Motor units in incomplete spinal cord injury: electrical activity, contractile properties and the effects of biofeedback

R B Stein, B S Brucker, D R Ayyar

Abstract
The electrical and contractile properties of hand muscles in a selected population of quadriplegic subjects were studied intensively before and after EMG biofeedback. Spontaneously active motor units and units that could only be slowly and weakly activated were observed in these subjects, in addition to units that were voluntarily activated normally. This suggests a considerable overlap of surviving motor neurons to a single muscle that are below, near or above the level of a lesion. Despite the common occurrence of polyphasic potentials and other signs of neuromuscular reinnervation, the average twitch tension of single motor units in hand muscles of quadriplegic subjects was not significantly different from that in control subjects. Nor did it increase after biofeedback training that typically increased the peak surface EMG by a factor of 2–5 times. The percentage of spontaneously active units was also constant. The surface EMG may be increased during biofeedback by using higher firing rates in motor units that can already be activated, rather than by recruiting previously unavailable motor units.

Recording from single motor units gives a unique window into the integration of inputs to human motor neurons in a variety of states. Although there have been a few studies of motor units following spinal cord injury, motor unit recordings in chronic, spinal cord injured patients (a year or more after injury) is rarely done clinically, perhaps because of the widely held view that the situation has stabilised and little prospect for improved function remains. However, treatment using biofeedback, based on surface EMG recordings from major muscle groups, combined with physiotherapy and electrical stimulation may improve function substantially in some patients. The efficacy of such treatment remains controversial and the neuronal mechanisms underlying any increase in the surface EMG signals through biofeedback are obscure.

The purpose of this study was to examine the electrical and contractile properties of single motor units in detail, using techniques developed over the last fifteen years. For this study subjects were selected who had incomplete spinal cord injuries and showed some activity in hand muscles, although hand function remained grossly defective more than a year after injury. The subjects selected had responded well to therapy including biofeedback of the larger muscle groups of the arm, but had not received any biofeedback to the hand muscles. They were studied before and after intensive biofeedback therapy to assess the nature of the functional deficits and possible neuronal mechanisms underlying any improvements shown.

Methods
Three hundred motor units were studied from 18 muscles of six adult, quadriplegic subjects (three male and three female; time since injury one to seven years; level of injury C4–C6). The subjects gave their written informed consent to participate in the study. The needles used for the quadriplegic subjects and the normal controls consisted of two teflon-coated stainless steel wires (diameter = 75 μm) inserted into a 25 gauge needle. The wires were fixed in place with epoxy, and ground flush with the barrel of the needle. Needles were gas sterilised before use. This design has proved to be a good compromise between the selectivity needed for single unit recording at medium to high force levels and the stability required to hold a unit for several minutes of voluntary contraction during spike-triggered averaging. Recordings were made bilaterally from thenar and first dorsal interosseous (FDI) muscles (about 10 units from each muscle studied) in which there was evidence of some voluntary electrical activity from surface EMG recordings.

Surface electrodes (Beckman type Ag/AgCl, 8 mm recording diameter) were placed over the muscles of interest on either side of the site where the needle was inserted. Recordings from these electrodes were used to check for synchronisation between motor units (see below) and to estimate the contribution of the single unit recorded by the needle to the surface EMG. Isometric force was recorded by means of a Grass FT03 strain gauge. The methods for recording force from the FDI muscle and the thenar muscles have been described previously.

The action potentials from a single motor unit were amplified (Leaf Electronics Model QT5B, Edmonton, Canada), highpass filtered using a 100 Hz RC filter and then discriminated using a Bak Electronics (Rockville, MD) Model DI-5-1 Window Discriminator. Pulses from the discriminator were used to trigger
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signal averaging using a Cadwell Laboratories (Kennewick WA) Model 7400.

Four signals were averaged as shown in fig 1: 1) the motor unit action potential after a time delay of 5–10 ms (Bak Electronics Model AD-6 Analog Delay); 2) the surface recordings after a 100 Hz high-pass filter and time delay of 5–10 ms; 3) the same surface signals after additionally being full-wave rectified and 4) the force signals after being band-pass filtered over a range of 0.16–100 Hz. The averaging of the surface signals, both rectified and unrectified, permitted a test of motor unit synchronisation to be used. The use of AC filtering of the force records with a long time constant (1s) permitted the force fluctuations associated with the unit action potentials to be determined. If synchronisation is absent, this permits the motor unit twitch tension to be estimated.

Biofeedback EMG measurements were obtained with gel-filled Beckman electrodes similar to those described above. Two active electrodes were placed over the belly of the muscle of interest (thenar or FDI) and a third ground electrode was placed on the distal end of the forearm. A prototype NeuroEducator manufactured by Therapeutic Technologies (Dayton, Ohio) was used to analyse and provide feedback of the EMG signal. The NeuroEducator measures the root mean square (RMS) voltage with an internal noise level of less than 0.2 μV with a bandwidth of 10–10,000 Hz, common mode rejection better than 140dB. EMG signals were integrated over 0.1s, calibrated in mV and displayed on a colour monitor in the form of a continuous line updated every 0.1s for a sweep of 20s duration.

Subjects sat 1–2 m in front of the monitor and were instructed that the line on the screen represented the amount of signal recorded from the muscle being studied. They were to make the line go as high as possible when told to do so and to stop when told. After the first try a cursor was placed on the monitor at the highest recorded level of EMG and verbal reinforcement was given for EMG responses that exceeded the maximum level, after which the cursor was set at the new maximum. Training sessions lasted 45 minutes to an hour, during which 5–10 training trials for the thenar and FDI were conducted. Each trial consisted of approximately a 10s attempt to increase the EMG signal, preceded and followed by a five second rest. Inter-trial intervals were 2–4 minutes. Data were stored on disc and a hard copy of average EMG responses every 0.5s was produced for further data analysis.

Results

As the patients who were selected for detailed study had continuing difficulty with hand function, it was not surprising that the number of motor units encountered at any needle location was grossly reduced and not uniform. Activity could be absent in large regions of one muscle, for example, first dorsal interosseous (FDI), but present in an adjacent muscle, adductor pollicis, innervated by the same nerve from approximately the same spinal roots. The number of active motor units could also vary enormously between different portions of the same muscle (for example, deep and superficial compartments).

Active units could be divided into three classes: 1) those under approximately normal control with a clear threshold for activation and a range of firing rates comparable to those in control subjects; 2) spontaneously active units that could not be activated by the subject. These tended to fire at low rates (<5 impulses/s), but the rate could often be increased by muscle stretch and/or by spasms which involved the hand muscles. Overall, 20% of the units studied in these quadriplegic subjects were spontaneously active, but the numbers varied from 0–50% in different muscles and different subjects; 3) units that could only be slowly and weakly activated. When a subject was asked to turn off a unit, it might take 5–10s for the unit to stop firing. These units comprised less than 5% of the units overall, but could represent up to 20% of the units in some muscles.

A similar, long latency was often required to reactivate the unit, and unusual methods might be used. In an exteme example, a subject noticed that when she tried to move her big toe, her thumb also moved and this proved the best way for her to activate some units in her thenar muscles. When activated, these thenar units tended to fire at low initial rates similar to those of the spontaneously active units, but with effort the rate might

![Figure 1](http://jnnp.bmj.com/)

**Figure 1** Action potentials from a single motor unit recorded with a needle electrode (top trace) were used to trigger an averaging program which averaged the needle EMG, the surface EMG before (trace 2) and after (trace 3) rectification and the force fluctuations in the muscle (trace 4). The top three traces were delayed 5 ms. The bottom trace is on a slower time scale and provides a measure of the twitch tension of the motor unit, assuming that the discharges of different motor units are independent (no synchronisation). The peak-to-peak amplitudes are 1.38 mV (needle EMG) and 0.10 mV (surface EMG) and the twitch tension is 66 mN. The absence of synchronisation can be checked using the surface EMG, as described in the text. Records from the FDI muscle of a quadriplegic subject (JB).
increase to more normal values. Thus it was difficult to determine with any assurance the percentage of motor units which fell into this category, but again it varied greatly between muscles and subjects.

Once a unit was activated, by whatever means, the motor unit action potential recorded by the needle electrode (trace 1 in fig 1) was used to trigger signal averaging of the unrectified surface EMG (trace 2), the rectified surface EMG (trace 3) and the force fluctuations in the muscle (trace 4). The reason for averaging the surface EMG both before and after rectification was to test for the presence of motor unit synchronisation.

Absence of motor unit synchronisation. The rectified surface EMG may contain 1) a non-zero steady level corresponding to the mean rectified EMG at the surface of the muscle, 2) one or more peaks corresponding to the phases of the unrectified surface EMG and 3) a broad peak representing the tendency of motor units in the muscle to fire at about the same time (synchronisation). In the recordings from this and other motor units of the quadriplegic subjects, no broad peak (item 3 above) was seen, so evidence of synchronisation was lacking.

Assuming that the motor units fired independently of each other, the force change seen in trace 4 gives an estimate of the twitch force and contraction time of the single motor unit shown above. Subjects were required to maintain firing rates less than 8–10 impulses/s to minimise the effects of partial fusion on estimated twitch tensions. Trace 4 has a time scale that is five times longer than the other traces and no delay was used, so the action potentials were largely over before force generation began.

Polyphasic potentials. In trace 1 of fig 1 the average motor unit action potential was highly polyphasic (six phases separated by zero crossings can be seen). This was quite common as shown in fig 2 where averaged motor unit action potentials for three successive units recorded from the same subjects are displayed. Motor unit action potentials from the first two impulses are highly polyphasic, whereas the third had a relatively simple triphasic shape. However, this unit had a small linked potential occurring about 5 ms after the main action potential. Such linked potentials were added to the number of phases if they represented additional zero crossings and were significantly above the noise level.

On average, the motor unit action potentials recorded from 16 muscles of subjects with spinal cord injuries had a mean and standard deviation (SD) of 5.5 (1.1) phases (the mean values ranged for different muscles from 4.4 to 9.6). In contrast, the values with the same electrodes had a mean of 4.1 (0.1) in control subjects and a range from 3.9 to 4.3. Thus there was no overlap in the two samples, but the values in the quadriplegic subjects varied from nearly normal to grossly abnormal.

Polyphasic potentials are a sign that the impulses from muscle fibres belonging to the motor unit recorded by the needle occur at slightly different times. This can arise, for example, from the presence of fine and hence slowly conducting branches of nerve fibres that sprout to innervate nearby, denervated muscle fibres.

Force generation. If substantial sprouting at nerve terminals had occurred, the force generated by motor units should increase considerably. Table 1 shows the values found for subjects in which spike-triggered averaging was used to estimate motor unit twitch tension from hand muscles, as explained in the Method and fig 1. The values of motor unit force for the quadriplegic subjects were not significantly different from those of normal subjects.

The force of the spontaneously active units described above for quadriplegic subjects was slightly smaller than the average unit in both

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**Table 1**  Mean twitch tensions of motor units from FDI and thenar muscles in normal subjects and quadriplegic subjects before and after receiving EMG biofeedback training

<table>
<thead>
<tr>
<th></th>
<th>Number of subjects</th>
<th>Units/subject</th>
<th>Mean Force (SE) (mN)</th>
<th>Range (mN)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FDI Muscles</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal subjects</td>
<td>7</td>
<td>5–72</td>
<td>28 (7)</td>
<td>13–64</td>
</tr>
<tr>
<td>Quads before biofeedback</td>
<td>8</td>
<td>2–12</td>
<td>23 (6)</td>
<td>8–57</td>
</tr>
<tr>
<td>Quads after biofeedback</td>
<td>6</td>
<td>5–13</td>
<td>22 (4)</td>
<td>11–32</td>
</tr>
<tr>
<td><strong>Thenar Muscle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal subjects</td>
<td>5</td>
<td>14–30</td>
<td>16 (5)</td>
<td>6–34</td>
</tr>
<tr>
<td>Quads before biofeedback</td>
<td>6</td>
<td>7–13</td>
<td>12 (2)</td>
<td>7–19</td>
</tr>
<tr>
<td>Quads after biofeedback</td>
<td>6</td>
<td>7–13</td>
<td>15 (4)</td>
<td>6–26</td>
</tr>
</tbody>
</table>
the force generated (86%) and the surface EMG recorded (84%). These differences from the average values were significant (p < 0.05 using Student's t-test). Possible explanations for these findings will be considered in the Discussion.

The order of recruitment was also normal in that small units were recruited first (their thresholds for recruitment were low) and increasingly large units were recruited at progressively large threshold force values (fig 3). An orderly pattern of recruitment is seen in patients after pressure block of the nerve or when the number of motor units is reduced in amyotrophic lateral sclerosis. An apparently random pattern of recruitment is seen after cutting and resuturing a nerve, but this may be due to misdirection of regenerating nerve fibres.

Biofeedback

The patients, whose hand muscles were studied in detail, were given intensive biofeedback using surface EMG and computerised displays of the RMS voltages recorded during periods of voluntary activity lasting about 10s (see Method). Figure 4 shows the range of peak EMG values (averaged over three successive 0-5s intervals) recorded on eight sessions over a three week period with one subject. The circled values show a more than five-fold increase from a low, initial value of 29 μV to a high, final value of 160 μV. The curves are parabolas fitted so as to minimise the root mean square error to the maximum and minimum values for each session. They are included more to guide the eye than to suggest a particular form for the increase, but they are more representative of the trend than simply picking high and low values.

Figure 4 was typical of the 14 hand muscles in which biofeedback was attempted. The ratio of highest to lowest values for these muscles was 5-6 (1-0) (mean SE) with a range from 1-4 to 13-5. A more conservative approach is to fit a curve, as was carried out in fig 4, to all the stored values. When this was done the mean ratio on the final and initial sessions was 2-0 (0-3) (range from 1-0 to 4-3).

In contrast, when we examined the twitch force of the single motor units that the subjects could activate in a follow up session after the biofeedback, no significant difference was seen (table 1). Nor was the EMG contributed by these single units to the surface recording significantly higher. The mean values (SE) were 109 (36) μV before biofeedback and 106 (31) μV after biofeedback. Finally, the percentage of spontaneously active units in the 14 muscles was 17%, before biofeedback and 18% following biofeedback. The reasons for these results will now be discussed.

Discussion

This is the first study to consider in detail the contractile as well as the electrical properties of motor units following spinal cord injury, and the changes in motor unit properties that take place following biofeedback using surface EMG. The results provide information concerning several important questions. For example, are there distinct classes of motor units that survive spinal cord injury? What is the nature of the very slow recovery which is often seen in these patients? What are the neural mechanisms underlying increases in the surface EMG during biofeedback? Answers to these questions will be proposed in separate sections below and experiments will be suggested for future tests of the proposals.

Classes of motor units after spinal cord injury

A mixed picture emerges in studying motor units of muscles innervated from levels of the spinal cord close to the injury. Some motor units are spontaneously active and modified by reflexes but not voluntary commands. Their motor neurons presumably lie below the level of injury and the spontaneous activity reflects the loss of the normal descending inhibition. Their somewhat smaller size, as measured by their force output or their EMG, probably does not imply that they are a distinct type of motoneuron. The reductions in size could easily result from their continuous activity at low firing rates. This pattern is well known to convert muscle fibres to oxidative metabolism, with the result that they are fatigue-resistant, but generate less force and contract more slowly.
Other motor units in the immediate vicinity of spontaneously active ones seemed to be activated quite normally and presumably had motor neurons situated above the level of the injury. The presence of a third class of very slowly activated units is intriguing. By using fast cortico-spinal connections normal subjects can activate motor units in a fraction of a second, yet the quadriplegic subjects showed substantially different latencies. If previous work had demonstrated a mechanism whereby the corticospinal motor neurones (possibly the slowest) are activated by first order neurones, it is possible that this was the case in the quadriplegic subjects. As a result, it was necessary to determine whether all the motor fibres had a normal latency or whether there was a spread of latencies that could be accounted for by the slowest units. 

It was necessary to establish the presence of a third class of very slowly activated units, and this was done by systematically changing the recording conditions. Two procedures were employed. In one, the subjects were asked to perform a task that activated only a small number of motor units. In the second, the same procedure was used, but with a much stronger electrical stimulus. The results of both procedures indicated that a large number of units had a latency of more than 500 ms. This is substantially greater than the latency of any motor units observed in normal subjects. It is possible that this was due to some form of reticular activation. It is also possible that the latency was increased due to the presence of a large number of units that were not normally activated. In either case, the results suggest that the latency of the units is not determined by the speed of conduction in the motor fibres, but rather by a factor that is not presently understood.

The presence of a third class of very slowly activated units is of interest because it suggests that the mechanism of activation is different from that observed in normal subjects. It is possible that this difference is due to the presence of a large number of units that are not normally activated, or to the presence of a large number of units that are activated by a different mechanism. In either case, the results suggest that the latency of the units is not determined by the speed of conduction in the motor fibres, but rather by a factor that is not presently understood.

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Since an orderly recruitment of motor units according to size could be demonstrated in these subjects (fig 3), any extra units recruited should have been very large ones. The lack of change in the size of the motor units, either in terms of increased electrical or contractile properties recorded after biofeedback, also argues against a substantial recruitment of previously unavailable units as a result of biofeedback. Finally, no change was observed in the percentage of motor units which were spontaneously active, so this group was not brought under voluntary control.

Thus by a process of elimination the evidence points towards the first possibility listed above and suggests that the increased biofeedback scores may result mainly from increased firing rate of motor units that could previously be activated. Technical differences, however, are such that other possibilities cannot be completely excluded. The peak EMG values recorded during biofeedback were only measured over a 1.5s period. In contrast, spike-triggered averaging required a contraction to be maintained for several minutes, so quite different mechanisms could be operative. For example, a voluntarily triggered spasm might be generated for a brief period, but not maintained. Similarly, during biofeedback muscles may be restrained or even stretched during effort, which could reflexly facilitate neurons in a way not matched during the needle EMG examinations. It is also technically very difficult to measure firing rates of motor units during brief, maximum voluntary contractions, but this should be attempted in future studies to confirm directly that increased rate is the most important factor in generating the greater surface EMG resulting from biofeedback.

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