Avoiding Mandibular Nerve Impairment, Part 3

Management of Neurosensory Impairments After Dental Implant Surgery

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A major concern in association with dental implant surgery is sensory impairment of the skin and mucosa innervated by branches of the trigeminal nerve. The reported incidence of such nerve injuries following dental implant procedures is highly variable (zero percent to 44%). It is vital that the dental implant surgeon be able to recognize the type and extent of any nerve injury, and be able to provide the most appropriate postoperative care. Iatrogenic and traumatic nerve complications may involve total or partial nerve resection, stretching, thermal, crushing, or entrapment injuries. The resulting sensory deficits can include a minor, nonpainful loss of sensation (hypoesthesia), or a more permanent and severe debilitating pain dysfunction (dysesthesia). The authors have developed postoperative guidelines for diagnosis and possible management of neurosensory deficits following dental implant surgery that is dependent on the history, type, and nature of the injury.

TRIGEMINAL NERVE ANATOMY

The mandibular nerve is the largest of the trigeminal branches and is the most common branch involved with neurosensory disturbances following dental implant surgery. The inferior alveolar branch carries sensory information from the mandibular teeth, periodontium, buccal mucosa, and the lower lip.

As the inferior alveolar nerve (IAN) runs downward and forward within the bony mandibular canal, a division occurs leading to the dental (alveolar) and the mental nerve. To understand the etiology of neurosensory impairments, a thorough knowledge of these anatomic structures is needed.

Histologically, this IAN consists of connective tissue and neural components in which the smallest functional unit is the individual nerve fiber. The IAN fibers may be either myelinated or unmyelinated. The myelinated nerve fibers are the most abundant, which consist of a single axon encased individually by a single Schwann cell. The individual nerve fibers and Schwann cells are surrounded by the endoneurium, which acts as a protective cushion made up of a basal lamina, collagen fibers, and endoneurial capillaries.

The individual nerve fibers of the IAN are situated in fascicles, which are bundles or groups of nerve fibers. The IAN is classified as polyfascicular, which is characteristic of more than 10 fascicles surrounded by an abundance of interfascicular connective tissue. Within the fascicles, there exist approximately 7,000 to 12,000 axons in various fascicular arrangements. The number of fascicles varies along the intramandibular course of the IAN, as there exists...
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approximately 18 to 21 fascicles in the third molar region, decreasing to 12 fascicles in the mental foramen area.3 Because of the polyfascicular nature of the IAN, there exists a greater ability to regain sensation after injury via compensatory innervation from the uninjured fascicles.

Surrounding the polyfascicular makeup of this nerve is the perineurium, which consists of dense, multilayered connective tissue. The perineurium’s function is to maintain intrafascicular pressure, acting as a diffusion barrier to protect the individual nerve fibers. There are 2 types of connective tissue, the inner and outer epineurium, which surround the fascicles. The inner epineurium is composed of loose connective tissue with longitudinal collagen bundles. This tissue protects the nerve fibers against compressive and stretching forces, thus maintaining the structural continuity of the nerve. The outer epineurium is continuous with the meso-neurium, which is the outer loose areolar tissue that suspends the nerve trunk within the soft tissue and contains the blood supply to the nerve. The mesoneurium allows the nerve to have longitudinal movement within the surrounding tissue (Figure 1).

In the event that any of these extraneural tissues (epineurium, perineurium, endoneurium, or mesoneurium) are injured or traumatized, impaired neural transmission may result in a sensory disturbance. The resultant neurosensory impairment is dependent on the varying functional units of the individual fiber type involved. A-alpha fibers are the largest fibers that mediate position and fine touch by way of muscle spindle afferents and skeletal muscle efferents. The A-beta fibers are mainly proprioception in nature and A-delta mediate initial pain impulses along with temperature information. The C-fibers are unmyelinated and slow conducting, which allows the perception of pain and temperature.4

Neurosensory Deficit Classification

When describing the histologic changes that occur from nerve injuries, there exist 2 widely accepted classifications of nerve injuries. In 1943, Seddon5 postulated a 3-stage classification, which was later reclassified by Sunderland6 in 1951 into 5 different subclassifications. These nerve injury classifications are described by the resultant morphophysiologic type of injury, which is based on the time course and amount of sensory recovery (Table 1).

Neuropraxia, or first-degree injury, is characterized by a conduction block, with no degeneration of the axon or visible damage of the epineurium. Usually, this type of injury is consistent with stretching or manipulation (reflection of tissue) of the nerve fibers, which results in injury to the endoneurial

### Table 1. Neurosensory Impairment Classification and Injury Response5,6

<table>
<thead>
<tr>
<th>Sunderland</th>
<th>Seddon</th>
<th>Description</th>
<th>Causes</th>
<th>Responses</th>
<th>Recovery Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Neurapraxia</td>
<td>Temporary interruption of nerve transmission “conduction block.”</td>
<td>Nerve Compression Edema</td>
<td>* Neuritis</td>
<td>Complete (Fast— days to weeks)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hematoma Minor Stretching Thermal</td>
<td>* Paresthesia</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Axonotmesis</td>
<td>Endoneurium, perineurium, and epineurium remain intact. Some axon degeneration may occur.</td>
<td>Nerve Compression Traction Hematoma Partial Crush Edema Stretching</td>
<td>* Paresthesia</td>
<td>Complete (Slow— weeks)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Episodic Dyesthesia</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Disruption of axon and connective tissue (endoneurium) causing disorganized regeneration.</td>
<td>Crush Puncture Severe Hematoma Stretching</td>
<td></td>
<td>* Paresthesia</td>
<td>Variable (Slow— weeks to months)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Dyesthesia</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Damage involves entire fascicle. Axonal, endoneurium, and perineurium changes occur. The epineurium is intact. Scar tissue formation.</td>
<td>Full Crush Extreme Stretching High Thermal Direct Chemical Trauma</td>
<td></td>
<td>* Hypoesthesia</td>
<td>Unlikely</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Dyesthesia</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Neurotmesis</td>
<td>Complete transaction or tear of the nerve with amputation neuroma forming at injury site.</td>
<td>Complete Transection (Overpreparation with implant drill)</td>
<td>* Anesthesia</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Intractable Pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Neuroma Formation</td>
<td></td>
</tr>
</tbody>
</table>
capillaries. The degree of trauma to the endoneurial capillaries will determine the magnitude of intrafascicular edema, which results in various degrees of conduction block. Usually, resolution of sensation and function will occur within hours to weeks.5,6

Axonotmesis (second-, third-, or fourth-degree injuries) consist of degeneration or regeneration axonal injuries. The varying injury classification depends on the severity of axonal damage. This type of injury involves the endoneurium, with minimal disruption to the perineurium and epineurium. The most common types of injuries are traction, stretching, and compression, which can lead to severe ischemia, intrafascicular edema, or demyelination of the nerve fibers. Initially, complete anesthesia is most common, which is followed by paresthesia as recovery begins. Improvement of the related sensory deficits occurs within approximately 2 to 4 months with usual complete recovery within 12 months. However, in some cases, painful dysesthesias are possible with resulting neuroma formation.5,6

Neurotmesis (fifth-degree injuries) is the most severe type of injury, which results from severe traction, compression, or complete transection injuries. Initially, patients exhibit anesthesia, followed by paresthesia with possible dysesthesias. A very low probability of neurosensory recovery exists with immediate referral for a neurosurgical evaluation recommended.5,6

Classification of Sensory Symptoms
The literature involving peripheral nerve injuries is vast with a significant variation in the nomenclature used to describe the associated clinical signs and symptoms. Table 2 describes the most commonly used nomenclature in describing these defects in medicine today.7

Physiologic Response To Nerve Injury
There exist many local and host related factors, which determine the neurologic response to a nerve injury. A patient’s age and gender are the most significant host factors related to implant-related nerve issues. Studies have shown, for most types of nerve injuries, females and increasing age are at greater risk of neurosensory deficits.5 Older individuals exhibit slower and less dramatic cell body regeneration in comparison to younger individuals. The type of injury is the most significant local factor relating to the neurologic response after trauma. Injuries that occur at the proximal site of the peripheral nerve are usually more significant in comparison to distal sites.9

When complete transection of a nerve occurs, within 96 hours, the proximal end of the nerve fiber shrinks approximately 20% to 50% in diameter and usually will not recover more than 80% of its original size.10 Shortly thereafter, axonal nerve sprouts will seek and extend out to the degenerating distal branch. Each axon may contain up to 50 collateral sprouts that advance approximately one to 3 mm per day, which eventually attempt to reinnervate the target tissue. If the nerve sprouts are unable to reconnect, then forward progress is stopped and Wallerian degeneration will occur.
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Wallerian degeneration usually results in neuroma (benign growth) formation, which is associated with increased mechanical and chemical sensitivity, resulting in chronic neurosensory deficits. If during surgery, known traction or compression of the nerve trunk has occurred, the topical application of Dexamethasone may be used to minimize deficits. Upon removal of an encroaching implant on the mandibular canal, one to 2 mL of the intravenous form of Dexamethasone (4 mg/mL) is topically applied (Figure 2). This direct steroid application will reduce neural inflammation and may enhance recovery from neurosensory deficits. Studies have shown no morbidity associated with the topical application of glucocorticoids at the injury site and post-surgical recovery has also been shown to improve significantly.

CLINICAL ASSESSMENT

When a neurosensory deficit occurs postoperatively, a comprehensive sensory evaluation must be completed. This initial examination is done to determine whether a sensory deficit exists, to quantify the extent of injury, document a baseline for recovery, and to determine if referral for microneurosurgery is indicated. Table 3 shows the most common diagnostic tests utilized for the neurosensory evaluation (Figure 3).

After confirmation of the neurosensory deficit, the first-line treatment is to include physiologic and pharmacologic therapies. The most important physiological therapy includes removal or repositioning of any irritant (implant, bone screw) in close approximation to the neurovascular bundle. If the implant is repositioned, a shorter implant should be placed, as “backing” the implant out may lead to impingement on the available interocclusal space, leading to compromised aesthetics and retention. Additionally, cryotherapy (ice packs) should be applied to the paraneural tissues intensely for the first 24 hours and then episodically for the first week. Cryotherapy has been shown to be beneficial in minimizing secondary nerve injury from edema-induced compression, decreasing the metabolic degeneration rate of trigeminal ganglion cells from undergoing degeneration, and slowing potential neuroma formation.

Pharmacologically, acute nerve impairment should include the use of corticosteroids and nonsteroidal anti-inflammatory agents. Studies have demonstrated that the use of systemic adrenocorticosteroids (eg, Dexamethasone) have been shown to minimize neuropathic symptoms following nerve trauma if administered in high doses within one week of injury. It has been advocated that a tapering dose of a corticosteroid for 5 to 7 days following trigeminal nerve injury is beneficial. Dexamethasone (~ 8 mg) is specifically recommended because of its greater anti-inflammatory effects in comparison to other corticosteroids such as methylprednisolone or prednisone.

In certain situations, patients may need to be referred in a timely manner to a practitioner experienced in nerve injury assessment and repair. The decision and timing to refer should be based on the patient's symptoms and the type of injury, along with the experience of the implant dentist in treating nerve injuries. Usually, sufficient time is given for neurosensory recovery;
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However, in cases of dysesthesia, anesthesia, or known nerve transection, prompt surgical intervention may allow for the best chance of neurosensory recovery. Early, aggressive treatment has been shown to minimize possible transition to chronic refractory neuropathies (Table 4).16

IN SUMMARY

Peripheral trigeminal nerve branch impairments are becoming more common with the popularity of dental implants and associated bone grafting. Neurosensory changes in the orofacial region can be devastating to the patient and have significant medico-legal implications for the doctor. In parts one and 2 of this article series, possible risk and etiologic factors to prevent nerve impairment in the mandible were discussed. In part 3, a summary of neurosensory impairment classifications has been presented along with a protocol which stresses early physiological and pharmacologic treatment and emphasizes prompt referral based on the type and degree of injury.17

Table 4. Neurosensory Deficit Treatment Algorithms

<table>
<thead>
<tr>
<th>POST-SURGERY</th>
<th>DOCUMENTATION</th>
<th>PHARMACOLOGIC INTERVENTION</th>
<th>TREATMENT</th>
<th>REFERRAL</th>
</tr>
</thead>
</table>
| ~ 48 HOURS         | 3-D radiographic examination (CBCT) Neurosensory examination | *Corticosteroids (Dexamethasone 4 mg) 2 tabs AM for 3 days 1 tab AM for 3 days | Implant evaluation:  
  • Removal or reposition if impingement within the mandibular canal  
  • No Bone Grafting  
  • Cryotherapy (one week) | None, unless unfamiliar with neurosensory testing |
| One Week Post-Op   | Neurosensory examination (Testing should be continued every 2 weeks thereafter) | High dose NSAIDs (600 to 800 mg ibuprofen tid) | Palliative | Refer to microneurosurgeon if:  
  • Known nerve transection  
  • Dysesthesia  
  • Complete anesthesia |
| Eight Weeks Post-Op| Neurosensory examination | NSAIDS (as needed) | Palliative | IF NO SIGN OF IMPROVEMENT:  
  Refer to oral maxillofacial surgeon or microneurosurgeon |

References

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POST EXAMINATION QUESTIONS

1. As the inferior alveolar nerve (IAN) runs downward and forward within the bony mandibular canal, a division occurs leading to the dental (alveolar) and the mental nerve.
   a. True b. False

2. Because of the polyfascicular nature of the inferior alveolar nerve, the ability to regain sensation after injury is much reduced.
   a. True b. False

3. Neuropraxia, or first-degree injury, is characterized by a conduction block, with no degeneration of the axon or visible damage of the epineurium.
   a. True b. False

4. Axonotmesis (second-, third-, or fourth-degree injuries) are the most severe type of injury, which results from severe traction, compression, or complete transection injuries.
   a. True b. False

5. When complete transection of a nerve occurs, within 96 hours, the proximal end of the nerve fiber shrinks approximately 20% to 50% in diameter and usually will not recover more than 80% of its original size.
   a. True b. False

6. After confirmation of the neurosensory deficit, the first line treatment is to include physiologic and pharmacologic therapies.
   a. True b. False
7. Cryotherapy has been shown to be beneficial in minimizing secondary nerve injury from edema-induced compression, decrease the metabolic degeneration rate of trigeminal ganglion cells from undergoing degeneration, and slow potential neuroma formation.
   a. True  b. False

8. Studies have shown that the use of systemic adrenocorticosteroids (eg, dexamethasone) has little or no effect on minimizing neuropathic symptoms following nerve trauma when administered in high doses.
   a. True  b. False
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ANSWER FORM: VOLUME 34 NO. 2 PAGE 120

Please check the correct box for each question below.

1. ☐ a. True ☐ b. False
2. ☐ a. True ☐ b. False
3. ☐ a. True ☐ b. False
4. ☐ a. True ☐ b. False
5. ☐ a. True ☐ b. False
6. ☐ a. True ☐ b. False
7. ☐ a. True ☐ b. False
8. ☐ a. True ☐ b. False

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