Pathological stretch reflexes on the "good" side of hemiparetic patients

A F Thilmann, S J Fellows, E Garms

Abstract
The reflex EMG responses from a tendon tap or an imposed, medium amplitude (30°) stretch at a range of stretch velocities have been recorded from the triceps and biceps muscles of normal human subjects and in both the affected and "unaffected" arms of hemiparetic patients under relaxed conditions. In the hemiparetic arm, exaggerated tendon jerks were, as expected, observed in both muscles. The response of the biceps to elbow extension was also exaggerated compared with normal values and displayed both an additional, earlier component and a much reduced velocity threshold. The triceps, in contrast, showed depressed responses to elbow flexion, with a much higher velocity threshold than normal subjects. Furthermore, on the supposedly "unaffected" side of the hemiparetic subjects, the reciprocal pattern was seen, with depression of the biceps response and a raising of its threshold, along with considerably exaggerated responses in the triceps including earlier components not seen in the normal subjects. The increased excitability of the flexor musculature on the spastic side may be paralleled by increases in activity in the segmental pathways responsible for modulation of agonist/antagonist activity in the ipsi and contralateral limb, leading to an inhibition of the ipsilateral extensors and contralateral flexors and excitatory input to the contralateral extensors. Thus the "good" side of hemiparetic patients also receives pathological changes, and studies of the mechanisms of spasticity should avoid the use of the "unaffected" side of hemiparetic subjects as a control for monitoring pathological reflexes.

Spasticity is a motor disorder which occurs after CNS lesions affecting the pyramidal tract at a variety of locations. Clinically it is defined by exaggerated tendon jerk reflexes, increased muscle tone (velocity-dependent increases in the resistance to movement), muscle weakness and, in some cases, clonus. If the cause of the spasticity is an ischaemic lesion in the vicinity of a middle cerebral artery, the disability is visible clinically only on the contralateral side of the body. Given the overwhelmingly one-sided nature of this deficit, it is perhaps not surprising that little study has been made of the "unaffected" side of hemiparetic patients. Indeed, it has become common practice to use the "good" side as a fully matched control for the description of changes on the affected side. This study concerns the responses to mechanical disturbance of antagonist muscle groups at the elbow on both sides of hemiparetic subjects and the comparison of these responses to those of a group of normal subjects. The results indicate that the so-called "unaffected" arm of the hemiparetic subjects does not respond with the normal pattern, and that pathophysiological changes have occurred on both sides of hemiparetic subjects.

Methods
Experiments were performed on a group of ten hemiparetic patients (for clinical details, see table 1) and a group of ten age-matched normal subjects, all of whom gave their informed consent to the procedures employed. All procedures were approved by the local ethical committee. The subjects were seated side on (see fig 1a) in an adjustable chair and the arm under study was placed in a moulded plastic cast (c) which restrained, by means of a series of straps, the forearm and hand. The cast was in turn attached to an aluminium support mounted on a bearing (b), its position being adjusted so that the elbow joint lay directly over the axis of rotation. The height of the chair was adjusted to bring the elbow and shoulder joint to the same level, with the subject's back and shoulder supported by adjustable rests. Displacements were applied by a powerful electric motor (Servalco, MC24P(3000W)) with 4-quadrant servo control system (Seidel, series 5000), via a rigid connecting rod (r) attached to the cast at the level of the wrist. The motor was strong enough to impose fully stereotyped movements, irrespective of arm inertia or resistance generated in the subject's arm. Thus, for a given set of motor parameters, an identical displacement was applied to all subjects. Displacement amplitude was 30°. Extending stretches moved the arm from 75° flexion to 105° flexion, flexing stretches from 105° to 75°, 90° flexion corresponding to a right angle at the elbow joint. Displacement velocities up to 262°/s were applied, under the control of a laboratory computer (DEC, PDP11/73). Position and velocity curves for the extending stretches are shown in fig 1b. All velocity curves were calculated by the computer direc-
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Table 1  Clinical details of hemiparetic subjects

<table>
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<tr>
<th>Case no</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Localisation of ischaemia</th>
<th>Imaging method</th>
<th>Duration of spasticity</th>
<th>Paresis</th>
<th>Tone</th>
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<td>m</td>
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<td>l.mca</td>
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<td>5 months</td>
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<td>3</td>
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<td>CT</td>
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<td>2</td>
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<td>r.mca</td>
<td>CT</td>
<td>9 months</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

1 l.mca = left middle cerebral artery. r.mca = right middle cerebral artery.
2 Imaging method(s) used to establish lesion localisation: CT = computed tomography; MRI = magnetic resonance imaging; A = angiography.
3 Paresis of the hemiparetic arm, expressed according to the British MRC scale: 0 = no voluntary power; 1 = visible contraction without effort; 2 = movement without the influence of gravity; 3 = movement against gravity; 4 = movement against resistance; 5 = normal.
4 Tone of the hemiparetic arm, expressed on the Ashworth scale: 0 = normal; 1 = slight increase in tone, giving a "catch" when the limb is moved; 2 = more marked increase in tone, but limb easily flexed; 3 = considerable increase in tone-passive movement difficult; 4 = limb rigid in flexion or extension.

The mechanical construction of the equipment, the same motor pulses produced slightly higher peak velocities during extending stretches than during flexing stretches. Ten stretches were applied at each velocity, at random intervals between 5 and 8 seconds. The subjects were instructed to remain fully relaxed and not to react to the displacement.

Displacement was monitored using a light sensitive detector (United Detector Technology, LSC5D), which followed the movements of a light source rigidly attached to the drive shaft. This detector yielded a signal which, after suitable processing, could resolve movements of <0.06° at the elbow, with no detectable phase shift. Electromyograms (EMG's) were recorded from the biceps and triceps muscles using custom built surface electrodes which contained a small pre-amplifier and were designed to reduce movement artifacts to a minimum. The signals obtained were amplified and filtered (bandwidth 20Hz to 1KHz), then passed, along with the position signal, to the ADC boards of the computer, which sampled at a rate of 1KHz for each channel. Both EMG's were then rectified and averages constructed from the ten displacements applied at each stretch velocity. Position, raw EMG data and the motor control pulses were also recorded on tape (Racal, Store7D).

At the start of each experimental series, tendon jerk reflexes were obtained from the biceps and triceps muscles, using a reflex hammer containing a microswitch which triggered computer sampling. Five responses were obtained from each muscle and peak amplitude, onset latency and duration subsequently determined.

For the displacement experiments, each averaged record was analysed offline to yield the peak EMG amplitude, onset latency and duration of the responses. Peak EMG amplitude was chosen as a measure of response magnitude after trial measurements showed that this value was strongly correlated to the integrated area of the EMG response. Onset latencies of the responses were measured from the onset of movement, not from the control pulse to the motor, which preceded movement onset by 10 ms. The statistical significance of differences between the groups was assessed by variance analysis.

Figure 1  (A)
Displacement apparatus. For explanation of symbols, see text.

(B) Position (lower) and velocity (upper) curves for the five rates of extending displacement applied.
Results

Elbow Extension

In the normal subjects, under relaxed conditions, elbow extension at velocities greater than 200°/s resulted in a burst of activity in the biceps EMG in eight out of 10 cases. In the remaining two subjects no response was obtained, even with the maximum rate of stretch applied (262°/s). Figure 2a shows the responses of a single subject over a range of stretch velocities. The response was generally small (average 38 µV, SE13). No correlation existed between its magnitude and the magnitude of a particular subject’s biceps tendon tap response. The response occurred with a latency of between 40 and 70 ms (average 56 ms, SE3) and lasted for between 25 and 45 ms (average 30 ms, SE3). As the stretch velocity was lowered, the response disappeared, usually when stretch velocity fell below 200°/s. In the subject illustrated, a small response remained at 172°/s, but in no case was a response seen if stretch velocity was slower than 165°/s.

On the affected side of the hemiparetic patients, as would be expected from clinical experience, the tendon tap reflex was exaggerated in both the elbow extensor and flexor muscles, in the latter to a significantly higher level (p < 0.001) than that seen in the normal subjects. In addition, it was apparent that the response to rapid elbow extension was also exaggerated. In all of the patients studied, the EMG of both the elbow flexors and extensors showed no sign of background activity at the onset of the displacements and no subject who exhibited contractures was selected for this study. Figure 2b shows the response in the biceps EMG of a typical patient to the same range of stretch velocities applied to the normal subject in fig 2a. The shaded lines represent the standard error bars of the group average for onset latency and duration of the normal subjects. It can be seen that the response is not only larger, but much more complex in form: while a large part of the response (average amplitude at 262°/s: 82 µV, SE22) occurs within the time period of the normal response, activity is apparent both before and after this period. The late EMG activity, occurring after 90–100 ms, cannot be assumed to be purely reflex in nature, and its origin and significance will be considered in a later paper. The earlier activity (average amplitude at 262°/s: 60 µV, SE15), appearing 25–35 ms (average 27 ms, SE1) after the onset of movement, was not observed in a normal subject, but was seen in the biceps EMG of the affected side in seven out of 10 of the hemiparetic subjects. As the stretch velocity was lowered, the medium latency activity, as in normals, quickly disappeared, never being observed with stretch velocities below 200°/s. The early activity, however, persisted and appeared following stretches as slow as 117°/s, well below the threshold of the most sensitive of the normal subjects (165°/s).

A similar range of displacement was applied to the “unaffected” arm of these hemiparetic patients. Somewhat surprisingly, in all but two of the subjects, no response to extending stretch was seen. Similarly, the biceps tendon tap reflex was significantly smaller (p < 0.001) than that of the normal subjects. Figure 3 shows the average EMG amplitude following biceps stretch plotted against stretch velocity for the normal subject (n) and for the spastic (s) and “good” (g) arm of the hemiparetic, with the corresponding tendon jerks. For the affected side of the hemiparetic, the early (s1) and medium (sII) latency activity is shown separately. It can be seen that while the affected arm of the spastic subjects responds more markedly (p < 0.01) and at lower velocities of the stretch than the normal arm, the so-called “unaffected” side shows significantly less
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activity \( (p < 0.001) \) than the affected side, but also less than the normal subjects \( (p < 0.005) \), indicating that changes from the normal state have also occurred in this arm.

**Elbow flexion**

In the normal subjects, elbow flexion produced more considerably EMG responses in the triceps than elbow extension did in the biceps. Only one subject showed no response to stretch and the responses persisted at lower stretch velocities. Figure 4a shows the triceps EMG responses of a representative normal subject over a range of stretch velocities. At the most rapid rate of stretch, a burst of activity can be seen (average amplitude 89 \( \mu \)V; SE27), appearing with an average latency of 58 ms (SE6) and average duration of 36 ms (SE3). As stretch velocity was reduced, this component gradually reduced in both amplitude and duration, but was not abolished until stretch velocity fell below 100°/s.

On the affected side of the hemiparetic subjects, stretch of the triceps, even at the most rapid stretch velocities applied, produced a reflex response in the triceps EMG in only 50% of the cases. In contrast, the tendon jerks tended to be exaggerated over normal values, although not significantly (fig 5). In those subjects who did show a response to imposed displacement, it was significantly smaller \( (p < 0.025) \) than in the normal responses (average amplitude at 252°/s: 23 \( \mu \)V; SE12), but occurred with similar latency and duration. The threshold of this response, however, was considerably higher than normal values, with no responses being seen for stretch velocities below 150°/s and only one subject showing a response at velocities of stretch lower than 200°/s.

On the “unaffected” side of the hemiparetic subjects, the pattern of response was, as for the biceps, far from the norm. Figure 4b shows the responses of a typical “good” side at a range of
stretch velocities. As in fig 2, the shaded lines represent the standard error of the group average for onset latency and duration of the normal subjects. It shows that the dominant part of the response occurs within this time period. Magnitude and duration of this component do not differ from normal values. In addition, however, it may be seen that shorter latency activity is present (average latency at 252°/s: 29 ms SE1; duration: 27 ms SE1). Such activity was present on the “unaffected” side in half the hemiparetic subjects, but unlike the situation in the flexor musculature on the affected side, it was never accompanied by late, tonic activity. Figure 5 shows the triceps EMG amplitudes of the spastic (s) and “good” (g) sides of the hemiparetic subjects and those of the normal subjects (n). As can be seen, the threshold of the activity on the “good” side did not differ significantly from that of the normal response. The threshold of the response on the affected side, however, was considerably raised and its amplitude significantly smaller than in the normal subjects (p < 0.025) and on the unaffected side of the patients (p < 0.005).

If figs 3 and 5 are compared, a clear picture of interaction between the two sides of the hemiparetic subjects becomes apparent: on the spastic side, the magnitude of the flexor response is increased relative to normal values and its threshold lowered, while the response of the extensor musculature is depressed and its threshold raised. On the “unaffected” side of the spastics, the reciprocal pattern is seen, with depression of the flexor and increase in the extensor response.

**Discussion**

On the affected side of the hemiparetic subjects, as would be expected from typical descriptions of spasticity, the response of the biceps to stretch was strongly exaggerated. This EMG activity was also more complex in form, resembling the modulated response that would, in normal subjects, be obtained only from the stretch of a voluntarily activated muscle. In all cases, however, the spastic subjects were relaxed at the time of displacement, with their muscles electrically silent. Furthermore, this pattern was also obtained in subjects who had no voluntary power at the arm, suggesting that the pattern does not depend on descending voluntary drive. The occurrence of this more complex pattern in the relaxed arm seems to represent a difference from the situation at the hemiparetic ankle, where it was uncommon to find more than a single component in the relaxed state.

Nevertheless, to discuss the pattern observed in this study in terms of the M1/M2 pattern (described solely for voluntary activated muscle) and the changes that may occur in it due to supraspinal lesions would seem unlikely to clarify our findings, given the many differences between the motor system in the relaxed and the voluntarily activated state. A breakdown in the phasic nature of the agonist burst in a rapid voluntary elbow flexion movement, leading to a prolongation of the burst has recently been reported in hemiparetic subjects and it could be argued that the current changes are more analogous to this loss of modulation than the incidence of new reflex components. An objection to this argument is that the EMG response on the hemiparetic side is not only prolonged, but contains a component occurring with a considerably shorter latency. This would suggest that, somewhere in the reflex pathway, the threshold at which a component begins to respond has fallen and/or the magnitude of its response considerably increased. Given that no evidence has yet been found for increased sensitivity of muscle spindles in spasticity, it would seem more likely that these changes have occurred centrally, probably at a spinal inter-
neuronal level. This idea is supported by the differences in the behaviour of the tendon jerk responses and the response to imposed displacement in the hemiparetic arm: while the tendon jerks of both biceps and triceps are exaggerated, the response to displacement is enhanced in the biceps but strongly depressed in the triceps. As the tendon jerk is known to be mediated predominantly (but not exclusively) over a monosynaptic pathway, it seems clear that the modulation of the responses to imposed stretch, which is not reflected in a similar modulation of the tendon jerks, must be occurring at a higher level than the motor neuron pool. More precise location of the site of this action is not possible from the present results. One conclusion to be drawn from the differing behaviour of the tendon jerk and the responses to imposed stretch is that the tendon jerk response does not reveal the full complexity of the changes in the reflex system in spasticity and may not provide a true reflection of the likely magnitude of the reflex response to a more physiological stimulus.

In the triceps of the affected arm, in contrast to the enhanced responses seen in the biceps, the reflex response to imposed displacement is both smaller to that of the normal subjects and displays a much higher velocity threshold. Such a modulation of excitability in an antagonist muscle group in spasticity is well known from studies of the lower limb, where the relative weakness of the pretibial muscles and hyperactivity of the ankle extensors is an important contributing factor to the impairment of gait in spasticity. This imbalance has been attributed, in the relaxed state, to increased reciprocal inhibition of the pretibial muscles by the ankle extensors. It is reasonable to assume that a similar mechanism is in operation in the muscles of the upper arm, given the general principle that an increase in excitation in a muscle group is accompanied by parallel changes in the activity of its associated segmental pathways, leading to increasing activity in excitatory pathways to synergists and in inhibitory pathways to antagonists.

The appearance of the reverse pattern of changes on the supposedly “unaffected” side of the hemiparetic subjects has, to our knowledge, previously been reported. All of the subjects studied were selected because their lesions were clearly localised by CT scanning and additionally, in most cases, by NMR techniques and angiography and in all cases the symptoms of spasticity were one-sided. Nevertheless, a marked depression of responses in the biceps and exaggerated responses in the triceps was seen on the “unaffected” side of all of the hemiparetic subjects. As this pattern was clearly reciprocal in the hemiparetic arm, it is tempting to attribute these changes to inter-limb innervation at a spinal level, specifically over pathways which normally deal with inter-limb coordination in complex movement patterns, with the increased excitability of the affected biceps leading to inhibition of the contralateral flexors and excitation of the extensors. Our results do not, however, allow the exclusion of other mechanisms, including interactions at supraspinal levels. Such alternatives cannot be excluded, as it is known that, while the greater arc of abstraction fibres cross to the contralateral side, around 10% remain on the ipsilateral side of the supraspinal lesion, and may thus directly influence the “unaffected” side. Brodal, has suggested that disturbances of fine motor control on the “unaffected” side of a hemiparetic subject may arise from disruption of cortico-cerebellar pathways with bilateral actions on motor neurons: again, a possible contribution from this source cannot be excluded.

The changes in the supposedly “good” side of a hemiparetic subject are of significance for neurophysiological studies of spasticity, which have, in the past, commonly utilised the “unaffected” side of hemiparetic subjects as a convenient, matched control for the description of the pathophysiological changes that occur in the upper motor neuron syndrome. The present findings indicate strongly that this practice should be discontinued. The role of these changes in the disability of the patients has still to be clarified. The patients themselves reported no problems on their “unaffected” side, but given the severity of the disability on the hemiparetic side, such judgements of normality should be viewed as subjective. It seems desirable to investigate the extent of reflex dysfunction on the “unaffected” side, to establish the extent to which “hemiparesis” is merely a term for naming the most severely affected side following a localised supraspinal insult.

A final minor point to be considered is that, in the relaxed state, the triceps muscle of the normal subjects responded more markedly, and at lower velocities of stretch, than did the biceps. This is, at first sight, somewhat surprising: from the principle of physiological excitability, as proposed by Sherrington, it is to be expected that the biceps, which is the major anti-gravity muscle at this joint, would show the most marked reflex responses. If the amplitude of the reflex responses was the sole basis for comparison, it could be argued that this discrepancy could be attributed to the use of surface electrodes for EMG recording. The triceps, however, also responds at much lower stretch velocities, suggesting that this difference in reflex excitability cannot be accounted for entirely on these grounds. A possible explanation could lie in the different morphology of these muscles: the biceps has proportionally, much longer tendons than the triceps, which will tend, in the relaxed state, to reduce the transmission of the imposed stretch to the muscle receptors, and hence the magnitude of the reflex response. Thus if, due to its longer tendon, more of the early part of the movement was lost in taking up tendon slack, the biceps muscle spindles might be exposed to lower acceleration rates than those of the triceps, despite the attainment of the same peak stretch velocity in the two movements. The presence of such tendon compliance might also explain the relatively long latencies of the responses, although it would be expected from work on the relaxed triceps.
surae,17 that a greater difference in latency between the biceps and triceps response would have been observed.

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