

Review Article

Review on Management of Bell's Palsy- An Update

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Abstract:

Bell's palsy is generally a unilateral disease, affecting both sides of the face equally. Treatment of Bell's palsy is controversial, because as many as two-thirds of patients recover spontaneously. Corticosteroids alone or associated with antiviral agents have been recommended. In some cases, physiotherapy may provide extreme benefit in reducing the physical and social impairments commonly observed in patients suffering from Bell's palsy. This review aims at enlightening the treatment strategies which include medications, surgeries and exercises for bell's palsy.

Keywords: Bell's palsy, Electrodiagnostic tests, Surgical treatment, Physiotherapy treatment

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Introduction

Bell's palsy is a neuropathy of the peripheral seventh cranial nerve, usually resulting from traumatic, compressive, infective, inflammatory or metabolic abnormalities. However, in many cases no etiology is identified and the eventual diagnosis is idiopathic.¹ Bell's palsy can be defined as acute peripheral facial nerve palsy usually of unknown cause.² The condition is named after Dr. Charles Bell, who, in 1821, described complete facial paralysis after injury of the stylomastoid foramen.³

Pathophysiology

Bell's palsy induces a wide range of facial muscle movement dysfunction from mild paresis to total paralysis. Individual patients display a spectrum of symptoms: some maintain reduced movement throughout the course of the disorder while others rapidly become totally paralyzed over a 24-hour period. The pathophysiology of the neural injury is suspected to be due to edema

within the nerve induced by a viral infection.⁴

Acute inflammation and edema of the facial nerve are thought to lead to entrapment of the nerve in the bony canal (especially in the labyrinthine segment), which leads to compression and ischemia.⁵ Many viruses, such as HIV,⁶ Epstein-Barr virus⁷ and hepatitis B⁸ virus have been suspected in initiating this inflammation, but herpes simplex virus (HSV) is the most. According to one hypothesis, HSV, dormant in the geniculate ganglion cells, becomes reactivated and replicates, causing inflammation, primarily in the geniculate ganglion and in the labyrinthine segment of the facial nerve.

These inflammatory events (evident on magnetic resonance imaging) result in entrapment and ischemia, which lead to neurapraxia or degeneration of the facial nerve distal to the meatal foramen.⁵

Table: Causes of Bell's palsy ⁹

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| Birth-related | Forceps delivery, Dystrophia myotonica, Möbius syndrome |
| Trauma | Basal skull fracture, Facial injuries, Scuba diving (barotrauma) |
| Neurological | Guillian-Barré syndrome, Multiple sclerosis, Myasthenia gravis |
| Infection | External otitis, Otitis media, Mastoiditis, Lyme disease, <i>Herpes zoster cephalicus</i> , Encephalitis, Poliomyelitis, Mumps, Mononucleosis, Tuberculosis, Acquired immunodeficiency syndrome (AIDS) |
| Metabolic | Diabetes mellitus, Pregnancy, Hypertension |
| Neoplastic | Cholesteatoma, Seventh nerve tumour, Glomus jugulare tumour, Meningioma, Sarcoma, Schwannoma, Malignant parotid lesions, Paramalignant phenomenon |
| Iatrogenic | Parotid surgery, Temporal bone surgery, Embolisation |
| Idiopathic | Bell's palsy |
| Other | Melkersson-Rosenthal syndrome, Amyloidosis, Sarcoidosis |

Diagnosis

Several electrodiagnostic strategies have been devised to evaluate the neural damage following acute facial nerve paralysis. These tests attempt to measure the amount of neural degeneration that has occurred distal to the sight of injury by measuring the muscle response to an electrically evoked stimulus. Electrodiagnostic tests can be used to differentiate nerve fibers that have minor conduction block problems (neuropraxia) from those that have undergone Wallerian degeneration, but these tests cannot differentiate the type of Wallerian degeneration, axonotmesis type injury from neurotmesis. The rate of progression of Wallerian degeneration after injury can be measured and provides some information about the severity of the degeneration. More rapid Wallerian degeneration is associated with neurotmesis, while nerves that degenerate more slowly are more likely to exhibit axonotmesis.⁴ Indicators for poor prognosis include complete facial palsy, no recovery of symptoms by three weeks, age

over 60 years, severe pain, herpes zoster virus, co-morbid status e.g. hypertension, diabetes, pregnancy and severe degeneration of the facial nerve shown by electrophysiological testing.^{10,11}

NCV test may be done to check for the nerve damage. NCV stands for nerve conduction velocity. MRIs and CAT scans of the brain may be done to make sure that the problem is not caused by a stroke, a tumor, or an infection. If an MRI or CAT scan reveals a stroke, a tumor, or an infection, it is not likely that the 7th nerve will improve. The doctor can usually detect other signs of these symptoms during the examination.¹²

Treatment

Treatment of Bell's palsy is controversial, because as many as two-thirds of patients recover spontaneously. Corticosteroids alone or associated with antiviral agents have been recommended. Adour¹³ reported that patients with Bell's palsy treated with acyclovir and prednisone experience a

more favourable recovery and less neural degeneration than patients treated with placebo plus prednisone. The favourable response to the treatment of Bell's palsy with acyclovir–prednisone supports the theory that reactivated HSV causes a neuritis.⁵

Bell's palsy and physiotherapy treatment:

The literature review yielded recommended guidelines described by Brach and VanSwearingen (1999). The authors describe four distinct treatment based categories (initiation, facilitation, movement control and relaxation stages) matched with special treatment techniques for each category. The exercises consist of actively assisting special facial movements. Initially the patient is instructed on using his fingers to passively move the left corner of his mouth into a 'smiling' posture and then slowly release his finger pressure. In addition to the 'smiling' exercise, patient is instructed to passively raise his left eyebrow with his finger and activate the appropriate musculature upon release of his passive support finger. Then the patient is instructed to focus both eyes on an object (e.g. the tip of a finger) positioned down and in front of the patient. After that the resistive exercises are introduced and are aimed at facilitating the affected-sided musculature. Resistance is provided using fingers to the desired facial movements e.g. smiling, 'puckering' and raising the eyebrow on the affected side. Careful attention is given to avoid muscular fatigue of the involved side and overfacilitation of the uninvolved side. The patient is usually instructed to increase the number of repetitions to 20 and to perform the exercises (1-2 times per day) once in the morning and once before going to bed at night. The continuity of these exercises is required to prevent further problems related to symptoms of facial paralysis.¹¹

Surgical treatment

Decompression surgery for Bell's palsy started in the 1930s (Balance and Duel

1932). Technically it is difficult to reach the area of compression in the labyrinthine-meatal segments and furthermore there is risk for complications such as CSF leakage, infection, hearing loss, dizziness and intracranial haemorrhage. Fisch and Esslen stress the importance of decompressing the facial nerve through a middle cranial fossa approach to be able to decompress the labyrinthine, geniculate and tympanic segment of the nerve, and state that this could improve outcome (Fisch and Esslen 1972). May showed no significantly improved recovery in patients treated surgically using a transmastoid approach (May et al 1985).⁹ Decompression of the facial nerve can be accomplished by a delicate microsurgical procedure. This surgery can make a critical difference with some types of severe nerve damage, but not generally for Bell's Palsy. For Bells Palsy it remains highly controversial, even when nerve degeneration is severe. There is not likely to be any benefit over prompt treatment by standard meds, and there are serious risks involved. The most common complications are hearing loss and damage to the facial nerve, which can be permanent. If this procedure is done for any reason, it should be done within 3 weeks of nerve damage. After this time statistics show no benefit for enduring the surgery and the potential risks.

Cosmetic surgeries such as brow-lifts, face-lifts, muscle shortening, removal of excess upper eyelid skin, muscle relaxing procedures and static slings are available to improve appearance, but they will not improve muscle function. Nerve and muscle grafts or transpositions can offer functional improvement as well as improve appearance. These are complex procedures that should be considered carefully. Take care to insist that the surgeon fully explains the procedure, recovery, and risks. Risks include nerve damage that can leave the patient with worse paralysis than prior to surgery.

One type of nerve transposition involves connecting the hypoglossal nerve (controls

the tongue) to the facial nerve. After surgery, the patient learns how to move the face by moving the tongue. Ideally, the motion becomes automatic in time. There is likely to be a loss of sensation at the tongue. A muscle transposition can be performed using a muscle that isn't controlled by the facial nerve. The temporalis or masseter muscle can be connected to the corner of the mouth. The intention is that the enervated muscle will increase motion in the muscles around the mouth.

In a combination muscle and nerve graft, two procedures are performed several months apart. Free muscle tissue is grafted from the leg to the face following a cross-facial nerve graft. The nerve graft becomes the nerve supply for the healthy, transplanted muscle.¹⁴

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