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PRACTICE POINTER

Assessment and management of facial nerve palsy

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The facial nerve is important for both communication and expression, and impairment of its function can severely affect quality of life.[1] The main concern at first presentation of a facial nerve lesion is to exclude the possibility of a stroke or other serious cause.[2] The figure outlines possible causes. Correct management within the first few days may prevent long term complications.

How is it assessed?

The facial nerve is responsible for motor supply to the muscles of facial expression (frontalis, orbicularis oculi, buccinators, and orbicularis oris) and stapedius, parasympathetic supply to the lacrimal and submandibular glands, and sensory input from the anterior two thirds of the tongue. Thus, as well as a facial droop, patients may present with a dry eye, reduced corneal reflex, drooling, hyperacusis, altered taste, otalgia, and speech articulation problems.[5]

Upper motor neurone

After identifying the affected side, it is important to establish whether an upper motor neurone lesion is responsible for the facial weakness. Although not an infallible sign,[3] classic neurology describes a bilateral innervation of that part of the facial nuclei supplying the forehead, and thus preserving forehead movement in upper motor lesions. Lower motor neurone disorders of the main nerve trunk result in a weakness of the entire side of the face.

Patients may have risk factors for stroke, which include older age (>60 years), hypertension, previous stroke or transient ischaemic attack, diabetes, high cholesterol, smoking, and atrial fibrillation.[6] Corroborative evidence may also be found by examining for abnormalities in other cranial nerves and the peripheral nervous system—increased tone, limb weakness, hyper-reflexia, upgoing plantars, and sensory loss.[4]

When an upper motor neurone lesion is suspected, it may help to determine whether this is localised to the brainstem or cerebral cortex. Brainstem disease may present with vertigo, ataxia, or crossed neurology signs (ipsilateral cranial nerve involvement and contralateral hemiplegia). A cortical lesion often affects the contralateral limbs and involuntary movements of the face, such as spontaneous smiling, may be spared.[6] Urgent referral to secondary care (neurology or acute medical unit) is needed at this stage to assess the need for thrombolysis.

Lower motor neurone

Once a central cause for facial palsy has been excluded, perform a focused examination of the ears, mastoid region, oral cavity, eyes, scalp, and parotid glands to look for the specific signs in the table. Bell's palsy is an idiopathic lower motor neurone (LMN) facial nerve paralysis that accounts for most new cases (incidence 10-40/100 000 population each year).[3 7] However, 30-41% of patients with LMN facial nerve weakness will have another cause that requires specific management and is often associated with a poorer prognosis.[2-4]

Bell's palsy is a diagnosis of exclusion, made only after excluding features in the table. Epidemiological studies suggest that it normally develops fully within 24-48 hours.[3 4] Most cases (60%) are associated with mild post-auricular pain. Incidence does not differ significantly with ethnicity or sex, but diabetes is associated with as many as 10% of cases, and it occurs more often in the last trimester of pregnancy (three times baseline risk).[8] Incomplete facial nerve paralysis at presentation (some residual muscle movement and partial closure of the eyelid) is a good prognostic sign, indicating a 94% chance of full recovery, as opposed to 61% in those with complete paralysis.[3]

The next most common cause of facial nerve paralysis is trauma (accidental or surgical). Accidental trauma includes any sharp or blunt mechanism of injury, such as facial laceration, stab injury, or temporal bone fracture. Detection of a temporal bone fracture is essential because of the 90% risk of associated intracranial disease.[9] A post-mortem correlation study indicates that periorbital bruising (racoon sign), mastoid bruising (Battle's sign), and blood in the ear canal have a positive predictive value of 85%, 66%, and 46%, respectively, for detecting a temporal bone fracture.[10] Cerebellopontine angle and middle ear surgical procedures are the main causes of iatrogenic injury, which may also be seen after procedures carried out on the parotid gland or any other region along the facial nerve.[4]

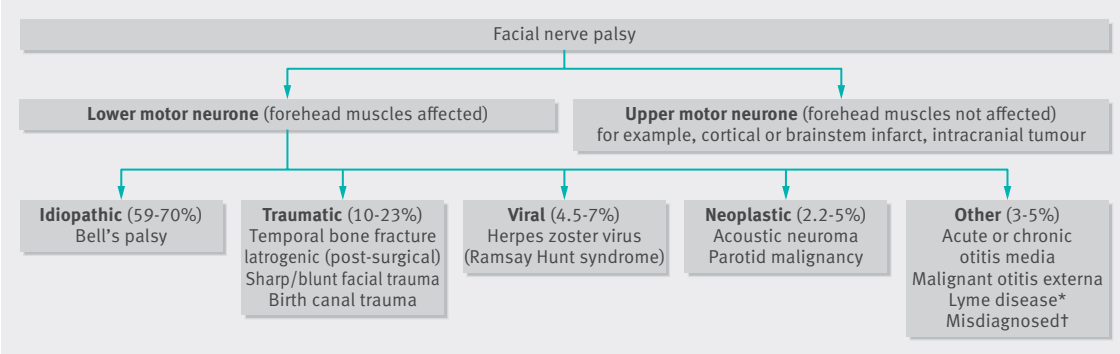
After infection by herpes zoster virus, a geniculate ganglionitis causes a prodrome of otalgia and vesicular eruption within the ear canal, with or without spread to the oral cavity. Facial paralysis (Ramsay Hunt syndrome) normally follows this and is associated with sensorineu-

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

We sought feedback on the paper from patient and medical representatives of the charity Facial Palsy UK. We incorporated their comments into the paper and developed a patient consultation guide for management and prognosis of Bell's palsy (see box below)

THE BOTTOM LINE

- In patients presenting with facial weakness, the first priority is to exclude an upper motor neurone lesion; important associated signs may include concurrent limb weakness, hyper-reflexia, upgoing plantars, or ataxia
- Check for causes of a lower motor neurone lesion by examining the ears, mastoid region, oral cavity, eyes, scalp, and parotid glands
- Bell's palsy is a diagnosis of exclusion, and oral steroids are needed within 72 hours to increase the chance of complete recovery. Prognosis is usually good compared with other causes of lower motor neurone weakness, such as tumours and Ramsay Hunt syndrome
- Eye protection is crucial if lid closure is impaired



Differential diagnosis of a unilateral facial palsy. Percentages are based on combined epidemiological data from 6024 patients with lower motor neurone facial palsy (rarer conditions including mumps, syphilis, HIV, Guillain-Barré syndrome, otitic barotrauma, myasthenia gravis, systemic lupus erythematosus, sarcoidosis, and multiple sclerosis have been excluded).[3 4] *Endemic in forested regions; †misdiagnosed cerebrovascular disease evident in about 1.5% of all patients[3]

ral hearing loss and vertigo in 40% of cases owing to involvement of cranial nerve VIII.[6] Patients with Ramsay Hunt syndrome generally have a poorer prognosis than those with Bell's palsy, with only 21% showing full recovery at 12 months.[3]

A slowly progressive onset of facial weakness is suggestive of cancer.[3 5] In addition, cancer may be associated with pain or paralysis of select branches of the nerve, such as zygomatic (eyelid) or marginal mandibular (angle of mouth) branches.[7] There may be a history of regional cancer. A thorough examination of the head and neck region will be needed to look for cervical lymphadenopathy, a parotid mass, or a scalp lesion in particular. Acoustic neuromas account for about 80% of cerebellopontine angle lesions and most cases of tumour related LMN facial nerve paralysis; they can be differentiated from other causes by ipsilateral sensorineural hearing loss (95% of cases) and absence of the corneal reflex (60%).[4 11]

Bacterial infections are responsible for 1-4% of new cases of LMN facial palsy.[3 4] Acute otitis media accounts for most and is associated with systemic sepsis, a bulging tympanic membrane, conductive hearing loss, and pinna lateralisation. Malignant otitis externa (or, more accurately, skull base osteomyelitis) is characterised by lack of sleep due to otalgia. More than 95% of cases are seen in older people (>65 years), immunocompromised people, and those with poorly controlled diabetes.[12] The condition is associated with *Pseudomonas aeruginosa* infection.[12] Lyme disease is a bacterial infection caused by a tick bite that results in facial nerve paralysis in one in 10 seropositive patients.[13]

How is it managed?

Management is influenced strongly by the initial clinical review and provisional working diagnosis. With an LMN lesion, the main priority is to treat the underlying cause, to improve symptoms, and to reduce associated morbidity, such as contracture of the facial muscles, synkinesis (involuntary movement of one part of the face due to aberrant re-innervation), and autonomic dysfunction (crocodile tears or hemi-facial spasm). In Bell's palsy, routine investigations in the primary care setting are no longer recommended.[2]

Red flags for urgent referral

These include potential upper motor neurone causes (such as limb paresis, paraesthesia of the face or limbs, involvement of other cranial nerves, postural imbalance), trauma, features suggesting cancer (such as gradual onset, persistent facial paralysis >6/12, pain within the facial nerve distribution, ipsilateral hearing loss, suspicious head or neck lesion, previous regional cancer), and acute systemic or severe local infection. Urgent paediatric referral is warranted in children, for whom Bell's palsy is less likely to be a cause of facial weakness (<50% of cases).[5]

Treatments applicable to all patients

Eye care is paramount for those with corneal exposure. To prevent ulceration or dehydration of the cornea, apply artificial tears (such as hypromellose drops) every one or two hours during the day. At night, keep the eye moist by using a thin strip of paraffin based ointment (such as Lacrilube) and secure the upper eyelid in the closed position by applying permeable synthetic tape (http://www.facialpalsy.org.uk/advice/guides/how-to-tape-eyes-shut/433). All cases of incomplete eyelid closure require urgent ophthalmology consultation at presentation.

Some cases of LMN facial palsy will need to be referred to the ear, nose, and throat department or another hospital specialty. Such cases include all patients with atypical symptoms (see table) and those with suspected Bell's palsy who do not respond to a trial of oral prednisolone (observe for maximum of two to three weeks). Post referral tests may include computed tomography or magnetic resonance imaging to visualise the skull base, stylomastoid foramen, and parotid gland; blood tests (full blood count, urea, and electrolytes); pure tone audiography; and topographical studies (such as Schirmer's test, taste sensation, and stapedial reflex). Electroneuronography can guide prognosis in cases of complete paralysis, but this test is expensive, time consuming, and it has a short window of opportunity after onset of symptoms (less than three weeks). In the case of longstanding facial palsy, impairment of eye closure may require insertion of a gold weight into the upper eyelid or lateral tarsorrhaphy.[14]

Clinical signs to check in lower motor neurone facial nerve palsy	
Are the ears clear on otoscopy?	Assess the tympanic membrane and ear canal; acute or chronic otitis media (±cholesteatoma) and malignant otitis externa can all cause lower motor neurone facial palsy and will require further urgent assessment in secondary care (ear, nose, and throat)
Is there ipsilateral hearing loss?	Perform Weber's and Rinne's tuning fork tests; although Ramsay Hunt syndrome will be associated with sensorineural hearing loss (SNHL), it is important to exclude a cerebellopontine angle lesion (normally presents with gradual onset unilateral SNHL); acute otitis media or cholesteatoma may be associated with conductive hearing loss, whereas patients with Bell's palsy normally have abnormal sensitivity to loud sounds
Is there a rash?	Small vesicular eruptions that affect the tympanic membrane, ear canal, external pinna, or oral cavity may indicate Ramsay Hunt syndrome; Lyme disease is restricted to heavily forested regions and presents with a characteristic erythematous "bullseye" lesion on the limbs or trunk (70% of cases), arthralgia, and facial swelling; it is caused by a bite from a tick carrying *Borrelia burgdorferi* (US) or *B afzalii/garinii* (Europe), and an antibody test may help confirm the diagnosis[13]
Are there any bruises or scars in the head and neck region?	The palsy may be secondary to a skull base fracture (common associated signs include periorbital or mastoid bruising, blood in the ear canal, and haemotympanum)[9] or recent mastoid, parotid, or submandibular gland surgery
Is the corneal reflex intact on both sides	In addition to facial muscle weakness, a cerebellopontine angle lesion (such as an acoustic neuroma) may cause a reduced or absent corneal reflex on the affected side ± aural fullness (owing to trigeminal nerve deficit)
Is the mastoid region tender or swollen?	A tender swelling of the mastoid with associated middle ear inflammation or pinna lateralisation (or both) may suggest acute mastoiditis
Is the parotid gland enlarged?	Palpation of a parotid lump may suggest cancer (particularly if associated with a history of regional skin cancer, delayed onset facial palsy, or pain); if cancer is suspected, thoroughly examine the rest of the head and neck and refer urgently (for example, through the "two week wait suspected head and neck cancer pathway" in the UK)

Alternative rehabilitation methods include physical therapy (facial retraining exercises, transcutaneous electrical stimulation, acupuncture), botulinum toxin injections (to reduce facial muscle contractures, synkinesis, and hemifacial spasm), dynamic facial reanimation surgery, or counselling. Of these interventions, only physical therapy has been subjected to controlled trials,[15] and no overall benefit over placebo was found. Tailored facial retraining exercises show limited evidence of earlier recovery of nerve function but this result needs to be confirmed by future trials that are adequately powered with low risk of bias.

Patient consultation guide for management and prognosis of Bell's palsy

What is Bell's palsy?

This condition involves swelling adjacent to the facial nerve as it passes through the skull base into the ear. Compression of this nerve can stop the muscles that it supplies from working. The cause of the swelling is currently unknown.[22]

How is it managed?

Steroid tablets (usually prednisolone) help to reduce inflammation and are normally taken for 10 days.[18 19] This short course of drugs is unlikely to have notable side effects if you have no history of high blood sugar levels (diabetes), hypertension, gastric ulcer, or glaucoma.[22] To ensure maximum benefit, the steroid tablets should be started within three days of the facial weakness appearing.[5]

If the eyelid cannot shut completely, the surface of the eye may dry up and be harmed. In addition, the tear ducts may not function temporarily, which could dry the eye further.[22] Treatment is needed to keep the eye moist. This normally involves frequent lubricating eye drops during the day. At night, eye ointment can be applied before closing the eye shut with tape.

What is the outcome?

Most studies indicate that if a steroid is not prescribed, seven of 10 patients will recover completely. If a steroid is taken, eight of 10 patients will recover completely.[16] Most patients will note an improvement in their facial weakness within three weeks, with the remainder resolving by three to five months.[3]

In the 20-30% of cases where facial weakness does not recover fully, further interventions may be considered. These may include physiotherapy to undergo "facial retraining exercises" or Botox injections to help with muscle spasms. It is important that other potential causes of facial palsy are excluded by referral to a specialist if recovery fails to progress. A considerable proportion of patients are left with psychosocial concerns because of their limited facial function and may need psychological support.[23] Surgical treatments also help improve the functional and cosmetic appearance of the face.

Treatments in primary care for Bell's palsy and Ramsay Hunt syndrome

Corticosteroids in Bell's palsy

A Cochrane review (1569 patients, eight randomised trials) found that significantly more patients taking oral steroids recovered complete motor function, compared with those taking placebo, if started less than 72 hours after symptom onset (77% *v* 67%; relative risk 0.71, 95% confidence interval 0.61 to 0.83).[16] They also had significantly fewer motor synkinesis symptoms (0.60, 0.44 to 0.81).[16] The results of one randomised trial that recruited patients to oral steroids versus placebo up to one week after symptoms began were significantly inferior to those of trials that recruited within 48 hours.[17 18] Randomised trials showed two steroid regimens to be of similar, significant benefit—prednisolone 25 mg twice daily for 10 days or 60 mg once daily for five days (the last dose should be tapered by 10 mg/day over the subsequent five days).[18 19] A systematic review of 10 randomised controlled trials found no significant difference in adverse event rates between oral steroids and placebo,[20] although most studies excluded patients with specific contraindications (such as poorly controlled diabetes, immune compromise, hypertension, peptic ulcer disease, glaucoma, active tuberculosis, first and second trimester of pregnancy, sepsis, renal or hepatic impairment, and psychosis).[2 5 21] It is important to discuss specific risks versus benefits of treatment with patients (box).

Antivirals

A meta-analysis of combination therapy (anti-viral (aciclovir or valaciclovir) plus oral steroid (prednisolone)) for Bell's palsy, suggested a marginal benefit only when small poorer quality trials are included.[24] The conclusion suggested that combined therapy should be reserved for patients with suspected Ramsay Hunt syndrome (herpes zoster virus infection). In these patients, primary care physicians may wish to start a trial of oral steroids (dose as described above) if there are no contraindications, together with an antiviral agent (such as 1 g valaciclovir three times daily for one week.[5 25] Because of the relatively poor functional outcome in this group and the high rate of coexistent sensorineural hearing loss, it is advisable (where appropriate) to consider referral to ear, nose, and throat specialists for repeat assessment and imaging of the cerebellopontine angle.