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BALLISTIC FLEXION MOVEMENTS OF THE HUMAN THUMB

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SUMMARY

1. In response to an auditory stimulus normal subjects made ballistic flexion movements of the top joint of the thumb against a lever attached to the spindle of a low-inertia electric motor.

2. Electromyographic (e.m.g.) activity was recorded from pairs of fine wire electrodes inserted into flexor pollicis longus and extensor pollicis longus, respectively the sole flexor and extensor of the joint.

3. Movements of 5°, 10° and 20° were made from initial angles of 10°, 20° and 30° flexion against torques of 0.04, 0.08 and 0.16 Nm.

4. The e.m.g. activity initiating such movements was characterized by a 'triphasic' pattern of sequential bursts of activity in the agonist (flexor pollicis longus), then in the antagonist (extensor pollicis longus), and then in the agonist again.

5. The duration of the first agonist and first antagonist bursts ranged from about 50 to 90 ms and there was no significant change of burst length in the different mechanical conditions.

6. In movements of differing angular distance, the rectified and integrated e.m.g. activity of the first agonist burst could be correlated with the distance moved. The rectified and integrated e.m.g. activity of the first antagonist burst could not be correlated with the distance moved.

7. Responses of the muscles to perturbations either before or during the ballistic movements were studied. Current in the motor could be altered so to extend the thumb ('stretch'), to allow it to accelerate ('release'), or to prevent further movement ('halt').

8. Suitably timed stretch increased the e.m.g. activity of the first agonist burst while release decreased it.

9. There was a small response of the agonist to stretch or halt timed to act during the interval between the first two agonist bursts; the major response was an augmentation of the second agonist burst.

10. Stretch, timed to act between the first two agonist bursts which released the antagonist, diminished the activity of the first antagonist burst while halt virtually eradicated it in all but one subject. Release, at this time, which stretched the antagonist, increased the activity of the first antagonist burst.

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11. It is concluded that the individual components of a ballistic movement are relatively fixed in duration and the amount of e.m.g. activity is altered within this time interval to produce the different forces required for fast movements of different amplitude.

12. Both agonist and antagonist muscles remain under some feed-back control during the entire course of a ballistic movement, but the amount of influence of feed-back depends on the supraspinal command signal and the changes in the spindle during the course of the movement.

INTRODUCTION

The electromyographic e.m.g. pattern of a rapid or ballistic movement is distinctive. In all muscle pairs that have been studied a burst of activity in the agonist is followed by a burst of activity in the antagonist which is followed in turn by a second burst in the agonist (Wachholder & Altenburger, 1926; Angel, 1974; Hallett, Shahani & Young, 1975*a*). Slower, smooth ramp movements are characterized by continuous agonist activity without significant participation of the antagonist. Differences of the e.m.g. pattern of ballistic and ramp movements, and differences in responses to perturbations, have suggested that the central command signals for the two types of movement may differ (Gottlieb, Agarwal & Stark, 1970; Hallett *et al.* 1975*a*). Motor unit behaviour is different in the two circumstances (Desmedt & Godaux, 1977). It has even been suggested that the two types of movement are controlled from two different parts of the brain (Kornhuber, 1971).

Previous studies utilizing a standard rapid 10° flexion movement of the elbow showed that the burst durations of the first agonist and antagonist bursts were relatively fixed in normal subjects (Hallett *et al.* 1975*a*). In contrast, patients with lesions of the cerebellum or cerebellar pathways, who often overshoot in such a task, produced longer bursts (Hallett, Shahani & Young, 1975*b*). Although the duration of the first agonist burst in such patients did not seem to correlate with the amount of overshoot, the question arose as to how burst duration normally varied with amount of movement. The initial parts of the present study were designed to investigate the precise mechanical consequences of each part of the characteristic e.m.g. pattern in ballistic movements and changes of the pattern with different movements. The major issue is whether changes in force needed to produce different movements are produced by changes in duration of the component bursts. Rapid flexion movements of the top joint of the thumb were studied. This movement is ideal since there is only one muscle which flexes the joint and one which extends it. Additionally, the movement can be considered to be an important example of a distal limb movement, which is presumably under the powerful influence of a large area of motor cortex and large input via the corticomotorneuronal pathway.

The precise nature and origin of the command signal responsible for the generation of a ballistic movement are not known, but it has been suggested that the signal is pre-programmed (Hallett *et al.* 1975*a*), by which it is inferred that the distinctive pattern can be produced by the central signal itself and that feed-back is unnecessary. The question arises as to what extent such a 'pre-programmed signal' may be influenced by peripheral information. Previous studies have suggested that feed-

back has little effect on the initial part of the pattern (Garland & Angel, 1971; Angel, 1975; Hallett *et al.* 1975*a*). The second part of this study investigates the extent to which peripheral information can modify the characteristic e.m.g. pattern responsible for ballistic movement. The major issue is whether perturbations of the joint before or during the movement will alter the e.m.g. pattern.

Preliminary accounts of some of the findings were presented at several meetings (Hallett & Marsden, 1977*a*, *b*).

METHODS

In most of the experiments the e.m.g. was recorded from flexor pollicis longus and extensor pollicis longus, the only flexor and extensor of the interphalangeal joint of the thumb, with pairs of wire electrodes. Fine platinum wires, insulated except for a few mm at the tip, were inserted into the muscle with a hypodermic needle. In some experiments pairs of surface electrodes were utilized, but such techniques proved useful only for flexor pollicis longus. Extensor pollicis longus is too deep and too near other muscles to record the activity from the surface. E.m.g. activity was amplified (Devices 3120) and continuously integrated (Devices 4010) using analogue devices.

Subjects were normal volunteers from the laboratory workers who gave informed consent to the experimental procedures. They sat in a chair with their forearms resting on a table; the proximal phalanx of the thumb was firmly clamped with the pad of the distal phalanx resting on a lever attached to the spindle of a low-inertia electric motor so that movement was possible only at the interphalangeal joint. By varying the current to the motor the torque pressing against the thumb could be altered. A strain gauge was incorporated into the lever so that the force exerted by the thumb could be measured. A potentiometer was incorporated into the motor's spindle so that the angular position of the thumb could be measured. This positional information was converted to the horizontal position of a vertical line displayed on an oscilloscope screen easily observed by the subject. Details of these experimental arrangements have been described previously (Marsden, Merton & Morton, 1976).

In one set of experiments subjects were asked to flex the top joint of the thumb 'as rapidly as possible' following a click stimulus, so as to move the line on the oscilloscope screen from one designated position to another. The angular distance to be moved, the initial angle of the joint and background torque of the motor were varied in different trials. Only well practised movements were analysed. E.m.g. and mechanical data were collected and stored with a PDP-12 computer using programmes written by H. B. Morton.

In another set of experiments, subjects were asked again to make rapid movements so as to move a line on the oscilloscope screen from one designated position to another. At a pre-set time, either with respect to the click stimulus or with respect to the onset of e.m.g. activity in the agonist, perturbations could be introduced. At random either (1) the torque of the motor would increase, *stretching* the agonist, or (2) the torque would decrease, *releasing* the agonist, or (3) the torque would be altered via a feed-back circuit, *halting* the thumb in its current position or (4) the torque would remain unchanged, a *control*. E.m.g. and mechanical data were averaged separately for the different conditions. Usually the average of twenty-four trials for each condition was recorded. Subjects practised the movements before collecting data. Perturbations were introduced in fewer than half of the trials and subjects were told to try to perform the task the best they could despite the perturbation.

RESULTS

Time of the bursts

The e.m.g. pattern underlying rapid movements of the thumb is usually 'triphasic', identical to the pattern underlying rapid movements of proximal muscles (Fig. 1). The basic pattern is characterized by a burst of activity in the agonist, followed by

a pause during which there is a burst in the antagonist, followed by a second burst in the agonist.

Identification of the beginning and ending of the first agonist burst was usually unambiguous. Occasionally there was a distinct pause in the low level base-line firing of the muscle prior to the onset of the first agonist burst, but even if not, the beginning of this burst was distinctly characterized by the rapid firing of action potentials and quick rise in the amplitude of the rectified e.m.g. signal. The first

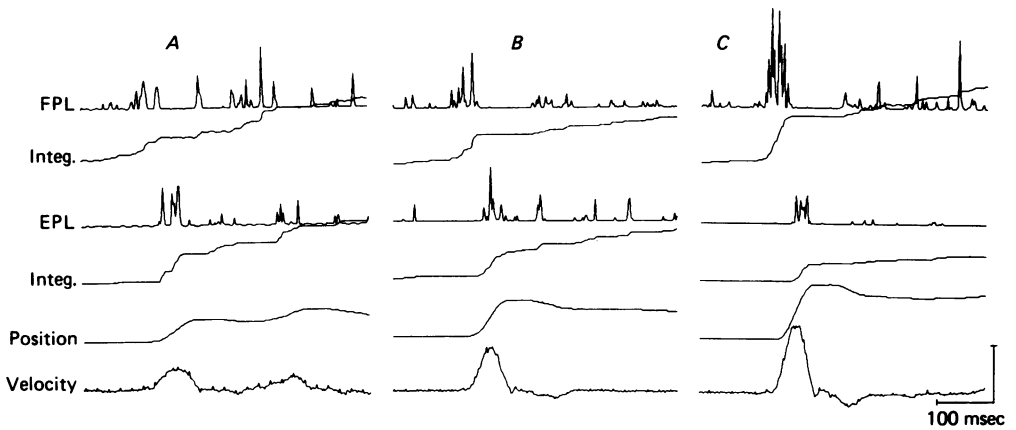


Fig. 1. Ballistic flexion movements of the top joint of the thumb. The records are individual trials from the same subject attempting movements 5° (*A*), 10° (*B*) and 20° (*C*) from an initial angle of 20° against a constant torque of 0·08 Nm. The e.m.g. activity of each muscle (FPL = flexor pollicis longus; EPL = extensor pollicis longus) is shown first rectified and then rectified and continuously integrated. The vertical calibration line corresponds to 1 mV for the rectified e.m.g. records and 25° for the position record.

agonist burst usually terminated at about the time of onset of the first antagonist burst and the second agonist burst usually began at about the time of termination of the antagonist burst. The period between the agonist bursts was usually quiet. Occasionally there was a distinct 'co-contraction' burst of the agonist during the first antagonist burst, in the middle of the pause between the two major agonist bursts; at times there would even be a pause during the first antagonist burst corresponding to the co-contraction. More will be said about this behaviour below. Although the beginning of the second agonist burst was usually clear, the time of termination of this burst was less certain. Indeed, the second agonist burst is extremely variable in appearance and no attempt was made to analyse it.

Identification of the limits of the first antagonist burst was usually clear, but occasionally difficult. Sometimes there would be antagonist activity during the first agonist burst. Usually such activity would be clearly separate in time from activity considered to be the first antagonist burst. This co-contraction activity was irregular in its appearance and was disregarded in the analysis. At other times co-contraction activity of the antagonist was not clearly separated from antagonist activity occurring after the first agonist burst ended; this behaviour could represent early initiation of the first antagonist burst or merging of co-contraction and the first antagonist burst. Often in this latter circumstance the limits of the first antagonist

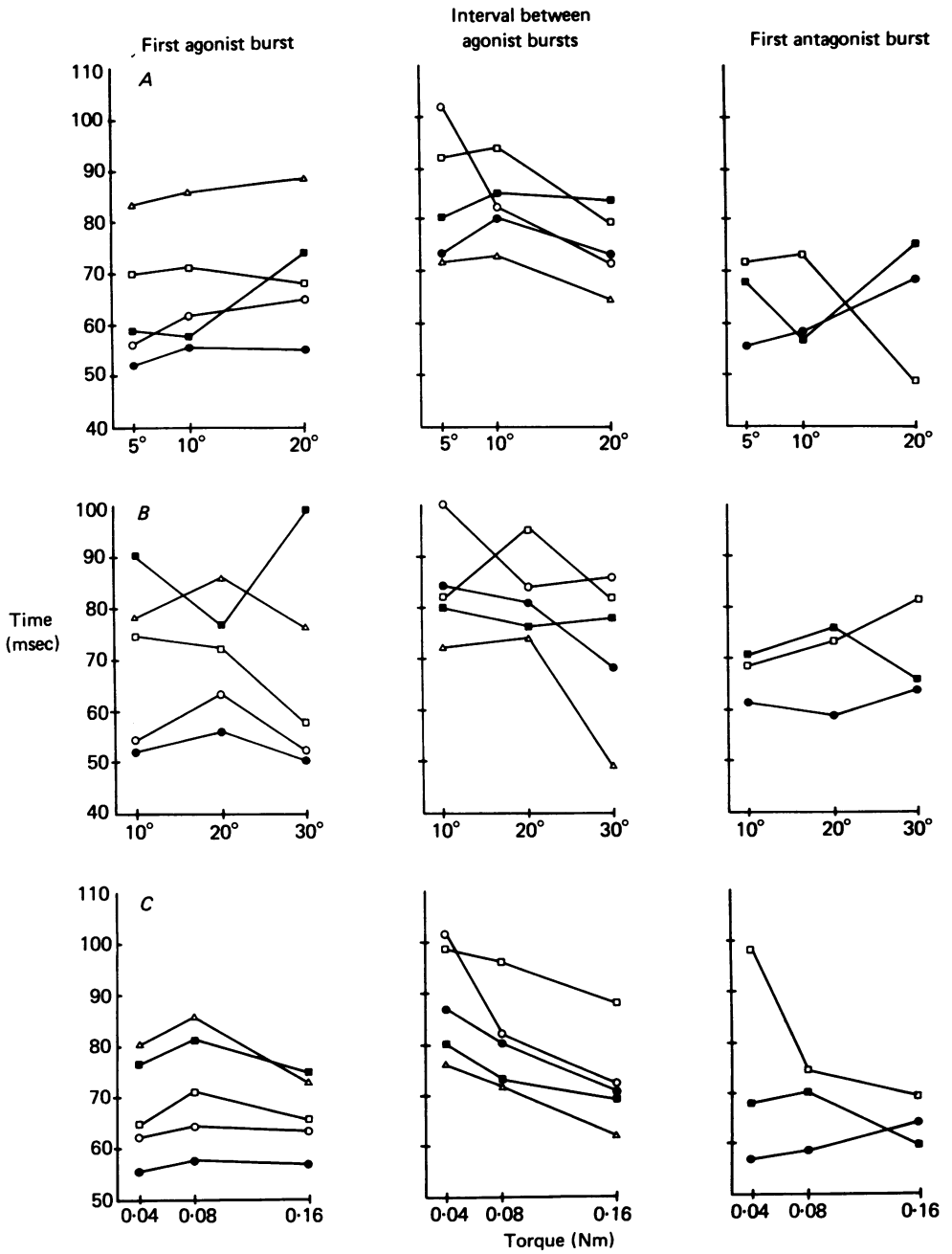


Fig. 2. Average durations of the components of the ballistic movement pattern in different mechanical circumstances. *A*, movements of 5°, 10°, 20°. *B*, movements of 10° from starting angles of 10°, 20° or 30° of flexion. *C*, movements against initial torques of 0.04, 0.08 and 0.16 Nm. Each point is the average value for the component in ten movements. The values for each of the five subjects are identified in the different parts of the Figure by the same symbol.

burst could not be identified with certainty. In two of five subjects the first antagonist burst was only present occasionally and thus could not be analysed.

With preceding considerations in mind, the durations of the first agonist burst, the first antagonist burst and the pause between the agonist bursts were measured for five subjects during rapid thumb flexions with different mechanical parameters. In the first set of experiments each subject made movements through different

TABLE 1. Ranges of duration (ms) of e.m.g. components of ballistic movements

	First agonist burst	Interval between agonist bursts	First antagonist burst
Thumb flexion*	52–88	64–104	48–76
Elbow flexion†	60–105	40–150	30–85

*Data from Fig. 2*A*.
†Data from Hallett, Shahani & Young (1975*a*).

distances from the same initial position, and against the same constant resisting torque (Fig. 1). The mean duration of each component for each subject is shown in Fig. 2*A*. Standard deviations of the means varied from about 10 to 30%. There was no significant variation in the durations of the components for the different movements. The range of durations of these components was remarkably similar to the values previously determined in biceps and triceps for a 10° rapid elbow flexion (Table 1).

In a second set of experiments, the subjects made the same movement, but from a number of different starting points (Fig. 2*B*). In the third set of experiments of this type, the subjects made the same movements from the same starting point but against different resisting torques (Fig. 2*C*). The durations of the first agonist and antagonist bursts showed no systematic changes in these two sets of experiments, though in each subject the interval between the agonist bursts decreased as the torque of the motor increased.

Relation of the bursts to force

In order to make movements of different distances (against constant torque and from the same initial angle), it is necessary to generate different impulses (impulse is the product of force and time). The results of the previous section show that the e.m.g. burst durations are constant, so that changes in impulse are produced by alterations of force and not of time. These changes of force should be reflected in the amplitude of the e.m.g. within the fixed time periods.

The examples in Fig. 1 qualitatively demonstrate that the 'amplitude' or the 'amount of e.m.g. activity' in the first agonist burst relates to the distance moved and hence to the initial impulse producing that distance. The amount of e.m.g. can be measured as the height of the step in the rectified and continuously integrated record produced by the burst. Likewise, the amount of e.m.g. activity in the first antagonist or second agonist could be measured. The relation between the amount of e.m.g. activity in the first agonist burst was compared to the actual distance moved using the data from the experiment where subjects moved 5°, 10° and 20°

from an initial angle of 20° against a background force of 0.08 Nm. The correlation was good for all five subjects (Fig. 3*A*). A good correlation was also demonstrated between the amount of e.m.g. in the first agonist burst and maximum velocity of the movement.

Attempts to relate the first antagonist burst with distance did not meet with similar success (Fig. 3*B*). There was not a good correlation of these two parameters except in occasional experiments, and then only with just the short distances.

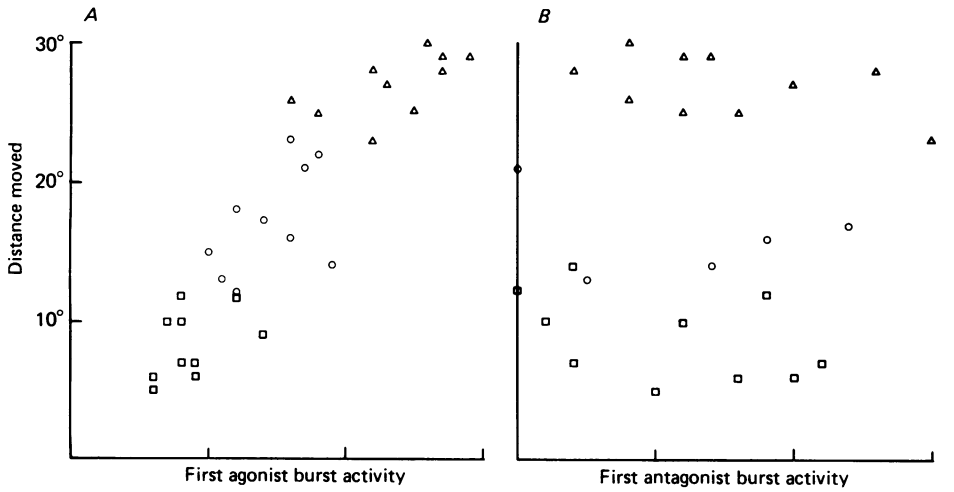


Fig. 3. The relations between integrated activity of the first agonist (*A*) and first antagonist (*B*) bursts to the angular distance moved. The values are from one subject attempting ten moves each of 5° (□), 10° (○) and 20° (△). (Five values of the first antagonist burst for the 10° move were lost due to a technical problem.)

Several facts made it clear that a close correlation between distance and antagonist activity should not have been expected. In two subjects the first antagonist burst occurred only occasionally. The amount of e.m.g. activity in the antagonist, even when present, seemed quantitatively insufficient to provide the total decelerative impulse. Before beginning the ballistic movement experiments, the quality of the e.m.g. recording from each muscle was tested by determining the relation between integrated electrical activity and isometric force. Using the amount of electrical activity for each muscle to generate the same force as a crude measure of force in the ballistic situation, it would be seen that the antagonist was not generating forces equal to the agonist. Additionally, while the first agonist burst was always completed prior to termination of the accelerative phase of the movement, the first antagonist burst often outlasted the period of deceleration.

Careful observations of the first antagonist burst revealed that it occasionally appeared to be composed of two parts (Fig. 4). The two parts were rarely further divided by brief firing in the agonist (arrow in Fig. 4*B*). The first part completely preceded the period of deceleration and qualitatively seemed related to the amplitude of deceleration. The second part preceded a secondary corrective movement. This secondary corrective movement was usually an extension movement since the primary movement had usually slightly overshot. It appears that each part of the first antagonist burst can be varied independently, even to the extent that either part might be absent. With subtle changes in strategy and with variable accuracy requiring different corrective movements, the amount of electrical activity in the antagonist becomes highly variable.

Perturbations

In these experiments six subjects were asked to make stereotyped rapid flexion movements and, at a pre-selected time, by altering the torque of the motor one of four events would occur randomly: (1) nothing, a *control*; (2) an extension of the joint, a *stretch*; (3) a flexion of the joint more rapid or further (or both) than would

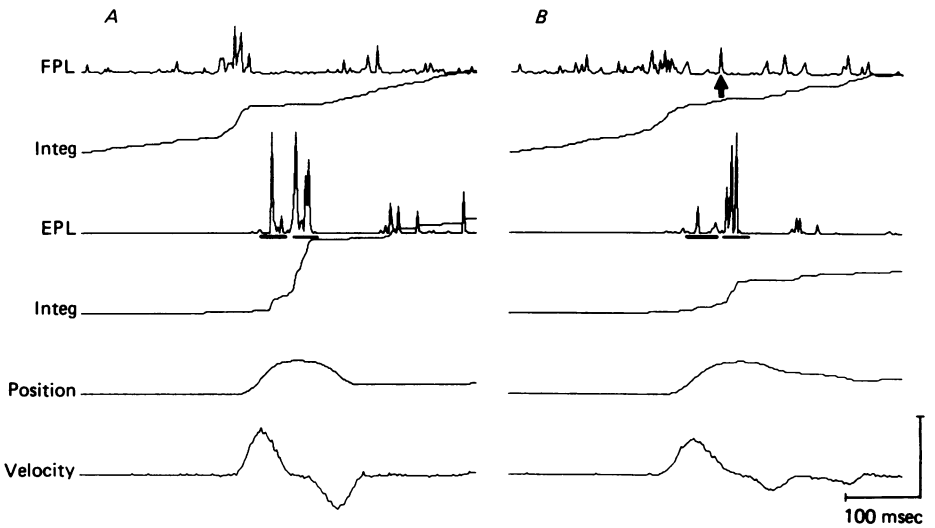


Fig. 4. Ballistic flexion movements showing two parts of the first antagonist burst. *A* and *B* are two individual trials from the same subject. The first part of the antagonist burst is indicated by a thick line and the second part by a thin line. In *B* the arrow points to activity in the agonist occurring between the two parts of the first antagonist. The vertical calibration line corresponds to 2 mV for the rectified activity of flexor pollicis longus (FPL), 0·2 mV for the rectified activity of extensor pollicis longus (EPL) and 20° for the position record. Background torque was 0·12 Nm.

be produced by the movement itself, a *release*; or (4) a maintenance of the joint in its current position, a *halt*. Usually eight trials of each event were averaged in one set, and three sets were obtained and averaged together.

In the first experiment of this type, designed to assess the effect of perturbations on the first agonist burst, the perturbation was set to occur at a fixed time after the auditory click which signalled the subject to move. This time was altered for each subject depending on his average reaction time so that the expected reflex response, if it would occur, would be in the middle of the first agonist burst. In practice this was difficult to do because the variability of the reaction time of most subjects. In most experiments, however, the same results could be demonstrated (Fig. 5). The stretch produced a stretch reflex superimposed upon the agonist burst. As is the case with isometric movement or ramp movements, this stretch reflex usually was composed of two phases beginning at about 40 and 55 ms with an occasional additional small earlier phase beginning at 25 ms. The release decreased the activity of the agonist burst at a latency of 40 ms or shortly thereafter. The halt had no effect at these early latencies, and would not be expected to have effect, since the

effect of halting is not apparent until after the thumb would ordinarily begin to move, about 25 ms after the onset of e.m.g. activity. There is a late rise in the e.m.g. activity following the halt and this will be analysed further below.

To assess the effect of perturbation on the end of the first agonist burst and other parts of the ballistic movement pattern, the timing of the perturbations was set with respect to the initial action potentials of the first agonist burst. This method

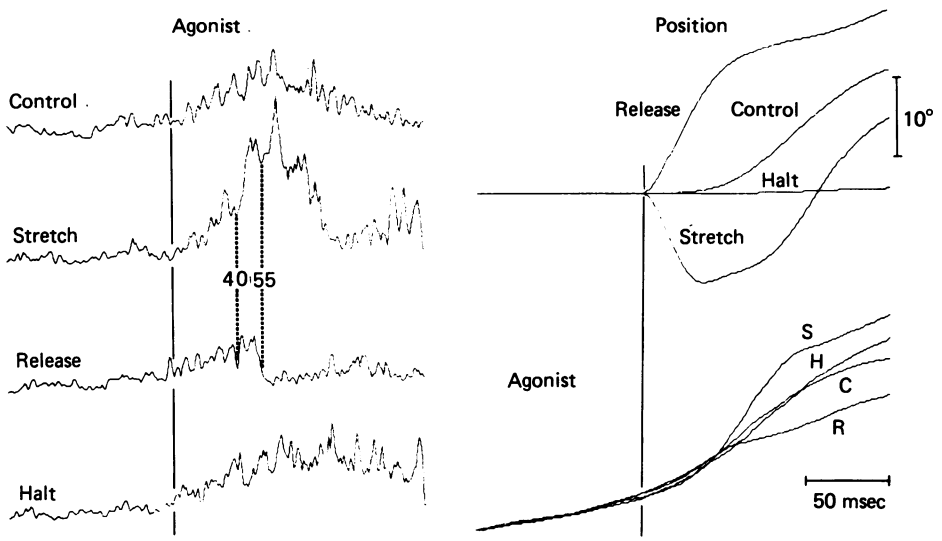


Fig. 5. The effect of perturbations on the first agonist burst. The subject attempted to make repeated ballistic movements of 15° following an auditory click, which occurred at the beginning of the traces. The control, stretch, release and halt conditions occurred randomly and each record is an average of twenty-four individual trials. The time of the perturbation is indicated by the thin line. The records on the left are rectified activity from flexor pollicis longus. The records at the top right are the superimposed position records for the four different conditions. The records at the bottom right are the superimposed rectified and integrated activity from flexor pollicis longus (a 'tulip'). The reflex responses are indicated by their latencies, 40 and 55 ms. The background torque was 0.10 Nm; stretch was produced by a step increase of torque to 0.20 Nm; release was produced by a step decrease to 0.02 Nm.

circumvents the variability of reaction time and aligns the e.m.g. bursts. The perturbations were triggered at various times after the first agonist burst began. Fig. 6 shows the response of agonist and antagonist muscles of one subject to perturbations at 10, 30 and 50 ms. The major responses occurred on average at about 25, 40, and 55 ms; for the subject illustrated in Fig. 6 they occurred at 25, 45 and 65 ms.

At 10 ms (Fig. 6*A*) the agonist responses to stretch occurred at the end of the first agonist burst. Stretch produced a stretch reflex with the largest components beginning at 45 ms, and the second agonist burst became much larger than in the control. Release produced a lessening of activity at the end of the first agonist burst and this silencing of activity merged with the expected silence of the interval between the agonist bursts. The halt did not produce a significant rise in e.m.g. activity until the time of the second agonist burst, even though the thumb position

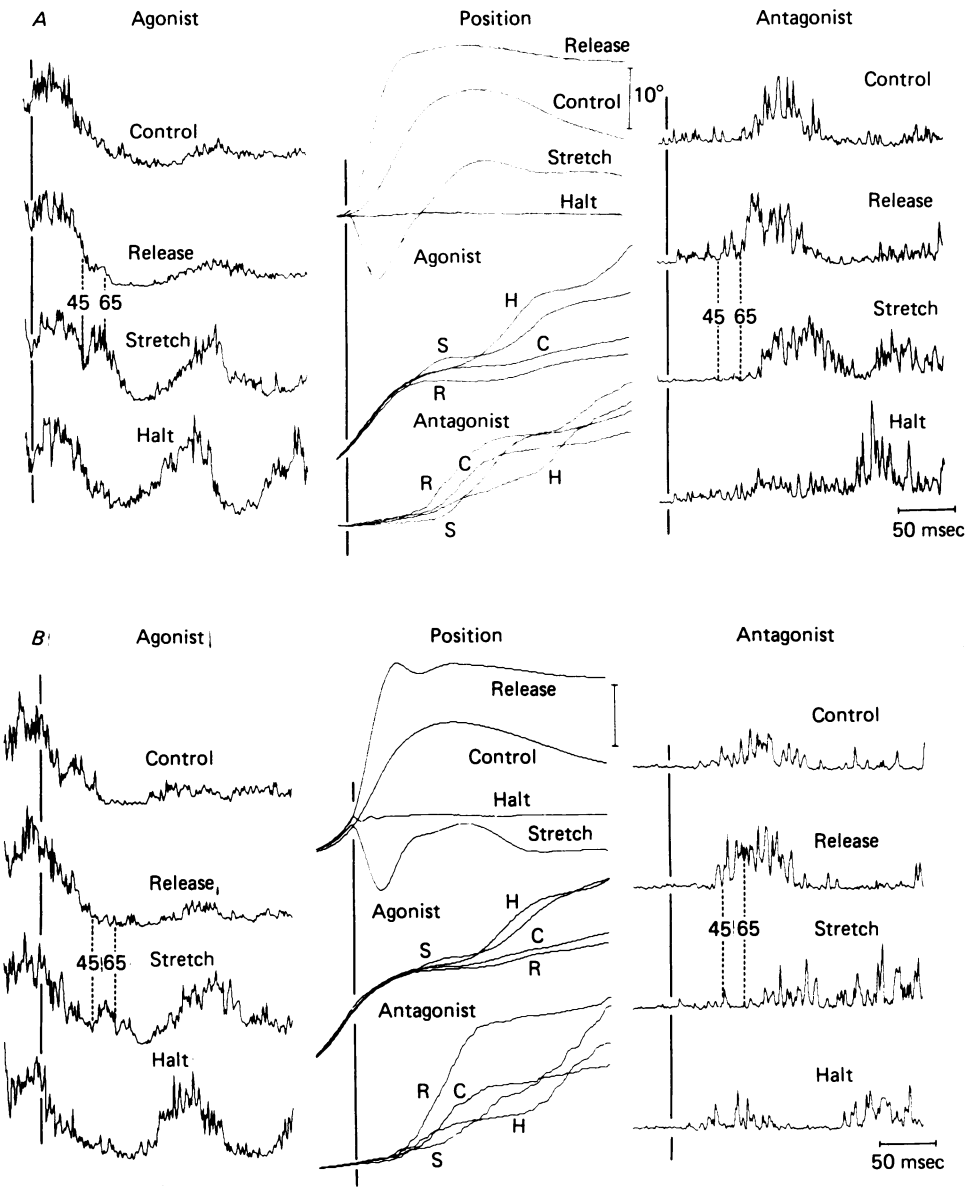
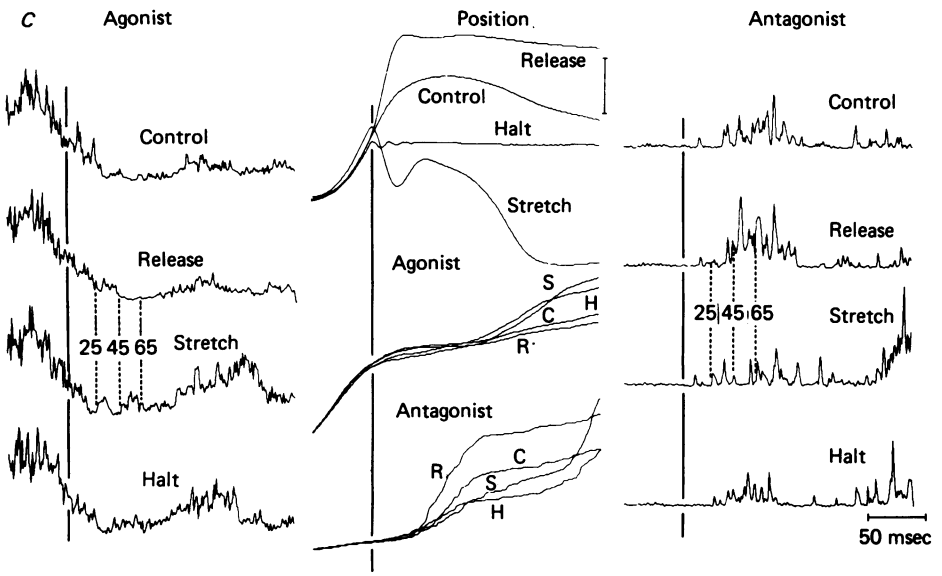


Fig. 6. The effect of perturbations on the components of the ballistic movement pattern. The subject made repeated rapid movements of 10° following an auditory click, which occurred from about 100–150 ms before the start of the traces. The traces begin with the first action potentials of the first agonist burst as detected by a Schmidt trigger. The control, stretch, release and halt conditions occurred randomly and each record is an average of twenty-four individual trials. The perturbations occurred 10 ms (*A*), 30 ms (*B*) and 50 ms (*C*) after the start of the first agonist burst, and the time of the perturbations is indicated by a thin line. The records on the left are rectified activity from flexor pollicis longus and those on the right are rectified activity from extensor pollicis longus. In the middle from the top downwards are the superimposed position records and the superimposed rectified and integrated activity ('tulips') from the two muscles. The reflex responses are indicated by their latencies, 25, 45 or 65 ms. The background torque was 0.12 Nm; stretches were produced by step increases of torque to 0.26 Nm and releases were produced by dropping the torque to zero.

began to differ from the expected position at the same time as during the stretch or release.

In considering the antagonist responses to such perturbations it is important to keep in mind that the 'stretch' will release the antagonist muscle and the 'release' will stretch it. The stretch shown in Fig. 6*A* produced a subtle silence prior to the onset of the first antagonist burst and delayed its onset slightly. For this subject and this early perturbation, most of the first antagonist burst occurred after the



stretch reflex in the agonist was completed. In other subjects, with an earlier first antagonist burst, the stretch perturbation reduced the expected activity of the initial part of the first antagonist burst, but it was never eliminated. The release caused a definite stretch reflex, but it is of note that there is a larger response at 65 ms than there is at 45 ms. The halt virtually eliminated the entire first antagonist burst. The halt had this dramatic effect on all of the subjects except for one for whom the amplitude of the first antagonist burst was only slightly decreased.

At 30 ms (Fig. 6*B*) the agonist's response to the stretch occurred early in the interval between the agonist bursts with a latency of about 45 ms; again the second agonist burst was enhanced. The silence with the release occurred synchronously with the expected silent period. The response to halting again seemed to be an enhancement of the second agonist burst. The antagonist's response to the release was a stretch reflex that now occurred with a relatively larger component at 45 ms and coincided with the first antagonist burst, increasing its magnitude. Stretch, which released the antagonist muscle, reduced the expected activity of the antagonist burst. Halting again considerably reduced the first antagonist burst.

At 50 ms (Fig. 6*C*) the agonist's response to stretch occurred in the middle of the interval between the agonist bursts. The stretch reflex is small with a component at about 25 ms and another component beginning after 45 ms. The major response to the stretch, as with the halt, is at the time of the second agonist burst. Again the

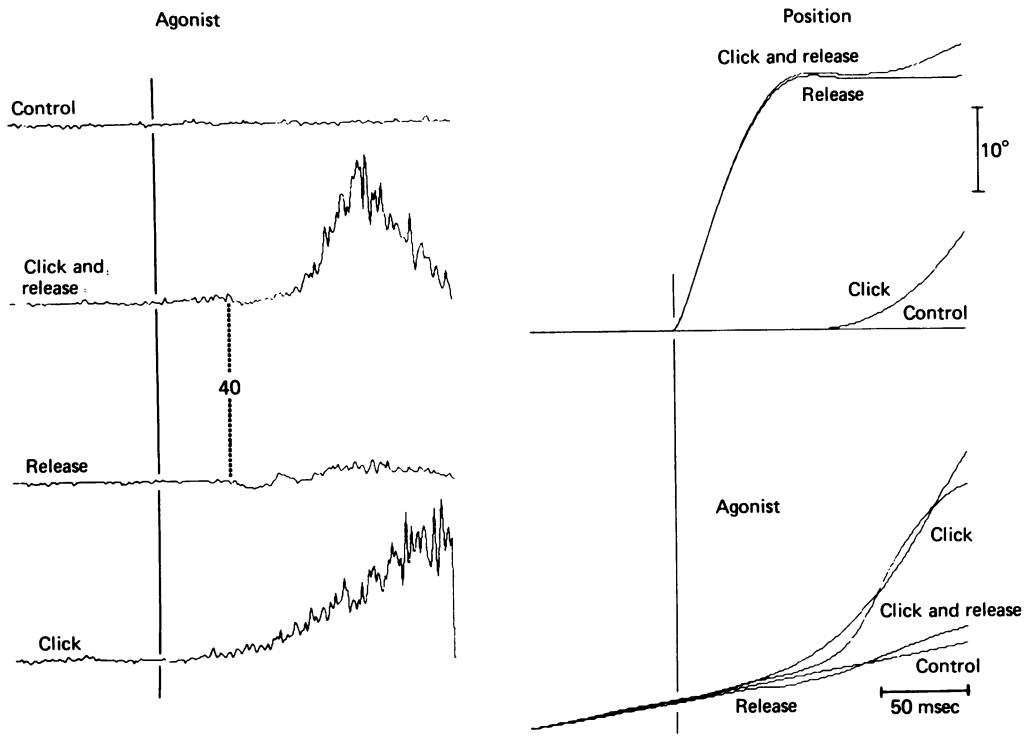


Fig. 7. The effect of release on the initial part of the first agonist burst. The subject held his thumb flexed at 20° against a torque of 0.12 Nm and attempted to make 10° ballistic movements whenever he heard an auditory click. In the 'click' condition(4) the auditory stimulus occurred at the beginning of the trace. In the 'click and release' condition (2) the auditory stimulus was similarly timed and the perturbation adjusted in time so that the reflex release response occurred at the time of initiation of agonist firing. In the 'release' condition (3) there was no click, the trace was begun at a random time after the subject began the tonic holding (waiting for a click to occur), and the perturbation was introduced at a similar time interval after the trace began as in the 'click and release' condition. In the 'control' condition (1) the subject was carrying out the tonic holding task and neither click nor perturbation occurred. The conditions occurred randomly and each record is an average of sixty-four individual trials. The time of perturbation is indicated by the thin line and the reflex response is indicated by its latency, 40ms. The rectified e.m.g. activity from flexor pollicis longus is on the left and the superimposed position records and superimposed rectified and integrated e.m.g. activity ('tulip') from the muscle are on the right. The background torque was 0.12 Nm and release was achieved by reducing the torque to zero.

response to release merges with the pause between the agonist bursts. The antagonist's response to release was an increase in activity beginning at 45 ms. The stretch and the halt both diminished the size of the first antagonist burst.

Another experiment was designed to study the influence of a release on the initial part of the first agonist burst. A release was timed with respect to the click stimulus so that the onset of reflex silencing of e.m.g. activity approximated the time of onset of the first agonist burst. The subject pressed his thumb against the lever and

made a rapid flexion movement when he heard the click. Four conditions were randomized: (1) no click, no release, (2) click, release, (3) no click, release, and (4) click alone. Similar results were obtained in eight subjects (Fig. 7). Comparing the release (3) and no release (1) trials without the click, a silent period to the release was apparent at about a latency of 40 ms (3). Comparing the two trials with the click (2) and (4), a silent period to release was again apparent, now superimposed upon the onset of the first agonist burst (2). (This result is similar to the results previously described when the release had its effect in the middle or end of the first agonist burst.) Comparing the two release trials (2) and (3), the release while the subject was just holding the position (3) was more effective in silencing e.m.g. activity than was the release prior to the ballistic movement (2). At the very beginning of the e.m.g. silencing, however, there were few milliseconds where the two releases produced a similar degree of silencing.

DISCUSSION

Invariance of time intervals

The triphasic e.m.g. pattern of the initial phase of ballistic movements (agonist, antagonist, agonist bursts) exists in distal muscles of the arm, as has been shown previously for proximal muscles (Wachholder & Altenburger, 1926; Basmajian, 1967; Dijkstra & Denier van der Gon, 1973; Hopf, Lowitzsch & Schlegel, 1973; Angel, 1974; Hallett *et al.* 1975*a*), and the timing of the bursts also is similar (Table 1). The results demonstrate that for movements of various distances and in different mechanical circumstances the durations of the first agonist and antagonist bursts are relatively constant. Freund & Büdingen (1978) have come to a similar conclusion in a study of ballistic movements of several hand and forearm muscles; the duration of the movement and initial e.m.g. burst were constant (approximately 100 ms) with movements of different amplitude. F. Lestienne (in preparation) has shown that the first biceps burst in fast elbow flexion has constant duration despite changes in inertia or velocity. Rapid isometric movements of different magnitude in the cat have a constant time to peak force (Ghez & Vicario, 1978).

Mechanical consequences of the bursts

It is clear from these results that ballistic movements requiring different forces are generated by varying the amount of e.m.g. activity within a fixed time frame. Desmedt & Godaux (1977) have demonstrated elegantly that this is possible by altering the number of active motor units and their frequency of firing.

A relation between the first antagonist and the distance moved could not be demonstrated. A correlation should not be expected because the first antagonist burst is not present in some subjects; even when present it appears to be small and often it outlasts the period of decelerative impulse. Understanding of the role of the first antagonist burst begins with the observation that occasionally it is divided into two parts. The first part totally precedes deceleration and may contribute to it. Our qualitative impression is that when the first part of the antagonist burst is larger the decelerative phase is more rapid. Major contributions to deceleration also

must come from passive mechanical features, such as 'springs' acting about the joint. F. Lestienne (in preparation) has shown that antagonist (triceps) activity appears in elbow flexion movements only when the agonist force exceeds a certain threshold.

The second part of the first antagonist burst precedes a secondary corrective movement. In trying to make a 'fast, accurate' thumb flexion movement most subjects overshoot slightly and make a small corrective extension movement. Our qualitative impression is that the magnitude of the second part of the first antagonist burst relates to the extent of the corrective movement. The second part of the first antagonist burst can be varied independently of the first part.

Nature of controlling signals

For some years many physiologists have viewed ballistic movements as being so rapid that there is 'no time for feed-back control' (Stetson & McGill, 1923). Certainly the triphasic e.m.g. pattern of a ballistic movement is pre-programmed in the central nervous system and not the consequence of activity in peripheral loops, for such a phasic e.m.g. pattern was present in a patient with a severe pan-sensory neuropathy which essentially caused peripheral differentiation (Hallett *et al.* 1975*a*).

An issue, independent of whether the movement is originally pre-programmed or not, is whether the muscles remain under feed-back control during a ballistic movement. Marsden *et al.* (1976) have shown that isometric or smooth ramp movements are under continuous feed-back control of several reflex loops. The precise role of these reflexes in the control of movement has not been clarified, but one function is presumably to help to correct for peripheral perturbation of a limb while it is carrying out a willed task. The monosynaptic reflex (with onset at 25 ms) in the thumb is small, and a quantitatively more important reflex is the 'long-latency stretch reflex' (with onset at 40 ms) (Marsden *et al.* 1976). The long-latency stretch reflex of a muscle working as an agonist seems divisable into two parts with latencies in the thumb of about 40 ms and 55 ms (Marsden, Merton, Morton, Adam & Hallett, 1978). When a muscle is serving as an antagonist, the response to stretch is smaller and often is delayed (Adam, Hallett, Marsden, Merton & Morton, 1976). The response at 40 ms may be absent and there may even be a brief period of inhibition at this latency.

The extent to which the characteristic triphasic e.m.g. pattern of ballistic movement is susceptible to peripheral influence has been debated. Angel and colleagues suggested that the first agonist burst was not affected by peripheral perturbations, while the second agonist burst was (Garland & Angel, 1971; Angel, 1975). Hallett *et al.* (1975*a*) suggested that the first antagonist burst was not very susceptible to perturbation. These studies appeared to support the notion that the initial part of the triphasic e.m.g. pattern was not only pre-programmed, but also relatively immutable.

The present studies were designed to measure carefully the sensitivity of the thumb muscles to peripheral influence throughout the initial phases of ballistic movement. Perturbations timed between the signal to move and the beginning of the first agonist burst have clear influence on the burst. Stretch superimposes a reflex increase in e.m.g. activity at about 25, 40 and 55 ms just as in slower ramp movements. Release causes a decrease in e.m.g. activity at 40 ms or shortly after.

Thus the agonist remains under some feed-back control even in this first phase of a ballistic movement. This result is contrary to most previous published experiments. Garland & Angel (1971) studied 'passive shortening' (similar to a release) of the pectoralis major prior to rapid adduction of the shoulder and found no alteration of the peak amplitude of the rectified and filtered first agonist e.m.g. burst. Hopf *et al.* (1973) studied weight-loading, muscle vibration and direct electrical stimulation of the biceps prior to brisk elbow flexion and found no effect on the first agonist e.m.g. burst. Angel (1975) studied passive shortening of the posterior deltoid prior to abduction and external rotation of the shoulder and found no change of the amplitude or duration of the first agonist e.m.g. burst. Hallett *et al.* (1975*a*) studied stretch of the biceps prior to elbow flexion; there was no change in the duration of the first agonist burst but its amplitude was increased.

There are a number of possible explanations for the discrepancy between these previous studies and the results of the present investigation. All the previous studies were of proximal muscles whereas the present study was of a distal muscle-pair. In the previous studies there was no background torque exerted by the agonist prior to the movement, while in this study there was such a torque; reflex responses are proportional to background force exerted by the muscle (Marsden *et al.* 1977). The present method also is much more sensitive for detecting changes than those previously employed. Which of these explanations is correct will be the subject of further studies.

The experiment in which a release was timed to act just at the onset of the first agonist burst (Fig. 7) was designed to obtain some quantitative information about the level of feed-back control. The degree of silencing of the e.m.g. activity produced by such a release was compared in a steady isometric contraction and in a ballistic movement on top of that isometric contraction. If the ballistic movement is initiated solely by increasing the activity in the gamma loop, then the silencing of the ballistic movement should exceed (or at least equal) the silencing of the isometric contraction. In fact, the silencing of the ballistic movement was less than the silencing of the background contraction, indicating the existance of a significant alpha component to the initial agonist burst. This result is compatible with previous studies of Vallbo (1971) and Hagbarth, Wallin & Löfstedt (1975) showing activity in an agonist muscle prior to increased firing in Ia afferents from that muscle. However, the fact that some silencing occurred indicates a degree of peripheral support of the initial phase of the ballistic movement.

Further evidence comes from the effect of halting the movement shortly after the onset of the first agonist burst so as to block the movement from its onset. The deviation from expected position occurs almost at the same time as the deviation caused by the stretch, yet there is no significant increase in e.m.g. activity of the agonist to overcome this increased torque until the time of the second agonist burst. Thus mismatch alone, without stretch, is insufficient to increase the size of the initial agonist burst or to overcome the silence between the agonist bursts. Halt does not fail to influence the agonist at early times because it is an impotent stimulus; halt does have an effect on the antagonist muscle quite early, virtually eradicating the first antagonist burst in all but one of the subjects. The antagonist burst is not useful to the movement in this circumstance since it is already halted.

This significant effect on the antagonist muscle produced by mismatch indicates the importance of feed-back loops in supporting the e.m.g. activity of the first antagonist burst. Similar results on antagonist activity in this circumstance have recently been obtained also by Angel (1977) and Denier van der Gon & Wadman (1977) in studies of proximal muscles.

The small agonist response to stretch timed to act in the pause between the two agonist bursts is a result similar to one obtained by Gottlieb & Agarwal (1975) who pointed out the discrepancy between the small or absent response to stretch and the enhanced H-reflex during this period. Evarts & Fromm (1977) have shown that neurones in area 4 which fire in association with ballistic movements, and which ordinarily would be influenced by limb perturbation, do not show response to stretch shortly after movement onset. Explanation of these results must come in part from observations of Hagbarth and colleagues who have studied Ia spindle afferent activity during rapid movements (Hagbarth *et al.* 1975). Ia afferents are active in a brief burst at the onset of the movement, but then fall silent (at about the time of the silent period of the agonist). This silence is due either to pre-programming of the gamma motoneurone signals or unloading of the spindle by the movement of the joint. If the Ia response to the perturbation also is small, significant responses in cortical neurones or alpha motoneurones cannot be expected. There may be also a change in the sensitivity of these cortical command neurones at this time.

The small agonist responses to stretch in this time period shortly after movement onset can be contrasted to the significant antagonist responses: release (which stretches the antagonist) increases the first antagonist burst, while halt and stretch (which releases the antagonist) diminish it. Ia afferents show activity during the antagonist phase of rapid alternating movements (Hagbarth *et al.* 1975); this can reflect pre-programming of gamma motoneurone activity and/or the response of the spindle to the stretch produced by the agonist muscle. This raises the question as to whether the first antagonist burst is merely a 'stretch reflex' induced by the movement generated by the first agonist burst. However, the first antagonist burst was present in the patient with the pan-sensory neuropathy who presumably had no peripheral input (Hallett *et al.* 1975*a*). Furthermore, the size of the antagonist burst was not related to the amplitude of the ballistic movement (i.e. to the size of stretch of the antagonist). Thus it appears that the first antagonist burst is a product of both pre-programming and feed-back, with the latter being quite important in most normal subjects.

In conclusion, the central command signal of a ballistic movement begins with a precise signal to the agonist for a pulse of accelerative force with the goal of moving a limb to the desired place. The next phase is a pre-planned decelerative force with the goal of stopping the limb appropriately. This decelerative force is often aided by activity of the antagonist muscle functioning as an agonist and is accompanied by reciprocal inhibition of the agonist itself. Subsequent activities are chiefly corrective in nature, reflecting the match between the desired and achieved results. Peripheral feed-back influences rapid ballistic movement, but the agonist spindles are subject to unloading which lessens the effect of perturbations on the agonist, while the antagonist spindles are subject to stretch which augments the effect of perturbations.

As a result of these various mechanisms, stretches and releases can have effects on

both agonist and antagonist muscles at all times during a ballistic movement. Their influence will be less than maximal to the extent that they compete with a supraspinal command signal and to the extent that the spindle is 'inactivated' by unloading. Thus even if the triphasic e.m.g. pattern that generates fast ballistic movements is pre-programmed it remains modifiable by peripheral input.

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