

Inferior Alveolar Nerve and Lingual Nerve Repair

Leon Assael

Armamentarium

#1 Periosteal elevator	Dental curette	Needle holder
#9 Periosteal elevator	Fissure/round bur	Rongeurs
#15 Scalpel blade	Gilmore probe	Small and large luxator
#150/#151 Forceps	Hemostat	Small and large straight elevator
#190/#191 Elevators	Irrigation syringe/sterile saline	Surgical handpiece
Appropriate sutures	Local anesthetic with vasoconstrictor	Suture scissors
Bone file	Minnesota cheek retractor	
Cryer elevators	Molt curettes	

History of the Procedure

The great golden age anatomist Galen was uncertain of the role of peripheral nerves and initially did not distinguish them from tendons. Subsequently he sectioned the recurring laryngeal nerve in pigs to demonstrate peripheral nerve action.¹ Injuries to peripheral nerves were poorly understood due to the nondiscrete nature of these injuries. However, in his 1795 treatise, John Haighton² reported that “an animate machine differs from an inanimate one in nothing more conspicuously than in its power of repairing its injuries.” He identified the need for peripheral nerve continuity to preserve diaphragmatic function. His experiments on dogs, apparently after division of the vagus nerve in the neck (although he called it the VIII nerve), demonstrate the apparent ability of peripheral cranial nerves to undergo repair. He sectioned one side, both sides, and both sides in sequence over weeks to demonstrate not only the action of the peripheral nerve, but also its ability to undergo repair. His drawings of dissections completed in functionally restored animals demonstrate the spontaneous repair of peripheral nerves.

The first description of technique for reanastomosis of peripheral nerve neurotmesis is likely that of Gabriel Ferrara of Venice in 1608.³ The first successful modern peripheral nerve repairs were performed during hand surgery for traumatic neurotmesis. In 1973 Millesi⁴ emphasized the importance of fascicular alignment and perineural suturing to achieve favorable results in hand surgery. The results of hand surgery demonstrated the proof of motor and

sensory recovery after neurotmesis could be obtained in humans.

Modern peripheral trigeminal nerve surgery was hampered by the development of access techniques and the irregular nature of referral for these sensory injuries which, compared to motor injuries of the hand, did not create as much disability for most. However, the presence of anesthesia dolorosa ineffectively treated with nerve ablation and the vexing issue of lingual nerve anesthesia and dysesthesia prompted surgeons in the 1970s to develop techniques for repair. Hausamen et al.⁵ demonstrated a technique for the inferior alveolar nerve with interpositional grafting. Phillip Worthington, Ralph Merrill, Bruce Donoff, Tony Pogrel, and John Gregg, among others, pioneered the advancement of these techniques in contemporary surgical practice. Today large outcome studies demonstrating functional sensory recovery have established the utility of direct repair and interpositional grafting of peripheral trigeminal nerve injuries.

Indications for the Use of the Procedure

Peripheral trigeminal nerve injury can result from mechanical injury to the affected nerve after facial trauma or surgical intervention (Figure 16-1). Mandible and zygomatico-orbital fractures commonly injure afferent V3 and V2, respectively. Removal of impacted mandibular third molars, mandibular fracture, mandibular tumors, and placement of dental implants commonly affect V3. In addition to mechanical injuries, chemical nerve injury of the trigeminal nerve results from

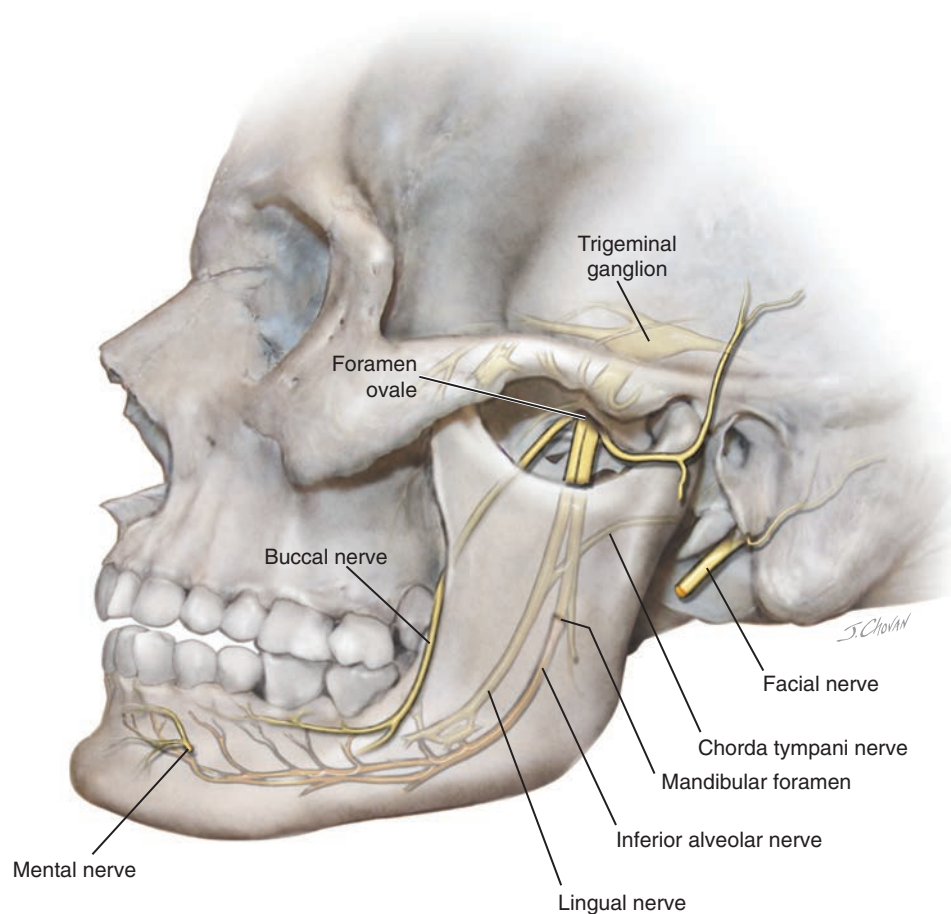


Figure 16-1 Location of the lingual and inferior alveolar nerves.

endodontic medicaments, amide local anesthetics, and anti-septics such as alcohol. Thermal injury to the trigeminal nerve can occur from electrocautery, heat from rotary instruments, or warm gutta-percha (Figure 16-2). Ischemic injury may occur due to endoneural injection of epinephrine, radiation therapy, or infarction of the peripheral nerve vas neurosum.

Injuries to the sensory branches of the trigeminal nerve result in afferent defects characterized by alterations or absence of sensation. The alterations in sensation can be noxious or painful, or they may be innocuous, with mild tingling or just dullness.

Neuropathic pain may be associated with peripheral nerve injuries of branches of the trigeminal nerve. All injuries to the sensory branches of the trigeminal nerve result in afferent defects characterized by alterations or absence of sensation. Although in many cases there is simply a diminution of sensation, these alterations in sensation can be noxious or painful. Surgical management of hypoesthesia or anesthesia is an established method, whereas procedures for dysesthesia are less well defined, and recommendations for surgical management have not been as well characterized.

Over the course of surgical and nonsurgical treatment, the improvement of patients to the point of elimination of pain applies only to a small minority. Despite these shortcomings,

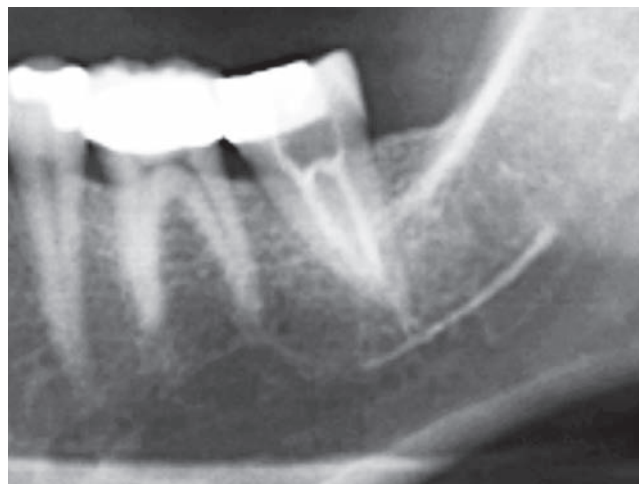


Figure 16-2 Extrusion of root canal filling material past apex of second molar.

treatment is capable of reducing the impact of pain from an unbearable burden on the patient's well-being to a tolerable condition with substantial improvements in quality of life. For patients with well-defined injuries and those with nociceptive inflammatory components, the prognosis is better yet with surgical intervention.

A variety of definitions of these conditions are used and are consistent with the characterization of neuropathic pain in a patient with a trigeminal nerve injury. The following definitions and clinical presentations are noted.

Hypoesthesia: A diminution of sensation, compared to a control stimulus, with the absence of pain. In such patients, sharp may feel dull. The patient produces errors in two-point and directional stimuli. No areas of anesthesia are present, and the findings are due either to decreased neural density (e.g., incomplete repair) without neurotmesis or to neuropraxia and thus are transient.

Anesthesia: The absence of sensation with the absence of pain to any stimulus. These patients often treat the anesthetic part as a foreign body (e.g., a bolus). Hyper-salivation, dysarthria, dysphagia, and speech articulation issues, among others, are noted when the perception of an absent body part is noted. This typically is due to neurotmesis, which may be physical separation, neuroma in continuity, lateral adhesive neuroma, kinking of the nerve, or some other impingement or infarction of the nerve.

Elicited neuropathic pain: With the absence of stimulus there is an absence of pain, but upon stimulus with what would normally produce no pain, a painful response is elicited. In such cases the patient withdraws from normally nonpainful activities, such as shaving, using lipstick, kissing, or chewing. This may be due to reafferentation without somatosensory modulation of pain fibers.

Spontaneous neuropathic pain: Prolonged neuropathic pain occurs and is persistent after the stimulus has been removed.

Dysesthesia: A noxious response occurs to a stimulus; it may be elicited or spontaneous.

Hyperalgesia: An increased response occurs to a minimally noxious stimulus; for example, a pin produces a greater than expected and more prolonged pain response or a response not typical of the stimulus (e.g., burning).

Allodynia: A painful response occurs to an innocuous stimulus.

Hyperpathia: A prolonged and explosive pain response occurs to an innocuous stimulus; it may be continuous.

Anesthesia dolorosa: The persistence of pain after neurectomies or amputation of a body part. Although there is no afferent function (e.g., anesthesia is seen on examination), pain is perceived in the body part previously supplied by the nerve.

Diagnosis of the patient with an afferent sensation defect or neuropathic pain due to peripheral nerve injury includes an anamnestic assessment to determine the likely cause of injury, the time since injury, the evolution of the injury and sensation over time, and the effect on activities of daily living. It is important to determine whether there remain

nociceptive components to the pain (due to inflammation, infection, a persistent lesion, local inflammatory aspects, or mechanical impingement) that would explain afferent defects. Palpation of the site for redness, swelling, and pain, in addition to imaging with computed tomography (CT) to assess for pathologic defects, can be helpful. Limited areas of cherrylike redness in the tissue may be due to a neuroma, which in the case of inferior alveolar and lingual nerve injuries can fill third molar extraction sites and replace normal mucosal tissue.

Afferent sensory defects should be carefully mapped. This can provide assistance in understanding the expected findings at surgery if intervention is carried out. The characteristics of any pain, whether constant or due to stimulation, are important in proportion to the stimulus or if sustained beyond the stimulus.

The diagnostic nerve block is a useful indicator of whether the injury is generating pain in the peripheral nerve and where in the nerve it is located. In general, the absence of both pain and sensation after peripheral nerve block indicates a peripheral mediation and source of that pain, generally due to a neuroma at the site of injury. However, the risk of false positives should be noted with regard to the potential that centrally mediated pain will result in an absence of pain after peripheral nerve block, but that pain is still dependent on a central nervous system (CNS) modulation. Thus, in many cases the patient with centrally mediated pain has but an alteration in that pain experience after peripheral nerve block. That alteration is usually toward mitigating the level of pain. For the patient with a peripheral source of pain, beginning the block with lidocaine anesthesia at the most peripheral site may determine whether a neuroma or perineural inflammation is a factor. For example, if the intent is to determine whether the injury is due to mechanical disturbance of a nerve at the time of surgery or whether it is in the location of the nerve block, it would be best for the patient to have a diagnostic block first at the site of possible mechanical injury (e.g., at the lingual crest for a third molar removal). If painful dysesthesia is persistent after this block, a Gow-Gates block (e.g., proximal to the site of nerve block) can be done to determine whether the location of injury is due to injection rather than surgery.

Diagnostic radiology for the patient with trigeminal nerve injury includes a panoramic radiograph, other local views of the injured site if needed, and a maxillofacial CT scan. Magnetic resonance imaging (MRI) is sometimes helpful in locating sites of injury, inflammation, impingement, or masses. Imaging for neuropathic pain differs somewhat from that used for other patients with trigeminal nerve injury. An attempt to define the inflammatory aspects of the injury can be made with MRI of the region, including mapping of the site of nerve injury with three-dimensional reconstruction (Figure 16-3).

Neuromas can be identified, in addition to inflammation in the perineural tissues, bone, or associated soft tissues. The extent of perineural inflammation is particularly important

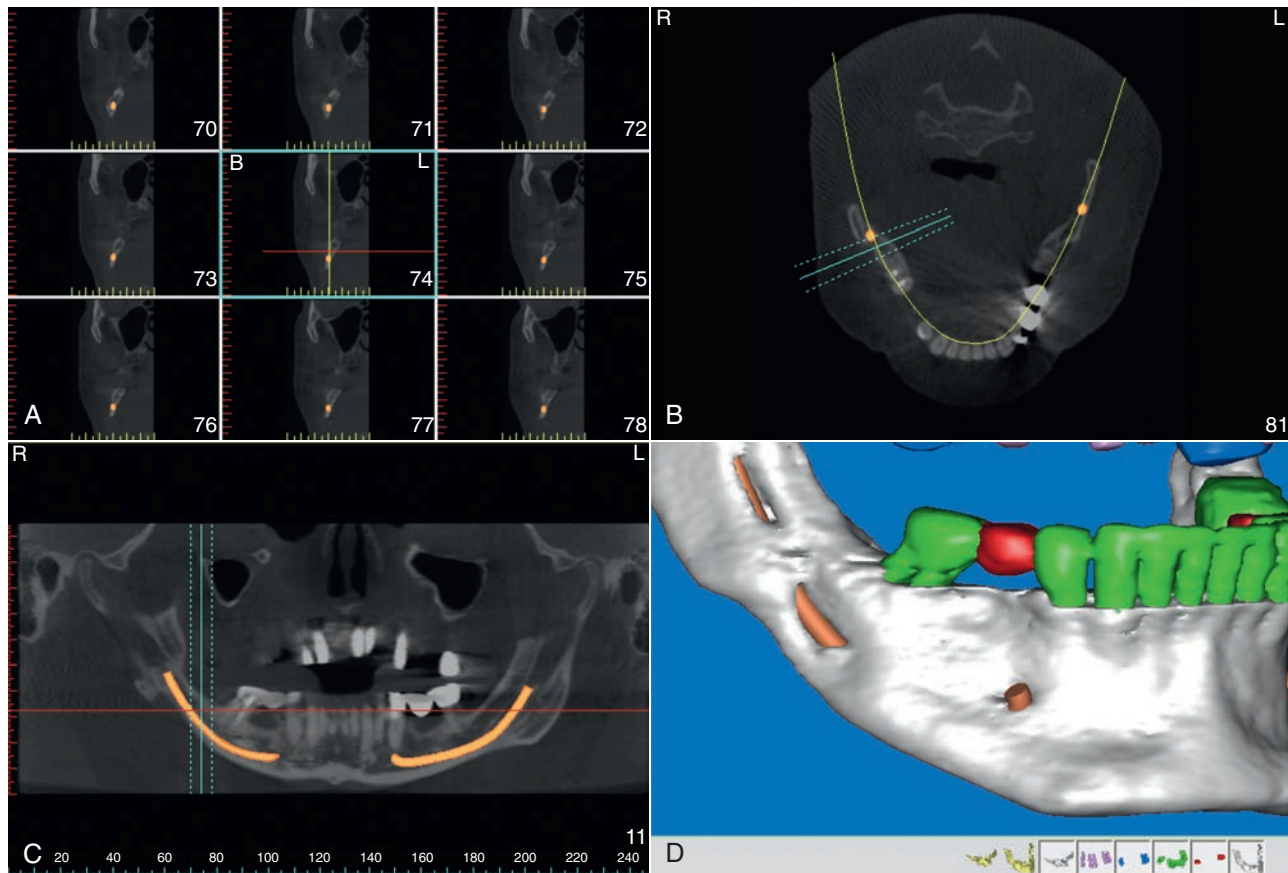


Figure 16-3 Preoperative planning. Cone beam CT scan-generated views of inferior alveolar nerve position.

in chemical nerve injury because this may not be completely apparent under the operating microscope. The need to resect associated bone or soft tissue also can be assessed with MRI. MRI neurography can be used to determine whether an inflammatory reaction or neural tissue turgor is apparent proximal to the injury. Deafferentation distal to the nerve can also sometimes be indicated on MRI neurography. CT or MRI of the brain is indicated to rule out a CNS lesion associated with the presence of neuropathic pain or to assess neurodegenerative findings, such as demyelinating diseases.

Limitations and Contraindications

All patients should be counseled regarding the lack of improvement of symptoms from peripheral nerve surgery. The most important prognostic factor in improvement is the length of time from injury to surgical intervention. In addition, the medical status of the patient must be considered. Medical management with neuroleptic medications also should be considered as a nonsurgical treatment option.

TECHNIQUE: Inferior Alveolar Nerve Repair**STEP 1: Patient Preparation**

The patient is immobilized with a head rest, towels and tape, sandbags, or similar methods.

STEP 2: Incision

Ability to access the injured site transorally is usual, with full access to the inferior alveolar nerve from the lip to high in the infratemporal fossae; to the lingual nerve from the oral tongue to the chorda tympani insertion; and for the infraorbital nerve from the cheek to the internal orbit via a transoral/transantral approach. All repairs should be done under a two-headed, binocular operating microscope with the ability to visualize the operative site on

a camera monitor; the ability to record is recommended. This allows both the surgeon and the assistant to visualize exactly the same site and from the same angle. It also allows the surgical team to operate via the microscope objectives or by observation on the monitor. This enables the surgical team to change hand position and gain access across a far greater range of variables than when operating with loupes (Figure 16-4, A).

STEP 3: Exposure and Osteotomy

Inferior alveolar nerve access can be performed via a crest or buccal osteotomy. Using a saw or rotary instrument, the lateral cortex is removed; generally the osteotomy cut is to 3 to 5 mm. Of note is that the inferior alveolar nerve courses to the buccal as it proceeds to the mental foramen. It often takes a buccal loop in the third molar area before returning toward the lingual in the

body. In addition, it may be tethered to the lingual in the mental foramen as it releases the incisive nerve to the anterior mandible. Nearly one in five inferior alveolar nerves are bifid in the angle and posterior body of the mandible. Once the bone has been removed from the lateral cortex, nerve probes can enter the canal, and microcurettes can be used to relieve the bony canal laterally (Figure 16-4, B).

STEP 4: Lateralization of the Inferior Alveolar Nerve

The inferior alveolar nerve should be removed from its canal and lateralized at least 1 cm on each side of the injury. Small branches to the pulps of teeth are neurovascular in nature; they sometimes can be preserved and should be, if possible. Although the main nutrient artery to a pulp may be severed, anastomotic blood supply from the periodontal ligament is likely to maintain vitality if the

apices are not disturbed. Such proximity is especially an issue for lower second molar teeth. If held by the mental foramen distally, separating the incisive nerve or lateralizing it as well may be needed. The osteotomy for inferior alveolar nerve access should determine that any entrapments have been eliminated and that any inflamed bone or granulation tissue has been removed.

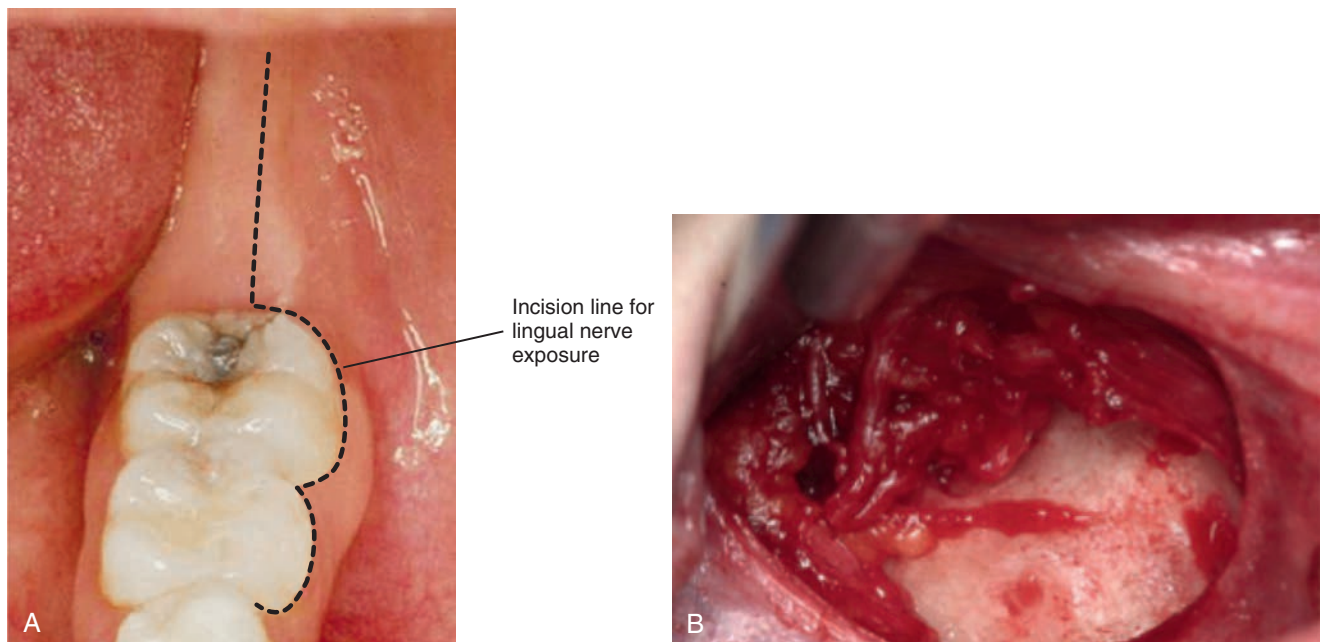


Figure 16-4 A, Access incision and osteotomy to inferior alveolar nerve. B, Exposure of injured site on inferior alveolar nerve.

TECHNIQUE: Lingual Nerve Repair**STEP 1: Incision**

Lingual nerve access can be obtained by repeating a third molar or sagittal osteotomy incision from the temporal crest lateral/superiorly to the distal of the second molar and with an anterior buccal vestibular release. No releasing incisions are desirable on

the lingual mucosa because these often tear into the parenchyma of the tongue. Once the buccal flap has been released, the lingual flap is elevated from the temporal crest to the premolar area, including the gingival papillae.

STEP 2: Retraction

With a peanut or sponge, the sublingual gland can be brought up with the lingual flap, revealing the distal lingual nerve and Wharton's duct.

STEP 3: Nerve Skeletonization

The proximal end of the lingual nerve must be distinguished from the temporalis tendon; careful preservation of this tendon when the flap is raised to its lingual aspect makes this distinction easier. Of note in the proximal dissection is that the chorda tympani joins the lingual nerve about 2 cm above the occlusal

plane from the posterior and anterior branches to the pterygoid muscle; it should be preserved if noted. This dissection should reveal about 2.5 to 3 cm of lingual nerve, making the whole injury, in addition to tension-free repair or grafting, easier to understand (Figure 16-5, *A* and *B*).

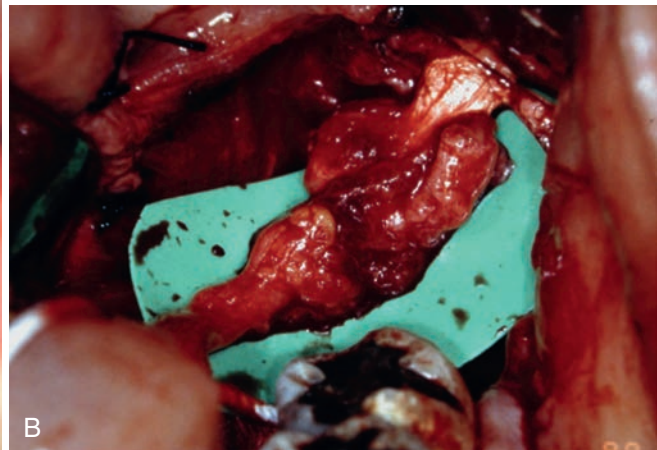
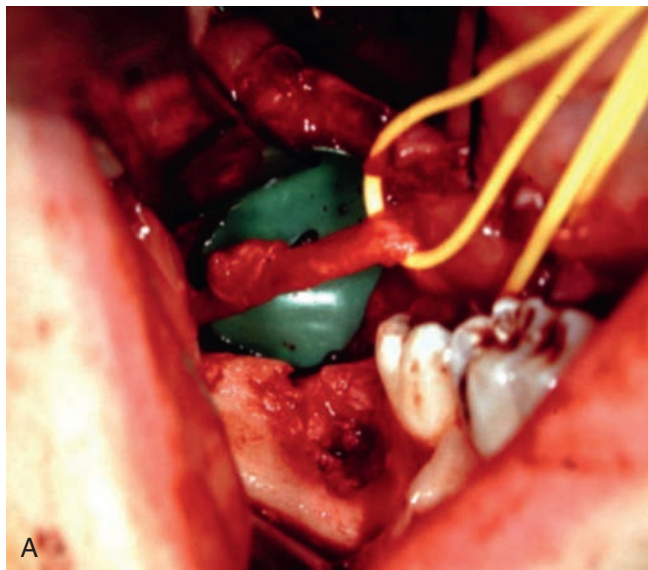


Figure 16-5 **A**, Exposure of injured site on lingual nerve. **B**, Removal of neuromas, lateral or in continuity.

TECHNIQUE: Lingual Nerve Repair—cont'd**STEP 4: Nerve Anastomoses Common to Both the Lingual and Inferior Alveolar Nerves**

The goal in surgical repair is to have a contiguous nerve free of pathologic components. Amputation neuroma, lateral adhesive neuromas, and other neuromas in continuity should be removed. External pressure or kinking should be relieved. If the nerve is intact and a compressive neuropathy is the underlying cause of symptoms, external or internal neurolysis can play a role in improving sensation and reducing pain. For nerves with

neurotmesis or resected neuromas, tension-free epineurial repair is indicated. This typically is performed with sutures in the epineurium (e.g., proline 7-0 to 9-0, at least three to orient the nerve). Insufficient suturing may result in loss of coaptation during healing. Sealing the epineurium with fibrin glue or Avitene can be done as a way to promote early adaptation. Animal studies of nerve repair demonstrate partial restoration of tensile strength of the nerve in as little as 1 week after surgery (Figure 16-5, *C* to *E*).

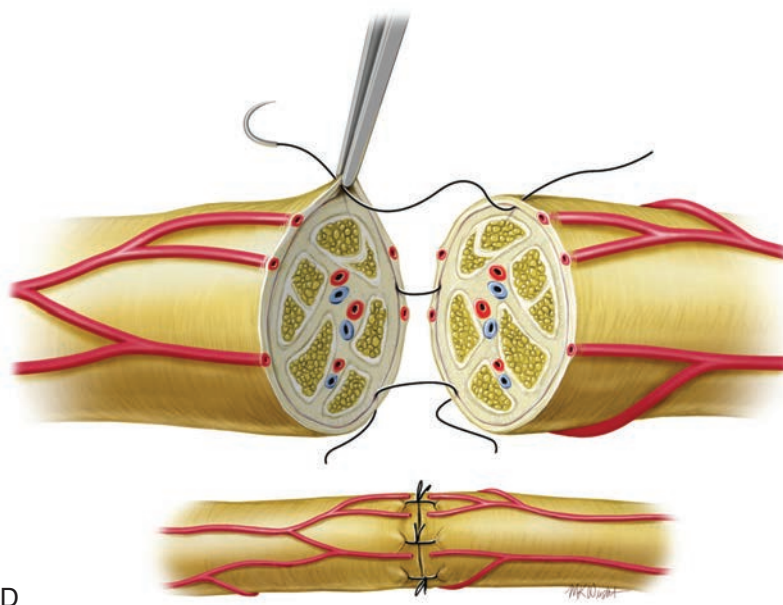
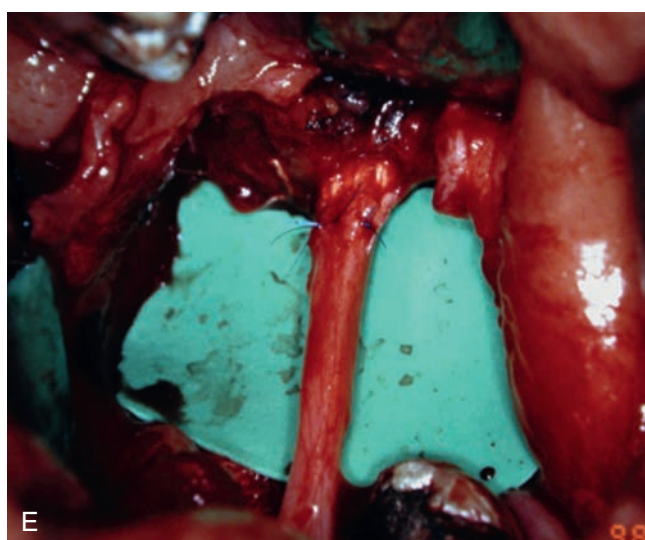
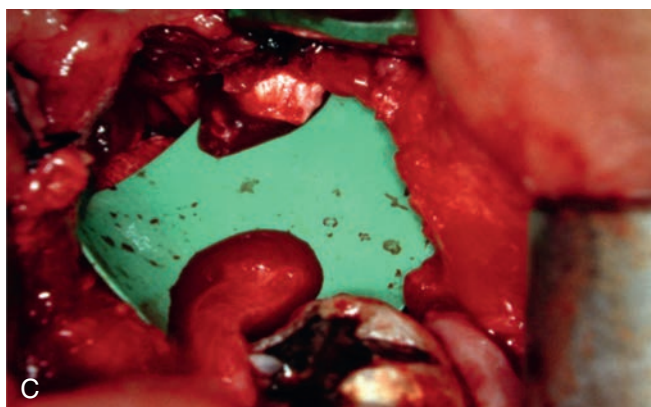


Figure 16-5, cont'd *C*, Identified proximal distal nerve stumps. *D*, Epineurial repair. *E*, Intraoperative view of completed epineurial nerve repair.

Alternative Techniques and Modifications

The surgical procedures for the patient with neuropathic pain associated with trigeminal nerve injury are not the same as for patients with simple sensory nerve injury. Consideration for greater resection of the injured portion of the nerve and replacement with a nerve graft is more likely because the persistence of pain may be due to continued perineural inflammation across any portion of the injured nerve. For example, chemical nerve injury due to endodontic procedures often leaves a leathery-appearing perineurium across up to centimeters of the inferior alveolar nerve. Identification of all the portions of the nerve demonstrating visual or MRI injury is needed to determine the amount of resection and nerve reanastomosis with cable graft. Experience in the past several years has been with axogen and Avance grafts for this purpose. Vein grafts or autogenous great auricular or sural grafts also may be performed.

Neuropathic pain due to osteoradionecrosis or osteonecrosis often is also associated with ischemia. Relieving ischemia, such as with hyperbaric oxygen therapy or free flap surgery, often can reduce pain. It is unknown whether this pain reduction is due to increased tissue oxygen tension or to some other etiology.

Neurectomy, cryotherapy, and chemical denervation remain infrequent options for neuropathic pain. The time-honored technique of peripheral neurectomy remains in use despite continued concerns about recidivism. In one recent study,⁶ the rate of pain return after neurectomy was just 2 of 30 patients after 3 years of follow-up.

Avoidance and Management of Intraoperative Complications

Prevention of associated tissue trauma is especially important during dissection of the floor of the mouth. The sublingual

plexus of veins as well as the sublingual artery and perforators on the lingual aspect of the mandible should be addressed carefully to ensure hemostasis. Muscles such as the superior pharyngeal constrictor should be released judiciously from their aponeurotic attachments if needed to avoid muscle bleeding and edema. Dissection within the mandible during inferior alveolar nerve repair should be performed to avoid damage to the teeth and perforation through the lingual aspect of the mandible where possible. The use of bone curettes can be helpful to avoid further injury to the inferior alveolar nerve during dissection of the mandible.

Postoperative Considerations

Peripheral nerve stimulators have been used to mitigate post-traumatic trigeminal neuropathic pain after repair.⁷ Large case series and extensive follow-up are available to assess peripheral stimulators for the treatment of trigeminal postherpetic neuralgia. Johnson uses implanted subcutaneous pulse generators. Although these have provided 50% pain relief in 70% of patients, they may have less practical utility in V3-located injuries.⁸

Patients are advised not to smoke after nerve repair. Vitamin B complex can be useful in the treatment of neuropathic pain. In a blinded study,⁹ the combination of B₁, B₆, and B₁₂, when used in an animal model, diminished pain behaviors associated with trigeminal induced traumatic neuropathic pain.

Neurosensory retraining exercises, with the assistance of occupational therapists, have been shown to mitigate symptoms and facilitate functional sensory recovery.

References

1. Gregg J: Historical perspectives on trigeminal nerve injuries. In Miloro M, editor: *Trigeminal nerve injuries*, Berlin, 2013, Springer-Verlag.
2. Haighton J: An experimental inquiry concerning the reproduction of nerves, *Philos Trans R Soc Lond* 85:190, 1795.
3. Ferrara G: Nuova selva di chirurgia, divisa in tre parti. Nella prima sono gli avvertimenti del manual, & artificioso modo di curare molte, e gravi infermità del corpo humano. Nella seconda sono molti medicamenti esquisiti, con le figure de' ferri, ò instrumenti necessarii per essercitar l'arte della chirurgia. Nella terza parimente si contengono molti rari medicamenti per distillationi, con le figure in ultimo de' vasi, e fornelli appartenenti all'arte distillatoriaio.
4. Sebastian Combi, Venice (1608). [Translated in Little K, Zomorodi A, Selznick L, Friedman A: An eclectic history of peripheral nerve surgery, *Neurosurg Clin North Am* 15:109, 2004.]
5. Millesi G: Microsurgery of peripheral nerves, *Hand* 5:157, 1973.
6. Hauamen J, Samii M, Schmidseider R: Restoring sensation to the cut inferior alveolar nerve by direct anastomosis or by free autologous nerve grafting, *Plast Reconstr Surg* 54:83, 1973.
7. Agrawal SM, Kambalimath DH: Peripheral neurectomy: a minimally invasive treatment for trigeminal neuralgia—a retrospective study, *J Maxillofac Oral Surg* 10:195, 2011.
8. Lenciq S, Cohen J, Patin D: A minimally invasive surgical technique for the treatment of posttraumatic trigeminal neuropathic pain with peripheral nerve stimulation, *Pain Physician* 15:E725, 2012.
9. Johnson MD, Burchiel KJ: Peripheral stimulation for treatment of trigeminal postherpetic neuralgia and trigeminal posttraumatic neuropathic pain: a pilot study, *Neurosurgery* 55:135, 2004.
10. Kopruszinski CM, Reis RC, Chichorro JG: B vitamins relieve neuropathic pain behaviors induced by infraorbital nerve constriction in rats, *Life Sci* 10(91):1187-1195, 2012.