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Evaluation of Clinical and Electrophysiological Effects of Electrical Stimulation on Spasticity of Plantar Flexor Muscles in Patients with Stroke

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Abstract

Objective: The aim of this study is to measure the clinical and electrophysiological effects of electrical stimulation on the spasticity of plantar flexor muscles in hemiplegic patients who have plantar flexor spasticity.
Material and Methods: Thirty-two hemiplegic patients having spasticity in lower extremities were included. Study group patients underwent electrical stimulation of the spastic agonist muscles for 20 min per day for 15 days in addition to the conventional program. On the other hand, control group patients underwent the conventional rehabilitation program only. The hemiplegic patients were clinically and electrophysiologically evaluated twice before and after the treatment within 24–48 h.
Results: A noticeable increase in Functional Independence Measure (FIM) scores, strength of ankle dorsiflexion, and range of motion of passive ankle dorsiflexion and a noticeable statistical decrease in the tonus of the ankle plantar flexor were found in the group that underwent electrical stimulation when pretreatment findings were compared to post-treatment findings. While the H/M ratio was found to be noticeably high in both groups ($p=0.005$) in the pretreatment period, no statistical difference was seen in the posttreatment period between groups compared to that in the pretreatment period in terms of electrophysiological statistical parameters ($p>0.05$).
Conclusion: Electrical stimulation can be a good functional option for treating patients having plantar flexor spasticity because it can be applied at home, it has no side effect, it is cheap, it is easy to apply, and it has a good functional performance in addition to the conventional treatment for spasticity.
Keywords: Electrical stimulation, spasticity, H reflex, Ashworth measurement, stroke

Introduction

One of the main goals of rehabilitation is recovery from gait disorder, which hinders functional independence to a great extent in stroke patients. In these patients, spasticity is prominent in the lower extremity, particularly in the extensor muscles of the knee as well as in the ankle plantar flexors and

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inverters; hence, spasticity is one of the most important causes of gait disorder (1).

Spasticity is a finding that is easy to recognize but difficult to evaluate. For the evaluation of spasticity, electrophysiological and biomechanical assessment techniques are used in addition to clinical scales. With electrophysiological techniques, quantitative data, including H-reflex, the ratio of H-reflex amplitude to compound muscle action potential amplitude (H/M ratio), F-response latency, T-reflex latency, tonic vibration latency, and flexor withdrawal response, can be obtained (1).

Spasticity must be treated considering the profit/loss ratio. Pharmacological therapy, surgical interventions, physical therapy, and rehabilitation applications are used for treatment. The effect of electrical stimulation on spasticity is explained by reciprocal inhibition that develops in the spastic muscles after stimulation is applied to the antagonist muscles, but the effect of electrical stimulation that occurs after it is applied to the spastic agonist muscles is defined by recurrent inhibition that develops through fatigue or Renshaw cells (2). The facilitation of Ib inhibitory pathways as a result of the application of stimulation to the muscle–tendon junction of the spastic muscle can be explained by mechanisms such as sensory habituation that occurs because of desensitization at the spinal cord level after recurrent sensory stimulation (2, 3).

The aim of this study was to evaluate the clinical and electrophysiological effects of electrical stimulation applied to the plantar flexor muscles of patients with plantar flexor spasticity.

Material and Methods

The study included 32 patients with stroke in subacute and chronic phases who were hospitalized and were enrolled in a rehabilitation program between September 2009 and February 2010. These patients had spasticity of the lower extremity, particularly in the plantar flexors, and were not given any other treatment for spasticity. They also did not have ankle contractures, a history of diabetes mellitus and similar systemic disease that could cause peripheral neuropathy, or a history and clinical finding of radiculopathy in the lower extremity. Ethical approval for the study was obtained from the local ethics committee of the hospital. Patients were informed about the procedure, and written informed consent was obtained from them.

Patients were randomly divided into two groups. All patients were administered conventional treatment methods (range of joint motion, progressive resistive, stretching, and neurophysiological exercises). Nineteen patients included in the study group were subjected to electrical stimulation for 20 min per day for 15 days (for 5 days per week during 3 weeks), in addition to conventional methods. On the other hand, 13 patients in the control group were subjected to only conventional treatment methods during 3 weeks.

Patient demographic parameters were obtained before treatment. However, clinical and electrophysiological evaluations were performed before treatment and within 24–48 h after treatment.

Clinical Evaluations

The Brunnstrom motor staging approach was used for evaluating the motor functions of patients (4). Extensor spasticity of the knee and plantar flexor spasticity of the ankle were evaluated using the Modified Ashworth Scale (MAS), and ambulation levels were assessed using the Functional Ambulation Scale (FAS) (5-8). The range of motion (ROM) for dorsiflexion of the ankle was measured at the sagittal plane by placing the foot at a right angle to the leg. The axis of the goniometer was placed on the plantar surface of the foot, and the arms of the device were placed parallel to the fifth metatarsal bone and the fibula. The dorsiflexion ROM was evaluated actively and passively. Clonus score, deep tendon reflexes, and dorsiflexion strength of the ankle were evaluated, and deep sensory evaluation was performed. Functional Independence Measure (FIM) was used for assessing the functional states of patients.

Electrophysiological Evaluations

All patients were electrophysiologically evaluated by studying motor and sensory conduction of the ulnar and median nerves in the upper extremity, motor conduction of the tibial and peroneal nerve, and sensory conduction of the sural nerve in the lower extremity. Moreover, H-reflex and M-response of the bilateral gastrocnemius–soleus (triceps surae) muscle and the tibial nerve F-wave response recorded from the abductor halluces longus muscle were measured for all patients before treatment and within 24–48 h after treatment.

Considering external factors that affect spasticity, patients were allowed to rest before the process in a comfortable position in a quiet, moderately bright room with a temperature of 22°C–24°C. A Medtronic Keypoint 4C electromyography (EMG) device (Medtronic, Skovlunde, Denmark) was used for all electrophysiological studies, and stimulation and recordings were performed using superficial electrodes. For recording, silver disc electrodes with a 1-cm diameter were used.

Patients were maintained in the prone position for H-reflex. A pillow was placed under the ankle, with the knee being flexed to approximately 30° and ankle being plantar flexed to approximately 20°. For evaluating H- and M-responses in the triceps surae muscle, a recording electrode was placed in the medial gastrocnemius at the midpoint between the inflection line in the popliteal fossa and the upper edge of the medial malleolus. The tibial nerve was stimulated at the popliteal fossa. Initially, the M-response with the maximum amplitude was obtained and recorded. Then, by increasing the severity of stimulation, the H-wave with the largest amplitude was obtained and recorded. The shortest latency of H-reflex was used. After completing electrophysiological evaluations and calculating the amplitudes of all waves separately, the H/M ratio was obtained by dividing the H-reflex amplitude by the M-response amplitude.

Electrical Stimulation

Patients were laid in a prone position, and the feet were placed in neutral flexion. Electrodes were placed in the medial and lateral gastrocnemius bodies about one-hand width below the popliteal line. The Intelect TENS (D) 77724 device (Chattanooga Group, Hixton, USA) was used for the process. This

device had dual-channel outputs, and the strength of current could be independently adjusted for each channel. For electrical stimulation, biphasic square waves with a frequency of 20 Hz and current width of 300 μs were used. The strength of the applied current was 60–80 Ma, similar to that used for contractions. Patients underwent electrical stimulation for 20 min per day for 15 days (for 5 days per week during 3 weeks).

Statistical Analysis

The data obtained from the study were statistically analyzed using SPSS 15 (Statistical Package for the Social Sciences, Inc., Chicago, IL, USA) software. Dependent or independent sample t-tests were employed for the evaluation of normally distributed data, and Mann–Whitney U test was used for the evaluation of non-distributed data. The Chi-square test was used for the comparison of the groups in terms of their percentage values. In addition, in the comparison of groups in terms of time, variance analysis was used for repetitive measurements for normally distributed data, but Bonferroni correction and Wilcoxon tests were used for non-distributed data. Spearman's test was also beneficial while evaluating the relationship among variables. The data were presented as mean ± standard deviation and median, and a p value of ≤0.05 was considered to be significant.

Results

The study included 19 patients with the mean age of 57.42±12.51 years and 13 patients with the mean age of 58.38±12.9 years. No significant difference was found between the study and control groups in terms of mean ages, etiology, distribution of right–left sides, and hemiplegia times (Table 1).

There was no significant difference between the groups with regard to the Brunnstrom motor stages of the lower extremity, knee extensors, plantar flexor MAS of the foot, clonus score, FAS, FIM, and dorsiflexion of the ankle (p>0.05). Moreover, no significant difference was detected between the groups in terms of the pre-treatment H amplitude, H-reflex latency, M-wave amplitude, H_{max}/M_{max} ratio, F-wave latency, and F-wave persistence values (p>0.05).

A negative correlation was observed between the plantar flexor MAS of the ankle and H-reflex latency (r= -0.45, p=0.009) before treatment. No correlation was observed between the modified Aswhorth scale and H amplitude, M-wave amplitude, F-wave latency, F-wave persistence, and passive ankle dorsiflexion joint ROM (p>0.05).

Table 1. Demographic features of patients

Parameter	Study group (n=19)	Control group (n=13)	p
Age	57.42±12.51	58.38±12.59	0.8[a]
Gender (n; male/female)	14/5	4/9	0.01[b]
Etiology (n; hemorrhage/TECVE)	4/15	4/9	0.6[b]
Lateralization (n; right/left)	6/13	6/7	0.47[b]
Hemiplegia time (month)	10.89±16.85	17.69±20.96	0.147[c]

[a]t-test
[b]Chi-square test
[c]Mann–Whitney U test
TECVE: Thromboembolic cerebrovascular event

Table 2. Pre-treatment electrophysiological comparison of hemiplegic and healthy sides in all patients

Parameter	Hemiplegic side[a] (n=32)	Healthy side[a] (n=32)	p[b]
F-wave latency (ms)	51.51±6.43	50.6±6.29	0.21
F-wave persistence (%)	90.62±14.24	88.43±14.77	0.28
H-reflex latency (ms)	32.3±3.32	33.12±4.41	0.11
H/M ratio	0.37±0.27	0.21±0.24	0.005
H-reflex amplitude (mV)	1.9±1.66	1.41±1.71	0.07
M-wave amplitude (mV)	6.3±3.42	7.58±4.69	0.063

[a]Mean ± standard deviation
[b]Mann–Whitney U test

Table 3. Pre-treatment and post-treatment clinical values of the study and control groups

	Study group (n=19)			Control group (n=13)			
	Pre-treatment[a]	Post-treatment[a]	p[b]	Pre-treatment[a]	Post-treatment[a]	p[b]	p[c]
Brunnstrom	2.78±0.71	3.05±0.84	0.025	3.15±1.14	3.3±1.18	0.15	0.271
FIM	83.1±22.23	86.1±21.62	0.007	87.7±26.88	89.53±28.13	0.007	0.345
FAS	1.42±1.64	1.57±1.57	0.18	2.76±1.78	2.76±1.78	1	0.056
Clonus score	0.84±0.89	0.78±0.91	0.31	0.53±0.77	0.38±0.65	0.31	0.161
DF strength of the ankle	1.84±1.5	2.05±1.58	0.046	2.76±1.64	2.84±1.67	0.31	0138
PF spasticity of the ankle	3.0±3.0	2.6±3.0	0.008	2.61±0.76	2.46±0.96	0.31	0.072
DF ROM of the ankle	12.15±4.64	14±4.72	0.008	12.76±6.3	14.53±6.37	0.043	0.37
10-m walking score	28.37±10.9	24.37±8.12	0.063	36.5±30.04	29.69±23.7	0.028	0.042

[a] Mean ± Standard deviation (median)
[b]Wilcoxon test (pre- and post-treatment comparison within the group)
[c]Wilcoxon test (post-treatment comparison between groups)
FIM: functional independence measure; FAS: functional ambulation scale; DF: dorsiflexion; PF: plantar flexion; DF EHA: dorsiflexion range of motion

Table 4. Pre-treatment and post-treatment electrophysiological values of hemiplegic sides in the study and control groups

	Study group (n=19)			Control group (n=13)			
	Pre-treatment[a]	Post-treatment[a]	p[b]	Pre-treatment[a]	Post-treatment[a]	p[b]	p[c]
F-wave latency (ms)	52.68±7.55	53.55±.92	0.71	49.81±4	50.02±4.7	0.57	0.067
F-wave persistence (%)	93.94±8.09	87.77±19.49	0.19	93.94±8.09	87.77±19.49	0.73	0.086
H-reflex latency (ms)	32.51±3.72	32.85±4.26	0.92	31.75±2.74	31.49±2.27	0.92	0.092
H-reflex amplitude (mV)	2.24±1.79	1.9±1.66	0.6	1.42±1.36	1.21±.69	0.68	0.089
M-wave amplitude (mV)	6.75±3.53	6.5±3.52	0.73	5.64±3.27	5.28±3.93	0.5	0.091
H/M ratio	0.37±0.27	0.35±0.22	0.67	0.37±0.28	0.28±0.2	0.28	0.082

[a] Mean ± Standard deviation (median)
[b] Wilcoxon test (pre- and post-treatment comparison within the group)
[c] Wilcoxon test (pre- and post-treatment comparison between the groups)

Pre-treatment electrophysiological values of hemiplegic and healthy lower extremities of all study and control group patients are shown in Table 2. Accordingly, although the pre-treatment H/M ratio was found to be significantly high on the hemiplegic side (p=0.005), the H amplitude was high on the hemiplegic side, but the difference was not significant (p>0.05).

In the study group, compared with pre-treatment values, there was a significant increase in the post-treatment FIM score, dorsiflexion strength of the ankle, and ankle dorsiflexion ROM and a significant decrease in the plantar flexor tone. On the other hand, in the control group, compared with pre-treatment values, there was a significant increase in the post-treatment FIM score and ankle dorsiflexion ROM, but no significant difference was observed in the lower extremity tone. In both the study and control groups, no significant difference was observed in terms of post-treatment Brunnstrom motor staging, FAS, and clonus scores. There was a post-treatment reduction in 10-m walk time in both the groups, but it was significant in the control group (Table 3).

No significant difference was observed with regard to the post-treatment Brunnstrom motor staging, FIM, FAS, clonus score, dorsiflexion strength of the ankle, plantarflexor MAS, and ankle dorsiflexion ROM values between the groups ($p>0.05$). However, with regard to the 10-m walk time, a significant decrease was observed in the study group compared with the control group (p>0.05) (Table 3).

In both the groups, there was no significant difference in electrophysiological parameters (H amplitude, M-wave amplitude, H_{max}/M_{max} ratio, H-reflex latency, F-wave latency, and F-wave persistence) measured before and after treatment (Table 4).

Discussion

Spasticity is a complication that is easy to identify, but it is difficult to evaluate its treatment quantitatively. In addition, its measurement has always posed a problem for clinicians and researchers (9, 10). Although the Ashworth scale is commonly used for measuring spasticity, it is not sensitive enough to determine small changes in spasticity, and its reliability has been demonstrated to vary depending on different muscles and joints (11). Sloan et al. (12) suggested the use of MAS in testing upper extremity spasticity to be more reliable among people evaluating spasticity but they concluded that it was not reliable for lower extremity spasticity. In particular, in plantar flexor spasticity, the short lever arm of the ankle makes it difficult to determine its resistance to motion (13). In the evaluation of spasticity, goniometric measurement of ROM and posture has been found to be reliable at varying rates among evaluators (14-17). The Ashworth scale or other scales such as MAS and ROM are subjective, but they are not always sufficient for distinguishing spasticity from other biomechanical and neural factors (18). Electrophysiological parameters (H-reflex and H/M ratio, F-wave response, T-reflex, tonic vibration reflex, flexor withdrawal response) can measure spasticity more objectively and can be used as a part of clinical evaluation (19).

In 1963, Hoffman and Angel (20) found an increase in the H-reflex amplitude in spastic patients. Some studies revealed that compared with healthy individuals, a significant increase in the H-reflex amplitude is observed on the spastic side in spastic patients (21, 22). The H/M ratio compares the total and maximal counts of motor neurons that are activated with reflex and are affected by excitatory and inhibitory stimuli, and it shows a decrease in the presynaptic inhibition level with the level of motor neuron excitability (23). This ratio is more sensitive to the stimulation of motor neuron excitability (24, 25). In our study, although the H/M ratio was found to be significantly higher on the hemiplegic side, it was not correlated with MAS. In patients with spasticity, H-reflex latency is observed to be shortened, but the values are close to normal. In healthy individuals, the maximal difference in the soleus H-reflex latency between two extremities is 1.5 ms at the most and 0.4–0.5 ms on average (26, 27). On the other hand, in patients with unilateral spasticity, the difference in H-reflex latency between two sides can exceed the upper limit. Although H-reflex latency tended to decrease on the spastic side, the mean difference was <1 ms. A negative correlation was detected between plantar flexor MAS and H-reflex latency on the spastic side.

F-wave, which is important for obtaining information about distal and proximal segments of motor nerves, is also used for spasticity. Although F-wave persistence and the F/M ratio in-

crease in spasticity, F-wave latency has been found to be prolonged (28). F-waves were previously used for evaluating alpha motor neuron activity in 120 stroke patients, and they were found to be more advantageous than H-reflex. It was thought that F-waves demonstrated only alpha motor neuron activity and were not affected by presynaptic inhibition of H-reflex. In addition, it was found that F_{max}/M, Fort/M, and Fort/F_{max} ratios increased on the hemiplegic side (29, 30). In our study, a significant difference was not observed in hemiplegic and healthy extremities before treatment, but electrophysiological evaluation revealed a prolonged tendency of F-wave latency prolongation and an increase in F-wave persistence on the hemiplegic side.

Electrical stimulation is used for decreasing the pain, developing and strengthening the muscle, facilitating learning of motor function again, taking over the task of lost motor function, increasing blood flow, accelerating wound healing, increasing the growth and fusion of bone, and improving motor and sensory peripheral nerve regeneration. Furthermore, studies on spasticity suppression have been commonly conducted, and contradictory results have been obtained (31). Moreover, some studies have found that the spasticity increases or remains same after electrical stimulation (32, 33). The varying results have been attributed to the variability of stimulation parameters, application method, and measurement parameters (19, 34).

In the study conducted by Alfieri (35), electrical stimulation was applied on the antagonists of spastic muscles in stroke patients. He found a decrease in muscle tone and the effect of electrical stimulation to be associated with the reciprocal inhibition principle. In a study conducted with electrical stimulation applied to the muscle–tendon junction of the spastic gastrocnemius muscle in stroke patients, the inhibition of gastrocnemius spasticity was targeted by facilitating the 1b inhibitor pathway. At the end of the study, a decrease was detected in plantar flexor spasticity (3). In another study, neuromuscular electrical stimulation was implemented in the extensor muscles of the hand, and functional magnetic resonance imaging revealed an increase in the cortical density of the ipsilateral somatosensory cortex (36). Bogataj et al. (37) reported that neuromuscular electrical stimulation increased sensory inputs into the central nervous system and thus accelerated motor learning by increasing neuronal plasticity. In some studies, such as this, the mechanism of electrical stimulation for decreasing spasticity was associated with its effect on the central nervous system (38, 39). In our study, compared with before treatment, the plantar flexor tone of the ankle was decreased after treatment in the group receiving electrical stimulation. On the other hand, in the control group, no difference was observed in the tone. The aim of electrical stimulation application in spastic agonist muscles was to create fatigue in the spastic muscle and to decrease spasticity by increasing recurrent inhibition that developed through Renshaw cells.

Chen et al. (3) found that electrophysiological findings correlated with motor excitability after applying electrical stimulation to the muscle–tendon junction of the gastrocnemius muscle on the spastic sides of stroke patients, but they could not show the long-term effect of electrical stimulation. Similarly, in our study, a significant increase in the H/M ratio was observed on the hemiplegic side before treatment. However, despite decreased spasticity after electrical stimulation, no significant difference was observed between the pre-treatment H/M ratio and post-treatment H/M ratio. According to the study of Bakhtiary and Fatemy (40), electrical stimulation affected spasticity positively, but this effect was observed only soon after the application. The long-term effect of electrical stimulation and its effect on functional activity could not be evaluated.

According to Lin et al. (41), the walking speed and temporal asymmetry are predominantly influenced by the strength of the dorsiflexor muscle; in fact, plantar flexor dynamic spasticity affects the spatial gait asymmetry in socially ambulating patients. A study has shown that spasticity does not have any effect on walking speed (42). A previous study (43) has shown that 10-m walking speed is the evidence of social life in patients who can be ambulated after stroke; hence, this test was performed and their walking speed was evaluated. In our study, an increase was observed in the post-treatment 10-m walking speed in both the groups, but the difference was not significant.

The effect of electrical stimulation on spasticity within 24 and 48 h was evaluated in our study. However, further studies conducted on larger patient populations are needed for demonstrating a longer effect. In our study, a single parameter was used in electrical stimulation, but different stimulation parameters should have been used, considering the fact that electrical stimulation could increase spasticity in some parameters or for determining parameters in which it could decrease spasticity. Another limitation of our study was that healthy extremities of hemiplegic patients were used as controls. The absence of a significant difference between hemiplegic and healthy sides, except in the H/M ratio, can suggest that the healthy extremity was also affected. In a study conducted on stroke patients, it was specified in the electrophysiological evaluation that the healthy extremity may have been affected, and healthy individuals had to be involved in the control group. However, personal differences could not be ruled out in this situation (44).

Conclusion

We suggest that when appropriate parameters are used, in addition to conventional treatment methods, electrical stimulation can be a good choice for the treatment of spasticity in stroke patients. This is because it provides a better functional performance and its application is easy and inexpensive, with fewer side effects.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Ankara Physical Medicine and Rehabilitation Training and Research Hospital.

Informed Consent: Verbal informed consent was obtained from patients and patients' parents-who participated in this study.

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Collection and/or Processing - A.G., B.Ö.; Analysis and/or Interpretation - A.G.,B.S.; Literature Search - A.G., A.A.Y.; Writing Manuscript - A.G., B.S.; Critical Review - M.A.

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