



Trigeminal Nerve Injuries

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Injuries to peripheral branches of the mandibular division (V3) of the trigeminal nerve (TN5) should be of concern to dentists who place dental implant fixtures into the mandible. The TN5 supplies sensation to the face, mouth, teeth, and jaws (Fig. 1) These anatomical locations are involved in many important activities of daily living which depend on intact sensory input (Table 1). An injury to a branch of V3 (i.e., inferior alveolar nerve, IAN; mental nerve, MN; lingual nerve, LN; long buccal nerve, LBN) during dental implant surgery is a known and accepted risk of such procedures.

Despite knowledge of the anatomy, thorough preoperative evaluation, and proper surgical technique, TN5 injuries cannot always be avoided. Unfortunately, such injuries can cause loss or alteration of sensory perception (*paresthesia*) and/or painful sensation (*dysesthesia*). Patients may experience interference with orofacial activities on a spectrum from mild discomfort or annoyance to severe prostrating or debilitating pain and/or hypersensitivity. The resulting symptoms and/or functional impairments are distressing to patients, especially if the symptoms do not resolve

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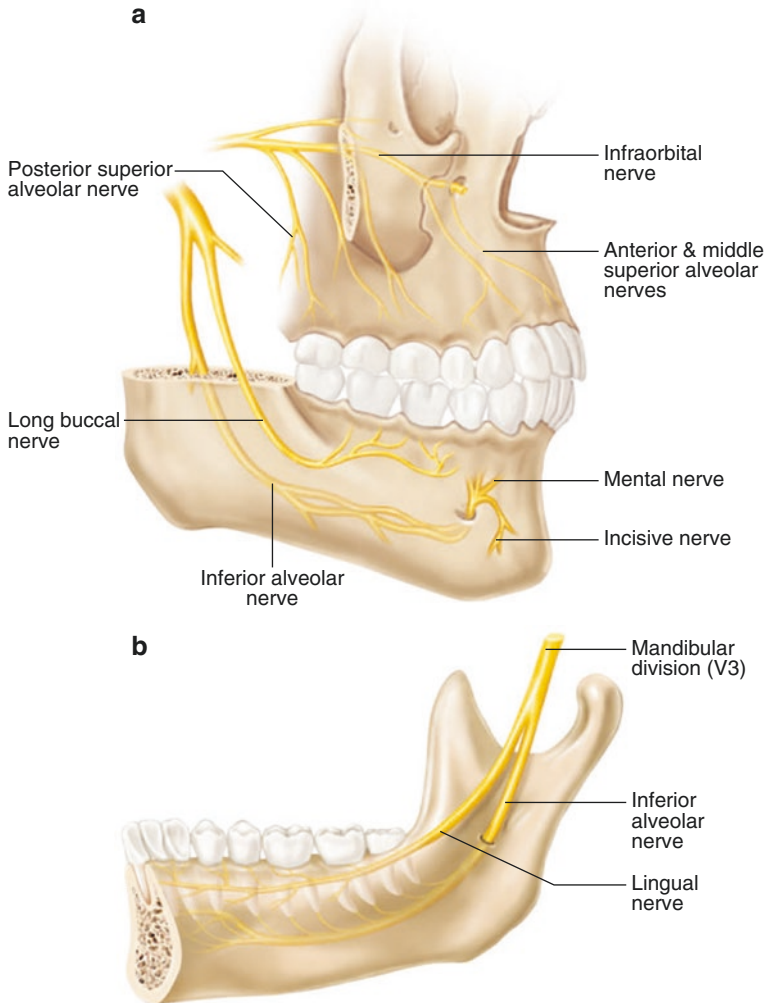


Fig. 1 (a) lateral aspect of maxilla and mandible; (b) medial aspect of mandible. Miloro M (ed): Trigeminal Nerve Injuries, Springer, 2013, Fig. 3.2, p. 29

Table 1 Important orofacial activities of daily living which can be adversely affected by an injury to a peripheral branch of the trigeminal nerve

Chewing food	Speaking/singing
Drinking liquids	Kissing and other oral sexual activity
Tooth brushing and flossing	Playing wind musical instruments
Face washing	Applying lipstick/make-up
Shaving	Smoking

promptly and the patient was not informed of the risk before treatment. Nerve injuries are currently the second leading cause of litigation against dentists in the USA. The “informed consent” doctrine in most states requires that the patient be informed of the risk of nerve injury during the preoperative discussion of dental

implants and the signing of a consent form. These injuries do not always heal, nor do the unpleasant sensations resolve, spontaneously. Such sequelae, if allowed to persist, can have devastating effects on a patient's quality of life. Therefore, prevention or early treatment of nerve injuries is essential in the care of the dental implant patient [1, 2].

1 Etiology and Prevention

Preoperative evaluation is integral to the successful completion of a dental implant procedure. Inadequate imaging of the mandible to determine the exact location of the inferior alveolar canal (IAC) and the mental foramen (MeF) is a common source of error leading to improper placement of an implant, especially when the mandibular alveolar ridge has become atrophic (Fig. 2). In such cases, the vertical distance from the crest of the alveolar ridge to the superior wall of the IAC may be inadequate for the length of the implant. Knowing this in advance enables the clinician to alter the treatment plan by either choosing an implant of lesser length or doing a nerve-repositioning procedure. A panoramic film, calibrated to reduce distortion, is the minimum requirement. In situations where the IAN or the MN is at risk of encroachment by the drilling procedure or implant placement, a three-dimensional imaging study (i.e., computed tomographic scan, CT; or, cone-beam computed tomographic scan, CBCT) provides additional important information (Fig. 3).

Every step of the implant procedure poses a risk of injury to the IAN, MN, LN, and LBN during the placement of dental implants into the mandible [1]. The well-trained, experienced, and proactive clinician will be able to modify his or her technique according to the needs of each patient to minimize the risk of nerve injury. Below are discussed those situations where nerve injuries may occur during dental implant procedures.

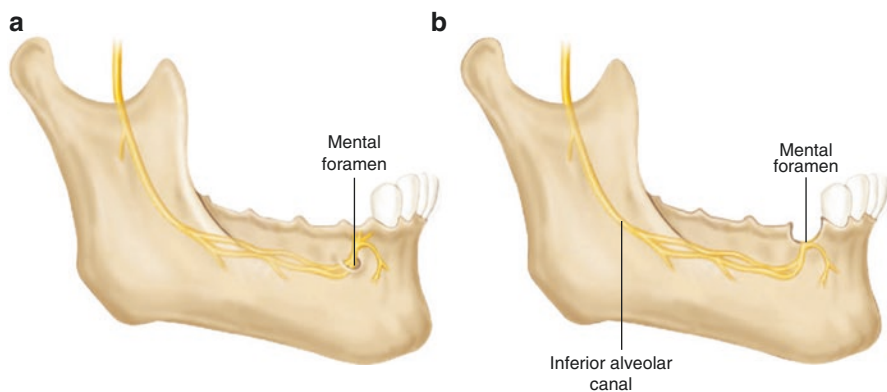


Fig. 2 (a) Superior position of the mental foramen due to resorption of alveolar bone in the area; (b) Inferior alveolar nerve in proximity to alveolar ridge. Miloro M: Trigeminal Nerve Injuries, Fig. 6.5 (a, b only), p. 93

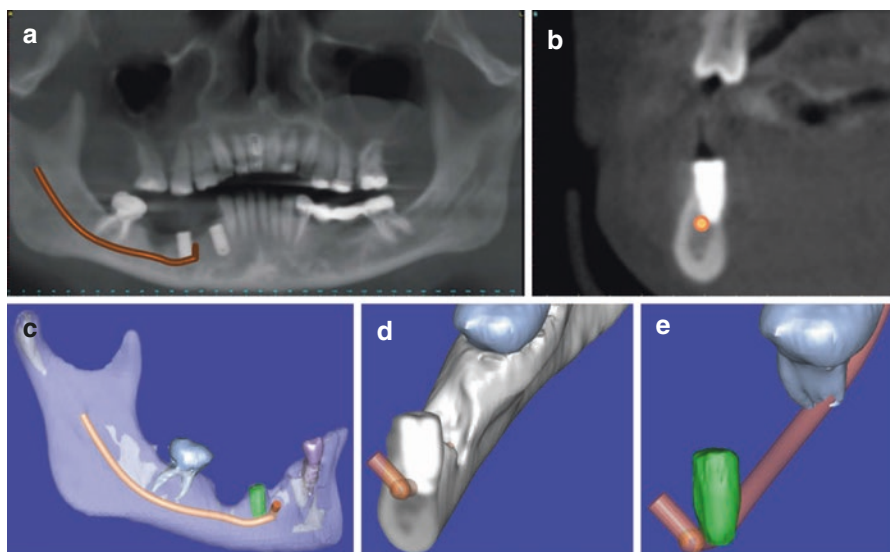


Fig. 3 (a) CBCT generated panoramic radiograph with right inferior alveolar canal in proximity to #29 dental implant; (b) Coronal slice of the same patient demonstrating #29 dental implant impingement on the right inferior alveolar canal; (c) 3D reconstruction with transparency of the osseous structures; (d) Cross-section of 3D reconstruction; (e) Osseous structures removed from previous image. Miloro M: Trigeminal Nerve Injuries, Fig. 6.6, p. 95

1.1 Errors in Diagnosis and Treatment Planning

Identification of the position of the inferior alveolar canal (IAC) can be determined with radiographic evaluation. The IAC defines the boundaries of the inferior alveolar neurovascular bundle. Anatomical analysis demonstrates that the IAN occupies approximately 80% of the canal space, the other 20% occupied by the inferior alveolar artery and vein. There is great variation in the position of the artery and vein in relation to the nerve. The panoramic radiograph (orthopantomogram) is useful as a primary imaging study to evaluate the vertical distance from the crest of the alveolar bone to the position of the IAC. Each panoramic system should be calibrated with its software to account for distortion and magnification. There is generally a magnification of 10–40% with more magnification of areas that are outside the focal trough of the beam. A typical magnification of the mandible is 20%. As a two-dimensional image, the mediolateral position of the IAC and path of the mental nerve anterior loop cannot be accurately assessed.

If there are anatomical concerns or if virtual surgical planning is indicated or desired, a computed tomographic (CT) scan or cone-beam CT scan is necessary. There have been many advances with implant planning software which is available to develop CT-guided implant planning and custom surgical guides. Regardless of the radiographic modality used, errors in interpretation and application may lead to errors in implant positioning. Errors in digital implant planning may also be transferred to the surgical procedure. With regard to the surgical guide, attention should be given to the accuracy and stability of the guide in situ. Surgical guides supported

by an edentulous mandible will have a margin of error related to the soft tissue despite correct digital planning. The use of a “flapless” technique for implant placement has gained popularity for a variety of reasons, it is accepted as less accurate than direct visualization of the alveolar bone. The surgeon should never hesitate to reflect a mucoperiosteal flap for better visualization and accuracy. Bone-borne and tooth-borne guides may help to improve accuracy and stability of a surgical guide by providing a fixed landmark. When planning implant length and depth of placement, it is important to allow an additional distance (2 mm) from the superior aspect of the IAC to allow for any margin of error in planning or placement.

1.2 Local Anesthesia

Injection of local anesthetic might cause nerve injury by direct trauma to nerve tissue or adjacent blood vessels or chemical toxicity to the nerve [3]. Introduction of a local anesthetic needle into the pterygomandibular space (PTMS) in close proximity to the IAN and LN is essentially a blind procedure. It is a testament to the clinician’s careful technique that such a small percentage of injections result in significant nerve injury. In order to minimize this risk, the authors recommend a protocol for local anesthetic injections [4]. In the fully conscious patient, the local anesthetic needle is inserted into the proper location in the PTMS. If the patient does not complain of sudden sharp pain or shocking sensation (*dysesthesia*) which may radiate to the lower teeth, lower lip, lower jaw, or tongue, the syringe is aspirated. If the aspirate contains no blood, the local anesthetic solution is injected with the needle position unchanged. However, if there is a bloody aspirate, the needle is withdrawn 2–3 mm and aspiration is repeated. If the aspirate is now clear, the local anesthetic solution is injected with the needle in the new position. If the patient experiences dysesthesia, the clinician proceeds similarly as with a bloody aspirate. After withdrawal of the needle (as above), the injection proceeds. If there is a bloody aspirate or a dysesthesia during the injection, the incident is noted in the patient’s record, and an evaluation of sensory function is done at the patient’s next visit. When the patient is under intravenous sedation or general anesthesia, he/she will not be able to react to a dysesthesia. Therefore, aspirate before the injection and proceed as described above.

1.3 Surgical Flap

During the incision, elevation, or retraction of mucoperiosteal flaps, attention to the position of the IAN, MN, LN, or LBN is especially important when the mandible has undergone significant alveolar resorption. The mental nerve branches are within the buccal and labial mandibular soft tissues and at risk of iatrogenic injury during a vestibular incision. They are also at risk of thermal injury from use of electrocautery in close proximity. Recognition of the anatomical position of the mental foramen, the intra-bony mental loop and the approximate position of the branches within the soft tissues is particularly important in preventing injury to this portion

of the nerve. It is also important to recognize the changing anatomy of the edentulous mandible. As mandibular alveolar bone resorbs with age, the position of the mental foramen approaches the crest of the alveolar ridge. In some patients, there is an actual dehiscence of the IAC and the IAN and MN come to lie on the crest of the alveolar ridge (Fig. 2). Therefore, incision design must take this anatomical position into consideration.

Injury to the MN and its branches may also be sustained as a stretch or retraction injury. During the elevation and retraction of a mucoperiosteal flap, which may also contain a nerve, gentle manipulation and retraction with frequent brief periods of relaxation of retraction pressure is indicated.

1.4 Nerve-Repositioning

A nerve-repositioning procedure is sometimes helpful to relocate the IAN or MN out of harm's way when preoperative imaging studies indicate that the nerve would be in the path of a properly positioned implant [5]. These procedures demand precise and careful microsurgical technique and should only be undertaken by a surgeon with training in microneurosurgery. The procedure can be done at the same operation as the placement of the dental implant (Fig. 4). An autogenous bone graft, either from the bone removed to unroof the IAC or elsewhere (e.g., from the ipsilateral mandibular ramus) is placed between the repositioned nerve and the associated implant(s) to prevent direct contact and thermal transmission between the implant(s) and the nerve. Alloplastic material, such as calcium hydroxyapatite, should never be placed in direct contact with a nerve. A severe inflammatory reaction producing dense scarring of the nerve, accompanied by intractable pain, is often the unfortunate result. Surgical or other treatment of such injuries is problematic [1]. Exposure and retraction of the nerve, although not causing anatomical disruption, is always followed by one to several months of decreased sensation [6]. Careful exposure, retraction, and repositioning of the nerve will reduce, but not entirely eliminate the risk of permanent nerve dysfunction. However, in most patients the sensory function returns ultimately to normal or acceptable (to the patient) status.

1.5 Implant Osteotomy

An osteotomy in preparation for implant insertion can cause injury to the IAN. Errors in planning due to inaccurate measurements from distorted imaging studies and miscalculation of drill depth allow penetration of the inferior alveolar canal (IAC) and direct trauma to the IAN (Fig. 5). Indirect nerve trauma can occur, if inadequate cooling of the drill allows generation of excessive heat, thereby causing a thermal nerve injury. Taking preoperative measurements from calibrated imaging studies, careful drilling technique with frequent intraoperative verification of drill dimensions (diameter and length), and irrigation with adequate coolant minimize such risks to adjacent nerves.

1.6 Implant Placement

Over-insertion of the implant with either indirect contact by dislodged bone impacting on the IAC or entrance into the IAC with direct nerve contact cause compression injury of the IAN. Disruption of the superior wall of the IAC during the drilling procedure or by over-insertion of the implant may cause a *delayed nerve injury*. As osseous regeneration occurs, production of new bone may be greater than that which

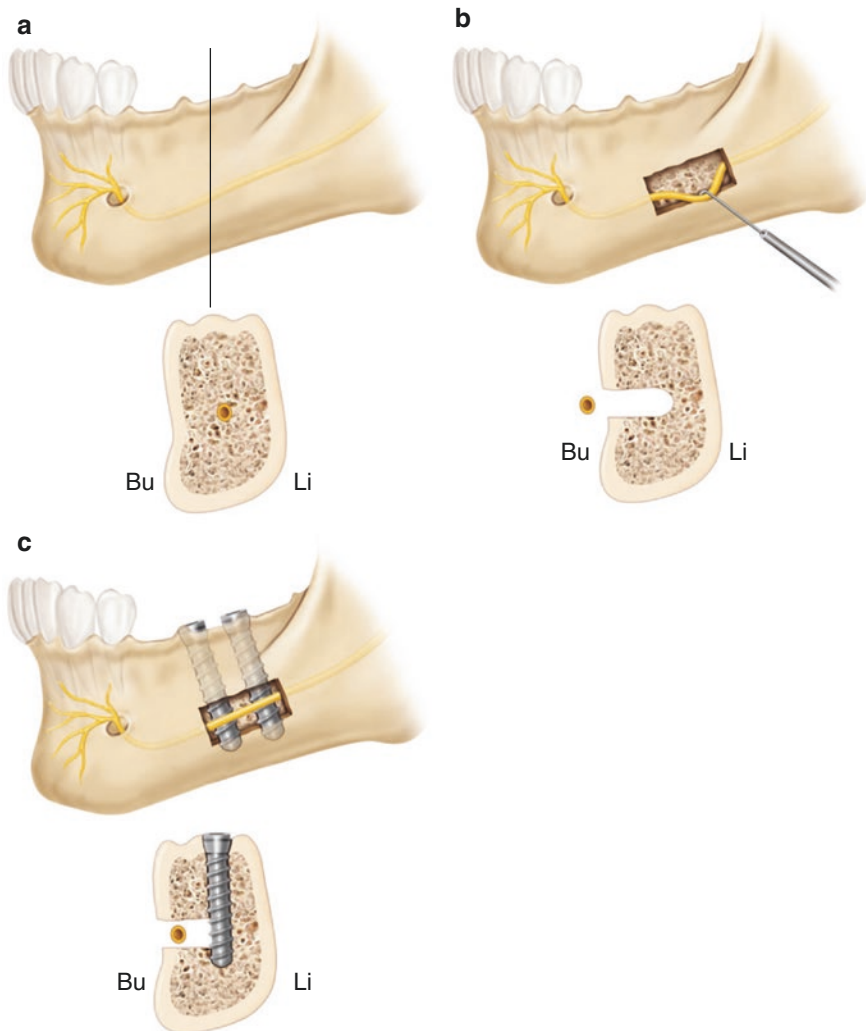


Fig. 4 (a) Schematic representation of anticipated site for dental implant placement, posterior mandible; (b) Inferior alveolar nerve lateralization; (c) Placement of two dental implants beyond the inferior alveolar canal; (d, e) Panoramic radiographs, pre- and post-operative clinical example. Miloro M: Trigeminal Nerve Injuries, Fig. 6.10, pp. 100–101

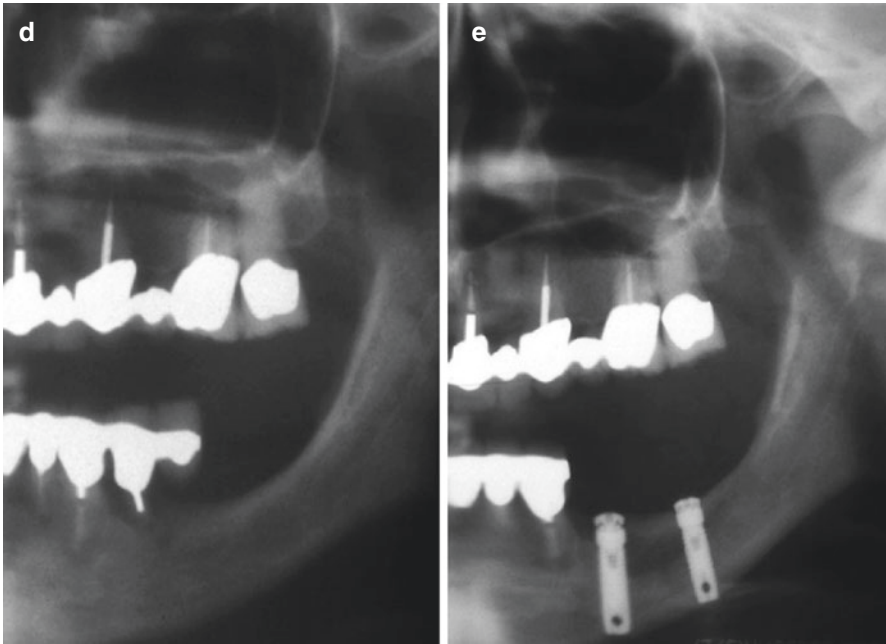


Fig. 4 (continued)

Fig. 5 Diagram of direct trauma to the inferior alveolar nerve during an osteotomy. Miloro M: Trigeminal Nerve Injuries, Fig. 6.2, p. 90



existed before, causing narrowing of the canal and compression of the nerve. In such case, the onset of sensory symptoms and signs may occur weeks to months after the implant procedure [7] (Fig. 6). Such delayed injuries are difficult to prevent and predict, but should be suspected when the onset of sensory dysfunction develops late following an implant procedure.

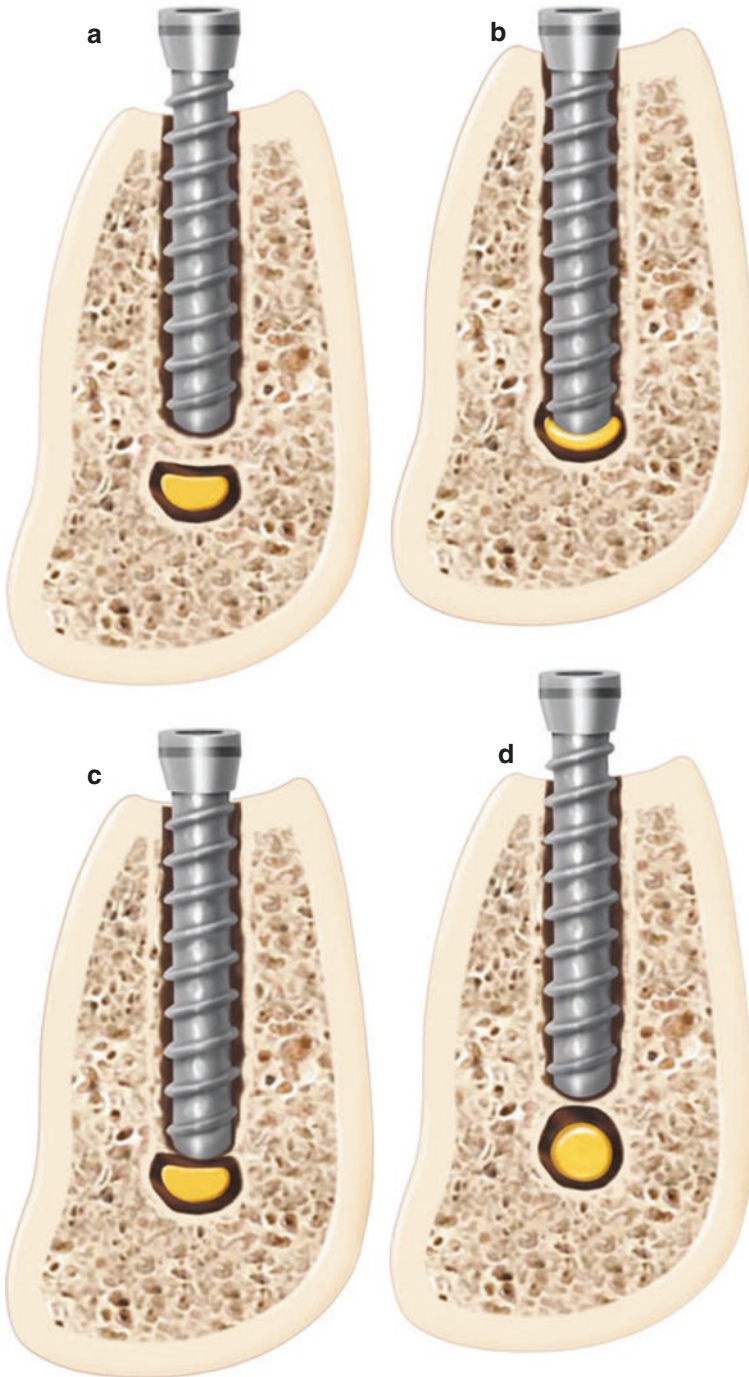


Fig. 6 (a) Compression and collapse of the superior aspect of the inferior alveolar canal due to implant placement beyond the planned osteotomy; (b) Direct injury to the inferior alveolar nerve by implant contact; (c) Over-insertion of implant disrupting superior wall of inferior alveolar canal resulting in *immediate* inferior alveolar nerve injury; (d) Over-insertion of implant disrupting superior wall of inferior alveolar canal resulting in *delayed* osseous regeneration and narrowing of the canal. Miloro M: Trigeminal Nerve Injuries, Fig. 6.4, p. 92

1.7 Medications

The perioperative administration of medications which limit the inflammatory response has been advocated for patients undergoing procedures such as dental implants, mandibular osteotomies, and lower third molar removals which are associated with a risk of nerve injury [8, 9]. It is recommended that the dental implant patient be given a single appropriate preoperative oral or intravenous dose of a corticosteroid (e.g., dexamethasone or hydrocortisone).

1.8 Bone Graft

Another etiology of nerve injury associated with implant surgery is placement of bone grafts. This can be in implant site development, ridge preservation technique, or bone grafting around implants. This is a less common cause of injury, but can be the result of impingement, compression, or crushing the IAN with overpacking or placing grafting material with excessive force. In the authors' experience, there have even been cases of material (autogenous, allogenic, xenogenic) migration around the MN and resulting in impingement of the branches exiting the mental foramen. Scarring in this area can result in neurosensory disturbance or even dysesthesia.

2 Evaluation

If a nerve injury is directly observed (open injury) during treatment, a nerve injury specialist (generally, an oral and maxillofacial surgeon who has had additional training and experience in the overall evaluation and management, including microsurgery, of TN5 injuries) should be contacted promptly and the patient referred without delay for further evaluation and treatment [10]. If no nerve injury was observed (closed injury) at the time of dental implant placement, but the patient subsequently returns complaining of numbness or pain, the nature and intensity of the painful symptoms are noted (use of a visual analog scale, VAS, is recommended). The patient is examined and responses to pain (pinprick or algometer), static light touch (cotton wisps or Semmes–Weinstein monofilaments), two-point discrimination (calipers), and moving brush stroke direction identification (cotton wisp or Von Frey hairs), so-called neurosensory testing (NST), are documented [11]. Record the history and examination findings in the patient's chart for comparison with subsequent patient VAS pain estimates and examinations, including NST, to assess progress of recovery (if any). Take a panoramic radiograph which clearly shows the implant(s) and the surrounding bone to determine the relationship of the implant to the IAC and mental foramen (MeF). If the implant is seen on the screening film to be superimposed on the IAC or MeF, a CT or CBCT scan must be obtained in order to accurately determine the mediolateral position of the implant and to ascertain if there is direct contact of the implant with the IAC or MeF. Remove or reposition the implant only if there is evidence upon imaging of encroachment of the implant upon the nerve. An implant that is not directly in contact with the IAC as seen on an imaging study should not be removed. Its removal will not have any effect on possible recovery of the nerve, and the patient will have lost a potentially functional

implant. The authors' protocol for management of closed (unobserved) TN5 injuries from dental implant surgery is summarized in Fig. 7.

The benefit of initiating corticosteroid or anti-inflammatory (NSAID) medications after a nerve injury has occurred is questionable [1, 9]. The patient is followed by the dentist at regular intervals (i.e., weekly), and a review of the progress of symptoms and reevaluation of sensory responses is done at each visit and noted in the patient's record.

Seddon's classification of peripheral nerve injuries (Sir Herbert Seddon, 1903–1977, a British neurosurgeon during and after WWII) is based on clinical findings and typical time frames, and is helpful in making a diagnosis, a prognosis, and timely treatment decisions [12].

NEURAPRAXIA is a benign injury similar to a concussion. Temporary interruption of nerve conduction without axonal discontinuity is produced. There is no demonstrable anatomic disruption of the nerve, and axonal degeneration does not occur. Spontaneous recovery is complete within 4 weeks. Surgical intervention or other treatment is not necessary.

AXONOTMESIS is a more significant injury. There is loss of continuity of some axons, but the body (e.g., connective tissue/epineurium) of the nerve remains intact. Prolonged (greater than 4 weeks) conduction failure occurs. Initial symptoms of returning sensation (tingling, itching, crawling, burning, hypersensitivity) do not begin until 5–11 weeks after injury. Eventual recovery of sensation is often less than normal, and it may be accompanied by dysesthesias. Surgical repair for removal of scar tissue, compressing bone, foreign material, or

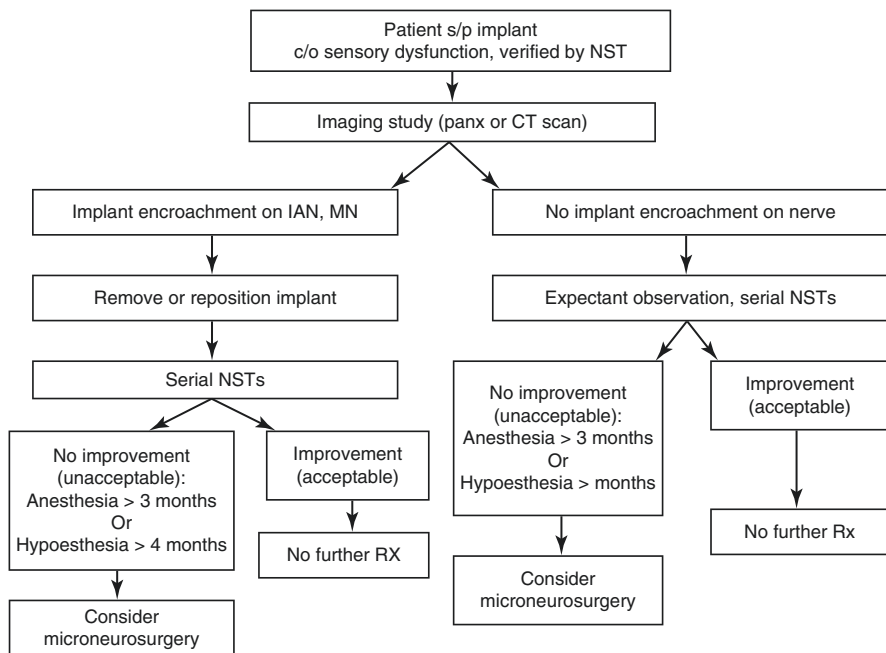


Fig. 7 Algorithm for the management of trigeminal nerve injury from dental implant surgery. Miloro M: Trigeminal Nerve Injuries, Fig. 6.1, p. 94

neuromas is often helpful in improving sensation or resolving dysesthesias, but the best results are achieved **ONLY** if done in a timely fashion (within 4 months in painful conditions, 6 months in others). After this time, nerve degeneration proximal and distal to the injury, deafferentation (loss of peripheral sensory input to an area of the central nervous system, CNS), and/or development of a “learned pain response” places the patient at risk to develop intractable neuropathic pain not amenable to peripheral nerve surgery, and which often fails to respond to any other treatment.

NEUROTOMESIS is complete physical separation (severance) or internal physiologic disruption of the nerve with total and permanent failure to transmit sensory impulses from the periphery to the CNS. Without timely surgical repair of the nerve, there is little chance of spontaneous recovery of sensation. Permanent anesthesia is the result of nontreatment. Disabling dysesthesias often develop as well. In our clinical experience and that of others, no patients with documented total anesthesia persisting beyond 3 months have spontaneously regained significant sensation at a later date [13]. Therefore, for best chance of recovery of sensation, surgical nerve repair should be done within 3–6 months after injury before distal axonal tubules begin to atrophy and become unable to accept new axonal sprouts regenerating from the proximal nerve stump [1, 2, 10, 12, 14]. The process of axonal tubular atrophy probably becomes irreversible at between 9 and 15 months post-injury (depending at least partially upon the age and general health status of the patient). Attempted surgical repair of neurotmesis beyond this time is generally less than satisfactory, and it should not be electively delayed this long in any patient.

3 Treatment

Patients with documented closed nerve injuries which do not resolve completely within 4 weeks should be referred promptly for evaluation to a nerve injury specialist. If nerve repair becomes necessary, it can be done at the optimal time to maximize the chance for satisfactory sensory recovery. In general, the following guidelines will assist you in determining the best course of action for your patient with a nerve injury:

1. An **OBSERVED** (open) nerve injury should be referred **WITHOUT DELAY**.
2. Patients in **SEVERE PAIN** should be referred promptly.
3. A **CLOSED** (unobserved) injury can be followed and re-examined weekly for 4 weeks. If normal sensation has not returned by that time, refer the patient to a nerve injury specialist.

Microneurosurgery, or nerve repair surgery, is technically demanding and requires additional training, specialized instruments, and magnification with loupes or an operating microscope. Microneurosurgery is done in the hospital operating room under general anesthesia. Depending on the type and location of the injury, the nerve is exposed by an intraoral incision or by an inconspicuous submandibular skin incision. After the nerve is visualized, the surgeon may perform one or more of the following procedures: (Figs. 8 and 9)

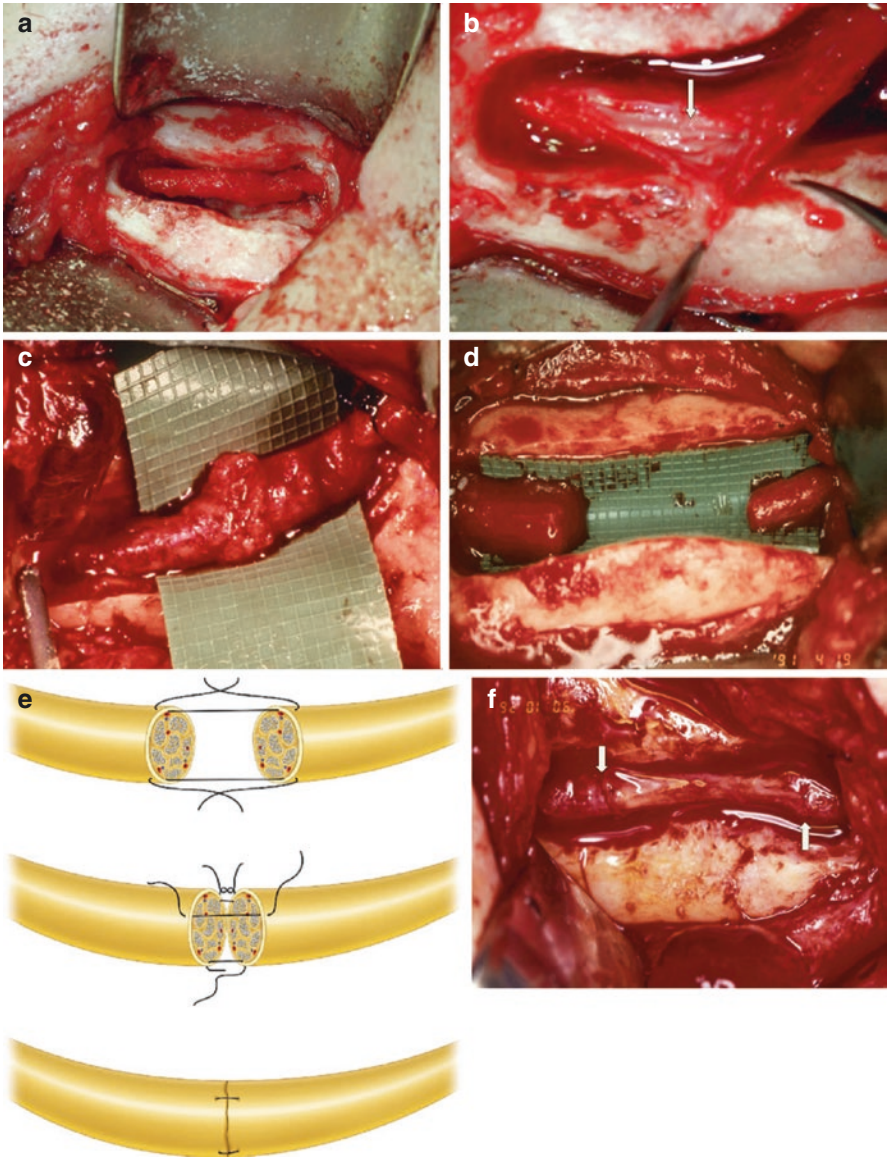


Fig. 8 (a–h only) (a) External decompression of the inferior alveolar nerve; (b) Internal neurolysis; (c) Neuroma-in-continuity; (d) Inferior alveolar nerve after excision of neuroma; (e) Diagram of direct neurorrhaphy; (f) Use of an autogenous sural nerve graft for inferior alveolar nerve reconstruction; (g) Use of an human nerve allograft for inferior alveolar nerve reconstruction; (h) Diagram of guided tissue regeneration with a conduit repair. Miloro M: Trigeminal Nerve Injuries, Fig. 6.7, pp. 97–98

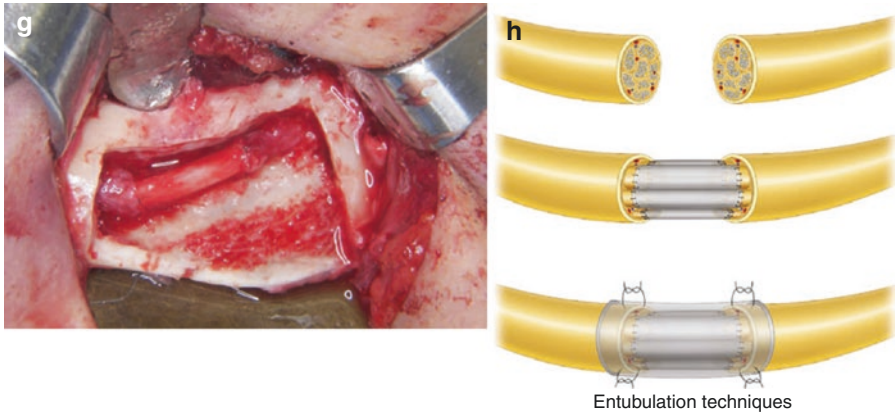
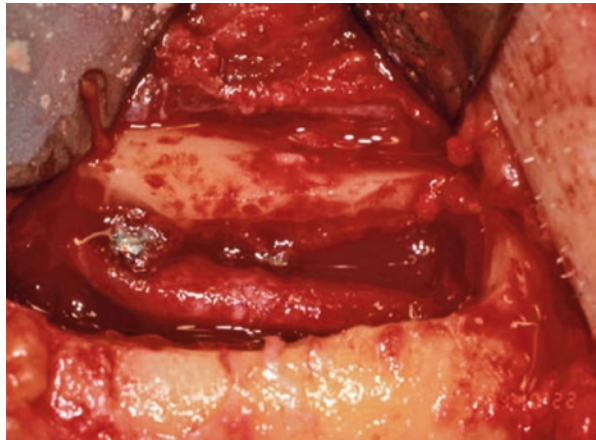


Fig. 8 (continued)

Fig. 9 Example of a dental implant impinging and deforming the integrity of the inferior alveolar nerve. Miloro M: Trigeminal Nerve Injuries, Fig. 6.8, p. 99



1. **Decompression:** removal of surrounding scar tissue, bone or foreign material. (Note: Seldom is removal of a well-integrated implant necessary or desirable.)
2. **Repositioning** of the nerve away from direct contact with the dental implant(s).
3. **Internal neurolysis:** examination of the internal structure of the nerve and removal of scar tissue from between or within nerve fascicles.
4. Excision of a neuroma or other abnormal nerve tissue followed by reconstruction.
5. **Neurorrhaphy:** dissection and mobilization of the proximal and distal limbs of a severed nerve to allow passive coaptation and tension-free suturing of the nerve.
6. **Nerve graft** to reconstruct a gap in nerve continuity that cannot be brought together without tension. Whereas in the past autogenous nerve grafts (ANGs; i.e., sural nerve from the lower extremity, great auricular nerve from lateral neck), were the standard of care in reconstructing nerve gaps [15], it is now common practice to use processed homologous nerve grafts (HNGs) or nerve allografts (obtained from donors and rendered immunologically inert). HNGs spare the patient a second

surgical site from which to obtain the graft and do not require immunosuppressive therapy. In recent clinical experience they appear to have a success rate similar to ANGs [16].

The prognosis for recovery of sensation after microsurgical repair of a peripheral nerve injury is dependent upon: (a) the length of time between injury and repair (the sooner, the better), (b) the age of the patient (young better than old, especially <45 years of age), (c) the type of sensory dysfunction (restoration of sensation is easier to achieve than relief of pain, especially if chronic, i.e., >4 months duration), and (d) the technical skill of the surgeon. There is a learning curve for microneurosurgery which requires about 100 operations before a steady and predictable rate of success is possible. Success rates (based on functional sensory recovery (FSR) or a Medical Research Council System grade of 3.0–4.0) [14, 17, 18] in an experienced surgeon's hands should be 80% or better in patients operated at 6 months or sooner after injury, but may drop to 30% or less in patients operated at longer than 1 year after injury [1]. In one report on immediate repair of the IAN as part of reconstruction of ablative oncologic surgery of the mandible, all patients regained useful sensory function in the lower lip, chin and gingiva [19]. This would seem to indicate that the best results occur when the injured nerve is repaired at the time of its injury. In another retrospective study, early nerve repair (defined as <90 days after injury) resulted in a higher rate of FSR than did late repair (i.e., >90 days after injury) [14]. Therefore, the sooner the repair of the nerve, the more likely is a successful outcome. However, in instances where nerve repair is delayed for various reasons, even a repair done beyond the favorable period might result in a partial recovery which is acceptable to the patient, especially if dysesthesias are decreased or resolved.

4 Surgical Case Examples

CASE 1: A 48-year-old man presented 6 weeks after #31 dental implant placement with profound numbness to his right V3 distribution. He was referred by the primary implant provider (PIP) after 6 weeks of no improvement in his paresthesia. Subjectively, he denied any spontaneous or provoked pain in the affected area and denied any improvement in his level of numbness. His examination was significant for right V3 severe hypoesthesia without dysesthesia or hyperalgesia. His intraoral examination was unremarkable, with a healing abutment on #31 implant and normal soft tissues. A panoramic radiograph and CBCT revealed #31 implant in proximity to the right IAN canal and violation of the superior cortex of the canal. The surgeon and patient decided on neurosurgical intervention to include right IAN exploration, decompression, and repair with nerve allograft as needed through an intraoral approach (Fig. 10).

CASE 2: A 58-year-old woman presented 4 months after extraction of multiple mandibular teeth and placement of four dental implants in the areas of first molars and canines. Immediately post-op, she had numbness in her left lower lip and chin and she developed intractable pain in her left mandible with radiation to her lip and chin that was only minimally relieved with analgesics. The left, more posterior implant was removed by her PIP 7 days after placement, following which there was some diminution of her pain. At 4 weeks status post left posterior implant removal,

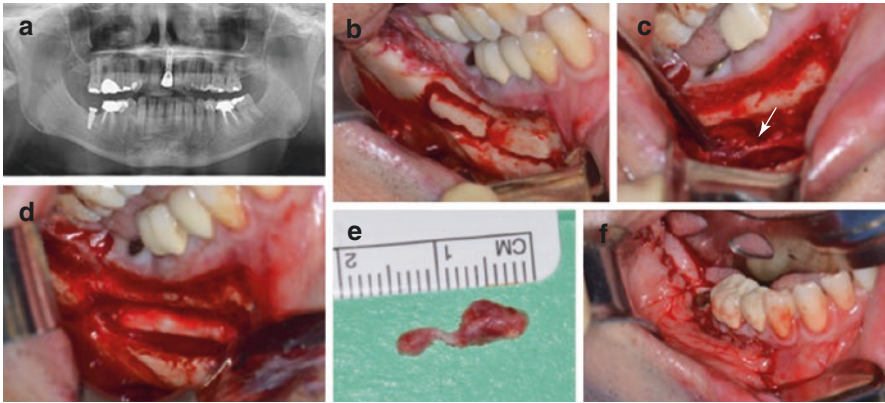


Fig. 10 (a) panoramic radiograph; (b) intraoral approach with initial osteotomy; (c) injured IAN with adjacent implant apex; (d) nerve allograft in place; (e) injured nerve segment; (f) final closure

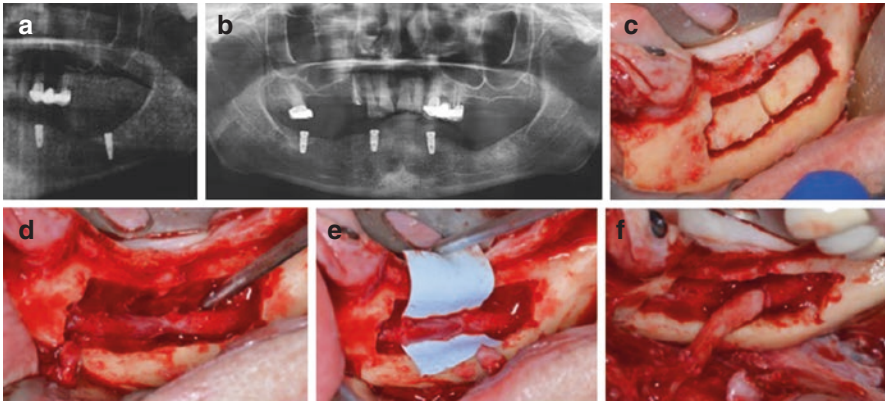


Fig. 11 (a) Copy of panoramic radiograph at initial implant placement; (b) panoramic radiograph 4 months status post explant; (c) intraoral approach with initial osteotomy; (d) injured IAN; (e) injured IAN with background; (f) coaptation of nerve allograft

she continued to have numbness and pain, and burning to her lip and chin had returned. She was referred by her PIP after nonsurgical interventions were unsuccessful. Her examination was significant for moderate hypoesthesia of the left V3 distribution with dysesthesia and hyperalgesia. Her intraoral examination was mostly unremarkable as she had three remaining mandibular implants with healing abutments and the site of the previously removed implant had healed normally. Her imaging studies were normal. The surgeon and patient decided on neurosurgical intervention to include left IAN exploration, decompression, and possible repair as needed via an intraoral approach (Fig. 11).

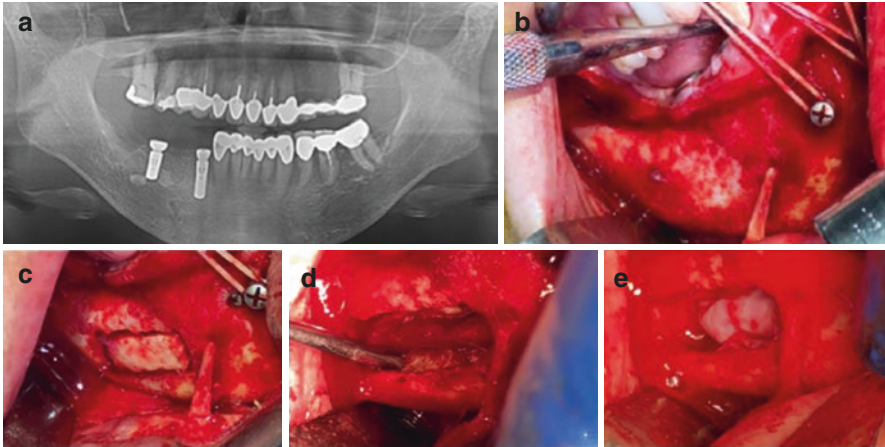


Fig. 12 (a) panoramic radiograph; (b) intraoral approach with intact mental nerve branch and evidence of bone grafting; (c) initial osteotomy; (d) IAN external decompression; (e) IAN protected with membrane after decompression

CASE 3: A 59-year-old woman presented 2 months after implant placement at #28 and #30 sites after staged alveolar bone grafting to the right mandible. She complained of persistent numbness and a constant severe burning sensation of her right lower lip. She was referred by her PIP after several weeks of no improvement in her symptoms. Her examination was significant for mild hypoesthesia of the right V3 distribution and severe dysesthesia. Her panoramic radiograph and CBCT displayed dental implants at #28 and #30 sites; #28 implant was unremarkable, #30 implant was in contact with the superior cortex of the IAN canal and there were areas of bony radiopacities around the canal in this area. The surgeon and patient decided on neurosurgical intervention with immediate decompression of the right IAN and possible repair as needed via an intraoral approach (Fig. 12).

CASE 4: A 33-year-old man presented 3 months after extraction of remaining mandibular teeth and placement of four dental implants in the areas of #19, #22, #27, and #31. Immediately post-op he reported numbness in his right lower lip and chin. Three days later his PIP removed the implants at the #27 and #31 sites and placed new implants at the #29 and #30 positions, but his numbness persisted. He was referred for further evaluation and treatment after unsatisfactory progress over 3 months. His examination was significant for severe right V3 hypoesthesia and hyperalgesia. His intraoral examination revealed four mandibular implants with healing abutments and normal healing of the sites of the previously removed implants. His panoramic and CBCT examinations displayed normal bony healing of previous implant and extraction sites. The surgeon and patient decided on microsurgical exploration of the right IAN with decompression and possible repair with a nerve allograft (Fig. 13).

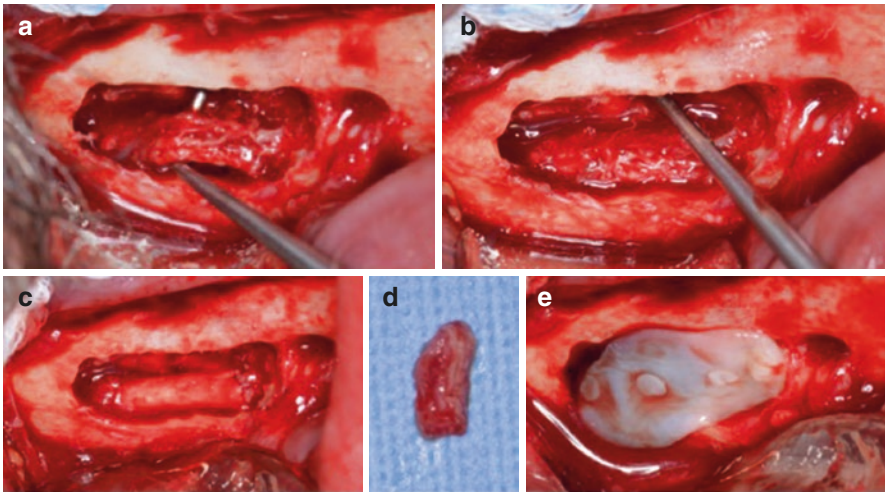


Fig. 13 (a) Injured nerve segment; (b) nerve hook within previous implant osteotomy adjacent to injured IAN; (c) nerve allograft in place; (d) injured nerve segment; (e) platelet-rich fibrin covering osteotomy

5 Nonsurgical Treatment and Sensory Rehabilitation

Sensory nerve injuries which do not require surgical intervention, or those recovering from a microneurosurgical operation for nerve repair, may benefit from neurotropic medications to relieve persistent neuropathic pain or hypersensitivity [20]. Physical therapy, exercise, yoga, psychological counseling, and psychiatric therapy including behavioral modification have helped many patients with post-traumatic, persistent neuropathic pain [21]. Such treatment is often provided in pain-management clinics staffed by specialists in the fields of anesthesiology, neurology, psychiatry, neurosurgery, and physiatry (physical medicine and rehabilitation).

Measures to enhance sensation and restore related orofacial functions are routinely included in the rehabilitation of the patient with a sensory nerve injury in order to maximize the end result of treatment [1]. Younger patients generally achieve better functional recovery after peripheral nerve injury and repair than mature adults (>45 years of age). Clinical experience indicates that the efficiency of tissue regeneration decreases with age. However, neuropsychological factors also influence the ability of the patient to recover successfully from a peripheral nerve injury and its repair. During the recovery process following nerve injury and repair, there may be new axonal connections with referral of sensory input to different areas of the CNS. Processing of that information requires time and practice to relearn correct interpretation of sensory input. A healing nerve's conduction speed is slowed, which requires further adaptation. Although the older patient is slower to adapt to changes imposed on the CNS after peripheral nerve injury, neuroplasticity (the ability of the brain to adapt and learn), even after traumatic injury or ablative tumor surgery, is

still viable into advanced age [22]. One can teach an old dog new tricks; it just might take longer!

The concept of sensory reeducation (SRed) was introduced by Wynn Parry in the 1960s for the rehabilitation of hand and upper extremity injuries [23]. SRed has been adapted to the oral and maxillofacial regions and shown to be successful in improving sensory function, especially in the patient's subjective interpretation of input [18, 24]. Daily SRed exercises are initiated following nerve repair, after responses to pain and light touch have been restored, and they are continued for at least 1 year, or longer if needed to achieve patient satisfaction. Long-term follow-up indicates that sensory nerve injury patients experience more favorable sensory function when SRed is included in their postoperative care regimen. SRed is also often useful in the rehabilitation of sensory function in patients whose TN5 injury did not require surgical repair.

6 Summary and Conclusions

There is a risk of injury to peripheral branches of V3 during dental implant surgery. Such injury is a known and accepted risk, and it should be included in the preoperative surgical consent process. Accurate preoperative evaluation and imaging studies and careful surgical technique can minimize this risk. If a nerve injury does occur, prompt evaluation and treatment gives the patient the best chance for a successful recovery of useful sensory function. Because the majority of dental implant patients are mature adults (>45 years of age), the potential for less than satisfactory healing or development of chronic neuropathic pain is magnified, especially if not evaluated and treated in a timely fashion according to the protocol discussed above. In developing a useful philosophy regarding the treatment of peripheral nerve injuries in general, and TN5 injuries in particular, two statements from the past literature on this subject have stood the test of time and govern our current principles of peripheral nerve injury management. In 1947 Seddon wrote, "If a purely *expectant* (i.e., observation only; emphasis by the authors) policy is pursued, the favorable time for operative intervention will always be missed..." [12]. In 1992 Colin and Donoff advised, "We emphasize that the current standard of care for these complex injuries is *early referral* to clinicians *familiar with their management* (emphasis added by the authors)" [25].

The incidence of nerve injuries associated with dental implants is unknown, although nerve injury specialists (including the authors' practice) see such injuries frequently. Reliable statistics are lacking at present. A typical article in the literature contains an uncontrolled study with small numbers of patients and inadequate data on sensory evaluation from a single practice or center [26]. It seems logical that when a new surgical procedure is introduced, practitioners' early experience is fraught with more complications, which hopefully diminish in frequency and severity, as the "learning curve" is surmounted. The development of a national dental implant data collection center to which all patients who receive dental implants would be registered and followed by their practitioners, with mandatory reporting

of complications (including nerve injuries and/or whether an implant was removed), would do much to elucidate the magnitude of dental implant-associated TN5 injuries and provide information which might assist nerve injury specialists in their care.

Acknowledgment *Disclaimer:* The views expressed in this material are those of the author(s) and do not reflect the official policy or position of the U.S. Government, the Department of Defense, or the Department of the Air Force.

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