

EVALUATION OF ELECTRICAL STIMULATION AS A TREATMENT FOR THE REDUCTION OF SPASTICITY^a

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The purpose of this study is to examine ways in which electrical stimulation, both transcutaneously and via implanted stimulators, may be used to treat spasticity. Spasticity is one of the complications which frequently follows CNS (central nervous system) lesion, and it is commonly identified in the following ways: The involved muscles develop a pathological resistance to passive stretch. This resistance increases with the rate of stretch and also exhibits the "knife-clasp" release phenomenon. The involved muscle groups also exhibit exaggerated flexor and extensor reflexes. Another neuromuscular complication, the muscle spasm (sometimes included as a characteristic of spasticity), frequently accompanies the spasticity of CNS lesion and is often observed in the spinal-cord-injured patient.

Spasticity is generally considered to be detrimental to the patient (1). Gross spasm in the paralyzed limbs of a spinal-cord-injured patient can obviously interfere with activities of daily living. Also, spasm can lead to contractures, dislocation of joints, and can mask volitional control in case of paresis.

Currently, spasticity is managed in a variety of ways (2). These methods include drug therapy, chemical and surgical denervation, physical therapy, and sometimes surgical alteration of tendons. The physical therapy treatment involves passive exercise of the spastic muscles. This treatment, in addition to preventing contracture, has a lasting effect in quieting the spasticity. Apparently, when the paralyzed muscles are exercised, muscle stretch receptors and tendon organs are activated. This sensory information flow into the spinal cord and/or the passive activation of the reflex loop appears to quiet the spasticity. Unfortunately, the therapeutic effect that exercise has on the spasticity lasts only for a period of hours. With electrical stimulation we hope to utilize the same exercise-triggered mechanisms that reduce spasticity in passive exercise. The scheme includes equipping the patient with a portable electrical muscle stimulator. Thus equipped, the patient is free to exercise his paralyzed muscles at his convenience. The potential secondary

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benefits of this treatment include discontinued use of drugs, with their undesirable side effects, and elimination of the need for regular physical therapy. Also, the electrically exercised muscle acts as a pump returning blood, which otherwise might pool in the legs, to the heart. Because 70 percent of spinal-cord-injured patients suffer from deep vein thrombosis (thrombophlebitis), a condition arising from pooling of venous blood, this secondary effect could be very beneficial.

Our program begins with electrical exercise of recently injured spinal-cord patients with transcutaneous stimulation of motor points and motor nerves. If this phase of the treatment shows promise, the patient is considered for implantation of a permanent radio-frequency coupled transmitter-receiver-type electrical stimulator. The electrodes of the implanted receiver are placed around the motor nerves, and the transmitter is worn on the patient's belt.

Thus far, effectiveness of the treatment has been evaluated subjectively with the aid of a standard neurological examination. Here, judgments are made regarding changes in the indicators of spasticity, such as resistance to passive stretch, strength of the stretch and flexor reflexes, and duration of clonus. We are also in the process of developing two additional measures to evaluate the effectiveness of the treatment. Both of these measures are attempts to define changes in spasticity in as quantitative a manner as possible. The first of these two measures is the reflexogram. Our use of this measure is based on the knowledge that the stretch reflex is a primary indicator of level of spasticity. Calibrated taps are delivered to the Achilles tendon with a motor driven hammer. The resulting isometric torque produced by the stretch reflex is measured with a torque transducer attached to the foot. Both the tendon tap and the isometric torque are recorded on FM tape for later processing by computer. The second measure we are employing to quantify level of spasticity is the standard H-reflex (3). This reflex, except for bypassing the muscle spindle, follows the same pathway as the Achilles tendon stretch reflex. Use of this reflex as a diagnostic tool is standard practice (4). Also, its use as a measure of motor neuron excitability, a prime indicator of level of spasticity, is common and is well documented (5).

Our progress to date on this project includes a paraplegic patient who has been implanted with bilateral peroneal nerve stimulators (Oct. 1972). The equipment used in this case was a portion of Medtronic Neuromuscular Assist Device (Fig. 1) which has been developed to correct the footdrop condition that frequently accompanies hemiplegia. This device is supplied with a cycling module which may be used to exercise paralyzed muscle. In this case the anterior tibial groups of both legs were exercised. Because our objective measure of spasticity (reflexogram and H-reflex) had not yet been developed when this

patient received his implant, only the subjective evaluation of the changes in his spasticity was made. This evaluation indicated a significant reduction in the clinical indicators of spasticity. In addition to these primary effects, an additional beneficial side effect was noted. The electrical exercise initially arrested atrophy of the stimulated muscle, and as time passed, resulted in an increase of muscle mass not only of the stimulated muscle but also of the adjacent musculature above the knee (Fig. 2). Further, the patient was able to discontinue the use of muscle relaxant drugs as well as regular physical therapy sessions.

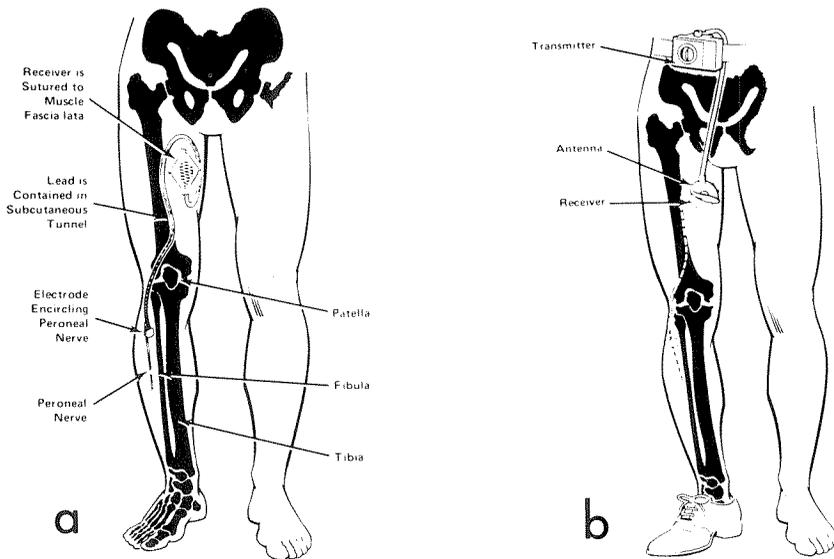


FIGURE 1.—Scheme illustrating method used to electrically exercise the paralyzed musculature. a. Receivers, lead, and peroneal electrodes are implanted in the patient. b. Battery-powered transmitter is worn on a belt and an antenna is taped over an implanted receiver. Copyright 1972 by Medtronic, Inc. Reprinted with permission.

Our immediate plans for the future in this part of our research include providing a second paraplegic patient with an implanted bilateral peroneal stimulator system similar to the one shown in Figure 1. Evaluation of the effectiveness of the treatment in this patient will include the use of both our subjective and quantitative measures.

The above study constitutes only part of our effort in evaluating the effectiveness of electrical stimulation for the treatment of spasticity. The

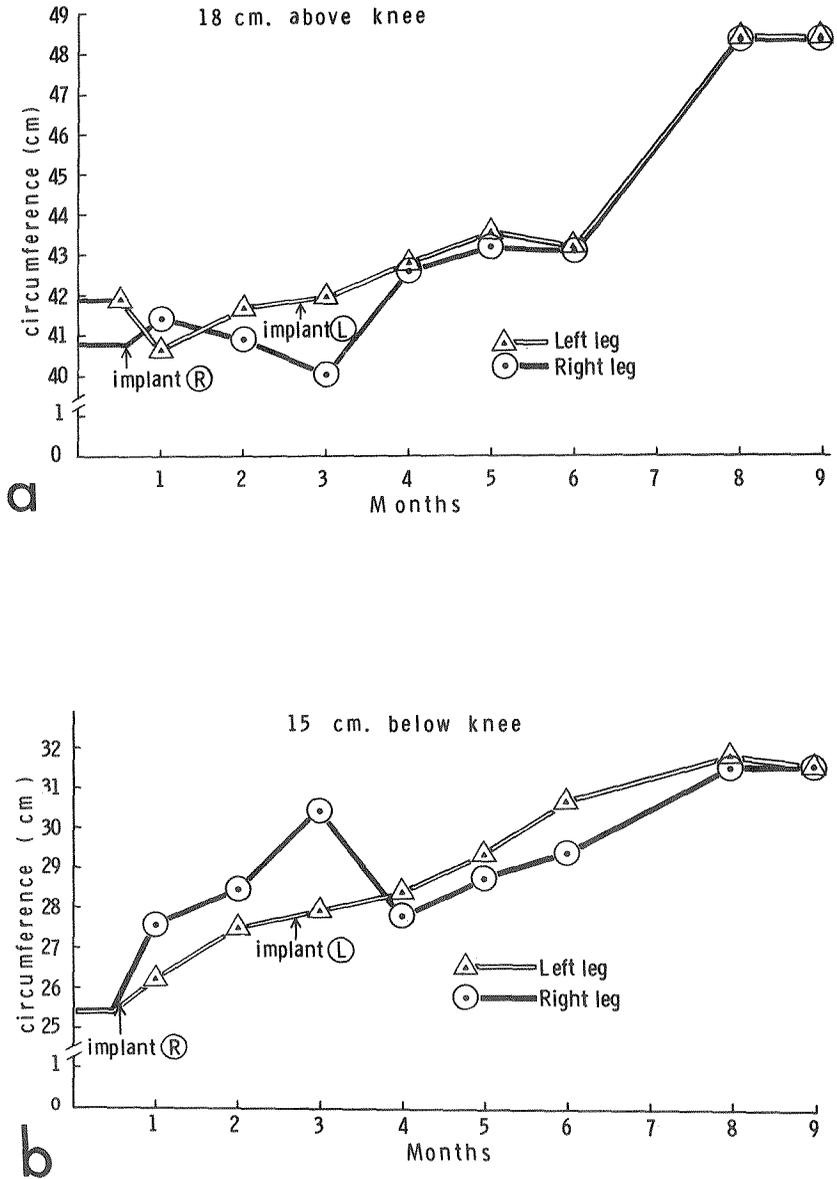


FIGURE 2.—Data illustrating hypertrophy of the leg following regular electrical stimulation of the peroneal nerves with implanted electrical stimulators. Hypertrophy is indicated by an increase in leg circumference. a. Circumference of the leg at a level 18 cm. above the knee. b. Circumference of the leg at a level 15 cm. below the knee.

second part of our effort is aimed at treating the spasticity that frequently follows severe brain damage such as cerebral palsy, stroke,

and head injury. While this type of spasticity (cerebral spasticity^b) has many characteristics that are similar to spinal spasticity, there are several differences. Spasticity of cerebral origin is frequently less violent and more regular than that arising from spinal lesions (2). Also, cerebral spasticity frequently masks voluntary muscular control, while in spinal spasticity, because the neural pathways for volitional control are frequently transected, there is no control left to mask.

Thus, successful treatment of cerebral spasticity potentially can be doubly beneficial in that it both reduces the spasticity and unmasks voluntary control. Many of the current treatments for cerebral spasticity are the same as those already discussed. Medication such as Valium (Roche), Dantralene (Eaton Laboratories), and other muscle relaxants have been partially successful with mild and moderate spasticity. Physical therapy and stereotaxically placed deep thalamic and cerebellar lesions have also been used to manage the spasticity. Very recently a treatment using electrical stimulation has been described (6). This treatment involves electrical stimulation of the cerebellum through electrodes placed directly on its cortex. The rationale for this treatment is based on a broad spectrum of neurophysiological studies which indicate that the cerebellar cortex acts as a significant inhibitor of the rest of the brain (7). This powerful inhibitory influence of the cerebellum was demonstrated before the turn of the century by Sherrington (8) who showed that decerebrate rigidity in cats could be inhibited by stimulation of the anterior cerebellar cortex. More recently, Moruzzi (9) observed that the stimulation of the anterior cerebellar cortex could increase or decrease decerebrate rigidity depending upon the frequency of stimulation. He found that a frequency of 100 to 300 Hz decreased rigidity while a frequency of 10 Hz resulted in an increase.

These and other observations led to the conclusion that the cerebellar cortex acts primarily in an inhibitory capacity, and that this inhibitory function can be increased and modulated with chronic electrical stimulation. The pioneering work in implanting cerebellar electrodes for chronic stimulation of the cortex was done by Cooper (6). He has

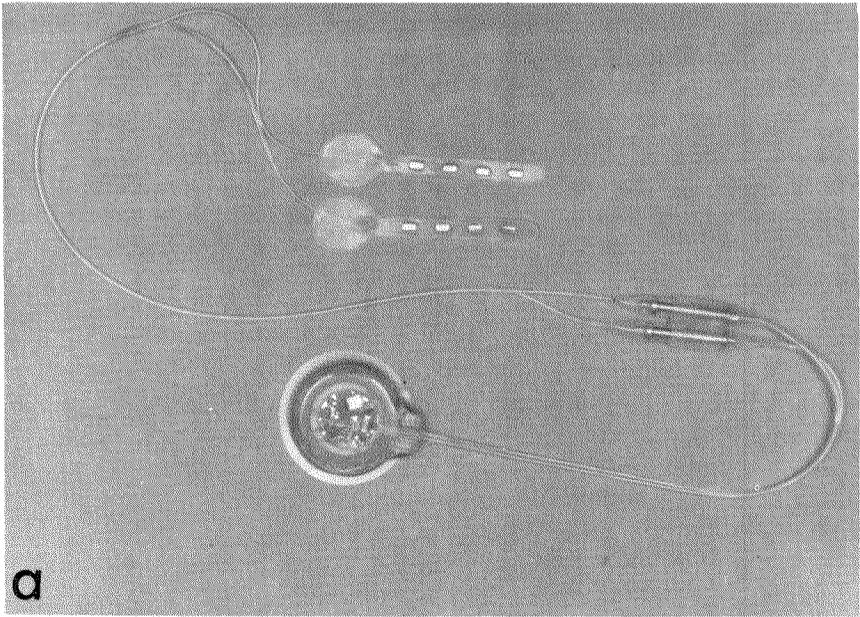
^bIt should be noted that all spasticity originates in the spinal cord and is the result of a hyperexcitable reflex loop. The hyperexcitability, however, can result from lesions at any point in the CNS. The terms "cerebral spasticity" and "spinal spasticity" thus refer to the location of the lesions which resulted in the hyperexcitability at the reflex loop.

reported at least 32 cases where the treatment was beneficial in reducing chronic cerebral spasticity.

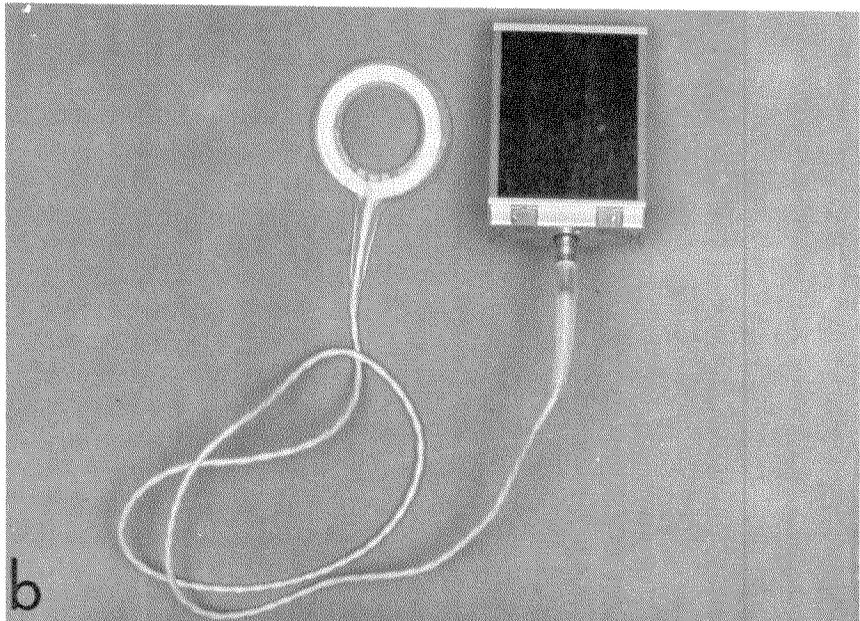
The aim of our effort in this project is to continue and extend the work of Cooper. Thus far we have moved toward this objective by developing a new technique which significantly reduces the complexity of implanting the cerebellar stimulators (10). Using the technique, we have implanted two patients suffering from cerebellar spasticity. In both cases, two arrays of silicon rubber-backed platinum button electrodes were used (Fig. 3). In the first case, the electrodes were placed on the anterior and posterior lobes of the right cerebellar cortex (Fig. 4). In the second case, the electrodes were placed on the anterior cerebellar lobes bilaterally. Electrical stimuli were delivered to the electrodes by means of a transcutaneous RF-coupled transmitter-receiver system supplied by Avery Laboratories (Fig. 3). Eight minute bursts of stimulus at a rate of 200 Hz were delivered alternately to the two electrode arrays. Stimulation was continuous. At this time we are still evaluating the effectiveness of the treatment in reducing the patient's spasticity. Preliminary subjective observations in the first patient indicate an improvement in speech and in gait and, in both patients, a somewhat reduced level of spasticity. H-reflex measurements on the first patient were made both pre- and post-implantation. These measurements indicate that the stimulation produced a decrease in spasticity, in that they showed a significant reduction in H-reflex detectable motorneuron excitability.

While our study of these two patients is not yet complete, our observations and measurements have led us to a number of conclusions regarding the cerebellar stimulation treatment. First, the treatment appears to be most effective when the electrodes are placed on the anterior cerebellar cortex. Second, placement of the electrodes on the posterior cortex has no detectable effect on reducing spasticity. Our third finding is that the beneficial effects of the treatment can be achieved even when the stimulation frequency is reduced from 200 Hz to 100 Hz. This reduction is beneficial in that it significantly reduces the rate of discharge of the batteries of the patient-worn transmitter.

Because this procedure appears to provide some relief from a pathological condition for which there are not other proven treatments, we plan to extend the study. Additional patients with cerebral palsy and dystonia are being considered for implantation and study. Also, followup studies are continuing on those patients who have already had an implant.



a



b

FIGURE 3.—Transmitter and implantable receiver—electrode system used in chronic electrical stimulation of the cortex. a. Implantable receiver, leads, and platinum button electrode arrays. Electrodes are placed on the cerebellar cortex. A receiver stimulator is implanted in the clavicular region. b. Battery-powered transmitter and antenna of stimulator system. The antenna is taped over the receiver.

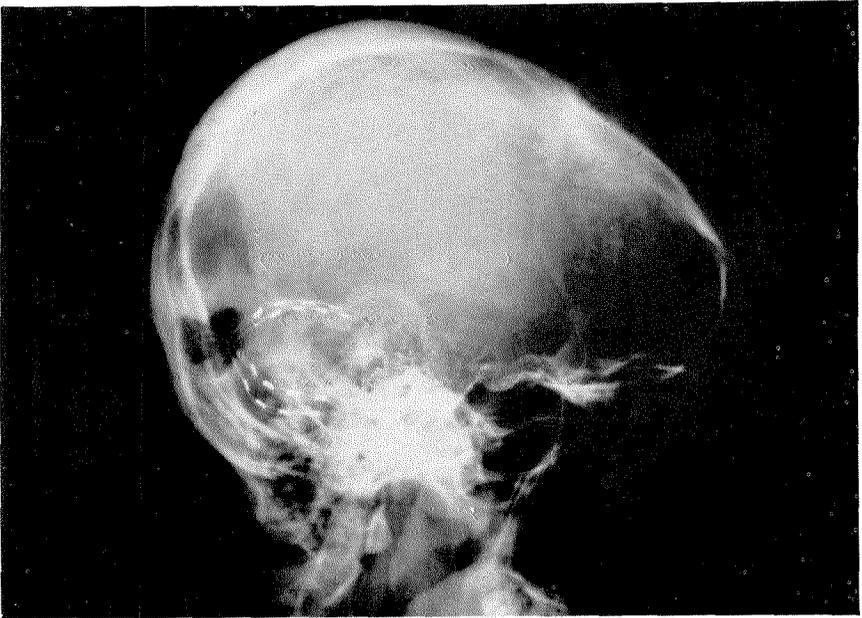


FIGURE 4.—X-ray illustrating placement of chronic cerebellar stimulating electrodes arrays on the anterior and posterior cerebellar cortex.

REFERENCES

1. Khalili, A.A.: Pathophysiology, Clinical Picture and Management of Spasticity. Clinical Anesthesia, F.A. Davis Co., 1967.
2. Pedersen, E.: Spasticity. Charles C. Thomas, Springfield, Ill., 1969.
3. Hoffman, P.: Über die Beziehungen der Sehnenreflexe zur willkürlichen Bewegung und zum Tonus. *Z. Biol.*, 68:351-370, 1918.
4. Braddom, R.I. and E.W. Johnson: Standardization of H-reflex and Diagnostic Use in S1 Radiculopathy. *Arch. Phys. Med. & Rehab.*, 55:161-166, 1974.
5. Angel, R.W. and W.W. Hoffman: The H-reflex in Normal, Spastic and Rigid Subjects. *Arch. Neurol.*, 8:591-596, 1963.
6. Cooper, I.: Cerebellar Stimulation Aids Victims of Intractable Hypertonia, Epilepsy. Interview by Gail McBride, Associate Editor, *J. Amer. Med. Assn.*, 225(12), Sept. 17, 1973.
7. Cooper, I., M. Riklan, and R.S. Snider: *The Cerebellum, Epilepsy, and Behavior*. Plenum Press, New York—London, 1974.
8. Sherrington, C.S.: Double (Antidrone) Conduction in the Central Nervous System. *Proc. Roy. Soc. London*, 618:243-246, 1897.
9. Moruzzi, G.: *Problems in Cerebellar Physiology*. Charles C. Thomas, Springfield, Ill., 1969.
10. Davis, R. and M. Dimancescu: Technique for Implanting Cerebellar Stimulator. Exhibit, Congress of Neurological Surgeons, Vancouver, Canada, 1974.