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Movement-related phasic muscle activation.

III. The duration of phasic agonist activity initiating movement

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Abstract To test the hypothesis that phasic muscle activation is related to the acceleration-deceleration characteristics of the resulting movement, we examined the relation between the duration of the acceleratory phase of a variety of movement types and the duration of the phasic muscle activity producing the acceleration (the initial agonist burst, AG1). Movements of five types were studied: (1) step-tracking movements of different amplitudes (10–90 deg) and durations (200–800 ms), (2) movements of the same amplitude (40 deg) and duration (600 ms) varying only in their symmetry ratio (SR, ratio of acceleration to deceleration durations), (3) movements in which acceleration duration was changed while acceleration magnitude was held constant, (4) oscillatory movements of different frequencies and peak amplitudes, (5) step-tracking movements against different inertial loads. Subjects made movements about the elbow joint in the horizontal plane. Surface electromyographic (EMG) activity was recorded from the biceps and the lateral head of the triceps muscles. Under all movement conditions tested and with acceleration duration ranging from 100 to 500 ms, acceleration duration varied linearly with the duration of AG1. Correlation coefficients for the linear regression lines ranged from 0.8 to 0.99. The slope of the best fit linear regression lines ranged from 0.5 to 1.6 and tended to be higher for extensions than flexions. The variations in slope may arise from differing mechanical properties of the biceps and triceps muscles, as well as from active forces produced in the antagonist. AG1 duration was unchanged by inertial loading when subjects kept acceleration duration constant. If subjects responded to an increase in inertial load with an increase in acceleration duration, there was a corresponding increase in AG1 duration. The data demonstrate a general relation between one characteristic of muscle activation (AG1 duration) and the resulting movement. The linear form of the relation is invariant across movement amplitude (range 10–90 deg), speed, duration (range 200–800 ms) and temporal profile (SR range 0.3–2.7), and is also independent of movement type (step, oscillatory). Such a general and simple relation between EMG and movement suggests that, at least to a first approximation, the nervous system can rather simply determine the muscle activation patterns needed to produce movements with desired characteristics.

Key words Movement · EMG · Acceleration
Burst duration · Human

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Introduction

This paper is the third in a series of studies on the relation between phasic muscle activation and voluntary movement in humans (Brown and Cooke 1990; Cooke and Brown 1990). The aim of these studies has been to determine what movement characteristics the motor system considers when planning or programming the motor commands required for a desired movement. We have approached this fundamental question by studying the relation between muscle activity (EMG) and the resulting movement. The EMG is reflective of the muscle forces involved in the movement and must therefore reflect the final commands responsible for movement generation. A finding of general or invariant EMG –movement relations would then suggest common or general rules used by the CNS in formulating movement commands.

What movement characteristics might the central nervous system consider when formulating the motor commands for a desired movement? In many simple, single-joint movements, muscle activation occurs in a typical 'triphasic' pattern (Brown and Cooke 1981; Hal-

lett et al. 1975; Wacholder and Altenburger 1926). An initial burst of agonist activity (AG1) is followed sequentially by a burst in the antagonist (ANT) and a second, less well defined agonist burst (AG2) (Hallett et al. 1975). We have recently shown that the antagonist burst consists of two separable parts, ANT1 and ANT2 (Cooke and Brown 1990). In trying to determine the function of the components of the triphasic pattern, most investigators have concentrated on the relations between the characteristics of the EMG pattern and such movement properties as amplitude, duration or speed. For instance, AG1 duration and magnitude increase with both movement amplitude and speed (Benecke et al. 1985; Berardelli et al. 1984; Brown and Cooke 1981, 1984; Hallett and Marsden 1979; Hoffman and Strick 1990; Mustard and Lee 1987). ANT magnitude has also been described as varying with movement amplitude (e.g., Brown and Cooke 1981; Hoffman and Strick 1990; Marsden et al. 1983; Mustard and Lee 1987; Wadman et al. 1979), although whether the relation is proportional or inverse is still unclear. The time of occurrence of ANT also varies with movement duration, appearing to be linked to the time of peak movement velocity (Hoffman and Strick 1990; Lestienne 1979; Sherwood et al. 1988; Wadman et al. 1979)

The function of the triphasic bursts has also been studied through altering the load being moved by the subject. Thus, for example, AG1 duration may increase with inertial load in the situation in which the subject is free to choose his own movement characteristics (movement strategy) (Angel 1974; Benecke et al. 1985; Berardelli et al. 1984). If, however, total movement duration is held constant, AG1 duration does not change with inertial load (Sherwood et al. 1988). Both AG1 and ANT magnitudes increase with increasing inertial load (Lestienne 1979; Karst and Hasan 1987; but see also Cooke et al. 1992).

In part, the dependence of the EMG on such movement characteristics as amplitude and speed was investigated because these movement properties appeared intuitively to be important. Until recently they were also the only movement characteristics which could be readily manipulated by the investigator. The development of the technique of phase plane tracking (Cooke and Brown 1986) provided us with a method for training normal humans to make movements having different temporal structures. Step-tracking movements (the most commonly studied movements) normally have a typically bell-shaped velocity profile (Nelson 1983; Ostry et al. 1987). The observation that the form of the velocity profile is invariant under transformations of movement amplitude and duration suggested that such movements belong to an equivalence class. That is, a common organizing principle may underlie the production of all such movements; the triphasic EMG pattern being, perhaps, indicative of a common motor program. However, using the phase plane-tracking technique to alter the shape of the velocity profile, we demonstrated that (1) a triphasic EMG pattern is used to produce movements not belonging to the same equivalence class and (2) striking changes in the characteristics of the triphasic EMG pattern occurred independent of any changes in movement amplitude, duration, or speed (Brown and Cooke 1990). This and our subsequent study on the EMG activity associated with movements made at constant velocity (Cooke and Brown 1990) led us to postulate that the properties of the various components of the triphasic pattern might best be related to the acceleration and deceleration properties of the desired movement (Brown and Cooke 1990). The same hypothesis may be arrived at by remembering that the EMG must, in some way, reflect the force being exerted by the muscle. If acceleration provides a measure of force (as is likely at least to a first approximation) then EMG should relate to acceleration. Such a relation has been suggested from the study of Wallace (1989) who found a strong correlation between EMG burst duration and the period of oscillatory forearm movements.

In the present study we have tested this hypothesis by determining the relation between AG1 duration and acceleration duration under a variety of movement conditions. Our aim was to determine whether the AG1 – acceleration relation has an invariant form independent of the type of movement being made (e.g., step tracking, oscillatory) and of various manipulations in movement characteristics (e.g., amplitude, duration, temporal profile). We further hypothesized that the increase in AG1 duration which results from increasing the inertial load (Angel 1974; Berardelli et al. 1984; Benecke et al. 1985) should be associated with an increase in acceleration duration. If acceleration duration is held constant, AG1 duration should not change with inertial load.

We will present data showing that the form of the AG1 – acceleration duration relation is very close to linear under all conditions tested and that manipulations such as loading – which alter acceleration duration – are associated with changed AG1 duration. Parts of the present data have been presented elsewhere in abstract form (Cooke and Brown 1989).

Materials and methods

Experiments were performed on 14 subjects aged 21–50 years with no known history of neurological disorders. A minimum of three subjects performed each of the experimental conditions to be described. Presented data are representative of all subjects tested. Each subject was seated comfortably and grasped a vertical rod attached to a horizontal manipulandum handle. The subject's upper arm was abducted 90 deg and supported at the elbow directly beneath the pivot point of the handle. Several different movement tasks were employed requiring flexion and extension movements about the elbow in the horizontal plane. In all tasks, both target and handle positions were displayed on an oscilloscope positioned approximately 1 m in front of the subject.

Movement paradigms

Step-tracking movements

In this task target position was displayed on the oscilloscope by a vertical bar which switched between two positions every 4 s. Target positions were symmetrically located about an elbow angle of approximately 90 deg. The angular position of the handle (and thus of the forearm) was displayed as a thin vertical line. The subject was required to superimpose the handle cursor on the target bar by making alternate flexion/extension movements. Subjects performed movements of four different amplitudes (10, 30, 60 and 90 deg) at two to four nominal durations. Durations ranged from 175 to 900 ms depending on movement amplitude, the durations being chosen in order to ensure that movements were initiated with phasic activity. The angular velocity was monitored by the experimenter, and the subject was verbally instructed to increase or decrease speed as required. Movements of each amplitude/duration combination were grouped into blocks of 30 movements (15 flexions, 15 extensions).

Movements with different symmetry ratios

Data for this set of experiments was derived from our previously published study (Brown and Cooke 1990) in which the paradigm was described. In brief, a template of the movement to be performed (based on a triangular velocity profile) was presented to the subject in the form of a phase plane (velocity vs position). This template remained on the oscilloscope screen while the subject reproduced it by moving a dot on the screen. The vertical and horizontal positions of the dot were controlled by the angular velocity and position of the handle respectively. Utilizing phase plane tracking as described above, subjects made 40 deg movements with different temporal profiles. All movements were of the same amplitude, duration, peak, and mean velocities. The temporal profiles were varied by altering the relative durations of the acceleratory and decelerator phases of the movement (termed the symmetry ratio, SR). SR ranged from 0.2 to 2.0. In this paradigm acceleration magnitude and duration covaried as SR was changed.

Phase plane-tracking movements

As noted above, in the preceding experiments acceleration duration and magnitude covaried. Experiments were thus performed in which the duration of the acceleration phase was systematically varied while acceleration magnitude was held constant. This was accomplished under three conditions: (1) Mean acceleration magnitude was kept constant with SR = 1.0. In this paradigm, movement amplitude, duration and maximum velocity varied as acceleration duration was varied. (2) Mean acceleration magnitude and total movement duration were constant. In this paradigm, movement amplitude and maximum velocity varied as acceleration duration was varied. (3) Mean acceleration magnitude and movement amplitude were held constant. Here, movement duration and maximum velocity varied with acceleration duration.

Oscillatory movements

In this paradigm, two target positions were displayed simultaneously. Movements of three amplitudes (10, 30, and 60 deg) were made with targets symmetrically located about an elbow angle of approximately 90 deg. The subject was required to move continuously back and forth between the two targets in time with an audio tone. Emphasis was placed on entering the target and reversing movement direction when the tone sounded. The desired frequency of the oscillatory movements (approximately 0.5–5.0 Hz) could be varied by changing the interval between the audio tones.

Inertial loading

In a further set of experiments, the inertial load was changed by adding mass to the manipulandum handle. Masses of 1.1, 2.3, or 3.4 kg were attached to the handle above the vertical rod held by the subject, approximately 30 cm from the pivot point of the handle. Subjects made step-tracking movements (30 deg, 'accurate') and phase plane tracking movements (30 deg, 0.5 s duration, SR = 1.0).

Data recording and analysis

The angular position and velocity of the manipulandum as well as surface EMGs from the biceps and lateral head of the triceps muscles were recorded. Angular position was derived from a precision potentiometer and angular velocity from a tachometer. In some experiments angular acceleration was directly recorded with an accelerometer on the manipulandum handle. Acceleration was otherwise obtained by digital differentiation of the velocity signal. Surface EMGs were recorded with paired disk electrodes (0.8 cm diameter) positioned about 2 cm apart over the bellies of the muscles. EMGs were filtered (20–2000 Hz) and full wave rectified before digitizing. Data were digitized on line with a sampling rate of 500 Hz.

The beginning and end of accelerations and decelerations were automatically determined using an acceleration threshold value of 120 deg/s^2. These computer-selected timing points were obtained for each movement individually and were later confirmed by visual inspection. Movements having obviously false timing points were rejected from the analysis. The start and end of AG1 were determined from individual movements using interactive graphics. Only those movements in which a clearly identifiable AG1 was

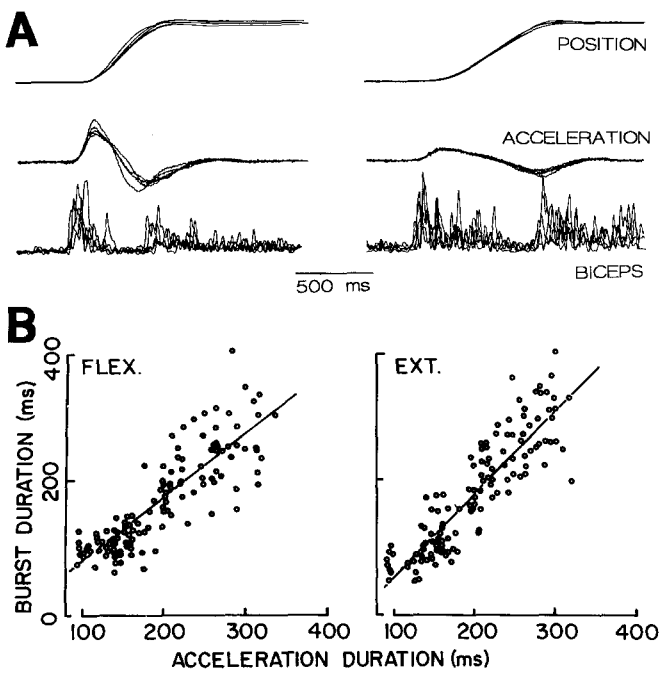

Fig. 1A,B Step-tracking movements. **A** Overplotted records from individual 30 deg flexion movements of two different durations Position, acceleration and the agonist (biceps) EMGs are shown. Records aligned to movement onset for plotting (DC161089). **B** Plots of AG1 duration (ordinate) as a function of acceleration duration for flexion and extension movements. Each plotted point is from an individual movement. Data are from movements of four amplitudes (10, 30, 60, and 90 deg made at different nominal durations (175–900 ms). Lines are best-fit linear regressions

present were used for analysis. An approximate threshold of 2 × baseline activity was used in defining burst start and end. Using these criteria some 10–15% of the records were rejected. All quantitative data to be presented were obtained from measurements on individual movements. Where averaged records are shown, the individual movement records were aligned to acceleration onset for averaging.

Results

Step-tracking movements

Subjects cannot readily exert direct control over acceleration duration in step-tracking movements. However, when the total duration of such movements is increased with movement amplitude held constant, there is a parallel increase in the duration (and decrease in the magnitude) of the acceleratory phase. Representative records from such step tracking movements are shown in Fig. 1A. Movements were initiated with phasic agonist activity, the duration of which increased with increasing movement duration.

The relation between AG1 duration and acceleration duration from such step tracking movements is shown in Fig. 1B. Data are shown from flexion and extension movements made at two to four different nominal durations at each of four amplitudes (10, 30, 60, 90 deg). AG1 duration increased with acceleration duration for both flexions and extensions. Correlation coefficients for the linear regression lines were 0.83 (flexions) and 0.88 (extensions). Since these graphs include data from all duration/amplitude combinations it is clear that the relation between AG1 duration and acceleration duration did not depend strongly on movement amplitude. The equations for the regression lines for movements of different amplitudes from this subject are given in Table 1. At each amplitude the AG1 duration - acceleration duration relation was linear with correlation coefficients ranging from 0.83 to 0.92. There was no consistent trend in either the slope or the intercept of the regression equations with movement amplitude, although the slope tended to be greater for extension than for flexion

Table 1 Regression equations of AG1 duration vs acceleration duration from step-tracking movements (AG1 duration = a + b × acceleration duration)

Amplitude (deg)	Flexions			Extensions		
	a	b	*r*	a	b	*r*
10[a]	–	*	–	− 35	1.12	0.74
30	−16	1.03	0.86	−122	1.55	0.92
60	−67	1.22	0.84	−118	1.47	0.90
90	4	0.81	0.84	− 90	1.19	0.85

[a] Insufficient points to establish a regression line for flexions

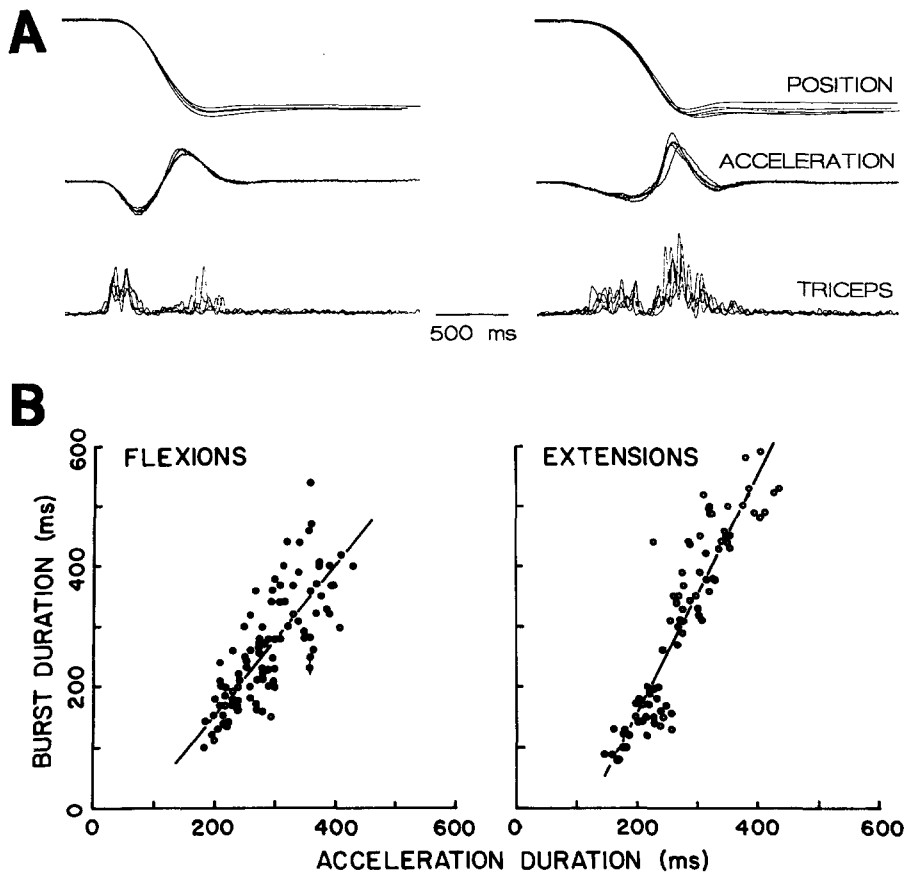

Fig. 2A,B Phase plane-tracking movements with varied symmetry ratios. **A** Overplotted records from individual extension movements with SRs of 1.0 (*left hand set*) and 2.5 (*right hand set*). Records aligned to movement start for plotting (DC151089) **B** Plots of AG1 duration as a function of acceleration duration from flexion and extension movements of different SRs. Each plotted point is from an individual movement. Movement amplitude was 40 deg and movement duration 0.6 s. SRs ranged from 0.3 to 2.7. Lines are best-fit linear regressions, ranging from 0.3 to 2.7

movements. This was also seen in most other movement conditions tested.

Phase plane-tracking movements

In the step-tracking movements just described, acceleration duration changed secondary to total movement duration. In order to allow us to explicitly control acceleration duration, experiments were performed using phase plane tracking. Two general types of tracking movements will be described: (1) movements in which the ratio of acceleration to deceleration durations (SR) was altered while maintaining movement amplitude, duration, and maximum velocity constant. These movements were the subject of a previous study (Brown and Cooke 1990). (2) Movements in which acceleration magnitude was held constant while acceleration duration was varied. These movements were studied under three conditions: constant SR, constant total movement duration, and constant movement amplitude.

Varying symmetry ratio

Figure 2A shows representative records from movements of two different SRs. For the left hand set, acceleration and deceleration durations were approximately equal (SR = 1). For the right hand set, acceleration duration was greater than deceleration duration (SR > 1). At each SR, movements were initiated by phasic agonist activity.

As we have recently shown, AG1 was often absent in movements with large SRs, appearing more as a gradual increase in tonic activity which was abruptly shut off around the time of peak velocity (Brown and Cooke 1990). The present movements were chosen from those with clearly 'burst-like' initial agonist activity. In Fig. 2B are shown the relations between AG1 duration and acceleration duration from this subject. Each point represents data from an individual movement with SRs ranging from 0.3 to 2.7. AG1 duration increased linearly with acceleration duration for both flexion and extension movements. The linear regression lines had correlation coefficients of 0.80 (flexions) and 0.93 (extensions).

Constant acceleration magnitude

In both the step tracking movements and those with variable SRs, acceleration duration and acceleration magnitude co-varied. Experiments were designed to investigate the relation between AG1 duration and acceleration duration when the acceleration duration was varied independently of acceleration magnitude. Utilising phase plane tracking, three conditions were tested, with acceleration magnitude constant in each condition. Figure 3 shows representative data from one condition. As seen in the velocity records in Fig. 3A, the subject made movements having the same SR (SR = 1.0) and acceleration magnitude but with different acceleration durations. Movement duration, maximum velocity, and amplitude varied with acceleration duration. The EMG records in Fig. 3B–D are from movements with the shortest to longest acceleration durations respectively. The duration of the agonist activity initiating the movement (AG1) increased with acceleration duration.

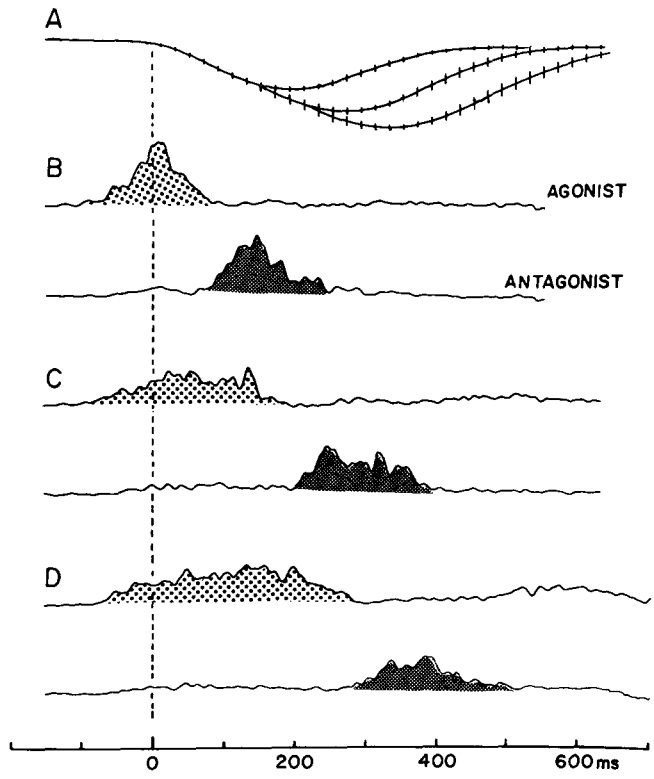

Fig. 3A–D Movements with varied acceleration duration. **A** Averaged velocity records from phase plane tracking, extension movements. Movements and EMGs were aligned about movement onset (*vertical dashed line*) for averaging. Movement parameters were chosen so that acceleration duration varied while acceleration magnitude and SR were constant. **B–D** show records of agonist (triceps) and antagonist (biceps) EMGs from movements with the shortest to longest acceleration durations respectively. AG1 and ANT bursts are indicated by *shading*. Note the increase in AG1 duration as acceleration duration increases

Figure 4 shows plots of AG1 duration as a function of acceleration duration under three different conditions as indicated by the diagrammatic velocity profiles in each panel. Data in Fig. 4A are from movements of constant SR as just described for Fig. 3. As noted above, changing the acceleration duration resulted in movements in which movement amplitude, duration, and maximum velocity varied. In Fig. 4B are shown data from movements in which the total movement duration was held constant. Changing acceleration duration resulted in movements of different amplitudes and maximum velocities. In Fig. 4C are shown data from movements of constant amplitude. In this case, changing acceleration duration produced movements of different total durations and maximum velocities. Under each of

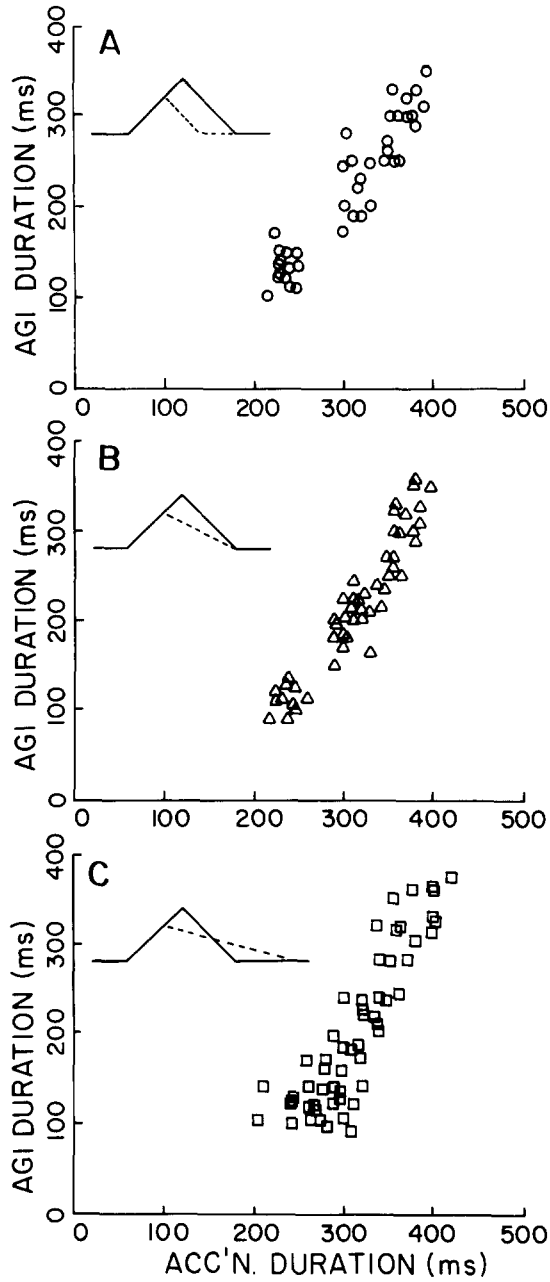

Fig. 4A–C AG1 – acceleration relations in movements with constant acceleration magnitude. Plots of AG1 duration as a function of acceleration duration from phase plane tracking extension movements in which acceleration magnitude was constant. Three conditions are shown (see velocity profiles in each panel): **A** movement SR constant and equal to 1.0; **B** total movement duration constant; **C** total movement amplitude constant. Each plotted point is from an individual extension movement

these three conditions, with acceleration magnitude constant (and the same across conditions), AG1 duration increased linearly with acceleration duration. Correlation coefficients for linear regression were 0.80, 0.90, and 0.87 respectively.

Table 2 Regression equations of AG1 duration vs half the cycle duration of oscillatory movements [AG1 duration = a + b × (cycle period)/2]

Amplitude (deg)	Isotonic movements					
	Flexions			Extensions		
	a	b	*r*	a	b	*r*
10	44	0.6	0.98	0	1.0	0.99
30	53	0.66	0.98	67	0.68	0.99
60	−3	0.84	0.99	45	0.72	0.97

Oscillatory movements

Typical records from movements made by one subject are shown in Fig. 5A. Desired movement amplitude (distance between flexion and extension targets) was 30 deg. As described in Materials and methods, the subject made cyclic, isotonic movements between the targets in time with an auditory cue. Records are shown from movements made at three frequencies. Alternating phasic activation of the biceps and triceps muscles occurred at all frequencies. The duration of the phasic activity increased with increasing period (decreasing frequency) of the movements (left to right panels).

The relations between AG1 duration and acceleration duration from such cyclic, isotonic movements are shown in Fig. 5B. Mean cycle durations (as measured from peak to peak acceleration) and EMG burst durations were calculated at each amplitude/frequency combination. Burst duration is plotted as a function of half the cycle duration which, for a pure sinusoid, would equal the acceleration and deceleration durations. Although the movements were not purely sinusoidal, the errors involved in using half the cycle duration will be small given the absolute cycle durations. At each amplitude, burst duration increased linearly with the half cycle duration. Correlation coefficients were 0.97 or greater (Table 2).

Inertial loading

In this series of experiments, subjects made step tracking movements with different masses attached to the handle. As illustrated in Fig. 6A, both AG1 duration and acceleration duration increased when the inertial load was increased. This was not, however, seen in all subjects. Figure 6C shows data from one subject in whom neither AG1 duration nor acceleration duration changed in response to increased loads. This observation led us to hypothesize that AG1 duration should be unchanged if subjects were forced to maintain a constant acceleration duration in the face of an increased inertial load. Utilizing phase plane tracking, we thus had subjects make movements in which they held the duration of the acceleration phase constant when different loads were applied. As illustrated in Fig. 6B, under

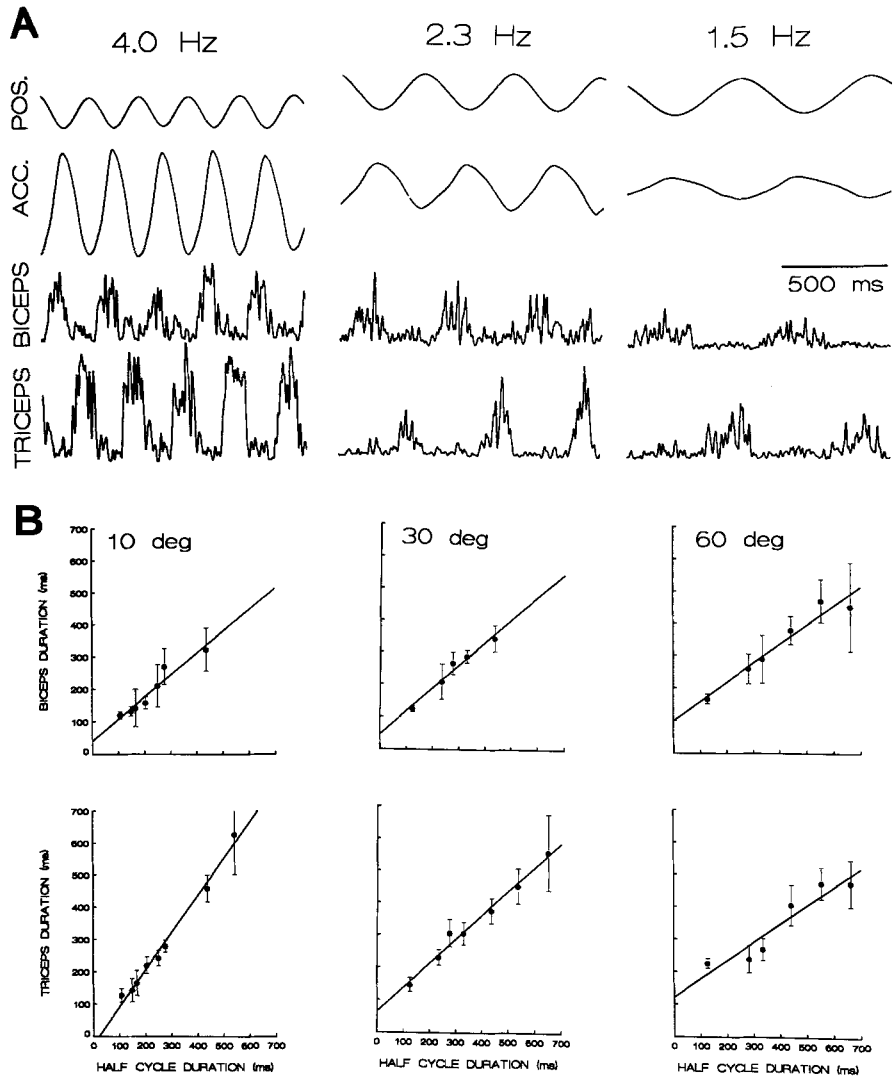

Fig. 5A,B Oscillatory movements. **A** Records of position, acceleration and biceps and triceps EMGs from oscillatory movements of three different frequencies (left to right panels). Note the increase in burst durations as movement frequency decreases (left to right) (DC161089). **B** Plots of average burst duration (±1 SD) as a function of half cycle period for movements of three different amplitudes. The *upper set of graphs* show data from the biceps and the *lower set* from the triceps. Each plotted point is the average from 10–15 movement cycles. Lines are best-fit linear regressions

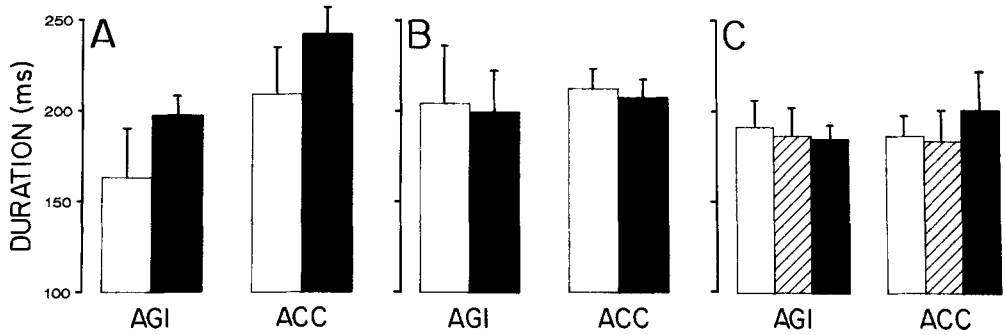

Fig. 6A–C Changes in AG1 duration with inertial loads. Mean AG1 and acceleration durations (±1 SEM) are shown under three conditions. **A** Forty-degree step-tracking movements. *Open bar* no load, *solid bar* 3.4 kg. **B** Phase plane-tracking movements (amplitude 40 deg, duration 0.6 ms, symmetry ratio 1.0). *Open bar* no load, *solid bar* 3.4 kg. **C** 30 deg step-tracking extension movements under three loading conditions; *open bar* no load; *hatched bar* 1.1 kg; *filled bar* 2.3 kg. Each histogram is the mean of 10–15 movements. **A** and **B** are from the same subject, **C** from another subject

these conditions AG1 duration did not change with load.

Discussion

As has been known since the time of Wachholder and Altenburger (1926), many movements are initiated by a brief period of phasic activation of the agonist muscle. This burst – termed AG1 – is the first in a regular se-

quence of phasic muscle activations which came to be called the 'triphasic' pattern (Hallett et al. 1975). The function of this burst of agonist activity is to accelerate the limb, and its magnitude has been shown to vary with such movement parameters as amplitude and speed (Brown and Cooke 1981; Hallett and Marsden 1979; Mustard and Lee 1987; Schmidt et al. 1988), as well as the inertial load being moved (Angel 1974; Benecke et al. 1985; Berardelli et al. 1985; Lestienne 1979). For many years the duration of AG1 was considered to be unaffected by changes in movement amplitude or duration. There were, however, brief reports of changes in AG1 duration with movement amplitude (Angel 1974; Wadman et al. 1979) which were subsequently confirmed and extended (Berardelli et al. 1984; Brown and Cooke 1984). As noted previously, Wallace (1989) described strong correlation between EMG burst duration and the period of oscillatory forearm movement. Benecke et al. (1985) also noted that the duration of AG1 increased with increasing total movement duration, a finding confirmed by Schmidt et al. (1988). Benecke et al. suggested that "...the duration of the first agonist burst determined movement time". This view of the functional role of the initial agonist burst was also put forward by Schmidt et al. (1988), who suggested that movement time was related to AG1 duration whereas movement velocity was controlled by both AG1 magnitude and duration. In a later paper, Sherwood et al. (1988) found that AG1 magnitude, but not duration, increased with the inertial load in movements of constant total duration. By manipulating the relative durations of acceleration and deceleration (called the symmetry ratio or SR), we have shown that the duration of AG1 varies with the temporal structure of movement independent of movement amplitude, total duration, maximum velocity, or mean velocity (Brown and Cooke 1990). This finding, coupled with those described above, suggested that there was a fundamental characteristic of movements to which AG1 duration was related and which was modulated under the various conditions tested by the different groups of experimenters. The most likely candidate for such a fundamental characteristic was the duration of the acceleratory phase of the movement (Brown and Cooke 1990).

The present experiments have, in fact, demonstrated a strong and simple relation between AG1 duration and the duration of the acceleratory phase of the resulting movement. This relation was linear with a very high correlation under all conditions tested. A similar finding has recently been reported for oscillatory movements by Wallace (1989), who reported that movement frequency changed as a result of the subject voluntarily altering movement amplitudes. It is of interest that the mean value of the slope of the biceps burst duration -half wave length of oscillation in that study was 0.76, a value in excellent agreement with that found in the present study. Whether the apparent difference in slopes of the relation for flexion and extension movements is real cannot be determined from our experiments. Wallace also found considerable variation in the slopes of the relation (ranging from about 0.14 to about 1.06). It is possible that the differing mechanical properties of the responsible muscle groups could lead to a different gain in this input-output relation while preserving its overall form. Viscous and elastic forces would also affect the relation, as would any early activity in the antagonist muscle. Thus, although in a purely inertial system the slope of this relation should be 1.0, a number of factors may affect the actual value in different movements.

Our data nonetheless indicate a relatively simple and robust relationship between part of the command for movement (the duration of the phasic muscle activation initiating movement) and a kinematic feature of the resulting movement (acceleration duration) which may be common to many types of movement. This finding helps unify a rather disparate collection of observations on the relations between AG1 duration and various kinematic features of movement. While it is certainly correct, for example, that AG1 duration normally changes with movement amplitude or duration as well as with other manipulations, the relations between AG1 and such kinematic variables are clearly secondary to the relation uncovered here between AG1 duration and acceleration duration. For example, the suggestion that " ... the duration of the first agonist burst determined movement time" (Benecke et al. 1985) is not correct. In an earlier paper (Brown and Cooke 1990) we showed that AG1 duration can vary widely with no change in movement duration. It is certainly correct that, under many conditions, AG1 duration and movement duration are related. This is not due, however, to AG1 determining movement duration but rather to the fact that such movements remain relatively time-symmetric as movement duration is increased. As a result, acceleration duration must be increased and this is accomplished by increasing AG1 duration.

In terms of determining the desired duration of the initiating muscular activity we would distinguish between the control of movement characteristics through muscle activation - movement relations and the determination of the desired movement characteristics in relation to different behavioral 'strategies' (Corcos et al. 1989; Gottlieb et al. 1989; Sherwood et al. 1988). This is perhaps best illustrated by our experiments with inertial loading. As has been reported previously (Angel 1974; Benecke et al. 1985; Berardelli et al. 1984) AG1 duration may increase with inertial load in the situation in which the subject is free to choose his own movement characteristics (movement strategy). However, it is clear from the present work and from Sherwood et al. (1988) that AG1 duration does not *of necessity* increase with load. If the subject is required to maintain the relevant movement characteristics (in particular acceleration duration) constant, then AG1 duration does not change with inertial load. While it is not the usual response to an increased inertial load, subjects may choose this behavioral strategy spontaneously, as did one of our four subjects. Thus, while the subject may by choice or through

the constraints of the task alter his behavioral response under different conditions, the control of movement characteristics through the EMG - movement kinematic relations does not change.

A word of caution must be interjected, however. In a previous paper (Cooke and Brown 1990) we showed that paired agonist/antagonist activation was related to limb acceleration when acceleration was temporally independent of deceleration in movements made at constant velocity. Movement (acceleration) was initiated with an agonist burst (AG1) closely followed by an antagonist burst (ANT1) which could be partially co-extensive in time with the agonist burst. Our interpretation of the functions of these two bursts was that AG1 produced the buildup of acceleration and ANT1 actively decreased acceleration to zero. [Note that ANT1 in this context is not the antagonist burst commonly associated with the triphasic pattern. Rather it corresponds to the early antagonist activity which is often seen in the triphasic pattern partially coextensive with the initial agonist burst (e.g., Brown and Cooke 1981 1986; Hallett and Marsden 1979).] This function for the antagonist is similar to that suggested by Ghez and his coworkers for the antagonist burst seen in rapid isometric movements (Gordon and Ghez 1984). For isometric movements, Ghez and Gordon (1987) described a range of movement durations in which no antagonist activity was present (their 'b' range; see Fig. 5 in Ghez and Gordon 1987). In this case muscle force decreased passively from its maximum value. In isotonic movements in the absence of ANT1, the force produced by AG1 would passively decrease resulting in a decrease of acceleration with a time course determined by the muscle and limb properties (Lestienne 1979). If, however, ANT1 is present, it is clear that the duration of the acceleratory phase will depend both on the duration of AG1 and the properties of ANT1. We have been unable thus far to have subjects independently manipulate the rising and falling phases of acceleration and therefore can only speculate on the interaction between AG1 and ANT1 in determining the temporal characteristics of the acceleratory phase of movement. It is, however, possible that the variations in slopes of the AG1 – acceleration duration curves seen in this study are in part related to the presence or absence of this early antagonist activity in some situations.

One apparently unexplained observation is that of Sherwood et al. (1988), who found a small (29 ms) increase in AG1 duration with amplitude (range 20–70 deg) in movements in which acceleration duration was quite constant. The movements studied by Sherwood et al. had acceleration durations of approximately 140 ms and AG1 durations of about 150–170 ms, which are at the very low end of the movements studied here (see Fig. 2). In previous studies (Benecke et al. 1985; Brown and Cooke 1984) it was shown that with AG1 durations of about 140 ms or less, the initial agonist burst was segmented and consisted of one or two sub-components each of approximately 70 ms duration. It is thus possible that the motor system utilizes a different strategy in generating movements with such short acceleration durations.

The finding of commonality between movements of different types is in agreement with our earlier suggestion (Brown and Cooke 1990) that the brain may be able to utilize relatively simple rules in setting up the commands for movements. In the case of determining or choosing the appropriate duration for AG1, the least information needed is the desired duration of the resulting acceleration. This information is simply obtained from knowledge of the desired temporal properties of the movement to be made. In the case of step movements, this would be movement duration and the relative times to be spent in acceleration and deceleration (which for most movements are approximately equal). In the case of oscillatory movements, it would be the cycle period of the oscillation. Although, as discussed previously, acceleration duration will be affected by early antagonist activity, this simple relation would at the least provide a good approximation for choosing burst duration.

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