BRITISH JOURNAL OF HOSPITAL MEDICINE
MMC Modernising Medical Careers

MODERNISING MEDICAL CAREERS

IN NEXT MONTH'S MMC SUPPLEMENT

How to make the most of your imaging department

Dealing with complaints 1: a guide for the foundation doctor

Tips on giving a good handover

Facial nerve palsy: assessment and management

Introduction

The facial nerve (cranial nerve VII) is the most common site of unilateral cranial nerve palsy. Patients with facial nerve palsy may present in either the emergency department or primary care setting and it is important to be familiar with appropriate assessment and management. The most common cause of facial palsy is idiopathic (otherwise known as Bell's palsy), accounting for around 50% of cases. This is essentially a diagnosis of exclusion and therefore a systematic approach to history, examination and investigation is required to ensure that the correct diagnosis is made.

Anatomy

A sound knowledge of the anatomical supply of the facial nerve is important in order to interpret symptoms and signs. The facial nerve has a larger motor component which supplies the muscles of facial expression, posterior belly of digastric, stapedius and the platysma layer of the skin. There are smaller sensory components which supply taste to the anterior two thirds of the tongue and sensation around the external auditory meatus. The facial nerve also carries parasympathetic secretomotor fibres to the parotid, submandibular, sublingual and lacrimal glands.

History

The most common presenting complaint is that of unilateral facial weakness. Other associated symptoms include facial or retroauricular pain, altered taste, hyperacusis and dry eyes. Chronicity of symptoms is important in that a facial palsy that has shown no sign of improvement after 3 weeks is a red flag for a diagnosis other than a simple Bell's palsy. However, the patient with facial nerve palsy usually presents early because of the disfiguring nature of the condition and so follow up is mandatory to assess progression. Marked otalgia is not common in Bell's palsy and is suspicious of aural causes of facial palsy, e.g. Ramsay–Hunt syndrome, cholesteatoma or acute suppurative otitis media.

The patient should be asked about any recent history of fevers, rashes and arthralgia as the tick-borne disease Lyme disease is a cause of facial nerve palsy in endemic areas. Diabetes mellitus is present in more than 10% of patients with Bell's palsy and given that corticosteroids may be prescribed it is sensible to ask specifically about this condition. Recent immunization and history of neurological infection can result in mononeuropathies or polyneuropathies and so are also relevant points in the history.

In the emergency department, a history of significant head trauma is relevant as there may be an underlying temporal bone fracture.

Examination

A concise combined head and neck plus neurological examination is the key to adequate assessment as there are numerous possible causes of facial nerve paralysis (*Table 1*).

The first part of the examination involves discerning whether the paralysis is likely to be caused by an upper or lower motor neurone lesion. A central upper motor neurone lesion will only result in weakness of the lower facial muscles as there is bilateral cortical innervation of the forehead. In this way, when the patient is asked to raise the eyebrows there should be no impairment if he/she has an upper motor neuron lesion but there will be unilateral impairment with a lower motor neuron lesion. This is not a definitive test for diagnosis of a central lesion as forehead sparing may be present with selective distal lesions in the facial nerve distribution. Therefore, it is important to carry

Mr Irfan Syed *is Specialty Trainee Year 2 in Ear, Nose and Throat Surgery and*
Mr Mahmood Bhutta *is Specialist Registrar in Ear, Nose and Throat Surgery in the Department of Otolaryngology/Head and Neck Surgery, Wexham Park Hospital, Slough SL2 4HL*

Correspondence to: Mr I Syed

Table 1. Causes of facial nerve palsy

Idiopathic	Bell's palsy
Trauma	Temporal bone fracture
Infective	Acute suppurative otitis media
	Malignant otitis externa
	Herpes zoster oticus (Ramsay–Hunt syndrome)
	Tuberculosis
	Lyme disease
	Meningitis
	Encephalitis
	Other viruses, e.g. HIV, influenza, coxsackie, mumps
Iatrogenic	Post surgery, e.g. parotidectomy
	Following vaccination, e.g. tetanus
Neurological disorders	Guillain–Barré syndrome
	Motor neurone disease
	Multiple sclerosis
Inflammatory	Sarcoidosis
	Autoimmune disease, e.g. myasthenia gravis
Congenital	Mobius syndrome
	Melkersson–Rosenthal syndrome
Neoplasia/mass	Cholesteatoma
	Meningioma
	Facial nerve tumour
	Vestibular schwannoma
	Parotid tumour
	Glomus jugulare

out a full examination of all the cranial nerves and a peripheral nervous system examination. For example, a central lesion would be a more likely diagnosis in the presence of associated nerve palsies and hemiparesis or hemiplegia of the ipsilateral upper limb.

If a lower motor neurone is suspected the affected muscle groups can be rapidly assessed. This may help identify the site of lesion and is also useful for documentation of the progression of nerve palsy. Gross tests for facial nerve function include eyebrow raising (frontalis supplied by temporal branches), eye closure (temporal and zygomatic branches supplying orbicularis oculi), smiling and blowing cheeks out (buccal and marginal mandibular branch innervated muscles).

The House–Brackmann classification (House and Brackmann, 1985) is the most commonly used system of facial nerve injury classification. It is important to document the extent of facial nerve function so that one can ascertain whether nerve function is improving or worsening at subsequent consultations (*Table 2*).

A comprehensive examination should be carried out using the information gathered from the primary neurological assessment. The purpose of examination is to clinically exclude pathology adjacent to the facial nerve that could be causing its malfunction. Therefore, having established that the facial palsy is a lower motor neurone lesion, it is prudent to:

- Examine the function of adjacent cranial nerves (to exclude a lesion of the facial nerve at the pons, e.g brainstem tumour)
- Test hearing (to exclude a lesion of the facial nerve at the cerebellopontine angle or internal auditory meatus, where it is adjacent to the vestibulocochlear nerve, e.g. vestibular schwannoma). Tuning fork tests may detect any gross sensorineural hearing loss
- Examine the ear (to exclude a lesion affecting the facial nerve as it courses through the middle ear or mastoid, e.g. acute suppurative otitis media, cholesteatoma). The clinician should examine the external ear for any vesicles of herpes zoster infection that may have progressed to Ramsay–Hunt syndrome. If there is a history of head injury, the mastoid should be examined for any ecchymoses which may suggest basal skull fracture
- Examine the parotid gland (to exclude extrinsic compression of the extracranial facial nerve as it courses through this gland, e.g. acinic cell carcinoma). Examination of the parotid also includes inspection of the oropharynx as a tumour of the deep lobe of the parotid may present as an ipsilateral oropharyngeal mass.

Investigations

At the primary consultation, a full history and examination should guide the choice of further investigations that need to be organized. Formal audiological testing in the form of pure tone audiogram is useful as the presence of asymmetry helps to localize the lesion. It also guides the need for and mode of further imaging, e.g. magnetic resonance imaging.

If the facial nerve palsy has a gradual onset with a history of worsening facial nerve function or there is associated cranial nerve involvement suggesting an anatomical lesion then further imaging is indicated. Magnetic resonance imaging with gadolinium enhancement is the modality of choice for identifying tumours of the facial nerve and can also indicate inflammation or oedema around the nerve. It may also be of benefit in identifying impalpable parotid gland tumours. Topographical testing, e.g. stapedial reflexes, taste testing or salivary flow rates, is now obsolete.

There is no benefit of electrophysiological testing, e.g. electromyography, in the acute investigation and management of facial nerve palsy. Electromyography is useful as a prognostic tool in assessing reinnervation potential by detecting fibrillation potentials but these only appear around 2 weeks after symptom onset.

Table 2. House–Brackmann classification of facial nerve function

Grade	Description
I (Normal)	Normal symmetrical function in all areas
II (Mild dysfunction)	Slight weakness noticeable only on close inspection. Complete eye closure with minimal effort
III (Moderate dysfunction)	Obvious weakness, but not disfiguring. Complete eye closure. Strong but asymmetrical mouth movement with maximal effort
IV (Moderately severe)	Obvious disfiguring weakness. Incomplete eye closure. Asymmetry of mouth with maximal effort
V (Severe dysfunction)	Motion barely perceptible. Incomplete eye closure. Slight movement corner mouth
VI (Total paralysis)	No movement

From House and Brackmann (1985)

Management

Figure 1 shows an example protocol for the initial assessment and management of facial nerve palsy. If there is no obvious cause for the facial palsy, the patient should be treated as having Bell's palsy and reviewed again after 2–3 weeks. If the facial palsy has worsened or not improved then ear, nose and throat referral is indicated for further investigation.

Obvious precipitating causes should be treated with specialist expertise when necessary. For example, acute suppurative otitis media will require systemic antibiotic therapy and may require emergency grommet insertion. Ramsay–Hunt syndrome can be treated with acyclovir or valaciclovir antiviral therapy. Direct trauma may require surgical intervention depending upon the extent of the injury, e.g decompression or nerve repair directly or with interposition graft.

Bell's palsy

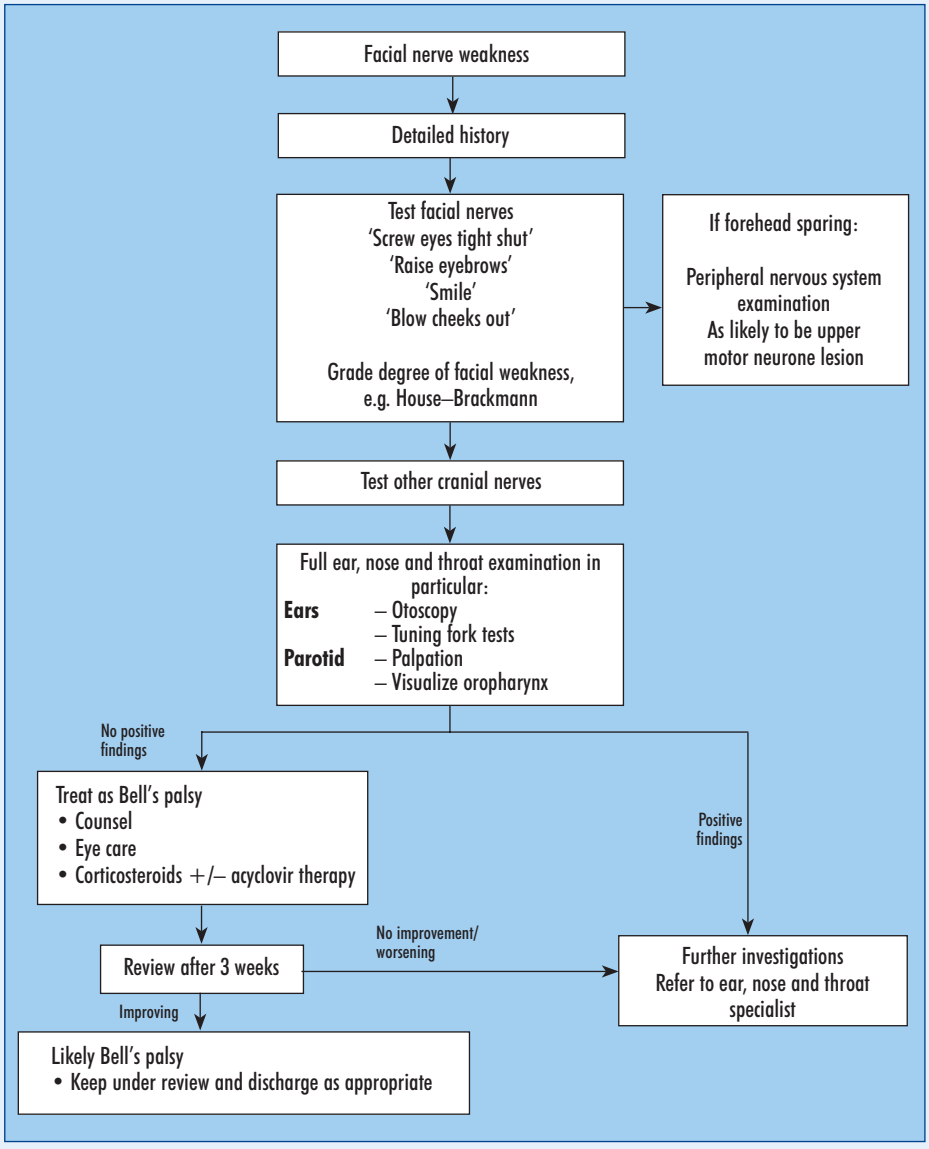
Sir Charles Bell was a Scottish surgeon-anatomist who was first believed to have described the presentation of idiopathic facial paralysis in the early 19th century.

If after completing the thorough history, examination and indicated investigations, there is no obvious cause of the facial palsy then the diagnosis of idiopathic or Bell's palsy can be made. Such a diagnosis accounts for around 50% cases of unilateral facial palsy.

Figure 2 shows a patient with a unilateral facial palsy. There is unilateral weakness of both upper and lower facial muscle groups.

Figure 2. Right-sided facial nerve palsy. The lesion is more obvious when the patient is asked to smile.

Figure 1. Example protocol for initial assessment of facial nerve palsy.



On gross examination, drooping of the eyebrow and corner of the mouth may be noticeable (the latter more obvious when the patient is asked to smile). Bell's phenomenon refers to the upward diversion of the eye when the patient attempts to close the eyelids. At rest there may be incomplete closure of the affected eyelid.

The precise aetiology is not completely understood but the most popular theory is of inflammation secondary to viral infection in particular herpes simplex viruses (Murakami et al, 1996). The condition affects males and females equally and is bilateral in less than 1% of cases.

Eighty five per cent of patients will recover completely. Around 10% will have recovery with some facial asymmetry, whereas 5% are left with severe sequelae.

The most important prognostic indicators are the extent of the palsy and the time taken for recovery to begin. A partial palsy that is associated with improvement in muscle weakness early has a very high probability of complete recovery.

In cases of Bell's palsy, there are a few general key management points:

Counselling

Bell's palsy in the acute stage can be severely disfiguring and is therefore associated with significant anxiety particularly in the young patient where cosmesis is very important. This should be taken into consideration in the patient consultation and it is important to make clear that complete recovery is highly likely.

Eye care

When there is incomplete lid closure (House–Brackmann grade IV–VI), the cornea is left unprotected and vulnerable to drying and injury. Such patients should be provided with artificial tears to be used regularly throughout the daytime. At night the affected eye should be taped shut or covered.

In the case of perceived Bell's palsy, the patient should be encouraged to report any new symptoms or signs that develop following the consultation which may suggest an alternative cause.

Follow up

If following history, examination and relevant investigations at the primary consultation a diagnosis of Bell's palsy seems likely it is important to follow up the patient after a few weeks to assess progress (earlier if there are any new symptoms or signs).

If there is no improvement then imaging and other investigations may be indicated as discussed earlier and the patient should be referred to an ear, nose and throat specialist. If no other diagnosis is established then the patient should be counselled of the fact that improvement may still occur but that this is less likely and may take up to several months.

Medical treatment

There has been much debate regarding the pharmacological treatment of Bell's palsy. Acyclovir and prednisolone are the two most commonly prescribed drugs for treatment of Bell's palsy in the acute stage. The latest Cochrane reviews have not shown any conclusive evidence of any benefit from either drug (Allen and Dunn, 2001; Salinas et al, 2004). However, a more recent randomized double blind trial has shown that early treatment with prednisolone significantly improves the chances of recovery at 3 and 9 months. There was no benefit in the use of acyclovir either alone or in combination with prednisolone in this study (Sullivan et al, 2007).

It has been suggested that the failure of antiviral therapy to show significantly positive results in treating Bell's palsy is a result of the poor bioavailability of the active drug. Further trials using drugs with higher bioavailability are required to establish any worthwhile benefit. The pro-drug valaciclovir has much better bioavailability that acyclovir and it may be that future studies will demonstrate benefit from this therapy.

If there are no contraindications to steroid use, the risk–benefit analysis probably errs in favour of the use of high dose steroids (e.g. 1 mg/kg/day prednisolone) in the short term (10 days) for treatment of Bell's palsy, if treatment is commenced within a few days of onset. The authors would also give valaciclovir therapy (1 g three times daily for 7 days), because again the risks of such treatment are small.

There is insufficient evidence to recommend a role for other therapies directed at improving the impaired microcirculation to the facial nerve including stellate ganglion block and low molecular-weight dextran infusion.

Physiotherapy

There is no strong evidence for the use of facial exercises in improving outcome in the acute phase of Bell's palsy. Facial muscles do not suffer from rapid onset of disuse atrophy and so the aim should be to allow the inflammation around the nerve to resolve. In patients with long term and possibly permanent palsy, facial exercises may be useful in correcting synkinesis, spasms and inappropriate movements. More sophisticated techniques including neuromuscular training are now available which involve the use of surface electromyography to guide facial muscle movements.

Surgery

There are a number of surgical procedures that can be offered depending upon the circumstances of the patient. Cosmetic surgical techniques can be used that aim to minimize the asymmetry of the face that can be disfiguring, e.g. brow lifts or Botox injections. There are other techniques which have a functional role rather than purely cosmetic. For example, gold weight can be inserted into the eyelid via a blepharoplasty incision to allow eye closure. Tarsorrhaphy allows eye closure but has cosmetic implications and may cause a blinkering effect to the vision towards the side of surgery. Cross facial nerve grafts and muscle transfer surgery can be used to reanimate the paralysed side of the face. Such facial reanimation surgery is only indicated in certain carefully selected cases.

Facial nerve decompression surgery has also been considered as treatment for Bell's palsy. Surgery has only shown efficacy shortly after the onset of symptoms and given that most cases resolve spontaneously or with medical treatment, the risks of surgery are too high to recommend such an invasive intervention routinely.

Conclusions

A thorough initial clinical assessment to establish the likely aetiology of facial nerve palsy is key to providing appropriate effective intervention. Whenever there is uncertainty regarding the cause, on initial or subsequent assessment, early consultation with an ear, nose and throat specialist is advisable. **BJHM**

Conflict of interest: none.

Allen D, Dunn L (2001) Aciclovir or valaciclovir for Bell's palsy (idiopathic facial paralysis). *Cochrane Database of Systematic Reviews*, Issue 2

House JW, Brackmann DE (1985) Facial nerve grading system. *Otolaryngol Head Neck Surg* **93**(2): 146–7

Murakami S, Mizobuchi M, Nakashiro Y, Doi T, Hato N, Yanagihara N (1996) Bell's palsy and herpes simplex virus: identification of viral DNA in endoneurial fluid and muscle. *Ann Intern Med* **124:** 27–30

Salinas RA, Alvarez G, Ferreira J (2004) Corticosteroids for Bell's palsy (idiopathic facial paralysis). *Cochrane Database of Systematic Reviews*, Issue 4

Sullivan F, Swan I, Donnan P et al (2007) Early treatment with prednisolone or acyclovir in Bell's palsy. *N Engl J Med* **357:** 1598–607

KEY POINTS

- Perform a full neurological and ear, nose and throat assessment.
- Bell's palsy is a diagnosis of exclusion.
- Facial nerve palsy, even if temporary, is disfiguring and patients should be counselled appropriately.
- Refer to an ear, nose and throat specialist for review after assessment and initiating primary management.