Electrical Stimulation To Improve Movement: Practical Application of Electroneuromyographic [ENMG] Assessment to Patient Selection

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Abstract

The purpose of this presentation is to consider criteria for including electrical stimulation [ES] in clinical protocols to improve motor capability. Based upon inquiries to the IFESS website, there is a need to provide rationale for selection of ES candidates and for identification of potential surgical patients. The information presented has immediate application to direct consumers of ES [including patients, physicians and therapists], to third-party payers and to the selection of target populations for ES technology development and clinical trials.

1 Introduction

When the goal of including ES intervention is to improve muscle performance and movement, there are specific questions to be asked with special reference to the patient’s diagnosis. While the basic questions appear to be simple, the answers are critical to the success of ES protocols. For example, if muscles are “weak,” what are the possible causes for the muscle weakness? Why is fatigue rapid? Or, why is the individual unable to use the muscles in functional tasks such as walking?

Patients, physicians, therapists, case managers and engineers have requested information through the IFESS Consumer Education website. The enthusiasm for implementing ES often has not been accompanied by an understanding of the criteria for patient selection. The purpose of this presentation is to consider criteria for implementing ES to improve muscle performance and to discuss the practical application of ENMG assessment [electromyography (EMG) and nerve studies (NSS)] in clinical decision making for cutaneous and implanted ES as well as for surgical procedures [tendon lengthening or transfer and procedures to inactivate muscle including neurectomy and botulinum toxin].

1.1 ES Considerations In Disabilities With Peripheral Nerve Pathology

Orthopaedic disabilities, including fracture, joint injury and repair, “frozen shoulder,” or immobilization after injury/repair: muscle weakness may be attributed to muscle disuse, loss of joint mobility or muscle inhibition secondary to pain. In each case, ES may improve muscle performance by pain modulation, increase in joint ROM and improvement in force, work and fatigue resistance. When muscle weakness is associated with peripheral nerve injury, however, ES may not be of value and may potentially interfere with the final stages of recovery. ENMG assessment can reveal the presence of neuropraxia [nerve block] as well as the presence of partial muscle denervation [axonotmesis] and the state of recovery. While ES may not be effective in regaining motor control until the nerve block has cleared, it could help to minimize the sequelae of disuse. When the muscle is partially denervated, ES will not be of value in strengthening and the overuse of the remaining motor units is thought to inhibit the nerve cell adhesion molecule [NCAM] and other chemical mediators required for “reattachment” of axonal terminals with the denervated muscle fibers [1]. Electrical stimulation of the denervated fibers will suppress fibrillations, as well. In partial denervation, reinnervation may occur by axonal regrowth [1 mm/day] or by terminal sprouting of the remaining axons within the muscle, which should occur within 6 months. Each motor unit has the potential to enlarge to approximately 5 times the original size. So, unless the partial denervation is severe, full reinnervation is possible by 6 months. Efforts to improve motor performance, including ES, would be appropriate at that time, with the understanding that return to normal, pre-injury status may not be possible if the nerve injury was severe enough to leave only a small population of very large, hard working motor
Peripheral Nerve Injury as Primary Diagnosis:
including Facial Nerve [Bell’s Palsy]; Ulnar Nerve [elbow]; Deep Fibular Nerve [knee]; Brachial Plexus; or multiple root pathology.

When the muscle is weak, but not paralyzed, the same questions asked above would apply. How much of the weakness is due to neuropathia and how much is due to partial denervation? The nerve block may clear at any time, axonal regrowth is at a rate of 1 mm/day and terminal sprouting should complete within 6 months.

If the muscle is completely paralyzed, is it a complete nerve block, complete denervation, or severe denervation with complete block of the remaining intact motor fibers? The implications are quite different for short-term and long-term planning. If the problem is a complete nerve block, ES can be comfortably employed for maintenance of muscle, ROM and it may provide sensory input to encourage recovery once the block begins to clear. If, on the other hand, the muscle is completely denervated, the only mechanism of reinnervation is by axonal regrowth. This may take 4-6 months for a Facial Nerve injury and 1-2 years for a Lower Trunk of the Brachial Plexus lesion. ES of the completely denervated muscle, with pulse durations at chronaxie, or longer, may be advisable to maintain the mobility of the intramuscular connective tissue [until the first ENMG signs of reinnervation] to provide a mobile end-organ once reinnervation has taken place. When there is no potential for axonal regrowth [multiple nerve root avulsion or proximal injury with retrograde degeneration of the anterior horn cells], ES cannot be justified.

Despite the research interest in the functional control of completely denervated muscle, commercially available technology is not available. Patients with multiple root avulsions in the cervical spine, for example, often elect to have amputation of their arm.

The stimulus requirements for sarcolemma versus neurilemma as well as the potential for muscle reinnervation in a particular diagnosis are worthy of attention by research teams. Development of ES technology for complete paralysis due to neuropraxia will be successful with 300 uS pulse durations, but the application may not be required for more than a few weeks or months. In the case of complete denervation, pulse durations must equal or exceed chronaxie of the individual muscle and once reinnervation has taken place, the device will not be needed [or the pulse duration will be excessive].

Systemic Disease with Peripheral Neuropathy, including diabetes and rheumatoid arthritis: While ES may have application to wound healing and pain modulation, expected outcomes of strengthening are contingent upon the extent and severity of neuropathy and medical management of the primary disease. In the presence of active synovitis, ES of the muscles crossing the joint would potentially increase intra-articular pressure and joint pain, resulting in further inhibition of the weak muscles [2-3].

Disease of Peripheral Nerve: It is commonly recognized that over-work, including ES, of muscles compromised by polio is not indicated. This same consideration is not afforded patients with Guillian-Barre Syndrome [GBS]. It is commonly believed that demyelination is the primary problem and that the axonal form of GBS is rare in the USA. In our experience, 95% of GBS individuals demonstrated current and/or past denervation on ENMG [n=22]; In a questionnaire, 99% of 150 patients indicated that they were referred for strengthening and gait training as soon as their paralysis plateaued [4] When motor recovery is slow, prescription of ES is common for strengthening. Weakness due to demyelination will not be remedied by ES and all of the considerations regarding partial denervation and the formation of giant, hard-working motor units would be appropriate in GBS. It appears ES is being utilized for muscle strengthening without regard for the neurological status of the GBS patient.

In Charcot-Marie-Tooth disease, muscle weakness and sensory loss are the result of degeneration of the peripheral nerves in the feet and legs, and in more severe cases in the hands. There is no evidence that ES can alter this process or be of functional benefit.

1.2 ES Considerations In Disabilities With Central Nervous System Pathology

Kinesiological EMG [fine wire, intramuscular (IM) recording of multiple muscles during functional] is crucial for decision making in stroke, brain injury and cerebral palsy. Questions include: Is the muscle being recruited?; Is the recruitment sufficient for function?; Is the recruitment tardy?; Is the antagonist muscle[s] active inappropriately due to spasticity or is it out of phase? Intramuscular EMG recordings are the only way to obtain this
information, because of the contamination of cutaneous EMG recordings by volume conduction [5]. If the problem is one of insufficient or tardy recruitment, ES may improve performance. If there is no recruitment, ES may substitute for volitional effort [and when recovery is inadequate, long-term neural prosthetic use is indicated]. If spasticity of the antagonist is the culprit, ES may modulate spasticity. If the muscle is truly out of phase, no therapeutic strategy has been shown to change the phase, and surgical transfer/inactivation of the individual muscle[s] is worthy of consideration. [6] For example, the cerebral palsy child who has excessive plantar flexion in swing may not have a problem with dorsiflexor recruitment. The culprit may be a tibialis posterior muscle that is out of phase. ES will not change this. Transfer of the tibialis posterior tendon to the dorsum of the foot where it can function in phase as a dorsiflexor of the ankle is appropriate. The upper motor neuron [UMN] patient with excessive ankle plantar flexion in stance may be improved by ES to modulate spasticity of the plantar flexors, allowing the tibia to ride forward during stance. Paralysis, or weakening, of the plantar flexors by lengthening or chemical injection may reduce spasticity but also lead to loss of tibial control for stance with knee buckling and reduced walking ability. The UMN patient who lacks hip, knee and ankle flexion for limb advancement as a result of insufficient recruitment may use ES of the dorsiflexors to elicit a flexion reflex for swing.

1.3 ES Considerations In Disabilities With Both Central And Peripheral Nervous System Pathology

Spinal Cord Injury: In addition to the questions related to UMN disability, it is appropriate to ask if there is lower motor neuron [LMN] involvement. Partial denervation distal to the zone of cervical and thoracic cord injury may contribute to weakness in the first six months after injury [7]. Strengthening efforts prior to 6 months [for terminal sprouting] would prematurely exhaust funding for therapy to strengthen muscles. Amyotrophic Lateral Sclerosis: ES has resulted in some muscle hypertrophy in the early stages of ALS. The improvement in motor use of the upper extremities, however, was reported to be the result of spasticity modulation which unmasked existing capability and resulted in functional gain [8]. Once denervation has progressed, ES cannot improve motor ability. Myelomeningocele: Lower limb weakness may be associated with UMN recruitment and spasticity problems or secondary to partial or complete absence of motor innervation. ENMG findings provide a basis for comprehensive consideration of muscle strengthening, spasticity modulation and surgical management to minimize deforming forces in the lower limb.

2 Discussion and Conclusions

Successful clinical outcomes require knowledge of each patient’s profile of muscle weakness. ENMG, including IM EMG, allows evaluation of present and past motor unit status. Dynamic IM EMG is the only definitive way to answer questions regarding recruitment and antagonist interference in function. ENMG findings lead to reasonable “strengthening” goals and treatment, including ES. Modulation of spasticity with ES is preferable to muscle paralysis and may unmask existing motor function. When ES does not adequately reduce spasticity, combination with other strategies such as drugs, botulinum injection and/or surgery, is feasible. [See ES in Spasticity References, www.ifess.org.]

References