ABC of ENT

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FACIAL PALSY

Two-thirds or more of cases of lower motor neurone facial palsy are idiopathic. But idiopathic (or Bell's) palsy should be diagnosed only after a full investigation and after no other explanation can be found.

Intracranial causes of lower motor neurone facial palsy include neoplasia, multiple sclerosis, brain-stem infarction, cerebellopontine angle lesions, and poliomyelitis. Intratemporal causes include trauma from an accident or surgery, otitis media (especially chronic with cholesteatoma) herpes zoster oticus (Ramsey-Hunt syndrome), and carcinoma of the middle ear or neurofibroma of the facial nerve. The commonest extracranial causes are trauma and carcinoma of the parotid gland. Other causes include infectious mononucleosis, sarcoidosis, and polyneuritis.

For a full diagnosis the doctor needs to identify the site of the lesion, its cause, and its severity.

Clinical assessment

Recognising facial palsy depends on carefully examining the face during voluntary and involuntary (emotional) movement. The amount of weakness in the forehead, eyelids, nostrils, and mouth should be assessed. An unaffected forehead and paralysis on voluntary but not involuntary movement suggests an upper motor neurone lesion, which demands neurological examination.

The ear drums must be carefully examined. Cholesteatoma hidden by a crust over the outer attic wall is easily overlooked, but if it is suspected the patient must be referred urgently for surgical exploration. Chronic destructive middle ear disease should be suspected if the patient has discharge, deafness, or symptoms of other complications such as vertigo. Inspection of the pinna and ear canal may show the rash and blisters typical of zoster infection in the Ramsey-Hunt syndrome, in which paralysis and rash is preceded by pain.

Assessing the integrity of branches of the facial nerve requires examination of lacrimation and taste.

Lacrimation is easily tested by Schirmer's test. A folded strip of sterile filter paper is hung on the lower eyelids and the rate of lacrimation along each strip compared.

Taste on the anterior two-thirds of the tongue is harder to test, and the patient's observations about disturbed taste at the time of onset of facial paralysis are often more useful than tests. The tongue should be dried with a swab and salt or sugar deposited on the anterior two-thirds and the patient asked to comment.

Radiographs of the temporal bone are necessary if the cause of the palsy is not apparent.

Site of the lesion

Branches of the facial nerve affected by paralysis will suggest the site of the lesion in the nerve. From the geniculate ganglion in the petrous temporal bone the great superficial petrosal nerve carries fibres to innervate the lacrimal gland. Just before it leaves the temporal bone (above the stylomastoid foramen) the facial nerve is joined by the chorda tympani, which carries sensory fibres from taste endings on the anterior two-thirds of the tongue (plus secretory motor fibres for submandibular and anterior lingual salivary glands). Slightly above this a small branch supplies motor fibres to the stapedius muscle.

A lesion in the brain stem will produce only weakness of the facial muscle with no loss of taste or lacrimation. There are usually other neurological signs such as palsy of the VIth nerve, which causes diplopia due to weakness of the lateral rectus muscle.

Loss of lacrimation and taste from the anterior two-thirds of the tongue suggests a lesion between the brain stem and geniculate ganglion.

Facial weakness with loss of taste but normal lacrimation indicates a lesion between the geniculate ganglion and the origin of the chorda tympani.

If both taste and lacrimation are preserved the lesion is peripheral to the stylomastoid foramen. There are no other cranial nerve defects and the lesion is usually obvious.

If the nerve has been affected by injury or disease this is usually apparent from the history or clinical examination—for example aural discharge. In rare cases surgical exploration may be necessary to establish the diagnosis. In most cases, however, if the facial paralysis is of sudden onset and there is no apparent explanation on clinical or x-ray examination it is Bell's palsy.

Severity of lesion

It is important to discover whether the nerve has suffered damage from which it will recover (neuropraxia) or more severe damage that will require regeneration for partial recovery (neuronotmesis or axonotmesis). Severity is usually assessed accurately by electrodiagnostic tests, but neuropraxia may be assumed unless one side of the face is completely paralysed.

If a nerve is suddenly damaged, for example, by injury that divides it, no electrical test will show any abnormality until the peripheral part of the nerve has degenerated to the region where the test stimulus is applied. This distal degeneration will take from three to five days to reach the stylomastoid foramen, where nerve conduction tests are performed. Indeed, if the tests are normal one week after onset degeneration has not occurred even though clinically the face may be completely paralysed.

Treatment

The aims of treatment of any facial palsy are to treat any lesion, alleviate the disability, and provide the nerve with the best chance of recovery.

The cornea of the eye on the affected side must be protected. An eye cover or spectacle wing may be adequate for a short period. But if recovery is expected to take months a lateral partial tarsorrhaphy is advisable.

Facial appearance may be improved by lifting the corner of the mouth with a hook or filling out the cheek with a plumper, both of which can be attached to an upper denture. Massage or electrical stimulation of the paralysed muscle does not prevent atrophy or encourage recovery but it may boost the patient's morale.

Help for the nerve depends on whether the fibres remain in continuity or whether they have been severed and their ends separated. If some voluntary movement remains, or if electrical responses remain normal for a long period, continuity is certain. If total degeneration is indicated by electrical tests and there is any doubt about the continuity as after injury or chronic middle ear disease, surgical exploration is necessary to determine the state of the nerve. At operation breaches of continuity may be mended by rerouting the facial nerve within its temporal course or by inserting a nerve graft taken from the lateral cutaneous nerve of the thigh or the sural or the great auricular nerve.

Treatment of Bell's palsy

Contraindications to steroids	
Hypertension	Pulmonary tuberculosis
Pregnancy	Peptic ulcer
Diabetes	Middle ear infection

Bell's palsy is possibly due to ischaemia of the nerve within its bony canal. The value of systemic steroids is controversial but there is enough evidence in their favour to warrant their use when there are no contraindications such as hypertension, pregnancy, diabetes, pulmonary tuberculosis, peptic ulcer, or middle ear infection. Prednisolone 25 mg four times a day should be started as soon as possible after the onset of palsy, continued for five days, and tailed off gradually over a further five days.

If total degeneration has occurred in Bell's palsy there may be a case for surgical decompression to allow the oedematous contents within the sheath to swell.

Prognosis

The prognosis depends on whether the nerve has degenerated. If there is no degeneration (neuropraxia) recovery is always complete provided the cause is controlled. If there is degeneration recovery will depend on whether the nerve is in continuity. If it is, regeneration can occur, but it will take many months and the paralysis will recover only partially, with associated movements, contractures, and sometimes involuntary clonic spasm.

In Bell's palsy 90% of patients recover fully. Seventy-five per cent suffer neuropraxia and can expect normal movement to return within three weeks of onset. Of the remainder (those who suffer degeneration) about half face the prospect of slow and incomplete recovery. The extent of neural damage is usually set at onset but a few patients experience further deterioration during the first week or so. Repeat attacks are less likely to resolve than initial ones.

Treatment of failed degeneration

If function has not recovered after a year and electrical tests indicate a dead nerve the choice of treatment lies between plastic surgery to raise the paralysed side of the face with a facial sling or an operation such as anastomosis between the hypoglossal and facial nerves. Faciohypoglossal anastomosis generally produces better results than plastic surgery but it cannot be performed after the muscle fibres have atrophied—a process that usually takes two or three years.

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