

# NEUROMUSCULAR ELECTRICAL STIMULATION AND DYNAMIC BRACING AS A TREATMENT FOR UPPER-EXTREMITY SPASTICITY IN CHILDREN WITH CEREBRAL PALSY

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**We have investigated a therapeutic regimen using neuromuscular electrical stimulation (NMES) and dynamic bracing to assess their effectiveness in reducing upper-extremity spasticity in children with cerebral palsy. Nineteen patients between 4 and 21 years of age with documented diagnoses of spastic cerebral palsy were treated. The patients included in the study followed a regimen of two 30-minute sessions of NMES of the antagonist extensors combined with dynamic orthotic traction during the day. A static brace was used at night. Spasticity of the wrist and fingers was assessed periodically using the Zancolli classification. Treatment ranged from 3 to 43 months. After treatment with electrical stimulation and dynamic bracing, all the patients moved up 1 to 3 levels in the Zancolli classification and showed a marked improvement in upper-extremity function. These results show that combining NMES and dynamic orthotic traction dramatically decreases spasticity of the upper extremity in young patients with cerebral palsy.**

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Spasticity in patients with cerebral palsy is a result of pathologically increased muscle tone and hyperactive reflexes mediated by a loss of upper motor neuron inhibitory control. The increased muscle tone may vary from mild to severe. Associated abnormal clinical findings include co-contractions, muscle clonus, the "clasp-knife phenomenon", rigidity (Gilman and Newman, 1987), muscle weakness, and loss of dexterity (Young and Wiegner, 1987).

Current treatments use several approaches. The spastic muscles can be weakened by using oral neuropharmacological agents (Joynt and Leonard, 1980; Rice, 1987) or injectable material such as botulinum-A toxin (Albright et al., 1991; Calderon-Gonzalez et al., 1994; Koman et al., 1993). The neuromuscular apparatus can be blocked temporarily with alcohol and phenol (Carpenter, 1983; Carpenter and Seitz, 1980). Surgical treatment is by tendon transfer to balance the spasticity (Goldner, 1988) or selective neurectomy (Young and Wiegner, 1987). Three other treatment options are strengthening the antagonist musculature with electrical stimulation (Hazelwood et al., 1994; King, 1996; Shindo and Jones, 1987), physical therapy (Palmer et al., 1988), and stretching the spastic muscle with an orthosis (Hoffer et al., 1974).

Although orthoses have been used with passive manipulation techniques to assist in "stretching out" the spastic muscles, most clinicians do not consider this form of treatment to be effective. Spasticity does not diminish with bracing probably because the added resistance to the isotonic contracting muscle seems to increase the tone of the spastic muscle (Manske, 1990). Upper-limb orthoses are temporary expedients and do not seem to be effective in encouraging function or in correcting contractures (Bleck, 1987). However, the Contracture

Reduction Orthosis (CRO) (Lunsford, 1993) for the lower extremity, which is actually a form of dynamic splinting, is currently an accepted technique to treat cerebral palsy patients but is described by Lunsford as static progressive stretching. This concept is based on the use of a powered orthosis to stretch the contracted tissues slowly until a predetermined resistance is met. Stretch is maintained for a preset period; then the actuator backs off to allow the tissues to relax. This is repeated automatically as often as desired, and the therapist can program all the parameters.

A few articles document the use of upper-extremity casting in children with cerebral palsy. Groen and Dommissie (1964) reported good results, i.e., a gain in range of motion (ROM), in one of three cerebral palsy children on whom an arm cast extending from above the elbow to the fingertip was applied. The second child had poor results, and the third child withdrew from the study, thereby yielding no outcome. Yasukawa (1989) reported improved strength and control of the hemiplegic arm in a child on whom inhibitory casting and a bivalve night splint were applied. The use of an orthosis alone addresses only the static component of spasticity (muscle shortening), but does not address the dynamic component (abnormal tone and imbalance) of spasticity, thus giving little or no improvement to the impaired upper extremity.

Electrical stimulation has also been used in treating paralysis. Reports have shown its efficacy in treating paralysis secondary to peripheral nerve lesions (Sisken et al., 1993), spinal cord injuries (Formal et al., 1997), cerebrovascular accidents (Roper, 1987), as well as in sports injuries and athletic training (Goldspink et al., 1991; Kramer and Mendryk, 1982). The use of electrical stimulation to treat spasticity is not a new concept. As early

as 1952, Levine et al. reported that stimulation of the antagonist to a spastic muscle, followed by vigorous ROM exercises, led to a dramatic decrease in muscle tone. Alfieri (1982) also demonstrated a long-term reduction of muscle tone by administering multiple treatment sessions of electrical stimulation to the antagonist of spastic muscles.

Despite its long and varied history, there are only a few reports considering the use of electrical stimulation in patients with cerebral palsy. Dubowitz et al. (1988) reported improved muscle power and gait patterns using chronic low-frequency electrical stimulation in two hemiplegic children. Laborde (1986) also reported improved muscle power and gait patterns using electrical stimulation applied to the quadriceps muscles of children with cerebral palsy. Carmick (1993) reported improved locomotor efficiency and gait patterns after applying electrical stimulation to various muscle groups for variable lengths of time to three children with cerebral palsy. Logan (1987) reported improved mechanical efficiency as an immediate effect of one treatment with NMES in spastic cerebral palsy patients. Pape et al. (1990) reported significant clinical improvement after therapeutic electrical stimulation (TES) to the tibialis anterior and quadriceps muscles in six children with mild cerebral palsy.

In most studies, there has been no consistency in frequency of delivery, intensity, and amplitude of electrical stimulation, and therefore no standards have been formulated. These studies have been primarily focused on the lower extremity, where goals and treatment protocols differ from those for the upper extremity.

However, Pape et al. (1990) applied TES to the triceps and wrist extensors of 26 hemiplegic children, but no measurable change in function was reported. Baker et al. (1979) reported an increase in wrist and finger extension in 16 adult hemiplegic patients with unilateral flexor spasticity. In that study, patients received three 30-minute periods of electrical stimulation per day for 4 weeks. However, patients seen 1 and 2 months after the cessation of electrical stimulation had developed increased flexor contractures despite attempts to maintain range of motion with passive exercise and splinting.

On the basis of these previous studies, we decided to treat upper-extremity spasticity in patients with cerebral palsy using a combination of multiple treatment sessions of NMES to address the dynamic component of spasticity and dynamic orthotic traction to address the static component of spasticity. Static orthoses were also used at night to maintain the gain in length of the flexors while keeping the extensors in the resting and shortened position.

We hypothesized that this combined therapy would improve limb function. The dynamic bracing would produce an effective extrinsic stretch of the flexor musculature, an extrinsic shortening of the extensor musculature, and a strengthening of the intrinsic musculature. NMES applied to the antagonist muscles would reduce agonist muscle tone and increase strength of antagonist muscles.

In this retrospective cohort study, we assessed the effects of dynamic orthotic and static traction and NMES on upper-extremity spasticity in cerebral palsy patients.

## PATIENTS AND METHODS

### Patients

Twenty-six patients seen between August 1992 and July 1997 were initially considered for the study. From this initial group, one patient who required a heart pacemaker, five patients who did not comply with the protocol, and one patient whose family moved out of state were excluded from the study. The remaining 19 patients met the following criteria: between 3 and 21 years of age; with true spastic hemiplegia; with mild to moderate spasticity in the scapula, shoulder, and elbow region that allowed them to place the hand in the desired position in space; and with moderate to severe spasticity in the wrist and digits. They had good sensation in the affected extremities, and they demonstrated enough cognition to understand and to follow directions. The mean age of the children was 10 years and 3 months (range, 3 years and 4 months to 20 years and 11 months). There were ten boys (four with left hemiplegia and six with right hemiplegia) and nine girls (six with left hemiplegia and three with right hemiplegia).

The 19 patients included in this study were classified according to the Zancolli classification (Zancolli et al., 1983): 11 were classified as type III, seven as type IIb, one as type IIa, and none was classified as type I (Table 1).

### Electrical stimulation

The electrical stimulation system consisted of three parts: a stimulator unit (EMS 400, Skylark Device Company, Louisville, KY), electrodes, and connecting wires (Medi-Stim Inc., Delaware, OH) (Fig 1). The reusable, self-adhering, carbonized rubber electrodes were connected to the stimulator by leads that were snapped to the button of the electrode. The adhesive electrodes were placed on the dorsum of the forearm over the bellies of the wrist and finger extensor muscles at the distal and proximal positions. The electrical stimulator consisted of a dual-channel battery-powered device with a current output that could be set between 0 and 100 mA. The stimulus waveform consisted of biphasic symmetric rectangular pulses with a 200  $\mu$ sec duration. The pulse rate ranged between 40 and 60 pulses per

**Table 1—The Zancolli classification**

Type I	Complete extension of the fingers with wrist in neutral position or with less than 20° flexion
Type II a	Active extension of the wrist with fingers flexed
Type II b	No active extension of the wrist even with fingers flexed
Type III	No active extension of the fingers even with maximal wrist flexion

