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REVIEW ARTICLE

Single-joint rapid arm movements in normal subjects and in patients with motor disorders

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Summary

In normal subjects the execution of single rapid one-joint movements is characterized by an electromyographic (EMG) pattern composed of three discrete bursts of activity: two bursts (first and second agonist bursts, or AG1 and AG2) are present in the agonist muscle separated by an almost complete period of electrical silence. During this pause, another burst (antagonist burst, or ANT) occurs in the antagonist muscle. If a rapid movement is executed during tonic activation of the agonist muscle, tonic activity is inhibited just prior to AG1 onset (agonist inhibition). Similarly, if the movement is performed during tonic activation of the antagonist muscle, such activity is also inhibited prior to AG1 onset (antagonist inhibition). Antagonist inhibition also starts prior to AG1 onset and lasts until ANT onset. A general descriptor of the kinematic features related to the EMG pattern described above is a symmetrical and unimodal velocity profile that is bell-shaped and shows an acceleration time roughly equal to the deceleration time. This holds true for movements performed under low accuracy constraints; as accuracy demands become stricter and stricter, the peak velocity decreases but, as long as the movement is made with one continuous trajectory, the velocity profile remains roughly symmetrical. In general terms, the function of AG1 is to provide the impulsive force to start the movement; the function of ANT is to halt the movement at the desired end-point; and the function of AG2 is to dampen out the oscillations which might occur at the end of the movement. The timing and size of the bursts vary according to the speed and amplitude of the movement. The origin of the EMG pattern is a central programme, but afferent inputs can modulate the voluntary activity. In this paper, we also review the EMG and kinematic abnormalities that are present during the execution of single-joint, rapid arm movements in patients with Parkinson's disease, Huntington's disease, Sydenham's chorea, dystonia, athetosis, cerebellar deficits, upper motor neuron syndrome, essential tremor and large-fibre sensory neuropathy. The data from these studies lead us to the following conclusions: (i) the basal ganglia have a role in scaling the size of AG1, reinforcing the voluntary command and inhibiting inappropriate EMG activity; (ii) the cerebellum has a role in timing the voluntary bursts and probably in implementing muscle force phasically; (iii) the corticospinal tract has a role in determining spatial and temporal recruitment of motor units; (iv) proprioceptive feedback is not necessary to produce the triphasic pattern but it contributes to the accuracy of both the trajectory and the end-point of rapid movements.

Keywords: rapid movements; arm; normal subjects; motor disorders

Abbreviations: AG1 and AG2 = first and second bursts of EMG activity in the agonist muscle; ANT = burst of EMG activity in antagonist muscle

Introduction

More than 50 years ago Wacholder and Altenburger (1926) made the first EMG recording of a single-joint voluntary movement of the upper arm. These authors reported that slow movements were characterized by continuous EMG activity that was present in both agonist and antagonist muscles. In contrast, rapid movements were characterized by alternating EMG bursts in the agonist and antagonist muscle. These observations have been confirmed and extended. Movements performed at different joints and under different types of instructions have been studied, and the EMG activity has been matched with kinematic parameters such as position, velocity and acceleration of the arm.

Here we review what is known about the control of rapid limb movements in normal subjects and in patients with movement disorders. We define such movements by both the instruction given to the subject, and the resulting velocity profile of the movement. The instruction must emphasize speed: for example 'As fast and accurate as possible', or 'As fast as possible'. In addition, subjects must make the movement in a single action, without the need for corrective adjustments during its course. The resulting velocity profile is usually bell-shaped, with a single peak reached about halfway through the movement (*see* below). Similar velocity profiles are often observed in movements involving more than one joint, suggesting that it reflects some basic principle of rapid movement control.

Normal subjects

The EMG pattern of rapid movements is characterized (Angel, 1974; Hallett *et al.*, 1975*a*) by two bursts of phasic muscle activity in the agonist muscle (AG1 and AG2), separated by an almost complete electrical silence. During this pause, another burst of phasic activity occurs in the antagonist muscle (ANT). The combination of AG1, ANT and AG2 is commonly called the 'triphasic pattern' (Figs 1 and 2). In general terms, the function of the AG1 is to provide the impulsive force to start the movement; the function of the ANT is to halt the movement at the desired end-point; and the function of AG2 is thought to be to damp out oscillations which might occur at the end of the movement.

This pattern of EMG activity produces a smooth movement from one position to another in which the velocity is maximum approximately halfway through the trajectory. The velocity time course characteristically has a bell-shaped profile in which the acceleration time (the time from start to peak velocity) is roughly equal to the deceleration time (Brown and Cooke, 1990; Hallett *et al.*, 1991). If the movement has to be terminated with significant accuracy, then the peak velocity decreases but, as long as the movement continues to be made in a single action, without mid-trajectory correction, the velocity profile remains roughly symmetrical (Brown and Cooke, 1990). Fitt's law (Fitts, 1954) describes the logarithmic relationship between the degree of accuracy and the speed of movement.

The first agonist burst

The behaviour of the AG1 has been studied extensively in movements at the elbow, wrist and thumb. For a given load, the integrated size of AG1 is proportional to the peak acceleration of the movement (Gottlieb *et al.*, 1989) and hence the EMG provides an estimate of the force produced by the muscle. The amount of EMG activity in AG1 can be adjusted by varying the duration and/or the amplitude of the burst. For example, when movements are made over increasing distances or with increasing loads, the duration of AG1 remains constant over the initial range of movement, whilst its amplitude increases (Freund and Budingen, 1978; Hallett and Marsden, 1979; Brown and Cooke, 1981). Beyond a certain range, which depends on the strength of the subject as well as the nature of the load (e.g. unloaded wrist movements made to amplitudes >30°), the duration may also increase. Moreover, when a muscle is fatigued, or if the inertial load is increased, the duration of AG1 may lengthen (Angel, 1974; Berardelli *et al.*, 1984*a*; Brown and Cooke, 1984; Benecke *et al.*, 1985; Pantaleo *et al.*, 1988; Hoffman and Strick, 1993) (Fig. 3). As is often the case with volitional movement, this behaviour can be modified by instruction. For example, Cooke and Brown (1994) have shown that the duration of the AG1 can remain constant over a wide range of inertial loads if subjects are given feedback to maintain the duration of the initial limb acceleration constant.

There have been many attempts to describe how the duration and amplitude of AG1 vary in different tasks. However, no one set of rules has been found that is adequate for all types of movement. One suggestion that has been put forwards is that the duration of AG1 is linked to the duration of the initial phase of limb acceleration (i.e. the time to peak velocity) (Cooke and Brown, 1994). Although this is often the case, it does not hold true if we compare movements made against different loads. In unloaded movements, the duration of AG1 may be relatively short, but if the viscosity of the load is increased, the duration of AG1 lengthens. Despite this, the maximum velocity of both movements may occur at the same time (Gottlieb *et al.*, 1995). One reason for this is that the time of maximum velocity can be influenced by the timing of the antagonist activity as well as by the duration of AG1.

Two other models, proposed in order to explain the form of the AG1, have been described by Gottlieb *et al.* (1989), and Corcos *et al.* (1989). Neither are sufficient on their

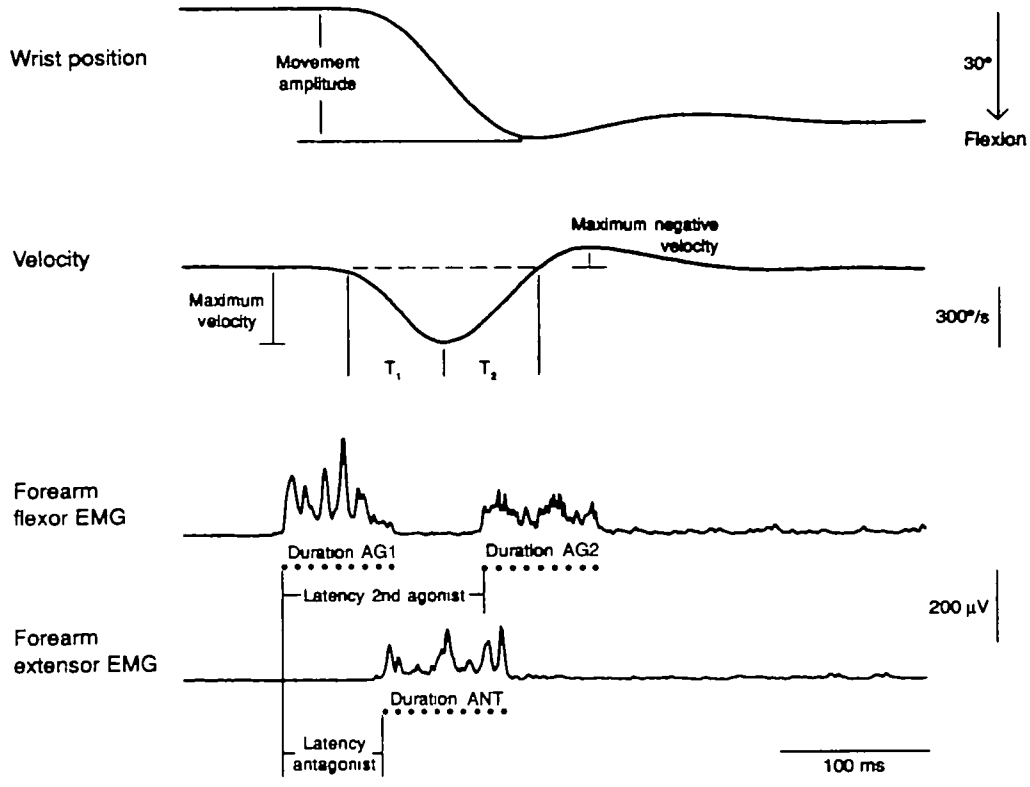

Fig. 1 Representative example of a rapid wrist flexion movement in a normal subject. Wrist position and movement velocity together with the rectified surface EMG activity in the forearm flexor and extensor muscles are shown. T1 indicates the acceleration time and T2 indicates the deceleration time. From Britton *et al.* (1994), with the authors' permission.

own to account for all aspects of the behaviour of AG1. Nevertheless, it is useful to review them here since they give some insight into how the nervous sytem may control rapid limb movements in certain situations. Before describing these in detail it is important to remember that both models encompass a description of the nature of the input to the motor neuron pool rather than the EMG signal that is actually recorded. The importance of this distinction is that motor neuron discharge in the spinal cord, and motor unit firing in muscle are likely to filter this input. Thus, if the input consisted of a pulse, the EMG would not be expected to rise and fall instantaneously. It would probably rise and fall exponentially at a rate proportional to the height of the input. The two models which describe this input are known as the 'pulse height' and the 'pulse width' hypothesis. Pulse height control assumes that the input to the agonist motor neuron pool is of constant duration, and hence that amount of excitation is graded only by changing the height of this pulse. Because of the filtering properties of the neuromuscular system, this model does not automatically imply that the EMG will be of constant duration. In fact, the amplitude of the pulse will determine the rate at which EMG activity rises at the start and then declines at the end. So, a higher pulse will give a wider, as well as higher, EMG burst. The relationship between height and width depends on the time constant chosen for the system. There are no full models in which this has been calculated. However, it is easy to see that a pulse height model on its own would be inadequate to describe the very large increases in duration of AG1 which occur without a concomitant increase in amplitude during movements made with a fatigued muscle, or against large viscosities (e.g. Gottlieb *et al.*, 1995).

The second model is that of width control. A constant excitatory pulse is given to the motor neurons for different lengths of time. Obviously, the duration of the measured EMG will change for wider pulses. However, the height of the EMG burst will also increase since more time is available for activity to rise towards its maximum when the length of the input is extended. Like the pulse height model, pulse width cannot account for all types of contraction. The model predicts that the rate of rise of the EMG activity in AG1 is proportional to the amplitude of the input pulse. This should be constant in the pulse width model, but would vary in the pulse height model. At the elbow, the initial rate of rise of the AG1 during flexion movements is approximately constant over a wide range of types of movements, suggesting a prominent role for pulse width control. However, the data also show that when movements are small and performed without any external load, then the slope of the initial part of the agonist EMG activity becomes smaller (Corcos *et al.*,

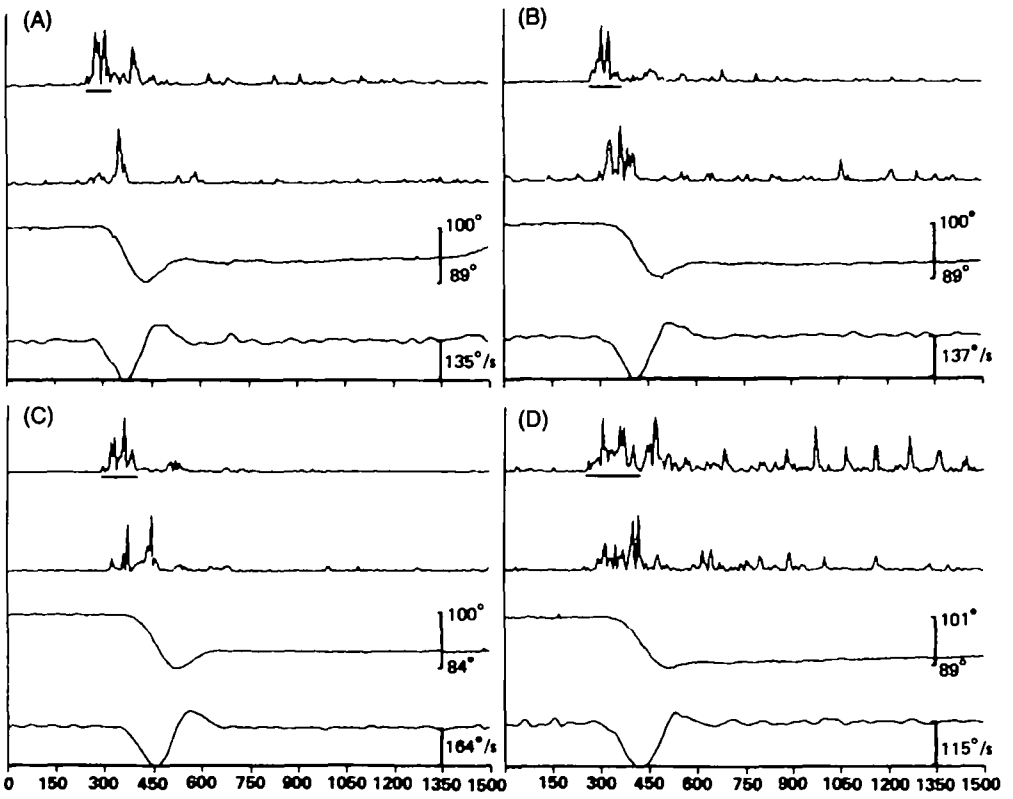

Fig. 2 Kinematic and EMG features of individual rapid flexion movements at the elbow in a normal subject. The four traces in each panel are, from top to bottom, rectified biceps and triceps EMG, angular position, and velocity. The line under the EMG bursts of the biceps records indicates the measurement of the duration of AG1 in each record. **A** shows a clearly formed triphasic pattern, while **B–D** show different degrees of co-contraction of the antagonist muscle during AG1. From Hallett *et al.* (1991), with the authors' permission.

1989). This suggests that for these movements, the pulse height model is also important.

A physiological hypothesis to account for the mixture of results, which is actually observed, is that the excitatory input to motor neurons has a finite minimum which is of the order of 70 ms or so. This would explain why voluntary EMG bursts also have a minimum duration of 70–80 ms. Such a limit would have the effect that when a small pulse of force was required to move the limb over a small distance, then the amount of activation could only be controlled by varying the height of this pulse. When larger (or longer lasting) force pulses are required then both the height and the width could be varied. There is no physiological reason why these two processes should be independent. Increasing the duration of the burst should be a particularly effective way of increasing the force produced by AG1 (Garland *et al.*, 1994). In a burst of 70 ms or so, many motor units discharge two or three times but this is insufficient for the production of maximum tension. A train of 5–10 impulses per motor unit is needed before maximum tension is achieved. Thus, increasing the duration of the AG1 beyond its minimum 70 ms allows summation of the force output from a larger number of impulses and produces a larger impulsive force for the movement.

Finally, it is pertinent to ask why the AG1 changes in size at all with different amplitudes of movement. If we wish to move as fast as possible, then why not use the maximum possible AG1 for all movements, and then activate the antagonist at an appropriate point in order to halt the motion at the desired end position? Apart from the problems that there might be in stabilizing such a system, it is also important to consider the relationship between EMG excitation and force production in a muscle which is allowed to shorten. Muscles act as a low pass filter of EMG activity. In the simplest isometric case, with the AG1 lasting a minimum of 70 ms, the peak force produced by muscle occurs only after 100–150 ms. The muscle then relaxes to its baseline force level after a further 200 ms or so. Thus, the total minimum duration of a phasic voluntary contraction would be of order of 300 ms. In a shortening muscle, it is much more difficult to predict the time course of the force pulse, since the force exerted declines (following the force–velocity: length–tension relationships) as soon as the muscle begins to change length. However, at least in small amplitude limb movements which are complete within 50–100 ms, it seems likely that the applied muscle force will outlast the movement itself and be wasted. This excess force must be balanced by overlapping antagonist activity if the limb is not to move beyond a desired

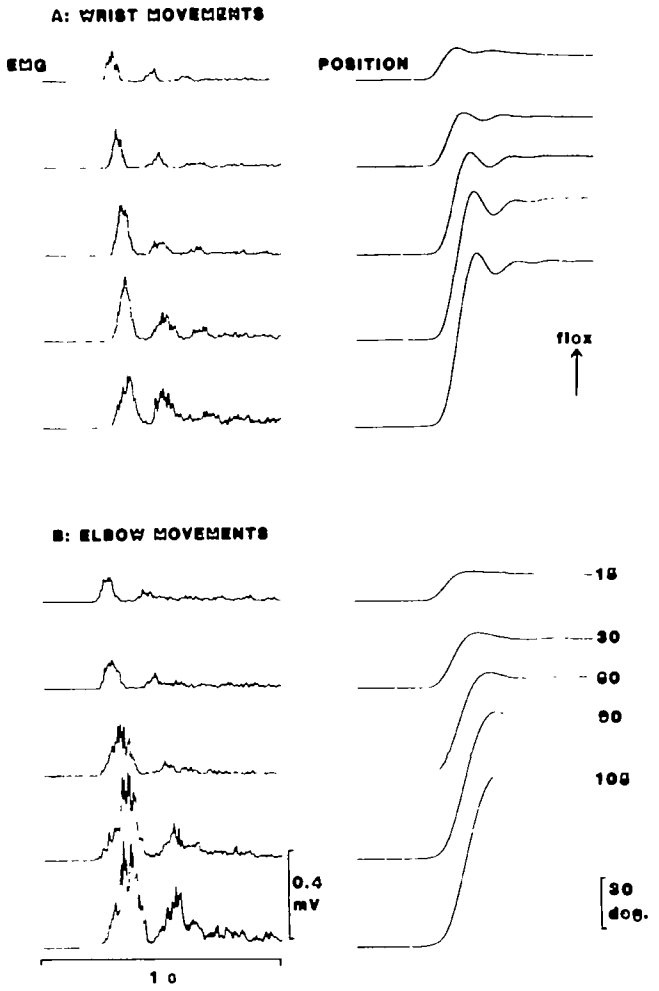

Fig. 3 Behaviour of AG1 during rapid wrist (**A**) and elbow (**B**) movements of different amplitude in a normal subject. The EMG signal from wrist flexors or biceps is on the left, while the angular position of the wrist or elbow is on the right. Each record is the average of 15 trials. Note that during both wrist and elbow movements the size and duration of AG1 increases as movement amplitude increases. From Berardelli *et al.* (1984*a*), with the authors' permission.

end-point. Reducing the size of the AG1 in small movements may be a way to minimize this wasted energy.

Inhibition of the agonist muscle

If a rapid movement is executed during tonic agonist activation, tonic activity is inhibited just prior to AG1. The agonist inhibition lasts from 20 to 140 ms (Conrad *et al.*, 1983; Mortimer *et al.*, 1987) and occurs in both prime movers and synergists (Mortimer *et al.*, 1987). The duration of agonist inhibition correlates positively with the amplitude of AG1 and movement distance, peak velocity (Conrad *et al.*, 1983) and peak acceleration (Mortimer *et al.*, 1987). Agonist inhibition exhibits similar features during reaction time or self-paced rapid movements. It has been proposed that its function is to increase the synchrony of the motor neuron pools upon which the voluntary command will impinge. An alternative hypothesis is that put forward by Aoki *et al.* (1989). These authors found that the agonist inhibition during rapid elbow movements had two effects on dynamic extension force: it reduced the force prior to movement, whilst increasing the force measured at the beginning of elbow extension. Aoki *et al.* (1989) suggest that the function of agonist inhibition might be that to increase the agonist force production by a 'pre-stretch' of agonist muscle fibres.

Antagonist burst

During rapid movements the antagonist muscle is activated phasically after the onset of AG1, and during the electrical pause between AG1 and AG2 (Hallett *et al.*, 1975*a*; Angel *et al.*, 1977). Co-contraction of the antagonist with the agonist often occurs and while it is usually possible to separate the co-contraction from the antagonist burst itself, it is sometimes difficult to decide whether the start of the antagonist contraction was co-contraction or an early start of the burst. The function of the antagonist burst is to help to decelerate the movement so that the limb halts at the intended end position (*see* Barnett and Harding, 1955; Brown and Cooke, 1990). If a subject knows that the movement will be terminated by a mechanical stop, then the size of the antagonist burst is greatly reduced and may even disappear (Waters and Strick, 1981; Marsden *et al.*, 1983). The same behaviour appears if the end-point of a given rapid movement is not specified (Mustard and Lee, 1987). In other words, the presence of ANT is linked to the intent to terminate a movement actively. The findings indicate that ANT can be generated independently from AG1.

The size of the antagonist burst depends upon the amount of force needed to halt the limb. This in turn depends on the inertia of the load, the speed of movement and the passive mechanical braking forces which act at the joint (Barnett and Harding, 1955; Lestienne, 1979; Brown and Cooke, 1981; Marsden *et al.*, 1983; Meinck *et al.*, 1984). Thus, small fast movements in the mid-range of joint position which are made against a large inertia require a large antagonist burst of activity to halt them; larger movements made to the end of the range of joint positions without any inertial load require much less activity since much of the braking force is provided by the visco-elastic properties of the joint. Thus, if movements of constant load are made to the same end position from different starting points, the amplitude of the antagonist activity is approximately proportional to the velocity of movement (Flament *et al.*, 1984). Since the load and the visco-elastic properties of the joint are approximately the same in these movements then the amount of braking force depends only upon the kinetic energy acquired by the limb (*see* Karst and Hasan, 1987). The slope of the relationship between antagonist size and velocity, however, changes if the end-point (Marsden *et al.*, 1983) or the load (Lestienne, 1979) are varied. Additional factors which influence the size of ANT are the length–tension and force–velocity relationships of the muscle. The ongoing movement passively

stretches the antagonist, so that the force it develops depends upon the length and the velocity of stretch at the time of activation. Such factors obviously complicate the relationship between ANT size and parameters of movement (Gottlieb *et al.*, 1989).

The latency and duration of the antagonist muscle burst is affected by movement parameters (Brown and Cooke, 1981). When movements are made over a small distance, then their duration is short, and the antagonist muscle burst shows an early onset and a short duration. As the distance to be moved is increased, the duration of the movement lengthens, and the antagonist muscle burst shows a later onset and a longer duration. Similar effects can be seen with changes in limb inertia.

Inhibition of the antagonist muscle

Tonic antagonist activity is suppressed prior to movement. This is sometimes called the Hufschmidt phenomenon (Hufschmidt and Hufschmidt, 1954; Hallett *et al.*, 1975*a*). Inhibition of ANT begins before the onset of AG1 and lasts until the onset of ANT (Hallett *et al.*, 1975*a*). Lack of overlap of motor programmes for tonic holding and phasic movement may help in the co-ordination of the overall movement. Additionally, this helps to generate force in the direction of the intended movement, although a recent study (Agostino *et al.*, 1992) has shown that the precise relationship between movement parameters and the amount and duration of antagonist suppression is complex.

The second agonist burst

The ANT is usually followed by the appearance in the agonist muscle of a second phasic activity. Studying the relationship between AG2 and the kinematic features of the movement, Brown and Cooke (1981) have shown that the amplitude of AG2 varies linearly with movement distance, whereas the latency shortens when the movements become faster. The AG2 is more variable than AG1 and ANT and this is true for both proximal (Wadman *et al.*, 1979) and distal (Hallett and Marsden, 1979) arm movements. The necessity for AG2 arises from the low pass filtering properties of muscle. As noted above, the minimum duration of a voluntary force pulse is of the order of 200–300 ms. Since some movements are shorter than this, additional EMG bursts are required to cut short the force produced by the primary agonist and antagonist activities. The AG2 is the first of these and actively terminates the decelerative force pulse produced by ANT (Wadman *et al.*, 1979; Brown and Cooke, 1990). As an example, consider a short movement reaching its peak amplitude after only 150 ms. The minimum duration force pulse generated voluntarily by AG1 may last for 300 ms. The job of the ANT is therefore not only to halt the limb movement by 150 ms but also to cancel out the agonist force pulse at any time after that. The problem is that to achieve this, the antagonist force profile cannot be a time shifted version of the agonist force pulse, otherwise the limb will not halt after 150 ms. The solution that the nervous system adopts is to start ANT very shortly after AG1 so its force exceeds the agonist force early in the movement and reverses the direction of limb movement after 150 ms. Thereafter, antagonist force usually exceeds that of AG1. The AG2 is then necessary to halt the reversal in limb position. In more generalized terms, the function of AG2 is stabilization of the limb after movement termination by dampening oscillations (Ghez and Martin, 1982; Meinck *et al.*, 1984).

Co-contraction and co-activation

One difficulty in measuring the triphasic EMG pattern is due to the presence of co-contraction of the muscle pair during the different bursts of the triphasic pattern (Fig. 2B–D). Co-contraction is reduced if the subjects are relaxed and may be related to stiffness of the joint during the movement. A different phenomenon is co-activation. By this we mean the overlap in time of AG1, ANT and AG2 EMG bursts. As explained above, this results from the necessity to cut short the force output produced by consecutive EMG bursts. It is particularly prominent in rapid, small amplitude movements.

Relationship between the 'triphasic pattern' and the movement velocity profile

Virtually all rapid limb movements between two points have a bell-shaped velocity profile. This is true whether single-joint or multi-joint movements are examined. Movements with different kinematic and kinetic features show the same velocity profile after scalar transformation of amplitude, duration or inertial load (*see* Brown and Cooke, 1990). Since it is so ubiquitous, the question arises as to whether the bell-shaped profile reflects a fundamental constraint in the output of the CNS which tells us something about the rules used by the brain to control movement. However, the pattern is not immutable (Brown and Cooke, 1990); by changing the instruction to subjects, it is possible to change the shape of the velocity profile whilst keeping peak velocity and movements duration constant. Thus, subjects can produce movements with short acceleration time and longer decelerations, or vice versa. When they do this, the EMG bursts change their timing and size in an appropriate fashion. The authors make clear, however, that the EMG changes related to changes of movement amplitude (*see* Hallett and Marsden, 1979) or movement amplitude and duration (*see* Berardelli *et al.*, 1984*a*; Brown and Cooke, 1984; Benecke *et al.*, 1985) are not due to modifications of the symmetry ratio *per se*, since this measure is relatively constant across these different movements. What these movements have in common are the concomitant changes in magnitude and duration of the acceleration and deceleration. Brown and Cooke (1990) and Cooke and Brown (1990) suggest that during a rapid voluntary movement, changes in the EMG

Table 1 *Electromyographic and kinematic features of single-joint rapid arm movement in patients with motor disturbances*

	Athetosis	Dystonia	Huntington's disease	Cerebellar ataxia	Upper motor neuron syndrome	Parkinson's disease	Essential tremor
Amplitude variability	Increased	Increased	Increased	Increased	—	Normal	—
Peak velocity	Decreased	Decreased	Decreased	Decreased	Decreased	Decreased	Normal
Velocity profile	—	Normal	—	Prolonged acc. phase	—	—	High peak deceleration
AG1 duration	Prolonged	Prolonged	Prolonged	Prolonged	Prolonged	Normal	Normal
ANT1 duration	Prolonged	Prolonged	Prolonged	Prolonged	Prolonged	Normal	Normal
AG/ANT pattern	Co-contracting	Co-contracting	Co-contracting	Normal	Normal	Normal	Normal

— = not studied.

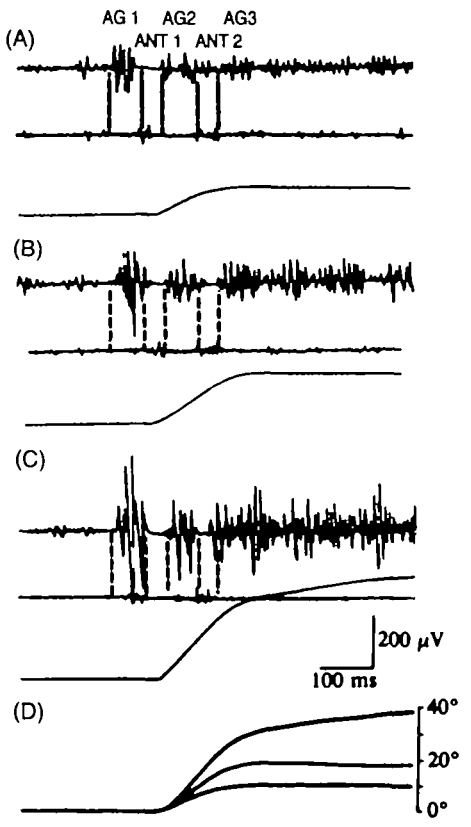

Fig. 4 Individual rapid flexion movements at the elbow in a patient with Parkinson's disease. The amplitude of the movements were of 10° (**A**), 20° (**B**) and 40° (**C**). The three traces in these panels are, from top to bottom, biceps and triceps EMG and angular elbow position. **D** shows the three position traces superimposed. Note that the patient required additional cycles of EMG burst to complete the movements. Note also that this patient was able to increase the amplitude of AG1 as the length of the movement increases. From Hallett and Khoshbin (1980), with the authors' permission.

bursts are determined by the characteristics of acceleration and deceleration phases (*see* also Flament *et al.*, 1984). A more general conclusion is that the bell-shaped velocity profile is only a favoured and not an exclusive solution for moving between two stationary points.

Role of peripheral feedback

The three EMG bursts of the triphasic pattern and premovement inhibition of antagonist activity are present and of normal duration in deafferented patients with large-fibre sensory neuropathy (Hallett *et al.*, 1975*a*; Rothwell *et al.*, 1982; Cooke *et al.*, 1985; Sanes *et al.*, 1985). However, both the trajectory and the end-point of rapid movements were inaccurate for most patients (Sanes *et al.*, 1985). Forget and Lamarre (1987) have suggested that was due, in part, to improper adjustment of the size and time of onset of the antagonist burst, which was small and poorly correlated with the size of AG1 in the three patients they studied. The conclusion is that somaesthetic afferent information is not necessary to produce the triphasic pattern, but that it can contribute to the accuracy of the final output [*see* also studies in normal subjects by Hallett and Marsden (1979) and Sanes and Jennings (1984)].

Besides the observations in deafferented patients, other data support a central origin of the triphasic pattern. After blockage of the motor nerve for the antagonist muscles with lidocaine, AG2 was present (Garland *et al.*, 1972); when the forearm was passively extended during a rapid flexion movement, ANT was almost unchanged (Hallett *et al.*, 1975*a*); when the movements were not voluntarily braked ANT was absent (Waters and Strick, 1981; Marsden *et al.*, 1983; Mustard and Lee, 1987); finally, during a voluntary movement, ANT was present, but disappeared during a passive movement of similar distance and velocity (Mustard and Lee, 1987). On the other hand, AG2 and ANT can certainly be influenced by stimuli travelling along Ia afferents (Barnett and Harding, 1955; Terzuolo *et al.*, 1974; Ghez and Martin, 1982).

Patients with motor disturbances

The results obtained for patients with motor disturbances are summarized in Table 1.

Parkinson's disease

In their pioneering study, Draper and Johns (1964) asked patients to perform rapid pronation/supination movements of

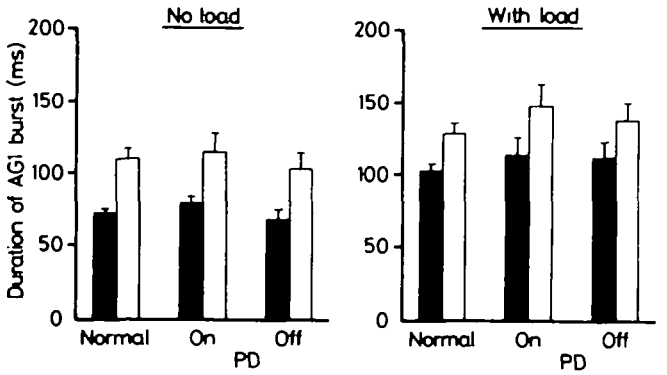

Fig. 5 The duration of the first agonist EMG burst in eight normal subjects and in 10 patients with Parkinson's disease (PD), on and off therapy, performing rapid wrist flexion movements of 15° (hatched bars) and 60° (open bars) amplitude. Data on the left refer to unloaded movements, data on the right to movements made against an opposing load of 2.2 Nm. Each pair of bars represents the mean +1 SE of the group data. In both groups the duration of AG1 was longer during the 60° than during the 15° movements and during loaded than unloaded movements. From Berardelli *et al.* (1986), with the authors' permission.

the wrist. Movement velocity was reduced and the ability to modify the velocity in movements of different amplitude was also limited. Important information about the nature of bradykinesia in Parkinson's disease came during the second half of the 1970s from studies by Flowers (1975, 1976). He found that parkinsonian patients were able to perform rapid movements of small amplitude at almost normal speeds, but that they made larger movements more slowly than controls. Normal subjects were able to increase velocity when performing movements of increasing amplitude, but parkinsonian patients tended to execute movements of different amplitude with similar velocity. During the execution of rapid movements, parkinsonian patients depended more than normal subjects on ongoing visual information.

EMG studies have clarified some of the mechanisms underlying these findings. When patients with Parkinson's disease are requested to perform rapid movements, they activate agonist and antagonist muscles in the correct sequence and show normal antagonist inhibition (Hallett *et al.*, 1977). They also bring in appropriate anticipatory activity in the postural muscles (Dick *et al.*, 1986). Their movements are, however, slow and are accomplished with multiple cycles of EMG bursts of normal duration (Hallett *et al.*, 1977; Hallett and Khoshbin, 1980; Berardelli *et al.*, 1986) (Figs 4 and 5). These abnormalities affect proximal (Hallett *et al.*, 1977; Baroni *et al.*, 1984) as well as distal (Berardelli *et al.*, 1984*b*) muscles; they worsen during movements of large amplitude (Hallett and Khoshbin, 1980), and are improved by dopaminergic treatment (Baroni *et al.*, 1984; Berardelli *et al.*, 1986; Benecke *et al.*, 1987).

The presence of multiple EMG burst cycles led Hallett and Khoshbin (1980) to suggest that in Parkinson's disease the amount of EMG activation in AG1 is not adequate for the movements to be executed. The multiple EMG cycles presumably serve to compensate for the lack of initial AG1 activation. This phenomenon might be due to saturation of the mechanisms that generate the first EMG burst. If this is true, movements below saturation level might be executed normally or almost normally (*see* Flowers, 1975), whereas movements above this level might have a greater number of EMG cycles, because the amount of EMG activation in AG1 could not be further improved. This hypothesis has been tested measuring the change in both duration and amplitude of AG1 during rapid movements of different amplitude (Berardelli *et al.*, 1986). In normal and in parkinsonian subjects, the duration and amplitude of AG1 increases with the amplitude of movement (Figs 4 and 5), as it does during rapid movements executed with an opposing load (Fig. 5). Patients with Parkinson's disease can modulate the duration and 'size' of AG1 so the mechanisms that generate AG1 do not saturate. The important abnormality in Parkinson's disease appears to be an inability to scale AG1 activation to the movement parameters. Parkinson's disease patients consistently underestimate the size of the impulsive force generated by AG1 required to produce the desired movement.

Huntington's disease

Hefter *et al.* (1987) observed that the maximum frequency of rapid alternating finger movements was lower in Huntington's disease patients than in controls. Thompson *et al.* (1988) studied rapid wrist flexion movements of 15° and 60°. Some Huntington's disease patients were unable to execute the movements. In the remaining patients, the relationship between movement velocity and amplitude was abnormal and movements were abnormally slow. Movement slowness was worse in the akinetic-rigid form of Huntington's disease than in the patients with mild chorea. Only the patients with mild chorea had a normal EMG activation pattern; those with severe chorea or the akinetic-rigid form had an abnormal pattern. The duration of AG1 and ANT was prolonged. In the severest cases, AG1 was often poorly defined, varying from a single prolonged and dispersed burst of activity to repeated smaller bursts. The ANT also varied, either co-activating or starting before or after AG1 (Fig. 6). Finally, movement velocity and amplitude, and AG1 duration varied more (as measured by the coefficient of variation) in the patients than in normal subjects.

Bursts of EMG activity with prolonged duration have been described in a patient with Sydenham's chorea (Hallett and Kaufman, 1981).

The slowness of movement in Huntington's disease is similar to that of Parkinson's disease. However, the mechanisms in these two conditions are probably different. The prolongation of EMG bursts and the variable movement trajectories of choreic patients with Huntington's disease are more reminiscent of those described in dystonia and athetosis (*see* below), in contrast to the small and repetitive EMG bursts of Parkinson's disease.

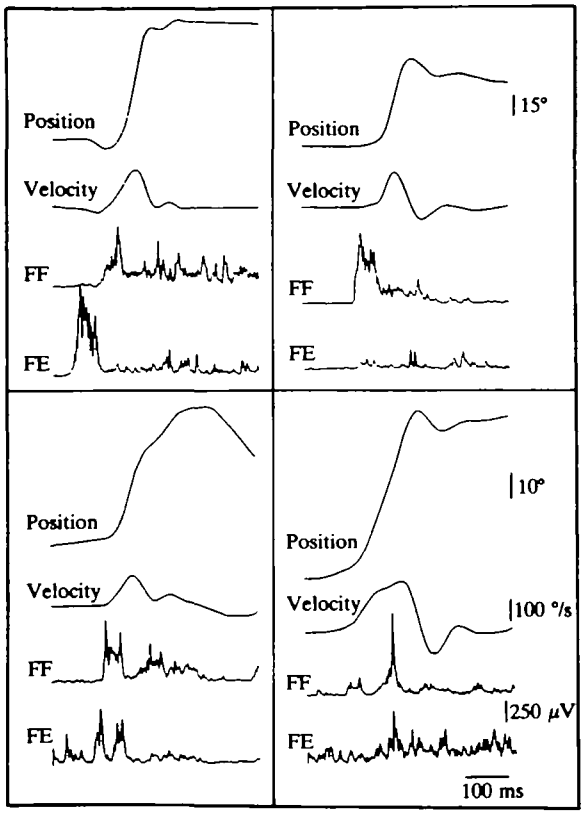

Fig. 6 Individual rapid 60° flexion movements at the wrist in a patient with Huntington's disease. The four traces in each panel are, from top to bottom, wrist position, angular velocity, and rectified EMG signals from forearm flexor (FF) and extensor (FE) muscles. Note the variability in movement trajectory and patterns of agonist and antagonist muscle activation during consecutive movements. Note also that the voluntary movement depicted in the upper left panel is preceeded by a choreic movement. From Thompson *et al.* (1988), with the authors' permission.

Dystonia and athetosis

Studies of rapid elbow flexion movements in patients with dystonia affecting the upper arm (van der Kamp *et al.*, 1989) have shown that movements are slower and more variable than those recorded in normal subjects. The AG1 lasted longer in these patients than in normal subjects. This was true even when normal subjects made slower movements to match the maximal velocities of the dystonic subjects. Many patients also exhibited co-contraction and a spread of activation into muscles not acting at the elbow joint. Despite these abnormalities, acceleration and deceleration times were approximately equal and the velocity profile symmetric, suggesting that this aspect of motor programming is intact in dystonic patients (Fig. 7). Finally, movement amplitude varied more (as measured by the coefficient of variation) in patients than in normal subjects even when the latter moved at a slow velocity.

Hallett and Alvarez (1983) found similar changes in patients with athetosis who also exhibited a triphasic EMG pattern with bursts of long duration, often with synchronous bursts in agonist and antagonist muscles.

The voluntary movements of patients with dystonia are very similar to those of patients with athetosis (Hallett and Alvarez, 1983) and patients with Huntington's disease (Thompson *et al.*, 1988), although there is lack of data on the velocity profiles in the latter conditions.

Upper motor neuron syndrome

In a patient with traumatic cerebral palsy (Angel, 1975) performing rapid abduction movements of the shoulder, AG1 had an abnormally long duration. Sahrmann and Norton (1977) observed the same abnormality during the execution of rapid alternating movements in patients with various spastic conditions. Analysing rapid flexion elbow movements in patients with amyotrophic lateral sclerosis, Hallett (1979) observed that patients had AG1 and ANT of longer duration than normal subjects. He proposed that the prolongation of the voluntary bursts could represent a compensatory mechanism due to reduction of motor neuronal recruitment in patients with pyramidal tract damage. Fagioli *et al.* (1988) also found prolonged burst durations in patients with an upper motor neuron syndrome following stroke. During movements of increasing amplitude, the size and duration of AG1 increased, as occurs in normal subjects. These authors suggested that the prolonged duration of the voluntary bursts might be due to the fact that a certain level of motor neuron recruitment specifies the duration of the EMG bursts. These two parameters could therefore vary only in parallel.

Patients with cerebellar deficits

Altenburger (1930) found that during rapid arm movements the antagonist burst 'was foreshortened or absent'. Terzuolo and Viviani (1974) studied rapid alternating movements and found that EMG activity in the contracting muscles frequently overlapped. Rapid flexion movements at the elbow were studied by Hallett *et al.* (1975*b*) in 20 patients with cerebellar deficits. Abnormalities were characterized by prolongation of AG1 and ANT or both and a reduction or absence of antagonist inhibition before the onset of AG1. Hallett *et al.* (1975*b*) proposed that these AG1 and ANT abnormalities might underlie the phenomenon of dysmetria; the abnormality of antagonist inhibition might be responsible for the dysdiodochokinesia and the rebound phenomenon. Similar observations have been made on thumb muscles (Marsden *et al.*, 1977).

More recently, Brown *et al.* (1990) have studied the kinematic aspects of step-tracking movements about the elbow in mildly affected patients. The movements were of different velocity, from slow to fast, and different distance (10–70°). The most important finding was that movements of medium and slow velocity and of 30–70° distance had an asymmetric velocity profile. The duration of acceleration was shorter and that of deceleration was longer than normal. The authors left open the question of whether this was due to abnormalities of the phasic voluntary bursts (not recorded)

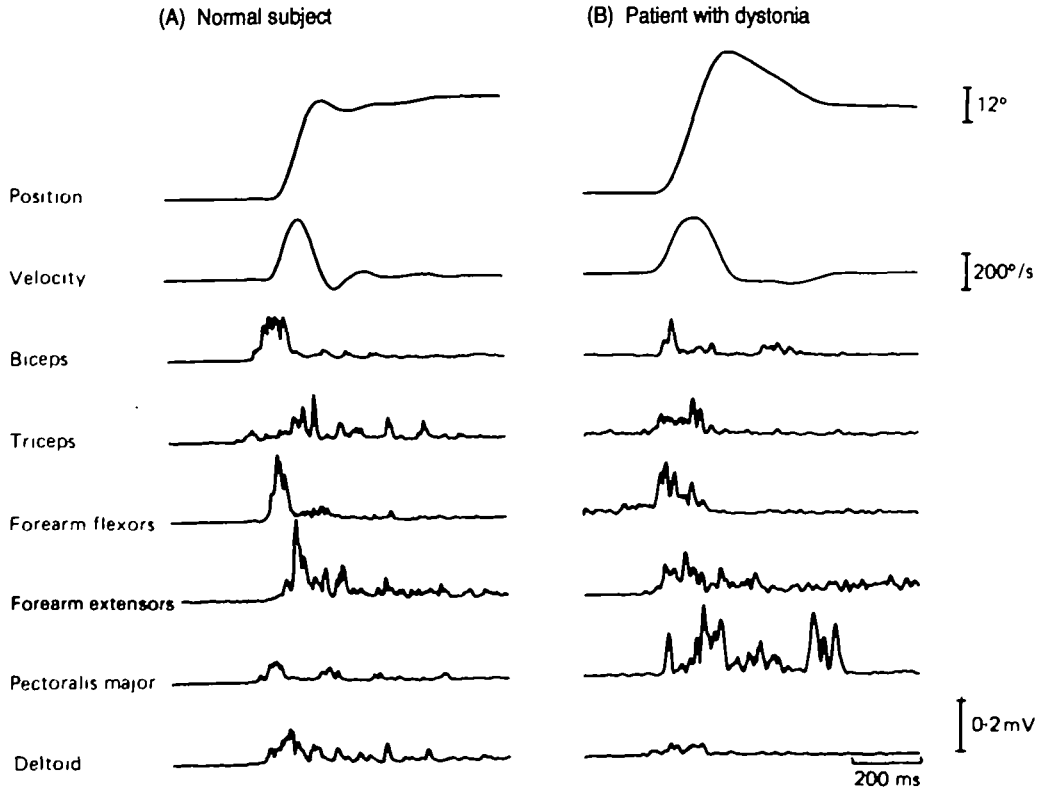

Fig. 7 Individual rapid 30 flexion movements at the elbow in a normal subject (**A**) and in a patient with dystonia (**B**). The eight traces in each panel are, from top to bottom, position, velocity, rectified EMG activity from biceps, triceps, forearm flexors, forearm extensors, pectoralis major and deltoid. Note that the duration of the agonist muscle (biceps) is prolonged in the patient with dystonia, and the timing of agonist and antagonist contractions is inappropriate for the movement. Note also the prolonged activation of pectoralis major (overflow) and co-contraction of the forearm and shoulder muscles. From van der Kamp *et al.* (1989), with the authors' permission.

or involved changes in movement strategy. The requirement for accuracy of these movements was probably responsible for the velocity profile that they observed.

Hallett *et al.* (1991) studied the execution of 10°, 20° and 30° rapid elbow flexion movements in 13 patients with cerebellar ataxia (Fig. 8). Movements were executed as fast as possible. The AG1 had a prolonged duration for the movements attempted even when compared with normal subjects making movements of similar distance and velocity. Moreover, this abnormality correlated positively with the severity of ataxia. Patients were more hypermetric and variable than normal subjects and the shortest movements displayed the greatest variability. Movements were performed slowly and acceleration time was prolonged. Cerebellar patients may, in some circumstances, choose to move more slowly than they are able to do in order to improve their accuracy. It seems clear, however, that even their best performance is slow. While occasional movements may be faster and are associated with shorter AG1s, this appears to represent one side of a variable distribution of performance. In addition, the prolongation of AG1 correlated positively with the prolongation of acceleration time. The authors suggested that prolongation of the acceleration time was the most important alteration and proposed two possible explanations for this finding: (i) an inability to turn off agonist activation; or (ii) a difficulty in implementing muscle force phasically.

Hore *et al.* (1991) studied six patients performing movements at three different joints (finger, wrist and elbow), with three distances (5°, 30° and 60°), under the instructions 'as fast possible'. All amplitudes and joints studied exhibited hypermetria, more evident for the shortest movements. A comparison of patients' movements and normal movements of similar peak velocity, showed a decreased peak acceleration and an increased peak deceleration. EMG recordings revealed an increased AG1 duration and a delayed ANT onset. In some patients the agonist activity developed more gradually than in normal subjects. The authors suggested that the cerebellum plays an important role in shaping the agonist command and contributes to the braking process of limb movements.

Essential tremor

In a study of patients with hereditary essential tremor (Britton *et al.*, 1994), with a postural and action tremor of the arm (Fig. 9), the peak velocity of movement was found to be normal or faster than normal, with the result that the patients

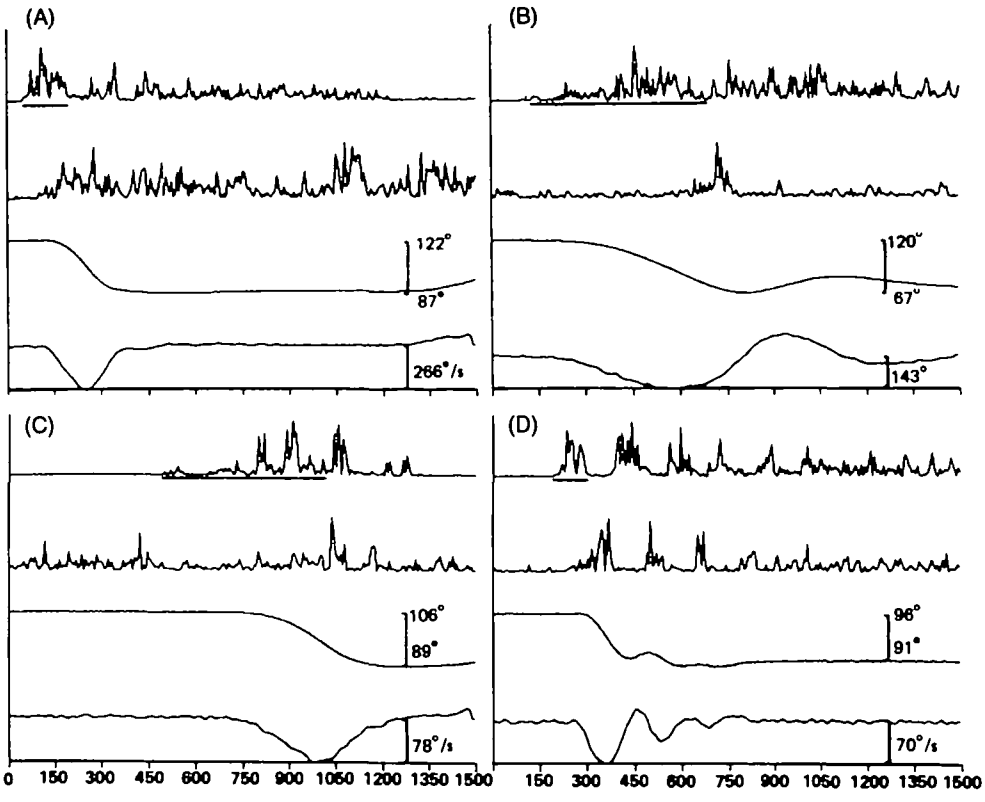

Fig. 8 Individual rapid flexion movements at the elbow in three patients with cerebellar deficits (**A** and **D**, same patient with moderate ataxia; **B** and **C**, two other patients with severe ataxia). The four traces in each panel are, from top to bottom, rectified biceps, and triceps EMG, angular position, and velocity. The line under the EMG of the biceps record indicates the measurement of the duration of AG1 in that record. The AG1 is mildly prolonged in **A** and markedly prolonged in **B** and **C**. In **C**, AG1 is fragmented. **D** shows the relatively rare event of a tremulous movement.

tended to overshoot the target. More important was the finding of an asymmetric velocity profile with higher peak deceleration. It was at this part of the movement that tremor was initiated. The EMG activity responsible for these ballistic movements showed a triphasic agonist/antagonist/agonist pattern but the onset of AG2 was delayed. The extent of delay correlated with the frequency of tremor. The delay in the onset of AG2 resulted in the unopposed antagonist contraction leading to an inappropriate and excessive deceleration. This activity interfered with the accuracy of the movement. Subsequent agonist bursts were attempts to correct this, with the end result of tremor. These findings are reminiscent of those in cerebellar disease discussed above. Indeed, there is good evidence linking a cerebellar abnormality to the production of tremor: (i) damage to one cerebellar hemisphere may abolish ipsilateral tremor (Dupuis *et al.*, 1989); (ii) abnormal increases in cerebellar blood flow are seen in patients with essential tremor (Colebatch *et al.*, 1990; Brooks *et al.*, 1992; Liao *et al.*, 1992); (iii) in a group of parkinsonian patients, the suppression of tremor by thalamic stimulation was accompanied by a decrease of cerebral blood flow in the cerebellum (Deiber *et al.*, 1993). It may be that AG2 and possibly other subsequent agonist and antagonist bursts are generated or timed by central mechanisms that involve the cerebellum.

Conclusions

The triphasic pattern characterizes rapid movement in proximal and distal joints of upper and lower extremities and even in the trunk (Friedli *et al.*, 1984). The pattern is present not only in highly skilled, intended movements, but also in automatic postural adjustments. Additionally, the timing of the triphasic pattern is strikingly similar in the different body parts. This remarkable generality must have important conclusions for motor control. The motor control programme for the triphasic pattern is one of the fundamental building blocks of the motor system, and helps to reduce redundancy in computing the biomechanical solution for specific motor tasks. Similarity of timing of the pattern in different body parts may well help in co-ordination of complex, multi-joint rapid movements.

How the triphasic pattern is generated in the CNS is not well understood. One possibility is that the cortical command does not contain the pattern itself, but is converted by pattern generators in subcortical and spinal structures. Another is that the pattern is developed completely in the cortex and delivered as such to the spinal cord. In favour of this latter view is the subtlety of detail of the pattern for different movements. The triphasic pattern has not been found in cortical recordings from performing primates, but it has not been looked for specifically either.

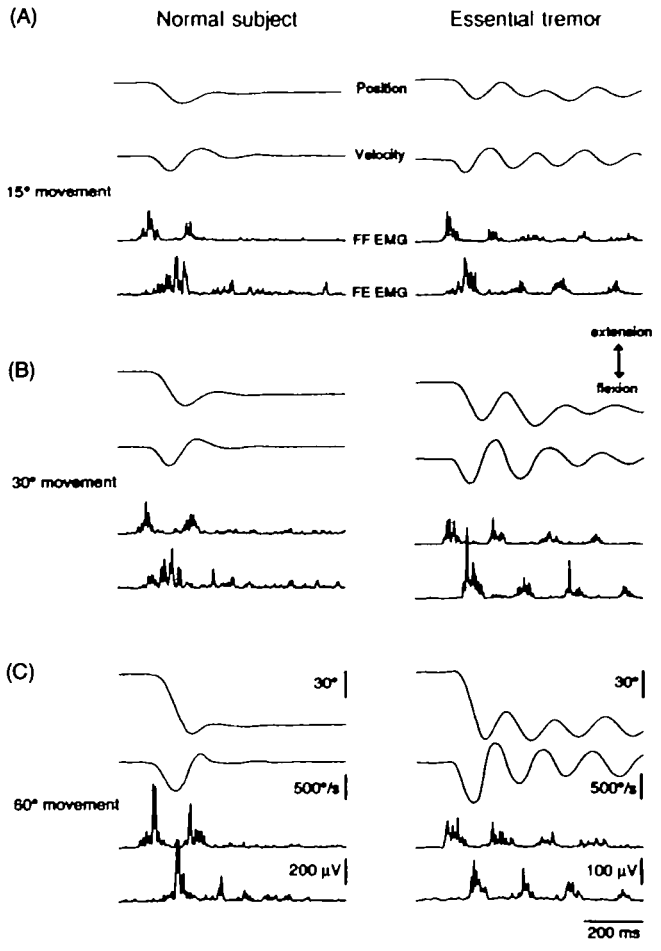

Fig. 9 Individual rapid flexion movements at the wrist of 15° (**A**), 30° (**B**) and 60° (**C**), in a normal subject (*left*) and in a patient with hereditary essential tremor (*right*). The movements performed by patients were slightly larger than normal but of similar duration and peak velocity and were accompanied by a triphasic pattern. However, patients showed difficulty in halting their movements, with further bursts of EMG activity producing oscillations of the wrist (tremor) about its end position. From Britton *et al.* (1994), with the authors' permission.

Abnormalities of triphasic pattern are typically represented by slowness of movements, irregularities of trajectory and loss of accuracy. The pathophysiology of each motor disturbance can be understood when both kinematic and EMG features are taken into consideration. The generality of the triphasic pattern means that even minor disturbances of the pattern may have a profound influence on motor behaviour. The derangements of the triphasic pattern summarized here for different movement disorders are highly relevant for understanding the pathophysiology of motor disorders.

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