Indirect effect of electrical stimulation of tibialis anterior on gastro-soleus muscles in children with spastic hemiplegic cerebral palsy

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ABSTRACT

Objectives: To investigate the effects of tetanic faradic stimulation in an antagonist muscle (tibialis anterior) on agonist muscles (gastro-soleus). In addition, to show the effects of maintaining agonist muscle strength on antagonist spastic muscle group to improve gait parameters in children with spastic hemiplegia.

Methods: We carried out the study at Pamukkale University, School of Physical Therapy, Denizli, Turkey between June 2001 and December 2002. We included 16 ambulant cerebral palsied children with a mean of age 6.25 ± 2.89 years in the study. Each subject practiced a 20-minute electrical stimulation session plus neurodevelopmental approach (Bobath Technique) once a day, 4 days a week for 5 weeks. We performed faradic tetanic stimulation to stimulate and strengthen the tibialis anterior (antagonist muscle). We performed all tests, including goniometric measurement, the Modified Ashworth Scale, and gait analysis at the start of the treatment program, after the program, and one month after first follow up.

Results: After treatment, we found significant improvements in goniometric measurements and Modified Ashworth Scale in comparison with the baseline measurements ($p<0.001$). However, the improvements did not continue after the first month of treatment ($p>0.05$). The results of the gait analysis showed only a significant difference concerning step width ($p>0.05$).

Conclusion: The results suggest that tetanic faradic stimulation was effective in improving aspects of ankle function, decreasing muscle tone and increasing range of motion in children with hemiplegic cerebral palsy during a physical therapy program.

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Cerebral palsy (CP) is a disorder of movement and posture resulting from a defect or lesion of the immature brain before the second year of life.[1-3] It is characterized by pathological changes especially in the musculoskeletal system, and is associated with incoordination movements, abnormal postural defects, and decreased perception and sense. It often manifests as spastic hemiplegia, spastic diplegia or spastic quadriplegia in the athetoid form, associated with basal ganglia involvement involuntary twisting movement of one or all extremities.[3-6] Hemiplegia is the second most common syndrome seen in preterm infants and the most common in term infants. Children with hemiplegia are an able, self-sufficient group, achieving standing and walking by 2-3 years at the latest. They tend to reject the affected side and to lead and lean towards the unaffected side. Body asymmetry and spasticity can result in unnecessary limitation, discomfort, gait problems and deformity.

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Children with spasticity show coactivation of agonist and antagonist muscles and exaggerated stretch reflexes on either one or both sides of the body. The following signs characterize spastic CP: 1. Increased muscle tone and increased stretch reflexes; 2. Exaggerated deep tendon reflexes; 3. Positive Babinski signs; 4. Loss of control of voluntary movement; 5. Weakness marked by increased tone and spasticity.[7] In the treatment of infants and children with spastic CP, the main goal is to inhibit the muscle spasm to gain voluntary movement. It is a cornerstone for improving the child's problem in neurological, motor developmental, biomechanical or functional terms. Health providers, especially physical therapists who work with spastic cerebral palsied children, must also pay attention to the neurological and developmental aspects of muscle spasm.[8] We aimed to investigate the effect of tetanic faradic stimulation in the antagonist muscles of spastic muscles (gastro-soleus) on the affected side of children with spastic hemiplegia (SH) secondary to CP. In addition, to show the effects of maintaining agonist muscle strength on the antagonist spastic muscle group to provide normal gait parameters in children with SH.

Methods. The current study was carried out in Pamukkale University, School of Physical Therapy and Rehabilitation, Denizli, Turkey between June 2001 and December 2002 under supervision by trained physical therapists working in the Pediatric Neuro-Rehabilitation Unit. Sixteen children (8 boys and 8 girls) with an average age of 6.25 ± 2.89 years were selected among 62 cerebral palsied children who are registered members of the Pediatric Neuro-Rehabilitation Unit at Pamukkale University. Inclusion criteria were: 1. A diagnosis of SH, 2. Moderate spasticity of the ankle flexors, 3. Able to walk independently without physical or equipment aid, 4. Sufficient cognitive function demonstrated to understand the requirements of the study, 5. No fear of electrical stimulation. All subjects' parents gave their informed consent for participation. The subjects of this study were 4 children with left SH and 12 children with history of right SH. The participants medical charts were reviewed and preliminary data including the child's name, nick name, age, and gender were recorded. Demographic and physical data belonging to the sample are shown in **Table 1**.

The electrical stimulation system consists of 3 parts: 1. Stimulator unit; 2. Electrodes (active and passive); 3. Connecting cables. The electrodes are connected to the stimulator unit by leads that are snapped to the button of the electrode. The stimulator operates in cyclical mode for a preset length of time. The stimulator provides tetanic faradic current to stimulate human muscles. Surface electrodes were placed and fixed with Velcro on the tibialis anterior muscle (TAM) of each subject's affected side. The TAM was selected as the antagonist muscle for the agonist spastic gastro-soleus muscles (GSM). Stimulation patterns were created using tetanic forms of the faradic current. The tetanic form was generated by modulating pulse duration up to 1 ms and pause duration up to 2 ms. The frequency of stimulation was 80 Hz for subject's TMA. Stimulation levels for the TMA were set to allow the maximal contraction, which could be tolerated by the subjects during the treatment session. Each subject practiced a 20-minute stimulation session 4 days (once a day) a week. During the stimulation session, each subject was in a long sitting position on the treatment table. A 20-minute stimulation session was performed by a physical therapist. After stimulation calibration, 2 conditions (stimulation on and off) were controlled automatically by the stimulator. All subjects participated in a daily neurodevelopmental physical therapy program based on the Bobath approach. The program was applied by a physiotherapist with at least 3 years experience and qualified in Bobath technique. Each physical therapist was instructed to continue their baseline physical therapy program unchanged throughout the study. The spastic hemiplegic cerebral palsied children (SHCPC) did not receive any other therapy during their time in the experimental program with the exception of medication they were already taking and physician visits.[9-11] Each subject was evaluated by a physical therapist and all tests were performed at the beginning of the stimulation program and repeated at the end of the program and one monthly intervals after the first follow-up. The effects of a 5-week stimulation program were assessed through spasticity, gait analysis, and goniometric assessment.

The Modified Ashworth Scale (MAS), which measures a patient's muscle spasticity in a 6-point scale. The lower score indicates less muscle spasticity. Each subject was assessed in the supine position and score was recorded according to the MAS.[12-16] Each subject was also evaluated concerning walking parameters, such as support surface, step length, step width, heel strike, and cadence. Each subject was allowed to try a trial walking period and was informed of the test procedures. The 5-meter walkway was covered with white colored powder before gait analysis. Each subject was asked to walk without shoes and with underwear only within the 5-meter walkway independently, and encouraged to finish the test. Gait parameters, including support surface, step width, and step length were measured using a tape and scores were recorded in centimeter (cm). Cadence was also assessed over one minute periods on ground surface (stable and smooth) and at the

Table 1 - Demographic data of the studied children (n=16).

Variable	Mean ± SD	Min -Max	Median
Age (years)	6.25±2.89	3 - 12	6
Mass (kg)	21.62±8.64	12 - 40	19.5
Height (cm)	110.75±18.58	88 - 140	113
The MAS score (at the baseline)	1.93±0.61	1.33 - 3	2
Gender	n (%)		
Male	8 (50)		
Female	8 (50)		
Affected side	n (%)		
Left	4 (25)		
Right	12 (75)		

MAS - Modified Ashworth Score, SD - standard deviation, Min - minimum, Max - maximum

end of the one minute period, the stopwatch was turned off and the total number of steps recorded.[17,18] Standardized clinical (universal) goniometry was used to measure dorsi flexion range of motion (ROM) of the ankle joint on the affected side. Each subject was assessed in the long sitting position (with back support) and measurement of ROM of the ankle joint (dorsi flexion) was performed passively. The measurement was repeated 3 times, and the average ROM in degrees was measured with the full-circle goniometry placing the ankle joint in the neutral position (00), following which ROM of dorsi flexion was measured passively.[19,20]

The results were analyzed using SPSS for windows, 2000 (version 10). The results obtained from the study (before treatment, after treatment, and first follow-up after one month) were compared to look for within group change over time among before treatment, after treatment, and one-month follow-up. Paired difference t test (Wilcoxon Test) were used to obtain final findings. The alpha level was set at 0.05 because of the exploratory nature of the present study.[21-22]

Results. **Table 1** illustrates the demographic data of the study participants. After treatment, the results of the goniometric measurement of ankle joint dorsi flexion on the affected side showed a high significant improvement. However, the improvement after treatment did not continue one month after first follow-up ($p<0.001$). When the considerations after the treatment, and at the end of the control period, compared with the considerations before the treatment in the MAS were examined, the spasticity in the plantar flexor muscle of the ankle joint (agonist spastic muscle) showed a highly significant decrease from 1.93 to 1.41 ($p<0.001$). When the gait analysis results are revised, according to the considerations before the treatment, the end of treatment and one month later, only a significant difference was detected regarding step width ($p<0.05$). However, the difference between before and after treatment was not observed one month later ($p>0.05$). Namely, the stimulation program did not have any positive effect on gait parameters (**Table 2**).

Discussion. Cerebral palsy causes disturbances of voluntary motor function and produces a variety of symptoms, such as abnormal posture, and loss of sense and perception.[5] Abnormalities within the spinal cord can increase spasticity and pain can exacerbate it. Spasticity associated with CP can lead to musculoskeletal complications, including contractures, deformities, or subluxation and so forth.[23,24] Locomotion of spastic subjects, for example, SHCPC, requires more energy than that of healthy subjects.[25,26] In spastic and paretic patients,

Table 2 - Means of all outcome variables at pretest (before treatment), post-test (after treatment), and one month after first follow-up.

Variable	Pretest Mean ± SD	Post-test Mean ± SD	Follow-up one month later Mean ± SD	Paired test *p*-value*
The MAS score	1.93±0.61	1.47±0.57	1.41±0.43	<0.001
Goniometric measurement				
Degree of dorsiflexion on the affected side	11.93±93	19.56±7.1	19.25±8.38	<0.001
Gait parameters				
Support surface (cm)	9.06±3.29	10.43±2.6	10.43±3.07	<0.05
Step length (cm)	66.56±17.83	68.75±16.55	69.43±18.79	NS
Step width (cm)	32.37±10.07	33.56±9.55	32.93±9.78	NS
Cadence (steps/min)	115.5±18.69	115.25±16.47	115.5±15.17	NS

MAS - Modified Ashworth Score, SD - standard deviation, *$p<0.05$ - significant difference, NS - not significant

motor control is disturbed and the efficiency may be decreased as a result of inappropriate muscle activation, namely, muscles could expend useless energy during excessive co-contraction.[27] Gait deviation, that is most often related to spastic paralysis, is common among CP children.[25,28] In a study by Cavlak and Kavlak,[29] it was found that spastic type CP lead to ankle-foot deformities much more than other types of CP. They also found a high percentage of equinovarus deformity resulting from SH in children with CP. Deformity to lower extremities decreases mobility and functional independence resulting in a decrease in standing, walking, running, climbing stairs in the CP children.[30] Failure to contact the floor with the heel at the onset of stance frequently is observed in persons with CP.[31,32] Perry et al[28] showed that the reduced movement in healthy subjects was accompanied by an increase in electromyographic activity of the soleus and medial gastrocnemius. They suggest that clinical decisions concerning the necessity of therapeutic intervention to reduce equinus should consider not only the effect on internal movements, but also the anticipated changes to the muscles' active and passive force generating capability. In ambulatory children with CP, equinus deformity is a well-recognized challenge in orthopedic treatment. A force foot landing and a small area of support adversely affects stability during the stance phase of gait.[33-35] Pierce et al[36] also approved that the children with CP demonstrate deficits in gait as compared with age-matched able-bodied peers. As CP cannot be cured, most children who have any of type of CP receive multimodal therapy – for example, physical, occupational and speech therapies; orthopedic surgery; spasticity management and special educational support services. From infancy to adulthood, physical therapy for children with CP focuses on the prevention of disability by minimizing the effects of functional limitation and impairment, preventing or limiting secondary impairments and helping the child compensate for function when necessary. Achieving these goals involves the promotion and maintenance of musculoskeletal integrity, prevention of secondary deformity, the enhancement of optimal postures and movement to promote functional independence and optimal levels of quality of life.[5,7,30]

The purpose of our study was to determine the effect of tetanic faradic stimulation applied to the TMA (antagonist to the GSM) during a 5-week physical therapy program on spasticity, range of motion and gait parameters in SH children. It is well established that children with SH CP demonstrate deficit, namely, plantar flexor spasticity and plantar flexor contractures.[28,33,37,38] That is why, all physical therapists, who work in pediatric rehabilitation should consider plantarflexor spasticity and contractures. Functionally, toe-walking resulting from GSM spasticity or contracture is associated with premature and prolonged muscle activity of the ankle plantar flexor spasticity and contractures. Stance stability is compromised as a result of the reduced portion of the foot in contact with the ground.[28,31,39,40] These functional limitations often result in reduced velocity and shorter stride length. As known, there are several studies showing the positive effect of electrical stimulation on spastic muscle in various disorders, such as CP, stroke, multiple sclerosis, and so forth.[41-45] However, Carmick[46,47] used neuromuscular electrical stimulation to strengthen spastic calf muscles in children with CP. He found that gait, balance and passive ankle ROM were to improve, while spasticity did not increase.[46,47] In a study by Hazlewood,[48] it was found that therapeutic electrical stimulation has a positive effect on knee and ankle motion (passive ROM) in children with hemiplegic CP. The data obtained from our study also suggests that tetanic faradic stimulation improved aspects of ROM of the ankle joint for example dorsiflexion, muscle tone, and gait parameters in children with SH secondary to CP. Stimulation of the tibialis anterior resulted in a more dorsi-flexion movement and less spasticity in gastro-soleus muscles. The improvements were observed during the treatment program and immediately after the program. However, one month after the first follow-up, the improvements were not observed. Namely, the electrical stimulation provides a temporary effect on muscle spasticity and ROM. Improvements in the ankle joint with application of stimulation are temporary. To our knowledge based on the literature, a permanent improvement has not previously been shown. Yet, we think that the electrical stimulation can be used to increase the muscle strength (antagonist), and ROM during a neurodevelopmental rehabilitation program in order to gain functional independence in children with SH. Beck[49] and Pape[50] also support that this type of sensory stimulation which increases the awareness of the involved extremity, thereby improving function. However, Steinbok et al[51] point out that the Botulinum toxin A, intrathecally baclofen pump and selective posterior rhizotomy may be beneficial in children with spastic CP to reduce tone and increase ROM, whereas functional improvement is achieved to only a limited extent without adding other treatments such as intensive physiotherapy, orthoses, or electrical stimulation. However, the mechanism behind these improvements requires additional studies. Controlled investigations are warranted to determine the efficacy of tetanic faradic stimulation applied to the tibialis anterior to gain some indirect effects on gastro-soleus muscle in SHCPC.

Finally, the increase in normal joint movement, namely, dorsiflexion, and the decrease in spasticity according to the MAS are obtained with the practice of tetanic faradic stimulation at the end of the study, however, the improvement did not continue one month after follow-up. Toe walking is an obligated gait for persons with calf muscle spasticity or primitive control that prevents heel contact with ground. Based on experience and gait studies of patients impaired by CP-induced hemiplegia, most physical therapists and clinicians have concluded that restoration of a heel-toe gait improves the patients function. In our pediatric rehabilitation unit, we sometimes use electrical stimulation to reduce plantarflexor muscle spasticity in these CP children scoring 2 and below according to the MAS. We believe that the appropriate combination of intervention must be considered to prevent and manage deformities and to delay and prevent arthritis, pain, progressive deformity, and contracture in order to facilitate ambulation, reduced pain, decreased muscle spasticity and an increase in quality of gait in CP children, especially the spastic type. Hence, tetanic faradic stimulation, although a temporary effect, can be used to reduce muscle spasticity and to increase ROM in pediatric neuro-rehabilitation units.

This study shows that the tetanic faradic stimulation applied to TMA in SH children had temporarily an indirect effect on gastro-soleus muscles. It strengthens the view that the muscle spasticity negatively affects ROM and gait parameters in CP children with SH. Further investigations should be focused on understanding the mechanism of spasticity and factor related to spasticity, and future treatments directed on decreasing spasticity.

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