CLINICAL NOTES

Electrical Stimulation in Treating Spasticity Resulting from Spinal Cord Injury

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ABSTRACT. Bajd T, Gregoric M, Vodovnik L, Benko H: Electrical stimulation in treating spasticity resulting from spinal cord injury. Arch Phys Med Rehabil 66:515–517, 1985.

• To study the efficacy of electrical stimulation in treating spasticity of six spinal cord injured patients, transcutaneous electrical nerve stimulation (TENS) was applied to the dermatomes belonging to the same spinal cord level as the selected spastic muscle group. Spasticity was assessed in knee extensors by a pendulum test in which the knee joint angle of a swinging lower leg was recorded with an electrogoniometer. TENS was found to produce a noticeable decrease of spasticity in three of the patients, but had little effect on the others.

KEY WORDS: Electric stimulation; Muscle spasticity; Spinal cord injuries

There are three major approaches currently in the treatment of spasticity: pharmacologic, rehabilitative (physical) and surgical. This paper will focus on one type of rehabilitative approach—transcutaneous electrical nerve stimulation (TENS).[5] Although afferent nerve excitation is primarily intended for pain relief, subcutaneous stimulation of median, radial and saphenous nerves has proved to inhibit clonus in multiple sclerosis patients.[7] Since spinal cord stimulation[3] and cerebellar stimulation[2] have already been used to alleviate pain and reduce spasticity, this study was designed to determine whether cutaneous afferent stimulation would reduce spasticity.

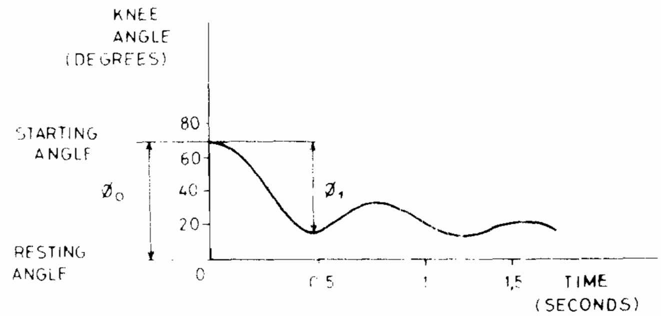

Fig 1—Knee joint goniogram displaying moderate spasticity of knee extensors. Starting angle (horizontal position of the lower leg), resting angle (vertical position of the lower leg), and amplitude of first swing are denoted.

Subjects

Six spinal cord injured patients (table) were randomly selected from the in- and outpatients of the spinal cord injury department of the rehabilitation institute. The six patients included those with clinically incomplete lesions who had retained some voluntary movements of their lower extremities, and those with lesions resulting in completely paralyzed lower limbs. All showed at least moderate spasticity on manual testing of passive resistance about the knee joint. This criterion resulted in the choice of the patients having thoracic and cervical lesions only. Their spasticity was symmetric in both extremities.

Stimulation

TENS was delivered through large (6 × 4cm) stainless steel sheet-metal electrodes covered with water-soaked gauze. The electrodes were placed over the L3,4 dermatomes,[4] one medially below the knee and the other laterally above it, to influence the spasticity of the knee extensors. No attempt was made to consistently place the cathode proximally. The electrical pulses were rectangular and monophasic. A stimulation frequency of 100 pulses per second (Hz), a pulse duration of 0.3ms, and a stimulation amplitude of up to 50mA was used. The electrical stimulation did not cause muscle contraction. Such a stimulation pattern was applied continuously for 20 minutes.

During the electrical stimulation treatment the patients were supine with both lower extremities extended. All treatments were performed in the morning before other physical therapy exercises.

Measurement

Spasticity was tested with the pendulum test.[6] For this test of spasticity of the knee extensor muscles the patient is supine on a tilt table with both legs bent over the edge of the table and hanging free at the knee. He is asked to relax as much as possible. The examiner grasps the patient's foot and brings the leg to a horizontal position. The limb is allowed to fall freely as the knee angle is recorded with an electrogoniometer.[1]

In a healthy subject the leg swings around the resting position. With slight spasticity, there is an irregularity in only the first swing of the lower leg; with moderate spasticity, the leg slowly oscillates toward the resting position (fig 1); and with extreme spasticity, the lower leg does not move from the fully extended starting position. The level of spasticity was evaluated from the initial drop of the leg.

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General Data on Six Spinal Cord Injured Patients

Case no.	Age, yr	Time postinjury, mo	Lesion	Cause
1	20	10	T8,9 comp	Fall
2	51	5	T6 incomp	Tumor
3	52	10	C6,7 incomp	Fall
4	26	48	C5 comp	Diving
5	11	9	T5,6 comp	Gunshot wound
6	21	19	T6 comp	Gunshot wound

A relaxation index R was defined as the ratio between the amplitude of the first swing ϕ_1 and the difference between the starting and resting angle ϕ_0 (fig 1). In normal subjects this ratio was found to be 1.6, therefore the relaxation index was normalized as follows[1]: $R = \phi_1/1.6\phi_0$. Thus, $R \geq 1$ would signify a nonspastic limb whereas $R < 1$ would quantify various degrees of spasticity.

To improve the reliability of the proposed measurement the pendulum tests were performed ten times one after another at 30-second intervals. At such fast repetitions of the measurements the test itself influences the next tests and the degree of spasticity is usually lessened. To quantify this repetitive testing the average value of ten relaxation indices was calculated and denoted as R_{10}. In previous work[1] we found that in the same patient the parameter R varies considerably from day to day, while R_{10} shows steady values describing some base line of the patient's spasticity.

The first series of ten pendulum tests was performed prior to the application of electrical stimulation, the second series was performed immediately after the treatment, while the third series of tests was made two hours thereafter. The patients had been relaxing for five minutes prior to the first measurement. During the two hours between the second and third measurements the patients were sitting in the wheelchair and received no therapeutic treatment.

RESULTS

The results obtained for the average relaxation index R_{10} are shown in figure 2. In patients 1, 5, and 6, spasticity significantly decreased immediately after the stimulation. The difference in R_{10} before and after the application of TENS was 0.32 in patient 1, 0.68 in patient 5 and 0.41 in patient 6. The patients' descriptions of their state of spasticity were consistent with the measurements. Patients 1 and 5 reported a decrease of spasticity after TENS. Patient 6 claimed that he had never been as relaxed as after the stimulation.

DISCUSSION

When the relaxation indices obtained before and after the electrical stimulation are compared, TENS appears to have reduced spasticity in three of six patients. However, the stimulation seems to be effective only for a short time, not more than two hours. With TENS, sensory information is delivered predominantly through the large diameter afferent fibers conveying information from the mechanoreceptors to the spinal cord. The decrease of spasticity is presumably due to electrical stimulation of the large diameter afferent fibers. The important role of afferent stimulation in decreasing spasticity is also evident from the positive results obtained by spinal cord stimulation.[3]

To speculate on the specific neurophysiologic mechanisms

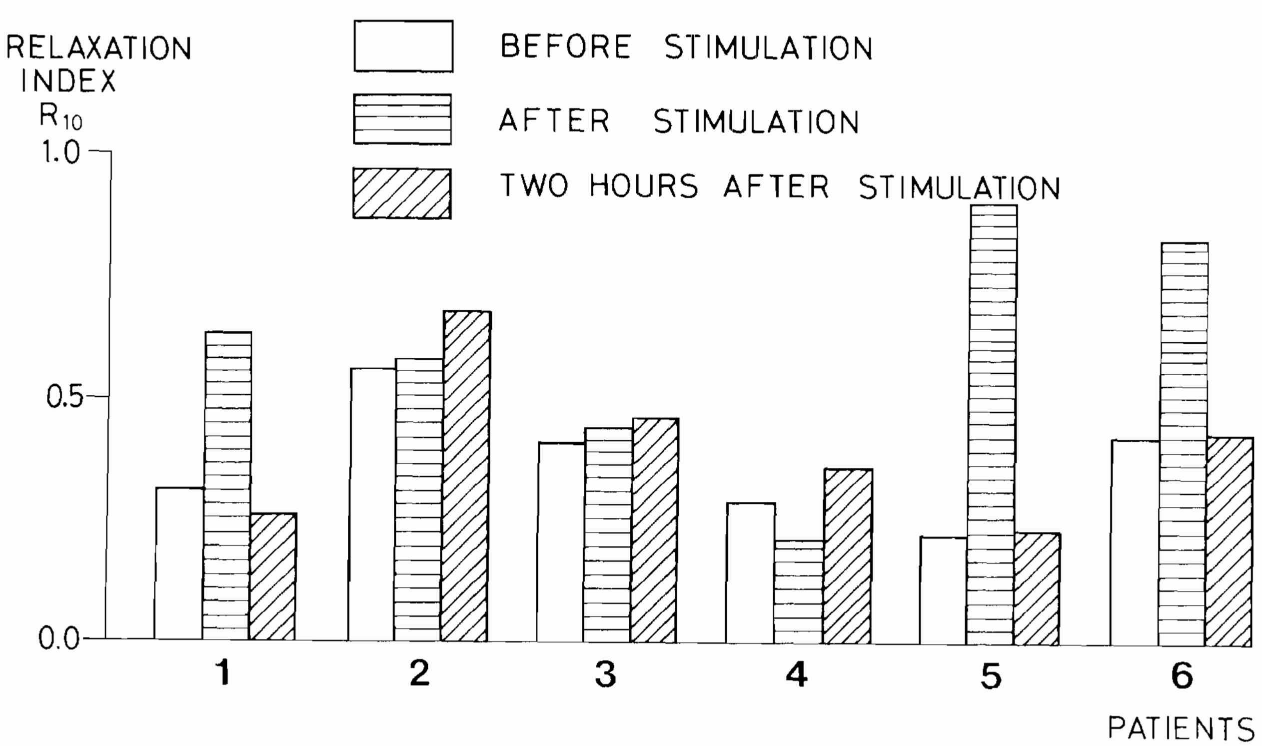

Fig 2—Relaxation before and after application of TENS

responsible for the effect seems to be premature at present. Of course any of the proposed mechanisms such as I_b autogenetic inhibition, Renshaw inhibition, reciprocal inhibition or decreased α-motoneurone excitability may be involved. The fact remains that with basically unspecific stimulation, specific and beneficial improvements can be obtained. Perhaps the diffuse stimulation applied at the periphery or in the spinal cord activates the atrophied inhibitory synapses but has no effect on the active excitatory synapses whose efficacy is already at saturation level.[6]

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BOOK REVIEWS

PEDIATRIC REHABILITATION by *Gabriella Molnar, MD*. Hardcover. Price $50.00. 478 pages. Williams and Wilkins, PO Box 969. Waverly Press Lane, Easton, MD 21601.

In his foreword as the editor of the series of books begun by Sidney Licht as the Rehabilitation Medicine Library, John V. Basmajian points out that this is the 19th such foreword he has written over the past six years. This observation illustrates the rapid expansion of these texts. The editor, Dr. Molnar, has assembled 16 national authorities working from New York City to Sioux Falls, and their team effort has produced a well-rounded text for the physician dealing with rehabilitation problems of childhood and infancy.

The book is divided into two parts. Part One deals with the principles of diagnosis and treatment, touching on the foundations of assessment and basic treatment in such areas as growth and development, psychology and communication, therapeutic exercise and orthotics. An entire chapter on electrodiagnosis addresses the special problems of children. A separate chapter on psychosocial issues speaks to this important point.

The second part is devoted to rehabilitation of specific childhood disabilities and contains the traditional diagnoses of myelodysplasia, arthritis, cerebral palsy, and limb deficiencies. This section also includes unique chapters on skeletal disorders, burns, spinal and head injuries. A chapter on diseases of the motor unit complements the chapter on electrodiagnosis presented in the first half of the text.

The book is well illustrated, current, and contains extensive bibliographies supporting each chapter. This publication continues in the finest tradition of Sidney Licht who began the Rehabilitation Medicine Series years ago. It should be in the library of every physician who sees disabled children (*Vincent D. Stravino, MD*)

ANATOMICAL CORRELATES OF CLINICAL ELECTROMYOGRAPHY (2nd ed.) by *Joseph Goodgold, MD*. Hardcover. Price $33.00. 194 pages. Williams and Wilkins, PO Box 969, Waverly Press Lane, Easton, MD 21601.

In reviewing Dr. Goodgold's book, I felt it necessary to compare it with the first edition of 1974. The new edition contains 44 more pages, dealing with the more esoteric muscles and their anatomical locations.

The basic format of the book remains unchanged. Anatomical drawings and photographs of models are presented with cross sections at various levels, providing information on the muscle location and nearby neurovascular structures.

There are five major sections in the book: I. The Head and Neck, II. Pectoral Girdle and Thorax, III. Upper Extremity, IV Trunk, Pelvic Girdle and Perineum, and V. Lower Extremity. The first section, for example, now describes the temporalis, internal pterygoid, and aryngeal and pharyngeal muscles. Special attention is paid to the facial and accessory nerves. The second section now contains the diaphragm, plus information on locating the phrenic and long thoracic nerve. Similar additions have been made in the other sections.

The book is straightforward in content, and highly recommended for anyone involved with electromyography. Some of the newly added muscles may never need to be examined by the average electromyographer, but a reliable source is now available in this book should the need arise (*Asa G. Hubbard, MD*)