The Effect of Transcutaneous Electrical Stimulation on Spinal Motor Neuron Excitability in People Without Known Neuromuscular Diseases: The Roles of Stimulus Intensity and Location

Background and Purpose. The Hoffmann reflex (H-reflex) is widely acknowledged as an indirect indicator of spinal motor neuron excitability. The purpose of this study was to determine whether transcutaneous electrical stimulation (TES), applied over the dorsiflexors or plantar flexors of the ankle, would alter the soleus muscle’s H-reflex. Attention was focused on the roles of stimulus intensity and location.

Subjects. Thirty-two volunteers without known neuromuscular diseases (17 women [53%]; mean years of age = 27, SD = 7.3, range = 21–48) were studied.

Methods. Subjects were randomly assigned to 1 of 4 groups, and TES was administered for 15 minutes. Stimulation site and intensity varied according to group assignment. H-reflexes were recorded before and for 10 minutes after TES.

Results. H-reflex amplitudes increased following TES at sensory threshold, whereas H-reflex amplitudes did not change following TES at 1.5 times motor threshold. The site of stimulation did not influence the resulting H-reflexes.


Key Words: H-reflex, Motor neuron excitability, Transcutaneous electrical stimulation.

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Transcutaneous electrical stimulation (TES) has been used to diminish the characteristics of upper motor neuron (UMN) syndrome (eg, a velocity-dependent increase in muscle tone [spasticity], hyperreflexia, clonus). The reduction of these characteristics was usually temporary, lasting from 30 minutes to 24 hours. In some cases, however, the reduction of characteristics was viewed as permanent. In these and other studies, it is interesting that the methods used to administer the TES often differed in regard to the stimulation site and stimulation intensity. Giebler reported that some authors stimulated over the antagonists of the involved muscles, whereas other authors chose to stimulate over the involved muscles. Stimulus intensity also has varied, with some authors reporting a reduction hypertonicity and tonus when TES was below sensory threshold (ST). Other authors, however, have suggested that the stimulation should be above ST and just below motor threshold (MT). Furthermore, although some authors have suggested that stimulation in excess of the MT exacerbates hypertonicity, other authors have observed that stimulation intensities above MT result in a prolonged reduction of hypertonia and hyperreflexia.

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All authors provided concept/research design and consultation (including review of manuscript before submission). Dr Hardy and Dr Stokic provided writing. Mr Spalding, Dr Liu, and Mr Hayes provided data collection. Dr Nick provided data analysis. Dr Hardy provided subjects, institutional liaisons, and project management. Dr Stokic provided fund procurement and facilities/equipment.

This study was approved by the Institutional Review Boards at the University of Mississippi Medical Center and Methodist Rehabilitation Center for research involving human subjects.

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Because of the previously cited claims that TES can be effective in reducing spasticity, hyperreflexia, and clonus, we concluded that TES may also be effective in reducing the activity of hypersensitive spinal motor neurons that are typically present in people having UMN syndrome. Furthermore, we conjectured that TES may also be effective in reducing the sensitivity of spinal motor neurons in people with no known neuromuscular diseases. Evidence supporting this hypothesis has been gathered in a variety of studies of subjects with no known neuromuscular diseases and will be discussed below.

In these studies, the Hoffmann reflex (H-reflex) as a monitor of spinal motor neuron excitability. The H-reflex used in these studies is similar to, and involves the same neural circuits as, the stretch reflex. Instead of using a reflex hammer, however, an electrical stimulus is administered directly to the nerve innervating the muscle to be tested (usually the soleus muscle). The stimulus activates sensory (Ia afferent) fibers within the nerve that, in turn, synapse upon spinal motor neurons, presumably via a monosynaptic pathway. The output of these spinal motor neurons then travels down the motor fibers of the previously stimulated nerve and causes a muscle membrane depolarization that can be recorded using surface electromyography (EMG) electrodes. The amplitude of the evoked EMG response indirectly reflects the excitability of the spinal motor neurons. The H-reflex amplitude has been found to have a high intrasubject reliability and can be obtained in people with and without known neuromuscular diseases. The validity of measurements obtained for the H-reflex, as an indicator of increased reflex activity, has been demonstrated in pharmacological studies involving the drug baclofen. In these studies, it was found that baclofen caused a reduction in clinical measures of reflex activity that was paralleled by a decrease in the H-reflex.

Delwaide and associates observed, in people with no known neuromuscular diseases, that a strong (painful) stimulation of the sural nerve caused a brief decrease in the amplitude of the H-reflex of the soleus muscle, whereas a mild stimulus (2–3 times the ST) caused an increase in the H-reflex. In similar experiments, Goulet and associates made observations that conflict with Delwaide and associates’ finding, regarding the effects of a mild stimulus. Goulet and associates found that a mild stimulus applied to either the sural or common peroneal nerves caused a decrease in the soleus muscle’s H-reflex. In both studies, the electrical stimulus was administered to nerve trunks rather than over the muscle bellies. Consequently, for TES administered over muscle groups, the effects of site and intensity on spinal motor neuron excitability are still not clear.

Our study was designed to determine what effect TES would have on spinal motor neuron excitability, as reflected by the H-reflex of the soleus muscle, in volunteers having no known neuromuscular diseases. We focused on the potentially differential effects that stimulus intensity and location might have on this reflex. Our hypotheses were that TES would influence the H-reflex and that this influence would vary in magnitude and perhaps direction, depending on the stimulus intensity and location. We used subjects with no known neuromuscular diseases in this study so that we could better understand the basic mechanisms underlying TES influences on spinal motor excitability. The results of this study can be used as points of comparison in future studies of people with central nervous system pathology.

**Method**

**Subjects**

Thirty-two volunteers (17 [53%] women) with a mean age of 27 years (SD = 7.3, range = 21–48) were recruited for this study. As determined by questionnaire, the subjects in this study had no history of neurological disease. The experimental procedures were explained to each subject, and each subject gave informed consent.

**Procedure**

**Electrode placement.** As Figures 1 and 2 illustrate, stimulating and recording electrodes were placed on the right lower extremity of each subject to stimulate the tibial nerve and thereby elicit the H-reflex. A pair of
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available electrodiagnostic apparatus (Viking IV-D
an experienced EMG technologist, using a commercially
H-reflex recording was performed by
H-reflex protocol.

| Subject positioning. Each subject rested supine on a
bed. Towel rolls were placed under the right knee and
ankle, so that the heel was off the surface of the bed and
so that reflex movements of the ankle were unrestricted.

H-reflex protocol. H-reflex recording was performed by
an experienced EMG technologist, using a commercially
available electrodiagnostic apparatus (Viking IV-D). First, an H-reflex recruitment curve was recorded to determine the current necessary to produce a maximal H-reflex and a maximal M-wave (short-latency orthodromic motor response). The current was subsequently adjusted to elicit reproducible H-reflexes that measured between 20% and 40% of the maximum M-wave, a size known to be sensitive to both excitatory and inhibitory influences. Following this procedure, the current was not changed, and 10 H-reflexes were recorded in complete relaxation before and 3 times after TES, with an H-reflex being evoked every 10 seconds using a 1-millisecond stimulus duration.

TES stimulation apparatus. Transcutaneous electrical stimulation was administered with a Grass S-88 stimulator through 2 carbon-rubber surface electrodes (4.3 cm long × 3.7 cm wide) that were separated from the skin by 2 saline-impregnated sponges. These electrodes were centered over the muscle group of interest (either the right-sided dorsiflexors or the right-sided plantar flexors). The stimulus was delivered as 3-second trains, with a 2-second intertrain (off) period. Each train consisted of 100-microsecond square waves occurring at 20 Hz (total of 60 pulses). These stimulus parameters are similar to those used to reduce spasticity, hyperreflexia, and clonus. Using these stimulus parameters, each subject was tested to determine the sensory or motor threshold over the muscle group of interest. The TES was administered either at ST or at 1.5 times MT (1.5MT), depending on group assignment.

Experimental protocol. Subjects were randomly assigned to 1 of 4 groups in which electrical stimulation was administered for 15 minutes. Each group was composed of 8 subjects. Three of the 4 groups contained 4 (50%) women, and only group 2 contained 5 (63%) women. The mean ages for each of the 4 groups were: 26 years (SD = 5.7, range = 22–40) for group 1, 26 years (SD = 8.7, range = 23–48) for group 2, 28 years (SD = 7.8, range = 23–43) for group 3, and 25 years (SD = 7.7, range = 21–44) for group 4. No age differences were found in the groups’ means (P = .86, one-way analysis of variance [ANOVA] test) and variances (P = .70, Levene test).

The stimulation intensity (intensity factor) and site (location factor) were assigned as follows: group 1 received ST intensity to the plantar flexors, group 2 received 1.5MT intensity to the plantar flexors, group 3 received ST intensity to the dorsiflexors, and group 4 received 1.5MT intensity to the dorsiflexors. Thus, the intensity had 2 levels—ST and 1.5MT—and there were 2 levels for location—over the plantar flexors and over the dorsiflexors. Immediately before the period of electrical stimulation, 10 consecutive H-reflexes were elicited, and their amplitudes were averaged to yield the baseline value. Following the period of stimulation, 3 sets of 10 H-reflexes each were obtained starting (1) at the end of stimulation, (2) 5 minutes following the end of stimulation, and (3) 10 minutes following the end of stimulation.

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‡ Nicolet Biomedical, 5225 Verona Rd, Madison, WI 53711.
§ Grass Instruments, Div of Astro-Med Inc, 600 E Greenwich Ave, West Warwick, RI 02893.
¶ Promatek Industries Ltd, 8390 Mayrand, Montreal, Quebec, Canada H4P 2C9.
Table 1. Means, Standard Deviations, and Ranges (Low, High) for Percentage of Change in the H-reflexes During the Three Post-Transcutaneous Electrical Stimulation Trials *

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intensity</td>
<td>1</td>
<td>35200</td>
<td>35200</td>
<td>10.29</td>
<td>.003</td>
</tr>
<tr>
<td>Location</td>
<td>1</td>
<td>22</td>
<td>22</td>
<td>0.01</td>
<td>.94</td>
</tr>
<tr>
<td>Trials</td>
<td>2</td>
<td>3878</td>
<td>3878</td>
<td>11.41</td>
<td>.001</td>
</tr>
<tr>
<td>Intensity × trials</td>
<td>2</td>
<td>1300</td>
<td>1300</td>
<td>3.82</td>
<td>.03</td>
</tr>
<tr>
<td>Location × trials</td>
<td>2</td>
<td>39</td>
<td>39</td>
<td>0.12</td>
<td>.89</td>
</tr>
</tbody>
</table>

*Statistical significance at P ≤ .05.

Table 2. Three-Way, Repeated-Measures Analysis of Variance Comparing the Percentage of Change in the Amplitude of the H-Reflex From Baseline Among Intensity, Location, and Trials (Repeated Factor)

<table>
<thead>
<tr>
<th>X</th>
<th>SD</th>
<th>Low</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td>86</td>
<td>61</td>
<td>18</td>
<td>197</td>
</tr>
<tr>
<td>93</td>
<td>50</td>
<td>35</td>
<td>165</td>
</tr>
<tr>
<td>93</td>
<td>50</td>
<td>33</td>
<td>168</td>
</tr>
</tbody>
</table>

*Group 1 received stimulation to the plantar flexors at sensory threshold, group 2 received stimulation to the plantar flexors at 1.5 times motor threshold, group 3 received stimulation to the dorsiflexors at sensory threshold, and group 4 received stimulation to the dorsiflexors at 1.5 times motor threshold.

Data Analysis
For each subject, the average amplitude of 10 consecutive H-reflexes was calculated for each trial. The percentage change of the average H-reflex amplitude for each of the 3 post-TES trials was calculated for each subject as [(trial − baseline)/(baseline)] × 100. For example, a subject with a baseline H-reflex of 2 mV and a post-TES trial H-reflex of 4 mV would have a 100% change from baseline. We compared changes in H-reflexes for the trial factor, intensity factor, and location factor using a 3-way, repeated-measures ANOVA (the trial factor was the repeated measure). All statistical tests were 2-tailed, and the type I error rates for the collection of tests for each factor were set at .05 (familywise error rate). The mean percentage of change of the 2 levels in the factors of location (ie, plantar flexors and dorsiflexors) and intensity (ie, ST and 1.5MT) were not assumed to be parallel over the 3 post-TES trials. That is, trial by location and trial by intensity interaction effects were included in the ANOVA model.30,31

The interaction effects were tested first, and if the interaction was not rejected, we then proceeded to test the main effect. If the interaction was rejected, we then tested the hypothesis of equal factor means separately for each trial using the usual unadjusted ANOVA F test. To adequately control the familywise error rate at .05, these separate F tests used a significance level of .05/3 = .017, if needed. If differences were found between the 2 levels within each of the factors, then significant increases or decreases in adjusted mean percentage of change were assessed using 95% confidence intervals (95% CIs). Significant changes from the control were determined if the 95% CI did not bracket 0. As a control measure, a statistical strategy identical to the one stated above was performed on the M-waves.

Results
Table 1 presents the means, standard deviations, and ranges of the percentage of change in the H-reflex for each post-TES trial in the 4 experimental groups. Table 2 presents the ANOVA summary.

The effect of the intensity factor (ST and 1.5MT) on H-reflex amplitudes (Fig. 3) was related to the post-TES trials (the interaction of intensity and trials was significant F = 3.82, P = .03). Because this interaction was significant, the main effects of intensity and trials could not be used as hypothesis tests; therefore, we tested equal intensity means separately for each trial with the usual ANOVA F test. Using a significance level of .017 for the tests in each trial,31 we concluded that there were differences between the intensity levels in all 3 trials. The difference in the mean percentage of change in H-reflexes (ST − 1.5MT) resulting from the 2 stimulus intensities was greater in trial 1 than in the other trials (Fig. 3). The change resulting from ST stimulation in trial 1 was 81% greater than change resulting from 1.5MT stimulation (P = .001, 95% CI = 37%-125%). The change brought about by ST stimulation was 59% more than the change resulting from stimulation at 1.5MT in both trial 2 (P = .01, 95% CI = 16%-102%) and trial 3 (P = .01, 95% CI = 15%-103%). In all 3 trials, the mean percentage of change resulting from the 1.5MT stimulation was not different from 0 (Figs. 4A and 3B). Conversely, in all 3 trials, the mean percentage of change for the ST stimulation was different from 0 (Figs. 4C and 3D), which was the baseline comparison. Table 3
presents the means and 95% CIs for all 6 baseline comparisons. The effect of the location factor (plantar flexors and dorsiflexors) on H-reflex amplitudes (Fig. 5) was not related to the trials. The interaction of location and trial was not significant (F\textsubscript{138}/H1\textsubscript{1105}.12, P\textsubscript{170}/H1\textsubscript{1105}.89). Because the interaction was not significant, the main effect of stimulus location was tested and was found to be insignificant (F\textsubscript{54}/H1\textsubscript{1105}.01, P\textsubscript{86}/H1\textsubscript{1105}.97). The pooled mean percentage of change for the plantar flexors was 45% (95% CI\textsubscript{16%-75%}), and the mean percentage of change for the dorsiflexors was 47% (95% CI\textsubscript{17%-77%}).

For the M-wave amplitudes, the effects of stimulus location and intensity were not related to the trials (F\textsubscript{45}/H1\textsubscript{1105}.60, P\textsubscript{50}/H1\textsubscript{1105}.55 for both interaction tests). In addition, there were no differences between the plantar flexors and dorsiflexors (F\textsubscript{70}/H1\textsubscript{1105}1.14, P\textsubscript{107}/H1\textsubscript{1105}.29) or between the ST and 1.5MT levels (F\textsubscript{54}/H1\textsubscript{1105}1.34, P\textsubscript{96}/H1\textsubscript{1105}.26). Overall, there was no difference between the M-wave amplitudes at baseline and after TES (F\textsubscript{76}/H1\textsubscript{1105}3.02, P\textsubscript{114}/H1\textsubscript{1105}.09).

In summary, H-reflex amplitudes increased following ST stimulation (P\textsubscript{102}/H1\textsubscript{1102}.001) (Figs. 3, 4C, and 4D). This effect was prolonged (>10 minutes), typically persisting through the third post-TES trial (Fig. 3). Following 1.5MT stimulation, changes in H-reflexes were not significant (Figs. 3, 4A, and 4B). Conversely, in the first post-TES trial of the group receiving 1.5MT stimulation of the plantar flexors, a transient 21% decrease in H-reflex amplitude did occur (Tab. 1) that fell just short of being significant (P=.06). The site of stimulation (plantar flexors versus dorsiflexors) was a variable that did not influence the resulting H-reflexes (Fig. 5).

**Discussion**

In our study, the results supported the hypothesis that stimulus intensity would play an important role in determining the extent to which TES influenced the H-reflexes. In subjects with no known neuromuscular diseases, we found that low-intensity TES increased H-reflex amplitudes; high-intensity stimulation, however, did not alter the H-reflexes and, in some cases, may actually have tended to cause a transient decrease in H-reflexes. The results did not support the hypothesis that stimulus location would play an important role in determining the extent to which TES influenced the H-reflexes. Stimulation over the dorsiflexors exerted no more influence on H-reflexes than did stimulation over the plantar flexors.

These results tend to be in agreement with the findings of some researchers, but not other researchers. Delwaide and associates found that mild stimulation (2–3 times ST) of the sural nerve caused a brief increase of the soleus muscle’s H-reflex, whereas a strong (painful) stimulus caused a brief decrease of the soleus H-reflex. The observations of Delwaide and associates correlate well with our findings that TES delivered at ST resulted in an increase in the soleus muscle’s H-reflex, whereas TES delivered at above MT did not alter the H-reflex.

The fact that our results only partly support the findings of Delwaide and associates and did not support the findings of Goulet et al may be attributed, in part, to the fact that we used different methods than those used by the other research groups. The primary difference was that the other researchers focused on changes in individual H-reflexes that occurred within a 1-second period, whereas we based our study on an average of 10 H-reflexes, which took a longer time to elicit (ie, approximately 100 seconds). Nevertheless, our data seem to indicate that TES utilizing a strong stimulus may have transiently depressed the H-reflexes occurring during the first post-TES trial (Tab. 1, Fig. 3), thus supporting the findings of Delwaide and associates. In other studies involving volunteers with no known neuromuscular...
cular diseases, transient depressions of the H-reflex (lasting a few seconds or less) have been elicited during the recovery from a 2- or 3-second isometric muscle contraction.\textsuperscript{32,33} Similar depressions of the stretch reflex, involving subjects with no known neuromuscular diseases, have been elicited by active joint movement.\textsuperscript{34} In our study, a tendency toward a transient depression of the H-reflex was observed in group 2, a group in which TES elicited both muscle contraction and joint movement.

In view of the reports\textsuperscript{1–13} indicating the efficacy of TES in decreasing the characteristics of UMN syndrome

![Figure 4](image)

**Figure 4.**
A comparison of H-reflexes (arrows) in 2 subjects before (A and C) and after (B and D) the administration of transcutaneous electrical stimulation (TES) to their plantar flexors. High-intensity TES (1.5 times motor threshold) was administered to one subject (A and B), and low-intensity TES (sensory threshold) was administered to the other subject (C and D). Note: low-intensity TES in the latter subject caused the H-reflex amplitudes to be increased, with no change in the M-wave (not visible at this display sensitivity).

<table>
<thead>
<tr>
<th>Trial</th>
<th>Sensory Threshold X</th>
<th>95% CI</th>
<th>1.5 Times Motor Threshold X</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>74</td>
<td>43, 105</td>
<td>-7</td>
<td>-38, 24</td>
</tr>
<tr>
<td>2</td>
<td>80</td>
<td>49, 110</td>
<td>21</td>
<td>-9, 51</td>
</tr>
<tr>
<td>3</td>
<td>84</td>
<td>53, 115</td>
<td>25</td>
<td>-7, 56</td>
</tr>
</tbody>
</table>

*An interval that does not include 0 indicates significant differences from baseline measurements.
(eg, spasticity, hyperreflexia, clonus), we found it surprising that TES, as administered in our study, did not attenuate H-reflexes. One possible explanation for this is the fact that none of the subjects in our study had any known neuromuscular diseases, whereas the subjects of the clinical reports had neurological deficits. It is conceivable that people with known neuromuscular diseases may respond to TES differently than people without known neurological diseases. In support of this hypothesis, it has been noted in people having hyperreflexia and hypertonia that those having the most pronounced symptoms obtained the greatest decrease in hyperreflexia and hypertonia following TES. Furthermore, TES-elicited decreases in the H-reflex are greater in people with lesions in the central nervous system than in people without lesions in the central nervous system. Consequently, we suggest that the state of excitability of spinal motor neurons or associated interneurons in people with no known neuromuscular diseases versus people with lesions in the central nervous system may be fundamentally different in regard to how they are influenced by TES.

What are the possible explanations for our findings? Perhaps an alteration in Ia afferent input (possibly due to a change in position of the stimulating electrodes) could result in findings similar to those we found. However, this is unlikely to have occurred in this study because both recording and stimulating electrodes were securely kept in place throughout the experiment. Furthermore, there were no alterations in M-wave amplitudes to accompany the alterations in H-reflexes. Consequently, we believe that it is unlikely that the results of this study were due to technical artifacts. Conversely, it is conceivable that electrical stimulation at ST resulted primarily in the depolarization of low-threshold cutaneous afferents. However, with 1.5MT stimulation, high-threshold deep afferents (cutaneous and muscle) also were likely recruited. The results of our study suggest that afferents that have different thresholds for electrical stimulation exert differential effects on spinal motor neuron excitability, with low-threshold cutaneous afferents being primarily excitatory and high-threshold afferents possibly serving in a more inhibitory capacity.

The excitatory effects of cutaneous stimulation on H-reflex amplitude have been previously reported. For example, H-reflex was found to be augmented after spraying the skin of the posterior aspect of the calf with either lidocaine or a placebo solution. However, stronger input such as massage had the opposite effect. The increase in H-reflex amplitude after low-intensity ST stimulation, as observed in our study, is also supported by the finding that iontophoresis of either lidocaine or a placebo facilitated the H-reflex for 30 minutes. The authors of this study concluded that the low-voltage galvanic electrical stimulation administered above ST was primarily responsible for the increase in spinal motor neuron excitability. Indeed, low-intensity sural nerve stimulation is capable of facilitating the soleus muscle's motor neuron pool, as demonstrated by the H-reflex and by the increased probability of single motor unit firing.

The amplitude of the H-reflex can be modulated by modification of Ia afferent input or by alterations in the excitability of spinal motor neurons. In our study, the magnitude of facilitation was independent of the sites of stimulation and their different segmental innervations. This tends to indicate that the effects of stimulation were mediated through a convergence of different spatial inputs upon a common interneuronal system. Furthermore, this suggests the involvement of a presynaptic mechanism. In our opinion, low-threshold cutaneous afferents probably share common interneurons with low-threshold muscle afferents that mediate the H-reflexes. In our study, it is therefore plausible that low-threshold cutaneous afferents diminished the presynaptic inhibition of Ia afferent fibers terminating on...
the soleus muscle’s motor neuron pool, thus increasing the soleus muscle’s H-reflex. Indeed, low-level electrical stimulation of the sural nerve, and of other peroneal cutaneous branches of the dorsum of the foot, is capable of reducing presynaptic inhibition of soleus muscle Ia afferent fibers.40

Conclusion
High intensity TES, as administered in this study, does not increase the H-reflex in people with no neurological impairments and presumably has little influence on the excitability of spinal motor neurons or spinal interneurons. Conversely, low-intensity TES significantly increases the H-reflex in these individuals, presumably through the excitation of spinal motor neurons. Future studies will need to determine what influence TES may have on the excitability of spinal motor neurons in people having UMN syndrome.

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