Post-extraction inferior alveolar nerve neurosensory disturbances – A guide to their evaluation and practical management

Précis: This paper focuses on the classification, causes, prevention, management, treatment options and prognosis of inferior alveolar nerve injuries, post dental extraction. Algorithms are provided to guide the practitioner on monitoring or referring these injuries and as to the pros and cons of surgery.

Abstract: Inferior alveolar nerve injuries are a recognised complication of mandibular third molar extractions. This paper describes the different types of nerve injuries that may occur. A differential of possible causes is provided and an approach to the immediate and follow-up management is outlined. The prognosis of such injuries is reviewed so that patients can be informed of the possible postoperative outcome. The algorithm shows the timeline for monitoring/referring and the included tables outline the advantages and disadvantages of surgery versus watchful waiting.


Introduction
Neurosensory disturbances (NSDs) are a possible side effect of many dental treatments. This study is a review of the current literature on how to manage this complication. The purpose of this paper is to generate an understanding of the mechanisms, causes, management and prognosis of nerve injuries.

Mechanism and classification of nerve injuries
The possible mechanisms of neural injuries are mechanical, chemical and thermal trauma.1 Nerve injuries were historically classified by Seddon in 1943 based on three types of nerve fibre injury and whether there is continuity of the nerve.2

1. Neurapraxia – The least severe form of nerve injury, commonly resulting in temporary numbness/paralysis with subsequent complete recovery within hours to months of the injury (average six to eight weeks). The structure of the nerve remains intact but the conduction impulses are interrupted by compression/ischaemia of the nerve. The sensory deficit is usually a mild paraesthesia.4 A microsurgical intervention is not indicated.5
2. Axonotmesis – A more severe form of nerve injury. Recovery takes weeks to years and occurs only by the regeneration of axons. There is disruption of the neuronal axon with Wallerian degeneration and yet the myelin sheath remains intact. This injury is usually caused by a severe crush, contusion or stretching of the nerve. The sensory deficit is usually a severe paraesthesia.4 A microsurgical intervention is not indicated.5
3. Neurotmesis – The most severe form of nerve injury. There is internal disruption of the architecture of the nerve with perineurium and endoneurium involvement. If the nerve has been completely divided, a neuroma may form
at its proximal stump and Wallerian degeneration occurs at the distal stump. Causes of neurotmesis include a severe contusion, stretching, laceration, local anaesthetic (LA) toxicity, and transection. These injuries are likely to be permanent without repair or will only achieve partial recovery. The sensory deficit is characterised by anaesthesia or dysaesthesia.4 Thus, a microsurgical intervention may be indicated. 5 This classification was further developed by Sunderland in 1951.6 Sunderland’s classification subdivides neurotmesis into a further three divisions (Table 1). Both classifications are still commonly referred to in the literature, but in light of advances in molecular biology, they now have their limitations.

The limitation with these classifications is that they only deal with the anatomical disruption of neural tissue, which we can only presume as we do not see the nerve unless we operate. However, other factors may be involved. When a surgical procedure has been performed, neuronal plasticity occurs. This is the potential of the nervous system to adapt to inputs by changing temporarily or permanently their biochemical, physiological, and morphological characteristics. Inflammation and injury occurs at the surgical site releasing inflammatory mediators, i.e., prostanoids, which, by activating intracellular pathways, lower the threshold of nociceptors (peripheral sensitisation). This increased excitability of nociceptors can sweep back centrally to the dorsal horn nuclei of the central nervous system (CNS), changing their protein structures, facilitating an increased number of ion channels. This increases excitatory transmitters and reduces inhibitory transmitters, resulting in an abnormal perceptual response to a normal sensory input (central sensitisation). This is usually reversible when the inflammation resolves, but if nerve injury has occurred, both the injured neurons and their non-injured neighbours give action potentials spontaneously – “etopic pacemaker activity”.

Alterations in gene expression are produced and this changes the function of neurons. It may be reversible, or non-reversible. If the injured axon and its target are not re-opposed, unmylelinated axons start to die and sensory inflow is permanently disturbed.7

### Types of sensory nerve impairment

Sensory impairment may be transient or permanent. Nerve damage can be subjectively described by patients as paraesthesia, dysesthesia, anaesthesia or pain.3,5,8,9

- **Paraesthesia** – represents abnormal sensations.10 It may be divided into spontaneous paraesthesia or elicited paraesthesia.11,12 Patients present with symptoms of “pins and needles”, “tingling”, “burning”, “prickling”, “itchiness”, or “partial numbness”, which may not be particularly unpleasant.1,3

- **Dysaesthesia** – Unpleasant abnormal sensation, whether spontaneous or evoked.3,11,12 It includes:
  - hyperalgesia: exaggerated amplified response to a noxious stimulus.3
  - allodynia: pain evoked by innocuous stimuli such as light touch or gentle pressure to deep tissue, not normally painful when applied elsewhere in the body.11

- **Anaesthesia** – Total absence of sensation, including pain or an insensitivity to all forms of stimuli that would normally be painful.3,11,12 Symptoms that are akin to post-dental injection anaesthesia.

- **Pain** – An unpleasant sensory and emotional experience, associated with actual or potential tissue damage and/or described in terms of such damage.12
  - Neuroma pain: pain aggravated by mechanical stimulation.13

### Differential of the possible causes of nerve injury

#### The extraction

It is estimated that 42.9-69% of all iatrogenic injuries to the inferior alveolar nerve (IAN) are caused by third molar surgery, especially those molars impacted in close proximity to the IAN.14,15 Renton stated that nerve injury can be temporary in 8% of these cases and permanent in 3.6% of these cases.9,14 Rates of paraesthesia are quoted as 0.4-8.4%.17,18,19,20,21,22 This may be caused by direct trauma to the nerve (root elevators may cause blunt trauma to the nerve via

<table>
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<tr>
<th>SEDDON 1943</th>
<th>SUNDERLAND 1951</th>
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<tr>
<td>Neurapraxia</td>
<td>First-degree injury</td>
</tr>
<tr>
<td>Axonotmesis</td>
<td>Second-degree injury</td>
</tr>
<tr>
<td>Neurotmesis</td>
<td>Third-degree injury - Endoneurium disruption but epineurium and perineurium intact</td>
</tr>
<tr>
<td></td>
<td>Fourth-degree injury - Perineurium injury with only epineurium intact</td>
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<tr>
<td></td>
<td>Fifth-degree injury - Complete transection</td>
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</table>
Neurotoxicity of the local anaesthetic
If the LA is injected intrafascicularly, or is deposited in the nerve during needle withdrawal, this can induce a localised chemical injury to the nerve. Smith and Lung explain how this results in demyelination, axonal degeneration and inflammation of the surrounding nerve fibre within the fascicles. Thus, the nerve blood barrier breaks down and endoneurial oedema occurs, followed by ischaemia, and subsequently the formation of reactive free radicals, which can cause cytotoxic injury to the nerve. Haas and Lennon implicated prilocaine and articaine as having a higher incidence of neurotoxicity than lidocaine. It is speculated that because these anaesthetics are present at higher concentrations (i.e., 4% articaine), they will produce a larger amount of toxic metabolites after being metabolised. Articaine is thought to be impossible for a needle to shear all nerve fibres, thus neurotmesis should not occur and therefore a transient paresis is a high possibility or the formation of a haematoma caused by trauma to the intraneural blood vessels by the needle. This results in epineuritis, which compresses nerve fibres, inducing a reactive fibrosis and subsequent scar formation. Thus, a neuropaxia or axonotmesis may occur depending on the amount of pressure applied to the nerve. For this type of injury, recovery may take weeks as remyelination and remyelisation must occur. Pain or an electric shock sensation is not a definite indicator of having made contact with the nerve.

Compress or the nerve may be disrupted by rotating burs. It may also be caused by indirect trauma via compression of the nerve, due to oedema or formation of a haematoma post extraction. A minor compression usually only causes a temporary conduction block (i.e., Seddon’s neuropraxia/Sunderland’s first-degree injury). Extraction of a tooth which required elevation of a mucoperiosteal flap adjacent to the mental nerve may cause a more severe type of stretching injury, causing rupture of the endoneurium and perineurium (Sunderland’s third- and fourth-degree injuries). Neurotmesis can occur if the IAN were passed through the root of the tooth and during elevation of the tooth, the nerve may have been subsequently transected. If the patient had severe resorption of the mandible, the position of the inferior alveolar nerve/mental nerve may be at a high level in the bone and easily crushed.

The inferior alveolar nerve block
Pogrel and Thamby in 2000 estimated that every full-time practitioner will have one patient in their lifetime of work who will suffer from permanent nerve involvement as a result of IAN block. IAN injury has a lesser incidence than lingual nerve injury because it can be deflected by the needle, unlike the more vulnerable lingual nerve. The causative factor may be a) the needle makes direct contact with the nerve, traumatising it; or, b) the needle tip became barbed on contacting the bone especially in the case of multiple injections. Smith and Lung in 2006 described how these barbs can rupture the perineurium, herniate the endoneurium and cause transection of multiple nerve fibres and even entire fascicles, especially on withdrawal. (Seddon’s axonotmesis/Sunderland’s second- and third-degree nerve injuries). However, due to the small diameter of the needle – 0.45mm – compared to the larger diameter of the inferior alveolar nerve – 2-3mm – it is thought to be impossible for a needle to shear all nerve fibres, thus neurotmesis should not occur and therefore a transient paresis is a high possibility or c) the formation of a haematoma caused by trauma to the intraneural blood vessels by the needle. This results in epineuritis, which compresses nerve fibres, inducing a reactive fibrosis and subsequent scar formation. Thus, a neuropaxia or axonotmesis may occur depending on the amount of pressure applied to the nerve. For this type of injury, recovery may take weeks as remyelisation and remyelisation must occur. Pain or an electric shock sensation is not a definite indicator of having made contact with the nerve.

### Table 2: Factors which initiate or perpetuate disorders of neuronal tissue (systemic diseases which also cause neuropathies)

<table>
<thead>
<tr>
<th>Bacterial infections</th>
<th>Viral infections (herpes zoster)</th>
<th>Arthridities</th>
<th>Neoplasia</th>
<th>Metastases</th>
<th>Haematological diseases (leukaemia and lymphoma)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple myeloma</td>
<td>Cysts</td>
<td>Metabolic disorders (diabetic neuropathy)</td>
<td>Vitamin deficiencies</td>
<td>Alcohol abuse</td>
<td>Drug-induced diseases (chemonecrosis)</td>
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<tr>
<td>Bone diseases (osteomyelitis)</td>
<td>Multiple sclerosis</td>
<td>Sarcoïdosis</td>
<td>Epilepsy (seizure may have caused a fracture)</td>
<td>Collagen vascular diseases</td>
<td>Psychological disorders</td>
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</table>

**Mandibular fracture**
Mandibular fractures can occur during or post extraction. The incidence of this is estimated as 37 in 750,000 extractions. A fracture can cause a partial or total transaction, a laceration or a stretch on the nerve, especially if displaced. This stretching results in Sunderland third-degree and fourth-degree injury. Libersa et al. in 2002 indicate that fractures can occur in all grades of tooth impaction, especially in male patients over 25 years of age. Woldenberg et al. in 2007 implicate the relative volume of tooth in the jaw, pre-existing infection, not maintaining a soft diet in the postoperative period and surgical technique as factors predisposing to mandibular fractures.

**Post-extraction infections**
Delayed onset wound infections are defined as infectious swellings
with onset generally one week after extraction. Figueiredo et al. in 2005 estimate their incidence after third molar extraction as 1.5%. Infection was evident between 10 and 84 days post extraction, usually before one month. Haematomas or food debris trapped beneath the flap can also be a nidus for infection.

Infection can cause:
1) mechanical/local pressure due to accumulation of purulent exudate;
2) ischaemia associated with inflammatory process; or,
3) toxic metabolic products of bacteria, which can breach the perineurium. Gram-negative bacteria are often involved in the production of neuropathies. In cases of non-persistent episodes of nerve irritation, paraesthesia should resolve within days or weeks as the cause is removed.

Other possibilities
Concurrent dental treatments at the time of the extraction could also be considered a potential cause, e.g., root canal treatment, implant placement and orthodontic treatment have all been implicated as causing NSDs.

Management
The management of a patient who presents post extraction with sensory dysfunction involves: history taking; examination; radiographs; sensory testing; provision of information regarding possible treatments; prognosis; and, a decision to monitor or refer.

History taking
WHO guidelines suggest that nerve injuries should be assessed in terms of impairment, activity limitation and participation restriction.

Ask the patient the following questions:
1) When did the sensory impairment begin? (Delayed onset may indicate infection, or post-op fracture.)
2) Can you describe the symptoms, i.e., complete numbness, burning, pain, tingling, pins and needles, etc.?
3) Is there a family history of chronic postoperative pain?
4) Is the sensation precipitated by touching the affected area?

Further a change in character of the sensation (i.e., a complete numbness may change to a paraesthesia indicating possible return of sensation)?
Could you point to the area of altered sensation?

Examine the patient extraorally and intraorally for: lymphadenopathy; swelling; heat; redness; tenderness on palpation; step deformities; rash (herpes zoster); discharge; haematoma formation; sequestra; and, trismus.

Medical history
Ask the patient if they have a personal or family history of the aforementioned systemic diseases and review their current and previous medication. Enquire regarding the family’s pain threshold.

Radiograph
Consult pre-operative radiographs for proximity of the tooth to the IAN. Take a postoperative radiograph and look for a dislocated root fragment in the mandibular canal or a dislodged bone fragment from the roof of the canal compressing the nerve. If there is deviation or disruption of the canal, then decompression is indicated.

Quantitative sensory testing
Nerve injuries may affect:
1) mechanoreception (touch pressure, positional sense);
2) thermoreception (hot, cold); and,
3) nociception (pain).

These sensations must be assessed to monitor whether a sensory disturbance is persistent or improving. Robinson et al. in 1992 described a method of sensory testing and, with this in mind, a similar way of testing may be employed using everyday equipment in the general dental practice. Hillerup in 2008 has shown that there is an excellent correlation between NSD expressed in the patient’s words and the objective findings by clinicians.

An objective assessment is achieved by testing the injured side and comparing it to the non-injured side, which is used as a control. The room should be quiet; the patient should close their eyes and raise their finger to indicate they feel a stimulus. An objective assessment involves:

1) Light touch perception (Figure 1)
Using a cotton pellet to lightly touch the affected area, one can map and measure an area within which no stimulus can be felt.

2) Pain (pin-prick) perception (Figure 2)
Repeat as above using a dental probe and again map the area of anaesthesia or pinch the lower lip within a tissue forceps to ascertain if the stimulus is felt.

3) Two-point discrimination threshold (Figure 3)
Place a tweezers on the skin and enquire if one point or two points can be felt. Lips can distinguish two points 2-4mm apart, whereas skin over the lower border of the mandible can distinguish only 8-10mm. The two-point discrimination is usually higher on the injured side.

4) Thermal assessment (Figure 4)
Test the affected area using a cotton pellet firstly dipped in cold water then in hot water. (Recommended: Heat 45-50 degrees Celsius. Cold 0-20 degrees
Celsius. This exact measurement is difficult to achieve in practice.)

5) Directional sense (Figure 5)
Brush the area with a brush or probe asking the patient in which direction the instrument is moving.

6) Pointed dull discrimination (Figure 6)
Alternate between the blunt end and the sharp end of a probe and ask the patient is the sensation sharp or dull.

7) Location of touch
Ask the patient to point to the area that has just been touched by a probe.

8) Photograph (Figure 7)
By taking a photo of the mapped area, this may be used for comparison at the next visit. It is difficult to achieve reproducibility with photos so reproducible markings should be placed. In Figure 7, a line is drawn from the midpoint of the tragus to the commissure of the mouth and another line drawn from the mid point of the vermilion border of the lower lip, to the mid point of the lower border of the chin (black line). The distance from the affected area (red shaded area) to the reproducible markings may be measured with a ruler and recorded (green lines).

Robinson et al. stated that light touch stimuli, pain (pin-prick) stimuli and two-point discrimination thresholds are adequate to detect evidence of early sensory recovery and that the latter two tests are the most likely to reveal a persistent sensory neuropathy. A patient’s objective assessment may disimprove at a future visit and this may be a sign that a neuroma has formed, which is interfering with nerve conduction.

Treatment options

1) “Wait and see”
- Reassure the patient as it has been reported that 96% of injuries recover spontaneously.
- However, if the injury does not improve within three months it is likely to be permanent.
- Monitor by regular objective and subjective sensory testing at one-week, one-month, two-month and three-month intervals.
- Advise the patient to take care with shaving, oral hygiene, and hot food or drinks, and warn them against cheek biting.

2) Treat painful neuropathies
Anticonvulsant drugs (gabapentin 300mg on the first day, 600mg on the second day, 900mg on the third day to be titrated to 1,800mg or to a maximum dose of 3,600mg), tricyclic antidepressants (amitriptyline, starting at 10mg and titrating to a maximum dose of 75mg), 5% lidocaine patches, topical clonazepam, benzocaine lozenges, Botox injections,
benzodiazepines, carbamazepine, steroids and vitamin B supplements have been used.\textsuperscript{3,4,13,39,54,55,56} (NB: Adverse side effects of drugs - dizziness, visual disturbances, depression, rash.)\textsuperscript{15}

3) Treat suspected infection
Antibiotics, surgical drainage, Corsodyl mouthwashes and irrigation of the socket may reduce infection and subsequently decompres the nerve.\textsuperscript{57}

4) Refer to psychologist for cognitive behavioural therapy.\textsuperscript{54}

5) Refer to a speech therapist if speech function is impaired.\textsuperscript{54}

6) Refer for further assessment, surgical exploration, +/- microsurgical repair.
If there is minimal/no resolution of a large neuropathic area, poor mechano-sensory function, or poor daily function with moderate to severe pain, then exploration may be warranted.\textsuperscript{54}

This may involve one of the following procedures.

Decompression - This is indicated when a retained root fragment, a fragmented part of the roof of the mandibular canal or a foreign body is compressing the nerve, thus relieving pressure on the nerve by the surgical excision of constricting bands or widening of a bony canal.\textsuperscript{56}

Primary direct re-anastomosis - This is indicated where there has been a complete transection of the nerve with both ends lying in close proximity to one another. The ends are then slightly stretched, reapproxosed and then sutured together using epineurial sutures.\textsuperscript{56}

External neurolysis - This procedure frees the nerve from inflammatory adhesions.\textsuperscript{58}

Internal neurolysis - This procedure removes the inflammatory adhesions between the nerve fascicles.\textsuperscript{13}

Neurectomy - This is the complete removal of the injured nerve.

Nerve grafting - This is indicated where both cut ends of the nerve are far apart and a graft is needed to bridge the continuity defect, e.g., the sural nerve, the great auricular nerve and medial antebrachial nerve may be used.\textsuperscript{59,60}

Vein graft - This reconstructs the nerve gap between the cut ends of a nerve. The vein is placed over each cut end and is inserted via an extraoral approach following decortification of the mandible, e.g., facial vein.\textsuperscript{60}

Nerve conduits - These are used to reconstruct a nerve gap. Examples are alloplastic conduits such as gortex.\textsuperscript{60}

Muscle graft - Muscles such as the masseter, the tongue or the anterior digastric may be used. Muscles may be freeze-dried and the neuronal elements of the injured nerve can grow over the laminin sheath of the muscle (denatured muscle autografts).\textsuperscript{59,60}

Excision of neuromas - Neuromas (benign tumours of nervous tissue) need to be excised before anastomosis can be achieved.

7) Refer to a pain management specialist
In cases of intractable dysesthesia, urgent referral may be necessary. This condition can severely affect a patient’s quality of life, producing significant psychological effects, and has even led patients to take their own life.\textsuperscript{9,15,61} The plan may include a multi-disciplinary approach with therapeutic medication, counselling and CBT.\textsuperscript{54}

Prognosis
The prognosis for recovery from an IAN nerve injury has been estimated as 96% of the injuries recovering within four to eight weeks.\textsuperscript{20} Gregg in 1995 indicated that higher levels of recovery can be expected in young patients, in good health, where local tissues are well perfused with no foreign bodies obstructing healing and where epineurial sheaths are intact or severed ends are passively opposed.\textsuperscript{13}

Persistent injury is more likely to occur if it was a severe injury, an older patient, a delayed presentation or when the injury is more proximal to the cell body.\textsuperscript{54} Loescher et al. in 2003 described how following an injury, the nerve often remains in position and regeneration begins.\textsuperscript{62} The advantage that an IAN injury has over a lingual nerve injury is that it has a bony canal, which acts as a conduit for the regeneration of nerve fibres;\textsuperscript{14,15} conversely, the bony canal can predispose it to ischaemic trauma and resultant permanent nerve damage may occur.\textsuperscript{3}

However, if the nerve is displaced into the socket, or if a fragment of bone from the roof of the canal is causing an obstruction, regeneration may only be aided by surgery.\textsuperscript{36} The visual sighting of the IAN bundle implies intimate relationship of the tooth to the nerve and carries a 20% risk of paraesthesia.\textsuperscript{2} Observed injuries should be repaired within 90 days.\textsuperscript{61} Knowledge of the mechanisms of nerve injuries should influence our decision to refer or monitor (Table 3). Neuapraxias, usually as a result of compression, are represented as a paraesthesia and result in complete recovery.\textsuperscript{4} Compression injuries often resolve within four months.\textsuperscript{55} Paraesthesia tends to subside in six months.\textsuperscript{20} Axonotmesis is usually represented as a severe paraesthesia and incomplete recovery has been described.\textsuperscript{4} Neurotmesis is characterised by anaesthesias or dysesthesias and may not recover, thus may require surgery.\textsuperscript{4} Anaesthesia beyond one month is likely to have permanent impairment.\textsuperscript{25}

Hillerup in 2008 noted how recovery of lesions differed depending on their aetiology, with lesions due to third molar removal recovering more significantly.\textsuperscript{15} He expressed how these lesions had an impressive potential for recovery to a level where microsurgical repair may not be necessary.\textsuperscript{15} He also found that there was no convincing recovery from lesions caused by endodontic procedures or LA.\textsuperscript{15} In contrast to this, Pogrel stated that studies have shown that LA-induced nerve injuries usually result in a spontaneous recovery in an eight-week period and Smith and Lung found that they have an excellent prognosis.\textsuperscript{1,2,5} Exploratory surgery has been unhelpful in patients with permanent nerve involvement due to LA and may even exacerbate symptoms.\textsuperscript{25} Surgery for LA-induced NSD is hazardous, as a need to mobilise the medial pterygoid to gain access may be necessary.\textsuperscript{25} Surgical outcomes may improve the sensory function, but often a complete recovery of nerve function is not achieved and patients must be informed about this.\textsuperscript{14} Pogrel and Lam estimate a 50% and 55% improvement after surgery, respectively.\textsuperscript{64,65}

Neurogenic, central pain, and anaesthesia dolorosa is not affected by peripheral surgery and such surgery may worsen the situation.\textsuperscript{36} Many
patients can adapt to their neurological deficits and hypoaesthesias are well tolerated.15,55 This must be considered in light of the morbidity of surgery. Patients with mild hypoaesthesia or paraesthesia are unlikely to benefit from surgical intervention.36 The most rapid phase of recovery occurs within the first six months.14 Most authors agree that a deficit present beyond 12 months is usually permanent.14,15,57 There is still disagreement as to the timing of referral for exploration/microsurgery. Some authors recommend early repair,4,66 another encourages late repair,13 Robinson et al. in 2004 found no significant correlation between delay and surgery outcomes.14 Most surgeons want patients referred within three months of injury.14 Based on cellular and biomechanical events, the ideal time for surgical repair is two to three weeks post injury, to maximise resolution of sensory function, and minimise neuronal cell death and central changes.54 Straus et al. in 2006 state that if microsurgery is performed within one year of injury, recanalisation or neotisation of distal end organs can be expected to occur.34 After one year, there is significant distal nerve scarring and atrophy, making surgery more difficult and less predictable.14,63 Renton et al. in 2010 state that the injury is permanent after three months, as it is after three months that permanent central and peripheral changes occur within the nervous system that are unlikely to respond to surgery.9,54 However, in reality, defining the exact type of nerve injury that has occurred and predicting recovery is not so straightforward. Patients must be evaluated with other factors such as: systemic co-morbidities; age; gender; pain threshold; psychological reaction to pain; and, their expectations accounted for. The multifactorial aspects of nerve injuries make prediction of a definite prognosis more difficult.

Prevention of nerve injuries

Firstly, consult NICE Guidelines to ensure that the tooth needs to be extracted and plan to avoid using high concentration LA and multiple blocks.23 Precautions to prevent nerve injury include:

1) Radiographs (Figure 8: Taken from Kosit Bowornchai et al.67)

An OPG and/or periapical radiograph should be taken to assess the likelihood of damage to the IAN. Signs including: a) darkening of the root where it crosses the inferior alveolar canal; b) deflected or hooked roots around the inferior alveolar canal; c) narrowing of the root, implying perforation or grooving by the nerve; d) a bifid root apex, representing intimacy of the apical periodontal membrane; e) interruption or obliteration of either of the cortical lines of the inferior alveolar canal; f) diversion of the inferior alveolar canal in the region of the root apices; g) narrowing of the inferior alveolar canal; and, h) presence of a juxta-apical area warrant caution/further investigation.9,15,16,22,68,69 Renton indicated that (f) and (h) were found to be most predictive of nerve injury.9

2) CT scans (Figure 9: Taken from Kosit Bowornchai et al.67)

These are expensive but are being used more often nowadays. As an x-ray only gives a two-dimensional view, errors may arise.20,22 If a panoramic +/- a periapical x-ray reveals a suspected close proximity between the tooth to be extracted and the mandibular canal, then dental CT scans are advised to determine the precise relationship between these structures so as to be able to accurately assess the extractive risk.23,68

Table 3: Risk assessment of a wait-and-watch approach versus surgical repair following the development of an IAN complication.

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<tr>
<th>SURGICAL REPAIR</th>
<th>MONITORING</th>
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<tbody>
<tr>
<td><strong>PROS</strong></td>
<td><strong>CONS</strong></td>
</tr>
<tr>
<td>Repair of nerve damage;</td>
<td>Risk associated with general anaesthetic (GA) or sedation;</td>
</tr>
<tr>
<td>Decompression of compressed nerve;</td>
<td>Improvements shown in only 50-55%;</td>
</tr>
<tr>
<td>Conduit/graft repair of severed nerve;</td>
<td>Extra-oral approach may cause scarring or damage to the mandibular division of the facial nerve;</td>
</tr>
<tr>
<td>Removal of adhesions or neuromas;</td>
<td>Morbidity of the harvest site;</td>
</tr>
<tr>
<td>Method of choice for complete anaesthesia beyond three months, profound hypoaesthesia with no improvement beyond three months, dysesthesia beyond three months and clinically observed nerve sevarence; and,</td>
<td>Risk of further damage to the nerve;</td>
</tr>
<tr>
<td>May cure intractable/neuropathic pain that is unresponsive to medication.</td>
<td>May not be appropriate treatment in a medically compromised patient; and,</td>
</tr>
<tr>
<td></td>
<td>May be ineffective if there is an excessive delay following injury.</td>
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</table>

**CONS**

3) Alternative procedures

Procedures such as a coronectomy (removal of the crown only) or an orthodontic extraction (extruding the tooth before extraction) have been described in the literature to avoid IAN damage for a ‘high-risk’ extraction.23,69

4) Referral to a specialist

Operator expertise has repeatedly been implicated as a factor in predicting nerve damage.11,18,20,21,57,70 Robert et al. in 2005 showed that IAN injury rates decrease with years of experience.70 Trainee
surgeons showed a higher prevalence of paraesthesia in their patients than their more experienced colleagues, and permanent nerve damage was four times more likely in the trainee group of patients also.\textsuperscript{20} It is important that practitioners recognise the need to refer a patient if the extraction does not appear to be within his/her field of expertise.

Consent and legal implications

A patient must be given an informed consent to sign before the extraction. Any extraction that appears to pose a risk to the IAN should be considered for consent, including third molars and surgical extractions near the mental foramen. Risk of a 2\% permanent injury and 20\% temporary injury should be declared.\textsuperscript{9} Consent should mention hyperaesthesia and pain and not only numbness.\textsuperscript{5} LA injection injury is considered a ‘no culpa’ incident, i.e., insurance coverage is not conditioned by proven malpractice.\textsuperscript{11} Financial compensations are usually paid after a two-year period has elapsed since the time of injury. A spontaneous recovery within this period will not be compensated for.\textsuperscript{5} Unfortunately, this delay also results in a patient not seeking exploration/microsurgical repair till the money has been paid and by this time recovery, even after surgery, is unlikely.

Conclusion

Iatrogenic NSDs are a rare complication of dental procedures. IAN injuries are less debilitating than lingual nerve injuries and also have a higher incidence of spontaneous recovery.\textsuperscript{5} Although there is disagreement within the literature about protocols for managing these patients, some common findings are acknowledged. Most authors agree that an urgent referral for surgery is recommended if an observable injury occurred (nerve appears sectioned in the extraction socket, a bleed requiring packing to control it, retained root or a foreign body in the canal).\textsuperscript{4,6,63} Another shared belief is that most injuries resolve within three to six months and a lack of improvement at three-month follow up warrants specialist referral.\textsuperscript{14} There is also agreement among authors that nerve impairment beyond one year is usually permanent and surgery after one year may be unsuccessful or have a worse prognosis.\textsuperscript{14,57} Surgery may potentiate neuropathic symptoms and patients need to be warned about this.\textsuperscript{54} The literature also outlines how many patients become tolerant to their sensory impairment and that surgery may not be indicated in this situation. Most of the literature refers to nerve injuries in the form of neurapraxia, neurotmesis and axonotmesis and predicts the prognosis based on this; however, we must be aware that this anatomical classification is becoming outdated and may be oversimplifying the situation. It is important to realise that nerve injuries have more complex molecular pathways and patients may have multifactorial aetiologies; therefore, this paper serves as a guideline only. These patients can be very difficult to treat. Equipped with this knowledge, general dental practitioners should be able to assess, monitor, refer and advise their patient as to the best individualised treatment for them.

References

4. Donoff, R.B. Surgical management of inferior alveolar nerve injuries (Part 1): The case
Table 4: Neurological post extraction review.

<table>
<thead>
<tr>
<th>Time</th>
<th>Asymptomatic</th>
<th>Anaesthesia</th>
<th>Parästhesia</th>
<th>Hypoesthesia</th>
<th>Dyästhesia</th>
<th>Pain Mx</th>
</tr>
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<tbody>
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<td>One to two weeks</td>
<td>No treatment required</td>
<td>Monitor/OPG and refer</td>
<td>Monitor/OPG and review 3/12</td>
<td>Monitor/OPG and review 3/12</td>
<td>Monitor/OPG and review 3/12</td>
<td>Pain Mx</td>
</tr>
<tr>
<td>Three months</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 3/12</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 3/12</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 3/12</td>
</tr>
<tr>
<td>Six months</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 3/12</td>
<td>No improvement or worsens or develops Dyästhesia</td>
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<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 3/12</td>
</tr>
<tr>
<td>12 months</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 6/12</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 6/12</td>
<td>No improvement or worsens or develops Dyästhesia</td>
<td>Improving Monitor 6/12</td>
</tr>
</tbody>
</table>

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