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BRAKING OF FAST AND ACCURATE ELBOW FLEXIONS IN THE MONKEY

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SUMMARY

1. The processes responsible for braking fast and accurate elbow movements were studied in the monkey. The movements studied were made over different amplitudes and against different inertias. All were made to the same end position. Only fast movements that showed the typical biphasic or triphasic pattern of activity in agonists and antagonists were analysed in detail.

2. For movements made over different amplitudes and at different velocities there was symmetry between the acceleration and deceleration phases of the movements.

3. For movements of the same amplitude performed at different velocities there was a direct linear relation between peak velocity and both the peak acceleration (and integrated agonist burst) and peak deceleration (and integrated antagonist burst).

4. The slopes of these relations and their intercept with the peak velocity axis were a function of movement amplitude. This was such that for large and small movements of the same peak velocity and the same end position (i) peak acceleration and phasic agonist activity were larger for the small movements and (ii) peak deceleration and phasic antagonist activity were larger for the small movements.

5. The slope of these relations and the symmetry between acceleration and deceleration were not affected by the addition of an inertial load to the handle held by the monkey.

6. The results indicate that fast and accurate elbow movements in the monkey are braked by antagonist activity that is centrally programmed. As all movements were made to the same end position, the larger antagonist burst in small movements, made at the same peak velocity as large movements, cannot be due to differences in the viscoelastic contribution to braking (cf. Marsden, Obeso & Rothwell, 1983). Instead we suggest it results from the property of symmetry of movements, i.e. these smaller movements have larger phasic agonist bursts. This symmetry may reflect the nature of the central mechanism that generates the phasic antagonist burst.

INTRODUCTION

Below a certain threshold of agonist force, slow human arm movements can be braked by passive viscoelastic forces (Lestienne, 1979). Above this threshold there is an additional active component to braking. This active component appears as a

burst in the electromyogram (e.m.g.) of the antagonist muscle and is proportional to the velocity of the movement (Lestienne, 1979). Initially it was reported that for human thumb (Hallett & Marsden, 1979) and elbow movements (Brown & Cooke, 1981) the size of the antagonist burst was independent of movement amplitude. However, a more recent study demonstrated that the antagonist burst is proportional to both movement amplitude and peak velocity (Marsden, Obeso & Rothwell, 1983). This was taken as evidence that the central 'programme' that generates the triphasic pattern must be specifically adjusted for the precise velocity and amplitude of each movement.

To study the neural activity of central structures responsible for these e.m.g. patterns it is necessary to perform experiments on animals. The present study was undertaken as a forerunner of such neural recording experiments. The aim was to investigate the characteristics of fast and accurate elbow movements of monkeys and thereby to determine whether the monkey acts as a model for human arm movements. The results indicate that the characteristics of braking are similar in monkeys and humans. They also indicate that new interpretations of the data are necessary. For example, in both human and monkey, large movements of the same peak velocity as small movements have a smaller antagonist burst. For the human movements this result was interpreted as being due to the greater viscoelastic braking forces at extremes of joint rotation (Marsden *et al.* 1983). However, this cannot be the case for the present movements, which were made to the same end position. Instead we suggest that it is due to an attempt by central programming to maintain symmetry between acceleration and deceleration.

METHODS

Experiments were performed on five Cebus monkeys. Monkeys were trained to hold a handle, pivoted at the elbow, within an extension target zone and then, after the target jumped to a new position, to move the handle to the new flexion position. Monkeys were rewarded with grape drink if the handle arrived in the new target within 0·7 s of the target jump and remained within the target for 0·4 s. Thus monkeys had to make prompt and accurate movements to gain reward. Target and handle position were displayed on an oscilloscope in front of the monkey. Monkeys were required to make a series of twenty-five to a hundred movements between the same extension and flexion positions. Then the starting extension target was shifted to a new position while the terminal flexion position remained fixed and another series of movements was recorded. The displacement between extension and flexion position was varied from 20° to 60° with target widths of 7° to 12°. Inertia was increased by attaching metal clamps to the handle.

Recordings were made of handle position from a thin film potentiometer. E.m.g. activity was recorded by means of fine wires inserted into biceps, brachialis, brachioradialis and triceps muscles. These signals were digitized on-line with a sampling rate of 500 Hz by a PDP 11/44 computer, block averaged into 4 ms bins and stored in digital form on magnetic tape. Movement onset and termination were defined as the points at which the velocity, determined by off-line differentiation of individual position records, reached or fell below a threshold of 25 deg/s. The computer also determined the time and magnitude of peak velocity and, by differentiating velocity, the value of peak acceleration and deceleration (defined as acceleration and deceleration magnitude: Fig. 1*A*). The duration of the acceleration phase was defined as the time from movement onset to peak velocity and the duration of the deceleration phase as the time from peak velocity to movement termination (Fig. 1*A*). Integration of e.m.g. activity was performed by computer analysis. E.m.g. activity of the agonist burst was integrated over a time period from 75 ms before onset of movement to 125 ms after onset of movement, and that of the antagonist from 80 ms before peak velocity

to 160 ms after peak velocity. Movements were selected for analysis which showed a biphasic or triphasic e.m.g. pattern in the agonist and antagonist muscles (Hallett, Shahani & Young, 1975) and which terminated in the flexion target without oscillations or secondary corrections. Such movements constituted about 80 % of the movement trials.

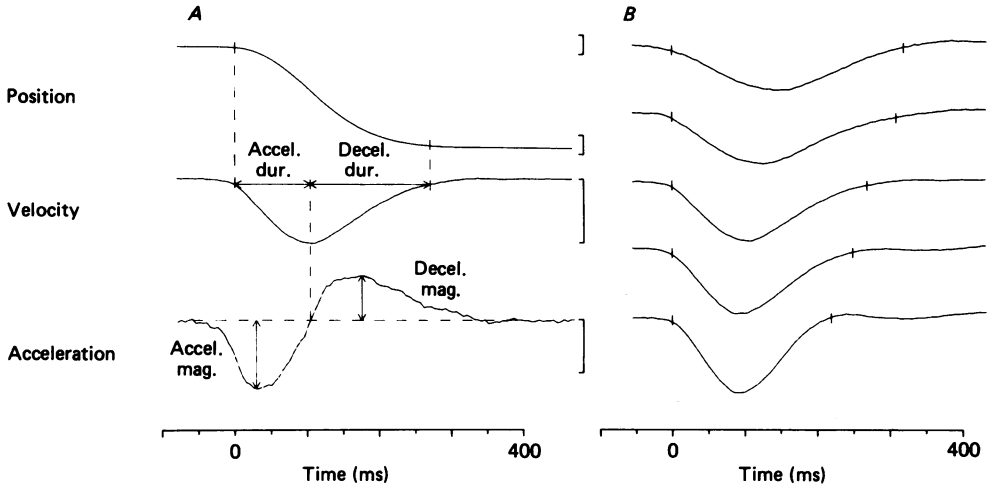

Fig. 1. *A*, records of handle position, velocity and acceleration during an elbow flexion. Vertical bars on position and velocity traces represent movement onset and termination determined when a velocity threshold of 25 deg/s was crossed. Acceleration and deceleration durations were measured as the time from movement onset to peak velocity and the time from peak velocity to movement termination respectively. Acceleration and deceleration magnitudes were measured to peak values as shown. Calibrations: target width, 8°; velocity, 250 deg/s; acceleration, 2000 deg/s^2. *B*, velocity traces of five flexion movements with different peak velocities. Vertical bars represent onset and termination of the movement. Monkey DU.

RESULTS

As do humans (Lestienne, 1979), trained monkeys made accurate flexions of the elbow in which the velocity was unimodal. For a given amplitude of target displacement movements were made with a range of peak velocities and movement durations. In spite of the range of peak velocities the rising and falling phases of velocity retained a strong tendency for symmetry (Fig. 1*B*). Thus small-velocity movements had relatively long durations and small magnitudes of both acceleration and deceleration while large-velocity movements had relatively short durations and large magnitudes of both acceleration and deceleration. This symmetry was examined quantitatively by plotting the magnitude of acceleration against the magnitude of deceleration for movements of different amplitudes. Fig. 2 shows these relations for two monkeys. Although individual movements show some variability in the relation of these parameters (Flament, 1983; Flament, Hore & Vilis, 1982), over-all there was a linear relation and therefore a tendency for symmetry. In some monkeys there was a tendency for the symmetry to be slightly skewed, with the magnitude of acceleration being slightly greater than the magnitude of deceleration.

A consequence of this symmetry is that, for movements of the same amplitude,

there is an increase in the magnitude of both acceleration and deceleration as velocity increases (Fig. 1*B*), and for movements of the same peak velocity but different amplitudes, the magnitudes of acceleration and deceleration are larger for the smaller movements (Fig. 3*A* and *B*). For deceleration this indicates that the braking force is dependent not only on velocity but also on movement amplitude. As deceleration is

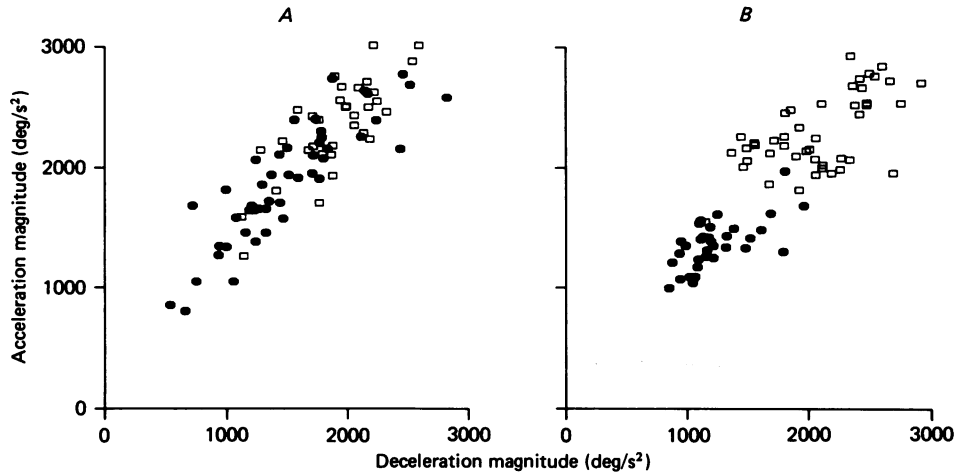

Fig. 2. Symmetry between magnitudes of acceleration and deceleration. Movements between targets separated by: 60° open symbols, 30° filled symbols (*A*); 52° open symbols, 18° closed symbols (*B*). *A*, monkey Ho; *B*, monkey DU.

due in part to active antagonist contraction, this suggests that for movements of two different amplitudes but the same peak velocity, the antagonist e.m.g. burst should be larger for the smaller movements.

To confirm this, recordings were made of e.m.g. activity for movements of different amplitudes performed at various velocities. Fig. 4 shows movement parameters and e.m.g. activity in brachialis and triceps for four representative movements – two through 64° (Fig. 4*A*) and two through 40° (Fig. 4*B*) – from the same experiment. First it is clear that for both movement amplitudes there is an increase in phasic agonist and phasic antagonist activity as peak velocity increases. Secondly for movements of the same peak velocity but different amplitude (movements 1 and 4 in Fig. 4) the phasic burst of the antagonist (triceps) is larger for the smaller movement.

The relations between peak velocity and integrated activity of the first agonist and first antagonist bursts for all the movements recorded from this same experiment are shown in Fig. 3*C* and *D*. To represent total agonist activity as accurately as possible, integrated activity from the three agonists for each movement was summed together. Presumably the scatter on the graphs for triceps would have been reduced if e.m.g. activity from the three triceps heads had been recorded and summed together. These relations are similar to those for the corresponding movement parameters shown in Fig. 3*A* and *B*. As predicted they illustrate that for movements of different amplitude but the same peak velocity the phasic agonist and antagonist bursts are larger for the smaller-amplitude movements.

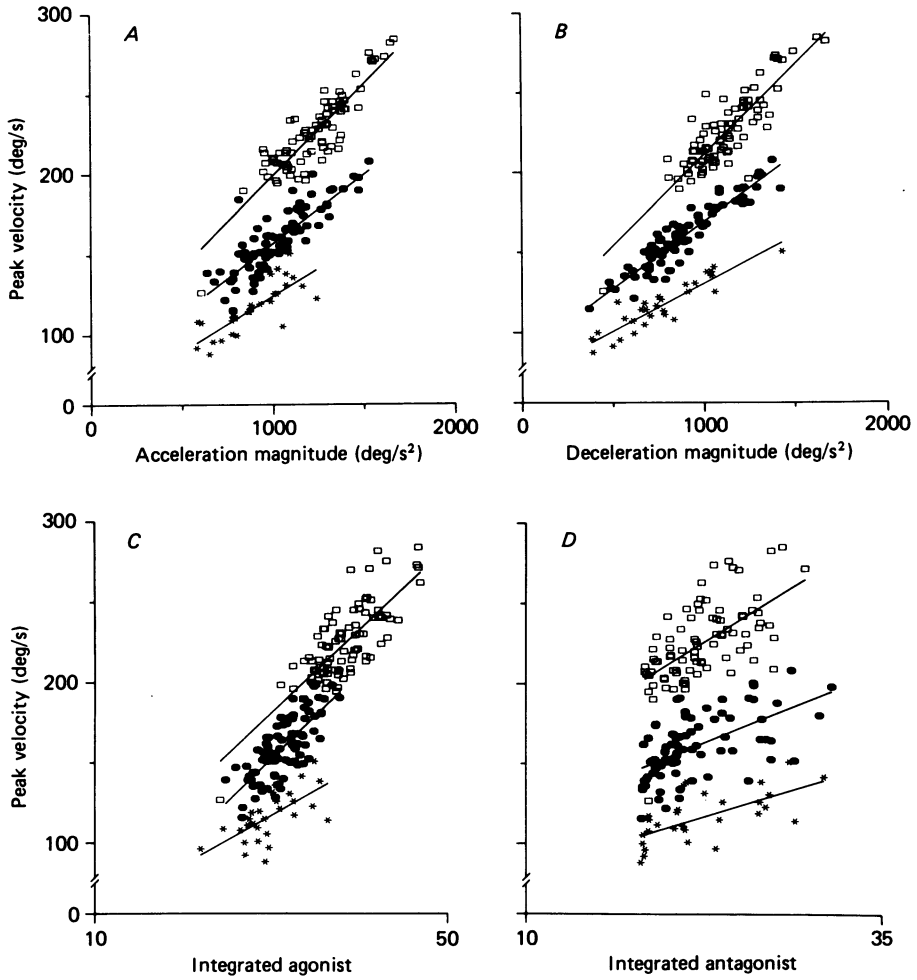

Fig. 3. Relations between peak velocity and magnitude of acceleration (*A*), magnitude of deceleration (*B*), integrated activity of the first agonist burst (*C*) and integrated activity of the first antagonist burst (*D*). Movements were between targets separated by: 60° open symbols, 35° closed symbols, 18° star symbols. E.m.g. activity was in arbitrary units. Regression line correlation coefficients were: *A*, open symbols 0·89, filled symbols 0·85, star symbols 0·79; *B*, open symbols 0·87, filled symbols 0·93, star symbols 0·91; *C*, open symbols 0·78, filled symbols 0·77, star symbols 0·65; *D*, open symbols 0·61, filled symbols 0·58, star symbols 0·65. Monkey MI.

The effects of added inertia on these relations were also investigated. Fig. 5 compares two movements of similar amplitudes and peak velocities but different inertias. As shown by Lestienne (1979), for human elbow movements, increasing the inertia (Fig. 5*B*) increased the size of the phasic agonist and antagonist bursts in movements of similar velocity. In addition, the onset of the antagonist burst was earlier relative to peak velocity as was the agonist relative to start of movement. The consequence of these e.m.g. changes was that the acceleration and deceleration magnitudes were very similar in movements made against different inertias. While

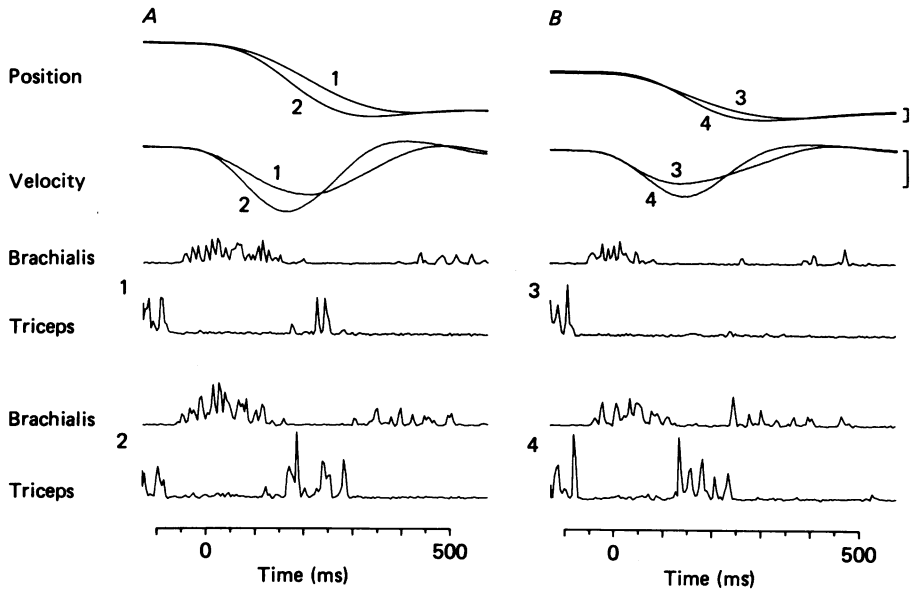

Fig. 4. Rectified e.m.g. activity from agonist (brachialis) and antagonist (triceps) during single trials of movements of two different amplitudes performed at two different velocities. All records were obtained in the same recording session. Movements 1 and 2 were 64° in amplitude (*A*); movements 3 and 4 were 40° in amplitude (*B*). Peak velocity of movement 4 was the same as that of movement 1. Position calibration represents flexion target of width 12°. Velocity calibration: 100 deg/s. Monkey MI.

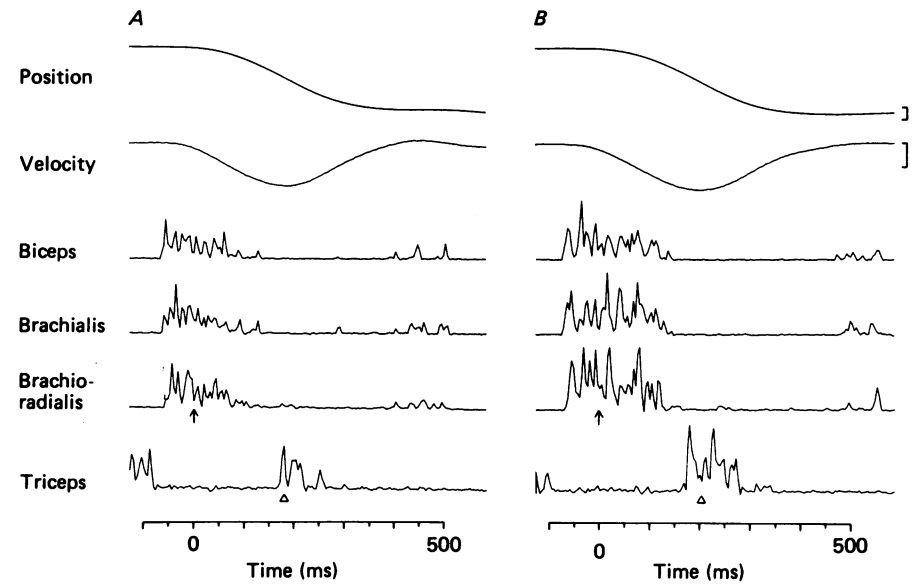

Fig. 5. Recordings of position, velocity and e.m.g. activity of biceps, brachialis, brachioradialis and triceps for movements of similar amplitudes and similar peak velocities performed under conditions of different inertia. *A*, normal handle; *B*, 100 g mass added to handle. Movements were from the same experiment and were made between targets separated by 52°. Arrow, start of movement; triangle, time of peak velocity. Position calibration represents flexion target width 12°. Velocity calibration: 100 deg/s. Monkey MO.

on the average, movements against added inertia were slower, the relations between peak velocity and acceleration magnitude and peak velocity and deceleration magnitude were the same for movements of the same amplitude made against different inertias (Fig. 6).

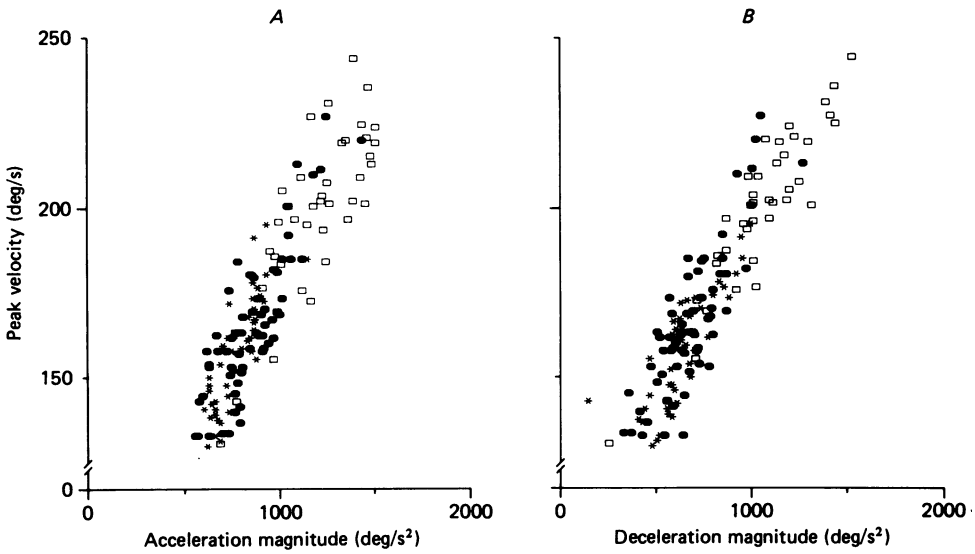

Fig. 6. Relations between peak velocity and acceleration magnitude (*A*) and deceleration magnitude (*B*) for movements performed with different inertias. Open symbols, normal handle; filled symbols, 40 g mass added to handle; star symbols, 100 g mass added to handle. Movements between targets separated by 52°. Monkey MO.

DISCUSSION

These results on monkey elbow movements confirm those of Marsden *et al.* (1983) on human thumb and elbow movements. Both studies found that there was a linear relation between peak velocity and the amount of antagonist activity needed to stop the movement and that this relation was dependent on movement amplitude. A dependence on movement amplitude was also found by Hoffman & Strick (1982) for the antagonist burst in human wrist movements.

These findings indicate that braking of movements in a step tracking task cannot be due to simple stretch reflexes. If it were due to simple stretch reflexes it would be expected that movements of different amplitude but the same peak velocity made to the same end position should have the same-sized antagonist burst. In fact all studies show that for a given peak velocity, the smaller the movement the larger is the antagonist burst. This indicates that the braking of these movements of different amplitude is under some degree of central control.

Similarly if movements were braked solely by simple reflexes the same antagonist burst would be generated for movements of the same peak velocity irrespective of the inertia. Consequently if the inertia were increased there would be a smaller deceleration magnitude. However, for an increase in inertia a larger and earlier antagonist e.m.g. burst is generated such that the deceleration magnitude is

unchanged (Figs. 5 and 6). Thus the central control adjusts the antagonist e.m.g. burst for movements of different amplitude and for movements made under different load conditions.

A different conclusion about the mechanism of braking has come from studies using a different behavioural task. In a task which involved monkeys performing an elbow flexion after overcoming a resistive load which rapidly decayed, Soechting, Ranish, Palminteri & Terzuolo (1976) found that the e.m.g. of the agonist and antagonist was related to velocity. They concluded that this was incompatible with the motor output being generated by central pre-programming. Similarly Ghez & Martin (1982) found that if a brake was unexpectedly released as cats made isometric adjustments in force, the time of occurrence of the antagonist burst was similar to that of a stretch reflex. In this latter experiment the magnitude of the antagonist burst was found to be inversely related to that of the first agonist burst. In contrast in the present study and that of Marsden *et al.* (1983), which both involved a step tracking task, the magnitude of the antagonist burst was shown to be directly related to that of the agonist. This emphasizes that the mechanism of braking may be different in different behavioural tasks.

The present results and those of Marsden *et al.* (1983) emphasize a major role of central programming in generating the phasic antagonist activity. The finding that there was relatively less antagonist activity for large movements was previously explained by the fact that the viscoelastic forces were larger at the extremes of joint rotation (Marsden *et al.* 1983). Conversely large antagonist activity was thought to be required for small movements made in the mid-range of joint rotation where the viscoelastic forces were smaller. This explanation is unlikely for our results because in our paradigm both large and small movements of the same peak velocity had the same end position and thus were subject to the same viscoelastic forces. Instead we suggest that the larger antagonist burst for the small movements is due to an attempt by central programming to maintain symmetry between the acceleration and deceleration phases of the movement. Thus for movements of the same peak velocity the smaller movements have a larger deceleration magnitude and a larger phasic antagonist burst because they have a larger acceleration magnitude and a larger agonist burst than the larger movements.

One speculation as to why symmetry occurs is that for movements of a given peak velocity symmetry will minimize the sum of the magnitudes of acceleration and deceleration, which may in turn minimize energy expenditure (Flament, 1983). Another possible significance of symmetry is that it is a feature which may simplify the way in which movements are generated. The results show that for movements over a particular distance both acceleration magnitude (and agonist activity) and deceleration magnitude (and antagonist activity) covary with peak velocity (Figs. 3 and 4). As concluded by Marsden *et al.* (1983) it follows that for movements of a certain amplitude a direct relation exists between the size of the first agonist and antagonist bursts. One way this could occur would be if the antagonist command was generated on the basis of the agonist command (efference copy).

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