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Assessment of Stimulated Single-Fiber Electromyography in Paralytic Muscles of Patients with Spinal Cord Injury

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Neuronal dropout accompanied with axonal degeneration may occur below the level of cord lesion after spinal cord injury (SCI). By using a sensitive electrodiagnostic method of stimulated single fiber electromyography (SFEMG), it is possible to assess the functional integrity of neuromuscular transmission in paralytic muscles of SCI. Neuromuscular jitter and fiber density were measured in 21 anterior tibial muscles of 18 patients with SCI and 11 muscles in 11 normal controls. The results showed an increased mean jitter value but without increased fiber density in SCI patients. There is a positive correlation between the increased jitter and the disease duration from the onset of cord lesion to the time of SFEMG test. This provides an electrophysiological evidence of axonal degeneration and dysfunction of neuromuscular transmission below the level of cord lesion in SCI. Also the neural degeneration may become prominent in the later course of the disease.

Key words: Stimulated single fiber electromyography, jitter, fiber density, spinal cord injury

INTRODUCTION

Degeneration of motor neurons occurring in the spinal cord following upper level of cord injury has been approved anatomically [1,2] and electrophysiologically [3-6]. Denervation of the peripheral nerve leading to unstable neuromuscular transmission was recognized while chronic ongoing changes in neurologic examinations as patients progressed from spinal shock to the spastic phase in spinal cord iniury (SCI).

The measurement of neuromuscular jitter by single fiber electromyography (SFEMG) is a sensitive clinical method to assess the functional integrity of neurological function of anterior horn cells caudal to the lesion site and signify the degradation of the neuromuscular function in spinal cord lesion. The originally described SFEMG is based on recording from muscle fibers of voluntarily activated motor units [1]. This however, is not always practical, for example in unco-operative or unconscious patients, young children and even in severely paretic muscles, whether there is an upper motor neuron lesion or weakness due to some peripheral pathology. Electrical stimulation of motor axons has been suggested for such cases and re-

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cently become increasingly used as an alternative way of activation [8-11].

The present study was undertaken by stimulated SFEMG to assess the integrity of the neuromuscular function of the paralytic muscles in patients with SCI.

SUBJECTS AND METHODS

Eighteen patients with SCI between neurological level at C6 and T11 participated in this study. Their age were between 19 and 44 years, mean 30.5 years. There were 13 males and 5 females, all in good health and without evidence of past history of a neurological disease. The duration from onset of the cord injury to the SFEMG examination was 2 to 28 months.

According to Frankel's classification for neurological function in SCI [12],12 patients were categorized as scale A with complete loss of both motor and sensory functions below the segmental level involved. Three patients were categorized as scale B with some sensation present below the level of the lesion, but the motor paralysis was complete at that level. The other 3 patients were scale C with some muscle power present below the lesion, but it was of no practical use to the patient.

Stimulated SFEMG was performed in the anterior tibial muscle of the patients and 11 age-matched normal persons (mean age, 28.1 years). A teflon coated monopolar needle electrode with a bared tip of 1 mm was inserted into the motor point of the muscle, while a SFEMG needle electrode was inserted 2 to 2.5 cm distal to the stimulating electrode. A surface electrode with 1 cm diameter of silver plate was placed 2 cm proximally for reference. It was advanced slowly to locate the stimulated fibers by observing sharp potentials with a rise time less than 300 μs and an amplitude greater than 200 μV. The stimulus frequency was raised from 5 to 10 Hz and the intensity was adjusted to about 10 to 30 V above the threshold for the studied motor unit. Skin temperature measured at knee was between 32° and 35°C.

The recording equipment was a Medelec MS60 electromyograph. The filters were set to 500 Hz for the high pass and 16 KHz for the low pass filter. The mean consecutive difference (MCD) was calculated as the mean of the absolute differences of more than 50 consecutive interpotential intervals between stimuli and single-fiber potentials. The resolution of time measurement is 0.1 μs. We use an MCD limit of 5 μs to identify low jitter which may suffice to recognize direct muscle fiber stimulation and do not rely on visual impression, nor on the size of jitter at threshold stimulaton.

Fiber density was measured as the mean value of the number of spiky components in the single-fiber action potentials recorded at more than 10 sites.

RESULTS

On supraliminal stimulaton, the mean MCD obtained from 21 anterior tibial muscles in 18 SCI patients was 36.4±15.5 (mean ± SD) μs. This is significantly higher than the result of 24.2±8.4 μs obtained from 11 anterior tibial muscles in 11 normal controls (t-test. $p<0.05$). The fiber density of 3.0±0.4 (mean ± SD) measured from anterior tibial muscles in SCI patients was not different to the findings of 2.6±0.3 obtained in normal controls ($p>0.1$).

In patients with SCI, mean MCD value was positively correlated to the disease duration from the onest of iniury to the stimulated SFEMG test. The linear correlation was shown in Figure 1 ($r=0.57$, d.f. = 19, $p<0.01$). The mean MCD values in different groups as presented by the scale of Frankel's functional classification were shown in Table 1. There is no significant difference of mean MCD among these three groups of SCI patients (one-way ANOVA, $p>0.1$).

DISCUSSION

The assessment of SFEMG with measurement

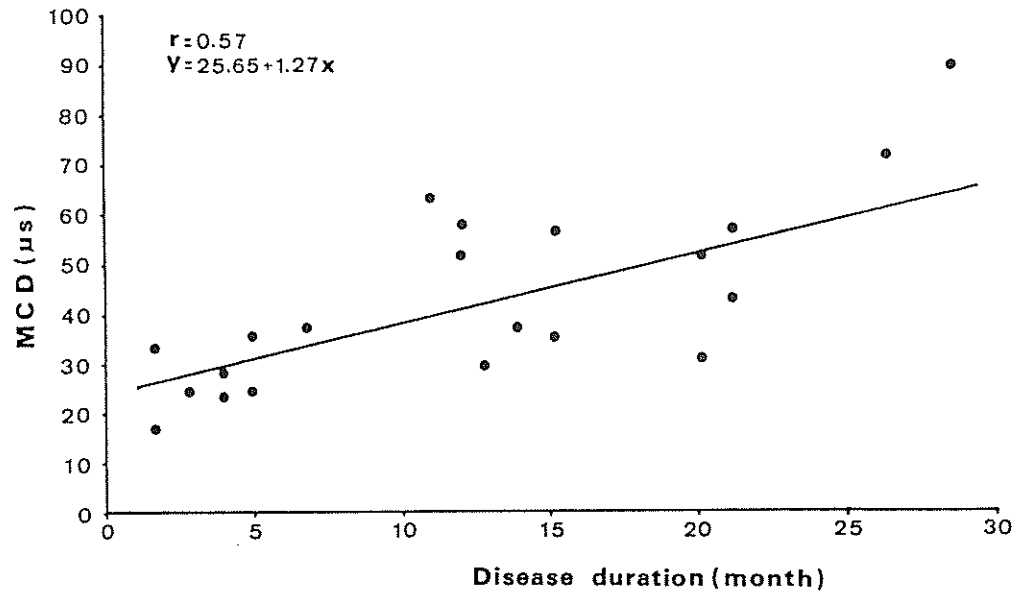

Fig. 1. The positive correlation between MCD (mean consecutive difference) and disease duration from cord injury to the time of test in 21 paralytic anterior tibial muscles of 18 patients with spinal cord injury. (r=0.57, d.f. =19, p<O.01).

Table 1. The mean consecutive difference (MCD) values (mean ± SD) obtained in 18 patients with different scales of Frankel's classiffcation for spinal cord injury.

Frankel's scale [12]	A	B	C
Number of patient	12	3	3
MCD	32.1±19.3	38.4±14.9	35.3±11.7

of neuromuscular jitter is a sensitive clinical method to assess the functional integrity of neuromuscular transmission [7]. Usually the jitter is measured between action potentials of two muscle fibers of the same motor units firing in voluntarily activated muscle. The magnitude of jitter is correlated to the safety factor of neuromuscular transmission [7,13]. Stimulated SFEMG with electrical stimulation of intramuscular axons has been recognized as a practicable method of studying the motor end-plate jitter with the following advantages: 1) the stimulation rate can be controlled, 2) it is applicable in severely paralytic muscles, 3) there is little need for patient cooperaton, 4) it is easy to stimulate the selected axon with a low stimulation intensity, and, 5) it may also be useful in animal studies.

The jitter in muscle fibers measured in our normal controlled group is lower than the published normal material by voluntary activity [9]. The expected jitter on axonal stimulaton should thus be mean MCD obtained by voluntary activity divided by $\sqrt{2}$ [9]. This result was possibly related to the differences of electrical threshold of the individual axons for their respective order of recruitment in the elicited response. The findings are also compatible with our results in control groups by using

stimulated SFEMG and normal data obtained by volitional SFEMG [7]. Voluntary contraction preferentially activates low threshold small motor units, while electrical stimulation most likely activates both small and large motor axons.

In the previously electrophysiological and anatomical studies of SCI, Rosen and his colleagues [3] have found positive sharp waves and fibrillation potentials in 6 of 7 patients studied during spinal shock, which largely resolved after the spastic phase developed. These findings suggested that dropout of anterior horn cell occurred when spinal shock resolved. Aisen et al [6] used a serial EMG studies on paraspinal muscles and lower extremities in 8 young patients commencing 2 to 9 weeks after acute cervical cord injury and found a strong evidence of trans-synaptic distal end dysfunction following SCI. Van Alphen and his colleagues [1] found anterior horn cells loss well below the site of cord injury at postmortem in 3 quadriplegic patients. Brown et al [2] demonstrated that hemisection of the macaque spinal cord in the thoracic region resulted in a 20% loss of anterior horn cells in the lumbosacral cord. These findings indicate that anterior horn cell degeneration may progress after SCI.

In stimulated SFEMG findings, the increased jitter in patients with SCI may signify evolving the unstability of neuromuscular transmission. These findings may correlate to the dropout of motor neurons and their proceeding collateral reinnervation. It rises the possibility that distant neuronal degeneration may occur in upper motor neuron lesion as it provides evidence of reinnervation and reorganization of motor unit during the chronic phase of SCI. The positive correlation between jitter and the disease duration after onset of injury in our study may strongly reflect a prominent evidence of dropout motor neurons with axonal neuropathy in the later course of the disease. Although the long-term follow-up study has not done. the degradation of nerve may persist for several years and then lead to a progressive fade-out of the neuron and the distal nerves.

The fiber density obtained in anterior tibial muscles from SCI patients is somewhat higher than the normal controls, but there is no difference statistically. This is possibly related to a small number of the data base, or its obtaining value is limited in differentiation by supraliminal stimulation with near-threshold stimulation of other axons. The reinnervation process was expected to be less in SCI with some groups of motor neuron degeneration.

To summarise, the stimulated SFEMG by axonal stimulation is a valuable and convenient method to study neuromuscular tranmission and apply in paralytic muscles of SCI patients. The increased mean jitter value suggested an evolving motor neuron degeneration as its providing evidence of reinnervation and reorganizaton of the motor unit during the chronic phase of SCI. Also, the increasing jitter is good correspondence to the longer duration of the disease. This suggests a degradation of neuromuscular transmission may progress in the later course of SCI.

REFERENCES

1. Van Alphen HA, Lammers HJ, Walder HAD: On remarkable reaction of motor neurons of lumbosacral region after traumatic tansection in man. Neurochirurgia 8:328-330, 1962.
2. Brown WF, Milner-Brown HS, Ball M, Girvin JP: Control of the motor cortex on spinal motoneurons in man. In: Desmedt JE, ed. Cerebral Motor Control in Man: Long Loop Mechanisms. Progress in Clinical Neurophysiology, Vol. 4. Basel, Karger 1978, pp. 246-262.
3. Rosen JS, Lerner IM, Rosenthal AM: Electromyography in spinal cord injury. Arch Phys Med Rehabil 50:271-273, 1969.
4. Taylor RG, Kewalramani LS, Fowler WM: Electromyographic findings in lower extremities of patients with high spinal cord injury.

Arch Phys Med Rehabil 55:16-23, 1974.

5. Ludolph AC, Hugon J, Roman GC, Spencer PS, Schoenberg BS: A clinical neurophysiologic study of tropical spastic paraparesis. Muscle Nerve 11:392-397, 1988.
6. Aisen ML, Brown W, Rubin M: Electrophysiologic changes in lumbar spinal cord after cervical cord injury. Neurology 42:623-626, 1992
7. Stalberg E, Trontelj JV: Single Fiber Electromyography. Old Woking, Surrey, UK, Mirvalle Press, 1979, pp. 1-244.
8. Trontelj JV, Stalberg E, Mihelin M, Khuraibet A: Jitter of the stimulated motor axon. Muscle Nerve 15:449-454, 1992.
9. Trontelj JV, Mihelin M, Fernandez JM, Stalberg E: Axonal stimulation for end-plate jitter studies. J Neurol Neurosurg Psychiatry 49:677-685, 1986.
10. Trontelj JV, Stalberg E: Single motor end-plates in myasthenia gravis and LEMS at different firing rates. Muscle Nerve 14:226-232, 1991.
11. Trontelj JV, Khuraibet A, Mihelin M: The jitter in stimulated orbicularis oculi muscle: technique and normal values. J Neurol Neurosurg Psychiatry 51:814-819, 1988.
12. Frankel HL, Hancock DO, Hyslop G, Melzak J, Michaels S, Ungar GH, Vernon JDS, Walsh VJ: The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and quadriplegia, Part 1. Paraplegia 7:179-192, 1969.
13. Sanders DB. Howard JF: AAEM minimonograph #25: single-fiber electromyography in myasthenia gravis. Muscle Nerve 9:809-819, 1986.

脊髓損傷病人麻痺肢之刺激單纖維肌電圖檢查

張權維　連倚南

脊髓損傷後的麻痺肢，因中樞神經之控制失常，可導致受傷部位以下的神經細胞及末梢神經之退化，本研究以18位脊髓損傷病人之麻痺肢脛前肌作刺激單纖維肌電圖檢查，病人中有13位男性，5位女性，年齡由19歲至44歲(平均30.5歲)，受傷部位在第六節頸脊髓至第十一節胸脊髓之間，其受傷至檢查時間在2個月至2年4個月不等，以單極針離記錄電極約2公分作神經軸電刺激，電刺激頻率每秒5至10次，並以肌電圖儀器配合單纖維肌電圖電腦程式記錄其平均肌纖維振顫差異(mean consecutive difference)與肌纖維密度(fiber density)。

結果顯示由脊髓損傷病人21條脛前肌所得平均肌纖維振顫差異為36.4±15.5 μ s，比11位同年齡層正常人11條肌肉所得24.2±8.4 μ 高(t test, $P<0.05$)，而肌纖維密度在脊髓傷病人所得為3.0±0.4，與對照組所得2.6±0.3比較並無明顯差異($P>0.1$)，在脊髓損傷病人的平均肌纖維振顫差異上，其變化與患者受傷至檢查之疾病期間成正相關($r=0.57$, d.f. = 19, $P<0.01$)，但與脊髓損傷病程度，依Frankel分類等級並無明顯相關。此顯示脊髓損傷後，由於中樞神經的控制失常可導致患者麻痺肢的肌肉神經交接處功能失常與退化現象，而受傷越久，其退化可能更明顯。

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