

# Control Variables in Mechanical Muscle Models: A Mini-Review and a New Model

*Mark B. Shapiro and Robert V. Kenyon*

A new mechanical model of isolated muscle is proposed in which spring with variable slack length is the force-generating element. Based on the review of experimental studies in isolated muscle, it is suggested that spring slack length  $X_0$  is the control variable in the model and is a function of motor unit firing rate. In the presence of sensory feedback, the Sliding Spring model is equivalent to the Rack and Pinion model. However, sensory feedback is essential in the Rack and Pinion model but complementary in the Sliding Spring model. How the new control variable in the model of isolated muscle affects the interpretation of control processes up the motor system hierarchy is discussed in light of certain controversies associated with the Lambda and Alpha models of control of movement. It is argued that the Sliding Spring model of isolated muscle can be used as a basis for developing models of control of movement.

**Key Words:** human, animal, biological models, skeletal muscle/physiology, muscle contraction/physiology

Currently, models of control of voluntary movement reflect different views on the relative roles of central control and sensory feedback in generating patterns of muscle activation. Intimately related to this issue is the question as to what degree the apparent elasticity of the neuromuscular apparatus is due to intrinsic muscle properties and what can be attributed to sensory feedback. Numerous experiments with isolated muscle demonstrated strongly pronounced apparent elastic properties which should be reflected in the structure of muscle models and the choice of control variables. Despite the fact that identification of the control variables in models of movement control has been a hotly debated issue, this problem at the level of isolated muscle has been given limited attention. In this paper we present an analysis of how the control variables in a model of isolated muscle ultimately determine the possible central controls compatible with the results of experiments in neurologically normal and deafferented subjects.

Our objective is to ascertain whether some of the controversies associated

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with the existing models of control of movement necessarily follow from the properties of force-generating element in models of isolated muscle. Mechanical muscle models used in studies of motor control have been borrowed from the field of muscle physiology. The emphasis in their development may have been placed on the muscle properties essential for elucidating the physiology of muscular contraction but less important for studying motor control. In particular, series elastic element (SE) accounts for the data from the quick release experiments (Levin & Wyman, 1927) and affects dynamic behavior of a model but does not alter its static characteristic: a spring in series with a force generator is equivalent to the force generator, while two springs in series can be substituted by an equivalent spring. Similarly, viscous damper does not affect the system's static force-length characteristic. Neither SE nor viscous damper accounts for the force-generating ability of activated muscle. On the other hand, properties of the force-generating element for less than maximal activation remain unclear.

Since muscle models are used in simulation studies of the *control* of voluntary movement, we approach muscle modeling from the control point of view. We concentrate on the analysis of the force-generating element which models relations between muscle activation, force, and length. It can be either a force generator, a spring, or a combination of both. To determine the structure of the force-generating element, a number of questions need to be answered: (1) How to model force-length dependence for less than maximally activated muscle? (2) What are the relations between variables in a muscle model and parameters of muscle activation? (3) Which variables in a muscle model are control variables and what are the criteria for choosing them?

First, in Section 1 we review the studies of isolated muscle in which the parameters of activation were manipulated by the experimenter. In Section 2 we briefly review the models developed by Gasser and Hill (1924) and Hill (1938). A new mechanical muscle model with slack length  $X_0$  of the sliding spring as the control variable is presented in Section 3. In Section 4 we discuss the consequences of the qualitative differences between the models of isolated muscle for the EP models of control of movement (Bizzi et al., 1992; Feldman & Levin, 1995; Shapiro, 1997).

## 1. Review of Studies of Activation-Force-Length Relations in Isolated Muscle

Of all the studies of isolated muscle, we are particularly interested in the experiments in which muscle activation was varied by the experimenter. Activation is the control input to muscle generated by the central nervous system (CNS), while force, length, and velocity are determined by the interaction of muscle and external loads. Consequently, in muscle models the variables related to motor unit (MU) firing rate and/or recruitment are recognized as controls in distinction to all the other variables considered mechanical inputs, outputs, or parameters.

Partridge (1966, 1967) was the first to show that isolated muscle stimulated with varying frequency exhibits spring-like behavior. The muscle was stimulated through the cut ventral root with sinusoidally modulated pulse rate. Partridge's conclusion in the analysis of the load-moving response of the muscle was that it should be viewed not as a force generator but "as a servo-system following a position command on its motor nerve with an appropriate load position, while force used to accomplish this positioning is adjusted as needed" (1966, p.

1190). In a follow-up experiment (Partridge, 1967), the muscle was contracting against a constant gravity load while the stimulus rate was slowly varied. When plotted in force-length coordinates, the relations between force, length, and stimulus rate appeared as a family of force-length curves that could be obtained from one another by translation along the length axis and change in the slope.

Observations that a change in the stimulus rate resulted in a translation of the static length-tension characteristic along the length axis have been confirmed in a number of later studies (Crago, 1979, 1992; Rack & Westbury, 1969). Moreover, Roszek et al. (1994) showed that a change in stimulus rate was associated with a translation of the length-tension curve along the length axis with almost equal change in both muscle "optimal" length and slack length. In all these experiments the slope of the length-tension curve increased with an increase in the stimulus rate. The slope, however, has been shown to depend particularly strongly on MU recruitment (Houk et al., 1970; Nichols, 1973).

It can be argued that muscle activated through the intact efferents behaves differently than when stimulated through the cut ventral root. In the experiments in intact muscle activated by way of stimulation in the central structures of the decerebrate preparation (Feldman & Orlovsky, 1972; Matthews, 1959a, 1959b), the slack length effect was observed but was attributed to the centrally set stretch reflex threshold (Feldman & Orlovsky, 1972). However, it might be expected that a response of deafferented muscle to stimulation in the central structures can also be described as a shift in the slack length of the muscular spring. An accidental occurrence of such an effect was reported by Matthews (1959b). In this experiment the size of the stretch reflex in the decerebrated cat was reduced by paralyzing the gamma efferent nerve fibers by applying procaine to the muscle nerve. Procaine blockade resulted in a gradual shift of the slack length without changes in the shape of the length-tension characteristic. After the gamma fibers had been paralyzed, the slack length assumed its maximum value. Matthews also reported, "A more striking effect was obtained by chance in a preparation in which the 'tone' increased spontaneously at a time when the gamma efferents were paralyzed. . . . In this isolated occurrence there was a striking displacement of the curve with little change of its slope" (p. 561).

The results reviewed above suggest that the static length-tension characteristic of less than maximally activated isolated muscle exhibits the translation along the length axis and change in its slope. If muscle is modeled by a spring with stiffness as a single control variable, then the equations describing the shape of the length-tension characteristic can be quite complex. Alternatively, introducing a second control variable that governs the translation of the characteristic along the length axis may lead to simpler models. This approach is similar to modeling systems with time delay in which case the accuracy can be improved either by significantly increasing the order of a polynomial approximation or by adding a new variable, time delay, which makes a model more succinct and conceptually clear.

## 2. Force-Generating Elements in Mechanical Models of Isolated Muscle

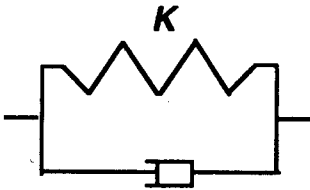
### *Gasser-Hill Model: Damped Spring With Controlled Stiffness*

In the early 1900s the skeletal muscle was viewed as a damped elastic actuator.

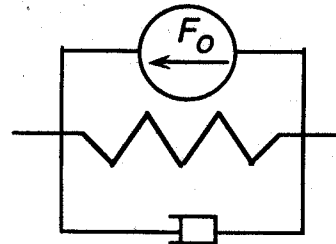
Gasser and Hill experimented with the isolated frog muscle stimulated directly and obtained the length-tension and force-velocity curves confirming the previously established linear relation between the force and *instantaneous* velocity of shortening *at a given length* (Gasser & Hill, 1924). As a second part of the study, Gasser and Hill attached a vibrating spring to a pair of muscles. They compared the motion of the muscle-spring system with the passive and maximally tetanized muscle and obtained the relation:

$$ma + (b_{spr} + b_{muscle})v + (k_{spr} + k_{muscle})x = 0 \quad (1)$$

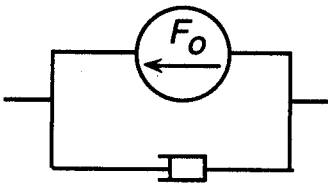
where  $m$  is mass of the spring,  $a$  is acceleration,  $v$  is velocity,  $x$  is displacement,  $b_{spr}$  is viscous coefficient of the apparatus,  $b_{muscle}$  is viscous coefficient of the muscles, and  $k_{spr}$  and  $k_{muscle}$  are stiffness of the spring and muscles, respectively. Muscle stiffness was considered a function of activation. Gasser and Hill proposed a model that included a spring and viscous damper in parallel (Figure 1). Force generator was not included in the model. An implicit assumption was that the spring stiffness is the only parameter related to activation.



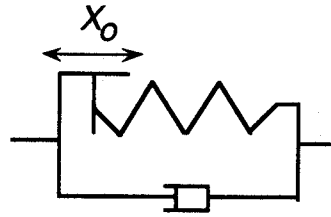
**Gasser-Hill model**



**Augmented Hill model**



**Hill model**



**Sliding spring model**

Figure 1 – Contractile elements (CE) in the mechanical muscle models. Spring stiffness  $K$  is the control variable in the Gasser-Hill model, force  $F_0$  is the control variable in the Hill and augmented Hill models, and slack length  $X_0$  is the control variable in the Sliding Spring model.

### Hill Model: Damped Force Generator

Muscle research at that time was focused on the apparent muscle viscosity, and the Gasser-Hill model was rejected after the discovery of the hyperbolic force-velocity relation (Hill, 1938):

$$(F + \underline{a})(v + \underline{b}) = (F_{f.g} + \underline{a}) \underline{b} = \text{const}$$

or, in a different form:

$$\begin{aligned} F &= F_{f.g} - (F_{f.g} + \underline{a})v/(v + \underline{b}) \\ &= F_{f.g} - F_{visc} \end{aligned} \quad (2)$$

where  $F$  is force of contracting muscle,  $F_{f.g}$  is isometric muscle force, and  $F_{visc}$  is resistance of the viscous damper. Note that in the Hill model (Figure 1) a force generator  $F_{f.g}$  is included instead of the spring. This substitution represents a major qualitative change for a control model.

The force-length dependence of the contractile element (CE) is of primary interest as far as control models are concerned, and in the following the taxonomy of models is based on whether the CE contains a spring or a force generator. The models in which the CE includes a spring with activation-dependent stiffness are considered Gasser-Hill models, while those with force generator are considered Hill models. The major qualitative difference between the Gasser-Hill and Hill models bears strong implications for the analysis of stability of posture and equilibrium as well as for the sensitivity to disturbances and control errors during movement. For example, antagonistically arranged force generators require position feedback in order for the limb to achieve and remain at a desired final position in the presence of disturbances or a mismatch in the force commands. In contrast, a joint actuated by two (damped) springs will always settle at an equilibrium position determined by the spring controls.

The reason that two qualitatively different models were developed based on studies of the same system is that muscle length-tension characteristic is strongly nonlinear. One approach for describing a highly nonlinear system is to use a global model that describes the system's behavior within a broad range of conditions. Alternatively, it may be desirable to use a number of local models. The advantage of the second approach is that the resulting models are simpler. However, it is essential to identify the region within which the system is operating and check the conditions for applicability of a particular local model.

The Gasser-Hill and Hill models are local. The Gasser-Hill model could be valid for a muscle working on the ascending limb of the length-tension characteristic. However, as discussed below in the analysis of control of a static position in the Alpha model (Bizzi et al., 1992), the Gasser-Hill model leads to predictions about muscle activation that have not been corroborated by the experimental observations. Hill's 1938 results were obtained with the muscle length kept in close proximity of the plateau on the length-tension curve so that the assumption about the constancy of isometric tension was justified. Later it was shown that Hill's force-velocity relation holds for shorter muscle lengths with an added assumption that  $F_{f.g}$  decreases with length (Abbott & Wilkie, 1953), and Hill suggested a force-length dependence to be included in the CE

(Hill, 1964). In the following, the Hill model with a parallel spring within the CE is called the augmented Hill model (Figure 1). However, Hill-based models are generally too compliant for quasi-static conditions, especially for inverted pendulum postural systems (Winters, 1995). Finally, for less than maximal activation, the force-length dependence in Hill-based models remains unclear (see Winters, 1990, for a review of Hill-based models).

### 3. New Mechanical Model of Isolated Muscle: Damped Sliding Spring

#### *Spring Slack Length as a Control Variable*

Gasser and Hill's assumption that the spring stiffness is the only parameter related to activation does not hold. First, consider an ideal mechanical spring in static conditions:

$$F_{ext} - KX = 0 \quad (3)$$

where  $K$  is stiffness, and  $X$  is the coordinate of the end of the spring to which the load  $F_{ext}$  is attached. Equation 3 is valid if the formalism of *free body diagram* can be applied, that is, if the origin of the coordinate system can always be brought to coincide with the point at which the spring starts generating tension. However, if the spring is part of a larger structure, it may not be possible to freely move the origin of the system of coordinates, and Equation 3 takes the form:

$$F_{ext} - K(X - X_o) = 0 \quad (4)$$

Assume that the origin of the system of coordinates is kept at one of the tendon attachments (Figure 2). In this case the muscular spring in general is described by Equation 4.

Both muscle stiffness and slack length depend on muscle activation  $A$ , and a muscle model should generally include the relations  $K = f(A)$  and  $X_o = f(A)$ . However, if a model is to be simplified, it is often necessary to choose between  $K$  and  $X_o$  which variable is considered the control while the other is a parameter. For example, consider an approximated static dependence between muscle force and length and MU recruitment and firing rate (Crago, 1992):

$$F = K(X - X_o) \quad (5)$$

with

$$K = c_1 A_r A_f \quad (6)$$

$$X_o = X_{max} - c_2 A_f + c_3 \quad (7)$$

where  $X$  is muscle length,  $F$  is muscle force,  $A_f$  is instantaneous MU firing rate,  $A_r$  is the scaling factor representing MU recruitment level, and  $c_1, c_2, c_3, X_{max}$  are constants. Crago suggested Relations 5 through 7 to be incorporated in Hill based models. However, in the later simulation studies (Lemay & Crago, 1996; Shue & Crago, 1998; Shue et al., 1995), slack length  $X_o$  was a constant and the force was

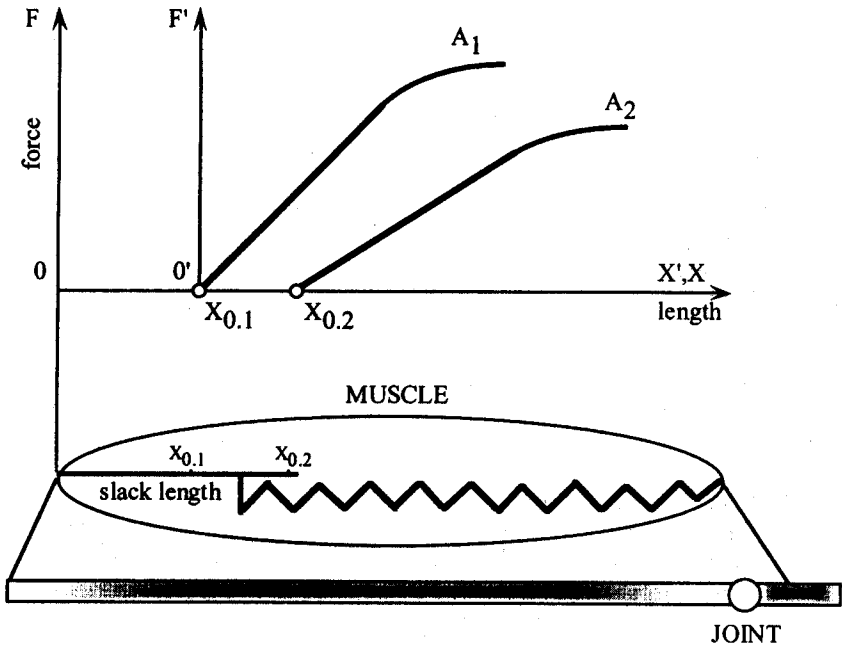


Figure 2 – Coordinate systems for muscular spring. Slack lengths  $X_{0.1} < X_{0.2}$ , muscle activation (firing rate)  $A_1 > A_2$ . Coordinate system  $0FX$  is fixed while  $0'F'X'$  moves along the length axis with change in activation.

calculated as a product of three components as in the muscle model developed by Zajac (1989):

$$F = F_o * F_v * F_L \tag{8}$$

where isometric force of the CE is a function of muscle activation  $F_o = f(A)$ ,  $F_v$  is Hill's force-velocity term, and  $F_L = f(x - X_o)$ . With  $X_o$  being a constant, Equation 8 corresponds to the Gasser-Hill model with the spring stiffness as the control variable.

In Zajac's model it is assumed that the muscle force-length characteristic scales with activation. Scaling of the characteristic changes the height of the flat region and slope of the ascending limb but does not affect its length axis intercept (Figure 5 in Zajac, 1989). Zajac's model is global and is equivalent to the Hill model for muscle operating within the plateau on the length-tension curve, and to the Gasser-Hill model for muscle operating on the ascending limb. Consequently, the arguments presented later against the Hill and Gasser-Hill models can be applied to Zajac's model as well.

We suggest that muscle operating on the ascending limb of the static length-tension characteristic is modeled by a sliding spring with slack length  $X_o$  as the control variable (Figure 1). While stiffness of the spring in the CE is very important for giving quantitative account of muscle behavior, the stiffness alone

should not be considered the control variable unless an invertible relation between the stiffness and slack length is assumed:

$$K=f(X, X_o) \Leftrightarrow X_o=f^{-1}(X, K) \quad (9)$$

in which case either  $K$  or  $X_o$  can be considered the control variable (e.g., Lan & Crago, 1994).

A spring with variable slack length  $X_o$  can be realized with a Rack and Pinion model (Houk, 1979). The Rack and Pinion model, however, has been proposed as a mechanical analogue for Feldman's model of intact muscle in which the slack length of the spring is determined by the centrally set stretch reflex threshold (Feldman, 1966a, 1974). To avoid confusion we use the name "sliding spring" instead of "rack and pinion" to emphasize that slack length  $X_o$  is attributed to the intrinsic muscle properties.

### *The Augmented Hill Model vs. the Sliding Spring Model*

The Hill model remains the model of choice in motor control and biomechanics studies, and any new model is bound to be compared with it. It can be shown that the augmented Hill model can be made equivalent to the Sliding Spring model by reversing the direction of the force generator. Consider the ascending limb of the muscle static length-tension characteristic (sketched in Figure 3):

$$F_{ext} - K(X - X_o) = 0 \quad (10)$$

The range of  $X_o$  in Equation 10 requires special attention. From the data reported by Rack and Westbury (1969), it follows that the range of  $X_o$  includes positive values:

$$X_o > 0 \quad (11)$$

With  $X_o$  varying within range (Equation 11), Equation 10 generates a family of length-tension curves (Figure 3A).

Next, consider the CE of the augmented Hill model:

$$F_{ext} - KX + F_{f.g} = 0 \quad (12)$$

Since muscle can only pull but not push, the force generator in Hill-based models is codirectional with the elastic force of the spring and oriented opposite to the  $X$  axis:

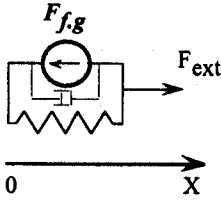
$$F_{f.g} < 0 \quad (13)$$

With  $F_{f.g}$  varying within range (Equation 13), Equation 12 generates a family of length-tension curves (Figure 3B).

The Sliding Spring model (Equations 10 and 11) generates curves (A) which in the first quadrant exhibit translation along the *length* axis similar to the experimental results. To the contrary, curves (B) generated by the augmented Hill model (Equations 12 and 13) exhibit vertical shift along the *force* axis. This dif-



Augmented Hill model, curves B



Sliding spring model, curves A

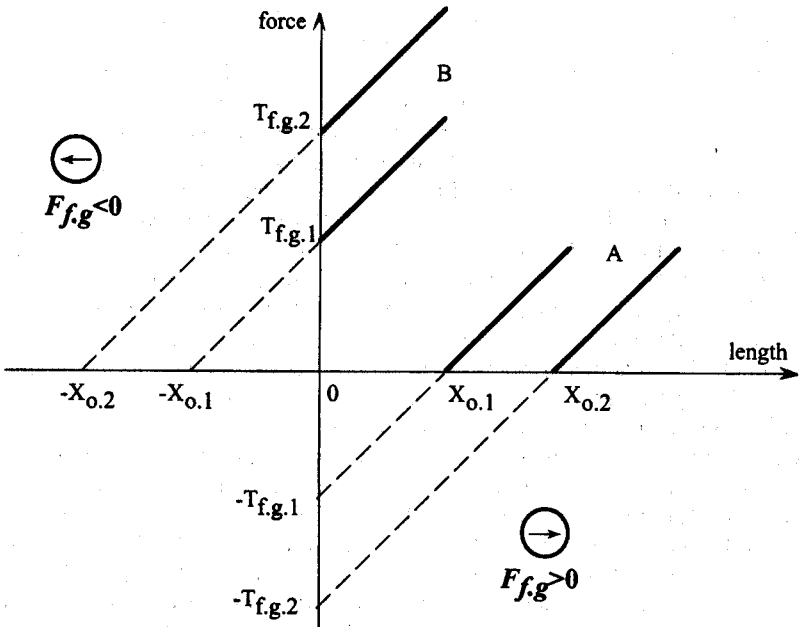
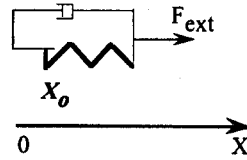


Figure 3 — Length-tension characteristics of augmented Hill and Sliding Spring models.  $X_{o,i}$  is slack length, force axis intercept  $T_{f.g.} = -F_{f.g.}$ . The curves A are generated with  $X_o > 0$  ( $F_{f.g.} > 0$ ) while the curves B are generated with  $F_{f.g.} < 0$  ( $X_o < 0$ ). The segments of the curves in the first quadrant (solid lines), where muscle characteristics are directly measurable, translate along the length axis (A) and along the force axis (B).

ference is due to the range (Equation 13) of allowed values for  $F_{f.g.}$  determined by the orientation of the force generator. Equations 10 and 12 are equivalent if  $F_{f.g.}$  and  $X_o$  have the same sign, meaning that inequality (Equation 13) is reversed so that the force generator pushes against the parallel spring. The unidirectional force generator has been thought to represent collective behavior of cross-bridges, and the reversal of its direction presents a conceptual quandary for Hill-based models.

## Explanation of the Slack Length Effect

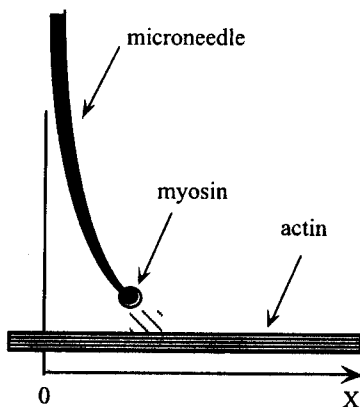
Mechanical behavior of the whole muscle is determined largely by the characteristics of cross-bridges because of the succession of the series and parallel arrangements of the muscular structures. If the cross-bridges behave as force generators, then the sarcomeres and muscle as a whole behave as a force generator, provided that the number of active cross-bridges does not change. Therefore, to explain the ascending limb of the length-tension characteristic, it was suggested that the number of active cross-bridges changes because of meeting and then double overlapping of the thin filaments (Gordon et al., 1966). A key question, however, is whether cross-bridges behave as force generators at short muscle lengths. Gordon and colleagues stated that the slack length effect in the sarcomere could not be explained on the then current formulation of Huxley's (1957) theory. Rack and Westbury (1969) proposed a hypothesis that at short lengths the process of activation may function less effectively.

The evidence has been accumulating that the ascending limb is due to incomplete activation of myofibrils at short sarcomere lengths (Close, 1972; Lopez et al., 1981; Schoenberg & Podolsky, 1972; Stephenson & Wendt, 1984; Sugi et al., 1998), also reviewed in Huijing (1998). There is also some experimental support for the idea that the muscle force-length relation reflects the basic properties of individual actin-myosin linkages (Oiwa et al., 1991; Schutt & Lindberg, 1992; Sugi et al., 1983; 1998). Without attempting to review the extensive literature on the sliding filament theory, we bring to the reader's attention some experimental results that appear to lend support for the Sliding Spring model.

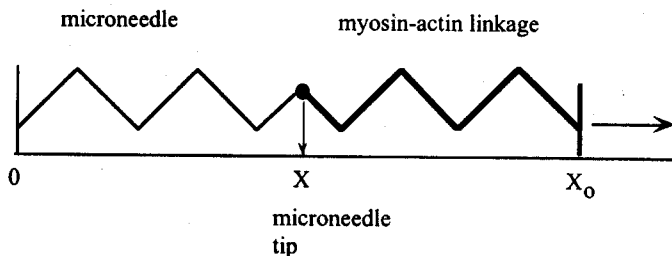
Echoing Partridge's conclusion regarding the whole muscle, Sugi et al. (1993) noted that "there appears to be a kind of load sensor within a cross-bridge." These authors studied actin-myosin interaction using an *in vitro* assay system consisting of a glass microneedle with myosin-coated tip and an array of actin cables (Figure 4A) (Chaen et al., 1989). The needle tip first attached to the actin cables due to rigor links. With an application of ATP, the tip moved along the actin cables and stopped at a new position when the ATP diffused. Subsequent applications of the same amount of ATP induced the needle-tip movement starting from an increasingly higher baseline force attained at the preceding position. Yet despite the fact that the ATP-induced tip movements started at the various initial baseline forces, the magnitude of the successive movements was nearly the same within the range of baseline forces from 0 to 0.4-0.6 of maximal attained force. Sugi and colleagues (1993) concluded that the apparent efficiency of actin-myosin sliding in producing work increases with the initial baseline force. This phenomenon could not be attributed to the varying number of myosin heads, since the authors estimated that the number of myosin heads involved was very small (around 500) and all were activated almost simultaneously.

To model this phenomenon, we assume that an ensemble of cross-bridges in the presence of ATP acts as a spring with one end sliding along the actin filament (Figure 4B). The springs representing the myosin-actin linkages and microneedle have stiffness  $K_{ma}$  and  $K$ , respectively, and the deflection  $X$  of the microneedle tip is given by:

$$X = \frac{K_{ma}}{K_{ma} + K} X_0 \quad (14)$$



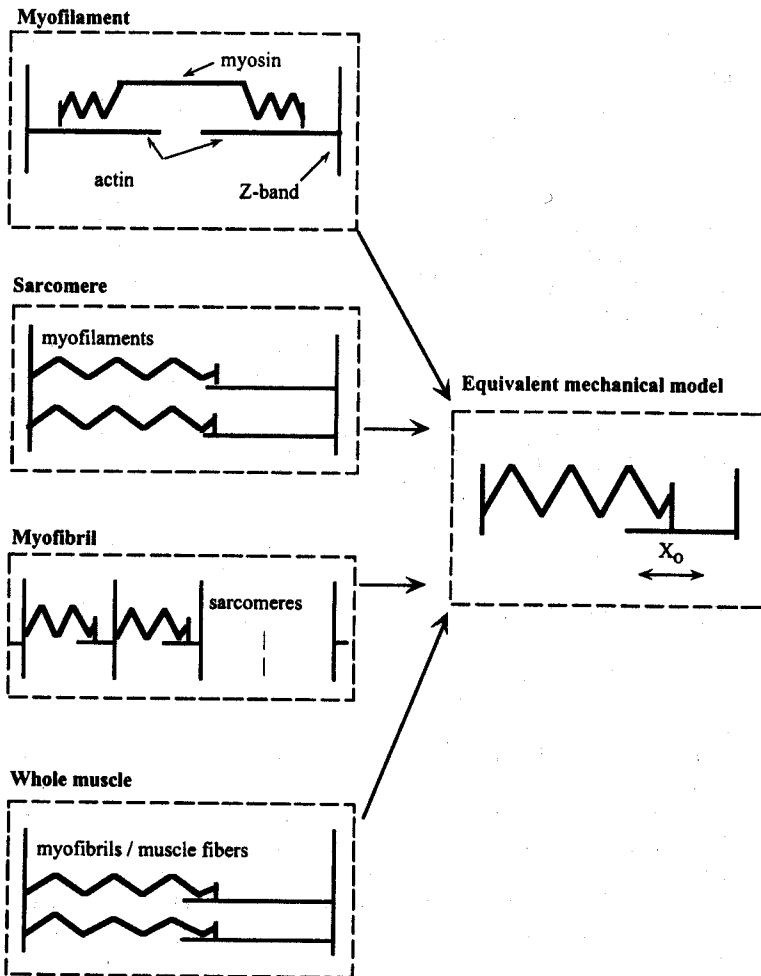
a) Schematics of the in vitro assay system.



**Figure 4 — Schematics and equivalent mechanical diagram of the in vitro assay system. (See text for further explanations.)**

If the effect of one ATP application can be described as a shift  $\Delta X_o$  of the sliding end of the spring representing myosin-actin linkages, then application of equal amounts of ATP induces equal successive shifts  $\Delta X_o$  and, consequently, equal tip displacements  $\Delta X$ , provided the spring stiffnesses do not change.

The gap between the sliding spring model of actin-myosin interaction and a model of whole muscle can be bridged if we consider the process of excitation-contraction coupling. Muscular contraction occurs due to the interaction between the proteins of the thick and thin filaments regulated by calcium. The sarcomere length at which the ascending limb of the length-tension characteristic intersects zero tension is dependent on the  $\text{Ca}^{2+}$  concentration (Allen & Moss, 1987). Therefore, slack length  $X_o$  of the spring in the model of sarcomere (Figure 5) is a function of the concentration of intracellular calcium, which in turn depends on MU firing rate. The shifting of the slack length and translation of the force-length characteristic as a whole with changes in the stimulus rate have been demonstrated for single muscle fibers (Balnave & Allen, 1996) as well as for fiber bun-



**Figure 5 — Mechanical models of various structural elements within muscle. (See text for further explanations.)**

dles (Zuurbier et al., 1998). Because of the internal structure of muscle, the equivalent mechanical model remains the same for myofilaments, sarcomeres, myofibrils, muscle fibers, and whole muscle (Figure 5).

### *The Sliding Spring Model With Sensory Feedback*

In the presence of sensory feedback, the central and sensory inputs to the alpha motoneuron pool complement each other and the resulting level of muscle activation sets the slack length. For completely relaxed muscle, the slack length  $X_0$

$$X_o = X_{max} - A \quad (15)$$

where  $A$  is muscle activation scaled such that it has units of length. To illustrate the effect of sensory activity, we consider only the position feedback:

$$A_{sens} = K_x X \quad (16)$$

where  $A_{sens}$  is the sensory contribution to muscle activation and  $K_x$  is the gain of position feedback. Assuming that the central  $A_{centr}$  and sensory components of muscle activation are additive (Crago et al., 1976; Nichols & Houk, 1976), Equation 15 takes the form:

$$X_o = X_{max} - (A_{centr} + K_x X) \quad (17)$$

Isometric force generated by the sliding spring is a function of the difference between the slack length and actual length:

$$\begin{aligned} F &= f(X - X_o) \\ &= f((I + K_x)(X - (I + K_x)^{-1}(X_{max} - A_{centr}))) \end{aligned} \quad (18)$$

The centrally set spring slack length is  $X_{centr,0} = X_{max} - A_{centr}$ . The resulting slack length is:

$$X_o = (I + K_x)^{-1} X_{centr,0} \quad (19)$$

As follows from Equation 19, it is possible for the "central controller" to control the slack length  $X_o$  independently of the sensory input, provided that the gain of position feedback  $K_x$  is known and does not depend on muscle length. However,  $K_x$  most likely depends on muscle length, given at the very least the strongly non-linear properties of muscle spindles (Gielen & Houk, 1984; Gielen et al., 1984; Hasan & Houk, 1975a, 1975b). We hypothesize that the inability to independently control slack length requires that the central controller predict the level of sensory activity in order to produce a desired time profile of  $X_o$  during movement. The need to predict motor outcome makes a concept of internal model of the limb and load a *sine qua non* for analysis of voluntary movement. Internal models of the limb and environment developed by the CNS should include an exact representation of what will occur at the periphery during movement (Arbib, 1984), and form an essential part of a general hypothesis about the brain's active mental modeling of the environment (Bernstein, 1967).

#### 4. Implications for Models of Control of Movement

##### *Equilibrium Point Hypothesis*

Apparent elasticity of the neuromuscular apparatus is reflected in the Equilibrium Point (EP) hypothesis proposed by Feldman (1966a; 1966b). However, in the motor control literature the EP hypothesis has been blended with the models of control of movement. To distinguish between the EP hypothesis per se and auxiliary assumptions, we will adopt the following explicit formulation:

- (i) The joint spanned by antagonistic muscles exhibits elastic behavior during movement and maintenance of posture;
- (ii) Joint equilibrium position is the control variable in mechanical models of the joint when (i) is valid.

Statements (i) and (ii) are applicable at the level of the joint and do not bear upon models of isolated muscle. While muscle with intact afferents should be modeled by a spring in accord with (i), the spring can be equally well realized by a force generator with position feedback or by a spring with position feedback. Once (i) is accepted, however, the models of voluntary movement in which joint is activated by torque generators, such as the Minimum Torque Change model (Uno et al., 1989) and Minimum Muscle Tension Change model (Dornay et al., 1996), must be thought of as the models of force/torque *planning* rather than *control*.

### The Lambda Model

Isolated muscle in the Lambda model has been described by the Hill model (St-Onge et al., 1997). Since the force-generating element in the model of isolated muscle is a force generator, the elastic characteristic of intact muscle can only be realized by position feedback provided by the tonic stretch reflex, and slack length  $X_0$  (denoted  $\lambda$  in the literature on the Lambda model) is related to the centrally set stretch reflex threshold. Consequently, the Lambda model cannot be applied in analyzing experiments in deafferented humans and animals that demonstrated elasticity of the neuromuscular apparatus (e.g., Bizzi et al., 1978; 1984; Goldberger, 1988; Gordon et al., 1995; Lajoie et al., 1996; Nougier et al., 1996; Rothwell et al., 1982).

Slack length in the Lambda model is associated with a hypothetical central input to the alpha motoneuron pool kept at a subthreshold level. This contradicts the observations that after acute deafferentation in the human subjects, the central input alone was sufficient to activate the muscles (Gandevia et al., 1990; 1993). It was also estimated that the afferent feedback accounted for only about one-third of the output of the alpha motoneuron pool at all levels of voluntary drive (Macefield et al., 1993).

### The Alpha Model

**Muscle Model.** According to the Alpha model, the neural input to muscle determines the intrinsic muscle stiffness linearized by neural feedback. It can be shown that in the simulation studies, isolated muscle was modeled by the Gasser-Hill model. Isometric muscle torque was given as:

$$T_{ag} = (T_{max} - K\Theta)U_{ag} \quad (20)$$

$$T_{ant} = -(T_{max} + K\Theta)U_{ant} \quad (21)$$

where  $T_{ag}$  and  $T_{ant}$  is the agonist and antagonist torque, respectively,  $T_{max}$  is maximal isometric muscle torque in the joint middle position,  $\Theta$  is joint angle with a range of  $-\pi/2 < \Theta < \pi/2$ , values of muscle neural controls have a range of  $0 \leq U_{ag}, U_{ant} \leq 1$ , and muscle stiffness is within the bounds  $0 \leq K \leq 2T_{max}/\pi$ .

Depending on the assumed maximal value of stiffness, Equations 20 and 21 represent two different muscle models. To show that, we introduce a new coordinate:

$$\Theta^* = +\pi/2 + \Theta$$

so that  $0 \leq \Theta^* \leq \pi$ . For example, if positive  $\Theta$  corresponds to flexion, then  $\Theta^* = 0$  for fully flexed limb and  $\Theta^* = \pi$  for fully extended limb. If the assumed maximal stiffness is

$$K_{max} = 2T_{max}/\pi$$

then Equations 20 and 21 can be rewritten as:

$$T_{ag} = K_{max}\Theta^*U_{ag} \quad (23)$$

$$T_{ant} = K_{max}(\Theta^* - \pi)U_{ant} \quad (24)$$

Equations 23 and 24 represent two springs with constant slack angles  $\Theta^*_{ag,0} = 0$  for agonist, and  $\Theta^*_{ant,0} = \pi$  for antagonist. If, on the other hand, assumed maximal stiffness  $K_{max} < 2T_{max}/\pi$ , then by introducing a term  $T_{f.g.}$ :

$$T_{f.g.} = T_{max} - K_{max}\pi/2$$

Equations 23 and 24 become:

$$T_{ag} = K_{max}\Theta^*U_{ag} + T_{f.g.}U_{ag} \quad (25)$$

$$T_{ant} = K_{max}(\Theta^* - \pi)U_{ant} - T_{f.g.}U_{ant} \quad (26)$$

Equations 23 and 24 correspond to the Gasser-Hill model, while Equations 25 and 26 represent a model in which activation controls both force of the force generator and stiffness of the parallel spring. The Gasser-Hill model (Equations 23 and 24) was used in the simulation studies in which the muscle model equations were given explicitly (Flash, 1987; Hogan, 1984).

*Control of Movement.* Initially, it was suggested that the CNS controls movement and posture by making appropriate changes in the stiffness of the agonist and antagonist muscles spanning the joint (Bizzi et al., 1982; Cooke, 1980). However, it has been argued that when applied to a task of maintaining joint static positions throughout the joint angular range, the concept of stiffness control is contradictory (Berkinblit et al., 1986; Feldman, 1986). In particular, the stiffness commands to the antagonistic muscles should be similar when the joint is in the middle of its angular range, while near the boundaries the stiffness of the shortened muscle is maximal and the stiffness of the lengthened muscle is minimal. Muscle stiffness is determined mainly by MU recruitment, and the highly unequal stiffness commands are expected to be associated with the large difference in the levels of MU recruitment in the antagonistic muscles which can be estimated by measuring surface EMG. The experimental data, however, do not support these predictions (Hasan & Enoka, 1985; Leedham & Dowling, 1995).

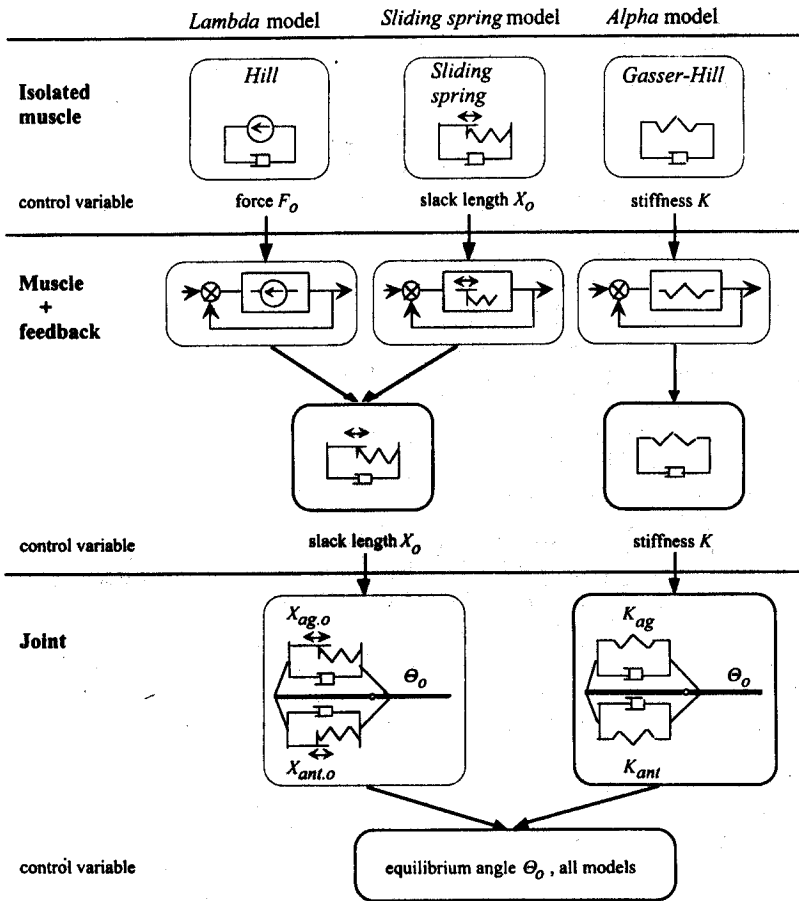


Figure 6 — Control variables in mechanical models of isolated muscle, muscle with sensory feedback, and joint in the EP models of control of movement. Control variables are the same in the Lambda and Sliding Spring models of muscle with intact afferents. However, sensory position feedback is essential in the Lambda but complementary in both the Alpha and Sliding Spring models. At the joint level, equilibrium angle is the control variable in all three models.

*Control of Movement Based on the Sliding Spring Model*

The Sliding Spring model is mathematically equivalent to the model of muscle with intact afferents included in Merton's (1953) "length servo" and the Lambda model. Moreover, models of muscle with intact afferents in the Lambda, Alpha, and Sliding Spring models lead to the same model of joint spanned by the antagonistic muscles (Figure 6). Consequently, joint equilibrium position is the control variable in all EP models.



Control of the joint equilibrium position in a model of control of movement based on the Sliding Spring model is realized by changing the slack lengths of the antagonistic muscles as described by Feldman (1966a). When maintaining a static position, the MU firing rates in the antagonistic muscles should be similar when the joint is in the middle of its angular range. For the joint to be held close to the boundaries of the range, the MU firing rate should increase in the shortened muscle and decrease in lengthened muscle. Also, the MU firing rate in a muscle holding against a constant load at different lengths should be higher at the shorter lengths. Intramuscular recordings showed that the MU firing rates increased with a decrease in length in isometrically contracting muscles (Vander Linden et al., 1991); (however, see Bigland-Ritchie et al., 1992). Note that the level of contraction was much higher in the latter study than in the former.

The Sliding Spring model of isolated muscle leads to a new interpretation of some previously published results. In this model, muscle and joint equilibrium trajectories are related to measurable patterns of MU firing rate and, provided that a relation between MU firing rate and recruitment is established, to surface EMG. In this context, the estimated N-shaped equilibrium trajectories during fast movements (Bellomo & Inbar, 1997; Latash & Gottlieb, 1991) reflected the *output* of the alpha motoneuron pool. From these data, however, no details can be directly inferred about the central and sensory *inputs* to the alpha motoneuron pool.

The Sliding Spring model of isolated muscle is applicable for the analysis of motor performance of neurologically normal as well as deafferented subjects. It is parsimonious in the sense that it can account for all the experimental data previously explained by either the Lambda or Alpha models. It can also be used in simulations of eye movements since oculomotor muscles exhibit spring-like behavior despite absence of the stretch reflex (Collins et al., 1975; Robinson, 1981). A model of control of movement based on the Sliding Spring muscle model has been used successfully in simulations of both postural and movement tasks (Shapiro, 1997).

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