SUPRESSION OF SPASTICITY BY NEEDLE ELECTRODE ACTIVATING Ib FIBER

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ABSTRACT
The therapeutic effect of electrical stimulation (ES) on spasticity is still controversial. It might be due to different application strategy. In this study, we demonstrated a mechanism of spasticity suppression by Ib fiber activation. 15 stroke cases suffered from spasticity were included. They were all neurologically stable and were 1-3 years post-stroke. We inserted 2 needle electrodes to stimulate the muscle-tendon junction of Gastrocnemius with a 0.2 msec width, 20 Hz bipolar rectangular wave current for 20 minutes. Spasticity was evaluated before ES, 10 minutes after ES and 24 hours after ES. The modified Ashworth Scale showed a trend of reduced spasticity 10 minutes after ES, but not 24 hours after ES. The F/M ratio was significantly decreased 10 minutes after ES (p<0.05), while not 24 hours after ES (p>0.05). The H-reflex latencies showed a trend of prolongation, but were not significantly prolonged statistically in both 10 minutes and 24 hours after ES. H-reflex recovery curves showed upward shift 10 minutes after ES, while not 24 hours after ES. We concluded that the spasticity suppression effect of needle electrode ES is significant 10 minutes after ES, but it can not well last for 24 hours. We suggest the spasticity suppression in our study is by Ib fiber activation.

Keywords: Spasticity, Ib Fiber, Golgi tendon organ, needle electrode, electrical stimulation (ES), modified Ashworth Scale, Fmax/Mmax ratio, H-reflex latencies, H-reflex recovery curves

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 study is to show a new strategy of spasticity suppression. It is based on the mechanism of Ib fiber activation. We inserted 2 needle electrodes to stimulate the muscle-tendon junction of Gastrocnemius 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]

METHOD

15 stroke cases with evident spasticity were included. They were all neurologically stable and were 1-3 years post-stroke. There are 10 male patients and 5 females. The mean age is 58 years old (from 43 to 70). 8 cases are right hemiplegic, 7 cases are left hemiplegic. The cases with diabetic mellitus and peripheral neuropathy were excluded. Some cases taking antispastic drugs were asked to maintain on regular schedule.

To treat spasticity, we inserted 2 needle electrodes with 2.5 cm distance to stimulate the muscle-tendon junction of Gastrocnemius. 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.

RESULTS

The modified Ashworth Scale shows trend of reduced spasticity 10 minutes after ES, but not reduced 24 hours after ES.

Fmax/Mmax ratio before, 10 minutes and 24 hours after ES are (8.0±6.7)%, (4.3±2.5) %, (5.9±2.5)% respectively. Statistically, it shows significantly decrease 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 28.4±4.2, 28.8±4.3, 28.6±3.57 respectively. Although by statistically analysis they are not prolonged significantly in both 10 minutes and 24 hours after ES, but it shows a trend of 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. 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</th>
<th>10min Post-ES</th>
<th>24hrs Post-ES</th>
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<tbody>
<tr>
<td>Mean</td>
<td>8.0%</td>
<td>4.3%</td>
<td>5.9%</td>
</tr>
<tr>
<td>S.D</td>
<td>6.7%</td>
<td>2.5%</td>
<td>2.5%</td>
</tr>
</tbody>
</table>
Table 2: H-reflex latency before and after ES:

<table>
<thead>
<tr>
<th></th>
<th>pre-ES</th>
<th>10min Post-ES</th>
<th>24hrs Post-ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>msec</td>
<td>28.4±4.2</td>
<td>28.8±4.3</td>
<td>28.6±3.57</td>
</tr>
<tr>
<td>t</td>
<td>&gt; 0.05</td>
<td>&gt; 0.05</td>
<td>&gt; 0.05</td>
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DISCUSSION

The therapeutic effect of ES on spasticity is still controversial. Reviewing the literature, a wide variety of stimulation parameters, application methods and quantification measurement of spasticity made the results different.[2-7]

As we know, the Golgi tendon organ can sense the stretching of muscle-tendon junction. When the stretching force exceeding threshold, Golgi tendon organ activate Ib fiber. The impulse goes back to spinal cord to inhibit the contraction of the same muscle. This is a self-protecting reflex to prevent over stretching of muscles.

In our study, stimulating needle electrodes were inserted into the muscle-tendon junction of Gastrocnemius. The results demonstrated an effective spasticity suppression by this stimulation strategy. We suggest that the spasticity suppression mechanism is based on activation of Ib fibers.

REFERENCES