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ORIGINAL RESEARCH

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Electrodiagnostic testing in acute facial palsy: Outcomes and comparison of methods

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Abstract

Objective: To study the relationship between various electrodiagnostic modalities in acute facial palsy.

Setting: Academic tertiary care center.

Patients: One-hundred and six patients who presented with traumatic or non-traumatic acute facial paralysis (House-Brackmann, HB, grade 6/6) between 2008 and 2017 and underwent acute electrodiagnostic testing.

Intervention: Electroneurography (ENoG) using nasolabial fold (NLF) or nasalis muscle (NM) methods, and volitional electromyography (EMG) in all patients.

Main outcome measures: Percent degeneration of ipsilateral facial nerve compound muscle action potentials (CMAP) on NLF- and NM-ENoG, presence or absence of muscle unit potentials (MUPs) on EMG.

Results: Extent of facial nerve degeneration measured by NLF- and NM-ENoG were highly correlated ($r = 0.85$, $P < .01$) on each test and on serial testing. NLF- and NM-ENoG concordantly diagnosed ≥90% degeneration in 44 patients (80%), of whom 32 patients were diagnosed to have 100% degeneration by both methodologies. Absence of MUPs on EMG was 63% sensitive and 92% specific for ≥90% degeneration on ENoG, with a positive predictive value of 90%. For patients with Bell's palsy, percent degeneration on ENoG was also correlated to HB score at 1 year. Surgical decompression resulted in mean HB scores of 2.2 and 3.0 for patients with Bell's palsy and trauma, respectively.

Conclusions: NM-ENoG may be a valid and comparable method to NLF-ENoG for predicting the recovery of facial nerve function in acute paralysis. Absence of MUPs on EMG is a specific measure of severe degeneration and highly predictive of candidacy for surgical decompression.

Level of evidence: Level 3.

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KEYWORDS
Bell's palsy, electrodiagnostic testing, electroneurography, facial nerve decompression, facial paralysis, herpes zoster, temporal bone fracture

1 | INTRODUCTION

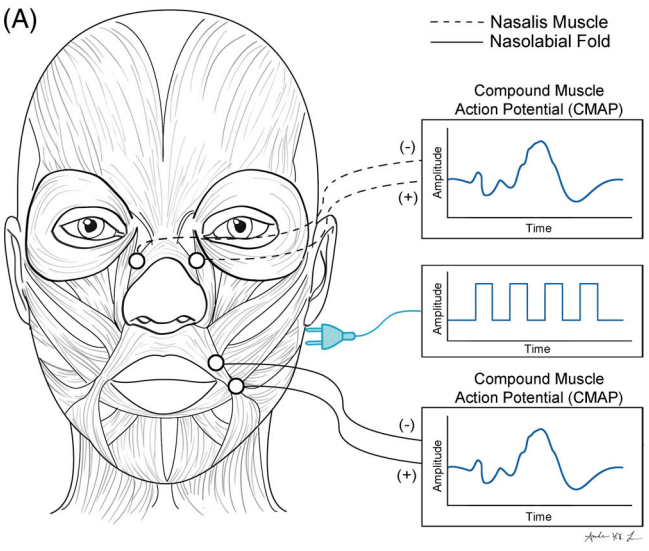
Facial palsy is a debilitating condition with pervasive physical, functional, and psychological impacts.[1] Acute idiopathic facial palsy, also known as Bell's palsy, and temporal bone fracture account for the majority of acute facial palsies due to peripheral etiologies.[2] Whereas many patients with acute facial palsy can expect excellent spontaneous recovery of nerve function,[3-5] a select group of patients are at risk for poor recovery of function despite maximal medical therapy.[6-10] Since its development in 1974 by Esslen and Fisch,[11-13] electrophysiologic testing of the facial nerve via electroneurography (ENoG) and volitional electromyography (EMG) has demonstrated value as both a diagnostic and prognostic tool in managing patients with peripherally-related acute facial palsy.[4,5,7,8,14]

ENoG testing relies on an evoked, supramaximal electrical stimulus delivered at the stylomastoid foramen to activate the ipsilateral facial nerve (Figure 1).[15] The amplitude of the consequently generated compound muscle action potential (CMAP) is dependent upon the synchronous discharge of viable nerve fibers, and reduction in CMAP amplitude is associated with Wallerian degeneration of the nerve.[6] The CMAP amplitude from the affected side is compared to the CMAP on the normal side, which serves as a control, and a percentage of degenerated nerve fibers is calculated.[15] Studies suggest that patients with Bell's palsy or temporal bone fracture with 90% or greater degeneration on ENoG and absent muscle unit potentials (MUPs) on volitional EMG in the acute phase are at high risk for poor recovery of facial nerve function despite maximal medical therapy and may benefit from surgical decompression.[6-8,14,16]

ENoG is also often performed by neurologists as part of a battery of tests to assess facial nerve function.[17] Several important methodological differences exist between ENoG testing performed within the neurology and otolaryngology communities. Whereas Esslen and Fisch[15] advocated recording from the nasolabial fold (NLF) using a hand-held electrode that is moved by the operator to find the maximum achievable CMAP and referencing to the same NLF, neurologic protocols[17] use the nasalis muscle (NM) as the recording site. A surface electrode is adhered to the skin overlying the NM on each side that is immobile, and the contralateral NM serves as a reference.

Under certain circumstances ENoG testing may have large amounts of variability[18] and must be carefully standardized.[15] Further, a limited number of studies have investigated the comparative efficacy of different records sites for ENoG testing. One study compared the variability in CMAP waveforms between NLF and NM testing and found NM testing to produce more consistent waveforms.[19] Other studies have tested the efficacy of placing electrodes on the midline or orbicularis oculi.[20,21] Likewise, the correlation between findings on ENoG and volitional EMG in the acute phase of paralysis has also not been systematically examined.

Presently, the specific ENoG methodology used for the electrophysiologic assessment of FN function is often heterogeneous and institution-dependent. Divergent practice patterns in electrodiagnostic testing can have important implications for clinical management. Differences in measured CMAP amplitude not only affects prognosis for facial nerve outcome but also drives clinical management decisions such as candidacy for surgical decompression in select patients. Therefore, we sought to study the correlation between NLF- and NM-ENoG methodologies and the role of volitional EMG testing in the acute phase of facial palsy in a large series of patients with acute facial palsy. In addition, we investigated the prognostic value of these testing modalities on eventual facial nerve outcome.



(B)

	ENoG Method	
	NLF	*NM*
Stimulus location	Stylomastoid foramen	Stylomastoid foramen
Recording location	NLF	NM
Reference location	Ipsilateral NLF	Contralateral NM
Electrode	Hand-held, moved by operator	Adhered to skin, immobile

FIGURE 1 Schematic of NLF- and NM-ENoG, A. Similarities and differences between testing methods, B. NLF, nasolabial fold; NM, nasalis muscle; ENoG, electroneurography

2 | MATERIALS AND METHODS

2.1 | Patient selection

Institutional review board approval was obtained for this study. Retrospective chart review was performed for patients who presented between 2008 and 2017, meeting inclusion and exclusion criteria. Inclusion criteria were patients who presented with unilateral acute facial nerve paralysis within 2 weeks of onset of total paralysis who underwent ENoG testing using NLF and NM methods, as well as volitional EMG. Those with iatrogenic or central causes (ie, stroke) of facial palsy were excluded. Diagnosis of acute facial paralysis was made by an otolaryngologist at time of presentation and facial function was assessed using the House-Brackmann scale.[22] Etiologic work up at time of presentation varied based on clinical symptoms, comorbidities, and assessment of the treating physician and typically consisted of neurologic exam, otoscopic exam, audiometry, with or without imaging and serum studies. Demographic, clinical, and electrophysiologic data were extracted from patient charts and compiled in a database. Unless contraindicated, all patients were treated with high dose steroids during the acute course of facial paralysis. Use of antivirals could not be consistently and reliably extracted from patient charts and therefore was not coded as a study variable.

Secondarily, we also sought to evaluate the prognostic value of various electrodiagnostic modalities on eventual facial nerve outcome. Whereas all study patients underwent concurrent NLF- and NM-ENoG to allow comparative analysis of CMAP amplitudes, only patients with at least 1 year of clinical follow up were included in the analysis of facial nerve outcomes. A further subgroup of patients underwent facial nerve decompression via a middle cranial fossa (MCF) approach. In Bell's palsy, the target of decompression was the labyrinthine segment and geniculate ganglion as these regions are suspected to be the sites of critical compression.[6-8,23-26] In trauma, the target of decompression was based on the site of injury through radiographic evaluation and most commonly involved the peri-geniculate region.

2.2 | ENoG protocol

Per institutional protocol, all patients underwent initial ENoG testing as part of a standard facial nerve test battery between days 3 and 14 after onset of total paralysis (HB 6/6) in a single neurology lab by experienced technicians and staffed by an attending neurologist. ENoG was performed using both NLF and NM methods within the same testing session.

Regardless of ENoG method, a supramaximal electrical stimulus is delivered at the stylomastoid foramen to activate the ipsilateral facial nerve. In the NLF-ENoG method, two hand-held bipolar surface electrodes (two 7 mm pads, 18 mm apart) are placed on the ipsilateral NLF and moved by the operator until the maximum evoked biphasic CMAP (Figure 1) is found. One electrode serves as a reference electrode whereas the other serves as a recording electrode. The same methodology is used to obtain a CMAP of the contralateral, unparalyzed side.

In the NM-ENoG method, an adherent surface electrode is placed on the skin over the NM on each side of the face. To record a CMAP amplitude of the paralyzed side, the ipsilateral NM electrode is used as the recording electrode and the contralateral NM electrode as reference; and vice versa for the CMAP on the unparalyzed side. For both NLF- and NM-ENoG, the ratio of CMAP amplitudes (uV) between the paralyzed and unparalyzed side is calculated to determine percent degeneration.

During the study period, although both ENoG methods were performed concurrently, only NLF-ENoG values were used for clinical counseling and management including consideration of surgical intervention. Patients whose NLF-ENoG demonstrated less than 90% degeneration were recommended to undergo repeat ENoG testing in several days unless improvement in facial function was noted

TABLE 1 Demographic, clinical, and demographic characteristics of cohort (n = 106)

Age, median (range)	36.4 (2-80)
Gender, n (%)	
Female	44 (42)
Male	62 (58)
Etiology of paralysis, n (%)	
Bell palsy	64 (60)
Trauma	31 (29)
Other	11 (10)
Maximum degeneration on NLF-ENoG, n (%)	
0%-49%	20 (19)
50%-89%	46 (43)
90%-100%	40 (38)
Maxiumum degeneration on NM-ENoG, n (%)	
0%-49%	17 (16)
50%-89%	42 (40)
90%-100%	47 (44)
EMG MUPs present, n (%)	
Absent	40 (38)
Present	66 (62)
Treated with steroids, n (%)	101 (95)
Surgical decompression, n (%)	29 (27)
Patients with at least one-year follow-up, n (%)	46 (44)
Surgery	18 (39)
No surgery	28 (61)
HB score at 1 year for non-surgical patients, n (%)	
1	14 (50)
2	4 (14)
3	6 (21)
4	2 (7)
5	0 (0)
6	2 (7)

Abbreviations: NLF, nasolabial fold; NM, nasalis muscle; ENoG, electroneurography; EMG, electromyography; MUP, muscle action potential.

clinically. This was repeated approximately every 3 days until no further increase in percent degeneration was found, degeneration reached 90% or higher (in which case they were assessed for surgical candidacy), or spontaneous facial motion returned (HB 5 or better). ENoG testing was not performed greater than 14 days following the onset of paralysis as surgical decompression would no longer be considered at this point.[7]

2.3 | Volitional EMG protocol

Per institutional protocol, volitional EMG was also performed as part of the standard test battery. Volitional EMG is especially important when ENoG reaches 90% degeneration and patients may be a candidate for decompression surgery. Presence of motor unit potentials (MUPs) on volitional EMG suggests early deblocking, portends a favorable prognosis for functional recovery, and is a contraindication to surgical decompression. EMG was performed using a concentric (bipolar) needle electrode inserted by the attending neurologist as part of the standard facial nerve test battery during the same session as ENoG testing, in the acute phase of paralysis. The concentric needle electrode consisted of a needle with a central cannula that contained an insulated wire, which served as the recording electrode whereas the outside cannula served as the reference electrode. Whereas there is discomfort associated with needle insertion, this method was chosen because it allows for precise measurement of motor unit potentials with minimal background noise, as compared to surface

TABLE 2 Correlation between severe degeneration on NLF- or NM-ENoG and the presence of MUPs on EMG

n	≥90% degeneration	<90% degeneration
MUPs absent	36	4
MUPs present	21	45

Abbreviations: ENoG, electroneurography; MUP, motor unit potentials; EMG, electromyography.

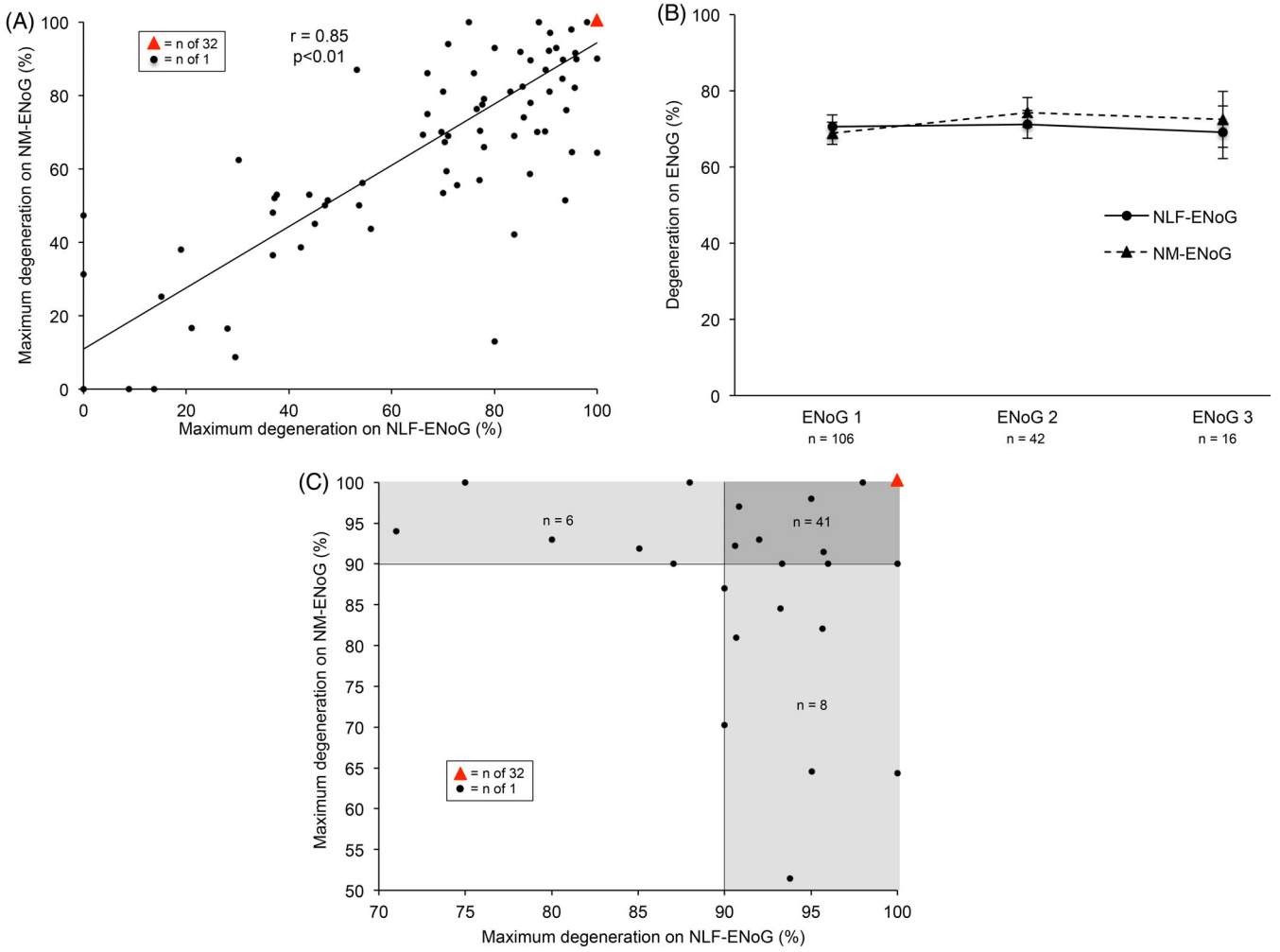


FIGURE 2 Panel A, maximum degeneration on NLF- and NM-ENoG are highly correlated ($r = 0.85$, $P < .01$) for all patients (n = 106). Panel B, degeneration on NLF- and NM-ENoG were similar on serial testing. Error bars, SE of mean. ENoG 1, $P = .26$; ENoG 2, $P = .53$; ENoG 3, $P = .26$. Panel C, plot of patients with ≥90% maximum degeneration on either NLF- or NM-ENoG. NLF, nasolabial fold; NM, nasalis muscle; ENoG, electroneurography

electrodes. The specific muscle group(s) tested for each patient was at the discretion of the attending neurologist and most commonly involved orbicularis oculi or oris. After electrode insertion, patients were asked to make forceful contractions of the muscle group under study and MUPs were documented as present or absent. EMG testing was performed serially in cases of repeat ENoG testing.

2.4 | Statistical analysis

Percent degeneration of ipsilateral facial nerve at presentation on NLF-ENoG was correlated with percent degeneration on NM-ENoG using Spearman rank correlation analysis. NLF- and NM-ENoG scores were averaged at each testing interval and differences in percent degeneration between NLF- and NM-ENoG were compared with students paired *t* test with significance defined as $P < .05$ on two-tailed test. The sensitivity and specificity of the presence of MUPs on EMG testing for degeneration of >90% on ENoG testing was calculated using values of maximal degeneration on NLF- or NM-ENoG and EMG test results from the date that maximum degeneration was measured on ENoG testing. HB scores were stratified by maximum percent degeneration reached on ENoG (0%-49%, 50%-89%, 90%-100%) and NLF versus NM methodology. To specifically investigate the prognostic value of ENoG on the natural history of facial function recovery, patients who reached 90% degeneration or greater were additionally stratified by non-surgical versus surgical intervention. Differences between mean HB score at 1 year were assessed using Mann-Whitney *U* test. SPSS version 25[27] was used for statistical analysis.

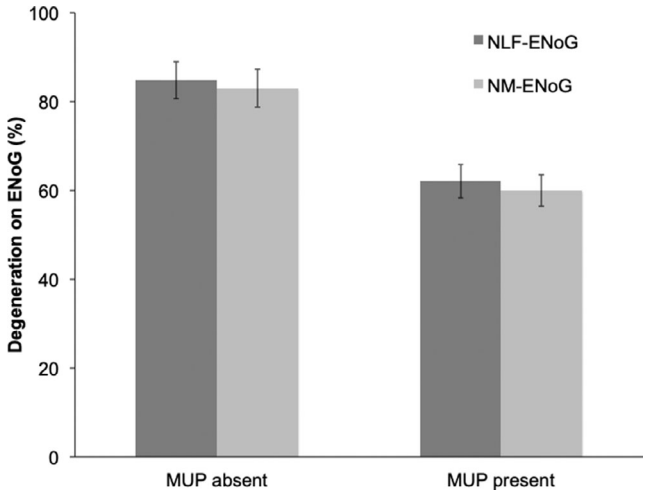


FIGURE 3 Absent MUPs on EMG are associated with greater degeneration on EnoG. No statistically significant difference existed between ENoG methods stratified by the presence or absence of MUPs. MUP, muscle action potentials; EMG, electromyography; ENoG, electroneurography

3 | RESULTS

One hundred and six patients met inclusion and exclusion criteria and their clinical information are shown in Table 1. The average age was 36 years (range 2-80) and 44 (42%) patients were female. Sixty-four (60%) patients suffered paralysis due to Bell's palsy (idiopathic), 31 (29%) due to trauma, and 11 (10%) due to other causes, including Ramsay Hunt syndrome (n = 10) and complications of otitis media (n = 1). On NLF-ENoG 20 (19%) patients had 0%-49% maximum degeneration, 46 (43%) had 50%-89% degeneration, and 40 (38%) had 90%-100% degeneration. On NM-ENoG 17 (16%) patients had 0%-49% maximum degeneration, 42 (40%) had 50%-89% degeneration, and 47 (44%) had 90%-100% degeneration. One-hundred and one (95%) patients were treated with steroids and five patients (5%) were not. Of the five patients who did receive treatment with steroids, three patients suffered paralysis secondary to trauma. A rationale was not specified in four patients and one patient was not offered steroids due to a belief by the treating physician that there was inadequate evidence to support steroid use at that time. Twenty-nine patients (27%) underwent facial nerve decompression.

Overall, there was a high degree of concordance ($r = 0.85$, $P < .01$) between maximum percent degeneration measured by NLF- and NM-ENoG for all patients (Figure 2A). As serial ENoG is often performed to determine the trajectory of neural degeneration, we also compared differential NLF- and NM-ENoG performance in patients who underwent up to three serial ENoG studies and the mean CMAP amplitude was found to be similar between NLF- and NM-ENoG both at initial and subsequent testing (Figure 2B). Differences in mean

TABLE 3 Average HB score at 1 year stratified by maximum degeneration on NLF- or NM-ENoG and etiology of facial palsy (non-surgical patients)

	0-49%	50-89%	90-100%
Maximum degeneration on NLF-ENoG			
Bell palsy; average (n)	1.2 (6) Range, 1-2	1.9 (10) Range, 1-6	2.5 (2) Range, 2-3
Trauma; average (n)	4 (1)	3 (1)	3 (1)
Other; average (n)	1 (1)	–	3 (6) Range, 1-6
Maximum degeneration on NM-ENoG			
Bell palsy; average (n)	1 (6) Range, 1	1.8 (10) Range, 1-6	3.5 (2) Range, 3-4
Trauma; average (n)	–	3.5 (2) Range, 3-4	3 (1)
Other; average (n)	–	1.5 (2) Range, 1-2	3.2 (5) Range, 3-6

Abbreviations: HB, House-Brackmann; NLF-ENoG, nasolabial fold electroneurography.

percent degeneration between NLF- and NM-ENoG were not statistically significant (ENoG 1, $P = .26$; ENoG 2, $P = .53$; ENoG 3, $P = .26$) for all testing intervals. There were six of 42 (14%) patients who had ≥90% degeneration on either NLF- or NM-ENoG at their second round of ENoG testing who had <90% degeneration on either modality previously. No patients had ≥90% degeneration on either NLF- or NM-ENoG at their third set of testing that had not had ≥90% degeneration on at least one ENoG modality previously.

As patients with ≥90% degeneration are at highest risk for poor outcome and may be candidates for surgical decompression, we specifically examined the impact of differential ENoG methodology in patients with severe degeneration (Figure 2C). Overall, 55 patients had ≥90% degeneration on at least one ENoG methodology. NLF- and NM-ENoG concordantly diagnosed ≥90% degeneration in 41 patients (75%), of whom 32 patients were diagnosed to have 100% degeneration by both methodologies. Five patients diagnosed to have ≥90% degeneration on NLF-ENoG were found to have <90% degeneration on NM-ENoG. Conversely, six patients were found to have ≥90% degeneration on NM- but not NLF-ENoG, including two patients with 100% degeneration who recovered to HB 3 and 4, respectively.

Sixty-six (62%) patients had muscle action potentials on volitional EMG. A high degree of concordance was also noted between absence of MUPs on EMG and percent degeneration regardless of ENoG methodology (Table 2; Figure 3). Indeed, MUPs were present in 92% of patients who sustained <90% degeneration on either NLF- or NM-ENoG, and absent in 63% of patients who sustained ≥90% degeneration (Table 2), yielding sensitivity and specificity of 63% and 92%, respectively, for predicting severe degeneration (≥90% degeneration) using absence of MUPs as a stand-alone indicator. In particular, the absence of MUPs had a 90% positive predictive value for ≥90% degeneration.

In a secondary analysis to investigate the prognostic value of ENoG in the natural history of facial paralysis, 46 patients (43%) had at least one-year clinical follow-up for assessment of facial nerve outcome as a function of percent degeneration on ENoG. We first examined HB outcomes of patients who did not undergo surgical decompression (n = 28). Table 3 shows the one-year HB scores of patients who were managed non-surgically stratified by etiology of paralysis and by maximum percent degeneration reached on NLF-ENoG. In patients suffering from Bell's palsy, there was a trend of poorer recovery of function with HB scores of 1.2, 1.9, and 2.5 for degeneration of 0%-49%, 50%-89%, and 90%-100%, respectively, on NLF-ENoG. Similarly, Bell's palsy patients who reached maximum percent degeneration of 0-49%, 50-89%, and 90-100% on NM-ENoG and who did not undergo surgical decompression had mean one-year HB scores of 1.0, 1.8, and 3.5, respectively (Table 3). Interestingly, one patient with Bell's palsy remained HB 6/6 at 1 year despite maximum ENoG degeneration of 70% and treatment with oral steroids. Imaging was not available for this patient at the time of paralysis, but an MRI 2 years prior to the onset of facial paralysis showed evidence of cerebrovascular accident. In both NLF- and NM-ENoG, one-year HB scores of patients whose facial paralysis was due to trauma or other causes were found to be more variable. Ramsay Hunt was the cause of paralysis in four patients, with recovery to HB 1, 2, 3, and 4.

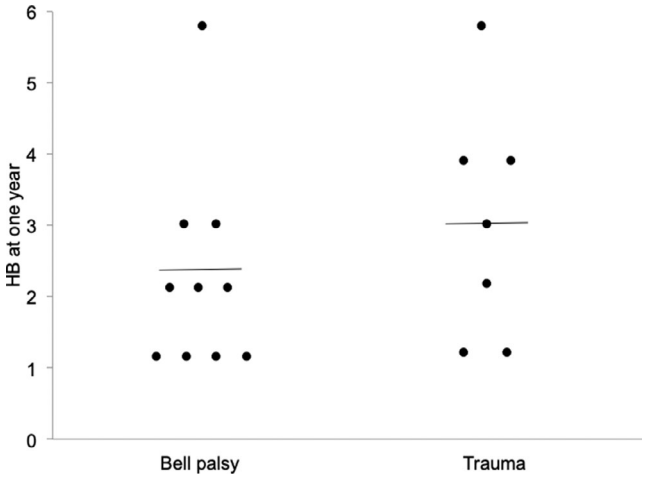


FIGURE 4 Outcomes (HB score) of patients with surgical decompression at 1 year ($P = .35$). HB, House-Brackmann score

Next, we examined the 1-year HB scores of patients who met electrodiagnostic criteria and elected to undergo surgical decompression (n = 18). Patients with Bell's palsy and trauma recovered to a mean HB scores of 2.2 and 3.0, respectively, representing a non-statistically significant difference ($P = .41$) (Figure 4). In 10 patients with Bell's palsy, four recovered to HB 1, three to HB 2, two to HB 3 and of note, intraoperative iatrogenic injury during decompression was suspected in one patient who did not recover from HB 6 at 1 year.

4 | DISCUSSION

To date, this study presents one of the largest cohorts of patients with acute facial palsy who underwent ENoG testing. Findings in this study demonstrate a high degree of concordance between two common ENoG methods and at multiple testing intervals. Whereas NLF-ENoG has traditionally been practiced by otolaryngologists for the assessment of facial nerve palsy[3-5,7,8,23,28,29] as a result of pioneering work by Ugo Fisch and Erlo Esslen,[11-13] it has distinct methodologic and electrophysiologic differences compared to NM-ENoG more commonly practiced in clinical neurology laboratories.[17] In addition to recording from distinct facial muscle groups, NM-ENoG also uses a reference electrode on the contralateral face that is more electrophysiologically accurate, whereas NLF-ENoG uses a reference electrode adjacent to the recording site ipsilaterally. Further, NLF-ENoG lacks a consistent recording site and requires manipulation by the operator to find the site of maximum amplitude. NM-ENoG is less operator-dependent as electrodes are fixed to the skin. As part of a standardized facial electrophysiology battery for many clinical neurology laboratories, NM-ENoG is also used more broadly at clinical centers regardless of available neurotologic expertise. For both testing methodologies other waveform characteristics such as latency may also

play a role, but was not a part of the specific protocols evaluated in this study, and historical studies[13,30] establishing the validity of ENoG have found amplitude the most accurate predictor.

Importantly, there was also a high degree of concordance for patients with severe degeneration (≥90%) who are most at risk for poor functional recovery and the correlation was even stronger for patients who had 100% degeneration (89%). Both tests are predictive of the return of facial nerve function at 1 year. Thus, NM-ENoG may be a suitable alternative to NLF-ENoG and permit the evaluation of acute facial palsy patients in centers where no NLF-ENoG protocol exists. In particular, the use of a contralateral reference electrode in NM-ENoG is a superior physiologic design compared to the use of an ipsilateral electrode in NLF-ENoG that may receive the same stimulus as the recording electrode. Technicians at our institution have observed much less variability when performing NM-ENoG as compared to NLF-ENoG. Further, NM-ENoG is a more commonly used neurologic test that is more widely available and its adoption may lead to greater penetrance of ENoG use for patients suffering from acute facial nerve paralysis. It is our opinion that, where possible, NM-ENoG should be common practice for assessing patients with acute facial nerve paralysis.

Whereas EMG is most commonly used in the chronic phase of paralysis to identify activity at the neuromuscular interface, findings from this study also reinforce the importance of volitional EMG in the acute phase of paralysis, as a complementary nerve conduction study to ENoG. Absence of MUPs on EMG is an important confirmatory test in patients with severe degeneration on ENoG who may be candidates for surgical decompression as presence of MUPs may be indicative of early deblocking and favorable functional recovery.[6] Interestingly, findings from the present study additionally suggest that the absence of MUPs is also highly specific for 90% degeneration on ENoG. Indeed, we found that if MUPs were used in isolation without ENoG in evaluating patients with Bell's palsy or trauma for ≥90% degeneration, it would have a positive predictive value for surgical candidacy (≥90% degeneration and absent MUPs) of 90%. The convergence between these two distinct electrophysiologic modalities may additionally suggest unique and important neurophysiologic phenomenon occurring at the 90% degeneration threshold that has been identified in clinical studies to be prognostic of poor functional recovery.[6-8,14] However, the prognostic significance of EMG alone for the recovery of facial nerve function remains unstudied and should continue to be used in conjunction with ENoG testing.

Moreover, the present study also included a subset of patients with available facial nerve outcomes at 1 year after paralysis. Findings in this group demonstrate the prognostic value of ENoG in that maximum percent degeneration reached on ENoG in the acute period after total paralysis is correlated to facial nerve function at 1 year (Table 3), in concordance with previous studies.[2,4,5,21,31] Although larger sample sizes are necessary for statistical comparison, the relationship between ENoG amplitude and facial nerve function was stronger in Bell's palsy than traumatic nerve injury. This is likely related to the variability of the mechanism of traumatic nerve injury, which may include impingement, crush, or penetrating types of facial nerve injury. This in turn may lead to heterogeneity in the extent and pattern of axonal degeneration and regeneration and underlie differences in functional recovery.

Subgroup analysis of patients who underwent surgical decompression of the facial nerve after meeting clinical and electrodiagnostic criteria demonstrated clinical outcomes that are broadly comparable to previous studies.[7,8] However, the current series is limited by its retrospective nature and lack of adequate statistical power in the surgical subgroup to draw direct comparisons in outcome to previous studies. Furthermore, as only one patient with Bell's palsy, who met electrodiagnostic criteria for surgical decompression during the study period, elected for medical management (recovered to HB 3), a non-surgical comparison group could not be established. Consistent with the most recent Academy of Otolaryngology—Head & Neck Surgery consensus statement that was unable to recommend for or against facial nerve decompression for Bell's palsy,[32] clinical outcomes in this study relate to both the potential benefits and serious risks associated with surgical decompression. The majority of patients at risk for unfavorable FN outcome recovered to HB 1 or 2 following decompression, consistent with previous prospective and retrospective studies.[7,8] However, one patient likely sustained iatrogenic injury during decompression with serious consequences to facial nerve outcome. Whereas this study lacks a control group of patients who met criteria for surgical decompression but did not receive surgery, making it impossible to formally assess the number needed to treat, it highlights the potential risks and challenges associated with MCF FN decompression, even for experienced surgeons. The importance of surgeon-related factors is seldom studied but likely plays an important role when evaluating the efficacy of delicate and challenging surgical procedures. These factors should be carefully discussed with the patient when weighing the decision to pursue facial nerve compression.

Whereas our current data series represents one of the largest studies to date in electrodiagnostic testing for acute facial paralysis, only 43% had follow-up at 1 year for assessment of long-term functional outcome, which limits the statistical power of secondary analyses investigating the prognostic value of electrodiagnostic modalities. Further, the patient cohort is highly selected based on clinical decision algorithms and referral patterns and is not likely to be reflective of all patients with acute onset of facial paralysis. Additionally, this study was performed by retrospective chart review and the grading of facial nerve function was both non-blinded and subject to inter-rater variability. Although this study focused on electrodiagnostic characteristics of acute facial paralysis, medical management has an important impact on functional recovery. Strong evidence suggests that steroid therapy improves functional recovery in Bell's palsy,[33] whereas the evidence regarding antiviral therapy is mixed.[34] Almost all patients in the present study received steroid therapy and information on anti-viral treatment was not consistently available. Furthermore, as a retrospective study spanning a study period during which there was on-going evolution in optimal medical therapy, the impact of differential medical management strategies on functional outcome could not be controlled.

In summary, we report on the correlation between several electrodiagnostic tests used in the acute phase of facial paralysis and their prognostic value for functional recovery. NM-ENoG may be a valid and comparable method to NLF-ENoG for predicting the recovery of facial nerve function in acute paralysis whereas absence of MUPs on EMG was also highly predictive of severe degeneration. Both testing

methodologies are comparable at predicting facial nerve function at 1 year and this study provides outcomes for a limited number of patients who received facial nerve decompression. As NM-ENoG is more broadly practiced in clinical electrophysiology, this may present an opportunity for increased penetration of electrodiagnostic testing in appropriate patients presenting with acute facial nerve paralysis.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ETHICS STATEMENT

This study received institutional review board approval.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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