

Lingual Nerve Injury

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Lingual nerve injury is a common complication following dental and medical procedures. The clinical presentation of lingual nerve injury, its epidemiology, predisposing factors, and anatomy are explored in an attempt to identify those patients at risk for developing neuropathic pain. Nonsurgical and surgical therapies also are discussed.

Key words: lingual nerve, neuropathic pain, third molar removal

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CASE HISTORY

Three years ago, a 24-year-old woman underwent local anesthetic blocks and removal of upper and lower molars by her dentist. She subsequently reported numbness and tingling of the left side of her tongue and the floor of her mouth that have persisted up to the present time. About 3 or 4 times per week, she experiences “aching and throbbing” in the same distribution, reduced but not relieved by naproxen. She also has had disturbance of taste since the procedure; she does not like the flavor of any type of meat and has become a vegetarian. Findings from the neurologic examination are normal, except for decreased pinprick sensation over the anterior two thirds of the left side of her tongue and the floor of her mouth. Three oral surgeons have seen her, all of whom recommended nonsurgical treatment.

INTRODUCTION

Lingual nerve injury causing numbness, dysesthesia, paresthesia, and dysgeusia may complicate invasive dental and surgical therapies.¹ Lingual neuropathy also may be precipitated by infection and various metabolic, toxic, or systemic disorders. The lingual nerve is a branch of the third division of the trigeminal

nerve. Although injury to the first or second trigeminal divisions may occur, this is less common (with the exception of herpes zoster, which typically affects the first division). The increased risk for surgical injury to the mandibular division may relate to anatomical issues. Trigeminal nerve injuries have been reported following tooth removal, tumor removal, osteotomy, distal wedge techniques, implant placement, and general dental therapies such as nerve block, crown preparation, and endodontic procedures.²⁻⁷ Laryngoscopy, intubation, tongue manipulation, chemotherapy, radiation, and ischemic events may cause injury, also.⁸⁻¹² While all nerves respond similarly to injury, there may be genetic, hormonal, anatomical, physiologic, behavioral, or other factors that influence recovery.¹³

CLASSIFICATION

In 1943, Seddon classified nerve injury as: (1) neuropraxia—conduction block resulting from mild trauma, without axonal damage, and with resolution of sensory deficit within days to months; (2) axonotmesis—more severe injury, with preservation of the nerve sheath but afferent fiber degeneration, and incomplete sensory recovery; neuroma formation may occur, and the typical clinical presentation involves severe dysesthesia; (3) neurotmesis—most severe injury, with nerve severance and anesthesia in the nerve distribution, and no sensory recovery (especially if the nerve course is in soft tissue; if the nerve course is in bone, regeneration may occur).¹⁴

In 1951, Sunderland classified nerve injury based on the degree of tissue injury.¹⁵ Under his system,

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first-degree injury, of which there are 3 types, is similar to Seddon's neuropraxia. Type 1 results from nerve trunk manipulation, mild traction, or mild compression and is thought to reflect transient ischemia. If blood flow is restored, nerve function usually returns to normal; with more prolonged ischemia, permanent injury and anesthesia may occur. Type 2 results from more prominent traction or compression that produces intrafascicular edema, decreased blood flow, and a conduction block. Recovery is variable. Type 3 injuries result from severe nerve traction or compression causing segmental mechanical disruption of the myelin sheaths and demyelination. Recovery is delayed and sensory loss may be permanent.¹⁶

Second-, third-, and fourth-degree injuries correspond with Seddon's classification of axonotmesis. The afferent or efferent fibers are damaged, but endoneurium, perineurium, and epineurium remain intact. Surgical decompression may be necessary, and recovery requires axonal regeneration. Third-degree injury occurs when the intrafascicular tissue components (axons and endoneurium) are damaged. If there is poor clinical recovery, surgical reconstruction may be needed. Fourth-degree injury implies fascicular disruption: all components are damaged and only the epineurium remains intact. The prognosis is poor, and surgical reconstruction may be indicated.

Fifth-degree injury implies nerve transection. Surgical approximation and coaptation may be required.

MacKinnon and Dellon, in 1988, added sixth-degree injury to describe a variation wherein a combination of Sunderland's 5 degrees of injury coexist within the same nerve trunk.¹⁷

Attempting to correlate histological findings with clinical data, Rood concluded that the clinical course is most reflective of the degree of initial injury.¹⁸ If there is complete numbness that improves over time, this is indicative of first- or second-degree injury per the Sunderland classification. The presence of complete numbness initially does not indicate nerve severance.⁵

CLINICAL PRESENTATION

Following trigeminal sensory nerve injury, patients present with various types of sensory disturbance including anesthesia, paresthesia, dysesthesia,

hyperesthesia, hyperalgesia, hypoalgesia, and allodynia.¹⁹ The taste fibers from the chorda tympani are conveyed to the tongue via the lingual nerve, and disturbance of taste also occurs frequently. The disturbance is compensated partially by the other taste receptors within the oral cavity.²⁰ Recovery of altered taste is not expected in fourth- and fifth-degree Sunderland injuries.²¹

INCIDENCE

The incidence of lingual nerve injury consequent to surgery depends upon the procedure being performed, the surgeon's experience, procedure methodology, and certain patient-specific factors.²²⁻²⁶ Following mandibular third molar removal, the incidence of permanent nerve damage is reported to vary between 0.5% and 2%.^{21,24-26} In a US survey of oral and maxillofacial surgeons, 76% reported having had cases complicated by lingual nerve anesthesia, dysesthesia, or paresthesia. In 18.6% of cases, the sensory symptoms failed to resolve, and only 3 cases underwent surgical nerve repair.

Permanent injury to the nerve following nerve block is rare, occurring in 1:175 000 procedures.³ In the non-English literature, the incidence is cited as between 1:67 000 and 1:200 000.²² In a retrospective study, Haas reported the incidence as 1:785 000 injections. Krafft reported an incidence of 0.15% of lingual sensory disturbance associated with local anesthetic administration. The incidence of permanent injury was 0.008%.

The location of impacted teeth and a person's age also contribute to the incidence of injury. Wisdom teeth that are lingually oriented, or those where there is direct contact with the inferior alveolar canal, are more likely to be associated with injury. It has been reported that if the teeth are partially erupted, the risk of lingual nerve paresthesia is increased.²² There is a greater incidence of injury as persons become older; those over aged 35 years are at greatest risk. Injury seems to adversely affect females more often than males. Surgical duration is another variable that may contribute.²²

Incidence of lingual nerve injury following mandibular third molar removal may depend on the surgical technique. Raising and retracting a lingual

mucoperiosteal flap with a Howarth periosteal elevator may result in more frequent injury, but this is temporary. Rood reported an initial incidence of 6.6%^{18,26}; Blackburn and Bramley, 11%²⁴; and VonArx and Simpson, 22%.²⁷ The majority recover over a few days or a few months, but symptoms remain in 0.5%²⁴ to 0.6% of patients.²⁵ Using a lingual flap and Obwegeser periosteal elevator also may increase the incidence, especially when distal site ostectomy is performed.²²

PREDISPOSING FACTORS

Nerve Location.—Close proximity of the lingual nerve to the cortex of the mandible may cause entrapment.⁵ A number of studies agree there is great variability in lingual and inferior alveolar nerve position. Carter and Keen indicate that although the inferior alveolar nerve is considered to be localized to the bony canal, in as many as 40% of cases there are large branches outside the canal subjecting them to possible injury.²⁸ Variability in the lingual nerve anatomy associated with location of the third molar provides for higher risk of injury. The nerve may lie above the lingual plate or even on the alveolar crest.^{4,29,30} The number of cases varies from 8.8% to 17.6% in results from anatomical dissections, and 15% in a magnetic resonance imaging (MRI) study.^{29,30} It is emphasized in these studies that the clinician can not use only the lingual plate as a reference to ensure lingual nerve protection, as often the nerve is in the soft tissue above the bone. It may lie in contact with the lingual plate in 57.4% to 62% of cases as seen in anatomical dissections,^{4,29} and in 25% of cases evaluated by MRI.³⁰

Hormonal Changes.—Most orofacial trigeminal dysesthesia occurs in women usually in their 40s.^{31,32} Sex-based differences have been seen in many pain disorders. The relationship and role of sex hormones in the generation and perpetuation of central sensitization is not fully understood, but is obviously important.³³ In a neuropathic pain model using partial sciatic nerve ligation, female rats were more likely to develop allodynia.³⁴ In studies comparing female rats that had been ovariectomized, there was a greater chance that those with estrogen were more likely to develop allodynia after injury than those without estrogen.³⁵ Further understanding of central pain inhibition may help explain these phenomena. In a recent report by Pogral

and Thamby, more females than males were affected with neuropathic pain.³⁶ This is also consistent with reports of trigeminal deafferentation following dental procedures such as prosthodontic and endodontic interventions.³²

Genetics.—Although there is still no direct evidence of a genetic predisposition for nerve injury, the recovery and resultant pain may have a genetic base.

ANATOMY

The lingual nerve branches from the third division of the trigeminal nerve after it exits the foramen ovale. It carries with it taste fibers from the chorda tympani that supply the anterior two thirds of the tongue. The lingual nerve may be round, oval, or flat and varies in size from 1.53 mm to 4.5 mm.⁴ The nerve is either monofascicular or oligofascicular in structure at the pterygomandibular space, making it susceptible to injury by injection in this area.⁷³ It runs deep to the lateral pterygoid muscle parallel to the inferior alveolar nerve, lying anterior and medial to it. It then runs between the internal and medial pterygoid muscles and passes obliquely over the superior pharyngeal constrictor and styloglossus muscles before approaching the side of the tongue. The nerve courses submucosally in contact with the periosteum, covering the lingual or medial wall of the third molar socket. It crosses the Wharton duct and then loops back to cross it again. It may run below and behind the tooth before swerving superficially across the surface of the mylohyoid muscle.

NEUROPHYSIOLOGY POSTINJURY

Compression.—Peripheral nerve compression may result in a neuropathic pain syndrome or sensory deficit. The acute response to compression is edema and inflammation. The first stage is blood-nerve barrier changes resulting in subperineural and endoneural edema. This leads to connective tissue changes with perineural and endoneural thickening, followed by localized nerve fiber changes exhibited as demyelination. Wallerian degeneration then becomes apparent. The mechanism of compression includes both mechanical deforming forces and ischemic changes.³⁷⁻⁴² Epineural hematoma, due to a local anesthetic injection, may be a common cause of nerve injury.⁴³

In nerve compression neuropathy, large myelinated fibers are lost and A delta and C fibers spared. There is an increase in circulating substance P and calcitonin gene-related peptide, and a decrease in dynorphin at the dorsal horn. There also may be activation of the *N*-methyl-D-aspartate (NMDA) receptor.³⁹ These changes are responsible for the dysesthesia or paresthesia. The response to peripheral or central nerve injury may involve neuroinflammation. This is defined as an infiltration of immune cells into the injured site.⁴⁰ An experimental neuritis provokes a local immune response, characterized by endoneural infiltration of granulocytes and CD4 and CD8 T lymphocytes.⁴¹ This reaction occurs even without significant axonal damage. These findings support the presence of a distinct neuroinflammatory event without significant nerve injury in generating persistent neuropathic pain and paresthesia.⁴² As we begin to understand the inflammation/immune-induced tissue damage and repair and how it relates to neuropathic pain, the opportunities for targeting the neuroinflammatory system in chronic pain prevention and therapy can be developed. Nerve compression may occur with tracheal intubation, use of a laryngeal mask airway, tongue retraction, or jaw protrusion.^{10,11,44,45,74}

Compartment Syndrome.—The nerve injury seen in a compartment syndrome is similar to that seen in compression, but the effects are largely due to ischemia caused by diminished flow in the compartment. Increased venous pressure results from increased local tissue pressure, such as from inflammation or edema. There is a decrease in the arteriovenous gradient, diminished blood flow, and decreased oxygen delivery to the nerve. The reversibility is dependent on how long and how much pressure is applied. Early decompression or anti-inflammatory therapy may be useful in preventing long-term pain and numbness. Because the lingual nerve is often adjacent to the lingual plate, the pressure created by suturing the mucosa accompanied by postsurgical edema and bleeding may result in a compartmentlike syndrome.

Stretch Injury.—Injury following stretch or traction varies considerably from fascicle to fascicle and along the nerve length. The injury may begin with axonal rupture followed by rupture of endoneural tubes, perineurium, and finally endoneurium. There is also

a theory that the epineurium may be the first structure to rupture, followed by perineurium axons and then endoneurium.⁴⁶ The injury may occur in several places along the length of the nerve. Lundborg and Rydevik have studied the deleterious effects stretching may have on the physiologic function of a nerve. As little as 8% stretch produced changes in intraneural blood flow. Complete cessation in arterial blood flow occurred with 15% elongation.³⁷ Injuries to the lingual nerve from laryngoscope, intubation, and possibly jaw retraction have postulated stretching as a mechanism.

Chemical Injury.—In dentistry, chemicals that create neural sensitization are commonly used. Some agents that have been implicated include eugenol, alcohol, phenol, and paraformaldehyde-containing endodontic filling materials.^{47,75} Once the nerve is exposed to the chemical, an inflammatory response ensues. Depending on the nerve involved, a compartment syndrome or neuroinflammatory nerve injury may develop. Depending on the duration and severity of the damage, as well as other predisposing factors, a neuropathic pain may develop.

Nerve Injection Injury.—Unexplained nerve injury following dental procedures, especially tooth removal, may be caused by intraneural injection, creating permanent damage.⁴⁸⁻⁵¹ Scarring and fibrosis that prevent axonal regeneration disrupt the fascicular architecture. There is a resultant conduction block, and dysesthesia may develop. Local anesthetic may produce persistent neurosensory deficit following enzymatic hydrolysis.^{2,50-52} Neural hematoma also has been postulated as a cause of persistent sensory change. The anesthetic itself injected into the nerve along with the vasoconstrictors and preservatives may injure the nerve permanently.⁵³⁻⁵⁶ Nickel suggests that under exceptional conditions, lidocaine metabolism may produce nerve dysfunction. If the lidocaine molecule is hydrolyzed while still bound to the receptor and the alcohol by-product persists, the metabolite may disrupt nerve conduction causing paresthesia.⁵⁶ Using 5% lidocaine in proximity to the peripheral nerve may cause permanent neural ablation.⁵⁷ The mechanism of neurotoxicity is unknown. Contaminating the anesthetic-containing Carpules with alcohol may cause nerve injury. Some anesthetics appear more likely to cause nerve injury. Articaine and prilocaine have been

implicated, perhaps because of the higher concentrations injected.⁵²

Stacy et al have proposed that after a needle encounters bone, it may develop a barb on its end. They noted barbs (inward and outward) occurred in 50% of 50 needles evaluated in dental student clinics and in 30% of the needles evaluated in the faculty clinic. They propose neural damage occurs as the needle is withdrawn through the nerve.⁵⁸ This is not unique to trigeminal nerves. Selander et al showed techniques where axillary blocks deliberately elicited paresthesia-caused damage to the perineurium, nerve fibers, and blood vessels. The damage was greatly reduced if a short-beveled needle was used, suggesting the barb in the needle would be less severe.⁵⁹ This has been supported by studies on nerve damage with short-beveled needles.⁶⁰ Harn and Durham reported a 3.6% chance of lingual nerve traumatization with a 15% chance of postinjection complication that could vary in severity.⁴⁹ In a closed-claims analysis, the presence of permanent peripheral nerve injury was described following local anesthetic injection and direct needle trauma.⁶¹

Transsection, Laceration, Rupture, or Avulsion.—

This injury is classified as a Sunderland type 5. The repair requires approximation of the 2 ends and suture. Scar tissue prevents the natural regrowth. Factors that minimize scar development may improve the prognosis. If the injury occurs intraosseously and the neurovascular canal is present, surgical repair should be delayed unless sensory testing over time shows no improvement.

EXAMINATION

Typical workup includes a detailed history, behavioral assessment, and physical examination. Details of the examination have been described by Canavan and Graff-Radford, and will not be reviewed in detail here.⁶² Specific sensory testing may be necessary to monitor progress over time, and responses to von Frey tactile sensory stimuli and Minnesota Thermal Disks, as well as responses to heat, are useful but not essential. Two-point discrimination also may help identify recovery over time. There is a significant false-positive and -negative rate with this test. Taste testing may be performed, but it is difficult to obtain accurate

responses. Unfortunately, all these tests are subjective, requiring a self-response. Operate factors may therefore change responses. Palpation over the injury site may produce a local sensitivity or an evoked sensation in the tongue. This may be due to the presence of axonal sprouts in the scar tissue or central sensitization and mechanical allodynia. These phenomena can be demonstrated where the damage to the nerve leaves it intact (compression).^{13,63} In animal models, spontaneous neural activity and mechanical sensitivity occurred at the same rate despite the type or extent of injury.⁶⁴

THERAPY

Nonsurgical.—Corticosteroids.—Immediately following injury, there may be reason to consider high-dose corticosteroids to reduce immune inflammatory reaction. This has been studied in neuritis involving the seventh nerve (Bell palsy), but not in the developing sensory nerve deficit. It is, however, common for neurosurgeons to prescribe corticosteroids following intracranial surgery. Additionally, in a patient in the prone position, the use of an NMDA antagonist may be beneficial. Further research is needed in this arena.

The therapy for trigeminal dysesthesia is aimed at reducing peripheral nociceptive inputs and simultaneously enhancing central nervous system pain inhibitory systems.⁶⁷

Topical Applications.—The use of topical therapies as not been well studied. There is some evidence that capsaicin applied regularly will result in desensitization and pain relief. The recommended dose is 5 times per day for 5 days, then 3 times per day for 3 weeks. If the patient cannot withstand the burning produced by the application, the addition of topical local anesthetic, either 4% lidocaine or EMLA, is useful. Clonidine can be applied to the hyperalgesic region by placing the proprietary subcutaneous delivery patch where it is most tender. Alternatively, the use of 4% gel can be compounded and delivered over a larger area. For local intraoral application, a neurosensory stent has been created. After an oral impression, an acrylic stent is manufactured to cover the painful site.⁶⁷ The topical agent is applied to the gingival surface and placed intraorally 24 hours per day.

Topical clonazepam (0.5 to 1.0 mg 3 times per day) has been effective at reducing burning oral pain.⁶⁸ Patients were instructed to suck on a tablet for 3 minutes (and then discard it) 3 times per day for at least 10 days. Serum concentrations were minimal (3.3 ng/mL) 1 and 3 hours after application. Woda hypothesized a peripheral not central action at disrupting the neuropathologic mechanism.⁶⁶

Procedures.—Neural blockade is very effective in differentiating sympathetically maintained pain (SMP) from sympathetically independent pain. It may also be effective in controlling SMP if used repetitively. Stellate ganglion blocks, phentolamine infusion, and sphenopalatine blocks have been described as useful in obtaining a chemical sympathetic block. The authors have not had significant benefit using phentolamine infusion in facial pain. This is supported by Scrivani et al who used 30-mg infusions without benefit.⁶⁷

Lidocaine infusion (200 mg over 1 hour) may be used therapeutically in various forms of neuropathic pain.^{68,69} It is suggested that response to intravenous lidocaine may predict who responds to the lidocaine analog, mexiletine. Sinnott et al used an animal model to demonstrate that minimal lidocaine concentration (2.1 mg/mL) is needed to abolish allodynia.⁷⁰ They also describe a ceiling effect. Many animals with experimentally induced allodynia did not obtain persistent relief. They suggest separate physiological mechanisms, with differing pharmacologies, may account for variability, and they suggest there are different aspects of neuropathic pain.

Pharmacology.—**TRICYCLIC ANTIDEPRESSANTS.**—It is well documented that tricyclic antidepressants are effective in many pain problems. Solberg and Graff-Radford have studied the response of amitriptyline in traumatic neuralgia. It is noted that the effective range is 10 to 150 mg per day usually taken in a single dose at bedtime.³² Many antidepressants may be used.

MEMBRANE STABILIZERS.—These medications include the antiepileptic agents, lidocaine derivatives, and some muscle relaxants. They have been classically used in intermittent, sharp, electriclike pains. The newer generation of medications that affect γ -aminobutyric acid appears to be effective in con-

tinuous as well as intermittent pain. These include gabapentin, topiramate, and zonisamide.⁶⁷

Behavioral Strategies.—Before beginning therapy, it is common to perform a behavioral assessment with appropriate testing. Following the behavioral evaluation, management is directed at the factors that may affect treatment and determine the most appropriate interventions. Consideration should be given to the following factors: (1) behavioral or operant, (2) emotional, (3) characterological, (4) cognitive, (5) side effects, (6) medication use, and (7) compliance. Therapy such as cognitive and behavioral management techniques, relaxation, and biofeedback with psychotherapeutic and psychopharmacological interventions may be useful.

Surgical.—Microsurgical techniques for nerve repair have been used for many years. There is little standardized manner in assessing outcome, and the numbers studied are very limited, especially when it comes to the lingual nerve. Repair may entail decompression, direct suture, or grafting. As mentioned previously, using an animal model, Robinson suggested excision and apposition with suture was the most effective repair procedure.⁷¹ They have prospectively assessed 53 patients, and although on an individual basis the outcome is variable, there was some benefit in most patients. Light touch improved from 0% to 51%, pinprick response increased from 34% to 77%.^{5,71} About half of these patients had some continuity in the nerve despite the lack of recovery. The nerves were found trapped in dense scar tissue and often a neuroma was evident. Unfortunately, there was little benefit in the dysesthesia or pain that was present. Gregg has reported a 49% reduction in pain in 31 patients following lingual nerve repair.⁷² Pogral and Kaban also reported good pain reduction following repair.² In 1996, Robinson reported 13 patients in whom the lingual nerve was repaired by apposition and epineural suture. The mean duration postinjury was 16 months.⁷¹ There was some sensory restoration and some taste recovery. Patients' subjective assessment of the value of the surgery ranged from 0 to 10 with a median of 7.

Microvascular decompression is very effective in the compression neuropathies. Trigeminal neuralgia

and other intermittent neuralgias respond exceptionally well. In continuous neuropathy, surgical intervention that may create further injury is not advised.

Chemosensory regeneration or the improvement in taste is incomplete following nerve repair. Zuniga et al demonstrated 50% of 12 patients improved after microneurosurgical repair. They also suggested an increase in fungiform papillae and pores over time.²¹ Taste is usually compensated for over time, and no known treatment is helpful.

CONCLUSION

Although quite rare, lingual nerve injury results in a variety of clinical presentations. Many patients have no problem adjusting to the change, but in some, it may be irritating. There does not seem to be any specific change unique to the lingual nerve other than the anatomical locations. Research into the genetic and other susceptibility issues will be enlightening and may provide us with the ability to better prevent these deficits.

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