Post-surgical neuropathy of the trigeminal nerve

I. Incidence of pain

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Abstract

Few papers have addressed the clinical presentation or the implications of trigeminal nerve injuries in relation to dentistry. This study aimed to describe the cause, clinical signs and symptoms of patients with iatrogenic lesions to branches of the trigeminal nerve (n = 93 iatrogenic lingual nerve injuries (LNI); n = 90 iatrogenic inferior alveolar nerve injuries (IANI)). Pain history, pain scores using the visual analogue scale and mechanosensory testing results were recorded and analysed using the SPSS statistical software. Lingual nerve injuries were more prevalent than inferior alveolar nerve injuries, and more females were referred than males. Third molar surgery caused 74% of LNI, followed by 17% being caused by the local anaesthesia. More diverse procedures caused inferior alveolar nerve injuries, including third molar surgery (60%), local anaesthesia (19%), implants (18%) and endodontics (7%). Approximately 70% of patients presented with neuropathic pain, despite the additional presence of anaesthesia and/or paraesthesia. Neuropathy was demonstrable in all patients with varying degrees of loss of mechanosensory function, paraesthesia, dysaesthesia, allodynia and hyperalgesia. In conclusion, pain, as well as numbness, frequently occurs following iatrogenic trigeminal nerve injury similar to other post traumatic sensory nerve injuries. Neuropathic pain caused by dental procedure must be acknowledged by clinicians as a relatively common problem thus current informed consent for patients at risk of trigeminal nerve injuries in relation to dentistry requires revision.

Introduction

Iatrogenic injuries to the third division of the trigeminal nerve remain a common and complex clinical problem. Many authors have reported on the incidence, risks and causes of iatrogenic trigeminal nerve injury in relation to dentistry however few papers have addressed the clinical presentation and implications of these injuries (Hillerup, 2007; Hillerup 2008a). In addition there are no working criteria for trigeminal post surgical or post traumatic nerve injuries (Okeson 1996). Many chronic pain patients present with a distinct history and clear association with craniofacial or oral trauma (Benoliel et al 1994) and due to the lack of clarity of diagnostic criteria, the incidence of painful trigeminal permanent sensory dysfunction in the trigeminal system is unclear. MacDermid JC. **Measurement of health outcomes following tendon and nerve repair.** J Hand Ther. 2005 Apr-Jun;18(2):297-312.

The World Health Organization's model of health suggests that nerve injury outcomes should be assessed in terms of impairment, activity limitations, and participation restrictions. These surely should reflect the outcomes that should be evaluated for interventions for these conditions.

There are several reports on troublesome paraesthesia subsequent to infraorbital fractures (Benoliel et al 2005; Jungell et al 1987; Fogaca et al 2004; Vriens et al 1998). Previous studies also include reported. 3.3% of patients developed neuropathic pain after traumatic zygomatic fractures followed up for 6 month (Benoliel et al 2005) in comparison with 5-17% in other body regions (Macrae 2001 and Beniczzky et al 2005).

There are relatively few reports of persistent pain subsequent to dental procedures. Persistent pain after endodontics was found to occur in 3-13% of patients (Marbach et al 1982, Lobb et al 1996; Polycarpou et al 2005) whilst surgical endodontics resulted in chronic neuropathic pain in 5% of patients (Campbell et al 1990). Significant factors associated with persistent post endodontic pain included prolonged preoperative pain, female gender and previous chronic pain symptoms (Polycarpou et al 2005). In 135 patients with inferior alveolar nerve injuries caused by dental treatment or malignancy 22% presented with dysaesthesia which was significantly associated with the female gender (Caissei et al 2005). In another study of 449 patients with trigeminal

nerve injuries caused by dental treatment, paraesthesia was the most prevalent neurogenic symptom (53.5%) but more incapacitating symptoms such as dysesthesia (17.1%) and allodynia (4.5%) counted for a lot of suffering (Hillerup 2007a). In another report lingual nerve injury subsequent to dental surgery only 14% of 67 patients undergoing repair presented without painful symptoms. Also contrary to the findings of POGREL & KABAN 2003, surgical repair of LNIs provided no significant resolution of these neurogenic disturbances (Robinson etal 2000; Hillerup 2007b) providing an extremely poor prognosis for these patients.

A review of the epidemiology of painful diabetic peripheral neuropathy, postherpetic neuralgia, and less commonly studied neuropathic pain conditions.

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Although the burden of neuropathic pain is well-recognized, the descriptive epidemiology of specific neuropathic pain conditions has not been well-described. While painful diabetic peripheral neuropathy and postherpetic neuralgia have been widely evaluated, many other peripheral and central neuropathic pain syndromes have been less frequently studied. This review summarizes incidence and/or prevalence information about two relatively frequent neuropathic pain conditions-painful diabetic peripheral neuropathy and postherpetic neuralgia-and similarly summarizes the more limited epidemiologic information available for other peripheral and central neuropathic pain conditions. The data suggest that while our knowledge is still incomplete, the high frequency of several of these conditions in specific populations should be considered an important impetus for further studies designed to evaluate their contribution to the overall burden of neuropathic pain.

Neuropathic pain (NP) syndromes are chronic pain disorders that develop after a lesion of the peripheral or central nervous structures that are normally involved in signalling pain. It is estimated that about 35% of chronic pain patients suffer from NP, and that up to 5% of the population is affected (Rehm et al 2008).. 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. Furthermore, NP states require different therapeutic approaches such as anticonvulsants, which are not effective in nociceptive pain. (Scadding 2003).

Neuropathic pain is characterised by a variety of sensory symptoms. The most typical traits of NP that are often described by patients are paraesthesia, burning pain, shooting, electric-shock-like pain and evoked pain (hyperalgesia, allodynia). There is almost always an area of abnormal sensation (except in Trigeminal neuralgia) and he patient's maximum pain is often co-extensive with the area of sensory deficit. This is an important diagnostic feature for neuropathic pain. The sensory deficit is usually to noxious and thermal stimuli, which indicates damage to small-diameter afferent fibres or to the spinothalamic tract (Rehm et al 2008).

As well as the existence of negative somatosensory signs (deficit in function) there other features that are characteristic of neuropathic conditions. Paresthesias (ant crawling = formication and tingling) are symptoms typically described by patients that are bothersome but not painful. Painful positive signs are spontaneous (not stimulus-induced) or evoked types of pain (stimulus-induced pain, hypersensitivity). Spontaneous pain is separated into spontaneous ongoing pain, which often has a burning character, and spontaneous shooting, electric shock- like sensations. Evoked types of pain include mechanical hypersensitivity and hypersensitivity to heat and cold. Two types of hypersensitivity can be distinguished. First, allodynia is defined as pain in response to a non-nociceptive stimulus. The most common example is dynamic mechanical allodynia, which means that even gentle stroking of the skin may cause severe pain. Second, hyperalgesia is defined as increased pain sensitivity to a nociceptive stimulus. Another evoked feature is summation, which is the progressive worsening of pain evoked by slow, repetitive stimulation with mildly noxious stimuli, for example a pinprick. A small percentage of patients with peripheral nerve injury have a nearly pure hypersensitive syndrome in which no sensory deficit is demonstrable. Although all of these characteristics are neither universally present in nor absolutely diagnostic of neuropathic pain, the diagnosis of NP is likely when they are present. (Rehm et al 2008).

Neuropathic pain conditions involving the orofacial region include, Mono neuropathies including post traumatic nerve injury, Trigeminal neuralgia, Atypical TN, post herpetic neuralgia, Glossopharyngeal neuralgia, connective tissue disease, malignant or radiation plexopathy and possibly burning mouth syndrome. Poly neuropathies causing neuropathic pain in the orofacial region include diabetes, multiple sclerosis, HIV, chemotherapy, alcoholism, amyloidosis, drugs and idiopathic small fibre neuropathy.

Thus symptoms experienced by patients with iatrogenic trigeminal nerve lesions can range from next to nothing, such as minimal anaesthesia in a small area to devastating effects on the patient's quality of life. Various assessment methods are required to quantify the level of discomfort experienced by patients, the associated functional disability as well as diagnose the sensory impairment. Assessment should also provide accurate monitoring of sensory and functional recovery ideally with criteria for intervention where necessary. The tests available to the clinician, for the assessment of trigeminal nerve injury, are predominantly subjective, although occasionally objective tests are used. The results of any subjective (psychophysical) clinical test will depend on good communication between the patient and the clinician; ultimately the outcome of the assessment will relate to the patient's perceived experience and their interpretation of how to report it. Objective assessment excludes the higher cognitive responses of the patient, relating accurately to neurophysiological events, but omitting the patient's perceived effects. Tests applied in the three longitudinal studies of lingual nerve injury were two point discrimination, light touch, pin prick, noxious heat and functional questions (Mason 1988, Blackburn, 1990, Renton et al 2005). The emphasis in most trigeminal neurofunctional studies is on using conventional mechanical tests which are subjective, and due to the variability in methodology and reporting, are of limited value for inter study comparisons and bare little clinical significance in relation to pain and functionality. Recently several investigators have recommended the use of the patient's report alone (Westermark et al 1998, Ylikontiola et al 2000), in combination with subjective and objective neurosensory tests (Zuniga et al, 1998, Essick, 2004) or utilising quality of life questionnaires (OHIP 14- Susarla et al 2007) for a more holistic approach for the assessment of patients with trigeminal nerve injury.

Subsequent to surgery patients often expect and experience significant improvements in their jaw function, dental, facial and even overall body image after oral rehabilitation (Kiyak et al, 1990). Due to neuropathic pain many patients experience difficulties with daily function. This particularly impacts on patients with trigeminal nerve injuries as most social interactions involve this nerve (speaking, eating, drinking, kissing, facial expression, make up application and shaving). Similarly if you experience evoked pain due to touch or cold this will also significantly impact on sleeping or going out. Thus, when these iatrogenic injuries occur, the patients' quality of life may significantly diminish and lead to significant psychological problems (Abarca et al 2006). This inevitably results in increased patients' complaints, litigation and malpractice suits, as well as great embarrassment to the practitioner who caused the damage (Cassie et al., 2005; Hillerup, 2007). The functional and psychological impact of these injuries, are reported in a separate papers.

The aims of this study were to therefore describe the signs, symptoms and functional status of patients with iatrogenic lesions to branches of the trigeminal nerve. The presentation is divided into three papers; Part1 Incidence of neuropathic pain, Part 2 Functional difficulties and Part 3 Psychological sequelae.

Methods

Subjects

A total of 221 patients with trigeminal nerve injuries collected over 3 years were consulted at the Dental Institute in King's College Hospital, London. 38 patients presenting with trigeminal neuropathy caused by neurological disease, malignancy, multiple sclerosis, sickle cell disease, known alcoholism, injury caused by non dental trauma, orthognanthic surgery, diabetes, HIV, post herpetic neuralgia, stroke and patients on chemotherapy. The aetiology and functional status of 183 injuries to lingual or inferior alveolar nerves were evaluated. A simple scheme of a clinical neuro-sensory examination was applied to enable a quantified rating of the perception.

Assessment methods

Assessment of patients with iatrogenic trigeminal nerve injury included psychometrics, pain history, related functionality and mechanosensory testing. Examinations took place in a quiet room with the patients at ease, and they were urged to concentrate on the neurosensory test. A detailed history was taken to include the date and mode of injury and the patients' self assessment of neurosensory function in terms of reduced function (hypoesthesia, anaesthesia), and neurogenic discomfort (paraesthesia, dysaesthesia, allodynia, dysgeusia, ageusia, etc.) Provoking factors and pain characteristics were also obtained from the patients. The related interference with daily function, explored on a task basis, and psychological effects were specifically, identified the details of which are described elsewhere (Renton and Yilmaz, 2009 in press).

The neurosensory status of the injured nerve was clarified by carrying out a series of standardised tests of neurosensory functions (Hillerup, 2007) on all patients by the same observer (TR). The clinical examination was based on recommendations by Robinson et al. 1992 [33], which utilised a similar kit of instruments and each of the following neurosensory qualities:

1. Mapping neuropathic area % of dermatome (extra-oral and intra-oral)

2. Subjective function score. The patients were requested to assess their overall level of sensory function of the affected nerve using a subjective function scale ranging from 0–10 [0 = no perception of touch and 10 = normal perception] (Renton et al., 2005). All assessments/ratings were based on a comparison with the uninjured side.

3. Feather light touch—corner of tissue paper was gently pulled over the area to be examined and repeated 5 times, 3 positive responses was recorded as positive outcome.

4. Pin prick—the pointed end of a dental probe was gently touching the area to be examined with minimal pressure (assessment for hyperalgesia) and repeated 5 times, 3 positive responses was recorded as positive outcome.

5. Sharp blunt discrimination—the pointed and dull ends of a dental probe were gently touching the area to be examined with minimal pressure and repeated 5 times, 3 positive responses was recorded as positive outcome.

6. Brush stroke direction—a number 8 sale brush was gently drawn in a direction that would be recognized immediately in the healthy side (forwards, backwards, towards the middle, towards the side), and tested in the injured side (test for mechanical allodynia).

7. If the neuropathic area was large enough two point discrimination thresholds—a discriminator was employed, and the patients' ability to discriminate distance between points if the neuropathic area was large enough.

8. Pain VAS at rest and after mechanical and cold stimulation.

9. Patients with injury to the lingual nerve were examined for the presence of a traumatic neuroma. An unpleasant, radiating sensation in the injured side of the tongue induced by digital pressure to the region of suspected injury at the medial aspect of the mandibular ramus was interpreted as caused by a traumatic neuroma.

10. Fungiform count compared with contralateral uninjured side.

Statistics: All data was analysed using the SPSS statistical programme. Side differences between the healthy and the injured side were tested with Students' t test, and a Chi-square test was applied for non-parametric testing of frequencies. The value of p≤0.05 was chosen as level of statistical significance. Appropriate correlations were also carried out between certain data sets. The Microsoft Office 2003 program package was used to create the illustrations.

Results

Clinical characteristics of trigeminal nerve injury referrals to a university centre.

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The aim of this retrospective study was to determine the aetiology and characteristics of trigeminal nerve injuries referred to a university centre with nerve injury care. Fifty-nine patients with 73 injured trigeminal nerves were referred in 10 months. The most common aetiologies were odontectomy (third molar surgery) (52.1% of nerves), local anaesthetic (LA) injections (12.3%), orthognathic surgery (12.3%) and implant surgery (11.0%). The inferior alveolar nerve (IAN) was most commonly injured nerve (64.4%), followed by the lingual nerve (LN) (28.8%). About a quarter of IAN injuries (27.3%) and half of LN injuries (57.1%) from odontectomy had severe sensory impairment. There were twice as many LN than IAN injuries from local anaesthetic injections, but all had mild or no sensory impairment. Nerve injuries from implant surgery occurred only in IAN injuries; none had severe sensory impairment. Neuropathic pain occurred in 14.9% of IAN injuries and only in those with mild or no sensory impairment. Nerve surgery was offered to 45.8% of patients; a third underwent surgery.

Demographics

Injury to the lingual nerve was the most prevalent type of lesion (n=93; 52%), followed by the inferior alveolar nerve (n=90; 47%), and the buccal nerve (n=3; 1%). Patients were referred to us from all parts of the UK. The majority of nerve injuries were referred from specialist practitioners in secondary care trust (LNI = 50% and IANI = 32%) whereas general dental practitioners referred 40% of LNI and 51% of IANI patients (Figure 1). Time from injury to examination followed a skewed distribution with an arithmetic mean of 14.5 months (SD 28.0), and a median value of 8 months, range 0–430 months. Most patients were seen within a year after the injury. Injuries were regarded as being permanent if the patient had their symptoms for more than 3

months. Many of the LNI and IANI patients had permanent injuries (63.4% and 54.8%, respectively) and females were more likely to suffer from permanent nerve injury ($p>0.001$). Only 12.9% and 5.4% of the LNI and IANI cases were temporary. Permanency of the remaining 23.6% LNI and 39.8% IANI cases could not be elucidated because the consultation took place within 3 months after the injury and females were significantly more likely to suffer from permanent nerve injury ($p<0.001$).

LNI patients presented with a mean age 38.4 years (range 20-64) and IANI patients presented with a mean age 43.8 years (range 22-85). Significantly more females suffered from injured nerves (63% of LNI patients, $p=0.01$ and 61% of IANI patients, $p=0.003$), but there was no significant difference in the severity of affection between females and males. Although IANI patients suffered from a larger mean neuropathic area, their mean subjective function was slightly better than the LNI patients (Table 2). The range of subjective function values indicated that more IANI patients suffered from hypersensitivity and possibly hyperalgesia/allodynia. The size of the extra-oral and intra-oral neuropathic area significantly correlated with the gender of only the IANI patients ($p < 0.001$ and $p = 0.01$, respectively).

Third molar surgery (TMS) and local anaesthesia caused the majority of IANIs and LNIs (Figure 2). A more diverse range of procedures, including implant placement and endodontic treatment caused IANIs. TMS carried out under general anaesthesia resulted in significantly larger intra-oral neuropathic areas (80%) in comparison to TMS carried out under local anaesthesia amongst both groups of patients ($p<0.01$).There appeared to be no significant difference between the incidences of IANI or LNI caused on the right or left side of the mouth ($p>0.05$). Likewise, the cause of injury did not correlate with the permanency of the injury.

Subjective signs and symptoms

Approximately 70% of all patients presented with neuropathic pain, despite the additional presence of anaesthesia and/or paraesthesia. As summarised in figure 3, 70% of IANI patients suffered from paraesthesia predominantly in association with pain (47%) or numbness (48%). 59% of IANI patients complained of anaesthesia in combination with pain and/or paraesthesia. 67% of LNI patients complained of anaesthesia. 70% of LNI patients complained of pain or

discomfort often in combination with numbness (49%) and or paraesthesia (54%). 77% of LNI patients suffered from paraesthesia predominantly in association with pain (54%) or numbness (50%) (Figure 3). A significantly greater percentage of female patients in general, complained of evoked and spontaneous pain, paraesthesia and anaesthesia ($p<0.05$). A greater number of IANI and LNI patients reported evoked pain if their nerve injury was more than 4 or 6 months duration. However, the duration of the injury did not significantly affect the incidence of spontaneous pain. Patients who had their TMS carried out under local anaesthesia were significantly more likely to complain of evoked pain, evoked and spontaneous paraesthesia and numbness in comparison to those patients who had their TMS carried out under general anaesthesia. Age of the IANI and LNI patients did not correlate significantly with symptoms, neuropathic area or permanency of the injury. There was also a significant reduction in the number of LNI patients reporting spontaneous paraesthesia if they had their symptoms for more than 6 months.

The majority of patients with IANIs or LNIs suffered from painful altered sensation (dysaesthesia) (Figure 4). Most lingual nerve injury patients suffered from spontaneous either intermittent or constant paraesthesia (48%) however 30% of IANI discomfort was evoked. The most commonly reported character of pareasthesia was pins and needles (72% of IANI patients and 68% of LNI patients). Other reported altered sensation included burning (13% in the IANI patients; 25% in LNI patients), swollen sensations (8% more in the lip than the tongue), formication (2% in lip) and cotton-wool type feeling in the mouth of a patient with LNI (Figure 5).

Neuropathy was demonstrable in all patients with varying degrees of loss of mechanosensory function, paraesthesia, dysaesthesia (in the form of burning pain) allodynia and hyperalgesia (Figure 6). Patients with IANI suffered mostly from mechanical allodynia, followed by cold allodynia (Figure 7). Consequently, these patients tended to avoid ice-cream and covered up well in cold weather. A lower percentage of LNI patients complained of mechanical and cold allodynia in comparison to IANI patients. Intra-oral heat allodynia, taste allodynia and allodynia to spice was only present in (**how many?**)LNI patients (Figure 7). Some of the other tastants that provoked symptoms amongst the LNI patients included salty food, red wine, ginger, mint, citrus flavour and fizzy drinks/flavours. Despite the presence of taste allodynia amongst LNI patients,

the number of fungiform papillae on the injured side of the tongue decreased in comparison to the contralateral, uninjured side amongst 38% of LNI patients.

Sensory impairment

IANI and LNI patients in this study suffered significantly from reduced mechanosensory function, such as light touch (LT), two-point discrimination (TPD), sharp-blunt discrimination (SBD), moving-point discrimination (MPD) (Figure 8). A greater percentage of IANI patients, however, showed elevated responses to more than one of the tests carried out, which correlates positively with increased incidence of cold and mechanical allodynia amongst these patients.

1. Feather light touch (LT): Almost half of the IANI and LNI patients had reduced or no response to light touch (LT) (Figure 8). An elevated response to LT was seen in approximately 15% of the patients, suggesting hypersensitivity to touch, and possibly mechanical allodynia. The main difference between the two groups of patients was that more IANI patients had a normal response to LT than the LNI patients.

2. Sharp blunt discrimination

Pin prick, or sharp-blunt discrimination (SBD) responses varied between the IANI and LNI groups. More LNI patients had reduced responses to SBD than IANI patients, who showed equally reduced or elevated responses to SBD. %?? What about mechanical hyperalgesia?? Increased pain on pin prick assessment

3. Brush stroke direction (moving point discrimination [MPD]):

Although the percentages of IANI and LNI patients with reduced responses to MPD were similar (at 19% and 23% respectively), 6% of IANI patients showed hypersensitivity. An elevated MPD response was not indicated by any of the LNI patients.

4. IANI and LNI patients had mostly lost or decreased their ability to discriminate between two points. Conversely, a small percentage of patients showed elevated TPD thresholds (Figure 8).

5. Pain VAS at rest and after mechanical and cold stimulation:

IANI and LNI patients reported moderate to severe pain at rest and at its worst. VAS scores upon mechanical stimulation of the tongue and gingivae increased immensely, to a mean score of 12/10, therefore indicating mechanical allodynia. Stimulation with EC did not cause significantly more pain amongst the patients.

6. An unpleasant, radiating sensation in the injured side of the tongue upon palpating the region of suspected injury at the medial aspect of the mandibular ramus, may indicate that a traumatic neuroma was present amongst 19% of LNI patients.

Discussion

Although the incidence, risks and causes of iatrogenic trigeminal nerve injury in relation to dentistry has been discussed previously (Hillerup, 2008; Hillerup, 2007; Zuniga et al., 1998; Robinson, 1988), very few papers have addressed the clinical presentation and implications of these injuries. Our study therefore aimed to identify and describe the signs and symptoms experienced by patients with post-traumatic inferior alveolar nerve injury (IANI; n=90) and lingual nerve injury (LNI; n=93). Functional and psychological effects of these nerve injuries are addressed in separate papers.

Cause of injury

Injury to the third division of the trigeminal may occur due to a variety of different treatment modalities, such as major maxillofacial and minor oral surgery including; third molar surgery (Blackburn, 1990), implant treatment (Kraut & Chahal, 2002; Wismeijer et al., 1997), injection of local analgesics (Hillerup and Jensen, 2006),and endodontic treatment (Grotz et al., 2005). The wide variety in the cause of trigeminal nerve injuries makes it very difficult to establish the true incidence of these injuries. However, the prevalence of temporarily impaired lingual and inferior alveolar nerve function is thought to range between 0.15–0.54% whereas permanent injury caused by injection of local analgesics is much less frequent at 0.0001–0.01% (Hillerup, 2007). Persistence of any peripheral sensory nerve injury depends on the severity of the injury, increased age of the patient which did not correlate with permanency in tis study but other factors include, the time elapsed since the injury and the proximity of the injury to the cell body (the more proximal lesions have a worse prognosis) (Birch R 2006).

What is rarely highlighted is that the lingual and inferior alveolar nerves are different 'beasts'. The lingual nerve sits in soft tissue and is more likely to be prone to compression mechanical type injuries, particularly related to lingual access third molar surgery, compared with the IAN that sits in a bony canal is more likely to be exposed to mechanical or hemorrhagic compression and chemical endodontic injuries which may explain the slight preponderance of LNIs in this study in contrast.to previous reports (Hillerup 2008). Causes of lingual nerve injury (LNI) include third molar surgery and dental local anaesthetic injections, intubation, ablative surgery

and submandibular gland surgery (Hillerup and Jensen, 2006; Pogrel and Thamby, 2004). The most common cause of LNIs is third molar surgery in this study and has previously been reported with an incidence of 11.5% temporary and 0.6% permanent (Mason, 1988). Many reports have highlighted increased lingual nerve injury with lingual access surgery (Robinson et al 1986; Renton et al 2003; Pilcher and Bierne 2001) This may explain why LNIs were more prevalent in our patient cohort as frustratingly lingual access surgery for third molar surgery remains the norm in many parts of the UK contrary to USA, Asia and Europe. The authors hope that with formalized oral surgery training pathways this high risk lingual access surgical approach for mandibular third molar surgery will phase out.

Third molar surgery (TMS) and local anaesthesia caused the majority of IANIs and LNIs in this study. Implants and endodontic caused the rest of the IAN injuries however the cause of injury did not correlate with the permanency of the injury in this study. Third molar surgery causes the highest incidence of nerve injuries in our study similar to previous reports (Bataineh, 2001; Carmichael & McGowan, 1992; Fielding et al., 1997; Hillerup 2007, 2008a and b). The inferior alveolar nerve injuries (IANIs) are most likely occur due to the close anatomical location of lower third molars to the inferior alveolar nerve (Howe and Poynton 1966; Rud 1988 a and b, Rood & Shebab 1991). The incidence of nerve injury in relation to these 'high risk' third molars can be reduced with the coronectomy approach (Renton et al 2005; Pogrel 2004) however none of the patients seen in this study with third molar related IAN injury were offered a coronectomy procedure which may have ben appropriate. TMS carried out under general anaesthesia resulted in significantly larger intra-oral neuropathic areas (80%) in comparison to TMS carried out under local anaesthesia amongst patients with either IANI and LNI, which may reflect the increased difficulty of the surgical procedures being selected for GA (Brann et al 2001??). The inferior alveolar nerve (IAN) neuropathy related to third molar surgery or inferior alveolar block injections is usually temporary but can persist and become permanent (at 3 months) (Hillerup, 2007).

Local analgesic (LA) related trigeminal nerve injuries was the second most common cause of IANI and LNIs in this study 19 and 17% respectively. Reports of incidences include 1:588,000 for Prilocaine and 1/440,000 for Articaine IAN blocks which is 20-21 times greater than for

Lidocaine injections.[15,16] Perhaps every full time practitioner will find he or she has one patient during his or her career who has permanent nerve involvement from an inferior alveolar nerve block and there is no means of prevention'.[15] However the true incidence is difficult to gauge without large population surveys. These injuries are associated with a 34%[15] and 70%[2] incidence of neuropathic pain which is high when compared with other causes of peripheral nerve injury. Recovery is reported to take place at 8 weeks for 85-94% of cases.[17] The problem with these injuries is that the nerve will remain grossly intact and surgery is not appropriate as one cannot identify the injured region, thus the most suitable management indicated is for pain relief if the patient has chronic neuropathic pain.[2] 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). Some authors infer that the direct technique involving 'hitting' bone before emptying cartridge and withdrawal of needle may cause additional bur deformation at the needle tip thus 'ripping' the nerve tissue.[20] 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, thus this is not a specific sign. Also 81% of IAN block nerve injuries are reported to resolve at 2 weeks post injection.[18] Chemical nerve injury may also be related to specific chemical agents[21] and the LA components (type of agent, agent concentration, buffer, preservative). It may be the concentration of the local anaesthetic agent that relates to persistent neuropathy, based on evidence provided in studies by Perez Castro et al (2009)[22] where increasing concentration of local anaesthetic agent significantly affected the survival rate of neurons in vitro. Epidemiologically several reports have highlighted the increased incidence of persistent nerve injury related to IAN blocks with the introduction of high concentration local anaesthetics (Prilocaine and Articaine both 4%) Hillerup and Jenson (2006)[18] Pogrel and.[23]

Implant related nerve injuries. A major concern that should not be ignored is the increased incidence of IANIs occuring as a complication to implant treatment (Bartling et al., 1999; Dao and Mellor, 1998; Kraut and Chahal, 2002; Worthington, 2004; Hillerup, 2008). The incidence of implant related inferior alveolar nerve (IAN) nerve injuries vary from 0-40%.[13,18,34-39] This study illustrates that 17% off IANIs were caused by implant surgery which is the highest incidence reported thus far. (Hillerup 2008a). Minimisig implant related nerve injury involves appropriate peoperative radiographic evaluation Harris et al (2002)[41]. Nazarian et al (2003)[44] noted several

modalities of implant related nerve injury which may include direct trauma, inflammation and infection are postoperative neural disturbances main causes. These injuries most likely occur during preparation rather than placement (Renton et al 2008 in press). They may be directly related to the depth of preparation, implant length or width.[45] There are rare reports of resolution of implant related IAN neuropathies at over 4 years (Elian et al., 2005) but these do not comply with normal reports of peripheral sensory nerve injuries (Robinson, 1988). The use of BiOss (pH 8.4) in close proximity to the nerve bundle should be avoided. Post operatively the patient should be contacted after the LA has worn off. If IAN injury is evident then consideration should be given to removing the implant within 24 hours of placement as removal later is unlikely to resolve the nerve injury (Khawaja and Renton 2009). Bone graft harvesting is also associated with IAN injuries. Again it is crucial that appropriate training, planning, assessment and training should be undertaken in order to minimise nerve injury.[47] Avoidance of implant nerve injury is sometimes attempted by using techniques including inferior alveolar nerve lateralization and posterior alveolar distraction, however, these high-risk procedures are more likely to result in inferior alveolar nerve defect regardless of the surgeon's experience.

Endondontic related nerve injury Serious mechanical and chemical damage may also occur from endodontic procedures (Grotz et al., 1998). Any tooth requiring endodontic therapy that is in close proximity to the IAN canal should require special attention. If the canal is over prepared and the apex opened chemical nerve injuries from irrigation of canal medicaments is possible as well as physical injury precipitated by overfilling using pressurised thermal filling techniques.[48,49] Post operative RCT views must be arranged on the day of completion and identification of any RCT product in the IAN canal should be reviewed carefully. If IAN function is compromised after LA has worn off then immediate arrangements should be made to remove the over fill. The optimum pH of an endodontic medicament is close as possible to that of body fluids, i.e., around 7.35 but current commonly used endodontic medicaments have pHs ranging from 2.9 to 12..45 which are likely to cause tissue necrosis and permanent nerve injury if placed close to nerve tissue. If endodontic nerve injury is suspected the post operative radiograph must be scrutinised for evidence of breach of apex and deposition of endodontic material into the IAN canal. If this is suspected the material, apex and or tooth must be removed within 24 hours of placement in order to maximise recovery from nerve injury.

Referral of patients

Time from injury to examination followed a skewed distribution with an arithmetic mean of 14.5 months (SD 28.0), and a median value of 8 months, range 0–430 months. Most patients were seen within a year after the injury. Injuries were regarded as being permanent. Most of the IANI's were caused by third molar surgery (TMS) carried out by the general dental practitioner (GDP) which may reflect inadequet specialist training in oral surgery. However LNI patients were mainly referred from secondary care clinics, where the main cause of injury was TMS which may reflect persistence in the use of lingual access third molar surgery which the authors believe is inappropriate. . Patients who had their TMS under general anaesthesia were more likely to have an IANI with a larger neuropathic area, possibly because these were more complex cases. These results indicate that both GDP's and specialist oral surgeons in secondary care clinics need to take more care when carrying out such procedures. IANI's may be avoided by carrying out coronectomies, a procedure that involves removal of the crown of the wisdom tooth and leaving behind the roots (Pogrel et al., 2004, Renton et al 2005; Dolanmaz et al., 2009).

Unfortunately, many of the patients had permanent nerve injury, whereby the time from injury to examination ranged from 0-430 months. Full recovery of nerve function following injury is less likely when the patient is seen a long time after the injury, possibly due to the lack of neuronal regeneration. The cascade of events that occur following peripheral nerve injury closely recapitulate the events seen in development. Interruption of the transport of neurotrophic factors from the peripheral target tissue to the cell body causes cellular deprivation of trophic factors resulting in cell death depending on the proximity to the cell body (Aldskogius & Arvidsson, 1978). Neuronal regeneration involves a variety of cells (Schwann cells, macrophages, fibroblasts and endothelial cells) and processes (apoptosis, neurotropism and path finding) (Rath, 2001). The expediency of referral of these patients will depend on the type and cause of injury. There is increasing evidence that implant or endodontic related injuries should be managed within 24 hours in order to maximise resolution of neuropathy (Khawaja and Renton 2009). Faster referral of cases to specialist oral surgeons within 3 months after third molar surgery injury may therefore help prevent the injury from becoming permanent by interrupting and possibly reversing this cascade of events that occur after nerve injury (Susarla 2005; Ziccardi 2007). There will, however, be the unfortunate cases where the nerve injury caused by local anaesthetics or delayed

referral of implant or endodontic injuries which can not be treated surgically in which case the patients should be reassured about their condition and referred for counselling if required with medication for the associated pain.

Patient age and gender

LNI patients presented with a mean age 38.4 years (range 20-64) and IANI patients presented with a mean age 43.8 years (range 22-85). These age ranges were similar to previous reports of patient cohort with iatrogenic nerve injury (Hillerup 2007; Hillerup 2008a and b). Age did not have any significant effect on the permanency, neuropathic area or the symptoms experienced by the patients. This was rather surprising, since an increase in age has been shown to influence neuronal death and ability to regenerate (Griffin and Hoffman, 1993).

Significantly more females had IANI and LNI, showing similarities to previous studies (Hillerup, 2007; Gerlach et al., 1989; Sanstedt and Sorensen, 1995). Although Hillerup (2007) showed no gender-related difference in the severity of impairment of the nerve injuries, our study indicated that females were more likely to suffer from permanent IANI and LNI. Females may appear be more at risk of iatrogenic trigeminal nerve injuries because they are more likely to visit the doctor in general than males and are more likely to seek advice regarding pain (Ref??).

Significantly more female patients with IANI and LNI reported evoked and spontaneous pain, paraesthesia and anaesthesia, therefore supporting the study by Standstedt and Sörensen (1995) but in contrast to studies on post endodontic pain (Caissei). Increased reports of pain amongst females may be possibly due to their lower pain thresholds (Hurley and Adams, 2008) or related to their increased tendency to communicate their problems. Female patients with IANI suffered from significantly more of the dermatomes affected by neuropathy for extra-oral and intra-oral areas; a phenomenon which deserves further analysis.

Neurosensory assessment:
1. Pain and altered sensation

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The purpose of this study was to evaluate the opinions and practices of peripheral nerve surgeons regarding assessment and treatment of pain in patients following nerve injury. Surgeons with expertise in upper extremity peripheral nerve injuries and members of an international peripheral nerve society were sent an introductory letter and electronic survey by email (n = 133). Seventy members responded to the survey (49%) and 59 surgeons completed the survey (44%). For patients referred for motor or sensory dysfunction, 31 surgeons (52%) indicated that they always formally assess pain. In patients referred for pain, 44 surgeons (75%) quantitatively assess pain using a verbal scale (n = 24) or verbal numeric scale (n = 36). The most frequent factors considered very important in the development of chronic neuropathic pain were psychosocial factors (64%), mechanism of injury (59%), workers' compensation or litigation (54%), and iatrogenic injury (48%). In patients more than 6 months following injury, surgeons frequently see: cold sensitivity (54%), decreased motor function (42%), paraesthesia or numbness (41%), fear of returning to work (22%), neuropathic pain (20%), and emotional or psychological distress (17%). Only 52% of surgeons who responded to the survey always evaluate pain in patients referred for motor or sensory dysfunction. Pain assessment most frequently includes verbal patient response, and assessment of psychosocial factors is rarely included. Predominately, patient-related factors were considered important in the development of chronic neuropathic pain.

The majority of patients with IANIs or LNIs suffered from painful altered sensation (dysaesthesia). Most lingual nerve injury patients suffered from spontaneous either intermittent or constant paraesthesia (48%) however 30% of IANI discomfort was evoked. The most commonly reported character of pareasthesia was pins and needles (72% of IANI patients and 68% of LNI patients). A greater number of IANI and LNI patients reported evoked pain if their nerve injury was more than 4 or 6 months duration. However, the duration of the injury did not significantly affect the incidence of spontaneous pain which may indicate that these symptoms remain stable.. Patients who had their TMS carried out under local anaesthesia were significantly more likely to complain of evoked pain, evoked and spontaneous paraesthesia and numbness in comparison to those patients who had their TMS carried out under general anaesthesia. Age of the IANI and

LNI patients did not correlate significantly with symptoms, neuropathic area or permanency of the injury. There was also a significant reduction in the number of LNI patients reporting spontaneous paraesthesia if they had their symptoms for more than 6 months.

Both IANI and LNI patients reported moderate to severe pain at rest and at its worst. VAS scores upon mechanical stimulation of the tongue and gingivae increased immensely, to a mean score of 12/10, therefore indicating mechanical allodynia. **Cold allodynia??** A large proportion (70%) of patients with post-traumatic trigeminal nerve injury in our study, contrary to popular belief, presented with neuropathic pain. In a previous report only 10 patients (14%) presented without neurogenic discomfort in patients undergoing Lingual nerve repair (Hillerup 2007) however 30% of LNI patients reported pain which reduced to 26% after surgical intervention (Robsinson et al 2000).. Altered sensation in relation to lingual nerve injuries reported in this study was similar to previous reports including paraesthesia (60%), dysaesthesia (16%) allodynia (3%) (Hillerup and Stoltze 2007) and paraesthesia (50-80%) (Robinson et al 2000). Many patients in this study experienced pain together with other neurogenic malfunctions, such as paraesthesia and/or anaesthesia. 70% of IANI patients suffered from paraesthesia predominantly in association with pain (47%) or numbness (48%). In previous reports (Akal et al., 2000; Fielding et al, 1997; Gerlach et al., 1989; Haas and Lennon, 1995; LaBanc and Gregg, 1992) many authors simplistically separate out these symptoms implying that the patients only experience one or the other, this study illustrates that this is clearly not the case. These troublesome symptoms inevitably resulted in a severe reduction of their overall quality of life and the functional difficulties and associated psychological distress experienced by these patients are discussed in greater detail elsewhere (Renton & Yilmaz -2009 in press).

Long-term alteration of taste sensation following lingual nerve injury has been reported by Sandstedt and Sorensen (1995) who found that in 226 patients with trigeminal nerve damage, 56 percent had a taste alteration, Morton et al 2005 reported 97 percent had a sensory disturbance (hypoaesthesia or anaesthesia) and 92 percent had a paraesthesia (of varying intensity). In this study we considered that altered activity of the Chorda tympani was manifested by 'tastant allodynia' i.e. pain elicited with spice flavours, salty food, red wine, ginger, mint, citrus flavour HP sauce and fizzy drinks/flavours despite the decreased number of fungiform papillae on the injured side of the tongue. This is the first study to report such a phenomenon in iatrogenic nerve

injuries and causes the patients siginificant problems with eating. A decrease in the number and quality of fungiform papillae following lingual nerve section supports previous studies (Cheal & Oakley, 1977; Ogden, 1989; Robinson and Winkles, 1991; Ogden, 1996; Hillerup and Stoltze 2007; Robinson et al 2000) and possibly explains the lack of trophism to the fugiform papillae due to damage to the chorda tympani nerve.Tastant allodynia may be due to specific up-regulation in neural receptors that respond to these adjuvants including TRPV receptors and sodium channels (NAv 1.7 and 1.8) that have been shown to be upregulated in other trigeminal pain conditions (Renton et al 2003).

Surgically induced injury resulting in chronic neuropathic pain is now well established.(IASP 2008, Jung etal 2003; Kehlet 2002; Perkins 2000) Estimated incidences of chronic pain after various procedures are: leg amputation about 60%, thoracotomy about 50%, breast surgery about 30%, cholecystectomy 10–20%, and inguinal herniorrhaphy about 10%. Predictive risk factors for chronic postoperative pain are: preoperative pain, repeat surgery, psychological vulnerability, workers compensation, a surgical approach with risk of nerve damage, moderate or severe intensity of acute postoperative pain, radiation therapy, neurotoxic chemotherapy, depression, neuroticism, and anxiety. The main cause is injury to a major nerve at the time of surgery, which leads to neuropathic pain, in contrast to inflammatory pain. Injury to a nerve causes release of pain-causing molecules, setting up a "pacemaker-like" process. Mysteriously, chronic postoperative neuropathic pain develops in only a fraction of patients with a nerve injury, implying that other factors, such as genetic susceptibility or psychosocial factors are important. The mechanism for neuropathic pain is eloquently discussed by Woolf et al 2008 (Lancet ??)

The estimated incidence of chronic pain following surgery is higher than most surgeons realize, ranging from 10% following cesarean section or herniorrhaphy to 30% to 50% following amputation or coronary bypass surgery (Kehlet et l 2006). Most dental surgery is undertaken on an outpatient basis and not kept in hospital allowing better monitoring of post operative persistent pain. As a result one would expect the incidence of post surgical neuropathic pain relating to dentistry to be lower than other surgical procedures. However this study highlights that the incidence of pain, dysaesthesia and hyperaesthesia in post surgical trigeminal nerve injuries was very high compared with other post surgical neuropathic pain incidence. This high incidence of neuropathic pain may be explained, in part, by this cohort being self selected in that patients had

to persist or even demand referrals for their complex and painful symptoms. Possibly those patients with anaesthesia perhaps are not so debilitated, thus not seeking secondary or tertiary referrals. Patients with pain are more likely to have severe functional and psychological difficulties even without due consideration to the additional damaging effects of the iatrogenic nature

Measurement of health outcomes following tendon and nerve repair.

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The World Health Organization's model of health suggests that tendon and nerve injury outcomes can be assessed in terms of impairment, activity limitations, and participation restrictions. A tendon injury results in impairment of motion and strength of affected digits. Literature on outcome of tendon surgery has focused on active motion. Recently developed devices can be used to measure strength impairments associated with individual digits after tendon injury, although the importance of either grip or digital strength measures as indicators of post-tendon recovery has not been fully delineated. Published impairment rating scales have expressed outcome based on regained total active motion of relevant joints. These scales also tend to classify outcomes on a subjective four-point scale ranging from poor to excellent. Subjective ratings have not been validated, vary across scales, and inhibit meaningful comparisons by diluting information. Nerve injuries result in an impairment of motion, strength, sensibility, and sympathetic nerve function. Development of quantitative measures of sensibility continues to evolve, although all current methods have some limitations. Two-point discrimination was once a mainstay of assessment, but current evidence suggests it is less valid and responsive than other quantitative sensory testing. Cold sensitivity is common and can be measured through rewarming responses or by self-report. A comprehensive impairment rating scale for nerve injury with subscales addressing sensory, motor, and pain/discomfort domains has been developed. Use of this validated instrument will facilitate more meaningful comparisons across centers and studies. Recent literature on treatment outcomes has focused on impairment measures with minimal attention to activity limitations and participation

restrictions. Validation of appropriate scales and inclusion of both impairment and disability measures in future clinical studies is required to fully understand health outcomes after tendon and nerve injury.

Ruijs AC, Jaquet JB, van Riel WG, Daanen HA, Hovius SE **Cold intolerance following median and ulnar nerve injuries: prognosis and predictors.** J Hand Surg Eur Vol. 2007 Aug;32(4):434-9. Epub 2007 May 4.

The study population consisted of 107 patients 2 to 10 years after median, ulnar or combined median and ulnar nerve injuries. Patients were asked to fill out the Cold Intolerance Severity Score (CISS) questionnaire and sensory recovery was measured using Semmes-Weinstein monofilaments. Fifty-six percent of the patients with a single nerve injury and 70% with a combined nerve injury suffered abnormal cold intolerance. Patients with no return of sensation had dramatically higher CISS-scores than patients with normal sensory recovery. Females had higher CISS scores post-injury than males. Cold intolerance did not diminish over the years. Patients with higher CISS scores needed more time to return to their work. Age, additional arterial injury, site or type of the injury and dominance of the hand were not found to have a significant influence on cold intolerance.

2. Neuropathy descriptors Pins-and-needles were the main complaints, followed by burning sensations in both groups. Fizzing and swollen sensations were also experienced by both groups. However, only the IANI patients complained of 'ants crawling across the area'-type sensations, otherwise known as formication. Itchiness and prickling, dull sensations were also experienced by only the IANI patients. A LNI patient also reported a cotton-wool type sensation within the mouth. The variation in reported symptoms probably reflects the difficulty patients have in describing their sensations and association of them with specific factors. Previous studies

3. Neuropathic area % of dermatome (extra-oral and intra-oral)

Extraorally IANI patients suffered form a mean of 55.5% of the dermatome affected by neuropathy and 57.7% intraorally. LNI patients suffered from 44% of the dorsal apect of the tongue 'dermatome' and 26.6% of the lingual gingivae. What was particularly of relevance was the significant prevalence of hyperalgesia on the affected gingivae.

4, Subjective function score Although there have been numerous studies evaluating trigeminal neurosensory disturbance, due to oral surgery, there seems to be no consensus as to the ideal choice of methods with which to measure such impairments. While such methods should be

precise enough to match the requirements of modern science, they should also be pragmatic enough to be used in an outpatient setting. IANI patients in our study showed elevated responses to more than one of the tests carried out, which correlated positively with increased incidence of cold and mechanical allodynia amongst these patients. In this study most patients suffered significantly from reduced mechanosensory function, such as light touch (LT), two-point discrimination (TPD), sharp-blunt discrimination (SBD), moving-point discrimination (MPD). A greater percentage of IANI patients, however, showed elevated responses to more than one of the tests carried out, which correlates positively with increased incidence of cold and mechanical allodynia amongst these patients.

5. Feather light touch (LT): Almost half of the IANI and LNI patients had reduced or no response to light touch (LT). An elevated response to LT was seen in approximately 15% of the patients, suggesting hypersensitivity to touch, and possibly mechanical allodynia. The main difference between the two groups of patients was that more IANI patients had a normal response to LT than the LNI patients suggestive of lingual mechanical hyperaesthesia.

6. Pin prick, or sharp-blunt discrimination (SBD) responses varied between the IANI and LNI groups. More LNI patients had reduced responses to SBD than IANI patients, who showed equally reduced or elevated responses to SBD. %?? What about mechanical hyperalgesia??

7. Brush stroke direction Although the percentages of IANI and LNI patients with reduced responses to MPD were similar (at 19% and 23% respectively), 6% of IANI patients showed hypersensitivity with dynamic mechanical allodynia. An elevated MPD response was not indicated by any of the LNI patients.
8. Two point discrimination thresholds. IANI and LNI patients had mostly lost or decreased their ability to discriminate between two points. Conversely, a small percentage of patients showed elevated TPD thresholds. Hillerup and Stoltze 2007 reported that.two-point discrimination thresholds of 2PD >20 mm.on the injured side compared with 6.3 mm(SD2.3) on the non injured side. Similar to this patient cohort.

9. Patients with injury to the lingual nerve were examined for the presence of a traumatic neuroma. An unpleasant, radiating sensation in the injured side of the tongue upon palpating the region of suspected injury at the medial aspect of the mandibular ramus, may indicate that a traumatic neuroma was present amongst 19% of LNI patients which is less than a previous study by Hillerup & Stoltze who reported that 53% displayed this positive sign which interestingly did not improve after reparative surgery.

Recommendations

There is a need for a consensus and standardisation of inferior alveolar and lingual nerve injury assessment in order to identify injury, whilst to simultaneously differentiate temporary from permanent injuries in the early postoperative period in order to expedite the appropriate selection of candidates for appropriate interventions. The authors recommend that a holistic approach would be of benefit to all clinicians and patients recognising the incidence of pain, related effect on functionality and psychological implications.

It is imperative that all patients undergoing procedures that place the trigeminal nerve at risk must be appropriately consented, surgical methods must be modified to minimise risk to the nerve and if injury does occur it must be recognised early on and appropriately referred to a specialist. Many authors recommend referral of injuries before 4 months (Hegedus & Diecidue 2006) but this may be too late for many peripheral sensory nerve injuries. More recently we have recommended early removal of implants as a strategy to optimise neuropathy resolution (Khawaja and Renton 2009). We now understand that after 3 months, permanent central and peripheral changes occur within the nervous system subsequent to injury that are unlikely to respond to surgical intervention (Susarla 2007; Ziccardi and Assael, 2001).

Consent procedures should be modified to alert patients to the possibility of chronic pain.
With regards informed consent all patients deserve to be provided with realistic expectations as to the risks and consequences of trigeminal nerve injury. Assessment of risk must be undertaken in order to appropriately advise the patient with regard to alternative treatment plans and include this possibility in the consent forms (Nazarian et al 2003). The information should be explicit with ensuring that the patient is aware that nerve injury may cause altered sensation (numbness,

pain or troublesome altered sensation) that may be intermittent or constant, temporary or permanent. The patient must also be warned that the neuropathic area may affect all or part of the IAN dermatome; extra- and intra-orally (whole of skin and vermillion of lip and chin on each side and all lower quadrant teeth and associated buccal gums) or LN dermatome (whole side of tongue and lingual gums. It may be important for clinicians to perform a neurosensory examination of mandibular nerve function before placing implants to determine whether there is pre-existing altered sensation as up to 24% of patients with edentulous mandibles may present with IAN neuropathy (Walton 2000). If the tooth is high risk (crossing both IAN canal LD on plane film) then the patient should be advised of increased risk of nerve injury and offered alternative surgical techniques that may minimise nerve injury. The possible outcomes of nerve injury for the patient are acceptable or unacceptable complete or partial resolution. The resolution may not be complete but if the patients are comfortable and functioning normally, they will not pursue further treatment however, if these persistent injuries are troublesome to the patient then surgical intervention may be indicated.

Conclusion:

Pain, as well as numbness and/or paraesthesia, may occur following iatrogenic trigeminal nerve injury. Based on these findings the authors hope to recommend best practice for informed consent for patients at risk of iatrogenic nerve injury in relation to dental procedures.

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3 independant reviewers

consensus of reviews

4. apply the findings to patient care.

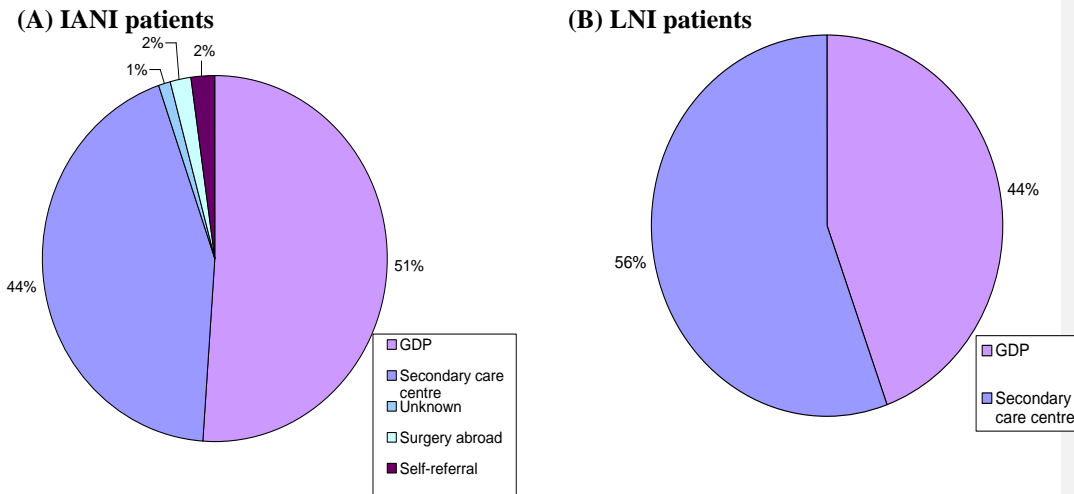**[Include this in part VII (consent paper) of the papers?]**

Table 1: Key clinical terms used for the analysis of patients' symptoms with nerve injuries (Burket *et al*., 1994; Dirckx and Stedman, 2001; Hillerup, 2007).

Term	Definition
Ageusia	Absence of gustatory perception.
Allodynia	Pain due to a stimulus that is not normally painful.
Anaesthesia	Complete loss of sensation due to pharmacologic depression of Aδ- and C-fibre activity within nerves, or from neurological dysfunction due to injury.
Dysaesthesia	Impairment of sensation that is either evoked or spontaneous, such as painful paraesthesia and burning.
Dysgeusia	Altered gustatory perception
Hyperaesthesia	Increased sensitivity to stimulation that is not necessarily painful but an exaggerated response to a specific sensory mode, such as touch, temperature or vibration.
Hyperalgesia	Increased response to a stimulus that is normally painful.
Mechanical allodynia	Pain to a mechanical stimulus, such as touch, that is not normally painful.
Paraesthesia	Abnormal sensation whether spontaneous or evoked, such as tingling, itching, pricking, or tickling.
Thermal allodynia	Pain to a thermal (warm/cold) stimulus that is not normally painful.

**Table 2: Summary of the neuropathic area affected and subjective function of the LNI and IANI patients.**

		Neuropathic area (%)	Subjective function	
			Minimum	Maximum
IANI	Intra-oral	57.7 [4-100]	2.0 [0-4]	8.0 [1-18]
	Extra-oral	55.5 [0.8-100]	4.3 [0-18]	7.6 [1-20]
LNI	Lingual	44.0 [2-100]	3.0 [0-8]	5 [0-12]
	Lingual gingivae	26.6 [2-100]		

Figure 1: Referral of (A) IANI and (B) LNI from primary or secondary care.

(A) IANI patients

2%
1%
2%
51%
44%

- GDP
- Secondary care centre
- Unknown
- Surgery abroad
- Self-referral

(B) LNI patients



44%
56%

- GDP
- Secondary care centre

Figure 2: Cause of the IANI and LNI.

The greatest majority of IANI and LNI were caused by third molar surgery, followed by the local anaesthetics (LA). Implants and endodontics were only caused IANI's.
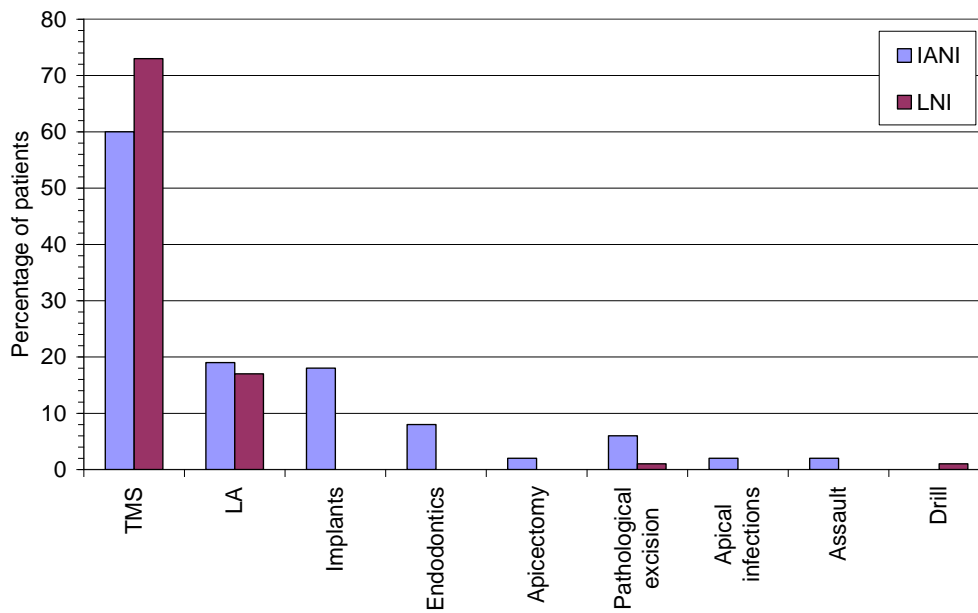
Figure 3: Summary diagrams showing the incidence of paraesthesia, anaesthesia and pain amongst the (A) IANI and (B) LNI patients.

(A) IANI patients
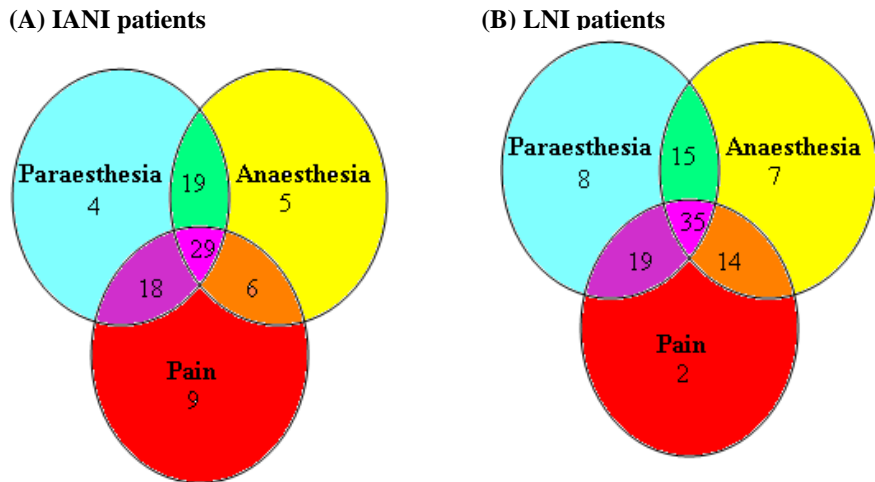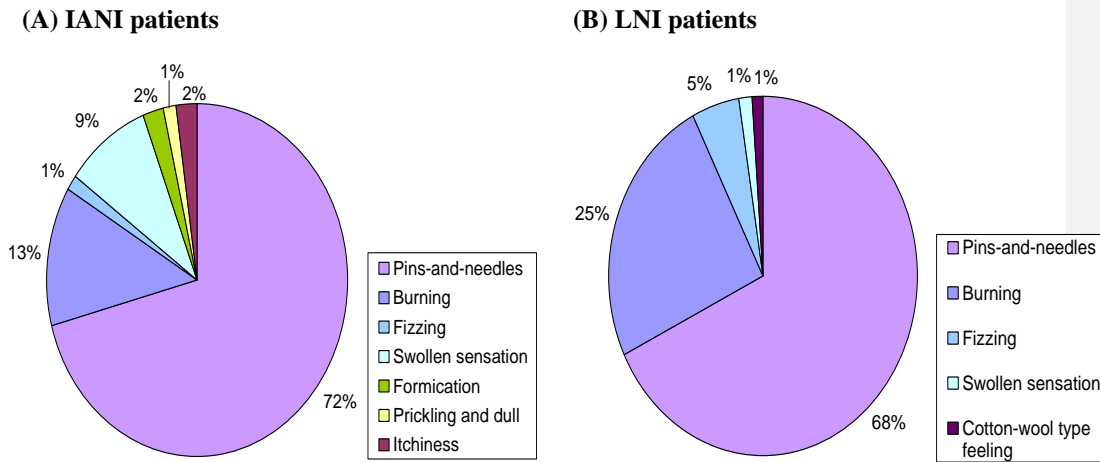

(B) LNI patients

Figure 4: Incidence of neuropathy amongst the IANI and LNI patients.

IANI and LNI patients mostly complained of dysaesthesia, followed by numbness and paraesthesia. Evoked paraesthesia was only seen amongst the IANI patients.
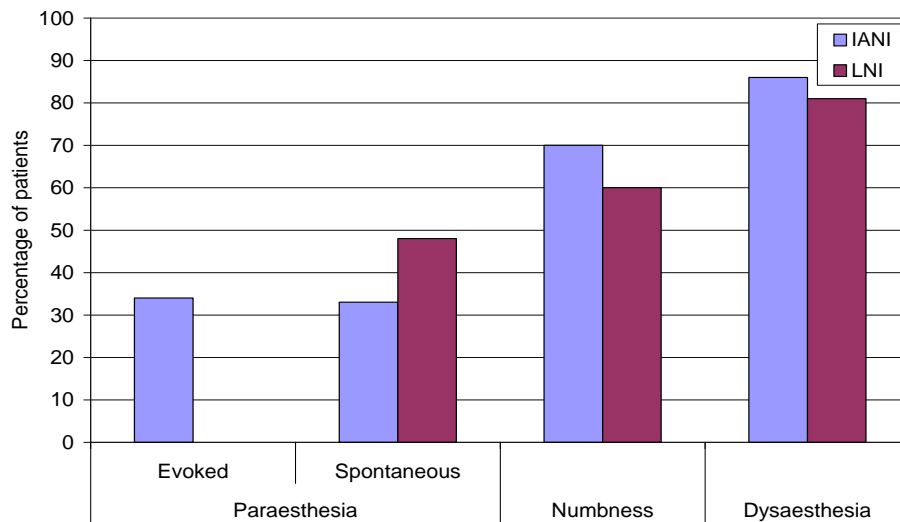
Figure 5: Presentation descriptors of neuropathy for (A) IANI and (B) LNI patients.
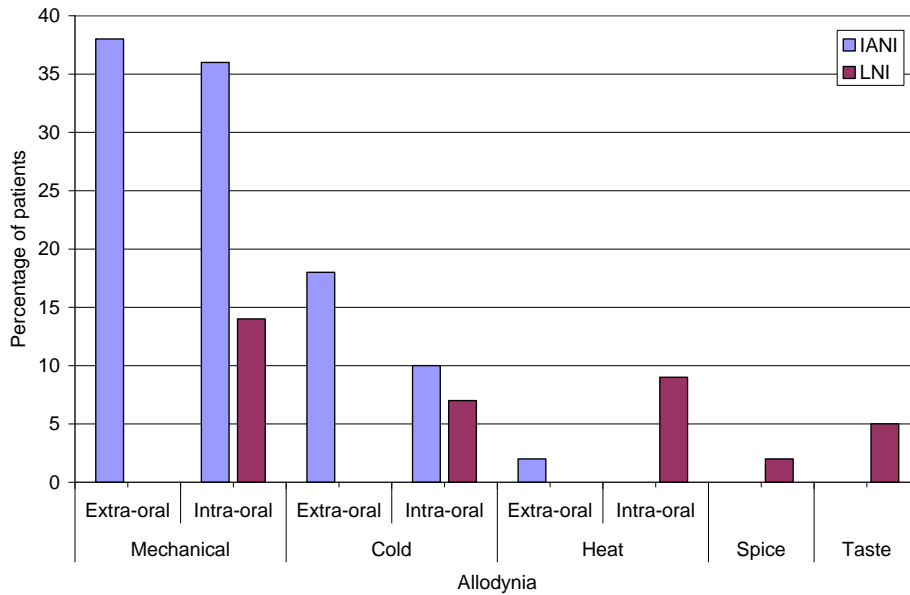
(A) IANI patients

(B) LNI patients

Figure 6: Incidence of pain amongst the IANI and LNI patients.

IANI patients tended to suffer more from evoked pain, allodynia and hyperalgesia than spontaneous pain. LNI patients, conversely, did not suffer as much from allodynia but had greater problems with evoked and spontaneous pain, and hyperalgesia.
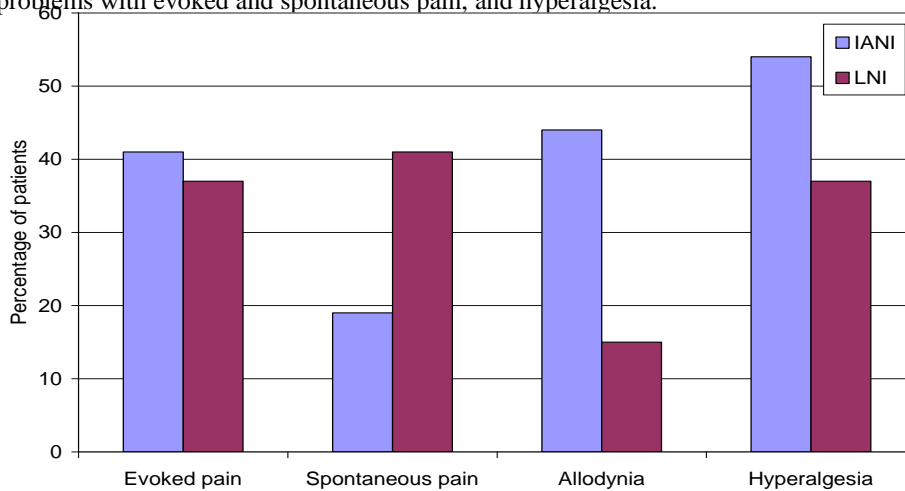
Figure 7: Incidence and types of allodynia experienced by the IANI and LNI patients.
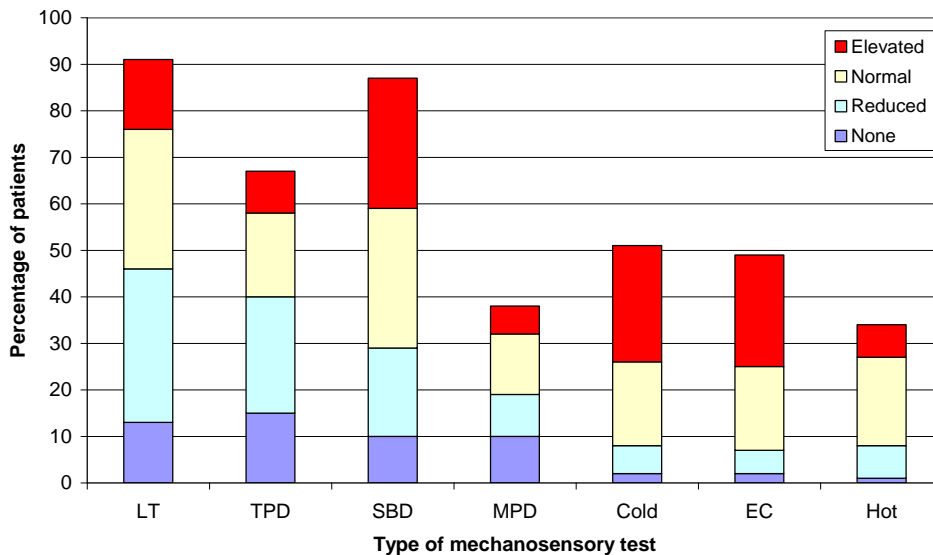

Figure 8: Mechanosensory results for (A) IANI and (B) LNI patients.

A**bbreviations: LT = Light touch; TPD = Two-point discrimination; SBD = Sharp-blunt discrimination; MPD = Moving point discrimination; EC = Ethyl chloride**

A greater percentage of IANI patients had elevated responses to TPD, SBD, MPD, cold and EC than the LNI patients, who were more likely to have decreased sensitivity to the mechanosensory tests.

(A) IANI patients

(B) LNI patients