Some Aspects of Facial Nerve Paralysis*

PART II. ELECTRICAL TESTS AND THE SUBMAXILLARY SALIVARY TEST

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SUMMARY

The submaxillary salivary flow test gives reliable information as to whether neurapraxia, axonotmesis, or neurotmesis of the facial nerve is present. This can be corroborated by electrical studies. This test can make an important contribution to the topognosis and prognosis of facial paralysis, especially when elaborate electrical equipment for nerve excitability tests and electromyography is not available.

S. Afr. Med. J., **47**, 65 (1973).

ELECTRICAL TESTS

In 1872 Duchenne described the technique of nerve excitability testing for facial paralysis.

According to the severity of the condition, peripheral nerve lesions can be classified on the bases of electrical tests, as: (*a*) neurapraxia; (*b*) axonotmesis; and (*c*) neurotmesis.

This classification for peripheral nerve lesions was introduced by Seddon[1] in 1943. It is of attractive simplicity and it has been accepted by most investigators, and particularly for paralysis of the facial nerve.

Neurapraxia, or physiological block, causes a temporary paralysis and is characterized by normal muscle responses. There is loss of conductivity only across the lesion, and recovery is usually rapid and complete, regardless of treatment, within 2 to 3 weeks.

Neurotmesis, or complete degeneration, is characterized by the absence of nerve excitability; strength-duration curves are shifted to the right and fibrillations are seen by electromyography. In this condition the axis cylinder and myelin sheath have undergone complete degeneration. Functional restoration is possible only by regeneration of the peripheral portion of the nerve. Regenerating nerves grow approximately 2,5 cm per month. Return of function is never complete and is always associated with complications.

Axonotmesis is caused by incomplete degeneration and varies in severity and prognosis between neurapraxia and neurotmesis. Nerve excitability is decreased, or lost, and some fibrillation potentials may appear on electromyography.

A conduction block results because of demyelination in the region of histological damage. The fast-conducting fibres are the first to be affected and when there is regeneration, the new fibres conduct slowly.[2]

The maintenance of nerve and muscle excitability depends greatly upon sodium and potassium cations. States of polarization and depolarization are dependent on the shift of sodium and potassium cations across the cell membranes. With injury to the nerve,[3,4] elimination of the membrane potential takes place and this causes a lack of electrical conduction. A shift of potassium from the cell takes place. An over-all increase in the potassium content of the injured nerve exists where, for instance, bone fragments cause a foreign body reaction. Hyaluronic acid from an inflammatory response, occurs as a potassium hyaluronate. Fibroblast and mast-cell activities increase and elaborate mucopolysaccharides. This may explain the high potassium values, maintained in the injured area of the nerve, inhibiting transmission. With decompression after traumatic facial paralysis, the bone fragments irritating the nerve are removed and the stimulus and build-up of the potassium pool are reduced according to Schiff,[3] and conduction may again take place.

As soon as the biochemical block is dispersed, the neural servo-mechanism (muscle spindles) apparently functions again. This may explain the increase in muscle tone found in some cases after decompression.

Matthews[5] pointed out that degeneration is not an all-or-none phenomenon. Some nerve fibres may degenerate and others remain intact, or in a state of reversible block. Such a reversible block may persist for as long as 6 - 12 weeks after the onset of facial paralysis.[6-8] This may explain the rapid return of at least some function within a few days after decompression of the facial nerve, as described by other authors.

The concept of facial palsy as an acute lesion which either undergoes degeneration from the start or not at all, is not valid in all cases. It may be a slow, progressive condition. Electrodiagnostic methods may show nerve degeneration as early as the third or fourth day after onset, by which time it is too late to prevent degeneration by decompression. One may be able to prevent the progression of degeneration only in those slowly developing cases where some of the nerve fibres are still neurapraxic at the time of decompression. Electrical tests are of great value in deciding the prognosis for facial paralysis.

The Nerve Excitability Test

When a lesion blocks conduction in a motor nerve, the muscle it supplies will not function voluntarily, but if the

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nerve is intact distal to the lesion, the nerve conducts normally if stimulated beyond the lesion. This is neurapraxia.

In a complete lesion where the axons are damaged, as in axonotmesis, and some of the nerve fibres degenerate, an increase of intensity is required to cause a muscle twitch. If the nerve distal to the lesion degenerates, as in neurotmesis, no conduction occurs, no matter how intense the stimulus.

Landau[9], and Gilliatt and Taylor[10] observed that a completely sectioned nerve may continue to conduct distal to the lesion for as long as 72 hours after the injury. For this reason the nerve excitability test has no value until 24 to 72 hours after the onset of paralysis.

When a peripheral nerve is cut, the distal segment of the axon receives sufficient nutrients and oxygen, probably through the Schwann cells that surround it,[11] to survive for 2 or 3 days with continued excitability.

Gilliatt and Taylor[10] found, in a nerve cut at operation, that the threshold of the evoked response increased progressively from the first day. The amplitude of the evoked potential remained unaltered until the third day after the operation, when the response started to decrease and could not be evoked after 7 days. The impulses are conducted at normal velocity as long as they are present. The nerve excitability test is of value only for as long as the nerve remains intact; when the nerve degenerates the test is no longer useful. Degeneration may set in very slowly, as pointed out by Landau,[9] Gilliatt and Taylor.[10] The test can be performed with any stimulator of which the strength and duration of the electrical current can be varied.

The Hilger nerve stimulator is designed to test the facial nerve. It is portable and functions within a few minutes without discomfort to the patient. It delivers a square-wave pulse lasting 0,6 milliseconds at a rate of 6 per second. This stimulus is not long enough to cause a denervated muscle to respond; the response of a denervated muscle is characterized by a sluggish contraction as compared with the brisk movement of the innervated.

Campbell *et al.*,[12] working with Cawthorne, utilized a unipolar electrode over the nerve trunk and an indifferent electrode on the back of the patient's neck. A square-wave impulse lasting 1 millisecond is applied at intervals of 1 second and the smallest intensity of stimulus in milli-amperes which will evoke a visible muscle twitch in any part of the face, is determined. The important figure is not the absolute measure, but the difference between the two sides. Gilliatt and Taylor[10] test with impulses of 0,1 millisecond, Richardson[13] and Wynn-Parry[14] and Laumans[15] use 0,3 millisecond impulses.

It is impossible to set a standard, normal threshold of intensity for the stimulation of contraction because of variations in skin temperature, thickness of the layer of soft tissue between the active electrode and the nerve, the position of the electrode, and anatomical variations in the course of the facial nerve. The stimulation intensity may also be affected by movements and tension in the facial muscles, especially in cases of partial paralysis.

The threshold intensity in normal patients, may vary from 2 - 16 mA in Jongkees's and from 3 - 8 mA in Richardson's[13] series. Laumans[15] found the average threshold intensity to be 6,5 mA. In normal subjects, differences between left and right are, as a rule, very small and vary from 0,2 mA to 0,4 mA.

May[5] states that it is not enough just to stimulate over the main nerve-trunk at its exit from the stylomastoid foramen, as outlined by Hilger.[16] He suggests that each part of the face should be tested for viability of the facial nerve. He tests 5 general areas: (*i*) the forehead and eyebrow; (*ii*) the peri-orbital region; (*iii*) the area of the cheek, upper lip and nasal ala; (*iv*) the lower lip; and (*v*) the cervical or platysmal area (Fig. 1).

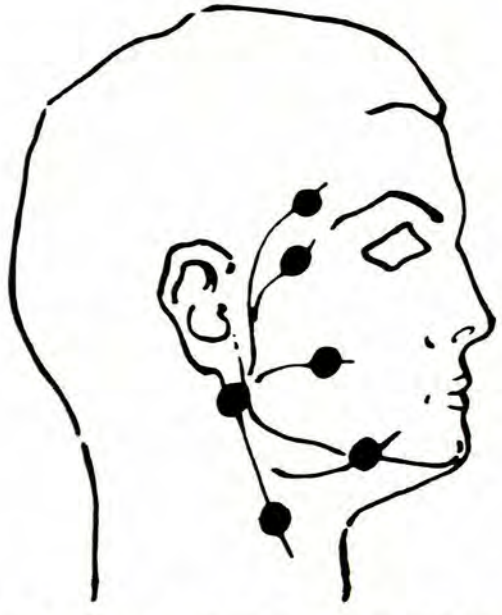

Fig. 1. Facial motor points to stimulate major divisions of the facial nerve.

In this way the upper, middle, and lower facial branches are tested. The peripheral branches of the facial nerve are superficial and the general anatomical pattern is relatively constant. The branch of the facial nerve that supplies the lower lip can be readily isolated where it crosses the mandible just anterior to the masseter muscle, in close relation to the facial artery where it grooves the mandible. May[5] uses this branch to determine the threshold on the normal side. The other major divisions can be located by stepping the stimulating probe along a line drawn halfway between the sideburn and the lateral aspect of the eyebrow. By using the muscle twitch as a threshold, each of the 5 areas is tested. If it takes more than 3,5 mA to stimulate the area on the involved side compared with the normal side, it indicates partial denervation, or axonotmesis. If no response can be elicited, even with maximal stimulation, complete degeneration or neurotmesis exists. If the involved side responds to a stimulus of equal intensity, or of not more than 3,5 mA compared with the normal side, neurapraxia, or a physiologic block, exists.

Jongkees and Laumans[17] use a difference of 3,5 mA as the critical difference indicating degeneration between the normal and involved nerves. By utilizing this critical difference of 3,5 mA, they found, during the first week of facial palsy, that in 4 out of 7 patients the prognosis was correctly considered to be unfavourable; during the first two weeks, in 8 out of 10 patients, the prognosis was correctly considered unfavourable, and during the first three weeks in 12 out of 13 patients.

Richardson[19] feels that the source of error in basing a prognosis on nerve excitability measurement during the first week, appears to be due to progress of the disease, rather than failure of the method.

Laumans and Jongkees[17] recommend daily or twice daily testing as soon as deviating results of more than 3,5 mA are obtained; if the nerve excitability then suddenly drops, one should decompress the nerve.

According to May, the rationale for testing each major area supplied by the facial nerve depends on the fact that part of a bundle, or certain fibres in the temporal bone, can be affected more than others, depending on the nature, location and severity of the lesion. One may observe neurotmesis in one part of the face and neurapraxia in another, especially during the acute phase and during the phase of recovery. He describes 3 cases of Bell's palsy where nerve excitability was lost in one part of the face and not in the other. This was correlated with electromyographic studies and the clinical course. Mixed lesions have been reported by others. Campbell *et al.*[12] also mention the necessity to test all the branches. They describe 3 cases where excitability was retained in some branches and was absent in others. Kettel[15] also stressed the existence of mixed lesions, and that, while some nerve fibres and branches undergo degeneration, others remain in a condition of reversible injury and capable of recovery.

These clinical findings can be substantiated histopathologically. Cajal[19] demonstrated that fibres of equal size, lying next to one another, could have different degrees of vulnerability. He tied a ligature around a nerve and showed the peripheral fibres to be more severely injured than the central axons which seemed to be spared. Reddy *et al.*[20] reported the histopathology of the facial nerve of a patient who had Bell's palsy for 17 days before death from a pulmonary embolus. Only 30% of the facial nerve fibres showed degeneration.

May[5] also states that the facial nerve is spatially orientated as it is in the cortex and pons. In the pons, the upper face is located dorsally, and the lower face, ventrally. May described several cases to prove that the nerve is similarly orientated in the tympanomastoid portion; this is in agreement with Miehlke.[24] Injury to the dorsal mastoid portion causes changes in the upper face, whereas injury to the ventral tympanic portion causes changes in the lower face (Fig. 2). In the literature there are numerous reports to support this view of May.

Schiff[3] reported a case of temporal bone fracture which resulted in facial paralysis. A bone chip was noted to be cutting through the dorsal portion of the facial nerve. There was recovery from the paralysis except for marked weakness of muscle movement in the upper face. Similar

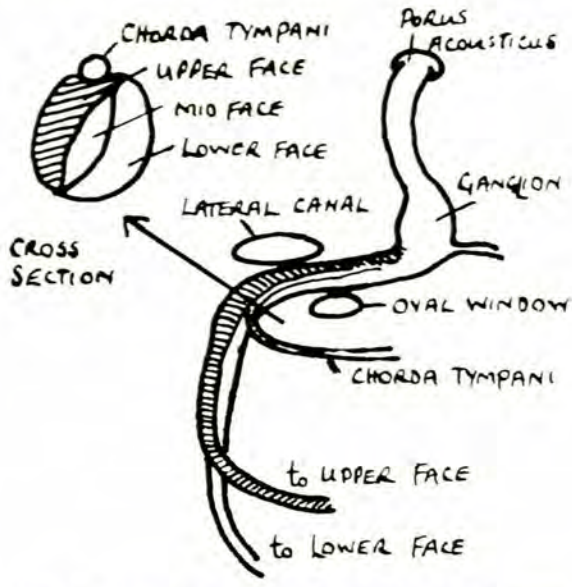

Fig. 2. Orientation of axons in the facial nerve.

findings were reported by Morrow and Broder,[21] Rulon and Hallberg,[22] Scheer[23] and Miehlke.[24] Mawson[25] states the first symptom of facial paralysis as a complication of otitis media, is weakness of the corner of the mouth and a tendency to drool. The most common site of injury to the facial nerve in otitis media, is the ventral portion just above the oval window, because of dehiscences of the Fallopian canal.

Each portion of the facial nerve may be differently involved and this should encourage electrical analysis of each portion for topognostic and prognostic purposes.

Jongkees[26] found the nerve excitability test of undoubted value in assessing the facial nerve, but he warns against entire reliance on this test alone, as it is not exact in every case.

Jongkees and Laumans measured the nerve excitability in 141 patients with facial paralysis. In 71 patients, no reaction of the facial muscles to an impulse strength up to 20 mA was seen at a given moment. These patients were not surgically treated and not one made a complete recovery. Electromyographic studies were performed in 64 of these 71 patients and profuse fibrillation potentials were found in all. This suggests that absence of nerve excitability to applications of pulses up to 20 mA is indicative of a degeneration of the nerve fibres. This is in agreement with the finding of Richardson[19] and Wynn-Parry.[14] A stimulus of 20 mA is also the maximal intensity tolerated by adults. In this series of 141 patients there were 30 cases of Bell's palsy who recovered completely. In these it appeared that the excitability differences in the patients with Bell's palsy, did not differ significantly from those in normal test subjects. In such cases a good prognosis is indicated.

Richardson[19] did nerve excitability tests on 197 patients and classified his results thus:

(i) excitability **unimpaired,** in which the affected side responds fully with the same current intensity, as the normal side.
(ii) excitability **diminished,** in which the affected side responds fully, but requires more current than the normal.
(iii) excitability **absent,** in which the affected side fails to respond to very high currents.

In theory such results indicate neurapraxia, partial nerve degeneration, and complete degeneration.

Groves, in conjunction with Campbell *et al.*[12] studied nerve excitability on a day-to-day basis, and often found a sudden worsening or diminishing of nerve excitability, which appears a day or two before spontaneous recovery of voluntary movement. To avoid operating unnecessarily they use a loss of all excitability to a 1 millisecond impulse as an indication for surgical intervention. On this basis they had 10 patients who qualified for decompression. Of these, 5 refused the operation and 5 were decompressed. Four of these 5 patients had swollen nerves. Subsequent to operation these 5 patients did no better than the 5 who had not undergone operation. This is logical. To decompress after total degeneration, when nerve excitability has been lost, is too late, and in cases of Bell's palsy, of no avail.

The value of nerve excitability tests as used today, is: (*i*) as a guide to prognosis; and (*ii*) that it informs, 3 days after the event, that degeneration has taken place, and if this has taken place slowly and this test is used as an indication for decompression, we may be able to prevent some axons from degeneration.

Richardson[13] found in his series of 197 patients, that only in 12% was nerve excitability lost in between the 7th and 21st day. This is a very limited group.

Campbell,[27] in a study of 137 patients, underlined the possibility of late degeneration of the facial nerve. He found that 18% of patients with retained nerve excitability at the first examination, eventually showed some degree of nerve degeneration. He also found that 12% of all cases with retained nerve excitability at first examination, failed to recover completely.

Electromyography

Electromyography is the recording of the electrical characteristics of what Sherrington in 1925 called the motor unit which comprises the motor cell, its axis cylinder and all muscle fibres connected to it. The technique is based on the detection of the skeletal muscle fibre action potentials which accompany activity of these fibres. A needle electrode is inserted into a muscle and connected to an oscilloscope which indicates the electrical activity in the motor units with which it is in contact. It has the limitation, however, that only a few of many thousands of motor units can be sampled.

A denervated muscle, being hyperirritable;[13] produces spontaneous, electrical fibrillation potentials. These usually appear 10 - 21 days after degeneration has set in.

Five forms of muscle action potentials are relevant to the study of facial palsies:

(i) positive potentials, and
(ii) fibrillation potentials. Both occur spontaneously 10 or more days after denervation of which they are the cardinal electromyographic signs.
(iii) Motor unit potentials. These are the action potentials derived from the almost synchronous contraction of physiologically grouped muscle fibres. Motor unit potentials are produced up to 30 per second, during a normal volitional effort, and their presence in denervated muscle is indicative of an incomplete lesion.
(iv) The reappearance after degeneration of polyphasic potentials, is the first sign of regeneration.
(v) motor unit potentials may also occur in groups, when they are called grouped or repetitive motor unit potentials, or discharges. This may be seen in post-ischaemic lesions of the proximal part of the lower motor neurones. They usually appear after regeneration of a nerve.

Kettel[28] regards electromyography as useless in an emergency. Fibrillation potentials are seen only 10 - 14 days after denervation, by which time the damage has been done. Campbell *et al.*[12] found that electromyography could be misleading. Fibrillation potentials have been detected when only a few nerve fibres have degenerated, suggesting a worse prognosis than was justified. They detected fibrillations in patients who recovered fully. May[8] describes 2 similar cases. Fibrillation potentials may be absent in some cases of denervation where the intramuscular temperature is low, or in hypoxia of the muscles. Some patients with severe paresis do not develop fibrillation potentials[2] and yet they do not recover, and there are patients with fibrillation potentials who recover fast. For reasons unknown, the frontal muscle does not show any sign of spontaneous activity after denervation, and this muscle should not be utilized for electromyography.

It should be pointed out that electromyography is painful; it requires the patient to be willing and relaxed and

TABLE I. NERVE EXCITABILITY RESULTS IN 197 PATIENTS

No.		Full recovery	Distortion on movement	Distortion at rest and on movement
Unimpaired	**77**	**88%**	**9%**	**3%**
Diminished	**35**	**40%**	**46%**	**14%**
Absent	**85**	**13%**	**31%**	**56%**
Total	**197**			

it should be performed by an experienced physician. Electromyography may be distorted by artifacts created by alternating currents, or even arterial pulsations, and requires an expensive complicated instrument.[29]

Alford[30] found electromyographic evidence of denervation in all cases where nerve excitability responses on the paralysed side were significantly altered (more than 3,5 mA. He found electromyography very useful to demonstrate the continuity of the facial nerve in traumatic cases where early information is desirable.

Laumans and Jongkees[17] find electromyography a very sensitive test of minimal denervation in peripheral nerve lesions, and the most reliable test of both nerve continuity and motor nerve regeneration; it can differentiate between complete and partial paralysis, and between neurapraxia and axon degeneration. Buchthal[2] agrees that electromyography is a very sensitive test which may pick up activity in only a few motor units. Clinical observation may indicate activity in a muscle, which is due, in fact, to passive movement of the paretic side by the non-paretic side. This may be confirmed by electromyography. The orbicularis oris muscle may also receive nerve fibres from the opposite side. In order to exclude this, the unaffected side needs to be anaesthetized.

The presence of fibrillation potentials is a bad prognostic sign because this indicates nerve fibre degeneration. Jongkees and Laumans[17] found fibrillation potentials at one stage or another, in 128 patients with Bell's palsy, surgical trauma, head injuries, etc. Of these patients only 6 made a complete recovery. All these patients were followed-up for at least one year. Of 31 patients with facial paralysis without fibrillation potentials, 28 made a complete recovery. In the absence of fibrillation potentials, a complete recovery may be expected.

In 1955 Taverner[31] reported the results of an electromyographic study of 96 cases of Bell's palsy of recent onset. He found fibrillations in 51 and of these, 49 made an incomplete recovery. There was no evidence of degeneration in 45 patients and 44 of these made a complete recovery.

Strength-Duration Determination

The strength-duration curve depends on the basic principle that a denervated muscle requires a stimulus of long duration when it is compared with an innervated one which requires a very short stimulus to excite it. The longer the duration, the more the curve is shifted to the right. As the muscle becomes re-innervated, the curve shifts back to the left. The first sign of re-innervation is a kink in the curve. The strength-duration determination is useful as a guide, but it represents the most excitable fibres present and does not take into account other less active, or degenerate fibres.

Conduction Time

Taverner[31] is the main exponent of this test, but most authors regard it to be without advantage over nerve excitability tests. Taverner, in testing the conduction time of the facial nerve over a distance of 3,5 cm, found 4 milliseconds to be the upper limit of normal. He tested 167 patients divided into 3 groups:

Group I—no denervation (47 patients): In all cases, at every examination, the conduction-time was less than 4 milliseconds. Clinical recovery was complete and it may be assumed that simple conduction block had been present. There were no fibrillation potentials and after 6 months or more, no sign of associated movement, or evidence of denervation, was detected.

Group II—complete denervation (57 patients): 3 or 4 days after complete denervation, conduction-time and excitability were completely lost. In none of these patients was an electrical response obtained 7 days or more after onset. Of 22 patients followed-up for more than 1 year, 16 were satisfied with the end result (73%), although a number had associated movements, and all had some disfigurement.

Group III—partial denervation (63 patients): Denervation was established by the detection of fibrillation-activity, and associated movement which appeared later. Response to electrical stimulation was always present and the conduction-time remained unchanged or slowed considerably. After 6 weeks the average percentage of recovery for this group was 80. After 9 months, several cases achieved full muscle power, although minute associated movements were present. All these patients believed complete recovery had occurred.

THE SUBMAXILLARY SALIVARY TEST WITH SPECIAL REFERENCE TO ITS PROGNOSTIC VALUE

Intratemporal facial nerve involvement is accompanied by impaired chorda tympani function. This becomes clinically evident when the visceral motor, secretory salivary, preganglionic nerve fibres which activate the submaxillary salivary glands, are utilized as an index of chorda tympani nerve function. Magielski and Blatt[32] advocated the submaxillary salivary test:

(i) as an aid in localizing the site of facial nerve damage;
(ii) to compare the degree of impaired chorda tympani function with the degree of facial paralysis; and
(iii) as an aid in measuring and predicting spontaneous recovery of the damaged facial nerve.

They believed that the spontaneous recovery from complete facial paralysis may be predicted if the chorda tympani retains its function, or rapidly becomes functional early in the course of Bell's palsy. The pathological process in Bell's palsy is not severe if the chorda is partially spared. A very late and only partial return of chorda tympani function, is a poor prognostic sign. Magielski and Blatt[32] found testing for taste to be very unreliable, but the submaxillary salivary test to be objective and very reliable.

In normal individuals, submaxillary salivary flow on the two sides is identical. Salivary flow can be easily stimu-

lated (by lemon juice) and in calculating results, the normal side is used as a control.[29]

Cocaine (10% solution) is applied to Wharton's duct which is dilated by puncta lacrimalia dilators and Bowman's blunt-tipped probes, Nos 1 - 4. Polyethylene tubing (0,5 mm diameter) of lengths (8 - 10 cm), sterilized in 1:500 aqueous benzalthonium chloride for 24 hours, is inserted into each submaxillary salivary duct and advanced 3 - 4 cm (Fig. 3).

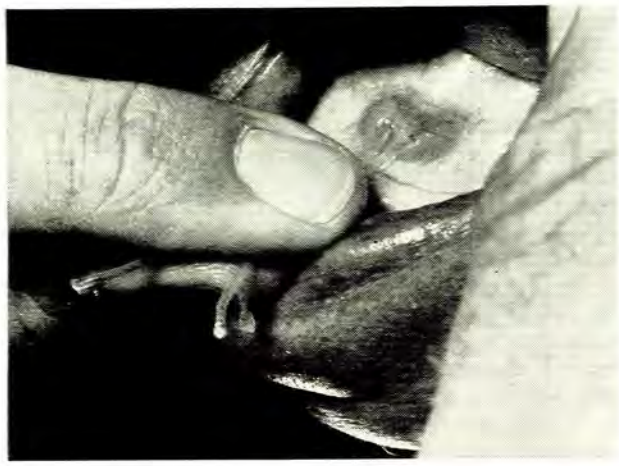

Fig. 3. Polyethylene tubes inserted into Wharton's ducts.

The patient sucks a slice of lemon to stimulate salivary flow and the drops from each tube are counted for 60 seconds. In this way the salivary flow on the affected side, is expressed as a percentage of the flow on the normal side.

May[5] recommends that a minimum of 10 drops be collected from the normal side, even if it requires a flow period longer than 60 seconds. If the test gives unsatisfactory results, e.g. no drops on the affected side and 4 drops on the unaffected side during the first minute, collection may continue for 60 seconds more.

Thick, stringy, tenacious saliva is very often seen on the affected side, and it results from the uninhibited sympathetic influence which is seen in impaired chorda tympany function. In more than 90% of patients tested by May, the salivary flow on the unaffected side was more than 20 drops per minute.

Blatt[29] proposed criteria based on the submaxillary salivary test for predicting the outcome of facial paralysis.

Prognosis for spontaneous recovery: when the chorda tympani visceral motor function is estimated to be 40% or more of that on the normal side.

Prognosis for permanent sequelae (or index for decompression): (*a*) salivary flow compared with the normal side: 25 - 40%—observe the patient for as long as 3 weeks, and if the study is not in the 40th percentile, or better, Blatt advises decompression; (*b*) salivary flow 10 - 25% compared with the normal side—observe for 2 weeks and if not in the 40th percentile, decompress; (*c*) salivary flow less than 10% compared with the normal side—decompress within 24 - 72 hours.

Magielski and Blatt[32] also challenge the concept of the reflex formation of saliva in the absence of secretory innervation. If the chorda tympani is severed, then no secretion from the submaxillary salivary gland is forthcoming.

May[5] tested salivary flow in 9 patients within 24 hours after section of the chorda tympani. In every case salivation was less than 10% of normal. He also found evidence of degeneration in every case in which the salivation test results were 25% of normal, or less. He recommends that decompression should be considered in every case in which salivation is 25% of normal, or less, and that surgery should be carried out within 12 hours, or sooner, after abnormal salivation results are obtained.

The advantage of chorda tympani function testing seems to be, according to May, that it enables accurate prediction of the cases that would degenerate before any abnormality was detected by the nerve excitability test. In 6 of his cases, significant changes in the affected nerve were detected, 2, 2, 2, 6, 8 and 10 days, respectively, by the salivation test, before they appeared electrically.

The chorda tympani fibres are preganglionic, parasympathetic fibres classified as type B fibres. The facial nerve motor fibres are classified as type A fibres. Experiments have demonstrated that the quick-conducting type A fibres are affected first and only then the type B fibres.

It seems that, whereas a neurapraxic facial motor fibre is characterized by a facial palsy, a state of neurapraxia of the parasympathetic fibres of the chorda tympani is not characterized by a complete absence of salivary flow from the submaxillary salivary gland.

In cases of neurapraxia of the facial nerve, with normal nerve excitability, salivary flow from Wharton's duct is seldom less than 40% of normal. When degeneration sets in, the submaxillary salivary test is quick to show this, as salivation drops below 25% of normal. Nerve excitability will indicate degeneration as late as 72 hours after the event.

The chorda tympani function test is a critical part of the topognostic work-up for facial paralysis. In cases of cerebellopontine angle lesions, facial motor function is involved much less frequently than is chorda tympani function. Cawthorne,[33] quoting Harvey Cushing, noted how remarkable it is that facial motor function escaped involvement by acoustic nerve neuromas.

Pulec and House[34] reported a series of 53 acoustic nerve neuromas, 5 with facial weakness and 3 with facial twitching. In this same series, 19 of 28 had a change in taste. Zilstorff-Pedersen[35] described impairment of taste and lacrimation as the earliest effects of cerebellopontine angle tumours upon the facial nerve. The nervus intermedius of Wrisberg which carries the sensory and autonomic nerve fibres, is involved early because it lies between the acousticovestibular nerve and the facial nerve and is subject earlier than the facial nerve to pressure and stretching.

The limitations of the salivary flow test, according to May,[5] are:

1. It can be used to prognose only for lesions proximal to the stylomastoid foramen.
2. It is of no value in the rare, unusual case where the paralysis is bilateral.
3. It is not practical in infants, young children, and unco-operative adults.
4. Like nerve excitability tests, it has a limited value, since it is useless after degeneration has set in.
5. It is tedious and time-consuming (15 - 30 minutes) and requires dexterity.
6. It is difficult to perform by the bedside of the hospitalized patient.

During the past year I performed 218 submaxillary salivary flow tests on 36 patients with unilateral facial palsy, and averaged 6 successful readings per patient. In another 7 patients I did not carry out the test because either the patient refused to co-operate, or there were technical difficulties. False passages, due to haste or a tortuous submaxillary salivary duct, are easy to make. The operating microscope is a great help in cannulating the ducts.

The quantity of saliva in testing for salivary flow is not the important measurement, but the difference in flow on the 2 sides. In 10 normal subjects this flow was identical. Sticky, thick, tenacious saliva is seen in most cases who develop axonotmesis of the facial nerve, but care should be taken not to jump to conclusions, because also in normal subjects the first few drops may be thick and mucoid at times.

In 9 patients, 7 days after one chorda tympani was severed during a radical mastoidectomy, or stapedectomy, salivary flow was 10% of normal, or less, on the affected side.

In 7 cases on whom radical mastoidectomies had been performed, more than 2 years before testing, the salivary flow varied from 33% to 100%. This could mean that the chorda tympani were intact, or that visceral secretory motor fibres reach the gland from another source. There may be a pathway via the otic ganglion and lingual nerve. In 2 patients with radical mastoidectomies of more than 2 years standing, there was less than 10% salivary flow, compared with the normal side.

In 100 testings of salivary flow on the normal side in different patients, a maximal flow rate of 83 drops per minute, to give an average flow of 30 drops per minute was obtained and a minimal of 4 drops per minute. May[5] found, in more than 90% of patients, a flow of more than 20 drops per minute.

The submaxillary salivary test may be of great value in localizing facial nerve damage, as stated by Magielski and Blatt,[32] but care should be taken not to accept the facial nerve as being normal proximal to the junction of the chorda tympani, in cases with equal, or near equal, salivary flow. In my experience, the salivary flow in cases of neurapraxia, is equal to, or in most cases, better than, 50% of the normal flow. This was so in 61% of this series.

A typical illustration of salivary flow in neurapraxia of the facial nerve follows:

A 41-year-old Bantu man developed a right-sided Bell's palsy on 6 October 1970; there was very little aural pain. There was epiphora on the right side and he was aware of a metallic taste. He had a complete right-sided Bell's palsy, but muscle tone was not affected. The stapedial reflex was absent on the right side. By the classification of May, and by taking into account the normal muscle tone, it was estimated that the patient had a 90% facial paralysis.

Nerve excitability tests performed on 9, 12 and 14 October 1970 were equal on both sides (4,0 mA).

Submaxillary salivary flow on 7, 8, 12 and 14 October 1970 was equal on both sides and on 29 October 1970 submaxillary salivary flow on the affected side was 90% compared with the normal side. Four weeks after the initial paralysis, this patient had made a complete recovery.

May found evidence of degeneration in every instance in which the salivation test results were 25%, or less. I had 14 patients (39%) who presented with a salivary flow of less than 33% on the affected side, at some stage. In retrospect, all but 1 of these patients made an incomplete recovery. A residual weakness or paralysis with or without synkinesis, was present 3 months later. This figure corresponds very closely with May's;[5] it is indicative of axonotmesis which, in most cases, was also shown by nerve excitability tests. The 1 case who did not develop permanent sequelae in spite of a salivary flow of less than 33%, was difficult to assess, for on no occasion could her normal, innervated, submaxillary salivary gland be stimulated to excrete more than 6 drops of saliva per minute. She made a speedy recovery within 6 weeks.

The submaxillary salivary flow test gave unreliable information in 1 case, a 23-year-old Coloured man who showed in 6 consecutive tests, a salivary flow varying from 50% to 75% of normal, and in spite of this excellent retention of chorda tympani secretomotor function, he had a 50% facial paralysis 3½ months later, with gross synkinesis.

Comparison of results obtained by means of the submaxillary salivary flow test with that of nerve excitability, did not substantiate that axonotmesis is detectable earlier by means of the salivary flow test.

As an aid to prognosis for facial palsy, this test is invaluable, but its greatest drawbacks are that it is time-consuming; it is not easy to perform; and for the patient it may be an uncomfortable experience, which may even, although rarely, culminate in a submaxillary salivary gland infection.

(To be continued)

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