JOURNAL OF NEUROPHYSIOLOGY
Vol. 76, No. 5, November 1996. *Printed in U.S.A.*

On the Voluntary Movement of Compliant (Inertial-Viscoelastic) Loads by Parcellated Control Mechanisms

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SUMMARY AND CONCLUSIONS

1. Experiments were performed to characterize the trajectories, net muscle torques, and electromyogram (EMG) patterns when subjects performed voluntary elbow flexions against different compliant loads. Subjects made movements in a single-joint manipulandum with different loads generated by a torque motor. Some series of movements were performed under entirely known and predictable load conditions. Other series were performed with the same known loads, interspersed, just before movement onset with occasional, unpredictable changes in the magnitude of the load.

2. To move a larger load, subjects increase the impulse (torque-time integral) by prolonging the duration of the accelerating torque while keeping its rate of rise constant. Subjects modulate torque most for inertial loads, less for viscous loads, and least for elastic loads, and modulation is greater under predictable than unpredictable load conditions.

3. Even when the loads are predictable, subjects move large inertial and viscous, but not elastic, loads more slowly than small. Unpredictable changes in load have a larger effect on movement kinematics than do known changes of the same magnitude.

4. Subjects prolong the duration and increase the area of the agonist EMG burst but do not change its rate of rise to move larger, predictable loads. Subjects change the area of the antagonist burst according to the torque requirements of the load, increasing it only for increases in inertial loads. These effects are usually greater for predictable than unpredictable loads but in either case, are highly variable across subjects.

5. Predictable loads that slow the movements delay the onset of the antagonist burst. When changes in load are unpredictable, only inertial changes affect antagonist latency.

6. The initial change in muscle force when there is an unexpected change in the external load is due to the viscous properties of muscle tissue. Electromyographic evidence of reflex changes in muscle activation follow this intrinsic mechanical response by 50–70 ms. Elastic neuromuscular properties may also be important but only late in the movement as the final position is approached.

7. We propose that the central command for a voluntary movement should be described by three elements. The first element (α) specifies the muscle activation pattern expected to generate dynamic forces adequate and appropriate to produce a satisfactory trajectory. This feed-forward control program uses simple rules, based on an internal model of task dynamics constructed from prior experience. The second element (λ) is a kinematic plan or reference trajectory utilizing the negative feedback of reflex action to partially compensate for errors in α or for unexpected perturbations during the movement. It defines the locus of a moving, instantaneous equilibrium position of the limb, a "template" for the intended trajectory. As movements become slower and require smaller dynamic (velocity and acceleration dependent) forces, λ will become the dominant control signal. It is also used for correction and updating of the internal model used to generate α. The third element (γ) modulates *volitional set,* the degree and manner in which multiple reflex mechanisms can contribute to the muscle activation patterns if the actual trajectory deviates from the planned one. Reflex mechanisms work in parallel with intrinsic muscle compliance to provide partial adaptation of neuromuscular system dynamics to external load dynamics. These controlled compliant mechanisms maintain the stability of the motor system, without which both posture and movement would not be possible.

INTRODUCTION

Fast, voluntary flexion of the elbow against an inertial load is produced by brief contractions of the agonist flexor and antagonist extensor muscles. The electromyogram (EMG) patterns associated with these contractions consist of a series of two or three partially overlapping bursts of activity (Angel 1975; Hallett et al. 1975; Wachholder and Altenburger 1926) followed by a prolonged, much lower contraction (Lestienne et al. 1981). These "pulse-step" patterns are planned in a stereotyped way to produce the desired features of an intended movement task such as distance and speed of the expected inertial load. Expectation of other kinds of external loads, such as viscous or elastic ones, produce different effects. No one has yet described general rules for how contraction patterns adapt different types and magnitudes of external load.

Load planning

How is the *central command* to the muscles adapted to accommodate known loads of different types and magnitudes? We address this here by systematically examining how the EMG patterns change when the subject is asked to move different known loads. The most commonly described pattern for fast, single-joint movements is a pulse step (Freund and Budingen 1978; Ghez 1979), reviewed in Gottlieb et al. (1989b). The "pulses" are the partially overlapping bursts of agonist and antagonist muscle activation. The "steps" are the much lower level of activity in both muscles that slowly decline (Lestienne et al. 1981). This pattern is adapted to larger inertial loads by prolonging the durations of both pulses, increasing the areas of the rectified EMG bursts (Angel 1975) and delaying the antagonist burst (Gottlieb 1993b; Gottlieb et al. 1989a; Smeets et al. 1990).

External viscous loads reduce the antagonist burst (Sanes 1986; Stein et al. 1988), similar to the effect of intrinsic limb viscosity (Lestienne 1979). Elastic loads demand static torques that are produced by changes in the step activation component (Richardson and Simmons 1985; Stein et al. 1988). No studies have reported the electromyographic consequences of systematically varying the magnitudes of the loads nor have they contrasted the effects of loads of different types.

We tested the hypothesis that the scheme of pulse-width modulation that is used to adapt the central command to the magnitude of known inertial loads (Gottlieb et al. 1989a) can be generalized to loads with other dynamic characteristics. While finding this to be true, different features of the EMG modulation patterns depended on the type of load being moved. Load type determines features of the joint torques needed to accelerate and decelerate the movements.

Loading reactions

MECHANICS. How do *forces and movements* change when the motor system is required to move compliant loads of different magnitudes or dynamic properties? If the CNS generated load-invariant muscle forces, the resulting motion would be load dependent. To exactly preserve the trajectory, the CNS must properly tailor muscle forces to the characteristics of the load. Angel (1974), Gottlieb (1993b), Gottlieb et al. (1989a), and Smeets et al. (1990) reported that increasing inertial load invariably slows a movement. However, no consistent picture has emerged across different types of loads (Stein et al. 1988). The intrinsically compliant nature of the neuromuscular system, created in part by both the length-tension and force-velocity properties of muscle and by length and velocity sensitive reflexes, makes it likely that natural behavior will lie between force and trajectory invariance. In generating the central command, the CNS will not ignore differences in loads but neither will it completely compensate for them in order to perfectly preserve a trajectory.

Compliant neuromuscular properties have been explicitly exploited by the equilibrium point (EP) hypotheses as the mechanism underlying voluntary control of movement. Both the α version of the EP hypothesis (Bizzi et al. 1982, 1992) and the λ version (Asatryan and Feldman 1965; Feldman 1986) presume that descending control signals that ultimately activate muscles emerge from a *kinematic* plan of the intended movement. This plan can be described in terms of "virtual trajectories" or as shifting "frames of reference" (Feldman and Levin 1995) that, for each individual muscle, specify a time-varying displacement command [$\lambda(t)$] as an input to the proximal end of a neuromuscular "spring" (Houk 1979). That spring is attached at its distal end to the compliant limb/load. Because of the limb/load's viscous and inertial properties, the command "stretches" the neuromuscular spring and thereby creates the forces for movement. These models characterize the compliant properties of the neuromuscular system as predominantly elastic. More recent versions of both models (Feldman et al. 1990; McIntyre and Bizzi 1993; St-Onge et al. 1993) have added a viscous component. The α version has both intrinsic muscle viscosity and velocity-sensitive feedback, whereas the λ version appears to create viscous behavior predominantly by feedback. The application of an external force to perturb a voluntary movement is necessary to distinguish between these two approaches.

A second aim of this study was to examine the hypothesis that the elastic properties of the neuromuscular system are sufficient to account for the forces produced by the muscles to accelerate fast movements. We find that over much of the time in which movement speed is increasing, the muscles' viscous properties are much more important in influencing forces than are its elastic properties.

NEURAL MEDIATION. What are the *mechanisms* that underlie the changes in muscle activation and ultimately changes in EMG, force, and motion that follow from alterations in external loading? The compliant properties of muscle tissue are altered by neural excitation from motoneuron pools that are excited by converging signals of both central and peripheral origin. Central mechanisms can successfully plan to match forces to loads only if the load is known ahead of time. Both central and peripheral mechanisms can also react to the course of the ongoing movement but with different delays.

Load-dependent responses can be studied by applying unexpected disturbances to the moving limb and observing the kinematic and EMG consequences. Unexpected changes in the elastic stiffness of the load produce little change in phasic EMG patterns (Gottlieb 1994; Richardson and Simmons 1985; but see Levin et al. 1992) and lead to errors in final position that require correction by subsequent movement commands. Unexpected changes in the viscous load can alter the phasic EMG patterns and sometimes produce positional errors (Sanes 1986). Large changes in inertial load may lead to changes in phasic EMG patterns (Smeets et al. 1990), but smaller ones may produce little or none (Gottlieb 1994; Latash 1994). Unexpected changes in inertial load have not been found to lead to changes in final position.

A third aim of this study was to test the hypothesis that feedback control by length-sensitive reflexes play a major role in determining the EMG patterns that are observed during fast voluntary elbow flexions. One version of the λ EP hypothesis postulates that length- and velocity-sensitive reflexes are the predominant mechanism for generating EMG patterns (Feldman et al. 1990; Feldman and Levin 1995), whereas another λ version (Latash and Gottlieb 1991a) as well as the α version (McIntyre and Bizzi 1993) give more of a role to central planning of feed-forward control. We find that reflexes play a small and variable role in patterning the EMGs and that role is less stereotyped than classical descriptions of spinal reflex mechanisms would predict.

METHODS

Protocol

Normal human subjects sat with the right shoulder abducted 90°, and they grasped a vertical handle while the forearm rested on a manipulandum that allowed free horizontal rotation about the elbow. They viewed a computer monitor on which a cursor moved according to elbow angle. Zero degrees was defined with the forearm and upper arm forming a right angle and extension defined positive. From a starting position of 30° into extension, identified by a narrow marker on the monitor, subjects performed sets of 36 or 54° flexion movements to a second marker, 3° wide, centered at the desired target position. They were instructed to move as "fast and accurately" as possible when they heard a computer signal (a tone). Asking subjects for both speed and accuracy induces them to make less than maximally fast movements (Fitts 1954). No further instruction was given regarding what to do if errors were made. These methods are described in greater detail in Gottlieb et al. (1989a).

Joint angle and acceleration were transduced and low-pass filtered at 30 Hz. The moment of inertia was estimated by applying a triangular torque pulse to the manipulandum and measuring the resulting acceleration. Typical values were 0.086 $Nm \cdot s^2$/radian (or equivalently $kg \cdot m^2$) for the manipulandum and ~0.095 $Nm \cdot s^2$/radian for the limb. All measured signals were digitized

with 12-bit resolution by a data acquisition computer at a rate of 1,000/s. Joint velocity was computed from the measured angle.

An external load was generated by a torque motor (PMI model JR25MCH), controlled by a servo computer dedicated to simulate a controllable load compliance with specified combinations of elastic, viscous, and inertial properties. The servo computer continuously digitized (at 1,500/s) angle, velocity (from an analog differentiater) and acceleration signals from the manipulandum and a load control voltage (v_l) generated by the data acquisition computer. The controllable compliance was the sum of fixed (M_0, B_0, or K_0) and variable components (M_v, B_v, or K_v) that were controlled by v_l. The ideal motor torque (τ_{motor}) is given by *Eq. 1*, where θ_0 is the initial position of the joint

$$\tau_{motor} = M_0 \frac{d^2\theta}{dt^2} + B_0 \frac{d\theta}{dt} + K_0(\theta - \theta_0) + v_l \left[M_v \frac{d^2\theta}{dt^2} + B_v \frac{d\theta}{dt} + K_v(\theta - \theta_0) \right] \quad (1)$$

For the servo system to be stable, the signals were filtered until satisfactory performance was obtained. Figure 1 illustrates the output of the servo and the controlling kinematic variable in three examples in which a single type of fixed load was simulated. There is a small amount of phase shift between the torque (——) and the angular variable (– – –) due to the filtering. Nevertheless, the servo "felt" like the intended type of load to both the subjects and the experimenters.

The net muscle torque (τ_m) was computed by *Eq. 2* as the sum of the passive inertial torque of the limb/device plus the measured motor torque

$$\tau_m = (M_{manipulandum} + M_{arm}) \frac{d^2\theta}{dt^2} + \tau_{motor} \quad (2)$$

PREDICTABLE LOAD EXPERIMENTS. Fixed load components (M_0, B_0, or K_0) were specified from the servo computer keyboard. The load control voltage was set to zero, and the subject practiced movements until they felt comfortable. A series of 11–15 movements was then recorded. This sequence was repeated with two to four different values of the fixed load component.

UNPREDICTABLE LOAD EXPERIMENTS. For these experiments, one nonzero variable load component (M_v, B_v, or K_v) was also specified. A series of 50–80 movements was performed, 60–90% were against the fixed component with $v_l = 0$. On the other 10–40% of the movements, 100 ms after the tone began to sound, the acquisition computer altered to add or subtract the variable load component. In an experiment, there might have been from two to five different values. It always changed before motion had started and so was imperceptible to the subject until the altered load compliance affected the evolution of movement-dependent external torque. In early experiments, the frequency of the most likely load (the fixed component alone) was 90%, and one variable load component value was used for the other 10%. We concluded that a higher frequency of less likely values did not alter the relevant behaviors of the subjects, and most experiments were performed with between 80 and 60% for the most likely load and two other equally likely variable component values. In the presence of unpredictable load changes, some subjects moved slightly slower with the most likely load than they would have with the same load under predictable conditions. This change in speed was accomplished by a reduction of the intensity of motoneuron pool excitation. This intensity is unaffected by changes in movement distance or known inertial loads (Gottlieb et al. 1989a, 1990a). One of the aims of this study was to see whether intensity was affected by changes in the magnitudes of other types of loads.

A total of 19 subjects, 14 male and 5 female, ranging in age from 17 to 52 yr, performed movements with one or more types of known loads. Of these, 12 also performed movements with unpredictable inertial loads, 8 with unpredictable viscous loads and 5 with unpredictable elastic loads. In the unpredictable load experiments, the value of load for each movement was chosen from a Latin-squares series of integers with the desired load frequency distribution. We used three variations of presentation for unexpected loads, all of which are illustrated in RESULTS. We either presented a single unpredictable magnitude of load that was either larger or smaller than the most likely magnitude, several unpredictable magnitudes that were all larger or smaller than the most likely magnitude, or several unpredictable magnitudes that mixed magnitudes larger and smaller than the most likely. This variation had no effect on subject responses. Subjects were informed before each series whether or not the load might change during the experiment. After obtaining informed consent according to Rush Medical Center approved protocols, data were collected.

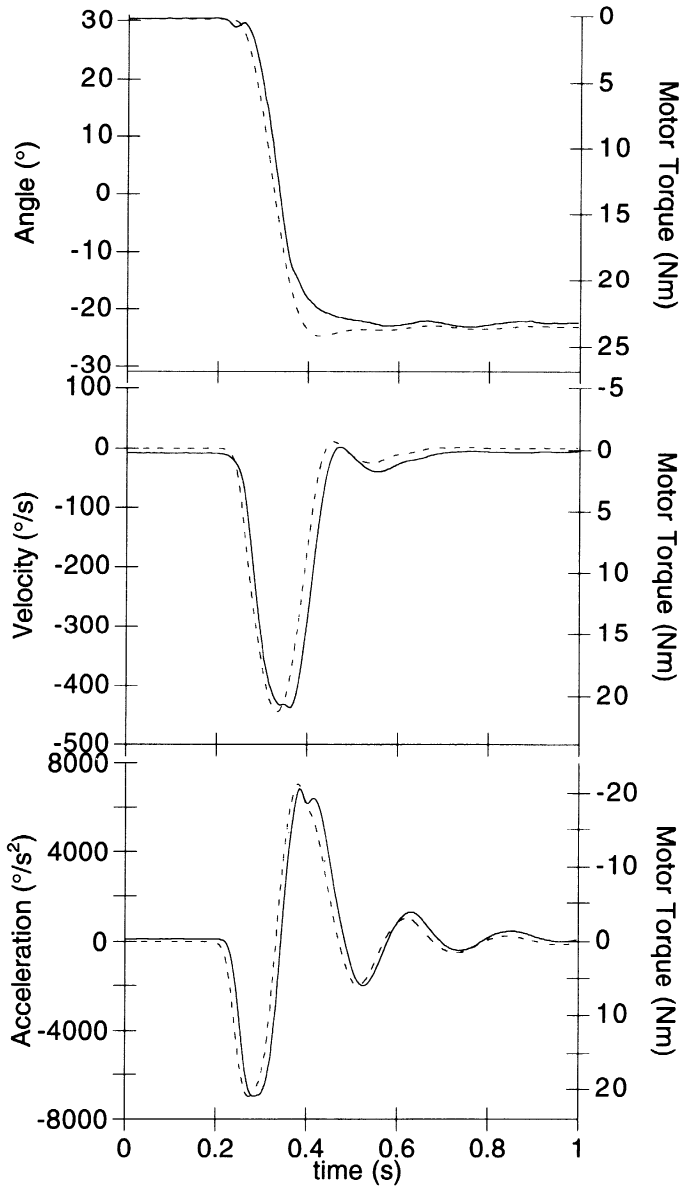

FIG. 1. By adjusting the feedback to the servo computer, the torque motor can be made to simulate different types of mechanical loads. Simulations of an inertial, a viscous, and an elastic load are shown by solid lines in the 3 parts of the figure, along with the kinematic variable (– – –) to which the force is supposed to be linearly proportional. The small lag between the torque and the kinematic variables is a result of the need to low-pass filter the kinematic variables to maintain stability of the servo system.

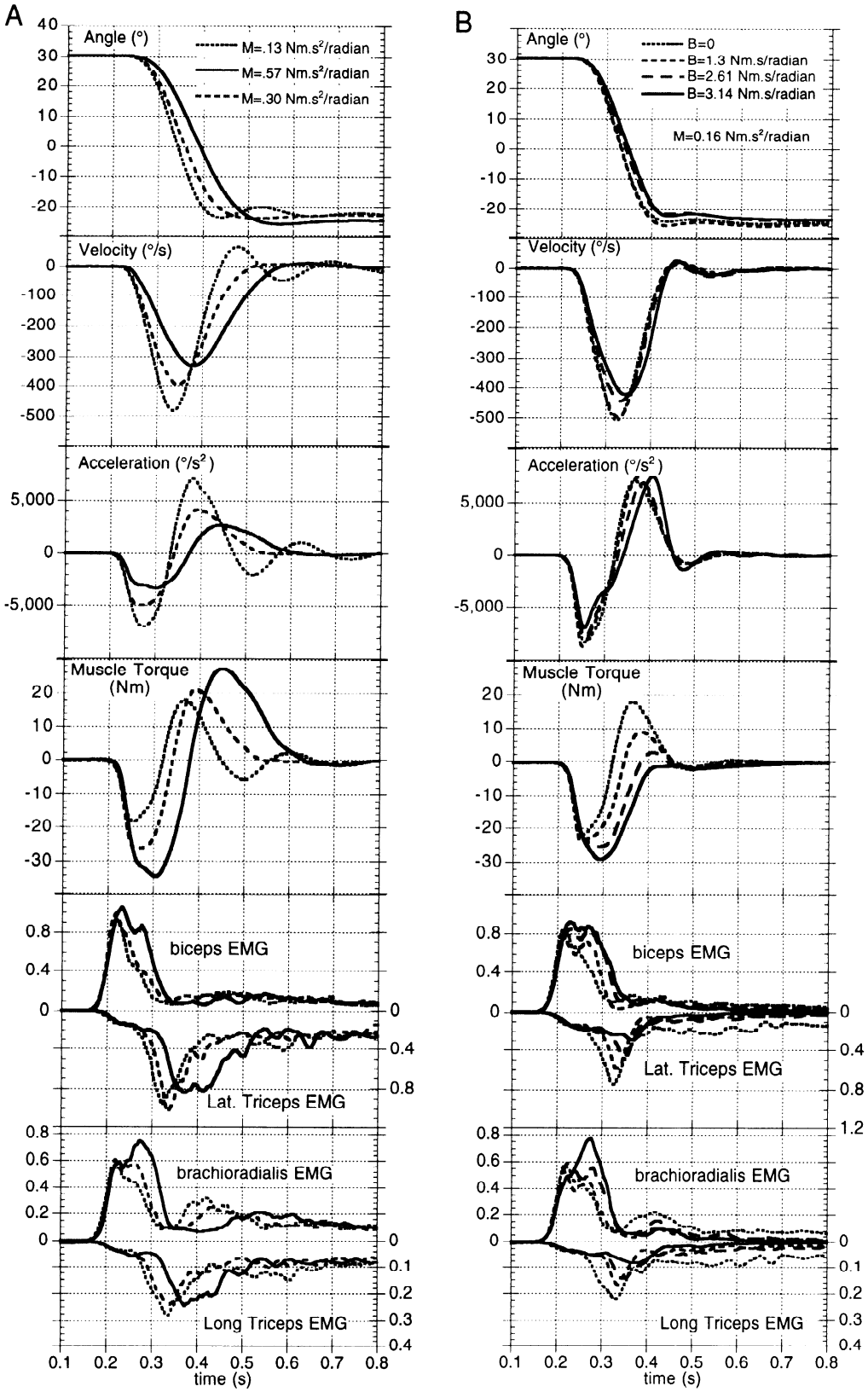

FIG. 2. Practiced movements of 54° performed against 3 types of known loads. Average kinematic, net muscle torque, and electromyographic (EMG) records. Changes in load magnitude produce systematic, quantitative changes in measured variables. Changes in load type produce a qualitative change in patterns of force and muscle activation. *A*: inertial loads: legend shows the net inertia of arm, manipulandum, and motor-controlled component. *B*: viscous loads: 3 different motor-controlled viscous loads added to the fixed inertia of the arm and manipulandum. *C*: elastic loads: 2 different motor-controlled elastic loads added to the fixed inertia. EMGs in this and subsequent figures are rectified and filtered (25 ms rectangular moving average). Flexors (biceps and brachioradialis) increases upward and Extensors (lateral and long heads of triceps) increases downward.

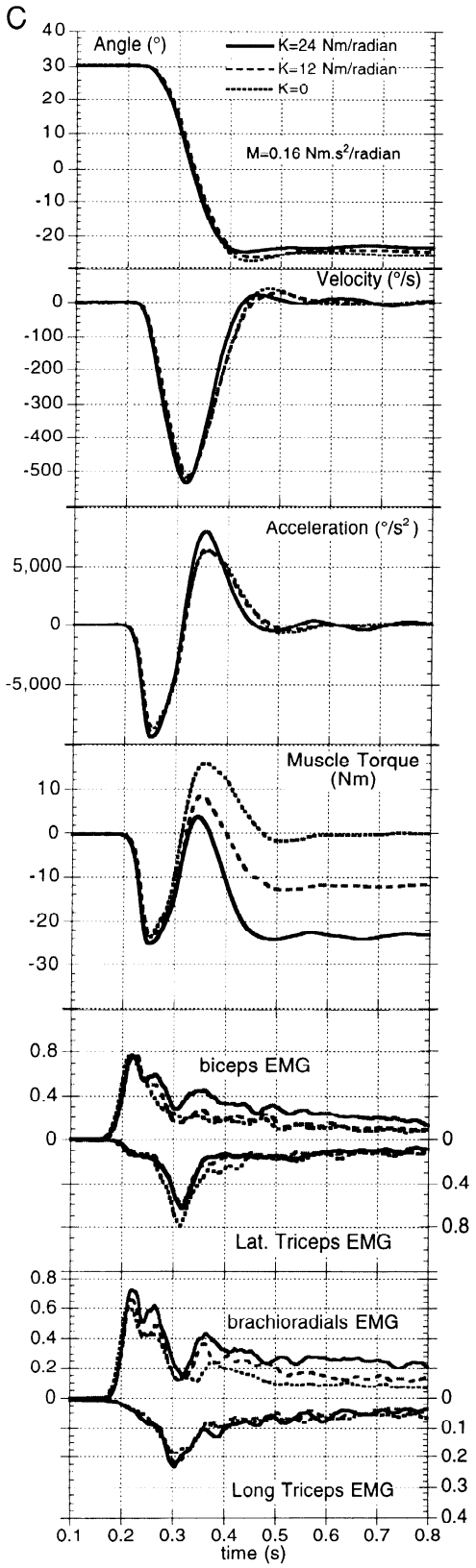

FIG. 2. (*continued*)

Data analysis

We measured EMG signals with pediatric electrocardiogram electrodes (1 cm diam, 2 cm between centers), taped over the bellies of the biceps brachii, brachioradialis, and triceps (lateral and long heads) muscles. The EMG signals were amplified (×2,000) and band-pass filtered (60–500 Hz) before digitization. They were full-wave rectified off-line and when plotted as time series, were smoothed by a 25-ms wide, realizable, rectangular moving average filter. To quantify the EMG signals, we integrated the areas under the rectified EMG bursts. The agonist EMG (Q_{ag}) was integrated from the burst's onset over a fixed interval of 150–300 ms, chosen after reviewing a subject's movements for all load types and identifying an interval long enough to just encompass the longest agonist burst and exclude a second agonist burst if it was present. The agonist burst's onset was defined by visual inspection of the EMG to locate the first rise of the rectified signal above the baseline. Since the agonist muscle was quiescent until the start of the burst, this was an unambiguous event. The antagonist burst (Q_{ant}) was integrated from the onset of the agonist EMG burst until the angular velocity fell to 5% of it maximum.

Visual measures of antagonist "burst" latency could not be consistently made across all load magnitudes and types. This burst onset was defined, not as for the agonist by its first detectable rise in activity (which occurred ~30 ms after the agonist onset), but as the more abrupt rise, which started ~100 ms later. This onset was particularly difficult to determine for large viscous loads. To more consistently evaluate the latency of the antagonist burst across all conditions, we used the following equation

$$C_{ant} = \frac{\int_{t_0}^{MT} t \cdot emg(t) \cdot u(t)\,dt}{\int_{t_0}^{MT} emg(t) \cdot u(t)\,dt} \qquad (3a)$$

$$u(t) = 1 \quad \text{if } emg(t) \geq K\, emg_{max}$$

$$u(t) = 0 \quad \text{if } emg(t) < K\, emg_{max} \qquad (3b)$$

The quantity C_{ant} has units of time and if $K = 0$, it is identical to the location of the centroid or "center of mass" of the EMG signal. For inertial or elastic loads, we set K to zero. We verified with inertial loads, where visual latency measurements were easily made, that there was a very high linear correlation between them and C_{ant}. For viscous loads where it was often not possible to visually identify a latency for the antagonist burst with confidence, *Eq. 3a* with $K = 0$ usually showed almost no dependence of C_{ant} on load. The reason for this can be explained by reference to Figs. 2*B* or 6*A* where visual inspection suggests that the antagonist bursts are delayed by larger viscous loads. Visual inspection defines our judgment of "truth" in a qualitative but not a quantitative sense. The centroid of these bursts is not delayed because as the burst moves to the right it diminishes in size and the two effects cancel each other in *Eq. 3a*. If $K = 0.75$, lower level activity is ignored, and *Eq. 3a* resolves the location of the burst. This algorithm is similar to locating the peak of the EMG burst but is less sensitive to the details of the EMG waveform.

Kinematic performance was evaluated by measuring movement time (MT) and peak movement velocity. Movement time was defined as the interval from 1% of peak acceleration to the fall of the velocity below 5% of its peak. To evaluate the force production of the agonist muscle we used impulse, defined by the integral of *Eq. 2* from acceleration onset (1% of peak acceleration) to the end of MT or to the first reversal of torque sign, which ever occurred first. This computation is only an approximation because it underestimates true agonist muscle force because it is based on net torque, which is reduced by antagonist contraction. We did not quantitatively evaluate the force output of the antagonist muscle.

To make quantitative comparisons between movement parameters for loads of different sizes, we chose the largest and smallest loads of each type for which we had movements with the same two loads under both predictable and unpredictable loading conditions. For five measures (peak velocity, impulse, Q_{ag}, Q_{ant}, and C_{ant}) we computed the ratio of the responses of the larger to the

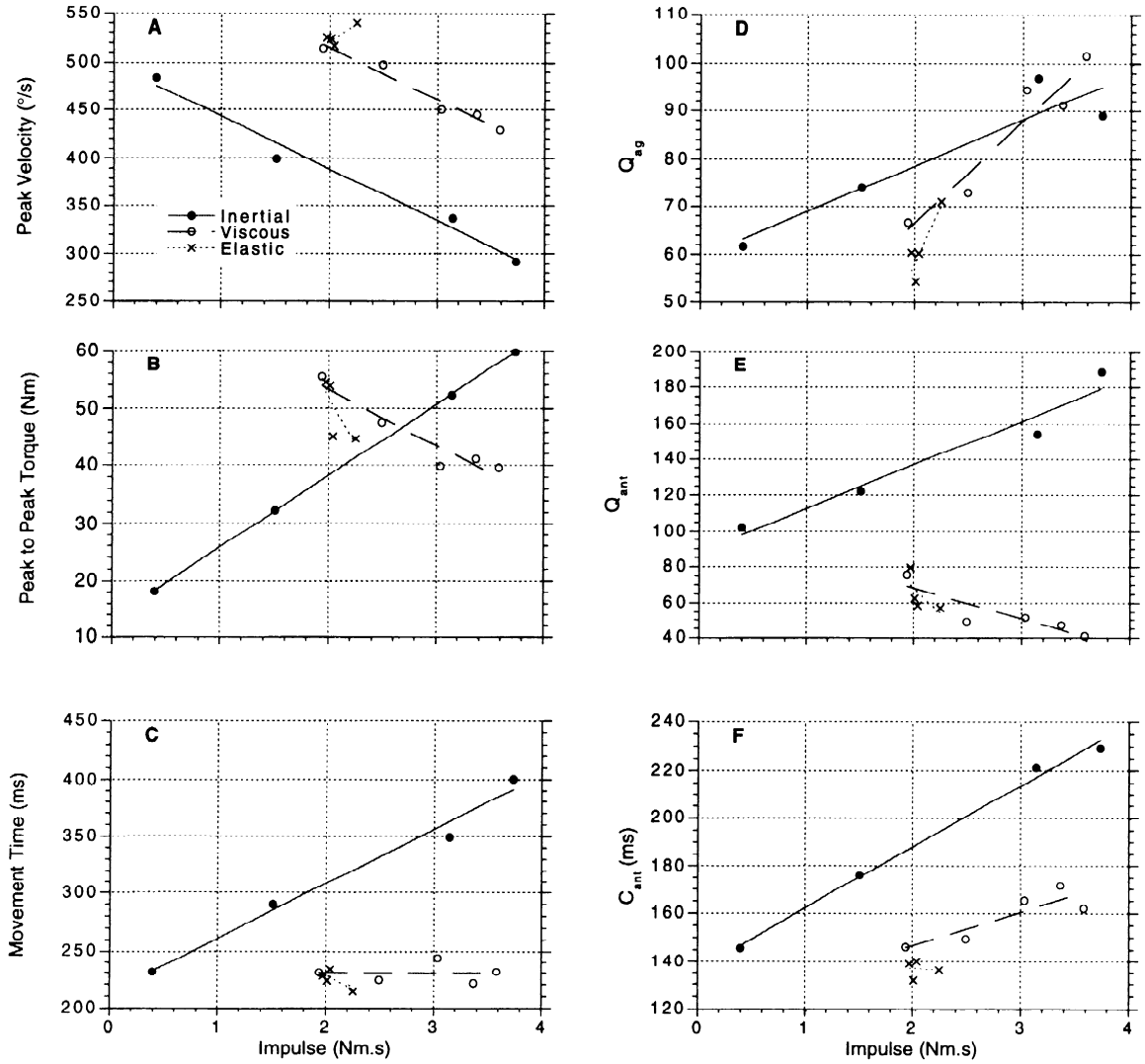

FIG. 3. Effects of different load magnitude and type are illustrated. For inertial and viscous loads, an increase in load magnitude lead to an increase in impulse, and this is plotted on the abscissa. Increasing the elastic load had only small effects on the variables in this figure. *A*: inertial and viscous loads reduce the peak movement velocity. The plotted linear regression lines both have $r > 0.98$. *B*: peak-to-peak torque is increased only by inertial loads ($r > 0.99$). *C*: only inertial loads increased movement time ($r > 0.99$). *D*: areas of the agonist (biceps) EMG bursts increased with the size of the inertial ($r = 0.96$) and viscous ($r = 0.93$) loads. *E*: areas of the antagonist (triceps) EMG bursts increased with the size of the inertial load ($r = 0.97$) and decreased with the size of the viscous load ($r = 0.88$). This decreasing trend can also be seen for elastic loads but is weaker ($r = 0.67$). *F*: the centroid of the antagonist burst is delayed by increases in inertial ($r = 0.98$) or viscous ($r = 0.89$) loads. For inertial loads, $K = 0$ in *Eq. 3*, whereas for viscous loads, $K = 0.75$.

smaller load as a percentage. If a response was increased by a larger load, we obtained a positive value. Each subject provided us with two ratios for a load type, one for the predictable loads and one for the unpredictable loads. Plotting predictable versus unpredictable ratios showed both the effects of load increases and the effects of predictability. This analysis, shown in Figs. 10 and 11, summarizes the data from 11 subjects in 27 experiments (13 with inertial loads, 8 with viscous and 6 with elastic).

RESULTS

Known loads of different amplitudes

Figures 2 and 3 show movement performed in a single session against predictable inertial, viscous, and elastic loads by a male of average strength. The *top three graphs* in Fig. 2 show the average angle, velocity, and acceleration records. Movements shown in Fig. 2*A* are significantly slowed as the moment of inertia increases. This reproduces previously reported findings (Gottlieb et al. 1989a). Predictable viscous loads have a much smaller effect as shown in Fig. 2*B* and elastic loads a negligible effect as shown in Fig. 2*C*. This is summarized in Fig. 3*A*, where we have plotted peak velocity versus impulse. Although load is not explicitly specified in the figure (because inertia, viscosity, and elasticity have different units), increases in the magnitude of a viscous or inertial load always resulted in increased muscle-generated impulse. The elastic loads used in this experiment did not usually increase the impulse because, as shown in the fourth panel of Fig. 2*C*, muscle torque reversed in sign after only a short part of the angular trajectory was traversed. These elastic loads were insufficient to affect torque significantly over this short distance where inertial effects dominated. Figure 3*B* illustrates the different effects of load type on peak-to-peak torque. Only inertial loads cause impulse to be

positively correlated with peak-to-peak torque. Viscous loads reduce peak decelerating torque more rapidly than they increase peak accelerating torque. Elastic loads have little effect on peak accelerating torque and decrease decelerating torque. Fewer movements are illustrated in Fig. 2 than are summarized in Fig. 3 to avoid overcrowding the time series graphs. The movements illustrated in Figs. 2*B* for viscous loads also demonstrate the unusual property of having changes in peak velocity without corresponding changes in movement time (Fig. 3*C*). Inspection of the trajectory in Fig. 2 shows that there are changes in the symmetry of the viscous loaded movements that caused this seeming paradox as a consequence of our choice of defining the end of the movement at 5% of peak velocity.

The *fourth row* of Fig. 2 shows net muscle torque (i.e., the algebraic sum of overlapping flexing agonist and extending antagonist contractile torques), calculated with the use of Eq. 2. Although impulse varied with the type and size of the load, the initial rate of rise of muscle torque was independent of both. This was to be expected from our earlier finding that the rate of rise is independent of movement distance (Gottlieb et al. 1990b) or inertial load (Gottlieb et al. 1989a). Early in the movement trajectory, displacement and velocity have not yet changed, and only acceleration-dependent torques initially resist the contracting muscle so this invariance is preserved across the other types of external loads.

The *fifth* and *sixth rows* of Fig. 2 show the associated EMG patterns that are similar in synergistic muscles. Agonist EMG bursts rise at a rate that is independent of the type or magnitude of the load. Visual inspection of the flexor EMG burst suggests a trend of increasing burst duration with load magnitude, but the strength of this effect is dependent on the load type. Inspection of the EMG patterns does not provide a clear definition of burst duration that will be adequate across all load types. Although we have not made a direct measurement of duration, because the rates of burst rise are load invariant, increases in duration will lead to increases in the areas of the agonist EMG bursts. These increases are summarized in Fig. 3*D* for the biceps. We found no qualitative differences between the behaviors of synergistic muscles and only present EMG data from a single agonist/antagonist pair in the remaining figures.

As the size of the load increased, the area of the antagonist bursts increased for inertial loads, decreased with viscous loads, and decreased slightly with elastic loads as summarized in Fig. 3*E*. Increasing inertial and viscous loads delayed the antagonist bursts as shown in Fig. 3*F*. Elastic loads had little effect (see also Fig. 2 in Gottlieb 1994).

Unpredictable changes in inertial load

An unpredicted change in inertial load alters the movement, the torque, and the EMG patterns. This is illustrated in Fig. 4 by three series of movements with two inertial loads. In Fig. 4*A*, both loads were known in advance of each movement, and the trials for each were presented in blocks. Even though the subject activated the muscles more strongly and produced larger torques for the larger load, he did not prevent slowing. These movements, qualitatively the same as those in Fig. 2*A*, can be compared with those shown in Fig. 4, *B* and *C*, made with the same two inertial loads, randomly ordered with 80% with one size load and 20% with the other. In Fig. 4*B*, 10 heavier inertial loads were interspersed with 40 of the lighter inertia. Movements of the more frequent, lighter load (the most frequent load is marked with ● symbols in this and succeeding figures) were almost indistinguishable from those of the same load in Fig. 4*A* in terms of kinematics and muscle torque. Movements with the unexpectedly larger load in Fig. 4*B* failed to reach the same peak acceleration of Fig. 4*A* and evolved more slowly. This lower acceleration of the heavy load in Fig. 4*B* is a direct consequence of the fact that, although muscle torque with the unexpectedly heavier load in Fig. 4*B* is greater than with the light load, it is not as great as the torque in Fig. 4*A* for the expected heavy load. In Fig. 4*A* the differences in muscle torque between the two movements are associated with similar differences in the sizes of the EMG bursts. In Fig. 4*B* the two agonist EMG patterns are indistinguishable (and the same as the burst in Fig. 4*A*) for ~200 ms, even though there is a difference in muscle torque in this interval.

In Fig. 4*C*, where the relative frequency of the two loads is reversed from that in Fig. 4*B*, movements, torques, and EMG patterns against the heavier, more frequent load (●) were very much like those for the heavier load in Fig. 4*A*. Movements with the less frequent, light load were faster than those with the same light load in Fig. 4*A*, and peak torques were greater. Presentation of the less frequent, larger load (Fig. 4*B*) decreased the early antagonist EMG (around $t = 0.4$) and increased it later (around $t = 0.7$). Presentation of the less frequent, smaller load (Fig. 4*C*) produced an earlier antagonist burst that was considerably larger than for the same load when known (Fig. 4*A*). However, its area is smaller than that of the larger load in Fig. 4*C* because of the difference in integration (movement) times (see also Fig. 2*A*).

The effects of changing load are made more evident in Fig. 5 by the differences in the kinematic, torque, and EMG patterns between the movements of the two loads. The time scale has been expanded, and the plot begins from $t = 0.2$ s, the onset of the agonist burst. The two angular trajectories diverge at about $t = 0.3$ s. Their difference is greatest (25–40°) near $t = 0.5$ s. The peak difference is over 10° smaller for the predictable load condition. Divergence of the angular trajectories is preceded by separation of the velocity and torque trajectories. The velocity differences have been multiplied by the scale factor β shown in the *top right* of the figures. By a proper choice of β,[1] the scaled velocity difference is identical to the torque difference for ~100 ms, after which it falls below the torque.

The agonist flexor muscles become active at $t = 0.2$ s in the figure. The difference between the EMG patterns under

[1] The value of β was chosen by visually matching the rising slope of the velocity to the rising slope of the torque. In all cases, when we did this, there was a clear divergence between the 2 curves between 300 and 400 ms after the onset of the agonist burst, and in every case, the measured torque exceeded the scaled velocity in the following time interval. We also tried to do this with the angle trace but could not. For example, if the angle trace in Fig. 4*B* were multiplied by 2 [i.e., postulating a stiffness of 2 Nm/deg (115 Nm/radian)], at $t = 0.3$ the postulated elastic torque would only be 1.8 Nm, whereas the measured torque would be 3.05 Nm. The 2 curves would cross at $t = 0.332$, and the elastic torque would rise to a peak of ~69 Nm at $t = 0.496$ s. To argue that the difference in torques between the two movements is the result of muscle elastic properties requires extremely high values of stiffness that have never been measured in human experiments and also requires that there be a rapid yielding of this stiffness after ~3° of joint rotation.

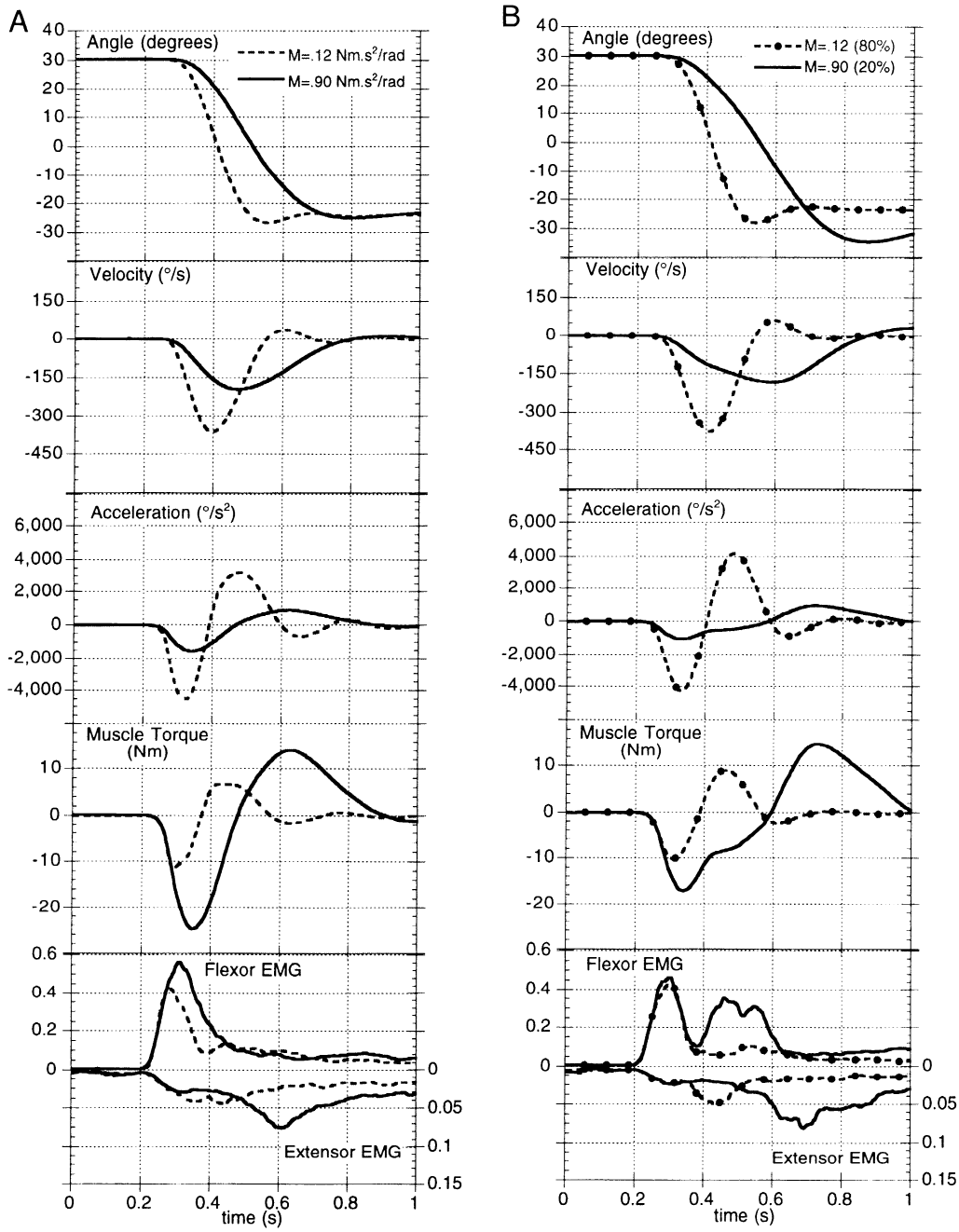

FIG. 4. Movements of 54° performed against 2 different inertial loads (0.12 and 0.9 Nm·s²/radian). Average kinematic, net muscle torque, and EMG records. *A*: both loads known. *B*: lighter load presented 80% with heavier load interspersed 20% in an unpredictable sequence. The average is of 10 of the 40 movements with the lighter load (randomly selected) and of all 10 of the movements with the heavier load. *C*: heavier load presented 80%.

the two loading conditions is zero for ~60 ms in Fig. 5*A* and longer in the other parts. This demonstrates that the flexor EMG bursts for the two loads are almost identical for ≥60 ms after agonist onset in all three cases. When the EMGs under the different conditions become different from each other, the agonist burst for the larger load condition usually exceeds that of the smaller. When the loads were unpredictable (Fig. 5, *B* and *C*), differences between the agonist EMG patterns did not appear until >120 ms later than under the predictable load conditions in Fig. 5*A*.

The antagonist burst is delayed with a heavier load, and the antagonist EMG in the 200-ms interval, starting 100–150 ms after agonist onset is smaller than it was with a smaller load. With interspersed, unpredictable loads, separation of the EMG patterns is observed later (by ~50 ms) than in the known load condition. The delay of the antagonist leads to a relative increase in the antagonist burst for the larger load, 300 ms after agonist onset. There is usually a net increase in the area of the antagonist burst with a larger inertial load (see Figs. 3 and 11).

Comparing the mechanical and EMG parts of Fig. 5 shows that with predictable loads, separation of the two torques is simultaneous or slightly after the separation of the agonist bursts. With unpredictable loads, torque records under the

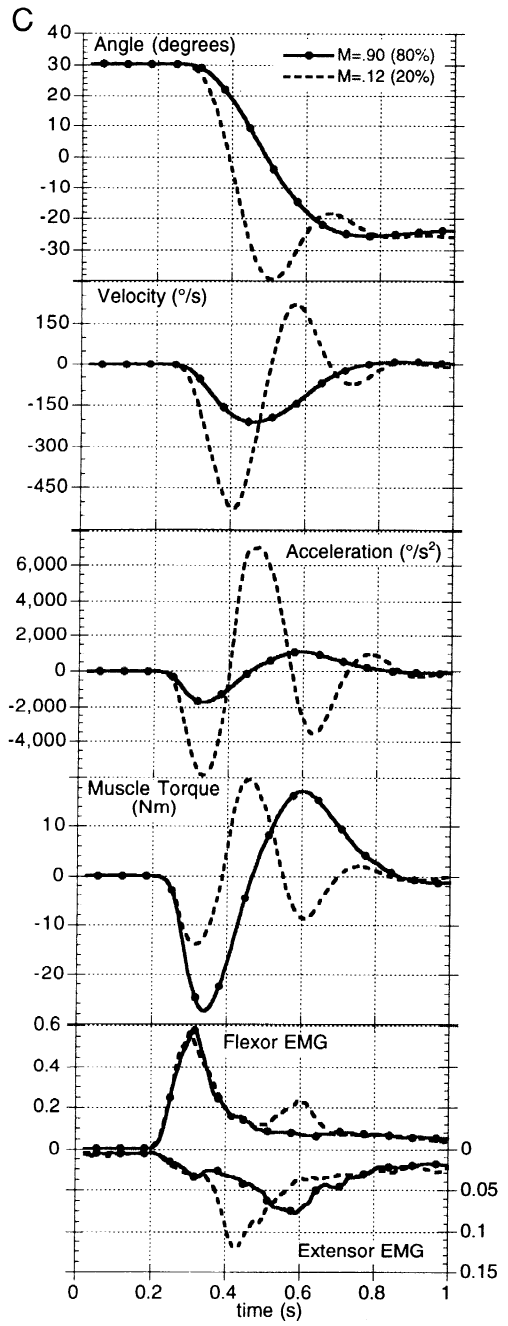

FIG. 4. (*continued*)

two loads diverge before the EMGs differ from each other. In the predictable case, differences in torque can always be associated with (and attributable to) differences in muscle contraction, but this is not possible with unpredictable loads until late in the movement.

This subject is representative of all subjects in the way predictable changes in inertial load affected kinematic and kinetic patterns. The subject's antagonist EMG responses to unpredictable changes in load are also representative. For the agonist EMG he shows one of the weaker responses to unpredictable changes in load (similar to the subject in Fig. 3 of Gottlieb 1994). After illustrating the effects of the other two load types with representative individuals, we will present data to summarize the population responses.

Unpredictable changes in viscous load

Figure 6 shows that predictable viscous loads slowed subjects to only a modest extent. This is illustrated in Fig. 6*A* by three series of movements against different levels of viscous load (0, 1.43, and 2.87 Nm·s/radian). This behavior is similar to that illustrated in Fig. 2*B*. With a load of 1.43 Nm·s/radian, this subject stopped beyond the target (by ~4° for a 54° movement), but there were no consistent positional errors with either predictable or unpredictable viscous or inertial loads nor with predictable elastic loads.

The movements with unpredictable loading were not identical to those under predictable conditions for the same load. This can be seen by comparison of Fig. 6*A* with *B* and *C*. Of interest here are the preserved similarities. Initial accelerations, torques, and agonist EMG bursts all increased at load-independent rates. Larger loads increased the torque and the areas of the agonist bursts while decreasing those of the antagonist bursts. A clear difference between predictable and unpredictable conditions is that only with predictable loads was the antagonist burst delayed by larger viscosities.

Subtraction of the movement against zero added viscosity from that with the largest value is shown in Fig. 7. Just as with the inertial loads shown in Fig. 5, divergence between angular trajectories with two different viscous loads was smallest when the loads were predictable. Angular divergence was preceded by divergence of the velocities that by proper scaling was identical to the torque divergence for the 1st 100 ms, after which it fell below the torque.

The EMGs of both agonist and antagonist muscles diverged 100 ms after agonist onset when the load was predictable and 175 ms after when load was unpredictable. Added viscosity increased the agonist and decreased the antagonist burst.

Figure 7 shows that divergence of the torques with different predictable loads to be simultaneous or slightly after the divergence of the agonist bursts. With different unpredictable loads, the torque divergence precedes the EMG divergence by >50 ms. In most respects this figure resembles Fig. 5 for the inertial loading condition.

Unpredictable changes in elastic load

Predictable elastic loads had only small effects on the trajectory of 36° movements against three different spring constants as illustrated by Fig. 8*A*. Although the subject of Fig. 8 was weaker than the subject in Fig. 2*C* and produced significantly smaller accelerating torques (smaller even than the static torques), the effects of elastic loads are similar in the two subjects. The trajectory was initially load invariant, and we found no consistent relation between the size of a predictable elastic load and any kinematic variable. Muscle torques rose at load-independent rates, and the rise was prolonged to a higher peak with the largest elastic load. The area of the agonist burst increased with load, but this figure does not show clear changes in the antagonist EMG.

Movements against unpredictable elastic loads all started out with similar trajectories but ended short of the target if the spring was stiffer than the most likely value and overshot the target if the spring was softer. The subjects made corrective movements that can be seen as load-dependent changes in agonist EMG activity that can be seen in Fig. 8*B* as starting at about $t = 0.45$ s.

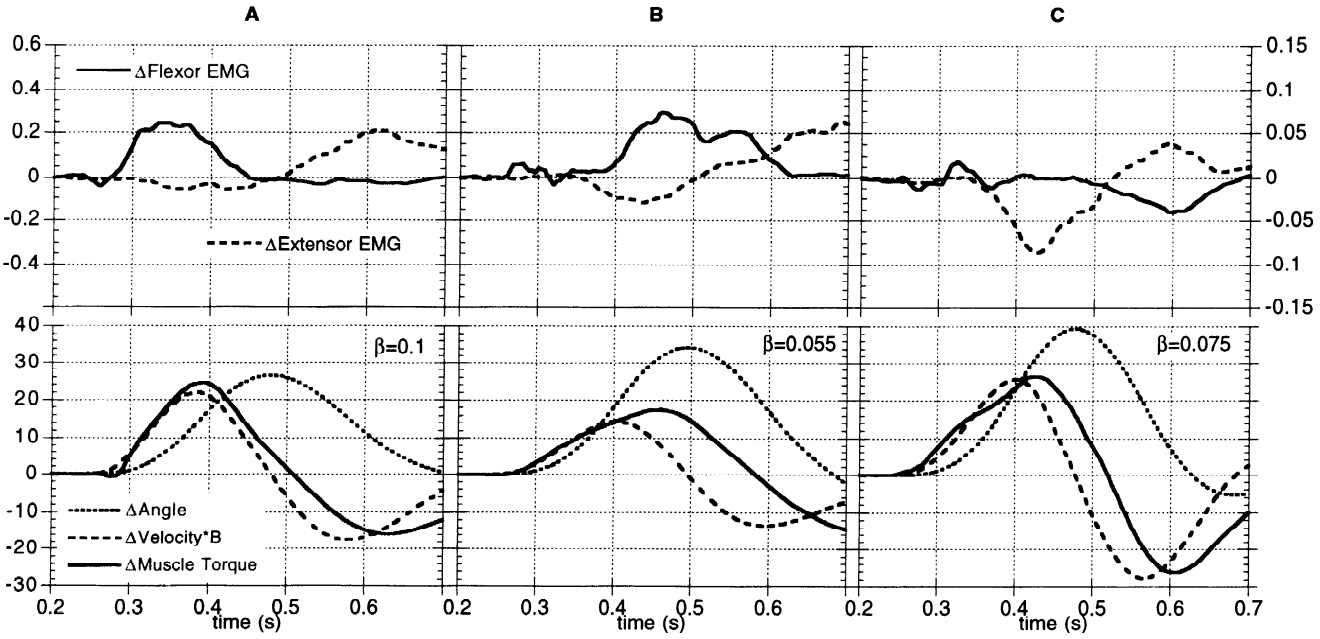

FIG. 5. Differences between movements with the 2 different inertial loads shown in Fig. 3. *Top*: differences in flexor and extensor EMG records. Time axis has been expanded and starts at the onset of the agonist burst. *Bottom*: differences in angle (deg), muscle torque (Nm), and velocity. Velocity trace has been multiplied by the value β in the *top right corners* of the figures.

Trajectory differences between the larger (K = 34.4 Nm/radian) and the intermediate (K = 22.9 Nm/radian) elastic loads are shown in Fig. 9 for predictable (A) and unpredictable (B) conditions. Angular divergence was again smallest in the case of predictable loads. Small changes in EMG and torque both appear within 100 ms of agonist onset under predictable load conditions and are much smaller and later with unpredictable load changes. Comparing the mechanical and EMG parts of Fig. 9 again shows that divergence of the torques is simultaneous or slightly after the divergence of the agonist bursts with predictable loads. With randomized loads, the torque separation precedes the EMG separation.

The preceding data show representative individuals moving the three types of loads under predictable and unpredictable conditions. Although the loads differ qualitatively, they have some similar mechanical consequences that are summarized by Fig. 10. Each data point in Fig. 10 represents the percentage by which an increase in load reduced peak velocity (A) or increased impulse (B) in one experiment. The size of the reduction depends, of course, on the magnitudes of the two loads. The reduction in peak velocity produced by an unpredictable increase in load is plotted on the ordinate. The abscissa shows that velocity also was reduced with known increases (identical in magnitude to those used for the ordinate) in viscous (squares) or inertial (circles) load. Known increases in elastic (triangles) load had a small, variable effect.[2] The loose clusters of the three symbols show that inertial loads produced the largest change and elastic loads the smallest. The intermingling of filled symbols (which denote that the smaller load was the most frequent) with the open symbols (the larger load was the most frequent) produced effects comparable in magnitude.

If the effect of a load change were related only to its magnitude, the data points would be distributed on both sides of the dashed ("indifference") line. The solid line, the linear regression curve through the pooled data set, lies below the indifference line as do all but two points showing that an unpredicted change in load had a greater effect on peak velocity than did a change of equal magnitude but known in advance. The wide range in the magnitudes of these effects reflects differences in the sizes of the two loads, strength differences between subjects, and differences in the size of the movements.

Larger inertial or viscous loads were associated with the generation of greater impulse whether the change was predictable or not so the indifference line lies above the regression line. This is shown in Fig. 10B. We used Student's t-test to compare impulse for larger loads to that generated against smaller loads. All subjects increased impulse with load, although for some elastic loads, the increases did not always reach statistical significance. For impulse, as with peak velocity, inertia had a larger effect than did viscosity, which, in turn, had a larger effect than did elasticity.[3] That observation is consistent with the load-induced changes in velocity.

Figure 11 shows some features of the myoelectrical re-

[2] Subjects sometimes produced larger peak velocities with larger, predictable elastic loads, although only one of those increases in velocity (the largest in the figure) is significantly different from zero ($P < 0.001$). This occurred only with predictable changes in elastic load, not with other types or with any type of unpredictable change. We interpret this increase as "unintentional" and unknowing overcompensation for the predicted increase in load. It is a failure of the subject to use the same effort from one experimental series to the next.

[3] The relative size of kinematic effects for a fixed load type obviously depends on the relative magnitude of the load. In comparing the effects of different load types, there is no direct equivalence between inertial, viscous, and elastic loads that allows a direct comparison of relative effects. As an operational matter we were limited in the peak torque we could generate (~25 Nm) by the amount of current we could provide to the motor. Under that constraint, inertial loads were more effective than viscous loads, which were, in turn, more effective than elastic loads in slowing the movements.

sponses. In Fig. 11*A*, larger loads usually increased the agonist burst area. However, in some subjects there was no significant change in the agonist burst. Data points lie scattered on both sides of the indifference line. Figure 11*B* shows the effects of load on the area of the antagonist burst. Inertial and viscous loads produced EMG changes in the antagonist that significantly differed from each other, not merely in magnitude but in direction. Although increase in either type of load slows the movement and increases impulse (Fig. 10), an increase in an inertial load increases the area of the antagonist burst, whereas an increase in a viscous load decreases it. Viscous effects are smaller than inertial, and some subjects demonstrated statistically significant (*t*-test, $P < 0.01$) decreases in the antagonist burst with unpredictable changes but not with predictable changes.

Figure 11*C* shows the influence of loading on the effective latency at which the antagonist burst acts. Inertial loads alter the effective latency of the antagonist burst by an amount proportional to the size of the load. Unpredictable increases in inertial load delay the antagonist burst by an amount similar to the delay with a known load. Unpredictable decreases in inertial load shorten the latency of the antagonist burst, but the effect is smaller. Unpredictable changes in viscous or elastic loads usually had little effect on antagonist latency as also illustrated by the two subjects in Figs. 6 and 8.

DISCUSSION

Voluntary movements are successfully accomplished under diverse conditions and degrees of knowledge about the intended task. How the nervous system copes with incomplete knowledge about the task has long been a question of interest. One proposed approach is to rely on feedback mechanisms to adjust muscle forces to the encountered load. Merton (1953) considered a servomechanism, based on the segmental stretch reflex, as a way of controlling muscle length, but the low gain of the reflex arc and problems with time delays showed this to be too simple an hypothesis. Feldman and his colleagues (Adamovitch and Feldman 1984; Asatryan and Feldman 1965; Berkinblit et al. 1986; Feldman et al. 1990; Feldman and Levin 1995; St-Onge et al. 1993) have pursued progressively more complex extensions of this notion, based on less well specified anatomic structures. As we will discuss below, we do not believe that reflex mechanisms make a major contribution to the activation patterns of the muscles when generating fast voluntary movements. The data indicate that they do play some role, however, and therefore disagreements may arise that are matters of emphasis and perspective. In the following, we will consider the behaviors that we think an adequate theory of motor control must account for from our own perspective.

The first task of any theory of voluntary movement is to account for the kinematic and EMG patterns of movements under the simplest of conditions. Such conditions would include full and complete knowledge of the movement task and simple performance criteria. Repeated, single-joint movements over different distances, at different speeds, or with different loads have been long used as the experimental paradigm for this. However, it has not been possible for human experiments of this type to distinguish between control mechanisms based on reflexes from those that exclude them completely. One way to make such distinctions is to apply some form of unpredictable, external perturbation to the movement. This deprives the subject of complete knowledge of the task and allows us to see whether, when, or how reflex mechanisms, or any other mechanisms based on feedback signals from the actual movement are incorporated into the muscles' activation patterns. It is important, however, that the perturbations not cause the subject to alter the central command itself. Such alteration may be less likely if the perturbation is applied smoothly during the movement (such as when it is dependent on the movement's kinematics) rather than delivered abruptly or in advance of movement as has often been done with pulse and step perturbations. In the sections below we will first consider movements under conditions of full knowledge and then proceed to those of incomplete knowledge.

Moving known loads

The "input" to the motoneuron pool is the net excitation converging from all sources, central and peripheral. Whether this signal, or components of it, can be given a physical interpretation such as a "virtual trajectory" or a "nominal isometric force" for example is an important question to which we will return. This input, filtered by the motoneuron pools, is transmitted to the muscles and drives both the contractile processes that lead to force and motion and the bioelectrical processes that produce the EMG.

The data show that when subjects are required to move a larger load, they produce greater impulse and their EMG bursts are prolonged and have greater area. The EMG bursts rise at load-independent rates, however, and there are often only slight changes in their peak amplitudes. All load types prolong the agonist bursts but differ significantly in the strength of their influence, inertial loads having the largest and elastic loads the smallest effects for the load magnitudes used here. The relation between agonist EMG and torque and load also applies to the antagonist muscles with the recognition that different types of loads require and receive very different decelerating torques. Larger inertial loads require larger decelerating torques. Larger viscous loads, and to a lesser extent elastic loads, require smaller decelerating torques from the muscles. The effects of load on the antagonist EMG parallel its effects on the deceleration torque.

Although increases in torque production and EMG activity due to increased loading are associated with reduced movement speed in these experiments, increases in torque and EMG activity will be associated with higher movement speeds if the load is constant and subjects deliberately increase speed or movement distance (Corcos et al. 1989; Gottlieb et al. 1989a). Larger EMG bursts always increased the impulse produced by the muscle, regardless of the task. In contrast, only under constant load conditions are EMG and speed positively correlated. For this reason, we suggested that muscle activation patterns (and therefore the EMG patterns) are planned in terms of the dynamic muscle torques expected to move the net load rather than in terms of features of the angular trajectories that result from those torques (Gottlieb et al. 1995b).

It is important to note that even when subjects knew that the load had changed, they did not alter the initial rates of

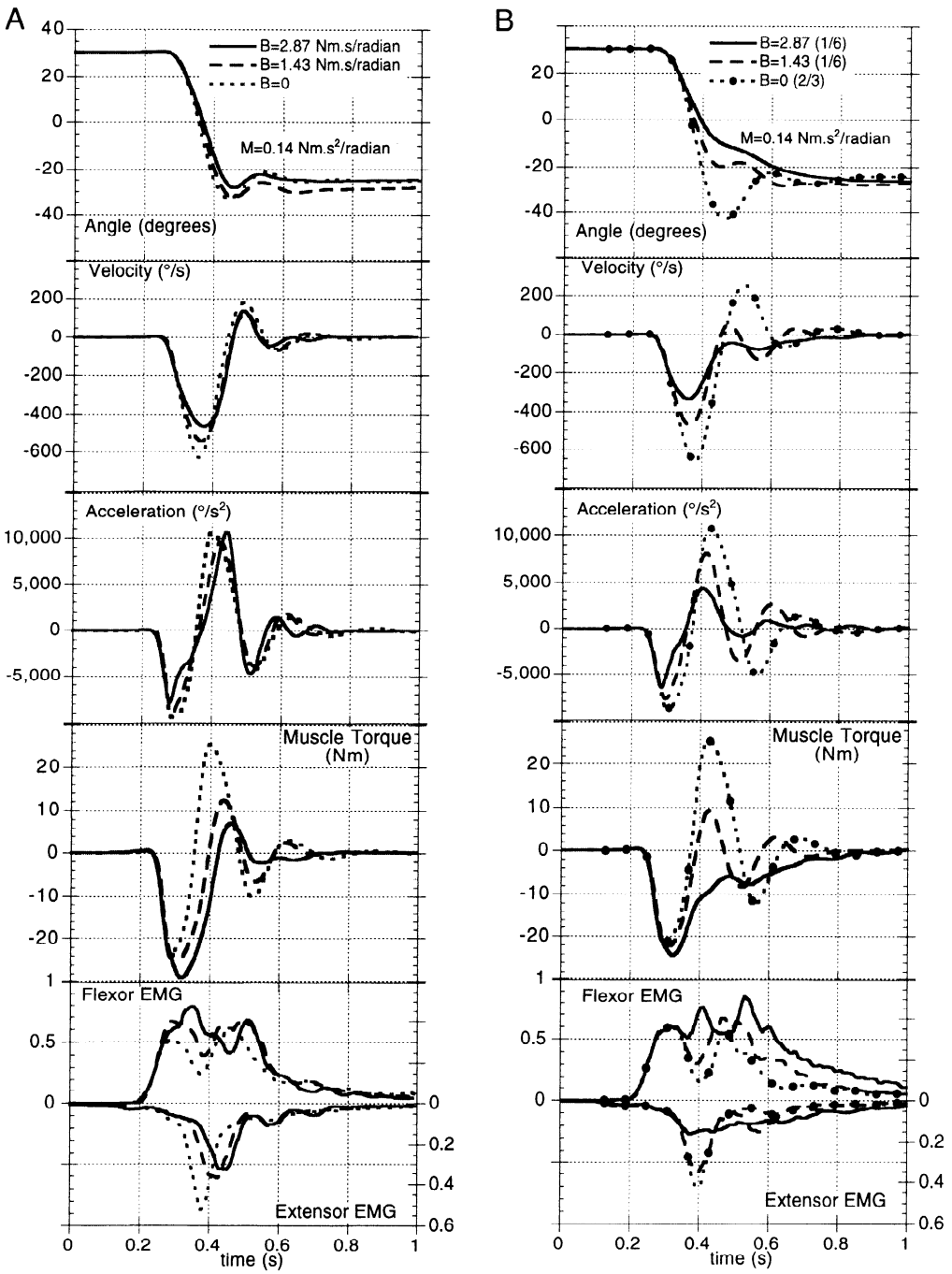

FIG. 6. Movements of 54° performed against 3 different load viscosities (0, 1.4, and 2.87 Nm·s/radian). Average kinematic, net muscle torque, and EMG records. *A*: all loads known. *B*: zero viscosity encountered 67% of the time with the 2 viscous loads interspersed 16% each in an unpredictable sequence. The average is of 10 of the 40 movements with the lighter load (randomly selected) and of all 10 of the movements with the heavier loads. *C*: largest viscosity (2.87 Nm·s/radian) presented 67%.

rise of the EMG bursts or of the muscle torque. By assuming that both EMG and torque represent measurable expressions of motoneuron pool excitation, low-pass filtered by the neuromuscular apparatus (Gottlieb 1993a; Gottlieb et al. 1989a,b), the load-independent rise is consistent with a model for centrally originating components of motoneuron excitation as constant height, rectangular pulses that produce agonist/antagonist bursts. To accommodate known changes in load, the agonist and antagonist pulses are modulated in duration and latency to provide appropriate accelerating and decelerating forces. The use of uniform pulse heights is an adequate strategy for the central command (Gottlieb et al. 1995a), not the result of rate-limiting physical or physiological constraints such as saturation of a neuron pool (Gottlieb 1993a; Gottlieb et al. 1990a). To accommodate changes in static force required to maintain the desired equilibrium position, the heights of the steps that outlast the pulses are adjusted.

This model accounts for load-independent rates of rise of the EMG bursts, and for areas that scale with load. However,

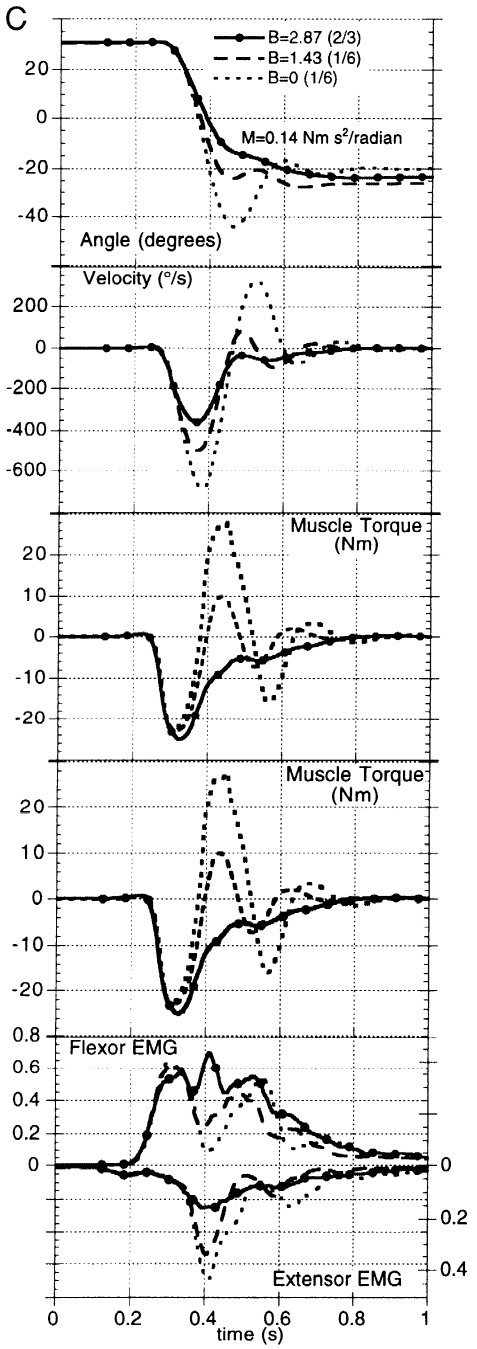

FIG. 6. (*continued*)

not all features of the EMG patterns can be explained in this way. The peaks of the EMG bursts often do not vary with load,[4] which may sometimes be due to the fact that strong, phasic contractions are recruiting a large fraction of the motoneuron pool. Peak EMG values that do not change with load can also be seen at lower levels of contraction with movements that are slower. Low-pass filtered, rectangular excitation pulses and steps are simplifications that fit some aspects of the data in a qualitative manner quite well but are not adequate to reproduce all the details of the actual EMG patterns.

[4] Of the data presented here, Figs. 4*A* and 8*A* show the clearest scaling of the EMG peak, and it is more often found at lower levels of effort on the part of the subjects (Gottlieb et al. 1990a).

The central command increased the latency of the antagonist pulse by an amount proportional to the expected effectiveness of the load in slowing the movement. This was appropriate to the expected torque requirements of the task and not a simple copy of the agonist command. The independence of the commands to the two muscle groups was implied by the fact that the area of the antagonist burst does not increase with movement distance as does the area of the agonist burst (Gottlieb et al. 1992). This independence is not evident in experiments with different inertial loads because the areas of the two bursts are positively correlated. It becomes clearer with viscous and elastic loads for which the areas of the two bursts are negatively correlated. Thus the areas of the two bursts can be independently controlled, based on the torque requirements of an external load.

Under predictable load conditions, the nervous system is able to do a very effective job of preserving the kinematic trajectory by appropriate anticipation for elastic and viscous loads. This is a kinematic consequence of excitation-based control rules that act early enough in the movement (by pulse-width and pulse latency modulation) to be effective with dynamic loads that affect forces late in the trajectory. Larger load changes and weaker subjects reveal greater trajectory perturbation. This strategy does a less effective job for inertial loads, although for light loads (or strong subjects) it can do quite well (Gottlieb 1994). If a subject wished to more closely preserve the trajectory, they would have to adopt a different movement strategy that involved pulse-height modulation and altered rates of rise of EMG bursts and muscle torques. This presumably could be observed with an appropriate experimental paradigm different from the one used here. It is not the strategy that is normally used, however.

Mechanical response to unpredictable changes in load

Unpredictable changes in load *always* alter the movement trajectory. Viscous or inertial changes affect movement speed but not the final position. The reported exceptions (e.g., Sanes 1986) probably represent a secondary, corrective reaction by the subject. By contrast, unpredictable changes in elastic loads produce static errors in final position that require a corrective movement (Levin et al. 1992). The changes in movement time and in peak movement velocity following changes in elastic load shown here and in Gottlieb (1994) are small and scale with changes in the actual distance moved, just as they would if a subject intentionally changed movement distance.

Equation 4 models muscle torque as depending on a central command (α) plus two components that are proportional to muscle length and its rate of change. The latter two are elastic and viscous terms that arise from the summed effects of both intrinsic muscular properties (i.e., the classical length-tension and force-velocity relations) and from time-varying changes in muscle activation that are reflex driven. Although there is a small delay to be expected between the onset of the EMG and muscle torque, it is too small to be resolved by this form of data analysis (Corcos et al. 1992). Muscle torque is equal and opposite to the algebraic sum of the motor and inertial torques

$$\tau_{motor} - M\frac{d^2\theta}{dt^2} = (K_{muscle} + K_{reflex})(\theta - \lambda) + (B_{muscle} + B_{reflex})\frac{d\theta}{dt} + f(\alpha) \quad (4)$$

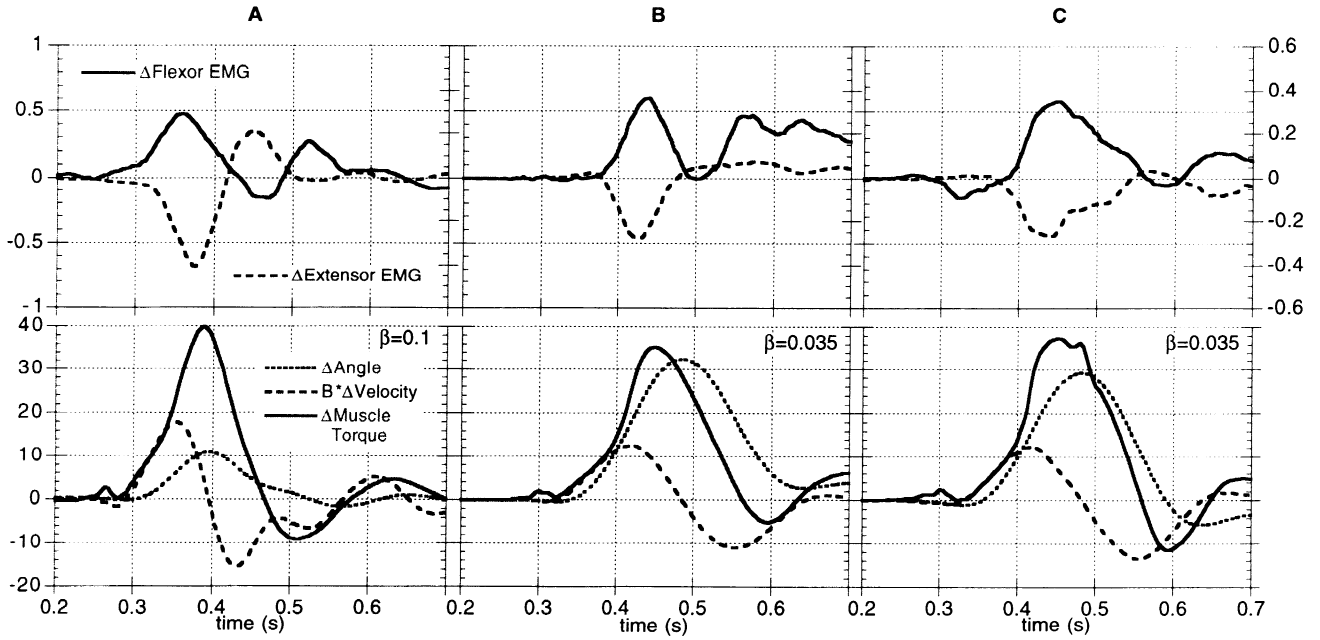

FIG. 7. Differences between movements with the 0 and 2.87 Nm · s/radian viscous loads shown in Fig. 6. *Top*: differences in flexor and extensor EMG records. Time axis has been expanded and starts at the onset of the agonist burst. *Bottom*: differences in angle (deg), muscle torque (Nm), and velocity. Velocity trace has been multiplied by the value β in the *top right corners* of the figures.

The figures that illustrate movements against different known loads (Figs. 2 and 4–9*A*) show simultaneous load-dependent differences in EMGs and torques, ~75 ms after the onset of voluntary agonist contraction. We interpret both as resulting from load-specific changes in the central command (α) that lead to a change in contractile torque and a consequent change in trajectory. The EMG changes cannot be angular or velocity reflex driven because they are not preceded by divergence of the angle or velocity trajectories.

When the load changes unexpectedly, there is delay before any central response can alter muscle contraction, and changes in muscle force and in kinematic variables precede changes in the EMG patterns by ≥75 ms. The earliest difference in torque between the two loaded conditions is matched by a scaled version (scaled by the factor β in Figs. 5, *A–C*, 7, *A–C*, and 9, *A–C*) of the divergence in velocity. The force-velocity properties of muscle imply that at any level of contraction, the faster a muscle shortens, the less force it is capable of generating against an external load. Therefore, in the time interval t = 275–375 ms (or more), those torque differences can be thought of as resulting from the differences in movement velocity under the assumption that viscosity is between 0.035 and 0.1 Nm s/deg (2.0–5.7 Nm s/radian), the scale factors (β) used in Figs. 5, 7, and 9. The limb moves more slowly with the larger load and consequently, the agonist muscles produce greater force. Toward the end of that interval, elastic properties may become significant as well. It is the elastic properties of the system that account for torques when the limb is moving slowly, or is at rest and at an equilibrium, but they play a negligible role at the onset of a fast movement.

The above arguments apply to inertial loads (Figs. 4 and 5) and viscous loads (Figs. 6 and 7). Divergence between mechanical trajectories begins somewhat later when viscous loading is compared with inertial loading because the difference in the externally applied torque is proportional to differences in velocity rather than acceleration. Nevertheless, the differences in muscle torque parallel the velocity differences. This is repeated at a lower level in Figs. 8 and 9 with elastic loads. The perturbation of the trajectory by elastic loads is smaller and later in time than that produced by viscous and inertial loads, and the differences between the muscle torques for the two loads are also smaller and later. Those torques still correspond to the deviation in velocities of the two movements.

Compliant properties of the joint affect fast voluntary movements by altering muscle torque in a motion-dependent fashion. The conclusion of this section is that at movement onset, the viscous rather than the elastic attributes of joint compliance are the dominant ones. Constructs based predominantly or exclusively on joint elasticity such as the "invariant characteristic" (Feldman et al. 1990) or "joint compliant characteristic" (Latash and Gottlieb 1991b), although appropriate to describing posture and slow movements, cannot account for torque production and therefore are not adequate for understanding fast voluntary movements.

At this point we can answer the second question of the INTRODUCTION concerning how forces and movements change with load. Increases in load, of any type and whether expected or not, lead to increases in joint torque. The effectiveness of a load in reducing movement speed is of course proportional to its magnitude but is also strongly dependent on the dynamic properties of the load that determine where in the trajectory, load-dependent forces develop.

Neurally mediated responses to unpredictable changes in load

When the loads are known in advance, load-dependent changes in trajectory and muscle torque are simultaneous

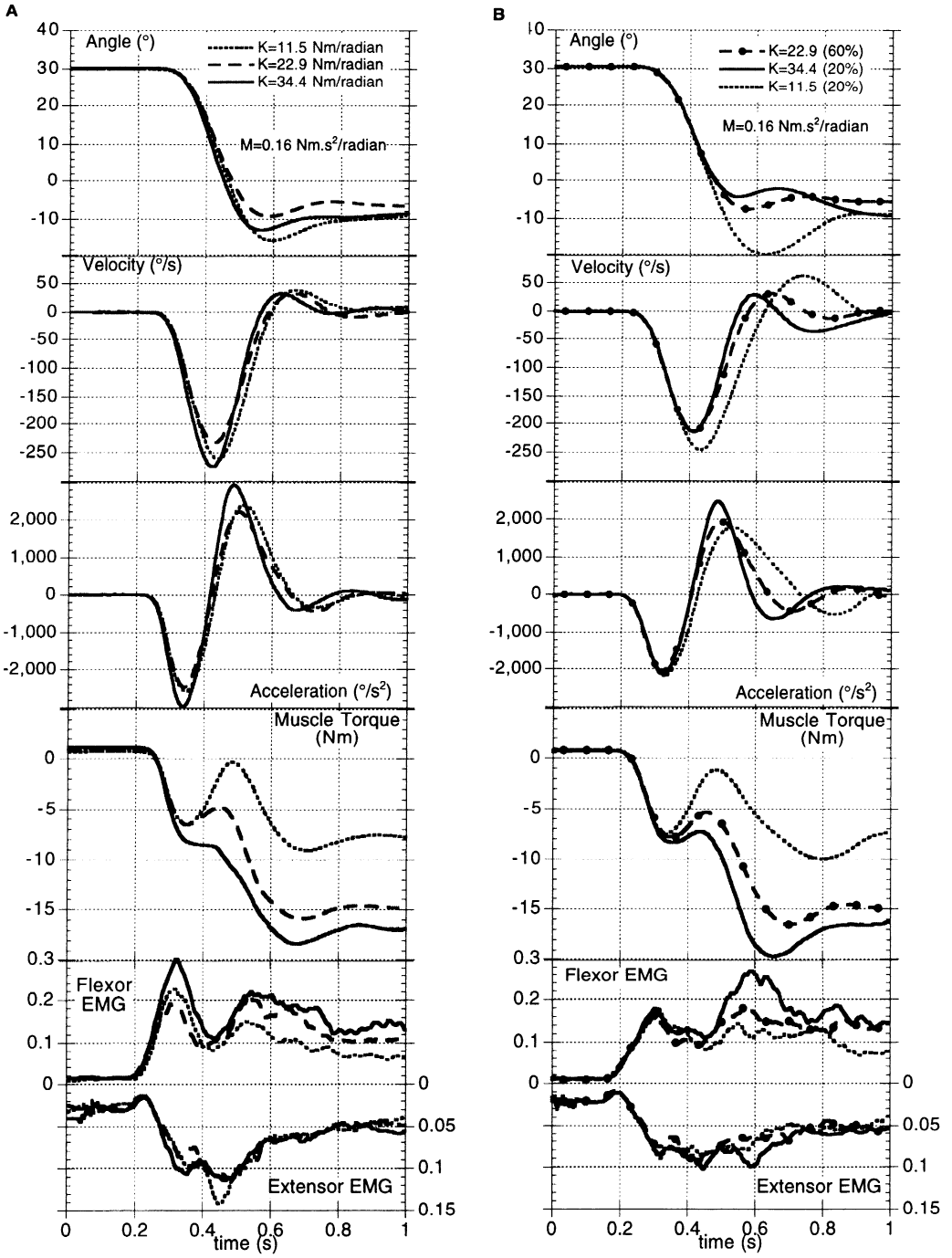

FIG. 8. Movements of 36° performed against 3 different elastic load (11.5, 22.9, and 34.4 Nm·s/radian). Average kinematic, net muscle torque, and EMG records. *A*: all loads known. *B*: middle elastic load (22.9 Nm/radian) presented 67% of the time with the other 2 elastic loads interspersed 16% each in an unpredictable sequence. Average is of 10 of the 40 movements with the middle load (randomly selected) and of all 10 of the movements with the other loads.

with the appearance of changes in the EMG patterns. These we have attributed to changes in the central command. When loads change unexpectedly, changes in trajectory and torque precede changes in the EMG patterns. These mechanical changes we have thus far attributed to intrinsic viscouslike and to a lesser extent elastic-like muscle properties.

One question that remains is what mechanisms produce the changes in EMG that eventually follow unpredicted changes in load. Length- and velocity-sensitive reflexes are likely candidates. Looking only at the *earliest differences* in the EMG patterns, unpredicted increases in inertial or viscous loads increase the agonist and decrease the antagonist bursts (Figs. 5 and 7). The timing of these changes is compatible with Smeets' et al. (1990) calculation that short-latency stretch reflexes appear ~37 ms after the actual velocity of the loaded movement deviates by 34°/s from the expected velocity of the unloaded movement. The changes in agonist burst activity are summarized by Fig. 11*A*, which compares the relative changes for unpredicted load changes (ordinate) with those found for known changes (abscissa). However, there is a great deal more intersubject variability in this EMG measure than in the velocity measures of Fig.

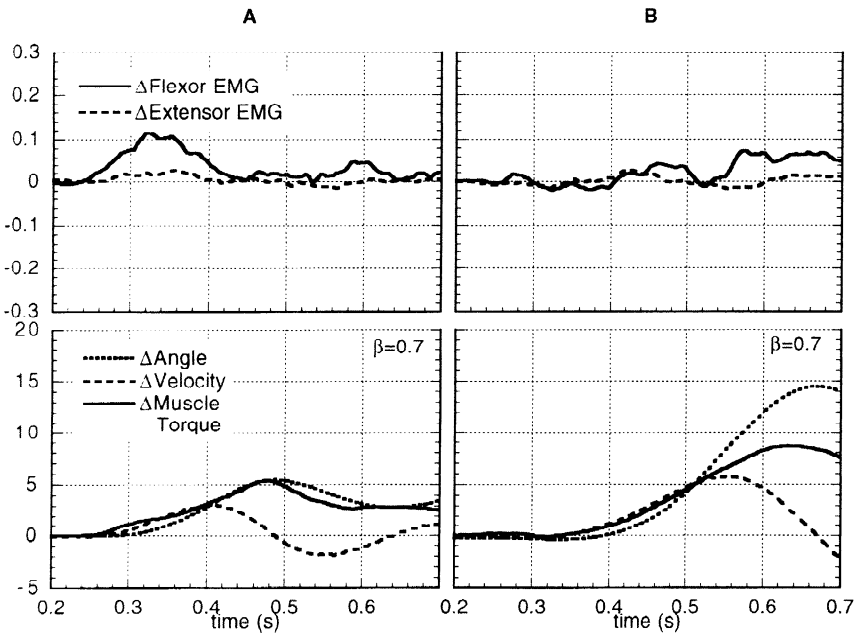

FIG. 9. Differences between movements with the 11.5 and 34.4 Nm/radian elastic loads shown in Fig. 8. *Top*: differences in flexor and extensor EMG records. Time axis has been expanded and starts at the onset of the agonist burst. *Bottom*: differences in angle (deg), muscle torque (Nm), and velocity. Velocity trace has been multiplied by the value β in the *top right corners* of the figures.

10*A* that presumably produced these responses. Some subjects demonstrated large EMG changes with unpredicted loads, but some produced little or no change at all. Inertial and viscous loads had similar effects, although the inertially induced were usually larger in our apparatus.

The picture is more complex for the antagonist burst. There is an initial relative decrease in the antagonist EMG following an unpredicted increase in inertial load (Figs. 4 and 5), that is simply a consequence of the increase in the latency of the burst. The net area of the antagonist burst, integrated over the total time of the movement, is increased as shown by Fig. 11*B*, where the symbols for inertial experiments are in the first quadrant. In contrast to this, increased viscous loads reduced the antagonist burst, and so those symbols are in the third quadrant. Figure 11*C* illustrates that the latency of the antagonist burst was consistently altered by predictable inertial and viscous load changes and unpredictable inertial changes, and not by other types or conditions.

Force-time models

The notion that a central plan for the production of muscle force plays a dominant role in the production of voluntary movement has a long history (Bock 1994; Henry and Rogers 1960; Lashley 1917; Schmidt et al. 1979) that is congruent with our model of programmed excitation pulses (Gottlieb 1993b). This mode of feed-forward control, along with feedback control, is also used in robotics (Hollerbach and Bennett 1992). Force planning does not explicitly address either the role of reflexes nor does it provide a basis for postural equilibrium. On these counts it cannot deal with either external perturbations or equivalently, with an incorrect program, i.e., one not accurately matched to the external requirements of the task. A control system that explicitly specifies muscle force or joint torque will produce an error in final position (and can even drive a limb to its mechanical limits) if the command is not exactly matched to the external load. As shown here, changes in external inertial or viscous loads (which impose only a transient change in net joint torque) do not prevent the limb from achieving its intended target. Similarly, it has been shown that other forms of transient torque perturbations do not change final position (Bizzi et al. 1976; Latash and Gottlieb 1990). Transient torques would affect the final position of a force-controlled inertial system, so force program control systems must operate in parallel with additional, equilibrium-producing mechanisms to enable kinematic success in spite of transient errors in force production.

Kinematic (α/λ) models

The equilibrium point models formulate planning in kinematic terms with an explicit role for position-dependent muscle forces. The α-model (Bizzi et al. 1992; McIntyre and Bizzi 1993) is based on the combined action of compliant muscle and reflex mechanisms to produce a moving but stable equilibrium point. The λ-model relies more strongly on reflex action, although it has added several independent control parameters and reciprocal inhibition as necessary elements to try to reproduce the agonist and antagonist burst patterns (Feldman and Levin 1995; St-Onge et al. 1993). While reciprocal inhibition is likely to play a role, Figs. 2 and 3 demonstrate that very different load-dependent variations in antagonist bursts (which imply different reciprocal inhibition) do not necessarily lead to differences in the agonist bursts. Neither of these equilibrium point versions can yet account for movements against different types of loads. McIntyre and Bizzi (1993) explicitly excluded even load magnitude changes from consideration. Moving equilibrium models can account for the data shown here (Gottlieb 1992), but in doing so, the control trajectory must be more complex than the actual trajectory (Gomi and Kawato 1996; Hasan 1986; Hogan 1984; Latash 1994; Latash and Gottlieb 1991b).[5]

[5] Hodgson (1994) has shown that if the muscle viscous element is attached to the moving equilibrium point (λ) rather than to a fixed reference as in Fig. 12*A*, the virtual trajectory is smoother and more like the actual trajectory. It is still not invariant over changes in limb/load dynamics, however.

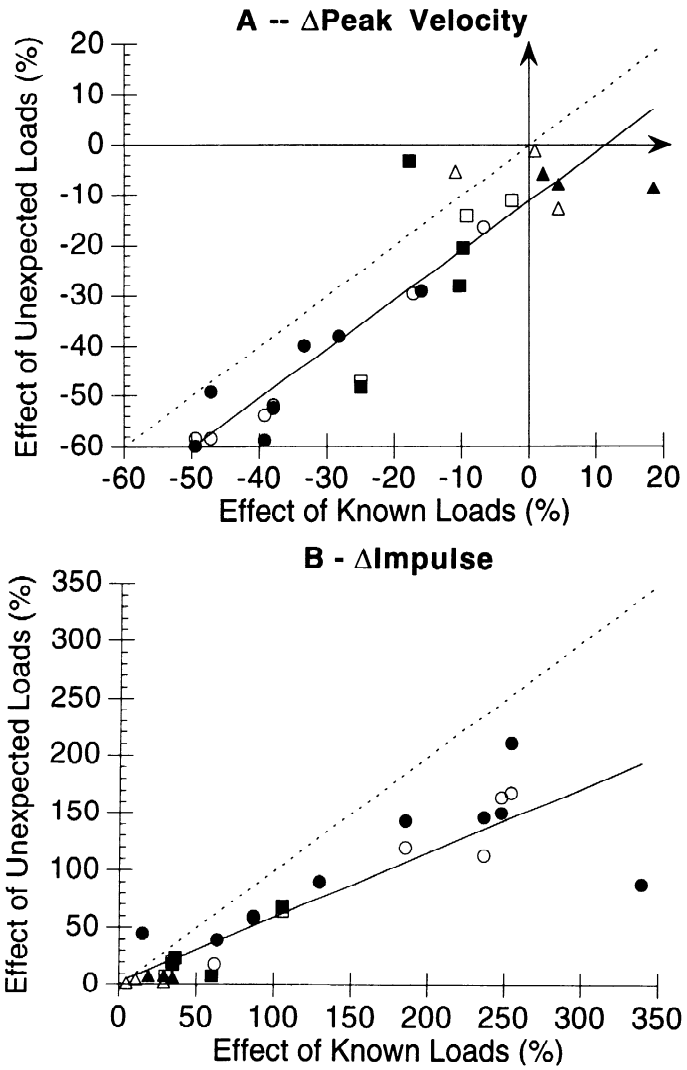

FIG. 10. Effects of changing load on the peak velocity and torque: the effect of any change in load depends on whether the change is predictable (plotted on the abscissa) or unpredictable (plotted on the ordinate). If predictability had no effect, all data points would lie about the dashed "indifference" line. Each data point represents the mean change for 1 subject in 1 experiment. *A*: decrease in peak velocity (%) produced by the larger of 2 loads. Solid line is the linear regression ($y = -11.2 + 0.98x$, $r = 0.91$) curve and lies below the indifference line. Larger loads slow movements, an effect that is reduced with predictable loads. The degree of slowing also depends on the magnitude of the load change and on the type of load. Inertial loads are plotted with circles, viscous loads with squares, and elastic loads with triangles. Open symbols are used when with unpredictable loads, the most likely load was the largest and solid symbol when it was the smallest. *B*: increase in impulse (%) produced by the larger of 2 loads. Larger loads lead to an increase in the impulse, an effect that is augmented with unpredictable loads. Solid line, the linear regression curve ($y = -2.6 + 0.56x$, $r = 0.89$), lies below the indifference line showing that predictable loads enable subject to increase the impulse produced by the muscles for any change in load. Changes in inertial load have the largest effect, and changes in elastic load a very small effect.

The λ-model rejects, in favor of reflex action, the notion that major features of the EMG patterns such as the antagonist latency are centrally determined (Feldman and Levin 1995; St-Onge et al. 1993). It is not clear how, or even if, according to that model, central commands adapt to different inertial or viscous loads or differences between inertial and viscous loads. It seems clear, however, that central commands must change when either of these loads change in a

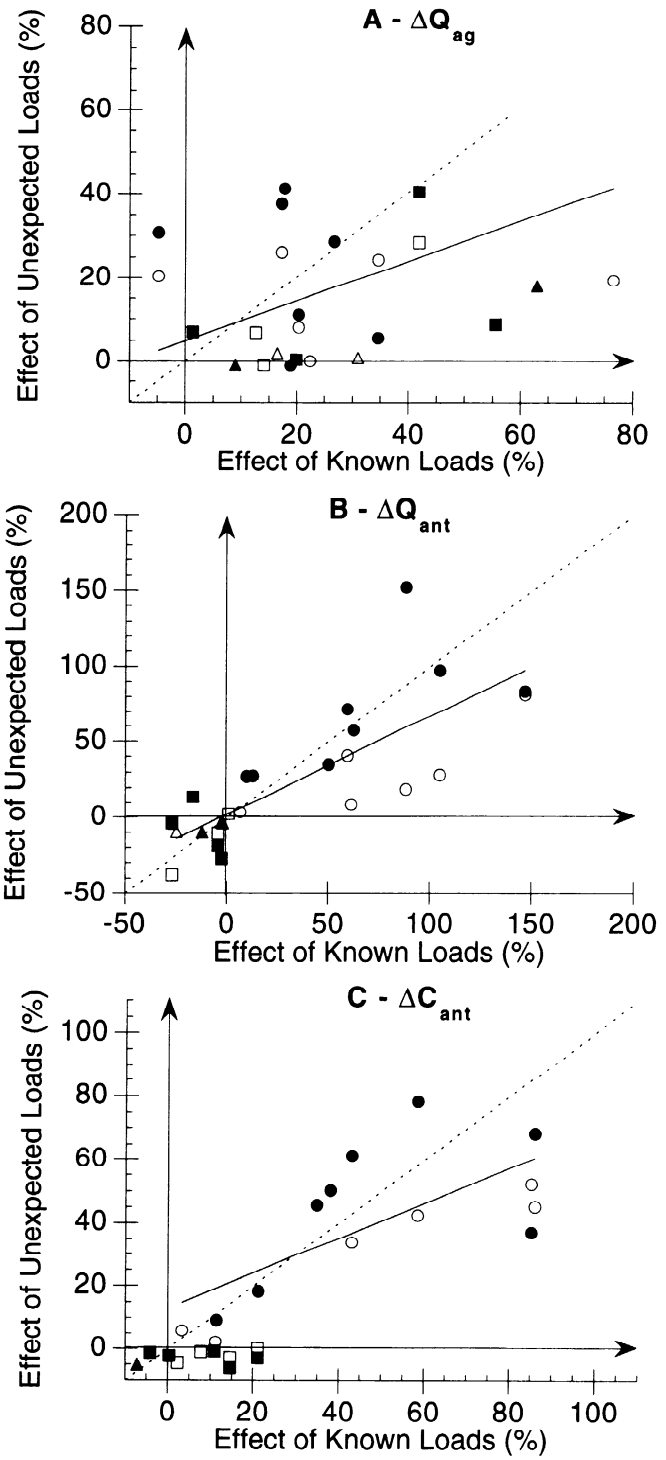

FIG. 11. Effects of changing load on the EMG patterns: the effect of any change in load depends on whether the change is known (plotted on the abscissa) or unpredicted (plotted on the ordinate). All data points would lie about the dashed "indifference" line if prior knowledge of the load had no effect. Each data point represents the mean change for 1 subject in 1 experiment. *A*: effects of load changes in the area of the agonist burst. Inertial loads are plotted with circles, viscous loads with squares, and elastic loads with triangles. Open symbols are used if the most likely load was the largest and solid symbols when it was the smallest. Solid line is the linear regression curve for pooled data of all 3 loads ($y = 4.8 + 0.45x$, $r = 0.42$). *B*: effects of load changes in area of the antagonist burst, computed over the full movement time. Solid line is the linear regression curve for pooled data of all 3 loads ($y = 1.9 + 0.65x$, $r = 0.78$). *C*: effects of load changes on the effective latency of the antagonist burst. Solid line is the linear regression curve for only the inertial loads ($y = 13 + 0.553x$, $r = 0.71$). There was no effect on latencies for unpredictable viscous or elastic loads.

predictable manner. If this were false, then we would not expect there to be differences between movements of identical loads based on whether or not the subject had prior knowledge of the load.

Because the α-model explicitly incorporates muscle mechanical properties in centrally mediated adaptation to external loads, the findings reported here are more compatible with it than they are with the more reflex-dependent λ version. Our results lead to the hypothesis that most of the adaptation in the patterns of muscle activation are centrally governed by a controller that "knows physics." That is to say, the controller knows which kinds of loads require larger agonist bursts, implements them by planning longer agonist activation pulses, and if those loads are expected to prolong the movement, it delays the antagonist burst accordingly. The controller knows the difference between inertial, viscous, and elastic loads and also adjusts the area of the antagonist burst. For movements in which the dynamic forces are small however, a feedback model is sufficient.

Three-mode model: programs, plans, and volitional set

Our new model, based on *Eq. 4,* has three parallel elements; an elastic spring, a viscous damper, and a force generator and is illustrated in Fig. 12. It is useful to treat the central command as consisting of three components, a *force*[6] *program* command α, a *trajectory plan* command λ, and a *volitional set* or reflex modulation command γ. They are described as follows.

1) The first component, α, is an excitation pattern to activate the motoneuron pools based on estimates of the dynamic forces needed to move the expected load at the intended speed over the desired distance (Gottlieb 1993b). The changes in EMG patterns seen with known load changes are programmed into this variable. For fast movements, it produces a biphasic force pulse and is the largest component, dominated by excitation pulses for acceleration and deceleration. The pulses will diminish in size and eventually vanish as the intended speed of the movement is reduced. This programming requires extensive knowledge about the task (which can be considered an internal model) in order to launch the movement correctly.

2) The second component, λ, is a kinematic plan analogous to what can be termed a "virtual trajectory." It is specified in terms of the expected sensory correlate of a successful movement and functions as a kinematic reference, to which sensory feedback signals are compared and from which reflex reactions are produced. The existence of a λ component is implicit in the existence of reflexes with similar properties at both the initial and final limb positions and that respond to and partially compensate when the trajectory deviates from the expected result of the α-command. For fast movements performed under predictable load conditions, α is usually accurate and adequate to perform the task, and λ would contribute little to the transient muscle torque. For movements that are sufficiently slow that the velocity- and acceleration-dependent forces are relatively small, however, α would vanish, and the λ component might be sufficient (Flash 1987).

Shadmehr and Mussa-Ivaldi (1994) used unusual dynamic loads to cause curved movements that the subject gradually straightened with repetition. The interpretation of this experiment was that with practice, subjects refined an internal dynamic model of the task that enabled muscle forces to be adapted to straighten the trajectory. We concur and in terms of our model, this is accomplished by the progressive adaptation of the α-command over a series of repeated movements based, in part, on the load-induced deviations of the actual trajectory from the λ command.

There must be another level of movement planning in addition to this, however, to account for the findings that when straight trajectories are made to appear curved, there is a gradual adaptation of the trajectory to straighten its visual appearance by curving its kinematics (Flanagan and Rao 1995; Wolpert et al. 1995). In this case, there must be some "higher" criterion that "prefers" the appearance of straightness over the reality. It adapts the kinematic plan (λ) to the curvature needed to produce a straight appearance, and the force program (α) is matched to produce that.[7] It evidently does this without conscious appreciation of what it is doing, however.

The action of λ here is as a position reference. We have not used its first derivative as a velocity reference and therefore have defined the proximal end of the viscous element in Fig. 12 as stationary. This allows us to segregate the control of load dynamics into the α command and seems physiologically reasonable because velocity-dependent muscle properties diminish the externally expressed contractile force of a shortening muscle and increase it in a lengthening one. Therefore α will be increased to voluntarily shorten a muscle and decreased if the muscle is lengthening, which is compatible with the observed EMG dependency on external load. The alternative of anchoring the viscous element on the moving λ has been explored by (Hodgson 1994).

Because λ represents the equilibrium position of the limb, this command must act in an anatomically distributed manner because the instantaneous equilibrium position depends on several factors. These include intrinsic muscle properties, the activation level of the muscle, reflex thresholds within the spinal cord, and spindle receptor properties that are under fusimotor control.

An alternative model is to have λ and α combined into a single control signal. For example, our proposed "force" command could be replaced by a complex, time-varying equilibrium trajectory input to the proximal end of the spring that is proportional to $\lambda + \alpha/K$. This is exactly analogous to the Thevenin-Norton Equivalent transformation in electrical circuit theory that describes going be-

[6] Strictly speaking, no central command to the α-motoneuron pool can be exactly congruent with muscle force of length because of neuromuscular compliance. What we imply by the use of these descriptive terms is that the α-command will closely resemble the change in muscle force and the λ-command the change in muscle length but only under external conditions that allow the intended movement to be realized. For example, when you accurately move a correctly anticipated load over a planned distance, the α- and λ-commands are matched to the conditions of the task and resemble the actual force and length trajectories, respectively. Otherwise, force and motion will be modified by the compliant interaction of muscle and load and differ from their planned values.

[7] It has also been found that the blind make straight, 2-dimensional hand paths (Miall and Haggard 1995). This could imply that there is a proprioceptive alternative to vision, at least in the blind. In normally visioned subjects, this alternative information source is presumably overridden if it is present because straight paths are not preserved in the presence of visual distortions.

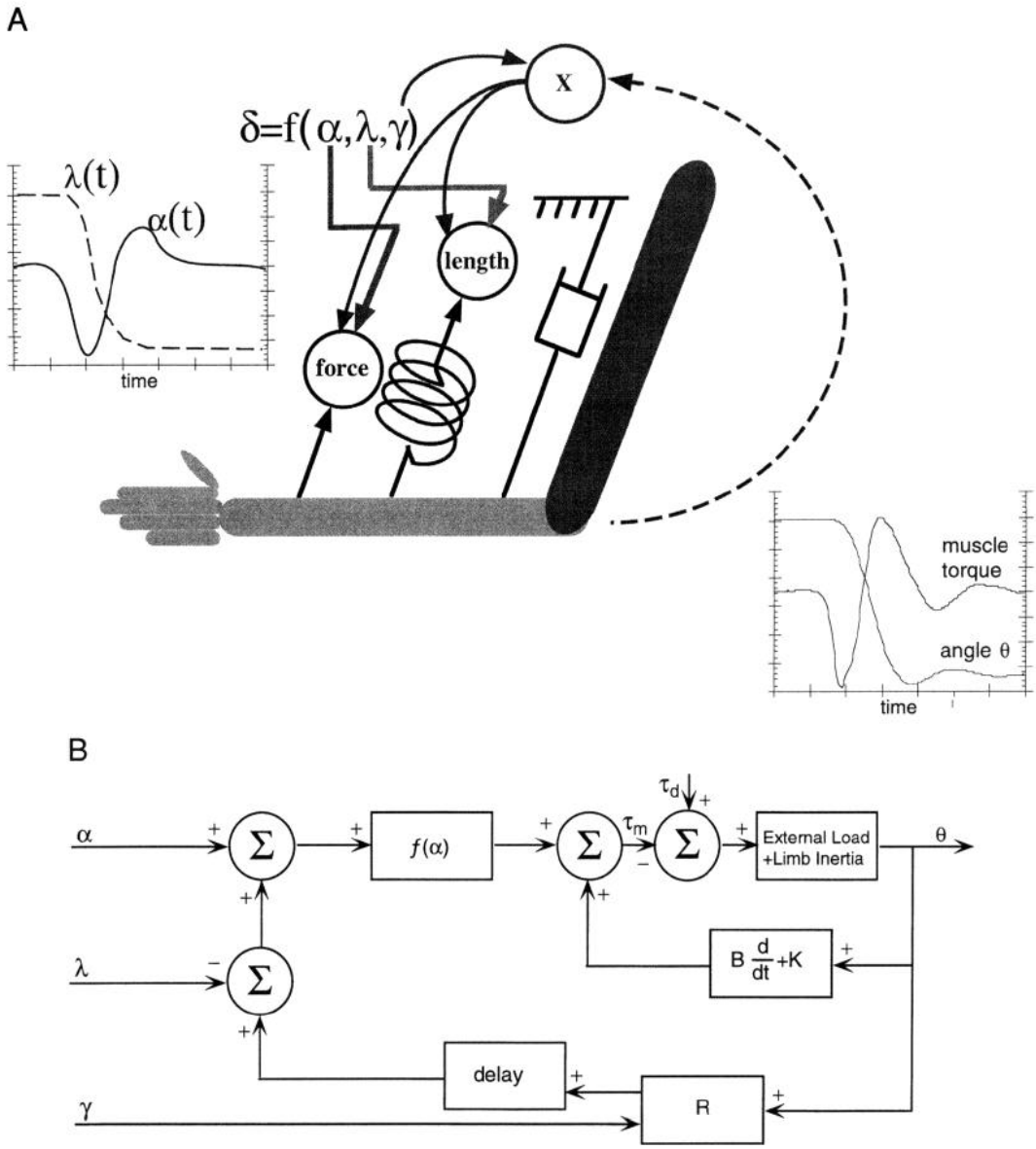

FIG. 12. *A*: a triad of control signals is used to control movement. These 3 signals, a "force" command (α), a "length" command (λ), and a reflex "gain" command (γ) control the behavior of a viscoelastic mechanical system. Although the elastic properties determine the equilibrium position of the limb, the viscous and inertial properties dominate the initial kinematics of a fast movement. *B*: one form of block diagram equivalent of the neuromechanical model in *A*, combining feed-forward and feedback control. Signs at each arrowhead indicate whether the signal entering a block causes the block's output to increase (+) or decrease (−). The muscle consists of a force generator [$f(\alpha)$] and the viscoelastic compliance. The receptor (R) is the muscle spindle apparatus. The α-input increases muscle force and causes muscle length (θ) to decrease. External forces increase muscle length. Reflexes increase muscle contraction. Reflex inputs are assumed to make relatively little contribution to muscle contraction in which the α-command is sufficient to produce a sensory correlate of the movement (θ) that matches the λ-command.

tween series voltage and parallel current sources. In the present case, this kind of a transformation results in a single motoneuron pool activation signal that cannot be closely identified with actual muscle force or length but only with their weighted sum that can be described by an "N"-shaped virtual trajectory (Gomi and Kawato 1996; Latash and Gottlieb 1991b). The two approaches are functionally equivalent (Gottlieb 1992) and differ primarily in the way that they characterize the nature of the centrally controlled independent variable, a dispute that is not presently resolvable by experiment.

3) The third element, γ, sets the gains and thresholds of the segmental reflexes to account for the differences in the effects of load-induced slowing between inertial and viscoelastic conditions. This we term "volitional set" of the segmental reflex apparatus, analogous to the more familiar "postural set" (Hore et al. 1990) that allows reflex compensation to be tuned to the task. Reflexes decrease the antagonist burst when the movement is slowed by a viscous load, a conventional behavior of "stretch reflexes," but they are not recruited soon enough by viscous loads to alter antagonist latency. In contrast, when inertial loads slow the move-

ment, deviations in movement velocity emerge earlier in the trajectory, and so the antagonist burst is delayed and increased in magnitude. Reflex actions for both loading conditions are appropriate to the dynamic requirements of the loads, but the inertially induced increase in magnitude is counter to the normal behavior of stretch reflexes. It is analogous to reflex reversals seen, for example, during gait (Duysens et al. 1990; Pearson and Collins 1993). Such context-dependent changes are analogous to the findings of Koshland et al. (1991), who found that the coupling between elbow and wrist muscles following elbow perturbations could be changed by pronation of the wrist. This flexibility here requires a third, independent γ-command to adjust the velocity sensitivity of the agonist and antagonist motoneuron pools and the antagonist latency.[8]

In answer to the first question in the INTRODUCTION about how central commands adapt to known loads, we suggest that it is the combination of α- and λ-commands that sculpts the joint torque patterns to the specific requirements of each load. We also suggest that the γ-command is used in differing degrees by different subjects because of the variability of the various components of the EMG responses shown in Fig. 11. In some subjects, reflex gain for the agonist is quite low, and the agonist burst changes little, even when the movement progresses at an unplanned rate (e.g., Fig. 2) (also Fig. 2 of Gottlieb 1994). Even the forces and kinematics of the movements themselves are not without variability among subjects. In performing voluntary movement, this may be interpreted as showing that individuals have different "styles" of movement, even at the level of single-joint elbow flexion, just as we recognize them as having different styles of handwriting, speaking, or walking.[9]

Figure 12*B* shows a more abstract formulation of this model. It illustrates the dual, negative feedback control system that responds to external torque perturbations. The inner loop represents muscle properties, and the outer loop passing through the receptor R represents (mostly segmental) reflex mechanisms. The experiments here have illustrated some of the properties of both these loops.

The control model we have proposed is a combination of three types of control; feed-forward, feedback, and active feedback control. The α-command is the primary, feed-forward control signal that drives the system for movements in which dynamical forces are significant (especially fast movements and inertially loaded movements). Operating the system by way of the λ-command appears as a standard feedback control system approach and suffices for movements that are slow enough. In fact, to state the case in its most extreme form, we suggest that for faster movements, γ opens the loop of the segmental reflexes such that they contribute relatively little to movements that have a well-planned α-command. The λ-command is quite important, nevertheless, because if α is poorly chosen (as it is when we provide the subjects with unpredicted loads), reflexes can augment muscle properties in reducing errors. If there is a discrepancy between λ and the afferent correlate of the movement, this can be used to learn a better form for α (Shadmehr and Mussa-Ivaldi 1994). The active feedback control signal, mediated by γ, enhances the quality of the feedback signal as well as its processing. [For simplicity, we have treated λ and γ as acting separately and discretely at different points in the reflex loop. In reality, both are likely to be distributed commands involving peripheral receptors (i.e., muscle spindles) and spinal interneuron synaptic gains. Such detail would greatly complicate any block diagram representation and has been omitted.]

The diagram in Fig. 12*B* shows that α and λ could easily be combined into a single command that we could call a "virtual trajectory" if we chose. Such a trajectory would differ significantly from the actual trajectory, however (Gomi and Kawato 1996). Our model separates the control of dynamics or movement from that of statics or posture at a conceptual level. Whether this separation can be supported on an anatomic or physiological basis is well beyond our current abilities to address experimentally. What the model here emphasizes by its focus on the α-command is the necessity for the CNS to deal explicitly with limb/load dynamics. They cannot be delegated to the peripheral feedback controller. Although this is implicit in most formulations of the equilibrium point hypothesis (even the λ-model has, in addition to a shifting equilibrium point, several control parameters that take the role of α and γ in our model), those models focus almost exclusively upon the λ-command. This can lead to the attractive misapprehension that if the nervous system has specified a virtual trajectory, it has "solved" the movement control problem. It has not.

Conclusions

The three-mode model has three essential features. First, muscles and reflexes act together as a feedback controller to determine the viscoelastic behavior about the joints. Second, there is a relatively smart central, feed-forward controller that "knows dynamics" and plans a force pattern based on the desired trajectory that pattern is expected to produce.

[8] For example, we previously suggested (Gottlieb 1993b) that the antagonist latency was entirely a product of central computation (i.e., specified solely by α). *Equation 5* shows how we might combine that with peripheral feedback

$$T_{ant} = T_0 + \frac{X}{F + V_{fb}/V_\gamma} \quad (5)$$

In this equation, the latency of the antagonist burst (T_{ant}) is proportional to X ("Extent," which is a function of distance and load) and inversely proportional to F ("Effort," which is proportional to intended speed), both of which are elements of α. V_{fb} is a sensory signal that presumably arises in the muscle spindles and is proportional to actual movement speed. V_γ is a scaling factor that is part of the γ-command. Antagonist latency will by reduced by the intention to move fast (F) and further reduced if the actual speed (V_{fb}) is greater than planned. The strength of this reflex effect is controlled by V_γ. This might be easily done by converging central and reflex inputs (F and V_{fb}) on antagonist motoneurons that have a controllable threshold.

[9] Smeets et al. (1990) reported strong reflex effects of large inertial load changes. Latash (1994), using inertial load changes smaller than those used here, found statistically significant changes in the rising phases of the antagonist bursts by carefully identifying the appropriate 20-ms interval in the EMG record to evaluate. No other EMG changes were reported. The magnitudes of results presented here and in Gottlieb (1994) span the range of both of those observations. It is possible that not only is intersubject style a source of variability but that the reflex phenomena are not linear. Small stimuli may have undetectable effects, and very large stimuli may be "unphysiological." Perhaps reflex mechanisms must be studied in a range of behaviors and with a range of evocative perturbations in which they are at least potentially effective and useful in order to be properly observed.

Third, reflexes are variable and adaptable, not stereotyped. In this scheme, the CNS improves the on-line load compensation of the feedback system. Most of the agonist and antagonist activation patterning for fast movements is specified by the central control program, and if that program does not produce the expected trajectory, the command will be modified by trial and error until subsequent movements are satisfactory.

In contrast to the various versions of the EP hypothesis that have tried to incorporate movement and posture under a single, unifying control scheme, this approach parcellates these aspects of control into separate categories. Looking at the nervous system as a modular collection of functionally discrete, semiautonomous but intimately linked processing centers is not a new idea. It has extensive roots in the clinical literature that has described numerous losses of highly specific, cognitive functions due to localized central lesions. Gardiner (1993) has argued that even the concept of intelligence should not be looked at as monolithic but as consisting of distinguishable components that are realized in each individual to different degrees. In robotics, Raibert (1986) has described a three-part controller for a one-legged, hopping robot where hopping height, forward progression, and attitude are controlled separately. Flanders and Soechting (1990) have proposed that some of the computation involved in visual and kinesthetic sensorimotor transformations should also be treated as multiple, functionally distinct processes.

Here we have begun to formally develop the argument that the mechanisms underlying voluntary motor control are best described as at least two, functionally distinct systems by distinguishing the problem of dynamics or movement from that of statics or posture. We propose that the successful solutions to the problems of dynamics that we achieve in almost all of our routine movements are the results of plans for the production of force; that is, of muscle activation patterns. At the same time, the accuracy and precision we achieve in positioning our limbs emerges more directly from central, kinematic plans. In performing both tasks, the compliant properties of the neuromuscular system are critical to kinematic success and system stability. At the onset of movement, inertia and viscous muscle properties dominate, and these are awkwardly controlled in purely positional terms. As time progresses, the elastic behavior of both muscle and reflex grows in importance, and this is characterized by notions of moving equilibrium states. To account for the variability among subjects, we have proposed that the modulation of reflex function be treated as a third distinguishable controlled element. Hence a "three-mode" model.

To finally answer the third question of the INTRODUCTION concerning the mechanisms by which the CNS deals with different loads, it must be a combination of all three elements. At first glance, it is not a particularly simple model that emerges from trying to describe this wide variety of experimental observations. It has points of similarity with previous theories but points of disagreement as well. On the latter, we believe the three-mode model is consistent with a much broader experimental data base. It has omissions (such as reciprocal inhibition) and oversimplifications (such as rectangular excitation pulses) that will require further work.

From a different perspective, however, the model is extremely simple. It does not require any neurological mechanisms that are not well recognized in animal and human studies. A small number of parameters (excitation pulse height, width, and latency), correlated according to the expected dynamic requirements of the load, are used to match each muscle's excitation pulse to the estimated dynamical properties of the load. This is sufficient to "launch" a movement in the right direction, even in the multiple joint case (Almeida et al. 1995; Gottlieb et al. 1996a,b; Hong et al. 1994). Accurate final position does not depend on the accuracy of this launch but rather on the specification of a stable equilibrium, established by the tonic level of muscle coactivation (Koshland and Hasan 1994; Lestienne et al. 1981). Only for movements that do not require large dynamic forces is a moving equilibrium point alone sufficient to control the movement, however. Thus the nervous system can use a rather simple set of control rules that under well-known conditions will produce consistent and accurate movements. Under unknown conditions, it will either slow the movement to give higher centers time to append corrections or else just hope for a lucky guess. Fast, accurate movements require knowledge, usually obtained by extensive practice, as we have always known.

I thank Dr. Bryan Flaherty for programming the servo, D. Odo for assistance in performing these experiments, D. Corcos for advice and counsel, and P. Cordo for constructively generous criticism.

This work was supported in part by National Institutes of Health Grants RO1-AR-33189, RO1-NS-28176, KO4-NS-01508, and RO1-NS-28127.

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Received 4 April 1996; accepted in final form 3 July 1996.

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