



Premiere Publications from The Triological Society

Read all three of our prestigious publications, each offering high-quality content to keep you informed with the latest developments in the field.



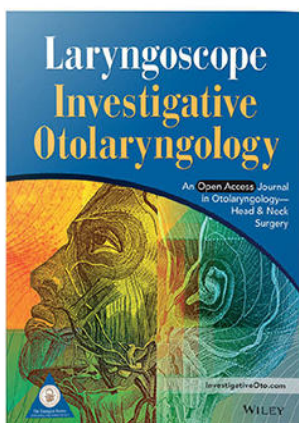
THE Laryngoscope

FOUNDED IN 1896

Editor-in-Chief: Samuel H. Selesnick, MD, FACS

The leading source for information in head and neck disorders.

Laryngoscope.com



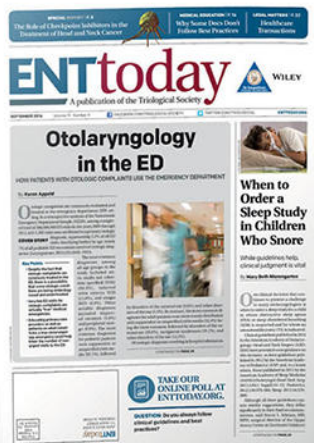
Laryngoscope Investigative Otolaryngology

Open Access

Editor-in-Chief: D. Bradley Welling, MD, PhD, FACS

Rapid dissemination of the science and practice of otolaryngology-head and neck surgery.

InvestigativeOto.com



ENTtoday

A publication of the Triological Society

Editor-in-Chief: Alexander Chiu, MD

Must-have timely information that Otolaryngologist-head and neck surgeons can use in daily practice.

Enttoday.org

WILEY

Best Practices in Facial Nerve Monitoring

Jack M. Kartush, MD, FASNM; Kent S. Rice, MS, CNIM, DABNM, R.EPT; Robert E. Minahan, MD, ABPN;
Gene K. Balzer, PhD, FASNM, FAAA; Charles D. Yingling, PhD, DABNM;
Christoph N. Seubert, MD, PhD, DABNM 

Objectives/Hypothesis: Facial nerve monitoring (FNM) has evolved into a widely used adjunct for many surgical procedures along the course of the facial nerve. Even though majority opinion holds that FNM reduces the incidence of iatrogenic nerve injury, there are few if any studies yielding high-level evidence and no practice guidelines on which clinicians can rely. Instead, a review of the literature and medicolegal cases reveals significant variations in methodology, training, and clinical indications.

Study Design: Literature review and expert opinion.

Methods: Given the lack of standard references to serve as a resource for FNM, we assembled a multidisciplinary group of experts representing more than a century of combined monitoring experience to synthesize the literature and provide a rational basis to improve the quality of patient care during FNM.

Results: Over the years, two models of monitoring have become well-established: 1) monitoring by the surgeon using a stand-alone device that provides auditory feedback of facial electromyography directly to the surgeon, and 2) a team, typically consisting of surgeon, technologist, and interpreting neurophysiologist. Regardless of the setting and the number of people involved, the reliability of monitoring depends on the integration of proper technical performance, accurate interpretation of responses, and their timely application to the surgical procedure. We describe critical steps in the technical set-up and provide a basis for context-appropriate interpretation and troubleshooting of recorded signals.

Conclusions: We trust this initial attempt to describe best practices will serve as a basis for improving the quality of patient care while reducing inappropriate variations.

Key Words: Facial nerve monitoring, best practices, technical set-up, facial electromyogram, facial nerve stimulation.

Level of Evidence: 4

Laryngoscope, 131:S1-S42, 2021

INTRODUCTION

Iatrogenic facial nerve injury can be a devastating consequence of surgical procedures performed along the course of the seventh cranial nerve. Because of anatomic variations and the extent of underlying disease, even the most experienced surgeon may injure the facial nerve.^{1,2} Injury may lead to a range of sequelae—from transient

mild paresis to severe long-term paralysis, including impairment of cosmetic appearance and function. Severe injury may lead to claims of malpractice.

Iatrogenic injury may occur at any point along the course of the facial nerve and therefore is seen during intracranial, intratemporal, and extratemporal procedures. Injury ranges from mild stretching or compression to complete transection. During temporal bone surgery, trauma from a high-speed drill may not only cause direct injury, but progressive ischemic neuropathy due to the fallopian canal exacerbating the effects of edema. In otologic surgery, it is not possible to accurately predict the severity of disease or the presence of anomalies beforehand. Green et al.³ have shown that there is a significant risk of iatrogenic facial nerve injury during presumably minor cases, for example, simple widening of the ear canal for exostoses. As detailed later in section Correlative Facial Nerve Anatomy, the high incidence of fallopian canal dehiscence is another factor that must be considered even in the otologic case that seems routine and low risk.

Facial nerve monitoring (FNM) was first described by Fedor Krause in 1898 and represents the first attempt at intraoperative neurophysiological monitoring (IONM).^{4,5} However, monitoring was rarely used until the 1960s when otolaryngologists used simple electric stimulation and facial twitch observation during parotid and acoustic tumor (vestibular schwannoma) surgery. Subsequently,

From the Michigan Ear Institute (J.M.K.), Farmington Hills, Michigan, U.S.A.; NuVasive (K.S.R.), San Diego, California, U.S.A.; Department of Neurology (R.E.M.), Georgetown University, Washington, District of Columbia, U.S.A.; Real Time Neuromonitoring Associates (G.K.B.), Real Time Neuromonitoring Associates Nashville, Tennessee, U.S.A.; The Golden Gate Neuromonitoring, Department of Otolaryngology and Head and Neck Surgery (C.D.Y.), Stanford University, San Francisco, California, U.S.A.; and the Department of Anesthesiology, Division of Neuroanesthesiology (C.N.S.), University of Florida College of Medicine, Gainesville, Florida, U.S.A.

Additional supporting information may be found in the **online version** of this article.

Editor's Note: This Manuscript was accepted for publication on February 2, 2021.

This study and its publication were funded by the authors and the University of Florida Department of Anesthesiology. Additional funding was provided by Real-Time Monitoring Associates after completion of the writing/revision of the manuscript.

The authors have no other funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Christoph N. Seubert, MD, PhD, DABNM, Department of Anesthesiology, University of Florida College of Medicine, 1600 SW Archer Road, PO Box 100254, Gainesville, FL 32610. E-mail: cseubert@anest.ufl.edu

DOI: 10.1002/lary.29459

recording was performed using accelerometers or intramuscular needle electrodes. IONM has since evolved to include many other modalities and surgical procedures, many of which cannot be performed by the surgeon alone. This in turn led to a new industry wherein technologists, neurophysiologists, neurologists, and anesthesiologists have developed important roles, especially in complex multimodality monitoring. Today, many surgeons continue to perform FNM on their own, while others work cooperatively with their IONM team members. A detailed history of FNM is available Appendix S1, in the **online version** of this article.

IONM of the facial nerve has become an important adjunct to reduce the incidence and severity of iatrogenic facial nerve injury. FNM can assist the surgeon in four ways. First, localizing the nerve via electric stimulation (“triggered EMG”) is of particular value when pathology or anomalies distort normal anatomical landmarks. Second, ongoing monitoring can detect injury during surgical maneuvers that may not otherwise be apparent. Third, post-dissection stimulation can confirm the function of the nerve prior to closure. Finally, surgeons-in-training may learn new technical skills more quickly based on the immediate electromyogram (EMG) feedback of stretch-induced trauma.

Despite these benefits, monitoring is an adjunct—not a replacement—for surgical training, experience, and skill. The information conveyed by monitoring must always be considered in context with anatomy and the ongoing surgical events. Once monitoring becomes an integral part of the procedure, the surgeon will come to rely on monitoring information. However, any deficiencies in the technical or interpretive aspects of monitoring—by the surgeon or any member of the team—can increase the chance of patient injury. Consequently, the benefits of monitoring can be optimized only when all members of the monitoring team have proper training.

Surgeons who perform monitoring on their own must have *procedure-specific* training and experience on both the technical and interpretive aspects of monitoring. Surgeons who choose to delegate monitoring to their IONM team members must nonetheless have sufficient training to properly apply monitoring information to the surgical procedure. Likewise, the training of technologists and supervising neurophysiologists must include an understanding of the sequential steps in a surgical procedure as well as recognizing periods of high- versus low-risk surgical dissection.

Despite the widespread acceptance of FNM, malpractice lawsuits for cranial nerve injury have actually increased over the last 25 years. Svider et al.⁶ have shown that facial nerve injury is the most commonly litigated cranial nerve injury. Their study could only assess cases that went to trial even though most settle out of court or are dismissed before trial. Because only 15% of surgical malpractice litigation reaches the courtroom, the actual number of iatrogenic nerve injury cases is much higher. Svider et al. identified 391 cranial nerve injury cases of which 209 were suitable for review. Of these, 33% of trials resulted in damages awarded—and otolaryngologists were the most commonly named defendant.

When iatrogenic facial nerve injury occurred in the past, the focus of investigation was on informed consent, indications for surgery, and details of the operative procedure. Today, there are new allegations that relate to IONM: 1) failure to monitor, and 2) failure to monitor correctly.⁷

Failures in IONM may occur at many levels, including procedure-specific training, technical set-up, troubleshooting, interpretation, communication, adherence to protocols, and implementation of the neurophysiologic data to the surgical procedure. While each of the aforementioned situations will be detailed in other sections, analyzing FNM failures reveals a number of key factors. First, there is no universally recommended curriculum for resident training of IONM, thus methods and extent of training vary. As detailed later, an American Academy of otolaryngology poll of department chairs by Gidley and Maw noted formal training in only 61% of their programs.⁸ Second, expertise in one application of an IONM modality (e.g., EMG) does not convey expertise in other applications. For example, simple electrical stimulation with EMG recording during spine surgery is a procedure commonly assigned to technologists-in-training given its relative simplicity and low-perceived risk for injury. However, documented competency in this particular use of nerve stimulation and EMG recording is a useful but insufficient foundation to effectively monitor the facial nerve. Likewise, surgeons trained in laryngeal nerve monitoring with a dedicated nerve monitor must be taught the critical differences when applying the same device to FNM. Otherwise, variations in nerve latency and default settings of the stimulus-ignore period could result in false-negative errors.

Once the benefits of monitoring became apparent in high-risk procedures such as acoustic tumor surgery, FNM became increasingly used in lower-risk procedures such as operations of the middle ear and mastoid. As detailed here, over the last two decades, FNM has become nearly a routine adjunct whenever the surgeon perceives the facial nerve may be at risk during surgical dissection including tympanomastoidectomy, cochlear implantation, labyrinthectomy, endolymphatic sac decompression, middle ear surgery (ossicular chain reconstruction and tympanoplasty), aural atresia repair, acoustic neuromas, parotidectomy, submandibular gland excision, and skull base surgery, for example, meningiomas and glomus tumors. According to Hughes et al.,⁹ there are nearly 13,000 otolaryngologists in the United States—but FNM is no longer within the sole providence of otolaryngologists, being often used by neurosurgeons, general surgeons, and oral surgeons. It is therefore surprising to note the lack of any recommended guidance on best practices in this arena.

Numerous clinical practice guidelines are available in otolaryngology, including guidelines on removing ear wax¹⁰ and treating swimmer's ear,¹¹ but surgeons have no resource on facial nerve monitoring. The inertia of national organizations to organize along these lines has led groups of individuals to publish their own recommendations to improve patient care. For example, Gregory Randolph, Henning Dralle, and others informally organized like-minded thyroid surgeons into an “International Neural Monitoring Study Group” that published dozens of articles describing best

practices of recurrent and superior laryngeal nerve monitoring that have immeasurably benefitted surgeons and their patients.^{12–14}

We likewise assembled a multidisciplinary team of subject matter experts from various fields to describe best practices for FNM, including surgeon, neurophysiologist, neurologist, monitoring technician, and anesthesiologist, working in both academic and private practice settings.

A search for evidence-based literature in MEDLINE, PubMed, Cochrane Reviews, Guidelines International Network, and the National Guidelines Clearinghouse was performed using appropriate MeSH headings for facial neurophysiological monitoring without a backward date limit. The sorted material formed the basis to support and describe: 1) procedure-specific best practices and 2) indications for when FNM should be considered. Background material is presented not only for the surgeon but also as a resource for non-surgeon clinical stakeholders who are now commonly involved in IONM.

A review of 1,340 publications revealed only one prospective, randomized controlled trial for parotid surgery¹⁵—and not a single one for surgery of the ear or skull base. As detailed in the historical online material, the benefit of FNM during acoustic tumor surgery appeared so significant within a short period that those who were once skeptical rapidly came to believe that not only was a prospective randomized controlled trial not needed, but that it likely would be considered unethical to withhold monitoring. In 1988, after using FNM for just 2 years at the Mayo Clinic, Dr Stephen Harner stated, “I don’t think I could convince anybody at our institution with experience to give up monitoring under any circumstances.”¹⁶ In fact, the current literature on Clinical Guidelines demonstrates that, due to a dearth of high-level evidenced-based studies, the great majority of *all* “Guidelines/Standards/Clinical Recommendations” are based predominately on expert opinion. This has just been acknowledged by the American Academy of Otolaryngology wherein they state: “The change from “Clinical Consensus” to “Expert Consensus” is being made to highlight the use of expert evidence in the development of these documents.” And: “The current (*Consensus Development*) Manual¹⁷ aligns with these criteria above and therefore the name change from “Clinical Consensus Statements” to “Expert Consensus Statements” is appropriate.”¹⁸

With the aforementioned absence of facial monitoring randomized controlled trials, this report relies on the literature’s best available evidence and our multidisciplinary panel of experts to provide a rational basis for best practices to improve the quality of patient care during FNM.

FNM BEST PRACTICES, NOT A MEDICOLEGAL DOCUMENT

Our aim is to define best practices under typical circumstances. Because each surgical procedure has unique circumstances, a lack of adherence to some aspects of these practices cannot be construed to imply negligence or breach of duty.

INDICATIONS FOR FNM

FNM can provide useful information whenever the surgeon believes the nerve may be at risk along its anatomic course. While there are no societal sponsored clinical practice guidelines that specify indications or specific methodologies for FNM, monitoring is commonly used for intracranial, skull base, otologic, and parotid surgery.

Following a 1991 presentation summarizing the benefits of FNM by Jack Kartush to the National Institutes of Health (NIH), the NIH published a national “Consensus Statement on Acoustic Neuroma” recommending that FNM be used routinely. This led to FNM becoming a de facto standard of care in the United States during acoustic tumor surgery.¹⁹ In a survey by Kartush,²⁰ 100% of 230 North American otologists in the American Neurotology Society reported routine use of FNM during every acoustic tumor surgery.

Establishing the cost-effectiveness for lower-risk procedures, however, has only been addressed more recently through expert opinion and surgeon surveys—not controlled trials. The reasons for this are twofold. First, researchers would need to withhold monitoring from their patient’s control group. But despite any theoretical questions that remain of clinical equipoise (i.e., uncertainty about the benefits of FNM), no such study has ever done so, likely for the reasons espoused by Harner, noted above. Second, even if this obstacle was surmounted, the need for an extremely large sample size powered to demonstrate differences when complication rates are small has meant that no such study has been done—nor likely ever will be. Consequently, as detailed here, recent literature now strongly supports the use of FNM for lower-risk procedures such as middle ear and mastoid surgery, as well as skull base surgery.

Despite a general reluctance to publish recommendations that could be construed as suggesting a standard, in 1988, Herbert Silverstein and colleagues²¹ published one of the early articles that “encouraged” its readers to use FNM in every otologic surgery. In 1994, Pensak et al.²² published a strong recommendation for FNM in training programs: “Facial nerve monitoring should be performed in all chronic ear cases in which the facial nerve may be at risk. The benefits to the patient and the resident surgeon are real.” In the aforementioned ANS survey by Kartush, FNM was used in 95% of cochlear implant operations and nearly 90% of mastoid surgeries.⁷ In contrast, monitoring was only used in 50% of tympanoplasties (Fig. 1).

Likewise, Gidley et al. described broad agreement that FNM is indicated in acoustic neuroma surgery, skull base surgery, atresia surgery, cochlear implant surgery, mastoid surgery, and revision chronic ear surgery among the 268 respondents of an American Academy of Otolaryngology-sponsored survey.⁸ The only otologic cases monitored less than 50% were tympanoplasty and stapedectomy.

A systematic review of FNM during parotid surgery found that “a majority of otolaryngologists in the United States are employing facial nerve monitoring during parotid surgery some or all of the time, even though no studies to date have demonstrated improved outcomes with its use.”²³

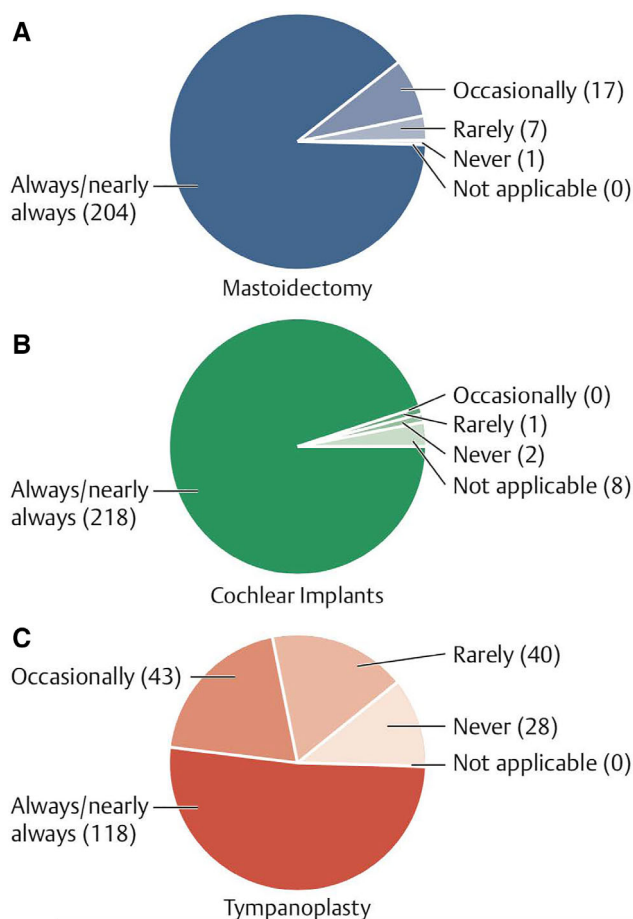


Fig. 1. Use of intraoperative facial nerve monitoring during (A) mastoidectomy, (B) cochlear implantation, and (C) tympanoplasty. Results of a survey of 230 otologic surgeons reporting on the use of intraoperative monitoring during routine middle ear and mastoid surgery. Used by permission Jack M Kartush MD

Following years of reluctance, the otologic literature has now published a quartet of articles unequivocally advising otologic surgeons to routinely use FNM for middle ear and mastoid surgery. Here are the key recommendations from each article:

To enhance the safety of facial nerve during ear surgery, *the otolaryngologist should consider the use of monitoring in middle ear and mastoid surgery.*²⁴

Facial nerve monitoring is *cost-effective*, and *its routine use should be adopted* to reduce the risk of iatrogenic facial nerve injury during otologic surgery.²⁵

The current evidence suggests that intraoperative facial nerve monitoring *is of value in identifying the facial nerve* that is at surgical risk during middle ear and mastoid surgery, and it is also *cost-effective.*²⁶

Although facial nerve monitoring is not legally the standard of care (as shown in one of our cases), *its routine use should be adopted* to reduce the risk of facial nerve injury during middle ear and mastoid surgery.²⁷

With these strong statements supporting routine FNM, may it be considered a “Standard of Care” in the United States? This is a complex question that eludes a simple answer, especially as the standard varies in different locations. The medicolegal aspects specific to nerve monitoring and iatrogenic injury have been detailed by Slattery and Kartush.⁷ Given the aforementioned literature, however, surgeons who cause an iatrogenic injury in a case they elected not to monitor can expect to be asked at deposition to justify their decision to forego FNM. Although each surgeon or surgical association may individually consider the pros and cons of specific indications for FNM, the earlier-cited literature and the analysis of current practice patterns should be strongly considered. A useful analogy on how standards evolve can be seen in the field of anesthesiology regarding the slow adoption of pulse oximetry during general anesthesia (Appendix S2, in the **online version** of this article).

Regarding costs, in the Wilson article cited above, the cost of monitoring was estimated between US \$222.73 and \$525.00 per case.²⁵ The authors conclude that this cost was offset by avoiding the high management costs of facial nerve paralysis.

EVOLVING MODELS OF FNM

Proper monitoring requires integration of technical aspects and interpretation of FNM into the ongoing conduct of a specific surgery for a specific patient and a specific disease. Such integration may involve just one person, as is the case when the surgeon employs a FNM device on their own. The surgeon assumes responsibility for all aspects of monitoring: technical, interpretive, and application to the surgical procedure. Conversely, monitoring may be performed as a team, which may include a technologist and an interpreting neurophysiologist where expertise as well as communication with the surgeon are critical components. In this setting, the surgeon must integrate the information conveyed by the team and apply it to the operation. We now discuss the unique duties of surgeon, technologist, and interpreting neurophysiologist for FNM and their training, before considering models of FNM.

Duties of the Surgeon

With deference to the many skilled members of the operative team, the surgeon holds a unique position. No others have the breadth and length of context-specific surgical training, nor are they responsible for the final interpretation, integration, and implementation of FNM into the surgical procedure. The surgeon must ensure that every aspect of the patient’s care, over which they have control, is optimized. They must decide the proper

indications for FNM and then take into consideration the equipment and personnel at the hospital facility they choose. Finally, just as a surgeon must always self-assess in deciding whether they have the necessary skills to perform an operation, they must also honestly assess their FNM training and skills.

Surgeon FNM Training. Although surgeons have an extremely broad and in-depth medical education, training in FNM has not been highly formalized. This potential deficit in training has occurred because of the relatively recent adoption of monitoring as a standard practice in many procedures.

Gitley and Maw²⁸ surveyed 1,500 otologists as well as 120 otolaryngology program directors in a report directed by the American Academy of Otolaryngology—Head and Neck Surgery Intraoperative Nerve Monitoring Task Force. They noted that FNM “plays a significant role in the training of residents in ear surgery.” Almost 90% of practicing surgeons reported that they had received training on FNM and over 90% monitored mastoid operations. Placement of recording needles and set-up of the monitor was performed by the surgeon in 60% of cases, whereas this responsibility was “delegated to a monitoring service (18.1%), a resident (15.2%), an audiologist (4.1%), or a nurse (1.5%) in a minority of cases.” Responding to the facial nerve monitor during the case “was handled overwhelmingly by the primary surgeon (85.5%).”

While this survey paints an encouraging picture, there were some concerning findings. Only 43% of respondents used electrical stimulation during mastoid surgery, even though—as discussed later—such stimulation is the only way to assure a functioning monitoring set-up. The converse means that without routine nerve stimulation, 57% of otolaryngologists did not obtain baseline current flow testing or a baseline facial nerve response. Furthermore, program directors affirmed formal training in only 61% of their programs; the remainder presumably receiving their training informally through other residents, staff, or vendors. Informal training (e.g., “see one, do one, teach one”) methods may result in a dilution of the original teachings that were introduced years before, often by the device vendor. Nor do most departments have a Policy and Procedure manual to follow. Only 22% of program directors were aware that nerve monitoring is now considered a competency of the American Board of Otolaryngology. Inconsistencies in technique and interpretation need to be remedied by a standardized core curriculum followed by testing for competency.

At this time, there are no surgical societies that mandate a uniform FNM training program with credentialing for monitoring expertise. Corollaries to such a requirement exist. For example, many hospitals have required special training or a letter from their department chairs for new, higher-risk procedures such as endoscopic, laser, or robotic surgery until such time as these modalities become well established over the years in residency programs. Consequently, for now, it is the surgeon's duty to take a realistic assessment of his or her own monitoring training to determine which model of FNM, (surgeon-directed or assisted) is in the best interest of his/her patient.

Duties of the Technologist

The technologist plays a critical role in ensuring a safe and proper technical set-up, vigilantly monitoring the facial EMG, identifying and resolving technical issues, differentiating artifacts from legitimate EMG events, and documenting and communicating these events to the surgeon. If multimodality neuromonitoring is being performed during facial nerve neuromonitoring (e.g., brainstem auditory-evoked potentials), the technologist will also be responsible for reporting evoked potential waveform changes to the surgeon. Due to the nature of EMG events and the potentially immediate risk to the facial nerve, the technologist must assume the responsibility of identifying EMG activity and reporting this to the surgeon without delay, whether or not concurrent use of audio feedback to the surgeon serves as a first alert. When triggered EMG (tEMG) is performed, the technologist will set initial stimulation parameters and will report the evoked waveforms, including their validity or reliability, if there are confounding technical issues.

Technologist FNM Training. There are no universally accepted criteria for the educational requirements to become a monitoring technologist. Nonetheless, Certification in Neurophysiologic Intraoperative Monitoring (CNIM) is now well-recognized for technologists and is offered through the American Board of Registered Electroneurodiagnostic Technologists. The extent to which the underlying training confers competency in FNM will vary among technologists.

The Neurodiagnostic Society (originally founded as American Society of Electroencephalographic Technicians) has published standards that speak to the technical performance of motor cranial nerve monitoring.²⁹

Duties of the Supervising Neurophysiologist

The supervising neurophysiologist is focused on the interpretative aspects of the monitoring and oversight of the technologist's duties (or performance thereof if the neurophysiologist fills both roles). Interpretative aspects involve the correct identification of EMG patterns, the correct identification of nerve stimulation results, including unexpected amplitude or latency findings, ensuring proper communication of these findings to the surgeon, explanation of implications of the activity as needed, and availability for consultation as desired by the surgeon. Interpretations include implications of any possible confounding factors present such as preoperative nerve dysfunction or the presence of neuromuscular blockade. The neurophysiologist may delegate some of the hands-on aspects to the technologist as they deem necessary,³⁰ and if a question of data integrity arises, the neurophysiologist, in concert with the technologist, need to be proactive in troubleshooting and attempting correction of the root causes of poor data. The oversight role includes differentiation of physiologic from non-physiologic signals.

The surgeon should receive *real-time feedback of EMG findings* via verbal report from the monitoring team and/or auditory alerts from the monitoring system. Subsequent interpretation of this feedback typically requires

correlation to ongoing surgical events that in turn requires an intimate understanding of the surgical anatomy and the surgical actions preceding and during electrophysiological events. The surgeon has the deepest understanding of these aspects and must always be an active participant in the interpretative process. The degree to which the overseeing neurophysiologist adds to the correlative aspect of the interpretation may vary substantially depending on the depth of information the surgeon wishes to receive for a given case.

Neurophysiologist Training. The training and background of interpreting neurophysiologists is not uniform. There are no universally accepted medical or societal criteria to become a neurophysiologist specializing in IONM. Typically, those applying their neurophysiology skills toward neuromonitoring will have advanced training in neurophysiology and neuroanatomy; will have a basic understanding of the technical performance of neuromonitoring; will have completed a clinical neurophysiology fellowship or have trained with a senior specialist in IONM; and will have spent time in the operating room observing and supervising IONM procedures. The heterogeneity of backgrounds and training is reflected in the number of boards that confer certification in intraoperative neurophysiology. Each credentialing/eligibility board requires continuing education for renewal of their specific certification. Credentialing requirements may be specified to gain privileges at individual hospitals, and these privileges should be obtained by overseeing neurophysiologists for each hospital or surgery center where they practice. Hospitals may require a specific number of monitored cases within the past year and they often include one of the following board certification/eligibility relevant to neuromonitoring:

- American Board of Neurophysiologic Monitoring (ABNM) (physicians and doctoral-level non-physicians)
- American Board of Clinical Neurophysiology (ABCN) (physician only)
- American Board of Psychiatry and Neurology added qualification in Clinical Neurophysiology (ABPN) (physician only)
- American Board of Electrodagnostic Medicine (ABEM) (physician only)
- American Audiology Board of Intraoperative Neuromonitoring (audiologist only)

While current trends in neuromonitoring point toward practitioners holding one or more of these credentials, as stated in the ASNM practice guidelines,^{31,32} “many Board-certified anesthesiologists, surgeons or proceduralists, and neurologist IONM-Ps, without specific certification by recognized IONM Boards, have contributed significantly to IONM literature/education/practice and will, in the near-term, continue to appropriately serve as qualified neuromonitoring professionals.”

Models of FNM

FNM has evolved into a variety of clinical models. The most common models in the United States are

TABLE I.
Models of Facial Nerve Monitoring.

Surgeon only
Surgeon + Technologist
Surgeon + Technologist + On-site Neurophysiologist
Surgeon + Technologist + Remote Neurophysiologist

depicted in Table I. FNM began in the 1960s as the surgeon performed both the technical and interpretive aspects of monitoring and then integrated this information directly into the surgical procedure. No other IONM personnel were available at that time to assist the surgeon. Decades later, monitoring of spinal and neurovascular surgeries led to technologists and neurophysiologists who were trained in multiple modalities such as somatosensory-evoked potentials and transcranial motor-evoked potentials, in addition to electromyography.

Other models occur more rarely such as 1) supervision performed by the anesthesiologist or 2) both technical and interpretive components performed by the neurophysiologist. The choice of model typically falls to the surgeon but is dependent on the facilities and personnel available at a given site. Additional factors include case complexity, other monitoring modalities used, and the availability and expertise of other monitoring personnel. To date, no studies have been performed to assess differences in safety and efficacy across the various models of FNM.

Surgeon-Only Monitoring. For surgeons who are trained in the interpretative and technical aspects of FNM, surgeon-only monitoring remains a common approach. The surgeon can set up the system, apply electrodes before draping, listen for EMG reactivity during the case, and map the location of the facial nerve with a stimulating probe provided that the response to stimulation is made audible by a time-locked EMG response or a tone triggered by EMG exceeding a pre-set threshold.

For the surgeon to evaluate waveforms (e.g., to distinguish true EMG responses from artifacts), the monitoring device must have visual representation of the EMG and the surgeon must be trained in waveform interpretation. The ability to alter current intensity is crucial to optimize nerve mapping. Surgeon-directed monitoring requires that the surgeon has had procedure-specific training in neural stimulation and evaluating the EMG responses. In surgeon-directed monitoring, the surgeon directs other operating room personnel such as nursing staff to adjust the stimulus intensity as needed.

Surgeon-only monitoring is limited to cases where facial nerve EMG is the only monitoring modality. When other monitoring modalities such as auditory brainstem responses (ABR), somatosensory-evoked potentials, motor-evoked potentials, or monitoring of other cranial nerves are used, additional technical and/or professional personnel are required.

Assisted Monitoring

Surgeon + Technologist Monitoring. Some surgeons choose to augment their monitoring with the

assistance of an in-room technologist who can connect electrodes, set initial device parameters, and make real-time adjustments such as altering stimulus intensity and the volume of auditory feedback. Technologists, at the direction of the surgeon, can also provide valuable assistance in troubleshooting the device and connections.

Furthermore, in the past, surgeons often entrusted experienced technologists to assist them in interpretation. However, as detailed below, the American Medical Association in 2008 declared interpretation of IOM as the “practice of medicine.” Thus, just as in the surgeon-only model, the surgeon takes responsibility for FNM interpretation—and must consequently ensure that the technologist’s training is adequate for the task.

The surgeon-only and surgeon + technologist models have been validated in studies that were the basis for the information presented to the NIH for the Consensus Statement on Acoustic Neuromas. No studies are available specifically assessing the model of remote monitoring by an off-site neurophysiologist.

Surgeon + Technologist + On-Site Neurophysiologist Monitoring. If the supervising neurophysiologist is present in the operating room (“on-site”), he or she is able to directly communicate with the surgeon and provide physiological context to surgical actions, thus negating the need for a remote connection. Situational awareness and communication are optimized among surgeon, technologist, and neurophysiologist.

Unfortunately, there are not enough qualified IONM professionals to staff every operating room. The burgeoning need for monitoring personnel, especially at a doctorate supervisory level, could not be met by the few initial pioneers who came from diverse backgrounds, including neurology, physiology, psychology, anesthesia, and audiology. While academic departments and monitoring companies began training programs, it quickly became clear that the manpower shortage required alternate solutions. This led to the rise of “remote monitoring” models of care where a CNIM-level technologist in the room is supervised by an online professional, viewing the data in real time, offering advice and feedback when necessary.

Surgeon + Technologist + Remote Neurophysiologist Monitoring. In 1987, Robert Scلابassi and his team began reporting on NeuroNet, a distributed real-time system for monitoring neurophysiologic function remotely.³³ It leveraged the ability of each supervisor to be able to monitor *multiple cases simultaneously*. This was particularly important in the United States because the valuation of IONM interpretation (based on the country’s work relative value unit model) has been so far below fair market value (average neurologist revenue requirements) that transferring one’s traditional neurologic practice to “in-room, one-on-one monitoring” is unsustainable.

For many hospitals, the multiple/simultaneous remote/teleneurology monitoring model is the only available model for the provision of professional supervision and clinical interpretation. This model works well for modalities such as evoked potentials, which are on a time scale of minutes rather than seconds. Somatosensory-

evoked potential and motor-evoked potential changes indicate potential impending problems, but there is generally a window of several minutes during which the causes of such changes can be explored and remedial actions taken. The causes may be technical, positional, physiological, or anesthesia-related, or due to surgical maneuvers, but in most instances can be identified and reversed in time to avoid permanent deficits. Inclusion of remote professional input into the decision process is therefore of unequivocal value.

In contrast, facial nerve EMG data are collected and displayed on a second-by-second basis that requires virtual instant communication between the surgeon and the IONM team. If the spontaneous EMG is made audible and appropriate steps are taken to avoid non-EMG sounds (such as electrocautery artifact), then the surgeon has immediate access to the relevant data and no intermediaries are necessary. The remote neurophysiologist, using standard connection methods, is subject to internet time delays, lack of ability to visualize the surgical field, and dependence on chat functions for feedback (which must be relayed to the surgeon through an in-room technologist). These combine to make instantaneous remote interpretation of EMG responses almost impossible.

Despite this, the remote professional may still provide value in FNM, provided real-time feedback of EMG activity is given by the person in the room as discussed above. Examples include suggestions to explain unusual response patterns (trigeminal, nervus intermedius), differentiation of noise from physiologic signals, assistance in identifying and eliminating artifacts, confirmation or clarification of specific EMG patterns observed, and literature-based prognostic assessments.

Given the need for instantaneous feedback with EMG techniques, the “monitorist in the room” is critically important. The technologist in the room must be adequately trained and qualified to provide detailed, real-time feedback to the surgeon. In this setting, the remote professional’s interactions with the technologist remains a critical link. The immediate report of EMG is the most vital aspect so that it can be correlated to surgical events in real time. There is a broad range of experience among technologists, even among those who have achieved CNIM certification. Therefore, the supervising neurophysiologist can provide additional benefit based on his or her advanced training and expertise.

This input from the neurophysiologist also fills a teaching role. The neurophysiologist has substantial expertise in the clinical and scientific basis of neuromonitoring. Furthermore, the remote neurophysiologist garners experience on an ongoing basis at a faster rate than a person in the operating room because he/she is able to focus on critical aspects of cases. The nature of neuromonitoring in general and FNM, in particular, involves a watchful waiting for relatively rare events. For any trained neuromonitoring clinician, it is the experience of these rare events that provides the primary impetus for ongoing learning. A neurophysiologist working with the technologist allows transfer of this clinical experience. For the highest-level technologists, learning

curves flatten and discussions of data become more of a collaborative discussion between experts.

Remote monitoring can therefore provide considerable value but it is not without its limitations. As Skinner et al.^{34,35} have reported, remote monitoring as it is currently implemented results in reduced situational awareness and compromised communication, which can increase the chance of errors. In the great majority of cases, the remote neurophysiologist views the ongoing EMG but is “blind to the surgical procedure and deaf to the surgeon’s comments.”

Skinner also notes that collaboration between the surgeon and neurophysiologist during critical decision-making is enhanced when there is a foundation of trust. Trust, however, is typically earned rather than simply assumed. In current remote monitoring models, however, trust is more difficult to develop when there is typically little or no communication between the surgeon and neurophysiologist. When communication does occur, rather than face-to-face interactions, most often it is a few lines of text chat conveyed secondhand to the surgeon by the technologist.

As noted earlier, some of the original descriptions of remote monitoring by Sciabassi et al.^{30,36} went well beyond the current model of waveform reading supplemented by technologist chats. They described use of audio-video feeds from the operating room so that remote neurophysiologists could correlate electrophysiological data with real-time surgical events. They advised of the importance of enhancing situational awareness by conveying audio-video information, not just of waveforms and “talking heads,” but of the surgical field, that is, “video-as-data.” In so doing, the remote neurophysiologist’s ability to correlate electrophysiological data with real-time surgical events is meaningfully improved.

At the time of Sciabassi’s initial description, however, limitations in bandwidth and technology prevented such a desirable system from being adapted. In the ensuing decades, there has been an exponential growth in technology allowing heretofore unimagined abilities to convey enormous amounts of data wirelessly. Witness our current routine ability to now have a stadium of 100,000 football fans all receiving streaming video. And yet today, such critical audio-video feeds remain a rarity in IONM. Root causes of this include the reluctance to introduce video into the operating room, the lack of commercial systems, neurophysiologist staffing, HIPAA concerns, and simple inertia.

Maw and Gidley²⁸ reviewed remote monitoring from the surgeon’s perspective. They expressed concerns that remote supervision “has multiple elements that can impede the transmission of information, including communication delay, communication failure, lack of waveform, and lack of attention. For optimum performance, FNM needs to be instantaneous. The only way to achieve this instantaneity is by auditory signal from the nerve monitor and by perception of the operating surgeon without a middle party.” Regarding the exclusion of reimbursement to the operating surgeon, Maw and Gidley²⁸ commented that “the notion that an operating surgeon is incapable of devoting adequate attention to monitoring

while performing surgery is discordant with allowing remote monitoring by a second party who is monitoring multiple simultaneous procedures.”

Alternative Models: “Interpreter in the Room”.

Remote models have drawbacks that have led to a desire to have the “expert in the room” and a hope that higher-level technologists could fill this role. The only acceptable model in the United States at this time, assuming there is no IONM professional in the room, is an IONM technologist who has been adequately trained and qualified to provide detailed, real-time feedback to the surgeon.

An alternative to the American remote monitoring model is being developed in Canada to address both the manpower shortfall and the need to “have the expert in the room.” A non-doctorate individual is trained to serve in the operating room for both technical and interpretive roles. A two-year Canadian Association of Neurophysiological Monitoring educational program is followed by a one-year apprenticeship. The practitioner is then required to perform at least 300 independently monitored cases and have 36 months of experience before qualifying to take the Certified Intraoperative Neurophysiology Practitioner examination. This means that “the IONM interpreter is physically present in the operating room bolstered by clinical situational awareness and the ability to contribute to remedial measures should signal changes occur.”³⁷ Such awareness “affords the IONM practitioner the interpretive context to prioritize relevant patient data and expeditiously initiate IONM alerts so that remedial efforts can be implemented.” However, the Canadian requirement of “300 independently monitored cases” is likely to include only a small percentage of FNM cases. Thus, the role for a more experienced neurophysiologist in teaching and online oversight could add value and help fill any gaps in the technologist’s experience.

CORRELATIVE FACIAL NERVE ANATOMY

We highlight here certain key points of facial nerve anatomy as they apply to interpretation during FNM.

In 1778, a 23-year-old medical student, Samuel Soemmering, proposed that cranial nerves be numbered I through XII, ranked according to the nerves rostral-caudal exit points. This nomenclature has since become universally accepted. However, Corrales et al.³⁸ have demonstrated through surgical and radiographic study that in the majority of cases, the seventh cranial nerve actually emerges caudal (below) the eighth cranial nerve. Furthermore, the sixth cranial nerve was noted to be caudal to both of these nerves in 93% of cases. This means that Soemmering’s classic 1778 cranial nerve numbering system is in error. Furthermore, medical illustrators have perpetuated the error by drawing what they expect versus what they have observed. After centuries, it is unlikely that the numbering system will be changed, but surgeons and monitoring personnel must take note of the actual anatomy rather than the dogma.

The facial nerve motor nucleus lies in the caudal pons. As intra-axial motor fibers exit the nucleus, they sweep rostrally and dorsally where branches bend around the

dorsal surface of the abducens nucleus (genu of the facial nerve). From there, the fibers run laterally and caudally to exit the pons ventrolaterally at the pontomedullary junction.

The nerve comprises a central nervous system segment and a peripheral nervous system segment separated by a transitional zone approximately 2 to 3 mm from the root entry zone (REZ) of the pons, known as the Obersteiner-Redlich zone. The central segment is covered by oligodendrocytes and the peripheral segment is covered by more robust Schwann cells. The peripheral segment is further supported by three additional layers of connective tissue in a funicular organization: epineurium, perineurium, and endoneurium. The transition zone is clinically important because the paucity of connective tissue protection makes the nerve more vulnerable to injury at this location. Consequently, the surgeon and monitoring team can expect a higher likelihood of stretch-induced EMG potentials during cerebellar retraction or direct surgical dissection. Furthermore, the fragility of nerves at the REZ make it a common site of origin for facial nerve spasms (VII) or trigeminal neuralgia (V) that may be treated with microvascular decompression.

Extra-axial motor fibers run laterally in the cerebellopontine angle (CPA) and reside superolateral to the vestibulocochlear nerve within its course from the brainstem to the internal auditory canal (IAC). In this location, the nerve is most at risk during resection of acoustic neuromas (vestibular schwannomas) and other CPA tumors. The proximity to the vestibulocochlear nerve impacts dissection when hearing preservation is attempted. Furthermore, the trigeminal nerve is only millimeters away from the facial nerve at the REZ. This proximity is important to consider because in situations where a tumor alters or obscures the usual anatomy, the trigeminal nerve may not be easily differentiated from the facial nerve within the surgical field. Further complicating this scenario, electrical stimulation of the trigeminal nerve consistently yields compound motor action potential (CMAP) responses in recordings from facial nerve-innervated muscles due to volume-conducted responses from the masseter and temporalis muscles.^{5,39–41} Electrical differentiation is based on the latency of the CMAPs generated as described in the section on facial nerve conduction studies. Relative amplitudes of CMAPs may also help in this differentiation when both trigeminal- and facial nerve-innervated muscles are monitored simultaneously.

A small facial nerve branch carrying sensory and parasympathetic fibers runs between these two larger nerves, earning the name *nervus intermedius* (of Wrisberg) and projecting centrally to the tractus solitarius, spinal nucleus of the trigeminal nerve, and glossopharyngeal nucleus. Electrical stimulation of the *nervus intermedius* may yield very long latency CMAP responses (~11 msec).

The subsequent course of the facial nerve through bony structures of the skull consists of the meatal, labyrinthine, tympanic, and mastoid segments that together comprise the fallopian canal. The tympanic segment is at particular risk during tympanomastoidectomy as this is the most common site of fallopian canal dehiscence. In a study of surgical patients, Selesnick and Lynn-Macrae⁴²

identified dehiscences in one of three patients of which 80% were at the tympanic segment. They advised that surgeons be highly vigilant when dissecting in this area and suggested that FNM be considered as an adjunct. In addition to exposing the nerve during dissection, these dehiscences can allow local anesthetics to inadvertently cause a temporary chemical paralysis of the nerve, *rendering monitoring useless*.

Within the parotid gland, the facial nerve divides into a plexus of five major extracranial branches comprising the parotid plexus or *pes anserinus* (“goose’s foot”). Following ramification at the *pes anserinus*, the nerve branches become progressively more delicate as they extend peripherally. Four-channel monitoring is advised to optimize electrical mapping during parotidectomy.

ANESTHETIC CONSIDERATIONS OF FNM

General anesthetic and amnestic protocols or agents have no clinically significant effect on the sensitivity or specificity of facial nerve neuromonitoring during surgical procedures as the neurophysiologic monitoring circuit for FNM only involves the facial nerve distal of the brainstem, neuromuscular junction, and facial musculature. In addition, derangements in systemic homeostasis such as changes in blood pressure, oxygen delivery, and body temperature will profoundly affect the patient before the neuromonitoring circuit is affected to a significant degree.

One way the management of a general anesthetic can affect FNM occurs when the level of anesthesia becomes “light” and the patient begins to emerge from anesthesia. This emergence is associated with profound increases in facial muscle activity that will manifest as polymorphic spikes on the EMG monitor that increase in frequency and become continuous unless anesthesia is deepened (Fig. 2).

Local anesthetics and neuromuscular blocking (NMB) agents may interfere with or prevent FNM. A local anesthetic may track to the proximal portion of the extracranial facial nerve and block nerve conduction, thus rendering monitoring useless. Using NMB facilitates airway management and decreases airway-related morbidity, thus NMB agents are frequently employed during induction of general anesthesia. However, any residual neuromuscular blockade during the monitoring phase of the case has the potential to decrease the sensitivity of FNM as discussed in detail in a separate section later.

Two strategies may achieve the goals of improved airway management and absence of neuromuscular blockade in the monitoring period. The first uses the drug succinylcholine and the second strategy uses a reduced dose of a non-depolarizing NMB followed by reversal of residual neuromuscular blockade before FNM begins. Because adequate neuromuscular transmission is critical to the success and sensitivity of FNM, it may be prudent to include the approach to management of neuromuscular blockade in the preoperative briefing. Likewise, prudence would dictate the documentation of return of neuromuscular function on the anesthesia record prior to surgical interventions that pose a risk to the facial nerve.

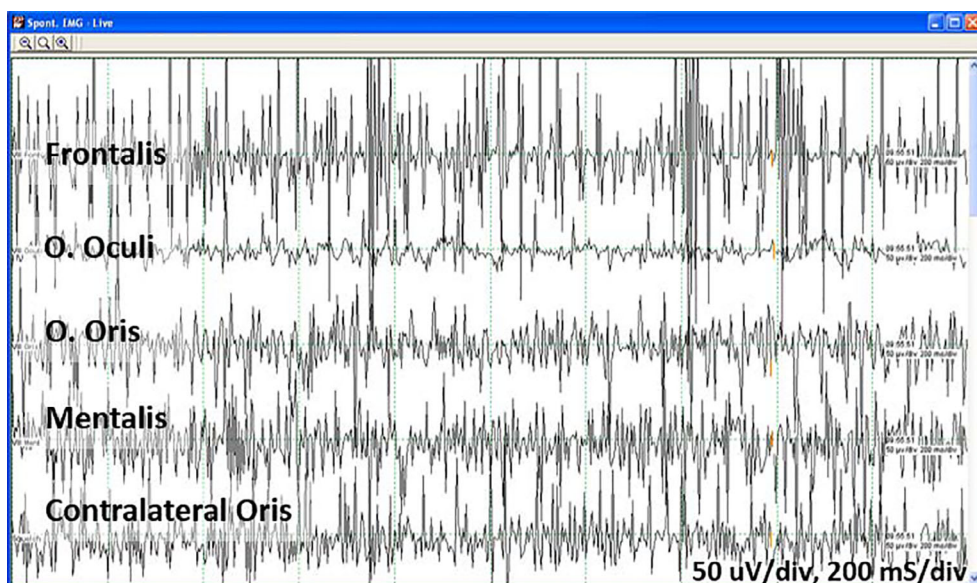


Fig. 2. Generalized irregular pattern of EMG consistent with light anesthesia.

Use of Succinylcholine

Succinylcholine is the only available depolarizing NMB agent. Succinylcholine is short-acting with a half-life of less than 1 minute and a clinical duration of action of 9 to 12 minutes. The termination of succinylcholine's effect is due to metabolism by an enzyme in plasma called pseudocholinesterase. There are many diseases (e.g., advanced liver disease) that prolong the effect of succinylcholine by decreasing the concentration of the pseudocholinesterase enzyme, but rarely to an extent that more than doubles duration of action. The Achilles heel of the strategy to use succinylcholine to facilitate FNM is a patient with a genetically determined type of pseudocholinesterase unable to effectively metabolize succinylcholine. Such a patient is entirely clinically asymptomatic, that is, without prior exposure to succinylcholine, he/she is completely unaware of the pseudocholinesterase deficiency. In these patients, the half-life of succinylcholine will extend to 4 to 9 hours and FNM will be impossible for the duration of succinylcholine action. Pseudocholinesterase deficiency occurs in 1 of every 3000 to 5000 people. It is more common in certain populations such as people of Persian Jewish or Alaskan Native ancestry. Although relatively rare, this argues for the use of low doses of non-depolarizing NMB agents rather than succinylcholine.

Use of NMB Reversal

The second strategy uses a reduced dose of a non-depolarizing NMB agent followed by reversal of residual neuromuscular blockade before FNM begins. Non-depolarizing NMB agents suitable for this strategy such as rocuronium, vecuronium, atracurium, or cis-atracurium have an intermediate duration of action with a clinical duration of action of 20 to 50 minutes. To be reversible after 20 to 25 minutes, the dosage needs to be restricted to an

ED95, that is, 0.3, 0.04, 0.2, and 0.04 mg/kg for rocuronium, vecuronium, atracurium, and cis-atracurium, respectively.⁴³ There are two pharmacological approaches for reversing non-depolarizing neuromuscular blockade. The first relies on functional antagonism and uses a cholinesterase inhibitor such as neostigmine and an anticholinergic such as glycopyrrolate and works for all available non-depolarizing NMB agents (to ensure intact neuromuscular transmission, reversal with neostigmine/glycopyrrolate should only be done once the train-of-four [TOF] has returned to four twitches). The second pharmacological approach relies on binding and thus inactivating the non-depolarizing NMB agent by encasing it in the cyclodextrin, sugammadex.⁴⁴ The binding cavity is designed to accommodate rocuronium or vecuronium and thus will not work if another non-depolarizing NMB agent has been used. One advantage of reversal with sugammadex is that sugammadex use allows for the reversal of more profound neuromuscular blockade (e.g., a TOF of only one of four twitches).⁴⁵

NMB and FNM: Physiology and Clinical Practice

Partial neuromuscular blockade during FNM requires identification and possible intervention. Except under investigational conditions, however, anesthesiologists rarely monitor neuromuscular blockade quantitatively due to a lack of suitable clinical equipment. Instead, they rely on a visual or tactile assessment of twitch counts. The gold standard for assessing neuromuscular transmission is the measurement of muscle force generated in response to electrical stimulation. The stimulation is a train of four stimuli at 2 Hz. The force of the first and fourth muscle contraction is measured and expressed as a ratio; a ratio of 0.9 represents full strength. IONM equipment allows for measurement of the amplitude of the CMAP in response to a TOF

BOX 1. Pitfalls IN FNM - Case 1

A 57-year-old man presents for a superficial parotidectomy for a pleomorphic adenoma. He is of normal weight and has a reassuring airway examination. His medical history is noteworthy for alcohol use disorder and severe gastroesophageal reflux disease. On the day of the procedure, rapid-sequence induction anesthesia is performed with rocuronium (1.2 mg/kg) to minimize aspiration risk. Facial nerve monitoring (FNM) is set up using a dedicated monitor. A “tap” test results in prompt auditory feedback from the monitor. One hour later, after exposure of the parotid gland, stimulation of the main trunk of the facial nerve posterior to the parotid gland fails to elicit a compound muscle action potential (CMAP) of the facial muscles, despite evidence for the delivery of an adequate stimulating current. Evaluation of a train-of-four (TOF) response at the adductor pollicis shows a complete absence of twitches.

The Issue

High doses of non-depolarizing neuromuscular blocking agents may result in prolonged neuromuscular blockade that may interfere with FNM. Some of the interindividual variability in the duration of action of a bolus dose of neuromuscular blocking agents is difficult to predict based on patient characteristics. This may be most frequently seen with rocuronium because its rapid onset of action makes it the non-depolarizing neuromuscular blocking agent of choice for rapid-sequence induction and because its recommended dose range includes higher doses than those recommended for other non-depolarizing neuromuscular blocking agents.

In the scenario described above, reversal with functional antagonists such as neostigmine will not normalize neuromuscular transmission and restore the sensitivity of FNM to full, particularly in passive mode. A full reversal of profound neuromuscular blockade with neostigmine typically takes 30 to 60 minutes to normalize neuromuscular transmission, with one-quarter of patients still showing significant residual weakness 1 hour after reversal.⁷⁸ In contrast, a high dose of sugammadex (≥ 4 mg/kg) restores neuromuscular transmission typically within 5 minutes.

Lesson Learned

The choice and dose of muscle relaxant for airway management should strike a balance between competing medical objectives such as rapid airway management at minimal morbidity and the need for FNM. Neuromuscular transmission monitoring is obligatory when FNM is planned. Newer reversal agents offer advantages over functional antagonists in reversing profound neuromuscular blockade.

stimulation paradigm. The resulting amplitude ratio approximates the gold standard force measurement and is a much better quantitative assessment of neuromuscular function than the visual or tactile assessment commonly used by anesthesiologists. Documentation of intact neuromuscular function should be systematically incorporated into the set-up for FNM before taking any steps that would put the facial nerve at risk.

Muscles differ in their sensitivity to neuromuscular blockade. In general, working muscles such as the diaphragm and the muscles of the airway show a rapid onset of neuromuscular blockade because of rapid drug delivery due to the increased blood flow required to sustain that work. The degree of neuromuscular blockade, that is, the extent to which a given muscle is weakened by a stable concentration of NMB agent also differs between muscles.^{46,47} In general, muscles that do all-or-none bulk movements such as the diaphragm or the orbicularis oculi (blink reflex) are relatively resistant to neuromuscular blockade, whereas muscles that do finely graded adjustments are more susceptible, for example, the laryngeal muscles that modulate the pitch of the voice, extraocular muscles, and hand muscles.^{48,49} A practical consequence of this physiology is that compared to a given TOF ratio maintained at the adductor pollicis (the typical

monitoring site used by anesthesiologists), the orbicularis oculi will show a lesser degree of neuromuscular blockade.

This differential sensitivity is a partial explanation for reports of successful FNM with direct electrical stimulation at constant levels of partial neuromuscular blockade. Because a constant level of neuromuscular blockade may be difficult to maintain for systemic reasons such as changes in blood volume through blood loss and for practical reasons such as bolus administration of medications through the infusion line of the NMB agent, most anesthesia providers refrain completely from using neuromuscular blockade during the time the facial nerve is at risk during surgical intervention. EMG activity can be recorded in the presence of partial neuromuscular blockade although implications for the sensitivity of different forms of facial nerve EMG monitoring are complex and available data directly addressing the issue are sparse.

The first consequence of residual neuromuscular blockade is a decrease in the amplitude of recorded electrical responses. Typical non-depolarizing NMB agents compete with acetylcholine at the postsynaptic muscle receptors. When present in sufficient concentration within the neuromuscular junction, some muscle fiber depolarization will be prevented despite activation of the

muscle's innervating nerve axon. While for individual muscle fibers, depolarization is an all-or-none phenomenon, facial nerve EMG monitoring typically examines electrical activity from groups of muscle fibers. Thus, as long as sufficient numbers of muscle fibers are within the EMG recording field, recorded amplitudes will decline in a graded manner with increasing degree of neuromuscular blockade as greater numbers of underlying muscle fibers fail to depolarize. At profound levels of neuromuscular blockade or when only few muscle fibers are within the EMG recording field, no response may be recorded despite activation of the facial nerve.

The second consequence of residual neuromuscular blockade may be a qualitatively different response to repeated depolarization of motor nerves. When repetitive axonal depolarization occurs, there is reduced presynaptic release of acetylcholine upon each subsequent depolarization through the first four or five depolarizations after which release typically plateaus. Because curare-like blocking agents competitively block postsynaptic receptors, the lower concentration of acetylcholine allows a greater impact of the blocking agent to manifest as stimulation continues and therefore fewer muscle fibers successfully reach their threshold for depolarization. This well-known phenomenon is manifested as the "fade" or "decremented response" following repetitive nerve stimulation.

Stimulated EMG is easier to place within the framework of the altered neuromuscular physiology caused by partial neuromuscular blockade. When a single electrical stimulation of the facial nerve is delivered in the presence of a qualitatively low level of neuromuscular blockade, a CMAP is likely to result with slightly lower amplitude than that obtained in the absence of neuromuscular blockade but otherwise no appreciable difference in response morphology or stimulation threshold. This simple picture of a quantitative decrement in response becomes complicated as levels of neuromuscular blockade rise. Because nerve stimulation is usually done at or near the threshold for a response, CMAPs are often of initial small amplitude and may simply decrease below a detectable amplitude in the presence of neuromuscular blockade. Even with a relatively high stimulation level of 1 mA directly on the nerve, one study showed that 10% of patients did not have a recordable response when the neuromuscular blockade was sufficient to decrease the hypotenar twitch amplitude to 25% of baseline twitch amplitude.⁵⁰ If, on the other hand, the stimulation intensity is raised to restore CMAP amplitude in the face of residual neuromuscular blockade, the measured threshold would rise. Studies examining facial nerve CMAPs under conditions of high levels of neuromuscular blockade demonstrate these effects showing markedly increased stimulation thresholds and reduced CMAP amplitudes.^{51,52} Unfortunately, there is no reasonable expectation that threshold increases will be linear or otherwise predictable within individual patients and thus no clear compensatory stimulation strategy is available. Because an increasing threshold may also indicate new facial nerve dysfunction and not just the presence of qualitatively high levels of neuromuscular blockade, threshold determinations would fail as a prognostic tool to assess

facial nerve function. Furthermore, the facial nerve is often stimulated repetitively, which will amplify the effects noted above due to the resultant "fade" in CMAP responses. These effects may also be amplified with the use of automated detection systems, as amplitudes may fall below preset levels for detection, whereas direct visualization may still identify reproduced responses with lower amplitude.

The effect of neuromuscular blockade on the sensitivity of monitoring free-running facial EMG is even less clear because in many situations when facial EMG provides useful feedback to the surgeon, the evoked motor unit potentials (MUPs) represent a less robust response than a stimulated CMAP. As with a CMAP response, one would expect only minor amplitude effects on a single burst of EMG activity with qualitatively low levels of neuromuscular blockade. However, free-run EMG must frequently assess single MUPs, which, as a rule, are generated by far fewer muscle fibers than a stimulated CMAP or even a burst potential. With fewer muscle fibers and their associated all-or-none response to neuromuscular blockade, there is greater likelihood of a complete dropout of MUPs as neuromuscular blockade increases, depending on their innervation ratio, fiber number within the recorded field, and the cutoff amplitude for detection of a response. Furthermore, examination of single MUPs occurs when they are depolarizing repetitively and thus the resultant "fade" will increase the chance for a complete dropout. As a result, the character of the EMG and the rates of activity could be affected by the dropout and fade in of MUPs. Finally, the reduced tension generated by the muscle under neuromuscular blockade might alter feedback mechanisms that impact the propensity of EMG activity. No studies have attempted to define the sensitivity of monitoring free-running facial EMG in the presence of neuromuscular blockade. Because of the concerns outlined above, in clinical practice, most centers refrain completely from using neuromuscular blockade during the time the facial nerve is at risk from surgery.

TECHNICAL CONSIDERATIONS OF FNM

Preparation for Monitoring

Because the technologist is part of the operating room team, he/she must become aware of and participate in the patient safety initiatives recommended by the Institute of Medicine and the Joint Commission, including the aforementioned World Health Organization's Surgical Safety Checklist. Although this checklist has often been modified for each hospital's local needs, there are core elements that must be included such as confirming surgical side, site, patient identity, consent, prophylactic antibiotics, and a functioning pulse oximeter. Before skin incision, the entire team confirms these elements in an oral recitation. Any possible variances or concerns are addressed before proceeding.

Preoperatively, the technologist must confirm the monitoring plan with the surgeon, including the site and side of surgery. The technologist must confirm the site again just prior to placing electrodes.

BOX 2. Pitfalls in FNM - Case 2

A 46-year-old patient presents for mastoidectomy for a cholesteatoma. The patient is otherwise healthy and has a reassuring airway examination. The surgeon requests succinylcholine as a muscle relaxant for airway management because he intends to monitor facial nerve function during the procedure. Induction and intubation are accomplished without difficulties. Fifteen minutes later, as the facial nerve monitor is being placed, the anesthesiologist reports that there are still no twitches apparent at the adductor pollicis on TOF stimulation of the ulnar nerve. Stimulation of the facial nerve through the skin over the styloid process fails to elicit a CMAP of the facial muscles even though the stimulator output is maximal; a “tap” test is positive and the monitor confirms current delivery. The case is cancelled and the patient is transferred to the intensive care unit for respiratory support with a presumed diagnosis of pseudocholinesterase deficiency.

The Issue

Succinylcholine is metabolized by enzymes found in the plasma. Patients carrying genetic variants that significantly reduce the activity of these enzymes are asymptomatic and typically diagnosed at first exposure to succinylcholine. The failure to metabolize succinylcholine prolongs the duration of neuromuscular blockade from 5 to 10 minutes in normal patients, to 4 to 6 hours in homozygous carriers.

Lessons Learned

Checking for reversal of neuromuscular blockade is essential for every facial monitoring case and requires assessment by TOF, stimulation of another preplanned muscle used for control, and/or early stimulation of the facial nerve using volume-conducted mapping techniques. Although pseudocholinesterase deficiency is rare (1:3000 in North America), it is not apparent in everyday life and is typically not screened for by current pharmacogenetic testing.

Marking the incision has only recently become a standard. Prior attempts to do so were ridiculed as needless. One of the first suggestions in 1984 was described by Kartush wherein not only did the surgeon mark the side in consultation with the patient preoperatively, but an erasable marker board was also placed in the operating room where surgeon, nurse, and resident/fellow all had to independently document their confirmation of the correct side after discussion with patient and confirming the consent^{53,54} (Fig. 3, A and B). Now surgical site marking has

become mandatory, the technologist should *confirm the surgical marking before placing electrodes*.

The technologist must also consult with the anesthesiologist regarding considerations for monitoring, as detailed above. If additional monitoring modalities will be used, those must be discussed as well.

The technologist will be responsible for placing and securing the recording and stimulating electrodes, connecting them to the monitoring system, and verifying adequate electrode impedances. Electrode placement should commence following taping of the patient's eyes to avoid accidental corneal abrasion from the electrode needle. Similarly, electrodes should be removed at the end of the procedure *before* the tape is removed from the eyes. When placing electrodes, the technologist should use standard precautions and aseptic technique. Gloves are worn and alcohol skin preparation precedes the insertion of subdermal needle electrodes. Electrode wires should be securely taped to the skin, avoiding placing tape over hair or eyebrows, and the wires directed away from the surgical site. The use of twisted-pair recording electrodes reduces extraneous electrical interference. Individual electrode impedance should be tested and confirmed to be less than 5000 ohms, whereas inter-electrode impedance should be less than 1000 ohms.

Documenting the number of electrodes placed on the patient may help to avoid missing electrodes during removal. Careful removal of the needles by holding the wires and peeling the tape away followed by immediate disposal into a sharp container will minimize accidental needle sticks. Pressure applied to the skin with gauze during needle removal will help avoid bleeding and ecchymoses.



Fig. 3. Surgical safety process. (A) Surgical site marking by the surgeon in consultation with the patient prior to sedation. (B) Erasable marker board design used at Michigan Ear Institute. Surgeon, nurse, and resident/fellow must independently confirm surgical site and then document. The board is then checked at time-out and again just before prepping.

FNM Protocol and Checklist

The following protocol elements are recommended to ensure optimal conditions for FNM:

First, it should be established that the neuromuscular blockade used to facilitate intubation will have worn off or be reversed prior to critical surgical events. A quantitative measure of neuromuscular block should be performed and documented by the anesthesiologist, the monitoring technologist, or preferably by both. For the technologist, a quantitative measure of neuromuscular blockade consists of a supramaximal TOF stimulus (four stimuli at 2 Hz) of a motor nerve and recording of the resultant CMAPs. Absence of neuromuscular blockade is confirmed if the amplitude of the first and last CMAP are within 10% of each other. Any residual blockade should be communicated to the surgeon before critical surgical events begin. Repeat confirmation of TOF values may be advisable, for example, with a change in anesthesia staff or during troubleshooting a loss or absence of expected facial response to stimulation.

Second, the surgeon should be cautious about injecting local anesthetic such as lidocaine or bupivacaine—especially avoiding the stylomastoid foramen. Local anesthetics, whether directly at the injection site or via drug entry into the middle ear where a dehiscent facial nerve may be exposed, will cause a conduction block of the facial nerve. Injection in the external auditory canal in the presence of a tympanic membrane perforation should prompt additional caution. An inadvertent nerve block will prevent spontaneous and stimulus-evoked EMG responses, thereby creating a dangerous false sense of security. As an alternative, the surgeon could use 1:100,000 epinephrine without lidocaine or any other anesthetic agent; the resulting vasoconstriction will minimize bleeding without affecting facial nerve function.

Third, care should be taken to place subdermal electrodes accurately and safely within the appropriate facial muscles, keeping 5 to 10 mm of spacing between electrodes. Intracranial procedures should be monitored minimally using the orbicularis oculi (electrodes placed near the eyebrow) and orbicularis oris (electrodes placed in the nasolabial groove) because the threat to the intracranial and intratemporal facial nerve is typically global, that is, through stretch or compression, and not confined to a specific facial branch. Therefore, monitoring orbicularis oris and orbicularis oculi captures the aspects of facial nerve function most important to the facial nerve outcome of intracranial surgeries. Additional muscle sites, frontalis and mentalis, should be considered for parotid surgery to help identify distal individual facial nerve branches. Electrodes should be secured with tape and wires drawn away from the sterile field and avoiding trauma to the patient's eyes. An anodal stimulating electrode should be placed on the sternum or shoulder. The ground electrode should be placed similarly and positioned between the anode and surgical site.

Fourth, electrode contact should be confirmed with an impedance check, ensuring that each electrode has an impedance of less than 5000 ohms and inter-electrode impedances of less than 1000 ohms. Any electrodes with

impedances outside this range should be inspected for placement and corrected or replaced.

Fifth, a brief “tap test” should be performed to further establish the continuity of the electrode to the recording system by tapping on the electrode sites while viewing or listening for the electrical artifact that will be produced. The absence of the expected tap-induced artifact will prompt troubleshooting of the electrode connections, amplifier, recording parameters, or audio functions of the monitoring system before proceeding. Note, however, that the tap test *does not confirm proper placement of electrodes nor does it provide any information about nerve or neuromuscular function*. Consequently, although the test has value, it is not a substitute for assessing neuromuscular blockade or performing intraoperative electrical stimulation.

Sixth, once the surgeon has begun exposure, the stimulating probe should be verified as positively delivering current. A monitoring device that measures the stimulus current and displays the information visually or through an auditory signal will confirm *current flow* when the stimulating probe is placed in contact with soft tissue. If the monitoring device has no such confirmation of current flow, then stimulation can only be verified by muscle tissue stimulation that causes local contraction or by facial nerve stimulation with a resultant CMAP.

Seventh, at an early point in the surgery (i.e., prior to any surgical manipulations), the surgeon should stimulate the facial nerve directly or through bone or other tissue. This step will establish a *baseline* response that confirms the functioning of the entire monitoring circuit: stimulus delivery, nerve and neuromuscular conduction, electrode connection, and recording system. A very low stimulus intensity of 0.05 mA intracranially will typically produce a CMAP when directly in contact with normal facial nerve, whereas higher intensities will be required when stimulating through other tissue such as bone, cholesteatoma, or tumor where levels of 0.2 to 1 mA are commonly used. Frequent stimulation of the facial nerve using near-threshold stimulation is recommended thereafter to assess the location and integrity of the nerve. An absence of any response to baseline stimulation will require checking the level of neuromuscular blockade and confirming adequate current delivery through the stimulator. The surgeon should also consider the possibility of a nerve block from local anesthetic.

These fundamental steps are included in the FNM protocol checklist shown in Table II.⁵⁴ This concise checklist has been refined and vetted over decades and is useful for both surgeon-directed and -assisted monitoring. Audio and video files of proper monitoring setup and typical EMG responses are available in Appendix S3, in the **online version** of this article.

Technological Considerations

Electrode Placement. Bipolar percutaneous parallel placement of two standard 13-mm or similar length subdermal needle electrodes is recommended for facial EMG recordings with a spacing of 5 to 10 mm. Surface electrodes may be insufficiently sensitive to detect

TABLE II.

Facial Nerve Monitoring Protocol Checklist

Based on the Kartush Facial Nerve Monitoring Protocol. Used with permission

Steps	Considerations	Check box
1	Consult with surgeon regarding procedure	☐
	- Avoid local anesthesia near the facial nerve	
2	Consult with anesthesia to avoid long-acting neuromuscular blockade	☐
	- Full return of function upon incision	
3	Verify absence of neuromuscular blockade via either:	☐
	- Train of Four	
	- Transcutaneous facial nerves stimulation	
	- Facial nerves stimulation within the operative field	
4	Assure the monitor's loudspeaker is audible	☐
	- Sufficient loudness to be heard over the ambient noise of the operating room	
5	Check recording electrode impedance	☐
6	Verify performance of recording electrodes ability to record EMG activity	☐
	- "Tap test"	
	- Alternate validation of system recording	
7	Verify stimulus current flow through soft tissue	☐
8	Obtain a baseline facial nerve response to electrical stimulation	☐
9	Map the location of the facial nerve with electrical stimulation	☐
10	Selectively use the most advantageous stimulation technique	☐
	- Flush tip monopolar stimulator	
	- Concentric bipolar stimulator	
	- Electrified surgical dissection instruments	
11	Titrate stimulation current intensity	☐
	- Based on nerve location and surrounding tissue in consideration of bone, soft tissue, blood and cerebrospinal fluid	
12	Obtain a final facial nerve response to stimulation prior to closure	☐
13	Provide adequate documentation (monitoring and operative)	☐
	- Confirmation of protocol (and adherence thereof)	
	- Highlight key events	

spontaneous EMG activity.⁵⁵ Bipolar recordings offer greater specificity of spontaneous and evoked EMG responses from the local muscle while attenuating unrelated far-field EMG responses as well as volume-

conducted artifact via common-mode rejection at the differential amplifier.⁵⁶ Differential-linked muscle recordings (e.g., orbicularis oculi-orbicularis oris) are sensitive to EMG activity in both muscle groups and

therefore achieve more with a single channel; however, this type of recording will be much more susceptible to artifact in addition to EMG potentials from adjacent muscles.⁵⁶ A minimal recording set-up for monitoring the proximal facial nerve (e.g., acoustic neuroma, tympanomastoidectomy) should include bipolar electrode pairs placed in the orbicularis oculi and orbicularis oris. The use of a standard color code (for example, “blue eyes” and “red lips”) helps to maintain the consistency of set-up and reduce errors (Fig. 4). See Figure 5 as an example of a two-channel facial nerve

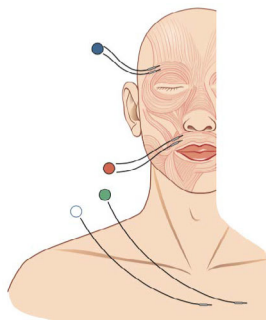


Fig. 4. Electrode placement and suggested color scheme for facial nerve monitoring.

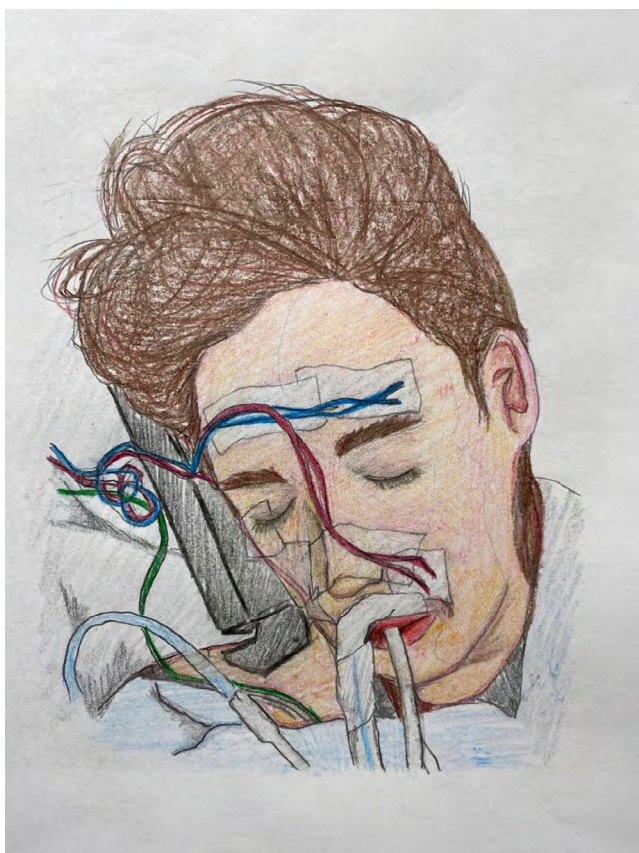


Fig. 5. Bipolar subdermal needle placements at orbicularis oculi and orbicularis oris for intracranial facial procedures. Drawn by Zoe Rice.

recording set-up (note that all of the electrodes have not yet been secured with tape in this depiction). The recording sites must be placed ipsilateral to the site of surgery following proper confirmation of side.

Rampp et al. note that although two-channel monitoring has been the standard for intracranial surgery, increasing the number of channels (muscle sites on the face) can increase the sensitivity to high-frequency A-train activity, which in turn may be helpful in predicting postoperative function.⁵⁷

For procedures involving the facial nerve through the parotid gland, a more extensive set of EMG recording channels is recommended to represent innervation of each of the main facial branches. Bipolar EMG recordings are recommended from frontalis, orbicularis oculi, orbicularis oris, and mentalis muscles. Figure 6 shows a representative set-up of bipolar subdermal needle placements in preparation for a parotidectomy. The stimulating anode



Fig. 6. Bipolar needle set-up for four-channel facial nerve EMG used in parotid surgery (frontalis, orbicularis oculi, orbicularis oris, and mentalis muscles).



Fig. 7. Placement of green ground electrode and white stimulating anode at the sternum.

BOX 3. Pitfalls in FNM - Case 3

A 49-year-old woman is brought to the operating room for resection of a 2-cm left acoustic neuroma via a trans-labyrinthine approach. Following completion of the Surgical Safety Checklist by the entire surgical team, the patient is intubated and the operating room table is turned 180° per protocol to allow maximal access to the patient's head away from the anesthesia equipment. Preparation of the operative site is initiated by the intraoperative neuromonitoring technologist who places bipolar recording electrodes in the facial musculature followed by shaving the patient's hair and sterile preparation by the senior surgical resident. At this point, the surgeon turns from confirming the left tumor on the MRI scan to see that the technologist and resident have mistakenly inserted electrodes and shaved the opposite right side.

The Issue

The technologist may be the first person to prepare the patient and once a preparation or procedure has begun, other well-meaning individuals will have a much higher chance of perpetuating the error by assuming that proper confirmation of the site was made by the "first touch". The technologist in this actual case claimed afterward that they were misled because "following intubation, anesthesia left the patient with their head tilted to the left thereby exposing the right ear and scalp," which they wrongly assumed was tilted in this direction to intentionally expose the right side for preparation.

Lesson Learned

While a disaster of operating on the wrong side was averted in this case, it was only because of the redundancy of a third person re-checking after the checklist, technologist, and resident had still allowed the error to occur. Each person in the team must do their due diligence and independently re-confirm the side, including checking for the surgical site marking. Given the technologist's role at the start of every case, this is a key step that must never be overlooked. Rotation of the operating room table 180° is common in middle ear, mastoid, and skull base surgery and demands even greater diligence by the entire team to prevent errors.

may be placed on the patient's shoulder or at the sternum, with a ground electrode placed above the anode (Fig. 7).

Whether using a dedicated EMG monitoring system designed specifically for facial or other cranial nerve monitoring or using a multimodality system that is also used concurrently for evoked potential monitoring, there are important equipment features—such as a loudspeaker for EMG and a stimulus delay—that are helpful to optimize feedback to the surgeon regarding facial nerve function.

Recording Parameters, Data Display, and EMG Event Capture. A minimal filter bandpass of 10 to 1000 Hz is suggested for recording EMG potentials. Sensitivity for viewing spontaneous EMG activity may range from 20 to 400 mV/division but may need to be adjusted downward to view large CMAPs during facial nerve stimulation.

Viewing spontaneous or free-run EMG from a variety of sweep lengths has advantages and drawbacks. For example, a long sweep of 2 to 10 seconds allows time to view and consider the patterns of activity that occur; however, the MUPs are quite compressed, and detailed waveforms cannot be appreciated. Thus, differentiating certain EMG activity from artifact may be more difficult. In contrast, viewing EMG with a short sweep of 30 to 200 msec allows for clear identification of artifact versus EMG potentials; however, unless the waveforms are captured and suspended on the screen, the presentation is so brief that details of the waveform can be very difficult to assess. The equipment should offer a choice of long, short, and intermediate timebases for free-run EMG. Alternatively, a display option that allows two windows of the

same data with independent timebases (e.g., a 2-second sweep and a 50-msec sweep) may be ideal.

A waveform capture capability is important because it allows the suspension of EMG waveforms on the screen that meet a certain amplitude criteria or "threshold" that can be set by the user. This feature permits a longer period of time in which the technologist can view the data, and the threshold can also be used to trigger an auditory alert to the surgeon of an event. Automated data archiving of captured EMG activity or triggered data are also useful for documentation and review, although it can lead to large data files. At the least, manual archiving of representative samples of facial activity and triggered responses is recommended.

Avoiding capture of the stimulus artifact during stimulus, tEMG may be desirable—especially if the surgeon is listening for a tone associated with a successful facial CMAP. Thus, a capture delay feature or "stimulus ignore period" that prevents the capture of any signal occurring during the first few milliseconds of the sweep is helpful. Despite this, an excessive-stimulus artifact could still extend beyond the capture delay period and trigger a false response. Therefore, an appropriate capture delay time must be selected depending on the anticipated evoked CMAP latency. It must be late enough to adequately ignore the stimulus, yet early enough to still capture the peak amplitude of the CMAP.

Auditory Functions: Squelch. Auditory feedback has become an essential feature of FNM, particularly in surgeon-directed monitoring systems. Common types of facial EMG activity such as bursts and trains produce

easily distinguished acoustic patterns when played over speakers and they provide instant feedback to surgeons relating the EMG to surgical manipulations.⁵⁸ Some artifacts can also often be differentiated by listening to the free-run EMG. In addition to listening to the raw EMG activity over the speakers, audible tones are frequently used to signal-captured EMG activity, both mechanically and electrically elicited. By assigning a different tone to each recording channel, the surgeon knows immediately which muscle is being activated. Additionally, when stimulating the facial nerve, the tone may be easier for the surgeon to distinguish versus the sound of the EMG response. One caveat, however, is that tones that play based on a capture threshold are not differentiated in terms of EMG versus artifact. This underscores the importance of the raw EMG audio as well as the value of having an experienced individual visually assessing and reporting the data.

A muting or audio squelch function is necessary to prevent electrocautery and perhaps other artifacts from startling the entire operating room team with a sudden loud noise from the speakers. The most common technique is using an induction coil placed around the cautery cables to detect cautery use and immediately signal the monitoring unit to mute the speaker output. With or without muting, the surgeon should be aware that, with current technology, monitoring is not in effect during electrocautery. Consequently, if the nerve is injured mechanically or thermally during cautery,

injury will not be detected. Any suspicion of injury during cautery should be immediately assessed using electrical stimulation proximal to the site of possible injury.

Audio squelch can also be triggered by the software by assigning an unrelated recording channel (i.e., not a facial EMG recording site) as the “squelch channel” such that any unexpected activity occurring in this channel over a set threshold criterion will be assumed to be an artifact (whether cautery related or other artifact) and will trigger muting of the speaker until it returns within the threshold amplitude.⁵⁸ Regardless what form of squelch is used, it must be sensitive enough to mute the speakers before the cautery noise can be heard.

Surgeon-directed FNM devices have a loudspeaker that enables the surgeon to receive immediate real-time feedback, including EMG response tones as well a “current-delivered” tone, during stimulation. In addition to the global squelch feature, stimulus artifact suppression technology is also required to prevent the surgeon from hearing the stimulus artifact at nearly the same time as a possible tEMG response. Loudspeaker volume should be set at the beginning of the procedure to ensure that it is sufficient to be heard above the operating room din. In analogy, note that American Society of Anesthesiologists “Standards for Basic Anesthetic Monitoring” now include requirements that not only must a pulse oximeter be used but that “variable pitch pulse tone and the low threshold alarm shall be audible.”⁵⁹

BOX 4. Pitfalls in FNM - Case 4

A 65-year-old patient was undergoing a left tympanomastoidectomy. Bipolar pairs of subdermal needles were placed at the orbicularis oculi and orbicularis oris muscles. A ground needle was placed at the top of the sternum and a return anode needle placed just distal to the ground. The electrodes were secured with 1-in clear plastic tape. A “tap” test revealed appropriate artifact from each recording channel. TOF testing demonstrated complete reversal of neuromuscular blockade upon exposure. During the mastoidectomy, a monopolar probe was connected to the stimulating cathode and the surgeon requested stimulation at 0.5 mA. Upon stimulation of the mastoid bone, the “capture” tone sounded, indicating that the EMG signal exceeded the amplitude threshold, which was set for 50 mV. Reassured, the surgeon continued to stimulate the surrounding bone only to discover that everywhere the probe touched, the machine indicated a positive response with a tone. Annoyed, the surgeon tested a remote area of tissue far away from the facial nerve and still received a capture tone. Examination of the triggered EMG waveform revealed a tall swooping wave descending into the recording sweep.

The Issue

The capture tone is giving a false result. Its intended function is to indicate only when the EMG trace exceeds its given capture threshold (usually following an initial delay period of a few milliseconds) but it cannot differentiate between an artifact and a true EMG response. In this case, the capture function is detecting a large amplitude stimulus artifact that still exceeds the 50-mV threshold after the capture delay period. The source of the stimulus artifact is likely to be a fault in the stimulus path (e.g., loose or disconnected anode or cathode) and should be investigated. An open circuit in the stimulus path will also result in a failure to stimulate. Positive confirmation of stimulation is an important step in any triggered EMG application.

Lesson Learned

Capture tones as sole feedback to the surgeon can give erroneous results. Waveform analysis will assure the validity of triggered and spontaneous EMG data. Confirmation of results by testing multiple known and unknown structures will also provide assurance that stimulation is giving accurate results or conversely that there is reason to suspect technical errors.

Unfortunately, the expectation of effective auditory feedback can be a challenge for multimodality systems during FNM. For example, when additional cranial motor nerves are being monitored with EMG, detecting which nerve is being irritated or stimulated based on the EMG audio output or even assigned tones can be difficult or impossible without communication from a technologist. Stimulus artifact from electrical stimulators used when simultaneously monitoring SEPs can sometimes generate an annoying repetitive popping sound and falsely trigger EMG capture and tones. Some monitoring systems have the ability to suppress the stimulus artifact or briefly mute the audio during the artifact to prevent it from interfering with audio EMG monitoring. Without that feature, EMG may need to be monitored without the use of audio functions, necessitating even greater diligence of the technologist in communicating EMG events to the surgeon.

Stimulus Verification. Whenever performing stimulus-evoked EMG to search for the presence of the facial nerve or for direct testing of nerve integrity, the verification of stimulus delivery is a simple but crucial step in the interpretation of the results. The absence of an evoked facial CMAP should not be interpreted as meaning the nerve is not nearby until it is established that the stimulator is functioning and the stimulating probe is actually delivering current. Without this verification, failure of the stimulator could falsely lead the surgeon to believe it is safe to proceed with drilling when the facial nerve is perilously close. It is recommended that

the equipment have hardware or software checks to confirm the successful delivery of the desired current by displaying the true measured current or by giving a warning when delivered current does not match what is set on the machine. Unique auditory tones (i.e., current delivered tones) that indicate successful stimulus delivery to the surgeon are a desirable feature, especially when there is no dedicated technologist operating the equipment. In some instances, the surgeon may be able to test the stimulating probe on another nearby nerve (the spinal accessory nerve, for example) or local muscle tissue to confirm stimulus integrity by eliciting a muscle twitch before testing for the facial nerve. Because excitable structures are not always available to the surgeon, equipment verification of stimulus is crucial. The presence of a stimulus artifact in the recording is not sufficient evidence that effective stimulation is occurring. Only a physiologic response (e.g., a muscle twitch) is absolutely unequivocal evidence of successful stimulation. Measured current from the monitoring device is the next best, but still potentially erroneous, means to verify proper stimulation. A short-circuited stimulating cable, for example, will still register proper delivery of current even though the current is not reaching the patient.

Stimulus Considerations: Monopolar Versus Bipolar. Nerve activation for motor nerve conduction studies—also referred to as triggered EMG—may be accomplished using monopolar or bipolar stimulating electrode configurations and each has advantages and disadvantages. Bipolar stimulators offer a more discrete stimulation as the shape of the stimulation field is more focal and directed to a very specific location on the nerve (Fig. 8). On the other hand, monopolar stimulation creates greater current spread through adjacent tissues. It is therefore less discriminating than bipolar stimulation but is superior for mapping the approximate location of the nerve.⁶⁰ For either stimulation application, it is critical to have a dry field, as fluid in the field can cause the current to “shunt” away from the nerve and falsely indicate a safe distance from the nerve by reducing the actual stimulation energy from reaching the nerve.

A monopolar probe is connected to the *cathode* output of the stimulator, while the return electrode or anode is placed on the patient's sternum. To avoid extraneous current flow during stimulation, the shaft of the monopolar probe should be insulated close to the tip. Because of the relative long distance between anode and cathode, current will spread in all directions from the tip of the monopolar probe. As stimulus intensity is increased, the effective current spread increases. This has the advantage of permitting current flow through intervening tissues such as bone or tumor when searching for the facial nerve. On the other hand, current can also spread to other nearby nerve fibers. Thus, it is necessary to *properly titrate the stimulus intensity* to adjust the relative sensitivity and specificity of monopolar stimulation according to the situation. Bencsoter and Kartush discuss that too high a level of monopolar stimulation could result in a false positive due to current spread (“jump”) to a more distal segment of the nerve that may bypass a conduction block near the site of stimulation. As such, they recommend using a bipolar

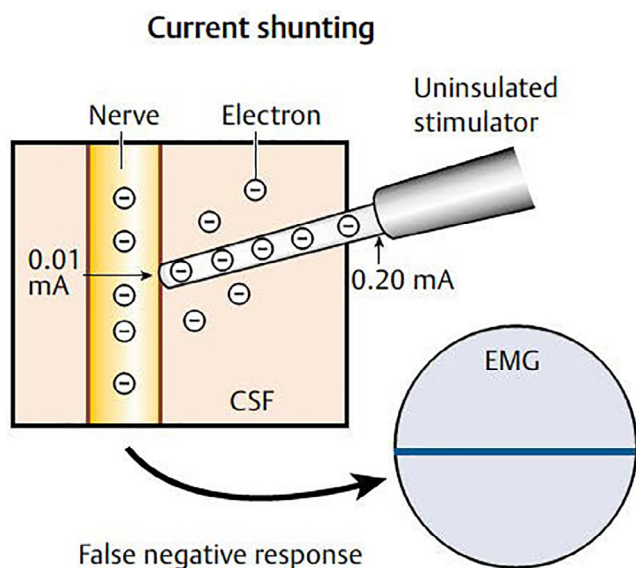


Fig. 8. False-negative response due to current shunting through cerebrospinal fluid (CSF). Contact of the stimulating electrode with anything other than target tissue allows flow of current away from the target. This cartoon models an exposed electrode contact within a CSF bath. In this case, 0.2 mA flows at the base of the exposed electrode contact but the low impedance pathway through the CSF siphons off most of the intended current with only 0.01 mA reaching the nerve surface, which in this case is insufficient to trigger depolarization.

stimulator or “only low-to-moderate current intensities with a fine-tipped monopolar stimulator” when assessing nerve function.⁵

A bipolar probe has the anode and cathode immediately adjacent to each other at the tip of the probe. These can be arranged side by side, or in a concentric configuration with the anode surrounding a central cathode. Because the relative spread of current is limited for a bipolar probe, it is best reserved for direct or near direct nerve stimulation, for example, when attempting to differentiate the facial nerve from adjacent cochlear, vestibular, or trigeminal nerves. In contrast, facial nerve mapping is best performed using monopolar stimulation while titrating the current level.

In tumor-removal surgeries where the normal anatomy and position of the facial nerve may be significantly distorted or other circumstances where the precise location of the nerve is in question, frequent use of triggered EMG is recommended to provide the surgeon with continuous feedback regarding the position and integrity of the facial nerve. Frequent exchanging of surgical instruments with the monopolar probe, however, can be disruptive and time consuming. Dissection and facial nerve testing can occur simultaneously by using stimulating dissecting tools. For example, Kartush stimulating instruments (KSI; Magstim Neurosign Surgical, Carmarthenshire, Wales, UK) are a commercially available set of micro-dissectors that are insulated along the shaft and can be connected to the cathode of the stimulator. In this

instance, the stimulus can be delivered continuously at a low intensity of 0.05 to 0.1 mA while the surgeon dissects tumor away from the nerve and will trigger an evoked EMG response immediately when the surgeon comes in contact or very close proximity to the nerve. The standard method of titrating current for *nerve mapping* is to increase the current until a response is generated, then progressively reduce current as the nerve is approached. This technique both minimizes excess stimulation and allows monopolar mapping to be progressively more discrete. Similarly, the assessment of *nerve integrity* at the end of dissection is based on minimal stimulation threshold (MST). For example, following acoustic tumor removal, the proximal facial nerve is stimulated at low levels, progressively lowering the current to as low as 0.05 mA. Significant elevations in MST may indicate some degree of mild (neuropraxic) or significant neural trauma. The common use and value of MST is confirmed by Huang et al.⁶¹ where EMG response to MST at 0.05 mA accurately predicted better facial nerve outcomes than that of their control group. In contrast, Schmitt et al.⁶² suggested a new method wherein a dropoff from supramaximal stimulation (using distal pre-auricular transcutaneous electrodes) had improved predictability of facial nerve function.

Stimulus Parameters. Repetitive stimulation at 4 Hz using a pulse duration of 100 msec is most common. Stimulus intensity must be adjusted according to the procedure and purpose of stimulation. Direct facial nerve

BOX 5. Pitfalls in FNM - Case 5

A 52-year-old female was undergoing a parotidectomy. Ipsilateral bipolar pairs of subdermal needles were placed at the frontalis, orbicularis oculi, orbicularis oris, and mentalis muscles. In addition, electrodes were placed at the contralateral orbicularis oris to detect bilateral EMG and artifact. Stimulation with a monopolar probe was performed during exposure at 3 mA and a CMAP was recorded that indicated the presence of facial nerve trunks. The tumor proved difficult to remove, with much bleeding in the surgical site. No extraordinary EMG activity was noted. Before closure, the surgeon stimulated again at 3 mA and received a somewhat reduced but reproducible CMAP from multiple channels. Postoperatively, the patient awoke with complete facial nerve paralysis. Postoperative electroneurography identified a loss of nerve conduction at the proximal portion of the facial nerve.

The Issue

The stimulus used to determine the integrity of the facial nerve was not applied appropriately, leading to erroneous interpretation. While intensity as high as 3 mA may be necessary initially when searching for the nerve, once the nerve is exposed, more selective stimulation should be applied (e.g., 0.3–0.5 mA). Proximal facial nerve injury occurred and yet CMAP responses were still present because 1) stimulation was performed distal to the site of injury or 2) because the stimulating current was set at an unnecessarily high level, creating spread of current distally beyond the injury site where nerve activation could occur. Spontaneous EMG monitoring may not have detected the injury if it was a sharp dissection or a possible thermal injury induced during cautery when EMG was obscured by electrical noise.

Lesson Learned

Although spontaneous EMG monitoring can provide some warning about potentially injurious maneuvers with the facial nerve, certain injuries will produce minimal or no EMG activity. Thus, it is important to frequently apply an “active” form of monitoring using triggered EMG during the case to assess the position and function of the nerve—not simply at the beginning and end of the procedure. To properly assess the functional integrity of the nerve, stimulation must occur proximally, progressively titrating the current level down as the nerve is approached.

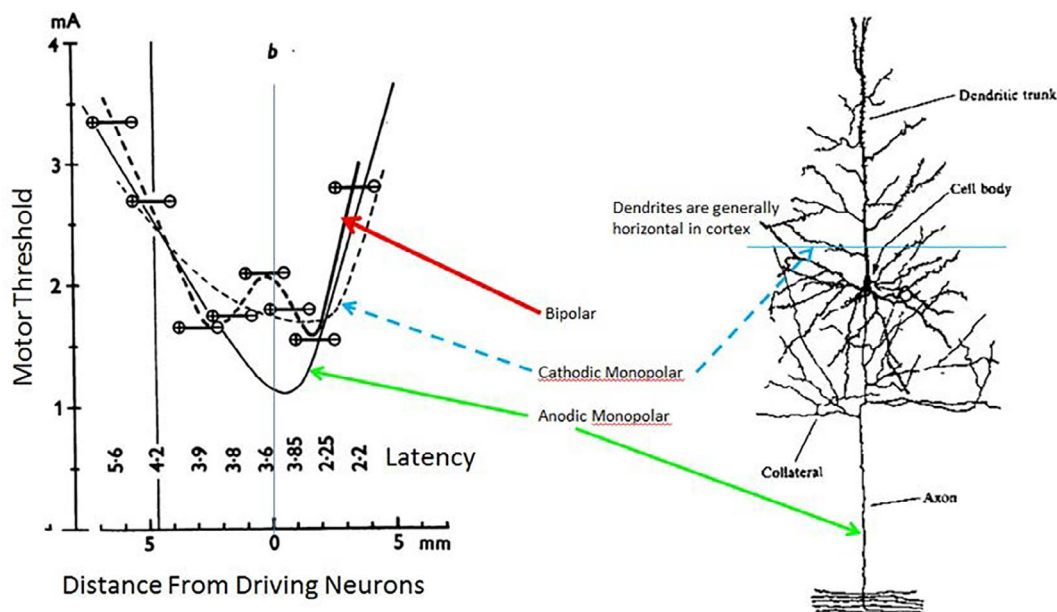


Fig. 9. The electrical field generated by bipolar or anodic monopolar stimulation. Comparison of bipolar and monopolar stimulation in both a “dry” field and “wet” field (current shunting). Note the reduction in signal amplitude when using bipolar stimulation in a “wet” field compared with the monopolar stimulation. Depending upon where one places the anode for the monopolar field, there may be excessive stimulation artifact as shown in this figure.

stimulation can be performed at very low intensities intracranially. Normal facial nerve thresholds near the brainstem will be ≤ 0.1 mA, whereas slightly higher thresholds may be observed extracranially (0.1–0.3 mA). An injured facial nerve may require intensities as high as 1.0 mA to elicit a CMAP.⁴⁰ Stimulus intensities will also be higher when testing for the presence of facial nerve through bone (0.5–2 mA) or soft tissue (0.3–1 mA).

Stimulation may be generated by constant current or constant voltage sources. Constant current stimulation has at least theoretical advantages over constant voltage in that constant current stimulators continually vary the delivered voltage to allow for a constant driving current (current density and charge per phase) that is invariant of the impedance of the surgical field⁶⁰ (Figs. 8 and 9). Although there has been some debate on the advantages of each, most IONM systems today use constant current and therefore we use milliamperes (mA) unless citing a source that used constant voltage.^{63,64} Under routine conditions where the field has been cleared of fluid to prevent current shunting, clinical experience has shown that 1 mA and 1 Volt provide similar neuromuscular responses.

Differentiating evoked facial nerve responses from trigeminal nerve responses may be necessary in some skull-base tumor surgeries. Because of the proximity of trigeminal-innervated muscle to facial nerve EMG recording sites, evoked CMAPs from stimulation of the trigeminal nerve can appear in the facial EMG recording despite the use of closely spaced bipolar electrodes. If one is recording from both facial and trigeminal EMG sites, identifying which nerve is stimulated is based largely on amplitude differences. However, without trigeminal EMG recording, the difference in response can

be based on the latency of the evoked CMAPs. Intracranial stimulation of the facial nerve produces a CMAP with a latency of about 6 to 7 msec, whereas trigeminal stimulation will produce a CMAP with a latency of 3 to 5 msec. Hence, the mnemonic: “7 (facial) is about 7 msec, and 5 (trigeminal) is less than 5 msec.” The data in Figure 10 show a comparison of facial nerve CMAP (Fig. 10A) to trigeminal nerve CMAP (Fig. 10B) using recordings from the orbicularis oculi, the orbicularis oris, and the masseter. Note the differences in amplitude and latency of the responses. When the trigeminal nerve is stimulated, a substantial response is picked up by the oculi and oris channels, yet the very early latency identifies it as trigeminal in origin.

Types of Spontaneous Facial EMG. Spontaneous EMG activity occurs in the form of MUPs presented singly (spikes), as repetitive patterns (spike train), or as roughly synchronized burst potentials. These patterns of activity relate to different forms of neural irritation. For example, thermal changes and various forms of trauma such as stretching or pressure tend to produce train activity, whereas bursts arise from direct mechanical contact with the facial nerve, causing multiple motor units to become briefly and immediately activated. Figures 11 and 12 show examples of burst potentials recorded from different monitoring devices using different timebases.

Train EMG has been largely characterized by a low versus a high frequency of spike presentation. Low-frequency trains have been acoustically described as sounding like popcorn popping, whereas high-frequency trains sound more like an airplane engine and are sometimes referred to as “bomber” potentials. Figure 13 shows

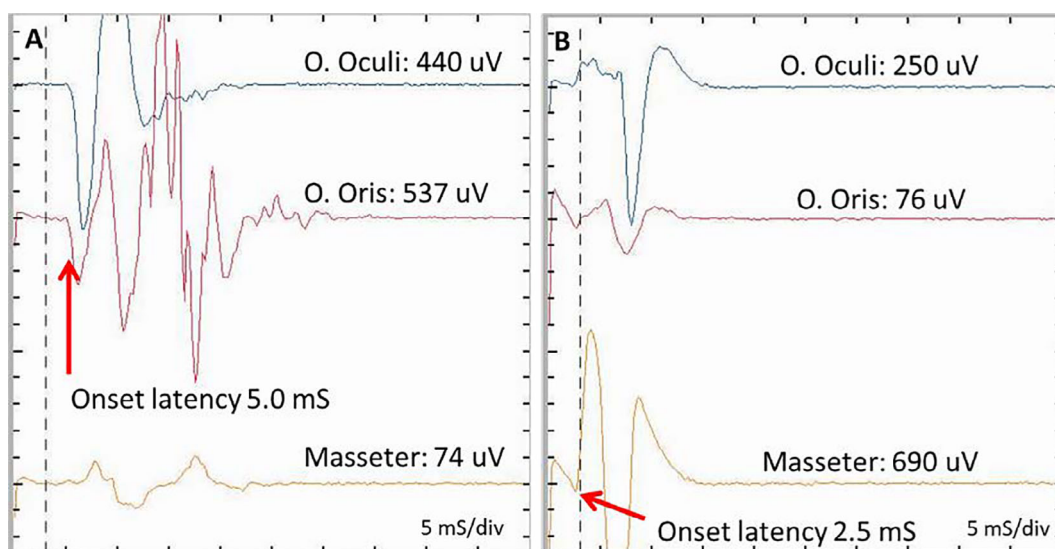


Fig. 10. Differentiating facial from trigeminal responses. (A) Facial nerve stimulation; (B) Trigeminal nerve stimulation. Note that the responses can be seen in all recorded channels but differ in onset latency and waveform detail.

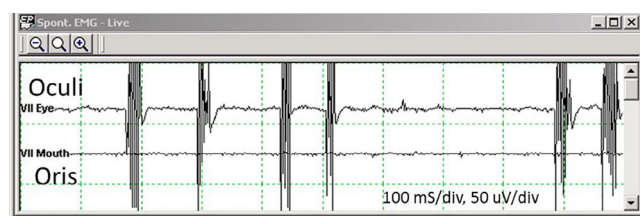


Fig. 11. Burst EMG potentials recorded with a long timebase.

an example of a low-frequency spike train recorded using a 500-msec sweep. Figure 14 is an example of high-frequency train recorded with a 50-msec sweep.

Triggered Facial CMAP. Direct electrical stimulation of a facial nerve via a stimulating probe is a crucial element of FNM, permitting the surgeon to achieve the following:

1. Positively identify the facial nerve fibers and differentiate them from other structures;
2. Map the position and course of the facial nerve in relation to other structures;
3. Establish the functional integrity of the facial nerve.

Suprathreshold facial nerve electrical stimulation activates a synchronized volley of neuronal action potentials that conduct orthodromically from the point of stimulation to the facial muscles to generate a CMAP thereby positively establishing the functional continuity of nerve fibers. Once stimulation has been verified and the absence of neuromuscular blockade has been confirmed via the TOF, the absence of response to stimulation can provide some reassurance that the nerve is not in the near vicinity. Figure 15 shows an example of identifying

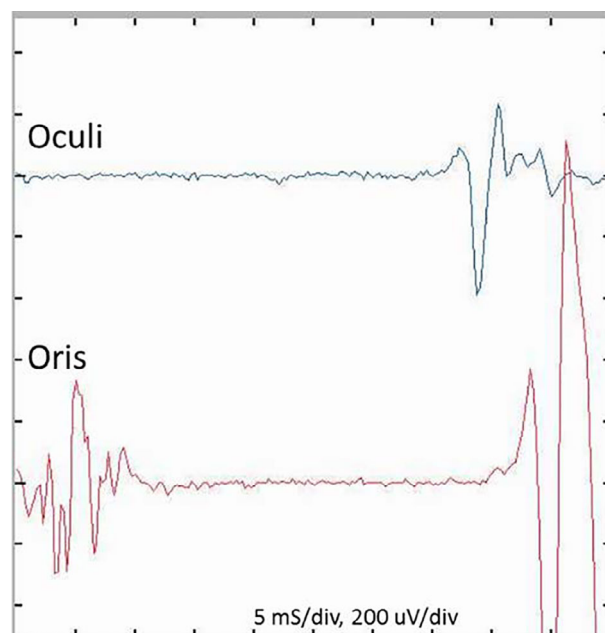


Fig. 12. Burst EMG potentials at higher resolution.

the lower trunk of the facial nerve with stimulation during a parotidectomy. Frequent or continuous awareness of the position of the facial nerve can help the surgeon to avoid injuring the nerve.

Recognizing Artifact. A variety of artifacts can be observed during facial EMG monitoring. One of the chief roles of the technologist is to optimize recording conditions to minimize electrical noise and to further differentiate artifacts from EMG activity. Artifacts can often be identified by their morphological features (i.e., waveform

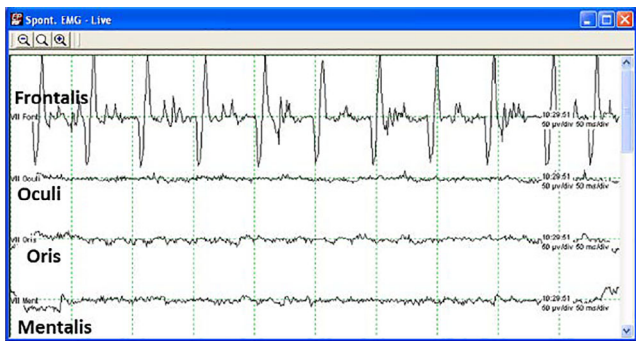


Fig. 13. Example of low frequency "B-train" EMG activity. The timebase is 50 msec/division.

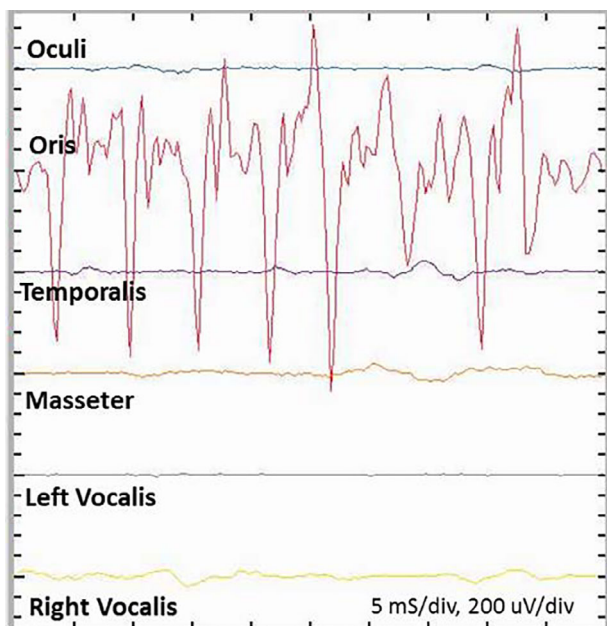


Fig. 14. Example of high frequency "A-train" EMG activity. The timebase is 5 msec/division.

characteristics), pattern of distribution, or by their presentation in correlation with events in the operating room (e.g., the anesthetist disturbing the electrodes wires while checking the endotracheal tube). One of the most common sources of transient artifact is dissimilar metal potential caused by the touching of metal surgical instruments within the surgical site, creating a brief electrical discharge that is volume-conducted to each of the recording sites.⁶⁵ In this instance, the artifact is presented synchronously to each of the recording channels, a feature that distinguishes it from an EMG event. Figure 16A shows an example of this "instrument artifact" observed during acoustic neuroma surgery. Note the precise synchronicity and similarity of waveforms appearing in the three recording sites. The larger amplitude of the artifact in the masseter recording reflects its proximity to the source at

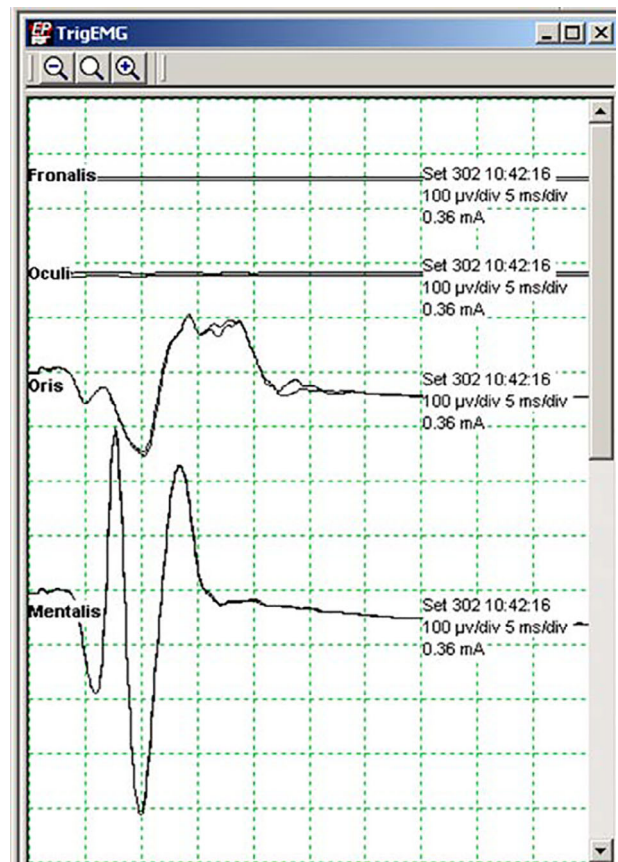


Fig. 15. Lower facial compound muscle action potential recorded during a parotidectomy following monopolar stimulation.

the surgical site. In contrast, Figure 16B shows an example of true EMG potentials from the orbicularis oris.

Line interference from 60-Hz power cables or equipment is another common source of artifact. In some cases, a complex 60-Hz periodic waveform can appear similar in shape to a repetitive high-frequency motor unit discharge. Figure 17 shows an example of an electrical artifact caused by a power cord coming in contact with the recording cable. Although the biphasic waveforms resemble spike train activity, they are occurring at exactly 60 Hz (three repeating cycles indicated by the arrows occurring within 50 msec), and the spikes are perfectly synchronous across the two channels; a characteristic that is not consistent with EMG train activity.

Yet another form of artifact is voluntary facial muscle activity that may occur with unexpected emergence from anesthesia. Facial muscle contractions will yield an irregular pattern of random EMG activity that may progressively increase in amplitude if left uncorrected. Aside from recognizing this pattern of general EMG recruitment, the activity will also occur bilaterally. Thus, having an additional facial EMG channel on the contralateral side as a control may be advantageous in identifying this. An example is given in Figure 2.

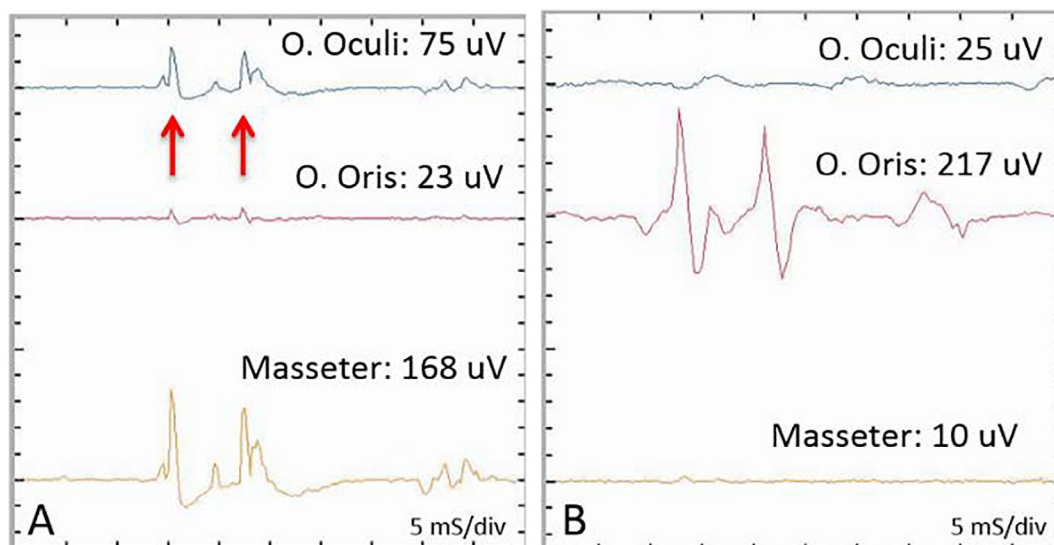


Fig. 16. Differentiating instrument artifact from EMG activity. (A) Example of instrument artifact displaying synchronized potentials across all channels. (B) Example of true EMG activity from orbicularis oris.

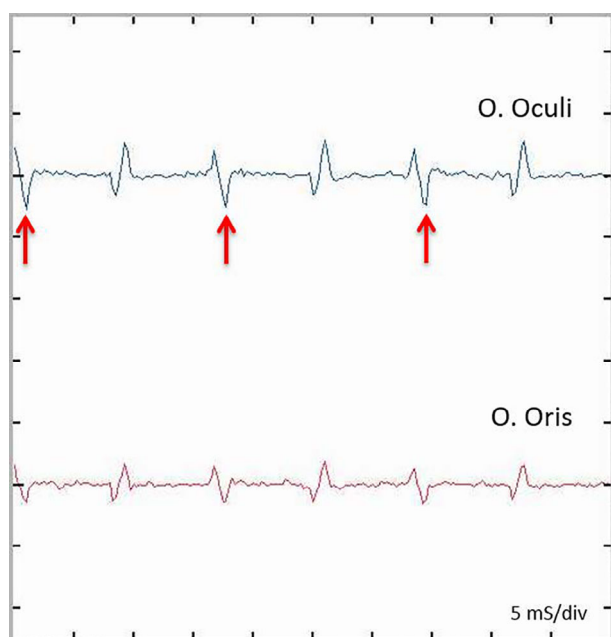


Fig. 17. Periodic noise derived from a power cord touching the recording cable. The arrows indicate the repeating cycles occurring at 60 Hz.

Alternative FNM Techniques

Continuous FNM. Two methods have been proposed for continuous FNM. One involves placing a stimulating electrode at the brainstem REZ and stimulating it repeatedly to elicit EMG responses without pausing dissection. Obviously, such an electrode cannot be placed until the REZ has been localized and confirmed with stimulation, so this method of monitoring is not feasible until considerable exposure and/or tumor

resection has already occurred. This is particularly true for larger tumors and limits its utility, as continuous monitoring cannot be used during the entire period that dissection may place the nerve at risk. Placement of such electrodes is non-trivial and must be accomplished in such a way that the electrode and its connecting wire are not in the way during tumor removal. For these reasons, this method of continuous monitoring has not gained wide acceptance despite its theoretical appeal.

The other method for continuous monitoring is to use *transcranial corticobulbar MEPs*.⁶⁶ Facial MEPs elicited via transcranial electrical stimulation can be monitored from the facial muscles and may permit assessment of facial nerve integrity when large tumors make the facial nerve inaccessible to direct stimulation.⁶⁷ The technique uses multi-pulse stimulation over the contralateral scalp to activate corticobulbar fibers in the same fashion described for corticospinal tract monitoring.⁶⁸ Although a number of studies have demonstrated a correlation between facial MEP deterioration and postoperative facial palsy,^{66,69–71} several technical barriers remain that limit its use. In addition to necessitating strict anesthetic requirements, transcranial stimulation can cause unacceptable movement and stimulus artifact. Stimulating electrodes placed over the facial motor cortex are much closer to the recording sites on the contralateral side of the head than the muscles in the upper and lower extremities used for spinal cord monitoring. Thus, stimulus artifacts are much larger and may be many times larger than EMG responses. Furthermore, the short pathway for corticobulbar tracts produces EMG responses at much shorter latencies than those mediated by longer corticospinal tracts. The combination of large-stimulus artifacts and short-latency responses means that EMG responses may overlap the end of stimulus artifacts, complicating interpretation.

A final problem is that strong transcranial stimulation may activate the facial nerve extracranially at the stylomastoid foramen and thus can elicit a facial EMG response even if the nerve were transected intracranially. This possibility can be controlled by presenting a single stimulus pulse with the same parameters used for MEP train stimuli. If the nerve is being activated extracranially, then a single pulse will elicit an EMG response, but if the site of activation is intracranially (proximal to the facial nerve nucleus), then a single pulse will not be effective and a response will only be evoked by an appropriate pulse train, which overcomes the anesthetic suppression of central synapses by means of temporal summation of excitatory postsynaptic potentials.

These technical issues can be minimized by careful attention to electrode placement (both stimulation and recording), grounding, appropriate stimulus parameters, and optimal anesthetic management. Furthermore, the method is in principle applicable to the monitoring of other cranial motor nerves because the transcranial stimulation is relatively diffuse and non-specific.⁷² However, given these technical difficulties, as well as the movement that typically accompanies transcranial stimulation, transcranial corticobulbar MEP monitoring has yet to gain wide use as a means of monitoring facial nerve integrity. Future developments may bring this promising modality into more mainstream use.

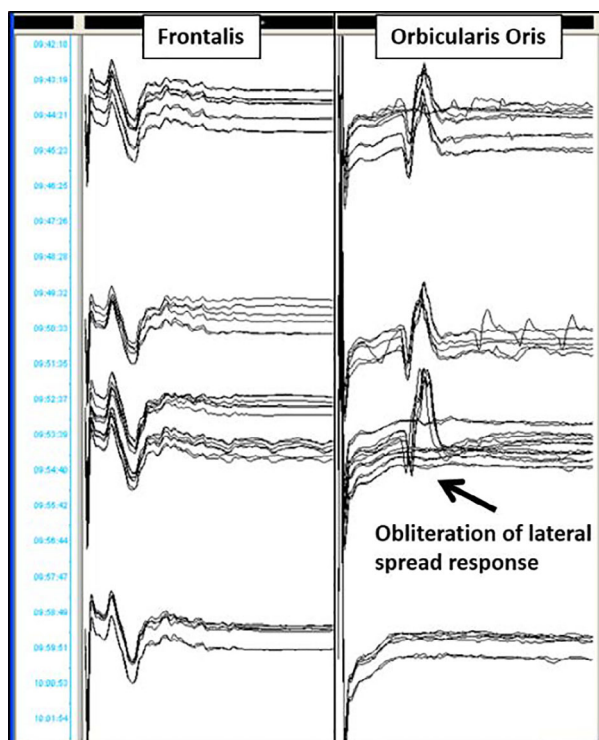


Fig. 18. Lateral spread response to temporal branch stimulation recorded from orbicularis oris. The direct orthodromic response from frontalis is also displayed. Upon decompression of the facial nerve, the abnormal lateral spread response is lost.

Another recently published potentially feasible technique includes the blink reflex, a trigeminal-to-facial nerve reflex that is elicited by a train stimulus to the supraorbital nerve and recorded at the orbicularis oculi.⁷³ However, the value of the blink reflex to monitoring facial nerve integrity has yet to be determined.

Lateral Spread Response During Hemifacial Spasm Surgery. Microvascular decompression is a surgical procedure to treat patients with hemi-facial spasm, a condition in which compression of the facial nerve by an intracranial blood vessel causes continuous involuntary twitching of facial muscles on the affected side. During microvascular decompression, the surgeon identifies the offending vessel and separates the vessel from the facial nerve, placing shredded Teflon or another soft material in between. In addition to using standard FNM in microvascular decompression surgery for hemi-facial spasm, an additional technique developed by Møller and Janetta is performed to determine the adequacy of the decompression.⁷⁴ Unique to patients with hemi-facial spasm, the antidromic volley following the stimulation of a distal branch of the facial nerve activates an orthodromic return volley involving additional, separate facial nerve branches. Thus, stimulation of the temporal branch, for example, will produce a delayed muscle potential from the orbicularis oris. This so-called lateral spread response may occur as a result of a hyperactive facial nucleus or ephaptic transmission; nevertheless, obliteration of the lateral spread response is highly predictive of successful facial nerve decompression.^{75,76} Figure 18 demonstrates the loss of lateral spread response that correlates with facial nerve decompression. A recent study evaluated the blink reflex during microvascular decompression surgery with promising results.⁷⁷

Documentation. A record of the events and communications related to IONM should be generated that includes patient demographics, diagnosis, and procedure information. If a remote supervising neurophysiologist is involved, a pertinent patient history and physical examination should be made available. Preoperative communications with the surgeon and anesthesiologist, confirmation of full recovery from muscle relaxants, surgical stages, reports of remarkable EMG events, and results of stimulation along with the stimulus intensity should be noted in the monitoring log in a contemporaneous fashion. If the supervising neurophysiologist makes an interpretive comment about EMG data, the technologist is responsible for communicating this to the surgeon immediately and logging the communication. The technical report completed by the technologist should include what monitoring took place, a statement about any EMG activity that occurred and the use of stimulation to identify and/or assess the facial nerve, and a statement about the results of stimulation.

FNM Safety Issues. FNM has been an extremely safe endeavor. Nonetheless, there are three factors to consider: 1) risk of burns, 2) risk of needle electrodes in the face, and 3) risk of nerve overstimulation.

The first concern is the risk of electrode-induced burn injuries. As detailed by Netherton and colleagues,^{78–80} this may occur during any monitoring procedure where

electrocautery cables could create a burn by inducing a capacitive coupling current into nearby stimulating or recording electrodes. Operating room personnel should implement standard precautions with the use of electro-surgical devices, with special attention to ensuring proper placement and adherence of the electrocautery-dispersive ground pad. Spills of surgical solution or blood near electrodes and the monitoring device must be immediately addressed to avoid burns from aberrant electrical pathways. Monitoring cables must be widely separated from the cables of the electrosurgical unit and its return electrode (sometimes incorrectly called a ground pad). The surgeon must be aware that a stimulator probe must not be placed near cautery tips. When attempting to cauterize a blood vessel adjacent to the facial nerve, a surgeon might be tempted to have a stimulator in one hand and bipolar cautery tips in the other. However, this is contraindicated, as current may flow from cautery tips back into the stimulator.

Reports of burns from electrodes are not uncommon. A review of the literature identified one article specifically related to burns during FNM.⁸¹ Burns were detected on the face at the needle electrode sites. This occurred with a single Magstim Neurosign monitoring unit that was found to be defective. Following a manufacturing change, no subsequent occurrences were described.

A second safety issue relates to the placement of facial needle electrodes. As noted, prior to inserting needle electrodes, the eyelids should be taped closed. At the end of the procedure, leave the eyes taped until the electrodes have been removed. During electrode placement, identify and avoid subcutaneous blood vessels before inserting. Superficial veins are common in the supraorbital area and personnel should use caution if applying needles near the vermilion border of the lips to avoid the labial artery. Pressure should be applied to the needle for a few moments as it is withdrawn. This pressure duration should be increased in patients with fragile skin and those who are on blood thinners.

The third safety factor relates to stimulus intensity. Decades ago, there was a single report of a stimulator burn to the nerve with a battery-powered, DC disposable unit.⁸² In contrast, there are no reports of injury using modern-day pulsed stimuli generated by constant-current or constant-voltage amplifiers. Typical stimulus settings range from as low as 0.05 mA when testing the threshold of the intracranial nerve to 1 to 2 mA for brief mapping when distant to the nerve or stimulating through bone and soft tissue.

Dedicated facial nerve monitors typically limit their maximal stimulus output to 3 to 5 mA. In this range, with pulsed stimuli and for the short durations used, there are no clinical reports of nerve injury. Kelley and Leonetti⁸³ demonstrated the safety of modern facial nerve stimulus parameters in an animal model. Using a Xomed Nerve Integrity Monitor with monopolar constant-current stimulation, they demonstrated that the canine facial nerve can tolerate at least 50 stimulations at 1 mA and still remain functionally and histologically normal.

Multimodality devices, however, require a much broader range of stimulus intensity to be used in other procedures such as pedicle screw stimulation, SEP, and MEP. This means that greater attention and caution must be directed to ensuring proper stimulus intensity. Direct stimulation of the facial nerve at 0.3 mA is common, as is using 1 to 2 mA to briefly map out a region where the facial nerve may be located. In contrast, a stimulus level of 30 mA that might be proper during pedicle screw stimulation would be improper and potentially unsafe for direct facial nerve stimulation.

INTERPRETATION

Interpretation of Facial Nerve EMG

Correct characterization of EMG patterns provides insight into the real-time physiological and pathophysiological processes in play during a surgical procedure. In the interest of effective communication, it is important that the many participants in the surgical procedure use consistent terminology. The simplest descriptor used is the term “spike.” A spike is a generic term used to describe a short deflection in the free-running EMG baseline that in the usual neuromonitoring setting typically reflects the activation of a single axon leading to depolarization of the muscle fibers that it innervates, its motor unit. Thus, in the typical setting, the electrical activity underlying a spike is the MUP. However, the term “spike” is noncommittal in the sense that spikes may also represent artifact (electrical noise) or may rarely reflect other physiological phenomena (e.g., positive sharp waves, fibrillation potentials, or end plate potentials; Fig. 19). Nevertheless, in almost all cases, EMG monitoring is in place when a surgeon feels motor nerves are at risk and as such, it is axonal depolarization that is of interest. Hence, it is the myogenic manifestation of an axon's depolarization, the MUP, that is our primary monitoring focus. The MUP serves as the building block of the more complex EMG patterns we discuss later.

Mechanical manipulation of a motor nerve may result in the near-simultaneous depolarization of multiple local axons, and the superimposed MUPs from these axons yields a polyphasic “burst” potential (Fig. 20). An axon may continue to depolarize repetitively due to ongoing irritating manipulation or as a lasting irritation from an earlier stimulus. The resulting string of recorded MUPs is termed a “train” of activity.⁵⁸ When the total component spike frequency of burst or train activity exceeds 30 Hz, it is termed “neurotonic”^{84–86} (Fig. 21). If neurotonic trains of activity are produced by the activation of two or more axons, then each of the component MUPs will have its own morphology and individual firing rate; the associated activity has been described as “asynchronous” neurotonic activity and “popcorn” activity.⁵⁸ Finally, MUP activity may increase to the point that it fills the recording channel without a clear return to a flat baseline. A diagnostic electromyographer would typically call this an “interference pattern,” whereas in the neuromonitoring setting, it has been called a “C-train.”⁸⁷

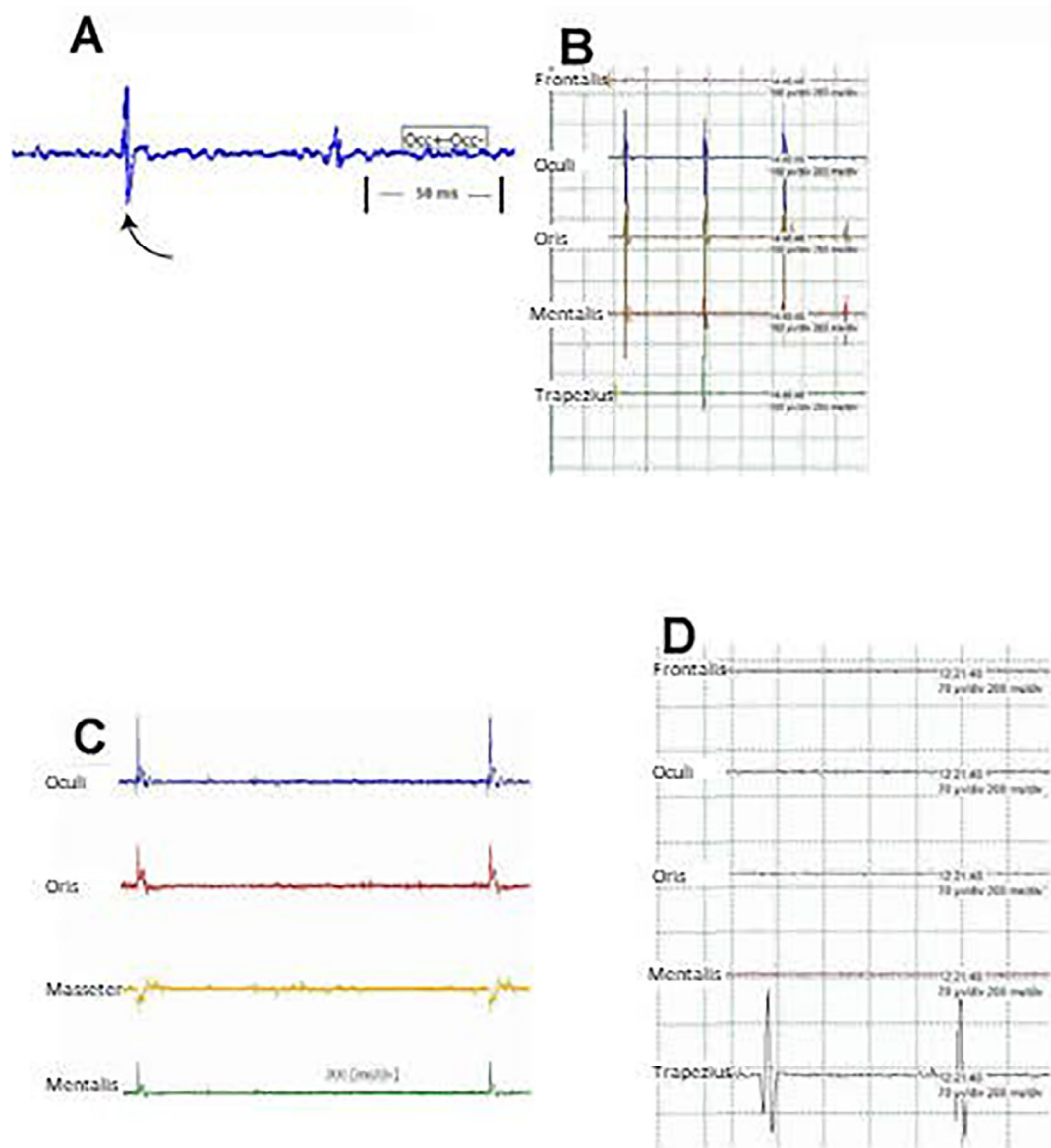


Fig. 19. Spikes. (A) Spike in the orbicularis oculi muscle (arrow) associated with dissection of tumor margin and following earlier train activity in the same muscle. The clinical setting suggests this is a single motor unit potential due to previous or ongoing surgically induced axonal depolarization. A later smaller and less well-formed deflection may represent a distant motor unit potential. (B) Spikes in all monitored muscles resulting from stimulation artifact. (C) Spikes in all monitored muscles resulting from metal-on-metal contact (microcurrent artifact). (D) Spikes representing EKG artifact in the trapezius muscle.

When firing rates of a single, paired, or several MUPs increase further (>60 Hz), it has been described as “A-trains”⁸⁷ (Fig. 22). These may occur as simple runs of sinusoidal, high-rate trains, or as grouped repetitive discharges of similar character. The latter pattern is also known as myokymia.⁸⁸ Myokymia occurs in the setting of axonal pathology and although the specific pathophysiology is not defined, likely results from ephaptic transmission (transmission of neural signals due to coupling at the axon–axon interface where two axons run next to each other) in one or more axons. In line with the expectation that underlying axonal pathology is present, A-trains are more consistently seen in patients with preoperative

nerve dysfunction and are most closely correlated to worsened postoperative nerve dysfunction,⁸⁹ especially if the total amount of this activity (“train time”) exceeds 10 s cumulatively during the procedure.⁹⁰ Despite the expectation that some degree of axonal pathology is necessary to produce A-trains in the first place, this pathology may remain subclinical, as postoperative deficits are not inevitable when A-trains are present.⁹¹ Romstock et al.⁸⁷ correlated different types of train EMG, spike, and burst potentials with postoperative facial nerve function. Although spikes, bursts, and trains are associated with surgical manipulations and provide important feedback to the surgeon, among all of these patterns, only the A-train

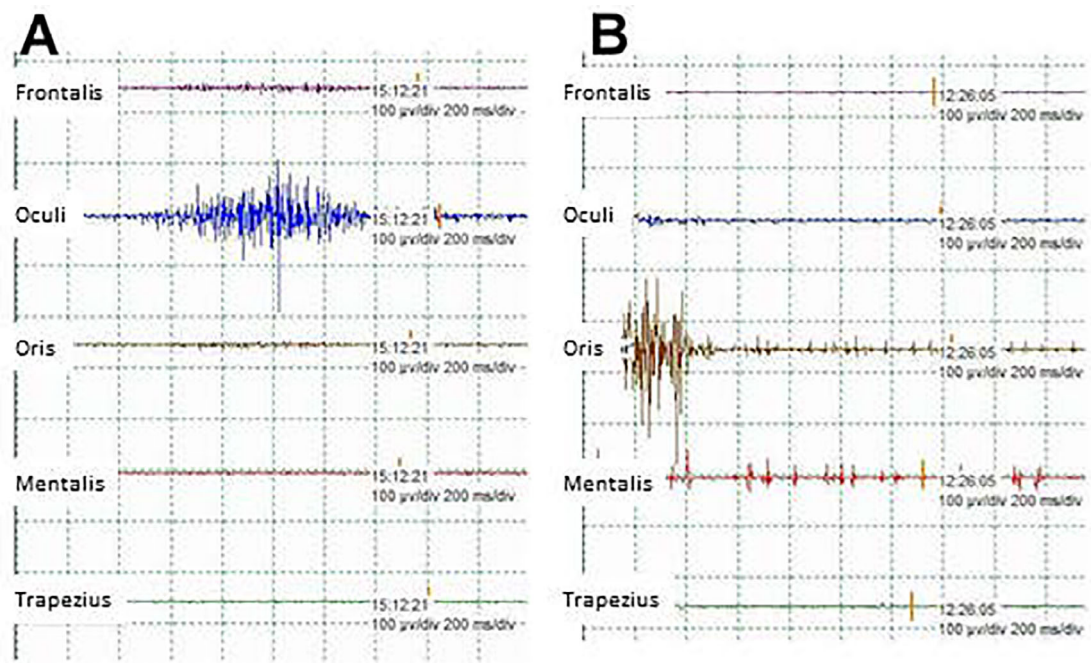


Fig. 20. Bursts. (A) Burst of EMG activity in the orbicularis oculi muscle. (B) Burst of EMG in the orbicularis oris with persisting low-level train of activity following the burst.

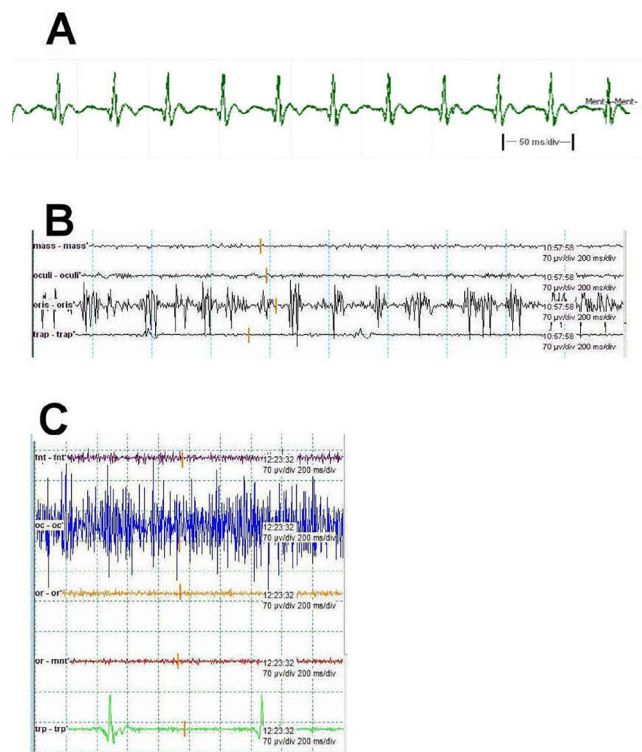


Fig. 21. Neurotonic trains. (A) Train of activity. The stereotyped morphology of the individual spikes suggests repetitive activation of a single motor unit. (B) Train of activity with multiple motor units (asynchronous). (C) C-train with EMG activity filling the entire trace with no return to baseline (interference pattern).

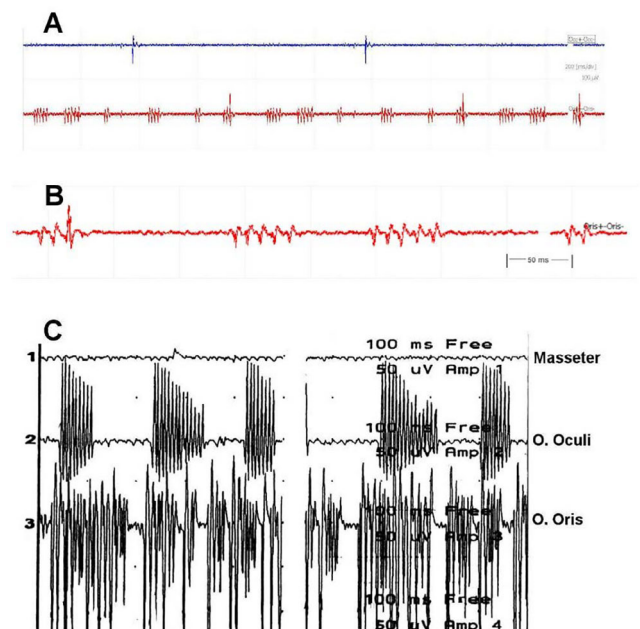


Fig. 22. A-trains. (A) Myokymic pattern with small repetitive groups of high-frequency discharges in orbicularis oris. (B) Small repetitive group A-train with shorter time base to show signal morphology. A-trains elicited with dissection of tumor from the facial nerve. (C) Simultaneous A-trains in orbicularis oculi (myokymic form) and orbicularis oris (non-myokymic form).

TABLE III.
Types of Spontaneous EMG Responses.

EMG Primary Forms	Muscle Correlate	Comments
Spike	MUP	Spikes may represent artifact or other physiologic phenomena
Burst	Near simultaneous depolarization of multiple axons	Polyphasic, typically not associated with nerve damage
Train	Repeating activation of one or more MUPs	Repeating single MUPs have mostly stable spike morphology and a gradual change in frequency if the external influence on the facial nerve is unchanged, although cessation may be abrupt
Train subtypes		
Multiple MUPs	Independently firing MUPs	Individual MUPs will have their own morphology and firing rate. At sufficient frequency, the activity is described as “asynchronous” and the resulting sound as “popcorn” EMG
Interference pattern	Multiple MUPs filling a recording channel	Also known as C-Trains
A-Trains—continuous form	Sinusoidal train of activity at >60 Hz	Duration of A-Train activity correlates with postoperative facial nerve function; sound is described as “bomber potentials”
A-Trains—myokymic form	Repetitive short duration A-Trains	Classic sound is described as “marching soldiers”
Mixed burst/train	Repetitive burst activity	At low intensity, polyphasic MUPs as a sign of chronic denervation; at high intensity, a form of myokymic A-Trains

MUP = motor unit potential.

showed significant correlation with postoperative facial palsy. The different patterns of spontaneous EMG are summarized in Table III.

Prell et al.⁹⁰ related the duration of A-train activity to the degree of postoperative facial palsy, demonstrating good facial nerve outcome with a train time of less than 0.5 s particularly in patients without preexisting facial weakness, mixed outcome between 0.5 and 10 s of train, and poor outcome associated with 10 s or greater train time. Prell et al.⁸⁹ later developed software to measure and display the cumulative train duration in real time. However, this technological advancement is not currently incorporated into commercially available monitoring systems.

Understanding Free-Run EMG Patterns in the Clinical Setting

Under anesthesia, muscles innervated by the facial nerve are typically electrically silent prior to any manipulation of the facial nerve. As such, there is a basic

assumption that baseline EMG is flat and that muscle activity during the procedure will reflect a change in this status. At least two scenarios may alter this expectation. First, in cases where facial neuropathy exists preoperatively, fasciculations (MUPs) or denervation potentials (fibrillations and positive sharp waves) may be generated unrelated to surgical actions. Under this scenario, only activity above the observed baseline level would be considered relevant during the procedure. Alternatively, baseline EMG activity may be present under conditions of relatively light anesthesia when muscle tone and/or voluntary muscle activation may be present (Fig. 2). In fact, facial monitoring can sometimes serve as an additional warning signal to the anesthesiologist because common signs that the patient may be getting light (i.e., tachycardia and elevated blood pressure) may not occur until after the patient begins to move. In this scenario, efforts would be directed toward deepening anesthesia and thereby restoring a quiet baseline.

Once surgical activity places the facial nerve at risk, one can consider the degree of neuronal irritation as the extent to which motor axons are depolarized independent of normal neural function. The density of observed MUPs will reflect a general sense of the degree of neural irritation at the time from the total number of axons that are depolarized as well as the frequency with which each axon depolarizes repetitively. The density of MUPs is of the greatest importance, whereas the amplitude of a MUP—determined by the recording field—has little import. However, in composite EMG signals when multiple MUPs are present, amplitude may become an additional indicator of MUP density due to superimposition of MUPs (e.g., bursts or interference pattern). Irritation in itself does not equate to injury, and some highly irritating triggers may have benign implications (e.g., osmotic/thermal irritation associated with wound irrigation). Therefore, irritation is an indication that a source of irritation should be identified and once a cause is known, the implications of the degree of irritation, specific patterns of EMG, and persistence of irritation beyond the inciting factor *for that specific factor* can be weighed in terms of risk to the facial nerve. To obtain maximal benefit from monitoring, burst and train potentials must trigger a troubleshooting algorithm for the surgeon and monitoring team. Depending on the clinical scenario, it may prompt relaxation of retractors, a switch to warmed irrigation, cessation of laser use, or, when in doubt, re-testing the nerve electrically to confirm its integrity.

Identifying a specific cause of irritation typically starts with an examination of the relation of the irritation to the ongoing surgical activity at the time. When EMG does not have a clear relation to ongoing surgical actions, the EMG findings are often less concerning, especially if a low density of MUPs is present (single or several MUPs at low rates). This activity may still be related to the surgery as residual irritation from earlier manipulation or as ongoing irritation due to a surgical factor outside the main area of current activity (e.g., traction from a retractor or irritation from a surgical patty). If no source of ongoing irritation is apparent, such activity is commonly unrelated to irritation at the surgical site and instead

BOX 6. Pitfalls in FNM - Case 6

A 58-year-old woman presents with a left 4-cm acoustic neuroma and with minor mouth droop on close inspection (House-Brackmann Grade II). A left translabyrinthine approach was used with succinylcholine administered at the time of intubation. Cranial nerve VII was monitored, along with upper and lower sensory-evoked potentials, and cranial nerves V, IX, X, and XI with electrodes placed in the obicularis oculi, obicularis oris, mentalis muscles for VII, masseter for V, soft palate for IX, endotracheal tube for X, and trapezius for IX. The stimulation return electrode was placed 3 cm above the contralateral right eye. "Tap" tests were performed on all facial electrodes to ensure proper stimulation artifact. The initial stimulation began at 1.0 mA during drilling, and a muscle near the incision was stimulated to ensure that technical aspects were operational. Median nerve TOF showed full recovery of neuromuscular blockade. Once facial nerve activity was located, stimulator output was decreased to 0.2 mA, and then to 0.1 mA once inside the dura. Free-run activity demonstrated appropriate drilling and cautery artifact. During the procedure, bursting activity was noted and the surgical team was informed. About 10 minutes later, the individual bursts transformed into a train of bursts (B-trains). A remapping of the area was performed, thereby locating the facial nerve. Given the activity and size of the tumor, the surgeon continued resection in another area and the bursting stopped. About 45 minutes later, the surgeon returned to the area of B-trains and restarted his resection. About 10 minutes into the resection, A-trains were noticed and the neurophysiologist recommended cessation of surgical activity in that area. Once the surgeon stopped manipulating that area, the activity slowed and eventually stopped in about 15 seconds.

The Issue

The appearance of A-train activity prompted a surgical pause at the area of resection. Individual EMG activity not only has to be interpreted in relation to location of the nerve relative to the surgical tools but also to the specific type of activity. For example, trains may be initiated by non-traumatic causes such as irrigation with cold saline and a colder temperature in general. Some patterns of activity are predictive of postoperative deficits more so than others. Consequently, proper training and experience of the surgeon and/or his monitoring team are required to differentiate the patterns because incorrect interpretation of a non-traumatic train activity may stop a resection when a gross resection is in fact possible. During this procedure, activity was noted in all three distributions of the nerve tested. This may not always be the case. Activity may be noted in only one division of the nerve. For example, if the activity noted in one division is noncritical, one can focus on the other two divisions, but care must be taken to not ignore the original firing because the activity may change to one representing a traumatic injury. In this case, a stop alarm was suggested to the surgical team by the neurophysiologist and the team paused and reassessed the situation. The surgeon decided to not completely remove the tumor from the nerve at the region where the A-train activity was noted. Surgery continued at other regions of the tumor and no A-train activity was noted. Upon completion of the procedure, nerve integrity was tested proximal and distal to the lesion with nerve function noted at 0.2 mA.

The patient woke with some left-sided facial weakness and drooping of the mouth (House-Brackmann Grade III) that resolved to that patient's baseline after 3 months.

may result from the preoperative state of a pathologic nerve, return of muscle tone, or early voluntary activation. The surgeon's response to uncorrelated activity is typically a brief assessment and, if no irritating factor of concern is identified, the procedure continues. Should this pattern persist without correlation to or modulation by the ongoing surgical activity, then this activity may represent a new baseline that must be incorporated into the interpretation of subsequent activity that rises above this baseline, especially if correlated to surgical activity.

When EMG activity is correlated to surgical actions in the vicinity of the monitored nerve, a cause and effect relation is assumed. Surgical actions may result in mechanical, osmotic, thermal, or ischemic irritation of the facial nerve resulting in observed EMG activity and each has its own implications in terms of the postoperative health of the nerve.

Mechanical manipulation of the facial nerve is typically the primary focus and concern. This manipulation may be

due to an inadvertent impact on the nerve while surgical activity is meant to be directed elsewhere or the facial nerve may be known to be in direct surgical contact and manipulation of the nerve is a necessary part of the procedure (e.g., extricating the nerve from the surrounding tumor).

Sudden insults to the nerve may result in a spectrum of EMG responses. A brief burst of moderate EMG activity would typically suggest minor contact with the nerve with a low expectation of a significant insult. A more intense contact might result in more extended and intense bursts of activity, possibly followed by trains of EMG activity even after ongoing contact/manipulation ceases, suggesting a more irritating event with a greater likelihood of injury, especially if repeated over time. This course of events may be more likely in an abnormal nerve with a less irritating cause.

Initial minor manipulation of relatively normal nerves typically does not elicit EMG activity. With

repeated manipulation, it is likely that protective layers of the nerve are disrupted, with EMG activity more readily elicited. Brief burst potentials result from blunt contact with the nerve and only rarely indicate significant injury. With progressive neural pathology, the full spectrum of EMG patterns described above might be seen, including more ominous patterns such as A-trains (Fig. 22). Subsequent diminution of EMG activity may reflect lesser degrees of manipulation but can also reflect the progression of dysfunction with loss of axonal ability to produce a MUP. This phenomenon appears to be reflected in early observations by Prass and Luders where relatively normal nerves produced more burst activity, but nerves that experienced trauma distal to the surgical activity demonstrated less burst activity.⁵⁸ Of course, with sufficient trauma, neural injury need not be progressive as described above. If some degree of axonal transection occurs, the general correlation of degree of irritation on EMG and extent of the insult may not hold. Sharp transection of the nerve may be a silent event⁹¹ and in other abrupt transection events, only a brief burst of EMG activity may be apparent before subsequent silence.⁹² Likewise, any insult or transection occurring during electrocautery would be undetected unless EMG activity persisted after cessation of the cautery.

EMG may also be elicited by non-mechanical factors, including osmotic, thermal, and ischemic effects. Wound irrigation with fluid that differs in osmolarity from that of the facial nerve may induce an intense EMG reaction despite its usual benign implications. As the irrigation gradually warms to body temperature, the asynchronous potentials will subside. Some nerves are hypersensitive to cold irrigation and can benefit by having the irrigation gently warmed in a blood warmer. If thermal-evoked responses are generated during laser surgery, however, they may indicate heating of the nerve and thus laser use should be discontinued temporarily followed by irrigation of the operative site with cool saline or Ringer's solution. If the buildup of heat is slow, there initially may be little or no discrete evoked potentials. Instead, a widened baseline can often be identified in the recorded activity seen on the monitor display. Because these initial responses to heating may be of low amplitude and because an automated monitor is typically set to alarm only when a response is greater than 100 mV, the surgeon and/or monitorist must actively look for the silent increase in baseline or transiently change the baseline alarm to signal at lower response levels.

Induced temperature differences in the surgical field, most commonly from nearby electrocautery, may elicit EMG activity. Although bipolar cautery helps to restrict the area of electrical current compared to monopolar cautery, neural injury can still occur due to the spread of heat by convection or conduction. Therefore, when cautery is required adjacent to the facial nerve, it should be performed using brief, intermittent applications at the lowest possible power. The experienced surgeon learns that when cautery must occur in close proximity to a nerve, it should be done at the lowest possible setting and then *the nerve should be electrically stimulated immediately following cautery (or other risky maneuvers) to confirm that it has not been injured.*⁴¹

"Benign" causes of EMG activity may mask the recognition of EMG activity due to surgical manipulation and thus a brief pause in surgery (seconds or minutes) may be warranted to allow a return to baseline.⁵ If such potentials persist beyond a pause of a few seconds or minutes and the surgeon chooses to resume dissection, these potentials can obscure the identification of new burst responses. This situation can sometimes be circumvented by temporarily deactivating the monitoring of nasolabial activity and monitoring only orbicularis oculi activity because this recording site is generally less prone to train potentials. As soon as possible, monitoring through both EMG channels should be resumed to maximize recording sensitivity.

Ischemic insults may elicit EMG activity from stretching or compression of the nerve. Note, however, that ischemia and other insults may go undetected (electrical silence) if earlier trauma has compromised axonal function.⁹² Differentiation of true EMG responses from non-physiologic responses is a key aspect of monitoring interpretation. For example, the stereotyped runs of A-trains could at first be mistaken for external noise, or the continuous deflections of an interference pattern might be mistaken as a dislodged electrode. An awareness of these patterns and an awareness of the "at-risk" portions of the procedure must be maintained to avoid this mistake. During periods of risk, a "notify first and sort out later" posture must be maintained while the level of certainty versus uncertainty is also communicated to the surgeon.

During FNM, EMG silence may be reassuring—or not, based on the particular circumstances and moment in time. Free-run EMG monitoring is the only form of neuromonitoring in which the state of complete normalcy matches the state of complete dysfunction, that is, a lack of any activity. In most situations, quiet EMG simply suggests an absence of irritation; however, reduced EMG activity may also be related to waning neural function. The latter scenario should be particularly considered in procedures when EMG reactivity is high in relatively early portions of the procedure but then decreases or ceases despite continuing surgical actions that previously elicited concerning degrees of EMG irritation. A proximal nerve stimulation to assess nerve function should be used whenever doubt arises. A differential diagnosis for changes in spontaneous facial EMG activity is provided in Table IV.

The correlation of EMG activity to surgical activity is most efficiently accomplished with the immediate feedback provided by audio output from the neuromonitoring system. The use of audio output is obligatory when using automated systems. When the procedure is being monitored by a technologist and/or neurophysiologist, the surgeon may request that the free-run audio be turned off. If so, it is the responsibility of the technologist and/or neurophysiologist in the operating room to immediately let the surgeon know of any changes or free-running EMG activity that occurs; the person in the room becomes that audio warning. That person must maintain constant vigilance regarding the EMG activity and be able to differentiate noise from critical EMG responses. Visual examination of the EMG can add further information, allowing additional description of the patterns, distribution, and even authenticity of EMG activity versus artifact. Unfortunately, the use of visual

TABLE IV.
Differential Diagnosis of Unexpected Changes in Free-Run EMG Activity.

	Pathophysiology	Causes	Actions
Increase	Mechanical Irritation	Ongoing nerve irritation	Modify surgical activity in proximity to the facial nerve Stretch due to retraction or other cause Ischemia
		Residual nerve irritation after removal of inciting factor	May denote a more severe trauma Consider pause in surgical activity
	Non-mechanical Irritation	Heating—Cautery	Cool irrigation
		Heating—Laser	Cool irrigation
		Cold irrigation	Warm irrigation
		Osmotic effect	Isotonic irrigation
	Technical Factors	Microcurrent artifact	Identical onset/offset in all recording channels, including nearby non-facial innervated, similar component spike elements
		Electrical noise mimicking EMG activity	As above when external source, stereotyped morphology possible, unrelated to surgical actions
		Displaced electrode	High amplitude characteristic “activity” without modulation by surgical activity
	Anesthetic Factors	Light anesthetic with return of muscle tone or reflexive/voluntary contraction	Increase anesthetic depth
Decrease	Mechanical Injury	Diminished facial nerve function	Assess function as indicated
	Improved Function	Diminished irritation	Desired result
	Technical Factors	Recording system failure/deficiency	Troubleshooting:
			Assess for drop out of expected activity
			Confirm continued presence of previously noted surgical/artifactual noise
			Confirm presence of stimulation artifact
			Ensure correct volume settings of audio output
			Confirm correct configuration of programmable settings:
			Electrode inputs selection
			Stimulation artifact rejection/exclusion parameters
			Automated response detection sensitivity
			Confirm appropriate filter settings, amplifier gain settings
	Confirm appropriate electrode impedance		
	Eliminate excessive noise or artifact that may obscure target signal acquisition		
If possible, obtain control CMAP from another nerve to confirm integrity of <i>both</i> recording and stimulation systems			
Anesthetic Factors	Neuromuscular blockade	Reverse neuromuscular blockade	
	Local anesthetic	Appropriate care with local anesthetic use	

CMAP = compound motor action potential.

interpretation alone runs the risk of missed activity should the monitoring team ever be diverted from their screen (e.g., replacing a dislodged electrode). Free-run EMG is most useful as a real-time indicator of irritation of the facial nerve, but no EMG pattern clearly indicates the functional status of the nerve. In addition to audio feedback to the surgeon, an in-room video display of the surgical procedure from the microscope provides invaluable information for IONM personnel. This allows real-time information to convey where the surgeon is working (e.g., adjacent to vs. remote from neural elements), differentiation of source of muscle activity (e.g., mechanical vs. irrigation), and identification of artifact (e.g., metal-on-metal contact).

Motor Nerve Conduction Study Interpretation (Stimulated EMG)

Direct electrical stimulation of the facial nerve may be used to elicit a CMAP in target muscles. This technique is a form of a motor nerve conduction study (as the diagnostic electromyographer might call it), and in the neuromonitoring literature it is often referred to as stimulated EMG, tEMG, or evoked EMG. This technique is essential to evaluate the integrity of the monitoring system and also serves to locate the facial nerve within the surgical field, map its course, and assess its function. Stimulation in the expected general vicinity can be initiated at approximately 0.8 mA and titrated up to threshold or a maximum of

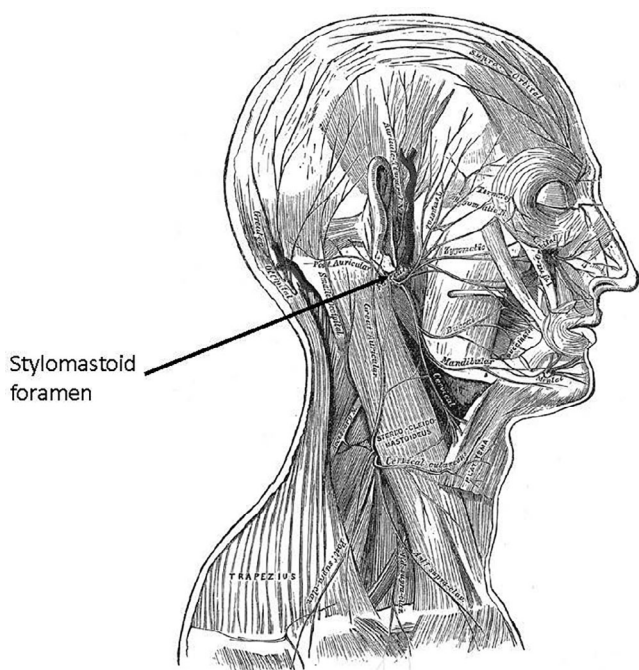


Fig. 23. The facial nerve can be activated transcutaneously near the stylomastoid foramen.

2.5 mA.⁵ Alternatively, the facial nerve can be activated transcutaneously near the stylomastoid foramen (Fig. 23), which confirms function of the system but does not yield information regarding its proximal function. Finally, for intracranial procedures, an alternative nerve such as the trigeminal or spinal accessory may be chosen for activation if the facial nerve is not yet clearly accessible. Note that such activation would confirm at least incomplete neuromuscular blockade and the effectiveness of stimulation but would not exclude the possibility of a temporary, chemically induced facial nerve dysfunction due to inadvertent application of local anesthetics.

A primary goal of the direct electrical activation of the facial nerve is to provide information on the proximity of the nerve to the stimulator. As described in section Technical Considerations of FNM, constant current monopolar stimulation with a 0.1-msec pulse width requires ≤ 0.3 mA when the nerve is directly activated, whereas a higher current is needed when initially locating the nerve or when there is intervening tissue or bone. Once a facial nerve response is obtained, relative thresholds can guide as to location, proximity, and course of the nerve through the surgical field. Some surgeons might instead depend on free-run EMG activity to alert them to facial nerve proximity. However, this approach has numerous concerns, for example, activity in proximity to the nerve that is not irritating, instances where the first episode of irritation may also be damaging, sharp transection of the nerve that may be electrically silent, and irritation occurring during electrocautery, which will mask useful recording. As such, when the nerve is at potential risk, early electrical stimulation to localize the facial nerve is advised.

The assessment of facial nerve function is also a primary goal. Stimulation of the nerve proximal to a point of

TABLE V.
Unexpected Responses to Stimulation.

Observation	Causes	Actions
Typical response in unexpected location	Atypical location of facial nerve	True positive
	Nerve splayed over tumor	Map surface of tumor
	Abnormal stimulation spread	Dry surgical field Reduce stimulation intensity Consider bipolar stimulator
Response with atypical latency	Volume conduction	Trigeminal stimulation (latency <5 msec)
	Nervus intermedius response	Latency ~11 msec
Atypical/inconsistent responses	Misidentification of background free-run EMG activity	CMAPs will repeat with similar latency, activity noted on free-run EMG
	Misidentification of external artifact	CMAPs will repeat with similar latency with each stimulation
	Misidentification of stimulation artifact	Improve recording technique such that stimulation artifact does not contaminate signals with targeted latency

CMAP = compound motor action potentials.

potential injury early in the procedure assesses the conduction of the nerve through the more distal segment. The presence of a response of similar amplitude and at a similar threshold compared to that of prior testing suggests retained function in that segment whereas the absence of a response suggests conduction block. If current shunting (Fig. 9) is avoided, a rise in the stimulation threshold at previously tested sites may reflect partial dysfunction. Similarly, decreased CMAP amplitudes using previous stimulation parameters may reflect partial dysfunction if the stimulation site is identical and no current shunting is present. If distal and proximal nerve segments are accessible, the presence of a response with distal but not proximal stimulation strongly suggests the presence of interval conduction block. Motor nerve conduction studies in its many forms constitute “active” monitoring as opposed to the “passive” monitoring of free-running EMG. Only active monitoring provides assurance that facial function remains intact. Tables V and VI summarize causes and approaches to troubleshooting unexpected responses when attempting to stimulate facial nerve activity.

Interpretation Specific to the Type of Surgery Monitored

FNM During Acoustic Tumor and Skull Base Surgery. Preservation of facial nerve function during resection of acoustic neuroma or other skull base tumors depends on many factors, including tumor size, surgical approach, and the microanatomic relationship of the nerve to the tumor.⁹³ The following section highlights

TABLE VI.
Unexpected Absence of a Response to Stimulation.

Causes		Actions
Nerve damage	Visible injury	Consider repair
	Conduction block	Compare distal and proximal responses
Signal acquisition problems	Nerve more remote than expected	Continue mapping
		Continue surgical exposure
	Nerve insulated by intervening tissue	Increase stimulation current
		Increase stimulation current
Technical issues—overall	Current shunting	Dry field
		Avoid contact between stimulator and surgical instruments
		Obtain control CMAP from adjacent nerve to confirm BOTH stimulation and recording
Technical issues—recording	Drop-out of expected activity	Confirm continued presence of free-run EMG
		Confirm presence of stimulation artifact
		Confirm presence of electrical noise from surgical actions
		Confirm correct volume of audio output
	Configuration of settings	Check electrode inputs
		Check artifact rejection settings
		Check stimulus exclusion settings
		Check filter settings
	Electrode issue	Check impedances
		Mitigate/eliminate sources of electrical noise
	Current flow absent	Confirm connection and programming correct
		Replace system components
Technical issues—stimulation	Current flow present	consider replacing stimulation system components
Anesthetic issues	Neuromuscular blockade	Reverse neuromuscular blockade
	Local anesthetic on nerve	Appropriate care during administration

CMAP = compound motor action potential.

monitoring details specific to each of the three main surgical approaches to removing acoustic tumors.

A *translabyrinthine* approach can minimize the risk to the facial nerve and minimize cerebellum retraction, but any residual hearing is sacrificed. The anatomy of the facial nerve is extremely consistent within the temporal bone. This approach allows unequivocal early identification of the facial nerve at the fundus of the internal canal even before the tumor is exposed and without regard to tumor size. Both *middle* and *posterior fossa (retrosigmoid)* approaches have the potential of sparing hearing if the

tumor is small. During the middle fossa approach, the facial nerve often overlies the tumor and must therefore be retracted to expose the tumor. In the posterior fossa approach, large tumors typically obscure the facial nerve on the anterior side of the tumor, whereas small tumors will require drilling of the porous acousticus before nerve and tumor can be visualized.

Considerations specific to each of these approaches will be discussed later after general considerations of the stages of tumor removal and their implications for FNM.

In the CPA, the facial nerve is most commonly displaced anterior to the tumor and is thus not initially evident. Nonetheless, the posterior aspect of the tumor should always be electrically mapped to exclude an atypical facial nerve course on the posterior face of the tumor, where immediate debulking could result in its transection. This is accomplished by sweeping the exposed face of the tumor with a stimulating electrode set to a level well above the expected threshold of the nerve. Periodic stimulation at a suprathreshold level (e.g., 0.5–1 mA) without eliciting a response confirms that adjacent portions of the tumor can be safely resected without damaging the facial nerve.⁹⁴

Although acoustic neuroma is the most common CPA tumor, other types may be found in this location and can lead to different anatomical relationships between the tumor and cranial nerves. Facial neuromas may appear identical to acoustic tumors, thus initial stimulation of the tumor prior to dissection is again indicated. If a response is noted on the posterior surface of tumor, the surgeon must differentiate between facial neuroma versus an atypical, posterior nerve location on an acoustic tumor.

Meningiomas rising from the anterior surface of the apical petrous bone may also displace nerves posteriorly, whereas a large lesion of jugular foramen origin may displace nerves superiorly. Such distinctions are usually, but not always, evident from careful evaluation of preoperative MRI scans. In any event, it is important to confirm that the anatomical relationships between the tumor and cranial nerves are as expected and to modify the surgical dissection if initial monitoring data indicates atypical anatomy.

Small tumors may be removed en bloc but large tumors require debulking before the adherent capsule can be separated from the nerve. During this time, intermittent stimulation and close monitoring for spontaneous EMG should be performed. If significant or prolonged activity is seen, the stimulator should again be used to confirm 1) the absence of the nerve in the region of dissection, and 2) the integrity of the nerve. A healthy nerve will typically elicit robust EMG responses at a threshold of <0.2 mA. The proximal nerve should be periodically stimulated because an intact response confirms the functional integrity of the nerve in its entire course through the CPA.

During dissection of the tumor capsule, the use of stimulating dissection instruments with continuous electric stimulation can provide greater safety from ongoing concurrent surgical dissection. Whether dissecting instruments or a separate stimulator is used, the best practice

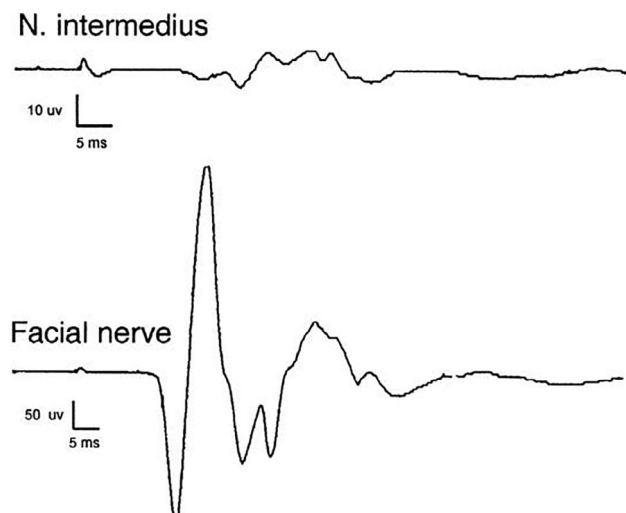


Fig. 24. Differentiating the facial nerves from nervus intermedius responses. Note the longer latency and smaller amplitude of the nervus intermedius response (different voltage scale), which is seen in orbicularis oris but not orbicularis oculi channels.

is to frequently modify the stimulus current setting to maximize sensitivity and selectivity while mapping the nerve and assessing its function. A “high” mapping setting (0.5–1.0 mA) will provide greater sensitivity due to current spread; if no responses are elicited, the dissection can safely proceed. If EMG responses are elicited, the stimulation can be lowered to 0.1 to 0.2 mA, which will minimize current spread and permit better localization. With ongoing stretching, microtrauma, and devascularization, the nerve threshold might become progressively increased thereby requiring small progressive increases in the current intensity.

It is important to recognize the characteristic response pattern from the stimulation of the nervus intermedius. The nervus intermedius may be contiguous with the facial nerve itself or it may run as a separate nerve over a different portion of the tumor. If it is incorrectly identified as the facial nerve itself, the surgeon may falsely assume that other portions of the tumor can be safely resected without endangering the facial nerve. Its response is typically of a longer latency and lower amplitude than true facial nerve responses and is seen only in the orbicularis oris channel (Fig. 24).⁹⁵

The final stage of tumor resection is usually the most difficult. The point of maximal adherence between the tumor and the nerve is typically at the porous acousticus. During this stage, it is vital to closely monitor spontaneous facial EMG and if significant activity is encountered, the nerve should be stimulated proximally to confirm continuity. On occasion, with large tumors, if stimulation demonstrates a significantly elevated threshold indicative of neural trauma, the surgeon may opt for a subtotal resection, leaving a small portion of tumor adherent to the nerve to preserve facial function. Any future evidence of significant regrowth can be addressed with staged surgery or stereotactic radiation.⁹⁶

Following tumor resection, the nerve should be stimulated at the most medial location accessible and the response amplitude evaluated as discussed above.

If proximal electrical stimulation demonstrates an increased threshold but with an intact facial EMG response, this is evidence that the facial nerve, while possibly injured, is anatomically and functionally intact. In this case, optimal long-term functional recovery will most likely be achieved by allowing the nerve to heal. However, if no response can be obtained at high levels of stimulation, the decision is more difficult. Because the intact distal segment of the nerve will continue to conduct action potentials for several hours even after a complete transection, stimulation mapping can be used to locate the most proximal segment that is still functioning. If clear anatomical continuity between the most proximal stimulation site and the brainstem REZ can be demonstrated, then the long-term prognosis may be best with no further intervention. If a clear discontinuity is evident, then re-anastomosis may be considered. This is best accomplished by identifying the most distal segment of the nerve exiting the brainstem and joining it to the most proximal segment in the temporal bone. Although a nerve graft can be performed, better results will usually be achieved with a direct end-to-end anastomosis. This may require further drilling in the temporal bone to free a long enough segment with constant monitoring of EMG reactivity and stimulation mapping to ensure intact function of the distal segment.

Retrosigmoid Approach. The retrosigmoid approach to the posterior cranial fossa and temporal bone places the nerve at risk during cerebellar retraction, tumor dissection, microvascular decompression, and vestibular neurectomy.

Cerebellar retraction may elicit EMG responses due to stretching of the VII/VIII complex, so this is the first stage in the procedure where monitoring is important. If ipsilateral hearing is intact enough to support ABR generation, the I to V interpeak latency should also be carefully observed because cerebellar retraction can cause ABR prolongation before facial EMG responses are elicited.

Spontaneous EMG activity should be carefully observed if drilling of the IAC is required for exposure. Because the vibration from the drill causes visible and audible artifacts, the surgeon may periodically pause the drilling to allow detection of any EMG activity secondary to facial nerve irritation from either heat or contact.

Translabyrinthine Approach. Different considerations come into play during a translabyrinthine approach. Translabyrinthine removal of an acoustic neuroma begins with an incision behind the ear, followed by a mastoidectomy, meaning that vigilance is required by both surgeon and monitoring team even during this initial exposure.

In addition, the facial recess between the nerve and the chorda tympani is widely opened to allow for soft tissue packing of the middle ear and Eustachian tube to reduce the chance of postoperative cerebrospinal fluid leak. The incus and head of the malleus are removed to

BOX 7. Pitfalls in FNM - Case 7

A 47-year-old woman presented with total right-side hearing loss and mild weakness of her right facial muscles (House-Brackmann Grade II). An MRI revealed a 3.5-cm right acoustic neuroma, and she was scheduled for a translabyrinthine craniotomy for resection of her tumor with multiple cranial nerve monitoring. Recording electrodes were placed in the ipsilateral orbicularis oculi, orbicularis oris, mentalis, masseter, and trapezius muscles, and in the contralateral orbicularis oris. A signal ground was placed just anterior to the ipsilateral ear canal, and a “tap” test confirmed the integrity of the recording system. After exposure and dural opening, a stimulus anodal return was placed in the posterior margin of the incision, and a flexible-tip probe was used to stimulate across the exposed surface of the tumor at 0.5 and then 1.0 V, with no EMG response noted, although the system indicated that current was being delivered and stimulation of an exposed muscle elicited twitching. Assured that the facial nerve was not on the exposed face of the tumor, the surgeon proceeded to debulk the tumor with ultrasonic aspiration. After the core of the tumor was removed, the stimulation probe was used to examine the superior and inferior surfaces, probing into the dissection planes as the capsule was retracted. No response was obtained from the inferior pole at 1.0 V, but superior stimulation produced a response at 0.3 V in the orbicularis oculi, orbicularis oris, and masseter channels with a latency of 4.5 msec. Based on this finding, the surgeon began the dissection from the inferior pole, with no significant EMG activity noted. After removal of tumor from the internal auditory canal, the VIIth nerve was stimulated and a robust response was noted at a threshold of <0.1 V. This was periodically rechecked during tumor removal, and the response remained intact with a low threshold. After removal of most of the tumor, the surgeon was able to visualize the root entry zone of the VIIth and VIIIth nerves at the brainstem. However, stimulation of the VIIth nerve produced no response, even with stimulation up to 1.0 V. Stimulation at the internal auditory canal still produced a robust, low-threshold response. As the last of the tumor was being removed, it was apparent that the facial nerve had been transected in the mid-CPA, and postoperatively, the patient had complete ipsilateral facial palsy. A hypoglossal/facial anastomosis was performed 10 days later, and after several months, the patient eventually attained House-Brackmann Grade IV facial function.

The Issue

When the motor portion of the trigeminal nerve is stimulated, the response generated in the masseter and temporalis muscles is frequently also seen in facial muscles due to volume conduction. The distribution of the responses is thus not a foolproof indicator of which nerve has been stimulated. However, the onset latency is quite different: responses to trigeminal stimulation always begin before 5 msec post-stimulus, whereas responses to facial nerve stimulation typically range from 6 to 8 msec onset latency (Mnemonic: 5 less than 5, 7 about 7). Failure to recognize this distinction led the surgeon to believe that the facial nerve was on the superior pole of the tumor and that it was thus safe to aggressively remove tumor from the inferior pole. Unfortunately, this resulted in inadvertent transection of the facial nerve, and there was not a significant EMG response. Loss of the nerve may have been avoided if inferior dissection had proceeded more carefully, with frequent stimulation to identify the location of the facial nerve.

Lessons Learned

Latency, not distribution of responses, is the best way to distinguish between responses to trigeminal versus facial nerve stimulation. The facial nerve is often splayed out into a wide sheet when distorted by large tumors, thus a response at one location does not exclude the presence of viable nerve fibers a considerable distance away. Frequent intracranial stimulation should always be used when removing acoustic tumors. In the future, transcranial corticobulbar motor-evoked potentials may prove useful in large tumors because the location of the facial nerve may not become apparent until a majority of the tumor has been removed.

increase exposure of the Eustachian tube. Consequently, the tympanic segment of the facial nerve, which is often dehiscent, is at risk during this process. Proactive stimulation when opening the facial recess may identify a dehiscence, prompting greater caution during the procedure.

The translabyrinthine approach to the CPA encounters risks to the nerve at the fundus of the IAC (at the vertical crest aka “Bill bar”) as well as intracranially within the CPA. Risks of injury to the facial nerve during tumor resection can be minimized by 1) intraoperative monitoring using stimulating dissection instruments, 2)

using Brackmann fenestrated suction tips to reduce suction trauma to the nerve,⁹⁷ and 3) when possible, using sharp instead of blunt dissection to reduce traction injury.

After tumor removal, an abdominal fat graft is placed to reduce the chance of a cerebrospinal fluid leak. The team must remain vigilant because any evoked EMG potentials noted during packing can signify that the graft is placing too much pressure or traction on the nerve. Marked EMG responses may require that the graft be temporarily removed, the nerve reassessed electrically, and the graft then gently replaced.

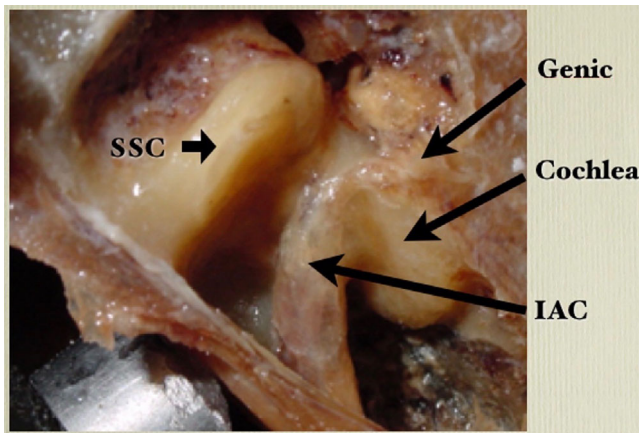


Fig. 25. A left cadaver dissection viewed through a microscope reveals the complex anatomy within the temporal bone. Seen from above as in the operating room following initial drilling exposure, the superior semicircular canal (SSC) is approximately 60° from the plane of the internal auditory canal (IAC). Stimulation near the geniculate ganglion can help identify the greater superficial petrosal nerve and a potentially dehiscent geniculate.

Middle Fossa Approach. In the middle cranial fossa approach, after craniotomy and elevation of the temporal lobe, the floor of the middle cranial fossa is exposed, beneath which are the unseen contents of labyrinth, the IAC, and the carotid artery (Fig. 25).

Because there are few anatomical landmarks to help orient the surgeon to the underlying structures, careful attention to EMG reactivity and periodic stimulation at a suprathreshold level is necessary to identify the location of the facial nerve. The facial nerve is at greatest risk during middle cranial fossa surgery at the perigeniculate area. In particular, the meatal foramen tightly binds the nerve (average diameter of 0.68 mm)⁹⁸ and is located within a 4-mm area bound by the ampullated end of the superior semicircular canal and the basal turn of the cochlea.⁹⁹

Key landmarks to use when beginning the temporal bone dissection include the greater superficial petrosal nerve extending anteriorly from the geniculate ganglion and the arcuate eminence posteriorly. In 15% of patients, the geniculate is dehiscent and therefore at risk simply during elevation of the temporal lobe dura. Stimulation in the expected area of the greater superficial petrosal nerve may not only identify a dehiscence, but also aid in retrograde dissection toward the meatal segment of the facial nerve.

Once the IAC is opened, the facial nerve may be just below the incised dura, particularly if the tumor originates from the inferior vestibular nerve, resulting in displacement of the nerve superiorly. Thus, once again, baseline stimulation should precede tumor removal.

It is necessary to carefully dissect the nerve free and gently move it aside to access the underlying tumor and cochlear nerve. If the facial nerve is adherent to the tumor, the tumor-nerve complex must be rotated into a favorable angle for sharp microdissection. This manipulation may cause stretch-induced injury with EMG

reactivity. Because the REZ is not typically accessible with a standard middle cranial fossa approach, the facial nerve should be periodically stimulated at its most medial aspect to confirm its integrity. Auditory brainstem responses should also be monitored closely during this procedure to confirm integrity of the cochlear nerve and artery.

Responses from Other Nerves During Acoustic Neuroma Resection. In addition to the facial nerve, responses from the activation of other cranial nerves may be encountered, particularly with larger tumors. It is vital to understand which nerve is responding so that the surgeon can accurately determine the anatomical course of each nerve.

The trigeminal (Vth) cranial nerve, a mixed sensory and motor nerve, is commonly encountered with large acoustic tumors. Techniques to reliably assess the sensory component during surgery are not available. Thus, the motor component (Vm, which is part of the V3 division) is the only reliable marker. It innervates the temporalis and masseter muscles and can in principle be monitored with spontaneous and triggered EMG from either of these muscles. Unfortunately, crosstalk between muscles innervated by V and VII is quite common due to their anatomical proximity and the large bulk of the muscles of mastication. Thus, distinctions based on the channel where responses are recorded are prone to error. More reliable is latency: responses to stimulation of Vm are of shorter latency (~4 msec to onset) than those to stimulation of VII (~5–7 msec). A convenient mnemonic is “V less than 5 msec, VII close to 7 msec.”

The IX-X-XI complex may also be encountered with large tumors but is less likely to be confused with VII than is V. The Xth nerve can be monitored with an EMG endotracheal tube, which records responses from the vocalis muscle, innervated by the recurrent laryngeal nerve, a branch of the vagus (Xth) nerve. Another option is EMG activity from the trapezius muscle, innervated by the spinal accessory nerve (XI). XI can often be identified relatively early, and thus is used as a confirmation of stimulus delivery by recording responses elicited in the trapezius, which is distinct and does not exhibit crosstalk with facial muscles. This muscle is also a convenient early indicator of light anesthesia, which often announces itself by an increase in spontaneous EMG activity in muscles innervated by multiple nerves. The trapezius seems to be particularly sensitive to light anesthesia, and sudden increases in EMG activity often precede overt patient movement. When stimulating the jugular foramen complex, care must be taken not to overstimulate the Xth nerve, as this can cause bradycardia or even asystole.

Finally, the abducens (VIth) nerve is often encountered in the late stages of tumor resection, running at approximately right angles to the VII/VIII complex. Although EMG monitoring from the lateral rectus is feasible, it is technically demanding and should not be attempted by individuals without special training and experience. Fortunately, responses from the lateral rectus usually can be recorded from the orbicularis oculi channel used for VII if one of the electrodes is placed at the lateral canthus and thus overlies this muscle. Although

spontaneous activity is not seen with this montage, electrical stimulation of VI can elicit small, short latency responses that are limited to the orbicularis oculi channel. A schematic of the responses to stimulation of Vm, VI, VII, and XI is shown in Figure 26.

Infratemporal Approach. To obtain adequate exposure, the infratemporal approach to the skull base

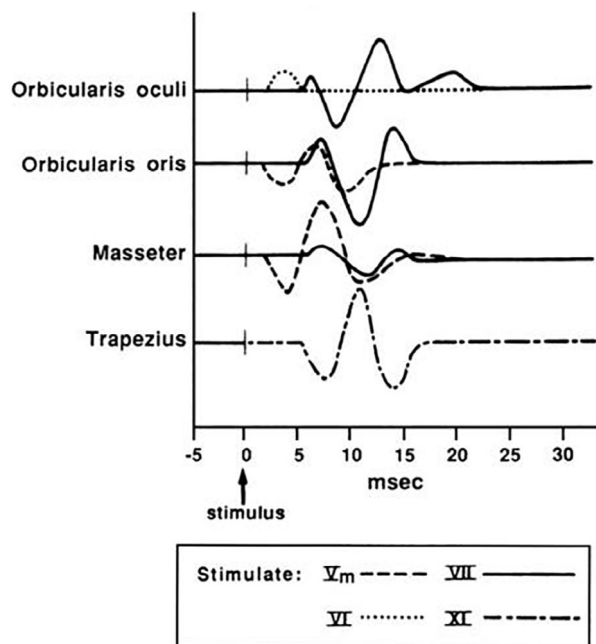


Fig. 26. Schematic representation of traces obtained in a four-channel montage after intracranial stimulation of cranial nerves Vm, VI, VII, and XI. Despite crosstalk in Vm and VII channels, these nerves can be clearly distinguished by the shorter latency of responses to Vm stimulation. Stimulation of VI produces a short latency response in the orbicularis oculi channel due to volume-conducted activity from the nearby lateral rectus. Responses to cranial nerve XI stimulation are restricted to the trapezius; care must be taken not to stimulate XI at too high a level as it can cause significant patient movement.

may include facial nerve decompression with anterior transposition. Intraoperative monitoring is especially helpful in detecting microtrauma by the drill as well as by self-retaining retractors. Although preoperative embolization of glomus jugulare tumors can provide a significant reduction in blood loss, it should be noted that preoperative devascularization of the facial nerve can markedly reduce the responsiveness of the nerve to electrical and mechanical stimulation.

Transcochlear Approach. Meningiomas and epidermoid tumors of the CPA are often located anterior to the facial nerve, which increases the risk of trauma during tumor resection. When necessary, a transcochlear approach provides anterior exposure by transposing the facial nerve posteriorly following decompression and transection of the greater superficial petrosal nerve.¹⁰⁰ However, because of devascularization, postoperative facial paralysis is common with nerve transposition, although satisfactory recovery usually occurs. Consequently, a modified “transotic” approach that avoids nerve transposition can be a useful alternative depending on the degree of exposure required.¹⁰¹

Monitoring During Microvascular Decompression for Hemifacial Spasm. Hemifacial spasm surgery presents an unusual situation in which the surgical procedure is intracranial but the primary monitoring is extracranial. Although ABR and EMG monitoring during cerebellar retraction are still relevant, the primary technique involves recording what has been termed the “lateral spread” response.¹⁰² EMG recordings are obtained from the orbicularis oculi (innervated by the zygomatic branch of the facial nerve) and mentalis (marginal mandibular branch). Stimulating electrodes are placed over the respective branches of the peripheral facial nerve. Normally, stimulation of peripheral branches distal to the pes anserinus should elicit EMG responses only in the target muscles. However, in hemifacial spasm, stimulation of the zygomatic branch typically elicits a short latency response (~2 msec) in the target muscle (orbicularis oculi) and a longer latency response (~10 msec) in the mentalis. The converse may also be

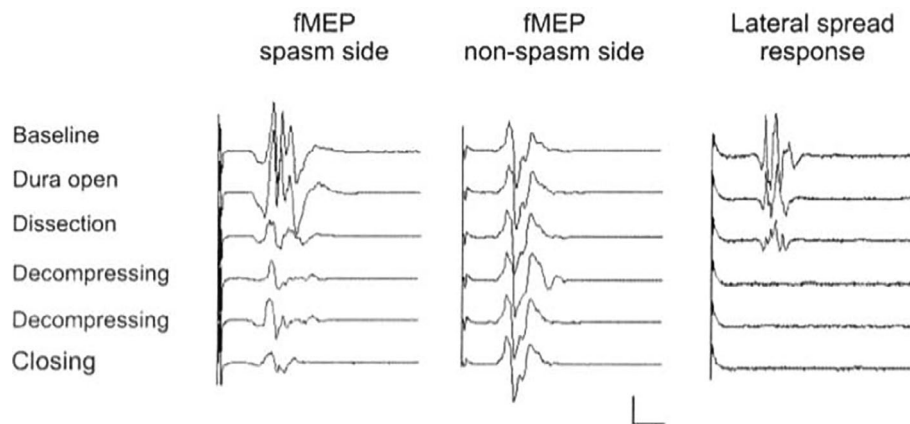


Fig. 27. Representative EMG recordings from the mentalis muscle and indicated intervals during microvascular decompression for left hemifacial spasm. Note the reduction in the ipsilateral facial motor-evoked potential corresponding to changes in lateral spread, with stable contralateral (non-spasm side) motor-evoked potential.

observed—later responses in the orbicularis oculi after stimulation of the mandibular branch. The response may disappear after dural opening, even before any vascular dissection has taken place, but can often be facilitated by delivery of a high-frequency (50 Hz) train prior to testing with single pulses. Irritated nerves also tend to exhibit greater sensitivity to mechanical manipulation. The lateral spread response may also be elicited by transcranial stimulation (Fig. 27).

The exact mechanism of this abnormal response has been attributed to ephaptic transmission between facial nerve axons at the site of arterial compression or to abnormalities in the facial nerve nucleus.¹⁰³ Regardless of the exact mechanism, the goal is to identify the vessel compressing the facial nerve in the CPA, particularly at the VII nerve exit from the brainstem or more laterally (usually a vascular loop of anterior inferior cerebellar artery) and move it away from the nerve with an insulating pledget to keep it isolated. When this is accomplished, the abnormal lateral spread response disappears or is significantly reduced. This may happen immediately after decompression, although the change may be delayed. If the abnormal response is not abolished, it is important to alert the surgeon because there may be a second vascular loop that is only apparent if the viewing angle of the operating microscope is changed.

Middle Ear Surgery. Middle ear surgery typically has a very low risk to the facial nerve but this risk may be significantly increased by congenital malformations or chronic ear disease, which can create granulation tissue or cholesteatoma that obscures the normal anatomical landmarks. Furthermore, cholesteatoma may erode the fallopian canal, making the nerve more susceptible to trauma during surgical dissection.

Stapes surgery carries an extremely low risk of permanent facial nerve injury and therefore is infrequently monitored. Nonetheless, the oval window is in close proximity to the tympanic segment of the nerve, which is a common area of bone dehiscence. Lasers and microdrills are often used to create a new fenestra for a prosthesis.^{104,105} Even with a normal facial canal, thermal or direct mechanical injury is possible. Stapes surgery is often performed under local anesthesia. Monitoring, if indicated, can be performed under a local anesthetic with intravenous sedation given just before placing the needle electrodes. However, voluntary movements of the face (e.g., grimacing) can trigger the alarm, creating “false-positive” responses that must be excluded based upon the surgeon’s activities. If a patient is referred with a known prolapsed facial nerve identified at a prior surgical attempt, general anesthesia with monitoring may be preferred.

As noted previously, dehiscences of the fallopian canal can allow local anesthetics to inadvertently cause a temporary chemical paralysis of the nerve, making monitoring useless. Knowing this, the surgeon must not only avoid injecting lidocaine near the stylomastoid foramen, but when injecting the ear canal in the presence of a tympanic membrane perforation, local anesthetic solutions must be prevented from dripping into the middle ear, for example, such as by using gelfoam or other similar materials.

Mastoid Surgery. The anatomical course of the facial nerve through these regions is relatively consistent compared with its variable course in the CPA. However, with extensive disease or anatomical anomalies, even experienced surgeons may have difficulty identifying these more distal segments of the facial nerve and avoiding iatrogenic injury. Noss et al.²⁴ reported that electrical thresholds were less than 1.0 V in 55% of such cases, suggesting dehiscence of the nerve even though the nerve was visibly dehiscent in only 13%. In a few cases, an aberrant facial nerve course through the temporal bone was identified, resulting in cancellation of surgical treatment.

The facial recess is a small triangular area only 4 to 5 mm at its largest opening thus an inadvertent slip of the drill in this area could result in facial paralysis, taste disturbance, or hearing loss. In addition to careful control of the drill tip, as the drill extends through the recess, attention must also be paid to the drill shaft, which could injure the nerve via direct contact or heat generation from the rotating shaft. One must look not only for EMG bursts due to direct trauma, but also for small repetitive responses, that is, “drill potentials.” Such potentials should be carefully scrutinized to determine if they represent only vibrations from the drill upon adjacent tissues (bone or epineurium) versus actual direct traumatic contact by the drill bit or the drill shaft.

During intracranial surgery, burst and train potentials may be common, especially during tumor resection. In contrast, during routine mastoid surgery, one expects to see only triggered EMG responses during mapping. Therefore, *any* burst or train activity is of concern. When such “trauma potentials” occur, the procedure should be immediately halted, followed by stimulation proximal and distal to the site of the surgical dissection.

When disease, infection, tumor, or congenital anomalies suggest that the facial nerve may not be in its typical location, electrical mapping becomes of even greater importance. Under these circumstances, the surgical dissection may also need to be modified to identify the facial nerve more proximally in the epitympanum or more inferiorly at the digastric ridge.

Congenital Aural Atresia. Surgery for congenital aural atresia poses unique risks to the facial nerve because of inconsistent anatomy. However, even when the anatomy is anomalous, proper precautions can minimize risk of injury. Jahrsdoerfer and Lambert¹⁰⁶ reported on greater than 1000 cases of atresia surgery and identified a nerve transection in one patient and nerve injuries other than transection in six patients (This report was of cases prior to the implementation of FNM.). They reported that the nerve is most vulnerable when 1) making the skin incision, 2) dissecting the glenoid fossa, 3) during canalplasty, 4) when transposing the nerve, and 5) when dissecting soft tissue in the preauricular area. The nerve is displaced in 25% to 30% of atresia cases, and the inexperienced surgeon is most likely to cause injury in the inferoposterior portion of the atretic bone lateral to the middle ear. Low-set ears, canal stenosis, and comorbid cholesteatoma tend to increase the risk for injury even in experienced hands.

Since that report, however, additional facial nerves have been injured during atresia surgery, which prompted Jahrsdoerfer to begin using monitoring on a routine basis (R.A. Jahrsdoerfer, personal communication to J.M. Kartush, 1999).

Parotidectomy. Facial nerve injury is the most concerning complication of parotidectomy. This risk is increased with large tumors, deep lobe tumors, and patients with a history of parotitis.¹⁰⁷ In a 2003 survey, 60% of head and neck surgeons in the United States used FNM but others thought “anatomic landmarks were sufficient.”⁸⁴ This seems somewhat surprising given that facial nerve injury during parotid surgery has accounted for more than 19% of U.S. civil cases that went to trial for facial nerve injury between 1985 and 2000.^{108,109} FNM does appear to be increasingly common among more recently trained surgeons where IONM is often used in academic training programs.

Although the parotid gland is often regarded as being divided into superficial and deep lobes by the facial nerve, the relationship of the gland tissue to the nerve is variable, with no true anatomic division into lobes. The nerve emerges from the mastoid bone through the stylomastoid foramen at the base of the styloid process. This “valley of the nerve” is in a tightly constricted area behind parotid gland tissue and anterior to a portion of the tragal cartilage referred to as the “pointer.” Within 1 or 2 cm, the main facial nerve trunk then divides at the pes anserinus into the previously discussed main branches. The pattern of facial nerve branching within the gland is highly variable from individual to individual, therefore, frequent stimulation throughout the entire procedure is essential.

Unlike surgery of the proximal facial nerve in the temporal bone or CPA, FNM during parotid surgery greatly benefits from four recording channels. A typical set-up would involve bipolar recording electrodes at the frontalis muscle (temporal branch of facial nerve), orbicularis oculi (zygomatic branch), orbicularis oris (buccal), and mentalis (marginal mandibular). This allows for more accurate mapping as the dissection proceeds peripherally.

CONCLUSIONS

Facial nerve monitoring (FNM) has evolved into a widely used adjunct for many surgical procedures along the course of the facial nerve. Nonetheless, a review of the literature and medicolegal cases reveals significant variations in methodology, training, and clinical indications.

Over the years, two models of monitoring have become well-established: 1) monitoring by the surgeon using a stand-alone device that provides auditory feedback of facial electromyography directly to the surgeon, and 2) a team, typically consisting of surgeon, technologist, and interpreting neurophysiologist. Regardless of the setting and the number of people involved, the reliability of monitoring depends on the integration of proper technical performance, accurate interpretation of responses, and their timely application to the surgical procedure.

ACKNOWLEDGMENTS

Grateful acknowledgment is made to Jay Shils, PhD, Lawrence Pitts, MD, and Michael McCaffrey, PhD, for their assistance and comments. We also thank Corey Astrom, ELS, for her editorial expertise and assistance with this manuscript.

BIBLIOGRAPHY

1. Wiet RJ, Herzon G. Surgery of the mastoid. In: Carrau R, ed. *Complications in Otolaryngology—Head and Neck Surgery*. Toronto: BC Decker; 1986:25–31.
2. Porter R, Kartush J. Diagnosis, evaluation and treatment of facial nerve disorders. In: Sanna M, Kirtane MV, Devaiah A, de Souza C, eds. *Otology Neurotology*. Noida: Thieme Publishers; 2012:329–347.
3. Green JD Jr, Shelton C, Brackmann DE. Iatrogenic facial nerve injury during otologic surgery. *Laryngoscope* 1994;104:922–926.
4. Krause F. *Surgery of the Brain and Spinal Cord*. New York: Rebman; 1912:738–743.
5. Benscoter B, Kartush J. Intraoperative facial nerve monitoring. In: Guntinas-Lichius O, Schaitkin BM, eds. *Facial Nerve Disorders and Diseases: Diagnosis and Management*. Stuttgart: Thieme; 2016:200–212.
6. Svider PF, Sunaryo PL, Keeley BR, Kovalerchik O, Mauro AC, Eloy JA. Characterizing liability for cranial nerve injuries: a detailed analysis of 209 malpractice trials. *Laryngoscope* 2013;123:1156–1162.
7. Slattery E, Kartush J. Medical and legal aspects of iatrogenic facial nerve paralysis. In: Guntinas-Lichius O, Schaitkin BM, eds. *Facial Nerve Disorders and Diseases: Diagnosis and Management*. Stuttgart: Thieme; 2016:380–392.
8. Gidley PW, Maw J, Gantz B, et al. Contemporary opinions on intraoperative facial nerve monitoring. *OTO Open* 2018;2:1–7.
9. Hughes CA, McMenamin P, Mehta V, Pillsbury H, Kennedy D. Otolaryngology workforce analysis. *Laryngoscope* 2016;126(Suppl 9):S5–S11.
10. Schwartz SR, Magit AE, Rosenfeld RM, et al. Clinical practice guideline (update): earwax (cerumen impaction). *Otolaryngol Head Neck Surg* 2017;156(Suppl 1):S1–S29.
11. Rosenfeld RM, Schwartz SR, Ron Cannon C, et al. Clinical practice guideline: acute otitis externa. *Otolaryngol Head Neck Surg* 2014;150(Suppl 1):S1–S24.
12. Randolph G, Dralle H, Abdullah H, et al. Electrophysiologic recurrent laryngeal nerve monitoring during thyroid and parathyroid surgery: international standards guideline statement. *Laryngoscope* 2011;121(Suppl 1):S1–S16.
13. Schneider R, Randolph GW, Dionigi G, et al. International neural monitoring study group guideline 2018 part I: staging bilateral thyroid surgery with monitoring loss of signal. *Laryngoscope* 2018;128:S1–S17.
14. Wu C, Dionigi G, Barczynski M, et al. International neuromonitoring study group guidelines 2018: part II: optimal recurrent laryngeal nerve management for invasive thyroid cancer—incorporation of surgical, laryngeal, and neural electrophysiologic data. *Laryngoscope* 2018;128:S18–S27.
15. Graciano AJ, Fischer CA, Coelho GV, Steck JH, Paschoal JR, Chone CT. Facial nerve dysfunction after superficial parotidectomy with or without continuous intraoperative electromyographic neuromonitoring: a prospective randomized pilot study. *Eur Arch Otorhinolaryngol* 2018;275:2861–2868. <https://doi.org/10.1007/s00405-018-5130-1>.
16. Harner SG, Daube JR, Beatty CW, Ebersold MJ. Intraoperative monitoring of the facial nerve. *Laryngoscope* 1988;98:209–212.
17. Rosenfeld RM, Nnacheta LC, Corrigan MD. Clinical consensus statement development manual. *Otolaryngol Head Neck Surg* 2015;153:S1–S14.
18. American Academy of Otolaryngology–Head and Neck Surgery. “Clinical consensus statements” are now “expert consensus statements”. *Bulletin* 2020;39:17.
19. National Institutes of Health. Consensus statement: acoustic neuroma. Presented at the NIH Consensus Development Conference, Bethesda, MD, 1991. Available at <http://consensus.nih.gov/1991/1991AcousticNeuroma087html.htm>
20. Bronstein D. Otolaryngologists report greater widespread use of intra-operative facial nerve monitoring. *ENT Today*, May 2014.
21. Silverstein H, Rosenberg SI, Flanner J, Seidman MD. Intraoperative facial nerve monitoring in acoustic neuroma surgery. *Am J Otol* 1993;14:524–532.
22. Pensak ML, Willging JP, Keith RW. Intraoperative facial nerve monitoring in chronic ear surgery: a resident training experience. *Am J Otol* 1994;15:108–110.
23. Sood AJ, Houlton JJ, Nguyen SA, Boyd Gillespie M. Facial nerve monitoring during parotidectomy: a systematic review and meta-analysis. *Otolaryngol Head Neck Surg* 2015;152:631–637.
24. Noss RS, Lalwani AK, Yingling CD. Facial nerve monitoring in middle ear and mastoid surgery. *Laryngoscope* 2001;111:831–836.
25. Wilson L, Lin E, Lalwani A. Cost-effectiveness of intraoperative facial nerve monitoring in middle ear or mastoid surgery. *Laryngoscope* 2003;113:1736–1745.
26. Heman-Ackah SE, Gupta S, Lalwani AK. Is facial nerve integrity monitoring of value in chronic ear surgery? *Laryngoscope* 2013;123:2–3.

27. Ruhl DS, Hong SS, Littlefield PD. Lessons learned in otologic surgery: 30 years of malpractice cases in the United States. *Otol Neurotol* 2013;34: 1173–1179.
28. Maw J, Gidley PW. Brief commentary on Gidley et al., “Contemporary opinions on intraoperative facial nerve monitoring”. *Otolaryngol Head Neck Surg* 2018;159:601–602.
29. ASET Board of Trustees. National Competency Skill Standards for Performing Intraoperative Neurophysiologic Monitoring. Available at: https://www.aset.org/files/public/IONM_National_Competency_Skill_Standards_Approved_2011.pdf
30. Nardi BA, Schwarz H, Kuchinsky A, Leichner R, Whittaker S, Sciabassi R. Turning away from talking heads: the use of video-as-data in neurosurgery. *Proceedings of the INTERCHI93 Conference on Human Factors in Computing*. Amsterdam, The Netherlands, April 24–29, 1993: Netherlands: IOS Press; 1993:327–334.
31. Skinner SA, Cohen BA, Morledge DE, et al. Practice guidelines for the supervising professional: intraoperative neurophysiological monitoring. *J Clin Monit Comput* 2014;28:103–111.
32. Gertsch JH, Moreira JJ, Lee GR, et al. Practice guidelines for the supervising professional: intraoperative neurophysiological monitoring. *J Clin Monit Comput* 2019;33:175–183.
33. Sciabassi RJ, Lofink R, Doyle E. NeuroNet: a distributed microprocessor network for clinical neurophysiology. In: Geisow MJ, Barret AN, eds. *Microcomputers in Medicine*. New York: Elsevier Science Publishers; 1987.
34. Skinner S, Holdefer R, McAuliffe JJ, Sala F. Medical error avoidance in intraoperative neurophysiological monitoring: the communication imperative. *J Clin Neurophysiol* 2017;34:477–483.
35. Skinner S, Sala F. Broca’s area: communication and collaboration in spine neuromonitoring: time to expect more, a lot more, from the neurophysiologists. *J Neurosurg Spine* 2017;27:1–6.
36. Krieger DN, Lofink RM, Doyle EL, Burk G, Sciabassi RJ. Neuronet: implementation of an integrated clinical neurophysiology system. *Med Instrum* 1987;6:296–303.
37. Wilkinson M. Message from the President. *Can Assoc Neurophysiol Monit Newslett* 2018;7:1–2.
38. Corrales E, Mudry A, Jackler R. Perpetuation of errors in illustrations of cranial nerve anatomy. *J Neurosurg* 2017;127:192–198.
39. Sugita K, Kobayashi S. Technical and instrumental improvements in the surgical treatment of acoustic neurinomas. *J Neurosurg* 1982;57: 747–752.
40. Kartush J, Bouchard K. Intraoperative facial nerve monitoring. In: Kartush J, Bouchard K, eds. *Neuromonitoring in Otolaryngology and Head and Neck Surgery*. New York: Raven Press; 1992.
41. Kartush J, Lee A. Intraoperative monitoring. In: Babu S, ed. *Practical Neurotology for the Otolaryngologist*. Austin, TX: Plural Publishing; 2013: 165–191.
42. Selesnick SH, Lynn-Macrae AG. The incidence of facial nerve dehiscence at surgery for cholesteatoma. *Otol Neurotol* 2001;22:129–132.
43. Naguib M, Lien CA, Meistelman C. Pharmacology of neuromuscular blocking drugs. In: Miller RD, ed. *Miller’s Anesthesia*. 8th ed. Philadelphia, PA: Saunders; 2014:958–994.
44. Lu IC, Wu CW, Chang PY, et al. Reversal of rocuronium-induced neuromuscular blockade by sugammadex allows for optimization of neural monitoring of the recurrent laryngeal nerve. *Laryngoscope* 2016;126: 1014–1019.
45. Yang LPH, Keam SJ. Sugammadex: a review of its use in anesthetic practice. *Drugs* 2009;69:919–942.
46. d’Honneur G, Slavov V, Merle JC, et al. Comparison of the effects of mivacurium on the diaphragm and geniohyoid muscles. *Br J Anaesth* 1996;77:716–719.
47. Rimaniol JM, Dhonneur G, Sperry L, Duvaldestin P. A comparison of the neuromuscular blocking effects of atracurium, mivacurium, and vecuronium on the adductor pollicis and the orbicularis oculi muscle in humans. *Anesth Analg* 1996;83:808–813.
48. Ibebunjo C, Srikant CB, Donati F. Morphological correlates of the differential responses of muscles to vecuronium. *Br J Anaesth* 1999;83: 284–291.
49. Zhou RY, Xu J, Chi FL, Chen LH, Li ST. Differences in sensitivity to rocuronium among orbicularis oris muscles innervated by normal or damaged facial nerves and gastrocnemius muscle innervated by somatic nerve in rats: combined morphological and functional analyses. *Laryngoscope* 2012;122:1831–1837.
50. Cai YR, Xu J, Chen LH, Chi FL. Electromyographic monitoring of facial nerve under different levels of neuromuscular blockade during middle ear microsurgery. *Chin Med J (Engl)* 2009;122:311–314.
51. Brauer M, Knuettgen D, Queser R, Doehn M. Electromyographic facial nerve monitoring during resection for acoustic neurinoma under moderate to profound levels of peripheral neuromuscular blockade. *Eur J Anaesthesiol* 1996;13:612–615.
52. Hester TO, Hasan A, McDonnell F, Valentino J, Jones R. Facial nerve monitoring under neuromuscular blockade. *Skull Base Surg* 1995;5: 69–72.
53. Shah R. Errors in otology: ahead of the curve. Interview of J.M. Kartush. *AAO-HNS Bulletin*. June 30, 2009. Available at: <https://bulletin.entnet.org/issue-archive/>
54. Kartush J. Kartush facial nerve monitoring protocol. *ENT Today*, May 2014. Available at: <https://www.enttoday.org/article/kartush-facial-nerve-monitoring-protocol/>?singlepage=1
55. Skinner SA, Transfeldt EE, Savik K. Surface electrodes are not sufficient to detect neurotonic discharges: observations in a porcine model and clinical review of deltoid electromyographic monitoring using multiple electrodes. *J Clin Monit Comput* 2008;22:131–139.
56. Calder HB, White DE. Facial nerve EMG monitoring. *Am J Electroneurodiagnostic Technol* 1996;36:28–46.
57. Rapp S, Rächinger J, Scheller C, Alfieri A, Strauss C, Prell J. How many electromyography channels do we need for facial nerve monitoring? *J Clin Neurophysiol* 2012;29:226–229.
58. Prass RL, Luders H. Acoustic (loudspeaker) facial electromyographic monitoring part 1: evoked electromyographic activity during acoustic neuroma resection. *Neurosurgery* 1986;19:392–400.
59. American Society of Anesthesiologists. Standards for basic anesthetic monitoring. Available at: <https://www.asahq.org/standards-and-guidelines/standards-for-basic-anesthetic-monitoring>
60. Kartush JK, Niparko JK, Bledsoe SC, Graham MD, Kemink JL. Intraoperative facial nerve monitoring: a comparison of stimulating electrodes. *Laryngoscope* 1985;95:1536–1540.
61. Huang X, Ren J, Xu J, et al. The utility of “low current” stimulation threshold of intraoperative electromyography monitoring in predicting facial nerve function outcome after vestibular schwannoma surgery: a prospective cohort study of 103 large tumors. *J Neurooncol* 2018;138: 383–390.
62. Schmitt WR, Daube JR, Carlson ML, et al. Use of supramaximal stimulation to predict facial nerve outcomes following vestibular schwannoma microsurgery: results from a decade of experience. *J Neurosurg* 2013;118: 206–212.
63. Prass R, Lüders H. Letter to the Editor: constant-current versus constant-voltage stimulation. *J Neurosurg* 1985;62:622–623.
64. Israel Z, Moffitt M, Montgomery EB, et al. The rationale driving the evolution of deep brain stimulation to constant-current devices. *Neuromodulation* 2015;18:85–88.
65. Pearlman RC, Isley MR, Ganley JC. Electrical artifact during intraoperative electromyographic neuromonitoring. *Am J Electroneurodiagnostic Technol* 2008;48:107–118.
66. Acioly MA, Liebsch M, Carvalho CH, Gharabaghi A, Tatagiba M. Transcranial electrocortical stimulation to monitor the facial nerve motor function during cerebellopontine angle surgery. *Neurosurgery* 2010;66(6 Suppl Operative):354–362.
67. Dong CC, MacDonald DB, Akagami R, et al. Intraoperative facial motor evoked potential monitoring with transcranial electrical stimulation during skull base surgery. *Clin Neurophysiol* 2005;116:588–596.
68. MacDonald DB. Intraoperative motor evoked potential monitoring: overview and update. *J Clin Monit Comput* 2006;20:347–377.
69. Akagami R, Dong CC, Westerberg BD. Localized transcranial electrical motor evoked potentials for monitoring cranial nerves in cranial base surgery. *Neurosurgery* 2005;57(1 Suppl):78–85.
70. Fukuda M, Oishi M, Takao T, Saito A, Fujii Y. Facial nerve motor evoked potential monitoring during skull base surgery predicts facial nerve outcome. *J Neurol Neurosurg Psychiatry* 2008;79:1066–1070.
71. Matthies C, Raslan F, Schweitzer T, Hagen R, Roosen K, Reiners K. Facial motor evoked potentials in cerebellopontine angle surgery: technique, pitfalls and predictive value. *Clin Neurol Neurosurg* 2011;113: 872–879.
72. Deletis V, Fernandez-Conejero I, Ulkatan S, Constantino P. Methodology for intraoperatively eliciting motor evoked potentials in the vocal muscles by electrical stimulation of the corticobulbar tract. *Clin Neurophysiol* 2009;120:336–341.
73. Deletis V, Urziza J, Ulkatan S, Fernandez-Conejero I, Lesser J, Misita D. The feasibility of recording blink reflexes under general anesthesia. *Muscle Nerve* 2009;39:642–646.
74. Möller AR, Jannetta PJ. On the origin of synkinesis in hemifacial spasm: results of intracranial recordings. *J Neurosurg* 1984;61:569–576.
75. Kim CH, Kong DS, Lee JA, Park K. The potential value of the disappearance of the lateral spread response during microvascular decompression for predicting the clinical outcome of hemi-facial spasms: a prospective study. *Neurosurgery* 2010;67:1581–1588.
76. Sekula RF, Bhatia S, Frederickson AM, et al. Utility of intraoperative electromyography in microvascular decompression for hemifacial spasm: a meta-analysis. *Neurosurg Focus* 2009;27:E10.
77. Choi J, Yang S, Kim J-S, Han JH, Park KS. Predictive value of intraoperative blink reflex monitoring for surgical outcome during microvascular decompression for hemifacial spasm. *Clin Neurophysiol* 2020; 131:2268–2275. <https://doi.org/10.1016/j.clinph.2020.06.025>
78. Stecker MM, Patterson T, Netherton BL. Mechanisms of electrode induced injury. Part 1: theory. *Am J Electroneurodiagnostic Technol* 2006;46: 315–342.
79. Patterson T, Stecker MM, Netherton BL. Mechanisms of electrode induced injury. Part 2: clinical experience. *Am J Electroneurodiagnostic Technol* 2007;47:93–113.
80. Netherton BL, Stecker MM, Patterson T. Mechanisms of electrode induced injury. Part 3: practical concepts and avoidance. *Am J Electroneurodiagnostic Technol* 2007;47:257–263.
81. Haenggeli A, Richter M, Lehmann W, Dulguerov P. A complication of intraoperative facial nerve monitoring: facial skin burns. *Am J Otol* 1999; 20:679–682.
82. Love JT Jr, Marchbanks JR. Injury to the facial nerve associated with the use of a disposable nerve stimulator. *Otolaryngology* 1978;6:61–64.

83. Kelley TF, Leonetti JP. Facial nerve stimulation at the internal auditory canal: an animal model and experimental assessment. *Laryngoscope* 2003;113:643–646.
84. Lowry TR, Gal TJ, Brennan JA. Patterns of use of facial nerve monitoring during parotid gland surgery. *Otolaryngol Head Neck Surg* 2005;133:313–318.
85. Harper CM. Intraoperative cranial nerve monitoring. *Muscle Nerve* 2004;29:339–351.
86. Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 2nd ed. Philadelphia, PA: F.A. Davis Company; 1989.
87. Romstöck J, Strauss C, Fahlbusch R. Continuous electromyography monitoring of motor cranial nerves during cerebellopontine angle surgery. *J Neurosurg* 2000;93:586–593.
88. Prell J, Rampp S, Rachinger J, Scheller C, Naraghi R, Strauss C. Spontaneous electromyographic activity during microvascular decompression in trigeminal neuralgia. *J Clin Neurophysiol* 2008;25:225–232.
89. Prell J, Rachinger J, Scheller C, Alfieri A, Strauss C, Rampp S. A real-time monitoring system for the facial nerve. *Neurosurgery* 2010;66:1064–1073.
90. Prell J, Rampp S, Romstock J, Fahlbusch R, Strauss C. Train time as a quantitative electromyographic parameter for facial nerve function in patients undergoing surgery for vestibular schwannoma. *J Neurosurg* 2007;106:826–832.
91. Nelson KR, Vasconez HC. Nerve transection without neurotonic discharges during intraoperative electromyographic monitoring. *Muscle Nerve* 1995;18:236–238.
92. Minahan R, Mandir A. Neurophysiologic intraoperative monitoring of trigeminal and facial nerves. *J Clin Neurophysiol* 2011;28:551–565.
93. Jackler RK. *Atlas of Skull Base Surgery and Neurotology*. 2nd ed. New York: Thieme; 2009.
94. Yingling CD, Gardi JN. Intraoperative monitoring of facial and cochlear nerves during acoustic neuroma surgery. *Neurosurg Clin N Am* 2008;19:289–315.
95. Ashram YA, Jackler RK, Pitts LH, Yingling CD. Intraoperative electrophysiologic identification of the nervus intermedius. *Otol Neurotol* 2005;26:274–279.
96. Porter RG, LaRouere MJ, Kartush JM, Bojrab DI, Pieper DR. Improved facial nerve outcomes using an evolving treatment method for large acoustic neuromas. *Otol Neurotol* 2013;34:304–310.
97. Brackmann DE. Fenestrated suction for neuro-otologic surgery. *Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol* 1977;84:975.
98. House WF. Surgical exposure of the internal auditory canal and its contents through the middle, cranial fossa. *Laryngoscope* 1961;71:1363–1385.
99. Goin DW. Proximal intratemporal facial nerve in Bell's palsy surgery. A study correlating anatomical and surgical findings. *Laryngoscope* 1982;92:263–272.
100. House WF, Hitselberger WE. The transcochlear approach to the skull base. *Arch Otolaryngol* 1976;102:334–342.
101. Gantz BJ, Fisch U. Modified transotic approach to the cerebellopontine angle. *Arch Otolaryngol* 1983;109:252–256.
102. Möller AA, Jannetta PJ. Monitoring facial EMG responses during microvascular decompression operations for hemifacial spasm. *J Neurosurg* 1987;66:681–685.
103. Wilkinson MF, Kaufmann AM. Monitoring of facial muscle motor potentials during microvascular decompression for hemifacial spasm: evidence of changes in motor neuron excitability. *J Neurosurg* 2005;103:64–69.
104. Harner SG, Daube JR, Ebersold MJ. Electrophysiologic monitoring of facial nerve during temporal bone surgery. *Laryngoscope* 1986;96:65–69.
105. Sargent E, Kartush JM. Lasers in otology. *Adv Otolaryngol Head Neck Surg* 1994;8:187–201.
106. Jahrsdoerfer RA, Lambert PR. Facial nerve injury in congenital aural atresia surgery. *Am J Otol* 1998;19:283–287.
107. Schwartz D, Rosenberg S. Facial nerve monitoring during parotidectomy. In: Kartush J, Bouchard K, eds. *Neuromonitoring in Otolaryngology and Head and Neck Surgery*. New York: Raven Press; 1992:121–130.
108. Lydiatt DD. Medical malpractice and facial nerve paralysis. *Arch Otolaryngol Head Neck Surg* 2003;129:50–53.
109. Hong SS, Yheulon CG, Sniezek JC. Salivary gland surgery and medical malpractice. *Otolaryngol Head Neck Surg* 2013;148:589–594.