Nerve Excitability Testing: Technical Pitfalls and Threshold Norms Using Absolute Values

George A. Gates, MD

Percutaneous stimulation of the facial nerve is used widely in tests to judge the severity and prognosis of facial paralysis. Several test paradigms are used including nerve excitability threshold (NET), the maximum stimulation test (MST), and electroneuronography (EnoG). Consistent technique and careful control of variables are essential to achieve accurate test results. The sources of variability examined in this study were age, gender, body weight, and the use of electrode paste; the NET was used as the test method. The facial NET in 120 adults without a history of facial paralysis increased linearly with age ($P = .0004$) and with body weight ($P < .0001$) and was higher in men than in women adjusted for age and weight ($P = .0001$). The mean NET $\pm$ SD was $0.7 \pm 0.27$ mA in the upper division using the eyelid twitch as an end point, and $1.2 \pm 0.40$ mA in the lower division. There was no statistically significant difference in the results between sides. The NET was falsely elevated by the use of electrode paste, presumably due to current shunting away from the nerve.

Based on the technique described herein, an absolute NET of $\geq 1.25$ mA in the upper division or an absolute NET $\geq 2.0$ mA in the lower division of the human facial nerve is statistically abnormal. These norms are not applicable to grossly obese patients or patients with facial edema or inflammation. Statistical norms allow the NET results to be reported on a continuous scale rather than the dichotomous scale used in the past. The predictive power of the NET will be greatly enhanced by basing test interpretation on both statistical and clinical significance.

INTRODUCTION

Electrical stimulation of the facial nerve is used clinically to determine the prognosis of patients with facial paralysis. Three types of tests are in current use: nerve excitability threshold (NET), maximum stimulation test (MST), and electroneuronography (EnoG). Each involves percutaneous stimulation of the nerve using a direct current (DC) pulse. Other stimulation tests, such as nerve conduction time, intensity-duration curves, and chronaxie/rheobase determination, are no longer used in most centers. The applied current alters the perineural electrical field to initiate depolarization, if the stimulus exceeds the neural threshold, and subsequent propagation of an impulse to the motor end plate. Any resultant twitch may be detected visually or electrically. In threshold testing the least amount of current required for a just visible twitch is the outcome measure. In the other tests, a suprathreshold stimulus is given and the qualitative strength of contraction is noted by inspection in the MST (or the speed of contraction by an accelerometer$^1$) or the amplitude of the neuromuscular compound action potential is noted in the EnoG. The tests compare the results from each side and use the difference as a criterion of normalcy. Although this strategy avoids the need for absolute values and compensates somewhat for intersubject variability, it does not address intrasubject variability. The principal use of electrical tests in patients with facial paralysis is to determine neural integrity and status; that is, whether the nerve to the paralyzed face is intact and, if so, whether it is neuapraxic or degenerating. The results of these tests are combined with the history and physical examination to establish a diagnosis (principally, viral neuropathy vs. neoplasm) and a prognosis (neuapraxic lesions have normal test results and recover fully and promptly, whereas nerves that degenerate take longer to recover and do so incompletely, although satisfactorily in most cases).

The history of electrical testing dates to 1855 when Duchenne$^2$ first described his clinical experience with electrical testing of facial nerve function. Erb,$^3$ using faradic/galvanic current, noted persistent stimulability 1 to 2 weeks after nerve injury. However, Landau$^4$ tested a patient in 1953 after transection of a facial nerve branch and found loss of stimulability after the fourth day. It was not until the early 1960s, however, that electrical tests were applied widely for the evaluation of patients with Bell's palsy. Laumann and Jongkees from Amsterdam and Richardson and colleagues from London were among the earliest investigators. Both groups applied a DC
pulse over the region of the stylomastoid foramen. The Netherlands group used a 0.3-msec pulse width and the English group used a 1-msec pulse width. The range of NET was from 2.4 to 16.2 mA with a mean value of 6.5 mA. Both groups reported close agreement between the right and left sides. Laumans and Jongkees noted a mean difference of 0.4 mA with a standard deviation of 0.2 mA in 20 normal subjects aged 20 to 30 years.

Early investigators sought a critical difference between the normal and paralyzed sides that would indicate a sufficient degree of neural degeneration, taking into account the great variance in threshold among individuals, to predict those cases with poor enough outcomes to warrant surgical therapy. For cases of Bell's palsy, a clinically significant difference was considered to be 3.5 mA in Amsterdam and 2.0 mA in London. These differences were proposed after correlating NET with subsequent outcome. However, the choice of a dichotomous end point (< 3.5 mA = no or clinically insignificant degeneration; ≥ 3.5 mA = clinically significant degeneration) ignores elevated values (e.g., 3.3 mA) that may, in fact, indicate levels of degeneration that would result in a poor outcome. Moreover, neither criterion has received critical study outside the clinics in which they were developed.

Hilger presented his facial nerve stimulator at the 1963 meeting of the then American Academy of Ophthalmology and Otalaryngology. It featured a 0.6-msec pulse, a repetition rate of 6 per second, and a constant current circuit that was said to compensate for variance in skin resistance. The modal threshold with this device was reported to be around 3 mA, but the data upon which this observation was based were not presented. Therasonic electrode paste is distributed with the clinical version of the stimulator (Model N) and the users are advised to use two applications to increase skin contact with the electrode.

Clinicians have generally accepted the 3.5-mA difference between sides in patients with facial paralysis as clinically significant. However, this criterion has not been validated for the Hilger stimulator, a substantially different device from the one on which the criterion was developed. Therefore, transferring this criterion to tests done with the Hilger stimulator without additional validation may not be appropriate. Moreover, the purposes for which electrodiagnosis is used have changed and the clinical significance of abnormal findings has also changed.

Over the years, there has been general dissatisfaction with NET testing using the dichotomous outcome criterion as being too insensitive to the presence of nerve degeneration. Interestingly, instead of questioning the criterion and revising the test paradigm, there has been a trend toward the use of supramaximal testing, which is held to be of greater validity because all fibers in the nerve are stimulated. MST has had advocates but ENoG has become the de facto gold standard for evaluation because of greater objectivity. However, the same problems with percutaneous stimulation apply to ENoG as to NET and MST. In addition, ENoG is also affected by technical artifacts and by neural dysynchronization. Therefore, a review of electrical testing technique as well as further study of electrical testing theory seemed in order.

This paper provides the results of NET testing with the Hilger stimulator in normal subjects and discusses sources of variance in percutaneous motor nerve stimulation based on testing of humans and laboratory animals. An argument is made for using the NET test because it is simpler, less expensive, has the same sensitivity and specificity as suprathreshold tests, and, based on statistical norms, should permit more precise assessment than the dichotomous interpretation used in the past.

**MATERIALS AND METHODS**

One hundred twenty patients in the clinics of the University of Texas Health Science Center at San Antonio voluntarily participated in this study. The subjects gave informed consent as per the protocol approved by the institutional review board. Equal numbers of men and women were selected across age-group categories from the second through seventh decades. None had a history of facial palsy and all had symmetric facial motion and the absence of otologic or salivary gland disorders. The subjects' height and weight were also recorded, and the body mass index was calculated as weight in kilograms/m² divided by the height in square centimeters. The body mass index is a convenient technique to classify people's height and weight relation. A body mass index (BMI) of <23 is considered normal, 25 is obese, and in between is overweight. An additional 25 grossly obese subjects recruited from the obesity clinic were studied in the same manner, as described, but analyzed separately.

Nerve excitability thresholds were established for the upper and lower divisions of the facial nerve on both sides. The skin was cleaned with an alcohol sponge and allowed to dry. No electrode paste was used on the stimulating electrode tip, although a small dab of paste was used on the ground electrode. The Hilger bipolar clinical electrode, which consists of an active and ground electrode in a single assembly connected by a hinge that permits varying the distance between the two electrode tips, was used. The electrodes were positioned at right angles to the nerve branch. A supramaximal current from the Hilger stimulator model N was used to locate the nerve at two sites: 1 where the upper division crosses the zygoma, just lateral to the lateral orbital rim; and 2 at the mandibular notch (Fig. 1). Contraction of the orbicularis oculi was used as the end point for the upper branch stimulation and the orbicularis oris for the lower branch. Several ascending and descending stimulations were used to bracket the threshold. Once the nerve was located, the stimulation intensity was reduced to the point where twitching of the muscle disappeared and, once below threshold, it was increased to the point where a just noticeable twitch occurred. The difference between the ascending and descending thresholds was taken as the true
threshold. To differentiate the absolute NET from the other dichotomous technique (NETa) the symbol NETb is used.

Six Sprague-Dawley rats weighing 200 to 250 g were used to determine the effect of electrode paste on NET. Thresholds for plantar flexion of the toes in response to stimulation of the posterior tibial nerve at the lateral malleolus were obtained with the same Hilger stimulator without, with a small amount, and with a large amount of Therasol electrode paste.

The English literature was reviewed for the results of NET, MST, and EMG testing for patients with Bell's palsy. All articles were used in which the patients' outcome in relation to the test results were reported. Results are presented as means ± SD. All tests are two-tailed. The level of significance for a type I error is P < .05. NET, is calculated as the algebraic mean of the absolute excitability thresholds of the upper and lower branches from each side. The following statistical tests were used: 1. Students t test for the comparison of NETb by side, gender, branch, and between normal and obese subjects; 2. one-way analysis of variance for the effect of three levels of electrode paste on NET; and 3. multivariate analysis of covariance of NETb using age, gender, and body mass as covariates. Normal is defined as the range of values encompassed by the mean ± 2 SD. Sensitivity is the percentage of the total number of positive tests that are truly positive; specificity is the percentage of the total number of negative tests that are truly negative.

RESULTS

The normal subjects consisted of 60 men and 60 women aged 21 through 79 with equal numbers in each decade group for each gender. The mean age was 49 years. The body mass index of the subjects is displayed in Table I. Women had a slightly greater body mass index: 24.6 vs. 24.1; this difference was not significant. Of the obese subjects, 5 were men and 20 were women. Their average weight was 287 lb with a range of 183 to 479 lb.

Table II displays the mean values and ranges of absolute nerve excitability thresholds of the upper and lower branches for the 120 normal subjects. The NETa for the upper branch was less than the lower branch in all but 5 subjects, 4 of whom were men. The

<table>
<thead>
<tr>
<th>Table I: Distribution of Subjects by Gender and Body Mass Index.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>≤ 25</td>
</tr>
<tr>
<td>25–30</td>
</tr>
<tr>
<td>&gt; 30</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

*Body mass index of ≤ 25 is considered normal, above 30 is considered obese.

<table>
<thead>
<tr>
<th>Table II: Mean Nerve Excitability Thresholds (mA) for 120 Normal Adults.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stimulation Site</td>
</tr>
<tr>
<td>------------------</td>
</tr>
<tr>
<td>Upper branch</td>
</tr>
<tr>
<td>Right</td>
</tr>
<tr>
<td>Left</td>
</tr>
<tr>
<td>Lower branch</td>
</tr>
<tr>
<td>Right</td>
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<td>Left</td>
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</tbody>
</table>

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difference is highly significant statistically ($t = 10.2$, $P = .0001$). The difference between the right and left sides averaged 0.01 mA and was not significant (upper branch $t = 0.04$, $P = .992$; lower branch $t = 0.007$, $P = .902$).

The mean nerve excitability thresholds for the 60 men and 60 women are listed in Table III. For both the upper and the lower branches, the thresholds are lower for the women, even though their body mass index is slightly higher than that of the men. This difference was statistically significant ($t = 2.86$, $P = .005$). There was no interside variation by gender.

**TABLE III.**

Mean Nerve Excitability Thresholds (mA) for 60 Men and 60 Women.

<table>
<thead>
<tr>
<th>Stimulation Site</th>
<th>Men (Mean ± SD)</th>
<th>Women (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper branch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>0.78 ± 0.24</td>
<td>0.62 ± 0.29</td>
</tr>
<tr>
<td>Left</td>
<td>0.79 ± 0.23</td>
<td>0.63 ± 0.28</td>
</tr>
<tr>
<td>Lower branch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>1.24 ± 0.37</td>
<td>1.09 ± 0.43</td>
</tr>
<tr>
<td>Left</td>
<td>1.23 ± 0.37</td>
<td>1.09 ± 0.43</td>
</tr>
</tbody>
</table>

Table IV displays NETs for 25 grossly obese subjects.

The figure displays the change in NET, with age (by decade) for each gender. The increase in electrical threshold with age is gradual in the men and rapid in the women.

Multivariate analysis of covariance of NET, by age, gender, and body mass revealed significant effects of age ($F = 13.41$, $P = .004$), gender ($F = 17.46$, $P = .0001$), and body mass ($F = 46.89$, $P < .0001$). This analysis indicates that the nerve excitability threshold increased with increasing age, male gender, and increasing body mass, with each variable ad-

### Table IV

Nerve Excitability of the Obese Subjects ($N = 25$).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean*</th>
<th>SD</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper A</td>
<td>1.03</td>
<td>0.44</td>
<td>0.55</td>
<td>2.14</td>
</tr>
<tr>
<td>L</td>
<td>1.02</td>
<td>0.41</td>
<td>0.58</td>
<td>1.90</td>
</tr>
<tr>
<td>Lower A</td>
<td>2.79</td>
<td>1.06</td>
<td>0.92</td>
<td>5.50</td>
</tr>
<tr>
<td>L</td>
<td>2.68</td>
<td>0.99</td>
<td>0.90</td>
<td>4.95</td>
</tr>
</tbody>
</table>

*The difference in means in nerve excitability threshold (NET) between the obese ($N = 25$) and not-obese subjects ($N = 120$) was highly significant ($t = 11.53$, $P = .0001$).
TABLE V.
Compilation of Test Results for Nerve Excitability Test (NET), Maximum Stimulation Test (MST), and Electroneuropography (ENoG).

<table>
<thead>
<tr>
<th>Test</th>
<th>TN</th>
<th>FN</th>
<th>FP</th>
<th>TP</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>NET</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laumanns and Jongkees</td>
<td>20</td>
<td>1</td>
<td>6</td>
<td>12</td>
<td>39</td>
</tr>
<tr>
<td>Campbell</td>
<td>55</td>
<td>6</td>
<td>17</td>
<td>39</td>
<td>117</td>
</tr>
<tr>
<td>Leclaire, et al</td>
<td>129</td>
<td>1</td>
<td>12</td>
<td>32</td>
<td>174</td>
</tr>
<tr>
<td>Saade and Karam</td>
<td>22</td>
<td>4</td>
<td>2</td>
<td>11</td>
<td>39</td>
</tr>
<tr>
<td>May, et al.</td>
<td>34</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>42</td>
</tr>
<tr>
<td>Totals</td>
<td>260</td>
<td>17</td>
<td>37</td>
<td>97</td>
<td>411</td>
</tr>
<tr>
<td>MST</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May, et al.</td>
<td>29</td>
<td>4</td>
<td>3</td>
<td>15</td>
<td>51</td>
</tr>
<tr>
<td>ENoG</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May, et al.</td>
<td>12</td>
<td>2</td>
<td>9</td>
<td>16</td>
<td>39</td>
</tr>
</tbody>
</table>

*Data for TN/FN relate to 70% degeneration level or less, and for FP/TP to a 75% or greater level of degeneration.
TN = true negative, FN = false negative, FP = false positive, TP = true positive.

justed for the effects of the other covariates. There was also a significant interaction between age and gender, but only at the upper extreme of age (Fig. 2).

The NET (mean ± SD) of the posterior tibial nerve in six animals was 0.21 ± 0.03 mA without electrode paste, 0.30 ± 0.08 mA with a normal amount of electrode paste, and 0.35 ± 0.08 mA with excess electrode paste. These values were significantly different (F = 23.08, P = .002) using a one-way analysis of variance.

Table V was abstracted from the literature and indicates the sensitivity and specificity of NET, MST, and ENoG in predicting degeneration in Bell's palsy cases. Based on this table, the sensitivity of NET is 85%, MST is 79%, and ENoG is 89%, and the specificity of NET is 87%, MST is 93%, and ENoG is 57%. The data for ENoG were extrapolated from a single study in which the criterion levels differed from standard norms. Therefore, these sensitivity and specificity data should be interpreted with caution.

**DISCUSSION**

**Technique**

This study demonstrates several intrinsic and extrinsic factors that affect the NET. That the NET increases with body mass emphasizes the concept that percutaneous nerve stimulation is influenced by the bulk of tissue separating the skin surface from the nerve. This point is further illustrated by the difference in mean NET in subjects of average weight compared to the obese subjects. It is likely that the gender difference in NET is due to skin thickness, but a true gender difference in neural threshold cannot be excluded. Although age influences the NET, the magnitude of the effect is very small: the NET increased 0.08 mA every decade from the 2nd through the 7th decades. Thus, over the age range studied, age alone does not elevate the NET outside the group's normal range. In contradistinction, the NET of the lower branch of the obese subjects (R = 2.79 mA, L = 2.68 mA) were clearly abnormal, and those over the upper division were elevated but still in the normal range. In addition to the statistically significant effects of age, gender, and obesity noted in this study, Lewis, et al. also found that hypertension and diabetes significantly elevated the NET.

The chief extrinsic variable delineated in this study was the use of electrode paste. The greater the amount of electrode paste applied, the greater the NET. The larger the area of anode contact with the skin, the larger is the current-flow field and the lower the current-flow density at any point in the field. Therefore, to achieve a critical electrical density at the axon membrane, more current is required as the field size increases. In effect, the size of the paste dab enlarges the size of the stimulating electrode. If there is variation in the size of the paste dab, there will be variation in the NET. A related factor that also affects the functional size of the anode is the pressure of the electrode tip on the skin. Increasing the pressure causes a greater contact area of the electrode tip as the skin is indented and wraps around the electrode. It is conceivable that variations in the amount of electrode paste and skin contact pressure may have contributed to the variability in NET observed in other studies.

Lewis, et al. reported their experience with the Hilger stimulator using the no-electrode paste technique. They obtained slightly higher NET values than in this study: 0.8 vs. 0.7 mA over the eye branch and 1.35 vs. 1.20 mA over the ramus mandibularis. However, the standard deviations in their study were nearly twice as large as those in the present report. This greater variability may reflect the presence of subclinical contralateral disease, given that all their patients had a unilateral facial palsy, or it may be due to differences in technique. This attests to the need of each clinic and even each examiner to establish norms using their own patients. Having absolute values for thresholds should increase the use of NET testing, especially when serial examinations are done. This avoids the problem of bilateral neuropathy in which a symmetric elevation of thresholds could lull one into a false sense of security.

Nerve excitability thresholds obtained by stimulation of the main trunk of the nerve through the parotid gland vary greatly, probably due to differences in tissue volume. The higher stimulus levels required there also reduce patient compliance. Stimulation of the lower branch has less variability and the eyelid branch has the least variability. Therefore, studies of NET should use the peripheral branches as stimulation points because the variability is less and patient compliance is higher.

The importance of technique to percutaneous
stimulation testing should be stressed. If an abnormal response is obtained, it must be verified that the abnormal response was not due to technical flaws; otherwise the abnormal result might be falsely attributed to neural pathology. As such, it is clear that a normal response has greater predictive value than an abnormal one. Thus, one would have more confidence in advising patients about the status of the nerve when the NET is within a normal range than when the response is abnormal.

Analysis and Interpretation

Interpretation of any electrodiagnostic test must be made in light of the etiology of the facial paralysis, the degree of paralysis (partial or complete), the rate of progression of the paralysis (velocity), the timing of the test, other neurologic findings, and associated conditions.

Nerve excitability threshold results have traditionally been analyzed dichotomously, e.g., the difference between sides must exceed a certain value to be interpreted as abnormal. This method was established with different equipment and at a time where the principal treatment decision was whether to decompress the nerve or not. Although two different criteria were proposed, a 2-mA difference and a 3.5-mA difference, the rationale for the acceptance of either value has never received critical appraisal.

Today, electrodiagnostic tests are used in patients with Bell's palsy primarily to provide an accurate prognosis. Selection of cases for surgical therapy has become more complex based on electrical tests, site of lesion, age of patient, progression of paralysis, and patient's desires. Given that the principal determinant of outcome for cases of Bell's palsy with complete paralysis is the presence of degeneration, the greater the accuracy of the test in detecting evidence of degeneration, the greater the value of the test. (Paretic cases almost always recover fully and do not, as a rule, require electrodiagnostic testing). Because degeneration is evidenced clinically by incomplete recovery or the presence of synkinesis, the value of the test can be assessed by comparing the test results with the occurrence of residual facial weakness and synkinesis.

The finding of similar values for sensitivity and specificity of the three tests suggests that each is measuring the same phenomenon. Although the reports vary in test methodology, outcome criteria, definitions of satisfactory and unsatisfactory results, and in the timing of testing, because the tests were done for the same purpose, i.e., to predict an unsatisfactory outcome, and that a single disease entity was studied, it may well be the case that this comparison is relevant and will have to suffice until better, prospectively designed comparisons are available. This analysis shows that each technique has errors and that the general range of the errors is similar. Such a conclusion is in keeping with well-known epidemiologic principles, which indicate that all types of tests used in clinical medicine suffer from false-negative and false-positive results, and that the trade-off between sensitivity and specificity must be taken into account in the design and interpretation of clinical tests.

Groves and Gibson deny that there is a critical NET difference on which to justify surgical intervention. Of 27 cases with rising NET, they found 10 who could not be stimulated yet had satisfactory, though delayed, recovery. The false-positive test rate was 63% at a criterion difference of 2 mA, 66% at 3 mA, 55% at 4 mA, and 0% at 5 mA. In most of the remaining 17 cases the NET improved after exceeding the differences just noted. Interestingly, Laumans noted an increasing difference in NET just prior to the recovery of motion in patients with Bell's palsy. Esselen noted that full recovery occurs in most cases with <90% reduction in amplitude on ENO and that, even up to 98% reduction in amplitude, recovery is good for most patients.

Theoretical Considerations

Wallerian degeneration probably occurs incrementally and non-uniformly in the nerve involved in Bell's palsy, and mixed lesions (i.e., neurapraxia and degeneration) are common. Except for the extreme cases where stimulability is completely normal (neurapraxia) or completely absent (total degeneration), there is a theoretical spectrum of abnormal NET values that would parallel the degree of nerve loss. Therefore, it is a logical hypothesis that partial degeneration of a nerve would elevate the threshold in relation to the degree of degeneration. Although it is held that the NET is most sensitive to the status of large, well-myelinated facial nerve fibers, there are no data to indicate that selective preservation of "threshold" fibers occurs in Bell's palsy. Given the rather narrow size range (7 to 9 μm) of myelinated motor fibers within the human facial nerve and the rather small number of unmyelinated fibers, which are probably sensory, it is difficult to conceive how a significant degeneration of motor fibers could occur without elevating the NET. Logic would suggest that even mild elevation of the NET would have clinical importance, and that NET values outside the normal range should be interpreted on a continuous scale rather than as two-choice (i.e., abnormal vs. normal), dichotomous scale NET. With complete absence of stimulability on NET, MST or ENO, the prognosis for full recovery is virtually nil, although many cases recover to a satisfactory degree.

This hypothesis is in contradistinction to that of Laumans and Jongkees, who suggest that a critical threshold of degeneration must be reached before the NET elevation is clinically significant, that is, to predict a poor enough outcome to warrant surgical decompression. In today's climate of cost-control and greater patient participation in medical decision mak-
ing, a test that will predict extent of recovery and time
to recovery has great value in its own right. The
problem of identification of cases that would benefit
from surgical decompression has not been satisfac-
torily solved to date with any of the electrodiagnostic
methodologies. Using the NET in the manner
described herein will prove to be highly satisfactory for
prediction of recovery from Bell's palsy.

The validity of NETₐ, as proposed herein, in
predicting outcome of patients with Bell's palsy
should be tested in a prospective study that compares
the results of carefully done NETₐ, MST, and ENoG
done in the same patients and which also examines
the timing of electrical test methodology. The results
of such a study would refine the role of electrodiagnostic
Testing in the selection of therapy for patients with
facial paralysis.

**ADDENDUM**

Subsequent to the presentation of this paper, a
report by Coker, et al., compared NETₐ and ENoG in
77 patients with acute facial palsy. They found high
correlations between NETₐ over the branches (as ad-
vocated herein) and ENoG. They concluded that “the
two tests are complementary in establishing the in-
tegrity of the facial nerve in acute facial paralysis.”

**BIBLIOGRAPHY**

try: A Sensitive and Accurate Method for Evaluating Facial
2. Duchenno, G.B.: *De l'Electriscation Localisee* (3rd ed.). Baillier-
iere, Paris, 1872.
New York, 1866, 1883.
4. Landau, W.M.: The Duration of Neuromuscular Function After
5. Laumenos, E.P.J. and Jongkees, L.B.W.: On the Prognosis of
Peripheral Facial Paralysis of Endometrial Origin. Part II.
Nerve-Excitability Measurements in Prognosis of Facial Pal-
8. Yanagihara, N. and Kishimoto, M.: Electrodiagnosis in Facial
Accuracy of the Maximal Stimulation Test Compared With
That of the Nerve Exidability Test in Bell's Palsy. **Laryngosco-
Excitability Testing Versus Neuromyography: Prognostic
11. Gavilán, J., Gavilán, C. and Sarria, M.J.: Facial Electroneu-
13. May, M., Blumenthal, F. and Klein, S.R.: Acute Bell's Palsy:
Prognostic Value of Evoked Electromyography, Maximal
Stimulation, and Other Electrical Tests. **Am J Otol**, 5:1–7,
1983.
16. Salman, B.L.: Bilateral Pathology in Bell's Palsy. **Arch Oto-
17. Groves, J. and Gibson, W.F.R.: Bell's (Idiopathic Facial) Palsy:
The Nerve Excitability Test in Selection of Cases for Early
19. Easen, E.: Electromyography and Electroneurography. In:
**Facial Nerve Surgery**, F. Fisch Ed. **Aesculapius P.O.O.**,
Facial Nerve: Quantitative Features. **Acta Otolaryngol Sup-
22. Leclaire, R., Tremblay, P.T. and Dupuis, M.: Prognostic Value of
Bell's Palsy: The Salivary Flow Test and Other Prognostic
Excitability Test and Electroneurography in Acute Facial