Trigeminal Nerve Injuries

25

Tara Renton

There are infinite opportunities in dentistry to damage the trigeminal nerve. Nerve damage from surgery can cause chronic post-surgical pain; however, this is limited in dentistry as a result of Local anaesthetic (LA) infiltration injections but more commonly associated with injuries to the nerve trunks of division two and three caused by implants, endodontics and third molar surgery (or other high-risk extractions). Fortunately, painful post-traumatic trigeminal neuropathy (PPTTN) is rare in dentistry compared to other common general surgical procedures where 20–45% of patients experience persistent pain after surgical limb amputation, thoracotomy and breast surgery. This chapter highlights the prevention (using risk assessment, optimal surgical techniques, early post-surgical follow-up protocols and other strategies) and optimal management of trigeminal nerve injuries.

Trigeminal nerve injury (TNI) associated with chronic pain is the most problematic consequence of dental surgical procedures with major medico-legal implications [1]. The incidence of lingual nerve injury has remained static in the UK over the last 30 years, but is increasing in the US, as is the incidence of inferior alveolar nerve injury in the UK, with the latter being due to implant surgery and endodontic therapy [2]. Third molar surgery-related inferior alveolar nerve (IAN) neuropathy or inferior alveolar block injections are usually temporary but can persist and become permanent (by definition at 3 months). There are rare reports of resolution of implant and other cause-related IAN neuropathies at over 4 years, [3] but these are not similar reports of other peripheral sensory nerve injuries [4–7]. In dentistry, frequently, a treat delay is 3–6 months [5], which is inappropriate when compared to other peripheral sensory nerve injuries where immediate repair and exploration are recommended. We now understand that known or suspected, restorative (endo and implant)-induced nerve injuries require intervention ideally immediately, within 30 h or within 3 months, dependent upon the mechanism of injury, to optimize resolution from injury and prevent the permanent central and peripheral changes within the nervous system [6, 7].

Paraesthesia is often inappropriately used in the dental literature to mean neuropathy. However, paraesthesia is only a descriptive term, meaning altered sensation and not a diagnosis. When sensory nerves are injured, a neuropathy (malfunction) may arise, and this may be painful or non-painful.

The trigeminal nerve has the largest representation in the sensory cortex, reflecting the disproportionate sensory input from the orofacial region. It protects vital structures that underpin our very survival, providing sensory supply to the eyes, airway, brain, mouth and ears. It is no 'wonder' that when the threat or actual pain arises in the trigeminal nerve area that the patient is neurophysiologically wired to 'run for the hills' from the dental chair. Latrogenic (caused by surgery or medicine) trigeminal nerve injuries (TNIs) result in 70% pain in patients seen seeking treatment on our clinic [7]. The ongoing or evoked pain results in interference with eating, speaking, sleeping, applying makeup, shaving, kissing, tooth brushing and drinking; just about every social interaction, we take for granted. As a result, these injuries have a significant negative effect on the patient's self-image, quality of life and psychology [7].

With the increasing age of the patient, the time elapsed since the injury and the proximity of the injury to the cell body (the more proximal lesions have a worse prognosis) will dictate the persistence of any peripheral sensory nerve injury.

There are many non-surgical causes for trigeminal neuropathy, and these must be borne in mind if the patient presents with an unclear onset of motor or sensory neuropathy [8].

Sensory nerve injuries caused by implant and endodontic treatments are mainly permanent. Only LA nerve injuries have a 75% likelihood of recovery and lingual access third molar surgery and have 90% potential for recovery [9].

T. Renton (✉)
Oral Surgery, Kings College London, London, UK
e-mail: Tara.renton@kcl.ac.uk

K. Bonanthaya et al. (eds.), *Oral and Maxillofacial Surgery for the Clinician*, https://doi.org/10.1007/978-981-15-1346-6_25

Referral of patients with these nerve injuries before 4 months [2] is too late for optimal resolution or management as inferior alveolar nerve injuries often require an immediate implant or endo-treated tooth removal within 24–30 h. We now understand that after 3 months, permanent central and peripheral changes occur within the nervous system subsequent to injury, which are unlikely to respond to surgical intervention [10].

Nerve damage is likely to result from a combination of poor risk assessment, poor technique, lack of recognition and the acute management of intra-operative and post-operative signs of neuropathy. Risk assessment involves the patient selection, pre-operative planning, both clinical and radiographic, appropriate selection of implant site and type (width and length) and suitable treatment protocol and follow-up.

It is important that the clinician is familiar with the nerve injury risk factors, specific for each of the types of invasive procedures. For example, in the case of protrusion through the IDC and resultant direct IAN mechanical injury by implant drill, a "sudden give" or an "electric shock" type feeling, even with local anaesthesia working, is reported by most of the patients seen in our clinic with post-traumatic neuropathy. This should result in the clinician stopping surgery, not reaching for another LA block injection and reassessing their surgical position with regard to the injured nerve.

The problem with implant-related nerve injuries is that they are entirely avoidable as this is elective surgery, thus negligent, and likely to be permanent and painful for the patient [11]. In addition, persistent nerve injuries cannot be resolved. Surgical intervention for hypoaesthetic nerve injuries does not return the patient to normality [10], and surgery for patients with pain and hyperaesthesia is not appropriate as the pain is not abated and patients are faced with long-term anti-epileptics or anti-depressants for chronic pain [11].

When assessing patients with surgically induced nerve injuries, we recommend a more holistic approach in assessing patients with nerve injury [8]. The definition ICHD 3 (International Craniofacial pain and Headache Disorders) of painful post-traumatic trigeminal neuropathy (PPTTN) includes development of neuropathy within 3 months of injury with sensory neuropathy and pain. The author believes that the neuropathy develops immediately after trauma, unless related to endodontic procedure where there may be a 2–3 days delay in neuropathy development. Features of iatrogenic trigeminal nerve injury worthy of assessment include

- Focal sensory neuropathy (mostly present). There is almost always an area of abnormal sensation (neuropathy with the exception in Trigeminal neuralgia which is NOT post-traumatic), and the maximum reported pain is associated with the area of sensory deficit (i.e. suffering from a mixture of pain, numbness and altered sensation). This is an important diagnostic feature for sensory nerve neuropathy.
- Pain discomfort, altered sensation and numbness (anaesthesia). Neuropathic pain is commonly present with allodynia (pain on non-noxious stimuli), hyperalgesia (increased pain to noxious stimuli) and hyperpathia (continuous altered sensation or pain after stimulation ceases). In 50–70% of patient reports, a combination of numbness, altered sensation and pain is experienced, the pain may be spontaneous ongoing pain, which often had a burning character, spontaneous shooting or electric shock-like sensations (neuralgia) [7]. Evoked pain due to touch or cold often leads patients to have difficulties with daily function, such as eating, socializing, kissing, speech and drinking. As a consequence, patients are often anxious and tearful and had psychological repercussions of surgery. These symptoms were often compounded by the lack of informed consent, which was given by only 30% of patients, most of whom were not specifically warned about potential nerve injury [7].
- Daily function problems (drinking, kissing, eating, sleeping, speaking, tooth brushing and avoidance) [12],
- Psychological (anxiety, stress, post-traumatic stress disorder and anger) [12].

The following sections address the prevention and management of trigeminal nerve injuries related to

- Local anaesthesia.
- Implants.
- Third molar and other high-risk extractions.

25.1 Local Anaesthetic-related Nerve Injuries

Local block injection-related nerve injury is an acknowledged complication in relation to surgery [13]. Dentistry is the only speciality that still trains clinicians to aim for nerves rather than avoiding neural contact (often using ultrasound), which likely explains the continued prevalence of LA-related nerve injuries in dentistry. All other block injections are undertaken using ultrasound in order to avoid nerve injury. One report highlights that the prevalence of IDB-related nerve injuries in UK General dental practise is 1:14,000 blocks, or 1:56 K IDB patients experience permanent lingual or inferior alveolar nerve injury of which this 25% of nerve injuries are permanent [9]. It is estimated that every practising dentist will experience causing 4–6 temporary nerve injuries and one permanent nerve injury related to IDBs during their working life based upon current practice.

Nerve injury may be due to many causes including physical (needle, compression due to epineural or perineural haemorrhage), ischaemic or chemical (haemorrhage or LA contents). The site of the injury may also vary and combine peri-, epi- and intra-neural trauma causing subsequent haemorrhage, inflammation and scarring, resulting in demyelination (loss of nerve lining) [14]. Only 1.3–8.6% of patients get an 'electric shock' type sensation on application of an IAN block and 57% of patients suffer from prolonged neuropathy having not experienced the discomfort on injection, and thus, this is not a specific sign [15]. Routine practice in Germany includes warning patients of potential nerve injury in relation to dental block injections. Risk factors for persistent local anaesthesia nerve injuries are summarized in Table 25.1.

The lingual nerve (LN) is at increased risk of permanent injury compared to the IAN during local anaesthesia, possibly related to the reduced number of fascicles in the LN compared to the IAN [16].

Higher concentration agents are more neurotoxic and, therefore, more likely to cause persistent inferior dental block (IDB)-induced nerve injury [17–22]. Irrefutably, Schwann cell death is related to increased concentration and time exposure to LA [22]. Articaine is provided in 4% concentration and Lidocaine in 2% solution in most countries. A recent prospective randomized study reports that there is no benefit or using 4% Articaine IDBs compared to 2% Lidocaine [23], which is substantiated by other evidence [24–27]. Thus, logically why would anyone use a higher concentration agent for an IDB when there is no increased efficacy and higher risk of nerve injury? [28]

Intra-operatively, all clinicians should document unusual patient pain reactions occurring during injections or surgery (such as sharp pain or an electrical shock-like sensation), as neuralgia during injections is associated with increased persistence of nerve injury [15]. Thus, it is important that the clinician uses an appropriate LA method to prevent proximity of the injection/surgical instruments to the IDC, for example, infiltration anaesthesia for implant surgery.

Table 25.1 Risk factors for persistent neuropathy related to IDBs

In order to minimize complications related to dental LA, you need to consider modifying the following risks:
• **Block anaesthesia:** nerve block injections should be undertaken without intent on direct 'hit' of the nerve. 60% of patients who experience the 'funny bone' neuralgia due to the IDB needle being placed too close to the lingual or inferior alveolar nerves experience persistent neuropathy
• **Lingual nerve > IAN:** is this technique related or anatomically related (less fascicles in LN lower capacity for recovery). Perhaps, the direct IDB approach may place the lingual nerve at increased risk compared to the indirect technique
• **Concentration of LA:** any increased concentration of any agent leads to increased neural neurotoxicity
• **Volume of LA:** there is no evidence to support this suggestion, but all chemicals are neurotoxic, and depending upon the proximity, LA concentration and neural damage, additional volume would add to potential neurotoxicity
• **Multiple injections:** second or subsequent injections that impede directly on or in neural tissue may not be associated with the usual 'funny bone' neuralgic pain. Thus, the patient does not self-protect as effectively possibly rendering the nerves more at risk of direct damage
• **Severe pain on injection:** 60% increased occurrence of persistent neuropathy after IDBs
• **Type of LA:** agent bupivacaine most neurotoxic of all LA agents
• **Type of vasoconstrictor?** The role of vasoconstrictor in nerve damage is unknown
• **Sedated or anaesthetized patients?** There is no evidence to support unresponsive patients, who are less likely to protect themselves when neuralgia (funny bone reaction) occurs as the IDB needle encroaches too close to the nerve
• **Lack of LA aspiration?** There is no evidence to support that aspiration during IDB results in lower persistent neuropathies, but a pragmatic view may infer less chemical injected intraneurally will cause less chemical nerve injury

25.1.1 Avoiding Block Anaesthesia by Using Infiltration Dentistry

Daublander et al. reported that in a 2014 [23] survey of German dental LA practise, 74% were using infiltration dentistry routinely and rarely giving IDBs (personal communication). Improved patient comfort is reported by patients with preference for having full lingual sensation and shorter duration LA anaesthesia after dental treatment.

There is further evidence to support the notion of infiltration dentistry can be successful in many aspects of dentistry

- *Maxillary infiltration anaesthesia*
  Studies report that 4% Articaine to be more effective than 2% lidocaine for lateral incisors but not molars [25], differing from other reports [23, 25]. A recent randomized controlled trial reported a statistically significant difference advocating the use of 4% Articaine in place of 2% lidocaine for buccal infiltration in patients experiencing irreversible pulpitis in maxillary posterior teeth [24, 26]. This has been superceded by a metanalysis that reports there is no advantage in using 4% Articaine for maxillary infiltration anaesthesia and that 2% Lidocaine is suffice for dental interventions.
- *Pulpal anaesthesia in the anterior mandible compared to inferior dental block (IDBs) [25].*
  Meechan provides evidence supporting the significantly increased rates of pulpal anaesthesia using infiltration anaesthesia when compared to IDB anaesthesia particularly for premolar and incisor teeth (Fig. 25.1).
- *Pulpitic mandibular molars in adults [23, 25, 29, 30].*
  A recent systematic review reports that Articaine is 3.4 times more effective for pulpitic mandibular molars when compared to lidocaine, but there is no difference between Articaine and Lidocaine maxillary infiltrations or IDBs [31].

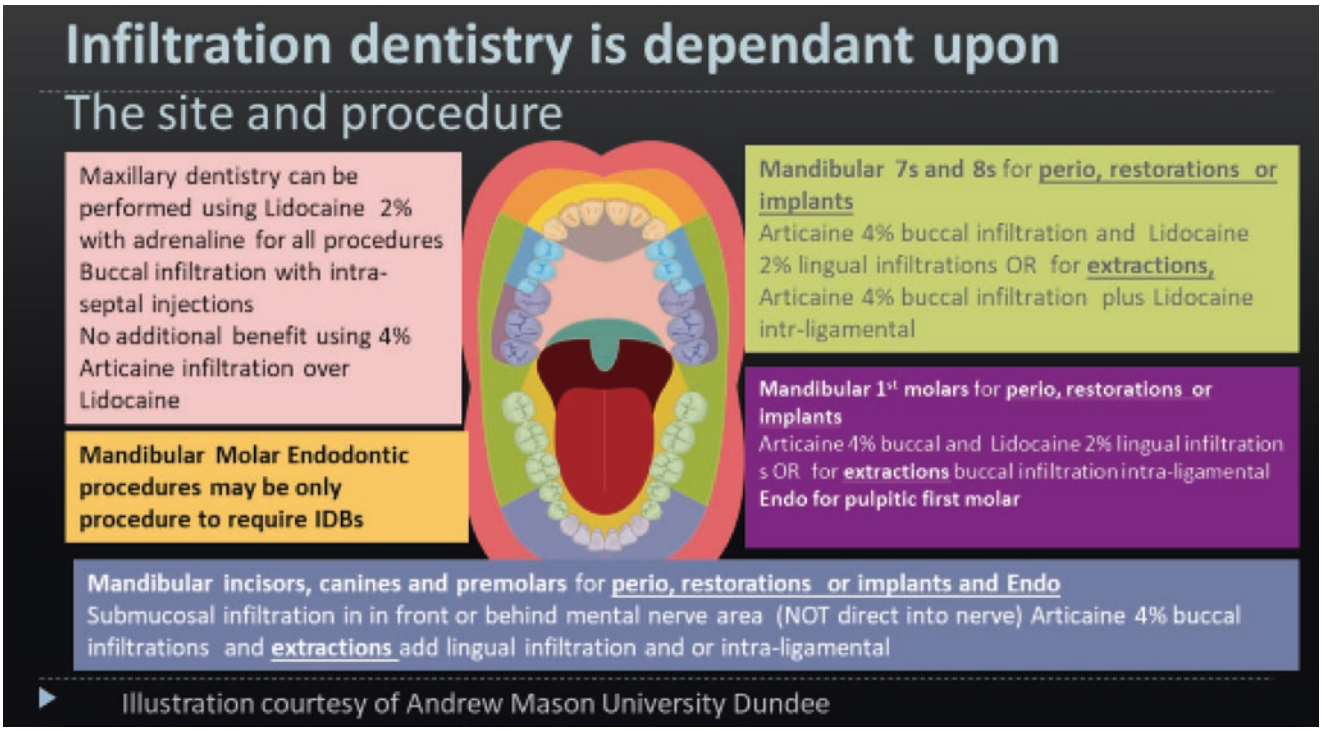


Fig. 25.1 Summary of infiltration methods to minimize nerve injury

- *for exodontia in adults and children [32].*
  Paedodontic extractions do not require IDBs as the bone is very porous and susceptible to absorption of infiltrative anaesthesia.
- *is ideal for implant surgery,*
  Several reports of supra-periosteal infiltration anaesthesia not only are sufficient for posterior mandible implant surgery but also may be protective of the IAN [33].
- *is suitable for periodontal surgery.*
  The standard care for periodontal and implant surgery is infiltration LA [34].

Intra-ligamental anaesthesia for extractions and avoiding IDBs is also gaining population [34].

Key Facts for Prevention of local anaesthetic nerve injuries.

Thus, prevention of LA nerve injuries is possible and some simple steps may minimize LA-related nerve injuries:

- Avoid high concentration LA (Articaine 4%) for block injections and for ID blocks (use 2% Lidocaine as standard) as the efficacy is equal.
- Avoid multiple blocks where possible.
- Avoid nerve contact during block injections.
- Avoid block anaesthesia by using Infiltration dentistry and, thus, prevent LA-related nerve injury, for which there is no cure.

25.2 Management of LA Nerve Injuries

Evidence base remains limited for managing dental LA-related nerve injuries; we only know that 25% are permanent and that there is no 'magic bullet' to fix them. A sit and wait approach has to be adopted with reassurance of the patient and therapeutic management of their symptoms

- HOMECHECK—If you cause pain during an IDB injection in your patient, do follow them up the next day and check they are OK. If the patient reports numbness, altered sensation and/or pain, reassure them.
- Continue to support, reassure your patient and advise them to visit to confirm the presence of neuropathy. If the neuropathy affects most of the dermatome ± associated with severe neuropathic pain, nerve injury must be suspected. Reassure your patient that 75% of these injuries resolve.
- Say SORRY as this is NOT an admission of guilt.
- Initiate medical management (recommended for other peripheral sensory nerve injuries).
  - High-dose oral NSAIDs (400–800 mgs Ibuprofen PO QDS) for 2 days only. Bandolier Oxford league table summarizes the optimal analgesia for post-operative pain, and combined Ibuprofen and paracetamol have the smallest number needed to treat.

  - GMP prescription for Prednisolone 5 day step down does 50-40-30-20-10 mg PO (not for patients with contraindications for steroids or NSAIDs).
  - Vitamin B complex (Riboflavin 400 mg once daily for maximum of 3 months plus other Vit B complex).
- Arrange a review of the patient. All advice is summarized on the Trigeminalnerve.org.uk website.
- Long-term management of patients with non-resolving LA nerve injuries. The reality for these patients is that if they have persistent neuropathic pain and have to be treated as such with psychological and medical management. Topical local anaesthetic (Lidocaine 5%) patches may assist the patient in sleeping and playing sports in cold weather [35]. Psychological interventions play a significant role in managing these patients, and recommendations for treatment of trigeminal neuropathic pain are also well described by Renton & Zakzrewska [36].

25.3 Implant-related Nerve Injuries

Implant-related IANI's incidence varies from 0–40% [37]. Two recent studies highlight persistent neuropathic pain due to implant IANIs [38, 39]. Prevention of nerve implant IANIs can be attributed to the avoidance of direct damage to the Inferior dental canal (IDC) during preparation [40]. A good clinical and radiographic pre-assessment protocol is required to mitigate damage due to proximity of implant bed preparation to the inferior dental canal (IDC) and are recommended [41, 42]. The risk factors for implant nerve injury are summarized in Table 25.2 [41, 42, 45–52].

A limited window is available to maximize inferior alveolar nerve injury resolution in relation to dental implants, endodontics and mandibular wisdom teeth. A report illustrated that early removal of implants (within 30 h) may maximize neuropathy resolution; however, the evidence remains weak [37]. Prevention of implant-related nerve injuries includes;

- *Pre-operatively*
  - undertake a good risk assessment,
  - ability to read and use CBCT to plan a sufficient safety zone (know difference between drill length and implant length),
  - extra diligence in planning implants in the parasymphyseal region near the mental nerve (loop and incisal branches),
  - Screen out neuropathic pain pre-implant.
- *Operatively:*
  - make sure the implant bed preparation is above the safety zone,
  - stop drilling if patient reports intraoperative pain and reassess depth,
  - use drill guides and stops,
  - intra-operative reassessment of implant bed depth using marker and LCPA films at 60% of planned depth,
  - Record any events that may indicate operative nerve injury,
  - extreme pain during LA IDB, or during implant bed preparation
  - Suddenly give and/or profuse haemorrhage arising from the implant bed (possible breech of IDC).
  - In such situations, stop surgery, do not reach for more LA and reassess your surgical position,

Table 25.2 Risk factors for implant-related nerve injury

A. Inadequate preoperative assessment and planning due to
• Surgeon lack of knowledge/inexperience/training
• Inadequate informed consent-all options provided and related risk benefit for each option of treatment. Implants are elective treatment. Sublingual haematoma that can require the need for tracheostomy post-implant treatment and rare events of death
• Lack of identification of existing pre-surgical neuropathy (especially important in edentulous patients)
• Poor planning in risk assessment and positioning the implant. A sectional DPT is recommended as a minimum for mandibular implant planning. If there is limited bone depth, a CBCT may be used to quantify and qualify bone density and volume. The clinician must be able to read and analyse the CBCT, depending upon technicians, software or radiologist specialist (who are not present with you intra-operatively)
– Bone assessment quality and quantity
– Know where the nerve is. Nerve localization, risk factors when assessing IAN position (mental loop, characteristics of IAN position in various sites of mandible) and Parasymphyseal zone that is of high risk.
– The accuracy of estimating the position of the IDC based on plain films or CT scans is highlighted in the radiographic assessment section
– Safety zone- the recommendation is 2 mm (by ITI and ADI), which may be insufficient considering that most-implant drills are 1.5 mm longer than implants. This increases the risk perforation of a canal surrounding IDC or even direct perforation and damage to the nerve
• Selection of implants 10 mm + (short implants <8 mm to simplify procedure and minimize morbidity)
B. Surgical procedure should include the execution of
• Local Anaesthesia (use infiltration LA techniques to allow patients to notify the surgeon or intraoperative neuralgia; if pain is reported, intra-operatively stop surgery and reassess preparation depth and width)
• Flap design
• Use surgical guides to minimize morbidity
• Surgical stents [43]
• Using intra-operative radiographs, ITI recommends stopping drilling after 60% of planned depth and reassess with bed marker and Long cone PeriApical radiograph
• Drill stops [44]
C. Post-operative care should attend to
• Early post-operative recognition of neuropathy (HOMECHECK).
• Prompt management of neuropathy (removal of implant if indicated) [44]
– Acute phase
– Late phase
• Early or late post-operative infection

- *Post-operatively*.
- take appropriate post-operative periapical radiographs (CBCTs not indicated) to confirm proximity and/or breech of the inferior dental canal or around the mental foramen before patient discharge.
- If nerve injury is suspected or identified, the patient should be informed, Immediate removal of the implant should be arranged and appropriate medical management should be instituted with arranged review with the treating clinician and specialist if required.
- HOMECHECK -Contact must be made by the clinician and the patient between 6 and 24 h after surgery to confirm that the patient is experiencing any persistent neuropathy. This builds on the relationship of the clinician with the patient, which will be premised upon good consent process.
  - Continue to support and reassure your patient and advise them to visit return to your clinic. If the neuropathy affects most of the dermatome ± associated with severe neuropathic pain, nerve injury must be suspected.
  - Say SORRY this is NOT an admission of guilt. When neuropathy is confirmed, check who you must notify as in many countries, IANIs are reportable events. It is essential to be honest with your patient.
  - Additional scanning or radiography may not be essential. Post-traumatic neuropathy is a clinical diagnosis. You will already be aware of the proximity of the implant bed to the IDC, and whether there was likely breach into the IAN canal.
  - If nerve injury is suspected, the implant must be removed within 24–36 hours of placement in order to maximize recovery from nerve injury [37].
  - Arrange a review of the patient to confirm neuropathy.
  - Initiate medical management (recommended for other peripheral sensory nerve injuries).
    High-dose oral NSAIDs (400–800 mgs Ibuprofen PO QDS) for maximum 2 days.
    GMP (General Medical Practitioner) prescription for Prednisolone 5 day step down does 50-40-30-20-10 mg PO (not for patients with contraindications for steroids or NSAIDs).
    Vitamin B complex (Riboflavin 400 mg once daily for a maximum of 3 months plus other Vit B complex).
- Arrange a further review of your patient.
- Long-term management of patients with non-resolving nerve injuries. The reality for these patients is that if they have persistent neuropathic pain and have to be treated as such with psychological and medical management. Psychological interventions play a significant role in managing these patients, and recommendations for treatment of trigeminal neuropathic pain are also well described by Renton & Zakzrewska [36].

25.4 Mandibular Third Molar Extraction-related Nerve Injuries

The nerves at risk of damage in mandibular third molar extraction are the terminal nerves of the third branch of the trigeminal nerve [53, 54], i.e. the inferior dental nerve (IDN) and lingual nerve (LN). The reported risk of neurosensory deficit ranges from 0.26 to 8.4% for IDN [55] and from 0.1 to 22% for LN [7]. Patients with IDN injury suffer from paresthesia, anaesthesia or dysesthesia in the lip, chin or gingiva on the affected side, while patients with LN injury have a sensitivity deficit at the homolateral half of the tongue, with or without taste alteration [7]. Transient and permanent lesions should be differentiated; permanent lesions often remain after 6–12 months, and spontaneous recovery cannot be expected in these cases [56].

Damage to the LN or IDN during third molar extraction is among the most frequent causes of litigation in dentistry [57]. Highly varying results have been published by numerous studies on risk factors related to neurosensory deficit in lower third molar surgery. The objective of a recent literature review was to identify and analyse studies on factors related to IDN and/or LN injury in lower third molar extraction, allowing clinicians to take appropriate measures to minimize this risk [58]. Several radiological risk factors have been identified that increases the risk of nerve injury during removal by ten-fold (from 0.2 to 2% permanent injury and 2–20% temporary nerve injury) [59, 60].

Key Facts

Factors that may be implicated in nerve injury after lower third molar surgery were classified into four groups:

- Risk assessment—diagnostic radiographic techniques.
- IDN injury risk factors.
- LN injury risk factors.
- Alternative surgical approaches.

25.4.1 Risk Assessment

A recent review included three cohort studies and various randomized clinical trials (RCTs) on the influence of diagnostic radiographic techniques. They generally reached similar conclusions, finding that the non-utilization of CBCT was not an additional risk factor for nerve injury in patients examined by conventional panoramic radiography [61–63].

Korkmaz et al. [64] and Lee et al. [65] reported a lower frequency of transient but not permanent IDN damage when

CBCT was also used. This may be because in cases where the relationship between third molar and the IDN is doubtful, there is likely to be no direct contact and the injury would result from pressure due to haemorrhage or haematoma so that the association would be less detectable on panoramic radiology. In contrast, cases of direct contact are readily observed using both radiographic techniques.

25.4.2 Patient Factors

Various authors reported a significantly lower frequency of nerve injury with younger age. [66–70] Thus, no cases of nerve injury were observed among patients under 23 years of age in the cohort study of 1050 patients by Zhang et al. [71], while Kjolle et al. [66] confirmed a significant association with age ($p = 0.007$), finding a higher frequency of permanent injury in patients over 30 years of age. These findings may be attributable to an increased difficulty of the surgery at older ages due to a greater likelihood of hypercementosis, lower bone elasticity and, above all, completed root formation, in addition to lesser vascularization, reducing the regenerative capacity of the nerve. Nevertheless, other researchers found no significant relationship with age [72] although the sample sizes were smaller than that in the aforementioned studies. All reviewed articles observed a higher frequency of nerve injuries in females [62] although this difference was only statistically significant ($p = 0.005$) in the multiple logistic regression analysis of 320 cases conducted by Selvi et al. [68] Gender differences have been attributed to the generally smaller mandible of females, implying a smaller gap between third molar root and IDN.

25.4.3 Anatomical

The mandibular canal is evidently more susceptible to nerve injury with greater depth and, therefore, closer proximity of the impacted third molar, reducing the surgical accessibility and visibility. A statistically significant association was demonstrated by all three articles that studied this risk factor [62]. A higher risk of IDN injury was associated with mesioangular impactions and with horizontal impactions [62], but these associations were not found to be statistically significant.

25.4.4 Radiological Factors

In 1990, Rood and Shehab [59] proposed seven radiological signs identifiable by panoramic radiography, which indicate a close relationship between lower third molar and IDN: root narrowing, root darkening, apex darkening and bifid images, changes in root direction, dental canal narrowing, dental canal diversion and interruption of the white line of the dental canal. Only four of these signs were reported to be significant indicators of IDN risk in the reviewed articles: interruption of the radiopaque band of the canal [62, 66, 69, 73–76], canal diversion [59, 74, 76], root darkening [59, 62, 74, 76] and mandibular canal narrowing. [70, 74, 76] In contrast, a retrospective study by Pippi et al. [72] found that none of these signs were significantly associated with nerve injury, even when two or more were observed. CBCT radiological signs have also been associated with IDN damage. Detection of contact between lower third molar and mandibular canal has been found to potentially influence the resulting nerve damage, [62, 68, 69, 72, 77] which is associated by Kim et al. [70] with a 21-fold higher risk of paraesthesia. Various studies [67, 69, 73, 78, 79] have associated nerve injury with the lingual position of the mandibular canal with respect to the third molar root, attributed to the more likely interruption of the mandibular canal cortex due to the direction of extraction manoeuvres. The RCT reported by Ghaeminia et al. [62] found the risk of nerve injury to be 16-fold higher when the localization was lingual versus buccal. In addition, some authors have described a higher risk of IDN injury for dumbbell-shaped versus round-, oval- or drop-shaped canals [72, 73, 76, 77].

Key Facts

There are seven radiological signs identifiable by panoramic radiography, which indicate a close relationship between lower third molar and IDN: root narrowing, root darkening, apex darkening and bifid images, changes in root direction, dental canal narrowing, dental canal diversion and interruption of the white line of the dental canal.

25.4.5 Surgical

Two studies related the type of anaesthesia to IDN injury. Nyugen et al. [67] found a significantly higher ($p = 0.007$) frequency of permanent damage in lower third molar surgery under general versus local anaesthesia, and Costantinides et al. [80] reported a 2–16-fold greater risk of IDN injury under the former. One explanation is that the absence of patient feedback with general anaesthesia means that surgeons are less aware of the force applied. Hasegawa et al. [69] observed a significantly higher ($p < 0.05$) IDN injury rate in patients with versus without nerve exposure during the surgery. However, Pippi et al. [72] reported nerve injuries in only 6.5% of cases in which the nerve was exposed versus

9.3% of cases in which it was not, suggesting that IDN exposure may simply reflect the close proximity of tooth and nerve and cannot per se be considered an indicator of potential nerve damage. Three studies associated haemorrhage during third molar extraction with IDN injury [62, 75, 81], without elucidating whether the bleeding resulted from mandibular canal fracture or a haematoma or other causes of nerve compression.

With respect to the experience of the clinician, Nguyen et al. [67] found a significantly higher frequency of permanent IDN injury ($p = 0.026$) amongst inexperienced dentists in comparison to oral specialists or maxillofacial surgeons, possibly related to inappropriate force and less instrumental control in the hands of those with less experience. The same study also explored the effect of surgery duration, finding a higher nerve injury rate when this was more than 20 min (from incision to completed tooth extraction), mainly because a longer surgical time implies a more challenging extraction. With regard to the surgical approach, Jain et al. [73] reported a significantly ($p = 0.04$) higher nerve injury rate in patients who underwent odontosection versus those who did not. This may be explained by the less extensive ostectomy often associated with this procedure although odontosection can be a direct risk factor for IDN injury in the extraction of horizontal third molars [82].

25.5 LN Injury Risk Factors

Demographics A prospective study by Charan Babu et al. [83] reported that older age was a significant risk factor for LN injury ($p < 0.05$), but Kjoelle et al. [66] found no differences in permanent nerve damage amongst age groups. No significant gender differences in LN injury rate were found in any study.

Anatomical Charan Babu et al. [83] observed a significantly ($p < 0.01$) higher risk of LN injury with greater impaction depth, attributed to the more difficult extraction and, therefore, more extensive osteotomy. A higher LN injury rate was observed for distoangular impactions [67, 83] generally due to the more difficult extraction and for horizontal extractions [83], possibly because of the larger amount of bone removed. However, these associations were not statistically significant.

Surgical Charan Babu et al. [83], Osunde et al. [84] and Yadav et al. [85] reported a significantly higher LN injury rate ($p < 0.01$, $p < 0.001$ and $p < 0.001$, respectively) in patients who had undergone lingual flap retraction before third molar extraction than in those who had not.

This injury was found to be transient in the RCT by Shad et al. [86], who suggested that permanent injury can be produced when the lingual flap is not separated from the bone. Three studies [62, 85, 87] observed a significant association between higher LN injury risk and the requirement for odontosection in third molar surgery.

25.6 Role of Alternative Surgical Techniques

Various authors have proposed alternative surgical techniques to avoid nerve damage in lower third molar extraction, but their findings should be considered with caution due to major study limitations (e.g. no control group and small sample size), and there has been little research on this issue. In a study of 53 patients, Bataineh et al. [88] reported a modified flap that appeared to reduce LN lesions caused by flap retraction, considering all known anatomical variations of LN. Ge et al. [89] observed lower nerve injury rates when type III lower third molars in lingual position were extracted by piezosurgery in a lingual split approach in comparison to published rates reported using the conventional lingual split technique. The persistence in defending lingual access third molar surgery is inappropriate, in that it significantly increases the risk of temporary lingual nerve injury and 10–12% of these injuries will be permanent [90]. A recent literature review recommends avoidance of lingual flaps in third molar surgery to minimize lingual nerve injury [91].

A recent surgical technique to mitigate the risk of high risk third molars in close proximity to the IDC is a coronectomy. Coronectomy, in which the dental crown is removed and the root is retained in the jaw, has been recommended to reduce IDN injury risk in cases of close proximity between nerve and third molar, and the majority of the reviewed studies described any IDN injury with the utilization of this approach [60]. Nevertheless, this technique is not free of controversy, even when clearly indicated, given the possibility of infectious complications around the root or its migration [92–95]. Extraction by orthodontic traction may also be useful when there is a high risk of nerve injury, and Wang et al. [96] found no cases of nerve injury in patients undergoing this procedure although account should be taken of study design limitations and the small sample size. There is a strong evidence base to support the prevention of IDN nerve injuries using the coronectomy technique [43, 44, 53, 54, 97].

Table 25.3 Risk factors for third molar surgery-related nerve injury

Lingual nerve injury	
	Increased patient age
	Increased duration surgery
	Lingual access surgery
	Inexperience of surgeon
	Distoangulation of third molar
	Depth of impaction
Inferior alveolar nerve injury	
	Proximity of tooth root to inferior dental canal
	Increased patient age
	Increased duration surgery
	Inexperience of surgeon
	Distoangulation of third molar
	Depth of impaction

Summary prevention of third molar surgery-related nerve injury (Table 25.3) [98, 99].

- Nerve injury risk does not appear to be influenced by the diagnostic imaging technique used (CBCT or panoramic radiograph) although the utilization of CBCT may possibly reduce the risk of transient injuries. Studies with larger sample sizes are needed to clarify this issue.
- Older age, female sex, mesioangular position, impaction depth, utilization of general versus local anaesthesia, haemorrhage, inexperience of the clinician and certain signs on panoramic radiography and CBCT appear to be associated with a higher risk of IDN injury.
- Lingual flap retraction, older age, horizontal and distoangular positions, impaction depth and odontosection may be possible risk factors for LN injury.
- Coronectomy is evidence based to prevent IDN injuries in selected cases, High-risk third molar, healthy and cooperative patients and vital tooth.
- Definitive conclusions are limited by the variability in study design (with the inclusion of some retrospective studies), reduced sample sizes and differences in the experience of clinicians, amongst other factors. However, buccal approach technique (when undertaken properly) will minimize lingual nerve injury.

25.7 Prognosis of Nerve Injuries (Table 25.4) [43]

It is not possible to classify the degree or outcome of a sensory nerve injury based on patients' presentation early post-injury. Just as with phantom limb pain patients, who may express non-existence or existence of a 'normal feeling' limb (after amputation, the most catastrophic nerve injury) with or without pain, numbness or altered sensation, these symptoms do not reflect the degree of injury or prognosis. Thus, in order to assess the end results of nerve injury, the patient must be reassessed and/or treated if indicated. The type and

Table 25.4 Resolution rates of inferior alveolar nerve injury (IANI)

Procedure	Recovery rate
Third molar surgery [7]	IANI – 67%; LNI – 72% Buccal access TMS LIN – Lingual access TMS 88%
Mandibular fractures [7]	IANI – 91%
Orthognathic surgery	IANI – 87% Bilateral sagittal split osteotomy (BSSO) IANI (patients 80–92%)
Local anaesthesia inferior dental block (mainly Lidocaine) [14]	75%
Implant-related IANI [87]	Complete recovery – 50% Partial recovery – 44% No change – 6%

A review of common operations such as groin hernia repair, breast and thoracic surgery, leg amputation and coronary artery bypass surgery found an incidence of chronic post-surgical pain in 10–50% of patients [100].

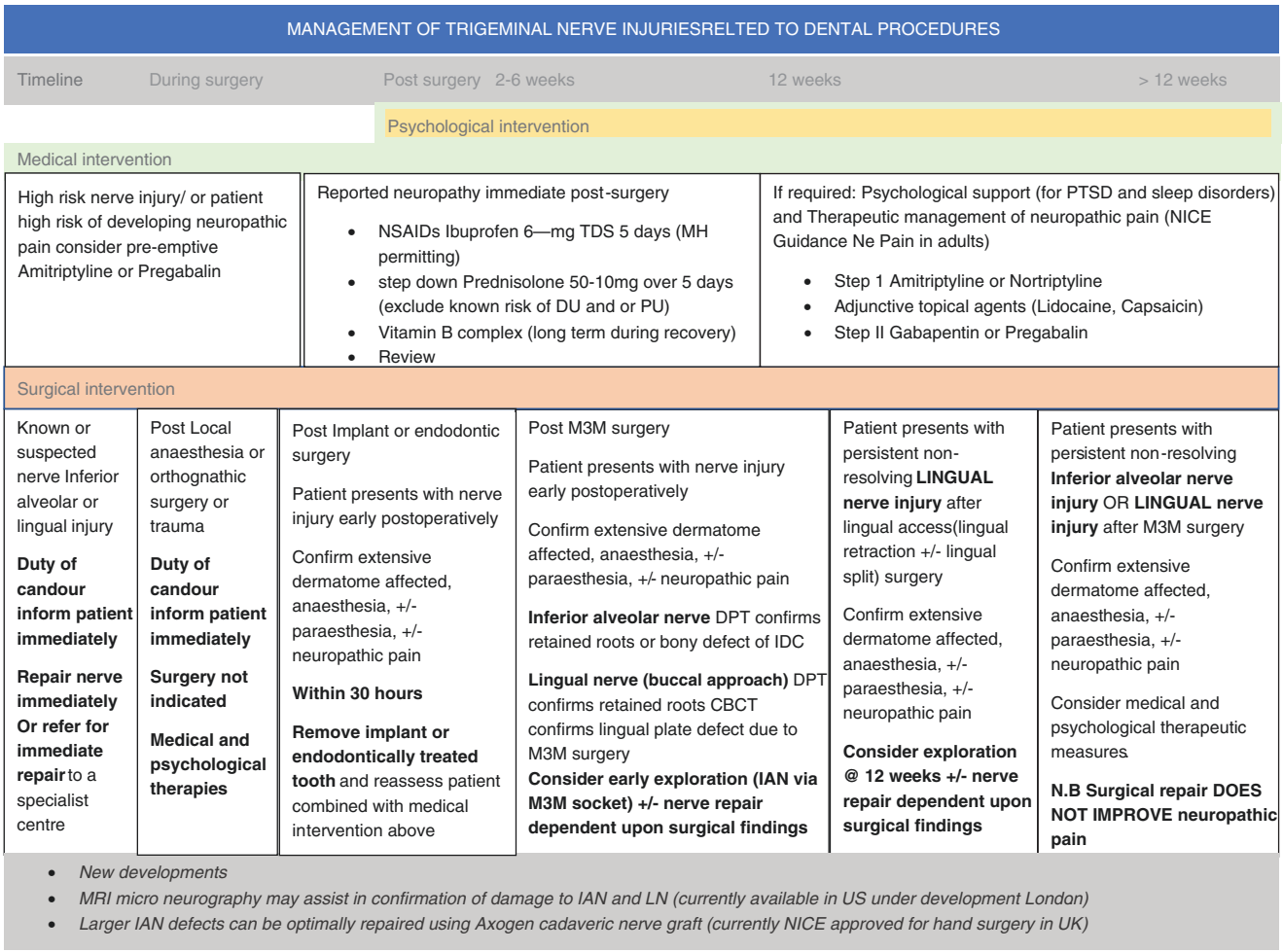
Table 25.5 Timing for intervention of Trigeminal nerve injury

Event	Recovery	
Endodontic	<24–36 h	Remove tooth and remove over fill or over instrumentation
Implant	<24–36 h	Remove implant
Wisdom teeth- inferior alveolar nerve injury	<2 weeks	Consider earlier intervention
Radiographic evidence of retained tooth fragments or IDC damage		Access via extraction socket and remove retained roots ± repair nerve
Wisdom teeth -lingual nerve injury	>3–6 months	Consider earlier intervention
If CBCT confirmation of breech, lingual plate	Consider earlier intervention	Access via extraction socket and remove retained roots ± repair nerve
Local anaesthetic nerve injuries (LN or IAN)		Therapeutic management only
Orthognathic nerve injuries		Therapeutic management only
Mandibular fracture nerve injuries		Therapeutic management only

A known or suspected sectioned/damaged nerve should undergo immediate exploration repair

related permanency of trigeminal nerve injuries are summarized in Table 25.4.

Summary of type and timing of management (Table 25.5 and Fig. 25.2) Management of third molar-related nerve injuries will depend upon the presentation of the patient (pain, functional and psychological implications) duration and cause of the nerve injury [56, 98]. Figure 25.2 summarizes the management and timing of intervention for trigeminal nerve injuries based upon the current evidence base [56, 98]. It is recognized that neuropathic pain does not respond to surgical intervention, and thus, prevention and early management are paramount in preventing chronic life-long pain after routine surgery in these patients. Advice is summarized on the Trigeminalnerve.org.uk website.



©Association of Oral and Maxillofacial Surgeons of India

Fig. 25.2 suggested management of nerve injuries related to mandibular third molar surgery

The patient with the nerve injury must be treated, NOT the nerve injury in isolation. The neuropathy, pain, numbness or pareasthesia, with associated functional and psychological impact, will be the driving force behind the patient seeking treatment [98]. These factors must be assessed, and the potential outcomes, good or bad, should be discussed and agreed with the patient [99].

Patients sustaining LA, orthognathic, oncology and trauma-related nerve injuries will mainly be managed therapeutically [101–104].

Overall, there is poor evidence to support late surgical intervention for Trigeminal nerve injuries [6, 105]. Most studies report on repair procedures undertaken too late, and early repair is imperative to minimize central irreversible changes and possibly chronic pain. Generally, surgical repair of the trigeminal nerves never returns the patient to peroperative neural function; in addition, there is a risk of making a numb patient into one with chronic post-surgical pain [100, 106]. As with other post-trauma sensory neuropathies, it is recognized that immediate repair is optimal; [107, 108] however, this is rarely applied to dental nerve injuries with the misconception that we should sit and wait for resolution (only for 3 months for lingual nerve injuries related to lingual access third molar surgery), resulting in long delays before surgical intervention [109–111].

Some recent studies have highlighted immediate repair with cadaveric-treated human nerve graft successful in managing various sized defects in planned resection of nerves related to benign tumour resection or trauma [112, 113].

Recent reports have also concluded that, similar to other surgical sites, neuropathic pain does not resolve with surgery, with this being the main driver for surgical repair [114, 115].

Many reports have recommended the use of conduits (venous, prosthetic), sural nerve grafts and other techniques without sufficient evidence and many with poor outcomes including neuropathy and pain from the donor sites! The future may prove that nerve growth factors, other growth-promoting chemical and anti-neuropathic pain agents and specialized conduits may play a role in improving

repair of trigeminal nerve injuries, and the overall conclusion from reviews in this area is that we have a lot of evidence base to harness [105, 106, 116]. The singular consensus is that prevention of these nerve injuries is possible and optimal.

The timing of intervention and mechanism of injury are paramount in decision making in the treatment of trigeminal nerve injuries (summarized in Table 25.5).

1. Counselling is the most useful effective tool for managing patients with problematic permanent sensory nerve injuries.
2. Medical intervention is indicated for patients with pain or discomfort or with anxiety and/or depression in relation to chronic pain. However, due to the multiple noxious side effects of chronic pain medication, less than 18% of patients remain adherent with medication.
   - acute (medical),
   - late (chronic pain management with psychological interventions).
3. Surgical intervention is indicated for:
   - Immediate surgical repair for suspected or known nerve injury or intended surgical defect after removal of benign tumour or recent trauma [98].
   - Removal of implant.
   - or overfill or RCT-treated tooth with 36 h if related to the development of neuropathy [99].
   - Within 2–4 week, exploration if clinical presentation of persistent neuropathy is paramount and radiographic follow-up is not necessary; however, if there is CBCT evidence of breech of lingual plate or IDC, consider immediate action-nerve exploration ± repair;
   - Lingual nerve neuropathy patients with CBCT evidence of damage to lingual plate adjacent to third molar surgical site.
   - Inferior alveolar nerve with retained roots or evidence of bone inclusions or compression of IDC.
   - Within 3 months of injury;
   - Non-resolving lingual or inferior dental nerve injuries: Exploratory surgery for lingual or inferior alveolar nerve injuries within 3 months post-injury. Surgical intervention is not effective for neuropathic pain, and if this is the driving force behind seeking surgery, it should be reconsidered.
   - There are reported exciting results of allografting lingual and inferior alveolar nerve injuries. Using a pre-prepared human-treated cadaveric allograft, the IDN and LN can be repaired with minimal tension. This is undertaken using microscopy and described in several publications by John Zuniga and Michael Miloro [109]. This is likely to be the treatment of choice if repair is indicated and direct re-anastomosis cannot be undertaken most commonly for the IDN. One of the main issues regarding nerve repair is the early identification of the neuroma related to the patients 'symptoms and the connectivity of the nerve itself, i.e. is the nerve actually functioning. Recent developments with Magnetic Resonance Neurography (MRN) have availed the surgeon to identify the nerve lesion and neural functionality to facilitate appropriate and earlier nerve repair intervention [117, 118].

25.8 Conclusions

Unfortunately, none of these interventions 'fix' the patient, but the aim is to manage their symptoms as best as possible, improve function and allow them time to accommodate to these unfortunate events, which is often not very satisfactory.

This chapter was intended to acknowledge and share some key issues around iatrogenic trigeminal nerve injuries and to provide some key take home messages including:

- Neuropathic pain as well as altered sensation and numbness is what most patients experience with iatrogenic sensory nerve injury. This has a significant and unpleasant effect on the patient (improve your consent!)
- The majority of iatrogenic nerve injuries are avoidable.
- Inferior alveolar nerve injuries in relation to implant and endodontic dentistry are permanent and 'unfixable' unless treated quickly within 30 h.
- Owing to the significant problems following nerve injury, pre-operative strategies for minimizing this risk of nerve damage need to be considered carefully. Peri-operative planning, operative execution and post-operative care need improving to minimize and hopefully abolish these injuries.
- Several strategies are presented to assist in preventing nerve injuries.
- There is a need for a consensus and standardization of risk assessment and management, a holistic approach in managing the pain, related effect on functionality and psychological implications caused to the patients affected by iatrogenic nerve injury.

References

1. Caissie R, Goulet J, Fortin M, Morielle D. Iatrogenic paresthesia in the third division of the trigeminal nerve: 12 years of clinical experience. J Can Dent Assoc. 2005;71(3):188–92.
2. Hillerup S. Iatrogenic injury to oral branches of the trigeminal nerve: records of 449 cases. Clin Oral Investig. 2007 Jun;11(2):133–42. Epub 2006 Dec 22.

3. Elian N, Mitsias M, Eskow R, Jalbout ZN, Cho SC, Froum S, Tarnow DP. Unexpected return of sensation following 4.5 years of paresthesia: case report. Implant Dent. 2005 Dec;14(4):364–7.
4. Loescher AR, Robinson PP. The effect of surgical medicaments on peripheral nerve function. Br J Oral Maxillofac Surg. 1998 Oct;36(5):327–32.
5. Hegedus F, Diecidue RJ. Trigeminal nerve injuries after mandibular implant placement--practical knowledge for clinicians. Int J Oral Maxillofac Implants. 2006;21(1):111–6.
6. Ziccardi VB, Assael LA. Mechanisms of trigeminal nerve injuries. Atlas Oral Maxillofac Surg Clin North Am. 2001;9(2):1–11.
7. Renton T, Yilmaz Z. Profiling of patients presenting with post-traumatic neuropathy of the trigeminal nerve. J Orofac Pain. 2011 Fall;25(4):333–44.
8. Carter E, Yilmaz Z, Devine M, Renton T. An update on the causes, assessment and management of third division sensory trigeminal neuropathies. Br Dent J. 2016 Jun;220(12):627–35.
9. Renton T, Adey-Viscuso D, Meechan JG, Yilmaz Z. Trigeminal nerve injuries in relation to the local anaesthesia in mandibular injections. Br Dent J. 2010;209(9):E15. https://doi.org/10.1038/sj.bdj.2010.978.
10. Zuniga JR, Yates DM, Phillips CL. The presence of neuropathic pain predicts postoperative neuropathic pain following trigeminal nerve repair. J Oral Maxillofac Surg. 2014 Dec;72(12):2422–7.
11. Leung YY, Cheung LK. Longitudinal treatment outcomes of microsurgical treatment of neurosensory deficit after lower third molar surgery: a prospective case series. PLoS One. 2016 Mar 4;11(3):e0150149.
12. Smith JG, Elias LA, Yilmaz Z, Barker S, Shah K, Shah S, et al. The psychosocial and affective burden of posttraumatic neuropathy following injuries to the trigeminal nerve. J Orofac Pain. 2013;27(4):293–303.
13. Renton T, Janjua H, Gallagher JE, Dalgleish M, Yilmaz Z. Uk dentists' experience of iatrogenic trigeminal nerve injuries in relation to routine dental procedures: why, when and how often? Br Dent J. 2013;214(12):633–42. https://doi.org/10.1038/sj.bdj.2013.583.
14. Pogrel MA, Schmidt BL, Sambajon V, Jordan RC. Lingual nerve damage due to inferior alveolar nerve blocks: a possible explanation. J Am Dent Assoc. 2003;134(2):195–9.
15. Smith MH, Lung KE. Nerve injuries after dental injection: a review of the literature J Can Dent Assoc 2006 Jul–Aug;72(6):559–564. Review.
16. Pogrel MA, Thamby S. Permanent nerve involvement resulting from inferior alveolar nerve blocks. J Am Dent Assoc. 2000;131(7):901–7.
17. Haas DA, Lennon D. A 21 year retrospective study of reports of paresthesia following local anesthetic administration. J Can Dent Assoc. 1995;61(4):319–20, 323–316, 329–330
18. Hillerup S. Iatrogenic injury to oral branches of the trigeminal nerve: records of 449 cases. Clin Oral Investig. 2007;11(2):133–42.
19. Hillerup S, Jensen R. Nerve injury caused by mandibular block analgesia. Int J Oral Maxillofac Surg. 2006;35(5):437–43. https://doi.org/10.1016/j.ijom.2005.10.004.
20. Pedlar J. Prolonged paraesthesia. Br Dent J. 2003;195(3):119. https://doi.org/10.1038/sj.bdj.4810421.
21. Perez-Castro R, Patel S, Garavito-Aguilar ZV, Rosenberg A, Recio-Pinto E, Zhang J, Blanck TJ, Xu F. Cytotoxicity of local anesthetics in human neuronal cells. Anesth Analg. 2009 Mar;108(3):997–1007.
22. Kämmerer PW, Schneider D, Palarie V, Schiegnitz E, Daubländer M. Comparison of anesthetic efficacy of 2 and 4% articaine in inferior alveolar nerve block for tooth extraction-a double-blinded randomized clinical trial. Clin Oral Investig. 2017 Jan;21(1):397–403.
23. Daubländer M, Kämmerer PW, Willershausen B, Leckel M, Lauer HC, Buff S, Rösl B. Clinical use of an epinephrine-reduced (1/400,000) articaine solution in short-time dental routine treatments--a multicenter study. Clin Oral Investig. 2012 Aug;16(4):1289–95.
24. Evans G, Nusstein J, Drum M, Reader A, Beck M. A prospective, randomized, double-blind comparison of articaine and lidocaine for maxillary infiltrations. J Endod. 2008 Apr;34(4):389–93.
25. Oliveira PC, Volpato MC, Ramacciato JC, Ranali J. Articaine and lignocaine efficiency in infiltration anaesthesia: a pilot study. Br Dent J. 2004 Jul 10;197(1):45–6.
26. Vähätalo K, Antila H, Lehtinen R. Articaine and lidocaine for maxillary infiltration anesthesia. Anesth Prog. 1993;40(4):114–6.
27. Srinivasan N, Kavitha M, Loganathan CS, Padmini G. Comparison of anesthetic efficacy of 4% articaine and 2% lidocaine for maxillary buccal infiltration in patients with irreversible pulpitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009 Jan;107(1):133–6.
28. Meechan JG The use of the mandibular infiltration anesthetic technique in adults. J Am Dent Assoc. 2011 Sep;142 Suppl 3:19S–24S. Review.
29. Zain M, et al. Comparison of Anaesthetic efficacy of 4% Articaine primary Buccal infiltration versus 2% Lidocaine inferior alveolar nerve block in symptomatic mandibular first molar teeth. J Coll Physicians Surg Pak. 2016 Jan;26(1):4–8.
30. Poorni S, et al. Anesthetic efficacy of four percent articaine for pulpal anesthesia by using inferior alveolar nerve block and buccal infiltration techniques in patients with irreversible pulpitis: a prospective randomized double-blind clinical trial. J Endod. 2011 Dec;37(12):1603–7.
31. Kung J, McDonagh M, Sedgley CM. Does articaine provide an advantage over lidocaine in patients with symptomatic irreversible pulpitis? A systematic review and meta-analysis. J Endod. 2015;41(11):1784–94.
32. Thakare A, Bhate K, Kathariya R. Comparison of 4% articaine and 0.5% bupivacaine anesthetic efficacy in orthodontic extractions: prospective, randomized crossover study. Acta Anaesthesiol Taiwanica. 2014 Jun;52(2):59–63.
33. Etoz OA, Er N, Demirbas AE. Is supraperiosteal infiltration anesthesia safe enough to prevent inferior alveolar nerve during posterior mandibular implant surgery? Med Oral Patol Oral Cir Bucal. 2011 May 1;16(3):e386–9.
34. Dumbrigue HB, Lim MV, Rudman RA, Serraon A. A comparative study of anesthetic techniques for mandibular dental extraction. Am J Dent. 1997 Dec;10(6):275–8.
35. Khawaja N, Yilmaz Z, Renton T. Case studies illustrating the management of trigeminal neuropathic pain using topical 5% lidocaine plasters. Br J Pain. 2013;7(2):107–13.
36. Renton T, Zakrzewska JM Chapter 22 Orofacial pain. In: Shaw I, Kumar C, Dodds C, editors. Oxford textbook of anaesthesia for oral and maxillofacial surgery. Oxford: Oxford University Press; 2010. p. 283–298 isbn:978-0-19-956421-7.
37. Khawaja N, Renton T. Case studies on implant removal influencing the resolution of inferior alveolar nerve injury. Br Dent J. 2009;206(7):365–70. https://doi.org/10.1038/sj.bdj.2009.258.
38. Alhassani AA, AlGhamdi AS. Inferior alveolar nerve injury in implant dentistry: diagnosis, causes, prevention, and management. J Oral Implantol. 2010;36(5):401–7.
39. Fukuda K, Ichinohe T, Kaneko Y. Pain management for nerve injury following dental implant surgery at Tokyo dental college hospital. Int J Dent. 2012;2012:209474. https://doi.org/10.1155/2012/209474.
40. Renton T, Dawood A, Shah A, Searson L, Yilmaz Z, Renton T, Yilmaz Z. Post-implant neuropathy of the trigeminal nerve. A case series. Br Dent J. 2012;212(11):E17. https://doi.org/10.1038/sj.bdj.2012.497.
41. Juodzbalys G, Kubilius M. Clinical and radiological classification of the jawbone anatomy in endosseous dental implant treatment. J

Oral Maxillofac Res. 2013 Jul 1;4(2):e2. https://doi.org/10.5037/jomr.2013.4202.N.
42. Harris D, Horner K, Gröndahl K, Jacobs R, Helmrot E, Benic GI, Bornstein MM, Dawood A, Quirynen M. EAO guidelines for the use of diagnostic imaging in implant dentistry 2011. A consensus workshop organized by the European Association for Osseointegration at the Medical University of Warsaw. Clin Oral Implants Res. 2012 Nov;23(11):1243–53.
43. Leung YY, Cheung LK. Coronectomy of the lower third molar is safe within the first 3 years. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2012;70(7):1515–22.
44. Cervera-Espert J, Pérez-Martínez S, Cervera-Ballester J, Peñarrocha-Oltra D, Peñarrocha-Diago M. Coronectomy of impacted mandibular third molars: a meta-analysis and systematic review of the literature. Med Oral Patol Oral Cir Bucal. 2016 Jul 1;21(4):e505–13.
45. Greenstein G, Tarnow D. The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review. J Periodontol. 2006 Dec;77(12):1933–43.
46. Thoma DS, Cha JK, Jung UW. Treatment concepts for the posterior maxilla and mandible: short implants versus long implants in augmented bone. J Periodontal Implant Sci. 2017 Feb;47(1):2–12.
47. Bartlett G, Mansoor J. Articaine buccal infiltration vs lidocaine inferior dental block – a review of the literature. Br Dent J. 2016 Feb 12;220(3):117–20.
48. Al-Ani O, Nambiar P, Ha KO, Ngeow WC. Safe zone for bone harvesting from the interforaminal region of the mandible. Clin Oral Implants Res. 2013 Aug;24(Suppl A100):115–21.
49. Van Assche N, van Steenberghe D, Guerrero ME, Hirsch E, Schutyser F, Quirynen M, Jacobs R. Accuracy of implant placement based on pre-surgical planning of three-dimensional cone-beam images: a pilot study. J Clin Periodontol. 2007;34(9):816–21.
50. De Kok IJ, Thalji G, Bryington M, Cooper LF. Radiographic stents: integrating treatment planning and implant placement Dent Clin N Am 2014 Jan;58(1):181–192. https://doi.org/10.1016/j.cden.2013.09.008. Review.
51. Burstein J, Mastin C, Le B. Avoiding injury to the inferior alveolar nerve by routine use of intraoperative radiographs during implant placement. J Oral Implantol. 2008;34(1):34–8.
52. Greenstein G, Greenstein B, Desai RN. Using drill stops on twist drills to promote safety and efficiency when creating osteotomies for dental implants. J Am Dent Assoc. 2014 Apr;145(4):371–5.
53. Szalma J, Lempel E. Protecting the inferior alveolar nerve: coronectomy of lower third molars. Rev Orv Hetil. 2017 Nov;158(45):1787–93.
54. Ali AS, Benton JA, Yates JM. Risk of inferior alveolar nerve injury with coronectomy vs surgical extraction of mandibular third molars-a comparison of two techniques and review of the literature. J Oral Rehabil. 2018 Mar;45(3):250–7.
55. Cheung LK, Leung YY, Chow LK, Wong MC, Chan EK, Fok YH. Incidence of neurosensory deficits and recovery after lower third molar surgery: a prospective clinical study of 4338 cases. Int J Oral Maxillofac Surg. 2010;39(4):320–6.
56. Renton T, Yilmaz Z. Managing iatrogenic trigeminal nerve injury: a case series and review of the literature. Int J Oral Maxillofac Surg. 2012;41(5):629–37. https://doi.org/10.1016/j.ijom.2011.11.002.
57. Koskela S, Suomalainen A, Apajalahti S, Venta I. Malpractice claims related to tooth extractions. Clin Oral Investig. 2017 Mar;21(2):519–22.
58. Leung YY, Cheung LK. Risk factors of neurosensory deficits in lower third molar surgery: a literature review of prospective studies. Int J Oral Maxillofac Surg. 2011;40(1):1–10.
59. Rood JP, Shehab BA. The radiological prediction of inferior alveolar nerve injury during third molar surgery. Br J Oral Maxillofac Surg. 1990;28(1):20–5.
60. Renton T. Notes on coronectomy. Br Dent J. 2012;212:323–6.
61. Petersen LB, Vaeth M, Wenzel A. Neurosensory disturbances after surgical removal of the mandibular third molar based on either panoramic imaging or cone beam CT scanning: a randomized controlled trial (RCT). Dentomaxillofac Radiol. 2016;45(2):20150224.
62. Ghaeminia H, Gerlach NL, Hoppenreijs TJ, Kicken M, Dings JP, Borstlap WA, et al. Clinical relevance of cone beam computed tomography in mandibular third molar removal: a multicentre, randomised, controlled trial. J Cranio-Maxillo-Fac Surg: Official Publication of the European Association for Cranio-Maxillo-Fac Surgery. 2015;43(10):2158–67.
63. Matzen LH, Schou S, Christensen J, Hintze H, Wenzel A. Audit of a 5-year radiographic protocol for assessment of mandibular third molars before surgical intervention. Dentomaxillofac Radiol. 2014;43(8):20140172.
64. Korkmaz YT, Kayipmaz S, Senel FC, Atasoy KT, Gumrukcu Z. Does additional cone beam computed tomography decrease the risk of inferior alveolar nerve injury in high-risk cases undergoing third molar surgery? Does CBCT decrease the risk of IAN injury? Int J Oral Maxillofac Surg. 2017;46(5):628–35.
65. Lee B, Park Y, Ahn J, Chun J, Park S, Kim M, et al. Assessment of the proximity between the mandibular third molar and inferior alveolar canal using preoperative 3D-CT to prevent inferior alveolar nerve damage. Maxillofac Plast Reconstr Surg. 2015;37(1):30.
66. Kjolle GK, Bjornland T. Low risk of neurosensory dysfunction after mandibular third molar surgery in patients less than 30 years of age. A prospective study following removal of 1220 mandibular third molars. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116(4):411–7.
67. Nguyen E, Grubor D, Chandu A. Risk factors for permanent injury of inferior alveolar and lingual nerves during third molar surgery. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2014;72(12):2394–401.
68. Selvi F, Dodson TB, Nattestad A, Robertson K, Tolstunov L. Factors that are associated with injury to the inferior alveolar nerve in high-risk patients after removal of third molars. Br J Oral Maxillofac Surg. 2013;51(8):868–73.
69. Hasegawa T, Ri S, Shigeta T, Akashi M, Imai Y, Kakei Y, et al. Risk factors associated with inferior alveolar nerve injury after extraction of the mandibular third molar--a comparative study of preoperative images by panoramic radiography and computed tomography. Int J Oral Maxillofac Surg. 2013;42(7):843–51.
70. Kim JW, Cha IH, Kim SJ, Kim MR. Which risk factors are associated with neurosensory deficits of inferior alveolar nerve after mandibular third molar extraction? J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2012;70(11):2508–14.
71. Zhang QB, Zhang ZQ. Early extraction: a silver bullet to avoid nerve injury in lower third molar removal? Int J Oral Maxillofac Surg. 2012;41(10):1280–3.
72. Pippi R, Santoro M. A multivariate statistical analysis on variables affecting inferior alveolar nerve damage during third molar surgery. Br Dent J. 2015;219(4):E3.
73. Jain N, Thomas S, Prabhu S, Jain S, Pathak AD, Pillai A, et al. Influence of tooth sectioning technique and various risk factors in reducing the IAN injury following surgical removal of an impacted mandibular third molar. Oral Maxillofac Surg. 2016;20(2):149–56.
74. Huang CK, Lui MT, Cheng DH. Use of panoramic radiography to predict postsurgical sensory impairment following extraction of impacted mandibular third molars. J Chin Med Assoc. 2015;78(10):617–22.
75. Pathak S, Mishra N, Rastogi MK, Sharma S. Significance of radiological variables studied on orthopantomogram to predict

post-operative inferior alveolar nerve paresthesia after third molar extraction. J Clin Diagn Res. 2014;8(5):ZC624.
76. Ueda M, Nakamori K, Shiratori K, Igarashi T, Sasaki T, Anbo N, et al. Clinical significance of computed tomographic assessment and anatomic features of the inferior alveolar canal as risk factors for injury of the inferior alveolar nerve at third molar surgery. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2012;70(3):514–20.
77. Shiratori K, Nakamori K, Ueda M, Sonoda T, Dehari H. Assessment of the shape of the inferior alveolar canal as a marker for increased risk of injury to the inferior alveolar nerve at third molar surgery: a prospective study. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2013;71(12):2012–9.
78. Ye Z, Yang C, Abdelrehem A. Prediction of inferior alveolar nerve injury in complicated mandibular wisdom teeth extractions: a new classification system. Int J Clin Exp Med. 2016;9(2):3729–34.
79. Kim HG, Lee JH. Analysis and evaluation of relative positions of mandibular third molar and mandibular canal impacts. J Korean Assoc Oral Maxillofac Surg. 2014;40(6):278–84.
80. Costantinides F, Biasotto M, Maglione M, Di Lenarda R. Local vs general anaesthesia in the development of neurosensory disturbances after mandibular third molars extraction: a retrospective study of 534 cases. Med Oral Patol Oral Cir Bucal. 2016;21(6):e724–30.
81. Xu GZ, Yang C, Fan XD, Yu CQ, Cai XY, Wang Y, et al. Anatomic relationship between impacted third mandibular molar and the mandibular canal as the risk factor of inferior alveolar nerve injury. Br J Oral Maxillofac Surg. 2013;51(8):e215–9.
82. Mavrodi A, Ohanyan A, Kechagias N, Tsekos A, Vahtsevanos K. Influence of two different surgical techniques on the difficulty of impacted lower third molar extraction and their post-operative complications. Med Oral Patol Oral Cir Bucal. 2015;20(5):e640–4.
83. Charan Babu HS, Reddy PB, Pattathan RK, Desai R, Shubha AB. Factors influencing lingual nerve paraesthesia following third molar surgery: a prospective clinical study. J Maxillofac Oral Surg. 2013;12(2):168–72.
84. Osunde O, Saheeb B, Bassey G. Indications and risk factors for complications of lower third molar surgery in a Nigerian teaching hospital. Ann Med Health Sci Res. 2014;4(6):938–42.
85. Yadav S, Verma A, Sachdeva A. Assessment of lingual nerve injury using different surgical variables for mandibular third molar surgery: a clinical study. Int J Oral Maxillofac Surg. 2014;43(7):889–93.
86. Shad S, Shah SM, Alamgir AMM. Frequency of lingual nerve injury in mandibular third molar extraction: a comparison of two surgical techniques. J Ayub Med Coll Abbottabad. 2015;27(3):580–3.
87. Agrawal A, Yadav A, Chandel S, Singh N, Singhal A. Wisdom tooth complications in extraction. J Contemp Dent Pract. 2014;15(1):34–6.
88. Bataineh AB, Batarseh RA. The effect of modified surgical flap design for removal of lower third molars on lingual nerve injury. Clin Oral Investig. 2016;21(6):2091–9.
89. Ge J, Yang C, Zheng J, Qian W. Piezosurgery for the lingual split technique in lingual positioned impacted mandibular third molar removal: A retros pective study. Medicine. 2016;95(12):e3192.
90. Blackburn CW, Bramley PA. Lingual nerve damage associated with the removal of lower third molars. Br Dent J. 1989 Aug 5;167(3):103–7.
91. Pippi R, Spota A, Santoro M. Prevention of lingual nerve injury in third molar surgery: literature review. J Oral Maxillofac Surg. 2017 May;75(5):890–900.
92. Leung YY, Cheung LK. Long-term morbidities of coronectomy on lower third molar. Oral Surg Oral Med Oral Pathol Oral Radiol. 2016;121(1):5–11.
93. Agbaje JO, Heijsters G, Salem AS, Van Slycke S, Schepers S, Politis C, et al. Coronectomy of deeply impacted lower third molar: incidence of outcomes and complications after one year follow-up. J Oral Maxillofac Res. 2015;6(2):e1.
94. Monaco G, De Santis G, Pulpito G, Gatto MR, Vignudelli E, Marchetti C. What are the types and frequencies of complications associated with mandibular third molar coronectomy? A follow-up study. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2015;73(7):1246–53.
95. Frenkel B, Givol N, Shoshani Y. Coronectomy of the mandibular third molar: a retrospective study of 185 procedures and the decision to repeat the coronectomy in cases of failure. J Oral Maxillofac Surg: Official Journal of the American Association of Oral and Maxillofacial Surgeons. 2015;73(4):587594.
96. Wang Y, He D, Yang C, Wang B, Qian W. An easy way to apply orthodontic extraction for impacted lower third molar compressing to the inferior alveolar nerve. J Cranio-Maxillo-Fac Surg: Official Publication of the European Association for Cranio-Maxillo-Facial Surgery. 2012;40(3):234–7.
97. Long H, Zhou Y, Liao L, Pyakurel U, Wang Y, Lai W. Coronectomy vs. Total removal for third molar extraction: a systematic review. J Dent Res. 2012;91(7):659–65.
98. Renton T. Oral surgery: part 4. Minimising and managing nerve injuries and other complications. Br Dent J. 2013 Oct;215(8):393–9.
99. Ziccardi VB, Zuniga JR. Nerve injuries after third molar removal. Oral Maxillofac Surg Clin North Am. 2007 Feb;19(1):105–15, vii. Review.
100. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006;367(9522):1618–25. https://doi.org/10.1016/s0140-6736(06)68700-x.
101. Greenstein G, Carpentieri JR, Cavallaro J. Nerve damage related to implant dentistry: incidence, diagnosis, and management. Compend Contin Educ Dent 2015 Oct;36(9):652–659. quiz 660.
102. Juodzbalys G, Wang HL, Sabalys G, Sidlauskas A, Galindo-Moreno P. Inferior alveolar nerve injury associated with implant surgery. Clin Oral Implants Res. 2013;24(2):183–90. https://doi.org/10.1111/j.1600-0501.2011.02314.x.
103. Bede SY, Ismael WK, Al-Assaf DA, Omer SS. Inferior alveolar nerve injuries associated with mandibular fractures. J Craniofac Surg. 2012;23(6):1776–8. https://doi.org/10.1097/SCS.0b013e318266fda3.
104. Iannetti G, Fadda TM, Riccardi E, Mitro V, Filiaci F. Our experience in complications of orthognathic surgery: a retrospective study on 3236 patients. Eur Rev Med Pharmacol Sci. 2013;17(3):379–84.
105. Kushnerev E, Yates JM. Evidence-based outcomes following inferior alveolar and lingual nerve injury and repair: a systematic review. J Oral Rehabil 2015 Oct;42(10):786–802. https://doi.org/10.1111/joor.12313. Epub 2015 Jun 7.
106. Coulthard P, Kushnerev E, Yates JM, Walsh T, Patel N, Bailey E, Renton TF. Interventions for iatrogenic inferior alveolar and lingual nerve injury. Cochrane Database Syst Rev. 2014 Apr 16;4:CD005293.
107. Susarla SM, Kaban LB, Donoff RB, Dodson TB. Does early repair of lingual nerve injuries improve functional sensory recovery? J Oral Maxillofac Surg. 2007;65(6):1070–6. https://doi.org/10.1016/j.joms.2006.10.010.
108. Ziccardi VB, Steinberg MJ. Timing of trigeminal nerve microsurgery: a review of the literature. J Oral Maxillofac Surg. 2007 Jul;65(7):1341–5.
109. Zuniga JR. Sensory outcomes after reconstruction of lingual and inferior alveolar nerve discontinuities using processed nerve allograft--a case series. J Oral Maxillofac Surg 2015

Apr;73(4):734–744. https://doi.org/10.1016/j.joms.2014.10.030. Epub 2014 Nov 13.
110. Bagheri SC, Meyer RA, Khan HA, Kubmichel A, Steed MB. Retrospective review of microsurgical repair of 222 lingual nerve injuries. J Oral Maxillofac Surg. 2010;68:715–23.
111. Strauss ER, Ziccardi VB, Janal MN. Outcome assessment of inferior alveolar nerve microsurgery: a retrospective review. J Oral Maxillofac Surg. 2006;64(12):1767–70. https://doi.org/10.1016/j.joms.2005.11.111.
112. Salomon D, Miloro M, Kolokythas A. Outcomes of immediate allograft reconstruction of Long-span defects of the inferior alveolar nerve. J Oral Maxillofac Surg 2016 Dec;74(12):2507–2514. https://doi.org/10.1016/j.joms.2016.05.029. Epub 2016 Jun 14.
113. Yampolsky A, Ziccardi V, Chuang SK. Efficacy of Acellular nerve allografts in trigeminal nerve reconstruction. J Oral Maxillofac Surg. 2017 Oct;75(10):2230–4.
114. Zuniga JR, Yates DM, Phillips CL. The presence of neuropathic pain predicts postoperative neuropathic pain following trigeminal nerve repair. J Oral Maxillofac Surg 2014 Dec;72(12):2422–2427. https://doi.org/10.1016/j.joms.2014.08.003. Epub 2014 Aug 11. PMID:25308410.
115. Zuniga JR, Renton T. Managing post-traumatic trigeminal neuropathic pain: is surgery enough? J Neurol Neuromed. 2016;1(7):10–4.
116. Rosén A, Tardast A, Shi TJ. How far have we come in the field of nerve regeneration after trigeminal nerve injury? Curr Oral Health Rep. 2016;3(4):309–13.
117. Zuniga JR, Mistry C, Tikhonov I, Dessouky R, Chhabra A. Magnetic resonance Neurography of traumatic and nontraumatic peripheral TrigeminalNeuropathies. J Oral Maxillofac Surg 2018 Apr;76(4):725–736. https://doi.org/10.1016/j.joms.2017.11.007. Epub 2017 Nov 16.
118. Chhabra A, Madhuranthakam AJ, Andreisek G. Magnetic resonance neurography: current perspectives and literature review. Eur Radiol 2018 Feb;28(2):698–707. https://doi.org/10.1007/s00330-017-4976-8. Epub 2017 Jul 14.