Early and late losses of motor units after poliomyelitis

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Summary

Motor unit number estimation was employed to assess muscle innervation in 76 patients with prior poliomyelitis. Of the 68 patients who were <70 years of age, new musculoskeletal symptoms had appeared in all but four; the mean latent interval was 38.0 ± 10.1 years. As expected, there was a high incidence of denervation in previously affected limbs (87%). However, the incidence in supposedly unaffected limbs was also high (65%). Significant differences in the degree of denervation were found between muscles of the same hands and feet. Judged on the basis of their potential amplitudes, the surviving motor units in partially denervated muscles tended to be enlarged. The enlargement was proportional to the extent of the denervation and was comparable to that found in amyotrophic lateral sclerosis. In some muscles, possibly those innervated by failing motor neurons, motor-unit enlargement was not present. Needle examination confirmed the high incidences of denervation in affected and allegedly unaffected limbs. Of the 188 muscles with EMG features of chronic denervation, only nine exhibited fibrillations or positive sharp waves (4.8%). Ninety-five symptoms had appeared in all but four; the mean latent interval was 38.0 ± 10.1 years. As expected, there was a high incidence of muscles exhibiting denervation in previously affected limbs (87%). However, the incidence in supposedly unaffected limbs was also high (65%). Significant differences in the degree of denervation were found between muscles of the same hands and feet. Judged on the basis of their potential amplitudes, the surviving motor units in partially denervated muscles tended to be enlarged. The enlargement was proportional to the extent of the denervation and was comparable to that found in amyotrophic lateral sclerosis. In some muscles, possibly those innervated by failing motor neurons, motor-unit enlargement was not present. Needle examination confirmed the high incidences of denervation in affected and allegedly unaffected limbs. Of the 188 muscles with EMG features of chronic denervation, only nine exhibited fibrillations or positive sharp waves (4.8%). Ninety-five muscles of 18 patients were studied a second time after an interval of 2 years. Overall, there was a 13.4% reduction in motor-unit number and a 18.4% diminution in M-wave amplitude (P < 0.001). The rate of motor-unit loss was twice that occurring in healthy subjects aged >60 years. Analysis of individual patients indicated that some were deteriorating more rapidly than others. These studies confirm that denervation progresses in patients with prior poliomyelitis in both clinically affected and unaffected muscles, and indicate that this progression is more rapid than that occurring in normal ageing.

Keywords: poliomyelitis; motor units; EMG

Abbreviation: MUNE = motor-unit number estimation/estimate

Introduction

Despite the passage of time, there probably remain in North America >200 000 patients who contracted poliomyelitis prior to the introduction of vaccination programmes in 1955 (cf. Windebank et al., 1991). Of these patients, the majority either already have, or may be expected to develop, further weakness and fatiguability, as a delayed consequence of their earlier illness. Because of this late progression, there has been renewed speculation as to the chronic effects of poliomyelitis on the spinal cord. In general there are two opinions, which are not mutually exclusive. One is that the late changes are the inevitable accompaniment of normal motor neuron ageing, though occurring earlier and running a more rapid course because of the previous illness and subsequent compensatory adaptations by the motor neurons. Thus, motor neurons surviving the acute paralytic episode could nevertheless have been ‘weakened’ by the viral invasions. Further, the propensity of the surviving neurons to adopt and support orphaned muscle fibres would increase metabolic demands made on the motor neuron cell bodies. The second explanation for the late symptoms is less obvious and more intriguing, for it postulates that there is a continuing immunological reaction against previously infected motor neurons; indeed, perivascular lymphocytic infiltrates have been observed in spinal cords post mortem (Pezeshkpour and Dalakas, 1987) and it is possible that poliovirus RNA persists in some neurons (Dalakas, 1995).

Regardless of the pathophysiological mechanisms underlying the late deterioration, there is interest in studying
certain features of the spinal motor neurons in affected patients. The most obvious and important question is whether further loss of functioning motor neurons does indeed take place, a question which the motor-unit number estimation (MUNE) procedure should be ideally suited to answer. This type of analysis, which was first described in 1971 (McComas et al., 1971a), has been of considerable theoretical interest, and has been employed to study ageing as well as a number of neuromuscular disorders (for recent reviews, see McComas, 1991, 1995). Although a number of MUNE methodologies have been described, all entail the comparison of single motor-unit properties with that of the parent muscle. In the only such study of post-polioymyelitis patients reported to date, Daube et al. (1995), found no significant loss of motor units over a 5-year period; it could be argued, however, that the statistical approach used may not have been sufficiently sensitive (see Discussion). In addition to tackling this issue, we have employed MUNE to answer three subsidiary questions. First, can muscle denervation be demonstrated in limbs thought not to have been involved in the acute paralytic illness? Secondly, are neighbouring motor neuron populations in the spinal cord equally susceptible to destruction by the poliovirus? And finally, is collateral reinnervation as prominent after poliomyelitis as with a denervating disorder such as amyotrophic lateral sclerosis?

Methods

Subjects

The study initially involved 78 patients with a history of poliomyelitis. In those patients who contracted the disease in early childhood, the circumstances of the illness were accepted as those which had been told to the subject by his or her parents. This account included the identification of the affected limbs, although the presence or absence of subsequent weakness and wasting was obviously critical information also. Of the 78 patients there were 56 (21 male, 35 female) who had been referred by a family doctor or specialist for EMG testing at the McMaster Health Sciences Centre in Hamilton during the period 1983–1996; their ages ranged from 29 to 84 years. Some of the 78 patients had medical problems, in addition to the effects of their previous poliomyelitis, and there were two in whom the diagnosis of poliomyelitis was doubtful; these issues are considered in the Results section. The remaining 22 patients (13 male, nine female) were volunteers for the follow-up study of motor-unit number during 1986–1989. These last subjects were all those available to physicians at the Strong Memorial Hospital, Rochester, NY, USA. Of these 22, 18 attended for a second examination after a planned interval of 2 years; the mean age of the 11 men and seven women at the first study was 58.6 ± 7.9 years. For the study of reliability in MUNE, seven healthy subjects aged 23–55 years were examined in 1989. All subjects gave their consent to the investigation and the study carried the approval of the ethics committees of the two universities involved.

MUNE and EMG examinations

The EMGs were performed by two of the authors, one of whom (A.J.M.) was also responsible for all of the MUNEs in the follow-up and reliability studies. Of the 580 MUNEs in the entire study, 568 were carried out by the original incremental method of McComas et al. (1971a). In this method the stimuli to a motor nerve are gradually increased in intensity from a subthreshold value, causing the muscle response to enlarge in discrete steps, each of which is assumed to reflect the excitation of an additional motor unit. The MUNE is then determined by dividing the mean peak-to-peak amplitude of the increments into the peak-to-peak amplitude of the maximal response evoked in the same muscle (or muscle group). Stimulation and recording are performed with surface electrodes. Other details of the technique, with the underlying assumptions and limitations, are described elsewhere (McComas et al., 1971a). To enhance accuracy in the follow-up and reliability studies, the samples of motor-unit potentials were made as large as possible (up to 20). In the same studies the estimates were performed without knowledge of previous values. The control values, used for comparison, were those obtained in previous investigations of healthy subjects (McComas, 1977). Only 12 MUNEs were conducted with the automated incremental method (Galea et al., 1991), largely because most of the data had already been gathered. To ensure consistency, the automated method was not used for any of the muscles in the follow-up study.

All patients had measurements made of terminal motor-latency, and usually impulse conduction-velocities, for the nerves of the muscles examined by MUNE; sensory nerve conduction studies were made in the hands and feet. Coaxial needle electrodes were employed for intramuscular recordings. The EMG machines were a Dantec Neuromatic 2000 (with a Tektronix storage oscilloscope as slave), an Advantage (Advantage Medical, London, Ontario), and a custom-built unit incorporating a variable persistence storage tube (Hewlett Packard). The latter unit was used for all the MUNEs in the follow-up and reliability studies and was transported to Rochester for the studies on the local patients.

Statistics

Throughout the text mean values have been given with their SDs. The significance of differences between means was assessed with Student’s t test. The $\chi^2$ test was employed to analyse the results of the follow-up study.

Results

Characteristics of the patient population

Of the 78 patients, two were rejected from the study because the diagnosis of prior poliomyelitis was questionable. Seventy of the 76 patients had delayed musculoskeletal symptoms, which in all but two consisted of increasing weakness and
fatigue, or both. In 10 patients the weakness involved limbs not thought to have been affected in the original poliomyelitic illness; in two additional patients bulbar weakness appeared for the first time, as did respiratory weakness in another. Some of the patients volunteered that their muscles had wasted, but in two there was enlargement of partially denervated calf muscles. In two patients muscle cramps and pain respectively were the presenting symptoms. The mean interval between the poliomyelitic illness and the onset of new symptoms was 40.5 ± 12.2 years. In order to reduce the contribution of normal ageing to the motor-unit results, eight patients aged ≥70 years were excluded from the remainder of the study. Of the remaining 68, there were 28 men and 40 women aged 29–69 years (mean age, 53.9 ± 8.9 years). The mean age of these subjects at the time of their poliomyelitic illness was 9.8 ± 7.8 years (range, 6 months to 30 years). The mean delay before new symptoms appeared was 38.0 ± 10.1 years (range, 18–62 years). Within this younger group, a number of patients had additional medical problems, as follows: mild carpal tunnel syndrome (four), late-onset diabetes without neuropathy (two), alcoholism (two), previous myocardial infarction (three), sciatica (two) and rheumatoid arthritis (one). None of these complications were used as grounds for exclusion from the study.

**Motor unit numbers at the first visit**

At the first visit, MUNE was performed on two muscles in the most affected limb, usually with an additional estimate in another limb. In those patients in the follow-up study, however, the same six muscles were examined routinely; these were the extensor digitorum brevis and soleus muscles of both legs, together with the thenar and hypothenar muscle groups of one hand. Examination of the extensor digitorum brevis was omitted if a surgical scar, from a tendon transfer, extended over the normal site of the muscle belly.

In limbs known to have been affected in the original poliomyelitic illness, 149 of 170 muscles (87%) examined were found to have reduced numbers of motor units, the mean number of units remaining being 40.8 ± 49.2% of normal. Twelve muscles were completely denervated.

In limbs not considered to have been involved by poliomyelitis, 49 of 75 muscles (65%) showed loss of motor units, the mean number of units remaining being 60.4 ± 40.1% of normal. Figure 1 shows the distributions of the motor-unit estimates in the previously affected limbs and in supposedly unaffected limbs. In Table 1 the results for the affected and ‘unaffected’ limbs have been combined to show the degrees of involvement of the different muscles. It can be seen that there was almost three times as much denervation in the soleus as in the plantar muscle group. Similar differences persisted when comparisons were restricted to muscles in the same hand and foot. Thus, in 32 feet, the mean numbers of extensor digitorum brevis and plantar motor units were 42.9 ± 37.1% and 76.1 ± 68.3% of normal, respectively (P < 0.02), while in 27 hands the mean numbers of thenar and hypothenar motor units were 38.2 ± 32.5% and 69.2 ± 44.3% of normal, respectively (P < 0.01).

**Relative sizes of motor units**

In muscles that are partially denervated, the axons of surviving motor neurons will normally sprout and form new neuromuscular junctions with the denervated fibres. The extent of such collateral reinnervation was assessed after poliomyelitis by comparing the number of remaining motor

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**Table 1 Comparison of MUNEs in different muscles**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Observations (n)</th>
<th>MUNE</th>
<th>Percentage of control mean</th>
</tr>
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<tbody>
<tr>
<td>Soleus</td>
<td>38</td>
<td>254 ± 236</td>
<td>26.6 ± 24.7</td>
</tr>
<tr>
<td>Extensor digitorum brevis</td>
<td>86</td>
<td>71 ± 60</td>
<td>34.1 ± 28.6</td>
</tr>
<tr>
<td>Thenar</td>
<td>49</td>
<td>145 ± 119</td>
<td>42.3 ± 34.8</td>
</tr>
<tr>
<td>Hypothenar</td>
<td>31</td>
<td>269 ± 175</td>
<td>69.6 ± 44.9</td>
</tr>
<tr>
<td>Plantar</td>
<td>38</td>
<td>290 ± 254</td>
<td>74.3 ± 65.1</td>
</tr>
</tbody>
</table>

Values are means ± SDs. Control means as given in McComas (1977).
units with the mean amplitude of their motor-unit potentials, since the latter should be proportional to the sizes of the respective muscle fibre populations. In Fig. 2A it can be seen that there was considerable spread in motor-unit potential amplitude for a given degree of denervation, but that the largest potentials were associated with the greatest denervation. In the lower part of Fig. 2 are shown, for comparison, the corresponding results for the extensor digitorum brevis muscles of patients with amyotrophic lateral sclerosis (M. Dantes and A. J. McComas, unpublished data). Although the largest mean potentials (>240 µV) were restricted to amyotrophic lateral sclerosis muscles, there was no significant difference between mean sizes of the M-waves in the two disorders when the most severely denervated muscles were compared (Table 2). Since the M-waves are the products of the numbers of motor units and the mean motor-unit potentials, it appeared that the amounts of axonal sprouting were similar in the two disorders.

**Follow-up and reliability studies**

An attempt was made to detect any further losses of functioning motor units in individual patients with prior poliomyelitis. For this purpose 22 patients were recruited in Rochester and of these 18 returned for a second study after 2 years. Fourteen of the 18 patients admitted to some deterioration in their strength and exercise tolerance. Two patients were unchanged in their symptoms and two were improved, following diet-induced losses of weight and the introduction of exercise programmes. Regardless of the distribution of the weakness, on both visits a proximal and distal muscle (soleus and extensor digitorum brevis) were examined in each leg, and two intrinsic muscle groups (thenar and hypothenar) in one hand. By investigating six muscles in each patient and pooling the results, it was hoped that it would be possible to compensate for the methodological error in the MUNE and to determine whether or not an individual had deteriorated over the 2-year period. Critical to this type of analysis is the reliability of increment-based MUNE, and this was investigated by examining 10 thenar muscle groups and 10 extensor digitorum brevis muscles in adult controls. Each of the 20 muscles was examined on 10 occasions (five different days) by the same investigator responsible for the follow-up study of prior poliomyelitis patients. The consistency of the results was found to vary considerably among the control muscles, with coefficients of variation ranging from 11.4% to 30.9%. Nevertheless, when 180 consecutive pairs of MUNEs were compared, there were only four occasions when one result was twice as large, or greater, than the other. Hence the probability of a ‘doubling’ or ‘halving’ of the motor-unit estimate occurring by chance in controls was 4/180 = 0.02.

In the 18 patients with prior poliomyelitis who were examined a second time, it was possible to compare MUNEs in 96 muscles. In 15 of these muscles reliability was assessed by carrying out a second MUNE during the same recording session. The mean coefficient of variation for the pooled data was 7.7 ± 5.0%, and was smaller than values derived for control muscles (see above). One reason may have been the ability of the MUNE technique to sample a larger proportion of the motor-unit population in the patients; in those muscles with the greatest denervation all the motor units were included.

**Table 2** Comparison of M-wave amplitudes in severely denervated muscle in amyotrophic lateral sclerosis and post-poliomyelitis

<table>
<thead>
<tr>
<th></th>
<th>Observations (n)</th>
<th>MUNE (mV)</th>
<th>M-wave amplitude (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-poliomyelitis</td>
<td>13</td>
<td>13.9 ± 8.4</td>
<td>2.56 ± 2.76</td>
</tr>
<tr>
<td>Amyotrophic lateral sclerosis</td>
<td>35</td>
<td>11.1 ± 7.3</td>
<td>1.97 ± 1.72</td>
</tr>
</tbody>
</table>

Values are means ± SDs. All muscles with 25 or fewer motor units were included.
Table 3 Comparison of MUNEs and M-wave amplitudes after a 2-year interval

<table>
<thead>
<tr>
<th></th>
<th>MUNE (%)</th>
<th>M-wave (%)</th>
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<tbody>
<tr>
<td>Initial values</td>
<td>106.7 ± 24.7</td>
<td>109.2 ± 19.0</td>
</tr>
<tr>
<td>After 2 years</td>
<td>93.3 ± 24.7</td>
<td>90.8 ± 19.0</td>
</tr>
</tbody>
</table>

A total of 95 muscles were studied. Values were expressed as percentages of means for each pair of observations. Reductions in MUNE and M-wave were both highly significant (P < 0.001).

in the sample. Of the 96 muscles examined, it was found that the estimate had doubled in five muscles but had halved in 16 others, a difference which was highly significant (P < 0.01). The M-wave amplitude, which is a rather more robust index than MUNE, was also compared at the two visits and was found to double in one muscle, but to halve in seven. In five of the seven muscles there had also been halving of the MUNEs, a fact which supported the conclusion that these muscles had deteriorated. Of the 18 patients, there was one, a 60-year-old woman, in whom four of six MUNEs had halved, and another, a 58-year-old man, in whom two estimates and one M-wave had halved; these two patients showed the greatest clinical changes over the 2-year period. Six patients had no significant changes in the MUNEs and M-wave amplitudes.

The results of the follow-up study were analysed in another way, by expressing each MUNE as a percentage of the mean of the two values obtained 2 years apart. There was a 13.4% reduction in MUNE and a 18.4% diminution in M-wave amplitude, both changes being statistically significant. (see Table 3).

Needle electrode recordings
Coaxial needle recordings were performed on muscles of limbs thought to have been affected and those not affected by poliomyelitis. Attention was paid to the presence or absence of spontaneous activity, the density of the interference pattern, and the amplitudes, durations and phases of the motor-unit potentials. All assessments were made subjectively by the same two electromyographers, and only the results in patients aged <70 years were considered.

In 202 muscles in previously affected limbs, the needle recordings revealed evidence of chronic denervation in 154 (76.2%), while in 78 muscles of allegedly unaffected limbs denervation was noted in 34 (43.6%). Of the total of 188 muscles with signs of chronic denervation, fibrillations and/or positive sharp waves were seen in only nine.

Discussion
Patient characteristics
It is now accepted that patients who have suffered from acute paralytic poliomyelitis many years before may develop fresh or further weakness, muscle atrophy, fatiguability and pains in muscles and joints; this constellation of symptoms has been termed the post-polio syndrome (Halstead and Rossi, 1987). In a well-designed study the incidence of post-poliomyelitis patients with new musculoskeletal symptoms was given as 21% (Codd et al., 1985). With the elapse of time, however, and with increasing awareness of the post-polio syndrome, the reported incidence of the latter has risen, and it has even been suggested that eventually all post-poliomyelitis patients may manifest at least some features of post-polio syndrome (Munsat, 1991). Since our own sample of 76 patients was made up of referrals from other physicians, the incidence of 92% with post-polio syndrome is clearly higher than that in the general population of subjects with prior poliomyelitis. It may be noted, nevertheless, that approximately one-third of our patients had consulted their physicians to ascertain if they had post-polio syndrome, which they had learned of through the media or from an acquaintance. Of our 70 patients with new neuromuscular symptoms, the mean interval between the onset and the previous poliomyelitic illness was 40.5 ± 12.2 years, a value which is close to that of 36 years found by Jubelt and Cashman (1987).

Within the population of 76 patients, 185 body ‘parts’ were considered to have been involved in the original illness; we arbitrarily class as a ‘part’ an arm or a leg, the bulbar muscles, the respiratory muscles and the trunk. At the time of the initial electrophysiological examination, symptoms had appeared in a further 13 parts.

Motor-unit estimates
Regarding the use of MUNE in our study, it must be stated that, despite the considerable time which has elapsed since the introduction of this methodology (McComas et al., 1971a), the determinations remain approximate for the reasons given in the original publication. On a more positive note, the different techniques which have been devised since 1971 yield control values which are similar (e.g. Stein and Yang, 1990) and in reasonably good agreement with the sparse data on axon counts (McComas, 1977). The original incremental technique was used for all but 12 estimates, partly because of its speed and simplicity and, partly because of the extensive collection of control data already obtained with it, in our laboratory. The first of the automated methods for motor-unit estimation did not become available until the study was well advanced.

Since MUNE is the only methodology that can determine the number of functioning motor units and motor neurons during life, it has an obvious application to the study of post-poliomyelitis patients. In the only such study reported to date (Daube et al., 1995), MUNEs were carried out on thenar and extensor digitorum brevis muscles of 50 patients by an automated technique. In both muscles the mean estimates were less than half those of controls. Our own results are similar, the mean numbers of surviving units in the extensor digitorum brevis and thenar muscles being 34% and 42%
of normal, respectively. Interestingly, we found that the percentages of surviving plantar and hypothenar motor units, even in the same hands and feet, were significantly higher. Such differences in vulnerability have been noted before in ageing and in generalized peripheral neuropathies, such as that due to vincristine (McComas, 1977), but it was rather unexpected to find it a feature of a viral illness. The cellular basis for such differences remains unknown.

Of the limbs known to have been affected by the acute paralytic illness, reduced numbers of motor units were observed in the great majority of muscles examined (87%). Of rather more interest, was the high proportion (65%) of affected muscles in limbs which were not thought to have been involved originally. This has been briefly noted before (McComas, 1991) and has recently been described on the basis of macro-EMG and muscle histochemical results (Luciano et al., 1996). Our present findings with MUNE were supported by the results of the coaxial needle examination in more proximal muscles; of the latter, 44% showed evidence of denervation in limbs not thought to have been affected by poliomyelitis. These data may be viewed in the context of two very different findings. On the one hand, Halstead and Rossi (1987) reported that 50% of their post-polio myelitis patients had complained of weakness in previously unaffected muscles. On the other hand, Windebank et al. (1995) assessed 2700 muscles in 50 patients and found only four that were weakened and had supposedly not been involved in the paralytic illness. Our own MUNE results, and the findings of Luciano et al. (1996), suggest that subclinical infection of motor neuron pools by virus must have been common. In such patients weakness was presumably prevented by collateral reinnervation, and studies from our laboratory have shown that 80% of a motor neuron pool may be lost before twitch force is reduced (McComas et al., 1971b).

**Does further loss of motor units take place?**

In earlier studies, we used MUNE to show that ageing is accompanied by a loss of functioning motor neurons in the human spinal cord and that, in healthy subjects, the onset is delayed until after 60 years of age (Campbell et al., 1973; Sica et al., 1974). More recent investigations have confirmed that the rate of loss varies between muscles and, of those tested, is highest in extensor digitorum brevis and lowest in the biceps brachii (Galea, 1996). It is logical to enquire whether post-polio syndrome could be due to neuronal attrition by ageing. If this is the case, the fact that post-polio syndrome may develop well before 60 years of age, would indicate that the ageing effects are accelerated. As postulated for amyotrophic lateral sclerosis (McComas et al., 1973), it could be that motor neurons supporting enlarged muscle fibre colonies would be metabolically stressed (cf. Dalakas, 1995). In the only follow-up study to date employing MUNE, Daube et al. (1995) examined 50 extensor digitorum brevis and 50 thenar muscle groups in post-polio myelitis subjects after an interval of 5 years. Although the mean numbers of units were further decreased in both muscles, the changes were not statistically significant. As suggested by the authors, the lack of significance could have been due to the broad range of values. Thus a 50-unit decrease in MUNE, say from 400 to 350, would be well within the methodological error, whereas a similar loss, but this time from 60 to 10, would be highly significant. An alternative approach would be to express all the follow-up MUNEs as percentages of the mean of the initial ones. However, it is easily shown that, given random distribution of values, the mean follow-up MUNE would necessarily be higher than 100%. Taking the above considerations into account, we have expressed the initial and follow-up MUNEs as percentages of the mean of each pair of results. Using this approach, we found a highly significant reduction in MUNE, as well as in M-wave amplitude, in 2 years. The 13.5% reduction in MUNE in this period is approximately twice the rate of loss to be expected in the most vulnerable muscles of healthy subjects aged >60 years (cf. Campbell et al., 1973). Significant losses of motor units were also found in our patients, when a \(\chi^2\) test was employed to analyse the incidence of MUNEs that had halved or doubled among the 96 pairs of results. As noted by others, such EMG changes as decremental responses to repetitive stimulation (Hodes, 1948), increased single fibre jitter and blockings (Wiechers and Hubbell, 1981) and responses to anticholinesterases (Cashman and Trojan, 1995), could reflect impairment of neuromuscular transmission in failing motor neurons.

**Sizes of surviving motor units**

The amplitudes of motor-unit potentials give an indication of the relative sizes of the muscle fibre colonies, though not of their territorial boundaries. In post-polio myelitis patients, size measurements are important in indicating the avidity of surviving motor neurons for annexing denervated muscle fibres. At the other extreme, unusually small motor-unit potentials might reflect motor neurons that were failing, and had shed some of their muscle fibres, as appears to happen in amyotrophic lateral sclerosis (Dengler et al., 1989). Our findings reveal evidence of both situations. Thus, the largest motor-unit potentials were found in those muscles with fewest units, but there were also very denervated muscles in which motor-unit enlargement was not present. In this last respect, unusually small motor-unit potentials in prior poliomyelitis patients have also been found with macro EMG recordings (Lange et al., 1989).

We were interested to see whether the capacity of collateral reinnervation after poliomyelitis was any different to that in amyotrophic lateral sclerosis, a condition of different aetiology in which the mean rate of motor-unit loss is very much higher (typically 50% in 6 months; Dantes and McComas, 1991). Our comparison failed to show any difference in reinnervation capacity between the two conditions.
Conclusions
In conclusion, MUNE may be said to have fulfilled expectations in throwing new light on the extent of muscle denervation in poliomyelitis, and on the adaptive properties and subsequent loss of surviving motor units. Thus, motor-unit loss following the initial infection was shown to be more severe and more widespread than previously supposed. Axonal sprouting is as vigorous as in amyotrophic lateral sclerosis, but is not seen in some motor units, possibly because of impending motor neuron death. Indeed, in some patients with post-polio syndrome, further losses of motor units can be demonstrated.

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