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Electrophysiological measures in facial paresis and paralysis

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Facial nerve injuries are debilitating events for patients. In many cases recovery is spontaneous and complete; in others prompt medical and/or surgical intervention will be necessary to improve the patient's outcome. Objective measurements are valuable tools that can help identify candidates for intervention. Electroneuronography and electromyography are the most commonly used objective measures of facial function. Basic examination techniques and recording parameters are discussed. A literature review indicated that, depending on the criteria used, Electroneuronography was 50-91% accurate (Positive Predictive Value or PPV) in identifying individuals requiring intervention and 80-100% accurate in predicting those who recover spontaneously (Negative Predictive Value or NPV). Electromyography can be used to assess both volitional movements (PPV 75-91% and NPV 62-89%) and for spontaneous activity (PPV 80-100% and NPV 92-96%). Attention is paid to how grading criteria and the timing of the examinations impacts the accuracy of both Electroneuronography and Electromyography. Specific protocols are suggested for patients with facial nerve injury.

The onset of a facial weakness can be a horrifying and potentially debilitating event for many patients. Facial nerve paresis and paralysis have been shown to have a significant impact on a patient's quality of life.[1-4] Facial paresis or paralysis can result from several different conditions. The most common form of facial weakness is idiopathic, commonly referred to as Bell palsy. However, facial weakness can also result from trauma, iatrogenic injury, congenital craniofacial abnormalities, or from neoplasms, such as a vestibular schwannoma or a facial nerve neuroma. Treatment of acquired facial nerve paresis or paralysis varies and includes observation, medical management, and surgical intervention. Surgical options include palliative measures to protect the eye, facial nerve decompression, and various facial nerve reanimation procedures. One of the primary goals of any objective measure evaluating facial nerve function is to differentiate those patients who will spontaneously recover to an acceptable degree from those who could benefit from a more aggressive surgical intervention. Electroneuronography (ENOG) and electromyography (EMG) are the objective tests of facial function most useful in determining the prognosis of a facial nerve injury (FNI) and in guiding treatment. They are both electrophysiologic measures that indirectly quantify facial nerve function by recording motor unit action potentials (MUAPs) and/or compound muscle action potentials (CMAPs).

The purpose of this discussion is to describe how to perform and interpret EMG and ENOG. This information will help the novice examiner gain knowledge, and expand the diagnostic repertoire of experienced examiners. Finally, attention is made to the application of EMG and ENOG in medical decision-making protocols.

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Nerve injury classification

Understanding the basic fundamentals of peripheral nerve injury is critical to the correct implementation and interpre-

tation of electrophysiological facial nerve studies. Seddon[5] initially classified peripheral nerve injury into 3 types: (1) neuropraxia, (2) axonotmesis, and (3) neurotmesis. Sunderland[6] expanded on Seddon's work by further subclassifying the neurotmesis category, which resulted in 5 distinct degrees of nerve injury. Table 1 displays the theoretical characteristics of each type of nerve injury along with a range of possible outcomes from EMG and ENOG studies.

To fully understand nerve injury classification, it is helpful to review the basic anatomical structure of nerve fibers. Each nerve fiber consists of an axon that contains cytoplasm, which is aptly named axoplasm. It is the axoplasm that serves to physically conduct electrical impulses along the axon. The axon itself is surrounded by a myelin sheath made of Schwann cells (in peripheral nerves) and other connective tissues. This sheath is often referred to in the literature as the "endoneurium," "neural tubule," or "endoneural tube." Several axonal sheaths are then bundled and held together by other connective tissues (called the perineurium) to form the funiculus or fascicle. Finally, several fascicles are held together by areolar connective tissue, which becomes more compressed outside the temporal bone and is called the epineurium. See Table 2 and Figure 1 for an overview of the contents of a nerve fiber. The epineurium, perineurium, and endoneurium can be viewed as 3 "protective layers" of connective tissue that serve to shelter the axon.[9] Sunderland's 5 degrees of nerve injury are based on the extent of damage to various functional anatomical components and layers of the nerve. A summary of nerve injury classification based on the works of Seddon[5] and Sunderland[6] is given here as follows:

- First-degree injury (neuropraxia): first-degree injuries are the result of a conduction block, which is typically secondary to nerve compression or ischemia. In this category, injury may result in some form of localized damage to the myelin sheath. However, there is no axonal degeneration so that the axonoplasmic continuity remains intact distal to the lesion. Therefore, neuropraxic injuries will continue to conduct a neural impulse if an electrical stimulus is delivered at a point distal to the site of lesion. This is the basic premise on which ENOG testing is based. Recovery from this type of injury is spontaneous and complete.
- Second-degree injury (axonotmesis): in this degree of injury, there is a complete interruption of both the axon and the axoplasm contained within the axon. However, there is preservation of the endoneural tubule. Anterograde axonal degeneration (called Wallerian degeneration) occurs; this results in the peripheral end organ being isolated from its corresponding neuron. This loss of nerve supply to the end organ is referred to as denervation. Because the endoneurium remains intact, the axon can regenerate toward its original end organ through the intact tubule. This leads to a better prognosis in terms of recovering motor function following the injury.
- Third-degree injury (neurotmesis): this type of injury involves damage to the endoneural tube and its contents. Retrograde disturbances are more significant because regeneration can now occur across disrupted endoneural

Table 1 Nerve injury characteristics

Category	Degree of injury				
Seddon's classification[5]	Neurotmesis	Axonotmesis	Neuropraxia		
Sunderland's classification[6]	First-degree conduction block	Second-degree axonal continuity	Third-degree endoneural tubule	Fourth-degree funiculus	Fifth-degree complete nerve trunk
Recovery[6]	Complete <2 wks	Complete or mild	Residual deficit	Some spontaneous recovery that is rarely useful	If untreated rare, and residual deficit if treated
Wallerian degeneration[5,6,7,8]	Does not occur	6-21 d, often >14 d	6-21 d, often <14 d		100% by 3-5 d[a]
ENOG [7-18]	Normal	There is a >50% chance of an incomplete recovery when the ENOG response is reduced by ≥90%			100% denervation by 3-5 d[a]
EMG (resting)[17-21,26]	Absent pathological spontaneous activity	Pathologic spontaneous activity is present after 14-21 d, which heralds a high probability (80%+) of an incomplete recovery			
EMG (volitional)[17-22,26]	Volitional responses can be intact, reduced or, absent. Early intact volitional responses at multiple sites or improvement in the firing pattern suggest a good outcome. Absent (or minimal) volitional predicts a poor recovery				No volitional activity[a]

Lays out the theoretic framework of the different degrees of nerve injury and the range of responses from ENOG and EMG that might be expected from each degree of injury. Note that ENOG and EMG responses cannot definitively differentiate 2 to 5 degree injury. Data from the table are compiled from a number of sources.

ENOG, Electroneuronography; EMG, electromyography.

[a]A 5th degree injury will always result in a no response ENOG/EMG. An absent response does not always indicate a type 5 injury.

Table 2 Contents of a nerve fiber

Nerve axon	Extension of the neuron that contains axoplasm and is responsible for transduction of electrical impulses away from the cell body.
Endoneurium	Contains the axon. It is a single layer of Schwann cells and an inner layer of connective tissue called neurilemma. It serves as a protective sheath for myelinated nerve cells.
Perineurium	Connective tissue that bundles together a number of endoneural tubes to form the funiculus.
Epineurium	Funiculus bundles are held together by areolar connective tissue, which is more compressed at the surface. The bundles gradually become intermingled as fibers ascend along the nerve.

tubules. Axons may reach functionally related end organs or they may enter totally foreign endoneurial tubes. This results in internal disorganization because some axons do not regenerate to their original end organs. The resulting abnormal healing creates a distorted and less efficient firing pattern, with the clinical manifestations of synkinesis and/or contracture. Synkinesis is the involuntary contraction of an erroneously reinnervated muscle during a contraction of the normally reinnervated muscle.[9] For example, a patient's eye may blink when he attempts to smile. The overall recovery is longer and usually incomplete in third-degree injuries.

- Fourth-degree injury (neurotmesis): fourth-degree injuries involve damage to the funiculus and its contents. The entire funiculus is involved, and all bundles are breached. Funicular bundles become so disorganized that they are no longer distinguishable from the surrounding connective tissues of the epineurium. Large numbers of regenerating axons escape and infiltrate foreign tubes. Some spontaneous recovery can occur, but it is of little functional value.
- Fifth-degree injury (neurotmesis, complete nerve transection): a fifth-degree injury involves transection of the entire nerve trunk. The majority of axons do not reach their designated funiculi or endoneural tubule because of the separation of the nerve ends and scarring. Recovery will not occur without surgical intervention, and complete restoration of function is impossible even if the nerve ends are repaired.
- Mixed injuries: it is entirely possible that the degree of injury can be unevenly distributed across a nerve fiber. This results in different axons, endoneural tubes, and funiculi being at various stages of injury at the same time. Fifth-degree injuries by definition cannot coexist with lesser degree injures. It is possible to have a partial transection, although injuries severe enough to partially transect a nerve will most likely leave a significant injury in any nontransected fibers. Mixed injures can muddy the diagnostic waters and make it difficult for any objective measure to make a clear and definitive differentiation regarding the specific degree of injury.

Electroneuronography

The ENOG examination was originally proposed and popularized by Esslen[10,11] and Fisch[7,12,13] in late 1970s. Evoked EMG or facial nerve conduction evaluation are more accurate names as the procedure involves recording

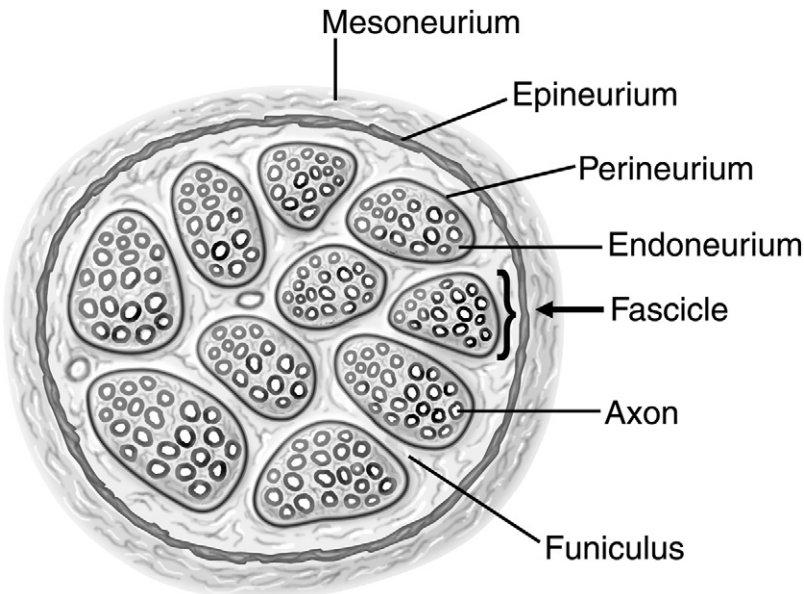

Figure 1 "Cross Section of a nerve fiber." The diagram depicts the 3 "protective layers" of connective tissue (endoneurium, perineurium, and epineurium), several axonal bundles (fascicle), and the axons themselves. The axons contain axoplasm, which serves to conduct the action potential.

Figure 2 "Abnormal ENOG." Electroneuronography responses 1 month postinjury from patient with bilateral temporal bone fracture. Patient presents with a grade VI weakness on the right and grade II weakness on the left. Responses demonstrated a large compound muscle action potential response from the left (top 2 responses) and no response (100% degeneration) on the right (bottom 2 responses).

the CMAP of the mimetic muscles from a proximally delivered electrical stimulus. The goal of the testing is to measure the amount of neural degradation that has occurred distal to the site of nerve injury by measuring the muscle response to an electrical stimulus.[8] The amount of denervation is represented by comparison of the peak-to-peak amplitude of the CMAP from the affected side with the response amplitude from the nonaffected side. Figure 2 depicts a 100% degenerated response from the right side and normal CMAP from the left side. The premise is that a nerve will no longer be able to propagate an electrical simulation distal to the site of injury once it has undergone anterograde axonal degeneration (ie, Wallerian degeneration). In effect, ENOG differentiates first-degree "conduction block" injuries (neuropraxia) from those that have developed Wallerian degeneration (second-fifth degree).

The CMAP is a result of the near-simultaneous firing of motor units in the same area. This results when the electrical stimulus depolarizes the distal portion of the facial nerve trunk, which causes the distal motor units to fire in sync.[9] The simultaneous response is recorded as single multipeaked response. Figure 3 diagrams show several near-simultaneous MUAPs being summated into a compound response. Another important factor to consider is that recording a CMAP from the surface of the skin requires a synchronous discharge of all "stimulable" fibers.[8] Neural synchrony is relevant when evaluating patients with facial nerve neuromas, which are in a state of constant simultaneous nerve injury and regeneration. This creates dyssynchrony, where the potentials from 2 axons occur out of phase, canceling their responses. The result is a greater than anticipated decline of the ENOG amplitude. Disproportionately, poor responses can also be witnessed during neural regeneration where the firing patterns of various axons are in different phases of regeneration. This can sometimes result in discrepancies between the ENOG and EMG responses; the implications for interpreting these discrepant results are discussed in the "Interpretation of EMG" section later in the text.

Performing ENOG

Stimulation and recording electrodes are typically saline-saturated felt pads mounted in a fixed-distance bipolar electrode receptacle. Alternatively, an angled tip stimulator can be used for stimulation. Conducting gel is applied to the probe tip in the latter case. The stimulation and recording sites are cleaned with standard preparation material, and the recording sites are confirmed to have impedance measurements $<5000\ \Omega$. A ground electrode is placed on the patient's forehead. The anode and cathode of the stimulating pair are positioned on the surface of the skin along the main trunk of the facial nerve as it exits the stylomastoid foramen; the anode electrode is typically placed proximally. The recording electrode is lined up with the nasolabial fold. Current pulses with a

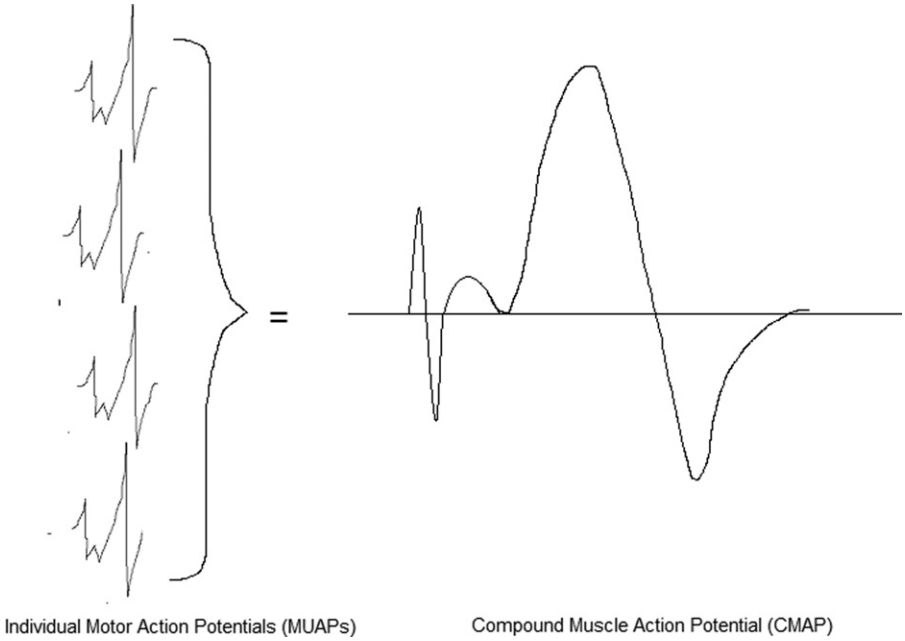

Figure 3 "Compound muscle action potential." This diagram depicts near-simultaneously firing multiple motor unit action potentials (MUAPs). This occurs when an electrical stimulus depolarized the entire nerve trunk. The resultant muscle contraction is recorded as a single biphasic compound response known as the compound muscle action potential (CMAP).

0.1-ms duration are gradually increased in a stepwise manner from 0 milliamperes (mA) until the CMAP reaches its maximum amplitude. Next, another 10% of the current mA is added to the stimulus level to obtain a supramaximal stimulation level. This is a real-time response and does not require signal averaging. In fact, averaging could obscure amplitude differences between an affected and normal side. A maximum of 30 mA is set as the absolute stimulation limit. See Table 3 for ENOG equipment settings. The alignment of the electrodes should be adjusted to obtain maximum responses. Stimulation 10 to 20 times before recording responses helps to reduce impedance and synchronize the nerve responses.[14] The peak-to-peak amplitude of the CMAP is measured on the affected side and compared with the response from the nonaffected side. Coker concluded that the side-to-side technique is most consistent when recorded from the midface.[15] This study also provided data to support for Esslen's original hypothesis that the absolute amplitude of the CMAP corresponds to the number of firing motor neurons. Thus, the percentage of denervated neurons can be estimated via ENOG.

Table 3 ENOG settings

	Recording parameters
High pass filter	20 Hz
Low pass filter	1000 Hz
Sensitivity	1 mV
	ENOG stimulator setup
Stimulus range	0-30 mA
Duration	0.1 ms
Pulse	(1) A single triggered pulse or (2) Continuous pulse train at a frequency of 3 Hz

ENOG simulation and recordings parameters used at the University of Michigan Medical Center. A grounding surface electrode is also necessary.

Interpretation of ENOG

ENOG's primary utility is in determining the long-term prognosis of facial function. Patients with ≥90% degeneration on the affected side (when compared with the nonaffected side) are considered to have a positive test. Some authors have suggested using a 75% degeneration criterion.[16,17,23] May et al[16] recommend using a 75% criterion, but their data also reflected that those who did degenerate by ≥90% were more likely to develop an incomplete recovery. Fisch[7] concluded that including patients who denervated <90% would excessively elevate the rate of unnecessary surgical procedures. Patients who demonstrate facial paresis in the setting of a normal ENOG study (ie, they have no signs of Wallerian degeneration) are likely to have a conduction block, and therefore, they will have a satisfactory recovery. ENOG cannot precisely differentiate second- through fifth-degree injuries; however, the timing of the onset of the Wallerian degeneration can provide additional information that can help to clarify the severity of the injury. Fisch[7] demonstrated that the velocity of complete denervation is proportional to the severity of injury. Wallerian degeneration was complete by 3 to 5 days after complete nerve transaction, whereas with axonotemsis (second-degree injury), the process took 14 to 21 days. One can interpolate that third- and fourth-degree injuries require

somewhere around 5 to 14 days range to completely degenerate.

Prognostic value of ENOG

One of the primary goals of ENOG testing is to identify patients likely to have a poor outcome without some type of intervention. May et al[16] found that ENOG was 88% accurate in predicting incomplete recovery when denervation was 75% or more. The positive predictive value (PPV) increased to 91% when a 90% reduction criterion was used. It is noteworthy that this article predates the publication of the House–Brackmann[17] (HB) facial nerve grading scale, making it difficult to compare with more contemporary studies. Grosheva et al[18] found that a ≥75% degeneration criterion predicted a poor facial nerve outcome (HB grade II or worse) in 59% of patients. Sillman et al[19] used a ≥90% degeneration criterion and demonstrated a 50% chance of developing an unacceptable outcome. Here, a poor recovery was defined as returning to HB grade III or poorer. In the Sillman et al[19] study, subjects were further subdivided into idiopathic and traumatic groups. For the idiopathic subset, ENOG demonstrated a PPV of 56%. ENOG was a less reliable predictor in the traumatic injury group. In this small subset, 6 out of the nine subjects with positive findings on ENOG went on to recover to HB grade I or II. This resulted in a PPV of only 33% for traumatic injury patients. Although it is difficult to directly compare individual study outcomes because of differences in diagnostic criteria, patient populations, and facial weakness grading systems, the overall range of reported PPV for ENOG testing was 50% to 91%.

Other authors have used ENOG to predict which FNI patients will have a good outcome. The facial function grading systems and the amount of residual function for a "good outcome" have varied across studies. Gantz et al[8] and Sillman et al[19] demonstrated that 100% of patients who had negative ENOGs returned to HB grade I or II. Furthermore, 89% of the negative ENOG subset fully recovered to a HB grade I. This study used ≥90% degeneration as the criteria for a positive ENOG. Fisch[7] reported good recovery in 80% to 100% of patients using ≤90% denervation as the criterion for a negative study; this study predates standardization on HB scale, so it is difficult to compare results. Using a ≤70% denervation criterion for a negative test, May et al[16] found that 84% of patients developed a complete recovery. Grosheva et al[18] found that 80% of patients returned to HB grade I (using a ≤75% degeneration as criteria for a negative test). It is noteworthy that 39 of 67 (58%) of patients in this study who had a "poor recovery" actually improved to a HB grade II. Many authors would argue that recovering to HB grade II would not warrant surgical intervention, and therefore, the negative predictive value (NPV) in this study would have been higher if a more lax (but reasonable) criteria for a "good outcome" had been used.

In summary, ENOG is a quick and relatively painless procedure. It has been demonstrated to be highly effective in identifying patients who are likely to recover to an acceptable degree from a facial nerve paralysis. There is also some evidence that ENOG can identify patients who will benefit from facial nerve decompression, which is discussed in greater detail later in the text.

Electromyography

EMG is the recording of MUAPs. MUAPs are the spikes in electrical activity generated when a motor unit fires. A motor unit consists of a motor neuron and the corresponding muscle fibers innervated by the neuron. There is an excellent description of motor units and MUAPs in Kileny et al.[9] Each motor unit consists of the neuron and its axon, which have multiple synaptic junctions that are affiliated with corresponding muscle fibers. These synaptic junctions are called myoneural junctions. Each myoneural junction and muscle fiber generate a small electrical potential when activated. The synchronized discharge arising from all of the axon's myoneural junction potentials combines to form the lager MUAP.

Performing EMG

EMG is recorded via a bipolar recording paradigm using a differential amplifier. Bipolar EMG recordings can be performed using several different recording needle types and methods. Recording can be done using: (1) a pair of monopolar needle electrodes, 2) a monopolar needle electrode and a reference surface electrode, or (3) concentric needles. The latter is the preferred method for recording facial EMG. Concentric needles were designed by Adrian and Bronk[24] in the 1920s. A thin recording electrode wire is positioned in the center of a small hypodermic-style needle. The exposed end of the electrode wire projects into the orifice of the needle and forms one electrode. The hypodermic needle shaft functions as the second electrode. This allows for recording from specific locations. Concentric needles offer the advantage of a single needle stick into the sensitive facial musculature. Given the nature of differential amplifiers, the likelihood of recording unintended EMG from adjacent muscle fibers is less when recording sites are close together. Howard et al[25] demonstrated that that MUAP amplitude, rise rate, and number of turns (ie, the number of times the waveform changes its direction) were significantly larger when using monopolar electrodes. There were no significant differences between monopolar and concentric needles in their overall response durations and/or firing rates. Both monopolar and concentric needle paradigms are adequate to detect target responses. The latter is more specific because it reduces the chances of recording errant EMG activity from unintended nearby muscles, such as the masticators.

Recordings are made from various mimetic muscles that are innervated by the peripheral motor branches of the facial nerve. See Table 4 and Figure 4 for an overview of recording sites; Table 5 covers standard equipment settings for EMG recordings. Electrical activity at each test location is

Table 4 EMG recording sites

Facial nerve branch	Recording location
Temporal branch	Frontalis muscle
Zygomatic branch	Orbicularis oculi
Buccal branch	(1) Nasolabial fold (levator labi alaque nasi/levator labii superioris) (2) Orbicularis oris
Marginal mandibular branch	Chin: (mentalis/depressor anguli oris/ depressor labi inferioris)

The 4 branches of the facial nerve responsible for facial expression and the recommended muscle locations for inserting the EMG recording electrode.

recorded both at rest and during attempted volitional movement. When the patient's face is at rest, the examiner is observing for the presence of abnormal resting potentials. These can consist of either spontaneous fibrillation activity or positive sharp waves. Positive sharp waves are involuntary potentials generated by a single muscle fiber. These responses are also sometimes referred to as unstable resting membrane potentials, abnormal spontaneous activity, or abnormal resting potentials. Figure 5 contains several images of abnormal resting potentials along with a depiction of some reduced volitional EMG. Fibrillation potentials characteristically have a polarity that is inverse to the normal motor potential.[9] Abnormal resting potentials are a sign of neural degeneration and appear as early as 7 days[20] but more frequently after 10 to 14 days postinjury.[18,23] According to Mills[20], acutely denervated muscle fibers develop acetylcholine receptors over the whole muscle fiber membrane rather than just at the neuromuscular junction (the normal state). This makes the nerve fibers extrasensitive and more likely to discharge spontaneously, resulting in fibrillations. Positive sharp waves have the same origin and significance as spontaneous fibrillations but are related to the EMG needle being inserted into a muscle fiber.

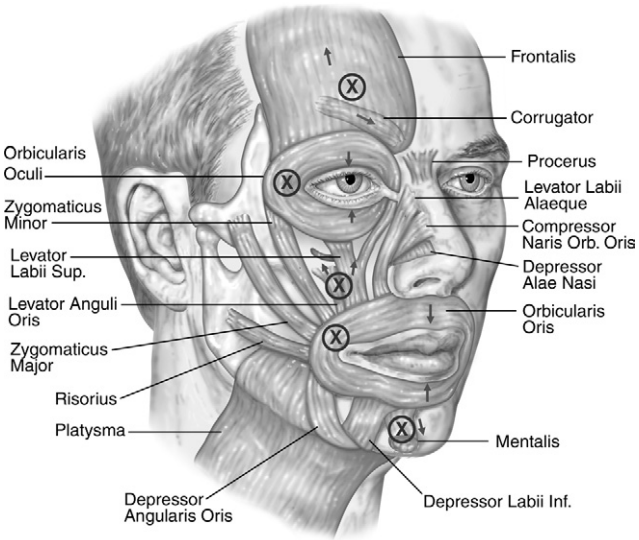

Figure 4 "EMG Recording Sites." Concentric needle recordings are made from the mimetic muscles innervated by the 4 motor branches of the facial nerve that contribute to facial expression. Recording sites are marked with an X. From top to bottom the concentric needle is inserted into the frontalis muscle in the forehead, in the orbicularis oculi near the lateral canthus of the eye, in the perioral muscles near the nasolabial fold and orbicularis oris, and finally into the mentalis muscle in the chin.

During the volitional EMG examination, the patient is asked to attempt to fully contract the muscle being recorded. During attempted volitional motion the examiner is observing for the presence or absence of any voluntary motor unit potentials. Normal volitional motor units will have between 2 and 4 phases. The normal MUAP starts with a relatively low amplitude–positive component that is followed by a major negative phase.[9] Any volitional motor unit with 5 or more phases, which are typically asynchronous, is considered polyphasic. This is associated with reinnervation and is attributed to randomized axonal regrowth. Most EMG equipment allows for the capture of patient responses; this allows the examiner to adjust the time base to zoom in and analyze individual MUAPs. The examiner also needs to assess if there is a normal recruitment pattern or not. In a normal EMG response, the frequency and amplitude of the motor unit potentials should be "proportional" to the amount of effort made to contract the muscle. During stronger contractions in normal patients, the motor unit potentials can start to overlap and interfere with each other, resulting in a random appearance. This is referred to as a normal recruitment or interference pattern.

Interpretation of EMG

If a patient is able to elicit volitional EMG, a complete nerve transection is effectively ruled out. Therefore, it is possible to use EMG to rule out a fifth-degree injury immediately after the onset of injury. It is also possible to record volitional EMG in the presence of a completely absent ENOG response. Fisch[12] initially described this phenomenon, which he called "early deblocking," and speculated that this occurs when the injured nerve fibers fire in a desynchronized pattern, which effectively cancels out the ENOG response. The muscle fibers respond randomly rather than with the single unified response that is necessary

Table 5 EMG equipment settings

Recording parameters	Setting
Recording window	2 s
High pass filter	20 Hz
Low pass filter	3000 Hz.
Sensitivity	100 μV
Electrode type	Concentric needle (preferred), monopolar electrodes can be used
Recording electrode	A disposable 25-mm 30-ga concentric EMG electrode

Listed in Table 5 are the standard recording parameters for a facial nerve EMG evaluation. A grounding surface electrode is necessary.

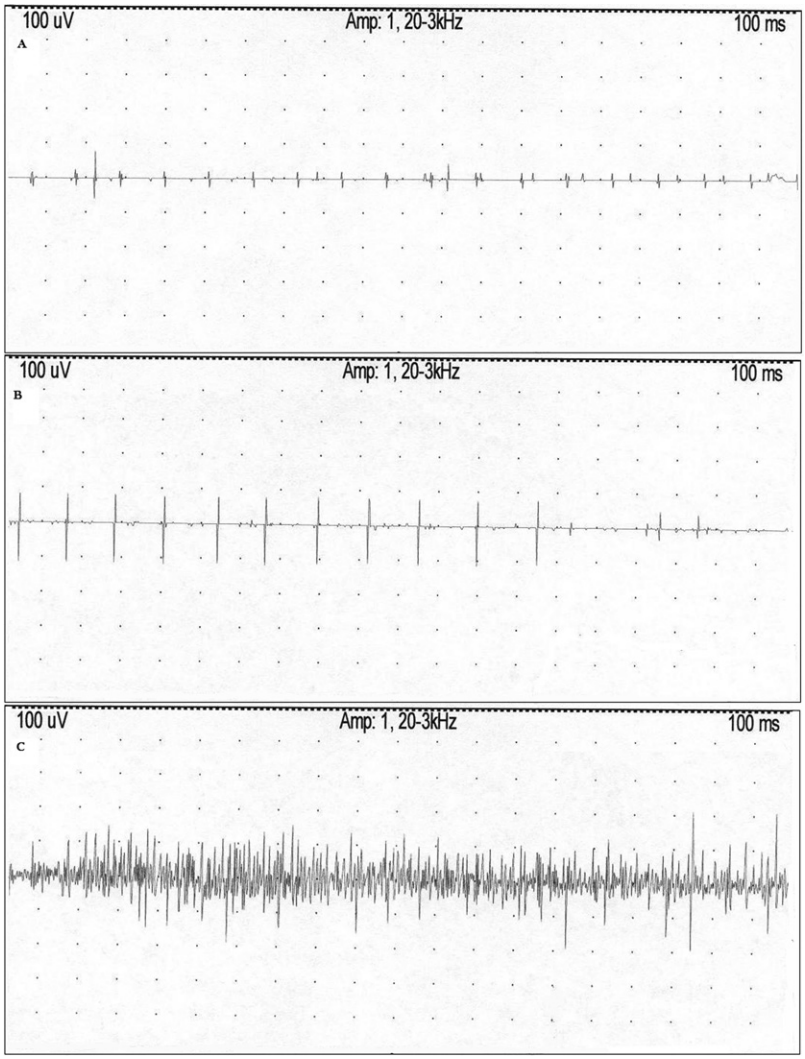

Figure 5 "Motor Unit Action Potentials": (A) Fibrillation potentials recorded from the temporalis branch (frontalis muscle), (B) spontaneous responses from the buccal branch (nasolabial fold), (C) volitional electromyography recorded from the marginal mandibular branch (mentalis muscle). All responses were measured from the right face of a 20-year-old man with a history of transverse temporal bone fractures secondary to a 4-story fall. Responses are recorded 1 month postinjury.

to register a CMAP. This pattern can also be seen in chronic conditions such as a facial nerve neuroma, where the facial nerve is in a constant state of injury and regeneration. Sillman et al[19] described 8 patients with signs of early deblocking. All patients had ENOG responses that degenerated to ≥90% within 14 days after onset of the injury. All 8 patients demonstrated volitional EMG (recorded from periorbital and perioral mimetic muscles) and returned to HB grade I or II. It is also important to consider the number of sites where volitional EMG remains intact, as this can impact the patient's chances of recovery. Granger[26] demonstrated that patients with voluntary potentials in 4 to 5 muscles 72 hours after paralysis onset have a 91% chance of a favorable outcome. If voluntary potentials were intact at 2 to 3 recording sites, the probability of a favorable outcome remained relatively high at 87%. If EMG was only intact at 0 to 1 site(s), a satisfactory outcome occurred in only 11% of patients. Granger[22,26] used his own facial nerve grading system and classified a satisfactory outcome as having 75% motor function; this was described as having full range of motion and moderate strength against manual resistance. This involved averaging the response from for upper and midface muscles. Wiet et al presented data where they performed a retrospective analysis of EMG studies and compared the results with long-term facial function, which was assessed via patient questionnaires. They used an EMG index that compares the number sites without volitional potentials with the number of sites tested (ie, a low index score is better than a high index score). Although no correlation was found between the EMG index and long-term HB grade, they did find that the presence or absence of volitional EMG from the periocular region was statistically significant for predicting facial nerve outcome. The forehead response was the next most important recording site (Wiet et al, unpublished data, May 2008).

Several authors have recommended using the presence or absence of pathological spontaneous activity (fibrillation potentials and/or positive sharp waves) with a goal of separating out those patients who will recover spontaneously versus those patients who require further intervention.[18,21,23] Under this

Table 6 Timing considerations for ENOG and EMG

Onset of injury	If the face is immediately weak at the time of trauma, this is concerning for severe injury. Later onset is more favorable than early onset.
0-3 d	ENOG will always be normal (unless injury is distal to point of stimulation). Presence of any volitional activity on EMG can rule out a complete nerve transection. Volitional responses at multiple sites are a positive sign.
3-5 d	Evidence of Wallerian degeneration (via ENOG) in this early stage after injury is concerning for a possible fifth-degree injury (complete transection).
6-14 d	Evidence of onset of Wallerian degeneration (via ENOG) in this time frame is suggestive of a grade 3-4 injury. The cut off for surgical decompression in idiopathic facial weakness is of 12-14 d.
14-21 d	Evidence of later onset Wallerian degeneration on ENOG suggest 2nd degree injury. EMG can now be evaluated for presence of abnormal spontaneous activity (which suggests a second-degree or worse injury and a less satisfactory outcome).
8-24 mon	EMG can be used to monitor for improved volitional responses and to help determine the patient's candidacy for a dynamic facial reanimation procedure.

The utility and implications of ENOG and EMG studies in terms of the amount of time that has lapsed between the onset of injury and the time of the evaluation.

system, a first-degree injury is defined as decreased or absent EMG without pathological spontaneous activity after 10 to 14 days. Inversely, the presence of pathological spontaneous activity along with decreased or absent volitional activity is categorized as a second- or higher-degree injury and thus predicts a poor outcome. These criteria also allow for mixed injuries. In mixed injures, there are signs of a first-degree injury at some recording sites and more severe injuries at other sites. Mixed injures are considered to be a predictor of a poor recovery. Outcomes using these criteria are discussed in the "Prognostic Value" of EMG section later. Grosheva et al[21] recommended taking multiple recordings from each recording site; this is critical to thoroughly evaluate for pathological spontaneous activity. It is important to understand that this classification system is not valid until after 10 to 14 days when the abnormal spontaneous responses will appear. This means that evaluating spontaneous activity does not add to the clinical picture during the acute stage of the injury. This issue is discussed in greater detail in the "Timing considerations" section.

Prognostic value of EMG

It has already been demonstrated that ENOG testing provides valuable prognostic information. EMG testing can certainly be an intimidating procedure as most people do not relish the idea of being stuck in the face with needles. It is essential to understand how EMG testing can add to the clinical picture to justify performing the additional test to both patients and insurers. May et al[16] found assessing for volitional EMG to be 75% accurate in predicting a poor outcome and 62% accurate in predicting a favorable outcome. All evaluations occurred within 10 days of onset of injury, and the criterion for predicting a good outcome was intact volitional EMG at ≥4 recording sites. This stringent criterion likely contributed to the lower predictive values that were reported. Furthermore, this study predated the HB grading system and thus cannot be directly compared with later studies. Grosheva et al[18] and Sittel and Stennert[23] both provided prognostic values using the protocol described in the preceding section (in which presence of abnormal spontaneous activity is used to differentiate first-degree injuries from more severe injures). Patients were classified into the "good outcome" group if they had a complete return of facial function (HB grade I). Grosheva et al[18] provided data for both an initial (<14 days) and a follow-up EMG obtained at least 15 days after the onset of injury. EMG examinations performed <14 days of onset had a PPV of 91% and a NPV of 89%. Both the NPV and PPV improved to 96% and 100% on the follow-up EMG studies. Sittel and Stennert[23] provided analysis of EMG evaluations 10 to 14 days after injury. They found a PPV of 80.8% and a NPV of 92.4%. Again, it is noteworthy to recall that these studies classified any patient who resulted in a HB grade II into the "bad outcome" group. Therefore, it is likely that the prognostic values would have been better if the HB II group were reclassified with the HB I group into a group of patients who had "acceptable outcomes." These data clearly show the utility of using EMG for facial nerve injures. The high HPV and high NPV of EMG studies performed 2 weeks after the onset of injury suggest that EMG may be the study of choice for patients who present to the clinic several weeks after the onset of their injury as well as those patients who are undergoing long-term monitoring.

Timing considerations for ENOG and EMG evaluations

When performing ENOG and EMG, it is important to consider how much time has elapsed since the onset of the injury. The reader is referred to Table 6, which suggests a timeline for EMG and ENOG evaluations. Key issues related to timing of the evaluation include:

1. ENOG should never be performed before 3 days postinjury. Abnormal ENOG responses rely on the onset of Wallerian degeneration, which requires a minimum of 72 hours to occur.

2. Early signs of denervation on ENOG are a poor prognostic sign because they herald a more severe nerve injury.
3. EMG can be performed at any time after the injury. Early responses, especially at multiple locations, are a positive indicator for recovery and help to rule out a complete nerve transection.
4. EMG's prognostic value improves when testing can be deferred until 10 to 14 days after the onset of injury. This will allow signs of abnormal healing/abnormal resting potentials to occur. However, at least in the case where a facial nerve decompression is being considered, delaying treatment may be detrimental to the patient's ability to recover.

Protocols

As we have previously discussed, ENOG and EMG can be used to evaluate facial weakness caused by a variety of disorders. Each group of patients has their own unique presentation and management needs that can impact timing, implementation, and interpretation of electrophysiological testing. Some of the unique features of each of these disorders are discussed as follows:

Chronic conditions

It has already been discussed that patients with a facial nerve neuroma may demonstrate poorer than expected ENOG responses when compared with the patient's observed facial function. This is because of disrupted neural synchrony. Other large cerebellopontine angle (CPA) lesions, such as a vestibular schwannoma or meningioma, also can produce facial nerve weakness. ENOG can be useful in the preoperative evaluation of patients with CPA lesions when there is concern that the tumor could be a facial nerve neuroma.

Idiopathic facial weakness

For patients with idiopathic facial weakness, our clinic follows the protocol outlined by Gantz et al[8] in their seminal article on facial nerve decompression in Bell palsy. This protocol dictates that patients who have ≥90% degeneration on ENOG and demonstrate no volitional activity on EMG undergo immediate surgical decompression of the facial nerve. All patients who do not meet surgical criteria are managed medically. The Gantz et al data demonstrated that patients who met surgical criteria but elected to defer decompression had a 58% chance of maintaining a HB grade III or, worse, facial weakness. Inversely, 100% of patients not meeting surgical criteria returned to HB grade I or II. Their data demonstrated that the probability of a good outcome for the surgical candidate group was tied to the time of intervention. The chance of a good outcome was 93% if the operation occurred by day 12 and was 82% by day 14. Patients who underwent surgery after 14 days did no better than the nonsurgical controls.

ENOG and EMG (when ENOG is abnormal) should be tested early in the acute phase (day 3) and then monitored during recovery (day 6, then 10-12), electing for surgical intervention when ENOG degenerates by ≥90% and no volitional EMG is recorded (or volitional activity is present at only 1 recording site). It is not advisable to delay decision making regarding surgical decompression until after day 14 when EMG can provide additional prognostic information. It is not likely a coincidence that the 12 to 14 days "deadline" for benefit from surgical decompression corresponds to the same time frame in which abnormal spontaneous resting potentials appear on EMG. It may be that the detection of abnormal resting responses is a harbinger that the window for surgical decompression has passed.

Despite the best efforts, there will be some patients who do not recover acceptable facial function. There will also be some patients who present outside the window of time for facial nerve decompression or for whom facial nerve decompression does not work. In these cases, EMG can provide further guidance. If there are little to no EMG potentials at 8 to 10 months postinjury, some type of dynamic surgery should be considered. Options include nerve substitution, temporalis tendon transposition, or free muscle transfer.

Trauma

As noted previously, Sillman et al[19] reported that ENOG correctly identified unsatisfactory outcomes in only 33% of trauma patients, albeit with a small number (9) of patients. In this population, EMG may be the more valuable tool. Often patients with FNI secondary to trauma are not referred for evaluation until several weeks after the onset of their injury and thus will be tested at the time when the EMG is most accurate. There are several reasons for delayed referrals, but the primary reason is that many patients have immediate life-threatening injuries or are incapable of undergoing the examination. Despite this, having an incapacitated or uncooperative patient does not necessarily preclude an evaluation as is illustrated in the following case study:

Case study

Patient "AD" was a 30-year-old woman who was a passenger in a small car that collided with a semitruck. A head computed tomography scan revealed a longitudinal fracture through the right temporal bone, with dislocation of the malleus from the incus. The trajectory of the fracture line extended toward the geniculate ganglion. Computed tomography also demonstrated an encephalocele on the right side associated with a tegmen tympani fracture. An audiogram demonstrated a moderate to profound mixed hearing loss on the right side. On physical examination, she demonstrated a grade VI right facial weakness on the HB scale.

Table 7 Good versus bad outcome indicators

Good outcome indicators	Bad outcome indicators
Normal ENOG after 14-21 d	Weakness at time of trauma
Late onset of Wallerian degeneration	Early onset Wallerian degeneration
Normal resting potentials on EMG after 14-21 d	Progressive decay of ENOG ≥90% amplitude reduction
Volitional EMG and a normal interference pattern	Abnormal spontaneous activity on EMG after 10-21 d
Improving volitional EMG	No volitional EMG or EMG at only 1 recording site
Volitional EMG recorded at 2+ sites (4-5 better)	

The initial plan was to perform a standard ENOG and EMG evaluation on this patient in the clinic. However, she was found to have an extreme fear of needles and was completely unwilling to participate in the facial nerve examination. We developed a plan to perform a "wake up" EMG evaluation while the patient was emerging from anesthesia for another procedure. Proactive arrangements were made with the anesthesia department to ensure that muscle relaxants were not used. At the time of the evaluation, the patient was approximately 3 months postinjury. Pairs of monopolar intramuscular needle electrodes were positioned on the right side in the frontalis, orbicularis oculi, orbicularis oris, and mentalis muscles to record free-running EMG. All EMG responses were monitored using a multichannel EMG system typically used for intraoperative facial nerve monitoring.

Initially the free-running EMG was observed with the patient at rest and still under the general anesthetic. The temporalis, zygomatic, and buccal branches were electrically silent. Possible fibrillation potentials were noted in the mentalis muscle. We then asked the anesthesiologist to slowly emerge the patient from the general anesthetic while watching for EMG activity on the right side of her face, particularly when she made volitional movements on the left. There was some brief and extremely reduced motor unit activity from the right orbicularis oculi muscle; this did not demonstrate a normal recruitment pattern as the patient gradually awakened. A stronger recruitment pattern was recorded from the marginal mandibular branch. The frontalis and buccal branches demonstrated electrical silence. The impression based on this collection of results was that the prognosis for recovery without intervention was poor. Unfortunately, the patient's other medical conditions, current medications, and ongoing brain injures contraindicated a middle cranial fossa decompression. She is currently being monitored at our facility, and facial reanimation surgery will be considered when the patient is more medically stable. In retrospect, it was not possible to definitively differentiate fibrillation responses from spasm trains potentially related to lightening anesthetic. Hindsight dictates that it would have been beneficial to record EMG from the nonaffected side to act as a control.

This case study illustrates several points: (1) electrophysiological tests do not and cannot be the sole factor in deciding a patient's course of treatment. This patient was not a candidate for immediate surgical intervention. (2) However, electrophysiological tests can provide a prognosis for facial nerve recovery. This allows the patient and the family to be counseled about possible interventions and allows the surgical team to develop a plan that will benefit the patient. It is our opinion that performing ENOG testing under sedation or a general anesthetic in conjunction with a "wake up" EMG can provide valuable prognostic information in cases where the patient is not capable or unwilling to participate using standard evaluation techniques. This includes evaluations in the pediatric population.

Trauma cases also have the added factor of being more likely to involve complete nerve transection. In many cases, test interpretation follows the same guidelines established for idiopathic cases. In cases of traumatic or iatrogenic injury, the examiner needs to take into account all of the possible signs indicating either a positive or negative outcome. Table 7 displays various possible objective findings and separates them in terms of being indicators for a good versus bad outcome. Intervention decisions are made based on several factors, including time of onset after injury and physical examination. Certainly imaging studies add tremendous value and are essential before considering surgical intervention. However, ENOG and EMG can be an important guide in the decision-making process. Some guidelines include:

1. Primary facial nerve repair or cable graft is indicated in the presence of a confirmed fifth-degree injury. The best indicator of this is a complete facial paralysis at the time of injury. However, many patients are unconscious at the time injury and may be poor historians while recovering. Other signs to support a nerve transection include the early onset of Wallerian degeneration as indicated by ENOG and absent volitional activity on EMG at all recording sites.
2. Decompression should be considered in the context of ≥90% degradation on ENOG, especially when onset of denervation is <14 days. This can be further supported by completely absent volitional EMG, volitional EMG at only one recording site, or signs of abnormal resting potentials. Surgeons also have the opportunity to explore and decompress the facial nerve and then convert to a nerve graft if their intraoperative findings reveal a complete nerve section.
3. Observation and medical management: patients with an ongoing negative ENOG (<90% degeneration) are suspected to have a first-degree injury and will be expected to recover. In our facility, patients with volitional EMG at 2 or more recording sites are observed for 8 months or up to a year and typically recover to acceptable levels.

4. Facial reanimation procedures: a complete discussion of these surgeries is beyond the scope of this manuscript. In general, facial reanimation procedures are a last resort when it is felt that the native facial nerve will not provide effective facial movement. It is generally agreed that the native facial musculature requires some type of innervation, either from the native facial nerve or a donor nerve, within 18 to 24 months of injury to prevent relatively irreversible atrophy and fibrosis.

Iatrogenic injury

ENOG and EMG can be used to monitor patients who have facial nerve weakness after a surgical procedure, such as CPA tumor resection or parotidectomy. ENOG testing is not beneficial if the site of lesion is distal to the stylomastoid foramen, and therefore, in those cases, EMG may be the most useful tool. Serial testing can be used to document improvement in facial function or support the need for a nerve graft or other facial reanimation procedures. Decision making is similar to the paradigm covered in the Trauma section.

Conclusions

EMG and ENOG are excellent at diagnosing first-degree injuries (neuropraxia). In other words, patients with a negative ENOG and/or voluntary motor unit potentials in several facial distributions have a high probability of returning to normal or near-normal facial function. The PPVs of both ENOG and EMG are slightly lower than their NPVs. Although surgical decisions are not made on diagnostic studies alone, electrophysiological measures can provide valuable prognostic information that can guide medical decision making. These evaluations can be performed reliably in the clinic and are usually well tolerated. It is possible to obtain valuable diagnostic information on uncooperative patients by performing ENOG under sedation or a general anesthetic during a "wake up" EMG. Additional research on the predictive value of these studies in specific patient populations, such as temporal bone fractures, is indicated. EMG and ENOG should both be a part of the armamentarium of any clinic evaluating and treating facial paralysis and paresis.

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