Short report

A method for detecting functional weakness

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SUMMARY A simple electrophysiological method is described for detecting weakness of psychiatric origin. The method involves the interpolation of a single indirect stimulus to a muscle during the course of a supposedly maximal voluntary contraction. Incomplete motor activation is revealed by the presence of a twitch superimposed on the recording of voluntary torque.

One of the most difficult problems that may confront the clinical neurologist is the decision as to whether muscle weakness is "organic" or "functional". During the past two years we have employed a technique originally devised by Denny-Brown¹ to examine motor unit recruitment. The technique was used by Merton² in his classic study of muscle strength and fatigue in the human adductor pollicis but, so far as we are aware, has never been exploited in a clinical situation. The principle of the method is to request the patient to make as strong a contraction as possible and, during this period, to deliver a single maximal stimulus to the appropriate motor nerve. If some motor units have not been recruited, or are firing at suboptimal rates for tension development, they will generate a twitch response which will be superimposed on the voluntary torque recording. On the contrary, if all the motor units are fully activated in the voluntary effort, the interpolated stimulus will not be able to produce any further torque.

Methods

The studies are performed on the dorsiflexors and plantarflexors of the ankle using a leg-holding device described elsewhere;³ this apparatus enables isometric twitch and voluntary torques to be measured for either of the two muscle groups, and at any chosen position of the ankle joint. During plantarflexion the tibial nerve is stimulated in the popliteal fossa through two surface electrodes constructed of aluminium foil; for evaluating dorsiflexion, similar electrodes are placed either over the motor point of

the tibialis anterior or else over the common peroneal nerve at the neck of the fibula. The stimuli are rectangular voltage pulses, 50 μ s or 100 μ s in duration, delivered from a Devices Ltd stimulator (type 3072) which, in turn, is triggered from a digital timing device (Digitimer type 3290, Devices Ltd). The torque developed by the contracting muscle, together with the evoked M-wave, is displayed on a variable persistence storage oscilloscope (Hewlett-Packard, model 141B).

Results

The predictions of the method can be readily tested by asking a normal subject to make voluntary contractions of different intensities (Fig (a)); as the voluntary force increases, the interpolated twitch declines. In the case of the dorsiflexors of the ankle, the complete disappearance of the twitch in virtually all healthy subjects indicates that full activation of motor units is usually achieved.⁴ In about half of normal subjects the plantar muscles cannot be maximally excited but the interpolated twitch is always a small fraction (less than 30%) of the control one.⁴ Consequently, the finding of an interpolated dorsiflexor twitch or of a plantarflexor twitch greater than 30% of its control value, is indicative of abnormally weak motor unit activation. In the absence of clinical evidence of a pyramidal or extrapyramidal disorder, a superimposed twitch can only be due to a lack of voluntary effort. Two brief examples of the usefulness of this technique will be given.

Case 1 Mrs SN, age 52 years, was a geologist who gradually developed weakness of both legs seven weeks prior to hospital admission. The weakness progressed to the stage that she had great difficulty in rising from a chair and could barely climb the stairs. She was not aware of weakness in her arms and she had no numbness or paraesthesiae. On examination, there was marked weakness of all movements

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Fig Responses to single stimuli interpolated (at arrow) during supposedly maximal voluntary contractions of plantarflexor muscles in control subject (a), case 1 (b), and case 2 (c) and of dorsiflexor muscles in case 2 (d). Note, in (a), the progressive reduction in twitch amplitude as the voluntary contractions became stronger, and the retention of large twitches in (b), (c), and (d). Horizontal calibrations, 2s; vertical calibrations, 30 Nm (a), 10 Nm (b), 5 Nm (c), and 4 Nm (d). Record (c) retouched.

of the hips and knees (MRC grade 3) and slight weakness of her ankle and shoulder muscles. Tendon reflexes were normal in her arms and diminished in her legs; the plantar responses were flexor. No sensory loss could be demonstrated but she displayed Rombergism. On investigation her sedimentation rate was 17 mm/hr, CK 27 μ /l (normal less than 120 μ /l), aldolase 4 μ /l (normal up to 6 μ /l) and an EMG failed to show any evidence of sensory or motor denervation or of defective neuromuscular transmission; similarly, there were no myopathic features. Cortical and subcortical responses evoked by visual and auditory stimulation were also recorded and were normal; somatosensory potentials evoked by stimulation of the peroneal nerves were also normal (Dr ARM Upton). Needle muscle biopsies were performed on the right deltoid and lateral vastus muscles and were stained for myosin ATPase and NADH activity, as well as for glycogen and lipid; none of these procedures, nor staining with haematoxylin and eosin and with the Gomori trichrome method, revealed any abnormalities. Isometric strength testing was performed on the right leg after the first EMG and before the results of the muscle enzyme titres and biopsy were available. It revealed

normal twitch torques for the tibialis anterior (dorsiflexor) and plantarflexor muscles (1.2 N.m and 14 N.m respectively). More informative still, however, were the results of maximal stimuli interpolated during maximum voluntary plantarflexion. Such stimuli evoked twitches which were larger than the maximum voluntary torque (Fig (b)). These results indicated that her weakness was of functional origin; a psychiatric interview disclosed anxiety surrounding an extra-marital relationship. The patient was informed of the nature of her weakness and was discharged from hospital; she recovered full strength in her legs within a few days.

Case 2 Mr ND, age 58 years, had fallen 3m from a ladder while working at a steel mill 5 years previously. Immediately after the accident the left ankle began to swell and became extremely painful, but no fracture was revealed by radiography. Since then the ankle had apparently remained painful and the patient also complained of pain in his lower back, together with weakness of the left leg. On examination there was no muscle wasting and the knee and ankle jerks were present and symmetrical on the two sides; the plantar responses were flexor. There was a moderately severe (MRC grade 3) weakness of all movements of the toes and ankle in the left leg; the patient did not, however, walk with a foot drop. He had been unable to work because of his symptoms and was receiving compensation. Two EMG examinations (October 1979 and January 1982) revealed a moderately reduced motor unit count in the left extensor digitorum brevis muscle with normal values in the left plantar muscles and in the right extensor digitorum brevis; the left sural nerve response was normal. Reduced interference patterns were recorded from the left tibialis anterior, medial gastrocnemius, and medial vastus muscles, without other abnormalities being present. On the basis of the reduced left extensor digitorum brevis motor unit count, it seemed probable that he had an L5 lesion; however, the weakness of his left leg was judged to be out of all proportion to the extensor digitorum brevis deficit and a "functional" component was suspected. Isometric strength testing was therefore performed. Although the plantarflexor and tibialis anterior twitch torques were normal (21.5 Nm and 3.4 Nm respectively) the maximum voluntary torques were very reduced (5 Nm and 2.5 Nm respectively). When stimuli were interpolated in the course of maximal plantarflexion and dorsiflexion, large twitches were superimposed (Fig (c,d)), indicating very poor motor unit excitation and confirming the presence of a large functional component in his symptoms.

Discussion

These two examples show the value of the interpolated stimulus technique in assessing psychiatric factors in muscle weakness. In our experience the technique is most useful in those patients in whom the diagnosis rests between a lesion of the motor unit and a functional syndrome. Patients with upper motoneuron or extrapyramidal disorders can usually be recognised as such on clinical grounds alone, but the technique could have a useful role in quantitat-

ing the severity of the muscle weakness. We have considered the possibility that misleading results could be obtained due to such factors as pain, anxiety, inattention, poor comprehension and weakness of muscles other than those being tested. The isometric nature of the muscle contractions eliminates pain associated with joint movement, while anxiety is more likely to enhance motor unit recruitment than hinder it. Even inattentive subjects should be able to manage the few seconds required for the voluntary task while a lack of understanding has not proved to be a problem in well over 100 normal subjects, aged 10–82 years. Finally, muscles other than the dorsiflexors and plantarflexors would not contribute to the maximum voluntary torque because of the firm fixation of the leg at the knee and ankle. In any case, the method is a test of the muscle stimulated rather than all the muscles which might participate in an intended movement; thus we have sometimes stimulated tibialis anterior alone, as in cases 1 and 2, rather than all the dorsiflexors of the foot. In promoting this technique, we wish to

emphasise its ease of performance and, in comparison with tetanic stimulation, its relatively painless nature.

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