

PERIPHERAL FACIAL WEAKNESS (BELL'S PALSY)

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SUMMARY – Peripheral facial weakness is a facial nerve damage that results in muscle weakness on one side of the face. It may be idiopathic (Bell's palsy) or may have a detectable cause. Almost 80% of peripheral facial weakness cases are primary and the rest of them are secondary. The most frequent causes of secondary peripheral facial weakness are systemic viral infections, trauma, surgery, diabetes, local infections, tumor, immune disorders, drugs, degenerative diseases of the central nervous system, etc. The diagnosis relies upon the presence of typical signs and symptoms, blood chemistry tests, cerebrospinal fluid investigations, nerve conduction studies and neuroimaging methods (cerebral MRI, x-ray of the skull and mastoid). Treatment of secondary peripheral facial weakness is based on therapy for the underlying disorder, unlike the treatment of Bell's palsy that is controversial due to the lack of large, randomized, controlled, prospective studies. There are some indications that steroids or antiviral agents are beneficial but there are also studies that show no beneficial effect. Additional treatments include eye protection, physiotherapy, acupuncture, botulinum toxin, or surgery. Bell's palsy has a benign prognosis with complete recovery in about 80% of patients, 15% experience some mode of permanent nerve damage and severe consequences remain in 5% of patients.

Key words: Bell palsy – diagnosis; Bell palsy – therapy; Acupuncture

Introduction

Peripheral facial weakness is a facial nerve damage that results in muscle weakness on one side of the face. It may have a detectable cause (secondary) or may be idiopathic without cause or without an obvious cause (primary, Bell's palsy)¹. Secondary peripheral facial weakness can be caused by metabolic diseases (diabetes, preeclampsia^{2,3}), stroke, infections (both viral and bacterial, e.g., herpes simplex infection, varicella zoster infection, influenza viruses, borreliosis), autoimmune diseases⁴ (Miller Fisher syndrome, Guillain-Barré syndrome, sarcoidosis), surgery⁵, trauma, tumors⁶, drugs⁷, and other causes⁸. Bell's palsy or idiopathic

peripheral facial weakness may only be diagnosed by exclusion of detectable causes. Due to the lack of sufficiently powered studies, therapy of primary and secondary facial weakness is controversially discussed, particularly if the causes of a secondary facial weakness coexist with Bell's palsy or if multiple causes of a primary facial weakness coexist in case of a secondary facial nerve palsy⁸.

The incidence of Bell's palsy is estimated to 20–25 cases *per* 100,000 population annually¹. Bell's palsy arises more frequently in the spring and fall than in any other time of the year. Women and men are usually equally affected⁸. Definitely, the prevalence is increased in pregnant women (43 cases *per* 100,000)⁸. The peak incidence occurs between the second and fourth decade (15–45 years)^{1,8}. It occurs with equal frequency on the right and left side of the face. Simultaneous, bilateral facial weakness is extremely rare with a prevalence of 0.3%–2% of facial palsies⁹.

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Etiology of Bell's Palsy

The etiology of Bell's palsy is unknown but viral infection, vascular ischemia, and autoimmune disease have been postulated as the possible pathomechanisms^{1,8}. Bell's palsy disproportionately occurs in pregnant women, patients who have diabetes, influenza, cold, some other respiratory infection, or have undergone tooth root extraction. Some patients report exposure to an air-condition outlet, or an open window before the attack¹⁰. Family manifestation has also been reported^{2,11}. Increasing evidence implies that Bell's palsy is caused by latent herpes viruses (herpes simplex, herpes zoster), being reactivated from cranial nerve ganglia¹². Reactivation of these viruses presumably causes inflammation of the facial nerve¹¹. Initially, inflammation of the nerve results in reversible neurapraxia, and ultimately in wallerian degeneration^{8,12}. Virus infection with herpes simplex type 1 or herpes zoster may predominantly occur if the immune system is simultaneously compromised. Herpes viruses have been detected by polymerase chain reaction (PCR) within the facial nerve¹³. There are confusing results concerning the role of *Borrelia burgdorferi* in the occurrence of Bell's palsy. Some studies found an increased prevalence of *Borrelia* antibodies among patients with Bell's palsy, whereas others could not confirm these results⁸.

Secondary peripheral facial weakness is caused by a number of different causes. Although it is often difficult to decide if any of these causes is responsible for the clinical picture, it is important to distinguish primary from the secondary form, which may have a significant influence on therapy and prognosis³. Among 180 patients, Bell's palsy was associated with arterial hypertension in 12%, diabetes in 11%, pregnancy or puerperium in 4% and neurocysticercosis in 1%³. In another study, facial weakness was most frequently associated with viral infections, borreliosis, or diabetes¹⁴. However, if Bell's palsy occurs simultaneously with a disorder, which also may cause secondary peripheral facial weakness, this does not necessarily imply a causal relation.

Pathophysiology

The precise cause of Bell's palsy is unknown. A theory proposes that swelling of the facial nerve trunk in the narrow confines of the facial canal leads to local compressive ischemia, which in turn leads to further swell-

ing and edema. The result is local interruption of blood supply to the facial nerve and thus extension of the ischemic injury. This compression of facial nerve has been seen in magnetic resonance imaging (MRI) scans¹⁵.

Clinical Presentation

Bell's palsy is acute palsy of the facial nerve that results in muscle weakness usually on one side of the face. The clinical picture may differ, depending on the location of the lesion of the facial nerve along its course to the muscles. Symptoms and signs may also differ because of the fact that the facial nerve carries not only motor fibers including fibers to the stapedius muscle but also supplies autonomic innervation of the lacrimal gland, submandibular gland, sensation to part of the ear, and taste to the anterior two thirds of the tongue *via* chorda tympani^{8,12}. Thus, Bell's palsy is diagnosed upon abrupt onset of impaired facial expression due to unilateral facial weakness of all facial nerve branches, dry eye, if saliva runs out of the mouth, the inability to close or wink the eye or close the mouth, to droop the brow or the corner of the mouth, numbness or pain around the ear, temple, mastoid, or angle of the mandible, an altered sense of taste, hypersensitivity to sounds, or decreased tearing¹⁶. Patients may also mention otalgia, aural fullness, or mild retroauricular pain, which may even precede the palsy⁸. Speech and eating may also be disturbed. Severe pain suggests herpes simplex or zoster infection and may precede vesicular eruption and progression to Ramsay Hunt syndrome, characterized by typical cutaneous vesicles and vesicles in the conchal bowl, soft palate, or tongue and by vestibulo-cochlear dysfunction^{8,12}. In almost half of the herpes zoster infections, vesiculation not necessarily appears or may be delayed (zoster sine herpette). Sometimes, the only clinical indication of herpes zoster is dysesthesia before vesiculation (described as preherpetic neuralgia). It is important to consider zoster sine herpette, since it is thought to be the cause of Bell's palsy in quite a number of cases¹².

Evaluation of the Severity of Peripheral Facial Weakness

To evaluate the severity of peripheral facial weakness clinically, various scoring systems are available. The most widely applied is the House-Brackmann fa-

cial nerve grading system (HBS). The degree of facial nerve palsy can also be evaluated by the Yanagihara grading system¹⁷, the Sunnybrook scales, the Jadad score of methodological quality, scales on computed systems, and various other systems¹⁸. Most grading systems rely on the evaluation of resting symmetry, degree of voluntary excursion of the facial muscles, and the degree of synkinesis (involuntary movement accompanying a voluntary movement) triggered by specific voluntary movements⁸. Facial weakness can be categorized as incomplete (partial) or complete (if there is inability to voluntarily contract the facial muscles, hyperacusis, or loss of taste)¹⁹. The progression of weakness may be additionally assessed by reviewing some old photos and comparing them with the actual status. The degree of nerve damage can also be evaluated by nerve conduction studies of the facial nerve. Reduction of the compound muscle action potential refers to the axonal degeneration, whereas increase in latency suggests demyelination of the nerve²⁰.

Diabetes and Peripheral Facial Weakness

There are indications that the facial nerve is subclinically involved in 6% of the patients with diabetes. Facial nerve affection, however, is less frequent than limb nerve affection. In one study, the rate of diabetes was 10% among 38 outpatients with Bell's palsy. This figure did not differ from the anticipated frequency of diabetes in the general population²¹, raising doubt whether diabetic mononeuropathy of the facial nerve really exists. Neither the severity of facial nerve degeneration, as assessed by facial nerve conduction studies, nor the clinical outcome was significantly different between diabetic and nondiabetic patients in a prospective study on 37 patients with Bell's palsy²².

Diagnosis

The diagnosis of peripheral facial weakness is based on clinical presentation with weakness of all facial nerve branches, drooping of the brow, incomplete eyelid closure, impaired closure of the mouth, drooping of the corner of the mouth, dry eye, hyperacusis, and impaired taste or pain around the ear. If eye closure is incomplete, Bell's phenomenon occurs (upward diversion of the bulb on attempted closure of the eyelid)⁸. Transcranial magnetic stimulation seems

capable of localizing the site of the lesion within the fallopian channel²³. Nerve conduction studies showing prolonged distal latency and reduced compound muscle action potential may provide useful information on the severity and nature of the lesion⁸, although more prospective studies are required to assess the validity of nerve conduction studies for the prognosis of facial nerve lesions. Assessment of the ear should include pneumatic otoscopy, tuning fork tests, otomicroscopy, and audiometry. Additional tests may include electronystagmography, videonystagmography, and videooculography. The stapedius reflex may be reduced or absent⁸. PCRs are basically used to demonstrate the presence of herpes viruses and antibody tests are necessary to demonstrate the presence of *Borrelia burgdorferi*. Cerebrospinal fluid investigations may show pleocytosis, increased or decreased glucose, increased protein, antibodies against *Borrelia burgdorferi*, or various viruses or virus DNA or RNA⁸. The main task of all investigations is to find or to exclude the cause of peripheral facial weakness.

Therapy

The main goals of treatment are to speed recovery, to make recovery more complete, to prevent corneal complications and other consequences, and if there is a viral infection, to inhibit viral replication¹². Psychological support is also very important. Therapy of secondary facial weakness aims to act upon the particular cause of the palsy. Patients with Bell's palsy should be referred to a specialist and treatment should start as soon as possible¹². Treatment may be divided into pharmacological therapy and nonpharmacological measures.

Pharmacological therapy

1) Steroids

Although steroids are often used in Bell's palsy, their efficacy in this indication has not been clearly shown. Most of the studies on steroids in Bell's palsy have a small sample size and a retrospective, observational design; rely on chart reviews, and lack randomization, control group, or blinding. On the one hand, there are studies, which clearly showed a beneficial effect of steroids in the treatment of Bell's palsy, but there also are studies, which did not⁸.

In one randomized, double-blind, placebo-controlled, factorial study on 496 patients with Bell's palsy, after 3 months 83% in the corticosteroid group recovered facial functions compared to 64% in the placebo group²⁴. After 9 months of follow up, this proportion increased to 94% in the corticosteroid group and 82% in the placebo group. The conclusion was that treatment with steroids within 3 days after onset significantly improved the chance for complete recovery at 3 or 9 months²⁴. In another study on 62 patients with Bell's palsy, high-dose intravenous prednisone given together with vitamins within 72 hours after onset resulted in better outcome as compared with controls, who received only vitamins²⁵. In a study on 71 patients with Bell's palsy, the administration of intravenous high-dose hydrocortisone together with low molecular dextran resulted in better outcome and less side effects in HBS I-II patients as compared to patients who received prednisone alone²⁶. In a study on 76 patients, simultaneous administration of methylprednisolone and acyclovir resulted in reversion of the deficits to HBS I or II in 92% of cases²⁷. All patients in HBS grade I-II recovered completely. For patients in HBS grade IV, V, and VI, complete recovery after 1 year was observed in only 94%, 86% and 50%, respectively²⁷. In a randomized study on 46 patients with Bell's palsy, of whom 23 received acyclovir and prednisone and 23 prednisone alone, those receiving combination therapy had better outcome on the facial nerve function index, as compared to those who were on steroids alone²⁷. If steroids were combined with valacyclovir, complete recovery was observed in 88% of 56 patients with Bell's palsy, whereas complete recovery was observed in only 68% of the patients who did not receive any therapy at all. Most studies recommend steroids for moderate to severe Bell's palsy within the first 72 hours after onset and for the one-fifth of patients in whom the palsy progresses⁸. A side effect of steroids for Bell's palsy is frequent temporary aggravation of pre-existing diabetes. This is the reason why patients with uncontrolled diabetes should receive corticosteroids only under close monitoring of blood glucose²⁴. According to two Cochrane reviews, there is no benefit of steroids alone or in combination^{28,29}. These reviews also admit that the available studies were insufficiently powered to detect treatment effect. From three randomized trials, one with steroids *versus*

placebo, one with steroids and vitamins *versus* vitamins, and one with steroids without a placebo group, including a total of 117 patients, the authors conclude that more randomized trials with a larger number of patients are needed to determine if there is potential harm or benefit from using steroids in Bell's palsy²⁹. In a study on 56 patients with Bell's palsy, steroids did not result in significant improvement of the lesions 3 and 6 weeks after onset³⁰. In a study on 221 patients with Bell's palsy, valacyclovir and corticosteroids were significantly better than corticosteroids alone³¹.

2) Antiviral agents

Antiviral agents for Bell's palsy are rarely given. Two Cochrane reviews on 246 and 200 patients, including three²⁸ and two randomized trials³² with acyclovir and steroids *versus* steroids alone, acyclovir *versus* steroids³³, and valacyclovir with steroids *versus* steroids³⁴ conclude that the results of all three trials were inconclusive with regard to short- or long-term benefit and that a large, multicenter, randomized, controlled, and blinded study with a minimum follow up of 1 year is required before definite recommendation regarding the effect of acyclovir or valacyclovir can be given^{8,34}. At least there does not seem to be a difference between oral acyclovir and steroids *versus* intravenous acyclovir and steroids^{8,14}. A study on 221 patients with Bell's palsy treated with valacyclovir and prednisolone within 7 days after onset showed better outcome for patients receiving combination therapy than corticosteroids alone³¹. In a study on 247 patients receiving acyclovir, complete recovery was observed in 71% after 3 months and in 85% after 9 months²⁴. The authors found no benefit of acyclovir alone or additional benefit of acyclovir in combination with corticosteroids²⁴. For patients with zoster sine herpette, however, acyclovir appears to be effective^{8,12}.

3) Pentoxifylline

The efficacy of pentoxifylline on the recovery of Bell's palsy has only been tested together with other drugs, particularly steroids and low-molecular dextran. Studies showed a beneficial effect of such a combination therapy, but which of these drugs is the one actually responsible for the beneficial effect, is so far unknown³⁵.

Nonpharmacological treatment

1) Mime and physiotherapy

Only a few controlled trials on the effectiveness of physical therapy for facial palsies are available¹². In a randomized trial on 50 patients with Bell's palsy, mime therapy, including automassage, relaxation exercises, inhibition of synkinesis, coordination exercises, or emotional expression exercises, resulted in improvement of facial stiffness, lip motility, and the physical and social indices of the facial disability index³⁶. Patients with remaining symptoms of Bell's palsy appear to experience positive effects from physiotherapy³⁷ and biofeedback training³⁸. In a controlled study on 24 patients with Bell's palsy, neuromuscular retraining exercises showed an effect in improving facial movements³⁹.

2) Acupuncture and moxibustion

Acupuncture is safe therapy with a low risk of adverse events in clinical practice⁴⁰. It is one of the most commonly used treatments for Bell's palsy in China. Although only limited experience has been reported with acupuncture for Bell's palsy¹², several studies provide increasing evidence for a beneficial effect of acupuncture and moxibustion as an adjunctive treatment for Bell's palsy^{41,42}.

Local treatment

In the acute phase, local treatment is very important because eye care is imperative in Bell's palsy. The patient's eye is at risk of drying, corneal abrasion, and corneal ulcers.

1) Eye protection

One of the greatest problems with Bell's palsy is drying of the eye if the lid fissure remains open. Eye care should be focused on the protection of the cornea from dehydration, drying, or abrasions due to insufficient lid closure or tearing¹². Eye ointment and wetting is proposed during day and night supported by a watch-glass bandage during the day or night.

Poor outcome treatment measures

1) Pulsatile electrical current (transcutaneous electrical stimulation)

In patients with poor outcome and chronic facial nerve damage, long-term electrical stimulation may be beneficial. In a study on 12 patients with chronic Bell's palsy and 5 patients whose facial nerves had been surgically sacrificed with a mean latency between onset and electrotherapy of 3.7 years, stimulation of the most affected muscles at a submotor level for 6 h *per day* during 6 months significantly reduced facial nerve latencies, the HBS, and collective scores of the 12 clinical impairment measures after 6 months. An improvement by 40%, 30%, or less than 10% was reported in 5, 4 and 8 patients, respectively. The beneficial effect was explained by facilitation of re-innervation through electrical stimulation⁴³.

2) Transmastoid decompression

In one study with 58 patients with Bell's palsy having denervation exceeding 95%, transmastoid decompression of the facial nerve resulted in significant improvement of the HBS and Yanagihara scores 60 days after onset⁴⁴. In a prospective, multicenter trial on patients with a chance of long-term sequels of Bell's palsy, as assessed by nerve conduction studies and electromyography, surgical decompression of the facial nerve through the middle cranial fossa exposure, including the tympanic segment, geniculate ganglion, labyrinth segment, and meatal foramen, significantly improved the chances for normal or near-normal return of facial nerve functions if surgery was carried out within 2 weeks after onset of total paralysis⁴⁵. Since middle fossa craniotomy carries the risk of bleeding, infection, seizures, deafness, leakage of cerebrospinal fluid, or facial nerve injury, this surgical approach cannot be routinely recommended for patients with acute Bell's palsy¹².

3) Gold weight implant

Implantation of gold into the upper eyelids of 16 patients with lagophthalmos due to Bell's palsy resulted in significant reduction of lagophthalmos and improved corneal coverage of 100%. Owing to delayed closure time and disrupted tear film, irritation of the cornea and sclera may persist, so that some patients require ongoing topical treatment of the eye, which may compromise visual acuity⁴⁶.

4) Facial nerve cable grafting

In a retrospective study on 27 patients undergoing facial nerve grafting between 1982 and 1997, those

who had the nerve grafted to a site distal to the meatal foramen had better outcome than those with an anastomosis proximal to the meatal foramen⁴⁷. Microneurovascular free muscle transfer and cross-face nerve grafting are other therapeutic options. The latter involves one of the nerves used for biting¹⁰. In a study on 29 patients who had undergone previous removal of cerebellopontine angle tumor, hypoglossal-facial nerve anastomosis resulted in significant improvement in all of them. A HBS of III or better was achieved in 65 of the included patients⁵.

5) Subperiosteal facial suspension (face lifting)

In an observational study on five patients with a HBS III-V, face lifting resulted in marked improvement in four of them⁴⁸.

6) Botulinum toxin

Synkinesis and facial spasms, common features of partially recovered facial nerve palsies, can be effectively managed by subcutaneous or intramuscular injections of botulinum toxin. In a study on ten patients with synkinesis during a period of 7 years on an average, periorbital injections of botulinum toxin A resulted in marked subjective and objective improvement in nine of them^{8,49}.

Prognosis of Peripheral Facial Weakness

Peripheral facial weakness can improve up to 1 year later. Patients with incomplete weakness have better prognosis than those with complete facial weakness, and the younger is the patient, the better is the prognosis⁸. In patients with incomplete facial weakness, up to 94% make full recovery¹². Without treatment, the prognosis of complete Bell's palsy is generally good, but 20%-30% of the patients are left with varying degrees of permanent disability^{11,34}. About 80%-85% of the patients recover spontaneously and completely within 3 months, whereas 15%-20% experience some kind of permanent nerve damage^{8,11}. About 5% may remain with severe consequences^{1,34}. About 10% of the patients with Bell's palsy experience one or more recurrences after a mean latency of 10 years⁵⁰.

Long-term sequels of Bell's palsy may be persisting weakness, contractures, facial spasms, synkinesis, decreased tearing, crocodile tears, or psychosocial effects¹. In patients who recover without treatment, ma-

ior improvement occurs within 3 weeks. A new wave of recovery of function starts 3 months after onset. If it does not occur within this time, then it is unlikely to be seen by 6 months.

Conclusion

Patients developing Bell's palsy should be treated by a neurologist, ENT specialist, and ophthalmologist with the least possible latency after palsy onset. In all patients suspected to have secondary facial nerve palsy, diagnostic work-up for the presence or absence of the possible causes should be promptly initiated. If any of these causes is detected, it should be assessed whether there is causal relation between the palsy and the detected cause or not. Although a final decision on optimal therapy for acutely developing Bell's palsy cannot be actually proposed, patients should be provided with all therapeutic measures to avoid secondary affection of the eyes if eyelid closure is insufficient or in case of impaired tearing.

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Sažetak

PERIFERNA LEZIJA SEDMOG MOŽDANOG ŽIVCA (BELLOVA PAREZA)

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Periferna lezija facijalnog živca označava oštećenje VII. moždanog živca koje rezultira mišićnom slabošću jedne strane lica. Može biti idiopatska (Bellova paraliza) ili može imati jasan uzrok. Gotovo 80% perifernih lezija facijalnog živca su primarne, a ostale su sekundarne. Među najčešće uzroke sekundarne periferne lezije facijalnog živca ubrajaju se sistemske virusne infekcije, trauma, operacijski zahvati, šećerna bolest, lokalne infekcije, tumori, imuni poremećaji, droge, degenerativne bolesti središnjega živčanog sustava itd. Dijagnoza se zasniva na tipičnoj kliničkoj slici, pretragama krvi, likvora, provodljivosti živca i neuroslikovnim metodama (MRI mozga, rtg lubanje i mastoida). Liječenje sekundarne periferne lezije facijalnog živca zasniva se na liječenju primarnog uzroka, za razliku od liječenja idiopatske periferne pareze facijalnog živca koje je još uvijek predmet nedoumice. Pojedine studije ukazuju na mogući pozitivan učinak steroida i antivirusnih lijekova, dok druge studije ne ukazuju na pozitivan učinak ovog liječenja. Dodatno liječenje uključuje zaštitu oka, fizioterapiju, akupunkturu, injiciranje botulinum toksina ili operacijski zahvat. Bellova paraliza ima uglavnom dobru prognozu s čak 80% potpunih oporavaka; u 15% slučajeva zaostane blaže oštećenje živca, dok u 5% bolesnika ostaju teže trajne posljedice.

Ključne riječi: *Bellova pareza – dijagnostika; Bellova pareza – terapija; Akupunktura*