Intraoperative monitoring of the facial nerve

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ABSTRACT

Objectives: Intraoperative facial nerve monitoring (FNM) was used to assess the anatomical and physiological integrity of the facial nerve during neurotological, otological, and parotid surgeries. Why monitor the facial nerve? There are several reasons to perform FNM. First of all, it alerts the surgeon when something potentially harmful was carried out to the nerve. When the regional anatomy is confusing, it allows positive identification of the nerve. Finally, the integrity of the facial nerve is assessed at the end of the procedure.

Methods: Eighty seven patients who underwent intraoperative monitoring of facial nerve between 1991 and 1996 at the King Fahad National Guard Hospital, Riyadh, Kingdom of Saudi Arabia were studied. The device used was the nerve integrity monitor manufactured by Xomed. The device works when a physiologically intact facial nerve is stimulated, the muscles that are innervated by this nerve will contract.

Results: In otologic surgery, FNM signals the unintentional mechanical stimulation of the facial nerve during surgery, it predicts the dehiscence in the bony covering of the nerve and it allows mapping the nerve through soft tissue and bone. Facial nerve injury is the most devastating complication of otologic surgery particularly when anatomy is distorted by previous surgery, granulation tissue or cholesteatoma distorts anatomy, and in rare instances by an anolomous course. However, it is unlikely to be injured when it has been identified. The surgical treatment of recurrent parotid tumor can be challenging. The main aim of surgery is complete removal of tumor and preservation of facial nerve.

Conclusion: Facial nerve monitoring assists early nerve identification and decreases the nerve trauma. However, as an adjunct, it has proved extremely beneficial. In this paper, the facial nerve was monitored intraoperatively in 87 cases with different pathologies. It is certainly helpful in revision mastoid surgery and in removal of recurrent parotid tumor. Intraoperative monitoring of the facial nerve plays an important role in identification and preservation of the facial nerve during otological and parotid surgeries.

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Intraoperative monitoring (IOM) of the facial nerve during surgery decreased trauma to the nerve, thereby preserving the function, which in turn is of great importance to both the physician and the patients. The surgeon who deals with the facial nerve has been concerned with how to achieve therapeutic success, and still maintain the integrity of the facial nerve. Application of electromyogram (EMG) and electro-neuronography techniques such as IOM is neither new nor revolutionary. Parsons[1] described EMG monitoring of facial nerve in 1966. Delgado et al[2] in 1979 and Sugita and Kobayashi[3] in 1982 described separately, constant EMG monitoring of facial nerve in acoustic neuroma surgery. The "facial nerve protectors" performed the first acoustic monitoring of the facial nerve, a surgical assistant was charged with the duty of continuously monitoring the patient's face and shouting out at the slightest facial movement.[4] Jako[5] introduced the photoelectric device applied to the cheek, which

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detected facial twitching and triggered an audible signal. Silverstein[6] modified the apparatus and incorporated a louder alarm and a more sensitive strain gauge sensor. Sugita and Kobayashi[3] used disc accelerometers to transduce facial motion into electric signal. These signals were amplified and fed into the operating room (OR) through a loud speaker. Acoustic monitoring had one significant weakness, namely, lack of specificity for facial muscle activity due to false positive responses and masseter muscle contraction sometimes led to facial nerve section. Moller and Janetta[7] developed techniques using the intramuscular EMG electrodes in which signals were amplified and fed into the OR through a loudspeaker thus providing immediate responses to the surgeon. The earlier techniques were relatively insensitive and required gross facial movements to elicit a response, and were less specific. The intramuscular EMG electrodes afforded absolute response specificity and sensitivity. Metson and Nadol,[8] and Prass[9] worked together and developed the more specific and sensitive, nerve integrity monitor (NIM-2), now distributed by Xomed. This unit consists of an EMG amplifier, oscilloscopic display, audible response mechanism, and a stimulator probe. Identification and preservation of the facial nerve during surgical procedures is at times difficult even in the most capable hands. Intraoperative facial nerve monitoring (FNM) has an important role in identification and preservation of the facial nerve during various otological and neurological procedures. A nerve integrity monitor was routinely used at the King Fahad National Guard Hospital (KFNGH), Riyadh, Kingdom of Saudi Arabia, from 1991 to 1996 in the following procedures: 1) Decompression of facial nerve in trauma (n=9), 2) Revision middle ear and mastoid surgery (n=60), 3) Iatrogenic facial paralysis (n=2), 4) Parotid surgery (n=15), 5) Stab wounds of neck and face (n=2).

Methods. Eighty-seven patients who underwent intraoperative monitoring of facial nerve between 1991 and 1996 at KFNGH were studied. Muscle relaxants are always avoided. Paired subdermal needle electrodes are used, one at the nasolabial fold and the other at lateral orbicularis oculi. A ground electrode was placed in the forehead and another needle electrode was placed in the shoulder for monopolar stimulator. Electrodes were secured with clear plastic adhesives for visual confirmation of placement. Electrode impedance were checked and kept below 5M ohm with electrode imbalances of less than 1 K ohm. The monitoring detector probe was clamped around the cables near their attachments to the electrosurgical units. Probe activates its speaker muting circuit during electrocautery use. The current stimulus was set at 0.2mA with a rate of 4 pulses per second. The NIM-2 speaker volume was set so that the surgeon could hear the acoustic response. During the procedure, the electrical stimulator was placed on soft tissue, and the measured stimulus current read on the NIM-2's display thus confirming current flow. An insulated stimulator, Prass Flush-tip probe, was used to prevent shunting the current away from the target structure. The NIM-2 provides continuous immediate monitoring of the resting function of facial muscles. It also allows for both visual and auditory feedback to the surgeon. The following are the common evoked facial EMG responses provided by the NIM-2 allowing the surgeon to interpret the ongoing surgical events. The pulse response is due to electrical stimulation, and it is a series of precisely timed clicks. The burst response is due to direct surgical manipulation, and the clicks of acoustic feedback are synchronous with manipulation. The train response is due to traction, pressure, irrigation, and thermal trauma, and the auditory feedback is a series of repetitive asynchronous clicks resembling popping popcorn.

Results. ***Revision surgery on middle ear and mastoid (n=60).*** In this surgery, it is difficult to determine what is cholesteatoma, granulation tissue or the facial nerve. In these 60 patients, only 3 had facial weakness after primary surgery. The remaining 57 had no facial weakness. The standard landmark of facial nerve was used during the procedure to identify the anatomy of the facial nerve. The facial nerve was identified in all cases by visual inspection. An exposed or dehiscent facial nerve was found in 27 cases. The most common area of dehiscence was the lower tympanic segment. This was either due to previous surgery or due to the disease process. Direct nerve stimulation and the acoustic response from the NIM-2 confirmed identification of the facial nerve. The pulse response of the later confirmed the facial nerve in 20 of 27 cases. However, the monitor did not identify the nerve in 7 patients, although the exposed nerve was seen to be in its normal anatomical position. An inadequately charged battery and dislodged electrodes were the cause in 3 cases but no reasons could be found in the other 4 patients. The failure of the monitor initially caused concern of possible nerve damage, though this was not the case. No muscle relaxants were used. In 5 other patients, where the exposed facial nerve was covered with cholesteatoma, fibrogranulation tissue and tympanosclerotic plaques, the NIM-2 clearly produced burst EMG responses when any attempt was made to remove this diseased tissue. The acoustic burst responses were obtained while dissecting the cholesteatoma from the nerve surface, when traction was applied to remove the fibrogranulation tissue and while attempting to remove tympanosclerotic plaques from the facial nerve canal. In 3 cases a segment of facial nerve was seen to bulging out on the oval window like a

neuroma and under the microscope resembled cholesteatoma. Manipulation elicited burst responses indicating a dehiscent nerve. In all revision cases, the total eradication of the disease was achieved. None developed postoperative facial paralysis. There were 3 patients who had partial preoperative facial weakness. None of these had any worsening of the weakness. One patient with diabetes mellitus (DM) and tympanosclerosis developed facial weakness on the 10th postoperative day, which responded well to conservative therapy (namely steroid therapy). In primary chronic ear surgery, an experienced Otologist may not need the NIM-2. It may be a valuable tool in a residency-training program to bolster the confidence of the trainee. In cases of revision surgery, NIM-2 may contribute to an improved outcome as it allows more complete removal of bone. In 2 of the cases, the use of NIM-2 in the primary procedure may have prevented iatrogenic neural damage. Extensive granulation tissue, excessive bleeding, and destruction of the bony canal by the disease process were implicated in this damage. Hepatic cirrhosis, DM and hypertension further complicated the procedure and outcome. Thus, in primary chronic ear surgery it may be recommended in the following situations: 1) Prolonged history of otorrhea associated with pain. 2) Moderate to severe mixed sensorineural hearing loss (SNHL) with cholesteatoma or granulation tissue. 3) Extensive tympanosclerosis. 4) Granulating otitis media namely the presence of granulation tissue polyp. 5) Radiological evidence of osteolysis (CT Scan) by cholesteatoma or granulation tissue. 6) Patients with hepatic disease, hypertension and diabetes. A literature review showed that chronic ear surgery carries a certain risk to facial nerve. Miehlke[10] reported an incidence of 1.6% facial nerve injury in primary surgery and 11% in cases of revision surgery. Wiet[11] described 10% iatrogenic facial palsy in revision cases. However, May[12] found an incidence of only 0.1% in a review of primary and secondary cases treated by 10 experienced Otologists. In congenital ear disease, particularly atresia, the potential for injury to facial nerve is the single greatest deterrent to surgery. Most leading Otologist performing these procedures recommended the use of intraoperative monitoring (Linstorm,[13] Jahrsdoerfer et al,[14] Molony and De La Cruz,[15] Silverstein et al[16]). Harner et al[17] recommended IOM in all skull-based surgery, and advocated that monitoring be carried out by experienced personnel in EMG, not by the surgical team or the OR personnel. However, during otologic surgery, continuous facial nerve monitoring offers the surgeon an additional degree of reassurance when drilling or dissecting close to the facial nerve.

Temporal bone fractures with facial paralysis (n=9). There were 9 cases of facial paralysis associated with temporal bone fractures out of 32 cases of temporal bone fractures treated since 1990. Seven of these had longitudinal fractures and 2 had transverse fractures. Associated lesions included conductive deafness due to ossicular disruption in 2 cases, total SNHL in one case, severe vertigo in 2 cases, and cerebrospinal fluid leakage in one case. Although facial paralysis was noted in 2 patients immediately after the injury, these were not treated as life threatening poly trauma took precedence. The other 7 cases were referred to the Ear, Nose and Throat Department on a delayed basis again due to other serious injuries. Electroneuronography facility was not available but EMG studies showed evidence of degeneration. Exploration of the facial nerve was carried out from 4 to 6 weeks after the injury in these 9 cases, using NIM-2. In the longitudinal fractures (n=7), localization of lesion during surgery was found as follows: a) Bony fragments impinging on the nerve involving the fallopian canal where it leaves the compact labyrinthine capsule to run along the medial wall of the tympanic cavity and also between the tympanic and mastoid segments in 4 cases. b) Intraneural hematoma and fibrosis in 3 cases in labyrinthine segment. Although these patients had clinical facial palsy, intraoperative monitoring identified the nerves with burst response, and train EMG activities were observed during dissection, drilling and manipulation of nerve trunk for repair work and drainage of hematoma. The fibres, which were intact, generated the potentials although at higher stimulus intensity (0.5-1mA). In 2 patients with transverse fractures, multiple lesions were noted, in the labyrinthine segment and in the mastoid segments, and the facial nerves were partially transacted distal to the geniculate ganglion with bony fragments lying between the sectioned parts. During surgery the bony fragments were removed, hematoma drained and the transacted nerves sutured, after excision of the fibrous tissue. In these cases, great auricular nerve grafts were used to repair the partial defects, as the ends of the nerve could not be approximated after excision of the fibrous scar. The NIM-2 recorded vigorous burst activity during dissection and a few episodes of train activity during repair.

Stab wound of the neck and face (n=2). In 2 patients with stab wounds in the face and neck, the upper trunk of the facial nerve to be sectioned was found in one, and the mandibular branch in the other. These cases were operated upon immediately within 4-6 days. One case with upper trunk injury was anastomosed without any difficulty but it took some time to isolate the ends of mandibular branch for repair, namely, end-to-end anastomosis. The NIM-2 recorded the pulse response when the main nerve trunk was electrically stimulated.

Iatrogenic facial paralysis (n=2). Iatrogenic paralysis occurred in 2 patients after tympanomastoidectomy. The incidence is 2 in 2250

cases of such procedures performed in this hospital. Both patients were female. One had hypertension and DM and the other had hepatic failure with bleeding diathesis. Extensive cholesteatoma and fibrogranulation with erosion or dehiscence of the tympanic segment of the facial nerve was noted during surgery. The surgeons were not aware of any accidents either with the drill or the micro instruments during the procedure. There was immediate weakness of the facial nerve after surgery. Nerve integrity monitor was not used in these cases during primary surgery. However, the monitor was used during re-exploration for repair of the nerve. In both cases, the facial nerve was partially transacted, and in one case, there was a bony fragment impinging on the nerve. This was removed and the nerve was repaired by direct end-to-end anastomosis. In the 2nd case, a great auricular nerve graft was used to repair the defect as 25% (approximately) of the nerve and trunk had been sectioned. The nerve was decompressed distally and proximally, and then grafting was carried out in a standard fashion. Again, vigorous burst activity and episodes of train activity were observed in NIM-2.

Parotidectomy (n=15). In 15 patients with parotid tumor, the facial nerve was identified and preserved in 13 cases with benign lesion. However, in 2 cases with malignancy the nerve had to be sacrificed due to its extensive involvement by malignancy. The train responses in nerve sectioning were observed in these 2 cases indicating complete sectioning of the facial nerve. During surgery in these cases, the NIM-2 often alerted the surgeon to neuorotonic activity even when there was no motion of the face. Rarely the assistant would note the facial muscle activity that was not detected by the monitor. Probably due to activation of the branches to the muscle that were not being monitored. The nerve stimulator can identify isolated nerve segments. It was thought initially that the monitor would be most beneficial to the occasional parotid surgeon, but now it may be considered worthwhile for even experienced operators. It is likely to call attention to unsuspected nerve activity. Olsen et al[18] discussed the importance of intraoperative monitoring of the facial nerve during parotidectomy in cases of recurrent pleomorphic adenoma. None of his patients had postoperative weakness even of the marginal branches.

Discussion. Prass and Luders[9] in 1986 divided the train activity potentials into 2 varieties according to the frequency, interval regularity, and pattern of build up and decline of motor unit potentials: 1) Bomber potentials - high frequency activity, uniform interval with sonic characteristics of an aeroplane engine. 2) Popping popcorn - low frequency, and irregular intervals with sonic characteristics of popping popcorn. Episodes of train activity were observed more frequently in cases of decompression of the nerve in temporal bone fracture patients than in revision mastoidectomy and parotidectomy patients. The rapid and high frequency mechanical trauma as produced by a drill may provide the best stimuli to excite the facial nerve axons as mechanoreceptors. So, the rapidity of compression is more important than the absolute amount of nerve compression. Intraoperative monitoring of neuroelectric potentials is a well-accepted modality in Neurotology and skull base procedures. Application of this monitoring in chronic ear surgery as routine is debatable, but in revision surgery in the middle ear and mastoid, it is preferable though perhaps not mandatory (Pensak[19] 1994). It is not a substitute for surgical skills in decompression of the facial nerve entrapped by temporal bone fractures and in parotid surgery.

Goals of intraoperative monitoring. The goals of intraoperative FNM are 3-fold: 1) Enhance early nerve identification. 2) Enhance neural preservation by minimizing trauma. 3) Assess neural integrity after the procedure is over.

Nerve localization. During surgery, the monitor will indicate unexpected facial contraction by noise. Mechanical stimulation of facial nerve may occur when: 1) a burr touches the facial nerve sheath; 2) the exposed nerve is touched with an instrument or suction nozzle; 3) The exposed nerve is stretched during removal of cholesteatoma of granulation tissue, and 4) thermal trauma due to irrigation with saline. Nerve localization is most readily performed by the use of electrical stimulator. Monopolar stimulation is more sensitive, but less specific than bipolar stimulation. The facial nerve stimulator was also used during surgery to map the position of the facial nerve in bone or tissue, to determine the integrity of the bony covering of the nerve, and to test the electrical integrity of the facial nerve during dissection. Prior to and during drilling the posterior canal wall, the stimulator probe was used to locate the nerve within the bone. "Burst" of EMG activity was elicited during skeletonization of the facial nerve or lowering the facial bridge during open mastoidectomy in revision cases. The normal distance from annulus to the facial nerve is 4 mm, since in revision cases less bone may be present, a lower current level will stimulate the exposed nerve (0.1-0.2mA) while higher levels (0.5-1.0mA) will be required if the nerve is cover by bone. In revision mastoidectomy cases where the fallopian canal was intact with no disease, current levels of 1mA were required to get an EMG response, but in patients where the bony facial nerve canal was thinned by cholesteatoma or granulation tissue, lower current levels were required (0.3-0.45) to get EMG responses. This information suggests that the disease process was destroying the bone. In cases where the nerve was exposed and surrounded by unhealthy

tissue, it was observed that manipulation produced burst activity, but much less than when an intact nerve was manipulated. Thus, NIM-2 helped to predict whether the bone covering the facial nerve was intact in the presence of chronic ear disease. The facial nerve has a higher stimulation threshold when covered by bone than it does when exposed. In revision cases where the nerve was already exposed and adherent to diseased tissue vigorous burst of EMG activity, namely, "train" responses were obtained when attempts were made to remove the diseased tissue, which caused lateral movement of the nerve trunk in the bony canal. In cases of decompression of nerve for fractured temporal bone, direct electrical stimulation of the intact nerve fibres produced typical "pulse" response at higher current intensity level namely 1mA. Burst responses were noticed while skeletonization was carried out to decompress the facial nerve proximal and distal to the site of lesion, namely, hematoma and bony fragments impinging on the nerve, indicating continuity of some of the fibres. In 2 cases with complete sectioning of the nerve, no response was observed after manipulation for dissection and suturing for anastomosis. Once the nerve was physically identified, direct electrical stimulation with NIM-2 monitoring allowed confirmation by production of the typical pulse pattern of EMG response.

Detection of neural trauma. Reliance on mechanically evoked responses caused by blunt manipulation and traction will minimize neural trauma. These responses are generated by changes in ion permeability consequent to neural deformation. Cold saline irrigation may also result in these changes. Direct manipulation of the nerve usually results in a single compound muscle action potential synchronous with a surgical maneuver. This is a burst response due to blunt trauma and it is not always time locked and thus the surgeon must interpret this in the context of ongoing surgical events. This is contrary to electrically stimulated response, which is always timed locked. Vigorous burst activity was noted occasionally during removal of cholesteatoma, granulation tissue, and bony fragments from the facial nerve. Removal of this tissue by cup forceps involved side-to-side or medial-lateral movements of the exposed facial nerve. This traction or prolonged dissection caused multiple asynchronous action potentials, which persisted for seconds. Prolonged periods of train potentials may indicate some degree of trauma due to surgical manipulation. Temporary cessation of dissection and release of traction was usually beneficial. The facial nerves that appeared to be relatively active in producing burst EMG activity and maintained this throughout the entire case exhibited the best early postoperative facial function. But nerves that were or had become quiet with respect to elicitation of burst activity during manipulation appeared to exhibit poor facial nerve function one week after surgery.

Assessment of neural integrity. At the end of each procedure, stimulating the nerve proximal to the site of dissection assessed neural integrity. Electrical stimulus was applied and pulse response was obtained. In an intact nerve, a fraction of the usual current intensity (0.2mA) was required to elicit the acoustic response. Damaged nerves as in cases of temporal bone fractures with bony impingement, and intraneural hematoma, required a higher current intensity (1mA). Where the nerve was partially sectioned or cut, no pulse or burst response was observed indicating no conduction of impulses except in one patient with iatrogenic trauma where less than one third of facial nerve was cut. Vigorous burst response was noted during manipulation for suturing the ends.

False alarms. A false alarm with regard to facial nerve monitoring is a relative term. Any facial muscle movement can cause EMG responses. Surgeon repositioning the head or touching the face can cause the movement. These can be triggered by external electrical signals when there is no facial movement, but muting devices in NIM-2 reduce these. Both mono and bipolar electrocautery can produce false alarms, but NIM-2 has a special muting device that cancels these alarms due to electrocautery.

In conclusion, intraoperative FNM with NIM-2 is not a replacement for sound anatomical knowledge and competence of an experienced surgeon. It provides immediate feedback to the surgeon in the form of burst, train and pulse acoustic EMG activity, and the surgeon has the opportunity to personally interpret various episodes of evoked EMG activities in the context of ongoing surgical events. Still, interpretation of mechanically evoked potential must be performed with great care. Electrical silence may be the consequence of absence of stimulation, severe injury, and complete transaction of nerve. Prolonged periods of train activity may indicate some degree of trauma, whereas most burst activity is probably generated by an abrupt change in ionic permeability secondary to cell membrane depolarization. Nerves that are significantly manipulated during surgery may demonstrate fatigue, wherein mechanically evoked potentials become more and more difficult to obtain. The most valuable feature of NIM-2 monitoring is for early localization and identification of the nerve.

Once the nerve is identified positively, the surgeon may use any technique to proceed in a known to unknown fashion to dissect along the facial nerve contour. The burst pattern of EMG activity was produced normally by blunt dissection therefore,

many will use sharp dissection when a frequent episode of burst activity was elicited. Frequent burst activity with mild mechanical manipulation, as happens during cholesteatoma, granulation tissue removal in revision mastoidectomies, and during decompression of facial nerves, may be considered a sign of functional integrity of the nerve. A decline in burst activity with a given degree of mechanical manipulation was often associated with poorer early postoperative facial function. Unless the burst activity is the result of sharp dissection of a cutting maneuver, it may be interpreted as an indication of relative integrity of the facial nerve distal to the site of stimulation. On the other hand, a decline in burst activity may give a false sense of security since more vigorous dissection often followed the period of time when the facial nerve "quieted down". Repetitive train activity is to be presumed due to increase in axonal membrane permeability to such a level that membrane depolarization is maintained above action potential threshold, thus indicating an established nerve injury. The pattern of train activity maybe regular, irregular or even bizarre in character with a relatively gradual or sudden build up and decline. These activities often are not timed locked hence the cause effect relationship is often difficult to appreciate in an ongoing manner. However, when the train activity disappears within a minute or after removal of an instrument it suggests that the nerve axons have recovered to a repolarized state, but when it fails to disappear it may indicate a more lasting change in axonal membrane permeability. Pre-existing neural ischemia renders the nerve more susceptible to ischemia with traction. This may explain the marked sensitivity of the nerve to traction in producing EMG activity in cases of patients with preoperative partial facial weakness. Facial nerve less involved by disease, for example cholesteatoma or fibrogranulation tissue, appeared to be more active in producing burst activity with a given degree of mechanical trauma than those grossly involved with disease. Also, previous trauma at or distal to the ongoing site of facial nerve manipulation seemed to have produced less burst EMG activity for mechanical trauma. This had been observed in cases of temporal bone fractures cases when distal to proximal dissection was carried out in order to remove the bone fragments, drain the hematoma and suturing or grafting the nerve. Safe handling of the facial nerve during ear surgery requires a sound knowledge of the course and position of the facial nerve. Intraoperative FNM does not replace the knowledge and skill, but does add an extra dimension of safety during ear surgery. Occasionally, even a most experienced surgeon may have considerable difficulty in preserving the facial nerve. Routine intraoperative monitoring and simulation of facial nerve confers certain advantages to the surgeon. A monitor cannot make the surgeon safe, and the absolute prerequisite for its safety use is sound otologic training. The monitor allows the surgeon to talk (stimulate) and listen (EMG response) to the facial nerve during the otology surgery. Intraoperative monitoring thus enhances the good care, which the otologists are already providing to the patients. It is an admission of wisdom not cowardice to use FNM.

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