Chapter 25

Inferior Alveolar Nerve Transpositioning for Implant Placement

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Additional information is available at the end of the chapter

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1. Introduction

Premature loss of posterior teeth in the mandible, failure to replace lost teeth as well as systemic factors may result in progressive resorption of the alveolar ridge. At present, oral and maxillofacial surgeons aim to reconstruct the lost bone and masticatory function via posterior mandibular grafting and/or implants. However, anatomic limitations such as the inferior alveolar nerve (IAN) may limit this. Various treatment methods are available for treatment of patients with posterior mandibular atrophy presenting with a superficial IAN; each has its own merits and drawbacks. [1,2] Use of removable or fixed prosthetics and reconstruction of the dentoalveolar system by dental implants are among the available treatment options; a superficial IAN often precludes use of the latter. Implant-based reconstruction has several advantages i.e. allows for placement of longer implants, bone preservation, better functionality etc. and is gaining more proponents. However, certain conditions should be met in order for an implant to be placed. The most important condition is the quality and quantity of the bone. The amount of resorption, density of the bone and level of the nerve may limit implant placement. Reconstruction and rehabilitation of the dentoalveolar system in cases with alveolar ridge atrophy is a challenge for maxillofacial surgeons and prosthodontists. To date, several treatment options such as augmentation techniques with bone grafts [3], cartilage [4] or hydroxylapatite [5], vestibuloplasty [6] and several osteotomy techniques [7] have been suggested. Such treatments are still indicated as alternatives for cases in which for some reason dental implants cannot be placed [8]. In order to place an implant, we need adequate bone volume (both mediolaterally and mesiodistally) with optimal bone density.

This condition is usually not met in atrophic areas of the posterior mandible especially in patients that have been edentulous for some time. As the alveolar ridge becomes atrophied, the bony height from the crest of ridge to IAN decreases and the bone height in this area is often not enough to place an implant. Due to the increasing demand of patients for dental implants, strategies have been presented to overcome the obstacle of deficient alveolar bone height. These include guided bone regeneration (GBR), onlay bone graft, inter-positional sandwich bone graft, distraction osteogenesis (DO), all-on-four technique, use of short implants, lateral (or Lingual) positioning of implants and nerve transpositioning. Each of the aforementioned treatment options has its inherent advantages and disadvantages as well as indications and contraindications. In this chapter we discuss nerve transpositioning.

2. Nerve transpositioning

2.1. History

The first case of inferior alveolar nerve repositioning was reported by Alling in 1977 to rehabilitate patients with severe atrophy for dentures [9]. Jenson and Nock in 1987 carried out IAN transposition for placement of dental implants in posterior mandibular regions [10]. In 1992, Rosenquist performed the first case series study on 10 patients using 26 implants. He reported an implant survival rate of 96% for this procedure [11] and therefore, this technique was accepted as a treatment modality for reconstruction of the dentoalveolar system with dental implants in the posterior mandible. Consequently, research studies started to evaluate various surgical techniques developed for this procedure; their advantages, disadvantages, pitfalls and methods for preventing or decreasing complications were presented. As a result, this technique constantly improved. When looking at the history of different treatment modalities and surgical techniques in various academic fields we notice that most of them had limitations and complications at first but significantly improved with time and advancement of technology. Nerve transposition is a young procedure that needs further refinements in terms of technique and instrumentation to decrease complications.

2.2. Anatomy of the inferior alveolar nerve

The inferior alveolar nerve (IAN) is a branch of the mandibular nerve (V3) which is itself the third branch of the cranial nerve V (Figure 1). It runs downward on the medial aspect of the internal pterygoid muscle and passes inbetween the sphenomandibular ligament and the mandibular ramus entering through the mandibular foramen into the inferior alveolar canal innervating the teeth posterior to the mental foramen. At the mental foramen, the IAN divides into two branches namely the incisal and mental nerves (Figure 2). The incisal nerve is often described as the extension of the IAN innervating mandibular canines and incisors by passing through the bone [12].

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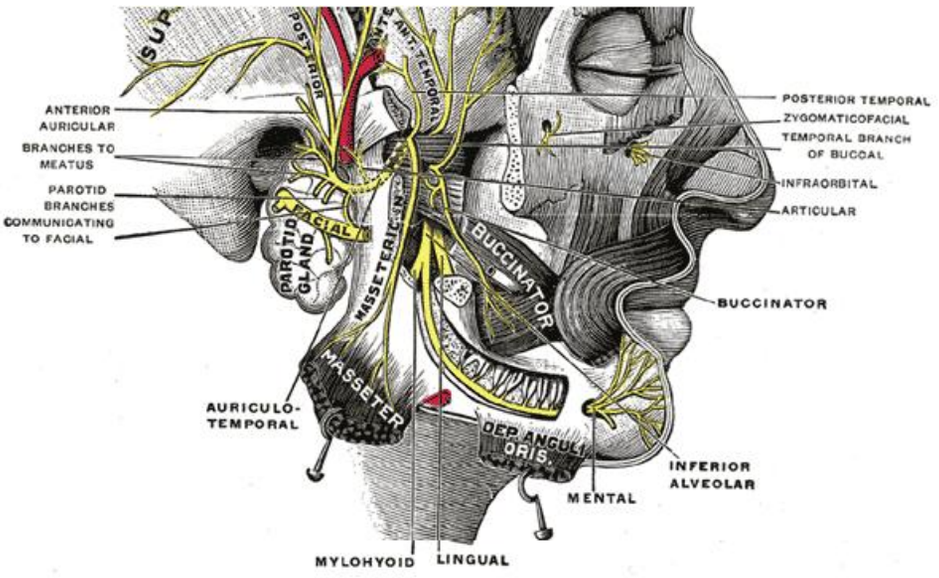

Figure 1. Inferior alveolar nerve path.

The inferior alveolar nerve gives off 3 branches inside the canal: Ramus Retromandibularis, Rami Molares or Molar Branch and Ramus Incisivus or Incisal Branch.

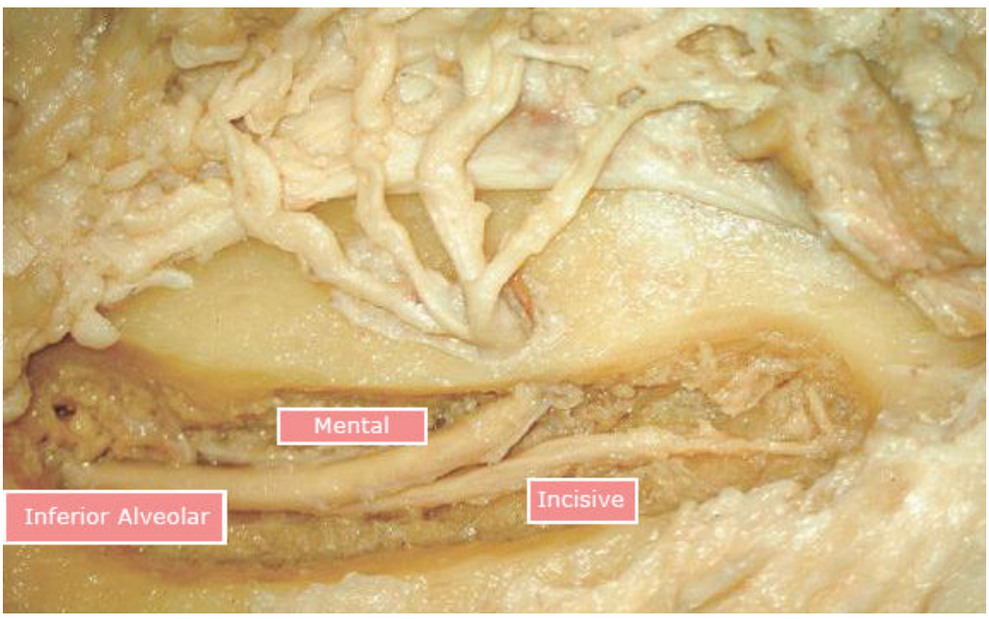

Figure 2. Branching of the inferior alveolar nerve into mental and incisive nerves at the mental foramen.

In some cases, the IAN canal is unilaterally or bilaterally bifid [13,14]. Thus, it is necessary to pay close attention to radiographic and CT examinations before nerve transposition in order to detect such cases and decrease the related risks (Figure 3).

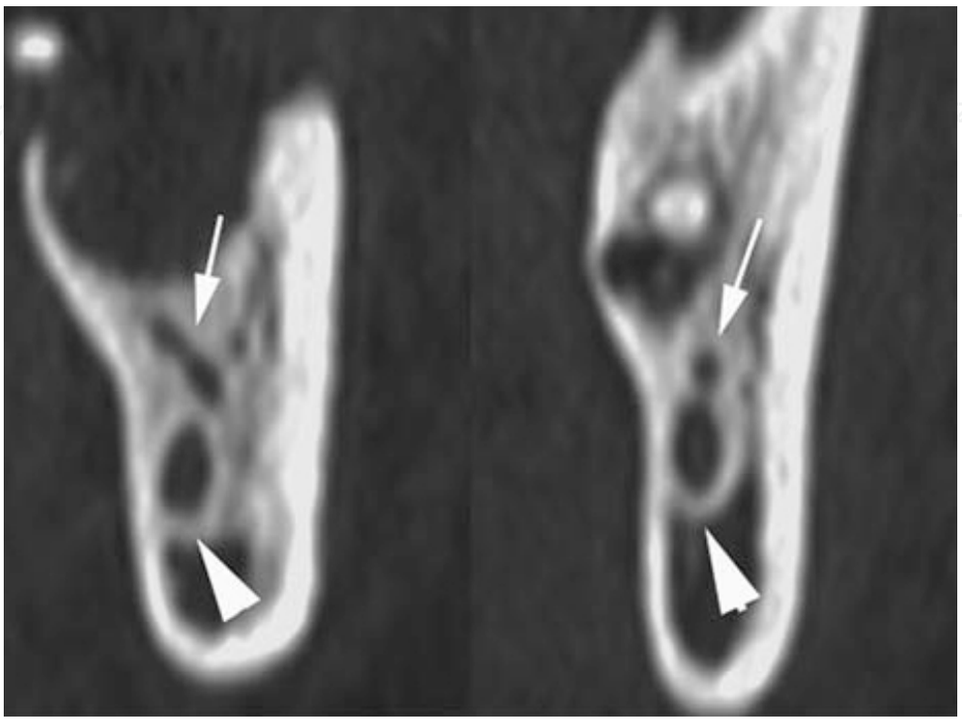

Figure 3. A coronal CT scan of a patient with a bifid mandibular nerve canal

2.2.1. Inferior alveolar nerve canal in edentulous patients

On panoramic radiographs of edentulous patients, the IAN canal in the body of the mandible is not very clear; thus, its path through the ramus and the opaque lines above and below the canal may not be clearly visible. Also, the closer we get to the mental foramen, the less visible the canal becomes [15,16]. Cesar et.al in their studies offered 2 types of classification for the IAN canal in edentulous patients. Vertically, the canal is located either in the upper or in the lower half of the mandible. In 73.7% of males and 70% of females the nerve is located in the lower half of the mandible (therefore, presence of the canal in the inferior half of the mandible is the most common occurrence). Branching of the IAN in edentulous patients falls into one of the following patterns: Type 1: Presence of one single trunk with no branching. Type 2: Presence of a series of separate nerve branches (most common type). Type 3: Presence of a molar plexus. Type 4: Presence of proximal and distal plexuses. Type 2 is the most prevalent pattern where a main trunk along with several single branches is directed towards the superior border of the mandible. The second most prevalent pattern is the presence of a small molar plexus at the proximal half of the IAN or Type 3 (Figure 4) [17].

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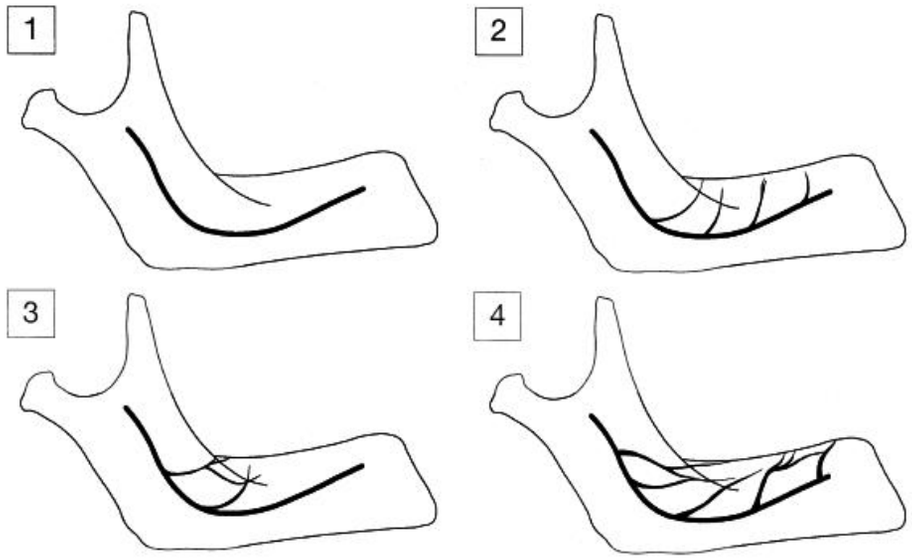

Figure 4. Variations of inferior alveolar nerve types in an edentulous mandible

2.2.2. The mental nerve

The mental nerve emerges at the mental foramen and divides beneath the depressor anguli oris muscle into 3 branches namely a descending branch that innervates the skin of the chin and 2 ascending branches innervating the skin and mucous membrane of the lower lip [13]. The patterns of emergence of the mental nerve at the mental foramen follows 1 of 3 patterns. Knowledge of these patterns is necessary for the surgeon before operating on this area. Type 1: The neurovascular bundle traverses anteriorly and then loops back to exit the mental foramen (anterior loop). Type 2: The nerve runs forward and exits the foramen along the canal path (absence of anterior loop). Type 3: The nerve exits the foramen perpendicular to the canal axis (absence of anterior loop). Type 1 is the most common pattern (61.5%) followed by type 2 (23.1%) and type 3 (15.4%) [18].

2.2.3. Contents of the mandibular canal and their location

Placing implants in areas adjacent to the IAN has increased significantly. Therefore, it is extremely important to know the contents of the canal and the exact location of components of the neurovascular bundle. According to histological examinations and MRI imaging, the inferior alveolar artery is located coronal to the nerve bundles inside the canal. Before entering the mandibular foramen, the artery is located inferior and posterior to the nerve. After entering the canal it changes its path at the mid length of the canal and runs superior and slightly medial to the nerve [18-20].The IAN usually has a round or oval cross section with a mean diameter of 2.2 mm. The mean diameter of the artery is 0.7 mm. The mean closest distance of the artery to a tooth apex is about 6 to 7 mm at the second molar area [20]. Yaghmaie et al. in 2011 confirmed the presence of lymphatic vessels in conjunction with the nerve trunks and blood vessels in all directions [21].The neurovascular bundle and its branches are responsi-

ble for sensation of pain, temperature, touch, pressure and proprioception of their innervated areas. The nerve is comprised of 1 or multiple fascicles. A collection of nerve fibers forms a fascicle. Microscopic examination of neurovascular bundles usually shows 2 to 8 axon bundles. Each fascicle contains about 500 to 1000 nerve fibers. Epineurium wraps around the fascicles, protects them and contains blood vessels for nutrition (Figure 5) [18-20].

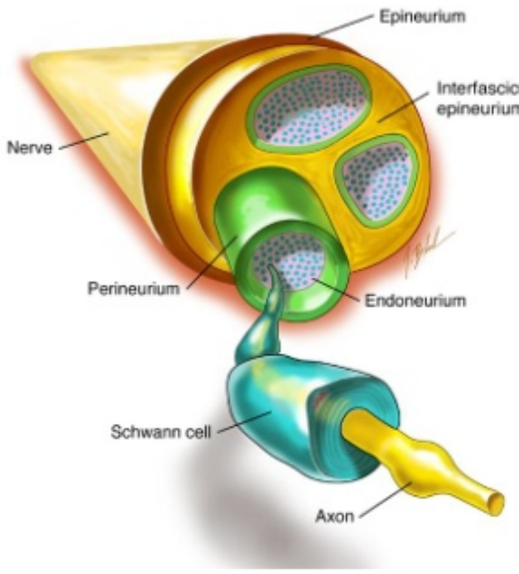

Figure 5. Schematic cross-section of the nerve. Nerve fascicles and fibers can be seen. Components in an orderly fashion from the outermost layer to the inner most include epineurium, perineurium, endoneurium and Schwann cells surrounding the axon.

2.2.4. Fascicular patterns

There are 3 fascicular patterns: The mono-fascicular pattern includes one big fascicle along with perineurium and epineurium layers surrounding it (i.e. the facial nerve). The oligo-fascicular pattern includes 2 to 10 fascicles each covered by perineurium. Fascicles are interconnected through the epineurium layer inbetween them; in this pattern, fascicles are usually of the same size (nerve roots C6 and C7 have the oligo-fascicular pattern). The poly-fascicular pattern includes more than 10 fascicles of various sizes i.e. inferior alveolar and lingual nerves (Figure 6) [18-20].

As mentioned earlier, the IAN has a poly-fascicular pattern. The outer nerve fibers of the bundle are called “mantle bundle”. They usually innervate the proximal areas (molars). Following the administration of local anesthesia, this area is affected sooner and more efficiently since it is close to the side of the nerve bundle; whereas, core bundles innervate distal areas (central and lateral) and are affected later and less efficiently by local anesthetics. Various senses are affected when administering local anesthetics depending on the nerve diameter and presence or absence of a myelin sheath. For instance, signal transmission is slower in thinner non-myelinated nerve fibers. These fibers are affected more efficiently and more quickly by the local anesthetics than large diameter, myelinated fibers that have faster signal

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transmission. Non-myelinated fibers (sympathetic C fibers responsible for vascular tonicity and slow transmission of pain) and partially myelinated fibers (A delta fibers, fast transmission of pain) are affected sooner by the local anesthetics and also return to their normal state more quickly. On the contrary, thicker myelinated fibers (like A alpha and A Beta) that transmit deep sensations, pressure and proprioception are affected by local anesthetics later. In conclusion, general senses are affected clinically by the local anesthetics in the following order: First cold sensation through the autonomic nerves, then heat, pain, touch, pressure, vibration and eventually proprioception. Contents of the canal are responsible for innervation of dental pulps, periodontium, dental alveoli and soft tissues anterior to the mental foramen. Dental pulps receive unmyelinated sympathetic nerve fibers from the superior cervical trunk which enter the pulp accompanied by arterioles. Dental pulps also receive A delta myelinated sensory nerve fibers as well as unmyelinated nerve fibers (both from the trigeminal ganglion); together they form a large plexus below the odontoblastic layer in the pulp (Raschkow's plexus). In the Raschkow's plexus myelinated fibers lose their myelin sheath and penetrate into the odontoblastic layer. Today, they consider the phenomenon of fluid mobility inside the odontoblastic tubes (hydrodynamic theory) to be responsible for stimulation of nerve endings and sensing pain [12]. There are 2 aspects in the sensation of pain namely, a physiologic aspect and a psychological aspect which together create the unpleasant psycho-physiologic and complex experience of pain. From the physiologic point of view, stimulation of specific nerves (like A delta and C fibers) and transmission of the signal to the trigeminal ganglion is called "transduction". Passing over the signal from this site to upper centers (thalamus and cortex) is called "transmission" and "modulation". The three mentioned pathways comprise the physiologic aspect of pain that combined with the psychological aspect (previous experience, cultural behaviors, psychological state and medical status) create the unpleasant complex experience of pain [12].

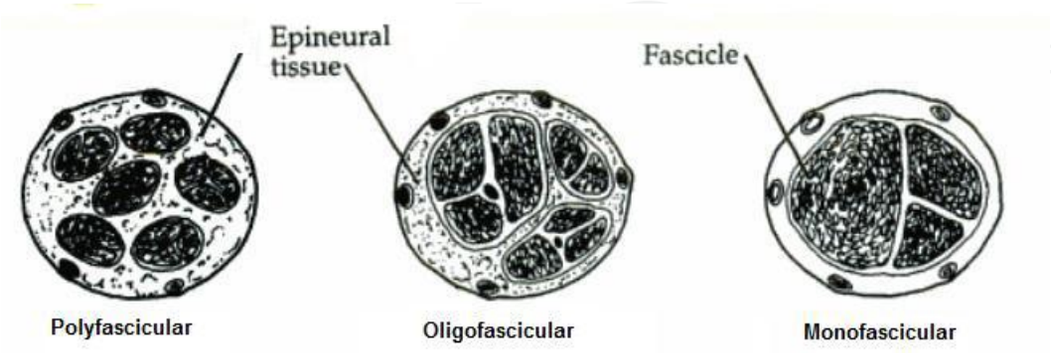

Figure 6. The three fascicular patterns. From right to left: Mono-fascicular, oligo-fascicular and poly-fascicular

2.2.5. Inferior alveolar nerve injury

Various factors can traumatize the IAN ranging from simple accidents like trauma from a needle during injection, bleeding around the nerve and even the local anesthetic drug itself, to maxillofacial traumas, pathologic lesions and surgical operations. Generally, the main nerve injuries are usually due to trauma or surgical operations among which, the most frequent ones are surgical extraction of mandibular third molars, endodontic treatment, implant placement, osteotomies (visor, sagittal, body of the mandible and subapical osteotomies), genioplasty, resection of mandibular cysts and tumors, partial mandibulectomy, fracture of the angle, ramus or body of the mandible, D.O. and IAN transpositioning. The nerve trunk is composed of 4 connective tissue sheaths. These membranes from the outermost to the innermost include mesoneurium, epineurium, perineurium and endoneurium. The mesoneurium suspends the nerve trunk within the soft tissue and contains vessels. The epineurium is a dense irregular connective tissue that protects the nerve against mechanical stress. The larger the epineurium (it usually measures 22 to 88% of the nerve diameter), the higher the nerve resistance against compressive forces compared to tensile forces. It should be mentioned that most nerve injuries are usually of a transient nature and will recover partially or completely. Epineural tissue wraps around nerve bundles and protects them against mechanical stress. Also, in many cases pressure due to severe inflammation or retention of fluid around the nerve trunk and subsequent development of transient ischemia in the epineurium cause clinical symptoms of neural dysfunction and disturbances. It is worth noting that the IAN is a poly-fascicular nerve. The smaller the number of nerve fascicles and the thicker the epineurium the more resistant the nerve is to pressure and vice versa (the greater the number of fascicles and the thinner the epineurium, the less resistant the nerves are towards pressure)[12,21-23]. It should be mentioned that poly-fascicular nerves like the IAN have a large number of small fascicles and therefore are more resistant to tensile forces compared to mono-fascicular or oligo-fascicular nerves [22].Perineurium wraps around the axon, Schwann cells and endoneurial sheath ; each nerve fiber is covered by the endoneurium sheath. Schwann cells are necessary for the axon to stay alive. They are the most sensitive cells to ischemia and radiation [12] (Figure 5).

2.2.6. Classification of nerve injury

There are 2 classifications available for nerve injury. The first was introduced by Seddon in 1943. He classified nerve injury into 3 types: Neuropraxia, Axonotmesis and Neurotmesis (from minor to major injury)[24]. The other classification was described by Sunderland [25] in 1987. He categorized 5 degrees of nerve injury : First degree where the axon and the covering sheath are intact. Epineural ischemia is probably the cause of the conduction block. Recovery is usually complete. Second degree where the axon is injured but endoneurium, perineurium and epineurium are intact. Recovery is often satisfactory. Third degree where the axon is injured but endoneurium is disrupted. However, of recovery. Fifth degree where there is complete transection with loss of continuity and less chance of spontaneous epineurium and perineurium are intact. Partial recovery may be achieved. Fourth degree where the axon, endoneurium and perineurium are all interrupted. However, epineurium is intact. There is a small chance recovery. Microscopic surgery is recommended (Table 1) [23].

Classification	Cause	Response	Recovery	Microscopic surgery
Neuropraxia(Sedd.) Grade 1 (Sunderland)	Compression, traction, small burn, acute infection	Neuritis, paresthesia, conduction block, no structural damage	Spontaneous recovery in less than 2 months	Not necessary unless a foreign body interrupts the process of nerve repair
Axonotmesis (Sedd.) Grade 2 (Sunder.)	Partial crushing, traction, burn, chemical trauma, hematoma, chronic infection	Intact epineurium, isolated axon loss, episodic dysesthesia	Spontaneous recovery within 2-4 months	Not indicated unless for decompression due to a foreign body or perineural fibrosis
Grade 3 (Sunder.)	Traction, crushing, contusion, burn, chemical trauma	Wallerian degeneration of axon, some internal fibrosis, peripheral pain	Poor sensory recovery, neuropathy for more than a year	Decompression and repair in case of poor function and continuous pain for 3 months
Grade 4 (Sunder.)	Complete crushing, severe traction, severe burn, direct chemical trauma	Neuroma-in-continuity, hypoesthesia, triggered hyperpathia	Permanent damage, minimal spontaneous recovery	Repair, resection of neuroma in case of unbearable pain after 3 months
Neurotmesis (Sedd.) Grade 5 (Sunder.)	Transverse incision on the nerve, laceration, laceration of the main nerve trunk	Neuroma at the site of incision, anesthesia, evolving deafferentation pain	Permanent damage, low spontaneous recovery	Resection of neuroma by neurorrhaphy or graft in case of poor function and lasting neuropathic pain

Table 1. Classification of nerve injury (Comparison of Sunderland and Seddon classifications)

2.2.7. Nerve changes following injury

Changes in central nervous system (CNS): The onset of such changes is 3-4 days or maximally 10-20 days after the injury. The neurons are in an anabolic state of protein synthesis. In humans, this can continue for years. The more proximal the location of injury, the higher the metabolic demand of the neuron. If the neuron is unable to supply such demand, cell death will occur. The best time for surgical repair when necessary is within 14 to 21 days after injury. After regeneration, the neuron gradually returns to its normal size and function.

Changes proximal to the site of injury: About an hour after trauma, a swelling develops within 1 cm proximal to the site of injury causing the area to enlarge up to 3 times its normal diameter. This swelling stays for a week or longer and then gradually subsides. On day 7, the proximal axon stump sprouts buds. These buds usually develop within a few millimeters distance from the site of injury from an intact node of Ranvier directed towards the dis-

tal end of the nerve. They cross the lesion on day 28, reconnect with the distal portion on day 42 and grow into it and advance (unless fibrous or scar tissue has formed). The more proximal the location of the injury, the longer it takes for a sprout to cross the lesion as the result of a more extensive inflammatory reaction.

Changes at the site of injury: During the next few hours after injury, proliferation of macrophages, perineural and epineural fibroblasts and Schwann cells occurs. On days 2 and 3, cell proliferation is seen proximal and distal to the site of injury. On day 7, Schwann cells play the major role. Fibrosis at the site of injury and imperfect positioning of regenerative fibers can result in formation of a neuroma.

Changes distal to the site of injury: Wallerian degeneration is the major characteristic of such changes in this area preparing the location for axonal sprouts budding out from the proximal stump. Death of all cellular components distal to the injury site is the key initiating event for Wallerian degeneration. On day 7 post-injury, the majority of cells at the distal portion disintegrate. This process is facilitated by the action of enzymes. By day 21, most cellular debris is engulfed and phagocytosed by Schwann cells. This cellular debridement is usually completed by day 42. Endoneurial tube becomes smaller, shrunken or even obstructed due to cell proliferation and excessive collagen formation. Its diameter is decreased by 50% after 3 months and only 10 to 25% of its primary diameter may be left open after 12 months. This phenomenon is called distal atrophy when the entire nerve trunk distal to the site of injury is shrunken and atrophied. Tubules formed by Schwann cells and surrounded by collagen guide the sprouts distally. Although the number of sprouts is various and may be up to 4 times the normal number, during regeneration volume and number of sprouts decrease and the final number will end up to be smaller than the original number and the diameter of the new axon will be smaller as well. When a sprout reaches the distal tube, the metabolic activity of the Schwann cells increases again and myelin is reproduced by the Schwann cells. However, the quality of the newly formed myelin is not as good as the quality of the primary myelin. The new axon has a smaller diameter and is placed in thinner endoneurial tubes. The new myelin is not similar to the old one. The nodes of Ranvier are shorter and therefore cause a decrease in nerve conduction velocity in this area. Axon regeneration speed is different in various circumstances but it is on average 1 to 3 mm a day.

Changes in the target organ: At the target sensory organ, receptors suffer from progressive deformities but following reinnervation even after several years the target organ will have no sensory impairment or disturbances. For skin flaps as well, reinnervation resumes pain, temperature and touch sensations perfectly. For target motor organs however, reinnervation of the respected muscle does not occur even 12 months after nerve transection. This is not because of changes in neuromuscular junction end plate but probably due to irreversible interstitial fibrosis in muscle fibers [13].

Clinical examination of sensory impairment of the lower lip following IAN injury:

Before discussing the clinical examinations, we explain the definition of common clinical terms (Table 2).

Anesthesia	Absence of any sensation
Paresthesia	Abnormal sensation even spontaneously or for no reason
Analgesia	No pain in response to a normally painful stimulus
Dysesthesia	An unpleasant abnormal sensation that can be spontaneous or for a reason
Hyperalgesia	Hypersensitivity to harmful stimuli
Hyperesthesia	Hypersensitivity to all stimuli except for special senses
Hypoalgesia	Decreased sensitivity to stimuli
Hypoesthesia	Decreased sensitivity to all stimuli except for special senses
Allodynia	Pain due to a stimulus that does not normally cause pain
Neuralgia	Pain that is distributed in one or several nerve fibers
Deafferentation pain	Pain due to decreased sensory afferents into the CNS
Neuropathic pain	Pain due to a primary lesion or nervous system dysfunction
Causalgia	Burning pain immediately or several months after injury
Anesthesia dolorosa	Pain felt in an area which is completely numb to touch
Synesthesia	Stimulation of one sensory or cognitive pathway leads to experience in a second cognitive or sensory pathway due to misdirected axonal buds resulting in misperception of the location of touch or pain
Central pain	Pain due to a primary lesion or central nervous system dysfunction
Hyperpathia	A painful syndrome characterized by hyper-responsiveness to a stimulus. Hyperpathia may be associated with hyperesthesia, hyperalgesia or dysesthesia

Table 2. Frequently used terms during clinical examination of neurosensory disturbances

2.2.8. Clinical tests

Static light touch: For this test a bunch of nylon filaments with same length and different thickness mounted on a plastic handle is used. The patient closes his eyes and says "yes" whenever he feels a light touch to the face and points to the exact spot where he felt the touch. Brush directional discrimination: For this test, the finest nylon filaments from the previous test or a brush with more filaments are used. The patient states if any sensation is detected and in which direction the filament or brush moved. **Two point discrimination**: In this test the distance between two points is altered. With the patient's eyes closed the test is initiated with the points essentially touching so that the patient is able to discriminate only one point. **Pin pressure nociception**: For this test the most common instrument is the algesimeter which is a simple instrument made from a no.4 Taylor needle and an orthodontic strain gauge. The sharp point of the needle is used to test nociception and the blunt end to test for pressure detection and hyperpathia. The needle is placed vertically on the skin. The pressure is increased every few seconds until the patient feels the sharpness (usually with 15 to 25 g) and then the needle is gently removed. The same is done for the affected area as well. No response to pin pressure up to 100 g is defined as anesthesia. An exaggerated response to pin pressure relative to an unaffected area is defined as hyperalgesia and a reduced response to touch relative to an unaffected area is considered as hypoalgesia. **Thermal discrimination**: This is an adjuvant test and is not es-

sential. Minnesota Thermal Disks are the most common instruments used for this assessment. Ice, ethyl chloride spray, acetone, and water are also used. The simplest method is to use an applicator dipped into acetone or ethyl chloride. When pain is a symptom of nerve injury, diagnostic nerve blocks using local anesthesia can be very helpful in deciding whether or not micro-reconstructive surgery is indicated. It is important to start with low concentrations of anesthetic drug. Injections should be performed starting from the periphery towards the center to ease the pain. If the pain is not alleviated there is a chance that collateral sprouts from the other side are present. If the persisting pain is aggravated by cold, is spontaneous, and of burning type and long lasting, then allodynia, hyperpathia, causalgia and sympathetic pain should considered in the differential diagnosis. In such cases, diagnostic stellate ganglion block is helpful in differentiating causalgia from sympathetic pain [10,12,24]. There are various causes of pain following traumatic nerve injury including nerve compression, neuroma, anesthesia dolorosa, causalgia and sympathetic pain, central pain and deafferentation, nerve laceration, nerve ischemia and chemical stimulation.

Clinical and radiographic evaluation. For clinical assessment of a patient who is a candidate for dental implants and suffers from atrophic mandibular alveolar ridge should first prepare study casts and then the occlusal relationship should be recorded. The following points should also be considered:

The area of the edentulous atrophic alveolar ridge: If the edentulous area extends interiorly up to the canine the surgeon should consider mental nerve transpositioning.[1]. In edentulous patients, absence of incisal sensation following nerve distalization does not cause problems but in patients with incisal teeth this can result in an unpleasant sensation in the anterior segment which is usually described as a sense of dullness in these teeth. The distance between the occlusal surface of maxillary teeth and mandibular alveolar ridge. In some cases, despite alveolar ridge resorption there is not enough space between the occlusal surface of the maxillary teeth and the mandibular ridge which is required for placing the implant prosthesis. It is usually due to the patient's previous occlusion (mainly in deep bite cases) or over-eruption of the opposing teeth. Augmentative methods often cannot be used (Figure 7]. In such cases, the only available option seems to be nerve transpositioning [3,22,26].

Evaluation of the relationship between the mandibular alveolar ridge and maxillary alveolar ridge in the horizontal plane: The necessity of lateral augmentation simultaneous with nerve transposition or vertical augmentation should also be evaluated by clinical examination and study of the patient's casts.

Radiographic evaluation: Every patient who is a candidate for nerve transposition is required to obtain panoramic radiography and CBCT scans (Figure 7).

The length of bone above the canal, anomalies, distance of the canal from the buccal cortex and also thickness of the cortex for ostectomy are all evaluated on panoramic radiography. Exact location and precise anatomy of the mental foramen and anterior loop can also be evaluated [27]. In rare cases, the IAN canal may be completely attached to the medial or lat-

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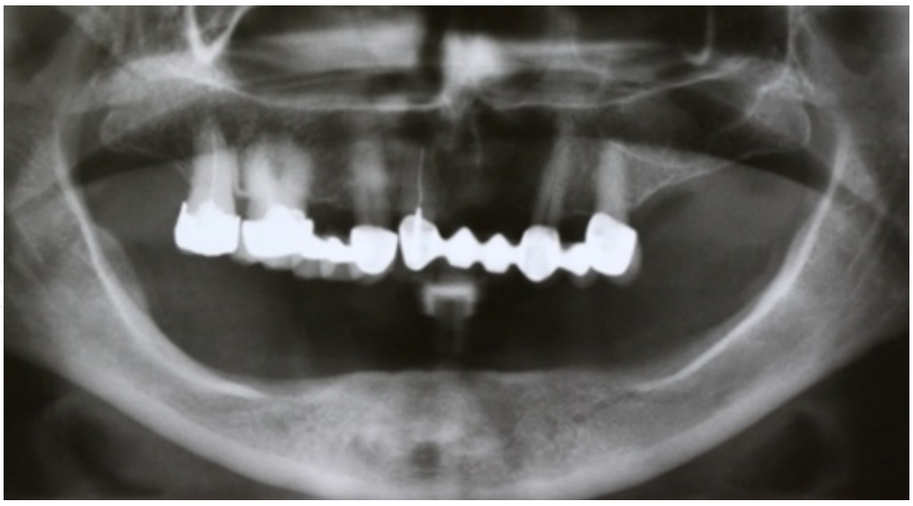

Figure 7. Panoramic radiography of an atrophic posterior mandible. Note the inadequate length of bone over the canal for implant placement.

eral cortex on CBCT. In such cases, implants can be easily placed buccally or lingually to the canal with no need for extensive surgery. Additionally, by analysis and reconstruction of scanned images using CAD-CAM, it is feasible to determine the path of the canal and place the implants in atrophic areas.

2.3. Indications, contraindications and limitations

Babbush mentioned several indications for nerve transpositioning; namely placement of removable prosthetics, stabilizing the remaining anterior teeth, stabilizing the temporomandibular joint, and establishing muscular balance following reconstruction of the dentoalveolar system. He also discussed some related limitations. This procedure is technically difficult and requires adequate expertise. The surgeon should have adequate experience, sufficient anatomical knowledge and necessary skills to fully manage peri-operative and post-operative complications. Accordingly, the most significant risk of surgery is nerve injury due to surgical manipulations and the surgical procedure itself. Although rare, mandibular fracture should also be considered as a risk factor especially in cases with severe mandibular atrophy (Figure 8) [28].

Resenquise et al. in their studies on nerve transpositioning procedure mentioned the following indications and contraindications for this operation:

Indications: Less than 10-11 mm bone height above the canal, when the quality of the spongy bone does not provide sufficient stability for implant placement

Contraindications: Height of bone over the canal is less than 3 mm. The patient has thick cortical bone buccally and thin neurovascular bundle. The patient is susceptible to infection or bleeding. Limitation in accessing the surgical site [9-11,29,30]

According to author's personal experience, nerve transpositioning in cases where the bone height over the canal is less than 3 mm is still feasible. We can transpose the nerve from the

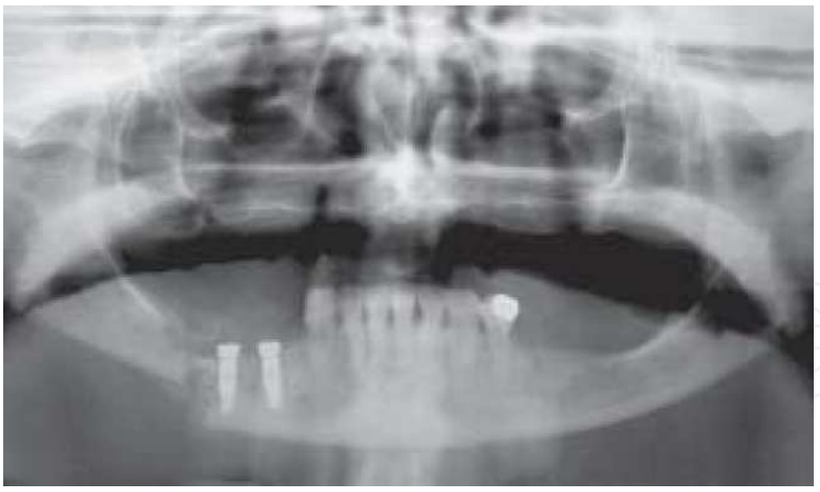

Figure 8. Mandibular fracture in a patient with severe mandibular atrophy following nerve transpositioning.

alveolar crest laterally, and after placing the implant with bone graft material. More details in this regard will be discussed subsequently.

2.4. Surgical procedure of nerve transpositioning

Pre-operative consultation: Before choosing nerve transpositioning, we should first scrutinize the required criteria. According to the literature, 100% of patients who undergo nerve transposition develop various degrees of sensory nerve dysfunction of the lips. Therefore, the patient and his/her family members should be well informed relevant to the phases of treatment, duration of surgery, post-operative general complications and most importantly provided with knowledge about the post-operative lip paresthesia which will definitely occur and may last for up to 6 months and in some cases it lasts longer or is very severe may require microscopic surgery [10,31-33]. Despite the above mentioned explanations, the patient may not fully comprehend what paresthesia actually feels like. In such cases, we recommend performing an inferior alveolar nerve block for the patient using bupivacaine for anesthesia so that the patient can experience anesthesia and paresthesia for 8 to 12 hours. We should also explain the advantages of this treatment modality for the patient including shorter treatment duration, no need for autogenous bone grafts and no donor site morbidity, minimum use of bone replacement material and obviating the need for additional surgery [9,10,33].

2.4.1. Technique

Inferior alveolar nerve transpositioning for implant placement is usually performed by 2 techniques: **IAN transpositioning without mental nerve transpositioning or involvement of mental foramen:** This is usually employed when the edentulous area and alveolar ridge resorption does not include the premolars. This technique has been called nerve lateralization in some articles (Figure 9-12 A). **IAN transpositioning with mental nerve transpositioning or involvement of mental foramen:** In cases where the edentulous area and ridge

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resorption include the premolar teeth: there is a need for transpositioning of mental neurovascular bundle and even transection of incisal nerve and transposing the nerve distally (associated with mental nerve and mental foramen involvement). This method has also been called nerve distalization by some [1,9,28,34] (Figure 12 B).

Phases of surgery: Nerve transpositioning can be performed under local anesthesia alone, local anesthesia along with sedation or under general anesthesia based on the patient's condition. Local anesthesia includes inferior alveolar nerve block plus local infiltrating anesthesia in the form of lidocaine plus vasoconstrictor at the buccal mucosa. 1-Incision is made on the alveolar crest starting from the anterior border of the ramus forward. At the mesial surface of the mandibular canine a releasing incision is made anteriorly and towards the vestibular sulcus in order to avoid injuring mental nerve branches. In cases where the treatment plan includes placement of dental implants in the same surgical step, soft tissue incision should be made in a way that part of keratinized gingiva is placed in the buccal and part of it on the lingual side of the healing abutment (Figures 10 and 11) [1,31-35].

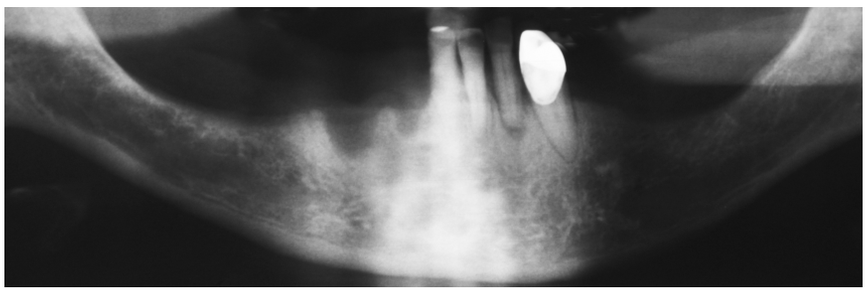

Figure 9. A patient with edentulous posterior mandibular region along with bone resorption who is a candidate for nerve transposition surgery.

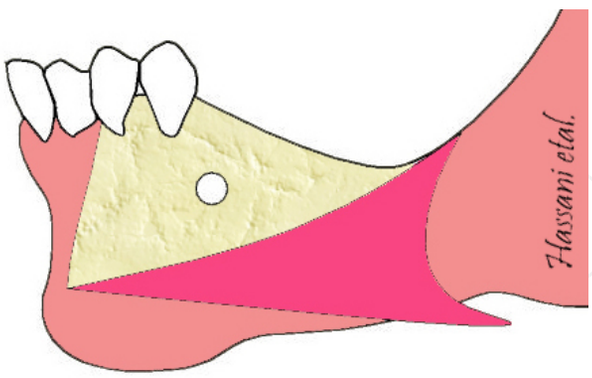

Figure 10. Flap design: An incision is made on the alveolar crest along with a releasing incision at the mesial of mandibular canine.

2-Retracting the mucoperiosteal flap is done so that the mental foramen is totally exposed and the dissection is extended towards the inferior border. Considering the radiographic and CBCT evaluations along with the fact that the neurovascular canal is usually located 2 mm below the mental foramen, it is necessary to expose the lateral surface of the body of the

mandible and release the periosteum around the mental nerve (Figure 10) [1,36]. 3-Bone removal on the lateral surface of the canal is done while preserving the maximum thickness of buccal bone as this especially important. Presence of adequate bone thickness in this area results in better and faster healing of the bone defect adjacent to the implant where nerve transposition has been performed. Bone can be removed using a diamond round bur or piezosurgery device [1,6].

In the first technique which is usually performed for treatments other than dental implants a piece of bone is removed as a block and then the canal is exposed. This method can be indicated for simultaneous implant surgery when there is adequate bone height over the canal. In such cases, even after resecting a bone block, a sufficient amount of bone still remains at the lateral side of the implant [26]. Rosenquist reported that in this method, it is difficult to maintain a proper angulation when placing the implant because a great extent of buccal bone has been removed for nerve transposition and accessing the canal [30]. In patients who are candidates for implants, cortical bone preferably should not be removed as a block because in such patients there is limited amount of bone available in the superior and lateral sides of the canal which should be preserved. If the surgical technique does not include manipulation of the mental nerve, bone is removed using a round bur number 700 or 701, a straight handpiece and copious normal saline for irrigation or a piezosurgery device. Bone removal is initiated 3-4 mm distal to the mental foramen and follows the canal path posteriorly and superiorly. Bone removal should extend 4-6 mm posterior to the intended location of the last implant. We should try to remove the smallest amount of bone possible from the buccal cortex. Excessive bone removal along with extensive drilling for implant placement can result in temporary mandibular weakening followed by increased risk of mandibular fracture which has been reported in the literature. Bone preservation helps in primary and final implant stability and shortens the recovery time. After removing the cortical bone, a curette may be used for removal of spongy bone and cortical layer of the canal in cases where the cortical layer surrounding the canal is not dense or thick. A special instrument (Hassani nerve protector) is required to protect the nerve while the cortical layer has to be removed using surgical burs or piezosurgery device. Bone removal in close vicinity to the neurovascular bundle should be performed patiently and thoroughly. This is usually performed using special curettes parallel to the surface of nerve bundles in an antero-posterior direction. Tiny bone spicules around the nerve should be removed. The area should be thoroughly irrigated so that the nerve bundle can be clearly seen (Figure 11 A - D) [1,2,4,9,10].

Another method that has been suggested is drilling the bone surrounding the canal using a hand piece and a round bur. The surgeon carefully enters a probe (round end with no sharp edge) into the canal through the mental foramen and determines the canal path. Then according to this test and after evaluating the canal path on the radiographs, the surgeon inserts the tip of the nerve protector into the canal. This instrument has been designed, patented and manufactured by the author (Hassani nerve protector). This instrument should be placed in between the nerve and the bone in order to protect the nerve. The buccal bone is drilled using a bur. By directing the bur distally, the nerve protector is also moved distally inbetween the nerve and bone to protect the nerve at all times. The bone chips are collected

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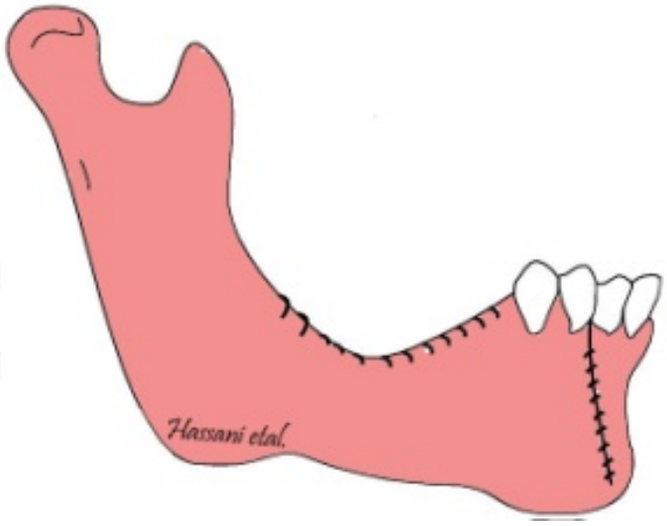

Figure 17. Gingival flap is put back in its location and sutured.

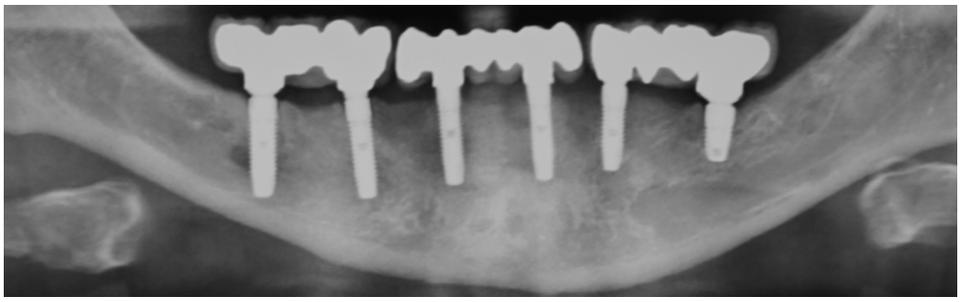

Figure 18. Same patient in Figure 16; Two years after loading the implants. Note the bone level. (Implantium Implants, Dentium Co.)

In patients with an atrophic alveolar ridge involving the premolar area or those with an edentulous mandibular ridge along with alveolar crest atrophy who need implant placement IAN transposition in the posterior mandible and mental nerve transposition is also necessary most of the time. This transposition is usually associated with incisal nerve transection. In such cases, the patients will not have any problems related to incisal nerve transection but in cases where transposition of the nerve is intended and the patient has vital anterior mandibular teeth, nerve transection may result in patient having an unpleasant sensation in these teeth. In some cases, even root canal therapy may be required. However, several studies have reported that no problems related to anterior mandibular teeth were seen [1,9,35].

Sectioning the incisal branch of the inferior alveolar nerve, releasing the neurovascular bundle and moving it backwards in order to avoid traction is called nerve distalization [9]. Based on the author's experience, in many cases it is possible to transpose the mental nerve without sectioning the incisal nerve. In the method of nerve transposition without releasing the mental nerve, great traction force is exerted on the nerve when keeping it out of the surgical site. According to the literature, the highest number of nerve injuries occurs during an-

terior osteotomy because the nerve trunk becomes thinner at mental foramen and is therefore more susceptible to injury. That is why nerve transposition without involving the mental foramen has the least sensory complications and side effects. According to the literature, by preserving 3-4 mm bone distal to the mental foramen during nerve transposition we can reduce inferior alveolar nerve damage because the nerve is thinner and more susceptible to injury at this specific location [32].

Vasconcelos et al. believes that at least 5 mm bone height above the canal is necessary in case selection for nerve transposition whereas, Kahnberg and colleagues believe that 2 to 3 mm bone thickness above the canal is adequate [9,10]. In cases where minimum requirement of bone height above the canal does not exist some authors suggest to do a bone graft before nerve transposition and implant placement [9]. However, fixing the grafts especially blocks of autogenous bone to the limited remaining bone above the canal is difficult and is associated with a risk of nerve injury by the screws. Based on the author's experience in such cases we can transpose the nerve from the alveolar crest laterally. Bone is removed from the alveolar crest, and when the nerve is exposed we move it upward and outward and start drilling for implant placement from inside the canal while the nerve is retracted laterally from the buccal cortex. Bone graft is placed inside the canal anterior and posterior to the implant. The nerve is placed into a newly formed groove from the posterior area of the last implant (Figure 19).

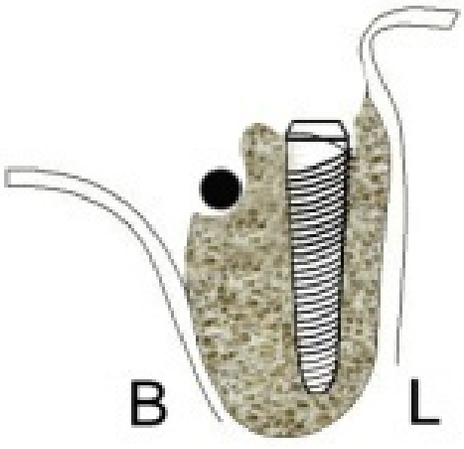

Figure 19. The IAN is located at the alveolar crest following ridge atrophy. The nerve is removed from the crest, implant hole is prepared from inside the canal, the implant is positioned and finally the nerve is repositioned in the lateral cortex of the mandible.

Histological findings associated with nerve transposition and implant placement. Yoshimoto and colleagues evaluated the condition of tissues surrounding the implant 8 weeks after nerve transposition surgery and simultaneous implant placement; they observed that none of the implants were exposed and all were perfectly stable. No infection or inflammation was observed at the site. In all cases bone formation between the implant and neurovascular bundle was observed and no direct contact was seen between them.

Research demonstrates that bone formation around the implant surface sand blasted with aluminum oxide was 2.5 times greater than a smooth titanium surface. Bone formation around the neurovascular bundle prevents the implant from having direct physical contact with the bundle and therefore the nerve structure will be protected from mechanical or thermal trauma. Microscopic sections show the formation of a vascular network in the adjacent tissues which proves that there is no need for placing a barrier or any kind of graft material to separate the nerve from the implant [35].

In Kahnberg et al. study on a dog, healing was not complete after 14 weeks but none of the implants were exposed. Histological examination showed that in cases where membrane had not been placed a small contact was present between the nerve bundle and the implant. Plasma cells, macrophages, polymorphonuclears, and granulocytes were alternately seen next to the membrane. Several giant cells and macrophages were also seen. Vascular buds were seen where membrane had been placed (compared to areas where no membrane had been used). In some cases, a capsule with less than 10 μm thickness was seen in some areas between the implant and the nerve. When membrane is used the distance between the nerve bundle and the implant will be 4 to 8 times greater. The mean distance between the implant and the nerve was 348.3 μm when using a membrane and 39.8 μm when not using it. There is no contact between the nerve and the implant when using a membrane but the bone was not seen around the implant either [38].

2.5. Important considerations in nerve transposition surgery

2.5.1. Patient selection

The surgical process is complicated and occurrence of sensory disturbances is definite. Therefore, the surgeon should evaluate the patient's mental condition. Some people are stressed out and over sensitive even towards the smallest surgical complications. Such patients do not have tolerance and compatibility skills and therefore are not good candidates for nerve transposition surgery. Providing data and acquainting the patient with phases of surgery and probable complications: Thorough explanation should be provided for the patient in an understandable and comprehendible manner regarding surgical and neural complications. The sense of anesthesia that may occur should be well described for the patient and it also should be mentioned that the anesthesia may be permanent and irreversible.

1. CBCT should be obtained for precise evaluation of the canal and bone thickness around it.
2. Dexamethasone should be administered before the surgery
3. The surgeon should have full knowledge regarding anatomy and physiopathology of nerve injury and be able to evaluate the clinical course of nerve dysfunction after the surgery.
4. The surgeon's skill and expertise are very important and magnification loops should be used.

5. Delicate instruments required for this type of surgery should be available (for minimal injury). Also, the surgeon should have the knowledge and skills for repairing the nerve in case serious damage is done to the nerve during surgery.
6. In cases where the canal is located in the center or lingually on CBCT, the surgeon should expect a more complex surgery.
7. In cases where the nerve transposition surgery extends further posterior and involves the 2nd molar area, the surgery can be more complicated due to the thicker cortical bone and limited access to the area.
8. Using low level laser after surgery reduces the inflammation and improves recovery.
9. The surgeon should be familiar with and have adequate skills regarding nerve reconstruction surgery and the instruments required for it.

2.5.2. Post-operative measures

Antibiotic therapy and administration of analgesics and NSAIDs post-operatively are similar to that of implant surgery and there are no specific recommendations in this regard in the literature. Antibiotic and corticosteroid prophylaxis is recommended because of the extensiveness and duration of surgery. Using corticosteroids pre- and post-operatively helps in decreasing the symptoms. However, there is no consensus in this regard but since inflammation can be among the causes of nerve dysfunction, corticosteroid therapy can be beneficial.

The most common sensory complications following nerve transposition are hypoesthesia, paresthesia and hyperesthesia. The most common causes of nerve dysfunction include the mechanical trauma to the nerve and ischemia following extracting the bundle from the canal, nerve traction during surgery, edema and probable hematoma and or chronic compression after the surgery [9,10]. According to Hirsch and Branemark, the main cause of sensory disturbances is nutritional impairment of the nerve due to injury to the microvascular circulation of nerve fibers as the result of mechanical trauma. Thermal and pain sensation nerve fibers are more resistant to compressive traumatic forces and ischemia than larger fibers responsible for touch sensation [1]. Therefore, great attention should be paid during and after surgery to minimize the factors responsible for ischemia and mechanical trauma such as;

1. Avoiding exerting too much traction upon the nerve and when lateralizing the nerve and during nerve transposition, try to transform the contact point to a contact area.
2. During ostectomy care must be taken not to injure the nerve with rotary instruments, curette or elevator. When removing the bone cortex over the nerve, the author recommends using the nerve protector designed specifically for this purpose by the author himself; it fits inside the nerve canal over the nerve (Figure12 C and D'). Direct contact of rotary or other surgical instruments with the nerve is among the most serious injuries in this type of surgery.
3. In order to lateralize the nerve, use instruments with minimal traction and prevent ischemia to the nerve. Instruments that have large contact area with the nerve and mini-

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mum thickness are preferred to be placed between the nerve and the location of drilling for implant placement.

4. The retracted bundle should be constantly moistened by normal saline.

5. Prevent development of hematoma because it applies pressure on the nerve trunk.

6. After inserting the implant, autogenous bone powder or collagen membrane should be placed between the implant and the nerve bundle (as discussed earlier).

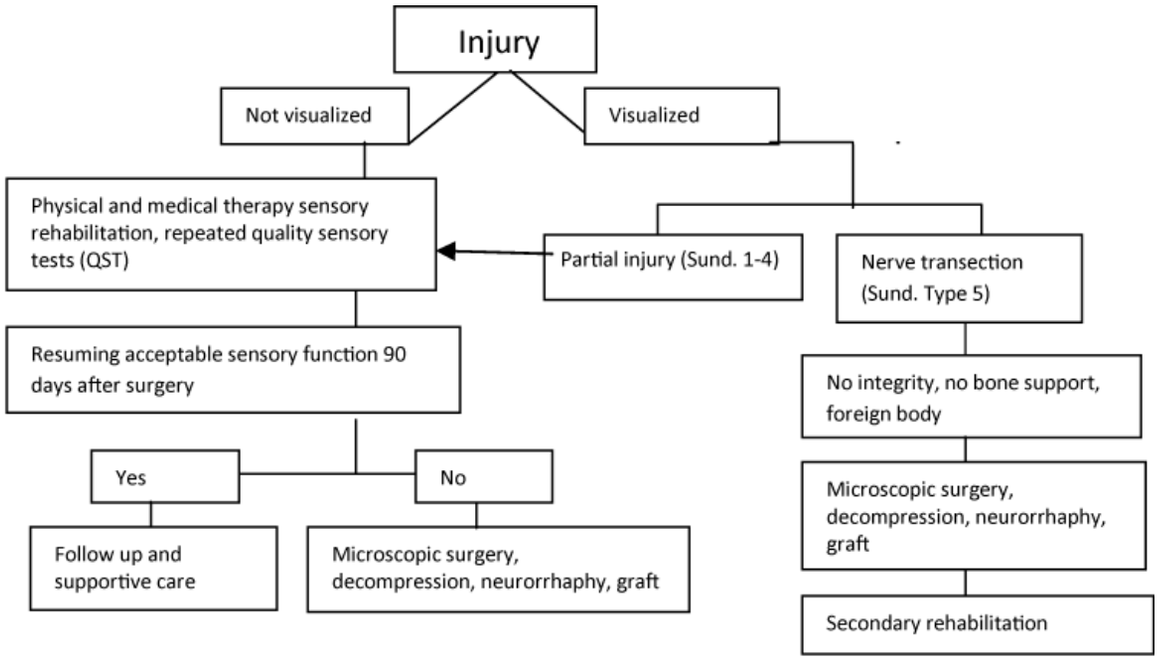
7. Use of anti-inflammatory drugs before and after surgery: Some articles have recommended administration of corticosteroids pre- and post-operatively or high dose ibuprofen 800 mg TDS for 3 weeks [39].

8. Using vitamin B complex supplements (studies have shown that B complex and vitamin E supplementation improves nerve function and decreases neuropathy. Vitamin B family especially B1 and B12 can prevent nerve injury and improve natural growth of the nerve by preserving and protecting the lipid-rich covering of nerve terminals. Alcohol consumption causes vitamin B deficiency and therefore should be avoided [40].

9. Use of low level laser (LLL) immediately after surgery 4 times a week for 10 sessions. Studies suggest using LLL as a non-invasive non-surgical method for faster recovery from paresthesia may obviate the need for surgery in nerve injuries. Use of GaA1As laser causes the patient's subjective and objective symptoms to disappear. Low level laser increases nerve function and capacity of myelin production [10,41]. Bleeding inside the canal can cause a hematoma or compartment syndrome [42]. The incidence of post-operative neuropraxia, permanent anesthesia and paresthesia decreases when only the thicker parts of the neurovascular bundle are manipulated compared to the manipulation of thinner parts or terminal branches. Therefore, although nerve transposition in more posterior areas like the 2nd molar area is technically more complex, it is usually associated with smaller risk of serious and long term injuries to the nerve because the neurovascular bundle is thicker in this region. Regeneration process of nerve following mild compression or crushing takes several weeks to 6 months [10]. If recovery does not occur in this time period, we should consider the possibility of permanent anesthesia. Some researchers believe that sensory changes following implant placement and nerve transposition should be considered as a normal consequence of treatment and not a sequel or complication [10,43].

2.5.3. Pharmaceutical therapy and treatment of traumatic nerve injuries

Course of nerve recovery and symptoms vary based on the type and severity of the primary injury. In most cases, only time and regular patient visits are required. Other cases may need drug therapy or microscopic reconstructive neural surgery (**Algorithm 1**). In case of nerve transection, we can suture the free ends without traction but primary and simultaneous graft should never be performed. If the nerve is under traction, greater fibrosis will develop at the site of repair. In cases with nerve compression or traction, the surgeon should release the nerve and eliminate the traction or compression and prevent ischemia due to mechanical



Algorithm 1. How to decide about the treatment and management of inferior alveolar nerve injury

trauma [12]. After nerve repair, clinical tests should be performed weekly during the first month and then monthly for 5 months. It is especially important to do the test in the first month to diagnose if neuroma or neuropathic pains develop [39]. In case of presence of neuropathic pains, primary management includes nerve block by local anesthetics, use of analgesics and nerve stimulation through the skin (30 min a day for 3 weeks). If post traumatic neural pains do not alleviate pain after 3-4 weeks, administration of various drugs have been recommended [12].

Some of the medications used for neuropathic pain control:

1. Fluphenazine 1 mg, 3 times a day along with Amitriptyline 75 mg before bedtime
2. Doxepin (tricylic antidepressant) 25 mg 3 times a day
3. Carbamazepine up to 100 mg/day
4. Baclofen up to 80 mg/day
5. For sympathetical pain we can do injections for stellate ganglion block. Alpha 2 adrenergic blockers (clonidine 0.1 to 0.3 mg/day based on tolerance) 5 times a week for 3 weeks; sympathectomy can also be used.
6. In case of acute pains fast-acting anticonvulsants (like clonazepam) 2 to 10 mg/day may be useful.
7. Titrated Gabapentin anticonvulsant 600 to 3000 mg is beneficial for chronic pains following traumatic injuries. If the patient also suffers from sleep disorders, antidepressants may be used at bedtime.

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8. Anti-inflammatory drugs, analgesics, anti-anxiety medications and sleeping pills can also be used in addition to the above mentioned medications.

9. Topical lidocaine gel (for mucus membrane) and 5% lidocaine patches (for skin) which are released slowly within 12 hours are used for the mucous membrane and skin of the irritated areas or trigger zones.

10. Intravenous injection of lidocaine may be used sometimes for diagnostic purposes. In such cases, first normal saline is injected as a placebo and then the patient's symptoms are evaluated and then 1 mg/kg lidocaine is slowly injected intravenously within 2 minutes and the patient is asked about its effects every 30 seconds. Pain relief (more than 30%) indicates the effectiveness of intravenous lidocaine injection which shows the neuropathic origin of the pain and we should consider the probable efficacy of medications with central effects such as anticonvulsants [12, 22].

Nerve reconstruction

In case of requiring inferior alveolar nerve reconstructive surgery, it is important to maintain the integrity of the nerve. First, the nerve is exposed and the surrounding tissues are released so that the extensiveness of injury is evident. Compression injuries result in development of fibrosis. In such cases, first lactate ringer's solution is subcutaneously injected in the fibrotic area with a 30 gauge needle to separate the epineurium from the fascicles and determine the extension of fibrous tissue. Then the fibrous tissue is eliminated by a fine longitudinal incision over the epineurium. If the fibrous tissue is extensive and has penetrated into the fascicles we have to dissect this area and suture the free ends of the nerve together. Inferior alveolar nerve is usually composed of 12 to 30 small fascicles with scattered epineurium wrapped around them. Therefore, extensive fibrosis between the fascicles rarely occurs unless in case of major injury. If there is a neuroma, similar to extensive fibrosis the lesion has to be removed and the two free ends should be sutured together. No traction should be applied to the free ends when suturing in order to avoid future fibrous formation. Approximation of the two ends of the nerve regardless of the direction of fascicles and placing the fascicles alongside each other is called coaptation. Since inferior alveolar nerve is a sensory nerve, often only approximation is sufficient. Sutures are applied to the epineural layer. If neural graft is intended, the most similar nerve to the inferior alveolar nerve in terms of diameter and consistency is the sural nerve and the second most similar is the greater auricular nerve [12,22].

Surgical intervention for a patient suffering from nerve injury has 2 main objectives: resuming the sensory function and managing the pain and discomfort due to nerve injury.

Indications of explorative surgery and nerve reconstruction include:

1. Visible injury

2. Presence of foreign body around the nerve

3. No change in anesthesia or hypoesthesia 2 months after nerve injury

4. Uncontrollable neuropathic pain

Contraindications of explorative surgery and nerve reconstruction include:

1. Signs of improved sensory function based on quantitative sensory testing (QST) which is a method for determining the exact threshold of sensory stimulation with the use of oscillatory, touch, thermal or painful stimuli)
2. Patient admission based on remaining dysfunction or present discomfort
3. Signs of central sensitivity (regional dysesthesia, secondary hyperalgesia)
4. Presence of clinical symptoms with autonomic origin (erythema, swelling, hypersensitivity, burning sensation) which are indicative of autonomic nerve dysfunction rather than sensory nerve injury)
5. Old age, presence of an underlying systemic or neuropathic disease
6. A long time has passed since the injury
7. Patient has unrealistic expectations (demands immediate full recovery or resuming of sensory function with no pain)
8. Neural pains that are not alleviated by local anesthesia [22]

Primary care of a patient with nerve injury includes:

The main goal of primary treatment of nerve injury is to eliminate the progressive cause, and prevent secondary nerve injury to allow formation of a peripheral tissue for maximum recovery of the nerve and avoid secondary neuropathic hypersensitivity. If the injured nerve is exposed, pressure from the foreign body, bony and dental chips, toxic materials or implant if present should be eliminated. The exposed nerve should be washed with isotonic solution and sutured with temporary epineural sutures. Infection and inflammation should be controlled precisely both locally and systemically. Anti-inflammatory medications, opioid analgesics and sedatives should be extensively used in order to control anxiety and minimal stimulation of the CNS. An appropriate method for this purpose is administration of a long acting local block anesthesia. A course of systemic corticosteroids like dexamethasone 8 to 12 mg/day can decrease the perineural inflammation in the first week following surgery [44]. Fast acting anticonvulsants like clonazepam in divided doses of 2 to 10 mg/day can further protect the CNS [45]. Topical lidocaine in the form of gel or 5% patch which is released and absorbed subcutaneously within 12 hours can also be used [46]. Basic examinations should be performed using QST and the injured nerve should be under follow up. The patient should be informed about the nature of nerve injury, importance of tests and examinations, constant and immediate care, possibility of requiring secondary consultation and microscopic reconstructive surgery and possibility of prolonged recovery.

Nerve surgery is categorized into 3 types based on the time of surgery following nerve injury:

1. Primary and immediate surgery: within the first few hours following injury.
2. Delayed surgery: within 14 to 21 days after injury.
3. Secondary surgery: 3 weeks after injury.

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Primary surgery is indicated when the nerve is exposed and becomes injured. It is usually performed in cases of trauma, orthognathic surgery, implant surgery, dentoalveolar pathologies and some cases of 3rd molar surgeries. From the biologic point of view, immediate primary surgery is preferred over other types. Despite limitations, primary repair is feasible even in the office. Use of surgical loop is recommended.

Delayed surgery following primary surgery, may also be require which is performed a few weeks following injury when the acute post-op condition of the area has subsided and the site is ready for the definite operation of nerve exposure and microscopic surgery.

Secondary surgery is done for invisible trigeminal nerve injury; this injury is not an uncommon event and requires secondary reconstructive surgery under controlled conditions following informing the patient about the indication of surgery, and explaining the situation according to clinical conditions and repeated QSTs. There is controversy regarding the optimal time for conduction of secondary surgery among researchers [47]. There are 3 reasons why the earlier reconstructive surgery within the first week following injury is preferred:

1. The high capacity for maximum recovery within the first week after surgery
2. Quick intervention can prevent traumatic neuroma from extension and subsequent chronic neuropathic hypersensitivity or fibrosis
3. Technical simplicity of the reconstruction (after a long delay, microscopic surgery would be very difficult due to the contraction and progressive atrophy of the nerve segments, increased collagen precipitation inside and outside the nerve and scarring of Schwann cells)

The first phase of nerve reconstruction includes:

1. Decompression of the injured nerve by extracting the foreign bodies and releasing the scar tissues and other tissues compressed around the nerve.
2. Detection of the injured area, incision and transection of the traumatic neuroma
3. Repair with microscopic sutures through neurorrhaphy (repeated direct anastomosis)
4. Reconstruction through an interstitial graft if neurorrhaphy is not feasible due to the extensive loss of nerve tissue.

Nerve graft: In some cases of severe injury, reconstruction through direct neurorrhaphy is not feasible. Clinical experience shows that distances wider than 15 to 20 mm cannot be repaired through neurorrhaphy and suturing without tension. In such cases, nerve grafting is indicated.

Autogenous graft: Our first choices for a nerve graft are the sural nerve, great auricular nerve, and anti-brachial skin nerve. All these donor nerves are easily accessible and provide sufficient length of the tissue (more than 6 cm)[48-50].In order to avoid tissue fragility, minimum number of sutures should be used. It would be ideal if the nerve is wrapped in a protective biodegradable barrier. The main complications in autogenous grafts are development of a sense of numbness and anesthesia/dysesthesia, and formation of a neuroma at the donor site. In cases where sural nerve is used, there is a risk of defect and difficulty along with hyperesthesia at the lateral

and posterior surface of the foot where is in contact with shoes and in the ankle. When the greater auricular nerve is used the patient may experience paresthesia at the lateral side of the neck and at the angle of mandible. This is especially troublesome in patients who have trigeminal neuropathy adjacent to this location. Another problem related to greater auricular nerve is the various diameters of this nerve [51].The greatest technical problem in autogenous nerve graft is the incompatibility in shape, size and number of fascicles between the grafted nerve and the inferior alveolar nerve. The inferior alveolar nerve has an average 2.4 mm diameter and is cylindrical. In comparison, the sural nerve has approximately 2.1 mm diameter versus 1.5 mm diameter of great auricular nerve. Both of these nerves have a significantly smaller number of fascicles than the inferior alveolar nerve [52]. It is not feasible to completely match the fascicles at the time of nerve grafting which amplifies the disorganized regeneration of the axon in between the grafted area [53].

Alternative strategies for autogenous grafts:

An alternative strategy for nerve graft is to use skeletal muscles [54]. To date, there is no definite report regarding the level of sensory recovery of the inferior alveolar nerve. Also, use of arteries and veins has been reported with varying levels of success clinically [55]. Use of vasculature for grafting has been considered because of the minimum tissue invasion and ease of access. However, this method has not shown acceptable results thus far. At present, some have suggested using alloplastic grafts which have caught great attention for their availability and avoiding the morbidity of the donor site. Their biocompatibility and efficacy are for the short grafts only. However, acceptable results have not been reported in this regard either.

Management of sensory function after nerve transposition surgery:

Inferior alveolar nerve transposition for implant placement almost in 100% of cases results in sensory impairment immediately after surgery [10,31,32]. Sensory disturbances are resolved in 84% of cases and in only 16% of patients may this complication be permanent and irreversible [10,24,32,33]. The important issue in management of nerve injury is to inform and educate the patient in this respect. The patient should be educated before and after the surgery and should be well aware that nerve reconstruction may take a long time and he/she may experience paresthesia or dysesthesia for a long period of time. The patient may be taught to massage the area (with lanolin or a moisture absorbing ointment). Massage should be started with mild movements and then the intensity is increased to improve the sense of touch. Massaging is indicated 4 to 6 times a day for 10 to 15 minutes. The first sense that resumes is the sense of cold followed by pain. At this time the patient still has paresthesia in the area. After 4 to 5 months, the patient would be able to differentiate between cold and heat sensations and feels the sharpness of needle with 25 to 30 g pressure. After 6 months, touch, pain and thermal sensations will resume more efficiently [12]. All patients should undergo treatment with low level laser for 10 sessions (4 times a week). The sessions start from the day of surgery. The sensitive area is detected using a simple anesthesia needle and is controlled monthly. The percentage of recovery is calculated by the proportion of the primary area suffering from paresthesia to the final area after 6 months. Researches indicate that chance of spontaneous recovery of the nerve is smaller in women compared to men

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[10]. As mentioned earlier, most surgeons believe that sensory disturbances should be considered as a normal predictable state following nerve transposition surgery and not a complication or sequel of treatment [10,32].

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References

[1] J.M. Hirsch, P.I. Branemark. Fixture stability and nerve function after transposition and lateralization of the inferior alveolar nerve and fixture installation. Br J Oral Maxillofac Surg.1995;33(5):276-281.

[2] Thoma Kh, Holland DJ. Atrophy of the mandible.Oral Surg Oral Med Oral Pathol. 1951;4(12):1477-95.

[3] Rocchietta I, Fontana F, Simion M. Clinical outcomes of vertical bone augmentation to enable dental implant placement: a systematic review.Clin Periodontol. 2008;35:203-15.

[4] Boyne PJ, Cooksey DE. Use of cartilage and bone implants in restoration of edentulous ridges.J Am Dent Assoc. 1965;71(6):1426-35.

[5] Chang CS, Matukas VJ, Lemons JE.Histologic study of hydroxylapatite as an implant material for mandibular augmentation.J Oral Maxillofac Surg. 1983;41(11):729-37.

[6] Jensen OT. Combined hydroxylapatite augmentation and lip-switch vestibuloplasty in the mandible..Oral Surg Oral Med Oral Pathol. 1985;60(4):349-55.

[7] Egbert M, Stoelinga PJ, Blijdorp PA, de Koomen HA. The "three-piece" osteotomy and interpositional bone graft for augmentation of the atrophic mandible.J Oral Maxillofac Surg. 1986;44(9):680-7.

[8] Matras H. A review of surgical procedures designed to increase the functional height of the resorbed alveolar ridge.Int Dent J. 1983;33(4):332-8.

[9] Vasconcelos Jde A, Avila GB, Ribeiro JC, Dias SC, Pereira LJ. Inferior alveolar nerve transposition with involvement of the mental foramen for implant placement.Med Oral Patol Oral Cir Bucal.2008;13(11):E722-5.

[10] Jensen O, Nock D.Inferior alveolar nerve repositioning in conjunction with placement of osseointegrated implants: a case report.Oral Surg Oral Med Oral Pathol. 1987;63(3):263-8.

[11] Rosenquist B.Fixture placement posterior to the mental foramen with transpositioning of the inferior alveolar nerve.Int J Oral Maxillofac Implants. 1992;7(1):45-50.

[12] Yaghmaei M.Mandibular Canal (clinical Aspects).1st ed.Tehran: Karvar Publishers; 2010.

[13] Hu KS, Yun HS, Hur MS, Kwon HJ, Abe S, Kim HJ. Branching patterns and intraosseous course of the mental nerve. J Oral Maxillofac Surg. 2007;65(11):2288-94.

[14] Sanchis JM, Peñarrocha M, Soler F.Bifid mandibular canal. J Oral Maxillofac Surg. 2003;61(4):422-4.

[15] Stella JP, Tharanon W.A precise radiographic method to determine the location of the inferior alveolar canal in the posterior edentulous mandible: implications for dental implants. Part2: Technique.Int J Oral Maxillofac Implants. 1990;5(1):23-9.

[16] Stella JP, Tharanon W.A precise radiographic method to determine the location of the inferior alveolar canal in the posterior edentulous mandible: implications for dental implants. Part 1: Technique.Int J Oral Maxillofac Implants. 1990;5(1):15-22

[17] Kieser J. Kieser D. Hauman T.The Course and Distribution of the Inferior Alveolar Nerve in the Edentulous Mandible. J Cranio fac Surg..2005;16(1):6-9.

[18] Chrcanovic BR, Custodio AL. Inferior alveolar nerve lateral transposition. Oral Maxillofac Surg. 2009;13(4):213-9.

[19] Wadu SG, Penhall B, Townsend GC.Morphological variability of the human inferior alveolar nerve. Clin Anat. 1997;10(2):82-7.

[20] Choukas NC, Toto PD, Nolan RF.A histologic study of the regeneration of the inferior alveolar nerve.J Oral Surg. 1974;32(5):347-52.

[21] Yaghmaei M, Mashhadiabbas F, Shahabi S, Zafarbakhsh A, Yaghmaei S, Khojasteh A. Histologic evaluation of inferior alveolar lymphatics: an anatomic study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011 Feb 16.

[22] Van Geffen GJ, Moayeri N, Bruhn J, Scheffer GJ, Chan VW, Groen GJ.Correlation between ultrasound imaging, cross-sectional anatomy, and histology of the brachial plexus: a review. Reg Anesth Pain Med. 2009;34(5):490-7.

[23] Hupp J.R. Ellis E.Tucker M.R.Contemporary Oral and Maxillofacial surgery.5th ed.Missouri:Mosby Elsevier;2008.p.281,285,620-622.

[24] Seddon HJ.A Classification of Nerve Injuries. Br Med J. 1942 Aug 29;2(4260):237-9.

[25] Sunderland S.A classification of peripheral nerve injuries producing loss of function. Brain.1951;74(4):491-516.

[26] Poort LJ, van Neck JW, van der Wal KG. Sensory testing of inferior alveolar nerve injuries: a review of methods used in prospective studies.J Oral Maxillofac Surg. 2009;67(2):292-300.

[27] Kahnberg KE, Ridell A. Transposition of the mental nerve in orthognathic surgery. J Oral Maxillofac Surg. 1987;45(4):315-8.

[28] Lindh C, Petersson A.Radiologic examination for location of the mandibular canal: a comparison between panoramic radiography and conventional tomography.Int J Oral Maxillofac Implants.1989;4(3):249-53.

[29] Babbush CA,Hahn JA,Krauser JT,Rosenlicht JL.Dental Implants:The Art and Science, 2th Ed. London:Saunders Elsevier ; 2010,232-250.

[30] Rosenquist Bo.Implant Placement in Combination With Nerve Transpositioning: Experiences With the First 100 Cases.Int J Oral Maxillofac Implants.1994: 9(5):522-531.

[31] Rosenquist BE.Nerve transpositioning to facilitate implant placement. Dent Econ. 1995;85(10):92-3.

[32] Kan JY, Lozada JL, Goodacre CJ, Davis WH, Hanisch O. Endosseous implant placement in conjunction with inferior alveolar nerve transposition: an evaluation of neurosensory disturbance.Int J Oral Maxillofac Implants. 1997;12(4):463-71.

[33] Peleg M, Mazor Z, Chaushu G, Garg AK. Lateralization of the inferior alveolar nerve with simultaneous implant placement: a modified technique.Int J Oral Maxillofac Implants2002;17(1):101-6.

[34] Friberg B, Ivanoff CJ, Lekholm U. Inferior alveolar nerve transposition in combination with Branemark implant treatment. Int J Periodontics Restorative Dent. 1992;12(6):440-9.

[35] Hashemi HM.Neurosensory function following mandibular nerve lateralization for placement of implants.Int J Oral Maxillofac Surg. 2010;39(5):452-6.

[36] Smiler DG. Repositioning the inferior alveolar nerve for placement of endosseous implants: technical note.Int J Oral Maxillofac Implants. 1993;8(2):145-50.

[37] Yoshimoto M, Konig B Jr, Allegrini S Jr, de Carvalho Lopes C, Carbonari MJ, Liberti EA, Adami N Jr. Bone healing after the inferior alveolar nerve lateralization: a histologic study in rabbits (Oryctolagus cuniculus).J Oral Maxillofac Surg. 2004;62:131-5.

[38] Kahnberg KE, Henry PJ, Tan AE, Johansson CB, Albrektsson T.Tissue regeneration adjacent to titanium implants placed with simultaneous transposition of the inferior dental nerve: a study in dogs. Int J Oral Maxillofac Implants. 2000;15(1):119-24.

[39] Brasileiro BF, Van Sickels JE.A modified sagittal split ramus osteotomy for hemimandibular hyperplasia and simultaneous inferior alveolar nerve repositioning. J Oral Maxillofac Surg. 2011 Dec;69(12):e533-41

[40] Kraut RA, Chahal O. Management of patients with trigeminal nerve injuries after mandibular implant placement. J Am Dent Assoc. 2002;133(10):1351-4.

[41] Kubilius R. Sabalys G. Juodzbalys G.Gedrimas V.Traumatic Damage to the Inferior Alveolar Nerve Sustained in Course of Dental Implantation.Possibility of Prevention.Stomatologija,Baltic Dent Maxillofac.2004; 6:106-10.

[42] Khullar SM, Brodin P, Barkvoll P, et al: Preliminary study of low-level laser for treatment of long-standing sensory aberrations in the inferior alveolar nerve. J Oral Maxillofac Surg 54:2, 1996

[43] Matsen FA,Winquist RA,Krugmire RB. Diagnosis and management of compartment syndromes. J bone joint surg Am.1980;62(2):286-291.

[44] Seo K et al..Efficacy of steroid treatment for sensory impairment after orthognathic surgery. Oral Maxillofac surg 2004; 62:1193.

[45] Bartusch SL. Et al..Clonazepam for the treatment of lancinating phantom pain. Clin J Pain 1996;12:59.

[46] Rowbotham MC et al.Lidocaine patch: double-blind controlled study of a new treatment method for postherpetic neuralgia.J Pain.1996;65: 39.

[47] Davis H, Ohrnell LO, Larson C, et al. Lateralizing of the inferior alveolar nerve to allow fixture placement. Proceedings of the UCLA Symposium on Implants in the Partially Edentulous Patient. Los Angeles, 1990:28–31.

[48] Susarla SM et al..Dose early repair of lingual nerve injuries improve functional sensory recovery?. J oral maxillofac surg;2007:65:1070.

[49] Brammer JP, Epker BN. Anatomic-histologic survey of the sural nerve: implications for inferior alveolar nerve grafting.J Oral Maxillofac Surg. 1988;46(2):111-7.

[50] Eppley BL, Snyders RV Jr. Microanatomic analysis of the trigeminal nerve and potential nerve graft donor sites.J Oral Maxillofac Surg. 1991;49(6):612-8.

[51] McCormick SU, Buchbinder D, McCormick SA, Stark M. Microanatomic analysis of the medial antebrachial nerve as a potential donor nerve in maxillofacial grafting.J Oral Maxillofac Surg. 1994;52(10):1022-5.

[52] Takasaki Y, Noma H, Kitami T, Shibahara T, Sasaki K. Reconstruction of the inferior alveolar nerve by autologous graft: a retrospective study of 20 cases examining donor nerve length.Bull Tokyo Dent Coll. 2003;44(2):29-35.

[53] Pogrel MA, Renaut A, Schmidt B, Ammar A. The relationship of the lingual nerve to the mandibular third molar region: an anatomic study.J Oral Maxillofac Surg. 1995;53(10):1178-81.

[54] Rath EM. Skeletal muscle autograft for repair of the human inferior alveolar nerve: a case report.J Oral Maxillofac Surg. 2002;60(3):330-4

[55] Pogrel MA, Maghen A. The use of autogenous vein grafts for inferior alveolar and lingual nerve reconstruction.J Oral Maxillofac Surg. 2001;59(9):985-8.

[56] Miloro M, Stoner JA. Subjective outcomes following sural nerve harvest. Oral Maxillofac Surg. 2005;63(8):1150-4.

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