

Ballistic elbow flexion movements in patients with amyotrophic lateral sclerosis

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SUMMARY Patients with amyotrophic lateral sclerosis made stereotyped 20° elbow flexion movements as rapidly as possible while surface EMG was recorded from biceps and triceps. The characteristic ballistic movement EMG pattern could be recognised in almost all the patients. The first agonist burst and the first antagonist burst, which are normally limited in duration, were prolonged in patients with clinical signs of pyramidal tract disease or alpha motor neurone disease or both. Prolongation of these components permits the muscles to generate sufficient forces to accomplish the movements.

Limb movements made as rapidly as possible are called ballistic. The initial part of the electromyographic (EMG) pattern underlying ballistic movements is characteristic (Wachholder and Altenburger, 1926; Hallett *et al.*, 1975a; Hallett and Marsden, 1978a,b). There is an initial burst of activity in the agonist (Ag 1), followed by a pause (Ag 1–Ag 2), followed by return of activity (Ag 2). At about the time of the pause of the agonist, there is a burst of activity in the antagonist (An 1). Studies of a 10° stereotyped flexion movement of the elbow in a group of normal subjects showed that the range of average durations of Ag 1, Ag 1–Ag 2, and An 1 was limited (Hallett *et al.*, 1975a). Preliminary studies of elbow flexion (Hallett, Shahani, and Young, unpublished), and careful studies of flexion of other joints (Freund and Büdingen, 1978; Hallett and Marsden, 1978b) show that these durations do not vary significantly if the angular distance of the movement or the initial angle of the joint varies. On the other hand, when attempting 10° stereotyped elbow flexion movements, patients with lesions of cerebellum or cerebellar pathways generated Ag 1 and/or An 1 bursts that were longer than normal (Hallett *et al.*, 1975b). Patients with Parkinson's disease showed

bursts of normal length in a similar task, but aspects of the patterning were abnormal (Hallett *et al.*, 1977).

The purpose of the present study was to see what changes there might be in the ballistic movement pattern in patients with "upper motor neurone lesions" or injury to the "pyramidal tract". The concept of pyramidal tract has been used loosely in neurology. The term should be restricted to those fibres originating in the cortex, traversing the medullary pyramid, and continuing in the spinal cord as the corticospinal tract. Above the level of the pyramid the fibres are intertwined with multiple other descending tracts, and lesions at these higher levels, therefore, usually give rise to deficits of multiple systems. The common hemiplegic stroke, for example, from internal carotid or middle cerebral artery occlusion gives rise to much more than injury to the pyramidal tract. Indeed, most patients who are said clinically to have the upper motor neurone syndrome do not have a pure pyramidal tract lesion.

Patients with amyotrophic lateral sclerosis were chosen for study. The pathological process in this disease appears to affect predominantly neurones of the pyramidal tract, defined strictly as above, and alpha motor neurones of the spinal cord and brainstem (Brownell *et al.*, 1970; Blackwood and Corsellis, 1976). Patients have a variable mixture of pyramidal tract injury, recognised clinically by "spasticity" (increased tone or tendon jerks or both), and alpha motor neurone injury, recognised clinically by muscle weakness and atrophy. An

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attempt was made to separate the physiological effects of these two pathological processes.

Methods

The 27 patients with amyotrophic lateral sclerosis were random patients admitted to the Peter Bent Brigham Hospital for a therapeutic trial of detoxified snake venom organised by Professor H. R. Tyler. It is of no consequence whether the patients were on snake venom or placebo since the venom has been shown to be ineffective (Tyler, 1978). The patients were divided into the following groups depending entirely on the examination of the right biceps and triceps: (1) normal, (2) spastic, with increased tendon reflexes or increased tone or both, (3) atrophic, with muscle weakness and atrophy, and (4) mixed, with clinical features of spasticity and atrophy. Four men and three women, ranging in age from 38 to 81 years, had normal examinations; six men and one woman, ranging in age from 32 to 69 years, were spastic; two men and one woman, ranging in age from 43 to 65 years, were atrophic; and three men and seven women, ranging in age from 35 to 72 years, were mixed. Five normal control subjects, all men ranging in age from 27 to 34 years, were also studied. The project was approved by the Peter Bent Brigham Hospital Human Subjects Committee.

Patients sat on a chair with their right arms resting on a table. The shoulder was abducted 70–90°, the elbow was at 90°, and the forearm was supinated. A large 20° angle was drawn on the surface of the table and patients initially placed their elbows at the vertex of the angle and directed their forearm along the side of the angle parallel to the edge of the table near where they were sitting. When given the verbal instruction, "go", the patients made rapid 20° elbow flexion movements moving their forearms to lie along the other side of the angle. Only well practised movements were studied, and the patients were continually urged to move as fast as possible.

The EMG was recorded from biceps and triceps muscles using pairs of surface electrodes placed longitudinally over the muscle bellies. The signals were amplified and recorded using a TECA TE4 electromyograph. The duration of EMG activity for each component of the ballistic movement pattern was measured manually from the paper records. Mean durations were calculated for each patient from five to ten individual movements.

Results

For all the normal control subjects and for 24 of

the 27 patients the characteristic pattern of agonist and antagonist muscle activity was easily recognised. Examples of EMG patterns from subjects in each group are shown in Figs. 1 to 4, and mean durations of Ag 1, An 1, and Ag 1–Ag 2 for each subject are plotted in Fig. 5. The duration of Ag 2 even for individual subjects, as has been noted before (Hallett *et al.*, 1975a), was quite variable and was not analysed.

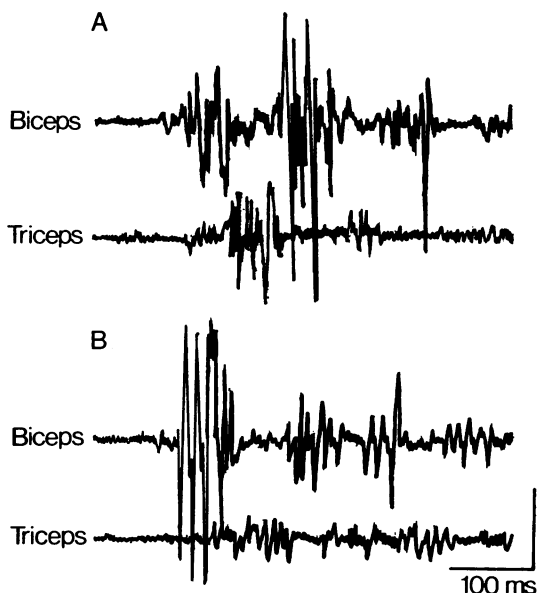


Fig. 1 Examples of the EMG pattern underlying rapid flexion movements in a normal subject (A) and an amyotrophic lateral sclerosis patient with a normal examination of the arm (B). Calibration bar for EMG amplitude represents 0.1 mV for A and 1.0 mV for B.

The normal control subjects had patterns (Fig. 1A) apparently identical to those seen in the 18 normal control subjects studied previously in a similar task (Hallett *et al.*, 1975a). The ranges of values from these earlier 18 subjects are indicated with bars in the normal columns in Fig. 5. The amyotrophic lateral sclerosis patients with clinically normal arms produced similar patterns (Fig. 1B). One amyotrophic lateral sclerosis-normal patient's results could not be analysed because the agonist did not silence well at the expected time of Ag 1–Ag 2.

Some patients in the spastic group produced patterns that appeared almost normal (Fig. 2A), while for others the durations of the components Ag 1 and An 1 were clearly prolonged (Fig. 2B). Interpretation and measurement of the com-

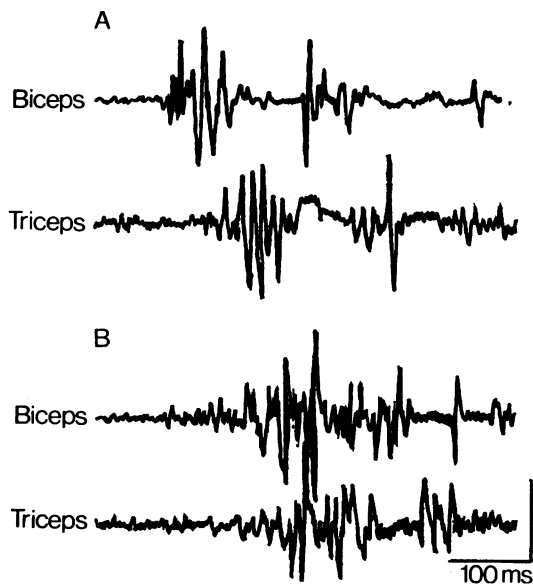


Fig. 2 Examples of the EMG pattern underlying rapid flexion movements in two patients with spasticity. The patients chosen for illustration had the longest and shortest average Ag1 durations in their group. Calibration bar for EMG amplitude represents 1.0 mV for A and 0.4 mV and 0.1 mV for biceps and triceps respectively for B.

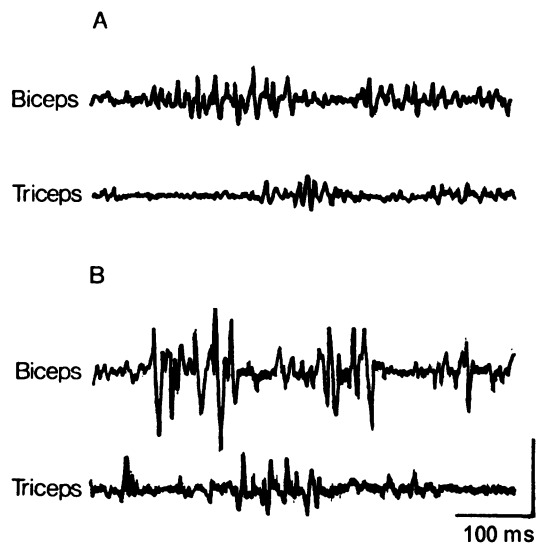


Fig. 3 Examples of the EMG pattern underlying rapid flexion movements in two patients with spasticity and atrophy. The patients chosen for illustration had the longest and shortest average Ag1 durations in their group. Calibration bar for EMG amplitude represents 1.0 mV for A and 0.4 mV for B.

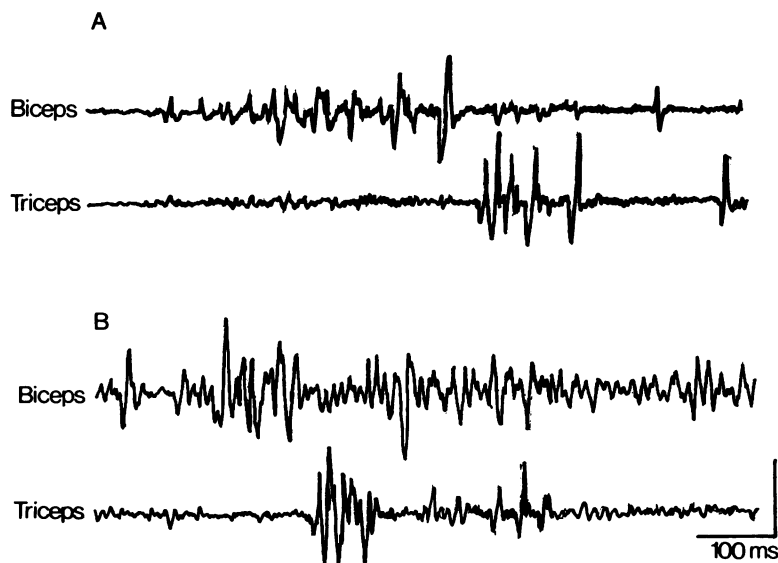


Fig. 4 Examples of the EMG pattern underlying rapid reflexion movements in two patients with atrophy. The patients chosen for illustration had the longest and shortest average Ag1 durations in their group. Calibration bar for EMG amplitude represents 1.0 mV for A and B.

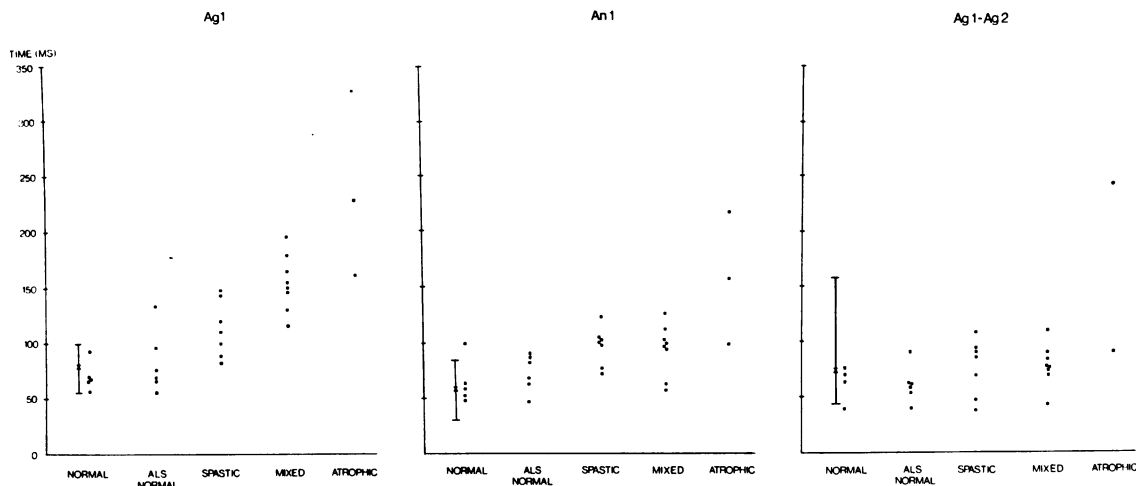


Fig. 5 Average durations of Ag 1, An 1, and Ag 1-Ag 2 for all the subjects separated by group. The range of values from the 18 normal subjects studied previously in a similar task (Hallett *et al.*, 1975a) is indicated with a bar in the normal column; the grand average duration is indicated by an X on the bar.

ponents in Fig 2B are difficult because of significant co-contraction of the triceps with Ag 1 of the biceps, and of the biceps with the An 1 of the triceps. Co-contraction activity, which can be seen in normal subjects, is usually variable in amount and was neglected in the analysis (see Hallett and Marsden, 1978a). The initial part of the Ag 1 burst in Fig. 2 B shows a gradual increase of EMG activity which contrasts with the rapid onset of maximal activity seen in normal subjects. This behaviour was characteristic only of some patients who showed the abnormally long Ag 1. Perhaps because it is difficult to quantify the amount of spasticity by usual clinical tests, there was no apparent relation between the durations of the components and the clinical degree of spasticity.

All the patients with spasticity and atrophy showed prolongation of Ag 1, and most showed prolongation of An 1 (Fig. 3). The durations of An 2-Ag 2 were within the normal range. Two patients in this group could not be analysed quantitatively because of ambiguity in defining the extent of the individual bursts.

The three patients with atrophy and no spasticity were all moderately to severely weak, but the basic ballistic movement pattern was still intact. The durations of Ag 1 and, to a lesser extent, An 1 were dramatically prolonged (Fig. 4). The duration of Ag 1 (and An 1) appeared to be related to the degree of weakness. Individual motor unit potentials can be recognised in biceps during Ag 1 in Fig. 4A, and this seems to be indicative of the

fact that only a few viable motor units remained in this muscle.

Discussion

The durations of the Ag 1 and An 1 bursts were similar for the normal subjects in this study making 20° movements and the normal subjects studied previously making 10° movements (Hallett *et al.*, 1975a). This is consistent with the emerging concept that the burst durations of the ballistic movement components do not change with movements of differing distance (Hallett and Marsden, 1977, 1978a,b; Freund and Büdigen, 1978). The patients with normal examinations of the arm had normal patterns, indicating that this test correlates with clinical impression. Patients with spasticity alone, presumably caused by pyramidal tract disease, showed mild but definite increases in duration of Ag 1 and An 1. Patients with muscular atrophy, presumably caused by lower motor neurone disease, showed marked increases in duration of Ag 1 and An 1. The extreme changes in these patients were probably a result of the fact that they were all moderately to severely affected and presumably less affected patients would show milder changes. Patients with spasticity and muscular atrophy had double cause for producing prolonged Ag 1 and An 1 bursts.

Prolongation of muscle bursts in rapid movements for patients with spasticity has been noted previously. Prolonged Ag 1 and An 1 were seen

in two patients with primary lateral sclerosis and one patient with a pure-motor-hemiparesis lacunar stroke in the 10° elbow flexion task (Hallett, Shahani, and Young, unpublished). Prolongation of the first agonist burst was seen in a rapid shoulder abduction movement in a patient with a traumatic cerebral injury (Angel, 1975). In rapid alternating movement tasks patients with spasticity of varying aetiology moved more slowly than normal, and the EMG burst durations when measured were prolonged (Neilson, 1972; Sahrman and Norton, 1977; McLellan *et al.*, 1978).

The mechanical task of Ag 1 is to provide force to accelerate the limb from one position to the next (Bouisset, 1973; Hallett and Marsden, 1977, 1978a,b). The task of An 1 is complicated, but one role, at least in certain circumstances, is to help stop the movement (Hallett and Marsden, 1978a,b; Lestienne, unpublished). Since, in normal circumstances, the durations of Ag 1 and An 1 are fixed, changes in force to generate movements of different length are accomplished by altering the amount of EMG activity in the fixed time frame (Hallett and Marsden, 1978a,b). Desmedt and Godaux (1977) have shown that this is possible by altering the number of active motor units and the frequency of their firing. If the number of available motor units is decreased or the firing frequency limited, it might not be possible to generate sufficient force in a fixed time period to make a desired movement. One method of possible compensation is to prolong the time period.

In lower motor neurone disease the number of functional motor units is decreased and the maximal voluntary force is reduced. Compensation for this deficit in ballistic movements does appear to occur by prolongation of the individual components of the ballistic movement pattern.

In upper motor neurone, or pyramidal tract disease the maximal firing frequency of motor units is limited. This fact is not well documented in the literature although most electromyographers recognise its validity (Rondot, 1977; Sahrman and Norton, 1977). Pyramidotomy in the monkey prolongs the "EMG summation time" required to make a rapid movement (Hepp-Reymond *et al.*, 1974). This reduction in firing frequency is one probable reason for the weakness caused by pyramidal tract lesions. In ballistic movements compensation for this deficit appears to occur by prolongation of the ballistic movement components.

Prolongation of Ag 1 and An 1 is characteristic of the ballistic movement pattern of patients with cerebellar deficits (Hallett *et al.*, 1975b) as well as patients with lower motor neurone and pyramidal

tract lesions. We have speculated that the problem in cerebellar malfunction is that the EMG activity in a burst is not truncated appropriately (and thus movements tend to be hypermetric). In the electrophysiological analysis of individual patients, to attempt to explain prolongation of the ballistic movement components on the basis of cerebellar deficit we must show that there is no denervation and that the interference pattern on maximal effort is full.

Ballistic movement component durations are similar for biceps and triceps (Hallett *et al.*, 1975a) and for the long flexor and extensor of the thumb (Hallett and Marsden, 1978b). Freund and Büdingen (1978) have shown similar contraction times for several forearm and hand muscles. These authors have speculated that this type of control is useful for the "synchrony of synergistic muscle contractions." Should this be the case, the abnormality of prolonged burst duration might explain some of the clumsiness in rapid movements seen in patients with spastic or denervated muscles.

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