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# MANDIBULAR NERVE INJURY AFTER DENTAL IMPLANT SURGERY: MANAGEMENT AND PROTOCOL

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**ABSTRACT** Sensory impairment of the skin and mucosa innervated by branches of the trigeminal nerve is a potential concern in association with dental implant surgery. The most common nerve traumatized in implant dentistry is the inferior alveolar nerve (IAN) and its mental branch. Other nerves at risk include the lingual nerve, long buccal nerve, and the infraorbital nerve because of the anatomical location of these structures.

More than 70% of dentists have experienced patients with postoperative paresthesia/dysesthesia or anesthesia as a result of dental procedures. The most common nerve affected is the mandibular nerve, which may be injured during either implant surgery or bone grafting procedures. However, no organized protocol directed specifically for general dentists, or for the vast majority of specialists, has been published.

The protocol presented in this article is divided into 5 periods: during surgery (1) nerve injury is suspectedor (2) known nerve transection, postoperative period (3) 1 week and (4) 12 weeks ( 5).

The appropriate treatment (pharmacology, monitoring, etc.) for each period is suggested, including the referral to a neurologist when appropriate.

**KEYWORDS :** Nerve injury, Paresthesia, Nerve injury protocol

Neurosensory impairment may occur during all phases of dental implant surgery, including anesthetic administration, incisions, soft tissue reflection, osteotomy preparation, bone augmentation, implant placement, suturing, or soft tissue swelling after surgery. The reported occurrence of such nerve injuries after dental implant procedures is highly variable (0%–44%).[1–5] When a nerve injury occurs, the dentist should be able to recognize the type and extent of injury and provide the most appropriate postoperative care. Traumatic and iatrogenic nerve complications may involve total or partial nerve resection, crushing, stretching, or entrapment injuries. The resulting sensory deficits may range from a nonpainful minor loss of sensation to a permanent and severe debilitating pain dysfunction. At present, no standardized protocol exists for the dentist in the management of nerve injuries after implant surgery. However, surveys at the Misch International Implant Institute (Beverly Hills, MI) indicate that 73% of dentists have encountered situations of neurosensory impairment within their practices. The purpose of this article is to present guidelines for the dentist in the diagnosis and possible management of mandibular nerve neurosensory deficits (including referral) after dental implant surgery that is dependent on the history, type, and nature of the injury.

### Peripheral Nerve Anatomy/Histology

The trigeminal nerve is the largest of the cranial nerves and has 3 major divisions:
the ophthalmic nerve (V1), the maxillary nerve (V2), and the mandibular nerve (V3).

The mandibular nerve is a peripheral nerve, which is the largest of the trigeminal branches, and, as previously stated, is the most common branch that is involved with neurosensory disturbances after dental implant surgery. This division carries sensory information from the lower lip, chin, lower teeth (and associated soft tissues), the mandibular bone, and parts of the external ear.

The mandibular nerve also contains motor fibers for supply of the muscles of mastication, tensor tympani muscles of the ear, and tensor veli palatine muscles of the soft palate.

However, these motor branches and many of the sensory fibers to the external ear are not injured during implant surgery, because the motor fibers are separated from V3 before its exit from the foramen ovale of the skull, and many of the sensory fibers enter the nerve above the lingula of the ramus.

A V3 injury in implant surgery usually occurs after the nerve enters the lingula of the mandibular ramus and anywhere along its pathway in the body and/or its exit from the mental foramen. The histology of the mandibular nerve consists of connective tissue and neural components. The smallest functional unit in the trigeminal nerve is the nerve fiber. These nerve fibers can be either myelinated or unmyelinated. Myelinated nerve fibers, the most abundant, consist of a single axon that is encased individually by a single Schwann cell.

The individual nerve fibers and Schwann cells are surrounded by a protective endoneurial connective tissue layer (endoneurium) that is made up of a basal lamina, collagen fibers, and endoneurial capillaries[6] The individual nerve fibers of the trigeminal nerve are situated in multiple groups termed fascicles. The IAN has been shown to contain between 7000 and 12,000 axons in various fascicular arrangements.[7] These nerves are classified as polyfascicular, which are characterized by an abundant amount of interfascicular connective tissue containing 10 fascicles. As a polyfascicular nerve, the IAN withstands stretch injuries better than monofascicular and oligofascicular nerves. The number of fascicles varies along the nerve trunk. There are approximately 18 to 21 fascicles in the third molar region decreasing to 12 fascicles in the mental foramen area.8

The IAN has more fascicles than the lingual nerve; therefore, the IAN has greater ability to regain sensation after injury via compensatory innervations from uninjured fascicles.9 Surrounding the bundles of fascicles is a thin, dense, multilayered connective tissue sheath called the perineurium. The perineurium maintains intrafascicular pressure and acts as a diffusion barrier in the protection of the individual fibers. Encompassing the fascicles are 2 types of connective tissue, the inner and outer epineurium. The inner epineurium is

composed of loose connective tissue with longitudinal collagen bundles, which serves to protect the nerve against compressive and stretching forces. The main function of the epineurium is to protect the nerve components and maintain the structural continuity of the nerve.[7] Continuous with the outer epineurium is the mesoneurium, which is the outside connective tissue sheath that suspends the nerve truck within the soft tissue and contains the blood supply to the nerve.6 If any of these extraneural tissues (epineurium, perineurium, endoneurium, or mesoneurium) are injured, impaired neural transmission of the individual nerve fibers may result in a sensory disturbance.[10] The neurosensory impairment is dependent on the extent of damage to the individual tissue type. A main concern related to injury of the IAN is the altered sensory functions (i.e., touch, pressure, temperature, or pain) after implant-related surgeries. Loss of sensation of the cheek and/or mandibular lip may lead to traumatic injury to the soft tissues during chewing and mastication and may affect the ability to drink. Significant pain may also be associated with these sensory functions, which may be debilitating to the patient.

### Terminology of Sensory Disturbances

The literature related to peripheral nerve injuries is abundant. However, there exists great variation in the nomenclature used to describe the clinical signs and symptoms. Several common terms are used, often with overlapping meanings. The most commonly used term to describe an altered sensation is paresthesia. For years, paresthesia has been used to describe any altered sensation including pain, numbness, tingling, aching, warmth, cold, and burning.[11] Recently, the Association for the Study of Pain has standardized a nomenclature system that defines the most frequently used neurosensory descriptive terms.[12] There now exists 3 distinct categories with related subcategories describing neurosensory deficits. The most significant change is paresthesia. It is limited to an altered sensation that is not unpleasant. Dysesthesia is defined as any altered sensation that is unpleasant.

Anesthesia is the total loss of feeling or sensation. These 3 main categories are used to describe, diagnose, and treat (including referral) the nerve injury in the protocol suggested in this article. Table 1 describes the most commonly used terms for neurosensory deficits (Table 1).

### Neurosurgery Classifications

For years, there have been 2 widely accepted classifications of nerve injuries. In 1943, Seddon postulated a 3-stage classification, which was later modified by Sunderland in 1951 into 5 subclassifications[13,14] (Table 2). These injury classifications depend on the severity of axonal damage. Neuropraxia or first-degree injury is distinguished by a conduction block with no degeneration of the axon or visible effects of the epineurium. Axonotmesis is characterized by axonal injury with resultant degeneration or regeneration. Neurotmesis is the most severe injury type caused by severe traction or complete transection of the nerve and the nerve sheath. Although partial recovery from this trauma may occur, complete recovery is impossible.

The nerve injury classifications of Seddon and Sunderland are useful for neurosurgery to determine the type of treatment based on the injury. However, predicting the amount of neural damage and resulting neurosensory deficit is usually difficult, unless the operator is aware that the nerve has been separated. The manner in which the neural damage manifests itself postoperatively and whether resolution follows can be perplexing. Therefore, these classifications are unclear for the dentist to determine an ideal time period to provide initial treatment or to refer a patient for further therapy.

### Related Host and Local Factors

Many factors, both local and hostrelated, will determine the neurologic response to an injury. The type of injury is the most significant local factor relating to the neurologic response. In general, injuries that occur at the proximal site of the peripheral nerve are more significant (i.e., ramus region) than those that occur at distal sites (i.e., the mental foramen area). The more proximal the nerve injury site, the higher the risk of trigeminal ganglion cell damage and the initiation of retrograde differentiation effects into the central nervous system.[15] Less traumatic injuries often are associated with early paresthesia or dysesthesias When a nerve is stretched or compressed, the perineurium will protect the nerve fibers within the fascicles. With greater tension, the fascicles will begin to elongate, thus increasing the intrafascicular pressure. Further pressure will lead to axon damage, and if elongation is 30%, structural failure with possible severance may occur.[16] Partial or incomplete nerve injuries will have different responses than complete nerve lacerations or resections. In contrast, more severe injuries are initially anesthetic lesions with poor orofacial function and associated referred, radiating forms of paresthesia. Although they may not be painful initially, they may eventually lead to formation of dysfunctional chronic neuroma formation.[17,18] The most significant host factors related to implant dentistry are the patients' age and gender. It is well documented for all types of nerve injuries that both females and increasing age are at greater risk of neurosensory deficits. [19,20] In older individuals, cell body regeneration has been shown to be slower and less dramatic than in younger individuals.

### Pharmacologic Therapy

The pharmacologic therapies for acute nerve injuries include the use of corticosteroids and nonsteroidal anti-inflammatory drugs (NSAIDs).The use of adrenocorticosteroids (e.g., dexamethasone) has been shown to minimize neuropathy after nerve injuries if administered in high doses within 1 week of injury.[21,22] In addition, adrenocorticosteroids have been shown to inhibit axon sprouting centrally and ectopic discharges from injured axons and prevention of neuroma formation.[23,24] Dexamethasone (8 –12 mg) is specifically recommended because of its greater anti-inflammatory effects in comparison with other corticosteroids.[25,26] It has been advocated that a tapering dose of a corticosteroid for 5 to 7 days be prescribed after trigeminal nerve injury[27] Further pharmacologic therapy includes the use of NSAIDs. NSAIDs have been shown to be excellent inhibitors of prostaglandin synthesis from damaged peripheral nerve endings.[28] Prostaglandins re-leased as a result of peripheral nerve damage sensitize peripheral nociceptor fibers and central spinal tract neurons.29 Accordingly, maintaining therapeutic blood levels of NSAIDs as an adjunct to the corticosteroid usage for 1 to 3 weeks after the injury is highly beneficial for the acute and intermediate stages of trigeminal nerve recovery.30 Because any altered sensation may be due to an inflammatory reaction, a postoperative course of steroid treatment followed by a high dose of nonsteroidal anti-inflammatory medication (such as ibuprofen 600 – 800 mg 3 times per day for 3 weeks) is used as soon as possible after any nerve injury. If necessary at 2 to 3 weeks after the injury on the basis of a repeated neurosensory examination, the clinician can prescribe an additional 3 weeks of NSAID treatment if no signs of gastric disturbances are present. Additional pharmacologic agents that have been advocated include antidepressants, anticonvulsants, antisympathetic agents, and topical medications. Precaution should be taken with these types of pharmacologic treatments because they should be prescribed and managed by a clinician familiar with the side effects of these drugs and experienced in treatment of nerve injuries.

**Table 1. Description of Neurosensory Impairment Deficits**[13]

| | |
|---|---|
| Anesthesia | Total loss of feeling or sensation |
| Dysesthesia | Abnormal sensation which is unpleasant |
| Allodynia | Pain due to a stimulus that does not normally provoke pain |
| Hyperpathia | Abnormally painful reaction to a stimulus |
| Causalgia | Persistent burning pain |
| Anesthetic dolorosa | Pain in an area that is anesthetic |
| Paresthesia | Abnormal sensation that is not unpleasant |
| Hypoesthesia | Decreased sensitivity to stimulation |
| Hyperesthesia | Increased sensitivity to stimulation |
| Hypoalgesia | Decreased response to a stimulus that is normally painful |
| Hyperalgesia | Increased response to a stimulus that is normally painful |
| Synesthesia | Sensation felt in an area when another area is stimulated |

## INITIAL MANAGEMENT OF NEUROSENSORY IMPAIRMENT

Various surgical and pharmacologictreatments have been advocated in the literature for nerve injury with varying degrees of success.[29] The management of the neurosensory deficits presented in this article (Table 3) should first include recognition and documentation of the type of injury and associated signs and symptoms. Initially, physiologic and pharmacologic nonsurgical therapies are indicated followed by surgical evaluation and/or treatment. If during surgery known or observed trauma (including traction or compression of the nerve trunk) has occurred, the topical application of dexamethasone is suggested. One to two milliliters of the intravenous form of dexamethasone (4 mg/mL) may be topically applied for 1 to 2 minutes.[31] The direct application of adrenocorticosteroids will reduce neural inflammation and reduce compression from swelling, which may enhance recovery from neurosensory deficits. No morbidity has been associated with topical steroid application at the nerve injury site; however, significant improvement in postsurgery recovery has been observed. This is followed by a 6-day regimen of oral dexamethasone (4 mg 2 tablets AM. for 3 days, 1 tablets AM. for 3 days). If known nerve trunk transection is clinically observed during the surgery, immediate referral to a nerve repair specialist is highly recommended. The most important physiologic therapy at the time of surgery includes removal or repositioning of any irritant (implant and bone screw) in close approximation to the neurovascular bundle. A radiograph or computed tomography scan immediately after implant placement is warranted to insure that the nerve is not violated. If a postoperative radiograph indicates that the implant may encroach on the IAN, it may be removed and dexamethasone introduced into the osteotomy site. A shorter length implant may be placed in the site in a more ideal location.[31] No bone grafting materials should be placed in the osteotomy site, because it may invade the mandibular canal and interfere with nerve repair. Cryotherapy should be applied extraorally to most implant and bone graft sites, but especially when nerve injury is suspected. The paraneural tissues should have ice applied intensely for the first 24 **hours postoperatively and then episodically for the first week. Cryotherapy has** been shown to minimize secondary nerve injury from edema-induced compression, decrease the metabolic degeneration rate of trigeminal ganglion cells from undergoing degeneration, and slow potential neuroma formation.[32] Ice, when applied to the tissues, has been shown to significantly improve postsurgical recovery.

**Table 2. Neurosensory Impairment Classification and Injury Response**[14,15]

| Sr no | Type | Description | Cause | Response | Recovery |
|---|---|---|---|---|---|
| 1 | Neurapraxia | Temporary interruption of nerve transmission "conduction block | Nerve compression, edema, hematoma, minor stretching, thermal | Neuritis, Paresthesia | Fast (Few days to 12 weeks) |
| 2 | Axonotmesis | Endoneurium, perineurium, and epineurium remain intact. Some axon degeneration may occur. | Nerve compression, traction, hematoma, partial crush,edema, stretching | Paresthesia episodic dysesthesia | Variable usually (2–4 mo) but may be incomplete |
| 3 | | Disruption of axon and connective tissue (endoneurium) causing Disorganized regeneration. Walter degeneration occurs | Crush, puncture, severe hematoma, stretching | Paresthesia, dysesthesia | Slow (months, incomplete |
| 4 | | Damage involves entire fascicle. Axonal, endoneurium, and perineurium changesoccur. The epineurium is intact. Scar tissue formation. | Full crush, extreme stretching, high thermal, direct chemical trauma | Hypoesthesia, dysesthesia, neuroma formation | None |
| 5 | Neurotmesis | Complete transaction or tear of the nerve with amputation neuroma forming at injury site | Complete transaction | Anesthetic, intractable pain, neuroma formation | None |

## POSTOPERATIVE MANAGEMENT OF NEUROSENSORY DEFICITS

### Neurosensory Testing

When the dentist discovers that aneurosensory deficit has occurred (often at the suture removal), a comprehensive sensory evaluation should be completed. The purpose of this initial examination is to ascertain whether a sensory deficit exists, to define and quantify the extent of nerve injury, record a baseline for recovery, and to determine whether referral for microneurosurgery is indicated.[10] There are several accepted protocols for neurosensory testing. Ideally, a range of tests are performed ranging from light mechanical to noxious stimuli. The authors have advocated 2 categories of clinical neurosensory testing, nociceptive and mechanoreceptive. Each test is specific for various neural receptors and axons. The responses are recorded and quantitatively compared with responses from contralateral uninjured tissues (control). The following are the more common tests suggested for the neurosensory evaluation.

### Nociceptive

1. "Pin-Prick" test: A 27-gauge needle is used to test for pressure detectionand anesthesia/paresthesia/ dysesthesia. A cosmetic pencil is used for mapping, and photographs are

used to evaluate the recovery period .

2. Temperature sensitivity tests: Ice chips or ethyl chloride spray and a heated mirror handle (warmed to 43°) are used to determine the patient's ability to feel cold and hot. Alternatively, test tubes may be filled with hot (43°C) water or cold water .

**Mechanoceptive tests**

1. Static touch detection tests: A cotton tip applicator is used to determine sensation .

2. Direction of movement test: The sensory modalities of mandibular nerve fibers are touch and vibration. A soft brush is used (with the patient's eyes closed) to determine the patient's ability to detect both sensation and direction of movement

3. Two-point discrimination test: With the patients eyes closed, thepatient's ability to discriminate varying distances between 2 points is determined. A caliper with the ability to vary the distance between 2 points can be used. The normal distance at which most patients can discriminate 2 separate points is 6 mm .[33]If the initial examination is within 1 week of the surgery, a course of steroids (decadron) is prescribed followed by 3 weeks of high-dose NSAIDs (600–800 mg ibuprofen). If paresthesia is reported after a 2-week period, only high doses of NSAIDs should be prescribed (600–800 mg ibuprofen three times a day for 3 weeks). If necessary, an additional 3 weeks of NSAIDs may be prescribed. If paresthesia is present, the neurosensory examination is repeated every 2 to 3 weeks. The patient should be periodically reexamined to evaluate whether nerve repair is occurring, signified by reduced symptoms and less soft tissue area involvement. Neurosensory improvement most often occurs by 2 to 3 months. If significant sensation has not improved by 3 to 4 months after the surgery, the prognosis typically is poor.

**Table 3. Neurosensory Deficit Treatment Algorithms**

| Time | Documentation | Pharmacologic Intervention | Treatment | Referral to Nerve Specialist |
|---|---|---|---|---|
| During surgery<br>Nerve injury suggested (i.e., compression) | Radiographic evaluation | Dexamethasone (4 mg/mL) topically 1 mL<br>Implant removal or reposition if impingement within mandibular canal | Dexamethasone 4 mg 2 tablets AM for 3 d<br>1 tablet AM for 3 d<br>Cryotherapy (1 wk ) | No |
| Nerve transection (observed) | | Dexamethasone (4 mg/mL) topically 1 mL<br>Implant removal or reposition if impingement within mandibular canal | Dexamethasone 4 mg 2 tablets AM for 3 d<br>1 tablet AM for 3 d<br>Cryotherapy (1 wk) | Yes |
| 1 wk Postoperatively | Neurosensory examination (testing should be continued every 2 wk) | High-dose NSAIDs (600–800 mg ibuprofen three times a day) | Palliative | Palliative Yes, if Dysesthesia or complete anesthesia |
| 12 wk Postoperatively | Neurosensory examination | | Palliative | Yes, if no sign of improvement |

**Referral of Neurosensory Deficits**

In certain situations, patients need to be referred to a practitioner experienced in nerve injury assessment and repair (Table 3). As previously mentioned, if known transection of the nerve occurs during surgery, dexamethasone should be applied and an immediate referral to a specialist in microsurgical repair should be made after surgery. Similarly, if the patient has dysesthesia or complete anesthesia at the initial examination after surgery, the patient should be referred to a nerve specialist. Prompt surgical intervention may allow for the best chance of neurosensory recovery. The decision postoperatively to refer should be based on the patient's signs and symptoms and the type of injury. A paresthesia should be given sufficient time for neurosensory recovery. However, referral to a nerve injury specialist is suggested after 3 months if the paresthesia has not improved during this time frame. Additional physiologic therapies have shown successful results in the treatment of nerve impairments and include transcutaneous electric nerve stimulation,[34] acupuncture,[35] and low level laser therapy.[36] It is suggested these physiologic therapies be used as indicated by a nerve specialist.

Various techniques to surgically treat nerve impairments have been advocated with varying results. This includes decompression, direct anastomosis[37] autogenous nerve grafts,[38] and alloplastic grafts.[39] Successful surgical intervention, when indicated, is generally agreed to be most predictable if performed before the onset of Wallerian degeneration (approximately 3 months).27 This early, aggressive treatment may prevent transition to chronic refractory neuropathies.[40,41]

## SUMMARY

The suggested treatment of mandibular nerve neurosensory deficit is summarized in Table 3. The protocol during surgery is divided into 2 aspects: nerve injury suspected (most often after a radiograph is made) or known nerve transection during surgery. Corticosteroids, NSAIDs, and cryotherapy are prescribed after surgery. The postoperative protocol is divided into the 1 week, initial treatment, and the 12-week period. During this timeframe, a paresthesia is documented and mapped every 2 to 3 weeks to monitor the condition. NSAIDs are prescribed for up to 3 weeks after the initial corticosteroid therapy. If dysesthesia or anesthesia is found at the initial postoperative appointment, a referral to a nerve specialist is suggested. Referral is also suggested to a specialist in nerve damage after 3 months if the paresthesia is not improved.

## CONCLUSION

With the popularity of dental implants and associated bone grafting, peripheral trigeminal nerve branch impairments may become more frequent. It is not expected that the practitioner make a definite neurosensory diagnosis. However, the clinician must be sufficiently knowledgeable in the causes, prevention, and treatment of such injuries to make an appropriate diagnosis and treatment and timely referral when necessary. Neurosensory changes in the orofacial region on rare occasions may be devastating. A protocol has been presented, which emphasizes early treatment with a wide range of modalities and guidelines for treatment—based on type and degree of neurosensory impairment.

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