Implications of Positive Feedback in the Control of Movement

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Prochazka, Arthur, Deborah Gillard, and David J. Bennett. Implications of positive feedback in the control of movement. *J. Neurophysiol.* 77: 3237–3251, 1997. In this paper we review some theoretical aspects of positive feedback in the control of movement. The focus is mainly on new theories regarding the reflexive role of sensory signals from mammalian tendon organ afferents. In static postures these afferents generally mediate negative force feedback. But in locomotion there is evidence of a switch to positive force feedback action. Positive feedback is often associated with instability and oscillation, neither of which occur in normal locomotion. We address this paradox with the use of analytic models of the neuromuscular control system. It is shown that positive force feedback contributes to load compensation and is surprisingly stable because the length-tension properties of mammalian muscle provide automatic gain control. This mechanism can stabilize control even when positive feedback is very strong. The models also show how positive force feedback is stabilized by concomitant negative displacement feedback and, unexpectedly, by delays in the positive feedback pathway. Other examples of positive feedback in animal motor control systems are discussed, including the β-fusimotor system, which mediates positive feedback of displacement. In general it is seen that positive feedback reduces the sensitivity of the controlled extremities to perturbations of posture and load. We conclude that positive force feedback can provide stable and effective load compensation that complements the action of negative displacement and velocity feedback.

INTRODUCTION

In the last few years there has been an accumulation of evidence for positive feedback in the control of motor tasks in animals. Positive feedback control of limb position or muscle length in different species has been discussed for many years (invertebrates: Bässler 1993; Burrows and Pflueger 1988; Cruse et al. 1995; mammals: Grill and Rymer 1985; Houk 1972, 1979; Kouchtir et al. 1995). More recently, interest has shifted to the possibility of positive feedback control of muscle or limb force (invertebrates: Bässler 1993; Bässler and Nothof 1994; Cruse 1985; Cruse et al. 1995; cats: Brownstone et al. 1994; Conway et al. 1987; Gossard et al. 1994; Guertin et al. 1994, 1995; Pratt 1995; humans: Dietz et al. 1992). In addition, positive feedback loops have been posited within sensorimotor control areas of the mammalian brain (Houk et al. 1993).

The theories and speculation regarding positive feedback mediated by sensory afferent input to the nervous system have not satisfactorily addressed the issue of stability. From a control systems point of view, this is the most immediate concern regarding the functional viability of any mechanism incorporating positive feedback. In engineering systems, positive feedback is generally only used intentionally if instability is desired, for example in circuits designed to switch from one extreme operating point to another, such as monostable or bistable multivibrators (flip-flops), or circuits that oscillate continuously, such as astable multivibrators or oscillators. Otherwise, positive feedback is avoided in the design process, because instability is undesirable and in many cases potentially destructive of the system under control.

Within the CNS, the oscillators generating the limit cycles of locomotion and respiration have been tacitly or explicitly assumed to be based on positive feedback mechanisms, whether in the form of neural networks or single neurons with pacemaker properties (Grillner et al. 1995; Roberts et al. 1995). However, the evidence for positive feedback connections in reflex systems controlling posture and movement has been something of a puzzle, precisely because of the potential for instability. On the one hand, it has been suggested that certain oscillatory behaviors such as rocking in stick insects (Bässler 1983) or paw shaking in cats (Prochazka et al. 1989) might reflect a ''deliberate'' augmentation of loop gain and therefore destabilization of stretch reflexes. Similarly, certain pathological tremors have been attributed to instability in the stretch reflex arc (Dimitrijevic et al. 1978; Jacks et al. 1988; Rack and Ross 1986) or in olivocerebellar and thalamocortical pathways (Lamarre 1984). In all these cases the neural loops are assumed to function as negative feedback systems under normal conditions, but they develop unstable positive feedback behavior because of a 180° net phase lag combined with an open-loop gain exceeding unity at a particular frequency. On the other hand, it has been argued that some reflex connections are ''designed'' to operate as positive feedback systems to provide nonoscillatory graded control of posture and movement (Cruse et al. 1995). It is here that the issue becomes problematic, for linear control theory predicts that to be efficacious in load compensation, the open-loop gain of positive force feedback (G_f) should be close to unity; but the closer it is to unity, the greater the risk of instability (Phillips and Harbor 1991).

Houk (1972) discussed the least disputable example of positive feedback in mammalian reflexes, the β-loop. This comprises β-skeletofusimotor neurons that exert fusimotor action on muscle spindle afferents, which reflexly excite the same β-skeletofusimotor neurons. Houk (1972) argued that even if the open-loop gain of the β-loop (G_β) exceeded 1, the system remained stable because the β-loop was nested inside a negative feedback loop controlling muscle displacement. In this view, activation of β-motoneurons would cause the spindle-bearing extrafusal muscle to shorten, counteracting β-fusimotor excitation of spindle afferents and thereby holding in check the reflex action of these afferents on the β-motoneurons. This argument was based on a simple static feedback model that ignored muscle and receptor dynamics and nonlinearities. Some years later, Grill and Rymer (1985) inferred G_β from differences in spindle afferent stretch sensitivity before and after dorsal

rhizotomy in decerebrate cats. The estimates of G_β were in the range of 0.2–0.8.

At the time, the reflex connections of force-sensing group Ib tendon organ afferents were assumed to produce negative feedback action. Accordingly, Houk (1972) showed that adding negative force feedback to negative displacement feedback produced springlike behavior of muscle in the face of externally applied loads. The greater the force feedback gain, the more compliant the muscle would be to loading. Whether force feedback gain was actually modulated in normal animal behavior remained an open question (Crago et al. 1976; Hoffer and Andreassen 1981; Rymer and Hasan 1980).

In the last five years numerous reports of positive feedback connections from force-sensing receptors to homonymous or heteronymous motoneurons have appeared. In spinal and decerebrate cats, heteronymous reflexes mediated by tendon organ Ib afferents switch from being inhibitory in static postures (Eccles et al. 1957; Harrison et al. 1983) to excitatory during locomotor behavior (Conway et al. 1987; Gossard et al. 1994; Guertin et al. 1995; Pearson and Collins 1993). Because Ia and Ib reflexes have the same sign under these circumstances, there has been a shift away from describing reflex action in terms of sensory modalities such as force and displacement, toward lumped descriptions such as ''group I excitatory action,'' whereby afferents are classified according to conduction velocity rather than modality (Angel et al. 1995; Jankowska and McCrea 1983). Yet the functional consequences of separate modalities cannot be avoided. If signals from force-sensing afferents reflexly increase the force the receptors sense, this constitutes positive force feedback, and, for the reasons discussed above, the issue of stability must then be dealt with.

In the preceding paper (Prochazka et al. 1997) we described experiments in human subjects whose muscles were controlled with electrical stimulation under positive force feedback control. These experiments showed that positive force feedback resulted in stable load compensation under a surprisingly wide range of feedback gains. The closed-loop behavior was characterized by reductions or even reversals in the yield that inertial loading would otherwise have caused. The reversals occurred when G_f had been set greater than unity. The very fact that the loop remained stable for $G_f >$ 1 was a surprise, and further investigation suggested that the muscle shortening associated with negative yield automatically reduced G_f to unity. Delays in the feedback pathway similar to those seen in cat locomotion stabilized positive force feedback. This was also unexpected.

The main advantage of the empiric approach whereby real muscles are incorporated in an artificial feedback loop is that the uncertainties of modeling nonlinear muscle properties are avoided. The disadvantage is that it is difficult to analyze closed-loop behavior in a general manner. However, by combining the two approaches, the underlying mechanisms can be understood and verified.

METHODS

Mathematical modeling

We recognized from the outset that to be general, conclusions regarding closed-loop behavior should not depend too heavily on the particular structure of a model or on the choice of parameters

within a given model. We therefore studied numerous models of mammalian neuromuscular systems, ranging from simple linear control loops to models that incorporated nonlinear elements such as conduction delays and nonlinear force-velocity and length-tension relationships. The modeling described in this paper was performed with the use of Matlab 4.2c and its associated graphics-based Simulink 1.3c control systems software. The ''raw'' Simulink versions of the models are detailed in the APPENDIX and may be replicated within minutes, allowing interested readers to verify our simulations and to explore and test the behavior of our reflex model. Stability was separately analyzed with numerous combinations of parameters with the use of Bode, Nyquist, and root locus plots, some of which are presented in the APPENDIX.

The unexpected features of positive feedback observed in the empiric studies, namely affirming reactions, delay-dependent stabilization, and automatic gain control, were also reflected in the models. These basic behaviors were robust in that they were seen over a very wide range of parameters.

Model 1: simple reflex model

Figure 1 shows a highly simplified model of a reflex system. In this model, key elements of real neuromuscular systems such as tendon compliance, dynamic transfer functions of sensor, and the length and velocity dependence of muscle force production are omitted or simplified. These elements are represented more rigorously in the nonlinear model below. Yet, despite the basic structural differences between the two models, they exhibited essentially the same closed-loop behaviors. Other variants of these models not detailed in this paper also showed the same basic behaviors. The generality of the main features of closed-loop behavior in the face of significant parametric and structural variation is our strongest argument that our main conclusions are not model specific.

The load in both models is a mass of 1 kg, representative of the inertial load supported either by a cat hindlimb or the wrist joint in the experiments reported in the previous paper. We concentrated on modeling the cat hindlimb because the physiological evidence for positive force feedback originates from studies of cat locomotion. However, as it turns out, parameters such as net muscle stiffness and damping are fairly similar for the wrist supporting the forearm, as inferred from the kinetic and kinematic responses in the companion paper.

In the simple model, muscle force is modeled by a first-order active component and a viscoelastic parallel stiffness. The force-velocity relationship (Hill 1938), the muscle length-tension curve (Rack and Westbury 1969), and tendon compliance are all neglected. The external force is summed with tendon force, the resultant force acting on the inertial load. Force feedback is represented without dynamic components, but an adjustable reflex delay is included because this was shown in the empiric work to stabilize positive force feedback. Feedback from muscle spindles is likewise represented without dynamics and without delays.

Figure 6 shows the model as it appeared in the Simulink graphic interface. Many combinations of parameters were tested in simulations. Figure 2 shows a small set of simulations chosen for their relevance to the main issues of this paper. All simulations were performed with the use of the Runge-Kutta-3 simulation algorithm in the Simulink program (minimum and maximum step sizes 0.5 ms). Two variables are plotted: external force (perturbing input) and displacement (output).

From our empiric results we were interested in the following questions.

1) What was the maximal positive force feedback gain before instability occurred?

2) Did positive force feedback provide effective load compensation?

3) Did muscle yield reverse when G_f exceeded unity?
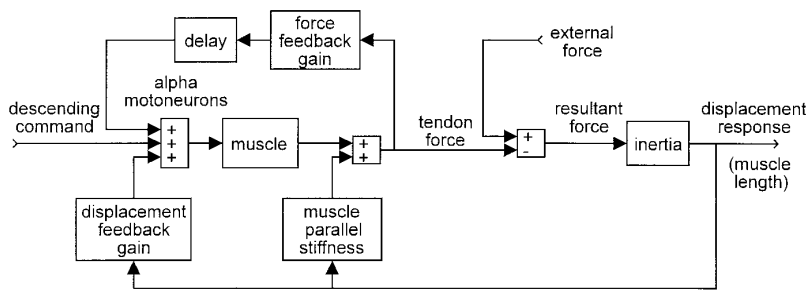
4) What was the effect of concomitant negative G_d?

5) Were delays in the force feedback pathway stabilizing or destabilizing?

The simulations in Fig. 2 illustrate the following results. Without displacement feedback or force feedback, the system, comprising the viscoelastic muscle parallel stiffness and the inertial load, showed a damped displacement response (Fig. 2*B*) to the external force (Fig. 2*A*). Adding positive force feedback at loop gains in the range of 0–1 produced stable load compensation (yield in Fig. 2*C* with $G_f = 0.5$ is about half that in Fig. 2*B* without feedback).
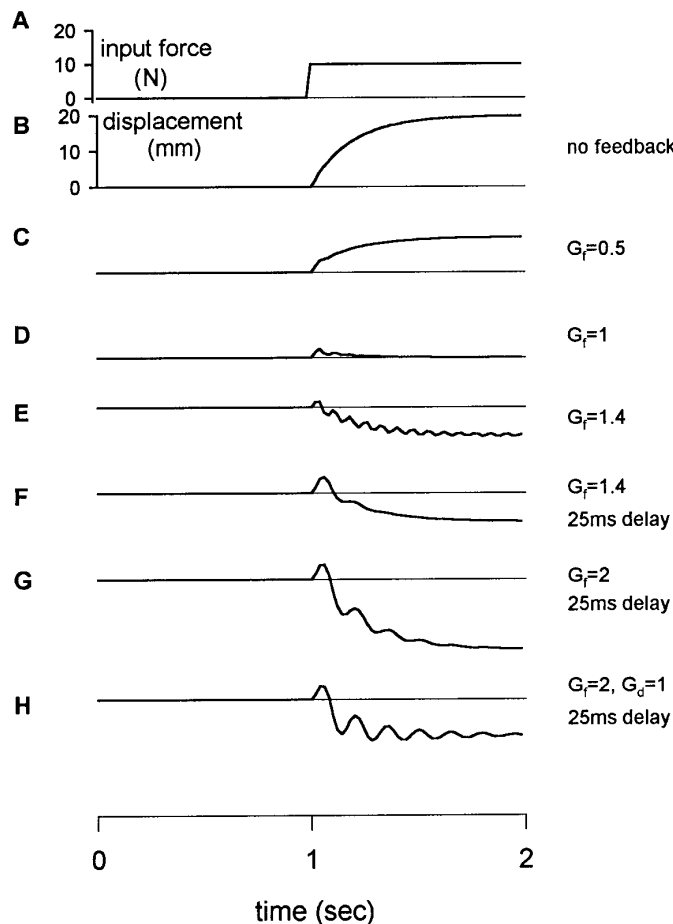

FIG. 2.   Simulations based on static reflex model of Figs. 1 and 6. *A*: input (external) force. *B*: displacement (yield) of load due to muscle stiffness; no feedback. *C*: reduced yield with addition of low-gain positive force feedback, open-loop gain of positive force feedback (G_f) = 0.5. *D*: zero net yield with unity-gain positive force feedback. *E*: underdamped overcompensation of load (negative yield or ''affirming reaction'') with $G_f = 1.4$. *F*: improved stability with 25-ms delay in positive force feedback pathway. *G*: delay allows higher force loop gain ($G_f = 2$), with larger affirming reactions. *H*: adding negative displacement feedback attenuates affirming reaction. Displacement calibrations in *B* also apply to *C–H*. G_d, displacement feedback gain.

In the simple model, G_f was determined by inspection as the multiple of the static gains around the force loop. Because displacement is held constant for this purpose, the only elements involved are the muscle contractile element and the force feedback element. In the nonlinear model, G_f could not be determined by inspection because of the nonlinear effects of tendon strain and muscle fiber length.

When $G_f = 1$ in Fig. 2*D,* apart from the small transients, the mass did not move, i.e., the system had infinite stiffness. The loop remained stable up to $G_f = 1.4$ (Fig. 2*E*), exhibiting an affirming reaction (i.e., instead of yielding, the mass now moved in the opposite direction of the imposed force). The reason that the system remained stable for values of G_f between 1 and 1.4 is that the parallel stiffness element provided negative displacement feedback and velocity feedback, which had a stabilizing effect (if the gain of this element was set to 0, the system went unstable at $G_f = 1$). Adding a 25-ms delay to the force feedback pathway had a stabilizing effect (Fig. 2*F*), allowing a higher loop gain to be attained with a larger affirming reaction (Fig. 2*G*). This stabilizing action of a delay in the positive feedback pathway was analyzed with the use of root locus techniques, as presented in Fig. 6 in the APPENDIX. Finally, the addition of explicit negative displacement feedback ($G_d = 1$) attenuated the affirming reactions. In this particular simulation displacement feedback without any velocity component to provide damping was slightly destabilizing. Note that the magnitude and sign of feedback gain are separately specified in this paper.

As expected from previous work (Houk 1972), negative force feedback in combination with negative displacement feedback resulted in a springlike response to external loading, the stiffness of which increased as G_d increased and G_f decreased (not illustrated).

Model 2: nonlinear reflex model

Figure 3 shows a more representative nonlinear model of the mammalian neuromuscular system. As in the simple model, the major functional elements are represented independently in each block (e.g., active and passive components of muscle force generation, tendon compliance, load, etc.). In natural movements of the limbs, agonist and antagonist muscles are often coactive, which tends to cancel out the rectifying nature of nonlinear stiffness and damping in individual muscles. In our first set of experiments, described in the previous paper, the muscles were coactivated to mimic this situation. However, the recent evidence for positive force feedback comes from cat locomotion, where extensor muscles are not coactive with flexor muscles during weight bearing. We therefore chose to model this situation with a single muscle supporting an inertial load.

Noteworthy features of the nonlinear reflex model are as follows.

1) A modified Hill (1938) model of muscle. This includes a passive parallel viscoelastic stiffness and a parallel contractile stiffness that depends on activation level, muscle length, and velocity, all based on relationships in the literature (Bawa et al. 1976; Rack and Westbury 1969; Winters 1990). The series compliance is equated to tendon compliance with the use of the parameters of Bennett et al. (1996). The muscle portion of the series compliance is neglected (Zajac 1989).
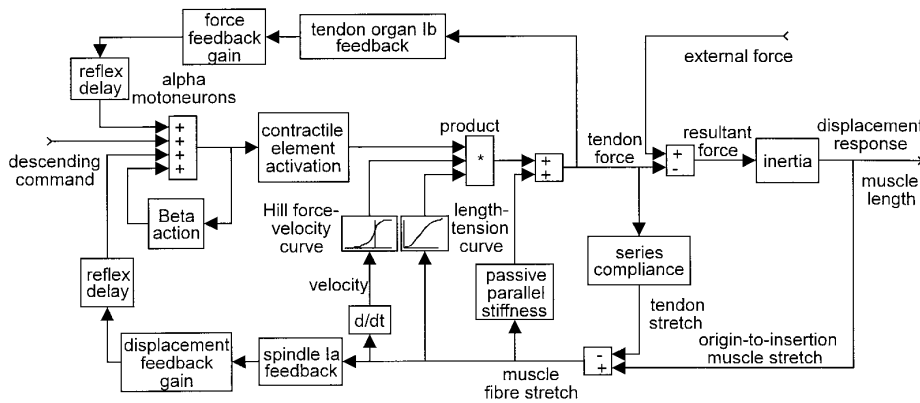
2) The length-tension and force-velocity relationships are represented by pairs of mathematical functions detailed in the APPENDIX. Variations in the shape parameters of these relationships were not critical in changing basic closed-loop behavior, as discussed in the APPENDIX.

3) Spindles are assumed to transduce muscle fiber displacement (i.e., length changes of the tendon are subtracted from muscle origin-to-insertion displacement).

4) Linear transfer functions of muscle spindle Ia afferents (Chen and Poppele 1978; Matthews and Stein 1969) and tendon organ Ib afferents (Anderson 1974; Houk and Simon 1967) are used. These have been found to give good fits of ensemble afferent responses in freely moving cats (Appenteng and Prochazka 1984; Gorassini et al. 1993). As alternatives, two variants of the Houk et al. (1981) nonlinear spindle Ia model were also tested (see APPENDIX).

5) Positive feedback via the β-fusimotor pathway. The activation of α- and β-motoneurons is assumed to be identical. The α-linked component of γ-fusimotor activity is lumped with the β-signal. Task-related fusimotor action (Prochazka et al. 1985) is represented as changes in Ia gain.

6) Separate delays in spindle Ia, tendon organ Ib, and β-fusimotor reflex paths. The Ia delay is assumed to be small (e.g., 10–20 ms). Current evidence from cat data indicates that positive Ib feedback has a delay or rise time of 30–40 ms (Gorassini et al. 1994; Gossard et al. 1994; Guertin et al. 1995). Consequently we tested Ib delays of up to 100 ms, or, alternatively, a single-pole, unity-gain, low-pass filter block with a turning point in the range of 10–100 rad/s.

7) Loading is provided as two step changes in force. The second load is added to explore the behavior of the loop after stabilization to the first load. In separate simulations not illustrated, step increments of force were applied through external springs.

RESULTS

The cost of the increased realism of nonlinear models such as that in Fig. 3 is an increase in the number of parameters. We experimented with many combinations of parameters and indeed numerous versions of the model itself. Our main conclusions regarding closed-loop behavior were robust across a large range of parametric and structural variations. The stability of the system was insensitive to large (2 orders of magnitude) variations in most of the parameters, as discussed in the APPENDIX. Figure 7 in the APPENDIX shows the version of the model that generated the representative simulations illustrated in Fig. 4. As in the simple model, simulations were performed with the use of the Runge-Kutta-3 simulation algorithm, although Euler and Linsim algorithms were also used. In Fig. 4 the initial con-

ditions at time $t = 0$ are such that muscle force and length are zero at the threshold of the length-tension curve (i.e., muscle just slack). The step in external force at $t = 0$ causes rapid muscle stretch and overshoot, because the stiffness and damping of the contractile element rise relatively slowly in the initial portion of the length-tension curve. The response to the second (test) load increment at $t = 1$ is more damped because the contractile element impedance is greater at the longer muscle length. Fig. 4, *left* and *right,* differ only in the time of onset of force feedback. In Fig. 4, *left,* force feedback is activated at the same time as the test loading. This mimics a linkage of positive force feedback with the onset of the stance phase of the cat step cycle, which is one of the possibilities to be inferred from the recent cat data. In Fig. 4, *right,* feedback is activated before loading (at $t = 0.5$).

The salient features of the simulations in Fig. 4 are as follows. Without displacement feedback or force feedback, the system, which comprises the viscoelastic muscle parallel stiffness, the in-series tendon compliance, and the inertial load, shows a damped displacement response to the test force. Adding positive force feedback at gains in the range of 0–1 produces stable and effective load compensation (stretch in Fig. 4*C, left,* with $G_f = 0.5$ is much less than in Fig. 4*B*). When $G_f = 1$ the mass moves very little, i.e., the system is very stiff.

At this point it is worth discussing the meaning of loop gain. As described in the previous paper, G_f may be determined by opening the force feedback loop, holding origin-to-insertion muscle length constant, and measuring the ratio of muscle force in response to a force input to the force sensor (see APPENDIX, Fig. 8). Tendon compliance is an intrinsic property of the force actuator and must therefore be included in the calculation of force loop gain. Internal shortening due to tendon compliance slightly reduces the gain that would otherwise be obtained. In the preceding paper it was shown empirically that force loop gain is length dependent. In the nonlinear model this results from the fact that the contractile element produces more force for a given change in activation as length increases. Strictly speaking, if we accept that force loop gain is length dependent, it should be viewed as a variable rather than a parameter. Indeed, the same argument can be applied to displacement loop gain.

In the ensuing discussion, G_f is understood to be the loop gain determined at some reference length and force. To differentiate this setpoint loop gain from the instantaneous loop
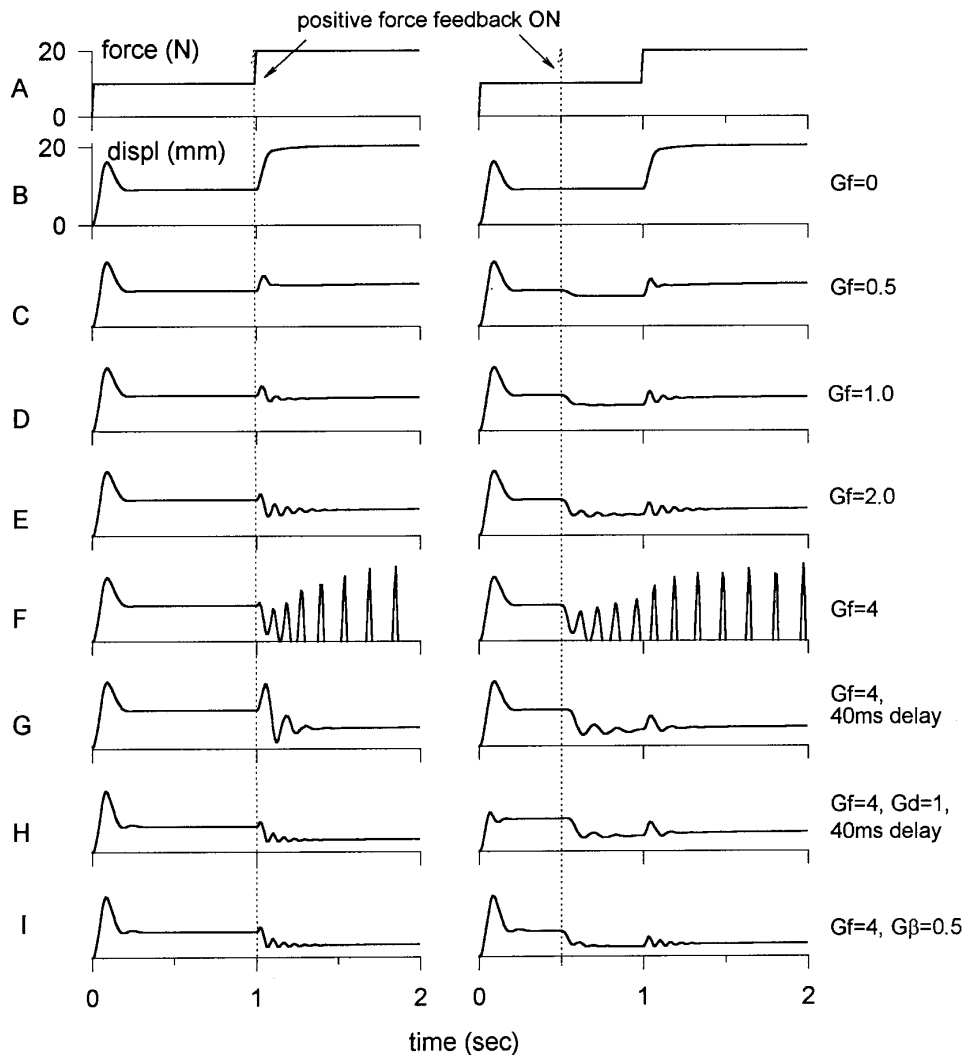
FIG. 4. Simulations based on nonlinear reflex model of Figs. 3 and 7. Positive force feedback is activated either at same time as test increment in external force (at $t = 1$ s, *left*) or earlier (at $t = 0.5$ s, *right*). *A*: external force. Two 10-N force increments are applied, the 1st at $t = 0$ to stretch muscle from slack to its operating length, the 2nd comprising test load. *B*: no feedback. Muscle displacement in response to force increments depends on inertial mass and muscle viscoelastic properties only. *C*: low-gain positive force feedback ($G_f = 0.5$) stiffens muscle, reducing displacement by test load. *D*: unity-gain positive force feedback. Displacement due to test load is very small when force feedback is activated at same time as loading (*left*), but some yielding is evident if force feedback is activated earlier (*right*) *E*: overcompensation (affirming reaction) to loading for $G_f = 2$. Underdamping suggests that system is close to instability. *F*: instability when G_f is raised to 4. *G*: stabilizing action of adding 40-ms delay in force feedback pathway. Similar effect is seen if a low-pass filter is used instead. *H*: adding negative displacement feedback to positive force feedback attenuates all of displacement responses, including affirming reaction. *I*: adding β-skeletofusimotor action stabilized unstable configuration of *F*. Displacement calibrations in *B* also apply to *C–I*. G_β, open-loop gain of β-loop.

gain, which varies with force and length, we will henceforth refer to it as G_{fs}. In the simulations of Fig. 4, the length and force reference points happen to be 7.3 mm and 10 N, respectively, in the midrange of the ascending portion of the length-tension curve for inputs to the contractile element of around unity (see Fig. 5). The system equilibrates to this length in Fig. 4*D, right,* after feedback is activated. The 10-N test load then stretches the muscle to a new equilibrium length (8.6 mm). The total force is now 20 N. With the use of the open-loop circuit of Fig. 8 in the APPENDIX, it can be shown that the gain is unity at this new length. In fact in all stable instances where $G_{fs} \geq 1$ the system equilibrates to the length and force at which loop gain is unity, regardless of the load. As is seen in Fig. 5, this automatic gain control is also evident in the length-tension trajectories for given values of G_{fs}. The loop remains stable up to $G_{fs} = 2$ (Fig. 4*E*). In Fig. 4*E, left,* where feedback is turned on at the onset of loading, the muscle shortens on loading (affirming reaction). In Fig. 4*E, right,* after feedback is activated, the system equilibrates to a length at which loop gain is unity, so the test load now causes a small length increase. For $G_{fs} = 4$, the system is unstable (Fig. 4*F*). Adding a 40-ms delay to the force feedback pathway stabilizes it (Fig. 4*G*). This stabilizing action of a delay in the positive feedback

pathway is confirmed with the use of root locus techniques in the APPENDIX. The addition of negative displacement feedback to the control loop attenuates and further stabilizes the load reactions (Fig. 4*H*, $G_d = 1$ starting at $t = 0$). Interestingly, delayed positive G_d via the β-pathway can stabilize positive force feedback in the same way as a delay in the force feedback pathway (Fig. 4*I*: $G_\beta = 0.5$, $G_{fs} = 4$, no delay in force feedback pathway, 20-ms delay in β-pathway).

The above closed-loop behaviors can be viewed from a different perspective by comparing open-loop (i.e., intrinsic) and closed-loop length-tension trajectories at different levels of contractile element activation and G_{fs}. Figure 5*A* shows a family of open-loop length-tension curves radiating from the origin. These were obtained from the nonlinear model by replacing the displacement feedback in Fig. 7 with a sinusoidal input function and applying increments of 0.5 to the input of the contractile element. In closed-loop trials similar to those of Fig. 4 but with the use of an 0.5-Hz sinusoidal loading signal, closed-loop length-tension trajectories were obtained, and portions of these are overlaid on the open-loop length-tension curves in Fig. 5. It should be stressed that the values of G_{fs} indicated in Fig. 5 are those evaluated for an operating point of 7.3 mm and 10 N. For $G_{fs} \geq 1$, all points in the closed-loop trajectories turned out
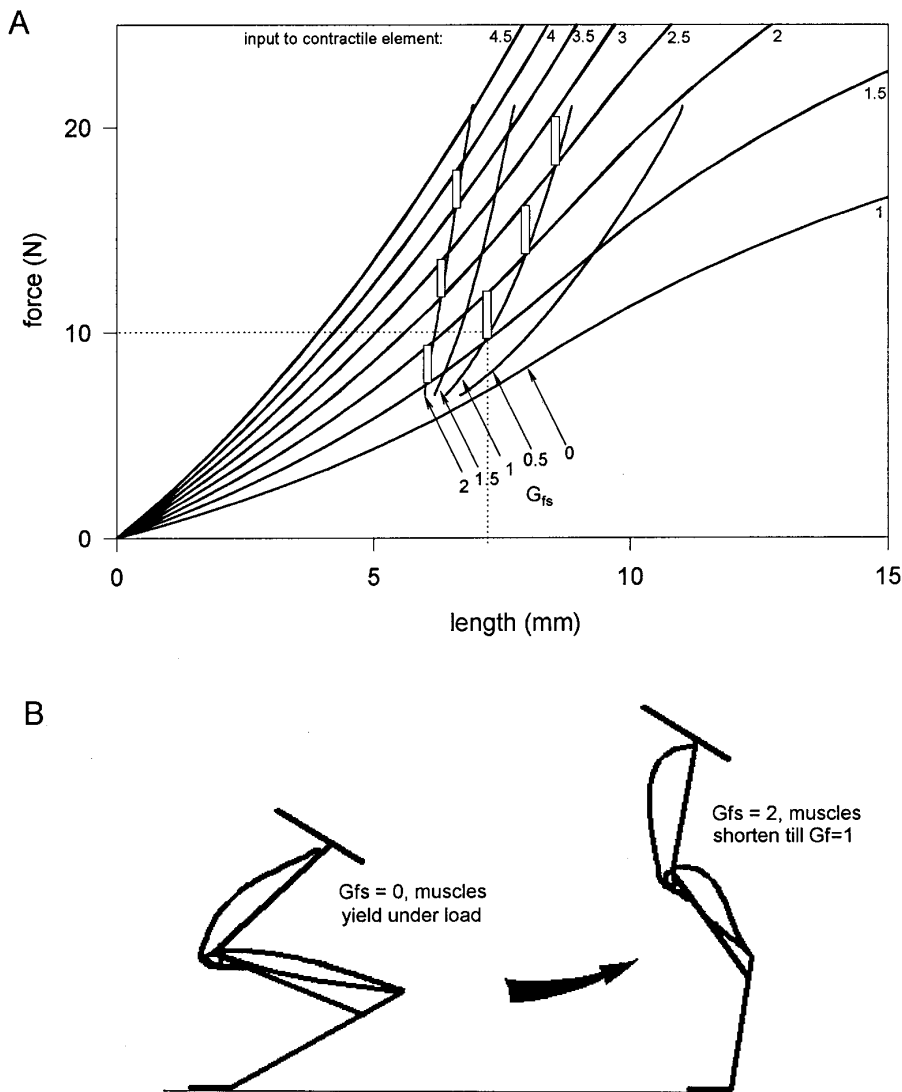
A

B

FIG. 5. *A*: length-tension curves due to intrinsic muscle properties alone, compared with those with positive force feedback. Family of open-loop length-tension curves radiating from origin obtained from nonlinear model, as described in text, is overlaid with portions of closed-loop length-tension curves where setpoint loop gain (G_{fs}) ranges from 0 to 2 (G_{fs} is force loop gain evaluated at an operating point of 7.3 mm and 10 N). Stiffness increases with increasing G_{fs}. Equilibrium length for a given force in Fig. 4 may be obtained from corresponding closed-loop length-tension curve in Fig. 5 (see text). Hollow bars show that along a closed-loop curve, a given increment in contractile element activation always produces same force increment, indicating that loop gain is held constant along each closed-loop curve. *B*: diagram illustrating predicted effect of activation of positive force feedback in extensor muscles of cat hindlimb. Extensor muscles shorten to a new equilibrium length, resulting in a more upright posture.

to correspond to a loop gain of unity. This was shown in separate analyses with the open-loop gain system of Fig. 8 in the APPENDIX. The constancy of gain in these trajectories is illustrated by the approximate constancy of the force increments for a given increment in contractile element activation, shown by the superimposed hollow bars in Fig. 5*B* (the bars on the $G_{fs} = 1$ and $G_{fs} = 2$ curves are not precisely the same because loop gain depends on contractile element output and muscle fiber length, the latter also depending on force and tendon compliance).

Any equilibrium point in Fig. 4 can be explained in terms of the curves of Fig. 5. For example, in Fig. 4*E, right,* the initial 10-N force step results in an equilibrated length before feedback activation of 9 mm, as predicted by the open-loop curve ($G_{fs} = 0$, force = 10 N) of Fig. 5. When positive force feedback is activated (Fig. 4*E, right, t* = 0.5 s, $G_{fs} = 2$), the muscle shortens, equilibrating at 6 mm, the equilibrium length predicted in Fig. 5 ($G_{fs} = 2$ curve, force = 10 N). The second load increment causes a stretch to 7 mm (Fig. 4*E, right*), again in agreement with the length predicted for 20 N by the $G_{fs} = 2$ curve in Fig. 5. In more general terms, Fig. 5 allows one to predict closed-loop displacement responses to any changes in force feedback gain and load,

although it does not provide information on stability. An affirming reaction (muscle shortening on increased load force) is then seen simply as the equilibrium point behavior of the system starting without feedback and moving to a new stable length as a result of concomitant loading and activation of positive force feedback.

In summary, the simulations in which the nonlinear reflex model was used confirm that positive force feedback can provide stable load compensation. The slopes of the closed-loop trajectories in Fig. 5 show that stiffness increases with G_{fs}. For a given load and G_{fs}, the system has a unique equilibrium length. If force feedback and inertial loading commence simultaneously, the muscle will either shorten or lengthen to this equilibrium point depending on the initial conditions. Delays in the force feedback pathway, inherent muscle viscoelasticity, concomitant negative displacement feedback, and/or β-mediated positive displacement feedback can all contribute to the stability of closed-loop operation.

DISCUSSION

Our experiments and analysis verify that positive force feedback in the neuromuscular system can provide stable

and effective load compensation. The analysis also shows that the conclusions regarding the stabilizing influence of muscle intrinsic properties, length feedback, and delays in positive feedback pathways were robust in the face of large parametric and structural variations in the systems considered (see APPENDIX). Stable behavior for large values of positive feedback gains was unexpected and initially quite puzzling. However, it became apparent that loop gain did not remain high, but rather it was automatically attenuated when muscles shortened and thereby reduced their force-producing capability.

Load compensation

Since the publication of the landmark papers by Pearson and Duysens (1976) and Conway et al. (1987) demonstrating reflex excitation of load-bearing muscles by their own load sensors during cat locomotion, most of the functionally related discussion in the mammalian literature has centered on the potential of such reflexes to reinforce load compensation, a role previously relegated to intrinsic muscle properties and reflexes mediated by muscle spindles (Feldman 1966, 1986; Marsden et al. 1977). Yet it was clear that the reflex mechanism in question represented positive feedback and this was normally associated with instability. It was tacitly assumed that the nervous system would somehow always limit positive force feedback gain within a range consistent with stability. Our results suggest that a combination of intrinsic muscle properties, concomitant negative displacement feedback, and reflex delays found in neuromuscular systems may provide this automatic gain control. The characteristic affirming reaction that results for loop gains that are initially greater than unity can only occur in a permissive framework such as this, and as far as we know, it has not been described before. From a functional point of view, the affirmation of ground contact, described many years ago as Rademaker's ''magnet reaction'' (Roberts 1979), has an obvious weight-supporting role during the stance phase of gait. It may also contribute to load compensation in other motor tasks and systems. In a more general sense the affirmation of contact is a possible solution for the problem of ''contact bounce'' encountered with the use of negative force feedback in robotic actuators (An et al. 1988; Hogan 1985). In a recent robotics study, Tarn et al. (1996) showed that positive acceleration feedback was useful in eliminating contact bounce.

Stability

It is a routine and essential aspect of the design of any control system to analyze the stability criteria. The two most common manifestations of instability are oscillation and monotonic unrestrained increases in the output variable. In a simple feedback loop, if the gain of the open-loop transfer function G is unity at a given frequency, the transfer function of the same loop when closed in positive feedback mode is $1/(1 - G)$, which has an infinite value at that frequency (Phillips and Harbor 1991). This in turn implies infinite amplitude oscillations in closed-loop operation. Monotonic instability occurs in cases where positive feedback $G = 1$ in the steady state (frequency = 0). In practice, unstable oscillations are constrained by nonlinearities to a finite range: ''limit cycles,'' provided the actuator survives.

The stability of our models was analyzed with root locus and Nyquist analyses of the system transfer functions. The root locus technique gave some insight into the mechanisms of stabilization (see APPENDIX). The parameters that most affected stability were intrinsic damping, inertia, and tendon compliance. As regards the stabilizing action of delays or poles (i.e., low-pass filtering) in the positive force feedback pathway, this presumably came about because negative displacement feedback, which opposes displacements caused by force feedback, requires a displacement error to generate the opposing force. In isometric contractions or in the absence of the delay, force builds up regeneratively in the positive force feedback loop before any significant displacement of the inertial load can occur, and the system goes unstable. This also highlights the stabilizing influence of length feedback via intrinsic muscle stiffness. Stated more generally, a phase lag in a positive feedback pathway tends to be stabilizing, just as a phase advance in a negative feedback pathway tends to be stabilizing. This provides a possible rationale for the puzzlingly long latencies (30–40 ms) of extensor reflexes after foot contact in cat gait (Gorassini et al. 1994).

Another factor that tended to stabilize positive force feedback was concomitant negative displacement feedback (e.g., compare Fig. 4, G and H), provided that muscle shortening was allowed. In the nonlinear model, this feedback is in fact provided by three separate elements: the passive parallel stiffness, the active parallel stiffness, and muscle spindles. A passing reference to stabilization of force feedback by negative displacement feedback was made by An et al. (1988) in relation to a robotic manipulator designed to avoid contact bounce. In a similar vein, Houk (1972) and Grill and Rymer (1985) argued that positive feedback mediated by the β-fusimotor loop would automatically be held below a loop gain of 1 by virtue of being nested within the negative displacement feedback loop mediated by muscle spindles. Indeed it has been suggested that under suitable conditions the very presence of multiple reflex pathways may have a stabilizing action (Oguztöreli and Stein 1976). The repeated observation that spinal interneurons and motoneurons tested during fictive locomotion receive excitation from both group Ia spindle and Ib tendon organ afferents (Angel et al. 1995) is strong evidence for concomitant positive force and negative displacement feedback, at least in the cat.

Our experimental data led us to identify the length dependence of muscle stiffness as a source of automatic gain control of positive force feedback and therefore as a stabilizing influence. It should be pointed out that this mechanism would only become relevant if the active muscle were allowed to shorten. During the shortening the system would in effect be transiently unstable, a possibility that has been raised previously in relation to reflex control (Prochazka et al. 1989; Rack et al. 1984). Stein and Kearney (1995) have pointed out another possible gain-attenuating nonlinearity in shortening muscles: the rectification of reflex responses. This property was not taken into account either in our experimental study or in our simulations. Whether it would stabilize or destabilize positive force feedback is not known. Furthermore, if a movement were being controlled by cocontracting antagonist muscles, the shortening of one muscle and the resulting reduction in its force loop gain would be counteracted by lengthening of the antagonist and a resulting increase in its force loop gain. In muscles that encounter im-

TABLE 1. *Qualitative summary of the interaction between displacement and force feedback*

	Initial force feedback gain			
	Negative	Zero	Positive and <1	Positive and >1
No length feedback	Very compliant	Compliant (intrinsic stiffness alone)	Stiff	Very stiff, stable with delay, muscle adjusts to equilibrium length
Negative length feedback (e.g., gain = 1)	Compliant	Stiff	Very stiff	Very stiff, muscle adjusts to equilibrium length

movable loads and contract isometrically, positive force feedback with gains exceeding unity could become unstable, even in the presence of the stabilizing influence of loop delays and the gain-attenuating effect of series tendon compliance. If muscles such as these had reflex connections that mediated positive force feedback, the strength of these connections would either have to be low or under the context-dependent control of the CNS. It may therefore be the case that positive force feedback will only be found in muscles, such as the leg extensors, that are inertially rather than isometrically loaded and are activated without their antagonists.

In Table 1 we summarize the effect on load compensation of different combinations of force and displacement feedback as inferred from our study.

Biological implications

What are the wider biological implications of the results? First, we feel that the paradox of homonymous excitatory reflex action from force receptors, with its underlying connotation of instability, is now partly resolved. As we have seen, the dynamic properties and nonlinearities of the mammalian neuromuscular system are well suited to compensate for the destabilizing tendencies of positive force feedback. Concomitant negative feedback of displacement and positive feedback of force is therefore a viable and effective configuration. At this stage the neurophysiological evidence for positive force feedback in mammals is largely restricted to the cat locomotor system (Conway et al. 1987; Guertin et al. 1995; Pearson and Collins 1993), although some evidence for a static postural role in cats and humans has been adduced (Dietz et al. 1992; Pratt 1995). There is also a body of evidence from invertebrates suggesting positive force feedback in certain behavioral states (Bässler 1993; Burrows and Pflueger 1988; Lindsey and Gerstein 1977; Pearson 1993).

Although it has become commonplace to assume that the gain of force feedback, whether negative or positive, is generally low (e.g., Bennett et al. 1994; Crago et al. 1976; Gottlieb and Agarwal 1980), the evidence is either indirect (e.g., Matthews 1984) or obtained in reduced preparations in which the nervous system may not be responding normally (e.g., Hoffer and Andreassen 1981). However, in light of the recent evidence in the cat locomotor system, this issue now deserves to be reexamined. In particular, the contribution of tendon organ Ib input should be sought in the medium- and long-latency (40–100 ms) components of stretch reflexes, given the stabilizing effect of such latencies in positive feedback pathways. There are some indications that the pathways mediating negative force feedback in some muscles may be active at the same time that positive force feedback is active in other muscles (Pratt 1995). Furthermore,

force feedback and displacement feedback are distributed across limb segments in ways that suggest global control strategies tailored to the motor task (Bonasera and Nichols 1994; Harrison et al. 1983; Nichols 1989). The effect on overall stability and load compensation of these distributed reflexes is beyond the scope of the present study, although clearly it is of basic importance in understanding the control of multisegmented limbs.

The gain and indeed the sign of force feedback may be strongly modulated according to motor task and behavioral set. This is suggested by the switch in force-mediated reflex action from inhibition to excitation in some muscles in the transition from static posture to locomotion in the cat (Conway et al. 1987) and stick insect (Bässler 1983) and also by the fact that many interneurons may be interposed in the long-latency and long-duration Ib pathways described by Gossard et al. (1994). No such task-dependent reversal has been found in short-latency Ia reflex action, although the magnitude of H reflexes is reduced when going from standing to walking and from walking to running (Capaday and Stein 1986, 1987).

What motor tasks other than locomotion might be under positive force feedback control? The grasp reflex in infants is certainly comparable with the affirmative reactions described above. The grasp appears to be triggered by cutaneous input from the fingers or palm, but its strength and tenacity suggest regenerative feedback control. As the nervous system matures, the grasp response in its most basic form declines (Forssberg et al. 1995), but it may reappear after upper motoneuron lesions (Eliasson et al. 1995). This suggests that the reflex connections are still present after infancy but that in adults they are under set-related control. The force of a grasp depends on prior evaluation of the load and also on sensory input related to the force of the load on the fingers and palm (Johansson and Westling 1988). In a series of experiments in which the load was suddenly altered during a pinch grip, Johansson et al. (1992a–c) showed that grip force was scaled to load force in two phases: a rapid "catchup" phase with electromyographic latencies in the range of 60–120 ms (Johansson and Westling 1988; Johansson et al. 1992c; Winstein et al. 1991) and a longer-latency and more gradual "tracking" phase (Johansson et al. 1992c). Local anesthesia of the thumb and finger tips disrupted but did not abolish these responses, suggesting cutaneous mediation with additional force-related sensory input from other receptors (Johansson et al. 1992c). Regardless of the sensory receptors involved, the scaling of grasp force to load is certainly consistent with segmental positive force feedback, both in latency and effect (D. Collins and A. Prochazka, unpublished data). One prediction would be that force-related medium-latency reflexes should be less subject

A

Simple Reflex Model

B
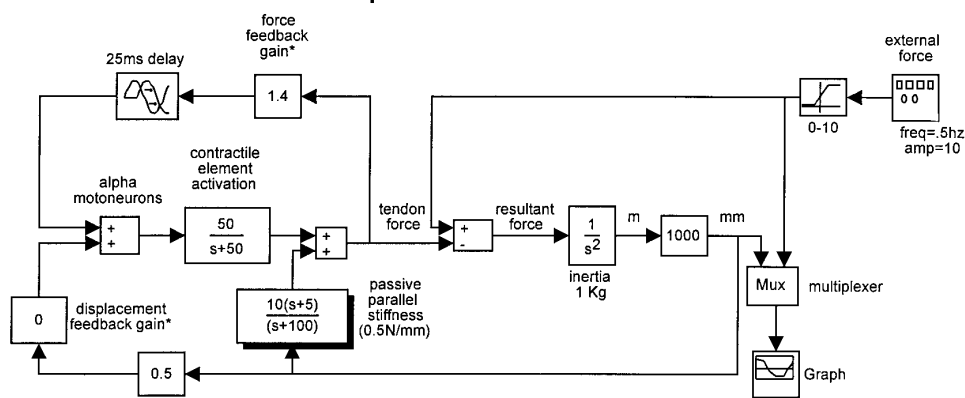
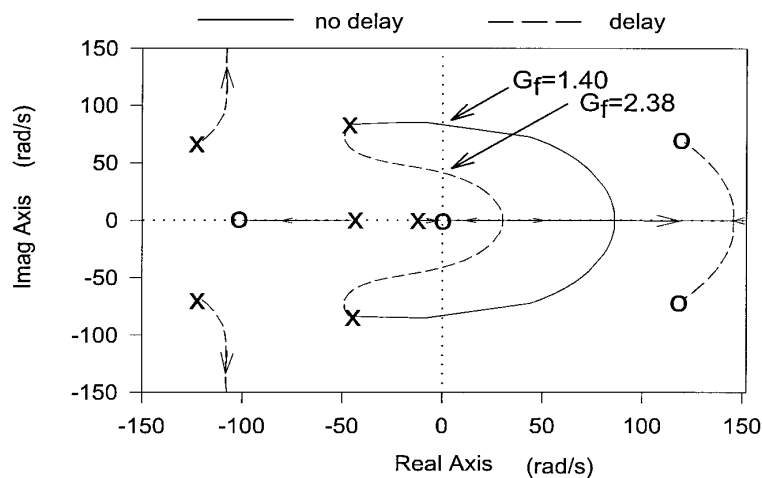### force gain root locus - simple reflex model

FIG. 6. *A*: system diagram of simple reflex model of Fig. 2 as it appears in Simulink graphic interface. Note that Matlab program does not allow an excess of zeros in a transfer function. This required inclusion of $(s + 100)$ term in shadowed box and corresponding gain adjustment. *B*: root locus plot of static reflex model, showing how 25-ms delay in positive force feedback pathway stabilizes system. Without delay (——), closed-loop poles cross into positive half plane for $G_{fs} = 1.4$. Inclusion of delay (- - -) pulls closed-loop roots back into negative half plane, thus allowing a higher gain ($G_{fs} = 2.38$) before instability occurs. Asterisk: element is a ''slider'' gain (i.e., an adjustable component).

to gating and may be exaggerated in amplitude in infantile and spastic grasp responses compared with those in normal adults.

Concluding remarks

In this paper we provide a theoretical analysis of the hypothesis that positive force feedback confers useful load compensation in the control of animal movement. Several nonlinear properties of the neuromuscular system appear to provide stabilizing influences. The neurophysiological evidence from different species indicates that the sign and gain of force feedback is highly task dependent. This suggests that positive force feedback may be appropriate in some muscles and motor tasks but not in others. Models such as those presented in this paper may help in understanding the reason for this task dependence in the future.

APPENDIX

Details of the models and root locus analysis

We analyzed several versions of the models presented in this paper with the use of Bode, root locus, and Nyquist plots. The idea was to see whether any generalizations could be made regarding

the stability of the systems and the sensitivity of the main conclusions to the choice of parameters. Figures 6–9 illustrate the Simulink versions of the models and corresponding root locus plots. The models may be implemented and run without modification in the Simulink program. For convenience, they may be obtained as digital files from the authors or from the following web site: http://gpu.srv.ualberta.ca/~aprochaz/hpage.html with the use of the file names shown in the bottom right corner of each figure.

Simple model

Figure 6*A* shows the simple reflex model. The muscle contractile element is modeled as a first-order low-pass filter with a cutoff frequency of 8 Hz, somewhat higher than the isometric frequency response characteristic of cat triceps surae muscles of Rosenthal et al. 1970. A mass of 1 kg represents the inertial load borne by a cat hindlimb. A force increment of 10 N represents the mean force developed by triceps surae during the stance phase of slow gait (Walmsley et al. 1978). Inherent muscle properties are simplified to a linear viscoelastic element with a stiffness of 0.5 N/mm. The force feedback and displacement feedback signals are represented without dynamics. The graph element allows a real-time view of the simulation and the multiplexer element chooses which signals are displayed. The simulation parameters in this and the dynamic reflex model were set for a 5-s run time of the Runge-Kutta-3 algorithm, with minimum and maximum steps of 0.5 ms.
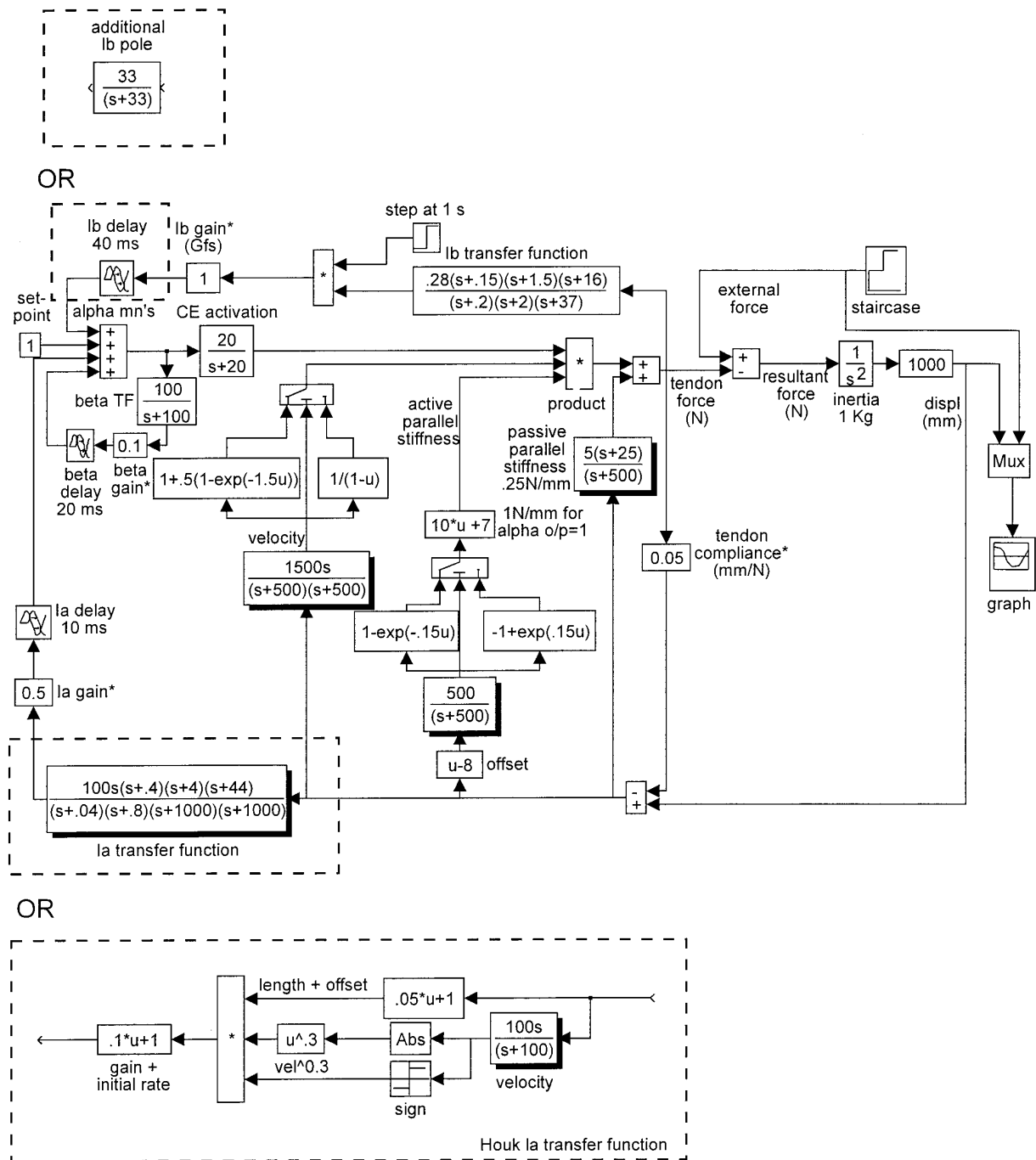
FIG. 7. Diagram of nonlinear reflex model of Fig. 3 as it appears in Simulink interface. As in Fig. 6A, because Matlab program does not allow an excess of zeros in a transfer function, extra poles and corresponding gain adjustments appear in shadowed boxes. In force feedback pathway we used either a time delay or a 1st-order low-pass filter element (dashed boxes at *top left*). Muscle spindle Ia responses were modeled either with a linear transfer function or nonlinear model described in text (dashed boxes at *bottom left*).

The model is highly simplified yet we found that, despite this, its closed-loop behavior showed all of the essential characteristics of the biologically more representative nonlinear models. This is the strongest argument for the generality of our conclusions.

Figure 6B shows the root locus analysis of the closed-loop behavior of the simple model with and without the 25-ms delay in the force feedback pathway. Without the delay the closed-loop poles cross into the positive half plane for $G_{fs} = 1.4$. Inclusion of the delay (represented by a 2nd-order Padé approximation, with pairs of zeros and poles at ± 120 and $\pm j69.3$ rad/s, respectively)

pulls the closed-loop roots back into the negative half plane, thus allowing a higher gain ($G_{fs} = 2.38$) before instability occurs. This stabilizing action of a delay in the positive feedback pathway was unexpected and mirrors the stabilizing action of phase advances in negative feedback loops.

Nonlinear reflex model

The nonlinear reflex model is more complex because it includes the β-skeletofusimotor loop, tendon compliance, and nonlinear
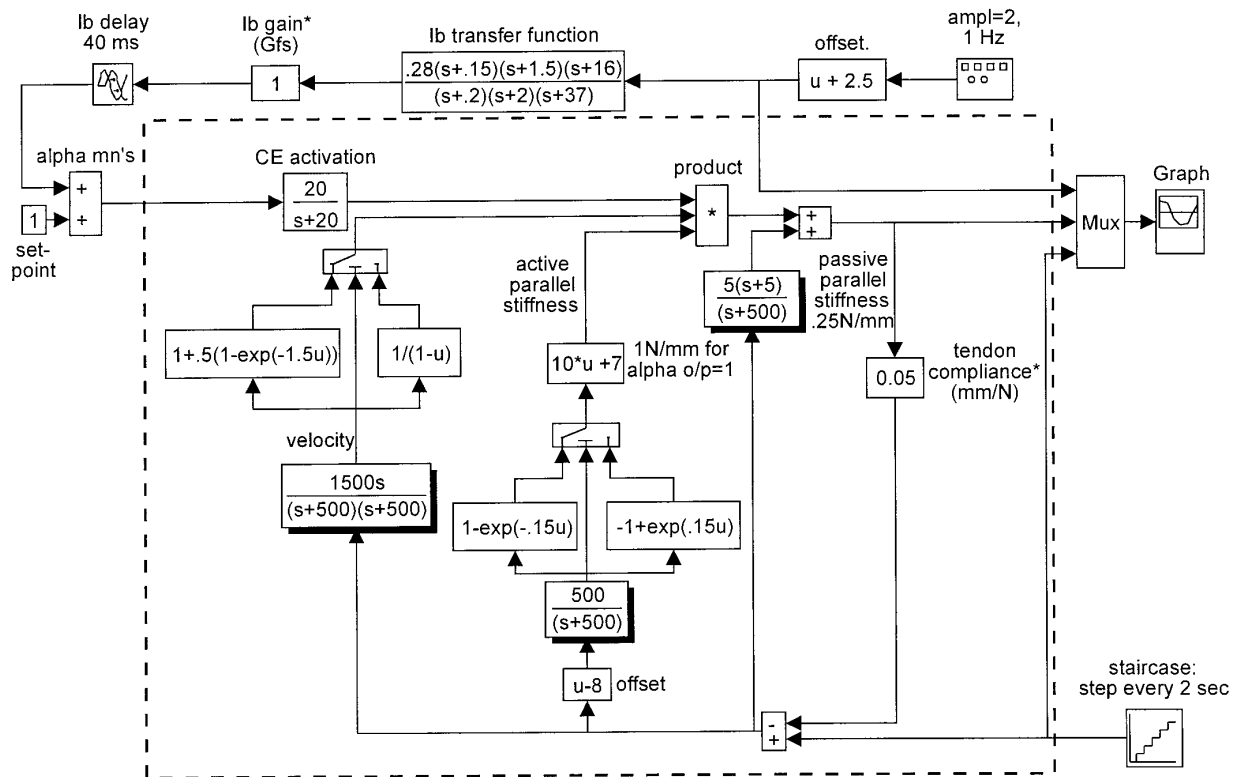
FIG. 8. System block diagram showing how a portion of nonlinear model of Fig. 6 is used to compute force loop gain. Length is treated as a contingent parameter (staircase generator at *bottom right*). A 1-Hz sinusoidal signal is supplied to input of force-sensing element. This transmits a signal via feedback pathway to contractile element, which, after combination with muscle intrinsic properties, results in a force output. Input and output force signals and displacement signal are gathered together and plotted against time. Force loop gain G_f is computed as ratio of output force signal to input force signal.

force-velocity and length-tension relationships that are combined multiplicatively with the neural input to muscle. There is also an alternative nonlinear model of spindle Ia transduction. Even so, several known properties of the neuromuscular system are neglected, such as force-dependent tendon compliance, high-pass filtering in the central nervous portion of the Ia reflex arc, Renshaw cell inhibition, etc. The validity of any model must be balanced against complexity (Winters 1990). We feel that our nonlinear model captures enough of the properties of the system to provide insight into the mechanism of interaction of muscle properties and force feedback and displacement feedback without getting lost in superfluous detail. The basic validity of the conclusions drawn from the modeling is supported by the data from the previous paper, in which real muscles were part of the feedback system.

Choice of parameters

The mass and external force parameters are the same as those of the simple model. Muscle properties are modeled with five main elements: the contractile element, the force-velocity relationship, the length-tension relationship, passive parallel stiffness, and tendon compliance. The contractile element is modeled as a first-order low-pass filter with a cutoff frequency of 3.2 Hz. Feedback via the series (tendon) compliance adds a second pole, also at ~3 Hz, giving an overall second-order characteristic in line with the isometric data of Rosenthal et al. 1970. Tendon compliance is represented as a constant of 0.05 mm/N (Elek et al. 1990; Rack and Westbury 1984). Force dependence of tendon compliance is neglected. The length-tension curve is a sigmoidal relationship made up of two exponential functions. The baseline shape constants and offsets were chosen to mimic the ascending portion of the curves described by Rack and Westbury (1969). The mean stiffness of this

element, assuming a unity value of output from the α-motoneuron element, is set to a baseline value of 1 N/mm (Bawa et al. 1976). The force-velocity curve was based on the work of Winters (1990) and is made up of a Hill-type hyperbolic function for negative (shortening) velocities and an exponential function for positive (stretch) velocities. The peak force for rapid stretches was assumed to be 50% greater than the isometric force (Winters 1990). Numerous shape constants were explored to mimic the range of length-tension and force-velocity curves in the literature. Force feedback was represented by a linear transfer function. That of Houk and Simon (1967) is shown in Fig. 7, but that of Anderson (1974) was also tested. Spindle Ia feedback was modeled by a linear transfer function (Chen and Poppele 1978), or, alternatively by the Houk et al. (1981) nonlinear model modified in the following way

$$\text{Ia response} = \text{constant}_1 + \text{constant}_2 * (\text{length-length}_o) * \text{velocity}^{0.3}$$

$$\text{Ia response} = \text{constant}_1 + \text{constant}_2 * (\text{length-length}_o) * \text{velocity}^{0.5}$$

where constant_1, constant_2, and length_o were derived from best fits to chronic cat Ia firing profiles (A. Prochazka and M. Gorassini, unpublished data). The Simulink representation of the Houk-type models is shown in Fig. 7. Note that this model assumes response symmetry for positive and negative velocities (the model of Houk et al. 1981 was not specified for negative velocities).

The 30- to 40-ms latency of autogenetic Ib excitation seen in cat locomotion (Gossard et al. 1994; Guertin et al. 1995) was modeled either as a simple time delay or as a single pole, $p/(s + p)$, where the baseline setting of the turning point frequency p was 33 rad/s, giving a time constant of 33 ms.

Parametric sensitivity

We studied the effect on closed-loop behavior of individually varying the main parameters of the nonlinear model from the base-

A

Linearized Reflex Model

B
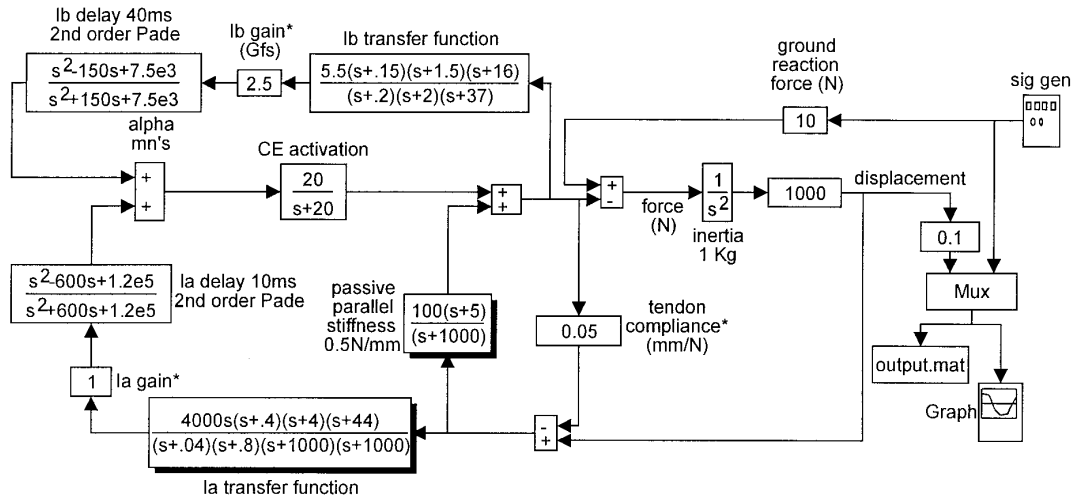
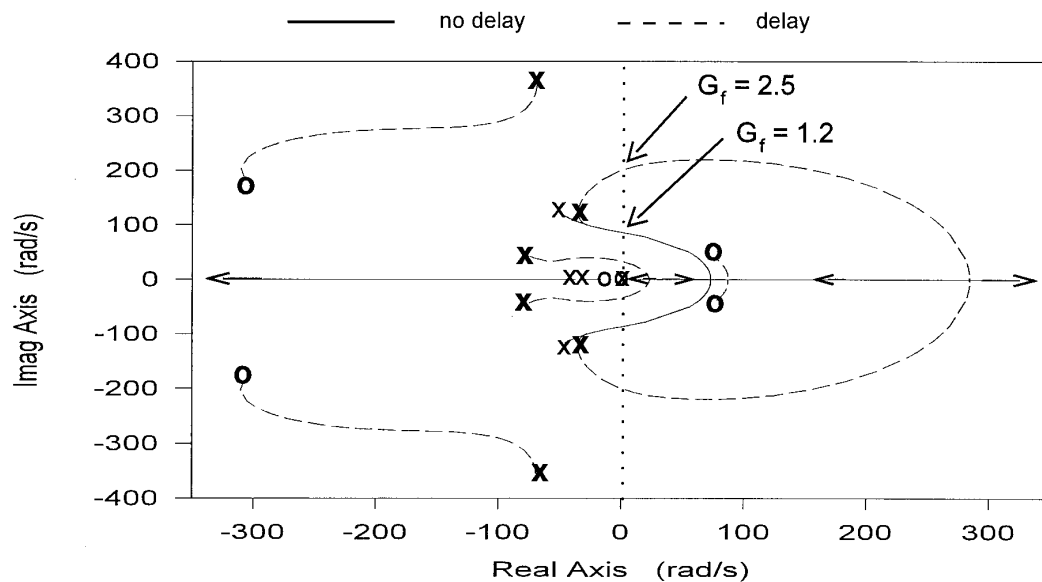## force gain root locus - linearized reflex model

FIG. 9. *A*: linearized version of nonlinear model of Fig. 7. *B*: root locus analysis of linearized model to show why 40-ms delay in positive force feedback pathway stabilizes system. *B*: without delay, closed-loop poles cross into positive half plane for $G_f = 1.2$. Inclusion of delay (represented by a 2nd-order Padé approximation, with pairs of zeros and poles at ±75 and $\pm j43.3$ rad/s, respectively) pulls closed-loop roots back into negative half plane, thus allowing a higher gain ($G_f = 2.5$) before instability occurs.

line operating settings shown in the block elements of Fig. 7. Table 2 shows the results. The range shown in Table 2, *third column,* is that over which the parameters could be varied without instability occurring or without any fundamental change in the closed-loop behavior in relation to our main conclusions. For all simulations in this analysis a step size of 0.5 ms was used. Larger step sizes (e.g., 1 ms) sometimes produced instability for computational reasons. In two cases the settings of several parameters were changed, as indicated in Table 2, *third column.* This was to explore parametric sensitivity in situations of particular interest, such as force feedback alone. The shape parameters a–c of the force-velocity

and length-tension curves are defined in the following equations used in the simulations

$$\text{for velocity} > 0, \text{force} = \text{constant} * (1 + 0.5 * e^{-a * \text{velocity}})$$

$$\text{for velocity} < 0, \text{force} = \text{constant} / (1 - b * \text{velocity})$$

$$\text{for length} > 0, \text{force} = \text{constant} * (1 - e^{-c * \text{length}})$$

$$\text{for length} < 0, \text{force} = \text{constant} * (-1 + e^{c * \text{length}})$$

(Length = 0 at the point of inflection of the sigmoidal length-

TABLE 2. *Parametric sensitivity analysis*

Parameter	Baseline Value	Range	Other Parameter Settings
Mass	1 kg	0.05–3 kg	Baseline
External force	10 N	0–100 N	Baseline
Setpoint (input)	1	−1–40	Baseline
G_d	0.5	0–2	Baseline
G_β	0.1	0–1	Baseline
G_f	1	0–17	Baseline
Force feedback delay	40 ms	0–1 s	Baseline
Force feedback delay	40 ms	25–100 ms	$G_d = 0$, $G_\beta = 0$, $G_f = 4$
Force feedback pole	33 rad/s	0–75 rad/s	$G_d = 0$, $G_\beta = 0$, $G_f = 4$
Displacement feedback delay	10 ms	0–50 ms	Baseline
Force-velocity shape, parameter *a*	1.5	0.1–10	Baseline
Force-velocity shape, parameter *b*	1	0.1–10	Baseline
Length-tension shape, parameter *c*	0.15	0.015–1.5	Baseline

G_d, open-loop gain of negative displacement feedback; G_β, open-loop gain of β-loop; G_f, open-loop gain of positive force feedback.

tension curve. Note that in the model, the offset block sets this point of inflection at a muscle displacement of 8 mm.)

The sensitivity analysis indicates that the main parameters could be varied over a large range without interfering with our main conclusions.

Determination of open-loop gain

Figure 8 shows the block diagram used to evaluate force loop gain in the nonlinear model. Spindle feedback and the β-loop are removed. Because force loop gain is length dependent, two inputs are provided, force and length. The length input is provided by a ''repeating sequence'' element, (*bottom right*) set up to produce a staircase of unity increments at 2-s intervals. The input force signal (*top right,* including offset block) is a 1-Hz sinusoid that varies between 0 and 4. This frequency and amplitude were chosen arbitrarily. The force input signal is transmitted via the Ib transfer function, gain, and delay blocks to the contractile element block, resulting in an output that is multiplied by the outputs of the length-tension and force-velocity feedback blocks. In essence, all of the components within the dashed line comprise the muscle model. The two input signals and the force output signals are gathered together and displayed by the graph block. Force loop gain is computed as the ratio of the output force signal to the input force signal and varies with displacement.

Root locus analysis of linearized version of nonlinear reflex model

Because there are no general methods for analyzing nonlinear systems, the feedback loop and the length-tension and force-velocity loops were removed from the nonlinear reflex model to facilitate a linear root locus analysis. The linearized version is shown in Fig. 9*A*. In addition, to represent the delay by a rational transfer function, a second-order Padé approximation was used.

As can be seen from the root locus plots in Fig. 9*B,* the dynamic response of each system is dominated by a pair of complex roots near the imaginary axis. The effect of the delay (or pole/zero roots of the Padé polynomial) is a modified root locus plot where the path of the dominant complex roots has been shifted left in the negative s plane. The addition of the delay not only provides more relative stability, it actually stabilizes an otherwise unstable system

for a certain range of gain. This allows for higher closed-loop gains before the roots cross into the positive half plane. However, this was only true over a limited range of delays (typically between 10 and 200 ms in the models we studied). Furthermore, with some parametric combinations, delays did not stabilize the systems. For example, if in-series (tendon) compliance was made very high in the reflex models, the system could not be stabilized by delays in the positive force feedback pathway. Thus, in much the same way that a properly designed phase-lead compensator will contribute a positive angle which may aid in the stabilization of a negative feedback system, we can conclude that delays in positive feedback pathways may have a stabilizing influence, but this is not necessarily the case in all such systems.

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