Efficacy of Neuromuscular Electrical Stimulation in Improving Ankle Kinetics During Walking in Children with Cerebral Palsy

Nerita N.C. Chan, MSc; Andrew W. Smith, PhD; Sing Kai Lo, PhD

Abstract: Neuromuscular electrical stimulation (NMES) applied to the triceps surae muscle is claimed to be effective in improving gait in children with cerebral palsy. The main aim of this study was to determine the effect of NMES on the triceps surae muscle in improving the gait and function of children with cerebral palsy. Twelve children with spastic diplegia or hemiplegia were recruited and randomly assigned to the two experimental groups. The period of the study was 8 weeks (2-4-2 week design). The initial 2 weeks was the control period, in which usual treatment was given to both groups of patients with a pre- and post-treatment assessment. The middle 4 weeks was the experimental period, in which the Treadmill+NMES group received NMES plus treadmill walking training and the Treadmill group underwent treadmill walking training only. Assessment was performed at 2-week intervals. The final 2 weeks was the carryover period, in which treatment to be tested was stopped and reassessment performed again at the end of week 8. An additional treatment and post-treatment assessment were given at weeks 2, 4 and 6 to test for the immediate effect of treatment. Altogether, eight repeated measures with three-dimensional gait analysis and five clinical measurements using the gross motor function measure (GMFM) were performed. Kinetic changes in ankle moment quotient (AMQ) and ankle power quotient (APQ) were not significant either immediately or cumulatively in both groups. Improvement in trend was observed in both groups immediately but not cumulatively. Using the GMFM, functional changes were detected in standing (GMST, \( p < 0.001 \)) and in walking (GMWK, \( p = 0.003 \)) using a “time” comparison. Significant interaction was also detected in GMWK using “treatment by time” \( p = 0.035 \). The difference between the two groups was not significant on “treatment” comparison of both GMST and GMWK. Both groups showed improvement in GMST and GMWK cumulatively but there was no difference between the two groups. The effects in both groups could be carried over to 2 weeks after interventions stopped. Both the Treadmill+NMES and Treadmill groups showed improvement in functional outcomes. The trend in the changes of the GMFM score suggested that improvements were greater in the Treadmill+NMES group. There was also a trend showing some immediate improvement in AMQ and APQ.

Key words: cerebral palsy, electrical stimulation, gait

Introduction

A common deviated gait pattern in children with cerebral palsy is equinus gait or toe walking. The presence of spasticity in the triceps surae muscle is the major cause of this equinus gait. It is believed that an imbalance in the ankle joint caused by a spastic calf muscle inhibits the development of the ankle dorsiflexors, making them weak and elongated [1]. Neuromuscular electrical stimulation (NMES) is one physiotherapy modality com-
monly used to decrease muscle spasticity [2–4] and to strengthen the weakened antagonist muscles [5–7]. Typically, NMES is applied to antagonist muscles (ankle dorsiflexor) to achieve an antispastic effect on agonist muscles (triceps surae) by way of reciprocal inhibition [8]. Previous studies suggest that applying NMES to ankle dorsiflexors should improve the gait pattern in children with cerebral palsy [9,10]. However, clinical experience and some literature indicate that this approach is not always successful. For instance, Carmick applied NMES to ankle plantarflexors, contrary to standard practice, and found improvement in foot posture, gait pattern, balance and efficiency of movement in children with cerebral palsy [11,12]. However, the efficacy of the application of NMES to agonist muscles has not been demonstrated to date. Thus, the purpose of the present study was to determine the efficacy of applying NMES to triceps surae muscles in order to improve ankle joint kinetics during walking in children with cerebral palsy.

**Ankle joint dynamics during gait**

Normally, the ankle joint will slightly dorsiflex at heel contact followed by plantarflexion as the foot is carefully lowered to the floor. This is achieved by slight concentric followed by eccentric contraction of the dorsiflexors. Once the foot is flat on the floor and the body is moving over the stationary foot, the ankle joint dorsiflexes as the tibia rotates forward over the foot. This motion is controlled eccentrically by the triceps surae muscle and continues until just after the heel rises slightly from the floor, at which point the triceps surae muscles reverse from contracting eccentrically to a more rapid and forceful concentric contraction. This period is referred to as “push-off” (or “pre-swing”). Push-off continues until the ankle is fully plantarflexed and, along with knee flexion, the foot leaves the ground, initiating the swing phase.

The observed kinematics of the ankle joint during stance are caused by the underlying kinetics. At initial heel contact, the ankle joint moment of force is slightly dorsiflexor, but for most of the stance, the moment of force is increasingly plantarflexor until the start of push-off, where it decreases rapidly to near zero when the foot leaves the ground (solid line, Figure 1). Ankle power throughout stance is mostly negative, indicating that the triceps surae works eccentrically absorbing energy. As push-off begins, a large positive power burst occurs, reflecting the forceful concentric contraction of the triceps surae as it works to propel the body forward (solid line, Figure 2).

**Ankle joint dynamics during gait in children with cerebral palsy**

The equinus gait pattern of children with cerebral palsy results from abnormal activities of the triceps surae complex throughout the stance phase of gait. During weight acceptance, overactivity of the ankle plantarflexor causes an abnormal first rocker [13], with the triceps surae preventing the heel from being the first point of contact with the ground. From mid-stance until late-stance (or “pre-swing”), as the triceps surae muscle is eccentrically stretched, there is an abnormal stretch reflex, resisting the lowering of the heel to the floor and producing the abnormal motion of the second rocker [13–15]. Due to the presence of plantarflexor weakness, push-off is prohibited and a crouched gait results. Children with hemiplegia have a lower average level of power generated by ankle plantarflexors [16]. The ankle on the hemiplegic side only provides one-third of total work, whereas two-thirds of the total concentric work is found in normal subjects [17].

In children with a spastic calf muscle, the advancement of the tibia over the foot in stance may result in a premature stretch of the spastic triceps surae and corresponding firing of the muscle [18], resulting in an “early heel raise” in mid-stance (Figure 3). A common pattern of “double bump” plantarflexor moment is usually found in children with cerebral palsy. The initial peak (a) will occur with greater magnitude before mid-stance than the peak (a’) occurring during terminal stance (dashed line, Figure 1). The pattern of plantarflexor moment in non-disabled children will only include the terminal peak. Any interventions that help to decrease the initial
peak and increase the terminal peak would improve the gait pattern in children with cerebral palsy. Thus, the ratio of initial peak over terminal peak \((a/a')\) or ankle moment quotient (AMQ) can be used to measure the change and, hence, the degree of improvement in ankle plantarflexor moment [19].

Similarly, double power generation occurs in children with cerebral palsy as opposed to substantial power absorption in the second rocker in non-disabled children [18,20]. The pattern of the total ankle power curve for children with equinus gait is typically “triphasic” (dashed line, Figure 2). An early initial deep trough of power absorption occurs as the child lands on his/her toes, followed by a premature burst of power generation in mid-stance \((b)\) before a final peak of power generation at terminal stance \((b')\). Ankle power quotient (APQ), the ratio of initial power generation over the final power generation in the ankle \((b/b')\), can be used to measure the change in ankle power after intervention [19]. A decrease in the ratio reflects an improvement in ankle joint power generation and absorption.

**Neuromuscular electrical stimulation**

NMES contributes to improving joint range of motion. Hazelwood and co-workers found an increase in passive range of movement at the ankle and ankle dorsiflexion strength in children with hemiplegic cerebral palsy after electrical stimulation of the anterior tibial muscles [10]. A randomized controlled trial of therapeutic electrical stimulation in children with cerebral palsy showed significant improvement in function with an increase in score on the gross motor function measure (GMFM) after treatment [21]. It is proposed that NMES applied to the calf muscle of children with cerebral palsy would not only improve the motor outcome, but also improve the child’s sensory awareness [22,23]. As an additional benefit, the sensory input provided by NMES incorporated in the task-specific approach in motor learning will enhance the pattern in the gait cycle.

**Methods**

**Subjects and design**

An ABA experimental design was adopted. Twelve subjects with spastic diplegia or hemiplegia aged 4–11 years were recruited and randomly assigned to one of two groups, Treadmill+NMES or Treadmill. All subjects met the inclusion criteria: spasticity over the triceps surae muscle of at least grade 1 on the Modified Ashworth Scale and an independent walker with equinus gait pattern. Subjects with fixed contracture and passive range of motion of the ankle of less than 10 degrees of dorsiflexion were excluded.

Table 1 shows the schedule for treatments, gait analyses and administration of the GMFM. Both groups were tested at the beginning and end of a 2-week period with their usual conventional physiotherapy to establish baseline measurements. Following this was a 4-week treatment period (weeks 3–6). Treatment frequency was three times per week. In weeks 4 and 6, a gait analysis was performed after the sixth treatment of that particular 2-week period. An additional treatment was performed in the gait laboratory session at weeks 2, 4 and 6, followed by post-treatment gait analysis to test the immediate effect. The carryover effect was tested 2 weeks after the treatment was terminated (week 8). Therefore, the whole study period was 8 weeks and involved five visits to the gait analysis laboratory with eight repeated measures.

**Procedures and measurement**

A portable NMES with remote control leads (Respond Select; Empi Inc, St Paul, MN, USA) was used in this study. NMES was applied to the triceps surae muscle while patients walked on a treadmill for a period of 15 minutes. A therapist triggered the stimulation during each stance phase of the affected limb(s). Children in the Treadmill group walked for 15 minutes with no NMES. The NMES parameters were set at a frequency of 30–35 pulses/second and an intensity of stimulation of visible muscle contraction. The speed of the treadmill was set according to the child’s usual walking speed (0.45–0.80 m/s). For gait analysis, a Vicon 370 (Vicon Motion Systems Ltd, Oxford, UK) with two AMTI force platforms (Advanced Mechanical Technology Inc, Watertown, MA, USA) was used to measure the kinetic change during gait. Five sets of kinetic data were taken in each session of gait analysis and the data averaged. The kinetic data were evaluated by comparing changes in AMQ and APQ. A decrease in either AMQ and/or APQ...
denotes improvement [19]. The kinetic data of both legs of children with spastic diplegia and the data on the affected leg for spastic hemiplegia were included in the analysis. In addition, children completed the “standing” and “walking” domains of the GMFM, which were designed to measure the change in function after intervention.

Statistical analysis
Data were analysed with SPSS version 10 (SPSS Inc, Chicago, IL, USA). Descriptive statistics were primarily used to analyse demographic data. Gait analysis data and GMFM scores were analysed by repeated measures analysis of variance. The change in the control period, immediate effect, cumulative effect and carryover effect of each group were compared.

Results
The demographic data of the subjects are summarized in Table 2. All subjects had 100% compliance with the treatment and tests.

Kinetic change
Immediate effect
The kinetic change in the AMQ immediately after treatment was compared between the Treadmill+NMES and Treadmill groups. Changes in AMQ at weeks 2, 4 and 6 are shown in Table 3 and Figure 4. Most tests showed a decrease in AMQ in both groups. However, no significant interactions were detected ($p = 0.533$). There were no significant changes on time comparison ($p = 0.146$) and treatment comparison ($p = 0.503$). However, from the trend in mean difference, we could see that both groups showed a positive immediate effect after treatment.

The same trend was observed in the APQ: the ratio decreased, showing a positive effect after treatment in both groups (Table 3, Figure 5). No significant interaction was found ($p = 0.138$). No significant changes on time comparison ($p = 0.402$) and treatment comparison ($p = 0.643$) were found. The only significant change was observed between treatments at week 4 ($p = 0.015$), meaning that there were significant differences between treadmill walking with NMES and treadmill walking alone.

Cumulative effect
The cumulative effect of NMES on the kinetic data during gait was assessed by comparing the AMQ and APQ at weeks 2, 4 and 6. There was no significant interaction ($p = 0.134$) in APQ. On time comparison, no significant difference was detected ($p = 0.199$); the only significant change was detected on treatment comparison between the two groups ($p = 0.037$), implying that the two groups were different, but there was no evidence of significant improvement over time in either group.

Functional change
The changes in function at weeks 2, 4 and 6 were significant in both walking and standing domains (Figure 6, Table 4). A significant interaction ($p = 0.035$) was observed in the walking domain for time comparison ($p = 0.003$), but there were no significant differences

Table 1. Schedule of tests

| Week | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|------|---|---|---|---|---|---|---|---|---|
| Therapy | N | N | TTT | TTT | TTT | TTT | N | N |
| Gait analysis | $X_1$ | $X_{TX_1}$ | $X_{TX_2}$ | $X_{TX_3}$ | $X_{TX_4}$ | $X_{TX_5}$ | $X_6$ | $X_7$ |
| GMFM | $G_1$ | $G_2$ | $G_3$ | $G_4$ | $G_5$ | $G_6$ | $G_7$ | $G_8$ |

$N =$ normal physiotherapy; $T =$ treatment (either Treadmill+neuromuscular electrical stimulation or Treadmill); $X_n =$ gait analysis performed in week number $n$; $GMFM =$ gross motor function measure; $G_n =$ GMFM administered in week number $n$.

Table 2. Demographic data of subjects

| Group             | Gender (M/F) | Diagnosis (diplegia/hemiplegia) | Limbs studied | Age (yr) |
|-------------------|--------------|---------------------------------|---------------|----------|
|                   |              |                                 |               | Range    | Mean ± SD |
| Treadmill+NMES    | 4/2          | 2/4                             | 8 ($n = 6$)   | 5–8      | 6.3 ± 1.03 |
| Treadmill         | 5/1          | 5/1                             | 11 ($n = 6$)  | 4–11     | 6.5 ± 2.74 |

$SD =$ standard deviation; NMES = neuromuscular electrical stimulation.
between treatments \( (p = 0.181) \). Paired t test showed that there were significant changes in both groups over time \( (p = 0.045 \text{ for Treadmill+NMES}; p = 0.014 \text{ for Treadmill}) \).

For the standing domain, no significant interaction \( (p = 0.212) \) was observed, the only significant change was noted on the time comparison \( (p < 0.001) \). However, from the trend of mean difference, there was greater improvement in the Treadmill+NMES group than in the Treadmill group. Moreover, on comparing the change at week 8 after treatment was stopped, no significant difference \( (p > 0.68) \) was detected. This non-significant difference, together with the fact that the mean values were almost identical at weeks 6 and 8, provided some indication that the effect gained at week 6 could be maintained and carried over to 2 weeks post-intervention.

**Discussion**

There was no obvious cumulative effect on the kinetics in the gait analysis but a positive effect was observed immediately after treatment. Both AMQ and APQ decreased, although the changes were not significant. This may be due to the small sample. As the change in gait was transient, we suggest that the effect of NMES on the triceps surae is due to a change in spasticity. This was also suggested by Comeaux et al in 1997 [24]: the constant state of activity in the spastic gastrocnemius may be interrupted by the on-off nature of the NMES so that the spasticity is decreased. The immediate positive effect of treadmill walking may be due to the decrease in spasticity caused by stretching of the calf. The forward momentum of the treadmill could decrease plantarflexor activity [25].
A significant difference between treatments ($p = 0.037$) was noted in the cumulative effect on the APQ, but not in the “time” and the interaction between “treatment and time”. This coincided with the finding that the Treadmill+NMES and Treadmill groups had different baseline APQs. In the Treadmill+NMES group, baseline APQ was more than 1.5, whereas in the Treadmill group, it was less than 1.0. A ratio above 1.0 means that the power generated in mid-stance is higher than that in terminal stance. Cupp et al showed that the magnitude of power generation in late stance (A2 event) increases as the subject’s age increases from 4 to 7 years [26]. No significant difference was seen between the ankle kinetics of the 8- to 10-year-old group and the adult group. Subjects in the Treadmill group were older than those in the Treadmill+NMES group, which may account for the difference in the kinetics variation between the groups.

Another drawback in comparison of kinetic changes is the use of AMQ and APQ. A ratio depends on two data to reflect a change. Either changing the first peak or the second peak may lead to a change in the ratio. Variation sometimes occurred in the peak power in the terminal stance phase, and some subjects used different maximum peak power in the push-off phase even in the same session of assessment. The variation in gait data may be due to various factors affecting the child’s performance, such as emotional state and concentration span. Gait analysis data are less repeatable in spastic children than non-disabled children [27].

The changes in functional outcomes measured using the GMFM standing and walking domains were significantly different. The score improved in both the Treadmill+NMES and Treadmill groups, and this improvement was maintained after treatment stopped. However, no statistically significant difference was found between the two groups. The functional items that changed were mainly balance items such as single-leg standing (items 57 and 58), single-leg hopping (items 82 and 83) and jumping (items 80 and 81). Improvement in these items means that the functional stability of the child improved.

Figure 6. Change in gross motor function measure.

NMES = neuromuscular electrical stimulation.

Table 4. Descriptive statistics of cumulative gross motor function measure (GMFM): walking (GMWK) and standing (GMST)

| Test no. | n  | Mean ± SD     | 95% Confidence interval |
|---------|----|---------------|-------------------------|
| GMWK    |    |               |                         |
| Treadmill+NMES | 2 | 8 | 77.588 ± 20.85 | 66.207, 88.968 |
|          | 3 | 8 | 79.238 ± 20.26 | 68.140, 90.335 |
|          | 4 | 8 | 83.163 ± 18.00 | 72.948, 93.377 |
| Treadmill | 2 | 11 | 88.618 ± 9.57 | 78.913, 98.324 |
|          | 3 | 11 | 89.655 ± 9.43 | 80.191, 99.118 |
|          | 4 | 11 | 89.909 ± 9.59 | 81.198, 98.620 |
| GMST     |    |               |                         |
| Treadmill+NMES | 2 | 8 | 89.725 ± 7.53 | 85.329, 94.121 |
|          | 3 | 8 | 90.688 ± 7.13 | 86.444, 94.931 |
|          | 4 | 8 | 92.638 ± 6.35 | 88.669, 96.606 |
| Treadmill | 2 | 11 | 93.427 ± 4.4  | 89.679, 97.176 |
|          | 3 | 11 | 93.436 ± 4.4  | 89.818, 97.055 |
|          | 4 | 11 | 94.855 ± 4.46 | 91.47, 98.239 |

SD = standard deviation; NMES = neuromuscular electrical stimulation.
acceleration phases [29]. Moreover, the improvement in balance items was obvious clinically, as reported by both parents and therapists. Motor learning on walking with a treadmill may also contribute to the functional gain.

**Conclusions**

NMES of the triceps surae muscle had a positive effect on gait and function. Improvement was observed in AMQ and APQ immediately but not cumulatively. The changes were noted from the trend as no statistical significance could be proved because of the small sample size. The change in the functional outcome was more dominant in this study. Both GMFM standing and walking domains were significantly different on “time” comparison (p < 0.003). Both groups showed improvement but there was no significant difference between the two groups. The effect of functional gain could be maintained to 2 weeks after the intervention stopped. Further study of the mechanism of effect in functional electrical stimulation and the effect of treadmill walking is beneficial for evidence-based practice.

**References**

1. Renshaw TS, Green NE, Griffin PP, et al. Cerebral palsy: orthopaedic management. *J Bone Joint Surg* 1995;77A:1590–606.
2. Alfier V. Electrical treatment of spasticity. *Scand J Rehabil Med* 1982;14:177–82.
3. Menkveld SR, Wuijm JR, Ancheta B. Functional electrical stimulation in pediatric patients with spasticity. *Orthop Trans* 1986;10:376–7.
4. Seib TP, Price R, Reyes MR, et al. The quantitative measurement of spasticity: effect of cutaneous electrical stimulation. *Arch Phys Med Rehabil* 1994;75:746–50.
5. Enoka RM. Muscle strength and its development: new perspectives. *Sports Med* 1988;6:146–68.
6. Delitto A, Brown M, Strube MJ, et al. Electrical stimulation of quadriceps femoris in an elite weight lifter: a single subject experiment. *Int J Sports Med* 1989;10:187–91.
7. Delitto A, Snyder-Mackler L. Two theories of muscle strength augmentation using percutaneous electrical stimulation. *Phys Ther* 1990;70:158–64.
8. Levin MF, Hui-Chan CWY. Relief of hemiparetic spasticity by TENS is associated with improvement in reflex and voluntary motor functions. *Electroencephal Clin Neurophysiol* 1989;2:131–42.
9. Dubowitz L, Finnie N, Hyde SA, et al. Improvement of muscle performance by chronic electrical stimulation in children with cerebral palsy. *Lancet* 1988;i:587–8.
10. Hazelwood ME, Brown JK, Rowe PJ, et al. The use of therapeutic electrical stimulation in the treatment of hemiplegic cerebral palsy. *Dev Med Child Neurol* 1994;36:661–73.
11. Carmick J. Clinical use of neuromuscular electrical stimulation for children with cerebral palsy. Part 1: lower extremity. *Phys Ther* 1993;73:505–13.
12. Carmick J. Managing equinus in children with cerebral palsy: electrical stimulation to strengthen the triceps surae muscle. *Dev Med Child Neurol* 1995;37:965–75.
13. Gage JR, Minnesota P, Deluca PA, et al. Gait analysis: principles and applications. *J Bone Joint Surg* 1995;10:1607–23.
14. Perry J. Gait Analysis: Normal and Pathological Function. Thorofare, NJ: Slack Inc, 1992.
15. Ounpuu S, Gage JR, Davis RB. Three dimensional lower extremity joint kinetics in normal pediatric gait. *J Pediatr Orthop* 1991;:11:341–9.
16. Olney SJ, MaPhail HA, Heddien DM, et al. Work and power in hemiplegic cerebral palsy gait. *Phys Ther* 1990;70:431–8.
17. Brown J, Walsh EG, Wright GW. Neurophysiology of lower-limb function in hemiplegic children. *Dev Med Child Neurol* 1991;33:1037–47.
18. Gage JR. Gait Analysis in Cerebral Palsy. London: MacKeith Press, 1991:101–17.
19. Boyd RN, Platsios V, Starr R, et al. Biomechanical transformation of the gastroc-soleus muscle with botulinum toxin A in children with cerebral palsy. *Dev Med Child Neurol* 2000;42:32–41.
20. Rose SA, Deluca PA, Davis RB, et al. Kinematic and kinetic evaluation of the ankle after lengthening of the gastrocnemius fascia in children with cerebral palsy. *J Pediatr Orthop* 1993;13:727–32.
21. Steinbok P, Reiner A, Kestle JRW. Therapeutic electrical stimulation following selective posterior rhizotomy in children with spastic diplegic cerebral palsy: a randomized clinical trial. *Dev Med Child Neurol* 1997;39:515–20.
22. Bertoti DB, Stanger M, Betz RR. Percutaneous intramuscular functional electrical stimulation as an intervention choice for children with cerebral palsy. *Pediatr Phys Ther* 1997;9:123–7.
23. Carmick J. Guidelines for the clinical application of neuromuscular electrical stimulation (NMES) for children with cerebral palsy. *Pediatr Phys Ther* 1997;9:128–36.
24. Comeaux P, Patterson N, Rubin M, et al. Effect of neuromuscular electrical stimulation during gait in children with cerebral palsy. *Pediatr Phys Ther* 1997;9:103–9.
25. Gardner MB, Holden MK, Leikaukas JM, et al. Partial body weight support with treadmill locomotion to improve gait after incomplete spinal cord injury: a single-subject experimental design. *Phys Ther* 1998;78:361–74.
26. Cupp T, Oeffinger D, Tykowskci C, et al. Age-related kinetic changes in normal pediatrics. *J Pediatr Orthop* 1999;19:475–8.
27. Steinwender G, Saraph V, Scheiber S, et al. Intrasubject repeatability of gait analysis data in normal and spastic children. *Clin Biomech* 2000;15:134–9.
28. Sutherland DH, Cooper L, Daniel D. The role of the ankle planter flexors in normal walking. *J Bone Joint Surg Am* 1980;62:354–63.
29. Katoh M, Mochizuki T, Moriyama A. Changes of sagittal-plane ankle motion and ground reaction force (fore-aft shear) in normal children aged four to 10 years. *Dev Med Child Neurol* 1993;35:417–23.