Some Aspects of Facial Nerve Paralysis

PART III. COMPLICATIONS, PROGNOSIS AND MANAGEMENT *

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SUMMARY

We have no test in facial paralysis to tell us the moment axonotmesis takes place, and because of this we cannot know for certain when to decompress the facial nerve.

When axonotmesis sets in, complications follow in all cases to a greater or lesser degree. It should be possible to set a definite prognosis within 2 weeks after the onset of facial paralysis, and in many cases even sooner.

In the prognosis of facial paralysis the aetiological and time factors involved, the completeness of paralysis, muscle tone and electrical tests are most important.

S. Afr. Med. J., **47**, 100 (1973).

COMPLICATIONS OF FACIAL PARALYSIS

The complications of facial paralysis depend on the degree of degeneration of the nerve.

Immediate Complications

During the acute phase of paralysis there is:

(i) Psychological alarm. The likely prognosis should be outlined sympathetically and the patient should be seen as often as possible. Kettel[1] recommends massaging the face with oil, exercises before a mirror, and electrical (galvanic) stimulation to the paralysed muscles in order to sustain the morale of the patient.
(ii) Speech impediment.
(iii) Discomfort when eating and drinking.
(iv) Epiphora.
(v) Corneal ulceration and infections of the eye.
(vi) Drooling.
(vii) Loss of voluntary and involuntary facial movement.

Patients with neurapraxia only, make a complete recovery within 2 - 3 weeks. In cases of complete degeneration there is always imperfect regeneration with subsequent long-term complications.

Delayed Complications

A *mask-like face* is an uncommon occurrence because the facial nerve has tremendous regenerative ability. The paretic, or flaccid, face results from an incomplete regrowth of fibres due to some form of obstruction, e.g.:

(i) Retrograde returning of fibres;
(ii) obstruction by scar tissue;
(iii) obstruction by bone spicule, or tumour;
(iv) fibres may form sterile clubs;
(v) fibres may be blocked by narrow, distal sheaths;
(vi) complete severance of the nerve with loss of substance.

A single growth cone from the proximal end of a cut or degenerate nerve will divide into several or many branches, each entering different tubules in the distal end. Misdirection of these fibres is regarded as the underlying cause of *synkinesis or associated movements.* The phenomenon is a voluntary or reflex contraction of one muscle accompanied by synchronous, involuntary contraction of one or several muscles, e.g. twitching of the muscles around the mouth when blinking.

As soon as the growing axon reaches a motor end-plate or a sensory organ, it proceeds to thicken by fresh axoplasm moving into it from the cell-body. Only when the axon attains a certain thickness does it become myelinated. These new, thread-like non-myelinated axons cause *mass movements and spasm* (tonic facial spasm) of the facial musculature. An impulse passing down 1 fibre, or bundle of fibres, can spread to neighbouring bundles.

As the regenerating axons thicken[2] and become myelinated, the spasm and mass movements lessen. However, many large axons enter smaller tubules so preventing them from bcoming thick enough to be myelinated. The distal segment of a regenerated facial nerve, therefore, has a higher proportion of small non-myelinated fibres than the proximal segment. Besides faulty myelination, these tics and spasms may arise from the short-circuiting of regenerating nerve fibres.

In peripheral nerve lesions, recovery may take place by the sprouting of fine[3] intramuscular branches of the motor units. This results in an enlargement of surviving motor units and is characterized by an increase in duration of motor unit potentials, by more than 20%. The first indication of recovery by sprouting may be an increase in polyphasic potentials.

Contractures in the face are unmistakable.[4] The facial muscles have no fixed point of insertion, but are inserted directly into the skin. Contracture may be welcome when it contributes to the symmetrical appearance of a previously drooping face. Contracture can be defined as the fixed shortening of fully relaxed muscle manifested by abnormally deep folds and narrowed palpebral fissures with the face in repose.[5] It may be

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related to a replacement of atrophied muscle fibres by connective tissue. In facial palsy there may be a return of tone without return of movement.

Gustatory tearing (crocodile-tear syndrome), *gustatory sweating* (Frey's syndrome) and *gustatory rhinorrhoea* are all signs of misdirected fibres in cases of faulty regeneration. Gustatory tearing is excessive lacrimation, during increased salivation, affecting the eye on the paralysed side only. This is seen months after recovery from paralysis of the facial nerve due to lesions in the geniculate ganglion region, and is attributed to misdirection of parasympathetic secretory impulses intended for the salivary glands. Golding-Wood[6] states that the misdirection of impulses is due to a sprouting of fibres which establish a physiological connection between the tympanic plexus and the greater superficial petrosal nerve. This symptom may be abolished by a tympanic neurectomy.

PROGNOSIS AND SOME ASPECTS OF MANAGEMENT OF FACIAL PARALYSIS

The prognosis for facial paralysis depends directly on the cause of the condition and on the degree of successful treatment of the primary condition. Some conditions are especially suitable for conservative treatment only e.g. aural herpes zoster and luetic neuritis. Other cases are best treated radically as early as possible, e.g. facial paralysis of direct onset after injury from surgery, or fractures of the skull. In the third group which includes Bell's palsy, facial paralysis of delayed onset after injury to the skull, or surgery to the mastoid, or acute otitis media, there appears to be an absence of any fixed rule for approach. This is due to the fact that we cannot distinguish in the important early stages of the paralysis, those cases unlikely to recover completely.

The chances of spontaneous recovery from facial paralysis due to injury of the skull and Bell's palsy are estimated to be as high as 75—90% by authors like Boone,[7] Cawthorne and Haynes,[5] Grove[9] and Kettel.[10] Other investigators, notably Matthews,[11] Park and Watkins,[12] Robson and Dawes[13] and Taverner,[14] do not share this optimism.

Completeness of Paralysis

Initial complete facial paralysis indicates a serious prognosis. The prognosis is greatly dependent on the severity of the paralysis. We should clearly distinguish between partial and complete paralyses, and between an immediate and delayed onset.[15] Recovery from incomplete paralysis is usual, especially if there is good muscle tone and surgery is not indicated. In a series of Cawthorne and Haynes[5] 47 out of 111 patients (42%) with complete Bell's palsy recovered fully, as against 57 out of 67 (85%) with incomplete paralysis. Laumans and Jongkees[4] agree with this. They also, report that paralysis, partial at the end of the first week, seldom became complete afterwards, and at the end of the second week the severity and course of the paralysis was well established.

Botman and Jongkees[16] say the chance for full recovery of a patient who shows a complete paralysis at the end of the first week is about 50% in cases of Bell's palsy. The chance for full recovery falls to zero when the paralysis is still complete at the end of the second week. For a patient with partial paralysis at the end of the second week, the chance for complete recovery is 85%; if the paralysis is partial from the beginning, the recovery rate is as high as 95%. This favourable prognosis is supported by the normal muscle tone and the presence of a normal nasopalpebral reflex.

Muscle tone is one of the most important signs in facial paralysis. This is stressed by Martin and Helsper,[17] and Kettel.[18] Jongkees and Laumans,[4] agree and state that paralysis with muscle tone still normal, or only slightly affected by the end of the second week, has a very favourable prognosis. Poor tone at the end of the second week, especially if this was present at the end of the first week, indicates a dubious prognosis.

It is agreeed, however, that the extent of facial paralysis is a more reliable criterion than the muscle tone. Return of tone was observed by numerous authors to occur within 3 - 7 days after decompression. After decompression it has been often reported that tone frequently returns long before there is any visible motor recovery.

Aural pain is a sign of poor prognosis according to May.[5] Of 10 patients who presented with severe pain, 7 developed severe damage of the facial nerve. May considers a painful onset indicative of severe damage. Laumans and Jongkees[4] obtained data on pain in 89 patients with Bell's palsy; 52 had pain and 37 were pain-free. In the group who made a complete recovery the numbers of painless and painful cases were the same. Of those who made an incomplete recovery, the number of painful cases exceeded that of the painless cases, by some 25%. Among the patients with aural pain, the numbers with *severe pain* were the same in the complete and incomplete recovery groups.

On the basis of these findings the authors differ over severe initial aural pain as being indicative of a serious prognosis. In 1909 Waterman noted that 41% of patients with mastoid pain in a study of 335 cases of facial paralysis made a quick recovery.

Taste: The values attached by various investigators to taste examination and disturbances in taste sensation vary greatly. According to Tumarkin, cases of Bell's palsy accompanied by marked pain and loss of taste, have an unfavourable prognosis. Park and Watkins[12] stated that loss of taste has no reliable prognostic value and Biemond pointed out that ageusia is an inconstant finding in facial paralysis. Jongkees also found that the presence or absence of a taste disturbance showed no correlation with the ultimate recovery.

Celis-Blaubach[19] noted that all taste fibres to the anterior two-thirds of the tongue may not reach the facial nerve through the chorda tympani. In an important percentage of persons some fibres may take an alternate pathway, perhaps through the petrosal nerves, otic ganglion and mandibular nerve, and reach the facial nerve at the geniculate ganglion.

Electrogustometry may give incorrect results in some cases. This is true when the threshold is above 120 μA and 240 μA, because of the possibility of the simultaneous stimulation of both taste and tactile receptors.

Taverner[20] regarded taste as an unsatisfactory test in 10% of patients because they were unable to identify the stimulus consistently. After division of the chorda tympani, 11 out of 13 patients were unable to appreciate the stimulus on the side of the lesion. In Bell's palsy with neurapraxia, and in partial denervation, transient changes in the taste threshold may occur, although the threshold seldom rises above 50 μA. In complete denervation there was a total loss of taste on the affected side. Taverner stated that a complete loss of taste always preceded the failure of the facial nerve to respond to electrical stimulation. He maintains that in facial paralysis after head injury, as in Bell's palsy, if the threshold of taste is normal, a good prognosis may be predicted.

Hyperacusis: The term refers to an abnormal sensitivity of the ear to sound, homolateral to the facial paralysis. The cause is not clear. It may be ascribed to paralysis of the stapedius muscle which results in dominance of the tensor tympani muscle. There is no betterment of auditory activity, but merely a disagreeable loudness of environmental sounds. Hyperacusis is often described as being associated with facial paralysis, but it is infrequently observed in practice. It offers no conclusion as to the prognosis for facial paralysis, since it is a subjective sensation of inconstant occurrence and transient nature.

Lacrimation: Zillstorff-Pedersen,[21] and Verjaal,[22] attach great importance to this method of investigation, but Laumans and Jongkees[4] believe the lacrimation test to be only of topical diagnostic interest and to have no prognostic indication. He does not subject his patients to this test.

The prognosis for facial paralysis resulting from *chronic otitis media, severe surgical trauma* and *tumours* involving the facial nerve is bad. In these cases it is not rational to adopt an expectant attitude. The prognosis when chronic otitis media and cholesteatoma cause facial paralysis, is bad because it seems that the facial nerve, being exposed to infection, is gradually transformed into connective tissue. If, after decompression, the facial nerve remains electrically and clinically dead, the string of connective tissue which has replaced the nerve, should be replaced by a nerve graft.

Kettel,[1] in 100 consecutive cases operated upon after immediate postoperative facial paralysis, found that 65 patients had completely severed nerves. He examined 105 patients with facial palsy of immediate onset after simple or radical mastoidectomy 5 years or more after surgery, and found complete paralysis in 81, severe paralysis in 16, and complete recovery in only 8 of these cases. An attempt at repair of the nerve is justified as long as the galvanic response is positive, indicating that the facial muscles have not atrophied. Only if the muscles have undergone atrophy, should one resort to plastic operations. Taverner[20] describes patients who had facial palsies for up to 5 years after the war and who were treated by means of nerve grafting. In some of these, good functional results were obtained.

The patient with a facial paralysis of immediate postoperative onset, should be brought back to the operating theatre and the wound inspected. The nerve may be exposed without any bony protection. There may be a haematoma in the nerve, in which case the sheath can be split. If the nerve is severed, it should be repaired immediately. If there is a gap in the facial nerve, the ends should not be sutured. It is much safer to leave the nerve with an undisturbed blood supply in the fallopian canal, to trim the ends, and to insert a graft.

In palsies of delayed onset after mastoid surgery, the continuity of the nerve is uninterrupted. The causes may be pressure by a tamponade, a haematoma, oedema, a spicule of bone pressed into the nerve sheath, or simply, the nerve being exposed during the operation. In most cases the palsy is due to an interference with the blood supply. In these cases the prognosis is good. However, there are exceptions. If the palsy comes on slowly, the pack should be removed and daily nerve excitability tests performed. If a nerve excitability shift of more than 3,5 mA is recorded, decompression should be performed, according to Kettel.[1] Taverner[20] agrees with this. If there are no signs of destruction of the wall of the facial nerve canal, Kettel suggests a decompression of the vertical segment if a simple mastoidectomy has been performed, and a decompression from the geniculate ganglion to the stylomastoid foramen, when a radical or semi-radical operation has been done and the sheath split.

Infection[1] should not delay immediate repair of the nerve. It is usually a low-grade infection and if the repair is postponed, scar tissue or granulations form to complicate the issue.

Fractures of the skull, or more specifically, of the temporal bone, causing facial paralysis are classified into longitudinal, transverse, and a type confined to the mastoid process by Grove.[9]

Transverse fractures may pass through the internal auditory meatus, shatter the cochlea, and injure the petrous segment of the facial nerve. The cochlea and labyrinth may be completely destroyed. A facial palsy is noted in 30 - 50% of such cases and is associated with perceptive deafness.[18]

Longitudinal fractures are by far the commonest and they are apt to extend from the vault to the base, starting in the temporal squama or the parietal bone and extending through the mastoid into the middle ear. The facial nerve is injured mainly in the pyramidal segment which seems to be the *locus electus* for trauma to the nerve due to fractures of the skull. Facial palsy is seen in 10 - 18% of such cases. Severe damage may also be done to the ossicular chain and the drum.

Ramadier and Caussé[23] have drawn attention to the uncommon fracture confined to the mastoid process; it may open into the external auditory meatus and the middle ear, and also reach the facial nerve in its vertical segment.

Very often even on tomography, the fracture lines may not be visible and it should be remembered that the fracture lines seen on X-ray films of the skull, are in the restored position. At the moment of impact the

fractured bones are widely separated and then return together, provided they are not held apart by soft tissue.

In cases of post-traumatic palsies[24] the general condition is often so serious that not enough attention is paid to the facial mobility of the patient who may be in deep coma. A bruised and swollen face makes it nearly impossible to detect the paralysis. Very often the diagnosis of delayed onset is wrong. Because of the bad prognosis in cases of immediate onset, any facial palsy which follows trauma to the skull or nerve should be regarded as one of immediate onset, unless there is proof that it is of delayed onset.

Turner reported the results of conservative therapy of facial palsies due to fractures of the skull. In 36 cases, the palsy was of immediate onset and 75% recovered completely. In 34 cases the palsy was of delayed onset and 32 of these recovered completely. In 40% of post-traumatic cases of facial paralysis that Jongkees[24] operated upon, the fracture had caused direct trauma to the nerve which was torn, severed, compressed, pierced by sharp pieces of bone, or the sheath damaged. In those cases where the fractures did not reach the Fallopian canal, no visible damage to the nerve was seen, but the nerve was oedematous as in Bell's palsy. The growth of connective tissue begins within a few days and may last months. Decompression and proper alignment will, to some extent, prevent fibrosis which spoils regeneration of the nerve.

If a complete facial paralysis due to a fractured temporal bone appears immediately after an accident the nerve should be explored as soon as possible. This also applies to cases in which the palsy is accompanied by signs of dislocation of the ossicles, according to Von Schulthess.[2] When the hearing is completely destroyed, only the condition of the facial nerve needs to be considered.

Kettel[1] advises that complete palsies, especially with loss of tone, which arise within the first 3 days, should be explored. Kettel[1] and Jongkees[24] agree that any post-traumatic facial paralysis, even that with slow onset, should be subjected to surgery if after 6 weeks the paralysis is still complete. Fibrillation potentials in the presence of complete paralysis are a surgical indication, according to Laumans.[25] In this respect one should rely on electromyography.

In those cases where oedema or intracanalicular bleeding was observed, the patient might have improved after further delay, but in Kettel's view it is fair to assume that decompression hastens recovery, especially when, improvement is seen so often after surgery.

Even the most favourable statistics show that 10 - 15% of patients with Bell's palsy do not recover completely. This percentage is too large to justify an exclusively conservative attitude. Experience has shown that, there is always the possibility that the paralysis might ultimately prove not to be Bell's palsy, but due to a latent otitis media or an incipient tumour.[4]

Like most eminent otologists, Jongkees[24] starts treating Bell's palsy conservatively, but switches to surgery when insufficient, or no improvement, is seen. There is still no consensus of opinion concerning criteria for changing from the initial conservative therapeutic approach, to surgery. Jongkees's[24] indications for facial nerve decompression are;

(i) complete paralysis with loss of tone;

(ii) nerve excitability difference of more that 3,5 mA between the normal and the affected side;

(iii) as soon as fibrillation potentials are demonstrated by means of electromyography, surgery may save nerve fibres still capable of recovery.

By these criteria some operations may be unnecessary, but more good than bad will come of it, according to Jongkees. The experienced ear surgeon does not agree with the objection frequently made that a major surgical procedure is involved, with grave risks for the patient.

No one has ever shown irrefutable statistics that decompression of the facial nerve gives better results than conservative treatment.[24] It is also wrong to fix a specific time to decompress; this is a compromise and not the result of thorough examination and scientific investigation.

Taverner[29] states that we do not have the experimental evidence to justify decompression of the facial nerve, routinely, even in selected cases of Bell's palsy. All cases with only a conduction block will recover completely. All patients with partial denervation eventually recover sufficiently to be satisfied. Not one of Taverner's patients with partial denervation complained about the end result, and in this group decompression may only speed up recovery a little. The group with complete denervation is much in need of help. Of these patients, 25% have such marred faces that they remain miserable for the rest of their lives. If Bell's palsy is due to compression of the nerve in the Fallopian canal, it would be a logical procedure to undertake surgical decompression, but unfortunately it is probable that complete electrical inexcitability means that the nerve is dead and decompression of no avail.

Surgical decompression appears least likely to help those very patients who most need help. Alford[26] reports the incidence of synkinesis to be 60% after decompression within 2 days of recording a nerve excitability shift of 3,5 mA. All patients decompressed later than 2 days after excitability was altered, had synkinesis on return of function. Degeneration may set in rapidly and since the information, when obtained by the nerve excitability test, is already 3 to 4 days old, degeneration may be complete by the time a shift of 3,5 mA is recorded, and then decompression is of little value.

When degeneration sets in slowly and progresses slowly, as observed in 12 - 18% of cases of Bell's palsy by May[5] and Campbell *et al.*[27] decompression after a nerve excitability shift of 3,5 mA or more, may prevent some nerve fibres from degenerating.

The question of decompression of the facial nerve for Bell's palsy, is not yet answerable. What is needed is a simple, trustworthy test to detect degeneration the moment it occurs.

To quote Jongkees: 'Really good statistics may prove things or may disprove things. In order to provide good

statistics we should follow a uniform formula and identical criteria in treating facial palsies. Only then can we start comparing results.'

I wish to thank Dr J. G. Burger, Superintendent, Groote Schuur Hospital, for permission to publish, Dr J. F. Jarvis and Dr S. Sellars for their interest in this project, Dr S. Goldberg for his kind assistance, and Sister O'Reilly for a helpful hand.

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