Biol. Cybern. 66, 463–477 (1992)

Biological Cybernetics

A mathematical analysis of the force-stiffness characteristics of muscles in control of a single joint system ★

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Received February 15, 1991/Accepted December 9, 1991

Abstract. Feldman (1966) has proposed that a muscle endowed with its spinal reflex system behaves as a non-linear spring with an adjustable resting length. In contrast, because of the length-tension properties of muscles, many researchers have modeled them as non-linear springs with adjustable stiffness. Here we test the merits of each approach: Initially, it is proven that the adjustable stiffness model predicts that isometric muscle force and stiffness are linearly related. We show that this prediction is not supported by data on the static stiffness-force characteristics of reflexive muscles, where stiffness grows non-linearly with force. Therefore, an intact muscle-reflex system does not behave as a non-linear spring with an adjustable stiffness. However, when the same muscle is devoid of its reflexes, the data shows that stiffness grows linearly with force. We aim to understand the functional advantage of the non-linear stiffness-force relationship present in the reflexive muscle. Control of an inverted pendulum with a pair of antagonist muscles is considered. Using an active-state muscle model we describe force development in an areflexive muscle. From the data on the relationship of stiffness and force in the intact muscle we derive the length-tension properties of a reflexive muscle. It is shown that a muscle under the control of its spinal reflexes resembles a non-linear spring with an adjustable resting length. This provides independent evidence in support of the Feldman hypothesis of an adjustable resting length as the control parameter of a reflexive muscle, but it disagrees with his particular formulation. In order to maintain stability of the single joint system, we prove that a necessary condition is that muscle stiffness must grow *at least* linearly with force at isometric conditions. This shows that co-contraction of antagonist muscles may actually destabilize the limb if the slope of this stiffness-force relationship is less than an amount specified by the change in the moment arm of the muscle as a function of joint configuration. In a reflexive muscle where stiffness grows faster than linearly with force, co-contraction will always lead to an increase in stiffness. Furthermore, with the reflexive muscles, the same level of joint stiffness can be produced by much smaller muscle forces because of the non-linear stiffness-force relationship. This allows the joint to remain stable at a fraction of the metabolic energy cost associated with maintaining stability with areflexive muscles.

1 Introduction

In the mid sixties, Asatryan and Feldman (1965) and Feldman (1966) proposed a unique approach for development of a theory for human motor control. Their theory was based on a series of experiments which examined the mechanics of movement in the elbow joint. In a typical test, the forearm was placed in an apparatus that allowed for radial movements about the elbow joint in the horizontal plane. In each trial, the elbow flexors (or extensors) would be loaded and the subject was asked to maintain a predetermined elbow angle. Based on their observations, when the flexors were loaded, judging from the EMG, the extensors were not active, and vice versa. After a sudden decrease of the load, the forearm would transfer to a new position. The subject was told not to intervene voluntarily to correct this deflection of the arm.

A series of such experiments was repeated for the same initial elbow position and load, but each time the decrease in the load was changed. The set of points that resulted consisted of two variables: elbow angle and muscle torque. These points were connected and formed an exponential-like curve (Fig. 1) that was characteristic of that initial elbow angle (Feldman 1980, experimental data comes from Asatryan and Feldman 1965). This curve was called an invariant characteristic (IC) because it preserved its shape despite rather wide time-space variations in the load change procedure (results of Feldman 1986 have shown that an IC maintains its shape regardless of whether it is obtained

★ This work was supported in part by grant no. 1R01 NS 24926 from the NIH (Michael Arbib, PI). R.S. was supported by an IBM Graduate Fellowship in Computer Science

through a sudden increase or a decrease in the load that the muscle is maintaining).

In Fig. 1, each IC originates from a line which represents the torque that is produced at the elbow joint (for a given joint angle) by completely relaxed muscles. Feldman (1966) hypothesized that when a subject is asked to, for example, maintain a flexion load, he does this by down-loading a set of control parameters to the spinal cord which set up the reflex system as well as directly activating the motoneurons. However, when the load changes, since there is presumably no voluntary change in these parameters, what is observed is the response of the muscle-reflex system for that particular set of control parameters. The instruction given to the subject, "do not intervene," is interpreted as meaning that the central input to the spinal servo loop is constant (Crago et al. 1976). If the change in load causes a change in the firing frequency of the α-motoneurons, it must be the result of afferent feedback.

Because of the shape of these torque-angle curves, Feldman (1966) hypothesized that to the supra-spinal centers, the static force-length behavior of a muscle and its reflex system appears as a non-linear spring with an adjustable resting length. In fact, it was proposed that the only controllable parameter of the muscle-reflex system (available for voluntary manipulation) is the threshold length of this non-linear spring (Feldman 1986).

Many elements of the Feldman model are in sharp contrast to other muscle models proposed for description of movement (Stern 1974; Hof and Van den Berg 1981; Zheng et al. 1984; Hogan 1984; Winters and Stark 1985, 1987; Ramos and Stark 1987; Mussa-Ivaldi and Giszter 1991). We will show that all of these "other" models belong to a single class where the static force-length relationship of a muscle is a non-linear elastic element with an adjustable stiffness. How do these models relate to the work of Feldman? Evidence is provided which indicates that when a muscle is without its reflexive control system, it behaves as a non-linear spring with an adjustable stiffness. On the other hand, using stiffness-force data from an intact muscle-reflex preparation (Hoffer and Andreassen 1981), we will show that the static characteristics of a reflexive muscle does indeed appear to the supra-spinal centers as a non-linear spring with an adjustable threshold parameter, providing independent evidence in support of Feldman's hypothesis. The key point is that when the controllable parameter in a muscle is its stiffness, then stiffness is always a linear function of force at isometric conditions (which is what was observed by Hoffer and Andreassen 1981 in the areflexive muscle). But the stiffness-force relationship becomes non-linear when reflexes are present, from which we can derive a force-length relationship that agrees with the principle of Feldman's hypothesis, albeit not his particular formulation.

Is there a functional advantage when a muscle has a non-linear stiffness-force relationship? This question has especial relevance because we will show that in a single joint system (an inverted pendulum supported by a pair of muscles), for the limb to remain stable, a muscle cannot obey Hooke's law and act like a linear spring with constant stiffness. In fact, the limb will be stable if and only if the stiffness of each muscle increases with force at isometric conditions. When a muscle's stiffness grows faster than linearly with force (as in the reflexive muscles), posture can be maintained at a fraction of the energy cost as compared to the case where stiffness grows linearly with force (as in the areflexive muscles).

2 The Feldman model

Feldman's thesis is that the family of curves in Fig. 1 represent the static torque-angle relationship that is imposed by the CNS on the spinal motor control apparatus, and movement arises as a consequence of *(1)* external load change, or *(2)* a decision by the CNS to change the torque-angle relationship of the synergists that are being controlled from a given curve to another. Feldman (1966) recognized that the force developed by a muscle at steady state for a particular length is a function of many variables:

$$\phi = \Omega(\lambda, c_1, c_2, \ldots, c_n) \tag{1}$$

where ϕ is the force developed by a muscle at length λ with a composite control signal consisting of $c_i (i = 1, \ldots, n)$ representing the neural command that acts on the spinal motor control circuitry. Voluntary

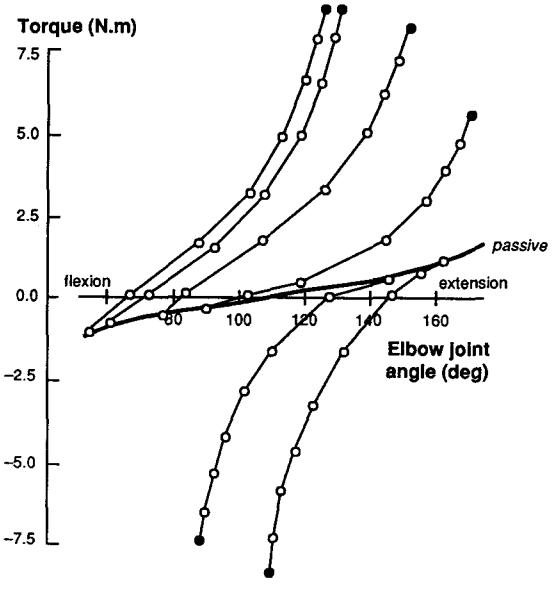

Fig. 1. A family of "invariant characteristics" (ICs) of elbow flexors (*upper curves*) and extensors (*lower curves*), measured through unloading the arm. The joint coordinate system refers to the angle of the elbow joint, and is 180° when the elbow is maximally extended. The instruction was "not to invervene voluntarily to forearm deflections when the load is removed." *Solid circles*: initial combination of muscle torque and elbow angle. *Open circles*: combinations of the same variables when they have settled after the unloading trials. Each IC merges with the characteristic of passive elbow joint (*heavy line*). (redrawn from Feldman 1980, Fig. 3A. The data from the figure recorded in Asatryan and Feldman 1965)

control of a muscle means changing the values of some of the independent variables c_i. These variables could represent gains or biases of the spindle feedback or Golgi tendon pathways, or the amount of "descending" activation received by the motoneuron pool.

It is generally believed that the essential components of the muscle-reflex system are as shown in the schematic of Fig. 2 (Houk and Rymer 1981). Here, a muscle produces a force which interacts with a load and leads to length changes. Muscle force depends on the level of activity of the skeleto-motoneurons as well as the operating length of the muscle. In this model, activity of the skeleto-motoneurons depends on three sources: *(i)* direct supraspinal activation, *(ii)* excitation from the spindle receptors mediated through interneurons in the segmental pathways, and *(iii)* inhibition from the Golgi tendon organs mediated through interneurons in the segmental pathways. The key assumption of Feldman (1966) is that an IC curve is produced when the CNS sets $c_i = \text{constant}$ for all $i = 1, \ldots, n$, and that this corresponds to a constant control signal in Fig. 2.

From Fig. 1, it appears that the effect of the operator Ω is to produce an exponential-like torque-angle relationship for a constant control vector **c**. Note that IC in Fig. 1 is the result of addition of three torque producing mechanisms: *(i)* a torque that is produced by the completely relaxed muscle (as judged by the absence of EMG) due to its passive mechanical property, *(ii)* a torque that is generated by the passive property of the antagonist muscle, accounting for the negative (or positive) torque region for the IC of the flexors (extensors), and *(iii)* a torque that is produced because of active contraction of the muscle. For example, in the case where the elbow joint is flexed to about 55°, the negative torque in the IC's of the elbow flexors is due to the passive properties of the stretched elbow extensors. Therefore the IC lines in Fig. 1 for the flexors include the passive properties of the extensors. In order to represent the torque-angle relationship for just the flexors, one need to account for this passive property of the antagonist muscles. In Fig. 3A we have done this by subtracting from the torque-angle relation of the flexors that region of the "passive" line in Fig. 1 where the torque was negative.

Each line in Fig. 3A is the static torque-angle relationship which results for a given control vector **c** in (1) for the elbow flexors. A new line is generated when the control vector changes. However, note that a change in **c** leads to more than just a shift in the torque-angle relationship; there is also a change in the shape of each line. Therefore, at least for the torque-angle relationship of this group of muscles, we cannot say that the control vector **c** is merely a threshold parameter beyond which torque develops. We can, however, consider the hypothesis that the change in the "shape"

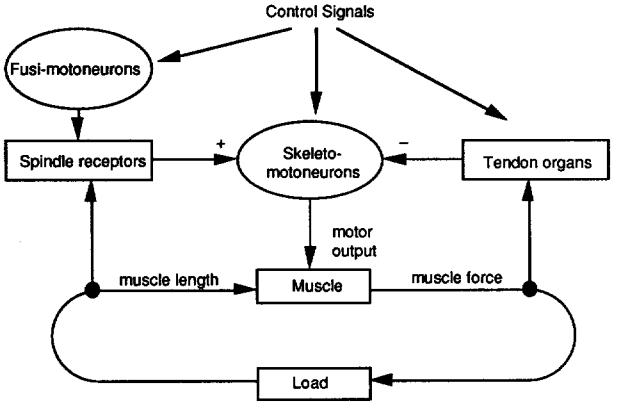

Fig. 2. Organizational plan of the autogenic reflex system acting on a muscle. Activation of a muscle produces a force which acts on a load to produce a length change. Muscle force is regulated by motor output from skeleto-motoneuron and also varies as a function of muscle length. Muscle length (and velocity) is monitored by spindle receptors and force by Golgi tendon organs. These signals provide excitation and inhibition, respectively, so skeleto-motoneurons by way of segmental (and possibly supra-segmental) pathways. Neural control signals are sent to skeleto-motor and fusi-motor neurons and to interneurons in the reflex pathway (from Houk and Rymer 1981)

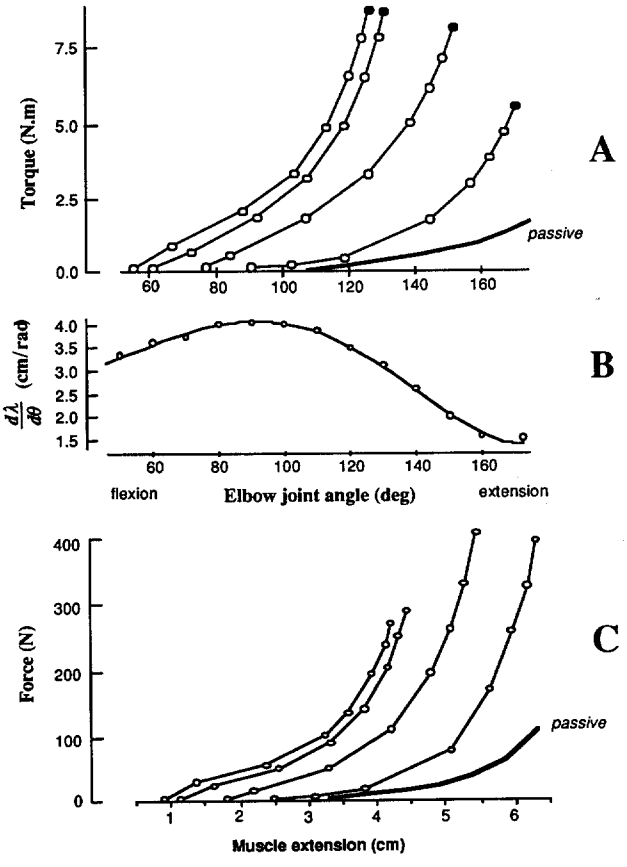

Fig. 3A–C. The static characteristics of elbow flexors during unloading tasks where the subjects were told not to intervene voluntarily to correct the deflection of the arm. Here we derive the force developed in the muscles from joint torques as a function of arm configuration. **A** The torque developed in elbow flexors. These are the curves in Fig. 1, but here we compensate for the effect of the passive torque as developed by the elbow extensors. **B** The moment arm for the biceps as a function of elbow joint angle. Data points from Feldman (1966), Fig. 3C. The fitted function is a fifth order polynomial. **C** Computed force as a function of muscle stretch

of the torque function is mainly due to the change in the moment arm of the muscles as the joint rotates. This is the argument that Feldman (1966) used to suggest that muscle force as a function of joint angle can indeed be approximated as a non-linear spring with an adjustable threshold length. In order to show this, we have used Feldman's (1966) model regarding the change in the moment arm of biceps brachii (an elbow flexor), as shown in Fig. 3B, to transform the torque-angle relation in Fig. 3A to a force-length relationship in Fig. 3C. The procedure is as follows: Initially, we need to derive the mathematical relationship between the force developed by the muscle, denoted by variable ϕ, and the resulting joint torque τ. This depends on the kinematics of the joint and how muscle length changes as a function of joint angle. If we assume that skeletal segments are rigid links and the muscles have a single point of origin and insertion (e.g., Hogan 1985), then the kinematics can easily be defined and the principle of virtual work (Craig 1986) can be used to relate joint torque to muscle force. We summarize the principle as follows:

The principle of virtual work

When forces act on a mechanism, work is done if the mechanism moves through a displacement. Work is defined as force acting through a distance, is a scalar with units of energy, and is the same regardless of the coordinate system in which it is measured. The principle of virtual work allows us to make certain statements regarding the transformation between forces in different coordinate systems by allowing the displacement to go infinitesimal. In our case, we have forces produced by muscles which act on a multi-joint skeletal system, and we want to understand how these forces can be represented in terms of joint torques. Initially, we represent work in each coordinate system, where work is the dot product of vector force or torque and a vector displacement. Assuming the "right hand rule," where a positive torque indicates an increase in the joint angle, work in each coordinate system is represented by the following equality:

$$-\phi \,.\, \mathrm{d}\lambda = \tau \,.\, \mathrm{d}\theta \tag{2}$$

where ϕ is an $n \times 1$ vector of muscle forces, $\mathrm{d}\lambda$ is an $n \times 1$ vector of infinitesimal stretch of the muscles, τ is an $m \times 1$ vector of torques at the joint, and $\mathrm{d}\theta$ is an $m \times 1$ vector of joint displacements. Assume that muscles can only pull, and this force is defined to be positive. The reason for the negative sign on the left part of this equality is as follows: Consider the case when a muscle is stretched. This requires positive work by the mechanism which stretches the muscle, thereby transferring energy to the muscle, which in turn stores this energy and releases it when the muscle shortens. Since muscle force has a direction that is always opposing the direction of length change for which positive work is done, the left side of (2) must have a negative sign in order for a stretch (positive length change) to result in positive work. On the right side of this equality, torque acting on a joint and the resulting displacement have the same direction, resulting in positive work. We can rewrite (2) as: $-\phi^T \mathrm{d}\lambda = \tau^T \mathrm{d}\theta$, where $\mathbf{x}^T$ is the transpose of $\mathbf{x}$. It is useful to represent the differential transformation from joint angles to muscle lengths by the Jacobian $\mathbf{J}_M$, which satisfies the following: $\mathrm{d}\lambda = \mathbf{J}_M \, \mathrm{d}\theta$, so that we may rewrite (2) as: $-\phi^T \mathbf{J}_M \mathrm{d}\theta = \tau^T \mathrm{d}\theta$, which must hold for all $\mathrm{d}\theta$, and so we have: $-\phi^T \mathbf{J}_M = \tau^T$. Transposing both sides:

$$\tau = -\mathbf{J}_M^T \phi \tag{3}$$

For a muscle acting on a single joint, this reduces to:

$$\tau = -\frac{\mathrm{d}\lambda}{\mathrm{d}\theta}\phi \tag{4}$$

Equation (4) gives us a way to transform joint torques in Fig. 3A to muscle forces, if we knew the moment arm of elbow flexors (i.e., $\mathrm{d}\lambda/\mathrm{d}\theta$). For the biceps (an elbow flexor), Feldman (1966) has approximated $\mathrm{d}\lambda/\mathrm{d}\theta$. We fitted a fifth order polynomial to his data (Feldman 1966, Fig. 3C), and have plotted the results in Fig. 3B. The curve fit equation is as follows:

$$\frac{\mathrm{d}\lambda}{\mathrm{d}\theta} = 2.8 - 0.02\theta + 7.4 \times 10^{-4}\theta^2 - 2.3 \times 10^{-7}\theta^3 - 6.4 \times 10^{-8}\theta^4 + 2.5 \times 10^{-10}\theta^5 \tag{5}$$

Figure 3c is the computed muscle force for the elbow flexors as a function of muscle length. Muscle length was derived by solving the differential equation in (5)[1]. From Fig. 3C it appears that the static force-length relationship of a muscle depends strongly on a threshold parameter beyond which muscle force is developed. Feldman (1966) has argued that since there is little change in the shape of this force function as the threshold parameter changes, the force-length relation of a muscle may be represented by the following model:

$$\phi = \Omega(\lambda - \beta) \tag{6}$$

where the control parameter β has the units of length and the operator Ω specifies the "invariant" relationship between muscle force and length. Based on the measured and derived data, Feldman (1966) proposed that muscle force as a function of the control parameter β is an exponential function:

$$\phi = k(\exp(\alpha(\lambda - \beta)) - 1) \tag{7}$$

which basically says that the equivalent circuit model of the muscle-reflex system, from the point of view of the supra-spinal control centers, is a non-linear spring with an adjustable resting length.

In order to arrive at this model, Feldman (1966) has made two important assumptions; *(i)* that the central control signals descending on the servo loop (as in Fig. 2) are not varied during sudden changes in load, and *(ii)* in Fig. 3B, we have a reasonable model of how the

[1] Since the initial condition for the differential equation in (6) is not known, the length axis of Fig. 3C is muscle stretch beyond an arbitrary constant, rather than absolute muscle length

moment arm of the elbow flexors, taken as a whole, changes as a function of joint angle. Unfortunately, both assumptions are difficult to verify in humans. However, one could test the validity of the model in (7) in a decerebrate animal by simulating the effect of the first assumption through systematic alteration of the control signals to the motor servo, while bypassing the second assumption through direct measurement of the developed muscle force as a function of muscle extension.

Feldman and Orlovsky (1972) have taken this approach in measuring the static force-length behavior of the reflexive gastrocnemius in a decerebrated cat. Their results suggest: *(i)* an exponential-like force-length relationship for a constant central input, and *(ii)* a simple shift in the threshold of the force-length relation as the central input is varied. Figure 4A shows a family of force-length curves obtained by Feldman and Orlovsky (1972) at different stimulation levels of Deiters' nucleus (a cerebellar nucleus). Force was measured from the gastrocnemius muscle while it was stretched slowly to its maximal physiological length. Deiters' nucleus stimulation at fixed rate was delivered before and throughout the period of stretching to mimic a constant level of central motor command. The salient effect of a change in stimulation was a shift in the force-length relation along the abscissa, i.e., a change in the "threshold" with little change in the shape of the curve. Excitatory input lowered this threshold, while inhibitory input moved it to a longer muscle length. The same qualitative results were obtained when stimulation was delivered to the pyramidal tract and the reticular formation.

From the data in Fig. 4A we can ask whether the model in (7) is an accurate description of the static force-length behavior of the gastrocnemius muscle and its associated spinal reflex pathways in the cat: logarithmic transformation of the points belonging to a single curve in Fig. 4A should result in a straight line. In Fig. 4B we have plotted the function $\ln(1+\phi)$, where ϕ is the muscle force in Fig. 4A. Although the model of (7) appears to be an accurate description of the data for medium and large forces, it fails to capture the subtle behavior for smaller forces. Based on this, it would appear that Feldman's (1966) model in (7) can be improved upon. In the next section, we will take up this issue when we consider the role of the reflex system in the development of this force-length relationship, i.e., how much of the force is due to intrinsic muscle stiffness, and how much due to the action of the stretch reflex.

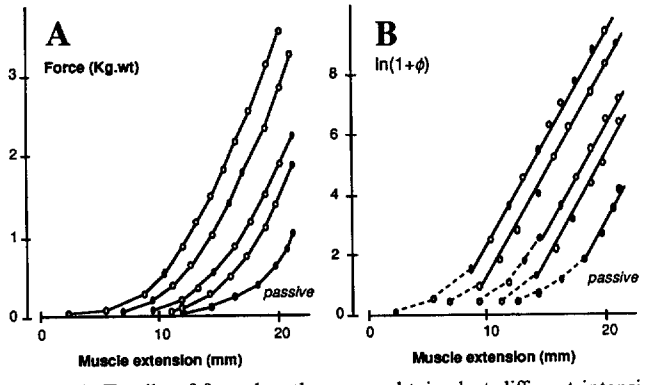

Fig. 4. **A** Family of force-length curves obtained at different intensities of Deiters' nucleus stimulation. Reflex force was registered while the muscle (gastrocnemius in decerebrate cat) was stretched slowly to its maximal physiological length. To arrive at each curve, a constant level of stimulation was delivered before and throughout the period of stretching to mimic a constant level of central motor command. The larger muscle force was obtained at higher stimulation intensities (redrawn from Feldman and Orlovsky 1972). **B** Test of whether the curves in **A** are of exponential shape. $\ln(1+\phi)$ is plotted as a function of muscle extension, where ϕ is muscle force. If the curves in **A** fit the model $\phi = k\exp(a(\lambda-\beta))-1$, then all the points in **B** should fit a set of parallel lines. For small muscle forces, there is a poor fit

3 Two types of muscle models

Although the term β in (7) has been suggested by Feldman to be the parameter that the nervous system uses to control the static behavior of a muscle-reflex system, its physiological basis remains unclear. Static muscle force depends on muscle length because of the mechanical stiffness of the muscle, and because of the role of the stretch reflex in increasing motoneuron activity: The traditional explanation of the stretch reflex is that when the whole muscle is stretched, since the spindle organs are mechanically in parallel with the muscle, they are stretched by the same relative amount (Ghez 1985). But it has been argued that the feedback gain of this loop (which, after all, is responsible for further activating the muscles as it is stretched) is simply too low to account for the appreciable gain in force that is obtained in Fig. 1 as the muscle is stretched beyond its set length (Matthews 1981). In fact, many computational models of the muscle-skeletal system tend to view muscles as spring-like elements with variable stiffness, rather than variable resting length (Stern 1974; Hof and Van den Berg 1981; Zheng et al. 1984; Hogan 1984; Winters and Stark 1985, 1987; Ramos and Stark 1987; Mussa-Ivaldi and Giszter 1991). In what follows we discuss implications of both kinds of approach and present a discussion which will reject the variable stiffness model as an accurate description of the static force-length behavior of a muscle.

Models which view a muscle as a non-linear spring with variable stiffness are usually based on data such as that shown in Fig. 5 (from Rack and Westbury 1969), which describes the steady-state tension measured in an isolated soleus muscle vs. muscle length for various levels of activation (Fig. 5A), and tension vs. activation rate for various muscle lengths (Fig. 5B). For example, in the work of Hogan (1984), a muscle was modeled as a linear spring with variable stiffness (a change in the activation of the muscle led to a change in the muscle's stiffness), and this approximation was used to predict the amount of co-activation in the elbow muscles of the human arm for a lifting task. In the work of Mussa-Ivaldi and Giszter (1991), the condition for linearity of

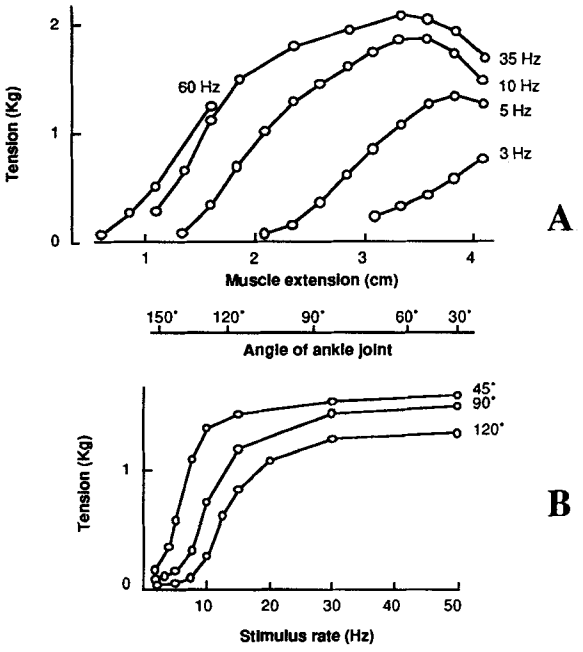

Fig. 5A,B. Static force-length behavior obtained from electrically stimulated areflexive cat soleus muscle under isometric conditions (from Rack and Westbury 1969). **A** The effect of length on active tension at different stimulus rates. **B** The effect of stimulus rate on active tension at three different muscle lengths

the spring was removed and the static behavior of a muscle was described by a factored form of a functional dependence of the output force upon a length parameter λ and activation parameter u:

$$\phi = h(u)g(\lambda) \tag{8}$$

where u is meant to represent the amount of depolarization received by the motoneurons of the particular muscle. This basic formulation also describes the static force-length relationship of muscle models used in Stern (1974) (cf., their p. 413, equation for P), Hof and van den Berg (1981) (cf., their equation 2), Winters and Stark (1985) (cf., their Fig. 2b and equation 4), and Winters and Stark (1987) (cf., the contractile element's active muscle torque-angle process, their p. 407): In all these cases, initially a force-length relationship for a maximally activated muscle is described and then a change in activation is represented by a scaling process (note that force in 8 may be a non-linear function of activation, as is the case in Fig. 5B). For example, in Zheng et al. (1984), a muscle model based on the work of Hatze (1977) was described where steady-state force developed in a single muscle fiber was:

$$h(u) = a_1(1 - b_1 \exp(a_2 u) - b_2 \exp(a_3 u))$$

$$g(\lambda) = c_1 + c_2 \exp(c_3 \lambda) \sin(c_4 \lambda) \tag{9}$$

where $h(u)$ produces a saturating exponential relation similar to that of Fig. 5B, and $g(\lambda)$ is defined over a range for λ (i.e., muscle length) such that it initially rises to a maximum and then declines, resembling to some extent the behavior in Fig. 5A[2]. In Zheng et al. (1984), the force developed in a whole muscle is a sum of the forces developed in single muscle fibers (since the fibers are more or less in parallel).

Although with this kind of model one can produce any force at any muscle length (the same can be said for the Feldman type muscle model), there is a crucial difference between it and the formulation in (7): The formulation in (8) suggests that the control parameter u has the units of stiffness (force divided by length), rather than length (which is the unit of the control parameter in the Feldman-type model). This crucial difference between the models will allow us to refute the stiffness control model based on experimental data.

Theorem 1. *If static muscle force $\phi(\lambda, u)$ can be written in factored form $\phi(\lambda, u) = h(u)g(\lambda)$, then $d\phi/d\lambda$, i.e., muscle stiffness, evaluated at some length λ_0, is proportional to $\phi(\lambda_0, u)$.*

$$\left.\frac{d\phi}{d\lambda}\right|_{\lambda_0} = h(u)\left.\frac{dg(\lambda)}{d\lambda}\right|_{\lambda_0} = \phi(u, \lambda_0)g^{-1}(\lambda_0)\left.\frac{dg(\lambda)}{d\lambda}\right|_{\lambda_0}$$

Note that since the function g only depends on λ, all the g terms in the above equalities are constants determined by λ_0, and therefore not a function of u. It follows that if at some operating length, stiffness is not proportional to force, then force is not a factorizable function of u and λ.

The experimental data of Hoffer and Andreassen (1978, 1981) allow us to reject the model of (8): In their work, decerebrate cats were held rigidly and a force increment was measured as the soleus muscle was stretched by a small amount. When the experiment was repeated over a wide range of different initial levels of muscle force, it was reported that: *(i)* stiffness of the muscle-reflex system increased with force at low force levels, but soon reached a plateau, so that stiffness was almost constant at moderate and high force levels (Fig. 6), and *(ii)* the force-stiffness curve was found not to vary as a function of operating muscle length. The first conclusion contradicts the predictions of (9).

This result suggests that the static force developed in a muscle cannot be modeled as a factorizable function of muscle length and activation. In fact, it also contradicts the predictions of Feldman's formulation in (7) since $d\phi/d\lambda$ (stiffness) in this model is also a linear function of ϕ (force) at a given muscle length. However, recall that (7), i.e., Feldman's (1966) model, was not a very good representative of the experimental data of Feldman and Orlovsky (1972) for small muscle forces, as illustrated in Fig. 4B. We will show that although the spirit of the Feldman's hypothesis (as in 6) can be supported by the Hoffer and Andreassen (1981) data, its particular formulation needs to be changed.

Based on the results of Hoffer and Andreassen (1981), the stiffness characteristics of a reflexive muscle

[2] The rationale for this formulation of $g(\lambda)$ is based on the work of Gordon et al. (1966). We discuss this work in section 4 where we describe the behavior of a single muscle fiber

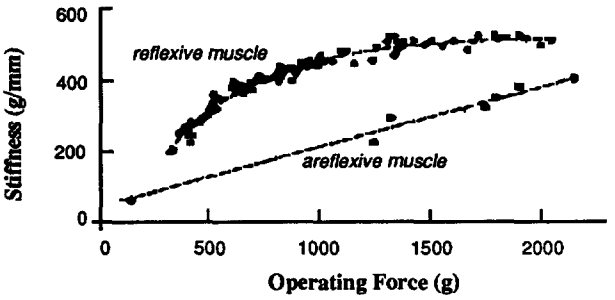

Fig. 6. Muscle stiffness vs. force in the cat soleus. When the stretch reflex is intact, muscle stiffness is a sharply rising function of force at the low end of the force range, but remains nearly constant at moderate and high forces. By contrast, when the reflex is eliminated by cutting the efferent soleus nerve and electrically stimulating the cut end to maintain tension, the isolated muscle shows a lower stiffness which is an approximately linear function of force (from Hoffer and Andreassen 1978)

(i.e., a muscle with its spinal reflex system) can be approximated by:

$$\frac{d\phi}{d\lambda} = k(1 - \exp(-\alpha\phi)) \tag{10}$$

which presumes that the stiffness-force curve goes through the origin. This is a reasonable assumption because at zero force, a muscle will have a positive stiffness only if it happens to be at that length beyond which passive mechanical force develops (at zero force, this stiffness cannot be negative because it implies that the muscle produces a negative force as it is stretched). This passive stiffness is likely to be a fraction of the stiffness measured at maximum muscle tension (McMahon 1984) and is ignored in the formulation of (8).

The solution to the differential equation in (10) is:

$$\phi = \frac{1}{\alpha} \ln(\exp(\alpha k(\lambda - \beta)) + 1) \tag{11}$$

where β is the constant of integration and depends on the initial conditions for (10). Unfortunately, it is not possible to define β precisely because the initial condition for this kind of experiment cannot be known: In the decerebrate animal, reflex response of a muscle is measured at various initial force levels, and changing this initial muscle force is done by stroking the fur, blowing on the face, or by moving the contralateral limb (Nichols and Houk 1976; Hoffer and Andreassen 1981). However, it is clear that the only way muscle force can be varied at some operating length is for the animal to vary β, therefore β can be thought of as a controllable parameter (note that it has to have the units of length).

Consider how force varies as a function of length λ in (11): At a given β, when λ is such that $\exp(\alpha k(\lambda - \beta)) \ll 1$, then force in (11) is approximately $1/\alpha \exp(\alpha k(\lambda - \beta))$, i.e., when the control parameter is such that the muscle is producing a small force, as we stretch it to new lengths, force will grow exponentially with length (i.e., faster than linearly). When λ is such that $\exp(\alpha k(\lambda - \beta)) \gg 1$, then force is approximately $k(\lambda - \beta)$, i.e., when the muscle is producing a large force, as we stretch it, force should grow linearly with length.

By varying β, i.e., the only controllable parameter of the system, one is in effect changing the *resting length* of a non-linear (exponential-like) spring. Interestingly, it can be shown that the formulation in (11) is an excellent estimate of the data in Fig. 4A. A measure of goodness of fit for this model can be visualized by plotting the function $\ln(\exp(\alpha\phi) - 1))$ for the data points in Fig. 4A. Our result should be a family of straight lines with the same slope. This is in fact the case, as is illustrated in Fig. 7 (except for the passive curve, which does not follow the behavior of an actively controlled muscle). Therefore it appears that the model derived from the data of Hoffer and Andreassen (1981) strongly supports the contention that a muscle and its associated reflex system can be viewed as a non-linear spring with a variable resting length, and is a better model of the Feldman and Orlovsky (1972) data than the formulation in (7).

Hoffer and Andreassen (1981) also reported on the stiffness of the areflexive muscle (i.e., a muscle without the reflex system) after cutting the efferent soleus nerve and electrically stimulating the cut end to produce various force levels. Their results are shown by the straight line in Fig. 6. The isolated muscle shows a lower stiffness which is a steadily rising function of force. Based on there results we can conclude that the model in (8), where a muscle is seen as a non-linear spring with variable stiffness, cannot express the observed non-linear stiffness-force characteristics of a reflexive muscle, although its predictions regarding a linear stiffness-force relationship appears to be valid for an areflexive muscle[3].

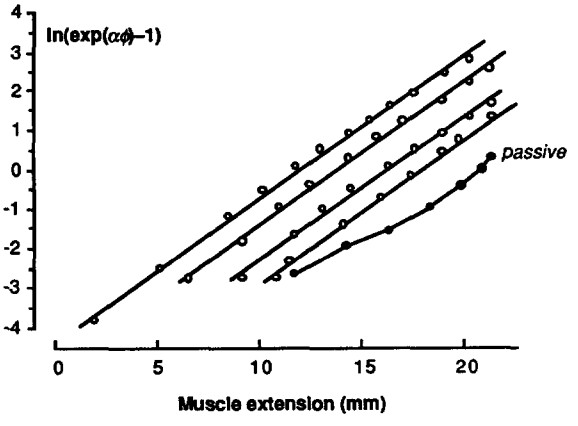

Fig. 7. A test of the hypothesis that the family of force-length curves obtained at different Deiters' nucleus stimulation by Feldman and Orlovsky (1972) (in Fig. 4A) represents the model of (7). If so, then the transformation $\ln(\exp(\alpha\phi) - 1)$ should result in a family of straight lines with constant slope. This prediction matches the behavior of the data, except for the passive length-tension curve

[3] The basic results of Fig. 6 regarding behavior of a reflexive muscle have been validated in other experiments (Greene and McMahon 1979; Allum et al. 1982; Nichols 1985)

What has not been discussed thus far is to what extent the force-length relationship in (11) is due to the inherent mechanical properties of the muscle: Recall that two factors contribute to the compensatory force developed by a muscle which undergoes a length change, one is recruitment and variation in frequency of the motor units because of reflex action, and the other is from the mechanical properties of the muscle (its force-length relationship). In order to establish the contribution of each factor to the observed force-length relationship in Fig. 1, Vincken et al. (1983) repeated the experiments of Feldman while recording motor unit activity from the loaded elbow muscles. Subjects were asked to move their arms to a prescribed position against an applied torque, and unexpected changes in the torque were made while activity of a single motor unit was recorded. The torque-joint angle relationship obtained matched the findings of the Feldman experiments. Firing frequency of motor units changed significantly when the torque acting on the joint was altered and a new position was reached: The direction of change in torque, e.g., an increase, corresponded to the same direction of change in the firing frequency. For a given motor unit, a plot of firing frequency vs. joint torque was obtained: The larger the torque acting on the joint, the larger the firing frequency. In order to determine the contribution of the elastic properties of the muscle to the restoring force, motor unit firing frequencies were recorded for a large number of arm positions and joint torques. This allowed the authors to construct isotonic torque-joint angle plots for a given motor unit. Because there was a very small change in the muscle torque as a function of muscle length (for the entire tested region of the joint space) at a given frequency of motor unit firing, Vincken et al. (1983) concluded that the influence of intrinsic mechanical properties of the muscle on the recorded force-length relationship is very small, and nearly all of the restoring force is due to an increase in the motor unit firing frequency and recruitment of new motor units. Since this is presumed to occur only through reflex action (the subjects being told not to intervene voluntarily), it appears that afferent feedback plays a determinant role in establishing the exponential-like force-length behavior for constant central activation.

It is worth noting that in Fig. 6, at maximum force, stiffness of the reflexive muscle is only marginally greater than that of the areflexive muscle, so one cannot say that the reflexes significantly increase the maximum stiffness of a muscle. However, at smaller muscle force, there is a marked increase in stiffness (experiments of Vincken et al. 1983 were presumably conducted in this region). Therefore the question is, what is the advantage of the non-linear relationship between force and stiffness in the intact muscle-reflex system, i.e., what is gained by having a muscle which has its stiffness at near maximum levels at a fraction of the maximum force?

In order to answer this question, in the remainder of this work we have compared the characteristics of each type of muscle when they are used to control a single joint system. This modeling effort will also be useful in solidifying the concept of maintaining an equilibrium position, modulating joint stiffness through co-activation of the muscles, and introduces some issues of stability. It turns out that because in an areflexive muscle, stiffness grows linearly with force, and because of the non-monotonic nature of the muscle moment arm (e.g., Fig. 3B), it is possible that co-contraction may actually de-stabilize the limb. A stable equilibrium position is not guaranteed simply because muscles have positive stiffness. The advantage of the non-linear stiffness force relationship in the reflexive muscle are readily observed in this framework.

4 A model of an areflexive muscle

The terms areflexive and reflexive are a short-hand way of separating the instances of a muscle which is without its spinal reflex control circuitry, and one which is behaving under normal, intact conditions. In this section we provide a model of an areflexive muscle and show that it does indeed exhibit the linear relation of stiffness to force shown in the lower trace of Fig. 6. Our approach is to describe a two component active state model of an areflexive muscle, where the two components are composed of an elastic component in series with a contractile component (Fig. 8A). The "active" component of the force is due to the action of the pure force generator. This is the model proposed by Gasser and Hill (1924) to explain the tension dynamics of various isolated frog muscles. We can write the force-length relationship of this model by relating the force produced by each component at the node (a) of Fig. 8A:

$$\phi = K_{SE}(\lambda_1 - \lambda_1^*) \tag{12}$$

$$= K_{PE}(\lambda_2 - \lambda_2^*) + B\frac{d\lambda_2}{dt} + P(\lambda, f(t)) \tag{13}$$

where K_{SE} is the series elastic component which lies in the contractile machinery, K_{PE} is the parallel elastic component which with K_{SE} accounts for the passive tension properties of the muscle, B represents the viscous resistance opposing force development during shortening, and $P(\lambda, f(t))$ is the active force produced by the contractile mechanism in the muscle (which depends on the length of the contractile element and the history of muscle activation $f(t)$). λ_1 and λ_2 are the lengths of the series and parallel elastic elements and λ_1^* and λ_2^* are the resting lengths of these elements. We can derive the differential equation relating muscle force ϕ to muscle length λ and muscle resting length λ^* (i.e., the length beyond which a passive force develops) with the following procedure: Noting that $\lambda = \lambda_1 + \lambda_2$ and $\lambda^* = \lambda_1^* + \lambda_2^*$ (Glantz 1974), (13) can be rewritten as:

$$\phi = K_{PE}(\lambda - \lambda^*) - K_{PE}(\lambda_1 - \lambda_1^*) + B\left(\frac{d\lambda}{dt} - \frac{d\lambda_1}{dt}\right) + P(\lambda, f(t))$$

Using the relation in (12) we have:

$$\phi = K_{PE}(\lambda - \lambda^*) - \frac{K_{PE}}{K_{SE}}\phi + B\frac{d\lambda}{dt} - \frac{B}{K_{SE}}\frac{d\phi}{dt} + P(\lambda, f(t))$$

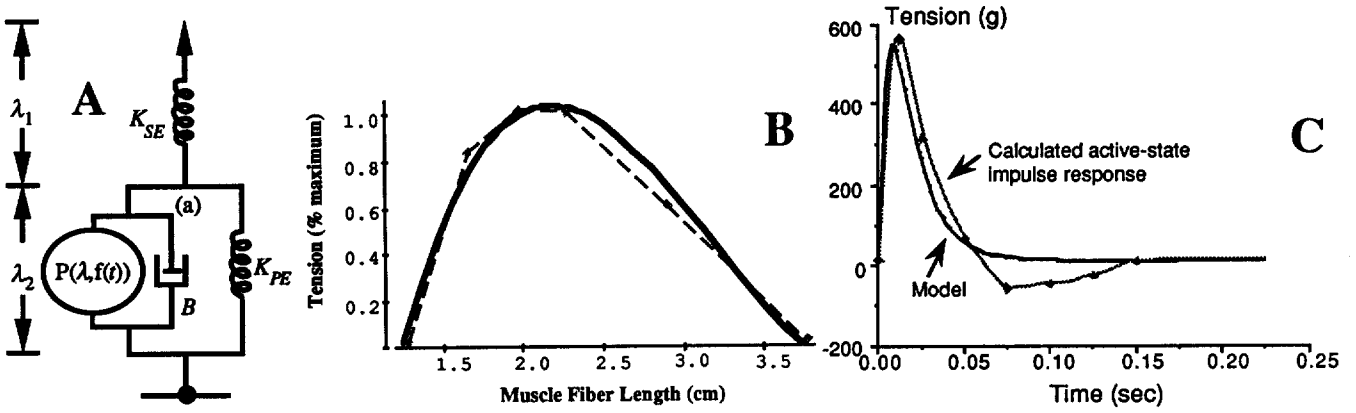

Fig. 8A–C. An active state muscle model. **A** The mechanical model of the muscle: λ_1 and λ_2 are taken as the displacement about the resting length, while $P(\lambda, f(t))$ is the active tension developed by the force generator. **B** Active tension vs. length for a single fiber of frog semitendinosus muscle (dashed line and data points from Gordon et al. 1966). This function was approximated by a third order polynomial: $S(\lambda) = -6.3 + 8.1\lambda - 2.9\lambda^2 + 0.3\lambda^3$. **C** The estimated impulse response of the active state component of the frog gastrocnemius muscle (redrawn from Inbar and Adam 1976), and the model approximation: $h(t) = 1200(\exp(-70t) - \exp(-210t))$

Solving for the rate of change in force:

$$\frac{d\phi}{dt} = \frac{K_{SE}}{B}\left(K_{PE}\Delta\lambda + B\frac{d\lambda}{dt} - \left(1 + \frac{K_{PE}}{K_{SE}}\right)\phi + P(\lambda, f(t))\right) \tag{14}$$

where $\Delta\lambda = \lambda - \lambda^*$, i.e., the displacement beyond the resting length (length at which passive force develops). The forces which develop at lengths smaller than λ^* are solely due to the action of active component. The length that a muscle occupies in the body is thought to be at or very near this length (McMahon 1984, chap 1).

The function $P(\lambda, f(t))$ is the active force produced by the contractile component of the muscle and depends on the history of muscle stimulation and the operating length. When a muscle is stimulated, a number of muscle cells become depolarized (each cell is an individual muscle fiber). Sufficient depolarization leads to a calcium release from the sarcoplasmic reticulum which eventually leads to formation of crossbridges and development of tension (Huxley 1974). In a depolarized muscle fiber, the tension developed is a function of the degree of overlap between thick and thin filaments in the sarcomeres (Huxley 1974). Gordon et al. (1966) experimented on a single fiber of a frog muscle and found that the developed tension was an increasing function of fiber length and reached a plateau at a length corresponding to that which the muscle occupies in the body, and then declined as the muscle was stretched beyond this point (this is the $g(\lambda)$ that was used in Zheng et al. (1984) in the discussion of Sect. 3). Results of Gordon et al. (1966) and our curve-fit (a third order polynomial) are plotted in Fig. 8B. Let us call this function $S(\lambda)$: it represents the tension in a single muscle fiber as a percent of maximum tension in that fiber.

It turns out that for different muscle fibers, the muscle length for which $S(\lambda)$ is a maximum (call this length λ_m) is not constant. For different muscle fibers of the same muscle, λ_m, obeys a statistical distribution whose variance depends on the type of muscle (Hatze 1977). In the whole muscle, the effect of this is that the region of near-maximum tension is sometimes broader than in the isolated fiber (Hatze 1977). However, the discussion for the remainder of this section is independent of the particular function used to approximate $S(\lambda)$ for a single muscle fiber.

In a whole muscle, if we assume that muscle fibers are arranged in parallel (Hatze 1977, a reasonable assumption for a large number of muscles[4], e.g., sartorius, McMahon 1984), then the tension produced by each fiber adds to the tension present in other fibers:

$$P(\lambda, f(t)) = \sum_{i=1}^{q(f(t))} c_i S_i(\lambda)$$

where $q(f(t))$ is an integer specifying the number of muscle fibers depolarized (as a function of the history of activation), and $c_1 S_i(\lambda)$ is the tension produced by the depolarized muscle fiber i. If we further assume that a muscle is composed of a nonhomogeneous set of muscle fibers and this property is represented by a scaling of $S(\lambda)$ (which presumes that the variance of λ_m is small), we have:

$$P(\lambda, f(t)) = S(\lambda) \sum_{i=1}^{q(f(t))} c_i = S(\lambda) y(t)$$

Suppose that the input function $f(t')$ is the temporal history of activation for $t' \leqslant t$. Using a linear systems approach, the active force developed in the contractile

[4] There are two types of arrangements for muscle fibers in a muscle (Pansky 1979). Fibers are either arranged in parallel to the long axis of the muscle, or obliquely. In the oblique case, muscle fibers are arranged as in a feather (this organization is called pennate), and tension developed in each fiber results in a force vector that is not directed along the long axis of the fiber. In this kind of muscle, the fibers are arranged symmetrically along the long axis of the muscle so the total force in the muscle is directed along the long axis

mechanism can be written in terms of its impulse response:

$$P(\lambda, f(t)) = S(\lambda) \int_{-\infty}^{t} f(t')h(t-t')\,dt' \qquad (15)$$

Using a parameter estimation technique, Inbar and Adam (1976) have described $h(t)$ for a frog gastrocnemius muscle. We approximated their results by a difference of two exponentials: $h(t) = 1200(\exp(-70t) - \exp(-210t))$, where the unit of tension is in grams[5]. In Fig. 8C we have plotted our approximation of $h(t)$ along with the results of Inbar and Adam (1976). Note that the impulse response of the contractile component in the experimental data is composed of a very large positive force region followed by a small negative force region. In the present paper we only modeled the positive force region since it is not clear how a muscle can produce an acive negative force[6].

Figure 9 shows the simulation results of this muscle model during isometric conditions (at $\lambda = \lambda^*$) for various simulation rates (the input to the system was modeled as a series of unit impulses with frequency of $1/\Delta t$). For the general model, we used the parameter values estimated by Inbar and Adam (1976) (in their Table 2) for a frog gastrocnemius muscle: $K_{SE} = 1500$ g/cm, $K_{PE} = 1460$ g/cm, and $B = 225$ g.s/cm. The overall isometric response of our model compares favorable with the experimental data of Inbar and Adam (1976): Maximal tension (185 g), the frequency of activation required for tetanus (79 Hz), contraction time (40 ms), half relaxation time (54 ms), and twitch-tetanus ratio (0.27) are all with in 12% of the experimental data.

Since our intention is to eventually use this muscle model for controlling a limb, it is useful to know how much activation we need to provide in order to produce a given amount of force at a given muscle length, and how stiffness varies as a function of muscle parameters. Therefore, we solved the differential Eq. of (14) and derived an expression for force (units are in grams) produced in the entire muscle as a function of the number of impulses, n, and their period, Δt (the derivation is summarized in Appendix 1). The following is the force produced by the muscle of (14) at Δt seconds after the arrival of the nth impulse, i.e., just before the arrival of impulse number $n+1$:

$$\phi = \xi a_1 S(\lambda)\left(\frac{\exp(-210\Delta t)}{210 - a_1 a_2} - \frac{\exp(-70\Delta t)}{70 - a_1 a_2} + a_3 \exp(-a_1 a_2 \Delta t)\right) + \frac{1}{a_2}\left(K_{PE}\Delta\lambda + B\frac{d\lambda}{dt}\right)(1 - \exp(1 - a_1 a_2 n \Delta t)) \qquad (16)$$

where

$$\xi = 1200\,\frac{\exp(-a_1 a_2 n\Delta t) - 1}{\exp(-a_1 a_2 \Delta t) - 1}$$

$$a_1 = K_{SE}/B$$

$$a_2 = 1 + K_{PE}/K_{SE}$$

$$a_3 = \frac{1}{70 - a_1 a_2} - \frac{1}{210 - a_1 a_2}$$

In Fig. 10 we have used (16) to describe the tension developed in this areflexive muscle as a function of stimulation rate at $n\Delta t = 1$ s, i.e., we wait one second after we begin the stimulation to measure the force in the muscle. This graph gives us a way to go from a desired muscle force to the activation necessary to produce that force in isometric conditions.

From (16) we can prove that for a reflexive muscle, force and stiffness at some operating length λ_0 will be linearly related. The first point to note is that because $a_1 a_2 \gg 1$ (in our case, it is 13), in (16), the term $\exp(1 - a_1 a_2 n\Delta t)$ is almost zero at $n\Delta t = 1$. Therefore the steady-state force will be related to frequency of stimulation Δt as follows:

$$\phi(\lambda, \Delta t) = bS(\lambda)g(\Delta t) + \frac{K_{PE}}{1 + K_{PE}/K_{SE}}(\lambda - \lambda^*)$$

where $g(\Delta t)$ and b are substitutions for the frequency dependent and constant terms in the first term of (16).

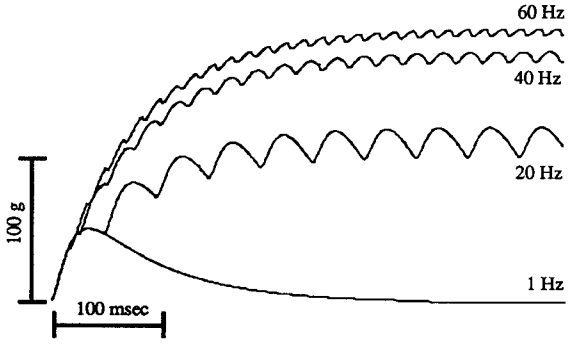

Fig. 9. Force in the active state muscle model of Fig. 8 at resting for a series unit impulses at various frequencies

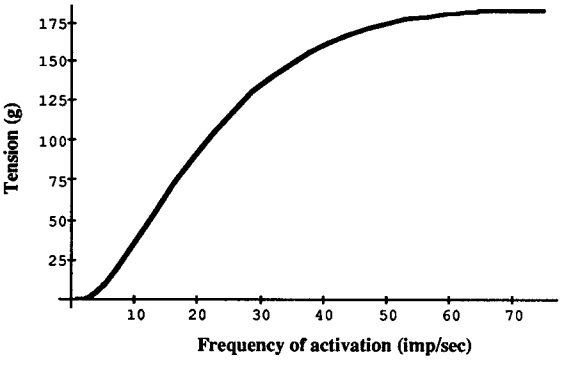

Fig. 10. Steady state tension output of the muscle model at $\lambda = \lambda^*$, i.e., length beyond which passive force develops

[5] In measuring the tension developed in a muscle, it is often the case that experimentalists report their results in terms of the amount of weight the muscle could hold, e.g., 5 g. The force developed by this muscle is $0.005\ \text{g} \times 9.8\ \text{m/s}^2 = 0.049$ N

[6] In a later modeling effort (Inbar and Ginat 1983), this negative region was also ignored

Differentiating above with respect to λ gives us muscle stiffness as a function of length and activation rate:

$$\frac{d\phi(\lambda, \Delta t)}{d\lambda} = b\frac{dS(\lambda)}{d\lambda} g(\Delta t) + K$$
$$= \frac{dS(\lambda)}{d\lambda}\frac{\phi(\lambda, \Delta t) - K(\lambda - \lambda^*)}{S(\lambda)} + K$$

where

$$K = \frac{K_{PE}}{1 + K_{PE}/K_{SE}}$$

At a given muscle length $\lambda = \lambda_0$ (i.e., isometric conditions), because $S(\lambda)$ and its derivative with respect to length are constant, stiffness varies linearly with force. This agrees with the experimental data of Fig. 6 regarding stiffness-force relationship of an areflexive muscle as measured at isometric conditions.

In order to understand what is gained when this areflexive muscle in endowed with a reflexive control system, we next consider control of a single joint with a pair of areflexive muscles. The results are then compared to the case where the single joint is being controlled by a pair of reflexive muscles.

5 Actuator redundancy and stability of a single joint system

Let us consider the problem of how to assign muscle activation rates so that the single joint system in Fig. 11 moves from a (previously) stable equilibrium at one joint angle, to a stable equilibrium at another position. If the limb in Fig. 11 is an inverted pendulum, then its dynamics are described by the following:

$$\tau = mc^2\frac{d^2\theta}{dt^2} + v\frac{d\theta}{dt} + mcg\cos(\theta) \tag{17}$$

where τ is the joint torque, m is the point mass at the end of the limb, c is the length of the limb, θ is the joint angle, v is the joint's viscous parameter, and g is the gravitational constant. Parameter values used in simulation are: $m = 0.1$ g, $c = 1$ cm, $v = 0.01$ g.cm^2/s. In order to relate joint torque to muscle forces, we first need to describe muscle-link kinematics. In Fig. 11, muscle lengths are related to the joint angle by:

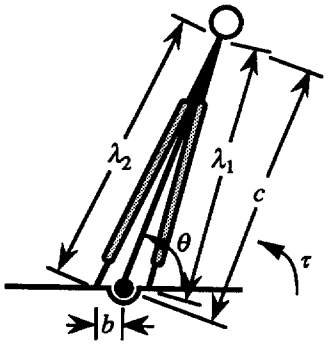

Fig. 11. A ball-and-socket joint with two muscle-like actuators

$$\lambda_1 = \sqrt{b^2 + c^2 - 2bc\cos(\theta)} \tag{18}$$

$$\lambda_2 = \sqrt{b^2 + c^2 + 2bc\cos(\theta)} \tag{19}$$

where $b = 0.2$ cm. Differentiating each muscle length with respect to the joint angle defines the muscle Jacobian $\mathbf{J}_M = [d\lambda_1/d\theta, d\lambda_2/d\theta]^T$, whose elements are the moment arm of each muscle as a function of joint configuration:

$$\frac{d\lambda_1}{d\theta} = \frac{bc\sin(\theta)}{\lambda_1} \tag{20}$$

$$\frac{d\lambda_2}{d\theta} = -\frac{bc\sin(\theta)}{\lambda_2} \tag{21}$$

This kind of muscle geometry captures the salient quality of muscles such as biceps where the moment arm is maximum near the middle of the joint-span: For example, in Fig. 3B we see that the moment arm of the biceps has its maximum value near 90 deg, which is the case with the muscles in Fig. 11 as well.

From the principle of virtual work, muscle forces counter-act each other to produce a joint torque:

$$\tau = -\mathbf{J}_m^T\phi = -\frac{d\lambda_1}{d\theta}\phi_1 - \frac{d\lambda_2}{d\theta}\phi_2 \tag{22}$$

Note that for a given joint torque τ, muscle forces ϕ_1 and ϕ_2 cannot be calculated because there is an infinite set of antagonistic muscle forces that can lead to the generation of the same joint torque (Shadmehr 1991a). A simple example of this is evident in postural control: one can hold a limb at the same position while changing the amount of co-contraction in the antagonistic muscles – increasing muscle activation leads to an increase in muscle force, but the same effective torque is produced at the joint because the limb does not move. This is one example of actuator redundancy.

It is commonly believed that co-contraction is a technique for modulating joint stiffness. Joint stiffness, $\mathbf{K}_J$, is in general a matrix which is the derivative of torque with respect to joint angle:

$$\mathbf{K}_J = \frac{d\tau}{d\theta} = -\left(\frac{d\mathbf{J}_M}{d\theta}\right)^T\phi - \mathbf{J}_M^T\frac{d\phi}{d\theta} \tag{23}$$

which can be written in terms of muscle force and stiffness as:

$$\mathbf{K}_J = -\left(\frac{d\mathbf{J}_M}{d\theta}\right)^T\phi - \mathbf{J}_M^T\frac{d\phi}{d\lambda}\mathbf{J}_M \tag{24}$$

In the case of our single joint system, muscle stiffness is a scalar:

$$\mathbf{K}_J = -\frac{d^2\lambda_1}{d\theta^2}\phi_1 - \frac{d^2\lambda_2}{d\theta^2}\phi_2 - \left(\frac{d\lambda_1}{d\theta}\right)^2\frac{d\phi_1}{d\lambda_1}$$
$$-\left(\frac{d\lambda_2}{d\theta}\right)^2\frac{d\phi_2}{d\lambda_2} \tag{25}$$

So we see that in this system, muscle forces counter-act to produce a torque at the joint, while their stiffnesses add to produce a joint stiffness. By specifying joint stiffness, one is describing, for example, how the limb

should react in case any part of it comes in contact with the environment. A certain amount of stiffness will also be required to ensure stability of the limb. For the system of Fig. 11, we can see that K_J should always be negative: if a disturbance displaces the limb, the resulting change in torque should be in the opposite direction. Setting a value on K_J depends on the stability requirements of the system: From (17) it is clear that for an inverted pendulum, given torque τ, the equilibrium of the system is at θ_E, where

$$\theta_E = \cos^{-1}\left(\frac{\tau}{mcg}\right) \tag{26}$$

Through non-linear stability analysis (Appendix 2, see also Shadmehr 1991b), one can show that in order to guarantee stability at this equilibrium, the stiffness must be:

$$K_J < -mcg \sin(\theta_E) \tag{27}$$

i.e., the stiffness of the joint must be more negative than the rate of change in the gravitational torque experienced by the limb at this equilibrium position. However, in order to produce such a stiffness, there might be complications: A muscle which has a moment arm that is not a monotonically increasing function of joint angle, such as the biceps and muscles used in Fig. 11, will at certain configurations have a negative value for its second derivative of length with respect to joint angle. The leads to a paradox: Assuming that muscle force is always positive (i.e., muscles cannot push), we see that in (25) an increase in muscle force will tend to destabilize the limb by making joint stiffness more positive! In other words, co-contraction may actually be a destabilizing mechanism in this system. Therefore, just because two muscles are in antagonistic geometric configuration, one cannot assume that co-activation (increase in muscle force) will necessarily lead to an increase in joint stiffness. In fact, co-activation may actually destabilize the limb

Theorem 2. *For a ball and socket joint fitted with muscle-like actuators, situated in a horizontal plane, muscle stiffness must increase at least linearly as a function of muscle force in order to generate a negative joint stiffness (the requirement for stability).* The proof is as follows: In (22), at equilibrium $\tau = 0$. Solve for ϕ_2, then substitute $d\lambda_1/d\theta$ and $d\lambda_2/d\theta$ from (20) and (21). This gives us:

$$\phi_2 = \phi_1 \frac{\lambda_2}{\lambda_1}$$

Therefore $d\phi_2/d\lambda_2 = \phi_1/\lambda_1$. These give us force and stiffness in muscle number 2 in terms of muscle number 1. Substitute these in (25), and after some algebra, it can be shown that:

$$K_J = \frac{b^2c^2 \sin^2\theta}{\lambda_1^3}\left(\phi_1 - \lambda_1 \frac{d\phi_1}{d\lambda_1}\right)$$

A necessary condition for stability of the system is for K_J to be negative, which can be realized if and only if:

$$\frac{d\phi_1}{d\lambda_1} > \frac{\phi_1}{\lambda_1}$$

This shows that at isometric conditions, muscle stiffness must grow at least linearly with force in order to guarantee stability (therefore, a muscle cannot obey Hooke's law). In light of this result, let us reconsider the data of Hoffer and Andreassen (1981) as plotted in Fig. 6. In the isometric areflexive muscle, stiffness grows linearly as a function of force until it reaches a maximum value. Approximately the same maximum stiffness is also reached by the reflexive muscle, but here the stiffness grows faster than linearly. The faster the rate of increase in stiffness of the muscles as a function of force, the more likely that co-contraction will lead to a further stiffening of the joint. For the problem of maintaining an equilibrium position and modulating stiffness, it would also be advantageous to be able to produce a larger muscle stiffness at lower muscle forces. The issue is taken up in the next section where both muscle models (i.e., Eq. 11 and Eq. 16) are used to control an inverted pendulum.

6 Programming an equilibrium position

Our concern here is to maintain posture with the muscle-skeleton system of Fig. 11, given that its dynamics are specified in (17), i.e., to assign muscle forces so the limb stays at a desired position θ_d. The procedure is to find the set of muscle forces which position the equilibrium of the system at θ_d, and to ensure that this position is stable. The initial step is to set $\theta_E = \theta_d$ and determine the amount of torque that the muscles must collectively produce: this can be calculated by solving for τ in (26): $\tau = mcg \cos(\theta_E)$. The next step is to assign a desired joint stiffness compatible with the constraint in (27). We can then solve for muscle forces in (22) and (25): recall that the stiffness terms can be written as a linear function of muscle force in the case of the areflexive muscles. We have plotted the muscle forces as a function of joint equilibrium position in Fig. 12 for the case where the desired joint stiffness was ten times the minimum requirement of (27) (qualitatively,

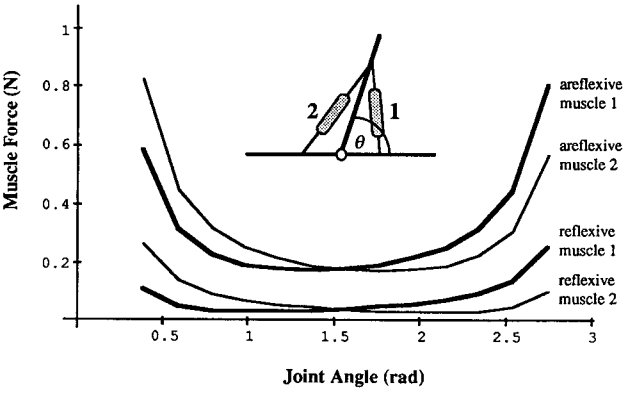

Fig. 12. A mapping from joint equilibrium to muscle forces at ten times minimum joint stiffness compatible with stability. The forces for the reflexive and areflexive muscles are shown. The same posture can be maintained with significantly smaller muscle force when the reflexes are available

the results do not change as this stiffness multiple is changed).

We formulated the reflexive muscle model in terms of the parameters of the areflexive muscle as follows: Hoffer and Andreassen (1981) showed that when the muscle was isolated by cutting the soleus nerve, maximum stiffness of the reflexive muscle was about 1.2 times the maximum stiffness of the areflexive muscle. Therefore in (11), $k = 12 \times \max\{d\phi/d\lambda\}$, where ϕ is the force produced by the areflexive and max $\{d\phi/d\lambda\}$ is the maximum stiffness of the areflexive muscle. For the areflexive muscle model introduced in the previous section, maximum stiffness is 1430 N/m. Therefore if the same muscle was under reflex control, we would expect maximum stiffness (or k) to be about 1716 N/m. This relatively small increase in the stiffness of the areflexive muscle is similar to the 25% increase that was found by Inbar et al. (1970) when comparing the stiffness of a passive areflexive muscle to a passive reflexive preparation.

The term α in (11) describes how rapidly stiffness reaches its maximum level as muscle force increases. A result of Hoffer and Andreassen (1978, 1981) was that the reflexive muscle achieves 65% of its maximum stiffness at 25% of its maximum force. Since the maximum force produced by a reflexive muscle is about the same as that of an areflexive muscle, α is approximately $4/\phi_{max}$, where ϕ_{max} is the maximum force possible in the areflexive muscle (in our case, this is 180 g or 1.77 N).

Unlike the situation for the areflexive muscles, it is not possible to find an analytical solution to (22) and (25) in terms of force for a pair of reflexive muscles. This is because for reflexive muscles, stiffness is a non-linear function of force, as in (11). We used a numerical technique to solve the simultaneous non-linear Eq. in (22) and (25). The results are plotted in Fig. 12. The joint torque and stiffness at each equilibrium position is the same as in the areflexive case, yet the forces at each muscle are significantly smaller. Since larger muscle force requires an exponentially higher level of muscle activation (as in Fig. 10) and consequently exponentially higher amount of metabolic energy, the energy cost of posture with reflexive muscles is much less than the energy cost of posture with areflexive muscles. This implies that control of muscles through modulation of the input to their spinal reflexes (i.e., via control of the spring's resting length) results in very significant improvements in the efficiency of the system: the same posture can be maintained at a far smaller metabolic cost. This is a result of the fact that a reflexive muscle is near its maximal stiffness while producing a fraction of its maximal force (the non-linear relationship in 10), as compared with an areflexive muscle where stiffness grows linearly with force.

7 Conclusions

We have considered how spinal reflexes affect the static length-tension characteristics of a muscle. Based on the experimental data on the response of the human elbow muscles, Feldman (1966) concluded that the static characteristics of the muscle-reflex system resembles a non-linear spring with an adjustable resting length. In contrast, many other researchers tend to model static characteristics of muscles as a non-linear spring with an adjustable stiffness. To understand the relative merits of each approach, we showed that the variable stiffness model would predict that at a given length, muscle stiffness would increase linearly as muscle force increased. This prediction does not agree with the data on the intact muscle-reflex system (Hoffer and Andreassen 1979, 1981), but it does resemble the behavior of that muscle when its reflex system has been removed (Fig. 6).

From the stiffness-force relationship in the intact muscle preparation, we derived the necessary length-tension characteristics of a reflexive muscle. The key point is that stiffness-force relationship in a reflexive muscle is a non-linear function that does not vary as a function of muscle operating length. Based on this, it was shown that a reflexive muscle does indeed appear as a non-linear spring with an adjustable resting length. This supports the spirit of the Feldman hypothesis, albeit not his formulation of the static force-length characteristics of a reflexive msucle. We showed that our formulation (in 11) fits the original Feldman and Orlovsky (1972) data better than the model proposed by Feldman (1966). So it would appear that there is independent experimental evidence supporting the hypothesis that the static behavior of a muscle-reflex system appears as a non-linear spring with an adjustable threshold length.

In order to show the functional difference between these two classes of models (i.e., non-linear springs with variable stiffness vs. variable resting lengths), we considered control of an inverted pendulum. To this end, we initially derived an active-state model of an areflexive frog muscle and then formulated a reflexive muscle based on the parameters of this model. Given our assumptions regarding the components of the areflexive muscle, it was proven that the stiffness-force behavior at isometric conditions will be linear (and this agrees with measurements of Hoffer and Andreassen 1981).

This model for the areflexive muscles was used to control an inverted pendulum where we introduced the problem of actuator redundancy: It was shown that unique muscle forces cannot be assigned to maintain the limb at a desired position since many degrees of co-activation can lead to generation of the same joint torque. To deal with this issue, it was suggested that description of posture must include not only the position of the limb, but also its stiffness. Lower bounds on the stiffness of the joint can be defined when one considers the stability requirements of the system at equilibrium. Using this information on stiffness of the system, the minimum amount of muscle force required for the pendulum to be maintained at equilibrium can be determined.

Stiffness of the joint can be varied when antagonist muscles co-contract. With the example of control of an

inverted pendulum we were able to present a paradox which challenges the notion that any pair of antagonist spring-like elements attached to a single joint system can produce a stable equilibrium position: In fact, it was shown that if the moment arm of one of the muscles is not a monotonically increasing function of joint angle (e.g., muscles such as biceps and triceps), then force produced by a muscle can act to make the joint's stiffness more positive (a destabilizing effect). Therefore, just because two muscles are in antagonistic geometric configuration, one cannot assume that co-contraction (increase in muscle force) will necessarily lead to an increased (i.e., more negative) joint stiffness.

Co-contraction will lead to an increase in joint stiffness if the stiffness of each muscle increases linearly with muscle force at isometric conditions. This condition may or may not be met in the areflexive muscle, but is more likely to be met in the reflexive case where stiffness grows faster than linearly with force (cf. Fig. 6). This means that co-contraction will stiffen the limb, rather than destabilize it.

Another advantage of the reflexive system on control of a single joint was illustrated when it was shown that posture may be maintained throughout the work space at a significantly reduced energy cost to the muscles if the muscles behaved as non-linear springs with an adjustable resting length (the model of a reflexive muscle). The reflexes enhance the stability of the system because stiffness of a reflexive muscle approaches its maximum value at a fraction of its maximum force, so that only a small amount of force is required of each muscle to maintain posture of an inherently unstable limb.

Appendix 1: solution to the areflexive muscle's differential equation

The force produced by a muscle is related to its mechanical parameters by the following relation:

$$\dot{\phi} = \frac{K_{SE}}{B}\left(K_{PE}\,\Delta\lambda + B\dot{\lambda} - \left(1 + \frac{K_{PE}}{K_{SE}}\right)\phi + P(t)\right)$$

where ϕ is the muscle force, K_{SE} is the series elastic component, K_{PE} is the parallel elastic component, B is the viscous component, λ is muscle length, $\Delta\lambda$ is the change in muscle length beyond its resting length, and $P(t)$ is the force pumped into the system by the active contractile component. This active force depends on the history of muscle stimulation, where the response to a series of stimuli of period Δt is modeled by the following:

$$P(t) = \sum_{n=0}^{\infty} h(t - n\Delta t)(u(t - n\Delta t) - u(t - (n+1)\Delta t))$$

where $h(t) = k(\exp(-\alpha t) - \exp(-\beta t))$. For the period $0 < t < \Delta t$, i.e. the period after the first stimulus, we have:

$$\dot{\phi}_1 = a_1(a_3 - a_2\phi + k\exp(-\alpha t) - k\exp(-\beta t))$$

where $a_1 = K_{SE}B$, $a_2 = 1 + K_{PE}/K_{SE}$, and $a_3 = K_{PE}\Delta\lambda + b\dot{\lambda}$. The general solution to this equation is:

$$\phi_1 = \frac{a_3}{a_2} + \frac{ka_1\exp(-\beta t)}{\beta - a_1a_2} - \frac{ka_1\exp(-\alpha t)}{\alpha - a_1a_2} + c_1\exp(-a_1a_2t)$$

where c_1 depends on the initial condition. Assuming the initial condition is $\phi(0) = 0$, the solution to this differential equation is:

$$\phi_1 = a_1k\left(\frac{\exp(-\beta t)}{\beta - a_1a_2} - \frac{\exp(-\alpha t)}{\alpha - a_1a_2} + \left(\frac{1}{\alpha - a_1a_2} - \frac{1}{\beta - a_1a_2}\right)\exp(-a_1a_2t)\right) + \frac{a_3}{a_2}(1 - \exp(-a_1a_2t))$$

After a period of Δt, the second impulse arrives. For the period $\Delta t < t < 2\Delta t$, set $t' = t - \Delta t$. It can be shown that the force in the muscle during this period is related to ϕ_1 by the following:

$$\phi_2(t') = \phi_1(t') + \phi_1(\Delta t)\exp(-a_1a_2t')$$

In general, for the period $(n-1)\Delta t < t < n\Delta t$, i.e., after n stimuli, we have:

$$\phi_n(t) = \phi_1(t - (n-1)\Delta t) + \phi_1(\Delta t) \times \sum_{m=0}^{n}\exp(-a_1a_2(t - (n-1)\Delta t + m\Delta t))$$

At time $t = n\Delta t$, i.e., just before the arrive of the $n+1$st stimuli, we have:

$$\phi_n = \phi_1(\Delta t)\sum_{q=0}^{n-1}\exp(-a_1a_2q\Delta t) = \phi_1(\Delta t)\frac{\exp(-a_1a_2n\Delta t) - 1}{\exp(-a_1a_2\Delta t) - 1}$$

Equation (16) represents the above force in the muscle.

Appendix 2: stability of an inverted pendulum with joint stiffness

The state variable equations for the system of Fig. 11 are:

$$F(\theta, \omega) = \begin{cases}\dot{\theta} = \omega \\ \dot{\omega} = \dfrac{\tau}{mc^2} - \dfrac{v}{mc^2}\omega - \dfrac{g}{c}\cos(\theta)\end{cases}$$

This system is in equilibrium when $\dot{\theta} = \dot{\omega} = 0$, which occurs at $\omega_e = 0$ and $\theta_e = \cos^{-1}(\tau/mgc)$. We will show that this equilibrium is stable if $d\tau/d\theta > -mcg\sin(\theta_e)$, where $d\tau/d\theta$ is joint stiffness. The proof is as follows:

$$\frac{d}{dt}\begin{pmatrix}\theta \\ \omega\end{pmatrix} = F(\theta, \omega) = F(\theta_e, \omega_e) + \left.\frac{dF}{d(\theta, \omega)}\right|_{\theta=\theta_e, \omega=\omega_e}\begin{pmatrix}\theta - \theta_e \\ \omega - \omega_e\end{pmatrix} + \cdots$$

$$= \begin{bmatrix} 0 & 1 \\ \dfrac{d\tau}{d\theta}\dfrac{1}{mc^2} + \dfrac{g}{c}\sin(\theta_e) & \dfrac{-v}{mc^2}\end{bmatrix}\begin{pmatrix}\theta - \theta_e \\ \omega\end{pmatrix} + \cdots$$

For the system to be stable, both eigenvalues of the matrix $dF/d(\theta, \omega)$, as evaluated at $\theta = \theta_e$ and $\omega = \omega_e$, must have negative real parts. These eigenvalues are:

$$\lambda_{1,2} = \frac{1}{2}\left(-\frac{v}{mc^2} \pm \left(\frac{v^2}{m^2c^4} + 4\left(\frac{K_J}{mc^2} + \frac{g}{c}\sin(\theta_e) \right) \right)^{1/2} \right)$$

where $K_J = d\tau/d\theta$. Suppose the term under the square root is positive, causing the eigenvalues to be real. It follows that if the term under the square root is larger than v/mc^2, then one of the eigenvalues will be a positive number, causing instability. Therefore we will have instability if $K_J/mc^2 + g\sin(\theta_e)/c > 0$, suggesting that one condition for stability is $K_J < -mcg\sin(\theta_e)$. Now suppose that the term under the square root is negative, causing the eigenvalues to be complex (damped oscillations). This would mean that $K_J < -1/4v^2/mc^2 - mcg\sin(\theta_e)$, which is a weaker condition than the previous constraint on K_J. Therefore the system is at stable equilibrium if $K_J < -mcg\sin(\theta_e)$.

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