

The management of Bell's palsy

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Bell's palsy is the most common diagnosis given to patients with acute facial palsy. The annual incidence of Bell's palsy is 15-40 per 100 000 population. There is an equal male to female ratio and a 3.3 times greater incidence in pregnant females. Familial inheritance has been found in 4-14% of cases with a recurrence rate of 10-12%. The left and right sides are equally involved and less than 1% of cases are bilateral. It is also considered to be four to five times more likely to occur in diabetics than the general population.

Pathogenesis

Approximately 80% of adults carry the herpes simplex virus in the ganglia of cranial and spinal nerves. Once the virus infiltrates the ganglion, it resides in the nucleus of the ganglion and becomes dormant. Reactivation leads to a polyganglionitis. Causes of reactivation could be preceded by trauma, environmental factors and metabolic or emotional disorders, thus suggesting that emotional, environmental (e.g. cold) and physical stress (e.g. trauma) may lead to reactivation.

Once reactivation takes place, the virus migrates proximally to cause a localised meningo-encephalitis at the brainstem and distally to cause mucocutaneous vesicles. After the virus exits the neural cell membrane, neurilipoprotein deposition leads to an immune mediated reaction and subsequent demyelination. An understanding of the pathogenesis quantifies the clinical signs and symptoms of Bell's palsy.

Diagnosis

Bell's palsy is a diagnosis of exclusion, by elimination of other reasonable possibilities (see Table 1). If dysgeusia and post-auricular pain occurs, it can only be a viral origin.

Taking a proper history is the most important step in the diagnosis of a patient with any form of cranial neuritis. From the outset, one must look for evidence of a benign disease. The criteria for benignity are acute onset, transient duration, lack of progression and clinically complete resolution of the inflammation.

Patients should be questioned regarding previous episodes, family history, associated symptoms (hearing loss, otorrhoea, otalgia, vertigo, headaches, blurred vision, paraesthesia), associated medical illnesses (diabetes, pregnancy, autoimmune disorders, cancer), history of trauma (recent or remote) and previous surgery (otologic, rhytidectomy, parotidectomy).

A complete head and neck examination must be performed, including examination of the ears, careful palpation of the parotid glands and a thorough neurological examination. It is important to assess the degree of voluntary movement present to document the grade of facial paralysis as described in the House Brackmann classification system (see Table 2).

It is unreliable to ask the patient to move his/her face and/or close his/her eyes. The normal moving side will cause movements on the affected side if the facial muscles are not pressed firmly against the skull in order to prevent movements over the midline (which can be surprisingly strong, because the patient tries his/her best to move the face) (see Figure 1).

Symptomatology

- Post-auricular pain
- Taste disturbance (dysgeusia)
- Hypersensitivity to touch (somatophobia)
- Hypoaesthesia of the cheek and decreased corneal reflex
- Reduced gag reflex
- Hypersensitivity to loud sounds (hyperacusis)
- Epiphora
- Vertigo
- Reduced tearing with xerophthalmia

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TABLE I: AETIOLOGY OF FACIAL PARALYSIS

Congenital	
	Möbius syndrome
	Congenital lower lip paralysis
	Melkerson-Rosenthal syndrome
	Sclerosing bone dysplasias (Marblebone disease)
Traumatic	
	Temporal bone fractures
	Birth trauma
	Facial lacerations
	Penetrating wounds to the face or temporal bone
Infection	
	Bell's palsy
	Ramsay Hunt Syndrome
Acute Otitis Media	
	Otitis Media with effusion
	Malignant Otitis externa
	Tuberculosis
	Syphilis
	Lyme disease
	Acquired immunodeficiency syndrome
	Infectious mononucleosis
Neoplasia	
	Facial nerve neuroma
Cholesteatoma	
	Acoustic neuroma
	Meningioma
	Glomus jugulare
	Parotid tumours
	Leukaemia
	Hemangioblastoma
	Rhabdomyosarcoma
Metabolic/Systemic	
	Diabetes mellitus
	Hyperthyroidism
	Pregnancy
	Auto-immune disorders
Neurological	
	Multiple sclerosis
	Guillain-Barré Syndrome

The observer should use four fingers on the forehead and the thumb on the nasion. The same method is used to observe movements to close the eye. For the mouth, use one or two fingers press on the upper lip against the teeth, and two fingers immobilise the lower lip.

On trying to close the eye, one will observe that the eyeball tilts upwards (Bell's phenomenon). This tilting of the eyeball must be kept in mind when examining for a return of function of the muscles which close the eye. One should then immobilise the lower eyelid with one finger and ask the patient to close the eye. If there is any real movement of the muscles below the eye, one will feel the muscles moving.

Any patient presenting with facial paralysis should undergo audiological testing, including pure tone, air and bone conduction, speech discrimination, acoustic reflexes, and tympanometry. If asymmetry is found on the audiogram, further tests need to be done.

Special investigations

Radiological tests

The need for radiological evaluation is based on the history and clinical course of each individual case. If radiological imaging is deemed necessary, high-resolution computed tomography (CT) is the study of choice for evaluation of traumatic seventh cranial nerve paralysis. In SA, ordinary x-rays of the skull need to be done to rule out sclerosing bone dysplasias (sclerosteosis, marble bone disease).

Magnetic resonance imaging (MRI) with gadolinium is the test of choice for facial nerve paralysis secondary to inflammation, neoplastic and other non-traumatic cases. Imaging is not used routinely for the diagnosis of Bell's palsy.

Bell's palsy is highly likely to be caused by Herpes simplex type 4

Grade	Degree	Description
I	Normal	Normal facial movements; no synkinesis
II	Slight	Mild deformity, mild synkinesis, good forehead function, slight asymmetry
III	Moderate	Obvious facial weakness, forehead motion present, good eye closure, asymmetry, Bell's phenomenon present
IV	Moderately	Obvious weakness, increasing synkinesis; no forehead motion
V	Severe	Very obvious facial paralysis, some tone present, cannot close eyes
VI	Total	Complete facial paralysis, absent tone

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Figure 1. Positioning of fingers during examination

Prognostic tests

Topographic tests

Although these tests are of historical interest, they have not been found to be of much use clinically for determining the site of the lesion in facial paralysis or for predicting the outcome (see Table III).

Lacrimation (Schirmer's Test)

This test evaluates the greater superficial petrosal nerve function (i.e. tear production) by determining the rate and amount of tear production.

Stapedial reflex

The stapedius muscle contracts reflexively when the ear is stimulated with a loud tone. This alters the reactive compliance of the middle ear, which can be measured. If the lesion involves the nerve proximally to the stapedius branch, the stapedial reflex will be absent

Salivary flow testing

By cannulising the submandibular gland duct orifice (Wharton's papillae), a measurement of salivary flow to gustatory stimulation can be obtained. This test is difficult to perform and is subject to a significant level of inaccuracy.

Electro gustometry

The tongue is stimulated electrically to produce a metallic taste and the two sides of the tongue are compared.

Electro diagnostic tests

Nerve stimulation tests using surface electrodes are used. It is recommended that the first stimulation test be done on day two or three. The MST, ENOG and VENOg tests will be discussed in this article. Patient participation is good because when the worried patient feels the muscles contract on stimulation, it is an immense relief. Both patient and doctor also gain confidence in managing the condition because prognostication is accurate.

Maximal stimulation test (MST)

For this method, the Hilger-type stimulator is used with the ground electrode on the back of the patient's hand. The observer is situated so as to see both sides of the face simultaneously. The stimulus is measured in milliamperes (mA).

The frontal, orbital and mandibular branches of the facial nerve are stimulated. The stimulating probe is applied to the nerve branch at an intensity that produces a just visible twitch. When the first contraction is observed, the area is explored to find the most sensitive point - that which displays the maximum amount of muscle motion. The current is then increased by 1-2mA above this threshold to obtain supramaximal stimulation.

Test results are expressed as the difference in facial muscle movement observed when comparing the affected side of the face with the normal side. If decreased movement is present, the observer notes whether this decrease is minimal, moderate or severe compared to the non-affected side. A scale of 0 to 4 is used, where 0 indicates no response, 1 is a severely decreased response, 2 is a moderate reduction, 3 is a minimal decrease and 4 is a normal response. A score of 4 is normal and 0 is total degeneration.

Electroneurography (ENOG)

An electromyogram is used. The stimulating probes are placed at the stylomastoid foramen and the recording surface electrodes on the muscles to be tested. Supramaximal stimulation is applied to the nerve trunk and the compound muscle action potentials of the muscles are measured (normal values are from 2.0 to 8.0mV). Several stimulations are given and the placement of the recording probe can be moved in order to determine the site of maximum response (optimised lead placement is the nasolabial fold and the cheek over the zygomatic muscles).

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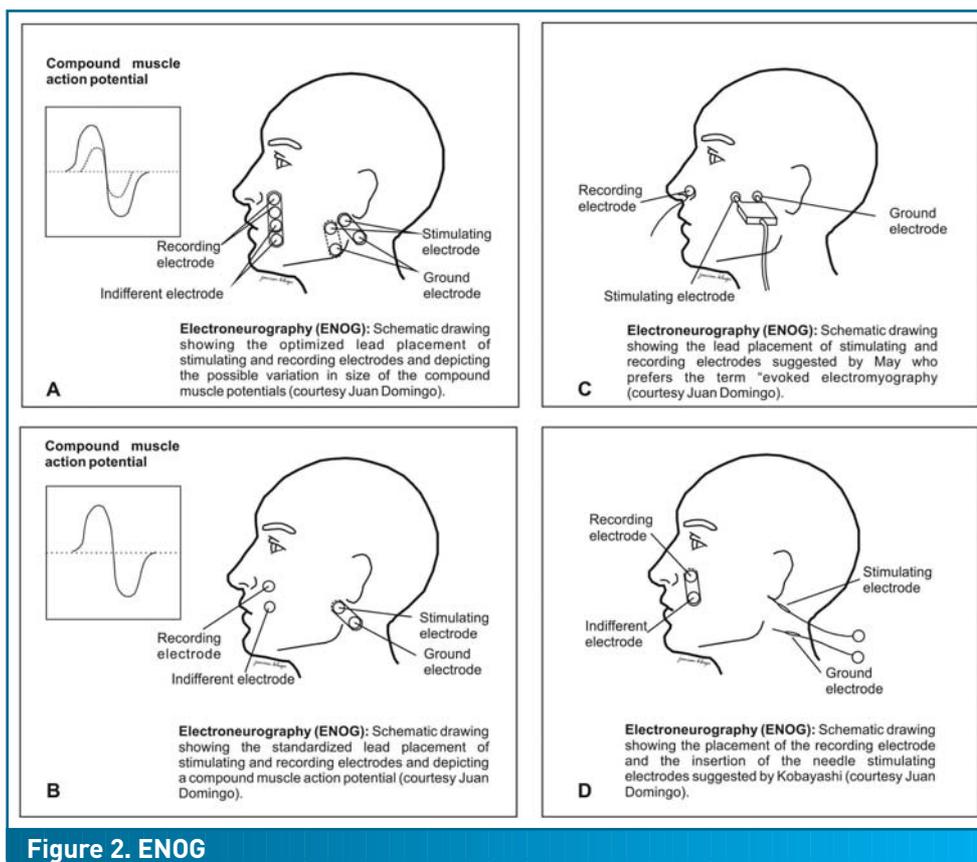


Figure 2. ENOG

ENOG is supposed to have the advantage of recording the extent of nerve degeneration more accurately than one can do with visual observation. When 90% degeneration is measured, this is regarded as critical and surgical decompression may be considered (see Figure 2)

Visual ENOG

A variation of the ENOG test is to observe the muscle contractions produced at the same time as measuring the compound action potentials. Additional information is obtained and this test can be called 'visual ENOG'. This method of testing is especially suitable for use with the new small nerve stimulators. If the evoked muscle contractions drop to 50% of normal, the patient can be referred for ENOG testing.

Management

Medical treatment: Every facial palsy should be regarded as an emergency. Prednisone treatment should be started immediately by the referring doctor and not delayed until a specialist is seen. When requesting an appointment with a specialist, the urgency should be stressed and the specialist should see the patient within 1-2 days.

Management consists of the following:

1. **Systemic steroids:** Prednisone 1mg/kg in daily divided doses is prescribed initially for a duration of five days for both complete and incomplete palsies.

If the paralysis remains incomplete (paresis) the dose is tapered to stop during the next six days. Improvement of function usually starts from day 10-14, and movement should be normal by day 28.

If the paralysis is complete, the 1mg/kg/day is continued up to day 16, and then tapered to stop during the next five days (total 21days). Prednisone is discontinued after day 21, even though return of facial function may not yet have taken place. Prednisone should never be stopped abruptly because rebound inflammation can lead to further denervation.

Prednisone treatment should be started immediately

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If the nerve does not degenerate totally (as determined by the electrical tests), some function will start from days 16-20, and normal movement is possible by day 42. If partial movement stops progressing by day 42, the rest will return by 12-14 weeks, and the final result will be good (more than 90% movement) with insignificant synkinesis.

In the case of total degeneration (as determined by electrical tests), function will start returning after 12-16 weeks, and no more than 70-80% movement can be expected (provided prednisone had been given, otherwise less movement can be expected). However sequelae will always occur.

2. **Antivirals:** Valacyclovir or famciclovir 500mg tds is recommended during the first week of the disease.
3. **Eye care:** Because the eye does not close, natural tears or a tear gel (e.g. Celluvisc) should be prescribed.
4. **Analgesics:** Although the prednisone usually helps to reduce the pain, liberal use of analgesics may be necessary.
5. **Serial electrodiagnostic testing**
6. **Decompression surgery:** Considerable difference of opinion still exists regarding surgery. The consensus is that, if surgery is performed, the proximal (labyrinthine) segment of the nerve should be decompressed via a middle fossae craniotomy approach. Recent studies done by Ganz (2007) revealed that 90% of patients receiving facial nerve decompressive surgery within 14 days of the onset of the facial nerve paralysis with 90% facial nerve degeneration, recovered to a House Brackmann score of 1 or 2. Comparative control groups that were managed non-surgically ended up with a House Brackmann score of 3 or 4. Unfortunately, the surgery carries a small risk of complications, as is the case in all surgical interventions.

After excluding other potential causes of a facial paralysis, treatment is medical

7. **Physiotherapy:** Most facial nerve researchers do not recommend physiotherapy treatment. The argument that muscle atrophy during the time of total paralysis is prevented does not apply to the facial muscles. Experience gained with patients who needed nerve grafts many months after the onset of the paralysis have shown that excellent movement recurs, even after many months of inactivity of the muscle, by utilising physiotherapy.

Conclusion

Bell's palsy is the most common cause of an acute facial palsy. It is unilateral and sudden in onset, often with a prodrome of post-auricular pain. After excluding other potential causes of a facial paralysis, treatment is medical. Surgery is reserved for those who meet electrodiagnostic criteria.

Fortunately, 85% of patients with Bell's palsy have full return of facial function but it is important to regard each facial palsy as an emergency. If there is no return of facial movement within four months after the onset of Bell's palsy, the diagnosis is wrong.

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