

**TITLE: Facial Nerve Trauma**

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The facial nerve is a very complex and unique nerve in both its anatomical course and function. Due to this complexity it is understandable that it is involved in many of the pathologic entities that affect the head and neck. One of these entities is trauma.

We will begin our discussion of facial nerve trauma by first examining the anatomy and function of the facial nerve. The anatomy of the facial nerve can be broken down into three major segments; intracranial, intratemporal, and extratemporal. The intracranial segment refers to that portion of the nerve that runs from the brainstem to the internal auditory canal (IAC). It can be further divided into two components, the motor root and the nervus intermedius. The motor root, as the name implies, carries the motor fibers of the facial nerve. The nervus intermedius on the other hand carries the facial nerve's preganglionic parasympathetic fibers and special afferent sensory fibers. These two components join near the IAC to form the common facial nerve. The intratemporal segments begin as the nerve enters the IAC.

The first intratemporal segment is referred to as the meatal segment. It is the portion of the facial nerve traveling from the porus acusticus to the meatal foramen of IAC. It travels in the anterior superior portion of the IAC along with three other nerves; the superior vestibular nerve in the posterior superior portion, the inferior vestibular nerve in the posterior inferior portion, and the cochlear nerve in the anterior inferior portion. The length of the meatal segment is roughly 8-10mm. At the end of the IAC near the meatal foramen, the diameter narrows from 1.2mm to 0.68mm. This is the narrowest portion of the IAC and just so happens to be where the next segment of the facial nerve is located. This segment is the labyrinthine segment. It runs from the fundus to the geniculate ganglion, and is the shortest of all the intratemporal segments at 2-4mm in length. The geniculate ganglion houses the sensory and taste cells to the anterior 2/3 of the tongue and palate. It is also where the first branch of the facial nerve comes off of, the greater superficial petrosal nerve. This branch joins the deep petrosal nerve to form the vidian nerve, and is responsible for providing parasympathetic fibers to the lacrimal gland. The portion of the facial nerve that runs from the geniculate ganglion to the second genu is termed the tympanic segment. It is roughly 11mm in length, and is the most commonly injured portion of

the facial nerve during middle ear/mastoid surgery. One of the major reasons for injury is secondary to the fact that the nerve is dehiscant in this area in 40-50% of the population. The next segment is termed the mastoid segment. It is the part of the facial nerve that runs from the second genu to the stylomastoid foramen, a length of roughly 12-14mm. Its course takes it between the incus and horizontal semicircular canal. It is at the end of this segment that the facial nerve gives off a branch to the stapedius muscle and the chorda tympani.

Once the facial nerve exits the stylomastoid foramen it gives off the postauricular nerve that supplies the external auricular and occipitofrontalis muscles as well as the branches to the posterior belly of the digastric and stylohyoid muscles. It then enters the parotid gland splitting the gland into a superficial and deep lobe. Within the parotid, the nerve splits into two major segments at a point termed the pes anserinus. The upper segment is termed the temporozygomatic segment, and the lower segment is termed the cervicofacial segment. These branches further split into the five major branches that supply the muscles of facial expression; the temporal, zygomatic, buccal, marginal mandibular, cervical branches.

The facial nerve fiber itself can be further divided anatomically. The three major components of the nerve fiber are the endoneurium, perineurium, and epineurium. The endoneurium surrounds each nerve fiber, and provides the endoneurial tube. This tube is needed for nerve regeneration. As such, if the endoneurium is disrupted, the prognosis for return of function is worse. The perineurium surrounds a group of nerve fibers. It provides tensile strength, protects the nerve from infection, and provides pressure regulation. The last component is the epineurium. This is the layer that surrounds the entire nerve. It is responsible for providing nutrition to the nerve through the vasa nervorum.

The function of the facial nerve can be broken into three major areas, motor, sensory, and parasympathetic. The motor component supplies function to the muscles of facial expression as well as the stylohyoid, posterior belly of the digastric, stapedius and buccinator muscles. The sensory component can be divided into the special visceral afferent and general sensory afferent. The special visceral afferent is responsible for providing taste to the anterior 2/3 of the tongue. The general sensory afferent provides sensation to part of the tympanic membrane, the wall of the EAC, postauricular skin, and concha. The parasympathetic component provides secretory function to the submandibular, sublingual, and lacrimal glands as well as many of the seromucinous glands of the nasal and oral cavities.

Before we go into the details of facial nerve injury we must understand the basic classifications of nerve injury. A commonly used classification is the Sunderland Nerve Injury Classification. It is broken into five different classes. Class I injury is referred to as neuropraxia. It is a conduction block caused by the cessation of axoplasmic flow due to compression. This class of injury is typically what is felt when one's leg "falls asleep." A full recovery is expected with this injury. Class II is termed axonotmesis. In this injury axons are disrupted and Wallerian degeneration occurs distal to the site of the injury. The endoneurial tube remains intact, so regeneration occurs. However, regeneration is very slow occurring at 1mm/day. One also expects complete recovery of function following class II injury. A class III injury is termed neurotmesis. In this injury, the neural tube is disrupted, thus regeneration potential and functional return are affected. If regeneration does occur, a high incidence of synkinesis exists. Synkinesis is the abnormal mass movement of muscles which do not normally contract together.

A class IV injury is classified as disruption of the perineurium, endoneurium, and axon. The epineurium remains intact. Poor functional outcome is expected if regeneration does occur with a high risk of synkinesis. Class V injury is the worst of the injuries, and is classified as complete disruption of the nerve. There is little chance of regeneration with this type of injury. The risk of painful neuroma formation is increased due to axonal sprouts that make their way out of the nerve sheath.

Trauma to the facial nerve is the second most common cause of facial nerve paralysis representing 15% of all cases of facial nerve paralysis. The most common site of injury in trauma is the temporal bone. There are many different types of trauma that lead to facial nerve paralysis. The ones that we will cover are temporal bone fractures, penetrating trauma, and iatrogenic trauma.

Most temporal bone fractures are due to blunt trauma and can be seen in up to 5% of all trauma victims. They are the most common traumatic cause of facial nerve paralysis. There are two distinct types of temporal bone fractures, longitudinal and transverse. Longitudinal fractures are the most common type making up 70-80% of all temporal bone fractures. The type of fracture seen is one that is parallel to the long axis of the petrous pyramid and results from blunt force delivered to the temporoparietal area. Typically, one can expect to see facial nerve paralysis in 25% of cases of longitudinal fractures. Transverse fractures are less common representing roughly 10-20% of all temporal bone fractures. The type of fracture seen is one that is perpendicular to the long axis of the petrous pyramid, and results from a frontal or occipital blow. This type of fracture results in facial nerve paralysis in 50% of cases. One may also see a mix of the two fracture types. This occurs in 10% of all temporal bone fractures.

In one study by Chang and Cass (1999), they reviewed the facial nerve pathologic findings of 67 longitudinal temporal bone fractures and 11 transverse temporal bone fractures where the patient was known to have facial nerve paralysis. In longitudinal fractures, 76% of cases showed bony impingement or intraneural hematoma while 15% showed a transected nerve. 9% either had no pathologic findings or just neural edema. In transverse fractures, 92% of cases showed transection of the nerve while 8% showed bony impingement or hematoma.

The next type of trauma that can result in facial nerve paralysis is penetrating trauma. This type of trauma typically affects the extratemporal segments of the facial nerve. However, gunshot wounds will cause both intratemporal and extratemporal injuries. Gunshot wounds to the temporal bone result in facial nerve paralysis in 50% of cases. This type of injury usually results in a much worse outcome than other types of trauma secondary to the fact that gunshot wounds typically result in a mixture of avulsion and blunt trauma to different portions of the nerve at the same time.

The next type of trauma is iatrogenic trauma. This can be further broken down into injury during surgery and birth trauma. Due to its complex course, the facial nerve is commonly encountered in many head and neck surgical procedures. The most common overall surgery where facial nerve injury occurs is the parotidectomy. The most common otologic procedure resulting in facial nerve injury is the mastoidectomy. Tympanoplasty and exostoses removal both account for 14% of cases of injury each. The mechanism of injury is either direct mechanical injury or heat generated from drilling near the facial nerve. The most common nerve

segment injured during otologic surgery is the tympanic portion due to its high incidence of dehiscence in this area, and relation to surgical field. Nearly 80% of all cases of surgical related facial nerve injury go unrecognized. Birth trauma is another type of iatrogenic injury to the facial nerve. It is typically the result of a forceps delivery with compression of the facial nerve against the spine.

The work-up of facial nerve injury related to trauma begins with a good history and physical examination. Important aspects of the history include the mechanism (recent surgery, facial/head trauma), timing of injury (progressive loss of function or sudden loss), and associated symptoms (hearing loss or vertigo hint more toward a temporal bone injury). The physical examination must include a full head and neck examination looking for facial asymmetry and signs of facial injury (lacerations, hematomas, and ecchymosis). One must examine the head/scalp for signs of injury to help determine the vector of force if head trauma is involved. Otoscopic examination is another important aspect of the examination. Canal lacerations or step-offs as well as hemotympanum, tympanic membrane perforation, drainage of blood or clear fluid from middle ear may all be seen in temporal bone injury. Tuning fork tests (Weber/Rinne) with a 512 Hz fork can help determine if there is a conductive hearing loss. In addition, the muscles of facial expression should be closely examined. Dysfunction can be classified by the House-Brackmann Grading System. This system is divided into gross inspection and motion ability. During examination one must be aware that movement of the upper eyelid should not be considered a criteria for partial function since the levator palpebrae muscle helps in this function, but is innervated by CN III. The HB grading system can be found in the table below.

<b>Grade</b>	<b>Characteristics</b>
I. Normal	Normal facial function in all areas
II. Mild dysfunction	<p><b>Gross</b></p> <ul style="list-style-type: none"> <li>• Slight weakness noticeable on close inspection</li> <li>• May have slight synkinesis</li> </ul> <p><b>Motion</b></p> <ul style="list-style-type: none"> <li>• Forehead - Moderate-to-good function</li> <li>• Eye - Complete closure with minimal effort</li> <li>• Mouth - Slight asymmetry</li> </ul>
III. Moderate dysfunction  -First time you can notice a difference at rest	<p><b>Gross</b></p> <ul style="list-style-type: none"> <li>• Obvious but not disfiguring difference between the two sides</li> <li>• Noticeable but not severe synkinesis, contracture, or hemifacial spasm</li> <li>• At rest, normal symmetry and tone</li> </ul> <p><b>Motion</b></p> <ul style="list-style-type: none"> <li>• Forehead - Slight-to-moderate movement</li> <li>• Eye - Complete closure with maximum effort</li> <li>• Mouth - Slightly weak with maximum effort</li> </ul>
IV. Moderately severe dysfunction  -First time you have incomplete eye closure -No forehead movement	<p><b>Gross</b></p> <ul style="list-style-type: none"> <li>• Obvious weakness and/or disfiguring asymmetry</li> </ul> <p><b>Motion</b></p> <ul style="list-style-type: none"> <li>• Forehead – No motion</li> <li>• Eye - Incomplete closure</li> <li>• Mouth - Asymmetric with maximum effort</li> </ul>
V. Severe dysfunction	<p><b>Gross</b></p> <ul style="list-style-type: none"> <li>• Only barely perceptible motion</li> <li>• At rest, asymmetry</li> </ul> <p><b>Motion</b></p> <ul style="list-style-type: none"> <li>• Forehead - None</li> <li>• Eye - Incomplete closure</li> <li>• Mouth - Slight movement</li> </ul>
VI. Total paralysis	No movement

The work-up for traumatic facial nerve injury may also include radiographic evaluation. This typically involves CT and MRI scans. CT scans tend to be better for bony evaluation while MRI scans are utilized more for soft tissue detail and CPA pathology.

Another integral part of the evaluation of facial nerve injury is facial nerve testing. Testing has many functions. It is used to assess the degree of electrical dysfunction, helps with pinpointing the site of injury, and helps with determining treatment options. It can also be used to predict recovery of facial nerve function; partial paralysis is a much better prognosis than total paralysis. The testing can be divided into two categories, topographic and electrodiagnostic tests.

Topographic tests are used to assess the integrity of specific facial nerve branches by testing the function of each branch. These tests are not utilized much anymore because they are not anatomically accurate and do not predict potential recovery of function.

The first of these tests is the Schirmer's test. It is used to assess the function of the greater superficial petrosal nerve which is an evaluation of the protective mechanism of the eye. A piece of filter paper is placed in the conjunctival fornix of both eyes and the patient is asked to close his eyes for 5 minutes. After 5 minutes, the paper is removed from both eyes and the length of the areas that are moist is compared. An abnormal test can be defined as either a unilateral length measuring only 25% or less of the total length measured from both eyes or a total length from both eyes only measuring 25mm.

The next topographic test is the salivary flow test. It is used to test the integrity of the chorda tympani nerve by measuring the function of salivation with gustatory stimulation. The test begins with the cannulization of Wharton's ducts. A gustatory stimulation is then applied. Measurements of salivary flow then occur over a 5 minute period. An abnormal test is defined as a reduction of 25% of the measured saliva when compared to the uninvolved side. The test is not utilized anymore since it is considered difficult to perform, causes significant patient discomfort and carries poor accuracy.

The third topographic test is the electrogustometry test. This test consists of stimulating the tongue electrically in order to produce a metallic taste. Both sides of the tongue are compared for results.

The next topographic test is the stapedial reflex or acoustic reflex test. This test is based on the ability of the stapedius muscle to contract in response to a loud sound. When contraction of the muscle occurs, the impedance of the middle ear changes. A loud sound is applied to one side, and the impedance of the middle ear from both sides is measured. If the stapedius muscle is out, there will be no impedance change on the affected side.

Electrodiagnostic tests utilize electrical stimulation to assess facial nerve function. All but the electromyography (EMG) test require a normal contralateral facial nerve to compare functional results with. The nerve excitability test (NET) compares the current thresholds required to illicit minimal muscle contraction on the normal side of the face to those of the paralyzed side. A stimulating electrode is applied over the stylomastoid foramen, and a DC current is applied percutaneously. The face is then monitored for movement. The electrode is then repositioned to the opposite side, and the test is performed again. A difference of 3.5 mA or greater between the two sides is considered significant. The main drawback to this test is that it relies on a visual end point making it very subjective.

The next test is the maximum stimulation test (MST). This test is similar to the NET, except that it utilizes maximal stimulation rather than minimal, and the main trunk as well as each major portion of the distal branches of the nerve are stimulated. The paralyzed side is compared to the contralateral side and the functional comparison is rated as equal, slightly decreased, markedly decreased, or absent. An equal or slightly decreased response is considered favorable for complete recovery. Markedly decreased or absent responses denote advanced degeneration with a poor prognosis. The response to this test becomes abnormal sooner than the response to the NET and is therefore considered superior to the NET. However, like the NET, this test is also subjective.

Electroneurography (ENoG) is another electrodiagnostic test utilized to assess the facial nerve. It provides quantitative analysis of the extent of degeneration without being dependent on observer qualification, and is thought to be the most accurate of the electrodiagnostic tests. The facial nerve is stimulated with an impulse applied at the stylomastoid foramen using bipolar electrodes. The summation potential is then recorded by a device utilizing bipolar electrodes placed near the nasolabial groove. The peak to peak amplitude of the evoked compound action potential is considered proportional to the number of intact axons. The two sides are then compared with the response on the paralyzed side of the face expressed as a percentage of the response on the normal side of the face. It is believed that surgical decompression of the nerve should be performed when 90% degeneration has occurred. Those with less than 90% degeneration within 3 weeks of facial nerve injury typically have an expected spontaneous rate of recovery of 80 - 100%. The disadvantages of this test include patient discomfort, cost, and test-retest variability that is due to positioning of the electrodes and excitation of the muscles of mastication.

The next test we will examine is electromyography (EMG). This test determines the activity of the muscle itself. A needle electrode is inserted into the muscle, and recordings are made during rest and voluntary contraction. Normally, voluntary movement will produce biphasic or triphasic potentials. When a lower motor neuron injury occurs, the muscles supplied by this nerve will undergo spontaneous movements called fibrillations that can be measured anywhere from 10-21 days following the injury. This test is typically not the first test utilized due to the amount of time needed to see signs of injury. Six to twelve weeks prior to the clinical return of facial function, polyphasic reinnervation potentials can be measured. These potentials are considered the earliest evidence of nerve recovery.

The treatment options for facial nerve injury differ by mechanism of injury and initial presentation. For instance, if the nerve is transected during surgery, it is recommended that the surgeon explore 5-10mm of the involved segment and stimulate both the proximal and distal segments. If there is a response with 0.05mA, full recovery and good function are expected, and thus further exploration is not required. However, if the nerve only responds distally a poorer prognosis is expected. As such, further exposure is warranted. If the loss of function is noted following surgery, wait 2-3 hours and then re-evaluate the patient. This should be ample time for any anesthetic to wear off. If the paralysis is still present following that time, the surgeon's next move is based on the understanding of the integrity of the facial nerve. If the surgeon is unsure of the nerve's integrity or the nerve was never identified during surgery, re-exploration is warranted. If the integrity of nerve is known to be intact, treatment can begin with a high dose of steroids. This is typically prednisone at 1mg/kg/day for 10 days and then a taper. After 72

hours, EnoG is utilized to assess the degree of degeneration. If there is greater than 90% degeneration, one should re-explore. If there is less than 90% degeneration, one can just monitor the patient. If worsening paralysis occurs the surgeon should re-explore. However, if no regeneration occurs, but the function does not worsen, the timing of exploration or whether to explore is controversial.

Quaranta et al (2001) examined the results of 9 patients undergoing late nerve decompression (27-90 days post injury) who all had greater than 90% degeneration. Seven of the patients achieved HB grade 1-2 after 1 year, and the other 2 patients achieved HB grade 3. They concluded that patients may still have a benefit of decompression up to 3 months out. Shapira et al (2006) performed a retrospective review looking at 33 patients who underwent nerve decompression. They found no significant difference in overall results between those undergoing early (<30 days post-injury) vs. late (>30 days post-injury) decompression. Most studies like these have been very small and lack control groups. Some studies have shown improvements with decompression occurring 6-12 months post-injury, but further evidence is required.

If there is facial nerve paralysis following birth or extratemporal blunt trauma, it is recommended that there be no surgical exploration since greater than 90% of these cases are expected to regain normal to near normal function. When dealing with temporal bone fractures the degree of paralysis guides the treatment options. If complete paralysis following a temporal bone fracture occurs then complete transection of the nerve must be assumed. For this reason exploration is warranted. If there is a partial or delayed loss of function following the fracture the treatment begins with high dose steroids and ENoG testing after 72 hours. If there is greater than 90% degeneration, explore. If there is less than 90% degeneration, one can monitor and explore at a later date depending on worsening or failure to regenerate.

When dealing with penetrating trauma there is a high likelihood of nerve transection, thus exploration is usually warranted. If the injury occurs in the extratemporal segments exploration is typically not recommended when the injury occurs distal to the lateral canthus since the nerve endings are very small and there is a rich anastomotic network from other branches in this area. However, when exploration is going to occur it should take place within 3 days of injury because the distal branches can still be stimulated, thus making it easier to locate them. With gunshot wounds, however, delayed exploration is actually recommended as this type of trauma results in extensive nerve damage, and waiting a little longer to identify the extent of injury can be beneficial in forming a surgical plan.

If decompression of the nerve must occur, the patient's auditory and vestibular function must be taken into account. If the auditory and vestibular function is intact, a transmastoid/middle cranial fossa approach is warranted. If the auditory and vestibular function is absent, a transmastoid/translabyrinthine approach is recommended. Since the nerve may be injured along multiple segments all attempts should be made to localize the injured site pre-operatively. This will make a big difference in the amount of exposure required while potentially decreasing the morbidity of the procedure. However, this may not be possible, and full exposure of the nerve from the IAC to the stylomastoid foramen may be required. During the decompression, diamond burs and copious amounts of irrigation should be utilized to prevent thermal injury. The thin layer of bone overlying the nerve is typically bluntly removed.

Whether to perform neurolysis or not to open the nerve sheath is debatable. However, if a hematoma is identified it should be drained.

If repair of the facial nerve is required, there are many options available to the surgeon. With neural repair, the surgeon should expect to start seeing some recovery starting around 4-6 months. The recovery can last up to 2 years following repair. The surgical options available differ based on the timing of injury since after 12-18 months, muscle reinnervation becomes less efficient even with good neural anastomosis.

When it comes to nerve repair, the goal is a tension free, healthy anastomosis. The rule is to repair earlier than later, but the exact timing of the repair is controversial. Some authors have reported improvement with repairs as far out as 18-36 months. May and Bienstock recommend repair within 30 days, but others have found superior results if done up to 12 months out. After 2 weeks of injury, collagen and scar tissue replace axons and myelin. For this reason unhealthy nerve endings must be excised prior to anastomosis.

Primary anastomosis should be attempted first since it provides the best overall results of any surgical intervention. It can be performed when the defect is less than 2cm since mobilization of the nerve can give nearly 2cm of length. However, if more than this is mobilized, the risk to further neural injury increases secondary to devascularization. The most important aspect of neural repair is ensuring that the endoneurial segments are aligned as this will promote regeneration. The nerve ends should be sutured together using three to four 9-0 or 10-0 monofilament sutures to bring the epineurium or perineurium together.

If the defect is greater than 2 cm or a tension free anastomosis cannot be obtained, then nerve grafting or transfer should be performed. The problem with this is that this results in partial or complete loss of the donor nerve function. The type of grafting/transfer performed depends on whether both the proximal and distal segments of the nerve are available. If they are available, then a simple graft can be used to bring them back together. A commonly used nerve for grafting is the great auricular nerve since it is usually in the surgical field already. It is located within an incision made from the mastoid tip to the angle of the mandible. However, only 7-10cm of this nerve can be harvested. The complication from this harvest is a loss of sensation to lower auricle. Another commonly used nerve for grafting is the sural nerve. It is located 1 cm posterior to the lateral malleolus, and can provide 35cm of length making it extremely useful in cross facial anastomosis. The complication that occurs with its use is the loss of sensation to lateral calf and foot. One study quoted that 92-95% of patients undergoing graft repair when proximal and distal portions of the nerve are available have some return of facial function. Of those, 72-75% have good results (HB 3 or above).

If the distal nerve segment is the only segment available, the surgeon must ensure that the facial musculature is suitable for reinnervation. This is done through EMG testing and/or muscle biopsy. The options for repair in this situation typically involve the use of the hypoglossal nerve or the contralateral facial nerve. The direct hypoglossal-to-facial graft is performed by attaching the distal segment of the injured facial nerve directly to the hypoglossal nerve. With this type of graft, 42-65% of patients are expected to experience decent symmetry and tone. However, the complications are difficult to deal with and include atrophy of the ipsilateral tongue and difficulties with chewing, speaking, and swallowing. A more tolerated grafting technique is the

partial hypoglossal-to-facial jump graft. This is done by the use of a nerve cable graft (usually the sural nerve) to connect the distal end of the facial nerve to a notch in the hypoglossal nerve. It results in much fewer complications, but increases the recovery time. May compared results of the direct VII-XII graft to the VII-XII jump graft. In his study, only 8% of patients experienced permanent complications from loss of the hypoglossal nerve in the jump graft compared to 100% in the direct graft. Of those patient undergoing jump grafting, 41% obtained good movement with less synkinesis. The motor function, however, was not as strong in the jump graft group.

Another option is the facial-to-facial graft. This procedure can either involve a single contralateral branch connected to the distal nerve or multiple anastomoses from segmental branches to segmental branches. The best results from this type of procedure have been seen when a sural nerve graft is utilized to connect the buccal branch on the contralateral side to the distal nerve stump. The VII-VII graft technique is typically not recommended secondary to the weakness caused to the contralateral facial nerve and lack of power to control the musculature resulting in poor results.

The next topic to be covered is one that many head and neck surgeons have found very useful when dealing with the facial nerve; facial nerve monitoring. The first monitors that were utilized relied on sensing muscle movement. They are rarely used now since a large threshold must be reached to illicit movement, and by that time injury may have already occurred. Also, there is a poorer response to facial nerve stimulation than what is seen in electrophysiologic techniques. The principle technique utilized now days involves electromyography. Electrodes are used to detect differences in electrical potential associated with a depolarizing current. A graphic and acoustic signal are then recorded.

There are two types of responses that one can expect. The first are repetitive responses. They occur following the cessation of surgical manipulation, and represent irritability of the nerve secondary to nerve injury. They are used to warn the surgeon of injury or impending injury. The next type of responses are the nonrepetitive responses. They are single responses secondary to direct mechanical or electrical stimulation, and are utilized to map the course of the nerve.

There are many uses for facial never monitors. These include identity, mapping, injury identification, and prognosis. The monitors can be utilized to help identify exactly where the nerve is located. This can be done through mechanical or electrical stimulation. Once located, the nerve can then be mapped by repeated stimulation. Most surgeons advocate bipolar stimulation as it is more precise, but does carry more false-negatives than monopolar techniques. Injury identification relies mainly on repetitive responses as described above. This allows the surgeon to alter his or her actions to prevent or lessen injury. The monitors can also provide prognostic information of facial nerve function following surgery. There are two different measurements that can be taken to determine prognosis. The first is a technique that utilizes stimulated compound action potentials. It is the least utilized as it relies on proper electrode placement, thus leading to poor reproducibility. During this technique, a 0.4mA stimulus is applied to the nerve and a compound action potential is recorded. If the compound action potential is greater than 500-800 microvolts, a HB I-II is expected. However, as the action potential drops below 500 microvolts, the outcome becomes poorer. The most commonly used

technique for determining facial nerve prognosis with monitors is termed the nerve stimulus threshold. This technique utilizes an electrical stimulus applied to the proximal end of the nerve. If the nerve responds with a stimulus that is less than 0.3mA, a HB I-II is expected. If a greater than 0.3mA stimulus is required to stimulate the nerve, one can expect a HB III-V.

Many have wondered whether the use of a facial nerve monitoring during surgery really makes a difference to the overall outcome following surgery. Dickinson and Graham reviewed the use of facial nerve monitors in surgical cases involving the excision of CPA tumors. Thirty-eight cases were performed without facial nerve monitoring, 29 cases with a pressure or strain gauge sensor, and 41 cases with monitoring by EMG. They reported poor outcomes (HB V-VI) in 37% of cases where no monitor was used and 21% of cases where the older pressure or strain gauge sensor was utilized. However, poor outcome was only noted in 4% of cases where EMG was utilized. A confounder with this study was that there was a higher incidence of larger tumors in the unmonitored group.

Pensak et al examined 250 cases involving surgery on chronic middle ear disease in which all cases were monitored. They reported that in 100% of cases the facial nerve was grossly identified. However, only 82% were confirmed with monitor stimulation. In cases where the nerve was exposed, they reported that the monitor alerted the surgeon to its location in 93% of cases. Silverstein and Rosenberg examined 500 cases in which facial nerve monitoring was utilized. They reported no cases of facial nerve injury, but did report that the monitor prevented injury to the facial nerve in 20 cases.

Terrell et al examined 117 cases of parotid surgery where 56 cases had monitors and 61 cases were performed without a monitor. They reported a statistically significant decrease in the rate of post-operative paresis in the monitored group, but found no difference in long term outcome. They did find that longer OR times were associated with decreased rates of post-operative paresis. Witt reviewed 53 cases of parotid surgery in which 33 had monitors and 20 did not. He found no difference in paresis rates as well as no difference in long term outcome.

Another question that is commonly asked when it comes to facial nerve monitoring is the safety of its use. Most ask the question "does repetitive stimulation lead to facial nerve injury?" Babin et al examined the use of pulsed current stimulation to stimulate cat facial nerves. A pulse of 1mA was applied to the nerve every 3 seconds for 1 hour. A transient decrease in nerve sensitivity following cessation of the stimulus was noted, but no permanent injury was reported. Hughes et al examined the use of pulsed and constant current models for stimulation of mouse sciatic nerves. In all cases in which pulsed current was utilized, no injury was reported. In some cases in which constant current was utilized, mild injury and axonal degeneration occurred. However, nearly all monitors now utilize pulsed currents. For this reason, the surgeon must be cognizant of the type of current being applied for stimulation.

In conclusion, the facial nerve is very complex in its function and anatomical course. As such, it can be easily injured by many mechanisms. A good understanding of its function and anatomical course can make it easier for the surgeon to prevent injury and identify where an injury has occurred. Combining this knowledge with the knowledge gained from diagnostic testing, a surgeon can come up with a good treatment plan.

## **Discussant's remarks 6/29/09**

### **Dr. Makishima:**

Thank you David, that was excellent. I did learn a lot from this. One of the things I wondered about was temporal bone fracture. Whether you do surgery or not, that's very controversial when it's a complete loss, and on film you know that it's likely transected.

I think there was a retrospective study on a large number of patients. It was probably about in the hundreds. They looked at whether or not it was effective to do decompression; the outcome was about the same. So even when you know that it's transected, and you decide to do something about it, that doesn't necessarily mean that it will help the patient's recovery. If you have the technique, and are confident that you can carry out the surgery, it seems reasonable to do it if the patient is willing. But remember that it is an invasive procedure.

If the outcome is the same, you have to think about doing the surgery after a long amount of time, eighteen months or one year or six months in Bell's Palsy. I've seen people come in as a last resort because we were in a big institute when I was in Japan. We did do decompression on patients six months, twelve months out. In 50% of the cases, we did have some improvement, so I would encourage you to decompress. But I don't know if it would be the same for people who have had trauma. I don't have anything to compare it with.

### **Dr. Quinn:**

What was the anatomical extent of your dissection in Bell's Palsy? Did you do middle fossa exposure and expose the facial nerve proximal to the geniculate ganglion?

### **Dr. Makishima:**

We only did mastoid segment decompressions, and it was just as effective. The whole idea is to decompress the nerve, and I believe that with Bell's Palsy it's kind of like a compartment syndrome. With an injury to the nerve, whatever release that frees up the segment will likely restore the function of facial nerve.

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