Causes and management of facial nerve palsy

Facial nerve palsy causes disfigurement with cosmetic, functional and psychological repercussions. The facial nerve can be affected anywhere along its course. A comprehensive assessment considering all differential diagnoses is critical to optimal management, as prompt, appropriate therapy leads to better outcomes.

Face to face contact is an essential component of human interaction. Facial nerve palsy is devastating and, without appropriate therapy, may result in permanent disfigurement. This review provides an overview of how to manage facial nerve palsy.

Injury to the facial nerve can occur anywhere along its course resulting in a sensorimotor deficit. The motor component predominates and facial nerve palsy causes significant morbidity including reduced emotional wellbeing and quality of life. Its diverse aetiology ranges from difficult delivery at birth (use of forceps) to neoplasia. The most common diagnosis is Bell's palsy, which is a diagnosis of exclusion. This review provides an overview of various causes of facial nerve palsy including their aetiology, pathogenesis, diagnosis, prognosis and therapy.

Applied anatomy of the facial nerve

The facial nerve is a mixed nerve consisting of both motor and sensory components. Its upper motor neurones originate as corticobulbar tracts, which traverse through the internal capsule to the pons, where they synapse at the facial nerve nucleus with crossover to the contralateral side. This explains the pathognomonic forehead sparing of upper motor neurone lesions seen in patients who have had a stroke as the forehead receives fibres from both hemispheres. In contrast, a lower motor neurone lesion affects the whole ipsilateral side. The lower motor neurone of the facial nerve has three parts: intracranial, intratemporal and extracranial.

Intracranial facial nerve

After leaving the pons (*Figure 1*), the facial nerve encircles the sixth cranial nerve (the abducent nerve) and exits the pons along with its sensory or parasympathetic root (nervus intermedius). An ipsilateral VI nerve palsy may suggest a pontine lesion. As it crosses the cerebellopontine angle and enters the fallopian canal via the internal acoustic meatus, the intracranial nerve becomes the intratemporal nerve.

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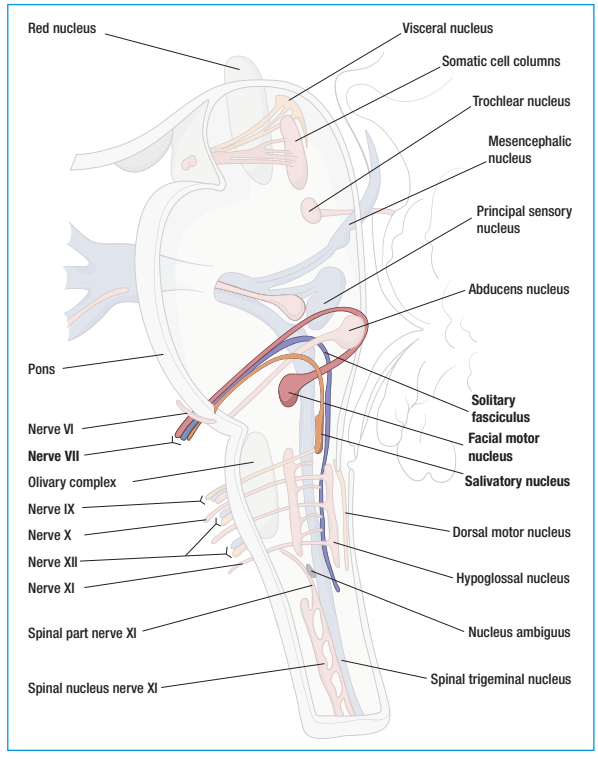
Intratemporal facial nerve

The fallopian canal has three segments: labyrinthine, tympanic and mastoidal. The labyrinthine segment courses from the internal acoustic meatus to the first genu where the geniculate ganglion is located. The greater superficial petrosal nerve and other fibres from the nervus intermedius emanate here (*Figure 2*). These supply the lacrimal glands and mucus glands of the nasal and oral cavities, explaining why lesions above this ganglion cause more severe ophthalmic symptoms (Portelinha et al, 2015). From the geniculate ganglion, the tympanic or horizontal segment of the facial nerve continues to the second genu along the medial wall of the tympanic cavity (*Figures 3a* and

Figure 1. The central tracts and brainstem site of facial nucleus and connections. Bold labels indicate components of the facial nerve.



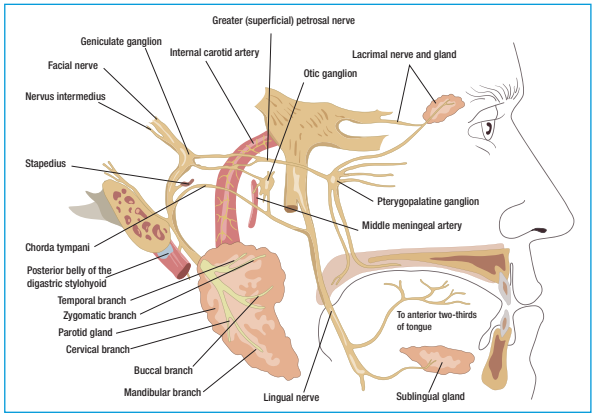


Figure 2. Intratemporal and extracranial course of the facial nerve.

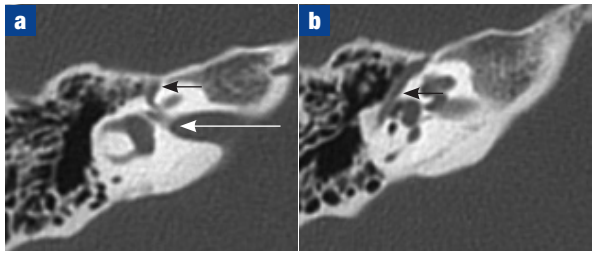


Figure 3. **a.** Here the facial nerve enters the internal acoustic meatus along with the cochlear and superior and inferior vestibular nerves (white arrow) The labyrinthine segment is the shortest and ends at the first genu (black arrow). **b.** From the first genu, the horizontal or tympanic segment of the facial nerve courses along the medial wall of the tympanic cavity (arrow).

b). The facial nerve descends vertically to the stylomastoid foramen, giving off branches to the stapedius muscle, chorda tympani and the sensory auricular branch.

Extracranial facial nerve

The facial nerve exits the stylomastoid foramen and runs through the parotid gland. It divides into five major branches to innervate the facial musculature: temporal, zygomatic, buccal, marginal mandibular and cervical.

Epidemiology

In neonates, the incidence of facial nerve palsy is reported as 0.6–1.8 per 1000 live births, mostly as a result of forceps delivery. In adults this ranges between 17 and 35 per 100 000.

Pathology

The aetiology of facial nerve palsy is diverse (*Table 1*). The most common causes are idiopathic, infective, trauma, iatrogenic and neoplasia (Siddiq et al, 2007; Hohman and Hadlock, 2014).

Idiopathic

Bell's palsy

Bell's palsy is a peripheral facial nerve palsy for which there is no detectable cause. It is essential to exclude other causes of facial nerve palsy before making a definitive diagnosis (Alaani et al, 2005).

Aetiology: The cause of idiopathic or Bell's palsy is incompletely understood, but there is some evidence that latent infection with herpes simplex type 1 plays a role in its aetiology (Murakami et al, 1996; Finsterer, 2008). Reactivation of the virus is postulated to lead to inflammation of the facial nerve, resulting initially in reversible neuropraxia and finally Wallerian degeneration (Holland and Weiner, 2004).

Epidemiology: The reported incidence of Bell's palsy ranges between 11 and 40 cases per 100 000 per year. Incidence peaks between the ages of 15–45 years (James,

Table 1. Causes of facial nerve palsy

Congenital	Möbius syndrome
	Goldenhar syndrome
	Melkersson–Rosenthal syndrome
Birth related	Forceps delivery
Idiopathic	Bell's palsy
Infection	Viral infection, i.e. varicella zoster (Ramsay Hunt), herpes zoster, herpes simplex, HIV
	Otitis media
	Cholesteatoma
	Necrotizing otitis externa
	Skull base osteomyelitis
	Lyme disease
Trauma	Temporal bone fracture
	Gunshot or penetrating injury
	Laceration
Neoplastic	Schwannoma
	Meningioma
	Haemangioma
	Parotid malignancy
Iatrogenic	Brain, middle ear, mastoid, parotid or facial surgery
Neurological	Lacunar or brainstem infarct
	Guillain–Barré syndrome
	Myasthenia gravis
	Multiple sclerosis
Metabolic	Diabetes mellitus
	Hypertension
	Pregnancy
	Vitamin A deficiency

Table 2. House–Brackman grading

Grade	Description
I	■ Normal symmetrical function in all areas
II	■ Slight weakness noticeable only on close inspection ■ Complete eye closure with minimal effort ■ Slight asymmetry of smile with maximal effort ■ Synkinesis barely noticeable, contracture or spasm absent
III	■ Obvious weakness but not disfiguring ■ May not be able to lift eyebrow ■ Complete eye closure with maximal effort ■ Strong but asymmetrical mouth movement with maximal effort ■ Obvious but not disfiguring synkinesis or hemifacial spasm
IV	■ Obvious disfiguring weakness ■ Inability to lift brow ■ Incomplete eye closure with maximal effort ■ Asymmetry of mouth with maximal effort ■ Severe synkinesis, hemifacial spasm, contracture
V	■ Barely perceptible motion ■ Incomplete eye closure ■ Slight movement of the corner of the mouth ■ Synkinesis, contracture and spasm usually absent
VI	■ No movement ■ Loss of tone ■ No synkinesis, contracture or spasm

From House and Brackmann (1985)

1996). Most studies report no sex or side preponderance, but an increased prevalence in pregnant women (Prescott, 1988; Holland and Weiner, 2004).

Clinical presentation: Bell's palsy presents with sudden onset of impairment of facial expression on one side. Bilateral facial nerve palsy accounts for 0.3–2% of cases of facial nerve palsy (Stahl and Ferit, 1989).

In Bell's palsy the facial nerve palsy progresses over a few hours to days. It is frequently preceded by periauricular paraesthesia or otalgia. This may be associated with dry eyes, xerostomia, dysguesia, tinnitus and hyperacusis.

In Bell's palsy the weakness involves half of the face, often with drooping of the eyebrow and corner of the mouth, and loss of the ipsilateral nasolabial fold. The House–Brackman grading score is used to assess the degree of facial nerve palsy. It objectively quantifies facial function to measure recovery and deterioration (*Table 2*).

The examination consists of a thorough assessment of the cranial nerve and head and neck including the parotid gland for masses. Otoscopy is performed to identify any vesicles around the ear suggestive of Ramsay Hunt syndrome or pus indicative of necrotizing otitis externa. Eye closure and corneal reflex are important in determining corneal risk. Regular topical lubricants are prescribed for day and night usage and eyes are taped at night. If problems occur with the eye referral to ophthalmology should be arranged.

Investigations: Pure tone audiometry is recommended when there is a hearing loss. Laboratory tests such as blood glucose or Lyme disease serology are obtained in atypical presentations. Electroneurography is of benefit to determine prognosis.

Treatment: For many years, Bell's palsy was treated with a combination of corticosteroids and antivirals. Sullivan et al (2007) demonstrated that early treatment with prednisolone alone significantly improved the chance of complete recovery. This was corroborated by similarly well-designed studies (Engström et al, 2008; Lockhart et al, 2009). In spite of the dissemination of these results, the proportion of untreated Bell's palsy cases was found to be only 40% in 2012 (Morales et al, 2013). Prednisolone should ideally be given within the first 24 hours at a dose of 1 mg/kg in adults to a maximum of 60 mg a day for 7 days (Gilden, 2004).

Prognosis: Most patients (85%) will have a partial recovery within 3–4 weeks and complete recovery within 6 months. Patients presenting with incomplete paresis have a 93–98% chance of complete recovery (Portelinha et al, 2015). Poor prognosis is indicated by complete paralysis, age over 60 years, minimal recovery by 3 weeks, pregnancy, nerve degeneration on electrophysiological testing, and underlying conditions such as diabetes mellitus.

Surgical decompression: While surgical decompression of the facial nerve may have a role in recurrent Bell's palsy (Doshi and Irving, 2010), in cases of acute facial paralysis its role is controversial (McAllister et al, 2011).

Imaging: Where recovery is not seen within 3 weeks, or the patient has spasm, other cranial nerve deficits or recurrence, a tumour must be ruled out using imaging. A combination of high resolution computed tomography scanning and magnetic resonance imaging, especially T1 weighted with gadolinium, is recommended.

Infection

Ramsay Hunt syndrome

Ramsay Hunt syndrome is caused by reactivation of the varicella zoster virus in the geniculate ganglion. It is characterized by a triad of facial nerve palsy, otalgia or vertigo, and vesicles in the ipsilateral external ear, palate or anterior tongue. It may be misdiagnosed as Bell's palsy in herpes zoster sine herpete as vesicles are absent in 2–35% of cases (Portelinha et al, 2015). The facial nerve palsy is more painful and more likely to be complete and associated with vestibulocochlear symptoms. Varicella zoster virus polymerase chain reaction distinguishes it from Bell's palsy. The treatment is steroids and antivirals. Prognosis is poor for these patients – fewer than 50% achieve complete recovery (Holland and Weiner, 2004).

Lyme disease

This is an uncommon cause of facial nerve palsy caused by the spirochaete *Borrelia burgdorferi*. It is transmitted by

tick bites in endemic areas. History taking should elicit any recent travel, outdoor activities and history of insect bites. Its incidence is higher in the summer. Its symptoms and signs include erythema migrans, joint pain, fever, fatigue or neck stiffness in association with unilateral or bilateral facial nerve palsy. Serology confirms the diagnosis. It is treated with doxycycline or amoxicillin for 2–3 weeks.

Acute otitis media

Facial nerve palsy secondary to otitis media is more common in young children. The majority of cases resolve with antibiotics. A myringotomy is indicated should the tympanic membrane not spontaneously perforate. This decompresses the middle ear and consequently the facial nerve. Occasionally a mastoidectomy is performed when there is lack of clinical improvement or a worsening palsy. Surgical decompression of the facial nerve is rarely indicated.

Cholesteatoma

Facial nerve palsy is a rare presenting feature of cholesteatoma. Its prevalence has decreased from 1–23% of cholesteatoma presentations in the 1950s to 0.04–0.16% these days owing to earlier diagnosis and treatment. When the disease is present in the petrous apex of the temporal bone, the incidence of facial nerve palsy rises to 50–81% (Siddiq et al, 2007). Early surgical decompression is indicated although improvement in facial nerve function can also be achieved when surgery is performed up to 7 months later.

Skull base osteomyelitis

This condition occurs in the elderly immunocompromised patient and is characterized by severe pain, aural discharge and progressive cranial neuropathies including facial nerve palsy. Diagnosis is based on imaging comprising computed tomography and magnetic resonance imaging and treatment is with long-term antibiotics.

Trauma

Temporal bone injury

Temporal bone fractures account for up to 22% of skull fractures. Road traffic accidents are the leading cause of temporal bone fractures and result in injury to the facial nerve in 31% of cases (Colbert et al, 2014).

Temporal bone fractures can lead to facial nerve palsy as well as conductive or sensorineural hearing loss, disequilibrium, vertigo and CSF leakage. Other signs are post-auricular bruising, Battle's sign and haemotympanum.

Management is based on the advanced trauma life support protocol. This is followed by neurological examination, otoscopy and assessment of facial nerve function and inner ear status (formal audiometric testing at the earliest opportunity). These fractures were traditionally classified into transverse, longitudinal or mixed with reference to the long axis of the temporal bone. However, as these did not often correlate well with clinical signs, 'otic-capsule sparing' and 'otic-capsule violating' have been suggested (Little and Kesser, 2006). The integrity of the nerve is evaluated by high resolution computed tomography.

Treatment is based on managing the facial nerve injury as well as the hearing loss, vestibular dysfunction and CSF leak. The decision to surgically explore the facial nerve in this context is based on a number of factors: penetrating injury, immediate onset of facial nerve palsy, a bony spicule on computed tomography, loss of inner ear function, persistent CSF leak and 90% or greater degeneration on electroneuronography at 10–14 days. Delayed onset or incomplete paresis usually recovers within 3 months and is treated with high dose corticosteroids, which are gradually tapered. In cases of persistent facial nerve palsy, facial reanimation and reinnervation techniques are considered after a period of observation (see below).

Iatrogenic

Temporomandibular joint replacement is the most common cause of iatrogenic injury to the facial nerve according to Hohman and Hadlock (2014). The tympanic segment of the facial nerve may be injured during middle ear surgery as it may be dehiscent or its canal eroded by cholesteatoma. The facial nerve is also at risk after surgery in the cerebellopontine angle where facial nerve palsy may be associated with decreased lacrimation and V-VIII cranial nerve palsy. Another common cause of iatrogenic injury is surgery to the parotid gland.

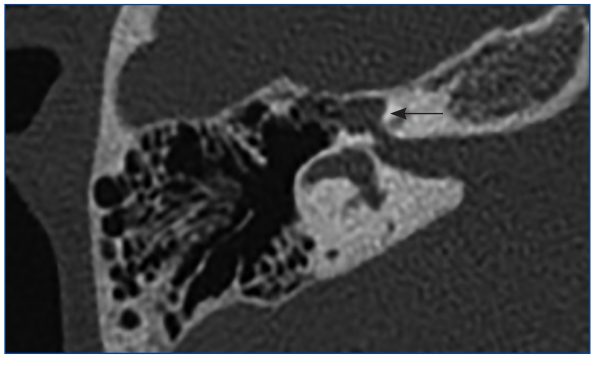
Iatrogenic injury should be repaired at the time of surgery or as soon as it is noticed, if possible within a few days. Corticosteroids should be administered if onset is delayed. Electroneurography is useful in determining prognosis. Patients with persistent palsy are initially observed before consideration of facial reanimation.

Neoplasia

Facial nerve tumours

Facial nerve tumours within the temporal bone account for 0.8% of all head and neck tumours. The most common intrinsic tumours are schwannomas (*Figure 4*), which cause up to 5% of cases of facial nerve palsy (Watson and Irving, 2015). Haemangiomas, the second most common intrinsic tumour, usually arise from the geniculate ganglion (Salib et al, 2001).

Figure 4. Axial computed tomography scan demonstrating a schwannoma (arrow) involving the geniculate ganglion. (Compare with *Figure 3a*.)



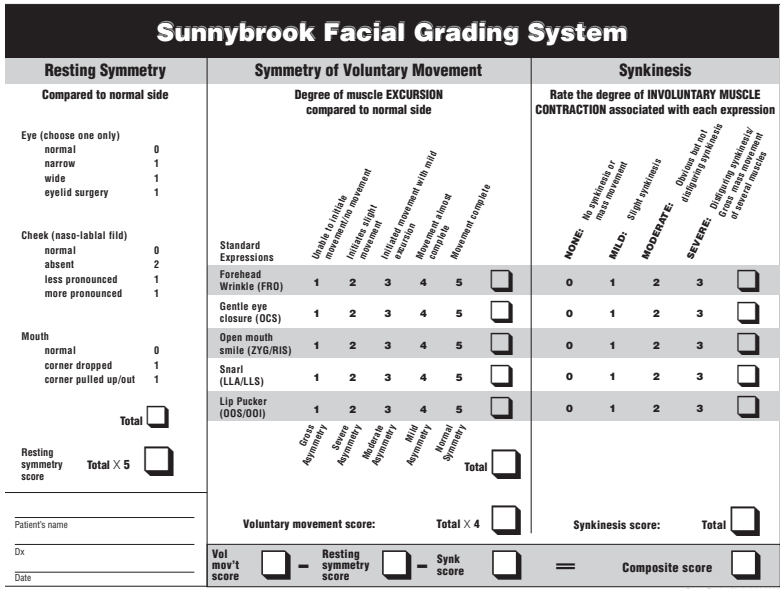
Secondary spread from malignant parotid tumours is also possible because of their propensity for perineural invasion. Symptoms vary depending on the site of the lesion on the facial nerve. Tumours involving the vertical or horizontal segment of the nerve may present with conductive hearing loss and a mass noted on otoscopy. Owing to inner ear erosion, those at the labyrinthine segment may cause sensorineural hearing loss, tinnitus and vertigo.

Suspicion is aroused by a history of facial spasm or twitching, a progressive weakness over a number of weeks, recurrent or slowly resolving facial nerve palsy. The facial nerve is assessed and graded according to the House–Brackman grading. A cranial nerve examination is indicated as well as otoscopy, assessment of cerebellar function, head and neck palpation, and pure tone audiometry. These tumours either involve multiple cranial nerves or the facial nerve in isolation. Both high resolution computed tomography scanning of the temporal bone and T1-weighted gadolinium-enhanced magnetic resonance imaging of the brain and skull base are arranged to visualize the entire nerve (Watson and Irving, 2015). Inadequate imaging may result in delayed or inappropriate diagnoses (Alaani et al, 2005).

Referral to a dedicated unit with adequate expertise in these lesions is advocated. Patients are discussed in a skull base multidisciplinary team meeting, followed by a review in a dedicated facial nerve palsy clinic.

Patients who are able to close their eyes and are stable are managed conservatively with observation, serial imaging and annual electromyography and/or electroneurography. Patients with House–Brackman grade III who have clinical deterioration and those with House–Brackman grade IV–VI are offered surgery. The size and position of the tumour as well as residual hearing should be considered when deciding on the surgical approach. A transmastoid and/or middle cranial fossa approach is recommended for patients with good hearing and a translabyrinthine approach for those with poor hearing.

Figure 5. The Sunnybrook facial grading system is used in the facial palsy clinic to monitor progress. It assesses the face across three domains: resting symmetry, symmetry of voluntary movement and degree of synkinesis.



Facial therapy and reanimation

In spite of optimal initial treatment, some patients do not recover their facial nerve function. This is estimated to occur in up to 30% of patients with idiopathic facial nerve palsy (Watson et al, 2015). In addition to the visible deformity, these patients also suffer with functional problems relating to lagophthalmos (inability to close the eyelids properly), oral continence and speech. This has an impact on their psychology and quality of life. A number of treatment modalities is aimed at improving their cosmetic appearance and function.

Facial therapy

The authors' institute holds a multidisciplinary clinic for patients with facial nerve palsy. Facial symmetry and tone are assessed using the Sunnybrook facial grading scale (*Figure 5*). The flexibility of the facial musculature is recorded and photographic evidence of facial expression collected to monitor progress. Initial therapy focuses on massage, which ameliorates mobility and circulation. Patients are also taught eyelid and brow stretching techniques, and are encouraged to tape their eyes on a regular basis. They are given individualized therapy dependent on the specific muscle groups identified as inactive. Neuromuscular retraining using sound productions such as 'ow' are used to encourage the action of a particular muscle group, in this case depressor anguli oris and depressor labii inferioris. Watson et al (2015) showed that this improved facial grading scores.

Facial reanimation

Early referral for reanimation is essential to achieve good results as muscle units are unlikely to survive beyond 18–24 months after nerve injury. The viability of the facial musculature determines the surgical approach.

Facial musculature viable

The best results in facial muscle recovery following iatrogenic injury, temporal bone fracture or oncological resection occur with early primary neurorrhaphy with accurate end-to-end anastomosis. If the defect is too big to achieve a tension-free repair, interposition grafts, most commonly from the auricular or sural nerves, can be used. These are preferred because of their desirable axon content, length and availability. Cross-facial nerve grafts encourage symmetrical movement. Donor branches of nerve from the normal side are used and rerouted to the paralysed side.

Facial musculature non-viable

Patients whose onset of paralysis predates 24 months have muscle units that are non-viable. Facial reanimation is achieved either by muscle transposition or free muscle

importation. Muscle transposition uses regional muscles such as masseter and temporalis. The masseter is used to animate the perioral area while the temporalis can also be used to achieve eyelid closure. Free muscle transfer is a two-stage procedure: the first stage is the positioning of the cross-facial nerve graft and the second, 9–12 months later, the free muscle transfer using gracilis, latissimus dorsi or pectoralis minor.

Static procedures

Static procedures achieve resting symmetry rather than dynamic symmetry. They are used in the correction of abnormal postures, lagophthalmos and oral continence. They can be used as sole therapy or as an adjunct to the aforementioned reanimation techniques. Lagophthalmos can result in corneal ulceration and blindness. Gold and platinum weights can be inserted to the upper lid to treat this. Lower lid repositioning can be used to correct or avoid a lower lid ectropion. Botulinum toxin injections can also be considered in cases to alleviate spasm or synkinesis. These have been found to achieve a good cosmetic outcome.

Conclusions

While Bell's palsy is the most common cause of facial nerve palsy, it is important to consider other pathology to avoid misdiagnosis. This review provides an overview of the diverse aetiology of facial nerve palsy and management thereof. It is imperative to treat the patient holistically and remember the impact on psyche and quality of life that facial disfigurement may impose. **BJHM**

Figure 5 *is reproduced by kind permission of Sunnybrook Health Sciences Centre, Toronto and Dr J Nedzelski.*
Conflict of interest: none.

KEY POINTS

- The facial nerve is an important cranial nerve with motor and sensory components.
- Facial nerve palsy is a devastating event with cosmetic, functional and psychological repercussions.
- There are many causes of facial palsy ranging from idiopathic to malignant tumours.
- It is important to optimally assess all facial nerve palsies to avoid misdiagnosing a Bell's palsy.
- Sparing of the forehead muscles suggests a cerebrovascular accident and not a lower motor neurone weakness.
- Recurrent unilateral weakness, slow onset of weakness, poor recovery and presence of facial twitch or spasm are all signs suggesting more serious underlying pathology and should prompt urgent referral.
- Most facial nerve palsies fully recover, but those that do not should be referred to a facial palsy clinic.
- Facial reanimation, botulinum toxin injections, gold weights and facial physiotherapy can achieve good outcomes for those with incomplete recovery.
- Early referral significantly improves outcomes as rehabilitative treatment carries a better outcome if the facial muscles are still viable, which is typically up to 18 months following the onset of paralysis.
- The psychological wellbeing of the patient should also be addressed.

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