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Relationship between EMG patterns and kinematic properties for flexion movements at the human wrist

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Summary. EMG patterns associated with voluntary wrist flexion movements were studied in normal human subjects. Initially, subjects were trained to produce movements within five specified velocity ranges while the amplitude of the movement and the opposing load remained constant. In a second set of experiments, subjects were required to produce movements at four different amplitudes, moving as rapidly as possible against a constant load. Finally, with movement velocity and amplitude kept constant, the external load was varied so that different forces were required to generate the movements. The slowest movements were associated with a prolonged burst of EMG activity from the agonist muscle with little or no antagonist activity. With increasing movement velocity, there was a gradual evolution to the characteristic "triphasic" pattern associated with rapid voluntary movements. As velocity of movement increased further, the amplitude and area of the EMG bursts increased while burst duration and interburst intervals decreased. Increases in movement amplitude were accomplished mainly by changing the timing of the EMG bursts; with larger amplitude movements the antagonist burst occurred later. With movements against larger loads there was an increase in the size of the agonist burst and a decrease in the antagonist burst, but no change in the relative timing of the EMG bursts. These systematic changes in EMG patterns associated with different types of movement provide an indirect method of obtaining information concerning the motor programs which generate the movements.

Key words: EMG patterns – Motor programming – Ballistic movements

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Introduction

Initial attempts to perform an unfamiliar movement are slow and awkward. With repeated practice the movement becomes more precise and stereotyped, suggesting that the subject has developed a "motor program" for that particular movement. If the movement is restricted to a single joint and directed to a specific target, a characteristic pattern of EMG activation is seen from the participating muscles (Hallett et al. 1975; Hallett and Marsden 1979; Brown and Cooke 1981). An initial burst of activity from the agonist muscle produces the force required to overcome inertia and set the limb moving. This is followed by an EMG burst from the antagonist muscle which provides a mechanism for braking the movement (Lestienne 1979). Subsequent smaller EMG bursts from both muscles generate the final adjustments to bring the limb to the target zone.

Variations in the speed, amplitude, or force of a movement can be produced by alterations in the motor program. For simple one-joint movements, these changes are associated with adjustments in the size and dimensions of individual EMG bursts or their relative timing. Therefore, a careful analysis of the EMG patterns associated with certain types of movement should provide at least indirect information concerning the motor programs which generated the movements. The goal of this work was to study, in normal humans, how EMG patterns change when subjects are required to produce wrist flexion movements with specified velocity, amplitude, or force. The subjects were trained to alter one of these kinematic properties while the other two were kept constant. The results show that EMG patterns do change in a systematic and predictable manner to produce movements with different properties.

Methods

Subjects and recording techniques

The subjects were normal human volunteers of both sexes, ranging in age from 20–45. Twenty subjects participated in the studies on movement velocity, twelve in the amplitude studies and twelve in the tasks examining the effects of varying loads. Some subjects participated in more than one component of the experiment.

The apparatus was designed to study flexion movements at the wrist. Subjects sat in a chair with the forearm resting on a firm support with adjustable devices which restricted movement to the wrist joint. The hand was strapped to a moulded surface attached to a handle which was coupled to a vertical rod. The arm was positioned so that the axis of rotation of the wrist joint lay directly over the rod. The rod was coupled to a precision torque motor (Aeroflex TQ82W) which was used to generate steady loads against which the movements were carried out. A potentiometer attached to the rod provided a recording of handle position or wrist angle, and subsequent differentiation of this signal was performed to indicate the velocity profile of the movement.

With the wrist in the neural position, the handle of the manipulandum rested against a mechanical stop on the extensor side of the wrist. This stop included a micro switch which was released when the subject initiated a wrist flexion movement. Individual trials were aligned and averaged using the time at which the micro switch was released as a reference point.

EMG activity was recorded with metal disc surface electrodes applied over the flexor carpi radialis and extensor carpi radialis muscles. Interelectrode distance was 3 cm. Following amplification (bandpass 10 hz–10 khz) the EMG signals were full wave rectified and then smoothed with further low pass filtering. Recordings was carried out on line to a PDP11/40 computer which also controlled the target display, identified correctly performed trials and averaged the results.

Experimental tasks

The subjects sat in front of a video monitor which displayed an open square representing the target and a slightly smaller closed square which was a cursor controlled by having the subject move the handle. At random times the target suddenly moved away from the centre of the screen and the subject was required to produce a flexion movement of the wrist to align the cursor to the new target position. The subjects were told this was not a test of reaction time and that it was not necessary to move as quickly as possible when the target moved; rather they were encouraged to use this cue to prepare for and execute a precise planned movement.

The initial set of experiments was designed to study movements of different velocities. The amplitude of movement (40°) and the opposing load (0.36 NM) were kept constant for this part of the experiment. The computer program permitted the investigator to specify the minimum and maximum times for the handle to reach the target zone once onset of movement had been signalled by release of the microswitch. At first the subject was instructed to move the handle to the target as quickly as possible. The maximum time was set at 80 ms and trials which were not completed within this time limit were rejected. Therefore these trials all consisted of movements at velocities of 500 degrees/second or more. For slower movements, a range of movement times was specified. Following each movement the computer provided feedback on the screen consisting of a cartoon indicating to the subject whether the movement had been too fast, too slow, or within the correct range of velocities. By trial and error, the subject learned to produce movements within the required time limits. These limits were set so that an average subject could learn the task and produce consistently correct movements after 20 to 30 practice trials. For relatively fast movements the range of times could be made quite narrow (e.g. 80–110 ms); for slower movements, subjects required a much wider range before they could respond consistently (e.g. 250–350 ms). Using this method it was possible to collect data for movements occurring at 5 specified mean velocities ranging from 140 to 500 degrees per second.

In a second set of experiments, the target position was adjusted to correspond to movements with four specified amplitudes; 15°, 27°, 40°, and 53°. Subjects were instructed to move as rapidly as possible and maximum movement times were set to reject trials which did not meet these criteria (60 ms for the smallest movement and 90 ms for the largest movement). The opposing load was kept constant at 0.36 NM.

In a third set of experiments, designed to study the effect of increasing external loads, the output of the torque motor was set to provide five different loads ranging from 0 to 1.8 NM. For these experiments the subjects were also required to move as rapidly as possible over a standard distance of 40 degrees.

For each set of conditions the subject was allowed to practice until his performance was consistent from trial to trial. Then 2 sets of 10 trials were collected and averaged. Inspection of the single trial data revealed that even after considerable practice there was some variability in the EMG bursts. Averaging of a small number of trials was therefore carried out to provide a more representative "mean" EMG pattern than what might be indicated by any single trial.

Another set of experiments was carried out to investitage the origin and role of the antagonist EMG burst. The size of the antagonist burst was determined for 3 different types of movements: 1) Rapid 40° wrist flexion movements to a target, 2) Rapid movements with no intent to stop (in this case the movement was terminated when the handle reached a mechanical stop after about 60° of wrist flexion) and, 3) Passive wrist flexions produced by having the investigator move the subject's hand at velocities comparable to those attained under the first two conditions.

Data analysis

The averaged trials were plotted and displayed on a graphics terminal. By moving a set of cursors on the screen, the investigator could obtain measurements for amplitude, timing, and area of relevant portions of the signals. Onsets and ends of EMG bursts were identified by positioning the cursors at the points where the signal left or returned to a baseline determined by visual inspection of the initial 200 ms of EMG prior to movement. All of these measurements were made by the same individual using consistent criteria with the gain of the display increased to a standard level for all trials.

Two averages of 10 trials were recorded for each condition. If there was any doubt concerning the onset or end of an EMG burst, the point which was most consistent in the two separate averages was selected. The two averages were then combined to provide a single average of twenty trials. The analysis program also permitted measurements of the area of the EMG bursts between the cursors.

Results

1. EMG patterns and velocity of movement

Results from one subject performing wrist flexion movements at five different mean velocities are

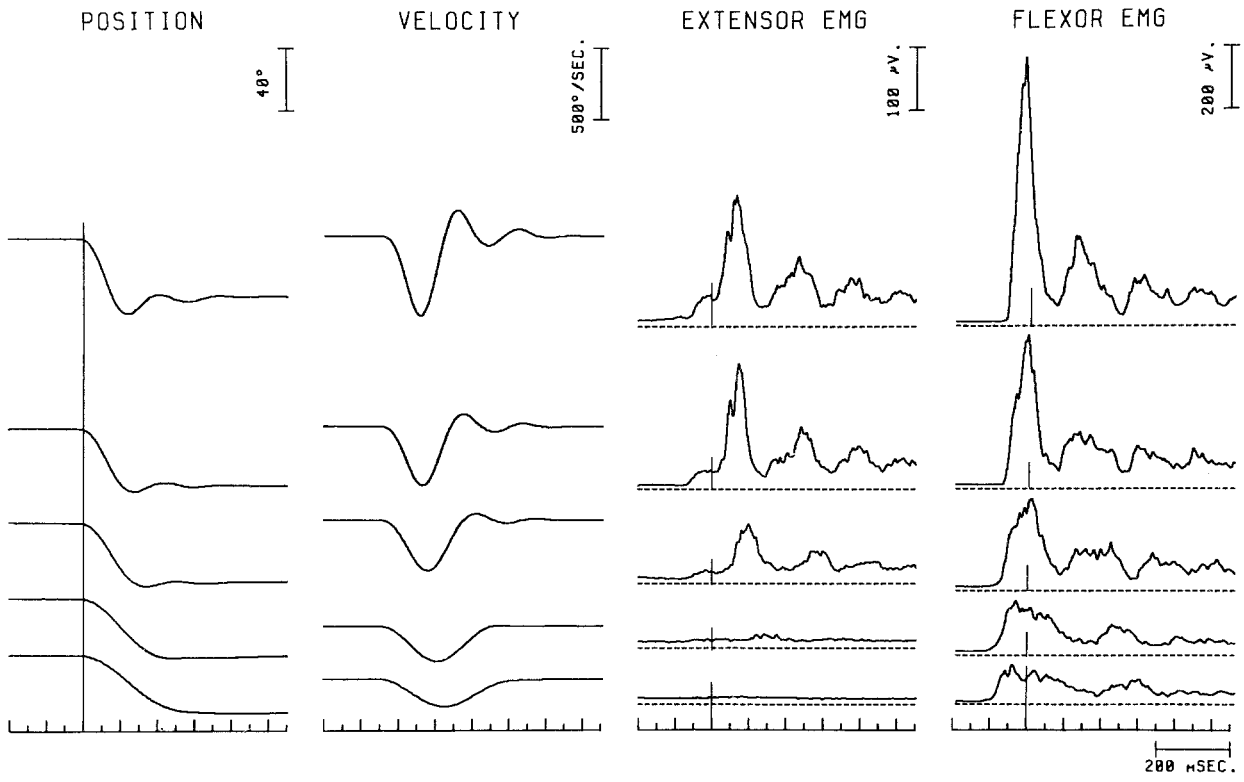

Fig. 1. Position and velocity changes and EMG bursts from wrist flexors and extensors for a normal subject performing wrist flexion movements at five specified mean velocities (150, 225, 300, 400 and 500 degrees/second). All movements were made over an angular distance of 40 degrees against a standard load of 0.36 NM. Each recording represents an average of 20 individual trials. Vertical lines on EMG traces correspond to vertical bar on position traces indicating onset of movement

shown in Fig. 1. The slowest movements were associated with a prolonged burst of EMG activity from the agonist muscle (wrist flexors). In this example, there was little or no acitivty from the antagonist muscle with the very slow movements. The most rapid movements shown at the top of Fig. 1 were associated with the characteristic triphasic pattern of EMG activity consisting of an initial burst from the agonist followed by an antagonist burst and then a second agonist burst. In this example there were also some subsequent EMG bursts following this initial pattern, but this later activity was not consistent from subject to subject and was therefore not considered in the analysis.

As the velocity of movement increased, the EMG acitivity showed a gradual evolution from the pattern associated with the slowest movements to the triphasic pattern. In the example shown in Fig. 1, a well defined antagonist burst did not appear until movement velocity exceeded 250 degrees/second.

In this subject, the main portion of the antagonist burst was preceded by a small hump which appeared to represent coactivation of the antagonist at the same time as the initial part of the agonist burst. A number of approaches including recording with intramuscular wire electrodes in some subjects were used to demonstrate that this did not merely represent "cross-talk" between the two groups of muscles. Other subjects showed some tonic activity in the antagonist muscle prior to the movement. When this was present, there was often a period of inhibition immediately preceding the onset of the antagonist burst. These variations created some minor problems in determining the exact time of onset of the antagonist burst. In all subjects, the onset of the major vertical deflection of the antagonist burst could be readily identified and it was decided to use this time as an indication of antagonist EMG onset.

With increasing movement velocity, the intervals between the three EMG bursts shortened. Interburst intervals were determined by measuring the time from onset of the first agonist burst to onsets of the antagonist burst and the second agonist burst. Mean values for these onset times for all 20 subjects are plotted as a function of movement velocity (Fig. 2 – left panel). Increasing velocity was also associated with changes in the size and configuration of individual EMG bursts. All three bursts increased in amplitude and decreased in duration with faster movements. This decrease in duration was most

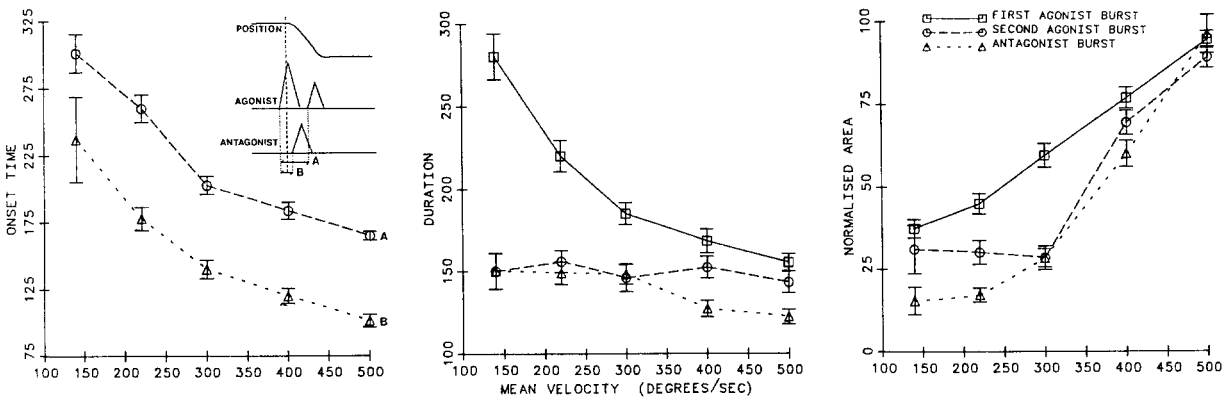

Fig. 2. Relationship between velocity of movement and properties of EMG burst. Left panel shows effects of increasing velocity of movement on timing of EMG bursts. As shown on inset interburst intervals were determined by measuring time from onset of first agonist burst to onsets of antagonist burst and second agonist burst. Middle panel shows duration of EMG bursts and right panel areas of bursts. For each of the 3 bursts area measurements were normalized with respect to the largest area for that burst. Points on the graphs represent mean values ± SE for 20 subjects

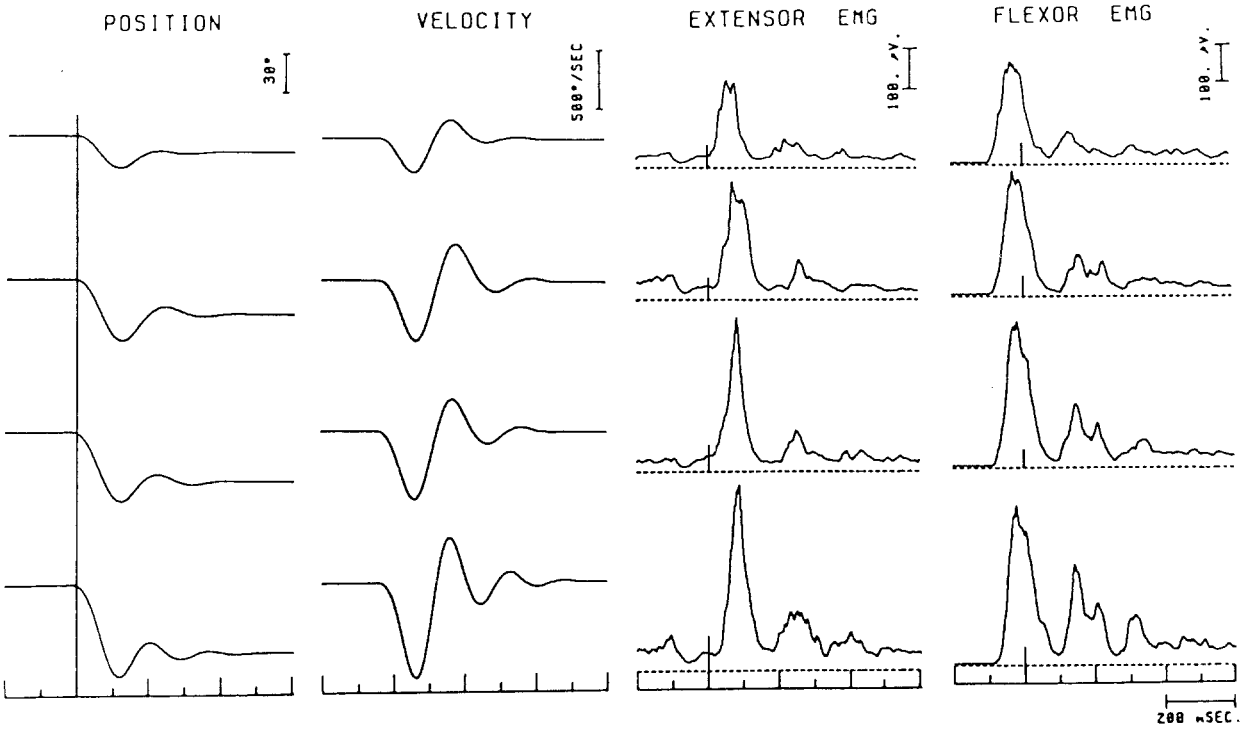

Fig. 3. Movement characteristics and averaged EMG bursts from a normal subject performing rapid wrist flexion movements over four different angular distances (15°, 27°, 40°, and 53°). Each recording represents an average of 20 individual trials. All movements were performed against a standard load of 0.36 NM

marked for the first agonist EMG burst (Fig. 2 – middle panel). The best indicator of amount of EMG activity was provided by measuring the area of each burst. As shown in the right panel of Fig. 2, the area of the first agonist burst increased in an almost linear fashion as movement velocity increased. The antagonist and second agonist bursts did not show consistent changes in area for the slower movements (in some subjects they were absent with the slowest movements). However, beyond movement velocities of 300 degrees/second, both of these bursts also showed a rapid increase in area.

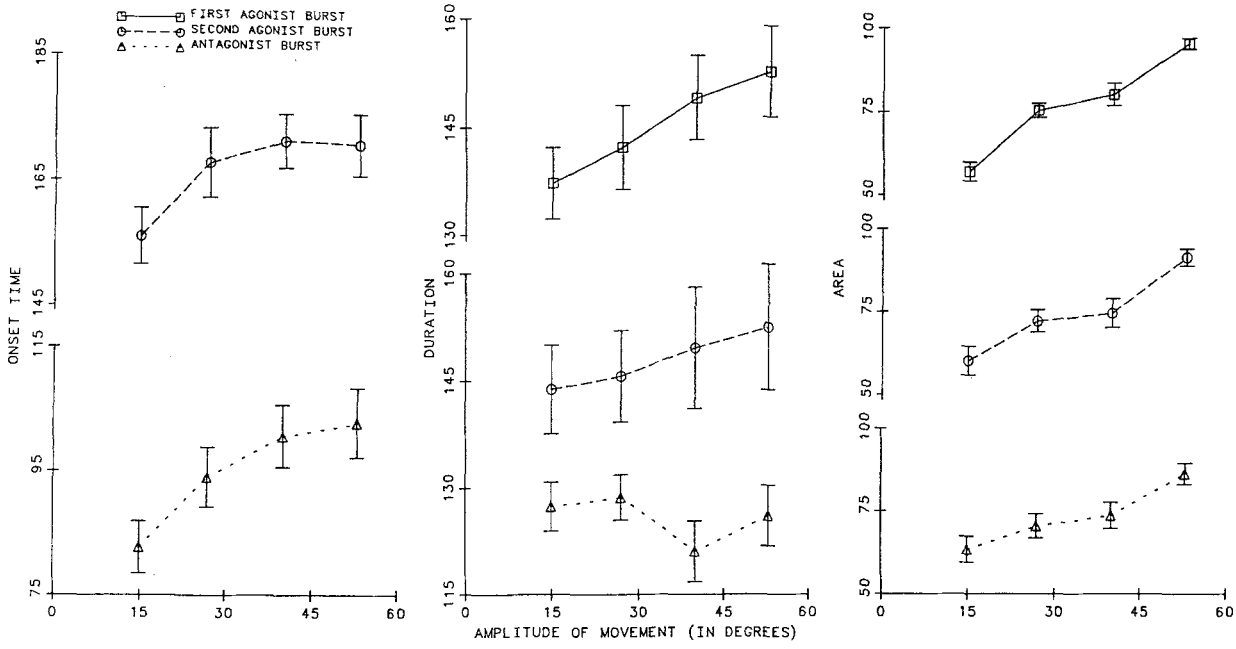

Fig. 4. Effect of increasing amplitude of movements on timing (left panel), duration (middle), and area (right) of EMG bursts from wrist flexors and extensors. Values shown represent mean ± SE for 12 subjects. Interburst intervals were determined by measuring onset time of bursts in the same manner as illustrated in Fig. 2. The interval between the first agonist burst and subsequent EMG bursts becomes progressively longer as movement amplitude increases. Durations of both agonist bursts increased slightly with increasing movement amplitude. Antagonist burst duration was not significantly changed

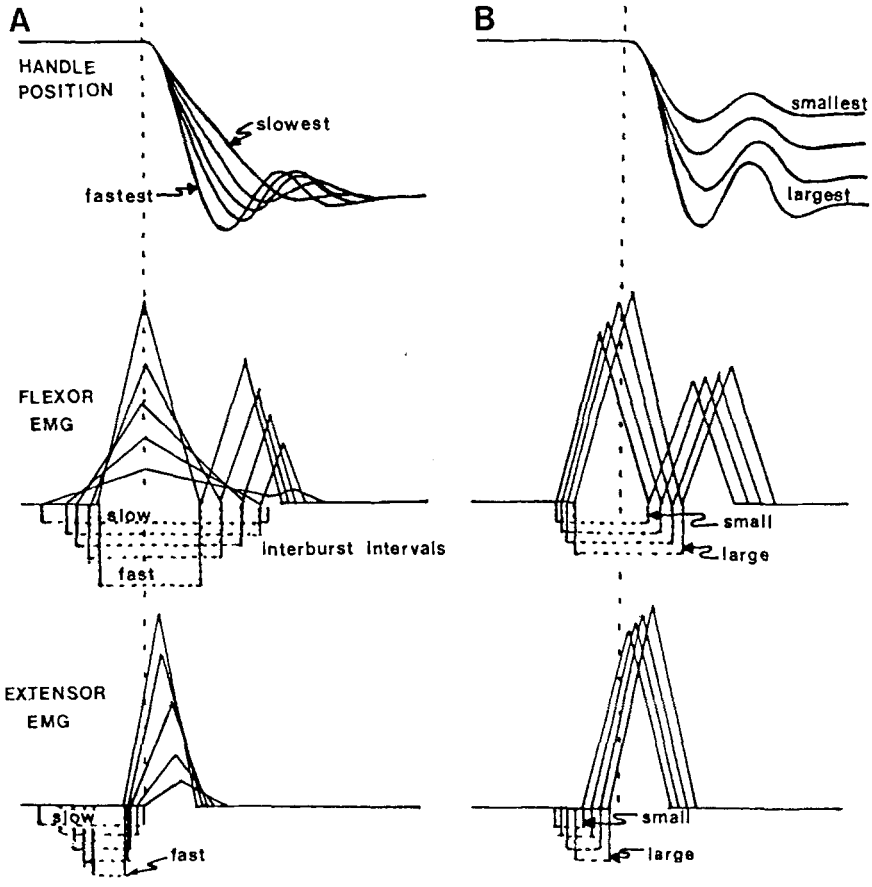

Fig. 5A, B. Schematic illustration showing changes in timing and duration of EMG bursts in relation to velocity (**A**) and amplitude (**B**) of flexion movements at wrist joint. These sketches were prepared using mean values obtained by measuring results from 20 subjects for the velocity experiments and 12 subjects for the amplitude experiments

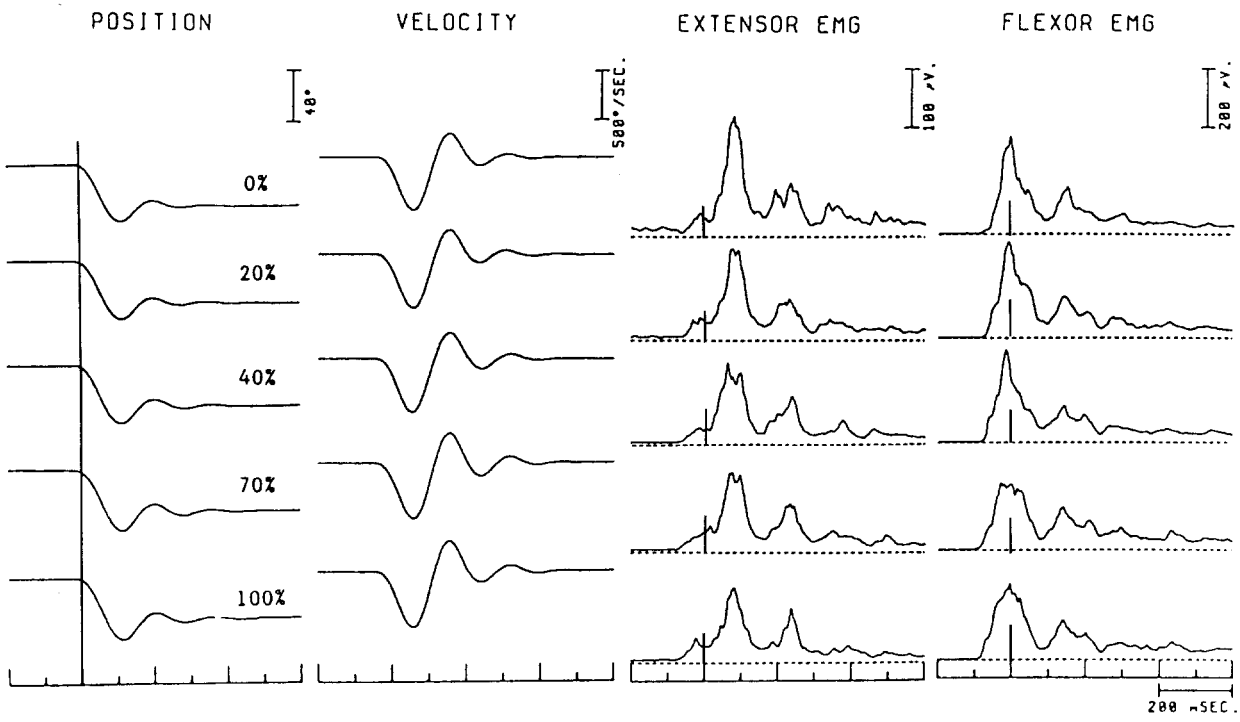

Fig. 6. Movement characteristics and associated EMG bursts from a subject performing rapid wrist flexion movements over an angular distance of 40 degrees against increasing external loads imposed by a torque motor. Load conditions are shown on the position trace and range from no load (0%) to 1.8 NM (100%). Note increase in duration of initial flexor EMG bursts and decrease in amplitude of extensor EMG bursts as the load becomes greater

2. EMG patterns and amplitude of movement

Figure 3 shows results from another subject performing movements of four different amplitudes, all against a constant load of 0.36 NM. In each case, the subject was instructed to move to the target as rapidly as possible. Therefore, the subject's intent was the same in each case, but with the larger amplitude movements, higher velocities were attained. As movement amplitude increased, the amplitude of both agonist bursts and also of the antagonist burst increased. This was accompanied by a slight but definite increase in the duration of the two agonist bursts, but no significant change was seen in the duration of the antagonist burst (Fig. 4 – middle panel). There was a linear increase in the area of all three EMG bursts with increasing movement amplitude (Fig. 4 – right).

The left panel of Fig. 4 shows mean values from twelve subjects for the timing of EMG bursts with movements of different amplitudes. The interval between the onset of the first agonist burst and the onset of the antagonist burst increased as the amplitude of the movement became greater. The onset of the second agonist burst also occurred progressively later with larger movements.

The changes in EMG bursts associated with different velocities and amplitudes of movement are summarized schematically in Fig. 5. For 40° movements, increases in velocity are accompanied by EMG bursts of increased amplitude and area, but decreased duration. Interburst intervals also decrease as velocity increases. Increases in movement amplitude are accomplished largely by changing the timing of the EMG bursts. Changes in the size and shape of individual bursts do occur but these are considerably less marked than those which are seen in relation to changes in velocity of movement.

3. EMG patterns with increasing external loads

Figure 6 shows results from one subject making movements against no load and also under four different external loading conditions. The amplitude of the movement was the same in each case (40 degrees) and the subject was instructed to move as quickly as possible. Even with the largest opposing load (1.8 NM) movement velocities were comparable to or greater than those associated with movements against no opposing load. How this was accomplished can be seen by examining the EMG bursts in Fig. 6

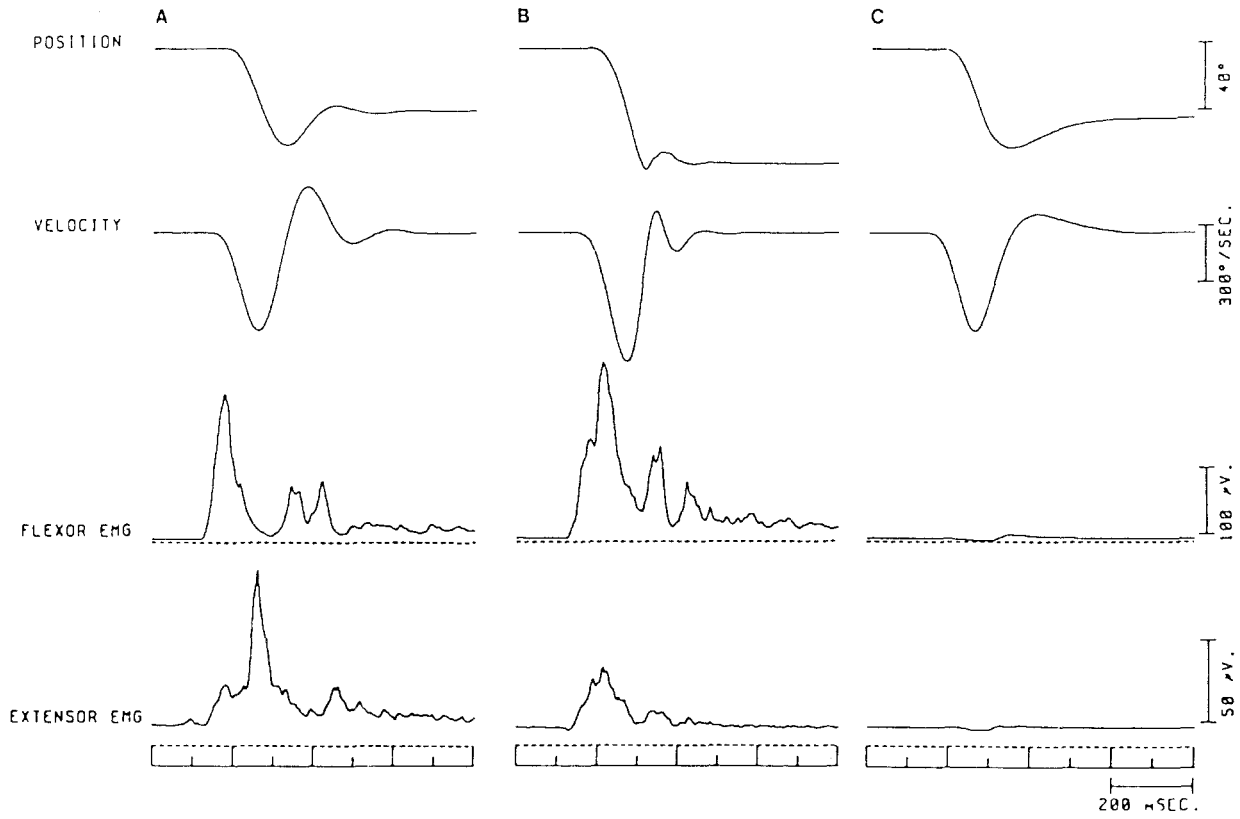

Fig. 7A–C. The behaviour of the antagonist (extensor) EMG burst with different types of movements. **A** Rapid 40° voluntary wrist flexion movements to a specified target. **B** Rapid voluntary movements with no intent to stop at the target. The movement is terminated when the handle hits a stop on the manipulandum. **C** Passive movements produced by investigator flexing subject's wrist at a velocity comparable to that in experiment **A**

which are representative of the changes seen in the twelve subjects who participated in this part of the experiment. With larger loads, the duration and area of the agonist bursts increased, although these changes were not very marked. The majority of trials showed a decrease in amplitude of the antagonist burst with larger loads but no consistent changes in duration were noted. Perhaps most interesting was the fact that there was no apparent change in the timing of the intervals between the EMG bursts with movements made against quite different loads.

4. The role of the antagonist EMG burst

Figure 7 shows results of an experiment carried out to determine the relative contributions of central programming and stretch reflexes to the generation of the antagonist burst.

When the subject performed rapid voluntary movements to a specified target (Fig. 7A) there was a well developed antagonist burst. As was seen in several of our subjects, the antagonist burst in this case had two components – a small initial "co-activation" component which occurred at the same time as the first agonist burst, and a larger "reciprocally timed" component occurring between the first and second agonist bursts. When the subject performed similar movements but with no intent to stop at the target (Fig. 7B), the main component of the antagonist burst was missing and only the co-activation component was seen. If the antagonist burst represented a stretch reflex it should have been even more prominent in this situation since the movement velocity and the rate of stretch applied to the extensor muscle was slightly greater than for the movements terminated at the target. In Fig. 7C, the subject's wrist was passively flexed by the experimenter. The velocity was comparable to that attained during voluntary movements to the target, but there was no antagonist EMG activity in this situation.

Discussion

The kinematic properties of simple movements can be adjusted by altering the amplitude or duration of individual EMG bursts, or by changing the relative

timing of the bursts from the participating muscles. The results of this study show that all of these mechanisms are used to varying degrees to specify the velocity, amplitude, and force of wrist flexion movements.

We must be careful not to attach too much significance to EMG bursts simply because they are available and convenient to measure. Obviously the central nervous system does not "think" in terms of amplitude and duration of EMG bursts. The central commands which generate movements activate pools of motoneurons, and the dimensions of the EMG bursts depend on the number of motor units activated, the frequency at which they fire, and the degree of synchronization (Desmedt and Godaux 1978).

Velocity experiments

When movement amplitude and the opposing load are constant, velocity of movement is increased by increasing the amplitude and decreasing the duration of the initial agonist EMG burst. With faster movements, the antagonist muscle is activated as well, and further graded changes in the amplitude and timing of the antagonist burst are observed as velocity increases to maximal levels.

Our results showed a gradual transition from the prolonged agonist EMG burst associated with slow movements to the triphasic pattern of activation seen with faster movements. There was no evidence for a sudden change in the pattern of EMG activation with increasing velocity such as might be expected if there were separate control systems for slow and fast movement as proposed by Kornhuber (1971).

Lestienne et al. (1981) have studied EMG patterns in opposing muscle groups during the transition from active movements to maintenance of a final position. During the dynamic phase, the EMG patterns of flexor and extensor muscles seemed to depend on the velocity of the movement and the initial position of the joint. The tonic EMG at the end of the movement correlated well with the final position but not with the velocity, duration or amplitude of the movement. Lestienne et al. (1981) concluded from their results that the processes controlling final position and trajectory are independent.

When a subject is required to terminate a fast movement at a specified target, there obviously must be some trade off between speed and accuracy. Other investigators (Hallett and Marsden 1979; Brown and Cooke 1981) have attempted to resolve this problem by giving the subjects specific instructions – e.g." move as rapidly and accurately as possible." However, we have found that different subjects adopt a variety of movement strategies even when the verbal instructions are quite specific.

For this reason we established time windows which required the subjects to learn to generate movements at several specified velocities. For faster movements, most subjects had no difficulty learning to produce movements within fairly narrow time windows (e.g. 100–110 ms); for slower movements the windows had to be considerably wider. Nevertheless, this approach helped overcome some of the problems with interpretation of EMG patterns where the subject's intent and movement strategy might be quite different.

Amplitude experiments

Several previous studies have shown that the duration of the EMG bursts, particularly the first agonist burst, remains constant for movements of different amplitudes (Freund and Budingen 1978; Hallett and Marsden 1979; Brown and Cooke 1981; Gordon and Ghez 1984). According to Hallett and Marsden (1979) the duration of the initial agonist and antagonist bursts remains constant when the amplitude of movement or the opposing load is changed. Different forces are produced by "varying the amount of EMG activity within a fixed time frame".

More recently Brown and Cooke (1984) have shown that the duration of the first agonist burst for movements at the elbow is increased for larger amplitude movements. However, they did not find evidence that the burst duration was continuously graded. For large amplitude movements (greater than 50 degrees) an additional burst was added so that the duration of the first agonist burst was essentially doubled.

Our results for flexion movements at the wrist joint were somewhat different. While the major changes associated with increasing amplitude of movement were increases in the amplitude and area of the first agonist burst, we also observed an increase in duration of the first agonist burst. Furthermore, the duration seemed to increase in a graded manner rather than in a stepwise fashion as reported by Brown and Cooke (1984). These apparent discrepancies may relate to the fact that different muscle groups were used and that the velocity and range of amplitudes were somewhat different in the two studies.

Marsden et al. (1983) studied movements of different amplitudes both at the thumb and at the elbow. When movement velocity was the same, the antagonist burst was smaller and occurred later for

larger amplitude movements. Similar findings were reported by Brown and Cooke (1984) for elbow flexion movement. Our results differed in that the size of the antagonist burst increased as the amplitude of movement increased. However, it should be noted that movement velocity was greater for the larger amplitude movements (see Fig. 3). Although the instructions to the subjects for this set of experiments were to move as rapidly as possible, velocities attained during the small amplitude movements were not as great as those during larger movements. The apparent discrepancies between our results and those of Marsden et al. (1983) and Brown and Cooke (1984) may also relate to the fact that a different set of agonist and antagonist muscles was used in our study. In addition to physiological differences between different muscle groups, the biomechanical characteristics of movements at distal and proximal joints are different and quite likely play a role in determining the characteristics of the associated EMG activity (Benecke et al. 1985).

Load experiments

To generate the force required to move quickly against an increased load, additional motor units are recruited and produce a larger initial agonist burst. In contrast to what was observed in the velocity and amplitude experiments, the relative timing of the EMG bursts did not change with movements performed against different loads. There was some variability in the size of the antagonist burst, but in most cases it was slightly smaller when the external load was increased, presumably because, with the additional load, there was less need for the antagonist to provide a braking mechanism to stop the movement at the target.

As will be discussed below, we believe that the major part of the antagonist burst is centrally programmed but the effects of increasing external loads indicated how programmed EMG bursts may be modified by peripheral inputs. The signals indicating that the load was greater and that there was less need for active braking must have originated from peripheral receptors in the extremity.

These results are similar in some respects to what was reported by Lestienne (1979) who studied the effects of increased inertial loads on EMG bursts associated with movements at the elbow. He observed increases in the level of activation of both the agonist and antagonist muscles when the inertial load was increased, but the relative timing of the bursts was not altered. Viscoelastic properties of the muscles and tendons obviously assist in deceleration of the movement, but when the driving force exceeds this passive resistance, an additional contribution from the antagonist muscle is required to prevent the movement from overshooting the target.

The role of the antagonist burst

The momentum associated with rapid limb movements is such that there is a need for some type of braking mechanism to ensure that the movement stops at the specified target. There is considerable evidence that this is provided by activation of the antagonist muscle, and most investigators consider that the antagonist EMG burst is generated as part of the centrally preprogrammed set of instructions for this type of movement (Hallett et al. 1975; Lestienne 1979; Waters and Strick 1981; Ghez and Martin 1982). However, the antagonist is undergoing rapid stretch during these movements and it is conceivable that at least some of the EMG burst could represent a stretch reflex, unless stretch reflexes are modified or gated out during certain types of movement (Terzuolo et al. 1981; Akazawa et al. 1985). Our observations that the antagonist burst is markedly reduced during fast movements with no intent to stop, and absent during rapid passive stretches, indicate that the stretch reflexes make only a small contribution to the antagonist burst. Similar conclusions concerning the antagonist burst have been reached by others (Waters and Strick 1981; Marsden et al. 1983).

Observations on deafferented monkeys (Polit and Bizzi 1979) and on patients with functional deafferentation due to sensory neuropathies (Hallett et al. 1975; Rothwell et al. 1982; Cooke et al. 1985) have shown that the triphasic EMG pattern including the antagonist burst can be generated independently of peripheral feedback. Despite these observations and the common belief that "ballistic" movements occur too rapidly to allow modification by spinal reflexes (Kornhuber 1971; Desmedt and Godaux 1979), there is evidence that the associated EMG patterns can be modified by peripheral inputs. In another study from our laboratory it was shown that, when a subject encounters unanticipated changes in load during rapid learned movements, the initial agonist and antagonist EMG bursts show compensatory changes occuring at times which would be compatible with spinal reflex activity (Lee and Lucier 1982; Lee et al. 1986). Thus, while the triphasic EMG pattern does represent the output of a learned motor program, it is important to realize that the pattern may undergo modification at the spinal level, and the final output which is recorded is determined by complex interac-

tions between central commands and afferent feedback from the extremity.

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