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ORIGINAL RESEARCH ARTICLE

Role of Electrical Stimulation Added to Conventional Therapy in Patients with Idiopathic Facial (Bell) Palsy

ABSTRACT

Tuncay F, Borman P, Taşer B, Ünlü İ, Samim E: Role of electrical stimulation added to conventional therapy in patients with idiopathic facial (Bell) palsy. Am J Phys Med Rehabil 2015;94:222–228.

Objective: The aim of this study was to determine the efficacy of electrical stimulation when added to conventional physical therapy with regard to clinical and neurophysiologic changes in patients with Bell palsy.

Design: This was a randomized controlled trial. Sixty patients diagnosed with Bell palsy (39 right sided, 21 left sided) were included in the study. Patients were randomly divided into two therapy groups. Group 1 received physical therapy applying hot pack, facial expression exercises, and massage to the facial muscles, whereas group 2 received electrical stimulation treatment in addition to the physical therapy, 5 days per week for a period of 3 wks. Patients were evaluated clinically and electrophysiologically before treatment (at the fourth week of the palsy) and again 3 mos later. Outcome measures included the House-Brackmann scale and Facial Disability Index scores, as well as facial nerve latencies and amplitudes of compound muscle action potentials derived from the frontalis and orbicularis oris muscles.

Results: Twenty-nine men (48.3%) and 31 women (51.7%) with Bell palsy were included in the study. In group 1, 16 (57.1%) patients had no axonal degeneration and 12 (42.9%) had axonal degeneration, compared with 17 (53.1%) and 15 (46.9%) patients in group 2, respectively. The baseline House-Brackmann and Facial Disability Index scores were similar between the groups. At 3 mos after onset, the Facial Disability Index scores were improved similarly in both groups. The classification of patients according to House-Brackmann scale revealed greater improvement in group 2 than in group 1. The mean motor nerve latencies and compound muscle action potential amplitudes of both facial muscles were statistically shorter in group 2, whereas only the mean motor latency of the frontalis muscle decreased in group 1.

Conclusions: The addition of 3 wks of daily electrical stimulation shortly after facial palsy onset (4 wks), improved functional facial movements and electrophysiologic outcome measures at the 3-mo follow-up in patients with Bell palsy. Further research focused on determining the most effective dosage and length of intervention with electrical stimulation is warranted.

Key Words: Bell Palsy, Rehabilitation, Electrical Stimulation, Facial Disability Index, Electrophysiologic

Idiopathic facial palsy (Bell palsy [BP]) is the most common peripheral lesion of the cranial nerves and the most common mononeuropathy.[1–3] The clinical presentation of facial nerve palsy depends on the location, pathophysiology, and severity of the lesion.[4] Although two-thirds of patients progress to full recovery within 3 mos, residual symptoms may persist in about one-third of the patients and approximately 5% are seriously handicapped with permanent disfigurement or sequelae. These patients are usually associated with significant axonal loss, and some permanent facial weakness may remain.[2,4,5]

Local heat therapy, facial exercises, massage, or taping to lift drooping flaccid face muscles are the treatments of choice in the rehabilitation of patients with BP.[6] Electrical stimulation (ES) continues to be included (or at least considered by some practitioners in some countries) as a clinical intervention for BP, but evidence to support its use is limited, and there is controversy over whether ES in various forms is helpful, has no substantial effect, or may inflict harm to patients with BP.[7]

A previous Cochrane analysis reported that all ES studies related to BP had low quality. Comparisons of ES with prednisolone (149 participants) or the addition of ES to hot packs, massage, and facial exercises (22 participants) yielded insignificant differences between the groups. A single low-quality study with 56 patients reported worse functional recovery with ES.[7] However, most of the included study samples were too small to reach sufficient statistical power, and most of them were of poor quality and not well designed, with inadequate descriptions of methods: mode of delivery, apparatus, randomization, and follow-up. Despite the accumulating research, incongruity exists between the study designs and the conclusions drawn from them. It is difficult to generalize these conclusions and use these results in the clinical setting.

There are conflicting data in last century textbooks on the effect of initiating ES late after denervation injury. Most of the data suggest that maximal maintenance of normal motor unit characteristics in a short-term stimulation program depends on the very early initiation of stimulation.[8] In BP, some previous studies have indicated that ES produced no benefit over placebo, with incomplete recovery at 6 mos.[7] Low-quality studies reported worse functional recovery with ES, but most applied ES in the chronic phase[9] or used eutrophic stimulation on different nerves of the face.[10] The data supporting the efficacy of ES in human studies are less clear than noted in animal models.[8] Cederwall et al.[11] have indicated that the use of ES was disruptive to reinnervation and thus might be contraindicated for individuals with facial nerve disorders. In contrast, Foecking et al.[12] have studied ways to enhance the regeneration of peripheral nerves using daily ES after a facial nerve injury on animal models, and their findings demonstrated that ES enhanced the recovery of most functional parameters in a rat model.

The aims of this study were to evaluate the effect of ES added to conventional physical therapy during the early period of recovery from BP on clinical, functional, and electrophysiologic outcome measures.

MATERIAL AND METHODS

This study was a prospective, randomized controlled trial performed on 60 BP patients, who were recruited from the Department of Physical Medicine and Rehabilitation and the Department of Otorhinolaryngology, Ankara Training and Research Hospital, between March 2010 and May 2012. The study protocol was approved by the local ethical committee, and all participants gave informed consent before treatment.

Seventy-three patients with BP were recruited to the study, and 60 consecutive patients were enrolled. Six patients dropped out before the beginning of the study and seven patients were excluded in the follow-up visit. Figure 1 summarizes the flowchart regarding patients' enrollment. Patients were randomly assigned to one of two treatment groups by drawing a sealed envelope from a box; envelopes were identical in size and color. All patients were first assessed by the same ear, nose, and throat specialist (I. Ünlü) within 48 hrs after onset. The patients were diagnosed as having BP after having ruled out a tumor, stroke, and Lyme disease, by physical examination, magnetic resonance imaging studies, and laboratory tests including blood work. All patients were treated with oral corticosteroids, beginning at a dose of 60 mg/day within the first 48 hrs after onset of symptoms and progressively tapered down during the next 10 days. The patients were then assessed for eligibility by the same physiatrist (B. Taşer). Criteria for inclusion were as follows: (1) new onset of idiopathic facial paralysis within 48 hrs and (2) either sex in the age group of 18 to 79 yrs. Patients with central nervous system disease, diabetes mellitus, varicella zoster virus infections, and recurrence of facial paralysis and those who were noncompliant and not presenting for follow-up visits were excluded.

Clinical and Functional Evaluation

During the clinical examination, each patient's ability to wrinkle his/her forehead, close his/her eyes,

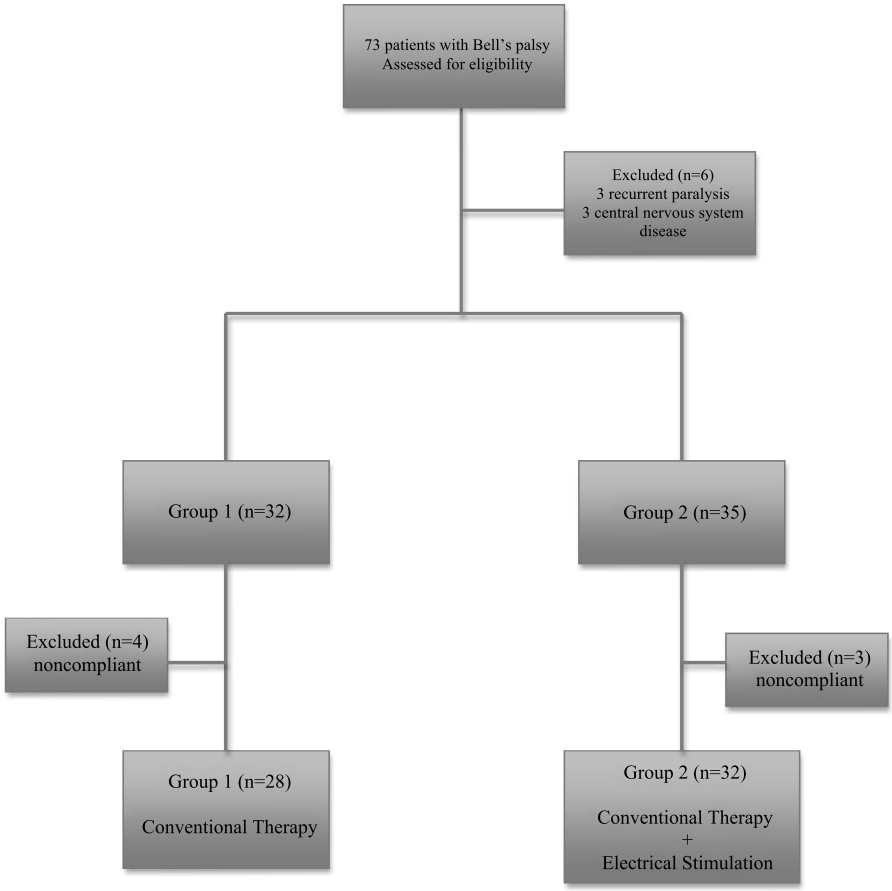

FIGURE 1 *Flow diagram of the study.*

and smile was evaluated. Static and dynamic facial asymmetry was assessed, and facial strength was tested manually.[13] Subsequently, electrophysiologic tests were done 4 wks after the onset of paralysis. Functional outcome measures were defined by scores on the House-Brackmann (HB) scale and Facial Disability Index (FDI), whereas electrophysiologic outcome measures were defined as latency and amplitude of compound muscle action potentials (CMAPs) of the frontalis and orbicularis oris muscles. Grading facial nerve function was performed using the HB scale (grade I, normal, to grade VI, total paralysis).[2] The assessments of the physical and social/well-being function were performed using the FDI. The FDI is a self-report, disease-specific instrument designed to provide the clinician with information about disability and is related to the social and emotional well-being of patients with facial nerve palsy. Voluntary movement is rated on a scale of 1 to 5, with 1 representing no movement and 5 indicating facial movement equal to the movement of the uninvolved side of the face in physical function.[14] The outcome measures were collected before the therapy at the fourth week after the onset of the palsy and 12 wks after the therapy.

The blinded functional assessment was done at 4 and 12 wks after the participant's decision to be enrolled in the study (P. Borman).

Electrophysiologic Evaluation

Motor nerve conduction studies and electromyography were performed between days 21 and 30 to assess the presence of denervation signs and to determine the presence of axonal degeneration. Needle electromyography was carried out at rest, during minimal to moderate and maximal voluntary contraction. The presence of abnormal spontaneous potentials such as fibrillations and positive sharp waves showed denervation of facial muscles. Motor unit action potential and recruitment analyses were performed to evaluate the presence and the severity of axonal degeneration and the signs of reinnervation. Recruitment analysis showed four types of patterns, which were full, reduced, discrete, and absent.[4,5,15,16]

Motor nerve conduction was performed stimulating the tragus and was recorded using bipolar concentric needle electrodes, which were placed 2 cm above the level of the frontalis muscle (temporal branch) and 2 cm below the lower lip and 2 cm

lateral of the midline for the orbicularis oris (buccal branch) muscle. The surface ground electrodes were placed on the proximal upper extremity of the affected side. Tendon-belly principle was considered in the placement of needle electrode. Supramaximal stimulus intensity was delivered for the motor nerve conduction studies.

Distal motor latency was measured from the stimulus onset to the initial deflection of the CMAP. All subjects were examined in the supine position using Nihon Kohden Neuropack M1 QP-954 BK equipment (Tokyo, Japan) with standard filter settings of 2–10 kHz. Sweep speed was 10 msecs per division and was adjusted to allow recording of peak-to-peak motor unit potentials. All needle electromyographic examinations were performed by the same physician (F. Tuncay), who was blinded to the subjects' identity and clinical data.

Treatment Methods

All patients were also instructed to protect their eyes and were educated regarding compensation strategies, posture, and diet modification. The patients were also instructed as to the correct practice of facial expression exercises, and balloon blowing and chewing gum on the paralyzed side were advised to all patients.

The study-related physical therapy and ES interventions began approximately 4 wks after diagnosis and after the first blinded assessment. The patients in group 1 ($n = 28$) received physical therapy including hot pack, massage to the facial muscles, and facial expression exercises via a mirror,[11] five times per week over 3 wks. Patients in group 2 ($n = 32$) received ES daily in addition to the same physical therapy provided to group 1. A monophasic waveform having 100 msecs of pulse duration, 300 msecs of interpulse interval, and a pulse rate of 2.5 pulses/sec were used. ES was produced by a Dynatron 438 device (Enraf, Germany) and delivered via carbon-rubber electrodes; a 3-cm^2 anode was placed over each muscle, and a 7-cm^2 cathode was placed over the proximal part of ipsilateral arm. ES was applied to each of 11 facial muscles (frontalis, corrugator supercilii, palpebral part of orbicularis oculi, levator labii superioris alaeque nasi, levator labii superioris, levator anguli oris, risorius, orbicularis oris, depressor anguli oris, depressor labii inferioris, and levator menti) to evoke three sets of 30 minimal contractions, five days a week for a period of 3 wks. Both groups were treated by the same physiotherapist.

Statistical Analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences v 13.0 (SPSS Inc, Chicago, IL) program. The data were analyzed using the Wilcoxon's signed-rank test to compare the HB scale scores, FDI scores, and motor nerve latencies and amplitudes within each group at baseline and 3 mos after the therapies. Mann-Whitney U test was used to compare the measures (HB score, FDI scores, and motor nerve latencies and amplitudes) between groups.

The data of HB scores and FDI scores were analyzed using nonparametric methods, which is appropriate based on the type of data. Therefore, the median ranks were calculated for these parameters. A value of $P < 0.05$ was considered statistically significant.

RESULTS

Sixty patients (mean age, 44.8 ± 17.6 yrs; range, 18–79 yrs) were included (29 men and 31 women). The demographic data of the patients are given in Table 1. The mean age, body mass index, sex, side of paresis, and duration of symptoms were similar between the groups. Sixteen of 28 (57.1%) patients in group 1 and 17 of 32 (53.1%) patients in group 2 had no axonal degeneration, while 12 (42.9%) patients in group 1 and 15 (46.9%) patients in group 2 had axonal degeneration. The baseline HB and FDI

TABLE 1 Demographic and clinical characteristics of the groups

Variable	Group 1 ($n = 28$)	Group 2 ($n = 32$)	P
Age, yrs	41.5 ± 18.1	47.7 ± 17.3	0.17
BMI, kg/m^2	27.8 ± 3.1	27.8 ± 3.6	0.94
Sex, F/M	16/18	15/17	0.43
Duration of symptoms, day	21.3 ± 12.0	21.6 ± 12.5	0.90
Side of paresis, R/L	20/8	19/13	0.33
Axonal degeneration, %	12 (42.9)	15 (46.9)	0.23

Data are presented as mean ± SD.
Levene test, $P < 0.05$ significant value.
BMI indicates body mass index.

TABLE 2 Median rank HB scores of the patients before and after the treatment periods

	Group 1	Group 2	*P*
Pretreatment	3 (2–4)	3 (2–4)	0.43[a]
Posttreatment	2 (1–4)	1 (1–3)	0.03[a]
P	0.03[b]	0.0001[b]	

Data are presented as median (range).
$P < 0.05$ significant value.
[a] Mann-Whitney *U* test.
[b] Wilcoxon's test.

scores (physical function and social being function) were similar between the groups ($P = 0.43$, $P = 0.34$, $P = 0.54$).

None of the patients had total paralysis (H5 and H6). Posttreatment HB scores, indicating the clinical state, were better in group 2 than in group 1, as shown in Table 2 ($P = 0.03$). The FDI scores, representing functional status, improved significantly in both groups after the therapies, but posttreatment scores in group 2 were statistically higher than in group 1 (physical function, $P = 0.02$; social/well being function, $P = 0.03$) (Table 3). The volitional movement of the facial muscles also improved substantially in both groups, as evidenced by increases in mean FDI physical function scores.

Table 4 indicates the pretreatment and posttreatment values of latency and amplitude recorded from the orbicularis oris and frontalis muscles. No meaningful difference existed between the two groups at baseline in terms of CMAP amplitudes and latencies of the frontalis and orbicularis oris muscles ($P = 0.45$, $P = 0.07$, $P = 0.23$, $P = 0.87$). Both CMAP amplitudes and latencies of the tested facial muscles improved in group 2 after the therapies. In the patients of group 2, the posttreatment mean distal motor latencies in the frontalis and orbicularis oris muscles were significantly shorter ($P = 0.04$, $P = 0.01$), and the CMAP amplitudes of these muscles were significantly increased ($P = 0.02$, $P = 0.02$) compared with group 1. In group 1, the only significant electrophysiologic improvement was found in the CMAP latency of the frontalis muscle ($P = 0.03$).

DISCUSSION

ES has achieved recognition as a suitable method in partial nerve palsies and has been used to minimize atrophy and maintain contractile properties of the muscles. If there truly are benefits of ES, these may be a result of enhancing reinnervation and/or helping to maintain existing motor units.[17]

Physical therapy for patients with facial paralysis has consisted traditionally of local therapeutic heat, generic facial exercises, massage, and sometimes ES.[18] However, there is still controversy regarding the use of ES for peripheral facial nerve paralysis. The presented findings should help to resolve the controversy. This randomized controlled study assessed the adjunctive effect of ES applied shortly after BP onset and combining it with conventional physical therapy treatment options. The results presented herein indicate a significant improvement in electrophysiologic outcome measures as well as a better recovery on the HB grading system, in favor of the additional ES therapy. The randomization, sample size, stimulation protocol, and clearly defined intervention period of this study followed the guide of credible experimental design that differed from earlier published studies.

Unlike this study, Targan et al.[19] provided monophasic short-duration 86-μsec pulses at low intensity to only four facial muscles in their experimental group with chronic BP. After 6 mos of ES, significant improvements were observed in recovery of the HB scale and nerve conduction latency abnormalities, but

TABLE 3 Pretreatment and posttreatment FDI scores in both groups (median and ranges)

FDI Components	Group 1 ($n = 28$)	Group 2 ($n = 32$)	*P*
Physical function			
Pretreatment	40 (25–90)	50 (20–80)	0.34
Posttreatment	85 (30–100)	100 (65–100)	0.02[a]
P	**0.000[b]**	**0.000[b]**	
Social/well-being function			
Pretreatment	68 (20–96)	68 (16–88)	0.54
Posttreatment	88 (28–100)	96 (56–100)	0.03[a]
P	**0.001[c]**	**0.001[c]**	

Data are presented as median (range).
[a] $P < 0.05$ significant value.
[b] $P < 0.0001$.
[c] $P < 0.001$.

TABLE 4 Electrophysiologic outcome measures of the two groups according to treatment period

	Group 1			Group 2			Group 1-Group 2 *P*	
	Pretreatment	Posttreatment	*P*	Pretreatment	Posttreatment	*P*	Pretreatment	Posttreatment
Frontalis								
CMAP amplitude, mV	1.1 ± 0.6	1.2 ± 1.0	0.12	1.3 ± 1.3	1.6 ± 1.2	**0.02**	0.45	**0.02**
CMAP latency, msec	4.4 ± 2.1	3.9 ± 1.1	**0.03**	4.9 ± 2.7	3.7 ± 1.2	**0.01**	0.07	**0.04**
Orbicularis oris								
CMAP amplitude, mV	1.2 ± 1.1	1.3 ± 1.6	0.09	1.0 ± 0.9	1.5 ± 0.9	**0.02**	0.23	**0.02**
CMAP latency, msec	3.6 ± 1.6	3.2 ± 2.5	0.34	3.8 ± 2.7	2.8 ± 1.2	**0.01**	0.87	**0.01**

Data are presented as mean ± SD.
Boldface values indicate $P < 0.05$.

not in clinical presentation like synkinesis, tearing, and drooling.[19,20] Farragher et al.[10] also studied chronic BP subjects, averaging 74 mos of paresis, all having markers of denervation, and applied ES at a frequency designed to mimic the pattern of motor activity characteristic of healthy facial muscles. They attempted to exert a trophic effect that would enhance reinnervation. Thirty-nine patients were allocated to either the ES group or the control group. In contrast to this study's design but similar to Targan et al.,[19] ES was provided twice a day for 3–5 hrs with a stimulation intensity set at visible motor threshold using short-duration 80-μsec compensated rectangular monophasic pulses.[10] Farragher et al.[10] suggested a benefit from ES when added to facial exercises and massage, but the absence of a true control group (patients in the control group were lost to follow-up) makes it difficult to determine whether the therapeutic benefits were related to ES or simply to prolonged attentive therapy. Accordingly, the use of short (microsecond) pulses and pulse frequency limited to 8–10 pps, even if applied for 6–12 mos during the chronic phase of facial palsy, remains controversial and can be questioned.

Mosforth et al.[9] studied 86 patients with acute BP and, similar to this study's protocol, used 100-msec duration pulses given daily. However, they stimulated the facial muscles for 6 mos and did not include a control group. In contrast to this study's findings, they reported neither harm nor therapeutic benefit with regard to the conduction block or denervation. In this study, ES was performed 5 days a week for a period of only 3 wks and yet efficacy of ES in terms of improving HB and FDI scores and electrodiagnostic parameters is reported. It is conceivable that the stimulation dose reported here providing three sets of 30 minimal visible contractions to each of 12 facial muscles was a primary contributor to a better recovery of patients receiving ES.

Monophasic waveform, pulse duration of 100 msecs, and pulse rate of 2.5 Hz were used based on the classic knowledge that relatively long pulse durations should satisfy the prolonged chronaxies of the denervated muscle fibers.[21] However, unlike in humans, in a couple of published animal studies, ES has been reported to suppress sprouting and cause delayed nerve outgrowth.[22,23] On the contrary, Foecking et al.[12] used rats with unilateral facial nerve crush injury and demonstrated that one session of ES was as effective as daily stimulation at enhancing the recovery of most functional parameters. They applied the stimulation directly to the nerve proximal to the crush site, using a single 30-min session of ES, and suggested such protocol as a possible treatment strategy for humans with paralysis as a result of acute nerve injuries.[12] Whether such animal model data are relevant to humans with damaged facial or other peripheral nerves remains untested and unknown.

There are some limitations of this study. The value of electrophysiologic testing for outcome prediction of acute peripheral facial nerve palsy remains controversial. The nerve excitability test can be used in the acute phase, and it is recommended to define the prognosis.[24] The authors did not aim to indicate prognosis and therefore did not perform nerve excitability tests in this study. Moreover, the finding of the present investigation may not be applicable to patients with HB grading of 5 or 6 as these were not included in this study's sample. Future studies are needed to evaluate the efficacy of ES in BP patients with complete paralysis.

A total of 15 stimulation sessions were provided over 3 wks while the recovery continued (at least in some patients) for 3 mos. It could be possible to

enhance the recovery of the experimental group *vs.* the control if the stimulation would be continued for longer periods.

Another shortcoming is the failure to assess patients' satisfaction. Further studies considering patient satisfaction would be valuable.

CONCLUSION

Within the limits of study design, it was demonstrated that the addition of ES to traditional physiotherapy intervention during the early phase of recovery from BP was superior to not adding it, when functional classification and electrophysiologic recovery profiles were considered after 3 mos of follow-up. Continued research is needed to determine the correct dose, stimulation intensity, frequency, or the number of ES treatments required in BP patients to maximize recovery. The use of therapeutic ES in the early phase of BP should be encouraged. It seems worthy to continue with a large clinical trial on the use of ES in the treatment of BP to standardize the ES treatment intervention in this patient group.

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