Chapter 149: Tests of Facial Nerve Function

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In facial paralysis, as in most medical problems, history and physical examination usually provide information that is more useful than the results of any laboratory test. Sometimes, however, it is useful to monitor facial nerve function to know the severity of a facial nerve lesion or to localize it to a particular intracranial, intratemporal, or extratemporal site; tests of facial nerve function (most of which are electrical and topognostic) are then used.

Sunderland (1977) has provided a sample classification of nerve injuries based on the histology of the nerve trunk; it is helpful in understanding the results of electrical tests (Fig. 149-1). There are five classes of injury.

Class I. Pressure on a nerve trunk, provided that it is not too severe, caused conduction block, also referred to as *neuropraxia.* No disruption of axonal continuity occurs, and the connective tissue elements remain intact. When the pressure or other insult is removed, the nerve can recover quickly. During the conduction block, no impulses can cross the area of the lesion, but electrical stimulation distal to the lesion still produces a propagated action potential and a visible muscle twitch. Everyone has experienced an arm or leg "going to sleep"; this is a Sunderland class I injury.

Class II. A more severe lesion, whether caused by pressure or some other insult, such as viral inflammation, may cause wallerian degeneration. The axon degenerates distally from the site of injury to the motor endplate and proximally to the first node of Ranvier. In a class II injury the connective tissue elements remain viable, so regenerating axons may return precisely to their original destinations. Removal of the original insult permits complete recovery, but this is considerably delayed because the axon must regrow from the site of the lesion (or slightly proximal to it) to the muscle, at a rate of about 1 mm/day, before function returns. A class II injury is also referred to as *axonotmesis.*

Class III. If the lesion disrupts the endoneurium, wallerian degeneration occurs as in a class II injury, but in addition, the regenerating axons are free to enter the wrong endoneurial tubes or may fail to enter an endoneurial tube at all; this aberrant regeneration may cause incomplete recovery and synkinesis. Sunderland class III to V nerve injuries, in which aberrant regeneration can occur, are also referred to as *neurotmesis.*

Class IV. Perineurial disruption obviously implies an even more severe injury, in which the potential for incomplete and aberrant regeneration is greater. Intraneural scarring may prevent most axons from reaching the muscle.

Class V. A complete transection of a nerve, including its epineurial sheath, offers almost no hope for useful regeneration unless the ends are surgically reapproximated.

The Sunderland classification system is useful as an intellectual framework, but it is important to realize that even traumatic facial nerve lesions are usually mixed; that is, some fibers may be in conduction block while others are disrupted, with varying degrees of connective tissue injury. Electrical testing can distinguish class I from classes II to V lesions, but cannot distinguish class II (with an excellent prognosis for perfect spontaneous recovery) from class V lesions (with a poor prognosis for useful recovery without surgical repair).

Electrical Tests

When a conduction block (class I injury) exists, the patient is unable to move his face voluntarily, but a facial twitch can still be elicited by percutaneous electrical stimulation of the nerve distal to the lesion. The electrical response of the facial muscles to voluntary, mechanical, or electrical activation of the nerve may also be recorded. Tests based on these two principles - electrical stimulation and recording of the electromyographic (EMG) response - are useful in prognosis and in the selection of patients for different treatments. However, they are rarely useful in differential diagnosis. In Bell's palsy and traumatic facial nerve paralysis, electrical tests are most often used to identify patients whose nerves have begun to degenerate, because these patients may be candidates for decompression surgery. In fact, the use of these tests in the outpatient evaluation of facial paralysis is probably unnecessary unless one is prepared to recommend decompression in the event that degeneration occurs. However, intraoperative monitoring of facial nerve function (usually using EMG) is rapidly becoming standard in many types of intracranial and intratemporal surgery.

Nerve excitability test

The simplest and best-known test for facial nerve degeneration is the nerve excitability test (NET) introduced by Laumans and Jonkees (1963). The stimulating electrode is placed on the skin over the stylomastoid foramen with a return electrode taped to the forearm. Beginning with the normal side, electrical pulses (0.3 msec in duration) are delivered at steadily increasing current levels until a facial twitch is just noticeable. The lowest current eliciting a twitch is the threshold of excitation. Next, the process is repeated on the paralyzed side, and the difference in thresholds between the two sides is calculated.

In a simple conduction block, such as occurs after infiltration of the perineural tissues with lidocaine (Xylocaine), no difference exists between the two sides; the paralyzed nerve is as easy to stimulate (distal to the point of the conduction block) as is the normal nerve. After a more severe injury (Sunderland class II to V), in which distal axonal degeneration occurs, electrical excitability is gradually lost. Unfortunately, this takes 3 to 4 days, even after a total section of the nerve. This means that the findings of the NET always lag several days behind the biologic events themselves (Gilliatt and Taylor, 1959).

In most cases of Bell's palsy with complete paralysis, some degree of degeneration takes place (Esslen, 1976), evolvnig over a period of 1 to 2 weeks. The proponents of the NET thus urge frequent, even daily examinations so that any trend toward degeneration can be detected as early as possible. A difference of 3.5 milliamperes (mA) or more in thresholds between the two sides has been proposed as a reliable indicator of progressive degeneration and has been used as an indicator for surgical decompression. With this criterion, complete or incomplete recovery can be predicted with 80% accuracy (Laumans and Jonkees, 1963).

Some investigators insist on conducting two consecutive tests showing differences greater than 3.5 mA so as to reduce the chance of test error.

The NET is useful only during the first 2 to 3 weeks of complete paralysis before complete degeneration has occurred. It is unnecessary in cases of incomplete paralysis, in which the prognosis is always excellent - and the test will be normal when the area distal to the lesion is stimulated. If the paralysis becomes total, the test can determine whether a pure conduction block exists or whether degeneration is occurring, as indicated by progressive loss of excitability. Once excitability is lost and that result is confirmed by repeat testing, further excitability tests are pointless because clinically evident recovery always begins *before* any apparent electrical excitability returns. This disparity occurs because the regenerating axons are smaller, more irregular in size, and fewer in number than before the lesion occurred. Electrical stimulation is generally relatively ineffective in eliciting a synchronous and thus observable twitch in the early stages of regeneration. Similarly, if a paralysis that has become complete begins to recover clinically before any degeneration is noted, continuing stimulation is unnecessary because recovery will be rapid and complete.

Remember that partial degeneration and a bad outcome are not synonymous. In a Sunderland class II lesion, nearly complete recovery may occur without serious complications. Laumans and Jonkees (1963) state that even patients who show some degeneration (threshold difference greater than 3.5 mA) have a 38% chance for *complete* spontaneous recovery; the remainder develop complications such as permanent weakness (not total paralysis) and synkinesis.

Because relatively large intersubject variations occur in threshold, compared to the small differences between the two sides of a single individual's face, stating NET results in proportional terms rather than in absolute threshold differences may be more appropriate. For example, Melchese et al (1971) used as their criterion for decompression a 150% increase in threshold as compared to the normal side.

Maximum stimulation test

The maximum stimulation test (MST) is similar to the NET in that it involves visual (ie, subjective) evaluation of electrically elicited facial movements. Instead of measuring threshold, however, maximal stimuli (current levels at which the greatest amplitude of facial movement is seen) or supramaximal stimuli (current levels above maximal stimuli) are employed. The electrode type and placement and the nerve-stimulating equipment are the same as in the NET. Increasing current levels are used until maximal movement is seen, and the paralyzed side is compared to the normal side. As performed by May et al (1976), peripheral branches are the preferred site of stimulation, and the movements on the paralyzed side are subjectively expressed as a percentage (0%, . 25%, 50%, 75%, 100%) of the movement on the normal side.

The theoretical basis of the MST is that by stimulating all intact axons, one can estimate the proportion of fibers that have degenerated; this information should be a more reliable guide to prognosis and treatment than the NET is. Unfortunately, there are no good data comparing these or other electrical prognostic tests *in the same patients,* so this claim has not been proved.

According to May et al (1976), when MST remained normal in Bell's palsy, 88% of patients recovered completely; reduced movement presaged only a 27% chance of complete recovery; an absence of electrically stimulated movement was always associated with incomplete recovery.

The MST is painful for some patients; Molina (1977) suggests that using a pulse duration of less than 400 microsec (as opposed to the 1 msec proposed by May) can eliminate this discomfort, even though higher currents are required.

Electroneurography

In electroneurography (ENOG) the facial nerve is stimulated transcutaneously at the stylomastoid foramen, as in the NET, although a bipolar stimulating electrode is used. Responses to maximal electrical stimulation of the two sides are compared, as in the MST, but they are recorded electrically by a second bipolar electrode pair placed (usually) in the nasolabial groove. The average difference between the two sides in normal patients is only 3% (Esslen, 1976). Despite the name *electroneurography*, the responses recorded are actually compound muscle action potentials (CMAP) from the facial musculature itself. Some workers have used the term *evoked electromyography* (EEMG) synonymously with electroneurography. Clearly this method offers the potential advantage of an objective registration of electrically evoked responses, and the amplitude of response of the paralyzed side (in mV) can be expressed as a precise percentage of the normal side's response. If the amplitude of the response on the paralyzed side is only 10% as large as that on the normal side, one may estimate that 90% of the fibers have degenerated on the paralyzed side. However, this method has some practical difficulties that must be mastered before reliable results can be expected (Fisch, 1980). Test-retest errors in the 20% range have been reported by Raslan et al (1988), despite standardization of electrode positions. Most investigators would require a 30% or greater asymmetry (or change over time) to be considered significant.

The use of ENOG or other electrical stimulation tests to prognosticate in Bell's palsy has become widespread. The most valid data supporting the use of ENOG come from studies limited to cases of *complete* paralysis (incomplete paralysis is known to have an excellent prognosis). In 37 such cases May et al (1983) showed that severe ENOG amplitude reductions (to less than 10% of the unaffected side) were highly correlated with incomplete recovery. Although Canter et al (1986) could find *no* correlation of final outcome with ENOG in 23 patients with complete paralysis, they did not specifically test the predictive value of the 10% criterion widely used by other authors. More recently, Sillman et al (1990) found excellent correlations between ENOG and outcome in Bell's palsy (N=66) but not in traumatic facial palsy (N=29).

Electrical recording of the muscle response also offers the possibility of measuring latency, which is the time elapsed between stimulus and response. Only slight interest in this variable has developed; although one might expect a slowing of nerve conduction velocities to be an early indicator of degeneration, the available data are in conflict. Joachims et al (1980) state that increased latency in the first 72 hours (before any observable change in threshold or response amplitude) was a very reliable predictor of a poor outcome. However, Esslen (1976) showed that in 145 patients latency never increased before the fifth day and never preceded changes in evoked potential amplitude; he concluded that latency

measurements were of no clinical value.

The limitations described for the ENT - namely its inapplicability in cases of partial paralysis, after the beginning of clinical recovery, and after excitability has been lost - also apply to the MST and to ENOG. In acute facial paralysis, all these tests are useful only in tracking the early course of a completely paralyzed nerve until it begins to recover or shows complete loss of excitability.

Esslen (1976) has used ENOG to study the time course of Bell's palsy, finding that the acute phase rarely exceeds 10 days. Decreases in ENOG amplitude after the tenth day were always associated with substantial latency increases and were attributed to desynchronization of fibers rather than to increasing degeneration. Thus the time elapsed since the onset of paralysis must be taken into account in the interpretation of ENOG results. Patients reaching 95% degeneration (amplitude of response equals 5% of that on the normal side) within 2 weeks had a 50% chance of a poor recovery, whereas patients exhibiting a more gradual decrease in ENOG amplitude had much better prognosis.

Most proponents of ENOG use it mainly to obtain an early prognosis in acute facial paralysis (Bell's or posttraumatic) or to select patients for decompression surgery. Kartush (1989) has pointed out that ENOG can also document subclinical facial nerve involvement by tumors, especially acoustic neuromas. Because those of his patients who had ENOG evidence of nerve involvement (despite clinically normal facial movement) were more likely to have postoperative weakness, ENOG may be helpful in counseling patients about the risks of surgery and may also play a role in the selection of therapy when nonsurgical options exist. However, because ENOG amplitude reductions were highly correlated with increasing tumor size (which also predicts poorer facial nerve outcome), it is unclear whether ENOG really *adds* prognostic information when tumor size is known.

Electromyography

The recording of spontaneous and voluntary muscle potentials by needles introduced into the muscle is called *electromyography* (EMG). Its role in Bell's palsy is rather limited because it does not permit a quantitative estimate of the state of the nerve (the percentage of degenerated fibers). However, EMG may be helpful in certain other situations. Several authorities who favor decompression for Bell's palsy base their decisions for surgery primarily on NET (Laumans and Jonkees, 1963) or ENOG (Fisch, 1980), but they also require a confirmatory EMG. If the EMG shows voluntarily active facial motor units, despite a nearly complete loss of excitability of the nerve trunk, the prognosis for a good spontaneous recovery is excellent (Esslen, 1976). This application of EMG in the early stages of Bell's palsy is probably underused.

As previously discussed, after loss of excitability in a case showing degeneration, tests of electrical stimulation are no longer useful. However, EMG may give prognostically useful information during this phase of the illness. After 10 to 14 days, fibrillation potentials may be detected, confirming the presence of degenerating motor units. More useful are the polyphasic reinnervation potentials that may be seen as early as 4 to 6 weeks after the onset of paralysis. These precede clinically detectable recovery and predict a fair to good recovery (Esslen, 1976). Because very few surgeons advocate decompression surgery for Bell's palsy

so late in the course of the disease, this use of EMG is uncommon. It may be helpful in the assessment of long-standing facial paralysis, along with muscle biopsy (see the next section), to determine the possible success of substitution anastomosis or cross-facial anastomosis as a mechanism of restoring facial motion. EMG can also be helpful in assessing whether a nerve anastomosis, for example, one in the cerebellopontine angle, is unsuccessful. If no clinical recovery occurs and EMG shows no polyphasic reinnervation potentials at 15 months (or at 18 months at the latest), the anastomosis should be considered a failure and consideration given to another operation such as a substitution anastomosis.

Antidromic potentials

If a motor nerve is electrically or mechanically stimulated at some point between its cell body and its synapse on a muscle fiber, action potentials will be propagated in two directions: an orthodromic or antegrade impulse will travel distal toward the muscle, while an antidromic or retrograde impulse will travel proximally toward the cell body. The orthodromic impulse will cross the neuromuscular junction, resulting in an observable muscle contraction and a recordable compound muscle action potential. Although the antidromic impulse will not cross a synapse, stopping at the cell body of the motor neuron, it can be recorded by electrodes on the proximal nerve (near-field) or at a distance (far-field). Kashima et al (1990) recorded electrical responses from the geniculate ganglion region in guinea pigs while stimulating the facial nerve at the stylomastoid foramen. These near-field antidromic responses were reliably altered by surgical lesions placed between the stimulating and recording electrodes.

Far-field antidromic potentials have been recorded from the scalp after electrical stimulation near the stylomastoid foramen, in hopes that this would yield a noninvasive test of the integrity of the intratemporal and/or intracranial portions of the facial nerve. Unfortunately, these responses are difficult to record and of questionable origin. Metson (1988) concluded, based on his studies in cats, that the intracranial nerve was the main generator, but Kartush egt al (1987) found (in dogs) that the mastoid segment was primarily responsible. No useful clinical application has been reported.

Acoustic reflex evoked potential

Hammerschlag et al (1987) reported a scalp-recorded potential at 12 to 15 msec latency in response to acoustic stimulation contralateral to the recording site, and attributed this to facial motor pathway activation. The response persisted after paralysis during anesthesia, and the authors proposed its use for intraoperative monitoring of facial nerve function. However, the response was extremely small (much lower in amplitude than the auditory brainstem response), which would make it difficult and slow to record, requiring prolonged averaging. This response seems unlikely to be useful, because audible EMG monitoring (discussed later) gives essentially instantaneous feedback for intraoperative assessment of facial nerve function.

Magnetic stimulation

A rapidly varying magnetic field, produced by a surge of current in a coil placed over the skin, will induce electrical currents in underlying tissue, and can be used to stimulate nerves. This method offers two potential advantages over conventional electrical stimulation of the facial nerve. First, the nerve can be maximally stimulated without pain or discomfort. Second, if the coil is placed in the temporoparietal area (transcranial stimulation), it appears that the nerve is stimulated in the region of the geniculate ganglion (Kartush et al, 1989; Schriefer et al, 1988) which could obviously be useful for site-of-lesion determination. However, Schriefer et al (1988) found no response to transcranial magnetic stimulation in serial follow-up of two patients with Bell's palsy, even after rapid and complete clinical recovery (2 and 3 weeks respectively), suggesting that this technique may not be useful for prognostic purpose.

Muscle biopsy

Although obviously not an electrical test of facial nerve function, muscle biopsy is included here to remind clinicians of the importance of verifying the existence of viable facial musculature before trying to bring neural impulses to it. Biopsy is a useful adjunct to EMG in patients with very longstanding paralysis (several years) in whom hemifacial atrophy is evident, and in infants with congenital facial paralysis in whom unilateral absence of the facial nerve is suspected.

Facial Nerve Monitoring

During the 1980s, audible EMG monitoring of facial nerve function became routine in many centers (perhaps most) during acoustic neuroma surgery, and its application is spreading to other operations involving risk to the facial nerve, as well as to operations involving other cranial nerves. Although it is possible for the surgeon or an associate to watch for facial movements in response to mechanical or electrical stimulation of the nerve, simple observation will fail to detect many small muscular contractions and demands constant vigilance. Electrodes in or near the facial muscles, on the other hand, record EMG potentials that can be amplified and made audible by the use of a loudspeaker. The surgeon's ears can then monitor the facial nerve while his hands and eyes operate. Harner (1986) points out that this technique is not really new, but it has only recently become popular.

Although needle electrodes, not surface electrodes, are usually used during surgery, the activity being recorded is the same compound muscle action potential (CMAP) that is recorded in ENOG. When the surgeon stimulates the nerve electrically, the CMAP can be seen on an oscilloscope (if visual display is being used), and the loudspeaker emits a characteristic "thump". Gentle mechanical stimulation, such as touching the nerve with an instrument, will produce a similar sound; stretching the nerve or thermal stimulation, such as irrigation, will often produce a prolonged irregular series of discharges that sound like popcorn popping. Prass et al (1987) have labeled these characteristic sounds "bursts" and "trains". Learning to identify these sounds is easy and gives surgeons instant feedback regarding the location of the facial nerve as well as their handling of it.

Electrical stimulation can be done by using either a monopolar electrode or a bipolar electrode (such as is used for bipolar electrocautery), which is insulated except at the tips. Monopolar stimulation activates a wide area (depending on current intensity) and can be quite sensitive for locating and mapping the facial nerve. However, stimulation of adjacent nerves (vestibular and auditory) with a monopolar electrode will often activate the facial nerve as well, leading to a false-positive identification. Commercially available monopolar electrodes are flexible with blunt tips, which makes them convenient for access to cramped areas. With bipolar stimulation, the current is mostly confined to the tissue between the forceps tips; with wide tip separation, this is very similar to monopolar stimulation, but with the tips close together, bipolar stimulation is quite specific. Both types are satisfactory if the user understands these differences.

As with any technical aid, there are pitfalls. Failure to obtain audible facial nerve responses can be caused by detached electrodes, an inoperative stimulator, or many other causes (including a nonfunctional nerve). Conversely, stimulation of the trigeminal nerve can occasionally cause confusion; the facial muscle electrodes may pick up EMG signals from the nearby masseter muscle. Electrode position should always be checked by voluntary movement, transcutaneous electrical stimulation, or simply by tapping over the stylomastoid area before the surgical site is draped. The most useful intraoperative check on the monitoring equipment is the stimulus artifact, a "ticking" sound produced when the stimulating electrode touces tissue remove from the facial nerve. This sound is quite distinct from the "thump" produced by nerve stimulation and confirms that the stimulator is delivering current, that the electrodes are in place, and that the loudspeaker is working.

The fact that audible EMG monitoring makes facial nerve preservation in acoustic tumor surgery easier, faster, and probably more successful has become widely accepted on an anecdotal basis, and Harner et al (1988) have shown that postoperative facial nerve function is better in patients who have been monitored. Although most surgeons use EMG monitoring, it is worth mentioning that others have monitored facial contractions by using a motion sensor connected to an electronic circuit that produces an audible signal (Silverstein et al, 1988), or even by using small bells that are sutured to facial skin (Williams and Lehman, 1988).

Kartush (1989) states that both excessive train activity intraoperatively and elevated thresholds for nerve stimulation at the end of the operation predict poor outcome; this has been our experience as well.

Topognostic Tests

The topognostic tests discussed here were initially intended to reveal site of lesion by using a simple principle: lesions below the point at which a particular branch leaves the facial nerve trunk will spare the function subserved by that branch. An injury to the nerve in the face thus does not affect lacrimation, salivation, taste, or the stapedius reflex. Conversely, the combination of facial paralysis and decreased lacrimation can be caused only by a lesion in that part of the nerve where voluntary motor and parasympathetic fibers to the lacrimal gland run together: from the cerebellopontine angle to the geniculate ganglion.

In complete focal lesions, such as commonly occur after trauma, the topognostic tests can be relied upon. Bell's palsy, however, is usually a mixed lesion with varying degrees of conduction block and degeneration changes; one would therefore not expect topognostic tests to provide very precise information about the level of the lesion, which is itself partial. Indeed, many investigators have noted paradoxic and misleading results in patients with Bell's palsy. These tests are more often used as an index of severity for the purposes of prognostication and treatment selection.

Lacrimal function

Schirmer's test has the advantages of simplicity, speed, and economy: the physician places a folded strip of sterile filter paper into the conjunctival fornix of each eye and then compares the rate of tear production of the two sides. Normally the portion of the filter paper in contact with the conjunctiva acts as an irritant, stimulating an increased flow of tears, which are then wicked along the filter paper strip by capillary action. The length of the wetter portion of the strip after a fixed interval (usually 5 minutes) is measured and is proportional to the volume of tears produced. It is apparent that a defect in either the afferent or efferent limb of this reflex could cause a reduced flow. The reflex is consensual; that is, the irritating stimulus in *either* eye causes tearing in both eyes, and a unilateral sensory (trigeminal) deficit will reduce tearing bilaterally. However, unilateral corneal anesthesia reduces tearing asymmetrically, with a greater reduction on the anesthetized side (Crabtree and Dobie, 1989); when a sensory deficit is present, one should consider bilateral corneal anesthesia and stimulation of lacrimation by other noxious stimuli (for example, inhalation of ammonia).

Schirmer's test is usually considered positive if the affected side shows less than half the amount of lacrimation seen on the normal side. Indeed, this corresponds well to the normative data of Fisch (1977), who found that 95% of normal subjects had "relative reduction" scores of less than 30%; this means that the lesser response was more than 54% of the greater response. Fisch also points out that tearing is often inexplicably reduced bilaterally in Bell's palsy, and this bilateral reduction even persists after unilateral resection of the geniculate ganglion. Thus, judging not only the symmetry of the response but also its absolute magnitude is important: a total response (sum of the lengths of wetter filter paper for both eyes) of less than 25 mm is considered abnormal.

Fisch (1977) correlated the results of Schirmer's test and ENOG in patients with Bell's palsy and herpes zoster oticus and found that all cases with a degeneration by ENOG of 90% or greater also had an abnormal Schirmer's test. However, Schirmer's test did not give an indication of degeneration earlier than ENOG and was abnormal in several cases in which ENOG correctly predicted a good spontaneous recovery.

May (1982) added Schirmer's test to salivary flow and MST in his battery of prognostic tests; a decrease to 25% of normal in any of these is said to predict a 90% change of a poor recovery.

Stapedius reflex

The nerve to the stapedius muscle branches off the facial trunk just past the second genu in the vertical (mastoid) part of the nerve. The stapedial reflex and the methods of testing it are described in detail in Chapter 148. The most common use of acoustic reflex testing is to assess the afferent (auditory) limb of the reflex, but in cases of facial paralysis the same test is used to assess the efferent limb. An absent reflex or a reflex that is less than half the amplitude of the contralateral side is considered abnormal.

The reflex can be elicited by either ipsilateral or contralateral acoustic stimulation, or in cases of bilateral severe hearing loss, by tactile or electrical stimulation. It is absent in 69% of cases of Bell's palsy (84% when the paralysis is complete) at the time of presentation (Koike et al, 1977); the reflex recovers at about the same time as clinically observed movements do. The prognostic value of this test therefore seems limited.

Taste

Because the chorda tympani carries fibers subserving taste from the anterior two thirds of the tongue, many investigators have studied abnormalities of taste in Bell's palsy. Psychophysical assessment can be performed with natural stimuli, such as aqueous solutions of salt, sugar, citrate, and quinine, or with electrical stimulation of the tongue. The latter, termed *electrogustometry,* has the advantages of speed and ease of quantification. In normal subjects, the two sides of the tongue have similar thresholds for electrical stimulation, rarely differing by more than 25% (Diamant, 1977). The major problem with taste testing is that the results of this test will be abnormal in almost *all* patients who are in the acute phase of Bell's palsy (Tomita et al, 1972); thus this test cannot be used to select patients with a poor prognosis. However, taste function apparently *recovers* before visible movement in some cases, so if the results of electrogustometry are normal in the second week or later, clinical recovery is imminent.

Salivary flow

The salivary flow test requires cannulation of the submandibular ducts and the comparison of stimulated flow rates on the two sides. It is somewhat time-consuming and unpleasant, especially if performed repeatedly; this is probably the main reason why it is not widely used. Obviously, reduced submandibular flow implies a lesion at or proximal to the point at which the chorda tympani nerve leaves the main facial trunk; this is variable and may occur anywhere in the vertical (mastoid) portion of the nerve.

Ekstrand (1977) states that reduced salivary flow (less than 45% of the normal side, after stimulation with 6% citric acid) correlates well with eventual outcome in Bell's palsy. Complete or incomplete recovery could be predicted with 89% accuracy. It is not clear whether these data, all collected within 10 days of the onset of the paralysis, provide an earlier or more reliable indication of prognosis than electrical or other tests do.

May and Hawkins (1972) stated that salivary flow *does* change sooner than NET in idiopathic facial paralysis. They argued that a flow rate of 25% or less compared to the contralateral side was an indication for surgery.

Salivary pH

At least one report (Saito et al, 1977) has shown that a submandibular salivary pH of 6.1 or less predicts incomplete recovery in cases of Bell's palsy. Presumably only the duct on the affected side needs to be cannulated because in this study, the control sides of all had pH levels of 6.4 or more. The overall accuracy of prediction was 91%. This test seems quite attractive for another reason: pH measurements are likely to be more reliable than flow measurements. Unfortunately, there has been very little reported experience with pH measurements and it is not known whether this technique gives an earlier prognosis than other tests.