

IN BRIEF

- Most cases of altered sensation are related to trauma.
- Facial palsy is often due to Bell's palsy.
- However, many disorders of orofacial sensation and movement can be an indicator of serious underlying disease.



Oral Medicine – Update for the dental practitioner

Disorders of orofacial sensation and movement

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This series provides an overview of current thinking in the more relevant areas of oral medicine for primary care practitioners, written by the authors while they were holding the Presidencies of the European Association for Oral Medicine and the British Society for Oral Medicine, respectively. A book containing additional material will be published. The series gives the detail necessary to assist the primary dental clinical team caring for patients with oral complaints that may be seen in general dental practice. Space precludes inclusion of illustrations of uncommon or rare disorders, or discussion of disorders affecting the hard tissues. Approaching the subject mainly by the symptomatic approach – as it largely relates to the presenting complaint – was considered to be a more helpful approach for GPs rather than taking a diagnostic category approach. The clinical aspects of the relevant disorders are discussed, including a brief overview of the aetiology, detail on the clinical features and how the diagnosis is made. Guidance on management and when to refer is also provided, along with relevant websites which offer further detail.

ORAL MEDICINE

1. Aphthous and other common ulcers
2. Mouth ulcers of more serious connotation
3. Dry mouth and disorders of salivation
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10. Orofacial pain

Sensory innervation of the mouth, face and scalp depends on the fifth cranial (trigeminal) nerve, so that lesions affecting this nerve can cause sensory loss or orofacial pain, or indeed both – sometimes with serious implications.

The facial (seventh cranial) nerve controls the muscles of facial expression, so that lesions of this nerve (lower motor neurone lesions) or its central connections (upper motor neurone lesions), can lead to facial weakness. The facial nerve also carries nerve impulses to the tear glands, to the salivary glands, and to the stapedius muscle of the stirrup bone (the stapes) in the middle ear and also transmits taste from the anterior tongue, so that lesions may also affect taste and hearing, lacrimation and salivation.

It is evident therefore that dental surgeons should be able to carry out examination of these and other cranial nerves (Table 1), as follows.

THE OLFACTORY NERVE (1st CRANIAL NERVE)

Bilateral anosmia is common after head injuries, but in practice the patient may complain of loss of taste rather than sense of smell. Unilateral anosmia is often unnoticed by the patient.

An olfactory lesion is confirmed by inability to smell substances such as orange or peppermint oil. Ammoniacal solutions or other substances with a pungent odour must not be used since they stimulate the trigeminal rather than the olfactory nerve.

THE OPTIC NERVE (IInd CRANIAL NERVE)

Blindness or defects of visual fields are caused by ocular, optic nerve or cortical damage but the

type of defect varies according to the site and extent of the lesion.

If there is a complete lesion of one optic nerve, that eye is totally blind and there is no direct reaction of the pupil to light (loss of constriction). If a light is shone into the affected eye, the pupil of the unaffected eye also fails to respond (loss of the consensual reflex). However, the nerves to the affected eye that are responsible for pupil constriction, run in the IIIrd cranial nerve and should be intact. If, therefore, a light is shone into the unaffected eye, the pupil of the affected eye also constricts even though it is sightless.

Lesions of the optic tract, chiasma, radiation or optic cortex cause various defects involving both visual fields but without total field loss on either side.

An ophthalmological opinion should always be obtained if there is any suggestion of a visual field defect.

THE OCULOMOTOR NERVE (IIIrd CRANIAL NERVE)

The oculomotor nerve supplies the muscle that raises the upper eyelid, most of the orbital muscles that move the eye (except the lateral rectus and superior oblique), and the ciliary muscle and pupil constrictor.

Normally the medial rectus (supplied by the IIIrd nerve) moves the eye medially (adducts). The lateral rectus (VIth nerve) abducts the eye. When the eye is abducted it is elevated by the superior rectus (IIIrd nerve) and depressed by the inferior rectus (IIIrd nerve). The adducted eye is depressed

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Table 1 Examination of cranial nerves

Nerve	Examination	Examination findings in lesions
I	Olfactory	Sense of smell Impaired sense of smell for common odours (do not use ammonia)
II	Optic	Visual acuity Visual fields Pupil responses Visual acuity reduced using Snellen types ± ophthalmoscopy: nystagmus. Visual fields by confrontation impaired; may be impaired pupil responses
III	Oculomotor	Eye movements Pupil responses Diplopia; strabismus; eye looks down and laterally; movements impaired; ptosis; pupil dilated Pupil reactions: direct reflex impaired but consensual reflex intact
IV	Trochlear	Eye movements Pupil responses Diplopia, particularly on looking down; strabismus; no ptosis; pupil normal and normal reactivity
V	Trigeminal	Sensation over face Corneal reflex Jaw jerk Taste sensation Reduced sensation over face; ± corneal reflex impaired; ± taste sensation impaired; motor power of masticatory muscles reduced, with weakness on opening jaw; jaw jerk impaired; muscle wasting
VI	Abducens	Eye movements Pupil responses Diplopia; strabismus; eye movements impaired to affected side; pupil normal and normal reactivity
VII	Facial	Motor power of facial muscles Corneal reflex Taste sensation Impaired motor power of facial muscles on smiling, blowing out cheeks, showing teeth, etc; corneal reflex reduced; ± taste sensation impaired
VIII	Vestibulo-cochlear	Tuning fork at 256 Hz Impaired hearing; impaired balance; ± nystagmus
IX	Glossopharyngeal	Gag reflex Taste sensation Voice Reduced gag reflex; deviation of uvula; reduced taste sensation; voice may have nasal tone
X	Vagal	Gag reflex Voice Reduced gag reflex; deviation of palate; voice hoarse
XI	Accessory	Ability to shrug shoulders and rotate head against resistance Motor power of trapezius and sternomastoid reduced
XII	Hypoglossal	Tongue protrusion Motor power of tongue impaired, with abnormal speech; ± fasciculation, wasting, ipsilateral deviation on protrusion



Fig. 1 Herpes zoster, palate

by the superior oblique muscle (IVth nerve) and elevated by the inferior oblique (IIIrd nerve).

Disruption of the oculomotor nerve therefore causes:

1. Ptosis (drooping upper eyelid).
2. Double vision and divergent squint. The affected eye points downwards and laterally—‘down and out’ in all directions except when looking towards the affected side.
3. Paralysis of internal, upward and downward rotation of the eye.
4. A dilated pupil which fails to constrict on accommodation or when light is shone either onto the affected eye (negative direct light

reaction) or into the unaffected eye (negative consensual light reaction).

THE TROCHLEAR NERVE (IVth CRANIAL NERVE)

The trochlear nerve supplies only the superior oblique muscle which moves the eye downwards and medially towards the nose.

The lesion is characterised by:

1. The head tilted away from the affected side.
2. Diplopia, maximal on looking downwards and inwards.
3. Normal pupils.

There is often damage to the IIIrd and VIth nerves as well.

Damage to the trochlear nerve causes serious disability, because there is diplopia maximal on looking down and the patient may therefore have difficulty reading, going down stairs or seeing obstructions on the ground.

THE TRIGEMINAL NERVE (Vth CRANIAL NERVE)

The trigeminal nerve supplies sensation over the whole face apart from the angle of the jaw, and the front of the scalp back to a line drawn across the vertex, between the ears. It also supplies sensation to the mucosa of the oral cavity, conjunctivae, nose, tympanic membrane and sinuses.

The motor division of the trigeminal nerve supplies the muscles of mastication (masseter, pterygoids, temporalis, mylohyoid and anterior belly of the digastric).

Taste fibres from the anterior two-thirds of the tongue, and secretomotor fibres to the submandibular and sublingual salivary glands and lacrimal glands, are also carried in branches of the trigeminal nerve.

Damage to a sensory branch of the trigeminal nerve causes hypoaesthesia in its area of distribution; infection such as with herpes zoster causes pain (Fig. 1). Lesions of the sensory part of the trigeminal nerve initially result in a diminishing response to pin-prick to the skin and, later, complete anaesthesia. Lesions involving the ophthalmic division also cause corneal anaesthesia: this is tested by gently touching the cornea with a wisp of cotton wool twisted to a point. Normally this procedure causes a blink, but not if the cornea is anaesthetised (and the patient does not see the cotton wool).

It is important, with patients complaining of facial anaesthesia, to test all areas but particularly the corneal reflex, and the reaction to pin-prick over the angle of the mandible.

If, however, the patient complains of complete facial or hemifacial anaesthesia, but the corneal reflex is retained or there is apparent anaesthesia over the angle of the mandible, then the symptoms are probably functional rather than organic.

Taste can be tested with sweet, salt, sour or bitter substances (sugar, salt, lemon juice or vinegar) carefully applied to the dorsum of the tongue.

Damage to the motor part of the trigeminal nerve can be difficult to detect and is usually asymptomatic if unilateral but the jaw may

deviate towards the affected side on opening. It is easier to detect motor weakness by asking the patient to open the jaw against resistance, rather than by trying to test the strength of closure.

THE ABDUCENS NERVE (VIth CRANIAL NERVE)

The abducens nerve supplies only one eye muscle, the lateral rectus. Lesions comprise:

1. Deviation of the affected eye towards the nose
2. Paralysis of abduction of the eye.
3. Convergent squint with diplopia maximal on looking laterally towards the affected side.
4. Normal pupils.

Lesions of the abducens can, however, be surprisingly disabling.

THE FACIAL NERVE (VIIth CRANIAL NERVE)

The facial nerve carries:

- The motor supply to the muscles of facial expression
- Taste sensation from the anterior two-thirds of the tongue (via the chorda tympani)
- Secretomotor fibres to the submandibular and sublingual salivary glands
- Secretomotor fibres to the lacrimal glands
- Branches to the stapedius muscle in the middle ear.

The neurones supplying the lower face receive upper motor neurones (UMN) from the contralateral motor cortex, whereas the neurones to the upper face receive bilateral UMN innervation.

An UMN lesion therefore causes unilateral facial palsy with some sparing of the frontalis and orbicularis oculi muscles because of the bilateral cortical representation. Furthermore, although voluntary facial movements are impaired, the face may still move with emotional responses, for example on laughing. Paresis of the ipsilateral arm (monoparesis) or arm and leg (hemiparesis), or dysphasia may be associated because of more extensive cerebrocortical damage.

Lower motor neurone (LMN) facial palsy is characterised by unilateral paralysis of all muscles of facial expression for both voluntary and emotional responses (Fig. 2). The forehead is unfurrowed and the patient unable to close the eye on that side. Attempted closure causes the eye to roll upwards (Bell's sign). Tears tend to overflow on to the cheek (epiphora), the corner of the mouth droops and the nasolabial fold is obliterated. Saliva may dribble from the commissure and may cause angular stomatitis. Food collects in the vestibule and plaque accumulates on the teeth on the affected side. Depending on the site of the lesion, other defects such as loss of taste or hyperacusis may be associated.

In facial palsy, facial weakness is demonstrated by asking the patient to:

- Close the eyes against resistance
- Raise the eyebrows
- Raise the lips to show the teeth
- Try to whistle.



Fig. 2
Facial nerve palsy

Full neurological examination is needed, looking particularly for signs suggesting a central lesion, such as:

- Hemiparesis
- Tremor
- Loss of balance
- Involvement of the Vth, VIth or VIIIth cranial nerves.

The following investigations may be indicated:

- Imaging with MRI, or CT, of the internal auditory meatus, cerebellopontine angle and mastoid may be needed to exclude an organic lesion such as a tumour – particularly in progressive facial palsy
- Study of evoked potentials to assess the degree of nerve damage. Facial nerve stimulation or needle electromyography may be useful, as may electrogustometry, nerve excitability tests, electromyography and electroneuronography
- Blood pressure measurement (to exclude hypertension)
- Blood tests that may include:
 - Fasting blood sugar levels (to exclude diabetes)
 - Tests for HSV or other virus infections such as HIV may need to be considered
 - Serum angiotensin converting enzyme levels as a screen for sarcoidosis
 - Serum antinuclear antibodies to exclude connective tissue disease
 - In some areas, Lyme disease (tick-borne infection with *Borrelia burgdorferii*) should be excluded by ELISA test.
- Schirmer's test for lacrimation, carried out by gently placing a strip of filter paper on the lower conjunctival sac and comparing the wetting of the paper with that on the other side
- Test for loss of hearing
- Test for taste loss by applying sugar, salt, lemon juice or vinegar on the tongue and asking the patient to identify each of them
- Aural examination for discharge and other signs of middle ear disease
- Blood pressure measurement (to exclude hypertension)
- Lumbar puncture occasionally.

THE VESTIBULOCOCHLEAR NERVE (VIIIth CRANIAL NERVE)

The auditory nerve has two components:

- The vestibular (concerned with appreciation of the movements and position of the head)
- The cochlear (hearing).

Lesions of this nerve may cause loss of hearing, vertigo or ringing in the ears (tinnitus).

An otological opinion should be obtained if a lesion of the vestibulocochlear nerve is suspected, as special tests are needed for diagnosis.

THE GLOSSOPHARYNGEAL NERVE (IXth CRANIAL NERVE)

The glossopharyngeal nerve carries:

- The sensory supply to the posterior third of the tongue and pharynx
- Taste sensation from the posterior third of the tongue
- Motor supply to the stylopharyngeus
- Secretomotor fibres to the parotid.

Symptoms resulting from a IXth nerve lesion include impaired pharyngeal sensation so that the gag reflex may be weakened; the two sides should always be compared. Lesions of the glossopharyngeal are usually associated with lesions of the vagus, accessory and hypoglossal nerves (bulbar palsy).

THE VAGUS NERVE (Xth CRANIAL NERVE)

The vagus has a wide parasympathetic distribution to the viscera of the thorax and upper abdomen but is also the motor supply to some soft palate, pharyngeal and laryngeal muscles.

Lesions of the vagus are rare in isolation but have the following effects:

1. Impaired gag reflex.
2. The soft palate moves towards the unaffected side when the patient is asked to say 'ah'.
3. Hoarse voice.
4. Bovine cough.

THE ACCESSORY NERVE (XIth CRANIAL NERVE)

The accessory nerve is the motor supply to the sternomastoid and trapezius muscles. Lesions are often associated with damage to the IXth and Xth nerves and cause:

1. Weakness of the sternomastoid (weakness on turning the head away from the affected side).
2. Weakness of the trapezius on shrugging the shoulders.

Testing this nerve is useful in differentiating patients with genuine palsies from those with functional complaints. In an accessory nerve lesion there is weakness on turning the head away from the affected side. Those shamming paralysis often simulate weakness when turning the head towards the 'affected' side.

THE HYPOGLOSSAL NERVE (XIIth CRANIAL NERVE)

The hypoglossal nerve is the motor supply to the

muscles of the tongue. Lesions cause:

1. Dysarthria (difficulty in speaking)
 - particularly for lingual sounds.
2. Deviation of the tongue towards the affected side, on protrusion.

The hypoglossal nerve may be affected in its intra- or extracranial course. Intracranial lesions typically cause bulbar palsy. In an upper motor neurone lesion the tongue is spastic but not wasted; in a lower motor neurone lesion there is wasting and fibrillation of the affected side of the tongue.

FACIAL SENSORY LOSS

Normal facial sensation is important to protect the skin, oral mucosa and especially cornea from damage. Lesions developing and affecting the sensory part of the trigeminal nerve initially result in a diminishing response to light touch (cotton wool) and pin-prick (gently pricking the skin with a sterile pin or needle without drawing blood) and, later there is complete anaesthesia. Facial sensory awareness may be:

- Completely lost (anaesthesia) or
- Partially lost (hypoesthesia).

The term paraesthesia does not mean loss of sensation, rather it means abnormal sensation.

Lesions of a sensory branch of the trigeminal nerve may cause anaesthesia in the distribution of the affected branch. Facial sensory loss may be caused by intracranial or, more frequently, by extracranial lesions of the trigeminal nerve and may lead to corneal, facial or oral ulceration (Table 2).

If the patient complains of complete facial or hemifacial anaesthesia, but the corneal reflex is retained then the symptoms are probably functional (non-organic) or due to benign trigeminal neuropathy. If the patient complains of complete facial or hemifacial anaesthesia and there is apparent anaesthesia over the angle of the mandible (an area not innervated by the trigeminal nerve) then the symptoms are almost certainly functional (non-organic).

Extracranial causes of sensory loss

Extracranial causes of facial sensory loss include damage to the trigeminal nerve from:

- Trauma, the usual cause
- Osteomyelitis and
- Malignant disease.

Common extracranial causes of facial sensory loss are shown in Table 2. The mandibular division or its branches may be traumatised by inferior alveolar local analgesic injections, fractures or surgery (particularly surgical extraction of lower third molars or osteotomies). Occasionally there is dehiscence of the mental foramen in an atrophic mandible leading to anaesthesia of the lower lip on the affected side, as a result of pressure from the denture. The inferior alveolar or lingual nerves may be damaged, especially during removal of lower third molars, or arising

Table 2 Causes of sensory loss in the trigeminal area**Extracranial**

Trauma (eg surgical; fractures) to inferior dental, lingual, mental or infraorbital nerves

Inflammatory

- Osteomyelitis

Neoplastic

- carcinoma of antrum or nasopharynx
- metastatic tumours
- leukaemic deposits

Intracranial

Trauma (eg surgical; fractures or surgical treatment of trigeminal neuralgia)

Inflammatory

- multiple sclerosis
- neurosyphilis
- HIV infection
- sarcoidosis

Neoplastic

- cerebral tumours

Syringobulbia

Vascular

- cerebrovascular disease
- aneurysms

Drugs

- Labetalol

Bone disease

- Pagets disease

Benign trigeminal neuropathy

Idiopathic

Psychogenic

Hysteria

- Hyperventilation syndrome

Organic disease

close by. Osteomyelitis or tumour deposits in the mandible may affect the inferior alveolar nerve to cause labial anaesthesia.

Nasopharyngeal carcinomas may invade the pharyngeal wall to infiltrate the mandibular division of the trigeminal nerve, causing pain and sensory loss and, by occluding the Eustachian tube, deafness (Trotter's syndrome).

Damage to branches of the maxillary division of the trigeminal may be caused by trauma (middle-third facial fractures) or a tumour such as carcinoma of the maxillary antrum.

Intracranial causes of facial sensory loss

Intracranial causes of sensory loss are uncommon but serious and include:

- Multiple sclerosis
- Brain tumours
- Syringobulbia
- Sarcoidosis
- Infections (eg HIV).

Since other cranial nerves are anatomically close, there may be associated neurological deficits. In posterior fossa lesions for example, there may be cerebellar features such as ataxia. In middle cranial fossa lesions, there may be associated neurological deficits affecting cra-

nial nerve VI (abducent nerve), resulting in impaired lateral movement of the eye.

Benign trigeminal neuropathy

This is a transient sensory loss in one or more divisions of the trigeminal nerve which seldom occurs until the second decade. The corneal reflex is not affected. The aetiology is unknown, though some patients prove to have a connective tissue disorder.

Psychogenic causes of facial sensory loss

Hysteria, and particularly hyperventilation syndrome, may underlie some causes of facial anaesthesia.

Organic causes of facial sensory loss

These include diabetes or connective tissue disorders.

Diagnosis in facial sensory loss

In view of the potential seriousness of facial sensory loss, care should be taken to exclude local causes and a full neurological assessment must be undertaken. Since, in the case of posterior or middle cranial fossa lesions, other cranial nerves are anatomically close, there may be associated neurological deficits. Thus in the absence of any obvious local cause, or if there are additional neurological deficits, patients should be referred for a specialist opinion.

Management of patients with facial sensory loss

If the cornea is anaesthetic, a protective eye pad should be worn and a tarsorrhaphy (an operation to unite the upper and lower eyelids) may be indicated since the protective corneal reflex is lost and the cornea may be traumatised.

OROFACIAL MOVEMENT DISORDERS

The facial nerve not only carries nerve impulses to the muscles of the face, but also to the tear glands, to the saliva glands, to the lacrimal glands and to the stapedius muscle of the stirrup bone (the stapes) in the middle ear. It also transmits taste from the anterior tongue. Since the function of the facial nerve is so complex, several symptoms or signs may occur if it is disrupted.

The main movement disorder is facial palsy, which can have a range of causes (Table 3), and may be due to UMN or LMN lesions, as discussed above.

The common cause of facial palsy is stroke, an UMN, and this is a medical emergency for which specialist care is indicated. The GDP should be able to differentiate UMN from LMN lesions (see above and Table 4).

The facial nerve should be tested, by examining facial movements and other functions mediated by the nerve. Movement of the mouth as the patient speaks is important, especially when they allow themselves the luxury of some emotional expression. The upper part of the face is bilaterally innervated and thus loss of wrinkles on one-half of the forehead or absence of blinking suggest a lesion is in the lower motor neurone.

**Key points for patients:
Bell's palsy**

- This is fairly common
- It affects only the facial nerve; there are no brain or other neurological problems
- It may be caused by herpes simplex virus, or other infections
- It is not contagious
- There are usually no serious long-term consequences
- X-rays and blood tests may be required
- Treatment takes time and patience; corticosteroids and antivirals can help
- Most patients recover completely within three months
- It rarely recurs

**Key points for dentists:
Bell's palsy**

- This is fairly common
- It affects only the facial nerve
- It may be caused by herpes simplex virus, or other infections
- It is not contagious
- It disproportionately attacks pregnant women and people who have diabetes, hypertension, influenza, a cold, or immune problems
- There are usually no serious long-term consequences
- Corticosteroids and antivirals can help
- Most patients begin to get significantly better within two weeks, and about 80% recover completely within three months
- It rarely recurs, but can in 5–10%

Table 3 Causes of facial palsy

Upper motor neurone lesion	
• Cerebrovascular accident	
• Trauma	
• Tumour	
• Infection	
• Multiple sclerosis	
Lower motor neurone lesion	
• Systemic infection	
	Bell's palsy (herpes simplex virus usually)
	Varicella-Zoster virus infection (+/- Ramsay-Hunt syndrome)
	Lyme disease (<i>B.burgdorferii</i>)
	HIV infection
• Middle ear disease	
	Otitis media
	Cholesteatoma
• Lesion of skull base	
	Fracture
	Infection
• Parotid lesion	
	Tumour
	Trauma to branch of facial nerve

Table 4 Differentiation of upper (UMN) from lower motor neurone (LMN) lesions of the facial nerve

	UMN lesions	LMN lesions
Emotional movements of face	Retained	Lost
Blink reflex	Retained	Lost
Ability to wrinkle forehead	Retained	Lost
Drooling from commissure	Uncommon	Common
Lacrimation, taste or hearing	Unaffected	May be affected

If the patient is asked to close their eyes any palsy may become obvious, with the affected eyelids failing to close and the globe turning up so that only the white of the eye is showing (Bell's sign). Weakness of orbicularis muscles with sufficient strength to close the eyes can be compared with the normal side by asking the patient to close his eyes tight and observing the degree of force required to part the eyelids. If the patient is asked to wrinkle their forehead, weakness can be detected by the difference between the two sides.

Lower face (round the mouth) movements are best examined by asking the patient to:

- Smile
- Bare the teeth
- Purse the lips
- Blow out the cheeks
- Whistle.

Corneal reflex

This depends on the integrity of the trigeminal and facial nerve, either of which being defective will give a negative response. It is important to test facial light touch sensation in all areas but particularly the corneal reflex. Lesions involving the ophthalmic division cause corneal anaesthesia, which is tested by gently touching the

cornea with a wisp of cotton wool twisted to a point. Normally, this procedure causes a blink but, if the cornea is anaesthetic (or if there is facial palsy), no blink follows, provided that the patient does not actually see the cotton wool.

Taste

Unilateral loss of taste associated with facial palsy indicates that the facial nerve is damaged proximal to the chorda tympani.

Hearing

Hyperacusis may be caused by paralysis of the stapedius muscle and this suggests the lesion is proximal to the nerve to stapedius.

Lacrimation

This is tested by hooking a strip of Schirmer or litmus paper in the lower conjunctival fornix. The strip should dampen to at least 15 mm in one minute if tear production is normal. The contralateral eye serves as a control (Schirmer's test). Secretion is diminished in proximal lesions of the facial nerve, such as those involving the geniculate ganglion or in the internal auditory meatus.

BELL'S PALSY

Bell's palsy is the most common acute LMN paralysis (palsy) of the face. There is inflammation of the facial nerve which may be immunologically mediated and associated with infection, commonly herpes simplex virus (HSV), leading to demyelination and oedema, usually in the stylomastoid canal.

The condition is usually seen in young adults; predisposing factors, found in a minority of cases, include:

- Pregnancy
- Hypertension
- Diabetes or
- Lymphoma.

Aetiopathogenesis

LMN facial palsy is usually associated with infections mainly with herpes simplex virus (HSV), rarely, another virus such as:

- Varicella-Zoster virus (VZV) infection
- Epstein-Barr virus (EBV) infection
- Cytomegalovirus (CMV) infection
- Human herpesvirus-6 infection
- HIV infection;

occasionally with bacterial infections such as:

- Otitis media
- Lyme disease (infection with *Borrelia burgdorferii*).

Clinical features

Damage to the facial nerve may result in twitching, weakness, or paralysis of the face, in dryness of the eye or the mouth, or in disturbance of taste.

There is:

- Acute onset of paralysis over a few hours, maximal within 48 hours.
- Paralysis of upper and lower face, usually only unilaterally.

- Diminished blinking and the absence of tearing. These together result in corneal drying, which can lead to erosion, and ulceration and the possible loss of the eye.

Occasionally:

- Pain around the ear or jaw may precede the palsy by a day or two.
- There may be apparent facial numbness, but sensation is actually intact on testing.

If the lesion is located proximal to the stylo-mastoid canal, there may also be (Table 5):

- Hyperacusis (raised hearing sensitivity; loss of function of nerve to stapedius), or
- Loss of taste (loss of function of the chorda tympani) and/or
- Loss of lacrimation.

Up to 10% of patients have a positive family history and a similar percentage suffers recurrent episodes.

Diagnosis of Bell's palsy

The history should be directed to exclude facial palsy caused by other factors, such as:

- Stroke
- Trauma to the facial nerve (eg in parotid region or to base of skull) or by underwater diving (barotrauma)
- Facial nerve tumours (eg acoustic neuroma)
- Facial nerve inflammatory disorders
- Multiple sclerosis
- Connective tissue disease
- Sarcoidosis
- Melkersson-Rosenthal syndrome
- Infections
 - Viral
 - HSV
 - VZV
 - EBV
 - CMV
 - HIV infection
 - HTLV-1 infection (rare-Japanese and Afro-Caribbean patients mainly).
 - Bacterial
 - Middle ear infections (eg otitis media)
 - Lyme disease (from camping or walking in areas that may contain deer ticks that transmit *Borrelia burgdorferii*).

The examination and investigations are discussed above.

Management of Bell's palsy

Treatment with systemic corticosteroids results in 80-90% complete recovery. There is

Table 5 Localisation of site of lesion in and causes of unilateral facial palsy

Muscles paralysed	Lacrimation	Hyperacusis	Sense of taste	Other features	Probable site of lesion	Type of lesion
Lower face	N	-	N	Emotional movement retained + monoparesis or hemiparesis + aphasia	Upper motor neurone (UMN)	Stroke (cerebrovascular accident) Brain tumour Trauma HIV infection
All facial muscles	-	+	-	+ Vth nerve damage	Lower motor neurone (LMN) Facial nucleus	Multiple sclerosis
All facial muscles	-	+	-	+ VIIIth nerve damage	Between nucleus and geniculate ganglion	Fractured base of skull Posterior cranial fossa tumours Sarcoidosis
All facial muscles	N	+	N or ⁻	-	Between geniculate ganglion and stylomastoid canal	Otitis media Cholesteatoma Mastoiditis
All facial muscles	N	-	N	-	In stylomastoid canal or extracranially	Bell's palsy Trauma Local analgesia (eg misplaced inferior dental block) Parotid malignant neoplasm Guillain-Barre syndrome
Isolated facial muscles	N	-	N	-	Branch of facial nerve extracranially	Trauma Local analgesia

N = normal + = present ⁻ = reduced

thus a strong argument for treating all patients with prednisolone 20mg four times a day for five days, then tailing off over the succeeding four days.

Since HSV is frequently implicated, antivirals are also justifiably used. The combination of oral aciclovir 400mg five times daily with oral prednisolone 1mg/kg daily for 10 days is more effective than corticosteroids alone.

WEBSITES AND PATIENT INFORMATION

<http://www.entnet.org/bells.html>

http://www.ninds.nih.gov/health_and_medical/disorders/bells_doc.htm

<http://www.bellsalsy.ws/>

Patients to refer

Any patient with a cranial nerve defect as further investigation is outwith the scope of primary dental care