SPINAL CORD STIMULATION

AND

MOTOR UNIT ACTION POTENTIALS

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Summary

Electrical stimulation of spinal cord has produced improvement in some clinical aspects of patients with multiple sclerosis and other central nervous system disorders. The exact mode of action of spinal cord stimulation is not understood. In this study the effect of electrical stimulation of the spinal cord on the recruitment of motor unit action potentials in ten patients with spastic and flaccid weakness has been evaluated and the possible mechanism of action of spinal cord stimulation is discussed.

Electrical stimulation of the thoracic spinal cord has been reported to improve motor control in patients with multiple sclerosis. (1) Technical aspects of the procedures had been reported by Cook and Illis, et al (1.2). Basically the spinal cord is stimulated with rectangular pulses of 200 microseconds duration at frequency about 33-50 HZ and adjustable voltage. Similar techniques had been used for control of intractable pain (3.4). The purpose of this study is to evaluate the effect of stimulation of the dorsal spinal cord on motor neuron discharges participating in the activation of skeletal muscles during voluntary muscle contractions.

Method and Materials

Ten patients were studied, all of whom were selected according to strict criteria (1). Eight of the patients were clinically diagnosed as having multiple sclerosis; one patient had poliomyelitis 17 years prior to the admission for application of posterior column stimulation; and one patient had both poliomyelitis and multiple sclerosis for several years. All patients were alert and fully cooperative. The purpose of examination was fully explained to each patient, who was then examined in supine position lying on a bed in a warm room. Motor and sensory nerve conduction were recorded in median, ulnar, peroneal, posterior tibial and sural nerves under standard techniques (5.6). The weakest muscles were selected for electromyographic study (0 to 3+ on 0 to 5+ scale). Standard monopolar needle electrodes 37 to 55 mm. in length (Teca MG 37, MG 50)* were used. Two to four needles were simultaneously inserted into the different parts of a single muscle. Care was taken that the needle electrode and the position of the extremity under evaluation remained unchanged throughout recording. A Teca TE4 apparatus was used. The output was amplified and displayed on the single or double beam oscilloscope and recorded on a photo-sensitive Kodak direct printing paper, type 1895. Stimulation of the spinal cord was discontinued 24 to 36 hours prior to each examination. Muscles were sampled before and after stimulator was turned on. Because of severe weakness, muscles were sampled only during maximum voluntary effort. All muscles were used as a prime mover during the entire procedure.

Results

None of the patients showed abnormally slow conduction. Patients were initially evaluated with the stimulator off. In patients with spastic weakness one or sometimes a few motor units could be found firing regularly at a slow rate before muscles were fatigued. The patient with old poliomyelitis showed typical so called "neuropathic pattern" with hypertrophic motor units, decreased interference pattern and occasional fibrillation and positive waves. No spontaneous activity at rest was noted in sampling of the other patients' muscles, including the patient with both conditions (poliomyelitis and multiple sclerosis). In some cases although the willed effort was maintained after a short period, depending on the patients' clinical state and the muscle power, a gradual decline in frequency and finally cessation of discharge of the units was noted (fatigue). When the stimulation was turned on, some of the spastic patients felt a sense of well-being. Electromyography repeated ten to fifteen minutes later showed clearly more prolonged electrical discharges in three cases, accompanied by more prolonged sustained contraction. Fig. 1. In two of these cases, also, an increase in the number of motor units accompanied by clinical improvement in motor functions, was noted each time the stimulator was turned on. These changes were not present in all the muscles in the same patient or in even different portions of the same muscle. A sample of vastus lateralis muscle of case number one is illustrated before and after the stimulation was turned on. Fig. II. Simultaneous recording from the vastus medialis of the same patient showed no change during the entire procedure. In the remaining patients there was no appreciable change in the rate of firing or in the shape of the motor units. The amplitude of the motor units was constant except for occasional minor fluctuations attributed to accidental needle movement during the contraction of the muscles. In one patient when the stimulator was turned off (case number 5) several samplings of the left anterior tibialis muscles produced absolute silence during maximal voluntary effort and no contraction was observed clinically. With the stimulation on, two units were recorded firing regularly and slowly, one from the depth and one from the surface of the muscle. However, because just before the turning on of the stimulation the needle electrodes were moved from the original position, the record was interpreted as invalid. The patient with old poliomyelitis showed no clinical or electromyographic change when the stimulator was activated. The patient with both conditions (multiple sclerosis, old poliomyelitis) responded to stimulation by subjective feelings of improvement but his weakness remained unchanged; simultaneously, in some muscles, both an increase in the number of firing units and a delay of the muscle fatigue which was manifested by more prolonged firing time of the units was noted. Fig. III.

Discussion

By definition a motor unit includes an anterior horn cell, its axon, neuromuscular junction and all the muscle fibers on which terminal fibers of the axon ends. When impulses reach the anterior horn cell and muscle fibers the motor unit fires, the response being a brief twitch of the muscle accompanied by electrical discharges called motor action potentials. The term recruitment is defined as an increase of motor neuron discharges associated with increase of the level of effort during reflex or voluntary contractions either as a result of increasing the firing rate of the individual motor units or an increase in the number of the simultaneously activated units. In general, with minimal effort, a primary unit begins firing at a lower limiting frequency and then increases to a higher level when recruitment of the second unit occurs. During the muscle activation, as the level of effort increases the number of the motoneurons firing also increases either as a result of an increased firing rate of individual motoneurons or an increased number of motoneurons being activated. There are some differences in recruitment at low and high levels of effort (7,8,9). The firing frequencies and recruitment patterns of motor units vary also with differences in size, prime function and diverse actions by individual muscles (9). Whether the contraction is isometric or isotonic is also important in regard to which motoneuron participates in the contraction (10,11).

In multiple sclerosis focal demyelination and its pathophysiological effect interferes with conduction in the demyelinated fibers (12), or a defect in the transmission (13) slows the conduction at the synaptic regions. Whatever the cause of slowing or blocking of conduction in the upper motor neuron fibers, the end result is a delay or permanent blocking of impulses before reaching their final destination, the anterior horn cells, resulting in clinical weakness and in defective firing and recruitment, with the appearance of the characteristic interference pattern on electromyography. The order of recruitment of the motor units was not studied in these patients (a study regarding this is in progress). This study has demonstrated a decrease in fatigueability with faster, more sustained firing of functional motor units; as well as augmented recruitment of silent motor units, in some patients, as a result of electrical stimulation of the spinal cord. At the present time there is no clear explanation for the mechanism of action of spinal cord stimulation. The fact that the patient with pure lower motor neuron disease (poliomyelitis) had no improvement suggests that the observed results of spinal stimulation are not due to a placebo effect. Induced ionic changes in the cerebro-spinal fluid and/or extracellular fluid might be of great significance since conduction velocity in the demyelinated fibers could change with alterations of their ionic environment (15). However, stimulation of the spinal cord had been of

no benefit in complete cord transection (14) suggesting that continuity between some fibers of the spinal cord is necessary. Increase in the number of motor units and decrease in fatigueability of the units might be due to the use of alternate pathways (2), alteration in the conduction velocities through the spinal cord, or lowering the threshold of the motoneuron pool, so that some faulty conduction mechanism is now able to fire the motoneurons. Whatever the action mechanism of spinal stimulation there remain many questions which need further evaluation and studies. It was not surprising that recruitment patterns were often dissimilar in single and in different muscles since their component fibers probably are not affected to the same degree either by the demyelinating process or by the stimulator. As a result the expression of cortical control over anterior horn cells (16) would necessarily be affected in same measure as well.

Footnote

* Teca Corporation, White Plains, New York.

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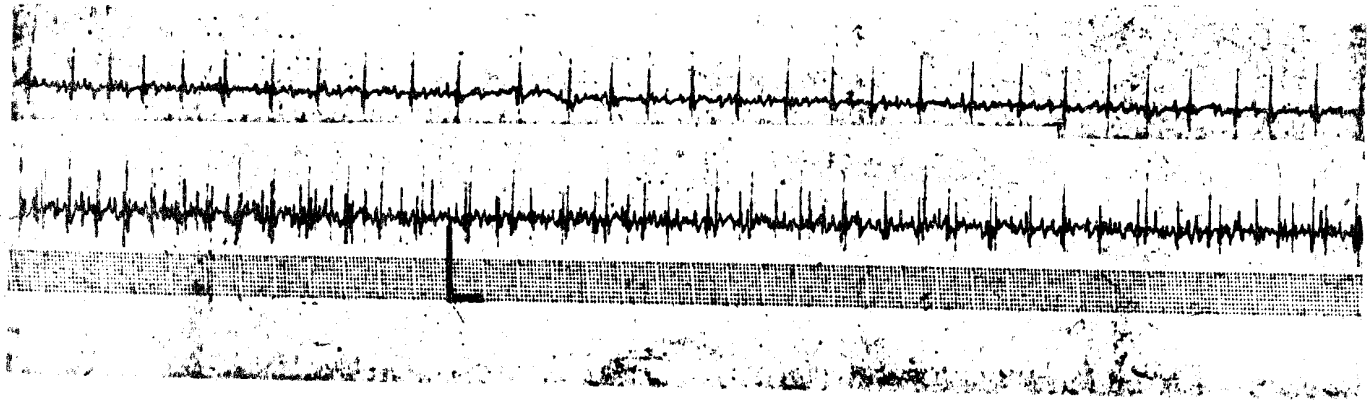

Fig. 1 Left anterior tibialis muscle.
Note more prolonged and sustained firing of motor units (lower tracing): after stimulator was turned on when compared to upper tracing, stimulator off.
Calibration 100 msec. and 1 mv.

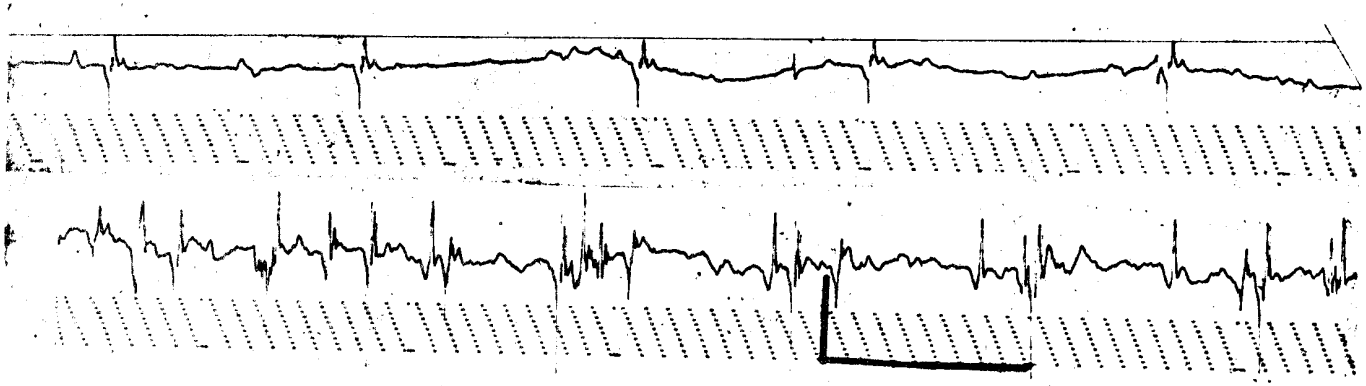

Fig. II Left vastus lateralis muscle.
Note clear increase in the motor unit number (lower tracing) after stimulator was turned on, when compared to the upper tracing (stimulator off).
Calibration 100 msec. and 1 mv.

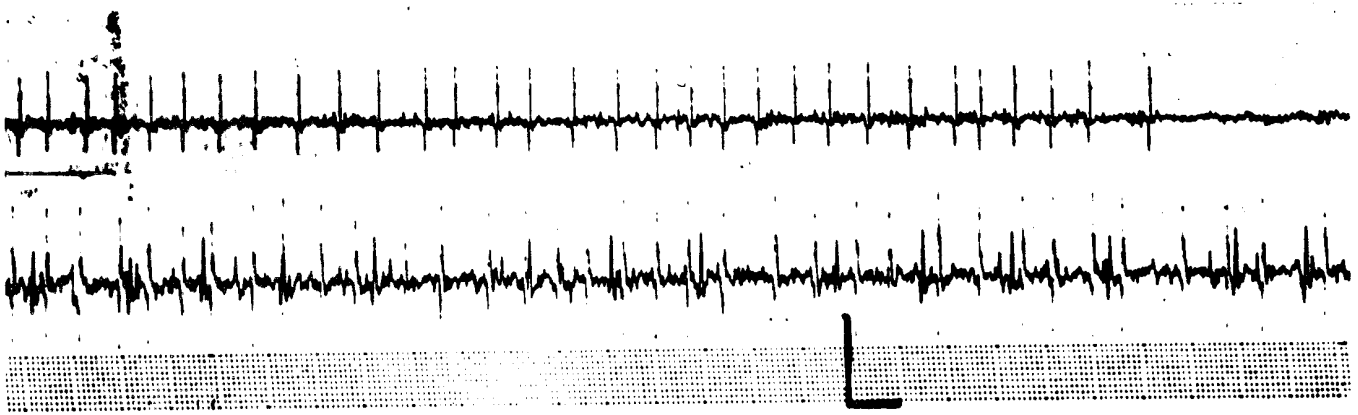

Fig. III Left anterior tibialis muscle.
Note increase in the number of firing units and delay of muscle fatigue (lower tracing end of 60 sec. contraction when compared to upper tracing (end of 15-20 sec. contraction).
Calibration 100 msec. and 1 mv.