

Altered motor unit architecture in hemiparetic patients. A single fibre EMG study

Sir: The electrophysiological features and pathogenesis of hemiplegic muscle atrophy remain unexplained. Electrophysiological¹⁻¹⁰ and morphological studies¹¹⁻¹⁵ have suggested a transneuronal degeneration of corticospinal fibres. The presumption that altered motor unit architecture could be reflected in single fibre electromyography (SFEMG) is the subject of this letter.

Twenty hemiparetic patients, 10 males and 10 females, aged 14 to 80 years were studied. In 16 cases the cause of the hemiplegia was a cerebrovascular lesion, in two a glioma, and head injury in two. The hemiplegia at time of electrophysiological investigation had been present from 2 months to 8 years. Mild muscle wasting was evident in the most severe cases. All but one patient had recovered movements in the affected lower limb and were able to walk. All the cases had at least a slight control of the extensor digitorum communis muscle. Causes of peripheral neuropathy were excluded, and the neurological examination in the opposite non-paretic limbs was normal. Single fibre EMG was performed in both extensor digitorum communis muscles. Medelec MS6 equipment was used. The fibre density of the motor unit¹⁶ within uptake area of the special electrode (SF25) was calculated for 20 random positions. The jitter interval was calculated manually (R-10)^{17,18} in some complex potentials, but only in the few patients in which a smooth activation of the paretic extensor digitorum communis was possible. Motor and sensory conduction velocities were measured according to conventional methods.

In the unaffected extensor digitorum communis the mean motor unit fibre density ranged from 1.40 (in a 40-yr-old patient) to 1.80 (70-yr-old patient). In the affected muscles the fibre density ranged from 1.70 (74-yr-old patient after two months of hemiparesis and with a good voluntary control in lower and upper limbs) to 2.50 (59-yr-old patient after 6 months of hemiparesis and with mild muscle wasting). In the 20 paretic limbs the mean value of the motor unit fibre density was 1.97 fibres per recording area (SD = 0.24), and in unaffected limbs was 1.55 (SD = 0.11). The difference between means was statistically significant ($p < 0.001$) (fig). Fibre density was not correlated with duration of the disease ($p > 0.2$) (fig). Jitter was determined in the

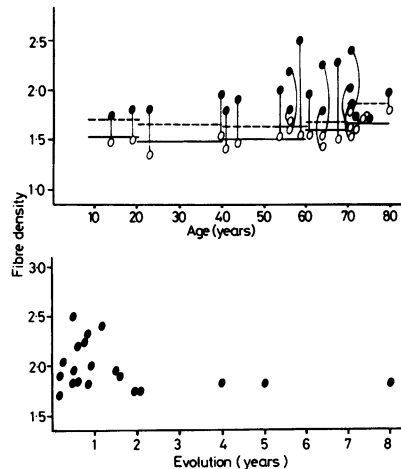


Fig Upper—Fibre density mean value (full line) and upper normal limit (interrupted line) in control subjects of different ages. Fibre density of the motor unit is less in unaffected (○) than in affected (●) extensor digitorum communis of 20 hemiparetic patients ($p < 0.001$).

Lower—The same results of motor unit fibre density in hemiparetic patients according to the time following onset of the illness. In paretic limbs no ratio between the increase in fibre density and time after hemiplegia was found ($p > 0.2$).

affected extensor digitorum communis only in a few patients. In patients with disease for longer than 10 months, none of the potentials showed increased jitter. In patients with disease for less than 9 months, some potentials presented increased jitter and occasional intermittent impulse blocking. Motor and sensory conduction velocities were within the normal range.

Muscle wasting in hemiplegia is well documented,^{21,22} and causes such as disuse, pressure or traction neuropathies, vasomotor changes, and loss of upper motoneuron trophic influence have been proposed.^{9,14} Abnormalities of motor end-plates, simple atrophy and selective atrophy of type II fibres have been described.¹¹⁻¹⁴ The observation of altered pattern of the terminal innervation is the most striking morphological finding to correlate with our study. Axonal sprouting with formation of very complex end-plates,²³ small angular fibres, fibre-type grouping, and grouped atrophy¹⁵ have been found in muscle biopsy of paretic limbs. Single fibre EMG can provide early infor-

mation about the pathology and architecture of the motor unit. The fibre density of the motor unit measures the average number of fibres within the uptake area of the electrode, and expresses the arrangement of the motor unit fibres.^{16,18} In the present study, the motor unit fibre density was mildly increased in the paretic upper limb of hemiparetic patients, compared with the unaffected extremity. These results suggest a rearrangement of the muscle fibres within the motor unit. Early plurisegmentary fibrillation potentials recorded in upper and lower limbs after 2 to 3 weeks following the onset of hemiplegia,³ and increased fibre density, do not suggest disuse as the cause of the electrophysiological findings. The absence of nerve conduction alterations and the normal amplitude of sensory evoked potentials are points against peripheral nerve or brachial plexus lesions suggested in other papers.^{19,20} Our previous EMG studies³ in a series of 87 patients support the hypothesis of trans-synaptic changes in lower motoneurons following degeneration of corticospinal tracts.⁹

Increased fibre density constitutes a sensitive index of collateral reinnervation. It is found very early after denervation in neuropathies, and can be observed before fibre type grouping in histochemical studies.¹⁸ In our patients, increased fibre density was found in the first 2 months, but was not correlated with the time following the onset of the illness nor the age of the patients. The logical progression should be: spontaneous activity after 2-3 weeks,³ axonal sprouting,²³ collateral reinnervation, increased fibre density with possible increased jitter, and increased size of the motor unit^{9,15} with disappearance of the spontaneous activity.³ In fact, spontaneous activity was rarely seen after 6-12 months,^{3,10} and increased size of the motor unit was observed some months following the onset of the hemiplegia.⁹

In some patients, jitter is difficult to study owing to the difficulty in obtaining smooth activation, and the inability of motoneuron to produce approximately constant intervals,²⁴ which is a source of error in jitter measurement.^{17,18} For this reason jitter was only calculated in some potential pairs, in patients with good recovery of movement in upper limb. Increased jitter was observed only in patients within 10 months after onset of hemiplegia, but not in cases of longer standing, who already presented stable neuromuscular transmission. Similar features were previously observed in the

response to repetitive nerve stimulation.^{4,9} The same behaviour is described in non-progressive neuropathies that show increased fibre density and unstable complex in the earlier phases of reinnervation; however stable neuromuscular transmission is observed after some months of evolution.^{18,25}

Single fibre EMG study in hemiparetics, not previously referred to in the literature, provides new information on the electrophysiological features of this condition. The early increment of the motor unit fibre density is in accord with other electrophysiological findings,^{3,9} and supports the possible collateral reinnervation suggested by biopsy studies.^{15,23} The architecture of the motor unit is altered in hemiplegia.

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Listeria monocytogenes abscess in the basal ganglia

Sir: Meningitis due to *Listeria monocytogenes* occurs in healthy people as well as in patients with preexisting disease. *Listeria* brainstem encephalitis¹ and two cases of brainstem and spinal cord abscess have been reported in otherwise healthy people.^{2,3} Brain abscess by *Listeria monocytogenes* is a rare occurrence. Five cases have been published to our knowledge.⁴⁻⁸ A sixth patient is presented.

A 45-year-old left handed man received a cadaveric renal transplant at the age of 39 yr. The cause of his renal insufficiency was chronic poststreptococcal glomerulonephritis. Rejection was suppressed by 25 mg azothioprine and 15 mg prednisolone daily. The transplant functioned satisfactory. After two days of headache, drowsiness and weakness of the left arm he was admitted with a temperature of 39.1°C, but no signs of meningitis. Neurologic examination showed mild left hemiparesis and a left extensor plantar response. ESR was 80 mm/h; haemoglobin 6.4 mmol/l (10.3 g/l); leucocyte count normal. The CSF contained 68 polymorphonuclear leucocytes/mm³, protein was 0.69 gram/l, and CSF cultures remained sterile. Blood cultures yielded *Listeria monocytogenes*. CT scan on the day of admission showed a hypodense area in the region of the right basal ganglia with a small central ring (fig A). Initial treatment consisted of iv ampicillin (12 g/24 hr) and 10 mg daily by intrathecal injection; azothioprine was continued. In spite of bactericidal levels of the antibiotic in the CSF, the patient deteriorated. Subsequent CT scans showed an enlarging ring after contrast enhancement (fig B and C). On the 31st day a drain was introduced via a right parietal burrhole. No pus could be aspirated. Postoperatively the hemiparesis became worse, and CT scan showed an unchanged abscess cavity. The azothioprine was discontinued. On the 44th day a drain was inserted through a right frontal burrhole and aimed with a stereotactic device. One ml of turbid fluid was aspirated, which on culture remained sterile. CT scan demonstrates the tip of the drain lying in or near the third ventricle. After the operation high fever and marked nuchal rigidity developed. The CSF cell count was 3000/mm³ with an elevated protein content but culture yielded no growth. Within six days the CSF became clear and nuchal rigidity disappeared. The condition of the patient then improved, and 10 weeks after admission the antibiotic treatment