

Evaluation of the facial nerve via electroneuronography (ENoG)

By Douglas L. Beck and James W. Hall III

Facial nerve paralysis is a debilitating condition.¹ Patients with the condition often experience severe emotional and psychological impacts because of facial disfigurement and the resulting physical limitations and difficulties associated with speaking, drinking, eating, and making facial expressions. For many of these patients, socialization is extraordinarily limited and difficult.

The evaluation of facial nerve viability by means of electroneuronography (ENoG) is critically important in the management of facial nerve disorders. Depending on the outcome of the ENoG evaluation, the physician may choose to “watch and wait” or may decide to intervene surgically. Surgery is by no means trivial, and its utility is often directly determined via the ENoG evaluation.

Facial nerve disorders have a variety of etiologies including: Bell’s palsy (BP), iatrogenic (surgically induced) injury, trauma to the temporal bone secondary to motor vehicle accidents (MVA), otitis media, herpes zoster oticus, multiple sclerosis, Melkersson-Rosenthal syndrome, mastoiditis, mumps, chicken pox, Guillain-Barré syndrome, central nervous system disorders (e.g., stroke glomus jugulare, meningioma, and facial nerve neuroma.²

AUDIOLOGISTS AND ENOG

ENoG is used by audiologists to evaluate the integrity of the facial nerve. This procedure involves electrical stimulation of the facial nerve at or near the stylomastoid foramen and the subsequent measurement and interpretation of the motoric response as recorded at or near the nasio-labial fold. Although other procedures are used as well (see below), the ENoG test is the only relatively objective measure of facial nerve integrity.

The ENoG compares the neurophysiologic response of the normal side of the face to that of the abnormal side. The findings help in determining (1) whether or not surgical intervention is recommended and (2) the probable prognosis.

This procedure falls within the scope of practice of audiologists as defined both by the American Academy of Audiology and the American Speech-Language-Hearing Association.^{3,4} Audiologists have been evaluating facial nerve function for many decades, starting perhaps with

measurement of the acoustic reflex during immittance test batteries.

Alternative tests of facial nerve function

Many other tests of facial nerve function have been, and continue to be, used. These include, among others: the Hilger test, electromyography, acoustic reflex testing,⁵ evoked accelerometry, antidromic nerve potentials, MRI and CT radiologic evaluations, maximal nerve stimulation tests, minimal nerve stimulation tests, transcranial magnetic stimulation,⁶ and blink reflex tests.⁷

THE HOUSE-BRACKMANN SCALE

The most commonly used tool for grading facial nerve function is the House-Brackmann (HB) scale.⁸ The HB scale is used to approximate the quantity of volitional motion the patient has based on clinical facial presentation. Although the HB scale is derived from clinical observation, and variation among observers exists, this scale does allow us to grossly describe the characteristics and degree of facial nerve motion.

The HB scale has six grades. Each grade is reported as a fraction (e.g., 1/6 = grade one). A grade one presentation is perfectly normal. Grade two indicates a slight or mild weakness. Grade three is a moderate weakness with good (or normal) eye closure. Grade four is a moderate weakness with no volitional eye closure. Grade five is a severe weakness. Grade six is a total facial paralysis.

In some respects, one might argue that only grade six (6/6) presentations require ENoG testing. After all, the purpose of the test is to determine if the facial nerve is neurophysiologically intact. And, since it can be inferred that in a patient who has any volitional motion (as would be indicated by grades one through five) the facial nerve is indeed intact, then there is no need for ENoG testing.

Nonetheless, it is useful to chart the progress of facial nerve disorders via ENoG even in cases without a grade six presentation. Additionally, it is sometimes difficult to discern if an apparent grade five presentation is really a grade five or if it is actually a grade six. In some cases, the extremely limited motion seen is from the masseter, or from musculature motion from the contralateral (normal) side. In such

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cases, the motion seen does not indicate an intact facial nerve.

EnoG evaluation allows a subclinical analysis which may impact medical or surgical decisions. However, once a visible improvement is apparent regarding facial nerve reanimation, further ENoG testing provides little information.

PHYSIOLOGY OF THE FACIAL NERVE

Humans have 12 pairs of cranial nerves, but our discussion will be limited to the seventh nerve, the facial nerve. Each facial nerve has some 10,000 fibers.⁹ About two-thirds of the fibers are motor fibers, and about one-third are sensory. The sensory portion of the facial nerve is the *nervus intermedius*. It is estimated that only half of the motor fibers need to be functioning for a person to have essentially normal facial nerve function.

As the facial nerve exits the brainstem, it traverses the cerebellopontine angle (CPA) to the medial end of the internal auditory canal (i.e., the *porus acusticus*). Progressing distally to the stylomastoid foramen, the facial nerve includes the labyrinthine segment, the tympanic segment, the pyramidal bend, the mastoid portion, and, finally, the stylomastoid foramen, where the nerve exits the skull and is readily positioned within the parotid gland.

As the main trunk of the facial nerve enters the parotid region, it separates into two main divisions—superior and inferior. The superior division then divides into three sections—temporal, zygomatic, and buccal divisions. The inferior division divides into the buccal, mandibular, and cervical branches.¹⁰

BELL'S PALSY:

Bell's palsy (BP) is probably the single most common cause of facial nerve disorders. In the general population, BP occurs in about 15 of every 100,000 people (0.00015%). The condition will recur in 5% to 10% of patients.

The time from onset of BP to total, unilateral facial paralysis is usually about 24 to 48 hours. Spontaneous recovery is common, occurring among some 75% to 80% of all patients, usually in about 3 to 4 weeks. However, about 15% to 20% of patients maintain a lifelong residual weakness following resolution of the BP. About 5% of all patients have permanent weak-

ness worse than HB grade 4/6.

The etiology of BP is unknown. Viral neuropathy, bacterial infections, genetics, environmental and many other causes have been suggested. It is likely that there are a number of reasonable etiologies. Various treatment options are available for BP, including surgical intervention, "waiting and watching," acyclovir treatment,¹¹ and other medical options.

Wallerian degeneration and timing of ENoG

In cases of BP and other facial nerve injuries, it takes some 72 hours for Wallerian degeneration (WD), which is the denervation of the neural fibers, to occur.¹² That means that if a patient is found to have an HB grade six facial presentation 1 hour after the onset of BP, administering ENoG would be likely to produce a somewhat normal result. That is, because the facial nerve would not yet have not completed WD, the fibers would still be physiologically intact, although non-functional volitionally. The resultant ENoG would produce a false negative result if the test was performed prior to complete WD.

Therefore, it is important to wait approximately 72 hours before performing the first ENoG. However, at the other

end of the timing window is the 21-day maximum.

Specifically, if ENoG and subsequent surgical intervention are delayed past a 21-day post-onset window, the test and possible surgical intervention are of questionable value. In essence, the ENoG should be performed for the first time at about 72 hours post-onset and again at intervals of 3 to 5 days until a trend is observed and confirmation can be obtained.

If the trend and confirmation are determined prior to 21 days post-onset, surgical intervention may be an option. However, if the ENoG is not conducted until more than 21 days post-onset, it is of little clinical use. Specifically, an ENoG result obtained 8 weeks after onset of facial paralysis is difficult, if not impossible, to interpret.

TYPES OF FACIAL NERVE INJURY

Fisch has used three terms—neuropraxia, neurotmesis, and axonotmesis—for the three primary types of facial nerve injury.¹³ It should be noted that ENoG cannot differentiate between neurotmesis and axonotmesis.

Neuropraxia is the most common find-

Stimulus

❖ Transducer	pair of electrodes
❖ Site	stylomastoid foramen region
❖ Orientation	horizontal with cathode (negative) posterior
❖ Type	electrical pulse (shock) of constant current or voltage
❖ Mode	continuous
❖ Duration	0.2 msec (200 µsec)
❖ Rate	1.1/sec
❖ Laterality	unilateral, assess uninvolved side first
❖ Intensity	milliamperes (mA) sufficient to produce a supramaximal response (usually 15 to 25 mA)

Acquisition

❖ Amplification	X 5000 (or less if response exceeds 1000 µV)
❖ Filter setting	3 to 5000 Hz
❖ Notch filter	none
❖ Analysis time	20 msec
❖ Pre-stimulus time	1 msec
❖ Number of sweeps	1 to 20
❖ Electrodes	Channel 1-Nasolabial fold (corner of mouth to base of nose) on side ipsilateral to stimulation Ground (common)-forehead
❖ Interelectrode impedance	less than 5000 ohms

Table 1. Summary of ENoG measurement parameters.

ing associated with BP. In this situation, the patient experiences paralysis without peripheral nerve degeneration. The ENoG demonstrates a normal or reduced response. The nerve fibers and the sheath are anatomically intact, but they are not responsive to volitional commands.

Neurotmesis is the worst possible outcome. The ENoG will demonstrate essentially no response (i.e., flat line). Neurotmesis represents a total anatomic separation with a very poor prognosis.¹⁴

Axonotmesis has been described as an inner nerve fiber disruption despite an intact outer casing (epineurium). The ENoG will show no response. An analogy to help describe axonotmesis is that of an insulated cable with thousands of wires inside that has been manually bent back and forth. Although the cable remains intact, many of the internal wires have snapped and separated.

ENOG PROTOCOL

At first glance, ENoG may appear to be a relatively simple electrophysiologic measurement technique. The stimulus electrode is placed directly over the facial nerve within a broad anatomic region. The electrodes located in the nasolabial fold (base of the nose to the corner of the mouth) do not require precise placement. The amplitude of the response is, by auditory electrophysiologic standards, gigantic, often exceeding 2000 microvolts (over 2 millivolts).

A protocol for recording ENoG is summarized in Table 1. As with other clinical protocols, the procedure is explained to the patient at the outset. Most patients undergoing ENoG readily agree to the discomfort of electrical stimulation since they are very troubled by their facial nerve dysfunction and want to do whatever possible to cure the condition.

The clinician should take care to use terms that do not alarm patients. For example, say “we will present stimulation to the nerve that goes to your face” rather than “we will give you a little electrical shock to test your facial nerve.”

Prior to beginning the procedure, the clinician should carefully assemble necessary supplies (tape, skin-abrading material, conducting paste, electrodes), prepare the skin for stimulus and response electrode application, and check interelectrode impedance for all electrode combinations.

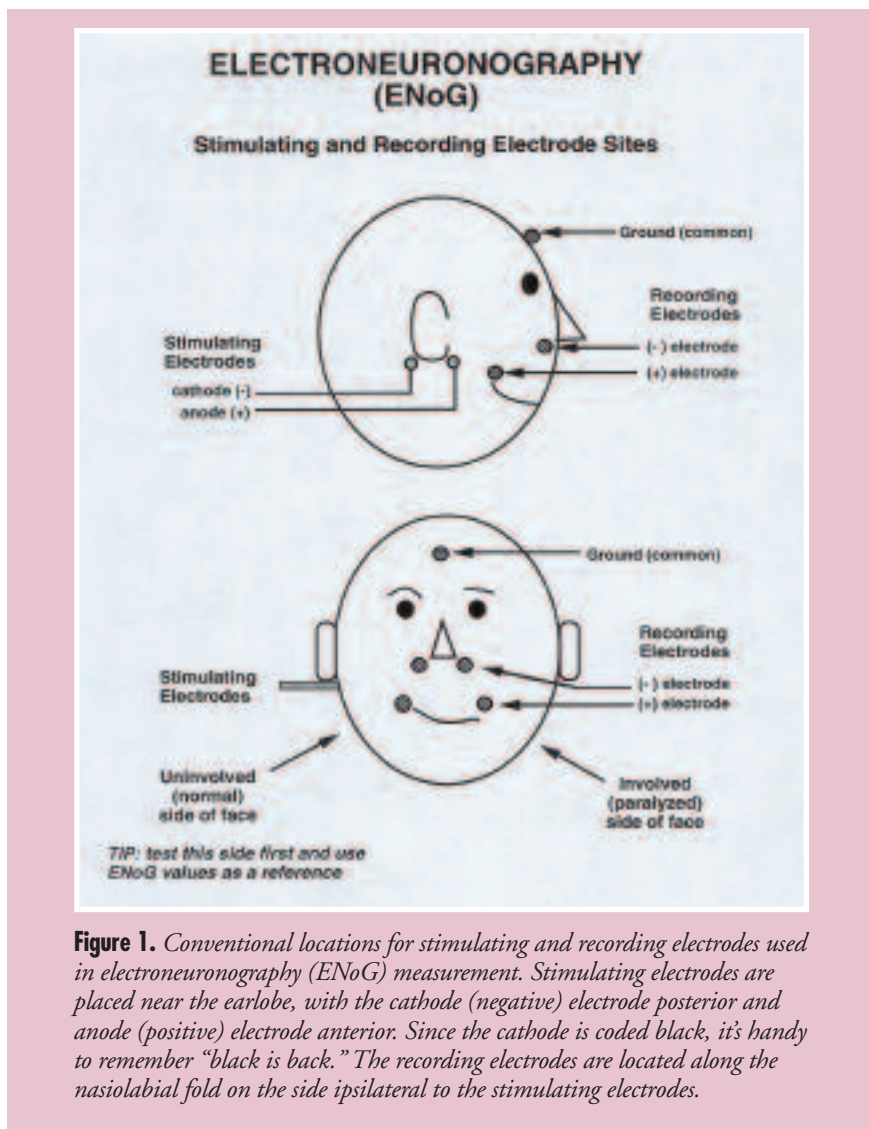


Figure 1. Conventional locations for stimulating and recording electrodes used in electroneuronography (ENoG) measurement. Stimulating electrodes are placed near the earlobe, with the cathode (negative) electrode posterior and anode (positive) electrode anterior. Since the cathode is coded black, it's handy to remember “black is back.” The recording electrodes are located along the nasolabial fold on the side ipsilateral to the stimulating electrodes.

ANALYZING THE RESULTS

The fundamental principle of ENoG analysis is a quantified comparison of the distal facial nerve response comparing ipsilateral to contralateral sides. Therefore, in ENoG measurement, the same technique should be used for each side. Conventionally, the “good” side is stimulated first. Bipolar electrical stimulation (in milliamperes, mA) is delivered to the region at the base of the ear, where the facial nerve trunk exits the stylomastoid foramen in the mastoid and then courses anteriorly toward the face.

Note in Figure 1 that the negative (black) stimulation electrode is located just behind the earlobe. Avoid inching the stimulating electrodes anteriorly ahead of the ear to enhance detection of an ENoG response, as the likelihood of inadvertently stimulating a masseter muscle response (and mistaking it for a facial nerve

response) is greater in the anterior electrode location. Typically, testers will sample several stimulation locations in this region to verify that they are measuring the largest possible response. As a rule, the optimal response is recorded with the two electrodes positioned parallel to the nerve (along a horizontal line from just behind to just in front of the earlobe).

If you suspect that the response is contaminated by masseter response or may be solely a masseter response, here are two things to observe: If the response is “triphasic,” it may well be the masseter. Additionally, watch the patient’s mandible closely. If the jaw is moving in rhythm with the stimulation, it is likely that the response includes some/all masseter response rather than being what you want—an isolated, facial nerve response.

The measurement electrodes are located on the nasolabial fold (Figure 1),

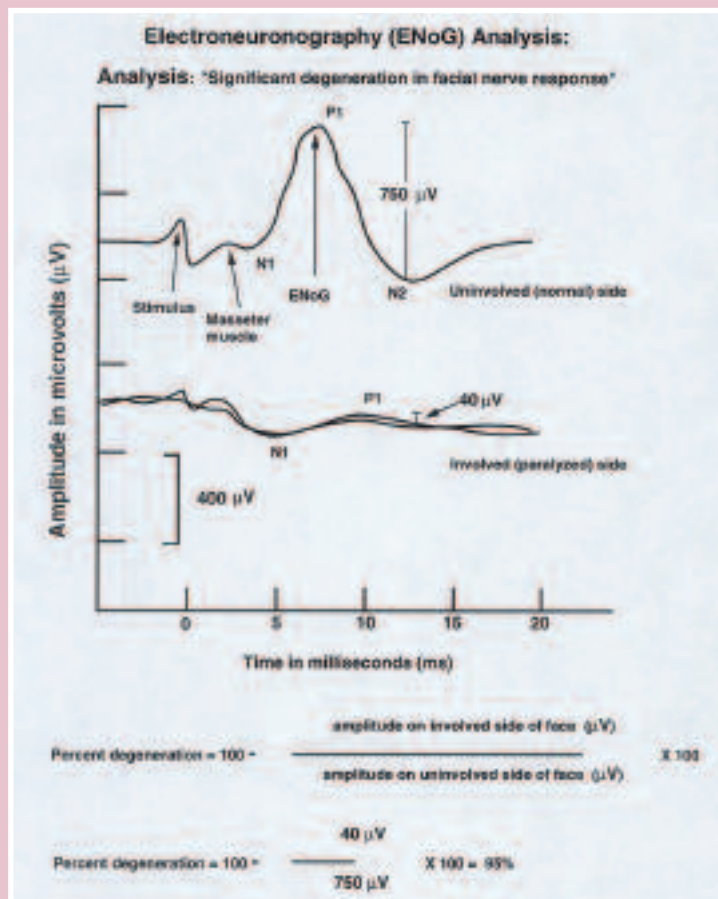


Figure 2. An example of a normal ENoG waveform (top) and a very abnormal waveform (bottom). The major peak in the ENoG waveform usually occurs within the region of 5 msec to 8 msec. Earlier latency peaks may be masseter muscle artifact rather than facial nerve/muscle responses. Normal ENoG amplitude ranges from about 500 microvolts to over 2000 microvolts. Facial nerve degeneration is calculated by comparing ENoG amplitude for the involved (paretic) side to the uninvolved (normal) side, as illustrated in the equations at the bottom of this figure. Significant degeneration is >90%.

ipsilateral to the side of stimulation. Be sure to check inter-electrode impedance for each side (below 5000 ohms is desirable) and verify that the ipsilateral electrodes are plugged into the electrode box/strip prior to stimulation. Measurement electrodes may be fixed (disc electrodes taped to the base of the nose and corner of the mouth) or the electrodes (within a bar) may be placed in these locations by the tester and systematically moved for subsequent stimulations in an attempt to maximize the response. Two measurement electrodes may also be placed near the forehead, outside the eyebrows. Then, either a two-channel or two single-channel ENoG recordings can be made from two branches of the facial nerve. This approach is often taken during intraoperative monitoring of facial

nerve function.

A basic principle of ENoG is to verify that the stimulus is delivered efficiently and effectively. That is, we wish to test using the lowest possible stimulus while obtaining a maximal response. If we accomplish this, a further increase in stimulus intensity will not yield any increase in response amplitude.

Although the literature does not appear to offer a maximal stimulus level, good clinical protocol suggests there is no reason to stimulate above 40 mA. The intensity level that first produces a maximum response varies from patient to patient, and even from side to side within a patient. Usually, however, the stimulus intensity must exceed 20 mA to 25 mA before a supramaximal level is reached.

Symmetry in the intensity level from

one side to the other is not important. However, it is critical to assure that the biggest possible facial nerve response is obtained from each side. Because the ENoG is such a large physiologic response (often exceeding 1000 mVolts) and because few (1 to 10) stimulus presentations are needed to average a response, an experienced tester can confidently find the supramaximal response in several minutes. Examples of a normal and an abnormal ENoG waveform are illustrated in Figure 2.

Despite the size of the ENoG response and the apparent simplicity of its measurement, problems can and do arise clinically with patients with suspected facial nerve dysfunction. The most common measurement problems, and some helpful solutions, are summarized in Table 2. A robust and reliable response is unequivocal.

There are, clearly, many possible explanations for a suboptimal response. Some can be quickly and effectively addressed by slight modification in test protocol, but others (e.g., obesity) may be difficult to resolve.

Considerable clinical experience and judgment are required to consistently record quality ENoG responses and avoid errors in interpretation. One must bear in mind the serious consequences of the two types of errors in interpretation. If a response is mistakenly identified on the involved side, i.e., artifact or perhaps masseter muscle is accepted as a response, this "false-positive" error can lead to a decision to withhold potentially effective surgical therapy. If, on the other hand, the tester incorrectly concludes that there is no ENoG (due to measurement problems summarized in Table 2), then potentially irreversible and damaging therapy (e.g., surgery) may be implemented for a patient who, in fact, has a normal facial nerve response.

Latency is not critically important for ENoG. Typical N1 latencies are less than 6 milliseconds. Of primary importance is amplitude. As mentioned earlier, the "normal" side is evaluated first, the "abnormal" side second.

The result of the ENoG is reported as a percentage. In essence, the amplitude of the normal side is the denominator and the amplitude of the weak side is the numerator. Therefore, if the weak side amplitude is 1240 microvolts and the normal side has an amplitude of 3265 micro-

volts, the response is reported as:

$$\frac{1240 \text{ microvolts (weak side)}}{3265 \text{ microvolts (normal side)}} = 0.379 = 38\% \text{ response.}$$

Alternatively, some clinics report the percentage in terms of “denervation,” indicating the percentage of neural fibers that are no longer responding. Therefore, the 38% response indicated above, would be considered a 62% “denervation.” Either protocol is fine, as long as the audiologist and the physician use consistent terminology.

Any response greater than 10% (or conversely, a 90% denervation) is consistent with a spontaneous recovery and may trigger the “watch and wait” response from the physician. However, a response of less than 10% may suggest a surgical alternative. It is a good clinical protocol to test the patient approximately every 3 to 5 days until a plateau (or direction) can be determined.

ILLUSTRATIVE CASE ONE

MRN is a 61-year-old male. He had been in the office previously for evaluation of his noise-induced, SN loss. His last office visit was 18 months ago. MRN presents today with right facial paralysis.

He states that after he woke up 4 days ago, he noted peculiar sensations while shaving, and found that he couldn't drink his coffee without “drooling all over the place.” His wife told him to go to the emergency room, as she thought he had had a stroke. He said he felt fine, assumed he had “slept on it funny,” and he went to work. That evening his wife noticed that when he was sleeping, his eyelid did not close all the way. The next morning, he went to his primary-care physician (PCP). The physician diagnosed “probable Bell's palsy” and referred MRN to the audiology department for additional work-up.

MRN presented to the audiologist with a grade six (6/6) HB facial weakness. The audiology work-up included the comprehensive audiometric evaluation and complete immittance battery. The audiogram was consistent with the test obtained 18 months ago (symmetric, 55-dB SN loss, excellent word-recognition scores), normal tympanograms AU, and normal ipsilateral reflexes on the left ear. Ipsilateral reflexes were absent in the right ear. With

Symptom	Problem(s)	Solution(s)
Poor response bilaterally	<ul style="list-style-type: none"> ❖ ineffective stimulus ❖ obesity ❖ bilateral dysfunction ❖ edema at stimulus site ❖ tenderness precludes proper stimulus electrode pressure (in ICU or OR) 	<ul style="list-style-type: none"> ❖ use needle electrodes ❖ compare patient's results to normative database ❖ relieve pain ❖ defer testing
No response bilaterally	<ul style="list-style-type: none"> ❖ chemical paralysis (in ICU or OR) ❖ improper electrode placement 	<ul style="list-style-type: none"> ❖ reverse neuromuscular blocking agent medically ❖ verify correct electrode placement and usage
Poor response unilaterally	<ul style="list-style-type: none"> ❖ inappropriate electrode site 	<ul style="list-style-type: none"> ❖ relocate stimulating electrodes ❖ increase stimulus intensity to supramaximal level
Short latency response	<ul style="list-style-type: none"> ❖ masseter muscle response 	<ul style="list-style-type: none"> ❖ move stimulating electrodes posteriorly
Excessive artifact rejection	<ul style="list-style-type: none"> ❖ stimulus artifact 	<ul style="list-style-type: none"> ❖ increase distance between stimulus and recording electrodes ❖ avoid crossing stimulus and electrode wires ❖ use post-stimulus time delay
	<ul style="list-style-type: none"> ❖ very large response 	<ul style="list-style-type: none"> ❖ decrease amplification (gain) and/or increase sensitivity limits

Table 2. *EnoG recording problems and solutions.*

the stimulus in the right ear, the acoustic reflex was recorded from the left ear (contralateral response). With the stimulus in the left ear, no response was obtained from the right ear. Tuning fork tests were consistent with the audiometric findings. A mid-line Weber response was obtained and air conduction was greater than bone conduction via the Rinne.

The above are classic findings consistent with Bell's Palsy. Therefore, the ENoG test was completed to obtain a “baseline” response.

The left-side response was obtained first. The maximal stimulus (40 mA) was required to obtain a “plateau” response. The response obtained from the left side was 2140 microvolts (µV). Using the same stimulation and recording parameters on the right side, the response obtained was 965 µV. The response was obtained and recorded from each side three times. Therefore, the right-side response was determined to be (965/2140) 45% of the normal side, also referred to as a “55% denervation.” The responses were consistent with neuropraxia and BP.

The audiologist instructed the patient to use artificial tears to keep the eye moist and lubricated and to protect the eye with glasses/sunglasses during all waking hours, since the normal “blink” response, which protects the eye from debris and insult, was not functioning. Additionally, the audiologist suggested that the eyelid be gently taped closed (with an adhesive tape)

during sleep to protect the eye. MRN was scheduled for the next ENoG 4 days later. An audiology report was sent to the PCP.

During the second office visit, MRN's facial paralysis was again determined to be a grade six. Although the audiogram was not repeated, acoustic reflexes were measured again. The left and the right ipsilateral responses were present, as were the contralateral responses. The second ENoG showed the left amplitude at 2310 µV, and the right side was determined to be 1405 µV. The response was determined to be 61% of the normal side, or 39% denervated. A follow-up (third) visit was scheduled 4 days later. An audiology report was sent to the PCP.

At the third visit, the audiologist noted that the facial presentation had improved to a grade three. Volitional eye closure had returned. The audiologist told the patient to discontinue the previously recommended eyecare protocols. Acoustic reflexes were again tested and were determined to be present via ipsilateral and contralateral pathways. The ENoG test revealed a left-side amplitude of 2275 µV. The right side had improved to 1885 µV. The response was determined to be 83% of the normal side. The patient was released from additional ENoG tests. A report was sent to the PCP. The patient was instructed to follow up with the PCP weekly until complete resolution of the BP had occurred, which it did about 2 weeks later.

ILLUSTRATIVE CASE TWO

The ENT service requested a bedside consult on patient TRW, who had been involved in a motor vehicle accident 7

neither the muscle relaxants or the edema interfered with the ENoG. The right response was not repeatable nor was it apparent (a.k.a. "flatline"). A report was

"...the ENoG test is the only relatively objective measure of facial nerve integrity..."

days earlier. TRW had been ejected through the windshield at a high rate of speed and had suffered multiple facial lacerations, a variety of broken bones (collarbone, ribs, pelvis) and a collapsed lung.

At the initial consult, TRW was conscious and able to respond to commands. Both sides of his face appeared puffy and swollen. His right blink reflex was absent. The left blink reflex was intact.

The audiologist and the ENT resident reviewed the chart. It was determined that the patient was on muscle relaxants and significant facial edema was present. Therefore, ENoG testing was deferred until 24 hours after muscle relaxants were discontinued and until the apparent swelling was reduced. A facial grading was deferred.

Three days later, the patient reported he could not hear out of his right ear. A bedside audiogram demonstrated a mild SN loss in the left ear; no responses were

sent to the ENT service.

A second ENoG 3 days later (13 days post-accident) demonstrated essentially the same response pattern. A CT scan showed a longitudinal fracture across the right temporal bone.

A combined middle fossa-mastoid facial nerve decompression was scheduled for the next day. Intraoperative monitoring of the facial nerve was done throughout the surgery. General anesthesia without muscle relaxants was employed. The otologist/surgeon determined that the fracture had severed the facial nerve at the geniculate ganglion. A microscopic re-anastomosis was obtained. Intraoperative monitoring revealed that when the distal segment of the nerve was stimulated, a reduced but repeatable response was obtained at the orbicularis oris, confirming physiologic facial nerve integrity.

The patient awoke with a grade six

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obtained in the right ear. Bedside screening tympanometry confirmed intact and mobile tympanic membranes AU (type A). The right ear was diagnosed with profound SN loss.

The left-side ENoG response was obtained. The test required a stimulus of 35 mA to obtain a plateau response of 2800 μ V. This response confirmed that

presentation. The facial nerve slowly regained limited function. At 8 months post-op, a grade four result was obtained. At 1 year post-op, a grade three result was obtained. The patient has maintained a grade three function for 2 years. This is not likely to improve.

CONCLUSION

ENoG is an extremely important test within the audiologist's scope of practice. We strongly advise those who intend to record ENoG clinically to gain experience under supervision prior to providing this service, and then to proceed cautiously and deliberately. Training for ENoG can be very lengthy, since individual offices may go weeks, months, or years between presenting patients.

Nonetheless, clinical training in this protocol should receive the same rigorous thought, study and practice as does intra-operative monitoring. **HJ**

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REFERENCES

1. Jackson CG, von Doersten PG: The facial nerve: Current trends in diagnosis, treatment and rehabilitation. *Med Clin North Am* 1999;83(1):179-195.
2. Beck DL, Benecke JE: Electroneurography: Electrical evaluation of the facial nerve. *JAAA* 1993;4:109-115.
3. American Speech-Language-Hearing Association. Ad Hoc Committee on Scope of Practice in Audiology: Scope of practice in audiology. See www.asha.org/library/scop_e_aud.htm (on line Dec. 2000).
4. American Academy of Audiology: Audiology: Scope of practice position statements. See www.audiology.org/professional/positions/scope.php (on line Dec. 2000).
5. de Bisschop G, Sarabian A, de Bisschop E, Sarabian N: Selection of electrophysiological investigations for diagnosis in idiopathic facial palsy: Twenty years experience in an ENT department. *Rev Laryngol Otol Rhinol (Bord)* 1998;119(2):75-85.
6. Rimpilainen I, Eskola H, Laippala P, Karma P: Prognostication of Bell's palsy using transcranial magnetic stimulation. *Acta Otolaryngol* 1997;Suppl 529:111-115.
7. Kimura M, Nakagawa I, Miimai H, et al.: Evaluation with blink reflex of bilateral facial palsy. *Masui* 2000;49(2):159-162.
8. House JW, Brackmann DE: Facial nerve grading system. *Otolaryngol Head Neck Surg* 1985;93:146-147.
9. Hughes GB, Josey AF, Glasscock ME, et al.: Clinical electroneurography: Statistical analysis of controlled measurements in twenty-two normal subjects. *Laryngoscope* 1981;91:1834-1846.
10. Beck DL: Facial nerve electrophysiology: Electroneurography and facial nerve monitoring. *Sem Hear* 1993;14(2):123-133.
11. Peiterwsen E: Bell's Palsy treated with Acyclovir (comment). *Ugeskr Laeger* 1997;159(14):2105.
12. Bendet E, Vajtai I, Maranta C, Fisch U: Rate and extent of early axonal degeneration of the human facial nerve. *Ann Otol Rhinol Laryngol* 1998;107(1):1-5.
13. Fisch U: Maximal nerve excitability testing vs. electroneurography. *Arch Otolaryngol* 1980;106:352-357.
14. Cai Z, Yu G, Ma D, et al.: Experimental studies on traumatic facial nerve injury. *J Laryngol Otol* 1998;112(3):243-247.