A Single Trial of Transcutaneous Electrical Nerve Stimulation (TENS) Improves Spasticity and Balance in Patients with Chronic Stroke

Hwi-young Cho,1 Tae Sung In,2 Ki Hun Cho2,3 and Chang Ho Song2

1Department of Physical Therapy, Gachon University, Incheon, Republic of Korea
2Department of Physical Therapy, Sahmyook University, Seoul, Republic of Korea
3Department of Physical Therapy, Seoul Bukbu Hospital, Seoul, Republic of Korea

Spasticity management is pivotal for achieving functional recovery of stroke patients. The purpose of this study was to investigate the effects of a single trial of transcutaneous electrical nerve stimulation (TENS) on spasticity and balance in chronic stroke patients. Forty-two chronic stroke patients were randomly allocated into the TENS (n = 22) or the placebo-TENS (n = 20) group. TENS stimulation was applied to the gastrocnemius for 60 min at 100 Hz, 200 µs with 2 to 3 times the sensory threshold (the minimal threshold in detecting electrical stimulation for subjects) after received physical therapy for 30 min. In the placebo-TENS group, electrodes were placed but no electrical stimulation was administered. For measuring spasticity, the resistance encountered during passive muscle stretching of ankle joint was assessed using the Modified Ashworth Scale, and the Hand held dynamometer was used to assess the resistive force caused by spasticity. Balance ability was measured using a force platform that measures postural sway generated by postural imbalance. The TENS group showed a significantly greater reduction in spasticity of the gastrocnemius, compared to the placebo-TENS group (p < 0.05). TENS resulted in greater balance ability improvements, especially during the eyes closed condition (p < 0.05). However, these effects returned to baseline values within one day. This study shows that TENS provides an immediately effective means of reducing spasticity and of improving balance in chronic stroke patients. The present data may be useful to establish the standard parameters for TENS application in the clinical setting of stroke.

Keywords: postural balance; rehabilitation; spasticity; stroke; transcutaneous electrical nerve stimulation

Introduction

Upper motor neuron (UMN) diseases, such as stroke, spinal cord injury (SCI), multiple sclerosis and traumatic brain injury are often accompanied by spasticity, which is defined as a velocity-dependent increased resistance to passive lengthening of muscles or tendons caused by a hyperexcitability of the stretch reflex (Mukherjee and Chakravarty 2010). Spasticity increases resistance to normal movement, interrupts motor performance, and induces gait disturbances, pain, and contracture in joints and muscles (Lundqvist et al. 1991; Sosnoff et al. 2011). Furthermore, increased muscle tone due to spasticity impedes self-care activities and results in balance disorders, thereby hindering the independence of activity of daily living (ADL) or increasing dependency when performing ADL (Doan et al. 2012). Therefore, spasticity intervention is important for improving the quality of life of people with UMN diseases and for achieving functional recovery.

Various interventions have been used to reduce spasticity, such as, surgery, anti-spastic drugs, and rehabilitation exercise-therapy. Botulinum toxin, chemical nerve-block anesthesia, analgesic-drug treatment and neurosurgery effectively decrease spasticity, but can also induce muscle weakness or paralysis (Gallichio 2004; Koussoulakos 2009). Furthermore, although anti-spastic drugs that are orally or intrathecally administered such as baclofen, tizanidine, diazepam and morphine, reduce spasticity, they are inappropriate for prolonged use due to drug tolerance and addiction (Satkunam 2003). Given these facts, there is an increasing demand for alternatives to surgical methods and medications for spasticity intervention.

Transcutaneous electrical stimulation (TENS) is the most commonly used noninvasive treatment method in physical therapy, TENS reduces pain by inducing the release of inhibitory neurotransmitters, such as, opioids and gamma amino butyric acid (GABA) agonists from the spinal cord, the rostral ventromedulla and the periaqueductal...
gray, by promoting enhancing causing inducing descending inhibition (Sluka et al. 1999; Kalra et al. 2001; Desantana et al. 2009). TENS also delays H-reflex via N2-fiber mediates presynaptic inhibition (Hiraoka 2002). Recently, it has been reported that TENS reduces spasticity and ankle clonus in UMN disease and improves joint movement and gait function (Ng and Hui-Chan 2007, 2009; Chung and Cheng 2010). However, other studies have concluded that TENS is ineffective at reducing the spasticity caused by UMN (Armütlu et al. 2003; Miller et al. 2007). Accordingly, the effect of TENS on spasticity remains unclear. On the other hand, TENS has been found to be effective at improving postural balance in healthy individuals (Dickstein et al. 2006), though no study has addressed its effects on balance ability in hemiplegic stroke patients with postural imbalance.

Thus, we investigated the effect of a single trial of TENS on spasticity and balance in patients with chronic stroke, in the hope that the results obtained would serve as basic data for future studies on the reduction of spasticity or on rehabilitation methods to improve balance ability in stroke patients.

**Subjects and Methods**

**Participants**

This study was based on a randomized, placebo-controlled trial design. Subjects were recruited according to the following criteria: those who (1) had hemiplegia from a single stroke that occurred at least six months previously, (2) had spasticity on lower limbs, (3) were able to stand over 10 minutes independently without an assistive device, (4) a score of ≥ 21 on the Mini-Mental State Examination (MMSE) (Folstein et al. 1975; Cho et al. 2012). Subjects with (1) psychiatric disorders or dementia, (2) a cardiac pacemaker, (3) neurologic or orthopedic disease that could affect balance, (4) metallic implants, (5) severe communication disorder like severe aphasia, (6) skin problems associated with electrode placement, and (7) previous experience of TENS stimulation were excluded. All experimental protocols and procedures were explained to each subject and approved by the Institutional Review Board of Sahmyook University, and all subjects signed a consent form.

**Procedures**

Fifty patients were randomly allocated into the TENS (n = 25) group or the placebo-TENS (n = 25) group using random allocation software (Saghaei 2004) to minimize the bias of experimenter. G-Power 3.13 software was used to calculate sample size. The power and the alpha level were set as 0.080 and 0.5, respectively. And, the effect size was set at 0.8. According to a priori analysis, at least 21 subjects were required in each group. Thus, we prepared 25 participants in each group for preparation for dropout. Three subjects in the TENS group and five in the placebo-TENS group dropped out due to health conditions, personal reasons, and unwillingness to continue. Thus, 22 and 20 patients with stroke in the TENS group and the placebo-TENS group, respectively, finished the study. Both groups received physical therapy based on the Bobath-concept for 30 min before TENS application (Fig. 1).

Spasticity and balance ability were measured before and after intervention and one day after intervention. Balance ability was assessed by measuring postural sway while standing (1) with eyes open condition, (2) with eyes closed condition, and (3) on an unstable surface with their eyes open condition. The Modified Ashworth Scale (MAS) and the hand held dynamometer (HHD) were used to measure spasticity following stroke. This study was designed such that the experimenters were unaware of group identities, and the different experimenters participated to measure and apply TENS stimulation. TENS stimulation (frequency 100 Hz, pulse width 200 μs, with 2 to 3 times the sensory threshold) was applied to the belly muscle of the gastrocnemius for once only for 60 min using a 2 channel TENS unit (TENS-7000, Koalaty Products Inc., USA). In order to measure the sensory threshold of each participant prior to the experiment, threshold levels were determined by administering electrical stimulation at different intensities from 0.01mA until subjects felt the stimulation. Participants were then stimulated at their individual sensory threshold intensities and at 200–300% of these sensory thresholds (Ng and Hui-Chan 2007; Hui-Chan and Ng 2009; Chung and Cheng 2010). In the placebo-TENS group, electrodes were placed but no electrical stimulation was administered.

**Outcome measurements**

Forceplate (PDM Multifunction Force Measuring Plate; Zebris, Germany) was used to measure postural sway length (PSL) for analyzing postural imbalance. A total of 1504 force sensors embedded in every 1cm² on a plate (32 × 47 cm) measure the static and dynamic pressure of feet when standing or walking (1–120 N/cm², 2–5 Hz). Participants were instructed to stand comfortably on the forceplate with their arms down to the side. When measuring with their eyes open, they stood at a point with a diameter of 15 cm and that was placed 3 m in front of them; when measuring with eyes closed, an assistant stood alongside for safety reasons. Participants positioned their feet 8 cm apart between the two medial malleoli at an angle between the feet of 10°. PSL was measured three times over 30 seconds, and mean values were calculated.

Spasticity of ankle plantarflexor was measured using the MAS and the HHD. The MAS is a validated method for assessing spasticity in stroke patients (Pizzi et al. 2005), and involves for measurement of resistance to passive dorsiflexion motion in maximal ankle plantarflexion. The MAS is composed of 6 grades (0, 1, 1+, 2, 3, and 4), where grade 0 is “normal” or “no increase in muscle tone” and grade 4 means “the affected part(s) are rigid in flexion”. This study measured spasticity three times using grades of 0, 1, 2, 3, 4 and 5, and then calculated mean values. Active contraction was eliminated by instructing patients to relax in order to minimize muscle tone in the lower limbs. The HHD (Model 01163 Lafayette, USA) is a device use to measure the muscle strength (Sisto and Dyson-Hudson 2007), and in the present study, it was used to measure the resistive force (kg) caused by spasticity. Maximal resistance force (kg) was measured with the patient supine during passive dorsiflexion to maximal range of motion by grasping the head of the HHD unit perpendicular to metacarpophalangeal joints of their feet.

**Data analysis**

The statistical analysis was performed using SPSS version 15.0 software. After confirming the normality of data using the Shapiro-Wilk test, the independent t-test, the Mann-Whitney U-test or χ²-test were used to compare the two groups. Repeated measure ANOVA and the Friedman test were conducted to determine overall changes in intergroup variances, and post-hoc test was performed using the LSD test. The significance level was set at 5% for all statistical analyses.
Results

Table 1 shows the general characteristics of the forty-two subjects with chronic stroke. No significant difference was found between the TENS group and the placebo-TENS group for any baseline value ($p > 0.05$).

Although both groups showed significantly reduced spasticity after therapeutic intervention, the TENS group appeared significant more reduction of spasticity than the placebo-TENS group. For the MAS measurement, Spasticity of the TENS group was reduced by 29% with a 13% decrease of spasticity in the placebo-TENS group. Spasticity returned to the baseline values one day after therapeutic intervention in both groups. For the HHD-based resistance measurement, the TENS group was reduced by 30% and this was similar to the result of MAS measurement, and the placebo-TENS group was also reduced by 19%. The same as the MAS measurement, spasticity measured by the HHD returned to the baseline values 24 hours after the therapeutic intervention in the HHD. Measurements using the MAS and the HHD both showed similar degrees of changes (Table 2) ($p < 0.05$).

TENS application reduced the postural imbalance in the eyes-open condition by 16% from 64.5cm to 54.3cm, and the postural imbalance of the placebo-TENS group was reduced by 9% after the intervention. Both groups returned to the baseline values after a day (Table 3) ($p < 0.05$).

With eyes closed condition, the TENS group showed a significantly decreased PSL of 23% from 89.8 cm to 69.1 cm after TENS application, while the placebo-TENS group decreased by 8% from 85.3 cm to 78.2 cm. Both groups returned to the baseline values after a day (Table 3) ($p < 0.05$).

In the TENS group, postural imbalance on an unstable surface with eyes open condition decreased by 16% from 209.4 cm to 174.6 cm, but similar to the other conditions, it returned to the pre-application values after one day. Also, placebo-TENS application decreased postural imbalance by 9% from 218.3 cm to 197.5 cm, and it returned almost to the baseline values after a day (Table 3) ($p < 0.05$).
TENS application was more effective in improving the balance function compared to that of placebo-TENS application, and its effect was most obvious in the eyes closed condition than in any other condition.

**Discussions**

This study has several important findings. First, it shows that a single trial of high-frequency TENS stimulation effectively reduced spasticity in chronic stroke patients. Second, the anti-spastic effects of a single trial of high-frequency TENS stimulation lasted for less than a day. Third, TENS stimulation effectively increased balance in stroke patients, especially in the eyes closed position. Fourth, the improved balance conferred by TENS stimulation also lasted for less than a day.

According to our results, TENS stimulation more effectively decreased spasticity than did placebo-TENS application (a reduction of 29% versus 13~19%) (Table 2). Anti-spastic effects were also observed in the placebo-TENS group, and it was probably due to the physical therapy applied during intervention. Similarly, previous studies have also shown that TENS reduces stroke-induced spasticity more effectively than exercise alone (Ng and Hui-Chan 2007; Bakhtiary and Fatemy 2008; Yan and Hui-Chan 2009).

Ng and Hui-Chan (2007) reported a 9% anti-spastic effect for TENS combined with exercise, whereas Yan and Hui-Chan (2009) reported anti-spastic effects of approximately 30%. Such discrepancies are undoubtedly due to the different methods used: Ng and Hui-Chan (2007)

### Table 1. Characteristics of study subjects.

<table>
<thead>
<tr>
<th>Variable</th>
<th>TENS (n = 22)</th>
<th>Placebo-TENS (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>55.2 ± 11.49</td>
<td>55.65 ± 8.62</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male / Female</td>
<td>14 / 8</td>
<td>13 / 7</td>
</tr>
<tr>
<td>Stroke type</td>
<td>15 / 7</td>
<td>14 / 6</td>
</tr>
<tr>
<td>Ischemic / Hemorrhage</td>
<td>15 / 7</td>
<td>14 / 6</td>
</tr>
<tr>
<td>MMSE</td>
<td>26.1 ± 1.91</td>
<td>25.8 ± 2.12</td>
</tr>
<tr>
<td>The time of stroke onset</td>
<td>15.0 ± 4.91</td>
<td>13.9 ± 5.08</td>
</tr>
<tr>
<td>MAS score</td>
<td>3.5 ± 0.91</td>
<td>3.4 ± 0.68</td>
</tr>
<tr>
<td>HHD of plantarflexors (kg)</td>
<td>12.07 ± 3.34</td>
<td>12.32 ± 5.49</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± standard deviation (s.d.). MMSE, Mini Mental State Examination; MAS, Modified Ashworth Scale; HHD, Hand Held Dynamometer.

Table 1 showed the general characteristics of forty-two subjects with chronic stroke and who fulfilled the inclusion criteria for the study. There were no significant differences between TENS and placebo-TENS groups for all the baseline values.

### Table 2. The Effects of TENS on spasticity (MAS and HHD).

<table>
<thead>
<tr>
<th>Variable</th>
<th>TENS (n = 22)</th>
<th>Placebo-TENS (n = 20)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAS (scores)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>3.50 ± 0.91*</td>
<td>3.40 ± 0.68*</td>
<td>0.516</td>
</tr>
<tr>
<td>Post</td>
<td>2.50 ± 0.60**</td>
<td>2.95 ± 0.69*</td>
<td>0.029</td>
</tr>
<tr>
<td>Follow-up</td>
<td>3.32 ± 0.89*</td>
<td>3.30 ± 0.57*</td>
<td>0.796</td>
</tr>
<tr>
<td>P-value</td>
<td>0.000</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean ± s.d. MAS, Modified Ashworth Scale; HHD, Hand Held Dynamometer. * means a significant difference from the baseline value and ** indicates a significant difference compared with post value (p < 0.05). ** represents a statistically significant difference between TENS and Placebo-TENS groups (p < 0.05).

Ankle plantarflexor spasticity was measured using the Modified Ashworth Scale (MAS) and the Hand Held Dynamometer (HHD). Although both groups showed significantly reduced spasticity after therapeutic intervention, TENS group appeared significant more reduction of spasticity than placebo-TENS group. However, spasticity returned to the baseline values one day after therapeutic intervention in both groups.

According to our results, TENS stimulation more effectively decreased spasticity than did placebo-TENS application (a reduction of 29% versus 13~19%) (Table 2). Anti-spastic effects were also observed in the placebo-TENS group, and it was probably due to the physical therapy applied during intervention. Similarly, previous studies have also shown that TENS reduces stroke-induced spasticity more effectively than exercise alone (Ng and Hui-Chan 2007; Bakhtiary and Fatemy 2008; Yan and Hui-Chan 2009).

Ng and Hui-Chan (2007) reported a 9% anti-spastic effect for TENS combined with exercise, whereas Yan and Hui-Chan (2009) reported anti-spastic effects of approximately 30%. Such discrepancies are undoubtedly due to the different methods used: Ng and Hui-Chan (2007)
used a home-based program, whereas Yan and Hui-Chan (2009) and we used 30-min physical therapy. In placebo group, Ng and Hui-Chan (2007) observed a 6% improvement, whereas Yan and Hui-Chan (2009) and we observed an anti-spasticity improvement of > 9%. These results show physical therapy is effective at reducing spasticity, and that TENS can be used as an additive anti-spastic intervention.

In the present study, electrodes were attached to muscle bellies in calves, which are innervated by the sural nerve communicating branch of the common peroneal nerve. In other studies on the anti-spastic effects of TENS, electrodes were applied to acupuncture points (Ng and Hui-Chan 2007; Yan and Hui-Chan 2009) or posterior to the fibular head (Chung and Cheng 2010), and these sites are innervated by the common peroneal and sural nerves. Therefore, although electrodes were placed on different sites, TENS stimulation reduced spasticity by enhancing presynaptic inhibition on the sural or peroneal nerves.

Baclofen (GABA receptor agonist) and morphine (opioid receptor agonist) are used in clinics to reduce spasticity caused by UMN diseases (Penn and Kroin 1987; Lewis and Mueller 1993). These agents hyperpolarize presynaptic terminals at the spinal cord, decrease excitatory neurotransmitter secretions, suppress $\alpha$-motor neuron activation (Milanov 1992), and inhibit mono- or multi-synaptic reflexes on the dorsal horn of the spinal cord (Erickson et al. 1985; Kita and Goodkin 2000). In animal studies, the application of high-frequency TENS to rats increased GABA (an inhibitory neurotransmitter) release from the dorsal horn of the spinal cord (Sluka et al. 2007). TENS applied on rats with reduced expression of dynorphin following SCI, which is both an opioid precursor and an endogenous opiate peptide, also resulted in reduced spasticity (Dong et al. 2005). Thus, it is assumed that the anti-spastic effects of TENS increased the expressions or release of endogenous GABA and opiates, with both being inhibitory neurotransmitters on the dorsal horn of the spinal cord, and this achieved similar anti-spastic effects as those of baclofen and morphine as a result.

TENS stimulation at two to three times the sensory threshold produces vibrations in stimulated muscles and surrounding regions. Moreover, the rapid stimulation of vibration triggers primary afferent neurons and increases the release of acetylcholine, a major neurotransmitter in the context of muscle contraction. However, prolonged stimulation may reduce muscle contraction by lowering the excitability of homonymous motor neurons by depleting acetylcholine, as occurs during muscle fatigue (Desmedt 1983). Although not suggested by our results, spasticity tends to increase temporarily at the beginning of TENS stimulation, but then progressively diminishes. However, the anti-spastic effects of TENS last for less than a day. The short-term nature of these effects is thought to be due to acetylcholine depletion, which is a transient phenomenon in peripheral neuromuscular junctions, rather than being due to any spinal cord change.

To measure spasticity in this study, we used MAS, which are commonly used for this purpose, and the HHD, which has been previously used to measure the muscle strength (Sisto and Dyson-Hudson 2007), and to measure hypertonia in SCI (Lamontagne et al. 1998) and cerebral

<table>
<thead>
<tr>
<th>Variable</th>
<th>TENS ($n = 22$)</th>
<th>Placebo-TENS ($n = 20$)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EO (cm)</td>
<td>Baseline 64.45 ± 12.53°</td>
<td>Placebo-TENS 66.07 ± 12.35</td>
<td>0.676</td>
</tr>
<tr>
<td></td>
<td>Post 54.31 ± 10.24*</td>
<td>Placebo-TENS 59.82 ± 10.16</td>
<td>0.088</td>
</tr>
<tr>
<td></td>
<td>Follow-up 63.30 ± 12.08°</td>
<td>Placebo-TENS 66.63 ± 10.17</td>
<td>0.342</td>
</tr>
<tr>
<td></td>
<td>$P$-value 0.010</td>
<td>Placebo-TENS 0.101</td>
<td></td>
</tr>
<tr>
<td>EC (cm)</td>
<td>Baseline 89.79 ± 21.78°</td>
<td>Placebo-TENS 85.31 ± 16.30</td>
<td>0.458</td>
</tr>
<tr>
<td></td>
<td>Post 69.05 ± 71.11**</td>
<td>Placebo-TENS 78.20 ± 15.17</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Follow-up 85.28 ± 16.64°</td>
<td>Placebo-TENS 83.58 ± 15.79</td>
<td>0.737</td>
</tr>
<tr>
<td></td>
<td>$P$-value 0.000</td>
<td>Placebo-TENS 0.338</td>
<td></td>
</tr>
<tr>
<td>USEO (cm)</td>
<td>Baseline 209.37 ± 55.12°</td>
<td>Placebo-TENS 218.26 ± 35.84°</td>
<td>0.543</td>
</tr>
<tr>
<td></td>
<td>Post 174.61 ± 52.29°</td>
<td>Placebo-TENS 197.51 ± 25.04°*</td>
<td>0.083</td>
</tr>
<tr>
<td></td>
<td>Follow-up 199.76 ± 47.75</td>
<td>Placebo-TENS 215.59 ± 31.35</td>
<td>0.216</td>
</tr>
<tr>
<td></td>
<td>$P$-value 0.079</td>
<td>Placebo-TENS 0.080</td>
<td></td>
</tr>
</tbody>
</table>

Values are expressed as mean ± s.d.
EO, eyes open; EC, eyes closed; USEO, unstable surface with eyes open.
*means a significant difference from the baseline value and °indicates a significant difference compared with post value. **represents a statistically significant difference between TENS and Placebo-TENS groups ($p < 0.05$).
palsy (Boiteau et al. 1995). In the present study, the anti-spastic effect of TENS stimulation was 29% according to MAS and 30% by the HHD, and similar effects were observed in the placebo-TENS group (13% by MAS and 19% by HHD). Although these two measurement methods showed a positive correlation ($r = 0.502, p < 0.05$), the HHD is believed to be more appropriate for measuring spasticity, and in fact, provides a useful clinical method for measuring spasticity caused by stroke.

Our results also demonstrate that a single trial of high-frequency TENS stimulation reduced postural imbalance following stroke. The placebo-TENS group, which only received physical therapy, showed an 8–10% improvement, whereas TENS group showed an effective boost in these tendencies to 16–23%. Similarly, TENS stimulation in healthy subjects has been reported to significantly reduce postural sway in the standing position (Dickstein et al. 2006), and to improve postural instability in the sitting position when applied to the neck muscles of stroke patients (Perennou et al. 2001). Additionally, TENS stimulation improved gait function, which is a balance-related movement in chronic stroke patients (Ng and Hui-Chan 2009).

Balance improvements elicited by TENS are believed to be the result of increased proprioception input out of the somatic sense in lower limbs. TENS stimulation of calves, which plays a pivotal role in the control and maintenance of the standing posture, probably produces higher somatosensory inputs than the standard rehabilitation (Lord et al. 1991). Somatosensory inputs can affect motor function because they are related to balance (Hatzitaki et al. 2004), and the somatosensory area in the cerebral cortex is connected to the primary motor cortex (Farkas et al. 1991). Therefore, TENS stimulation on both calves may increase somatosensory inputs from the lower limbs and cause changes in or a reorganization of the sensory and/or motor cortex, and thus, increase balance.

Our results support this hypothesis. Postural control is predominantly based on three sensations (visual, vestibular, and somatic sensations). According to our results, the placebo-TENS group showed a 10% decrease in postural sway with eyes open condition, but an 8% improvement with eyes closed condition. On the other hand, the TENS group showed a 16% improvement with eyes open condition and a 23% improvement with eyes closed condition. The maintenance of balance with eyes closed condition is more dependent on proprioception and vestibular sensation (de Oliveira et al. 2008), and the vestibular system was not limited in our subjects. Thus, the improvements in postural function observed after TENS stimulation are believed to have been caused by increased proprioception input.

Impaired balance in stroke patients is related to diminished ankle proprioception (Tyson et al. 2006). In the present study, the TENS group (16%) and the placebo-TENS group (9.5%) showed similar percentage improvements in postural sway with eyes open condition on stable and unstable surfaces, the latter of which hinders proprioception for postural orientation. This finding suggested that postural control in hemiparetic stroke patients is more dependent on visual function due to restricted proprioception.

Spasticity not only deteriorates balance function by causing asymmetrical postural alignment, it also hinders motor skills by limiting movement and increasing energy consumption due to co-contraction of agonists and antagonists (Knikou et al. 2007; Miller et al. 2007; Ng and Hui-Chan 2009). This study demonstrates that TENS stimulation to calves following chronic stroke reduces spasticity. Furthermore, a positive correlation was found between decreased spasticity and improvements in the eyes-open condition ($r = 0.210, p < 0.05$). Therefore, spasticity reductions by TENS effectively improve balance by improving asymmetrical postural alignment, reducing limb function limitations, and by facilitating more efficient energy usage.

According to a previous study on the effect of TENS on motor cortex excitability (Tinazzi et al. 2005), excitability was attenuated in cortical areas corresponding to TENS-stimulated muscles, but elevated in antagonist brain areas. Stroke patients are commonly accompanied by ankle plantarflexion contracture due to spasticity of the calf muscle and tibialis anterior weakness (Mulroy et al. 2010). As was observed in a previous study (Tinazzi et al. 2005), TENS stimulation of calf muscles in this study changed the excitability of brain cortical areas, or rather reciprocal inhibition and facilitation that enabled efficient movement and increased balance function was observed.

However, although a single trial of TENS was found to be effective at reducing spasticity and improving balance, the effects of long-term TENS application have not been determined. Therefore, more research is required on the effects of long-term TENS treatment on spasticity and balance in stroke patients.

This study demonstrates that TENS application to spastic muscle is immediately effective at reducing spasticity and improving balance in stroke patients. We hope that our data are useful to establish standard parameters for TENS in the setting of stroke.

Acknowledgments

This study was supported by Sahmyook University.

Conflict of Interest

The authors declare that there are no conflicts of interest.

References


Transcutaneous Electrical Nerve Stimulation in Stroke


