Complications of Local Anaesthesia in Endodontics

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3.1 Introduction

Problems resulting from local anaesthesia during endodontic treatment can result from many causes. They can be restricted to a local area or result in systemic signs and symptoms. Problems can arise from a direct (primary) cause involving the injection process or as a reaction to this (secondary cause). Symptoms can be transient or permanent and can be mild to severe in intensity. There are many textbooks that deal with technical aspects of local anaesthesia used in dentistry and complications resulting from its use. There is also excellent visual material available on various internet sites giving detailed explanations of dental anaesthetic techniques. This chapter will deal with complications arising during/after the administration of local anaesthesia with particular reference to its use in endodontics.

Pain management of the patient during endodontic treatment is critical. The clinician and their team must use different resources to holistically manage the patients (and their family or escort) to ensure that they feel well cared for, are in control of the situation, understand fully what is happening to them and realise the risks and benefits of the intended treatment. Research has shown that fear of pain associated with the delivery of local anaesthesia is a common cause of anxiety, and this alone can prevent many from obtaining dental treatment. Dental phobia is the most common phobia reported, and over 80% of patients are anxious about dental treatment, which of course lowers their pain threshold. Most patients with endodontic

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emergencies no longer have a choice regarding attendance or the need for anaesthesia. They require profound anaesthesia. Needle fear and also the patient's fear of numbness are issues to consider. Many patients arrive at the operatory anxious, tired and in pain, requiring immediate diagnosis and treatment. Anxiety can be compounded by fear of dental injections. It also influences perception of pain. Thus it is important in endodontic treatment to reduce the anxiety and pain associated with delivery of effective anaesthesia. This delivery should be an almost pain-free process if care is taken with the use of an empathetic gentle patient management and slow delivery of the local anaesthetic. By avoiding block anaesthesia where possible, by adopting an infiltration technique and routinely using aspiration effectively during block anaesthesia, the majority of complications can be avoided.

3.2 Failure to Deliver Pain-Free Local Anaesthesia

Explanation for the need of the injection and reassurance of the patient are important in patient care. Distraction using visual aids, music or any patient preferred media is advised. Pain and burning during anaesthesia can also be minimised by careful anaesthetic technique. Tissues at the injection site should be dried, and a small amount of topical anaesthetic should be applied exactly to the site of the injection. Sixty seconds is a long time when treating an emergency in a busy dental practice. Nevertheless, to be effective, topical anaesthetic should be left in place for at least this long, preferably longer. It is not effective if used for a shorter period of time. To obviate any pH changes in old local anaesthetic solution, ensure solutions are in date before use. The anaesthetic should be kept at and administered at room temperature. Expression of a few drops of anaesthetic from the cartridge before the injection will ensure that the bung is moving freely. If possible tissues at the injection site should be stretched taut at the time of needle insertion. Slow advancement of the needle tip through tissues and slow deposition of solution once the target area is reached are helpful to prevent pain during injection procedures. Positive communication with the patient and keeping the syringe out of sight are important habits along with the technical process itself. Distraction techniques, e.g. involving tugging the tissues over the needle tip or asking a patient 'to see if they can try opening even wider than they are', 'wiggling their toes' are essential parts of the local anaesthetic process. Prewarning the patient of a possible electric-like feeling when administering an inferior alveolar nerve block (IANB) injection can be helpful in patient management.

Disposable anaesthetic needles are invariably sharp on first use. Nevertheless if direct contact is made though with bone, the tip can become damaged. Any tiny barb produced at the tip on the needle can cause needle insertion to become painful if a second delivery is made, or result in bleeding and possibly contribute to nerve injury when the barbed needle is withdrawn from tissues. If contact is made with bone, the needle should be inspected before a second injection is made and replaced if necessary. Block injections should be preferred where local tissues are inflamed or

swollen. Palatal infiltrations should be given in areas where the tissues are not tightly attached to the palate.

3.3 Failure to Obtain Adequate Pulpal Anaesthesia in Mandibular Teeth

Failure to obtain adequate pulpal anaesthesia is the most frequent untoward outcome of local anaesthesia in endodontics and is often associated with delivery of an IANB injections. Though it goes without saying that lack of lip numbness is a clear sign of lack of pulpal anaesthesia, lack of pulpal anaesthesia can occur despite the presence of lip numbness, especially in mandibular posterior teeth with symptomatic irreversible pulpitis. Lack of pulpal anaesthesia is often objectively tested as an inability to obtain two consecutive reading of 80 with electric pulp tester (EPT) or after prolonged application of a cold stimulus. However, every endodontist is aware that pulps can be sensitive even when this test appears to confirm complete anaesthesia. Many studies have highlighted the inadequacy of IANB providing sufficient pulpal anaesthesia to allow for restorative treatments with less than 50% of healthy anterior teeth that are likely to be anaesthetised by an IANB. The pulpal anaesthesia rate will further decline with pulpitis and other local factors.

Lack of lip numbness is a sign of a misdirected block and is reported to occur from 5% up to 23% of the time [1, 2]. While lip numbness usually occurs in 4–6 minutes after an IANB, pulpal anaesthesia in a mandibular first molar regularly takes longer (up to 9 min). Lower central incisors may take longer still (up to 19 min) [3]. In addition, 12–20% of patients can have slow onset of pulpal anaesthesia, defined as an 80 EPT reading occurring after 15 min [3]. Thus sufficient time should be allowed for pulpal anaesthesia to develop before endodontic therapy is initiated. The slower onset in anterior teeth should be considered when treating these teeth, though the latter could be mitigated with a buccal infiltration.

Incorrect techniques, failure to identify landmarks, insufficient dosage and anatomical variations such as bifid IAN canals are often cited as the many reasons for lack of lip numbness. An inability to identify the three critical landmarks, namely, the coronoid notch, the pterygomandibular raphe and the occlusal plane of mandibular teeth, are the most common causes for failure of IANB injections. Many anxious patients half open their mouth and posture the jaw forwards. Anecdotally one of the major reasons for missing an IANB injection is identifying injection landmarks while the mouth is not completely open. Injections given in this jaw position often result in the solution being deposited ineffectively distal and below the IAN. Perhaps the single most important adjunct to the delivery of an IANB is to ensure that the patients open their mouth, ‘as wide as they can’, before the injection is placed. Asking the patient if they can open ‘even wider’ just before injection ensures that landmarks can be seen and also provides useful distraction at the time of delivery.

The loss of multiple mandibular posterior teeth can result in confusion regarding the orientation of the cranio-caudal plane of injection site. An unusually larger

buccal pad of fat and a larger than usual tongue can also obscure vision, preventing accurate location of the site of insertion. It is worthwhile, especially in these cases, to let the assistant retract the tongue with a mouth mirror to the non-operating side and to identify the cranial end of the pterygomandibular raphe rather than the caudal end. The latter is more difficult to identify when mandibular molars are absent. Simultaneously, the face of the mouth mirror can be used to compress the buccal pad of fat against the medial surface of the ramus of mandible, and this will surprisingly often reveal an otherwise obscured pterygomandibular raphe. While repeating of IANB will often fix problems with anaesthesia, it increases the risk of nerve injury as the usual protective reflexes will no longer be operating effectively.

Popular theories and their drawbacks on causes of failure of an IANB in symptomatic irreversible pulpitis in mandibular teeth are discussed.

Obtaining profound pulpal anaesthesia in mandibular molars with symptomatic irreversible pulpitis is one of the most common problems facing clinicians. Success rates have been reported to be as low as 14–33%. [2]. Before embarking on strategies to manage failed IANB injections in symptomatic irreversible pulpitis, it is important to dispel some of the popular misconceptions:

1. *Misdirected injection*: While a misdirected block injection can lead to lack of anaesthesia, lack of pulpal anaesthesia following an IANB in the presence of lip numbness is not necessarily due to an inaccurate injection site [1]. IANBs performed using an ultrasound or peripheral nerve stimulator to accurately locate the neurovascular bundle have not always increased the success rate of mandibular first molar pulp anaesthesia when compared to conventional techniques [4, 5].
2. *Accessory innervation*: The two sources of accessory innervation to the mandible that are considered to be associated with failed pulpal anaesthesia are the mylohyoid nerve and the cutaneous branches of the cervical plexus. Contrary to popular belief, accessory innervation from mylohyoid nerve is unlikely to be the cause of failure of an IANB in symptomatic irreversible pulpitis or even in healthy subjects. Blocking of the mylohyoid nerve has not been associated with increased success of an IANB in healthy subjects [6, 7] or in patients with irreversible pulpitis [8]. A recent systematic review concluded that additional lingual infiltrations of mandibular teeth did not improve the anaesthetic success of pulpal anaesthesia in mandibular molars, premolars and canines irrespective of whether 2% lignocaine or 4% articaine was used [9]. However, it did increase success rates for mandibular incisors [9]. Accessory innervation from the cervical plexus has been shown to play a role in pulpal anaesthesia, but in a recent study, only 60% of subjects with symptomatic irreversible pulpitis were shown to have gained pulpal anaesthesia with an IANB coupled with intraoral cervical plexus anaesthesia [10]. Anaesthesia of the cervical plexus is made by depositing the solution by infiltration distal and below the apex of the mandibular first molar.
3. *Ion trapping hypothesis*: Diffusibility and binding are two critical factors in the clinical effectiveness of local anaesthesia [11].The lipid soluble, uncharged

base form (RN) of the local anaesthetics is responsible for the diffusion of the solution into a nerve sheath. The amount of base form in the solution is determined by the pKa (dissociation constant) of the solution and the pH of extracellular environment. The ion trapping hypothesis states that the lower pH associated with infection reduces the amount of active RN of the local anaesthetic. This does not in itself explain why IANB can fail in infected teeth, as the site of infection is away from the injection site and often only limited to the tooth apex [1].

The use of buffered lignocaine, which should theoretically result in significantly higher amount of available RN, has not been shown to increase the success rate of IANBs in symptomatic irreversible pulpitis using either 2 or 4% lignocaine formulations [12, 13]. The lack of sufficient RN of local anaesthesia due to low pH within or around the tooth is unlikely to be the reason for a failed IANB. This may nevertheless be of significant in infiltrations where the site of injection and location of abscess may be in close proximity.

4. *Central core theory*: This is based on the anatomy of IAN, where central fibres have been shown to supply the molars and peripheral fibres, supplying the anterior teeth. Additionally, the lip is supplied by the external and myelinated A β fibres of the IAN, which are relatively easily anaesthetised as compared to the core fibres and the unmyelinated C fibres, and the unregulated nociceptive elements in symptomatic irreversible pulpitis. While the inability of a solution to diffuse through the entire nerve bundle, particularly if the block is misdirected or if there is insufficient solution deposited at the site, may seem a plausible explanation why IANB anaesthesia may fail, it does not explain failure to anaesthetise inflamed pulps in molar teeth [14].
5. *Difference in susceptibility of different category of nerve fibres to anaesthesia*: A negative response to an EPT does not correspond with pain-free treatment. In one study over 40% of pulps in patients with irreversible pulpitis were not completely anaesthetised, even though teeth did not react to an EPT [15]. This may be because local anaesthetic solutions are four times more effective in blocking the myelinated Aδ fibres, which are stimulated by EPT, than the unmyelinated C fibres, which are often involved in the dull, boring, radiating pain associated with symptomatic irreversible pulpitis [16, 17]. This difference in susceptibility to local anaesthetic of a nerve fibre group might be a contributing factor in lack of pulpal anaesthesia in symptomatic irreversible pulpitis.

Most likely explanation for failure of IANB in symptomatic irreversible pulpitis:

1. *Flow of anaesthetic to path of least resistance*: Considering the failure to obtain profound pulpal anaesthesia in up to 23% of healthy mandibular molars anaesthesia, it is important to recognise that a single factor is unlikely to cause anaesthetic failure in irreversible pulpitis [3]. Hence some of the factors which may play a role in failure to achieve anaesthesia in healthy molars, such as the flow of anaesthetic along the path of least resistance away

from the pterygomandibular space, may contribute to failure in symptomatic molar teeth [3].

2. *Neurogenic inflammation and susceptibility to local anaesthetic*: There is evidence to suggest that nerves arising from the inflamed tissue have altered resting potentials and lower thresholds for excitability and that they release neuropeptides capable of maintaining neurogenic inflammation [18]. A plausible explanation of anaesthetic failure in inflamed pulps is that local anaesthetic administration may thus not prevent evocation of central cortical action potentials in such situations [19–21]. There is also enhanced expression of Nav 1.8 tetrodotoxin-resistant Na + (TTX-R) voltage-gated sodium channels (VGSCs) in inflamed pulps [22, 23]. TTX-R Na + channels have been shown to be four times less sensitive to lignocaine [24]. Hyperalgesic agents such as prostaglandin E2 are also able to increase the activation-inactivation rate, decrease the threshold and increase maximal conductance of TTX-R Na + channels [25]. Injury to peripheral nerves which occurs in pulpitis may in addition result in a dramatic shift in transcription of neuropeptides, thus resulting in neuroplasticity (axon sprouting) to a variable length along the inflamed nerve [18].
3. *Demyelination and susceptibility to local anaesthesia*: Recently, pulp tissue extracted from human subjects with severe spontaneous pulpal pain has been shown to exhibit increased accumulations of multiple isoforms of sodium channels at atypical nodal sites with evidence of demyelination [26, 27]. These sites of demyelination could theoretically change the axon excitability characteristics with spontaneous nerve activity and pain paroxysms, which may not be blocked by local anaesthetic agents [17, 27].
4. *Central sensitisation*: Increased excitability of central neurons caused by a barrage of impulses sent to the trigeminal nucleus and brain from a tooth with severe pulpitis could result in exaggerated CNS responses to minimal peripheral stimuli and widening of the sensory receptive field [17]. Local anaesthetics are unable to block these signals transmitted from the brain and are considered by some to contribute to anaesthetic failure [17, 19]. Central changes such as increased c-fos expression in brain have been used as precise indicator of acute and persistent central neuronal reaction to peripheral nerve injury, inflammation and neuropathy [28]. It has been shown that c-fos expression is upregulated with pulpal inflammation. The role of central sensitisation in teeth with symptomatic irreversible pulpitis is well documented [18, 29]. Local anaesthesia can produce only partial and slow reversal of expansion in receptive fields and hyperexcitability associated with post-injury central sensitisation [30, 31].

From a practical perspective, it is unlikely that any of these processes act independently, in isolation to one another. It is more likely that multiple factors contribute to failure to achieve pulpal anaesthesia. Hence the strategies to manage should also be multipronged.

Strategies to manage failure of pulpal anaesthesia with an IANB in symptomatic irreversible pulpitis:

1. Premedication**:** Antibiotics have no role in the treatment of symptomatic irreversible pulpitis [32]. However, oral non-steroidal anti-inflammatory agents, particularly 600 mg ibuprofen administered 1 hour before an injection, have consistently been shown to be effective in improving the efficacy of an IANB in painful teeth [33, 34]. This technique should be employed whenever practically possible.

   Supplementary injections:

2. Several supplemental injections can be considered when block injections fail to provide sufficient anaesthesia. Options include:
   (a) *Block reinjection*: Where there is a suspicion that a block has been unsuccessful after a reasonable length of time, reinjection is warranted. However, if a patient has profound lip numbness, but still experiences pain with the endodontic treatment, reinjection is usually an unsuccessful procedure. Alternative block techniques, including a Gow-Gates block, can be sometimes helpful.
   (b) *Buccal infiltration*: Supplementing an IANB with a buccal infiltration of articaine has been shown to be more successful than using an IANB alone in difficult cases [35]. Two recent systematic reviews have confirmed that the odds of getting successful pulpal anaesthesia with supplemental buccal infiltration with 4% articaine are higher compared to buccal infiltration with 2% lignocaine [33, 36]. However, with inflamed pulps there are still around 20% of patients who still experience operative pain even after premedication and using a buccal infiltration with articaine.
   (c) *Intraligimentary injection*: This involves firm administration of an anaesthetic solution into the periodontal ligament adjacent to the symptomatic tooth, either with a standard syringe, with a proprietary intraligimentary injection devices or with computer controlled devices, e.g. The Wand® or CompuDent® (CompuDent®, Milestone Scientific Inc., Deerfield, IL). Intraligimentary injections can provide immediate but short-term anaesthesia of a painful pulp.

      An intraligimentary injection is the most common supplementary injection used by endodontists when block anaesthesia does not provide sufficient anaesthesia**.**
   (d) *Intraosseous injection*: Correctly given, an intraosseous injection delivers a solution directly into the cancellous bone immediately adjacent to a painful tooth. An intraosseous injection given in the region of the first molar, after an IANB, will usually provide effective anaesthesia sufficient for pulp extirpation [37]. While this technique is not without problems, is cumbersome, requires special armamentarium and can increase the heart rate when administered with adrenaline-containing anaesthetic, it is nevertheless a very effective supplemental injection technique for anaesthetising a symptomatic tooth when IANB injections have proved to be unsuccessful. Anatomical considerations prevent its use in all parts of the mouth. A number of proprietary devices are available, e.g. Stabident and X-tip intraosseous systems.

(e) *Intra-pulpal injection*: This technique, which involves the administration of local anaesthesia directly into a pulp, with sufficient back pressure, results in 100% success, but the duration of action is only short lived (10–20 min) [38]. While used as last resort, when properly administered, it is an extremely useful anaesthetic technique, particularly where the pulp is large and the exposure point is small. The patient must be prewarned that 'they will experience sharp pain' for just an instant. The jaw should be the firmly stabilised with firm finger rests. When the pulp is not exposed and all reasonable attempts have been made to anesthetise the pulp, access to the pulp can be obtained relatively atraumatically with a small thin bur. Once this drops into the chamber, it can be withdrawn and the local anaesthetic administered through this, using as much force as the syringe allows. Anaesthesia is obtained by the pressure applied to the pulp and not by the solution used. The success rate depends on whether adequate back pressure is maintained [1]. If the operator does not notice any back pressure, then it is unlikely that the procedure will be effective. Anecdotally the use of this technique is far less stressful to the patient than multiple attempts at anaesthesia, testing each time whether the tooth is 'completely numb now'.

(f) *Intracanal injection*: On occasions, removal of the coronal pulp can be completed, but the patient continues to experience sensitivity in one or other root canal. Anaesthesia can be obtained usually without pain in the same way making sure sufficient back pressure is developed.

(g) *Combination techniques*: Intraligimentary and intraosseous injections can provide pulpal anaesthesia but sometimes for only a short duration of time. Where it has been possible to gain sufficient anaesthesia to access the pulp through one of these techniques, it is still advantageous to immediately proceed to an intra-pulpal injection to ensure complete anaesthesia for pulp removal.

(h) *Sedation*: Managing a very nervous or uncooperative patient with a very painful molar can be stressful for both the operator and the patient, particularly when local anaesthesia has initially proved unsuccessful. Where oral and intravenous sedation or similar facilities are available, the use of these at least during the initial stages of treatment is a good adjunct to treatment ease and patient management.

3.3.1 Achieving Anaesthesia in Maxillary Teeth

Failure to gain satisfactory anaesthesia with maxillary teeth with acute pulpitis is not as common as with mandibular teeth. This is primarily due to the ease of access to the neural system via infiltrations. Nevertheless, problems still occur and supplemental anaesthesia may be required. There is no evidence to support the use of both buccal and palatal injections, although there is also no reason not to do so in difficult cases, provided the palatal injections are given slowly and not into tissues that are firmly attached to the tissues. Sloughing can occur

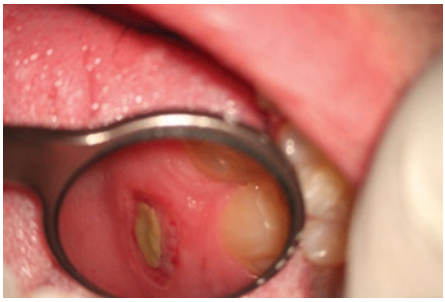

Fig. 3.1 Indirect view of palate where 4% articaine with 1:80,000 adrenaline was administered. Sloughing following vasoconstrictor induced ischemia is a more likely reason than the anesthetic per se

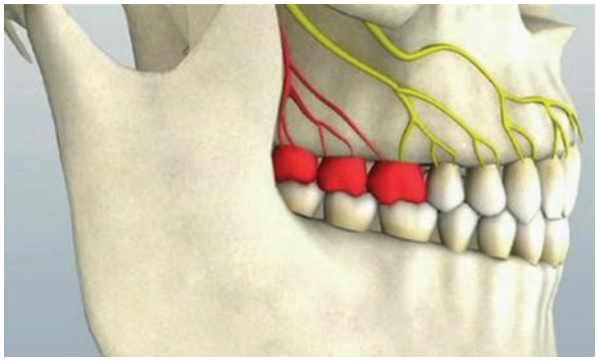

Fig. 3.2 Diagrammatic representation of nerve supply to apices of apices of maxillary teeth http://www.merckmanuals.com/professional/dental-disorders/symptoms-of-dental-and-oral-disorders/toothache-and-infection

(Fig. 3.1) due to prolonged ischemia, especially with use of higher concentration of adrenaline (1:50,000). The management is symptomatic and it resolves in a few days.

When anaesthetising symptomatic maxillary teeth, it is important to appreciate that the nerves to the teeth pass postero-anteriorly and that infiltrations for a first molar must be given both mesially and distally to anaesthetise all buccal roots (Fig. 3.2). Where pulpal anaesthesia is difficult for a molar, the injection should be given more distally and apically to the tooth.

Of note also that with long maxillary teeth, e.g., some canines, failure to achieve anaesthesia may be due the fact that the anaesthetic has not been delivered sufficiently close enough to the apex of the tooth.

With many maxillary teeth, infection and inflammation are in the area where an injection would normally be made. Effectiveness of a local anaesthetic solution is pH dependant. For a local anaesthetic solution to work effectively, the pH should be closer to physiological levels. Infected tissues usually have a lower pH. Thus, if local anaesthesia is delivered to a site that is inflamed or infected, its effectiveness may be severely diminished. The use of block injections, particularly an infraorbital block, is often necessary in these events.

Ophthalmic complications of maxillary injections and inferior alveolar nerve block can occur. Most of the reported cases are from single injections and due to inadvertent intravascular injections. If these do occur, it is important to reassure the patient about the transient nature of the problem and cover the eye until the corneal

reflex returns. The patient should be escorted home and not drive until vision returns completely to normal. Ophthalmic advice should be sought if symptoms persist for more than 6 hours.

3.4 Complications of Local Anaesthesia and Endodontic Treatment

3.4.1 Possible Mechanism of LA Nerve Injury

Nerve injury due to LA is complex. The nerve injury may be physical (needle, compression due to epineural or perineural haemorrhage) or chemical (haemorrhage or LA contents). Thus the resultant nerve injury may be a combination of peri-, epi- and intraneural trauma causing subsequent haemorrhage, inflammation and scarring resulting in demyelination (loss of nerve lining (Fig. 3.3) [39]. Local anaesthesia practice can cause injury to nerves in many ways. The location of nerve injury may also be important as well as mechanism. Factors that need to be considered are that only 1.3–8.6% of patients get an 'electric shock' type sensation on application of an IAN block and 57% of patients who suffer from prolonged neuropathy have not experienced the discomfort on injection, thus this is not a specific sign [78].

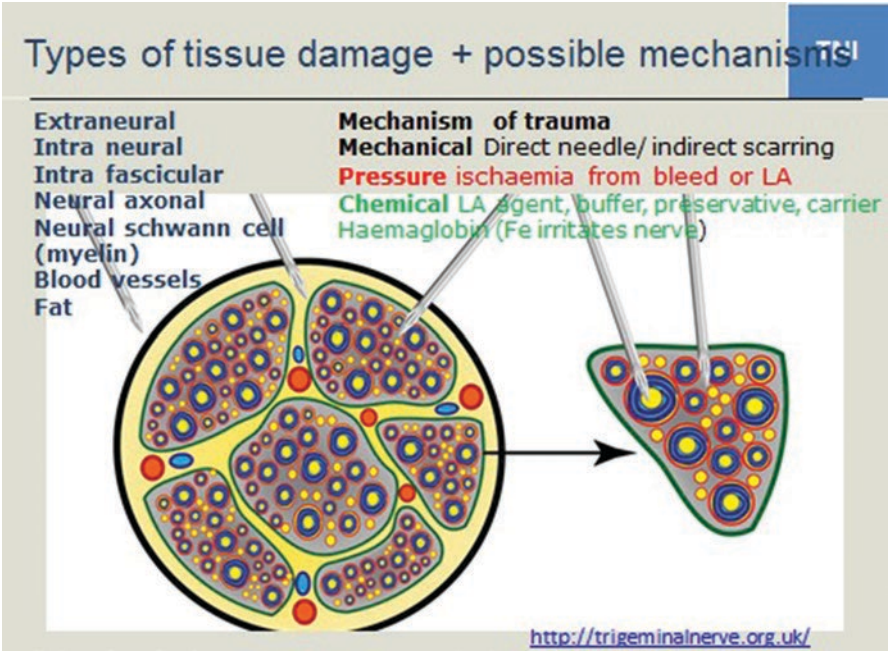

Fig. 3.3 Diagrammatic representation of mechanisms of nerve injury from IANB

3.4.2 Damage to the IANB

3.4.2.1 Neuropathy

Any damage to sensory nerve tissue may cause a mixture of anaesthesia (numbness), paraesthesia (altered sensation which is not painful), dysaesthesia (altered sensation which is uncomfortable/painful) and neuropathic pain. Neuropathy in the orofacial region must be taken seriously and the cause established, as there are some serious conditions that need to be eliminated if a cause is not obvious [42]. Often there is a history that helps determine the origin. Sensory deficits should be mapped on the face so that an estimate can be made of resolution or expansion of the deficit. Any patient with an expanding area of paraesthesia for which a cause has not been established should be referred for specialist evaluation. Amongst other things, paraesthesia can result from trauma, nerve injury, surgery, infections and prolonged reactions to local anaesthesia, viruses, malignancy or serious pathoses. If a dental cause is suspected, patients require reassurance, careful documentation and follow-up.

Unilateral paraesthesia and facial paralysis can occur in a misdirected block where local anaesthetic solution is deposited in the parotid gland. It usually occurs when the depth of needle penetration is nearly to the hub of a long needle. It is more likely to happen when the ramus of the mandible is flared laterally making it difficult for the operator to 'hit the bone'. The unilateral paralysis of facial muscles is reversible. This resolves over hours. The patient needs to be reassured, and the eye has to be protected until the blink reflex returns, as corneal reflex is often lost.

3.4.2.2 Neurological Injuries Resulting from Untreated Periapical Infections

It is not uncommon in endodontic practice to encounter patients with apical periodontitis of endodontic origin leading to sensory impairment of the inferior alveolar or mental nerve. This invariably resolves with the reduction in inflammation and local swelling. A number of authors have reviewed and documented clinical cases of neurological disorder with paraesthesia and hypaesthesia of the mental nerve resulting as a sequel of apical periodontitis of a mandibular second premolar and second molar teeth [40–42].

3.4.2.3 Motor Nerve Palsy Due to Endodontic Treatment

There are reports of facial motor nerve deficit as a result of endodontic treatment due to extrusion of endodontic irrigants, hydrogen peroxide and ethanol rinses [43, 44].

3.4.2.4 Paraesthesia and Pain Associated with Endodontically Related Nerve Injuries

Endodontic treatment of premolar and mandibular teeth has the potential to damage the inferior alveolar nerve via direct trauma, pressure or neurotoxicity. Trigeminal nerve injury is the most problematic consequence of dental surgical procedures with major medicolegal implications [44]. While incidence of lingual nerve injury has remained static over many years, the incidence of inferior alveolar nerve injury has increased; the latter likely being due to implant surgery and endodontic therapy [45].

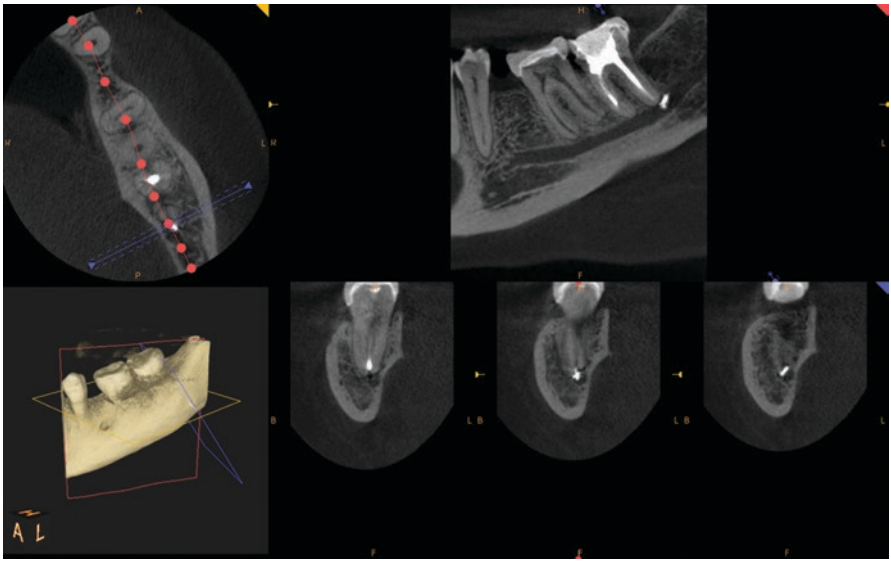

Fig. 3.4 CBCT imaging of a tooth that shows extrusion of endodontic sealant into the inferior alveolar canal causing pain and paraesthesia

Iatrogenic injuries to the third division of the trigeminal nerve remain a common and complex clinical problem (Fig. 3.4). Altered sensation and pain in region may interfere with speaking, eating, kissing, shaving, applying make-up, tooth brushing and drinking, in fact just about every social interaction we take for granted [46, 78]. Thus these injuries have a significant negative effect on the patient's quality of life, and the iatrogenesis of these injuries lead to significant psychological effects [47].

Persistence of any peripheral sensory nerve injury depends on the mechanisms, the severity of the injury, the increased age of the patient, the time elapsed since the injury and the proximity of the injury to the cell body (the more proximal lesions are having a worse prognosis). Most sensory nerve injuries related to dentistry are permanent. LA-induced nerve injuries have nearly 80% likelihood of recovery. Many authors recommend referral of injuries before 4 months [50], but this may be too late for many endodontically related peripheral sensory nerve injuries. Inferior alveolar nerve injuries related to endodontic treatment, particularly where there is extrusion of filling material, require immediate attention with permanency likely after 24–30 h [50]. After 3 months, permanent central and peripheral changes occur within the nervous system subsequent to such injury, and they are unlikely to respond to surgical intervention [48].

The IAN is at risk from a variety of endodontic procedures. The IAN is contained within a bony canal, predisposing it to ischemia, trauma and subsequent injury in relation to dental procedures. This may explain the higher incidence of permanent damage for inferior alveolar nerve injuries compared with lingual nerve injuries [45]. Inferior alveolar nerve injury during endodontic treatment can result from

local anaesthetic accidents, from irrigant extrusion and from over instrumentation or overfilling, in all cases resulting in one or a combination of mechanical injury, haemorrhagic, ischaemic or chemical injury to the nerve.

There are relatively few detailed reports on nerve injuries resulting from endodontic treatment [42]. The largest series of endodontic-related trigeminal nerve injuries included 61 patients reviewed over an 8-year period [9], with most of these patients presenting with persistent pain. There are relatively few reports of persistent pain subsequent to endodontic procedures [49]. In a recent study of 216 patients with trigeminal nerve injuries related to dentistry, 70% are reported to have chronic neuropathic post-traumatic pain [50].

Neuropathic pain (NP) syndromes are chronic pain disorders that develop after a lesion involving the peripheral or central nervous structures that are normally involved in signalling pain. The characteristics of NP differ substantially from those of other chronic pain states, i.e. chronic nociceptive pain, which develops while the nervous system that is involved in pain processing is intact. Apart from the existence of negative somatosensory signs (deficit in function), there are other features that are characteristic of neuropathic conditions (allodynia, hyperalgesia and hyperpathia) [51]. Paraesthesia is typically described by patients as bothersome but not painful. Furthermore, NP states require different therapeutic approaches such as anticonvulsants, which are not effective in nociceptive pain [52].

Thus symptoms experienced by patients with post-traumatic neuropathy of the trigeminal nerve can range from next to no symptoms, such as minimal anaesthesia in a small area to devastating effects on the patient's quality of life [53].

3.4.2.5 Local Anaesthesia-Related Neuropathy During Endo Procedures

As stated earlier, local anaesthesia is often complex in endodontics as the patients often experience difficulty in achieving analgesia. Patients undergoing endodontic treatment often have multiple injections and may be more at risk of local anaesthetic-related nerve injuries [54]. More recently the incidence of nerve injury in relation to IANBs has been calculated as 1:609,000 but with a reported increase in injury rate when 4% anaesthetic agents are used [55]. These LA injuries are associated with a 34–70% incidence of neuropathic pain, which is lower when compared with endodontic-related nerve injuries. Recovery is reported to take place at 8 weeks for 85–94% of cases [56].

LA injuries may have a better prognosis than nerve injuries resulting from endodontic treatment. In both cases, the nerve injury may be physical (compression due to epineural or perineural haemorrhage or extruded material) or chemical (haemorrhage or local anaesthetic or endodontic compound contents). There may be elements of direct mechanical trauma, which generally would be worse in endodontically caused nerve injuries.

Intraoperatively all clinicians should document unusual patient reactions occurring during application of local analgesic blocks (such as sharp pain or an electrical

shock-like sensation), and multiple blocks should be avoided if possible as they may also increase the risk of local anaesthetic-related nerve injuries.

3.4.3 Assessment of Trigeminal Nerve Injuries

The emphasis in trigeminal neuro-functional studies has been on using conventional mechanical tests, which are subjective. Due to the variability in methodology and reporting, they are of limited value for inter-study comparisons and little clinical significance in relation to a patient's pain and functionality. Recently several investigators have recommended the use of the patient's report alone [53], in combination with subjective and objective neurosensory tests [50] or utilising quality of life questionnaires (OHIP 14–31) for a more holistic approach for the assessment of patients with trigeminal nerve injury [57]. In this way hopefully to make studies easier to compare.

3.4.4 Management of Neuropathy Related to Endodontics

Differentiation between the possible causes of nerve injury, whether it is of local anaesthetic, periapical inflammation [50] or endodontically origin, is difficult. An accurate diagnosis is dependent on a thorough history and specific identification of the neuropathic area distribution. On occasions the use of adjunctive antibiotics or NSAIDS and paracetamol may enable the clinician to exclude inflammatory or infection-related pain as against post-traumatic NP.

3.4.4.1 Management

There is limited evidence base for managing dental LA-related nerve injuries. Unfortunately, a quarter of the cases are permanent and never recover. There is no 'magic bullet' to fix them; we have to wait and reassure the patient. In order to maximise resolution of any sensory neuropathy, it has been recommended to institute early medical intervention. The general medical practitioner should be involved and should prescribe the medication:

- *Steroids*: Step-down 5-day course of prednisolone, oral 50 mg, 40 mg, 30 mg, 20 mg and10 mg for 5 days
- *NSAIDs:* Ibuprofen oral 400–600 mg 6 hourly
- *Vitamin B* complex

Patients who report severe electric-like pain (though it is not a definite sign) following the administration of an IANB should be followed up [63]. What is important, however, is to inform all patients of the risks of problems that may result from IANB, however slight the risk, beforehand and do everything in your power to minimise the risks. Such as minimising repeat blocks and using supplementary techniques.

Some factors to consider in planning procedures which may influence the safety of the procedures include:

- *Block anaesthesia*: **Consider first whether block anaesthesia is really required**. By avoiding IANBs there is less risk of injury to the lingual and inferior alveolar nerves, which though rare, is debilitating to the patients and has no cure. Infiltrations provide more localised and shorter lasting anaesthesia which is of benefit to the patients. This technique requires less skill and less discomfort for the patient during the injection and avoids unnecessary lingual anaesthesia after dental treatment. Recent studies [9, 59–62] have suggested that infiltration of 4% articaine in the mandibular molar region can result in anaesthesia of the lower first molar that is in most cases as effective as an inferior dental block.
- *Concentration of LA*: Any increased concentration of any agent leads to increased neural neurotoxicity [64].
- *Volume of LA*: There is no evidence to support the suggestion that increased volumes of solution result in more nerve damage, but as all chemicals are neurotoxic, it is dependent upon the proximity to the nerve, concentration, neural damage and additional volumes that 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. Alternatives to block injections may be indicated.
- *Type of LA agent*: Bupivacaine is the most neurotoxic of all LA agents.
- *Type of vasoconstrictor*: The role of vasoconstrictor in nerve damage is unknown.
- *Sedated or anaesthetised patients*: There is no evidence to support unresponsive patients are prone to nerve injury as they are less likely to protect themselves when the IDB needle encroaches too close to the nerve.
- *Lack of LA aspiration*: There is no evidence to support that aspiration during an IANB results in lower persistent neuropathies though it is always advisable.
- *Patient factors*: Patient factors include age (>50 years of age), migraines, patients with existing neuropathic pain conditions (fibromyalgia) and those predisposed to developing peripheral neuropathy.

Acute Management

Should endodontically induced nerve injuries be suspected, acute management involves urgent referral. Some patients may require surgery. The largest series reported to date is by Pogrel [65], who described the management of 61 patients with endodontically induced nerve injuries over an 8-year period. Eight patients were asymptomatic and received no treatment. Forty-two patients exhibited only mild symptoms or were seen more than 3 months after undergoing root canal therapy. They received no surgical treatment. Only 10% of these patients experienced any resolution of symptoms. Eleven patients underwent surgical exploration. Five of these patients underwent exploration and received treatment within 48 h, and all recovered completely. The remaining six patients underwent surgical exploration and received treatment between 10 days and 3 months after receiving endodontic therapy. Of these six patients, four experienced

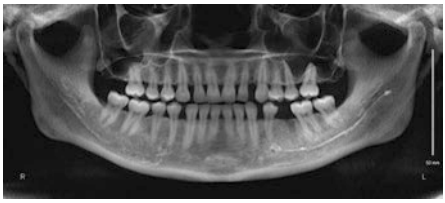

Fig. 3.5 Panoramic radiograph showing extruded sealer material in the left inferior alveolar canal resulting from endodontic treatment

partial recovery and two experienced no recovery at all. Thus Pogrel [65] concludes that early surgical exploration and débridement may reverse the side effects (pain and paraesthesia) of endodontic treatment on the inferior alveolar nerve. If the patient has continued symptoms of paraesthesia or pain once the local anaesthetic has worn off and the radiograph obtained after endodontic therapy shows sealant in the inferior alveolar canal (Fig. 3.5), then immediate referral to an oral and maxillofacial surgeon is indicated. Immediate surgical exploration and débridement may provide satisfactory results.

Escoda-Francoli et al. [66] described a case of endodontic treatment of a permanent right mandibular first molar in which the sealer cement overextended in large amounts and damaged the right inferior alveolar nerve. The condition reverted a few months after the surgical removal of the material. There are several reports of nerve injury in relation to over instrumentation and overfilling [67, 68]. Endodontic retreatment for inferior alveolar nerve injury was also reported by Yatsuhashi et al. [67]. Gatot and Tovi [68] recommended steroid therapy for early postoperative neuritis. More recently Grotz et al. [69] described the management of 11 patients with endodontic-associated neuropathy. They similarly reported that the neurological findings were dominated by hypaesthesia and dysaesthesia with 50% of patients reporting pain. Initial X-rays showed root filling material in the area of the mandibular canal. Nine cases were treated with apicectomy and decompression of the nerve; in two cases, extraction of the tooth was necessary. Only one patient reported persistent pain after surgery. Both Scolozzi et al. [70] and Brkic et al. [71] describe a limited series of patients successfully treated with surgical decompression (removal of material within the canal or apicecting the tooth) resulting in resolution of the endodontically related nerve injury. As stated earlier, patients must be assessed on a case-by-case basis, but immediate surgical exploration and débridement may provide satisfactory results.

Delayed Management

If the neuropathy is longer standing and the patient has chronic pain, then post-traumatic neuropathy must be diagnosed and appropriate treatment prescribed. Most patients accept a full explanation and reassurance for their symptoms. Many patients can find the explanation for their neuropathic pain symptoms very helpful. Antibiotics may be prescribed to exclude apical infection. NSAIDs and paracetamol will also exclude inflammatory pain, but neuropathic pain due to post-traumatic nerve injury will not respond to these medications.

Oshima et al. [72] reported that 16 patients of 271 patients presenting with chronic orofacial pain were diagnosed with chronic neuropathic tooth pain subsequent to endodontic retreatment. Most of these patients were treated for maxillary teeth, and 70% of the patients responded to tricyclic antidepressant therapy which

highlights the importance of establishing whether the patient has neuropathic pain to start with. A recent Cochrane review highlights the lack of robust evidence in managing trigeminal nerve injuries [73]. Recommendations for treatment of trigeminal neuropathic pain are also well described by Truelove [52].

3.4.5 Recommendations Based on the Current Evidence

Before undertaking endodontic care, practitioners should:

1. Preoperatively, identify teeth proximal to the IAN and take special care in preventing over instrumentation or extrusion of irritants.
2. Intraoperatively, recognise and record certain events that may be indicative of nerve injury, e.g. extreme patient discomfort during treatment including:
   - Intraoperative pain during irrigation
   - Intraoperative pain during preparation and filling
   - Inferior alveolar vessel bleed during preparation and delay filling
3. Postoperatively, continue to support and reassure your patient and advise them to visit websites designed to assist by post-traumatic neuropathy related to dentistry. *Always* undertake a *Homecheck* and arrange to contact your patients postsurgically the following morning. Neuropathy related to endodontics can be delayed, and the patient must be encouraged to report any change in sensation up to 3–4 days post-treatment (Renton et al. unpublished). If nerve injury is suspected, due to over instrumentation or deposition of endodontic material into the canal, the apex and/or tooth must be removed within 48 hours of placement in order to maximise recovery from nerve injury [65].
4. If the patient is insistent on keeping the tooth, urgent referral of the patient may be indicated for mandibular decompression and saline irrigation of the nerve and canal (Fig. 3.6) [65].

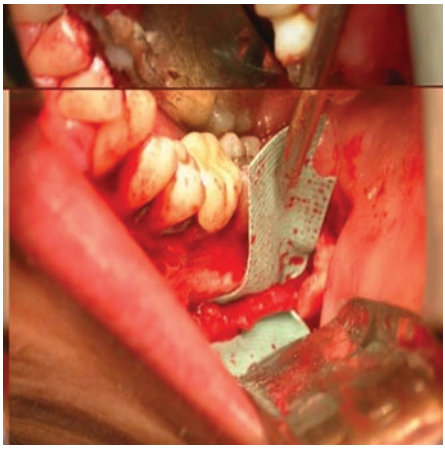

Fig. 3.6 Clinical picture of traditional approach for decorticating buccal aspect of mandible to gain access to inferior dental canal and IAN. This technique was recommended for IAN injuries presenting late, but some authors prefers access via the socket for recent injuries

Pain following root filling a tooth is a common occurrence, and this usually responds well to occlusal adjustment and the prescription of anti-inflammatory medication. If there is no evidence of overfill present, then pain may be due to:

- Apical inflammation (neuritis) confirmed by prescription of anti-inflammatories
- Chemical nerve injury from irrigants or filling material
- Thermal damage

However in the any event once neuropathy is identified, the clinician must reassure the patient, prescribe steroids (prednisolone step down such as 15 mg 5 days, 10 mg 5 days, 5 mg 5 days and high-dose NSAIDs and 600 mg ibuprofen) and make a timely referral to an appropriately trained micro-neurosurgeon if necessary; the clinical evidence is poor but despite this remains recommended practice; however, in vitro studies have demonstrated the possible benefit of these regimes [74].

3.4.6 Incidence of LA Nerve Injuries

Nerve injuries related to local anaesthetic injections are thankfully rare. Incident reports vary considerably. The incidence of IANB nerve injuries was first comprehensively reported by a retrospective examination of voluntary reported nonsurgical paraesthesia cases in Ontario, Canada, during the period from 1973 to 1993 [55]. Based on the number of cartridges used, these authors reported an incidence to be between 1.2 and 2.27 per 1 million injections depending on the type of anaesthetic used. The frequency of paraesthesia with articaine being twice that observed with other agents. Sambrook and Goss [64] estimated the incidence to be 1 in 27,415 cases of prolonged neuropathy related to IAN blocks per year in Australia. On the other hand, Haas and Lennon [55] found the incidence to be 1 in 785,000 injections, while Garisto et al. reported a rate of IANB paraesthesias to be 1 in 13,800,970. Renton et al. [75] reported a much higher incidence based upon surveys of dentists and specialists.

Two studies examining reports of paraesthesia from 1994 to 1998 and 1999 to 2008 have reported similar findings, implicating the local anaesthetics 4% articaine and 4% prilocaine with paraesthesia [76]. Other studies have corroborated these findings [56, 58, 76, 77]. All of the above studies found that the lingual nerve was most often affected during mandibular anaesthesia as compared to the inferior alveolar nerve.

3.4.6.1 Significance of IANB Nerve Injuries

The long-term significant problems seen in patients are severe. There is no ‘fix’ for these injuries, only prevention. Thus we can only wait for resolution, while managing the patient therapeutically, using medical and psychological interventions. Eighty-one percent of the IAN block nerve injuries resolve in 2 weeks [78]. Those with permanent injury often result in high levels of dysaesthesia and pain, mainly affecting the tongue with attendant social and psychological impacts [78].

While with consent, a patient may be aware of these rare but possible injuries, as no one else has ever heard of them or understands their nature; the resultant isolation for the patient is severe. There is significant stress to both dentist and patients.

3.5 Needle Breakage and Equipment Failures

With modern delivery systems, needle breakage is now a very rare complication. If breakage occurs, it invariably does so where the needle meets the hub. The needle may break if there is forceful contact made with bone or if the patient moves suddenly. Almost all needle breakages reported in the literature involve using 30 gauge needles used in block injections or in posterior superior nerve blocks. Long needles should therefore be used for block injections. Needles should not be inserted to the hub or bent at the hub prior to the injection in order to gain a better anaesthetic result. Both these procedures invite needle breakage if the patient moves suddenly. *Treating stressful patients with painful pulps is stressful, and care should not to rush any procedure, including anaesthetic procedures, as this may lead to problems including needle breakage and nerve injury.*

Should needle breakage occur during a block injection, it is imperative to keep the patient's mouth opening wide (mouth prop). If a long needle has been used and if the needle is visible, it can be grasped with a needle holder and removed. If the needle is not visible, then immediate referral is mandatory.

Conclusion

In this chapter, complications with dental anaesthesia in endodontics have been discussed. By far the greatest complication is failure to obtain good anaesthesia, particularly in mandibular molars with symptomatic irreversible. The causes and management of anaesthetic failure have been discussed. Complications relating to paraesthesia including that due to nerve injury from the delivery of local anaesthesia and endodontic treatment have been discussed in detail. These are often serious injuries. Guidelines are given for the prevention and management of these.

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