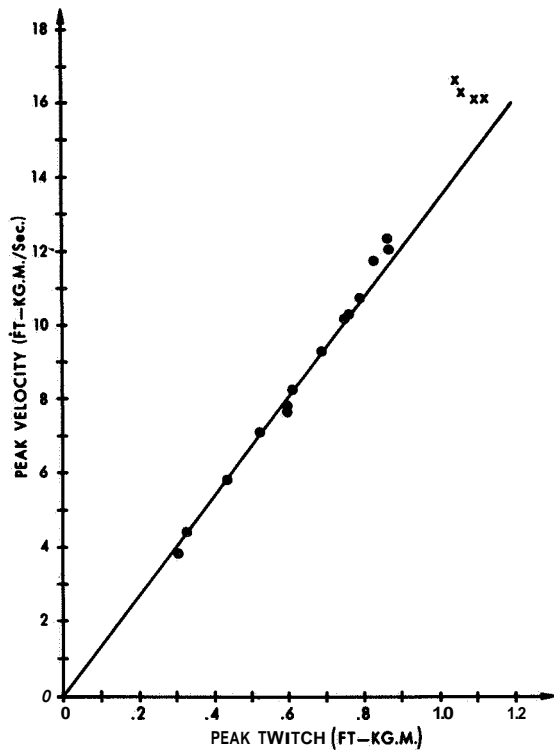FIGURE 6.—Effect of the velocity limiter.

FIGURE 7.—A plot of the peak velocity of the isometric twitch contraction vs the peak twitch in the soleus at different levels of stimulus voltage.

the M-wave (ref. 9). A plot of the peak velocity (with plateau) versus the peak twitch in such electrically evoked twitches is shown in figure 7. The circles are for H-wave response and the crosses are for M-wave response. Note that this relationship is nearly linear. The peak velocity is a function of the response, i.e. the input level itself. Thus the major change that is required in this model is that the mechanical parameters must be made functions of the input.

It seems clear, though, that a relatively simple model can be used to predict isometric muscle activity from the EMG.

which have a plateau in their response. That, at least, will improve the impulse response but its impact on the response with EMG inputs is not clear.

The impulse response of the physiological system is much more complex than what is shown in figure 3. In a slightly different experiment the tibial nerve at the back of the knee in popliteal fossa is stimulated with different levels of voltage stimulus. The EMG at the soleus at the lower levels of stimulus shows the familiar H-wave. As the stimulus level is increased, the alpha motor fibers are directly stimulated producing

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