Assessment of a patient with trigeminal neuropathic pain

Tara Renton

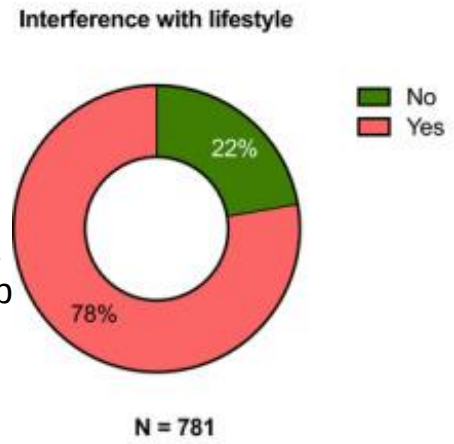
A patient presents with ‘nerve injury’

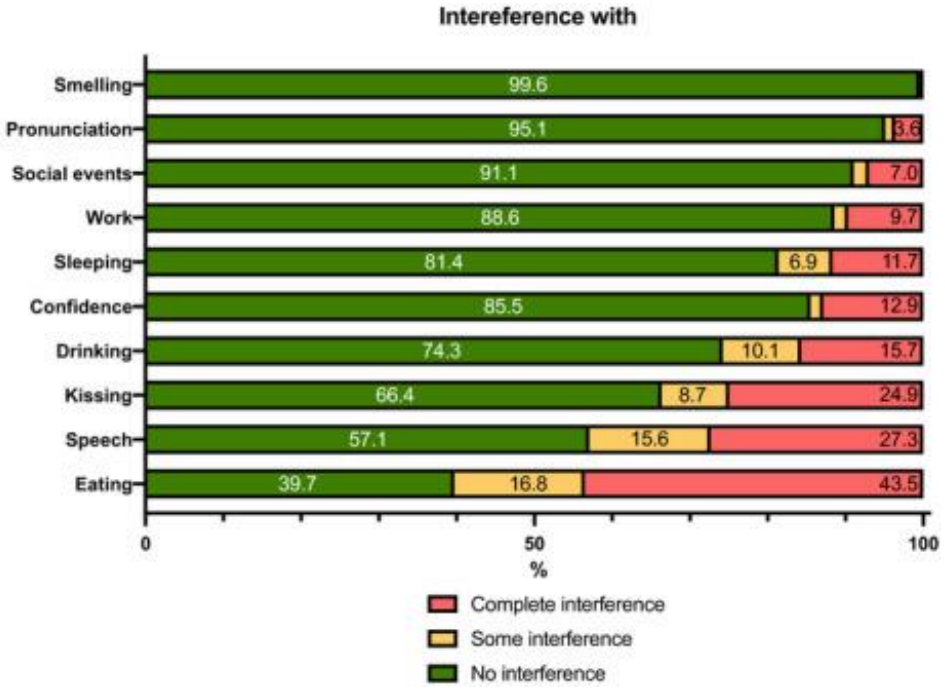
- Holistic assessment
  - Assess functional limitations
  - Assess AXIS 2 psychosocial impact
- Confirm diagnosis
  - Diagnostic criteria
- Then you can treat the patient with post traumatic neuropathy (PTN) optimally

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

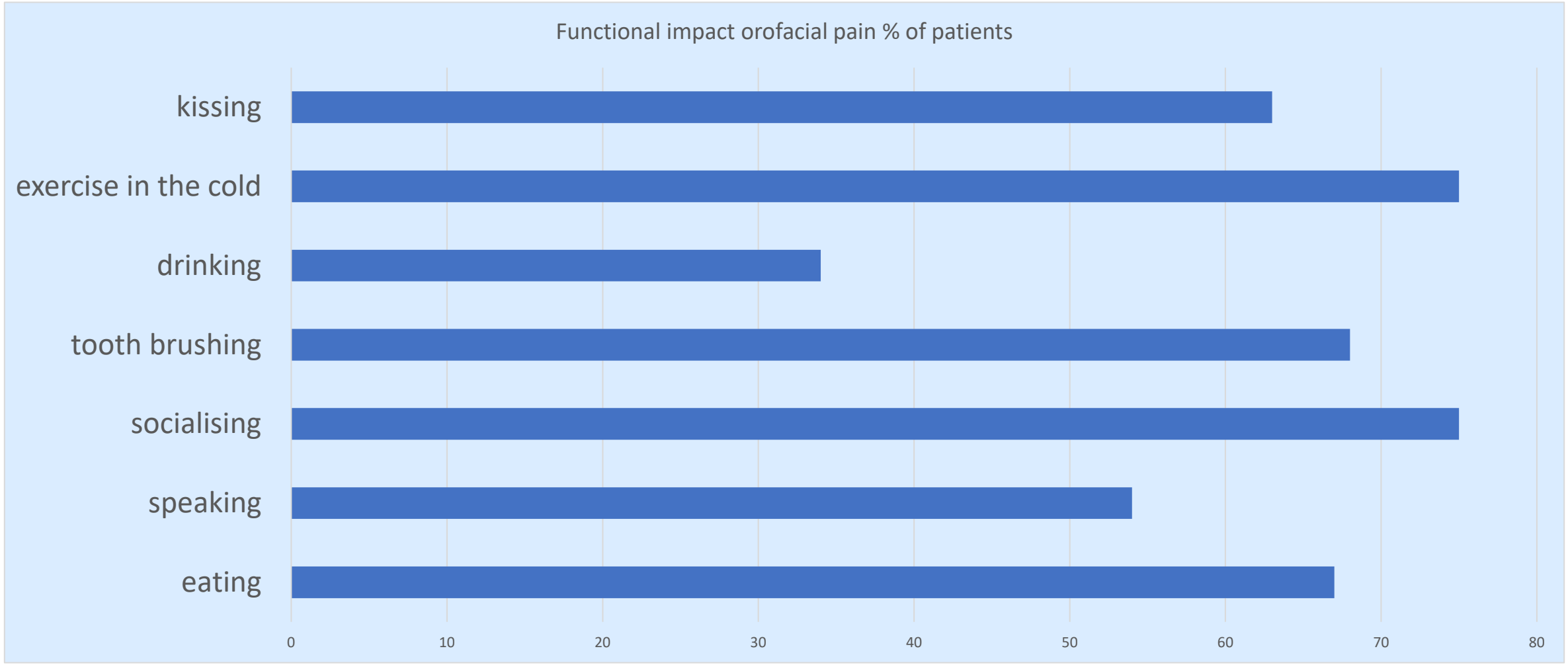
Renton T, Dawood A, Shah A, Searson L, Yilmaz Z. Post-implant case series. Br Dent J. 2012 Jun 8;212(11):E17. doi: 10.1038/sj.b





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.

Functional impact of post traumatic neuropathy



Renton T, Yilmaz Z. Profiling of patients presenting with posttraumatic neuropathy of the trigeminal nerve. J Orofac Pain. 2011 Fall;25(4):333-44. PMID: 22247929.

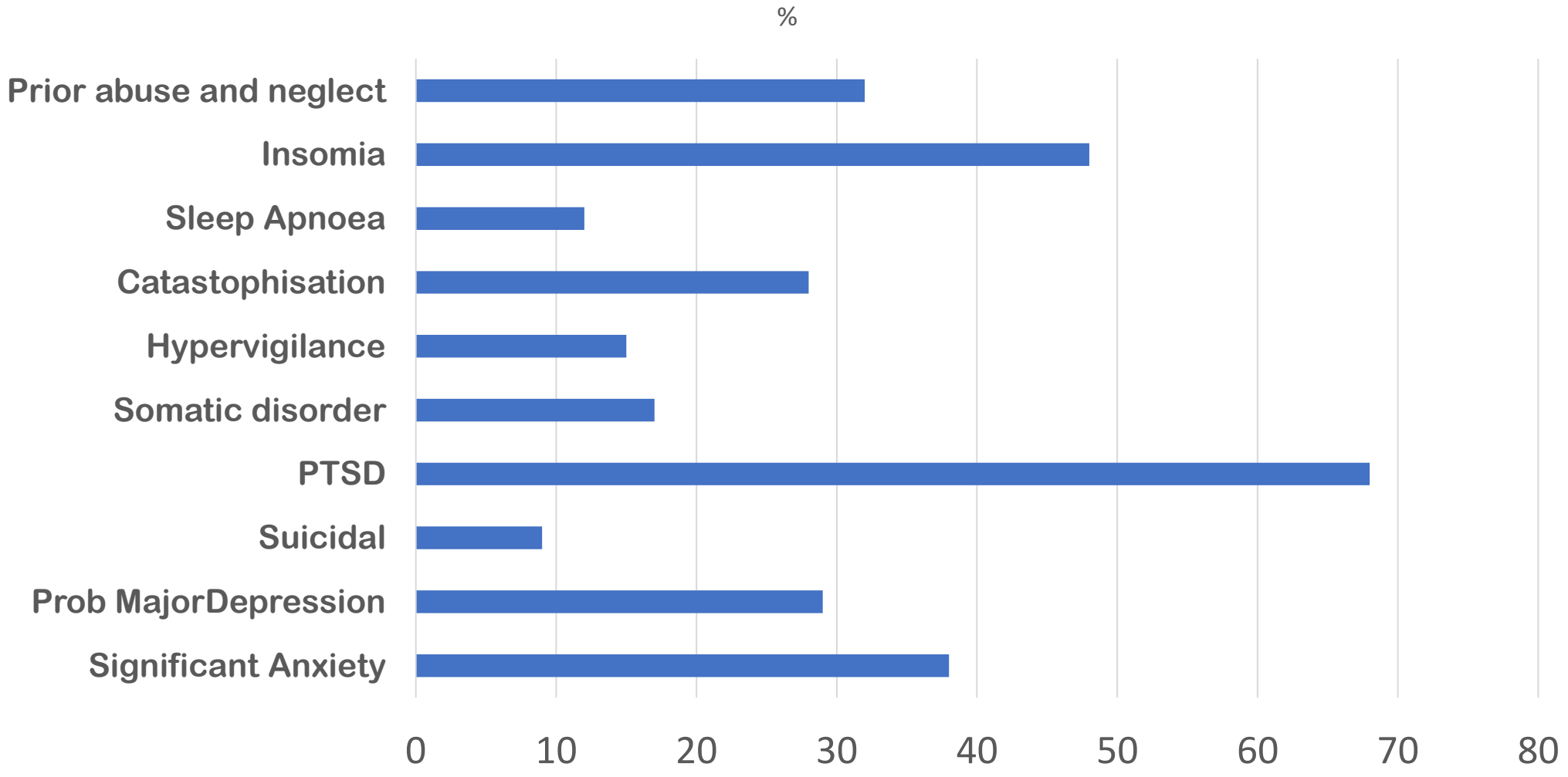
AXIS II pre consultation extensive psychosocial assessment

- All patients:
  - **EQ-5D**
  - **GAD7 generalised ANXIETY disorder**
  - **PHQ9 Patient Health Questionnaire- depression**
  - **SF-MPQ-2 Short-form McGill** Pain Questionnaire-2
  - **BPI Facial pain**
  - **ISI insomnia**
  - **STOP Bang and International sleep inventory** ISI (sleep)
  - **IES-R** (abuse)
  - **Hit-6 migraine**
  - **Fibromyalgia-19 (ARA)**

  - ~~(PHQ 15 MULTIDIMENSIONAL SCALE OF PERCEIVED SOCIAL SUPPORT)~~
  - ~~**Global Chronic Pain Scale (GCPS)**~~
  - ~~**Post traumatic neuropathic scale**~~
- Dash board with red flags suicidal thoughts/ depression, anxiety and somatic disorders

Severe Anxiety
Probable Major depression
Somatic disorder
PTSD
Likely NP

What AXIS II diagnoses patients with PTN?



% of patients presenting with OFP

Diagnosis of Post traumatic neuropathy (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 chan in cold sensation)
   c. Hypersensitivity to brush or pinprick in or around the painful
4. No other condition (eg, inflammation, tumor) better explains tl pattern of the clinical features (eg, radiculopathy) that could pl bly account for persisting pain in the affected dermatome or dermatomes.

*This pain may occur even if there was a deliberate attempt to spare the nerves crossing the surgical area (eg, in breast surgery).
†There is a spontaneous decline in reporting of pain >12 mo after su trauma. Relevant citations in support of these diagnostic criteria are Bru Duffy et al,[77] Guo et al,[107] Haldar et al,[109] Pappagallo et al,[187] Teerijoki- et al,[224] and Wildgaard et al.[247]

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Focus Article

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

Roy Freeman,* Robert Edwards,† Ralf Baron,‡ Stephen Bruehl,§ Giorgio Cruccu,¶ Robert H. Dworkin,‖ and Simon Haroutounian**

*Center for Autonomic and Peripheral Nerve Disorders, Beth Israel Deaconess Medical Center, Boston, MA
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The IASP classification of chronic pain for ICD-11: chronic neuropathic pain

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

PTNP diagnostic criteria

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   b. Hyposens
      in cold se
   c. Hypersens
4. No other co
   pattern of th
   bly account
   dermatome

*This pain may
nerves crossing t
†There is a spor
trauma. Relevan
Duffy et al.[77] G

Traumatic event = onset

Neuropathic area

Positive signs Allodynia / Hyperalgesia = hyperaesthesia

Negative signs Anaesthesia/paraesthesia = hypoaesthesia

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.

Probability 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

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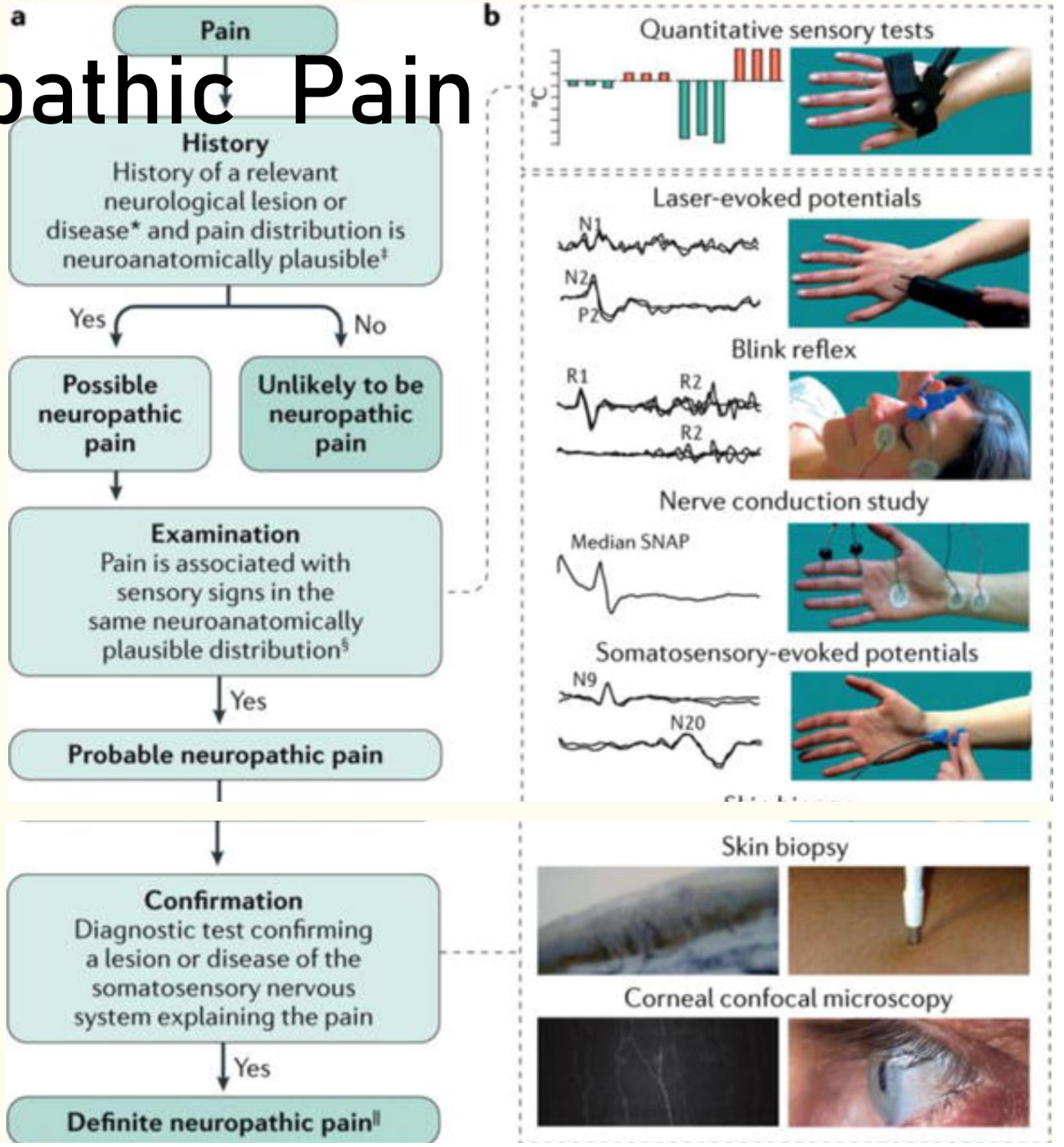
Neuropathic pain: an updated grading system for research and clinical practice

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



Grading of neuropathic pain

Comprehensive Review

PAIN

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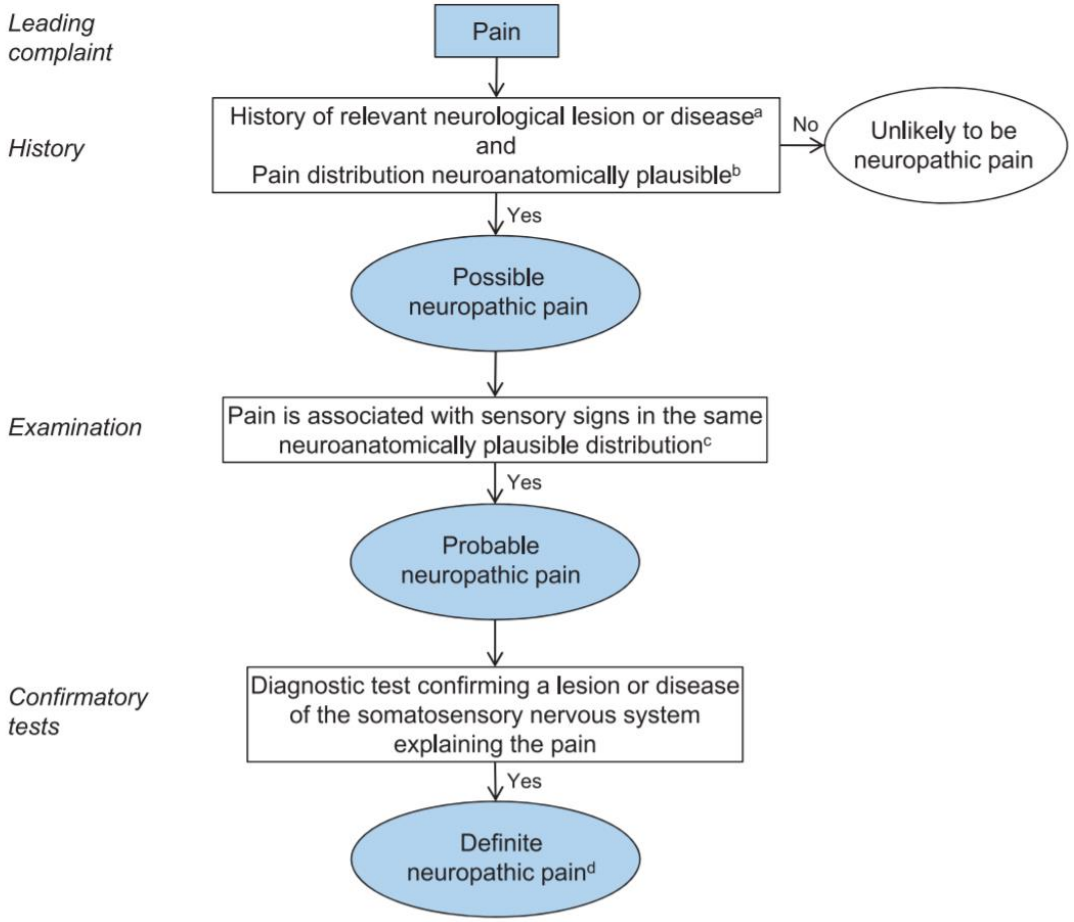


Compared to the grading system published in 2008, we have (1) changed the order of the grading criteria to better reflect clinical practice. (2) annotated the terms used to improve clarity. (3) recognized the role of screening tools (questionnaires) in neuropathic pain evaluation. (4) emphasized that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

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/dull**
  - **Elicited neuralgic/sharp/ shooting**
  - **Or a combination of both**

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

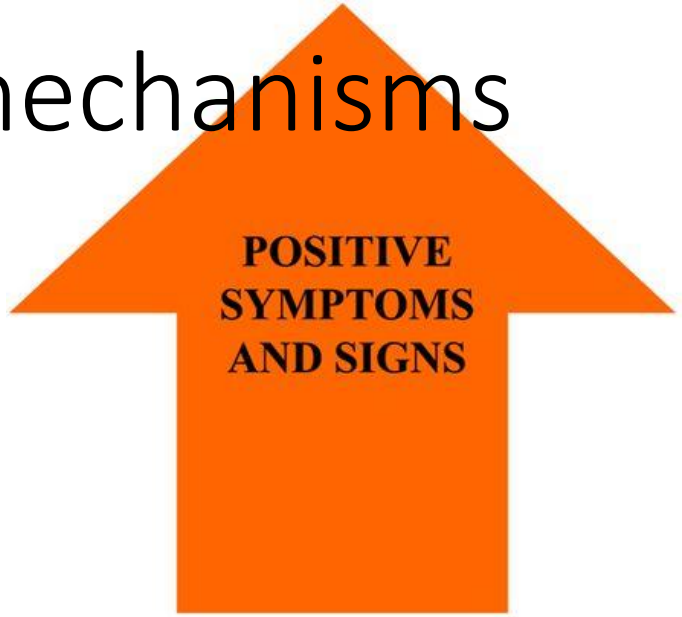
Paresthesia	An abnormal sensation, whether spontaneous or evoked
Dysesthesia	An unpleasant sensation, whether spontaneous or evoked
Hypoesthesia	Decreased sensitivity to stimulation (tactile or thermal; both are frequent)
Hyperesthesia	Increased sensitivity to stimulation (tactile or thermal; both are 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

Neuropathic area

+

Positive or negative signs



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

Hyperaesthetic

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)

Hypoaesthetic

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

Exclude non-traumatic Neuropathic pain

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; and MOUNZER Y. KASSAB, MD, *Michigan State University College of Human Medicine, East Lansing, Michigan*

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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.

COLOR

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

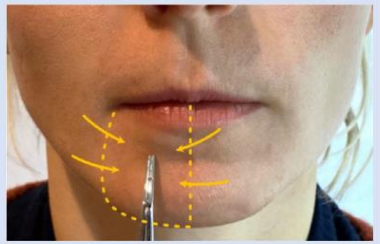
Area affected

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

Neuropathic area?

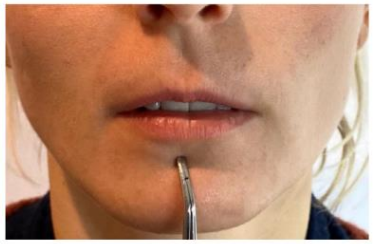
Map out the area and record pictorially or by photograph using pen marks on patient's face.
Estimate the % or extra-oral dermatome is affected by the neuropathy.

(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.



Hypo or Hyperaesthetic??

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

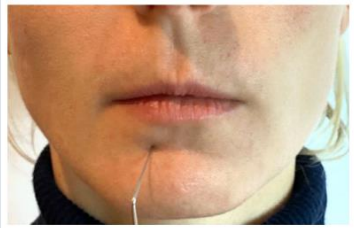
Light touch

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.

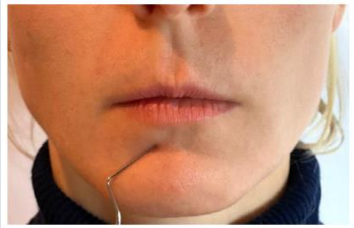
Tactile / mechanical allodynia?

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.



Tactile / mechanical hyperalgesia?



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.

Apply Cold metal mirror back Thermal allodynia

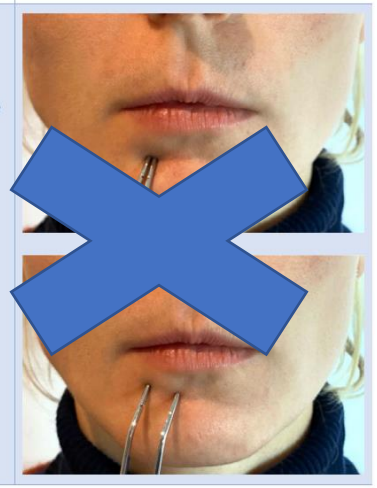


Figure 2

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ORIGINAL ARTICLE

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

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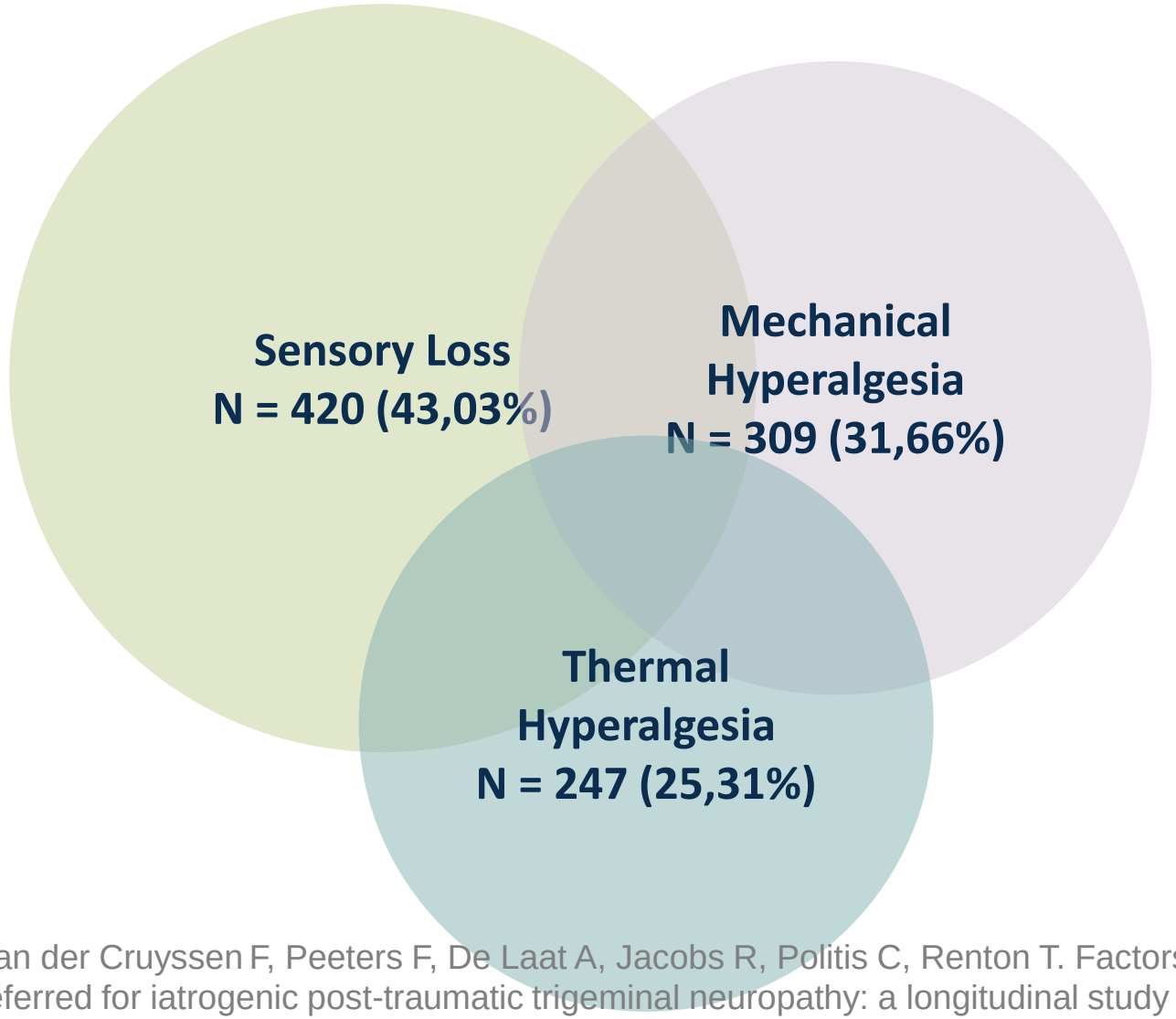
Neuropathic pain Trigeminal PTN (N = 976)



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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.

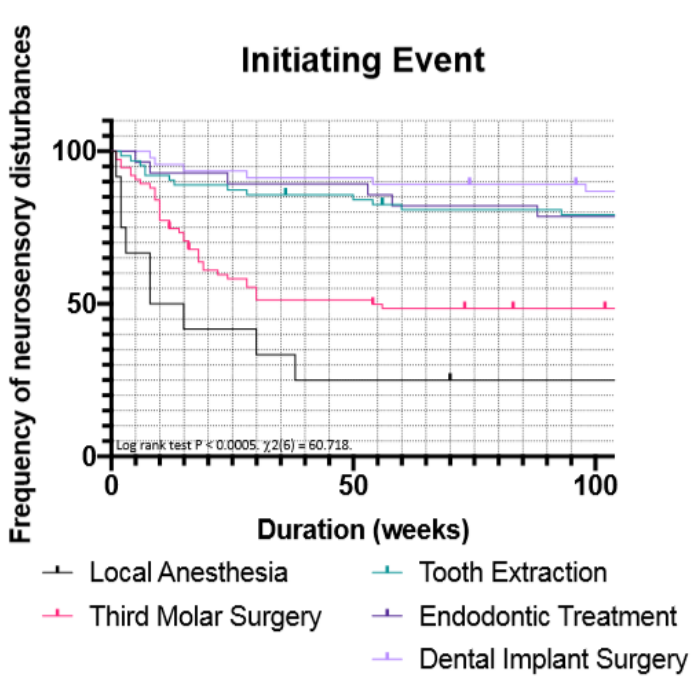
Conclusion: Patients with painful PTN had different clinical profiles and lower QoL scores than those with non-painful PTN. Sensory profiles may provide important prognostic and therapeutic information; however, more research is needed to assess the clustering procedure and link these clusters to therapeutic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

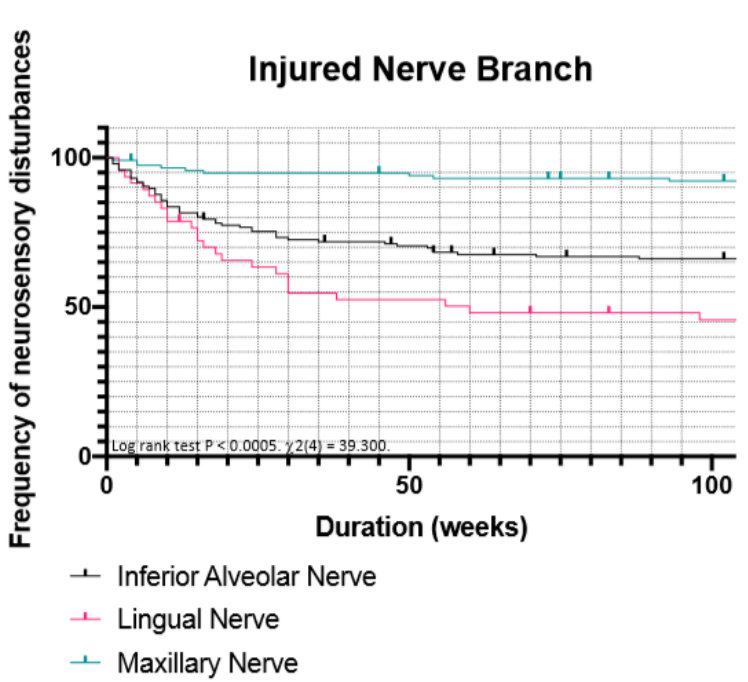
KEYWORDS
diagnosis, neuropathic pain, quality of life, trigeminal nerve, trigeminal nerve disorder

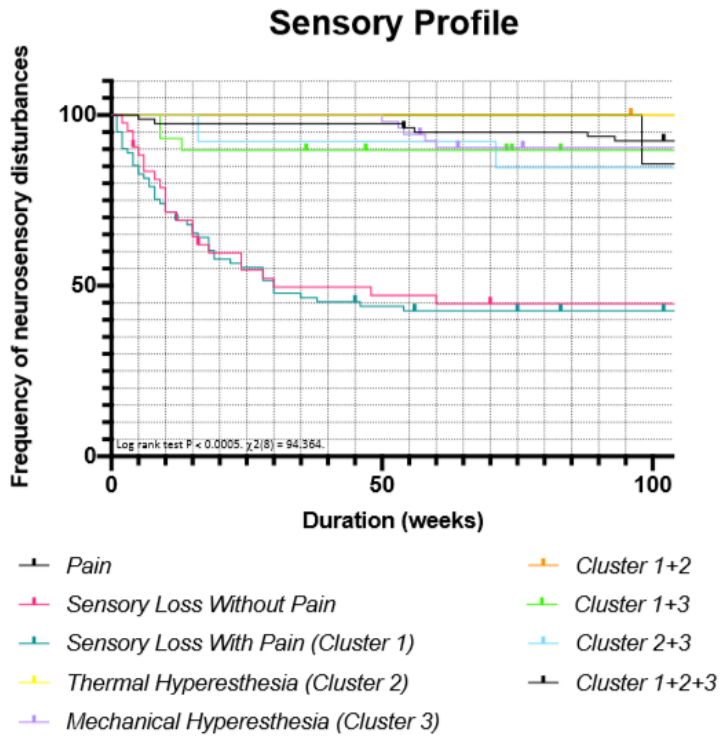
Van der Cruyssen F, Peeters F, De Laat A, Jacobs R, Politis C, Renton T. Factors affecting evolution of symptoms and quality of life in patients referred for iatrogenic post-traumatic trigeminal neuropathy: a longitudinal study in two tertiary referral centers in UK and Belgium. Pain 2020
Van der Cruyssen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

Prognosis of Post Traumatic Neuropathy N=1331

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







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