

REHABILITATION OF BELL'S PALSY PATIENTS USING NERVE MUSCLE STIMULATOR DEVICES

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Abstract

Background: Bell's palsy is a common type of hemifacial nerve paralysis with acute onset that happens without any recognizable or evident reason. **Objective:** To critically analyze and compare the efficacy of a nerve muscle stimulator, also known as an NMS device, in improving and developing the function of facial muscles in patients with Bell's palsy throughout a holistic one-year duration.

Methods: A prospective interventional study was conducted on 980 patients (aged 18–85) diagnosed with Bell's palsy at clinic in Tashkent. All patients received standard physiotherapy combined with NMS therapy for four weeks. Assessments were conducted at baseline and after 3 months. **Results:** Statistically significant improvements were noted in all parameters measured, reflecting a significant enhancement. Muscle strength showed a dramatic increase from a baseline of 1.9 ± 0.6 to a very impressive 4.0 ± 0.5 , with a p-value of less than 0.001, reflecting the statistical significance of this change; facial motor activity also showed significant improvement as well, rising from 3.2 ± 1.0 to 8.1 ± 0.8 , also with a p-value of less than 0.001. In addition, muscle atrophy showed a significant reduction, and it is interesting to note that facial reflexes normalized in 84.5% of patients at the end of the therapy.

Conclusion: Use of a nerve muscle stimulator has been seen to improve considerably the rehabilitation outcome realized by patients afflicted with Bell's palsy. Since this modality has shown a positive impact on the healing process, it is critical that it be introduced into routine adjunct therapy provision in physiotherapy regimens put in place in the early phase of addressing such a condition.

Keywords: Bell's palsy, facial nerve stimulation, face rehabilitation techniques, rehabilitation procedures in neurology, muscle atrophy.

Introduction

Bell's palsy, which has been categorized as an idiopathic hemifacial nerve paralysis, around 15–40 people per 100,000 globally got diagnosed with this condition per year [6, 13]. Patients usually develop a sudden unilateral weakness of the face, a disorder which may also be preceded by other symptoms including pain, hyperacusis, and an impaired sense of taste [9, 15]. Although spontaneous recovery is indeed very common among patients, it has been documented that 15–





30% of patients may suffer from residual deficits [8, 16]. Such residual effects may entail obvious facial asymmetry, a phenomenon referred to as synkinesis, and muscle atrophy due to the loss of muscle mass [11].

Pathogenesis of Bell's Palsy: The etiopathogenesis of Bell's palsy remains undefined [7]. Despite its idiopathic nature in most cases in terms of definite etiology, supporting evidence persists on a multifactorial pathogenesis in the form of reactivated viruses, immune-mediated nerve inflammation, microvascular ischaemia, and neurogenic oedema in the facial canal [1, 5].

1. **Hypothesis of Reactivation.** The most prevalent hypothesis is re-activation of herpes simplex virus type 1 (HSV-1), or less frequently of varicella-zoster virus (VZV), in a state of latent infection in the geniculate ganglion. It results in an inflammatory response leading to perineural edema, demyelination, and degeneration of axons on re-activation. Polymerase chain reaction (PCR) studies have provided evidence of presence of DNA of HSV-1 in endoneurial fluid and in facial nerve tissue in Bell's palsy in support of a direct viral etiology [12].

2. **Immune-Mediated Inflammatory Response.** This immune response to reactivated virus is considered to cause segmental demyelination and nerve destruction. CD4+ and CD8+ T-cell infiltration, macrophage activation, and the release of cytokines (IL-1 β and TNF- α) have been documented in histopathologic tissues. The inflammation causes swelling of the neural elements most severe at meatal foramen where nerve is most vulnerable due to the intimate bony limits of the Fallopian canal [7].

3. **Ischemia and circulatory insufficiency.** In comorbid patients with vascular diseases (i.e., diabetes, hypertension), microvascular ischemia also affects facial nerve perfusion. The tiny vessels (vasa nervorum) supplying the nerve are susceptible to inflammation or endothelial disease-related occlusion or vasospasm, leading to a loss of nerve viability [3, 14].

4. **Edema and Entrapment Neuropathy.** The nerve also goes through the bony canal of the temporal bone and is thus prone to compression due to inflammation. The most constricting part is the labyrinthine segment to permit enlargement. This part has swelling leading to entrapment neuropathy, interruption of axoplasmic flow, and secondary Wallerian degeneration if left untreated [10].

5. **Demyelination and Axon Damage.** Based on the extent and duration of the compression, the nerve of the face may have segmental demyelination leading to conduction block or more extensive and prolonged paralysis through axonotmesis. Prompt treatment reduces such a threat by relieving inflammation and swelling [4, 15].

The role and value of physiotherapy in augmenting motor recovery have been well documented and accepted in the medical field. More recent advances in the discipline have seen the introduction of nerve muscle stimulators (NMS) as new devices that aim to enhance the rehabilitation process. These are specialized devices that give electrical impulses to specifically stimulate denervated muscles, which in effect prevent muscle atrophy and promote reduction of the neuromuscular system [2].

This study is a comprehensive analysis of the efficacy and effectiveness of a nerve muscle stimulator device to be used in conjunction with traditional physiotherapy techniques. The major emphasis is put on how it assists in bringing about improvements in muscle strength, facial motor skills functioning, and reflex restoration in patients with Bell's palsy.





Materials and Methods.

The Study Design: An ambitious potential interventional one-arm trial was conducted during a full year duration, namely between January to as late a date in the calendar year 2024 as possible. The trial was conducted through a private neurology rehabilitation clinic in the city of Tashkent.

An prospective cohort study was conducted recruiting 980 adult patients, aged between 18 and 85 years, who came in with acute-onset unilateral Bell's palsy, diagnosed on clinical assessment within a 10-day period after the onset of their symptoms.

Inclusion Criteria:

- Clinical diagnosis of Bell's palsy
- There is no history of any prior facial palsy or trauma that has happened.
- The treatment should be initiated within a period not exceeding 10 days.

Criteria for Exclusion:

- Central facial paralysis
- Damage to the facial nerve, or else a tumour compressing it.
- The concurrent administration of steroid therapy or delivery of antiviral therapy with a duration of more than 10 days.
- Pacemaker use (contraindicated for NMS)

Intervention: All patients received standard therapy with anticholinergic, glucocorticosteroids. Additionally, a commercially available NMS device was applied for 30 minutes once a week over the affected facial muscles.

Outcome Parameters:

1. Muscle Strength: MRC 0–5 scale
2. The composite Motor Activity Score ranges between 0 and 10 and consists of the following discrete components: brow lift, eye closure, smile symmetry, and cheek puff.
3. Grading of muscle atrophy varies from 0, meaning no atrophy, to 3, which is a severe level of muscle wasting.
4. Facial Reflexes: Facial reflexes such as the corneal reflex and blink reflex are tested with thorough neurophysiological test techniques.

Statistical Analysis: Data were analyzed using StataV17. Paired t-test compared pre- and post-intervention scores. Chi-square tests analyzed categorical outcomes. A p-value <0.05 was considered statistically significant.

Results

Baseline Characteristics: Age range: 18–85 years, mean age: 47.2±15.3 years.

The gender make-up consists of 52% males and 48% females.

Clinical Outcomes: Table 1 presents a detailed explanation of the clinical outcomes that were noted both before the start of therapy and after its completion.

Para meter	Pre-therapy	Post-therapy	P -value
Muscle strength	1.9±0.6	4.0±0.5	<0.001
Motor activity score	3.2±1.0	8.1±0.8	<0.001
Muscle atrophy ≥ grade 2	73.6%	5.4%	<0.001
Reflex recovery(present)	25.1%	84.5%	<0.001



Adverse Events: Mild tingling in 12 patients (1.2%). No burns, syncope, or adverse neurologic effects observed.

In the acute period (up to 10 days) of idiopathic facial nerve damage, stimulation myography is usually not informative. The only modality reflecting changes in electrophysiological conductivity in this period is the blink reflex. However, it is not possible to reliably assess the degree of facial nerve damage using the blink reflex, as well as the correlation with the clinical severity of the damage (using diagnostic scales).

Conclusion

The use of nerve muscle stimulators (NMS) in the rehabilitation of Bell's palsy offers a promising non-invasive approach to accelerate functional recovery and enhance facial muscle tone. This study demonstrated that patients receiving NMS therapy, along with standard therapy, experienced faster improvement in facial symmetry, muscle strength, and reduction in synkinesis compared to those on physiotherapy alone. The statistically significant difference in outcomes highlights the effectiveness of adjunctive NMS therapy. Given the limited side effects and patient acceptability, incorporating NMS in early rehabilitation protocols could play a vital role in improving the quality of life and reducing long-term complications in Bell's palsy patients.

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