Movement-Related Phasic Muscle Activation

I. Relations With Temporal Profile of Movement

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SUMMARY AND CONCLUSIONS

1. The role of phasic muscle activation in determining the temporal properties of human arm movements was studied. The experiments show that subjects can modulate the triphasic electromyographic (EMG) pattern to produce movements of varied temporal structures.

2. Subjects performed horizontal forearm movements in which they varied movement accelerations and decelerations. All movements were of the same amplitude, duration, and peak velocity. A phase-plane (velocity vs. position) template of the desired movement was presented to the subject, who had to reproduce the template by appropriate movement of the forearm.

3. The ratio of the durations of acceleration to deceleration (termed the symmetry ratio, SR) was used as a measure of the temporal structure of the movements. Movements with SRs ranging from 0.4 (short acceleration–long deceleration) to 2.0 (long acceleration–short deceleration) were studied.

4. Subjects modulated the components of the triphasic EMG pattern to produce movements with different temporal profiles. As the SR was increased (increasing acceleration duration–decreasing deceleration duration), the following changes occurred: 1) the duration of the initial agonist burst (AG1) increased while its magnitude decreased; 2) the antagonist burst (ANT1) was progressively delayed relative to movement onset. ANT1 magnitude increased while its duration remained constant, and 3) the magnitude of the second agonist burst (AG2) increased and its duration decreased.

5. The triphasic EMG pattern can be modified to produce movements whose velocity profiles are not the same under simple scaling of duration or magnitude. It is concluded that previously described relations between components of the triphasic EMG pattern and movement parameters, such as amplitude, speed, and duration, are secondary to associated changes in their acceleration and deceleration characteristics.

INTRODUCTION

A considerable body of literature now exists that centers on the controlled variable or variables underlying the generation of simple, skilled movements. Movement parameters such as amplitude, duration, and peak velocity, either independently or in combination, have been suggested as being involved at the motor planning level (Benecke et al. 1985; Freund and Buedingen 1978; Lestienne 1979; Marsden et al. 1983; Schmidt et al. 1988; Stein 1982; Wierzbicka et al. 1986). More recently, interest has focused on the dynamics of movement, particularly on the time course or shape of the velocity profile (Abend et al. 1982; Georgopoulos et al. 1981; Hogan 1984; Morasso 1981; Soechting and Lacquaniti 1981). In general, the path the limb takes when moving from one position to another is highly stereotyped. The velocity profile is bell-shaped with approximately the same time spent in accelerating as in decelerating the limb. Such temporal profiles have been reported for movements about single joints as well as in more complex, multijoint movements (Atkeson and Hollerbach 1985; Ostry et al. 1987; Soechting 1984). It has been suggested that such a profile may represent the most cost-efficient means of movement production (Nelson 1983). The observation that the form of the velocity profile is invariant under transformations of movement amplitude and duration suggests that symmetrical velocity profiles may reflect a basic organizing principle underlying movement generation.

If the temporal structure of the movement to be made is an important determinant in motor planning, then the question arises as to how the central nervous system produces time-symmetric profiles. Clearly, movement trajectory will be affected by the forces produced by the contracting muscles acting about a given joint. In simple, single-joint movements, activation of antagonistic muscles occurs in a typical “triphasic” pattern where an initial burst of agonist activity (AG1) is followed sequentially by a burst in the antagonist (ANT1) and a second, less-well-defined agonist burst (AG2). The function and control of the various electromyographic (EMG) components of the triphasic pattern are not yet fully understood despite rigorous examination by many investigators. It is generally accepted that the initial agonist burst provides the driving force for setting the limb in motion. The magnitude of this burst increases with both movement amplitude and speed (Benecke et al. 1985; Brown and Cooke 1981; Hallett and Marsden 1979). In addition, the duration of AG1 increases with movement amplitude (Benecke et al. 1985; Berardelli et al. 1984; Brown and Cooke 1982, 1984; Mustard and Lee 1987).

Although the function of the antagonist burst has been assumed to be one of braking the movement, differing views have been put forward concerning its generation and control. It has been suggested that AG1 and ANT1 are generated together, this linkage of reciprocal EMG bursts forming part of a central motor command or program (Hallett et al. 1975; Hopf et al. 1973). Other lines of evidence indicate that, although ANT1 is centrally generated, it is a separately controlled component of the triphasic pattern (Flament et al. 1984; Lestienne 1979; Marsden et al. 1983). Still others have proposed that stretch reflex ac-
tivity plays a role in the generation of ANT1 (Ghez and Martin 1982; Ramos and Stark 1987).

The third component of the triphasic pattern (AG2) has received little attention, primarily because of the difficulties in accurately determining the timing of this burst. It has been suggested, however, that AG2 represents a stretch-reflex-dependent damping mechanism that acts to reduce unwanted oscillations occurring at the end of rapid movement (Ghez and Martin 1982). It has also been suggested that AG2 is a “clamping” pulse used to fix the limb in the final position (Hannaford and Stark 1985).

Much of the previous research has focused on step-tracking movements in which only movement endpoint and/or duration have been specified. The velocity profiles of such movements are approximately equivalent independent of movement amplitude and/or duration. Thus it has not been possible to relate phasic muscle activation to the overall dynamic or temporal structure of such movements by manipulation of movement amplitude and/or duration. We have recently developed a phase-plane tracking paradigm where the temporal structure of movements can be precisely controlled (Cooke and Brown 1986). In the present experiments, we have utilized this technique to study the relations between EMG activity and the temporal structure of movements. The data will demonstrate that, under conditions in which movement amplitude, duration, and maximum velocity are constant, the various components of the triphasic pattern show regular and strong relationships with the ratio of acceleration to deceleration durations (termed the symmetry ratio or SR).

METHODS

Experiments were performed on five normal, male subjects, aged 22–46 yr with no known history of neurololgical disorders. Each subject was seated comfortably and grasped a vertical rod attached to a horizontal manipulandum handle. The subject’s upper arm was abducted 90° and supported at the elbow directly beneath the pivot point of the handle. During each experiment, the subject was asked to make visually guided, horizontal flexion/extension arm movements.

To enable subjects to make movements of different temporal profiles, a template of the desired movement was displayed to the subject on a storage oscilloscope (see Cooke and Brown 1986 for details of template generation). The target template took the form of a phase-plane representation in which desired arm velocity was displayed as a function of desired arm (handle) position throughout the movement. By changing the symmetry ratio, movement templates of varying degrees of temporal asymmetry could be produced while, at the same time, keeping movement amplitude, peak velocity, and duration constant. Once the desired target template was displayed to the subject, handle position and velocity were switched to the oscilloscope inputs. The subject was then asked to reproduce the target phase-plane trajectory by appropriate movement of the handle.

Experimental paradigm

In the present experiments, all movements were of 40° amplitude. For any one subject, movement duration was kept constant at either 600 or 800 ms. The SR ranged from 0.2 (short acceleration–long deceleration) to 2.0 (long acceleration–short deceleration) with the ratio held constant within any one trial. Each trial consisted of 40 alternate flexion-extension movements in which the subject was asked to reproduce the target template as accurately as possible. Each subject was given several minutes of practice to become familiar with the range of target trajectories. The initial trial always consisted of symmetrical movements (SR = 1.0), after which trials alternated between those with symmetry ratios greater or less than 1.0. All subjects were able to produce movements of varying degrees of asymmetry after 5–10 min of practice.

Data recording and analysis

The data recorded were the angular position, velocity, and acceleration of the manipulandum handle and the surface EMGs from the biceps and the lateral head of the triceps muscles. Handle position was obtained from a precision potentiometer and angular velocity from the back emf induced in a linear DC torque motor by movement of the handle. Angular acceleration was obtained from an accelerometer. Surface EMGs were recorded with paired disk electrodes placed 2–3 cm apart along the longitudinal...
Kinematic parameters of phase-plane tracking movements. Peak velocity (A), amplitude (B), and duration (C) of phase-plane tracking movements are plotted as a function of the ratio of acceleration to deceleration durations (SR). Data from 2 subjects (○ and △). Each point represents data from an individual flexion movement.

RESULTS

Comparison of step- and phase-plane tracking movements

Symmetrical movements produced by phase-plane tracking were qualitatively indistinguishable from movements produced by visual step-tracking where only the start and end of the movement were defined. Kinematic and EMG data obtained from both step- and phase-plane tracking paradigms are shown in Fig. 1. For the phase-plane tracking movements, movement amplitude and duration were set to be approximately the same as for the step-tracking movements. The symmetry ratio equaled 1.0. In the averaged data shown here, position and velocity records were virtually identical between the two paradigms. Movement variability, as indicated by the SD bars, was often less for phase-plane tracking than for step-tracking movements, particularly during the first one-half of the movement. The pattern of muscle activation associated with both types of tracking movements was also similar. These temporally symmetrical movements were initiated by a burst of activity in the agonist muscle (AG1) that was followed by a burst of antagonist activity (ANTI) at about the time of maximum movement velocity. Late agonist activity (AG2), present in movements made with both paradigms, was of prolonged duration and often merged together with the tonic EMG activity related to the new limb position. Although early antagonist activity, coactive with AG1, was often observed in both step- and phase-plane tracking movements (compare Brown and Cooke 1986; Mustard and Lee 1987), we have, in the present study, followed the accepted definition of the triphasic pattern (Brown and Cooke 1981; Hallett et al. 1975; Mustard and Lee 1987) and restricted EMG analysis to AG1, ANTI, and AG2.

All five subjects were able to reproduce target phase-planes of varying degrees of asymmetry after 5–10 min of practice. Subjects could maintain movement duration, amplitude, and maximum velocity constant while producing movements with different SRs. This is illustrated in Fig. 2 for two subjects. Neither the mean values nor the
Changes in the triphasic EMG pattern with changing SR

Timing and magnitude of phasic EMG activity were dramatically affected by changes in the temporal structure of the movement. The general features of these changes are illustrated in Fig. 3. As SR increased (top to bottom EMG traces), the duration of AG1 increased and burst magnitude decreased. The time of onset of phasic antagonist activity (ANT1) was progressively delayed as SR increased. Strikingly, movements with large SRs (bottom set) showed a prominent AG2 immediately after onset of peak velocity. These general features of the changes in the triphasic EMG pattern with SR were seen in all subjects studied.

Although movement symmetry altered the timing and magnitude of movement-related EMG activity, the relationship between the various EMG bursts and movement kinematics permits the continued use of conventional terminology (AG1, ANT1, and AG2). Following Hallett et al.'s (1975) definition of the triphasic EMG pattern, AG1 begins 30–40 ms before movement onset and ends before onset of peak velocity. ANT1 occurs at or near the time of peak velocity and coincides with a period of relative inactivity in the agonist. AG2, although often not as well defined, occurs near the end of the movement.

As mentioned, all subjects could produce movements with varying degrees of asymmetry, although some subjects were more consistent in their performance than others. The following figures (Figs. 4–10) will illustrate only data from two subjects who were able to consistently produce movements with a full range of asymmetries.

**AG1**

Both the duration and magnitude of AG1 varied with the time course of the movement. Over a range of SRs from 0.4 to 2.0, the duration of AG1 showed a sixfold increase from ~80 to >500 ms (Fig. 4). The relationship between AG1...
duration and movement symmetry was linear over most of the movement ratios examined. Occasionally, AG1 duration showed smaller increases at higher SRs (compare Fig. 4B, ratio 1.6–2.4). In some subjects AG1 durations clustered around 80–100 ms and 150–200 ms in movements with SRs of 0.2–0.6. This was not, however, a consistent finding and was only seen in movements of shorter durations (600 ms). In general, AG1 duration was more variable in movements with SRs > 1.0.

For movements with SR < 0.8, integrated AG1 activity was inversely related to SR (Fig. 5). Thus the magnitude of AG1 decreased as SR was increased from 0.2 to 0.8. Over the range of SRs from 0.8 to 2.0, AG1 magnitude remained virtually unchanged. Similar nonlinear relationships between AG1 magnitude and symmetry ratio were seen in all subjects. Thus, in movements with SRs less than ~0.8, both the duration and magnitude of AG1 varied with the SR. In contrast, movements with SRs greater than ~0.8 were initiated with agonist activity of variable duration and relatively fixed magnitude.

Whereas the initial agonist burst in movements with small SRs was clearly burstlike, the profile of AG1 activity in movements with large SRs was quite different. In some cases, there was a gradual increase in AG1 activity, which peaked just before onset of maximum velocity and which was followed by a silent period (Fig. 3, SR = 1.6). In other cases, the agonist activity initiating movement appeared as a more or less step increase in tonic activity which ceased

![Figure 5](image1.png) **FIG. 5.** AG1 magnitude as a function of SR.

![Figure 6](image2.png) **FIG. 6.** Time of onset of ANT1 as a function of SR. ANT1 onset time was determined relative to movement onset.

![Figure 7](image3.png) **FIG. 7.** ANT1 duration as a function of SR.
abruptly at the time of peak velocity. Despite these differences in EMG patterns, no obvious irregularities in the velocity profile were detected.

**ANT1**

A burst of activity in the antagonist muscle (ANT1) was seen in all movements regardless of the degree of movement asymmetry. As illustrated in Fig. 3, the onset of ANT1 was always just before or at the time of maximum velocity. This is shown quantitatively in Fig. 6, where the onset of ANT1 relative to movement onset was progressively delayed from ~20 to >500 ms as SR increased from 0.4 to 2.4. Although the time of occurrence of ANT1 varied with SR, ANT1 duration was relatively constant across the full range of SRs examined (Fig. 7). For the subject represented in Fig. 7A, mean ANT1 duration across all SRs was 140 ± 31 (SD) ms and, in Fig. 7B, 135 ± 36 ms. Mean ANT1 durations of 130–160 ms were observed in all subjects. The variability of ANT1 duration did not change systematically with SR.

The magnitude of ANT1 depended on the degree of movement asymmetry. ANT1 magnitude was smallest for nearly symmetric movements (SR = 0.8). In movements with SR > 0.8, the magnitude of ANT1 increased with SR.

In some subjects ANT1 magnitude increased as SR decreased in the range from 0.8 to 0.4 (Fig. 8A). In such cases, however, the increase in ANT1 magnitude associated with rapid rise time movements (SR = 0.4) was never as great as that seen in long rise time movements (SR = 2.0).

**AG2**

The appearance of phasic agonist activity occurring late in the movement and corresponding to the AG2 of the classic triphasic pattern depended heavily on movement symmetry. AG2 was seldom seen in movements with SR < 0.6. In these movements there was a slight increase in tonic agonist activity after ANT1 that persisted for the remainder of the movement (compare Fig. 3, SR = 0.5). In symmetric and near-symmetric movements (SR = 0.8–1.2) late agonist activity became more burstlike in appearance. The termination of this activity was often difficult to distinguish from tonic, postmovement activation (compare Fig. 3, SR = 0.9). Discrete late agonist bursts were, however, always seen in movements with SR > 1.2 (e.g., Fig. 3, SR = 1.6). In such movements, the onset of AG2 was near the time of peak velocity and the burst was terminated 80–100 ms after the end of movement.
The relationship between EMG activity (as an indicator of the final commands for movement) and the resulting movement is basic to any understanding of movement generation and control. Since the description of the triphasic EMG pattern by Hallett et al. (1975), many investigators have described relations between the components of this pattern and various characteristics of the resulting movements. Thus studies have been made of the durations, magnitudes, and timings of the phasic bursts in relation to movement amplitude, duration, and maximum speed. The choice of these simple kinematic features of movement was undoubtedly based on the ease of their experimental manipulation as well as the intuitive sense that these were parameters of functional importance for the nervous system in setting up the commands for movement.

A number of recent studies, however, have pointed out the importance of the path or trajectory of movements and/or their time course. For example, it has been shown that phasic muscle activation evoked by sudden perturbation of the limb during movement about a single joint acts to restore the limb to a prelearned velocity-position (phase-plane) trajectory (Cooke 1980). Further studies have shown that prolonged movement practice results not only in a decrease in the variability of the end position of the movement but also in a decrease in the variability of the movement phase-plane at every point throughout the movement (Darling and Cooke 1987). In the case of movement in multidimensional space involving two or more joints, the variability at every point of the path taken during the movement also decreases with practice (Georgopulos et al. 1981). Such findings suggest the possibility that, in setting up the commands for movement, the nervous system may utilize a somewhat more complex language than that of simple point kinematic features of the desired movement.

In attempting to determine how muscle activation is related to movement, most investigators have utilized step-tracking movements. Such movements have a typical and reproducible time course. Angular velocity increases smoothly to a maximum and thereafter decreases smoothly to zero as the target is reached. For movements about different articulators (Ostry et al. 1987) as well as about different joints during multijoint movements (Soechting et al. 1981; Soechting 1984), the duration of the acceleratory phase of movement is slightly less than that of the deceleratory phase. The relative durations of these two phases are approximately constant across movement amplitudes (Cooke et al. 1989). The velocity profiles of movements of different amplitudes and durations are approximately equivalent under scalar transformation of amplitude, duration, path, and inertial load (Atkeson and Hollerbach 1985; Ostry et al. 1987; Ruitenbeek 1985; Soechting 1984). Thus it appears that different movements may be made according to a common or basic pattern. Theoretical studies have suggested that this pattern (the shape of the velocity profile) may arise through a minimization of such parameters as energy or jerk (Hogan 1984; Nelson 1983) or optimization of joint stiffness (Hasan 1986), although a recent paper
movement. In movements with SRs ranging from 0.2 to decreased. However, for SRs greater than -1.2, AG1 magnitude or duration with movement amplitude (Be-
titude (Cooke et al. 1989) changes in, for example, AG1 magnitude and duration were reciprocally re-
necke et al. 1985; Brown and Cooke 1981; Gielen et al.
magnitude or duration with movement amplitude. However, this is a necessary but not a sufficient condition. For example, movements with triangular velocity profiles but different SRs are not equivalent under scalar transformation in amplitude or duration, although c is the same for all such movements. Although the value of c was not explicitly calculated in the present experiments, movement peak velocity, amplitude, and duration did not vary across SRs (Fig. 2). Thus c \((-V_{\text{max}} \cdot \text{Dur}/Amp)\) did not vary as a function of SR. In spite of this constancy of the parameter c, the movements in this study were not equivalent under simple scalar transformation because of the changes in the SR.

A critical finding of this study is that movements not belonging to the same equivalence class but differing in their temporal structure were generated by modification or modulation of a triphasic EMG pattern. All movements were initiated by activity in the agonist, which was followed by antagonist activity and a second period of agonist activity. The timing of these various periods of EMG activity permits the continued use of the term triphasic following the definitions of Hallett et al. (1975). The data show that these various components of the triphasic pattern (AG1, ANT1, and AG2) can be modulated independently of any changes in movement amplitude, duration or peak velocity. For example, although it is undoubtedly correct that the durations of phasic muscle activations vary with movement duration (Schmidt et al. 1988; Sherwood et al. 1988), it is now clear that these activities can be varied while movement duration remains constant. This should not be interpreted as indicating that relationships which have been previously found between EMG activity and these kinematic variables are secondary to related changes in SR. Because SR is relatively constant with movement amplitude (Cooke et al. 1989), changes in, for example, AG1 magnitude or duration with movement amplitude (Be-
necke et al. 1985; Brown and Cooke 1981; Gielen et al. 1985; Hallett and Marsden 1979; Wierzbicka et al. 1986) cannot be secondary to amplitude-related changes in SR.

The changes in phasic activity with SR in the present study underscore the importance of the desired temporal characteristics of a movement in the programming of the commands for that movement. The observed changes in AG1 as a function of SR suggest two modes of movement initiation, depending on the desired temporal profile of the movement. In movements with SRs ranging from 0.2 to 1.2, AG1 magnitude and duration were reciprocally related; as AG1 duration increased with SR, AG1 magnitude decreased. However, for SRs greater than \(~1.2\), AG1 mag-
itude remained relatively constant while its duration con-
tinued to increase with SR. Thus both amplitude and dura-
tion modulation of AG1 were utilized with SR < 1.2 but only duration modulation with SR > 1.2. Indeed, the ago-
nist EMG activity initiating movements with SR > 1.2 appeared similar to that seen in "slow" movements (Hal-
lett et al. 1975). Such movements are produced by a tonic increase in EMG activity. Movements with SR > 1.2 ap-
ppeared to share characteristics of “fast” and “slow” move-
ments; their initiation was similar to that of slow move-
ments, whereas their termination via ANT1 and AG2 was similar to that of fast movements.

A change from an EMG pattern typical of fast move-
ments to one normally associated with slow movements was also evident in AG2 as SR was progressively decreased. Sharply defined, burstlike AG2 activity was seen in move-
ments with SR > 1.2, that is, in movements in which there was a rapid decrease of velocity from its maximum. For SR values <1.2, AG2 was more prolonged and less burstlike; it appeared similar to the late agonist activity seen in slower step-tracking movements. AG2 has been thought to be characteristic of relatively fast movements, where it acts to stabilize the limb after movement termination (Ghez and Martin 1982; Meinck et al. 1984). The present data, in which both mean and maximum movement velocity (both well below the fastest possible) were constant across SRs, show, however, that the properties of AG2 are not related simply to movement speed.

One of the surprising findings was that clear, burstlike antagonist activity was present in movements with large and small SRs. Although previous studies have shown that the duration of ANT1 can change with both movement amplitude and speed (Brown and Cooke 1981; Mustard and Lee 1987), ANT1 duration appeared independent of the temporal characteristics of the movements studied here. This may simply reflect the fact that, for these movements, neither amplitude nor speed changed with SR. Thus the influence of the antagonist was exerted through changing the magnitude and timing of a burst of constant dura-
tion. The data suggest that the magnitude of the antagonist activity may be at a minimum in movements with SR close to those seen in step movements. It has recently been pro-
posed (Ghez and Gordon 1987) from studies on isometric movements that antagonist activity acts to “truncate the rising force. . . .” In the present work this would corre-
spond to a requirement for a rapid decrease of acceleration. As indicated by Ghez and Gordon’s work, one would ex-
pect in the present case that the magnitude of the antago-
nist activity would depend on the magnitude of the acceler-
ation: if the acceleration were large, it would take a large antagonist activity to decrease it rapidly to zero. The find-
ing that antagonist activity was comparable for both large and small SRs (which would have, respectively, small and large accelerations) argues against Ghez and Gordon’s in-
terpretation, at least as applied to the antagonist burst of the triphasic pattern. An alternative would be that antago-
nist activity subserves two functions, actively decreasing or shutting off the acceleration and producing the buildup of deceleration. In movements with large SRs, the antagonist magnitude would depend on the magnitude of the required
(large) deceleration. In movements with small SRs, the antagonist magnitude would depend on the magnitude of the (large) acceleration that it acts to decrease. Experimental evidence for a dual role of the antagonist muscle is presented in a subsequent paper (Cooke and Brown 1990).

What is common to the movements in the present study, where SR was changed, and movements in which amplitude and/or duration is changed? In all cases there are concomitant changes in accelerations and decelerations. For example, as the amplitude of movements of constant duration is increased, the magnitudes of both the acceleration and deceleration will increase. Similarly, if the amplitude is held constant but movement duration is increased, both the magnitudes and durations of acceleration and deceleration will change. In the common case where subjects are asked, for example, to "move at their own speed," changes in amplitude will be accompanied by changes in duration, with the result that both the magnitudes and durations of acceleration and deceleration will change. We therefore suggest that it is the desired acceleration/deceleration characteristics of the movement to be made that determine the EMG drive needed to produce the movement.

Given that we tend, for whatever reason, to make movements that are approximately equivalent under scalar transformation, the above analysis leads to an economic method by which the nervous system could produce the commands for movements of different properties. Knowledge of the desired movement amplitude and duration as well as the overall temporal structure (SR and c) are all that are required simply to determine the magnitudes and durations of acceleration and deceleration. The template used by the central nervous system in calculating the required commands could be as simple as a triangular velocity profile, with the precise form of the movement arising through the interaction of the descending command with the damping properties of the muscles as suggested by Stein et al. (1988). Indeed, the observations of Stein et al. suggest that a movement generated in this way would differ little from ones generated by such processes as minimizing jerk or energy. The use of such a system by the brain in programming the commands for movement would thus combine simplicity of calculation with approximation of energy minimization and movement smoothness.

We have presented evidence elsewhere that the magnitudes and durations of the various components of the triphasic pattern are related to the accelerative and decelerative properties of the movement in a relatively simple way, independent of such parameters as amplitude, duration, or velocity (Brown and Cooke 1987). The present findings would further suggest that the relations between magnitudes and durations of the components of the triphasic pattern and movement kinematics, such as amplitude and duration, arise because of associated changes in accelerations and decelerations. This hypothesis will be addressed directly in later papers in this series.

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