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RESEARCH PAPER

Effects of surface electrical stimulation on the muscle–tendon junction of spastic gastrocnemius in stroke patients

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Abstract

Purpose: The purpose of this study was to explore the effects of spasticity suppression by surface electrical stimulation (ES) on the muscle–tendon junction of spastic gastrocnemius muscles in stroke.

Methods: Twenty-four neurologically stable stroke patients (aged 41 – 69 years, 12 – 35 months post-stroke), with spasticity graded 2 or 3 on the modified Ashworth scale, were recruited and divided into two groups. In the ES group, each patient received 20 min of surface ES once daily, 6 days per week for 1 month. In the control group, ES was used with stimulation intensity kept at zero. To evaluate the therapeutic effect, the modified Ashworth scale, Fmax/Mmax ratio, H-reflex latency, H-reflex recovery curve, and the 10-m walking time were tested before and after the 1-month treatment.

Results: In the ES group, the modified Ashworth Scale showed a trend toward reduced spasticity after 1 month of treatment. The Fmax/Mmax ratio decreased from $8.10\% \pm 4.84\%$ to $4.00\% \pm 1.36\%$; the H-reflex latency increased from 28.87 ± 2.45 ms to 29.40 ± 2.57 ms; the H-reflex recovery curves indicated a downward shift; and the 10-metre walking time significantly decreased after ES. In the control group, none of the measures showed a statistically significant change.

Conclusions: In the study, we demonstrated a way to suppress spasticity at a metameric site and to increase walking speed effectively by applying surface ES on the muscle–tendon junction of spastic gastrocnemius muscles.

Introduction

Spasticity, a debilitating outcome of upper motor neuron lesions, is a common complication of stroke patients and represents a major problem in long-term management. It often interferes with an individual's functions, limits activities of daily living, and results in pain and contracture.

Electrical stimulation (ES) has been used in a variety of ways to suppress spasticity [1 – 18]. In the previous literature, therapeutic ES for spasticity was based on mechanisms of facilitating Renshaw cell recurrent inhibition [9, 14, 16], antagonist reciprocal inhibition [1], and cutaneous sensory habituation [4, 18]. However, using ES to suppress spasticity by facilitating the autogenic Ib inhibitory pathway has never been studied. Delwaide and colleagues [19] assessed the Ib inhibitory effect on the spastic limbs of six hemiplegic adults. The results indicated that the Ib inhibitory effect was absent in hemiplegic spastic limbs. Reduction of Ib inhibition is an additional mechanism contributing to spasticity. Based on the theory, the facilitation of the Ib inhibitory pathway may be a way to suppress spasticity.

Although some studies have shown a significant decrease in spasticity after ES, the therapeutic effects of ES on spasticity remain contradictory [1 – 4, 6 – 8, 12, 13, 15 – 18]. Differences in outcomes may be due to different application methods.

The purpose of this study was to explore the effect of spasticity suppression by surface ES on the muscle–tendon junctions of spastic muscles. Furthermore, a possible mechanism of Ib inhibitory pathway facilitation is discussed.

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Methods

Subjects

After excluding patients with diabetes mellitus and peripheral neuropathy, 24 selected stroke subjects (14 males and 10 females, aged 41 – 69 years, mean = 57), with evident ankle spasticity graded 2 or 3 on the modified Ashworth scale, were recruited and divided into two groups randomly. All patients were neurologically stable. Among the 24 subjects, 13 had right hemiplegia, and 11 had left hemiplegia. The duration of time from the stroke to study inclusion was from 12 – 35 months. Patients taking antispastic drugs were asked to maintain the dosage on a regular schedule.

Procedure

Each patient was in the programme for 1 month. Before and after the 1-month treatment, all patients were tested for the modified Ashworth scale of the ankle, tibial Fmax/Mmax ratio, H-reflex latency, and the H-reflex recovery curves for quantification of spasticity under a double-blind process. In addition, a 10-m walking time was measured. Medelec Premiere equipment was used for electrophysiological evaluation.

Quantification of spasticity

(1) The modified Ashworth scale: The modified Ashworth scale was used following the method described by Bohannon [20].
(2) Tibial Fmax/Mmax ratio: [21]. F-waves and M responses were recorded from the abductor hallucis with 10 mm-diameter metal surface disc electrodes. The intensities of the stimuli of the tibial nerves halfway between the medial malleolus and the Achilles tendon were 25% supramaximal for recording the maximal amplitude of M responses. A stimulation duration of 0.2 ms was used. Ten consecutive supramaximal stimuli were delivered to record the maximal F-wave amplitude.
(3) H-reflex Latency: [22 – 24]. The H-reflex was recorded from the area just below the margin of the belly of the gastrocnemius and was elicited using a submaximal stimulus with a duration of 0.5 ms over the tibial nerve at the popliteal fossa. The positive deflection point from the isoelectrical line was measured as the H-reflex latency.
(4) H-reflex recovery curve: The H-reflex recovery curve was recorded using paired equal stimuli of the tibial nerve in a varying temporal arrangement. Intervals between the paired stimuli were 10, 20, 50, 75, 100, 150, 200, 400, 800, and 1000 ms. The peak-to-peak amplitude ratio of the second H-reflex (H2) over the first H-reflex (H1) was determined at different intervals of stimulation.

Patients were prone on a bed during electrophysiological evaluation, with a pillow under the chest and face downward in a neutral position. The temperature was stably kept at 32°C.

Ten-metre walking time assessment

In the 10-metre walking time measure, patients were asked to walk straight for a distance of 10 metres at a comfortable speed they might use during daily life. The time needed to complete the test was recorded.

Electrical stimulation treatment

After initial evaluation, subjects received 20 min of ES once daily, six times per week for 1 month. An Enraf 982 stimulator was used. Electrical stimulation was delivered through 35 × 45 mm disposable electrodes. The active electrode was placed on the junction of the gastrocnemius muscle and Achilles tendon, while the reference electrode was set on the distal end of the Achilles tendon. To apply the active electrode precisely on the muscle–tendon junction, a mark on the skin was made under ultrasonographic guide. The electrical pulses were bipolar symmetrical rectangular waves. A stimulation frequency of 20 Hz and a pulse duration of 0.2 ms were used. In the ES group, the intensity was adjusted to a maximum without inducing muscle contraction, then kept with a constant-current mode. In the control group, the intensity was kept at zero. After a 1-month course, all quantification measures of spasticity and 10-metre walking time were tested again.

Statistical analysis

The Wilcoxon signed ranks test was employed to compare measurements of spasticity before and after the 1-month programme. One-tailed p values < 0.05 were considered statistically significant throughout.

Results

Age, gender, and duration of time were all matched between the ES group and the control group.

In the ES group, eight of 12 patients showed a decrease in the modified Ashworth scale after 1 month of treatment. Two patients decreased from 3 to 2, five patients decreased from 2 to 1 +, and 1 patient decreased from 2 to 1. The other four patients remained at the same status. In the control group, only 1 of 12 patients decreased from 2 to 1 + . The other 11 patients remained at the same status (Table I).

Table II shows the Fmax/Mmax ratio, H-reflex latency, and 10-m walking time before and after the 1-month programme. In the ES group, the Fmax/Mmax ratio decreased significantly from 8.10% ± 4.84% to 4.00% ± 1.36% after ES ($p < 0.01$); and the H-reflex latency increased significantly from 28.87 ± 2.45 ms to 29.40 ± 2.57 ms after ES ($p < 0.01$). Furthermore, the 10-m walking time significantly improved from 89.75 ± 20.69 to 80.75 ± 19.23 s after ES ($p < 0.01$). In the control group, the measures showed no statistically significant changes.

Table III shows the peak-to peak amplitude ratio of H2/H1. In the ES group, the ratio decreased significantly after ES ($p < 0.01$). The H-reflex recovery curve demonstrated a downward shift after ES (Figure 1).

These results demonstrated a significant spasticity reduction and walking speed improvement using surface ES on the muscle–tendon junctions of spastic muscles.

Discussion

Electrical stimulation has been widely studied for spasticity suppression, however, the results have been contradictory [1 – 18]. The different outcomes may be related to the wide variety of stimulation parameters, application methods, and quantification measurements used. Alfieri reported that spasticity was dramatically reduced during ES by stimulating the antagonistic muscles of the wrist and finger flexors of hemiplegic patients [1]. However, some studies indicated that either increased or unchanged spasticity was found after ES [8, 13]. Robinson and coworkers reported that spasticity increased by stimulating the quadriceps of spinal cord-injured patients required to perform isometric exercises for 20 min twice a day, for 4 – 8 weeks [13]. In our study, using different methods from those used previously, the active electrode was applied to the muscle–tendon junction of spastic muscles to augment Ib fibre activation. Spasticity was significantly reduced after 1 month of ES. We supposed that the main therapeutic mechanism, in theory, was based on the facilitation of the Ib inhibitory pathway of the spastic muscle.

Table I Modified Ashworth scale before and after the 1-month programme in both groups

Modified Ashworth Scale							
ES Group				Control Group			
Case number	Initial	1 month later		Case number	Initial	1 month later	
ES-1	2	1 +	*	C-1	2	2	
ES-2	2	1 +	*	C-2	2	2	
ES-3	2	1 +	*	C-3	3	3	
ES-4	3	2	*	C-4	2	2	
ES-5	2	2		C-5	3	3	
ES-6	3	2	*	C-6	2	2	
ES-7	2	1 +	*	C-7	2	2	
ES-8	2	2		C-8	3	3	
ES-9	2	1 +	*	C-9	2	1 +	*
ES-10	2	1	*	C-10	2	2	
ES-11	2	2		C-11	2	2	
ES-12	2	2		C-12	2	2	

* Decrease in modified Ashworth scale.

Table II Comparison of Fmax/Mmax, H-reflex latency, and 10-m walking time before and after the 1-month programme in both groups

	ES group		Control group	
	Initial	1 month later	Initial	1 month later
F max/M max (%)	8.10 ± 4.84	4.00 ± 1.36**	8.16 ± 4.11	8.23 ± 4.01
H-reflex latency (ms)	28.87 ± 2.45	29.40 ± 2.57**	28.91 ± 2.53	28.85 ± 2.47
10-metre walking time (s)	89.75 ± 20.69	80.75 ± 19.23**	87.91 ± 23.05	88.05 ± 22.88

** $p < 0.01$.

Table III Comparison of H2/H1 ratio before and after the 1-month programme in both groups

Paired stimulating intervals (ms)	H2/H1 ratio (%) ES group Initial	ES group 1 month later	Control group Initial	Control group 1 month later
10	49.08 ± 16.24	39.50 ± 13.19**	48.90 ± 14.34	49.98 ± 13.78
20	14.58 ± 5.66	8.92 ± 4.62**	15.55 ± 7.76	14.58 ± 6.06
50	47.83 ± 13.01	33.25 ± 13.64**	45.98 ± 12.33	45.45 ± 13.67
75	88.08 ± 13.81	71.83 ± 14.98**	85.67 ± 15.46	87.24 ± 12.81
100	102.58 ± 10.95	92.33 ± 10.52**	100.37 ± 13.05	102.34 ± 11.67
150	122.25 ± 7.24	113.75 ± 7.65**	120.34 ± 8.45	119.75 ± 6.52
200	117.50 ± 5.66	112.83 ± 4.84**	117.94 ± 7.06	119.50 ± 7.66
400	110.92 ± 5.02	104.17 ± 3.41**	111.42 ± 6.05	112.95 ± 6.47
800	107.42 ± 3.09	103.25 ± 2.30**	107.95 ± 3.24	106.64 ± 4.38
1000	106.00 ± 3.19	102.75 ± 2.30**	105.82 ± 4.19	106.42 ± 3.50

**$p < 0.01$.

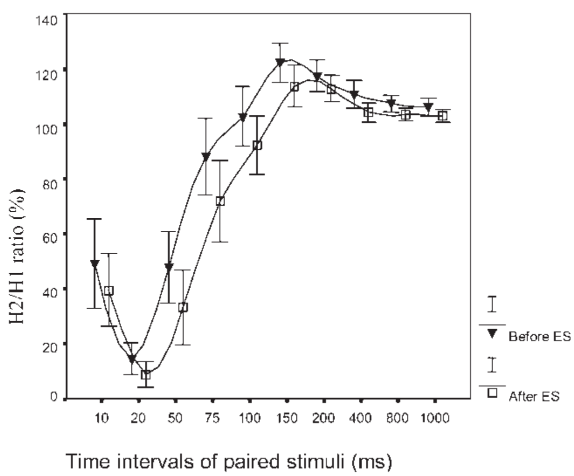

Figure 1 H-reflex recovery curve before and after 1 month of ES. The curve demonstrates a downward shift after 1 month of ES. The values of the H2/H1 ratio significantly decreased after ES in the ES group.

Measurements of spasticity and therapeutic effects of ES

In our study, the 6-level modified Ashworth scale [20] was used instead of the regularly used 5-level Ashworth scale. Although the Ashworth scale is a widely used measure of spasticity, it may not be sensitive enough to detect minute changes. A modification of the scale has been created that adds an additional intermediate grade (1 +) [20]. The modified Ashworth scale is a 6-level scale to grade spasticity. The lowest grade (0) represents normal muscle tone. The highest grade (4) represents severe spasticity. In our study, 8 of 12 patients in ES group showed a decrease in the modified Ashworth scale. In the control group, only 1 of 12 patients showed a decrease. This result indicates that spasticity was more suppressed in the ES group than in the control group.

Difficulties in objective quantification were previously encountered in determining the therapeutic effect of ES on spasticity. In our study, except for the modified Ashworth scale, a subjective measurement with marginal reliability [25], spasticity was quantified by objective measurements, including the Fmax/Mmax ratio, H-reflex latency, and H-reflex recovery curve. Ten-metre walking time was also measured.

The Fmax/Mmax ratio is correlated with motor excitability, and has been found to be increased in spastic patients [21]. In our study, the Fmax/Mmax ratio showed a significant decrease after 1 month of ES. The suppression of spasticity is evident.

The H-reflex, a monosynaptic reflex, was used as a tool to explore the excitability of motor neurons [22–24]. H-reflex latencies have been found to be decreased in spastic limbs [24]. In our study, the result demonstrates that H-reflex latencies were significantly prolonged after 1 month of ES. The spasticity of the gastrocnemius muscle is significantly suppressed.

The H-reflex recovery curve reflects polysynaptic modulation of motor neuron excitability secondary to a segmental or suprasegmental mechanism. Paired equal stimuli of the tibial nerve are applied in a varying temporal arrangement. The resulting H-reflex recovery curve demonstrates various phases of inhibition and facilitation, which are shown by the downward and upward shifts of the H2/H1 curve, respectively. In our study, H-reflex recovery curves showed a downward shift after 1 month of ES. This result suggests that spasticity significantly decreased after ES.

Spasticity interferes with the functioning of hemiplegic limbs in stroke patients. In our study, the 10-m walking time significantly improved after ES. This result indicates that ambulation is improved with spasticity reduction using a 1-month ES treatment.

Mechanisms of the strategy

The strategy used to suppress spasticity in this study was developed based on the mechanism of the activation of the Ib inhibitory pathway. The Ib inhibitory pathway, originating from the Golgi tendon organs, is usually activated by passive stretching and active muscle contraction. Ib afferents project onto interneurons and inhibit the motor neurons of the agonist muscle to protect the muscle from over-contraction (Figure 2). Stimulation of the Ib fibres may lead to a reduction of muscle tone. Delwaide and colleagues found that the Ib inhibitory effect was depressed or absent in the spastic limbs of stroke patients [19]. Reduction of the Ib inhibitory effect is one of pathomechanisms responsible for spasticity in stroke patients. We conducted the study based on this mechanism—the stimulating electrode was placed on muscle–tendon junctions where Golgi tendon organs are located and Ib afferent fibres originate. The application of the electrode on the muscle–tendon junction was to augment Ib fibre activation. After a 1-month ES treatment, spasticity was significantly decreased.

Except for the possible cutaneous sensory habituation mechanism [4, 18], we suppose that the main mechanism of our strategy for spasticity suppression is ES facilitation and the re-establishment of the depressed Ib inhibitory pathway. To confirm the mechanism and to eliminate the cutaneous sensory habituation, direct needle ES in muscle–tendon junctions may be used in further studies.

The effect of surface ES on spasticity suppression is not yet well determined [1, 2, 4–7, 10, 12, 15, 16, 18]. Robinson and coworkers investigated the effects of ES on the spastic limbs of spinal cord-injured patients [13]. Thirty-one subjects underwent a surface ES programme, stimulating the quadriceps to perform 20 min of isometric exercise twice daily, 6 days per week for 4–8 weeks. The results demonstrated a tendency toward increasing spasticity in the subjects undergoing ES. However, in our study, using a new stimulation strategy, spasticity was significantly suppressed after 1 month of ES in stroke patients. We believe that different ES strategies will induce different electrical neuromodulation mechanisms.

Conclusion

Surface ES on the muscle–tendon junctions of spastic gastrocnemius muscles is an effective way to suppress spasticity at the metameric site and to improve 10-m walking time in stroke patients. Different ES strategies can induce different electrical neuromodulation mechanisms and produce different results. In our ES strategy, we suppose that the re-establishment and facilitation of the Ib inhibitory pathway is the possible mechanism for the suppression of spasticity.

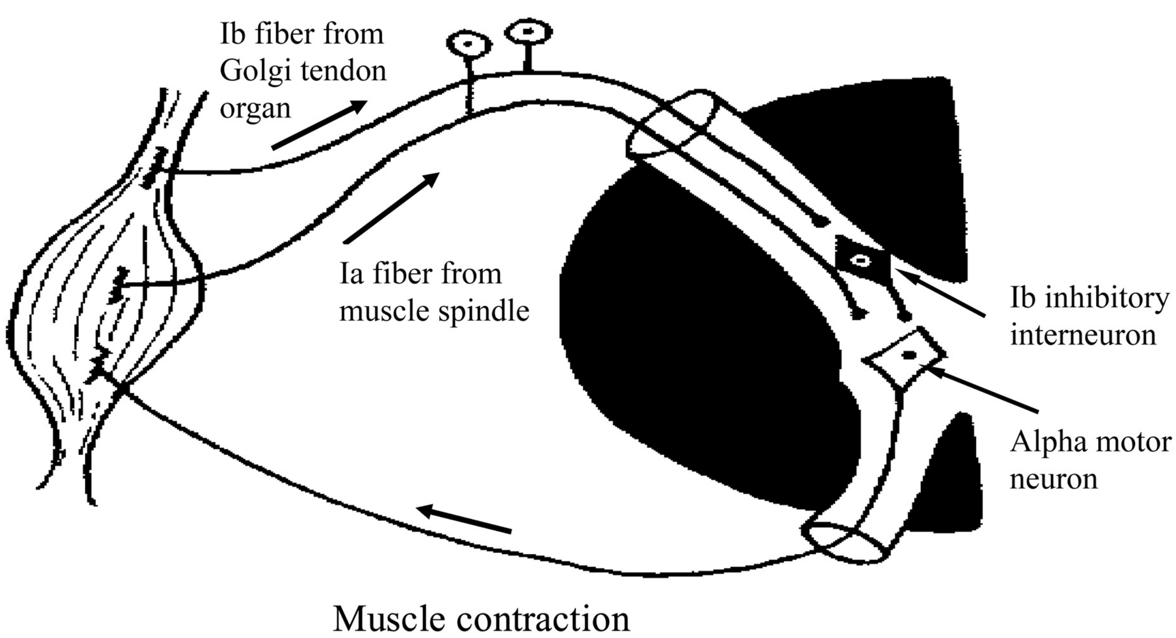

Figure 2 Diagram illustrating the stretch reflex and Ib inhibitory pathway. Activation of the Ia fibre induces reflex muscle contraction and increases the muscle tone. Ib fibres originate from Golgi tendon organs, which are located in the muscle–tendon junction. Impulses pass up the Ib fibre and activate the Ib inhibitory interneuron to suppress the excitability of the alpha motor neuron.

Although the effects are statistically significant, for approving this treatment as a promising therapeutic strategy, it needs to be further studied to learn how long the effects last.

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