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The effects of caregiver-supervised, home use of electromyographically-triggered electric stimulation on active dorsiflexion ROM of the ankle in children with spastic, diplegic cerebral palsy: a pilot study.

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Background and Purpose: The purpose of this pilot study was to measure the effect of electromyographically-triggered electrical stimulation (EMG-ES) on active dorsiflexion range of motion (ADROM) in a group of children with spastic cerebral palsy.

Subjects: The subjects were eight children with diplegic cerebral palsy between 8 and 14 years old.

Method: Each child participated in the study for eight weeks; a four-week no intervention period, and a four-week intervention period. ADROM was measured at the beginning and the end of the no-intervention period, and at the end of the intervention period. The intervention was a guardian-supervised program of EMG-ES for the dorsiflexors of one ankle. Results: There were statistically and clinically significant increases in ADROM during the intervention period versus the no-intervention period, but not between treated and untreated ankles.

Discussion and Conclusion: Our study suggests that home-based, caregiver-supervised EMG-ES could be a useful adjunct to traditional PT programs for children with spastic cerebral palsy if when one of the goals is the improvement of active dorsiflexion range of motion.

Introduction

The inability to actively and selectively dorsiflex the ankle is a common impairment among children with spastic diplegic cerebral palsy (CP).[1] There are a wide variety of therapeutic interventions in use to improve ankle function in this population, including active and passive exercise, bracing, participation in whole-body activities designed to improve overall ability to control and coordinate movement, electromyographic feedback, and electric stimulation.

The use of electromyographic (EMG) biofeedback has been suggested as a training tool to improve the ability to increase activation of weak and/or partially paralyzed muscles and/or to decrease the activation of muscles affected by spasm or spasticity without regard to specific diagnosis.[2] However, very few studies have examined reported on the the effects of EMG biofeedback on ankle function among children with spastic CP. In 1994, Colbourne, Wright and Nauman [3] reported that the

use of EMG biofeedback to train selective control of the gastrocnemius resulted in improved gait symmetry, and was associated with greater ankle power for push-off at the end of the stance phase. In 1998 Toner, Cook and Elder[4] reported improved active dorsiflexion ROM and strength after a six-week program that included EMG biofeedback to increase activation of the ankle dorsiflexors. In 2004, Dursun, Dursun and Alican reported that the use of EMG biofeedback to train selective activation of the plantarflexors and dorsiflexors in a group of 21 children with spastic CP significant resulted in significant increases in active dorsiflexion range of motion.[5]

The use of neuromuscular electric stimulation (NMES) has been suggested as an intervention for the purpose of increasing muscular strength and control of movement in a number of different populations.[6,7] In 2004, Kerr, McDowell and McDonough reviewed the literature related to the effects of electric stimulation on strength and function of children with CP.[8] They made a distinction between "therapeutic electric stimulation (TES), and "neuromuscular electric stimulation (NMES)". TES was described as a low-level, electrical stimulus applied at home during sleep with intensities and pulse rates too low to elicit tetanic muscle contractions; whereas NMES was described as the application of an electrical current with sufficient pulse rate and intensity to elicit tetanic muscle contractions. They concluded that evidence for the efficacy of NMES was stronger than that for TES, but noted that, "The scarcity of well-controlled trials makes it difficult to support definitively or discard the use of electrical stimulation." (p. 212).

Two recent studies have reported the effects of NMES applied to the ankle muscles of children with spastic CP while walking. Postans and Granat studied eight children and customized the locations and parameters of stimulation based on each child's gait defects. Only three of the eight children in their study showed significant improvement as determined by gait analysis.[9] Ho et al studied the effects of footswitch-triggered NMES of the gastrocnemius muscle on mechanical impulse and stiffness, stride length, cadence and walking speed in a group of 9 children with spastic cerebral palsy and six children without physical disabilities.[10] Subjects were fitted with a footswitch that turned the current on when they achieved foot-flat, and turned the current off when the swing-phase began. They reported that NMES increased the impulse generated during the push-off phase of the gait cycle among the children with CP, but that differences in the other variables were not statistically significant. Their discussion included speculation that this may have been due to the small sample size and limited statistical power, and the fact that measurements were taken during a single session, so that there was no opportunity for subjects to learn how to employ the increase in mechanical impulse to increase stride length, cadence and/or velocity.

Maenpaa et al employed a same-subject, repeated measures design to examine the effects of a four-week program of "sensory-level electric stimulation" of the tibialis anterior on active ankle dorsiflexion range of motion (ADROM) in a group of 17 children with cerebral palsy.[11] The "sensory level electric stimulation" was described as a biphasic, 300-microsecond duration pulse applied at pulse rates of 10 to 20 per second, and with and intensities too low to elicit visible muscle contractions, but high enough to elicit a tingling sensation. After a one-month intervention period, they reported a statistically significant increase in mean ADROM from 7.9 to 16.0 degrees.

Electromyographically-triggered NMES (EMG-ES) is a mode of treatment that combines EMG-biofeedback with NMES. The delivery of current in most commercially available NMES systems is controlled by timers that turn the current on and off for preset

intervals. Less commonly used are subject-controlled on/off switches as described in the article by Ho et al mentioned previously.[10] In contrast, EMG-ES is turned on, or triggered, when the user generates a predetermined magnitude of electromyographic activity. EMG-ES, therefore, has the potential to provide the benefits both EMG-biofeedback and NMES.

Bolton et al recently performed a meta-analysis regarding the use of EMG-ES in motor recovery following stroke.[12] Based on the results of seven experimental studies [13,14,15,16,17,18,19] that met the criteria they had established for inclusion they concluded that, "EMG-triggered neuromuscular stimulation causes improvements in arm/hand function capabilities during the acute, subacute, and chronic phases of recovery." (p. 125[12]).

Although the etiologies of stroke and cerebral palsy are quite different, the movement-related problems faced by people with both diagnoses are similar in many ways. Both are central nervous system lesions that frequently result in spasticity and difficulty with motor control. In 1991, Atwater et al studied the effects of a two-month, three times a week, 20 minute session of EMG-ES applied to the dorsiflexor musculature in a group of 10 children, aged 5 to 15.[20] Their results were inconclusive. Other than Atwater et al, there have been no other published studies regarding EMG-ES and children with CP.

Our review stimulated our curiosity as to the value of EMG-ES in the management of impairments associated with cerebral palsy. Because of the paucity of literature, we chose to focus our efforts on an easily measured, commonly occurring impairment with important clinical implications. Limited ADROM places children at risk for joint contractures, and often necessitates the use of an ankle-foot orthosis to prevent foot contact during the swing phase of gait. The specific purpose of this pilot study was to determine if the addition of caregiver-supervised, home use of EMG-ES to an ongoing program of physical therapy would improve active dorsiflexion range of motion in a group of children with spastic cerebral palsy.

Methods

Design Overview

We employed a pretest-posttest design for the study. The dependent variable (ADROM) was measured before a non-intervention period, at the end of the non-intervention period and the beginning of the intervention period, and at the end of the intervention period. All subjects demonstrated bilateral impairments in ADROM, but only one ankle was treated. Therefore we were able to compare measurements of ADROM at different times, and for the treated versus untreated ankles.

Setting and Participants

Subjects for this study were recruited with the assistance of several outpatient physical therapy centers in southeast Florida. Criteria for inclusion were: Age of 8 to 18 years, diagnosis of quadriplegic or diplegic spastic cerebral palsy (CP), ongoing participation in a physical therapy program that included a goal of increasing the active ankle dorsiflexion of both ankles, and permission of primary care physician. The protocol used for this study was approved by the Institutional Review Board of Florida

International University. Written consent for participation was obtained from the guardian of each subject and assent was obtained from each subject.

A total of 18 subjects agreed to participate in the study, but only 8 completed the protocol. The reasons for dropping out included: family travel, illness, hospitalization for surgical procedures, and/or failure to use the device as frequently as requested. None of the subjects dropped out because of operating difficulties or side-effects of the EMG-ES system. The eight subjects ranged in age from eight to 14, and included 4 boys and 4 girls.

Interventions

Each subject participated in the study for 8 weeks. During this time they continued their customary level of participation in physical therapy. The investigators traveled to the subject's home or to the location of their physical therapy to carry out the study. At the beginning of the eight-week period, the purpose and plan of the study was explained to the subjects and their caregivers, and active dorsiflexion range of motion (ROM) in a seated position with knees flexed to approximately 90 degrees was measured using standard goniometric technique.[21] Subjects were allowed three attempts; the greatest amount of active ROM was recorded. After recording of ROM, each subject continued with their usual course of physical therapy for four weeks with no intervention from this study.

At the end of the four week non-intervention period, active ROM measurements were repeated as described previously, and subjects and their caregivers were introduced to the Biomove 3000* EMG-ES system. Factory default settings were used for the stimulation. When triggered, the Biomove 3000* delivered a six-second period of electric stimulation consisting of 35 pulses per second of a 400 microsecond pulse-duration, biphasic square wave. This was followed by an enforced "off" period of 12 seconds, during which time it was impossible to trigger another stream of stimulating current regardless of EMG activity.

Caregivers could control the intensity of stimulation and the sensitivity of the EMG trigger with dials, as shown in Figure 1. Intensity was adjustable from 0 to 80 milliamperes, and sensitivity was adjustable so as to allow for initiation, or "triggering" of the stimulating current when the electromyographic signal reached a magnitude of 2 to 200 microvolts.

Caregivers were asked to use the Biomove 3000 (Curatronic Ltd, Israel) once a day for between 20 and 30 minutes. Caregivers were instructed to set the stimulation at the highest comfortable level, and the sensitivity of the EMG at the lowest level to that allowed for triggering during a maximal effort to produce active dorsiflexion. Caregivers were allowed to choose which ankle to treat during the course of the four-week intervention period. Four of the caregivers chose the ankle with greater limitations of ankle dorsiflexion range of motion (ADROM) and four chose the ankle with lesser limitation of ADROM.

The investigators chose the optimal electrode sites for stimulation based on published motor point charts for stimulation of the tibialis anterior and extensor digitorum longus, modified by a process of trial and error.[22] Placements that generated the strongest dorsiflexion with the least amount of current were chosen. A third reference electrode was placed midway between the two electromyographic (EMG) sensing and stimulating electrodes. The typical electrode placement is shown in Figure 2. Sites for placement of all three electrodes were marked with indelible pen, and guardians were asked to place the electrodes in the same location throughout the treatment period.

Investigators followed up via phone two days after the training session, answered any questions, and reviewed the instructions as needed. Caregivers recorded the day, time, and duration of EMG-ES use. Subjects who reported using the device fewer than five times per week for at least 20 minutes were eliminated from the study. At the end of the four week intervention period, ADROM measurements were repeated as described previously, and a brief, unstructured interview of the caregivers regarding the value of the EMG-ES trial was performed.

Statistical Analysis

SPSS for Windows 15.0 (SPSS Inc., 233 S. Wacker Drive, Chicago, Illinois 60606) was used to perform Wilcoxon signed-rank tests to assess the significance of differences in ADROM between treated and untreated ankles, and between measurements at three different times; at the beginning of the non-intervention period, the end of the non-intervention period, and the end of the intervention period. A p value < 0.05 was considered significant.

Role of the Funding Source

Curatronic LTD provided partial financial support for the study, and provided 20 ES-EMG systems for the study. **It had no role in the design, conduct, or reporting of the study.**

Results

At the beginning of the no-intervention period mean ADROM ranged from -35 to 10 degrees. By the end of the intervention period, which was also the beginning of the intervention period, 11 of the 16 ankles showed an increase in ADROM. Between the beginning and the end of the intervention period, 15 of the 16 ankles showed an ADROM increase.

ADROM measurements for each subject at the beginning and end of the non-intervention period and the end of the intervention period for the treated and untreated ankles of each subject are displayed in Table 1; means and standard deviations for treated and untreated ankles are displayed in Table 2. and displayed in Figure 3.

Results of the Wilcoxon signed-rank tests revealed that there were no significant differences in ADROM between treated and untreated ankles before the no-intervention period, at the end of the no-intervention period, or at the end of the intervention period. Differences in ADROM for both ankles between the beginning and the end of the no-intervention period were also insignificant. However, there were statistically significant differences in mean ADROM between beginning of the no-intervention period and the end of intervention period for both the treated ($p=0.012$) and untreated ($p=0.035$) limbs; and between the beginning of the intervention period and the end of the intervention period for both treated ($p=0.024$) and untreated ($p=0.018$) limbs. Results of the Wilcoxon signed-rank tests are displayed in Table 3.

At the end of the intervention period, all eight caregivers interviewed expressed the opinion that the intervention was useful. Five expressed disappointment that EMG-ES devices were not readily available, and three of those five expressed interest in

purchase of the device. None of the subjects reported discomfort, skin irritation, or difficulty operating the EMG-ES system during the course of the study.

Discussion

In our pilot study, a four-week period of guardian supervised, home-use of an electromyographically-triggered FES device (Biomove 3000) resulted in statistically and clinically significant improvements in ADROM of the treated ankle in a small group of children with spastic, diplegic cerebral palsy. Based on our review of the literature, we were not surprised by these results. We were surprised that intervention also caused a significant improvement in ADROM of the untreated ankles as well. This may be due to some combination of several different factors.

EMG biofeedback alone has been shown to be a useful tool for improving voluntary activation of muscles after in the presence of a variety of neurological disorders,[2] and sensory-level stimulation alone has been shown to improve active dorsiflexion ROM in a group of children with spastic CP.[11] Electric stimulation of sufficient intensities to elicit muscle contractions, also applied alone, has also shown to increase muscle strength and ROM in the presence of a wide variety of neurological disorders.[6] The intervention in this study provided biofeedback, sensory-level stimulation, and stimulation sufficient to generate muscle contractions, and it seems reasonable to assume that this combination would be at least as effective as any of the individual modes of treatment.

The use of EMG-ES might be viewed as imitating many of the features of the process of motor learning in people without disabilities; that is, attempts to activate a particular muscle are accompanied by sensations emanating from the muscle, and visual and proprioceptive feedback that the muscle is causing a particular movement. Because the motor learning is a function of the central nervous system as opposed to the muscle itself, improvements in ADROM of both the treated and untreated ankles occurred.

There was no attempt to blind the subjects and their caregivers to the purpose of the study, and knowledge that ankle dorsiflexion would be closely monitored may have improved motivation and effort that affected ADROM bilaterally. In addition, the intervention added at least 100 minutes per week of attempts at active dorsiflexion, above and beyond the regularly scheduled physical therapy program.

It is also possible that caregivers used the EMG-ES system on both ankles in spite of our instructions.

The limitations of this study should be considered when interpreting these results. The number of subjects was small, and the length of the intervention was brief. There was a large variance of initial ADROM, which would be expected in a group of children with spastic cerebral palsy. Additional research is recommended that would include larger, more homogenous groups, and comparisons of EMG-ES versus other interventions that might control for the amount of time and effort on the part of the subjects and/or their caregivers. Future research that examines the long-term effects of EMG-ES on gross motor function, the need for corrective surgery and orthotic devices, the ability to perform activities of daily living is also suggested.

In our study, a four-week course of home-based, guardian-supervised use of the Biomove 3000 significantly increased active ankle dorsiflexion range of motion.

This suggests that it would be a useful adjunct to traditional PT programs for children with spastic cerebral palsy when one of the goals is the improvement of active dorsiflexion range of motion. Furthermore, if children with spastic CP could develop the ability to active dorsiflex beyond neutral, it seems reasonable to suggest that they cease to require ankle-foot orthoses to allow for toe-clearance during the swing phase of gait, and their risk for development of plantar-flexion contractures would be decreased.

TABLES

Table 1. ADROM of each subject at the beginning and end of the no-intervention period and at the end of the intervention period.

Subject Number	Ankle	Beginning of No-Intervention Period	End of No-Intervention Period	End of Intervention Period	Change during the No-intervention period	Change during the intervention period
1	Treated	-10	-5	-8	5	-3
1	Un-treated	-24	-20	-10	4	10
2	Treated	-19	-13	2	6	15
2	Un-treated	-11	-8	5	3	13
3	Treated	-35	-25	-10	10	15
3	Un-treated	10	0	5	10	5
4	Treated	0	4	7	4	3
4	Un-treated	-10	-6	-4	4	2
5	Treated	-8	-10	-7	-2	3
5	Un-treated	-2	-4	-3	-2	1
6	Treated	-10	-14	-4	-4	10
6	Un-treated	-13	-6	-6	7	0
7	Treated	-21	-30	-6	-9	24
7	Un-treated	-22	-12	-1	10	11
8	Treated	-28	-14	-7	14	7
8	Un-treated	8	6	15	-2	9

Table 2. Mean and standard deviation of active ankle range of motion (ADROM) at the beginning and end of the no-intervention period, and at the end of the intervention period.

Ankle	**Mean (SD)at the beginning of No-Intervention Period**	**Mean (SD) at the end of No-Intervention Period**	**Mean (SD) at the end of Intervention Period**	**Mean (SD) change during the No-intervention period**	**Mean (SD) change during the intervention period**
Treated	-16.38 (12.08)	-13.38 (10.93)	-4.13 (5.55)	3.00 (7.16)	9.25 (8.61)
Untreated	-8.00 (12.57)	-6.25 (7.74)	0.13 (7.90)	4.25 (4.68)	6.38 (5.01)
Both ankles combined	-12.19 (12.43)	-9.81(9.72)	-2.00 (7.02)	3.63 (6.12)	7.81 (6.96)

Table 3. Results of the Wilcoxon signed-rank tests.

Matched Pairs	Mean Difference	Standard Error Mean Difference (±)	p value
Treated ankles, beginning and end of the no-intervention period	-3.00	2.68	0.23
Treated ankles, beginning of the no-intervention period vs end of the intervention period	.12.25	3.33	0.01*
Treated ankles, beginning of the intervention period vs. end of the intervention period	-9.25	3.04	0.02*
Untreated ankles, beginning vs, end of the no-intervention period	-1.75	2.21	0.29
Untreated ankles, beginning of the no-intervention period vs.end of the intervention period	-8.12	3.06	0.04*
Untreated ankles, beginning of the intervention period vs. end of the intervention period	-6.37	1.77	0.02
Treated vs. untreated ankles, at the beginning of the no-intervention period	-8.37	7.52	0.58
Treated vs. untreated ankles, at the beginning of the no-intervention period	-7.13	4.98	0.21
Treated vs. untreated ankles, at the end of the no-intervention period	-4.25	3.64	0.21

* $p < 0.05$

Figure legends

Figure 1. Control panel of the Biomove 3000

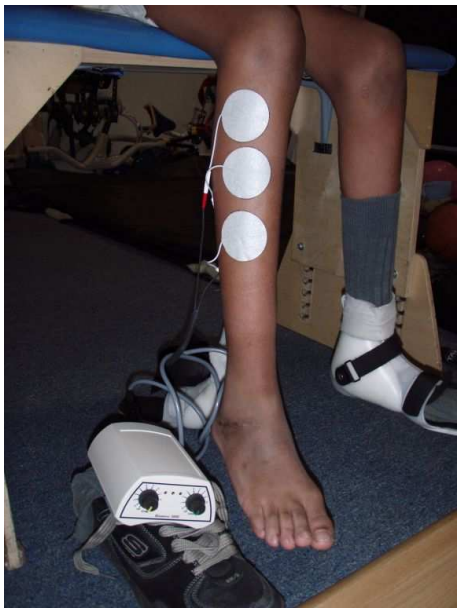

Figure 2. Typical placement of electrodes

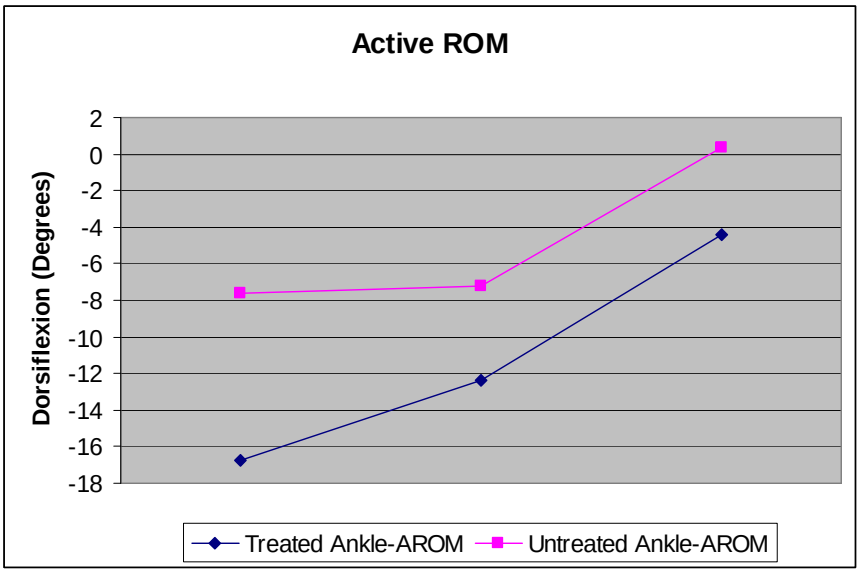

Figure 3. Mean ADROM of treated and untreated ankle before the no-intervention period, at the end of the no-intervention and beginning of the intervention periods, and the end of the intervention periods.

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