Description
Bell’s Palsy, or idiopathic facial paralysis, is a temporary facial paralysis that results from damage or trauma to the facial nerve (cranial nerve VII). This nerve travels through a narrow bony canal in the skull beneath the ear to each side of the face. The facial nerve controls the muscles on one side of the face as well as impulses to the tear glands, saliva glands, and the stapes in the middle ear. The nerve also transmits sensory signals for taste from the tongue.

Pathology, Pathophysiology
The etiology of Bell’s Palsy has not been definitively identified, but viral infection, vascular ischemia, or autoimmune diseases have been postulated as possible causes. In general, Bell’s Palsy occurs when function of the facial nerve is disrupted by swelling, inflammation, or compression. One of the most prevalent theories as to the cause of Bell’s Palsy is that a viral infection such as viral meningitis or herpes simplex (the common cold sore virus) causes the facial nerve to swell and become inflamed in reaction to the infection. It is suspected that this swelling can cause the nerve to become pinched in the bony canal and lead to death of nerve cells due to insufficient blood or oxygen supply. In some mild cases, there is only damage to the myelin sheath of the nerve.

Bell’s Palsy is reported to afflict 40,000 Americans each year. It is disproportionately found to affect pregnant women, people with diabetes, and people with upper respiratory ailments.

Presentation
The symptoms of Bell’s Palsy usually begin suddenly and reach their peak within 48 hours. Symptoms range in severity from mild weakness to total paralysis. These may include twitching, weakness or paralysis, drooping eyelid or corner of the mouth, drooling, dry eye or mouth, ringing in one or both ears, dizziness, pain or discomfort around the jaw, hypersensitivity to sound on the affected side, impairment of taste, and excessive tearing.

In patients for whom axonal damage has occurred, axonal regeneration is possible. One possible complication during this process is synkinesis which can occur when the facial nerve is recovering and some new nerves branches accidentally re-grow in close proximity to muscles they don’t normally innervate. This can result in unwanted involuntary movements of some muscles that accompany purposeful movements of other muscles. One example of this is the eye closing during purposeful mouth movement. The incidence of synkinesis ranges from 9-55% of patients recovering from Bell’s palsy. Research has not found a definite cause of what contributes to the development of synkinesis in some patients but not others.
Clinically facial nerve injury is most commonly classified using the House-Brackmann system. Grade 1 is normal function. Grade 2 is slight weakness but no synkinesis (involuntary movement accompanying a voluntary movement). Grade 3 is obvious weakness and some mass movement. Grade 4 is inability to elevate the brow, significant synkinesis, and obvious weakness. Grade 5 is barely perceptible motion, and grade 6 is no movement.

According to Peitersen, during an observational study of the natural course of Bell’s Palsy in 1,700 patients, 64% of patients will have “regained normal function” in 3 months without intervention. Longer term, this study found that 71% of people recovered normal function of the face, 13% had insignificant symptoms, and the remaining 16% had permanently diminished function, with contracture and synkinesis. The earlier the recovery starts, the better the prognosis for full recovery.

Management
There is no standard course of treatment or cure for Bell’s Palsy. Some cases are mild and do not require treatment since the symptoms usually resolve within 2 weeks with or without treatment.

Medical treatment often involves medications such as acyclovir to fight the viral infection as well as an anti-inflammatory drug such as the steroid prednisone to reduce injury due to swelling of the nerve. Since Bell’s Palsy can interrupt eye blinking, patients must keep the eye moist to prevent damage; typically lubricating eye drops and eye patches are used.

There is debate over the effectiveness of intervention for the facial paralysis following Bell’s Palsy. Treatments include acupuncture, exercise, and electrical-stimulation (e-stim). However, the research is inconsistent in demonstrating the effectiveness of these interventions. Peitersen points out that evaluation of different treatments for Bell’s Palsy is difficult because of the high rate of spontaneous recovery regardless of treatment.

Another treatment used for residual paralysis following Bell’s Palsy is neuromuscular retraining (NMR). NMR uses “selective motor training to facilitate symmetrical movement and control undesired gross motor activity (synkinesis)” Modalities such as surface EMG biofeedback and mirror exercises provide sensory information to assist with recovery. The Bells Palsy Information Site webpage offers information about the how-to’s of NMR for clinicians. However, as with other interventions, the research is still inconclusive as to the benefits of this treatment.

Role of NMES
Neuromuscular Electrical Stimulation (NMES) can only be used with patients diagnosed with Bell’s Palsy when the nerve has sufficiently healed and can respond to stimulation. NMES functions by stimulating intact peripheral nerves and is not indicated for patients with denervated muscles. Once the nerve is intact, the use of NMES may be beneficial in improving residual weakness resulting from Bell’s Palsy, although as with other treatments for Bell’s Palsy, the research is inconclusive. If NMES is attempted with a patient who has
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Bell’s Palsy and no muscle contraction is observed when the stim is applied, then the nerve is likely not sufficiently healed and the treatment is not recommended.

It is sometimes stated that e-stim can cause synkinesis in a patient recovering from Bell’s Palsy. However, given the high incidence of synkinesis regardless of the intervention, no one treatment has been shown to cause or prevent synkinesis. In cases in which synkinesis occurs, it is currently not possible to determine if any treatment component may have contributed to its development. Some case studies have suggested that the use of electrotherapy may have contributed to the onset of synkinesis. However during these studies many different interventions were used in addition to e-stim, making it impossible to draw reliable conclusions about the impact of e-stim. Additionally, in these case studies direct current was used, which is significantly different from the pulsed current delivered by the VitalStim Therapy device. Further research is needed to determine what role different external factors may play in the development of synkinesis.

Literature review

  Teixeira et al completed a review of the published research about the use of electrostimulation (e-stim) or exercise for patients with Bell’s palsy. In this review, one study found that e-stim appeared to speed recovery (Flores 1998) and one study found that the group treated with e-stim had worse quality of movement than a control group (Manikandan 2007). This review concluded that there is insufficient evidence of significant benefit or harm from e-stim or exercise.

  Ohtake et al also completed a review of the available literature and found 2 studies that showed positive associations between e-stim and clinical outcomes (Farragher 1987 and Targen 2000). However, since there were no control groups in these studies they did not feel that the outcomes could be contributed to e-stim and could have likely been due to “the natural tendency for spontaneous recovery from Bell palsy”.

- Cardoso JR, Teixeira EC, Moreira MD, Favero FM, Fontes SV, Bulle de Oliveira AS. Effects of exercises on Bell’s palsy: systematic review of randomized control trials. Otol Neurotol. Jun 29 2008(4): 557-60. OBJECTIVE: This study examined the effects of facial exercises associated either with mirror or electromyogram (EMG) biofeedback with respect to complications of delayed recovery in Bell’s palsy. RESULTS: Four studies of 132 met the eligibility criteria. The studies described mime therapy versus control (n = 50), mirror biofeedback exercise versus control (n = 27), ”small” mirror movements versus conventional neuromuscular retraining (n = 10), and EMG biofeedback + mirror training versus mirror training alone. The
treatment length varied from 1 to 12 months. CONCLUSION: Because of the small number of randomized controlled trials, it was not possible to analyze if the exercises, associated either with mirror or EMG biofeedback, were effective. In summary, the available evidence from randomized controlled trials is not yet strong enough to become integrated into clinical practice.

In conclusion, additional research is needed to conclusively determine when, how, and if NMES is of benefit to patients recovering from Bell’s Palsy. Given the inconclusive findings thus far in the research, treatment should involve educating the patient so that he or she can make an informed decision about the potential use of this modality in therapy.

References


