The Maximal Stimulation and Facial Nerve Conduction Latency Tests: Predicting the Outcome of Bell's Palsy

John M. Ruboyianes, MD; Kedar K. Adour, MD; David Q Santos, MD; Peter G. Von Doersten, MD

To test the hypothesis that the facial nerve conduction latency test is a better and earlier indicator of prognosis than other electrodiagnostic tests, 86 patients with Bell's palsy were followed for a minimum of 4 months. To select control subjects for our own research clinic and for comparison with the patient population, latency values in 25 normal volunteers (50 sides) were determined. Serial maximal stimulation tests (MST) and latency tests were conducted to determine disease severity and prognosis in Bell's palsy patients. Outcome was graded using the Facial Paralysis Recovery Profile (FPRP) and Facial Paralysis Recovery Index (FPRI) as well as the House grading system. The capability of the two tests to accurately predict outcome was evaluated. The MST accurately predicted outcome in 94% of patients studied. In the control group, normal latency values were a mean 3.5 msec with a standard deviation of 0.49. In the patient population, latency values were either within normal limits or absent. When done within 4 days of onset of Bell's palsy, neither test was capable of predicting axonal degeneration. Statistical analyses included Fisher's Exact Test, the paired Student's t test, and correlation coefficient calculations.

INTRODUCTION

Bell's palsy, formerly termed idiopathic facial paralysis, can be diagnosed on the basis of an appropriate history and physical examination while other possible causes of facial paralysis (trauma, infection, and tumor) are excluded. Although initial signs and symptoms may indicate severity and outcome, electrodiagnostic testing is needed to document disease progression and thus more accurately assess prognosis. Results of electrodiagnostic tests have been used both in selecting patients for treatment and in predicting outcome. The most commonly used electrical tests include the minimum nerve excitability test; the maximal stimulation test (MST), also termed the maximal nerve excitability test; and electroneurography (ENOG).1-3

These tests all rely on percutaneous stimulation of the facial nerve distal to the stylo mastoid foramen, and such tests do not show abnormality until 72 hours after degeneration has occurred. Gilliatt and Taylor4 found that facial nerve conduction latency is maintained as long as nerve conduction is still functional. A recent report5 suggests that latency is an accurate indicator of prognosis within 24 hours of onset of facial paralysis. If so, patients could be selected for treatment before degeneration occurred. For the otologist, this determination would identify a period in which surgical decompression of the facial nerve would be of maximal benefit to patients. To test this hypothesis, a prospective longitudinal study of 150 consecutive Bell's palsy patients was conducted, and the predictive values of the facial nerve conduction latency test and the MST were compared.

SUBJECTS AND METHODS

As part of a prospective treatment study of Bell's palsy patients at the Cranial Nerve Research Clinic, Kaiser Permanente Medical Center, Oakland, Calif., serial MSTs were conducted, and latency was determined in a series of 150 unselected patients. All were seen within 7 days of onset of palsy. Sixty-four patients were excluded from the study because of inadequate follow-up or patient inability to tolerate the electrodiagnostic tests. The remaining 86 patients were followed for a minimum of 4 months. The study protocol was approved by the Kaiser Permanente Medical Care Program, Northern California Region, Institutional Review Board, and informed consent was obtained.

Latency testing was done at the patient's initial visit, 2 weeks after onset of palsy, and at the last visit. The MST was done at the initial visit as well as 2 weeks and 1 month after onset of palsy. For both tests, the normal (control) side of the face was studied first, and then the affected side was studied.

Facial Nerve Conduction Latency Testing

To test facial nerve conduction latency, the Ampilaid mk 15 electrodiagnostic system (Ampilaid S.P.A., Caleppio di Settala, Milan, Italy) machine was used. The bipolar stimul-
Fig. 1. Electrode placement for facial nerve conduction latency test. Stimulating electrode is placed over stylomastoid foramen, and recording electrode is placed in nasolabial crease. In standard electrode placement, only stimulating electrode may be manipulated to obtain best response. (Adapted and reproduced by permission of the illustrator and publisher from: Adour, K.K.: Facial Nerve Electrical Testing. In: Textbook of Neuro-Otology. C.V. Mosby, St. Louis. In press.)

Lating electrodes were placed over the stylomastoid foramen, in the groove between the mandibular ramus and the mastoid process, with the stimulating electrode more cranial. The bipolar recording electrodes (Nicolet), encased in a plastic block, were placed in the nasolabial fold with the recording electrode placed adjacent to the nasal ala (Fig. 1). The stimulating electrodes were 8 mm in diameter, the recording electrodes 7 mm; the centers of the stimulating electrode pairs were 23 mm apart, and the centers of the recording electrodes were 25 mm apart.

The standard technique was used to determine latency: to maximize the muscle compound action potential (CAP) and to prevent masseter muscle stimulation, only the stimulating electrodes were repositioned. To induce supramaximal stimulation, the intensity was gradually increased until no further CAP increase was noted. Maximum intensity used was 50 mA. Intensity was sometimes limited by masseter contraction or patient tolerance. Measurements of the affected side were compared with those of the normal side to control for minor variations in technique used by different examiners. Both CAP and latency were determined (Fig. 2).

Fig. 2. Latency readout. Derivation of compound action potential (CAP) and latency.

To determine normal values and standard deviations for our clinic, the latency test was administered to 25 volunteers who had no neuromuscular or otologic symptoms.

Maximal Stimulation Testing

To conduct the MST, we used the Hilger Model 2-R and H-3 Facial Nerve Stimulators" (WR Medical Electronics Co., Stillwater, Mich.). The ground electrode was moistened with alcohol and was held by the patient to the back of the hand. The temporal, orbital, and marginal branches of the facial nerve were stimulated, and the results were recorded separately (Fig. 3). Starting at a low amperage, the stimulator setting was gradually increased until muscle activity was seen on the normal side. The area was then explored to find the point at which muscle response was greatest. Next, the stimulus was increased by 1 to 2 mA, thereby inducing supramaximal stimulation. Using the same setting, the corresponding nerve branch on the affected side was stimulated.

A numeric method of recording MST results was developed (Table 1) which enabled entry of data into calculations for statistical analysis on the basis of the mean score for stimulation of the three facial regions. The amount of muscle movement on the affected side was compared with that on the normal side, and relative response was graded on a

| TABLE 1. Maximal Stimulation Test (MST) Grading System Derived From Averaging Scores From Three Facial Sites Stimulated.* |
|---------------------------------|---------|
| Difference                      | Grade   |
| No response                     | 0       |
| Marked                          | 1       |
| Moderate                        | 2       |
| Minimal                         | 3       |
| Equal                           | 4       |

*Resultant visible muscle contraction on affected side recorded relative to contraction on normal side.

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TABLE II. 
Facial Paralysis Recovery Profile (FPRP) and Facial Paralysis Recovery Index (FPRI) .

<table>
<thead>
<tr>
<th>Site</th>
<th>0%–25%</th>
<th>26%–50%</th>
<th>51%–75%</th>
<th>76%–100%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forehead</td>
<td>+1</td>
<td>+1</td>
<td>+2</td>
<td>+2</td>
</tr>
<tr>
<td>Eye</td>
<td>+1</td>
<td>+2</td>
<td>+3</td>
<td>+4</td>
</tr>
<tr>
<td>Mouth</td>
<td>+1</td>
<td>+2</td>
<td>+3</td>
<td>+4</td>
</tr>
</tbody>
</table>

FPRP:† Points awarded for each unit of recovery.

Complication Points†

Contracture –1
Synkinesis –1

*Points awarded for facial function on affected side, relative to normal side, in increments of 25%.
†Points subtracted from FPRP to derive FPRI. (Adapted and reproduced by permission of the publisher from: Adour, K.K. and Swanson, P.J., Jr.: Facial Paralysis in 403 Consecutive Patients: Emphasis on Treatment Response in Patients With Bell's Palsy. Trans Am Acad Ophthalmol Otolaryngol, 75:1291, 1971.)

scale from 0 to 4. Zero was defined as no muscle response to stimulation of the peripheral nerve branch, and 4 was defined as no difference in response between the affected and normal sides. Intermediate numeric values were assigned to minimal, moderate, and severe reduction in muscle response of the affected side compared with that of the normal side.

**Reporting Methodology**

Final outcome was graded using the Facial Paralysis Recovery Profile (FPRP) and Facial Paralysis Recovery Index (FPRI). The FPRP (Table II) is obtained by measuring the precise amount of forehead and mouth movement and estimating the degree of eye closure. The affected side is assigned points for each 25% of function relative to the normal side. The forehead is less heavily weighted than the eye and mouth. Percentage of facial nerve function can be estimated by totaling the score from each of the three measurements and multiplying by 10. The FPRI (Table II) is an adjustment of the FPRP based on residua of nerve degeneration and regeneration. Each complication is subtracted from the FPRP to obtain the final FPRP. For instance, for a patient who had full recovery of volitional facial function (FPRP = 10) but who also had contracture and synkinesis, the FPRI score would be 8 (10 – 2).

Final outcome was reported using the House grading system as well (Table III). Conversion of FPRP and FPRI scores to the House system was done on the basis of voluntary facial function as well as presence or absence of synkinesis and contracture.

Latencies on the affected and normal sides were compared using the paired Student's t test. The strength of the relation between outcome (reported using the FPRP/FPRI and House systems) and the tests used (latency and MST) was evaluated using correlation coefficients. Statistical significance of the results was evaluated using Fisher's Exact Test.

**RESULTS**

Patients in the study and control groups were demographically similar. Of the 86 study group patients, 36 were men and 50 were women; mean age was 44 years (range, 18 to 75 years). Of the 25 control group patients, 10 were men and 15 were women; mean age was 40 years (range, 24 to 75 years).

In 66 patients (76.4%), recovery was complete. In 13 patients (15.1%), recovery of facial function was nearly complete, but mild synkinesis and contracture developed. The other 9 patients (10.5%) had mild-to-moderate residual facial weakness as well as moderate-to-severe contracture and synkinesis (Table IV).

**Facial Nerve Conduction Latency Testing**

In the control group, latency ranged from 2.9 to 4.9 msec (mean latency, 3.8 msec; SD, 0.49 msec). Side-to-side difference in the same group of patients ranged from .00 to .96 msec (mean difference, 0.27 msec; SD, 0.24 msec).

In all study group patients, latency was normal at initial testing, done during the first 4 days of palsy (Table V). Mean latency on the affected side was 3.7 msec (range, 2.6 to 4.9 msec) compared with 3.6 msec on the normal side. Mean difference between the two sides was 0.46 msec (range, 0 to 2.0 msec), and latency was shorter on the affected side in 42% of patients. During subsequent testing at 2 and 4 weeks, latency was either normal (range, 1.4 to 6.7 msec) on the affected side when compared with the normal side or unmeasurable (i.e., no CAP). No statistically significant difference in latency (when present) was found between the affected and normal sides of the face in study group patients at any time during the study.

**TABLE IV.** 
Patient Recovery Assessed Using Adour-Swanson and House Grading Systems.

<table>
<thead>
<tr>
<th>N (%)</th>
<th>FPRP/FPRI</th>
<th>House Gradea</th>
</tr>
</thead>
<tbody>
<tr>
<td>66 (76.7)</td>
<td>10/10</td>
<td>I/VI</td>
</tr>
<tr>
<td>13 (15.1)</td>
<td>10–8/8–6</td>
<td>II/VI</td>
</tr>
<tr>
<td>4 (4.7)</td>
<td>7/5</td>
<td>III/VI</td>
</tr>
<tr>
<td>3 (3.5)</td>
<td>4/2</td>
<td>III/VI</td>
</tr>
</tbody>
</table>

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Normal latency was associated with complete recovery (FPRI = 10, House grade I/V) in 94.8% of patients. Absent latency was predictive of incomplete recovery (FPRI ≤8, House grade II–III/VI) in only 71.4%. In the other 28.6% in whom latency was absent, recovery was complete (Table VI). Latency testing was 88.6% accurate in predicting outcome of Bell’s palsy when latency was interpreted as either normal or absent.

Maximal Stimulation Testing

An MST score of 2.6 or greater at the second and third visits was associated with complete recovery in 94.2% of patients (FPRI/FPRI = 10/10, House grade I/V). An MST score of less than 2.6 was associated with incomplete recovery and development of synkinesis and contracture in 94.1% of patients (FPRI ≤8, House grade II–III/VI) (Table VI).

Correlation coefficients relating both the MST score and latency (absent or normal) to the FPRP and FPRI were calculated and compared (Table VI). A positive correlation with both the FPRP and FPRI was found for each test, although the correlation was stronger for the MST score (Table VI).

DISCUSSION

Continued interest in electrodiagnostic testing for Bell’s palsy is aimed primarily at determining which patients would benefit from surgical decompression of the facial nerve. The MST and ENOG have both been accepted as good indicators of prognosis. The problem with these and other electrodiagnostic tests is that they can indicate axonal degeneration only 2 to 3 days after it occurs, potentially reducing any benefit of surgical intervention. An ideal test would identify which patients were at risk for axonal degeneration so that it might be prevented.

Facial Nerve Conduction Latency Testing

In peripheral neuropathy, nerve conduction velocity (NCV) is used in assessing a nerve’s integrity. The difference in conduction times, represented by distance/time, between two stimulated points (proximal and distal latency) can be measured only in relatively long peripheral nerves. NCV is decreased in neuropathy in which demyelination has occurred but is maintained in axonal neuropathy.9

Because the extratemporal facial nerve is relatively short, NCV cannot be determined. Furthermore, because of the lesion’s intratemporal location in Bell’s palsy, neither proximal nor distal latency is determined by stimulating the nerve at the stylomastoid foramen. Latency testing measures the time between initiation of electrical stimulation and first-recorded beginning of facial muscle action potential. When ENOG is used, this process creates dependence on measurable CAP to determine latency.

Compound action potential is considered an objective measurement of the number of axons remaining that respond to stimulation, including “blocked,” or neuropathic, axons. CAP is thus an estimate of the number of normal and neuropathic axons; when this estimate for the affected side is compared with that for the normal side, the result should be reported as percentage of electrically excitable fibers, not as percentage of degeneration.

In trauma cases in which axonal disruption has occurred, degeneration of both the distal axonal stump and myelin sheath begins immediately (within hours).10 However, electrical testing shows that the distal stump responds to stimulation for up to 72 hours. The same phenomenon accounts for delay in detecting axonal degeneration when current electrodiagnostic tests are used in examining Bell’s palsy patients.

Histologic examination of the intratemporal facial nerve in a Bell’s palsy patient11 showed an inflammatory infiltrate within the nerve bundles and degeneration of axons as well as associated myelin sheaths; findings for the nerve from the internal auditory meatus to the stylomastoid foramen were described. To our knowledge, no studies have described histologic findings for the extratemporal facial nerve after onset of Bell’s palsy. This deficiency is important because all current electrodiagnostic tests necessitate examining the extratemporal portion of the facial nerve.
If peripheral demyelination occurs in Bell's palsy, prolonged latency in the affected axons might be expected. However, testing individual axons is not possible using current methods. Liston and Kleid\(^{11}\) showed that not all axons participate in the inflammatory infiltrate and myelin degeneration seen in Bell's palsy. When ENOG is used, axonal response must be derived from the remaining nondemyelinated axons, and the delayed contribution to CAP of the affected axons is thus obscured. This process results in reduced CAP and normal latency.

If demyelination does not occur in the presence of peripherally degenerating axons (which seems unlikely on the basis of current knowledge), the result is the same. As long as electrically excitable axons are present (\textit{i.e.}, CAP is produced), latency should be normal.

Contrary to reports by Gilliatt and Taylor,\(^{4}\) those by Joachims, \textit{et al.}\(^{12}\) and Skevas, \textit{et al.}\(^{5}\) both showed that latency accurately predicted outcome in Bell's palsy within 72 hours of onset (within 24 hours, according to Skevas, \textit{et al.}).\(^{5}\)

Mean latency in control patients (3.8 msec) as well as on the affected side (when CAP was present) and the normal side in study patients in our study was similar to the mean normal values of 3.6 msec (range, 2.4 to 6.0 msec) reported by Joachims, \textit{et al.}\(^{12}\) and 3.2 msec (range, 2.5 to 4.0 msec) reported by Skevas, \textit{et al.}\(^{5}\). Their results thus validate our technique. In addition, knowing the normal latency ranges justifies our using the normal side of the face as a control, and also excludes the possibility that the latency values determined are affected by any contralateral subclinical palsy.\(^{13}\)

Latency was not prolonged in any patient examined by ENOG within 4 days of onset of palsy. Latency among patients seen 1, 2, and 3 days after onset of Bell's palsy was compared, but no statistically significant difference was found. Furthermore, in patients in whom denervation developed and CAP amplitude was reduced, latency was normal; in patients with denervation in whom CAP was completely lost, latency was unmeasurable. These findings remained the same throughout the recovery period. The MST also showed denervation in these patients, and the final FPRI score for each was \(\leq 8\), indicating development of synkinesis and contracture.

These results show that conduction time in electrically excitable axons is maintained. Stimulating the same axons, which produces measurable CAP results in normal latency. Because electrically responsive axons obscure the delayed contribution of affected axons, latency (unless absent; \textit{i.e.}, no CAP) cannot indicate axonal degeneration.

Our study shows that the latency test is 88.6% accurate when latency is interpreted dichotomously as either normal or absent. However, the false-positive rate (the percentage of patients in whom latency is absent but who recover completely) is 28.6%, which thus precludes relying on this test to determine the need for surgical decompression of the facial nerve in Bell's palsy. In addition, the latency test does not show abnormality during the first 4 days after onset of Bell's palsy. Like the other available electrodiagnostic tests, the latency test can detect axonal degeneration only after it occurs.

**Maximal Stimulation Testing**

A method was developed for converting MST-derived information into a numeric format, which provides for computations for statistical analyses. These analyses showed that the MST is 94% accurate as a predictor of prognosis in Bell's palsy. A mean MST score of less than 2.6 correlates with incomplete recovery. Furthermore, the same parameter correlates with presence of axonal degeneration as shown by development of synkinesis and contracture, as well as reduced FPRI.

**Facial Paralysis Recovery Profile (FPRP) and Facial Paralysis Recovery Index (FPRI)**

The FPRP and FPRI were used in reporting outcome because this grading system has been used successfully at the Cranial Nerve Research Clinic since 1969. However, several problems with the proposed House grading system\(^{8}\) (Adour, K.K., \textit{et al.}, unpublished data, 1983) have been identified.

First, the major difference among House grades II, III, and IV is degree of synkinesis and contracture, which is clearly demonstrated by the FPRP and FPRI. Other determinants are often scattered among the three groups. Grading degree of synkinesis and contracture is not necessary using the FPRP and FPRI because these complications are associated with a lower FPRP and thus a reduced FPRI. For instance, a face with mild functional asymmetry associated with a lower degree of synkinesis and contracture would have a higher FPRI score of 3 and an FPRI score of 5. A face with moderate residual dysfunction would be associated with a greater degree of synkinesis and contracture and would have an FPRI score of 6 and an FPRI score of 4. The FPRP and FPRI thus inherently indicate degree of synkinesis and contracture.

Second, grade III patients include two distinct groups: one in which patients have relatively normal facial movement as well as mild synkinesis and contracture, and one in which patients have decreased facial motion as well as moderate synkinesis and contracture. House grade III should thus be subdivided into two separate groups.

Finally, the House grading system cannot indicate rate of recovery because it depends on complications which are not evident until 3 to 4 months after onset of Bell's palsy. This deficiency is not true of the
FPRP, which depends only on function of the affected side of the face.

The Brackmann modification (based on the Adour-Swanson recovery profile) of the House grading system attempts to correlate objective volitional function at the eyebrow and corner of the mouth with the subjective House system. This system is similar to the FPRP but provides no means for including complications when reporting outcome, as can be done with the FPRI. We could not use the Brackmann modification because many of our patients had brow and mouth movement greater than 1 cm on the affected side even if they had not completely recovered.

For uniform reporting, outcome using the House system has been included. After conversion of the FPRP and FPRI scores to this system, none of our patients fit into grades IV, V, or VI. Criteria for grades IV and V do not include presence of synkinesis and contracture. Among the more than 4500 Bell's palsy patients seen at the Cranial Nerve Research Clinic, development of axonal degeneration always resulted in development of synkinesis and contracture. Devriesse, et al. reported similar findings in their analysis of more than 1000 Bell's palsy patients, so they used only voluntary motor function when converting their patients to the House grading system (called the Bordeaux classification in the Netherlands). Because all patients with Bell's palsy recover to some extent, it cannot be classified as House grade VI (total paralysis). We conclude that the House-Brackmann grading system is not adequate for reporting outcome in Bell's palsy patients.

CONCLUSION

Our study shows that the MST using the Hilger facial nerve stimulator is an accurate indicator of prognosis in Bell's palsy. Facial nerve conduction latency is a less accurate indicator of prognosis. Furthermore, latency cannot indicate axonal degeneration before other currently used electrodiagnostic tests.

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BIBLIOGRAPHY