Organizing Principles for Single-Joint Movements

I. A Speed-Insensitive Strategy

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1. Normal human subjects made discrete elbow flexions and extensions in the horizontal plane from a stationary initial position to visually defined targets at different distances with a constant inertial load or made flexions to a visually defined target with different inertial loads. We measured joint angle, acceleration, and electromyograms (EMGs) from two agonist and two antagonist muscles.

2. Subjects were instructed to move their limbs accurately but quickly to the targets. Movements of greater distances or lesser loads were performed at higher velocities.

3. Peak inertial torque, acceleration and velocity, movement time, and integrated, rectified EMG were all highly correlated with the task variables, distance and inertial load. We show that peak inertial torque can be used as a linking variable that is almost sufficient to explain all correlations between the tasks, the EMG, and movement kinematics.

4. The rate at which subjects initially developed torque to accelerate their movements was invariant over changes in the value of either task variable. The rising phase of the agonist EMG was also independent of the distance or load moved.

5. Two components were distinguished in the antagonist EMG. The first had a relatively constant latency and amplitude. It terminated on the onset of the second and larger component at a latency that was delayed as both distance and load increased.

6. The integrated, rectified antagonist EMG was proportional to inertial load and peak decelerating torque for changes in inertial load. When target distance varied, proportionality between peak decelerating torque and antagonist EMG could be found if correction was made for the effects of muscle length on the torque-EMG relationship.

7. We propose organizing principles for the control of single-joint human movements in which tasks are performed by one of two strategies. These are called speed-insensitive and speed-sensitive strategies.

8. A model is described in which movements made under a speed-insensitive strategy are executed by controlling the duration and the relative timing of amplitude invariant patterns of activation to the spinal motoneuron pools.

INTRODUCTION

When a person is asked to repeat a simple movement task such as flexing the elbow a given distance to a visually defined target, and a single aspect of that task is altered, regularities of behavior are observed. Regularities are exhibited by covariations between variables specified by the experimenter that prescribe the movement task ("task variables") and those that describe the performed movement ("measured variables"). An example of such a relationship would be that between target distances and agonist electromyogram (EMG) peak amplitudes in an experiment to move different distances. Covariations may also be found among different measured variables, such as between agonist EMG peak amplitudes and the peak movement velocities associated with different distances.

Covariations between two measured variables do not demonstrate the existence of a causal relationship or, if one does exist, the direction in which it works. Covariations of the first kind, between task and measured variables, are directional. Task variables such as target distance or inertial load may affect measured variables but not vice versa. Such relations can reveal rules used within the nervous system to accomplish the task. The following are examples of empirical rules found in the literature between task variables and measured kinematic variables for experiments in which subjects are told to move both accurately and quickly: 1) movement time and velocity both increase with target distance (Fitts et al. 1954, 1964; Hancock and Newell 1985); 2) movement time decreases and velocity increases with target size (Fitts et al. 1954, 1964; Hancock and Newell 1985); and 3) movement time increases and velocity decreases with inertial load (Benecke et al. 1985; Corcos et al. 1986; Danoff 1979). In the first rule, movement time and velocity are normally measured, whereas "distance" refers to the distance to the target at which the subject is aiming. In this case distance is a task variable. Distance can also refer to the actual distance moved, which would make it a measured variable. Because subjects cooperate in performing experiments, actual distance and target distance are usually in close agreement, so the distinction is generally not of quantitative importance. In terms of expressing causality, however, the distinction is profound. Investigators have attempted to extend these rules to describe the EMG associated with a movement. Based upon a variety of experiments (Freund and Rudinger 1978; Hallett and Khoshbin 1980; Lestienne 1979), Wallace (1981) suggested that: 4) "The duration of the initial agonist burst will be positively related to the total movement time." (p. 151) [See also Wallace and Wright (1982)]. However, this rule fails to describe the agonist burst accurately when different distances are moved in a constant time (Sherwood et al. 1988). Rule four is part of Wallace's impulse-timing theory from which other rules emerge. For example, the amplitude of the agonist burst is proportional to peak velocity when the load is constant (Mustard and Lee 1987; Shapiro and Walter 1986) or to inertial load for constant average velocity (Lestienne 1979).
These rules for scalar EMG measures present two kinds of problems. Either they are severely circumscribed or their generality is subject to considerable uncertainty (e.g., Rule 4 above). Although all the studies cited above (and many more) describe significant correlations, all are incomplete. None can account for all the correlations found throughout the literature, nor does any provide a rationale for deciding why correlations between a pair of variables should change from one set of experiments to another (Hasan et al. 1985b).

Our goal is to find rules which are: 1) sufficiently general to be independent of the task, 2) determined by the task, and 3) a rationale for specifying the appropriate rules to apply. In this paper and its companion (Corcos et al. 1989), we will formulate rules and provide a conceptual framework that attempts to account for all the contingencies and regularities between and among task, kinematic, and EMG variables (see also Gottlieb et al. 1989). The experiments described in this paper will be interpreted according to the following organizing principles:

I. Elements of a movement task lead to a strategy governing its control. We have identified two strategies to describe how single-joint movements are accomplished for a variety of tasks.

II. Strategies consist of sets of rules that determine the patterns of muscle activation.

III. Rules for muscle activation lead to patterns of muscle torques and EMGs. Well-chosen scalar measures of torque and EMG will be highly and consistently correlated irrespective of task because of their shared causal, neural activation.

IV. Muscle torques interact with limb loads to generate kinematics (angle, its derivatives, and movement intervals). Because of the role of load in determining kinematics, no general correlations between EMG and purely kinematic measures are possible.

This paper describes experiments in which subjects used a single strategy that we call “insensitive” to movement speed, namely, movements made accurately and rapidly to targets of fixed size at variable distances or with different inertial loads. The term insensitive is used to indicate that for this class of movements, selected aspects of the behavior
pattern remain invariant, in spite of the fact that movements are performed at different speeds.

**METHODS**

**Experimental protocols**

Seated subjects viewed a computer monitor that displayed a cursor, positioned horizontally by the angle of the elbow. A narrow marker on the screen corresponded to the starting position of the limb. A broad marker was located as a target at the desired angular distance. The width of the broad marker corresponded to 9° of angular elbow rotation in all the experiments reported here. The angle measurement origin defined 0° with the elbow physically flexed at 90°. Full extension was -90° and full flexion approached +90°.

After obtaining informed consent according to Medical Center-approved protocols, two kinds of experiments were performed. The first series of experiments involved flexion and extension movements to the target at 18, 36, 54, and 72° without added inertial loads. The moment of inertia of the manipulandum was 0.18 18 Nm \( \cdot \) s^2/rad (units equivalent to kg/m^2). These experiments were performed on eight subjects, two of whom were run twice to verify the repeatability of the data. For each experiment, the subjects were instructed to relax at the initial position, corresponding to an elbow angle of -30° or -45° for flexion movements and 36° for extension movements. They were instructed that when a computer-generated tone sounded, they should accurately move to the target zone, as quickly as possible. The instructions also explained that reaction time was not important. Only data for flexion movements will be presented here because no qualitative differences were observed for extension movements.

The second set of experiments, performed on six subjects, involved movements of 18, 36, 54, and 72° with weights added to the end of the manipulandum to alter its moment of inertia. The moments of inertia of the manipulandum with the four added weights were 0.2788, 0.789 1, 1.3 14, and 1.832 Nm \( \cdot \) s^2/rad. These weights were chosen to give approximately equal increments in moment of inertia. The total moment of inertia required estimating the moment of inertia of the hand and forearm was done using regression equations (Miller and Nelson 1976) and ranged from 0.07 to 0.1 Nm \( \cdot \) s^2/rad for these six subjects.

For each experimental condition (i.e., different distance or load), subjects performed 11 movements in ~90 s. The first
movement of each condition was discarded from all subsequent analyses. Experimental conditions were set in a randomized sequence with a brief rest between conditions.

Mechanical measurements

The forearm was strapped in a rigid manipulandum that could rotate in the horizontal plane. The axis of rotation was aligned with the elbow joint, with the arm abducted to the side at 90°. Joint angle was measured by a capacitative transducer mounted on the axis of rotation. Joint acceleration was measured by a piezoresistive accelerometer, mounted 46.7 cm from the center of rotation with its axis of maximal sensitivity oriented to measure tangential acceleration.

Angle and acceleration were digitized with 12-bit resolution at a rate of 1,000/s. Joint velocity was computed from the acceleration. Inertial torque was computed by multiplying the measured acceleration by the estimated total moment of inertia.

EMG measurements

EMG surface electrodes (Liberty Mutual Myoelectrodes) were taped over the bellies of the biceps brachii, brachioradialis, and triceps (lateral and long heads) muscles. EMGs were amplified (1,600×), band-pass filtered (60–500 Hz) and digitized as above.

The digitized EMGs were full-wave rectified, smoothed by a 10 ms moving average filter, and displayed at high gain on a computer monitor, where the onsets of EMG activity in the muscles were visually estimated. The onset was taken as the first sustained rise above the baseline. The onset of the second component of the antagonist EMG required a clear discontinuity in the level of antagonist excitation. This criterion is similar to that of Mustard and Lee (1987) who used the “major vertical deflection” (p. 749) to locate the antagonist onset. Trials that were ambiguous to mark were rejected from further analysis and accounted for two to three trials per condition.

Rectified EMGs were quantified by integration over two intervals for the agonist and one for the antagonist. For the agonist, the intervals were the first 30 ms after agonist EMG onset (Q0) and from the onset to the first zero crossing of the acceleration (Qacc) (See also Bouisset et al. 1977). Integration of a fixed interval was chosen to determine whether the slopes of the initial component of the EMG records were similar across experimental conditions. This method is discussed in the Appendix.

The kinematically defined interval for Qacc, which is also the
time to peak velocity, approximately corresponds to the duration of the first agonist burst. We could have also used a fixed, but sufficiently long, interval (e.g., 300 ms) to encompass the longest burst of any series. Because burst patterns are associated with all these movements, increasing the integration interval beyond the end of the body of the burst does not qualitatively alter any of the findings reported here.

For the antagonist, the selected interval was from the agonist onset to the projected end of deceleration. This time was determined by linearly extrapolating the deceleration to 0 from the point at which it had fallen to 50% of its negative (deceleration) peak. This kinematically defined interval encompasses the antagonist burst ($Q_{\text{inc}}$).

**EMG normalization**

To interpret EMGs in a more quantitative fashion, EMGs were normalized by the following method. With the manipulandum clamped at 88°, the subject made a brief, isometric, maximal voluntary contraction (MVC) to give an estimate of MVC torque ($t_{\text{MVC}}$) at that joint angle. With the use of a level of torque corresponding to ~50% MVC at 88°, three isometric contractions were recorded. This lower level was used to minimize fatigue and to obtain records with less oscillation. All contractions were performed in both flexion and extension.

For each 50% MVC isometric contraction, 500-ms time averages of EMG ($e_{500}$) and torque ($t_{500}$) were computed, starting 500 ms after the onset of the contraction. The averaged values for the three contractions were used. A normalizing scale factor ($K$) was computed by Eq. 1

$$K = \frac{t_{500}}{e_{500} \cdot t_{\text{MVC}}} \quad (1)$$

Flexor normalization factors were computed from isometric flexion contractions and extensor normalization factors from isometric extension contractions.

The method of EMG normalization was to multiply the EMGs measured during movements by an appropriate normalization constant. (The torque ratio in Eq. 1 was always about 1/2). This was performed for all plots of EMG versus time that follow (Figs. 1–3, 9).

If the relation between EMG and torque were not angle dependent (and neglecting the possible effects of motor-unit synchronization), this method of EMG normalization would give values of normalized EMG from 0 for relaxed muscles to 1 at maximal contraction. The implications of the fact that the constancy of this relation cannot be assumed will be discussed later.

At the onset of EMG activity, a high degree of motor-unit synchronization, i.e., the tendency for motor units to discharge at or nearly at the same time, is more likely than later in the contraction (Basmajian and de Luca 1985, see p. 206). This synchronization can produce larger voltage peaks than the same number of unsynchronized motor units and, for the EMG normalization method we have used, could produce numbers in excess of 1. Values approaching or in excess of 1 will be interpreted as showing levels of muscle activation approaching MVC but not greater than that.

The reason for this complex method is not apparent from inspection of any of the averaged records shown in Figs. 1–3 or 9. In occasional single records, however, deceleration returns to zero in an exponential or in a stepped pattern, which gives inappropriately long times if a measured-zero crossing is used. An extrapolation along the descending limb of the deceleration curve is one method that does a consistent job of estimating movement duration that is reasonable to human observers.
EMGs rise at similar rates for all loads. Peak EMGs are not well differentiated, but peak inertial torques for the first three loads are.

In all three figures, there is an early phase to the antagonist EMG, with a latency of <50 ms, which is insensitive to distance or load. In the subject in Figs. 2 and 3, the early phase is much more pronounced in the lateral head of triceps than in the long head. The later phase of antagonist activity is progressively more delayed by increases in either task variable.

In all three figures, the normalized values of these EMGs represent substantial fractions of MVC activity and in some cases even exceed it. The inertial torques also reach large peak values when measured in terms of MVC.

**Quantification of the agonist EMG**

The agonist EMG has been quantified using $Q_{30}$ and $Q_{acc}$. They are plotted for biceps as a function of peak inertial torque in Fig. 4. Similar plots could be made for brachioradialis and for both heads of triceps for extension movements over different distances.

The quantification of the EMG over the interval of acceleration toward the target (which is roughly the EMG asso-

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**FIG. 5.** A: peak velocity, B: movement time, and C: peak decelerating inertial torque plotted vs. peak accelerating inertial torque. The data are from the experiment in which 4 distances were moved against 4 inertial loads. The data are from the same subject as in Figs. 2 and 3.
associated with the first agonist burst) shows that most of this variation in $Q_{acc}$ can be accounted for by the variation in peak inertial torque. This torque was very sensitive to the distance moved but more variably affected by inertial loading.

In marked contrast, the EMG measure $Q_{10}$ does not share this dependence on peak inertial torque. This supports what might be surmised from Figs. 1-3. The initial EMG slope is relatively invariant over changes in movement distance or inertial load.

The dependence of $Q_{acc}$ on peak velocity has frequently been described (e.g., Corcos et al. 1988). It can be inferred from Figs. 4 and 5 that $Q_{acc}$ increases with peak velocity and acceleration when the task is to move different distances. However, this is not true when changes in velocity result from changes in inertial load. In this case, $Q_{acc}$ is inversely related to peak velocity and acceleration. The differential effects of the two task variables (i.e., distance and load) on peak velocity and movement time are illustrated in Fig. 5, A and B.

Figure 5A illustrates that peak velocity is poorly correlated with peak inertial torque across the two task manipulations and therefore cannot be used as a task-independent predictor of $Q_{acc}$ (and vice versa). The relationship shown between $Q_{acc}$ and peak inertial torque applies over a wider range of task types than relationships between EMG and any purely kinematic measure.

Figure 5C shows the relationship between peak accelerating and peak decelerating torque for the two tasks. For changes in distance there is a high correlation between the two peaks, but this is not true for changes in inertial load. Peak decelerating torque is consistently less than peak accelerating torque, with the difference accentuated at higher loads. This asymmetrical scaling can also be discerned from a comparison of peak torques in Figs. 1 and 3.

**Antagonist latency**

Inspection of Figs. 1-3 suggests that the antagonist can be partitioned into two components. In either head of the triceps, the first component begins ~30 ms after the agonist burst (Fig. 6 shows measurements in the lateral head). This component is small and not always noticeable in some subjects. Its size is also muscle dependent. When it is distinguishable, its amplitude is relatively invariant for changes in either task variable. This low level of activity is followed by a sharp increase that is usually described as “the antagonist burst.” The latency of this antagonist burst varies with both distance and load. Although there is a very strong relationship between the delay of this antagonist burst and movement time, the relationship is not a simple single-valued one but is clearly task dependent. For example, in Fig. 6A, the longest movement (72°) with the lightest load takes the same time (~250 ms) as the shortest movement (18°) with an added load (load 2). However, the difference in latencies (lagL) of the antagonist bursts under these two conditions is ~50 ms or 20% of the total movement time. The same latencies are plotted versus peak accelerating inertial torque in Fig. 6B. Here, as in Figs. 4 and 5, the

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4 This small EMG could have been the result of volume conduction from adjacent muscle groups, but we believe this not to be the case. We carefully inspected individual, digitally unfiltered, unrectified records to verify this. By the use of intramuscular wire electrodes, other studies that have also observed this early component have concluded that the signal originates in the antagonist and is not volume conducted from signals originating in the agonist muscle (Mustard and Lec 1987).
Quantification of the antagonist EMG

Figure 7 shows the antagonist burst, $Q_{\text{dec}}$, as a function of peak decelerating torque for the two task variables. The effect of adding inertial loads is unambiguous in increasing the size of the antagonist burst, but the effect on peak decelerating torque [but not peak accelerating torque (Fig. 5C)] is small. The effect of distance on antagonist EMG is less clear, in spite of its clear influence on the decelerating torque. In fact, in most subjects in the lightest load condition, we found the antagonist burst to decrease with increasing distance, whereas peak velocity, acceleration, and inertial torque always increased.

The failure of the antagonist burst to demonstrate a clear positive correlation with peak decelerating torque appears inconsistent with arguments that rationalize such a relationship between the agonist burst and peak accelerating torque. Therefore the observation that as distance increases, peak inertial torque increases, but the antagonist does not, led us to consider whether changing muscle length might play a role here. Muscle length influences both its intrinsic force at a given degree of excitation (Hasan and Enoka 1985a; Rack and Westbury 1969; Ramsey and Street 1940) and also the EMG measured at constant force (Rack and Westbury 1969). Muscle length is not a factor for the agonist burst, irrespective of the task variable, for two reasons. First, as can be seen by inspection of Figs. 1–3, the agonist burst occurs under almost isometric conditions and is completed before the movement is completed. Second, even after movement begins, it takes an appreciable length of time before the trajectories of the different tasks diverge so that the agonist bursts occur at substantially the same agonist muscle length, independent of movement distance. However, the antagonist bursts occur at significantly different antagonist muscle lengths.

To try to correct for this effect, we adjusted $Q_{\text{dec}}$ with angle-dependent scaling factors. Figure 8 shows $Q_{\text{dec}}$ plotted versus movement distance both with no normalization factor and an angle-dependent normalization factor. These measures were on the lateral head of triceps. We have also plotted peak decelerating torque for comparison. When this adjustment is made to compensate for the effects of muscle length on force and EMG, we change a relationship in which the antagonist burst decreases with distance to one in which the normalized antagonist burst increased with distance. One can infer from this and from the positively covarying relationship between distance and decelerating torque, which can be seen in Figs. 5C and 8, that adjusted EMG, distance, and torque would all positively covary in deceleration as well as in acceleration.

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5 We noted in the METHODS that the normalization factor was computed from isometric measurements made at an elbow angle of 88°. To directly address this problem, however, for one subject part of the procedure was repeated at four other positions over a range of angles that included the initial joint angle and each target angle. This gave us five measures of EMG ($c_{\text{emg}}$) as a function of joint angle at the same torque ($T_{\text{sm}}$). We calculated linear regressions on angle to estimate the normalization scale factor for the target angles. (We could not measure these at the exact target angles because the manipulandum could only be clamped in place to perform isometric measurements at 15° intervals.)

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![Figure 7](image-url)  
**FIG. 7.** Integral of the antagonist burst over the duration of the movement ($Q_{\text{dec}}$) is plotted vs. peak inertial torque during the decelerating phase of the movement. The data are from the experiment in which 4 distances were moved against 4 inertial loads from the same subject in Figs. 2 and 3. Distances increase monotonically from left to right for 2 sets and as labeled for the other 2.

![Figure 8](image-url)  
**FIG. 8.** Measured antagonist EMG quantity ($Q_{\text{emg}}$), length-adjusted quantity, and peak decelerating inertial torque plotted against movement distance for an experiment in which a subject made 18, 36, 54, and 72° movements to a 9° target against the lightest inertial load combination. Units are millivolts per second for EMGs and Newtons per meter for torque. The data are for the subject in Fig. 1.
FIG. 9. Averaged (of 10) angle, velocity, acceleration, inertial torque, and EMGs for movements of 18, 36, 54, and 72° to a 9° target with a constant load. The EMGs from the flexors biceps and brachioradialis, and the extensors triceps lateral and triceps long, are shown after full-wave rectification and smoothing with a 25-ms moving average digital filter. This figure illustrates an experiment identical to that in Fig. 1. The behavior of the subject is similar to that in the earlier figure except for the 72° movement. The arrows on the inertial torque plot correspond to the torque generated during maximal voluntary contractions in each direction.

An exception

The final experiment to be illustrated was procedurally identical to the distance experiments illustrated in Fig. 1. This subject was experienced and skilled. Figure 9 shows "unusual" behavior. Movements made at the three shorter distances followed the same patterns demonstrated in previous experiments of this type: inertial torque and EMG rose at uniform rates. The observation that the 72° movement violated the "principle" of invariance for the rise of inertial torque and EMG was only made when the data was subsequently analyzed. Neither the experimenter nor the subject had noted anything unusual at the time of data collection nor complained of fatigue. The 72° movement was the last of the four movement sets performed. This experiment demonstrates that an invariant rate of rise (Qm) is not an obligatory feature of EMG or torque in movements of this type.

DISCUSSION

Many studies of single-joint movement have described relationships among task variables, kinematic-measured variables, and EMG-measured variables. Different investigators have used a variety of experimental manipulations of the task to induce changes in their dependent variables. In fact, all the results presented in Figs. 1–8 can be found in several places in the literature, although perhaps in different forms. Nevertheless, no coherent pattern or rule has yet emerged to organize these diverse results in a unified way (Hasan et al. 1985b). We will approach this problem by considering, in reverse order, the four principles with which we concluded the INTRODUCTION.

IV. Muscle torques interact with limb loads to generate kinematics (angle, its derivatives, and movement intervals). Because of the role of load in determining kinematics, no general correlations between EMG and purely kinematic measures are possible.

Figures 4–7 show that for any single experimental protocol, there are many strong correlations among variables, a finding well documented in the literature. Correlation does not demonstrate causality, however. Across changes in movement task (such as from distance to load), some of these relationships can change not only in magnitude but in sign. To go beyond description, we need prescriptive
rules to organize the experimental results into a framework based on causal relations between physical variables. Such attempts have been made at the kinetoc and kinematic levels (Hancock and Newell 1985; Meyer et al. 1982; Schmidt et al. 1979), but no theory yet comprehensively relates such principles to EMG, although a number of papers have presented relations between EMG and force-related parameters (Benecke et al. 1985; Bouisset et al. 1973, 1974, 1977, Gielen et al. 1985, Shapiro and Walter 1986).

Based upon physical principles, we expect that all movement kinematics should be explainable in terms of Newtonian laws for torques applied to viscoelastic-inertial loads. Fast unidirectional movements of the human elbow are largely dominated by the inertial component of these forces. This implies that the analysis of task-induced changes in kinematic parameters must be made in conjunction with the analysis of the underlying torques that produced those kinematics. Fig. 5, A and B, shows that peak inertial torque remains well correlated with peak velocity and movement time (and of course acceleration) in a manner which is entirely consistent with Newtonian mechanics.

One reason that torque has not often been used in a quantitative manner is that in the absence of an external load on the limb, joint torques are not measurable in humans. Even with external loading, only a portion of the net torque (the difference between agonists and antagonists) is measurable. The torque required to overcome intrinsic muscle viscoelasticity remains unmeasured. Figures 1–3 show that the inertial torques required to accelerate and decelerate the limb are an indirectly measurable component that can constitute an appreciable fraction of MVC, but they remain an unknown fraction of the total torque produced in any movement.

III. Rules for muscle activation lead to patterns of muscle torques and EMGs. Well-chosen scalar measures of torque and EMG will be highly and consistently correlated irrespective of task because of their shared causal, neural activation.

Agonist muscle

The activation of a muscle by its α-motoneurons produces both excitation (EMG) and contraction (force or torque). This shared causal input is the rationale behind attempts to use EMG as a predictor of muscle force (Agarwal and Gottlieb 1982; Basmajian and de Luca 1985). The most general method would use EMG as a function of time to predict (model) torque (Hof and van den Berg 1981) and angle (kilmer et al. 1982) as time series, but no such general (and competent) model exists. At a much simpler level, many studies have used some scalar quantity, such as the area or height of the agonist burst, to predict some scalar kinematic quantity, such as distance or movement time (Benecke et al. 1985; Brown and Cooke 1981; Wallace 1981).

We have chosen Qacc and peak inertial torque as our scalar measures of EMG and torque. The correspondence between them is shown in Fig. 4. This correlation is preferable to any that can be obtained from measures of angle, velocity or acceleration but it too has its shortcomings, particularly for the lightly loaded limb. These shortcomings may follow from the fact that it is plausible to suggest that Qacc should be better correlated with total muscle torque than with just the inertial component. It is also plausible to believe that when the inertial load is smallest, the inertial component is also the smallest fraction of the total, and under such conditions, it should provide the poorest measure of Qacc. It is not surprising, therefore, to observe in Fig. 4 that the movements made with added inertial load appear to be associated with relatively larger values of Qacc than movements made with added inertia. Although these experiments demonstrate that the correlation between EMG and peak inertial torque preserves its sign across manipulations of both task variables, they demonstrate the same for movement time (Fig. 5B). In the companion paper (Corcos et al. 1989), we describe experiments where the correlation between Qacc and movement time becomes negative whereas that between Qacc and inertial torque remains positive. Therefore the relation between Qacc and movement time is as general (but no more general) than that between it and peak velocity, distance, or load.

Antagonist muscle

The arguments for correlating Qacc with peak inertial torque during movement acceleration appear equally compelling (if that is the proper word) for correlating Qdec with peak inertial torque during movement deceleration. A similar argument for covarying antagonist EMG with torque-related measures, for movements with different inertial loads, has been made by Lestincn (1979), Meinek and colleagues (1984), as well as by Karst and Hasan (1987). In Fig. 1, Qacc is inversely related to movement distance. This observation appears consistent with most reports by others (Benecke et al. 1985; Brown and Cooke 1981; Cheron and Gofaux 1986; Marsden et al. 1983; Wadman et al. 1979) that show increasing distances can be accompanied by unchanging or decreasing antagonist bursts. More important, however, is the negative covariation that exists between peak decelerating torque and Qdec.

It appears inconsistent to argue that the activation of the antagonist muscle provides, and is proportional to, braking torque when movements with different loads are performed and abandon this principle for movements of different distances. We considered the possibility that this apparent inconsistency in the relationship between Qacc and

Karst and Hasan (1987) observed that the antagonist EMG can be related to average braking torque \( (V^2/D) \), \( V = \) peak movement velocity, \( I = \) moment of inertia, and \( D = \) distance moved. This makes excellent physical sense and is similar to the relationships between EMG and peak accelerating torque for the agonist burst. Their experiments were not designed to explore the situation in which both distance and acceleration covaried, and it is unclear how well their data agree with their equation when distance is increased and speed increases with distance. They only examined movements of two different distances (15 and 30°), which clearly showed that movements of different distances at the same peak speed (requiring average acceleration to scale inversely with distance) obeyed the rule. However, in our experiments, average velocity increased with distance, and if one substitutes this into the equation, the result is that torque is proportional to \( ID \), which is consistent with our measurement of peak decelerating torque.
distance (when torque is increasing) was a consequence of the changing relations between EMG and torque when muscles shorten and lengthen (Bigland and Lippold 1954) and that eccentric contractions generate larger forces than concentric contractions (Gravel et al. 1987). There are many possible ways to use information about the relationship between EMG and torque at different muscle lengths. Although it is reasonable that such information should be exploited, the best way to do this is not obvious.

We chose, for simplicity, to adjust each antagonist EMG measure by multiplying $Q_{\text{acc}}$ by the scaling normalization factor appropriate for the final target angle. The effects of this procedure are shown in Fig. 8. The results suggest that the effective activation of the antagonist muscle, if not the actual EMG voltage measured, remains positively correlated with peak inertial torque for movement deceleration.

This explanation does not completely resolve the problem that $Q_{\text{acc}}$ increases with load but peak inertial decelerating torque does not (see Figs. 3 and 7). In fact, it can be inferred from Fig. 5C that with increases in inertial load, peak decelerating torque is not well correlated with peak accelerating torque. Were we to use a measure dependent upon peak velocity [such as the average braking torque $IV^2/D$ (Karst and Hasan 1987) or maximum kinetic energy $IV^2/2$ (Bouisset and Goubel 1973)], proportionality would be preserved. If we wish to use a torque based measure, then impulse [integral of torque $\int \tau(t)dt$] might be a better candidate than peak torque.

The more fundamental problem is that any correlation between scalar measures of performance (in this context, that is a measure that summarizes time-varying behavior by a single number) is probably bound to fail for some kinds of movements. Such correlations are major simplifications of relations that require sets of nonlinear differential equations rather than algebraic ones. They are useful for summarizing complex behaviors but must be interpreted most cautiously when trying to deduce causal physical or physiological relations from them.

II. Strategies consist of sets of rules that determine the patterns of muscle activation.

Having described the data and observed what we think are the causal relations from task to EMG and torque and from there to kinematics, we can proceed to deduce possible rules by which the nervous system chooses to accomplish the assigned movement tasks.

Control of muscle contraction

It is impossible to directly control muscle force or joint torque, but only to control some higher-level variables which, penultimate, excite motoneurons, activate muscles, and only then influence torque. In the experiments described here, subjects were asked to move as fast as the load, the distance, and the target size allowed. In all cases, changing the load or the distance resulted in changes of peak inertial torque, acceleration, velocity, and movement time.

What is of significance is that subjects always chose to increase their initial, accelerating torques in the same manner, even when the load or the distance changed (Wadman et al. 1979). This is revealed by the uniformity of the rising inertial torque curves in Figs. 1–3 and for the three shorter distances in Fig. 9. The longest distance in Fig. 9 demonstrates how early torque curves can be differentiated when the subject uses a different rule (Corcos et al. 1989; Gottlieb et al. 1989). Torque curves rise together for 50–100 ms before diverging. Peak movement velocity increases when torque continues to increase for a longer interval of time, but the initial rate of increase is unaffected by changes in the magnitude of the task variable.

Control of muscle activation

The pattern of force development is a consequence of the way muscle is activated. The way muscles are activated can be inferred from the EMG records. Irrespective of distance or inertial load, the agonist EMG bursts rose at similar rates. In Fig. 1, they rose for variable intervals depending on the distance, and therefore measures of areas ($Q_{\text{acc}}$) were highly correlated to the peak inertial torque of the movement. In Figs. 2 and 3, biceps brachii EMGs rose to similar plateaus at which they remained for variable intervals. Measures of $Q_{\text{acc}}$ were equally highly correlated to peak inertial torque. The inertial torque, in turn, was positively correlated with inertial load and movement distance.

This difference between Fig. 1 and Figs. 2–3 could suggest that the former is an example of EMG pulse height modulation (Ghez 1979), whereas the latter two are examples of EMG pulse duration modulation (Angel 1974; Berardelli et al. 1984). It is possible by means of a simple model to show how these may both be a manifestation of a single control rule.

Let us suppose that we can meaningfully characterize the total instantaneous input to the motoneuron pool by a single signal, "excitation," to reflect a weighted algebraic sum of all depolarizing and hyperpolarizing synaptic inputs to the motoneurons. Excitation leads to depolarization and the motoneuron pool acts as a low-pass filter, summing, smoothing, and attenuating its many converging inputs (Knox 1974; Stein et al. 1974). A low-pass filter relation between excitation $[N(t)]$ and the mean depolarization of the motoneuron pool $[e(t)]$ is

$$\frac{de(t)}{dt} + \frac{1}{a} e(t) = N(t)$$

(2)

The rate of discharge from the pool $[f(t)]$ will be proportional to $e(t)$ but is bounded because it cannot be negative nor exceed some maximum rate of discharge in a neuronal population $[0 < f(t) < F_{\text{max}}]$. This is illustrated in Fig. 10. From simulation studies (Agarwal and Gottlieb 1978; Moore 1967; Person and Libkind 1967), we expect that the rectified, filtered EMG [EMG(t)] will be a monotonically increasing function of $f(t)$. A model of the relation between the excitation pulse $[N(t)]$ and the electromyographic signal EMG(t) is described by the following equations.

---

3 The assumption of a linear relation is not vital but is used for simplicity. The assumption of only first-order dynamics is also not essential but the assumption of at least first-order dynamics is. Knox (1974) derives Eq 2 with $a = 8$ ms. A review of other more complex models can be found in Agarwal and Gottlieb (1982).

We are neglecting other dynamic processes that lie between the motoneuron and the EMG, such as the filtering effects of the tissue (Basmajian...
FIG. 10. This block diagram characterizes the motoneuron pool as a low-pass filter, followed by a limiter that restricts the number of impulses that can be discharged from the pool to some maximum value. The input to the diagram is the net presynaptic excitation to the pool \( N(t) \). Postsynaptic excitation \( e(t) \) is transformed directly into a composite action potential train in the motor nerve \( f(t) \), the output after amplitude limiting. This train is the source of both the externally measurable consequence of motoneuron activation (the EMG) and muscle contraction.

\[
f(t) = 0, \quad e(t) = 0, \quad N(t) = 0 \quad t < 0
\]

\( e(t) \) is described by Eq. 2 for \( t > 0 \).
\[
f(t) = e(t) \quad 0 \leq e(t) < F_{\text{max}}
\]
\[
f(t) = F_{\text{max}} \quad e(t) > F_{\text{max}}
\]
\[
\text{EMG}(t) = f(t)
\]

If we let \( N(t) \) be a rectangular pulse, this model gives rise to predictions of what the rectified EMG should look like. For constant-amplitude pulses, EMG area will scale with pulse duration, whereas the peak EMG will scale with duration up to \( F_{\text{max}} \). Figure 11A illustrates this. Note the uniform rising phases and that the duration of the output waveform appears proportional to the duration of the input. By way of contrast, allowing the amplitude of \( N(t) \) to change while keeping its duration constant causes EMGs to rise at different rates to different peaks before falling. Again, peak EMG will reach the limiting rate if the filter output is large enough. This is illustrated by solutions of Eqs. 2–6 in Fig. 11B. The duration of the output waveform might be estimated to be constant in this simulation, but this is arguable as we have previously discussed (Gottlieb et al. 1989). The integrated EMG always increases when either of the pulse parameters is increased. The peak EMG is only proportional to both parameters until limiting occurs.

If the motor control system specifies the width of the excitation pulse\(^8\) based upon the magnitude of the task variable, be it distance or load, then the model demonstrates that an integrated EMG quantity will correlate with pulse width and therefore with the magnitude of the manipulated task variable. The model also demonstrates that integrating the EMG over only its rising phase, such as we do for \( Q_{30} \), will be insensitive to the task variable. This behavior can be compared to the data in Fig. 4 that show task dependent \( Q_{\text{acc}} \) and task independent \( Q_{30} \).

One consequence of the low-pass filtering is that increases in neither peak nor integrated EMG uniquely define the controlling parameter of the pulse. Even when pulse duration is the independently controlled variable, the model shows how EMGs which show amplitude modulation with distance (Fig. 1) and those which do not (Fig. 2) can both arise. Similar observations have been made for different loads.

\( F_{\text{max}} \) is the intensity of the excitation pulse, \( a \) and \( W \) are the amplitude and width of the rectangular pulse, respectively. The time constant for equation is \( a \), the intensity of the excitation pulse is \( A \), and \( W \) is its width.

\( * \) It is not necessary that the higher centers have total control of the excitation of the motoneuron pool. There are multiple signals converging at the anterior horn, including afferent ones, which clearly depend in part on external factors. However, for the classes of movement with which we are concerned (series of discrete movements with no unpredictable perturbations), afferent signals are probably highly predictable. Therefore, if afferent input is significant, then their contribution can be corrected for in the generation of the descending signals to obtain the required degree of motoneuron activation.

\( 8 \) It is not necessary that the higher centers have total control of the excitation of the motoneuron pool. There are multiple signals converging at the anterior horn, including afferent ones, which clearly depend in part on external factors. However, for the classes of movement with which we are concerned (series of discrete movements with no unpredictable perturbations), afferent signals are probably highly predictable. Therefore, if afferent input is significant, then their contribution can be corrected for in the generation of the descending signals to obtain the required degree of motoneuron activation.

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FIG. 11. Equations 2–4 were solved on a microcomputer (Macintosh) using a general purpose equation solver (EZQ). A: rectangular pulses of the same amplitude but different durations were used as inputs. B: rectangular pulses of equal durations but different amplitudes were used. The model output represents the rectified EMG. The time constant for equation is \( a \), the intensity of the excitation pulse is \( A \), and \( W \) is its width.
Whether the antagonist muscle is regulated by rules identical to those for the agonist, but with an appropriate temporal delay, is not as clearly answered by the present data. Visual inspection of Figs. 1–3 and 9 shows that the early component of the antagonist is insensitive to the magnitude of the task variable and that the rising phase of the antagonist burst of the lateral head of triceps is also task insensitive. The long head, however, shows rising antagonist bursts with decreasing slopes for later onsets. Later onsets imply that the contraction occurs at longer antagonist muscle lengths, and applying an appropriate length dependent correction factor would make the slopes more uniform. Such a transformation would be analogous to that performed on Qdec in Fig. 8 to show that it could be interpreted as an indicator of antagonist muscle activation proportional to decelerating torque. We take it as obvious that there are sufficient numerical degrees of freedom in such a procedure to enable us to demonstrate the relationship we would like and too many to prove it. As a reasonable simplifying hypothesis, we would argue that agonist and antagonist muscles are controlled by similar rules.

1. Elements of a movement task lead to a strategy governing its control. We have identified two strategies to describe how single-joint movements are accomplished for a variety of tasks.

Our model suggests that the control of simple movements can be described in terms of an excitation pulse of adjustable width and height. The processing of such a pulse through appropriate filtering mechanisms can yield predictions for both muscle excitation, as reflected in the EMG and muscle contraction, as measured by joint inertial torque.

Strategy

We can characterize the rules by which the nervous system sets control parameters that ultimately determine movement kinematics as a strategy. That is, a strategy is a set of rules that specify, in terms of task variables and subject instructions, how to compute each parameter of the excitation pulse. The data presented here describe behavior that can be characterized by a single strategy, used to make accurate, fast movements to targets of different distances or with different inertial loads. We call this a speed-insensitive (SI) strategy.

The SI strategy adjusts muscle activation to the specific elements of a task by modulating only the durations and latencies of pulses of motoneuron excitation as diagrammed in Fig. 12. The amplitudes of those pulses are independent of changes in the task variables, although they clearly must be selected initially according to some consideration based upon the entirety of the intended movement. Once the amplitudes are selected, however, they remain invariant over changes of task variables such as movement distance or inertial load.

An SI strategy causes the initial excitation to the motoneuron pool to be insensitive to the speed at which the movement is performed. As a result, the initial rise in contractile forces are task insensitive. This implies that once the motor system has selected an acceptable level of excitation, different distances and load requirements are met by changing only the width of the agonist and antagonist pulses (W_{ag} and W_{ant}) and the latencies of the early antagonist activation (L_{ant}). For movements with this strategy, L_{ant} is relatively constant, and the others are all positively correlated with each other.\footnote{The initial excitation is insensitive to many things in addition to speed, such as distance or load. An alternative strategy which can meaningfully be called "speed sensitive" is described in the companion paper (Corcos et al. 1989, see also Gottlieb et al. 1989) and has motivated the present appellation.}

Rules

The foregoing discussion can be summarized into a set of rules which describe how subjects make movements under an SI strategy.

\textbf{SI RULE FOR THE EXCITATION PULSE.} Intensity is constant and duration is modulated.

\textbf{SI RULE FOR THE EMG.} The initial pattern of motoneuron pool discharge is independent of the magnitude of the task variable. This generates EMGs which rise at the same rate, irrespective of changes in distance or load. Changes in the task affect the area and duration of the EMG burst.

\textbf{SI RULE FOR KINEMATICS.} Because initial motoneuron pool activation is insensitive to the magnitude of the task variable, initial muscle force is also unaffected. For con-
stant inertial loads, this is reflected in constant initial rates of acceleration. For different inertial loads, acceleration scales inversely with load.

The first of these rules defines the way the nervous system implements an SI strategy at the level of the motoneuron pool. It does not specify how such an excitation pulse is generated (e.g., central programming or use of peripheral feedback) but only what parameters are modulated (see Gottlieb et al. 1989, reply to commentaries for a discussion of this issue). The second rule is a consequence of the first, combined with the postulated low-pass filtering effects of the motoneuron pool. The third rule is also a consequence of the first, based upon physiological considerations of muscle excitation-contraction coupling mechanisms.

Correspondence between theory and data

Our model makes several specific predictions about the amplitude and timing of the EMG peaks that are not as strongly supported by the data presented here as are the predictions concerning area and initial slope. This may be a shortcoming of our simple rectangular model of the excitation pulse or a result of the normal variance in EMG signals that has not been reduced by averaging a large enough number of movements.

Another shortcoming of the model is found in Fig. 9. It illustrates a subject who, by our definition of strategy, appears to have changed strategies for the 72° movement, a change not noticed at the time of the experiment. This experiment is of interest because it demonstrates a qualitative change in behavior which is not captured by examining changes in peak velocity, \( Q_{\text{acc}} \), or movement time, all of which increase for all four distances. It is captured by the quantitative measure of \( Q_{\text{acc}} \), which should be invariant over the four distances but is not, and initial rise of inertial torque, which should also be invariant but is not.

Why the subject changed strategies is unknown. We would exclude physiological fatigue since that should be associated with a change in the relationship between the EMG and force production (Bigland-Ritchie 1981). Perhaps it represents “psychological” fatigue. For this longest movement, on the last series of the experiment, the subject simply (and unconsciously) did not try as hard and slowed down. Perhaps it represents lability in the nature of human performance. Without highly specific forms of feedback or specific and extensive training, subjects may be unable to continue to reproduce certain patterns of behavior consistently.

Having concluded that subjects may fail to sustain a uniform strategy across an experimental session, we can look more carefully at the data in Figs. 1–3. Figure 1 shows evidence in both agonist EMGs that a smaller excitation pulse was used to perform the 18° movements. The lesser slope of the agonist burst is followed by a similar reduction in the rise of inertial torque. Similar observations can be made concerning the movements of the heaviest inertial load in Fig. 3. These deviations from the proposed stereotyped strategy are small compared to those in Fig. 9 and were ignored in our initial discussion of those data.

This imperfect match between the theoretical model of how movements are performed and our data could be interpreted as a falsification of the theory. We prefer to interpret it as an expression of normal variability, inescapably associated with human behavior (Meyer et al. 1988). The consistency with which subjects could sustain a uniform strategy ranged from excellent to poor, depending upon factors that we do not understand, but was never perfect. Part of the utility of a theory arises not just from the accuracy with which it predicts behavior but also from its provision of a norm from which deviations can be detected.

It is fair to ask whether an SI strategy uses a constant intensity excitation pulse by choice or because, for these “accurate and fast” movements, the driving signal is maximal and cannot be further increased (cf Gottlieb et al. 1989 commentaries and reply). It is our opinion that the constancy of the excitation pulse represents a choice and not a saturation effect. The data presented here do not prove or disprove this. Further experiments will be required to demonstrate it.

On nomenclature

The names speed-sensitive and speed-insensitive have the following rationale. In almost any circumstance, if a single task variable is experimentally manipulated, movement speed (peak and mean) monotonically covaries. The exceptions are experiments which are deliberately designed to control speed in some contrary manner. It is clear that although speed changes, this only sometimes occurs to satisfy task requirements. For example, making movements of a fixed distance at different speeds or of different distances in a fixed time both require speed control. Similarly, because of the speed-accuracy trade-off expressed by Fitts' Law (Fitts et al. 1954, 1964), changing the size of the target also requires speed control.

In contrast, changing distance or inertial load does not require speed control. One can make arguments, based on physical principles, that if such movements involve maximal activation of the muscles, changes in speed are unavoidably linked to changes in the task variable; but in this case, an additional level of analysis is required. Further, if maximal activation is not used, variations in speed are completely a matter of choice or strategy. The terms therefore imply that sometimes subjects use strategies that vary speed because of task requirements and are therefore speed-sensitive, whereas other times subjects use strategies that vary speed, although it is not necessary to satisfy task requirements, and are therefore speed-insensitive.

Concluding remarks

We began this discussion by observing that our data are not novel but rather are typical of many obtained in other laboratories that have performed similar experiments. The novelty provided here is in the forms of analysis that organize and allow us to interpret data found both here and in the literature in a more meaningful way. The rules for a speed-insensitive strategy have been found to hold across two different task manipulations. In the following paper, we will describe another class of movements in which this
strategy is not used and an alternative, speed-sensitive strategy is employed.

APPENDIX ANGEL

Estimating EMG slopes

The measure \( Q_0 \) provides a way of estimating the slope of the rising phase of the EMG. Such a method has the advantage of being relatively insensitive to the stochastic variability of the EMG. The inherent variability of the EMG makes an estimate of slope based on simple differences unreliable. The method is explained by the following geometric analysis. We assume that the rising phase of the EMG can be reasonably approximated for a short period of time by

\[
\text{emg}(t) = a^t \tag{A1}
\]

where \( a \) is an unknown constant and the exponent \( n \geq 0 \). Then the integrated EMG over the interval \( T/Q_0 \) is given by

\[
Q_T = a \int_0^T t \, dt = a \frac{1}{n+1} T^{n+1} \tag{A2}
\]

Solving for \( a \)

\[
a = \frac{n+1}{T^{n+1} Q_T} \tag{A3}
\]

Because for all values of \( n > 1 \), the slope of the EMG is continuously changing with \( t \), we want to define an average or representative value of the slope over the interval. The simplest is the slope of the chord between the two values of EMG at \( t = 0 \) and \( t = T \). Hence

\[
\text{chord slope} = \frac{\text{emg}(T) - \text{emg}(0)}{T} = a T^{n-1} \tag{A4}
\]

Substituting our computed value of \( a \) (Eq A3) into Eq A4 gives

\[
\text{chord slope} = \frac{n+1}{T^{n+1}} Q_T \tag{A5}
\]

Thus, we can use the integral of the recorded EMG over a short interval \( T \) to estimate the average slope. This estimate is directly proportional to the integrated EMG, irrespective of the order of the power function used to derive the equation. A similar conclusion can be drawn for various other choices of average slope. No one choice stands as obviously better, and so we will use the simplest.

In presenting our data, we show \( Q_T \), where \( T = 30 \) ms. This interval represents a compromise, arrived at by trial and error, between competing factors. Increasing \( T \) so that it intrudes into the interval when the EMGs start to diverge would make \( Q_T \) look progressively more like \( Q_{\text{acc}} \). Shortening \( T \) would make the measure describe very little of the recorded data. The interval of uniform slope is task dependent, and we chose the longest interval brief enough to include movements of the shortest distances and lightest loads which had the briefest excitation pulses.

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