Correlation Between Recruitment Order of Motor Units and Muscle Atrophy Pattern in Upper Motoneurone Lesion: Significance of Spasticity

Human muscle fibres are of two main types; type I with a low content of myofibrillar ATPase, type II with a high content of this enzyme[1]. To conform with animals, type I human muscle fibres must have a long contraction time and type II fibres a short contraction time. We have studied the order of recruitment of motor units electromyographically in subjects with upper motoneurone lesions and correlated the findings with a histochemical classification of the atrophic muscle fibres with the aim to evaluate the significance of spasticity for the use of different types of motor units.

The order of recruitment of motor units differs in tonic and in phasic voluntary activity in normal man[2], as well as in tonic and in phasic exteroceptive reflex activity in 'spinal' man[3]. In tonic activity, motor units which have a low maximum frequency are always recruited before motor units which attain higher discharge frequencies. In phasic activity, however, 'high frequency' motor units may be first recruited[4].

The difference in recruitment order, however, disappears on strong pre-existing facilitation of the motoneurone pool[3,5], and the recruitment order of tonic activity is then used also in phasic activity. The significance of tonic spasticity for recruitment order flexibility in a paralytic muscle is demonstrated in the Figure. The level of tonic spasticity is not constant and its role for the recruitment order can thus be studied in experimental situations.

The level of tonic spasticity does not usually influence the recruitment order in tonic exteroceptive reflex activity. The first unit recruited attains regular discharge intervals below the frequency of 10 sec and they should be of low frequency type (Figure A).

When the level of tonic spasticity is so high that sustained electromyographic activity appears already at minimal passive stretch of the muscle, the first unit in tonic activity is also the first unit in phasic activity (Figure B) and the second unit in tonic activity also the second unit in phasic activity. Thus, strong tonic spasticity causes a stereotyped recruitment order and only low frequency units are available as low threshold units.

When after a period of rest tonic spasticity is less marked, a different unit is first recruited in phasic than in tonic activity (Figure C). The first unit in phasic activity usually discharges only once, but on strong stimuli it may repeat in short bursts (Figure D). Its discharge frequency is then higher than the discharge frequency of the first unit in tonic activity. In the Figure the first unit in tonic activity discharges at a frequency of 6/sec, but the first unit in phasic activity at intervals corresponding to a frequency of more than 50/sec. The recruitment order in non-spastic paralysis is thus flexible and both low and high frequency units are available as low threshold units.

The staining method for myofibrillar ATPase by PADIKULA and HERMAN[6] separates the muscle fibres of extremity muscle in man into fibres with a low stainability known as 'type I' and fibres with a high stainability 'type II'[1].

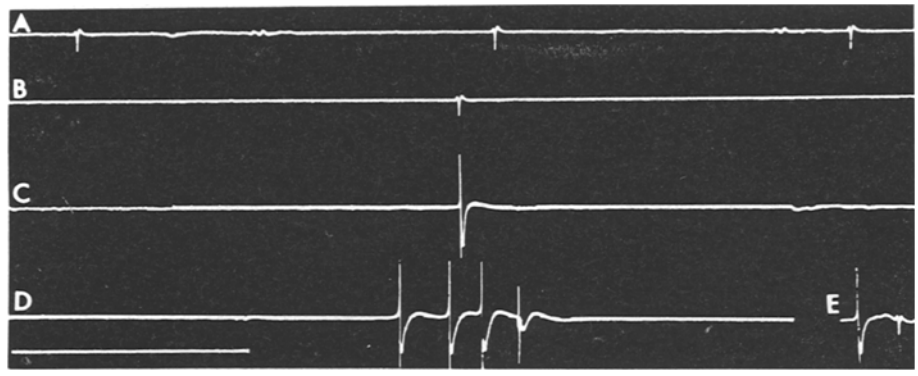
The size of muscle fibres decreases after long-lasting inactivity[7]. One way to quantitate the degree of atrophy in muscle biopsy specimens is to estimate the cross-sectional area of fibres[8] and determine the frequency of fibres less than 1500 μm^2. In normal human muscles the frequency of such small fibres does not exceed 2%[9]. In this study, the frequency of atrophic fibres is determined in muscles with strong and in muscles with weak tonic spasticity.

Case 1–13 in the Table are biopsies from muscles with great tendency to tonic spasticity. Most of the specimens are from antigravity muscles in the paretic limbs of patients with cerebral hemiparesis. It is clinically well known that these muscles tend to be continuously spastic after upper motoneurone lesions.

Case 14–25 in the Table shows the occurrence of atrophic fibres in biopsy specimens from paretic muscles with weak tendency to tonic spasticity. The specimens are obtained from muscles in 'spinal man' (case 14–22) or from antagonists to antigravity muscles in 'hemiplegic man' (case 23 and 24). It is clinically well known that the level of tonic spasticity is lower in these muscles than in the antigravity muscles in hemiplegics. There are, however, hemiplegic limbs in which spasticity never develops even in antigravity muscles. One biopsy from such a muscle is also included (case 25).

Specimen	Muscle	Fibres (%) Type I	≤ 1500 μm^2 Type II
1.	biceps br.	0	2
2.	biceps br.	1	5
3.	biceps br.	0	8
4.	biceps br.	1	33
5.	biceps br.	1	12
6.	biceps br.	19	95
7.	biceps br.	3	22
8.	flex. carp. uln.	3	40
9.	flex. carp. uln.	0	12
10.	flex. carp. uln.	0	29
11.	flex. carp. uln.	1	35
12.	vastus lat.	1	44
13.	vastus lat.	2	23
14.	tib. ant.	2	0
15.	tib. ant.	26	5
16.	tib. ant.	30	42
17.	tib. ant.	19	33
18.	tib. ant.	40	35
19.	vastus lat.	13	13
20.	vastus lat.	33	9
21.	vastus lat.	45	32
22.	vastus lat.	40	52
23.	tib. ant.	10	4
24.	tib. ant.	6	2
25.	flex. carp. uln.	55	75

[1] G. M. FENICHEL and W. K. ENGEL, Neurology *13*, 1059 (1963).
[2] L. GRIMBY and J. HANNERZ, J. Neurol. Neurosurg. Psychiat. *31*, 565 (1968).
[3] L. GRIMBY and J. HANNERZ, J. Neurol. Neurosurg. Psychiat. *33*, 562 (1970).
[4] J. HANNERZ, Experientia, *29*, 45 (1973).
[5] J. HANNERZ and L. GRIMBY, J. Neurol. Neurourg. Psychiat., in press (1972).
[6] H. A. PADYKULA and E. HERMAN, J. Histochem. Cytochem. *3*, 170 (1955).
[7] R. W. ADAMS, D. DENNY-BROWN and C. M. PEARSON, *Diseases of Muscle* (Harper and Brothers, London 1965), p. 177.
[8] L. EDSTRÖM and K. TORLEGÅRD, Z. wiss. Mikrosk. *69*, 166 (1969).
[9] L. EDSTRÖM, J. neurol. Sci. *11*, 537 (1970).



Influence of the level of tonic spasticity on the recruitment order of motor units in the exteroceptive reflex. Recordings from the soleus muscle. Reflexes obtained by skin stimuli on the heel. A: Tonic activity. B: Phasic activity on strong tonic spasticity. C and D: Phasic activity on weak tonic spasticity. E: The first recruited motor unit potential in phasic and the first recruited motor unit potential in tonic activity discharging together, proving that they belong to different motor units. Time bar 100 msec.

Muscles with strong tonic spasticity have an almost exclusive type II fibre atrophy. Only in one case with atrophy of nearly all type II fibres is a significant fraction of the type I fibres also affected (case 6). This selective atrophy of type II muscle fibres has earlier been described both from upper motoneurone lesions and parkinsonism[9,10].

In the muscles with weak tonic spasticity, a significant atrophy is found both within the type I and type II fibre group and there is no evidence for a selective involvement of one of the fibres types.

The present electromyographic study of paralytic muscles indicates that strong tonic spasticity causes a stereotyped order of recruitment of motor units, low frequency units being recruited before high frequency units irrespective of the mode of activation. This is in agreement with findings in decerebrate rigidity in cat, that small tonic motoneurones are always recruited before large phasic ones[11]. As maximal contractions hardly occur in paralytic muscles, some high frequency units should be continuously inactive.

The present histochemical study of paralytic muscles shows a selective type II muscle fibre atrophy in muscles with strong tonic spasticity but a non-selective atrophy in muscles with weak tonic spasticity. This difference in atrophy pattern can hardly be due to any other factor than tonic spasticity itself. We assume that the selective atrophy of type II muscle fibres is due to selective disuse of high frequency units discussed above.

The results also support the hypothesis that slow twitch units tend to discharge at a lower and fast twitch units at a higher frequency.

Zusammenfassung. Bei einer Läsion der zentralen motorischen Bahnen hat die tonische Spastizität eine stereotype Rekrutierungsordnung der motorischen Einheiten und eine selektive Typ II-Faser-Atrophie zur Folge.

L. Edström, L. Grimby and J. Hannerz

Dept. of Neurology, Karolinska sjukhuset, Fack, S–10401 Stockholm 60 (Sweden), 17 November 1972.

[10] L. Edström, Experientia *24*, 916 (1968).
[11] E. Henneman, G. Somjen and D. O. Carpenter, J. Neurophysiol. *28*, 560 (1965).

Hippocampal Lesions Produced by Prolonged Seizures in Paralyzed Artificially Ventilated Baboons

Patients having suffered from epilepsy often show hippocampal lesions described as Ammon's horn sclerosis[1,2] or mesial temporal sclerosis[3]. Such lesions resemble those seen after episodes of cerebral anoxia or vascular insufficiency, and it has been suggested that in epileptics they are commonly the result of systemic or local hypoxia occurring during generalised seizure activity[2,4,5]. Experimental studies in cats employing focal injections of alumina cream or tungsten have emphasised the possible role of local vascular factors and of local oedema[6,7]. Meldrum and Brierley[8] have recently shown in adolescent baboons that seizures (induced by bicuculline) lasting 82 to 300 min produce ischaemic cell change selectively in some neurones of the cortex, cerebellum and hippocampus. These animals showed severe systemic disturbances during the seizure (Meldrum and Horton[9]), but it was not possible to evaluate separately the contributions of the different systemic changes and the cerebral discharges themselves. We have therefore produced prolonged convulsions in adolescent baboons that are paralysed and artificially ventilated so that any contribu-

[1] W. Spielmeyer, Arch. Neurol. Psychiat. *23*, 869 (1930).
[2] W. Scholz, Epilepsia *1*, 36 (1959).
[3] M. A. Falconer, E. A. Serafetinides and J. A. Corsellis, Arch. Neurol. *10*, 233 (1964).
[4] C. Ounsted, J. Lindsay and R. M. Norman, *Biological Factors in Temporal Lobe Epilepsy*. Clinics in developmental medicine 22 (Spastics Society, London 1966), vol. 22.
[5] M. A. Falconer, Epilepsia *12*, 13 (1971).
[6] H. Gastaut, R. Naquet, A. Meyer, J. B. Cavanagh and E. Beck, J. Neuropath. exp. Neurol. *18*, 270 (1959).
[7] R. Naquet, P. Tiberin, M. Toga et R. Vigouroux, in *Physiologie de l'Hippocampe* (Ed. C.N.R.S., Paris 1962), p. 389.
[8] B. S. Meldrum and J. B. Brierley, Arch. Neurol. *28*, 10 (1973).
[9] B. S. Meldrum and R. W. Horton, Arch. Neurol., *28*, 1 (1973).