NUMB TONGUE, NUMB LIP, NUMB CHIN: WHAT TO DO WHEN?

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INTRODUCTION

Microneurosurgery of the trigeminal nerve has been in the spotlight over the last few years. The introduction of cone-beam scanning, three-dimensional imaging, magnetic resonance neurography, endoscopic-assisted surgery, and use of allogenic nerve grafts have improved the techniques that can be used for assessment and treatment of patients with nerve injuries. Injury to the terminal branches of the trigeminal nerve is a well-known risk associated with a wide range of dental and surgical procedures. These injuries often heal spontaneously without medical or surgical intervention. However, they sometimes can cause a variety of symptoms, including lost or altered sensation, pain, or a combination of these, and may have an impact on speech, mastication, and social interaction. These injuries also can cause significant morbidity when accompanied by neuropathic pain. Appropriate assessment and management of these injuries requires adequate training, knowledge, clinical skills, and experience. This chapter focuses mainly on injuries to the inferior alveolar nerve (IAN) and lingual nerve (LN) and their management.

MECHANISM OF NERVE INJURIES

When attempting to classify the various mechanisms of nerve injury in the maxillofacial region, it becomes clear that the overwhelming majority are iatrogenic in nature. The nerves that are most often affected in dento-alveolar procedures are the branches of the mandibular division of cranial nerve V, i.e., the trigeminal nerve. The lingual nerve and inferior alveolar nerve are most often affected, and third molar surgery is the most common cause of injury. [1]

None of these nerves provide motor innervation. However, damage to these nerves can cause a significant loss of sensation and/or taste in affected patients. When considering the full scope of maxillofacial surgery, branches of all three divisions of cranial nerve V are at risk. These can include the peripheral branches of the supraorbital and supratrochlear nerves as well as the infraorbital nerve. In addition, the greater auricular and auriculotemporal nerves are fairly superficial and can be injured in surgeries involving the neck. The nerve of greatest concern because of its motor function is cranial nerve VII, i.e., the facial nerve. While this nerve is not at risk in routine

transoral and maxillofacial procedures, extraoral approaches to the craniofacial skeleton can put this nerve and its branches at risk.

It is important that a discussion of the risk of nerve injury is included when taking patient consent for any proposed surgical procedure. However, not all nerve injuries are unplanned, as many pathologic resections of tumors in the maxillofacial region necessitate resection of a portion of the cranial nerve architecture. In addition to iatrogenic nerve damage, nerve injuries in this region also can result from traumatic events, such as facial fractures, gunshot wounds, and lacerations.

To classify the multiple types of nerve injuries that can occur, we have devised four categories: **chemical**, **blunt**, **irregular**, and **sharp**. Understanding the type of injury that has occurred helps to understand the biology of the injury that is present and further differentiate the relative urgency needed in offering surgical treatment. While there is overlap in many of the injury patterns that occur, we can classify the procedures by the nerves that can be injured, the type of injuries that can occur, and the mechanism of each injury.

When considering the four categories that we have introduced, it is critical to understand that each category results in a different pattern of nerve damage at the structural level. First, **chemical** injuries are unique in that they can be quite diffuse and result in direct toxic effect to the affected nerve. These often represent true emergencies and should be treated as such when identified. **Blunt** injuries are often the result of over-zealous retraction or compression between mobilized bone segments. This type of injury often leads to a neuropraxic state without structural disruption of the nerve fibers themselves. However, the ischemia that can occur results in death of cell bodies and subsequent Wallerian degeneration. Next, irregular injuries are those that result in partial or total disruption of the nerve fibers in a ragged and irregular fashion, leaving the nerve ends looking much like the fraying ends of a rope. These often result from nerve contact with high speed surgical burs, saws, files, or drills. Lastly, sharp injuries are those that result in a clean transection of the nerve fibers in such a way that, other than continuity, the morphology of the nerve has not been altered. Blunt injuries typically recover on their own with time. However, irregular and sharp injuries typically require surgical intervention. **Table 1** shows the procedures, nerves affected, types of injury, and mechanisms involved.

Procedure	Nerves affected	Mechanism
Injection of local anesthesia	LN, IAN	Toxic effect of anesthesia (C) Bleeding/hematoma (B) Direct needle trauma (I)
Endodontic	IAN, MN	Chemical application (C) Compression (B) Over-instrumentation (I)
Wisdom tooth removal	IAN, MN, LBN	Topical medications (C) Lingual flap retraction (B) Root/bone compression (B) Bur (I) Sutures (I, B) Incision (S)
Orthognathic surgery	IFN, IAN, LN, LBN	Nerve retraction (B) Segment fixation/compression (B) Drill, saw, screw (I) Incision (S)
Trauma	SON, ION, IAN, MN	Compression (B) Severed/Avulsed (I) Plate/Screw Placement (I,B) Retraction (B)
Dental Implant Placement	IAN, LN, MN	Canal Compression (B) Compartment Syndrome (B) Over-drilled (I) Overly long fixture (B,I) Suture (B, I) Incision (S)

Procedure	Nerves affected	Mechanism
Floor of mouth surgery	LN	Blunt dissection (B) Suture (B, I) Incision (S)
Trans-oral ablation	IAN, MN, LN	Planned nerve resection (I,S) Unplanned injury (B,I,S)
Extraoral approaches Ablative head and neck Cosmetic facial procedures	FN, ATN, GAN, SON	Retraction (B) Dissection (B) Incision (S)

Table 1: Procedures, nerves affected, and mechanisms for surgical intervention. (IAN, inferior alveolar nerve; LN, lingual nerve; MN, mental nerve; LBN, long buccal nerve; SON, supraorbital nerve; ION, infraorbital nerve; FN, facial nerve; ATN, auriculotemporal nerve; GAN, greater auricular nerve; C, chemical; B, blunt; I, irregular; S, sharp.)

As with any other surgery, appropriate patient selection is critical. An adequate and informed consent discussion including all other treatment options, risks, and complications should be held. Lastly, nerve repair surgery is incredibly technique-sensitive and implementation of correct surgical technique is critical to obtaining success.

Third molar odontectomy as mentioned above is the cause of the majority of injuries to the inferior alveolar and lingual nerves. These can occur at any stage during the extraction process, beginning with injection of local anesthetic and ending with closure of the incision with sutures. The incidence of IAN injury as a consequence of third molar surgery is 0.41%–7.5% and following sagittal split ramus osteotomy is 0.025%–84.6%, whereas the incidence of lingual nerve injury is 0.06% –11.5% following third molar removal. However, it is important to differentiate temporary from permanent paresthesia rates. After third molar surgery, temporary paresthesias affecting the IAN and lingual nerve range from 2%–6%, whereas permanent injuries are in the range of 0.5%–2%. Orthognathic surgery is another common procedure that may result in trigeminal nerve injury. The IAN is affected more often than the lingual nerve. For sagittal split ramus osteotomies, almost all cases will have some temporary IAN paresthesia, whereas the permanent rates are in the range of 1%–5%. The reported incidence ranges from less than 5% to in excess of 90% because of poorly controlled factors inherent in the study designs, including the experience of the surgeon, technical variability in the surgery, and lack of standardization of neurosensory testing. There are multiple risk factors that predict nerve injury, [2] including patient age, [3] increased procedural time, concomitant third molar removal or genioplasty procedures, nerve

entrapment in the proximal segment, manipulation of the IAN at the site of the osteotomy, [4] position of the inferior alveolar canal close to the inferior border of the mandible, a low mandibular body corpus height, and retrognathism in class II deformities. [5]

Fortunately, with proper informed consent, most patients tolerate the paresthesia well after orthognathic surgery.

In the past few years, a significant increase in implant-related injuries to the IAN have been observed, most likely secondary to the increase in the number of implants being placed and the more frequent reporting of the injuries. These injuries are not well documented in the literature and are difficult to manage appropriately. Further, there is no universally accepted algorithm to follow for management. There are multiple suggested mechanisms by which the implant surgery can cause nerve injury, including direct injury by the drill and indirect injury by damage to the neuromuscular bundle leading to edema and possible hematoma formation with a compartment-like syndrome. These injuries not uncommonly result in long-term dysesthesias.

Appropriate preoperative planning for implant cases is of paramount importance to prevent those injuries. Patients with a vertical height deficiency may benefit from bone grafting procedures, utilization of short implants, or repositioning of the IAN. Although this procedure protects the nerve from direct and indirect injuries, it has been associated with an increased incidence of long-term paresthesia, ranging from 0% to 77%, with a mean of approximately 30%–40%. [6]

Early recognition of implant-associated injury postoperatively is important. Nerve injury evident by postoperative paresthesia and confirmed radiographically warrants consideration of surgical removal of the implant and possibly replacing it with a shorter implant. When the patient presents after an observed period with no improvement, the option of exploration of the nerve and possible repair comes into play while retaining the implant. Patients with persistent paresthesia should be referred to a microneurosurgeon for evaluation and management in a timely fashion.

With the increased utilization of implants, there has been a noticeable drop in the number of preprosthetic surgeries performed. In cases where placement of an implant is not feasible, procedures such as mandibular vestibuloplasty prior to fabrication of a denture may still be performed and place the mental nerve at risk for injury. In such cases, repair is possible but carries the risk of fibrosis and scarring in the soft tissues adjacent to the repaired nerve and thus decreased quality of neurosensory recovery.

Other dento-alveolar procedures that carry a risk for possible nerve injury include autogenous bone graft harvesting from the mandible (both ramus and genial), bone grafting with or without placement of mesh or screws, periapical mandibular surgery depending on the position and proximity to the canal, and alveolar distraction.

Facial trauma involving the orbital and zygomaticomaxillary complex, mandibular fractures, and soft tissue injuries may cause injury to the branches of the trigeminal nerve. Appropriate evaluation and recognition of these injuries along with adequate reduction and stabilization of the fracture segments help to decompress and realign the segments with nerve injury. Accomplishing these two tasks will help to guide spontaneous neurosensory recovery. When it comes to soft tissue traumatic injuries with nerve involvement, appropriate identification of the injured nerve branches and repair should be accomplished.

Treatment of pathologic head and neck lesions with cranial nerve involvement may

result in permanent nerve injury. Perineural invasion leading to a nerve deficit is not uncommon with malignant tumors, and resection of the involved nerve is mandatory in these cases. Similarly, nerve damage whether from multiple debridements of fractured mandible in severe osteoradionecrosis cases will require resection of the involved nerve. While there is no consensus on immediate reconstruction, specifically when postoperative radiation is indicated or patients with history of radiation involving the nerve, several case reports and small series have shown promising results. [7]

There are multiple options for management of the associated nerve when resecting a benign tumor, including resection without reconstruction, resection with delayed reconstruction, which carries the risk of extensive scaring around the nerve ends and making reconstruction more challenging, and resection with immediate reconstruction of the nerve. Other techniques that have been proposed include the nerve pull technique and nerve preservation technique (the latter being controversial according to the type of tumor). In addition to defects caused by a continuity resection involving the cranial nerves, topical use of some medications in close proximity to the IAN or lingual nerve, such as Carnoy's solution, tetracycline, and Surgicel, has been associated with nerve injury.

Injection injuries to the IAN and LN continue to pose a dilemma, with as yet no consensus on the cause, diagnosis, or management. The incidence is estimated to be 1 in 100,000–500,000 blocks. A study of 9,587 mandibular blocks by Harn and Durham [8] found a 3.62% incidence of temporary paresthesia and a 1.8% incidence of long-term paresthesia lasting longer than 1 year. Multiple theories have been proposed to explain the mechanism by which these injuries occur. The most likely mechanisms are as follows: direct neural injury from insertion of the needle resulting in separation of the fascicles without direct neural disruption, [9] formation of a barb on the needle from bony contact with direct neural disruption on removal, post injection edema that can result in transient paresthesia and resolves spontaneously, formation of an epineurial hematoma due to the disruption of vessels in the epineurium and perineurium, and local anesthetic toxicity. When considering pressure related injuries, bleeding or edema in a confined area may lead to ischemic pressure on select groups of fascicles contained within the nerve resulting in localized paresthesia, not involving the entire distribution of the IAN. In most of these cases there will be a resolution of the clinical symptoms and that is owning to the lymphatic drainage of the localized hematoma over the few days to weeks after surgery. The possibility of local anesthetic toxicity is proposed to cause prolonged paresthesia after nerve block, especially if the solution is deposited within the confines of the epineurium. Prilocaine and articaine may be associated with an increased risk of long-term paresthesia when used in mandibular blocks compared with other local anesthetic solutions due to the concentration gradient effect. [10-11]

As already mentioned, the final step during injection is when the needle is withdrawn, which is the point at which the theory of the needle-barb mechanism of injury is proposed. [12] The barb that forms at the needle tip as a result of advancing the needle to the medial ramus in a mandibular block injection can pass in the vicinity of the LN or IAN during withdrawal, leading to fascicular injury. [13]

While much is still unclear regarding injection-related injuries, the following points have been deduced from the collective experience thus far [13]:

- Injection injuries are difficult to predict and prevent.

- Persistent symptoms are more common in female patients, and the LN is more commonly affected.
- The classic electric-shock sensation upon injection is not commonly reported by patients who suffer these injuries.
- Dysesthesia is more likely to result from injection injury than other types of nerve injury.
- Progressive demyelination along the injured nerve might lead to involvement of other divisions of the trigeminal nerve.
- Most of the cases resolve within 2 months, but when paresthesia persists for longer than 2 months only one third of the injuries resolve spontaneously.

Microneurosurgical intervention in these injuries has not been considered as an option for treatment because of limited surgical access, an unclear site of injury, and the presence of chronic dysesthesia. Medical management is preferable in the majority of these cases.

ANATOMY

The largest of the cranial nerves is cranial nerve V (the trigeminal nerve). This nerve provides sensation to most regions of the maxillofacial complex, so it is an important nerve to consider in day-to-day practice. The posterior division of the mandibular nerve has a general somatic afferent component that provides sensory information via the LN and IAN to the ipsilateral anterior two-thirds of the tongue, mucoperiosteum, and teeth of the mandible. The nerve also carries special visceral efferent motor fibers to the anterior digastric and mylohyoid muscles. The LN carries general somatic afferent sensory feedback from the anterior two-thirds of the tongue, floor of the mouth, and lingual gingiva. The nerve descends from the posterior division medial to the lateral pterygoid muscle in the infratemporal fossa where it joins with the chorda tympani. This is a branch of the facial nerve that carries taste sensation for the anterior two thirds of the tongue and parasympathetic fibers to the salivary gland. The LN then enters the oral cavity between the attachments of the superior pharyngeal constrictor and mylohyoid muscles to the mandible. The nerve continues in the floor of the mouth lateral to the hyoglossus muscle, crossing the submandibular duct, where it enters the tongue. Miloro *et. al.* analyzed the location of the LN in the third molar area and found it to be approximately 10% above the alveolar crest, 25% in contact with the lingual plate, and on average approximately 2.5 mm below and medial to the cortical plate [14] The LN averages 3.2 mm in diameter. In many cases, it is monofascicular at the level of the lingula and averages 20 fascicles at the level of the third molar [15]

The IAN carries general somatic afferent information for the teeth, anterior buccal gingiva, lip, and chin. The initial course has a close relationship to the LN. After separating about 5 mm caudal to the cranial base, it descends from the posterior division between the lateral and medial pterygoid muscles. The nerve passes around the lower border of the lateral pterygoid muscle and proceeds to the medial aspect of the ramus of the mandible to enter the mandible at the lingula. The cephalocaudal course of the nerve in the mandible is such that it descends to the lowest point near the first molar and then rises once again, whereas in a buccolingual position, the nerve is closest to the lateral cortical plate in the third molar area, but remains constant in its relationship to the medial cortical plate. [16] The IAN has been shown to take a variable route through the mandible and there are reports of bifid canals. The nerve can have an anterior loop of up to 5 mm

where it exits the mandible. It sends off an incisor branch to the first premolar and to the ipsilateral central incisor and gingiva. The nerve terminates in the mental branch, which supplies sensation to the lip and chin. The average diameter of the nerve is 2.4 mm; it averages 7 fascicles at the level of the lingula and 18 fascicles overall. [17]

CLASSIFICATION OF NERVE INJURIES

Classification of nerve injuries is useful in understanding their pathologic basis, making decisions on management strategies, and predicting the prognosis for recovery. Most systems aim to correlate the degree of nerve injury with symptoms and pathology at the microscopic level. The two most common classifications are the Seddon and Sunderland. The injury is graded using these classifications based on the degree of axonal injury at the histologic light microscope level according to the likelihood that an injured nerve will recover spontaneously after injury.

Learning to identify subjective patient descriptors and group them appropriately can aide the clinician in appropriate diagnosis. Common terms that the clinician may hear in a patient interview fall into three groups: "numb and swollen", "tingling and tickling", and "pricking and burning"; these descriptions represent hypoesthesia, paresthesia, and dysesthesia, respectively. Before embarking on a discussion of the classification systems that are currently used in nerve injuries, the reader should be familiar with the list of terms used when discussing nerve injury patterns and outlined in **Table 2**.

Allodynia	Pain attributable to a stimulus that does not normally provoke pain.
Analgesia	Absence of pain in the presence of stimulation that would normally be painful.
Anesthesia	Absence of any sensation in the presence of stimulation that would normally be painful or non-painful.
Anesthesia dolorosa	Pain in an area or region that is anesthetized.
Dysesthesia	An abnormal sensation, either spontaneous or evoked, that is unpleasant. All dysesthesias are a type of paresthesia but not all paresthesias are dysesthesias.

Hyperalgesia	An increased response to a stimulus that is normally painful.
Hyperesthesia	An increased sensitivity to stimulation, excluding the special senses (seeing, hearing, taste, and smell)
Hyperpathia	A painful syndrome characterized by an increased reaction to a stimulus, especially a repetitive stimulus. The threshold is also increased.
Hypoesthesia	Decreased sensitivity to stimulation, excluding the special senses
Paresthesia	An abnormal sensation, either spontaneous or evoked, that is not unpleasant.
Protopathia	The inability to distinguish between two different modes of sensation, such as a painful and non-painful pinprick.
Synesthesia	A sensation felt in one part of the body when another part is stimulated.

Table 2: Glossary of Terminology.

When considering the classification of nerve injuries, it is of the utmost importance to realize that the classifications we use are all based on histologic data. The first classification schema came from Sir Herbert Seddon in 1942, who based his three categories of nerve injury severity on histology in combination with motor function deficit [18, 21] (**Figure 1**) in 1951, Sir Sydney Sunderland further delineated the classification schema developed by Seddon into five categories by focusing on the severity of the damage deep to the epineurium. [19, 21] Finally, for completeness, we must mention the more recent addition by MacKinnon, who added a sixth category to this schema in 1988 to represent injuries where a mixture of the previous classifications exists simultaneously. [20] The classification categories will be discussed further here in greater detail; however; it should be noted that these categories were created to represent the histologic condition of the nerve injury site and based on that understanding can be used to guide care and predict recovery (see Figure 1).

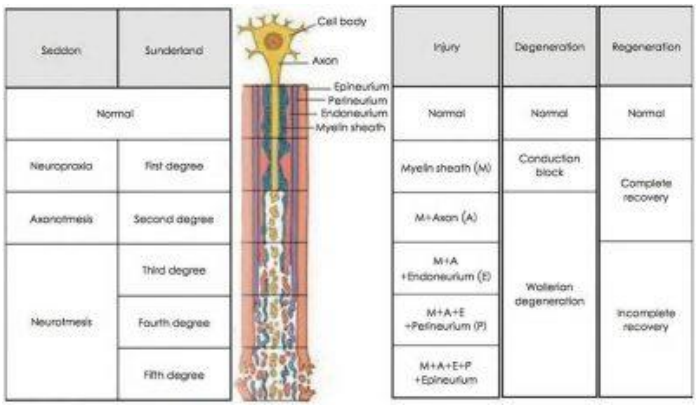


Figure 1: Overview of Seddon and Sunderland Classifications.

In breaking down the classically described classifications of nerve injury, the first category is neuropraxia. Neuropraxia is equivalent to a Sunderland first-degree nerve injury. Neuropraxia is a state in which there is no physical disruption of nerve fiber continuity, but despite this, the patient experiences loss of nerve function that may be motor or sensory. Symptoms may include paralysis, tingling, or numbness. These symptoms are temporary. This is perhaps the most commonly experienced nerve injury in oral and maxillofacial surgery. The authors frequently explain this to patients when being equivocal regarding a bruise on the nerve, and mention that, just like a bruise on the body, it will resolve with time. Another analogy that has proven helpful in describing this to patients is the tendency for the power to go out in heavy rain, and that most certainly, in the next hours to days, the power may flicker a bit (equivalent to the shocks and zings felt by the recovering patient) but will eventually come back on.

The next level of nerve damage is referred to as axonotmesis. Axonotmesis occurs when the nerve axon has been disrupted but the associated connective tissues (endoneurium, perineurium, and epineurium) are intact. This is represented by Sunderland as a second-degree injury. In this type of injury, Wallerian degeneration does occur at the injury site. Spontaneous recovery is expected in such injuries.

The next level of severity is neurotmesis. As originally described by Seddon, this stage represents a complete severance of the nerve. At this stage, spontaneous recovery becomes variable at best and in most cases unlikely without surgical intervention. However, this is where Seddon and Sunderland's classification systems diverge from one another. Degrees 3–5 in Sunderland's system all fall under Seddon's original neurotmesis heading. This subdivision more accurately describes the internal damage to the nerve and can better predict the likelihood of functional recovery without surgical intervention. A third-degree injury under Sunderland's system represents a disruption of the axon and endoneurium. This degree of nerve injury still can theoretically undergo spontaneous recovery but may require surgical intervention. Proceeding to a fourth-degree Sunderland injury, disruption of the axon, endoneurium and perineurium has occurred. In this degree of injury, spontaneous recovery is considered unlikely and surgical intervention is recommended. Lastly, a fifth-degree Sunderland degree represents a complete severance of the nerve at all levels, i.e., axon, endoneurium, perineurium, and epineurium. Surgical intervention is indicated for such an injury.

As previously discussed, the ability to classify nerve injuries is a crucial step in successfully managing patients that have sustained neurologic damage. Understanding not only the naming system but also the biology behind the degree of injury that has occurred allows the practitioner to better explain to the patient what they are experiencing and the possible indications for corrective surgery. In addition to nerve injury classification schemes, one must also learn to identify the common terms used by patients that correlate with the diagnosis being

provided. Ultimately, identification followed by appropriate management and timely treatment and/or referral are of the utmost importance in successful management of nerve injuries.

EVALUATION

Evaluation of the patient with a nerve injury is considered a key step in surgical management, and should be completed in a systematic fashion. Subjective information (from both the patient and the referring surgeon) is of paramount importance and should be reported appropriately. In addition, associated daily disturbances, such as biting of the lip or tongue and pain while shaving and others should be documented. Following documentation of this information, objective testing should be performed using the standardized clinical neurosensory testing. Advanced tests Gustatory assessment, may also be indicated in cases with lingual nerve injuries.

The McGill pain questionnaire and the pain analogue scale are useful tools for measuring the patient's subjective sensations as they wax and wane. After the patient rates their dysfunction, a quantitative measurement can be made regarding the altered sensations and how they affect the patient's life. If the nerve injury was witnessed and primarily repaired, it is important to begin testing immediately postoperatively in order to track the progression of the injury and repair. [22]

The distinction between patients who are experiencing pain associated with neurosensory dysfunction and those who are not must be made because of the potentially different treatment pathways for these patients. Hypoesthesia can be coupled with dysesthesia in response to noxious and non-noxious stimuli. This response should be measured, compared with that in the uninjured contralateral nerve, and utilized as a part of the decision-making process regarding whether or not to pursue surgical repair of the injury. Patients experiencing early pain are candidates for early surgical intervention than those who are not. The armamentarium for neurosensory testing should be consistently used for each patient. This testing should include light touch, sharp/dull detection to determine the level of hyper/hypoesthesia, and two-point discrimination. Cotton swabs may be used to determine light touch. Sharp/dull detection can be accomplished in several ways, First, a cotton swab may be used if the wooden sticks are broken into a sharp point. Alternatively, a sharp dental needle can be applied with gentle pressure. However, the best way to objectively measure hypo/hyperesthesia is by testing with Semmes-Weinstein monofilaments. Each monofilament bends when a known amount of force is applied, allowing for a quantitative measurement of the loss of sensation.

Two-point discrimination is measured using a graduated disc with points that are spaced at known distances. Performing these tests on both the affected and unaffected nerve distributions is extremely useful. We find it convenient to document both the patient's subjective fields of neurosensory impairment as well as objective measurements utilizing a skin marker and photographic documentation. This serves as a baseline for dysfunction and assists in monitoring the progression of recovery after surgical or non-surgical interventions. In cases of hyperesthesia, regional nerve blocks can be used proximal to the site of nerve injury to differentiate local versus central pain mechanisms.

Neurosensory testing for trigeminal nerve injuries can be divided into three types or levels that are based on the types of fibers being tested. Testing should begin at "level A" where the A-alpha and beta nerve fiber function is assessed by measuring two-point discrimination, fine touch and

directional discrimination. The rolled end of a cotton-tipped applicator works well for fine touch and directional perception. These higher-order fibers, which function in fine touch, are the last fibers to recover because of their complex anatomy and function. A mildly altered test at level A suggests a mild nerve injury with slowed stimulus responses secondary to conduction blockade. This is likely an injury without discontinuity in which full sensory recovery can be expected in less than 2 months in a patient who does not undergo surgery. Alternatively, this may represent progression from a more severe nerve injury. The two-point discrimination of the LN and IAN should be approximately 3 mm and 4 mm, respectively. [5] If dysesthesia secondary to nerve injury is part of the patient's complaint, this may not represent a contraindication to peripheral nerve repair, but further testing should be performed to rule out centrally mediated pain. **(Figure 2)**

Level B testing is performed on conclusion of abnormal sensory testing at level A. This level evaluates crude touch perception and should utilize Semmes-Weinstein monofilaments in order to quantify the patient's threshold. The nerve fibers represented include smaller diameter A-beta and delta fibers. A normal examination at this level suggests a mild impairment and an abnormal examination suggests a moderate nerve impairment.

Level C testing commences upon abnormal findings on level B testing. This level evaluates the slow conducting, unmyelinated C fibers, which solely carry information about noxious stimuli. Testing at this level is normally accomplished using pinprick testing, but quantitative testing can be performed using a thermal probe. A normal examination at this level suggests moderate nerve impairment while an increased pain threshold suggests severe impairment. No response is equivalent to anesthesia. [23]

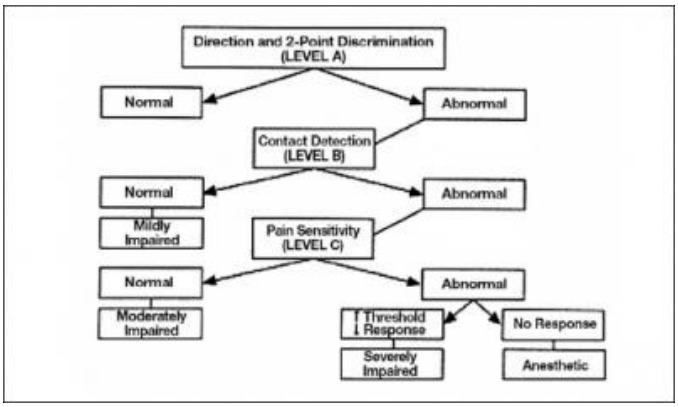


Figure 2: Algorithm for objective clinical neurosensory testing.

MANAGEMENT

A detailed and thorough evaluation will guide the surgeon to the best treatment option, including the timing for appropriate referral. In general, the indications for referral following nerve injury include but are not limited to the following:

- Lack of microsurgical training
- Observed nerve transection
- Complete postoperative anesthesia
- Persistent paresthesia with no improvement at 4-week follow-up
- Presence or development of dysesthesia

Nonsurgical Treatment

Patients with late dysesthesia following nerve injury are best managed medically. Some surgeons have the experience to manage these cases, but in most cases, a referral to a microneurosurgeon, neurologist, or facial pain specialist is warranted. Many systemic (**Box 1**) and topical (**Table 3**) medications are available.[24]

Box 1: Systemic Pharmacologic Agents

Local anesthetics
Corticosteroids
Nonsteroidal anti-inflammatory agents

Antidepressants
Narcotic analgesics
Anticonvulsants
Muscle relaxants
Benzodiazepines
Antisympathetic agents

Category	**Example**
Topical anesthetics	5% viscous lidocaine gel; 20% benzocaine gel; 2.5% lidocaine with 2.5% prilocaine
Neuropeptides	Capsaicin cream (0.025% or 0.075%)
Nonsteroidal anti-inflammatory drugs	Ketoprofen 10-20% PLO base; diclofenac 10-20% PLO base
Sympathomimetics	Clonidine 0.01% PLO base or patch
N-methyl-D-aspartate blocking agents	Ketamine 0.5% PLO base
Anticonvulsants	Carbamazepine 2% PLO base
Tricyclic antidepressants	Amitriptyline 2% PLO base
Antispasmodics	Baclofen 2% PLO base

PLO, pleuronic lecithin organogel

Table 3: Topical Agents.

Topical agents have the advantage of being easy to access over-the-counter in most instances and undergo very little systemic absorption. These medications can be combined with a eutectic mixture of local anesthetics, e.g., EMLA cream, which contains 2.5% lidocaine and 2.5% prilocaine. In contrast, systemic agents may have significant side effects and require long-term drug dosage adjustments based on clinical response.

For perioperative paresthesia subsequent to third molar extraction or other dento-alveolar procedures, a short course of systemic corticosteroid therapy is appropriate in an attempt to decrease perineural edema caused by the nerve injury. Several nutritional supplements and vitamins, such as B vitamin complex and L-methylfolate, have shown some limited success in improving regeneration of peripheral nerves.

INDICATIONS FOR MICRONEUROSURGERY

The decision of whether or not to perform a microneurosurgical repair of an injured peripheral trigeminal nerve depends upon multiple individual patient variables, including the history, initial presentation, and clinical course following the injury.

The majority of nerve injuries that we encounter during routine neurosensory evaluations are either closed or unwitnessed nerve injuries. In these patients, we have very limited clinical information to provide us with clues on the nature and extent of the injury. Therefore, these patients must be thoroughly examined and followed with serial neurosensory evaluations. Utilizing this approach will help to guide the surgeon in the correct identification and differentiation of low grade injuries that have the potential to improve spontaneously (Sunderland I and II) and those that

necessitate surgical intervention (Sunderland IV and V).

The general primary indications for nerve repair or nerve reconstruction include the following:

- Postoperative anesthesia or less than 50% residual sensation in an injury classified as a Sunderland grade III, IV, or V
- Early dysesthesia that may indicate early formation of a neuroma
- Injury or continuity defect in a nerve, resulting from trauma, pathology, or surgery, where surgical intervention is necessary to restore normal neural function
- Loss of normal neurologic function, resulting in anesthesia, paresthesia, and/or dysesthesia that persist longer than 3 months
- Progressively worsening hypoesthesia or dysesthesia

Hypoesthesia that is intolerable for the patient. In these instances, the patient must be informed of the reasonable expectation for recovery following microneurosurgery, which may be at or below their current level of sensation

Presence of a foreign body around the nerve (necessitates exploration and may lead to need for reconstruction)

A small number of patients present as candidates for immediate primary repair, and those include:

- Observed transection where the nerve ends are misaligned within mobile soft tissue. This often occurs in LN or mental nerve injuries
- Nerve injury where the nerve ends are exposed and surgically accessible. This often occurs in sagittal split osteotomies
- Ablative surgical procedures in which damage to the nerve is a planned event

Sometimes surgical intervention may not be needed or would not be helpful to the patient. Such situations include the following:

- Spontaneous improvement of neurosensory function based on quantitative sensory testing
- Late development of neuropathic pain
- Hypoesthesia that is acceptable to the patient
- Advanced age with the presence of an underlying systemic or neuropathic disease
- Extended delay from time of injury
- Signs of central sensitivity (e.g., regional dysesthesia, secondary hyperalgesia)
- Presence of clinical symptoms of autonomic origin (e.g., erythema, edema, hypersensitivity, burning sensation) that are indicative of autonomic nerve dysfunction rather than sensory nerve injury
- Unrealistic expectations on the part of the patient (e.g., demands immediate full recovery or resuming of sensory function with no pain)
- Neuropathic pain that is not alleviated via local anesthesia block [25-26]

In addition, there is no indication that surgery is helpful for neurosensory deficits associated with local anesthetic. Such patients can be particularly difficult to treat because of the difficulty in accessing the pterygomandibular space for microneurosurgical exploration. Further, a chemical lesion is very hard to visualize

clinically. However, Renton et al. showed that exploratory surgery improved symptoms and reduced the neuropathic area and improved quality of life in a small number of patients.

When unobserved IAN or LN injuries may have occurred, weekly follow-up assessments should be scheduled for a period of about 4–6 weeks. During these evaluations, if there is persistent or worsening paresthesia, an immediate referral to a microneurosurgeon is warranted.

Currently, the accepted recommendations are to consider microneurosurgery, when indicated, for the LN within 1–3 months after the injury and for the IAN within 3–6 months after the injury. The reason for the difference in time is that the IAN lies within a bony canal that provides a physiologic conduit to guide spontaneous regeneration. A LN injury lacks that conduit and lies in an area with continuous motion.

For an observed nerve injury, treatment is best tailored according to the severity of injury. For example, in traction injury, the patient should be evaluated weekly for 4–6 weeks to monitor for signs of spontaneous recovery, and then based on the last assessment, the decision to operate or not is made. When there is nerve compression with an implant of displaced bone into the canal from trauma or a bone grafting procedure, immediate decompression, and possibly surgical repair, should be performed, followed by serial neurosensory testing.

Chemical injury is another indication for immediate surgical intervention. When it comes to an avulsive injury such as a gunshot wound or when the LN becomes entangled with the bur during removal of a tori, a delayed primary repair performed at 3 weeks following the injury should be considered. This allows time to define the extent of injury and to assess the surrounding environment to determine if it is conducive to nerve repair surgery at a time when the levels of neurotropic and neurotrophic factors are highest.

After microneurosurgery, patients should be examined in the first few weeks to observe the initial healing following surgery and then again with serial neurosensory testing following the first evidence of return of sensation. Neurosensory re-education should be implemented early in the postoperative period.

OUTCOMES OF NERVE REPAIR

Many factors can influence the quality of neurosensory outcomes after nerve repair and outcomes often can be unpredictable. However, the more favorable factors a patient has (i.e., young age, clean transection, small gap), the greater the potential for a good outcome. One should keep in mind, however, that defining success following nerve repair varies widely among patients and surgeons since there is no accepted universal standardized assessment protocol and patients' perception of what is satisfactory as an outcome can vary widely.

The vast majority of the early reports on trigeminal nerve repair included minimal information regarding outcomes [27-29] and very little description of the methods applied to assess a successful result. [30] It was not until the late 1980s and early 1990s that publications began to report outcomes based on neurosensory testing. [31] In 1991, Zuniga reported on outcomes of IAN and LN repair in 10 patients using both objective and subjective measures. Patients and surgeons rated the overall outcomes as mostly good, although there were some differences in specific outcome ratings by surgeons and patients. [32] During this same time period, Donoff and Colin were also investigating outcomes in patients undergoing LN or IAN repair. They reported improvement in neurosensory function in 63% of patients who underwent

repair of 31 LN (77% in the anesthesia group and 42% in the pain-paresthesia group) and in 77% of patients who underwent IAN repair. [33] Although these studies reported very favorable results with nerve repair, other studies reported results that were not as favorable.

Overall, the bulk of the outcomes-based evidence suggests that nerve repair of the LN and IAN is a worthwhile endeavor. For example, Pogrel reported a review of his results for IAN and LN repair based on neurosensory testing. He reported that, of 51 trigeminal nerve injuries (17 IAN; 34 LN), 28 (54.9%) gained "some" or "good" improvement in sensory function. [34] In addition, Strauss et al. reported their results from repair of 28 IAN injuries evaluated by neurosensory testing. They found that 12 (42.9%) had "slight" improvement and 14 (50%) had "significant" improvement. Only 2 repairs resulted in "no improvement" (7.1%). [35] A long-term follow-up of 20 LN repairs by Rutner et al. using standardized neurosensory testing and patients' subjective evaluations of their degree of sensory recovery found that 15 patients (85%) gained improvement in all neurosensory testing parameters, whereas 18 patients (90%) judged the repair to have achieved "some improvement." [36] In a review of 60 repaired trigeminal nerve injuries including 4 IAN and 56 LN repairs, Susarla et al. reported that 45 (75%) achieved functional sensory recovery (Medical Research Council Scale score ≥S3) by 1 year post-surgery. [37] Finally, Bagheri et al. reported on their experience with repair of trigeminal nerve injuries. A total of 429 nerve repairs (186 IAN; 222 LN) were included and the success rate (achieving functional sensory recovery, Medical Research Council Scale grade >S3) varied from 81.7% for the IAN to 90.5% for the LN. The success rate for IAN repair increased to 87.3% when an autogenous nerve graft was used for reconstruction. [38]

MEASURING SUCCESS

The most common method used to measure sensory recovery in the maxillofacial region is the Medical Research Council Scale score. Successful outcomes are typically defined as anything at or above a grade of S3, which correlates with useful sensory function.

For repairs in the maxillofacial area, functional outcomes can include more than just recovery of touch sensation. Repair also can lead to recovery of the sense of taste. For example, Hillerup et al. and Zuniga et al. reported some recovery of taste response in 5 of 10 patients. [39-41] However, this contrasts with the results of Riediger et al., who found recovery of taste sensation in only one of their patients. [42] These studies help to demonstrate that repair in the maxillofacial region is still not ideal, given that some patients do not improve, others continue to have speech and taste problems, and recovery is rarely, if ever, complete.

Pain is another outcome that can be important to measure in maxillofacial nerve repair. Early studies on the effect of IAN repair provided little information on changes in the incidence of dysesthesia. However, when pain is present in the absence of a nerve defect, IAN decompression and neurolysis have shown positive results. [43] Interestingly, the presence of pain after surgery does not appear to impact functional sensory results. Bagheri et al. reported that the presence of pain after nerve injury did not affect the likelihood of achieving functional sensory recovery after repair in a statistically significant manner. Additionally, patients who did not have pain as a major complaint prior to nerve repair did not develop pain after repair. [44] In general, a deterioration in symptoms after nerve repair is very uncommon, and patients with no dysesthesia preoperatively

usually continue to be pain-free postoperatively. [45]

FACTORS AFFECTING SUCCESS OF NERVE REPAIR

In the literature, multiple factors have been associated with the success and quality of outcomes of nerve repair. The more favorable the factors, the more reliable and predictable the outcomes. These factors include:

1. Time Since the Initial Injury

As discussed above, the timing of nerve repair continues to be a topic of debate and the timing of surgical repair of trigeminal nerve injuries specifically has been accompanied by some uncertainty. This is likely because of the very limited number of prospective randomized studies evaluating the effect of treatment delay on outcomes of trigeminal nerve injuries in humans, which is most likely a result of the ethical difficulties in initiating such studies. However, a retrospective analysis of 41 cases of LN injuries that were treated, Ziccardi et al. identified that the most prognostic factor in the repair of LN injury is the interval between injury and surgery. [46]

Although microneurosurgical repair of mechanical peripheral trigeminal nerve injuries has generally been shown to be more successful when performed within weeks to a few months after the initial injury, parallel experimental and clinical evidence has shown that selected cases of partial nerve injuries may be successfully managed months to years after injury. As time passes after injury, the chance of a successful outcome diminishes as the distal nerve undergoes Wallerian degeneration, atrophy, and fibrosis, until it is replaced by scar tissue. Further, cell death occurs in the trigeminal ganglion, which decreases the total percentage of axons that can regenerate and negatively affects functional outcomes. Depending on several factors, including age and general health of the patient, ganglion cell death starts to occur approximately 12 months after injury. [47]

2. Type and Magnitude of Injury

It is inevitable that the extent of initial nerve injury will have a major impact on the outcome of nerve repair, and this has led to the commonly used classifications of nerve injury (e.g., Sunderland Grades I–V, Sunderland S (1951)). Neurosensory recovery tends to be better when the injury to the nerve is localized, as in a clean cut with a blade. Stretch-type or chemical injuries can lead to widespread and possibly irreversible damage to the nerve and result in poorer outcomes as compared with more localized types of injury. In addition, better preoperative sensory function is found to be associated with more rapid return of neurosensory function after repair. [48]

3. Vascularity of the Surgical Bed

Early revascularization at the neurorraphy site or within the nerve graft is paramount to the success of the nerve repair and the final outcome. Preparing a vascular surgical bed with no infection, scars, or debris will assure a faster revascularization process.

4. Length of the Nerve Defect

In general, the shorter the nerve graft, the better the outcome, and as the length of the graft increases, the predictability of recovery decreases. This has been attributed to the amount of time that is required for nerve regeneration to occur across each anastomosis area (7–14 days) and along the length of the nerve (0.2–3.0 mm/ day). The more time required for regeneration to reach the distal target, the higher the risk of atrophy and fibrous ingrowth in the distal nerve, resulting in a poorer outcome. However, reconstruction

of large gaps can be successful and in our experience, we have had quite good results utilizing allogenic nerve grafts for reconstruction of large nerve defects up to 7 cm.

5. Quality and Type of Repair

Surgeon experience and skill correlate well with the quality of repair, which ultimately affects the final neurosensory outcome. Correct handling of the nerve graft, adequate resection of the proximal and distal nerve ends, and meticulous neurorraphy techniques, including elimination of tension at the coaptation site, are important factors in high-quality nerve repair.

Care should be taken to handle the nerve graft only by the outer epineurium to prevent crushing the native basal lamina structure. Additionally, scar removal is critical because scar tissue impedes the regenerating axons and reducing the scar improves the potential for regeneration and better functional outcomes. [49]

Neurorrhaphy technique is another factor that can be critical to functional outcomes. Epineurial repair between the nerve ends of the graft and the host nerve used to be, and still is for some surgeons, the standard neurorrhaphy technique. However, in our experience, utilizing nerve conduits has usually eliminated the need for placing sutures at the coaptation face, which decreases the potential for scarring in the repair zone. Further, conduits provide a channel for direction of axonal sprouts from the proximal stump to the distal nerve stump, thus preventing the risk of axon escape and axonal misalignment. They also can limit the ingrowth of scar from the adjacent tissues and allow for diffusion of neurotrophic and neurotropic factors secreted by the Schwann cells of the distal stump. [50]

In addition to technique, selection of suture type is also important. The suture used should be thin and be minimally irritating to tissue; 7-0 to 10-0 monofilament nylon sutures are typically ideal. Repair of segmental nerve gaps can be accomplished in several ways. First, if the gap is small (<5 mm), a conduit may be used in a technique known as connector-assisted repair. This is where the nerve ends are approximated within the conduit and tension-relieving sutures are placed through the epineurium of the nerve at the outer edges of the conduit.

If the gap is larger, a nerve graft will be required. Autogenous nerve has been shown to provide a reconstruction with good neurosensory outcomes. However, it also can be associated with varying degrees of clinical complications, such as donor site morbidity, limited availability, diameter mismatch, and formation of neuromas at the harvest site. [51]

An alternative to autograft is human peripheral nerve allograft (PNA). PNA provides an unlimited graft source, is available in varying sizes and lengths, and is without the morbidities associated with autogenous nerve harvesting. Zuniga et al. have reported their experience using PNA for reconstruction of large discontinuity defects. Their retrospective analysis of the neurosensory outcomes in 26 patients with 28 LN and IAN discontinuities reconstructed with allogenic nerve was conducted to evaluate the effectiveness and safety of this treatment. Twenty-one subjects with 23 nerve defects met the inclusion criteria and 87% had improved neurosensory scores with no reported adverse experiences after surgery. Similar levels of improvement were achieved for both nerve types (87% for LN and 88% for IAN). Further, 100% sensory improvement was achieved in injuries repaired within 90 days of the injury compared with 77% in injuries repaired after 90 days. [52]

6. Age of Patient and General Medical Health

Patient age is another important factor that can influence outcomes. In general, the younger the patient, the better the results. Younger patients have greater potential for nerve regeneration, and better healing and metabolic rates than older patients. In LN repair, the potential for neurosensory recovery decreases by 5.5% per year for every year of age in patients over 45 years. Similar results have been observed with IAN repair, where there is a significant drop-off in success rates after the age of 51 years. [53, 54]

7. Patient Expectations

An important factor that is often overlooked yet plays a significant role in achieving acceptable outcomes is patient expectations. An appropriate evaluation, correct diagnosis, and thorough discussion of all potential outcomes, complications, and treatment options, including no treatment, is of paramount importance in avoiding or minimizing unexpected outcomes by the patient and the surgeon.

TIMING OF SURGICAL REPAIR

Immediate microneurosurgical repair/reconstruction is advantageous in that it limits the degree of neural degeneration and scar tissue formation. [55] In instances where an immediate repair is not feasible because of surgeon inexperience, surgical access limitations, or patient care problems, a delayed or early secondary repair within 7–10 days after the injury can achieve similar results to an immediate repair. [56]

Immediate repair appears to be desirable because several papers have suggested that delayed repair results in poorer outcomes as compared with early repair, especially when the repair occurs more than 12 months after the injury. [57-59] For example, Meyer [60] reported a 90% success rate for trigeminal nerve repair if the repair was undertaken within 3 months. This success rate was reduced to 10% at 12 months. However, the outcome measures used were not defined. In a subsequent study from the same group, the authors defined useful sensory recovery using the Medical Research Council Scale score. In this study, the authors calculated that 94% of their patients gained 'useful sensory recovery' when repair was undertaken within 6 months of the injury, but only 85.4% gained the same level of improvement if repaired later than 6 months after injury. [61] In a similar evaluation of 64 patients, Susarla et al., 2007 reported that 'functional sensory recovery' was achieved in 93% of patients who underwent repair within 90 days of injury, compared with 78% of subjects who had a later repair. [62] This difference was not significant, although recovery was achieved more rapidly in the early repair group. Further, as more than one type of surgical procedure was used and the groups were poorly matched, it is difficult to draw clear conclusions. Interestingly, a more recent study from the same group found no correlation between early repair and any positive outcome measurement. [63]

There does appear to be some discrepancy in the literature related to the impact of delayed repair on functional outcomes because several other studies have shown no effect. In one series of patients, there was no significant correlation between repair delay and any measure of outcomes. [64] Interestingly, another study also found no correlation when evaluating the whole group of patients, but when those patients with very poor recovery were excluded (where other factors are likely to have been involved), there were better outcomes with early repair. [65] A likely explanation for the discrepancy between the different clinical studies is that when a large population is studied, other factors may be dominant and may mask the effect. Arguably, this debate is of limited importance because few would dispute that early referral to a center that

manages trigeminal nerve injuries is appropriate and that surgical intervention should be undertaken as soon as it is clear that there will not be satisfactory spontaneous recovery. The difficulty is the

timing of that decision, so as to avoid unnecessary surgery in a patient who would recover adequate sensation spontaneously, while also avoiding a long period of monitoring that could hamper the healing potential. [66] In addition, several studies have shown that late repair can be worthwhile, [67] so this surgical intervention should still be considered in cases of late presentation.

In some cases, a small delay in repair may be a more appropriate option than immediate repair. For example, in trauma patients, where there is a grossly contaminated surgical bed, the patient is medically unstable with a high risk for additional anesthesia and surgery, and/or the surgeon lacks microneurosurgical training. Delaying the repair in these cases is acceptable and usually amounts to a delay of only days or weeks. [68]

Longer delay in repairing nerve injuries also may occur in cases where nerve injury is not suspected or recognized, the patient desires no further treatment of a subjectively acceptable neurosensory function, or the patient is lost to follow-up. In patients with nerve injury whose function is improving, nerve repair should be delayed and serial examinations repeated as long as they continue to show subjective and objective improvement at each subsequent visit.

SURGICAL TREATMENT

Microneurosurgical repair generally involves a set of surgical procedures starting with surgical exposure, identification of the nerve and the injured site after dissection of the nerve from the surrounding tissues, assessment of the degree of injury, neurolysis, and neuroma resection and the scar tissue in the area, if present, resection of the proximal and distal nerve stumps to a healthy-looking nerve, and repair with a direct anastomosis or the use of a nerve graft or conduit. The armamentarium includes typical micro-instruments, either surgical loupe or operating room microscope, either can be used based on the preference of the microsurgeon.

CASE PRESENTATIONS

Case 1

The first case is one of the most common presentations for a LN injury. A 19-year-old healthy woman was referred to our office for evaluation 2 months after removal of her wisdom teeth with reported anesthesia and ageusia of the right tongue. Her oral surgeon reported that he did not witness or suspect lingual nerve injury. The patient had been observed for spontaneous recovery and had taken a steroid dose pack after reporting the numbness at follow-up. The patient reported no improvement since the surgery and there was no pain. Clinical examination revealed well-healed extraction sockets, but a scar in the mucosa that was curved to the medial aspect of the ramus, which could have been the area where the LN was injured, and evaluation of the LN distribution showed complete anesthesia. The panoramic reconstruction of the CBCT radiograph taken on presentation to our office is shown in **Figure 3A**. It is always recommended to obtain a CBCT even in LN injury cases, because evaluation of the surgical area might give a clue as to how the injury happened, like a notching in the lingual cortical plate, and also unmask the presence of a foreign body compressing the nerve.

After a discussion of the findings with the patient, the plan was to undertake a surgical repair of the injured nerve. Once

in the operating room, the patient was intubated nasally, exposure of the injured area commenced, a mucoperiosteal flap was raised along the lingual surface of the molars on the affected side. After cutting behind the second molar to the distal buccal line angle of the crown, a buccal releasing incision was employed similar to that used in routine third molar removal (**Figure 3B**). This tissue was then dissected in a subperiosteal plane and a 2-0 silk suture was used to suture the soft tissue to the contralateral posterior maxilla, thus providing retraction (**Figure 3C**). This approach rapidly exposed the LN immediately deep to the periosteum. Incision through the periosteum freed the LN and the site of injury was often readily apparent (**Figure 3D**). External neurolysis was performed by freeing the nerve from the surrounding tissues, including the lateral adhesive neuroma that is commonly seen with the periosteum or from within the healing third molar extraction site itself. At this point, nerve stump preparation followed, and both the proximal and distal nerve ends were freed and excised clear of any appearance of neuroma. The clinical end point of this procedure is fascicles that appear to mushroom out of the nerve end. In a hospital setting, frozen section specimens can be used to guide resection but this is time-consuming and often the pathologist must be made aware that the surgeon is resecting to healthy nerve or too often a read of "neuroma" or "neural tissue" may be returned. In general, direct repair is possible when the LN defect is <10 mm and when the IAN defect is <5 mm, keeping in mind the more dissection to free the nerve, the more the risk of scarring and possible injury to the nerve in other places.

In this patient, the required resection resulted in a defect that could not be reapproximated without undue tension at the repair site. A 2 cm × 4–5 mm Axogen Avance allogenic nerve interpositional graft was used with porcine small intestine submucosa nerve connectors to aid in the coaptation both proximally and distally. After determining the needed graft length, the nerve was prepared on a sterile Mayo stand (**Figure 3E**). The tension-free nature of the graft can be seen in **Figure 3F**. Tension must be avoided because it leads to vascular compromise and induced scarring. When in doubt the authors recommend grafting and avoiding any tension because this will lead to poor results. There are many different ways of performing neurorraphy, and a commonly used technique is epineural sutures. Generally, a 7-0 to 9-0 monofilament nonresorbable nylon suture is preferred. Two or three epineural sutures should be used on each side to prevent rotation of the nerve stumps. In cases where there is a small neuroma or there is no need for grafting, a direct neurorraphy can be made with epineural sutures and covered with a connector. A comparison of direct repair with a grafted repair can be seen in **Figure 3G**. Closure in these cases is similar to wisdom tooth removal and patients often report less discomfort than what they experienced at their original surgery (**Figure 3H**). Postoperatively, most patients have complete anesthesia. In general, the nerve regeneration process progresses at approximately 1 mm/day from the cell body to the target site. When an interposition graft is used, the process of regeneration slows through the graft site and recovery is variable. After the initial healing of the surgical site, the patient is requested to come for a follow-up visit at the 3-month mark or earlier if there is any evidence of return of sensation for sensory re-education exercises that include self-performance of a two-point discrimination or brushstroke directional discrimination test while looking in a mirror, and comparing the result with that on the contralateral side. This patient went on to have functional sensory recovery at 6 months.

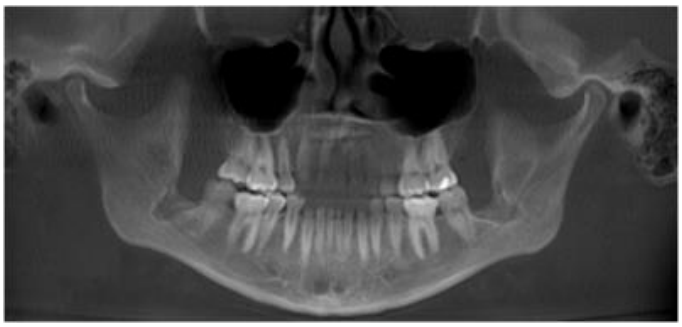


Figure 3A: A panoramic reconstruction of a CBCT showing the sites of previous wisdom tooth removal.

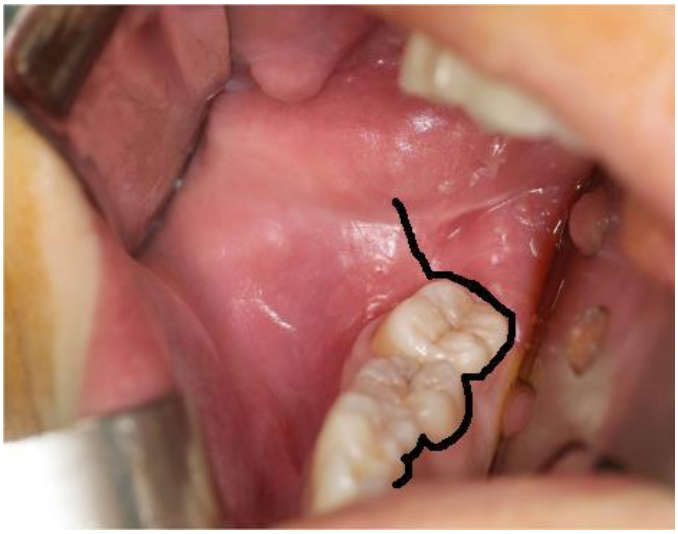


Figure 3B: The black line represents the typical incision used to raise the flap on approaching the lingual nerve.

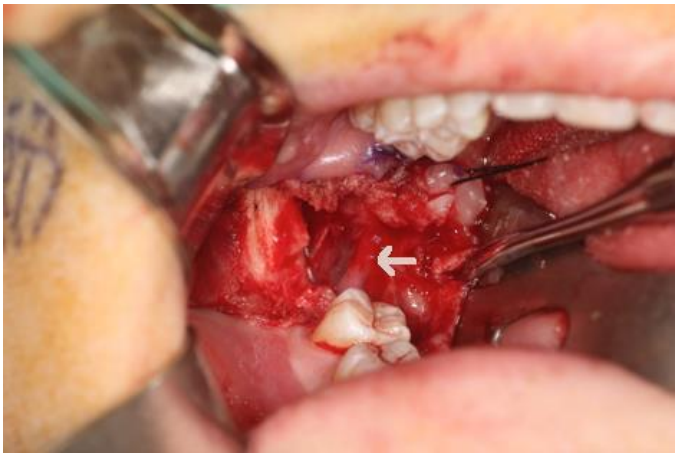


Figure 3C: Once reflected in a subperiosteal plane the lingual nerve is readily visible and easily exposed. Here the notching in the lingual cortical plate can be observed.

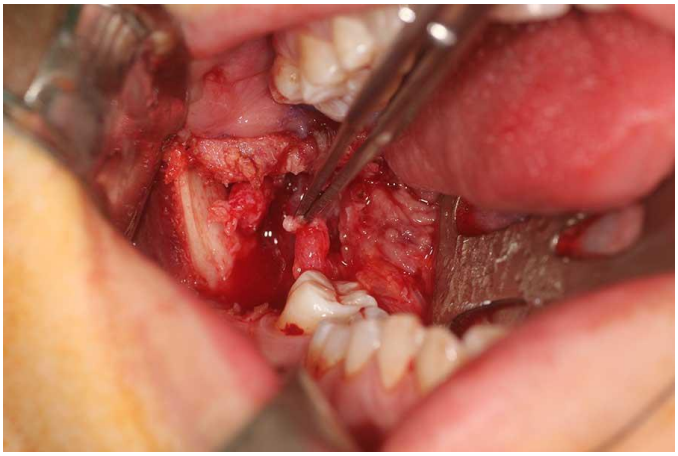


Figure 3D: After incision, the periosteum and proximal and distal nerve stumps can be freed. The distal portion of a neuroma is being held here prior to resection.

Figure 3E: An Axogen Avance 2 cm × 4–5 mm nerve graft is prepared prior to suturing in place. Doing this outside the mouth decreases the complexity of the procedure.

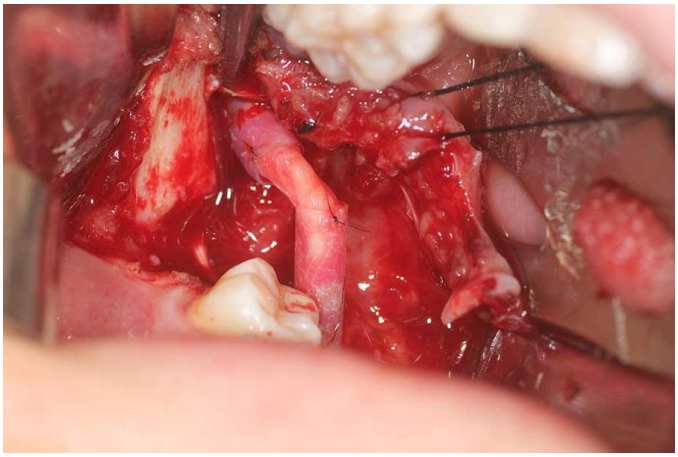
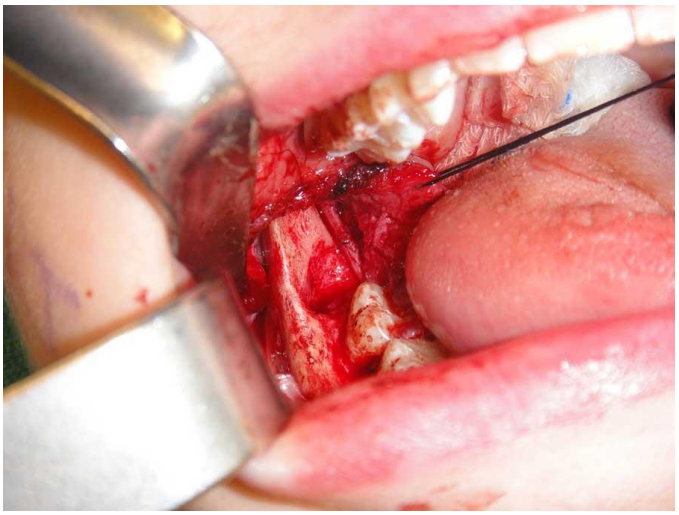


Figure 3F: The nerve graft is then sutured in place with no tension. The authors tend to do the proximal coaptation first as it is often the most difficult to perform.



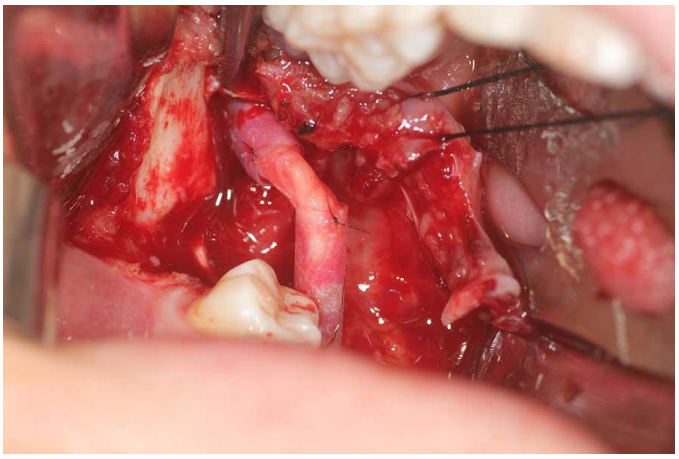


Figure 3G: The two repairs contrasted here show a direct repair (top) and our grafted case (bottom). The taut nature of the nerve at top likely indicates an unacceptable amount of tension at repair site.

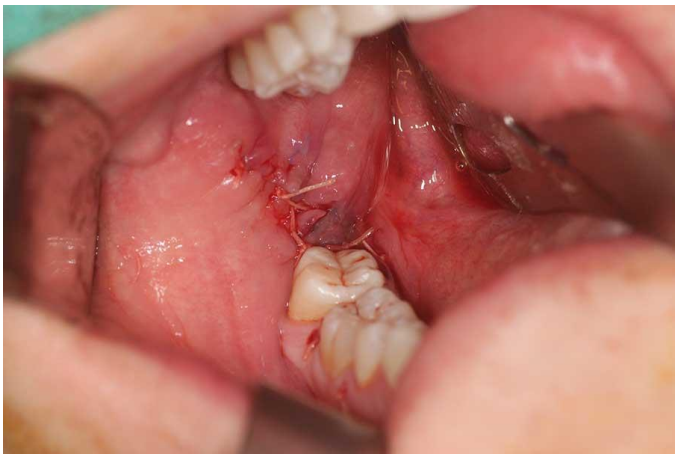


Figure 3H: Closure of the incision used in approaching the lingual nerve often resembles that used in a third molar removal.

Case 2

The second case is a 52-year-old patient who presented as referral from her dentist. The dentist reported that during placement of the implant, the patient felt a severe electric shock. A postoperative X-ray showed compression vs. transection of the nerve. (**Figure 4AD**)

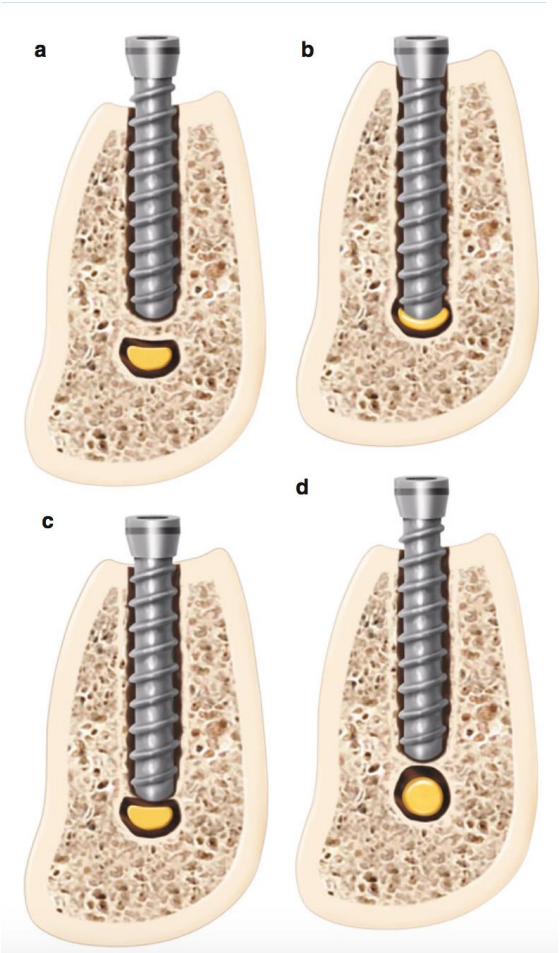


*Figure 4: Different mechanisms by which the nerve can be injured by placement of an implant. **A,** Collapse of the inferior alveolar canal by placing the implant beyond the planed osteotomy leading to inward fracture and compression of the nerve. **B,** Direct injury. **C,** Compression of the superior cortex of the IAC leading to compression of the nerve. **D,**Remodeling of the IAC cortex leading to narrowing and compression of the nerve.*

The patient was evaluated 5 hours after the surgery and found to have complete right lower lip and chin numbness and severe pain in the surgical site. In similar cases, when the nerve is partially compressed by the implant, removal of the implant and replacement with a shorter implant is recommended. In this case, the

injury was severe and an exploratory surgery was warranted. First, a full thickness mucoperiosteal flap was reflected, exposing the mandible and identifying the mental nerve. A lateral cortectomy was performed. This procedure can be performed even when the injury is in the area of the third molar or alternatively a sagittal split ramp osteotomy can be performed. Upon evaluation of the area, the nerve was found to be compressed and pushed inferiorly. The implant was removed gently. (**Figure 4E**)

Figure 4E: The implant is removed.

The injured site was inspected again, and revealed partial severance of the nerve by 30%, and the remaining part of the nerve was lateralized during placement of the implant and looked clinically healthy (**Figure 4F**).

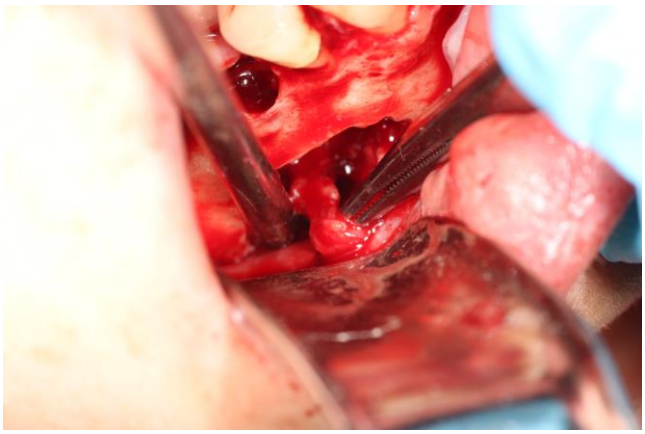


Figure 4F: Evaluation of the injured nerve after severance of the incisor branches and lateralization. Partial severance of the nerve is evident.

Internal neurolysis was performed, identifying the injured part of the nerve, which was trimmed to healthy looking fascicles. Minimal internal neurolysis is recommended to decrease scar formation. A nerve protector was placed over the injured area and secured in position to the epineurium with 8/0 proline sutures (**Figure 4G**).

The surgical site was closed, and the patient elected not to have the implant replaced. This patient went on to have functional sensory recovery at 4 months.

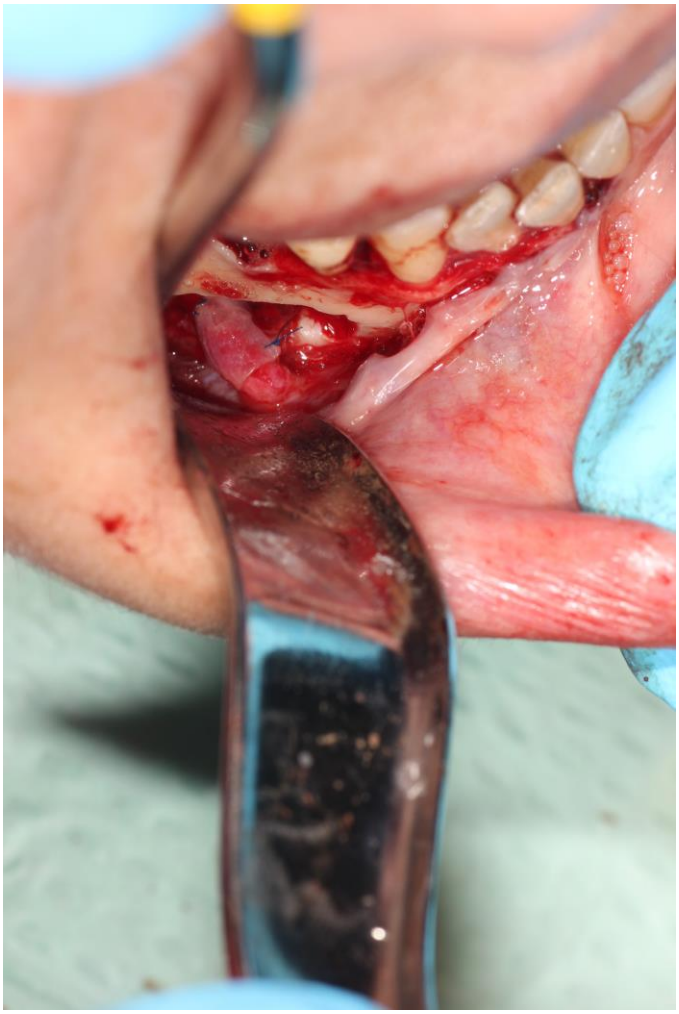


Figure 4G: Following internal neurolysis, a nerve protector was placed and secured in position with 8/0 proline sutures.

Case 3

A 30-year-old healthy woman was referred for evaluation of persistent anesthesia of the right lower lip and chin subsequent to surgical extraction of tooth #32 approximately 3 months earlier. The surgeon reported that the right inferior alveolar nerve was visualized but was not sure if it was injured. The patient reported that she bit her lower lip once in a while, and in the previous few weeks she had started to have considerable pain. Clinical examination revealed complete numbness in the distribution of the right inferior alveolar nerve. CBCT shows the close proximity of the IAN canal in association with the roots of the third molar, in addition to a small bone spicule at the level of the apices (**Figure 5A**).

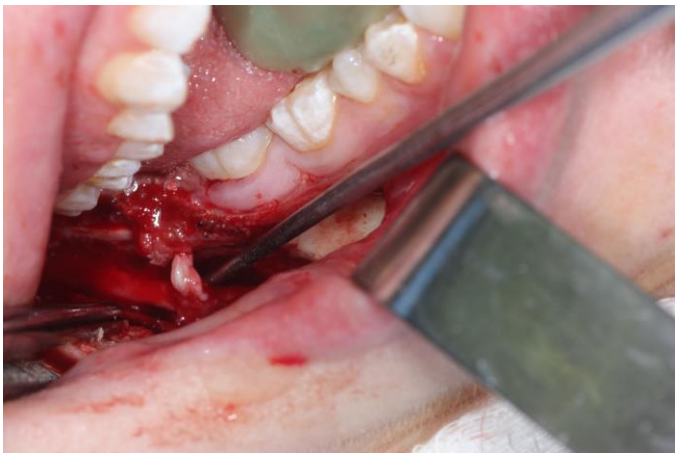


Figure 5A: Close association of the IAN canal and the roots of the third molar.

After discussion of the findings with the patient, the plan was to take the patient to the operating room for a nerve repair procedure. In general for IAN injury such as this, it is not unreasonable to observe the patient for few weeks for possible spontaneous recovery, but the fact that there was no improvement since the surgery and the patient reported pain that might indicate neuroma formation, the decision was to operate sooner. The surgical approach in these cases can be extra-oral or intra-oral. The latter is more commonly used, with the advantage of having an extra-oral scar, but the downside is the limited access compared to the extra-oral approach.

In this patient, the exposure was through an intraoral incision following the same old third molar surgery scar with extension anteriorly to provide better access. Exposure of the nerve can be performed utilizing a lateral cortectomy or a sagittal split ramp osteotomy similar to what was done in this case. Once the nerve is identified, a thorough evaluation should be performed to determine the extent of the injury and the presence of a neuroma (**Figure 5B**), which was present in this case.

Figure 5B: Following the sagittal split ramps osteotomy, the nerve was found to be totally severed and associated with neuroma.

Excision of the neuroma and preparation of the nerve stumps was performed. The nerve gap was 12 mm and the decision was to utilize allograft interposition graft. Two epineural sutures were used on each side and then the nerve was wrapped with a nerve protector (**Figure 5C**). The proximal segment was fixed to the distal segment with positional screws and the incision was closed. This patient went on to have functional sensory recovery at 6 months.

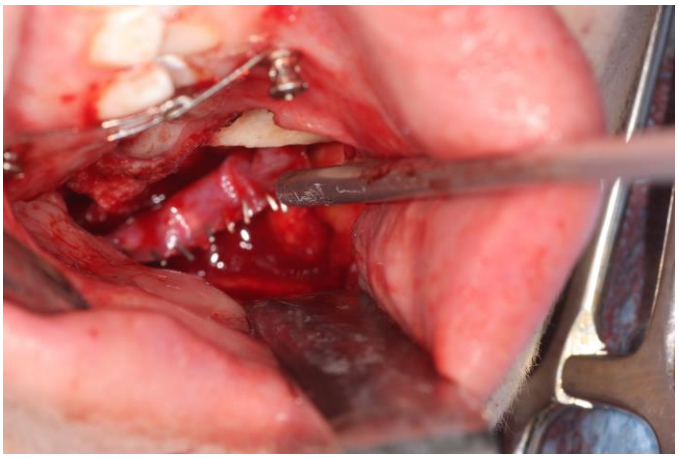


Figure 5C: Allogenic nerve interpositional graft and a nerve protector.

MEDICOLEGAL ISSUES

Since the "Medical Malpractice Crisis" in the 1970s, there has been an increase in the number of lawsuits and a marked increase in payments to plaintiffs. This has led the medical community to initiate litigation analysis to help understand the causes, and focus on the prevention of suits by health care provider education and management strategies. As part of the medical community, oral and maxillofacial surgeons have been through these changes and realized the potential medicolegal sequelae associated with the nerve injuries that may result from some of the most common procedures performed, like extraction of third molars and implant placements and others, and the subsequent adverse results that account for a significant number of malpractice claims. [69]

All oral and maxillofacial surgeons should have a basic knowledge of nerve injuries and should be able to recognize, evaluate, diagnose and manage nerve injuries, and if not, refer when appropriate following the guidelines of the legal parameters of care, with which the legal profession is familiar. [70]

These include the following:

- Spontaneous sensory recovery occurs in most but not all patients.
- IAN has a higher chance of spontaneous sensory recovery compared to LN.
- Appropriate documentation of nerve injuries including history, evaluation (neurosensory testing), diagnosis (Seddon and Sunderland), and referral to a microsurgeon in a timely fashion when appropriate, especially in observed injuries.
- Patients with nerve injuries should be followed up for a minimum of 4 weeks. When complete recovery occurs during that period no further treatment is required. On the other hand if neurosensory dysfunction continues past that period, that indicates a higher-grade injury with uncertain spontaneous neurosensory recovery. A referral to a microsurgeon should be considered.
- Nerve injuries that continue to improve (objective and/or subjective) may be followed up periodically in anticipation of neurosensory recovery. Once improvement ceases for a period of time, it usually does not restart again.
- Most nerve injuries resolve within 3 to 9 months, but only if improvement begins before 3 months. Patients who are anesthetic at 3 months usually do not achieve significant neurosensory recovery, and immediate referral to a microsurgeon is warranted.
- When objective and subjective assessment has not improved or normalized by 4 months, and the patient finds the partial sensory loss and/or painful sensations

unacceptable, the patient should be refereed for microsurgery. Delaying nerve repair decreases the potential for successful outcomes.

- As with any surgical procedure, full discussion of the procedure, alternatives, and complications including the potential of nerve injury should be performed. An uninformed upset patient with nerve injury is less likely to improve with any treatment.

- Microsurgical nerve repair is more likely to objectively improve the response to neurosensory testing and/or to decrease functional impairment than it is to diminish pain or the patient's perception of feeling.

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