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RESEARCH ARTICLE

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# Nonlinear control of movement distance at the human elbow

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**Abstract** The kinematic, kinetic, and electromyographic (EMG) patterns observed during fast, single-joint flexion movement have been widely studied as a paradigm for understanding voluntary movement. Several patterns have been described that depend upon the movement task (e.g., distance, speed, and load). A previous model that interpreted differences in EMG patterns in terms of pulse-height or pulse-width modulation of rectangular pulses of motoneuron pool excitation cannot explain all the EMG patterns reported in the literature. We proposed a more general version of that model, consisting of a set of four equations, which specify the parameters of the excitation pulses for a wide variety of movement tasks. Here we report experiments in which subjects performed fast elbow flexions over a range of distances from 2.8° to 45°. The EMG patterns that we observe are consistent with this more general model. We conclude that this model is sufficient to specify muscle excitation patterns that will launch a movement toward and stop it in the neighborhood of a target. This model operates on the basis of prior knowledge about the task rather than feedback received during the task.

**Key words** Voluntary movement · Force control model · EMG · Movement planning

## Introduction

The control of a task even as simple as flexing a single joint from one stationary position to another has at various times been described as being controlled by different rules, depending on how certain aspects of that task are defined. For example, many movements of the elbow show uniform rates of rise of joint torque and of agonist electromyographic (EMG) bursts. For such movements, the duration of the accelerating torque and the duration of the EMG burst covary. Other movements show varying rates of rise of joint torque and agonist EMG bursts that have constant durations. These patterns were described in terms of two strategies for muscle activation termed speed-insensitive (SI) and speed-sensitive (SS), respectively (Corcos et al. 1989, 1990; Gottlieb et al. 1989a, 1990, 1992). The patterns of motoneuron pool excitation were assumed to be rectangular in shape ("excitation pulses") and the EMG patterns that emerged were low-pass filtered versions of those pulses. The SI strategy covaried the duration of muscle excitation and the latency of the antagonist muscle burst to control movement distance or adjust to known changes in the external inertial load (Gottlieb et al. 1989a). The SS strategy modulated the intensity of muscle excitation to scale movement speed while inversely scaling antagonist latency (Corcos et al. 1989). Even taken together, the two strategies are not sufficient to describe how all single-joint flexion movements are controlled, such as those that are performed under certain temporal constraints (Gottlieb et al 1989b, 1995). These strategies also fail to account for the kinematic and EMG patterns of a series of seemingly SI-type tasks performed at the wrist (Hoffman and Strick 1986, 1990, 1993) and the finger (Freund and Budingen 1978). These studies used movement distances of 5–25° and showed approximately constant movement time (MT), although that was not an explicit part of the instructed task. This differs from the SI pattern in which MT covaries with distance. In the study by Hoffman and Strick (1993), the initial EMG slopes increased with movement distance, which is also in contrast to the SI pattern.

Hoffman et al. (1990) and Hoffman and Strick (1993) suggested that the central nervous system (CNS) changes the patterns of muscle activation because of the need to

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provide task-appropriate forces with muscles that have physiological constraints on their force-producing mechanisms. Two mechanisms used by the nervous system for developing larger amounts of force are to increase the number of activated motor units (recruitment) and to develop more force in each active motor unit by modulating the intervals between successive action potentials (frequency modulation; Stein and Parmiggiani 1979). Both of these correspond to height modulation of the excitation pulses. For the transient forces that are used to produce relatively fast, phasic movements, a third mechanism is to increase the duration of the action potential train in the set of active motor units (width modulation of the excitation pulses) and rely on simple twitch summation to raise the force levels. As originally described, SI and SS strategies corresponded to pulse-width and pulse-height modulation of agonist excitation pulses, respectively. We have argued that there is no evidence that this choice of pulse-width and pulse-height modulation is imposed upon the CNS by biomechanical, neural, or computational constraints. Rather, this represents a choice of control strategies in the sense that other patterns of modulation could perform very similar movements. This pattern is adequate to satisfy all the explicit (e.g., distance, load) and implicit (e.g., movement time, energy conservation, comfort, etc.) criteria with their unknown but certainly varying relative importance. In the absence of evidence that this is an imposed strategy, this is the arbitrary, default strategy (Gottlieb et al. 1990). It can be changed by the addition of constraints on the task such as requiring the subjects to simultaneously satisfy specific distance and MT requirements (Gottlieb et al. 1995). We can also manipulate the task to exploit some of the physiological properties of the neuromuscular system that act as constraints.

The experiments in this manuscript explore this latter condition. As movements get smaller, the needed forces get smaller and briefer. A pulse-width modulation strategy will be ineffective in reducing contraction force if the desired duration of the acceleration phase of the movement approaches the duration of a muscle twitch because further reduction of the excitation pulse width cannot further reduce the contraction duration. When this happens, further reduction in pulse width ceases to be mechanically effective and the nervous system must switch to some other control scheme such as pulse-height modulation to reduce contraction strength. This would be expressed by a change in EMG and torque patterns from those described as SI patterns of agonist modulation for longer movements to patterns similar (but not identical) to those of SS for shorter movements. Some evidence of this switch can be seen for the shortest movements at the elbow (Gielen et al. 1985, Fig. 8; Gottlieb et al. 1989a, Fig. 1, 1990, Fig. 1b,c) and at the wrist (Hoffman and Strick 1990, 1993).

These considerations lead to a model (Gottlieb 1993) of how the excitation pulses of the agonist and antagonist muscles are modulated under task conditions that include moving with different distance, inertial load, speed, and accuracy requirements. The mathematical expression of the model (described below) allows us to calculate specific quantitative features of the EMG patterns that depend upon parameters of the task. The experiments described here present the results of experiments that apply this model to data collected over a range of distances sufficiently wide to demonstrate its predicted nonlinear features and to also show how experiments with seemingly incompatible results could be the consequence of being performed over different parts of that nonlinear range.

## Materials and methods

### Data collection

Informed consent was obtained from eight neurologically normal male subjects between the ages of 20 and 30 years according to Medical Center-approved protocols before they participated in this study. Seated subjects abducted the right shoulder 90° and rested the forearm on a lightweight, horizontal manipulandum (moment of inertia 0.06 $Nm.s^2/rad$) that allowed free rotation about the elbow. They viewed a computer monitor that displayed a cursor, the horizontal location of which was determined by the angle of the elbow. The origin was defined with the forearm and upper arm forming a right angle, and flexion was positive. A narrow marker on the screen specified a starting position for the limb at 35° extension from the origin. A second marker (3.75° wide) was a target, centered at the desired final angular position.

An audio tone was delivered about every 8 s, signaling the subject to make an elbow flexion movement from the starting position to the target. Subjects remained on the target for about 2 s until the tone ended and then returned to the starting position. Movements were performed over seven different distances (2.8°, 5.6°, 8.4°, 11.25°, 22.5°, 33.75°, and 45°). The sequence of distances given to the subject was mixed (11.25°, 33.75°, 2.8°, 8.4°, 22.5°, 45°, and 5.6°). No special point was made to the subjects that we were investigating possible differences between "small" and "large" movements. The instructed task was to move "as fast and accurately as possible" to the target. Movements were performed at each distance 15 times and the first five trials automatically rejected from analysis. In addition, trials were rejected in which the peak movement displacement exceeded or was less than the adjacent target distance. This amounted to rejection of up to three trials for movements shorter than 12° and one trial for the longer movements.

Joint angle and acceleration were transduced and low-pass filtered at 30 Hz. Joint velocity was computed. EMG surface electrodes (pairs of pediatric electrocardiographic electrodes with 2 cm between centers) were placed over the bellies of the biceps brachii, brachioradialis, and triceps (lateral and long heads) muscles. EMGs were amplified (×2000) and band-pass filtered (60–500 Hz). All signals were digitized with 12-bit resolution at a rate of 1000/s. These methods are described in greater detail by Gottlieb et al. (1989a).

### Data analysis

EMG time series in Fig. 1 were normalized with respect to the maximum voluntary contraction (MVC) of the subject by dividing the EMG by the mean of a maximal isometric contraction (Corcos et al. 1993). Quantification of specific features of the EMG patterns was done with four measures, made on each individual record. The area of the agonist burst ($Q_{ag}$) was computed by integrating the rectified EMG from the first sustained rise of the EMG signal above base line to the time of peak velocity. The area of the antagonist burst ($Q_{ant}$) was computed over the full MT (Gottlieb et

al. 1989a). The rate of rise of the agonist EMG burst was estimated by integrating it over the first 30 ms ($Q_{30}$). We measured the latency ($T_{ant}$) of the antagonist burst by displaying the highly amplified antagonist EMG signal on a computer monitor. It was first full-wave rectified and smoothed with a 10-ms rectangular averaging window. The antagonist, almost always silent prior to movement onset, showed its earliest activity 20–30 ms after the agonist. What we refer to as the "burst," was visually identified as a sharp increase in this activity that took place 50–200 ms later (e.g., shortly after $t$=150 ms in Fig. 1A). If we could not identify such an event (which occurred in no more than three records per block), the record was rejected from further analysis.

### Model of excitation pulses and resultant EMG

The fundamental assumption of our model is that control of fast, single-joint movements is exercised by a combination of pulse-height and pulse-width modulation of rectangular pulses of excitation to the motoneuron pools of the agonist and antagonist muscles. From correctly planned excitation inputs, muscle force develops and, ultimately, the intended movement emerges as the interaction of the muscles with the mechanical load. This is functionally equivalent to having an inverse dynamical model of the limb and load, but such a model is only implicit in the rules for determining the pulse parameters rather than explicitly defined by dynamical equations. The model was originally defined by Gottlieb (1993) in terms of four task-specific variables (distance, load, speed, and accuracy) to compute the pulse parameters: i.e., agonist and antagonist heights and widths and antagonist latency. In the present study, only movement distance is an independent variable.

To test the model, predictions of measurable variables are required, and we proposed four features of the EMG patterns as estimators of the task-dependent but unobservable excitation pulses. The EMG patterns are assumed to be low-pass filtered views of the pulses. Hence, the areas of the EMG agonist and antagonist bursts ($Q_{ag}$, $Q_{ant}$) are assumed proportional to the areas of the pulses (i.e., the products of their heights, $H$, and widths, $W$). The rate of rise of the agonist burst (estimated by the area under the first 30 ms of the agonist EMG burst, $Q_{30}$) is assumed to be proportional to pulse height and the latency of the antagonist EMG burst is assumed to be proportional to the latency of the antagonist pulse. We also assume that the widths of the two pulses are the same.

In accordance with the proposed pulse model, we used the following four equations to describe the measurable variables in terms of pulse heights, widths, and latencies.

$$Q_{30} \propto H_{ag} = a_1 \cdot \left(1 - e^{\frac{-\theta}{\theta_{ag}}}\right) \tag{1}$$

$$Q_{ag} \propto WH_{ag} = a_2 \cdot \left(1 + \frac{\theta}{W_{min}}\right) \cdot \left(1 - e^{\frac{-\theta}{\theta_{ag}}}\right) \tag{2}$$

$$Q_{ant} \propto WH_{ant} = a_3 \cdot \left(1 + \frac{\theta}{W_{min}}\right) \cdot \left(1 - e^{\frac{-\theta}{\theta_{ant}}}\right) \cdot e^{\frac{-\theta}{\theta_1}} \tag{3}$$

$$T_{ant} = T_0 + K\theta \tag{4}$$

The coefficients were determined by fitting the equations to the data by least-squares methods [the Levenberg-Marquardt algorithm (Press et al. 1992)] with KaleidaGraph (Synergy Software), a commercial program for the Macintosh computer. The independent variable in all four equations for the experiments reported here is movement distance ($\theta$). All other symbols ($a_i$, $W_{min}$, $K$, $T_0$, $\theta_{ag}$, $\theta_{ant}$, $\theta_1$) are constants with the following interpretations:

1. $a_i$ are constants of scale.
2. $W_{min}$ represents the theoretical minimum excitation pulse width. EMG burst durations are difficult to quantify because of the low-pass filtering properties of the neuromuscular system.
3. $\theta_{ag}$, $\theta_{ant}$ characterize the transition distances at which pulse height gradually changes from distance proportionality to distance invariance.
4. $\theta_1$ is a scale factor for the rate at which the antagonist EMG burst will decrease as a function of movement distance.

These equations are simplified forms of the more general case for different inertial loads, and in which explicit control of movement speed and accuracy (the SS strategy) is included by changing some of those constants to functions of the additional task variables.[1]

Below we will describe our experimental results for elbow flexion movements over different distances and show the degree to which the above equations can account for the results. We will defer the rationale for choosing these equations to the Discussion.

## Results

Figure 1 shows the kinematics and EMG time series for one subject. Short movements (2.8–11.25°) are on the left (Fig. 1A) and long (11.25–45°) on the right (Fig. 1B). Note that the ordinate scales of the two parts are different and that the 11.25° movement is repeated in both parts of the Fig. as the longest distance on the left and the shortest on the right.

Longer distance movements (Fig. 1B) demonstrate all the defining features of the SI strategy that are seen with increasing movement distance (Gottlieb et al. 1989a,b, 1990). In the EMG records: (1) the agonist bursts rise together for almost 50 ms; (2) they fall off earlier for shorter movements and later for longer movements; (3) the areas and peaks of the biceps bursts scale with distance; (4) the antagonist bursts start progressively later with distance; but (5) show no clear trend for their area, a common finding for movements over this range of movement distances with a light manipulandum. The kinematics for the long movements are also typical of the SI pattern. Peak velocities (PVs) and accelerations as well as MTs increase with distance. The initial uniform activation of the agonist muscle produces muscle torques (acceleration multiplied by moment of inertia) that rise at uniform rates that are independent of distance for almost 75 ms after the initial activation of the agonist muscles. For these movement conditions, that is equivalent to uniformly rising rates of acceleration.

The left side of Fig. 1 illustrates the shorter distance movements. Several visually distinguishable differences can be found between movements in the left and right sides of this figure: (1) the slopes of the rising phases of the agonist EMG bursts and of the initial accelerations are not the same on the left but vary with distance; (2) differences in agonist burst durations can not be distinguished (Berardelli et al. 1984); (3) the areas of the antagonist bursts clearly increase with distance but differences in their onsets are difficult to distinguish; and (4) MTs are approximately constant.

[1] These equations also apply to the intentional control of movement speed according to the SS strategy. To do so, coefficients $a_1$, $a_2$, and $a_3$ scale directly with $K$ and inversely with intended speed. Data supporting this for movements of 18° and greater have already been published by Corcos et al. (1989; see Fig. 2 for $a_1$ and $a_2$, Fig. 5 for $a_3$ and Fig. 4 for $T$) and by Gottlieb.et al. (1990; see Fig. 3 for $a_1$ and $a_2$ and Fig. 3 for $a_3$).

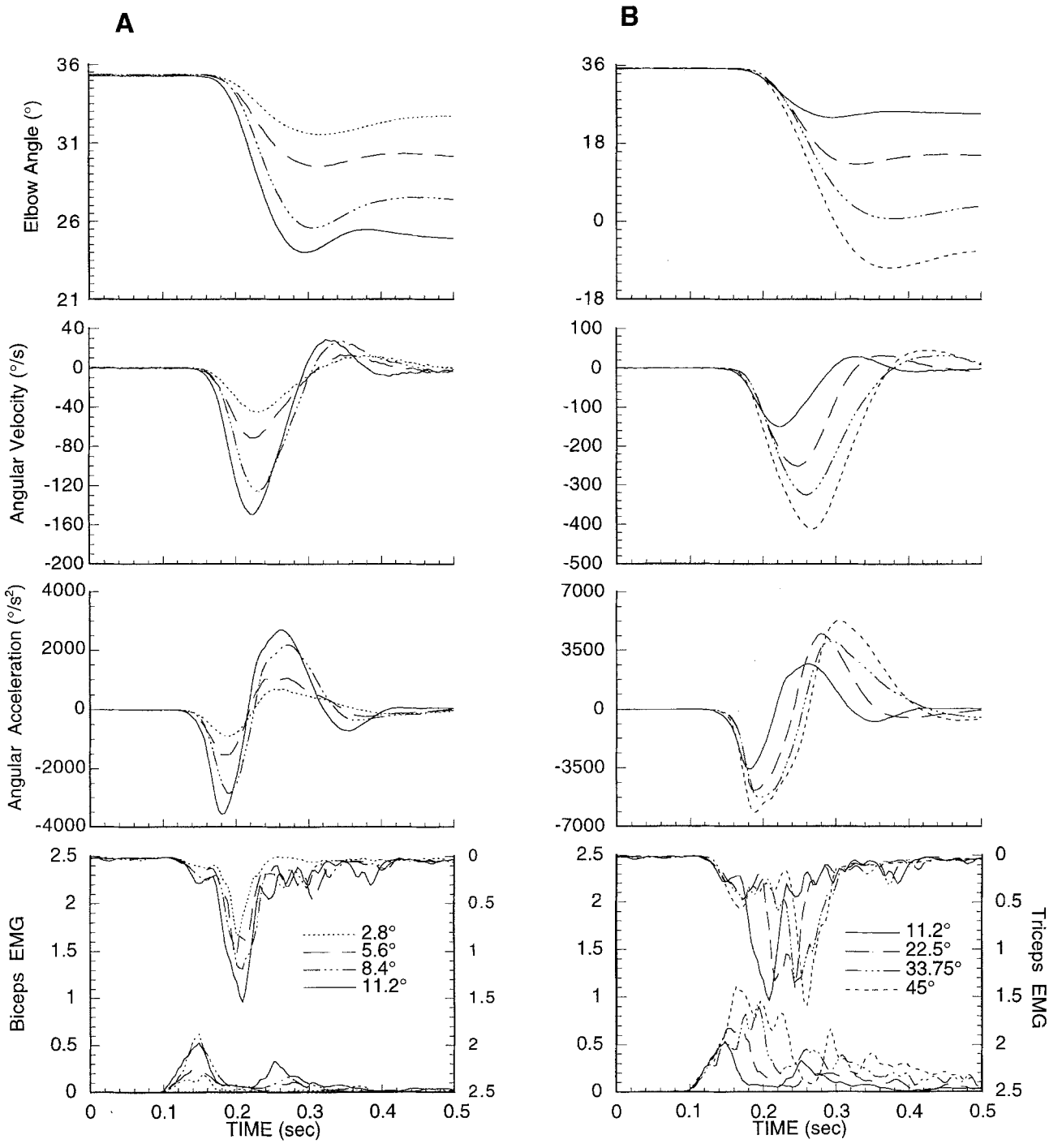


**Fig. 1A, B** Mean elbow angle, velocity, acceleration, biceps brachii (agonist) and lateral triceps (antagonist) electromyograms (EMGs) of one subject (1) for ten movements over each of the distances 2.8°, 5.6°, 8.4°, and 11.25° (**A**) and 11.25°, 22.5°, 33.75°, and 45° (**B**). EMGs are smoothed with a 10-ms, rectangular filter. The movement over 11.25° is the same in **A** and **B**. The triceps EMGs have been inverted. The mean record shows many of the features found in the single records but is not a reliable way to determine timing features that get blurred. The mean record was never used to measure the movement parameters plotted in Figs. 2–7. All those measures were from single records

The relationships between these EMG patterns and the kinematic patterns can be examined quantitatively by looking at parametric measures of the time series. An increase in MT with distance is commonly seen in experiments such as these (Fitts 1954). This relationship was described as nearly linear in our previous studies over the 18–72° range (Gottlieb et al. 1989a). However, visual inspection of Fig. 1 suggests that, for shorter distances, constant MT (isochrony) emerges as a spontaneous behavior. This was confirmed in two ways. Linear regression coefficients were computed for MT using all seven distances and recomputed for only the four longer dis-

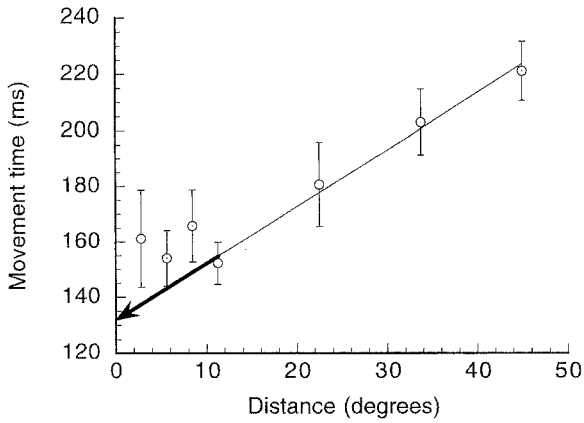


**Fig. 2** Mean movement time (±SE, $n$=8) is plotted versus seven different distances. A linear relationship has been plotted through the four longest distances ($MT_4$=132+2.03θ; $r$=0.99). The *heavy arrow* shows the extrapolation of this line to fall below the measurements at the three shorter distances

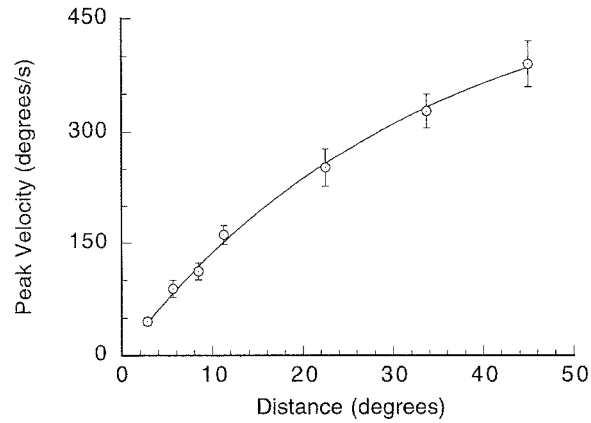


**Fig. 3** Mean peak movement velocity (±SE, $n$=8) is plotted against seven different distances. The data have been fit with a nonlinear curve given by Eq. 5:

$$PV = a \cdot \left(1 - e^{\frac{-\theta}{\theta_0}}\right)$$

Parameter values are $a$=507, $\theta_0$=31.6; $r$=0.99

tances. Regression lines for the four longer distances alone had lower intercepts and steeper slopes for five of the eight subjects and were almost identical coefficients for two (subjects 5 and 7). The kinematic features of one subject's movements (6) were contrary and this subject's EMG data also failed to fit the model. The net effect for the group is shown in Fig. 2, where the mean MT for all eight subjects is plotted against distance. The linear regression line computed on the four longest distances is extrapolated to zero to illustrate the deviation of the MT for short distances from the trend at long distances.

The PV data are well characterized by an exponential relationship between PV and movement distance (Eq. 5), which is based on assumed nonlinear control processes (Bahill et al. 1975).

$$PV = a \cdot \left(1 - e^{\frac{-\theta}{\theta_{PV}}}\right) \quad (5)$$

We have fitted Eq. 5 to the mean of all eight subjects in Fig. 3. The equation also fits the behavior of each individual, all of whom had $r$ values of 0.99. Milner (1986) proposed an alternative to Eq. 5 for the relationship between PV and movement distance at the interphalangeal joint of the thumb in the form:

$$PV = \alpha\theta^{\beta} \quad (6)$$

This equation also fits the data in Fig. 3 well (α=27.1, β=0.70; $r$=0.99; $n$=8).

The kinematic changes reflected by MT and PV are consequences of altered muscle contractile forces that result from changes in muscle activation patterns. Those patterns are described by Figs. 4 and 5 for all eight subjects, plotting $Q_{30}/a_1$ and $Q_{ag}/a_2$ as they varied with distance.

The measure of $Q_{30}/a_1$ for all eight subjects is plotted in Fig. 4. Thin lines show the least-squares fits of Eq. 5 for seven of the eight subjects. The heavy line uses the mean of the seven coefficients. One subject was omitted because of the poor fit of Eq. 5 to his data. The range of $\theta_{ag}$ was 3.44–10.31 (mean 6.35) and $r$ ranged from 0.77

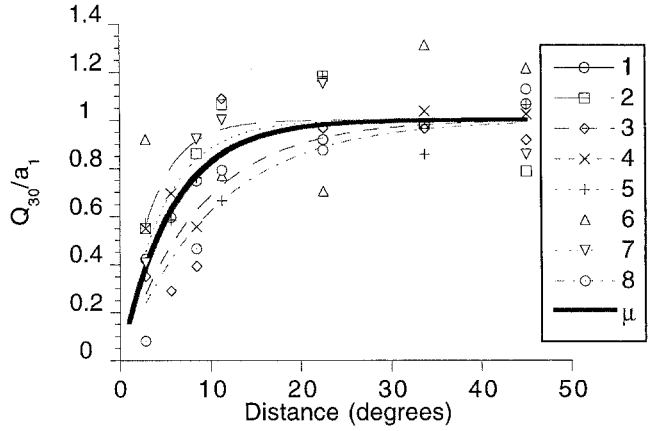


**Fig. 4** Agonist EMG integrated over the first 30 ms is plotted against movement distance. The data have been fit by Eq. 1:

$$\frac{Q_{30}}{a_1} = \left(1 - e^{\frac{-\theta}{\theta_{ag}}}\right)$$

*Thin lines* are of individual subjects, the *heavy line* is for the mean $\theta_{ag}$=6.35

to 0.98. If we were to speak of the angular range having distance-proportional and distance-invariant segments, the boundary would lie at about $2\theta_{ag}$ or about 7–20°.

Figure 1 suggests that $Q_{ag}$ always increases with distance (Godaux 1989). The model predicts that the steepness of the relationship will be greatest at short movement distances, a behavior that is satisfied by equation 2. Cheron and Godaux (1986) fit the area of the agonist burst with a similarly shaped function. Figure 5 shows data for all eight subjects. To estimate the coefficients of equation 2, we set $Q_{ag}$ for each subject to the value found from fitting $Q_{30}$ in Fig. 3 and optimized $a_2$ and $W_{ag}$. There were a wide range of values found for $W_{ag}$ (16.8–121.2, mean 70.5 for seven subjects). The solid line in Fig. 4 shows the fit of Eq. 2 with $\theta_{ag}$=6.35,

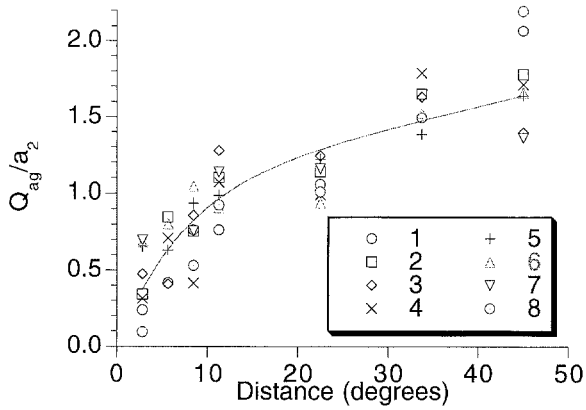


**Fig. 5** Agonist EMG integrated over the acceleration phase of the movement is plotted against seven different distances. The *solid line* shows Eq. 2:

$$Q_{ag} = a_2 \cdot \left(1 + \frac{\theta}{W_{ag}}\right) \cdot \left(1 - e^{\frac{-\theta}{\theta_{ag}}}\right)$$

for the mean parameter values, $\theta_{ag}$=6.35, $W_{ag}$=70.5

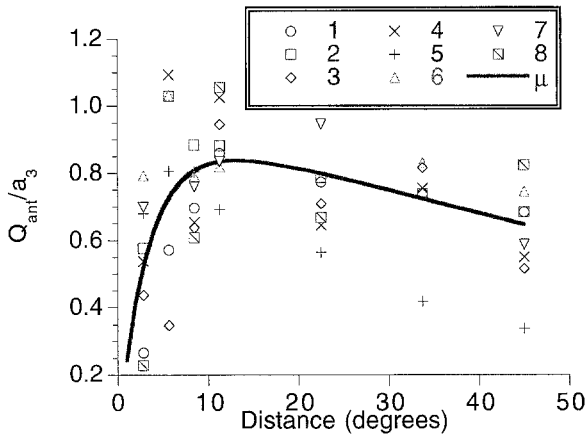


**Fig. 6** Mean antagonist EMG integrated over the time course of the movement (mean±mean of the SEs) is plotted against seven different distances. The data have been fit with Eq. 3:

$$Q_{ant} = a_3 \cdot \left(1 + \frac{\theta}{W_{ant}}\right) \cdot \left(1 - e^{\frac{-\theta}{\theta_{ant}}}\right) \cdot e^{\frac{-\theta}{\theta_1}}$$

The *heavy line* is for the mean parameter values, $\theta_{ant}$=4.15, $W_{ag}$=70, $\theta_1$=46

$W_{ag}$=70.5. The fits with the individual coefficients gave values of $r$ in the range of 0.82–0.98, but, in fact, $W$ was relatively insensitive to the coefficients so that the mean regression curve fit the individual data sets with values of $r$ from 0.79 to 0.96.

The area of the antagonist EMG burst ($Q_{ant}$) is plotted for all eight subjects in Fig. 6. Over longer distances, this variable usually has a low and often negative correlation with movement distance, as reported before (Benecke et al. 1985; Cheron and Godaux 1986; Gottlieb et al. 1989a, 1992; Marsden et al. 1983; Wadman et al. 1979). The model's Eq. 3 is based on the assumption that the area of the antagonist burst will be proportional to distance for short movements and negatively related for long

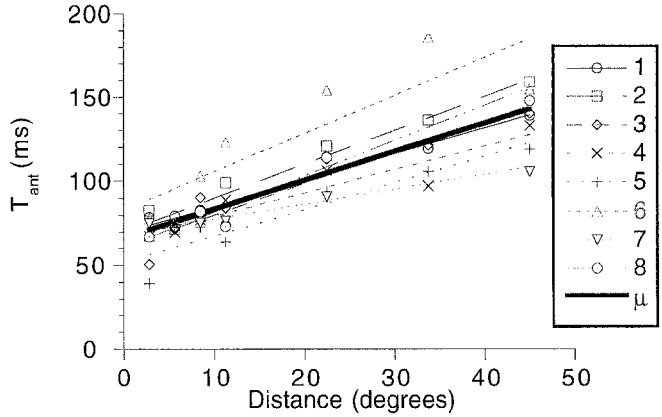


**Fig. 7** Antagonist latency (±SE) is plotted against seven different distances. A linear relationship (Eq. 4: $T_{ant}=T_0+K\theta$) has been plotted through the data. *Thin lines* are of individual subjects, the *heavy line* is for the mean $T_0$=66.4, $K$=1.7

movements. Fitting Eq. 3 to the data, using $\theta_{ant}$=6.35 and $W_{ant}$=70 gave $r$ values from 0.75 to 0.96 for four of the eight subjects (1, 3, 5, 8). This was because although the other four subjects (2, 4, 6, 7) did show a modest decline in $Q_{ant}$ with longer distances, $Q_{ant}$ only showed an increase with distance in going between the two shortest distances. Three of the subjects in this second group were fit reasonably well if $\theta_{ant}$ was reduced to 2.5, but subject 6 (who also did not conform for $Q_{30}$) could not be fit. The solid line in Fig. 6 shows Eq. 3 with the mean coefficient values ($\theta_{ant}$=4.15, $W_{ant}$=70, $\theta_1$=46).

The latency of the antagonist burst is proportional to distance over the entire range shown in Fig. 7. The straight lines were fit to those data by Eq. 4.

## Discussion

The control model described in the introduction postulates that the nervous system plans patterns of motoneuron excitation and, from the resultant muscle contractions, kinematic and EMG patterns emerge. Measures of EMG and kinematic parameters are not linear functions of distance if examined over a sufficiently wide range of movement distances, performed as fast and accurately as possible.[2] Reports in the literature that describe approximately linear scaling of EMG measures with distance have usually been over restricted ranges of movement conditions. Although some of our data are well described by linear regression, that approach offers no insight into causal relations between the correlated variables. Our model equations (Eqs. 1–4) are based upon a specific pulse-height/width control scheme that necessitates the greater computational complexity.

[2] Presentation of a small target and the inclusion of accuracy in the instruction is sufficient to ensure that these movements were not the very fastest the subjects were capable of performing (Fitts 1954) and that the invariant features we found are not simply an expression of physiological limits (Gottlieb et al. 1990).

With respect to kinematics, evidence in the eye movement literature suggests that, as in Fig. 2, velocity scales to keep MT constant for short movements and proportional to distance for longer ones (Bahill et al. 1975). Several studies (Bahill et al. 1975; Langolf et al. 1976; Milner 1986; Newell et al. 1984; Viviani and McCollum 1983) have all found, using diverse movement tasks, that increments in velocity progressively decrease as a function of distance, as in Fig. 3. Bahill (1981) fit saccadic eye movements that ranged from 3 min of arc to 50° with Eq. 5 ($a$=850, $\theta_0$=10.6) and his MT data with a linear equation (MT=20+1.7θ). Although Eqs. 5 and 6 fit the data well, neither are based on specific underlying physical mechanisms. This kinematic nonlinearity might be an intrinsic property of muscle contractile processes. It might instead (or also) emerge from a nonlinear control process that generates the muscle activation signal. The existence of such processes may be inferred from observations of similar nonlinear features in the EMG signals that are shown in Figs. 4–6. This nonlinear behavior of $Q_{ag}$ and the linear variation of $T_{ant}$ has also been reported by Cheron and Godaux (1986). They did not observe nonmonotonic behavior of $Q_{ant}$ but only a decline with distance. Their experiments were performed in the vertical plane and therefore made somewhat different demands on the forces required of the muscles. Thus, the nonlinear behavior of the EMG is consistent with the hypothesis that the kinematic features that characterize Figs. 2 and 3 can be, at least in part, emergent properties of the neural control mechanisms.

The observation that $Q_{30}$, which characterizes the onset of the agonist contraction, is reduced for short movements has not been previously reported at the elbow joint. This is a key result because $Q_{30}$ is our measure of excitation pulse height, which we now demonstrate is dependent on movement distance. The dependencies of various EMG quantities on movement distance that are plotted in Figs. 4–7 cover a different angular range than we have previously published. The results over the four longer distances reproduce findings that have frequently been reported (see Gottlieb et al. 1989b for a review). Over the shorter distance range, these results at the elbow are similar to those of Cheron and Godaux (1986) and to Hoffman and Strick (1986, 1990, 1993) for wrist movements.

The published descriptions of antagonist scaling have been ambiguous or contradictory (see Gottlieb et al. 1989b) when they have not been omitted. The quality of our model's fit to these data is weakest for the antagonist and this is the expected outcome of a control mechanism that is most influenced by distance and load effects on the force-producing properties of a lengthening muscle.

## The structure of the model

Do Eqs. 1–4 represent more than just functions that fit the data? We suggest that they represent two sets of neurophysiological constraints. One set is of linear but arbitrary[3] rules for pulse timing. The other set is of nonlinear rules imposed on pulse height by neuromuscular mechanisms.

From observations of the agonist EMG patterns, we can see that the durations of the agonist EMG bursts are proportional to movement distance but do not approach zero, even as distance does. We can also see that this is also true of the latency of the antagonist burst. This leads us to write the following two equations for pulse duration and antagonist latency.

$$W = W_0 + k_w\theta \tag{7}$$

$$T_{ant} = T_0 + K\theta \tag{8}$$

These are examples of arbitrary rules, since it is easy to imagine that movement distance could be controlled by alternative schemes (e.g., $W_0$=$T_0$=$0$). We can speculate that these rules might result from the fact that reducing $W_0$ is an ineffective way of reducing the agonist contractile force to zero when $\theta \rightarrow 0$. Even the shortest pulse (a single action potential) would still produce a twitch contraction of 50–100 ms duration).

We also know that the slope of the agonist burst is constant for movements of longer distance and therefore $H$ must be constant. If $H$ were constant over the entire range of movement distance, agonist burst area could not be modulated at short distance very effectively (the smallest possible area would be $W_0H$). Hence, although $H$ can be constant for larger distances, it is necessary that it fall to zero as movement distance gets very small. Equation 9 is a function that has these properties. Equation 1 for $Q_{30}$ is the same as Eq 9. Equation 2 for $Q_{ag}$ is the product of Eqs. 7 and 9:

$$H = k_h\left(1 - e^{\frac{-\theta}{\theta_0}}\right) \tag{9}$$

The above premises imply a monotonic increase in the area of the excitation pulses throughout the full range of movement. However, many investigators have shown that this is not true for the elbow antagonists, which often show constant or slightly decreasing EMG activity at longer movement distances (Benecke et al. 1985; Gottlieb et al. 1989a; Wadman et al. 1979). We proposed (Gottlieb et al. 1992) that the antagonist is activated in a nonmonotonic manner, increasing with distance for shorter movements and then decreasing for longer movements (Lestienne 1979), because muscular viscous properties increased the mechanical effectiveness of the antagonists that are undergoing lengthening and reduce the needed strength of excitation. Therefore, we need a fourth equation to describe the nervous system's recognition and accommodation for this mechanical property of muscles. We modify Eq. 9 for the antagonist. Equation 3 for $Q_{ant}$ is the product of Eqs. 7 and 10:

[3] Why the rules we observe were chosen we do not know. The reason might be that they optimize some performance measure (e.g., minimum time, energy, jerk, etc.) but this has not been and probably cannot be established with any degree of certainty. By "arbitrary" we simply mean that similar movements could have been performed under alternative rules.

$$H_{ant} = k_h\left(1 - e^{\frac{-\theta}{\theta_0}}\right)e^{\frac{-\theta}{\theta_1}} \quad (10)$$

The data are well characterized by Eqs. 1–4, three of which are nonlinear, that constitute our model. This applies both to the pooled data set and the individual data sets. The individual parameters are mostly within a factor of 2–3 of each other, but, for every parameter computed, two or three of the eight subjects show widely divergent values if we simply accepted the values that produced the least-squared error. We find, however, that the EMG predictions of the model are relatively insensitive to the equation coefficients. One interpretation of this insensitivity is that we could use a simpler mathematical model, since linear regression also provides a good fit to all but the $Q_{ant}$ data. However, because we assumed the structure of the model and its two excitation pulses prior to fitting the data, this insensitivity becomes a major strength. It implies that the nervous system will produce repeatable excitation pulses, and as a result repeatable movements, in spite of neural noise, computational inaccuracy in specifying some of the control parameters, or any other cause of variability. Regression equations that are simply descriptive of the data are normally chosen by a trade-off between accuracy and simplicity. The approach taken here has chosen equations based upon a model that describes the modulation of muscle excitation pulses. That modulation is determined by certain desired features of the movement task (Gottlieb 1993). The model characterizes aspects of the physiological mechanisms from which experimental observations emerge rather than just describing those observations. An advantage of this approach is that, beyond describing this data set, the model allows us to make predictions about how all the features of the EMG patterns illustrated in Figs. 4–7 will vary with other movement tasks. Examples of such tasks are movements of constant duration made over different distances (Gottlieb et al. 1995) or movements performed over increasing distances with decreasing inertial loads.

## Omitted elements

This proposed model relies entirely on accurate prior knowledge of the dynamics of the movement task for successful operation. Thus, it omits two important features of the neuromuscular system. One is neuromuscular compliance. Although we have spoken as if the muscles were perfect force generators, they have internal compliance and therefore their mechanical performance depends upon the dynamic properties of the load (Gottlieb 1994). The second omission is of all reflexes which also create a functionally compliant system. Reflexes are unquestionably present, but the nature of their actual contribution to the control of these movements is speculative and unproved (Gottlieb 1995).

Neither of these features are necessary to the arguments presented here, because our subjects were well practiced and knowledgeable about the task. Given accurate knowledge to specify the coefficients of our equations, the proposed control scheme will generate muscle excitation pulses that can produce all the EMG patterns seen during movements of widely differing distances. In the absence of accurate knowledge, errors of movement would be produced (as they are produced in the actual system), in which case compliant and reflex mechanisms would play a more visible role. Experiments to explore this are soon to be reported elsewhere (G.L. Gottlieb, 1966).

## Conclusion

In this paper, we have shown by studying movements over a wide range of distances that pulse-width and pulse-height modulation strategies are not rigidly fixed by the kinematic task but are adjusted to both the kinetic requirements of that task and the physiological constraints imposed by muscle mechanisms. Thus, pulse-height/width modulation is not an either/or proposal but a graduated device for performing diverse movements.

From this we have generalized our previously proposed notion of "strategies" as "sets of rules that determine the patterns of muscle activation" (Gottlieb et al. 1989a, p. 343). *What we previously enumerated as two strategies, SI (which used pulse-width modulation of the agonist excitation pulse) and SS (which used pulse-height modulation), are experiment-specific aspects of a single algorithm that controls the heights, widths, and timing of the agonist and antagonist excitation pulses.* They are not independent entities but different views of the same entity, observed under two different task conditions, one of which is specifically concerned about movement speed and one of which is not. Small movements give a different view of the workings of the same algorithmic procedure in which pulse height and width are both modulated. This approach may be broadened to include additional kinds of movements, including those that involve fewer mechanical constraints and more than a single joint (Almeida et al. 1995; Hong et al. 1994).

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