

Electrical Stimulation in the Management of Spasticity: A Review

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Abstract

Despite successful reports of spasticity management by electrical stimulation [ES] over the past 246 years, this potentially effective and economical tool is often overlooked in clinical practice in favor of oral medications with serious side-effects, intrathecal drug administration, or surgical procedures including tendon lengthening and long-term muscle denervation. The body of literature on ES and spasticity provides a rationale for critical review of the relative merits of all strategies employed to manage spasticity as well as for the development of criteria to use these available tools in concert for optimal patient outcomes.

Index terms: Spasticity; Electrical Stimulation; Stroke; Brain Injury; Spinal Cord Injury; Cerebral Palsy; Multiple Sclerosis; Amyotrophic Lateral Sclerosis.

Historical Perspective

Electrical stimulation has been used therapeutically for over 2,000 years. It has been employed to manage spasticity for 246 years, or more. In 1752, Benjamin Franklin wrote a case report describing his use of electricity to manage involuntary muscle contractions in a young patient. In 1871, Duchenne used electrical stimulation [ES] to inhibit spastic antagonist muscles that interfered with function. [1-2]

Following the interest in the neurophysiological mechanisms underlying spasticity in the early 1900's, practical electrical stimulation devices became available in the 1960's. Success with cutaneous, or skin, electrodes was replicated when implanted electrodes were employed.

Cerebellar and spinal cord stimulation studies in the early 1970's appeared to reduce spasticity and improve the lives of the patients implanted, but the statistical outcomes were not universally rewarding. [3-4]

Despite the proliferation of studies in the past 20 years, this body of knowledge has been ignored by many clinicians. In many centers, medications are the first treatment of choice and intrathecal drug administration is commonly employed. Surgical intervention, including phenol nerve block and tendon lengthening is recommended without prior trials of electrical stimulation. It is time to consider the development of criteria for the use of electrical stimulation in concert with, or as an alternative to medications and surgery.

Consensus of Results in Peripheral Nerve Stimulation

Study protocols, using objective measures of spasticity, ranging from single assessment to two year follow-up have demonstrated statistically significant reduction in spasticity as a result of peripheral nerve stimulation. [5-16] Cutaneous electrodes, implanted electrodes and implanted neural prosthetic systems have led to reduced interference from spastic muscles, improvements in volitional control and positive changes in the energy demand of walking. [11-12] Carry-over effects ranged from 30 minutes to 24 hours, or more. Functional outcomes were realized when ES was combined with goal directed physical therapy. [7-8,17-18]

Results of Cerebellar and Spinal Cord Stimulation

Cerebellar ES in cerebral palsy and multiple sclerosis resulted in reduced spasticity in the majority of subjects along with improvement in bladder function, respiratory function, volitional control, active and passive movement and mood state. [3] Spinal cord ES outcomes were similar with carry-over effects lasting up to 24 hours.[4] Functional test scores in many of the studies did not reflect the other improvements observed. [3-4]

Problems in Study Design

Among the problems in some study designs have been small subject samples with extremely varied diagnoses and severity of disability. Objective measures of spasticity have not been uniformly employed. Gross functional scales have been used as the primary measurement tool and the statistical significance criteria imposed would have required relatively miraculous changes in function in order to be considered efficacious. In addition, many final publications have disregarded reports of improvement from patients, families and physicians. [2-4]

Critical Concepts

Confusion exists in terminology, selection of assessment tools, methods of patient evaluation and the importance of specificity in goal setting, treatment and outcome evaluations. Terms such as "muscle tone" and "muscle spasm" are not objectively measurable and should be replaced by "spasticity" which can be measured.

Spasticity must be assessed in the upright or most functional position if interference is to be accurately defined. It must be recognized that interfering muscle activity [ie in walking or transfers] may be the result of a spastic response to voluntary use of antagonist muscles or it may be that the inappropriate muscle is being recruited in the wrong phase of movement. In the latter case, spasticity is not the culprit and we have no evidence that therapy will alter the cortical recruitment pattern. Surgical relocation of the muscle's action to its functioning phase would then be appropriate. [19-20]

The only way to determine if inappropriate muscle activity is responsible [spastic or out of phase, or both] is to do intramuscular electromyography [EMG] recordings during movement such as walking or grasp and release. Cutaneous EMG recordings are contaminated by volume conduction from all muscles in the limb and are useless for diagnostic purposes of this nature. [21-22] It must be recognized that each patient has his or her own "neurological fingerprint" of

neurological dysfunction and treatment must be tailored to each individual.

It must be recognized that ES alone is usually not an encompassing treatment for spasticity. ES can "unmask" residual control and result in early recovery of selected functional movement. In most instances when the patient is in the phase of "neural recovery" after insult to the CNS, reduction of spasticity with ES is only the first step. ES can then be employed to improve muscle recruitment and performance [force, work, power, and fatigue resistance] as well as enhance timing of recruitment for function. When recovery is incomplete, ES may be used as a neural prosthetic for maintained daily function.

Goals must be individualized and small achievements that result in even minimal improvements in function and quality of life must be delineated. Whether the goal is improved sitting position and tolerance resulting in less frequent repositioning by an attendant or the ability to bring the body weight forward over the base of support in order to allow a reasonable contralateral step length, improved safety in walking, increased free pace velocity and reduced energy demand, objective documentation of goal achievement is invaluable. It is not necessary to change from bedridden to walking independently to show efficacy of ES in the modulation of spasticity.

Common Misconceptions

There are a variety of misconceptions in the treatment of spasticity. One serious pitfall relates to the amount of ES required per day to reduce spasticity and the need for immediate ES treatment when spastic episodes occur. There is agreement among researchers that 1-2 hours of ES per day will suppress spasticity on a 24 hour/day basis. In addition, the ES may be administered at any time, including nighttime. Even cutaneous, or sub-motor, intensities of ES result in 24 hour/day suppression of interfering spasticity. [15-16]

One of the biggest impediments to the use of spasticity is the misconception that ES for spasticity modulation is expensive or esoteric. There are a variety of ES devices available through wholesale vendors for less than the cost

of a single physician or therapy clinical visit [ie less than \$45-100.00, US currency]. Despite the body of literature and the inexpensive cost of ES devices, a relatively recent publication states that there is no basic research and there have been no multicenter trials on the use of ES to control spasticity, and the clinical availability of ES systems for this purpose in the U.S.A. is limited. [23] The survey results reported in this publication indicate that of 105 "FES" centers, only 4 centers listed spasticity management among their services [Public Hospital in Lonato, Italy; National University Hospital in Reykjavik, Iceland; "Shake-A-Leg" FES Research Program, University of Sydney, Australia; and the Cleveland Clinic, Ohio]. These four centers state that they charge from approximately \$1,000.00 to \$20,000.00 to treat spasticity. [23]

ES can be used anywhere in the world to modulate spasticity for a minimum cost [\$45.00 to \$100.00 and the cost of 1-3 physical therapy visits]. The lack of recognition of this opportunity by clinicians and publishers of consumer education materials is revealing of the lack of understanding of the clinical needs of patients and the ES research and clinical outcome reports available in the literature.

Considerations of Risk

ES for spasticity modulation is relatively risk free. Although it is possible that spasticity may be temporarily exacerbated, especially if abrupt muscle contraction is generated, any adverse effect is significantly reduced or absent within 30-60 minutes. If the patient uses spasticity to allow standing, transfers or limited stepping, the reduction of spasticity by ES may reduce function until ES control of muscle or ES facilitation of muscle recruitment can be instituted. When ES is applied as one component of an integrated rehabilitation protocol, this is not a problem.

In comparison to the muscle weakness, depression of CNS and respiration associated with drugs and the risk of infection with implanted medication dispensing devices, ES is extremely safe. [24] In comparison with the muscle weakness, severe post-operative pain, subluxation of the hip, spinal deformity, increased incidence of spondylolisthesis and

potential for recurrence of spasticity associated with rhizotomy, ES is very safe. [25] In comparison with the muscle weakness or even complete denervation associated with botulinum toxin and phenol nerve block, ES is a preferred initial treatment. [19-20,26-27]

Summary of Clinical Suggestions

Individual patient care can be improved through clarity of terminology [ie omission of terms like tone and spasm and replacement with spasticity when applicable] and the use of objective measurements [ie measurable resistance to passive joint movement, available joint range of motion, sitting tolerance, specific hand function assessment, manual muscle test or instrumented assessment of force, work, power and fatigue, and mechanical as well as metabolic characteristics of gait].

Attention to specific changes in function is critical. It is not necessary to improve from bedridden to independent ambulation, for example, to realize the benefits of reduced spasticity. Careful documentation of ES protocols is important. For example, the use of a comfortable, balanced pulse duration [ie 300 usec] and a long ramp in intensity [2 seconds or more] along with a minimal intensity will minimize the potential for aggravation of spasticity in the early days of an ES protocol.

When prospective data collection is planned, categorization of patients by diagnosis, severity and specific goals will improve outcome evaluation and statistical analyses.

Suggestions For Future Spasticity Studies

There are many options for successful use of ES to modulate spasticity and accomplish reasonable, efficacious goals for the patient. When ES alone is not adequate to resolve the penalties of severe spasticity, it may offer a first line of evaluation as well as an adjunct to the overall rehabilitation outcomes. [28-29]

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