The Therapeutic Effect of Surface Electrical Stimulation on Spasticity

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Abstract:
Purpose: The pathomechanism of spasticity is quite complex. The therapeutic effect of surface electrical stimulation (ES) on spasticity are controversial. To make it clear, the most favorable strategy of ours was shown. It reduced the spasticity significantly by stimulating Ib fiber. Method: Nine cases (8-24 months post-stroke) included in our study were all neurologically stable. We treated their spasticity by applying surface ES (0.2 msec width, 20 Hz bipolar rectangular waves) on the muscle-tendon junction of triceps surae for 20 minutes. The intensity is adjusted at maximum without inducing muscle contraction. Evaluations of spasticity are by Ashworth Scale, F/M ratio, H-reflex latency, H-reflex recovery curve before, 10 minutes and 24 hours after ES. Result: The modified Ashworth Scale shows trend of reduced spasticity 10 minutes after ES, but not reduced at 24 hours after ES. Fmax/Mmax ratio before, 10 minutes and 24 hours after ES are (7.87.5)%, (4.32.7)%, (5.62.3)% respectively; significantly decreased at 10 minutes (p<0.05), while not 24 hours after ES. H-reflex latencies before, 10 minutes and 24 hours after ES are 28.24.4, 28.53.6, 29.00.7 respectively; not significantly prolonged in both 10 minutes and 24 hours after ES statistically. But 7 of 9 cases show prolongation of latencies. H-reflex recovery curves show downward shift at 10 minutes, while not at 24 hours after ES. Conclusion: Surface ES is effective in spasticity suppression 10 minutes after ES, but it can not well last for 24 hours. We suggest the mechanism of spasticity suppression in our study is by stimulation of Ib fiber around the muscle-tendon junction inducing suppression of contraction of the same muscle.

Introduction
Spasticity is defined as “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex. as one component of the upper motor neuron syndrome.”[1] Spasticity in stroke, often interferes with function, limits activity of daily living, and results in pain and contracture.

The therapeutic effect of electrical stimulation (ES) on spasticity is still controversial.[2-7] The differences of these conclusions are due to different application methods. The purpose of this research is to show the most favorable strategy of ours, which suppresses spasticity effectively by stimulating Ib fiber. In this study, we applied surface ES over the muscle-tendon junction of triceps surae to suppress its spasticity and we evaluated spasticity by modified Ashworth Scale, [2] and objectively quantify it by H-reflex latency,[3] H-reflex recovery curve,[4] and Fmax/Mmax ratio.[5]

Subject and Method
Subjects:
Nine stroke cases with evident spasticity were all neurologically stable. There are 8 male patients and one female. The mean age is 58 years old (from 42 to 71). Five cases are right hemiplegic, the other four are left hemiplegic. The mean duration of stroke is 15 months (from 8 to 24). The cases with diabetic mellitus and peripheral neuropathy were excluded. Some cases taking antispastic drugs were asked to maintain on regular schedule.

Procedure:
We treated the spasticity by applying surface ES on triceps surae for 20 minutes. The active electrode was set on the junction of triceps surae muscle and achilles tendon, while the reference electrode was set on the distal end of achilles tendon. The 20 Hz bipolar symmetric rectangular waves with 0.2 msec pulse width were carried. The intensity is adjusted at maximum without inducing muscle contraction.

Evaluation of spasticity are by modified Ashworth Scale, Fmax/Mmax ratio, H-reflex latency ,H-reflex recovery curve before, 10 minutes and 24 hours after ES.

We analyzed data by paired t-test statistically.
Results

The modified Ashworth Scale shows trend of reduced spasticity 10 minutes after ES, but not reduced 24 hours after ES. One case was improved from scale 3 to 2, another one was improved from 2 to 1+, 10 minutes after ES. The scales of the nine cases at 24 hours post-ES were all the same as pre-ES conditions.

Fmax/Mmax ratio before, 10 minutes and 24 hours after ES are (7.87.5)% , (4.32.7) %, (5.62.3)% respectively. Statistically, it shows significantly decreased at 10 minutes (p<0.05), while not at 24 hours after ES (Table 1). It means spasticity was suppressed at 10 minutes, while not at 24 hours after ES.

H-reflex latencies before, 10 minutes and 24 hours after ES are 28.24.4, 28.53.6, 29.00.7 respectively. Although by statistically analysis they are not prolonged significantly in both 10 minutes and 24 hours after ES, but 7 of 9 cases show prolongation of latencies 10 minutes after ES (Table 2). Prolongation of H-reflex latency means suppression of spasticity.

H-reflex recovery curves show downward shift at 10 minutes, while not at 24 hours after ES (Fig 1). It means that the spasticity was suppressed at 10 minutes post-ES.

By our strategy, The effect of surface ES on spasticity suppression is evident 10 minutes post-ES, but it is not so definite 24 hours post-ES.

Table 1: Fmax / Mmax::

<table>
<thead>
<tr>
<th></th>
<th>pre-ES Mean 7.8%</th>
<th>10min Post-ES 4.3%</th>
<th>24hrs Post-ES 5.6%</th>
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<tr>
<td></td>
<td>S.D 7.5%</td>
<td>2.7%</td>
<td>2.3%</td>
</tr>
<tr>
<td>t (paired-t test)</td>
<td>* 0.047 (&lt;0.05)</td>
<td>0.2 (&gt;0.05)</td>
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* Significant

Table 2: H-reflex latency before and after ES:

<table>
<thead>
<tr>
<th></th>
<th>28.24.4</th>
<th>28.53.6</th>
<th>29.00.7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Msec</td>
<td>10min Post-ES</td>
<td>24hrs Post-ES</td>
<td></td>
</tr>
<tr>
<td></td>
<td>t &gt; 0.05</td>
<td>t &gt; 0.05</td>
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Fig 1: H-reflex Recovery curve before and after ES

Discussion

The therapeutic effect of ES on spasticity is still controversial. [2-7] Reviewing the literature, a wide variety of stimulation parameters, application methods and quantification measurement of spasticity made the results different. Alfieri [2] reported that 85-100 % efficacy in decreasing spasticity by using ES on hemiplegic subjects. Robinson et al [6] report increased spasticity by stimulating SCI cases 20 minutes twice a day for 4 week. All of these studies adopt different strategies of ES and measure the spasticity only by subjective evaluation method, such as Ashworth Scale. Ashworth Scale is a widely used measurement of spasticity, but the sensitivity may be not enough to discriminate miniature change. The inter-rater variability can not be neglect. A modification of these scales has been created that adds an additional intermediate grade and has been shown to have high inter-rater reliability. [12] The former difficulty in determining the therapeutic effect of ES on spasticity is objective quantification. In our research, except modified Ashworth Scale, we quantified spasticity by objective measurements, including, Fmax/Mmax ratio, H-reflex latency, H-reflex recovery curve.

In this research, One case was improved from modified Ashworth Scale 3 to 2, another one was improved from 2 to 1+, 10 minutes after ES. 24 hours later, the scales of the nine cases were all the same as pre-ES conditions. The modified Ashworth Scale is a scale to grade muscle tone from 0 (normal) to 4 (severe). [8] Although only 2 cases were improved, It shows trend of reduced spasticity 10 minutes after ES.

The F-wave is less affected by postural change than H-reflex. Fmax/Mmax ratio has been found to be increased in spastic patients. [10] Fmax/Mmax ratio shows significantly decreased at 10 minutes (p<0.05), while not 24 hours after ES (Table 1). It implies the suppression of spasticity is evident 10 minutes after ES, but not evident 24 hours after ES.

H-reflex latencies had been used to assess the Ib fiber autogenic inhibition in spasticity. Delwaide et al [11] found stimulating medial gastrocnemius nerve content with a large number of Ib fibers at the lower part of popliteal fossa inhibits a subsequent H-reflex to a varying degrees. In our study, 7 of 9 cases show prolongation of latencies 10 minutes after ES (Table 2). But statistically H-reflexes are not prolonged significantly in both 10 minutes and 24 hours after ES. It might be due to small number of subjects.

The H-reflex recovery curve reflects polysynaptic modulation of motor neuron excitability secondary to segmental or suprasegmental mechanism. Paired equal stimuli of the tibial nerve are applied in varying temporal arrangement. The resulting H-reflexes demonstrate various phases of inhibition and facilitation, which are shown by downward and upward shift of H2/H1 curve respectively. In our study, H-reflex recovery curves show downward shift at 10minutes (Fig 1), while not 24hours after ES.

In our study, All of the measures of spasticity show significant inhibition of spasticity statistically or show the trend of improvement at 10 minutes post-ES, while the therapeutic effect is not so definite at 24 hours post-ES.

Conclusion:

Surface ES is effective in spasticity suppression 10minutes after ES, but it can not well last for 24hours. We suggest the mechanism of spasticity suppression in our study is by stimulation of Ib fiber around the muscle-tendon junction inducing suppression of contraction of the same muscle.
Reference:


