*J. Physiol.* (1983), **335**, *pp.* 1–13
*With 9 text-figures*
*Printed in Great Britain*



# THE FUNCTION OF THE ANTAGONIST MUSCLE DURING FAST LIMB MOVEMENTS IN MAN

BY C. D. MARSDEN, J. A. OBESO AND J. C. ROTHWELL
*From the University Department of Neurology, Institute of Psychiatry and King's College Hospital, de Crespigny Park, London SE5 8AF*

(*Received 28 October 1981*)

## SUMMARY

1. We have examined fast flexion movements of the human thumb and fast extension movements of the elbow over three different distances at a variety of speeds in order to elucidate the function of the antagonist muscle in these circumstances.

2. All movements were of such a velocity that they showed the typical bi- or triphasic pattern of muscle activation in agonist and antagonist. Slower movements, with continuous agonist activity, were not analysed.

3. For movements made through the same angle at different velocities, there was a linear relationship between the amount of antagonist activity needed to halt the movement and the peak velocity. However, the slope of this relationship was a function of the distance moved. Movements made through large angles showed less antagonist activity than those made through small angles at the same speed.

4. The timing of the antagonist activity also changed as a function of both distance and speed. Fast, small movements showed earlier onset of antagonist activity than slow, large ones.

5. Movements which were halted mechanically with the subject's prior knowledge had little or no antagonist activity, since it was no longer necessary in these conditions.

6. The complexity of these relations indicates that the triphasic pattern of muscle activity underlying these movements can no longer be regarded as a simple immutable 'programme'. The size and timing of the bursts of muscle activity are subtly adjusted to the precise nature of the task.

## INTRODUCTION

A triphasic pattern of activation is seen in the electromyogram (e.m.g.) of the agonist and antagonist muscles during fast limb movements in man (Wachholder & Altenburger, 1928). The first agonist e.m.g. burst produces the impulsive force for the movement; it ceases before the limb has reached the final end-position. The function of the antagonist muscle burst is not clear; it is generally presumed to provide a counter-acting braking force. Indeed, Lestienne (1979) has shown that the amount of triceps activity during fast elbow flexion is proportional to the velocity of movements above a certain speed. Below this threshold, the visco-elastic properties of the joint alone are sufficient to halt the movement. However, both Hallett &

Marsden (1979) and Brown & Cooke (1981), analysing thumb and elbow movements respectively, have shown that the size of the antagonist burst is independent of movement amplitude. If the function of the antagonist activity is indeed to halt the movement, then it seems strange that its size should be independent of movement amplitude. We now show that the size and timing of the antagonist muscle e.m.g. burst are a function of both the amplitude and velocity of movement. The visco-elastic properties of a joint vary with its angle, rising very rapidly at the extremes of joint rotation so that, for any constant velocity, larger movements require smaller antagonist activity. Somehow the nervous system must be capable of computing this relation of distance to be moved and velocity of movement, so as to determine the size of the antagonist burst required to halt the movement.

## METHODS

As before (Hallett & Marsden, 1979), we have studied fast flexion movements of the top joint of the thumb produced by flexor pollicis longus (f.p.l.), whose e.m.g. activity was recorded by silver–silver chloride surface electrodes secured over the belly of f.p.l. The activity of the main antagonist extensor pollicis longus (e.p.l.) was recorded with platinum–iridium wire electrodes (3/1000 inch diameter) inserted via a needle into the muscle. The pad of the thumb rested on a lever attached to the spindle of a printed motor (G9M4H, Printed Motors Ltd), which exerted a fixed torque of 0·06 N m against the thumb. A sensitive potentiometer (Bourns servopotentiometer) was attached to the other end of the spindle to record thumb position, and its output was displayed on one channel of a cathode ray oscilloscope placed 18 cm in front of the subject.

In addition, in these experiments we also studied extension movements of the elbow. The shoulder was abducted to 90°, and the point of the elbow and the forearm rested on a soft padded platform whose axis of rotation was aligned with that of the elbow joint. The forearm was semi-pronated so that the subject could grasp a handle mounted vertically from the end of the platform. The platform was connected via a frictionless bearing to a potentiometer signalling elbow angle, whose output was displayed on a cathode ray oscilloscope as described above. The e.m.g. activity of the agonist triceps and antagonist biceps was recorded with surface electrodes.

In each session the subject first practised movement of the thumb from full extension to 14° of flexion (for the elbow these figures were from 80° to 95° of extension). Then he was asked to execute a series of such movements in his own time at a range of velocities, all of which were sufficiently fast to require triphasic activation of agonist and antagonist (for f.p.l. these were from about 200 to 800 deg/s; for triceps these were 125 to 275 deg/s). Each individual run was recorded for joint position, velocity (analogue differential obtained from the position signal), and the raw, rectified and integrated e.m.g. signals (Devices 3160 pre-amplifier with a time constant of 20 ms, low-pass filtered to attenuate the signal by 3 dB at 2·5 kHz, amplified by a Devices 3120 amplifier and processed by a Devices Signal Processor, type 4010). Thirty to forty trials were recorded. The subject then rested for 10 min or so before being asked to learn to move to a new position at different velocities. This sequence was repeated for three different positions (for f.p.l. to 14°, 30° and 7° of thumb flexion; for triceps to 15°, 30° and 4° of elbow extension).

The following variables were measured from each record: the amplitude and peak velocity of movement; the time of onset and cessation of the agonist and antagonist e.m.g. bursts; the integrated e.m.g. activity during the agonist and antagonist bursts. Five healthy subjects aged between 26 and 35 years were studied.

## RESULTS

Fig. 1*A* shows a typical single fast thumb flexion through 14° at about 350 deg/s. The triphasic pattern of activation of f.p.l. and e.p.l. is evident in these rectified e.m.g. records. Similar pictures were obtained for elbow extension (not illustrated). Proof that the antagonist burst of activity does assist in stopping such a movement is shown

in Fig. 2, in which the subject was asked to make a similar fast movement of thumb flexion through about 14° at the same velocity before and after radial nerve anaesthesia (6 ml 1 % lignocaine injected around the nerve at the elbow). Following the nerve block, antagonist activity was greatly reduced and the subject overshot the mark. This finding was confirmed in another two subjects.

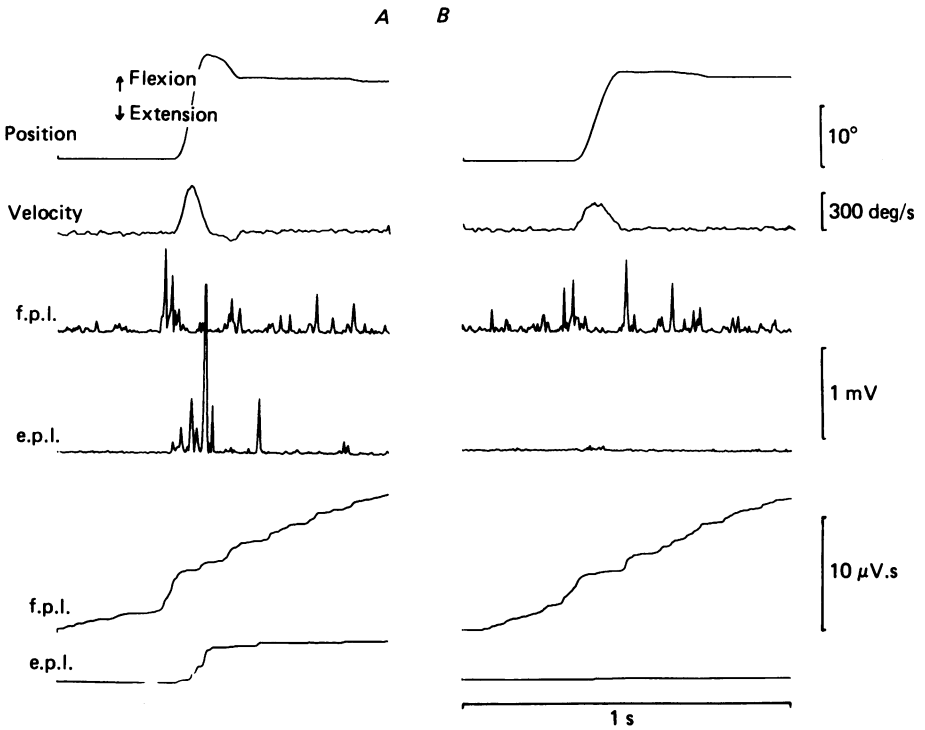



Fig. 1. Fast (*A*) and slow (*B*) single thumb flexions showing the triphasic pattern of muscle activation in agonist (f.p.l.) and antagonist (e.p.l.) muscles. Traces are, from the top down: thumb position, thumb velocity, rectified e.m.g. from f.p.l. and e.p.l., and integrated e.m.g. from f.p.l. and e.p.l. The size of each burst of activity is most clearly seen in the integrated records.

Slower thumb flexion (at about 275 deg/s) through the same angle (14°) is shown in Fig. 1*B*; the agonist burst was smaller, for less impulsive force was required, and the antagonist burst virtually disappeared. Fig. 3 compares the f.p.l. and e.p.l. integrated e.m.g. activity required to move through the same angle at different speeds (Fig. 3*A*), or through different angles at the same speeds (Fig. 3*B*). The size of these agonist bursts in f.p.l. is related mainly to the velocity of movement, while that of the antagonist is a function of both velocity and angle. These relationships are summarized for one subject in Fig. 4 for both the thumb flexion (Fig. 4*A*, *C*) and elbow extension (Fig. 4*B*, *D*). E.m.g. activity of the agonist of the thumb (f.p.l.) is shown in Fig. 4*A* and of the elbow (triceps) in Fig. 4*B*; activity of the antagonist of the thumb (e.p.l.) is shown in Fig. 4*C* and of the elbow (biceps) in Fig. 4*D*. Identical results were obtained in the other four subjects.

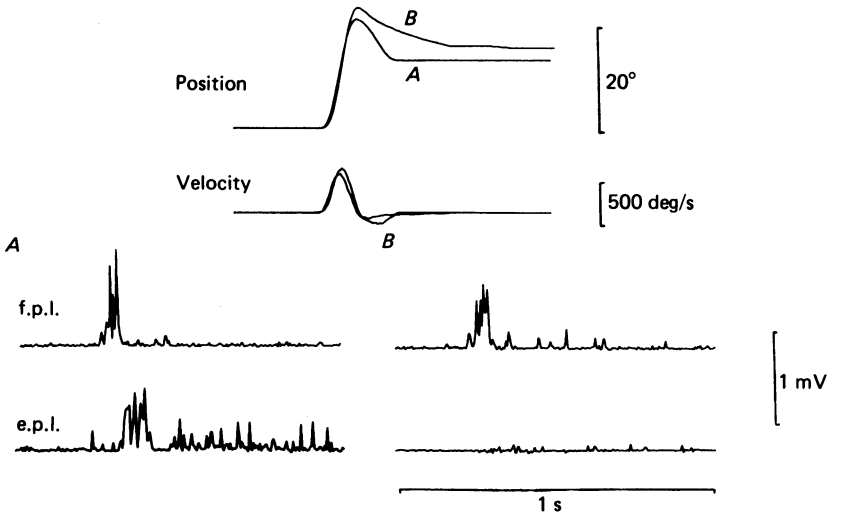



Fig. 2. The effect of radial nerve anaesthesia on the accuracy of rapid thumb movements. Single trials from the same subject are shown before and after the block, with the subject attempting to reach the same peak velocity of movement in each case. The upper two records show superimposed position and velocity traces without (*A*) and with anaesthesia (*B*), and the bottom two records the rectified e.m.g.s from f.p.l. and e.p.l. Virtual abolition of extensor muscle activity results in positional overshoot if the same velocity of movement is maintained.

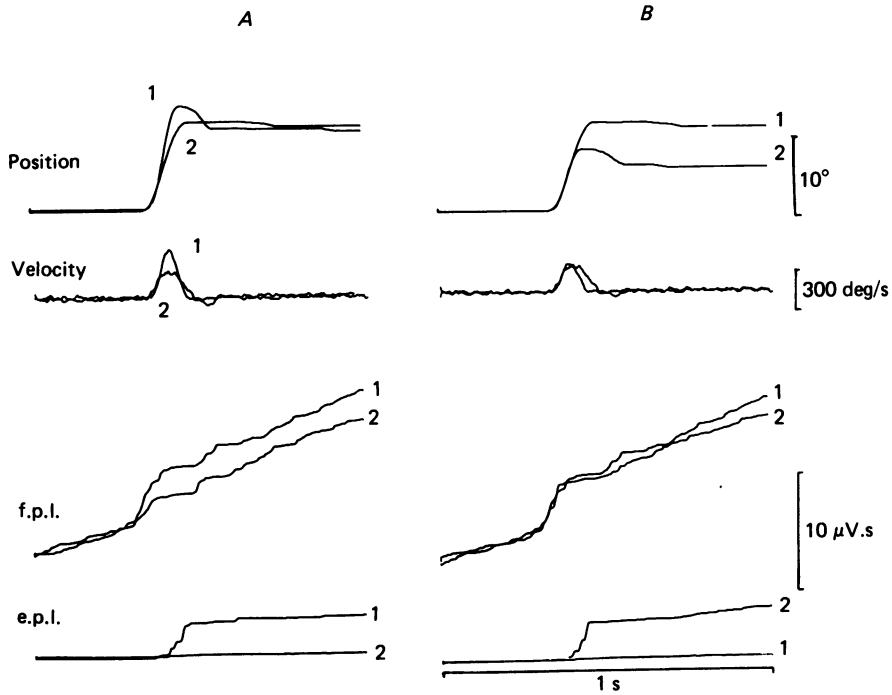



Fig. 3. Contrasting amounts of muscle activity in agonist (f.p.l.) and antagonist (e.p.l.) during thumb movements made at different speeds to the same end-position (*A*) and movements made at the same speed to different end-positions (*B*). Superimposed single records (1 and 2) are shown from the same subject, and are, from the top down: thumb position, velocity, and integrated e.m.g. activity from f.p.l. and e.p.l.

Agonist f.p.l. activity was related linearly to the velocity of movement over the range studied ($n = 88$, $r = 0{\cdot}81$, $P < 0{\cdot}001$) (Fig. 4*A*), confirming an earlier observation (Hallett & Marsden, 1979). The size of the movement also determined the amplitude of the f.p.l. burst, but this relation was much more obvious in records from triceps during elbow extensions (Fig. 4*B*). The exact relation between agonist activity and velocity of movement is complex, as illustrated in the values for triceps e.m.g. in movements through 30°, where an exponential curve best fits the data. However, we will not consider this matter further, for our attention here is focussed on the activity of the antagonist.

The size of the antagonist e.m.g. burst in e.p.l. (Fig. 4*C*) and biceps (Fig. 4*D*) obviously was determined by both velocity and magnitude of movement. The faster the movement through a given angle, the greater the size of the antagonist burst. The greatest antagonist activity was seen for small movements at high speeds; the smallest antagonist activity was for large movements at slow speeds.

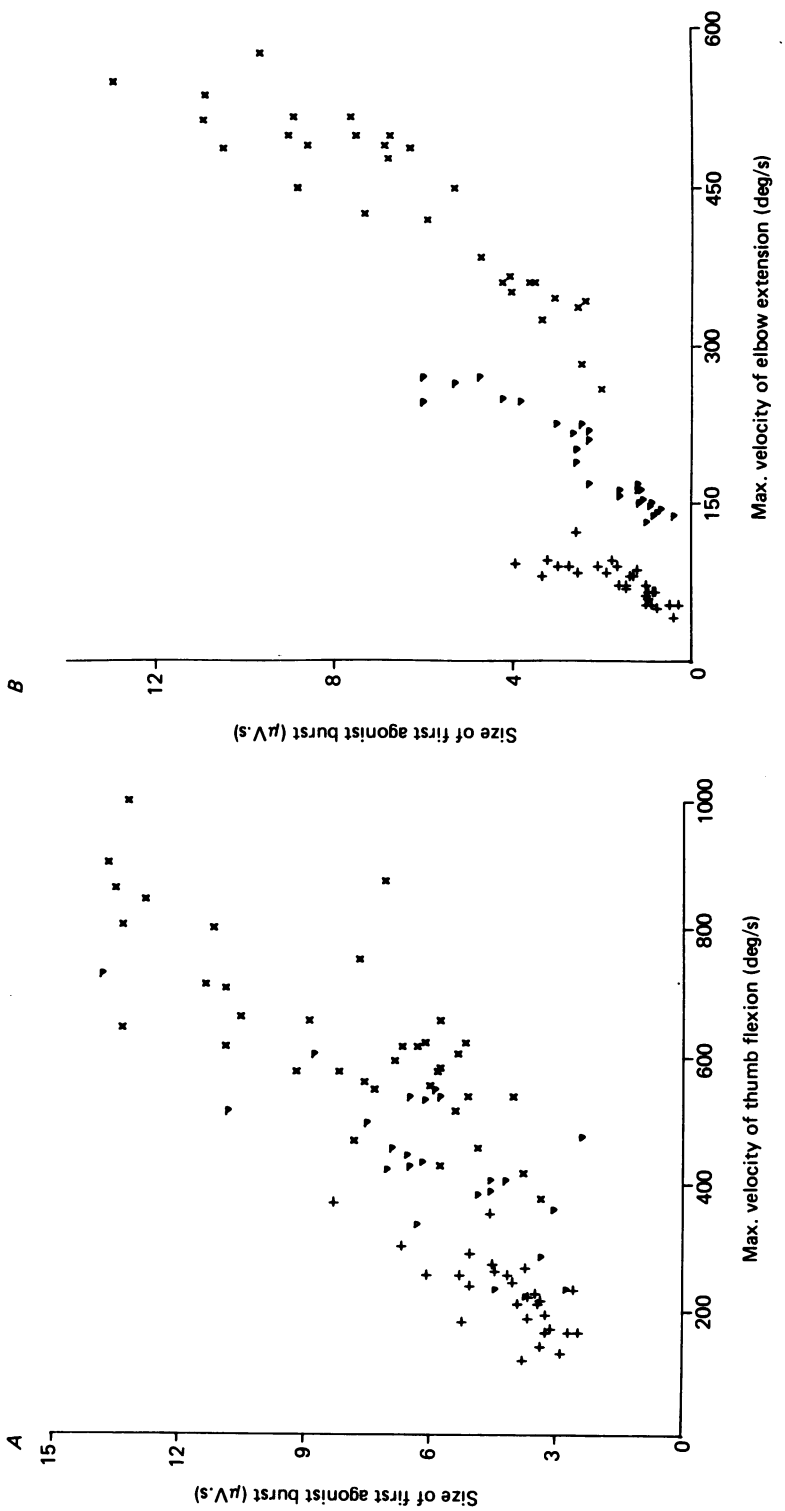
The time of onset of the antagonist e.m.g. burst relative to that of the agonist, also was determined by both the velocity and amplitude of the movement for the thumb (Fig. 5*A*) and elbow (Fig. 5*B*). The antagonist burst started earlier in fast-velocity low-amplitude movements. The antagonist muscle was not activated at a particular point nor at a particular time during these movements.

All the main experiments in this study were performed with movements starting from the same initial joint position. However, we also performed a small number of experiments in which the influence of final end-position on the size of the antagonist burst was investigated. The main result is illustrated by the example shown in Fig. 6. The same subject was asked to perform elbow extensions of the same extent (12°) and peak velocity from different initial joint positions. Fig. 6 *A* shows an elbow movement starting from 60° flexion; *B* shows a movement from 120° flexion. A smaller amount of antagonist activation was required to halt the movement when the end-position was near the extremes of joint rotation.

Finally, a third factor over and above the velocity and amplitude of the movement was shown to govern the activity of the antagonist, namely the subject's knowledge of whether it was required. In Fig. 7 the subject was asked to move quickly through about 14°, but in other runs an end-stop was placed to prevent further movement beyond that point of the lever on which the thumb rested. When the subject knew that the end-stop was in place, the antagonist burst disappeared after two or three trials; as a result, the movement was faster, illustrating the point that the antagonist burst not only normally halts the movement but also slows it.

## DISCUSSION

Action of the antagonist undoubtedly assists in halting a fast movement, but the amount and timing of such antagonist activity is subtly adjusted to circumstances. In the first place, it does not occur at all when it is not necessary, as when the subject knows that the movement will be halted by an end-stop. This partly explains the 'suitcase illusion', in which the subject, believing that he is going to lift a heavy weight, puts in too great a burst of agonist activity and fails adequately to activate the antagonist so that the light valise is forcibly and unexpectedly jerked off the


A
Size of first agonist burst (μV.s)
15
12
9
6
3
0
200
400
600
800
1000
Max. velocity of thumb flexion (deg/s)
B
Size of first agonist burst (μV.s)
12
8
4
0
150
300
450
600
Max. velocity of elbow extension (deg/s)

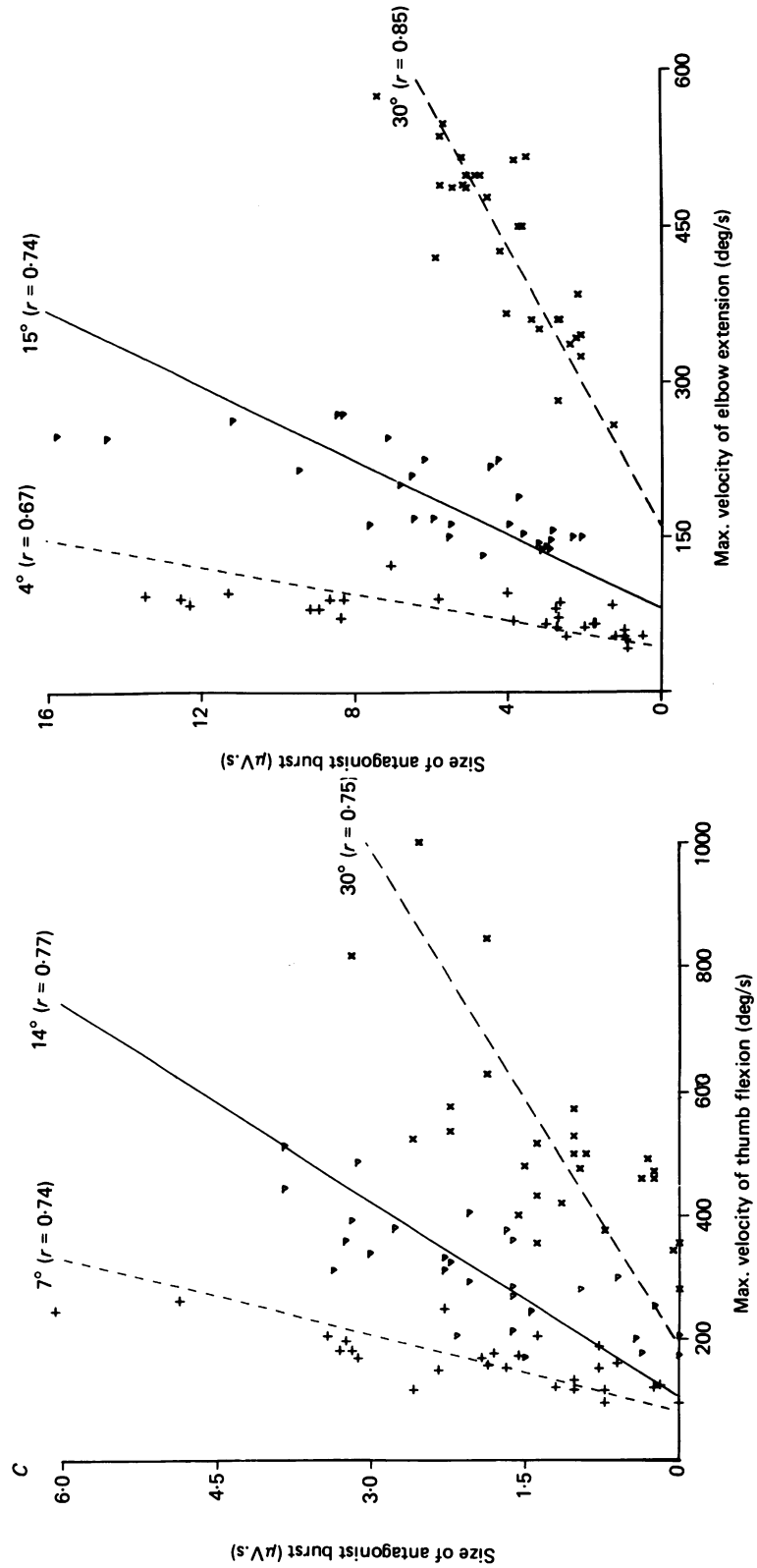



Fig. 4. Relationships between peak velocity of movement and size of first agonist burst (*A* and *B*) or size of antagonist burst (*C* and *D*) for fast thumb flexions or elbow extensions in one subject. Movements were made at a variety of speeds through three different angles (30°, 14° or 7° for the thumb; 30°, 15° or 4° for the elbow). The size of each burst of muscle activity was measured in individual records from integrated e.m.g. traces. The value of the correlation coefficient is given for each of the fitted linear regression lines. ×, large; ▽, medium; +, small movements.

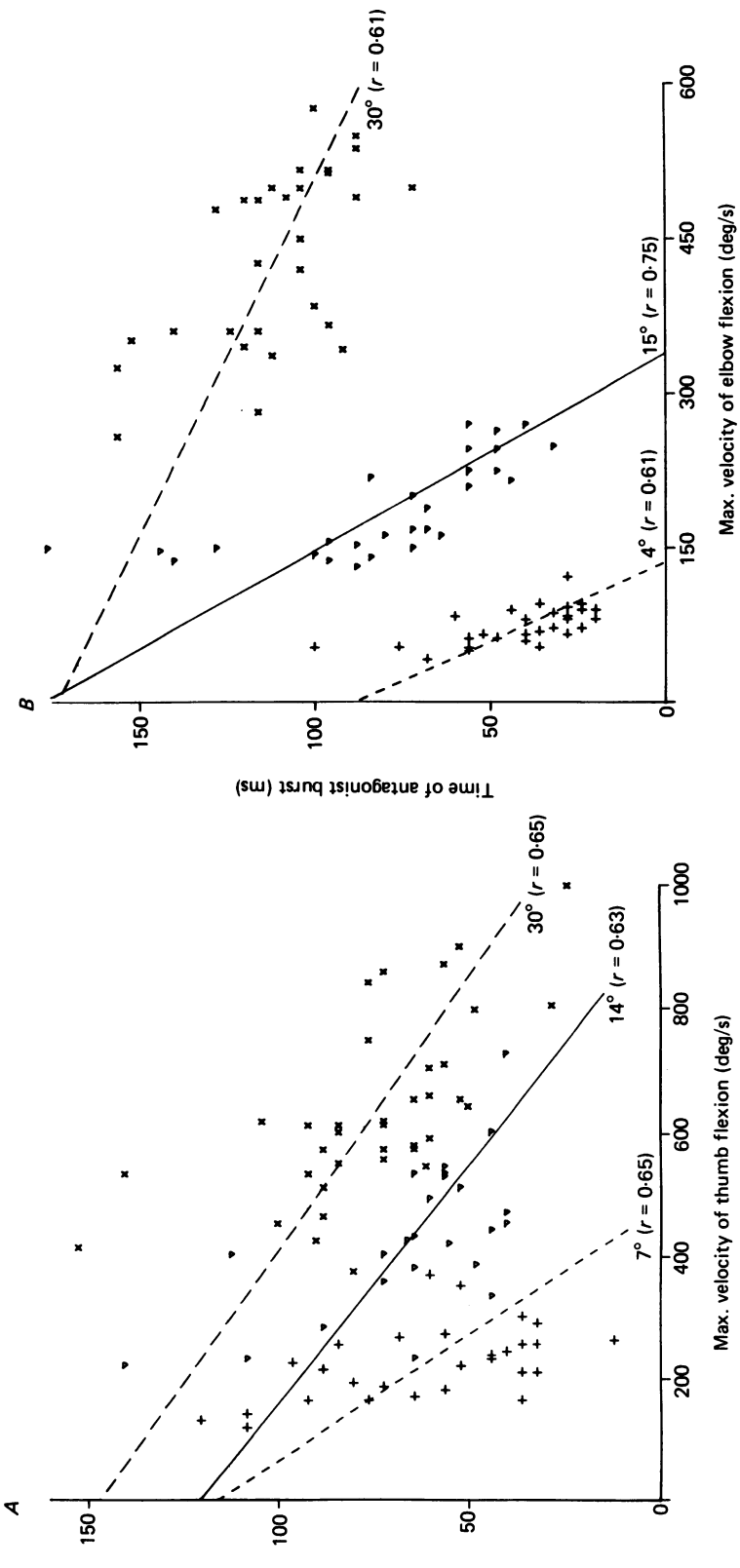



Fig. 5. Relationship between peak velocity of movement and the timing of the onset of the antagonist muscle burst, relative to the onset of the agonist activity, in rapid thumb flexions (*A*) and elbow extensions (*B*) in the same subject. Movements were made through three different distances at a variety of speeds as in Fig. 4. Correlation coefficients are given for the fitted linear regression lines. Symbols as in Fig. 4.

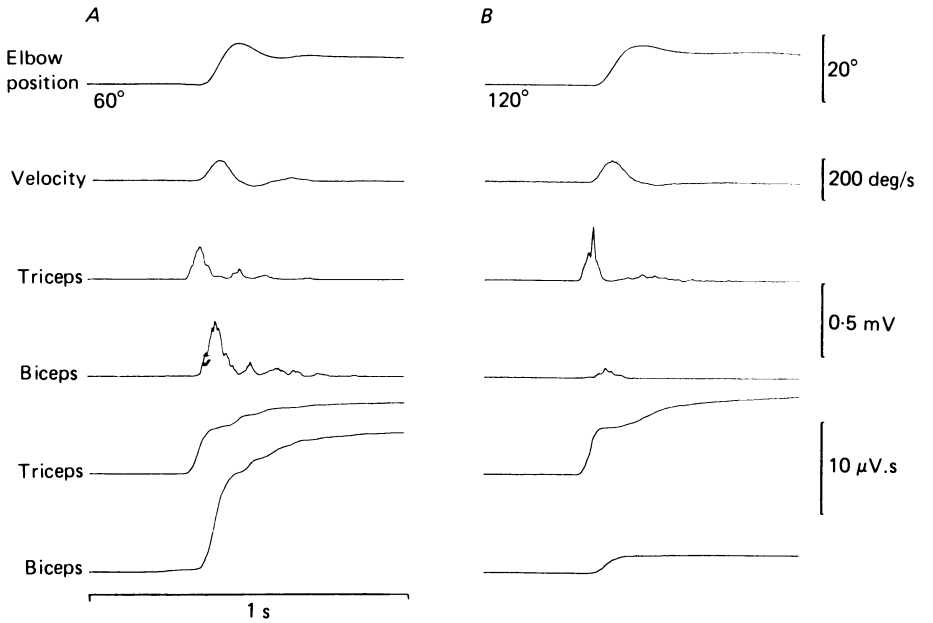



Fig. 6. Average (of eight) rapid elbow extensions of approximately the same extent and peak velocity made by a single subject from different initial starting positions. The movement shown in *A* was made from 60° elbow flexion, whereas that in *B* was made from 120° elbow flexion. The movement in *B* ends much nearer the point of maximum elbow extension than that in *A* and shows a correspondingly smaller amount of antagonist activity in biceps. Traces are, from the top down: elbow position, elbow velocity, rectified surface e.m.g. from triceps and biceps, and integrated e.m.g. from triceps and biceps. The size of each burst is seen most clearly in the integrated records.

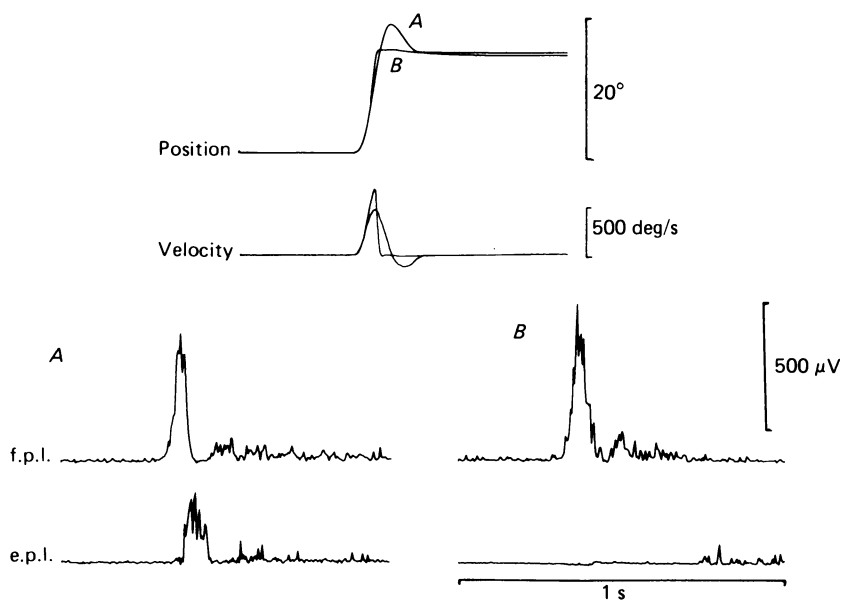



Fig. 7. The effect of a mechanical end-stop, placed in the apparatus with the subject's knowledge, on the activity of the antagonist muscle (e.p.l.) during fast thumb flexions. Traces are the average of ten trials each from the same subject, and are, from the top down: superimposed records of thumb position and velocity, and separated rectified e.m.g.s from f.p.l. and e.p.l. with (*B*) and without (*A*) the stop in place. In the presence of the end-stop antagonist activity is markedly reduced.

ground. The disappearance of antagonist activity in these circumstances also may be explained if one supposes that in the presence of an end-stop subjects attempt to make a movement of 'infinite' extent, knowing that the limb will be halted mechanically at the correct position. Thus, from Fig. 4*C* and *D* the slopes of the lines describing the relationship between velocity and size of antagonist burst decrease

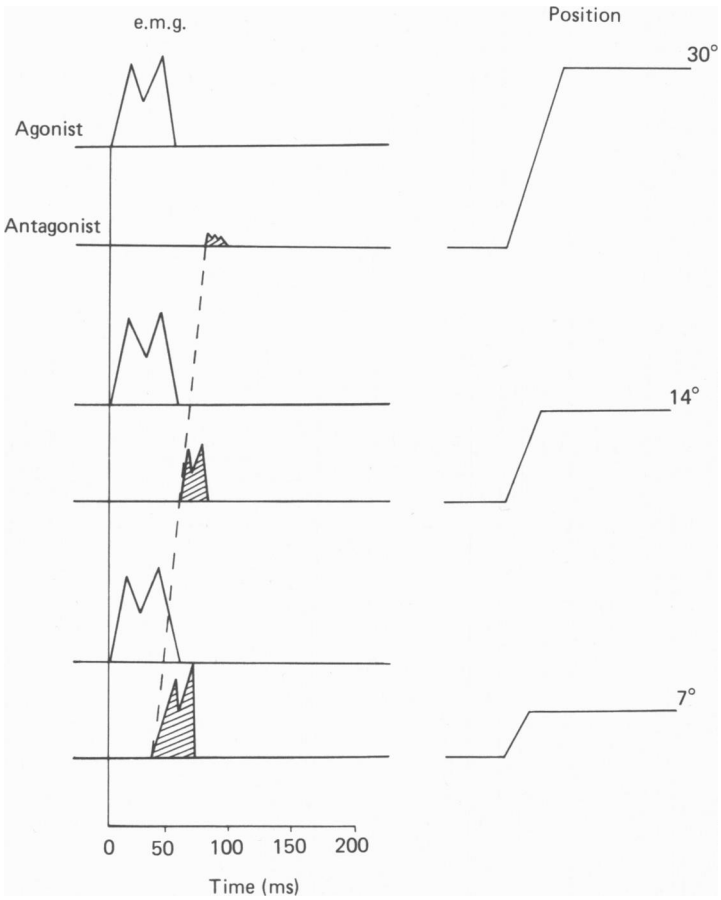



Fig. 8. Diagrammatic summary of the behaviour of the antagonist muscle activity during fast thumb flexions made at the same peak velocity over three different distances. As the distance moved becomes smaller, the antagonist activity is larger and starts earlier in the movement.

progressively for larger and larger movements, so that for movements of 'infinite' extent, no antagonist action would be necessary at any velocity of movement. However, it must be stressed that the antagonist activity does not disappear unless the subject is well aware of the mechanical effectiveness of the end-stop. Any attempt to brake the movement before the stop involves antagonist activity and may explain why this effect was not noted previously by Jacobs, Andrews, Ianonne & Greninger (1980).

It is difficult to predict the exact relationship between the e.m.g. bursts and the

force exerted when a muscle is contracting over different distances at various velocities. However, since all the movements investigated were of the same type, with the bi/triphasic pattern of muscle activation, we shall assume for the rest of the discussion that there is at least a qualitative relationship between e.m.g. and muscle force under these conditions.

When the subject knows that antagonist activity is required, the nervous system automatically and subconsciously adjusts its size and time of onset to brake the movement. These effects are illustrated diagrammatically in Fig. 8. The earlier the antagonist burst begins, the greater will be its effect on velocity. For a given time of onset, the greater its amplitude so the more it will restrict the size of the movement. Of course, this is just what is required for small, fast movements; slow, large movements encounter increasing restriction imposed by the passive mechanical properties of the limb. These comprise the visco-elastic components provided by joints, tendons and muscles themselves, all of which tend to restrict speed and amplitude of movement. In fact, such visco-elastic properties are not linear over the range of joint movement, but increase considerably at the extremes of rotation. Accordingly, smaller antagonist activity is required when the joint is moved throughout its range of excursion than if it is only moved through a few degrees in its mid-range. With regard to velocity of movement, undoubtedly the passive visco-elastic properties do tend increasingly to restrict faster movements. However, by themselves they are inadequate to halt fast movements, particularly since these generate additional inertial forces, which are obviously greater for the forearm than for the terminal phalanx of the thumb. Accordingly, antagonist activity occurs earlier and is greater for faster movements, particularly for those at the elbow compared with those at the top joint of the thumb. This difference between the elbow and thumb is evident when comparing Fig. 4*A* and *B*, and Fig. 5*A* and *B*.

Finally, there is one feature which may help simplify the present results. From the data in Fig. 4 it can be seen that movements made through the same angle at a variety of speeds will preserve a simple, linear relation between the size of the first agonist and antagonist bursts. The timing of the antagonist burst will also be a simple function of the size of the agonist burst. Thus, for movements made over a given distance, selection of the amplitude of the agonist burst will determine not only the peak velocity of movement, but also the size and timing of the antagonist burst. We suggest that the simplicity of this relation underlies the observation that many of our subjects made during the experiment, that once they had learned the distance to move, they could perform the movement at any required velocity without further practice and without substantial loss of accuracy.

In summary, if the subject wants to move through a small distance quickly, the antagonist burst is large and starts soon after the agonist fires; if they wish to move a long way slowly, the antagonist burst is small and late. The relation between these two parameters of timing and size of the antagonist burst is not simple (see Fig. 9). Thus, the timing of the antagonist burst is not specified solely by its size, but is also a complex function of the movement amplitude. The central nervous system appears to be able to regulate both parameters independently according to circumstance. When these results showing the complex control of antagonist muscle activity are coupled with those of Brown & Cooke (1981) on the independent control of the first

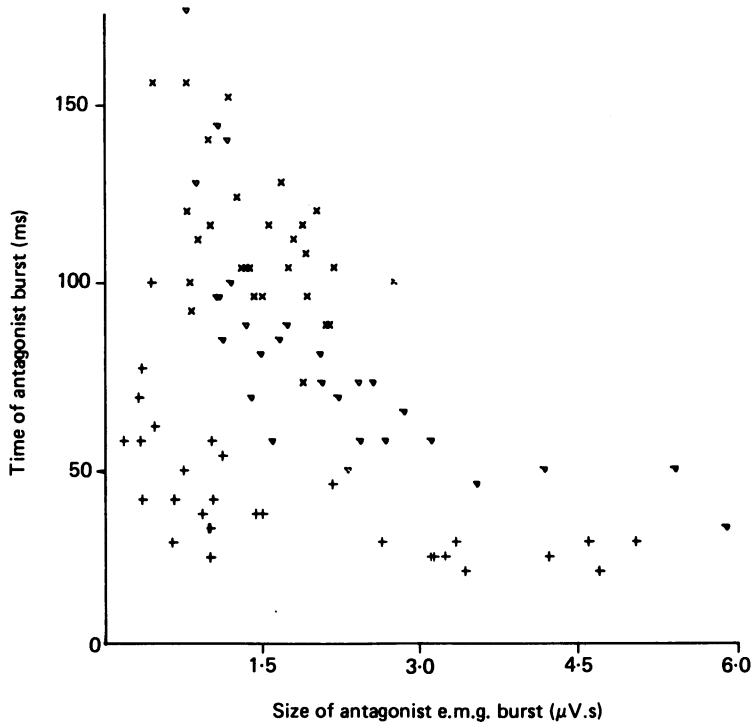



Fig. 9. The relationship between the size and timing of the antagonist muscle activity during fast elbow extensions. As in Figs. 4 and 5 the subject made a variety of movements at different speeds over three different distances. Symbols as in Fig. 4. No clear relationship emerges between these two parameters.

and second agonist bursts, the common triphasic pattern of muscle activation responsible for fast limb movements can no longer be viewed as a simple immutable 'programme' generated by the central nervous system, but can be seen to be carefully adjusted to the precise parameters of the task. What role afferent feed-back from the moving limb may play in these adjustments is unknown, but observations on deafferented men (Hallett, Shahani & Young, 1975; Day, Marsden, Obeso, Rothwell & Traub, 1982) show that the triphasic pattern can be generated in the absence of any feedback. However, details of the adjustment of size and timing of the bursts have not been investigated in these patients, and peripheral effects cannot be ruled out.

We should like to thank Mr H. C. Bertoya for his help in building and maintaining the equipment used in these experiments. This work was supported by the Medical Research Council.

## REFERENCES

Brown, S. H. C. & Cooke, J. D. (1981). Amplitude- and instruction-dependent modulation of movement-related electromyogram activity in humans. *J. Physiol.* **316**, 97–107.

Day, B. L., Marsden, C. D., Obeso, J. A., Rothwell, J. C. & Traub, M. M. (1982). Manual motor function in a deafferented man. *J. Physiol.* **320**, 23–24*P*.

Hallett, M. & Marsden, C. D. (1979). Ballistic flexion movements of the human thumb. *J. Physiol.* **294**, 33–50.

HALLETT, M., SHAHANI, B. T. & YOUNG, R. R. (1975). EMG analaysis of stereotyped voluntary movements in man. *J. Neurol. Neurosurg. Psychiat.* **38**, 1154–1162.

JACOBS, M. B., ANDREWS, L. T., IANONNE, A. & GRENINGER, L. (1980). Antagonist EMG temporal patterns during rapid voluntary movement. *Neurology, Minneap.* **30**, 36–41.

LESTIENNE, F. (1979). Effects of inertial load and velocity on the braking process of voluntary limb movements. *Expl Brain Res.* **35**, 407–418.

WACHOLDER, K. & ALTENBURGER, H. (1926). Beiträge zur Physiologie der wuhrlichen Bewegung. X. Einzelbewegungen. *Pflügers Arch. ges. Physiol.* **214**, 642–661.