

# A MUSCULOTENDON MODEL SUITABLE FOR USE IN NEUROMUSCULOSKELETAL CONTROL SIMULATION

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## ABSTRACT

A viscoelastic model has been developed for the dynamic behavior of a muscle and tendon pair suitable for use as an actuator in neuromusculoskeletal control simulation. This model accounts for the steady-state force generator characteristics of the musculotendon unit, such as force variation with length, velocity with length and velocity with load, as well as accounting for the transient behavior of the system in response to dynamic changes in activation and loading. This model is formulated as a bond graph in a manner to be implemented with standard system simulation software. A careful and thorough section on parameter estimation from available physiologic data is included. The bond graph is compatible with inclusion into an anatomically more comprehensive model, based on its inherent hierarchic capability for system building.

## INTRODUCTION

The static and dynamic characteristics of skeletal muscles and their accompanying tendons define the properties of the actuators in the neuromusculoskeletal system. Each musculotendon unit is similar in structure but has individual characteristics that tend to suit its particular role in producing the joint torques that ultimately cause skeletal motion. For example, muscles with a larger physiologic cross-sectional area are able to develop more total force, while muscles with a higher percentage of fast fiber composition are able to achieve a faster contraction velocity (Winters, 1988).

The musculotendon unit also provides feedback corresponding to the mechanical state of the muscle and tendon. Anatomically, these feedback transducers are the muscle spindles and the Golgi tendon organs. They are embedded within the musculotendon unit so as to experience the same relative length changes as the individual muscle and tendon. The feedback from these proprioceptors is integrated within the spinal chord and represents a control loop that operates beneath

higher-level control. A musculotendon model that is to be suitable for neuromusculoskeletal control simulation should make available signals corresponding to feedback from these musculotendon proprioceptors.

## MUSCULOTENDON ACTUATOR MODEL

A general musculotendon actuator model must be versatile and robust enough to be applied to many different types of skeletal muscle arranged in a very complex geometry. The model should be complex enough to account for commonly accepted behavior of muscle, yet be simple enough to be defined as a macro function for the actuators in a neuromusculoskeletal control simulation.

As an actuator, the musculotendon unit receives an activation level as an input and produces as outputs a force and velocity at the junction between the tendon and the load. Live muscle preparations are commonly tested *in-vitro* under isotonic and isometric conditions. Measurements made with these types of tests include the static tension versus length, velocity versus length at a constant load and velocity versus load. These types of tests characterize the force generator characteristics of the contractile unit after transient effects have decayed. Typical observations include the following.

The active force developed by the muscle reaches a maximum at the optimal length of the muscle and then declines as the muscle becomes shorter or longer than this length. Also, the developed active force sums with the passive force (Zajac, 1989). At a very light load, the velocity of contraction is constant in the midrange of sarcomere lengths. The velocity decreases at lengths below the midrange and increases at lengths beyond the midrange (Edman, 1979). The velocity of contraction is inversely related to load (Wilkie, 1950).

Other tests, such as the quick-release experiment, impose a sudden change in load to measure the transient response of the muscle. The results from these dynamic tests show that upon sudden change in the magnitude of the load, the muscle responds with a very rapid

length change that is dependent upon the difference between the initial and final load (Civan, 1968). This behavior correlates with the mechanical concept of a spring in series with the force generator.

A common theory for the molecular basis of muscle contraction contends that myosin and actin molecules form cross-linkages that actively cause the actin molecules to slide over the myosin molecules, thus developing tension and shortening the muscle. A simple mechanical model of a single cross-linkage is shown in Figure 1. The source of effort

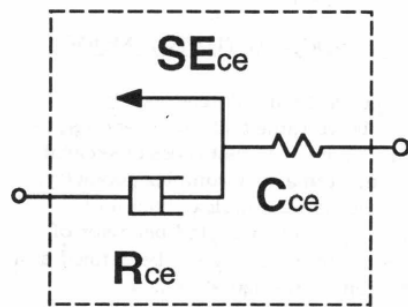


Figure 1. A mechanical schematic of a single cross linkage.

represents the ability of the contractile unit to develop tension in the muscle. The resistive dissipative element is included in parallel with the source of effort to account for the finite contraction velocity when the muscle is under no external load. The contractile element compliance represents the portion of the series elastic compliance that resides in the cross-linkages themselves (Huxley, 1971).

The remainder of the model is shown as a mechanical schematic in Figure 2. The

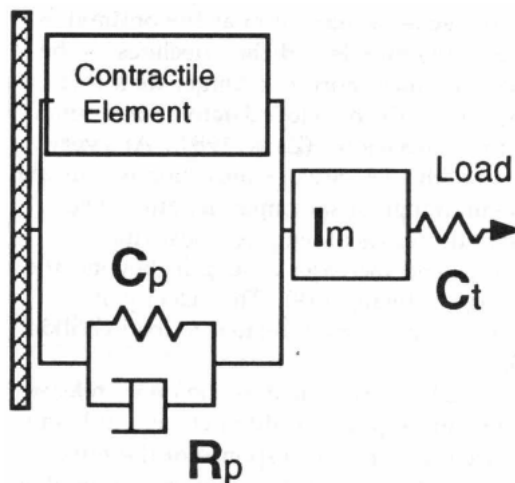


Figure 2. A mechanical schematic of a musculotendon unit.

passive compliance and resistance represents the behavior of the tissue that composes the ultrastructure of the muscle. The inertial element represents the mass of the moving muscle. The tendon compliance represents an elastic connection between the mass and the load.

### BOND GRAPH

Bond graph techniques were employed to build a model to simulate the behavior of this active mechanical system, as shown in Figure 3.

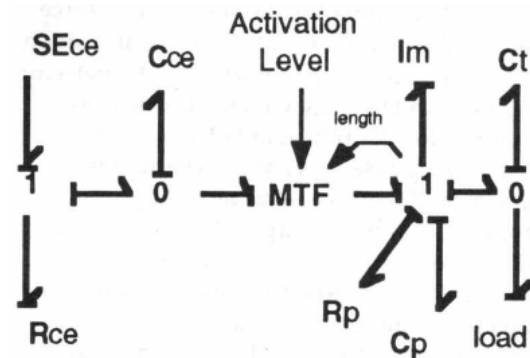


Figure 3. A bond graph of the musculotendon unit shown schematically in Figures 1. and 2.

The bond graph provides an effective mechanism for representing the dynamic energetic interactions and topology of the elements of this system in a format that contains inherently all of the information necessary to generate the governing system equations. In generalized terminology, 1 and 0 elements are nodes of common flow and effort. R, C, I, SE and SF elements represent ideal resistances, capacitances, inertias and sources of effort and flow. Half-arrows represent bonds between elements within the system. At each bond there is an identifiable effort and flow, the product of which is power. Bond graphs are of particular efficacy for biomedical systems because they are easily able to model the complex behavior of living systems which typically involve nonlinear phenomena and multiple coupled energy domains.

Some explanation of this particular bond graph is in order. The source of effort and the dissipative resistance of the contractile element,  $SE_{ce}$  and  $R_{ce}$ , are shown bound to a one junction. This indicates that the two elements have a common velocity, or flow. This is because the two elements are mechanically in parallel, as can be seen in Figure 1. The

adjacent zero junction has bound to it the compliance of the contractile element,  $C_{ce}$ . The zero junction specifies a common force, or effort. The compliance of the contractile element is bound to a zero junction because it is modelled as a massless spring and the force on both sides of a massless spring is of equal magnitude, as can be seen from the free-body diagram of a massless spring. A modulated transformer, MTF, connects the contractile machinery to the rest of the model. The modulation signal is the length of the muscle and the level of activation, which indicates that the number of active cross-linkages which may be formed is dependent upon the muscle length and the level of activation. The muscle inertia, parallel compliance and parallel dissipative resistance;  $I_m$ ,  $C_p$  and  $R_p$ , are shown bound to a one junction which, as previously mentioned, indicates a common flow. This can be seen in the mechanical schematic of Figure 2. The tendon compliance,  $C_t$ , is shown bound to zero junction because the tendon is modelled as a massless spring. Of interest is the causal stroke on the bond to the load. The causal stroke is towards the load, which indicates that the musculotendon unit is presenting an effort to the load, while the load is determining the flow. Thus, this model of a musculotendon unit is acting as a "force generator" rather than a "shortening generator". This is in agreement with published work (Julian, 1969).

#### PARAMETER ESTIMATION

These parameters were calculated so as to match the model to the triceps muscle and tendon of the arm; however the procedure for determining the parameters may be applied to any muscle and tendon pair for which appropriate data is available.

$SE_{ce}$  - The source of effort is determined by the maximum force that can be actively generated by the muscle. The maximum force is assumed to be dependent upon the physiologic cross-sectional area. The sum of the physiologic cross-sectional areas of the medial, long and lateral head of the triceps (An, 1981) was used to give a physiologic cross-sectional area of  $.00188 \text{ m}^2$ . A pressure of  $.5 \text{ MPa}$  (Winters, 1988) was used to determine a value of  $940 \text{ N}$  for the source of effort of the contractile element.

$R_{ce}$  - The dissipative resistance of the contractile element determines the unloaded shortening velocity of this model. The unloaded shortening velocity is assumed to be a function of fast versus slow fiber composition and muscle

length. Shortening velocity versus load data has been published for the triceps (Winters, 1988) that takes into account the fast versus slow fiber composition. This data is presented as angular velocity versus torque so a moment arm is necessary to convert this data to linear velocity versus force. A moment arm of  $.0247 \text{ m}$  at  $50$  degrees flexed (An, 1981) was used to give an unloaded shortening velocity of  $.424 \text{ m/s}$ . Edman (Edman, 1979) has published velocity versus length data for a single sarcomere. This data was assumed to be indicative of the velocity versus length for the entire muscle under maximum activation and was used to determine the dissipative resistance of the contractile element as function of muscle length as shown in Figure 4.

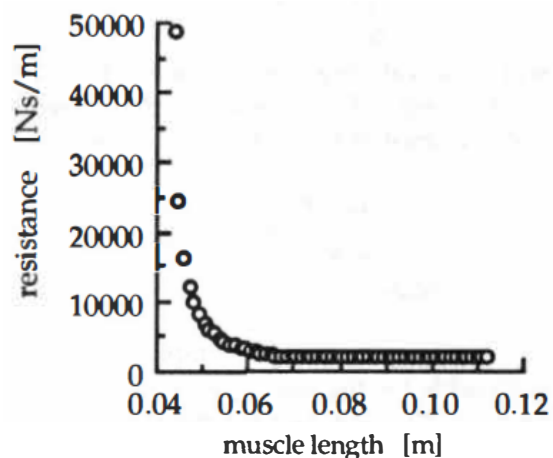


Figure 4. Contractile element resistance as a function of muscle length.

$C_t$  - A dimensionless force-strain curve (Zajac, 1989) was used to determine a value for the tendon compliance. The slope of this curve in the linear region is  $37.5$ . By using the maximum force of  $940 \text{ N}$  and a tendon length of  $.1933$  (Winters, 1988), a tendon stiffness of  $1.82 * 10^6 \text{ N/m}$  was calculated.

$I_m$  - The muscle inertia represents the mass of the moving muscle. The mass of the muscle may be found by multiplying the volume of the muscle times its density. Using a volume of  $152.6 * 10^{-6} \text{ m}^3$  (An, 1981) and a density of  $1000 \text{ kg/m}^3$ , the mass of the total muscle is calculated to be  $.153 \text{ kg}$ . The entire muscle is not, however, moving at the same velocity. Clearly, the portion of the muscle that is immediately adjacent to the fixed end will not be moving as fast as portions of the muscle that are farther away from the fixed end. If it is assumed that the velocity of the muscle is linearly increasing along its length, an

equivalent mass of 1/3 of the total mass may be calculated by requiring that the lumped mass have a kinetic energy equivalent to that of the distributed mass. In order to calculate the kinetic energy of the moving muscle, let:

KE = kinetic energy  
 m = mass of the muscle  
 V = shortening velocity of the muscle's free end  
 v = shortening velocity of an incremental mass element  
 l = length of the muscle  
 x = distance along the muscle, measured from the fixed end

$$dKE = (1/2)v^2 dm = (1/2)(Vx/l)^2 (mdx/l)$$

$$KE = \int_0^l (1/2)(mV^2 x^2 / l^3) dx = (1/6)mV^2$$

In order to calculate the equivalent mass, let the kinetic energy of the equivalent mass equal the kinetic energy of the distributed mass:

$$KE_{equiv} = KE_{distributed}$$

$$(1/2)m_{equiv}V^2 = (1/6)mV^2$$

$$m_{equiv} = (1/3)m$$

C<sub>p</sub> - The parallel compliance is responsible for the tension developed by the inactivated muscle as it is stretched beyond its resting length. By using a muscle fiber resting length of .083 m (An, 1981), the maximum active force of 940 N and a dimensionless force versus length curve (Zajac, 1989), the stiffness versus length of the parallel compliance shown in Figure 5. was calculated.

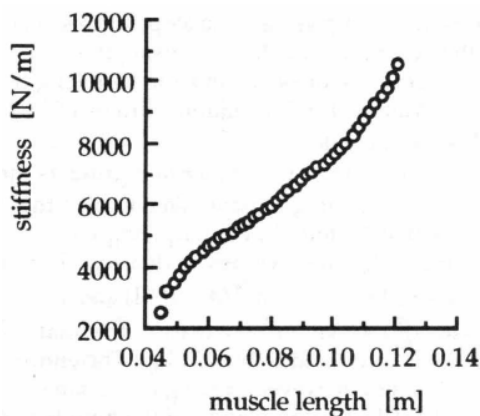


Figure 5. Passive stiffness as a function of muscle length.

R<sub>p</sub> - The parallel dissipative resistance was calculated to provide the unloaded

shortening velocity presented by Edman (Edman, 1979) for lengths greater than the resting length of the muscle.

C<sub>ce</sub> - The contractile element compliance is one of two undamped compliances in this model; the tendon compliance is the other. These compliances are arranged in series with each other and also in series with the source of effort. The total undamped series compliance is the sum of these two compliances. At the maximum muscle force its value is 8.96 \* 10<sup>-6</sup> m/N (Winters, 1988). The contractile element compliance is calculated by subtracting the tendon compliance from the total compliance which gives a contractile element compliance of 3.45 \* 10<sup>-6</sup> m/N.

MTF - The modulation function for the modulated transformer is determined by the number of cross-linkages that are actively producing force. In order to determine the modulation function, it was assumed that the number of cross-linkages that are actively producing force is directly proportional to the maximum active force that could be developed by the muscle at each muscle length. The general shape of the active force versus length curve is well known. A dimensionless active force versus length curve (Zajac, 1989) was combined with the muscle fiber length (An, 1981) to calculate the modulation function shown in Figure 6.

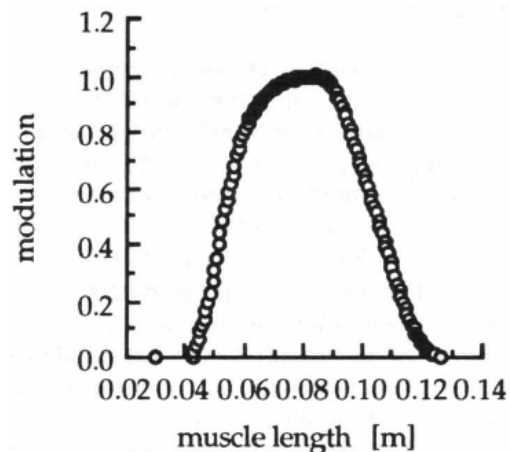


Figure 6. Modulation as a function of muscle length.

#### COMPUTER SIMULATION

The data represented in this paper was generated using Enport VII bond graph simulation software run on a VAX 11/750 computer. The model was given an activation level between 0.0 and 1.0 as the input. At 0.0 no

active force is developed and at 1 J the maximum active force available at the current muscle length is developed.

Figure 7. shows the force developed at the load during a simulated isometric test at full activation for a range of muscle lengths.

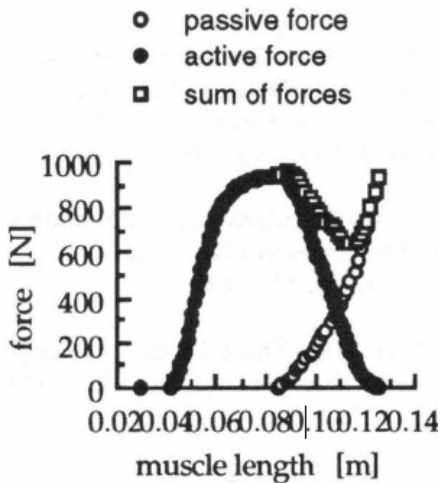


Figure 7. Simulation results showing the forces developed by the fully activated muscle as a function of muscle length.

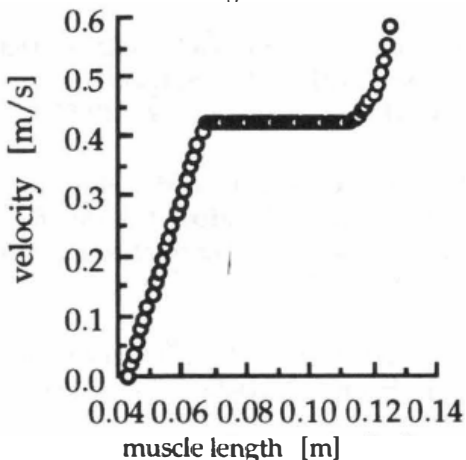


Figure 8. Simulation results showing the contraction velocity of a fully activated and unloaded muscle as a function muscle length.

Figure 8. shows the velocity of shortening versus muscle length at no load. The results show that the velocity is constant in the middle range of muscle lengths and then decreases as the muscle becomes shorter than this length. The velocity increases as the muscle is stretched beyond the midrange of muscle lengths due to the effect of the parallel compliance.

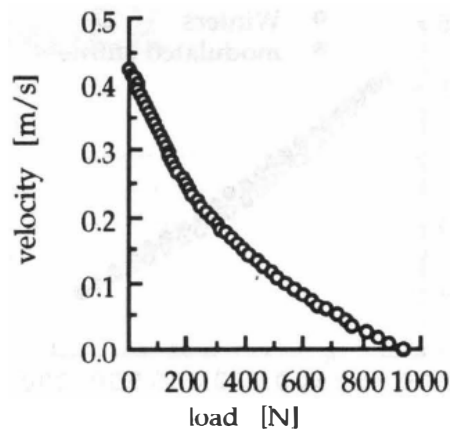


Figure 9. Simulation results showing the contraction velocity of a fully activated muscle at the rest length as a function of load.

Figure 9. shows the maximum active contraction velocity versus load from an isotonic simulation at full activation. The load was simulated by a source of effort resisting the muscle contraction. These results show that the velocity of contraction is inversely related to load, and occurs because as the load is increased, less net force is available to cause flow through the dissipative resistance of the contractile element. Live muscle preparations also show the characteristic that velocity of contraction is inversely related to load (Wilkie, 1950).

#### CONCLUSIONS

This viscoelastic model has been developed for use as an actuator in neuromusculoskeletal control simulation. The parameters of the elements were calculated so as to match the performance of the model to published data available for the triceps muscle and tendon of the arm; however the procedures presented for determining the parameters may be applied to any muscle and tendon for which appropriate data is available. State variables representing feedback from the muscle spindles and Golgi tendon organs are available for use in neuromusculoskeletal control simulation.

The total undamped series elasticity of the model as a function of force is shown in Figure 10. The series elasticity varies as a

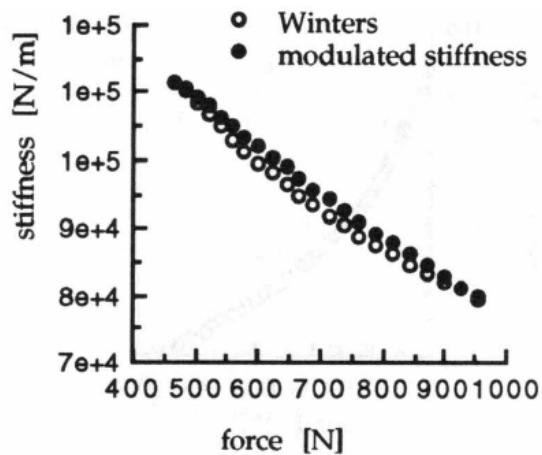


Figure 10. Total undamped series elastic stiffness as a function of muscle tension.

function of force because the series compliance of the contractile element is connected to the model through a modulated transformer. For comparison the series elasticity presented by Winters (Winters, 1988) is also shown. As can be seen, the modulation structure provides a series elasticity that is very similar to empirically derived data.

The modulation structure also predicts a severe penalty in performance when the muscle is operated away from its optimal length. The performance is degraded because the available contractile force decreases and the series compliance increases as the muscle moves away from its optimal length.

Extrapolation of the model to multiple musculotendon units operating in concert could be made easily by treating the model as a macro-function, many of which could be bonded together using appropriate 0 and 1 junctions to represent a more complex system.

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