



New method for the objective evaluation of injury to the lingual nerve after operation on third molars

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Summary Existing tests of function of the lingual nerve are either subjective or, when they elicit the jaw-opening reflex, are dependent on the cooperation of the subject. We report a study in 12 healthy volunteers and 12 patients with iatrogenic injury to the lingual nerve. A bite block (containing stimulating electrodes) was held between the teeth and the tongue was held on to the electrodes by suction. When the lingual nerve was intact, an electrical stimulus elicited brief inhibition of masseteric electromyographic activity. Local analgesia and iatrogenic injury to the lingual nerve altered nerve conduction and caused a reduction in reflex inhibition. Two methods, compatible with limited numbers of applications of the stimulus, were used to quantify responses. One used an indirect measurement of intervals between action potentials of muscle and the other used a measurement of rectified signals falling below the mean amplitude before and after the stimulus. Both methods gave values that correlated with subjective sensations. The first gave an estimate of the probability of defining major malfunction of the nerve objectively; the second gave a linear measurement that allowed recovery of the nerve to be followed.

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Introduction

Many studies have reported that the removal of lower third molars causes temporary injury to the lingual nerve in up to 23% of patients and permanent injury in up to 2%.¹ It is therefore desirable

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to have an objective test of function of the lingual nerve. Conventionally, the integrity of a nerve is tested by applying a stimulus to skin or mucosa and asking the patient what he or she feels. This is used as an indicator of the integrity of the sensory nerve fibres. However, in the orofacial region, the sensitivity to such tests varies widely across different sites² and the results of the tests do not correlate well with the damage to the afferent nerve that is found at operation.³ There is also the problem that these tests fail to differentiate between sensory loss resulting from a peripheral lesion and sensory loss from a central lesion. Afferent nerve function can, however, be assessed without involving a conscious response, simply by testing for the presence or absence of a local reflex (Fig. 1). If the central and efferent components of the reflex arc can be shown to be intact but the reflex is absent, then the afferent limb must be the defective element. In the orofacial region, an appropriate test is the jaw-opening reflex.⁴ The reflex comprises inhibition of the motor fibres that cause the jaw to close,^{5,6} and it can be elicited by electrical stimulation of various orofacial sites. The response is expressed as a brief reduction in the extent of the

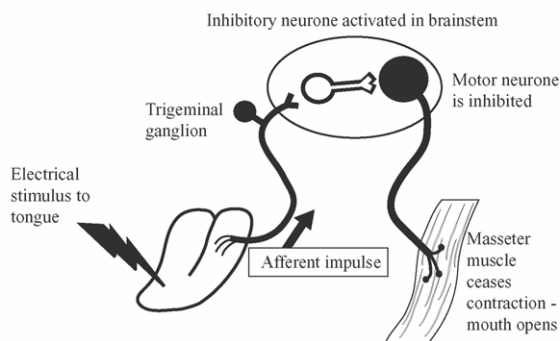
existing electromyographic (EMG) activity produced by conscious clenching of the muscles that close the jaw after the stimulus. When the stimulus is applied to an uninjured site, the inhibitory reflex is expressed bilaterally,^{4,7,8} so the central and motor components of the reflex can be tested by stimulation on the side on which the lingual nerve is intact. Provided that the central and motor components of the reflex are intact, stimulation of the tongue on the side of the damaged nerve will then test for adequate conduction in the afferent nerve.

Many different methods have been advocated for measuring the inhibition of the reflex by EMG activity. These vary from visual inspection to computer-based statistical methods.^{6,9,10-13} The EMG activity is usually measured by full-wave rectification and the data from successive stimuli are averaged. Reliable objective measurement of the reflex therefore requires the responses to a large number of stimuli (sometimes up to 72) to be averaged and, in all cases, the subject has to cooperate by attempting to maintain a steady level of EMG activity in the masseter muscle.^{11,12} However, after iatrogenic injury to the lingual nerve, some patients are apprehensive and may be unable to cooperate satisfactorily, particularly when there is an associated dysaesthesia or glossodynia. An additional problem, in medicolegal cases, is that some patients may not be motivated to cooperate fully.

In most published methods, the mean of the rectified value of EMG activity before stimulation is used as the reference with which the value after stimulation is compared. However, the value of the EMG signal before stimulation is substantially variable about its mean, so a reflex can be considered to be present only if the mean value after stimulation is significantly lower (more than 2 S.D.) than the mean before stimulation.¹¹ These conditions can be met satisfactorily in experimental subjects by using a large number of stimuli. If, however, the number of stimuli has to be limited to be acceptable to a patient with, for example, glossodynia, the S.D. of the value before stimulation increases considerably. The problem then is that weak inhibition, such as may be present in the initial stages of recovery of the lingual nerve, may not reduce the value of EMG activity after stimulation to a significant extent. Conventional methods can therefore make it impossible to show the presence of a weak reflex.

Our aim was to develop and test methods of establishing the presence or absence of a weak jaw-opening reflex, using the small number of stimuli likely to be used in subjects who were unable to cooperate fully. The method was tested on healthy volunteers, in some of whom local analgesia was used to mimic a lesion of the lingual nerve.

A. Healthy lingual nerve



B. Injured lingual nerve

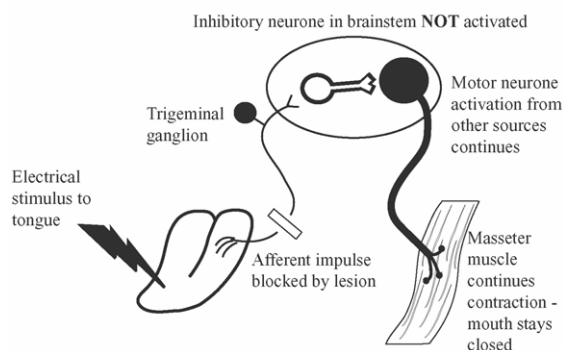


Figure 1 Diagrammatic representation of the neural pathway of the human jaw-opening reflex before and after a lesion of the lingual nerve. See Introduction for further explanation.

Method

Experiments on volunteer subjects ($n=12$, 8 men and 4 women, age range 18–64, mean 32 years) were made in accordance with the recommendations of the local ethics committee. All volunteers were healthy, fully dentate, and with no history of craniomandibular disorders. Local analgesia was used to mimic the effects of a lesion of the lingual nerve in 6 of them. A group of patients with injuries to the lingual nerve after operations on third molars ($n=12$, 9 men and 3 women, age range 19–58, mean 30 years) were studied to establish the nature of the jaw-opening reflex when elicited through a damaged lingual nerve. None of the patients had medical conditions that might affect neural function, and none had previous experience of the research method. All patients had reduced and altered sensation in the distribution of the injured lingual nerve with raised thresholds for two-point discrimination and light touch and decreased counts of fungiform papillae.

The subjects were seated upright in a dental chair in a quiet environment with no sight of the display of data. One side of the tongue was stimulated using an electrical stimulus set at 30% above the strength of stimulus that, when applied to an area innervated by an undamaged nerve, only just elicited the reflex (assessed visually from the display on the computer). The constant current electrical stimuli (usually in the range 3–5 mA) lasted 0.1 ms and were repeated 8–16 times at 2-s intervals. The stimulus was generated by a MacLab 8e system using Scope 3.5 software (ADI Electronics Ltd., Hastings, UK), with an isolation unit and constant current module.

The stimulating electrode consisted of 1 mm diameter, bipolar silver–silver chloride electrodes separated by 1 cm and housed within a perspex bite block (Fig. 2), the occlusal surfaces of which were covered with a layer of wax. When placed between the first molars, the bite block resulted in an interincisal distance of 10–15 mm and the electrodes contacted the edge of the tongue. A well (with a raised rim) surrounded each electrode and was connected to a vacuum line. During the test the lingual mucosa was therefore held firmly in contact with the stimulating electrodes, with the insulating perspex rims, and with the surrounding shield (earth electrode). Any loss of contact with the tongue was signalled immediately by a hissing sound.

The EMG activity of the masseter muscles was recorded bilaterally from bipolar surface electrodes (Medicotest N-00-S Blue sensor disposable

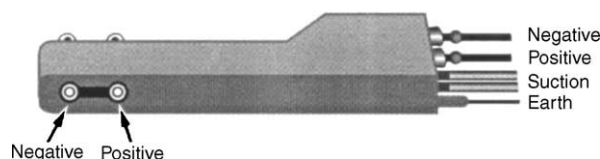


Figure 2 Tongue electrode-bite block. This was placed in the mouth with the electrodes (anode and cathode marked positive and negative) facing the edge of the tongue. The source of the stimulus was connected to the end of the electrode system by miniature plugs; a buried wire led from each socket to a silver rod 2 mm in diameter that had silver chloride at its tip. Suction was applied independently to the two wells surrounding the silver–silver chloride electrodes, the height of which could be adjusted. The metal strip covering the surface that contacted the edge of the tongue formed a shield and an earth electrode.

electrodes). These were arranged over the midline of the masseter muscles, separated vertically by 2 cm. The EMG signals were then differentially amplified (Bioamp with MacLab/8e system; AD Instruments, Hastings, UK) and band-pass filtered (10 Hz to 1 kHz). The EMG data were saved on the MacLab 8e system together with the stimulus. Scope 3.5 software was used to full-wave rectify the EMG signals and (solely for immediate visual inspection) to calculate the mean response to 4, 8, or 16 successive stimuli.

EMG activity was recorded while the subject clenched firmly on the wax-covered housing of the stimulating electrode held between the first molars. Auditory feedback by loudspeaker was used to assist the patient (and warn the operator of any failure) in maintaining a fairly constant extent of EMG activity. This was set at 20% of the peak to peak amplitude found in maximum biting and it produced signals over the masseter that, when full-wave rectified, had mean values in the range 50–150 μ V. While these conditions were regularly achieved in volunteer subjects, that was not always the case in patients with lesions of the lingual nerve. The EMG activity was digitised at 10 kHz (mainly to ensure recording of the stimulus artifact) and recorded for 80 ms before, and for 176 ms after, each stimulus so the visual display had a duration of 256 ms (Fig. 3a). Each 256 ms length of record, associated with a single stimulus, is referred to as a 'trial' while a set of 8 or 16 consecutive trials is referred to as a 'test group' (Fig. 3a).

The full-wave rectified EMG data generated in each test group were exported to an Excel spreadsheet for processing in two ways, using a 'runs counting' test and a 'normalised inhibition' test (Fig. 3b).

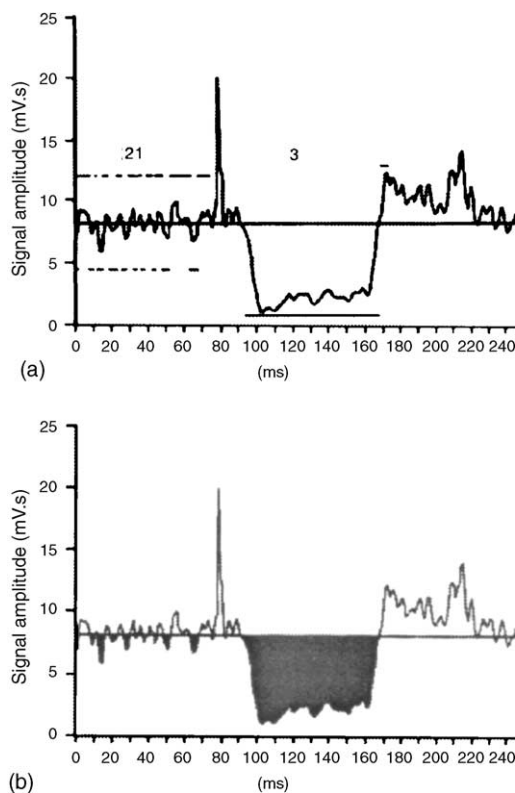


Figure 3 Quantification of reflex inhibition: a diagram of the two systems of reflex EMG measurement. In the examples shown, a full wave rectified EMG signal showing a series of peaks and troughs before the stimulus (at 80 ms) is followed by a maintained drop in the level of EMG activity. The reflex shown is typical of that elicited in a normal subject with a strong stimulus. The horizontal line indicates the mean/median of the activity before stimulation. (a) The number of signal reversals occurring about the median were counted in the periods before and after stimulation in each trial. In the diagram, 21 runs (20 reversals = 21 runs) are found in the period before stimulation and 3 (2 reversals) after stimulation. Only the runs in the first 80 ms after stimulation are shown. (b) Two sets of values, relative to the mean during the period before stimulation, were derived from the averaged signals of 8 or 16 trials: (i) the sum of all deviations below the mean in the pre-stimulus period and (ii) the sum of all deviations below the mean in the period before stimulation. These values were then divided by the respective durations of their periods and the normalised post-stimulus values were expressed as a percentage of the normalised pre-stimulus values.

Runs counting test

This test was used for every trial. The median of the values in the 80-ms control period was computed and this amplitude was used as the basis for dichotomising all the rectified data of that 256 ms trial. The data therefore resolved into a series of

values above and a series below the control median value. Each series constituted a run, so that the number of runs was equal to the number of reversals plus one.¹⁴ The number and the length of the runs reflected the varying intervals between, and the duration, of the rectified muscle action potentials. The number of runs in the 80-ms control period was then compared with the numbers of runs in two consecutive periods of the same duration after the stimuli. In each trial the number of runs in a particular period after stimulation was therefore either less than that in the control period or it was not. If no reflex was present, the number of trials in which the period after stimulation fell into the 'less than' or the 'not less than' categories should be equal or nearly so. On the other hand, recurring inequality between the number of runs in the control period and the period after stimulation indicated the presence of a reflex. The probability that there was a recurring inequality between the two periods in a number of trials was then tested by McNemar's test for the significance of changes,¹⁴ which yields a chi square value.

Normalised inhibition test

This test gave a measure of the inhibition that resulted from stimulation, normalised to the deviations below the mean signal in the control period. Successive constant time (5 ms) reset integrals were derived from the rectified signals of each trial and then averaged across the test group for each 5 ms interval. The starting point in measuring the value of activity caused by stimulation was to establish the grand mean of all activity preceding the stimulus, which will be referred to as the control value. To avoid the problem of measuring EMG activity when patients could not or would not maintain the desired extent of background EMG activity in successive tests, we adopted a new approach. Unlike traditional measures that concentrate on the absolute extent of EMG activity (measured from a zero baseline) we concentrated on the extent to which the fluctuating signal dropped below the pre-calculated control value. In view of the potentially differing latencies of inhibition in subjects with intact (Fig. 4a and b) and damaged nerves (Fig. 4c–f), we compared the mean deviations below the control value after stimulation (150-ms duration) and the same measurements in the control period (75-ms duration). The control period and the period after stimulation were separated by 15 ms, starting 5 ms before the stimulus and ending 10 ms after it. This ensured that no artifact from the stimulus contaminated the process of quantitation. The mean of the deviations below the control mean in the period

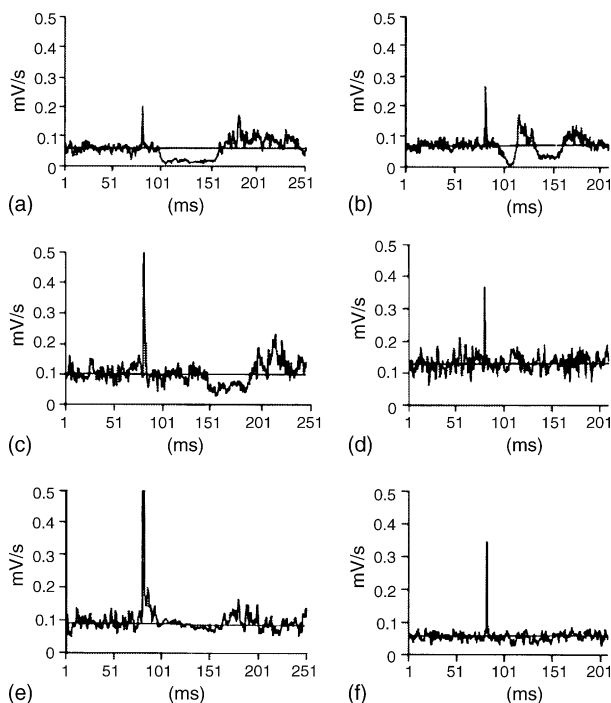


Figure 4 Variations in the patterns of reflex EMG activity on opening of the jaw in the masseter of normal subjects (a and b) and in patients with lesions of the lingual nerve (c–f). The stimulus used was 30% greater than the threshold stimulus for eliciting the reflex from the opposite side of the tongue and was timed to occur 80 ms after the start of each record. Other patients with lesions of the lingual nerve showed patterns that were similar to those in panels d–f (y-axis: mV/s; x-axis: ms—5 ms has been clipped from the end of these records).

after stimulation was normalised to the mean of the same values in the period before stimulation and expressed as a percentage. This measurement will be referred to as “normalised inhibition”. If no reflex response was present, the signals before and after stimulation should, on average, be the same so that the ratio of the two amplitudes would be 1 and the percentage value would be 100. If a reflex were present, the level of activity in the period after stimulation would fall further below the control mean and the normalised value would consequently be greater than 1, more than 100%.

In 6 of the 12 volunteer subjects, injection of lignocaine (Lignostab 1/80,000 adrenaline) was used to anaesthetise the lingual nerve on one side during a series of measurements. At each stage (1, 2, 3, 4, 5, and 10 min after the injection and then at 10-min intervals until recovery, Fig. 5) each subject estimated the sensation on the injected side compared with the non-anaesthetised side (scored as a percentage sensation, the stimulus being light stroking of the tongue).

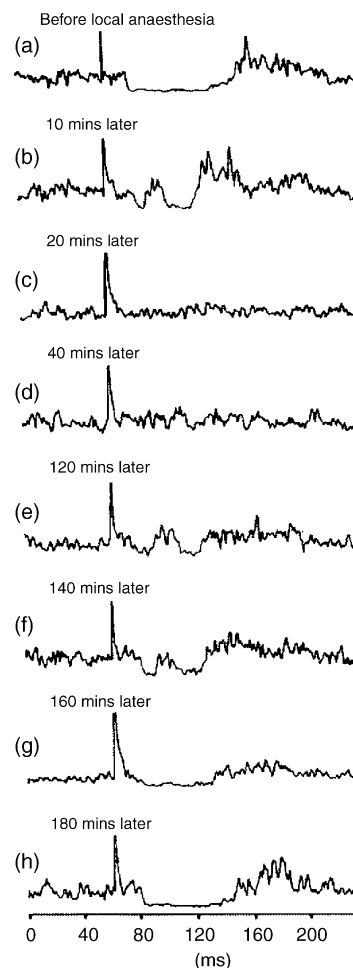


Figure 5 The effect of local analgesia of the lingual nerve on the pattern of reflex response. Averages of 16 full-wave rectified responses are shown. In this series of records the stimuli were applied 60 ms after the start of the record.

Results

The patterns of reflex activity in normal subjects were similar to those reported in other studies,^{5,15,16} there being responses with a single clear period of inhibition (Fig. 4a) and also responses containing a short period of increased signal activity that split the period of inhibition (Fig. 4b). In patients who complained of absent or abnormal sensation in the distribution of the lingual nerve after removal of a lower third molar, the reflex was weak or absent or showed unusual patterns of inhibition (Fig. 4c–f). Local analgesia of the lingual nerve in healthy subjects caused a gradual reduction in the size of the reflex response, a splitting of the inhibitory period, and eventually the disappearance of the inhibition (Fig. 5). These changes occurred in parallel with the subjects’ reports of the loss, and subsequent recovery, of sensation.

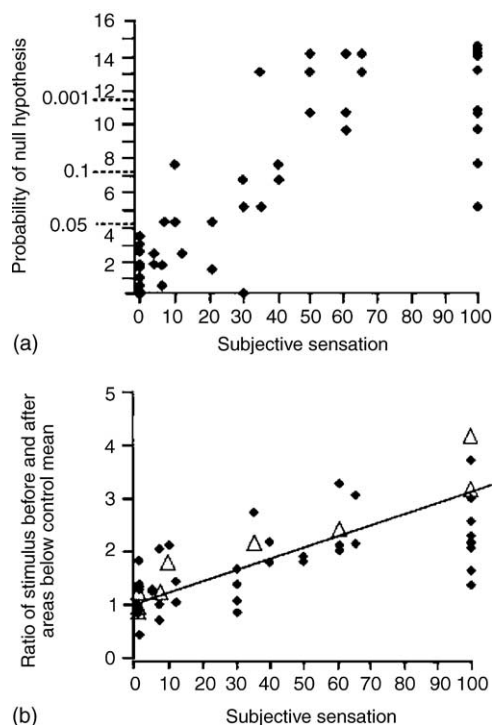


Figure 6 Graphs of values of runs counting test compared with subjective function and normalised inhibition compared with subjective function. In panel (a), the values of the runs counting test were first obtained from 12 control subjects with 100% subjective function. The test values at less than 100% subjective function are those of 2 series of responses from each of 6 subjects at varying stages after local anaesthesia of the lingual nerve. Note that increasing integer values of chi square on the vertical scale show a decreasing probability of the data before and after stimulation being the same i.e. there is an increasing probability of a reflex being present. The decimal values show the equivalent p values for 16 trials. Values less than 4 on the integer scale ($p > 0.05$ when 16 trials are used) correspond to a low probability that data before and after stimulation differ i.e. it indicates that the reflex is absent. In panel (b), the raw data were from the same series of experiments as in panel (a), but processed to obtain normalised inhibition values. The variation around the regression line is partly the result of inter-individual differences during the onset and recovery from local anaesthesia (6 subjects) but also reflects the influence on the regression line of the differences among the 12 normal subjects. The data points for one series of responses from a single individual are shown as open triangles.

In 11 out of the 12 normal subjects in this study, the runs counting count test indicated that the post-stimulus 80 ms period (corresponding directly to the inhibition) differed from the pre-stimulus period at the $p < 0.01$ level and, even in the one remaining normal subject, it differed at the $p < 0.05$ level (Fig. 6a). When the runs counting tests were

applied to the records obtained during the onset of, and recovery from, local analgesia there was an apparently linear fall in the test statistic with reduction in subjective sensation below 70% (Fig. 6a). When the subjective estimation of the difference in sensation between the two sides was below 5%, all runs counting test values were non significant; there were no significant differences in the signals before and after stimulation.

Under the same conditions of local analgesia, the values for normalised inhibition varied linearly with the reduction and the subsequent recovery of sensation (Fig. 6b). At zero subjective function the regression line for all data points passed close to 1 (100%) on the normalised inhibition axis, when signals before and after stimulation should be equivalent. The normalised inhibition then increased to a mean of nearly 3 (300%) at 100% subjective function.

Discussion

In contrast to the pattern of the jaw-opening reflex in subjects with healthy lingual nerves (Fig. 4a and b), the responses in subjects with lesions of the lingual nerve were absent or abnormal (Fig. 4c–f). Similar loss of the reflex also follows injury to the nerve caused by mandibular fractures¹⁷ or orthognathic operations.¹⁸ Local anaesthesia of the lingual nerve produced similar patterns of change in the reflex, although the initial change (Fig. 5a and b) was similar to the variation found in normal unanaesthetised subjects (Fig. 4a and b). Profound anaesthesia (Fig. 5c and d) and the early stages of recovery from local anaesthesia (Fig. 5e) produced patterns of response (or lack thereof) similar to those in subjects with iatrogenic nerve lesions (Fig. 4d–f) although big changes in latency of the type shown in Fig. 4c were not seen. The latency of the pathological response in Fig. 4c (of the order of 75 ms) is significantly longer than that of the second or late trigeminal inhibitory response reported elsewhere in healthy subjects (47–48 ms,¹⁶ 50 ms,⁸ 35–50 ms^{4,19} and 45 ms¹⁰). With the exception of the single case with unusually long latency after iatrogenic injury, local anaesthesia seems to be a reasonable and rapidly reversible model of an iatrogenic nerve lesion.

While it is a simple matter to distinguish visually between the extremes of reflex response, namely between clear inhibition (Fig. 4a and b) and no response (Fig. 4f), objective measurement of the apparently absent responses (Fig. 5c and d) and of reflex responses in the intermediate stages of

recovery (Fig. 5e and f; Fig. 4d) is more problematic. It is, however, the ability to quantify the reflex responses objectively (and so follow the recovery of the afferent nerve over a period of time) that is likely to be clinically useful.

We tested two methods of quantitation of the reflex in this study to see if either was more sensitive or more proportionate to different levels of subjective sensation. The main differences between the two methods of quantitation were, firstly that the runs counting method was designed to be sensitive to changes in the duration of (and intervals between) the compound muscle action potentials in individual trials, and secondly that the normalised inhibition method was sensitive to reductions in the amplitude of the rectified integrated reflex EMG, averaged over 16 trials.

The runs counting method had the advantage that the test result gave a value of probability. If this was greater than a critical value ($p=0.05$) then no reflex was deemed to be present (Fig. 6a). Although it might seem from Fig. 6a that lower probability values (such as $p=0.01$) would indicate partial loss of the reflex, the test unfortunately yielded values up to $p=0.05$ when applied to a larger group of normal subjects. The runs counting test also looks imperfect when it is used as a continuous measure of recovery of function of the nerve because the probability of a reflex being present becomes nearly certain when subjective recovery is only 60%. The test does, however, give a clear threshold ($p > 0.05$) for deciding whether or not the reflex (even a weak one) is absent. This suggests that its main use is in establishing whether or not a nerve was severely damaged (for medico legal purposes), and whether a nerve was taking the first steps to recovery. Because apprehension or anticipation of pain can reduce the extent of reflex inhibition substantially,²⁰ the runs counting test (which is sensitive primarily to the temporal changes in the structure of the EMG signal) might be preferable when dealing with a patient with glossodynia or nervousness to a test that measures the amplitude of EMG signals.

Objective tests of neurosensory function are quantitative rather than qualitative in the sense that they exclude cortical interpretation of the stimulus by the patient, so avoiding the possibility of bias. Liguori et al.²¹ using direct stimulation of the nerve, reported a reduced or delayed sensory action potentials in the lingual nerve associated with injury. Nerve conduction tests are, however, intrusive, time-consuming, and are not suitable for use in the outpatient department.

Somatosensory evoked potentials reflect the result of nerve conduction using scalp electrodes to

detect the cerebrocortical potentials that result from afferent volleys that reach the somatosensory cortex.²²

Thermographic images are symmetrical in normal subjects. After injury to a peripheral nerve (when sympathetic fibres travel within the nerve), sympathetic tone is reduced and vasodilatation ensues, which increases the peripheral blood supply and emission of heat from the surface. At present the images are diffuse and non-specific and further work is necessary to evaluate the sensitivity of this method of diagnosing nerve injuries.²³ In any event, as the mucosal surfaces have raised temperatures compared with skin, this may also limit the application of this method to intraoral tissues. The possible advantage of the EMG method over these conventional objective methods is that quantitative evaluation of the nerve injury is possible.

The normalised inhibition test produced results that were similar to those from the runs counting test but were more clearly linearly related to subjective function. It also had the advantage of a more intuitive scale; a ratio of 1 or a percentage of 100 indicated that there was no measurable difference between the periods before and after stimulation (meaning that the reflex was absent) and all higher ratios or percentages indicated varying degrees of the reflex. The variation in values around the regression line in Fig. 6b is partly the result of inter-subject variation. The plotted recovery of reflex in a single subject with recovery of sensation (shown by open triangles in Fig. 6b) suggests that the recovery or persisting dysfunction of an injured lingual nerve might be followed easily by looking at the trend in such values over a period of time. Such comparisons must, however, be made only between normalised inhibition values calculated from exactly the same number of trials on each occasion. Providing the 'normalised' inhibition is so calculated, the values can also be used to compare the reflex responses in different groups of patients.

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