Trigeminal Nerve Injuries After Mandibular Implant Placement—Practical Knowledge for Clinicians

Frederick Hegedus, DDS[1]/Robert J. Diecidue, DMD, MD[2]

Endosseous mandibular implant placement can result in injuries to the peripheral branches of the trigeminal nerve even with the most careful preoperative planning and intraoperative technique. In the past, many patients have been discouraged from seeking repair for such injuries because of the unreliability of the techniques for correcting the injury. It is now possible to perform microneurosurgical repair of such injuries. If the repair is done in a timely fashion, sensation can be improved or restored and painful nerve dysesthesia can be relieved. This article reviews the different types of nerve injuries, their symptoms and diagnosis, and provides information for clinicians to manage their implant patients with neurosensory disturbance. Int J Oral Maxillofac Implants 2006;21:111–116

Key words: *dysesthesia, mandibular implants, trigeminal nerve injury*

Implant dentistry is a very dynamic and exciting area of oral treatment. Whether autogenous or allogeneic, the reconstruction of teeth or their supporting structures can be rewarding in terms of restoration or enhancement of oral function and/or esthetics. Improvements in quality of life[1] and long-term maintenance of peri-implant tissue health[2] have also been reported with implant therapy. However, implant dentistry does not guarantee results, nor is it without complications. The risk of nerve injury is an important and inherent complication associated with oral implant placement. It is important to recognize such a risk and be aware of the treatment of such injuries should they occur.

Endosseous mandibular implant placement can result in injuries to the peripheral branches of the trigeminal nerve, even with the most careful preoperative planning and intraoperative technique. Causes of such injuries include compression, stretch, transection, tearing, laceration, or needle penetration of the nerve. Patients with sensory nerve injury may experience unexpected, unpleasant sensations and have difficulty performing common activities with the face and mouth. Such adverse effects can be unacceptable to patients and negatively impact their physiology and psychology. Patients with implantation-related nerve injuries have often been discouraged from seeking repair because of the unreliability of repair techniques. However, it is now possible to predictably perform microneurosurgical repair of such injuries.[3,4] Indeed, in the hands of trained and experienced clinicians, nerve continuity can be reestablished, sensation and motor function can be improved or restored, and painful nerve dysesthesia can be relieved. This is provided that the injury is recognized early and repair is done in a timely fashion. Careful evaluation, early recognition of the symptoms and stage of nerve injury, timely referral (ie, prior to nerve degeneration) and repair, as well as follow-up of the progress of nerve regeneration represent good clinical practice in the management of patients with nerve injuries following implantation. The objective of this article was to review the pathogenesis, symptoms, and manifestations of trigeminal nerve injuries associated with endosseous mandibular implantation. Practical suggestions for the recog-

[1]Resident, Department of Oral and Maxillofacial Surgery, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.
[2]Chair, Department of Oral and Maxillofacial Surgery, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

Correspondence to: Dr Frederick Hegedus, Department of Oral and Maxillofacial Surgery, Thomas Jefferson University Hospital, 909 Walnut Street, 3rd floor, Philadelphia, PA 19107. Fax: +215 923 9189. E-mail: oms.surgery@mail.tju.edu

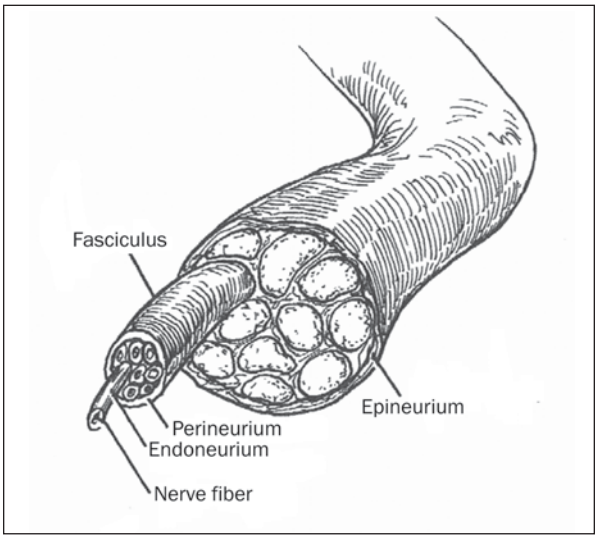

Fig 1 Drawing of a nerve showing the relationship between connective tissue components (epineurium, perineurium, and endoneurium) and axons or nerve fibers within a fasciculus. Reproduced with permission from Day.[5]

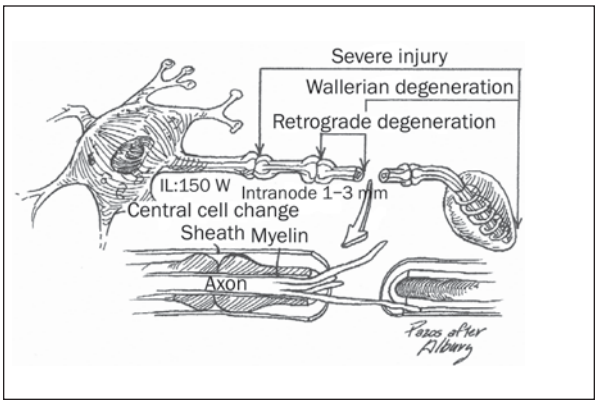

Fig 2 Schematic summary of postinjury events within the nerve. Reproduced with permission from Day.[5]

nition and repair of such injuries and minimization of associated deficits are provided to aid the clinician in the management of compromised patients. With proper care and early intervention by a qualified specialist, partial or complete restoration of nerve function can be achieved.

NEUROLOGIC RESPONSE TO INJURY

Both the anatomic organization of the nerve and the pathologic response to injury play an important role in the eventual regeneration of injured nerves and restoration of nerve function. The trigeminal nerve contains approximately 7,000 to 12,000 nerve fibers arranged in a regular pattern within its trunk (Fig 1).[5] The nerve trunk additionally contains connective tissue components that wrap the microvascular blood supply of the nerve, protect the nerve against mechanical forces such as compression and stretch, and maintain the internal milieu of the nerve.[4,5] Such nerve fibers bundle into fascicles, and nerve fascicules align as organized cords within the nerve trunk. The trigeminal nerve is a polyfascicular nerve; it contains 10 to 25 fascicles within its trunk. This complex anatomic pattern represents the greatest challenge for spontaneous recovery or regeneration following injury, as spaces between misaligned fascicles provide loci for intraneural fibrosis, which disrupts nerve continuity. The goal of microneurosurgical intervention is to align the fascicles as early as possible to prevent fibrosis and allow nerve regeneration and conduction continuity.

The neurologic response to injury is complex and involves proximal and distal changes (Fig 2).[5] The injured axon degenerates distally from the site of injury. This is known as Wallerian degeneration. The myelin sheath surrounding the nerve begins to break down within hours to days of the injury. The Schwann cells that compose the myelin sheath (or those that surround unmyelinated axons) usually survive axonal degeneration and play a key role in providing the axon with metabolites for nerve regeneration.[5] Proliferation of Schwann cells and macrophages provides the impetus for phagocytosis of axonal and myelin sheath debris. This is the first step toward regeneration.

As phagocytosis progresses, an empty endoneural area or tubular canal begins to form wherein Schwann cells align in a longitudinal pattern and lay the groundwork for new nerve fibers. Deregulation of endoneural tubes during this process will lead to twisted or misaligned nerve configurations, referred to as neuromas. During the regenerative process, altered cell body metabolism promotes axonal sprouting proximal to the site of nerve injury. This usually occurs around the second day following injury. The new budding axons begin to advance toward the injury site along the newly formed Schwann cell tubes.

After approximately 2 weeks, the new axons begin to cross the site of injury and try to traverse the empty distal segment, a process referred to as neurotization. Successful neurotization is dependent on the integrity of the endoneural tubes, which begin to gradually decrease in size. They are reduced by 50% within about 3 months. If neurotization does not occur, the diameter of the endoneural tubes can decrease by up to 90% by 12 months. The decrease in diameter is brought about by deposition of collagen and products

Table 1 Terminology of Altered Sensations After Nerve Injury

Terminology	Associated change in sensation
Hyperesthesia	Increased sensitivity to noxious or non-noxious stimulus
Allodynia	Pain resulting from a stimulus that normally doesn't provoke pain
Hyperalgesia	Increased pain response to a stimulus that is normally painful
Paresthesia	Abnormal sensation, provoked or spontaneous, not unpleasant
Dysesthesia	Unpleasant abnormal sensation, spontaneous or evoked
Anesthesia	Absence of perception of noxious or non-noxious stimulus
Hypoesthesia	Decreased perception of stimulation by noxious or non-noxious stimulus

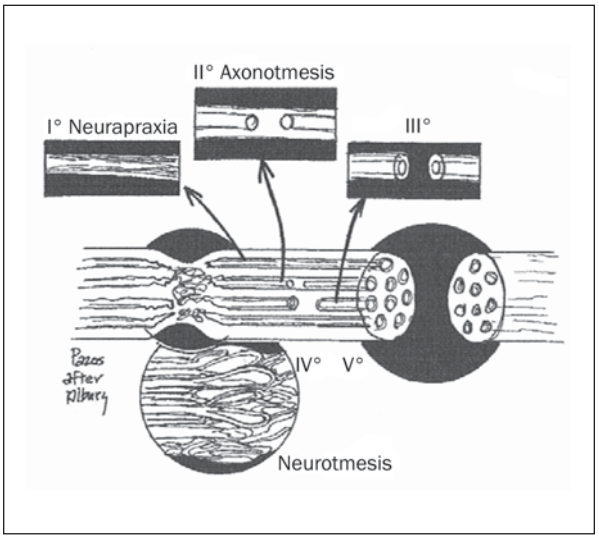

Fig 3 Classification of nerve injuries as described by Seddon and based on the extent of anatomic injury, recovery time, and prognosis. Reproduced with permission from Day.[5]

of cellular proliferation that form a scar tissue. Such scar tissue will inhibit penetration of axonal fibers or neurotization from occurring, thus preventing the recovery of any lost sensation. Should neurotization occur, the newly formed axons will occupy the endoneural tube, often in numbers up to four times in excess of the normal number of axons for that nerve.

With time, however, axonal proliferation slows down, and the regenerated nerve generally contains fewer axons than normal, which explains the rather primitive return of sensation that begins with pain and an increased perception of temperature.[5] Simultaneous with axonal regeneration, cell bodies also grow back, albeit at a rate of 50% to 85% of original numbers—even early repair has not been shown to minimize the almost 50% loss in cyton numbers.[5] Overall, successful regeneration depends on several factors, such Schwann cell proliferation, axon sprouting, preservation of endoneural tubes, survival of cell bodies (ganglions), achievement of fiber continuity, and proper alignment. It is obvious that for regeneration benefits to translate clinically, intervention should occur early to promote these factors. If this does not happen, neuropathic responses consistent with the formation of neuromas, development of collateral nerves, and abnormal sensation of pain will occur.

SYMPTOMS OF NERVE INJURY

Table 1 summarizes the terminology used to describe the various types of altered sensations after nerve injury. In general, symptoms of nerve injury may fall into 1 of the following categories: *(1)* Nonpainful hypoesthesia or anesthesia, representing decreased or lack of pain perception in an environment that is immune to other stimuli; *(2)* Painful hypoesthesia or anesthesia, representing pain associated with lack of perception of sensory stimuli; *(3)* Nonpainful hyperesthesia, representing a condition of intact response to fine static touch but loss of complex sensations (brushing, vibration) and no pain (although tingling or crawling sensations may be present); and *(4)* Painful hyperesthesia, representing pain (as stinging, flashing, burning) associated with low-intensity stimuli.[5,6] Microneurosurgery as a restorative therapy can provide variable success in each of these cases. Most patients with nonpainful anesthesia and hypoesthesia can hope to have restored ability to sense simple stimuli. Patients with nonpainful hyperesthesia may not achieve any improvement (there is anatomic preservation of the nerve but impaired sensory perception). In most other cases, function restoration or improvement may be expected in about 55% to 60% of cases.[5]

TYPES OF NERVE INJURIES

The well-known Seddon's classification describes nerve injuries based on the extent of anatomic injury, recovery time, and prognosis. This classification is helpful to clinicians in diagnosing nerve injuries and determining the prognosis for recovery and the need and timing of surgical intervention. The system recognizes 3 classes of injuries: neurapraxia, axonotmesis, and neurotmesis (Fig 3).[5] These levels of nerve injury may be thought of as increasingly severe perturbations of the nerve.[4]

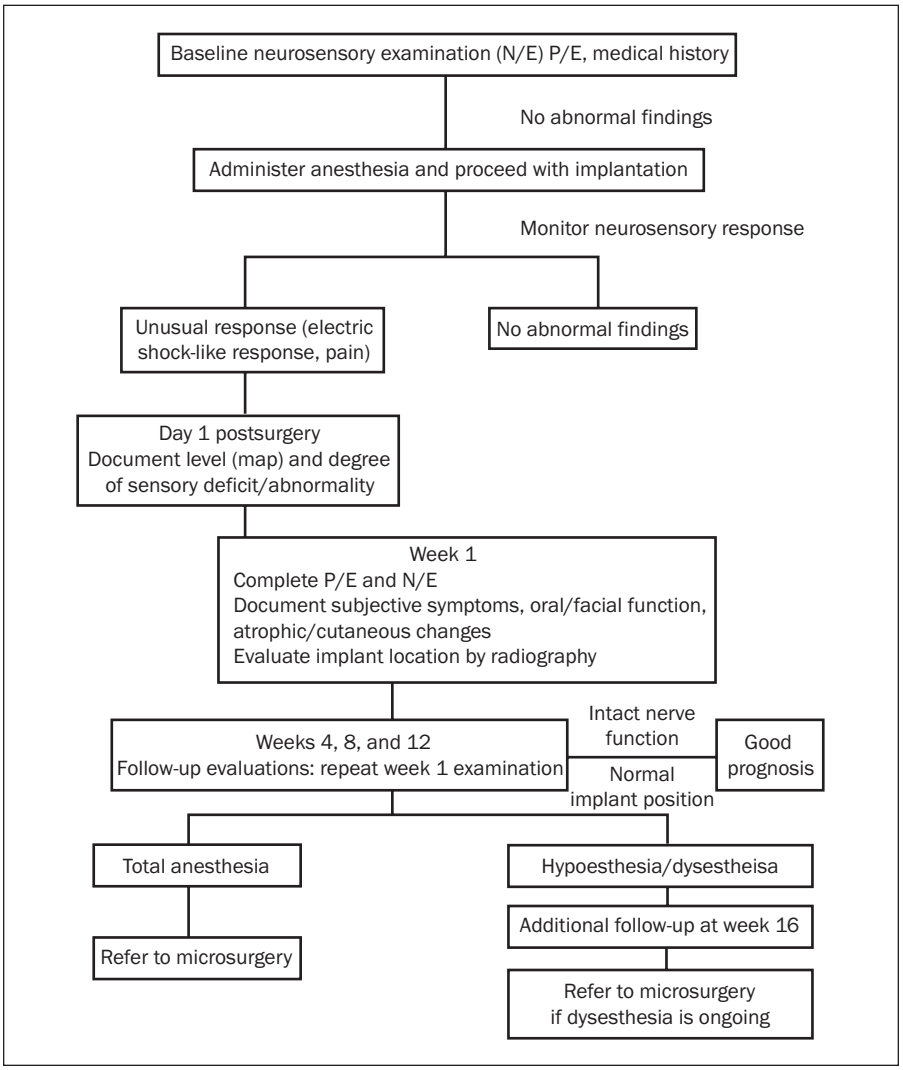

Fig 4 Algorithm of clinical evaluation and follow-up of patients with endosseous mandibular implants.

Neurapraxia (or first-degree injury) results from a mild injury in a temporary conduction block often caused by compression or prolonged traction of the nerve. The nerve axonal pathway remains intact, and injury results in only temporary failure of conduction. A local anesthetic block is an example of neurapraxia. Spontaneous recovery from neurapraxia usually is complete within 4 weeks, and surgery is not necessary.[7] Axonotmesis (or second-degree injury) is a more significant nerve injury, often caused by excessive traction or compression of the nerve. Severe intrafascicular edema, ischemia, and demyelination may occur. There may be loss of continuity of some axons although the general structure of the nerve remains intact. Initial symptoms of returning sensation (eg, itching, tingling) occur 5 to 11 weeks after injury and slowly improve for the next 10 to 12 months. The recovered sensation is often less than normal (hypoesthesia) and may be accompanied by unpleasant abnormal sensation or dysesthesia. Microsurgical reconstruction is typically not necessary, although surgery may be used to relieve persistent pain.[7] Early intervention is important for success. The third and most severe type of nerve injury is neurotmesis and includes so-called third-, fourth-, and fifth-degree injuries. Neurotmesis involves disruption of neural sheath elements, resulting in a block of conduction impulses. Sensory recovery following neurotmesis typically does not occur without intervention.[5] A transection injury needs microneurosurgical intervention for optimal alignment of the nerve sheaths. The prognosis for normal sensory recovery is generally poor and varies with the extent of damage and site (soft tissue versus intracanal) of nerve placement.

CLINICAL EXAMINATION

Pre- and perioperative procedures can help in rapid diagnosis and documentation of injury (Fig 4). Clini-

cians should document any unusual response (eg, unusual pain or an electric shock–like feeling) during administration of the local anesthetic or during implant surgery.[8] If a nerve injury is suspected, the clinician should perform a thorough examination and document the level of neurosensory function as soon as the injury is suspected. This typically occurs the day after surgery. When a clinician suspects a nerve injury in the immediate postoperative course, the patient should be reassured that injury of this sort is not unusual and that it is usually not permanent. He or she should also be informed that continued follow-up is necessary for maximal recovery. Evaluation should include assessment of the patient's subjective symptoms, physical examination, and neurosensory examination to ascertain the area and depth of sensory loss.[5,9]

The first step in the diagnostic process is to determine the type of neurosensory disturbance (Fig 4). Most of the necessary information can be obtained by taking a careful and detailed medical history. During the patient's interview it is important to distinguish between an unpleasant or painful sensory deficit (dysesthesia) and a decreased or abnormal sensation (paresthesia). Information regarding the location and size of the sensory deficit, the severity and duration of symptoms, presence/absence of pain, and the nature of any pain reported (eg, is pain constant, transient, spontaneous, evoked?) should be recorded. Factors that make the pain better or worse should be noted. Documentation of returning sensation at follow-up visits is also important since it is an indicator of the degree of underlying injury and prognosis following microneurosurgery. As discussed earlier, returning sensation within 4 weeks of injury typically indicates first-degree injury. Later onset of sensation recovery (5 to 11 weeks) is associated with second-degree injury. Total "numbness" for 12 weeks or longer suggests third-, fourth-, or fifth-degree injury with disruption of the nerve sheath.[7]

Inspection of the head, neck, and intraoral region should be made to help determine the degree of nerve trauma. Any atrophic changes or cutaneous changes should be documented. Subsequent clinical examination is important to document any physical findings. Repeated radiographic evaluation of the area of surgery may be necessary to verify the implant location and to evaluate the need for change in implant placement.

Finally, one must specify the level and extent of the deficit by performing a detailed localized neurosensory examination. Problems with lip or tongue biting, drooling, and difficulty with chewing, drinking, speaking, or brushing should be discussed and noted.

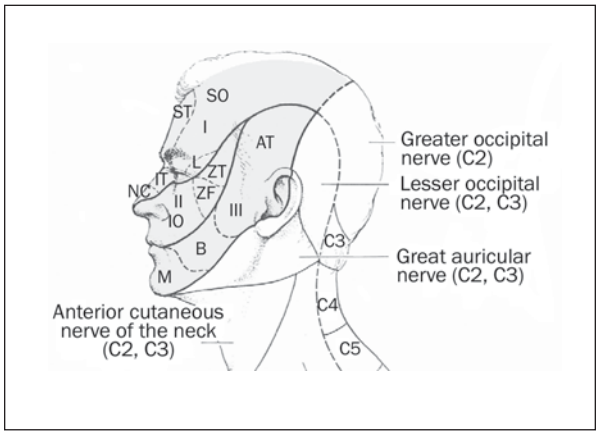

Fig 5 Distribution of the cutaneous fields of the 3 peripheral divisions of the trigeminal nerve: the ophthalmic division (I), the maxillary division (II), and the mandibular division (III). Abbreviations indicate the following nerves within the trigeminal divisions: AT = auriculotemporal, B = buccal, IO = infraorbital, IT = infratrochlear, L = lacrimal, M = mental, NC = nasociliary (external branch), SO = supraorbital, ST = supratrochlear, ZF = zygomaticofacial, ZT = zygomaticotemporal. There are no dermatomic overlaps between the 3 divisions of the trigeminal nerve. Reproduced with permission from Carpenter.[10]

The basic neurosensory examination should consist of light touch, brush directional discrimination, 2-point discrimination, pinprick, nociceptive discrimination, and thermal detection. The area of sensory deficit is mapped using a 19-gauge needle, moving from the unaffected area to the affected area.[5] An eyebrow pencil can be used to outline the area, which can be photographed or drawn on the patient's chart. Affected areas can be compared to the zones associated with trigeminal nerve sensitization (Fig 5).[10] A diagnostic nerve block may also be indicated in patients with dysesthesia. If a peripheral nerve block relieves the patient's pain, one may assume that the problem is confined to the peripheral sensory nerve and not to the central nervous system.[9]

Once the examination is completed, the findings serve as a baseline to which all other neurosensory examinations will be compared. This allows the clinician to monitor the patient's progress over time. Results of the clinical examinations are compared with findings at subsequent examinations conducted at 4-week intervals until sensation has returned to normal or a decision has been made to perform microsurgery.[9]

INDICATIONS AND TIMING

The process of phagocytosis of necrotic tissue distal to the site of injury and initial axonal sprouting from the proximal nerve stump both occur over the initial

Table 2 Indications and Contraindications for Microneurosurgery

Indications for microneurosurgery	Contraindications for microneurosurgery
• Observed nerve severance • Total anesthesia beyond 3 months • Dysesthesia beyond 4 months • Severe hypoesthesia without improvement beyond 4 months	• Dysesthesia not abolished by local anesthetic nerve block • Sensation improving • "Excessive" delay after injury • Patient medically compromised

1- to 2-month period. After this, the distal neurotubules and the Schwann cells begin to atrophy and are being replaced by scar tissue.

Patients who experience loss of sensation and/or painful unpleasant sensation that fails to resolve after four months are potential candidates for microsurgery.[5] Surgery is not necessary if the patient is pain-free and continues to progress normally. The indications and contraindications for microneurosurgery are presented in Table 2. With an understanding of the structure, pathogenesis, and classification of nerve injuries, the clinician can approach the patient with postimplantation neurosensory disturbance with greater confidence. Careful evaluation, early diagnosis, and meticulous follow-up of the progress of nerve regeneration are the hallmarks of good clinical practice in the management of patients with nerve injuries (Fig 4). Timely referral for microneurosurgery is particularly important, since surgery can re-establish nerve continuity, improve sensation and motor function, and possibly relieve pain.

SUMMARY

Trigeminal nerve injuries remain relatively uncommon events after mandibular implant placement.[11,12] However, given the frequency of dental and surgical procedures in the mandibular area and the growing field of implant dentistry, it is likely that this complication will occur in a dental professional's practice. If sensory disturbance appears after implant placement, a nerve injury should be suspected. The patient should be carefully examined, the findings documented, and progress or return of sensation monitored diligently (Fig 4). When there is no improvement in the patient's status, a referral should be made for definitive evaluation for microneurosurgery. Early referral and intervention can be essential to the restoration of sensation after trigeminal nerve injury. Therefore, it is important for clinicians to be familiar with these types of injuries and their management so that when such injuries do occur, patients can be properly evaluated and monitored or referred for microneurosurgery promptly. The current standard of care for significant trigeminal nerve injuries after mandibular implantation is early referral to a specialist experienced in the management of nerve injuries.

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