

MOTONEURON ACTIVITY AND MUSCLE FIBRE TYPE COMPOSITION IN HEMIPARESIS

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ABSTRACT. The firing of single motor units (MUs) in musculus tibialis anterior (TA) was studied during maximal voluntary effort and maximal speed of walking in 10 patients with severe chronic hemiparesis and the findings compared to normal data. As shown in a previous study, the paralysed TA exhibited an increase in proportion of type 2 fibres as compared with normal muscle. Thus, 57% of the muscle fibres were type 1 and 43% type 2, while the normal percentages were 80% and 20% respectively. The present findings indicate that in the paralysed muscles a little less than half of the fibres, i.e. roughly the equivalent of the type 2 population, was not tonically active either during sustained voluntary contraction or during locomotion. Normally high threshold MUs reached high rates during both modes of activation. The findings in paralysed muscles also indicate that a little more than half of the fibres, i.e. roughly the equivalent of the type 1 population, could be brought into tonic firing during voluntary contraction as well as during walking. Their maximal firing rate was, however, no more than two thirds of that of normal low threshold MUs.

Key words: motor unit, recruitment, firing rate, muscle fibre type, hemiplegia.

It is known from animal experiments that long-term electrical tetanization causes a transition of type 2 to type 1 fibres, and that immobilisation leads to the opposite transition. A transition from type 2 to type 1 may occur also during physiological training in man and back to type 2 during detraining. For references see reviews by Salmons & Henriksson (18) and Edström & Grimby (5).

We showed in a previous study (15) that hemiparetic patients who markedly disused musculus tibialis anterior (TA) during locomotion, that was the main function of the muscle, had a decreased proportion of type 1 TA fibres and an increased proportion of type 2B fibres. Ten patients in whom the TA surface electromyogram (EMG) during the ordinary step cycle or maximal voluntary effort did not reach 50% of that in

normal subjects were studied. $57.4 \pm 18\%$ of the fibres were type 1 and $42.6 \pm 18\%$ type 2. The normal fibre composition was $79.6 \pm 9\%$ type 1 and $20.4 \pm 8\%$ type 2. The difference was statistically significant ($p < 0.05$).

The aim of the present study was to determine to what extent the low TA global EMG in the hemiparetic patients was due to low firing rates and to what extent it was due to low recruitment, i.e. to relate the disuse to muscle fibre type.

MATERIAL AND METHODS

Four women and six men with severe chronic non-progressive hemiparesis were studied. Their mean age was 55.8 ± 8.8 years. The duration of the impairment varied between 3 and 18 years (mean 9.4 ± 4.4 years). In six subjects the cause was a cerebral infarct, two have had cerebral hemorrhagia, one had residuals after a brain concussion and one had residuals after an operation for a benign cerebral neoplasm. Eight subjects had leftsided and two rightsided hemiparesis. All patients could walk without aids.

Normal subjects were used as controls since the contralateral leg in hemiparesis was also disused during locomotion. In the studies of submaximal voluntary contraction 10 sex- and age-matched members of the laboratory staff were used. Normal data for locomotion and single MU discharge during maximal voluntary effort were obtained from previous studies (7, 8, 9, 10).

The global TA electromyogram (EMG) was recorded with surface electrodes (Medelec Ltd Woking Surrey UK) located 2 cm apart over the belly of the muscle about 2 cm from the tibia bone and 5-10 cm distal to the tuberositas. Time constants of the amplifiers were 10 ms (LF) and 100 ms (HF). The signals were rectified and the resulting surface was integrated over a period between 40 and 100 ms. TA power was not studied in the hemiparetic patients since it could not be made in a conclusive way because of cocontraction of strong antagonists.

EMG recordings with a selectivity and stability permitting the identification of single TA MU potentials were obtained during low to moderate voluntary tension with conventional single fibre needle electrodes (Medelec Ltd, Woking, Surrey, U.K.) and during locomotion and high voluntary tension with wire electrodes made from insulated silver wires 20-100 μm equipped with a hook for fixation in the muscle (7, 11).

Satisfactory recordings were obtained in the paralysed muscles without major difficulty since the level of activation was low. Normal studies of maximal tension and particularly locomotion were, however, laborious (8).

The following procedure was used to get an idea of the global EMG that would have been recorded in the hemiparetic patients if fully fused contraction had been achieved. The foot was fixed at 120 degrees in the foot joint. A supramaximal single electrical shock to the peroneal nerve was elicited in the paralysed and control TA. The integrator was started during the interval between the shock artefact and the evoked response. The amplification was adjusted so that the two potentials were of identical size. The EMG recorded during maximal voluntary tension in the control TA was then used as a reference in the paralysed TA as well. We assumed that differences in global EMG then were due mainly to differences in recruitment or firing rates.

During walking the recording electrodes were connected to a small pre-amplifier strapped to the leg and connected to the main amplifier by a cable permitting 40 m of locomotion. The EMG activities were related to the signals from two tape switches (Stig Wahlström AB, Box 64, S-123 22 Farsta, Sweden), the one strapped to the heel and the other to the anterior part of the shoe. The two switches operated at different voltages so that the signals could be distinguished when recorded simultaneously. All subjects used indoor shoes with low heels and low weight.

For statistical analysis, the Wilcoxon rank sum test (two-tailed) was used for intergroup comparisons. $p < 0.05$ was considered statistically significant. Values are given as mean \pm standard deviation.

RESULTS

Long-term recordings of the TA surface EMG in hemiparesis during activities of daily living and physiotherapy showed that the strongest activations occurred during voluntary contraction with biofeedback. Long-term studies in normal subjects indicated that the muscle was mainly used during walking (8). These two modes of activation were studied.

Control TA

Maximal voluntary tension. Normal subjects could maintain the maximal tension of TA for several seconds. This could be checked by observing that superimposed supramaximal electrical stimulation of the peroneal nerve did not cause any increase of tension. During the first second of maximal voluntary tension high threshold MUs fired at 30–65 Hz and low threshold MUs at 25–30 Hz (10). After a few seconds, however, 25–30 Hz were sufficient for tension in all MUs (8) because of the increased relaxation time of rapidly contracting muscle fibres during prolonged contraction (1). The integrated surface EMG record-

ed during prolonged maximal tension at 120 degrees in the foot joint was used as reference (cf. below).

Submaximal voluntary tension. During slowly increasing voluntary effort the MUs were orderly recruited. There was a correlation between threshold and minimal rate for tonic firing (no interval > 200 ms). The MUs with the lowest threshold had a minimal rate < 10 Hz. New MUs were successively recruited until low threshold MUs had reached 20 Hz. Just after recruitment a MU with a higher threshold fired at more irregular intervals and fewer times per second than a simultaneously recorded low threshold MU. During stronger voluntary contraction the differences decreased. The MUs with the highest threshold did not fire tonically (no interval > 200 ms) at a rate lower than 15–20 Hz. Simultaneously recorded low threshold MUs then fired at 20–25 Hz.

Tonically active MUs fired at 14.5 ± 2 Hz (Table I) when the integrated surface EMG was 25% of that recorded during prolonged maximal tension. Intermittently firing MUs were so few and their discharges so scarce that they probably did not make any major contribution to the global EMG.

Locomotion. TA was active during the swing phase with a marked peak lasting about 100 ms starting just before the heel strike. There was a reciprocity between TA and triceps surae activity. During comfortable walking the maximal integrated surface EMG was about half of that recorded during prolonged maximal tension. During rapid locomotion the global EMG exceeded that recorded during prolonged maximal tension (14).

The MUs were recruited during increasing speed of locomotion in the same order as during increasing voluntary tension. During comfortable speed of walking, low threshold MUs fired 5–10 times per step cycle at intervals corresponding to 10–15 Hz during most of the swing phase and 20–25 Hz during the heel strike peak. Intermediate threshold MUs fired mainly a few times at heel strike. High threshold MUs were not recruited in the ordinary step cycle but participated frequently in corrective movements (9).

Paralysed TA

Maximal voluntary effort. The maximal voluntary TA tension could not be measured conclusively due to marked co-contraction of antagonists. The maximal integrated surface TA EMG was, however, 25% to 50% of that recorded during prolonged maximal tension in normal subjects.

Few MUs reached > 15 Hz and no MU > 20 Hz.

Table I. Firing rates of tonically active MUs in the paralysed TA and the normal control subjects at the 25 % global EMG level

For further description see methods and results

Number	Hemiplegic subjects				Normal subjects			
	Hz	SD	Range	No. of MU studied	Hz	SD	Range	No. of MU studied
1	11	3.6	(6-20)	16	14.5	2.4	(11-20)	13
2	10.4	1.7	(8-12)	5	13.7	2.1	(11-18)	14
3	12	1.5	(10-16)	28	16.9	1.9	(14-20)	11
4	12.3	1.7	(10-14)	8	15.1	1.9	(12-19)	15
5	12.4	2.3	(9-16)	7	15.2	2.4	(12-20)	11
6	11.7	1.4	(8-13)	10	18.1	1.5	(16-20)	11
7	11.6	1.7	(9-14)	14	14.4	1.2	(12-16)	16
8	10.4	0.9	(9-12)	15	15.3	1	(14-17)	15
9	9.9	1.7	(7-12)	10	10.6	1.4	(8-13)	16
10	8.8	0.7	(8-10)	9	13.6	1.2	(11-16)	24
Total	Mean 11.1	2.1	(6-20)	122	Mean 14.7	2	(8-20)	146

Just recruited MUs fired only occasionally at very irregular intervals. Fig. 1A illustrates a finding during maximal voluntary effort in one patient. One MU with a relatively low threshold fired tonically at 12-13 Hz (low amplitude potential). Another MU with a relatively high threshold fired only occasionally (high amplitude potential).

At the 25% global EMG level reached by all patients tonically active MUs had a mean firing rate of 11.1 ± 2.1 Hz. As mentioned above the corresponding figures in normal subjects were 14.7 ± 2 Hz (Table I). The difference between firing rates was statistically significant ($p < 0.01$).

It can be argued that part of the integrated surface EMG is derived from muscles co-contracting with TA. The peroneal muscles (P) and the TA co-contracted regularly, but there was no significant difference in the TA/P relation between paralysed and normal subjects. The triceps surae (TS) muscle co-contracted with TA more in paralysed than in normal subjects. However, studies in normal subjects capable of selective TS activation indicated that TS activity comparable to that occurring during voluntary dorsiflexion in hemiparesis was hardly picked up by the surface TA electrodes.

Submaximal voluntary effort. During slowly increasing voluntary effort the MUs were orderly recruited. A low threshold MU fired at a higher rate than a simultaneously recorded later recruited MU. The higher firing rate of high threshold MUs than of

low threshold MUs found in normals during maximal effort was not seen in the patients.

Locomotion. In hemiparesis, only slow walking was possible. There was a less marked heel strike than normally and the heel strike EMG peak was absent. The global EMG in the ordinary step cycle exceeded the 25% level only in one patient. On the other hand, TA activity was recorded not only during the swing phase as in normal subjects but also during parts of the stance phase. The normal reciprocity between the TA and the triceps surae muscles was disturbed in hemiplegia.

At the 25% level tonically active MUs fired at about 10 Hz (Fig. 1). The same MUs were active during locomotion as during the corresponding voluntary contraction.

DISCUSSION

The MUs were orderly recruited during voluntary contraction as well as during locomotion (7, 12), and it can be assumed that the type 1 population was recruited before the type 2 population.

The main objective of the present study was to determine the proportion of TA muscle fibres that was not recruited in our paralysed subjects during sustained maximal voluntary contraction or locomotion at maximal speed. This could not be measured directly but was calculated from the surface EMG and the firing rate of recruited muscle fibres in the follow-

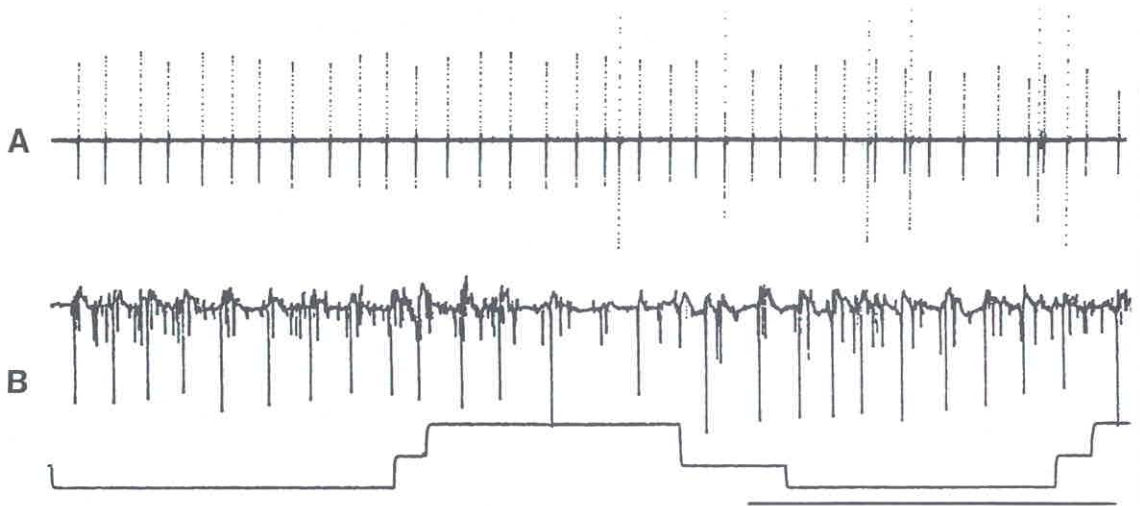


Fig. 1. Paralysed TA. (A) Simultaneous recording of two MUs (low and high amplitude potentials, respectively) during maximal voluntary effort. (B) Upper trace shows a recording of one MU (high amplitude potential) during the highest speed of walking possible. Lower trace shows signals from

foot switches: small upward deflexion denotes pressure on a switch at the toes, intermediate deflexion pressure on a switch at the heel, large deflexion pressure on both switches. Time bar 1 s.

ing way. The amplification of the surface EMG was adjusted so that the potential evoked by a supramaximal electrical shock to the peroneal nerve had the same size in all subjects. We then assumed that individual differences in surface EMG were due to differences in recruitment of muscle fibres and differences of their firing rate. The surface EMG recorded when all fibres fired at 25–30 Hz in normal subjects was used as reference (100% surface EMG = 100% of the muscle fibres firing at 27.5 Hz).

Most of our paralysed subjects reached only 25% of the reference surface EMG during the activities of daily living studied. Theoretically this might mean that 25% of the muscle fibres fired at 25–30 Hz or that 100% fired at 6–7 Hz. The actual firing rate was, however, 11 Hz suggesting that about 60% were recruited. We do not claim that the remaining 40% were never recruited only that they were not tonically active during the activities studied.

Normally all MUs were recruited during rapid locomotion and strong voluntary effort and then at high rates. It seems reasonable to assume that the disuse of 40% of the MUs in hemiparesis is the cause of the increase of type 2 fibres from about 20% to about 40%.

For optimal effect of the low firing rates in hemiparesis the contraction time of the muscle fibres should be as long as possible. The transition of slow to

fast myosin due to low amount of nervous activity must be a disadvantage for power production.

The 60% tonically active muscle fibres remained type 1. Their maximal firing rate was, however, no more than two thirds of that in normal muscles. Only partial fusion would be achieved if their contractile properties were unchanged. Bigland-Ritchie et al. (2) recently reported, however, that very low motoneuron firing rates were sufficient for full fused tension in chronic non-paralysed multiple sclerosis. They suggested that an abnormally low firing rate of the upper motoneuron in this demyelinating central nervous disorders resulted in an abnormally low firing rate of the lower motoneuron as well and in the long run, in a slowing of the contractile properties of the muscle fibres. We found that the mean firing rate of MUs recruited at 25% of the maximal surface EMG was lower in paralysed than in normal subjects indicating that a larger proportion of the MUs fired at low rates in the pathological state. Some intermediate threshold MUs normally responding mainly to relatively strong drive and then at relatively high rates seemed to adapt to the loss of drive by lowering their threshold and firing rate. This is in accordance with some previous findings (17, 20, 21).

In previously studied chronic lower motoneuron disorders the findings were the opposite, i.e. the motoneuron firing rates were abnormally high and the

proportion of type 2 fibres abnormally low (3, 4). This suggests that in the lower motoneuron patients, maximal power is easily reached but regulation of submaximal power is impaired.

Our findings are compatible with experiments in intact animals indicating that the amount of stimulation is more important than the rate of stimulation for muscle fibre properties (6, 13, 16, 19). Consequently, discrepancies may appear between the firing rate of a motoneuron and the stimulation rate optimal for its muscle fibres.

To conclude, in upper motoneuron disorders there was a discrepancy between the abnormally low firing rate of the motoneurons and the abnormally low proportion of muscle fibers expressing slow myosin isoforms in contrast to the lower motoneuron disorder where there was a discrepancy between the abnormally high firing rate of the motoneurons and the abnormally high proportion of fibres expressing slow myosin isoforms in the muscle fibres. These opposite problems in upper and lower motoneuron disorders should be considered in the training programs.

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