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Does electrical stimulation reduce spasticity after stroke? A randomized controlled study

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Objective: To investigate the therapeutic effect of electrical stimulation on plantarflexor spasticity in stroke patients.
Design: A randomized controlled clinical trial study.
Setting: Rehabilitation clinic of Semnan University of Medical Sciences.
Subjects: Forty stroke patients (aged from 42 to 65 years) with ankle plantarflexor spasticity.
Intervention: Fifteen minutes of inhibitory Bobath techniques were applied to one experimental group and a combination of 9 minutes of electrical stimulation on the dorsiflexor muscles and inhibitory Bobath techniques was applied to another group for 20 sessions daily.
Main measures: Passive ankle joint dorsiflexion range of motion, dorsiflexion strength test, plantarflexor muscle tone by Modified Ashworth Scale and soleus muscle H-reflex.
Results: The mean change of passive ankle joint dorsiflexion in the combination therapy group was 11.4 (SD 4.79) degrees versus 6.1 (SD 3.09) degrees, which was significantly higher ($P = 0.001$). The mean change of plantarflexor muscle tonicity measured by the Modified Ashworth Scale in the combination therapy group was −1.6 (SD 0.5) versus −1.1 (SD 0.31) in the Bobath group ($P = 0.001$). Dorsiflexor muscle strength was also increased significantly ($P = 0.04$) in the combination therapy group (0.7 ± 0.37) compared with the Bobath group (0.4 ± 0.23). However, no significant change in the amplitude of H-reflex was found between combination therapy (-0.41 ± 0.29) and Bobath (-0.3 ± 0.28) groups.
Conclusion: Therapy combining Bobath inhibitory technique and electrical stimulation may help to reduce spasticity effectively in stroke patients.

Introduction

Upper motor lesion may produce muscle spasticity which increases the resistance against normal movements.[1] As spasticity may disturb walking and functional abilities of patients,[2] there is a general agreement that its treatment is important.[3] Various treatments have been recommended to reduce spasticity, including surgical, medical and physiotherapy techniques.[4–6] Methods such as drug therapy, chemical nerve block or neurosurgical treatments may reduce spasticity but may cause muscle weakness or paralysis.[7] The aims of physiotherapy techniques used for the treatment of spasticity are to favour sensorimotor recovery, which leads to optimal independence in daily life activities.[8] For stroke

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and head injury patients there are several techniques, sometimes based on opposing principles.[9] In physiotherapy, various methods have been employed for spasticity reduction, such as Bobath inhibitory techniques that may reduce the activity of stretching reflex and spasticity.[10,11] It has been stated that the Bobath concept tries to inhibit spastic paralysis[12] and the associated reactions to improve the voluntary movement of limbs with the ultimate goal of enabling exercises in a functional situation.[9]

Another method for reducing spasticity is neuromuscular electrical stimulation over the agonist or antagonist muscles of spastic muscle.[2,13] There is some evidence that electrical stimulation of the antagonist muscles can reduce spasticity immediately following treatment.[7,13,14] It has also been claimed that spasticity reduction by this method is achieved without any muscle weakness or paralysis.[7] However, there are controversial reports about the spasticity reduction effect of electrical stimulation.[15,16] Bogataj *et al.* found that neuromuscular electrical stimulation may increase sensory inputs into the central nervous system and so accelerate nervous plasticity and lead to faster motor learning.[5] It has been claimed that electrical stimulation may reduce muscle tonicity via the reduction of the stretching reflex, causing lower spasticity and allowing a larger range of motion,[17,18] and preventing soft tissue stiffness and contracture.[19,20] However, as there has been no study on the combination effect of these methods, and also controversial reports about the effectiveness of electrical stimulation on spasticity rehabilitation, this study has been designed to investigate the effectiveness of combination therapy of Bobath and neuromuscular electrical stimulation methods on spasticity in spastic stroke patients.

Material and methods

The study was approved by the ethical committee of the Semnan University of Medical Sciences. Forty stroke patients, ranging in age from 42 to 65 years (average 55 years) with upper motor neuron lesion and ankle plantarflexor spasticity were recruited from the neurology clinic of the Semnan University of Medical Sciences and participated voluntarily in the study. Exclusion criteria included sensory deficit or taking medicine for reducing muscle tonicity.

A computer-generated randomization list was drawn up by the statistician for each group. It was given to the physiotherapy department in sealed numbered envelopes. When the subjects qualified to enter the study and had signed their informed consent forms, the appropriate numbered envelope was opened at the reception; the card inside indicated the subject's allocation to either the Bobath or the combination therapy group. This information was then given to the physiotherapist to administer the appropriate intervention (Figure 1).

Intervention

The subjects were randomly assigned to one of the two experimental groups: combination therapy (Bobath plus electrical stimulation method) or Bobath. Before starting the treatment protocol, the subject's lower limbs were exposed for 10 minutes to infrared at a distance of 50 cm to warm up the limbs. This was also done to ensure the same skin temperature in all subjects, as the afferent sensory signals may affect motor neuron pool excitability in the central nervous system.[21] The combination therapy group underwent 20 daily sessions of Bobath inhibitory techniques and neuromuscular electrical stimulation. Bobath inhibitory techniques included applying for 15 minutes passive movement of ankle joint dorsi-flexion, knee joint extension, abduction and external rotation of hip joint, which is known as the reflex inhibitory pattern.[4] Neuromuscular electrical stimulation included 9 minutes of supramaximal (25% over the intensity needed to produce maximum contraction of muscle) muscle stimulation. The stimulation current included 100 Hz pulse stimulation (pulse duration = 0.1 ms, pulse interval = 0.9 ms) which was applied in surge mode (surge duration = 4 seconds and rest between surge = 6 seconds), known as Faradic stimulation,[22] on the tibialis anterior muscle via cathode (over the neuromuscular junction of the muscle) and anode (over the fibula head) electrodes.

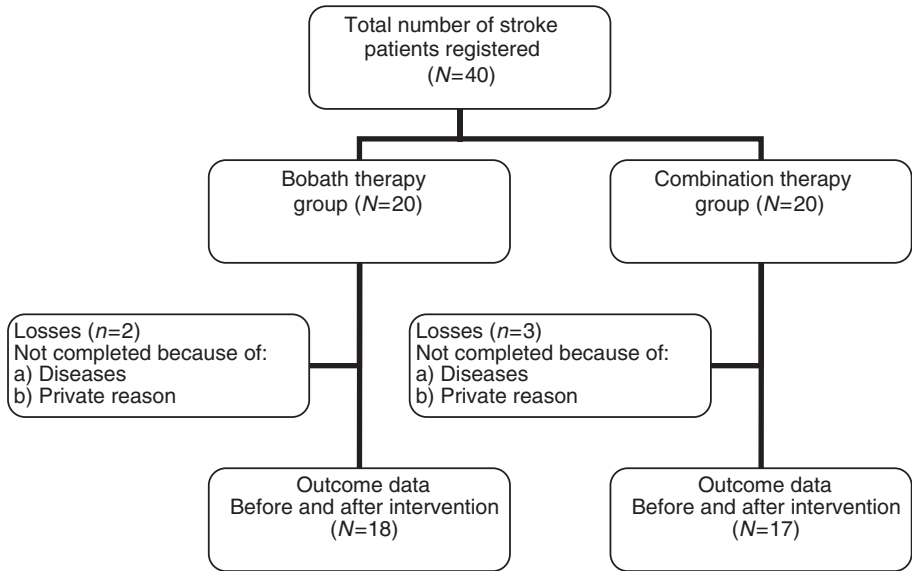

Figure 1 Flow diagram of study.

The control group received just the Bobath inhibitory technique after infrared exposure for 15 minutes as in the combination therapy group.

Measurements

The staff who assessed the outcomes measure were different from the staff administering the treatments and they were blinded from the type of treatment each patient received. All measurements were performed before and after the 20 daily therapy sessions. Outcome measures for each patient consisted of: (a) tonicity evaluation by Modified Ashworth Index,[23] (b) ankle joint dorsiflexion range of motion (ROM) by hand-held goniometer, (c) ankle dorsiflexor muscle manual strength test[24] and the evaluation of soleus muscle H-reflex amplitude.[25] A hand-hold goniometer was used to measure passive ankle joint dorsiflexion range of motion. The axis of the goniometer was placed 2 cm below the medial maleolous of the ankle joint, while its fixed arm was placed along the long axis of leg and its moving arm placed along the long axis of first metatarsal bone. The reference position was the right angle between foot and leg. The foot was then moved passively to the end of ankle joint dorsiflexion until any resistance was felt. Total free range of motion was measured by placing the moving arm to the new position of the first metatarsal. The average of three measurements was calculated and considered to be the dorsiflexion range of motion.

To record the H-reflex and M-wave, the subject lay prone in a comfortable and relaxed position while the legs were supported by pillows at the ankle joints, maintaining the knee joints in apposition of slight flexion throughout. A portable Dantec electromyography device (Keypoint Portable, Bristol, UK) was used to record the H-reflex and M-wave to calculate the H/M_{max} ratio.[26] Briefly, H-reflex and M-wave were elicited with a bipolar stimulating electrode on the tibial nerve at the popliteal fossa and recorded with a bipolar surface recording electrode placed on the midline of the soleus muscle. The active recording electrode was placed halfway between the crease of the popliteal fossa and the edge of the heel, so the recorded site was the same for all recording sessions. Low-stimulus intensity (0.5 Hz, 0.1 ms rectangular pulse) was applied to elicit H-reflex, then the stimulus intensity was increased to supramaximal stimulation until the maximal amplitude of M-wave was recorded. To ensure

recording maximal amplitude of H-reflex and M-wave, the measurements were repeated three times with a 1-minute rest between each.

Statistical analysis

To compare the possible effect of the Bobath inhibitory technique with the combination therapeutic effect of Bobath inhibitory technique plus electrical stimulation, an intention-to-treat analysis was used which involved all subjects who were randomly assigned to their groups. As all recorded measures were normal statistically, independent Student's *t*-test was used to compare the baseline values and also to compare the mean changes of the recorded values between the two experimental groups. Paired sample *t*-test was used to find any significant change in the recorded values before and after intervention within experimental groups.

Results

Table 1 shows the measured parameters before intervention in both groups; no significant difference was found in the baseline values.

Comparison between the measurement parameters showed significant changes within both combination and Bobath groups, so that significant increases in the ankle joint dorsiflexion ROM ($P=0.0001$) and dorsiflexion strength ($P=0.0001$ and $P=0.002$, respectively) and decrease in the gastrocnemius muscle tonicity ($P=0.0001$) and H-reflex amplitude ($P=0.0001$) were seen in both groups (Table 1).

Table 2 shows mean changes of measured parameters after intervention in both experimental groups. The comparison of mean changes showed significantly higher ankle joint dorsiflexion ROM ($P=0.0001$) in the combination group than in the Bobath group. Statistically, lower calf muscle

Table 1 Measured parameters before and after intervention in both experimental groups

Measured parameters	Before intervention			After intervention	
	Combination group	Bobath group	Between-groups	Combination group	Bobath group
	Mean (SD)	Mean (SD)	*P*-value	Mean (SD) Within-group *P*-value	Mean (SD) Within-group *P*-value
Ankle joint dorsiflexion ROM (degrees)	13.55 (9.52)	14.5 (9.27)	0.75	24.95 (9.57) $P=0.0001$	20.6 (9.68) $P=0.0001$
Ankle stiffness (Modified Ashworth Scale)	3.5 (0.76)	3 (1.08)	0.69	1.9 (0.72) $P=0.0001$	1.9 (0.97) $P=0.0001$
Dorsiflexor strength (graded from 0 (no contraction at all) to 5 (normal contraction))[24]	0.25 (0.55)	0.8 (1.15)	0.07	0.95 (0.83) $P=0.0001$	1.2 (1.51) $P=0.002$
H/M_{max} amplitude ratio	0.65 (0.37)	0.69 (0.37)	0.73	0.24 (0.19) $P=0.0001$	0.39 (0.16) $P=0.0001$

Table 2 Mean changes of measured parameters after intervention in both experimental groups

Measured parameters	Combination group Mean change (SD)	Bobath group Mean change (SD)	*P*-value
Ankle joint dorsiflexion ROM (degrees)	11.4 (4.79)	6.1 (3.09)	0.0001
Plantarflexor muscle tonicity (Modified Ashworth Scale)	−1.6 (0.5)	−1.1 (0.31)	0.001
Dorsiflexor strength (graded from 0 (no contraction at all) to 5 (normal contraction))[24]	0.7 (0.37)	0.4 (0.23)	0.04
H/M_{max} amplitude ratio	−0.41 (0.29)	−0.3 (0.28)	0.243

tonicity ($P = 0.0001$) and higher dorsiflexion strength ($P = 0.04$) were found in the combination therapy group by comparison of mean change recorded from both groups. However, no significant difference was found between mean changes of H/M_{max} amplitude ratio ($P = 0.23$) recorded from both experimental groups.

Discussion

This study has been designed to investigate the effectiveness of a combination of neuromuscular electrical stimulation and Bobath inhibitory techniques on spasticity in spastic patients. Our results indicated that the combination of neuromuscular electrical stimulation and Bobath techniques may be more effective in reducing spasticity, as it caused lower ankle stiffness, higher ROM in ankle joint dorsiflexion and higher ankle dorsiflexor muscles strength. Several studies have been designed to investigate the effect of electrical stimulation on spasticity either in transcutaneous or neuromuscular form.[15,27–29] By searching MEDLINE, 17 studies were found about the effects of neuromuscular electrical stimulation on spasticity. Five of these studies showed no significant change in spasticity, while the other 12 studies reported some benefits of neuromuscular electrical stimulation on spasticity reduction. Hazlewood and colleagues stated that neuromuscular electrical stimulation may increase passive range of movement among children receiving electrical stimulation by reduction of muscle tone.[17] However, in two separate studies, the authors concluded that electrical stimulation has no effect on the spasticity.[16,30] On the other hand it has been claimed that different parameters used for electrical stimulation may be the reason for the different reported results.[22] This was seen in the Hines *et al*. study that reported no decrease in spasticity in hemiplegic patients by functional electrical stimulation.[30] However, most of the studies indicate that neuromuscular electrical stimulation may be an effective method for rehabilitation of spasticity,[27,31] as was shown in our study, although the specific mechanism of this improvement remains uncertain.[32] In a more recent study, Ozer *et al*. showed that the combined use of neuromuscular electrical stimulation and bracing is more effective than either alone.[27] In 2005, Carda and Molteni showed in a case–control study that patients treated with adhesive taping and botulinum toxin achieved a greater reduction in spastic hypertonia than those treated with transcutaneous electrical stimulation therapy after botulinum toxin therapy.[16] In another study the therapeutic effect of transcutaneous electrical stimulation and oral baclofen was compared in the treatment of spasticity in patients with multiple sclerosis and authors suggested that transcutaneous electrical stimulation may be applied as a supplement to medical treatment in the management of spasticity.[28]

Functional electrical stimulation has been used for motor-complete spinal cord-injured patients and no benefit of such a therapy was found on spasticity.[29] However, it should be remembered that their subjects had no motor control from the upper motor neuron system, unlike patients in other studies who had some control from the upper motor neuron system.[32–34] These studies showed significant spasticity rehabilitation after neuromuscular electrical stimulation in hemiplegia and cerebral palsy patients.

Although our findings showed significantly improved spasticity indexes, such as joint stiffness and joint passive range of motion in the combined group, no significant changes were found in the H-reflex amplitude as reported by others.[28] H-reflex amplitude has been introduced as an index for the evaluation of spasticity,[35] although different studies present different reports about its changes. Geoulet and colleagues reported no H-reflex amplitude changes after reduction in the gastrocnemius colonus due to electrical stimulation therapy.[36] Conversely, Gaft and colleagues showed that electrical stimulation therapy may reduce spasticity and H-reflex amplitude as well.[37] Later in 2001, these contradictory reports were also reported by other authors.[38,39] It has been shown that an abnormal pattern in the H-reflex amplitude may be seen in spastic patients regarding the level of muscle tonicity.[35] Tanino and colleagues reported that after muscle electrical stimulation, there is no such pattern of H/M ratio changes in normal subjects.[40] They suggested that the main reason is the

amplitude reduction of M-wave due to muscle fatigue, which happens after electrical stimulation. This was also found in our study, as lower spasticity was found in the combination therapy group, but no further reduction was seen in the H/M_{max} ratio, which may be due to the muscle fatigue as a result of electrical stimulation therapy.[40] However, it should be remembered that the H-reflex is a variable electrophysiological value due to small changes in the level of activation of the motoneuronal pool during repeated trials.[41]

These results showed significant beneficial effects of electrical stimulation on spasticity reduction, although these effects were only assessed immediately after the intervention and no long-term effect of the therapeutic protocol was assessed by the study. However, other studies showed that the reduction of spasticity due to electrical stimulation may last for up to six months in spastic patients secondary to cerebral vascular accident and head injuries.[32,33]

As spasticity may impair functional activities in stroke patients, it is necessary to control it before applying any motor control therapeutic protocol. From our findings, it can be recommended to apply neuromuscular electrical stimulation to reduce spasticity in stroke patients so that they can receive more benefit from motor control programmes and improve their functional activity. The emphasis is on the clinical implications of electrical stimulation for spastic patients with impaired motor function.

Although our findings stated that electrical stimulation may reduce spasticity more effectively and so may help to improve motor performance,[32] no functional activity evaluation was assessed by our study to investigate any functional improvement after the therapeutic protocol. Therefore, it seems further study is needed to investigate any improvement in motor function following electrical stimulation.

Conclusion

The results of this study showed that combination therapy with neuromuscular electrical stimulation and Bobath inhibitory technique may reduce spasticity in patients with upper motor neuron lesions and may help to provide better functional performance for these patients. Therefore, it may be recommended from these findings that electrical stimulation may be used as a standard therapeutic protocol with Bobath inhibitory techniques for treatment of spasticity in the rehabilitation clinic, before starting any motor control therapeutic protocol. However, as this study showed the benefits of neuromuscular electrical stimulation on spastic muscles, further studies are needed to investigate the long-term effects of electrical stimulation on spasticity and also on the functional activity of spastic patients. It would also be recommended to design the study to compare the beneficial effects of neuromuscular electrical stimulation on agonist muscles versus antagonist muscles to find the most effective protocols for the treatment of spasticity.

Clinical message

- In stroke patients, therapy that combines Bobath inhibitory technique with electrical stimulation may help to reduce spasticity.

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