Its not numb, its painful!
Nerve injuries in implantology

ABAOMS
Association of British Academic Oral and Maxillofacial Surgeons

Updating in Upheaval

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21st-22nd May 2021

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Learning content

Aims

- An update on post-surgical traumatic sensory nerve injuries/ neuropathy
- Outline definitions and diagnostic criteria for neuropathic pain
- Risk factors and recommendations on how to avoid nerve injuries in relation to dental implants with regard;
  - Preop planning
  - Operative execution
  - Post-operative care

Objectives

- The delegate will better understand recent developments in sensory nerve injury/ post traumatic neuropathic pain (PTNP) diagnosis and management
- The delegates will have an improved understanding of the application patient and surgical risk factors for PTNP related to dental implants
- The delegate will know what methods to employ in prevention of trigeminal nerve injury in planning, operative execution and post-operative care
- The delegate will better understand patient selection and earl post surgical care that may mitigate PTNP in relation to dental implant practice
- To know When to refer or treat.

- According to the American Academy of Implant Dentistry (AAID), around 3 million people in the United States have dental implants.
- Dental implants are also growing in popularity. The AAID state that the number of people receiving them is increasing by around 500,000 per year.

Systematic Review
Incidence of Implant nerve injuries?

- The meta-analyses revealed that the short-term
  - 10 days after implant placement **13%** (95% CI, 6%-25%)
  - longterm (1 year after implant placement) incidence was and 3% (95% CI, 1%-7%),
- For the patients who initially reported altered sensation,
  - 80% (95% CI, 52%-94%) of them would return to normal sensation within 6 months
  - 91% (95% CI, 78%-96%) of them would return to normal sensation one year after surgery.
- In terms of long-term follow-up (1 year after surgery), the incidence is much lower (3%) and most patients (91%) would return to normal sensation
- What about those patients with pain?

PLOS ONE

RESEARCH ARTICLE

Systematic Review and Meta-Analysis on Incidence of Altered Sensation of Mandibular Implant Surgery

Chia-Shu Lin[1]*, Shih-Yun Wu[2,1], Hsin-Yi Huang[3], Yu-Lin Lai[1,4]

1 Department of Dentistry, School of Dentistry, National Yang-Ming University, Taipei, Taiwan, 2 Division of

Metanalysis Incidence Implant Nerve injuries

- 1589 articles; a total of nine articles were selected for the meta-analysis.
- The risk of neurosensory disturbance **13.50/100 person-years** (95% confidence interval (CI): 10.98–16.03),
- Greater risk with anteriorly placed implants: −0.02 (95% CI: −0.21–0.16) (*P* = 0.05).
- The overall recovery rate was estimated at **51.30/100 person-years** (95% CI: 31.2–71.4).
- **=49% permanent**

THE JOURNAL OF INDIAN PROSTHODONTIC SOCIETY
Official Publication of Indian Prosthodontic Society

REVIEW

Year : 2020 | Volume : 20 | Issue : 1 | Page : 17-26

Incidence of neurosensory disturbance in mandibular implant surgery – A meta-analysis

Harini Padmanabhan[1], Anand V Kumar[1], K Shivashankar[2]
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[2] Department of Public Health Dentistry, Indira Gandhi Institute of Dental Sciences, Kothamangalam, Kerala, India

Aim: Implantology has been widely accepted as the mainstay treatment for rehabilitating complete and partial edentulism. However, it is associated with some failures and complications, the most concerning being neurosensory disturbance. Although neurosensory disturbance has been extensively studied, the incidence and cause remains largely variable. Thus, the aim of this systematic review and meta-analysis was to evaluate the incidence, distribution, and recovery rate of neurosensory disturbance.
Settings and Design: This systematic review was conducted as per the Preferred Reporting Items for Systematic Reviews and Meta-analyses statement. A structured literature review was conducted using the following databases: PubMed, Science Direct, Cochrane, Ovid, and Google Scholar for reports related to neurosensory disturbance experienced after implant placement in the mandible.
Statistical Analysis Used: Incidence and recovery rate for 100 person-years was calculated using the Poisson regression model. The risk difference of incidence between anterior and posterior implants was calculated with a random effects model.
Results: Electronic database search yielded 1589 articles; a total of nine articles were selected for the meta-analysis. The risk of neurosensory disturbance was estimated at 13.50/100 person-years (95% confidence interval (CI): 10.98–16.03), with a greater risk with anteriorly placed implants: −0.02 (95% CI: −0.21–0.16) (*P* = 0.05). The overall recovery rate was estimated at 51.30/100 person-years (95% CI: 31.2–71.4).
Conclusions: Within the limitations of the study, it can be concluded that mandibular implant placement is associated with a considerable risk of neurosensory disturbance. A large proportion of these patients present with spontaneous recove
necessary precautions to avoid such complications. More randomized controlled trials are required to quar
altered sensation during implant placement.

Keywords: Implant surgery, incidence, neurosensory disturbance, paresthesia

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Permanence Implant Post traumatic neuropathy

- 13% of 1331 cases implant related
- 173 cases
- **96% permanency**

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

Van der Cruyssen F, Peeters F, Gill T, De Laat A, Jacobs R, Politis C, Renton T. Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries. J Oral Rehabil. 2020 Oct;47(10):1212-1221. doi: 10.1111/joor.13058. Epub 2020 Aug 2. PMID: 32687637; PMCID: PMC7540026

DOI: 10.1111/joor.13058

ORIGINAL ARTICLE

JOURNAL OF ORAL REHABILITATION WILEY

Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries

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Abstract

Background: Post-traumatic trigeminal neuropathy (PTN) is a disturbance of function or pathological change of the trigeminal nerve branches following trauma and has an important impact on patient's quality of life (QoL).

Objectives: To provide diagnostic data on PTN and illustrate differences in aetiology, injured nerve, pain distribution, sensory profile and QoL between PTN subgroups.

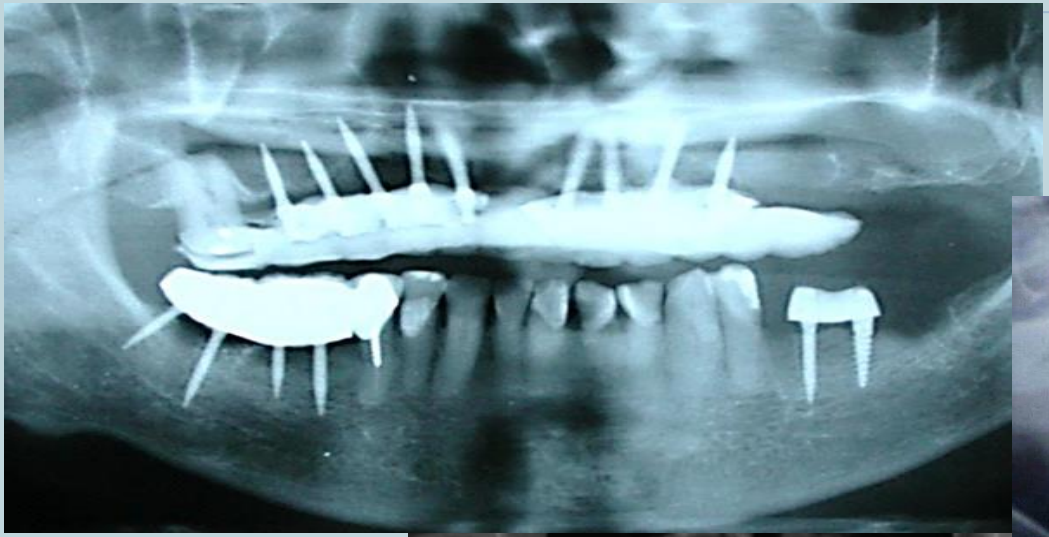
Methods: 1331 patients with painful or non-painful PTN were retrospectively re-viewed in two centres, extracting demographic data, time and cause of trauma, clinical findings including signs and symptoms, basic neurosensory testing, imaging modalities, treatments, and QoL or psychosocial assessment.

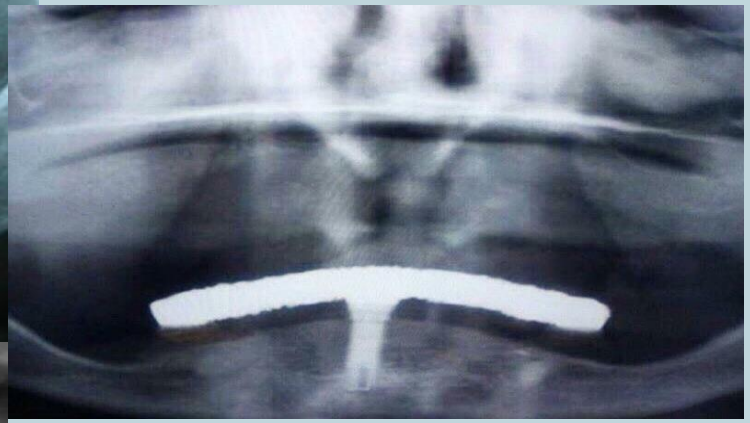
Results: More females were represented (70%) than males. The inferior alveolar nerve was most frequently damaged (60%) followed by the lingual nerve (28%). Wisdom teeth removal was considered the main cause (48%). Pain was reported in 63% of patients and pain frequency increased with age without clinically significant gender differences. Numbness was reported in 50% of PTN patients. Neurosensory testing showed larger affected dermatome involvement in persistent injuries, with no differences between the non-painful and painful PTN groups. Patient clustering indicated different sensory profile distributions when stratified according to aetiol-ogy or affected nerve branch. High interference with lifestyle was reported (78%), and patients suffering from painful PTN had worse QoL and psychosocial outcomes.

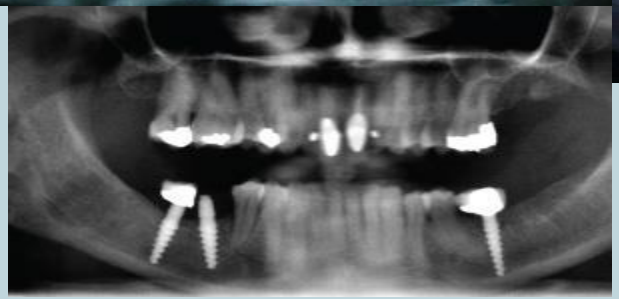
Conclusion: Patients with painful PTN had different clinical profiles and lower QoL scores than those with non-painful PTN. Sensory profiles may provide important prognostic and therapeutic information; however, more research is needed to assess the clustering procedure and link these clusters to therapeutic guidelines.

KEYWORDS
diagnosis, neuropathic pain, quality of life, trigeminal nerve, trigeminal nerve disorder

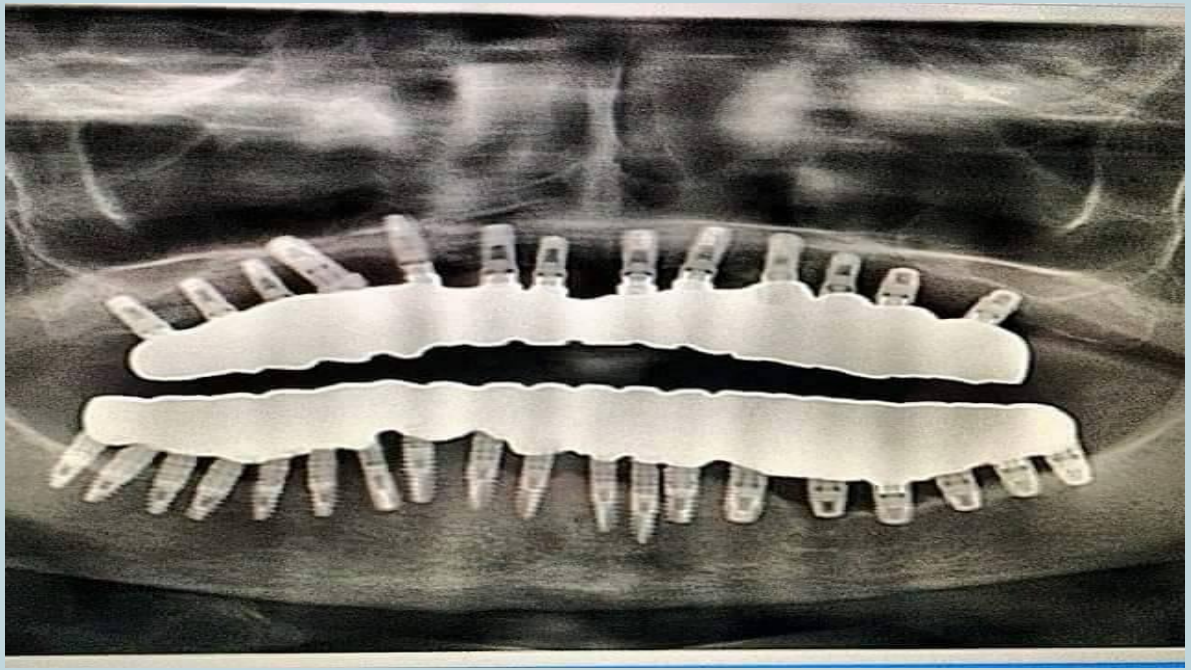
- There are three kinds of dental surgeons…….



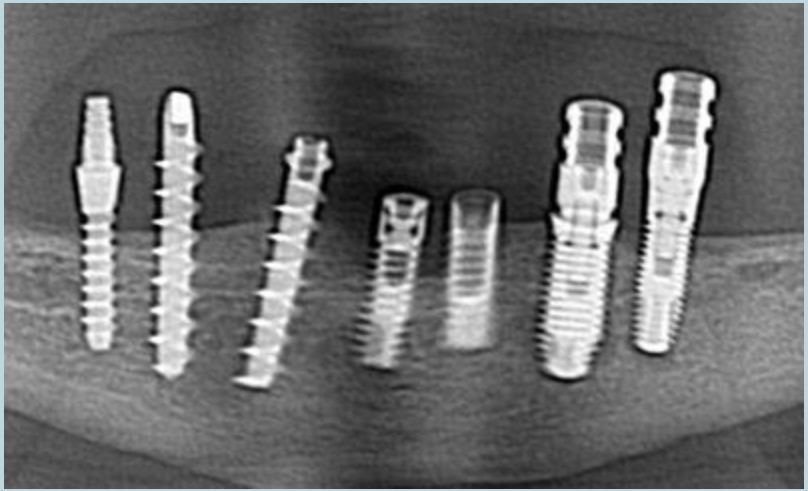


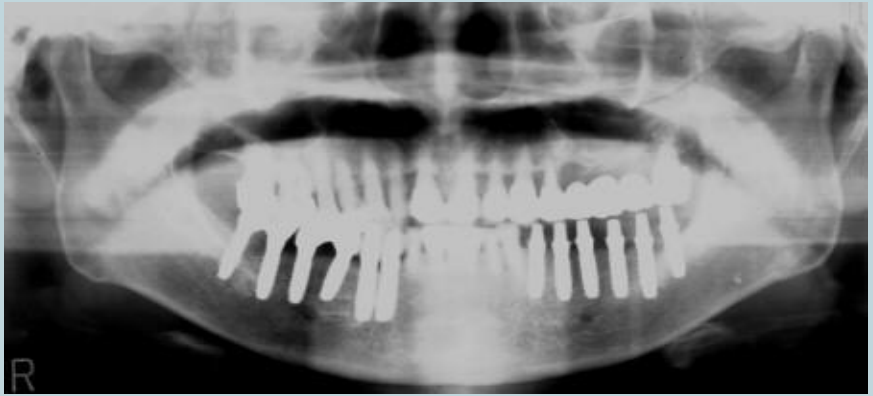


The Pessimists



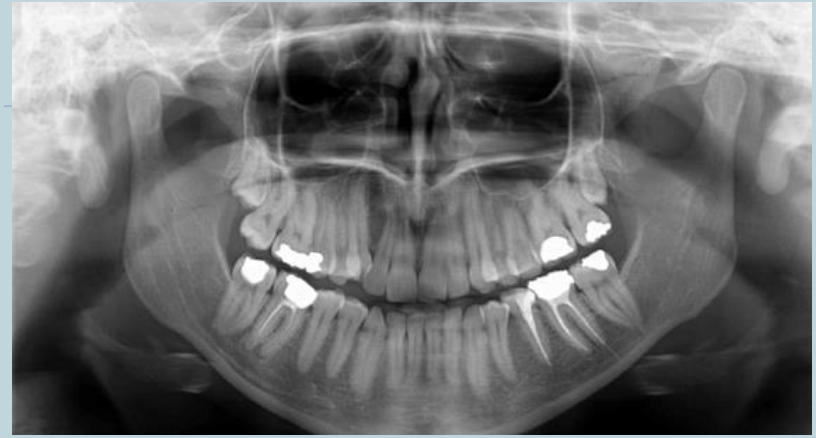
And the undecided.....

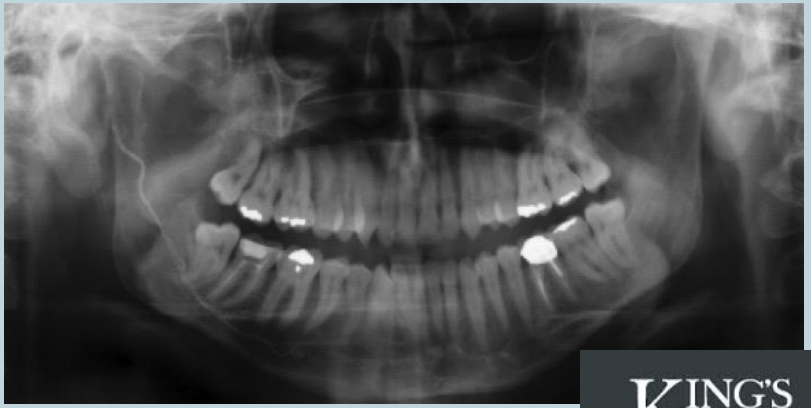




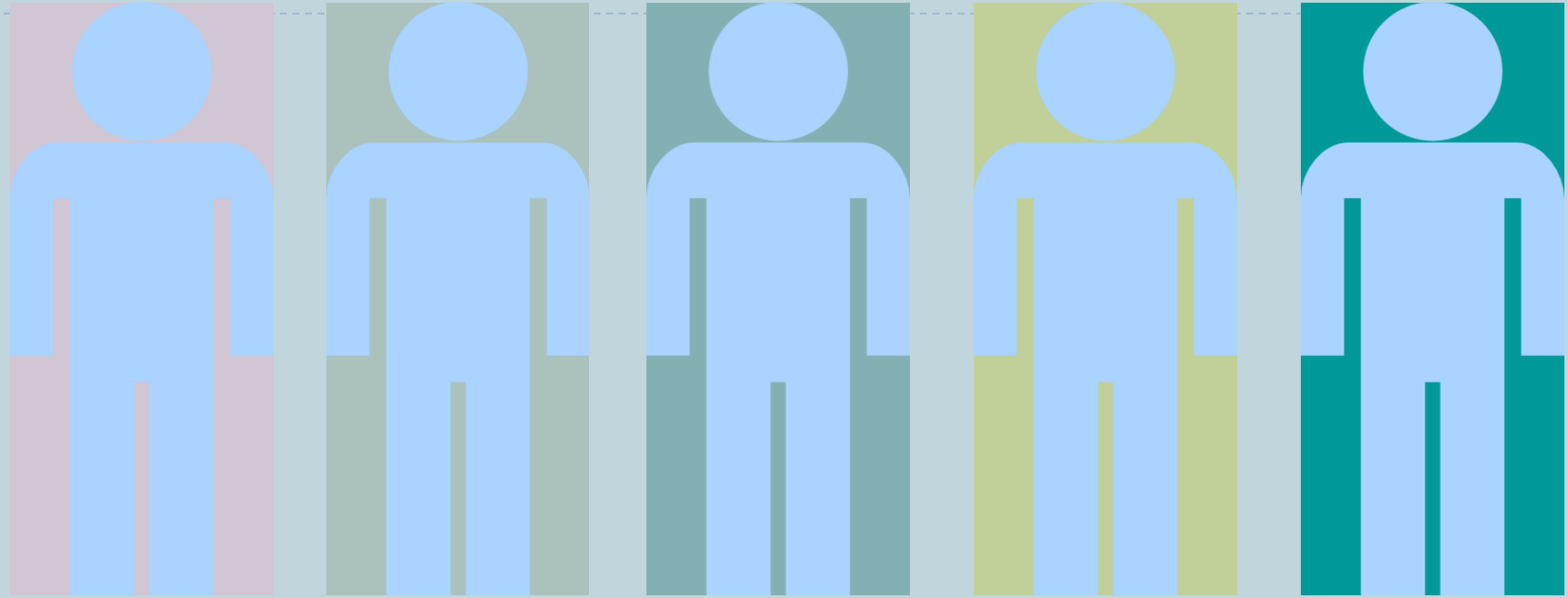
However.....

- All dentists are optimists when considering the outcome of ~~nerve injuries~~ Post traumatic neuropathy in their own patients!





Overview



Consequences of Trigeminal nerve injuries PTN

Neuropathic pain and diagnosis and assessment

Implant PTN related risk factors

Prevention of Implant related PTN

Management of patients with Implant related PTN

Overview

Consequences of Trigeminal nerve injuries PTN

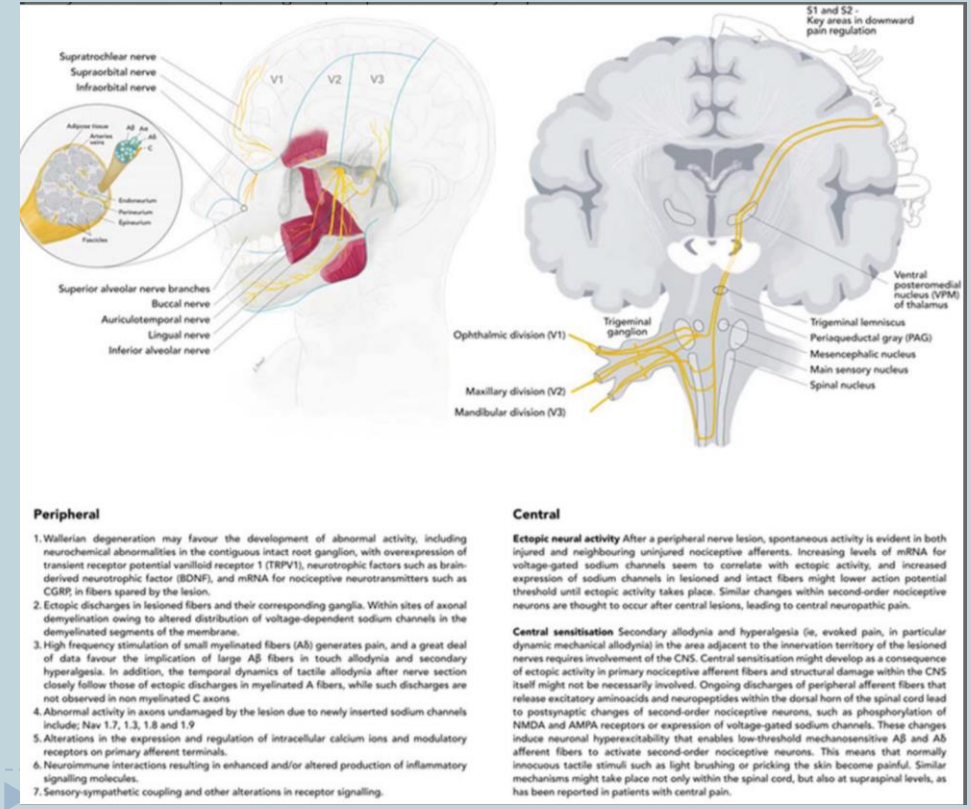
Neuropathic pain and diagnosis and assessment

Implant PTNP related risk factors

Prevention of Implant related PTN

Management of patients with Implant related PTN

Trigeminal nerve injury (PTN)



Peripheral

1. Wallerian degeneration may favour the development of abnormal activity, including neurochemical abnormalities in the contiguous intact root ganglion, with overexpression of transient receptor potential vanilloid receptor 1 (TRPV1), neurotrophic factors such as brain-derived neurotrophic factor (BDNF), and mRNA for nociceptive neurotransmitters such as CGRP, in fibers spared by the lesion.
2. Ectopic discharges in lesioned fibers and their corresponding ganglia. Within sites of axonal demyelination owing to altered distribution of voltage-dependent sodium channels in the demyelinated segments of the membrane.
3. High frequency stimulation of small myelinated fibers (Aδ) generates pain, and a great deal of data favour the implication of large Aβ fibers in touch allodynia and secondary hyperalgesia. In addition, the temporal dynamics of tactile allodynia after nerve section closely follow those of ectopic discharges in myelinated A fibers, while such discharges are not observed in non myelinated C axons
4. Abnormal activity in axons undamaged by the lesion due to newly inserted sodium channels include; Nav 1.7, 1.3, 1.8 and 1.9
5. Alterations in the expression and regulation of intracellular calcium ions and modulatory receptors on primary afferent terminals.
6. Neuroimmune interactions resulting in enhanced and/or altered production of inflammatory signalling molecules.
7. Sensory-sympathetic coupling and other alterations in receptor signalling.

Central

Ectopic neural activity After a peripheral nerve lesion, spontaneous activity is evident in both injured and neighbouring uninjured nociceptive afferents. Increasing levels of mRNA for voltage-gated sodium channels seem to correlate with ectopic activity, and increased expression of sodium channels in lesioned and intact fibers might lower action potential threshold until ectopic activity takes place. Similar changes within second-order nociceptive neurons are thought to occur after central lesions, leading to central neuropathic pain.

Central sensitisation Secondary allodynia and hyperalgesia (ie, evoked pain, in particular dynamic mechanical allodynia) in the area adjacent to the innervation territory of the lesioned nerves requires involvement of the CNS. Central sensitisation might develop as a consequence of ectopic activity in primary nociceptive afferent fibers and structural damage within the CNS itself might not be necessarily involved. Ongoing discharges of peripheral afferent fibers that release excitatory aminoacids and neuropeptides within the dorsal horn of the spinal cord lead to postsynaptic changes of second-order nociceptive neurons, such as phosphorylation of NMDA and AMPA receptors or expression of voltage-gated sodium channels. These changes induce neuronal hyperexcitability that enables low-threshold mechanosensitive Aβ and Aδ afferent fibers to activate second-order nociceptive neurons. This means that normally innocuous tactile stimuli such as light brushing or pricking the skin become painful. Similar mechanisms might take place not only within the spinal cord, but also at supraspinal levels, as has been reported in patients with central pain.

ORAL SURGERY

Oral Surgery ISSN 1752-2471

INVITED REVIEW

Diagnosis, pathophysiology, management and future issues of trigeminal surgical nerve injuries

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Key words:
chronic postsurgical pain, neuropathic pain, painful post-traumatic trigeminal neuropathy, post-traumatic trigeminal neuropathic pain, trigeminal nerve injury

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Abstract

The trigeminal nerve constitutes the largest sensory cortex representation in the brain compared with other sensory nerves. This is likely due to the fact that the trigeminal nerve underpins our very existence, as it sensorially protects, our five senses including the organs that provide sight, smell, taste, hearing, speech and meninges protecting our brain. Thus, when trigeminal nerve injuries occur, which in the main are preventable and painful, the majority of patients experience mixed symptoms including altered sensation, numbness and ongoing or elicited neuropathic pain. These neuropathic features cause significant impact on the patients' ability to function, for example cold allodynia prevents the patient enjoying cold foods and drinks and undertaking out-door activities or mechanical allodynia frequently interferes with eating, speaking, kissing and sleep. The resultant chronic symptoms and functional impedance result in significant psychological morbidity. Prevention of nerve injuries related to local anaesthesia (LA), endodontics, implants and third molar surgery is imperative as there is no magic bullet to repair these sensory nerve injuries with their related neuropathic pain. Some causes have higher levels of resolution (third molar surgery and LA) some lower levels of resolution (implant surgery and endodontics) and many patient factors will dictate the prevalence of chronic neuropathic pain. The patient must have appropriate consent and their expectations managed with understanding the potential benefits and risks for their chosen interventions. The authors have aimed to provide an up to date evidence base for diagnosis and management of trigeminal nerve injuries.

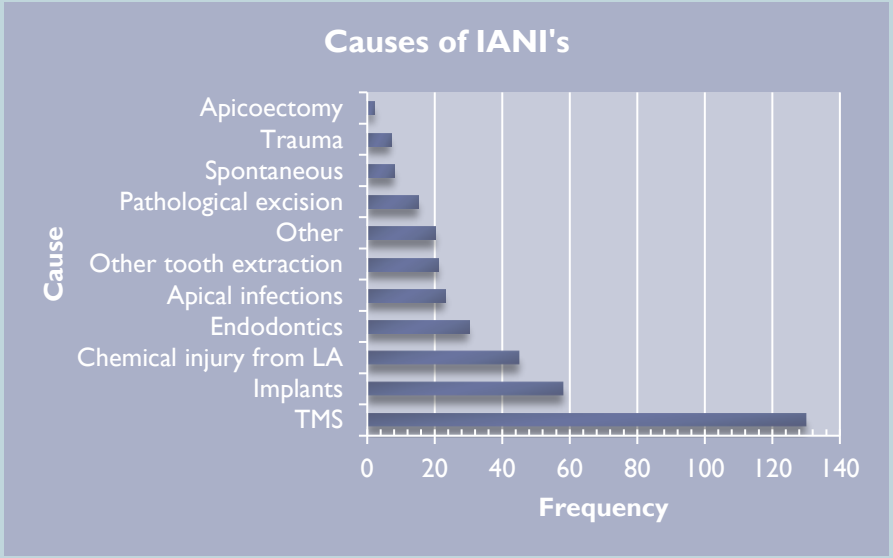
Background

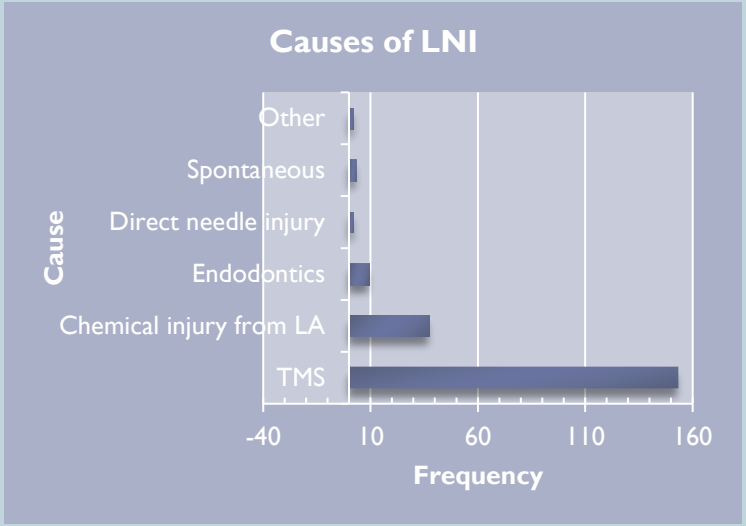
Trigeminal nerve injury (TNI) and subsequent post-traumatic trigeminal neuropathic pain (PTNP), is a problematic consequence of dental or oromaxillofacial surgical procedures with major medico-legal implications.[1] The incidence of lingual nerve injury has remained static in the UK over the last 30 years, but is increasing in the US, as is the incidence of inferior alveolar nerve (IAN) injury in the UK; the latter being due to implant surgery and endodontic therapy.[2] Trigeminal nerve injuries are generally classified as temporary but can persist and become permanent (by definition after 3 months). Based upon the limited evidence base, nerve injuries caused by implant and endodontic treatments are mainly painful and permanent.[3] Temporary nerve injuries are more likely related to local anaesthesia (LA) or third molar surgery, with mandibular related surgery patients are advised that the rate of permanent inferior alveolar or lingual nerve injuries occur between 0.1–2% of cases.[4,5] LA nerve injuries have a 75% likelihood of recovery.[6,7]

The fifth cranial nerve divisions two and three are the most commonly damaged, caused by implants,

https://onlinelibrary.wiley.com/journal/1752248x

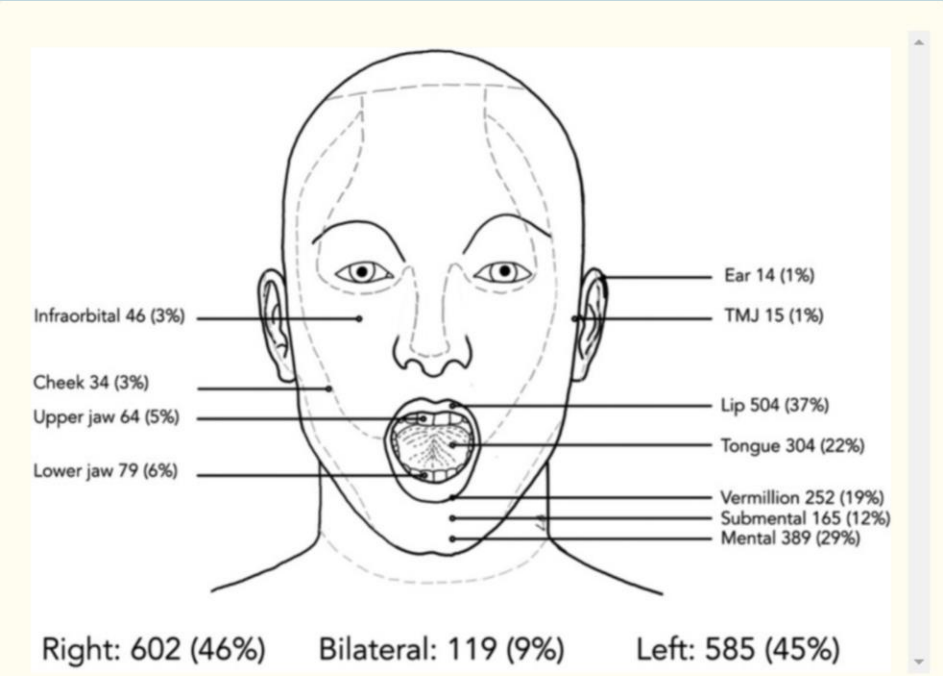
Causes of PTN in the trigeminal system(n=897)





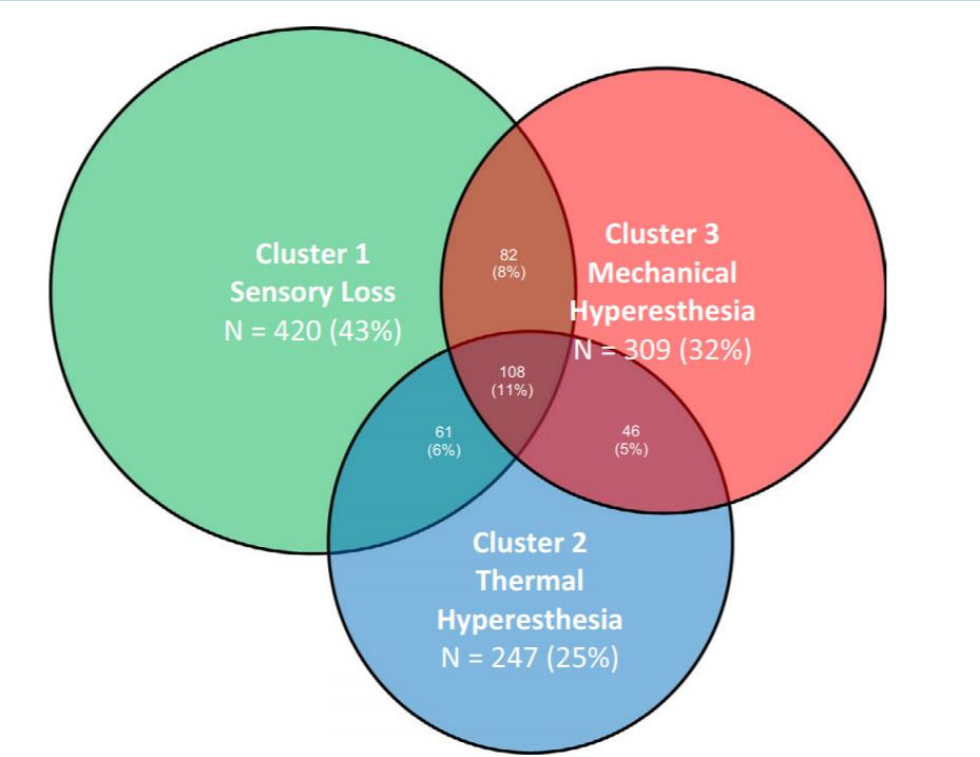
- **Summary of nerve injury patients** March 2008 –2016
- 400 IANI patients (73% F: 26.8% M; mean age = 46.5 years [range 18 – 85])
- 214 LNI patients (64.5% F: 34.6% M; mean age = 38.6 years [range 20 -73])

Distribution of PTN (N=1331)



Van der Cruyssen F, Peeters F, Gill T, De Laat A, Jacobs R, Politis C, Renton T. Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries. J Oral Rehabil. 2020 Oct;47(10):1212-1221. doi: 10.1111/joor.13058. Epub 2020 Aug 2. PMID: 32687637; PMCID: PMC7540026.

Consequences trigeminal PTN
63% of patients have pain! (n=1331)



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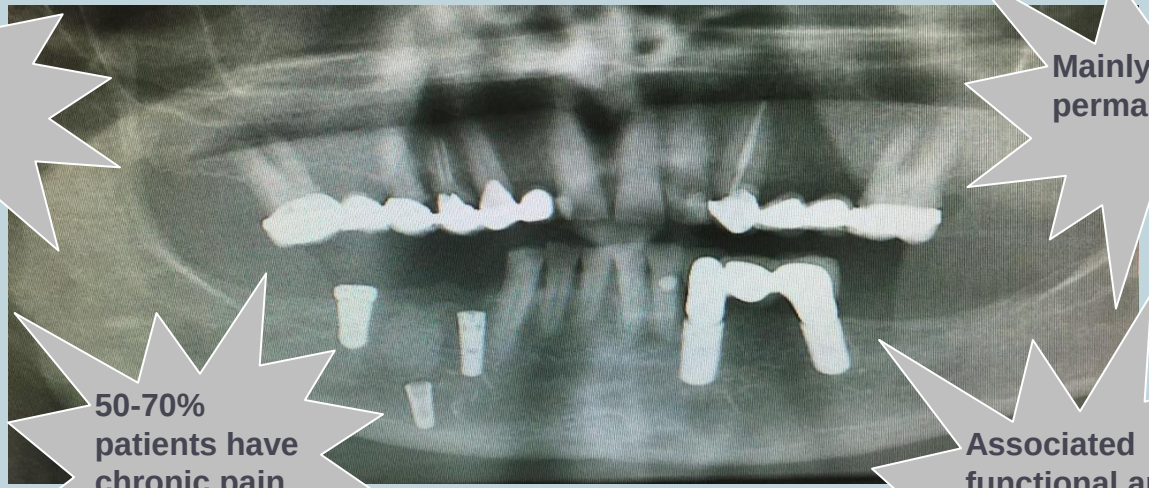
Why are nerve injuries (PTNs) such a big deal ?

Avoidable / negligent



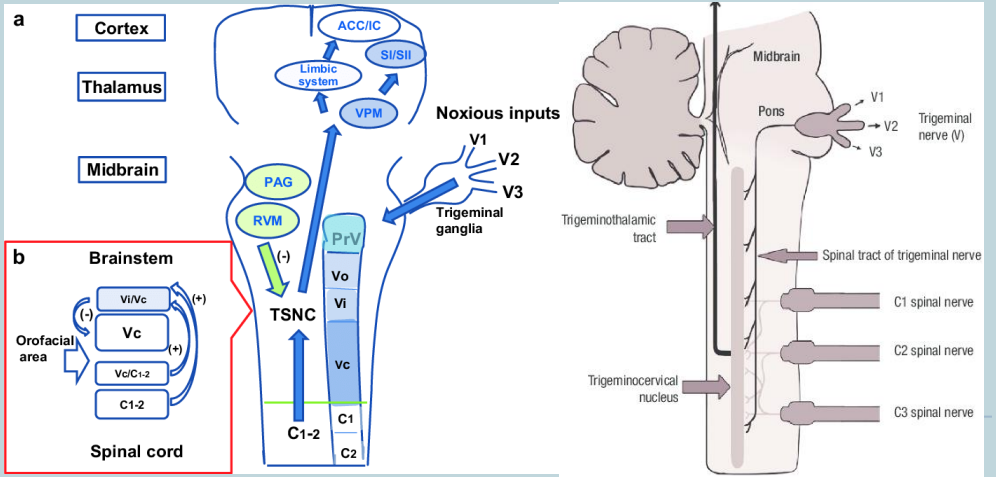
Mainly permanent

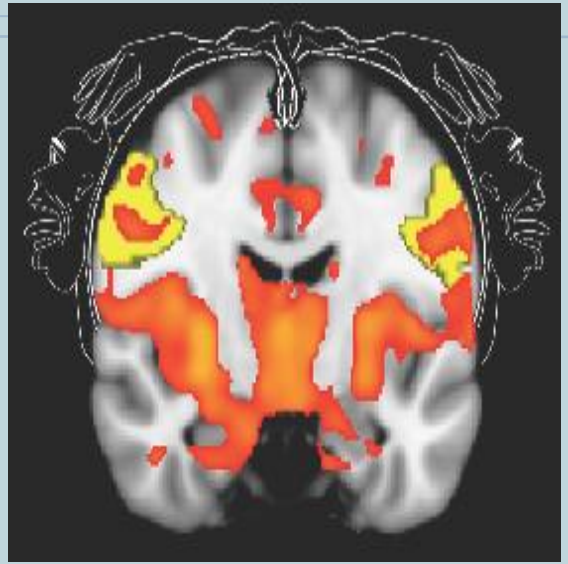
50-70% patients have chronic pain

Associated functional and psychological impact

The problem complexity of the Trigeminal nerve

- 50% Homunculus – sensory cortex
- Substantial Limbic component in V pain
- Trigemino-cervical complex
- Significant Autonomic input- Vagus
- Trigemino-vascular complex





On a Pain Scale of 1 to 10, Trigeminal Neuralgia Can Feel Like 11

Significantly higher affective component to trigeminal pain

- Noxious stimuli experienced by the head and facial region are detected and conveyed to the central nervous system (CNS) by sensory neurons located in the trigeminal (TG) ganglia, whereas noxious stimuli affecting extracranial regions are sensed and relayed to the CNS via primary sensory neurons residing in the dorsal root ganglia (DRG)

- Humans generally rank head and facial pain as much more severe and emotionally draining than body pain. For example, two of the arguably most severe chronic pain conditions are trigeminal neuralgia and cluster headaches1–3 .

- Craniofacial pain sensation is qualitatively different from bodily nociception as shown in human experiments, where repeated application of noxious heat to the face induces sensitization, yet similar stimulation applied to the hand induced habituation4 .

- Fear induced by pain in human subjects was rated higher for face than for extremities, despite comparable ratings of the pain intensity5 .
- fMRI studies further revealed that face pain resulted in higher levels of amygdala activation compared to the same intensity stimulation applied to the hand6 .

1. Waldman, SD. Atlas of common pain syndromes. Elsevier Health Sciences; 2011. 2. Zakrzewska JM, Wu J, Williams MM, Phillips N, Pavitt SH. Evaluating the impact of trigeminal neuralgia. Pain. 2017 3. Smith JG, et al. The psychosocial and affective burden of posttraumatic neuropathy following injuries to the trigeminal nerve. Journal of orofacial pain. 2013; 27:293–303. [PubMed: 24171179] 4. Schmidt K, Schunke O, Forkmann K, Bingel U. Enhanced Short-Term Sensitization of Facial Compared With Limb Heat Pain. The Journal of Pain. 2015; 16:781–790. [PubMed: 26043953] 5. Schmidt K, et al. The differential effect of trigeminal vs. peripheral pain stimulation on visual processing and memory encoding is influenced by pain-related fear. NeuroImage. 2016; 134:386– 395. [PubMed: 27015710] Moulton EA, et al. Capsaicin-induced thermal hyperalgesia and sensitization in the human trigeminal nociceptive pathway: An fMRI study. NeuroImage. 2007; 35:1586–1600. [PubMed: 17407825]

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A craniofacial-specific monosynaptic circuit enables heightened affective pain

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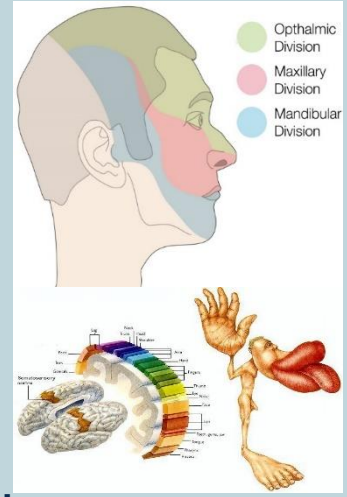
Abstract

Humans often rank craniofacial pain as more severe than body pain. Evidence suggests that a stimulus of the same intensity induces stronger pain in the face than the body. However, the underlying neural circuitry for the differential processing of facial versus bodily pain remains unknown. Interestingly, the lateral parabrachial nucleus (PB_L), a critical node in the affective pain circuit, is activated more strongly by noxious stimulation of the face than the hindpaw. Using a novel activity-dependent technology called CANE developed in our lab, we identified and selectively labeled noxious stimuli-activated PB_L neurons, and performed comprehensive anatomical input-output mapping. Surprisingly, a hitherto uncharacterized monosynaptic connection between cranial sensory neurons and the PB_L-nociceptive neurons was uncovered. Optogenetic activation of this monosynaptic craniofacial-to-PB_L projection induced robust escape/avoidance behaviors and stress calls, whereas optogenetic silencing specifically reduced facial nociception. The monosynaptic circuit revealed here provides a neural substrate for heightened

The problem of the significant burden of Trigeminal Pain

- V is the great protector
- Sensory Feedback for all craniofacial functions
  - Eyes
  - Part Meninges
  - Nose
    - Airway
  - Face
    - Expression and communication
  - Mouth
    - Breathing
    - Speaking
    - Eating



Underpins our own identity and pleasurable experiences in life

Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology
Volume 123, Issue 1, January 2017, Pages 58-66

Oral medicine

The impact of chronic orofacial pain on daily life: the vulnerable patient and disruptive pain

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JOURNAL OF ORAL REHABILITATION

REVIEW | Open Access

The impact of oro-facial pain conditions on oral health-related quality of life: A systematic review

Ibrahim Oghli, Thomas List, Naichuan Su, Birgitta Häggman-Henrikson

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Scandinavian Center for Orofacial Neurosciences: http://www.sconresearch.eu/
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Consequences
Pathophysiological consequences of nerve injury

Sensory nerve damage causes various symptoms because sensory nerves have a broad range of functions.

- Damage to large sensory fibers harms the ability to feel vibrations and touch, especially in the hands and feet. You may feel as if you are wearing gloves and stockings even when you are not. This damage may contribute to the loss of reflexes (as can motor nerve damage). Loss of position sense often makes people unable to coordinate complex movements like walking or fastening buttons or maintaining their balance when their eyes are shut.
- The "small fibers" without myelin sheaths (protective coating, like insulation that normally surrounds a wire) include fiber extensions called axons that transmit pain and temperature sensations.
- Small-fiber polyneuropathy can interfere with the ability to feel pain or changes in temperature. It is often difficult for medical caregivers to control, which can seriously affect a patient's emotional well-being and overall quality of life.
- Neuropathic pain is sometimes worse at night, disrupting sleep. It can be caused by pain receptors firing spontaneously without any known trigger, or by difficulties with signal processing in the spinal cord that may cause you to feel severe pain (allodynia) from a light touch that is normally painless. For example, you might experience pain from the touch of your bedsheets, even when draped lightly over the body.

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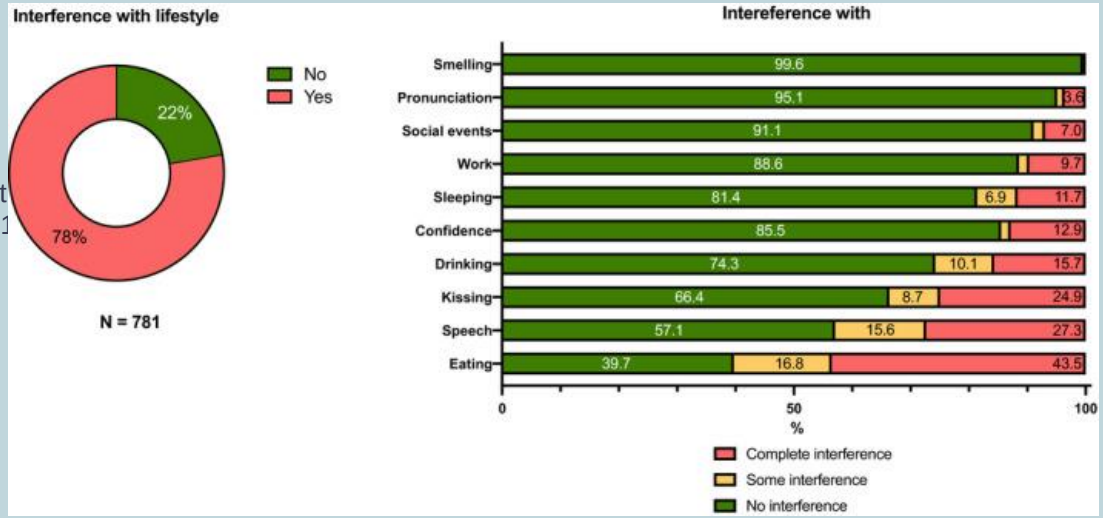
Dahlin et al. reported that nerves in rat tibia compressed at 200 to 400 mm Hg for 2 hours showed demyelination and axonal degeneration 3 weeks after compression

6 hours post ischaemic lan in IDC permanent demyelination in some neurons

Consequences
PT Neuropathy and pain causing functional problems

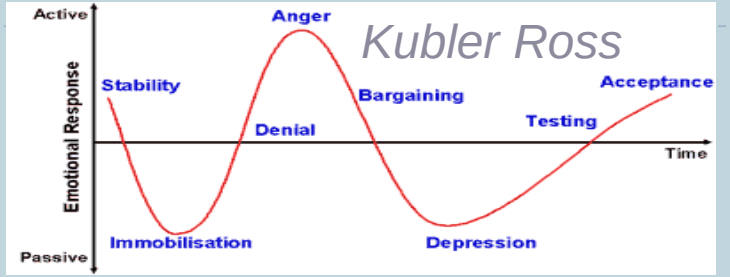
78% of patients have significant functional problems
Recent study @ KCL on 100 implant nerve injury patients
95% of implant nerve injury neuropathic pain
92% permanent
Functional and psychological impact



Renton T, Dawood A, Shah A, Searson L, Yilmaz Z. Post-implant
trigeminal nerve. A case series. Br Dent J. 2012 Jun 8;212(11):E1
10.1038/sj.bdj.2012.497

Van der Cruyssen F, Peeters F, Gill T, De Laat A, Jacobs R, Politis C, Renton T. Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries. J Oral Rehabil. 2020 Oct;47(10):1212-1221. doi: 10.1111/joor.13058. Epub 2020 Aug 2. PMID: 32687637; PMCID: PMC7540026.

Psychological consequences of Trigeminal PTN



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

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DOI: 10.1111/jop.13071
SPECIAL ISSUE ARTICLE
Journal of Oral Pathology & Medicine WILEY

The differential impact of neuropathic, musculoskeletal and neurovascular orofacial pain on psychosocial function

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Funding information
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Abstract

Background: While the psychosocial morbidity of orofacial pain (OFP) is widely recognized, the differential impact of musculoskeletal, neuropathic and neurovascular symptoms on pain and psychosocial function in individuals with and without coexisting OFP conditions is unclear.

Materials and methods: This was a comparative cross-sectional study of 350 consecutive patients attending an OFP clinic; 244 completed standardized self-report measures of pain experience, mood, and generic and oral health-related quality of life (HRQoL). The impact of musculoskeletal, neuropathic and neurovascular symptoms on measures was assessed using linear and logistic generalized linear models.

Results: Two hundred patients were diagnosed with a neuropathic condition: 125 with musculoskeletal pain and 101 with (neurovascular) headache disorders. 23% of patients presented with multiple OFP conditions; this was more common in patients with neurovascular (62%) than neuropathic (21%) and/or musculoskeletal orofacial symptoms (28%). Patients with neurovascular symptoms experienced significantly higher levels of pain, evidenced less pain self-efficacy and had poorer overall health. Neuropathic OFP was significantly associated with greater psychological and social oral health disability. Multiple OFP symptoms were not linked to pain severity or psychosocial function, although health scores were worse for patients with neurovascular pain and neuropathic/musculoskeletal symptoms compared with patients with only neurovascular symptoms.

Conclusions: The profile and degree of psychosocial morbidity in patients with OFP is significantly related to the types of presenting orofacial symptoms. Patients with neurovascular pain present with higher pain levels and have poorer health while those with neuropathic pain have higher oral functional morbidity; both may require more complex multidisciplinary management.

KEYWORDS
headache, health-related quality of life, musculoskeletal pain, neuropathic, orofacial pain, psychosocial

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

Medicolegal consequences PTN

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)

Analysis of Professional Malpractice Claims in Implant Dentistry in Italy from Insurance Company Technical Reports, 2006 to 2010

Vilma Pinchi, DDS[1]/Giuseppe Varvara, DDS[2]/Francesco Pradella, DDS[3]/ Martina Focardi, MD[4]/Michele D. Donati, DDS[5]/Gianaristide Norelli, MD[6]

. 2019 Jun;23(2):187-192.
doi: 10.1007/s10006-019-00756-3. Epub 2019 Apr 29.

Twenty four years of oral and maxillofacial surgery malpractice claims in Spain: patient safety lessons to learn

Sergio Bordonaba-Leiva[1], Esperanza I. Gómez-Durán[1], José M Baldrea[1], Josep Benet-Tuyá[1], Carlos Martín-Fumadó[1], Coro Bescos Atín[1], Javier Marrupe-Bueno[1], Josep Arimany-Manso[1]
Affiliations expand

PMID: 31037563

Review

J Oral Maxillofac Surg. 2020 Aug;78(8):1314-1318.
doi: 10.1016/j.joms.2020.03.015. Epub 2020 Mar 23.

Characteristics of National Malpractice Claims in Oral and Maxillofacial Surgery

Yui D Ji[1], Zachary S Peacock[1], Cory M Resnick[1]

Abstract

Purpose: Insight into the causes and outcomes of malpractice claims against surgeons will help inform practitioners and may support better patient care. The purpose of this study was to characterize national malpractice claims against oral and maxillofacial surgeons (OMSs).

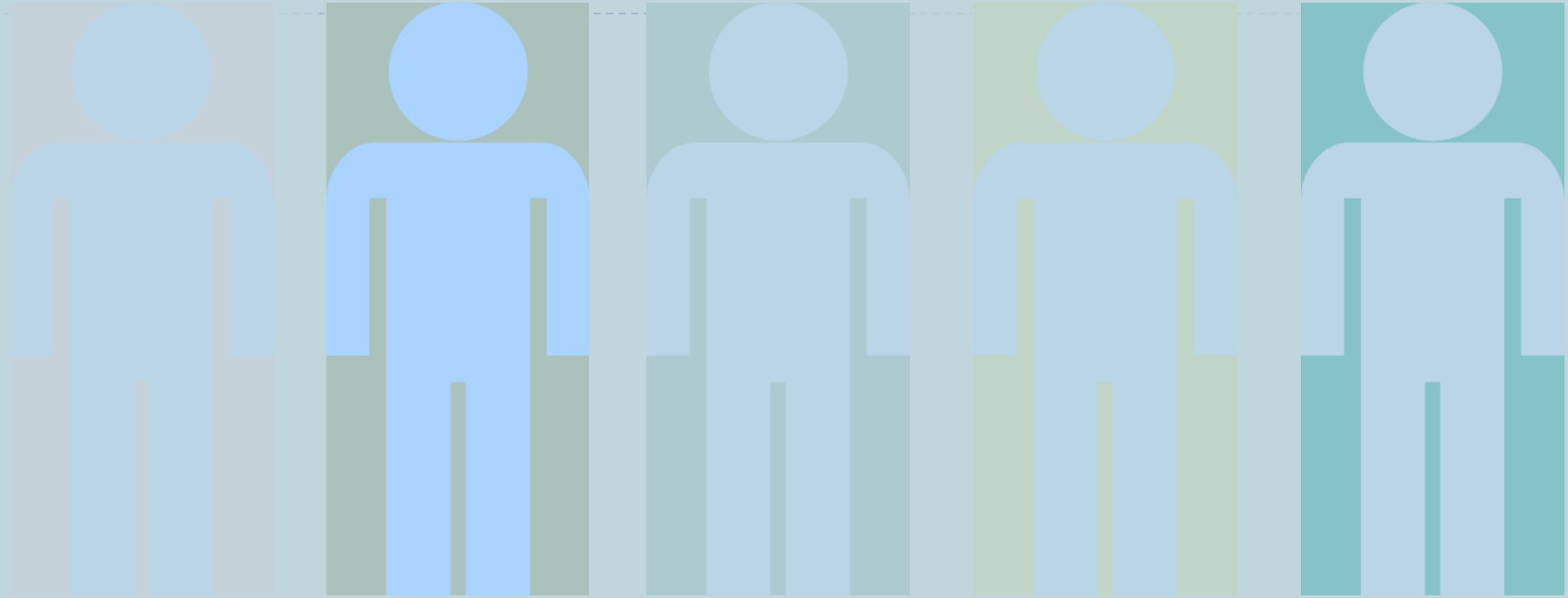
Materials and methods: A comprehensive review of all claims against OMSs from 2000 to August 2018 in the National Practitioner Data Bank was performed. Primary outcomes were claims against OMSs, payment amount, claim duration, and percentage of anesthesia-related claims. Other variables of interest included demographic characteristics, nature of allegations, clinical outcome of injury, outcome of claim, and number of payments of $1 million or greater ("catastrophic payments"). Student t tests and Wilcoxon rank-sum test were performed, and P < .05 was considered significant.

Results: This was a retrospective cohort study of malpractice claims against OMSs. There were 2,643 claims against OMSs during the study period. The average age of the claimant was 35.5 ± 18.4 years, and 47.6% were female patients. Most claims (94.7%) were settled out of court for a mean of $130,824 ± $402,633.8. Court-adjudicated claims had significantly higher payments with a mean of $247,554.69 ± $414,655.51 (P < .0001). The average

implant malpractice claims in Italy are most often settled out of court. The large number of intraoperative errors seen and the high proportion of injuries to surrounding structures suggest that implant dentists would benefit from further specific training. Also, clinical documentation vital to a defense against any claims relating to professional misconduct was incomplete or absent in more than half of the cases. Int J Oral Maxillofac Implants 2014;29:1177–1184. doi: 10.11607/jomi.3486

Ji YD, Peacock ZS, Resnick CM. Characteristics of National Malpractice Claims in Oral and Maxillofacial Surgery. J Oral Maxillofac Surg. 2020 Aug;78(8):1314-1318. doi: 10.1016/j.joms.2020.03.015. Epub 2020 Mar 23. PMID: 32305375; Castellano-Navarro JM, Castellano-Reyes JJ, Hirdina-Castilla M, Suárez-Soto A, Bocanegra-Pérez S, Vicente-Barrero M. Neurosensory issues after lateralisation of the inferior alveolar nerve and simultaneous placement of osseointegrated implants. Br J Oral Maxillofac Surg. 2019 Feb;57(2):169-173. doi: 10.1016/j.bjoms.2019.01.006. Epub 2019 Jan 31. PMID: 30712958.

Overview



Consequences of Trigeminal nerve PTN

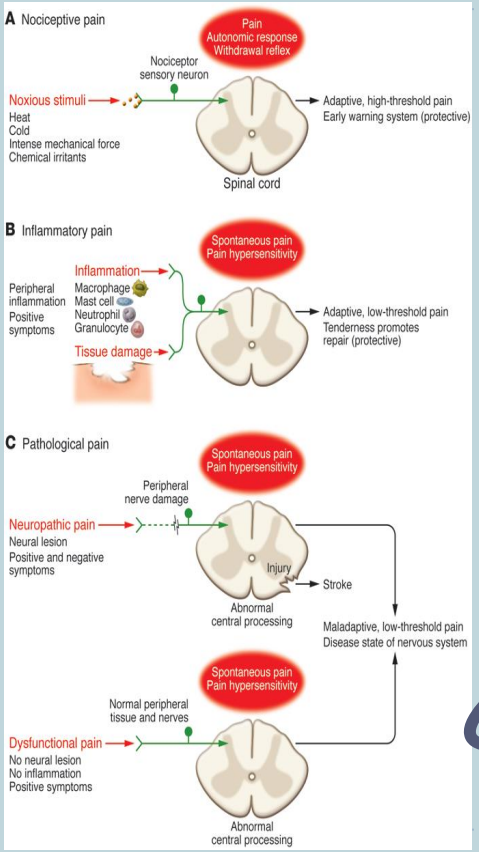
Neuropathic pain and diagnosis and assessment

Implant PTNP related risk factors

Prevention of Implant related PTN

Management of patients with Implant related PTN

Types of pain



Types of pain
Healthy acute pain

Nociceptive
healthy feeling pain 'pain'

NOCICEPTIVE PAIN

Inflammatory pain
healthy short lived after insult

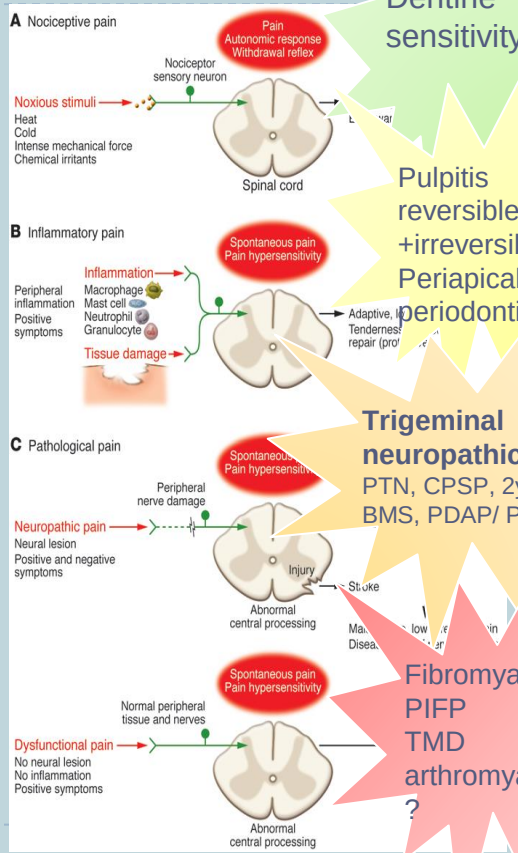
Chronic pain =
disease of neuromatrix

Neuropathic pain
Associated with nerve lesion

NEUROPATHIC PAIN

Dysfunctional or centralised pain
Unknown cause

NOCIPLASTIC PAIN



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*

Chronic pain is a disease not a symptom

Postgraduate Medicine

ISSN: 0032-5481 (Print) 1941-9260 (Online) Journal homepage: https://www.tandfonline.com/loi/ipgm20

Reframing chronic pain as a disease, not a symptom: rationale and implications for pain management

Daniel J. Clauw, Margaret Noyes Essex, Verne Pitman & Kim D. Jones

To cite this article: Daniel J. Clauw, Margaret Noyes Essex, Verne Pitman & Kim D. Jones (2019) Reframing chronic pain as a disease, not a symptom: rationale and implications for pain management, Postgraduate Medicine, 131:3, 185-198, DOI: 10.1080/00325481.2019.1574403

To link to this article: https://doi.org/10.1080/00325481.2019.1574403

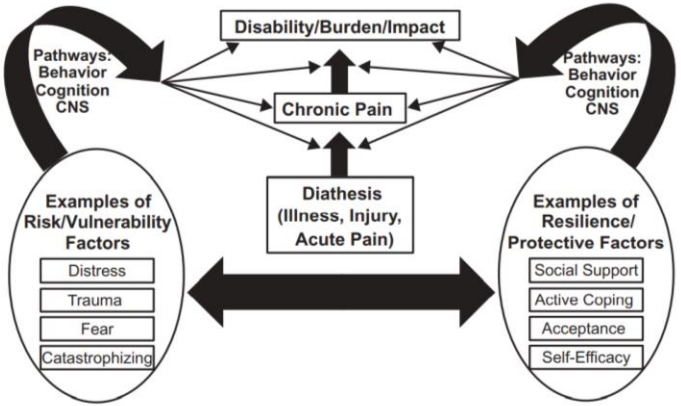


Figure 1. Impact of psychosocial factors on pain-related outcomes [24]. Reprinted from J Pain, 17(9 suppl), Edwards et al., The role of psychosocial processes in the development and maintenance of chronic pain, T70-92, © 2016 by the American Pain Society.

Table 1. Summary of standardized instruments for chronic pain assessment [133].

Measure			
Pain intensity, quality, and location	Pain interference with function	Pain interference with quality of life	Emotional distress
• Brief Pain Inventory (BPI) [142] • Pain intensity/interference with enjoyment of life/interference with general activity (PEG) [143] • PROMIS measures for pain intensity and quality [144] • Numerical Rating Scale (NRS) [145] • Verbal Rating Scale (VRS) [146] • Visual Analog Scale (VAS) [146] • Pain thermometer [147] • McGill Pain Questionnaire (MPQ) [148] • Short-form-McGill Pain Questionnaire-2 (SF-MPQ-2) [149] • Neuropathic Pain Scale (NPS) [150] • painDETECT Questionnaire (PD-Q) [140]	• BPI [142] • PEG [143] • Pain Disability Index [176] • PROMIS item banks for pain interference and behaviors [144] • Functional Independence Measure [151] • Oswestry Disability Index (ODI) [152,153] • Western Ontario MacMaster Osteoarthritis Index (WOMAC) [154] • Fibromyalgia Impact Questionnaire (FIQ) [155] or Symptom Impact Questionnaire (SIQR) [141,156] • Roland-Morris Disability Questionnaire (RDQ) [157]	• Medical Outcomes Study Short Form Health Survey (SF-36) [175] • West Haven-Yale Multidimensional Pain Inventory (WHYMPI) [158] • EuroQoL (EQ-5D) [159] • Sickness Impact Profile (SIP) [160]	• Beck Depression Inventory (BDI) [161] • Profile of Mood States (POMS) [162] • Symptom Checklist-90 Revised (SCL-90R) [163] • Pain Catastrophizing Scale (PCS) [164] • Coping Strategies Questionnaire (CSQ) [165] • Patient Health Questionnaire 9-item depression scale (PHQ-9) [166] • Generalized Anxiety Disorder 7-item anxiety scale (GAD-7) [167] • PTSD Checklist-Specific Version (PCL-S) [168] • Posttraumatic Diagnostic Scale for the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) (PDS–5) [169] • Clinician-Administered PTSD Scale (CAPS) [170] • PTSD Symptom Scale Interview for DSM-5 (PSSI-5) [171]

Definition of neuropathic pain

- Neuropathic pain is defined by IASP as pain arising as a direct consequence of a lesion or disease affecting the somatosensory system. It develops as a result of damage to, or dysfunction of, the nervous system.

- https://www.iasp-pain.org/

Biennial Review of Pain
PAIN

Neuropathic pain in the community: prevalence, impact, and risk factors

Blair H. Smith*, Harry L. Hébert, Abirami Veluchamy

1. Introduction

Neuropathic pain arises as a direct consequence of a lesion or disease affecting the somatosensory system.[87] It can be peripheral in origin, as a result of nerve injury or disease (eg, lumbar radiculopathy, postherpetic neuralgia, diabetic or HIV-related neuropathy, or postsurgical pain), or central (eg, poststroke or spinal cord injury). It is characterized by unpleasant symptoms, such as shooting or burning pain, numbness, allodynia, and other sensations that are very difficult to describe. Clinically, particularly in primary care (where time for assessment is limited), it is important to identify (possible) neuropathic pain, distinguishing it from other pain types (including nociceptive pain), as it generally fails to respond to standard analgesics (eg, nonsteroidal anti-inflammatories) but requires a different analgesic approach.[25] As all analgesics potentially cause harm as well as benefit, the distinction will promote safe and effective prescribing.

However, "definite" neuropathic pain can relatively rarely be confirmed, particularly in nonspecialist settings. According to the widely accepted grading system proposed by the International Association for the Study of Pain (IASP)'s Special Interest Group on Neuropathic Pain (NeuPSIG), this diagnosis requires (1) a history of a relevant neurological lesion or disease, and pain in a neuroanatomically plausible distribution; (2) sensory signs in the same distribution; and (3) a diagnostic test confirming the lesion or disease in the somatosensory system.[26] Diagnostic tests might include imaging (eg, magnetic resonance imaging to demonstrate nerve lesion), intraepidermal nerve fibre density measurement on skin biopsy, neurophysiological testing (eg, nerve conduction studies), or genetic testing to demonstrate a relevant hereditary disorder (eg, erythromelalgia). Note that the term "definite" in this grading system is itself relative, and the above tests do not always confirm causality.

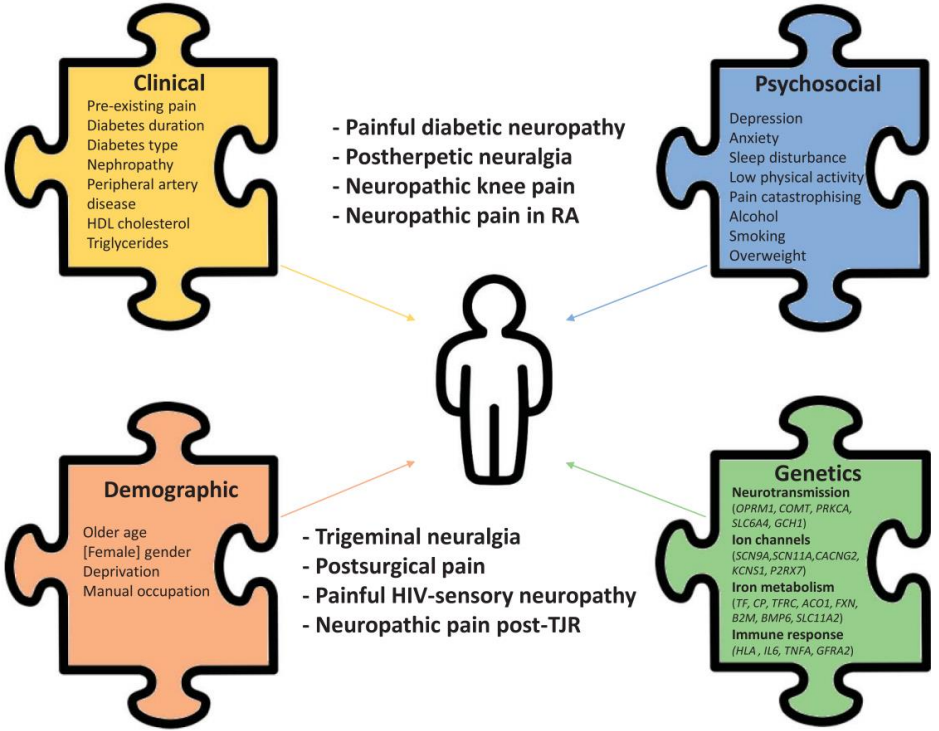


Figure 1. Summary of genetic and nongenetic factors shown to be associated with the presence and/or severity of neuropathic pain.

Genetic basis for Neuropathic Pain

Neuron
Review

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
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[5]Department of Clinical Medicine, Danish Pain Research Center, Aarhus University, Aarhus 8000, Denmark
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[10]These authors contribu
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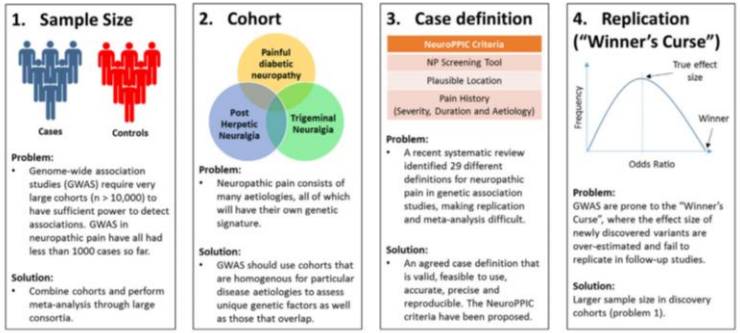
https://doi.org/10.1016/j

Neuropathic pain (N
disabling, renderin
conservation of pai



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

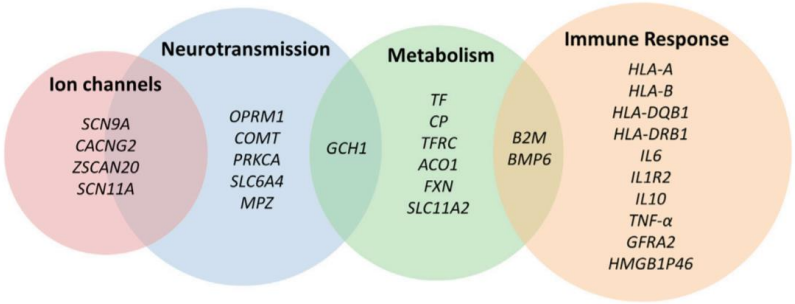


Figure 3. A Venn Diagram of Genes Reaching Study Specific or Suggestive Significance in Human Candidate Gene and Genome-wide Studies So Far in NeuP and the Overlap of Biological Pathways

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.

Neuropathic pain is increasing in prevalence!

COVID-19 and pain

Review

OPEN

PAIN REPORTS®

Potential for increased prevalence of neuropathic pain after the COVID-19 pandemic

Nadine Attal[a,*], Valéria Martinez[a,b,c], Didier Bouhassira[a,b]

Abstract

Although coronavirus disease 2019 (COVID-19) most commonly manifests with acute respiratory symptoms, one very common symptom of COVID-19 is pain. As COVID-19 often causes peripheral or central neurological complications, it is anticipated that a number of the chronic pain complications of COVID-19 will be neuropathic. This review first examines the most common viral infections responsible for neurological complications including neuropathic pain. These encompass herpes zoster, HIV, poliovirus, enteroviruses, and several tropical viruses. Neurological complications of COVID-19 including in particular Guillain–Barré syndrome, myelitis, and stroke are reviewed with regards to their potential risk of chronic neuropathic pain. Prospective longitudinal cohorts of patients should be implemented to evaluate the exact risk of neuropathic pain after COVID-19.

Keywords: COVID-19, SARS-CoV-2, Neurological complications, Neuropathic pain, Narrative review

Prevalence/ Incidence of OFP diagnoses
Common things happen commonly

Toothache Prevalence estimates for 5 case definitions identified were: 'toothache' 7-32%, 'pain in teeth with hot, cold or sweet things' **25-38%, 'pain and discomfort needing medication or treatment' 7-9%, 'pain or discomfort in the mouth, teeth or gums' 19-66%, and 'oral and facial pain'40-44%.** Pau AK, Croucher R, Marcenes W Prevalence estimates and associated factors for dental pain: a review. Oral Health Prev Dent.2003;1(3):209-20

Tension type headache Episodic TTH,occurring on fewer than 15 days per month, is reported by more than **70% of some populations.** http://www.who.int/mediacentre/factsheets/fs277/en/

Migraines 22.7% in the National Health and Nutrition Examination Survey, 16.6% of adults 18 or older reported having migraine or other severe headaches in the last 3 months in the 2011 National Health Interview Survey. In contrast, the AMPP study found an overall prevalence of migraine of 11.7% and probable migraine of 4.5%, for a total of 16.2%. Smitherman TA, Burch R, Sheikh H, Loder E.The prevalence, impact, and treatment of migraine and severe headaches in the United States: a review of statistics from national surveillance studies. Headache. 2013 Mar;53(3):427-36. doi: 10.1111/head.12074. Epub 2013 Mar 7.

Pain from TMD Males / Females **6.7% / 12.4%** Johansson et al2002

Post traumatic Painful neuropathic pain/ Chronic post surgical V pain

0.01-20% of patients undergoing third molar surgery/ **1:14-54k** post LA block / ? Post Implants

PDAP 1.6% -5%

Burning Mouth Syndrome prevalence 0.1% [Incidence over 55 years (3.7%), 11 men (1.6%) and 42 women (5.5%)] Bergdahl M Bergdahl J Burning mouth syndrome: prevalence and associated factors. J Oral Pathol Med. 1999 Sep;28(8):350-4.

Non traumatic secondary neuropathy???

Trigeminal neuralgia General population **0.1% and 0.3%,** although studies carried out in primary care settings suggest that it may be much higher, around 12% per 100,000 persons per year http://www.iasp-pain.org/files/Content/ContentFolders/GlobalYearAgainstPain2/20132014OrofacialPain/FactSheets/Trigeminal_Neuralgia.pdf

Fayaz et al 2016; Renton 2015; Nixdorf & Moano-Filho 2011;Kohorst etal 2015;Mueller et al 2015

OFP Neuropathic pain is rare but preventable in many cases

- Toothache Prevalence estimates for 5 case definitions identified were: 'toothache' 7-32%, 'pain in teeth with hot, cold or sweet things' **25-38%,** 'pain and discomfort needing medication or treatment' 7-9%, 'pain or discomfort in the mouth, teeth or gums' 19-66%, and 'oral and facial pain'40-44%. Pau AK, Croucher R, Marcenes W Prevalence estimates and associated factors for dental pain: a review. Oral Health Prev Dent.2003;1(3):209-20
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- **Post Herpetic Neuralgia**
- **Trigeminal neuralgia** General population **0.1% and 0.3%,** although studies carried out in primary care settings suggest that it may be much higher, around 12% per 100,000 persons per year http://www.iasp-pain.org/files/Content/ContentFolders/GlobalYearAgainstPain2/20132014OrofacialPain/FactSheets/Trigeminal_Neuralgia.pdf

Fayaz et al 2016; Renton 2015; Nixdorf & Moano-Filho 2011;Kohorst etal 2015;Mueller et al 2015

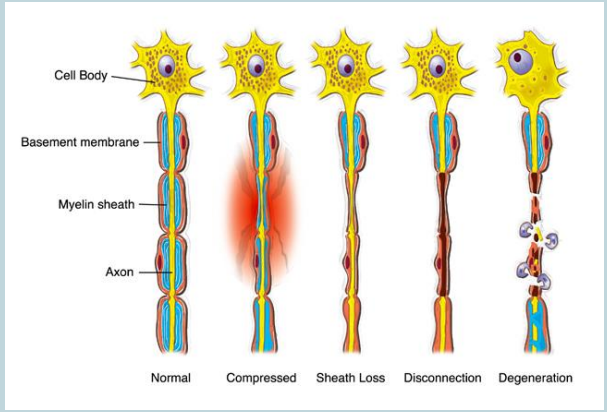
Causes of sensory neuropathy =/- pain

Trauma Surgery, LA,
mechanical Chemical, thermal, radiation

Infections
Dental abscesses close to ID
Bacterial TB Leprosy
Viral, Herpes Zoster (PHN)
HIV, Leprosy

Toxins
Chemotherapy
Heavy metals

Metabolic
Diabetes,
Hypothyroidism
Sickle cell
Acromegaly



Nutrition
Heavy metal poisoning
Vitamin deficiency
B, E

Alcoholism

Auto immune
Demyelination (Multiple sclerosis, Guillain Barre)
Connective tissue disorders
Sarcoidosis/amyloidosis

Neoplasia

Peripheral sensory neuropathy presents with:

- Anaesthesia (numbness) +
- Altered sensation (pins and needles) +
- 50-70% Pain ongoing and or elicited (allodynia, hyperalgesia, hyperpathia)

Exclude non-traumatic PTN

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

Exclude systemic causes of Peripheral neuropathy

Table 1. Causes of Peripheral Neuropathy

Cause	Type of neuropathy	Comments	Laboratory tests
Diseases			
Acquired immunodeficiency syndrome	A	Mainly sensory	Human immunodeficiency virus test
Carcinoma (paraneoplastic syndrome)	A	Usually sensory	Paraneoplastic panel (anti-Hu, anti-Yo, anti-Ri, anti-Tr, anti-Ma, and anti-CV2 antibodies)
Chronic liver disease	M	Mainly demyelinating, especially in viral hepatitis	Hepatic transaminase, bilirubin, albumin, and alkaline phosphatase levels
Critical illness neuropathy	A	Usually acute or subacute	No specific laboratory test
Diabetes mellitus	M	Chronic; axonal may predominate	Fasting blood glucose level, glucose tolerance test, A1C level
End-stage renal disease	A	—	Serum creatinine and blood urea nitrogen levels
Hypothyroidism	A	Usually acute or subacute, but can be chronic	Thyroid-stimulating hormone level
Leprosy	A	Usually sensory	Phenolic glycolipid-1 antibody, skin biopsy
Lyme disease	A	—	Lyme titers
Lymphoma	M	Mainly axonal	CBC, imaging
Monoclonal gammopathy		Usually chronic	Urine and serum protein electrophoresis with immunofixation
Amyloidosis	A	Usually sensory	
Multiple myeloma	M	Axonal damage predominates after treatment	
Plasmacytoma (osteosclerotic myeloma)	D	May have some axonal damage	
Monoclonal gammopathy of undetermined significance			
IgM	D	Most common; may have some axonal damage	
IgG or IgA	M	Demyelinating features often predominate	
Porphyria	A	Acute	Porphyrin titers
Syphilis	A	—	Rapid plasma reagin, VDRL, cerebrospinal fluid analysis
Vitamin B_6 deficiency	A	Sensory more than motor	Vitamin B_6 level
Vitamin B_{12} deficiency	A	Peripheral neuropathy is intermixed with upper motor neuron signs	CBC; vitamin B_{12} and homocysteine levels; methylmalonic acid test

continued

Peripheral Neuropathy: Differential Diagnosis and Management

HEND AZHARY, MD; MUHAMMAD U. FAROOQ, MD; MINAL BHANUSHALI, MD; ARSHAD MAJID, MD;

Table 1. Causes of Peripheral Neuropathy *(continued)*

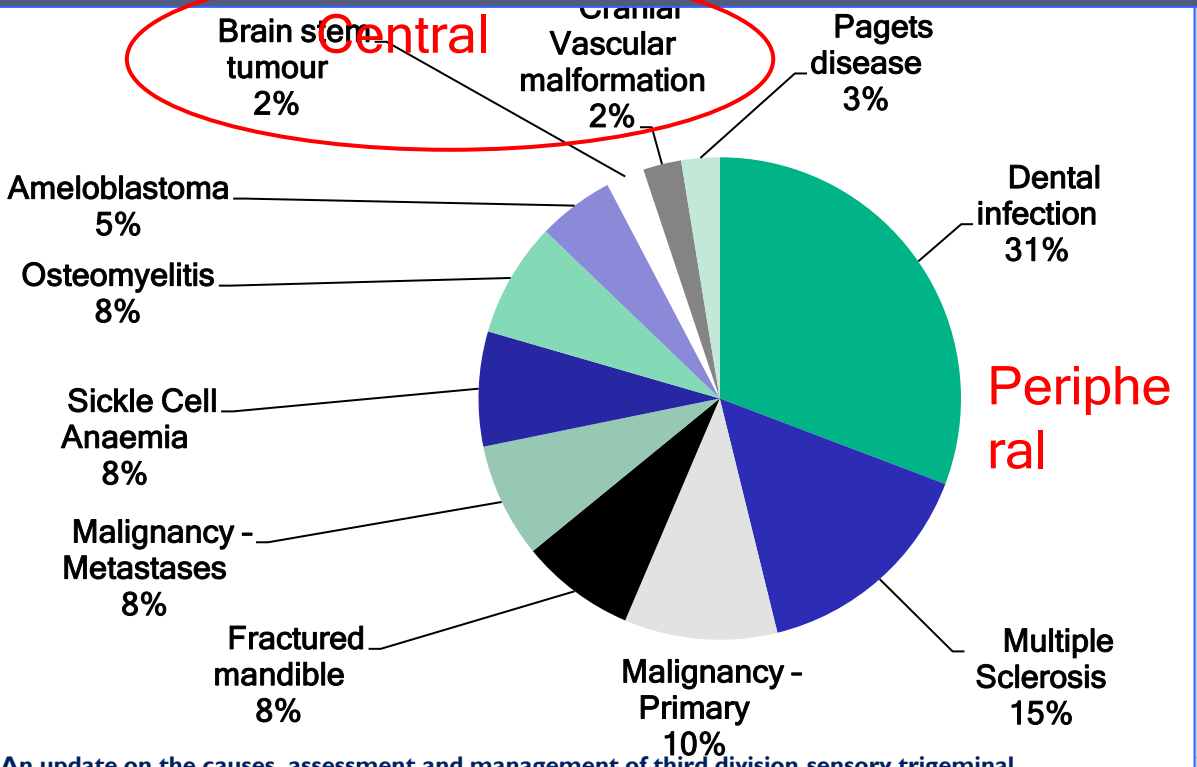
Cause	Type of neuropathy	Comments	Laboratory tests
Drugs*			
Amiodarone (Cordarone)	M	Mainly axonal with sensorimotor	No specific tests
Chloroquine (Aralen)	D	May have some axonal damage	
Digoxin	A	Mainly sensory	
Heroin	A	Sensorimotor	
Hydralazine	A	Mainly sensory	
Isoniazid	A	Mainly sensory	
Lithium	A	Sensorimotor	
Metronidazole (Flagyl)	A	Mainly sensory	
Misoprostol (Cytotec)	A	Motor	
Nitrofurantoin (Furadantin)	A	Sensorimotor	
Phenytoin (Dilantin)	A	Mainly sensory	
Procainamide (Pronestyl)	D	May have some axonal damage	
Statins	A	Mainly sensory	
Vincristine (Oncovin)	A	Sensorimotor	
Vitamin B_6 excess	A	Mainly sensory	
Genetic disorders†			
Charcot-Marie-Tooth disease			Genetic testing
Type 1	D	Also called HMSN-I	
Type 2	A	Also called HMSN-II	
Metachromatic leukodystrophy	D	—	
Neuropathy with liability to pressure palsies	D	—	
Refsum disease	D	Also called HMSN-IV	
Toxins*			
Diphtheria toxin	D	Acute presentation	Histopathology
Ethanol (alcohol)	A	Sensorimotor	No specific or practical laboratory test
Heavy metals (e.g., arsenic, lead, mercury, gold)	A	Lead and mercury mainly cause motor neuropathy Arsenic causes sensorimotor neuropathy Gold may cause some demyelination	24-hour urine collection for heavy metal titers
Organophosphates	A	Sensorimotor	No specific or practical laboratory test
Tetanus	A	Motor; acute presentation	No specific or practical laboratory test
Tic paralysis	A	Motor; acute presentation	No specific or practical laboratory test
Other causes			
Idiopathic polyneuropathy	A	Diagnosis of exclusion; usually chronic	No laboratory test

A = axonal; CBC = complete blood count; D = demyelinating; HMSN = hereditary motor-sensory neuropathy; Ig = immunoglobulin; M = mixed; VDRL = Venereal Disease Research Laboratory.

Exclude non traumatic Secondary Trigeminal PN

Trigeminal neuropathy

Retrospective analysis of the case notes of 372 patients referred to the specialist nerve injury clinic between 2007 and 2014 was carried out to establish the cause of numb chin syndrome

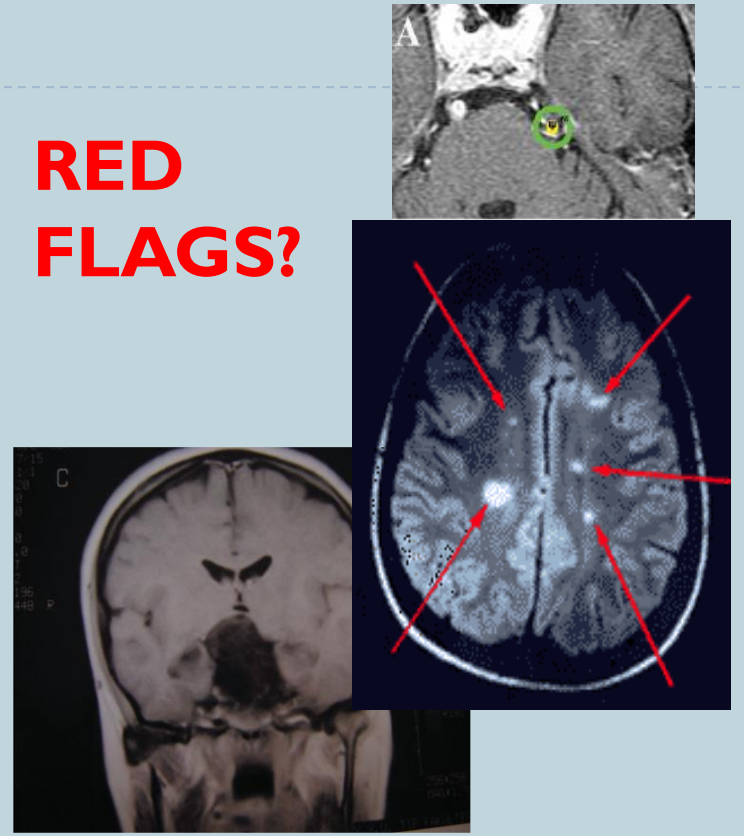


An update on the causes, assessment and management of third division sensory trigeminal neuropathies.Carter E, Yilmaz Z, Devine M, Renton T. Br Dent J. 2016 Jun 24;220(12):627-35. doi: 10.1038/sj.bdj.2016.444

Exclude Central Causes Peripheral Neuropathy

- Classical TN + NVC
  - vascular compression
- Multiple sclerosis
  - MRI plaques
- Stroke
- Vasculitis
- Central viral infection
- Tumours
  - Cervical pathology

RED FLAGS?



Exclude Neoplasia

Red Flags

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

- 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

NHS 2 (NICE 3) weeks Referral pathway

Pathophysiology of Neuropathic Pain

Damage to all sensory peripheral fibres (**Aβ, Aδ and C fibres**) alters transduction and transmission due to altered ion channel function. **Local nerve damage causes ectopic activity**

Altered spinal cord activity, leading to an excess of excitation coupled with a loss of inhibition.

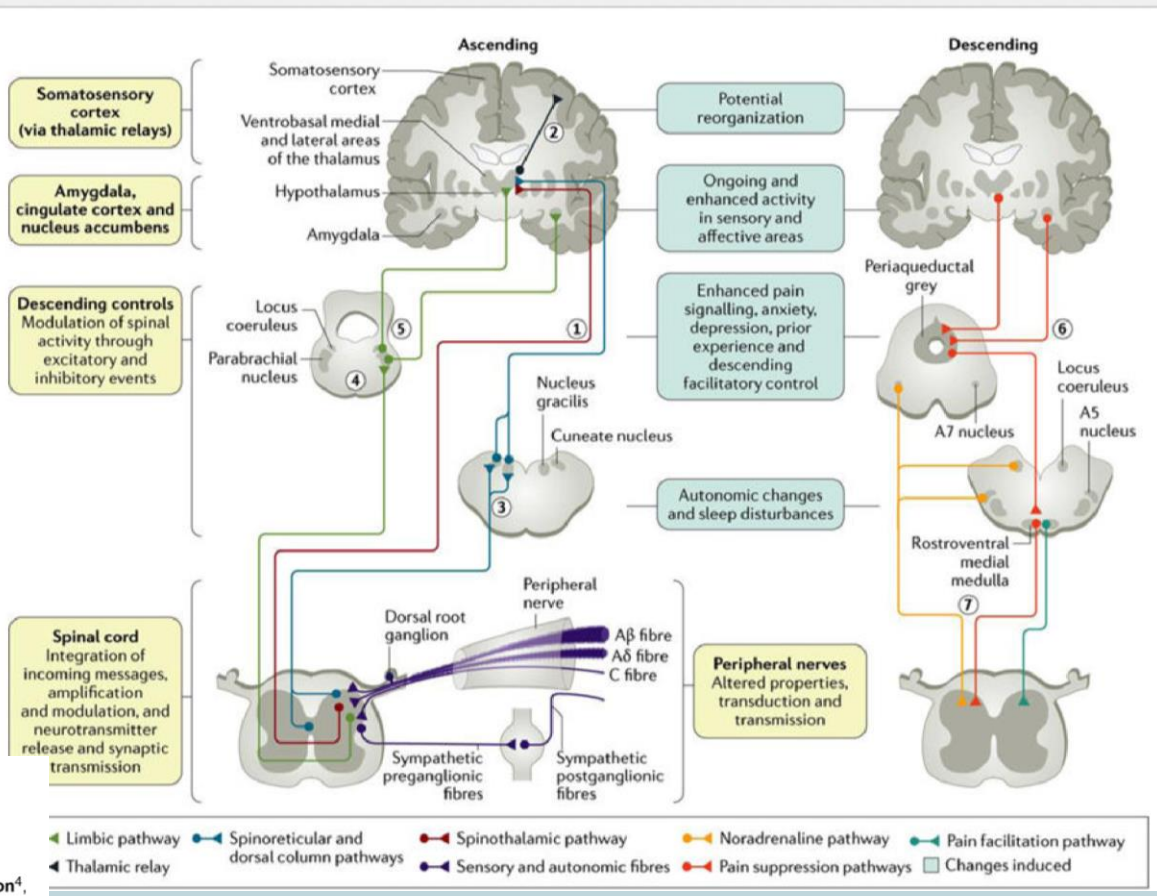
Afferent spinothalamic pathway to the **ventrobasal medial and lateral areas**

project to the **somatosensory cortex** allowing for the location and intensity of pain to be perceived

The **spinoreticular projections** and the **dorsal column pathway to the cuneate nucleus and nucleus gracilis**

Other limbic projections relay in the **parabrachial nucleus** before contacting the h**ypothalamus and amygdala, where central autonomic function, fear and «anxiety are altered**.

Descending efferent pathways from the **amygdala** and **hypothalamus** 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**



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],

Chronic post surgical pain (CPSP) is common (Is this NeP?)

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 low CPSP/ PTNP related to dentistry likely due to the use of Local Anesthesia (1.6-5% after endodontics)

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.

Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006 May 13;367(9522):1618-25. doi: 10.1016/S0140-6736(06)68700-X. PMID: 16698416.

Diagnostic Criteria PTN

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 et al,[224] and Wildgaard et al.[247]

Traumatic event = onset

Anatomically plausible site

Neuropathic area

Allodynia / Hyperalgesia = hyperaesthesia

Anaesthesia/paraesthesia = hypoaesthesia

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Focus Article

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

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‡University of Kiel, Division of Neurological Pain Research and Therapy, Department of Neurology, Kiel, Germany
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¶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.

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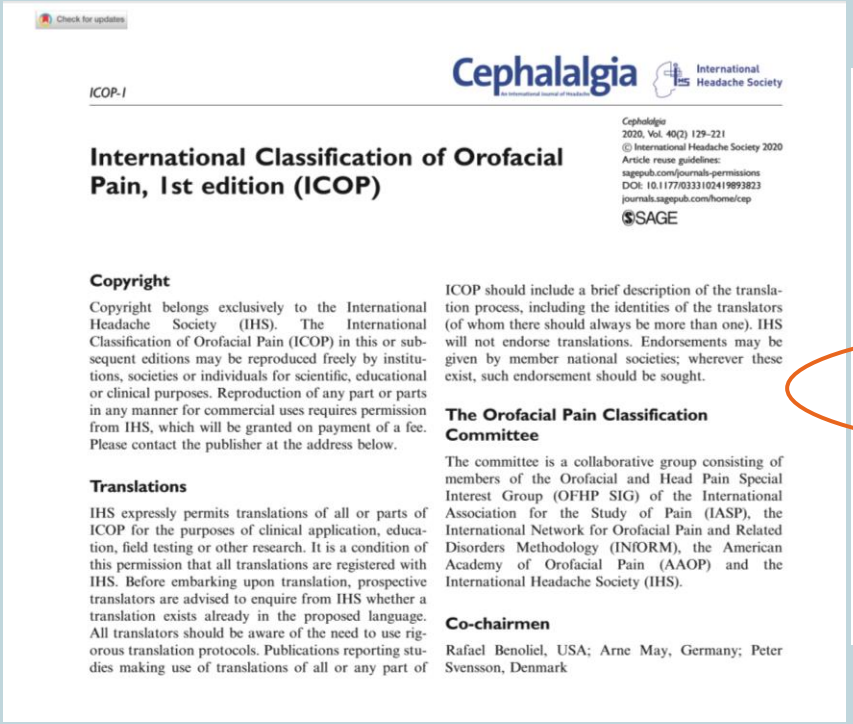
The IASP classification of chronic pain for ICD-11: chronic neuropathic pain

Joachim Scholz, 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)

International Classification orofacial pain (ICOP) Diagnostic Criteria PTNP



Check for updates

ICOP-1

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;

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.

Diagnostic algorithm for Neuropathic Pain

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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]

[1]Department of Pain and Translational Symptom Science, School of Nursing and Department of

Comprehensive Review

PAIN

OPEN

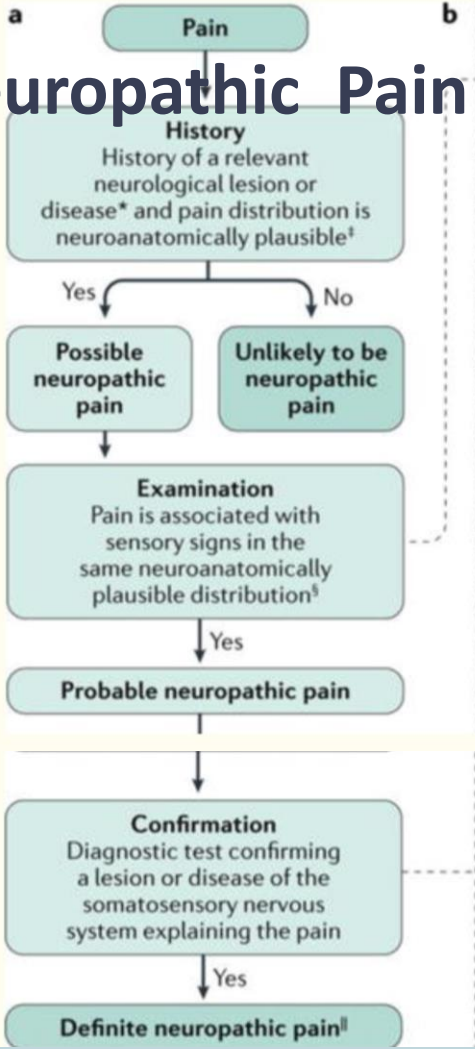
Neuropathic pain: an updated grading system for research and clinical practice

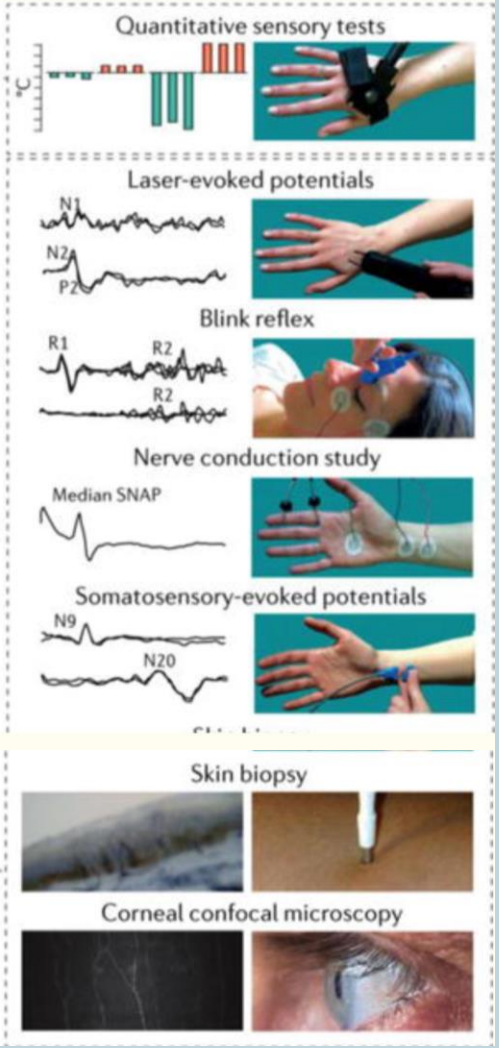
Nanna B. Finnerup[a,*], Simon Haroutounian[b], Peter Kamerman[c], Ralf Baron[d], David L.H. Bennett[e], Didier Bouhassira[f,g], Giorgio Cruccu[h], Roy Freeman[i], Per Hansson[j,k], Turo Nurmikko[l], Srinivasa N. Raja[m], Andrew S.C. Rice[n,o], Jordi Serra[p], Blair H. Smith[q], Rolf-Detlef Treede[r],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





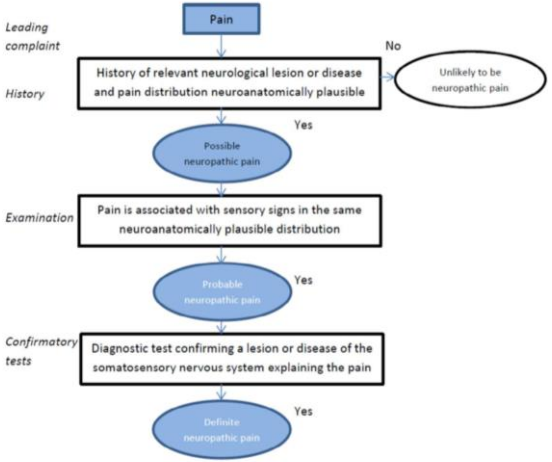
Clarifying the diagnostic criteria for V PTN

Vol. 125 No. 6 June 2018

Identifying criteria for diagnosis of post-traumatic pain and altered sensation of the maxillary and mandibular branches of the trigeminal nerve: a systematic review

Maria Devine, BDS, MFDS (RCS Ed), M Oral Surg, RCS, FHEA,[a] Murtaza
Justin Durham, BDS, MFDS (RCS Ed), PhD, FCS (OS) RCS,[b] Donald R. N
Tara Renton, PhD, MDSc, BDS, FDS, RCS, FRACDS (OMS), FHEA[a]

Objective. The aim of the study was to systematically identify criteria used to diag
Study Design. A systematic review of the literature registered in the PROSPERO



Proposed grading system for neuropathic pain (Finnerup et al 2016).

Table VI. Proposed diagnostic criteria for PPTTN

Diagnostic criteria	*Notes*
A Spontaneous or touch-evoked (stimulus dependent) pain predominantly affecting the receptive field of one or more divisions of the trigeminal nerve.	Pain tends to spread with time and is mostly unilateral without crossing the midline. Paroxysmal pain patients may also have constant background pain. Time pattern may change over the course of the disease.
Duration ranges widely from episodic (minutes to days) and may also be constant	
B Develops within 3 months of an identifiable traumatic event to the painful area or relevant innervation.	Trauma, surgery, invasive dental treatment.
Continues for >3 months.	*Usually localized pain
	†Likely to cause dermatomal pain, may spread due to central mechanisms
C At least one clinically evident neurologic dysfunction:	Must be a constant feature and reproducible. Nonvital tooth is evidence of nerve damage.
Positive sign	Clinical examination may be suitable.
- Hyperalgesia	If area is amenable, quantitative sensory testing may reveal changes.
- Allodynia	Advanced neurophysiologic testing is not always available but certainly valuable (e.g., nerve conduction studies, electromyography, laser-evoked potentials, blink reflex, masseter inhibitory reflex). Convincing data from C may be considered sufficient.
- Swelling or flushing	
And/or negative sign	
- Anesthesia	
- Hypoesthesia	
D Imaging or neurophysiology demonstrating a neurologic lesion and its location	Imaging may often be historical, e.g., zygomatic fractures affecting the infraorbital nerve that have been decompressed, dental implants that impinged on nerve bundles but may have been removed.
	Root canal therapy is considered evidence of nerve damage.
	Neurophysiology (see above)
E Not attributed to another disorder	Other causes are ruled out by history, physical examination, and special investigations, if necessary
Diagnostic level	Possible NP
Fulfils criteria A, B, and E	Probable NP

Features of Neuropathic pain

- Diagnostic features
  - **Neuropathic area either hypoaesthetic or hyperaesthetic**
  - **Allodynia**
  - **Hyperalgesa**
  - **Hyperpathia**
- Prior Multiple injuries or episodes of infection and pain
- Non-respondent to anti inflammatory pain killers (NSAIDs Paracetamol)
- Does not disturb sleep
- Better in mornings
- Worsens during day
- Worsens with stress, tiredness and illness
- Pain presentation
  - Constant burning
  - Elicited neuralgic
  - Or a combination

Table 2
Definitions of common features suggestive of neuropathic pain[29]

Paresthesia	An abnormal sensation, whether spontaneous or evoked
Dysesthesia	An unpleasant sensation, whether spontaneous or evoked
Hypoesthesia	Decreased sensitivity to stimulation (tactile or thermal; both are frequent)
Hyperesthesia	Increased sensitivity to stimulation (tactile or thermal; both are rare)
Hypoalgesia	Diminished pain response to a normally painful stimulus
Hyperalgesia	An increased response to a stimulus that is normally painful
Allodynia	Pain due to a stimulus that does not normally activate the nociceptive system

Signs and mechanisms

SYMPTOMS	PHYSIOPATHOLOGICAL MECHANISMS
Paroxysmal pain	Spontaneous activity in C-fibres
Superficial pain	Spontaneous activity in Aδ- and C-fibres
Deep pain	Spontaneous activity in articular/muscular nociceptors
Paraesthesia	Spontaneous activity in Aβ-fibres

SIGNS (EVOKED PAIN)	PHYSIOPATHOLOGICAL MECHANISMS
Cold hyperalgesia	Central sensitization/loss of central inhibition
Heat hyperalgesia	Peripheral sensitization
Punctate hyperalgesia	Central sensitization mediated by Aδ-fibres
Mechanical allodynia	Heterosynaptic central sensitization
Temporal summation of pain	Homosynaptic central sensitization
After-sensations	Homosynaptic central sensitization

Hyperaesthesia

NEUROPATHIC PAIN SYNDROMES

Coexistence of negative symptoms/signs (loss-of-function of the somatosensory system) and positive symptoms/signs (gain-of-function of the somatosensory system)

Hypoaesthesia

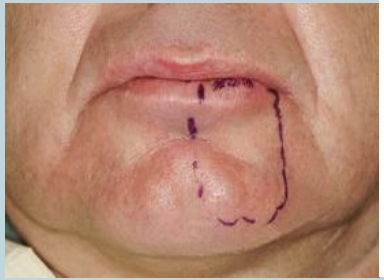
SYMPTOMS	PHYSIOPATHOLOGICAL MECHANISMS
Hypalgesia	Aδ-fibres lesion

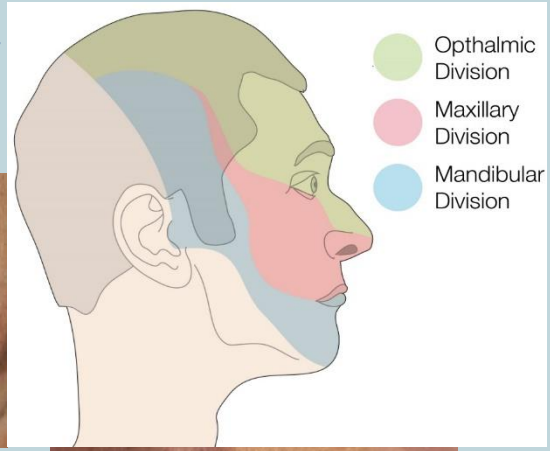
SIGNS	PHYSIOPATHOLOGICAL MECHANISMS
Tactile hypesthesia	Aβ-fibres lesion
Hypopallesthesia	Aβ-fibres lesion
Thermal hypesthesia	Aδ- and C-fibres lesion
Punctate hypesthesia	Aδ-fibres lesion

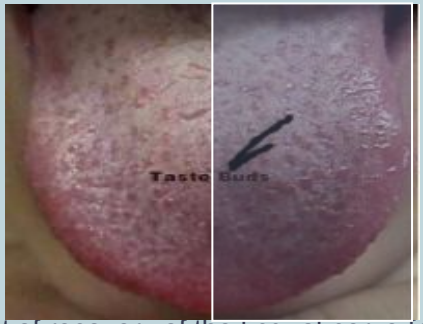
Diagnosis of nerve injury

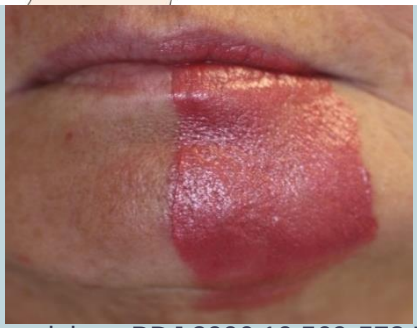
Confirm Nerve injury / Neuropathy

- Identify the extent of injury
  - Size neuropathic area
  - Subjective function
  - Mechanosensory function
  - Disability
  - Pain / discomfort
    - **Allodynia**
    - **Hyperalgesia**
    - **Spontaneous or elicited?**









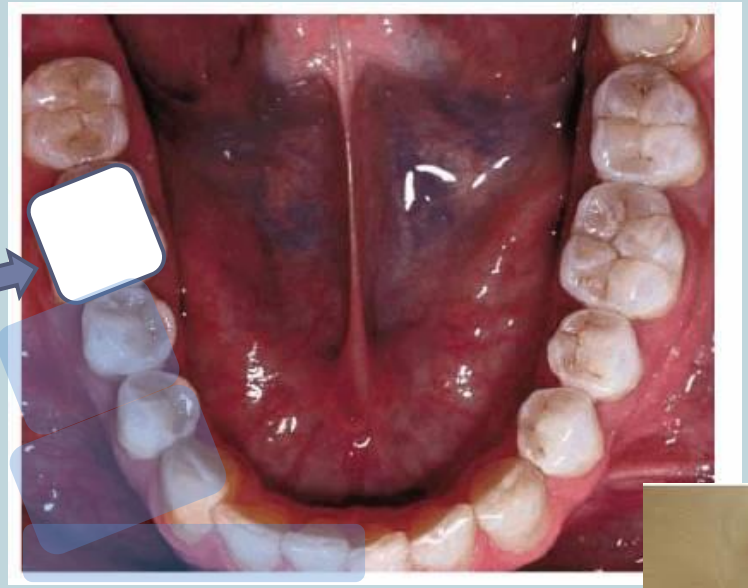
Renton T, Thexton A, SJ Crean, Hankins M. Simplifying assessment of recovery of the lingual nerve from injury. BDJ 2006 10:569-573 **Renton T**, Thexton A, Mcgurk M. New method for the objective evaluation of injury to the lingual nerve after operation on third molars.Br J Oral Maxillofac Surg. 2005 Jun;43(3):238-45. **Renton T**, Thexton A, Mcgurk M. Objective evaluation of iatrogenic lingual nerve injuries using the jaw-opening reflex. Br J Oral Maxillofac Surg. 2005 Jun;43(3):232-8

Assessment of neuropathic area
Know your anatomy!

Implant extraction or endodontic procedure undertaken with resultant numbness of mouth& lip with pain

Neuropathic area should affect 'DISTAL' domain of dermatome

In some cases only socket area can be affected with localised hypersensitivity



Neuropathic area you can use dental vitality tests but not very reliable

Extraoral area may be complete **or partial Below illustrates 40% affected**

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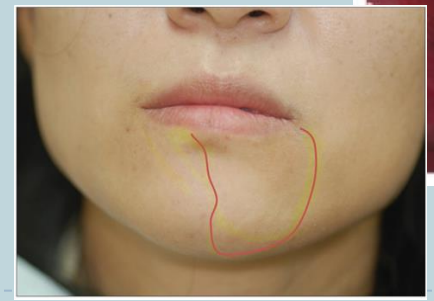
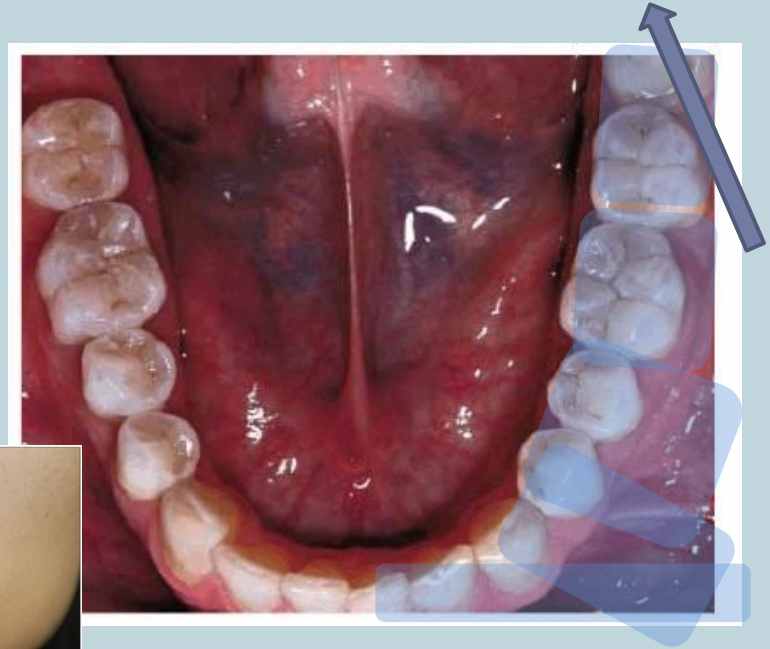
Assessment of neuropathic area
Know your anatomy!

Neuropathic area you can use dental vitality tests but not very reliable

Extraoral neuropathy affecting 9 of area0%





Inferior dental block undertaken with resultant numbness of mouth&lip with pain

Neuropathic area should affect 'DISTAL' domain of dermatome

KING'S College LONDON

Assessment of sensory Post Traumatic Neuropathy

- Pain presentation?
  - No pain
  - Ongoing paraesthesia
  - Elicited pain Allodynia
  - Ongoing deep burning pain
- Qualitative
  - Area, Light touch, sharp blunt, TPD
- Quantitative
  - Blink reflex, SEMs, nerve conduction
  - Neurophysiological
- Imaging

PAIN

Pain 117 (2005) 349–357

www.elsevier.com/locate/pain

Neurophysiologic and quantitative sensory testing in the diagnosis of trigeminal neuropathy and neuropathic pain

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Abstract

This study investigated the utility of neurophysiologic examination and thermal quantitative sensory testing (QST) in the diagnosis of trigeminal neuropathy and neuropathic pain. Fifty-eight patients (14 men), 34 with sensory deficit within the inferior alveolar nerve (IAN) and 24 within the lingual nerve (LN) distribution, were included. Twenty-six patients (45%) reported neuropathic pain. Patients underwent blink reflex (BR) test and thermal QST; sensory neurography was done to the IAN patients. Results of clinical sensory testing were available from the charts of 48 patients revealing abnormal findings in 77% of the IAN and in 94% of the LN patients. The BR test was abnormal in 41%, neurography in 96%, and QST in 91% of the IAN patients. In the LN group, BR was abnormal in 33%, and QST in 100% of the patients tested. Neurophysiologic tests and QST verified the subjective sensory alteration in all but 2 IAN patients, both with old injuries, and 4 LN patients who did not undergo QST. When abnormal, thermal QST showed elevation of warm and cold detection thresholds (hypo/anesthesia), hypoalgesia was less marked, and heat allodynia was only occasionally present. Contralateral thermal hypoesthesia after unilateral injury was found in 14 patients. It was associated with the occurrence of neuropathic pain ($P=0.016$). Axonal Aβ afferent damage was less severe in the IAN patients with pain than in those without pain ($P=0.012$). Neurophysiologic tests and thermal QST provide sensitive tools for accurate diagnosis of trigeminal neuropathy and study of pathophysiological features characteristic to human neuropathic pain.

Keywords: Inferior-alveolar-nerve; Lingual-nerve; Trigeminal-nerve; Neuropathy; Neuropathic-pain; Neurophysiologic-examination; Quantitative-sensory-

COLOR

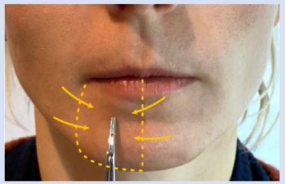
Examination protocol for mechanosensory evaluation of the extraoral dermatome of V3. This protocol could also be applied to other dermatomes.

Area affected

Using forceps run over normal to neuropathic area warning the patient that there may be hypersensitivity as well as hyposensitivity.

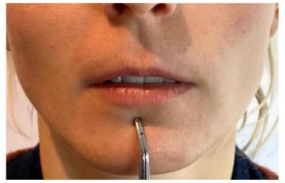
Map out the area and record pictorially or by photograph using pen marks on patient's face.
Estimate the % or extra-oral dermatome is affected by the neuropathy.

(yellow dotted lines indicate V3 dermatome and arrows indicate direction of testing from normal to neuropathic area)



Subjective function

Using forceps with beaks together firmly tap (minimum 5 times) the patient's hand several times explaining that is 'normal' 10 out of 10 subjective function. Then tap, with the same pressure, over the unaffected side of the face or tongue and repeat the stimulation explaining that should be 10 out of 10.
Move your forceps away and explain no stimulation at all is 0 out of 10.
Repeat over neuropathic area that you have already confirmed and ask the patient to report the level of stimulus according to the NRS scale below.



0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Hypoaesthesia											Hyperaesthesia									
No stimulus response										Normal function										Worst stimulus intensity imaginable

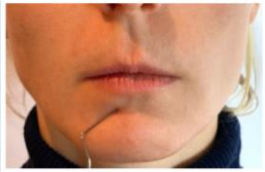
This test can be repeated over different domains of the neuropathy (lip vermillion, lip skin and chin skin or over tongue)

Light touch

To evaluate light touch thresholds von Frey filaments are highly recommended. If these are not be available, a pledget can be used instead, placing repeated (minimum 5 times) on normal side first then repeated on affected side; ask the patient to report differences. If the patient is experiencing numbness on stimulation, they will have reduced light touch detection thresholds. However, if the patient is suffering from hyperaesthesia and possible allodynia (pain on touch) this test can be very uncomfortable.

Sharp blunt discrimination

Using a dental probe sharp and blunt ends, the unaffected side is tested first. A minimum of five stimulations would be used and the number recognized by the patient (if less than 3 out of 5 then the test is negative). Whilst this test can illustrate hypoaesthesia with reduced sharp detection on the affected side, this test can also identify mechanical hyperalgesia (increased pain on sharp stimulation) which is often extremely uncomfortable for the patient. Sharp thresholds can be estimated using specially designed algometers not used in this study.



Two-point discrimination (TPD)

Using college forceps with beaks open and closed (both for five stimulations), TPD function can be estimated. Some authors prefer specially designed calipers which can be set to a specific distance. Normal TPD in the V3 dermatome extraorally ranges from 2-4mm on the lip vermillion to 6-8mm on the skin of the chin.

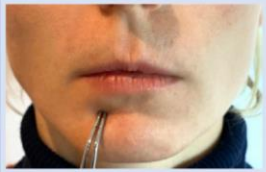


Figure 7

Oral Surgery ISSN 1752-2471

ORIGINAL ARTICLE

Diagnosis, pathophysiology, management and future issues of trigeminal surgical nerve injuries

T. Renton[1] & F. Van der Cruyssen[2,3]

[1]Department of Oral Surgery, Kings College London, Dental Hospital, Kings College Hospital Trust, London, UK
[2]Department of Oral & Maxillofacial Surgery, University Hospitals Leuven, Leuven, Belgium
[3]OMFS-IMPATH Research Group, Department of Imaging and Pathology, Faculty of Medicine, University Leuven, Leuven, Belgium

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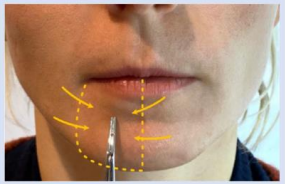

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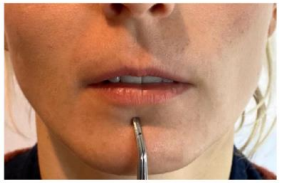

Hypo or Hyperaesthetic?

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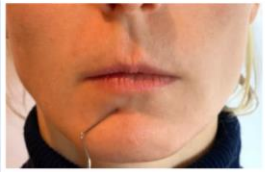
Tactile / mechanical hyperalgesia?



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Apply Cold metal mirror back Thermal allodynia

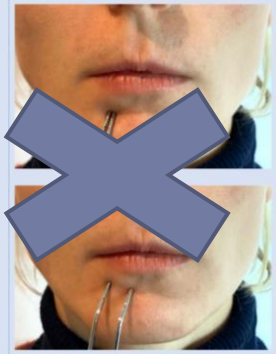

Figure 7

ORIGINAL ARTICLE

Diagnosis, pathophysiology, management and future issues of trigeminal surgical nerve injuries

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Sensory testing

Do we need Quantitative testing?

Possible Neuropathic pain-pain since event

Probable neuropathic pain (check patient not in remission)

- **Identify neuropathic area and +ve or –ve signs**
  - Mechanical and or thermal allodyn
  - Hyperalgesia
  - Hyperpathia
  - (Refractory period =TN)
- Qualitative sensory testing

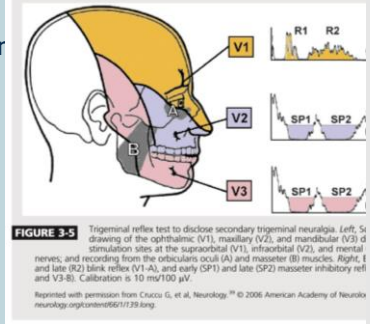

FIGURE 3-5 Trigeminal reflex test to disclose secondary trigeminal neuralgia. *Left,* S… drawing of the ophthalmic (V1), maxillary (V2), and mandibular (V3) … stimulation sites at the supraorbital (V1), infraorbital (V2), and mental … nerves; and recording from the orbicularis oculi (A) and masseter (B) muscles. *Right,* … and late (R2) blink reflex (V1-A), and early (SP1) and late (SP2) masseter inhibitory refl… and V3-B). Calibration is 10 mV/100 μV.

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Definite neuropathic pain

- Quantitative sensory testing
  - Trigeminal reflex testing is an established neurophysiologic assessment of nerve function, requires only standard nerve conduction study equipment. Blink, jaw closing, jaw opening)
  - Method of Limens thermo-sensory testing
  - Evoked potentials after electric or thermal stimuli have been studied in trigeminal neuralgia. In contrast to trigeminal reflex testing, which is normal in idiopathic or classic trigeminal neuralgia, evoked potentials may be altered, but their mean specificity of 64% is low

CONTINUUM **Review Article**

Address correspondence to Giorgio Cruccu, Department of Neurology and Psychiatry, Viale Università 30, Rome, Italy 00185, *giorgio.cruccu@uniroma1.it.*

Relationship Disclosure:

Trigeminal Neuralgia

Giorgio Cruccu, MD

Journal of Medicine and Life Vol. 6, Issue 4, October-December 2013, **pp.383-388**

Pain in trigeminal neuralgia: neurophysiology and measurement: a comprehensive review

Kumar S*, Rastogi S**, Kumar S**, Mahendra P**, Bansal M***, Chandra L**
Private Practice*
**Department of Oral and Maxillofacial Surgery and Oral Implantology, Institute of Technology and Sciences- Centre for Dental Studies and Research, Murad Nagar, Ghaziabad, India-201206
***Department of Periodontology, Institute of Dental Studies and Technologies, CCS University, Modinagar, Uttar Pradesh, India

Randomized Controlled Trial > J Oral Rehabil. 2017 Jan;44(1):30-42. doi: 10.1111/joor.12455. Epub 2016 Nov 16.

Agreement between quantitative and qualitative sensory testing of changes in oro-facial somatosensory sensitivity

J Agbaje [1], A De Laat [2], P Constantinus [1 3], P Svensson [4 5 6], L Baad-Hansen [4 5]

Affiliations + expand

PMID: 27770480 DOI: 10.1111/joor.12455

Abstract

Qualitative somatosensory testing (QualST) is a simple chairside test. It can be used to roughly assess the presence or absence of altered somatosensory function. To use QualST clinically, it is important to assess its agreement with quantitative sensory testing (QST). The aims of this study were to assess the agreement between QST and QualST when testing the modulation of facial sensitivity by capsaicin in healthy participants and to explore the agreement between QST and QualST in assessing the intraoral sensory function in clinical atypical odontalgia (AO) patients. Eighteen healthy pain-free adults and data from 27 AO patients were included in the study. Thirteen QST and three QualST parameters were evaluated at each site. Z-scores were computed for healthy participants, and Loss-Gain scores were

Sensory testing

Do we need Quantitative testing?

Possible Neuropathic pain-pain since event

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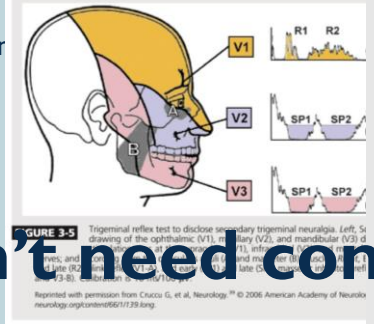

We don't need complex quantitative testing?

Definite neuropathic pain

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  - Method of Limens thermo-sensory testing
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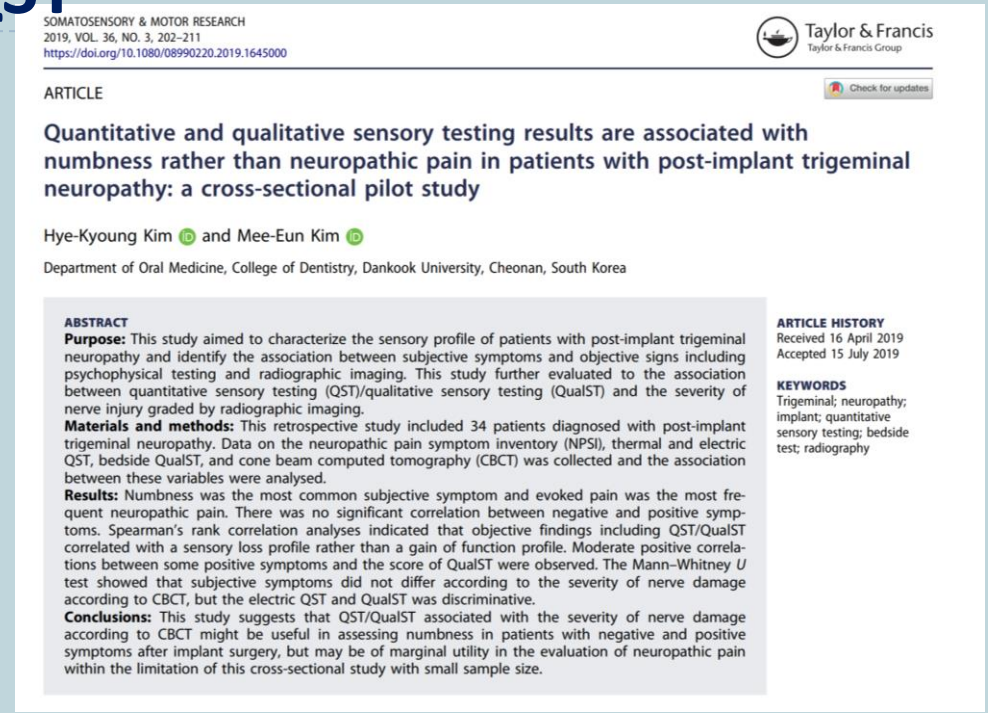
QST in implant related PTN

Self report as good as QST

- 34 patients
- Assessed using
  - QST
  - CBCT
- Numbness most common problem
- That subjective symptoms did not differ according to the severity of nerve damage according to CBCT,
- However, the electric QST and QualST were discriminative.



SOMATOSENSORY & MOTOR RESEARCH
2019, VOL. 36, NO. 3, 202–211
https://doi.org/10.1080/08990220.2019.1645000

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ARTICLE

Check for updates

Quantitative and qualitative sensory testing results are associated with numbness rather than neuropathic pain in patients with post-implant trigeminal neuropathy: a cross-sectional pilot study

Hye-Kyoung Kim and Mee-Eun Kim

Department of Oral Medicine, College of Dentistry, Dankook University, Cheonan, South Korea

ABSTRACT

Purpose: This study aimed to characterize the sensory profile of patients with post-implant trigeminal neuropathy and identify the association between subjective symptoms and objective signs including psychophysical testing and radiographic imaging. This study further evaluated to the association between quantitative sensory testing (QST)/qualitative sensory testing (QualST) and the severity of nerve injury graded by radiographic imaging.

Materials and methods: This retrospective study included 34 patients diagnosed with post-implant trigeminal neuropathy. Data on the neuropathic pain symptom inventory (NPSI), thermal and electric QST, bedside QualST, and cone beam computed tomography (CBCT) was collected and the association between these variables were analysed.

Results: Numbness was the most common subjective symptom and evoked pain was the most frequent neuropathic pain. There was no significant correlation between negative and positive symptoms. Spearman's rank correlation analyses indicated that objective findings including QST/QualST correlated with a sensory loss profile rather than a gain of function profile. Moderate positive correlations between some positive symptoms and the score of QualST were observed. The Mann–Whitney *U* test showed that subjective symptoms did not differ according to the severity of nerve damage according to CBCT, but the electric QST and QualST was discriminative.

Conclusions: This study suggests that QST/QualST associated with the severity of nerve damage according to CBCT might be useful in assessing numbness in patients with negative and positive symptoms after implant surgery, but may be of marginal utility in the evaluation of neuropathic pain within the limitation of this cross-sectional study with small sample size.

ARTICLE HISTORY
Received 16 April 2019
Accepted 15 July 2019

KEYWORDS
Trigeminal; neuropathy; implant; quantitative sensory testing; bedside test; radiography

Predicting outcome of Trigeminal PTNP

Received: 9 December 2019 | Revised: 7 May 2020 | Accepted: 10 July 2020
DOI: 10.1111/joor.13058

ORIGINAL ARTICLE

WILEY

Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries

Fréderic Van der Cruyssen[1,2] | Frederik Peeters[1,2] | Thomas Gill[3] | Antoon De Laat[4,5] | Reinhilde Jacobs[2,6] | Constantinus Politis[1,2] | Tara Renton[3]

[1]Department of Oral & Maxillofacial Surgery, University Hospitals Leuven, Leuven, Belgium
[2]OMFS-IMPATH Research Group, Department of Imaging and Pathology, Faculty of Medicine, University Leuven, Leuven, Belgium
[3]Department of Oral Surgery, King's College London Dental Institute, London, UK
[4]Department of Oral Health Sciences, KU Leuven, Leuven, Belgium
[5]Department of Dentistry, University Hospitals Leuven, Leuven, Belgium
[6]Department of Dental Medicine, Karolinska Institutet, Stockholm, Sweden

Correspondence
Fréderic Van der Cruyssen, Department of Oral & Maxillofacial Surgery, University Hospitals Leuven, Leuven, Belgium.
Email:frederic.vandercruyssen@uzleuven.be

Abstract

Background: Post-traumatic trigeminal neuropathy (PTN) is a disturbance of function or pathol
importan
Objectiv
injured n
Methods
viewed i
clinical fi
modalitie
Results:
nerve w
Wisdom
63% of p
gender d
testing s
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Conclusi
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> J Oral Maxillofac Surg. 2021 Jan 5;S0278-2391(20)31547-0. doi: 10.1016/j.joms.2020.12.049. Online ahead of print.

Degree of Compression of the Inferior Alveolar Canal on Cone-Beam Computed Tomography and Outcomes of Postoperative Nerve Injury in Mandibular Third Molar Surgery

Anton Sklavos [1], Seth Delpachitra [2], Tom Jaunay [3], Ricky Kumar [4], Arun Chandu [5]

Affiliations + expand

PMID: 33529607 DOI: 10.1016/j.joms.2020.12.049

Abstract

Purpose: Cone-beam computed tomography (CBCT) offers the advantage of a 3-dimensional representation of the anatomic relationship of the mandibular third molar tooth and the inferior alveolar canal (IAC), as compared to a panoramic radiograph. We hypothesized that a novel method of categorizing the degrees of compression of the IAC were reliable predictors for postoperative nerve injuries.

Methods: We conducted a retrospective analysis of the outcomes in third molar surgery for patients who obtained a CBCT scan in additional to a plain film radiograph over a 12 months period and underwent surgical removal of their mandibular third molars; 257 consecutive patients were identified, and 416 mandibular third molars were surgically removed.

Results: Patients who had severe compression of the inferior alveolar canal (IAC) on CBCT imaging

Journal of Oral & Facial Pain and HEADACHE
Rafael Benoliel, Editor-in-Chief

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The Diagnostic Value of Magnetic Resonance Imaging in Posttraumatic Trigeminal Neuropathic Pain

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DOI: 10.11607/ofph.2732

Aims: To evaluate the diagnostic value of non–nerve-selective MRI sequences in posttraumatic trigeminal neuropathic pain (PTNP). Methods: This study retrospectively analyzed all MRI protocols performed between February 2, 2012 and June 20, 2018 commissioned by the Department of Oral and Maxillofacial Surgery, University Hospitals Leuven. Demographic, clinical, and radiologic data were extracted from the records of patients with an MRI in the context of PTNP. A contingency table was constructed based on the opinions of the treating physician and the

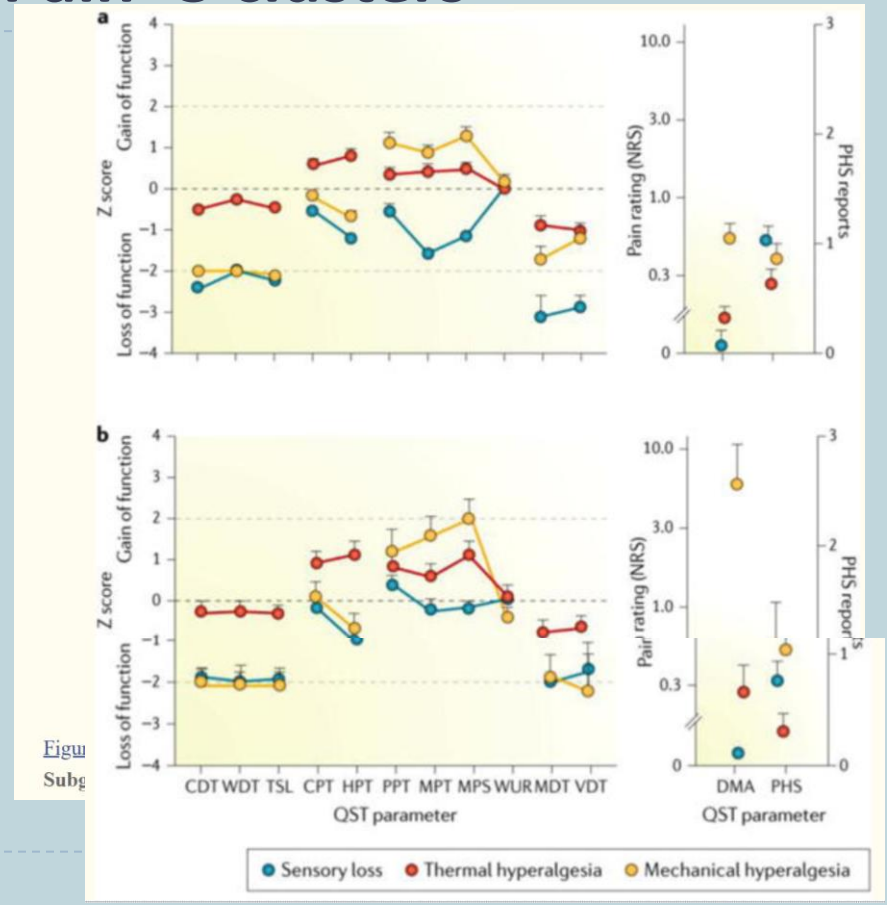
General sensory nerve injury recovery predictors

- Cause of trauma
- Degree and site of nerve damage
- Pain profile
- Delay in presentation
- Age of patient
- Psychological factors
- Medical factors?
- Social factors?

Sub types of neuropathic pain
Phenotyping patients with NePain- 3 clusters

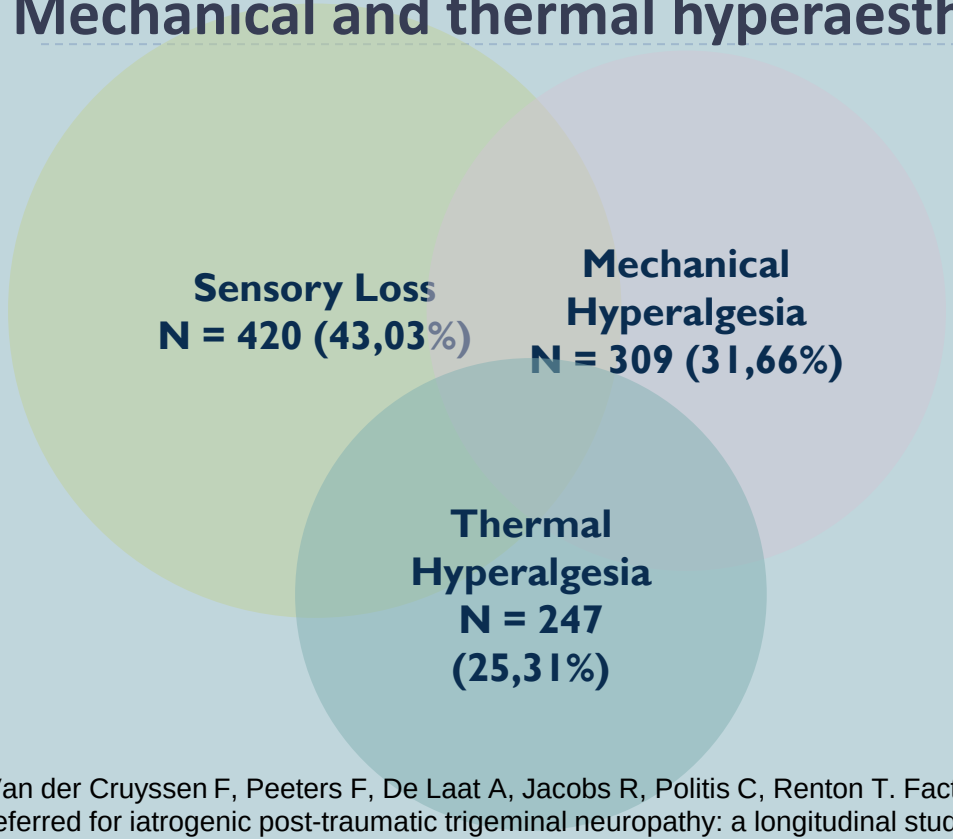
Furthermore, the first clinical trial to show phenotype stratification based on these sensory profiles has predictive power for treatment response.

Error bars are the graphical representation of the variability of the data present in the database. CDT, cold detection threshold; CPT, cold pain threshold; HPT, heat pain threshold; MDT, mechanical detection threshold; MPS, mechanical pain sensitivity; MPT, mechanical pain threshold; PPT, pressure pain threshold; QST, quantitative sensory test; TSL, thermal sensory limen; VDT, vibration detection threshold; WDT, warm detection threshold; WUR, wind-up ratio.



Baron, R. *et al.*, Peripheral neuropathic pain: a mechanism-related organizing principle based on sensory profiles, *Pain*, **158**, 2, 261–272,

Clustering of Sensory Profiles Trigeminal PTNP (N = 976)
Mechanical and thermal hyperaesthesia less likely to recover?



DOI: 10.1111/joor.13058

ORIGINAL ARTICLE

WILEY

Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries

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Abstract

Background: Post-traumatic trigeminal neuropathy (PTN) is a disturbance of function or pathological change of the trigeminal nerve branches following trauma and has an important impact on patient's quality of life (QoL).

Objectives: To provide diagnostic data on PTN and illustrate differences in aetiology, injured nerve, pain distribution, sensory profile and QoL between PTN subgroups.

Methods: 1331 patients with painful or non-painful PTN were retrospectively re-viewed in two centres, extracting demographic data, time and cause of trauma, clinical findings including signs and symptoms, basic neurosensory testing, imaging modalities, treatments, and QoL or psychosocial assessment.

Results: More females were represented (70%) than males. The inferior alveolar nerve was most frequently damaged (60%) followed by the lingual nerve (28%). Wisdom teeth removal was considered the main cause (48%). Pain was reported in 63% of patients and pain frequency increased with age without clinically significant gender differences. Numbness was reported in 50% of PTN patients. Neurosensory testing showed larger affected dermatome involvement in persistent injuries, with no differences between the non-painful and painful PTN groups. Patient clustering indicated different sensory profile distributions when stratified according to aetiol-ogy or affected nerve branch. High interference with lifestyle was reported (78%), and patients suffering from painful PTN had worse QoL and psychosocial outcomes.

Conclusion: Patients with painful PTN had different clinical profiles and lower QoL scores than those with non-painful PTN. Sensory profiles may provide important prognostic and therapeutic information; however, more research is needed to assess the clustering procedure and link these clusters to therapeutic guidelines.

KEYWORDS
diagnosis, neuropathic pain, quality of life, trigeminal nerve, trigeminal nerve disorder

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

Van der Cruyssen F, Peeters F, Gill T, De Laat A, Jacobs R, Politis C, Renton T. Signs and symptoms, quality of life and psychosocial data in 1331 post-traumatic trigeminal neuropathy patients seen in two tertiary referral centres in two countries. J Oral Rehabil. 2020 Oct;47(10):1212-1221. doi: 10.1111/joor.13058. Epub 2020 Aug 2. PMID: 32687637; PMCID: PMC7540026.

Predicting resolution of Trigeminal Post Traumatic Neuropathy

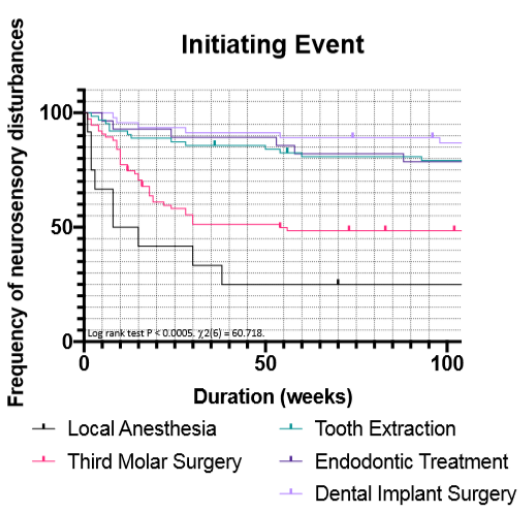
LA> M3M> Endo Implant & non M3M extraction
Lingual nerve > Inferior alveolar and maxillary nerve
Hypoaesthesia> hyperaesthesia

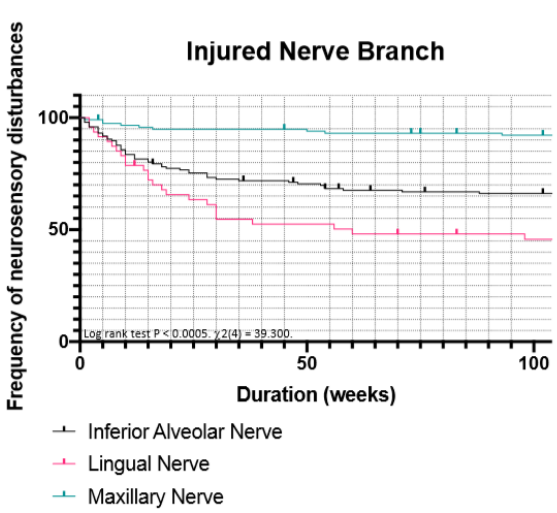
Positive factors for resolution
LA or M3M cause
EQ5D low pain
Lingual nerve
Sensory loss with or without pain

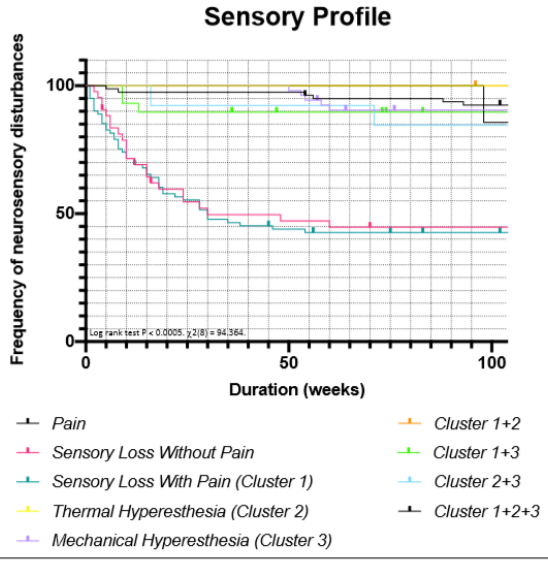
Negative factors for resolution
EQ5D poor activity
Allodynia

Endo Implant nerve injuries
Maxillary nerve
Duration of NI

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

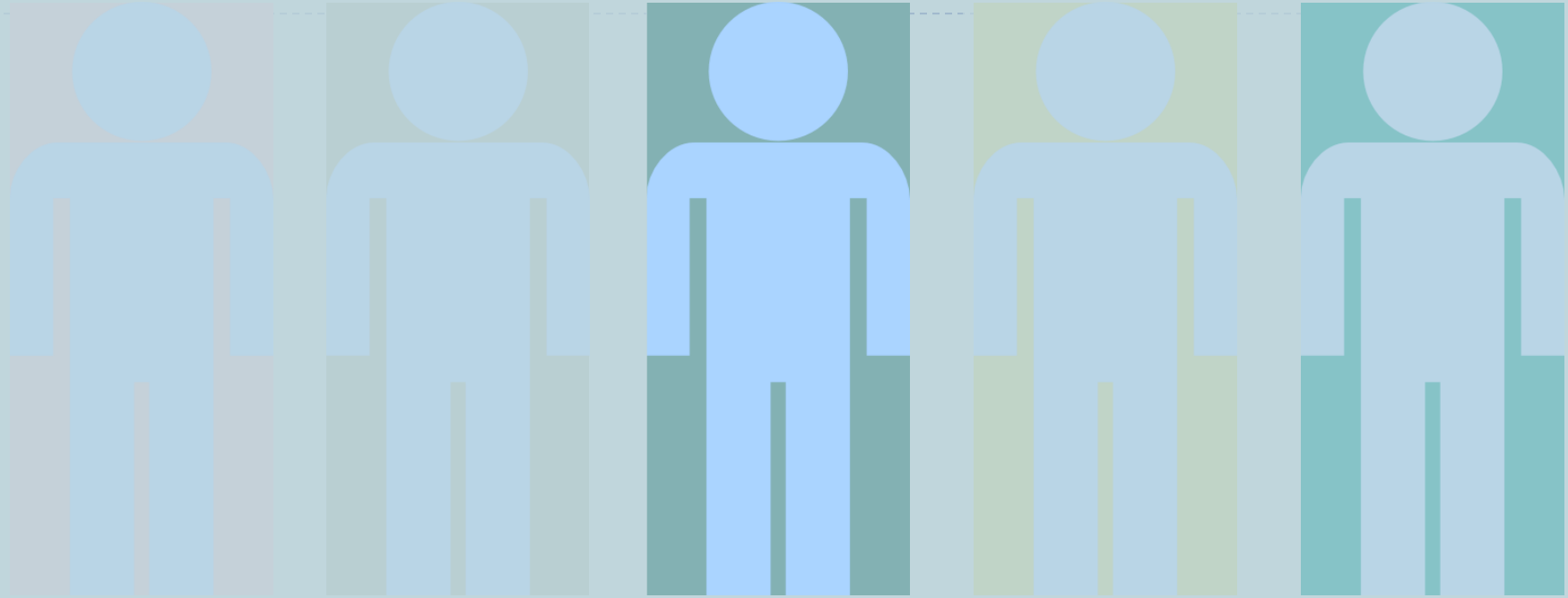






Van der Cruyssen F, Peeters F, De Laat A, Jacobs R, Politis C, Renton T. Factors affecting evolution of symptoms and quality of life in patients referred for iatrogenic post-traumatic trigeminal neuropathy: a longitudinal study in two tertiary referral centers in UK and Belgium. Pain 2020 in press.

Overview



Consequences of Trigeminal PTN

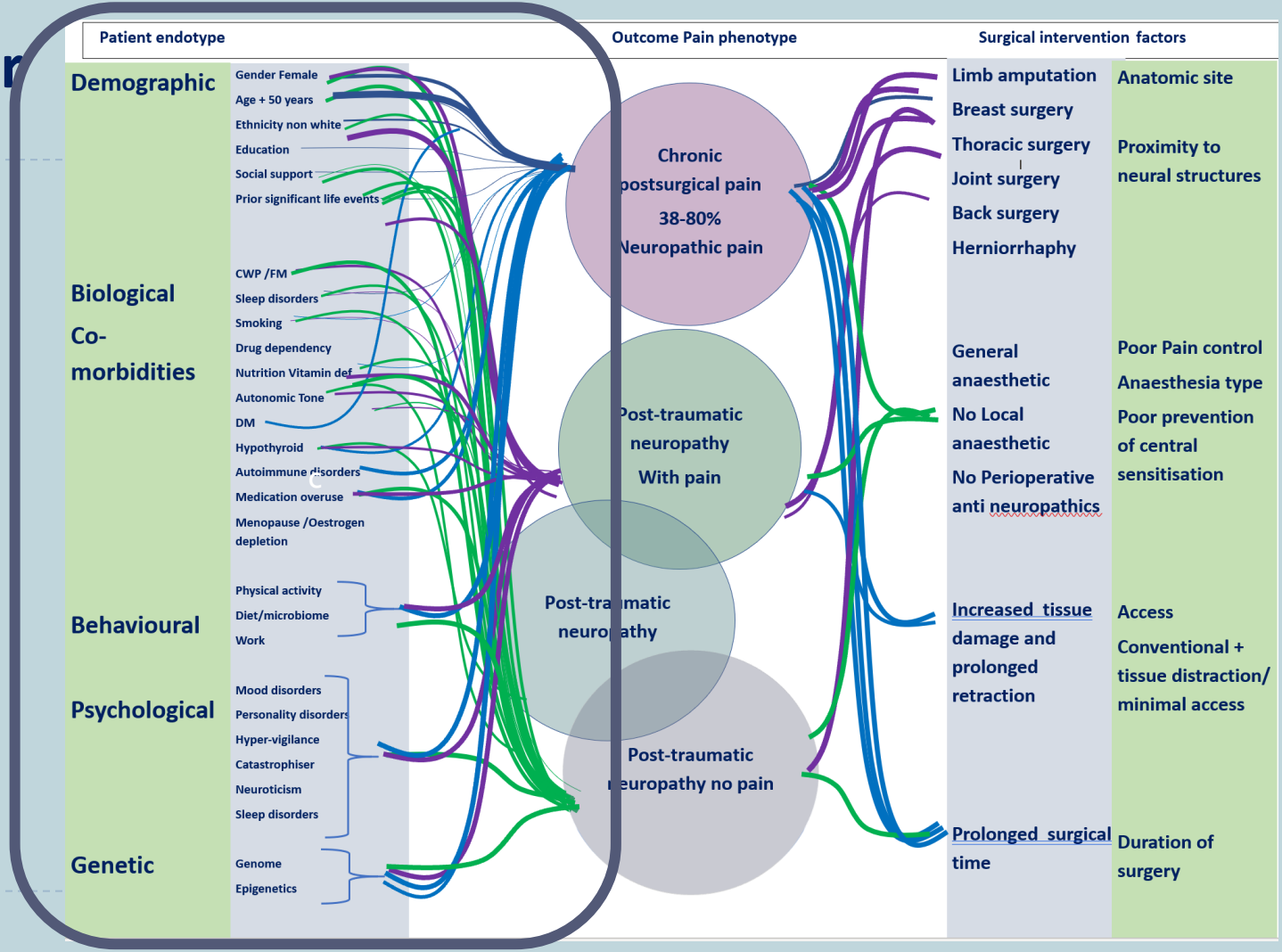
Neuropathic pain and diagnosis and assessment

Implant PTNP related risk factors

Prevention of Implant related PTN

Management of patients with Implant related PTN

Predictors for CPSP/PTNP



Risk factors for chronic post surgical (Ne) pain

PAIN: Clinical Updates
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Risk stratification for the development of chronic postsurgical pain

Stephan A. Schug[a,*], Julie Bruce[b]

Keywords: Chronic postsurgical pain, Persistent postsurgical pain, Risk stratification, Genetics, Psychosocial, Surgery

The past 20 years have seen an increasing recognition of the burden of chronic pain after surgery and other trauma. There is now good evidence that chronic postsurgical pain (CPSP) is by far more common and more severe than previously thought with far-reaching consequences for quality of life and function of those affected. There are also significant implications and costs for health care systems and society as a whole.[14] **Table 1** highlights this by showing the incidence of chronic pain after a number of surgical interventions, as well as the proportion of patients who experience severe pain and the contribution of neuropathic pain features to this presentation. The wide variability of these numbers is largely due to methodological differences, caused by the use of variable definitions for chronicity, in particular regarding the time frame applied for measurement (between 2 and 12 months). Other factors include differences in study design (eg, cross-sectional, prevalence surveys or prospective surgical cohort studies), as well as variable assessment of preoperative chronic pain and measurement of postoperative pain. Attempts to standardise a definition have been based on the initial proposal by Macrae and Davies,[17] modified by Werner and Kongsgaard,[27] which have now been presented by the IASP to the WHO for the upcoming revision of the *International Classification of Diseases Eleventh Revision* (*ICD-11*) as[25]: "Chronic postsurgical pain is pain developing after a surgical procedure and persisting beyond the healing process, ie, at least 3 months after surgery. The pain is either localized to the surgical field, projected to the innervation territory of a nerve situated in this area, or referred to

Key Points

1. Chronic postsurgical pain (CPSP) is a common complication of surgery with important consequences for the individual patient and society as a whole.
2. Risk stratification is best defined as the grouping of patients based on factors measured at baseline (in this context before surgery), to determine an individual's risk of suffering a particular condition and thereby the likely level of need for preventive interventions.
3. Risk factors for CPSP have been identified in the preoperative, intraoperative, and postoperative periods and cover 6 broad domains: genetic, demographic, psychosocial, pain, clinical, and surgical factors.
4. Risk stratification for CPSP enables clinicians to address these risk factors before surgery, to discuss the necessity of surgery or to change the surgical and anaesthetic/analgesic planning.

a dermatome (after surgery/injury to deep somatic or visceral tissues). Other causes of pain including infection, malignancy etc. need to be excluded as well as pain continuing from a preexisting pain problem."

These initial observations have resulted in a large research effort focussing on the topic of CPSP. This research effort has focussed initially on the epidemiology to establish the burden of

Table 2

Risk factors for CPSP by time line and domain.

Domain of risk factor	Preoperative period	Intraoperative period	Postoperative period
Demographic	Age Sex Others	N/A	N/A
Genetic	Multiple mutations	N/A	N/A
Psychological	Depression Psychological vulnerability Stress Anxiety Catastrophising	N/A	Depression Psychological vulnerability Stress Anxiety Catastrophising Poor coping skills
Pain	Preoperative chronic pain Preoperative opioid use Increased sensitivity to experimental pain Increased temporal summation Decreased CPM	N/A	Severe acute pain Acute neuropathic pain Acute secondary hyperalgesia
Surgical	N/A	Type of surgery Nerve injury Longer duration of surgery Traumatic approaches	Need for repeated revisions
Clinical	Severity and numbers of comorbidities Disability	N/A	Radiotherapy Chemotherapy

Compiled from multiple sources including Refs. 13,22,26.
CPM, conditioned pain modulation; CPSP, chronic postsurgical pain.

Psychosocial factors CPSP

Studies examining the influence of psychological factors on chronic post-surgical pain are few, with contradictory results.

Kock has suggested that chronic post-surgical pain can be caused by;

- **Hypervigilant state**
- **Fear of surgery**
- **Anxiety**
- **Psychological vulnerability, specifically pain-related fear and coping skills-**
- **Personality disorders These might reflect psychosocial vulnerabilities in coping skills that are antecedents to chronic pain.**
- **Depression and neuroticism might lead to higher incidences of chronic pain after surgery.**

The psychological factors that seem to be the risk factors for acute pain do not show the same association with chronic post-surgical pain.

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Psychosocial predictors in the transition from acute to chronic pain: a systematic review

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Abstract

Chronic non-cancer pain (CNCP) is a major health problem which psychosocial factors have significant implications in. There is a gap in regards to evidence for the prevention of chronicity specifically addressing psychological and social domains. Four databases were searched with terms related to "psychosocial", "acute pain", and "chronic pain". A total of 1,389 studies were identified in which titles, abstracts, and full texts were assessed for inclusion criteria. A data template was used to capture pertinent details, and overall themes and patterns were organized according to type of pain examined and psychosocial variables measured. Of the 18 articles that met inclusion criteria, fifteen (83%) of the articles reported an association between psychosocial factors and chronicity. A total of 5 of the studies (29%) demonstrated that depression was a possible predictor and 6 (35%) of the studies found fear-avoidance to be associated with chronicity. This review provides evidence that psychosocial factors are associated with chronicity within CNCP. These results suggest a need for targeting psychosocial predictors in prevention and early intervention through clinical guidelines and a national strategy to support a cultural change in pain care.

Keywords

systematic review; chronic pain; chronicity; psychosocial; mental health

Introduction

Chronic pain has significant medical, social, and economic implications. In the US, chronic pain affects 100 million adults and annual costs are estimated to be between $560 and 635 billion a year (Gaskin & Richard, 2012). The impact on individuals' quality of life, health care utilization, and social resource expenditures provides a compelling motive to better understand the mechanisms involved in the transition of acute to chronic pain (Häuser et al., 2014). While there is growing evidence that psychosocial factors play a significant role in the transition from acute to chronic pain (Katz & Seltzer, 2009; Liu et al., 2012; VanDenKerkhof, Peters, & Bruce, 2013) there is still a need to enhance the evidence

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Disclosure statement
The authors declare that they have no conflict of interest.

Risk factors for pain chronicity
Determinants for onset and maintenance of chronic pain
Biopsychosocial


COMMENTARY

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], ...

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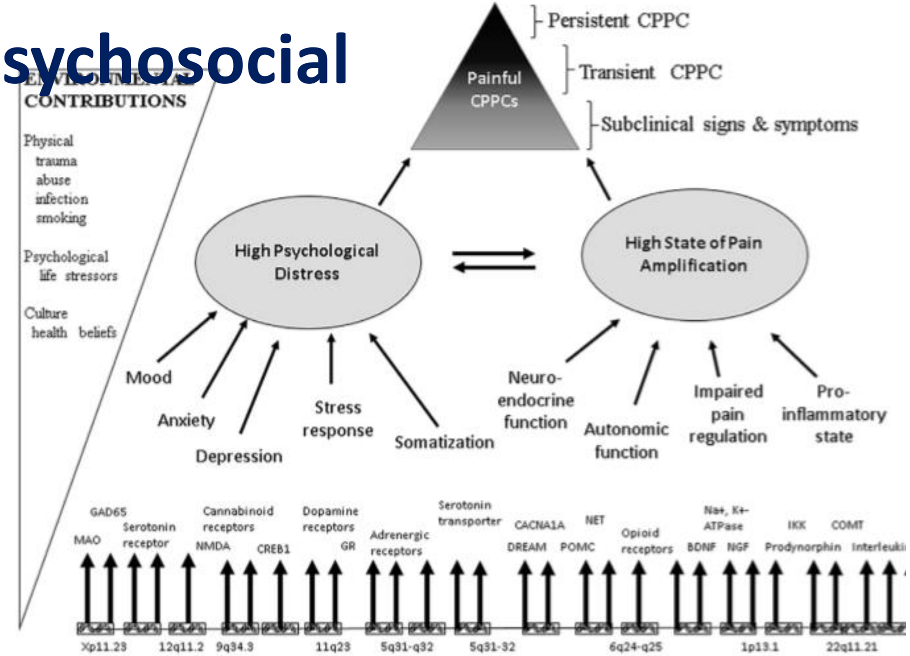
ABSTRACT
Objective: Pain is one of the most common reasons for an individual to consult their primary care physician, with most chronic pain being treated in the primary care setting. However, many primary

ARTICLE HISTORY
Received 18 December 2017
Revised 5 March 2018



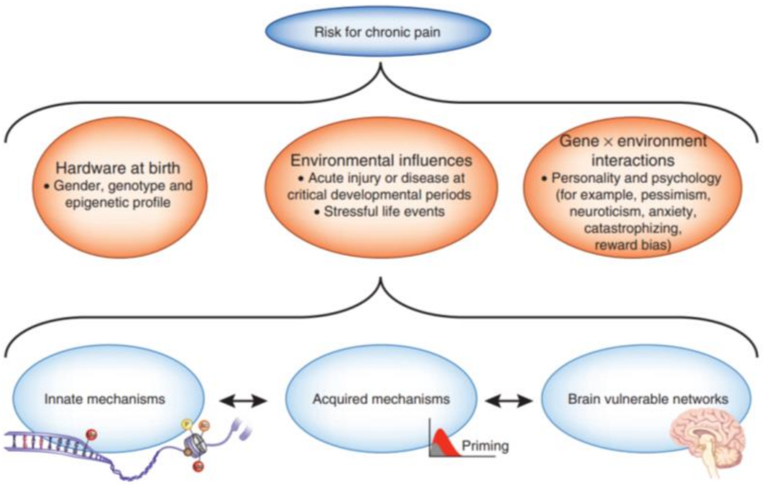


Figure 4. This model depicts likely determinants that contribute to the risk of onset and maintenance of common chronic overlapping pain conditions (COPCs). These factors are determined by genetic variability and environmental events that determine an individual's psychological profile and pain amplification 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; POMC, proopiomelanocortin; NET, norepinephrine transporter; BDNF, brain-derived neurotrophic factor; NGF, nerve growth factor; IKK, IκB kinase; COMT, catechol-O-methyl transferase.

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

Risk factors PTNP

The patient endotype & surgical factors

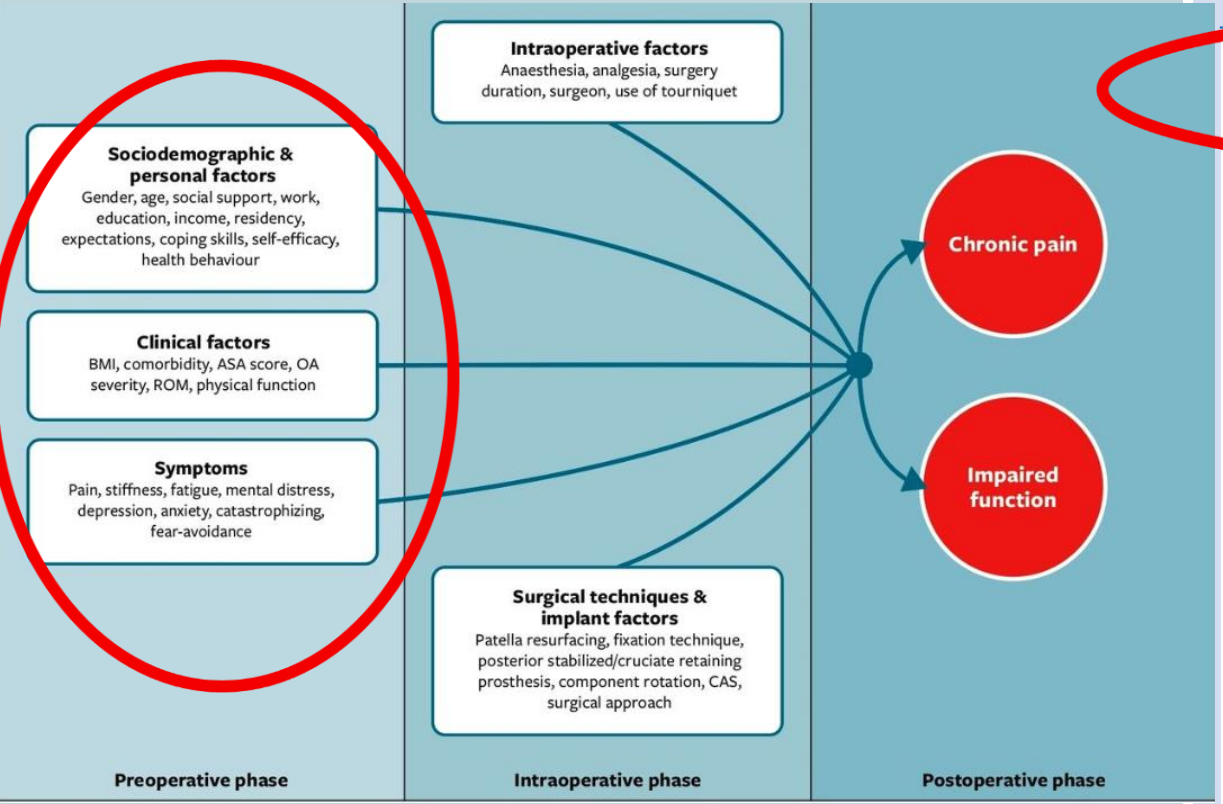


Table 1 Predisposing factors for chronic post-procedural pain

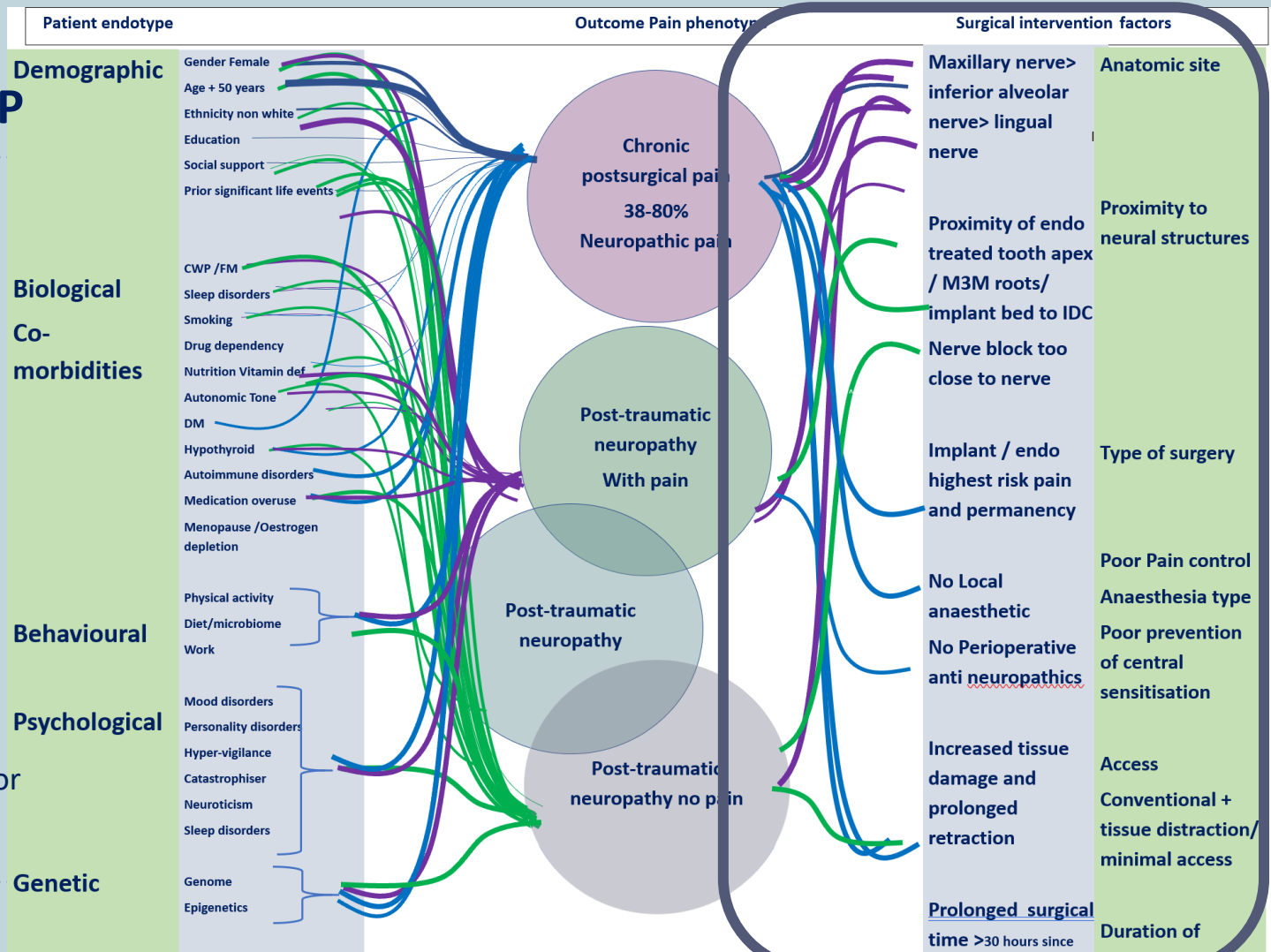
Preoperative pain at the site of surgery or other body regions
Psychosocial and mood factors
Coping skills
Surgical factors
1. Nerve damage (complicated aetiology likely than just nerve injury alone)
2. Factors predisposing to prolonged inflammatory states (foreign materials)
3. Volume of surgeries performed per year for given operation
4. Recurrence of operation
5. Type of surgery
6. Length of surgery
Genetic predisposition
Acuity of postoperative pain
Prolonged postoperative pain/inflammatory responses
Duration of postoperative pain treatment
Anaesthetic factors (general *vs* regional, type of general anaesthesia)
Gender (female)
Type of disease
Recurrence of malignancy
Adjuvant therapy: radiation, chemotherapy (conflicting reports)
Age (conflicting reports)

Surgical Predictors for Trigeminal PTNP

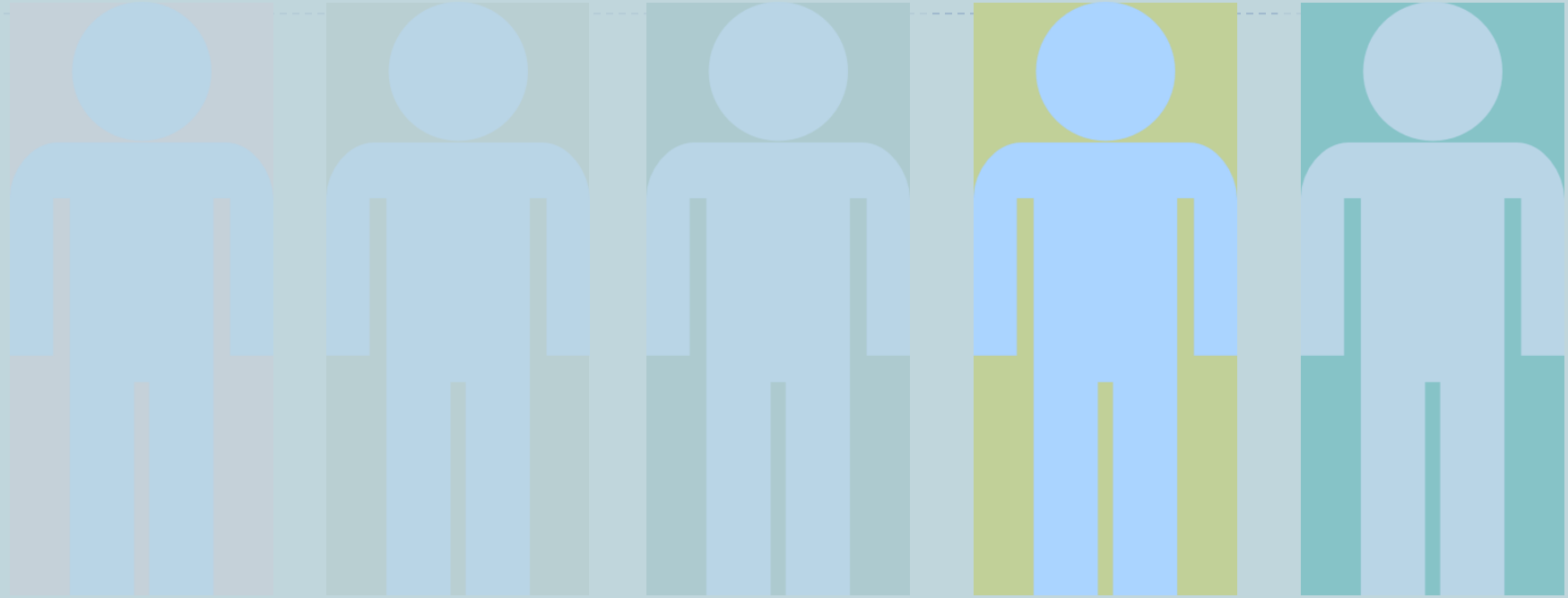
Site
Proximity to neural structures
pain during drill preparation

Type of surgery

Perioperative pain
-multiple prior pain episodes or ongoing pain
-high level intraoperative pain
-high level postoperative pain



Overview



Consequences of Trigeminal PTN

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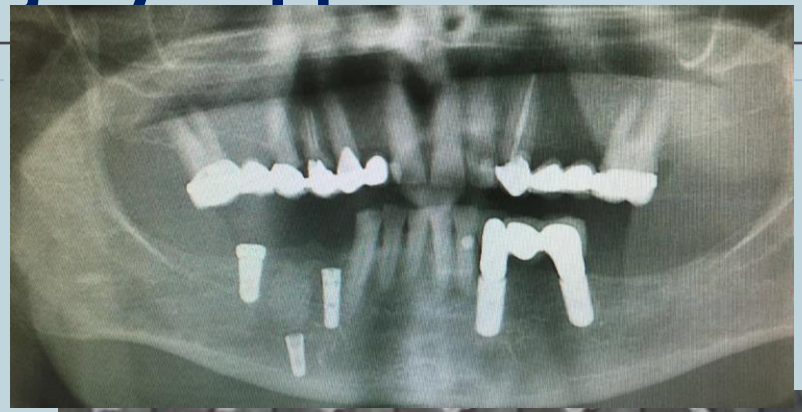
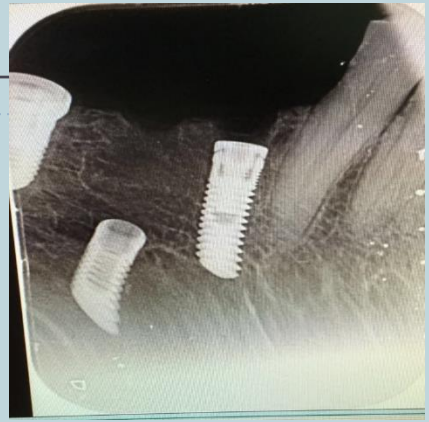
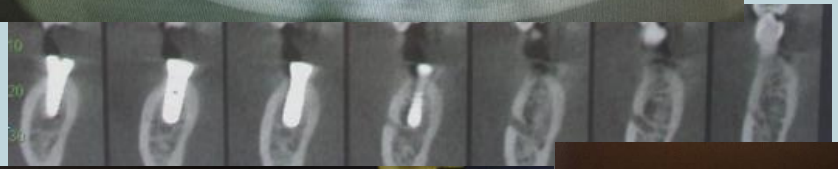
Prevention of Implant related PTN

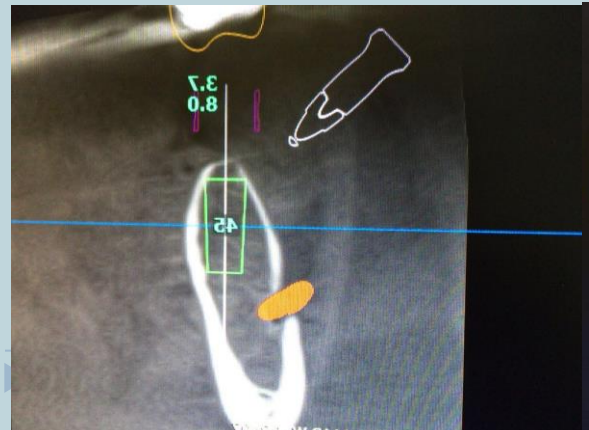
Management of patients with Implant related PTN

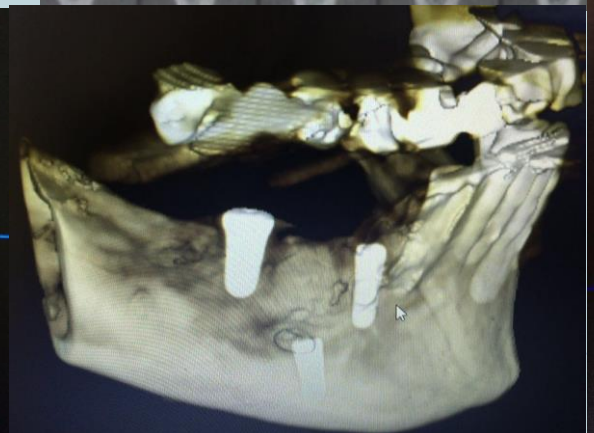
How does the injury happen?

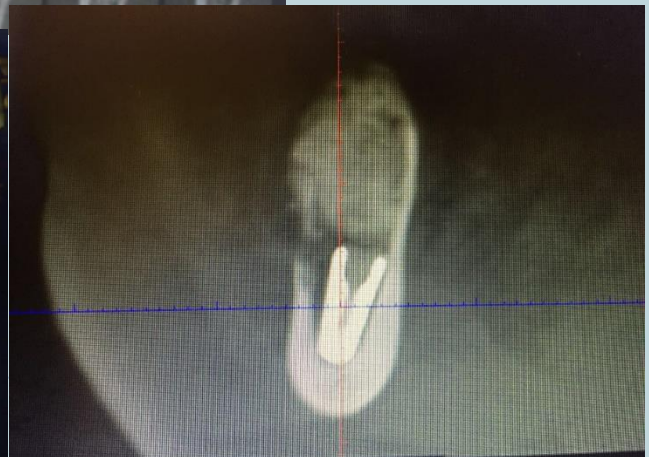












Possible aetiological factors in implant relatedPTN

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Hom-Lay Wang
Gintautas Sabalys
Antanas Sidlauskas
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Inferior alveolar nerve injury associated with implant surgery

Table 1. **Etiological factors and mechanism of traumatic inferior alveolar nerve (IAN) injury**

Intraoperative etiological factor	Indirect or direct and injury mechanism	Post-operative etiological factor	Indirect and injury mechanism
Traumatic local anesthesia			
Chemical (cytotoxic) injury by local anesthetic	Indirect; endoneurial edema, compression and secondary ischemia Direct; IAN degeneration	Injection needle trauma to epineurial blood vessels or inferior alveolar artery	Indirect; hematoma with reactive fibrosis and scar formation, compression and secondary ischemia
Injection needle	Direct; transection of multiple IAN fibers and entire fascicles		
Implant drill			
Partial intrusion into MC	Indirect; hematoma and secondary ischemia	Thermal injury	Indirect; inflammation of bone and IAN with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN		
Chemical (cytotoxic) injury	Direct; IAN degeneration		
Thermal injury	Direct; IAN degeneration		
Dental implant			
Partial intrusion into MC	Indirect; hematoma or/and deposition of debris, compression and secondary ischemia	Infection	Indirect; inflammation of bone and IAN with secondary ischemia
		Implant is too close to MC	Indirect; bone and IAN stress, compression with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN	Chronic stimulation	Indirect; implant is situated aside of or on top of the nerve with chronic neuropathy formation
Wrong operation technique			
Scalpel	Direct; mental nerve injury or transection	Soft tissue swelling	Indirect; mental nerve compression caused by soft tissue edema
Soft tissue reflection and retraction	Direct; mental nerve injury caused by reflection, retraction and pressure		
Soft tissue suturing	Direct; mental nerve compression caused by suture material		

MC, mandibular canal.

Possible aetiological factors in implant relatedPTN

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Inferior alveolar nerve injury associated with implant surgery

Table 1. **Etiological factors and mechanism of traumatic inferior alveolar nerve (IAN) injury**

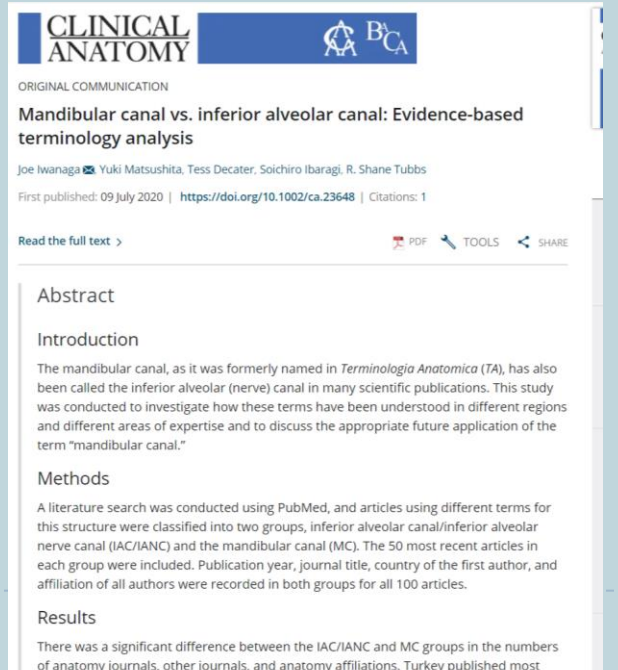
Intraoperative etiological factor	Indirect or direct and injury mechanism	Post-operative etiological factor	Indirect and injury mechanism
Traumatic local anesthesia			
Chemical (cytotoxic) injury by local anesthetic	Indirect; endoneurial edema, compression and secondary ischemia Direct; IAN degeneration	Injection needle trauma to epineurial blood vessels or inferior alveolar artery	Indirect; hematoma with reactive fibrosis and scar formation, compression and secondary ischemia
Injection needle	Direct; transection of multiple IAN fibers and entire fascicles		
Implant drill			
Partial intrusion into MC	Indirect; hematoma and secondary ischemia	Thermal injury	Indirect; inflammation of bone and IAN with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN		
Chemical (cytotoxic) injury	Direct; IAN degeneration		
Thermal injury	Direct; IAN degeneration		
Dental implant			
Partial intrusion into MC	Indirect; hematoma or/and deposition of debris, compression and secondary ischemia	Infection	Indirect; inflammation of bone and IAN with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN	Implant is too close to MC	Indirect; bone and IAN stress, compression with secondary ischemia
		Chronic stimulation	Indirect; implant is situated aside of or on top of the nerve with chronic neuropathy formation
Wrong operation technique			
Scalpel	Direct; mental nerve injury or transection	Soft tissue swelling	Indirect; mental nerve compression caused by soft tissue edema
Soft tissue reflection and retraction	Direct; mental nerve injury caused by reflection, retraction and pressure		
Soft tissue suturing	Direct; mental nerve compression caused by suture material		

MC, mandibular canal.

During Implant bed drill preparation
Insufficient safety zone

Anatomy Nomenclature

- we suggest that the mandibular car
  the **“inferior alveolar canal.”**



CLINICAL ANATOMY

ORIGINAL COMMUNICATION

Mandibular canal vs. inferior alveolar canal: Evidence-based terminology analysis

Joe Iwanaga ✉ Yuki Matsushita, Tess Decater, Soichiro Ibaragi, R. Shane Tubbs

First published: 09 July 2020 | https://doi.org/10.1002/ca.23648 | Citations: 1

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Abstract

Introduction

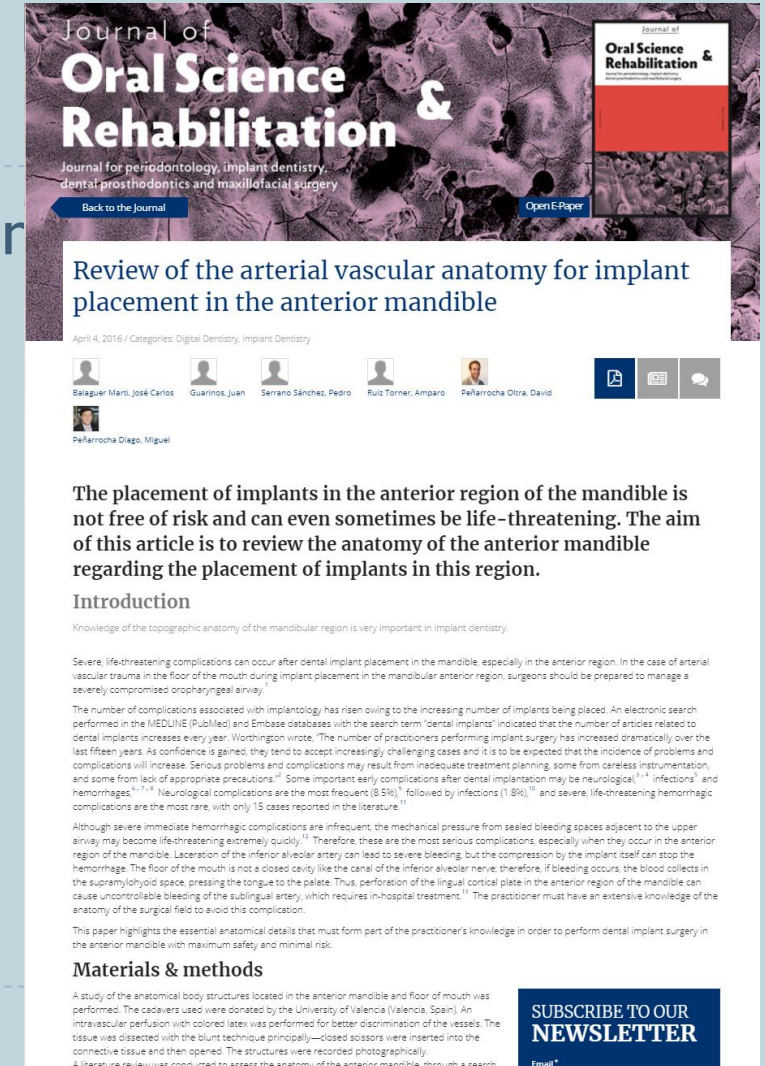
The mandibular canal, as it was formerly named in *Terminologia Anatomica* (*TA*), has also been called the inferior alveolar (nerve) canal in many scientific publications. This study was conducted to investigate how these terms have been understood in different regions and different areas of expertise and to discuss the appropriate future application of the term “mandibular canal.”

Methods

A literature search was conducted using PubMed, and articles using different terms for this structure were classified into two groups, inferior alveolar canal/inferior alveolar nerve canal (IAC/IANC) and the mandibular canal (MC). The 50 most recent articles in each group were included. Publication year, journal title, country of the first author, and affiliation of all authors were recorded in both groups for all 100 articles.

Results

There was a significant difference between the IAC/IANC and MC groups in the numbers of anatomy journals, other journals, and anatomy affiliations. Turkey published most



Journal of Oral Science & Rehabilitation

Journal for periodontology, implant dentistry, dental prosthodontics and maxillofacial surgery

Back to the Journal Open E-Paper

Review of the arterial vascular anatomy for implant placement in the anterior mandible

April 4, 2016 / Categories: Digital Dentistry, Implant Dentistry

Balaguer Martí, José Carlos Guarinos, Juan Serrano Sánchez, Pedro Ruiz Torner, Amparo Peñarrocha Oltra, David Peñarrocha Diago, Miguel

The placement of implants in the anterior region of the mandible is not free of risk and can even sometimes be life-threatening. The aim of this article is to review the anatomy of the anterior mandible regarding the placement of implants in this region.

Introduction

Knowledge of the topographic anatomy of the mandibular region is very important in implant dentistry.

Severe, life-threatening complications can occur after dental implant placement in the mandible, especially in the anterior region. In the case of arterial vascular trauma in the floor of the mouth during implant placement in the mandibular anterior region, surgeons should be prepared to manage a severely compromised oropharyngeal airway.[1]

The number of complications associated with implantology has risen owing to the increasing number of implants being placed. An electronic search performed in the MEDLINE (PubMed) and Embase databases with the search term “dental implants” indicated that the number of articles related to dental implants increases every year. Worthington wrote, “The number of practitioners performing implant surgery has increased dramatically over the last fifteen years. As confidence is gained, they tend to accept increasingly challenging cases and it is to be expected that the incidence of problems and complications will increase. Serious problems and complications may result from inadequate treatment planning, some from careless instrumentation, and some from lack of appropriate precautions.”[2] Some important early complications after dental implantation may be neurological,[3,4] infections[5] and hemorrhages.[6,7,8] Neurological complications are the most frequent (8.5%),[9] followed by infections (1.8%),[10] and severe, life-threatening hemorrhagic complications are the most rare, with only 15 cases reported in the literature.[11]

Although severe immediate hemorrhagic complications are infrequent, the mechanical pressure from sealed bleeding spaces adjacent to the upper airway may become life-threatening extremely quickly.[12] Therefore, these are the most serious complications, especially when they occur in the anterior region of the mandible. Laceration of the inferior alveolar artery can lead to severe bleeding, but the compression by the implant itself can stop the hemorrhage. The floor of the mouth is not a closed cavity like the canal of the inferior alveolar nerve; therefore, if bleeding occurs, the blood collects in the supramylohyoid space, pressing the tongue to the palate. Thus, perforation of the lingual cortical plate in the anterior region of the mandible can cause uncontrollable bleeding of the sublingual artery, which requires in-hospital treatment.[13] The practitioner must have an extensive knowledge of the anatomy of the surgical field to avoid this complication.

This paper highlights the essential anatomical details that must form part of the practitioner’s knowledge in order to perform dental implant surgery in the anterior mandible with maximum safety and minimal risk.

Materials & methods

A study of the anatomical body structures located in the anterior mandible and floor of mouth was performed. The cadavers used were donated by the University of Valencia (Valencia, Spain). An intravascular perfusion with colored latex was performed for better discrimination of the vessels. The tissue was dissected with the blunt technique principally—closed scissors were inserted into the connective tissue and then opened. The structures were recorded photographically.

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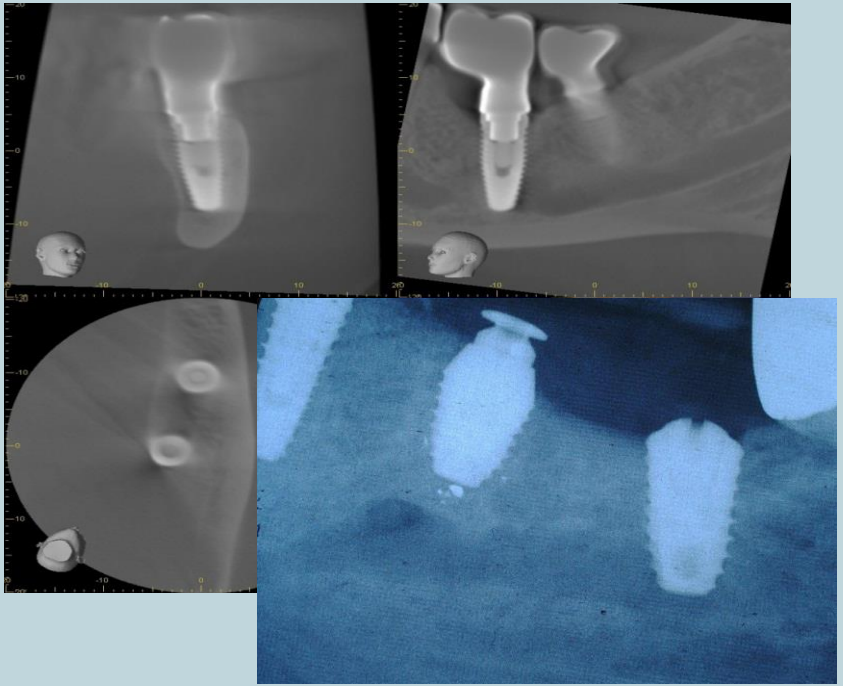
Preventing implant related post traumatic neuropathy

- **Treatment need?**
- **Correct patient?**
  - History clinical examination
- **Manage expectations Consent**
  - Indication for treatment
  - Guidelines
  - Risks
- **Planning Risk assessment**
  - Clinical
  - Radiographic When is CBCT recommended? Who reads CBCT?
  - Safety zone
- **LA protocol**
  - Articaine as infiltration only

  Peterson 2004; Heller & Shankland 2001
- **<u>Technique most drills longer than implants</u>**
- **<u>Early recognition Post operative care</u>**



Renton T. Prevention of iatrogenic inferior alveolar nerve injuries in relation to dental procedures. SADJ. 2010 Sep;65(8):342-4, 346-8, 350-1

Implant PTN risk factors

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

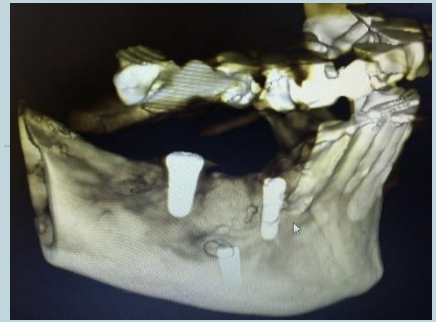
Operative

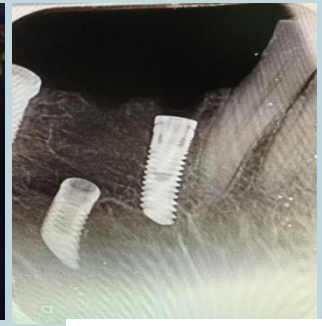
Poor technique reducing Safety zone/ lack use drill stops, guides/ intraoperative LCPAs
Lack of recognition risks bleeding/ drill sink

Post operative

Late recognition of nerve injury
Lack removal implant within 30 hours

Implant nerve injury Risk factors

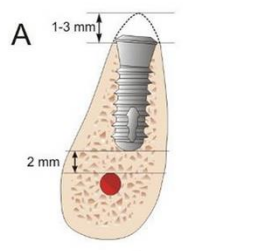


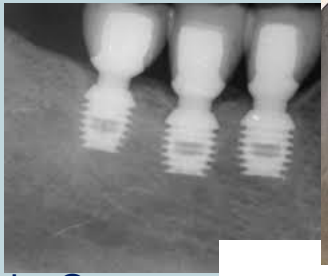


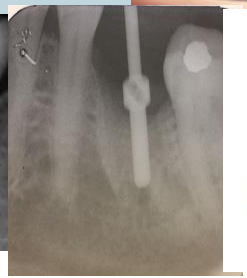
Most nerve injuries occur:

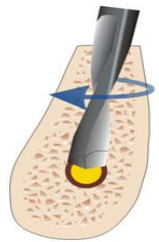
- In the parasymphyseal region
- During preparation of implant bed
- Using Implants >10mm
- When the patient experiences severe pain
  - during prep or implant placement
  - severe pain post surgery
  - Intraoperative bleed during prepping

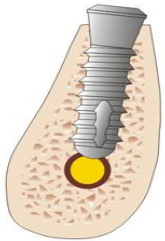
Safety zone of 2mm is insufficient with implant drills 1.5mm longer than the implants = resultant safety zone of 0.5mm!!!! 4mm!











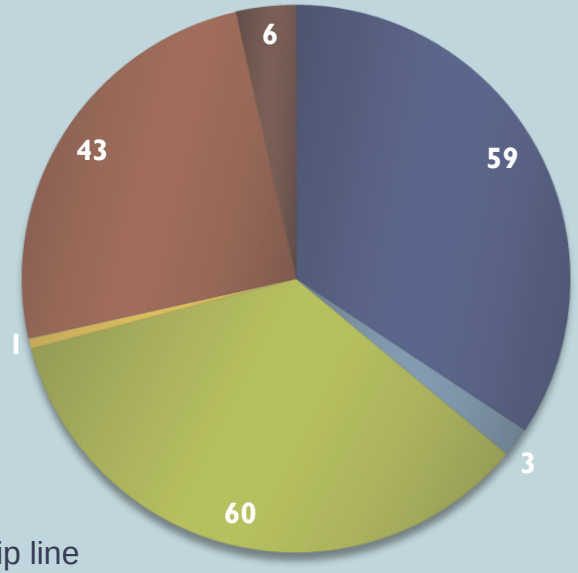
Yilmaz Z, Ucer C, Scher E, Suzuki J, **Renton T**. A Survey of the Opinion and Experience of UK Dentists: Part 1: The Incidence and Cause of Iatrogenic Trigeminal Nerve Injuries Related to Dental Implant Surgery. Implant Dent. 2016 Oct;25(5):638-45.

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

Preventing implant related nerve injury

Is there a need?

- Explore patients expectations
- Medical History
  - Smoker
  - Compromised immunity
  - MRONJ risk
- Clinical
  - Poor Oral hygiene
  - Periodontal disease
  - Bone mapping aesthetics, soft tissue, lip line
- Consent

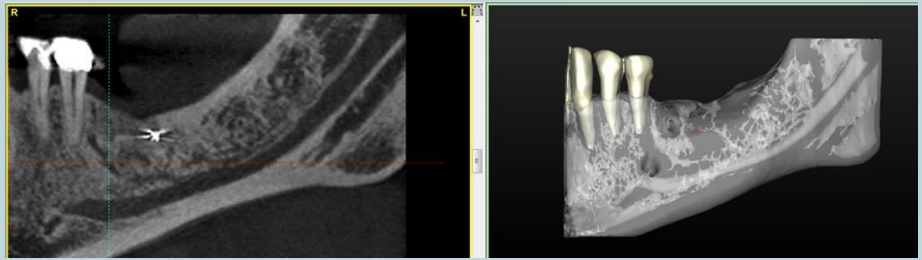


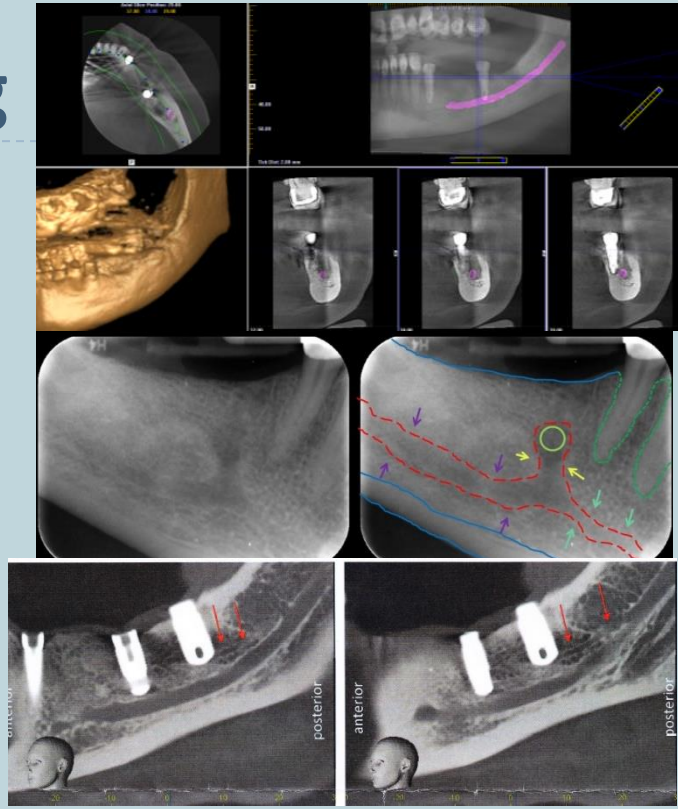
Contraindicated in patients with periodontal disease, smokers, bruxists, immunosuppressed.
The reality is only 57% of implants survive 10 years

Chanavaz M. Patient screening and medical evaluation for implant and preprosthetic surgery.J Oral Implantol. 1998;24(4):222-9. Revie

Maximising Safety Zone Planning

- Radiographic assessment
- Short implants





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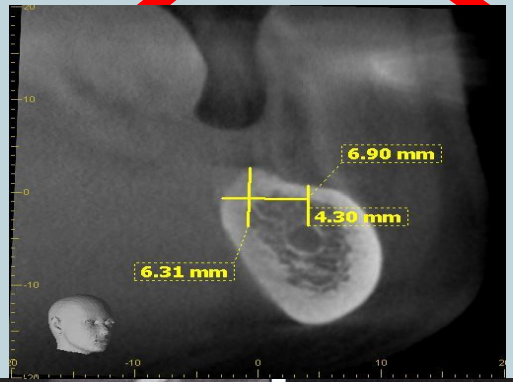
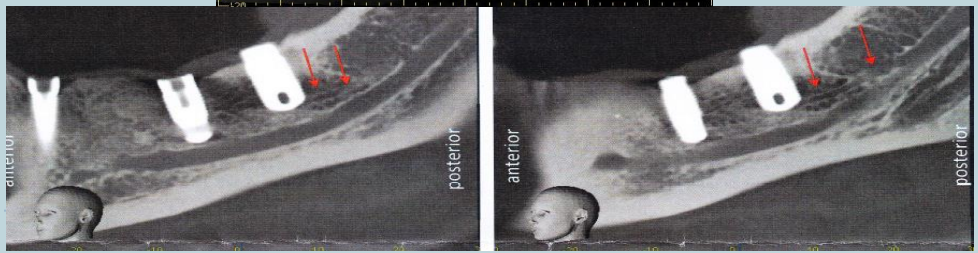
Managing preoperative risk
By good clinical and radiographic assessment

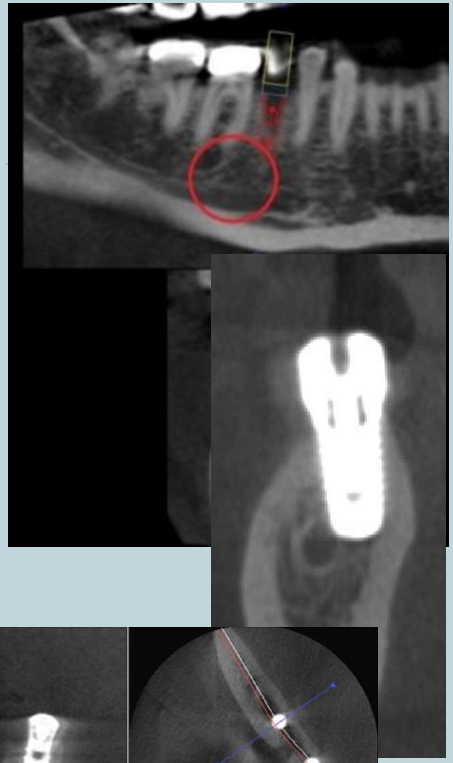
- LCPA inadequate for mandibular implants
- OPG/ DPT is standard
- Indications for CBCT
  - Mandibular implant surgery where depth and width of bone requires further assessment
  - Parasymphyseal region Premolar and first molar most problematic
  - **You MUST be able to read your own CBCTs**
  - Always get radiologist review to exclude pathology of all structures





Assessment clinical & Radiographic Guideline requirements

Preoperative assessment
Clinical assessment
Radiographic assessment
Patients expectations and treatment options ….Risk:Benefit



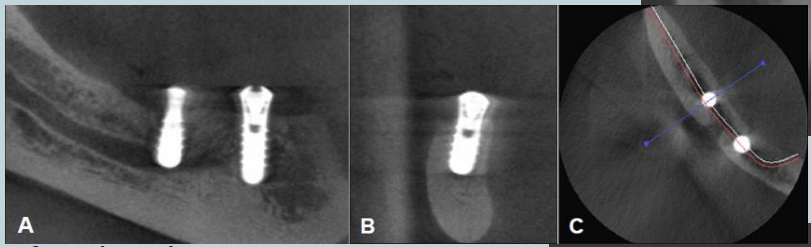
- **Radiographic EAO Sedentext CT guidance**

Clin Oral Implants Res. 2012 Nov;23(11):1243-53. doi: 10.1111/j.1600-0501.2012.02441.x. Epub 2012 Mar 21.

E.A.O. guidelines for the use of diagnostic imaging in implant dentistry 2011. A consensus workshop organized by the European Association for Osseointegration at the Medical University of Warsaw.

Harris D, Horner K, Gröndahl K, Jacobs R, Helmrot E, Benic GI, Bornstein MM, Dawood A, Quirynen M.
Dublin Dental School and Hospital, Trinity College, Dublin 2, Ireland. david@drdavidharris.com

Abstract
Diagnostics imaging is an essential component of patient selection and treatment planning in oral rehabilitation by means of osseointegrated implants. In 2002, the EAO produced and published guidelines on the use of diagnostic imaging in implant dentistry. Since that time, there have been significant developments in both the application of cone beam computed tomography as well as in the range of surgical and prosthetic applications that can potentially benefit from its use. However, medical exposure to ionizing radiation must always be justified and result in a net benefit to the patient. The as low a dose as is reasonably achievable principle must also be applied taking into account any alternative techniques that might achieve the same objectives. This paper reports on current EAO recommendations arising from a consensus meeting held at the Medical University of Warsaw (2011) to update these guidelines. Radiological considerations are detailed, including justification and optimization, with a special emphasis on the obligations that arise for those who prescribe or undertake such investigations. The paper pays special attention to clinical indications and radiographic diagnostic considerations as well as to future developments and trends.



- **Pre Existing neuropathy?**

25% of edentulous patients present with a degree of altered IAN function, thus reinforcing the guidelines on the necessity of preoperative neurosensory evaluation.

Chanavaz M.Patient screening and medical evaluation for implant and preprosthetic surgery.J Oral Implantol. 1998;24(4):222-9. Review

KING'S College LONDON

CBCT> Panoral

Evaluation of the risk of inferior alveolar nerve injury during an implant procedure: A comparative study between OPG and CBCT

Hassan A. Barzanji[1]; Alan A. Tahir[2]

Background and objectives: Dental implants are considered as one of the major options for replacement of missing teeth and this surgical procedure may be accompanied by trauma to the adjacent vital structure when there is inadequate information of the implant site. The use of OPG as a preliminary diagnostic instead of CBCT may expose the patient to a high risk of trauma to an inferior alveolar canal. To evaluate the possibility of the risk of endangering inferior alveolar nerve during implant placement using OPG or CBCT as a preoperative assessment tool.

Patients and methods: This study is a prospective cross-sectional study carried out in outpatient clinic of the college of dentistry and Denta Plus private center in Erbil city during the period from 1st of January to 31st of August, 2018. A sample of 49 patients was selected according to special criteria: Group I consists of 33 patients who had implant in molar and premolar regions, in this group pre-implant assessment done by Orthopantomogram (OPG). Group II; consists of 16 patients who had implant in molar and premolar regions, in this group pre-implant assessment done by Cone beam computed tomography (CBCT). The measurement of the distance between a dental implant and inferior alveolar canal were analyzed by CBCT which classified into four levels of parameters (distances) a-Safety zone ≥2 mm, b-Risky zone 1-2 mm, c-Error and high risk >0-1 mm, d-Traumatized ≤0 mm.

Results: the distance between implant and inferior alveolar canal (IAC) for group I (OPG) patients were as following: - in the safety zone for 30.3%, in the risky zone for 15.2%, in error & high risk for 21.2% and traumatized for 33.3%, while this distance for group II (CBCT) patients was in the safety zone for 75%, in the risky zone for 6.3%, in error & high risk for 12.5% and traumatized for 6.3%.

Conclusion: Cone beam computed tomography is the best choice compared to OPG in the pre-implant evaluation and planning for placement as it showed a lower risk of injury to an inferior alveolar canal.

Keywords: Dental implant, cone beam computed tomography, orthopantomogram.

[1]Department of Oral and Maxillofacial Surgery, College of Dentistry, Hawler Medical University, Erbil, Iraq.
[2]Department of Oral and Maxillofacial Surgery, College of Dentistry, Hawler Medical University, Erbil, Iraq.

Table 3: Showed the analysis of data for distances between the dental implant and inferior alveolar canal in both groups of study.

Distance groups	Pre-OPG (group I)		Pre-CBCT (group II)		P value
	No.	%	No.	%	
Safety zone	10	30.3	12	75.0	0.02*[S]
Risky zone	5	15.2	1	6.3	
Error and high risk	7	21.2	2	12.5	
Traumatized	11	33.3	1	6.3	
Total	33	100.0	16	100.0	

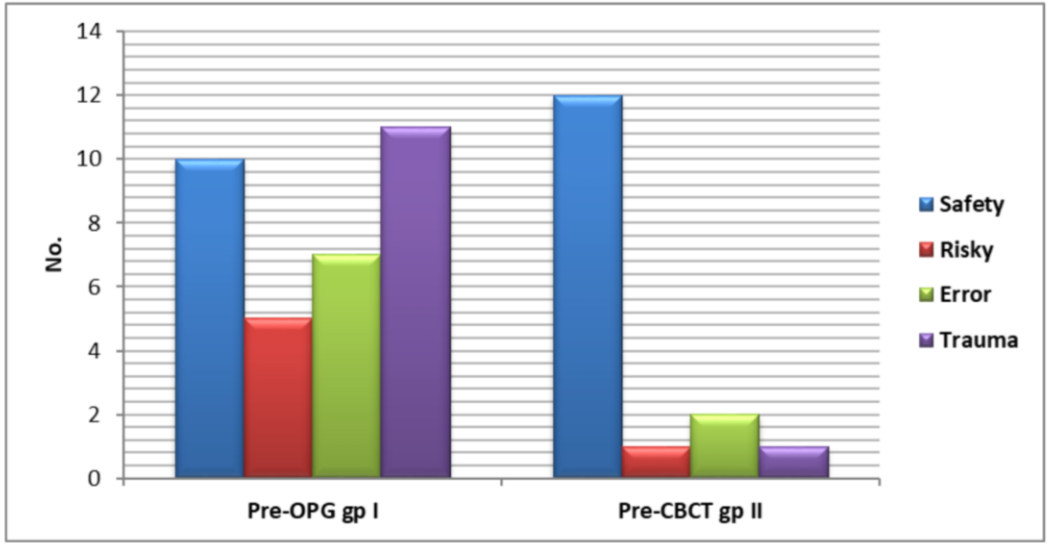


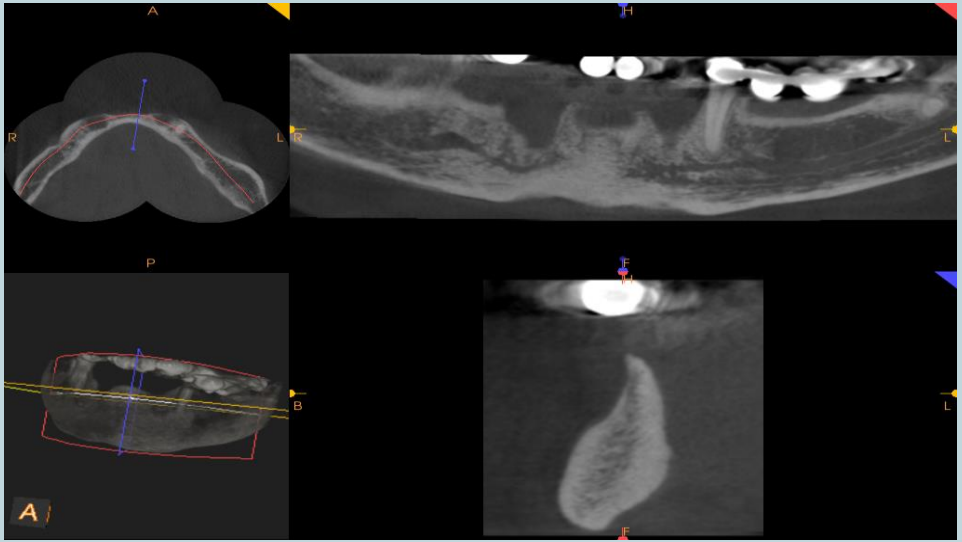
Figure 2: Distribution of different distance zones of an implant to IAC according to both groups of study.

Preoperative Risk assessment
Anterior extension of IAN

- OPG and DPT can over estimate the bone available for implants and CBCT can lead to over treatment

Both undesirable!!

- **Assess IDC**
  - **Position**
  - **Bifid**
  - **Cortication**
  - **lateral branches**
  - **Anterior extension**
- **No evidence that CBCT reduces morbidity related to implant treatment**



Courtesy of Dr. David R. Nelson BDS, MSc.(Imp.Dent), Clinical Director, Cranmore Clinical Tutor, Institute of Postgraduate Dental Education, University of Central Lancashire Tutor, School of Dentistry, Queen's University Belfast. Fellow, International Team for Implantology

Pre-extraction CT scans may present a useful diagnostic aid to assess the risk of inferior alveolar nerve injury and lingual plate perforation for IIP in the posterior mandible

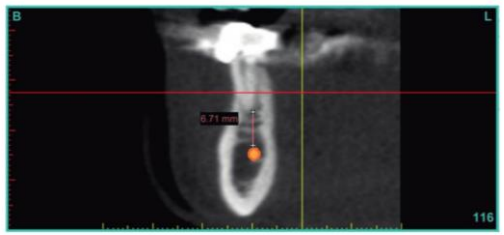


Figure 1.
RAC measurement. Measurement of available bone measured apical to the root apices to the alveolar canal. Orange area signifies the tracing of inferior alveolar dental canal drawn with implant software.[8]

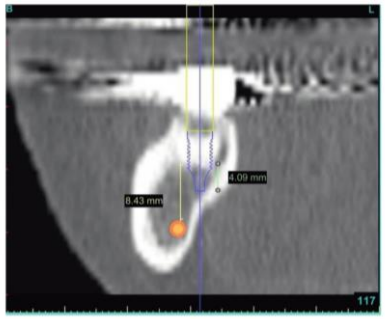


Figure 2.
RAC 26 mm. IIP does not present a high risk for inferior alveolar nerve injury. Orange area signifies the tracing of inferior alveolar dental canal drawn with implant software. Orange area signifies the tracing of inferior alveolar dental canal drawn with implant software.[8]

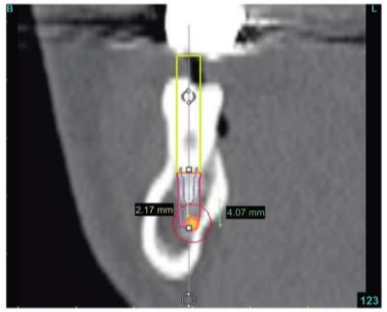


Figure 3.
RAC 10 mm. IIP presents a high risk for inferior alveolar nerve injury. Orange area signifies the tracing of inferior alveolar dental canal drawn with implant software.[8]

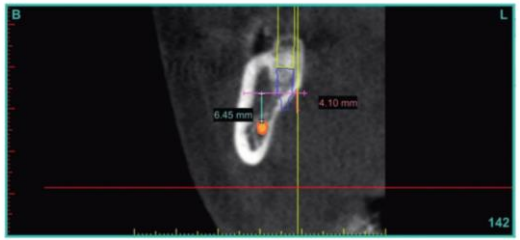


Figure 4.
RAC 26 mm. IIP presents high risk for lingual plate perforation when implant is placed in ideal position to support the replacement crown. Orange area

J Periodontol • March 2011

Risk Assessment Before Extraction for Immediate Implant Placement in the Posterior Mandible: A Computerized Tomographic Scan Study

Stuart Froum,* Leticia Casanova,* Sara Byrne,* and Sang Choon Cho*

Table 1.

Probability of Inferior Alveolar Nerve Injury According to Tooth Type

Tooth Type	No. Teeth Analyzed	High Risk for Inferior Alveolar Nerve Injury	
PM2	40	26/40	65%
M1	47	25/47	53%
M2	48	35/48	73%

Table 2.

Mean RAC for Each Tooth Type

Tooth Type	Mean (mm)	SD (mm)
PM2	4.86	2.82
M1	5.76	3.07
M2	4.41	3.04

Table 3.

Probability of Lingual Plate Perforation According to Tooth Type

Tooth Type	No. Teeth Analyzed	High Risk for Lingual Plate Perforation	
PM2	14	1/14	7%
M1	22	2/22	9%
M2	13	4/13	31%

4 mm in diameter. However, if a larger-diameter implant was selected, the probability for lingual plate perforation would increase, and if undiagnosed at the time of implant placement could present life-threatening complications.[32]

Currently in clinical practice, considering the high rates of implant survival, many posterior mandibular teeth requiring multidisciplinary treatment may be extracted and replaced by implant-supported restorations.[33,34] However, controversy remains as to when retaining a tooth may be considered hopeless and

KEY WORDS

Assessment

Who actually assesses the risk?

- Clinical
  - OH, Perio
  - Soft tissues and function
  - Hard tissue bone mapping, adjacent dentition
- Radiologic LCPA, DPT or CBCT?

  Guidelines Faculty Dental Practitioners 9FGDP

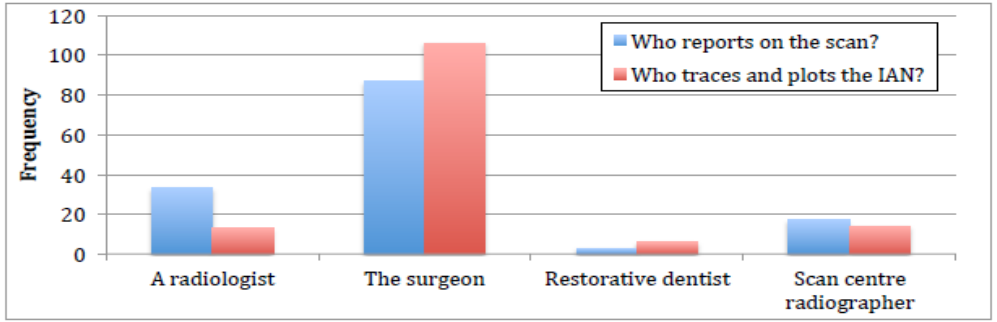
  SEDENTEXTCT
- Informed consent
- Who assesses the risk?

Cone Beam Computed Tomography in Implant Dentistry: A Systematic Review Focusing on Guidelines, Indications, and Radiation Dose Risks

Michael M. Bornstein, PD Dr Med Dent[1]/William C. Scarfe, BDS, FRACDS, MS[2]/ Vida M. Vaughn[3]/Reinhilde Jacobs, DDS, MSc, PhD, Dr hc[4]

Purpose: *The aim of the paper is to identify, review, analyze, and summarize available evidence in three areas on the use of cross-sectional imaging, specifically maxillofacial cone beam computed tomography (CBCT) in pre- and postoperative dental implant therapy:* (1) *Available clinical use guidelines,* (2) *indications and contraindications for use, and* (3) *assessment of associated radiation dose risk.* ***Materials and Methods:*** *Three focused questions were developed to address the aims. A systematic literature review*

Figure 3: Indications of who reports on the scans (CBCT) and who traces and plots the IAN.



Assessment

Who actually assesses the risk?

- Clinical
  - OH, Perio
  - Soft tissues and function
  - Hard tissue bone mapping, adjacent dentition
- Radiologic LCPA, DPT or CBCT?

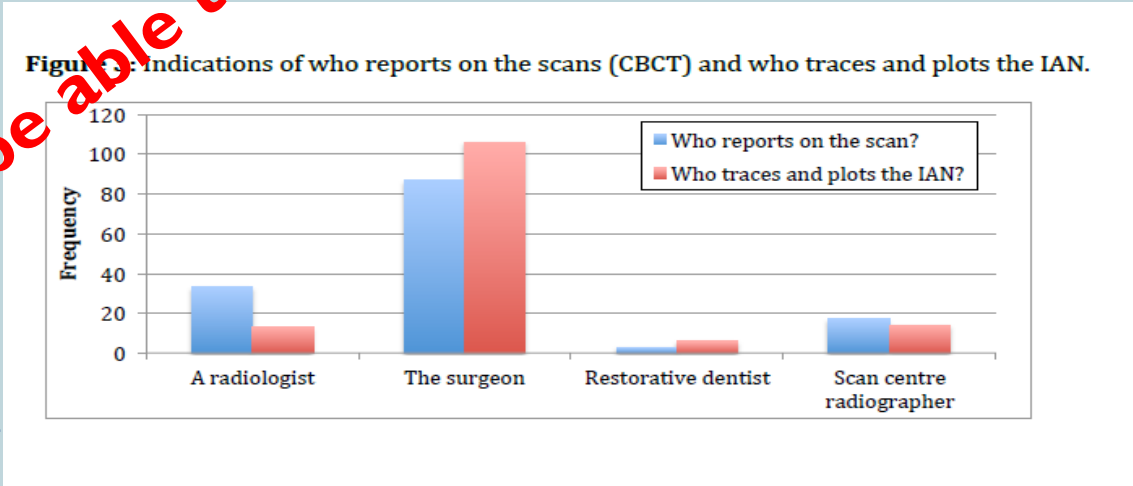
  Guidelines Faculty Dental Practitioners 9FGDP SEDENTEXTCT
- Informed consent
- Who assesses the risk?

Cone Beam Computed Tomography in Implant Dentistry: A Systematic Review Focusing on Guidelines, Indications, and Radiation Dose Risks

Michael M. Bornstein, PD Dr Med Dent[1]/William C. Scarfe, BDS, FRACDS, MS[2]/ Vida M. Vaughn[3]/Reinhilde Jacobs, DDS, MSc, PhD, Dr hc[4]

*Purpose: The aim of the paper is to identify, review, analyze, and summarize available evidence in three areas on the use of cross-sectional imaging, specifically maxillofacial cone beam computed tomography (CBCT) in pre- and postoperative dental implant therapy: (1) Available clinical use guidelines, (2) indications and contraindications for use, and (3) assessment of associated radiation dose risk. **Materials and Methods:** Three focused questions were developed to address the aims. A systematic literature review*

You MUST be able to read your own CBCTs



Prevention of Implant IAN

Surgeon assessment

- Panoral worse than CBCT
- Both led to underestimation of safety zone

RESEARCH ARTICLE Open Access

The reliability of surgeons to avoid traumatic insertion of dental implants into high-risk regions: a panoramic radiograph study

Firas A. Jamil[1*], Jamal A. Mohammed[1], Thair A. Hasan[1] and Mohammed G. Rzoqi[2]

Table 2 Descriptive statistics for the outcome variables

Variable	N	Premolars	Molars	Minimum (mm)	Maximum (mm)	Mean ± SD	*P* value
Underestimated sites:	**138**	**55**	**83**	0.2	6	1.58 ± 1.43	0.008[a,**]
Posterior maxilla	67	37	30				
Posterior mandible	71	18	53				
Overestimated sites:	**10**	**3**	**7**	−0.2	−1.6	0.36 ± 0.44	
Posterior maxilla	1	1	0				
Posterior mandible	9	2	7				

[a]By T-test (2-tailed)
[**]Highly significant

≥ 2 mm (26.1%). In the posterior mandible, overestimation was significantly higher than posterior maxilla. Five cases with transient paresthesia were reported in the mandibular overestimated implants.

Conclusions: This study specified that surgeon's choice of implants length, based on panoramic radiographs, was reliable regarding the incapability to insert implants with further length in the majority of underestimated cases, the low percent of overestimated measurements, and the minor associated complications.

CBCT Assessment of anterior loop

- Two thousand seven hundred eighty-four articles were further excluded by the reviewers after screening the abstracts which resulted in 37 studies
- Two thousand five hundred three subjects with anterior loop were found, which approximates 38% with 48.4% bilateral, 27.8% right side, and 23.8% left side.
- The loop distribution in males and females was also found to be different.
- There was highly significant ($P < 0.001$; $I2 = 98.81\%$) heterogeneity found in the included studies.
- Variations were found in the prevalence, length, gender, and side distribution of anterior loop in various populations.
- This systematic review highly recommends **NOT relying on any average values** and the clinician should compulsorily make use of imaging modalities available in each and every case, wherever surgical procedure is to be performed near mental foramen region.

Oral and Maxillofacial Surgery
https://doi.org/10.1007/s10006-020-00915-x

REVIEW ARTICLE

Identification of anterior loop in different populations to avoid nerve injury during surgical procedures—a systematic review and meta-analysis

Sunil Kumar Mishra[1] · Rajvi Nahar[1] · Reetika Gaddale[2] · Ramesh Chowdhary[3]

Received: 2 September 2020 / Accepted: 21 October 2020

Abstract
Exact recognition of the anterior loop is very important to avert any injury to the neurovascular bundle during surgical procedures. The purpose of this review was to evaluate the prevalence and length of the anterior loop in different populations. A comprehensive search of Medline/Pubmed and Cochrane database was done. The focused question was the presence of anterior loop (including loop length) of the inferior alveolar nerve in mental foramen region in CBCT images of the various subjects. Articles related to the presence of anterior loop (including loop length) were only included. Initial literature search resulted in 3024 papers, after removing duplicate articles, 2821 articles were left. Two thousand seven hundred eighty-four articles were further excluded by the reviewers after screening the abstracts which resulted in 37 studies. Hand searching resulted in 2 additional papers. Seven full-text articles were excluded for not fulfilling the inclusion criteria. Finally, 32 articles were included in the review. Two thousand five hundred three subjects with anterior loop were found, which approximates 38% with 48.4% bilateral, 27.8% right side, and 23.8% left side. The loop distribution in males and females was also found to be different. There was highly significant ($P < 0.001$; $I^2 = 98.81\%$) heterogeneity found in the included studies. Variations were found in the prevalence, length, gender, and side distribution of anterior loop in various populations. This systematic review highly recommends not relying on any average values and the clinician should compulsorily make use of imaging modalities available in each and every case, wherever surgical procedure is to be performed near mental foramen region.

Keywords Anterior loop length · Cone-beam computed tomography · Dental implant · Mandibular canal · Mental foramen

Nerve Injury is Preventable with adequate radiographic planning

- To avoid nerve injury during surgery in the foraminal area, guidelines were developed based on the literature with respect to verifying the position of the mental foramen and validating the presence of an anterior loop of the mental nerve
- These guidelines included leaving a 2 mm zone of an implant and the coronal aspect of the nerve;
  - observation of the inferior alveolar nerve and m panoramic and periapical films prior to implant p
  - use of CT scans when these techniques do not p with respect to the position of the nerve
  - surgical corroboration of the mental foramen's p anterior loop of the mental foramen is suspected or if it is unclear how much bone is present corc foramen to establish a zone of safety (in millimet placement; once a safety zone is identified, implar anterior to, posterior to, or above the mental for to placing an implant anterior to the mental fora deeper than the safety zone, the foramen must b exclude the possibility that an anterior loop is pr
  - In general, altered lip sensations are preventable foramen is located and this knowledge is employ performing surgical procedures in the foraminal a

J Periodontol • December 2006

Review

The Mental Foramen and Nerve: Clinical and Anatomical Factors Related to Dental Implant Placement: A Literature Review

Gary Greenstein*† and Dennis Tarnow†

Table 1.

Locations of the Mental Foramen: Percentage of Occurrence

Study	Population	N	Apical to Second Premolar (%)	Between Apices of Premolars (%)	Other Locations (%)
Neiva et al.[14]	Caucasian	22	42	58	
Fishel et al.[20]	Caucasian	1,000	18.9	70.4	Apex first premolar: 3.3 Mesial to first premolar: 1.5 Distal to second premolar: 6.0
Wang et al.[22]	Chinese	100	59	21	Between premolar/molars: 19 By the molar: 1
Ngeow and Yuzawati[23]	Malay	322	69	20	Apex first premolar: 3.4 By the molar: 1 Between premolar/molar: 6.6
Kekere-Ekun[24]	Nigerian	604	55.63	26.99	Apex first premolar: 1.66 Between premolar/molars: 12.3 By the molar: 3.3 Mesial to first premolar: 0.17
Bergman et al.[25]	Unknown	1,414	61	36	Canine: <1; molar: 2; first bicuspid: 0.15

Does CBCT preoperative assessment minimise risk?

- Reduced medicolegal claims since the introduction of CBCT

Clinical Oral Investigations (2019) 23:399–404
https://doi.org/10.1007/s00784-018-2448-4

ORIGINAL ARTICLE

CrossMark

Did malpractice claims for failed dental implants decrease after introduction of CBCT in Finland?

Magdalena Marinescu Gava[1,2] · Anni Suomalainen[1,2] · Tapio Vehmas[1,3] · Irja Ventä[4]

Received: 22 January 2018 / Accepted: 12 April 2018 / Published online: 20 April 2018

Abstract

Objectives To examine the role of cone beam computed tomography (CBCT) in preventing failures in implant treatment. We hypothesize that the number of malpractice claims related to dental implant treatment would decrease after the first CBCT device came available in 2002 in Finland.

Material and methods Data concerning malpractice claims related to dental implant treatment during the years 1997–2011 were collected from the Finnish Patient Insurance Centre ($N = 330$ subjects). We selected the cases that might have benefitted from the use of CBCT examination. These cases ($n = 131$) led to financial compensation due to permanent inferior alveolar nerve injury, improper implant position, or insufficient amount of bone for the implant. The annual total number of inserted dental implants, CBCT devices, and CBCT examinations in Finland were drawn from the national registers and used to estimate the impact of CBCT in preventing treatment failures.

Results The most common reason for all failures ($n = 268$ implants) was an improper implant position (46.3%). The most common area of malpractices was upper front teeth (34%). We have noticed a fall in the rate of compensable malpractice cases concerning implant failure, simultaneously with CBCT technology emerging on the market.

Conclusions There may be an association between the increasing availability of CBCT equipment and the reducing frequency of compensable malpractice claims.

Clinical relevance It is possible that the use of CBCT may result in fewer compensable malpractice claims.

Marinescu Gava M, Suomalainen A, Vehmas T, Ventä I. Did malpractice claims for failed dental implants decrease after introduction of CBCT in Finland? Clin Oral Investig. 2019 Jan;23(1):399-404. doi: 10.1007/s00784-018-2448-4. Epub 2018 Apr 20. PMID: 29679231.

Systematic review of CBCT in assessment Mental foramen

- 3 patterns

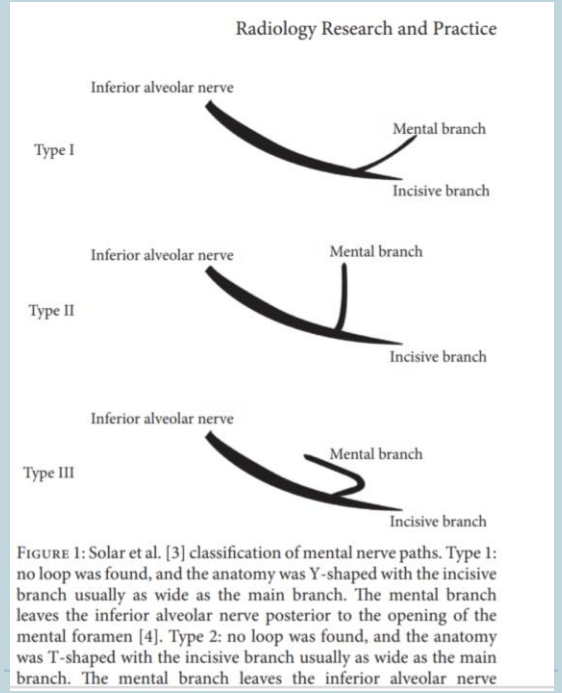


FIGURE 1: Solar et al. [3] classification of mental nerve paths. Type 1: no loop was found, and the anatomy was Y-shaped with the incisive branch usually as wide as the main branch. The mental branch leaves the inferior alveolar nerve posterior to the opening of the mental foramen [4]. Type 2: no loop was found, and the anatomy was T-shaped with the incisive branch usually as wide as the main branch. The mental branch leaves the inferior alveolar nerve

Hindawi
Radiology Research and Practice
Volume 2021, Article ID 8897275, 10 pages
https://doi.org/10.1155/2021/8897275

Hindawi

Review Article

Evaluation of Mental Foramen with Cone Beam Computed Tomography: A Systematic Review of Literature

Antoinette Pelé[1,2] **Pierre-Alexandre Berry**[1,2] **Charles Evanno**[2,3] **and Fabienne Jordana**[1]

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[2]*Private Practice, 34 Rue Jules Verne, 44700 Orvault, France*
[3]*Oral Implantology, Dentistry Department, University Health Centre, University of Nantes, Nantes, France*

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Academic Editor: Lorenzo Faggioni

Purpose. The aim of this systematic review is to assess whether the anatomy of mental foramen is precisely evaluable with cone beam computed tomography (CBCT) before implantation in humans. *Methods*. A systematic review was carried out to evaluate the anatomy of mental foramen (size, position, symmetry, anterior loop, and accessory mental foramen or multiple mental foramina). According to Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines, an electronic search of three databases (Medline, Web of Science, and Cochrane Library) was undertaken until June 2020 and was supplemented by manual searching. Two reviewers will independently perform the processes of study inclusion, data extraction, and quality assessment. Systematic reviews, studies about children, and case reports were excluded. Only studies using CBCT to do preoperative evaluation were selected. *Results*. From 728 potentially eligible articles, 72 were included in the qualitative analysis and quantitative synthesis. This systematic review provided an assessment of the anatomy of the mental foramen. The mental foramen was located mostly between the two premolars (between 50.4% and 61.95%) or apically to the second premolar (from 50.3% to 57.9%). The mean diameter of the mental foramen was bigger in males than in females; the difference between them could reach 0.62 mm. The anterior loop seemed to be longer in males (between 0.87 ± 1.81 and 7.25 ± 2.02 mm) than in females (between 0.81 ± 1.18 and 6.52 ± 1.63 mm) and with the presence of teeth (from 0.91 ± 1.18 to 2.55 ± 1.28 for dentate people and from 0.25 ± 0.61 to 2.40 ± 0.88 mm for edentate population). The anterior loop and the accessory mental foramina were detected more frequently with CBCT than panoramic X-ray: only between 0.0 and 48.6% AMFs detected with CBCT were also seen with panoramic images. *Clinical Significance*. The mental foramen (MF) is an important landmark for local anesthesia and surgical and implantology procedures. Its location, morphology, and anatomical variations need to be considered to avoid mental nerve injury. The aim of this review is to evaluate the mental foramen using CBCT through a systematic literature review to improve knowledge of this complex area for the clinician.

Prevention implant IANI

Anterior extension of IAN

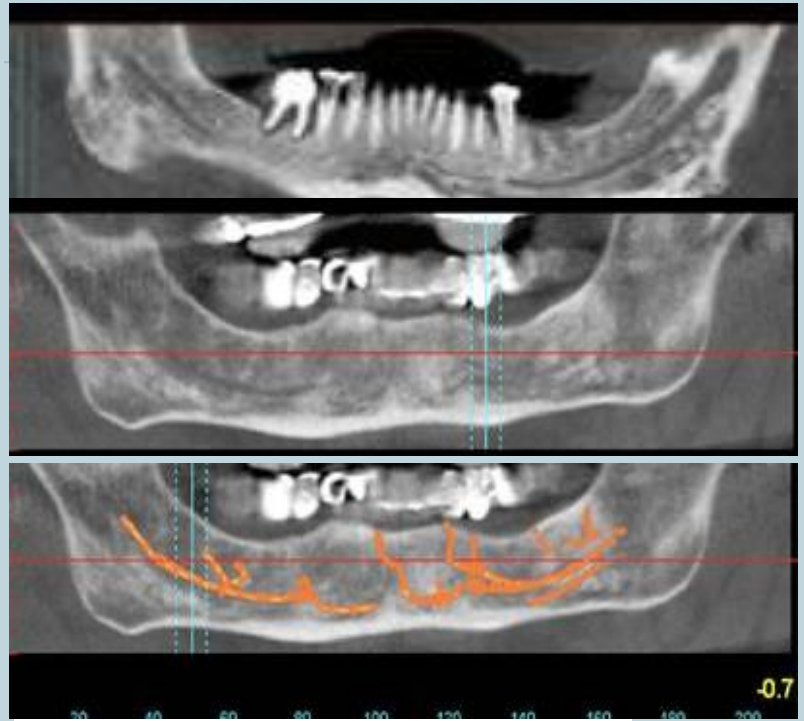
Data from five hundred (500) consecutive patients sent to i-dontics center from 9 centers located in 3 states for 3D dental CT studies, were evaluated

Nearly 97% of all mandibles had an anterior extension; nine patients did not have a measureable anterior extension of the IAN as seen on a 3D cone beam study.

Fourteen patients (4.73%) did not have an extension on the right side; eleven patients (3.72%) did not have an extension on the left side.

The **average length of the anterior extension extending from the mesial rim of the mental foramen is 12.0 mm** on the right and 11.8 mm on the left

A **continuous loop**, defined as an extension of the canal that emanates from both the right and left mental foramina and is seen to connect in the midline was viewed on **77 patients (26.01%).**



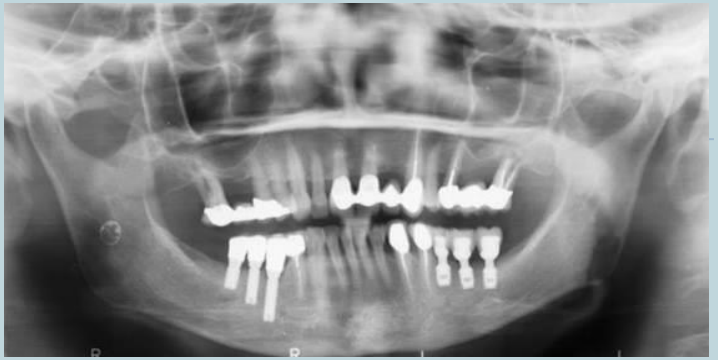
Winter, Baradarian, Sivitz 2014

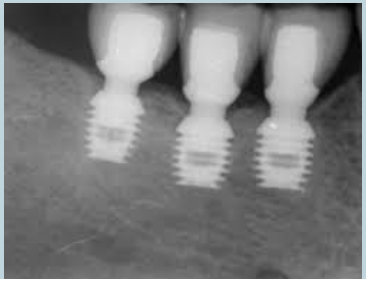
Dental 3D Cone Beam CT Imaging: Part IV Anterior Extension of IAN from Mental Foramen (Pre-surgical analysis for the insertion of dental implants)

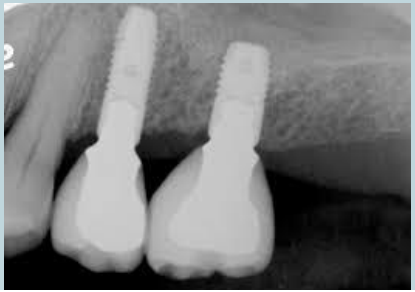
Managing risk

Short implants

- Strong evidence to support the use of short implants 5-8mm for mandibular cases
- Moderate evidence to support maxillary short implants (prevents the need for maxillary grafting)







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*,†

Survival of short dental implants in atrophied jaw: a systematic review

Fatimah Assaf[1], Guilherme Siqueira-Ibelli[1], Rogério Margonar[2], Pâmela Letícia Santos[3*], Ana Paula de Souza-Faloni[4], Thallita Pereira-Queiroz[3]

Chen MH, Shi JY **Clinical and Radiological Outcomes of Implants in Osteotome Sinus Floor Elevation with and without Grafting: A Systematic Review and a Meta-Analysis.** J Prosthodont. 2017 Jan 12. doi: 10.1111/jopr.12576. [Epub ahead of print] Fan T et al **Short Implants (5 to 8 mm) Versus Longer Implants (>8 mm) with Sinus Lifting in Atrophic Posterior Maxilla: A Meta-Analysis of RCTs.** Clin Implant Dent Relat Res . 2017 Feb;19(1):207-215. doi: 10.1111/cid.12432. Epub 2016 Jun 13.

Intra-operative risk factors

- LA blocks
- Drills longer than implant by at least 1.5-2 mm
- Continued implant bed drilling
  - Stop drilling @ 60% use drill guide to assess position (ITI)
- Acute pain during implant bed preparation even with efficacious LA
- Shooting nerve pain or paraesthesia along IAN to chin
- Implant bed Bleeding

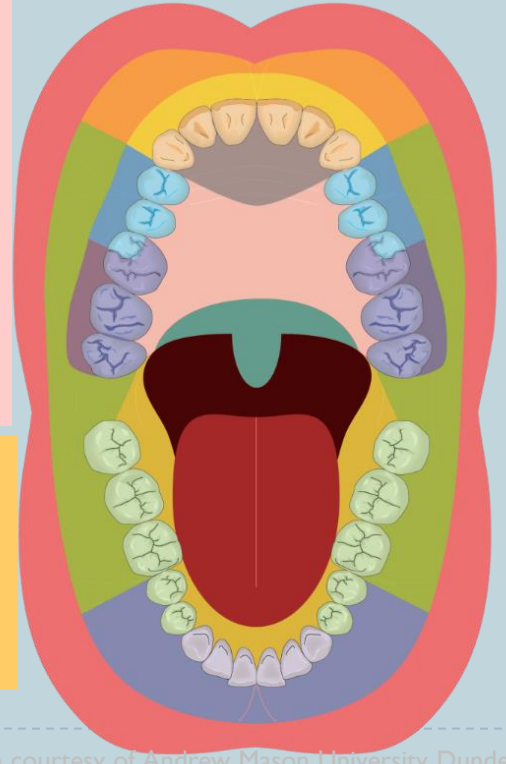
Intraoperative risk factors

LA related nerve injuries –can be mitigated by avoiding blocks

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

LA related nerve … avoiding blocks
Infiltration dent … e and procedure

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

IDBS needed for
Posterior mandibular m…
Endodontic procedure… require IDBs or higher techniques (Gow Gate… Akinosi)

…and 8s for **perio, restorations or**

…uccal infiltration and Lidocaine 2% …ns OR for **extractions**

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…canines incisors for **perio,**

…ion (incisal nerve block …jacent not in the mental …over region. If fails repeat or …filtration OR for **extractions,**

▶ Illustration modified from f…

Journal section: Oral Surgery
Publication Types: Research
doi:10.4317/jced.54330
http://dx.doi.org/10.4317/jced.54330

A randomized controlled trial comparing nerve block and mandibular infiltration techniques in posterior mandible implant surgeries

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The use of the mandibular infiltration anesthetic technique in adults

John G. Meechan, BSc, BDS, PhD, FDSRCS, FDSRCPS

ABSTRACT

Background. The author describes the use of the infiltration anesthetic technique to anesthetize mandibular teeth in adults and explores its mechanism of action.
Methods. The author reviewed articles describing randomized controlled trials of the mandibular infiltration anesthetic technique in healthy participants.
Results. The author found that using the mandibular infiltration anesthetic technique can produce anesthesia in adult mandibular teeth. The success was dose dependent and the choice of anesthetic solution was significant; 4 percent articaine with 1:100,000 epinephrine was more effective than 2 percent lidocaine with 1:100,000 epinephrine. Combining buccal and lingual infiltrations increased success in the mandibular

Local anesthesia in the mandible traditionally has been provided by means of one of the inferior alveolar nerve block (IANB) techniques such as the Halsted, Gow-Gates or Akinosi-Vazirani methods. The regional block anesthetic technique may be more difficult technically to perform than is the infiltration anesthetic technique, and it has additional disadvantages, including the potential for causing nerve damage and the failure to counter any accessory nerve supply such as the dual supply of midline structures. The infiltration anesthetic technique often is used in the maxilla, although the use of regional blocks is possible.

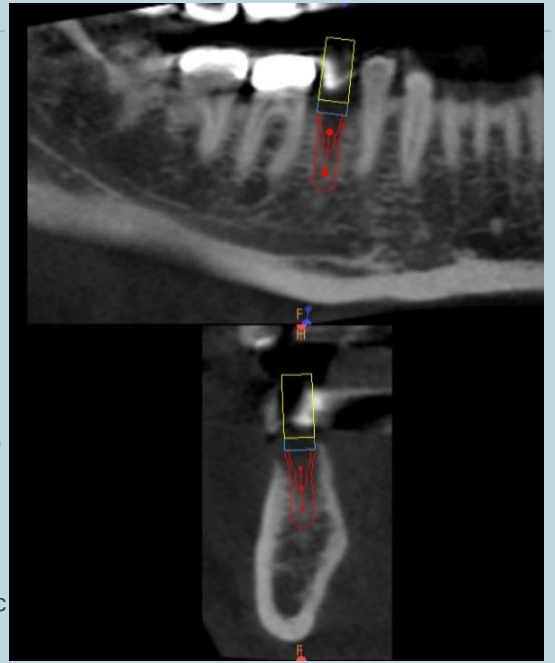
A reason that infiltration techniques may not be the first choice in the adult mandible is because practitioners tend to think that the thick cortical plate prevents diffusion of solution into the cancellous bone and, therefore, to the nerves supplying the pulps of the teeth. There are holes in the body of the mandible, however, and these could permit diffusion of solution into the cancellous space. Such holes

block provides a more profound analgesia than mandibular infiltration. When placing implants under mandibular infiltration, as getting closer to the canal does not increase the feeling of pain, it is not recommended to use the presence of pain as a preventive resource to avoid inferior alveolar nerve injuries.

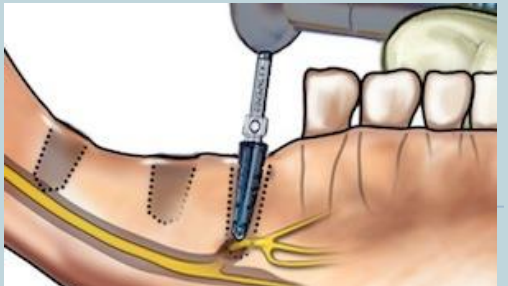
Managing Intraoperative risk

Intraoperative risks

- **Pain on injection of LA**
- Sudden give during preparation
  - Mylohyoid ridge fracture
  - Into Inferor alveolar nerve canal
- Brisk persistent bleed on preparation
  - Consider delaying placement of implants
- **Intense sudden pain during implant bed** preparation- Any protrusion into the IDC or breech causes severe neuralgic type pain intra-operatively FUNNY BONE PAIN!
- Stop and reassess



Leckel M, Kress B, Schmitter M. Neuropathic pain resulting from implant placement: case report and diagnostic conclusions. J Oral Rehabil. 2009 Jul;36(7):543-6. Epub 2009



KING'S College LONDON

Intra-operative risk factor
brisk bleed during implant bed preparation

Gintaras Juodzbalys
Hom-Lay Wang
Gintautas Sabalys
Antanas Sidlauskas
Pablo Galindo-Moreno

Inferior alveolar nerve injury associated with implant surgery

Authors' affiliations:
Gintaras Juodzbalys, Gintautas Sabalys, Department of Maxillofacial Surgery, Lithuanian University of Health Sciences, Kaunas, Lithuania
Hom-Lay Wang, Department of Periodontics and

Key words: alveolar nerve, cranial nerve injuries, dental implants, inferior, mandibular canal, mandibular nerve, paresthesia

Abstract

perforation of the mandibular **canal during drilling or positioning the implant close tooth canal** subsequent formation of an adjacent hematoma (Lamas Pelayo et al. 2008). Khawaja & Renton (2009)
"cracking" of the IAN canal roof by its close proximity to preparation of the implant bed
(millimeters)may cause hemorrhage into the canal or deposition of debris which may compress and cause ischemia of the nerve

Table 2. **Data for subjects and inferior alveolar nerve injury**

Subject no.	Gender	Age (years)	Affected JDS no.	Duration post-injury	Intraoperative risk factor	Etiological factor
1	Female	44	30	18 h	Change pre-planned implant size (wider)	Implant drill
2	Female	56	18	52 h	Not identified	Implant (partial intrusion)
3	Male	60	19	26 h	Pain during bone preparation, bleeding	Implant (partial intrusion)
4	Female	48	19	50 h	Not identified	Wrong operation technique (suturing)
5	Male	52	30	28 h	Bleeding	Implant drill
6	Male	47	31	36 h	Drill slippage, bleeding	Implant (partial intrusion)
7	Male	36	29	14 h	Not identified	Not identified
8	Male	61	20	46 h	Bleeding	Implant (full intrusion)
9	Female	47	21	13 h	Change pre-planned implant size (longer), bleeding	Implant drill
10	Male	55	19	2 weeks	Not identified	Implant, infection
11	Female	58	31	51 h	Drill slippage, bleeding	Implant drill
12	Male	63	18	5 days	Not identified	Implant (too close)
13	Female	65	19	14 h	Pain during bone preparation, bleeding	Implant (partial intrusion)
14	Female	44	20	24 h	Pain during local anesthesia	Injection needle
15	Female	46	30	14 h	Not identified	Implant (partial intrusion)
16	Male	53	28	14 h	Change pre-planned implant size (longer), bleeding	Implant (partial intrusion)

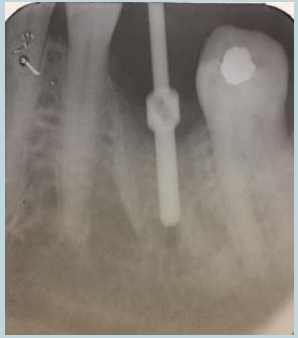
JDS, jaw dental segment.

Elian N, Mitsias M, Eskow R et al. Unexpected return of sensation following 4.5 years of paresthesia: case report. Implant Dent 2005; 14: 364–367

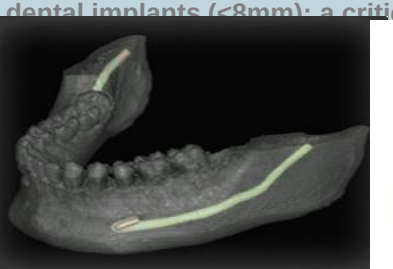
Managing Intraoperative risk

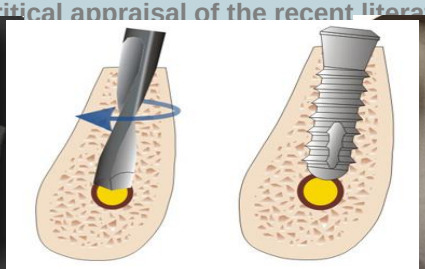
Implant prep and placement technique

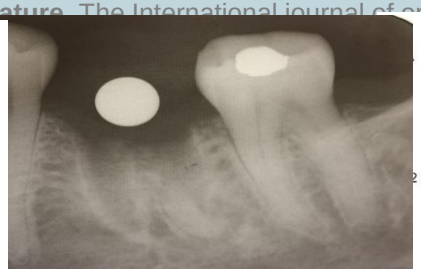
- High risk mandibular premolars and molars
- Infiltration anaesthesia
- **Maximise safety zone**
  - More than 2 mm as in most implant systems drills are 1.5 mm longer than implants Short implants
  - Implants should not need to be longer than 8 mm
  - Use short implants
  - Use system where drill shorter than implant
- **Be aware of intraoperative risk factors**
  - Bleeding, sudden drop
  - Severe pain
- **Avoid complex treatment**
  - Bone grafting both mental and post mandibular have high morbidity
  - Lateralisation of IAN has high morbidity and poor evidence
  - Many nerve injuries are caused by over use of extensive soft tissue flaps

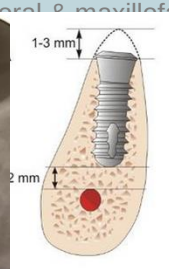


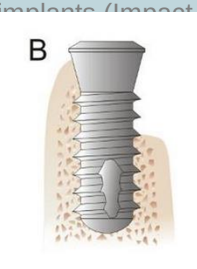
Leckel M, Kress B, Schmitter M. Neuropathic pain resulting from implant placement: case report and diagnostic conclusions. J Oral Rehabil. 2009 Jul;36(7):543-6. Epub 2009 M Srinivasan, Lydia Vazquez Philippe Rieder, Osvaldo moraguez, Jean-Pierre Bernard Urs C. Belser **Efficacy and predictability of short dental implants (<8mm): a critical appraisal of the recent literature.** The International journal of oral & maxillofacial implants (Impact Factor: 1.91). 11/201

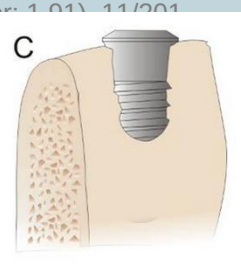








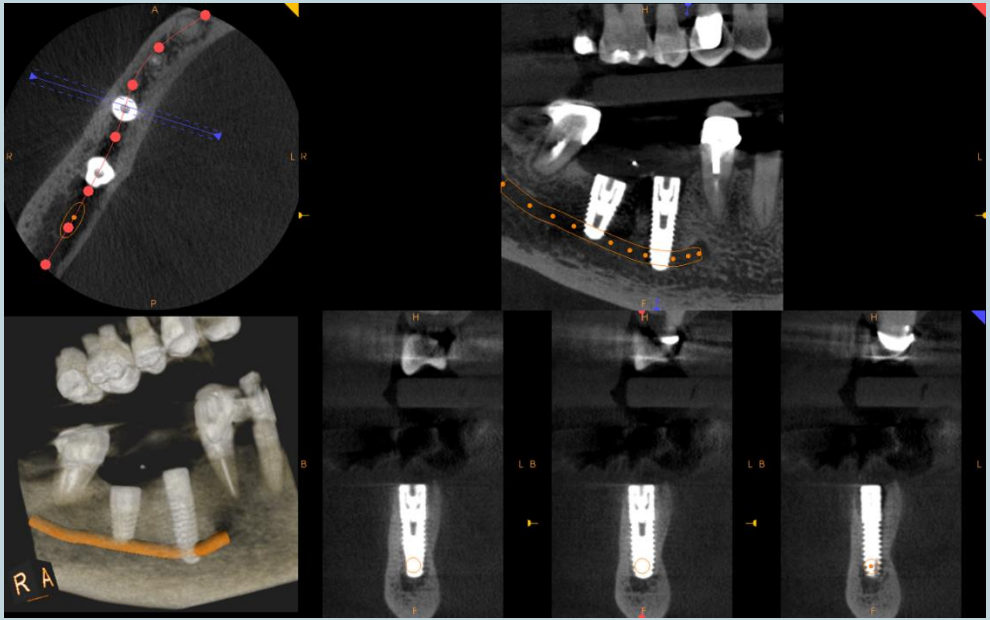




Managing Intraoperative risk

Computer guided surgery?????

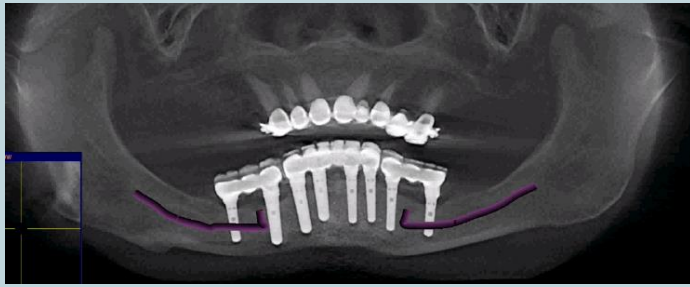
- Many types software
  - Helios
  - CDent
  - Simplant
- BUT still many injuries happen!



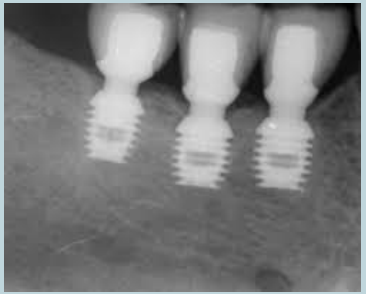
KING'S College LONDON

Evidence base
for managing Intraoperative risk

- 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



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

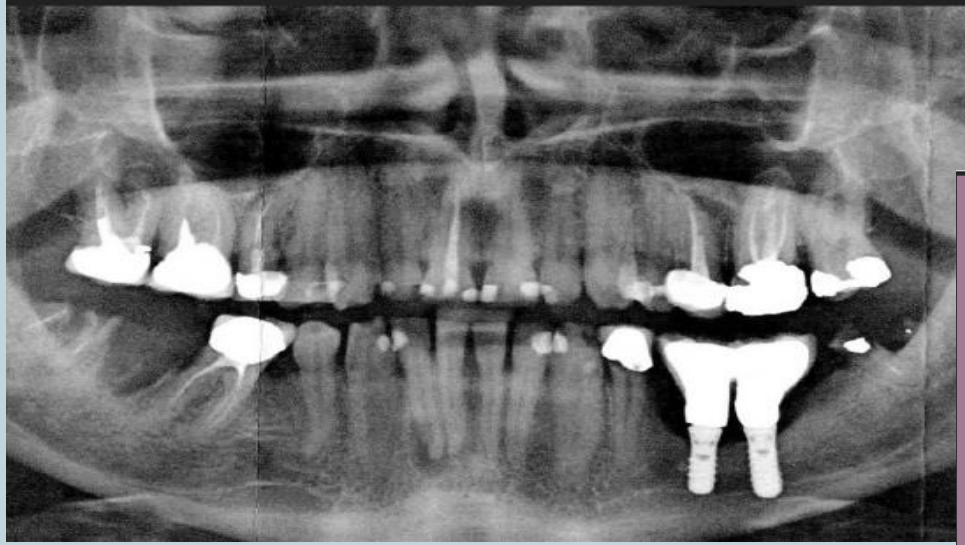


Guidance for prevention of implant nerve injury FOR.org, ITI and ADI

FOR SCIENCE EDUCATION HUMANITY
FOUNDATION FOR ORAL REHABILITATION

ePapers

Home check within 24 hours surgery



ADI Association of Dental Implantology

Risk Management and Prevention of Inferior Alveolar Nerve Injury (IANI) associated with Dental Implant Surgery

Author of original consultation paper: Professor Tara Renton, Kings College, London
Edited by the ADI Guidelines Subcommittee: Ucer, C; Wright, S; Scher, E; Slade, K.

Diagnosis and Management of Inferior Alveolar Nerve Damage Associated with Dental Implant Surgery

Tara Renton, Maria Devina

Prof. Tara Renton, BDS, MDSc, PhD, FDS, RCS, FRACDS (OMS), FHEA (Specialist in Oral Surgery) is a dentist with a particular interest in trigeminal nerve injuries and pain. She was awarded her chair at King's College London in 2006. She has established an academic training program, which has taught 5 Academic Oral Surgery Spls., of whom 3 are undertaking PhDs. She was recently appointed lead for orofacial pain at INPUT, a nationally recognised pain management program based at St Thomas' Hospital. Tara is the national advisor for oral surgery. She is an elected council member for BAOS and is an elected member of the RCS England FDS Board.

Maria Devina, BDS (Hnd), MFDS, RCSEd is an Academic Clinical Fellow/Specialty Registrar in Oral Surgery at Kings College Hospital. Her current research activity involves iatrogenic trigeminal nerve injury. She qualified from Newcastle University in 2009 and worked in a variety of hospital dentistry, general practice and maxillofacial surgery posts before taking up her specialty training in Kings College Hospital.

CLINICAL DILEMMAS

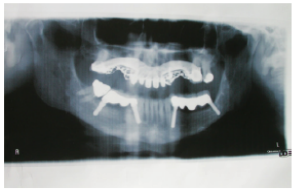


Fig. 1: OPT of patient with bilateral inferior alveolar nerve injury due to implant surgery

INTRODUCTION

Minimizing adverse events related to patient care is paramount. Trigeminal nerve injuries related to dentistry are a recognized complication and avoidable in most instances. Implant treatment and nerve injuries are an increasingly common cause for medico-legal complaints and resultant compensation to patients with lifelong orofacial neuropathy (Fig. 1).

The inferior alveolar nerve (IA N) is at risk from a variety of dental procedures as it is contained within a bony canal predisposing it to ischaemic trauma and a higher incidence of permanent damage. Causes of inferior alveolar nerve injury (IANI) include local anaesthetic injections, third molar surgery, implants, endodontics, ablative surgery, trauma and orthognathic surgery. IAN neuropathy related to third molar surgery or inferior alveolar block injections is usually temporary but can persist and

and drinking; in fact just about every social interaction we take for granted. Thus these injuries have a significant negative effect on the patient's self-image, and quality of life as well as significant psychological effects (Renton & Yilmaz 2011).

At the same time the predictability of dental implants and the expectations and demands of patients have moved to a position where dental implants are now seen as a routine treatment option when considering the restoration of missing teeth (Ghoi et al. 2011). More recently, complaints and legal action against dentists in the United Kingdom have increased significantly and many of these arise from implant-related treatment (Ghoi et al. 2011), which in part is contributed to by 'implant tourism' (Barrowman et al. 2010).

Although nerve injury occurs in a minority of patients undergoing implant therapy, the consequences can be devastating for both

scarce and mainly address the prescription of implants rather than their application (Gothreborn et al. 2008, Dawson & Cardaci 2006, van Waas et al. 1991, Academy of Osseointegration 2010).

The incidence of implant-related nerve injury is not fully established and is generally based on empirical estimates or rare prospective case series (Renton et al. 2012, Rubenstein & Taylor 1997, Wismeijer et al. 1997, Dao & Meiller 1998, Barting et al. 1999, Walton 2000, Zicconti & Assaal 2001, von Arx et al. 2005, Abarca et al. 2006, Greenstein & Tarnow 2006, Hagedus & Diacidus 2006, Tay & Zuniga 2007, Hillerup 2007, Misch et al. 2008, Alhassani & Aghumdi 2010, Misch 2010, Juodzbalys et al. 2011, Palma-Carrio et al. 2011, Juodzbalys et al. 2011, Park et al. 2012, Balshi 1985, Barbari et al. 1995, Dalcanho 1995, Fukuda & Ichinohe 2012). The incidence of implant-related IANI varies from 0-40% (Dawson & Cardaci 2006, van Waas et al.

Overview

Intro Trigeminal nerve injuries

Intro to neuropathic pain

Implant related nerve injury risk factors

Prevention of Implant related nerve injuries

Treatment of Implant related nerve injury

Management of Trigeminal Post-traumatic neuropathy

Currently, there is **no consensus** on the optimal management of neuropathic pain exists and practices vary greatly worldwide.

Possible explanations for this include **difficulties in developing agreed diagnostic protocols and the coexistence of neuropathic, nociceptive and, occasionally, idiopathic pain in the same patient**.

- **Treat the patient with the nerve injury! NOT the implant!**
- **Prevention is best!**
- **Treatment must depend upon the mechanism and duration of nerve injury**
- **Holistic approach**

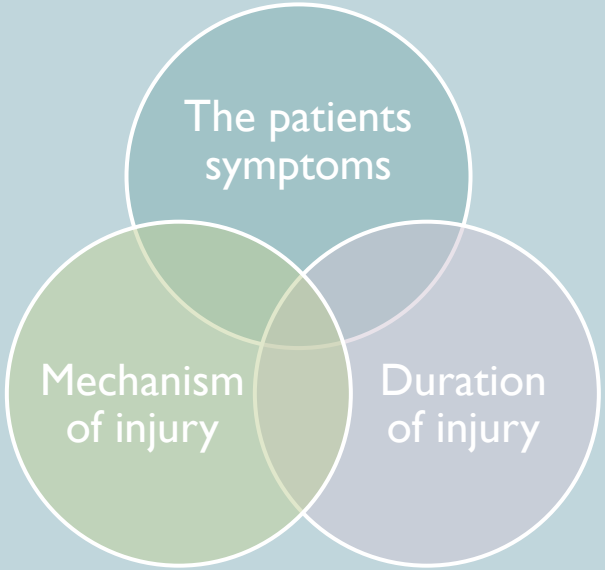
•Treat
  - *Pain*
  - *Functional disability*
  - *Psychological impact*

•Counselling
  - *Reaffirm nerve injury is permanent*
  - *Be honest with the patient*
  - *Reassurance and explanation*

•Medical for pain +/- depression
  - *Topical*
  - *Systemic*

•Surgical

•**Remove implant or Endo within 30 hours**




Int. J. Oral Maxillofac. Surg. 2012; 41: 629–637
doi:10.1016/j.ijom.2011.11.002, available online at http://www.sciencedirect.com

International Journal of Oral & Maxillofacial Surgery

Review Paper
Oral Surgery

Managing iatrogenic trigeminal nerve injury: a case series and review of the literature

T. Renton, Z. Yilmaz
King's College London Dental Institute, Denmark Hill Campus, London, UK

T. Renton, Z. Yilmaz: Managing iatrogenic trigeminal nerve injury: a case series and review of the literature. Int. J. Oral Maxillofac. Surg. 2012; 41: 629–637.


Int. J. Oral Maxillofac. Surg. 2018; 47: 794–801
https://doi.org/10.1016/j.ijom.2017.10.030, available online at https://www.sciencedirect.com

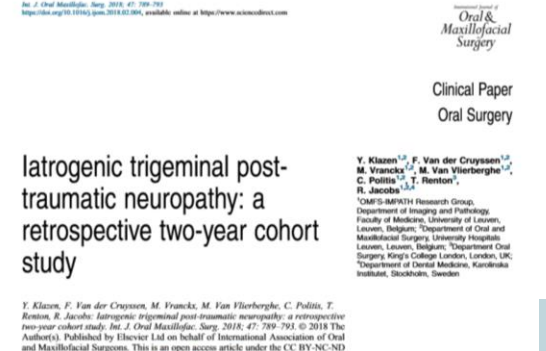
International Journal of Oral & Maxillofacial Surgery

Clinical Paper
Oral Surgery

Treatment modalities and risk factors associated with refractory neurosensory disturbances of the inferior alveolar nerve following oral surgery: a multicentre retrospective study

T. Hasegawa[1], S. I. Yamada[2], N. Ueda[3], S. Soutome[4], M. Funahara[4], M. Akashi[1], S. Furuno[5], H. Miyamoto[1], S. Hayashida[6], R. Amano[1], K. Mori[6], Y. Kojima[7], H. Kurita[3], T. Kirita[2], M. Umeda[4], Y. Shibuya[8], S. Fujita[9], T. Komori[1] Japanese Study Group of Cooperative Dentistry with Medicine (JCDM)
[1]Department of Oral and Maxillofacial Surgery, Kobe University Graduate School of Medicine, Kobe, Japan; [2]Department of Dentistry and Oral Surgery, Shinshu University School of Medicine, Nagano, Japan; [3]Department of Oral and Maxillofacial Surgery, Nara Medical University, Kashihara, Nara, Japan; [4]Department of Clinical Oral Oncology, Nagasaki University Graduate School of Biomedical Sciences, Nagasaki, Japan; [5]Department of Oral and Maxillofacial Surgery, Nagoya City University Graduate School of Medical Sciences, Nagoya, Japan; [6]Department of Oral and Maxillofacial Surgery, Wakayama Medical University,

T. Hasegawa, S.I. Yamada, N. Ueda, S. Soutome, M. Funahara, M. Akashi, S. Furuno, H. Miyamoto, S. Hayashida, R. Amano, K. Mori, Y. Kojima, H. Kurita, T. Kirita, M. Umeda, Y. Shibuya, S. Fujita, T. Komori: Treatment modalities and risk factors


Int. J. Oral Maxillofac. Surg. 2018; 47: 789–793
https://doi.org/10.1016/j.ijom.2018.02.004, available online at https://www.sciencedirect.com

International Journal of Oral & Maxillofacial Surgery

Clinical Paper
Oral Surgery

Iatrogenic trigeminal post-traumatic neuropathy: a retrospective two-year cohort study

Y. Klazen[1,2], F. Van der Cruyssen[1,2], M. Vranckx[1,2], M. Van Vlierberghe[1,2], C. Politis[1,2], T. Renton[3], R. Jacobs[1,2,4]
[1]OMFS-IMPATH Research Group, Department of Imaging and Pathology, Faculty of Medicine, University of Leuven, Leuven, Belgium; [2]Department of Oral and Maxillofacial Surgery, University Hospitals Leuven, Leuven, Belgium; [3]Department Oral Surgery, King's College London, London, UK; [4]Department of Dental Medicine, Karolinska Institutet, Stockholm, Sweden

Y. Klazen, F. Van der Cruyssen, M. Vranckx, M. Van Vlierberghe, C. Politis, T. Renton, R. Jacobs: Iatrogenic trigeminal post-traumatic neuropathy: a retrospective two-year cohort study. Int. J. Oral Maxillofac. Surg. 2018; 47: 789–793.

Mechanism
Cause and duration

URGENT treatment < 30 hours

- Any known or Suspected nerve trauma
- Implants
- Endodontics (neuropathy may develop 2-3 days post treatment)

- **Within 2 weeks**
- Buccal approach causing Lingual nerve
- Inferior alveolar nerve injuries related to third molar surgery

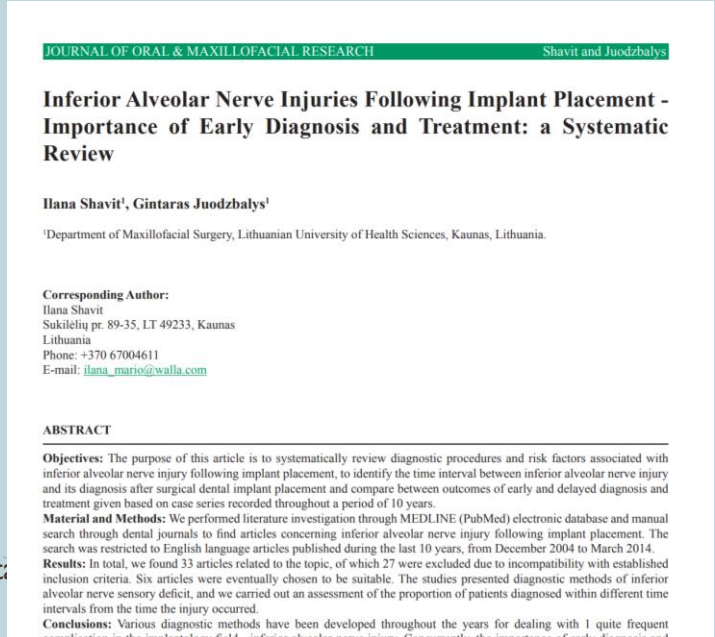
Consent patient properly…forearmed is for warned
Risk assessment in planning

- **> 2 weeks**
- Not ideal

Check on patients post operatively HOMECHECK
Acknowledge problem
No sit and WAIT !!!!!
You MUST reassure your patient but don't give them false expecta
Seek advice- Trigeminalnerve.org.uk- Medication and REFERRAL

Wait for resolution

- Lingual nerve injuries related to LINGUAL ACCESS third molar surgery (consider explore @ 12 weeks)
- LA
- Trauma
- Orthognathic



JOURNAL OF ORAL & MAXILLOFACIAL RESEARCH Shavit and Juodzbalys

Inferior Alveolar Nerve Injuries Following Implant Placement - Importance of Early Diagnosis and Treatment: a Systematic Review

Ilana Shavit[1], Gintaras Juodzbalys[1]

[1]Department of Maxillofacial Surgery, Lithuanian University of Health Sciences, Kaunas, Lithuania.

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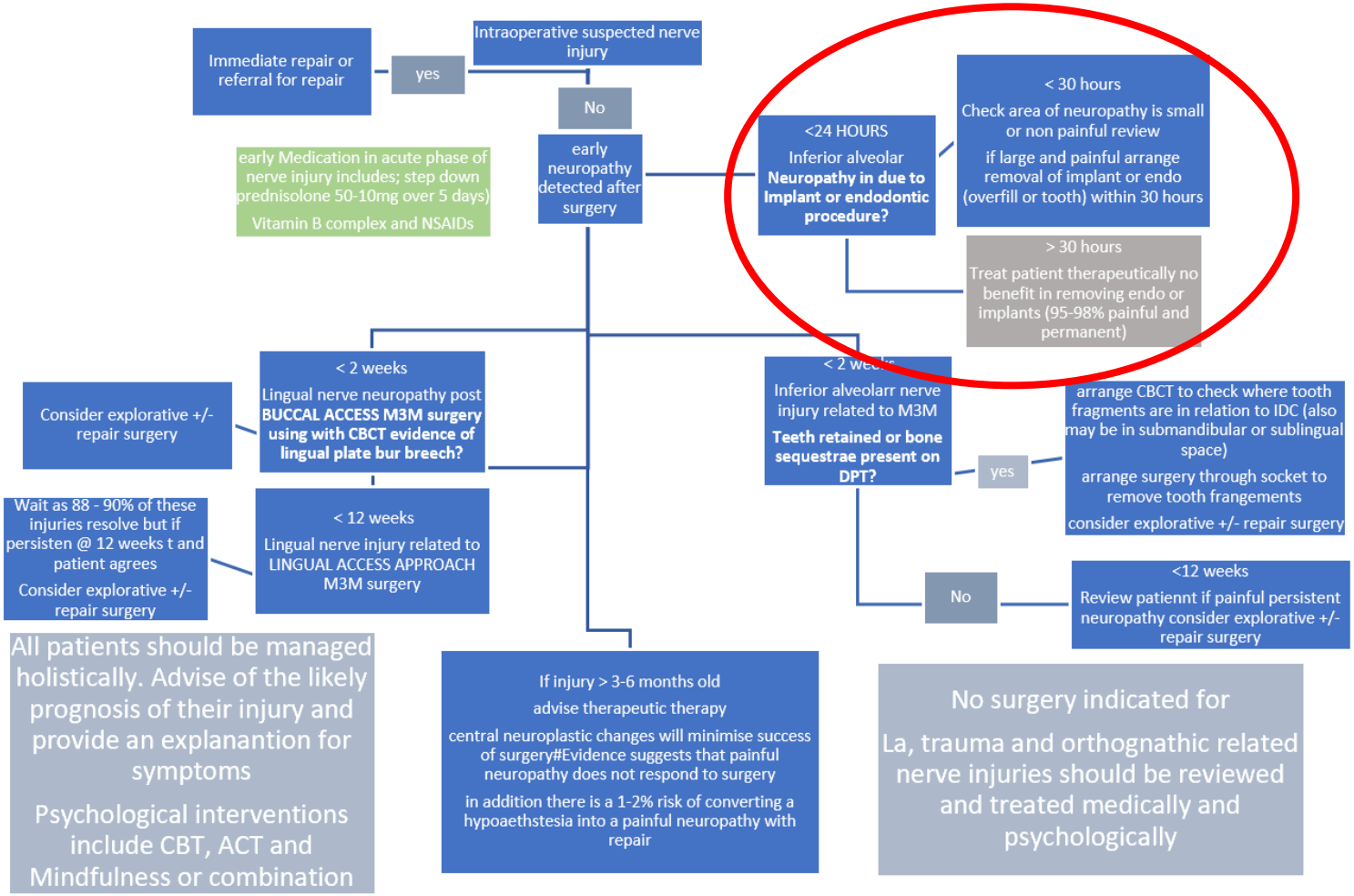
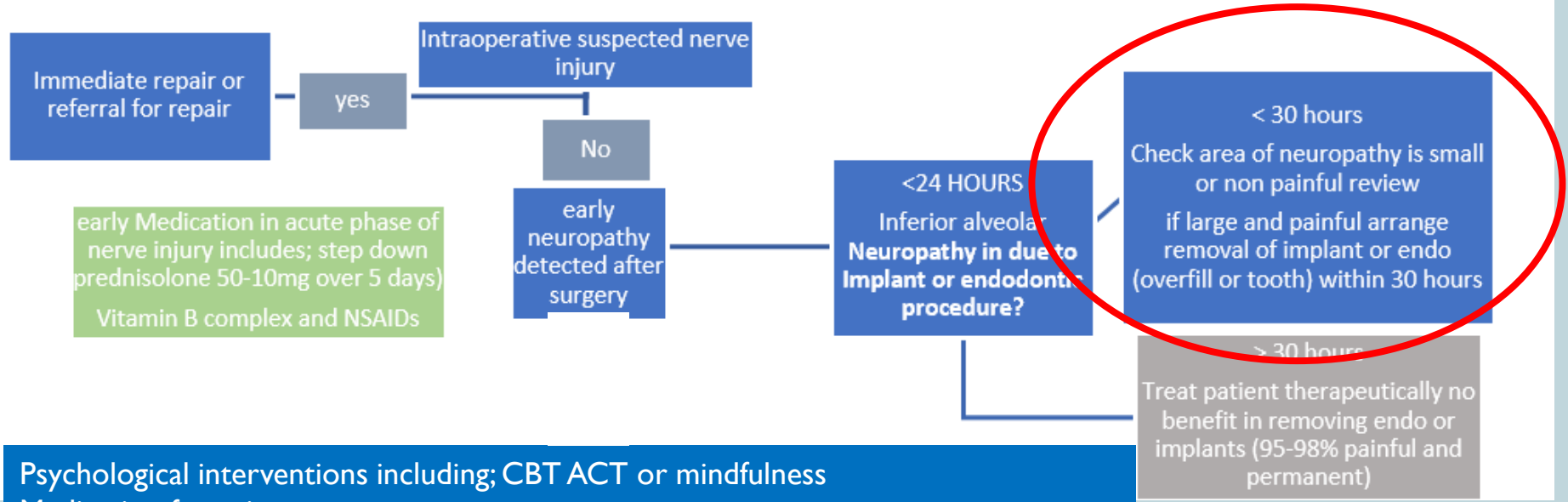
ABSTRACT

Objectives: The purpose of this article is to systematically review diagnostic procedures and risk factors associated with inferior alveolar nerve injury following implant placement, to identify the time interval between inferior alveolar nerve injury and its diagnosis after surgical dental implant placement and compare between outcomes of early and delayed diagnosis and treatment given based on case series recorded throughout a period of 10 years.
Material and Methods: We performed literature investigation through MEDLINE (PubMed) electronic database and manual search through dental journals to find articles concerning inferior alveolar nerve injury following implant placement. The search was restricted to English language articles published during the last 10 years, from December 2004 to March 2014.
Results: In total, we found 33 articles related to the topic, of which 27 were excluded due to incompatibility with established inclusion criteria. Six articles were eventually chosen to be suitable. The studies presented diagnostic methods of inferior alveolar nerve sensory deficit, and we carried out an assessment of the proportion of patients diagnosed within different time intervals from the time the injury occurred.
Conclusions: Various diagnostic methods have been developed throughout the years for dealing with 1 quite frequent


Immediate repair or referral for repair
yes
Intraoperative suspected nerve injury
No
early Medication in acute phase of nerve injury includes; step down prednisolone 50-10mg over 5 days)
Vitamin B complex and NSAIDs
early neuropathy detected after surgery
<24 HOURS
Inferior alveolar Neuropathy in due to Implant or endodontic procedure?
< 30 hours
Check area of neuropathy is small or non painful review
if large and painful arrange removal of implant or endo (overfill or tooth) within 30 hours
> 30 hours
Treat patient therapeutically no benefit in removing endo or implants (95-98% painful and permanent)
< 2 weeks
Lingual nerve neuropathy post BUCCAL ACCESS M3M surgery using with CBCT evidence of lingual plate bur breech?
Consider explorative +/- repair surgery
< 12 weeks
Lingual nerve injury related to LINGUAL ACCESS APPROACH M3M surgery
Wait as 88 - 90% of these injuries resolve but if persisten @ 12 weeks t and patient agrees
Consider explorative +/- repair surgery
< 2 wee
Inferior alveolarr nerve injury related to M3M
Teeth retained or bone sequestrae present on DPT?
yes
arrange CBCT to check where tooth fragments are in relation to IDC (also may be in submandibular or sublingual space)
arrange surgery through socket to remove tooth frangements
consider explorative +/- repair surgery
No
<12 weeks
Review patiennt if painful persistent neuropathy consider explorative +/- repair surgery
All patients should be managed holistically. Advise of the likely prognosis of their injury and provide an explanantion for symptoms
Psychological interventions include CBT, ACT and Mindfulness or combination
If injury > 3-6 months old
advise therapeutic therapy
central neuroplastic changes will minimise success of surgery#Evidence suggests that painful neuropathy does not respond to surgery
in addition there is a 1-2% risk of converting a hypoaethstesia into a painful neuropathy with repair
No surgery indicated for
La, trauma and orthognathic related nerve injuries should be reviewed and treated medically and psychologically

Management – implant related PTNs



Psychological interventions including; CBT ACT or mindfulness
Medication for pain
Neuralgic pain – Pregabalin Lyrica
Burning deep pain Nortriptyline
? Restoration or late removal of implant can exacerbate neuropathic pain and surgery doesn't work

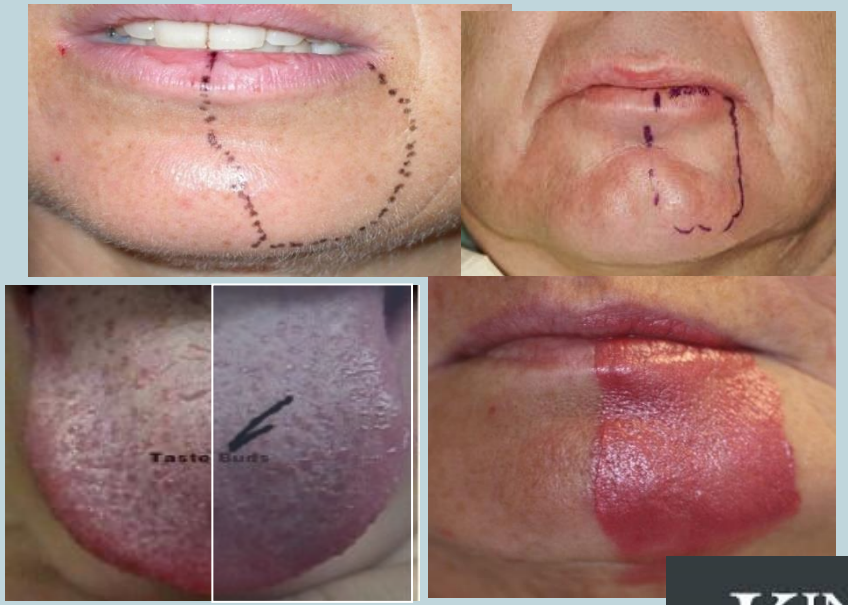
Management of Implant nerve injury

Confirm Nerve injury < 24 hours

HOMECHECK patie complains of pain and or numbness after LA worn off

Temporary or permanent?

- **Mechanism**
- **Duration**
- Identify the extent of injury
  - Size neuropathic area
  - Subjective function
  - Mechanosensory function
  - Disability
  - Pain / discomfort
    - Allodynia
    - Hyperalgesia
    - Spontaneous or elicited?



Patient's story and expectations?

Renton T, Thexton A, SJ Crean, Hankins M. Simplifying assessment of recovery of the lingual nerve from injury. BDJ 200
T, Thexton A, Mcgurk M. New method for the objective evaluation of injury to the lingual nerve after operation on third mola

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Post implant placement radiograph should be available
But if not Post-operative DPT as good as CBCT

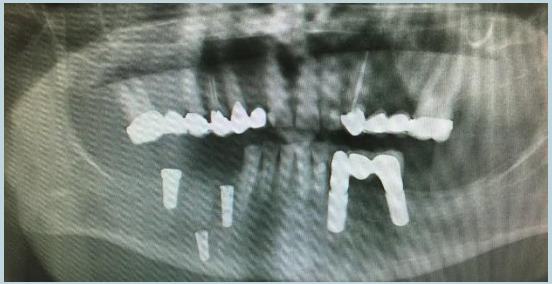
J Oral Implantol. 2020 Jun 1;46(3):206-213. doi: 10.1563/aaid-joi-D-19-00005.

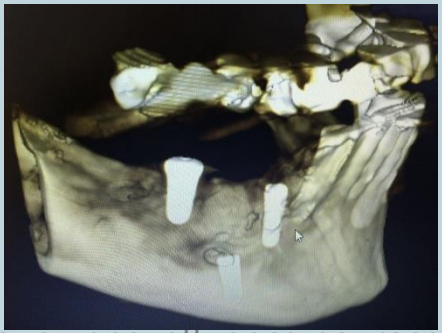
Diagnostic Potential of Panoramic Radiography and CBCT in Detecting Implant-Related Ex Vivo Injuries of the Inferior Alveolar Canal Border

Yigit Sirin[1], Senem Yildirimturk[1], Sinan Horasan[2], Koray Guven[1]

Abstract

The aim of this ex vivo study was to compare the diagnostic performances of panoramic radiography and cone beam computerized tomography (CBCT) in detecting implant-related injuries of the inferior alveolar canal. Monocortical bone windows were created in 60 fresh sheep hemimandibles, the inferior alveolar canals were revealed and 120 dental implants were inserted. Three types of injuries, described as pilot drill damage (PDRILL), collapsing of the superior border of the canal (COLL), penetration of the implant tip into the canal (PENET) and one control group, were simulated. Standard (PANO) and dentition mode panoramic (PANO-DENT) images as well as CBCT data presented as multiplanar reconstruction (MPR) and cross-sectional (CROSS) views were evaluated by 6 observers who had also expressed their level of confidence to their final diagnosis. Intra- and interobserver agreement scores were rated good. The area under the curve (AUC) values and the confidence scores for CROSS and multiplanar reformation (MPR) views were both significantly higher than those of PANO and PANO-DENT ($P < .05$ for each) in PDRILL group. In COLL group, observers showed less confidence to PANO and PANO-DENT compared to CROSS and MPR techniques ($P < .05$ for each). No other significant differences were found. Within the limits of this experimental study, it can be suggested that the standard and dentition modes of panoramic radiography can be as effective as CBCT in the detection of penetrating and collapsing injuries, but multiplanar and cross-sectional views of the CBCT are more accurate than panoramic radiography in the detection of pilot drill injuries in sheep mandible.

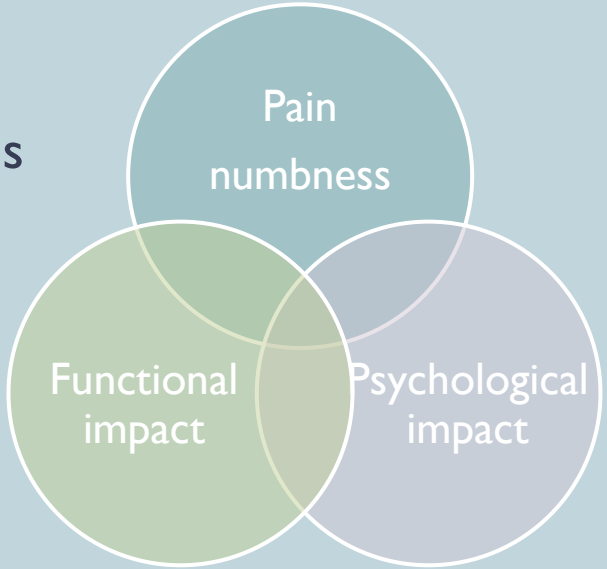




Suhaym O, Miloro M. Does early repair of trigeminal nerve injuries influence neurosensory recovery? A systematic review and meta-analysis. Int J Oral Maxillofac Surg. 2020 Nov 6:S0901-5027(20)30380-5. doi: 10.1016/j.ijom.2020.10.002. Epub ahead of print. PMID: 33168370.

Patient issues?

- Pain
  - Diagnosis and prognosis and info
  - Early and late Neuropathic pain meds
    - Systemic
    - Topical
    - Local
  - Psychological interventions
- Functional Impact
  - Psychological interventions
- Psychological impact
  - Psychological interventions



- **Continued support and review Trigeminalnerve.org.uk**

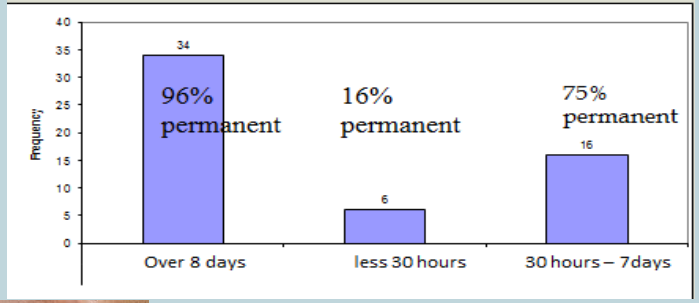
Implant PTN confirmed < 30 hours

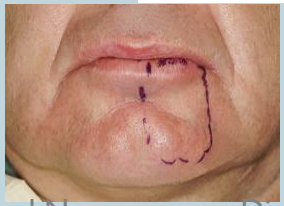
Early surgery and medication after nerve injury can improve resolution?

- Acute management < 30 hours (delayed onset neuropathy)
  - (LA IDB lasts 3 hours and 25minutes)
  - Check on Patient after 6 hours (Home check)
  - IAN NEUROPATHY? (extreme pain/ mixed symptoms large neuropathic area)
    - Yes
  - Consult patient, check for area of neuropathy and signs of nerve injury
    - Confirmed
      - **Remove Endo / tooth < 30 hours with neuropathy**
        - **+ High dose oral NSAIDs (600-800mgs Ibuprofen PO QDS)**
        - **Prednisolone 5 day step down does 50-40-30-20-10mg PO**
        - **Vitamin B Complex?**
        - **(check medical history!)**
        - Review

Only use plain films

Removing implant or endo filled tooth < 30 hours does Improve NI resolution





Bhavsar I[1], Khalaf M, Ferrin J, Al-Sabbagh M. Resolution of Implant-Induced Neurosensory Disturbance: A Procedural Failure. Implant Dent. 2015 Dec;24(6):735-41. Khawaja N, Renton T. Case studies on **implant removal** influencing the resolution of inferior alveolar **nerve injury**. Br Dent J. 2009 Apr 11;206(7):365-70.

Urgent Mx Implant related PTN is best

- Results were triangulated to evaluate their level of agreement.
- **The extraction of dental implants less than 36 hours after injury to the mandibular nerve results in the most successful resolution of neurosensory dysfunction.**
- Various microsurgical techniques have shown less success in obtaining neurosensory recovery than extraction of the implant.
- However, microsurgery is worthwhile, as it improves neurosensory dysfunction and reduces dysaesthesia in the majority of patients.
- Direct suturing and external decompression can result in good neurosensory recovery, and nerve grafts are also successful whenever tension-free direct suturing is not possible. Low-level laser therapy has been shown to achieve some neurosensory improvement

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BRITISH Journal of Oral and Maxillofacial Surgery
www.bjoms.com

Success of surgical interventions for direct dental implant-related injuries to the mandibular nerve: a review

L.M. Fee*

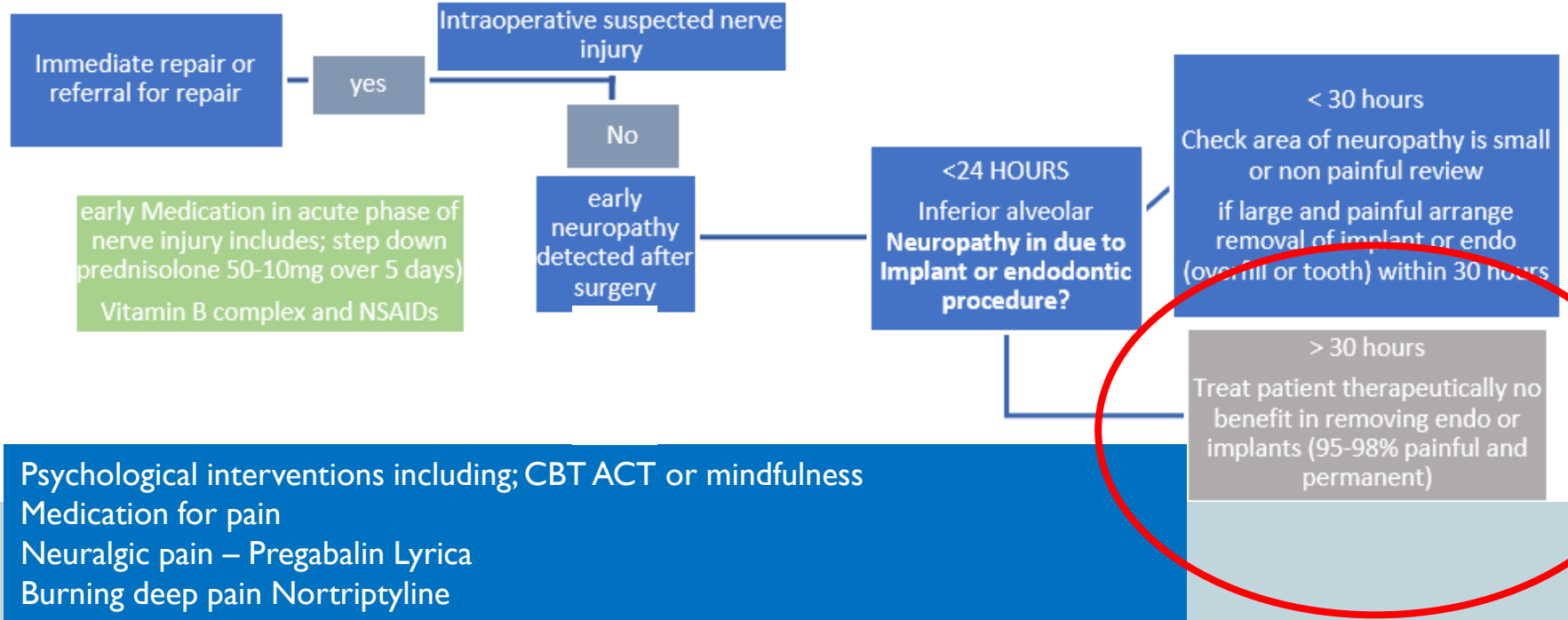
BA BDS, MSc in Dental Implants(Bristol University), Diploma in Pimary Care Oral Surgery, RCS(Eng), Diploma in Conscious Sedation (Newcastle University), Cert in Clinical Education (Edinburgh University)

Available online 12 May 2020

Abstract

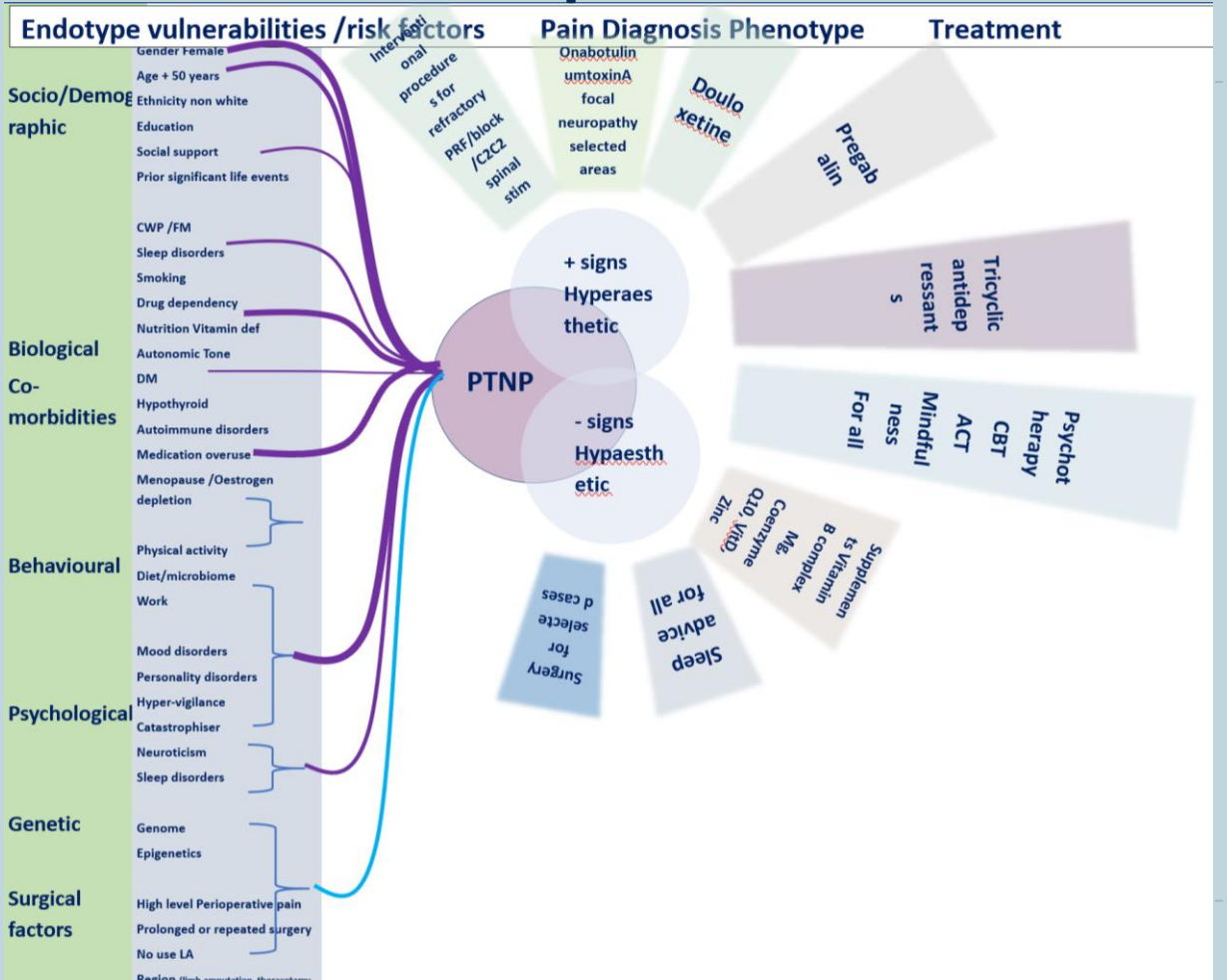
To the best of our knowledge there are no guidelines regarding the surgical management of dental implant-related injuries to the mandibular nerve. This review aims to investigate the success of different surgical interventions. Neurosensory injury to the mandibular branch of the trigeminal nerve can occur during administration of local anaesthetic, elevation of the flap, preparation for osteotomy, and placement of the implant. Surgical interventions include extraction of the implant, external decompression, internal neurolysis, excision of a neuroma, neurorrhaphy, nerve grafting, and low-level laser therapy. The following electronic databases were searched: MEDLINE, EMBASE, and the Cochrane Library. Primary outcome measures included patient-reported outcomes such as pain and altered sensation. A total of 185 publications were obtained, of which 21 were included in the qualitative synthesis (2 randomised controlled trials (RCT), 9 controlled cohort studies, and 10 case reports/series). They were all screened in consideration of the exclusion criteria and appraised using the Cochrane risk of bias tool, the Newcastle Ottawa scale, and the modified Newcastle Ottawa scale. Results were triangulated to evaluate their level of agreement. The extraction of dental implants less than 36 hours after injury to the mandibular nerve results in the most successful resolution of neurosensory dysfunction. Various microsurgical techniques have shown less success in obtaining neurosensory recovery than extraction of the implant. However, microsurgery is worthwhile, as it improves neurosensory dysfunction and reduces dysaesthesia in the majority of patients. Direct suturing and external decompression can result in good neurosensory recovery, and nerve grafts are also successful whenever tension-free direct suturing is not possible. Low-level laser therapy has been shown to achieve some neurosensory improvement.

Late Management – implant related PTNs



Psychological interventions including; CBT ACT or mindfulness
Medication for pain
Neuralgic pain – Pregabalin Lyrica
Burning deep pain Nortriptyline
? Restoration or late removal of implant can exacerbate neuropathic pain and surgery doesn't work

PTNP treatment options



- **Confirmation** of diagnosis, explanation and provision of realistic prognosis
- **Counselling**
- **Medication**
- **Surgery**
- Continued support/ monitor resolution

Mx Implant PTNPain

- Mainly pain related

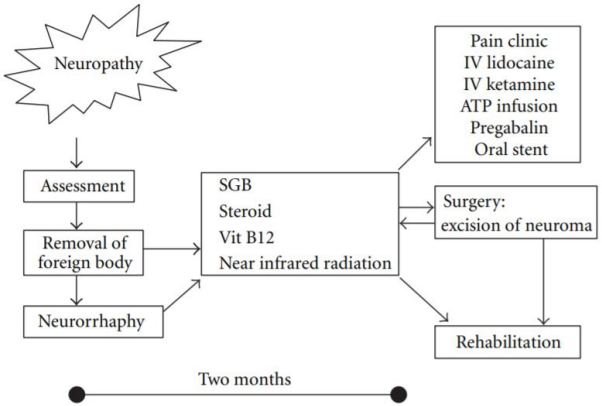


FIGURE 15: Therapies for neuropathy [18].

Review Article

Pain Management for Nerve Injury following Dental Implant Surgery at Tokyo Dental College Hospital

Ken-ichi Fukuda,[1] Tatsuya Ichinohe,[2] and Yuzuru Kaneko[2]

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[2] *Department of Dental Anesthesiology, Tokyo Dental College, 2-9-18, Misaki-cho, Chiyoda-ku, Tokyo 101-0061, Japan*

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Academic Editor: Toshiya Koitabashi

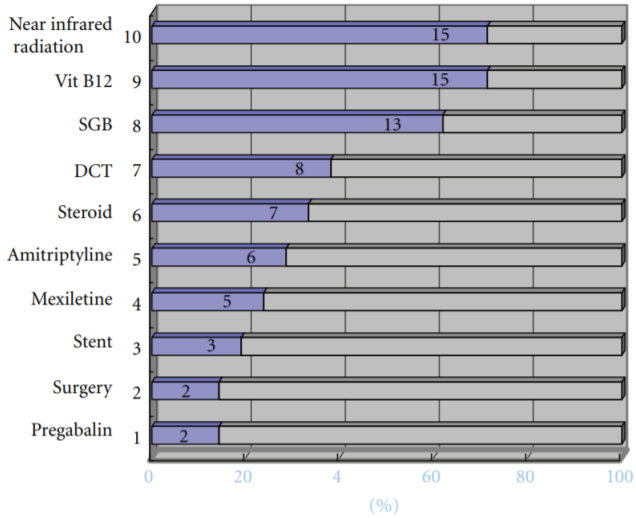

FIGURE 12: Methods of treatments after nerve injury used at Tokyo Dental College Suidoubashi Hospital (2008.4-2009.3) SGB: stellate ganglion block; DCT: drug-challenge test [18].

- **Management of patients with neuropathic pain**
- **Multidisciplinary Care**
- **a strong GRADE recommendation for use and proposal as first line for TCAs, SNRIs, pregabalin, gabapentin and gabapentin ER/enacarbil in neuropathic pain :**
  - NNTs
  - 3 ·6 (95 % CI 3 ·0–4 ·4) for **tricyclic antidepressants** (TCAs),
  - 6 ·4 (95 % CI 5 ·2–8 ·4) for **serotonin- noradrenaline reuptake inbibitor (SNRI**) antidepressants duloxetine and venlafaxine,
  - 7 ·7 (95 % CI 6 ·5–9 ·4) for **pregabalin**
  - 6 ·3 (95 % CI 5 ·0–8 ·3) for **gabapentin**.
  - ?? **capsaicin high concentration patches**,
- **a weak recommendation for use and proposal as second line for lidocaine patches, capsaicin patches and tramadol,**
  - opioids
  - Final quality of evidence was lower for lidocaine patches and BTX-A. Tolerability/safety and values/preferences were high for lidocaine patches and lower for opioids and TCAs.

Finnerup et al. Lancet Neurol. Author manuscript; available in PMC 201

HHS Public Access
Author manuscript
Lancet Neurol. Author manuscript; available in PMC 2016 February 01.

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Pharmacotherapy for neuropathic pain in adults: systematic review, meta-analysis and updated NeuPSIG recommendations

Nanna B Finnerup, MD[*,a], Nadine Attal, MD[*,b,c,1], Simon Haroutounian, PhD[d], Ewan McNicol, MS[e], Ralf Baron, MD[f], Robert H Dworkin, PhD[g], Ian Gilron, MD[h], Maija Haanpaa, MD[i], Per Hansson, MD[j], Troels S Jensen, MD[a,k], Peter R Kamerman, PhD[l], Karen Lund, MD[a], Andrew Moore, DSc[m], Srinivasa N Raja, MD[n], Andrew SC Rice, MD[o], Michael Rowbotham, MD[p], Emily Sena, PhD[q], Philip Siddall, MD[r], Blair H Smith, MD[s], and Mark Wallace, MD[t]

[1]Corresponding author: Nadine ATTAL. INSERM U 987 and Centre d'Evaluation et de Traitement de La Douleur, Hospital

Managing Neuropathic Pain

Robert Carter Wellford Jones III, MD, PhD[a], Erin Lawson, MD[a,b], Miroslav Backonja, MD[c,*]

KEYWORDS

- Neuropathic pain • Neuralgia • Peripheral neuropathy • Radiculopathy
- Anticonvulsants • Interventional treatments • Physical therapy
- Cognitive behavioral therapy

KEY POINTS

- Neuropathic pain (NP) arises from injuries or diseases affecting the somatosensory component of the nervous system at any level of the peripheral nervous system or central nervous system (CNS).
- Regardless of location of injury, NP is diagnosed based on common neurologic signs and symptoms that are revealed by history taking and on physical examination.
- NP is best treated with a combination of multiple therapeutic approaches, which starts with patient education, and the treatments include conservative, complementary, medical, interventional, and surgical treatment modalities.
- Goals of treatment are the same as in pain management in general, and they include improvement in pain control and in coping skills as well as restoration of functional status. Early identification of realistic treatment expectations is the key to building a successful relationship with a patient suffering from NP.
- In most instances when treating chronic NP, the approach to pain management begins with conservative therapies and advances to more interventional ones only when earlier modalities do not meet goals of pain relief and improved function, because risks increase with the invasiveness of the therapies. Most patients with NP benefit most from an individualized, multimodal approach that emphasizes both pain and function.

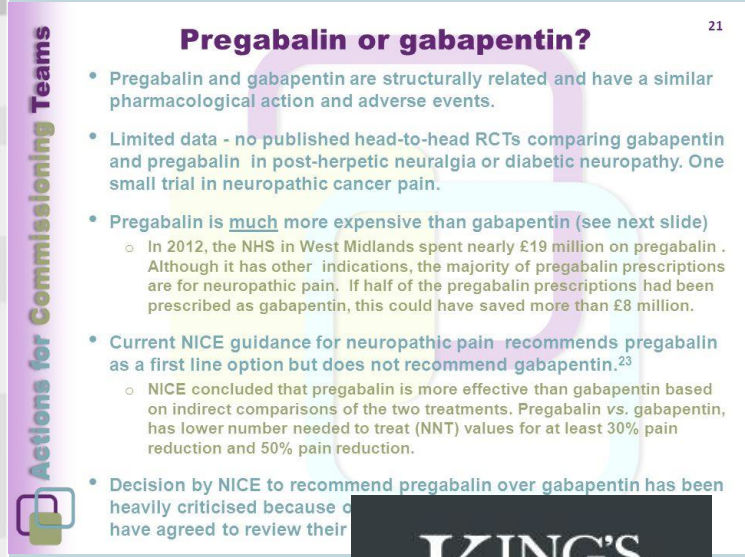
.ility, Grunenthal, Johnson and .stellas, Grunenthal and Astra Zeneca. aistry of Education and Research Research Foundation (DFG). He has .Medtronic, Eisai, Lilly, Boehringer fizer, Genzyme, Grünenthal, , Novartis, Bristol-Myers Squibb, .Drug Administration and US thods from Acorda, Adynxx, ioness, Bristol-Myers Squibb, , Depuy, Eli Lilly, Epicept, Flexion, Holdings, Nektar, Neura, NeurogesX, esis, Relmada, Sanofi-Aventis, Salix, ceived speaker's honorarium from Astellas. MH has received honoraria from Pfizer, Allergan, Astellas for om Pfizer, Grünenthal, Astellas, Orion oard for Reckitt Benckizer, and . EM reports grants from Richard ker's honorarium from Pfizer, a Grünenthal. SNR has served on the as share options in Spinifex ees from Spinifex Pharmaceuticals, as received funding for research R's laboratory are: Wellcome Trust rch Trust, International Association Research Council Industrial, sity of Kiel (Neuropain). ASCR is a

ING'S College NDON

Go to drugs
Nortriptyline (TCA) (10-40mgs nocte)
Lyrica Pregabalin (25mgs nocte / BD)

Indication	Dosing regimen	Maximum dose
DPN pain	3 divided doses per day	300 mg/day within 1 week
PHN	2 or 3 divided doses per day	300 mg/day within 1 week. Maximum dose of 600 mg/day
Adjunctive therapy for adult patients with partial onset seizures	2 or 3 divided doses per day	Maximum dose of 600 mg/day
Fibromyalgia	2 divided doses per day	300 mg/day within 1 week Maximum dose of 450 mg/day
Neuropathic pain associated with spinal cord injury	2 divided doses per day	300 mg/day within 1 week. Maximum dose of 600 mg/day

	Gabapentin	Pregabalin
Chemistry	Analog of GABA	Substituted analog of gabapentin
Absorption	Saturable	Non-saturable
Oral bioavailability	60% – 300 mg 33% – 3600 mg 27% – 4800 mg	90%
Onset of action	≥ 9 days	1–3 days
Renal elimination (half-life)	70–80% (5–7 hours)	90–99% (5–7 hours)
Dose (normal renal function)	300 mg po TID; ↑ q week as tolerated to maximum 3600 mg/day	75 mg po BID; ↑ every 3–7 days as tolerated to maximum 600 mg/day
T_{max}	0.7–1.5 hours	
Half-life	4.6–6.8 hours	5–7 hours
Percent excreted uncharged in urine	98%	



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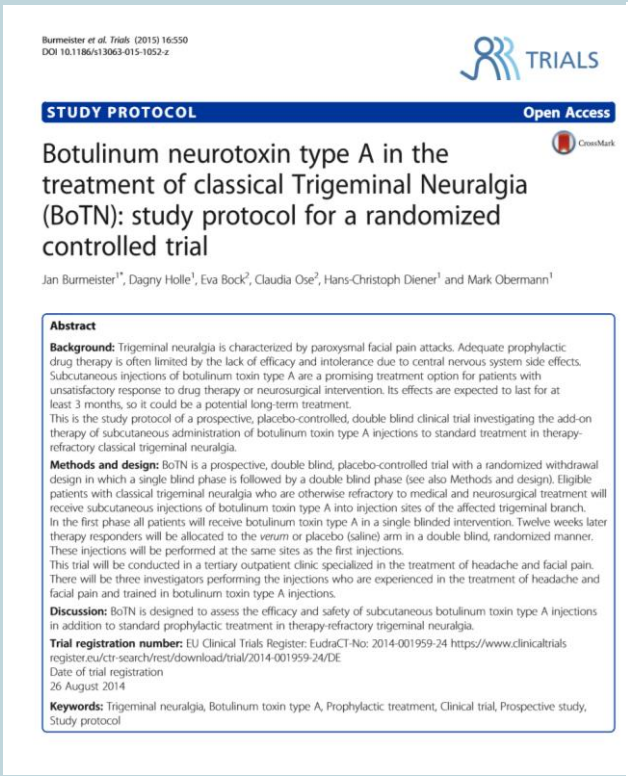
Botoxin A
Grade B for TN but low evidence for PTNP



Burmeister et al. Trials (2015) 16:550
DOI 10.1186/s13063-015-1052-z

TRIALS

STUDY PROTOCOL Open Access

Botulinum neurotoxin type A in the treatment of classical Trigeminal Neuralgia (BoTN): study protocol for a randomized controlled trial

Jan Burmeister[1*], Dagny Holle[1], Eva Bock[2], Claudia Ose[2], Hans-Christoph Diener[1] and Mark Obermann[1]

Abstract

Background: Trigeminal neuralgia is characterized by paroxysmal facial pain attacks. Adequate prophylactic drug therapy is often limited by the lack of efficacy and intolerance due to central nervous system side effects. Subcutaneous injections of botulinum toxin type A are a promising treatment option for patients with unsatisfactory response to drug therapy or neurosurgical intervention. Its effects are expected to last for at least 3 months, so it could be a potential long-term treatment.
This is the study protocol of a prospective, placebo-controlled, double blind clinical trial investigating the add-on therapy of subcutaneous administration of botulinum toxin type A injections to standard treatment in therapy-refractory classical trigeminal neuralgia.

Methods and design: BoTN is a prospective, double blind, placebo-controlled trial with a randomized withdrawal design in which a single blind phase is followed by a double blind phase (see also Methods and design). Eligible patients with classical trigeminal neuralgia who are otherwise refractory to medical and neurosurgical treatment will receive subcutaneous injections of botulinum toxin type A into injection sites of the affected trigeminal branch. In the first phase all patients will receive botulinum toxin type A in a single blinded intervention. Twelve weeks later therapy responders will be allocated to the *verum* or placebo (saline) arm in a double blind, randomized manner. These injections will be performed at the same sites as the first injections.
This trial will be conducted in a tertiary outpatient clinic specialized in the treatment of headache and facial pain. There will be three investigators performing the injections who are experienced in the treatment of headache and facial pain and trained in botulinum toxin type A injections.

Discussion: BoTN is designed to assess the efficacy and safety of subcutaneous botulinum toxin type A injections in addition to standard prophylactic treatment in therapy-refractory trigeminal neuralgia.

Trial registration number: EU Clinical Trials Register: EudraCT-No: 2014-001959-24 https://www.clinicaltrials register.eu/ctr-search/rest/download/trial/2014-001959-24/DE
Date of trial registration
26 August 2014

Keywords: Trigeminal neuralgia, Botulinum toxin type A, Prophylactic treatment, Clinical trial, Prospective study, Study protocol

Vol. 122 No. 1 July 2016

The efficacy of botulinum toxin for the treatment of trigeminal and postherpetic neuralgia: a systematic review with meta-analyses

Thomas Shackleton, DDS, MS,[a] Saravanan Ram, DDS, MS,[b] Misty Black, DDS, MS,[a] Jon Ryder, DDS, MS,[a] Glenn T. Clark, DDS, MS,[c] and Reyes Enciso, PhD[d]

Objective. To evaluate the efficacy of a botulinum toxin type A (BoTN-A) in treating trigeminal neuralgia (TN) and postherpetic neuralgia (PHN).
Study Design. Three databases were searched: Medline, Web of Science, and Cochrane Library. The search was restricted to English-language randomized, placebo-controlled trials. Three review authors evaluated the cases for risk of bias.
Results. Six studies were eligible for inclusion. Pooled results showed a difference in post-treatment pain intensity of −3.009 (95% confidence interval −4.566 to −1.453; $P < .001$) in favor of BoTN-A compared with placebo in managing TN or PHN. Of the six studies, five had unclear risk of bias, and one showed high risk.
Conclusions. Although the studies had unclear or high risk of bias, moderate evidence regarding the efficacy of BoTN-A in treating TN and PHN was found. BoTN-A might be an alternative treatment to those patients who are either unable to manage their pain medically or would like adjunct therapy. (Oral Surg Oral Med Oral Pathol Oral Radiol 2016;122:61-71)

Neuralgia is described as pain extending along the course of one or more nerves. Many varieties of neuralgia are distinguished according to the nerves affected, such as the trigeminal, brachial, occipital, and supraorbital nerves, or to the cause, such as postherpetic, anemic, diabetic, gouty, malarial, or syphilitic factors.[1] Pain from neuralgias is often debilitating to those who suffer from it. These patients often suffer for extended periods before any sort of beneficial therapy is suggested.[2] There are two major treatment strategies for neuralgias: pharmacotherapy and neurosurgery. Medical management is the mainstay treatment for most neuralgias, since it generally carries a lower risk compared with major surgical procedures and is suitable for medically compromised patients who are unfit for such surgery.[3] However, side effects from systemic medications, such as ataxia, dizziness, nausea, fatigue, rash, and somnolence, can be problematic and debilitating.

Botulinum toxin type A (BoTN-A) is a potent neurotoxin that blocks acetylcholine release from presynaptic nerve endings by interfering with the activity of SNARE (soluble *N*-ethylamide-sensitive-factor attachment protein receptors) proteins. BoTN-A has been reported to have analgesic effects independent of its action on muscle tone.[4] The most significant results have been observed in patients with neuropathic pain. Neuropathic pain caused by peripheral lesions has been the most widely studied. BoTN-A has shown its efficacy on pain and allodynia in various animal models of inflammatory neuropathic pain.[5] The objective of this review was to determine the efficacy of BoTN-A when used as a treatment in patients suffering from trigeminal neuralgia (TN) or postherpetic neuralgia (PHN).

MATERIALS AND METHODS

This systematic review followed the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement.[7]

Eligibility criteria

Studies were limited to randomized controlled trials (RCTs) on the efficacy of BoTN-A compared with

[a]Graduate, Master Science Program in Orofacial Pain and Oral Medicine, Herman Ostrow School of Dentistry of USC, Los Angeles, CA, USA.
[b]Associate Professor of Clinical Dentistry, Program Director, Oral Medicine, Herman Ostrow School of Dentistry of USC, Los Angeles, CA, USA.
[c]Professor of Dentistry, Program Director, Orofacial Pain, Herman Ostrow School of Dentistry of USC, Los Angeles, CA, USA.
[d]Associate Professor of Clinical Dentistry, Herman Ostrow School of Dentistry of USC, Los Angeles, CA, USA.
Received for publication Oct 10, 2015; returned for revision Jan 4, 2016; accepted for publication Mar 4, 2016.

2212-4403/$ - see front matter
http://dx.doi.org/10.1016/j.oooo.2016.03.003

Statement of Clinical Relevance

In this systematic review, the number of eligible studies was small, and the authors found unclear or high risk of bias in the included studies. However, moderate evidence regarding the efficacy of botulinum toxin A in treating trigeminal and postherpetic neuralgia was found; this evidence provides hope that this may be an alternative treatment for those patients who are either unable to manage their pain medically or would like an adjunct therapy.

Morra et al. The Journal of Headache and Pain (2016) 17:63
DOI 10.1186/s10194-016-0651-8

The Journal of Headache and Pain

REVIEW ARTICLE Open Access

Therapeutic efficacy and safety of *Botulinum Toxin A Therapy* in *Trigeminal Neuralgia*: a systematic review and meta-analysis of randomized controlled trials

Mostafa Ebraheem Morra[1†], Ahmed Elgebaly[1†], Ahmed Elmaraezy[1†], Adham M. Khalil[2†], Ahmed M. A. Altibi[3], Tran Le-Huy Vu[4], Mostafa Reda Mostafa[5], Nguyen Tien Huy[6,7*] and Kenji Hirayama[8*]

Abstract

Background: Several different interventions have been examined to alleviate pain and reduce frequency of trigeminal neuralgia (TN) paroxysms. However, some patients continue to have persistent or recurrent painful attacks. Using a systematic review and meta-analysis approach, we aimed to synthesize evidence from published randomized controlled trials (RCTs) regarding safety and efficacy of botulinum toxin type A (BTX-A) as a possible emerging choice of treatment for TN.

Methods: We conducted an electronic search in 10 databases/electronic search engines to access relevant publications. All articles in all languages reporting RCTs on the efficacy and safety of BTX-A in the treatment of TN were included for systematic review and meta-analysis.

Results: A total of four RCTs (n = 178) were identified for final meta-analysis. The overall effect favored BTX-A versus placebo in terms of proportion of responders (risk ratio RR = 2.87, 95 % confidence interval CI [1.76, 4.69], p <0.0001) with no significant detected heterogeneity (p = 0.31; I^2 = 4 %). Paroxysms frequency per day was significantly lower for BTX-A group (mean difference MD = -29.79, 95 % CI [-38.50,–21.08], p <0.00001) with no significant heterogeneity (p = 0.21; I^2 = 36 %).

Conclusion: Despite limited data, our results suggest that BTX-A may be an effective and safe treatment option for patients with TN. Further larger and well-designed RCTs are encouraged to translate these findings into better clinical outcome and better quality of life for TN patients.

Keywords: Botulinum, BTX-A, Trigeminal neuralgia, Clinical trials, Systematic review, Meta-analysis

Ngeow WC, Nair R Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010 Mar;109(3):e47-50.
Injection of botulinum toxin type A (BOTOX) into trigger zone of trigeminal neuralgia as a means to control pain.

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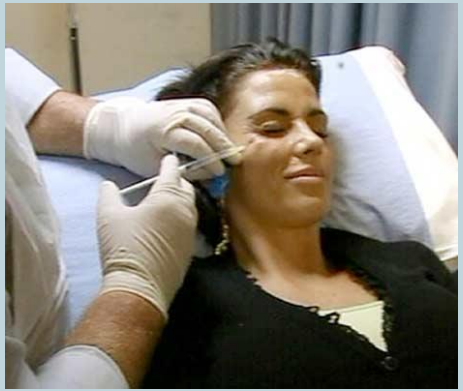
Management-
Pain medication - topical



- **Botox injections**
- Peripheral local anaesthetic block
- Check effect on local musculature
Facial nerve
- **Topical LA patches**

Ngeow WC, Nair R Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010 Mar;109(3):e47-50.
Injection of botulinum toxin type A (BOTOX) into trigger zone of trigeminal neuralgia as a means to control pain.

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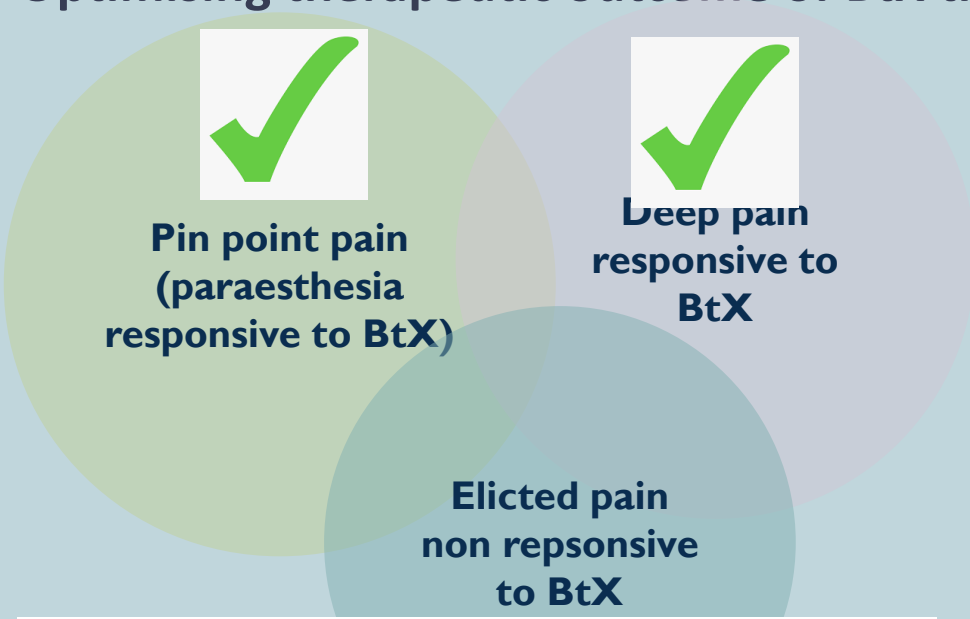
What Nepain phenotype predicts outcome of Btx?

Clustering Neuropathic pain presentation

Optimising therapeutic outcome of BtX therapy



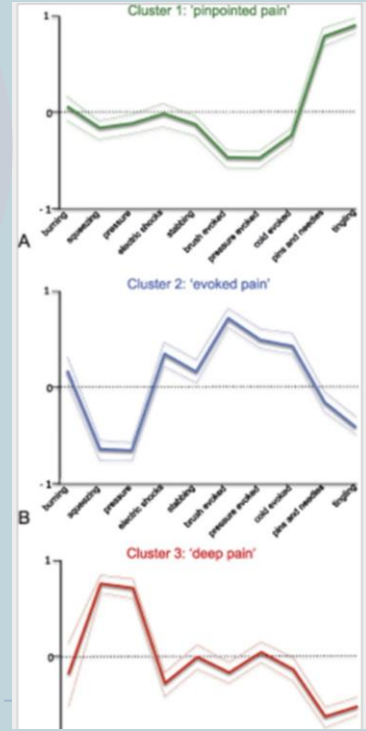


Figure 1.: Description of the 3 clusters of patients with distinct sensory profiles (ie, combinations of symptoms assessed with the 10 neuropathic pain descriptors included in the NPSI). Dashed lines represent confidence intervals (95% CI). (A) Cluster 1, "pinpointed pain," was characterized by above average scores for items relating to paresthesia/dysesthesia (ie, tingling and pins and needles) and below average scores for evoked pain (brush allodynia and pressure allodynia). (B) Cluster 2, "evoked pain," was characterized by above average pain provoked by brushing, provoked by cold or pressure and electric shocks and below average deep pain and paresthesia/dysesthesia. (C) Cluster 3, "deep pain," was characterized by above average pressure and squeezing pain and below average paresthesia/dysesthesia. NPSI, Neuropathic Pain Symptom Inventory.

› Pain. 2021 Apr 1;162(4):1038-1046. doi: 10.1097/j.pain.0000000000002130.

Stratification of patients based on the Neuropathic Pain Symptom Inventory: development and validation of a new algorithm

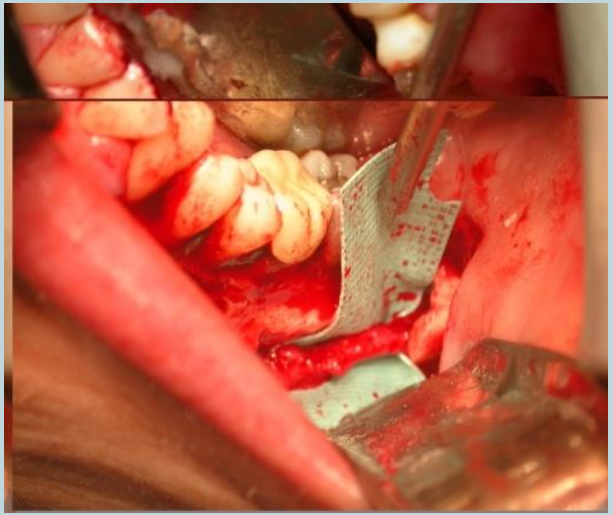
Didier Bouhassira [1], Samuel Branders [2], Nadine Attal [1], Ana Mercia Fernandes [3], Dominique Demolle [2], Julio Barbour [3], Daniel Ciampi de Andrade [3], Alvaro Pereira [2]

Late Management of Implant nerve injury Doesn't relieve neuropathic pain

If injury is > 36 hours days old or more

Manage therapeutically

- Surgery - removal of implant **doesn't work**
- Reassure patient
  - Apologise
  - Manage expectations
  - **Psychological support and interventions**
  - Reassure that lessons have been learnt
- Pain management **Medical management**
  - Topical Lidocaine patches, Capsaicin, Amitriptyline
  - Systemic Pregabalin / Tricyclic antidepressants



Surgery does NOT 'fix' injuries or resolve pain

Rodriquez-Lozano F, Sanchez-Perez A, Moya-Villaescusa MJ, Rodriguez-Lozano A, Saez-Yuguero MR. Neuropathic orofacial pain after dental implant placement: review of the literature and case report. OOOE 2010; **109:** e8-e12. Renton T, Yilmaz Z. Profiling of patients presenting with posttraumatic neuropathy of the trigeminal nerve. J Orofac Pain. 2011 Fall;25(4):333-44. Renton T, Dawood A, Shah A, Searson L, Yilmaz Z. Post-

ANESTHESIA/FACIAL PAIN

The Presence of Neuropathic Pain Predicts Postoperative Neuropathic Pain Following Trigeminal Nerve Repair

John R. Zuniga, DMD, MS, PhD, David M. Yates, DMD, MD,† and Ceib L. Phillips, MPH, PhD‡*

Purpose: The risk for the continuation or recurrence of neuropathic pain following trigeminal nerve repair has not been examined. The objective of this study was to determine which risk factors might be associated with the continuation or recurrence of neuropathic pain following trigeminal nerve microneurosurgery.

Patients and Methods: An ambispective study design was used to assess subjects who underwent trigeminal nerve repair of the inferior alveolar nerve and lingual nerve between 2000 and 2010. The primary outcome was the presence or absence of neuropathic pain at 3, 6, and 12 months after surgery. Explanatory variables, including age at surgery, gender, presence of neuropathic pain before surgery, site of nerve injury, etiology of nerve injury, classification of nerve injury, duration of nerve injury, and type of repair performed, were abstracted from patient charts. Fisher exact tests were used to compare the demographic and injury characteristics of patients who presented with pain before surgery and those who did not. The McNemar test was used to assess whether there was a significant change in neuropathic pain report from before to after surgery. The level of significance was set at .50.

Results: Of the 65 patients analyzed, two-thirds were women; the average age was 36 ± 16.1 years, and the median time between the injury and surgery was 6.4 months (interquartile range, 6.7 months). Lingual nerve injury type was the most frequent (62%). There was no statistically significant change in pain status

Conclusions: The presence of neuropathic pain prior to trigeminal microneurosurgery is the major risk factor for the continuation or recurrence of postoperative neuropathic pain. These findings suggest that trigeminal nerve surgery is not a risk factor for developing neuropathic pain in the absence of neuropathic pain before surgery.

J Oral Maxillofac Surg 72:2422-2427, 2014

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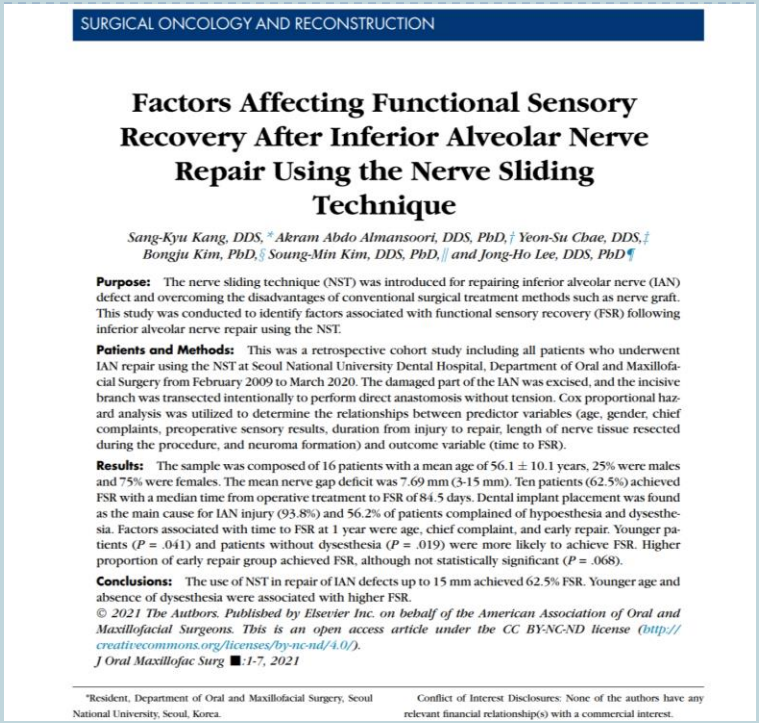
Surgery alone is not enough for neuropathic pain!



SURGICAL ONCOLOGY AND RECONSTRUCTION

Factors Affecting Functional Sensory Recovery After Inferior Alveolar Nerve Repair Using the Nerve Sliding Technique

Sang-Kyu Kang, DDS, Akram Abdo Almansoori, DDS, PhD,† Yeon-Su Chae, DDS,‡ Bongju Kim, PhD,§ Soung-Min Kim, DDS, PhD,‖ and Jong-Ho Lee, DDS, PhD¶*

Purpose: The nerve sliding technique (NST) was introduced for repairing inferior alveolar nerve (IAN) defect and overcoming the disadvantages of conventional surgical treatment methods such as nerve graft. This study was conducted to identify factors associated with functional sensory recovery (FSR) following inferior alveolar nerve repair using the NST.

Patients and Methods: This was a retrospective cohort study including all patients who underwent IAN repair using the NST at Seoul National University Dental Hospital, Department of Oral and Maxillofacial Surgery from February 2009 to March 2020. The damaged part of the IAN was excised, and the incisive branch was transected intentionally to perform direct anastomosis without tension. Cox proportional hazard analysis was utilized to determine the relationships between predictor variables (age, gender, chief complaints, preoperative sensory results, duration from injury to repair, length of nerve tissue resected during the procedure, and neuroma formation) and outcome variable (time to FSR).

Results: The sample was composed of 16 patients with a mean age of 56.1 ± 10.1 years, 25% were males and 75% were females. The mean nerve gap deficit was 7.69 mm (3-15 mm). Ten patients (62.5%) achieved FSR with a median time from operative treatment to FSR of 84.5 days. Dental implant placement was found as the main cause for IAN injury (93.8%) and 56.2% of patients complained of hypoesthesia and dysesthesia. Factors associated with time to FSR at 1 year were age, chief complaint, and early repair. Younger patients (P = .041) and patients without dysesthesia (P = .019) were more likely to achieve FSR. Higher proportion of early repair group achieved FSR, although not statistically significant (P = .068).

Conclusions: The use of NST in repair of IAN defects up to 15 mm achieved 62.5% FSR. Younger age and absence of dysesthesia were associated with higher FSR.

J Oral Maxillofac Surg ■:1-7, 2021

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Conflict of Interest Disclosures: None of the authors have any relevant financial relationship(s) with a commercial interest.

Journal of Oral Rehabilitation

Journal of Oral Rehabilitation 2016

Review

Evidence-based outcomes following inferior alveolar and lingual nerve injury and repair: a systematic review

E. KUSHNEREV & J. M. YATES *Department of Oral & Maxillofacial Surgery, University of Manchester, Manchester, UK*

Zuniga JR, Renton T. J Neurol Neuromed (2016) 1(7): 10-14
www.jneurology.com

J Neurology Neuromedicine
Journal of Neurology & Neuromedicine

Mini Review Open Access

Managing post-traumatic trigeminal neuropathic pain: is surgery enough?

John R. Zuniga[1], Tara F. Renton[2]

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ABSTRACT

In the absence of effective non-surgical methods to permanently resolve neuropathic pain involving the lip, chin, or tongue following inferior alveolar and/or lingual nerve injury, microsurgery of these nerves has been a recommended modality. In two ambispective clinical trials, we demonstrated that phenotypic differences exist between individuals with neuropathic pain and those without neuropathic pain of the trigeminal nerve. In those without neuropathic pain before microsurgery there was a 2% incidence of neuropathic pain after microsurgery whereas there was a 67% incidence of

The use of NST in repair of IAN defects up to 15 mm achieved 62.5% FSR. Younger age and absence of dysesthesia were associated with higher FSR

Suhaym O, Miloro M. Does early repair of trigeminal nerve injuries influence neurosensory recovery? A systematic review and meta-analysis. Int J Oral Maxillofac Surg. 2020 Nov 6:S0901-5027(20)30380-5. doi: 10.1016/j.ijom.2020.10.002. Epub ahead of print. PMID: 33168370

Laser therapy?

- No evidence
- Resolution likely due to spontaneous resolution?

ORAL SURGERY

Efficacy of photobiomodulation therapy on neurosensory recovery in patients with inferior alveolar nerve injury following oral surgical procedures: a systematic review

Neda Hakimiha, DDS, PhD/Seyed Hossein Bassir, DDS, DMSc/Georgios E. Romanos, DDS, PhD, Prof Dr med dent/ Ahmad Reza Shamshiri, MD, PhD/Neda Moslemi, DDS, DMSc

Objective: The present systematic review aimed to assess the efficacy of photobiomodulation (PBM) therapy on neurosensory recovery of patients with inferior alveolar nerve injury following third molar surgery or dental implant placement. **Method and materials:** An electronic search was carried out in Scopus, Embase, Medline, PubMed, Web of Science, Cochrane Library, and Google Scholar databases. Among 1,122 identified papers, seven articles (three RCTs, one observational study, and three case series) met the inclusion criteria. **Results:** Time lapse from nerve injury to the onset of PBM therapy varied widely from 2 days to 4 years. The number of patients in each study ranged between 4 and 74. In the majority of the studies, PBM was done using a diode laser at wavelengths in the range of 808 to 830 nm with power of 5 to 500 mW and radiation dose of 3 to 244 J/cm^2. Two out of three RCTs found significant neurosensory recovery in the patients who received PBM therapy compared to the controls. The observational study and all case series reported significant improvement in the neurosensory status following PBM therapy. The degree of neurosensory recovery was found to be greater in younger patients and those who received the treatment within 6 months following the injury. **Conclusions:** Due to the limited number of well-designed RCTs and small number of patients in each study, it is not possible to make a clear conclusion about the efficacy of PBM therapy on neurosensory recovery in patients with inferior alveolar nerve injury following third molar or implant procedures. Considering the possibility of spontaneous inferior alveolar nerve recovery during this period, the conclusion based on the studies with no control group should be interpreted with caution.
(Quintessence Int 2021;52:140–153; doi: 10.3290/j.qi.a45430)

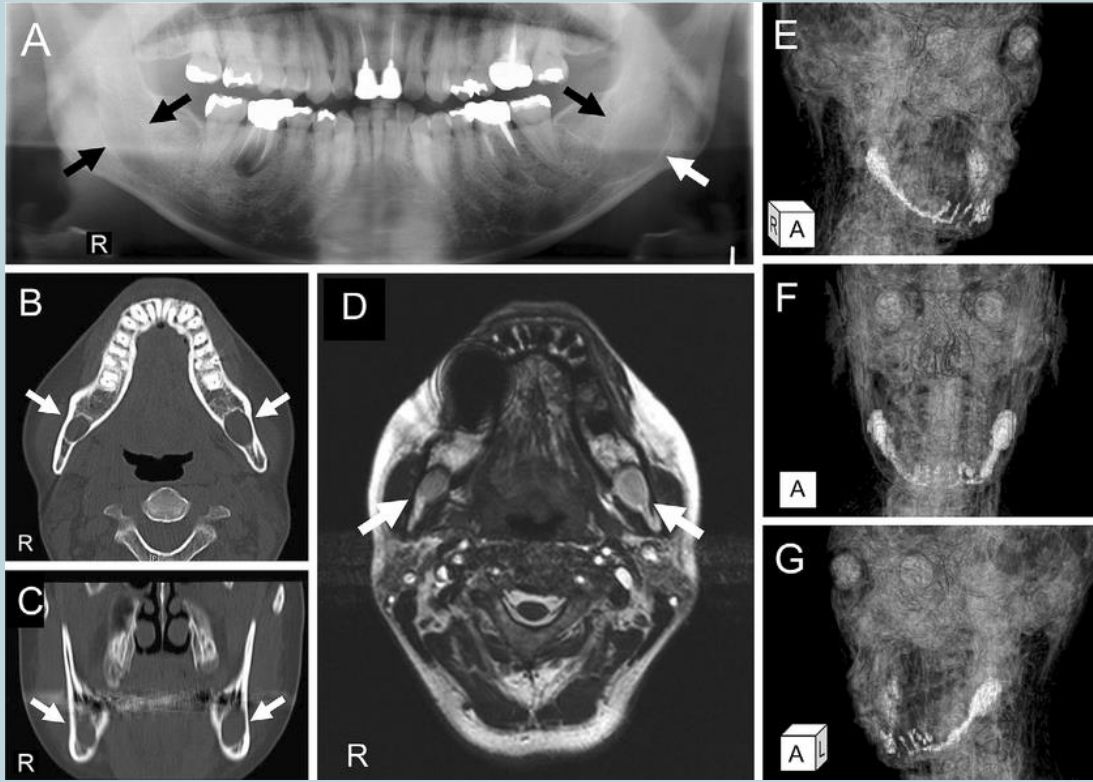
Key words: dental implants, inferior alveolar nerve, low-level light therapy, nerve regeneration, oral surgical procedures, photobiomodulation therapy

New developments

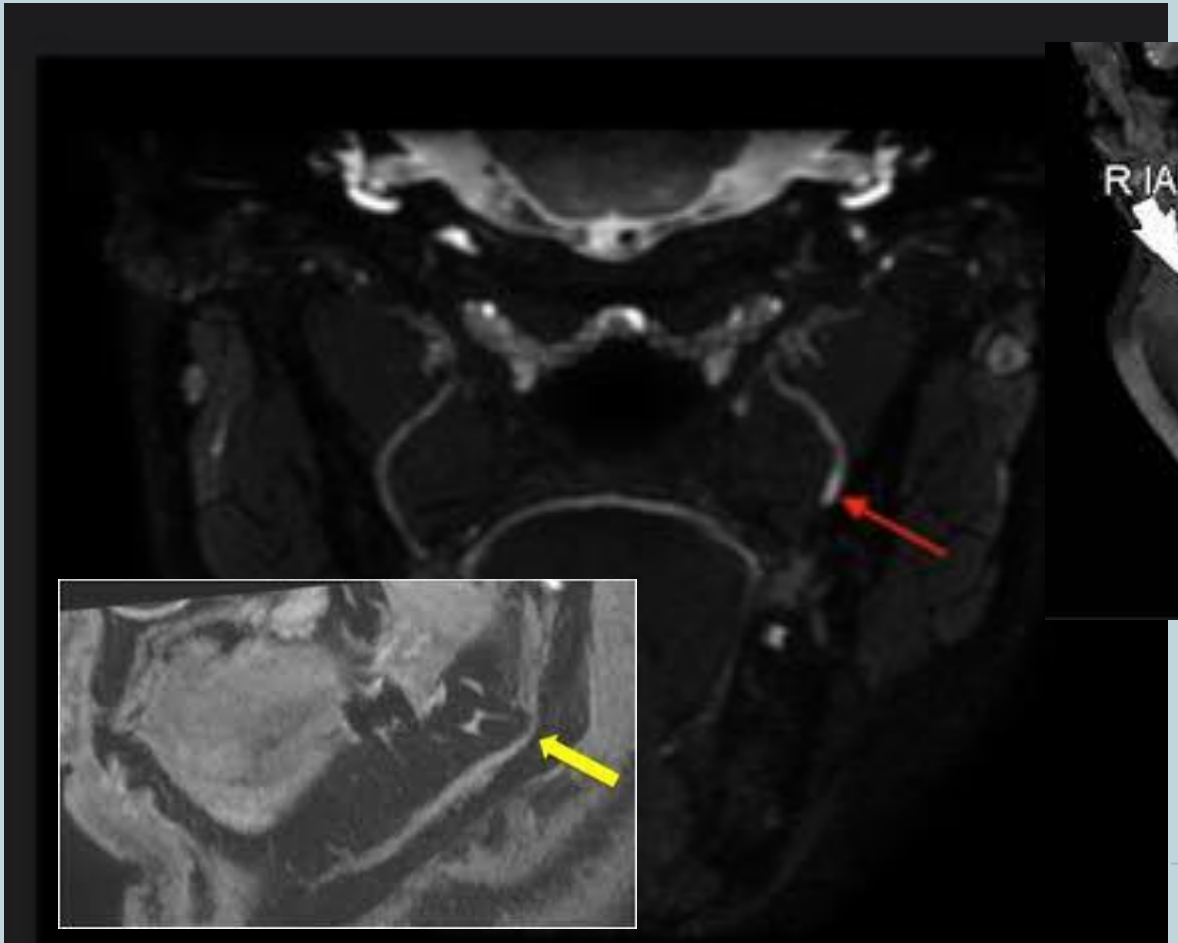
Zuniga JR, Mistry C, Tikhonov I, Dessouky R, **Chhabra** A Magnetic Resonance Neurography of Traumatic and Nontraumatic Peripheral Trigeminal Neuropathies. J Oral Maxillofac Surg. 2018 Apr;76(4):725-736. doi: 10.1016/j.joms.2017.11.007. Epub 2017 Nov 16.

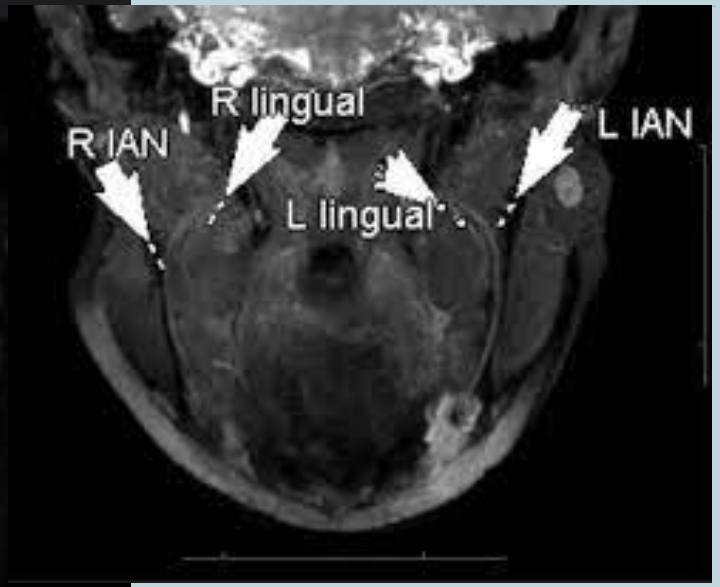
Dessouky R, Xi Y, **Zuniga J**, **Chhabra** A. Role of MR Neurography for the Diagnosis of Peripheral Trigeminal Nerve Injuries in Patients with Prior Molar Tooth Extraction. AJNR Am J Neuroradiol. 2018 Jan;39(1):162-169.

Cox B, Zuniga JR, Panchal N, Cheng J, **Chhabra** A. Magnetic resonance neurography in the management of peripheral trigeminal neuropathy: experience in a tertiary care centre. Eur Radiol. 2016 Oct;26(10):3392-400. doi: 10.1007/s00330-015-4182-5. Epub 2016 Jan 21

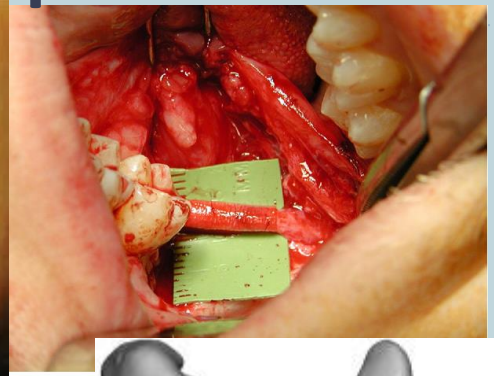


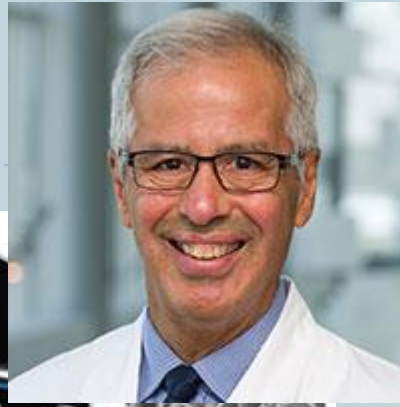
Lingual IAN PTN -MR Neurrography at KCL

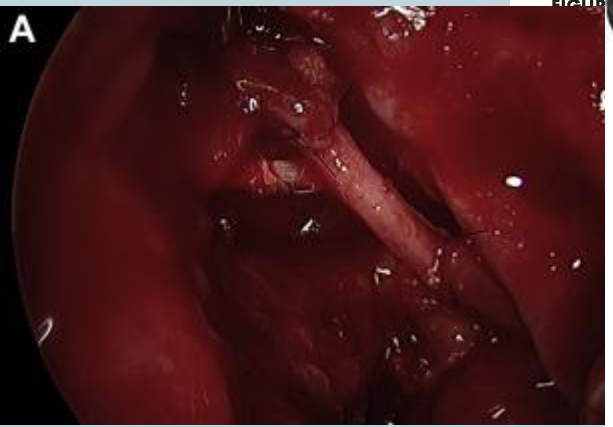


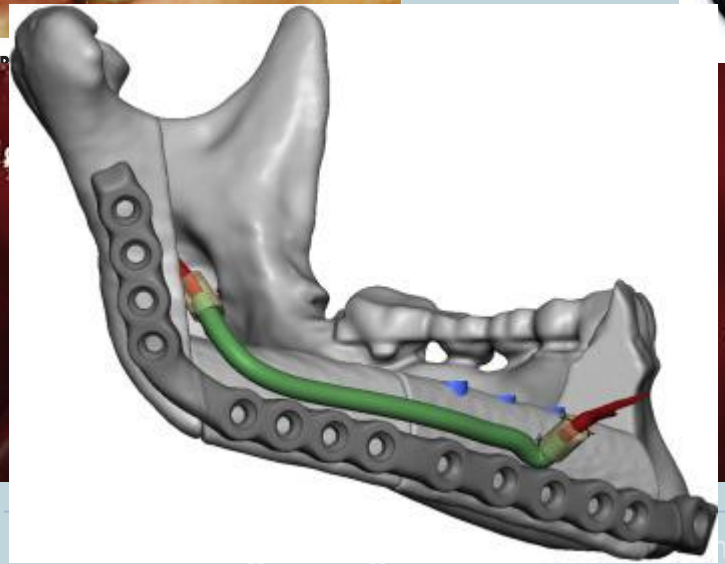


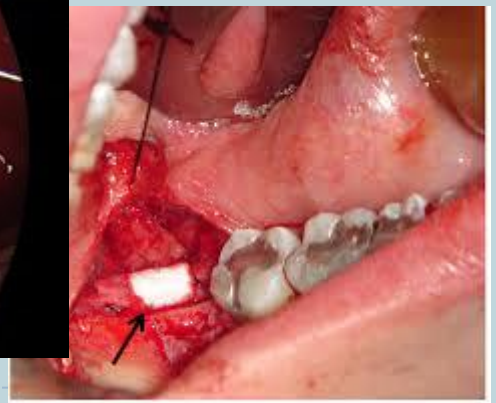
New developments





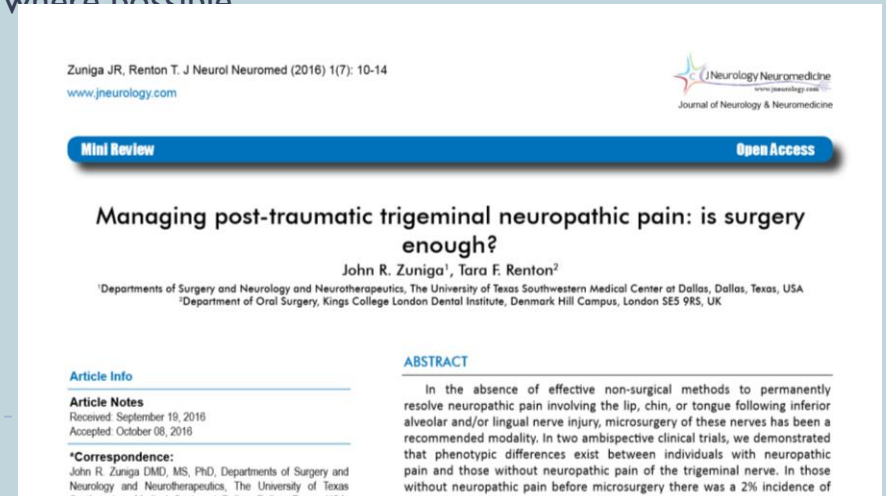






Summary

- **There is low evidence base for managing dental implant related PTN**
- we know that **38-95% are permanent** and **63-98% painful long term**
- **There is no 'magic bullet' to fix them**, we have to sit and wait and reassure the patient .
- **Early detection and management < 30hours** (medical, psychological and removal of implant f necessary)
- **You will be negligent** in causing the nerve injury but you should at least treat your patients humanely and prevent nerve injuries where possible



Zuniga JR, Renton T. J Neurol Neuromed (2016) 1(7): 10-14

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Mini Review | Open Access

Managing post-traumatic trigeminal neuropathic pain: is surgery enough?

John R. Zuniga[1], Tara F. Renton[2]

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ABSTRACT

In the absence of effective non-surgical methods to permanently resolve neuropathic pain involving the lip, chin, or tongue following inferior alveolar and/or lingual nerve injury, microsurgery of these nerves has been a recommended modality. In two ambispective clinical trials, we demonstrated that phenotypic differences exist between individuals with neuropathic pain and those without neuropathic pain of the trigeminal nerve. In those without neuropathic pain before microsurgery there was a 2% incidence of

Summary

- **There is low evidence base for managing dental implant related PTN**
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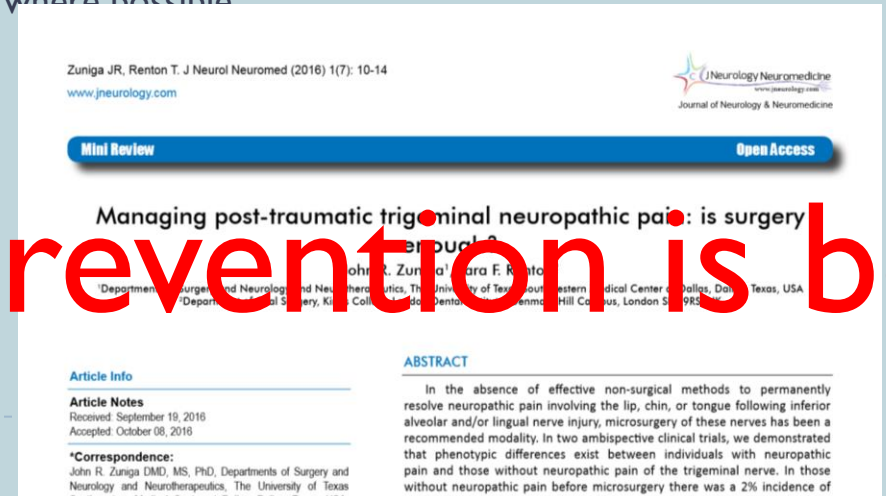

Prevention is best

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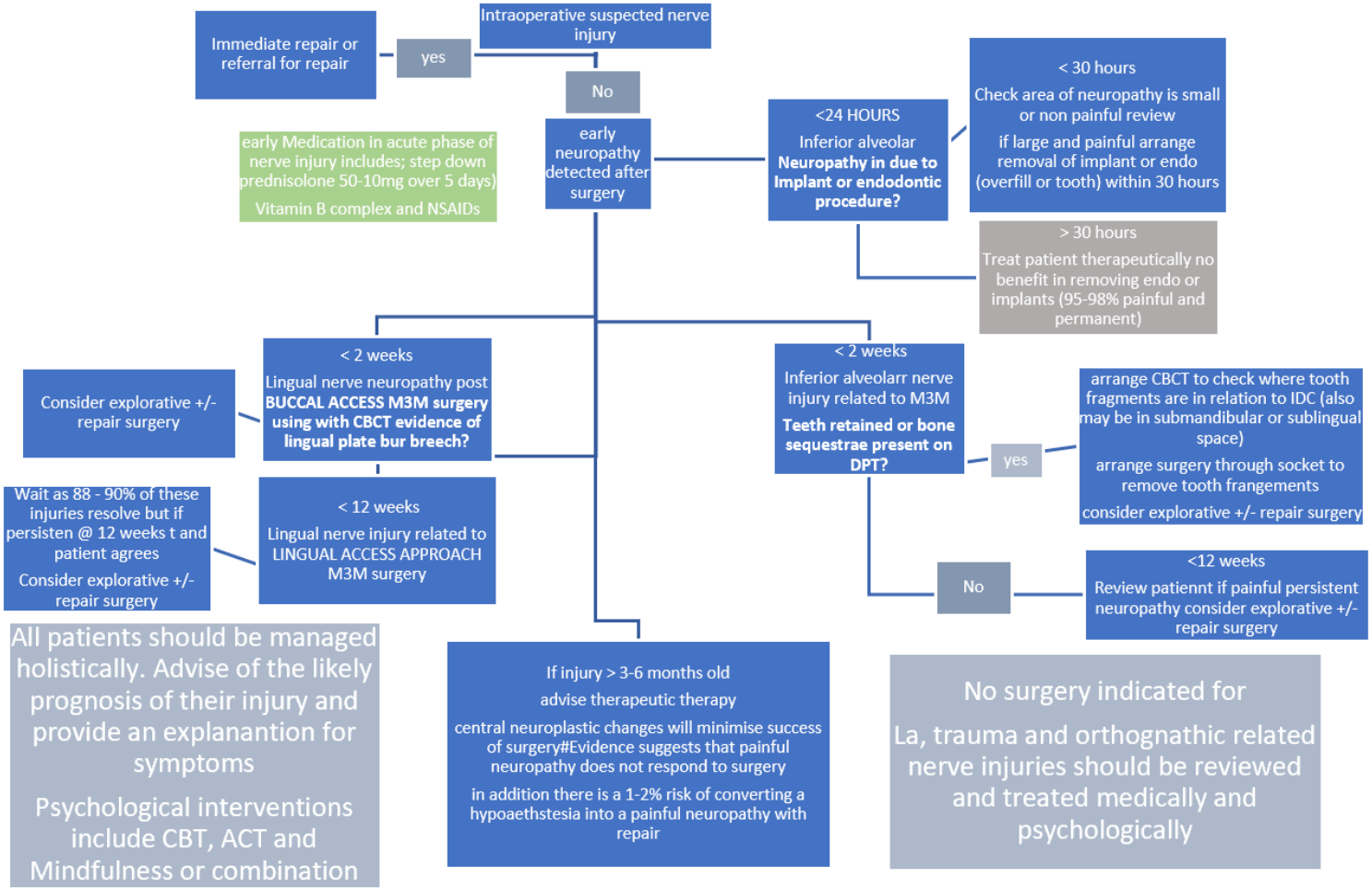
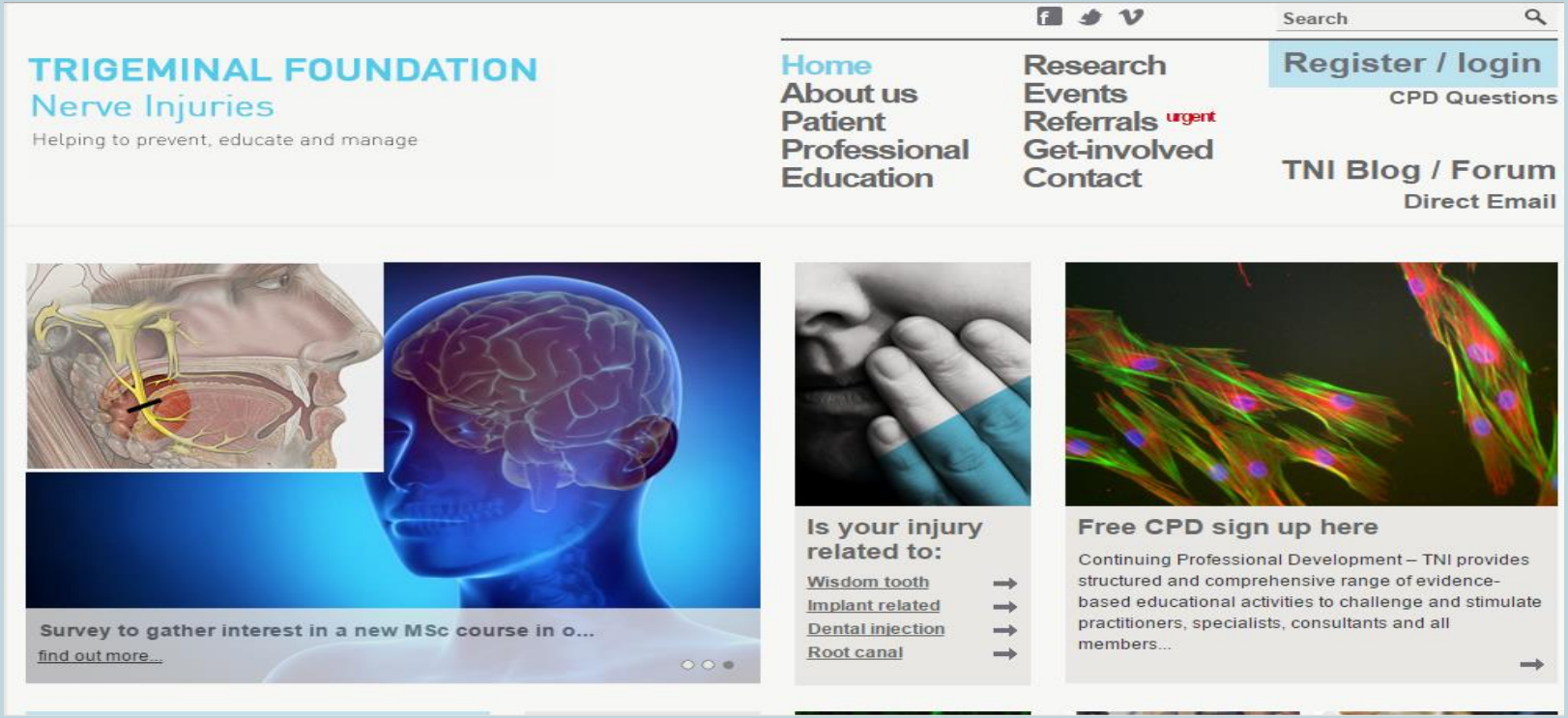

Immediate repair or referral for repair
yes
Intraoperative suspected nerve injury
No
early Medication in acute phase of nerve injury includes; step down prednisolone 50-10mg over 5 days)
Vitamin B complex and NSAIDs
early neuropathy detected after surgery
<24 HOURS
Inferior alveolar Neuropathy in due to Implant or endodontic procedure?
< 30 hours
Check area of neuropathy is small or non painful review
if large and painful arrange removal of implant or endo (overfill or tooth) within 30 hours
> 30 hours
Treat patient therapeutically no benefit in removing endo or implants (95-98% painful and permanent)
< 2 weeks
Lingual nerve neuropathy post BUCCAL ACCESS M3M surgery using with CBCT evidence of lingual plate bur breech?
Consider explorative +/- repair surgery
< 12 weeks
Lingual nerve injury related to LINGUAL ACCESS APPROACH M3M surgery
Wait as 88 - 90% of these injuries resolve but if persisten @ 12 weeks t and patient agrees
Consider explorative +/- repair surgery
< 2 weeks
Inferior alveolarr nerve injury related to M3M
Teeth retained or bone sequestrae present on DPT?
yes
arrange CBCT to check where tooth fragments are in relation to IDC (also may be in submandibular or sublingual space)
arrange surgery through socket to remove tooth frangements
consider explorative +/- repair surgery
No
<12 weeks
Review patiennt if painful persistent neuropathy consider explorative +/- repair surgery
All patients should be managed holistically. Advise of the likely prognosis of their injury and provide an explanantion for symptoms
Psychological interventions include CBT, ACT and Mindfulness or combination
If injury > 3-6 months old
advise therapeutic therapy
central neuroplastic changes will minimise success of surgery#Evidence suggests that painful neuropathy does not respond to surgery
in addition there is a 1-2% risk of converting a hypoaethstesia into a painful neuropathy with repair
No surgery indicated for
La, trauma and orthognathic related nerve injuries should be reviewed and treated medically and psychologically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What are the key diagnostic features of neuropathic pain?

- Onset related to surgical trauma
- A demonstrable neuropathic area n a viable anatomical distribution related to the prior surgery
- Positive signs including
  - elicited pain (mechanical and thermal allodynia),
  - ongoing spontaneous pain (burning, itching, pulling, stretching)
  - and altered sensation (dysesthesia or paraesthesia)
- Negative signs
  - numbness (anaesthesia) at rest,
- Confirmed neurosensory tests required for Definite diagnosis PTNP

What are the key patients risk factors for implant related post-traumatic neuropathic pain?

- *Age*
- *Gender*
- *Psychological co-morbidities (depression, anxiety, hypervigilance, catastrophising, narcissism, introversion*
- *High perioperative pain*
- *Medical co-morbidities, widespread pain, DM, haematinic, vitamin D, Magnesium deficiencies hypothyroidism, connective tissue disorders*
- *Mandibular mental nerve, premolar and molar region*

What are the key management strategies in managing patients with post-traumatic neuropathic pain related to implants?

- *ALWAYS review your patient by phone morning after surgery if confirmed area of neuropathy remove implant within 30 hours*
- *When checking the patient if they have a very small neuropathic area and no neuropathic pain then discuss with patient re implant removal*
- *Prescribe NSAIDs (if no medical contraindications) and vitamin B complex and you can ask GMP to prescribe step down Prednisolone over 5 days(50-10mg) (if no medical contraindications)*
- *If troublesome elicited neuralgic neuropathic pain consider GMP prescription of pregabalin and topical lidocaine patches (Versatis)*
- *If troublesome burning ongoing pain consider GMP prescription of Nortriptyline (10-40mg nocte)*
- *Long term Refer for clinical psychology support and referral to orofacial pain team*
- *Use resources on Trigeminalnerve.org.uk*

Prevention implant PTN using Lateralisation?

- After implant placement in the anterior mandible, the incidence of transient altered lip sensations was noted by severa investigators:
- 8.5% (N = 94),57 11% (N = 110),58 and 24 (N = 75) of patients.59 Walton59 reported that 1% of the patients manifested symptoms 1 year after therapy,
- Bartling et al.57 noted no permanent alterations of sensation 4 months posttherapy (N = 94).
- In another study, 7% (N = 110) of the patients reported sensory disturbance 16 months after treatment that was not present before therapy.58

Br J Oral Maxillofac Surg. 2019 Feb;57(2):169-173. doi: 10.1016/j.bjoms.2019.01.006. Epub 2019 Jan 31.

Neurosensory issues after lateralisation of the inferior alveolar nerve and simultaneous placement of osseointegrated implants

J M Castellano-Navarro[1], J J Castellano-Reyes[1], M Hirdina-Castilla[1], A Suárez-Soto[1], S Bocanegra-Pérez[1], M Vicente-Barrero[1]

Abstract

Our aim was to evaluate neurosensory symptoms after lateralisation of the inferior alveolar nerve (IAN). We studied a retrospective case series with one-year follow up that included 139 procedures in 123 patients. After the IA had been located it was deflected from the mandibular body and the implant placed. Sensitivity was mapped 24hours, one month, six months, and one year after the intervention by gently pressing the skin and lips with the t of a probe. A total of 337 implants were placed in 123 patients aged between 44 and 68 years. There were 33 men and 90 women and they all recovered. The IAN was mobilised by one of two procedures, one that involves th nerve directly (transposition) and one that does not (lateralisation). During lateralisation the nerve is deflected laterally through a mandibular osteotomy, while the mental nerve and mental foramen are not manipulated. The resulting hypoaesthetic area was drawn on a graph to assess its extension. Although different techniques are available for placing implants in atrophic jaws, mobilisation of the IAN is indicated in certain cases in which other techniques are not feasible or have a high risk of complications.

Keywords: bone implant interactions; implant; lateralization; soft tissue management; surgical procedure; wound healing.

- No neurosensory deficits after lateralisation for implant placement in 123 cases!!!!

Are there other operative strategies? NO

diagnostics

MDPI

Article

A CBCT Based Three-Dimensional Assessment of Mandibular Posterior Region for Evaluating the Possibility of Bypassing the Inferior Alveolar Nerve While Placing Dental Implants

Mohammed G Sghaireen [1], Kumar Chandan Srivastava [2], Deepti Shrivastava [3], Kiran Kumar Ganji [3], Santosh R. Patil [2], Anas Abuonq [4], Mohammed Assayed Mousa [1], Najla Dar-Odeh [5,6], Ghazi M. Sghaireen [7], Mohammad Amjad Kamal [8,9,10,*] and Mohammad Khursheed Alam [11]

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check for updates

Abstract: A high rate of nerve injury and related consequences are seen during implant placement in the posterior mandibular arch. An approach has been proposed to avoid nerve injury by dodging the inferior alveolar nerve (IAN) while placing an implant. A prospective study with a total of 240 CBCT (cone beam computed tomography) images of patients with three dentate statuses, namely, edentulous (group I), partially edentulous (group II) and dentate (group III) were included in the study. The nerve path tracing was done on CBCT images with On-demand 3D software. The three dimensions, i.e.,

Inferior Alveolar Nerve Lateralization and Transposition for Dental Implant Placement. Part I: a Systematic Review of Surgical Techniques

Boris Abayev[1], Gintaras Juodzbalys[1]

[1]Department of Maxillofacial Surgery, Lithuanian University of Health Sciences, Kaunas, Lithuania.

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ABSTRACT

Objectives: The purpose of this first part of a two-part series was to review the literature concerning the indications, contraindications, advantages, disadvantages and surgical techniques of the lateralization and transposition of the inferior alveolar nerve, followed by the placement of an implant in an edentulous atrophic posterior mandible.
Material and Methods: A comprehensive review of the current literature was conducted according to the PRISMA guidelines by accessing the NCBI PubMed and PMC database, academic sites and books. The articles were searched from January 1997 to July 2014 and comprised English-language articles that included adult patients between 18 and 80 years old with minimal residual bone above the mandibular canal who had undergone inferior alveolar nerve (IAN) repositioning with a minimum 6 months of follow-up.
Results: A total of 16 studies were included in this review. Nine were related to IAN transposition, 4 to IAN lateralization and 3 to both transposition and lateralization. Implant treatment results and complications were presented.
Conclusions: Inferior alveolar nerve lateralization and transposition in combination with the installation of dental implants is sometimes the only possible procedure to help patients to obtain a fixed prosthesis, in edentulous atrophic posterior mandibles. With careful pre-operative surgical and prosthetic planning, imaging, and extremely precise surgical technique, this procedure can be successfully used for implant placement in edentulous posterior mandibular segments.

Keywords: alveolar bone atrophy; dental implants; fifth cranial nerve injury; jaw surgery; mandibular nerve; paresthesia.

Novel nerve repair

- RESULTS DPSCs highly expressed several specific neural markers, including GFAP, S100, Nestin, P75, and NF200, and were inclined toward neural differentiation. Furthermore, neural-induced DPSCs (N-DPSCs) could express neurotrophic factors, including NGF, BDNF, and GDNF. The secretions of N-DPSCs could enhance the proliferation and migration of Schwann cells. In vivo, both DPSC and N-DPSC implants alleviated gastrocnemius muscle atrophy. However, in terms of anatomy and motor function, as shown by H&E staining, immunofluorescent staining, and walking track analyses, the repair effects of N-DPSCs were more sustained, potent, and effective than those of DPSCs and the controls.
- CONCLUSION In summary, this study demonstrated that DPSCs are inclined to differentiate into neural cells. N-DPSCs express neurotrophic proteins that could enhance the proliferation and migration of SCs. Furthermore, our results suggested that NDPSCs could help crushed nerves with functional recovery and anatomical repair in vivo. Thus, DPSCs or N-DPSCs could be a promising therapeutic cell source for peripheral nerve repair and regeneration.

World Journal of Stem Cells

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ISSN 1948-0210 (online)

ORIGINAL ARTICLE

Basic Study

Neurotrophic effects of dental pulp stem cells in repair of peripheral nerve after crush injury

Dian-Ri Wang, Yu-Hao Wang, Jian Pan, Wei-Dong Tian

ORCID number: Dian-Ri Wang 0000-0001-8128-7637; Yu-Hao Wang 0000-0002-3745-7624; Jian Pan 0000-0003-1346-048X; Wei-Dong Tian 0000-0003-0175-1132.

Author contributions: Wang DR and Pan J contributed to the conception and design of the study, collection and/or assembly of data, data analysis and interpretation, and manuscript writing; Wang DR performed the *in vivo* and *in vitro* experiments; Wang YH participated animal experiments; all authors read and approved the final version of the manuscript.

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Institutional review board statement: All experiments involving humans followed the guidelines of the Ethics Committee of West China College of Stomatology.

Institutional animal care and use committee statement: All experiments involved animals were proved by the Ethics

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Abstract

BACKGROUND

Nerve diseases and injuries, which are usually accompanied by motor or sensory dysfunction and disorder, impose a heavy burden upon patients and greatly reduce their quality of life. Dental pulp stem cells (DPSCs), derived from the neural crest, have many characteristics that are similar to those of neural cells, indicating that they can be an ideal source for neural repair.

AIM

To explore the potential roles and molecular mechanisms of DPSCs in crushed nerve recovery.

METHODS

DPSCs were isolated, cultured, and identified by multilineage differentiation and flow cytometry. Western blot and immunofluorescent staining were applied to analyze the expression levels of neurotrophic proteins in DPSCs after neural induction. Then, we collected the secretions of DPSCs. We analyzed their effects on RSC96 cell proliferation and migration by CCK8 and transwell assays. Finally, we generated a sciatic nerve crush injury model *in vivo* and used the sciatic function index, walking track analysis, muscle weight, and hematoxylin & eosin (H&E) staining to further evaluate the nerve repair ability of DPSCs.

Implants most common cause Younger patients (P = .041) and patients without dysesthesia (P = .019) were more likely to achieve FSR. Higher proportion of early repair group achieved FSR, although not statistically significant (P = .068).

Factors Affecting Functional Sensory Recovery After Inferior Alveolar Nerve Repair Using the Nerve Sliding Technique

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Purpose: The nerve sliding technique (NST) was introduced for repairing inferior alveolar nerve (IAN) defect and overcoming the disadvantages of conventional surgical treatment methods such as nerve graft. This study was conducted to identify factors associated with functional sensory recovery (FSR) following inferior alveolar nerve repair using the NST.

Patients and Methods: This was a retrospective cohort study including all patients who underwent IAN repair using the NST at Seoul National University Dental Hospital, Department of Oral and Maxillofacial Surgery from February 2009 to March 2020. The damaged part of the IAN was excised, and the incisive branch was transected intentionally to perform direct anastomosis without tension. Cox proportional hazard analysis was utilized to determine the relationships between predictor variables (age, gender, chief complaints, preoperative sensory results, duration from injury to repair, length of nerve tissue resected during the procedure, and neuroma formation) and outcome variable (time to FSR).

Results: The sample was composed of 16 patients with a mean age of 56.1 ± 10.1 years, 25% were males and 75% were females. The mean nerve gap deficit was 7.69 mm (3-15 mm). Ten patients (62.5%) achieved FSR with a median time from operative treatment to FSR of 84.5 days. Dental implant placement was found as the main cause for IAN injury (93.8%) and 56.2% of patients complained of hypoesthesia and dysesthesia. Factors associated with time to FSR at 1 year were age, chief complaint, and early repair. Younger patients (P = .041) and patients without dysesthesia (P = .019) were more likely to achieve FSR. Higher proportion of early repair group achieved FSR, although not statistically significant (P = .068).

Conclusions: The use of NST in repair of IAN defects up to 15 mm achieved 62.5% FSR. Younger age and absence of dysesthesia were associated with higher FSR.

- -is systematic review provided an assessment of the anatomy of the mental foramen. -e mental foramen was located mostly between the two premolars (between 50.4% and 61.95%) or apically to the second premolar (from 50.3% to 57.9%). -e mean diameter of the mental foramen was bigger in males than in females; the difference between them could reach 0.62 mm. -e anterior loop seemed to be longer in males (between 0.87 ± 1.81 and 7.25 ± 2.02 mm) than in females (between 0.81 ± 1.18 and 6.52 ± 1.63 mm) and with the presence of teeth (from 0.91 ± 1.18 to 2.55 ± 1.28 for dentate people and from 0.25 ± 0.61 to 2.40 ± 0.88 mm for edentate population). -e anterior loop and the accessory mental foramina were detected more frequently with CBCT than panoramic X-ray: only between 0.0 and 48.6% AMFs detected with CBCT were also seen with panoramic images. Clinical Significance. -e mental foramen (MF) is an important landmark for local anesthesia and surgical and implantology procedures. Its location, morphology, and anatomical variations need to be considered to avoid mental nerve injury. -e aim of this review is to evaluate the mental foramen using CBCT through a systematic literature review to improve knowledge of this complex area for the clinician

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Review Article

Evaluation of Mental Foramen with Cone Beam Computed Tomography: A Systematic Review of Literature

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Purpose. The aim of this systematic review is to assess whether the anatomy of mental foramen is precisely evaluable with cone beam computed tomography (CBCT) before implantation in humans. *Methods*. A systematic review was carried out to evaluate the anatomy of mental foramen (size, position, symmetry, anterior loop, and accessory mental foramen or multiple mental foramina). According to Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines, an electronic search of three databases (Medline, Web of Science, and Cochrane Library) was undertaken until June 2020 and was supplemented by manual searching. Two reviewers will independently perform the processes of study inclusion, data extraction, and quality assessment. Systematic reviews, studies about children, and case reports were excluded. Only studies using CBCT to do preoperative evaluation were selected. *Results*. From 728 potentially eligible articles, 72 were included in the qualitative analysis and quantitative synthesis. This systematic review provided an assessment of the anatomy of the mental foramen. The mental foramen was located mostly between the two premolars (between 50.4% and 61.95%) or apically to the second premolar (from 50.3% to 57.9%). The mean diameter of the mental foramen was bigger in males than in females; the difference between them could reach 0.62 mm. The anterior loop seemed to be longer in males (between 0.87 ± 1.81 and 7.25 ± 2.02 mm) than in females (between 0.81 ± 1.18 and 6.52 ± 1.63 mm) and with the presence of teeth (from 0.91 ± 1.18 to 2.55 ± 1.28 for dentate people and from 0.25 ± 0.61 to 2.40 ± 0.88 mm for edentate population). The anterior loop and the accessory mental foramina were detected more frequently with CBCT than panoramic X-ray: only between 0.0 and 48.6% AMFs detected with CBCT were also seen with panoramic images. *Clinical Significance*. The mental foramen (MF) is an important landmark for local anesthesia and surgical and implantology procedures. Its location, morphology, and anatomical variations need to be considered to avoid mental nerve injury. The aim of this review is to evaluate the mental foramen using CBCT through a systematic literature review to improve knowledge of this complex area for the clinician.

Neuropathic pain does not respond to surgery

Surgical impact on NP

Lingual nerve repair and recurrence of neuropathic pain

27 patients Various procedures

ANESTHESIA/FACIAL PAIN

Factors Determining Outcome After Trigeminal Nerve Surgery for Neuropathic Pain

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Purpose: Most patients who seek relief from trigeminal neuropathic pain by trigeminal microneurosurgery techniques do not show permanent pain relief after surgery. However, a small number of patients have permanent relief after surgery. The objective of this study was to determine factors that might be associated with the resolution, decrease, or recurrence of neuropathic pain after trigeminal nerve surgery in those patients who present with neuropathic pain before surgery.

Patients and Methods: An ambispective study design was used to assess patients who underwent trigeminal nerve repair of the inferior alveolar and lingual nerve who had documented neuropathic pain before surgery from 2006 through 2014. The primary endpoint was the difference in pain intensity at 3, 6, and 12 months after surgery compared with presurgical intensity levels. Explanatory variables, including age at surgery, gender, site of nerve injury, etiology of nerve injury, classification of nerve injury, duration from injury to repair, health comorbidities, and type of repair performed, were evaluated as potential factors in the outcomes. Wilcoxon signed rank analysis was used to compare demographic and injury characteristics of patients who had pain relief, partial pain relief, and no pain relief after surgery. Two-way analysis of variance and logistic regression analysis were used to evaluate the association between neuropathic pain and the explanatory variables.

Results: Twenty-eight patients met the inclusion criteria. Three cohorts of patients were identified and analyzed. The no-recurrence cohort included 7 patients who had neuropathic pain before surgery that was resolved with surgery. The complete-recurrence (CR) cohort included 10 patients who had neuropathic pain before surgery and complete recurrence of pain intensity after surgery. The incomplete-recurrence (ICR) cohort included 11 patients who had neuropathic pain before surgery and partial recurrence of pain intensity after surgery. There was no statistical difference in preoperative pain intensity levels among the 3 cohorts ($P = .16$), but there were statistical differences at 3 months ($P = .007$), 6 months ($P < .0001$), and 12 months ($P < .0001$). There were no statistical differences between the CR and ICR cohorts at 3 months ($P = .502$), 6 months ($P = .1$), and 12 months ($P = .2$). There was no effect by age, gender, injury type, Sunderland classification, injury etiology, duration from injury to repair, health comorbidity, or repair type on the outcome.

Conclusions: The recurrence of neuropathic pain after trigeminal nerve repair for neuropathic pain is likely multifactorial and might not depend on factors that normally affect sensory recovery in patients who have no neuropathic pain (ie, age, duration of injury, type of injury, or repair type) and undergo trigeminal nerve surgery. These differences indicate that the understanding of trigeminal neuropathic pain is

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If surgical reconstruction is used to treat allodynia, this often results in a decrease of complaints but symptoms almost never completely resolve.[10] Zuniga[26] reported only 3% of patients with neuropathic pain before surgery will completely recover following surgery. Occasionally, reconstruction can worsen complaints.[9,26]

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the 3 cohorts ($P = .16$), but there were statistical differences at 3 months ($P = .007$), 6 months ($P < .0001$), and 12 months ($P < .0001$). There were no statistical differences between the CR and ICR cohorts at 3 months ($P = .502$), 6 months ($P = .1$), and 12 months ($P = .2$). There was no effect by age, gender, injury type, Sunderland classification, injury etiology, duration from injury to repair, health comorbidity, or repair type on the outcome.

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