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 hypertonic 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 Wiegener, 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 Wiegener, 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 (Siskin 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 loco-motor 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 μsec duration. The pulse rate ranged between 40 and 60 pulses per

<table>
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<th>Table 1—The Zancolli classification</th>
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<td>Type I Complete extension of the fingers with wrist in neutral position or with less than 20° flexion</td>
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<tr>
<td>Type IIa Active extension of the wrist with fingers flexed</td>
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<tr>
<td>Type IIb No active extension of the wrist even with fingers flexed</td>
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<tr>
<td>Type III No active extension of the fingers even with maximal wrist flexion</td>
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second to produce tetanic muscle contraction. The stimulus amplitude was adjusted between 30 and 40 mA to produce tolerable muscle contractions. During the training session with the parents, electrical stimulation was applied first on the clinician's arm, then on the arm of one of the parents, and finally on the patient's arm. A 2 second-ON ramp, 2 second-OFF ramp, 10 second-ON duty and 7 second-OFF duty cycle was selected to produce rhythmic muscle contraction. The stimulus amplitude threshold was determined by increasing the amplitude of the stimulus until muscles started to contract. The amplitude was then gradually reduced until no contractions were apparent. This amplitude threshold was then doubled and used routinely. If necessary, the amplitude was decreased to make contractions tolerable.

Dynamic orthotic traction

The orthotic device consisted of two parts: a wrist/hand unit and an elbow unit. The wrist/hand unit consisted of a thermoplastic rigid support that positioned the wrist in the extension described below. This unit also had an adjustable dynamic flat pan that locked the PIP and DIP joints in extended position, allowing finger movement only at the Metacarpophalangeal joints (Fig 2). To resist the spastic muscle, but not facilitate a spastic response (that is, a stretch reflex), the wrist unit was positioned 10° short of maximum extension stretch with a flat spreading hand portion to mobilize the metacarpophalangeal joints and block the proximal interphalangeal joints. The elbow unit had a dynamic dual hinge with adjustable tension (0-7.2 kg) and an adjustable lockout.

This stretching technique resisted tight spastic flexors while strengthening intrinsic musculature and gave the antagonist musculature the chance to shorten. The custom-fitted elbow orthosis (Ultraslim, Malvern PA) was used to alter the elbow flexion contracture and pronation deformity by placing the forearm in maximum supination and then applying the elbow extension unit. Velcro was used to prevent slippage of the orthosis during pronation (Fig 3). With this technique, we achieved maximum mechanical leverage on the pronator teres muscle and strengthened the biceps' ability to supinate the forearm.

Treatment

Neuromuscular electrical stimulation and dynamic bracing protocol

This home-based programme consisted of 1 hour per day of electrical stimulation with dynamic bracing. Treatment time could be divided into two 30-minute sessions or three 20-minute sessions (once in the morning, once at noon, and once in the evening), depending on the working schedules of the parents. After the parents were instructed, children were fitted with the wrist and elbow unit. Adhesive, reusable electrodes were then placed on the bellies of the wrist and finger extensor muscles. Once the amplitude was determined, the extensor muscles were stimulated for 10 seconds to obtain a tetanic contraction, followed by 7 seconds with no electrical stimulation. During the period with no stimulation, the patient was told to flex the fingers repeatedly so that the patient learned which muscles to contract to flex the fingers and prevent the co-contraction. At the beginning of the treatment, the rubber bands in the outrigger part of the dynamic brace were strong enough to extend the fingers, and then, as the extensor tendons became stronger, the strength of the rubber bands was gradually decreased. After each session, children were allowed to continue with their daily activities. The orthotic devices and the electrode positioning were reviewed every time the
Fig 3  (a) Attaching the unit with the forearm in maximum supination to alter pronation deformity, allowing (b) flexion and (c) extension of the forearm.

Fig 4  Wrist unit with dynamic flat pan connected to the dorsal outrigger with a Velcro® strap to maintain the stretching gained during the day.

extrinsic flexors and maintain the ROM gained during the day (Fig 4).

Assessment

The children were assessed using the Zancolli classification (Zancolli et al., 1983) at the start of the treatment and every month thereafter. Patients' compliance was also assessed using a subjective 1 to 5 scale; those patients who did not miss any appointment and followed the protocol as it was explained to them were scored 5, and patients who cancelled appointments and did not follow the protocol were scored 1.

RESULTS

Patients who complied with the programme showed a significant improvement in the quality of movement of the upper extremity. They were able to extend the wrist and fingers leading to better control and use of the hand (Fig 5). Those who received 4 or 5 in the compliance scale showed a quicker improvement, noticeable as early as 4 to 6 weeks of treatment. The mean duration of treatment was 15 months (range, 3-43 months). After treatment, none of the patients was classified as type III, none as type IIb, nine as type IIa, and ten as type I (Fig 6). Fourteen patients moved up two grades in the Zancolli classification. The mean age in this group was 10 years and 3 months (range, 3 years and 4 months to 19 years), and the mean duration of treatment was 16 months (range, 6-43 months). Two patients moved up only one grade: a young man aged 20 years and 11 months old and a girl aged 17 years. The young man moved out of the country after 3 months of treatment, and the girl had been in the programme only 4 months at the time the charts were reviewed. Three patients moved up three grades. The mean age in this group was 5 years
and 6 months (range, 3 years and 8 months to 6 years and 7 months), and the mean duration of treatment was 18 months (range, 10–29 months).

None of the patients reported any pain or injury related to the stimulation. Eight patients reported some discomfort while wearing the splint because it did not fit perfectly. This was relieved by reforming the thermoplastic arm support. All the patients tended to have reduced co-contractions. One patient in whom treatment was discontinued showed signs of regression of spasticity and had to begin the programme again.

**DISCUSSION**

The results of this retrospective study show that the use of NMES combined with dynamic orthotic traction during the day and static orthosis at night is a relatively quick and dramatically effective method for treating children with spasticity of the upper extremity due to cerebral palsy. The efficacy of our combined treatment method was confirmed by the clear improvement observed in the Zancolli classification and in hand function.

This study confirms previous studies in which electrical stimulation has been used successfully in the treatment of spasticity in hemiplegic patients (Kraljić and Vodovnik, 1977; Liberson et al., 1961; Stanić et al., 1978; Takebe et al., 1975; Waters et al., 1975). In previous studies, electrical stimulation has been applied on antagonist muscles of the lower extremity. There are only a few studies in which NMES and bracing have been used on the upper extremity. Carmick (1997) reported improved hand function and a reduction of spasticity using NMES and a static dorsal wrist splint in a patient with spastic hemiplegia. We found no studies in the English and Spanish medical literature about the use of combined NMES and dynamic orthotic traction to improve the function of the upper extremity.

In most patients, we could observe a notable improvement after 4 weeks, which encouraged the patients to...
Fig 6 Patient distribution according to the Zancolli classification before and after treatment.

continue the therapy. The parents and patients also reported increased ability to perform their activities of daily living.

We are still unclear about the duration of the effectiveness of the treatment, and we are administering a maintenance programme. Patients who were classified as Zancolli I or higher after treatment are put on a maintenance programme consisting of NMES and dynamic traction once every other week for 30 minutes. They are also instructed to wear the brace once a week at night. They are periodically monitored by their local therapist and reviewed every 6 months. The first patient treated has been on this maintenance programme for 5 years and 5 months and has not shown any sign of regression of spasticity. With this protocol, we have decreased the spasticity of the flexor pronator mass and strengthened the extensor supinator mass. Moreover, we have taught the children which muscles need to be fired to obtain flexion and extension of the fingers, thereby reducing the co-contraction.

Since the pathophysiology of spasticity is not clearly understood, the mechanism of action of NMES and dynamic traction is not known. It may be that by stimulating the muscles through NMES and dynamic traction, a natural muscle contraction is simulated which polysynthetically inhibits the agonist or spastic muscle in the reflex arc.

NMES treatment has limitations. The fact that the one patient in whom the therapy was discontinued started showing signs of regression of spasticity leads us to conclude that this is a long-term, even life-long treatment. Another disadvantage is that the therapy has to be administered two or three times every day, which may compromise compliance and thus the results.

Although this method of treatment for spasticity is relatively quick and dramatically effective, more research is needed to determine whether the improvement shown by our patients was due to the NMES, to the dynamic traction, or to the combination of both.

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References


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