
2020 GLOBAL YEAR FOR THE
PREVENTION OF PAIN
IASP®

Preventing V nerve injuries and resultant neuropathic pain

Faculty of Dentistry, Oral & Craniofacial Sciences

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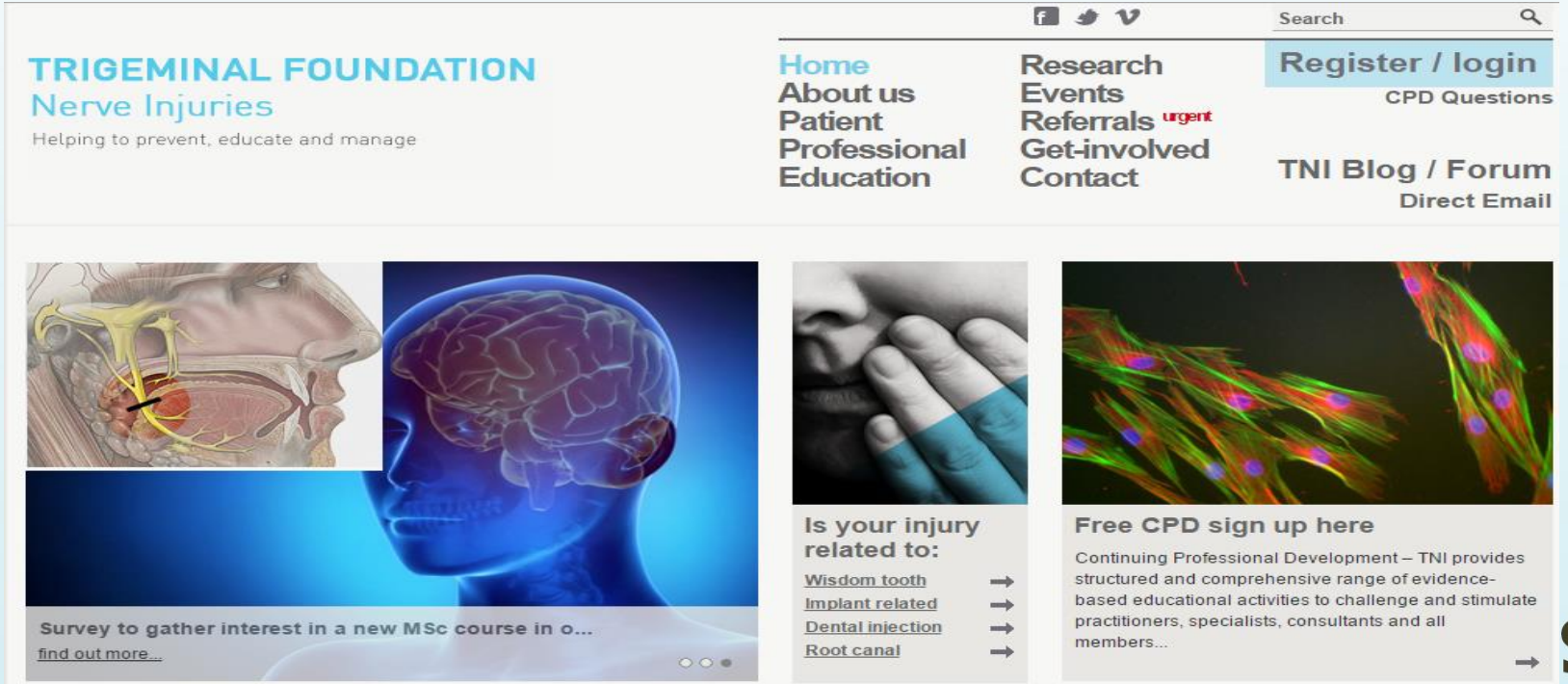
Trigeminalnerve.org.uk



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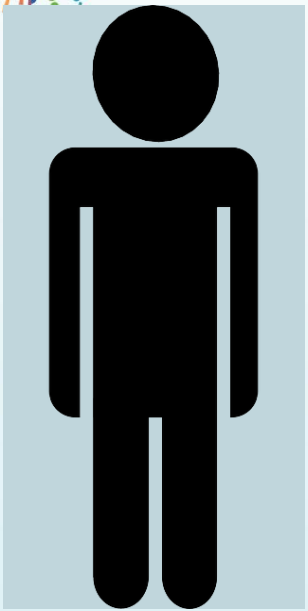
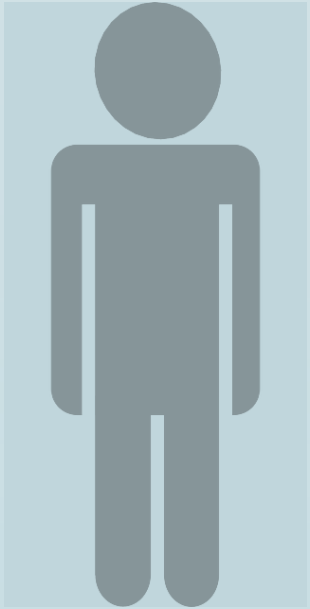
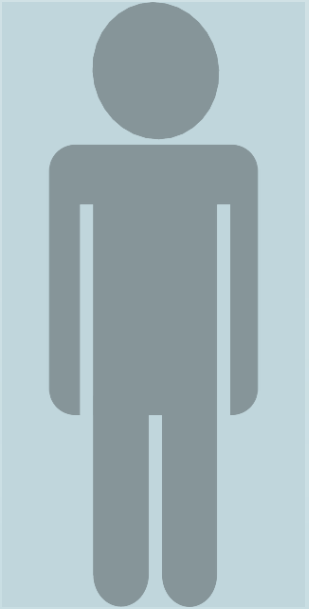
Overview



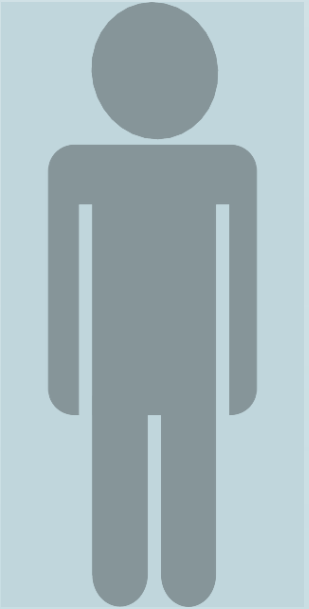
What is Post Traumatic Neuropathic pain PTNP?



Who gets PTNP?



Why prevent PTNP?



How to prevent these injuries?

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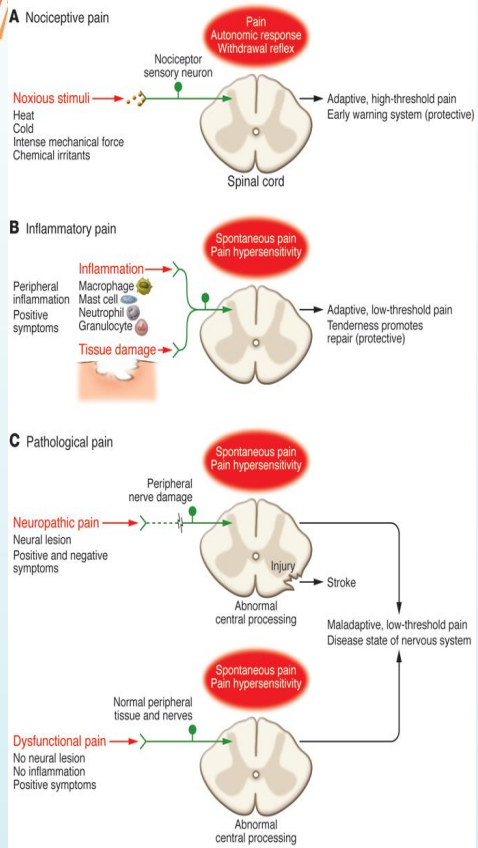


Types of pain
Healthy acute pain

NOCICEPTIVE PAIN

Nociceptive
healthy feeling pain 'pain'

Inflammatory pain
healthy short lived after insult

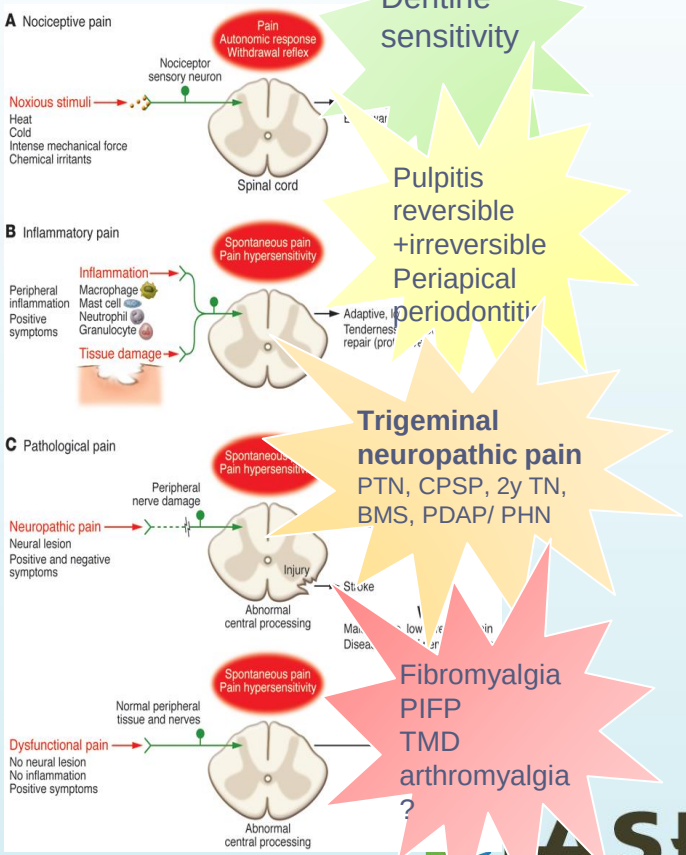
Chronic pain =
disease of neuromatrix

NEUROPATHIC PAIN

Neuropathic pain
Associated with nerve lesion

NOCIPLASTIC PAIN

Dysfunctional or centralised pain
Unknown cause



Dentine sensitivity

Pulpitis reversible +irreversible
Periapical periodontiti

Trigeminal neuropathic pain
PTN, CPSP, 2y TN, BMS, PDAP/ PHN

Fibromyalgia
PIFP
TMD
arthromyalgia
?

J Clin Invest. 2010 Nov 1; 120(11): 3742–3744. ***What is this thing called pain?*** *Clifford J. Woolf*

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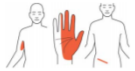
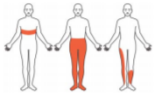
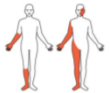
Neuropathic pain

- In 1994, the International Association for the Study of Pain (IASP) defined neuropathic pain as "pain initiated or caused by a primary lesion or dysfunction in the nervous system."
- In 2008, a task force initiated by the IASP Special Interest Group on Neuropathic Pain (NeuPSIG) noted the need to distinguish neuropathic pain from nociceptive pain arising indirectly from neurological disorders and pain conditions with secondary neuroplastic changes occurring in the nociceptive system, and proposed a new definition that omitted the term "dysfunction":
- "pain arising as a direct consequence of a lesion or disease affecting the somatosensory system."30
- A slightly modified version of this definition was proposed by the IASP Taxonomy Committee and accepted by the IASP: "**pain caused by a lesion or disease of the somatosensory nervous system."**

Table 1

Common neuropathic pain conditions and neuroanatomically plausible distribution of pain symptoms and sensory signs.

Neuropathic pain condition	Neuroanatomically plausible distribution of pain and sensory signs	Illustration of typical distribution
Trigeminal neuralgia	Within the facial or intraoral trigeminal territory.	
Postherpetic neuralgia	Unilateral distributed in one or more spinal dermatomes or the trigeminal ophthalmic division.	
Peripheral nerve injury pain	In the innervation territory of the lesioned nerve, typically distal to a trauma, surgery, or compression.	
Postamputation pain	In the missing body part and/or in the residual limb.	
Painful polyneuropathy	In feet, may extend to involve lower legs, thighs, and hands.	
Painful radiculopathy	Distribution consistent with the innervation territory of the nerve root.	
Neuropathic pain associated with spinal cord injury	At and/or below the level of the spinal cord lesion.	
Central poststroke pain	Contralateral to the stroke. In lateral medullary infarction, the distribution can also involve the ipsilateral side of the face.	
Central neuropathic pain associated with multiple sclerosis	Can be a combination of distributions seen in spinal cord injury and stroke.	

About 413 physicians completed a total of 3,956 patient records forms. Total annual direct health-care costs per patient ranged from €1,939 (Italy) to €3,131 (Spain).

Annual professional caregiver costs ranged from €393 (France) to €1,242 (UK), but this only represented a small proportion of total care because much care is provided by family or friends. Sick leave costs ranged from €5,492 (UK) to €7,098 (France), with 10%–32% patients prevented from working at some point by NP.

Total cost (including **direct and indirect costs**) of NP per patient was €10,313 in France (69% of the total cost), €14,446 in Germany (78%), €9,305 in Italy (69%), €10,597 in Spain (67%), and **€9,685 in the UK (57%).**

Indirect costs (ie, sick leave) constituted the majority of costs in all five countries: €7,098 in France, €11,232 in Germany, €6,382 in Italy, €7,066 in Spain, and **€5,492 in the UK**. In the subgroup analysis, total annual direct costs per patient were highest for neuropathic back pain and radiculopathy, and lowest for fibromyalgia.
Mean WPAI score range was 34.4–56.1; BPI interference was 4.1–4.8; and EQ-5D was 0.57–0.74. The results suggest that a significant proportion of the patient's work time in the previous week was affected by NP, and these are relatively high compared with other diseases such as diabetes, respiratory conditions, and arthritis.

The wider costs appear significantly higher to patients, carers/families, and society as a whole than to the health system alone.

ClinicoEconomics and Outcomes Research

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ORIGINAL RESEARCH

A burden of illness study for neuropathic pain in Europe

This article was published in the following Dove Press journal:
Clinico Economics and Outcomes Research
27 April 2016
Number of times this article has been viewed

Hiltrud Liedgens[1]
Marko Obradovic[1]
Jonathan De Courcy[2]
Timothy Holbrook[2]
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Purpose: Neuropathic pain (NP) is often severe and represents a major humanistic and economic burden. This study aimed at providing insight on this burden across France, Germany, Italy, Spain, and the UK, considering direct and indirect costs, productivity loss, and humanistic impact on patients and their families.

Methods: Physician questionnaires provided data on patients presenting with NP covering demographics, sick leave and retirement, number of consultations, drug treatments, and surgical procedures. Patients provided further demographic and disease-related data and completed the Work Productivity and Activity Impairment (WPAI), the EuroQol 5-Dimension (EQ-5D), and the Brief Pain Inventory (BPI) questionnaires. All health-related direct unitary costs were collected from relevant country-specific sources and adjusted to 2012 prices (€) where necessary. A subgroup analysis of costs based on diabetic peripheral neuropathy (n=894), fibromyalgia (n=300), and low back pain (n=963) was performed.

Findings: About 413 physicians completed a total of 3,956 patient records forms. Total annual direct health-care costs per patient ranged from €1,939 (Italy) to €3,131 (Spain). Annual professional caregiver costs ranged from €393 (France) to €1,242 (UK), but this only represented a small proportion of total care because much care is provided by family or friends. Sick leave costs ranged from €5,492 (UK) to €7,098 (France), with 10%–32% patients prevented from working at some point by NP. Total cost (including direct and indirect costs) of NP per patient was €10,313 in France (69% of the total cost), €14,446 in Germany (78%), €9,305 in Italy (69%), €10,597 in Spain (67%), and €9,685 in the UK (57%). Indirect costs (ie, sick leave) constituted the majority of costs in all five countries: €7,098 in France, €11,232 in Germany, €6,382 in Italy, €7,066 in Spain, and €5,492 in the UK. In the subgroup analysis, total annual direct costs per patient were highest for neuropathic back pain and radiculopathy, and lowest for fibromyalgia. Mean WPAI score range was 34.4–56.1; BPI interference was 4.1–4.8; and EQ-5D was 0.57–0.74. The results suggest that a significant proportion of the patient's work time in the previous week was affected by NP, and these are relatively high compared with other diseases such as diabetes, respiratory conditions, and arthritis.

Implications: Despite differences in practice between countries, these findings suggest a high opportunity cost for society in terms of lost work and productivity due to NP. The wider costs appear significantly higher to patients, carers/families, and society as a whole than to the health system alone.

Keywords: neuropathic pain, burden of illness, chronic lower back pain, produ

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Introduction

Chronic pain is a distinct and well-recognized condition experienced by
of the European adult population.[1] While the majority of chronic pain i

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http://dx.doi.org/10.2147/CEOR.S81396

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Pathophysiology

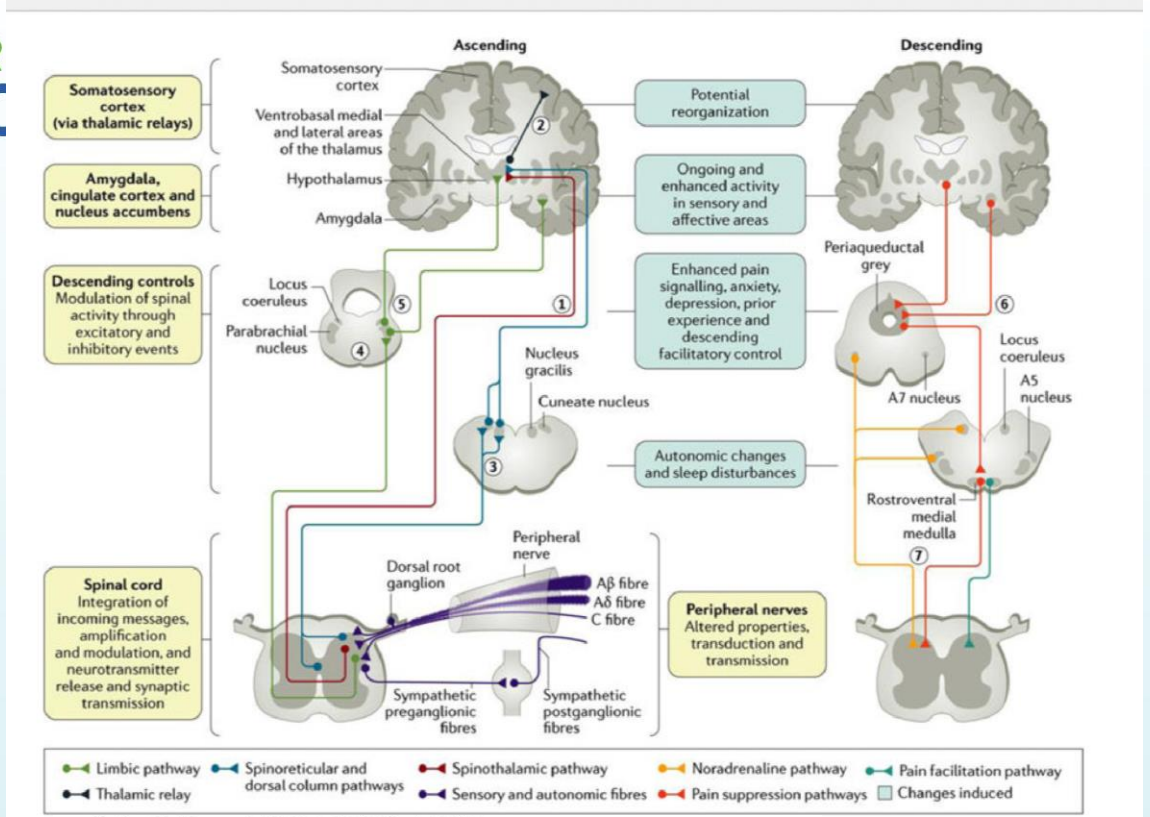

Figure 1. The peripheral and central changes induced by nerve injury or peripheral neuropathy Preclinical animal studies have shown that damage to all sensory peripheral fibres (namely, Aβ, Aδ and C fibres; BOX 1) alters transduction and transmission due to altered ion channel function. These alterations affect spinal cord activity, leading to an excess of excitation coupled with a loss of inhibition. In the ascending afferent pathways, the sensory components of pain are via the spinothalamic pathway to the ventrobasal medial and lateral areas (1), which then project to the somatosensory cortex allowing for the location and intensity of pain to be perceived (2). The spinal cord also has spinoreticular projections and the dorsal column pathway to the cuneate nucleus and nucleus gracilis (3). Other limbic projections relay in the parabrachial nucleus (4) before contacting the hypothalamus and amygdala, where central autonomic function, fear and «anxiety are altered (5). Descending efferent pathways from the amygdala and hypothalamus (6) drive the periaqueductal grey, the locus coeruleus, A5 and A7 nuclei and the rostroventral medial medulla. These brainstem areas then project to the spinal cord through descending noradrenaline (inhibition via α2 adrenoceptors), and, in neuropathy, there is a loss of this control and increased serotonin descending excitation via 5-HT3 receptors (7). The changes induced by peripheral neuropathy on peripheral and central functions are shown. Adapted with permission from REF. 38, Mechanisms and management of diabetic painful distal symmetrical polyneuropathy, American Diabetes Association, 2013. Copyright and all rights reserved. Material from this publication has been used with the permission of American Diabetes Association.

Nat Rev Dis Primers. ; 3: 17002. doi:10.1038/nrdp.2017.2.

Neuropathic pain

Luana Colloca[1], **Taylor Ludman**[1], **Didier Bouhassira**[2], **Ralf Baron**[3], **Anthony H. Dickenson**[4], **David Yarnitsky**[5], **Roy Freeman**[6], **Andrea Truini**[7], **Nadine Attal**[8], **Nanna B. Finnerup**[9], **Christopher Eccleston**[10,11], **Eija Kalso**[12], **David L. Bennett**[13], **Robert H. Dworkin**[14], and **Srinivasa N. Raja**[15]

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Definitions – do not confuse nomenclature!

- **Neuralgia –** nerve pain
- **Neuropathic pain (IASP)**
  Pain caused by a lesion or disease of the somatosensory nervous system.
- **Neuropathy (IASP)**
  A disturbance of function or pathological change in a nerve: in one nerve, mononeuropathy; in several nerves, mononeuropathy multiplex; if diffuse and bilateral, polyneuropathy.
- *Note:* **Neuritis** (q.v.) is a special case of neuropathy and is now reserved for inflammatory processes affecting nerves.
  - sensory (touch, heat, pain)
  - motor (movement)

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Chronic post surgical pain (CPSP) or NeP?

Haroutiunian S, Nikolajsen L, Finnerup NB, Jensen TS. The neuropathic component in persistent postsurgical pain: a systematic literature review. Pain. 2013 Jan;154(1):95-102. doi: 10.1016/j.pain.2012.09.010.

Persistent postsurgical pain (PPSP) is a frequent and often disabling complication of many surgical procedures.

Nerve injury-induced neuropathic pain (NeuP) has repeatedly been proposed as a major cause of PPSP. However, there is a lack of uniformity in NeuP assessment across studies, and the prevalence of NeuP may differ after various surgeries.

We performed a systematic search of the PubMed, CENTRAL, and Embase databases and assessed 281 studies that investigated PPSP after 11 types of surgery.

The prevalence of PPSP in each surgical group was examined. The prevalence of NeuP was determined by applying the recently published NeuP probability grading system. The prevalence of probable or definite NeuP was high in patients with persistent pain after thoracic and breast surgeries-66% and 68%, respectively. In patients with PPSP after groin hernia repair, the *prevalence of NeuP was 31%,* and after total hip or knee arthroplasty it was 6%.

The results suggest that the prevalence of NeuP among PPSP cases differs in various types of surgery, probably depending on the likelihood of surgical iatrogenic nerve injury. Because of large methodological variability across studies, a more uniform approach is desirable in future studies for evaluating persistent postsurgical NeuP.

	Estimated incidence of chronic pain	Estimated chronic severe (disabling) pain (>5 out of score of 10)	US surgical volumes (1000s)†
Amputation[2]	30–50%	5–10%	159 (lower limb only)
Breast surgery (lumpectomy and mastectomy)[3]	20–30%	5–10%	479
Thoracotomy[4–7]	30–40%	10%	Unknown
Inguinal hernia repair[8–10]	10%	2–4%	609
Coronary artery bypass surgery[11–13]	30–50%	5–10%	598
Caesarean section[14]	10%	4%	220

*Gall bladder surgery not included, since preoperative diagnosis of pain specifically from gall bladder is difficult and persistent postoperative pain could therefore be related to other intra-abdominal disorders. †National Center For Health Statistics, Ambulatory and Inpatients Procedures, USA, 1996.

Table 1: **Estimated incidence of chronic postoperative pain and disability after selected surgical procedures***

30% get persistent pain 10% are <u>severely</u> affected
Very few related to dentistry likely due to LA

Kehlet H *et al*, 2006 Lancet

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Table 3. Core Diagnostic Criteria for Persistent Posttraumatic Neuropathic Pain

1. History of traumatic nerve injury or surgery associated with known risk of nerve injury.*
2. Pain lasting ≥3 mo, with onset showing a temporal relation to known nerve injury (onset within days to weeks after the injury).†
3. Positive and/or negative signs of sensory disturbance in the innervation of the injured nerve as evidenced by ≥1 of the following:
   a. Mixed areas of hypo- and hypersensitivity to various sensory modalities
   b. Hyposensitivity to nonpainful warmth (with or without changes in cold sensation)
   c. Hypersensitivity to brush or pinprick in or around the painful area
4. No other condition (eg, inflammation, tumor) better explains the pattern of the clinical features (eg, radiculopathy) that could plausibly account for persisting pain in the affected dermatome or dermatomes.

*This pain may occur even if there was a deliberate attempt to spare the large nerves crossing the surgical area (eg, in breast surgery).
†There is a spontaneous decline in reporting of pain >12 mo after surgery/trauma. Relevant citations in support of these diagnostic criteria are Bruehl,[34] Duffy et al,[77] Guo et al,[107] Haldar et al,[109] Pappagallo et al,[187] Teerijoki-Oksa

American Pain Society RESEARCH EDUCATION TREATMENT ADVOCACY
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Available online at www.jpain.org and www.sciencedirect.com

Focus Article

AAPT Diagnostic Criteria for Peripheral Neuropathic Pain: Focal and Segmental Disorders

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†Department of Anesthesiology, Brigham & Women's Hospital, Harvard University School of Medicine, Boston, MA
‡University of Kiel, Division of Neurological Pain Research and Therapy, Department of Neurology, Kiel, Germany
§Department of Anesthesiology, Vanderbilt University Medical Center, Nashville, TN
¶Department Human Neuroscience, Sapienza University, Rome, Italy
‖Department of Anesthesiology and Perioperative Medicine, University of Rochester School of Medicine and Dentistry, Rochester, NY
**Department of Anesthesiology and Washington University Pain Center, Washington University School of Medicine, St Louis, MO

Abstract: Peripheral neuropathic pain is among the most prevalent types of neuropathic pain.

Published in final edited form as:
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The IASP classification of chronic pain for ICD-11: chronic neuropathic pain

Joachim Scholz[a], Nanna B. Finnerup[b,c], Nadine Attal[d], Qasim Aziz[e], Ralf Baron[f], Michael I. Bennett[g], Rafael Benoliel[h], Milton Cohen[i], Giorgio Cruccu[j], Karen D. Davis[k], Stefan Evers[l], Michael First[m], Maria Adele Giamberardino[n], Per Hansson[o], Stein Kaasa[p], Beatrice Korwisi[q], Eva Kosek[r], Patricia Lavand'homme[s], Michael Nicholas[t], Turo Nurmikko[u], Serge Perrot[v], Srinivasa N. Raja[w], Andrew S. C. Rice[x], Michael C. Rowbotham[y], Stephan Schug[z], David M. Simpson[aa], Blair H. Smith[ab], Peter Svensson[ac], Johan W.S. Vlaeyen[ad], Shuu-Jiun Wang[ae], Antonia Barke[q], Winfried Rief[q], Rolf-Detlef Treede[af], Classification Committee of the Neuropathic Pain Special Interest Group (NeuPSIG), and **Task Force for the Classification of Chronic Pain of the International Association for the Study of Pain (IASP)**

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   a. Mixed areas of hypo- and hypersensitivity to various sensory modalities Neuropathic area
   b. Hyposensitivity to nonpainful warmth (with or without changes in cold sensation) Allodynia / Hyperalgesia = hyperaesthesia
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*This pain may occur even if there was a deliberate attempt to spare the large nerves crossing the surgical area (eg, in breast surgery).
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†Department of Anesthesiology, Brigham & Women's Hospital, Harvard University School of Medicine, Boston, MA
‡University of Kiel, Division of Neurological Pain Research and Therapy, Department of Neurology, Kiel, Germany
§Department of Anesthesiology, Vanderbilt University Medical Center, Nashville, TN
¶Department Human Neuroscience, Sapienza University, Rome, Italy
‖Department of Anesthesiology and Perioperative Medicine, University of Rochester School of Medicine and Dentistry, Rochester, NY
**Department of Anesthesiology and Washington University Pain Center, Washington University School of Medicine, St Louis, MO

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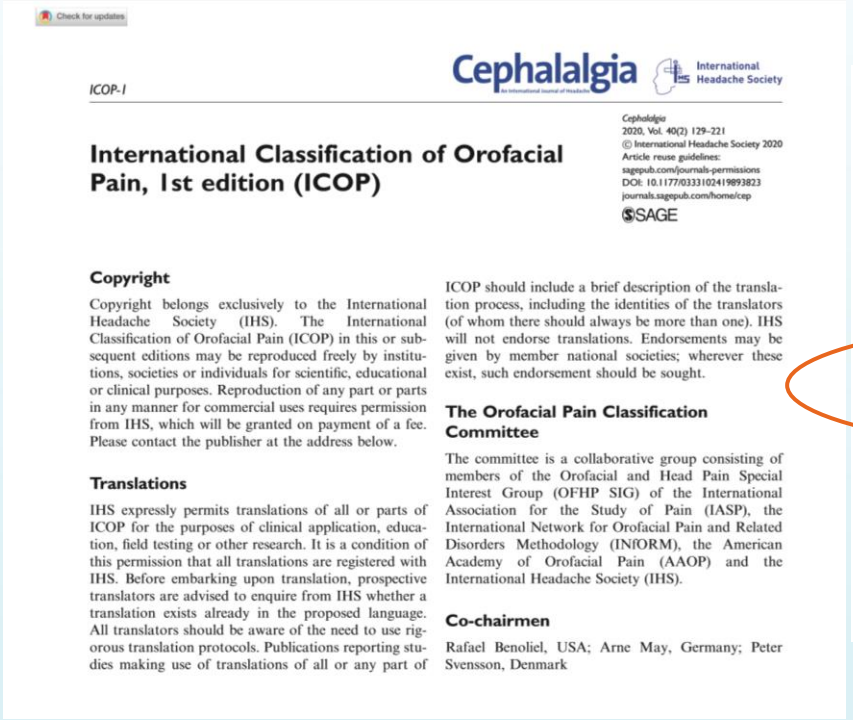
ICOP Definitions and Diagnostic Criteria PTNP



Check for updates

ICOP-I

Cephalalgia

International Headache Society

Cephalalgia
2020, Vol. 40(2) 129–221

DOI: 10.1177/0333102419893823
journals.sagepub.com/home/cep
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International Classification of Orofacial Pain, 1st edition (ICOP)

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The Orofacial Pain Classification Committee

The committee is a collaborative group consisting of members of the Orofacial and Head Pain Special Interest Group (OFHP SIG) of the International Association for the Study of Pain (IASP), the International Network for Orofacial Pain and Related Disorders Methodology (INfORM), the American Academy of Orofacial Pain (AAOP) and the International Headache Society (IHS).

Co-chairmen

Rafael Benoliel, USA; Arne May, Germany; Peter Svensson, Denmark

1. Orofacial pain attributed to disorders of dentoalveolar and anatomically related structures
2. Myofascial orofacial pain
3. Temporomandibular joint (TMJ) pain
4. Orofacial pain attributed to lesion or disease of the cranial nerves
5. Orofacial pains resembling presentations of primary headaches
6. Idiopathic orofacial pain

ICOP 2020

Orofacial pain attributed to lesion or disease of the cranial nerves Lene Baad-Hansen, Denmark (chairman); Eli Eliav, USA;

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Post Traumatic neuropathic pain PTNP (ICOP)

4.1.2.3 Post-traumatic trigeminal neuropathic pain

- Previously used terms: Anaesthesia dolorosa; painful post-traumatic trigeminal neuropathy.
- Description: Unilateral or bilateral facial or oral pain following and caused by trauma to the trigeminal nerve(s), with other symptoms and/or clinical signs of trigeminal nerve dysfunction, and persisting or recurring for more than 3 months.
- *4.1.2.3.1 Probable post-traumatic trigeminal neuropathic pain*
- *Diagnostic criterion: A. Pain fulfilling all but criterion B2 for 4.1.2.3 Posttraumatic trigeminal neuropathic pain.*

4.1.2.4 Trigeminal neuropathic pain attributed to other disorder

4.1.2.5 Idiopathic trigeminal neuropathic pain

Description: Unilateral or bilateral facial pain in the distribution(s) of one or more branches of the trigeminal nerve

Diagnostic criteria:

A. Pain, in a neuroanatomically plausible area within the distribution(s) of one or both trigeminal nerve(s), persisting or recurring for >3 months and fulfilling criteria C and D

B. Both of the following:

1. history of a mechanical, thermal, radiation or chemical injury to the peripheral trigeminal nerve(s)
2. diagnostic test confirmation1 of a lesion of the peripheral trigeminal nerve(s) explaining the pain2

C. Onset within 6 months after the injury

D. Associated with somatosensory symptoms and/or signs4 in the same neuroanatomically plausible distribution

E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

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Grading of neuropathic pain

2020 GLOBAL YEAR FOR THE PREVENTION OF PAIN

Comprehensive Review

PAIN

OPEN

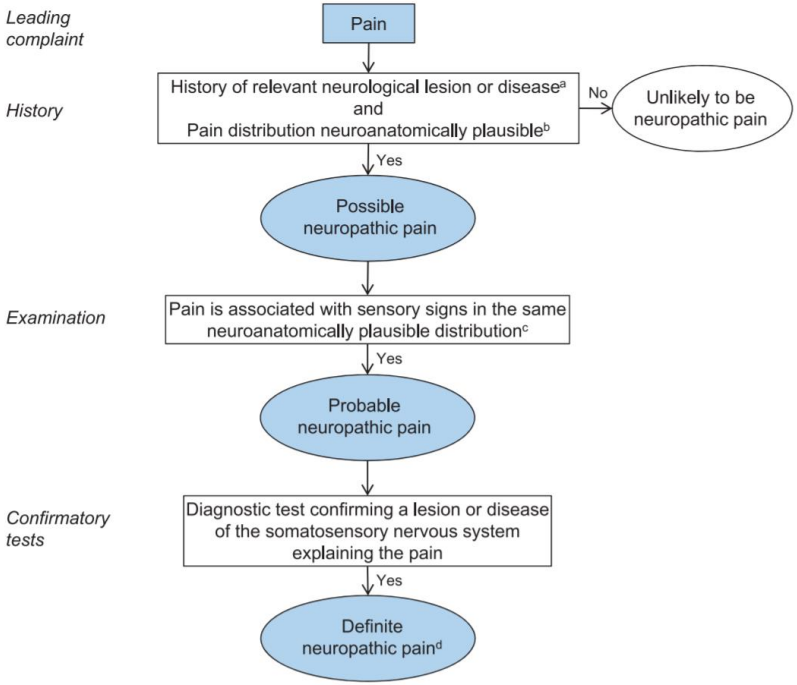
Neuropathic pain: an updated grading system for research and clinical practice

Nanna B. Finnerup[a,*], Simon Haroutounian[b], Peter Kamerman[c], Ralf Baron[d], David L.H. Bennett[e], Didier Bouhassira[f,g], Giorgio Cruccu[h], Roy Freeman[i], Per Hansson[j,k], Turo Nurmikko[l], Srinivasa N. Raja[m], Andrew S.C. Rice[n,o], Jordi Serra[p], Blair H. Smith[q], Rolf-Detlef Treede[r], Troels S. Jensen[a,s]

Abstract

The redefinition of neuropathic pain as "pain arising as a direct consequence of a lesion or disease affecting the somatosensory system," which was suggested by the International Association for the Study of Pain (IASP) Special Interest Group on Neuropathic Pain (NeuPSIG) in 2008, has been widely accepted. In contrast, the proposed grading system of possible, probable, and definite neuropathic pain from 2008 has been used to a lesser extent. Here, we report a citation analysis of the original NeuPSIG grading paper of 2008, followed by an analysis of its use by an expert panel and recommendations for an improved grading system. As of February, 2015, 608 eligible articles in Scopus cited the paper, 414 of which cited the neuropathic pain definition. Of 220 clinical studies citing the paper, 56 had used the grading system. The percentage using the grading system increased from 5% in 2009 to 30% in 2014. Obstacles to a wider use of the grading system were identified, including (1) questions about the relative significance of confirmatory tests, (2) the role of screening tools, and (3) uncertainties about what is considered a neuroanatomically plausible pain distribution. Here, we present a revised grading system with an adjusted order, better reflecting clinical practice, improvements in the specifications, and a word of caution that even the "definite" level of neuropathic pain does not always indicate causality. In addition, we add a table illustrating the area of pain and sensory abnormalities in common neuropathic pain conditions and propose areas for further research.

Keywords: Neuropathic pain, Definition, Grading, Possible, Probable, Definite



Compared to the grading system published in 2008, we have (1) changed the order of the grading criteria to better reflect clinical practice. (2) annotated the terms used to improve clarity. (3) recognized the role of screening tools (questionnaires) in neuropathic pain evaluation. (4) emphasized that reaching the final level of certainty (definite neuropathic pain) confirms clinically that a lesion or disease of the somatosensory nervous system can explain the pain but, as often in neurology, it does not establish causality (ie, there may still be other causes of the pain such as a diabetic ulcer). The main purpose of the grading system is to help in the classification of the pain as neuropathic.

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Exclude non-traumatic Neuropathic pain

Identified cause Neuropathic

V (TN),IX,VII classic neuralgias- TN classical

PDAP II

Ne pain/PTN (CPSP) metabolic, infection, MS, neoplasia, vascular autoimmune)

Nutritional deficiencies

Fe, Ferritin, Zinc, Magnesium,
Vit B complex, D, E

Malignancy

Compression by a space occupying lesion centrally or peripherally NEOPLASIA

Metabolic Acromegaly, Hormonal neuropathy (Hypothyroidism, Diabetes),

Infarction (sickle cell hypoxic neural damage, giant cell arteritis)

Demyelination (Multiple sclerosis)

Infection Post viral neuropathy, Bacterial, Leprosy

Toxic Heavy metal poisoning (lead, mercury) radiation, thermal, chemotherapy, drugs

Auto immune problems: Lupus, Rheumatoid disease

Sarcoidosis and amyloidosis

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Any spontaneous neuropathy think Red flags of malignancy

- Over 50 years
- Previous history of Carcinoma
- Smoking /alcohol/ Betel nut/ Pan
- Night fevers
- Weight loss
- Blood loss/ aneamia

NHS 2 (NICE 3) weeks Referral pathway

- Recent onset
- Rapid growth
- Neuropathy - sensory or motor
- Resorption of adjacent structures
- Localised mobility of teeth
- Progressive trismus
- Persistent painless ulcer
- Lymphadenopathy painless persistent
- Lack of response to conventional treatments:
  - Antibiotics
  - Endodontic surgery

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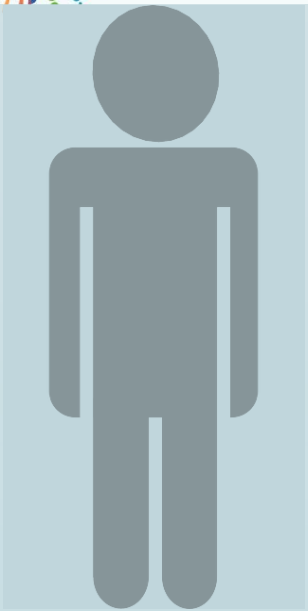
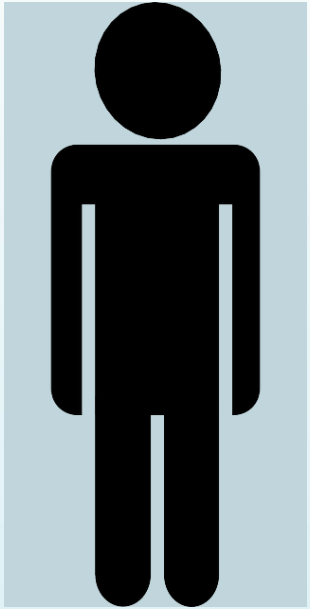
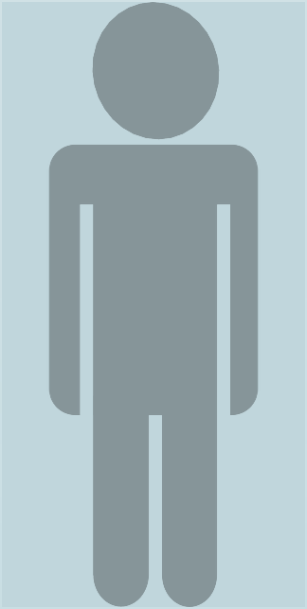
Overview



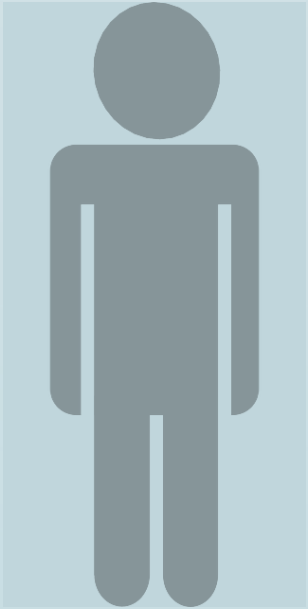
What is Post Traumatic Neuropathic pain PTNP?



Who gets PTNP?



Why prevent PTNP?



How to prevent these injuries?

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When pain gets stuck: the evolution of pain chronification and treatment resistance

David Borsook[1,2], **Andrew M Youssef**[1], **Laura Simons**[3], **Igor Elman**[4], and **Christophe Eccleston**[5,6]

[1]Center for Pain and the Brain, Boston Children's (BCH), McLean and Massachusetts Ho

(M

[2]D MGH)

[3]D

[4]V

[5]C

[6]D

REVIEW FOCUS ON PAIN

nature neuroscience

Pain vulnerability: a neurobiological perspective

Franziska Denk[1], Stephen B McMahon[1] & Irene Tracey[2]

There are many known risk factors for chronic pain conditions, yet the biological underpinnings that link these factors to abnormal processing of painful signals are only just beginning to be explored. This Review will discuss the potential mechanisms that have been proposed to underlie vulnerability and resilience toward developing chronic pain. Particular focus will be given to genetic and epigenetic processes, priming effects on a cellular level, and alterations in brain networks concerned with reward, motivation/learning and descending modulatory control. Although research in this area is still in its infancy, a better understanding of how pain vulnerability emerges has the potential to help identify individuals at risk and may open up new therapeutic avenues.

Considerable advances have been made in understanding the neurobiology of chronic pain over the last two decades. The molecular mechanisms leading to amplification of pain-related signals in chronic pain states have been dissected. An unexpected contribution of non-neuronal cells in the CNS has been discovered, and functional, as well as structural, neuroimaging studies have revealed a brain organization

likely to develop certain chronic pain conditions, as are older people, although age may function as a protective factor in some instances. The influence of genetics is supported by twin and population-based studies, which clearly indicate that painful conditions and acute pain sensitivity *per se* are heritable (see ref. 5 for a recent review). Other

COMMENTARY

Check for updates

Pain chronification: what should a non-pain medicine specialist know?

Bart Morlion[a], Flaminia Coluzzi[b], Dominic Aldington[c], Magdalena Kocot-Kepska[d], Joseph Pergolizzi[e], Ana Cristina Mangas[f], Karsten Ahlbeck[g] and Eija Kalso[h]

[a]Leuven Centre for Algology & Pain Management, University Hospitals Leuven, KU Leuven, Belgium; [b]Department of Medical and Surgical Sciences and Biotechnologies Unit of Anaesthesia, Intensive Care and Pain Medicine, Sapienza University of Rome, Rome, Italy; [c]Royal Hampshire County Hospital, Winchester, UK; [d]Department of Pain Research and Treatment, Jagiellonian University Medical College, Kraków, Poland; [e]Global Pain Initiative, Golden, CO, USA and Naples Anesthesia and Pain Associates, Naples, FL, USA; [f]Hospital de Santo André, Leiria, Portugal; [g]Capio St Görans Hospital, Stockholm, Sweden; [h]Pain Clinic, Departments of Anaesthesiology, Intensive Care, and Pain Medicine, Helsinki University Central Hospital, Helsinki, Finland

ABSTRACT

Objective: Pain is one of the most common reasons for an individual to consult their primary care physician, with most chronic pain being treated in the primary care setting. However, many primary care physicians/non-pain medicine specialists lack enough awareness, education and skills to manage pain patients appropriately, and there is currently no clear, common consensus/formal definition of "pain chronification".

ARTICLE HISTORY

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KEYWORDS

Chronic pain; chronification; pain; non-pain medicine specialist

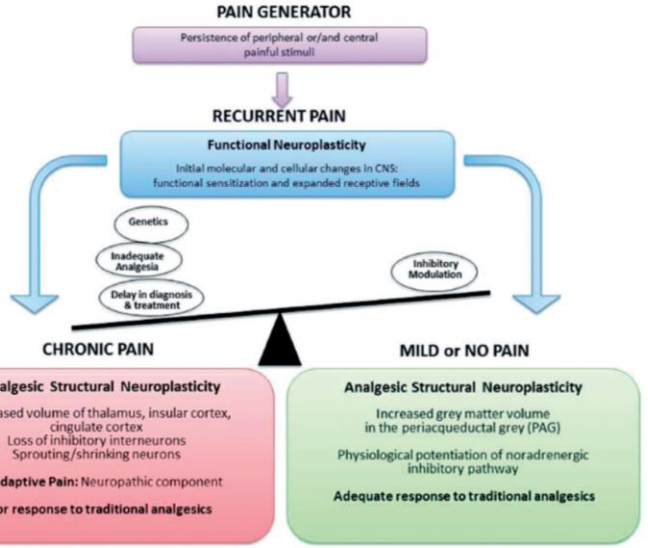

Figure 1. From the physiological perspective, an imbalance between enhanced ascending nociceptive inputs and inadequate inhibitory descending pathways is responsible for pain chronification[45]. Reproduced with permission from Coluzzi *et al.*[45]

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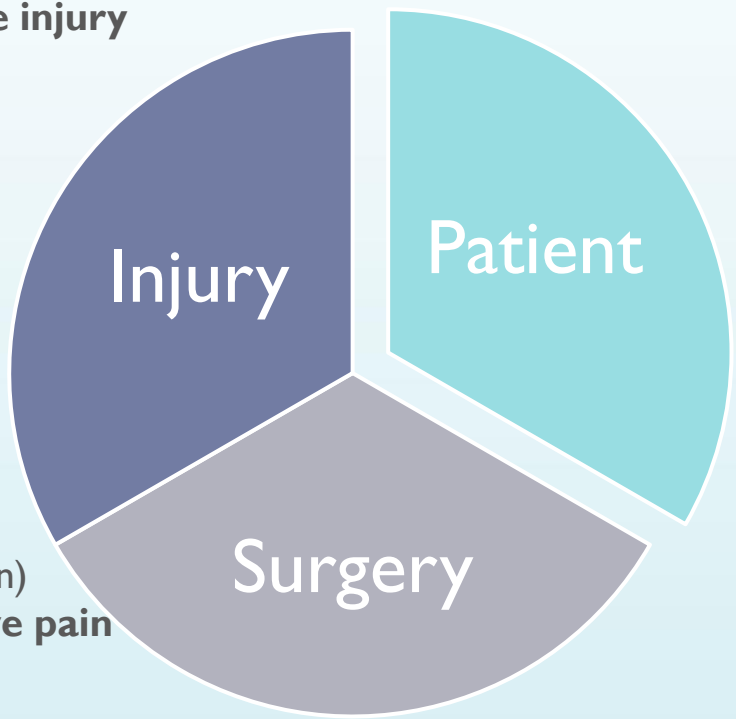
Summary risk factors for PTPN/chronic post surgical pain

Resultant sensory nerve injury
Large neuropathic area
Thermal allodynia
Mechanical allodynia
Hyperalgesia



Age > 50 yrs
Female
Multiple pain conditions
Social Factors

Axis II Psychological factors
Mood anxiety / depression
Introversion, neuroticism, hypervigilance, catastrophising
Fear of surgery
Fear of pain

Poor pain modulation DNIC positive tests
Genetics
COMPT CA channels
Epigenetics
Prior abuse and neglect
OMICS ????

Surgical factors
Type of surgery
Site
Minimise nerve injury
(Tissue tension & Duration)
High level perioperative pain
(Lack of local anaesthesia)

Joel Katz & Ze'ev Seltzer Transition from acute to chronic postsurgical pain: risk factors and protective factors. Expert Review of Neurotherapeutics Volume 9, 2009 - Issue 5

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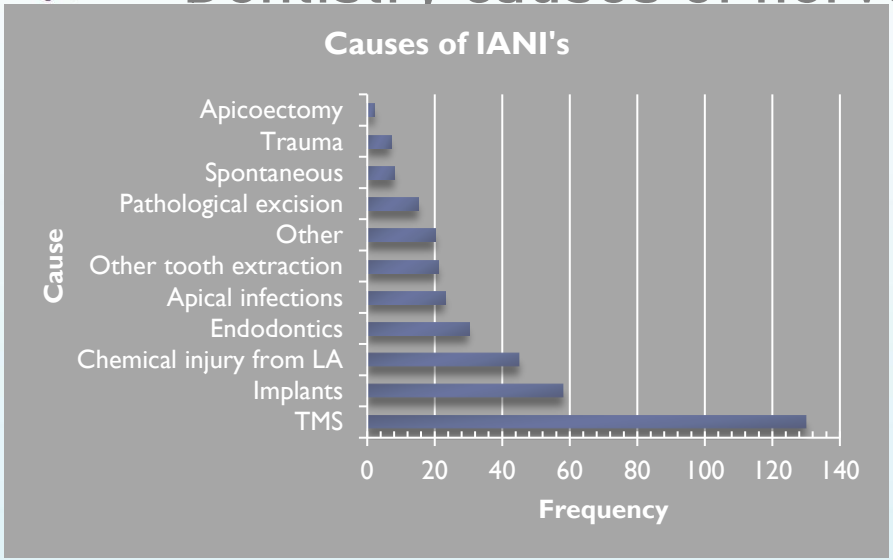
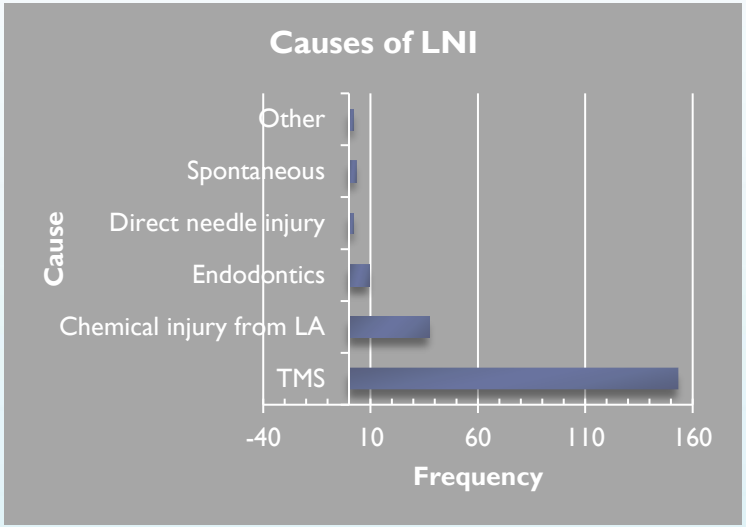
Dentistry causes of nerve injuries + neuropathic pain





- **Summary of nerve injury patients** March 2008 –2016
- 400 IANI patients (73% F: 26.8% M; mean age = 46.5 years [range 18 – 85])
- 214 LNI patients (64.5% F: 34.6% M; mean age = 38.6 years [range 20 -73])

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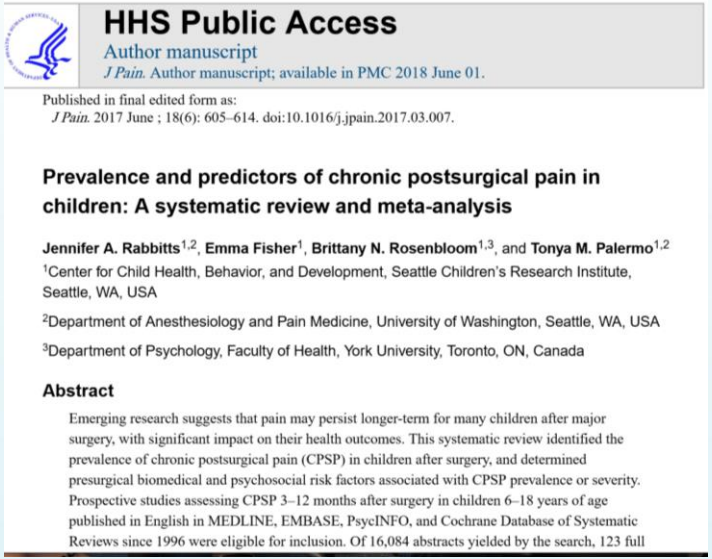
Predictive patient factors

- **Presurgical pain intensity, child anxiety, child pain coping efficacy, and parental pain catastrophizing** were the only presurgical factors identified as predictive of CPSP. Biological and medical factors assessed were not associated with CPSP in any study. Well-designed studies examining prevalence and predictors of CPSP are critically needed in children.
- The biopsychosocial model of pain is central to our understanding of factors involved in the development and maintenance of CPSP.
- **Several presurgical risk factors for CPSP have been consistently identified in adults undergoing surgery, including biological factors (older age, female sex), medical factors (greater presurgical pain), and psychosocial factors (higher levels of presurgical anxiety and pain catastrophizing)7–10.**



HHS Public Access

Author manuscript

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Prevalence and predictors of chronic postsurgical pain in children: A systematic review and meta-analysis

Jennifer A. Rabbitts[1,2], **Emma Fisher**[1], **Brittany N. Rosenbloom**[1,3], and **Tonya M. Palermo**[1,2]

[1]Center for Child Health, Behavior, and Development, Seattle Children's Research Institute, Seattle, WA, USA

[2]Department of Anesthesiology and Pain Medicine, University of Washington, Seattle, WA, USA

[3]Department of Psychology, Faculty of Health, York University, Toronto, ON, Canada

Abstract

Emerging research suggests that pain may persist longer-term for many children after major surgery, with significant impact on their health outcomes. This systematic review identified the prevalence of chronic postsurgical pain (CPSP) in children after surgery, and determined presurgical biomedical and psychosocial risk factors associated with CPSP prevalence or severity. Prospective studies assessing CPSP 3–12 months after surgery in children 6–18 years of age published in English in MEDLINE, EMBASE, PsycINFO, and Cochrane Database of Systematic Reviews since 1996 were eligible for inclusion. Of 16,084 abstracts yielded by the search, 123 full

Hinrichs-Rocker A, Schulz K, Jarvinen I, Lefering R, Simanski C, Neugebauer EA. Psychosocial predictors and correlates for chronic post-surgical pain (CPSP) - a systematic review. Eur J Pain. 2009; 13:719–30. [PubMed: 18952472] 8. Katz J, Seltzer Z. Transition from acute to chronic postsurgical pain: risk factors and protective factors. Expert Rev Neurother. 2009; 9:723–44. [PubMed: 19402781] 9. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006; 367:1618–25. [PubMed: 16698416] 10. Kehlet, H., Edwards, RR., Brennan, T. Persistent Postsurgical Pain: Pathogenic Mechanisms and Preventive Strategies, Pain 2014. In: Srinivasa, R., Sommer, C., editors. Refresher Courses, 15th World Congress of Pain. Washington, D.C: IASP Press; 2014.

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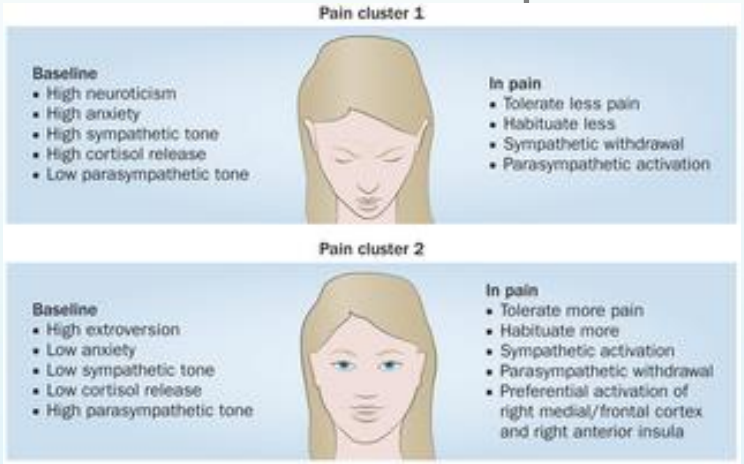
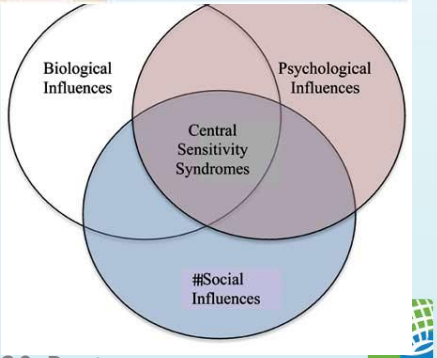
Psychosocial risk factors predictive of CPSP

- Cognitive
  - Fear of surgery and anxiety
  - Fear of pain
- Personality disorder
  - increased preoperative anxiety
  - Introverted personality
  - Catastrophizing
  - Poor coping skills
  - Hypervigilance state
- Psychological vulnerability – pain related fear
- Social support
- Solicitous responding
  - Empathetic spouse encouraging negative behaviour
  - Munchausen





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Type of patient

Nociception

Sensation

Behaviour

Suffering

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Type of patient

W

I

M

P

S

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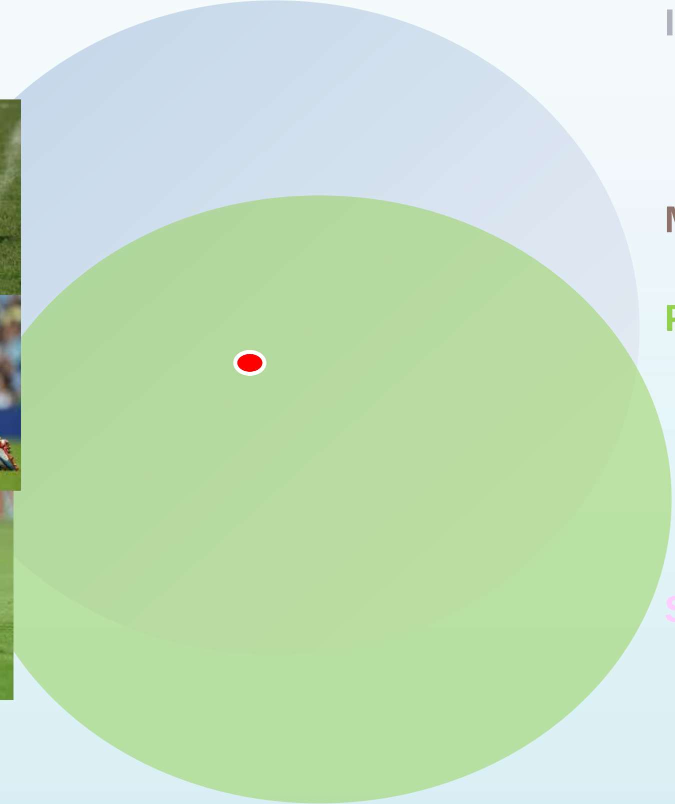
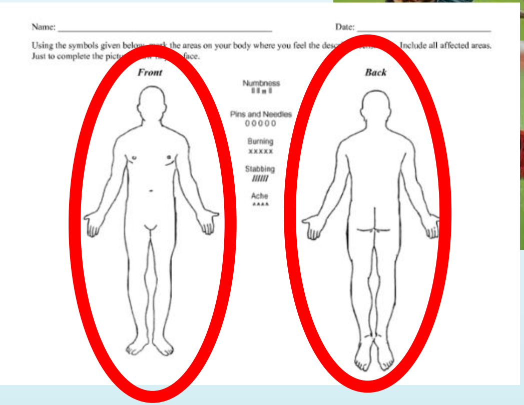
Type of patient

WW	**Women**
	GWAS
I I	**Injury- PTSD**
	Inhibition is poor with low pain modulation
M	**Mood disorders Anxiety & Stress**
PP	**Personality disorders**
	introspective, catastrophiser and hypervigilance
	Prior abuse and neglect
S	**Sleep deprivation Stress**




Name: ______ Date: ______
Using the symbols given below, mark the areas on your body where you feel the desc... Include all affected areas.
Just to complete the pict... face.
Front
Back
Numbness
Pins and Needles
Burning
Stabbing
Ache

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Determinants for onset and maintenance of chronic pain=AXIS

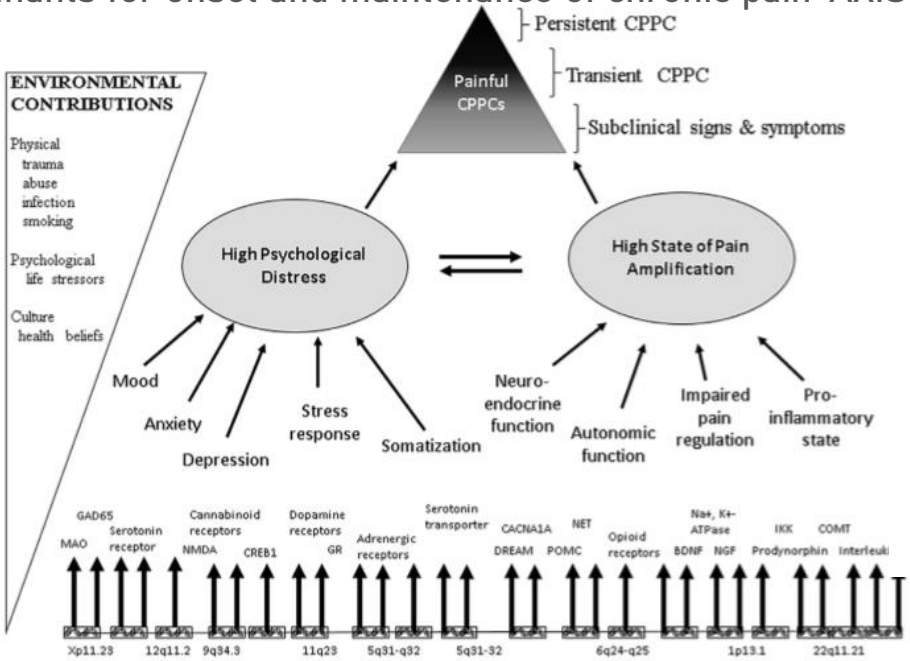

Figure 4. This model depicts likely determinants that contribute to the risk of onset and maintenance of common chronic overlapping pain conditions (COPCs). These factors are determined by genetic variability and environmental events that determine an individual's psychological profile and pain amplification status. These 2 primary domains are interactive and influence the risk of pain onset and persistence. Likely modifiers of the interaction between genetic and environmental factors include sex and ethnicity. Abbreviations: MAO, monoamine oxidase; GAD65, glutamate decarboxylase; NMDA, *N*-Methyl-D-aspartic acid; CREB1, CAMP responsive element binding protein 1; GR, glucocorticoid receptor; CACNA1, calcium channel, voltage-dependent, T type, alpha 1I subunit;

COMMENTARY

Check for updates

Pain chronification: what should a non-pain medicine specialist know?

Bart Morlion[a], Flaminia Coluzzi[b], Dominic Aldington[c], Magdalena Kocot-Kepska[d], Joseph Pergolizzi[e], Ana Cristina Mangas[f], Karsten Ahlbeck[g] and Eija Kalso[h]

[a]Leuven Centre for Algology & Pain Management, University Hospitals Leuven, KU Leuven, Belgium; [b]Department of Medical and Surgical Sciences and Biotechnologies Unit of Anaesthesia, Intensive Care and Pain Medicine, Sapienza University of Rome, Rome, Italy; [c]Royal Hampshire County Hospital, Winchester, UK; [d]Department of Pain Research and Treatment, Jagiellonian University Medical College, Kraków, Poland; [e]Global Pain Initiative, Golden, CO, USA and Naples Anesthesia and Pain Associates, Naples, FL, USA; [f]Hospital de Santo André, Leiria, Portugal; [g]Capio St Görans Hospital, Stockholm, Sweden; [h]Pain Clinic, Departments of Anaesthesiology, Intensive Care, and Pain Medicine, Helsinki University Central Hospital, Helsinki, Finland

ABSTRACT

Objective: Pain is one of the most common reasons for an individual to consult their primary care physician, with most chronic pain being treated in the primary care setting. However, many primary

ARTICLE HISTORY
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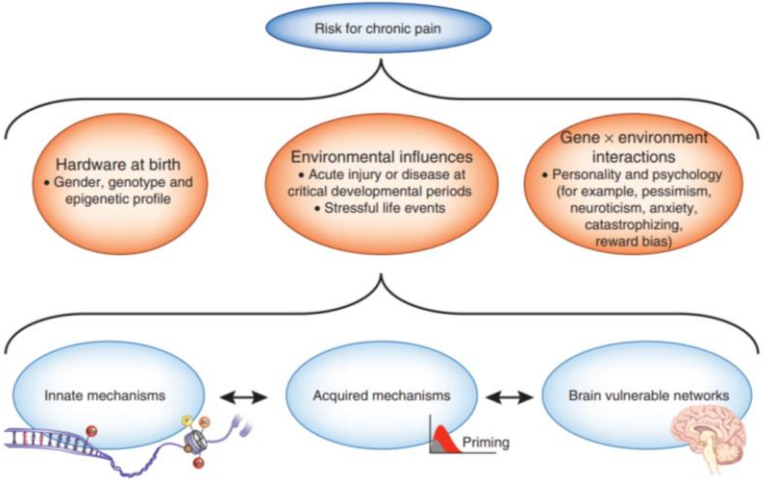

Denk F, McMahon SB **Neurobiological basis for pain vulnerability: why me?** Pain. 2017 Apr;158 Suppl 1:S108-S114.

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Neuron
Review

CellPress

The Genetics of Neuropathic Pain from Model Organisms to Clinical Application

Margarita Calvo,[1,10] Alexander J. Davies,[2,10] Harry L. Hébert,[3,10] Greg A. Weir,[2,9,10] Elissa J. Chesler,[4] Nanna B. Fi
Roy C. Levitt,[6] Blair H. Smith,[3] G. Gregory Neely,[7] Michael Costigan,[8,*] and David L. Bennett[2,*]
[1]Departamento de Fisiología, Facultad de Ciencias Biológicas, Pontificia Universidad Católica de Chile, Santiago, Chile
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[3]Chronic Pain Research Group, Division of Population Health and Genomics, Mackenzie Building, Ninewells Hospital & Medical S
University of Dundee, Dundee, UK
[4]The Jackson Laboratory, Bar Harbor, ME, USA
[5]Department of Clinical Medicine, Danish Pain Research Center, Aarhus University, Aarhus 8000, Denmark
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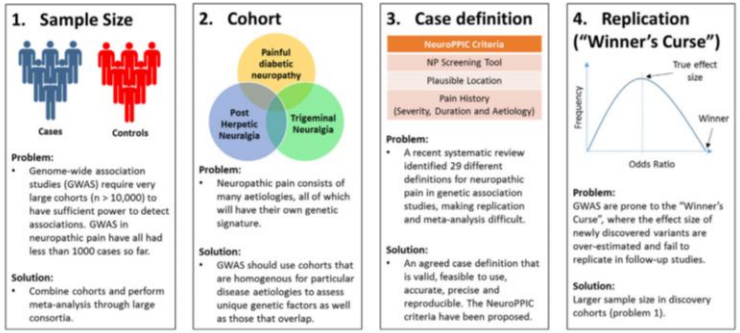
Genetics, Miller School of Medicine, University of Miami, Miami, FL, USA
[7]Dr. John and Anne Chong Lab for Functional Genomics, Camperdown, University of Sydney, Sydney, NSW, Australia
[8]Departments of Anesthesia and Neurobiology, Children's Hospital Boston and Harvard Medical School, Boston, MA, USA
[9]Present address: Institute of Neuroscience and Psychology, College of Medical, Veterinary and Life Sciences, University of Glas
Glasgow, UK
[10]These authors contribu
*Correspondence: micha
https://doi.org/10.1016/j

Neuropathic pain (N
disabling, renderin
conservation of pai



2. The Challenges of Conducting Genome-wide Association Studies in NeuP

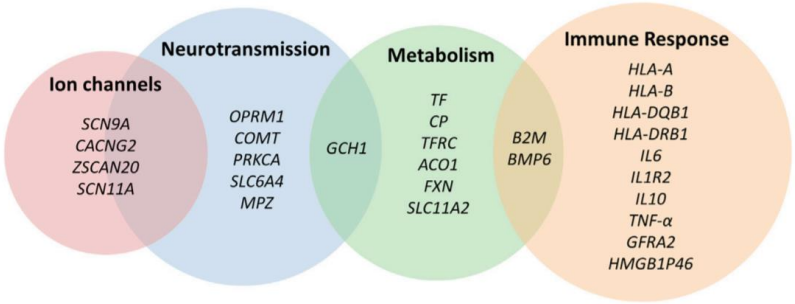


Figure 3. A Venn Diagram of Genes Reaching Study Specific or Suggestive Significance in Human Candidate Gene and Genome-wide Studies So Far in NeuP and the Overlap of Biological Pathways

These genes have been summarized in a recent systematic review of NeuP by Veluchamy et al. (2018), where the inclusion criteria were any study analyzing genetic variants in people with NeuP compared to people without NeuP. The number of genes and our understanding of their contribution within these pathways, in the context of NeuP, is likely to change as more studies are published.

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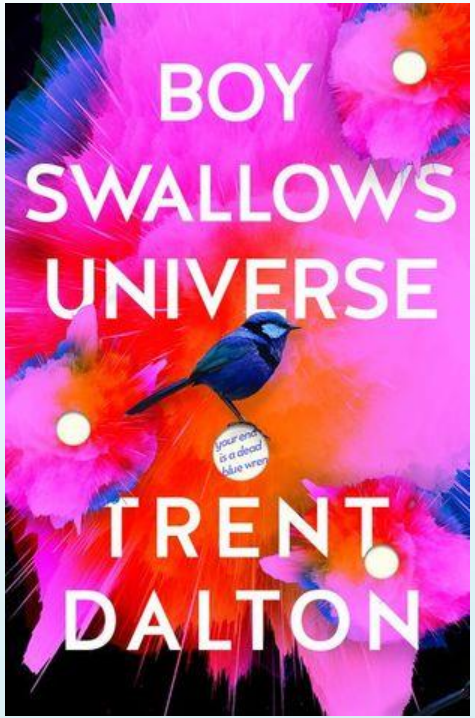
Past life events……



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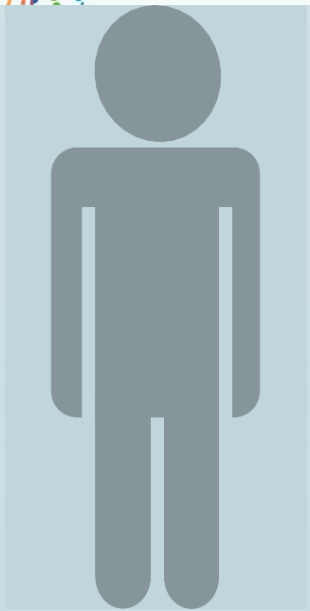
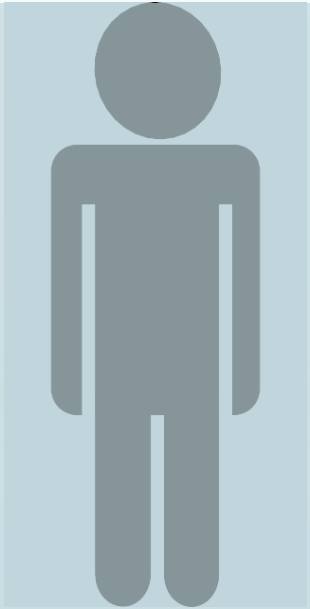
2020 GLOBAL YEAR FOR THE PREVENTION OF PAIN

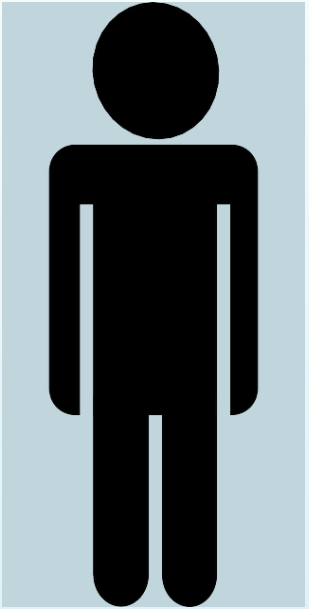
Overview



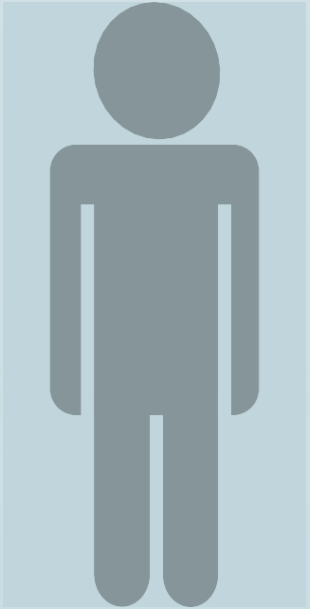
What is Post Traumatic Neuropathic pain PTNP?



Who gets PTNP?



Why prevent PTNP?



How to prevent these injuries?

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Incorrect diagnosis of Endo PTNP

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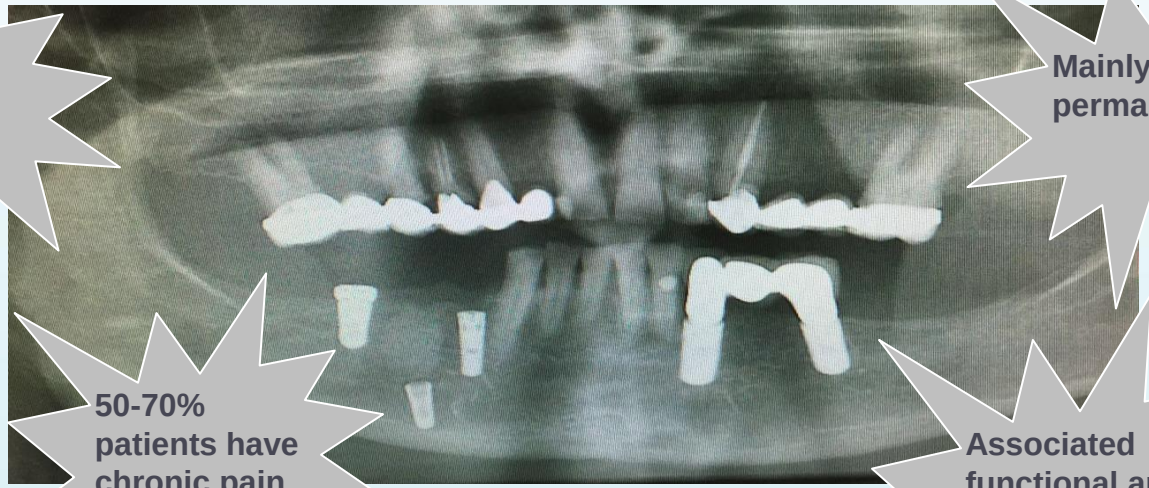
Why are nerve injuries such a big deal ?



Avoidable / negligent

Mainly permanent

50-70% patients have chronic pain

Associated functional and psychological impact

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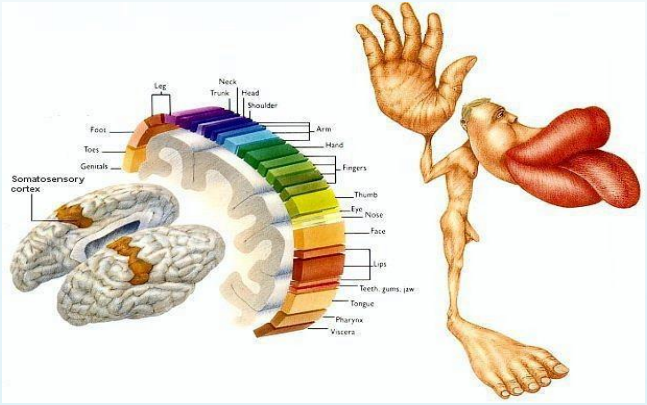
Particular issues with Trigeminal pain?

- Big part of our lives
- Underpins the primordial survival instincts
- Constant unavoidable activity
- Underpins daily pleasure in health
  - Eating
  - Drinking
  - Speaking
  - Smiling
  - Sexual interaction
- **Underpins our identity!**



Most nerve injuries are permanent and cannot be fixed

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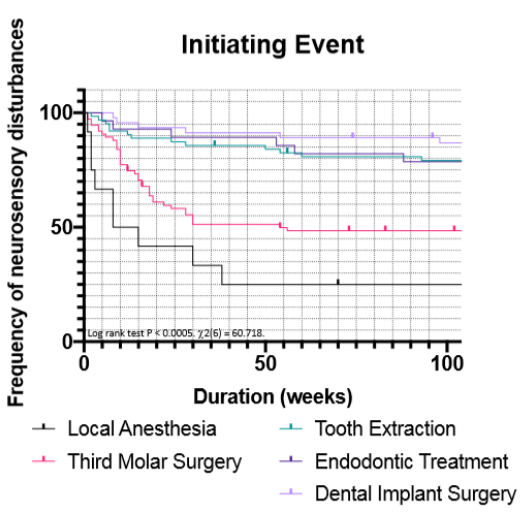
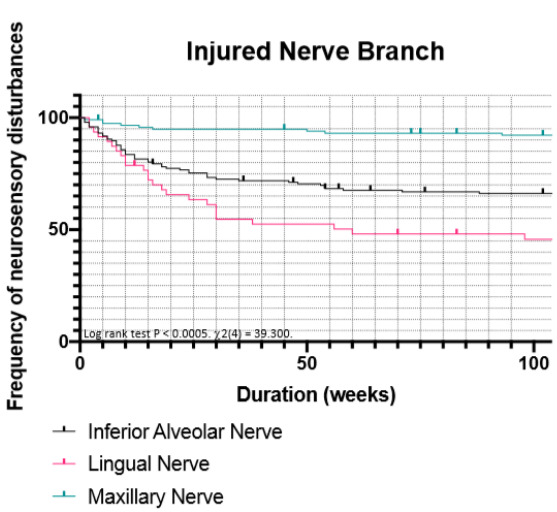
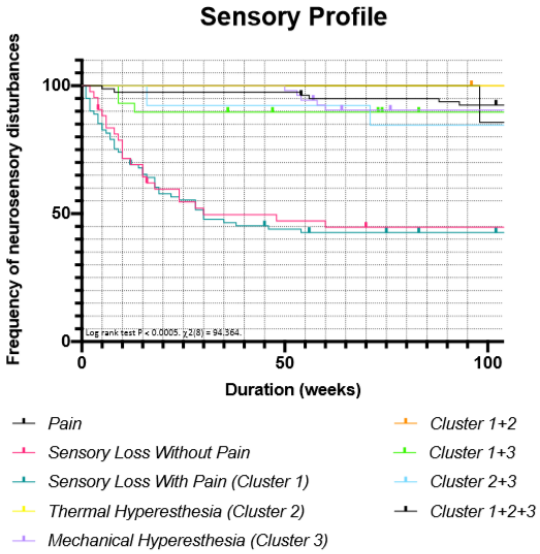
Prognosis V Nerve injuries N=1331

Kaplan–Meier analysis of neurosensory disturbances over time comparing the injured nerve branch (A), initiating event (B), and sensory profile (C).







Van der Cruyssen F, Peeters F, De Laat A, Jacobs R, Politis C, Renton T. Factors affecting evolution of symptoms and quality of life in patients referred for iatrogenic post-traumatic trigeminal neuropathy: a longitudinal study in two tertiary referral centers in UK and Belgium. Pain 2020 in press.

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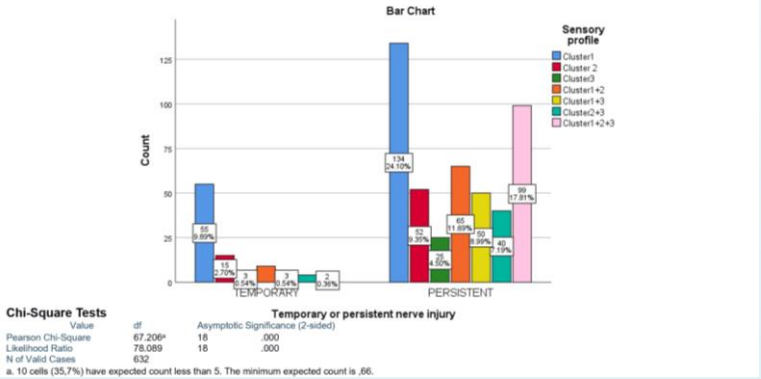
Predictive prognosis by clustering n=1331

Persistent vs temporary between clusters



Chi-Square Tests

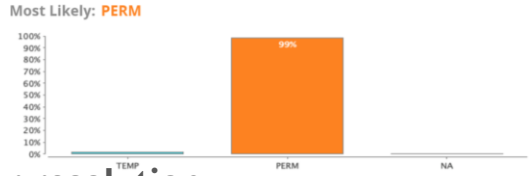
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	67,206ª	18	,000
Likelihood Ratio	78,089	18	,000
N of Valid Cases	632		

a. 10 cells (35,7%) have expected count less than 5. The minimum expected count is ,66.

Positive factors for resolution

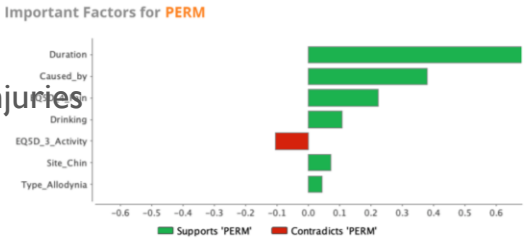
LA or M3M cause
EQ5D low pain
Lingual nerve
Sensory loss with or without pain

Prediction Model RapidMiner (generalized linear model)



Negative factors for resolution

EQ5D poor activity
Allodynia
Endo Implant nerve injuries
Maxillary nerve
Duration of NI



Collaboration with University of Leuven
Fréderic Van de Cruyssen

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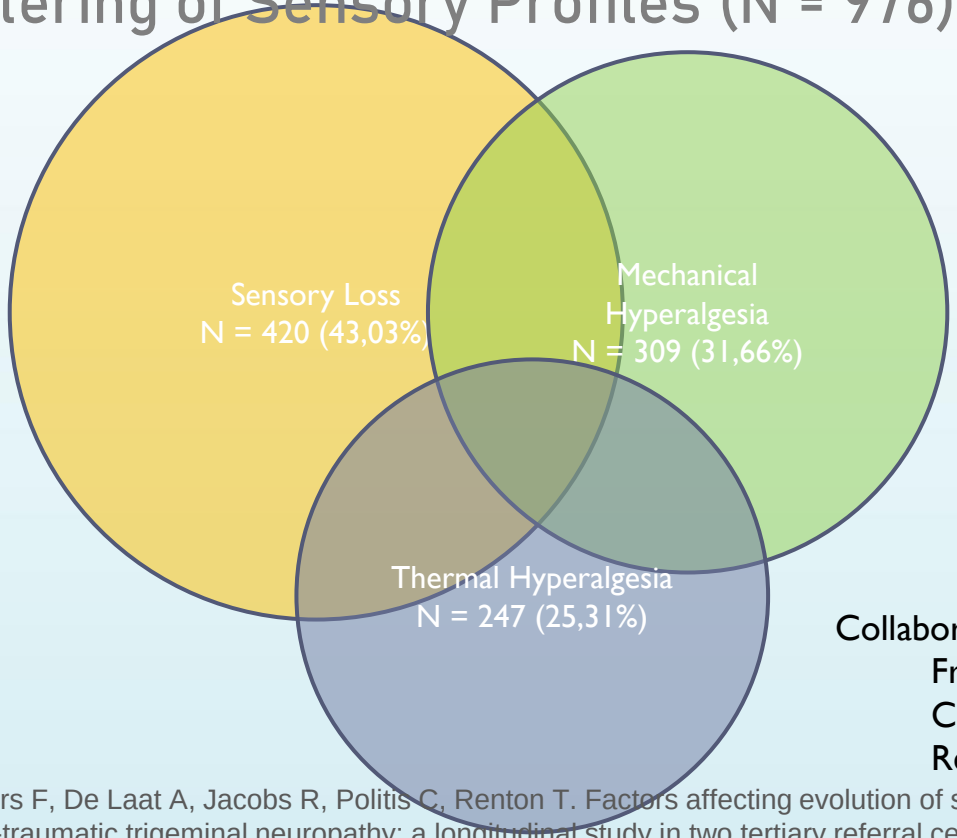
Clustering of Sensory Profiles (N = 976) in press



Collaboration with University of Leuven
Fréderic Van der Cruyssen
Constantis Politis
Reinhilde Jacobs

Van der Cruyssen F, Peeters F, De Laat A, Jacobs R, Politis C, Renton T. Factors affecting evolution of symptoms and quality of life in patients referred for iatrogenic post-traumatic trigeminal neuropathy: a longitudinal study in two tertiary referral centers in UK and Belgium. Pain 2020 in press

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Consequences

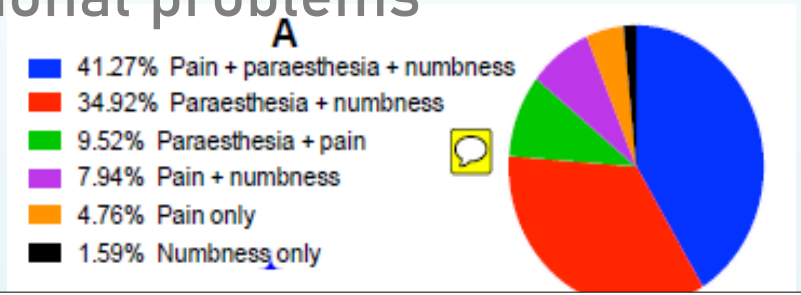
Neuropathy causing functional problems

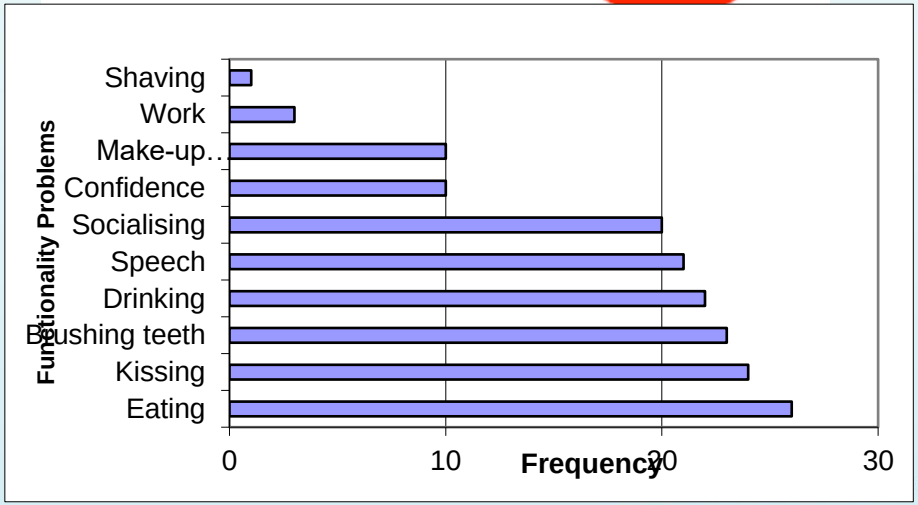
Recent study @ KCL on 100 implant nerve injury patients

95% of implant nerve injury neuropathic pain

92% permanent

Functional and psychological impact





Renton T, Dawood A, Shah A, Searson L, Yilmaz Z. Post-implant neuropathy of the trigeminal nerve. A case series. Br Dent J. 2012 Jun 8;212(11):E17. doi: 10.1038/sj.bdj.2012.497

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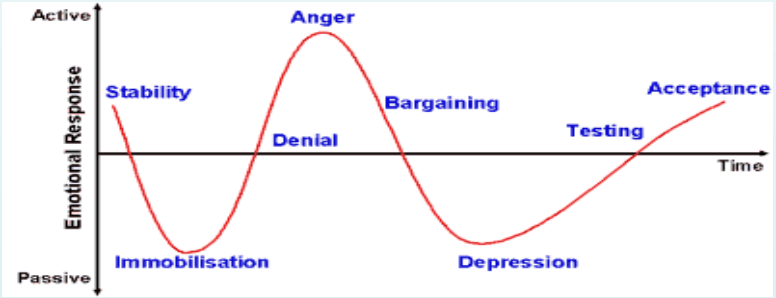
Psychological consequences

- Depression
- Anger
- Post traumatic stress disorder 68%
- Victim of abuse
- Loss of ability to trust

Kubler Ross



The psychosocial and affective burden of posttraumatic neuropathy following injuries to the trigeminal nerve. **Smith** JG, **Elias** LA, **Yilmaz** Z, Barker S, Shah K, Shah S, **Renton** T. J Orofac Pain. 2013 Fall;27(4):293-303. doi: 10.11607/jop.105 Sullivan MJ et al. Catastrophizing and perceived injustice: risk factors for the transition to chronicity after whiplash injury. Spine (Phila Pa 1976). 2011 Dec 1;36(25 Suppl):S244-9 Dec;92(12):2041-56. Review

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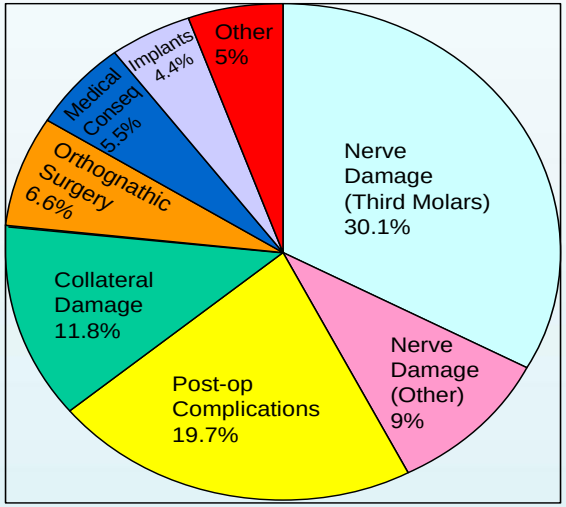
Medicolegal consequences

Nerve damage related to dental procedures are often NEGLIGENT as they are elective surgery and damage is avoidable.

- This results in litigation and Settlements getting more expensive
  - Implant related cases settlements $1-3 million (2011)



Other	1.5%	TMJ Surgery
	1.2%	Consent Only
	0.5%	Failure to diagnose/treat
	0.3%	Maxillary Sinus
	1.5%	Misc
	5%	

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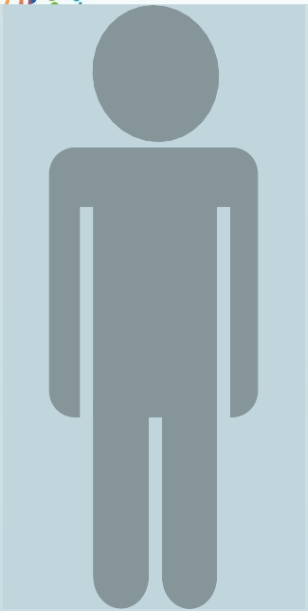
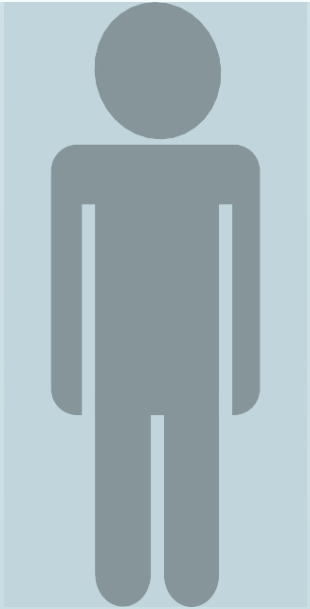
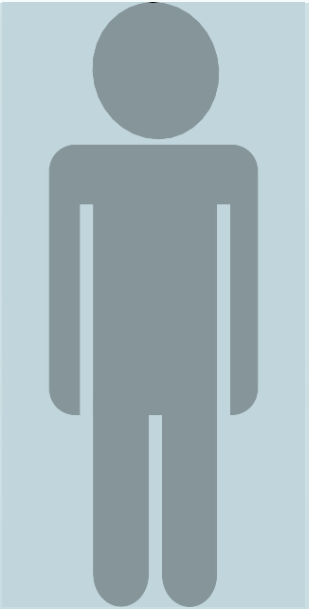
Overview



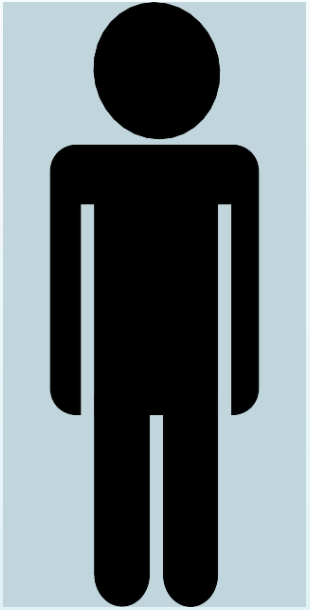
What is Post Traumatic Neuropathic pain PTNP?



Who gets PTNP?



Why prevent PTNP?



How to prevent these injuries?

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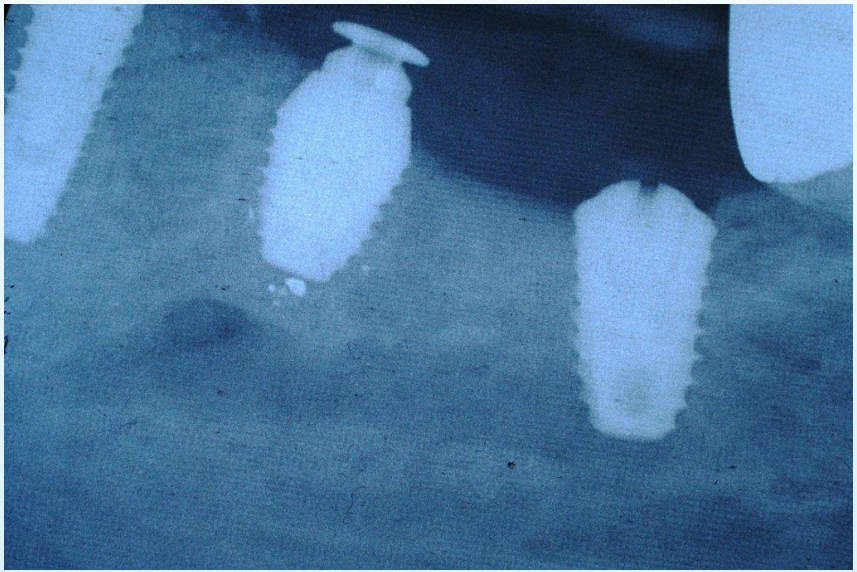
Preventing dentistry related nerve injury and PTNP



How do we prevent these injuries?

- Managing patients expectations
- Risk assessment and management
- Operative technique
- Post op follow up
- Recognition and early medical and or surgical intervention (if indicated)

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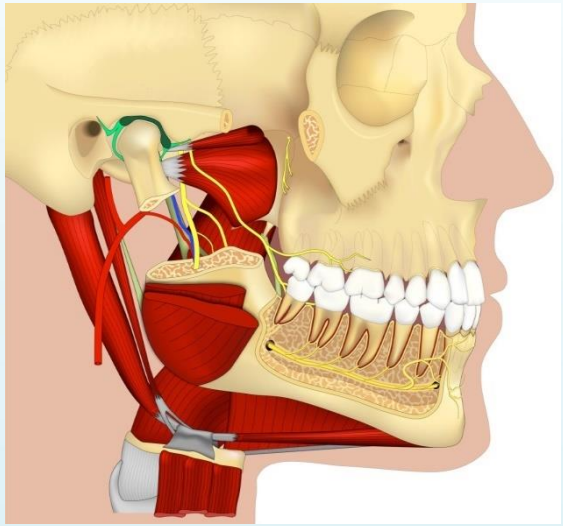
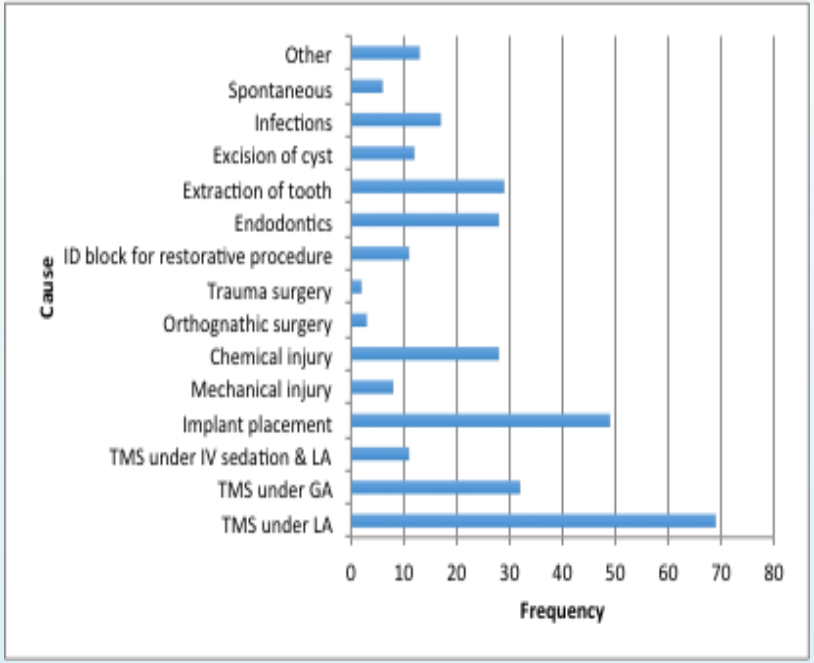
Prevention of Trigeminal Post Traumatic Painful Neuropathy?





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Risk factors for persistent neuropathy related to IDBs
In order to minimise complications related to dental LA you need to consider modifying the following risks;

- **Block anaesthesia** Nerve block injections should be undertaken without intent on direct [illegible] patients who experience the 'funny bone' neuralgia due to the IDB needle being placed too [illegible] inferior alveolar nerves experience persistent neuropathy (20)
- **Lingual nerve > IAN** Is this technique related or anatomically related (less fascicles [illegible] recovery). Perhaps the direct IDB approach may place the lingual nerve at increa[illegible] risk [illegible] indirect technique. (14)
- **Concentration of LA** Any increased concentration of any agent leads to incre[illegible] [illegible]ity (21)
- **Volume of LA** There is no evidence to support this sugg[illegible] [illegible]are neurotoxic, dependent upon the proximity, LA concentration, neural damage ad[illegible] potential neurotoxicity.
- **Multiple injections** Second or subsequent injections that impede directly o[illegible] associated with the usual 'funny bone' neuralgic pain. Thus the patient does not self-prot[illegible] rendering the nerves more at risk of direct damage.
- **Severe pain on injection** 60% increased occurrence of persistent neuropathy after IDBs (21)
- **Type of LA Agent** Bupivicaine most neurotoxic of all LA agents
- **Type of vasoconstrictor?** The role of vasoconstrictor in nerve damage is unknown[illegible]
- **Sedated or anaesthetized patients?** There is no evidence to support unresponsive[illegible] [illegible]y to protect themselves when neuralgia (funny bone reaction) occurs as the IDB needle encro[illegible] nerve.
- **Lack of LA aspiration?** Again there is no evidence to support that aspiration d[illegible] [illegible]ersistent neuropathies but a pragmatic view may infer less chemical injected intra neura[illegible] [illegible]mical nerve

Block injections

Multiple injections

Type and concentration of LA agents

Extreme pain during injections

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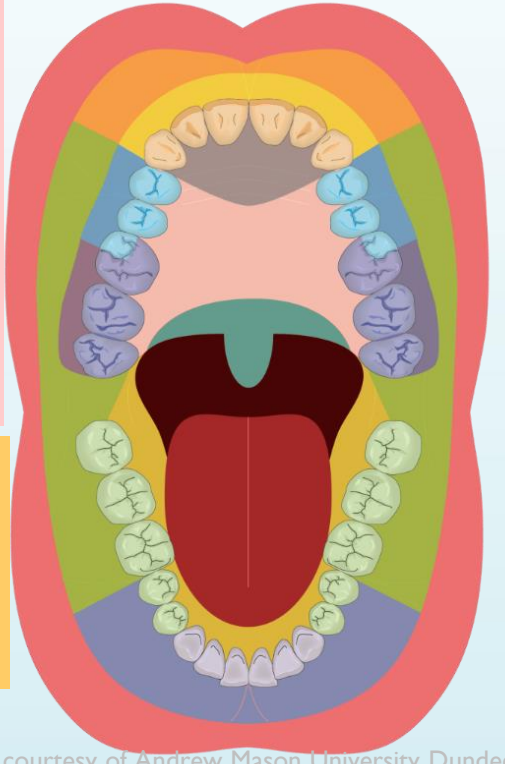
Infiltration dentistry is dependant upon the site and procedure

Maxillary dentistry can be performed entirely using Lidocaine 2% with adrenaline for all procedures
Buccal infiltration with intra-septal injections
No additional benefit using 4% Articaine
No palatal or incisal blocks are indicated

IDBS needed for
Posterior mandibular molar
Endodontic procedures may require IDBs or higher techniques (Gow Gates or Akinosi)



Mandibular 7s and 8s for **perio, restorations or implants**
Articaine 4% buccal infiltration and Lidocaine 2% lingual infiltrations OR for **extractions** intraligamental
If fails may need lidocaine IDB

Mandibular 1st molars for **perio, restorations or implants**
Articaine 4% buccal +/- Lidocaine 2% crestal or lingual infiltration s OR for **extractions** add lidocaine lingual **of** intra-ligamental

Mandibular premolars, canines incisors for **perio, restorations or implants**
Articaine buccal infiltration (incisal nerve block using 30% cartridge) adjacent not in the mental foramen and massage over region. If fails repeat or add crestal or lingual infiltration OR for **extractions**, intra-ligamental

Illustration modified from figure courtesy of Andrew Mason University Dundee

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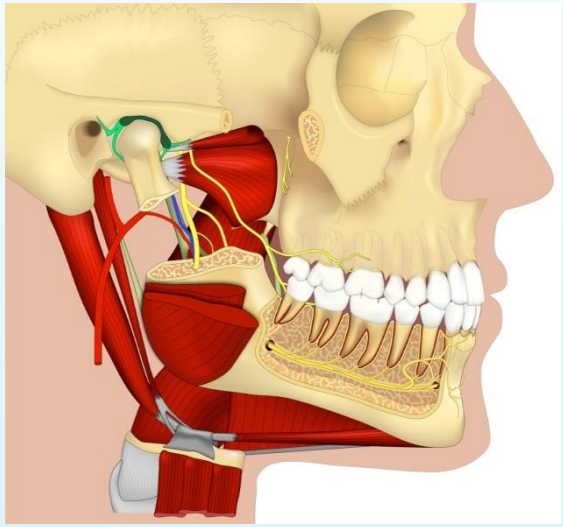
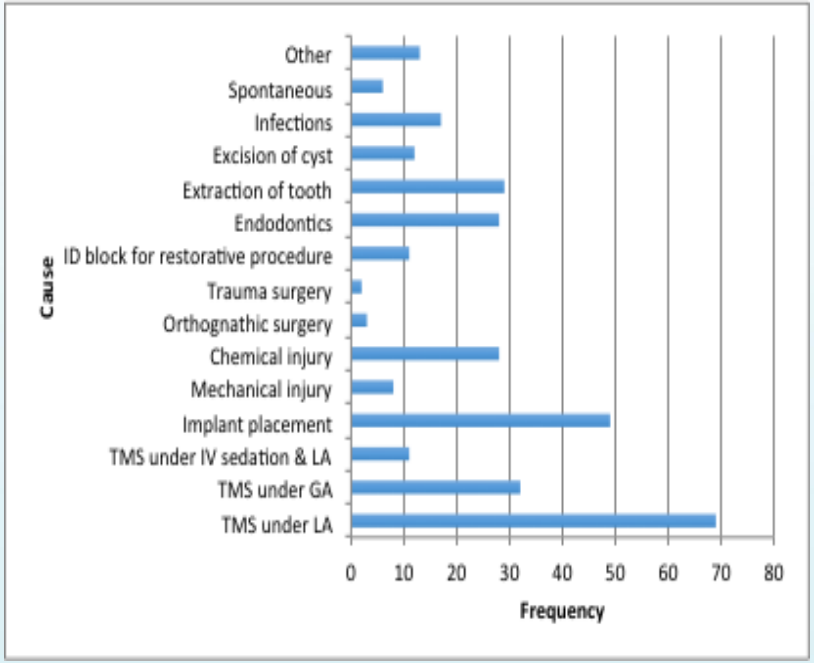
Prevention of Trigeminal Post Traumatic Painful Neuropathy?





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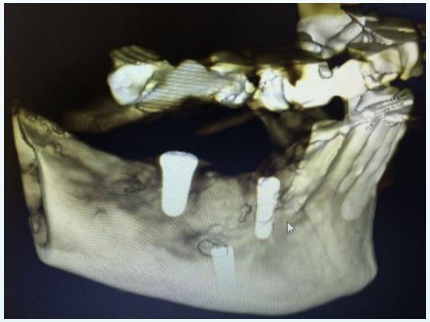
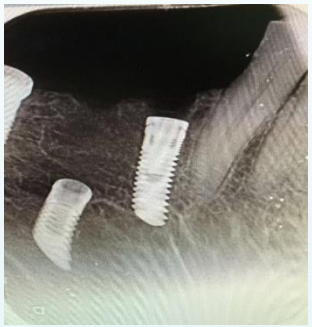
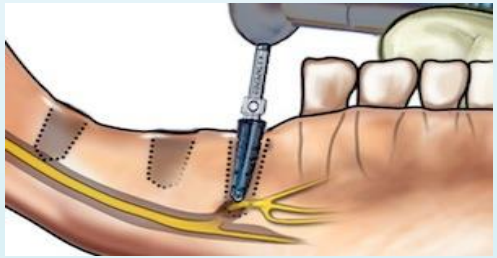
Prevention of Implant nerve injury
Risk factors

Most nerve injuries occur:

- **In patients over 47 years**
- **In the parasymphyseal region**
- **During preparation of implant bed**
- **Using Implants >10mm**
- **When the patient experiences severe pain**
  - during prep or implant placement
  - severe pain post surgery
  - Intraoperative bleed during prepping







Yilmaz Z, Ucer C, Scher E, Suzuki J, **Renton T**. A Survey of the Opinion and Experience of UK Dentists: Part 1: The Incidence and Cause of Iatrogenic Trigeminal Nerve Injuries Related to Dental Implant Surgery. Implant Dent. 2016 Oct;25(5):638-45.

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Risk factors 1

A. Poor risk assessment - Inadequate preoperative assessment and planning due to;

Lack of knowledge/inexperience

Inadequate informed consent and management of patient expectations

Lack of identification of existing pre-surgical neuropathy.

Additional risk assessment of mandibular premolars and m

Poor planning

Know where the nerve is. Nerve localisation, risk factors when assessi
(Mental loop, characteristics of IAN position in various sites of mandible)

Parasymphyseal zone high risk.

The accuracy of estimating the position o
or CT scans is highlighted in the radiograph

Insufficient Safety zone- Risk perf
to the nerve.

Poor surgical technique

Poor recognition of intraoperative problems

Poor implant placement

Selection of implants 10mm plus

(evidence supports shorter implants **-**short impla
procedure and minimise morbidity**)**

Poor Planning

Insufficient Safety zone

Inappropriate radiographs

Inability to read CBCT

Using implants > 8mm

Operative

Poor technique reducing Safety zone/ lack use drill stops, guides/ intraoperative LCPAs

Lack of recognition risks bleeding/ drill sink

Post operative

Late recognition of nerve injury

Lack removal implant within 30 hours

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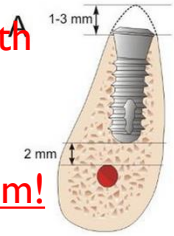
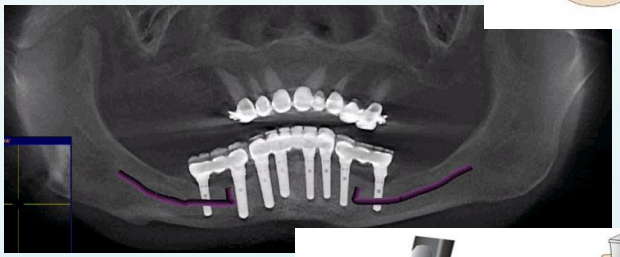
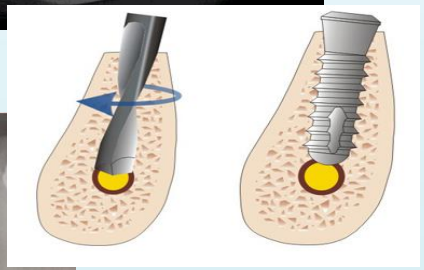
Evidence for prevention of implant related nerve iniuries

- Computer guided surgery (**none**)
- Use surgical guides (**moderate**)
  - (Chan, Chik, Pow, & Chow, 2013; Van Assche et al., 2007).
- Drill stops stock or tailored **(none)**
- ITI recommendation **(moderate)**
  - PAUSE after 60% planned depth OR 6mm
  - Take LCPA and check position

Safety zone of 2mm is insufficient with implant drills 1.5mm longer than the implants = resultant safety zone of 0.5mm!!!! 4mm!



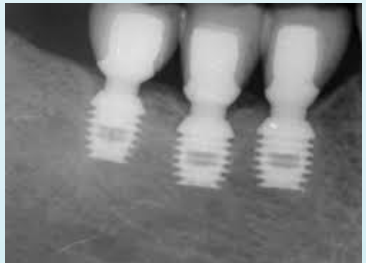


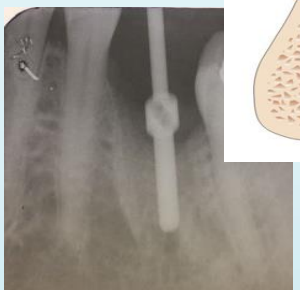


- **USE SHORT IMPLANTS** less than 10 mm for parasymphyseal region (**strong**) Implants should not need to be longer than 8 mm

Short Implants (5 to 8 mm) Versus Longer Implants (>8 mm) with Sinus Lifting in Atrophic Posterior Maxilla: A Meta-Analysis of RCTs

Tengfei Fan;* Yicun Li;* Wei-Wei Deng;* Tianfu Wu;* Wenfeng Zhang*,†





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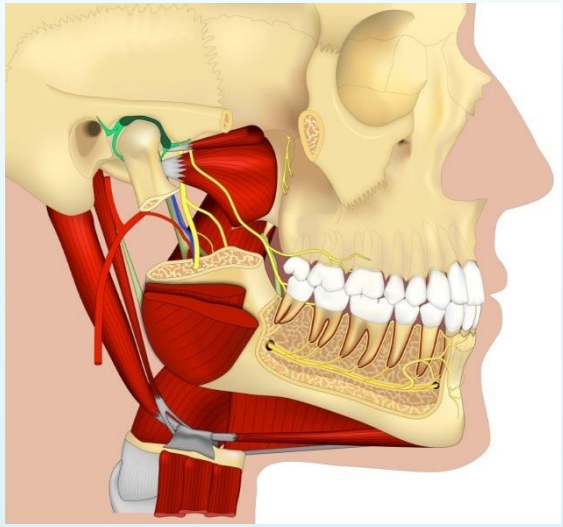
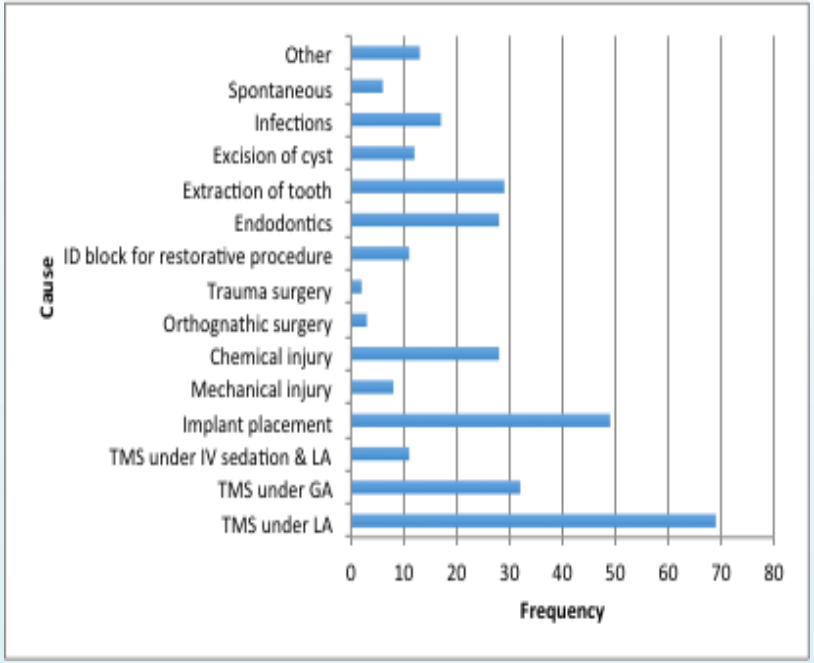
Prevention of Trigeminal Post Traumatic Painful Neuropathy?





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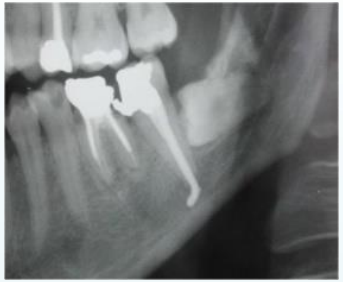
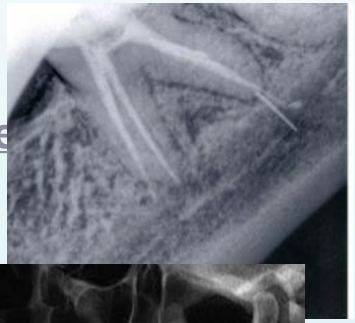
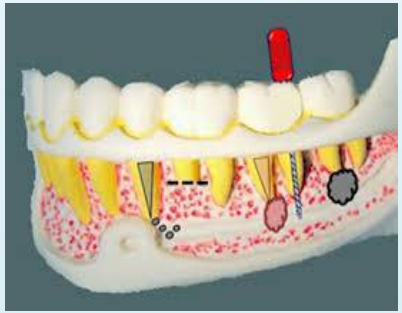
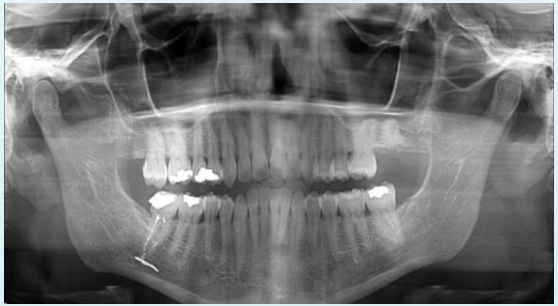
Endodontic related nerve injuries mechanisms

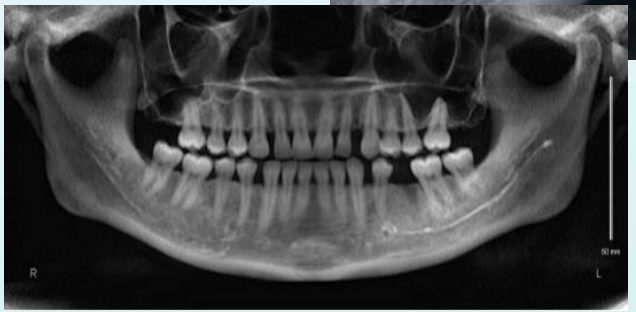
- Mechanical compression canal due to overfill
- Direct mechanical damage due to over instrumentation
- Haemorrhage with direct and indirect neural ischaemia
- **Loss of apical seal and CHEMICAL leakage and damage**
- Inflammation / infection











Fanibunda K, Whitworth J, Steele J (1998) The management of thermomechanically compacted gutta percha extrusion in the inferior dental canal. Br Dent J. 1998 Apr 11;184(7):330-2

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Prevention of Endodontic related neuropathy: Risk factors

A. Inadequate preoperative assessment and planning due to;

- Lack of knowledge
  - GDP (80% of referrals) GDP endodontic success rates are significan… …vs 85%)
  - The American Association of Endodontists have made several recc… …ral of these patients
- Inability to read the radiographs or CBCT
- Inadequate informed consent-all options provided and related risk benef…
- Lack of identification of existing pre-surgical neuropathy (periapical lesions)

Tooth apex position
Proximity to IDC
Related root morphology

B. Premolar teeth & Proximity of tooth apex to IDC – 90% of the mandibular teeth in this series, were close to the IAN canal or premolars adjacent to the mental foramen. Proximity to the apex to the IAN/ breach a… seal and over chemical or instrumentation

- Tantanapornkul et al (33) reported the specificity and sensitivity of … …the IAN to the tooth roots in 161 mandibular third molars 161; for it was … …70% and 63% which were not significantly different.
- Patel et al (34) have reported on the use of CBCT in managi… cone periapicals.

Poor technique
Lack apical seal
Over instrumentation
Over filling

C. Poor technique

- Breach of apex causing pain during surgery on irrigation or during instrumentati… …damage to periapical t…es
- Over instrumentation
- Overfill Detectable overfill occurred in 60% of cases and over instrumentation during preparation

D. Early recognition and intervention for Endodontic related nerve injuries

- ALWAYS undertake HOMECHECK , review patient and confirm neuropat…
- Neuropathy related to endodontics can be delayed and the patient must h… 3-4 days post treatment (Renton et al unpublished).
- If nerve injury is suspected, you will already be aware of the proximity of th… …was likely breach of apex, over instrumentation or deposition of endodontic material in…
- If there is suspected the material, the apex and or tooth must be removed within 4… …of placement in ord… …maximise recovery from nerve injury (9). If the patient is insistent on keeping the tooth urgent referral of the patient may be indicated for

Postoperative
Late recognition and late tooth or overfill removal

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Risk assessment Radiographic Proximity to the Inferior dental canal (IDC)

Mandibular teeth proximal to the IAN canal

- Apex of the tooth may be adjacent or intruding into the IDC canal and any small degree of leakage or overfilling may compromise the IAN.
- Assessment of the proximity of the tooth apex to the IAN canal has become significantly improved with Cone Beam CT scanning (CBCT) with the attendant risk of additional radiation and may not provide significantly more information than a plane long cone radiograph.
- Most of CBCT assessment of tooth positioning relation to the IAN canal is based on M3M prior to extraction

Is there a "safety zone" in the mandibular premolar region where damage to the mental nerve can be avoided if periapical extrusion occurs?

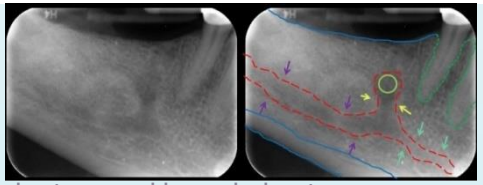
Wei Cheong Ngeow, BDS (Mal), FFDRCS (Ireland), FDSRCS (Eng), MDSc (Mal), AM (Mal)

Posted on June 16, 2010

Tags: adverse reactions endodontics radiology

Anatomic Relationship between the Inferior Alveolar Nerve and Dental Apex

Tilotta-Yasukawa and colleagues[11] determined the proximity of the apex of the premolars and molars in relation to the mandibular canal, as well



Tilotta-Yasukawa F, Millot S, El Haddioui A, Bravetti P, Gaudy JF.Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006 Oct;102(4):e4

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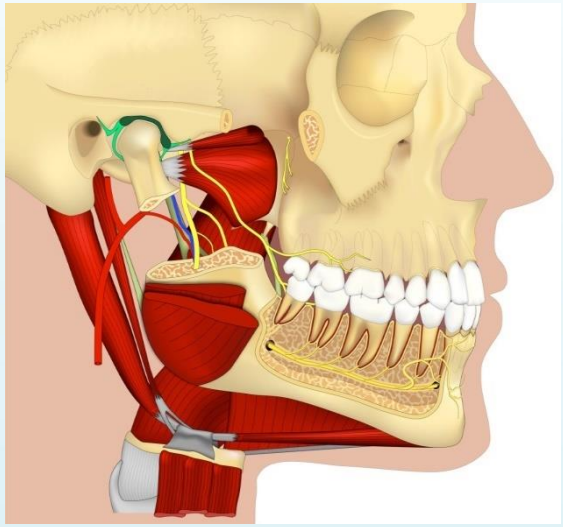
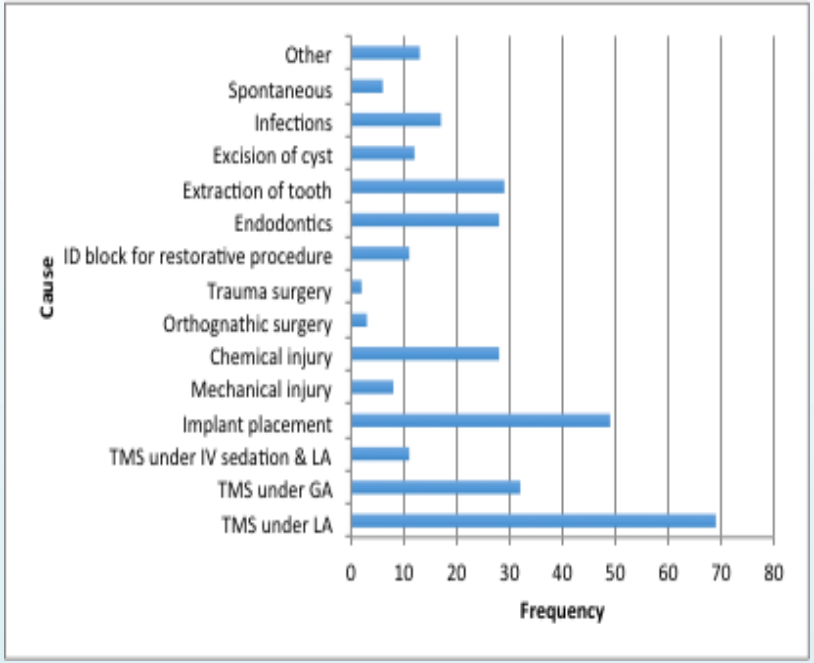
Prevention of Trigeminal Post Traumatic Painful Neuropathy?





Local anaesthesia
Dental Implants
Endodontics
Third molar surgery

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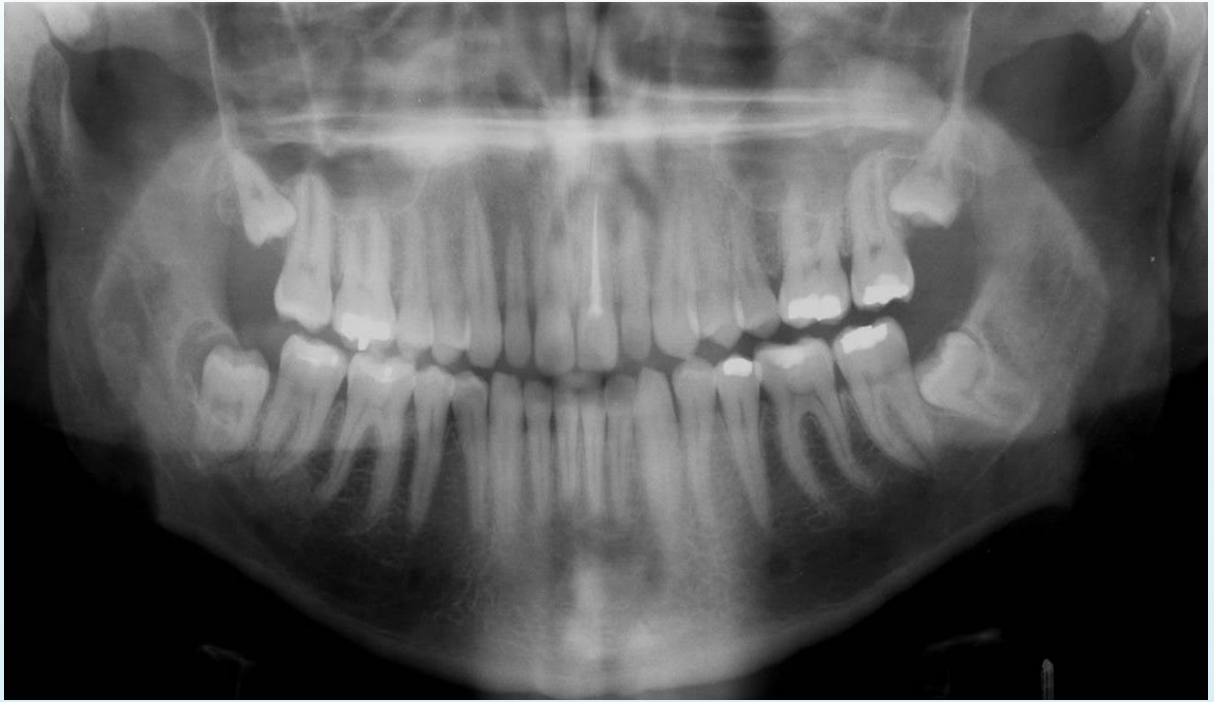
Preventing M3M surgery related PTPN



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Lingual nerve
Age of the patient
Poor surgical technique
- Junior surgeons
- Duration of surgery
- Lingual access surgery
- Distal bone removal and lingual nerve injury
  - Use Buccal approach
  - Minimal access

'aberrant' Lingual nerve anatomy
- 11-18% of lingual nerve above alveolar crest distal to M3Ms

Inferior alveolar nerve
Age of the patient
- Intra-operatory exposure of the nerve
- Un-erupted tooth

Poor Radiographic risk assessment
- Perforation of tooth roots by IDC
- Proximity of tooth roots to inferior dental canal (IDC)
- Plain film
  - IDC loss LD
  - Darkening of roots
  - Deviation of IDC
- CBCT lack cortication, distortion of canal.
- Lingual IDC

Acta Odontol Scand. 2013 Jul 4. The importance of a good evaluation in order to prevent oral nerve injuries: A review.Céspedes-Sánchez JM, Ayuso-Montero R, Marí-Roig A, Arranz-Obispo C, López-López J. 662 were obtained from the search, from which 25 were selected accomplishing the inclusion criteria. Moreover, seven important articles were selected from the references of the ones mentioned, obtaining a total of 32 articles for the review.
Renton T, McGurk M. Brit J Oral Maxillofac Surg 2001; 39: 423-428 Acta Odontol Scand. 2013 Jul 4. [Epub ahead of print]
The importance of a good evaluation in order to prevent oral nerve injuries: A review.Céspedes-Sánchez JM, Ayuso-Montero R, Marí-Roig A, Arranz-Obispo C, López-López J. 662 were obtained from the search, from which 25 were selected accomplishing the inclusion criteria. Moreover, seven important articles were selected from the references of the ones mentioned, obtaining a total of 32 articles for the review.

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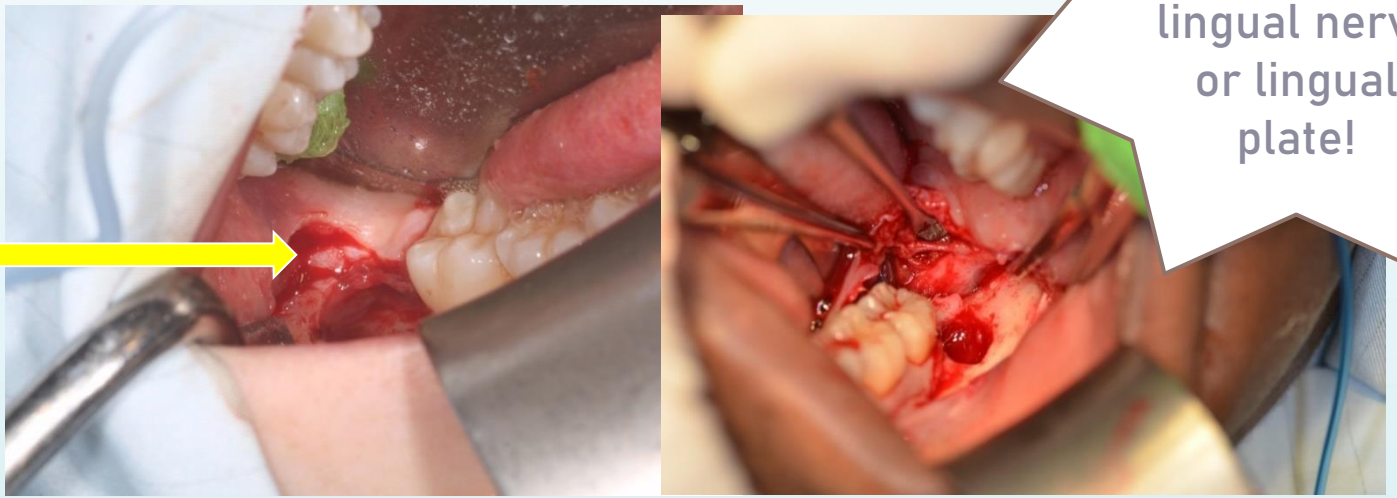
Prevention

Lingual nerve Injury in M3M surgery



Avoid going anywhere near the lingual nerve or lingual plate!

Spot the lingual nerve!

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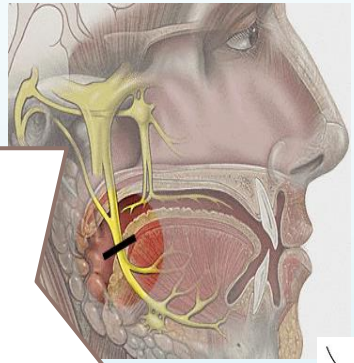
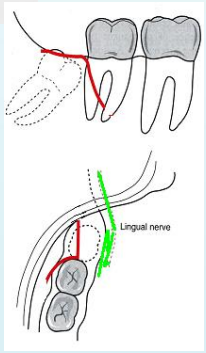
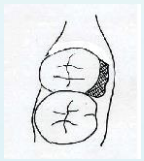
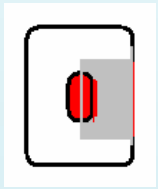
Minimal access prevents LNI

NEVER Remove distal bone OR section through the tooth

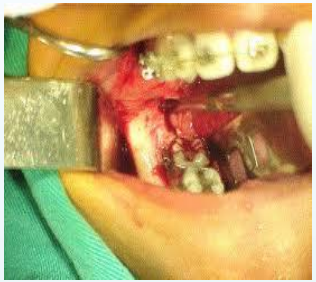


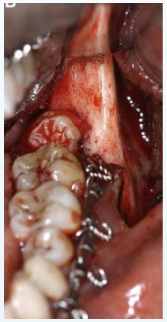




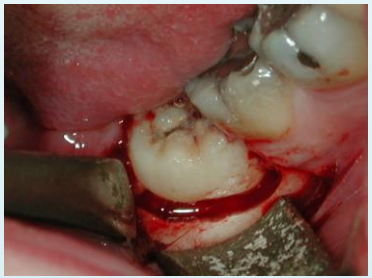


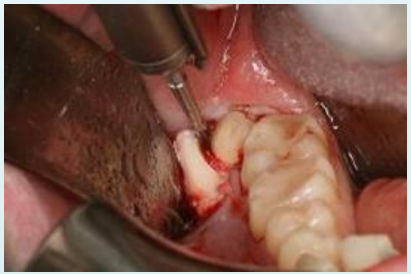
Old Technique ‘Explode the patient’





New technique minimal access





Evaluation of trigeminal nerve injuries in relation to third molar surgery in a prospective patient cohort. Recommendations for prevention. **Renton T**, Yilmaz Z, Gaballah K. Int J Oral Maxillofac Surg. 2012 Dec;41(12):1509-18.

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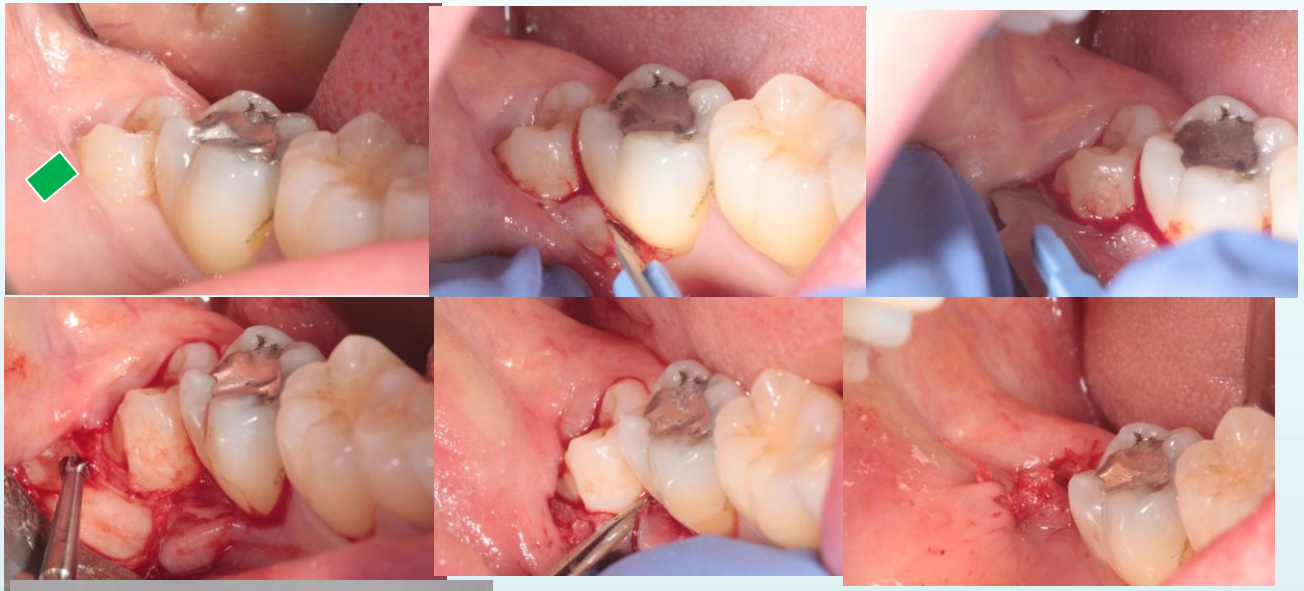
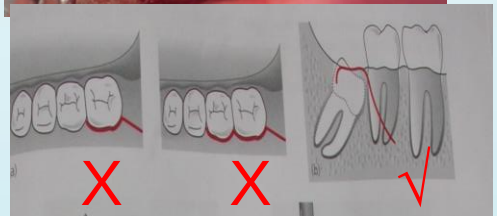
Prevention LNI related to M3M surgery

Buccal minimal access surgery



Fissure bur not rose head bur to get more accurate and minimal bone removal and tooth section



Triangular flap ensures minimal access and no exposure of distal bone behind M3M
Envelope flap increases trismus too

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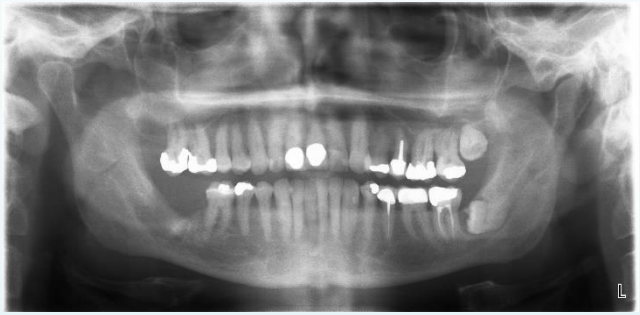
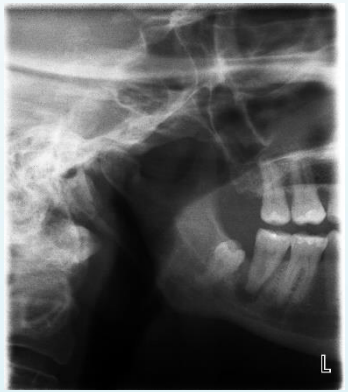
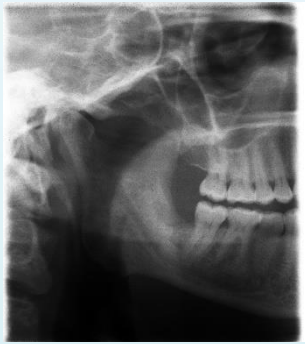
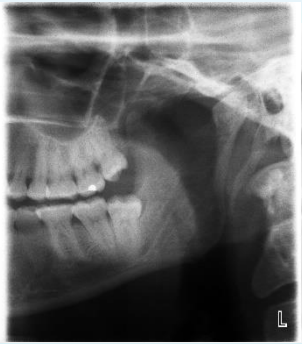
Preventing inferior alveolar nerve injury

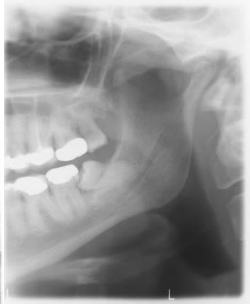
Risk assessment

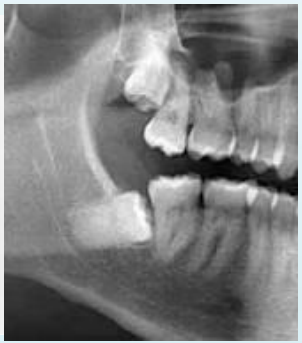












Céspedes-Sánchez JM, Ayuso-Montero R, Marí-Roig A, Arranz-Obispo C, López-López J The importance of a good evaluation in order to prevent oral nerve injuries: A review. Acta Odontol Scand.2013 Jul 4.

Factors that are associated with injury to the IAN in high-risk patients after removal of third Molars. Selvi, Dodson, Nattestad, Robertson, Tolstunov. BJOMS 51 (2013) 868–873. with permission.

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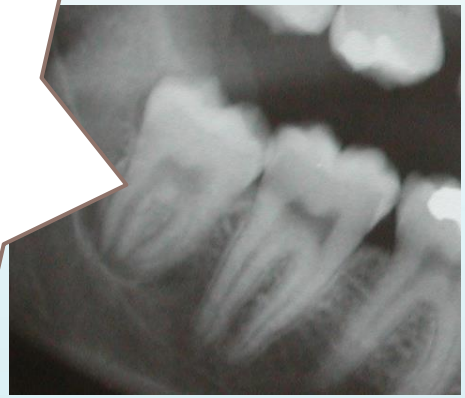
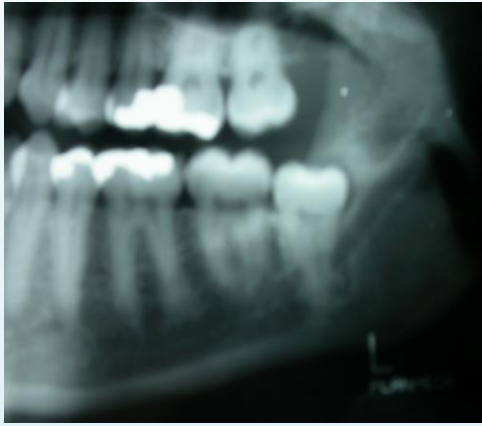
Risk assessment using plain films

Radiographic factors

- Diversion of the canal
- Darkening of the root
- Interruption of the canal LD

Recognise plain film risk factors
If high risk - CBCT





NEW

- Juxta-apical area
- Deviation of canal
- Narrowing / darkening of roots

Renton T, Hankins M, Sproate C, McGurk M. A randomised controlled clinical trial to compare the incidence of injury to the inferior alveolar nerve as a result of coronectomy and removal of mandibular third molars. Br J Oral Maxillofac Surg. 2005 Feb;43(1):7-12 Rood JP, Shehab BA.The radiological prediction of inferior alveolar nerve injury during third molar surgery.Br J Oral Maxillofac Surg. 1990 Feb;28(1):20-5 Rud J.Third molar surgery: perforation of the inferior dental canal through the root. Tandlaegebladet. 1983 Oct;87(19):659-67. No abstract available.

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Risk assessment using plain films

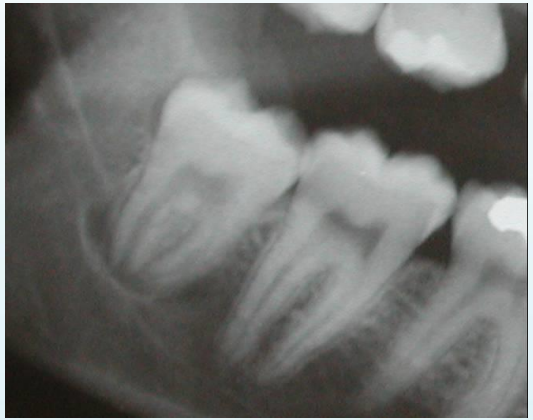
Risk

- 0.5% of cases permanently
- 2% of cases temporarily

BUT if the teeth are superimposed on the IAN canal

- 20% temporary
- 2% permanent

10 x



Risk factors

- increased age
- difficulty of surgery
- proximity to the IAN canal

- Renton T, Hankins M, Sproate C, McGurk M. A randomised controlled clinical trial to compare the incidence of injury to the inferior alveolar nerve as a result of coronectomy and removal of mandibular third molars. Br J Oral Maxillofac Surg. 2005 Feb;43(1):7-12
- Rood JP, Shehab BA.The radiological prediction of inferior alveolar nerve injury during third molar surgery.Br J Oral Maxillofac Surg. 1990 Feb;28(1):20-5
- Rud J.Third molar surgery: perforation of the inferior dental nerve through the root. Tandlaegebladet. 1983 Oct;87(19):659-67. No abstract available.

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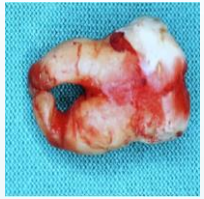
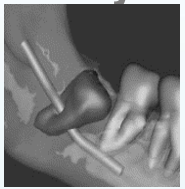
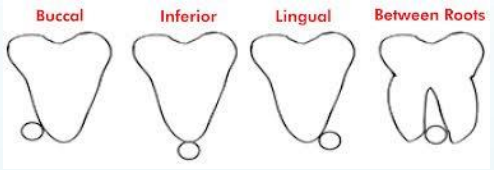
CBCT Risk assessment to IANI
Proximity to IDC and perforation





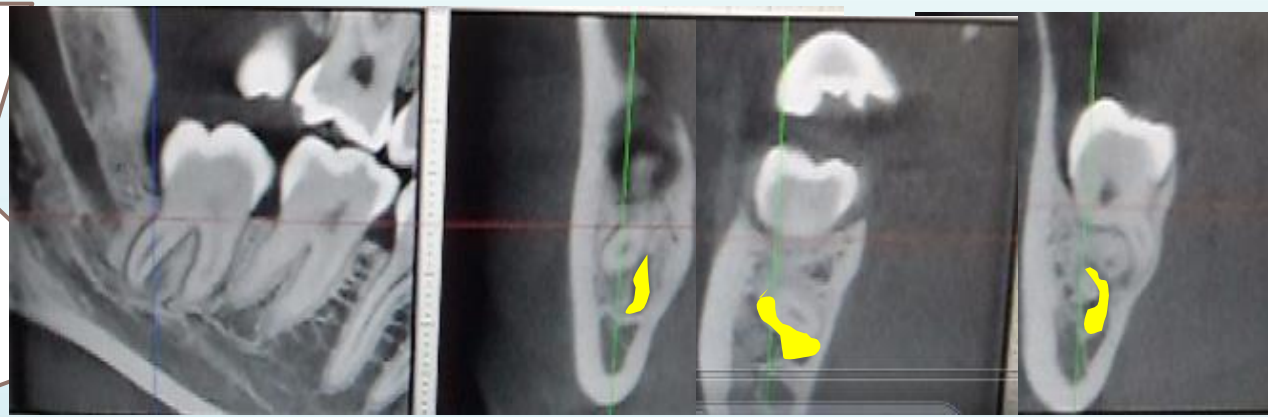


Perforation is very rare

How close does the nerve have to be?

The nerve doesn't have to 'perforate' tooth…

IAN at risk CBCT
Distortion of IDC
Lingual position IDC
Loss of cortication IDC
Bifid IDC
Inter proximal
IDC/perforation tooth root by IDC



ison between cone beam computed tomography and panoramic radiography in the assessment of the and impacted class C mandibular third molars. Dent Res J. 2011;8:203

trapment. J Oral Maxillofac Surg 68:1173-1178, 2010

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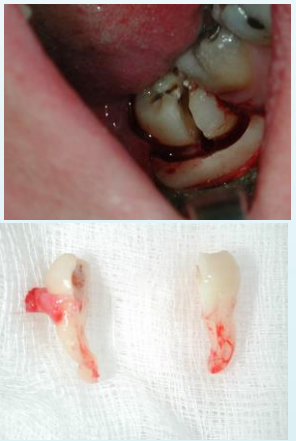
M3M Removal or Coronectomy?

- Patient healthy?
- Patient reliable?
- Tooth vital?
- Tooth high risk- confirmed on CBCT inter radicular IAN?

Yes to all → Coronectomy

No to any → Removal with sectioning



Guerrero ME, Botetano R, Beltran J, Horner K, Jacobs R Can preoperative imaging help to predict postoperative outcome after wisdom tooth removal? A randomized controlled trial using panoramic radiography versus cone-beam CT. Clin Oral Investig. 2014 Jan;18(1):335-42. doi: 10.1007/s00784-013-0971-x. Epub 2013 Mar 15.

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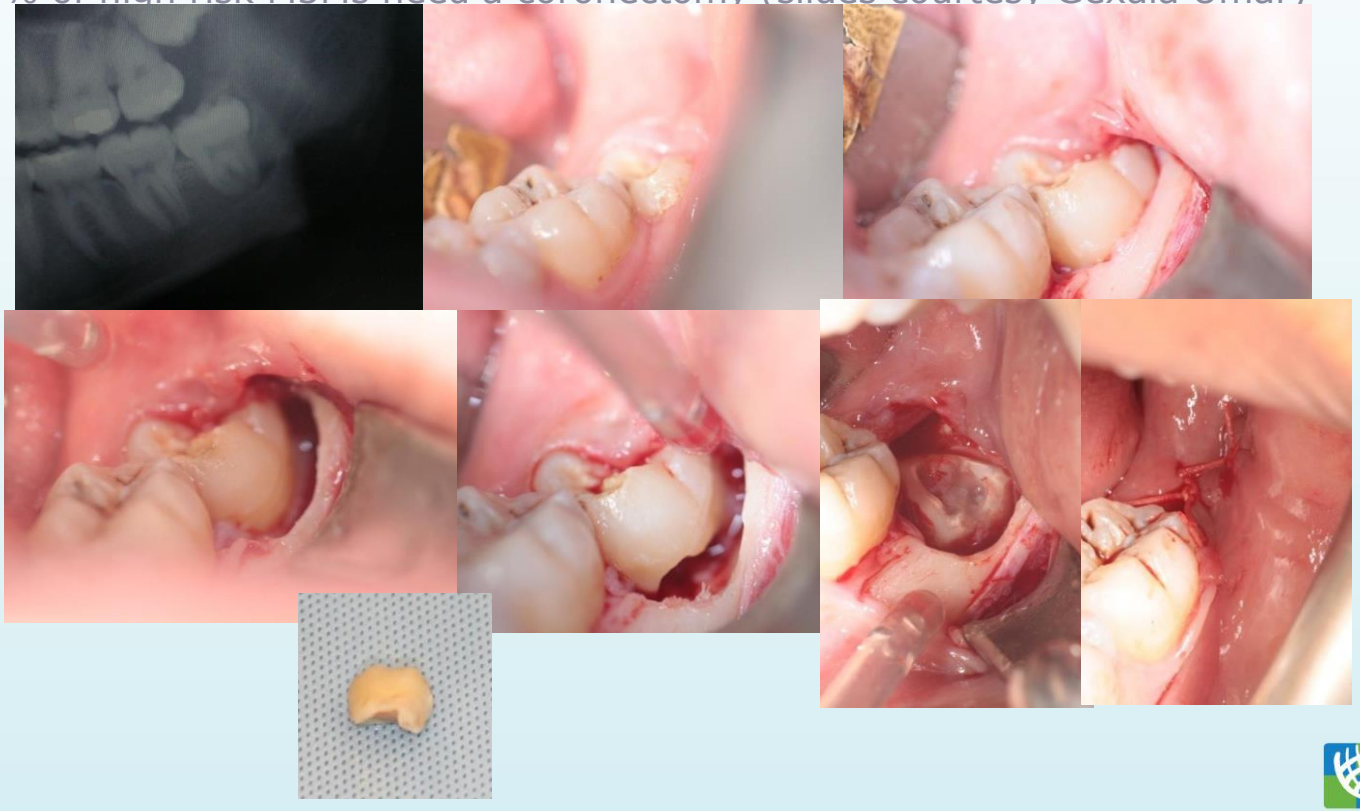
Prevention of M3M IANI Technique decision Coronectom

Less than 4% of high risk M3Ms need a coronectomy (slides courtesy Gexala Umar)



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Key messages…

Prevention of nerve injuries and related neuropathic pain is essential and possible

- Patient selection – preoperative psych assessment / pain comorbidity /age/ gender
- Good planning and risk assessment - Awareness of intraoperative risk factors
- Good surgical technique –minimal access avoid nerve injury and minimise pain

Manage the patients expectations

- **Surgery does not fix neuropathic pain**
- Most patients have pain with related functional, social and psychological sequelae
- We cannot 'fix' the patients with nerve injuries

DO NOT SIT AND WAIT for resolution

- Home check will facilitate timely urgent intervention < 24-30 hours
- Refer to resources at **Trigeminalnerve.org.uk**

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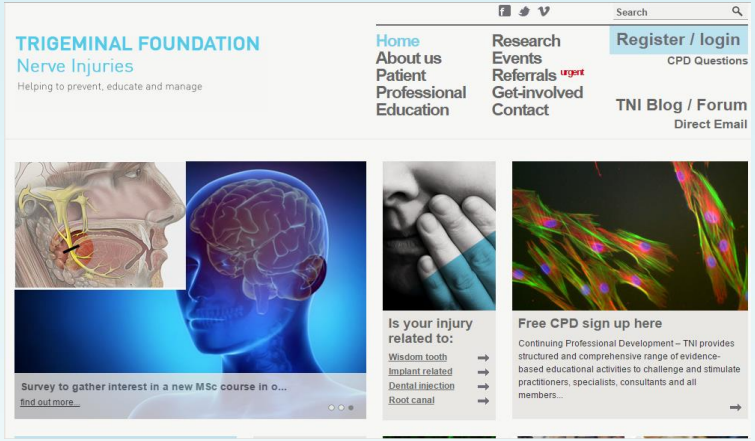
Thank you



Websites

Trigeminalnerve.org.uk
Orofacialpain.org.uk

IASP® King's College Hospital NHS NHS Trust

Thank you

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