Some Aspects of Facial Nerve Paralysis

PART I. INTRODUCTION, AETIOLOGY, APPLIED ANATOMY AND TOPOGNOSIS, AND DEGREE OF PARALYSIS *

M. G. POTGIETER,† M.B. CH.B. UNIV. PRET., M.MED. OTOL. UNIV. CAPE TOWN, *Department of Otolaryngology, Groote Schuur Hospital, Observatory, Cape*

SUMMARY

Paralysis of the facial nerve can cause a great deal of anxiety and discomfort to the patient, as it is intimately involved in emotional expression. A lesion anywhere on its entire course can usually be pinpointed and this is important in the treatment and prognosis. A uniform way of expressing the degree of facial paralysis is suggested.

S. Afr. Med. J., **47**, 27 (1973).

Paralysis of the facial nerve is, in fact, a medical emergency for the doctor, and a source of considerable discomfort for the patient. It deprives the patient of voluntary and involuntary facial movement and causes the loss of facial expression. A patient whose face is disfigured by paralysis, suffers psychological alarm. There is considerable discomfort when eating and drinking, speech impediment, and epiphora. In 1821 Sir Charles Bell demonstrated that the facial nerve is the motor nerve of the face. Although he described facial palsy due to gunshot wounds, tumours, syphilis, and even goring by an ox, the term Bell's palsy has come to be restricted to an idiopathic paralysis of the facial nerve.

There are many known causes of facial palsy. In some, the prognosis is excellent, in others not. Neurologists and other doctors in many centres continue to teach watchful expectancy, and when a patient is finally referred for consultation it may be too late. It has been the general opinion that facial paralysis following head trauma,[1] and especially after a free interval, has always a good prognosis and will eventually heal.

This attitude of 'wait and see and hope for the best' is to be deplored. Every case of facial palsy should be assessed as soon as possible by every means available to decide on treatment and the ultimate prognosis.

Laumans and Jongkees[2] classify facial paralysis as follows:

1. Cases exclusively suitable for conservative treatment, e.g. aural herpes zoster, and leutic neuritis.
2. Cases to be treated exclusively and as early as possible in a radical manner (surgery or radiotherapy), e.g. chronic otitis media; intratemporal tumours; after surgery to the temporal bone and of immediate onset; fractures of the temporal bone with paralysis of immediate onset; and external violence to the middle ear or mastoid bone.
3. Cases for which there is no fixed plan of therapy, e.g. Bell's palsy; acute otitis media; external otitis; facial paralysis of delayed onset after operation to the temporal bone; and fractures of the temporal bone with paralysis of late onset.

There is hardly any difference of opinion as regards treatment of facial paralysis due to causes mentioned in groups 1 and 2. The situation is entirely different for the cases in group 3. The absence of a fixed rule of approach is due to a lack of reliable tests; in the vital early stages of facial paralysis it seems impossible to distinguish cases which are unlikely to show complete recovery. There are, also, no accepted standardized criteria for determining the type and degree of paralysis before and after treatment; without such criteria, methods of therapy are difficult to evaluate.

THE AETIOLOGY OF FACIAL PARALYSIS

The most common cause of peripheral facial paralysis is a lesion within the temporal bone. Cawthorne[3] reported 347 cases of facial palsy, 93% of temporal bone origin.

Every effort should be made to determine the aetiology of facial paralysis. If the cause of the paralysis is recognized and treated (e.g. myasthenia gravis, sarcoidosis, neoplasms, and infection of the temporal bone), the progression of the disease and the paralysis may be changed.

Differential Diagnosis of Facial Palsy

At birth: due to moulding forceps delivery; dystrophia myotonica; and the Möbius's syndrome.

McHugh,[5] in a series of 18 139 consecutive births, found facial paralysis in 41 newborn infants, or 2,3 per 1 000 live births. Facial palsy at birth must be differentiated from agenesis of facial muscles.

Trauma: fractures of the base of the skull; facial injuries; penetrating injury of middle ear; and altitude paralysis.

Neurologic causes: Landry-Guillain-Barré ascending paralysis; multiple sclerosis; myasthenia gravis; opercular syndrome (cortical lesions in the facial motor area); Millard-Gubler syndrome (facial nerve and abducens nerve paralyses with contralateral hemiplegia due to

*Date received: 10 April 1972.

†Present address: 15 Medical Centre, 331 Burger Street, Pietermaritzburg, Natal.

lesions in the base of the pons, invading the corticospinal tract.)

Infection: external otitis, otitis media, especially tuberculosis; mastoiditis; chicken pox; Hunt's syndrome (herpes zoster may be extremely difficult to diagnose as the cause of facial paralysis. A single vesicle on the eardrum lasting only 24 hours, may easily be missed); encephalitis; poliomyelitis; mumps; infectious mononucleosis; leprosy; influenza; coxsackie virus infection; malaria; syphilis; and sarcoidosis (Heerfordts' syndrome).

Metabolic causes: diabetes mellitus; hyperthyroidism; and pregnancy as related to Bell's palsy.

Neoplastic causes: cholesteatoma; acoustic neuromas (Cawthorne[3,6] examined more than 200 patients with acoustic neuromas and found complete facial paralysis in only 2); tumours of the glomus jugulare; leukaemic infiltration; meningioma; haemangioblastoma; sarcoma; carcinoma (invasive or metastatic); an anomalous sigmoid sinus; haemangioma of the tympanum; hydradenoma (external canal); osteopetrositis; cylindroma; teratoma; Hand-Schüller-Christian disease; ependymoma of facial colliculus; and Wegner's granulomatosis.

Toxic causes: thalidomide toxicity (Miehlke's syndrome); tetanus; and diphtheria.

Iatrogenic causes: mandibular block anaesthesia; administration of antitetanus serum; vaccine treatment for rabies; after polio immunization; parotid surgery; mastoid surgery; and after tonsillectomy and adenoidectomy.

Idiopathic causes: Bell's palsy; Melkersson's syndrome; familial; and Möbius's syndrome.

In spite of all the known causes of facial paralysis, two-thirds of all cases still fall in the class of idiopathic or Bell's palsy. According to Jongkees,[1] 81% of his cases were Bell's. The figures for Cawthorne and Haynes[7] are 62%, and for Laumans,[8] 52%.

Alter[9] noted that almost 30% of his patients with Bell's palsy had a close relative with the same condition. In addition, 25% of his cases were hypertensive; 5% developed Bell's palsy during pregnancy, and at least 5% were diabetics.

Cawthorne[3] noticed that in nearly every case of Bell's palsy the mastoid was well pneumatized.

Most authors today attribute Bell's palsy to ischaemia of the facial nerve. Jongkees agrees with Hilger[10] that Bell's palsy may be caused by arteriolar spasm with ischaemia. The oedema compresses the nerve and obstructs the lympatic and venous circulations. In this way a vicious circle is created. Psychosomatic factors may also be involved.

Microscopic examination[11] of the facial nerve in Bell's palsy shows oedema, dilated capillaries, degeneration of the medullary sheaths and axis cylinders. Later, there is degeneration of the Schwann sheaths, and finally complete fibrosis of the nerve. The most important factor is the complete absence of inflammatory cells. The recent trend is to accept that the initial change is an interstitial oedema of the nerve and to regard all cellular infiltration as part of the reparative process.

Blunt[12] has shown that only 2 arteries supplying the vertical part of the facial nerve enter the bony canal (at the genu and stylomastoid foramen) between the bone and the sheath, and that they give off branches which enter perpendicularly into the nerve. The veins do not enter perpendicularly, but travel for some distance between the nerve and its sheath. Any minor swelling may compress the veins at this point. In 1943, Denny-Brown[13] of Boston showed that nerve fibres when deprived of oxygen rapidly lose their ability to conduct impulses. Ischaemia of the nerve can play a role in producing a paralysis of delayed onset after surgical intervention, and after head injury and acute otitis media. Facial paralysis of delayed onset after head injury can arise from a circulatory disturbance in the nerve from pressure exerted by loose bone fragments, from a haematoma at the fracture lines; dehiscences in the Fallopian canal; swelling of the mastoid mucosa; oedema secondary to infection; haemorrhage in the nerve sheath; or haemorrhage in the nerve itself.

It is important to know that facial palsy may arise simply from manipulation of the facial nerve, or pulling on the chorda tympani.

Dietzel examined 211 temporal bones microscopically and found dehiscences of the Fallopian canal in 57%. Their typical situations are: over the geniculate ganglion; above the oval window; and in relation to the retrofacial air cells.

APPLIED ANATOMY AND TOPOGNOSIS OF FACIAL NERVE LESIONS

Although lesions along its entire length can cause facial paralysis, of special interest to the aural surgeon is the part of the facial nerve distal to its entrance into the internal auditory meatus. It is essential to pinpoint the site of compression in order to decide which portion of the nerve to decompress. In theory, this sounds reasonable, but it has its limitations because some functions may be affected more than others, or may recover earlier than others. Jongkees[1] especially stresses that in late cases of facial palsy one should not rely heavily on the methods of topognosis.

Jepsen[14] repeatedly examined the stapedial function and taste after facial palsy and often found that these functions returned earlier than the motor function of the facial muscles. In a few cases, a complete return of stapedial function and taste were not followed by a complete return of function of the facial muscles.

Embryologically, the facial nerve belongs to the second branchial arch, and contains 4 different types of fibres:

(i) special visceral efferent fibres from the facial nucleus supplying striated muscle;

(ii) general visceral efferent fibres to the lacrimal glands, submaxillary and sublingual salivary glands, and glands of the oral cavity and nose. The superior salivatory nucleus is the site of origin of these fibres;

(iii) special visceral afferent fibres conveying taste impulses to the tractus solitarius with their neuronal cells in the geniculate ganglion;

(iv) somatic afferent fibres with their neuronal cells in the geniculate ganglion. These fibres are concerned with deep sensibility from the facial muscles, and sensory impulses from part of the tympanic membrane, the external auditory meatus, the tympanic cavity, and nasal mucosa. It is thought that these sensory fibres end in the descending nucleus of the brachial.

The motor nucleus of the facial nerve[23] gives rise to the motor fibres of the facial nerve. The motor neurons for the lower face are situated dorsally, and those for the upper face, in the ventral part of the nucleus. The corticobulbar tracts to the upper face are crossed and uncrossed, whereas the tracts to the lower face are only crossed, so that the upper face is bilaterally innervated.

Small lesions in the motor nucleus may paralyse some of the muscles of facial expression, and leave others intact. This may be seen in bulbar poliomyelitis, acute epidemic encephalitis, and multiple sclerosis. The whole nucleus may be involved to produce a complete paralysis of the face. In more extensive lesions the motor nucleus of nervus abducens may also be involved. The paralysis is of a flaccid lower motor neuron type.

In addition to fibres from the corticobulbar tracts, the motor nucleus of the facial nerve also receives fibres from: the superior colliculus (visual reflex centre); the nucleus of the solitary tract (taste); the sensory trigeminal nuclei; and the acoustic nuclei. These connections enable the motor nucleus to participate in a large number of reflexes, of which the acoustic stapedius reflex is the most important for assessing facial nerve function.

Other reflexes can be checked to determine the integrity of the motor root of the facial nerve: tapping the side of the nose produces an ipsilateral elevation of the upper lip and angle of the mouth; blinking, or palpebral reflex, i.e. contraction of the orbicularis palpebrarum as a response to tactile, optic, or acoustic stimuli. Impulses are carried via the spinal tract of trigeminus, superior colliculus or acoustic nuclei, respectively.

The preganglionic components of the facial nerve arise from cell clusters (the superior salivatory nucleus) on the facial motor root, after forming the genu around the abducens nucleus.

A small blood vessel supplying the area near the floor of the IVth ventricle at the level of the superior salivatory nucleus is apparently prone to thrombosis, and this may result in complete lower motor neuronal paralysis, an associated dry eye, and decreased submaxillary gland secretion because of involvement of the superior salivatory nucleus, as well as the motor fibres. A vertical or rotary nystagmus may result if the rostral end of the vestibular area is involved. The tracts from the cortex concerned with voluntary and following movements of the eye in the horizontal plane decussate and lie medial to the facial motor roots. A patient with a lesion involving this region cannot turn his eyes voluntarily toward the side of the paralysis. An ependymoma of the IVth ventricle or medulloblastoma may likewise present.

The gustatory fibres have their cells of origin in the geniculate ganglion. Their axons enter the brain with the nervus intermedius and pass to the fasciculus solitarius. The fibres decussate and in company with fibres which have arisen from the glossopharyngeal and vagus nerves, ascend to the thalamus. Together they form the secondary ascending gustatory tract; this tract gives off fascicles to the midbrain and mamillary body. According to Bradley, recognition of gustatory impulses takes place at midbrain levels. From the thalamus, impulses are relayed to the base of the central fissure, and toward the uncus. Lesions anywhere along this route may abolish or alter taste sensation.

The cutaneous sensory fibres of the facial nerve overlap similar components of the trigeminal nerve to supply part of the eardrum and the posterosuperior aspect of the external auditory meatus. Their cells of origin are in the geniculate ganglion. The axons of these cells descend in the spinal tract of nervus trigeminus to the spinal nucleus of the Vth nerve from which a secondary relay of fibres ascend and decussate at various levels to reach the thalamus where projections occur to the postcentral gyrus of the cortex.

If the cutaneous sensory fibres of the facial nerve are destroyed, the patient will not note the absence of pain in these areas, due to the overlap of the trigeminal nerve. However, pain due to an irritative lesion of the facial nerve, such as a tumour or herpes zoster oticus may be felt in the external auditory meatus and on the outer surface of the eardrum.

The cortical motor area of the face is situated in the inferior frontal cortical regions, rostral to the central fissure (area 4). A cortical lesion in the 'face' motor area is often associated with an interference with voluntary movements of the tongue on the same side as the facial paralysis. The upper face is not paralysed. Facial movement as emotional responses, reflexes, and automatic responses of facial muscle remain.

Lesions in the genu of the internal capsule cause paresis of the face on the opposite side, and paresis of tongue and uvula.

Lesions in the upper midbrain may involve the oculomotor roots with a homolateral loss of direct and consensual pupillary light reflexes and homolateral external strabismus and an oculomotor paresis.

Lesions lower down in the pons and brainstem may involve the abducens nucleus. Lesions at this level do not involve tongue and uvula because the nerve tracts supplying the hypoglossal nucleus and nucleus ambiguus, have separated from the main cortico-bulbar pathways and lie nearer the midline in the pons.

In supranuclear lesions, the lower face is paralysed, but wrinkling of the forehead and closure of the eye is preserved. The emotional innervation may not be affected and may be the only reliable sign of a supranuclear lesion.

Location of the lesion in peripheral facial paralysis: The length of the facial nerve (*i*) in the posterior cranial fossa is 23 - 24 mm; (*ii*) in the internal auditory meatus, 7 - 8 mm; (*iii*) in the facial canal the (*a*) labyrinthine segment is 3 - 4 mm; (*b*) the tympanic segment; 12 - 13 mm; and (*c*) the mastoid segment; 15 - 20 mm.

Lesions in the cerebellopontine angle cause, in addition to facial paralysis, involvement of the cochlear and vestibular nerves. Taste, lacrimation and salivation may be affected. At a later stage there may be involvement of the trigeminal, glossopharyngeal, vagus and accessory nerves.

Lesions in the internal auditory canal cause involvement of the facial and auditory nerves and nervus intermedius. Involvement in the internal auditory canal of the secretomotor fibres to the lacrimal gland causes decreased tearing, but in some early cases, apparently, an irritative reaction can cause increased tearing.

Lesions of the geniculate ganglion involve the facial nerve with hyperacusis because of paralysis of the stapedius muscle. Alterations in taste, lacrimation, and salivation occur. Involvement of the stapedius muscle may cause hyperacusis; this is a subjective sensation, and not very helpful in the topognosis of facial palsy.

Lesions in the tympanomastoid segment of the facial nerve, should not affect lacrimation.

Extracranial lesions or lesions below the origin of the chorda tympani show normal parasympathetic and taste functions. One may find a complete facial paralysis, but a branch may be spared. In 19 out of 23 cases examined by May,[4] one or more branches of the facial nerve were not involved.

Quantitative tearing (Schirmer's test) based on the nasolacrimal reflex has been described by Zilstorff-Pedersen.[16] Filter-paper strips, 0,5 cm × 10 cm, are hooked over the lower eyelid for 30 seconds while the reflex is stimulated by a sniff of spirits of ammonia. One should empty the conjunctival sacs before inserting the strips if pooling of tears is present. The test is abnormal if the strip on the affected side is less than 20% of the normal side. Zilstorff-Pedersen[16] found that in 99% of normal subjects tested by him, the value on the side with less lacrimation exceeded 74% of the values on the side with more lacrimation as measured in millimetres.

The stapedius reflex measured by the acoustic bridge, may be of great value to pinpoint a facial nerve lesion. The stapedial reflex is an objective test and trustworthy. One should remember that in 2 - 5% of normal subjects the stapedial reflex may be absent.

A sensory deficit of the external auditory canal was found to be a reliable sign of acoustic neuroma by Hitselberger and House.[17]

A deficit is determined thus: (*a*) during caloric testing of the patient he states whether there is a decrease in sensitivity to cold water in one ear canal compared with the other; (*b*) on probing the superior-posterior aspect of the external auditory meatus of the patient, any decrease in sensitivity there, is considered significant.

Chorda Tympani Function Tests

The chorda tympani contains taste fibres, preganglionic secretory fibres to the submaxillary and sublingual salivary glands, and fibres for common sensation at the intra-oral part of the 'geniculate zone'. These sensory fibres were postulated by Hunt,[18] and later confirmed by Costen and Bishop as originating in the geniculate ganglion.

The sense of taste: Electrogustometry[19] is clinically the most suitable technique for evaluating taste, compared with the conventional method of testing for bitterness, sweetness, sourness, etc., which is still a subjective method of testing but not always reliable.

The Elgustometer of Krarup[19] measures in microamperes the differences in thresholds between the normal and affected sides. It is based on the phenomenon that a galvanic current applied to the tongue evokes a sensation of an acid, metallic taste at the positive pole. The electrical stimulus can be varied from 2,5 - 300 μA. A difference in threshold, on right and left sides, exceeding 50% is pathological. Taverner[20] found that normal subjects appreciated a sour, metallic taste with a current of 20 μA.

Submaxillary salivary secretion in facial paralysis will be discussed later.

Radiological examination, especially polytomography of the temporal bone in cases of tumours, fractures, or infections leading to facial paralysis, may render invaluable information about the site of damage to the facial nerve (Fig. 1).

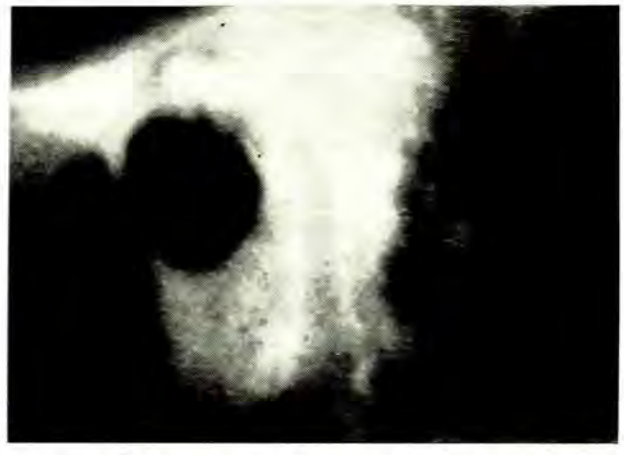

Fig. 1. Tomogram of normal Fallopian canal.

THE DEGREE OF PARALYSIS

Many patients, as well as some aural surgeons, tend to be very lax in their description of the degree of facial paralysis. There is no uniform way of measuring movement of facial muscles.

Jongkees pleads for the results of surgical and conservative treatment to be judged by the same standards, and he supports Janssen's[21] opinion that expressions like fairly good, very good, etc. are no good at all.

May[4] divides the face into 10 parts, 9 for muscle groups and 1 for tone (Table I). The face is classified on a percentage basis, and thus a clear concept of the degree of facial paralysis is formed. Each of the 9 muscle groups chosen, samples a major motor portion of the facial

TABLE I. ASSESSING THE DEGREE OF FACIAL PARALYSIS

	Normal	Weak	Absent
1. Tone	10	5	0
2. Wrinkle forehead	10	5	0
3. Close eyes tightly	10	5	0
4. Blink	10	5	0
5. Wrinkle nose	10	5	0
6. Grin	10	5	0
7. Whistle	10	5	0
8. Blow out cheeks	10	5	0
9. Depress lower lip	10	5	0
10. Tense platysma	10	5	0
	100%	50%	0%

nerve: wrinkling the forehead tests the highest branch; closing the eyes is a protective reflex; blinking aids the lacrimal system; wrinkling the nose tests the midface; grinning, whistling and blowing out the cheeks account for the lower midface. Depressing the lower lip depends on an intact mandibular branch. Testing the platysma, measures the integrity of the cervical branch of the facial nerve.

An absence of creases and folds results in flattening and sagging; the palpebral fissure widens and the mouth droops. One should study the entire face. Regional loss of tone may be observed in patients who begin to recover from a total flaccid paralysis.

(To be continued)

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