Positive Force Feedback Control of Muscles

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Prochazka, Arthur, Deborah Gillard, and David J. Bennett. Positive force feedback control of muscles. *J. Neurophysiol.* 77: 3226–3236, 1997. This study was prompted by recent evidence for the existence of positive force feedback in feline locomotor control. Our aim was to establish some basic properties of positive force feedback in relation to load compensation, stability, intrinsic muscle properties, and interaction with displacement feedback. In human subjects, muscles acting about the wrist and ankle were activated by feedback-controlled electrical stimulation. The feedback signals were obtained from sensors monitoring force and displacement. The signals were filtered to mimic transduction by mammalian tendon organ and muscle spindle receptors. We found that when muscles under positive force feedback were loaded inertially, they responded in a stable manner with increased active force. The activation attenuated the muscle stretch (yield) that would otherwise occur in the absence of feedback. With enough positive force feedback gain, yield could actually reverse. This behavior, which we termed the affirming reaction, was reminiscent of the mammalian positive supporting reaction, a postural response elicited by contact of the foot with the ground. Muscles under positive force feedback remained stable, even when the loop gain (G_f) was set at levels of 2 or 3. In a linear system, if $G_f > 1$, instability occurs when the loop is closed. On further investigation, we found that G_f changed with joint angle: it declined as the load-bearing muscle actively shortened. We inferred that in closed-loop operation, the active muscles always shortened until G_f approached unity. In other words, the length-tension curve of active muscle ensures stability even when force-related excitation of motoneurons is very large. Concomitant negative displacement feedback reinforced and stabilized load compensation up to a certain gain, beyond which instability occurred. In further trials we included delays of up to 40 ms in the positive force feedback pathway, to model the delays recently described for tendon organ reflexes in cat locomotion. Contrary to expectations, this did not destabilize the loop. Indeed, when instability was deliberately evoked by setting displacement feedback gain high, delays in the positive force feedback pathway actually stabilized control. The stabilization of positive force feedback by inherent properties of the neuromuscular system increases the functional scope to be expected of feedback from force receptors in biological motor control. Our results provide a rationale for the delayed excitatory action of Ib heteronymous input on extensor motoneurons in cat locomotion.

INTRODUCTION

Since the 1950s, it has often been assumed that force feedback to the spinal cord from tendon organ afferents caused reflex inhibition of the receptor-bearing muscles, whereas displacement-related feedback from muscle spindles caused reflex excitation. In both cases the reflex mechanism was equivalent to negative feedback, because a change in the sensed variable produced a muscle response that opposed the change. The force feedback loop tended to hold force constant in the face of perturbations, whereas the displacement feedback loop tended to hold length constant. The simultaneous operation of force and displacement feedback loops was shown on theoretical and empirical grounds to endow muscles with springlike properties (Houk 1972, 1979).

Whereas the reflex action of muscle spindle (group Ia and II) afferents on homonymous and synergistic muscles generally fits the simple scheme outlined above, tendon organ Ib reflex action, which is entirely mediated through interneurons, is more complex (Matthews 1972). First, the reflex action of flexor Ib afferents from a muscle diverges to many muscles of the limb (Bonasera and Nichols 1994; Harrison et al. 1983; Pratt 1995). Second, individual Ib interneurons receive input from Ib afferents of several muscles as well as input from Ia afferents (Jankowska and McCrea 1983; McCrea 1992). They also receive descending input, for example from the rubrospinal tract, which modulates their action on target motoneurons (Jankowska 1992). Third, reflex action from flexor Ib afferents tends to be much weaker and more variable than from extensor Ib afferents (Harrison et al. 1983; Rymer and Hasan 1980). Notwithstanding these complexities, in the immobile decerebrate cat the reflex action of extensor Ib afferents does in general inhibit extensor activity in the limb and promotes flexor activity, which is in keeping with the idea of negative force feedback (Bonasera and Nichols 1994; Jankowska 1992).

The issue was given a functionally broader slant when Pearson and Duysens (1976), extending the classical findings of Sherrington (1910) and Denny-Brown (1929), showed that force in hindlimb extensors was a crucial control signal for the transition from the stance phase to the swing phase of gait in the decerebrate locomotor cat. If extensor force was kept above a certain level, for example by mechanically constraining the ankle in a dorsiflexed position, extensor activity was maintained and the swing phase was delayed, in some cases indefinitely (Duysens and Pearson 1980). Analogous results have been obtained in invertebrates, where stimulation of load-sensing campaniform sensilla produces excitatory responses in load-bearing muscles during locomotion (Bässler 1993; Cruse 1985). In several studies since, it has become quite clear that extensor Ib afferents are primarily responsible for the effect in mammals (e.g., Conway et al. 1987; Pearson and Collins 1993). Obviously a Ib-mediated reinforcement of muscle force is the reverse of what occurs in the immobile decerebrate cat described above. It suggests positive rather than negative force feedback. At this stage it is unclear whether the positive force feedback effects are continuous and proportional, or whether they are used within another type of control strategy, such as finite-state control (Prochazka 1996a,b). To qualify as proportional feedback, an increment in Ib input should cause a more or less propor-

tional reflex increment in homonymous muscle activity. There is reasonable evidence to support this. Trains of extensor Ib stimuli in fictive locomotion have been shown to elicit proportional reflex excitation of extensor motoneurons (Brownstone et al. 1994; Gossard et al. 1994; Guertin et al. 1994, 1995). The motoneuronal depolarizations had a long latency (25 ms: Brownstone et al. 1994; Gossard et al. 1994), a slow buildup, and long durations (>100 ms: McCrea et al. 1995). In normal walking cats, input from afferents signaling ground reaction force was shown to reinforce extensor activity, also at unexpectedly long latencies (Gorassini et al. 1994; Hiebert et al. 1994).

Given the likelihood that positive force feedback is indeed present to some extent during locomotion and perhaps in some static postural tasks as well (Dietz et al. 1992; Pratt 1995), we decided to examine the implications of such feedback analytically. Little information was to be obtained from the control systems or robotics literature because positive force feedback is generally equated with instability and is therefore avoided in engineering design (Phillips and Harbor 1991). However, positive feedback has previously been posited for certain subsystems of the CNS (e.g., Houk 1972, 1979; Houk et al. 1993). In mammalian reflex mechanisms, positive length feedback has been considered before (Houk 1972), but, to our knowledge, the interaction of muscles and loads under positive force feedback control has not been analyzed quantitatively.

In this paper we describe the findings of trials on human subjects in whom muscles were electrically stimulated under force and displacement feedback control. Several interesting features were observed. First, positive force feedback control remained stable even when the isometric loop gain (G_f) had been set greater than unity. Closer investigation showed that this was probably due to the nonlinear intrinsic properties of active muscle, which automatically attenuated force feedback gain as the muscle shortened so that a stable equilibrium point was always reached. Thus, when unloaded muscles under positive force feedback were inertially loaded, they briefly yielded, but then quickly built up an opposing force and shortened to a new equilibrium point. Second, the inclusion of delays that mimicked those in cat Ib pathways stabilized the positive force feedback. This is opposite to the destabilizing effect of delays in negative feedback loops. To verify this unexpected effect, we first deliberately destabilized muscles under positive force feedback control by adding negative length feedback of a sufficiently high gain. This produced limit-cycle oscillation. Then we added delays ranging from 20 to 100 ms to the force feedback loop. This encompasses the range implicated in the cat locomotion data. The limit-cycle oscillations were immediately damped. The stabilizing effect of delays in the positive force feedback systems studied here is formally verified in the analytic models presented in the companion paper (Prochazka et al. 1997a).

The overall conclusion of our study is that positive force feedback control of muscles is an effective and surprisingly stable way of controlling load-bearing tasks such as locomotion. Automatic gain compensation due to intrinsic muscle properties ensures stability even when motoneuronal responses to force feedback signals are large. The long delays in Ib-mediated reactions previously observed in cat locomotion are well suited to stabilizing concomitant force and displacement feedback of high gain. The stabilizing effect of delays in positive feedback systems may be of general importance in control systems theory as well as in considerations of brain function and pathology.

METHODS

The experiments were carried out in human subjects with the use of a hybrid technique in which part of the control loop was the neuromuscular machinery of the limb of a subject and part was mimicked artificially—the sensory feedback, central processing, and generation of efferent commands to the muscles through electrical stimulation of the motor nerves (Bennett et al. 1994; Jacks et al. 1988). In this way it was possible to externalize and parametrically control just the feedback pathway while retaining the actuator properties of the muscle/load, which incorporate several important nonlinearities.

Four normal adult subjects (age range 29–49 yr) took part in the experiments with informed consent and the approval of the University of Alberta Hospitals Ethics Committee. A typical experiment lasted 3 h. In experiments on the hand, self-adhesive surface electrodes (ConMed Versa-stim: 45 × 45 mm) were placed on the skin overlying the motor points of the wrist flexor and extensor muscles (Fig. 1). A larger electrode (40 × 90 mm), which served as the indifferent, was attached to the skin proximal to the wrist crease. In experiments on the leg, similar electrodes were placed on the motor points of the foot dorsiflexors and plantarflexors (Fig. 1). The indifferent electrode was located laterally on the lower leg. Initially, subjects tended to intervene voluntarily, either assisting or resisting the external electrical control of their muscles. However, it was possible to train subjects to relax their muscles and eliminate any element of voluntary contribution (Bennett et al. 1994; Jacks et al. 1988). This lack of voluntary or reflex intervention was checked from time to time by unexpectedly turning off stimulation and looking for any residual activation of muscles. The setting up of stimulus parameters such as offset and gain for each muscle has also been detailed previously (Jacks et al. 1988) and is summarized below.

Force experiments

In a first set of experiments, force was measured with a load cell (Interface SM-50, stiffness > 200 N/mm) fixed under the knuckles of the hand or the ball of the foot (Fig. 1, force sensor 2). In three subjects an orthopedic elastic sock was stretched over the closed fist to prevent any hand opening during extensor stimulation and to provide a smoothly curved contact surface between the knuckles and the force transducer. A second, similar load cell (Fig. 1, force sensor 1), was used only to evaluate G_f and played no role in closed-loop trials (see below). Displacement was measured with a compliant spring sensor (Prochazka et al. 1992) or a linear variable displacement transducer (Prochazka et al. 1997b) that spanned either the wrist or ankle joint (Fig. 1*B*). The signals from force sensor 1 and the displacement sensor were conditioned with transfer functions that mimicked muscle spindles (Chen and Poppele 1978) and tendon organs (Appenteng and Prochazka 1984; Houk and Simon 1967), respectively (Fig. 2). In early trials this was achieved with programmable analog filters (Jacks et al. 1988). In later trials we used an on-line digital signal conditioning system comprising an analog-digital interface (CED 1401) and a personal computer running Matlab 4.2c, Simulink 1.3c, and Real-Time Workshop 1.1b software through a digital signal processor (dSpace DS1102, Real-time interface 2.2).

The conditioned force and displacement signals were summed to provide a single analog feedback signal (Fig. 2). The difference

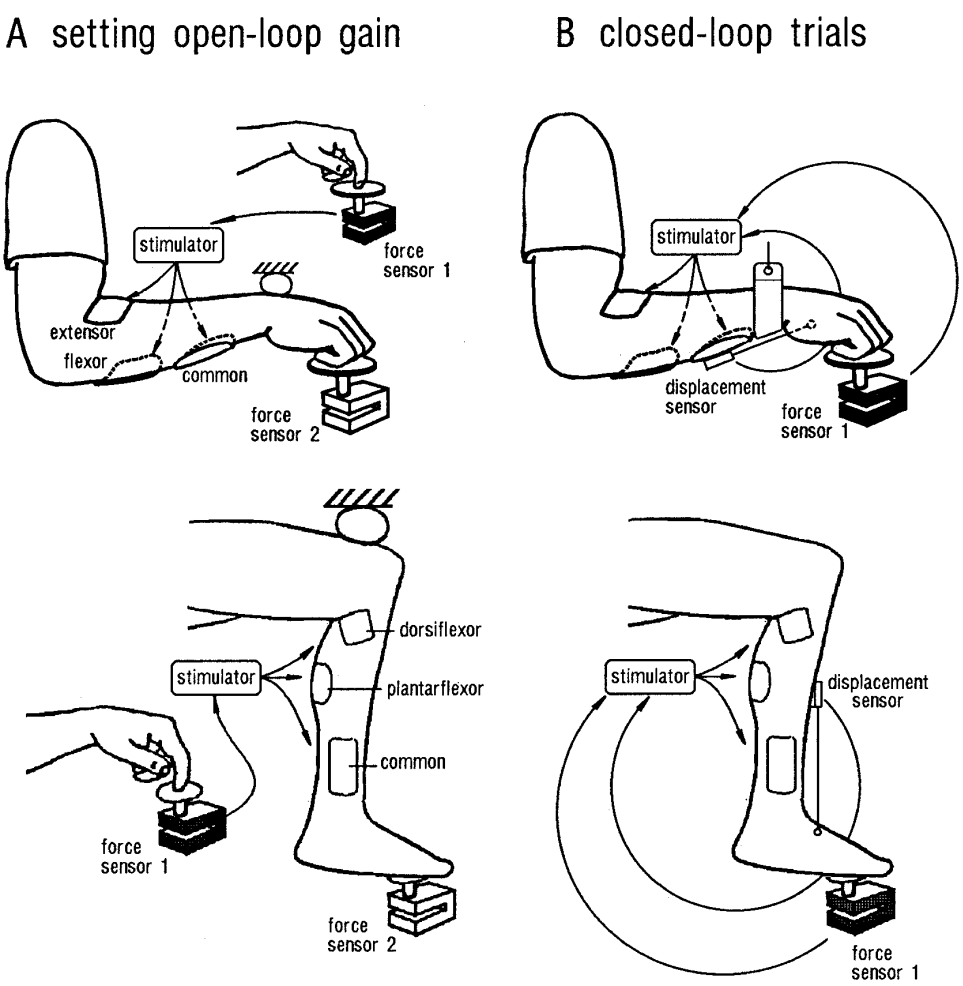

FIG. 1. Feedback control of electrically stimulated human muscles. *A*: setting open-loop gain (G_f). Force f_1 was applied manually to force sensor 1. Signal from this sensor, or a sinusoidal signal of same amplitude from a signal generator (Fig. 2, point A) was amplified and conditioned with the use of a transfer function mimicking tendon organ transduction (see Fig. 2, force feedback pathway). Conditioned signal and its inverse were used to amplitude modulate stimulus pulse trains activating flexor and extensor muscles, respectively (Fig. 2), through surface electrodes shown. Resulting force on force sensor 2 produced a signal f_2. Force gain $G_f = f_2/f_1$. G_f was set to values between 0 and 2. A similar method was used to set displacement open-loop gain (G_d) with the use of displacement sensor in *B*: details in text. *B*: closed-loop trials. Hand or foot was lowered onto force sensor 1. In hand trials, sling under wrist enabled experimenters to lower subjects' hands passively onto force sensor. This closed force feedback loop. Because feedback was positive, force applied to force sensor 1 resulted in increased activation of wrist flexors or foot plantarflexors and decreased activation of wrist extensors or foot dorsiflexors (see Fig. 2). Displacement sensors located as shown provided concomitant negative displacement feedback (conditioned feedback signals from force and displacement sensors were summed: Fig. 2).

between this signal and a set point signal was used to modulate muscle stimulation. As shown in Fig. 2, reciprocal control of flexors and extensors was achieved by the use of this difference signal to amplitude modulate a stimulus pulse train to the flexors. The inverse of the same signal amplitude modulated a stimulus pulse train to the extensors (30 stimuli/s, interleaved, biphasic current pulses, 1st phase rectangular 30–50 mA, 100 μs; 2nd phase 25 mA, exponential time constant 200 μs).

Gains were set under open-loop conditions as follows (see Figs. 1*A* and 2).

1) G_f: displacement feedback gain was set to 0. In the absence of muscle stimulation, force sensors 1 and 2 were pushed against each other and their amplifier gains were adjusted so that their signals, displayed on a Tektronix 5111 oscilloscope, were identical. Next, force sensor 2 was fixed either under the subject's distal joint or under the ball of the foot (Fig. 1*A*). The experimenter manually exerted force f_1 on force sensor 1. The signal from this sensor was now used to control muscle stimulation as described above, resulting in force f_2 measured by force sensor 2. (i.e., the loop was open at point A). Thus f_1 was the input, f_2 was the response, and so

$$G_f = f_2/f_1 \quad (1)$$

For convenience, we usually substituted the signal from force sensor 1 with a 1-Hz sinusoid from a signal generator (Fig. 2, point A) corresponding to a peak-to-peak force variation of 10 N. Note that G_f was determined under isometric conditions with the joints at neutral position (i.e., wrist angle 0° or ankle angle 90°). This is important, because we subsequently found that G_f varies with joint angle.

2) Displacement gain (G_d): this was determined by an analogous method. First, in the absence of muscle stimulation, the subject's joint was passively flexed and extended through ±30°, producing a signal of amplitude d_1 from the displacement transducer. A 1-Hz sinusoidal signal of amplitude d_1 was now supplied from a signal generator at point B in Fig. 2. This produced muscle stimulation that resulted in a sinusoidal movement of the subject's unloaded hand or foot, and a corresponding signal of amplitude d_2 from the displacement transducer. The loop was open at point B; d_1 was the input, d_2 was the response, and so

$$G_d = d_2/d_1 \quad (2)$$

Torque experiments

The point of contact of the closed hand with the force transducer illustrated in Fig. 1*A* shifted between the distal and proximal interphalangeal joints when wrist rotation was large. This meant that the moment arm from the wrist pivot to the contact point could vary by about ±5% of the mean through the range of motion of the wrist. The variation of the moment arm was reduced in the experiments in which the fist was bound in an elastic sock, which produced a more smoothly curved profile over the knuckles. However, to improve the precision of our results we performed a second complete set of experiments on three of the original subjects in which wrist torque and angle were the sensed variables. The sub-

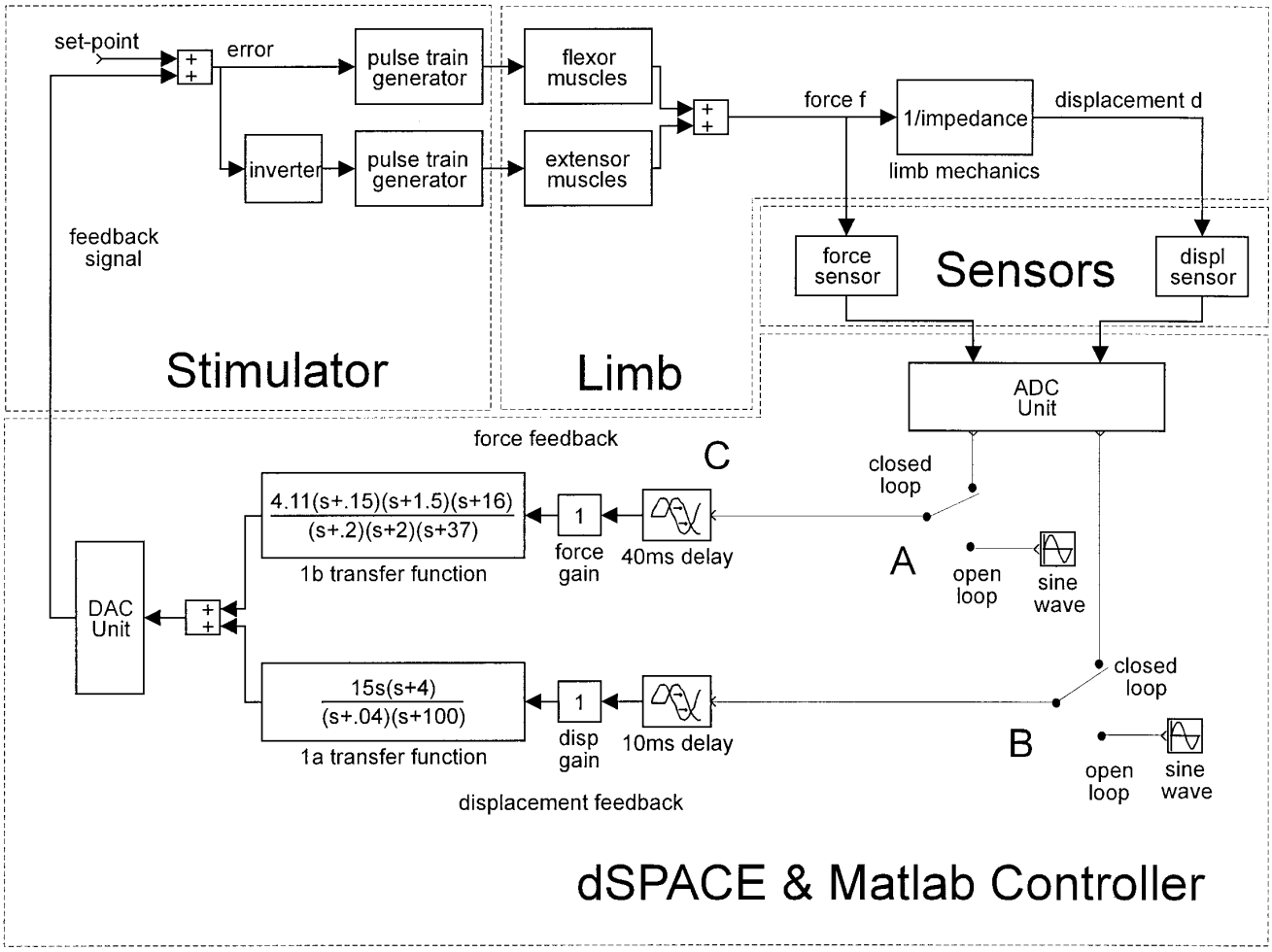

FIG. 2. Detailed schematic of experimental arrangement. Flexor and extensor muscles of limb were stimulated by reciprocally modulated pulse trains. Stimulator was driven by a control signal from D/A converter (DAC) of real-time dSpace/Matlab control system, running on a personal computer. This system implemented digital filtering that mimicked lumped conduction delays and transfer functions of ensembles of muscle spindle and tendon organ receptors in limb. Force and displacement signals that drove controller were obtained from force sensor 1 and displacement sensor illustrated in Fig. 1. Force sensor 2 was only used to determine G_f. Switches at points A and B allowed selection of open-loop or closed-loop operation. Sine wave generators at points A and B were used to determine G_f.

ject's closed fist was bound as before in an elastic sock. A light cast made of orthopedic Sandsplint was molded snugly over the fist (Fig. 3). A circular arc of Sandsplint fixed to the distal surface of the cast formed a vertical guide for a steel cable. The cable served to suspend the cast containing the subject's hand from a ceiling-mounted load cell. The cable wrapped part of the way around the arc from an attachment point at the bottom. It was kept in vertical alignment with the cast by virtue of a groove in the arc and two closely spaced sheet-metal guides. After separating tangentially from the arc, the 30-cm-long cable led vertically to a 1-m aluminum rod stiffener hanging from the load cell. The compliance of the force transducing system was 0.025 mm/N, equivalent to $0.2° \cdot N^{-1} \cdot m^{-1}$ about the wrist. The means of suspension of the hand cast ensured that there was negligible impedance to small lateral components of movement (abduction-adduction) as the wrist flexed and extended. The center of curvature of the arc corresponded to the center of flexion-extension rotation of the wrist. Tension in the cable was therefore proportional to the resultant flexor torque about the wrist. A Penny and Giles twin-axis goniometer was used to sense wrist angle. Comparisons with a joiner's level (Mayes Level and Angle Finder) showed the angle measurements to be accurate to within ±2.5° over the range of motion studied (±50°).

RESULTS

Figure 4 shows video tracings of the hand of a subject resting on the feedback force sensor, the forearm muscles being stimulated electrically under four types of control. The control parameters were set as follows. First, the subject's forearm was supported horizontally by one of the experimenters with the use of a sling just proximal to the wrist, as shown in Fig. 2*B*. Displacement and force feedback gains were set to 0 and tonic stimulation of wrist flexors and extensors was set to ~5% of maximal voluntary force so that the wrist stabilized at ~30° flexion. The hand was then lowered by the experimenter and placed on the feedback force sensor handle. The yield at the wrist in Fig. 4*A*, *top*, indicates that the intrinsic muscle stiffness under tonic stimulation was insufficient to compensate for the weight of the

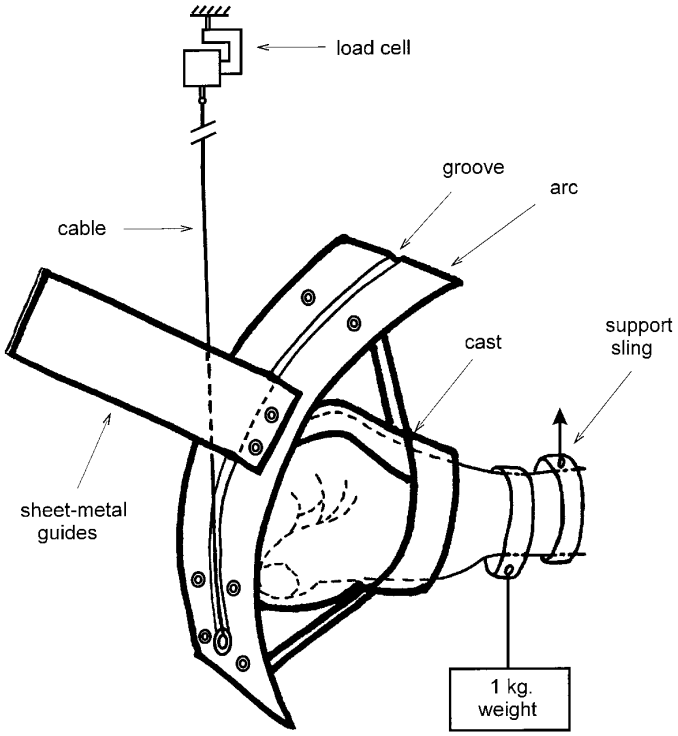

FIG. 3. Experimental arrangement whereby wrist torque and angle were sensed variables. Light orthopedic mold encased closed fist and provided curved surface or arc for a supporting cable. Center of rotation of arc coincided with axis of rotation of wrist joint. An angular goniometer (not shown) was used to monitor wrist angle and provide displacement feedback signal. Load cell mounted in ceiling above subject measured tension in cable, which, by virtue of mechanical arrangement, was proportional to torque. This load cell took the place of force sensor 1 of Fig. 1*B* in closed-loop trials.

forearm. With positive force feedback alone ($G_f = 0.8$, $G_d = 0$), the yield was reduced. Concomitant displacement feedback ($G_f = 0.8$ plus $G_d = -0.6$) further reduced the yield, the wrist equilibrating closer to neutral. When positive force feedback was raised further ($G_f = 1.5$, $G_d = -0.6$) the wrist muscles overcompensated and the hand equilibrated in the flexed posture shown in Fig. 4*A*, *bottom*. This behavior is reminiscent of the positive supporting reaction in decerebrate reflex standing (Creed et al. 1932; Denny-Brown 1929; Sherrington 1910).

Linear control systems theory would predict that when G_f exceeded unity, instability should have resulted and the wrist should either have oscillated or flexed maximally. We show below that the stable behavior observed is attributable to intrinsic mechanical properties of active muscle that automatically reduce G_f as the load-bearing muscles shorten. Similar results were obtained in trials on the legs of seated subjects, stimulating the ankle plantar- and dorsiflexor muscles (Fig. 4*B*).

A characteristic feature of the responses when positive force feedback was present was an active contribution to weight bearing that reduced yielding of the joint. The extent of this load compensation depended on G_f. Figure 5 shows averaged trials for five values of G_f: 0, 0.5, 1.0, 1.5, and 2. The mechanical arrangement was that illustrated in Fig. 1*A*. In the absence of force feedback ($G_f = 0$), the wrist joint yielded by 57° when the hand was lowered onto the force transducer. The flexor and extensor muscles were being tonically costimulated at ~5% maximal voluntary contraction, and so the 57° yield reflected the intrinsic stiffness of the muscles for this level of cocontraction. For $G_f = 0.5$, with the same initial stimulus conditions, net yield was reduced to 23°. For $G_f = 1$, net yield was only 11° (i.e., nearly complete load compensation). For $G_f = 1.5$ and 2.0, after initial brief yields, the wrist flexed back beyond the starting angle. This could be viewed as overcompensation or negative final yield.

In all three subjects we characterized the relationship between G_f (determined at neutral wrist position) and the final equilibrium angle after load compensation. Figure 6 shows that on average, there was a net yield for $G_f < 1$ and overcompensation (i.e., flexion beyond neutral) for $G_f > 1$. For $G_f = 0$, only the intrinsic stiffness of the tonically active muscles resisted the load and so yield was large. For $G_f = 1$, the wrist equilibrated close to the expected 0° equilibrium point.

Comparable data were obtained in ankle force trials. Thus in Fig. 7, trial 2 ($G_f = 0.8$, $G_d = 0$), the ankle initially yielded by ~30° and then stabilized with ~18° of net deflection. In trial 3 ($G_f = 1.0$, $G_d = -0.6$), the initial yield was ~25° and the ankle then returned nearly to the unloaded position (nearly complete compensation). In trial 4, a similar initial yield was followed by overcompensation ($G_f = 1.5$, $G_d = -0.6$). Damped oscillations in some of the records indicated that the control loop was only marginally stable at these gains in this subject.

Figures 8 and 9 show results of trials with the mechanical arrangement of Fig. 3, in which torque and joint angle were the sensed variables. The protocol of these experiments was developed to test results of the modeling presented in the companion paper and differed from the protocols of Figs. 5–7 in two respects. First, only the wrist flexors were stimu-

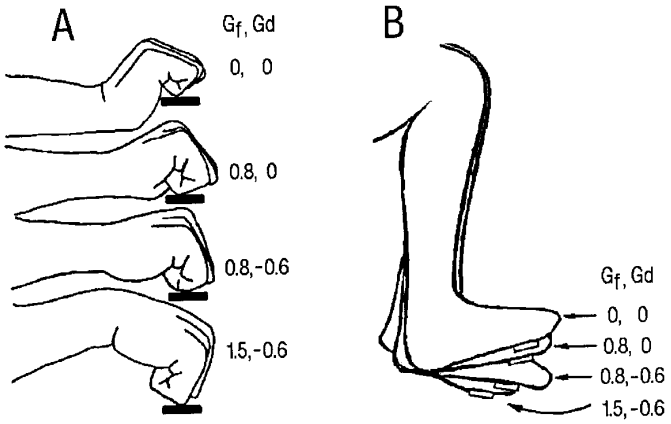

FIG. 4. Tracings of video frames showing equilibrium postures of (*A*) hand and (*B*) foot in closed-loop trials (see Figs. 1 and 2 for technique) under 4 feedback conditions. *Top tracings*: no active feedback. Intrinsic properties alone ($G_f = 0$, $G_d = 0$). Flexor and extensor muscles were tonically coactivated to ~5% maximal contraction. Weight of forearm and leg caused yield at wrist and ankle. *Second tracings*: intrinsic properties plus positive force feedback alone ($G_f = 0.8$, $G_d = 0$). Less yield at wrist and ankle. *Third tracings*: addition of negative displacement feedback ($G_d = -0.6$). Even less yield, joints equilibrated close to neutral position. *Bottom tracings*: strong positive force feedback and concomitant negative displacement feedback ($G_f = 1.5$), $G_d = -0.6$). Marked affirmative reactions at wrist (flexion) and ankle (plantarflexion).

lated. This was to simplify the analysis of muscle behavior under positive force feedback control. Because only the flexors were stimulated, the unloaded wrist, suspended by the support sling, adopted a flexed posture (−50°). The flexor muscles were therefore shorter than when G_f had been set (0° wrist angle). Second, an external load was added to the limb in the final part of each trial. This was to test the response of the positive feedback system to secondary loading.

Twenty-five trials, each lasting 60 s, were performed in each of four subjects. Between trials the forearm was passively suspended by a wrist strap for a 60-s rest period to minimize the effects of muscle fatigue. Each trial involved one of five randomized force feedback gains (0, 0.5, 1.0, 1.5, and 2.0). G_d was 0 throughout. A set sequence of events spaced by 10 s occurred in a given trial. Figure 9 illustrates these events as superimposed and averaged segments of data. At arrow 1, the unloaded wrist flexors were tonically stimulated at a level that had produced 5% maximal voluntary force at 0°. The unloaded wrist flexed to −50°. At arrow 2, the forearm was lowered to engage the cable to bear the weight of the forearm and hand. At arrow 3, the wrist yielded under the inertial load, equilibrating near 0° by virtue of the

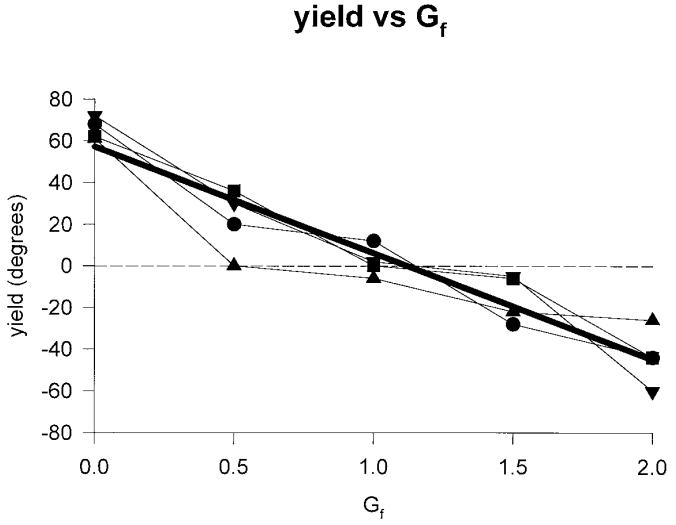

FIG. 6. Yield at wrist, plotted against G_f. Four subjects; each data point is mean of 5 trials. Thin lines join data points of individual subjects. Thick line: regression line. Note that for $G_f > 1$, yield is negative (overcompensation).

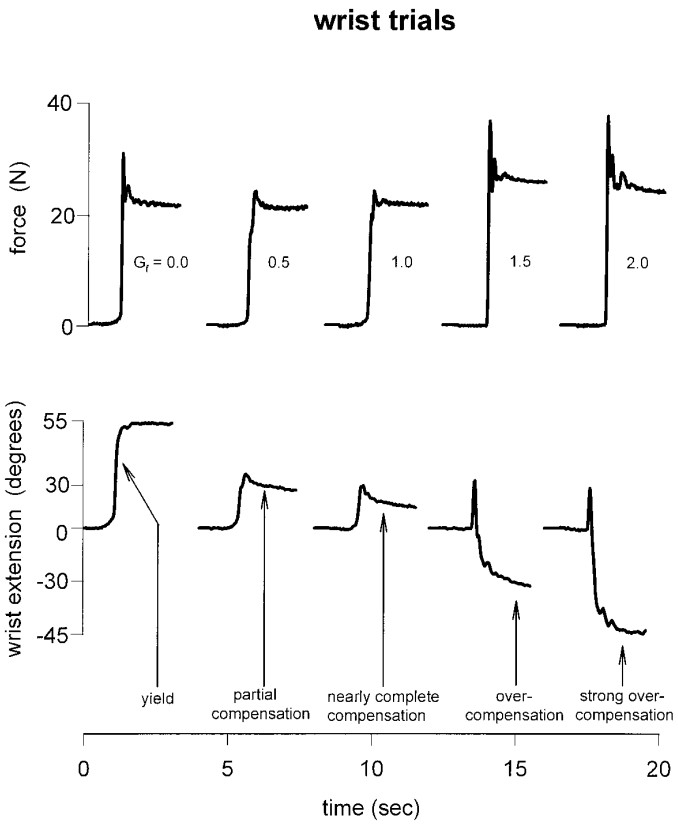

FIG. 5. Averaged force and displacement signals recorded in closed-loop wrist trials similar to those illustrated in Fig. 4*A*. Each pair of force and displacement traces represents 10 trials in which hand was lowered onto force feedback sensor and then was lifted away from it. Numbers within force traces: value of G_f. $G_d = 0$ in all cases. Initial stimulus conditions were such that flexor and extensor muscles were coactivated so that wrist started close to neutral (0°). For $G_f = 0$, wrist yielded (extended) to ~57°, as determined by intrinsic muscle stiffness. As G_f increased, yield progressively declined. For $G_f = 1.5$ and 2.0, yield actually reversed (overcompensation).

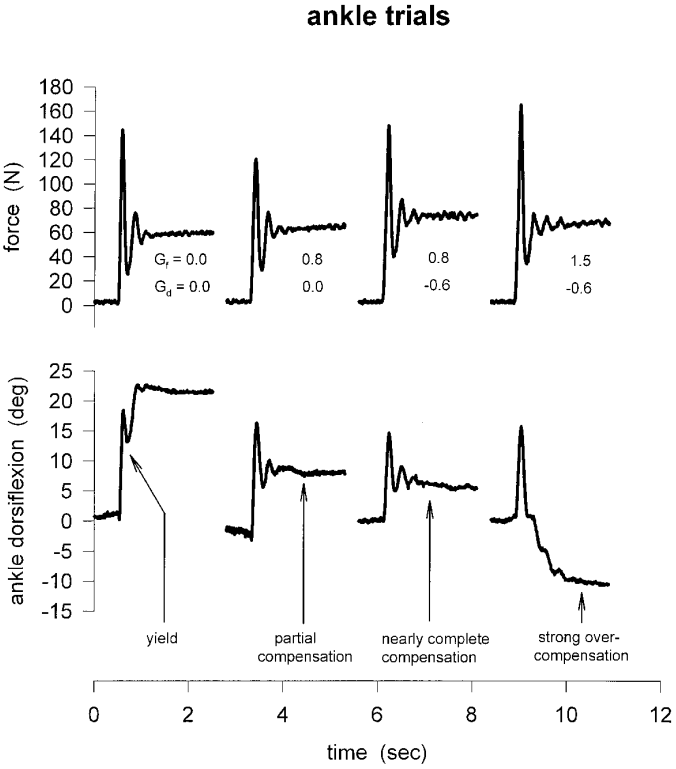

FIG. 7. Ground reaction force and ankle displacement as foot was lowered onto force sensor (as shown in Figs. 1*B* and 3*B*). Trial 1: no feedback, intrinsic muscle properties alone. Dorsi- and plantarflexor muscles were tonically coactivated to ~5% maximal contraction. Leg weight caused ~22° yield at ankle. Trial 2: intrinsic properties plus positive force feedback ($G_f = 0.8$, $G_d = 0$). Yield was reduced by 50% (''partial compensation''). Underdamped force response indicated marginal stability. Trial 3: $G_f = 0.8$, plus concomitant negative displacement feedback ($G_d = -0.6$), initial phasic yield, then nearly complete load compensation (final yield nearly 0). Trial 4: strong positive force feedback and concomitant negative displacement feedback ($G_f = 1.5$, $G_d = -0.6$). Initial yield, then marked overcompensation, so that foot equilibrated in plantarflexed posture: ''affirming reaction.''

intrinsic stiffness of the contracting flexor muscles. At arrow 4, positive force feedback was activated, causing the wrist flexors to contract more and the wrist to flex to a new equilibrium angle, i.e., to counteract some of the yield caused by weight bearing. In Fig. 8 this reduced the yield by 11° for $G_f = 0.5$ and by 21° for $G_f = 1.5$. At arrow 5, an additional weight of 1 kg was suspended from a sling at the wrist joint. For $G_f = 0.5$, this caused another 7° of yield, but for $G_f = 1.5$ there was no yield (i.e., the 1-kg weight was completely compensated for by the feedback response). This tested a prediction of the model presented in the companion paper, namely that when G_f is set greater than unity, weight bearing causes the muscles to equilibrate to a new length at which G_f approaches unity, and so further inertial loading is fully compensated for but overcompensation does not occur.

Figure 9 shows a complete set of averaged trials similar to those of Fig. 8 for five values of G_f: 0, 0.5, 1.0, 1.5, and 2.0. There was a clear dependence of load compensation, i.e., reduction in yield, on G_f in the initial weight-bearing trials. The data also show that compensation of the 1-kg secondary load was complete for $G_f = 1.5$ and 2.0. In fact there was a small overcompensation in the final part of the $G_f = 2.0$ trial in Fig. 9. At first glance there seems to be a discrepancy between these results and the marked overcompensations for $G_f > 1$ in Figs. 5 and 6. However, the length dependence of G_f alluded to above accounts for the paradox. In the protocol of Figs. 5–7 the muscles started near the length at which G_f had been determined. Furthermore, positive force feedback was active before first loading. This meant that for $G_f > 1$, weight bearing unleashed a strong feedback response that caused the flexor muscles to shorten beyond their unloaded length. In Figs. 8 and 9, on the other hand, the tonically contracting flexor muscles were at a much shorter length before loading. Feedback was activated only after initial weight bearing and yielding had occurred. In this case the effect of feedback was to reduce the yielding that had already occurred, but not to the point of reversing the yield. It is shown in the companion paper that the different behaviors in the two protocols were predicted on the basis of a length dependence of force feedback gain. This has important functional implications with respect to the modulation of force feedback gain in anticipation of loading. For the moment, however, it is sufficient to note that in all cases positive force feedback contributed to load compensation and the feedback system remained stable even when the G_f had initially been set greater than unity.

At the outset we were quite surprised that closed-loop operation remained stable for G_f values >1. For G_f values of 1.5, 2, and even 3, the wrist stabilized at progressively more flexed positions. This suggested that anatomic factors or intrinsic muscle properties were limiting the operating range under these conditions. The implication was that G_f was not the same at the very flexed position as it was at

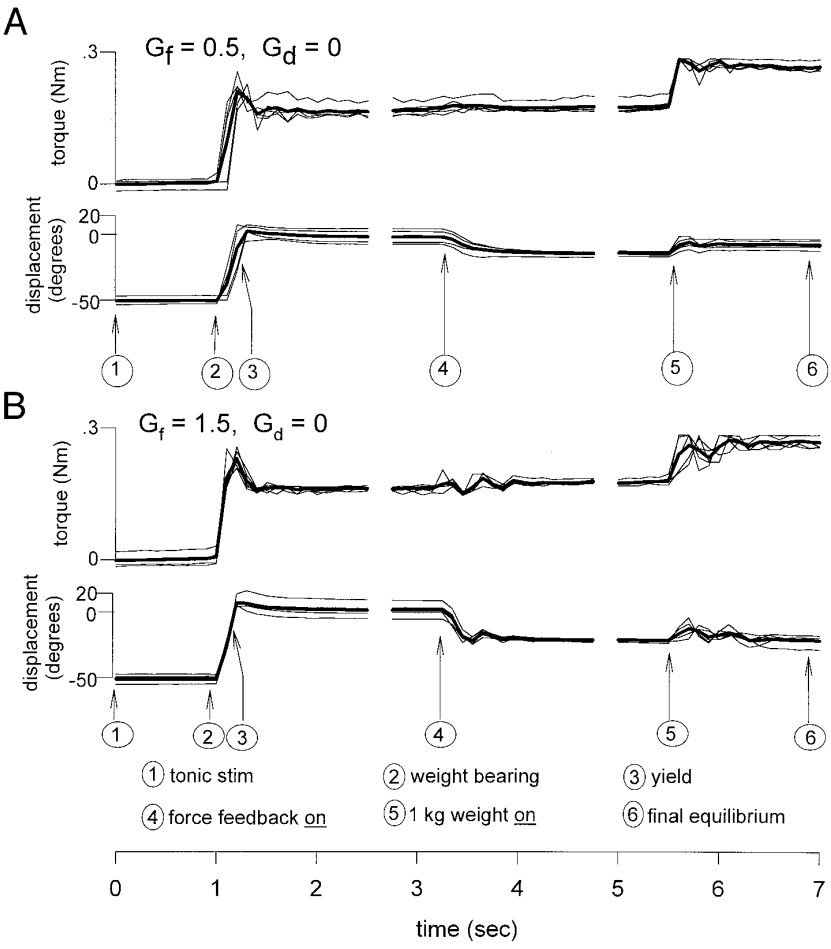

FIG. 8. Torque/angle trials in which mechanical arrangement of Fig. 3 was used. Protocol differed from that in Figs. 5 and 7 in that *1*) only wrist flexor muscles were stimulated; *2*) wrist therefore started in a very flexed position (arrow 1); *3*) initial weight bearing and yield (arrows 2 and 3) occurred before activation of positive force feedback (arrow 4); *4*) an additional 1-kg weight was suspended from wrist (arrow 5) to test response of control loop to a secondary load. Thin lines: 5 individual trials. Thick lines: averages of individual trials. Flexion responses after feedback was activated (i.e., after arrow 4) show that feedback contributed to load compensation and system remained stable for $G_f = 1.5$. There was complete load compensation (but not overcompensation) for 1-kg load when $G_f = 1.5$.

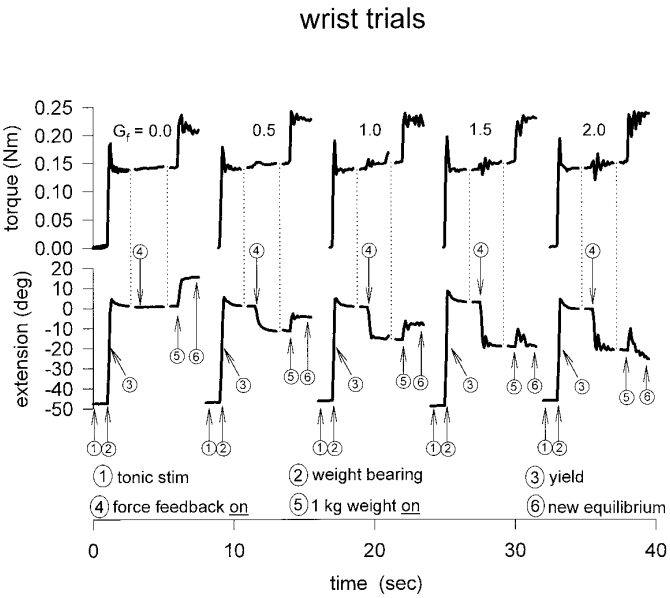

FIG. 9. Complete set of averaged trials of type shown in Fig. 8. Net yield caused by initial weight bearing was reduced in proportion to G_f after force feedback was turned on (i.e., after arrow 4 in each case). Secondary load caused additional yielding that was also reduced as G_f increased. For $G_f > 1$, 1-kg load was completely compensated for. Effects of positive force feedback on yield differed from those in Figs. 5–7 (i.e., little overcompensation) only because of differences in initial conditions and test protocols.

neutral. To verify this, we performed a series of G_f measurements at different wrist joint angles. This confirmed that G_f was indeed dependent on joint angle (Fig. 10). Furthermore, the data suggested that G_f was related to the length-tension curve of the active muscle. We feel that this is a key demonstration, because it implies that Ib-mediated excitation of homonymous motoneurons (positive force feedback) could be very strong and produce very high G_fs in the midrange of muscle length, and yet under inertial loading, stability would always be ensured because of an automatic attenuation of gain at shorter muscle lengths.

In the course of studying the analytic models described in the companion paper, it became apparent that a phase lag in positive force feedback pathways had a stabilizing effect on the closed-loop system. This was confirmed in all four subjects and is illustrated in the data from one subject shown in Fig. 11. In these trials, instability was deliberately provoked by setting G_d to ~ -6, in combination with $G_f = 2$. The hand oscillated in midair before being placed on the feedback force sensor. In Fig. 11*A*, when the hand was placed on the feedback force sensor (indicated by the horizontal bar), oscillation continued. In this case the delay at box *C* in the force feedback pathway in Fig. 2 had been set to 0. The delay was then set to 40 ms. As may be seen in Fig. 11*B*, this led to a rapid stabilization of the system as soon as the hand was lowered onto the force sensor. Instability resumed when the hand was lifted away from the force sensor. This effect was very reproducible from one trial to the next.

DISCUSSION

Our study was prompted by recent neurophysiological data showing that input to the CNS from receptors signalling extensor force results in excitation of extensor motoneurons, which in turn results in increased extensor force (Dietz et al. 1992; Gossard et al. 1994; Pearson and Collins 1993; Pratt 1995). Although it remains to be proven that proportional feedback is involved, we thought the evidence compelling enough to warrant a study of the behavior of limbs under positive force feedback control. In our experiments the ''real'' musculoskeletal apparatus of human subjects was used in the control loop. The nonlinearities of load-moving muscle that are otherwise difficult to model were thereby included. This turned out to be crucial, as shown by the stabilizing effect of length-dependent feedback gain.

The main results were as follows.

1) Positive force feedback was an effective means of generating load compensation.

2) For $G_f > 1$ (set under isometric conditions at midrange joint angles), inertial loading was resisted so strongly that instead of yielding, limbs actually pushed back beyond the starting position.

3) For $G_f > 1$, stable equilibrium positions were always reached. Stability at these gains was unexpected. We found that it was attributable to automatic reductions in G_f as the load-bearing muscles shortened (length-dependent gain).

4) A delay or phase lag in the force feedback path also improved overall stability, allowing higher levels of concomitant G_d. This was analogous to the stabilizing effect of a phase advance in a negative feedback loop.

The first result, that positive force feedback was effective in load compensation, could in fact have been predicted from standard linear control systems theory: as G_f approaches 1, inertial loading is resisted, with less and less yield of the active muscles. From a physiological point of view, the responses were reminiscent of the positive supporting reaction of Sherrington (1910) and also the ''magnet reaction'' of Rademaker (see Roberts 1979). Rademaker found in decere-

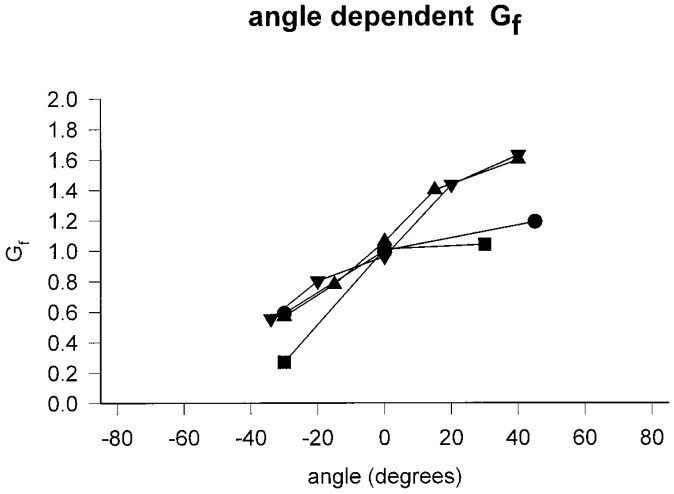

FIG. 10. Dependence of G_f on wrist angle. G_f was adjusted to be 1 at wrist neutral (0°) with the use of method illustrated in Fig. 1 and 2. Without changing stimulus parameters, G_f was then measured at various angles in range −30° to + 45°. Data are from 4 subjects. G_f is strongly dependent on wrist angle. For example, at 30° flexion, G_f is half that at 0°. This means that in closed-loop force feedback, as wrist moves into flexion, G_f declines. This suggests that in strong affirming reactions, such as in Fig. 6, G_f, which may significantly exceed 1 at long muscle lengths, equilibrates to unity as muscle shortens.

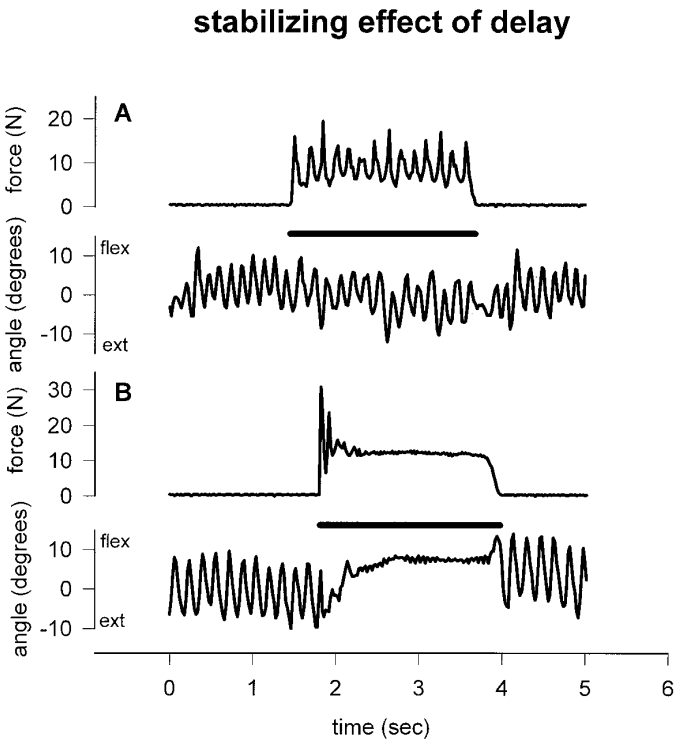

FIG. 11. Stabilizing effect of a delay in force feedback pathway. Wrist under a high and unstable level of displacement feedback ($G_d \sim -6$, $G_f = 2$) was supported in midair and then placed on force feedback sensor for period indicated in force trace. *A*: 0 delay in force feedback pathway (see Fig. 2). Limit-cycle instability resulting from high G_d persisted when hand was placed on force sensor. *B*: delay of 40 ms was added to force feedback pathway. When hand was placed on force feedback sensor, thus activating force feedback, loop was rapidly stabilized. Limit-cycle oscillations resumed when hand was lifted away from force sensor.

brate cats that when a pendant leg was supported by placing of the researcher's hand under the paw, the paw exerted an extensor force against the hand that then remained fairly steady even when the hand was moved down. In our subjects, this behavior occurred, for example, with moderate positive force feedback (e.g., $G_f = 0.8$) and weak concomitant displacement feedback (e.g., $G_d < 0.5$). The displacement feedback maintained a set position of the joint before inertial loading. The force feedback became active on contact with the support, in our experiments this being the feedback force sensor. We considered using the term ''positive supporting reaction'' to describe the way the limb pushed back beyond its starting point in our experiments. However, Sherrington (1910) associated nociceptive input with the positive supporting reaction. To avoid confusion, we have coined the term ''affirming response.''

From a functional point of view, result 1 above suggests a useful role of Ib-mediated positive force feedback from extensor muscles during gait. However, it has been recognized for some time that Ib autogenetic excitation also has the potential to be destabilizing if its gain is too high. Without the automatic gain control conferred by muscle length-tension properties, one would have to assume that Ib excitatory action at the reflex level was always kept well below levels corresponding to $G_f = 1$. This would greatly reduce the scope for effective load compensation. Furthermore, given the wide range of reflex gains in the normal population (Prochazka and Trend 1988; Rack et al. 1984), the potential for instability would be significant. The automatic stabilizing mechanisms described in results 3 and 4 are therefore very important, because they circumvent any need to posit safety margins within the nervous system to avoid unstable behavior. On the other hand, in muscles that encounter immovable loads and contract isometrically, positive force feedback could be unstable, even in the presence of the stabilizing influence of a delay in the force feedback loop. It could be that positive force feedback only exists in muscles, such as the leg extensors, that are always inertially rather than isometrically loaded.

The stabilizing effect of a delay in the positive force feedback path is particularly interesting. Just as a phase advance in a marginally stable negative feedback loop avoids overshoot by ''speeding up'' the attenuating effect of the feedback, a delay in a marginally stable positive feedback loop may avoid overshoot by retarding the amplifying effect of the positive feedback. The main extensor response to force feedback from extensor muscles comes at a latency of 30–40 ms (Gorassini et al. 1994; Pratt 1995). This latency, which is ~4 times as long as that of a segmental reflex, has led to the speculation that it is processed through the locomotor pattern generator (Pearson and Collins 1993) or through a finite-state or conditional control mechanism (Prochazka 1993, 1996a,b). The present results indicate that if the extensor response is viewed as proportional positive force feedback, the intrinsic muscle properties, coupled with a delay through interneurons, would ensure stability.

How might proportional positive force feedback be distinguished from other control strategies such as those alluded to above? One way is to disconnect a muscle from its tension receptors, stimulate the receptors in a graded manner, and look for graded force in the muscle. This is technically feasible in invertebrates: stimulation of load-sensing campaniform sensilla produced graded activity in detached flexor muscles in stick insects (i.e., the muscles that bear the animal's weight during the stance phase), thus demonstrating a positive force feedback effect (Bässler 1993; Bässler and Nothof 1994; Cruse 1985; Cruse et al. 1995; but cf. Libersat et al. 1987). In mammals, the force-sensing tendon organ receptors are embedded within muscles and cannot be separated from them. An alternative in this case is to cut the muscle afferent nerves and stimulate them. Because the nerves contain Ia and II afferents from muscle spindles as well as Ib afferents from tendon organs, the method relies either on small differences in the thresholds of the different types of afferent, on quirks of their connectivity, such as selective Ib reflex action from one muscle to another (e.g., plantaris to medial gastrocnemius), or on selective sensitivity to vibration (Angel et al. 1996; Pearson and Collins 1993). Gossard et al. (1994) found that heteronymous Ib stimulation evoked graded excitatory postsynaptic potentials in the 3,4-dihydroxyphenylalanine-treated spinal locomotor cat. This implies graded positive force feedback because Ib firing increases with increasing muscle force and so heteronymous motoneuronal excitation would also increase with increasing muscle force. As already stressed, the Ib autogenetic reflex excitation of extensors only becomes significant at latencies of 30–40 ms (Gorassini et al. 1994; Guertin et al. 1994, 1995; Pearson and Collins 1993). The excitatory

postsynaptic potentials recorded by Gossard et al. (1994) and Brownstone et al. (1994) were interneuronally mediated and although in some cases they were of short latency (3.5–4 ms), they facilitated slowly but strongly. Thus net depolarization in response to a train of Ib stimuli rose exponentially, also with a 30- to 40-ms time constant. These cat data therefore support the hypothesis underlying the present study. There are also indirect indications that graded positive force feedback may be present in the control of stance in humans (Dietz et al. 1992).

In conclusion, our study provides analytic and experimental evidence that positive force feedback can produce stable load compensation, with properties that complement negative displacement feedback. We coined the phrase affirming reaction to describe the pushing-back behavior that occurs when extremities are first loaded under positive force feedback. Length-tension properties ensure automatic control of force feedback gain when active muscles shorten during affirming reactions. This stabilizes the system even when Ib autogenetic excitation is very strong. The stabilizing effect of a delay in the force feedback pathway was unexpected and may explain why autogenetic Ib reflex excitation in cat gait has a relatively long latency. These conclusions are valid over a large range of system parameters, as shown in the companion paper, and so we feel that it is safe to extrapolate them to biological motor control systems in general.

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