

A general practice approach to Bell's palsy



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Background

Bell's palsy is characterised by an acute onset of unilateral, lower motor neuron weakness of the facial nerve in the absence of an identifiable cause. Establishing the correct diagnosis is imperative and choosing the correct treatment options can optimise the likelihood of recovery.

Objective

This article summarises our understanding of Bell's palsy and the evidence-based management options available for adult patients.

Discussion

The basic assessment should include a thorough history and physical examination as the diagnosis of Bell's palsy is based on exclusion. For confirmed cases of Bell's palsy, corticosteroids are the mainstay of treatment and should be initiated within 72 hours of symptom onset. Antiviral therapy in combination with corticosteroid therapy may confer a small benefit and may be offered on the basis of shared decision making. Currently, no recommendations can be made for acupuncture, physical therapy, electrotherapy or surgical decompression because well-designed studies are lacking and available data are of low quality.

Bell's palsy is characterised by an acute onset of unilateral, lower motor neuron weakness of the facial nerve in the absence of an identifiable cause.¹ The annual incidence is estimated to be 11–40 per 100,000, with a lifetime risk of one in 60. On average, general practitioners (GPs) encounter one acute case every two years.² Most cases of Bell's palsy resolve spontaneously. Indeed, 71% of patients notice clinical improvement within three weeks of symptom onset and achieve complete recovery within three months. The remainder of patients fail to recover completely and continue to have facial weakness, synkinesis and contractures. Facial dysfunction has a dramatic effect on a patient's appearance, psychological wellbeing and quality of life.³ Management of Bell's palsy is aimed at achieving complete recovery or reducing the negative sequelae in cases that fail to resolve. This article summarises our understanding of Bell's palsy and the evidence-based management options available for adult patients.

Case 1

Mr XY, 32 year of age, presents with a history of rapidly progressive, right-sided facial paralysis. Prior to this, he had been systemically well.

Case 2

Mrs PL, 76 years of age, presents with progressive weakness of the left forehead muscles over the course of two weeks. Prior to this, she had been systemically well.

How to diagnose Bell's palsy

The exact mechanism of Bell's palsy is unknown, although a viral aetiology is suspected.^{1,2} The unilateral facial weakness associated with Bell's palsy is thought to result from facial nerve inflammation and oedema induced by reactivation of *Herpes simplex* or *Varicella zoster* virus.^{1–3} In the temporal bone, the facial nerve travels in a narrow canal; swelling of the nerve may result in compression and subsequent damage. The facial nerve innervates the lacrimal glands, salivary glands, stapedius muscle, taste fibres from the

anterior tongue, and general sensory fibres from the posterior ear canal and tympanic membrane. In addition to unilateral facial weakness, patients may report dryness of the eye and mouth, taste disturbance and hyperacusis. Specific risk factors for Bell's palsy include pregnancy, severe pre-eclampsia, obesity, hypertension, diabetes and upper respiratory illnesses such as influenza.

Diagnosis of Bell's palsy is based on exclusion.¹⁻³ The aim of a thorough history and physical examination is to exclude a neurological, otologic, infectious, inflammatory or neoplastic cause, as well as cerebellopontine angle pathology and vascular insufficiency (Box 1). The history should specifically enquire about the onset and timing of symptoms. Onset of Bell's palsy is sudden, but not stroke-like, and tends to evolve over minutes to hours. Facial weakness often worsens while the patient is waiting in the emergency department.

Other more sinister causes of facial palsy can also evolve at the same rapid rate. Gradual progression of symptoms often indicates an infectious or neoplastic cause (eg perineural spread from a previous facial cutaneous squamous cell carcinoma). It is important to note whether a patient has a previous history of Bell's palsy. Although rare, recurrence of Bell's palsy is possible. Underlying medical problems such as previous stroke, brain tumour, cutaneous cancers of the face and neck, parotid tumour, head or facial trauma or recent infection can also predispose patients to facial weakness and should also be considered. Associated symptoms of diplopia, dysphagia, genuine numbness of the face or dizziness are not typical of Bell's palsy and are red flag symptoms suggestive of other diagnoses.

The physical examination should begin with careful inspection of the ear canal and tympanic membrane for *Herpes zoster* infection, suggestive of Ramsay Hunt syndrome.³ The head and neck should be inspected for cutaneous cancers and palpated for any masses. All cranial nerves should be assessed, with attention to the extent of the facial weakness and whether all nerve branches are involved. The House–Brackmann grading scale is useful for assessing dynamic facial nerve function (Table 1).⁴⁻⁶ Sparing of forehead movement may suggest a central pathology, such as a stroke, or a more peripheral lesion affecting only a single branch of the nerve.

Case 1 continued

Mr XY reported associated symptoms of aural fullness and hyperacusis in his right ear. He denied symptoms of diplopia, dysphagia, numbness of the face or dizziness. His past medical history was significant for chronic lower back pain, which was mechanical in nature. Physical examination revealed normal external auditory canals and tympanic membranes bilaterally. Assessment of facial nerve function on the right side was a House–Brackmann Grade VI, with no evidence of facial movement. The remainder of the head and neck and cranial nerve examinations were within normal limits. Mr XY was diagnosed with Bell's palsy.

Case 2 continued

Mrs PL denied any otalgic symptoms, diplopia, dysphagia, numbness of the face or dizziness. Her past medical history was significant for multiple cutaneous cancers (including a previous moderately differentiated squamous cell carcinoma excised from the left temporal region), hypertension and type 2 diabetes mellitus. Physical examination revealed normal external auditory canals and tympanic membranes bilaterally. Assessment of the facial nerve function on the left side showed loss of tone only in the forehead muscles. The remainder of the head and neck and cranial nerve examinations were within normal limits. Mrs PL was thought to have Bell's palsy.

The diagnosis is Bell's palsy – What to do now?

Bell's palsy is thought to result from facial nerve inflammation and oedema; thus, corticosteroids are the mainstay of treatment and their use has been supported by a number of well-designed, randomised controlled trials.^{1-4,7} Meta-analyses have shown that the rate of residual facial dysfunction at six months is 23% in patients who received corticosteroid treatment, compared with 33% in the control group. On the basis of a recent review, the

Box 1. Causes of facial weakness

Neurological

- Stroke (upper motor neuron palsy)
- Guillain–Barré syndrome
- Multiple sclerosis

Otologic

- Acute or chronic otitis media
- Malignant/necrotising otitis externa
- Cholesteatoma
- Schwannoma

Infectious

- *Herpes zoster* virus
- Mumps
- Rubella
- Epstein–Barr virus

Inflammatory

- Sarcoidosis

Neoplastic

- Cerebral tumour
- Cutaneous cancer of the face and neck
- Parotid tumour
- Metastatic tumour
- Lymphoma

Idiopathic

- Bell's palsy

Trauma

- Temporal bone fracture
- Surgical intervention with subsequent damage to the facial nerve

American Academy of Otolaryngology has recommended a 10-day course of oral steroids, with at least five days at a high dose (either prednisolone 50 mg daily for 10 days or prednisone 60 mg daily for five days, then tapered over five days).³ Oral steroids should be initiated within 72 hours of symptom onset. The benefit of treatment after 72 hours is less clear.

To date, there is no evidence to suggest that oral antiviral therapy alone is effective for the management of Bell's palsy.^{1,3} With regard to the rate of facial nerve recovery, antiviral therapy alone has been shown to be inferior to corticosteroid therapy. Well-designed clinical trials have shown that antiviral therapy in combination with corticosteroid therapy is of no additional benefit, compared with corticosteroid therapy alone; however, a small benefit cannot be completely excluded. Currently, the most extensively studied antiviral agents are acyclovir (400 mg five times daily) and valacyclovir (1000 mg three times daily). The main adverse effects associated with these drugs are nausea,

vomiting and diarrhoea. Given that there is a small potential for benefit and the adverse effects are of low risk, the American Academy of Otolaryngology has recommended that patients are offered combination corticosteroid and antiviral therapy within 72 hours of symptom onset, based on shared decision making.³ As previously discussed, it is important to exclude Ramsay Hunt syndrome on physical examination. Management of Ramsay Hunt syndrome entails antiviral therapy, often given intravenously, in addition to corticosteroids.

Patients with incomplete eye closure are at risk of foreign body deposition, exposure keratitis, corneal ulceration and eventual loss of vision.^{3,7} Eye-protective measures in these patients are imperative. To prevent corneal damage, patients are encouraged to wear sunglasses when outdoors, use lubricating eye drops/ointments regularly and tape the eyelid shut overnight when sleeping. GPs should also counsel patients to immediately report any symptoms of eye irritation, pain or changes to vision.

For patients who are interested in adjunctive forms of treatment such as acupuncture, physical therapy or electrotherapy, currently no recommendations can be made because there is a lack of well-designed studies.¹⁻³ Use of these treatments should be based on assessment of benefit versus harm and shared decision making. Similarly, no recommendation can currently be made regarding surgical decompression as a form of treatment for Bell's palsy, because the available data are of low quality.

Table 1. The House–Brackmann grading scale for assessing dynamic facial nerve function

Grade I	Normal function Normal facial function in all areas
Grade II	Mild dysfunction Slight weakness noticeable on close inspection Slight synkinesis Normal symmetry and tone at rest Moderate to good forehead movement Complete eye closure with minimal effort Slight asymmetry of the mouth
Grade III	Moderate dysfunction Obvious but not disfiguring weakness Noticeable synkinesis, contractures and/or hemifacial spasms Normal symmetry and tone at rest Slight to moderate forehead movement Complete eye closure with effort Slight asymmetry of the mouth with maximum effort
Grade IV	Moderately severe dysfunction Obvious and disfiguring weakness Normal symmetry and tone at rest No forehead movement Incomplete eye closure Asymmetry of the mouth with maximum effort
Grade V	Severe dysfunction Barely perceptible movement Asymmetry at rest No forehead movement Incomplete eye closure Slight mouth movement
Grade VI	Total paralysis No facial movement

When should a patient be referred?

GPs should refer patients with new or worsening neurological findings, facial diplegia, ocular symptoms or complications, or incomplete facial nerve recovery three months after initial symptom onset. These patients should be referred to a neurologist or otolaryngologist.³ A second opinion is warranted in these groups of patients, as a condition other than Bell's palsy may be the cause for facial weakness.

Investigations for neoplasms along the course of the facial nerve should be performed and include magnetic resonance imaging (MRI) or high-resolution computed tomography. MRI is useful for evaluating the brainstem, cerebellopontine angle, interfaces between bone and soft tissues, and the parotid gland. In red-flag cases where the initial MRI result is negative, it may be useful to repeat the scan in three months if the clinical suspicion is high. Computed tomography is better suited for evaluating the intratemporal segment of the nerve.⁷ Patients with incomplete eye closure, ocular symptoms or complications require a referral to an ophthalmologist for further evaluation and treatment.

From a psychological point of view, patients with facial dysfunction suffer from depression and a reduced quality of life as a result of their appearance.³ Patients may benefit from support and counselling to cope with the emotional and physical consequences of their condition. Reconstructive procedures are also available to improve the appearance and function of the face;

examples include brow lifts, eyelid weights and static and dynamic facial slings. Patients can be referred to a plastic and reconstructive surgeon for consideration of these procedures.

Case 1 continued

Mr XY was started on prednisolone 50 mg daily (for 10 days) and valacyclovir 1000 mg three times daily. In addition, he was prescribed ocular lubricants, to be applied periodically, and advised to securely patch his right eye while sleeping. Mr XY started to notice clinical improvement within three weeks of the symptom onset and achieved complete recovery within three months.

Case 2 continued

Mrs PL was managed with high-dose oral steroid and antiviral therapy. She failed to respond to treatment and at three months was noted to have progressive facial weakness now involving the lower face. Mrs PL was referred to an otolaryngologist, who organised for Mrs PL to have an MRI. The MRI scan showed enhancement along the left facial nerve. Mrs PL underwent a facial nerve biopsy; histopathology confirmed perineural invasion of squamous cell carcinoma. She then had a radical parotidectomy and excision of the facial nerve through to the second genu via a mastoidectomy to obtain clear margins; this was followed by postoperative radiotherapy.

Interestingly, a study has shown that over 20% of patients with perineural spread have no known primary skin tumour.⁸ In addition, over one-third of patients with a known primary skin tumour did not have perineural invasion detected in the primary. Therefore, in patients with progressive facial nerve neuropathy, it is important

to consider perineural spread even in the absence of a history of cutaneous cancers.

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