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# AMPLITUDE- AND INSTRUCTION-DEPENDENT MODULATION OF MOVEMENT-RELATED ELECTROMYOGRAM ACTIVITY IN HUMANS

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SUMMARY

1. Studies were made of the electromyogram (EMG) patterns associated with the performance of visually guided, step-tracking arm movements by normal humans. Subjects were instructed to make movements either 'accurately', 'as fast as possible' or 'fast and accurately'. Movements of 16, 32, 48 and 64 deg of arc were made with each instruction. Movements had durations of approximately 250–600 msec.

2. A 'triphasic' pattern of EMG activity was associated with all movements in this study. All bursts in this pattern were more clearly defined in faster movements whether the increased speed of movement was a result of increased movement amplitude or of the instruction-related 'intent' of the subject.

3. The magnitudes of the two agonist EMG bursts showed identical linear dependencies on movement amplitude. The slope of this relation was instruction-dependent, being greatest for 'fast' and least for 'accurate' movements.

4. The duration and time of onset of the initial agonist burst relative to the start of the movement were not dependent on movement amplitude or on instruction. In contrast, the time of onset of the second agonist burst depended on both movement amplitude and instruction, occurring earlier when movements were made faster.

5. The magnitude of the antagonist activity was instruction- but not amplitude-dependent. Duration and onset of this burst varied with both instruction and movement amplitude.

## INTRODUCTION

For at least the past fifty years students of motor behaviour have considered there to be two general types of movement, fast and slow (e.g. Stetson & McDill, 1923). A further type of fast movement, the 'ballistic' movement, remains a common concept (see for example Brooks, 1979; Ghez, 1979; McKay & Murphy, 1979). Indeed a recent hypothesis suggests that not only are fast and slow movements qualitatively different they are also generated or controlled by anatomically distinct parts of the brain (Kornhuber, 1971).

What are the distinguishing features of these different types of movement? The first obvious one is speed. Indeed, a common view of ballistic movements is that they are made at such a speed that the movement is completed before there has been time

for its possible modification by the action of afferent feedback. It is, however, clear that velocity of movement is dependent on (at least) two factors: movement amplitude and on what the person moving 'wants' to do. Thus there is a linear relation between peak velocity of movement and movement amplitude (Bouisset & Lestienne, 1974; Cooke, 1980). The slope of this relation is greater if the subject is instructed to move 'as quickly as possible' than if he is told to move 'as accurately as possible' (Cooke, 1980). Movements of equal speed can thus be made by different combinations of amplitude and 'instruction' and one can speak of amplitude- or instruction-dependent velocity

An apparently more precise distinction between fast and slow movements has been made on the basis of the electromyographic (EMG) activity associated with the movement. A 'triphasic' pattern has been associated with 'fast' movements (Garland & Angel, 1971; Hallett, Shahani & Young, 1975). This pattern is initiated by a burst of activity in the agonist which starts before movement onset. A second burst of agonist activity is separated from this initial burst by a period of relative EMG silence. During this 'silent period' a burst of activity is seen in the antagonist. In contrast, during so-called 'slow' movements more or less continuous electrical activity is observed from the agonist (Hallett *et al.* 1975) which may or may not be accompanied by co-contraction of the antagonist.

An alternating pattern of agonist–antagonist activity is also seen in 'ballistic' movements (Stetson & McDill, 1923; Conrad & Brooks, 1974; Desmedt & Godaux, 1978; Ghez, 1979; Hallett & Marsden, 1979). The distinguishing feature of these movements has been that the duration of the initial agonist burst is constant, only its magnitude being altered by experimental manipulation (Ghez, 1979).

Preliminary studies in this laboratory indicated that movements of different amplitude and made under a variety of instructions may have a number of features in common (Cooke, 1980). None of the movements studied was obviously ballistic; they were all made at speeds well below the maximum possible by the subjects. They thus appeared to fall somewhere between classical fast and slow movements. The present study was undertaken to investigate the detailed structure of EMG activity associated with movements made between the extremes of ballistic and slow movements.

## METHODS

### *General*

Experiments were performed on nine normal subjects with no known history of neurological disorder. All subjects were right-handed and experiments were performed utilizing alternate flexion–extension movements about the right elbow. Subjects were seated comfortably and grasped a vertical rod which was attached to a manipulandum handle (Thomas, Croft & Brooks, 1976). The subject's arm was positioned so that the elbow was beneath the pivot point of the handle, which was free to move horizontally. The arm was supported just distal to the elbow.

During experiments the subject was required to make visual step-tracking movements. The target which was to be followed was displayed as a vertical bar on an oscilloscope positioned about 1 m in front of the subject. Target width was indicated by the width of the bar. The targets were not mechanically detectable and were not bounded by mechanical stops. Also displayed (as a vertical line) was the handle position derived from a precision potentiometer placed at the pivot point of the handle.

*Experimental paradigm*

During an experimental session the subject was asked to make a series of step-tracking movements of each of four different amplitudes (16, 32, 48 and 64 deg of arc). Movements of each amplitude were made under each of three instructions. The instructions to the subject were:

(1) 'Make the movements as *accurately* as possible.' The subject was told to move from target to target in one smooth movement. He was not to overshoot the target and return, nor was he to undershoot and reach the target in a final corrective movement. These movements are termed 'accurate'.

(2) 'Make the movement as *fast* as possible.' The emphasis in this instruction was on speed. The subject was allowed to overshoot if he so desired. He was free to pick the strategy which he felt permitted him to reach the target as rapidly as possible. These movements are termed 'fast'.

(3) 'Make the movements as *fast and as accurately* as possible.' For these movements the subject was required to increase his speed as much as possible while retaining accuracy. These movements are termed 'fast and accurate'.

Each experimental session thus consisted of a series of twelve trials (three instructions by four movement amplitudes). Trials of different amplitudes and instruction were randomly ordered during the experiment, and were separated by a 2 or 3 min rest period during which new target positions were set, etc.

*Data recording*

The primary data recorded were the angular position and velocity of the manipulandum handle and the surface electromyograms (EMGs) from the biceps muscle and the lateral head of the triceps muscle. Handle position was obtained from a precision potentiometer and angular velocity from the back e.m.f. induced in a small linear d.c torque motor by the handle movement. EMGs were recorded from the muscle surface with disk electrodes, 0·8 cm in diameter, spaced 4–5 cm apart over the muscle surface. EMGs were filtered (low-frequency cut-off 20 Hz, high-frequency cut-off 2000 Hz) and full-wave rectified before recording. All data were digitized on-line with an effective sampling rate of 500 Hz (1000 Hz with two point averaging), collected on disk and transferred to magnetic tape for later off-line analysis.

*EMG standardization*

In order to compare quantitatively EMG data obtained from different subjects or from the same subject on different days, a method was needed to standardize the EMG recordings. The method chosen utilized the well-known linear relation between integrated surface EMG and isometric force (Lippold, 1952; Milner-Brown & Stein, 1975). This approach has recently been used by Hallett & Marsden (1979). In order to calibrate the curve, the subject opposed a torque of 0·8 Nm and the gain of the EMG amplifier was adjusted so that a fixed value was obtained for the integrated EMG in all subjects. The average linear regression equation for the EMG–isometric force relation from nine subjects had a correlation coefficient of 0·97 for biceps activity and 0·96 for triceps activity. The calibrated curve did not change over repeated trials or over the course of a tracking experiment. Although it is clear that such a relation between EMG activity and isometric force may not be applicable in determining force production during isotonic movements, this technique nonetheless provided a method for standardizing EMG recording between subjects that allowed averaging of data obtained from different subjects.

## RESULTS

### *Effects of amplitude and instruction on the triphasic pattern*

Shown in Fig. 1 are average records obtained from 'fast' flexion movements made by one subject. The typical triphasic pattern is apparent (Fig. 1*D*): an initial burst in the agonist (B1) (after Hallett *et al.* 1975) is separated from a later period of agonist activity (B2) by a period of relative EMG silence. During the interval between agonist bursts there is a burst of activity in the antagonist (T1). Antagonist activity was often seen preceding the antagonist burst (e.g. Fig. 1*C*), partially overlapping in time with

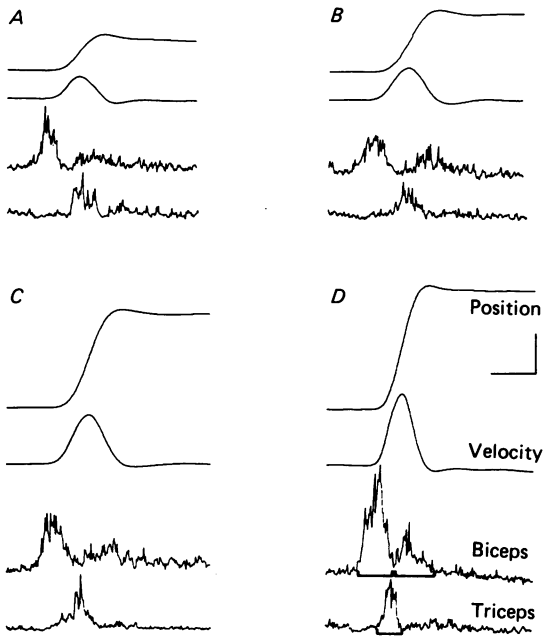


Fig. 1. Triphasic EMG activity associated with movements of different amplitude. The traces in *A–D* show averaged records of position, velocity, and biceps and triceps EMGs from flexion movements of different amplitudes (*A*, 16°; *B*, 32°; *C*, 48°; *D*, 64°). All traces represent the average of fifteen movements. The horizontal bars in *D* indicate the defined bursts as used for analysis (see Methods). The vertical calibration in *D* represents 25° for position and 300°/sec for velocity. The horizontal calibration represents 200 msec. (PM020379)

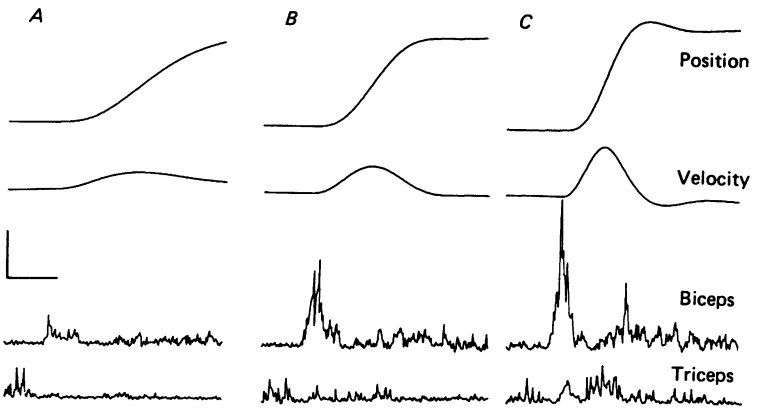


Fig. 2. Triphasic EMG activity associated with different instructions. Each set of traces in *A–C* shows records of average position, velocity, and biceps and triceps EMGs during performance of 48° flexion movements. Instructions to the subject were: *A*, 'move as accurately as possible'; *B*, 'move both fast and accurately'; and *C*, 'move as fast as possible'. Each set of traces is the average of fifteen flexion movements. The gain on EMG recordings in *A* and *B* is twice that in *C*. Vertical scale: 25°; 300°/sec; horizontal scale: 200 msec. (EB030479)

the first agonist burst. This activity occurred at a constant latency relative to the onset of the first agonist burst in movements of different amplitudes and speeds and became more 'burst-like' as movement amplitude was increased. It was also more prominent in 'fast' as opposed to 'accurate' movements.

The triphasic EMG pattern is apparent at the four amplitudes of movement shown in Fig. 1. All three components appear to increase in magnitude with increasing movement amplitude. Similar records are shown in Fig. 2 for movements of the same amplitude made under the different instructions. The triphasic pattern is most apparent in the 'fast' movements (Fig. 2*C*). An initial agonist burst is clear in both the 'accurate' and the 'fast and accurate' movements (*A* and *B*), as is a period of later increased agonist activity. Activity is present in the antagonist (triceps) in the interval during which the agonist is relatively silent. This antagonist activity also becomes more 'burst-like' as the speed of movement is increased. Additionally, the magnitude of each of the EMG bursts appears to increase as movement velocity is increased (*A–C*).

The movements performed and the EMG activity underlying them depended, as has been seen, both on the inter-target distance and on the instruction given to the subject. Common to both these manoeuvres was a change in movement velocity. The magnitude of the EMG bursts increased as movement velocity increased (Figs. 1 and 2). For the movements studied here there was a linear relation between peak velocity and movement amplitude. As was seen in an earlier study (Cooke, 1980) the slope of this relation was instruction-dependent, with 'fast' movements showing the greatest and 'accurate' movements the least slope. 'Fast and accurate' movements had a slope intermediate between the other two. There thus appear to be two distinct ways to alter the velocity of a movement: either by changing the inter-target distance or by asking the subject to change his speed voluntarily. Velocity at constant movement amplitude then provides a measure of the subject's 'intent'. Since the magnitude of the EMG bursts appeared velocity-dependent, a detailed analysis of EMG activity was split into two parts: the first is the dependence on movement amplitude and the second the dependence on instruction-dependent velocity.

*Modulation of timing and magnitude of EMG bursts*

*The initial agonist burst* (*B1*). Data on the onset, duration and magnitude of the initial agonist burst are shown in Fig. 3. The onset and duration of the EMG bursts were determined from inspection of computer-generated plots of averaged data (see for example Fig. 1*D*). Magnitudes were obtained by computer integration to obtain the area under the previously determined bursts. Graphs in the top row (*A–C*) show data from movements of different amplitudes and those in the bottom row (*D–F*) from movements made under different instructions. On the average across all movements the initial agonist burst started $55 \pm 8$ msec before movement onset. Analysis of the linear regression equations for the curves in Fig. 3*A* and *D* showed that this onset time was independent of both movement amplitude and the subject's 'intent' (instruction). The duration of this burst was also relatively constant under all conditions (Fig. 3*B* and *E*). Although the graph in Fig. 3*B* suggests a trend for this burst to increase in duration as movement amplitude increased, regression analysis indicates that any such change was non-significant.

The magnitude of the initial agonist response (determined by integrating the EMG activity over the duration of the burst and normalizing to a duration of 100 msec) was linearly related to both movement amplitude and instruction-dependent velocity (Fig. 3*C* and *F*). The dependence on movement amplitude was itself instruction-dependent, with 'fast' movements giving the greatest slope and 'accurate' movements the least. Note that since, for these movements, peak velocity is linearly related to

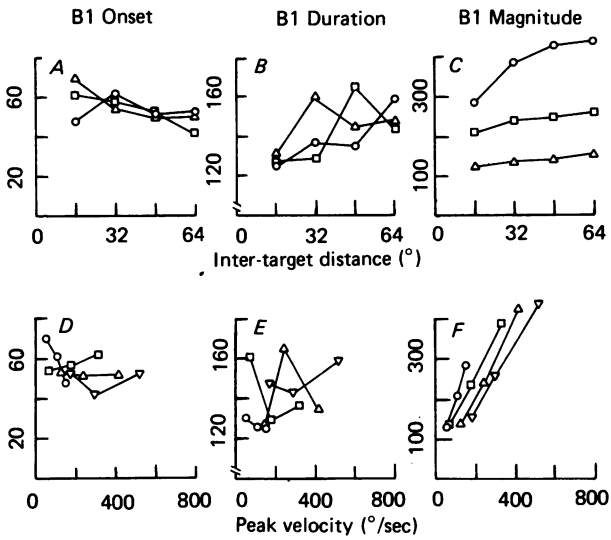


Fig. 3. Dependence of the first agonist burst (B1) on movement amplitude and instruction-dependent velocity. Onset, duration and magnitude are plotted as a function of movement amplitude (*A–C*) and instruction-dependent velocity (*D–F*). All data represent the averages from seven subjects. In *A–C* data are shown from 'fast' (circles), 'fast and accurate' (squares) and 'accurate' (triangles) movements. In *D–F* data is from 16° (circles), 32° (squares), 48° (triangles) and 64° (inverted triangles) movements. Onset and duration are in msec and magnitude in arbitrary units.

movement amplitude, the magnitude of the initial burst must change linearly with the amplitude-dependent velocity. The magnitude of this burst was also linearly related to instruction-dependent velocity (Fig. 3*F*). This relationship was, however, not strongly dependent on movement amplitude.

*The late agonist burst (B2).* In contrast to the findings on the initial agonist burst, both the duration and time of onset of the second burst of agonist activity were strongly dependent on both instruction and movement amplitude (Fig. 4). As inter-target distance (and thus movement velocity) increased, the later agonist activity was progressively delayed relative to the time of movement onset (Fig. 4*A*). However, the opposite was seen when movement velocity increased due to instruction to the subject (Fig. 4*D*): in this case the late agonist burst occurred progressively earlier with faster movements, the effect appearing more pronounced with movements of larger amplitude.

A similar difference between the effects of movement amplitude and instruction

was seen with the duration of this late agonist burst (Fig. 4*B* and *E*). Here the duration varied little with movement amplitude but progressively decreased as instruction-dependent velocity increased. It should be noted that the end of the second burst of agonist activity was sometimes difficult to determine. This was particularly so for the 'accurate' movements, where the magnitude of this response was rather low. A similar problem was encountered by Hallett & Marsden (1979).

Perhaps surprisingly, the magnitude of the second agonist response appeared to depend on movement amplitude and instruction in much the same fashion as did the

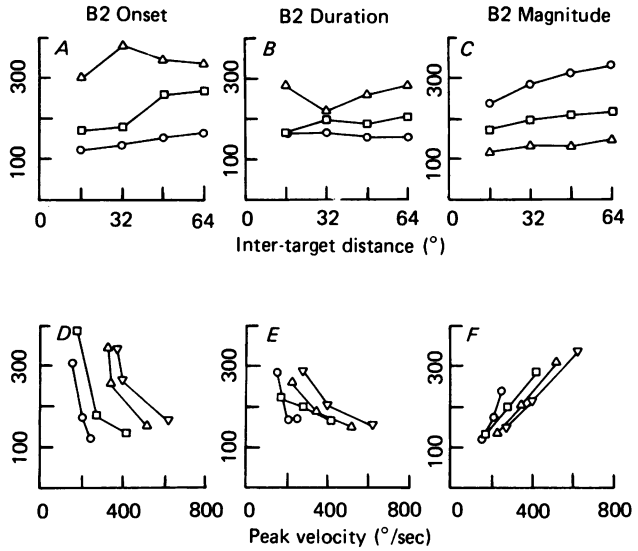


Fig. 4. Dependence of the second agonist burst (B2) on movement amplitude and instruction-dependent velocity. Onset, duration and magnitude are plotted as a function of movement amplitude (*A–C*) and instruction-dependent velocity (*D–F*). All conventions and symbols as in Fig. 3.

initial burst. That is, the magnitude of the second burst increased as a function of both movement amplitude and of instruction-dependent velocity (Fig. 4*C* and *F*). As with the initial burst, the dependence was greatest for the 'fast' movements and least for the 'accurate' movements.

*The antagonist burst (T1).* Unlike activity in the agonist, activity in the antagonist was strongly dependent on both movement amplitude and on instruction. Both the time of onset relative to movement start (Fig. 5*A*) and the duration of the antagonist burst (Fig. 5*B*) increased with movement amplitude. Thus, both these parameters increased as amplitude-dependent velocity increased. In contrast, both the time of onset and the duration of the antagonist burst decreased as the instruction-dependent velocity increased. That is the antagonist burst occurred earlier in the movement and was of shorter duration for 'fast' than for 'fast and accurate' movements. No data are available for the 'accurate' movements as their antagonist activity was small and not clearly 'burst-like'.

The magnitude of this antagonist activity was quite independent of movement amplitude (Fig. 5*C*). This is in contrast to the activity in the agonist where both agonist bursts increased as movement amplitude was increased. However, as was found with the agonist activity, the magnitude of the antagonist burst was instruction-dependent, being greater for 'fast' than for 'fast and accurate' movements.

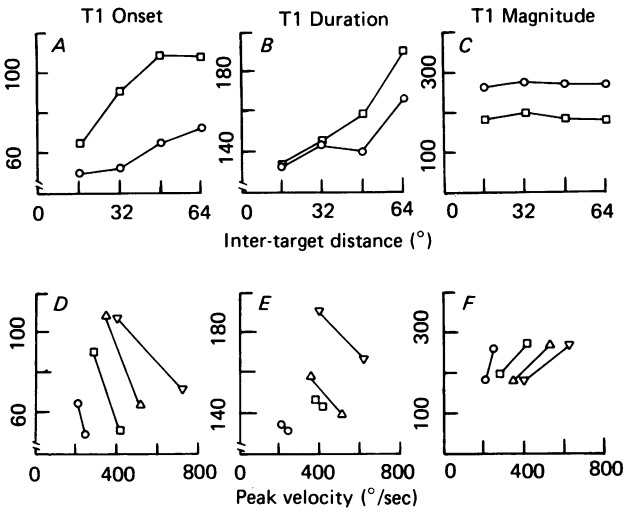


Fig. 5. Dependence of the antagonist burst (T1) on movement amplitude and instruction-dependent velocity. Onset, duration and magnitude are plotted as a function of movement amplitude (*A–C*) and instruction-dependent velocity (*D–F*). All conventions and symbols as in Fig. 3.

## DISCUSSION

### *Generality of the triphasic pattern*

A triphasic pattern of EMG activity has been described in association with fast movements (Wacholder & Altenburger, 1926; Angel, 1974; Hallett *et al.* 1975; Hallett & Marsden, 1979). It has also been stated that such a pattern is seen with 'ballistic' movements (Hallett, 1979; Hallett & Marsden, 1979). Clearly, as has been shown here, a triphasic pattern of EMG activity is associated with a wide range of movements, from 'fast' to 'accurate' in the terms used here. Movement peak velocities in the present study, for example, ranged from approximately 50–450 °/sec. Hallett *et al.* (1975) reported a more or less continuous, low-amplitude agonist EMG activity in what they termed 'smooth' movements. It would seem that in the present study movements ranged between the two extremes of 'smooth' and 'ballistic'. That is, on the basis of their burst-like EMG pattern our 'accurate' movements appear slightly faster that the 'smooth' ones of Hallett *et al.* On the other hand, none of the movements studied here fits the common meaning of ballistic; they were all made well below the subject's maximal velocity and their durations were such as to allow ample time for afferent feedback.

One other criterion which has been put forward as characteristic of ballistic

movements is an invariance in the duration of the initial burst of agonist activity (Ghez, 1979). Such a constancy of the initial agonist burst was also seen in all movements in this study. Since, as was just pointed out, these movements could not be considered as ballistic, it seems more likely that such a feature is characteristic of the performance of a step movement rather than of a particular type of step movement (i.e. ballistic). Certainly such a burst is not seen in what have been called ramp or smooth movements (Hallett *et al.* 1975).

*Control of burst magnitudes*

Studies by previous workers have indicated that the magnitude of the initial burst of activity in the agonist is related to the amplitude of the movement (Ghez, 1979; Hallett & Marsden, 1979). Such an effect was also seen in this study (Fig. 3).

Thus the magnitudes of the initial and the late agonist bursts are linearly related to the desired movement amplitude. This was seen for all movements studied here. The slope of the relation was instruction-dependent, being greatest for the 'fast' and least for the 'accurate' (and slowest) movements. Of particular interest is the fact that the dependence of agonist burst magnitude on desired movement amplitude is the same for both agonist bursts. That is, the curves relating the magnitudes of both agonist bursts to desired movement amplitude are parallel with a slope which depends on the instruction given to the subject. In contrast, the antagonist burst is independent of desired movement amplitude for the two instructions during which it could be accurately determined.

There thus appear to be (at least) two distinct ways by which the magnitudes of the agonist burst can be modified: by altering the desired movement amplitude or by altering the 'intent' or 'set' of the subject by instruction. Any such intent-related modification is performed equally on both bursts (the curves of agonist burst magnitudes *vs.* movement amplitude are parallel for each instruction). That is, in terms of changing the magnitude of the agonist activity, both agonist bursts are treated as a unit.

Activity in the antagonist is not modifiable in the same way as is the agonist activity. The agonist burst magnitude is independent of desired movement amplitude (Fig. 5) but can be graded by the instruction to the subject. Under any one instruction the antagonist burst thus appears to provide a constant background against which the modifiable agonist activity operates.

*Control of timing of bursts*

Although the observed changes in the magnitude of agonist bursts indicate that they share a common source or control, the observations on their onset and durations indicate that they are independent. Thus, although initial agonist burst onset and duration appear relatively fixed (Fig. 3), the onset of the late burst can indeed be altered (Fig. 4). This increased onset time of the late burst as a function of movement amplitude (Fig. 4*A*) implies that the duration of the interval between bursts has increased (since onset and duration of the early burst are relatively constant). At the same time as this interval is increasing, the duration of B2 is constant. The suggestion is thus that the late burst is an independent one of fixed duration whose onset is being delayed in the movements of larger amplitude. In addition, of course, the duration

of the late burst can be modified by the instruction to the subject (Fig. 4*E*), being shorter for 'fast' than for 'accurate' movements.

*Generation and modulation of the triphasic pattern*

Four conclusions may be drawn concerning the generation and control of the movement-related triphasic EMG activity. First, a wide range of movements appear to be generated by modulation of a common pattern of EMG activation. Secondly, in terms of their magnitude, the agonist bursts are treated as a unit. Thirdly, in terms of their onset time relative to movement onset and in terms of their durations they appear to be under separate control. Fourthly, activity in the antagonist is related to the subject's 'intent' and provides a background against which the agonist muscle operates. The so-called 'pulse–step' model of EMG activity (Ghez, 1979) would agree with the present observations, the pulse corresponding to the early and the step to the late agonist bursts. Thus, a command to move would result in the generation centrally of a pulse of activity followed by a longer duration step. Since the timing of the two agonist bursts appears independently variable, the pulse and the step generator may be separate. However, since their magnitudes appear to be under common control, the pulse and the step may go through some common gain control mechanism, perhaps at the spinal cord level.

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